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Recovery times of stance and gait balance control after an acute unilateral peripheral vestibular deficit

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Abstract.
BACKGROUND: Acute unilateral peripheral vestibular deficit (aUPVD) patients have balance deficits that can improve after several weeks. Determining differences in vestibulo-spinal reflex (VSR) influences on balance control and vestibular ocular reflex (VOR) responses with peripheral recovery and central compensation would provide insights into CNS plasticity mechanisms. Also, clinically, knowing when balance control is approximately normal again should contribute to decisions about working ability after aUPVD. Usually VORs are employed for this purpose, despite a lack of knowledge about correlations with balance control. Given this background, we examined whether balance and VOR measures improve similarly and are correlated. Further whether balance improvements are different for stance and gait.
METHODS: 26 patients were examined at onset of aUPVD, and 3, 6 and 13 weeks later. To measure balance control and thereby assess the contribution of VSR influences during stance and gait, body-worn gyroscopes mounted at lumbar 1–3 recorded the angular velocity of the lower trunk in the roll (lateral) and pitch (anterior-posterior) directions. These signals were integrated to yield angle deviations. To measure VOR function, rotating chair (ROT) tests were performed with triangular velocity profiles with accelerations of $20^\circ/s^2$ and $5^\circ/s^2$, and caloric tests with bithermal (44 and 30°C) water irrigation of the external auditory meatus. Changes in average balance and VOR measures at the 4 examination time points were modelled with exponential decays. Improvements were assumed to plateau when model values were to within 10% of steady state.
RESULTS: Balance improvement rates were task and direction dependent, ranging from 3–9 weeks post aUPVD, similar to the range of ROT VOR improvement rates. Stance balance control improved similarly in the pitch and roll directions. Both reached steady state at 7.5 weeks. However, changes in visual and proprioceptive influences on stance sway velocities continued to decrease in favour of vestibular influences for over 10 weeks with the visual influence being correlated with ROT deficit side responses ($R = 0.475$). Spontaneous nystagmus and stance roll velocity were weakly correlated ($R = 0.24$). Pitch control during gait tests improved faster than roll. Gait speed was slower and only recovered normal velocity at 6–9 weeks. Pitch velocity when walking eyes closed was correlated ($R = 0.38$) with ROT asymmetry. Other balance and VOR measures were more weakly correlated ($R < 0.2$) even if these had similar improvement rates.
CONCLUSIONS: These results indicate that balance control for stance improves equally fast in the pitch and roll directions. For gait, pitch control improves faster than roll. On average, stance and gait tests show normal balance control at 6–9 weeks post aUPVD onset. As few balance measures are correlated with those of VOR function and then with low ($R < 0.5$) coefficients, we suggest that VOR tests should not be used to assess improvements in balance control after aUPVD. The lack of strong correlations between balance and VOR measures included in this study during peripheral recovery and central compensation of aUPVD supports the hypothesis that recovery of balance function after an aUPVD involves different CNS pathways and neural plasticity mechanisms.

Keywords: Unilateral vestibular loss, balance control, recovery rates, stance, gait

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1. Introduction

If vestibulo-ocular reflexes (VOR) and vestibulospinal reflexes (VSR) contributing to balance control improve at the same rate following an acute unilateral peripheral vestibular deficit (aUPVD) and are strongly correlated with one another, then determination of the time-point of normal gaze and postural stability after the acute onset would be relatively straightforward because either could be used to determine if recovery had occurred. If, however, there are differences in VOR improvement rates depending on the amplitude of head acceleration used, and for VSR contributions to balance depending on whether stance or gait tests (that is slow versus fast body movements) are used to test the effect of VSR deficits on postural stability, then determining the time point of recovery would have to depend on a representative test battery of the different balance improvement rates and not VOR measures. A difference in balance improvement could also occur dependent on the direction of motion, that is, pitch versus roll, just as differences in VOR improvement with the direction of rotation, pitch versus yaw, have also been observed [9].

The immediate disabling symptoms of an aUPVD, which is often due to vestibular neuritis, disappear within a few weeks. Nausea and spontaneous horizontal nystagmus are generally absent at 3 weeks from onset despite a remaining VOR response asymmetry. At 3 weeks, the eye velocity responses for head accelerations to the deficit side are, on average, significantly lower than those of healthy subjects, and lower than those of responses for head accelerations to the non-deficit side [8]. Thus within VOR measures, spontaneous nystagmus and responses to rotation, different improvement rates emerge. The VOR response improvement rates may also depend on whether high or low accelerations are applied to the head with video head impulse tests (HIT) or clinical rotating chairs (ROT), respectively, to elicit a VOR. For HIT, recovery of ipsilesional gain to normal appears to take over twice as long [34,45] as the 12 weeks required with low ROT accelerations [8,13], although the VOR recovery for these tests has not been investigated in the same subjects. This difference might be explained by the inherent response non-linearities observed both in VOR and vestibular nuclei neural sensitivities [33,43], plus the reduced number of ipsilesional type I vestibular nucleus neurons post-deficit [32]. Both neural dysfunctions could lead to slower asymmetry improvement rates for high stimulus accelerations imposed with HIT. Considering these movement speed differences would seem important when judging the effects of oscillopia on stance and gait, and direct effects of an aUPVD on balance control during stance and gait. Improvements for slow accelerations might be appropriate for judging the ability of a PVD patient to stand in a stable position, whereas faster accelerations might be more appropriate in judging the ability to look from side-to-side or up and down and fixate gaze on a stationary or moving object while walking [3].

Regardless of the appropriate accelerations to use to measure VOR improvement, the question arises whether recovery of normal balance control during stance and gait is related to that of the VOR. That is, whether there are significant correlations between stance and gait measures and VOR measures, and further if the improvement time courses are similar. Pilot studies have indicated that VOR and balance measures based on trunk sway during stance and gait tests are not correlated with one another [6]. Furthermore the improvement rates for gait tasks (ca. 12 weeks) and stance tasks (between 3 and 12 weeks post-acute onset) appear to be different [4].

There are a number of neural mechanisms which would suggest differences in improvement rates are likely to be present for the VOR and VSR contributions to balance. Firstly, stimulation at the receptor level, produces VOR and VSR responses which appear to be very different. For example stimulation at the lateral canal ampulla produces horizontal eye movements, but anterior-posterior and medial-lateral body sway, rather than yaw sway [36]. Secondly, neuronal thresholds to head movements in vestibular nucleus neurons projecting centrally are elevated following vestibular loss and remain so years later [26]. However, vestibular neurons which post-loss received increased modulation by non-vestibular inputs, for example, neck [39] inputs, recover normal thresholds quicker [26]. The latter neurons are thought to be involved in the regulation of stance and gait [14]. In contrast VOR interneurons in the vestibular nuclei have a different reweighting of proprioceptive and vestibular inputs post vestibular lesion [38], suggesting that VSR improvement may be faster than that of the VOR. The use of non-vestibular inputs to improve balance control, particularly visual inputs, is well known. Such inputs help reduce balance deficits following UPVD [12]. Thus a third reason for differences between VOR and VSR improvement might lie in the ability of VSR neurons in the vestibular nuclei to use different non-vestibular signals to improve balance control as part of the compensation process.

Based on these differences in anticipated improvement rates it would seem important from both a neurophysiological as well as a clinical viewpoint to determine the differences in balance and VOR improvement rates. Clinically documented, the different rates would provide one basis, among others, for determining the time point when a patient has normal working ability and has achieved as much improvement as can be expected. Even for VOR responses, improvement cannot be predicted from VOR responses in the acute state of an aUPVD [11]. For these reasons, we examined improvement in VOR reflexes documented in a number of tests (caloric, and rotating chair tests) and compared these improvement rates with improvements in balance control, as observed in stance and gait posturography, following an aUPVD. Our assumption was that VSR and VOR improvement rates depend on separate neural pathways. Therefore improvement could be different because VOR and balance measures would not be correlated with one another. Further we expected differences in improvement to depend on the direction of sway (pitch or roll).

2. Methods

We retrospectively examined data of 26 patients (11 female, 9 male, mean age 51 years, range 23–73) referred to our emergency department with typical symptoms of acute unilateral vestibular neuritis with spontaneous nystagmus beating to the healthy side, nausea, and a falling tendency to the deficit side. The diagnosis of an aUPVD was suspected by a screening video head impulse test (HIT) with a pathologically low gain of 0.6, or less, and catch-up saccades, and confirmed with a caloric test (see below), which showed a canal paresis (CP) greater than 30% (mean 86.4%, sem 3.5%). All patients were treated intravenously with methylprednisolone (125 mg Solumedrol^{TM} per day) and then discharged 4 days later with oral medication. On discharge, patients were offered 10 sessions of balance oriented physical therapy.

During the in-patient stay patients were examined with a battery of standard video-oculography (VOG) tests including recording of spontaneous nystagmus, bi-thermal irrigation caloric test to determine a canal paresis (CP) value of peripheral vestibular deficit, optokinetic nystagmus, saccade, eye-tracking and rotating chair tests (ROT). Details of this test battery are described in Allum et al. 1996 [5] and the ROT analysis in Allum and Ledin 1999 [8]. Two whole body rotating chair velocity profiles were used. The first consisted of a 20°/s^2 acceleration for 6 s to 120°/s, then a 20°/s^2 acceleration in the opposite direction for 12 s, followed by a deceleration of 20°/s for 6 s to stop. The second profile consisted of 5°/s^2 acceleration for 40 secs to 200°/s followed by a deceleration of 40°/s for 5 secs to stop. Rotations were performed in the dark. Both velocity profiles were presented first in the clockwise (right) direction, then counter-clockwise. For the first profile, the peak amplitudes of the slow phase eye velocity (SPV) averaged over 2 s were measured with respect to the level of spontaneous nystagmus. This level was based on average SPV occurring over the 20 s before the start of each of the 4 irrigations of the caloric examination. To obtain a ROT measure for each direction, the peak amplitude of SPV at 6 s for one initial direction of chair rotation was averaged with that at 18 s for the opposite initial direction of rotation (that is, both averaged peak SPVs were in the same direction) after compensation for the different durations of acceleration [8]. For the second, 5°/s^2 profile, the average of the SPV for the last 20 s of the acceleration phase was used, again corrected by the level of spontaneous nystagmus. From the amplitudes, response symmetry was measured as (R-L)/(R+L) expressed as a percentage, where R means right SPV, L left.

Magnetic resonance imaging (MRI) was performed on any patients showing any central signs in the VOG examinations. This step was taken for 4 patients, all of whom had normal MRIs. We excluded any patients with pre-existing orthopaedic problems affecting balance, polyneuropathy, and co-existing central deficits, for example, multiple sclerosis.

Balance control was measured with a body mounted gyroscope system (SwayStar^{TM}, Balance International Innovations, Switzerland). This system quantified the body's angular displacement and velocity near its centre of mass (CoM), during a battery of 14 stance and gait tests [4]. The gyroscopes were mounted at lumbar segments 1–3 using a tight fitting elasticated belt. With this system we have documented improvements or lack thereof in balance control following an acute or chronic UPVD [4,10]. Peak-to-peak displacement and velocities in the pitch (anterior-posterior) and roll (lateral) directions were used as measures of balance control. From the standard test battery of 14 tasks we selected for analysis those tasks best indicating a UPVD [4]. These were one stance test (standing on two legs, feet shoulder width apart, eyes closed on foam), one semi-stance test (walking 8 tandem steps), and two gait tasks (walking 3 m while pitching the head up and

down, walking 3 m with eyes closed). A foam support surface reduces the efficacy of ankle proprioceptive inputs and reveals the ability of the vestibular inputs to control body sway because, with eyes closed, visual inputs are not available [24]. All four stance and gait tests have been used to optimally discriminate patients with vestibular loss from other patient groups with balance deficits and healthy controls [4,42]. However, we used, in addition, the differences in pitch sway velocity between eyes closed and eyes open stance tests summed for a foam and firm surface divided by the sum for all two-legged stance tests in the test battery (eyes open and closed on a normal and foam surface) to determine the visual contribution to balance control during stance and the difference between foam and normal surface results summed for eyes closed and for eyes open divided by the sum for all stance tests for the proprioceptive contribution.

In previous studies, we tested the balance control of patients who are suffering from UPVD at three time points: at onset of the deficit and then, on average, 3 weeks and 12 weeks later [4]. In our clinic, an additional test at 6 weeks is used, particularly if the patient would normally stand while working, or an instability could endanger others (for example, a bus driver). The 3 weeks interval was selected based on reports that at this time point aUPVD patients recover normal control of stance [16,40], neurochemical changes associated with compensation are complete [27,41], and spontaneous nystagmus has subsided [37]. The test point at 12 weeks coincides with the time point when VOR yaw responses to low acceleration (below $100^{\circ}/s^2$) whole body rotations have achieved normal symmetry and gains in the majority of patients [8,9].

At onset of the aUPVD and at the targeted 12 weeks the complete VOG test battery was recorded. However, at the targeted 3 and 6 weeks only the caloric and ROT tests were performed. HIT and balance tests were performed at all test times. Because HIT results provided no additional information to the current results, HIT results are not reported here. The actual mean test times were 3.0, 6.2 and 13.1 weeks, standard error of mean (sem) 0.1, 0.2, and 0.3 weeks, respectively.

Calculations of means and linear regressions between measures were performed with Excel 2010. All other calculations, including statistical tests were performed with MATLAB R2012b (Mathworks, Natick, MA USA). For the comparisons of the means between the acute values and the values after 3, 6.3 and 13.1 weeks we used a one sided t-test because we expected a priori a reduction in the values. We also tested the medians with a one sided Wilcoxon signed rank test. Significance is stated in the results when both statistical tests revealed significant differences ($p < 0.05$). The upper bounds for the p values stated in the results are thus valid for both tests.

The mean recovery time (as well as of the mean plus and minus the sem of measures was modelled by the following equation, $y = p_1 + p_2 \cdot e^{-p_3 t}$, where y is the measured mean at time, t, in weeks, p_1 the steady state mean value of the measure, p_2 the difference between onset and steady state means, and $1/p_3$ the exponential decay time constant of the mean between onset and steady state. The parameters of the exponential model function were estimated using MATLAB's *nlinfit* (nonlinear least-squares regression) function. The termination tolerance was set as 1e-8 for both the residual sum of squares and the estimated coefficients. For the data presented here less than 100 iterations were needed to terminate within the set tolerance.

The term

$$t_{error} = abs\left(\frac{\left(\frac{1}{p_{3,mean+sem}} + \frac{1}{p_{3,mean-sem}}\right)\cdot\frac{1}{2} - \frac{1}{p_{3,mean}}}{\frac{1}{p_{3,\,mean}}}\right) \times 100\%$$

was used to estimate the significance between model fits with a value of 10% considered borderline significance. We developed this error term representing the difference between one set of model fits to mean and mean $\pm$ sem in order to ensure only similar improvement times for one set of model fits were accepted. There is no clear-cut consensus of the method to use to compare non-linear regression parameters [30].

This retrospective study of routine clinical examinations, with permission of the patients to use their data anonymously, was approved by the local ethical committee (EKNZ Switzerland).

3. Results

We observed an average peripheral loss at aUPVD onset of 86.4% (standard error of mean (sem) 3.5%) as determined by the caloric canal paresis (CP) value. Consistent with our previous studies [1,8] peripheral vestibular VOR recovery as indicate by the CP value recovered to a value of 69.7 (sem 6.8), 62.3 (sem 7.6), 51.4 (sem 8.8) at 3, 6.2 and13 weeks, respectively. The 24.1 and 40.5% CP recovery at 6.2 and 13 weeks with respect to the acute onset value of 86.4% was signifi-

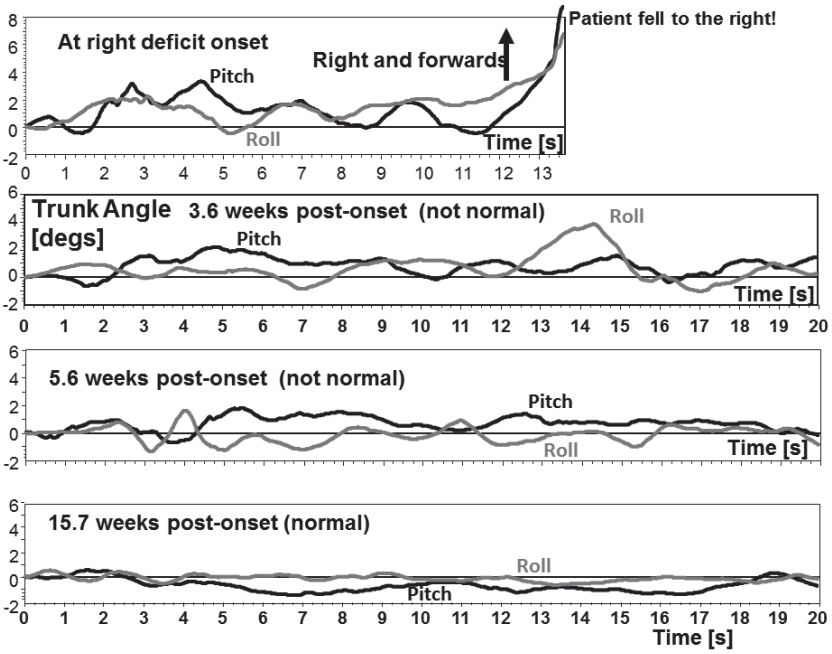

Fig. 1. Typical example of the changes in trunk pitch and roll motion for an aUVL patient while standing eyes closed on foam (patient with 100% canal paresis (CP) value at onset and only 14% CP peripheral recovery at 15.7 weeks). The upper recordings were taken at deficit onset when trunk motion was greater than normal for healthy persons of the same age as the patient (63 years). At 3.6 weeks (second set of traces) pitch and at 5.6 weeks (third set of traces) post-aUVL onset trunk motion was still abnormal. At 15.7 weeks (lower set of traces) trunk sway was normal.

cant ($p < 0.02$) with respect to onset CP and may be contrasted with improvements in stance and gait balance control described below.

Pathological stance and/or gait test results (based on exceeding the 95% normal limit of SwayStar normal reference values [21] were observed at onset of the aUPVD for all patients with 2 exceptions. These exceptions initially had only pathological VOR results at onset and developed pathological stance and gait test results at 3 weeks. Some 65% (17/26) of the patients had pathological gait at onset compared to 69% (18/26) with stance deficits.

Figure 1 shows a typical example of improvement for the most difficult stance test – standing on two legs eyes closed on a foam surface. 42% (11/26) of the patients lost balance control and had to be assisted at aUPVD onset during this test. At 3 weeks, 3 patients lost balance during the same test, and none at 13 weeks. On average, trunk sway for this stance test was normal at 6.2 weeks (Fig. 2). The rates of improvement rate for roll and pitch were identical (7.6 weeks to within 10% of the steady state level, see Fig. 2 and Table 1). The changes in roll and pitch sway velocities over the 13 week observation period were 12.7°/s and 26.2°/s, respectively. At 6.2 and 13.1 weeks roll and pitch velocities were significantly different from velocities at onset ($p < 0.02$). The improvement was 68.6% and 75%, respectively, in comparison to onset values and therefore greater than CP recovery. Similar improvement percentages were observed for exponential decay models of improvement (Table 1).

The visual and proprioceptive contributions to stance sway control decreased over time from elevated values at onset. These composite measures from all four stance tests took longer to reach a value within 10% of steady state than stance balance control measures for the eyes closed foam task, 9.9 and 12.1 weeks respectively, (see Fig. 3 and Table 1). The largest and most significant changes occurred with the visual contribution for which the change between onset and 13 weeks was, on average, from 47% to 17% ($p < 0.0001$). At 3 weeks the visual contribution was less than at onset ($p < 0.02$). The change from onset to 13 weeks in the proprioceptive contribution was from 46% to 37% on average ($p = 0.04$) and only significant different from onset at this time point. These differences imply that vestibular contributions increased from 7% at onset to 46% at 13 weeks (assuming that the sum of all contributions is 100%).

Improvement times for VOR and balance measures that could be modelled with a simple exponential function are listed in Table 1. Examining the VOR mea-

Table 1
Improvement times of VOR and balance measures fitted with the exponential decay model

	Mean						
Variable	p_1	p_2	p_3	t(5%)	t(10%)	t error%	% change
SPN	1.30	6.98	0.87	3.41	2.62	1.24	84.3
5 Asymm	19.72	27.74	0.42	7.22	5.55	0.03	58.5
5 Def	16.27	−8.15	0.29	10.33	7.94	4.18	100.4
20 Asymm	11.57	30.85	0.37	8.19	6.29	0.06	72.7
20 Def	34.91	−16.23	0.26	11.58	8.90	0.28	86.9
Caloric	44.55	43.34	0.17	18.17	13.96	1.48	49.3
s2ecf.pv	8.26	26.54	0.30	9.94	7.64	2.60	76.3
s2ecf.rv	5.78	12.67	0.30	9.88	7.59	4.46	60.5
Visual	14.89	32.20	0.23	12.93	9.94	1.07	68.4
Proprioceptive	34.90	11.65	0.19	15.76	12.11	4.41	25.0
w8tan.ra	9.94	4.64	0.23	13.10	10.07	0.07	31.8
w8tan.pv	53.49	15.52	0.77	3.88	2.98	2.02	22.5
w3mec.ra	4.42	3.14	0.08	36.81	28.29	0.30	41.5
w3mec.dur	4.06	2.23	0.34	8.71	6.70	4.82	35.4
w3mhp. pv	61.44	15.52	0.54	5.52	4.25	0.24	20.2
w3mhp. dur	4.43	2.12	0.48	6.29	4.83	0.49	32.4

$y = p_1 + p_2 \cdot e^{-p_3 t}$ where t is in weeks. The parameters p_1, p_2, p_3 are listed in the table as well as the time for the variable to decay to within 5% and 10% of its steady state value. The variables are listed separately for VOR values (taken from Allum and Honegger, 2014), stance and gait values. *t error* lists a significance value for the model (see methods). The mean percentage change between the steady state value p_1 with respect to the acute value $p_1 + p_2$ is provided in the last column. *SPN* is an abbreviation for spontaneous nystagmus slow phase velocity, 5 and 20 refer to the rotating (ROT) chair acceleration, Asymm means the ratio of deficit and healthy side responses. Def means response amplitude for the respective ROT acceleration. $s2$ means standing on two legs, *ec* eyes closed, f foam. $w3m$ means walking 3 m, *hp* head pitching. *w8tan* means walking 8 tandem steps. *pv* means pitch velocity, *rv* roll velocity, *ra* roll angle, *dur* duration. The terms for proprioceptive and visual contributions to stance are defined in the methods section.

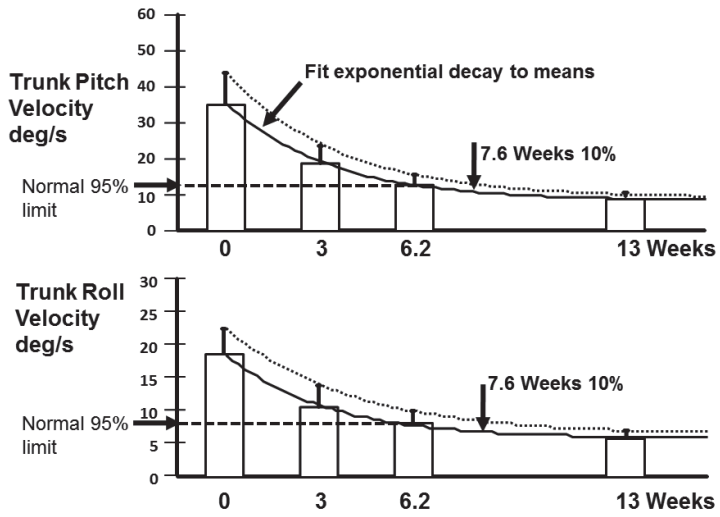

Fig. 2. Recovery time courses of trunk pitch and roll measures during stance. Pitch and roll velocity means and sem for the task of standing on two legs, eyes closed, on foam are shown. The column heights indicate mean value of the measure at aUVL onset (0) and 3, 6.2 and 13 weeks after onset. The vertical bars on the columns represent the standard error of the mean (sem). The thick full line joining the means is an exponential fit (see methods) to the change in the mean value over time. The dashed line above the full line is an exponential fit (same model form) to the means plus the sem. The recovery times to 10% of steady state are listed in table 1 and marked in the figure. Note the equally fast recovery for pitch and roll trunk sway. The upper 95% limit of normal sway is marked by a dashed horizontal line.

sures in Table 1 indicates that the VOR measures for the rotating chair deficit side responses for 5 and 20°/s^2 accelerations (to within 10% of steady state level reached after 7.9 and 8.9 weeks respectively) have similar improvement rates as the stance velocity measures (7.6 weeks). As we have determined that VOR measures with similar improvement time courses are correlated [7], we examined if this was the case

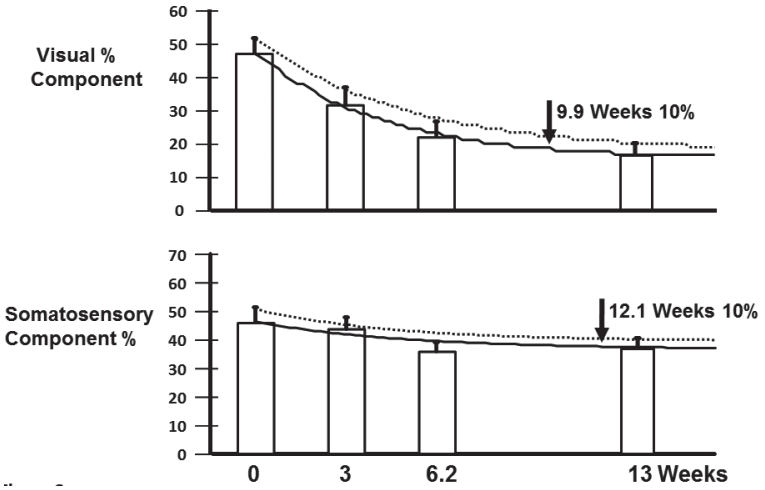

Fig. 3. Visual and proprioceptive contributions to stance based on pitch velocity during 4 different stance tests: standing on two legs eyes open and closed, on a normal and on a foam support surface. The layout of the figure is identical to that of Fig. 2.

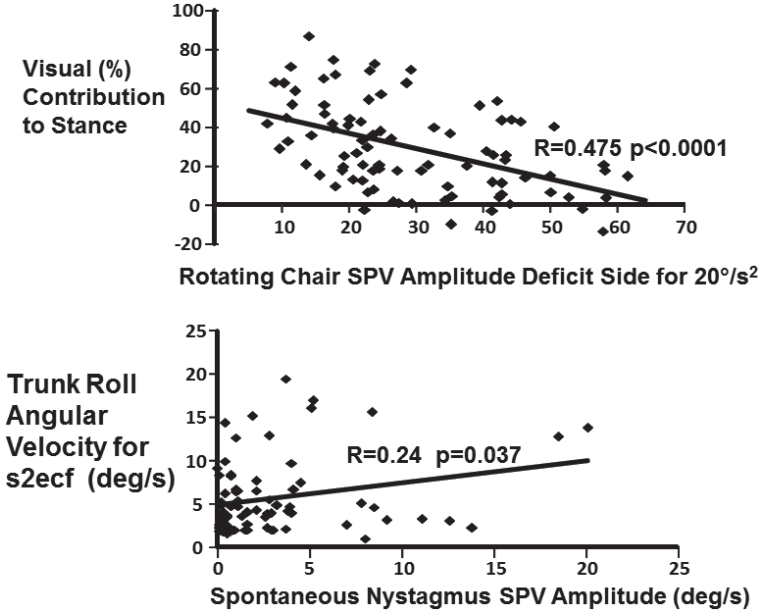

Fig. 4. A. Regression between the visual contribution to stance calculated as described in the methods section and the rotating chair response to the deficit side for a 20°/s^2 rotation. Each patient is represented in the figure up to 4 times – at onset, 3, 6.2 and 13 weeks. B. Regressions between trunk peak-to-peak roll velocity during the task of standing on 2 legs with eyes closed on foam and the slow phase eye velocity of spontaneous nystagmus. Data from onset, 3, 6.2 and 13 weeks. Only trials for which no near fall occurred were used. Note this regression is weak (R = 0.24) but significant ($p = 0.037$).

for stance and VOR measures with similar improvement times. Despite the overlap in improvement times for stance velocity and rotating chair deficit side VOR measures, there were no significant correlations ($p > 0.05$) between these stance and VOR measures. There was, however, a significant correlation ($R = 0.48$ $p < 0.0001$) between the visual component to sway during stance and the VOR rotating chair deficit side response for 20°/s^2 accelerations (see Fig. 4). Interestingly these 2 measures had similar recovery times (9.9 vs. 8.9 weeks, see Table 1). The change in afferent nerve resting activity on the deficit side following an aUVL causes a spontaneous nystagmus (SPN). We assumed that the resting discharge underlying the SPN

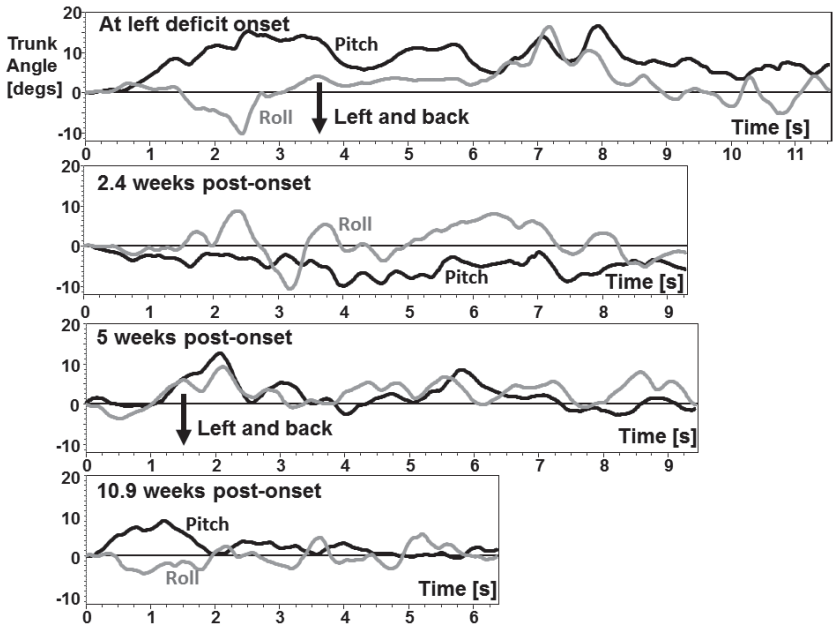

Fig. 5. Typical example of the changes in trunk pitch and roll motion over time for the task of walking 8 tandem steps recorded from an aUVL patient. The records at aUVL onset, 2.4, 5 and 10.9 weeks post onset are characterized by pitch and roll angular velocities greater than those of normal persons of the same age as the patient (61 years). As can be noted from the duration of the recordings, the task was completed quicker with recovery. The UVL recovered completely (as determined by a CP value of 10.4% at 10.9 weeks). Note the early stabilisation of pitch (at 2.4 weeks) compared to roll.

slow phase velocity (SPV) amplitude might affect the detection of low body sway velocities. Therefore, we examined correlations between this SPN SPV amplitude and stance sway velocities. A weak correlation ($R = 0.24$, $p = 0.04$) between roll velocity stance measures and SPN SPV (see Fig. 4) was found, even weaker for pitch velocity ($R = 0.18$, not significant). Other correlations that we investigated for stance measures including those with CP values yielded non-significant correlations with $R < 0.2$.

Four of the 5 patients that returned for testing at 6 months due to chronic balance problems had gait but not stance deficits. Therefore we expected recovery rates to be slower for gait than for stance. Tandem gait is a gait task dependent on stable control of body roll and pitch motion [23]. Figure 5 provides a typical example of improvements over time and Fig. 6 illustrate the times required for roll and pitch motion to reach a steady state value – at 3 weeks post aUVL onset for pitch velocity but later, 10 weeks, for roll angle. This difference occurred despite a considerable reduction in task duration at 3 weeks ($p < 0.0001$). For normal walking, increases in gait speed cause equal increases in roll and pitch measures [18]. The mean change in pitch velocity over 13 weeks was 15.8°/s (24.5% with respect to onset mean), that for roll velocity 12.6°/s (20.4%), pitch angle 5° (33%), roll angle 4.2° (29%) and duration 6.7 (37%) s. These changes were significant ($p < 0.02$) with respect to onset values, but less as a percentage than CP changes. Roll velocity amplitudes were correlated with the VOR measure of rotating chair amplitude asymmetry for 20°/s^2, however, the correlation was weak (R = 0.09) and not highly significant ($p = 0.03$). No other more significant correlations were found between tandem gait and VOR measures.

Another test commonly used to test vestibulo-spinal control during gait is walking 3 m with eyes closed. For this test, the duration required to walk 3 m is most affected by vestibular loss [4]. As may be observed in the example of Fig. 7 and the mean plots of Fig. 8, pitch velocity and roll angle are changed but less than duration. The mean changes between onset and 13 weeks were 18°/s (23.4% with respect to onset) for pitch velocity, 3.6°/s (6.3%) for roll velocity, 1.3° (15%) for pitch angle, 2.2° (29%) for roll angle, and 2.3 (35%) s for duration. The changes for pitch velocity, roll angle and duration were significant ($p < 0.01$) with duration having the highest significance. The highest correlation with VOR measures for this gait task was achieved by comparing pitch velocity during the eyes closed gait

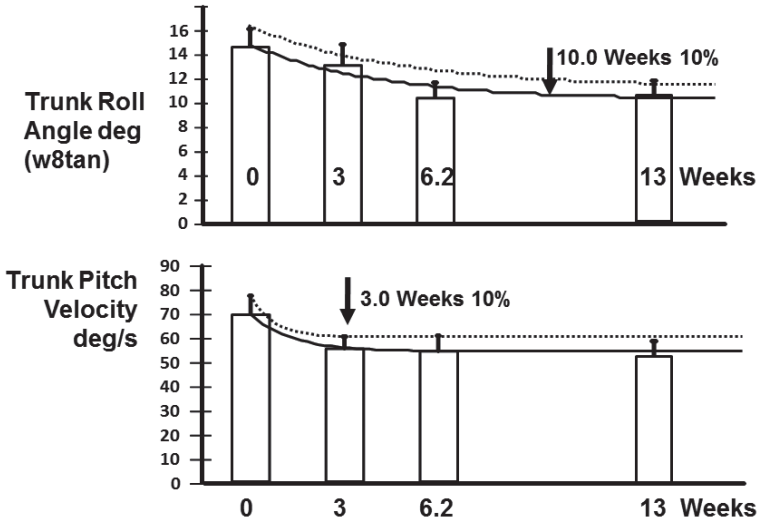

Fig. 6. Recovery time courses of trunk roll angle and pitch angular velocity for the task of walking 8 tandem steps. The layout of the figure is identical to that of Fig. 2. Note the faster recovery for pitch velocity compared to roll angle.

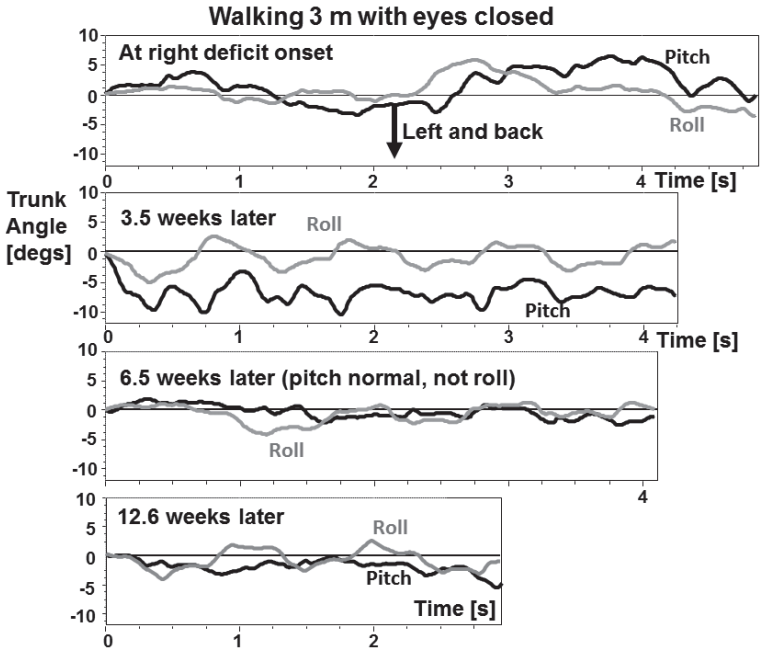

Fig. 7. Typical example of trunk pitch and roll motion during the tasks of walking 3 m eyes closed. The records are taken at right aUVl onset, 3.5, 6.5 and 12.6 weeks post onset. At 6.5 weeks post-onset the traces were normal. The aUVL patient, 48 years of age, had a 30% peripheral recovery from a complete (100%) loss.

task with the rotating chair asymmetry for 5°/s^2 accelerations ($R = 0.39$, $p < 0.001$, see Fig. 9).

A similar effect between onset of the aUPVD and 13 weeks later on trunk pitch angular velocity, roll angle and durations was noted for the task of walking 3 m while pitching the head up and down. The mean changes between onset and 13 weeks were 14.7°/s (19.1% with respect to onset) for pitch velocity, 0.2°/s (0.6%) for roll velocity, 1.4° (14%) for pitch angle, 1.8° (25%) for roll angle, and 2.4 s (36%) for duration. Again changes for pitch velocity, roll angle and duration were significant ($p < 0.01$) with duration having the highest significance. The pitch velocity recovery rates were equally fast as those for duration (Ta-

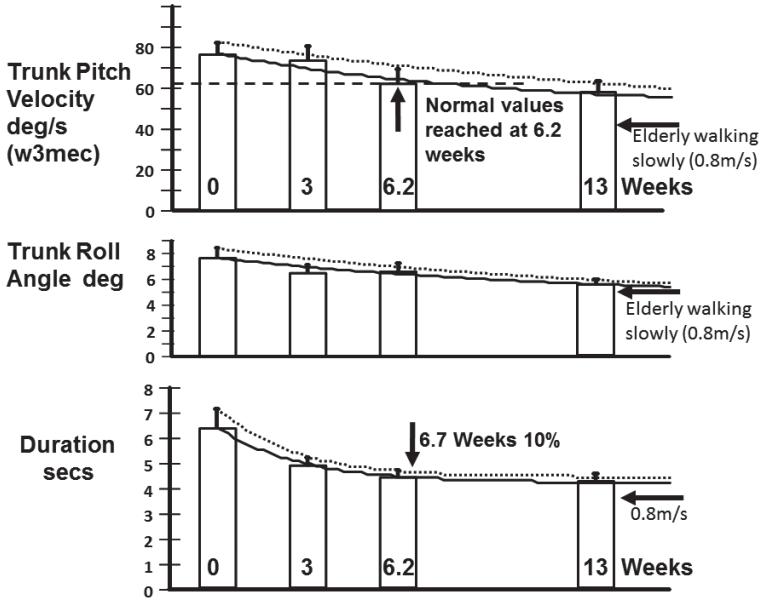

Fig. 8. Recovery rates of trunk peak-to-peak pitch angular velocity, roll angle, and task duration while walking 3 m eyes closed. The time for roll angle to recovery to within 10% of steady state is not shown as this value is at 28.3 weeks (see Table 1). The model curve for pitch velocity is not significant. The layout of the figure is identical to that of figure 2. For comparison the average pitch velocities and roll angle of elderly persons walking slowly (at 0.8 m/s) are marked (data from Goutier et al. 2010).

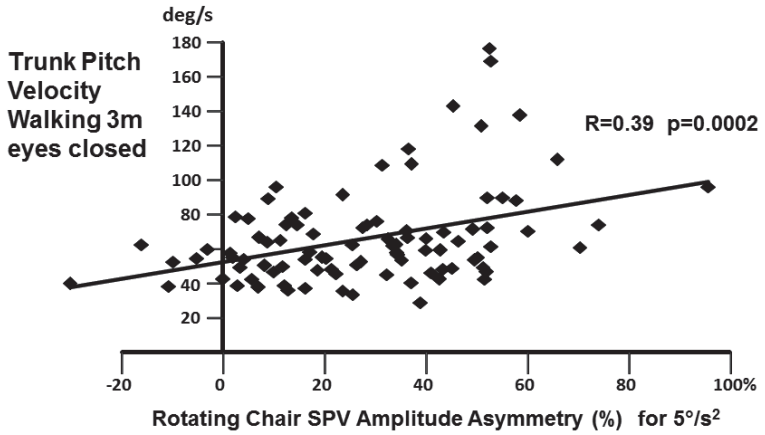

Fig. 9. Regression between trunk peak-to-peak pitch velocity during the walking 3 m eyes closed task and rotating chair asymmetry for $5°/s^2$ accelerations. Data from onset, 3, 6.2 and 13 weeks. Note the regression is weak ($R = 0.39$) but significant ($p = 0.0002$).

ble 1). Correlations of balance measures for this task with VOR measures were weak and not significant. Correlations between stance and gait measures were also not significant.

4. Discussion

This study has documented improvement times for balance control during stance and gait tasks following aUVD. Means of balance measures were normal at 13 weeks. Thus, these improvements in balance can be classified as to a recovery. One of our main findings was that recovery rates of pitch and roll velocities were equally fast for eyes closed stance tasks on foam, but that roll control had a slower recovery time course for all gait tasks. In terms of percentage changes between onset and 13 weeks, the effect on stance was greater than that on gait (see Table 1). The changes were greater than CP recovery for stance, less for gait. The

recovery of normal visual and proprioceptive contributions to stance was slower than that of stance velocities on foam alone, with visual contributions decreasing from high values at aUVD onset more rapidly than proprioceptive contributions. Presumably the shorter duration of visual dependence changes should be taken into account during physical therapy by attempting to increase proprioceptive and vestibular dependence. The other main finding was that strong correlations were not found between balance (trunk sway) measures influenced by VSRs and VOR measures commonly used to describe vestibular function.

For gait tasks either tandem gait, walking eyes closed, or walking with simultaneous head pitching movements, pitch velocity and roll angle movements were most affected but not roll velocities, possibly because roll velocities were reduced by carrying out the task more slowly, that is, slowing gait velocity. This was surprising, especially for roll velocity with tandem gait, as balance in this direction is more unstable than pitch. One method both young and elderly persons use to reduce trunk velocities and angles is to walk slower [19]. In Fig. 8, the average values of sway and gait velocity of the healthy elderly during slow (0.8 m/s) walking, eyes open, is marked for comparison. In contrast, roll and pitch sway velocity during walking is a factor 1.5 greater in the elderly prone to fall [15]. Thus reducing gait velocity by walking slowly would appear to be the strategy used by the UVL patients to control roll velocity if not pitch velocity. Thus, it appears that across gait balance tasks, pitch control recovery after an aUVD is different from that of roll.

The range of VOR improvement rates spans from 3 to 12 weeks based on the time to reach within 10% of steady state mean (see Table 1). The range of rates for balance tasks dependent on VSR contributions for stability was similar. Nonetheless, there were no or just weak correlations between most VOR and VSR measures. For example, the correlation between the SPV of the spontaneous nystagmus (SPN) present for approximately 3 weeks post onset of aUVL and trunk sway velocities during stance was weak for roll velocity although significant. We expected a correlation because presumably the change in the afferent nerve resting discharge affects both the level of SPN SPV and the detection thresholds of trunk sway [44]. There were other correlations between VOR dynamic measures (deficit side responses to $5°/s^2$ and $20°/s^2$ accelerations) with more significant, but still low, correlation coefficients. For example, the highest correlation that we could find ($R = 0.475$) was between the visual contribution to stance and deficit side responses to $20°/s^2$ accelerations. We also tested for correlations of balance measures with HIT responses and CP values without obtaining significant values. These results support our previous conclusion [6] that VOR and balance measures are generally not correlated with one another. The lack of correlation may be due to a difference in correlations between various patients' sub-groups. For example the elderly may be more susceptible to balance changes caused by an aUVD than the young. Future studies will need to address this point. As balance measures in healthy controls were highly repeatable after 3 weeks [1] we concluded that low individual repeatability is not the cause of the low correlations we observed.

Pitch values recovered much faster than roll values during gait tasks. Other authors have shown that roll deficits are much worse following UVD than pitch deficits [29,35]. Thus a longer recovery time for roll is an expected result. Furthermore, the unilateral reduction of vestibular signals would presumably lead to difficulties in discriminating roll vestibular and proprioceptive signals from one another as has been suggested for bilateral vestibular loss patients [2]. For this reason the CNS may preferentially increase visual rather than proprioceptive gains as we noted in stance tasks. The difficulties UVD patients may have in interpreting roll signals stem from the differences in processing times associated with proprioceptive versus vestibular inputs in the roll and pitch planes [2]. In the roll plane proprioceptive signals from the hip (the main axis of roll rotation during stance and gait) arrive simultaneously with roll vestibular signals at the CNS whereas in the pitch plane vestibular signals are present earlier than signals from the ankle joints (the main axis of pitch rotation). It has been proposed that the latter difference facilitates the use of alternative sensory inputs for pitch balance control [2] and presumably faster pitch plane balance recovery. It would be of interest to investigate in future studies the comparative VSR recovery rates for the yaw, pitch and roll planes.

One of the drawbacks of this study is that correlations have been limited to comparison with yaw plane VOR measures. Apart from the need described above to investigate correlations to pitch and roll plane VOR measures, an inclusion of otolith responses in the form of leg muscle vestibular evoked myogenic potentials (VEMPs) might offer additional insights. Neck muscle VEMP amplitudes appear not to be correlated with stance balance measures [25].

It was interesting to note in gait tasks little direct effects of the aUVD on roll velocities. In the elderly, walking slower than the normal speed reduces sway velocity [19] and the risk of falling. Therefore it was not surprising that UVD subjects reduced gait speed (increased duration to walk 3 m) in order to reduce excessive lateral sway. In this case, the recovery time course for trial duration to about 5–7 weeks for gait trials would be associated with the recovery of roll velocity control. The recovery of pitch velocity control during gait was relatively rapid (ca. 3 weeks), thus it is unlikely that heightened pitch velocity was used via a Coriolis effect [23] to reduce gait roll velocity. Thus our results indicate that reduced gait speed is the primary mechanism used to contain excessive, possibly fall-inducing, roll during gait. We cannot exclude the possibility that reduced gait velocity was the result of patients' being anxious about falling. However, the effect of being chronically anxious is to increase the VOR gain [17]. The effect on VSR gain for such persons is not known. If, however, healthy persons are put in a condition where they are anxious (at considerable height above the ground), then VSR gains increase [31]. It remains an open question how being chronically anxious affects VOR and VSR gain in those already with a vestibular loss.

Finally, our results help understand the controversy on whether balance tests can be used to estimate vestibular function [20,25] by pointing out that strong correlations do not exist between balance measures influenced by VSRs and VOR measures commonly used to describe vestibular function. Furthermore, balance measures involve both pitch and roll displacements with different recovery rates for gait, but not for stance. This reasoning underlines the necessity to test balance function separately from tests of gaze stability controlled by the VOR. In the case of a UVD, tests should be performed at 3–6 weeks post-acute onset in order to best capture whether recovery of balance control has occurred or not. If not, retesting should occur at 12 weeks when most balance measures should have reached a steady state (see Table 1).

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Conflict of interest

The authors declare a conflict of interest as they both worked as consultants for the company producing the SwayStar equipment used in this study.

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