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Nature and treatment of decompression sickness occurring after deep excursion dives

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Greene KM, Lambertsen, CJ. Nature and treatment of decompression sickness occurring after deep excursion dives. Undersea Biomed Res 1980; 7(2):127–139.—During Predictive Studies IV (PS IV), the fourth in a series of collaborative undersea investigations, plans were made for excursion compressions to 1200 and 1600 feet of sea water (fsw) from saturation depths of 800 and 1200 fsw, respectively. Three cases of decompression sickness (DCS) occurred in the excursion phases of PS IV; all were relieved by prompt treatment, and there were no residual effects. This paper describes the rationale and treatment regimen used for deep excursion DCS, reports the results of the treatment, and makes specific recommendations for the therapy of DCS occurring after such excursions.

decompression sickness
saturation-excursion diving
case history
therapy

The technique of making excursion dives from saturation depths has been used for over a decade. During the development of decompression profiles for excursion exposures, there have been surprisingly few reports of decompression sickness (DCS) occurring as a result of the excursion exposure per se.

In a collaborative investigation designed to determine both physiological and performance decrements during rapid compression and the rate of adaptation to compression effects, accelerated decompression procedures were developed for deep saturation-excursion dives (1, 2). This investigation, designated Predictive Studies IV (PS IV), offered an opportunity to study six subjects during saturation-excursion decompressions from 1200 to 800 fsw (37.4–25.2 ATA) and from 1600 to 1200 fsw (49.5–37.4 ATA). To achieve the repetitive exposures required by the experimental goals, we were required to use excursion decompressions more rapid than any available at the time. Techniques to generate the decompression profiles for PS IV are described in Lambertsen et al. (2). Reported here are the three incidents of DCS that occurred as a result of the excursion decompressions. The treatment procedures in these cases are discussed, and procedures, based on the PS IV experience, are recommended for the treatment of such incidents of DCS.

METHODS

The purposes and design of the PS IV program are described in detail elsewhere (2). Subjects for these experiments were six healthy, trained, male professional divers (age range, 23–29 years), who were informed of the risks of deep simulated diving experiments and of the nature of these experimental decompressions. None of them had any history of serious DCS.

Predictive Studies IV consisted of two phases; each comprised saturation exposures to a helium-oxygen atmosphere. Phase I included repetitive, rapid excursions to 1200 fsw from a base depth of 800 fsw. Phase II excursions reached 1600 fsw from 1200 fsw. The compression times for the excursions were either 20 or 40 min. Time at maximum depth was 55 min or less, followed by staged decompression. During compressions and at stable depths, the chamber Po_2 was kept between 0.21 and 0.23 ATA and the Pco_2 was kept below 6 torr (0.008 ATA). Ambient temperature was controlled for the subjects' comfort.

A low-resistance breathing system was used to administer hyperoxic helium-oxygen mixtures during decompression and treatment gases in the event of DCS. Normal medical instrumentation was supplemented by equipment for electronystagmography (ENG) and bone-conduction audiometry.

CASE REPORTS OF EXCURSION DCS IN PS IV

One incident of spinal cord DCS and one incident involving vertigo occurred in Phase I of PS IV. One incident involving vertigo occurred during decompression from an excursion in Phase II. All three subjects responded to prompt recompression, and there were no residual effects. Case histories of these incidents are provided. For PS IV purposes, subjects are designated by numbers.

Case 1

During his third exposure day in Phase I, Subject 6 was being decompressed back to 800 fsw from his third excursion from 800 to 1200 fsw in 3 days. The excursion profile (Fig. 1) involved a 40-min compression and an actual bottom time of 55 min. Normoxic helium-oxygen was breathed during the compression and bottom phases. The scheduled decompression duration was 89 min, during which time 2.7% O_2 in helium was breathed. At 830 fsw (26.2 ATA) (min 71 of the elapsed decompression time), Subject 6 noted the onset of paresthesias and numbness in his right leg and thigh. Although he was the exercise subject and was seated on the bicycle ergometer, no exercise was performed during the decompression. It was first considered possible that he had a simple sciatic neurapraxia from the pressure of his thigh against the seat. Decompression continued while he walked about the chamber or lightly pedalled the ergometer to aid diagnosis. After the return to 800 fsw, the paresthesias spread downward to his toes and upward, eventually reaching the T-6 dermatome on the right. He also had slight weakness and tremor of the right leg and aching in the right flank.

As part of the overall diagnostic procedures, one of the other subjects elicited the following signs under supervision: hyperesthesia to pin prick on the right trunk, leg, and thigh; decreased temperature sense on the right; and normal patellar tendon reflexes.

Recompression of all four subjects (for operational and safety reasons) was begun 52 min after the first mention of symptoms. Rate of pressurization averaged 16 fsw/min (0.5 atm/min). Subject 6's flank pain was relieved while passing 900 fsw (28.3 ATA), and recompression was stopped at 950 fsw (29.8 ATA) because the remaining symptoms began to regress. All signs

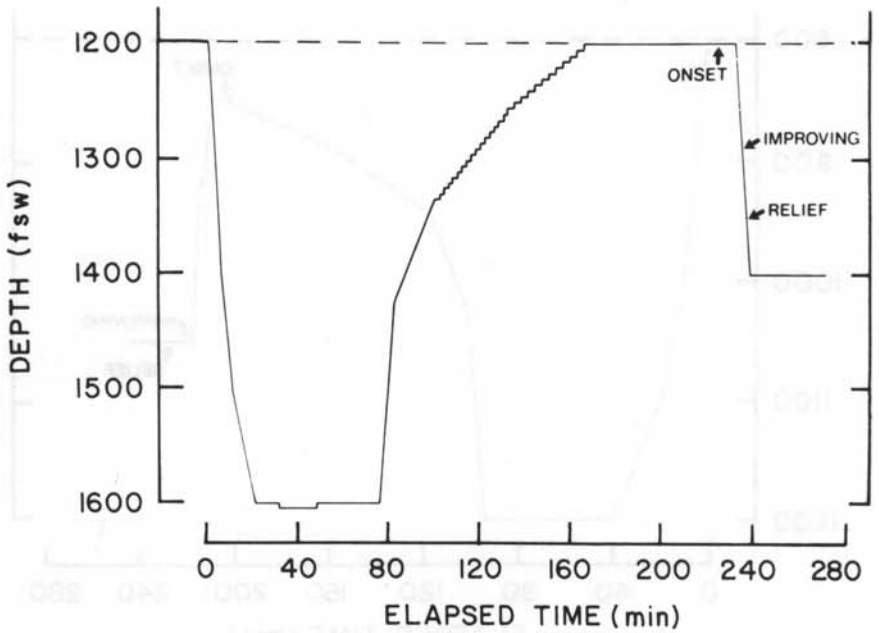

Fig. 1. Exposure profile showing time of onset and relief of decompression sickness in Case 1 (Subject 6) during Phase I of Predictive Studies IV.

and symptoms had resolved within 19 min after leaving 800 fsw. Administration of treatment gas was begun at 830 fsw. The gas was given by mask in 20-min cycles interrupted by 5 min of chamber atmosphere (Po_2 = 0.2 ATA); the first two cycles consisted of 7% O_2, with the balance helium, and were followed by 8% O_2 in helium for six more cycles.

Although the DCS was apparently overcome at 950 fsw, the subjects were recompressed to 1000 fsw (31.3 ATA) for added safety; that pressure was reached 30 min after leaving 800 fsw. Decompression back to 800 fsw at the rate of 7 min/fsw (231 min/atm) began 12 h and 34 min after the start of therapy and was uneventful. As a precautionary measure, Subject 6 was given two cycles of 7% O_2 breathing at the beginning of decompression. He made no further excursions and had no DCS during the final ascent to the surface. Detailed neurological examination after he surfaced revealed no residual deficit.

Case 2

During his fifth exposure day in Phase I, Subject 2 incurred inner ear DCS after his third excursion to 1200 fsw. This excursion was begun 25 h and 50 min after all of the subjects had returned to 800 fsw from the treatment of Subject 6. Compression lasted 40 min and the actual bottom time was 55 min (Fig. 2). The decompression schedule planned for this excursion would have required nearly twice the time of that used for Subject 6 (2).

Before decompression from his stay at the excursion depth of 1200 fsw, Subject 2 felt nauseated and dizzy, but nystagmus was not present. He did not report difficulty in equalizing his ears during either descent or ascent. The symptoms cited resolved at about 945 fsw (29.6 ATA) during decompression, but at 850 fsw (26.8 ATA) he reported he was "spinning" and a pronounced right-beating nystagmus was seen simultaneously on the ENG; this nystagmus

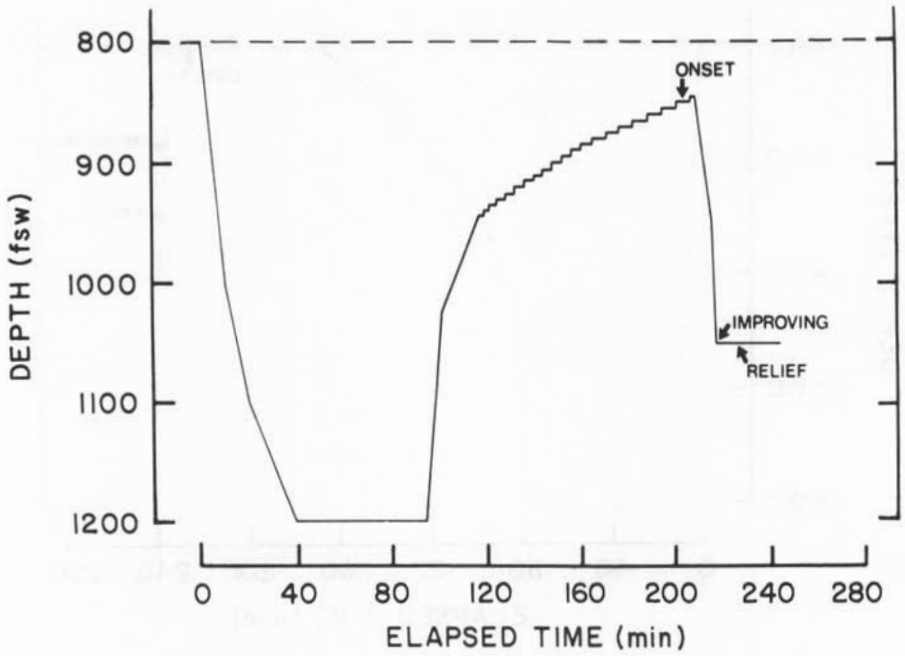

Fig. 2. Exposure profile showing time of onset and relief of decompression sickness in Case 2 (Subject 2) during Phase I of Predictive Studies IV.

reached a maximum of 36°/s for the slow component. He soon developed nausea but did not vomit.

Recompression from 845 fsw (26.6 ATA) was begun within 5 min of onset and was halted at 1050 fsw (32.8 ATA) 9 min later, because the vertigo and nausea were improving. Administration of therapeutic gas (7% O_2, balance helium) began at 875 fsw (27.5 ATA) in a total of eight 20-min cycles, interrupted by 5 min of chamber atmosphere breathing. The ENG leads were disconnected temporarily to allow Subject 2 to lie on a cot, but the recording was restored 7 min after reaching 1050 fsw and showed greatly reduced nystagmus that had changed to left-beating. No nystagmus and no vertigo were present 2 min later. Despite the change in direction of the ENG, Subject 2 did not feel any change in the direction of the vertigo (i.e., the environment spinning counterclockwise). No subjective hearing loss was reported, but transient right-sided tinnitus was noticed at 1050 fsw. Bone-conduction audiometry at this pressure showed an equivocal 15 dB decrease on the right at 4 kHz. More extensive repeated measurement about 3.5 h after onset showed no loss of auditory acuity, no spontaneous nystagmus, minimal positional nystagmus, and a slight decrease in the threshold for electrical vestibular stimulation (2).

Although Subject 2 had no further inner ear problems, he did have an episode of pain-only DCS during final ascent to the surface. Examination at the surface revealed normal vestibular responses to warm and cold caloric tests. Audiometry on the day after surfacing showed a uniform average decrease of about 15 dB bilaterally in bone and air conduction compared to the baseline examination. The next day the decrease averaged about 25 dB, but 5 weeks later the audiogram was the same as the one taken during the baseline examination. Upon surfacing, the otologic physical examination was normal, as were results of the remainder of the neurological examination.

Case 3

On his fifth exposure day in Phase II, Subject 4 developed inner ear DCS after decompression from his third excursion to 1600 fsw. The excursion profile required a 20-min compression from 1200 fsw to 1600 fsw, 55 min of actual bottom time with hard work underwater on a scaled-down oil wellhead, and an 89-min decompression (Fig. 3). While underwater the subject was immersed in fresh water (33.6°C) at a pressure equivalent to 1610 fsw (49.8 ATA). The breathing apparatus was a modified Kirby-Morgan Bandmask supplied with 2% O_2 in helium. The subject breathed this 2% O_2 mixture for 19 min, the final 16 min of which was in the water. For the remaining 28 min at 1600 fsw, he breathed chamber gas while acting as tender for another subject. The chamber oxygen partial pressure was initially normoxic, but it rose to 0.5 ATA by the end of the time at maximum pressure because of admixture with the hyperoxic breathing apparatus exhaust. A mixture of 3.2% O_2 in helium was breathed by mask during the decompression. Although Subject 4 did not dry himself or remove his wet clothes while serving as tender for another subject or during the decompression, he covered himself with a blanket and did not complain of being cold.

A sudden onset of vertigo was experienced by Subject 4 59 min after reaching 1200 fsw and just after eating dinner. Although he had no nausea, the vertigo was prominent, and he was assisted to a cot. Immediately before the onset of vertigo, he had performed the Valsalva maneuver repeatedly, commenting that his right ear felt "stuffy," and that he had been trying to clear it. He had no tinnitus and no noticeable hearing loss.

Recompression was begun 7 min after the initial complaint of vertigo, at a rate of about 30 fsw/min (0.9 atm/min). The vertigo "slowed down" at 1290 fsw (40.1 ATA) and stopped while passing 1350 fsw (41.9 ATA), 5 min after leaving 1200 fsw. Recompression was halted at 1400 fsw (43.4 ATA) 2 min later. The treatment gas mixture (5% O_2, balance helium) was started at 1290 fsw and was continued for six cycles of 20 min each. Electronystagmography was performed 5 min after reaching 1400 fsw, and no spontaneous nystagmus was seen. Positional stimulation did not produce nystagmus, and no significant change was seen in bone-conduction audiometry. Although this subject later suffered pain-only decompression sickness, he had no further inner ear symptoms. On the day after surfacing, he had normal caloric responses bilaterally and no significant change in audiometry from his baseline examination.

DISCUSSION

Subject 6 had a definite episode of spinal cord DCS, which presumably affected at least the left spinothalamic tracts and possibly the right pyramidal tract. Despite a delay of 52 min before recompression was begun, the subject was completely relieved of his symptoms after 19 min of treatment.

In Subject 2, both the onset and relief of vestibular DCS were dramatically confirmed by electronystagmography. Bone-conduction audiometry at 1050 fsw showed no residual loss, and there were no inner ear symptoms during subsequent ascent to the surface. A temporary bilateral decrease in auditory acuity later observed at the surface remains unexplained. The transient dizziness and nausea reported at 1200 fsw before the decompression began were not accompanied by nystagmus and therefore could not be attributed to inner ear barotrauma. These symptoms may have been caused by rapid compression from 800 to 1200 fsw.

For Subject 4, the interval of 59 min between return to saturation depth and the onset of symptoms was unexpectedly long. Because he had been seen attempting to clear his ears after

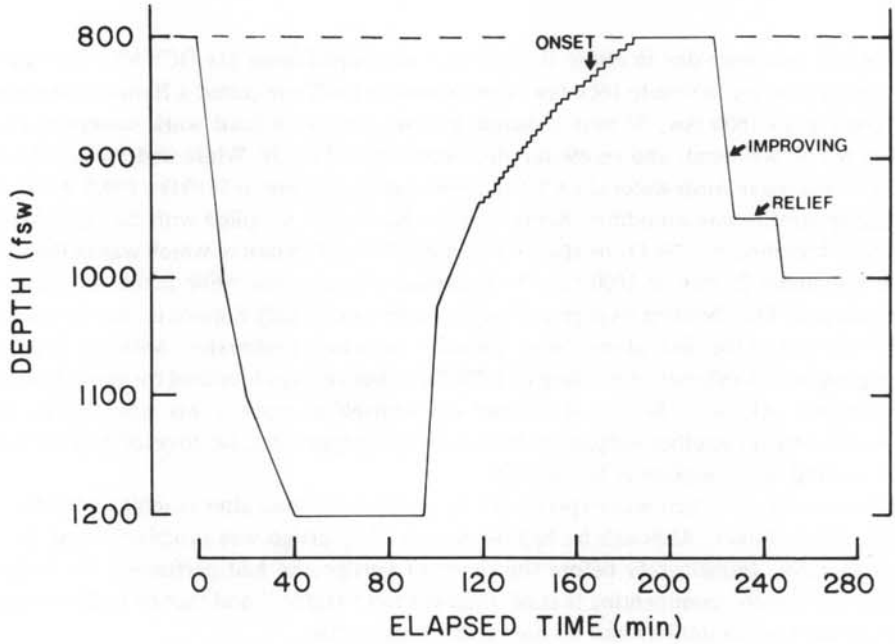

Fig. 3. Exposure profile showing time of onset and relief of decompression sickness in Case 3 (Subject 4) during Phase II of Predictive Studies IV.

decompression, alternative etiologies for his vertigo were considered, such as forceful autoinflation (alternobaric vertigo) and perilymph fistula from barotrauma (3). Nystagmus was not observed because the subject had complete relief before the ENG electrodes were reapplied. However, the severity, time course, and response to recompression all tend to support the diagnosis of DCS. Indeed, this subject may have felt the need to clear his ears because of a transient hearing loss caused by the onset of DCS.

All three subjects were completely relieved of their symptoms by recompression to less than the depth of the excursion. Subject 6 was symptom-free within 19 min at 950 fsw, a pressure increase ($\triangle P$) of 150 fsw (4.5 atm). Subject 2 had relief within 18 min at 1050 fsw ($\triangle P = 205$ fsw [6.2 atm]). Subject 4 achieved relief in 5 min at 1400 fsw ($\triangle P = 200$ fsw [6.1 atm]). The pressure increases represented by these recompressions were of sufficient magnitude to reverse the hypothetical supersaturation that controlled the decompression calculation (2). The ratios of treatment depth to depth of onset were all less than 1.24 : 1. This ratio would result in a volume reduction of only 19%, by Boyle's Law, of any gas volume contributing to the DCS. For a hypothetical spherical bubble, the corresponding reduction in diameter would be only 7%. In contrast, a recompression from surface pressure to 60 fsw (2.8 ATA), as in the standard U.S. Navy Treatment Tables 5 and 6 (4), causes a volumetric decrease by 64% and diameter reduction by 29%. In planning for PS IV, we recognized that it would not be possible to accomplish a recompression from 800 fsw or deeper that would approach the compression ratio of the standard treatment tables. Accomplishing such a ratio would require a recompression to more than 2300 fsw (70.7 ATA). To plan a rational approach to this therapeutic problem, we reviewed both the limited published reports of DCS after saturation-excursion dives and the larger body of information pertaining to DCS in deep diving in general.

Review of saturation-excursion DCS

The earliest reported systematic excursion trials consisted of 17 man-excursions (breathing air) to pressures equivalent to 100–165 fsw (4,0–6,0 ATA) from an air saturation pressure of 35 fsw (2.1 ATA), (5); these excursions were all free of DCS. U.S, Navy investigators continued this developmental work during the SEALAB series, using the Workman model of allowable gas supersaturation (6). After SEALAB, a total of 1123 man-excursions were made from saturation pressures ranging from 150 to 850 fsw, all using helium-oxygen, with no incidents of DCS (7, 8, 9, 10, 11). Furthermore, no cases of DCS were reported by Krasberg after another series of exposures involving 4000 h of excursion diving at sea and in the laboratory; schedules similar to those of the U,S. Navy were used for these dives (12). In light of more recent developments, it is clear that the successful decompressions of these early efforts did not even approach the DCS threshold.

In a recent program of decompression experiments at the U.S. Navy Experimental Diving Unit, the permissible excursion depths and ranges in helium-oxygen saturation diving have been extended (4). During that program, two episodes of DCS occurred, which were attributable solely to the excursion ascent. The first took place after a 5-h excursion to 450 fsw (14.6 ATA) from a saturation depth of 300 fsw (10,1 ATA); symptoms consisted of pruritus and chest discomfort, which were 95% relieved by recompression to 350 fsw (11.6 ATA) and by having the subject breathe a hyperoxic mixture (13). The second incident consisted of spinal cord involvement, which occurred after an ascending excursion to 800 fsw (25.2 ATA) from a saturation depth of 1000 fsw (31.3 ATA). Relief was achieved in this case by recompression to 1000 fsw (14).

There are five other published reports of DCS cases resulting from helium-oxygen excursions. Three occurred during a Compagnie Maritime d'Expertises (COMEX) LUDION procedure involving repetitive excursions from 120 meters of seawater (msw) to 180 msw (396–594 fsw, 13.0–19.0 ATA) (15). All three of these cases involved vestibular symptoms that were relieved by recompression and drug therapy; treatment pressures were not reported.

During the diving experiment known as JANUS IV, another diver was treated for vestibular DCS after returning to 400 msw (1320 fsw, 41.0 ATA) from an excursion to 460 msw (1518 fsw, 47.0 ATA). Treatment consisting of 20-msw (66 fsw, 2,0 atm) recompression, hyperoxygenation, and the administration of unspecified drugs, was completely successful. Two episodes of minor joint pain also occurred during this dive, but these incidents were not treated (16). Another reported incident of vertigo and nausea occurred after a diver had made six excursions in 2 days to 300 msw (990 fsw, 31.0 ATA) from a saturation depth of 250 msw (825 fsw, 26.0 ATA) during British Navy saturation diving trials (17); symptoms in this case were relieved by immediate recompression to 300 msw.

During an earlier phase of the British Navy trials, saturation decompression were accomplished in a stepwise fashion. Although these were not intended as excursion experiments, each step resembled a maximum upward (no-stop) excursion to a lower pressure (18). In the course of these exposures (see Phase I data in ref, 19), 17 decompression incidents occurred, of which two involved inner ear symptoms. In the Phase I series (19), there was no correlation between the depth of onset of symptoms and the degree of recompression (ΔP) needed for relief. One of the inner ear cases was managed successfully by recompression from 47 msw (155 fsw, 5.7 ATA) to 69 msw (228 fsw, 7.9 ATA); the other improved slowly after recompression from 47 to 75 msw (248 fsw, 8.5 ATA). In none of the cases was more than a 30-msw (99 fsw, 3.0 atm) recompression applied.

Review of DCS in other types of high pressure exposures

Because there had been so little experience with DCS resulting from deep excursion diving, other kinds of pressure exposures, which might be applicable to PS IV conditions, were reviewed. It seemed likely for two reasons that DCS caused by extreme excursions at great depths might resemble DCS occurring in very deep nonsaturation dives from the surface. First, large inert gas gradients are expected in both cases, and second, in each situation the degree of gas phase volume change in response to pressure changes at great depth would be similar.

A review of U.S. Navy experience (20) showed that in helium-oxygen subsaturation diving, onset depth of DCS was approximately proportional to dive depth. Further, the probability of successful treatment was related to the ratio of absolute pressure of treatment to pressure of onset. The probability of relief for serious DCS was greater than 96% if this ratio was 1.91 : 1.0 or higher.

In 23 cases of DCS occurring in the British Navy during decompression from deep, experimental helium-oxygen dives, six consisted of neurological or inner ear symptoms (21). Of the five incidents with onset deeper than 200 fsw (7.1 ATA), four involved the inner ear. The mean recompression differential pressure ($\triangle P$) for therapy in these cases was 178 fsw (5.4 atm) and the maximum was 270 fsw (8.2 atm); the recompression ratios were all less than 2 : 1.

Other investigators have noted that inner ear symptoms (vertigo or hearing loss or both) are the principal manifestation of DCS in deep subsaturation diving (22, 23). Immediate recompression to the depth of the dive is reported to have been successful in a series of experiments at 650 fsw (20.7 ATA), when DCS onset occurred at 470, 450, or 390 fsw (15.2, 14.6, and 12.8 ATA), (23). In another series (24), prompt treatment by recompressing 100 fsw (3.0 atm) greater than the depth of onset is reported to have relieved vertigo and deafness and to have left no residual effects.

Bühlmann and Gehring (25) reported 11 cases of DCS affecting the inner ear, which occurred during helium-oxygen dives to pressures between 16 and 31 ATA (495 and 990 fsw). The six patients who were completely cured were recompressed an average of 2.4 atm (average ratio 1.2 : 1). Five divers had incomplete relief, even though the group average recompression was greater. None of the total of 11 divers was recompressed to the full depth of the dive.

Recompression depth

In the subsaturation cases cited above, none of the recompression ratios was less that 2 : 1. Even that small ratio, however, would not be feasible from the 1200-fsw depth planned for PS IV. In the more analogous series reported by Hanson et al. (19), the average ratio for serious symptoms was 1.5 : 1, but none of those recompressions exceeded 30 msw (99 fsw, 3.0 ATA). Of the true saturation-excursion incidents cited, four were reported in sufficient detail to determine recompression ratios. These ratios ranged from 1.05 : 1 to 1.24 : 1. The largest $\triangle P$ was 200 fsw (6.1 atm). The latter values are quite similar to those that proved effective in PS IV.

These observations lend support to the hypothesis that guided planning for the treatment of DCS in PS IV, namely, that the successful treatment of DCS of deep onset depends more on the manipulation of the inert gas gradients between bubbles and tissue or blood, than on the volumetric compression of gas according to Boyle's Law (26).

The resolution of bubbles in blood or tissue has been discussed by Van Liew (27), Van Liew and Hlastala (28), and Hills (29). Hlastala and Van Liew (30) add a more rigorous treatment of

the transient state. However, most of these analyses are primarily concerned with bubble dynamics in the steady state or during decompression. It is possible to predict the factors and trends involved in the response to recompression, but none of the cited publications provides a concrete basis on which to propose guidelines for recompression depth. In the particular cases of saturation-excursion diving, the most difficult problem is to define a stopping point, a maximum recompression that will ensure cure without exceeding a safe depth.

As in the general case, three choices are available. The patient may be recompressed to the depth of relief, to the depth of the causative dive, or by some arbitrary increment (31). The depth of relief is the most straightforward concept, but has two serious problems. First, the end-point of relief may be difficult to determine when the medical observer is remote from the patient. Second, the tissue damage from DCS is sometimes apparently irreversible. In that case, complete relief would not be obtained and the recompression would continue until some limit was forced to be set.

The depth of the causative dive is an end-point sufficient to assure that supersaturation can no longer exist and inert gas gradients must be outward from any bubbles, provided the respired inert gas has not been changed or the Po_2 lowered. Even in the case in which gas remained at its site of origin and completely obstructed the local circulation, recompression to the depth of the dive would assure that all of the gas could eventually be re-dissolved in the same tissue if none were removed by the circulation.

The value of any arbitrary treatment depth, whatever the theory behind it, must be determined by experience. Before the PS IV studies, there was insufficient evidence to support any particular arbitrary approach to excursion DCS.

During PS IV, the three patients were compressed at moderate speed (16–30 fsw/min [0.5–0.9 atm/min]), under medical supervision, to the depth of relief. Since unequivocal improvement was seen well before reaching the depth of the excursion, recompression was halted long enough to confirm complete resolution of symptoms and signs. No attempt was made, however, to find the precise minimum recompression required. The least recompression previously reported as effective in excursion DCS was 50 fsw (1.5 atm), and the maximum, 200 fsw (6.1 atm). No case has been reported in which the depth of the excursion was exceeded during therapy. Our experience suggests that recompression to somewhat less than the depth of the dive can be sufficient. The rationale given above, and the experience of others, supports the use of the depth of the dive as the limit for recompression if relief is not obtained at a shallower depth.

Use of hyperoxia

Hyperoxic treatment gas was used in the three episodes of deep DCS that occurred in PS IV; the administration of hyperoxic breathing mixtures has been a treatment procedure of long standing at the Institute for Environmental Medicine for any DCS therapy. However, one of the subjects had already begun to improve before the gas could be given. Other investigators have not found hyperoxygenation to be necessary, either in deep diving from the surface (21, 25) or in at least one saturation-excursion incident (17). The desire to administer treatment gas should not be allowed to delay the primary therapy, recompression, and appropriate attention must be paid to oxygen toxicity when hyperoxia is used.

Use of ancillary agents

Other than oxygen, no drugs were used in the treatment of our patients. Whether any drug would be needed or effective in excursion DCS is not evident from the literature.

Final decompression

The optimal duration of treatment at depth and the optimal rate for decompressing divers to the surface after treatment of excursion DCS have not been established. Nevertheless, it is noteworthy that two of the three patients described in this report subsequently developed pain-only DCS during the final ascent to the surface. This correlation was not discussed in the case reports cited earlier. Spaur (32) states that knee pain following excursion ascents tends to recur during the final 100 fsw (3.0 atm) of ascent, but he is referring to cases of pain that, in fact, first appeared during the saturation decompression shortly after an excursion ascent (14).

Limitations imposed by high pressure neurological effects

In planning for PS IV, the potential handicap to therapy presented by the neurological effects of deep recompression was considered. Because of this potential problem, the initial excursion trials to 1200 fsw (Phase I) were made from a saturation depth of 800 fsw. It was not anticipated that the high pressure nervous syndrome (HPNS) would limit therapeutic compression from this base depth, even if recompression to the full depth (1200 fsw) was undertaken. Information about compression rate tolerance gleaned from Phase I of PS IV guided planning for Phase II; it was thought that HPNS might prove limiting at the greater pressures required in Phase II.

An illustrative example of the effect of HPNS on therapy is an instance of inner ear DCS, consisting of incapacitating vertigo, which occurred after a rapid decompression from saturation at 1500 fsw (46.5 ATA), (33, 34). Recompression from 1160 to 1535 fsw (36.2–47.5 ATA) ameliorated but did not eliminate DCS symptoms. In this case, however, the first diver's partner developed symptoms of HPNS, a development that curtailed both the depth and the rate of recompression that could be used. Because there is a clear correlation between promptness of treatment and successful outcome (20, 21, 35), these limitations of recompression undoubtedly affected the outcome.

In the cases that occurred during PS IV, recompression was not curtailed by symptoms of HPNS, in spite of compression rates up to 30 fsw/min (0.9 atm/min) to depths as great as 1400 fsw. The same subjects, in the rapid excursions earlier in the experiment, had been noticeably affected by tremor, nausea, dizziness, and decreased alertness. There was clear evidence of adaptation to these repetitive compressions (2). However, even the first 400-fsw (12.1 atm) excursion to 1600 fsw (49.5 ATA) was sufficiently well tolerated to make therapeutic compression at that rate and depth entirely acceptable.

The ease of achievement of treatment depth in our experiment contrasts dramatically with the case reported by Leitch (33). Because we are unable to identify with certainty the factors responsible for this difference, it is unwise to expect that therapeutic compression will not again be limited by HPNS. There are, however, no known pathologic sequelae to HPNS in humans. On the other hand, DCS can easily produce permanent damage. It is reasonable, then, to risk severe symptoms of HPNS if necessary to attain therapeutic depth promptly.

CONCLUSIONS

The helium-oxygen excursion DCS experience before PS IV, which has been reviewed in this paper, comprised four episodes of inner ear dysfunction, one episode affecting the spinal cord, and one involving chest discomfort with pruritus. Two more instances of inner ear symptoms and one incident of spinal cord DCS from PS IV excursions can now be added, as

well as the vestibular case reported by Gardette et al. (16). Presumably, any DCS syndrome is possible in deep excursion diving, but it can be predicted that inner ear lesions will occur frequently in decompression incidents resulting from this type of diving.

Clearly, it is possible to achieve complete relief by prompt treatment of deep excursion DCS and this should be the goal of treatment. If rapid and progressive improvement is noted during recompression, further recompressions may be halted briefly to ascertain whether complete relief follows. If there is any doubt about residual damage, it would be best to recompress the patient to the depth of the excursion. This is especially important when inner ear lesions are present and hearing cannot be tested with precision, as occurs in operational diving, where accurate diagnosis and evaluation are not possible. If the incident occurs in a submersible compression chamber (bell), recompression should be initiated in the bell before transfer to the deck decompression chamber, because any delay in starting treatment reduces the chance of complete cure.

Although the excursion schedule of PS IV did not allow more than one excursion per day, other techniques or circumstances do permit repetitive excursions in the same day. If DCS occurs after one of these, the appropriate treatment depth should be the depth of the deepest excursion of the preceding 24 h.

Our recommendations for the treatment of DCS that occurs as a result of excursions from helium-oxygen saturation dives include:

- Prompt recompression at a rate of approximately 30 fsw/min to the pressure of relief or to the pressure of the deepest excursion of the preceding 24 h.
- Hyperoxic gas mixture to be given after the recompression is initiated.
- Careful observation of unresolved deficits at depth to determine whether further recompression and adjunctive therapy are needed.
- Finally, the development of symptoms of HPNS during recompression should not be allowed to interfere with prompt recompression to treatment depth.

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Greene KM, Lambertsen CJ. L'origine et le traitement de la maladie de décompression laquelle se manifeste après des excursions sous-marines profondes. Undersea Biomed Res 1980; 7(2):127–139.—Au cours des Etudes Prédictives IV (PS IV), la quatrième d'une série d'enquêtes sous-marines en collaboration, des plans furent dressés pour accomplir des compressions en excursion à 1.200 et 1.600 pieds en eau de mer (fsw) à partir de profondeurs de saturation de 800 et 1.200 fsw, respectivement. Il y a eu trois cas de maladie de décompression au cours des phases d'excursion IV; tous furent remédiés par un traitement rapide, et il n'y eut aucun effet résiduel. Ce papier illustre l'explication et le système de traitement employé dans le cas de la maladie de décompression en excursion profonde, et rapporte les résultats de ce traitement, tout en avançant des propositions particulières touchant à la thérapeutique de la maladie de décompression résultant de telles excursions.

maladie de décompression
plongée saturation/excursion
passé médical
thérapeutique

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