RECOVERY OF POSTURAL CONTROL AFTER AN ACUTE UNILATERAL VESTIBULAR LESION IN HUMANS

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Abstract — Postural control during stance was investigated using the EQUITEST® system in 10 patients during recovery after an acute unilateral vestibular lesion and was compared to the time course of recovery of the static and dynamic vestibulo-ocular imbalance. During the acute phase the patients showed a characteristic pattern with normal upright stance as long as at least one accurate sensory input (visual or somatosensory) was provided and severe postural disturbances when they had to rely primarily on vestibular afferences. Both static vestibulo-ocular and vestibulo-spinal balance recovered very fast, showing basically normal results on postural testing within about 2 weeks after the lesion. Thereafter, no pathological pattern was detectable during postural testing even in patients with persistent complete unilateral vestibular lesions. Reflexive postural responses to unexpected rapid displacements of the support surface seemed not to be influenced by vestibular imbalance even in the acute phase of the lesion.

Keywords — posture; unilateral vestibular lesion; vestibulo-spinal reflexes; vestibulo-ocular reflex.

Introduction

Gait and balance in the upright position are dynamic processes that require continuous adjustments based on visual, vestibular, and proprioceptive information. This redundancy allows for almost normal equilibrium as long as at least one system is working adequately, even though the three control loops have working ranges in terms of their frequency response that only partly overlap. To test one of these control loops in isolation, one has to reduce or to abolish the contribution of the two others. For clinical testing of vestibular afferences this is usually done by the so-called "sharpened Romberg test." However, this method is highly subjective and does not really exclude proprioceptive inputs. Recently, methods of measuring postural disturbances under different sensory conditions have been developed to test the vestibular input (1,2). With this tool we were able to monitor closely the recovery of vestibulo-spinal control in 10 patients with acute unilateral peripheral vestibular hypofunction. The aim of the study was threefold: (1) to determine the early effects (as soon as 3 days after the onset of symptoms) of acute unilateral vestibular hypofunction on postural control; (2) to monitor the time course of postural recovery; (3) to compare the time course of recovery of postural control and of the static and dynamic vestibulo-ocular imbalances.

Methods

Ten patients (2 males, 8 females, mean age 37.6 ± 13.5) with acute unilateral vestibular lesions, due to suspected vestibular neuropathy in 9 cases and to surgical removal of an acoustic neuroma in one case, were investigated during the first 2 months after the onset of symptoms. Five of the patients had left-sided lesions, and 5 had right-sided lesions.

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The patient with the acoustic neuroma was tested also prior to the surgical procedure and showed at that time no spontaneous nystagmus, normal caloric and rotatory responses, and normal vestibulo-spinal functions.

Body sway was assessed on a movable platform and within a movable visual surround using the EQUITEST® system (1,2). The patients were tested under 6 different sensory conditions. Under the first three conditions, the platform support surface remained fixed. In condition 1 the visual surround was fixed also, in condition 2 the patients had to close their eyes, and in condition 3 the visual surround was moved proportionally to body sway. In conditions 4 to 6 the platform support surface was moved proportionally to body sway. In condition 4 the visual surround was fixed, in condition 5 the patients had to close their eyes, and in condition 6 the visual surround was also moved proportionally to body sway. Conditions 5 and 6 test the vestibular control loop in isolation, since subjects were largely deprived of proprioceptive input and since vision of body movement relative to the surround was almost entirely disrupted or distorted. Dynamic equilibrium was quantified by the extent to which the patient's sway remained within the limits of stability during a 20 s test period and was expressed by a score between 0 and 100. The score compares a maximum sway deviation during the trial (the difference between maximum forward and maximum backward sway angles) to the theoretical maximum of 12° between the forward and backward stability limits. Using this calculation, patients who have little sway receive scores near 100, while patients who approach their limits of stability or fall receive low scores or zero for a fall.

In a second measurement, strategy scores were calculated. The strategy score describes the extent to which the patient uses ankle versus hip joint motions to correct for sway. Hip motion is detected by the amount of shear force exerted on the platform. Movement strategy values near 100 occur with little if any hip sway, while values approach zero when the subject makes maximum use of hip flexion/extension.

Finally, initial and dynamic anterior–posterior orientation (sway angle) are calculated. This measure quantifies the extent to which the patient maintains the center of mass over the center of the area of foot support just before each sensory test (initial orientation) and as an average during the 20-s trial (dynamic orientation). Forward–backward sway angles in degrees are based on zero orientation (perfectly vertical) with the center of mass above a midpoint halfway between the heels and toes of the feet. The orientation is negative when the center of mass is positioned behind the center of the area of feet support. Orientation is positive when the center of mass is in front of this point.

Additionally, movement coordination was tested. This test assesses the patient's ability to coordinate automatic movement responses to unexpected sway perturbations of the surface. Sway is induced by unexpectedly displacing the foot plates backward or forward. The complete series consists of three sizes each of forward and backward displacements. For each translation size, amplitude is adjusted to compensate for differences in subject height. The overall effect of these adjustments is to make the angular amplitudes of anterior–posterior sway produced by a given size translation the same for individuals of all heights (small: 0.7°, medium: 1.8°, large: 3.2°). Delay times between trials are random between 1.5 and 2.5 s. The following parameters are calculated: (1) Lateral sway under static and dynamic conditions is calculated as a dimensionless measure of the relative weight bearing by the left and right leg before each trial (static) and during active force response (dynamic). Equal weight bearing or equal active force responses result in a sway score of 0. If the right leg bears more weight or produces relatively more active force, the value is positive. If the right leg bore the total weight, the sway score would be 100. If the left leg bore the total weight, the score would be -100. (2) Active force response latencies are calculated separately for each leg for medium and large perturbations. Latency is the time in milliseconds following onset of a surface translation at which the subject first begins to actively resist the sway. (3) Strategy scores are calculated the same way as for the sensory coordina-
Posture Control after Unilateral Lesion

Tests. The abnormal ranges for test scores have been defined as those below those achieved by 95% (about 2 standard deviations below the mean) of individuals in the age-matched normal population (EQUITEST® Normal Subject Study, Nashner, personal communication, 1990).

To assess static and dynamic vestibulo-ocular disturbances, open-eye movements were recorded by DC-electrooculography in a dark room using silver-silver-chloride electrodes. Static vestibular imbalance was measured by the amount of spontaneous nystagmus (slow phase velocity [°/s]). Dynamic imbalances were evaluated by measuring the maximum slow phase velocity of postrotatory nystagmus after constant velocity rotations about the earth vertical axis with 180°/s in both directions and expressed by a directional preponderance value (difference of the two responses divided by the sum of the two responses; absolute values; corrected for spontaneous nystagmus). Finally, the degree of the unilateral vestibular hypofunction was assessed by routine caloric testing including 44°C and ice-water calorics and expressed by a canal paresis score (CaIP), which is defined as the difference of the combined warm and cold caloric responses between the two ears divided by the sum of the responses (absolute values).

**Results**

Patients were tested first between day 3 and day 13, then between day 7 and day 25, and finally between day 43 and day 68 after the onset of symptoms. Table 1 shows the recording days for each patient. The patients were divided into two groups according to the side of their lesion. The patients of group R (patients 1 through 5) had a right-sided lesion, the patients of group L (patients 6 through 10) had a left-sided lesion.

### Table 1. Summary of the Days of Measurement after the Onset of Symptoms for Each Patient

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For initial anterior–posterior orientation of the center of the mass over the center of feet support, the majority of the patients (9 out of 10) preferred a position with the center of mass slightly behind the center of foot support. The only patient (9) with the center of mass consistently in front of the center of feet support was the patient who had undergone surgery for an acoustic neuroma. Normal individuals tend to lean approximately one-half degree forward of the perfectly vertical position (the center of body mass above a point halfway between the heels and toes of the feet) both before and during sensory testing. Most patients were in the normal range of anterior–posterior body orientation (between 2.5° and -1.5°). Only one patient (4) consistently exceeded the normal limits, with backward displacement of the center of mass of up to
Figure 1. Test scores (ordinate) for equilibrium (a and b) and for strategy (c and d) with the six different sensory conditions (abscissa; 1 through 6) for the five patients (1 through 5) of group R (right-sided lesions) and the five patients (6 through 10) of group L (left-sided lesions) at the first recording session are depicted. The crosshatched bar denotes the 5th percentile of normal behavior (95% of normal subjects have test results above this value). The strategy score identifies the extent to which the patient uses ankle versus hip joint motions to correct for sway. While most patients had close to normal scores as long as at least one accurate sensory signal (visual or somatosensory) was provided, most patients showed severe postural disturbances or even fell when they had to rely solely on the vestibular input.

-3°. For the average dynamic anterior-posterior orientation, similar results were found, with 9 out of 10 patients achieving normal scores on most or all tests.

**B. Movement coordination.** Lateral sway under static and dynamic conditions was expressed as a dimensionless measure of the relative weight bearing by the left and right leg before each trial of perturbation (surface translation) and during active force response.

No consistent asymmetry could be detected. In particular, there was no correlation between the side of the lesion and the side bearing more weight. Lateral sway under static conditions was within normal limits for all 9 subjects in whom this test was performed at the first recording day (movement coordination test was not performed in patient [2]). Lateral sway under dynamic conditions exceeded the normal range in some cases in individual trials (test 3 in patient 5, test 3 and 4 in patient 7, and test 6 in patient 8). In most patients, however, the lateral sway under dynamic conditions was within normal limits in most of the trials.

Latencies of active force response for the right leg and for the left leg were within nor-
nal limits for most of the patients. However, the results are compromised by the fact that values are missing for several cases in which the analysis program could not detect a clear onset of the active force response. In the group with right-sided lesions, only patient 1 showed increased latencies for medium and large forward-perturbations (tests 5 and 6) for both the right and the left leg, with about equal values for the two legs. Two other patients (3,4) showed increased latencies for the right leg for large forward-perturbations (test 6). In the group with left-sided lesions no increased latencies were found for the right leg. For the left leg one patient (8) showed increased latencies for medium and large forward-perturbations (tests 5 and 6), and another patient (6) only for medium forward-perturbations (test 5). All the other patients had latencies within normal limits. There was no difference between the latencies of the right and left legs, neither for group R with a mean latency for medium and large perturbations for the right leg of 167.5 ± 43.3 ms and for the left leg of 158.8 ± 39.4 ms ($P = 0.45$), nor for group L with identical mean latencies for the right and the left leg of 147.8 ± 13.0 ms.

Strategy scores for the movement responses fell mostly within normal limits. Only three patients with left-sided lesions (8,9, and 10) showed more than once a significantly increased use of hip-strategy. Two of them (8 and 10) were recorded very early after the onset of symptoms (3 and 4 days, respectively). The third patient (9) was the one with an acoustic neuroma (10 days after surgery).

2. Time Course of Postural Recovery

Since only the sensory organization tests (5 and 6) revealed specific disturbances in the acute phase after a unilateral vestibular lesion, the time course of the results with only these tests will be further presented in most circumstances. Since stimulus conditions (vestibular input only) of tests 5 and 6 and results were similar, their results were combined to an average score. In Figure 2 the time course of postural recovery is shown for the average equilibrium and strategy score of tests 5 and 6 (combined).

At the first recording session, only one patient (10) had an equilibrium score in the normal range. Five patients fell (1,2,4,6, and 8). The average equilibrium score amounted to 18.8 ± 21.6 and the average strategy score to 50.1 ± 16.7.

When measured the second time (7 to 25 days after the onset of symptoms), most patients had recovered to such an extent that they scored in the normal or even high normal range for both equilibrium and strategy. The average equilibrium score amounted to 60.1 ± 12.3 and the strategy score to 75.7 ± 8.8 (more movement around the ankles). In seven patients we had the chance for a third measurement 49 to 68 days after the lesion. Only slight further improvement was noticed in most patients, with an average equilibrium score of 70.1 ± 10.4 as compared to 63.9 ± 10.5 when recorded the second time. The average strategy score for these seven patients was 83.4 ± 6.1 at the third recording session compared to 77.4 ± 7.7 at the second recording session.

Figure 3 depicts the raw data (body sway and shear force) of patient 9 (complete lesion after removal of an acoustic neuroma) for tests 1 through 6 during postural recovery.

3. Time Course of Vestibulo-Ocular Recovery

The individual results for spontaneous nystagmus, canal paresis, and directional preponderance during rotatory testing during recovery are depicted in Figure 4.

Six of the 10 patients (3,4,5,8,9, and 10) had at least initially complete unilateral vestibular lesions with no caloric excitability, even with ice-water calorics. The remaining four patients had severely reduced caloric responses on one side. In three patients (4,5, and 9), the lesion remained complete (Figure 4b). On the first recording session all patients had spontaneous nystagmus in the dark ranging from 2° to 36°/s with an average of 16.4° ± 9.8°/s, the fast phase always beating toward the in-
tact side. With ice-water calorics on the intact side, the spontaneous nystagmus could be reversed in 7 out of 10 patients, indicating the existence of vestibular tonic activity on the deafferented side. The canal paresis score amounted to an average value of $85.5\% \pm 17.0\%$, with a range from 59% to 100%. The dynamic imbalance expressed by the directional preponderance of postrotatory nystagmus amounted to an average value of

Figure 2. The time course of postural recovery is shown for the equilibrium scores (a) and the strategy scores (b) for all 10 subjects as denoted by small numbers at the last recording day. The scores were calculated as the average of sensory test 5 and 6 (stabilized or missing visual input and reduced proprioceptive input). The dashed line denotes the 5th percentile of normal behavior. The graph shows that most of the postural recovery takes place within 1 to 2 weeks and only small further improvement occurs thereafter.
Figure 3. This graph shows the raw data for body sway (upper trace) and shear force (lower trace) of patient 9 (complete lesion after removal of an acoustic neuroma) for sensory tests 1 through 6 for the 3 recording days. Again, tests 1 through 4 show only small if any anterior–posterior sway and basically absent shear movement. In test 5 and even more in test 6 at the first recording day the anterior–posterior sway is drastically increased, even though this patient did not fall. During recovery this pathological sway markedly decreased to normal amounts despite the fact that the vestibular lesion remained complete.

35.6% ± 12.6%, ranging from 15% to 52%, with the better response when the intact side was excited (that is, on sudden cessation rotation toward the lesioned side). High velocity rotations toward the lesioned side produced a mean slow phase velocity toward the intact side of 52.4° ± 20.6°/s ranging from 22° to 79°/s.

At the second measurement (7 to 25 days after the onset of symptoms) the patients had reduced spontaneous nystagmus in the dark with an average value of 5.9° ± 4.4°/s, ranging from 0° to 14°/s. Ice-water caloric on the intact side reversed the spontaneous nystagmus in 9 out of 10 patients. In three patients (4, 5, and 9) there was no caloric excitability on the lesioned side. One patient (2) showed a better caloric excitability on the lesioned side with only minimal overall caloric excitability. In the remaining 6 patients, the canal paresis score was markedly diminished to an average value of 53.2% ± 21.5%, ranging from 15% to 71% compared to 82.8% ± 19.3% (for these 6 patients) at the first recording day. The directional preponderance with rotatory testing was reduced to an average value of 17.5% ± 15.7%, ranging from 0% to 41%. High velocity rotation toward the lesioned side produced a mean slow phase velocity toward the intact side of 83.6° ± 35.1°/s ranging from 43° to 142°/s.

Eight of the 10 patients were recorded a third time (43 to 68 days after the lesion). Spontaneous nystagmus was reduced to an average value of 2.8% ± 1.5%. In 2 of these 8 patients (4 and 9), ice water caloric elicited no response on the lesioned side. In the remaining 6 patients, the canal paresis score showed either no side difference (one patient [10]) or even better excitability of the lesioned
Figure 4. The time course of spontaneous nystagmus (a) and canal paresis with caloric testing (b) and direction preponderance with rotatory testing (sudden cessation of continuous rotation with $180^\circ$/s) (c) is shown. While all patients had reduced spontaneous nystagmus in a similar way as they regained postural stability, several patients showed a much slower recovery of their dynamic imbalance with rotatory testing. Six patients (3 through 5 and 8 through 10) had initially a complete unilateral vestibular lesion. Except 3 patients (4, 5, and 9) in whom the lesion remained complete, all the other patients reduced the canal paresis rapidly when the affected labyrinth regained function.
posture was further diminished and even reversed in 3 of the 8 patients.

Discussion

The vestibular signals are one of the three partly redundant sensory inputs that control upright stance and gait, the other two being proprioceptive and visual inflow (3,4,5,6). The role of labyrinthine afferents has been studied during stance (7,8) and gait (9). Nashner and coworkers (1,2) have hypothesized that if visual or somatosensory inputs conflict with the vestibular estimate of the direction of gravity, inappropriate postural reflexes are suppressed and inaccurate sensory inputs ignored. These authors suggested that, if the vestibular input is disturbed, the postural control system must appropriately reweight each sensory input, enhancing the influence of those senses providing accurate information and suppressing conflicting or inaccurate inputs. In this report, we have shown that even in the acute phase after a unilateral vestibular lesion with marked static and dynamic vestibulo-ocular asymmetries, most patients can maintain upright stance as long as sufficiently accurate orientation information is provided by proprioception and/or vision. Shortly after the lesion (as early as 3 to 4 days after the onset of symptoms) each of the remaining accurate sensory systems (visual or somatosensory) alone can provide postural stability despite a disturbed vestibular signal. This indicates that the proposed reweighting of sensory inputs occurs very early after the vestibular lesion, probably mainly by ignoring the inappropriate vestibular signal. Consequently, loss of balance occurs as soon as both accurately functioning sensory systems are disrupted. About 1 to 2 weeks after the vestibular lesion, however, vestibular inputs are certainly reused again by the central postural control system, since upright stance was basically unaffected in most patients, even in the case in which visual and somatosensory inputs were almost entirely disrupted or inappropriate (sensory tests 5 and 6). Thus, one can assume that the vestibulo-spinal imbalance must have been largely reduced 1 to 2 weeks after the vestibular lesion, even in the subjects in whom the lesion remained complete. This leads to the conclusion that after a vestibular lesion the proposed reweighting of the three sensory inputs may be only the first line of defense, but is not the main mechanism for the recovery of postural control. Postural control requires the rebalancing of the vestibulo-spinal system and hence the reutilization of centrally adjusted vestibular signals themselves in cases where labyrinthine function does not recover. Thus, provided that the static vestibulo-spinal asymmetry has been rebalanced, one functioning labyrinth can provide enough vestibular information to serve as vestibular input sufficient to keep balance under most circumstances.

Earlier findings suggested a rather independent time course of the recovery of the vestibulo-ocular reflex and vestibulo-spinal reflexes after a vestibular lesion (10,11). We found, however, that the static vestibulo-ocular asymmetry (as revealed by spontaneous nystagmus) was reduced in a manner similar to the reduction in the static vestibulo-spinal asymmetry, with most patients having a spontaneous nystagmus of less than 10°/s 1 to 2 weeks after the lesion. That indicates a similar fast time course for static vestibulo-ocular and vestibulo-spinal rebalancing mechanisms, with most of the recovery being completed within 1 to 2 weeks. In contrast, the recovery of the dynamic vestibular asymmetry was much slower in most of the patients, as has also been found in hemilabyrinthectomized monkeys (12). This may indicate that both static vestibulo-ocular and vestibulo-spinal symmetry are accomplished by the same neuronal process, namely, the restoration of tonic vestibular activity on the lesioned side (13,14). That tonic activity on the lesioned side was indeed restored was demonstrated in our patients by high velocity rotations toward the lesioned side and by ice-water caloricson the intact side (both of which silence the vestibular afferents from the intact labyrinth).
Both of these stimuli lead to slow phases away from the side of the lesion, which could only have been driven by vestibular activity on the lesioned side. One can speculate that deficits during locomotion with fast changes in movement direction might last longer, similar to the dynamic vestibular asymmetry. Unfortunately, the above comparison is hampered by the fact that we could test only the function of the horizontal semicircular canals. We were not able to adequately test for either the otolithic function or for the function of the vertical semicircular canals, which contribute more to the vestibular input for the postural stabilization, especially in the anterior–posterior direction, than does the horizontal canal system. It seems conceivable, however, that the basic mechanisms for restoration of central vestibular symmetry apply in a similar way for all labyrinthine subsystems.

Obviously, the sensory organization tests detect a unilateral vestibular lesion only during the acute phase, and do not show a pathological pattern about 2 weeks after the onset of symptoms. This is observed even in cases with persistent complete unilateral vestibular lesions, since most patients had test results falling into the normal or even high normal range, even in the conditions when they had to rely primarily upon the vestibular input. In our patients, we could not confirm earlier findings suggesting that abnormal vestibular function, even in well compensated states, disrupts the subject’s reference to gravity (earth) vertical. Neither could we confirm that in patients with vestibular lesions this loss of a spatial reference persists, the resolution of conflicting or inaccurate visual and somatosensory spatial references, resulting in postural instability roughly proportional to the degree of the vestibular deficit (1, 2, 15, 16).

Unlike the sensory test, the movement coordination test did not show any significant alteration, even during the acute phase of a vestibular lesion, which would distinguish patients with unilateral vestibular lesions from normal subjects. According to our data, these responses seem to be relatively independent of a disturbed vestibular input. There is some controversy in the literature about this notion. While a vestibulo-spinal modulation of motor activity (H-reflex) dependent on head position and comparable effects of visual–vestibular excitation in response to gazing at a large moving surround (17, 18) as well as transient EMG responses in gastrocnemius–soleus muscle after galvanic stimulation of one labyrinth (19) illustrate the functionality of this pathway at low frequencies, fast disturbances rather release a fixed proprioceptively elicited reflex-like pattern of motor responses that is largely independent of static vestibular and visual input and does not depend on skin and ankle joint receptors (6).

Nevertheless, even 2 months after the onset of a unilateral vestibular lesion, Allum and co-workers (20, 21) found a 10% reduction (amplitude) in EMG responses (though with normal latencies) of the ipsilateral tibialis anterior muscle; these responses were not exactly distinguishable from those observed during an examination at deficit onset.

Conclusions

Our study confirmed earlier findings in animals (10) that static vestibulo-ocular and vestibulo-spinal balance recover very quickly after a unilateral vestibular lesion, showing basically normal test results for postural testing within about 2 weeks after the lesion, and little improvement thereafter.

While the sensory organization tests revealed the vestibular disturbance during the acute phase after the lesion (up to about 1 to 2 weeks), further testing did not disclose any pathological pattern in our patients, despite a persistent unilateral lesion in some cases.

Postural responses to unexpected displacements of the support surface are automatic and stereotyped and not influenced by vestibular imbalances even in the acute phase of the lesion.

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