Hemoptysis and pneumomediastinum after breath-hold diving in shallow water: A case report

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ABSTRACT
We report the case of a healthy 21-year-old woman who performed iterative breath-hold dives in relatively cold water, not exceeding depths of 5 meters but with “empty lungs.” At the end of a dive, after experiencing an intense involuntary diaphragmatic contraction underwater, she presented hemoptysis followed by chest pain and cough. Chest radiography and computed tomography were performed 24 hours later, confirming the diagnosis of pneumomediastinum. The clinical course was benign: However, chest pain and effort dyspnea lasted for a few weeks. The pathophysiology of this accident may be explained by a combination of mechanisms involved in several clinical entities, namely pulmonary edema of immersion, pulmonary barotrauma and spontaneous pneumomediastinum.

INTRODUCTION
Pulmonary barotrauma and edema after underwater immersion have been described as a consequence of exposure to environmental stress and variations of intrathoracic pressures. Rather uncommon in breath-hold divers, these adverse events may cause failure of the blood-gas barrier with intra-alveolar hemorrhage and other symptoms, such as mediastinal emphysema [1,2].

We report the case of a young healthy woman who suffered hemoptysis and chest pain, revealing a pneumomediastinum, after a series of breath-hold dives with “empty lungs” in shallow water. The diagnosis and possible injury mechanisms of this condition are discussed.

CASE REPORT
A 21-year-old woman performed numerous breath-hold dives to a depth of about 5 meters over a period of two hours in relatively cold water (10°C). She was healthy, albeit overweight, not a current smoker, and she had 10 years of trouble-free experience in scuba diving. She exhaled before breath-hold diving in order to stabilize herself at the bottom in order to take underwater photographs, and between dives, swam at the surface without physical effort. Before ascent at the end of a dive, she felt an intense involuntary diaphragmatic contraction, which she later on compared to hiccup.

Upon surfacing, the presence of blood in her snorkel was evident and she interrupted her diving session soon afterwards. A few minutes later, she experienced retrosternal chest pain and mild cough with hemoptysis. That evening, she took paracetamol without improvement. The next day, because hemoptysis occurred once again and chest pain was still intense, she sought medical evaluation.

In the emergency room, the pulse oximetry and physical examination were normal: In particular, there was no evidence of dyspnea, dysphagia, voice alteration or subcutaneous emphysema. However, a chest radiograph revealed a pneumomediastinum confirmed by the chest CT scan (Figure 1, Page 214). Bronchoscopy showed diffuse inflammation of the airways.

The patient was discharged from the hospital after a few hours, with paracetamol as the sole treatment. Residual chest pain and effort dyspnea lasted for a few weeks. A chest CT scan performed three months later showed no abnormality.

DISCUSSION
The present case is remarkable in that hemoptysis was found associated with pneumomediastinum. These signs occurred after breath-hold diving to shallow depths with “empty lungs,” probably as a consequence of a combination of pulmonary edema and barotrauma. That the
bleeding was alveolar in origin, and not from the upper airways, larynx or sinus, can be ascertained both by the clinical presentation (association with cough, recurrence a few hours apart) and by the bronchoscopic examination. Initial hemoptysis is clear evidence of primary failure of the blood-gas barrier, which allowed air leakage and dissection throughout the bronchovascular sheath toward the mediastinum, thereby explaining the acute retrosternal chest pain felt secondarily.

Several plausible pathophysiological mechanisms exist to explain the lesions of the blood-gas barrier occurring during breath-hold diving. Increased central blood pooling (immersion plus exercise) added to the hemodynamic modifications of the diving response and of cold exposure (bradycardia, vasoconstriction and hypertension) contribute to the pressure elevation in pulmonary capillaries [3]. As a result of abnormally high transmural pressure, the capillary endothelium and alveolar epithelium can disrupt, leading to edema and hemoptysis [2,4]. In a report of three cases of alveolar hemorrhage, the breath-hold divers had taken aspirin, which may have aggravated the bleeding [5], but no drug was ingested before diving in our case.

A factor that may have triggered the pulmonary capillary stress failure in the present case is the violent diaphragmatic contraction experienced during a “struggle phase” of the last dive series, and the resulting negative intrapulmonary pressure. Such negative pressure effects have also been involved in the alveolar hemorrhage reported in three subjects diving beyond the start of diaphragmatic contractions endured at depth [6]. In this respect, Lambrechts et al. [7] speculated that intense involuntary diaphragmatic contractions could contribute to the increase of extravascular lung water (as measured by ultrasound lung comets) observed in deep, shallow maximal and shallow dynamic apneas.

Finally, diving after exhaling as did our diver in order to stabilize at shallow depth, is likely to have increased the strain on the pulmonary capillaries through the thorax/lung-squeeze effects that characterize deep diving [3]. Indeed, in an experimental study of 11 healthy subjects who dove to 6 meters after full exhalation to residual volume in order to simulate thoracic compression of deep diving, pulmonary edema was suggested by reduced dynamic spirometry, and slight hemoptys-
sis was noted in two subjects [8]. Obviously, a number of the above mechanisms are common with those at work in the so-called pulmonary edema of immersion reported in connection with scuba diving [9], strenuous swimming and breath-hold diving [10]. The latter meta-analysis comprised eight breath-hold divers (among 60 cases compiled), of whom three had taken aspirin and/or endured voluntary diaphragmatic contractions. Two of these cases were fatal, with alveolar hemorrhage and interstitial edema found at autopsy but no evidence of gas emboli or drowning [10]. It is noteworthy that none of the patients complained of chest pain in this series.

The pneumomediastinum that occurred in our patient after the hemoptysis is likely to be due to some degree of pulmonary barotrauma of ascent. The latter condition is thought to be quite uncommon in free divers who did not breathe at depth from a compressed gas source. The hypothetical mechanisms on rapid ascent involve:

(i) shear forces between lung compartments due to non-uniform reduction in their compliance; and

(ii) airway closure and air trapping in some areas of the lungs that become overdistended to the point of rupture (2).

Following alveolar disruption, air first enters the perivascular interstitium and dissects proximally within the bronchovascular sheath toward the mediastinum along a pressure gradient; then it may decompress into cervical soft tissues, to the retroperitoneum or even cause pleural rupture producing pneumothorax [11].

There are somanecdotal reports of pulmonary symptoms (including pneumomediastinum) with a benign self-limited course that appeared shortly after breath-hold dives and were attributed to pulmonary barotraumas of ascent. Predisposing factors such as lung blebs or asthma were present in some of these cases [1,12,13]. Besides, the possibility of fatal arterial air embolism due to this mechanism is illustrated by the well-documented case of a young healthy male performing forceful inspiration before swimming underwater at a 2-meter depth [14].

Similarly, transient tetraplegia attributed to cerebral air embolism was recently reported in a breath-hold diver who overinflated his lungs with glossopharyngeal insufflation prior to a dynamic apnea swim [15]. This maneuver of glossopharyngeal breathing (lung packing), routinely practiced by competitive free divers to add extra air to the lungs on top of a full inspiration, can increase the transpulmonary pressure up to 8 kPa, causing lung damage in itself [3].

Interestingly, a pneumomediastinum that was transient and asymptomatic has been detected on CT scans in five of six trained subjects performing lung insufflation independently of diving [16]. In our diver, no such provocative maneuver or risk factor could be found. However, as discussed above, she probably presented prior stress failure of the alveolar barrier that favored air escape into the mediastinum during ascent or shortly thereafter.

The mechanisms of barotraumatic alveolar rupture and dissection of air along the tracheobronchial tree are similar to those seen in spontaneous pneumomediastinum, a benign entity that usually affects healthy young adults without any obvious etiology such as iatrogenic injuries or infections [11,17,18]. Actually, the most common triggering events of spontaneous pneumomediastinum have been forceful emesis, asthma flare-ups, intense coughing and physical exertion during sports [17, 18,19]. The most frequently reported clinical manifestations are chest pain, followed by shortness of breath and subcutaneous emphysema sometimes associated with cervical pain, voice distortion or dysphagia, the latter symptoms being absent in our diver. Because spontaneous improvement is the rule, it is likely that a significant number of minor cases are undiagnosed, which may explain the paucity of reports [17,18].

The same probably holds true for both pulmonary barotrauma and pulmonary edema in free divers. Despite an apparent low frequency of the latter two conditions, they should be considered in case of suggestive respiratory signs in conjunction with free diving, because they require a high index of suspicion. Chest CT scan (more sensitive than conventional radiography) is an important diagnostic tool to reveal mediastinal and/or pleural air, early ground-glass opacifications in case of edema, as well as to rule out pre-existing lung disease. In our case, the CT scan showed only a pneumomediastinum, probably because it was performed too late (24-hour delay) to show minimal lesions of the blood-gas barrier, known for their rapid healing in experimental models [4]. Any pulmonary edema or barotrauma in a diver is cause for concern, because not all cases have a benign course. Instead, unpredictable life-threatening complications may ensue before the diver is rescued from the water [9,11,14,15]. We suggest that, during breath-hold diving, some fatal accidents (misreported as “drownings”) may be attributable to the aforementioned pathophysiological mechanisms.
REFERENCES


