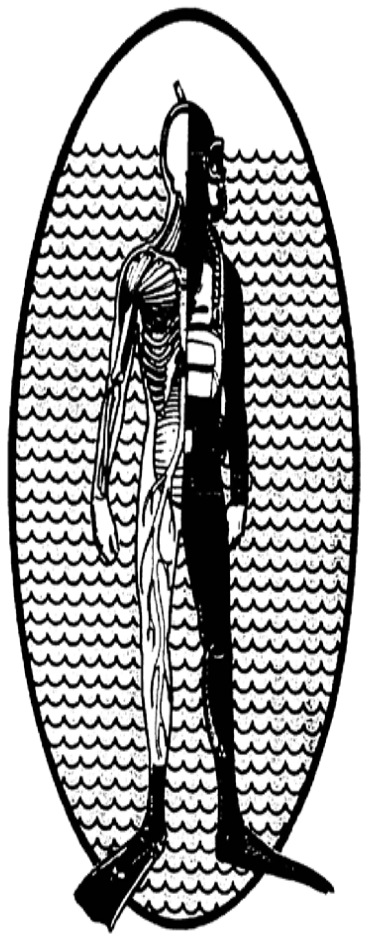
PHYSIOLOGY IN DEPTH



The Proceedings of the Seminar

December 6, 1980 - Santa Ana, California

Sponsored by

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OVERVIEW

This special seminar, held on December 6, 1980 in Santa Ana, California, was organized to assemble a panel of highly qualified individuals to explain complex and misunderstood aspects of diving physiology, to correct misconceptions associated with the topic, and to answer questions from participants.

The panel experts included Bruce E. Bassett, Ph.D. of the USAF School of Aerospace Medicine in San Antonio, Texas; Paul Linaweaver, M.D., an Undersea Medicine Specialist from the Santa Barbara Medical Foundation Clinic; Andrew A. Pilmanis, Ph.D. of the University of Southern California Institute for Marine and Coastal Studies; Christopher Dueker, M.D., author of diving medical texts and articles; Charles Brown, M.D., Medical Editor of SKIN DIVER Magazine; and other notable personalities.

The program consisted of individual presentations by the panel members, with sessions to answer participant questions held at the end of both the morning and afternoon presentations.

Eighty seven divers and instructors attended the highly successful event and gained new insights into diving physiology. The evaluation questionnaires from the attendees indicated a high degree of satisfaction with the program and the manner in which it was conducted by Dennis Graver, PADI Director of Training.

PHYSIOLOGICAL CONCERNS OF WOMEN SCUBA DIVERS

By Susan Bangasser, Ph.D.

Many women now enjoy the sport of scuba diving. The number of women certified from the basic diver through instructor levels is continuously increasing. Yet, many women divers experience anxiety over participating in some scuba diving activities due to personal problems. For example, can a female dive in the ocean when she is having her menstrual period? Most scuba classes do not touch on these subjects and most female students are reluctant or embarrassed to ask the instructor. I will cover most of the physiological areas that are of concern to women divers. These areas include diving in thermal comfort, diving during the menstrual period, diving while using birth control pills and diving during pregnancy.

The first subject, diving in thermal comfort, covers the sensitivity of females to cold and hot conditions. Women have a layer of subcutaneous fat which acts to retain body heat because of the low thermal conductivity of fat. This has been shown to be an asset to the Ama divers of Korea (1). However, in my experience as an instructor, many female divers suffer from diving in cold water sooner than their male colleagues. A recent study indicates that there is indeed a physiological basis for this phenomenon. In considering an individual's sensitivity to cold, two factors must be considered: 1 — the degree of body fatness, and 2 — the ratio of surface area to body mass. Lean women (less than 27% body fat) have a large surface area to body mass ratio and therefore cool at a faster rate (2). Above a fatness of 30%, men and women maintain similar low levels of heat production when placed in cool water (2). To solve this problem, anyone who cools easily should invest in an adequate and properly fitting wet or dry suit. If uncontrollable shivering begins, get out of the water.

Sensitivity to heat is important to divers carrying heavy gear and suiting up during the hot summer months. The female's body temperature will rise 2° to 3° higher than the male's before the cooling process of sweating begins (3). Also, the female has fewer functional sweat glands. The solution to this problem during prolonged periods of very strenuous activity is to periodically cool off. A dunk in the water before donning one's tank and weight belt is usually easy to do.

Another area of concern to women is diving during the menstrual period. Whether a woman should dive during this time depends on just how well she feels. If the diver feels fine, go ahead and dive. In the Olympics of 1972 and 1976, female medal winners were at all stages of the menstrual cycle. The menstrual period did not prevent top performances by the athletes. If, however, severe cramps or discomfort are experienced, the woman should postpone her diving. One complication of the menstrual period is fluid retention. Edema may make a woman more sensitive to decompression sickness (4). Females should follow conservatively the No Decompression Table of the Navy Dive Tables, if diving three days prior or during her period.

Women who dive in salt water during their period frequently ask, "What about sharks?" The average blood loss during a period is 25 to 70 cc, in three

to four days. Internally worn protection, e.g. a tampax, is preferable to an externally worn napkin. There is no evidence of increased shark interest in a menstruating female, so the woman diver should concentrate on the other factors of her dive and enjoy herself.

The third area of discussion is that of diving while using birth control pills. First, I would like to cover the possible susceptibility to decompression sickness of women in general. After a deep dive, a prolonged dive to moderate depths, or a repetitive dive, a diver may get decompression sickness. This is the formation of nitrogen bubbles in the tissues. Decompression sickness has been studied in nurses undergoing flight training at the United States Air Force School of Aerospace Medicine. Much of the work was done by Dr. Bruce Bassett, Major, USAF, from 1968-72. During these five years at the USAFSAM, there were nine cases of decompression sickness out of 12,246 exposures (5). The females have a tenfold greater incidence than the males undergoing the same exposures (5). More recent studies have verified the increased incidence of decompression sickness in women over men undergoing flight training. As a result of these studies, flight nurses are now exposed to different altitude chamber flight profiles.

In a later study by Dr. Bassett, the incidence of decompression sickness during the ten year period from 1968 through 1977 was analyzed. The incidence in men was 0.09% while in women it was 0.36%. This gave a 4-fold greater incidence of bends for the women (12). Other observations made include women having more skin symptoms, a more rapid onset of pain, more recurrences and lasting effects of decompression sickness when compared to men with identical exposures (12).

Since the greater incidence of decompression sickness occurred in altitude training, can this be relevant to the scuba diver? In order to answer this question, I included a portion on decompression sickness in the Medical Aspects of Women Divers Survey. This survey was conducted from October, 1977 through June, 1978, and had over 700 respondents. A portion of the survey was sent to male divers. The data indicated that a group of women divers (instructor level certifications) had a 3.3-fold greater incidence in suspected or treated decompression sickness, compared to their male colleagues (11). This was a significant increase in incidence. The woman diver should, therefore, avoid decompression dives and follow the No Decompression Table conservatively. By this I mean, stay less time at a particular depth than indicated.

Now, let's return to the question of birth control pills. The pill has caused blood clots in some women, and is suspected to cause a slowing of the circulation in some women, it may increase their susceptibility to decompression sickness. Birth control pills used today have lower hormone levels than those used even ten years ago. Possibly these pills are less of a problem. However, the woman diver should continue her safe diving practices of avoiding decompression dives and using the No Decompression Table conservatively. In order to avoid any increased chance of decompression sickness, the women scientists of Tektite II saturation project discontinued birth control pills three months prior to their saturation dive (6).

A question many women ask is "Can I dive while pregnant?" Some women

enjoy the sport and wish to continue as long as they are able. Others may be going on vacation, and the warm, clear water will be very inviting. Many obstetricians believe that if a woman has been active in a sport prior to pregnancy, she can continue the sport during pregnancy as long as she feels comfortable. However, are there any risks to the fetus if the woman continues to dive? Research is being conducted on this topic, and we do not know all the answers. Because of this lack of information, some medical authorities believe the pregnant woman should not dive at all. Nevertheless, many pregnant women have dived during pregnancy without any apparent problems. The pregnant diver must decide for herself if she chooses to dive and if so, under what conditions she can safely dive. To help the diver decide, I will present some of the information available.

The first possible problem is the fluctuation in oxygen partial pressures (pO_2). Will the fetus get sufficient oxygen? Will the fetus get too much oxygen? Both can be serious problems.

Research has been done on mammals on the transfer of gases across the placenta. Oxygen transfer to the fetus across the placenta must be rapid and continuous to assure successful growth. There are special mechanisms that assist the fetus in transplancental oxygen exchange: 1. higher hemoglobin concentration in the fetal blood, increasing its oxygen carrying capacity, and 2. higher affinity for oxygen of the fetal hemoglobin. Only severe hypoxia in the mother will cause changes in the fetal oxygen content (7). The placenta prevents transient fluctuation in fetal blood pO_2.

Scuba divers, however, must be concerned with increasing pO_2 in fetal blood. In experiments on gravid ewes, the maternal arterial oxygen tension was increased by providing 100% oxygen at one atmosphere. (This pO_2 is equivalent to breathing air at a depth of 132 feet of sea water.) There was only a very small increase in fetal blood pO_2 (7). In sheep, cow, pig, and primate, only small changes occurred in fetal blood pO_2 when maternal oxygen intake was increased (8). In contrast, an experiment on rats did demonstrate fetal wastage with hyperbaric oxygen (9). In most of the animal studies fetal oxygen content remained relatively stable during significant maternal pO_2 increase. Also, there appear to be differences in placental gas transfer between different groups of mammals.

A sport diver who limits her dives to 132 feet will probably not have problems with high-oxygen pressures (hyperoxia). However, if the diver suffers from decompression sickness, she will be treated in a hyperbaric chamber. During the treatment, divers are given oxygen at the equivalent pressure of around 60 feet. The partial pressure of oxygen at this depth would be almost three atmospheres. This pressure may cause some harm to the fetus. Therefore, the pregnant diver should make an effort to avoid decompression sickness.

Another potential problem for the fetus is the possibility of fetal decompression sickness. The question of fetal susceptibility to the bends has not been resolved, and several investigators are currently looking for the answers (13).

Studies done years ago on dogs and rats indicated that even when the mother was bent, the fetus did not appear to suffer from decompression

sickness. However, a more recent study on pregnant sheep had conflicting results. Sheep were selected because their placenta more closely resembles the human's. In these experiments, a dopler transducer, capable of detecting bubbles, was surgically attached to the umbilical artery of the sheep. The sheep were then exposed to pressures in a hyperbaric chamber for the duration of the maximum allowable time of the U.S. Navy No Decompression Dive Table. An interesting result was obtained. Dives at depths greater than 60 feet resulted in bubble formation in the fetus, while no bubbles were detectable in the maternal circulation either by dopler or by visual inspection (10). At depths of sixty feet and less, bubbles were not detected in the fetus. These tests infer greater infant susceptibility to the bends. Also, previous studies indicated that sheep are more bend resistant than humans. These findings are preliminary and there are differences in the placenta between humans and sheep. Because of these findings and because of the uncertain results in humans, diving at depths greater than 60 feet appears to be contraindicated for the pregnant diver.

Another group of researchers set out to repeat the experiments with sheep. This time a catheter was implanted, and bubbles were again detected. The same results were found — bubbles in the fetus but not in the mother. But then one day twins were born. The infant with the catheter had bubbles, but the untouched infant sheep was normal. More experiments revealed that if the fetus was left untouched during the dive, the baby was born without any signs of distress (14). If the fetus was surgically manipulated, this fetus would bubble. These findings indicated that the techniques used may have introduced or caused bubble formation. The earlier experiments on pregnant sheep did not use the control experiment of treating pregnant sheep in the hyperbaric chamber without violating the uterus. Maybe the resulting babies would have been healthy. Obviously more experiments are currently being conducted. In the meantime, it is my opinion that the pregnant woman dive at a maximum of thirty feet.

In order to gather retrospective information on human subjects, two questionnaires have circulated among diving women. One of the them, the Medical Aspects of Women Divers survey, incorporated a section on pregnancy. The result of this survey indicated that of all the women who were diving before pregnancy, 72% made at least one dive during pregnancy (11). Many women made deep dives, especially prior to knowledge of their pregnancy. Once aware of their pregnancy, most women limited themselves to around a forty foot dive, and made less strenuous dives. The pregnant diver usually discontinued diving either around the fourth or fifth month of pregnancy when size became a problem. Only 10% of the respondents dived through the ninth month. None of the 72 respondents reported any abnormalities of their babies.

Another survey, Pregnancy and Diving, was distributed to a selected group of women. Two groups of women were compared, those that dived during pregnancy and those that dived prior to but not during pregnancy (15, 13). The rate of physical abnormalities for the diving group exceeded the non-diving group, but was comparable to the rate for the general public (15).

Research on the pregnant diver will continue. Although the available information looks encouraging, particularly for shallow dives, not all the answers are known. A safe maximum depth and time has not been established. In my opinion, a pregnant diver should limit her dives to 30 feet, and avoid strenuous dives. This conservative approach to diving during pregnancy fits into the guidelines established by the available research.

The concerns of current and future women divers are finally being addressed (16). Hopefully, men and women divers will keep abreast of the information that will make diving safer and more enjoyable for everyone.

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PRACTICAL ASPECTS OF DIVING TABLES FOR SPORT DIVERS

Bruce E. Bassett, Ph.D.

Introduction

Over the years that divers in the United States have used scuba for recreational diving, the U.S. Navy Standard Air Decompression Tables have been the accepted reference for avoiding decompression, calculating repetitive dives, or for making correct decompression stops. Decompression tables were designed to prevent decompression sickness (DCS). How successful are these tables in preventing DCS? In the U.S. Navy, DCS occurs in only 0.03 to 0.04% of all dives made per year.[1] Is this record also attained in sport diving? No one can answer that question with certainty because the incidence of DCS in sport diving is, and will likely remain, unknown.

As the sport of scuba diving has grown and developed, most instructors and certifying agencies have tended to agree on certain basic safety principles which should be followed by sport divers. Thus, **most** agree that: 130 feet should be the maximum depth; all dives should be made within the no-decompression limits of the U.S. Navy tables; and divers should not "push" these no-decompression limits. It is within the third recommendation that there are a wide variety of "factors" espoused by different divers/instructors. Some reduce the limits by a fixed amount of time (i.e. 5 minutes), some use a percentage of the time (2 to 10%) to reduce the limits, some add "safety" decompression stops, and most merely say "don't push the tables."

It is proposed here that SPORT DIVER TABLES be designed to provide a standardized approach for sport divers to avoid "pushing" the limits of the U.S. Navy Tables. The rationale for altering the basis of the U.S. Navy tables will be explained as will the design of such tables. The proposed SPORT DIVER TABLES are not to be extrapolations of the U.S. Navy or any other tables per se, but they will be compared with others. Finally, the tables are strictly designed with the sport diver in mind. They are totally devoted to avoiding "pushing the limits."

Rationale for Altering the Tables

A. U.S. Navy Experience

If one looks at the U.S. Navy statistics regarding the incidence of DCS, i.e. 0.03 to 0.04%, the logical questions formed is "why alter the tables?" The logic of this question is reinforced by a lack of DCS incidence statistics for sport divers in general. However, in examining the U.S. Navy statistics, the question must be asked, "how does the U.S. Navy dive?" and more specifically, "how does the U.S. Navy dive with open-circuit scuba?" The U.S. Navy Diving Manual does not set **absolute** depth/time limits for the use of scuba, but states that "whenever possible, scuba operations **must** be conducted so that no decompression will be required" (Chap. 5, page 5-40, 5.3.6.). It also states, (Chap. 4, figure 4-17), that 60 feet is the **normal** working limit for open circuit scuba, and 130 feet is the **maximum** working limit. As far as these recommendations go we can assume that all, or nearly all, U.S. Navy scuba dives are conducted **within** the no-decompression limits. But what

fraction of Navy scuba dives are nowhere near these limits? If a high percentage of their scuba dives are shallower than 40 feet, or much shorter than the deep time limits, (which is probably the case by the nature of their use of scuba), then the DCS incidence figures are misleading by an inflated denominator, i.e. incidence = # DCS cases/# dives. The incidence figure of concern to sport divers would be # DCS cases/# dives **made to the exact no-decompression limits.** That statistic is not readily available for U.S. Navy dives.

B. Laboratory Findings

Merrill Spencer, M.D., conducted a large number of dives to the U.S. Navy no-decompression limits (and some beyond the limits) in a recompression chamber, and a smaller number in the open ocean, using the Doppler Ultrasonic bubble detector to monitor post-dive intravascular bubble formation.[2] In chamber dives, his results were as presented in Table 1. His experience, as others have noted with open water dives, was that a higher percentage had bubbles and bends than on equivalent "dry" dives in the chamber.

Andrew Pilmanis at the USC Catalina Marine Science Center found, also using the "bubble detector," that open water dives to 100 feet for 25 minutes produced intravascular bubbles in all subjects and that the degree of 'bubbling" was influenced by the level of work performed while at depth.[3] No bends occurred in his subjects, but one other significant finding was noted. In a subject who had repeated significant "bubbling" upon surfacing directly from an exposure of 100 feet for 25 minutes, the degree of bubbling could be drastically reduced by a short stop at 10 feet and eliminated by short stops at 20 and 10 feet. This may indicate that only minor alterations in the no-decompression limits may drastically alter the results, and that perhaps the present limits are on the "knifes-edge" with regard to bubble formation and bends.

Table 1 — Data of Merrill P. Spencer, M.D.

	Dive Profiles (Depth/Time)				
	70/50	60/60	25/720	165/10	165/10*
Number of exposures:	12	13	9	6	6
% subjects w/bubbles:	33.3	31	56	33	83
% subjects w/bends:	8.3	7.6	11	0	33

*all dives in chamber except**

Ulf Balldin from Sweden has made interesting observations regarding flying following dives made to the limits of the U.S. Navy no-decompression limits, specifically 50 feet for 100 minutes and 130 feet for 10 minutes.[4] These were also "dry" chamber dives, and the subjects were monitored with the bubble detector while exposed to altitude. The altitude exposures were made to either 3,000 meters (9,840 ft.), 2,000 meters (6,560 ft.) or 1,000 meters (3,280 ft.) after a three hour surface interval. The incidence of intravascular bubbling was as shown in Table 2.

Table 2 — Data of Ulf Balldin, Ph.D.

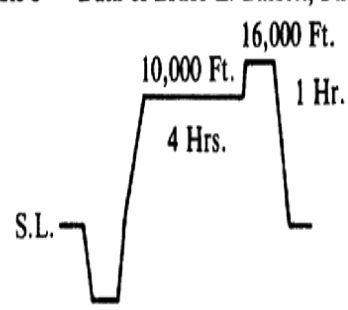
50/100 or 130/10 chamber dives followed by a 3 hour surface interval and then ascent to the indicated altitude.

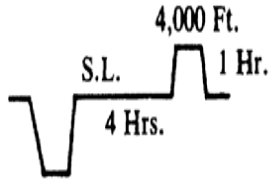
Altitude	% subjects w/bubbles
3,000 M (9,840 ft.)	60%
2,000 M (6,560 ft.)	30%
1,000 M (3,280 ft.)	10%

It is interpreted that silent bubbles were formed upon surfacing from the dives, which then became detectable upon further pressure reduction to altitude. There were no bends among his subjects, but again, these were "dry" chamber exposures.

In a tri-service project conducted at the USAF School of Aerospace Medicine concerning flying immediately after diving, further evidence was gained that the present no-decompression limits may not be sufficiently conservative.[5] Exposure times at depths to 130 fsw were calculated to give the same decompression stress with an ascent directly to 10,000 feet as diving to the no-decompression limits and ascending directly to sea level, i.e., the comparative profiles were as shown in Table 3.

Table 3 — Data of Bruce E. Bassett, Ph.D.





Dives:		Dives:	
	130/7		= 130/10
	100/10		= 100/25
	80/14		= 80/40
	60/20		= 60/60
	40/34		= 40/200
	10.75/24 hours		= 22/24 hours

The results were as shown in Table 4.

Table 4 — Data of Bruce E. Bassett, Ph.D.

Depth	%Bubbles	%Bends	#Subjects
130	0	0	20
100	38.9	5.5	18
80	37.5	6.3	16
60	27.8	5.5	18
40	22.2	5.5	18
10.75 (=22)	20.0	5.0	20

In summary, the laboratory evidence is that the "true" incidence of intravascular bubbling and bends on dives made to the U.S. Navy no-decompression limits:

1. ranges from 10-56% bubbles in dry chamber exposures;
2. ranges from 83-110% bubbles in open water exposures;

3. ranges from 0-11% bends in dry chamber exposures;
4. ranges from 0-33% bends in open water exposures;
5. long shallow dives produce higher bubble/bends incidence than short deeper dives.

This then is the rationale for proposing reduced no-decompression limits for sport divers. If Sport Diver Tables are to be designed to make diving safer, then additional consideration must be given to their design. One area of investigation that has been performed has been an analysis of the Repetitive Dive Tables of the U.S. Navy.

The repetitive group letters of the Navy system are based on a theoretically calculated difference in nitrogen pressure, equivalent to 2 fsw per group letter, in the slowest theoretical compartment/tissue (i.e. the 120 minute half time). When all dives within the U.S. Navy Standard Air Decompression Tables are calculated, there are many anomalies found. The range of mean differences in P_{N_2} between subsequent repetitive group is from 1.2 fsw (A to B) to 1.8 fsw (G to H and M to N). Also, the range of P_{N_2}'s found within a given repetitive group letter is from 2 fsw (A) to 6 fsw (M).[6]

Because of these anomalies, such conditions as the 60/30, 30 minute surface interval, 60/30 repetitive dive sequence that produces a decompression dive on the second 60/30 dive arise. While this example is obviously a safe, conservative approach, a more precise system of repetitive groups would reduce such anomalies and make the system produce more "realistic" results.

Design Criteria for Sport Diver Tables

A. Limits of Navy Tables

The limiting factors in the U.S. Navy tables are the calculated nitrogen tensions in six theoretical "tissues." These nitrogen tension limits were determined empirically (i.e. by trial and error) during the development and testing of the Standard Air Decompression Tables and are referred to as "M-values" for **maximum** nitrogen tensions allowed upon surfacing. These M-values, which are expressed in feet of sea water absolute (fswa), can also be expressed as ratios of nitrogen tension to total pressure, i.e. by dividing the M-value by 33 fswa (the absolute pressure at sea level). These are presented in Table 5.

Table 5

Half-time tissue (min.)	5	10	20	40	80	120
M-valve (fswa)	104	88	72	58	52	51
Ratio	3.15	2.67	2.18	1.76	1.58	1.55
No-decompression limits	110/20	90/30	70/50	50/100	40/200	
	120/15	100/25	80/40	60/60		
	130/10	110/20	90/30	70/50		
		120/15	100/25	80/40		

B. Reducing the Limits

Since these limits represent the driving force for gas phase separation (i.e. bubble formation), they are the limits which must be reduced to design a set of tables for sport divers. Considering the laboratory evidence presented earlier, the recommended revised M-values and ratios for proposed Sport Diver Tables presented in Table 6.

Table 6

Half-time tissue (min.)	5	10	20	40	80	120
M-value (fswa)	95	83.2	67	53.8	46.5	44
Ratio	2.88	2.52	2.03	1.63	1.41	1.33
Reduction in M-value (fswa)	-9.0	-4.8	-5.0	-4.2	-5.5	-7.0

C. Limits of Sport Diver Tables

Using the reduced M-values, the calculated "no-decompression" limits of the proposed Sport Diver Tables are given in Table 7.

Table 7 — SPORT DIVER TABLE LIMITS:

Schedule	Limiting Tissues
140/5	5
130/5	5
120/10	5
110/15	5
100/20	10
90/25	20
80/30	20
70/40	20, 40
60/50	40
50/70	40
40/120	40, 80
35/180 & 30/220	120

D. Repetitive Diving Revisions

In order to produce repetitive groups for strictly "no-decompression" Sport Diver Tables, to provide for more flexibility in repetitive dive planning and to eliminate most of the anomalies encountered in using the U.S. Navy repetitive dive tables, 18 repetitive groups are proposed (instead of the 15 involved in the Navy no-decompression tables) with a precise range of 1 fswa per repetitive group designation. In the proposed Surface Interval Credit Table, there is no minimum time of ten minutes, but with short surface intervals, the Residual Nitrogen times will always be equal to or greater than the Bottom Time of the first dive.

Finally, an additional part of the proposed Sport Diver Tables will be to include a three minute minimum stop at 10 to 20 feet on **all** dives made. This is again an attempt to standardize safety factors and put in an extra margin to make the tables as safe as possible.

Recommendations/Summary

A. Sport Diver Tables

In summary, the proposed Sport Diver Tables:

1. are more conservative in their design limits than the U.S. Navy tables;
2. provide standardized safety factors;
3. appear slightly more complex than the present tables;

4. are designed to be presented like the PADI tables — simple to use with practice;
5. reduce some of the anomalies present in the U.S. Navy repetitive dive system;
6. should be validated by manned testing, but can be used without because they are more conservative in all respects than the presently accepted standard;
7. can be used for multi-level/step diving.

B. Flying After Diving

Because of the results of the studies of Balldin and of Bassett, previously discussed, most, if not all of the current recommendations regarding flying after diving are suspect.

Edel's[7] two-hour surface interval rule does not appear to be sufficiently conservative, since 3 to 4 hours in the two studies cited was insufficient time to clear bubbles apparently formed as a result of dives made to the no-decompression limits. Additional evidence that bubbles, once formed, may persist for as long as 18 hours.

The "D group rule" may not be any better for the same reasons; Namely, if bubbles are formed, they may persist during the surface interval involved in allowing the decay of the repetitive group from some higher letter down to a "D" group. Surfacing from a dive with a D group and immediately flying to a maximum of 8,000 feet above sea level (unpressurized flight altitude or cabin altitude of pressurized aircraft) may be safe. However, C.L. Smith's[8] recommendations that it is safe to fly to 10,000 feet with a D group is unsafe, as shown by the Air Force tests, i.e. Table 8.

Table 8

Schedule	Group	%Bends	%Bubbles
130/7	D	0	0
100/10	D	5.5	38.9
80/14	D	6.3	37.5
60/20	D	5.5	27.8
40/34	E	5.0	22.2

Use of the "Cross Corrections"[9] for flying immediately after diving would also not be sufficiently conservative, particularly at the shallower depths as indicated in Table 9.

Table 9 — Cross Limits for 10,000 feet

	Tested Schedules	%Bubbles
130/5	130/7	0
100/5	100/10	38.9
80/15	80/14	37.5
60/30	60/20	27.8
40/60	40/34	22.2

In view of these findings, it is recommended that a **minimum** surface interval of 12 hours be allowed between any diving and flying to any altitude in excess of 3,000 feet.

C. Diving at Altitude

Related in most respects to flying after diving, the situation with diving at altitude is also unsettled. The "Cross Corrections," if used in conjunction with the Sport Diver Limits, should be sufficiently conservative. Decompression dives using the Cross Corrections should **not** be performed. An additional safety precaution is to not dive at altitude until 12 hours after arrival there.

As an alternative, there are some tables developed by the Swiss that appear to be very conservative and which have the added benefit of having been validated by manned testing.[10]

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GAS SPACES IN DIVERS

Charles V. Brown

Once upon a time, Robert Boyle discovered that if you raise the pressure around a bag of gas it gets smaller, and if you lower the pressure it gets bigger, or tries to. Any diver could have told him that. Yet divers still run afoul of Boyle's law. They break the rules. Violation is punishable by squeeze, direct or reverse, with results anywhere from annoying to fatal. The rules, violations, and penalties are the subject of this paper.

Two tracts in the body normally contain gas. One is the alimentary canal for food processing. The other is the breathing tract, along with its associated sinuses and middle ear spaces.

The Alimentary Canal

The esophagus, stomach, and intestine may harbor gas. They're not subject to direct squeeze because they're flexible and don't resist collapse. They are subject to reverse squeeze — damage by distension.

Two things that set one up for reverse squeeze are eating unwisely and swallowing air. Intestinal bacteria produce gas, sometimes in prodigious quantity if supplied the right fodder. Some people have aerophagia — they swallow air. If you're one of these, and you swallow repeatedly to clear your ears while diving, you can ingest a significant amount of compressed air. Belch it up quickly before it joins the gas already in your intestine. Gas in the gut expands during ascent, and if there's too much of it, you may decide you'd rather stay down.

There's a time not to swallow. A Hawaiian who dives a lot told me about it. By the end of dive number five, his throat felt awfully dry, and as he ascended, he swallowed repeatedly trying to moisten it. He felt pain under the breast bone, and the further up he went, the more it hurt. It eased some after surfacing but didn't go away, and swallowing food or water hurt like mad. He thought about pneumomediastinum and heart attack, but his doctor ruled those out. What he had was reverse squeeze of the esophagus. Expanding air had stretched it and damaged the lining. It was probably close to rupture.

Then there was a cave diver trapped in a tunnel when its roof collapsed. His air ran out, and like most people who are drowning, he began to swallow water. At that moment he somehow broke free. Another diver saw he wasn't breathing, popped a fresh regulator into his mouth, and took him up. The victim, by then semiconscious or unconscious, kept swallowing automatically and drank air. During the ascent, it expanded and ruptured his stomach. A timely operation saved his life.

Gas in the gut has two other effects you'd never guess. If you've carefully trimmed for neutral buoyancy at a precise depth so you can photograph something on a vertical wall and then gas up, you'll have to re-trim. More seriously, the gas generated by intestinal bacteria includes methane and hydrogen sulfide. As the saying goes, it all comes out in the end. If you're living in a saturation habitat or a submarine, it will need to be vented with fresh air, as is done with Hydrolab, or fitted with filters to scrub the offensive vapors.

The Sinuses

The sinuses have rigid walls, so are subject to both squeeze and reverse squeeze. Each is lined with a membrane that has blood vessels and mucus glands. The mucus drains through an opening into the back of the nose. Usually the opening permits easy equalization of air pressure when you dive, but occasionally the membrane swells enough to block it, and the squeeze is on.

If you re-seal an empty kerosene can and push it down through the water, in just a few feet it will buckle inward. That's the simplest kind of squeeze. It doesn't happen in the sinus or anywhere else in the body, because the presence of a circulatory system modifies the mechanism. As a diver descends, increasing pressure transmitted throughout his body pushes blood into the vessels of the sinus mucus membrane, swelling it and compressing the air in the sinus to satisfy Boyle's Law.

Those vessels can take just so much. To appreciate the stress, consider what diving does to blood pressure. At the surface, arterial pressure may be 130/80 and capillary pressure 20 mm of mercury. At 33 feet, add 760 mm to all values. Arterial pressure becomes 890/840 and capillary pressure becomes 780. That's no problem if the sinus clears, since the air within it will be at 760 mm (gauge) pressure, and the capillaries will only have to support their usual 20 mm differential. If the sinus doesn't clear, the differential will be immense and the capillaries can hardly be faulted for bursting and bleeding.

It's usually not that bad. The capillaries are elastic enough to accommodate some volume change. If descent is not too fast, there'll be time for fluid to leak through the capillary walls, swelling the membrane further and then dripping into the sinus. Mucus will be secreted too and add to the liquid pool. Together these factors may ease the squeeze enough to prevent capillary rupture.

Sinus squeeze may or may not hurt. When it does, maxillary sinus pain is felt in the cheek near the nose. It's easily mistaken for a toothache. An ethmoid sinus hurts over the nose, and a frontal sinus over an eye. Sphenoid sinus squeeze produces an ache deep in the head. It's rarely reported, probably because it doesn't happen often, and when it does is likely to be misinterpreted. There are a fifth pair of sinuses — the mastoids — located behind the ears. They drain through the middle ears and eustachian tubes. They share squeeze with the middle ears, and the ear symptoms are so much more compelling that the mastoids aren't even thought of.

Sinus squeeze usually heals by itself, but occasionally leads to infection that can be stubborn and, if not controlled, spread to the brain. Doctors handle it with decongestants and antibiotics, and sometimes irrigation or surgical drainage. You shouldn't dive with sinus infection. You can tough out the pain, but it's not smart. An instructor candidate developed a deep headache, and we cancelled her final checkout dive. X-rays later showed severe sphenoid sinusitis. Had she made the dive, she might have paid for her instructor's certificate with a brain abscess.

Reverse squeeze of a sinus is rarely a problem, but the possibility mustn't be ignored. A young man diving near Santo Domingo ignored the tightness and pressure above one eye, left over from a recent cold. He made it to 100 feet

and stayed 23 minutes. During ascent the pressure sensation became severe, and his eye began to hurt. At the surface, the eye was bulging as though trying to escape from the orbit. X-rays showed that the floor of the frontal sinus had blown out, letting compressed air escape into the orbit behind the eyeball. He recovered after intensive treatment.

One final sinus question pops up now and then. Neurosurgeons like to get at pituitary tumors by going through the sphenoid sinus, and the defect they make in its bony wall heals with scar tissue. The question is whether it's safe to dive afterwards. Expert opinion is divided. I think it's safe if there are no complications, and know of one such patient who did resume diving.

The Middle Ear

Middle ear squeeze is basically like sinus squeeze, but there are important differences. The drainage channel — the eustachian tube — is more easily blocked. The walls of the middle ear have windows, and the proximity of the inner ear makes it vulnerable to harm when the middle ear is stressed.

The eustachian tube is a little over an inch long. It opens into the nasopharynx (the back of the nose and throat) in a way that permits easy drainage but hinders retrograde flow. This is probably a defense against infection, but makes it hard to get air into the middle ear when you need to. The tube is opened periodically by several small muscles that attach near its end and contract when you swallow. This is necessary because middle ear air is absorbed into the bloodstream and must be replaced. It opens for a fifth of a second about once each minute while you're awake, and every five minutes during sleep.

Various things can make the membranes in and about the eustachian tube swell and block it. They include allergy, infection, chemical irritants, cold, autonomic nervous system activity, and drugs. When the tube stays blocked, even non divers get ear squeeze, because the pressure in the middle ear becomes lower than the ambient one atmosphere. Divers subject themselves to much greater pressure differentials, so get squeezed much faster and harder.

The windows of the middle ear are the large one in its outer wall covered by the eardrum, and two small ones in its inner wall, the oval and round windows, covered by membranes similar to the eardrum. A hinged chain of bones connects the eardrum to the oval window membrane to transmit sound waves from the outer to the inner ear. The membrane of the round window, by moving in opposite phase to that of the oval window, relieves the sound pressure and makes higher wave amplitude possible.

When the middle ear is squeezed, its mucus membrane, like that of a sinus, engorges with blood, swells with edema fluid that drips into the air space, and may bleed. Its windows are also affected. The eardrum bulges inward and may tear with as little as five or six feet of descent, though usually more. The tearing may or may not hurt, but it does equalize pressure and so relieves the pain of squeeze. Water may or may not enter through the tear. If it does, it cools the wall between the middle and inner ear, and sets up a convection current in one of the semicircular canals that tells the brain that you're spinning. The vertigo soon stops as the water warms up, and there's no danger if you don't panic.

With squeeze, the round window membrane also bulges into the middle ear. Like the eardrum, it can rupture, but the consequences are more serious. The inner ear fluid, called perilymph, will leak into the middle ear, disturbing the inner ear pressure relationships. Either the shock wave at the time of rupture or the loss of perilymph damages the organs of the inner ear. The result may be any combination of hearing loss, ringing in the ears, disorientation, dizzyness, and nausea.

The oval window membrane is stressed by squeeze too, but paradoxically in the opposite direction. Because it's connected to the eardrum, it can move only in the same direction. The geometry of the linkage gives the oval window membrane a mechanical advantage of 1.7, but the difference in area gives the eardrum an advantage of 10, so it wins the pushing contest. So if the oval window ruptures, it will be into the inner ear. The point is academic since the result is the same as for round window rupture.

Since ear squeeze is the most common cause of distress in diving, and its consequences can be disastrous, it's worthwhile to review the strategies available to counter it. Some fortunate folks equalize automatically and descend without thought. A few tell me they've learned to open their eustachian tubes voluntarily, like some people can wiggle their ears. Most of us don't have the magic and must resort to other measures.

The first is to descend feet first — the pressure differential in the nose makes clearing easier. The second is to descend slowly and try to clear frequently. The eustachian tube opens most easily when the pressure differential is low — about ten inches of sea water. When the differential reaches six feet or more, it may lock completely. The third measure is to try a clearing maneuver. Move your jaw about and swallow, or better, swallow with your nose pinched off. If these fail, fall back on the two big guns — the valsalva or frenzel maneuvers.

The valsalva is the most popular because it's simple. Close your mouth, pinch your nose, and blow. With a regulator in your mouth, close the airway by raising your tongue against your palate. The valsalva is fine when it works easily, but blowing hard for more than a few seconds is dangerous. It raises the pressure in your chest and tends to collapse the great veins. There are two grave consequences. First, blood denied entry to the chest swells the great veins and capillary beds elsewhere, including inside the rigid walled spinal canal and brain case. This exerts pressure on the cerebrospinal fluid. It also prevents reabsorption of the cerebrospinal fluid into the venous circulation. Since the fluid is constantly produced, the pressure rises more. This same cerebrospinal fluid bathes the inner ear, where it's called perilymph. Its increased pressure, added to the squeeze already pushing the round window membrane into the middle ear, makes rupture more likely.

The second danger from collapse of the great veins is underwater blackout. The strongest heart can't pump blood it doesn't receive, so cardiac output falls, and the brain is starved for oxygen. Without oxygen, it stops working in about eight seconds.

To avoid the hazards of a long, strong valsalva, we recommend the frenzel maneuver. It's a lot safer, and even more powerful. Most instructors don't teach it because they don't know what it is. Diving medicine experts I've

talked to don't teach it because it's difficult to learn. I don't think it has to be. To do a frenzel, shut off your mouth and nose as for the valsalva, then also close your throat. You've completely walled off a small space into which the eustachian tubes open. Now tighten the muscles of your neck and throat so they won't give, and push your tongue further upward and backward, compressing the air until you feel your ears clear.

The hard things are learning to close your throat and putting it all together. To get the feel of a closed throat, just cough. The instant before the cough came out, your throat was closed. Coordination comes with practice, and once learned, the frenzel is like bicycling or skating — you do it without thinking. A recent study in Sweden showed that experienced divers have as much trouble with their eustachian tubes as do beginners, but suffer less ear damage. The reason — without realizing it, is that most of them had switched from the valsalva to the frenzel.

Reverse squeeze of the middle ear is not common, but can be painful and even dangerous. The main causes are sensitivity to cold and rebound congestion. The nasal membranes are supposed to swell when it's cold so as to warm the incoming air. This does no good when you're scuba diving, since you're breathing through the mouth. However, the nose doesn't know that, so swells anyway. In sensitive people it may swell enough to block the eustachian tubes. Rebound congestion occurs when a decongestant medicine wears off and leaves your nose plugged worse than it was to start with. If you use a strong but short acting spray like neosynephrine, you may be able to dive with a bad cold or allergy. When the effect wears off underwater, you'll have to make choice between coming up and suffering, or staying down and drowning.

If you elect to come up, you risk more than pain. Reverse squeeze can blow out an eardrum or window. It can even paralyze your face. The seventh cranial nerve, which supplies the muscles of facial expression, passes close to the middle ear, and sometimes its bony canal is defective. Middle ear overpressure puts it to sleep by collapsing the blood vessels that nourish it, leaving you expressionless. Luckily, recovery is spontaneous.

Alternobaric vertigo, or change-of-pressure dizzyness, occurs when one eustachian tube works better than the other and makes the pressure in the two middle ears different. Probably 15% to 20% of all divers experience it at some time or other, more often during ascent than descent. To relieve the vertigo, simply reverse your direction for a few feet, equalize, and then proceed more slowly.

A final caution: Some divers accustomed to using the valsalva or frenzel during descent have thoughtlessly used the maneuver when they felt ear pressure during ascent. This of course just makes the overpressure worse and adds to the risk of ear damage. The way to deal with reverse ear squeeze is to descend a few feet and swallow, then ascend slowly, swallowing as needed. There is something called the Mueller maneuver — sucking inward against a blocked nose and throat, but it's not very effective.

Scientists hate to use simple words, so when they want to talk about pressure injury they translate it to Greek and say barotrauma. Barotrauma of the ear affects the organs concerned with hearing, spatial orientation, and sensing change in motion. It's essential that divers recognize the symptoms, so we'll repeat them: any combination of hearing loss, ringing in the ears, disorientation, dizzyness, and nausea. These same symptoms can be caused by decompression sickness, but the treatment is very different. Consultation with an ear specalist who knows diving medicine is indicated, and delaying it invites permanent damage.

Lung Squeeze

The lungs are subject to squeeze and reverse squeeze. Not easily, since they're designed for variable volume, but with life threatening potential.

Lung squeeze in the conventional sense probably doesn't happen. Theory had it that if a breathhold diver were to descend beyond the depth at which his lungs reached residual volume, squeeze would begin. For example, a diver's total lung capacity might be five times his residual volume — the volume of air remaining in his lungs after full exhalation. He'd reach residual volume at five atmospheres absolute or 132 feet, and beyond that his lungs would become engorged with blood, waterlogged with edema fluid, and susceptable to hemorrhage.

When Bob Croft of the U.S. Navy dived to 240 feet, the experts were shaken. Trained breathhold divers develop larger total lung capacities and smaller residual volumes than the rest of us, but not enough to account for Bob's exceeding the theoretically safe limit by so much. They didn't want their nice theory spoiled, so they postulated that Bob was a freak with extraordinary lung mechanics. Then a pair of Jacques — Maiorca and Mayol, exceeded 300 feet, and it was definitely back to the drawing board.

The fallacy in the theory was the assumption that lung squeeze should begin as soon as actual lung volume becomes lower than residual volume. It doesn't, for two reasons. First, sea pressure probably makes the chest cavity smaller than the diver can voluntarily make it, by pushing in on the rib cage and up on the diaphragm. Second, and far more important, the pulmonary capillaries are highly elastic and can accommodate a lot of extra blood before they rupture.

When the capillaries are over-distended they will leak fluid, but there's a time course involved. With a small squeeze stress, pulmonary edema develops slowly. When the stress is great, it develops rapidly. In the deep dives mentioned, the combination of squeeze stress (depth) and time wasn't enough to cause symptoms.

For any would-be heroes, let me emphasize that most of us don't have the physiological adaptations that Bob and the Jacques enjoy, and would not survive such profound plunges. If squeeze didn't get us, hypoxia would.

There is one way to get conventional lung squeeze. We've seen that it's virtually impossible if you start your descent with your lungs full. If you don't, all bets are off. A scuba diver blacked out for unknown reasons, exhaled passively to resting lung volume (which is much closer to residual volume than to total lung capacity), and then sank through the water. He was rescued, but died of acute pulmonary edema. Another scuba diver was too buoyant on the surface and had to fight to get down. He happened to be a bright engineer, and for his next dive, decided to use Archimedes' principle. He exhaled fully to reduce his buoyancy, and without inhaling, kicked down

to 20 feet. Immediately he became very short of breath and was rushed to the hospital in acute pulmonary edema — a victim of lung squeeze.

If it seems incredible that so short a descent could cause lung squeeze when much longer ones don't, let's look at the arithmetic. We'll assume that when ambient pressure pushes enough blood into the lungs to compress the air down to one half residual volume or less, squeeze is likely, at least for ordinary mortals. The breathhold diver who reaches residual volume at 132 feet will have to get down to 297 feet to reach half of residual volume. However, if he starts his dive after full exhalation, he'll reach half of residual volume at only 33 feet. Our engineer's situation was much worse. I'll estimate his residual volume on land as 1200 cc. However, he began his dive from a head-out immersion posture. With immersion, sea pressure offsets the effects of gravity and pushes an average of 700 cc of blood from the lower body into the chest. This volume shift may be increased by cold-induced vasoconstriction in the skin and by any elastic tension the wet suit exerts. The result is that when he exhaled completely, his actual lung volume was far below residual volume, much sooner. Besides, his lungs had already been stressed by negative pressure breathing during his previous dive, giving him a head start toward squeeze.

There is an unconventional form of lung squeeze. The essence of squeeze is negative pressure — a relative vacuum within the chest, and there are various ways to achieve it. One is to duck a few feet below the surface and breathe through a long snorkel. If you've tried this, you know it's practically impossible. That's fortunate, since if your chest muscles were strong enough to permit breathing down there, your lungs would soon fill up with blood.

The usual way to generate a relative vacuum is to use a maladjusted regulator. A quarry diver had a regulator so badly adjusted that he had to suck hard at the surface. Underwater, breathing dense air, he had to suck very hard. He was a macho type and toughed it out as long as he could. Finally he surfaced, too spent to clear his snorkel, and set off for shore still on the regulator. He lost consciousness but was spotted and quickly rescued. Witnesses said he was huffing and puffing like a steam engine. Pulmonary edema made his lungs stiff and hard to inflate, and fluid in the alveoli interfered with gas exchange. Whether you want to call it lung squeeze is a matter of semantics, but it amounts to the same thing.

Lung Overpressure

Reverse lung squeeze is disastrous to divers. You know it by another name — lung overpressure. The usual cause is a breathhold ascent. It's not necessary to be panicked or even stupid to accomplish this. If your buoyancy is positive, vomiting or convulsing will do. So will laryngospasm, which you might bring on by tickling your vocal cords with vomitus or sea water. Or you might forget to exhale during a buddy breathing ascent, or while engrossed in photographing something that's rising.

Lung overpressure has two evil results. The first is collapse of the great veins, reducing cardiac output. Like the valsalva, this can cause underwater blackout. It can also cause a torn lung.

If the lungs are full to start with, ascent from only three to four feet can tear them. The mechanism has usually been thought obvious — inflation beyond the lung's elastic limit and tensile strength pops it like a balloon. It's not that simple. Lungs removed at autopsy can be inflated more than would be possible within the chest, and not tear. Overpressure, per se, isn't the answer either. If you wrap a dog's chest so that it can't expand, it takes more overpressure to tear the lung. If you wrap the abdomen too, the overpressure required is still greater.

We believe that what tears the lung is neither over-inflation nor overpressure in the usual sense, but rather distortion. When a diver ascends with his airway closed, his lungs will expand normally until his rib cage reaches maximal size. With further ascent, they can't expand outward any more, but can and do expand downward by pushing the non-rigid diaphragm into the abdomen. The lungs aren't designed to withstand this kind of disproportionate stress, so they tear.

When alveolar walls are torn, blood can enter the airways, and coughing up blood is an important clue to the event. More importantly, air can enter the capillaries. It probably does so just after the diver has surfaced and released his breath, since before then the overpressure keeps the pulmonary capillaries collapsed. The air is swept down the pulmonary veins to the left heart, which pumps it out into the aorta. The first branch the bubbles encounter is the pulmonary artery. It's small, and they usually pass by, but if any do enter, you have a heart attack. The next branches are the carotid arteries. They're large, and their openings are at the top of the aortic arch, where bubbles would be expected to migrate. Bubbles swept into them block small branches within the brain, and you have a stroke.

Any symptom that occurs within a few minutes of surfacing and points to the central nervous system must be considered due to air embolism until proven otherwise. To steer any further bubbles away from the carotid openings, standard advice has been to place the patient in a reverse slant, left tilt position. It's doubtful that the purpose is served, since whatever air enters the circulation probably does so all at once. I do favor the position, but for other reasons. Animals embolized experimentally do better if immediately up-ended. Maybe the sudden gravitational boost to pressure in the carotid artery is enough to push the bubbles through. Then too, the slant and tilt reduce the chance that a victim who vomits will inhale the stuff into his lungs, thereby committing involuntary suicide. Most essential of course is to get the victim to a recompression chamber with all possible haste. And if possible, give him oxygen.

If air from the torn lung dissects its way along the bronchial tubes to the central compartment of the chest, you have pneumomediastinum (formerly called mediastinal emphysema). This isn't so bad except in the rare case when there is enough air to collapse the great veins and the atria of the heart; then it will have to be removed. Air in the mediastinum wants out and seeks the path of least resistance, which is usually into the soft tissues in front of the neck. Rub your fingers there, and you'll feel the crackling sensation that's a dead giveaway for subcutaneous emphysema.

If the lung tears at its surface, usually through an emphysematous bleb, air will escape into the pleural space between the lung and chest wall. Alternatively, air from a pneumomediastinum will sometimes break into the

pleural space. Either way, you have a pneumothorax. Ordinarily this isn't life threatening, even if the lung collapses completely, since you can get along on the other one. Still, you'll need medical help to re-expand the lung, and should seek it quickly, as a few cases will progress to tension pneumothorax.

The symptoms of pneumothorax are quite variable. Pain and shortness of breath run from absent to severe. With practice you can learn to recognize a severe case by its physical signs. Percuss the bare chest wall the way a carpenter would to locate a stud. If one side is definitely more resonant than the other, that's suspicious. Then place your ear against each side of the chest in turn. If you hear good breath sounds on one side and little or nothing on the other, you have your confirmation.

It's essential to recognize a pneumothorax if you're going to put a person into a chamber. With recompression, high pressure air will probably enter the pneumothorax space. Upon decompression it will expand and almost guarantee a tension pneumothorax. It will compress the mediastinum and push it against the good lung, compressing it too. That's not conducive to longevity.

Some chambers are supplied with a neat little instrument that will let you detect even a small pneumothorax. It's called the Sonicaid. It hears the noise made by lung tissue in motion. A small layer of air interrupts sound transmission between source and probe, and so announces itself as a pneumothorax.

Pneumothorax in divers is usually provoked by lung overpressure. Non divers get pneumothorax too, when an emphysematous bleb pops. Since there is no apparent provocation, it's called spontaneous pneumothorax. With so many non divers becoming divers, it was just a matter of time until spontaneous rupture would occur underwater. It has! The young man was cruising at 20 feet when he felt a pain in his chest. It wasn't bad, and he kept going. But when he turned downward to check out a hole and the pain shifted to his kidney area, he figured something weird was happening and decided to surface. During ascent he became progressively short of breath and at the surface was in extreme distress. A fast trip to the hospital and a chest tube saved his life. The point we want to make here is that while a provoked pneumothorax usually occurs near the surface, spontaneous pneumothorax will occur at any depth, and ascent will convert it to tension pneumothorax. Had our man been a little deeper, he'd not have survived.

The recurrence rate for spontaneous pneumothorax is substantial, so if you've ever had one, hang up your fins. If that thought is repulsive, you can request an operation that will prevent recurrence.

Since you're all alive and well, we can assume you've learned not to hold your breath during ascent. But don't feel too smug — there are other ways to tear your lungs. Blockage of a small airway will do very nicely. Last August a physician dived the wreck of the U.S.S. Butler off San Clemente Island in 80 feet of water. His bottom time was well within the no decompression limits, and his ascent normal. Upon surfacing, he coughed up blood, nearly collapsed, and had to be rescued. In the hospital, studies indicated obstruction of a small airway. It was not quite complete, so it allowed high pressure air to seep past during the course of the dive, but didn't allow it to escape fast enough during the much shorter time of ascent.

In this case, small airway blockage could not have been predicted, but in many cases it can. Professor Walder, who has extensive experience with British Navy divers, found that many who suffered air embolism had had a cold within the past ten days. Small airway blockage would be expected because infection causes swelling and excessive secretions. The same applies with chronic bronchitis, only it doesn't go away. The most common cause is smoking cigarettes. Spasm of the bronchial tubes traps air, so asthmatics shouldn't dive. Other lesions with air trapping potential include cysts, scars, tumors, and certain congenital defects. People with emphysema shouldn't dive, for even a slight blockage could pop one of those weak walled emphysematous blebs. Some of these conditions show on x-ray, so diving candidates ought to have chest films.

One last way to tear your lungs may be to exhale during ascent. If you blow down to residual volume and hold it there, some of your small airways will be collapsed. Air in the alveoli they serve will have to push open the collapsed segment to escape. The overpressure required may be enough to rupture the alveoli if they're weak (localized emphysema), or if they're overstressed for some other reason. This could explain embolization in students practicing free ascents at the proper rate and blowing bubbles all the way.

Summary

Unless you'd rather dive than hear, don't descend if your ears don't clear easily. Never do a long, strong valsalva. See a specialist if any ear symptom that begins during or after a dive doesn't quit soon. Don't hold your breath while moving up or down through the water. Don't dive with a condition that could compromise the small airways. Assume any distress soon after surfacing to mean air embolism, and arrange a fast run to the nearest chamber.

BREATHHOLD DIVING

Christopher W. Dueker, M.D.

In the long ago days of sport diving when scuba was rare, primitive, and expensive, many people dove with only the air supplied by a deep breath at the surface. Today breathhold diving is still important in training and for underwater hunting. It seems simple, but breathhold involves many complex physiological mechanisms.

Direct pressure changes affect all divers. The scuba diver has the luxury of time to solve any problems with equalization. One form of barotrauma affects only breathhold divers; that is chest (or thoracic) squeeze. The thorax is a semi-compressible air space. At shallow to moderate depths the lungs decrease in volume and body density increases. This doesn't happen in scuba diving since the inhaled high pressure air keeps the lungs expanded. At great depths it is possible to compress the lungs maximally. Beyond that point they act like a rigid air space (such as the sinuses) and proceeding deeper can cause severe lung damage. Formerly, it was believed that the lungs could safely be compressed only to their residual volume (the amount of air remaining after maximal exhalation). Lung compression could be calculated using Boyle's Law, and if the initial lung volume was known, it was easy to determine the depth which would reduce volume to residual. For the average sized person this would permit breathhold diving to about 100 feet. If the initial lung volume was small (less than full inhalation) the allowable depth would decrease. Later investigation showed that diving causes fluid to shift into the chest blood vessels. This shifting effectively reduces the air space volume. Thus, residual air volume decreases and more external compression can be tolerated. This is similar to the almost solidification of the lungs of diving mammals. These changes help explain why well trained persons can go very deep without lung damage. Lung damage can occur in shallower depths if the lungs are not well expanded before starting the dive.

Indirect pressure effects are less of a problem in breathhold diving than with scuba. There is no risk of nitrogen narcosis and very little danger of decompression sickness. It is possible to make enough closely spaced, deep dives to accumulate sufficient nitrogen to cause decompression sickness. This is not a practical danger for general diving, however.

Virtually every breathhold diver envies the capacities of seals, beavers, whales, etc. God has provided these animals with several mechanisms that permits impressive diving. The diving animal has profound heart slowing (bradycardia) and changes in blood distribution which act to reduce oxygen consumption. They also have forms of hemoglobin which store oxygen very efficiently and they can tolerate metabolism without oxygen very well.

Humans also have bradycardia after breathholding and immersing the face. This reflex is sometimes called the mammalian diving reflex, but it also occurs in birds. In some persons the pulse rate is more than halved. The slowing is generally not seen when a snorkel is in use although some investigators report bradycardia in scuba divers. Blood vessels constrict and this offsets the bradycardia so that blood remains normal. Unfortunately, immersion may cause abnormal heart beats (dysrhythmias). Diving deaths have occurred from acute dysrhythmias.

Three major factors affect breathhold duration: lung volume, gas changes, and motivation. It seems obvious that a big breath permits prolonged breathholding (apnea) since this breath provides more oxygen. Actually, the volume itself prolongs breathholding. If extra oxygen is supplied so that a constant lung oxygen tension is provided, a large lung volume still affects breathhold time.

During apnea, lung oxygen decreases and carbon dioxide increases. The consumption of oxygen exceeds the production of carbon dioxide so lung volume slowly decreases.

Very low oxygen tensions will stimulate breathing. But this may not occur until the oxygen has fallen enough to cause unconsciousness. Carbon dioxide is a much more potent stimulus to breathing. Oxygen and carbon dioxide act together to affect breathhold duration. That is, a higher carbon dioxide can be tolerated in well oxygenated lungs.

Breathing oxygen before breathholding will markedly increase the duration of tolerable apnea. Similarly, hyperventilating to decrease carbon dioxide can increase breathhold time. Hyperventilating with oxygen provides the largest apnea: up to three times normal.

Most breathhold divers do some hyperventilating. Unfortunately, this can be very dangerous. When hyperventilating with air, the lung oxygen is little changed, while carbon dioxide falls sharply. During the subsequent breathholding, the slow rise of carbon dioxide may permit apnea beyond the time when low oxygen causes unconsciousness. When this happens in underwater swimming, drowning may ensue. The problem is far greater in diving than in level underwater swimming. During a dive, lung compression raises oxygen tension. On ascent, this tension falls and consciousness may be lost. This is not an uncommon phenomenon among competitive spear-fishermen.

Motivation is difficult to study, but anyone who dives knows how it affects breathholding. The last moment sighting of a fish or abalone definitely lengthens the dive. Similarly, experienced divers can stay longer than neophytes. This is largely due to their familiarity with the sea. They are comfortable and do not exhaust themselves fighting the water. Prolonged breathhold diving does cause physiological adaptations. Lung volumes increase and the tolerance for carbon dioxide increases. These changes require intensive diving and are quickly lost when diving stops.

Breathhold diving is fun and is good training for all divers. It is not simple.

SOME PULMONARY ASPECTS OF DIVING

Paul G. Linaweaver, M.D.

During the course of man's evolution from a primordial aquatic environment to become a terrestrial air breathing mammal, he developed a highly efficient respiratory system. This system, consisting of a semi-ridged yet flexible thoracic cage, respiratory musculature, conducting airways, gas exchanging surfaces, and blood distribution elements is complex in design yet simple in function. The system causes air, with its life-essential oxygen, to enter the airways to be distributed to the gas-exchanging alveoli which are in intimate contact with circulating blood. Oxygen leaves the gaseous phase and dissolves in the blood and carbon dioxide, the byproduct of metabolism, released from the blood it passes into the gas phase and is partially eliminated when the pulmonary gas is expelled. The harder one works, the more oxygen is required, the more CO_2 produced and the need for increased breathing.

I plan to outline some of the anatomy of the pulmonary system and normal movement of gas flow and, finally, how diving affects these parameters.

The human chest is constructed in such a manner that it has sufficient rigidity to protect the vital organs which it contains and yet it provides pliability or flexibility which enables it to function as a bellows in the respiratory cycle. The rigidity results from the bony constitution of the ribs, sternum and spine, and there is pliability because each rib is attached by resiliant cartilage and has movable joints at its spine end. The first seven ribs are attached to the sternum. The cartilage of the next three ribs are attached to the cartilage of the seventh rib. The remaining two ribs, the "floating ribs" have no connection with the sternum or other ribs. The elasticity of the thoracic cage is evident in that when it is compressed in any direction, it always returns to its original position. The chest increases in volume with inspiration. This increase in lung volume normally takes place in three-dimensions, anterioposteriorly, transversely and longitudinally. This three-dimensional increase in volume occurs because the ribs are elevated as a result of the contraction of the scalene and intercostal muscles and because the diaphragm descends during inspiration.

The respiratory muscles act as force generators and include the intercostal muscles with separate sets for inspiration and expiration, accessory muscles of respiration, and the primary inspiratory muscle, the diaphragm. This powerful muscular organ makes up the inferior flexible boundry of the chest. When relaxed it is dome-shaped and flattens during inspiration contraction and flares the lower chest because of its attachment to the ribs. These facts have considerable significance to the diver. Also, the diaphragm has no significant role during expiration.

The airways conduct the air moved by the musculoskeletal bellows to the regions of the lung where gas exchange occurs. The larger airways have cartilage rings supporting their walls, but the distal smaller airways do not and are subject to collapse under certain conditions. Diving is one of those conditions.

The lung is conveniently divided into several capacities and volumes. These are not anatomical spaces but functional. For our purpose, the Functional Residual Capacity (FRC), holds the most important considerations regarding diving, since volumes below FRC involve the closure of airways.

The static volumes of the lung are functions of the elastic characteristics of the chest wall and muscular power of respiratory muscles. The dynamic changes in lung volumes or ventilation are a function of muscular power; elastic properties of lung and chest wall as well, but airway competence is an important factor. These can be tested by certain pulmonary function tests. We find that inspiration is limited by power and that expiration is limited by airway competence is an important factor. These can be tested by certain pulmonary function tests. We find that inspiration is limited by power and that expiration is limited by airway resistance and elastic recoil of the lung. Beyond a certain point, expiration cannot be affected by increasing muscular force or power and is effort independent, and is entirely dependent on lung recoil.

Just how much ventilation is required by an individual under various conditions? The amount of ventilation required is closely related to the oxygen requirement of any activity, the ventilation is roughly 20 times the oxygen consumption unit, usually expressed in liters per minute.

Studies performed on finned divers show a very high rate of oxygen consumption as compared to surface activities. **This fact should be emphasized to student divers.** The following table illustrates how hard aquatic activities are in terms of oxygen usage and how much ventilation is required to maintain normal CO_2 levels.

Activity	O_2 Consumption L/m	Ventilation L/m
Sitting	0.35	7
Standing	0.40	8
Walking 4 MPH	1.2	24
Running 8 MPH	2.0	40
Fin Swimming 0.8 Kts	1.2	24
Fin Swimming 1.2 Kts	2.5*	50
Fin Swimming 1.5 Kts	3.5*	70

*very hard work

Now, what happens to the lungs when a diver is in the water?

At U.C. San Diego, I studied the effects of immersion on pulmonary function. There are positive effects on pulmonary anatomy including thoracic shape, diaphragmatic position and configuration. In the immersed canine, because of pleural pressure changes, compression of the thorax decreased functional residual capacity to 55% of the baseline value. The change in diaphragm position results in a 2.8 fold increase in the ability of inspiratory muscles to exert force (Pmus). In spite of this increase in muscular contractility, the Tidal volume decreased during emersion, averaging only 83% of the surface value. Hence, immersion was associated with a marked stiffening of the respiratory system. In all, there were three factors effecting pulmonary functions during immersion: the FRC reduction, the change in the

thoracic configuration and diaphragm configuration, and the stiffening of the respiratory system.

We know that changes in lung volumes change airway resistance to air flow. We also know that airway size is dependent on lung volume. We, therefore, looked at airway configuration with tantalum. There was a marked decrease in length and most importantly the diameter of the airway. Many airways simply closed off. The resistance to air flow is also a function whether flow is smooth and laminar or turbulent, which requires the square of the power to move a certain volume. This means muscle power and elastic recoil power.

The amount of flow is also a function of work rate, as we have previously mentioned. Density or depth of diving has a direct effect on airway resistance. Studies on divers show that the ability to ventilate decreases with depth. Thus, a diver should be warned that the deeper he dives the less work he can expect to perform. It has been widely taught from my own initial training to present, that if a diver works too hard at depth, he can "over-breathe his regulator" which could lead to panic. Divers should be taught that he can **"over-breath his pulmonary system"** by working too hard at depth. Of course, regulators can add resistance to both inhalation and exhalation especially if not well serviced. Man's own airways and elastic recoil are the primary limitation factors to underwater work.

In closing, I would urge all instructors to emphasize that diving activities can be safe if the diver knows his limitations. These are many including thermal limitations, oxygen limitations, and decompression limitations. The limitation I emphasize today is that his work level is limited by his cardiopulmonary anatomy and function which are altered by the water environment. In simple words, you can't work as hard underwater as you can on land and your ability decreases the deeper you go.

NEW TECHNIQUES IN EXTERNAL CARDIAC COMPRESSIONS

Aquatic Cardiopulmonary Resuscitation

By Nico F. March, Richard C. Matthews, M.S.

Despite the 8,000 drownings that occur annually in the United States, procedures for aquatic first aid are currently limited to mouth-to-mouth resuscitation, while cardiopulmonary resuscitation (CPR) must be delayed until the victim is transported to a solid surface for conventional closed-chest cardiac compressions. We discuss techniques of positioning a victim on the rescuer's chest and initiating CPR, on site. Respiratory support was provided with a slightly modified scuba regulator, and procedures were tested on an instrumented aquatic CPR mannequin in the water and anesthetized dogs on land. Most results were within specified criteria for successful CPR. (*JAMA* 244:1229-1232, 1980)

WHEN the heart loses its capacity to provide adequate systemic perfusion, cardiopulmonary resuscitation (CPR) administered correctly can sustain vital functions.[1] This assumes a rescuer begins CPR within four minutes and maintains adequate ventilatory support as well as sufficient sternal compression and duration to guarantee cerebral blood flow.[2-4] Cardiorespiratory arrest that occurs in aquatic situations results from numerous factors, including trauma, drowning,[5] or asphyxia.[6]

Unfortunately, the American Medical Association, the American National Red Cross, and the American Heart Association currently lack description of acceptable techniques for CPR without back support in the water.[7,8] In some aquatics-related accidents, the time delay involved in transport of a victim to shore, boat, or other conveyance may prove fatal or seriously impair successful recovery.[9]

The purpose of this study is to validate techniques that use a pressure-limited, safety-designed scuba regulator as an emergency ventilatory device, and the rescuer's chest as a backboard for external cardiac compressions, as possible methods for sustaining victims of cardiorespiratory arrest in aquatic environments.

Materials and Methods

These studies used a Laerdal Recording Resuscitation mannequin, redesigned and instrumented for submersion in water. The mannequin measures 173 cm in length, with a dry weight of 52 kg. When immersed, the mannequin's weight is slightly negative, simulating dead weight. Statham transducers (P23-ID) were connected to airway and compression mechanisms to record proximal airway pressure and sternal deflection, respectively. Data were recorded on a two-channel polygraph recorder (Brush-mark 222). Correct sternal positioning was measured and recorded manually on the polygraph strip. Expired volumes were taken from one Laerdal polyethylene lung (model No. 040402, 1,000 mL) and recorded in milliliters per minute, using a 6-L water-filled spirometer (W.E. Collins).

Fig. 1 — Emergency regulator resuscitation, using factory-calibrated, saftey-designed scuba regulator.

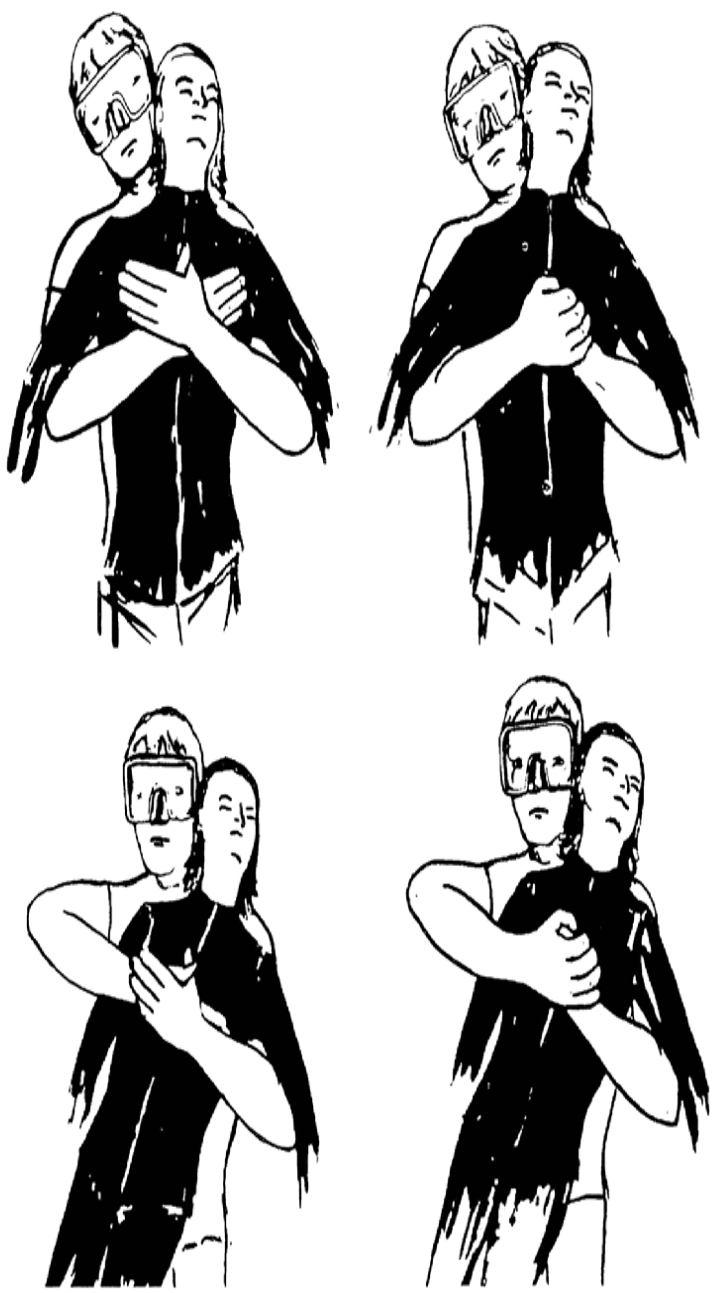

Fig. 2 — Aquatic cardiopulmonary resuscitation (CPR). Top left, Under-arm body positioning for aquatic CPR using butterfly hand position. Top right, Under-arm positioning using clenched-fist hand position. Bottom left, Cross-chest body positioning for aquatic CPR using butterfly hand position. Bottom right, Chest-chest body positioning using clenched-fist hand position.

A pressure-limited, safety-designed scuba regulator was used to provide positive-pressure ventilations, or IPPB (Fig. 1). The scuba regulator (SCUBAPRO Air Inhalation Regulator [AIR] 1) was factory calibrated to deliver peak inspiratory pressures not exceeding 40 mm Hg and 780 ± 20 L/min airflow from the mouthpiece on complete depression of the trigger or "purge" mechanism. These criteria were established by the National Research Council, Standards for Cardiopulmonary Resuscitation and Emergency Cardiac Care.[7] Modifications to the SCUBAPRO AIR 1 demand regulator have negligible effects on breathing resistances, making this a feasible alteration to all high-flow regulators.

Conventional scuba regulators were not used during these studies, owing to high inspiratory pressures developed by some and lack of sufficient pressure or flow in others. Peak inspiratory pressures exceeding 50 mm Hg in closed systems could possibly rupture lungs,[10] while pressures below 10 mm Hg might not provide adequate ventilation.[11]

Emergency regulator resuscitation (ERR) was administered by placing the regulator within the mannequin's mouth, hyperextending the neck to achieve a patent airway, and fully depressing the trigger mechanism for 1 s per ventilation. Full inflation of the lung is accomplished without occluding the nose or sealing the regulator completely within the mouth, owing primarily to the high flow rate (780 ± 20 L/min) of the SCUBAPRO regulator.

Six persons, all with previous scuba diving experience, were instructed in the performance of ERR and external chest compressions on the mannequin while stationary and out of water, after which their ability to perform these techniques for a period of 15 minutes was measured. All subjects were then tested while administering aquatic CPR and supplying propulsion in the water with the mannequin as the victim. All rescuers wore wet suits and back or vest flotation devices during aquatic trials. Several body and hand positions were attempted during these studies for correct sternal positioning and relative ease of applying compressions. The two body positions were an under-arm and cross-chest maneuver, while hand positions include the butterfly and clenched fist (Fig. 2). Laboratory tests were conducted with audiofeedback signals to rescuers, indicating correct position, compressions, and ventilations. All aquatic trials were single-blind and simulated emergency situations for rescuers with no audiofeedback indicators.

Results

Laboratory results of compression depth vs time (Fig. 3, top) show a slight decline from 4.4 to 3.0 cm, with all except one remaining within acceptable limits (i.e., 3 to 6 cm) for compression depth. Aquatic trials produced compression depths of up to 4.5 cm, with all persons attaining slightly more compression in the water than on land. Compression rate and duration, measured as sternal deflection per minute, remained relatively constant — between 45 and 55 deflections per minute during the laboratory sessions with audiofeedback — but decreased during aquatic trials to between 34 and 48 deflections per minute with longer compression durations (Fig. 3, bottom). Ventilatory rate was constant in the laboratory at 8/min and fluctuated from between 6 and 8/min during aquatic trials (Fig. 4, top). Expired volumes from

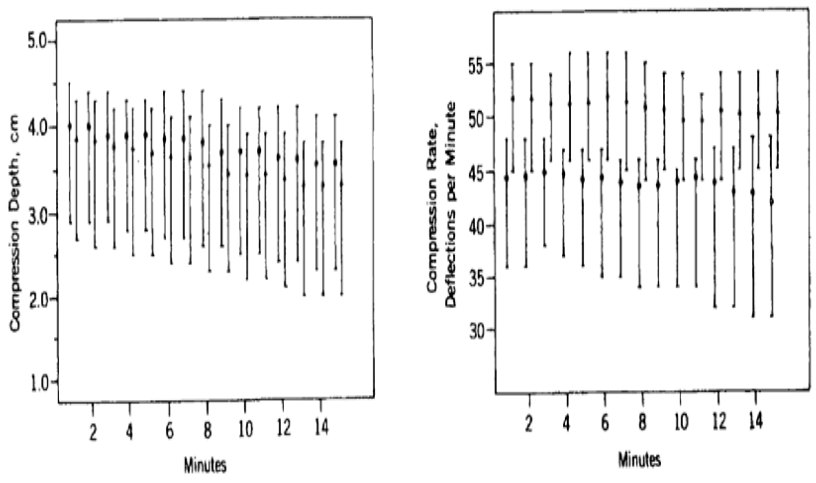

Fig. 3 — Laboratory test results of compression vs. time. Left, Compression depth generated by rescuers on submersible recording resuscitation mannequin while administering aquatic cardiopulmonary resuscitation (CPR). Decay in sternal deflection is greater in laboratory tests than in aquatic trials. Right, Compression rate maintained by rescuers administering aquatic CPR to simulated victim. Rate in laboratory was slightly higher, owing to audiofeedback system used during laboratory sessions. Triangles indicate laboratory trials; circles, aquatic trials.

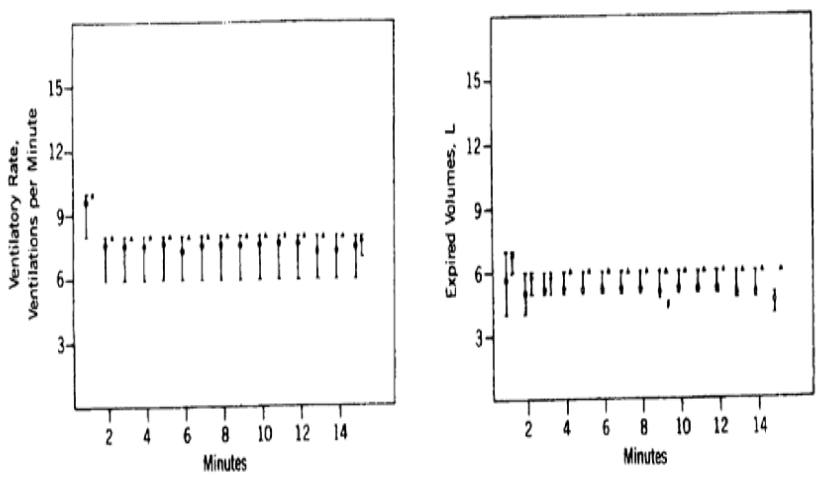

Fig. 4 — Ventilatory rate (left) during aquatic cardiopulmonary resuscitation (CPR) using safety-designed scuba regulator for positive-pressure ventilation. Some variation occurred in pool during first three minutes, owing to unfamiliarity of mannequin to some rescuers. Expired volumes (right) from simulated 1,000-mL lung during administration of aquatic CPR. Full inflations of lung were achieved only if mannequin's neck was hyperextended, maintaining patent airway. Triangles indicate laboratory trials; circles, aquatic trials.

laboratory tests were between 5 and 6 L/min, while pool sessions produced volumes from betwwen 4 and 6 L/min (Fig. 4, bottom). Rescuer-supplied propulsion shows distances traveled while administering aquatic CPR to be between 365 and 560 m/15 min while in the pool (Fig. 5).

Comment

Today there exists no medically proved technique for CPR applicable in water without firm support for the victim's back. Our studies suggest that these described methods could be used successfully if rescuers have access to a pressure-limited scuba regulator, and adequate oxygen or air supply, and flotation equipment commonly used by scuba divers.

Sternal deflection depth ranged from approximately 2.0 to 4.5 cm, with most values falling within established limits for CPR. The compression duration rate ranged from 34 to 55 deflections per minute, even over a

15-minute interval; again, most were within the published guidelines. Ventilatory rates between 6 and 8/min, and expired volumes from the simulated lung illustrate that the pressure-limited scuba regulator can be used safely for IPPB in an emergency aquatic situation.

By using a high-flow regulator, occlusion of the nose, mouth, and exhaust ports was unnecessary. As flow increases, so does resistance to flow. The exhaust ports and open nose impose sufficient resistance to high flows to cause inflation of the lungs, without the possibility of lung overexpansion.

Comparing the ERR device against a constant-volume respirator (Harvard model No. 607) and a demand-valve resuscitation respirator (Elder oxygen valve model No. 34-103) in ventilating anesthetized, aspirated dogs, we found that the modified SCUBAPRO regulator maintained blood gas values as well as the demand valve resuscitator. Taking into account that both the ERR device and the demand valve resuscitator maintained successful ventilation in the experimental animals, and that the scuba device is capable of full operation when immersed in water while the latter is not, we believe that a modified safety-designed scuba regulator could be an invaluable tool available immediately at the scene of many aquatic accidents.

This study shows that a trained person may successfully sustain a victim of cardiorespiratory arrest at the surface of an aquatic environment, even while moving toward advanced life support for periods of at least 15 minutes.

Data from this research indicate that these described techniques are applicable in many instances of aquatic emergencies. Because of the tremendous interest in aquatic environments for recreational and occupational pursuits, accidents are inevitable. Rapid response by a properly trained and equipped person could determine the difference between successful first aid and recovery or certain death.

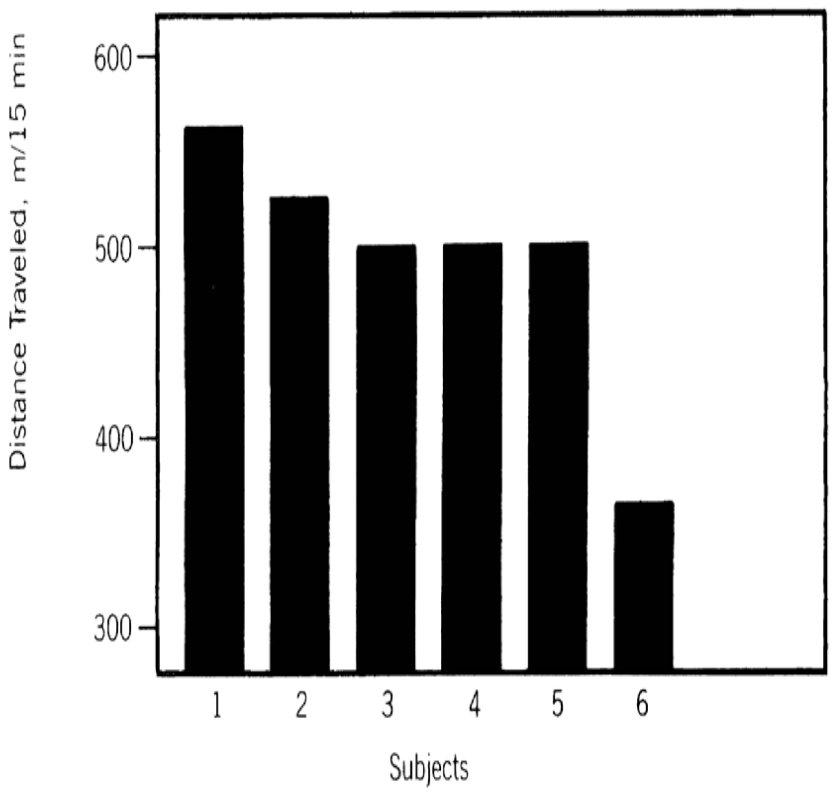

Fig. 5 — Distance traveled by single rescuer while administering aquatic cardiopulmonary resuscitation to submersible, recording resuscitation mannequin. All rescuers wore full scuba gear while performing closed-chest cardiac massage and administering emergency regulator resuscitation in pool.

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PHYSIOLOGY OF AN AQUATIC ACCIDENT

Nico F. March

One of the most prevalent types of accidents occuring in aquatic environments today is drowning, defined as the process by which air breathing animals or men succumb on submersion in a liquid. This can be attributed to numerous causes where the upper airway, bronchial tree or alveoli are blocked and gas exchange cannot occur.

In this short discussion we'll address the physiology and pathophysiology of this type of accident, which just happens to comprise a large percentage of the diving deaths and accidents occurring today and expected to occur in the future.

There are actually four types of drowning. The first is wet drowning, where the victim ingests large amounts of fluid. The second is dry drowning, where the glottis closes off the respiratory tract causing death by asphyxia. The third is secondary drowning, where the victim develops adult respiratory distress syndrome — or ARDS — up to 72 hours after a drowning or near-drowning episode. And the fourth is immersion or sudden death, due to vagally induced cardiorespiratory arrest in very cold water.

First, lets consider the effects on the respiratory system:

When an experimental animal or man is submerged, its initial reaction is to voluntarily hold its breath. After a short period of apnea, the CO_2 in the blood and tissues increases, stimulating the medulla, or respiratory center, and causing the animal to take a breath. Water may be swallowed at this point rather than being inhaled directly into the lungs. This is seen in many instances of human drownings or near-drownings. Involuntary water swallowing continues during inspiratory efforts, filling the stomach with a combination of air and fluid. This usually acts as an emetic, causing regurgitation of the stomach's contents.

This fluid and vomitus then contacts the mucous membranes of the larynx, which in turn, generates a characteristic reflex, which is referred to simply as laryngospasm. Physiologically, the laryngeal muscles contract, occluding the airway, causing obstructive asphyxia. Following a period of asphyxia, these muscles relax, allowing the airway to reopen. With this relaxation of the laryngospasm, and reopening of the airway, the terminal phase begins.

These involuntary breathing movements, or gasps, may continue even after the circulation has stopped. If the victim is still submerged, aspiration of more fluid into the respiratory tract may occur, thereby decreasing vital gas exchange between alveolar spaces and the blood stream, and the likelihood of the victim's survival.

In cold water or hypothermic drownings, "diver's reflex" or bradycardia causes an inhibition of the respiratory center, which may prevent water from entering the lungs.

It also causes the heart rate to decrease to approximately 10-20 beats per minute, depending on the temperature of the water and the thermal insulation available to the victim. Peripheral vasoconstriction reduces blood flow to the skin, muscles and other organs resistant to damage from anoxia, while

shunting available blood to the brain and heart. These effects are seen in warm and cold water, while most documented cases of survival without resulting cerebral damage occur in water with temperatures below 63 degrees Fahrenheit. The effects on the circulatory system depend primarily on the type and amount of fluid introduced into the respiratory tract.

In sea-water aspiration, the fluid is more concentrated than the salt content of the blood. Therefore, fluids are drawn from the bloodstream into the lungs, causing a rapid hemoconcentration, as well as severe pulmonary edema. The resultant loss of blood volume then causes hypotension, or abnormally low blood pressure, bradycardia, circulatory collapse and eventually death. In experiments by Gordon on dogs, cats and rabbits, sea-water drownings did not result in ventricular fibrillation, but death usually occurred within three to six minutes following immersion. The blood pressure response of all animals tested shows an initial rise with deep respirations, followed by a plateauing of the pressure and a gradual decrease to zero after the last terminal cases. The blood pressure response can be directly correlated to an electrocardiogram. This pattern is characteristic of all species tested during sea-water immersion.

In fresh water drowning, Gordon submerged horses, cows, pigs, sheep, dogs, cats, monkeys, rabbits, rats and guinea pigs. He found one of three responses in blood pressure. In one group of animals, there was an initial elevation of blood pressure, then a plateau and a marked decrease in blood pressure. In this group, there was also simultaneous and terminal ventricular fibrillation. The second group showed a response similar to the sea-water submersion, with no instances of ventricular fibrillation. The third group had a similar rise and plateau, followed by a gradual fall in the blood pressure. However, these animals had short intervals of decreasing blood pressure, which was found to be simultaneous with transient, brief, spontaneously reversible V-fibb. Gordon found that there was a definite species specificity for ventricular fibrillation in fresh water drowning, and that there is a direct relationship between the size of the animals and the incidence of permanent V-fibb with the larger animals being most susceptible and the smaller ones being most resistant.

The ventricular fibrillation seen so frequently in these animal experiments, has been reported in only a few instances of fresh water drowning in humans. It seems more likely that a combination of anoxia, acidosis and local myocardial changes would play a greater role in the production of V-fibb in humans rather than the water absorption theory as postulated by Gordon. Furthermore, other serious complications may occur if fluids aspirated into the lungs contain chemicals or other irritants which wash away the surfactant from within the alveoli. The surfactant keeps the air sacs from collapsing, and any alteration or destruction may lead to large areas of non-ventilated alveoli, through which pulmonary blood flows. In these cases there is usually a high incidence of pulmonary edema and aspiration pneumonitis, and prolonged respiratory support with high oxygen concentrations, as well as drug therapy is indicated.

The next question is, what can be done to safely and expeditiously rescue a drowning victim from a life-threatening aquatic environment? Since the major pathophysiologic changes of drownings result from anoxia, in most instances

experts agree that the immediate treatment of the near-drowning victim is imperative, even before one reaches shore, a boat, an underwater habitat in saturation diving, or some other conveyance. In the past, very little thought was given to medical emergencies occurring in the water and their possible prevention. Now, however, as one can clearly see by glancing through the available literature and recent accidents such as those occurring on the Oil Rig Alexander Kielland in the North Sea, the problems are evident, lives are at stake, and some viable solutions are being proposed. We have just completed experiments at the University of California, San Diego, School of Medicine, to determine the feasibility of a single rescuer administering CPR and emergency airway ventilation to an unconscious or incapacitated victim while in the water.

I must preface this and tell you that due to limited government funding available for this study, we were forced to work primarily on the surface and from the sport diving aspect first, but have just completed the preliminary experimental protocols to continue with underwater and saturation research. Other investigators at the University of Maryland, Institute for Emergency Medicine, have also carried out concurrent studies using cadavers rather than animal models; and much of the preliminary findings look promising for the future of aquatic CPR. We feel that these techniques, or slight modifications thereof, may prove invaluable in rescuing and resuscitating divers, swimmers and numerous other aquatics-related accident victims in cardiac or cardiorespiratory arrest. Until now the only accepted methods of emergency life support in the water are mouth-to-mouth and mouth-to-nose resuscitation, while conventional CPR must be delayed until the victim is either transported to shore or a boat or some other conveyance which will support the victim's back with a firm surface, as well as ample room for the rescuer to perform standard closed-chest compressions.

In October of 1978, George Arnoux of Comex Diving, Marseilles France, introduced a fouled or unconscious diver recovery procedure for use in all commercial diver operations. These procedures suggest mouth-to-mouth resuscitation and closed-chest cardiac massage be attempted immediately if the victim is to survive. The methods suggested for chest compression include a head-thrust technique, whereby the rescuer grasps the victim from the front and utilizes the top of the head for chest compression; and an alternative knee thrust technique, where the rescuer grasps the victim's shoulder straps or harness and applies compressions with the kneecap. Unfortunately, studies conducted in the early 60s by Dr. Archer Gordon of UCLA, indicate that little if any blood flows to the brain during CPR in vertical positions while the victim is suspended in air. This was attempted previously as a technique to resuscitate utility linemen on telephone poles, suffering from heart attack or electrocution.

Our studies utilized a very special recording resuscitation mannequin redesigned for submersion and weighted to simulate an actual diver in the water. The mannequin has pressure transducers which monitor airway and lung pressures, chest compression depth, rate and duration, as well as information pertaining to respiratory support.

Respiratory support was provided by using a safety-designed, pressure

limited scuba regulator for artificial respiration. This device has tremendous potential in situations such as drownings, surf or rough water accidents, scuba rescues, or any other situation — wet or dry — that requires respiratory support for a victim. We have conducted tests on various regulators and procedures at the University of California, San Diego School of Medicine, and have developed design specifications which allow a high flow regulator to be calibrated to deliver emergency respiratory support to an unconscious victim without the possibility of lung over-expansion or embolism. We call this technique Emergency Regulator Resuscitation, or ERR, and have followed criteria set forth in the Journal of the American Medical Association which means we adjusted these regulators to deliver 40 millimeters Mercury pressure, or approximately .7 psi at the mouthpiece. This is done only upon complete depression of the purge mechanism. The air flow from the mouthpiece is 780 ± 20 liters per minute, or about 25 cubic feet per minute. Very little blow-by goes out of the exhaust ports until the lung is filled or some other obstruction limits the flow into the lung. This, along with the fact that the nose and mouth don't necessarily have to be sealed, allows a safety-valve, or open-system effect, permitting the lungs to expand, but not overexpand. The mechanical limiting of the purge mechanism, or adjustment of the distance traveled between poppet and seal in the second stage is the only way, at this point in time, to adjust some regulators to these criteria. Regrettably, not all regulators can be modified to meet these specifications. Some regulators deliver pressures and flow rates too high for safe resuscitation, while still others don't create sufficient flow or pressure to inflate even the smallest victim.

We have conducted animal experiments to see if a modified scuba regulator could maintain a drowning victim as well as a standard resuscitator utilized in hospitals and ambulances. If it is possible to use a scuba regulator for artificial respiration, it might save valuable minutes in aquatic situations where mouth-to-mouth resuscitation might be impractical or unappealing.

We compared a standard hospital resuscitator, the Elder oxygen valve, to a SCUBAPRO AIR 1 regulator. During our experiments we measured three different values of the blood which determine the well-being of the animal. This is referred to as our blood gas data. The first variable is the oxygen content, or O_2. The second value is the carbon dioxide, or CO_2, and the third is the pH, which tells us about the acid-base balance of the animal. In the beginning, we ventilated the animals with a Harvard volume respirator pump until normal values for all three variables were obtained. We then hyperventilated the animals for 10 minutes with the scuba regulator, and another 10 minutes with the hospital type resuscitator. Following a return to baseline values on the volume respirator, we instilled seawater, and disconnected the volume respirator. Following aspiration, we again connected the volume respirator, which did virtually nothing to reverse the hypoxia, or lack of O_2, and the severe acidosis, seen by the dramatic drop in the pH to 7.1 and the accompanying build-up of carbon dioxide. The SCUBAPRO AIR 1 was then utilized for resuscitation and it performed equally as well as the Elder Oxygen valve for restoring the blood gas values to near normal. Finally, to be quite sure it was the regulators that were responsible for the marked increase,

we again connected the volume respirator, and the values reverted to life-threatening levels.

Previous studies have shown that, in certain circumstances, resuscitation is feasible 10, 22, and even as much as 45 minutes following immersion without permanent brain damage. This is due presumably to the "diving reflex" better known as bradycardia, which can occur in cold or frigid waters. Our experiments, and blood gas data obtained during these studies, confirms that immediate resuscitation using a mechanical device for artificial respiration will improve blood gas values following aspiration or near-drowning. Unfortunately, the volume respirator and the hospital type resuscitator have limited applicability during on-site aquatic emergencies, which involve the management of an unconscious or incapacitated victim suffering from heart attack or drowning. However, as this study shows, a scuba-type regulator, factory-calibrated and properly maintained, can safely deliver life-support in and around the water, without the possibility of lung overexpansion occurring. The other types of respirators can't function in these situations due to design or material characteristics which preclude immersion in water. More importantly, since immediate response and workability are the key points that usually determine survival rates, we feel very strongly that properly adjusted and calibrated scuba regulators represent an attractive alternative to rescuers at the site of an aquatic accident. Furthermore, this procedure provides an alternative to mouth-to-mouth resuscitation, is simpler to perform while in the water, and is much more aesthetically appealing.

Once we had solved the problem of airway resuscitation, we began experimenting with cardiac resuscitation. Several hand and body positions were attempted during our studies for correct sternal positioning and relative ease of performing CPR. These were based on previous methods attempted by Dr. Charles Guildner at Johns Hopkins University. The body positions utilized were an under-arm maneuver, and a cross-chest maneuver. The hand positions include a butterfly or interlocking thumb, clenched-fist position.

Data generated on the mannequin regarding compression depth showed a slight decline from 4.4 to 3.0 centimeters during laboratory trials, while all but one subject remained within the accepted criteria for sternal compression, which is considered anywhere between 3 and 6 centimeters for effective compressions. Aquatic sessions provided compression depths of up to 4.5 centimeters and all rescuers maintained slightly higher compression depth in the water than on land. Compression rate, measured as sternal deflections per minute, were relatively constant during the laboratory trials due presumably to an audio-feedback system used by the subjects while in the laboratory. The compression rate decreased slightly during aquatic sessions, due to the fact that all rescuers were required to compress, ventilate and transport the victim with no audio-feedback signals to assist them.

Data generated on the mannequin regarding ventilations show a constant respiratory rate was achieved in the laboratory, due again to the audio-feedback system. While in the water, the amount of ventilations fluctuated slightly. Expired volumes from the simulated lung show volumes from 6 to 7 liters during the laboratory sessions, while aquatic sessions produced volumes between 4 and 6 liters per minute. This of course is

proportional to the size of the lung utilized in these studies.

Rescuer supplied propulsion, and the distance traveled by each rescuer during the administration of aquatic CPR showed distances between 375 and 560 meters over a fifteen minute interval.

Besides conducting experiments in the laboratory and pool situations, we have also carried out the studies in open water and surf conditions with the San Diego City Lifeguard Service. We have also carried out detailed studies with the U.S. Coast Guard, Special Medical Operations Branch in preparing an aquatic CPR program for their emergency medical technicians and other rescue personnel.

As in the case with most medical research, further studies must still be carried out to substantiate our data regarding blood flow and pressure developed underwater and in head-out immersion during aquatic CPR. Some of this preliminary work has been carried out at the University of Maryland, Institute for Emergency Medical Services, under Drs. Mark Bradley and Roy Meyers.

On a more positive note, I am pleased to say that the standards for CPR and emergency cardiac care, established in 1974, have been revised in 1980 to reflect our findings regarding CPR in the water. Simply stated:

> In 1974, under special resuscitation situations it says, "External cardiac compression should never be attempted in the water, because it is impossible to perform it there effectively." And in the new 1980 standards it says, "external chest compression should not be attempted in the water unless one has had special training."

We are now in the process of preparing a reference and training manual which will be utilized as the basis for a 24 hour aquatic CPR training course.

This course will be offered through the PADI International College in San Diego and will include lifeguard and Coast Guard procedures as well as scuba and underwater rescue techniques.

In conclusion, we have shown that an incapacitated victim can be maintained for a period of at least 15 minutes, and even transported with advanced life support. Data collected during our experiments indicate the described methods are applicable in many cases of aquatic emergencies, and may be modified to incorporate underwater or other rescue situations as well.

Because of the tremendous growth and interest in aquatic or marine environments for both recreational and occupational pursuits, accidents are inevitable. Therefore, rapid response by a properly trained and equipped individual could mean the difference between successful first-aid and rescue or certain death.

MEDICAL QUESTIONS IN DIVING STUDENT SELECTION

By Christopher W. Dueker, M.D.

At the recent PADI Diving Physiology Seminar, several questions were raised regarding the medical standards for diving. These concerns are appropriate since maximum safety in diving requires careful evaluation of those who go diving. Some diving accidents involve people who should not have been participating in the sport. Diving instructors must carefully evaluate potential students, and each diver needs to consider his own health before making a dive.

The diving instructor has the responsibility for determining fitness for diving. To do this he relies on guidelines from his instructional agency, physician reports, and his knowledge and experience. Diving organizations vary in the rigidity and completeness of their standards. The prudent instructor does not violate standards, but will still have to deal with issues in student selection that are not specifically discussed.

Candidates are often referred to physicians for medical clearance. Many physicians are not aware of the specific health requirements for diving. The instructor should provide a brief summary of requirements or refer physicians to standard diving medicine texts or to diving medical specialists for consultation. Finally, the instructor must decide if a candidate should be permitted to become a diving student. The instructor's knowledge guides him in requiring chest x-rays (when they may not be required by "Headquarters") or in declining a candidate with a history of asthma without symptoms for three years.

A few principles govern the establishment of diving medical standards. It is much easier to understand these principles than to try to memorize facts about every medical condition.

1. Diving is a potentially vigorous sport.
2. Diving often is done in remote areas.
3. Diving is done in water, under increased atmospheric pressure.
4. Since health does not remain constant, reevaluation is essential.

Almost everyone has the physical ability to snorkel in a pool or very calm waters. This certainly doesn't mean all are fit for open water diving where routine or unexpected demands require great energy expenditures. Any health problem that limits activity makes ocean diving unwise.

In general, these health problems involve the cardiovascular and respiratory systems. This would include coronary artery disease or valvular heart disease. Persons with angina are easily eliminated, but what about the person who had a myocardial infarction (heart attack) and now has no angina? This candidate must have had coronary disease and most likely still has it. In rare cases the person may be very fit, but this determination requires careful evaluation by a medical specialist. Respiratory disease typically reduces capacity and should not be present in divers. As will be discussed, lung defects are especially dangerous underwater.

Divers who go to remote areas must not be dependent on drugs, equipment, diets, or medical supervision that may not be available. As an example, poor

vision is usually not disqualifying for sport diving. However, the travelling diver would need to bring spare visual equipment.

Water is a welcome environment, but it is still an alien one for divers. The diver must always be alert and capable of escaping unexpected trouble. The requirement for any drug that reduces alertness makes diving unsafe! These medicines include sedatives, tranquilizers, and antihistamines. Either the drug or the condition for which it is used may be the problem. A psychosis requiring major tranquilizers is not safe in diving. Mild hypertension (high blood pressure) which would by itself not be disqualifying is sometimes treated with tranquilizers — these would be disqualifying.

Seizure disorders (epilepsy) which cause convulsions with loss of consciousness (grand mal) or lapses in consciousness (petit mal) would make diving very dangerous. The damage that would result from an underwater seizure is so great that **even persons with well-controlled epilepsy should not dive.** This particular disorder may be hard for an instructor to deal with if there has not been a seizure in some time. Nonetheless, if a specialist considers the person to have a seizure disorder, that person should not dive. There are special cases (infant febrile seizures, some post trauma seizures) that may permit diving. This is not a decision for the instructor to make without excellent medical counsel.

Breathholding and immersion affects the circulatory system. Marked pulse slowing occurs and dysrhythmia (abnormal heart beat rhythm) is not uncommon. Most of these are harmless. However, persons with heart disease are prone to dysrhythmias that seriously disrupt the heart's function. This is another reason that people with heart disease should not dive. It appears that the incidence of diving accidents from heart disease is increasing.

Heart disease is not always easy to detect. The physician uses the medical history and examination plus laboratory tests such as x-ray and electrocardiogram in the diagnosis. The value of stress (exercise) electrocardiograms in the detection of asymptomatic coronary disesase is hotly debated. It may be of value in the sedentary middle aged person who wants to enter the vigorous, wet world of diving.

Ear problems secondary to pressure changes are the most common diving medical maladies. Sinus problems often accompany them. In diving, ear abnormalities can result in troubles varying from ear pain to deafness or incapacitating vertigo (dizziness and loss of orientation) that causes drowning. Thus, a thorough examination is essential. This is one area where apparently minor defects, such as an unhealed ear drum perforation, can be very serious. Physicians not familiar with diving physiology may not appreciate this. Even the erudite *British Medical Journal* suggested the use of ear plugs in external ear infections in divers! The examiner will note the defects; the instructor must be cognizant of the defect's importance when connected with diving.

Lung disease is very dangerous in diving, but not only because of its limitation of exercise tolerance. Abnormal lungs are prone to overexpansive injuries. Any lung rupture underwater has a catastrophic potential. Many people have had a spontaneous pneumothorax. Unfortunately, these persons should not become scuba divers since the chance of recurrent pneumothorax is fairly high. Chronic lung diseases (asthma, emphysema, bronchiectasis)

increase the risk of lung rupture as do structural defects such as bullae or broncholiths. Sometimes childhood asthma may heal completely, but this requires medical evaluation, not just the diver's opinion. X-rays are useful in the initial diving examination as they may detect symptomless defects. Fortunately, few screening x-rays show defects. But since the risk from any defect is enormous, the x-rays are worthwhile.

Unfortunately, the area of greatest risk in diving is hardest to evaluate; i.e., emotional stability. A history tends to detect only the most obvious of problems. Further hints can be obtained from patterns of drug or alcohol use or difficulties in driving. Astute observation of a person may help as well. The instructor carefully watches each student during all phases of instruction. A student who is not performing well in the pool should not be taken to the ocean.

Health does change. Having passed a diving physical does not confer lifelong suitability for diving. This should be understood by all instructors, students, and divers. Each person should consider his health before making a dive. In a class this is part of the instructor's responsibility. Respiratory infections or nasal congestion may temporarily make diving unwise. An acute illness, such as pneumonia, may make diving unsafe. Some medicines being used may cause dangerous sedation. Temporary illnesses can include emotional disorders such as depression or the aftermath of alcoholic indulgence.

Pregnancy is a very special transitory state lasting 40 weeks. For years, diving was part of a man's world; and even now we really don't know enough about women and diving. Investigations on diving in pregnancy often yield conflicting results. There is potential danger to the fetus from decompression sickness in the first trimester, even if the mother has no symptoms of "bends." This, however, remains unproven. Physical problems of size change may make diving difficult in late pregnancy. Since pregnancy is temporary, and since the fetus is so valuable, it seems prudent to be very cautious in diving. As always, sport divers should make only "no decompression" dives. But the pregnant diver needs to know that intravascular bubbles form even in some "no decompression" dives and that some scientists believe that these can harm the fetus.

This brief article does not answer questions about all health conditions. The diving instructor is not expected to know everything about medical examinations. The instructor's duty is to recognize that safe diving depends on healthy divers. Careful student evaluation will make instruction less worrisome and more enjoyable for all.

Q & A FROM PHYSIOLOGY IN DEPTH SEMINAR

By Charles V. Brown, M.D.

Q. What's the connection between the cerebrospinal fluid that bathes the brain and spinal cord and the perilymph fluid of the inner ear? How does it tie in with round window rupture?

A. The connection is direct, but to make sense out of this we'll have to start at the beginning. It starts with a diver who has difficulty clearing an ear as he descends through the water. He has ear squeeze — a relative vacuum in the middle ear. He blows against a closed mouth and nose (the valsalva maneuver) to force air up through the stubborn eustachian tube. It doesn't work, so he blows harder. This raises the pressure within his chest to a level above that in the great veins that return blood to the heart. They partially collapse, causing a rapid build-up of back pressure in the venous system.

The cerebrospinal fluid is formed from the arterial side and is reabsorbed into the venous side of the circulation. Back pressure in the veins prevents its reabsorption. Back pressure also increases the volume of blood within the brain case and spinal canal. Both effects elevate the pressure of the cerebrospinal fluid. The cerebrospinal fluid communicates directly with the perilymph in the inner ear — they're really both the same fluid — so perilymph pressure increases too.

The round window in the bony wall between the inner and middle ear is closed by a membrane something like the eardrum. With squeeze, the relative vacuum makes the membrane bulge into the middle ear. Any elevation of perilymph pressure increases the bulge and can rupture the membrane. This sends a shock wave through the inner ear and allows perilymph to leak out into the middle ear. The result is often permanent damage to the organs of hearing and balance. This is not just theoretical — it's happened to many divers. So don't descend when you can't clear easily, and don't use a strong valsalva.

Q. Isn't reverse ear block (reverse squeeze) mostly just a matter of pain? What if any actual damage occurs?

A. Since the eustachian tube normally lets air pass outward much more easily than inward, a diver who's able to descend rarely has trouble with his ascent. When it does occur, reverse block is usually mild, and the distress it brings is minor. If it affects the two ears unequally, alternobaric vertigo may result. That's Latin for change of pressure dizzyness. In most cases it's easily overcome by swallowing a few times or descending a few feet.

Severe reverse block can occur in two situations. A diver who uses a strong spray to open his stopped up nose, and stays down long enough for the effect to wear off, may be in for a lot of pain when he ascends. A diver whose nasal

membranes are very sensitive to cold may fare similarly. There's some evidence that barotrauma, including rupture of an eardrum or round or oval window, can result.

Never make the mistake of using a standard clearing maneuver (valsalva or frenzel) to relieve the discomfort of reverse squeeze. Forcing still more air into the already over-pressured middle ear will only add to the pain and danger.

Q. Why can smokers breathhold longer than non-smokers — a proven fact?

A. We're not convinced that they can. Breathholding ability depends upon so many things — lung volumes, carbon dioxide tolerance, metabolic rate, ability to relax, practice, motivation, etc. — that proving this premise would require studies set up in double blind fashion with multiple controls and involving vast numbers of people. In general, smoking reduces exercise tolerance by reducing maximum breathing capacity and the efficiency of gas exchange in the lungs. However, most smokers have adequate pulmonary function to keep their blood oxygen and carbon dioxide levels normal in the resting state. Since breathhold tests are conducted at rest, no significant difference between smokers and non-smokers would be expected.

Q. Review alternate methods for clearing the ears.

A. Clearing is easier if you descend slowly and feet first. Any clearing maneuvers should be utilized as soon as the slightest sensation of pressure is felt, for if the pressure in the middle ear is allowed to get much below ambient, clearing becomes difficult or impossible.

The smooth muscles that tug open the end of the eustachian tube are normally activated by swallowing, so swallowing is the most physiologic and safest way to clear the ears. Closing the mouth and nose while you swallow (a sort of mini-frenzel, of which more later) makes it more effective, without adding significant risk.

The valsalva (blowing against a closed mouth and nose to force air up the eustachian tube) is more powerful but decidedly more dangerous. Besides threatening window rupture, it increases air pressure within the chest. This collapses the big veins that return blood to the heart; and since the heart can't pump blood it doesn't receive, cardiac output falls. The brain, starved for oxygen, may stop functioning. That's underwater blackout.

The frenzel is the most powerful clearing maneuver we know, and it's safer than the valsalva because the pressure in the chest is not increased. To perform it, close off your mouth and nose as you would for a valsalva, then also close your throat. This is accomplished by closing the vocal cords, as you'd do just before a cough comes out. You've now isolated the small air space in the nasopharynx into which the eustachian tubes open. Make its walls rigid by

tensing your neck and throat muscles, and then compress the air within it by driving your tongue upward and backward as though it were a piston.

If the clearing maneuvers you try don't work, ascend a few feet and try again. If still no luck, hang it up and try again another day. It's a pity to miss the dive, but with permanent ear damage, you'll miss a lot more.

Next, turn to the druggist. A decongestant pill such as sudafed or a decongestant-antihistamine combination like actifed (or any of many others) may help. Or try a nasal spray. One of the most popular is afrin, a long acting decongestant. More appropriate for a few people is a spray that contains hydrocortisone, which inhibits swelling through several complex mechanisms.

If nothing else works, give up and see an ENT specialist. The problem could be a correctable allergy, deformity, or tumor.

Q. What information do you have on hearing loss caused by diving?

A. There have been conflicting reports as to whether divers of long experience hear less well than their non-aquatic age peers. It does seem that commercial and other divers who are frequently exposed to the loud noise of helmets and chambers being ventilated, compressors whining, and similar high amplitude cacophony do suffer significant loss in the high frequency range. Certainly anyone who violates the decompression guidelines invites cochlear damage by bubbles, and anyone who dives in spite of eustachian insufficiency invites otic barotrauma. However, we see no reason that diving per se should be injurious to the organ of hearing and know of no evidence that it is.

Q. Can diving, by flushing air in and out of the sinuses, help to eliminate allergies?

A. Negative. It's true that air supplied by dive shops is highly filtered, and this removes many of the pollens that might precipitate an allergic response. People who have sinus trouble induced by allergy may get relief when they switch from snorkel to regulator, but the allergic state is not changed, and topside the misery will recur.

PHYSIOLOGICAL SEMINAR SPEAKER QUESTIONS

By P.G. Linaweaver, Jr., M.D.

Virginia Barisich's question: 1) When teaching emergency ascent techniques, should the student exhale all the way to the surface, then at around 10 feet try to take a short quick breath?

The student should be instructed to normally exhale at the beginning of the ascent, continuing to hold the chest in this normal exhalation position with the airway and glottis open all the way to the surface and **not** try to take a short, quick breath at about 10 feet. However, the student should be advised that if he/she feels the need to breathe at anytime during the ascent, he/she should resume normal breathing through the regulator. The purpose of the instruction is to inform the student that due to the expanding gas, he/she should be able to exhale all the way to the surface in an emergency situation such as failure of the regulator or running out of air.

2) Please give the definition of the carotid sinus reflex.

The carotid sinus is an anatomical plexus of sensors and nerves near the bifurcation of the carotid artery. In some individuals this carotid sinus plexus is extremely sensitive to pressure and when pressure is applied, such as with a tight wet suit seal, it causes reflex slowing of the heart, at times to the point of fainting. I personally have never seen this happen in class, but it has been reported to have occurred, however I cannot quote a reference.

3) How do we teach shallow water black-outs?

Shallow water black-out, as used in the sport diving community, refers to passing out from anoxia due to prolonged breathholding at depth, particularly after hyperventilation which permits the diver to remain at depth holding his breath until the desire to breathe is stimulated by hypoxia and not elevated carbon dioxide. When the diver then ascends and drops the total ambient pressure, the oxygen partial pressure drops to anoxic levels and unconsciousness occurs.

Question by Steve Gold: Is it true that by keeping the regulator in your mouth while ascending, enough air will flow out without voluntarily exhaling?

No. Having the regulator in your mouth does not automatically open the airway and either voluntary exhalation maneuvers or normal breathing maneuvers must be performed in order to assure patency of the airway.

Question by Earl King: How do you recommend we condition ourselves to improve our working capability under water?

There is nothing special about physical conditioning for aquatic sports or diving activities as compared to any other form of cardiovascular conditioning. Any cardiovascular stimulating activity such as jogging, riding bicycles, or swimming is of value. Attention should also be given to maintaining ideal body weight and avoidance of such harmful activities as smoking. One exercise that I usually advise to people new to diving — particularly in cold water — is open water swimming with a full wet suit, buoyancy compensator, and enough

weights to remain neutrally buoyant. This helps them get used to the constricting sensations and added weight that requires increased effort.

Question from Eric Hanauer: What is the source of anatomical painting and your slides?

These are from the CIBA symposium series published by the CIBA Pharmaceutical Company, division of CIBA-Giegee Corporation, Summit, NJ 07901. The particular slides in question came from their series on the respiratory system.

It is hoped that these brief answers sufficiently clarify the interested party's questions.

ANSWERS TO QUESTIONS FROM "PHYSIOLOGY IN DEPTH"

Q. When using the Regulator to resuscitate an unconscious victim — how do you keep the regulator in the mouth of the victim? Is it designed to stay in the mouth? Also, how do you keep the nostrils closed?

A. In some cases the regulator remains within the mouth because of the design of the rubber mouthpiece.

There are certain instances though, where a slightly longer regulator hose will increase the probability of it staying in place.

During rough water or surf exits, the regulator should be held in the mouth to avoid aspiration of fluids.

When administering CPR there is no need to seal the nostrils or the mouth completely because of the high flow rate of the properly calibrated regulator. This open-water effect also prevents the possiblity of overexpansion or embolism.

Q. If you are using a standard regulator (not modified) and are faced with an emergency situation would you recommend using a regulator vs. mouth to mouth?

A. When faced with an emergency situation you should attempt all that you are capable of doing, but *only* what you have been trained to perform effectively and efficiently.

If you have a regulator that has not been tested and calibrated for Emergency Regulator Resuscitation (ERR) *do not attempt to administer resuscitation with it!!*

If you do use a non-calibrated regulator for ERR, it might have a pressure setting far above the safe limit for ventilation, and could possibly lead to embolism, or on the other hand, it may have insufficient pressure or flow to inflate even the smallest victim.

Please remember, if your regulator is not ERR calibrated, use it only for diving and not emergency resuscitation.

Q. How does a regulator, modified for emergency use, perform under "normal" sport diving conditions?

A. Regulators which have been modified for ERR performed almost as well as new regulators in breathing resistance tests, which means these units are capable of use as normal diving regulators as well as Emergency Regulator Resuscitation devices.

Q. What flow rate and pressure limits are necessary for the safe and effective use of a scuba regulator for resuscitation? Which regulators

have you found to be adaptable for this purpose?

A. The limits for safe and effective resuscitation can be found in our article, "New Techniques in External Cardiac Compressions: Aquatic Cardiopulmonary Resuscitation" in the Journal of the American Medical Association, *JAMA 244 (11): 1229-1232 September 12, 1980.*

These limits are determined by the National Association of Sciences — National Research Council, and are the same as those required for hospital type resuscitations.

Our data is still being compiled regarding which regulators will and will not be easily adaptable for ERR, and we hope to publish our preliminary findings shortly.

Q. Were gases checked during resuscitation using O_2 tank or air tank? Has this technique been used yet on real victims?

A. Blood gas data was monitored during resuscitation utilizing compressed air and 100% oxygen (O_2) as the source.

In the case of a diver-to-diver rescue, experiments were performed with 21% O_2 versus 100% O_2 while animal studies were done on both air and 100% oxygen.

Only in lifeguard or Coast Guard rescues, or in water less than 30 feet deep is 100% oxygen suggested for use with ERR.

In answer to the second half of this question please see next question.

Q. Has in-water CPR ever been done on a *real* non-breathing, non-circulating victim?

A. We had several unconfirmed reports through the Coast Guard, that the techniques have been utilized three times within the last few years and that two victims survived, but there was no definitive determination that the victims were actually in cardiac arrest.

One other documented case where the victim was actually drowned, took place during a scientific saturation dive and involved only ERR.

In this instance the victim was revived within a short time and survived without any resulting cerebral sequel.

Q. Do you have, or are you aware of any data that would support an approximate mean arterial pressure or cardiac output during in-water resuscitation.

A. There has been Aquatic CPR experimentation performed on human cadavers by Drs. Meyers and Bradley, at the University of Maryland, Shock Trauma Center. These studies were conducted in a simulated diving bell and showed that the arm and body positions we are proposing provided sufficient systemic perfusion and adequate cerebral bloodflow when the victim

was in near-horizontal attitudes.

Other research by Hong, et.al. has shown that immersion causes thoracic pooling of the blood, as does a wet suit, creating increased volumes of blood available during compressions in Aquatic CPR.

Q. Will your in-water CPR method work with all types of buoyancy compensators? (i.e. front mount, back mount, stabilizing jacket)

A. The methods of Aquatic CPR tested several types of buoyancy devices as well as no buoyancy whatsoever. Our studies have shown that most back mounted or jacket-type units will provide adequate flotation and support for the victims back.

More information on the various combinations of flotation systems are discussed in detail in the "Aquatic CPR: Training Manual" available soon through the PADI International College, San Diego.

Q. Have you considered the problem of finding a pulse in the studies with in-water CPR?

A. Finding a pulse in the water is difficult, but not at all impossible as some would insist. With proper training it is possible to determine both respiratory and cardiac arrests.

Actually, cardiac arrest must be determined by assessment of various different signals, and not just the absence of a palpable pulse.

Q. How much strength is required to administer in-water compressions?

A. Some arm strength is required to administer chest compressions, especially over extended time spans.

In our studies though, five out of six individuals were able to compress, ventilate and transport a victim, while successfully performing aquatic CPR within established American Medical Association limits.

Q. Where can we obtain more information concerning open-water CPR and the ERR?

A. More information regarding aquatic CPR and Emergency Regulator Resuscitation (ERR) may be obtained by writing Nico F. March at the U.C.S.D. School of Medicine M-023A, La Jolla, California 92093. Reprints are available at 25 cents each to cover postage and handling.

Further information on upcoming aquatic CPR training courses and reference/training manuals as well as audio visuals is available from the PADI International College in California, Inc., 1310 Rosecrans Street, San Diego, CA 92106.

BIOSKETCH
Susan Bangasser, Ph.D.

Dr. Bangasser began diving in 1970, and became a NAUI instructor in 1974. She earned her Ph.D. in biochemistry from the University of Illinois Medical Center, in 1975. She is active in the area of women diving. She co-authored the book *Women Underwater*, conducted the Medical Aspects of Women Divers Survey, and has written and spoken on various aspects of diving physiology. Currently, she is serving as co-branch manager of the NAUI Pacific and South Pacific Branch. She is co-director of Sea-to-Sea Scuba School, and teaches all levels of diving skills.

BIOSKETCH
Bruce E. Bassett, Ph.D.

Dr. Bassett has specialized in diving physiology and medical education for

over 16 years, having been the project officer responsible for establishing the U.S. Air Force's Hyperbaric Therapy Training Program. He has conducted programs in the field of diving medicine for the University of Southern California, the National Park Service, the Government of Mexico, NASA, NOAA, the Commercial Diving Center, and all major sport diving certification agencies. His research contributions have all been in the area of decompression/decompression sickness. He is president of *Human Underwater Biology Inc.* which specializes in the medical and physiological aspects of diving safety and education and is Program Director for *Diving Medicine in Depth.* He serves on many advisory boards, including the Western Regional Undersea Laboratory, the PADI International College, the National YMCA Underwater Activities Medical Advisory Board, and The Ocean Corporation.

BIOSKETCH
Charles V. Brown, M.D.

Dr. Brown has been a sport diver for 13 years, with UMCA basic and NAUI advanced training, and logs 100 dives yearly. He has practiced medicine for 21 years, now limited to emergency medicine and hyperbaric consultation. He is examiner and lecturer for the commercial diving school at Chino, California state prison, and for numerous other diving activities. He is medical editor for NAUI News, Medifacts columnist for *Skin Diver Magazine*, and a member of the Undersea Medical Society.

BIOSKETCH
Christopher W. Dueker, M.D.

Dr. Christopher Dueker is an anesthesiologist at the Palo Alto Medical Clinic and a Clinical Assistant Professor of anesthesia at Stanford University. He was trained in underwater medicine in the Navy and at the University of

Pennsylvania. He has written for *The Undersea Journal*, a PADI publication, since 1970. Publications include *Medical Aspects of Sport Diving* (under revision) and *Scuba Diving Safety*. Dr. Dueker's personal diving interests date to the ping-pong ball era.

BIOSKETCH
Paul Linaweaver, M.D.

Dr. Linaweaver's diving interests began in 1953 while in medical school and he did U.S. Navy reserve duty with the Experimental Diving Unit. He spent 25 years in the USN; major assignments included the Experimental Diving Unit, Submarine Medical Research Lab, Sec Lab Project, and Senior Medical Officer, Navy's Deep Submergence Organization. Since 1976, he has been at the Santa Barbara Medical Clinic and acted as a consultant to many commercial diving companies, Santa Barbara City College marine tech course, the Commercial Dive Center, and the Catalina Chamber. He is also on the advisory board of PADI International College, San Diego.

BIOSKETCH
Nico F. March

Nico F. March has been sport diving since 1974, and has done underwater research for Scripps Institute of Oceanography. Presently he is involved in medical research regarding aquatic and diving emergency situations at the University of California, San Diego School of Medicine. He has also developed and tested criteria for Emergency Regulator Resuscitation (ERR) and Aquatic CPR. His work has been published in the *Journal of the American Medical Association*, the *Journal of Undersea Biomedical Research*, and *Studies in Commercial Rescue Procedures* at Commercial Diving Center.