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Mechanisms of Death in Shallow-Water Scuba Diving

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THE first practical forms of SCUBA (self-contained underwater breathing apparatus) equipment were designed for escape from sunken submarines and were modified during World War II for use in clearing mines from ports and in clandestine military operations, e.g., "frogmen". These Scubas were oxygen re-breather gear with CO_2 absorbent canisters and they have not gone into civilian use. The open-circuit compressed air gear now so widely popular stems from the work of Cousteau and Gagnan in France during and just after the German occupation. Initial interest in the development of Scuba arose then partly because of the military implications. After the Second World War, when efficient compressed-air gear became available, there was a rapidly expanding interest in Scuba diving as a sport, and at present there are an estimated eight million divers in the U.S.A. alone. The sport has serious hazards, but even in the best medical centres there are few physicians who have adequate understanding of the physiological changes which may be incurred while diving, or knowledge of the wide range of complications that may result.

CASE REPORT

R.W., a 21-year-old male flying cadet, was brought to the Emergency Department of the Royal Victoria Hospital, Montreal, about four hours after losing consciousness during a Scuba diving exercise in a standard-sized indoor swimming pool. It was reported that this young diver had been in excellent health; his annual chest radiograph and physical examination were within normal limits.

Observers at the pool-side had noted that the diver rose to the surface without his equipment, expired forcibly and then fell back into the water. He was brought to the pool-side by another diver approximately one minute later and rushed to the local infirmary, where he was found to be comatose and to have pulmonary edema; a blood pressure of 80/0 was recorded.

On arrival at the Royal Victoria Hospital, the patient had regained consciousness and had an accurate memory of events up to the time of entering the pool. He complained of severe retrosternal chest pain and shortness of breath. On examination pulmonary edema was noted and was confirmed by radiography. An electrocardiogram indicated a massive anteroseptal myocardial infarction. Neurological examination revealed complete right-sided hemiparesis and absent right abdominal reflexes. Management was symptomatic, but his condition deteriorated and terminally the patient was oliguric. Vigorous resuscitation efforts were undertaken for some two hours, but although initially successful, eventually failed. He was pronounced dead approximately 23 hours after the accident had occurred.

Fig. 1.—The left ventricular muscle is hyperemic with diffuse mottling throughout. (× ¾.)

The Scuba equipment used by the diver was found at the bottom of the pool where he had apparently discarded it. It was fully examined and found to be fault-free and in perfect working order. Reportedly the pool was standard in size with a 9-foot maximum depth.

Autopsy Findings

The body was that of a well-developed muscular man, measuring 70 ins. in height and weighing 172 lbs. There was marked cyanosis of the lips, ear lobes and nail beds.

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The heart weighed 450 g. No air could be aspirated from the ventricles. The foramen ovale was closed; the coronary arteries were present in their normal position and distribution. There was no evidence of atherosclerosis in the coronary vessels or aorta. The entire left ventricular wall, including the posterior papillary muscle and the anterior and posterior septum, was mottled (Fig. 1). On microscopic examination the cardiac muscle cells showed fragmentation; the cytoplasm was eosinophilic, with diffuse clumping and granularity. Well-preserved acute inflammatory cells and many erythrocytes lined the interstitial areas of the left ventricular muscle. The lungs were consolidated and firm to palpation. There were no definite signs of pulmonary, interstitial or mediastinal emphysema or pneumothorax. The left lung weighed 1000 g. and the right 1200 g. They were inflated with 20% formalin and cut into sagittal sections after fixation. Microscopic examination of numerous sections showed widespread acute bronchopneumonia, pulmonary edema and hemorrhage (Fig. 2).

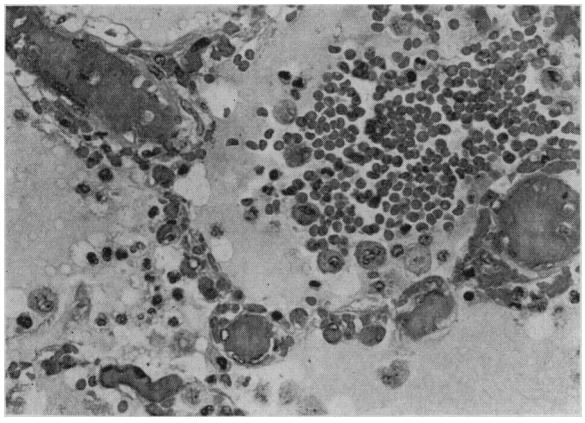

Fig. 2.—Microscopic appearance of the lung: the vessels are markedly congested. Intra-alveolar acute inflammatory cells are prominent. There is some intra-alveolar hemorrhage with diffuse intra-alveolar and interstitial edema. (Hemalum-phloxin-eosin, × 600.)

The left kidney weighed 160 g. and the right 170 g. Marked congestion of the calyces was noted. There was diffuse proximal tubular degeneration and widespread severe acute vascular congestion. The liver weighed 1850 g. and the spleen 360 g.; both were markedly congested. The brain weighed 1500 g. and was diffusely edematous. There was no evidence of cerebral infarction or laminar necrosis.

DISCUSSION

At the time of the accident the patient was engaged in a routine training procedure known as "ditching".[1] In this exercise the diver swims to the pool floor, takes a final breath from his tanks, abandons his equipment and rises to the surface; he then returns and re-dons the equipment. This procedure subjects the unschooled diver to the danger of lung rupture and pneumothorax, or air embolism. It is the diver who takes a full inspiration during the "ditching" procedure that runs the risk of lung rupture because his lungs are already filled to capacity. During training, candidates are advised by their instructors to take a full inspiration at the pool floor, then to rise slowly and exhale throughout the ascent (an action which is contrary to natural inclinations). Lung rupture with its consequences occurs because the regulator valve of Scuba equipment allows air into the lungs at ambient pressure. Thus, pressure in the lungs will exceed atmospheric pressure in proportion to the diver's depth. As he rises towards the surface, ambient pressure decreases and intrapulmonary air expands. This is according to Boyle's law, viz. $P_1 V_1 = P_2 V_2$. Therefore, the pressure volume product at any depth of water will equal the pressure volume product at the water surface. In the case under consideration, the volume at surface,

$$V_2 = \frac{P_1 V_1}{P_2} \text{ or } V_2 = 1.3 \times V \text{ (approx.).*}$$

A total lung volume of 5.5 l. (approx.) will expand to a volume of 7 l. (approx.) at the water surface. With the glottis closed, the volume increase in rising from a relatively shallow depth of 9 ft. is thus significant. Moreover, if this increase in volume were prevented by the resistance of the thoracic wall, the pressure in the alveoli would exceed that in the pulmonary vessels by more than 200 mm. Hg.

Apart from ignorance, fear and lack of instruction, the diver may also be subject to the same hazard through focal bronchial obstruction. This may either be congenital or acquired, and although clinically silent and radiologically inapparent, under diving conditions it may produce a ball-valve obstructive lesion. The diver with this deformity will be unable to rid himself of the focal expanding gas volume on rising to the surface and is therefore a candidate for pneumothorax and/or air embolism. There is little doubt that persons who have any special risk of ball-valve obstruction in the form of congenital or acquired lung disease should not engage in Scuba diving.

Presumably, on rising to the surface, increasing volume causes minute alveolar ruptures, and as the lung volume expands against a closed glottis there is a tendency for air to escape into the left heart circulation. This phenomenon is well illustrated in Duffner's monograph on

*Using Duffner's chart, the pressure at a depth of 9 ft. is approximately 1.3 x that at the surface.[2]

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underwater diving hazards.[2] The target organ, or organs, will depend entirely on the position of the diver at the time the air is released into the left heart circulation. While rather large volumes of air (80 to 100 c.c.) are needed in the right heart circulation to cause serious clinical consequences, small volumes can be fatal in the left heart circulation. As long ago as 1883 it was proved that moderate overinflation of lung parenchyma could result in air embolism without grossly apparent evidence of pneumothorax or severe lung damage.[3, 4] These experiments were carried out on animals and established that as little as 1 c.c. of air in the left heart circulation could result in serious damage to the central nervous system or the myocardium, depending on the position of the animal. Bichat demonstrated that when air is blown into the lung of a living animal, at a pressure no greater than that produced by the maximum expiratory effort of which the animal is capable, air will leave the alveoli and enter the pulmonary capillaries, provided that the pressure is maintained.[3, 6] Neuberger attributed the convulsions of whooping cough to cerebral air embolism from air forced directly into the bloodstream from the alveoli during a paroxysm of coughing.[7, 8]

In the case described in this paper, myocardial circulation was interrupted while the diver was still in the pool. We may assume that the actual occlusion happened as he reached the water surface. It is known from experimental work that coronary occlusion must exist for 15 to 20 minutes before infarction occurs.[5] Air is presumably quickly reabsorbed, and as death took place about one day after the accident occurred one would not expect to be able to aspirate air from the ventricles or to find it in the coronary arteries. No doubt the occlusion must in fact have existed and the air must have remained as an unabsorbed embolic occlusion in the left coronary artery for a length of time sufficient to produce the extensive infarction noted at autopsy. The widespread microscopic hemorrhage seen in most sections of the infarcted heart muscle implies that ultimately the air was completely reabsorbed and total coronary circulation was established. Presumably air emboli caused transient occlusion of the right internal carotid artery which resulted in transient hemiparesis.

THERAPEUTIC IMPLICATIONS

It is a well-known dictum among divers that they are their own best physicians. Medical men, by and large, are ignorant of the physiological hazards of underwater diving and therefore equally ignorant of the rationale of therapy. Besides air embolism, the following major hazards of diving have been described: drowning, usually secondary to physical exhaustion; the bends and asphyxia, suffocation or strangulation.[9] Squeeze or barotrauma is a hazard of the traditional helmet diver. It should not occur in Scuba diving unless the gear misfunctions, except locally as in face-mask squeeze affecting the eyes, and external ear squeeze from a closely fitting hood. Narcosis from CO_2 and oxygen poisoning are only remote possibilities with the modern equipment now used by most amateurs. Carbon monoxide and other toxic gases may get into the tanks, but probably overbreathing is a more frequent cause of clouded consciousness. Examples of the more minor hazards of Scuba diving include otitis (ruptured drum) and sinus barotrauma, disc herniation (from weight of gear in the dry), chilling and abrasions. The mechanisms involved are fully explained in the U.S. Navy Diving Manual.[9] Logical treatment in the case of air embolism is to shrink and dissolve the embolus. Recompression treatment should be put into effect at the very earliest suspicion of air embolism. Prevention of this hazard should be the prime objective of divers, who should be fully familiar with the physiological changes occurring in diving ascent and descent.

Summary A previously healthy young male cadet engaged in shallow water Scuba "ditching" exercises lost consciousness and died approximately 24 hours later. Having taken a full inspiration at the pool floor, the diver neglected to exhale; on rising to the surface, the intrapulmonary air expanded in accordance with Boyle's Law. Under such conditions, often in the absence of visible lung rupture, very small amounts of air may be forced into the left heart circulation and result in air embolism with serious clinical consequences. One of these complications, well illustrated in the case described, is "dysbaric cerebral embolism". In addition, transient occlusion of the coronary circulation in this patient led to extensive myocardial infarction. With the expanding interest in Scuba diving as a sport, it is important that physicians should acquaint themselves with the physiological changes that occur in diving, the potential hazards of this activity and the rationale of therapy.

Résumé Un jeune cadet, jusqu'alors en bonne santé et qui se livrait à des exercises de plongée libre en eau peu profonde perdit conscience et mourut environ 24 heures plus tard. Ayant pris une grande inspiration avant de plonger, il négligea

d'expirer; en revenant à la surface, l'air intra-pulmonaire se dilata suivant la loi de Boyle-Mariotte. Dans ces conditions, souvent même en l'absence d'une rupture pulmonaire visible, de très petites quantités d'air peuvent pénétrer dans le cœur gauche créant ainsi une embolie gazeuse qui peut avoir des conséquences cliniques graves. Une de ces complications, qu'illustre bien le cas qui nous occupe, est "l'embolie cérébrale dysbarique". En outre, chez notre malade, une occlusion transitoire de la circulation coronarienne a provoqué un important infarctus du myocarde. Etant donné l'intérêt croissant que l'on porte au sport de la plongée libre, il importe que le médecin se tienne au courant des modifications physiologiques qui surviennent pendant la plongée, des risques que peuvent courir les plongeurs et des mesures logiques à prendre pour traiter les accidents.

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Clinical Trial of a Broad-Spectrum Antimicrobial Preparation in Vaginitis

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INFECTIOUS vaginitis may be caused by many different pathogens. *Trichomonas vaginalis, Candida albicans* and a variety of bacteria are the organisms most commonly associated with the disease. More than one pathogen may be present, resulting in a mixed infection; for example, the concurrent appearance of Candida has been reported in 10 to 15% of trichomonal infections.[1, 2]

This multiplicity of etiological agents makes accurate diagnosis important to ensure adequate treatment of the disease. While each infecting organism is responsible for a characteristic clinical picture, diagnosis based on the clinical appearance is by no means wholly reliable, and for accuracy microbiological examination must be made.[3, 5]

The physician is thus presented with a dilemma. On the one hand, the patient usually presents with considerable discomfort and the desire for quick relief. On the other, accurate diagnosis is not possible without the time necessary for microbiological examination, making immediate treatment a problem. The typical mucopurulent discharge of bacterial (nonspecific) vaginitis, the frothy discharge of trichomoniasis or the curdy white discharge of candidiasis is not always present. In this situation a number of advantages could be suggested for the use of a broad-spectrum antimicrobial agent with activity against all the organisms commonly involved in vaginitis, either singly or collectively. The present study deals with the evaluation of such a preparation in a group of patients seen in a private practice in obstetrics and gynecology.

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MATERIALS AND METHODS

Forty-six patients were admitted to the study. Their ages ranged from 15 to 45 years, and they were all generally healthy, menstruating normally and presenting no obvious features of hormonal imbalance. An initial presumptive diagnosis was made on the basis of history, presence of itching, burning or discharge, physical characteristics of vaginal secretion and the gross appearance of the external genitalia, vaginal vault and cervix. No patient of the group admitted to allergy to sulfonamides when questioned directly on this point.

From the discharge present in the vagina, microscopic examination was made of wet mounts for Trichomonas and, after addition of potassium hydroxide, for spores and hyphae. The vaginal secretion was cultured by inoculation