FATAL FAT EMBOLISM FOLLOWING DECOMPRESSION SICKNESS IN AN EXPERIMENTAL DIVE

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Professor Bühlmann was unable to attend the Symposium to present this paper. Its contents are nonetheless considered pertinent to a study of osteonecrosis related to dysbarism, and hence the paper is included.

—Eds.

A simulated dive, involving two subjects, was undertaken at the Kantonsspital, Zurich, Switzerland, in February 1968. The subjects were compressed in 20 minutes to 31 ATA*, at which pressure they remained 3 hours. They were then decompressed to 1.0 ATA in 63.5 hours. The first subject experienced decompression difficulties only at the beginning of decompression and was asymptomatic thereafter.

The second subject, of concern to this report, was R.B., aged 22; he was 185.2 lb. (84 kg) in weight and 69.3 in. (167 cm) in height. Prior to the experiment no abnormal clinical findings were uncovered, other than a blood volume value of 56 ml/kg (normal value in the laboratory, 67 ml/kg). Hematocrit was 47%; total lipids were 505 mg %.

This subject experienced no difficulty during compression or during the period at maximum pressure. But on decompression he suffered "vertigo bends" at 18.5 ATA, which was relieved by recompression to 23 ATA.

During the final period of decompression, in which the breathing mixture was 50% O_2 and 50% N_2 at 2.6 ATA, the subject experienced mild pain in both knees. The pain dissipated spontaneously during decompression at the scheduled slower rate.

R. B. slept for five hours after decompression was completed, and then was mobilized for medical examination. His blood pressure at that time was 120/70; his pulse rate, 116; and his total lipids, 560 mg %. His blood volume had decreased to 46 ml/kg, his hematocrit had increased to 51%, and he showed evidence of dyspnea and cyanosis. Chest X-ray revealed a faint shadow on the left side. Because of R.B.'s increasing dyspnea and cyanosis, he was placed under intensive care. Such measures as intubation and artificial respiration with positive end-expiratory pressure (PEEP B) were undertaken, and plasma infusions were given. Arterial blood-gas levels, blood pressure, and diuresis were maintained satisfactorily.

On the third day of therapy the triglyceride level in the patient's blood was elevated to 328 mg % (normal, 30 to 190 mg %); chylomicron level was 111 mg % (normal, 1 to 3 mg %); and very low density lipoprotein was 217 mg % (normal, 30 to 190).

The patient suffered a cardiac arrest on both the second and third days of treatment, but was successfully resuscitated. Cardiac arrest occurred again on the sixth day; in this instance, unfortunately, efforts at resuscitation were unavailing.

Postmortem findings. Gross examination revealed a hemorrhage, 3 cm x 2 cm x 8 cm in size, in the bone marrow of the distal third of the left femur (Fig. 1). Microscopic study of the tissues revealed diffuse fat embolization of the lungs (Fig. 2), brain, heart, kidneys, and muscular system.

Diagnostic hypothesis. Hypovolemia, of unclear etiology, caused poor blood flow and tissue perfusion. The rate of gas elimination was inadequate and decompression sickness resulted. The decompression sickness then caused hemorrhage and fat-tissue destruction in the marrow of the left femur, which was followed by a diffuse, and fatal, fat embolization.

*A distinction should be made between references to *atmospheres absolute* as used by certain European and by U.S. and U.K. researchers:

1 ATA (as used here) = 1 atmosphere absolute, technical (or 735 mm Hg, which is also the normal atmospheric pressure at Zurich); whereas

1 atm abs = 1 atmosphere absolute, international, or 760 mm Hg

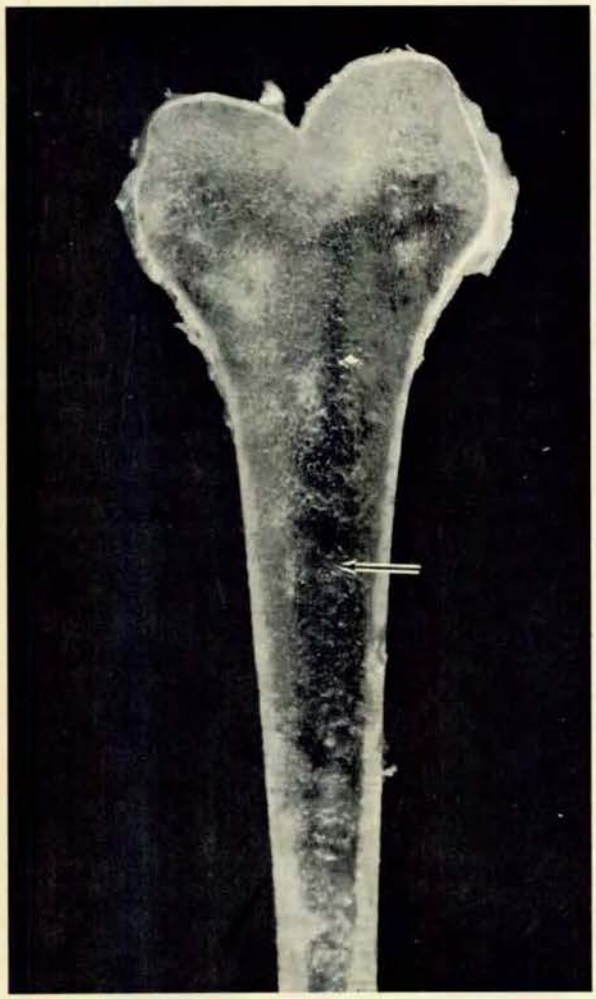

FIG. 1. Longitudinal section through distal third of left femur of patient R.B., showing intramedullary hemorrhage (arrow) in posterior segment.

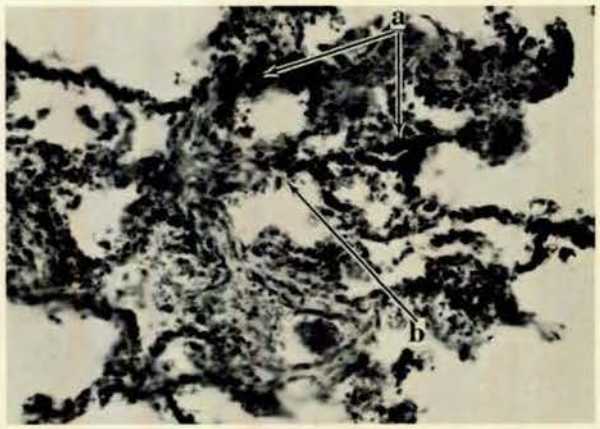

FIG. 2. Photomicrograph of lung tissue of patient R.B., showing vascular fat embolization; (*a*) droplets of fat in lung capillaries and (*b*) hemorrhagic extravasation.