Air Embolism as a Cause of Death in Scuba Diving in the Pacific Northwest

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Man's desire, in this rapidly changing and expanding world, to explore one of his alien environments - the sea, requires a physiologic constitution which he does not possess. The environmental factor of underwater living calls for a new storehouse of facts. He may explore the sea superficially by swimming, greater depths by surrounding himself with submarine machinery, by hard-hat diving suit gear or by the very mobile diving equipment of SCUBA (self-contained underwater breathing apparatus). Our problem here concerns air embolism, one of the very real pulmonary hazards of scuba diving.

It is estimated that there are three million scuba divers in this country alone. Of this group not all are properly educated and examined, in spite of the availability of excellent training program through scuba clubs, athletic associations, the Y.M.C.A. and other groups.

Scuba deaths in the Pacific Northwest, although not nearly as impressive in number as our household and automobile deaths, have the unique distinction of virtual total prevention - a facet not shared by many modes of accidental death. This being so, investigations into the mechanism of death and its prevention can result in a high percentage of effective accident prevention.

This study involves primarily air embolism, which is lethal and for which there is ordinarily no treatment available. The phenomenon of "bends" has no relationship to scuba air embolism. Pneumothorax has only incidental importance in this investigation and does not seem to be a major factor in deaths from scuba diving in our area. However, the rupture of a lung cyst into the pleural cavity under changing conditions of pressure certainly can happen, and most emphatically, anyone with a cyst, congenital or acquired, should not be scuba diving.

In August, 1963, it became apparent to the Seattle coroner that special necropsy techniques are necessary to detect and indict air embolism as the cause of death in scuba fatalities. In this area bordered generously by water, where there is one boat for every six people, only 181 drownings occurred between 1959-1963. In roughly the same period (1959-1965) ten scuba death drownings were listed. This previous lack of awareness of the factor of air embolism as the possible causative factor in scuba deaths is not unique to this area.

A review in 1963 by Chappell of 33 scuba deaths in as many months in Florida showed instances in which the actual cause of death was not that listed on the death certificate. Persons submitting the death certificate on a diving fatality, unless knowledgeable about diving, may not be aware of other divers' maladies which can and do occur and erroneously report the death as a drowning. Chappell points out that air embolism is a most serious diving disease and can occur at almost any depth including water as shallow as seven feet; this points out a potential hazard even in swimming pools.

The Los Angeles Fire Department reported 42 scuba deaths from 1953-1960.

Chappell quotes the Navy as reporting only seven deaths in one 15-year period of experience in training scuba, a very low incidence considering the extensive diving conducted by the Navy, - a direct result of supervision, excellent training, discipline

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and availability of recompression tanks at the site of the diving.

One representative of the Navy reported seven episodes of air embolism in a recent three-year period of training at Pearl Harbor. Two apparently had "scars on their lungs," and one had a demonstrable bleb on the roentgenogram that was noted afterwards. Another four had mucous plugs due to a bronchitis. All of these incidents would seem avoidable by rigorous screening of applicants before being permitted to dive. Most of these seven deaths occurred during buoyant ascent training.

Miles of England reported that as of 1962, over 50,000 training ascents from 90 feet were carried out in the British Training Tank with only 25 accidents, all of whom recovered completely. In 19 of the 25 there was evidence of cerebral air embolism.

Investigation

This study by the Seattle-King County Safety Council of air embolism as a cause of scuba deaths in the Seattle area has the substantial support of the Seattle Police Harbor Patrol, Sheriff's Patrol, Coast Guard and Coroner's Office. Further, neighboring county coroners are also cooperating. Law enforcement agencies have been alerted to: 1) inform us immediately upon finding a scuba death body, 2) impound the body in a refrigerator, and 3) to impound the scuba gear, especially the air tanks for analysis.

A thorough study of the cerebral and coronary blood vessels, with photographic documentation is made. The lung is meticulously examined grossly and microscopically for the mechanism of rupture of alveoli, especially for pre-existent segmental or subsegmental bronchostenosis.

Figure 1: Scuba diving is distinct from skin diving, the latter being, for all practical purposes, swimming. The scuba diver carries his own air supply to substantial depths. The hard-hat diver is connected to a source of air on the surface, but is able to go to greater depths than the scuba diver. This report concerns the problems of the scuba diver.
A clinical history is reconstructed by interviews with survivors, family doctors, hospital records and any other source available. A special effort is made to obtain any and all chest x-ray films, including tuberculosis mobile unit films.

Generally, our Seattle-King County Safety Council research divers inspect and report underwater environmental details surrounding the fatality.

Gas analysis of the air in the tanks is done when possible to rule out contamination of the air, not as usual as might be thought, as a cause of death. The importance of this factor was emphasized by the University of Washington School of Medicine Environmental Health Division in 1963 in a report stating that in an examination of 25 scuba tanks for the presence of carbon monoxide, only two tanks showed no carbon monoxide present and 18 tanks had carbon monoxide concentrations between 10-25 ppm. Five of the samples were greater than 25 ppm with one sample one of 75 ppm. At present, some groups define breathing all for diving as showing the maximum carbon monoxide for decompression dives as 10 ppm and for non-decompression dives as 20 ppm.

Hemoglobin takes up carbon monoxide 200 times more readily than it does oxygen; carbon monoxide in small amount can be very dangerous.

The usual postmortem examination used in hospitals and coroners' offices will not give accurate evidence of air embolism in the brain and coronary arteries; an underwater technique must be used.

Variations have been described by Saphir, Lovell, and Aldape. Some method of immersing the body or the organ examined must be used. Aldape has emphasized that a deviation from routine necropsy procedure by opening the skull first to examine the brain for air emboli is important; opening the chest first will drain blood, possibly dislodging air emboli in the brain.

Walter of England recommends fixation of the lungs in distended position while

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**Figure 2**: Lethal air embolism can be the result of partial bronchial obstruction. The diagram on the left indicates air passing by an obstruction on its way to the alveoli. The figure on the left demonstrates the narrowing of the diameter of the bronchus on expiration, causing an obstruction with air being trapped distally. This ball-valve mechanism results in eventual overdistension of the alveoli with rupture. Air then escapes into accompanying torn veins and proceeds to the heart.
they are inflated by pumping informalin, followed by a formalin bath for ten days to find maximum lung pathology. Pre-
necropsy x-ray film of the chest, he suggests, may demonstrate a pulmonary cyst.
He, too, feels the necropsy should be done under water.

AIR EMBOLISM

Air embolism occurs when air from ruptured alveoli enters the blood stream and lodges as bubbles in vital structures, preventing the normal flow of blood to and through that structure, resulting in death of that area. Unfortunately, this frequently occurs in the brain and in the myocardium, two structures vital to life.

The prime mechanism in air embolism is rupture of alveoli in a segment or sub-
segment distal to an obstructed bronchus. In obstruction, with air pocketed distally, the volume of air in this space doubles every 33 feet that the diver rises, producing gross distension adequate to virtually explode the alveoli. The obstruction need not be absolute; if a ball-valve effect is present permitting ingress but greatly hampering egress, complete obstruction is present for all practical purposes.

There are a multitude of reasons for significant obstruction of a bronchus. Acute bronchitis is a common offender. Chronic inflammatory causes include granulomas, bronchiectasis, mycotic disease, tuberculosis, especially a calcified lymph node at a segmental or subsegmental bifurcation or a broncholith. Smoking causes an increase in mucus and a chronic inflammatory change in the bronchial lining. In the opinion of this author, no diver should smoke at any time; it is recognized that many do, but the hazard involved hardly seems worth it. Asthma is a distinct contradiction to diving. Tumors, benign and malignant, obviously can and will block a bronchus. Ob-
structive emphysema is a contraindication. Foreign bodies, such as vegetable matter, can cause chronic bronchial partial obstruction. Congenital cysts rule out diving also.

Unfortunately, a single posteroanterior roentgenogram of the chest may not demon-
strate the presence or hazards of these lesions. Inspiration, expiration and lateral roentgenogram views are mandatory. An area of greater or lesser radiotranslucency in the lung is highly suspicious of partial bronchial obstruction. Presence of calcium in a lymph node at a bifurcation of bronchi should be looked upon with concern in a diver. It has been advocated elsewhere that divers have chest x-rays while under several atmospheres of pressure to disclose possible partial bronchial blocks and cysts not seen by usual x-ray techniques; a suggestion with merit, especially in problem cases.

WORK BY OTHER INVESTIGATORS

Others also have felt that air embolism is a serious and lethal complication of scuba diving and that it can occur at surprisingly shallow depths.

Two cases of death of Navy submariners being trained in free ascent were reported by Liebow in 1959, one with a broncho-
lith in a subsegment of the superior seg-
ment of the right lower lobe and the other a case of emphysematous bullae. He feels that poor risks liable to involuntary air trapping are those with obstructive emphy-
sema, significant pulmonary calcification and chronic respiratory disease.

Miles quoted a report by Lambert in 1958 for a similar case in England.

Lamphier stated that a diver who holds his breath on ascent is immediately a can-
idate for one of the most serious of all diving accidents, air embolism. When a breath is held on ascent under these condi-
tions, the volume of expanding gas may readily exceed the normal capacity of the lung. The resulting increase in intrapul-
monary pressure then causes over-expansion of the lungs and rupture may follow. He reported that this is a serious accident it-
self, but that the main danger lies in the fact that excess intrapulmonary pressure can apparently force air into the pulmo-

nary circulation and send a stream of bubbles through the left side of the heart.

Lamphier also stated that air embolism can result from breath-holding in ascent even from depths of ten feet or less.
Duffner\textsuperscript{19} agrees that the most serious injuries seen in scuba diving are those in which the alveoli are ruptured, resulting from failure to equalize pressure during ascent from deep water.

Morriset,\textsuperscript{14} describing scuba diving in relationship to the practicing California physician, states that the most dangerous condition that may arise on ascent is air embolism and the source of air is invariably from the lung.

Air embolism during ascent from the bottom of a swimming pool is reported in the \textit{U.S. Navy Diving Manual}.\textsuperscript{14} This manual goes on to affirm our impression that lethal air embolism can occur in depths of only 15 feet. The \textit{Navy Diving Manual} points out, quite correctly, that the most common reason for air embolism is the diver holding his breath on ascent. This is due to the diver's lack of knowledge of physics and physiology, or due to panic—a normal response when one is under water and in trouble. As the diver ascends he must exhale, because gases behave according to Boyle's Law; as pressure becomes less the gas expands, resulting in overdistention of the lung, if not vented by adequate exhalation.

Miles,\textsuperscript{14} a Surgeon Captain of the British Royal Navy, remarked in 1962 that evidence of the true mechanics of many cases or air embolism is sparse and there is an urgent need for more information, especially with regard to local intrapulmonary obstruction which potential danger no amount of training in free ascent technique will remove. He also pointed out that structured weaknesses of the lung, such as emphysematous bullae, may well be a predisposing factor.

Walter\textsuperscript{7} reported in experiments with pigs that bullae enlarged with air trapped by an obstruction of various types resulted in rupture of these cysts. The resulting tear opened a pulmonary vein permitting air emboli to enter the arterial circuit of the body. He feels that bronchial mucus and irritants, particularly tobacco, are prime offenders. Diving is hazardous, in his opinion, for tuberculosis and asthmatic patients.

\textbf{TREATMENT OF EMBOLISM}

The only effective treatment is immediate recompression. The tank must be where the diver is. The diver cannot be transported. By the time he is moved, he is doomed to die. There is no other effective treatment. The theory behind immediate recompression is that the bubbles of air are put into solution and blood permitted to flow through the brain and myocardium again. Brain tissue is lost so rapidly that one has only seconds to act.

Of interest are the portable chambers that are now being produced that can be placed at areas where divers enter the water; some of these are relatively inexpensive.

Obviously, the best treatment is prevention. A very detailed physical examination should be carried out with particular attention to pulmonary disease history, careful examination of the chest and at least three-view chest roentgenograms.

\textbf{CONCLUSION}

Air embolism is suspected to be at least a prime cause of scuba fatalities in the Seattle area. This tragedy can occur at less than 15 feet submersion. It is suggested that partial bronchial obstruction in a segmental or subsegmental bronchus acts as a ball-valve, permitting with each successive inspiration greater distal distention. Rupture of the alveoli results in air entering torn veins, proceeding to the heart from there. Air embolism in the coronary and cerebral vessels causes anoxia and death. Types of partial bronchial obstruction are discussed. A thorough investigation of every scuba death in the Puget Sound area of the Pacific Northwest is being carried out.

Further study of the mechanism of the phenomenon of air embolism is indicated, coupled with promulgation of the facts to both physicians and lay personnel in this field.

Whereas immediate recompression is the only practical treatment of these casual-
ties, elimination of students of 'scuba diving whose history, physical examination and chest x-rays show hazardous bronchial changes is a vital preventative step.

A combination of adequate instruction plus a proper examination can reduce these accidents to the category of a rarity.

Resumen
La embolia gaseosa es considerada la causa más frecuente de accidentes fatales en el buceo “scuba” en el área de Seattle, EE.UU. Este trágico percance puede ocurrir en una sumersión de menos de 15 pies. Se ha sugerido que una obstrucción parcial de un bronquio segmentario o subsegmentario puede ejercer un efecto valvular, provocando una distensión distal que aumenta con cada inspiración. Eventualmente la ruptura alveolar da lugar a la penetración del aire en las venas desgarradas, de donde pasa al corazón. La embolización gaseosa de las arterias cerebrales y coronarias produce una anoxia que puede ser fatal. Los diversos tipos de obstrucción bronquial parcial son objeto de comentario. Todos los casos de muerte súbita durante la práctica del buceo en el área de Puget Sound en el Noroeste de la costa del Pacífico de los EE.UU. estan siendo cuidadosamente investigados. Es necesario proseguir la investigación sistemática del fenómeno de la embolia gaseosa y hacer llegar los conocimientos adquiridos a los médicos y personal lego interesados en este campo. Si bien la recomposición inmediata es el único tratamiento de estos accidentes, la eliminación del scuba de los individuos afectos de alteraciones bronquiales predisponentes constituye una medida preventiva de la mayor importancia.

La instrucción adecuada de los principiantes, en conjunción con el examen físico acucioso, han de reducir estos accidentes a la categoría de raras.

Zusammenfassung


Eine Kombination adäquater Unterrichtung mit einer gezielten Untersuchung kann diese Unfälle zu einer Rarität werden lassen.

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