Should computed chest tomography be recommended in the medical certification of professional divers? A report of three cases with pulmonary air cysts

A S Toklu, E Kiyan, S Aktas, M Cimsit

Pulmonary barotrauma (PBT) is a recognised risk of compressed gas diving. Any reason that causes air trapping in the lung during ascent may cause PBT by increasing intrapulmonary pressure. Chest x-ray examination is mandatory for medical certification of the professional divers in many countries, but pulmonary air trapping lesions such as an air cyst in the lungs cannot always be detected by plain chest x-ray exam. Computed tomography (CT) is a reliable, but expensive measure for detecting pulmonary abnormalities in divers. Three cases with pulmonary air cysts are reported in which air cysts were invisible on the x-ray pictures, but well defined by CT. It is impractical and not cost effective to perform CT for medical certification of all divers, but it can be an option to recommend CT once during the initial examination of the candidates for professional diving, especially if there is a history of predisposing factors, such as smoking or pulmonary infections.

There are several medical standards in different countries for initial or periodic certification of professional divers. These standards are slightly different than each other and established to ensure that only fit candidates receive or renew their dive certification. Chest x-ray examination is mandatory in many countries, but the intervals vary from annual to every five years. One of the purposes of the chest x-ray examination is to check the presence of lung cysts, emphysematous blebs, or bullae, which might predispose pulmonary barotraumas (PBT) and preclude diving.

PBT is a risk for divers who breathe compressed gas while immersed at pressure. On ascent the gas in the air containing cavities of the body will expand, in accordance with Boyle's Law, since environmental pressure is reduced during ascent. A volume of gas at 10 m, where the pressure is 2 atmospheres absolute (ATA), will double at the surface since the pressure is decreased to 1 ATA. During ascent, the expanded gas in the lungs must be exhaled to prevent pulmonary overinflation. Any reason that causes air trapping in the lungs or breath holding during ascent may cause PBT by increasing intrapulmonary pressure. The air trapped in the lung can rupture the alveoli by expanding during decompression. Thus, the gas in the ruptured alveoli might escape to the interstitial space, track along perivascular sheaths, and cause mediastinal emphysema, pneumothorax, or subcutaneous emphysema. If the gas enters the pulmonary circulation via torn vessels next to the ruptured alveoli, it goes to the left heart and causes arterial gas embolism (AGE). AGE is the most dangerous complication of pulmonary barotrauma and is a major cause of mortality in diving. In a study of fatal diving accidents, 31% of the deaths were attributed to AGE.

Subpleural emphysematous blebs or air cysts may predispose to PBT since they may cause air trapping. Existence of air trapping lesions in the lung is therefore a reason for disqualifying divers. These lesions might be missed on plain chest x-ray examination, but can be well defined by chest computed tomography (CT). In this paper, three cases with pulmonary abnormalities are reported in which pulmonary air trapping lesions probably expanded during ascent, and resulted in PBT. The lesions were verified by high resolution computed tomography (HRCT) in all cases whereas the abnormalities could not be detected on plain chest x-ray pictures.

The cases reported in this paper were referred to the Department of Underwater and Hyperbaric Medicine, Istanbul Faculty of Medicine; CT (Siemens Somatom Plus) examinations were performed with 1.5 mm slice thickness HRCT scans at the Radiology Department of the Faculty.

**CASE 1**
A 56 year old male professional underwater photographer with 15 years of diving experience; smoker (20 cigarettes a day for 20 years).

He dived to 35 m and noticed he was out of air after 35 minutes at the bottom. He started ascending rapidly since he was alone. He experienced no problems when he surfaced. The next day he felt discomfort while breathing during his dive to 20 m and interrupted the dive. He had a pain in his chest and was dyspnoeic when he arrived at the surface. He did not dive for 12 days as the pain persisted. He underwent medical examination.

A pneumothorax on the left side was detected by chest x-ray examination (fig 1). A large bulla, located on the apical part of the left lung and in connection with pneumothorax, was detected on HRCT (fig 2). Normobaric 100% oxygen was administered for four hours a day for six days; the pneumothorax had subsided at control chest x-ray examination. He was excluded from diving.

**CASE 2**
A 36 year old male professional diver with 15 years of diving experience; smoker (25–30 cigarettes a day for 10 years).

He performed a dive to 38 m with 30 minutes bottom time, and obeyed decompression stops according to US Navy Standard Air Decompression Table as if he had dived 140 feet (42.6 m). He felt pins and needles in his right arm while he was in water at the 3 m decompression stop. He did not feel his right arm and could hardly stand with assistance when he

**Abbreviations:** AGE, arterial gas embolism; ATA, atmospheres absolute; CT, computed tomography; DCS, decompression sickness; HRCT, high resolution computed tomography; PBT, pulmonary barotrauma
came to the platform, after completing his decompression stop. After a few minutes he collapsed with hemiplegia on the right side, but there was no alteration of consciousness. There was no cough or any problem with breathing. The ascent was uneventful; the rate was about 10 m/min. The dive was his 62nd dive with similar profiles during an underwater pipeline installation. He had had no problems during his previous dives. Immediate recompression therapy was performed in 10 minutes according to US Navy Table 6. The symptoms started to decrease in the first few minutes and disappeared completely in the first 20 minutes at a depth of 60 feet in the recompression chamber.

Physical examination revealed no pathological findings at the end of treatment. Pulmonary barotrauma was suspected rather than decompression sickness. Investigation was performed for a possible precipitating factor for PBT in the lungs. HRCT of the chest showed an azygos lobe on the right side and multiple air cysts bilaterally (fig 3), which were not visible on the chest x-ray pictures (fig 4). He was excluded from diving.

CASE 3
A 25 year old male professional diver with six months diving experience.

He performed a scuba dive, and spent 35 minutes at 9–10 m after staying one minute at his maximum depth of 17 m. He surfaced slowly after spending three minutes at 5 m. He was dizzy and complained of visual disturbances, with numbness in the right leg, a few minutes after surfacing. The symptoms disappeared spontaneously within 10 minutes. He performed another dive with a similar profile the next day and had no problems. Three days later he performed a dive and spent 14 minutes at 37 m. He ascended slowly to 1–2 m in 10 minutes uneventfully, but did not remember surfacing from 1 to 2 m. He was unconscious at the surface. He became conscious 30 minutes after surfacing.

Physical examination revealed motor and sensory disturbances of the lower extremities when he reached the hyperbaric facility 48 hours later. He underwent repetitive recompression therapies to achieve complete recovery. HRCT of the chest revealed a small bleb on the upper part of the right lung, which could not be detected by chest x-ray examination. He was excluded from diving.

DISCUSSION
Pulmonary barotrauma is a recognised risk of compressed gas diving, which occurs mostly among inexperienced divers, as they might hold their breath during ascent, because of panic. Studies of accidents during submarine escape training have also revealed that the incidence of PBT among initial trainees is higher than among requalifiers. PBT occurs during ascent when there is either breath holding or regional gas trapping, resulting in an increase in intrapulmonary pressure. Some reports have stated that transpulmonic pressures of 60–70 mm Hg are sufficient to cause rupture of alveolar septa and allow escape of gas into interstitial spaces.

The onset of clinical symptoms of AGE is usually within the first minutes of surfacing. Leitch et al reported that onset time
of symptoms after surfacing was less than five minutes in 97% of their 117 AGE cases. In two of our cases the symptoms appeared while the divers were still in water.

In the first case the diver made rapid ascent in a dive but did not have any symptoms afterwards. He had symptoms after the second dive he performed on the next day. It is known that some PBT cases might be asymptomatic. It is more likely that an asymptomatic PBT occurred during the first dive, which became symptomatic when the diver dived on the next day. Probably the pneumothorax, which developed during the first dive, became symptomatic during the second dive.

Involvement of the central nervous system is common in cases of pulmonary barotrauma. In two of our cases there were symptoms that might be related to central nervous system involvement. A pneumothorax was evident in the dive who had no neurological symptoms.

Some predisposing factors, such as pulmonary infections or smoking, may increase the likelihood of emphysematous changes in the lung. There was a history of smoking in two of our cases, which might have contributed to the formation of bullous lesions in the divers’ lungs. It is especially important to investigate such lesions by HRCT, if the divers have history of smoking or lung infection.

It is evident that not all emphysematous bullae are visible on plain chest x-ray examination, but can be seen clearly on CT scans. Air cysts or blebs were detected in all of our cases on HRCT scans, but the lesions were not visible on the x-ray pictures. A pneumothorax was clear on the chest x-ray picture in case 1, but there was no image indicating a bulla.

Existence of subpleural blebs, pulmonary air cysts, or an emphysematous bulla should disqualify a subject from compressed air diving, as they may facilitate PBT to occur. There have been several reports where preexisting medical condition predisposed to PBT. Aktas at al reported four cases of PBT in which pulmonary lesions were detected on CT scan. They suggested that in doubtful decompression sickness (DCS) cases, CT scanning of the chest must be performed not only for differential diagnosis but also for disqualification.

We conclude that screening the divers by chest x-ray examination is not sufficient to detect all possible air cysts in the lung and may be excluded from the routine examination. The reliable measure for detecting pulmonary abnormalities during medical assessment of the divers is HRCT. However, it is impractical to perform CT for both recreational and professional divers, because of its cost and lack of facilities. It can, however, be a good decision to recommend performing it only during the initial examination of the professional divers, especially if there is a history of predisposing factors such as smoking or pulmonary infections.

Authors’ affiliations
A S Toklu, S Aktas, M Cimsit, Istanbul University, Istanbul Faculty of Medicine, Department of Underwater and Hyperbaric Medicine, Istanbul, Turkey
E Kiyanoğlu, Istanbul University, Istanbul Faculty of Medicine, Department of Respiratory Disease, Istanbul, Turkey

Correspondence to: Dr A S Toklu, Deniz ve Sualtı Hekimliği AD, Istanbul Tip Fakültesi, Capa İstanbul 34590, Turkey; akin@toklu.net

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