J Forensic Sci, Jul. 1978, Vol. 23, No. 3

C. D. Brown,[1] Maj., USAF MC; Watson Kime,[1] M.D.; and E. L. Sherrer, Jr.,[1] M.D.

Postmortem Intravascular Bubbling: A Decompression Artifact?

Rapid advances in diving technology over the past decade have opened the underwater world to thousands of professional and sport divers. As may be expected with any activity involving an element of risk, the number of diving-related accidents and deaths has also risen [*1-3*]. Unfortunately, at the present time too few physicians and pathologists have been adequately trained in the investigation of deaths occurring during diving activities.

The need for accurate statistical data on diving-related deaths is becoming more important for several reasons. First, it is essential to determine the risk factors involved in both commercial and sport diving so that corrective steps may be taken in diving procedures and diver training [*4,5*]. Second, the legal and political implications of diving accidents and deaths will require accurate determination of the causes for the intelligent formulation of future policy.

Intravascular bubbles found at autopsy following a diving accident are frequently interpreted as proof that death occurred because of air embolism or decompression sickness [*6*]. While this may be true in some cases, other explanations must be considered. If a diver dies under pressure, the simple act of bringing the body to the surface may precipitate bubbling sufficient to be detectable at autopsy.

This question therefore arises: In a drowned diver at depth, do the length of time spent at depth, both before and after death, and the rate of ascent on recovery have any effect on the development of the bubbles found in the vessels at autopsy?

Materials and Methods

Male and female guinea pigs, weighing from 310 to 1200 g, were selected as experimental models. Because we were interested in drowned divers, drowning was selected as the method of death. In this way, the panic reaction and involuntary respiratory motions occurring during drowning could be faithfully reproduced.

All animals were killed by being placed in small wire cages and immersed in water until respiratory efforts ceased. Autopsies were carried out immediately or after 24 h. Animals undergoing delayed autopsies were kept in a cold locker at 4°C during the interim. In addition, it was demonstrated that small bubbles introduced by intravascular injection could be recovered after overnight refrigeration. Initial studies were carried out on five animals drowned at atmospheric pressure (98 kPa). These studies showed that animals so drowned demonstrated no intravascular or extravascular bubbles.

Received for publication 24 June 1977; revised manuscript received 1 Nov. 1977; accepted for publication 14 Nov. 1977.

[1]Senior resident in surgery, director of Department of Laboratories, and adjunct attending staff member, Department of Pathology, respectively, South Baltimore General Hospital, Baltimore, Md.

Autopsies consisted of opening the chest and examining the jugular, innominate, and subclavian veins; superior and inferior vena cavae; right atrium; pulmonary veins and arteries; and thoracic aorta. Sections of muscles, fat, myocardium, salivary glands, and lungs were taken for microscopic study. The presence or absence of intravascular bubbles was noted, with qualitative estimates of the size and the number of bubbles.

Two experimental dive profiles were used. In the first, animals were taken to 30 m (100 ft) (392 kPa or 4 atm) and kept there until the maximum allowable human "no decompression limits" (25 min) had been exceeded. In the second study, animals were taken to 18 m (60 ft) (294 kPa or 3 atm) for less than the maximum human "no decompression limit" (60 min). All experiments under pressure were conducted in a double-lock hyperbaric chamber.

First Experiment

The dive profile is shown in Fig. 1. Fifteen animals were divided into three groups of five each. After 30 min bottom time at 392 kPa (4 atm), the animals were killed. Group A was then placed in the outer lock and brought to the surface as fast as the lock could be vented (about 4 min). Group B returned to the surface with the tenders, taking the standard decompression stops required for a total bottom time of 70 min. Group C was brought up on an extended decompression table patterned after saturation dives, the belief being that guinea pigs may saturate their tissues with nitrogen sooner than humans and that a longer, slower decompression might be required to release the saturated gas without causing bubbling (Table 1).

Autopsies of all 15 animals revealed bubbles in the arterial and venous systems (Figs. 2 and 3). In addition, qualitative measurement of bubbles found in the superior vena cava and internal jugular veins showed no significant difference in the number or size of the bubbles (Table 2).

It is interesting to note that one animal from Group B was autopsied by the tenders in the medical lock during decompression. Bubbles were observed to form in the superior vena cava of the animal at a depth of 6 m (20 ft) (156.8 kPa or 1.6 atm).

Second Experiment

Since the majority of diving deaths occur in shallow water without extended bottom times, we devised a second profile which we hoped would determine whether bubbling could be demonstrated after relatively short exposures to increased pressure. In addition, we wanted to demonstrate that respiratory gas exchange was necessary for the development of bubbles. A diver, drowned at or near the surface, who then sank, to be later recovered, should not demonstrate bubbles because he was not breathing gas at increased pressure.

Eighteen animals were divided into three groups of six each. Dive schedules for each group and autopsy results are shown in Fig. 4 and Table 3.

As expected, animals drowned on the surface and then compressed showed no bubbling. The group of animals taken to depth and drowned immediately showed no bubbling whether they were decompressed immediately or kept at 392 kPa (3 atm) for 60 min prior to decompression. The animals given 30 min of bottom time before drowning exhibited bubbling after both immediate and delayed decompressions.

Discussion

Boycott et al [7] warned as early as 1908 that "the presence of bubbles *in vivo* must be inferred from their discovery post mortem with considerable caution. The super saturation

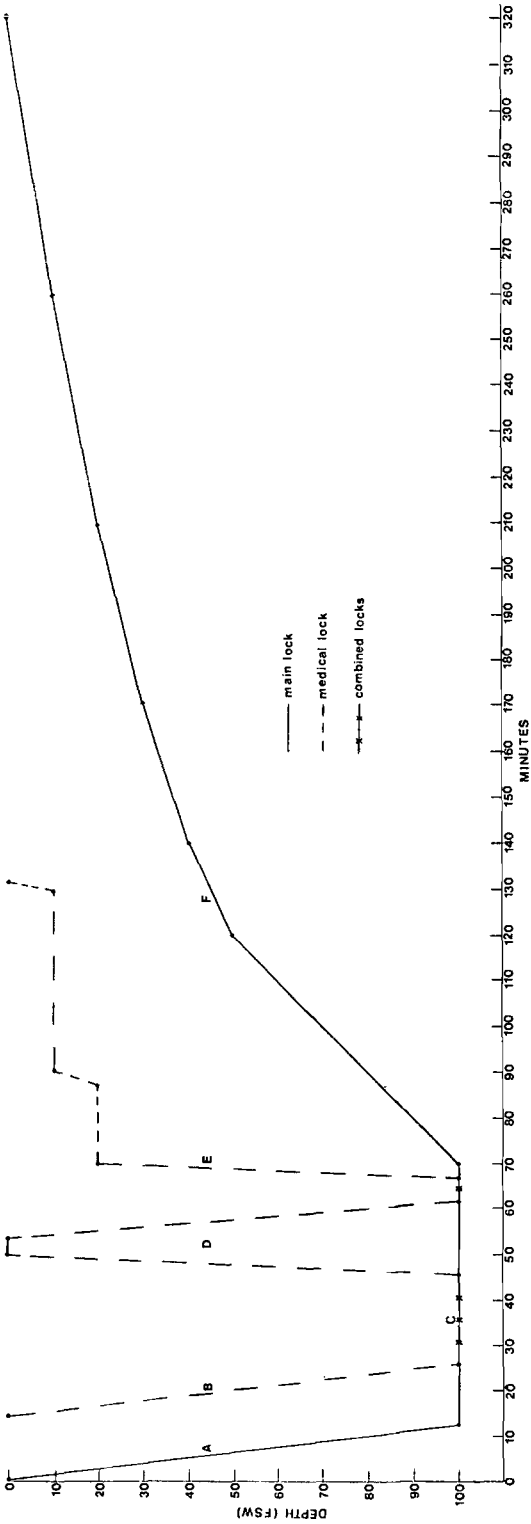

FIG. 1—*Schematic presentation of Dive 1.* (A) *All animals were taken to 392 kPa (4 atm) in main chamber.* (B) *Tenders were locked in to minimize bottom time.* (C) *All animals were drowned.* (D) *Group A animals were decompressed rapidly in medical lock.* (E) *Group B animals and the divers ascended on standard decompression schedule for 30 m (100 ft) and 70 min in medical lock.* (F) *Group C animals were brought up on extended saturation table (FSW = feet of seawater).*

TABLE 1—*Saturation decompression schedule for Dive 1, Group C. Total ascent time was 250 min.*[a]

Depths, ft			
From	To	Rate, ft/min	Total Time for Stage, min
100	50	1	50
50	40	2	20
40	30	3	30
30	20	4	40
20	10	5	50
10	surface	6	60

[a]1 ft = 0.3 m.

FIG. 2—*Internal jugular vein showing large (3 mm) bubble.*

of the body may be such that the separation of the gas bubbles may take place after death." Hanson and Young [*8*] reiterated in their discussion of diving accidents that postmortem gas bubbles are of no significance if a diver died under pressure. Hempleman's studies [*9*] in 1969 indicated that intravascular gas came from the reservoir of gas in lung tissue, and he implied that bubbles are probably present in all divers to some extent. In his studies, animals were killed by the inhalation of inert gas or by intraperitoneal injection of barbiturates. The Doppler flowmeter [*10,11*] has shown that Hempleman's postulation was correct by demonstrating intravascular bubbles in asymptomatic divers and laboratory animals during ascent. The implication is that if bubbles can be present intravascularly in a healthy individual on ascent, the finding of postmortem bubbles at autopsy

FIG. 3—*Pulmonary artery showing multiple bubbles.*

is of doubtful significance, especially if no clinical history or eyewitness account of the accident can be provided.

In spite of these studies, pathologists unaware of these facts may unknowingly equate intravascular bubbles with air embolism or decompression sickness without any corroborating history or findings such as obviously ruptured pulmonary blebs.

The hypothesis that we wished to examine in this study is that gas may be forced into the circulatory system postmortem by decompression during recovery or that the dissolved

TABLE 2—*Results of Dive 1.*

Group	Animal	Weight, g	Ascent Time, min	Bubbles
A	1	370	4	+
A	2	520	4	+
A	3	540	4	+
A	4	580	4	+
A	5	480	4	+
B	6	470	57.3	+
B	7	500	57.3	+
B	8	310	57.3	+
B	9	560	57.3	+
B	10	440	57.3	+
C	11	570	250	+
C	12	660	250	+
C	13	310	250	+
C	14	550	250	+
C	15	460	250	+

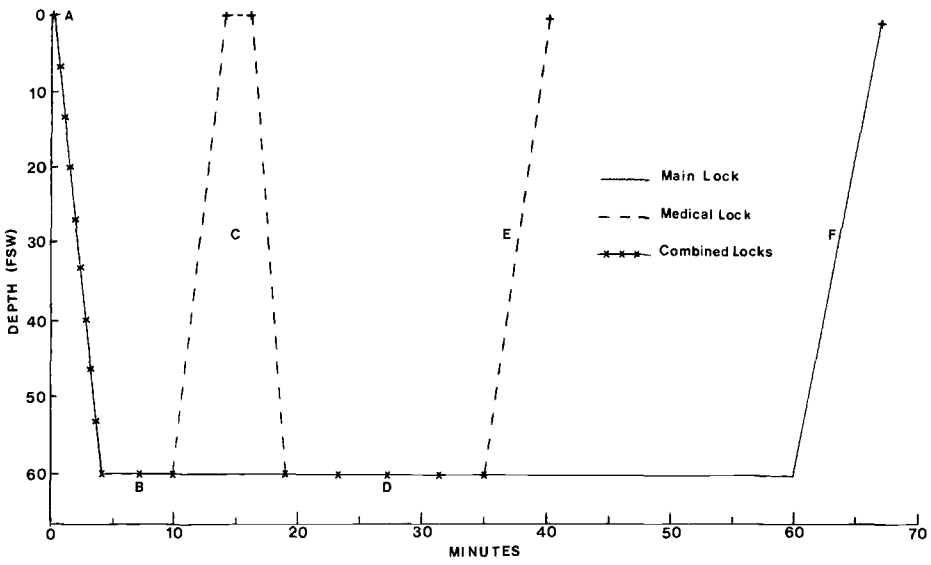

FIG. 4—*Schematic presentation of Dive 2.* (A) *All animals in Group D were drowned at 98 kPa (1 atm) and taken to 294 kPa (3 atm) with Groups E and F.* (B) *Group E animals were drowned.* (C) *Subgroups D' and E' were rapidly decompressed in medical lock.* (D) *Group F animals were drowned after 30 min bottom time.* (E) *Subgroup F' animals were decompressed with tender in medical lock at the standard rate of ascent [18 m (60 ft) per minute].* (F) *Subgroups D", E" and F" animals were decompressed after 60 min bottom time (FSW = feet of seawater).*

gas already present in the circulation expands during decompression as it does in a living individual and therefore will be seen as an artifact at autopsy. We demonstrated large amounts of foamy material in the lungs at autopsy. This could serve as a reservoir of gas to be forced into the pulmonary circulation by decompression.

Conclusions

Contemporary studies [*10,11*] have demonstrated bubbling in the circulatory systems of healthy divers and laboratory animals during ascent from relatively shallow depths.

Our studies tend to support the hypothesis that the observed intravascular gas was either there before death and expanded during decompression or was forced into the circulation from the lungs. In addition, the study seems to indicate the following:

1. Bubbling artifacts can be created even though "no decompression limits" are not exceeded.
2. The rate of ascent in recovering the body does not affect the formation of bubbles to any appreciable degree.
3. The depth at which death occurs need not exceed 18 m (60 ft).
4. The individual must be subjected to increased pressure before death to develop bubbles and a minimum time at pressure is required to dissolve sufficient gas [30 min at 18 m (60 ft)].

The probability that guinea pigs saturate more rapidly than humans in no way changes the implications of our results. The fact remains that we were able to demonstrate artifactual bubbling without subjecting the animals to decompression sickness or air embolism.

TABLE 3—*Results of Dive 2.*

Group	Animal	Weight, g	Drowning Depth[a]	Bottom Time, min		Bubbles
				Before Drowning	After Drowning	
D′	16	750	surface	0	10	0
D′	17	650	surface	0	10	0
D′	18	630	surface	0	10	0
D″	19	750	surface	0	60	0
D″	20	560	surface	0	60	0
D″	21	410	surface	0	60	0
E′	22	510	60 ft	5	0	0
E′	23	550	60 ft	5	0	0
E′	24	600	60 ft	5	0	0
E″	25	540	60 ft	10	50	0
E″	26	575	60 ft	10	50	0
E″	27	540	60 ft	10	50	0
F′	28	900	60 ft	30	0	+
F′	29	440	60 ft	30	0	+
F′	30	740	60 ft	30	0	+
F″	31	800	60 ft	30	30	+
F″	32	840	60 ft	30	30	+
F″	33	1200	60 ft	30	30	+

[a]1 ft = 0.3 m.

In the light of these findings it becomes apparent that investigation of diving accidents and deaths must be undertaken with caution. The best solution to the problem would be impractical: a pressure vessel would be lowered to the depth at which the victim was found and the body placed inside. Recovery to the surface could then be accomplished without decompression. At the surface, the pressure vessel would be mated to a dry recompression chamber and the autopsy done at the recovery pressure. By eliminating any decompression of the body until after autopsy, any bubbles found would have far greater significance.

While this procedure may one day find limited application, most examiners will have to rely on a full history of the accident, including witnesses' accounts, depth and duration of the dive, and evidence of ascent prior to the accident [*12*].

In all diving-associated deaths, a careful autopsy that uses special techniques for the identification of intravascular gas bubbles or free gas in the pleural cavities must be performed [*13,14*]. If no evidence of cardiopulmonary barotrauma is found and the pressure exposure is inconsistent with decompression sickness, the finding of intravascular gas bubbles does not justify the diagnosis of air embolism or decompression sickness.

Summary

The relationships between drowning occurring in divers using compressed air, with subsequent recovery of the body to the surface, and the finding of intravascular gas bubbles at autopsy were studied.

Guinea pigs were exposed to compressed air in a hyperbaric chamber at various depths [98, 294, and 392 kPa (1, 3, and 4 atm)] for various intervals and then drowned at depth. Various decompression schedules were used in returning the drowned animals to the surface.

Autopsy examination indicated that large and small intravascular gas bubbles may be found, in the absence of traumatic air embolism or decompression sickness, as a result of the process of decompression alone.

Postmortem findings of intravascular bubbles in drowned divers must be interpreted with caution and the importance of a full history of the incident, including depth and time of diving and evidence of ascent before drowning, are critical to the proper interpretation of intravascular gas bubbles.

Acknowledgments

We are indebted to Jackie Crowther for her assistance as hyperbaric chamber operator during our experimental dives. Hyperbaric facilities and financial support for the investigation were donated by Dive-Med International, Baltimore, Md.

References

[1] Fuller, R. H., "The Clinical Pathology of Human Near-Drowning," *Proceedings of the Royal Medical Society*, Vol. 56, 1962, pp. 34-38.
[2] Griffin, H. W., "Near-Drowning, Its Pathophysiology and Treatment in Man," *Military Medicine*, Vol. 131, 1966, pp. 12-21.
[3] Rivers, J. F., Orr, G., and Lee, "Drowning: Its Clinical Sequellae and Management," *British Medical Journal*, Vol. 2, 1970, pp. 157-161.
[4] Berbhage, T. E., "Summary Statistics, U.S. Navy Diving Accidents," *USN Experimental Diving Research Report 1-66*, Department of the Navy, Washington, D.C., 1966.
[5] McAniff, J. J. and Schenck, H. V., "Investigation of Scuba Deaths," *Journal of Sports Medicine*, Vol. 2, No. 4, 1974, pp. 199-208.
[6] Lanche, J. M., "Investigation of Diving Deaths in California in 1970," *California Medicine*, Vol. 116, No. 6, 1972, pp. 18-22.
[7] Boycott, D. M., Damant, G. C. C., and Haldane, J. S., "The Prevention of Compressed Air Illness," *Journal of Hygiene*, Vol. 8, No. 3, 1908, pp. 342-443.
[8] Hanson, R. G. and Young, J. M., "Diving Accidents," in *The Physiology and Medicine of Diving and Compressed Air Work*, Bennett and Elliot, Eds., Williams and Wilkins Co., Baltimore, Md., 1975, p. 554.
[9] Hempleman, H. V., "Bubble Formation and Decompression Sickness," *Revue de Physiologie Subaquatique*, Vol. 1, 1969.
[10] Gillis, M. F., "Research on Deep Submergence Diving Physiology and Decompression Technology Utilizing Swine: Evaluation of Swine as a Hyperbaric Analog to Man and Detection of Emboli by Use of the Ultrasonic Doppler Flowmeter," Battelle Memorial Institute Final Report, Richland, Wash., 20 May 1971.
[11] Spencer, M. P. and Campbell, S. D., "Development of Bubbles in Venous and Arterial Blood During Hyperbaric Decompression," *Bulletin of the Mason Clinic*, Vol. 22, 1968, pp. 26-32.
[12] Elliott, D. H., "Diving Accidents," in *Diving Manual*, B. Eaton, Ed., BSAC, London, 1966, pp. 111-117.
[13] Spitz, W. U. and Fisher, R. S., *Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, Charles C Thomas, Springfield, Ill., 1973.
[14] Ludwig, J., *Current Methods of Autopsy Practice*, W. B. Saunders, Philadelphia, 1972.

Address requests for reprints or additional information to
Cary D. Brown, Maj., USAF MC
USAF Hospital
Patrick AFB, Fla. 32925