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# HUMAN DECOMPRESSION TRIAL IN NITROGEN-OXYGEN DIVING

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and
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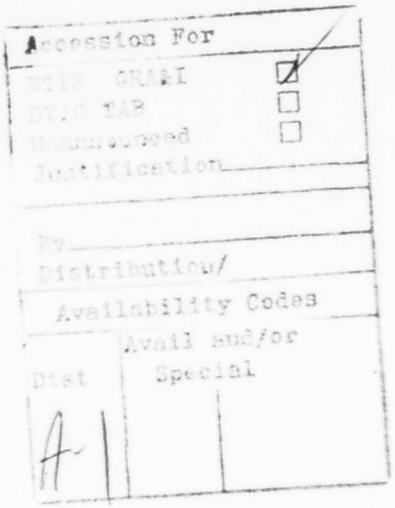
Kurt Sorensen — 5 Feb 87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A trial was performed to assess the relative risks of oxygen and nitrogen in human no-decompression dives. Dives (n=477) were performed with subjects breathing mixtures with low or high oxygen partial pressures. Dives were 30, 60, or 240 min in duration to various depths chosen by a sequential dose-response format. Despite exceeding the currently accepted safe limits by more than 20%, the incidence of demonstrable DCS was under 3%. Another 3% had ambiguous symptoms. An increase in DCS risk with higher $N_2$ pressure could not be strongly demonstrated. Oxygen had less than half of the DCS risk associated with nitrogen.

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INTRODUCTION

Human decompression sickness (DCS) results from a series of mechanisms sufficiently unknown as to prevent effective prediction of its occurrence. "Safe decompression" procedures are calculated by various means, but a final testing phase is always required. Typically, the first calculations produce an unsafe set of procedures, and revision and re-testing is the historic norm. Any information to increase the accuracy of prediction would save people and resources.

To calculate an acceptable decompression, numerous assumptions are made. With a single recent exception (Thalmann, 1984), a standard assumption is that only the inert gas needs to be considered. All inspired oxygen is ignored. A frequent application of this assumption is the Equivalent Air Depth (EAD) calculation used to consider any dive on a $N_2$-$O_2$ mixture as equivalent for decompression purposes to an air dive with the same inspired $N_2$ partial pressure. This assumption may be incorrect and unsafe, because there is no chemical reason why $O_2$ cannot be a component of a bubble in tissue (Forest and Ward, 1977). On the other hand, the assumption may be incorrect and safe, because higher levels of oxygen decrease the blood flow, and thus the rate of inert gas uptake, in at least some tissue beds (Carrier et al. 1964). Neither line of reasoning can be pursued much further, because the actual composition (and in fact the occurrence) of bubbles is unknown, as are the identification and characteristics of the tissue beds responsible for DCS.

Some experimental approaches to address this assumption have been reported. Donald (1955) demonstrated that animals can suffer a form of DCS when only $O_2$ is breathed. Berghage (1979) accumulated evidence that $O_2$ can add to DCS risk, but not in a simple way. Yount and Lally (1980) reinterpreted Berghage's data with allowance for direct solubility and

hemoglobin binding. A recent study of Lillo (1986) indicated a substantial contribution of $O_2$ to DCS risk in near-fatal DCS in small animals. The only direct study with humans appeared to show a slight contribution of $O_2$, but was terminated short of statistically significant results (Logan, 1960). An indirect implication of $O_2$ involvement in the decompression process is the failure of extrapolation from air to mixed $N_2$-$O_2$ decompression tables in a recent EDU experimental series (Thalmann, 1984).

This report describes a trial of 477 human exposures intended to establish the magnitude of any $O_2$ contribution to DCS risk under conditions of $N_2$-$O_2$ diving with immediate return to the surface ("no-decompression dives"). The trial was sequential in design, with the diving depths established by recent history of the trial rather than in advance. The raw data were then to be in the form of DCS dose-response observations that could be analyzed by appropriate models using maximum likelihood estimation (Weathersby, 1984). Statistical models of DCS incidence depend on dose-response formulations, and this trial was expected to provide an excellent opportunity to estimate the shape of the dose-response function.

## METHODS

### Experimental design

The experiment was intended to simulate operational diving with respect to conditions of water immersion, cold, exercise, and breathing gear; but to closely control gas composition, depth and time. Dives were divided into 6 "series" which specified a bottom time (30, 60, 240 min), and a gas mixture (high or low $O_2$) (Table 1). The starting depth for each series was chosen to be about 8% shallower and presumably safer than the U.S. Navy Diving Manual no-decompression limits on compressed air (U.S. Navy, 1985), when the assumption of no $O_2$ influence is applied. The entry of EAD (Equivalent Air

TABLE 1

| Series | Bottom Time (min) | $O_2$ | Depth (fsw) | Equivalent Air Depth (fsw) | $pO_2$ (ATA) |
|---|---|---|---|---|---|
| 1 | 30 | 10% | 66- 91 | 80-108 | .30 - .38 |
| 2 | 30 | 30% | 95-130 | 80-111 | 1.16 - 1.48 |
| 3 | 60 | 10% | 43- 59 | 53- 72 | .23 - .28 |
| 4 | 60 | 35% | 72- 96 | 53- 73 | 1.11 - 1.37 |
| 5 | 240 | 12% | 25- 38 | 32- 46 | .21 - .26 |
| 6 | 240 | 40% | 50- 74 | 30- 48 | 1.01 - 1.30 |

Depth) in Table 1 gives the depth equivalent for the same $pN_2$ with compressed air. For example, the Series 1 gas mixture at a depth of 66 fsw has the same nitrogen partial pressure as air at a depth of 80 fsw. The U.S. Navy no-decompression air limits are 90 fsw for 30 min, 60 fsw for 60 min, and 35 fsw for 310 min. The oxygen fraction was selected to maintain at least normoxia ($pO_2$ > 0.21 ATA) for Series 1,3,5 and to be below substantial risk of $O_2$ toxicity for Series 2,4,6.

Rules for depth change within each Series were as follows:

1. No DCS cases in 10 exposures at this depth, add 4% in pressure
2. 1 DCS case in 20 exposures at this depth, add 4% in pressure
3. 2 DCS cases in any number of exposures, or 1 serious case, subtract 2% in pressure.

Each subject was limited to 2 exposures in each series, for a total of up to 12 exposures. This limitation was a compromise between extensively studying a few people, and trying to get a very large population to have a few exposures each. In the only large controlled population study of decompression, Gray et al. (1947) found that DCS tended to be more random than reproducible in the same individual. The order of dives (i.e., which series next) followed a table of random numbers that was stratified to achieve equal numbers of exposures in each series at multiples of 72 exposures.

Subjects

A total of 61 active duty U.S. Navy divers volunteered after the study was approved by the local Committee for Protection of Human Subjects. With 3 exceptions, all were stationed at this command. Subjects participated in 1 to 12 successful exposures, with an average of 8 exposures per subject. Physical attributes of the subjects are included in Appendix 1. Volunteers for the trial were solicited from all male divers under age 40. Prospective subjects

were briefed individually and in groups on the purpose and design of the study before informed consent was elicited. Approximately 85% of those who were eligible volunteered. For acceptance as a subject, no chronic or acute medical condition that would cloud the diagnosis of DCS was allowed. No other blanket disqualifications were instituted in order to approach a representative sample of the total Navy diving population. Medical problems noted in subjects accepted into the study included: gas pockets in the pelvis (Subject 61) (Hart et al., 1986), recent arthroscopic knee surgery (Subject 23), twice ruptured tympamic membrane (Subject 17), history of fracture with internal fixation (Subjects 37 and 50), and history of low back pain (Subjects 15, 36, and 41). These conditions did not result in permanent disqualification from diving, and in fact, none of these problems recurred following any experimental dive.

Qualification standards for a particular scheduled dive were more severe. To avoid mistaking muscle strain with DCS, a subject was temporarily excused if he had participated in physical exercise on the scheduled day unless it was much less than his daily regimen. He was also excused if he had taken any systemic drug other than antibiotics. Aspirin and oral decongestants were not permitted. Divers were also excused for respiratory illnesses with significant sinus or joint involvement, less than average sleep the night before, or consumption of over 2 oz. of alcohol during the preceding 24 h. No diver was scheduled if he had been exposed to increased atmospheric pressure for any reason in the preceding 7 days. This last requirement attempted to avoid any acclimatization effect that is demonstrable in some pressure exposures (Golding et al. 1960) and suspected in others (Thalmann, 1985). If a diver suffered DCS, he was disqualified for 2 weeks in cases of simple

pain-only symptoms, and for 4 weeks after more serious cases. Only 1 subject declined to re-enter the trial after suffering DCS.

Facilities

The NMRI chamber with wet-pot was used for all exposures. Water was kept between 69 and 72 °F, except for the long dives when a rise to 76 °F was allowed for divers with significant susceptibility to cold. Divers were clothed in 1/4 inch wet suits, but some used an additional partial wet suit if they expected to be very cold. A target of less than 1 °C core temperature drop was established. The low risk of severe hypothermia was insufficient to justify the discomfort of continuous temperature monitoring. However all subjects in 4 h dives had a rectal temperature measurement immediately before and after the dive. Most measurements showed a drop of a few tenths of a degree, but more than 10 subjects actually had a temperature rise, and in 7 cases a drop of 1.0-1.5 °C was found. All subjects used the U.S. Navy Mk-1 Diver's Mask for gas supply and communications (U.S. Navy, 1985).

While at depth, subjects exercised using a locally built sled ergometer (Moritz and Mints, 1972), that allowed a standard work rate in a sitting position. The sled had a set of spring loaded pedals for each foot that was extended by the subject from a length of 28 to 44 inches (spring tension from 6.6 to 13.5 pounds) between 2 sets of magnetic switches. A timing light was placed near each subject to help him remain at 50 repetitions (each leg through a full extension-relaxation cycle) per minute. The work was performed on a schedule of 5-min work, 3-min rest, with an additional 10 min rest after each hour on the long dives. In preliminary measurements on 2 subjects, the exercise was found to produce an oxygen consumption of 1 - 1.5 l/min.

Gas mixtures were prepared from pure $O_2$ and $N_2$. Mixtures were made in large batches (60-180 cu.ft. at 4000 psig) and the analysis of each batch fell

within 0.2% $O_2$ of the target value by both paramagnetic oxygen analysis and mass spectrometry. Composition of the breathing gas was also verified before and during each dive with a paramagnetic oxygen analyzer.

Procedure

After equipment check-out, diver-subjects descended 9 ft into the wet pot while breathing compressed air from the Mk-1 emergency gas supply. Then the chamber above was compressed with air at a specified rate of 75 ft/min. Compression was stopped for 10 sec at 30 fsw for breathing gas to be switched to the experimental mixture, then compression resumed. If divers had difficulty in equalizing pressure in ears or sinuses, compression was slowed or reversed. Actual total descent times for each dive are included in the chronological record, Appendix 2. If the final pressure could not be reached within 2 min of the scheduled descent time (or 1 min in the case of 30 min dives), the dive was aborted. Time at depth was spent in the exercise described above.

Control of the depth was tight. Depth was defined as the air pressure in the chamber above the wet pot plus the water depth at a mid-chest level on the diver. A high precision differential digital pressure gauge (Mensor Corp, San Marcos TX, Serial 2237 with overall accuracy specification of 0.04%) was used to control depth via manually operated supply and exhaust valves. Variations in barometric pressure were ignored. A pen plotter was attached to the gauge auxiliary output and monitored through the dive. Deviation of 1 fsw for 30 sec, or any other cumulative combination of pressure excursions adding up to that value was reason to abort the dive. At the end of the dive (30, 60, or 240 min after the diver first left the surface), a large valve was opened for a target rate of 60 ft/min. Actual rates were within 10% of the target rate except for shallow depths. Travel from 10 ft to the surface took 18-20

sec, as measured on a separate recorder used on every dive. Again, a 10 sec hold at 30 fsw was used to switch the diver's gas back to compressed air. The Series 5 dives, being shallow, had the change of gas occur at the full dive depth; otherwise the procedures were identical.

After leaving the chamber, the divers were interviewed briefly by a medical officer, and examined in more detail 2 h after the dive. Subjects were strongly encouraged to report all symptoms of any kind for 18 h, and were generally interviewed by a medical officer the following morning. From the list of symptoms elicited (Appendix 3), and from numerous remarks made by the subjects, the record keeping appears more extensive than in most other decompression studies.

For about 2/3 of the exposures, a Doppler ultrasonic device was used. Tape recordings were made of pre-dive, 15 min, 1 h, and 2 h post-dive sounds in the precordial and left and right subclavian positions (Eatock and Nishi, 1986). The recordings were made with only a numerical identification of the actual dive, and neither the subject nor the attending medical officer heard the tape on the day of the dive. Analysis of the doppler data will be reported separately.

## RESULTS

### Symptoms

A total of 477 exposures were completed according to all specifications. Another 47 exposures were aborted during the dive for violation of the protocol. The most frequent reason to abort was the inability of a diver to clear his ears fast enough to reach bottom depth within the short allowable time. A chronological record of all successful dives is provided in Appendix 2.

Diagnostic outcome did not fall into the clean categories of DCS or no

DCS. The spectrum of symptoms were forced into final categories of definite DCS, no DCS, and marginal symptoms. Some 54 exposures were followed by a report of symptoms of one kind or another. Appendix 3 presents a brief report of each case, indentified by numbers in parenthesis in Appendix 2. Of the 54 incidents, 13 were treated on a U.S. Navy hyperbaric oxygen treatment table. As seen in the Appendix 3, most symptoms were rather mild and gradual in onset, and relief was prompt with recompression. No subject had any measurable or subjective symptom persisting for as long as a week after the dive. Four cases had no prompt relief of the ambiguous presenting symptom and were concluded on that basis to not represent DCS. Two of these unresponding cases treated within 4 h of the dive (cases 54 and 55 in Appendix 3) were then considered as interrupted post-dive observations and not included with the 477 exposures in Appendix 2 as final data. The rationale is that the unwarranted treatment masked any actual DCS that might have occurred later. The other 2 unresponsive cases (cases 24 and 47) were treated 18-20 h post-dive (assumed long enough to develop any sympton) and were declared to be acceptable uneventful exposures.

Upon reviewing the records after the entire experiment, a diagnosis of DCS was made in an additional 3 cases (13,20,38) based on the symptoms reported orally more than a day after a dive, but not treated with recompression. At the time of the review, another 18 cases were declared as having marginal decompression symptoms (frequently called "niggles") that did not warrant treatment. This category was defined as mild joint pain or discomfort that lasted for 2-60 min, or for a shorter period but on more than 1 occasion that day, or occurred in more than 1 site. These cases were not treated because the symptoms did not persist for long enough to better evaluate or to observe a possible improvement with treatment. The marginal

TABLE 2

Summary of Outcome

Series 1 (10% $O_2$) - 30 min -

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 66 | 2 | 0 | 0 |
| 67 | 10 | 0 | 0 |
| 71 | 21 | 1 | 2 |
| 75 | 9 | 0 | 1 |
| 79 | 10 | 0 | 0 |
| 83 | 11 | 0 | 0 |
| 87 | 10 | 0 | 0 |
| 91 | 3 | 0 | 0 |

Series 2 (30% $O_2$)

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 95 | 10 | 0 | 0 |
| 100 | 10 | 0 | 0 |
| 105 | 10 | 0 | 1 |
| 110 | 10 | 0 | 0 |
| 115 | 10 | 0 | 0 |
| 120 | 10 | 0 | 0 |
| 125 | 11 | 1 | 0 |
| 130 | 7 | 1 | 0 |

Series 3 (10% $O_2$) - 60 min -

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 43 | 10 | 0 | 0 |
| 44 | 10 | 0 | 0 |
| 46 | 10 | 2 | 0 |
| 47 | 11 | 0 | 0 |
| 50 | 10 | 0 | 0 |
| 53 | 10 | 0 | 0 |
| 56 | 10 | 0 | 1 |
| 59 | 8 | 0 | 1 |

Series 4 (35% $O_2$)

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 72 | 12 | 0 | 1 |
| 76 | 10 | 0 | 0 |
| 80 | 10 | 0 | 2 |
| 84 | 10 | 0 | 0 |
| 88 | 19 | 1 | 1 |
| 92 | 9 | 0 | 0 |
| 96 | 12 | 1 | 0 |

Series 5 (12% $O_2$) - 240 min -

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 25 | 10 | 0 | 0 |
| 27 | 10 | 0 | 0 |
| 29 | 10 | 1 | 0 |
| 31 | 10 | 0 | 0 |
| 33 | 11 | 1 | 0 |
| 34 | 10 | 0 | 2 |
| 35 | 7 | 1 | 1 |
| 36 | 11 | 0 | 1 |
| 38 | 2 | 0 | 0 |

Series 6 (40% $O_2$)

| Depth | Dives | DCS | Marginal |
|---|---|---|---|
| 50 | 20 | 1 | 0 |
| 54 | 10 | 0 | 0 |
| 58 | 10 | 0 | 1 |
| 62 | 10 | 0 | 0 |
| 66 | 10 | 0 | 0 |
| 70 | 10 | 0 | 1 |
| 74 | 11 | 0 | 2 |

477 Total Dives
11 Total DCS (2.3%)
18 Total Marginal (3.8%)

cases also include 4 cases of simple "skin bends" that presented as a mottled rash on the trunk.

A summary of outcome by exposure condition is presented in Table 2. Most series covered a 30% span in absolute pressure. The data within each series are striking in their variability; no clear increase in DCS incidence with exposure depth is evident. That observation clearly contradicts the common view that a threshold exists beyond which safe diving suddenly becomes hazardous. The variability is also evident across series: no oxygen or time grouping has a predominance of the symptoms.

Variability was evident in the response of individuals as well. No subject had DCS twice with the same exposure. In the 4 instances where subjects had 2 dives in a series, one of which resulted in DCS, his other dive was uneventful. In 3 of these 4 cases, the other exposure was to the same or greater depth as the problem dive.

Although not specifically part of the experimental design, questions of individual risk factors can be asked. Subjects covered the range expected of a healthy military population which omits the large differences found in a more general group. Two frequently cited variables concern the diver's age and physique. In the study population, average age was 29.4 years; average height was 70.4 inches; average weight was 178.2 pounds. Half of the subjects were measured for body fat by hydrostatic weighing, and they averaged 18% fat. Table 3 compares the DCS outcome by age, weight, and % body fat for the lower and upper half of the group by each measure. Columns labelled "Expected" used the overall incidence of DCS, or DCS + marginal, symptoms for the entire study, consistent with the null hypothesis that all exposures had equal risk. For example, the 236 dives performed by men under age 30, when multiplied by the overall incidence of 29 DCS + marginal in 477 dives, would be expected to

TABLE 3

Age and Physique Effects on Outcome

| | Total | | Expected | | Observed | |
|---|---|---|---|---|---|---|
| | Divers | Dives | DCS | DCS + Marginal | DCS | DCS + Marginal |
| AGE 20-29 | 31 | 236 | 5.4 | 14.3 | 5 | 13 |
| AGE 30-40 | 30 | 241 | 5.6 | 14.7 | 6 | 16 |
| WEIGHT <178 | 31 | 240 | 5.5 | 14.6 | 5 | 12 |
| WEIGHT 179+ | 30 | 237 | 5.5 | 14.4 | 6 | 17 |
| FAT < 17% | 15 | 160 | 3.7 | 9.7 | 1 | 5 |
| FAT 17% + | 15 | 140 | 3.2 | 11.7 | 5 | 13 |

result in 14.3 total dives with any symptom. The low total incidence produces fairly weak test statistics, and no effect of age, weight or body fat are visible. (Chi-square tests on the results are all consistent with no effect, though the % fat comparison shows a possible difference at $0.10 < p < 0.05$).

Analysis

Several questions need to be addressed by analysis: what is the shape of the decompression dose-response function? What effect does oxygen have? How do these results compare to other work?

Inspection of the raw data in Table 2 suggests that the dose-response function is not very steep, that is, a few feet deeper in the dive depth does not convert a very safe dive to one that is very hazardous. One indication of how "softly" the DCS risk increased with depth is an overall comparison of dives to the current U.S. Navy no-decompression limits using the EAD assumption. Dives allowed by present rules had 2.1% DCS and 5.4% DCS + marginals out of 186 exposures. On the 391 dives beyond the limit, there was a 2.4% incidence of DCS and 6.5% rate of DCS + Marginals. The "limits" clearly do not separate regions of greatly different DCS risk.

To better define the dose-response and oxygen effect a specific probabilistic model is needed. A number of models capable of dealing with fairly complex dive profiles were examined in a recent report (Weathersby et al., 1985), but the simple design of the present data allows simpler models. First, we define the probability of DCS to depend on an empirical risk model:

$$P(DCS) = 1.0 - \exp(-R) \qquad [1]$$

The R in Eqn. 1 is the decompression risk that results from a particular depth, time, and gas mixture. Note that a small value of R leads to only a small chance of DCS while a large value of R makes the probability of bends approach 1.0 (i.e., 100%). In principal, R could also include measures of

individual susceptibility (e.g., % body fat) were such measures available. For now we use only the following expression where R is "physiologically" formulated with a "tissue supersaturation":

$$R = [k1\ (pN_2 + kO_2 \cdot pO_2 - 1\ ATA)]^n \qquad [2]$$

The $pN_2$ and $pO_2$ are computed nitrogen and oxygen partial pressures in tissue immediately before decompression, which when decreased by the post-decompression ambient pressure of 1 ATA represent the calculated gas supersaturation. The parameter $kO_2$ allows for oxygen to have less (or more) of an impact on risk compared to nitrogen; parameter n allows for greater or lesser steepness of the dose-response curve compared to a simple exponential (with large n's producing sharper sigmoid curves); and parameter k1 has units of $ATA^{-1}$ to establish the pressure-probability scale conversion.

It is still necessary to calculate the tissue tensions of nitrogen and oxygen. Oxygen is assumed to achieve a tissue steady state value very quickly in comparison to the experimental time (30 min or more). Tissue levels of oxygen are subject to different metabolic and solubility effects than nitrogen, but for simplicity in the present analysis we will use inspired $pO_2$. Tissue nitrogen to a first approximation is assumed to respond exponentially to a change in ambient pressure.

$$pN_2 = .79\ ATA + (pIN_2B - .79) \cdot [1.0 - \exp(-T/TC)] \qquad [3]$$

Eqn. 3 states that some area of the body has been equilibrated with atmospheric $N_2$ (79% of 1 ATA) then increases over the dive bottom time, T, towards the bottom depth inspired nitrogen pressure, $pIN_2B$, with a characteristic time constant, TC.

The unknown parameters in Eqn. 1-3 which must be estimated by the data are k1, $kO_2$, n, and TC. The optimizing procedure of maximum likelihood was used to obtain the parameters. For that procedure the likelihood function

(Weathersby et al., 1984), which is the product over all dives of the probability of the event actually happening, is maximized. The probability is determined by the model, such as Eqns. 1-3, which relate details of the dive to p(DCS) if symptoms were observed, or to p(no DCS) = 1.0-p(DCS) is the dive was uneventful. Estimation was performed by a nonlinear least-squares Marquardt algorithm modified for maximum likelihood as previously described (Weathersby et al., 1984). The large number of marginal cases has a significant impact on the data structure. As before (Weathersby et al., 1984), we have run parallel analyses on 3 possible interpretations of marginal cases: all considered as DCS, none considered as DCS, and each marginal considered as 1/2 case of DCS and 1/2 safe exposure.

Results of the estimation from all 477 exposures are presented in Table 4 for the 3 diagnostic possibilities. Numbers in parentheses are approximate 1 standard error (SE) uncertainties in estimated parameters.

For each set of entries in Table 4, the first entry is a "null model" which denies any effect of pressure or time and considers each dive to have equal chance of DCS. The maximum log likelihood, LL, for this model can be considered a lower bound for more realistic models. The second entry in each section uses Eqns. 1-3 with mono-exponential gas exchange kinetics, no oxygen effect, and the dose-response exponent, n, fixed at 1.0. In each case, the fit to data is slightly better than the null model, but not outside the improvement (increase in likelihood) that may occur simply by chance with the additional parameter. The time constants are in the range of 75 to 95 min.

Entries are not listed in Table 4 for the effect of varying exponent, n. It proved difficult by normal estimation procedures to deal with this parameter. However the allowable range covered 1/4 to 4.0 in all cases without achieving a statistically significant difference in maximum LL from

TABLE 4

Probabilistic Model Analysis of Data

| Models and Parameters | LL |
|---|---|
| **DCS Cases only** | |
| None - null model, P(DCS) = 0.02306 | -52.338 |
| TC = 74.7(57), $kO_2$ = 0.0F, n = 1.0F, k1 = 3.84(2.3)x$10^{-2}$ | -52.150 |
| TC = 56.7(52), $kO_2$ = -.1(.3), n = 1.0F, k1 = 3.95(2.0)x$10^{-2}$ | -52.090 |
| NMRI Report 85-16, Model 5, Parameters ABCD | -61.852 |
| **DCS Cases + 1/2 Marginal Cases** | |
| None - null model, P(DCS) = 0.0419 | -83.010 |
| TC = 88.5(54), $kO_2$ = 0.0F, n = 1.0F, k1 = 8.0(4.3)x$10^{-2}$ | -82.114 |
| TC = 67.7(53), $kO_2$ = -.09(.25), n = 1.0F, k1 = 8.3(3.6)x$10^{-2}$ | -82.031 |
| NMRI Report 85-16, Model 5, Parameters ABCD | -89.632 |
| **DCS + All Marginal Cases** | |
| None - null model, P(DCS) = 0.0608 | -109.306 |
| TC = 94.0(40), $kO_2$ = 0.0F, n = 1.0F, k1 = .123(.05) | -107.641 |
| TC = 69.6(40), $kO_2$ = -.09(.17), n = 1.0F, k1 = .124(.04) | -107.526 |
| NMRI Report 85-16, Model 5, Parameters ABCD | -117.413 |

Note: entries with F indicate parameter fixed at that value and not estimated by data.

the entry in Table 4. Thus the data under all diagnostic categories do not allow a precise estimate to be reached about this exponent. Remember, however, that the effect of the exponent is most sharply seen in the mid-dose range of the dose-response curve (near 50% DCS). In fact all curves have a similar shape in the <10% range exhibited in the data.

The third entry in each section of Table 4 presents the best estimate of how strongly oxygen affects DCS risk, $kO_2$. In all cases the effect is small, that is $kO_2$ is much less than 1, and actually may be zero in each case. The uncertainty in the estimates of $kO_2$ is such that we can only conclude that $O_2$ has less than 50% of the impact of $N_2$ in generating risk of DCS. Examination of the 95% confidence limits on $kO_2$ allows rejection of the high values of $kO_2$ (approximately 0.8) that would be consistent with recent animal decompression experiments (Lillo, 1986). The negative estimate of $kO_2$ means that additional oxygen seems to decrease the risk of DCS.

How do these results compare to other studies? In general, other studies have used so few exposures that no result can be rejected as outside inherent binomial error of the data. The largest data set available is a combined set of over 1700 air dives combined from several sources (Weathersby et al., 1985). Excellent agreement with data was reported for several of the models in that study. A final model and parameter set for DCS incidence from that analysis (labelled Model 5, parameter set ABCD in that report) was applied to the present study in a purely predictive sense. That is, the previous model calculated a p(DCS) for each dive using its inherent assumption that oxygen has no effect. Adding predictions for each of the 477 dives, the present study was predicted to result in 22.6 cases of DCS. That disagrees with the actual results of 11 DCS cases, and is outside the 95% binomial confidence limits on the overall outcome. Examining the predictions more closely, most

of the discrepancy occurs on the 240 min dives, where the previous model predicts that 17.4 cases should have occurred (also outside the 95% confidence limits on the data). The discrepancy persists even if the limited precision of the predictions are accounted for. That is, use of all 95% lower confidence limits on the predictions for the 240 min dives only reduces the expected number of DCS cases from about 17 to about 13 cases which is far from the 3 cases recorded.

If one includes all marginal cases as actual DCS then the disparity between predictions and outcome disappears. However, we believe that such a diagnostic grouping is not comparable to the air data examined in the previous report. In reviewing old reports of DCS trials, one is struck by the severity of symptoms needed for a diagnosis of DCS. Not only would our marginal cases not have been scored as DCS by the original investigators; many cases that we diagnosed and treated in the present series would have been ignored.

Another statistical means to compare these data to the 1700 previous dives is by the likelihood function. Using the model previously shown to well describe the 1700 air dives, the likelihood function for the 3 treatments of data are the final entries in Table 4. For each diagnostic possibility the likelihoods are several units lower (less negative) than the null model. Thus regardless of how the marginal cases are considered, the previously successful model is a poorer descriptor of the present study than is a model which denies that the type of exposure matters at all! The data therefore shows much greater safety than expected.

A more extensive review of other recorded no-decompression dives is presented in Appendix 4. The results are not very helpful in explaining the present data.

DISCUSSION

This study was intended to obtain quantitative answers on the effect of oxygen in human decompression sickness, and on the steepness of the DCS dose-response curve. Despite the study length and the unusually strict quality control the results are frankly disappointing.

Historically, the focus in seeking to prevent DCS has been the inert gas. Donald (1955) reviewed scattered evidence that oxygen adds to decompression stress and presented a qualitative demonstration in goats. Subsequent work in England extended to quantitative studies in both goats (Eaton and Hempleman, 1973) and rats (Rashbass and Eaton, 1957). The latter study concluded that 1/4 to 1/3 of the oxygen adds to nitrogen. More recent intensive work, also in rats, estimates that oxygen is 40-80% as potent as is nitrogen in causing DCS (Lillo 1986). The animal studies have the usual problem of interspecies extrapolation and the further problem of using much more than the 1.3 ATA of $O_2$ that can cause oxygen toxicity in humans. One direct attempt at human measurement was inconclusive due to the small number of total exposures (Logan 1960). Nevertheless, the present basis of computing allowable decompression procedures ignores oxygen (Dwyer, 1955).

There is also literature on the physiology of blood flow dependence on oxygen. Gas uptake rates in tissues depend on flow, and DCS risk depends on the extent of uptake. Classic studies in skeletal muscle (Carrier et al., 1955) suggest a decrease in flow as inspired oxygen increases through the same range used in the present studies. Flow in other tissue beds may not have such a dependence (Hordnes and Tyssebotn, 1985), and of course the tissue beds important to DCS remain an object of speculation. Even more complex oxygen effects may occur in possibly relevant spaces such as synovium (Richman et al., 1982).

Consideration of human decompression sickness as a population dose-response problem has a fairly recent origin (Berghage and McCracken, 1979). Current decompression procedures are formulated as a boundary-avoidance problem and the most common perception is that the imminence of DCS suddenly increases at this boundary. Animal experiments have shown the inapplicability of this view. With the application of probabilistic models to animal (Weathersby et al., 1984; Lillo et al., 1985) and human (Weathersby et al., 1984, 1985) DCS, the need for proper dose-response formulations has come to the foreground. Human helium-oxygen saturation-excursion diving was found to be fit by several different response curves (Weathersby et al., 1984) while animal response data has much better defined shapes. Again, the question of extrapolating the curve shape from rats to man occurs and should be verified in some way.

The study was designed to provide the most complete data ever for both questions of dose-response and oxygen effects. The total study size was relatively large and control of the experiments was unusually precise. Conditions of water immersion, temperature, breathing apparatus, and exercise were consciously chosen to simulate the operational and acceptance testing environments under which most information on human DCS has been obtained. Care was taken to minimize the human subjective element in participants and observers. The pressure-time profiles were more carefully controlled than in any other human decompression study of which we are aware.

The major surprise in this study was the low overall incidence of DCS. Even as the trial was extended longer than originally planned, the feared numerous severe cases never materialized. The challenge now is to explain the unexpected level of safety. The only aspect of the trial that is clearly different from previous work is the tight pressure control.

Regardless of the reason(s), the low DCS incidence makes conclusions difficult: inferences on the cause of DCS are weak if DCS is rarely caused.

Some numerical bounds can be established from the study. The dose-response function is flat with the fractional increase in $p$(DCS) appearing to be less than the fractional increase in pressure. Oxygen does not increase DCS to the same extent as nitrogen; $O_2$ is less than 50% as potent and may actually protect against some of the $N_2$ risk.

Satisfactory answers to the original questions require additional work. Modeling could be improved, for example by exploring a functional dependence of $N_2$ exchange rate on $O_2$, and by including details on control of tissue oxygen content. The main problem however is the paucity of data. Because the present study is unlikely to be repeated or extended, other human trials will need to examined and combined with these results.

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SUBJECT DATA

| Diver number | Age (yrs) | Ht. (in.) | Wt. (lb.) | Fat % | Dives | DCS | Marginal |
|---|---|---|---|---|---|---|---|
| 1. | 40 | 67 | 172 | | 3 | | |
| 2. | 27 | 70 | 188 | | 12 | | |
| 3. | 34 | 70 | 192 | 22 | 4 | | |
| 4. | 32 | 70 | 176 | | 2 | | |
| 5. | 34 | 72 | 175 | | 3 | | |
| 6. | 26 | 69 | 179 | | 12 | | |
| 7. | 32 | 68 | 185 | 25 | 12 | 1 | |
| 8. | 31 | 68 | 155 | | 3 | | |
| 9. | 29 | 70 | 193 | 16 | 4 | | |
| 10. | 26 | 70 | 178 | | 2 | 1 | |
| 11. | 25 | 68 | 165 | | 3 | | |
| 12. | 30 | 74 | 190 | 22 | 4 | | |
| 13. | 30 | 70 | 177 | 17 | 12 | | |
| 14. | 24 | 72 | 195 | | 5 | | |
| 15. | 26 | 74 | 195 | | 2 | | |
| 16. | 30 | 69 | 174 | | 2 | | |
| 17. | 38 | 71 | 190 | 26 | 12 | 2 | 1 |
| 18. | 31 | 75 | 190 | | 1 | | |
| 19. | 37 | 67 | 156 | 15 | 12 | | |
| 20. | 40 | 70 | 185 | | 4 | | |
| 21. | 28 | 69 | 180 | | 2 | | |
| 22. | 27 | 74 | 209 | | 2 | | |
| 23. | 33 | 75 | 204 | 15 | 12 | | |
| 24. | 24 | 72 | 187 | 12 | 12 | | |
| 25. | 32 | 69 | 162 | | 6 | | |
| 26. | 38 | 70 | 176 | 22 | 12 | | 1 |
| 27. | 33 | 72 | 165 | | 12 | | 1 |
| 28. | 27 | 71 | 188 | | 12 | | |
| 29. | 27 | 67 | 158 | | 5 | 2 | |
| 30. | 31 | 73 | 175 | | 12 | | |
| 31. | 35 | 67 | 145 | | 5 | | 1 |
| 32. | 23 | 73 | 167 | 09 | 12 | | |
| 33. | 24 | 73 | 187 | 15 | 12 | | |
| 34. | 30 | 71 | 209 | 12 | 11 | | |
| 35. | 35 | 64 | 152 | | 12 | 1 | |
| 36. | 34 | 70 | 193 | 25 | 12 | 1 | |
| 37. | 30 | 71 | 178 | 15 | 12 | | 2 |
| 38. | 23 | 69 | 175 | 16 | 12 | | |
| 39. | 27 | 71 | 185 | | 12 | | 3 |
| 40. | 32 | 72 | 193 | 23 | 12 | | 1 |
| 41. | 23 | 71 | 165 | 14 | 12 | | |
| 42. | 28 | 72 | 190 | 16 | 12 | | 1 |
| 43. | 29 | 72 | 190 | 21 | 12 | | 1 |
| 44. | 34 | 74 | 222 | 28 | 8 | 1 | 1 |
| 45. | 26 | 67 | 157 | | 12 | | |
| 46. | 26 | 69 | 180 | 16 | 6 | 1 | 1 |

Subject data (continued)

| Diver number | Age (yrs) | Ht. (in.) | Wt. (lb.) | Fat % | Dives | DCS | Marginal |
|---|---|---|---|---|---|---|---|
| 47. | 32 | 68 | 160 | 15 | 12 | | |
| 48. | 33 | 71 | 159 | 17 | 12 | | 1 |
| 49. | 23 | 66 | 132 | 12 | 12 | | |
| 50. | 35 | 67 | 168 | 12 | 7 | | |
| 51. | 25 | 72 | 173 | | 7 | | |
| 52. | 29 | 68 | 162 | 17 | 10 | | 1 |
| 53. | 28 | 74 | 195 | 21 | 9 | | 1 |
| 54. | 30 | 72 | 181 | | 8 | | 1 |
| 55. | 24 | 68 | 192 | 23 | 3 | | |
| 56. | 20 | 73 | 200 | 23 | 6 | | |
| 57. | 28 | 70 | 165 | | 4 | | |
| 58. | 38 | 70 | 165 | | 2 | | |
| 59. | 21 | 71 | 163 | | 5 | 1 | |
| 60. | 20 | 68 | 174 | | 3 | | |
| 61. | 26 | 73 | 180 | | 2 | | |

CHRONOLOGICAL RECORD OF EXPOSURES

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 18APR83 | 1 | 67 | 1.9 | 13 | 20 |
| 19APR83 | 5 | 25 | 0.5 | 23 | 5 |
| 20APR83 | 4 | 72 | 1.7 | 3 | 19 |
| 21APR83 | 6 | 50 | 1.4 | 22 | 4 |
| 06JUN83 | 6 | 50 | 1.3 | 21 | 14 |
| 07JUN83 | 1 | 67 | 1.2 | 2 | 12 |
| 08JUN83 | 5 | 25 | 0.4 | 18 | 22 |
| 09JUN83 | 1 | 67 | 1.0 | 16 | 13 |
| 09JUN83 | 3 | 43 | 1.0 | 20 | 9 |
| 20JUN83 | 6 | 50 | 1.1 | 12 | 2 |
| 21JUN83 | 4 | 72 | 1.5 | 1 | 16 |
| 21JUN83 | 3 | 43 | 0.7 | 20 | 14 |
| 22JUN83 | 6 | 50 | 0.9 | 10( 1) | 4 |
| 30JUN83 | 3 | 43 | 1.1 | 13 | 5 |
| 30JUN83 | 3 | 43 | 1.1 | 8 | 2 |
| 21SEP83 | 2 | 95 | 2.0 | 14 | 7 |
| 22SEP83 | 3 | 43 | 1.0 | 19 | 13 |
| 22SEP83 | 3 | 46 | 1.0 | 8 | 3 |
| 26SEP83 | 5 | 25 | 0.4 | 15 | 21 |
| 27SEP83 | 2 | 95 | 1.5 | 1 | 9 |
| 29SEP83 | 2 | 95 | 2.1 | 12 | 14 |
| 030CT83 | 6 | 50 | 1.4 | 5 | 7 |
| 040CT83 | 2 | 95 | 1.3 | 3 | 15 |
| 040CT83 | 2 | 95 | 1.4 | 10 | 19 |
| 050CT83 | 6 | 50 | 1.1 | 1 | 9 |
| 110CT83 | 1 | 67 | 1.0 | 3 | 23 |
| 110CT83 | 2 | 100 | 1.9 | 8 | 20 |
| 120CT83 | 5 | 25 | 0.3 | 14 | 12 |
| 130CT83 | 4 | 72 | 1.1 | 13 | 9 |
| 27NOV84 | 4 | 72 | 1.3 | 39( 2) | 33 |
| 28NOV84 | 1 | 66 | 1.5 | 35 | 2 |
| 03DEC84 | 4 | 72 | 1.2 | 19 | 37 |
| 05DEC84 | 1 | 67 | 1.2 | 27 | 35 |
| 06DEC84 | 5 | 25 | 1.2 | 28 | 2 |
| 07DEC84 | 1 | 71 | 1.1 | 32 | 30 |
| 10DEC84 | 5 | 27 | 0.4 | 13 | 37 |
| 13DEC84 | 6 | 50 | 0.9 | 19 | 27 |
| 14DEC84 | 4 | 72 | 1.4 | 2 | 33 |
| 17DEC84 | 2 | 100 | 1.5 | 32 | 35 |
| 18DEC84 | 3 | 46 | 0.8 | 7( 3) | 37 |
| 07JAN85 | 2 | 100 | 1.7 | 33 | 13 |
| 07JAN85 | 4 | 76 | 1.7 | 35 | 17 |
| 10JAN85 | 1 | 71 | 1.1 | 19 | 32 |
| 22JAN85 | 6 | 50 | 0.9 | 13 | 37 |
| 22JAN85 | 1 | 71 | 1.8 | 17 | 7 |
| 23JAN85 | 2 | 100 | 1.9 | 35 | 39 |
| 29JAN85 | 6 | 50 | 0.8 | 17 | 7 |

Chronological record of exposures (continued)

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 30JAN85 | 5 | 27 | 0.3 | 33 | 32 |
| 04FEB85 | 5 | 27 | 0.4 | 19( 4) | 13 |
| 04FEB85 | 2 | 100 | 1.6 | 36 | 26 |
| 06FEB85 | 2 | 105 | 1.8 | 39( 5) | 32 |
| 11FEB85 | 3 | 46 | 1.1 | 2 | 33 |
| 12FEB85 | 3 | 46 | 0.8 | 26 | 7 |
| 13FEB85 | 4 | 76 | 0.7 | 39 | 32 |
| 19FEB85 | 3 | 46 | 0.7 | 17( 6) | 33 |
| 20FEB85 | 5 | 27 | 0.7 | 7 | 28 |
| 20FEB85 | 4 | 76 | 2.3 | 32 | 26 |
| 21FEB85 | 4 | 76 | 1.2 | 13 | 35 |
| 27FEB85 | 1 | 71 | 1.1 | 7 | 39 |
| 28FEB85 | 5 | 27 | 0.5 | 32( 7) | 2 |
| 05MAR85 | 6 | 50 | 1.9 | 13 | 27 |
| 06MAR85 | 1 | 71 | 1.1 | 36( 8) | 26( 9) |
| 06MAR85 | 6 | 54 | 0.9 | 33 | 39 |
| 07MAR85 | 5 | 29 | 0.4 | 7 | 19 |
| 12MAR85 | 3 | 44 | 0.8 | 32 | 27 |
| 13MAR85 | 5 | 29 | 0.7 | 39 | 33 |
| 13MAR85 | 1 | 71 | 1.3 | 37(10) | 26(11) |
| 14MAR85 | 1 | 71 | 1.2 | 40 | 19 |
| 14MAR85 | 4 | 76 | 2.9 | 28 | 6 |
| 14MAR85 | 2 | 105 | 1.6 | 13 | 24 |
| 19MAR85 | 4 | 80 | 1.4 | 7 | 27(12) |
| 19MAR85 | 6 | 54 | 0.8 | 2 | 32 |
| 20MAR85 | 3 | 44 | 1.4 | 36 | 39 |
| 20MAR85 | 5 | 29 | 1.1 | 26 | 35(13) |
| 21MAR85 | 6 | 54 | 1.0 | 11 | 6 |
| 21MAR85 | 3 | 44 | 0.8 | 40 | 28 |
| 21MAR85 | 2 | 105 | 1.8 | 33 | 31 |
| 26MAR85 | 2 | 105 | 1.7 | 19 | 2 |
| 26MAR85 | 3 | 44 | 1.1 | 32 | 37 |
| 28MAR85 | 1 | 71 | 1.4 | 6 | |
| 28MAR85 | 6 | 54 | 1.1 | 28 | 33 |
| 28MAR85 | 1 | 71 | 1.3 | 31 | 40 |
| 03APR85 | 5 | 29 | 0.6 | 24 | 39 |
| 03APR85 | 4 | 80 | 1.5 | 2 | 26 |
| 04APR85 | 6 | 54 | 1.2 | 32 | 19 |
| 04APR85 | 5 | 29 | 1.1 | 11 | 31 |
| 09APR85 | 6 | 58 | 1.0 | 37 | 40 |
| 10APR85 | 1 | 71 | 1.2 | 33 | 24 |
| 10APR85 | 2 | 105 | 1.7 | 36 | 7 |
| 10APR85 | 3 | 44 | 0.9 | 25(14) | 26 |
| 11APR85 | 6 | 58 | 1.2 | 6 | 11 |
| 16APR85 | 1 | 71 | 2.0 | 31 | 27 |
| 16APR85 | 2 | 110 | 1.7 | 40(15) | 37 |
| 17APR85 | 6 | 58 | 0.9 | 36 | 24 |

Chronological record of exposures (continued)

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 17APR85 | 4 | 80 | 1.2 | 26 | 7 |
| 17APR85 | 2 | 110 | 1.7 | 2 | 25 |
| 18APR85 | 5 | 31 | 1.2 | 35 | 34 |
| 23APR85 | 4 | 80 | 1.3 | 31(16) | 40 |
| 23APR85 | 1 | 75 | 1.4 | 37(17) | 33 |
| 24APR85 | 4 | 80 | 2.0 | 28 | 36 |
| 24APR85 | 5 | 31 | 2.3 | 41 | 24 |
| 24APR85 | 2 | 110 | 2.0 | 6 | 26 |
| 25APR85 | 3 | 47 | 1.0 | 35 | 19 |
| 25APR85 | 1 | 75 | 1.3 | 39 | |
| 30APR85 | 5 | 31 | 0.9 | 27 | 37 |
| 01MAY85 | 4 | 84 | 1.7 | 36 | 24 |
| 01MAY85 | 4 | 84 | 1.3 | 41(18) | 23 |
| 02MAY85 | 3 | 47 | 1.1 | 28 | 25 |
| 02MAY85 | 3 | 47 | 1.2 | 35 | 39 |
| 02MAY85 | 2 | 110 | 2.1 | 29 | 34 |
| 07MAY85 | 5 | 31 | 0.7 | 26 | 30 |
| 07MAY85 | 4 | 84 | 1.7 | 37 | 27 |
| 08MAY85 | 4 | 84 | 1.9 | 23 | 41 |
| 08MAY85 | 3 | 47 | 0.9 | 24 | 36 |
| 09MAY85 | 6 | 58 | 1.4 | 40 | 39(19) |
| 09MAY85 | 3 | 47 | 1.1 | 34 | |
| 14MAY85 | 3 | 47 | 1.0 | 27 | 30 |
| 14MAY85 | 1 | 75 | 1.6 | 28 | 25 |
| 15MAY85 | 5 | 31 | 0.5 | 23 | 36 |
| 15MAY85 | 3 | 50 | 0.9 | 24 | 41 |
| 16MAY85 | 6 | 58 | 1.2 | 35 | 42 |
| 16MAY85 | 3 | 50 | 2.3 | 34 | 29 |
| 21MAY85 | 5 | 33 | 0.5 | 43 | 25 |
| 21MAY85 | 4 | 84 | 2.9 | 30 | 40 |
| 22MAY85 | 5 | 33 | 0.5 | 46 | 41 |
| 22MAY85 | 1 | 75 | 1.4 | 24 | 23 |
| 23MAY85 | 5 | 33 | 0.9 | 34 | 29(20) |
| 23MAY85 | 2 | 110 | 2.0 | 37 | 42 |
| 29MAY85 | 4 | 88 | 1.8 | 30 | 43 |
| 29MAY85 | 3 | 50 | 0.9 | 40 | 23 |
| 30MAY85 | 4 | 88 | 1.6 | 24 | 46 |
| 30MAY85 | 1 | 75 | 1.5 | 42 | 41 |
| 30MAY85 | 4 | 88 | 1.5 | 34 | 29(21) |
| 04JUN85 | 2 | 115 | 2.3 | 27(22) | 28 |
| 04JUN85 | 6 | 62 | 1.2 | 26(23) | 45 |
| 05JUN85 | 5 | 33 | 0.5 | 36(24) | |
| 05JUN85 | 2 | 115 | 2.2 | 23 | 43 |
| 18JUN85 | 2 | 115 | 2.3 | 24 | 28 |
| 18JUN85 | 1 | 79 | 1.3 | 34 | |
| 20JUN85 | 6 | 62 | 1.5 | 36 | 35 |
| 25JUN85 | 1 | 79 | 1.3 | 28 | 42 |

Chronological record of exposures (continued)

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 25JUN85 | 2 | 115 | 1.9 | 38 | 43 |
| 26JUN85 | 3 | 50 | 0.9 | 46 | 6 |
| 26JUN85 | 6 | 62 | 1.2 | 24 | 45 |
| 27JUN85 | 1 | 79 | 1.4 | 36 | |
| 27JUN85 | 2 | 115 | 2.1 | 34 | 25 |
| 02JUL85 | 1 | 79 | 1.7 | 41 | 30 |
| 02JUL85 | 6 | 62 | 1.1 | 38 | 28 |
| 03JUL85 | 3 | 50 | 1.1 | 23 | 6 |
| 03JUL85 | 6 | 62 | 1.8 | 46(25) | 29 |
| 09JUL85 | 1 | 79 | 1.3 | 47 | 45 |
| 09JUL85 | 6 | 66 | 1.1 | 41(26) | 30 |
| 10JUL85 | 3 | 53 | 1.1 | 42 | 38 |
| 10JUL85 | 6 | 66 | 2.0 | 23 | 34 |
| 11JUL85 | 1 | 79 | 1.8 | 43 | 48 |
| 16JUL85 | 3 | 53 | 1.0 | 47 | 45(27) |
| 16JUL85 | 6 | 66 | 1.1 | 41 | 30 |
| 17JUL85 | 4 | 88 | 1.4 | 42 | 38 |
| 17JUL85 | 5 | 33 | 0.8 | 27 | |
| 18JUL85 | 4 | 88 | 0.7 | 46(28) | 43 |
| 18JUL85 | 5 | 33 | 0.6 | 48 | |
| 23JUL85 | 6 | 66 | 2.1 | 47 | 23 |
| 23JUL85 | 5 | 33 | 1.2 | 45 | 6 |
| 24JUL85 | 6 | 66 | 1.2 | 42 | 38 |
| 24JUL85 | 3 | 53 | 0.9 | 41 | 30 |
| 24JUL85 | 2 | 120 | 1.7 | 27 | 40 |
| 30JUL85 | 5 | 35 | 1.0 | 6 | 45 |
| 30JUL85 | 2 | 120 | 2.5 | 23 | 47 |
| 30JUL85 | 2 | 120 | 1.2 | 48 | 49 |
| 31JUL85 | 5 | 35 | 0.7 | 40(29) | 42(30) |
| 31JUL85 | 5 | 35 | 1.0 | 38 | 30 |
| 01AUG85 | 1 | 83 | 1.7 | 17(31) | 43 |
| 01AUG85 | 5 | 35 | 1.6 | 46(32) | |
| 06AUG85 | 4 | 88 | 1.3 | 47 | 49 |
| 07AUG85 | 5 | 34 | 0.6 | 42(33) | 40 |
| 07AUG85 | 2 | 120 | 2.1 | 41 | 30 |
| 08AUG85 | 4 | 88 | 1.6 | 17(34) | |
| 08AUG85 | 4 | 88 | 1.5 | 6 | 45 |
| 13AUG85 | 1 | 83 | 1.6 | 38 | 47 |
| 13AUG85 | 2 | 120 | 2.1 | 49 | 48 |
| 14AUG85 | 3 | 53 | 1.1 | 43 | 42 |
| 15AUG85 | 1 | 83 | 1.6 | 45 | 6 |
| 15AUG85 | 3 | 53 | 0.9 | 17(35) | |
| 04SEP85 | 4 | 88 | 2.5 | 42 | 34 |
| 04SEP85 | 1 | 83 | 1.0 | 38 | 48 |
| 05SEP85 | 5 | 34 | 1.5 | 50 | 17(36) |
| 05SEP85 | 2 | 125 | 2.2 | 41 | 30 |
| 05SEP85 | 3 | 53 | 1.2 | 45 | |

Chronological record of exposures (continued)

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 11SEP85 | 3 | 56 | 1.6 | 43(37) | 51 |
| 11SEP85 | 3 | 56 | 1.5 | 38 | 47 |
| 12SEP85 | 2 | 125 | 2.4 | 53 | 45 |
| 12SEP85 | 2 | 125 | 2.0 | 42 | |
| 12SEP85 | 6 | 70 | 1.3 | 34 | 52 |
| 01OCT85 | 2 | 125 | 1.9 | 38 | 17(38) |
| 02OCT85 | 3 | 56 | 2.9 | 50 | 44 |
| 08OCT85 | 1 | 83 | 0.9 | 53 | |
| 08OCT85 | 6 | 70 | 2.9 | 52(39) | 51 |
| 09OCT85 | 1 | 83 | 1.8 | 49 | 50(40) |
| 09OCT85 | 6 | 70 | 1.2 | 47 | 17(41) |
| 10OCT85 | 3 | 56 | 1.2 | 48 | 44 |
| 10OCT85 | 5 | 34 | 0.5 | 38 | 43 |
| 15OCT85 | 3 | 56 | 1.0 | 53 | 52 |
| 15OCT85 | 1 | 87 | 2.0 | 51 | |
| 16OCT85 | 2 | 125 | 2.0 | 47 | 45 |
| 17OCT85 | 3 | 59 | 1.0 | 48 | 54(42) |
| 17OCT85 | 6 | 70 | 1.9 | 43 | 44 |
| 22OCT85 | 2 | 125 | 2.2 | 52 | 17(43) |
| 22OCT85 | 6 | 70 | 1.6 | 53 | 51 |
| 23OCT85 | 4 | 88 | 1.2 | 38 | 47 |
| 23OCT85 | 4 | 92 | 1.6 | 45 | |
| 24OCT85 | 1 | 87 | 2.0 | 44 | 49(44) |
| 24OCT85 | 4 | 92 | 1.9 | 48 | 54 |
| 29OCT85 | 6 | 74 | 1.4 | 53 | |
| 29OCT85 | 1 | 87 | 1.6 | 52 | |
| 31OCT85 | 1 | 87 | 2.0 | 44 | 54 |
| 31OCT85 | 4 | 92 | 2.1 | 49 | 48 |
| 31OCT85 | 5 | 34 | 0.5 | 17 | 47 |
| 05NOV85 | 1 | 87 | 1.6 | 50 | 55 |
| 06NOV85 | 5 | 34 | 0.7 | 56 | 52 |
| 07NOV85 | 5 | 36 | 0.6 | 47 | 49(45) |
| 12NOV85 | 4 | 92 | 1.8 | 54 | 57 |
| 12NOV85 | 1 | 87 | 1.7 | 53 | 55 |
| 12NOV85 | 5 | 36 | 1.9 | 48 | 50(46) |
| 13NOV85 | 4 | 92 | 2.2 | 51 | 59 |
| 13NOV85 | 4 | 96 | 2.6 | 56 | 52(47) |
| 14NOV85 | 2 | 130 | 2.1 | 61 | 6 |
| 14NOV85 | 4 | 96 | 1.7 | 44(48) | 60 |
| 14NOV85 | 3 | 59 | 1.0 | 49 | |
| 19NOV85 | 3 | 59 | 1.0 | 54 | |
| 19NOV85 | 6 | 74 | 1.7 | 48(49) | 43 |
| 20NOV85 | 4 | 96 | 2.2 | 51 | 56 |
| 20NOV85 | 6 | 74 | 2.9 | 58 | 59 |
| 21NOV85 | 1 | 91 | 1.5 | 52(50) | 60 |
| 21NOV85 | 4 | 96 | 1.8 | 53 | |
| 21NOV85 | 6 | 74 | 1.7 | 50 | 49 |

Chronological record of exposures (continued)

| Date | Series | Depth (fsw) | Descent Time(min) | Subject Number (incident number) | |
|---|---|---|---|---|---|
| 26NOV85 | 4 | 96 | 3.0 | 61 | |
| 26NOV85 | 5 | 36 | 0.8 | 54 | |
| 27NOV85 | 4 | 96 | 3.2 | 59 | 58 |
| 27NOV85 | 5 | 36 | 1.1 | 51 | 56 |
| 03DEC85 | 1 | 91 | 1.7 | 57 | |
| 03DEC85 | 5 | 36 | 0.7 | 53(51) | 60 |
| 04DEC85 | 3 | 59 | 1.3 | 52 | 44 |
| 04DEC85 | 5 | 36 | 0.8 | 54 | 59 |
| 05DEC85 | 4 | 96 | 1.7 | 50 | |
| 05DEC85 | 5 | 38 | 1.2 | 49 | 56 |
| 10DEC85 | 3 | 59 | 1.2 | 57 | |
| 11DEC85 | 2 | 130 | 2.4 | 59(52) | 54 |
| 12DEC85 | 3 | 59 | 1.1 | 49 | |
| 17DEC85 | 6 | 74 | 1.8 | 48 | 56 |
| 17DEC85 | 2 | 130 | 1.7 | 55 | 57 |
| 17DEC85 | 4 | 96 | 1.4 | 53 | |
| 18DEC85 | 2 | 130 | 2.2 | 52 | |
| 19DEC85 | 6 | 74 | 1.2 | 44(53) | 49 |

SYMPTOMS REPORTED ALL DIVES

1. Subject 10. 22 June 83. Reported pressure on mid-back from seat during the dive. No symptoms for 2 1/2 hours after surfacing. After dinner that evening complained of mid-back pain and rash, left posterior flank pain, and fatigue. Exam showed erythema over T8 spinous process and over left anterior infracostal area, also hypoesthesia in left anterior infracostal area. Standard TT6 brought substantial relief within 1-2 minutes. All symptoms resolved in 20-30 min except for pinpoint tenderness over spinous process of T8. This local tenderness resolved over the next several days. Dx: DCS.

2. Subject 39. 27 Nov 84. Reported to have had "skin bends" about 4 hours after surfacing. Described as 6 cm round, raised, red lesion over right side of waist, slightly pruritic. No treatment. Exam was normal 2 hrs after surfacing. Dx: skin bends, not DCS

3. Subject 7. 18 Dec 84. Normal exam 2 hours after surfacing, although subject noted a "full" feeling. Awoke the next day with intermittent sharp pain in right thigh above the right knee. Found to have mildly decreased strength in right lower extremity, decreased right ankle reflex, decreased position sense in the right big toe, crossed abductor response on left, focal tenderness to touch on the right medial thigh, and tenderness in a 6-8 cm area in a T12 distribution on the left posterior ribs. TT6 brought relief of all symptoms and signs except for the right thigh tenderness within 20 min. The right thigh pain persisted several days and was felt to be due to mechanical injury. Dx: DCS.

4. Subject 19. 4 Feb 85. Seen about 20 min after surfacing for light-headed feeling immediately after a transient lower abdominal pain. Pale appearance, symptoms, and vital signs (P 60, BP 100/60) consistent with vasovagal reflex. Neurologic exam normal. Dx: not DCS.

5. Subject 39. 6 Feb 85. Blotchy, dusky, patchy skin rash over anterior chest and upper abdomen and near left axilla about two hours after surfacing. Not itchy or painful. Reported very transient (less than one min) ache in right shoulder. Rash spread, then faded over the following hour. Normal neurologic exam. Dx: skin bends, not DCS.

6. Subject 17. 19 Feb 85. Noted pain in left distal forearm 1 h 45 min after surfacing. Exam showed tenderness to palpation over wrist and ulnar head anteriorly on left, pain aggravated on grasping or pulling, and mild decrease in pinprick sensation over medial aspect of hand. Remainder of exam normal. Standard TT6, with normal sensation in 20 min and complete relief of all symptoms in an hour. Dx: DCS.

7. Subject 32. 28 Feb 85. Noted right knee pain on forced extension after dive. Had same pain during dive and in past with exercise. Exam unremarkable. Dx: not DCS.

8. Subject 36. 6 Mar 85. Dull left upper arm pain beginning about 4 hours after surfacing. Tender to palpation, otherwise exam normal. Standard TT6 brought 50% relief in five minutes, complete relief within an hour. Dx: DCS.

Symptoms reported all dives (continued)

9. Subject 26. 6 Mar 85. Brief (half minute) ache in left forearm at two hours after surfacing, 2-3 min pain in left elbow at 3 1/2 hours. Normal exam. No treatment. Dx: niggles.

10. Subject 37. 13 Mar 85. Four episodes of transient (5-10 min each) aches in right shoulder and elbow and left knee over a 3 hr period the evening after diving. Normal exam the following day. Dx: niggles.

11. Subject 26. 13 Mar 85. Two or three episodes of aching joints the evening after diving, each lasting 1-2 min. Had similar transient aches several days during the week prior to the dive. Normal exam the following day with no pains. Dx: not DCS.

12. Subject 27. 19 Mar 85. Brief ache in left heel about 20 min after surfacing. Dull ache in left knee lasting about 1 min, onset about 2 hr after surfacing. Normal exam on both occasions. Dx: niggles.

13. Subject 35. 20 Mar 85. Reported 8 hr after surfacing that both shoulders had dull, poorly localized ache for the previous hour, which then subsided. Normal exam. Dx: DCS, not requiring treatment.

14. Subject 25. 10 Apr 85. Normal exam 2 hr after surfacing. Mild soreness developed in left groin that evening. Exam the following day showed mild tenderness to palpation. Stiffness decreased with activity. Dx: Muscle strain, not DCS.

15. Subject 40. 16 Apr 85. Very mild dull ache in left midscapula 1 hr 45 min after surfacing. No variation with movement. Normal neurologic exam. Lasted about 15 min. Subsequently noted identical pain several days later following the same mechanical movement which had preceded pain on 16 April. Dx: not DCS.

16. Subject 31. 23 Apr 85. Mild left elbow ache lasting about 5 min the evening following the dive. Normal exam. Dx: niggles.

17. Subject 37. 23 Apr 85. Beginning about one hour after surfacing, had four episodes of dull aches in right knee, right shoulder, and left elbow, lasting about three minutes each. Last episode ended about four hours postdive. Exam normal. Dx: niggles.

18. Subject 41. 1 May 85. Reported ache in right ribs about one hour after surfacing, lasting about 5-10 minutes. Normal exam. Dx: not DCS.

19. Subject 39. 9 May 85. Developed skin itch just before 2 hr doppler, accompanied by erythema on trunk. Subject heard recording. Neuro exam normal except for generally increased muscle tone, including 3+ DTR's and 1-2 beat nonsustained clonus in both ankles. Pulse 108, BP 164/100. No nausea, dizziness, or dyspnea. Blotchy rash on trunk gradually shifted and became bluish and raised, but never tender. Pulse and blood pressure returned to normal within 1/2 hr, and subject became less anxious. At four hours postdive, subject was normal except for mild itching. Close examination throughout this period showed no neurologic abnormalities. Dx: Skin bends and nonspecific findings suggesting catecholamine release. Not DCS.

Symptoms Reported After All Dives (continued)

20. Subject 29. 23 May 85. Reported dull, deep ache in right shoulder the night after the dive. Pain gone the next morning. Not reported until the following week. Dx: DCS, not treated.

21. Subject 29. 30 May 85. Right shoulder pain beginning three hours after surfacing, gradual increase in intensity. Full resolution on standard Treatment Table 5. Dx: DCS.

22. Subject 27. 4 Jun 85. Very mild dull ache in right trapezius and right arm 4 hrs postdive, noted about 10 min after lifting a heavy object. No tenderness to palpation. Normal exam. Dx: Mechanical injury, not DCS.

23. Subject 26. 4 Jun 85. Several very brief (few seconds duration) episodes of wrist, shoulder, and foot pain the evening after the dive. Normal exam the following day. Dx: not DCS.

24. Subject 36. 5 Jun 85. No problems the day of the dive. Sharp medial left knee pain the following morning. Localized to a spot medial to the left patella, tender on palpation, aggravated on weight bearing and rotating foot laterally. Normal neurologic exam. Clinically diagnosed as mild medial collateral ligament strain. TT5 conducted with no change. Dx: not DCS.

25. Subject 46. 3 Jul 85. Reported two hours postdive that he was "very, very tired." No aches or other symptoms, normal exam. Felt fine the next day. Dx: not DCS.

26. Subject 41. 9 Jul 85. Mild tenderness of left knee reported the following day. Recalls hitting knee. Localized tenderness of anterior tibial tuberosity on exam, no other abnormalities. Dx: Mechanical injury, not DCS.

27. Subject 45. 16 Jul 85. Fatigued after dive, felt tired all evening. Normal exam two hours postdive. Also noted "pins and needles", while he was sitting that evening, extending from lateral right midcalf down through lateral aspect of foot, which disappeared upon walking. Felt fine the next day. Dx: not DCS.

28. Subject 46. 18 Jul 85. Several transient pains, each lasting 1-2 min: moderate pain in right ankle 20 min postdive, tingling of right foot 10 min later, moderate pain of right knee 15 min later. Exam normal. Also reported crepitance in left temporomandibular joint postdive. Dx: niggles. (TMJ symptoms probably mechanical, from mask).

29. Subject 40. 31 Jul 85. Several episodes of 3-4 min aches in right hip, ankle, and scapular region in the first hour postdive. Normal neurologic exam. Dx: niggles.

30. Subject 42. 31 Jul 85. Two brief (less than 5 sec each), sharp, stabbing pains in abdomen about 45 min after surfacing. Examined immediately, normal exam. Reported passing gas both before and after the dive. Dx: not DCS.

Symptoms reported all dives (continued)

31. Subject 17. 1 Aug 85. Reported a 5-10 sec pain in left elbow after dive. Normal exam. Dx: not DCS.

32. Subject 46. 1 Aug 85. Fleeting, 10-15 second pain over the left side of neck about 1 hour after surfacing, followed within a few minutes by left shoulder pain. Pain was described as deep-seated ache. Left upper extremity slightly weaker than right in wrist extension and elbow flexion. DTR's symmetric, sensation intact, remainder of neurologic exam normal. Recompressed to 60 FSW. Pain and weakness relieved within first 20 min of treatment. Treated with TT6 extended once at 30 FSW, and judged by medical officer to have had complete relief. Examined by neurologic consultant the following day and found to have slight weakness in left finger and wrist extensors. No improvement with repeat TT6. Neurologic examination one week later, including EMG, completely normal. Dx: DCS.

33. Subject 42. 7 Aug 85. Dull ache for about 5 min in right shoulder, occurring over two hours postdive, and dull pain for less than one minute in right foot the evening following the dive. Normal exam the next day. Dx: niggles.

34. Subject 17. 8 Aug 85. No symptoms for 24 hrs after surfacing. Flew on commercial airline the following day and had moderate, sharply localized pain in right wrist about 1 hour after landing. Subsided in 1-2 hrs. Dx: No DCS from the experimental dive, but niggles precipitated by further decompression of flight.

35. Subject 17. 15 Aug 85. Fleeting pain in right elbow and left fingers several hours after surfacing. No further symptoms; normal exam. Dx: not DCS.

36. Subject 17. 5 Sep 85. 4-5 episodes of brief (less than one minute) right shoulder pain. Normal exam. Dx: niggles.

37. Subject 43. 11 Sep 85. Two nearly identical episodes of dull right supraclavicular pain occurring at about 5 and 6 1/2 hrs after surfacing. Pain described as moderate, dull, not aggravated by movement, nontender, lasting about 5 min each time. Exam the following day normal. Dx: niggles.

38. Subject 17. 1 Oct 85. Normal exam two hours after surfacing. About four hours postdive, experienced marked fatigue and fleeting dull pains (less than one minute duration) in left wrist, both elbows, both shoulders, and a sharp pain in the sternoclavicular joint. Felt normal within about 45 min. Exam the next day normal. Dx: DCS, no treatment.

39. Subject 52. 8 Oct 85. Mild left shoulder ache about two hours after surfacing, lasted about 3 min, unaffected by movement. Normal exam. Dx: niggles.

40. Subject 50. 9 Oct 85. Very brief (few seconds) pain in right hand, neck. Normal exam. Dx: not DCS.

Symptoms reported all dives (continued)

41. Subject 17. 9 Oct 85. At one hour after surfacing, subject experienced sudden "flushed" feeling, followed by mild fatigue. No pains or other complaints, and exam was normal. Fatigued feeling lasted about 1/2 hr. Dx: nonspecific symptoms, not DCS.

42. Subject 54. 17 Oct 85. Blotchy, pruritic rash on anterior trunk about two hours after surfacing. Faded over 5-6 hrs. Normal exam. Dx: skin bends.

43. Subject 17. 22 Oct 85. Sharp pain in left shoulder of only a few seconds duration twice after surfacing. Normal exam. Dx: not DCS.

44. Subject 49. 24 Oct 85. Sharp pain in right shoulder of only a few seconds duration about 1 1/2 hr after surfacing. Also had low back pain the evening after the dive, identical to pain which has occurred before following exercise. Normal exam. Dx: not DCS.

45. Subject 49. 7 Nov 85. Brief lightheaded feeling about 5 min after surfacing, after removing tight wetsuit. Normal examination. Dx: not DCS.

46. Subject 50. 12 Nov 85. Very brief sharp pain in right elbow and left ankle late the night of the dive. Normal exam. Dx: not DCS.

47. Subject 52. 13 Nov 85. Noticed mild pain in third finger the evening of the dive. Although subject did not recall trauma, skin was scraped. Normal neurologic exam. No change with TT5 started the next morning. Dx: mechanical injury, not DCS.

48. Subject 44. 14 Nov 85. Mild pain superior and lateral to left eye about twenty minutes after surfacing, worse upon gaze to the right. Tender to palpation. Neurologic exam, including visual acuity, visual fields, extraocular movement, corneal reflex, and blink reflex, was normal. No change in symptoms for an hour, until recompression initiated. Symptoms resolved soon after treatment started, with full resolution on standard TT6. Dx: DCS.

49. Subject 48. 19 Nov 85. Three episodes of strong, throbbing pain the evening after surfacing, each lasting "only a minute or two": left wrist twice and left medial arm near shoulder. Normal exam. Dx: niggles.

50. Subject 52. 21 Nov 85. Dull pain posterior to right ear about 30 min after surfacing, lasting less than one minute. Dull pain in left trapezius about one hour after surfacing, lasted less than one minute. Normal exam. Dx: not DCS.

51. Subject 53. 3 Dec 85. Dull ache in right knee about 2 1/2 hours after surfacing, lasting 2-3 minutes. Normal exam. Dx: niggles.

52. Subject 59. 11 Dec 85. Dull, deep, right hip pain 3-4 min after surfacing from dive, felt as if penetrating through the hip. Not radiating in a dermatomal pattern, and no evidence of any neurologic involvement. Immediately recompressed, and pain disappeared within 10 fsw. Full relief on TT6A. Dx: DCS.

Symptoms reported all dives (continued)

53. Subject 44. 19 Dec 85. Well until 3 1/2 hours after surfacing, when he noted itching in right periumbical region. Pruritis progressed to a circumferential erythematous lesion which blanched on pressure. Examined about 6 hrs after surfacing, found to have two well-demarcated, purple skin lesions, periumbilical and over right ribs 9-11 posteriorly. Mild tenderness to palpation in abdomen. No neurologic abnormalities. Rash improved the following morning, but abdominal tenderness to deep palpation had changed in location. No symptoms or other signs to explain abdominal pain. Patient recompressed to 60 fsw, and abdominal tenderness improved within an hour. Treated on TT6 with an extension at 30 fsw, with full resolution of abdominal tenderness. Etiology of abdominal tenderness uncertain, but not felt to represent DCS. Rash also resolved with treatment. Dx: skin bends.

Note: the following two cases were excluded from the data because a treatment was instituted for other than DCS.

54. Subject 25. 18 Jun 85. Series 1, 79 fsw. Onset of dull, right upper chest ache about three hours after surfacing, aggravated by arm movement. Tender to palpation. Normal neurologic examination. Had lifted weights the day before diving and was stiff predive. No response to abbreviated TT6. Dx: not DCS.

55. Subject 53. 10 Dec 85. Series 2, 130 fsw. Mild, dull ache in left trapezius, with no tenderness to palpation, no history of trauma, and normal strength and sensation. Treated with TT6, with only slight improvement slowly over the duration of the treatment. Lack of response with prompt recompression felt incompatible with DCS. Dx: not DCS.

Note: "Niggles" in the above refers to mild musculoskeletal symptoms which appeared related to the dive but resolved without treatment over a short time. Joint symptoms were considered niggles if they lasted more than two minutes, recurred in the same location, or involved several locations.

## COMMENTS ON OTHER REPORTED NO-DECOMPRESSION DIVES

The previous report combined data from a number of sources into a data set of 1713 air dives (Weathersby et al, 1985). Though there seemed to be incompatibilities within parts of those data, the combination of a very wide range of exposures was pursued so that predictions of safety would interpolate within conditions found in the large data set rather than extrapolate far from known exposures.

No subset of the previously examined data closely matches the new experimental conditions. All of the previous data used 21% oxygen (i.e. air). One segment of the old data (set B) had exposures of 90 to 360 min, but 1.5 to 12 hours were spent in decompression. Another segment (set C) had no-decompression dives, but they were deep and very short - most under 3 min. Segment D of the old data included many different decompression profiles, but none had an average decompression rate as fast as 10 ft/min (the current study used 60 feet per minute). The fastest overall decompression in set D had 7 min at decompression stops. Only segment A (Dwyer 1956) had some nearly equivalent exposures. A total of 56 dives were done with no decompression or 2 min or less in decompression stops. All but 8 of these had a bottom time of 20 min or less. Using the same prediction applied in the text, a total of 0.07 cases of DCS would be expected for the 56 dives and zero were reported. This constitutes a weak agreement of prediction and outcome.

Several other studies were briefly examined in our previous report but not included in the large consolidated data file. One British report presented the results of 87 no-decompression dives (Crocker and Taylor 1952). (Called set H in the earlier report). These dives varied from 50 feet for 90

min to 150 ft for 11 min, apparently somewhat more hazardous dives than the present study. Using the prediction method described, 0.5 cases of DCS were expected and none were observed. This gives us another weak reconciliation of safe no-decompression dives.

Two reports had significant DCS cases. In the first, Van der Aue and colleagues (1949) reported 141 no-decompression exposures with 42 cases of DCS. The number fell short of their avowed goal of 50% DCS. The longest was 100 ft for 60 min: predicted incidence of 6.6%, outcome of 7 cases in 20 exposures. In 30 min exposures, they had 16 cases in 32 exposures from 150 ft. Prior modeling predicted only 4.3 cases total. To confound interpretation, the men were dry and sedentary during the pressure exposure but half exercised vigorously after decompression. Thus in very ambitious experiments, men can develop many cases of DCS, even more than predicted by the analysis of 1713 other dives.

The other old report was by Van der Aue and colleagues in 1951. In a major study of surface decompression, some 143 no-decompression exposures were performed. Bottom times ranged from 5 to 205 min but averaged only 35 min. With a single depth of 94 ft, 24 exposures of 32 to 48 min were performed and resulted in 5 DCS cases. The 94 ft dives are predicted to result in 0.1 cases of DCS, and only 1.3 cases are predicted overall. The total outcome of 9 cases in 143 trials is much greater than predicted. Again, an instance is found where DCS can be produced by no-decompression dives of high enough severity that extensive modeling underestimates the actual risk.

The final data are too new to be published (Thalmann, 1985, unpublished). Some 130 exposures were performed with no-decompression from dives of less than 66 min duration. All exceeded the current U.S. Navy limit, but none resulted in DCS.

No single theme unifies these observations. No-decompression diving can indeed produce DCS, but it has usually been cited only in dives much more severe than the present experiments. In the several hundred supporting dives for this experiment (training and tender breathing air) three clear cases of DCS developed when DCS stress should be low, e.g. 60 fsw for 30 min. Perhaps the underlying dose-response relationship has been poorly estimated; perhaps current practice and a modest region beyond is relatively safe, but exposures much beyond turn suddenly disasterous. Perhaps short dives of nearly any kind are relatively safe, and only the present 4 h dives are anomalous. No equivalent data for 4 hour shallow exposures has been located for comparison; clearly more analysis is needed.