

CLINICAL MEDICINE

Pulmonary Barotrauma After a Free Dive—A Possible Mechanism

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Pulmonary barotrauma during scuba diving is a life-threatening event. In a skin diver, who does not use compressed air, this complication is rare and its pathophysiology is not readily understood. We present a young, healthy skin diver who suffered pneumomediastinum and subcutaneous emphysema after a sequence of free dives to 5 m, and suggest a possible mechanism.

A SCUBA DIVER, who breathes compressed air, must exhale while surfacing, otherwise he or she will face the risk of pulmonary barotrauma as the air expands in the alveoli, possibly rupturing them. The air from the burst alveoli can enter the rich surrounding capillary network, causing arterial gas embolism, or gain access to other tissues, depending on the location of the trauma (e.g., mediastinum and subcutaneous spaces in the neck, causing subcutaneous emphysema; the pleural space, causing pneumothorax). During a free dive, however, lung volume decreases during descent in accordance with Boyle's law, and re-expands to its original volume upon surfacing. In such a case, therefore, pulmonary barotrauma is a rare event, and its mechanism is not readily understood. We report a young, healthy diver who suffered pulmonary barotrauma after a free dive and discuss a possible mechanism.

CASE REPORT

A 19-year-old male made a sequence of free dives to a maximum depth of 5 m. The patient denied breathing compressed air during his dives. His previous medical history was unremarkable. A thorough medical examination 16 months prior to the incident was normal, and

he was found fit to dive. About 2 h after returning from his dives he complained of dyspnea, neck pain, hoarseness, and later also dysphagia. Subcutaneous emphysema was found on examination, neurological examination was normal, and chest X-ray revealed pneumomediastinum without pneumothorax. He was admitted to hospital for overnight observation. The pain and emphysema gradually subsided. Repeated chest X-ray and computerized tomography of the chest 9 d later were normal. Spirometry 3 weeks after the pulmonary barotrauma, including a methacholine challenge test, was normal.

DISCUSSION

This apparently healthy young man suffered pulmonary barotrauma after an innocent sequence of free dives. His normal workup provides no explanation for the mechanism of his injury.

Spontaneous pneumothorax after swimming has been described very rarely (1-3,7). It might be speculated that some of these patients had been diving and not, as they thought, simply swimming. Bayne and Wurzbacher (1) describe a young man who collapsed and died minutes after surfacing from a free dive to a depth of 6 ft. On autopsy, large amounts of air were noted in the cerebral arteries and veins. Indeed, the victims of many accidents which occur every year around swimming pools and beaches are probably reported as drowning casualties; it is not known how many might in fact have suffered arterial air embolism secondary to pulmonary barotrauma.

Minor pulmonary barotrauma manifesting as spontaneous pneumomediastinum with uncharacteristic symptoms may also be missed. With the popularity of swimming and breath-hold diving in swimming pools, a considerable number of such cases may remain unreported.

Pierson (6) states that pneumomediastinum can sometimes be induced by acute changes in breathing pattern, resulting in an increase in lung volume or sudden pressure changes. Cadaver studies have shown that a gra-

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dient pressure as low as 95–110 cm H₂O is required to rupture the lung (5). Air is thought to gain entry to the mediastinum and subcutaneous tissue planes by rupture of marginal alveoli into perivascular sheaths and dissecting proximally.

Most cases can be managed by close observation, in order to intervene if airway obstruction is imminent. Even if the patient breathes air, the gas accumulated in the mediastinum should be absorbed. The rate of gas elimination is proportional to the "oxygen window," which builds up a pressure gradient. A simple way of speeding up this process is to administer normobaric O₂, which considerably increases the gradient. Compression therapy is not required, as the process is self-limited.

Arterial gas embolism (AGE) is a medical emergency. The only way of shrinking intravascular bubbles efficiently is by compression therapy in a hyperbaric chamber.

The mechanism of pulmonary barotrauma during a breath-hold dive is open to speculation. If the lungs are regarded as a passive reservoir inflated with air, Boyle's law will rule out the possibility of pulmonary barotrauma during a free dive. The fact that pulmonary barotrauma does occur after a free dive implies that during breath-holding there is a shift of air within the lungs, with accompanying minimal fluctuations of pressure, and partial or complete blockage of an airway leading to a lung segment into which air has been shifted.

However, the lungs are not a passive reservoir, because during a free dive they are subjected to considerable changes; i.e., blood shifts due to immersion, a pressure gradient along the vertical axis, with repeated body position changes leading to differential chest wall compression.

This proposed mechanism suggests two pulmonary pathologies: anatomic and functional. An emphysematous bulla is a lesion which may yield to minor fluctuations of pressure. Even during shallow dives, if a pressure gradient builds up, a bulla represents a lung

segment which is prone to rupture, with resultant pulmonary barotrauma.

On the other hand, hyperreactive airways may lead to a temporary blockage of air flow from a lung segment after it has shifted during the dive. This has no clinical significance in the normobaric environment. However, even a shallow dive to 5 m causes a 50% increase in pressure, with possible overinflation of the blocked lung segment upon surfacing.

In order to avoid the chance of recurrence, the patient was advised to refrain from activities involving possible exposure to extreme pressure changes, such as scuba diving, in which rapid decompression can cause life threatening pulmonary barotrauma. The Royal Navy's experience over a period of 20 years (4) indicates that recurrent pulmonary barotrauma tends to be more severe than the first incident, and hence the policy of advising divers who suffer pulmonary barotrauma to refrain from further diving.

We believe that a carefully selected experimental model may clarify the mechanism of pulmonary barotrauma in a free dive.

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