ASCENT AND SILENT BUBBLES

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In 1971, when the Doppler "Bubble Detector" became readily available to research, studies were initiated at the University of Southern California's Catalina Marine Science Center to document the degree of "silent bubble" occurrence after open ocean scuba dives. One hundred ten Doppler monitored subject-dives were done. Dives conformed to the limits of the U.S. Navy Standard Air Decompression Tables. Exact bottom times, depths and ascent rates were adhered to. No symptoms of DCS were seen. However, venous gas emboli (vge) were found to some degree in all subjects after all dives. It was found that with small increases in decompression times, vge scores could be greatly reduced. Thus, short "safety stops" could be beneficial in reducing the occurrence of "silent bubbles" in divers using the limits of the U.S. Navy Tables.

Preface

This paper is a summary of one part of a 3-year study done by the author between May, 1972 and June 1975. The complete report has the following reference:

Pilmanis, Andrew A. 1975. "Intravenous Gas Emboli in Man After Compressed Air Ocean Diving." USN Office of Naval Research, Final Technical Report, Contract No. N00014-67-A-0269-0026, May 1, 1972 to June 30, 1975.

Background

The condition of decompression sickness stems from the inert gas partial pressure gradients developed after hyperbaric exposure between the ambient breathing gas and the body tissues. The degree and rates of various tissue inert gas saturation and desaturation determine if, and to what extent, tissue gas emboli formation and growth occurs. Specifically, the primary variables associated with the evolution of gas bubbles in the body during and/or after hyperbaric exposure are:

1. ambient pressure (depth of water)
2. bottom time
3. decompression time.

In addition, under actual open ocean diving conditions, there are other factors that influence inert gas uptake and elimination, including:

1. degree of exercise
2. water temperature

3. water immersion
4. constrictive equipment worn
5. psychological factors.

The development of non-invasive methods of detecting *in vivo* intravascular gas emboli opened a relatively objective field of study for the problems of decompression sickness in man. Through the use of the Doppler ultrasonic flow-meter, modified for use as a bubble detector, definitive evidence has been obtained that circulating non-symptomatic venous gas emboli (vge) exist after certain dive profiles previously considered "safe". These non-symptomatic gas emboli have been termed "silent bubbles". Despite the lack of apparent symptoms from these vge, it is highly probable that there is some degree of tissue damage associated with frequent "silent bubble" foundation.

Most of the Doppler studies have been done in hyperbaric chambers under very controlled conditions. Thus, many of the inert gas uptake/elimination variables listed above for open ocean diving were not considered in these studies. The task of bubble detection in an immobile man in a hyperbaric chamber is much less complex than the task of bubble detection in a working diver in the ocean environment where pressure change is but one of the influencing factors.

The overall purpose of this study was to attempt to define the occurrence and extent of decompression "silent bubble" formation in man after ocean diving to the limits of the U.S. Navy Standard Air Decompression Tables (USN Tables). The initial objective was to demonstrate the feasibility of *in vivo* intravascular bubble detection during the post-dive period of open ocean air scuba dives. This was immediately followed by a characterization of the post-dive time course of vge occurrence.

Methodology

All experimental work was done at the University of Southern California Catalina Marine Science Center, located at Big Fisherman Cove, Santa Catalina Island. The diving site for these studies was approximately 300 yards from the dock in 100 to 200 feet of water. A powered diving platform was always anchored over the site during the diving operations.

A model A 5 MHz Precordial Doppler Ultrasonic Bubble Detector was acquired from the Institute for Environmental Medicine and Physiology, Seattle, Washington. This was replaced with a Model B in the second year. These units were successfully used during the project for the detection of venous gas emboli. The large precordial transducer consisted of two 1/2 inch square piezoelectric crystals separated 1.3 cm. and tilted at a 13° angle so that the ultrasonic transmitter and receiver beams cross in a region 3 to 4 cm. distant. The advantage of this unit was that it covered a large tissue volume at its focus and, thus, positioning was less critical and there was higher probability of detecting vge in the pulmonary blood.

During the course of the 3-year study, 18 resident scientific divers of the U.S.C. Catalina Marine Science Center were used as subjects. These people routinely perform working dives and are thoroughly familiar with the diving station. All dives were free-swimming air scuba dives. After extensive experience with several brands of depth gauges (in a test chamber and in the ocean) it was concluded that none of them had the reliability and accuracy required for these experiments. Thus, a steel cable lead-line was used as the primary depth sounder and was dropped and left suspended during all dives. Markers on

this line every ten ft. permitted accurate ascent rates. Horizontal visibility was between 40 and 80 ft. Water temperature was between 11 and 13°C. The subjects' descents were totally passive. During the resting dives, the subject knelt motionless on the bottom for the full bottom time. The ascents were at 60 ft./min., were passive, and controlled through buoyancy regulation. The Doppler recordings were made on the diving platform. Two-minute control recordings were made prior to each dive. Due to the time required for boarding the dive platform and the removal of diving gear, the earliest bubble detection recordings were made 3-5 minutes after the point of surfacing.

Eight two-minute post-dive recordings were made at 5, 15, 30, 45, 60, 90, 120, and 180 minutes after surfacing. The data was simultaneously recorded on tape and monitored with earphones. Subjects were seated and motionless during the recordings.

The following 3 dive profiles were used:

	Depth (ft.)	Bottom time (min.)	Decompression (min./ft.)
1.	100	25	none
2.	100	30	3/10'
3.	190	10	2-3/20'
			4-5/10'*

*(decompression was added to USN requirements for safety reasons)

Animal studies were done to verify the electronic characteristics of the intravascular bubbles as recorded by the bubble detector. The animals and human data were subjected to audio and oscillograph analysis and a method was then developed for quantification of the data. It is important to note that these signals are termed "events", not bubbles, because occasionally, there are cardiac events which also elicit above-control level characteristics. However, the majority of these electronic events are interpreted to represent gas emboli passing through the right heart. The events are tabulated for each 2-minute period and compared with the audio counts made for the same period (Figure 1). There were two problems encountered with this method of bubble quantification. The sensor placement had to be very critical, since it was found that signals produced by valve closures and/or peak flow were sometimes as high as those produced by bubbles and, thus, masked the bubble data. Secondly, a substantial portion of the bubbles that were audible in the earphones did not display above control frequencies. Because of these problems, the human ear continued to be considered the most accurate method.

Figure 1. Brush recording from the "Bubble Counter"

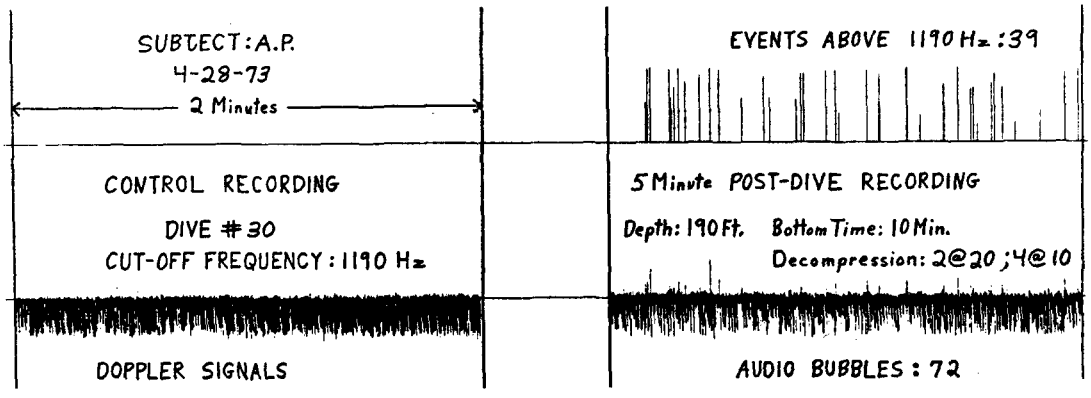
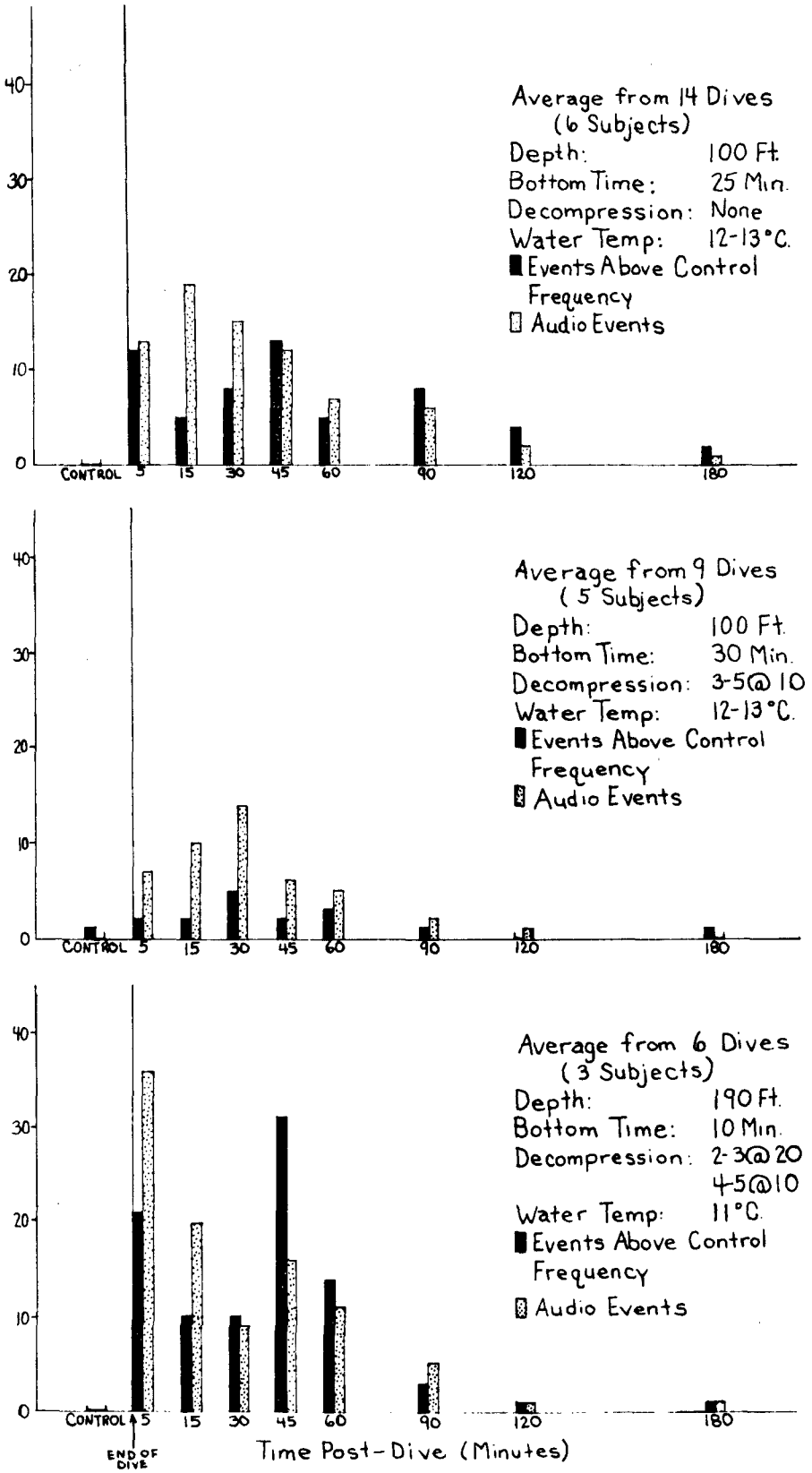


Figure 2. Averaged vge data from 3 dive profiles



Results

One hundred ten subject-dives were made in the open ocean. No symptoms of decompression sickness were seen as a result of any of the diving. Intravascular "silent bubbles" were present, to some degree, after all of the dives reported. The averaged vge occurrence time-courses of the three dive profiles are seen in Figure 2. "Silent bubbles" were present within a few minutes after surfacing from the dives. The number of events generally peaked within an hour post-dive, declined and was close to control levels by three hours post-dive. Table I gives the levels of significance of each averaged recording to the control levels. Significant differences occurred at the 5, 15, 30, and 45 minute post-dive recordings. A great range of individual variability was seen. Furthermore, each individual showed a relatively consistent degree of bubble formation on various dive profiles and repeated dives. In particular, the subject in Figure 3 consistently produced large numbers of events, even after a relatively "safe" dive profile. A bottom time of 25 minutes is the "no-decompression limit" for a depth of 100 feet according to the USN Tables. Yet, this subject always exhibited large numbers of events after such an exposure. **However, when relatively short decompression periods were added to the dive profile, the number of post-dive events was drastically reduced.**

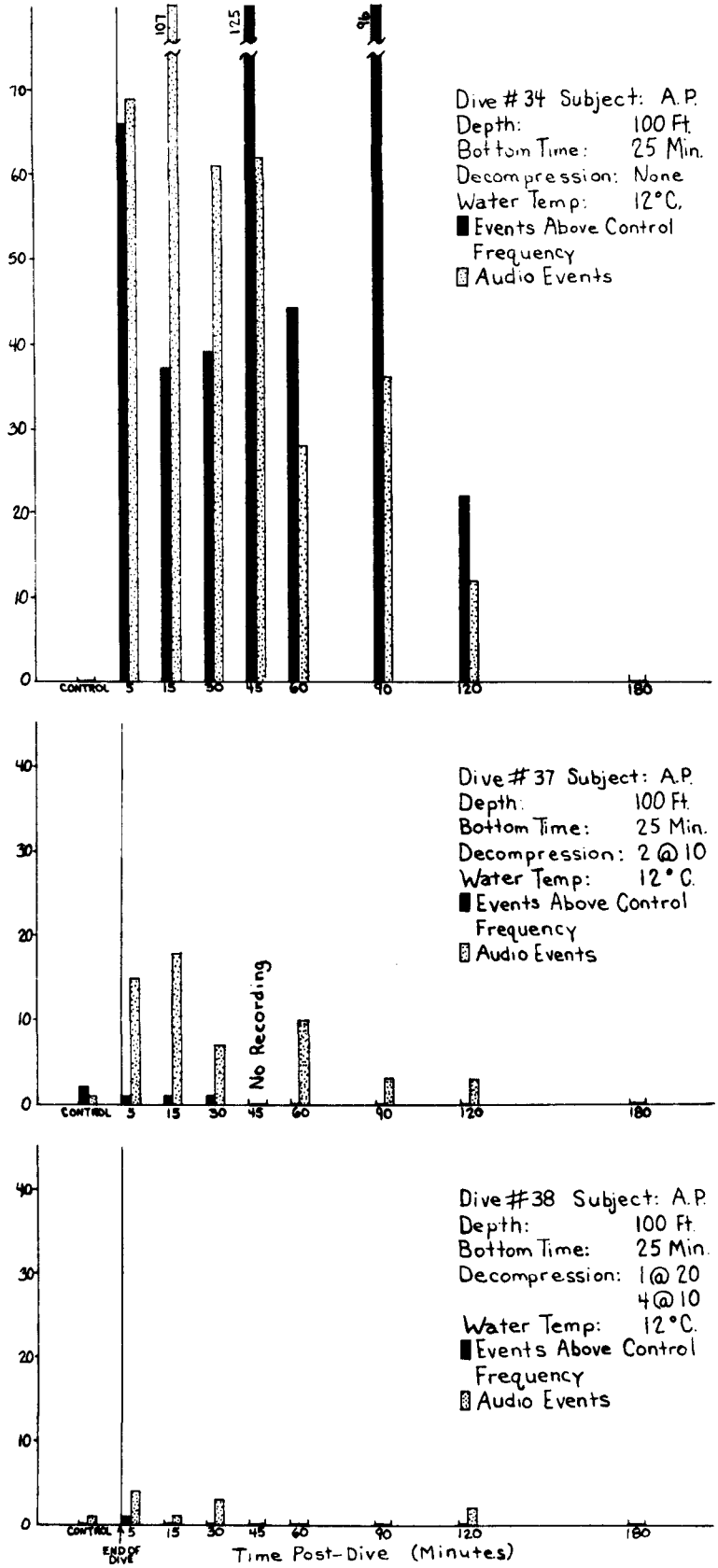
Table 1

Dunnet's t Statistic

	Dive Profiles	Recordings Post-Dive (minutes)							
		5	15	30	45	60	90	120	180
1.	100 ft / 25 min No decompression								
	Audio	**	**	**	**	NS	NS	NS	NS
	Electronic	**	NS	NS	NS	NS	NS	NS	NS
2.	100 ft / 30 min 3 min @ 10 ft								
	Audio	NS	*	**	NS	NS	NS	NS	NS
	Electronic	NS	NS	*	NS	NS	NS	NS	NS
3.	190 ft / 10 min 2 min @ 20 ft 4 min @ 10 ft								
	Audio	**	*	NS	NS	NS	NS	NS	NS
	Electronic	**	NS	NS	NS	NS	NS	NS	NS

NS = Not Significant
* = P less than 0.05
** = P less than 0.01

Figure 3. Vge data from subject A.P. after 3 dive profiles; depth and bottom times were identical, only the decompression was changed.



Discussion

The full extent of the pathophysiological complexity of decompression sickness has recently become more and more apparent. At the same time, many of the basic mechanisms of action that lead to the varied and interrelated clinical manifestations of decompression sickness remain elusive. The existence of asymptomatic venous gas emboli is now generally accepted. However, the pathophysiological significance of these bubbles has still not been adequately defined.

It is clear from this study, as well as others, that man can tolerate and eliminate at the lungs relatively large quantities of gas emboli from the venous system without developing clinical symptoms of decompression sickness. It is also clear that pre-symptomatic bubbles are present in large numbers after open ocean dives which strictly adhere to the limits of the USN Tables. It is suggested that silent bubble formation and clinically symptomatic bubble formation are not two distinct conditions, but rather, the same condition at various levels of gradation. In addition, the point of development of obvious symptoms is not necessarily synonymous with the start of tissue damage. The pathology from vge may simply be a milder form of "symptomatic" tissue damage. Any gas emboli in the tissues are potentially harmful.

It should be noted that the bubbles detected by the Precordial Doppler are, physiologically, relatively unimportant. These venous bubbles funnel into the right heart, pass to the pulmonary circulation and dissipate. It is unlikely that they cause any tissue damage, except perhaps for hematological alterations. Pathophysiologically, the stationary bubbles in the various tissues are the prime contributors to the disease. However, we cannot detect stationary bubbles. The circulating gas emboli may only be **detectable** indicators of the presence of bubbles in other tissues.

In conclusion, these data suggest that by increasing decompression times by a few minutes over those required by the USN Tables, "silent bubble" formation into the venous system can be significantly reduced. Thus, the routine use of "short safety stops" when diving the U.S. Navy Tables to the "no-decompression" limits is strongly encouraged. However, it is also emphasized that this conclusion is based on an N of 1, and additional research is needed to confirm these findings.