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Decompression sickness during saturation dives

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Berghage, T. E. 1976. Decompression sickness during saturation dives. Undersea Biomed. Res. 3(4):387–398.—Available Navy saturation diving data were analyzed for an evaluation of the therapeutic adequacy of decompression sickness treatment procedures and for delineation of precipitant factors in the etiology and treatment of decompression sickness during saturation dives. None of the cases of decompression sickness recorded during saturation dives involved more than musculoskeletal or joint pain, and in 96% of the cases the joint pain was confined to the diver's knees. In 89% of the cases symptoms appeared while the divers were still under pressure. The subsequent recompression treatment of these cases resulted in full relief in only 35% of the cases; the remaining 65% completed the therapy and subsequent decompression with residual pain which diminished over a period of weeks. The adequacy of the recompression appears to be inversely proportional to the depth of reported onset of symptoms and the time required to obtain even partial relief is directly related to the magnitude of the recompression ratio used. Four explanations are suggested for the limited recompression therapy common in saturation diving: increase in musculoskeletal pain with recompression, peer pressure to avoid extension of the chamber confinement, lack of severe neurological symptoms, and the tremendous depths required to obtain a reasonable recompression ratio. The author further suggests that future treatment procedures will require a departure from the accepted concept of radically decreasing the volume of inert gas bubbles by increasing pressure.

decompression sickness
saturation dives
recompression treatment procedures

In the past 10 years saturation diving has progressed from a laboratory experiment to an operational tool for both the U.S. Navy and commercial divers. Advancements in diving hardware have provided the fleet with three operational deep-dive systems; a Navy saturation diving school has been established; and, for the first time, the *U.S. Navy Diving Manual* (1973) contains a chapter on saturation diving. All of this technology has been designed to allow man to work at deeper depths for longer periods of time.

Saturation diving makes up less than 0.3% of the U.S. Navy diving. Yet it accounts for over 25% of the time spent under pressure and 20% of the cases of decompression sickness that occur (Fig. 1a, b, c) (Table 1). Although the overall incidence of decompression sickness is not high, it poses a hazard that must be overcome. The work of Goodman and Workman (1965) provides an adequate treatment regimen for the routine fleet subsaturation dive, but for

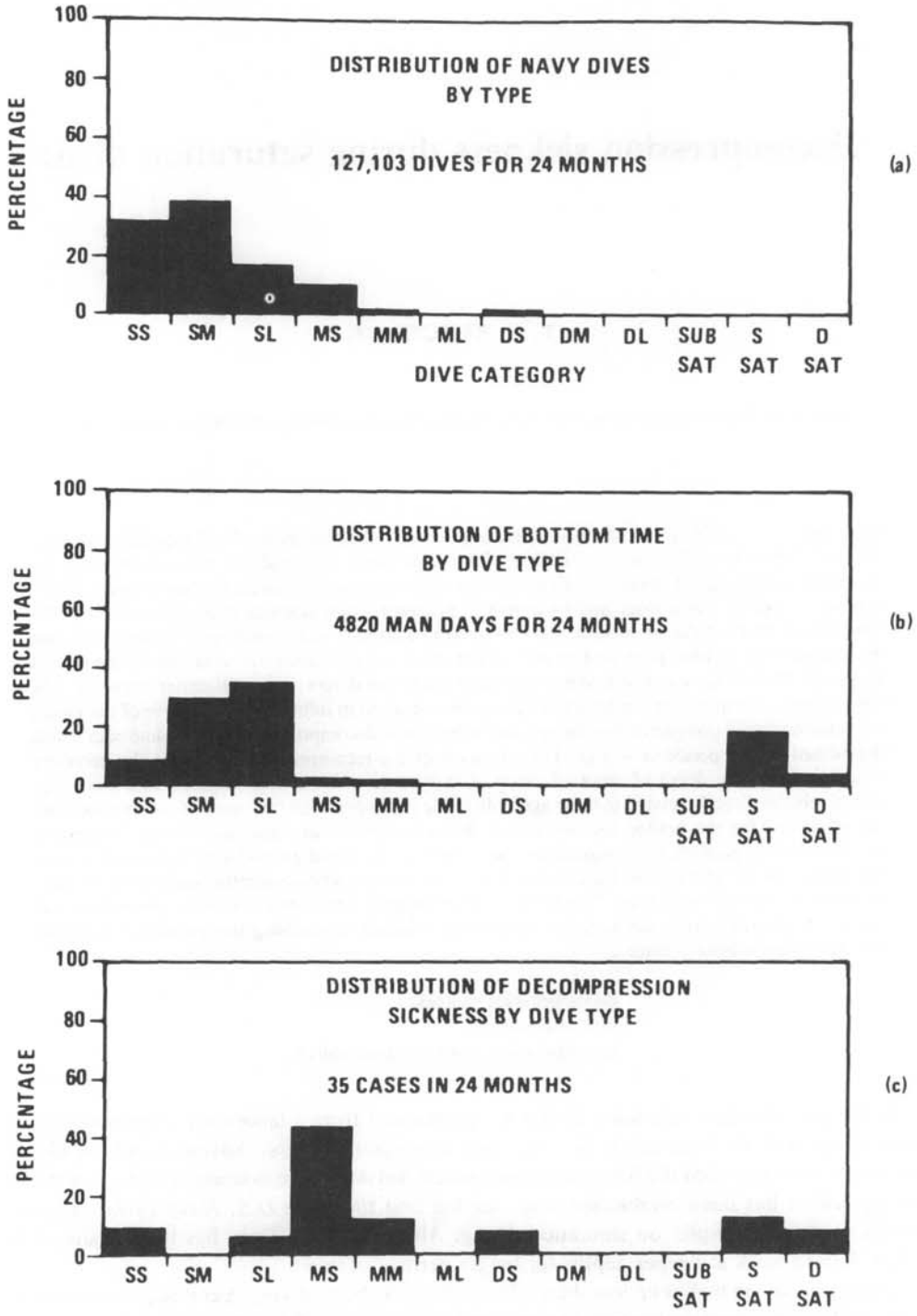

Fig. 1. Distribution of Navy dives and decompression accidents by dive category.

TABLE I

Description of dive categories*

Code	Depth	Bottom time	Depth (fsw)	Bottom time (min)
SS	Shallow	Short	<100	<30
SM	Shallow	Medium	<100	30-60
SL	Shallow	Long	<100	>60
MS	Medium	Short	100-200	<30
MM	Medium	Medium	100-200	30-60
ML	Medium	Long	100-200	>60
DS	Deep	Short	201-300	<30
DM	Deep	Medium	201-300	30-60
DL	Deep	Long	201-300	>60
Sub Sat	Subsaturation		>300	<12 (hr)
S Sat	Shallow	Saturation	≤300	≥12
D Sat	Deep	Saturation	>300	≥12

*From Berghage, Rohrbaugh, Bachrach, and Armstrong (1975).

saturation dives the *U.S. Navy Diving Manual* provides only treatment guidelines. A standard treatment technique for saturation dives suggested by Summitt, Berghage, and Every (1971) did not produce acceptable results; therefore, this review and analysis were undertaken.

I analyzed the available Navy saturation-diving data to evaluate the therapeutic adequacy of the procedures that have been used for treating the saturation diver stricken with decompression sickness and to delineate precipitant factors that may be important in the etiology or treatment of decompression sickness occurring during saturation dives.

METHOD

Data sources

Two sources of saturation-diving data were surveyed. First, the U.S. Navy Safety Center (NSC) in Norfolk, Virginia, was asked to search their computerized diving-data bank for the saturation dives made during the 24-month period between January 1972 and December 1973. During this 24-month period, 265 man saturation dives were made, of which 7 resulted in decompression sickness. We obtained and analyzed data on these dives to identify etiological factors suitable to estimate the likelihood that a case of decompression sickness would occur.

The historical files of the U.S. Navy Experimental Diving Unit (NEDU) provided the second source of saturation-diving data. These historical data, obtained from several sources, consisted of 27 saturation decompression-sickness cases from which adequate details were available. A number of these cases came from the NEDU in-house diving logs, which provided detailed descriptions of the sequence of actions taken during the therapeutic procedure. I used these diving-log data primarily to assess the adequacy of various treatment regimens.

Data analysis

NSC data Six experimental independent variables were identified and evaluated for a determination of whether they are related to the incidence of decompression sickness. These six independent variables included the environmental variables of dive depth, bottom time, and environmental temperature, along with the human variables of age, height, and weight.

NEDU data Nine experimental independent variables were selected for analysis, including dive depth, bottom time, an exposure index: $\frac{\text{depth} \times \text{bottom time}}{100}$, diver age, height, weight, weight-to-height ratio, and the recompression ratio used in treatment. These nine variables were correlated with three experimental dependent variables: depth of reported onset of symptoms, depth of reported relief of symptoms, and the time to reported relief of symptoms.

The systematic evaluation of the preceding variables involved the calculation of descriptive and inferential statistics; least-squares best-fit curves were calculated for those relationships reaching statistical significance.

RESULTS

The data analysis indicates the overall incidence of decompression sickness on these U.S. Navy saturation dives was about 2.64%, which is slightly higher than the 1.7% reported by one of the leading commercial diving companies. This slightly higher percentage may result, in part, from the deeper experimental saturation dives conducted by the Navy. The important point is that the incidence of decompression sickness on saturation dives is about seven times higher than that of routine subsaturation dives.

NSC data

The saturation-diving data obtained from the U.S. Navy Safety Center were correlated with the presence or absence of decompression sickness symptoms (Table 2). The nonsignificant results probably can be attributed to one or more of the following: (a) the small number of cases of decompression sickness in the sample, (b) the lack of precise dive-recording procedures, or (c) the fact that indeed there is no relationship between these variables and the incidence of decompression sickness. Until contrary results are obtained, however, we must assume that none of these variables substantially alters decompression outcome. Identification of any predisposing factors with the available NSC data was impossible, focus was shifted to evaluation of the therapeutic adequacy of present treatment techniques using the data from NEDU.

NEDU data

The saturation-dive data from the Navy Experimental Diving Unit logs provide some interesting and statistically significant results. Decompression sickness occurring during saturation dives within the U.S. Navy has been characterized by musculoskeletal pain alone (Type I) rather than the more serious symptoms involving the cardiopulmonary system, the central nervous system, and the organs of special sense (Type II). The onset is usually gradual and

TABLE 2

Correlations between various human and dive-profile variables and incidences of decompression sickness

	Variables	r
Human	Age	−.006
	Height	.032
	Weight	−.026
Dive Profile	Depth	.119
	Bottom time	.029
	Environmental temperature	.080

generally occurs while the diver is still under pressure. Of the 27 cases in this study, 11% of the stricken divers reached the surface (1 ATA) before reporting symptoms (Table 3). It is very likely that the symptoms were present before the divers reached the surface but were not sufficiently severe to overcome the individual's reluctance to report them. The depth of onset of symptoms for divers reporting symptoms while still under pressure is shown in Fig. 2. About 60% of the divers reported their symptoms within 1.5 atm of the surface; very few saturation decompression-sickness symptoms have been reported at pressures greater than 5.5 ATA.

The distribution of symptoms among various anatomical locations for different kinds of pressure exposures is shown in Table 4. Interestingly, the locations of symptoms reported during Navy saturation dives is closely aligned with the locations reported for tunnel workers. The concentration of musculoskeletal pain in the lower extremities seems to be associated with the length of the exposure. The knee may be one of the longest tissues in the body, or perhaps it is just a collection point for free gas. In either event, future saturation decompression schedules may have to be designed specifically for this anatomical location.

In addition to these general observations concerning decompression sickness, the NEDU data were also subjected to a correlational analysis (Table 5). The relationship between nine diver and dive-profile variables and three dependent variables (depth of reported onset of symptoms, depth of reported relief of symptoms, and time to reported relief of symptoms) were calculated.

The largest correlation coefficient in Table 5 is the 0.96 between depth of reported onset and depth of reported relief. If this correlation had not been high, it would have cast doubts upon

TABLE 3

Type of decompression sickness tabulated by location of onset of symptoms

Decompression sickness category	Onset of symptoms under pressure	Onset of symptoms after surfacing
Type I	24	3
Type II	0	0

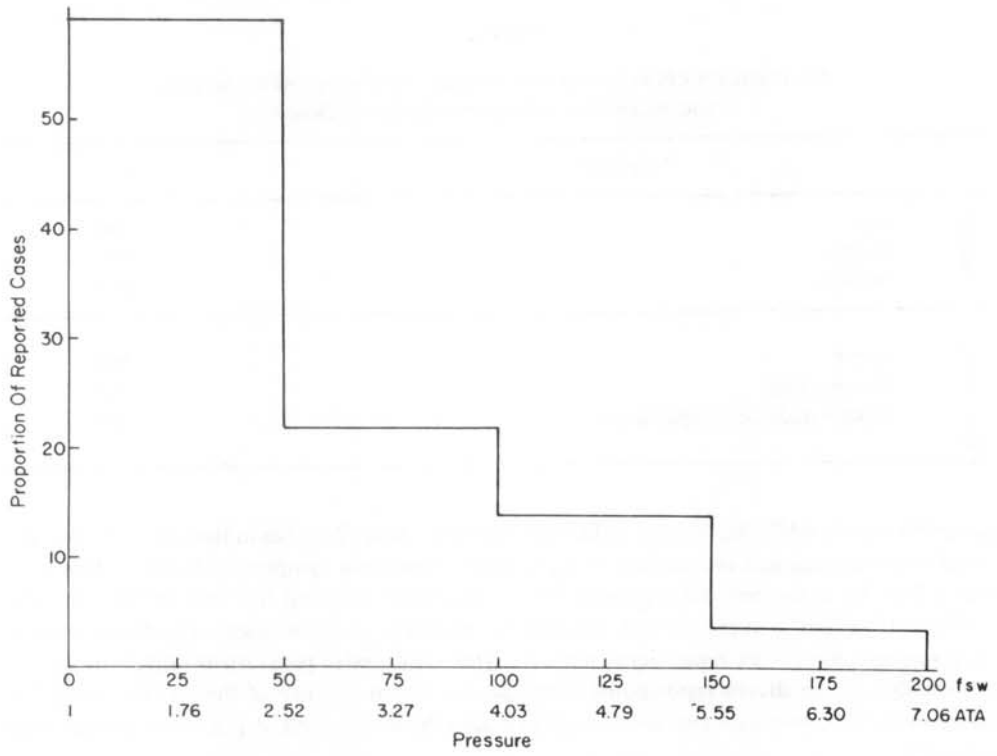

Fig. 2. Proportion of decompression sickness cases occurring at various depths following a saturation exposure.

TABLE 4

Percentage distribution of decompression sickness symptoms

Location of symptoms	Subsaturation divers*	Tunnel workers†	Saturation divers‡
Upper extremities	63	20	4
Lower extremities	26	70	96
Other	11	10	0

*Average derived from Rivera (1964), Behnke (1947), and Duffner, Van der Aue, and Behnke (1946).
†Average derived from Erdman (1907), Keays (1909), Bornstein (1912), Levy (1922), and Golding, Griffiths, Hempleman, Paton, and Walder (1960).
‡Berghage (present study).

the entire results; it is logical that the deeper one experiences decompression symptoms the deeper one will be when relief is obtained. This is true even if no recompression is used.

According to Boyle's Law, the pressure change necessary to reduce bubble volume is depth-dependent. A greater pressure change is required to compress a bubble formed at 4 ATA than is required to compress a bubble formed at the surface. To obtain equivalent bubble resolution regardless of the depth of onset, a constant ratio (P_1:P_2) between treatment depth

TABLE 5

Correlations between diver and dive-profile variables* and measures of decompression and treatment adequacy†

	Variables	Depth of onset	Depth of relief	Time to relief
Diver and dive-profile variables	Depth of dive	0.30	0.41	0.29
	Bottom time	−0.17	−0.17	−0.14
	Exposure index $\frac{D \times BT}{100}$	−0.02	0.22	0.17
	Diver age	−0.22	−0.26	−0.16
	Diver height	0.36	0.33	0.18
	Diver weight	0.39	0.39	0.08
	Weight-to-height ratio	0.35	0.37	0.04
	Excursion dives (yes-1, no-0)	−0.19	−0.24	−0.27
	Recompression ratio	−0.91	−0.52	−0.70
Decompression and treatment adequacy	Depth of reported onset of symptoms	--	0.96	0.38
	Depth of reported relief of symptoms	0.96	--	0.50
	Time to reported relief of symptoms	0.38	0.50	--

D is depth and BT is bottom time.
($r = .60, P < .001$)
*$n = 9$
†$n = 3$

(ATA) and depth of onset (ATA) must be maintained. If a constant ratio is maintained, there should be a zero correlation between the ratio and depth of onset of symptoms. The data provided a correlation coefficient of −0.91, which indicates that the deeper the depth of reported onset of symptoms, the smaller the recompression ratio that has been used. A least-squares regression curve has been fitted to these data (Fig. 3). In light of Boyle's Law, it is interesting that existing procedures run completely contrary to the physical concept.

A statistically significant correlation between recompression ratio and time to reported relief of symptoms is also shown in Table 5. Relief of symptoms here refers to the first reported indication of improvement. The reason for this distinction will become clear in a moment. The high negative correlation (−0.70) between recompression ratio and time to relief indicates that inadequate recompression results in a protracted treatment period (Fig. 4). Because there is also a high negative correlation between depth of reported onset of symptoms and the treatment-recompression ratio, it is possible that the protracted treatment period results from depth of onset rather than the recompression ratio. The partial correlation is a statistical technique for holding the effects of one variable constant while two other variables are evaluated. With the effects of depth of onset of symptoms partialled out, the correlation between treatment recompression ratio and time to reported relief of symptoms is increased to

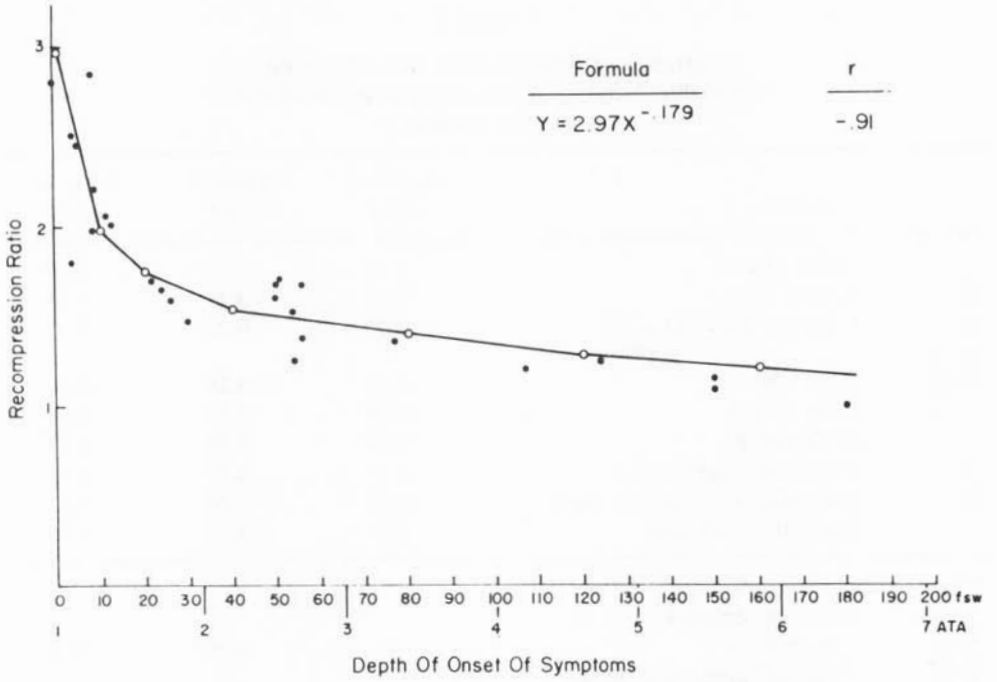

Fig. 3. Relationship between depth of reported onset of symptoms and the recompression ratio used in the treatment.

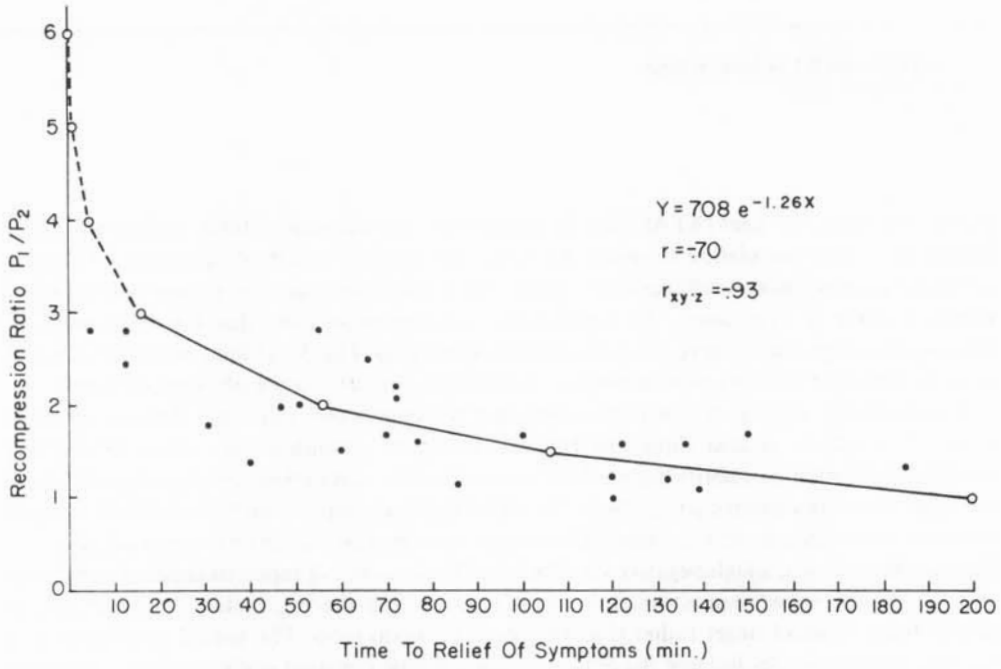

Fig. 4. Relationship between recompression ratio used and the time to reported relief of symptoms. The dashed line represents an extension of the best-fit curve beyond existing data points.

−0.93. The result strongly supports the idea that unless adequate recompression is used, the time required for treatment will be greatly extended.

The distinction made earlier between time to full relief of symptoms and time to the first indication of relief or partial relief is necessary because symptoms occurring during saturation decompressions rarely obtain the full and complete relief usually associated with recompression therapy initiated from the surface (1 ATA). Even after a full 2 hours of breathing an increased partial pressure of oxygen at an increased pressure, only about 35% of the patients obtained "full relief." This is in striking contrast to the 96% relief of symptoms usually obtained when recompression is initiated from the surface (Fig. 5).

DISCUSSION

The majority of the decompression sickness studies, including this one, have been descriptive in nature. They have described in fine detail the symptomatology that occurs and sometimes they have suggested therapeutic procedures. Very little attention, however, has been directed toward predicting the likelihood of the occurrence of decompression sickness. Once a phenomenon is adequately described, the next step in scientific procedure is prediction and control. Unless we can predict a phenomenon, we really do not understand it. The inability to predict which divers are likely to be stricken with decompression sickness is partially due to the probabilistic nature of the phenomenon. The failure of the U.S. Navy Safety Center data to provide even a hint of which divers might be stricken is an indication that we do not have an

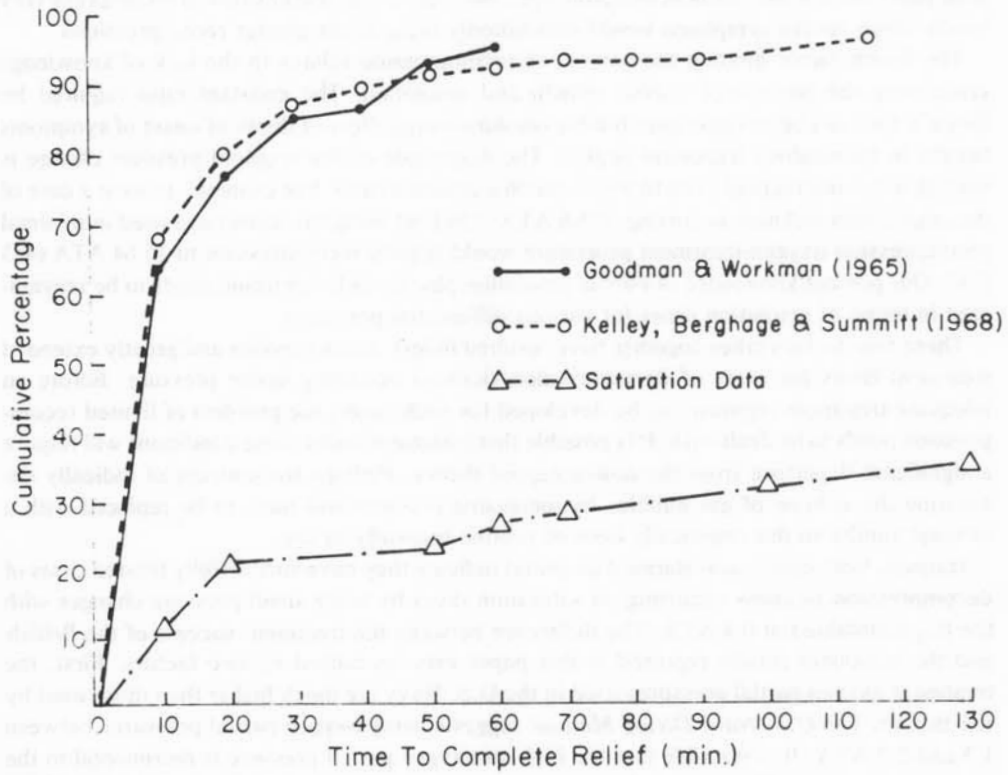

Fig. 5. Proportion of decompression sickness cases obtaining relief after various time intervals.

adequate understanding of the phenomenon and, consequently, have only limited control of the outcome of a saturation dive.

The apparent failure of the existing therapeutic procedures in saturation diving takes place largely because most of the cases occur while the divers are still under pressure. Recompression in these cases is usually limited to 1 atm or less; consequently, very little bubble-volume change can be expected. Four factors are responsible for this limited use of recompression. First, several attempts at recompressing patients stricken during saturation dives have resulted in an increase in the severity of the joint or musculoskeletal pain; this creates for the treating physician a dilemma of not being able to decompress or recompress. The only alternative has been to remain at the depth of onset and administer to the patient high partial pressures of oxygen.

The second factor that limits the extent of recompression is the total confinement time usually involved. Saturation dives are not normally made unless the depth of the dive is very deep or the dive requires an extensive period of bottom time. In either case, by the time the divers decompress to within 3 atm of the surface, they have been confined for extended periods of time. Any recompression at this point will extend this confinement and bring extensive peer pressure (either real or imaginary) on the stricken individual, the attending physician, and the topside supervisor. Because of this peer pressure, the tendency is to limit the recompression as much as possible.

The third factor that limits the amount of recompression is the clinical severity of the symptoms. As indicated earlier, the U.S. Navy is yet to have a Type II decompression sickness case during a standard saturation decompression. As long as musculoskeletal and joint pains are the only clinical symptoms present, physicians are hesitant to recompress very much. More severe symptoms would undoubtedly bring about greater recompressions.

The fourth factor limiting the amount of recompression relates to the lack of knowledge concerning the physics of bubble growth and resolution. The constant ratio required by Boyle's Law to obtain equivalent bubble resolution regardless of depth of onset of symptoms results in tremendous treatment depths. The magnitude of the required pressure change is such that it is impractical even to try to obtain a constant ratio. For example, to treat a case of decompression sickness occurring at 5.6 ATA (150 fsw) using the same ratio used in minimal recompression oxygen-treatment procedure would require recompression to 15.64 ATA (483 fsw). Our present knowledge of bubble-resolution physics in living tissue needs to be reevaluated in terms of resolution times for various differential pressures.

These four factors taken together have resulted in less recompression and greatly extended treatment times for cases of decompression sickness occurring under pressure. Before an adequate treatment regimen can be developed for such cases, the problem of limited recompression needs to be dealt with. It is possible that treatment under these conditions will require a significant departure from the now-accepted theory. Perhaps the concept of radically decreasing the volume of gas bubbles by increasing pressure will have to be replaced with a concept similar to the empirically derived routine presently in use.

Hanson, Vorosmarti, and Barnard (*in press*) indicate they have successfully treated cases of decompression sickness occurring on saturation dives by using small pressure changes with the Po_2 maintained at 0.4 ATA. The difference between the treatment success of the British and the treatment results reported in this paper may be caused by two factors. First, the treatment oxygen partial pressures used in the U.S. Navy are much higher than those used by the British. The *U.S. Navy Diving Manual* suggests using oxygen partial pressures between 1.5 and 2.5 ATA. It is possible that too high an oxygen partial pressure is detrimental to the treatment outcome. The results of Berghage, Conda, and Armstrong (1973) suggest that there may be an optimum oxygen partial pressure for each treatment pressure level.

A second major difference between the British procedure and that used in the U.S. is not related to the treatment but to the saturation decompression producing the symptoms. The British use a stage method with significant drops in pressure as opposed to the constant-bleed method used in the U.S. The periodic reductions in pressure associated with the British stage method provides discrimination points for assessing the condition of the divers. With the present U.S. method, a bubble may exist for a long time before its presence is detected in the form of clinical symptoms. The prolonged time between the formation of the bubble and the therapeutic recompression may result in tissue damage, which in turn complicates and extends the treatment process. The treatment problems described in this report could possibly be reduced if a stage decompression was used and the time between bubble formation and therapeutic recompression decreased.

In implementing a stage decompression for saturation dives, it might be worth considering variable step sizes depending upon the pressure level. The work of Berghage, Gomez, Roa, and Everson (1976) suggests that the magnitude of a safe pressure reduction decreases as one gets close to the surface. Therefore, rather than having consistent step sizes throughout the decompression, it may be better to vary the step size depending on the pressure level.

Experimental data should be gathered on the time course of symptom resolution with various oxygen partial pressures and pressure changes. This type of information is needed before an adequate evaluation can be made. Consideration should also be given to altering the saturation decompression from a continuous ascent to a variable-staged profile.

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A note of thanks should also be extended to Captain W. H. Spaur, of the U.S. Navy Experimental Diving Unit, for his critical review.—*Manuscript received for publication January 1976; revised manuscript received June 1976.*

Berghage, T. E. 1976. Maladie de décompression au cours de plongées à saturation. Undersea Biomed. Res. 3(4):387–398.—Les auteurs ont analysé les données de la Marine américaine sur les plongées à saturation pour évaluer l'éfficacité thérapeutique des traitements de la maladie de décompression et identifier les factèurs déclenchants de l'étiologie et du traitement de la maladie de décompression pendant des plongèes à saturation. Dans aucun cas de maladie de décompression survenue au cours d'une plongèe à saturation on n'a noté des symptômes plus graves que des douleurs articulaires ou musculosquelettiques; dans 96 p. 100 des cas il s'agissait des genoux du plongeur. Chez 89 p. 100 des patients les symptômes se sontmanifestés pendant que le plongeur était toujours comprimé. Un traitement de récompression n'a apporte de soulagement qu'à 35 p. 100 des patients; chez les autres il restait après la thérapie et la décompression suivante des douleurs résiduelles qui ont diminué au cours des semaines suivantes. L'éfficacité de la récompression est en rapport inverse à la profondeur à laquelle ont commencé les symptômes. Le temps nécessaire pour obtenir un effet thérapeutique même partiel dépend directement du rapport de décompression choisi. Quatre explications sont suggérées pour la thérapie de récompression limitée trouvée dans les plongées á saturation: augmentation de la douleur musculosquelettique par la récompression; influence des camarades pour éviter un prolongement de la période de confinement dans la chambre; manque de symptomatologie neurologique grave; profondeurs très grandes nécessaires pour obtenir un rapport de récompression raisonable. L'auteur suggère aussi que pour les procédés thérapeutiques de l'avenir il faudra abandonner l'idée acceptée de diminuer le volume des bulles de gaz inerte en augmentant la pression.

maladie de décompression
plongèes à saturation
procédés thérapeutiques de récompression

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