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Alterations of the human vestibulo-ocular reflex in a simulated dive at 62 ATA

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Gauthier, G. M. Alterations of the human vestibulo-ocular reflex in a simulated dive at 62 ATA. 1976. Undersea Biomed. Res. 3(2): 103-112.–In an attempt to investigate some aspects of the high pressure nervous syndrome, the vestibulo-ocular reflex (VOR) gain was measured in two professional divers undergoing a simulated dive at 62 ATA. The aquanauts in a seated position were rotated sinusoidally around the vertical axis at a frequency of about 0.3 Hz over a 20° range. Tests were performed at regular intervals prior to, during, and after the compression/decompression period. The rotations were applied either in total darkness or with a visual target rotating with the chair or with a target fixed to the chair-supporting frame. An infrared photoelectric system monitored eye movements. The results showed no spontaneous nystagmus, but two definite changes in VOR gain: (1) a slight but significant increase related to pressure increase, which may be due to an increase of the vestibular system excitability or a decrease of the cerebellar inhibition exerted upon the vestibular nuclei, and (2) an intermittently appearing increase (VOR gain between 1 and 1.3) during brief periods. The latter finding, not related to pressure, was interpreted as the expression of an underwater-adapted mode that may developed in professional divers submitted to the intensive use of magnifying diving-optical systems.

simulated dive
rotatory vestibular stimulation
heliox
vestibulo-ocular reflex adaptation

The function of the vestibulo-ocular reflex (VOR) is to maintain a stable fixation and provide stability of the visual world during head and body movement. In a normal situation, stable vision is achieved if the eye movement is equal, but opposite, to the head movement. The gain of the VOR (ratio of eye-to-head velocity) is then 1.0. The apparent stability of the outside world is achieved through a more complex operation that involves interpretation by the central nervous system of visual, oculomotor, and vestibular signals.

The localization of seen objects is based on the extraretinal knowledge of eye position from the efferent copy of the eye-muscle motor command (Skavenski, Haddad, and Steinman 1972). During head or body movement, the subjective calculation of relative target position must be the subjective estimate of the head-movement amplitude from the vestibular signal, minus the eye movement from the efferent copy, plus the retinal signal that is the distance of the target image with respect to the fovea. When the three internal signals are properly calibrated, the observer sees no illusory apparent movement. Illusory movements may be perceived when any one of the signals loses proper calibration. This may occur through internal alterations of the various settings (lesions) or after changing

externally the visual (prisms or magnifying lenses) or vestibular (caloric or electric stimulations) input references. When sufficient time is allowed in the above clinical and experimental situations, adaptation may occur and apparent stability may be regained.

When the visual field– or a specific target– moves with the head or the body, the VOR has to be suppressed (gain = 0) in order to maintain stable fixation. It has also been shown that a normal subject can mentally set his VOR gain during rotations in total darkness. For instance, a subject asked to imagine and fixate on a target rotating with him can lower his VOR gain to values close to 0.3 and increase it to about 0.9 with an imaginary target fixed to the wall (Gauthier and Volle *in press*). When a subject is given a mental arithmetic task, the gain tends towards values comprised between 0.45 and 0.6 in the frequency range around 0.1 Hz (Collins 1962; Guedry 1968; Melvill-Jones 1971; Meiry 1971; Gauthier and Volle 1974).

Similar visual and mental tasks were given to two professional aquanauts undergoing a COMEX-CNEXO-sponsored simulated dive at 62 ATA with helium-oxygen (heliox) in order to study the VOR alterations due to high pressure. Clinical observations and diver's subjective reports from an earlier simulated dive suggest that the vestibular system functions may be seriously disrupted by absolute pressure, gas nature, and compression/decompression dynamics. Such alterations of the vestibular functions may be responsible for the visual (nystagmus) and vegetative (nausea and vertigo) disorders described in the literature (Appaix and Demard 1972). This study of the VOR in men exposed to high pressure may help us understand some aspects of the high pressure nervous syndrome (Brauer, Dimov, Fructus, Fructus, Gosset, and Naquet 1969; Bennett and Towse 1971) and of adaptation to an underwater environment (Ross, Franklin, Weltman, and Lennie 1970).

METHODS

The VOR was tested by rotating the diver sinusoidally on a chair driven through a swinging adjustable-length pendulum seen to the left of Fig. 1A. The vertical-axis chair

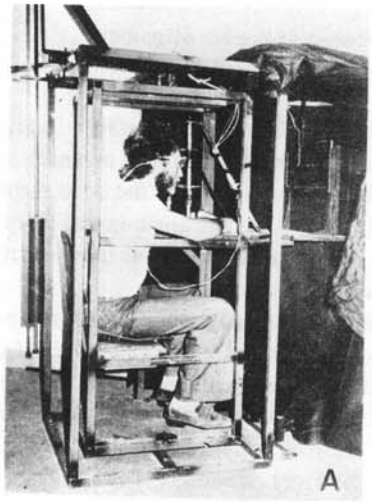

Fig. 1A. Experimental apparatus. The VOR was tested by rotating the subject sinusoidally through a pendulum which was installed in the lightproof lower chamber of the three-sphere diving simulator (see text for details).

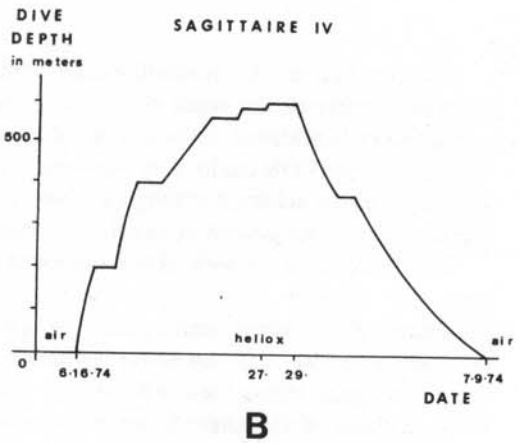

Fig. 1B. Simulated dive SAGITTAIRE IV. The compression/decompression rate is represented approximately in this graph. The two aquanauts spent 50 hours at 62 ATA in an experiment which lasted 23 days.

rotated over a 20° range at a near sinusoidal frequency of 0.3 Hz. A potentiometer provided head (chair) position with respect to the frame. The driving pendulum was manually started and stopped by the second diver. The low-amplitude decrement made it possible to obtain five to six successive oscillations with practically the same amplitude. A bite bar and a forehead rest immobilized the diver's head.

An infrared photoelectric system monitored eye movements (Gauthier and Volle *in press*). Calibration was obtained in the otherwise completely dark chamber by asking the diver to fixate alternately on targets (red photodiodes, 1 mm in diameter) presented over a 30° range on a mat-black screen situated 57 cm from his head (Fig. 1A). The target light intensity was adjusted to approximatively three times the visual subjective-threshold intensity. The center target of the black screen was subsequently used as a chair-fixed target while the frame-fixed target was a similar photodiode attached coaxially to the chair-fixed target, to the pendulum outerframe. It is to be remembered that in a normal subject, the chair-fixed target should induce a VOR gain of zero while the frame-fixed target should set it at one. A black cloth cover made the pendulum inner space perfectly lightproof. The pendulum was installed in the lightproof lower chamber of the three-sphere diving simulator.

The target light intensity (on/off), the head position, and the eye position were simultaneously recorded on a 1000-Hz band-pass paper-chart recorder. Tests were performed at regular intervals prior to, during, and after the compression/decompression period. Figure 1B shows the compression/decompression time course. Compression of the chamber was started on day 1. Successive rapid-compression periods followed by constant-pressure intervals gradually brought the chamber pressure, 11 days later, to an equivalent depth of 610 msw. The divers remained at maximum pressure for 50 hours. Then, 10 days of slow decompression brought the chamber back to atmospheric pressure. The whole experiment lasted 23 days.

RESULTS

General observations

Monitoring of eye movements in total darkness at regular intervals during the simulated dive did not reveal the presence of spontaneous pathological nystagmus. On reaching pressures above 41 ATA, the divers experienced mild discomfort but never reported nausea or vertigo. Rotation in darkness– with or without a fixation target– did not trigger spontaneous nystagmus nor did it induce motion sickness.

Figures 2-5 and numerical data in the text refer to one diver, although two were tested and both showed similar VOR alterations.

Rotation during fixation of a frame-fixed target

When tested in the laboratory and in the chamber at pressures below 41 ATA during the compression phase, the divers showed a smooth-pursuit eye movement of equal amplitude and in the opposite direction to head rotation (Fig. 2A). Only a few microsaccades of small amplitude were necessary to compensate for the mismatching of eye and target velocities.

As the pressure increased, the fixation error increased as shown in the recording of Fig. 2B, which was taken at 53 ATA. A full cycle was necessary for the VOR gain to build up to the required value of 1. During the following cycles, the mismatching of eye and target velocities (the VOR gain was inadequate) occasioned large and numerous saccades. This disorder persisted during the decompression phase of the dive and several days after return to atmospheric pressure.

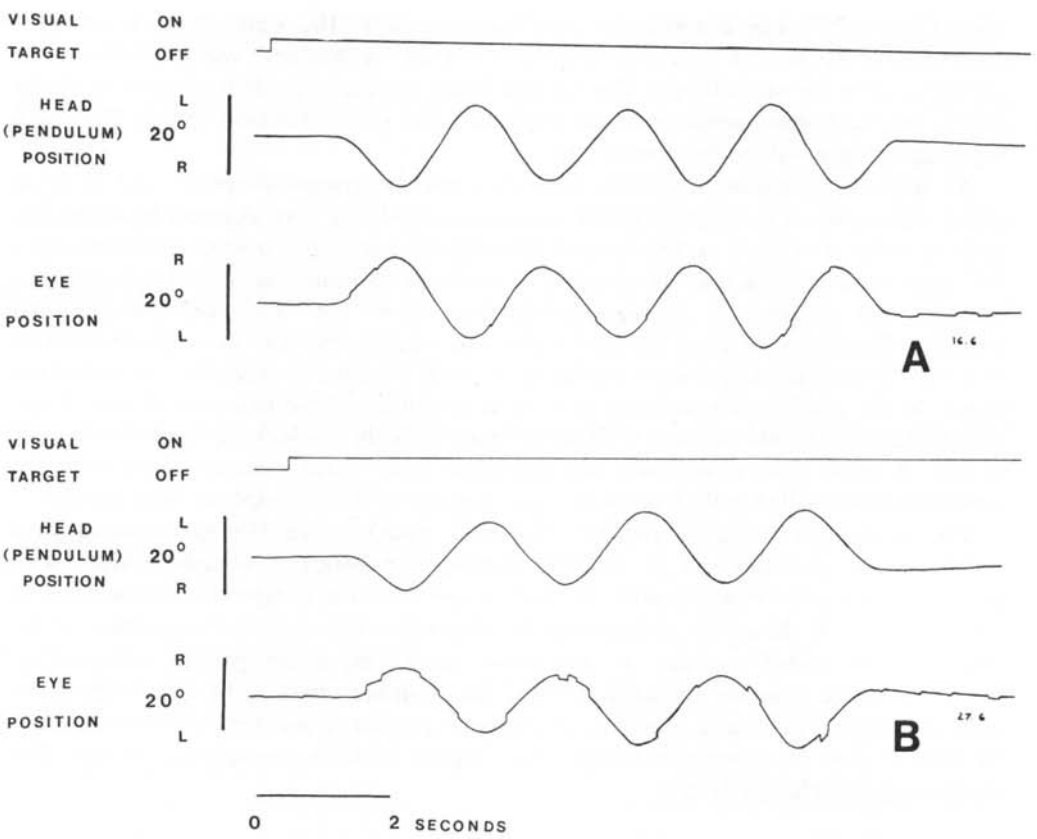

Fig. 2. Rotation-induced compensatory eye movements during fixation of a target attached to the pendulum outer frame. At ground level and at pressures below 41 ATA, rotations applied to the diver induced compensatory eye movements similar to those represented in A. At higher pressures and during decompression, the compensatory eye movements deteriorated. B shows recordings taken at 59 ATA during decompression. *In this and all the following figures, the target state, the head, the eye, and eventually the cumulative eye positions are successively represented as a function of time.*

Rotation during fixation of a chair-fixed target

At ground level and pressures up to 41 ATA, rotation during fixation of a chair-fixed target demonstrated eye movements of small amplitude as shown in Fig. 3A. The VOR gain decreased rapidly to zero so that the overall fixation error did not exceed 2-3°. As the pressure increased beyond 41 ATA, the fixation error increased up to between 6 and 8°. As noted in the previous test, the alterations of fixation stability persisted during the decompression phase and after return to normal pressure.

Rotation in total darkness

During rotation in total darkness, the diver was instructed to fixate on a target and count backwards by 3 from a given number between 100 and 1000. Soon after the fixation target was turned off and, about 1 s later, the chair was set into rotation. Under these conditions

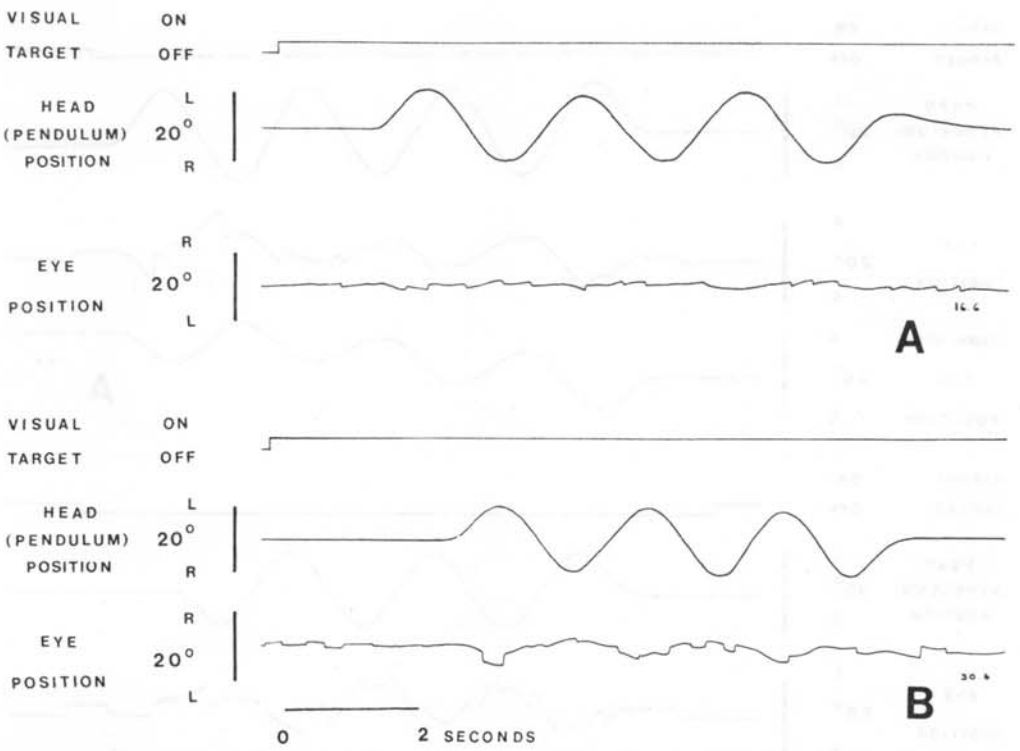

Fig. 3. Chair-fixed, target-fixation eye movements during rotation. In the laboratory and at low pressures, rotations applied to the diver during fixation of a chair-fixed target induced eye movements similar to those shown in A. At pressures above 41 ATA and during decompression, the fixation error increased markedly. The recordings in B were taken at 59 ATA during decompression.

rotations at pressures below 41 ATA induced a VOR with an average gain of 0.50 ± 0.06. Figure 4A shows the head position (pendulum), the overall eye position, and the cumulative eye position (that is the sum, end-to-end, of the smooth-pursuit segments) recorded at 21 ATA. As the pressure increased, the gain tended to increase. At 59 ATA (as shown in Fig. 4B) it was 0.65 ± 0.1. During the decompression phase the VOR gain remained high and the standard deviation increased even more. Four days after atmospheric pressure was resumed, tests carried out in the laboratory still showed a slightly hypernormal VOR gain.

A special examination of the VOR recordings showed no detectable phase variations between eye and head velocities due to the pressure.

VOR alterations not related to pressure

VOR tests also revealed an interesting phenomenon. The diver was rotated in total darkness and required to fixate on a frame-fixed imaginary target. The VOR gain was generally measured between 0.8 and 0.9 (Fig. 5A). Rapid increases of VOR gain to values up to 1.3 were observed, however, for brief periods of time (Fig. 5B). These randomly occurring changes, which lasted from 30-60 s, were not related to the experimental conditions inasmuch as they were observed in the laboratory prior to the dive as well as during pressure exposure. The high-gain periods could be terminated by asking the diver to switch fixation to a chair-fixed imaginary target.

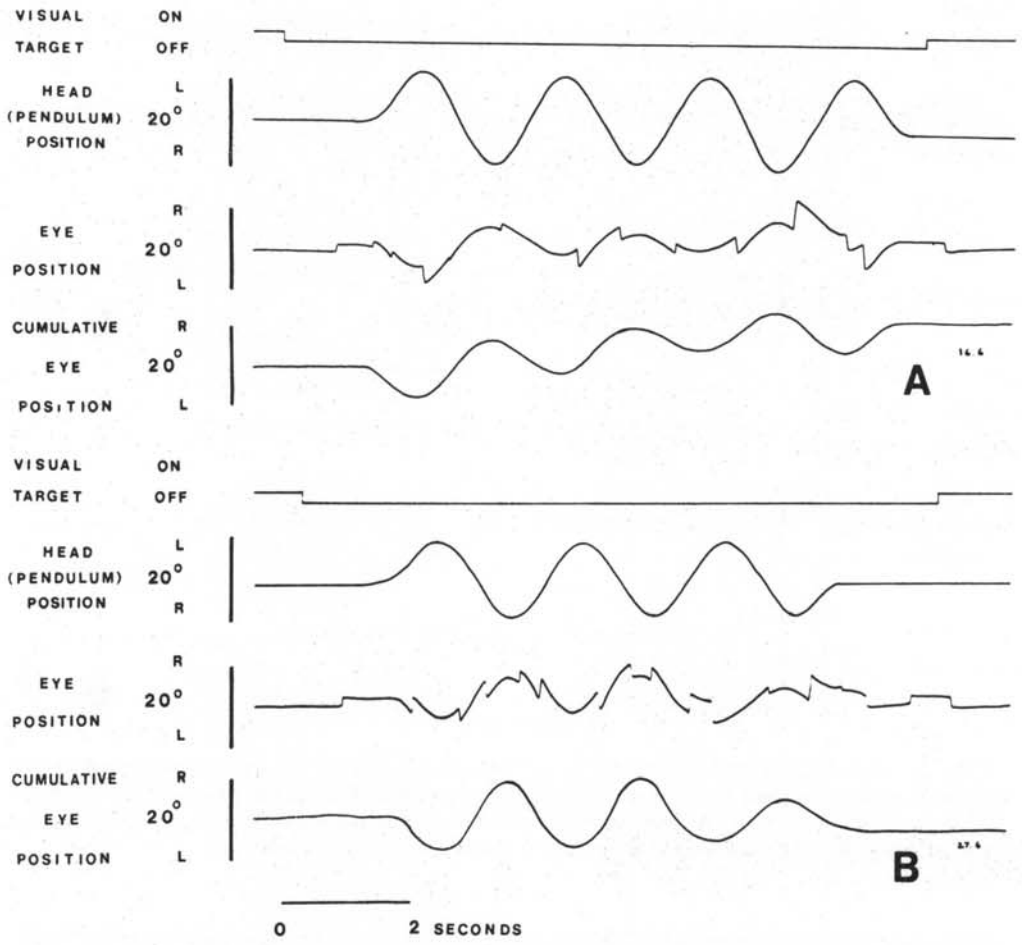

Fig. 4. Compensatory eye movements of a diver performing mental arithmetic at low pressures (A) and at pressures above 41 ATA (B) during rotation in total darkness. The lower trace of A and B shows the cumulative eye position.

DISCUSSION AND CONCLUSIONS

VOR gain increase in relation to the experimental conditions

Rotations during fixation of chair-fixed and frame-fixed targets showed a significant increase in the fixation error as shown in Figs. 2B and 3B. The oculomotor instabilities observed in the two situations had probably the same origin– that is, a dysfunction of the systems responsible for the setting of the gain value (1 or 0) from visual and vestibular inputs. The results of a previous experiment (Gauthier 1974), which were confirmed by the present study, showed that steady target-fixation error increased markedly as the pressure increased beyond 21 ATA. It was concluded that the fixation instability was probably caused by retinal and cortical dysfunctions (error detector and forward-loop controller), rather than by motor dysfunction, since saccadic velocity was not altered.

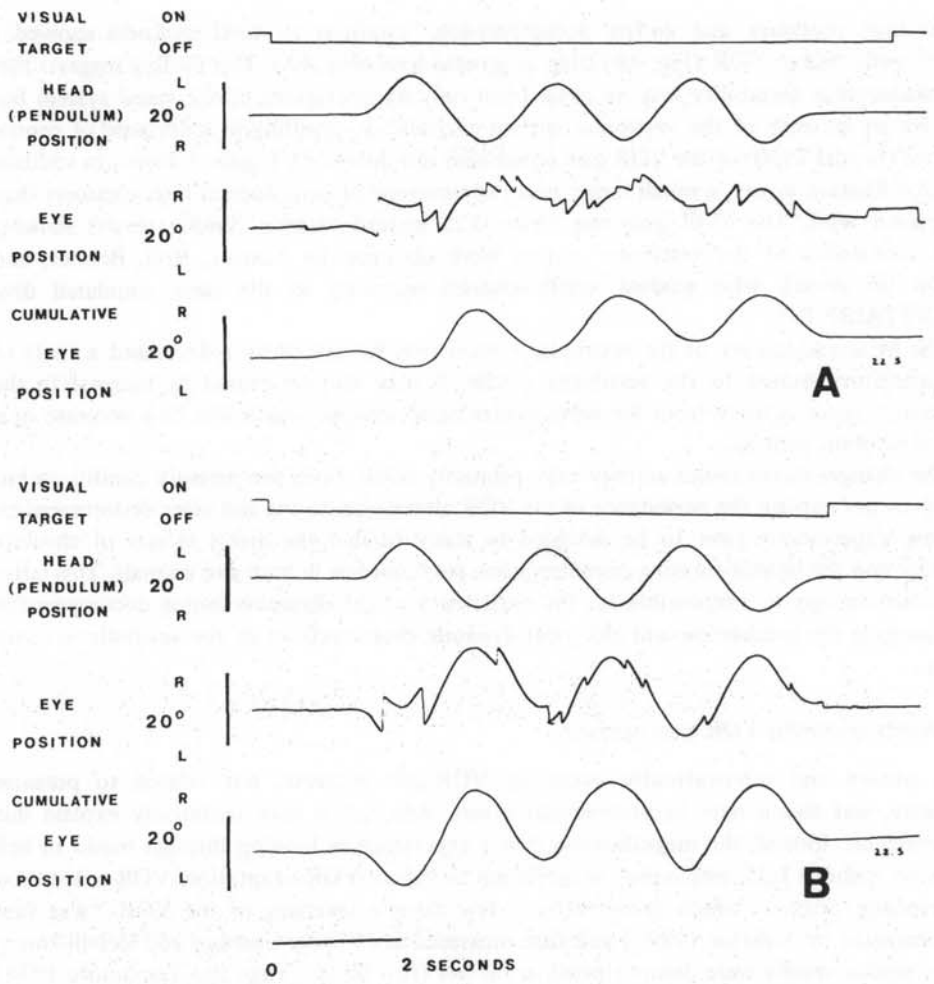

Fig. 5. VOR adaptation in professional divers. Compensatory eye movements during fixation of a frame-fixed imaginary target. A : regular mode (gain of 0.8 to 0.9) and B : underwater-adapted mode (gain up to 1.3).

In a normal situation, when a subject is rotated, the direction of the target-image slip over the retina is used at some level of the visual-vestibular system (cerebellum and/or colliculi) to set the VOR gain to the appropriate value of zero (chair-fixed target) or 1 (frame-fixed target). At high pressures, a decrease of velocity and position-detector sensitivity may disrupt the normal functioning of the gain-setting system, causing large errors in position and velocity.

The eye-movement instability may also result from a decrease in the central control of the vestibular output, causing, in turn, an inadequate VOR only slightly corrected by visually induced eye movements.

At high pressures and during decompression, rotations in total darkness showed a significantly higher VOR (Fig. 4B) than at ground level (Fig. 4A). This finding suggests that the oculomotor instability may be caused not only by alterations of the visual system but also by an increase of the vestibular system excitability, resulting in a decrease of central control (Brodal 1960) of the VOR gain (cerebellar disinhibition). Figure 3 shows, in addition to large fixation errors, a much larger mean eye-movement amplitude at high pressures than at ground level. The VOR gain was about 0.25 instead of zero. Similar results showing hyperexcitability of the vestibular system were obtained by Lacour, Roll, Bonnet, and Hugon (*in press*), who studied vestibulospinal reactivity in the same simulated dive (SAGITTAIRE IV).

The hyperexcitability of the vestibular system may be caused by a decreased activity of the inhibitory inputs to the vestibular nuclei. It may also be caused by increase in the excitatory input activity from the semicircular canal sensory organs due to a decrease or a lack of afferent control.

The changes in vestibular activity may primarily result from the pressure conditions but this does not explain the persistance of the VOR alterations during and after decompression.

New experiments have to be designed to study further the direct effects of absolute pressure and the bubble-forming decompression phenomenon in man and animals. This latter phenomenon may be responsible for the persistence of the disorders during decompression by changing the mechanical and electrical dynamic characteristics of the vestibular-sensory organ.

Randomly occurring VOR-gain increase

A sudden and intermittently occurring VOR-gain increase, not related to pressure exposure, was found only in professional divers. Adaptation may tentatively explain this phenomenon. Indeed, the magnification divers experience in looking through masks or bell windows is about 1.35, which may be sufficient to induce VOR adaptation. VOR adaptation to reversing prisms– which cause, after a few days, a reversing of the VOR– was first demonstrated by Stratton (1897) and first measured in 1973 by Gonshor and Melvill-Jones. Later, similar results were demonstrated in rabbits (Ito, Shiida, Yagi, and Yamamoto 1974) and in cats (Robinson 1975). Adaptive increases and decreases of VOR gain were demonstrated in monkeys (Miles and Fuller 1974). In an attempt to verify the hypothesis according to which the high gain measured in professional divers might be due to conditioned adaptation, Gauthier and Robinson (1975) demonstrated a clear increase of VOR gain in a man who was fitted for 5 days with × 2.1 magnification telescopic lenses.

These results and other studies (Ross, Franklin, Weltman, and Lennie 1970) strongly suggest that the professional divers studied in SAGITTAIRE IV may have developed a dual-mode VOR with one gain for land activity and another for underwater activity, either of which might appear sporadically during rotation in the dark.

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Gauthier, G. M. Les alterations du reflexe vestibulo-oculaire chez l'homme pendant une plongée fictive à 62 ATA. 1976. Undersea Biomed. Res. 3(2):103-112.–Pour étudier des aspects oculaires du syndrome nerveux des hautes pressions, l'augmentation du reflexe vestibulo-oculaire (RVO) a été mesurée chez deux plongeurs professionnels pendant une plongée fictive à 62 ATA. Les plongeurs, assis, ont subi une rotation sinusoïdale autour d'un axe vertical à une frèquence de 0,3 Hz sur un champ de 20°. Les tests ont eu lieu régulièrement avant, pendant et après la période de compression/décompression. Les rotations ont été accomplies soit dans l'obscurité totale, soit en présence d'un objectif visuel qui tournait avec la chaise, soit en présence d'un objectif attaché au support de la chaise. Les mouvements oculaires ont été suivis par un système photoélectrique infrarouge. Aucun nystagmus spontané n'a été observé, mais deux changements de l'augmentation du RVO ont été notes: (1) une augmentation modest mais significative, en rapport avec l'augmentation de pression qui est due soit à une excitabilité accrue du système vestibulaire, soit à une baisse de l'inhibition cérébellaire exercée sur les noyaux vestibulaires; et (2) une augmentation intermittente (augmentation du RVO entre 1 et 1,3) pendant des périodes courtes. On a interprété cette dernière donnée, sans rapport à la pression, comme l'expression d'une mode d'adaptation sous-marine, développée chez des plongeurs professionels qui se servent intensivement des appareils optiques de grossissement.

plongée fictive
hélium-oxygène
stimulation vestibulaire rotatoire
adaptation du réflexe vestibulo-oculaire

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