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Postural control in a simulated saturation dive to 240 msw.

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Goplen FK, Aasen T, Nordahl SHG. Postural control in a simulated saturation dive to 240 msw. *Undersea Hyperb Med* 2007; 34(2):123-130. **INTRODUCTION:** There is evidence that increased ambient pressure causes an increase in postural sway. This article documents postural sway at pressures not previously studied and discusses possible mechanisms. **METHODS:** Eight subjects participated in a dry chamber dive to 240 msw (2.5 MPa) saturation pressure. Two subjects were excluded due to unilateral caloric weakness before the dive. Postural sway was measured on a force platform. The path length described by the center of pressure while standing quietly for 60 seconds was used as test variable. Tests were repeated 38 times in four conditions: with eyes open or closed, while standing on bare platform or on a foam rubber mat. **RESULTS:** Upon reaching 240 msw, one subject reported vertigo, disequilibrium and nausea, and in all subjects, mean postural sway increased 26% on bare platform with eyes open ($p < 0.05$) compared to pre-dive values. There was no significant improvement in postural sway during the bottom phase, but a trend was seen toward improvement when the subjects were standing with eyes closed on foam rubber ($p = 0.1$). Postural sway returned to pre-dive values during the decompression phase. **DISCUSSION:** Postural imbalance during deep diving has been explained previously as HPNS possibly including a specific effect on the vestibulo-ocular reflex. Although vertigo and imbalance are known to be related to compression rate, this study shows that there remains a measurable increase in postural sway throughout the bottom phase at 240 msw, which seems to be related to absolute pressure.

INTRODUCTION

Diving may occasionally elicit vestibular symptoms including vertigo, disequilibrium, nausea, and vomiting, which lead the diving physician to suspect injury to the inner ear, eighth cranial nerve, brain stem or cerebellum. Known causes are ear barotrauma, asymmetric changes in middle ear pressure, decompression sickness (DCS) or asymmetric caloric stimulation. Vertigo, dizziness, nausea and vomiting may occur during fast compression to depths greater than 150 m (1). The symptoms are then interpreted as manifestations of the high pressure nervous syndrome (HPNS), a

complex syndrome affecting different levels of the nervous system during deep diving. HPNS appears to be related both to speed of compression and to absolute pressure. Some symptoms disappear quickly when the pressure is held constant, and others disappear only during decompression, implying different pathophysiological mechanisms.

Whether the cause of these symptoms is vestibular or not, they have the potential to pose serious problems to the immersed diver. On land, vision and proprioception contribute significantly to postural balance and sense of spatial orientation. Under water, the neutral buoyancy and poor visibility will make the diver more vulnerable to vestibular

symptoms, even when these are physiological, e.g. due to asymmetrical caloric stimulation. If the symptoms are strong enough, they may cause panic, irrational behaviour, and threaten survival. Even if vertigo is not present, an effect of pressure on the postural system could decrease the performance of working divers.

It is possible to measure changes in the vestibulo-ocular reflex (VOR) during diving, but the results seem to be equivocal and difficult to interpret (2-6). A more practical approach, which may also be more directly relevant to the total performance of the diver, is to use platform posturography, which is relatively sensitive, but not specific, to acute changes in vestibular function. Using this method, Adolfson et al (7-8) found severe postural imbalance in 10 divers when breathing air in a pressure chamber at 90 msw. Braithwaite et al (3) found marked postural imbalance in 6 divers breathing helium-oxygen in a pressure chamber at 485 msw. Nordahl et al (9, 10) measured postural sway in subjects exposed to simulated dives in heliox to 5, 100 and 450 msw and in air to 20 msw. Increased postural sway was found in all dives except to 5 msw in heliox. Results from the dive to 450 msw indicated that 200 msw could be a “critical point” in the development of postural sway. Thus, we had an interest in examining postural control more closely at a dive close to this depth.

The aim of the present study was to measure postural sway at 240 msw (2.5 MPa), a depth which has not been examined in previous studies on postural control. Since HPNS is known to be related both to compression rate and absolute depth, the time course of any changes in postural sway was of particular interest. Four null-hypotheses were formulated: First, only random variations in postural sway would occur throughout the dive; second, no difference would be found between postural sway before compression and after reaching storage depth. Third, no linear trend would be

found in the variations in postural sway during the bottom phase; and fourth, no difference would be found in mean caloric response before and after the dive.

METHODS

In October 2002, a simulated dry helium-oxygen saturation dive was carried out in the hyperbaric chamber complex of Norwegian Underwater Intervention A/S (NUI) in Bergen. The research protocol was approved in advance by the Regional Committee for Medical Research Ethics, which enforces the Helsinki Declaration on medical research involving humans. Participation in the study was based on written informed consent.

Eight male subjects participated. Mean age was 41 years (range 29 - 48). Seven participants were commercial divers certified for saturation diving by the Norwegian Petroleum Directorate. One participant was a medically-qualified scientist with experience in SCUBA and saturation diving. The dive lasted 19.3 days, the compression phase 20 h 35 min (0.86 days) with stops at 10, 80 and 160 msw. The bottom phase lasted 6.6 days and decompression phase 11.9 days (Figure 1). The mean rate of saturation decompression was 20.1 msw per day. The partial pressure of oxygen (pO_2) was ~35 kPa during the compression and the bottom phase, and 70 kPa during the excursions. During decompression pO_2 was ~50 kPa, except for eight hours around each of the first seven night-stops, when it was reduced to ~35 kPa. The saturation pressure was 240 msw. Four divers were subjected to one pressure excursion to 254 msw and three excursions to 250 msw, while the other four were subjected to six excursions to 250 msw (11). Exact rates of compression/decompression during excursions were not available to the authors.

The otoneurological examinations

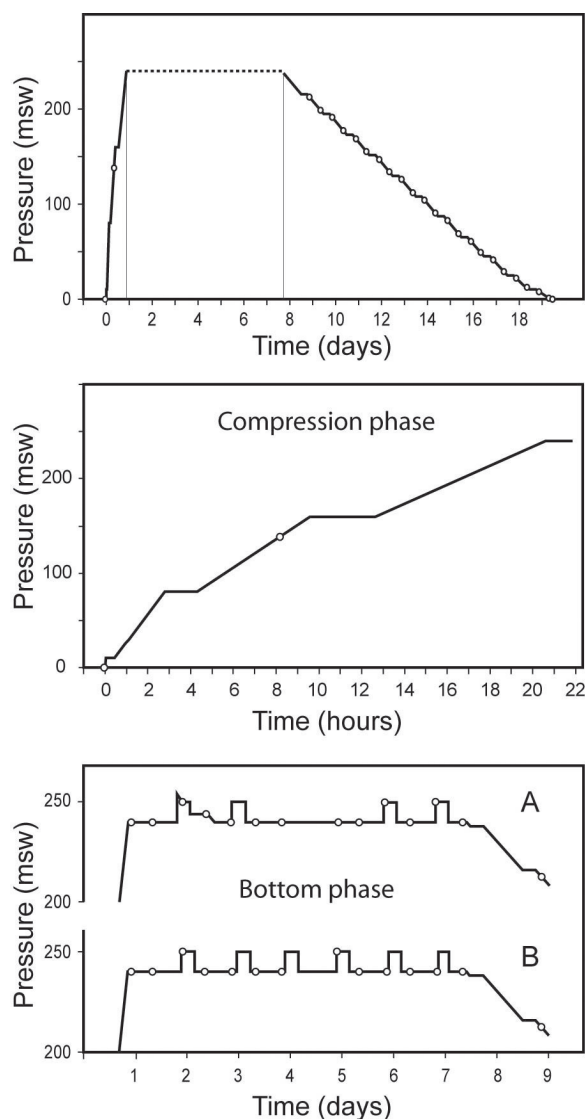


Fig 1. Pressure plotted against time after start of compression (Start: 12:25 PM). Circles identify measurements of standing steadiness. The dotted line indicates pressure changes during excursions. The lower panel shows excursion details for the bottom phase. Four divers followed profile A and the other four profile B. One from each group was excluded from analysis (see text).

before and after the dive were performed at The National Center for Vestibular Disorders, Department of Otolaryngology, Head and Neck Surgery, Haukeland University Hospital in Bergen. One otolaryngologist completed all examinations according to a standardized procedure. A medical history was taken, in addition to a clinical otoneurological

examination (including tests for spontaneous, gaze evoked, positional and post-head-shake nystagmus and Unterberger step test), static posturography (described below) and electronystagmography (ENG) to test for spontaneous and positional nystagmus, ocular smooth and saccadic pursuit, and bi-thermal caloric tests. Abnormal spontaneous nystagmus was defined as nystagmus with slow phase velocity (SPV) $\geq 5^\circ/\text{s}$ (12). Unilateral caloric weakness was defined as a difference of 25% or more in caloric response between the right and left ear according to Jongkees' formula.

In order to measure standing steadiness, we used a commercially available force platform (Cosmogamma[®], Italy) measuring 40 x 40 cm with three strain gauge pressure transducers (10). During the dive, the platform was placed in one of the smaller (11 m³) hyperbaric chambers. The platform was wired to a computer outside the chamber wall. The divers could be observed through a porthole in the chamber wall. They received instructions and practiced the tests before the chamber was closed. Two divers entered the chamber at a time, one instructing and helping the other while communicating with the outside experimental supervisor. The subjects were tested twice daily (approx 9:00 and 21:00) while in the pressure chamber, except on three occasions, when the test had to be cancelled due to other activities. Thus, each subject was tested 38 times during the whole dive. The first and last of these tests were made immediately before and after the dive, in the pressure chamber, with the doors open (i.e. breathing air at surface pressure). Only one measurement was obtained during compression (at 138 msw). The first measurement after reaching storage depth was obtained the morning after the dive started.

Postural sway was measured in four different test conditions according to the "sensory interaction" model. The rationale

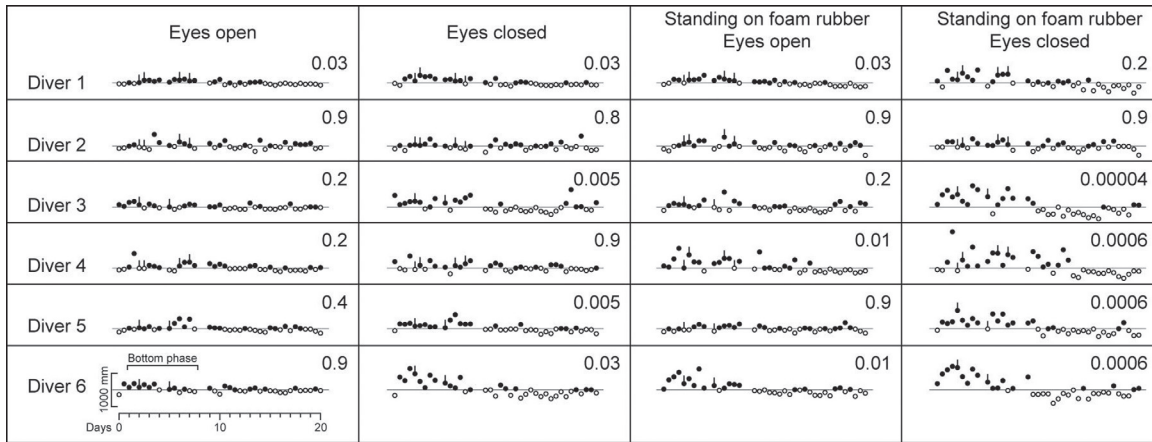


Fig. 2. Postural sway in six divers measured 38 times on a force platform in four different test conditions during a 19-day simulated saturation dive in helium-oxygen to 240 msw. Path length, i.e. the movement of the center of pressure (in mm) during one minute of quiet standing, is plotted against time (days after start of compression). Path length values were dichotomized using the median (indicated with a thin horizontal line) as cut-point. Dots and circles indicate sway above and below median respectively. P-values are shown for the null-hypothesis that postural sway varied randomly throughout the dive (runs test). A small vertical line indicates that the measurement was performed during an excursion. Note that in cells with significant results dots occur more frequently during the bottom phase, and circles more frequently during decompression.

behind this method has been described elsewhere (13). In each of the 38 tests, the balance was measured with the subject standing: 1. With eyes open (EO), 2. With eyes closed (EC), 3. On foam rubber with eyes open (EOF), and 4. On foam rubber with eyes closed (ECF). In condition 3 and 4, proprioception from the feet was disturbed by having the subject stand on a 10 cm thick foam rubber mat on top of the platform.

The center of pressure (COP) under the soles of the feet was sampled by the platform at a rate of 10 Hz. The movements of the COP reflected the corrective forces exerted on the platform by the subject in order to maintain steady posture. The length of the curve described by the COP while standing quietly for 60 seconds was used as test variable for statistical evaluation (path length).

Statistical procedures were performed in order to test the four null-hypotheses stated above: The first null-hypothesis was tested for each individual diver using the runs test (cut-point = median). The second null-hypothesis was tested using paired samples t-tests. The

third null-hypothesis was tested using linear regression and a one-sample t-test to find whether regression coefficients differed from zero. Caloric responses before and after the dive were compared. For this purpose the maximum SPV after the four irrigations were added and divided by four. The fourth null hypothesis, i.e. that no difference in response would be found between these two measurements, was tested using a paired samples t-test. The distribution of the path lengths did not differ significantly from the normal distribution (Kolmogorov-Smirnov and Shapiro-Wilk tests). The type I error level was set to $\alpha = 0.05$.

RESULTS

Before the dive, two subjects had abnormal ENG (unilateral caloric weakness). They were excluded from this study. Figure 2 shows how postural stability, as measured by path length, changed during the dive for each individual diver (rows) and in each test condition (columns). The first null-hypothesis, that only random variations in postural sway

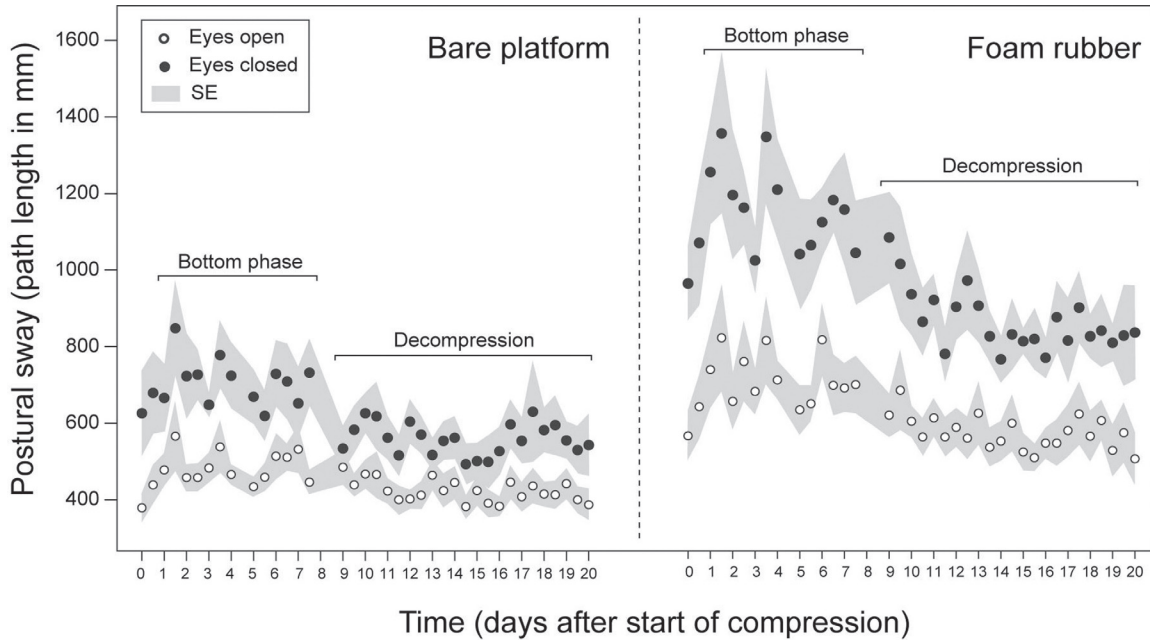


Fig. 3. Mean postural sway in six divers measured 38 times on a force platform in four different test-conditions during a 19-day simulated saturation dive in helium-oxygen to 240 msw. Path length, i.e. the movement of the center of pressure (in mm) during one minute of quiet standing, is plotted against time (days after start of compression). Left panel: with subjects standing on bare platform. Right panel: standing on a 10 cm thick foam rubber mat.

would be found during the dive, was rejected in all subjects except one. The figure shows how, in the sequences with significant changes, balance tended to be poorer (dots = worse than median) in the beginning of the dive, i.e. during the bottom phase, and better (circles = better than median) toward the end of the dive, i.e. during the decompression phase. The postural sway increased after reaching storage depth compared to before compression (paired-samples t-tests), disproving the second null-hypothesis. The mean increase (95% confidence intervals in parentheses) was 26% (8 – 45) with eyes open, 6% (-39 – 52) with eyes closed, 31% (8 – 53) when standing on foam rubber with eyes open and 30% (18 – 42) when standing on foam rubber with eyes closed. Figure 3 shows mean postural sway throughout the dive, reaching a maximum at the time of the fourth measurement, i.e. approx 12 hours after arriving at 240 msw, and slowly decreasing

TABLE 1. Linear Regression Coefficients of Path Length (MM) vs. Time (Days) during the Bottom Phase.

Diver	Eyes open	Eyes closed	Standing on foam rubber	
			Eyes open	Eyes closed
1	12	-18	-3	-16
2	1	-8	0	-8
3	-13	21	8	-21
4	-6	1	-25	-8
5	27	15	21	-5
6	-30	-62	-50	-102

during the decompression phase. Table 1 shows linear trends in postural sway vs. time during the bottom phase. The third null-hypothesis could not be rejected. The most consistent result was that there was a trend (one-sample t-test: $p = 0.1$) toward improvement in postural sway vs. time when standing on foam rubber with eyes closed. The mean improvement was 27 mm/day with a 95% confidence interval of -13 to 66 mm/day. Measurements performed during

(n=72) or directly after (n=108) excursions did not differ from the median during bottom phase (binomial distribution: $p > 0.2$).

The otoneurological examinations before and after the dive were unremarkable with the following exceptions: One subject reported vertigo, disequilibrium and nausea on reaching 240 msw. The symptoms improved gradually during the dive, and his postural sway returned to pre-dive values, but he still reported dizziness and a subjective feeling of disequilibrium after the dive. The pathogenesis remains unclear in spite of extensive investigations (including MRI). When reviewing the data, another subject was found to have had signs of a possible subclinical right sided vestibular lesion (caloric weakness, left-beating post-head-shake nystagmus and increased postural sway) on the first post-dive examination 1-2 days after the dive. Although DCS would explain these findings, he denied any symptoms of dizziness or imbalance at the time, and no DCS was diagnosed or treated. There was no difference ($p = 0.9$) in mean caloric response before the dive (mean SPV = 13.8 deg/s) and after (mean SPV = 14.1 deg/s). The fourth null-hypothesis could not be disproved.

The two divers that were excluded from this study due to unilateral caloric weakness before the dive, displayed the same changes as the others in postural sway during the dive, i.e. there were significant changes in postural sway during the dive ($p < 0.05$), postural sway increased after reaching saturation pressure, and there was a trend toward improvement during the bottom phase.

DISCUSSION

The aim of this study was to measure postural stability at 240 msw, to document the time course, and to discuss possible mechanisms. This depth was of interest, since a previous study had indicated that 200 msw

could be a “critical point” in the development of postural sway (9). The increase in body sway found at 240 msw is in agreement with previous findings at 100 and 450 msw (9,10). In addition, this study documents the great inter-individual differences ranging from severe vertigo, disequilibrium and nausea in one subject to no symptoms and only random variations in postural sway in another. In the remaining four, there was measurable postural instability, but to a moderate degree. It is also interesting to note that the two divers who were excluded because of possible unilateral vestibulopathy pre-dive displayed the same changes as the others in postural sway.

The severity of HPNS signs and symptoms is greater the deeper the dive and the faster the compression rate (1). The relatively slow compression rate in this dive to 240 msw was not expected to cause much HPNS. Nevertheless, the measured instability lasted throughout the 7-day bottom phase and returned to normal during the decompression, indicating an effect mostly related to absolute depth. This may still be consistent with HPNS, but means that platform posturography could be more sensitive than other methods in detecting this syndrome. In repeated testing, an improvement in balance may occur due to a learning effect, i.e. that the subjects learn to perform the test better, thus masking some of their symptoms. Such a learning effect has been documented previously (14). This effect would cause greatest improvement during the bottom phase, when the subjects had the opportunity to practice balancing on the platform 15 x 4 times over a period of eight days. However, in this study, most of the improvement in balance occurred later, during the decompression, which cannot be explained solely by a learning effect.

Platform posturography is a practical way of measuring the total performance of the balance system, which depends on input through vestibular, visual and proprioceptive

pathways. When used clinically, it provides relevant information about the degree of a balance disturbance, but there is no general agreement that this information can be used to localize any pathology within the central or peripheral vestibular system (15, 16). The method of decreasing visual and proprioceptive input by eye closure and by standing on a foam rubber mat respectively has been described previously by Norré (13). In the present dive, the most significant effects seemed to occur when standing on foam rubber with eyes closed, which is when balance is most dependent on vestibular function.

Other methods have been used in order to pinpoint effects of pressure to specific parts of the vestibular system, but the results have been equivocal and difficult to interpret. Since diving is known to cause changes in the function of the inner ear through a number of mechanisms such as barotrauma, decompression sickness, alternobaric vertigo and caloric stimulation, it would be very interesting to learn whether pressure in itself has an effect on the vestibulo-ocular reflex arc. Adolfson et al (2) reported severe imbalance and a statistically significant decrease in nystagmus beats and amplitude during passive head rotations in 10 divers breathing air at 90 msw. While this could be interpreted as an effect of pressure on the vestibulo-ocular reflex, the authors considered the decrease too small to explain the severe imbalance, and concluded that the latter was of central nervous origin and related to nitrogen narcosis. Braithwaite et al (3) found marked postural imbalance and a slight decrease in the amplitude of optokinetic and caloric nystagmus in 6 divers breathing heliox at 485 msw, but it was concluded that this was within normal limits, and that the measured imbalance was due to HPNS. Molvær (4) measured caloric response in eight divers during the bottom phases of two dives to maximum pressures of 300 and 504 msw. The caloric response

was reduced in six of six divers participating in the dive to 300 msw, but only in two of six in the deeper dive. Renon (5) found decreased nystagmus induced by caloric stimulation in six of eight divers breathing trimix at 450 msw. In contrast to this, Gauthier (6) found increased gain of the vestibulo-ocular reflex during passive sinusoidal rotations in two divers at 610 msw. Nordahl et al (9) found a decrease in caloric response in four divers after a heliox dive to 450 msw. In the present study, no change in caloric response was found 1-2 days after the dive. Thus, the results point in different directions. Even if one accepts the results of the majority of studies, indicating a small depressive effect on VOR-gain, interpretation of the finding is difficult, since a general decrease in alertness, as for instance in HPNS, inert gas narcosis or simply due to lack of sleep while in the chamber, would also be expected to cause a decrease in VOR-gain.

In conclusion, increased ambient pressure to 240 msw in helium-oxygen was associated with postural instability. The instability lasted throughout the 7-day bottom phase. The finding is consistent with HPNS, but the long duration in spite of the relatively slow compression rate in this dive to 240 msw indicates that platform posturography could be more sensitive than other methods in detecting this syndrome.

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