The diving doctor's diary

Editor's note:

Pulmonary barotrauma (PBT) and its consequences are well recognised in scuba diving, but less so in association with breath-hold diving. The same may be said for iatrogenic PBT and arterial gas embolism (AGE), and failure to recognise these may (and sadly often does) lead to tragic consequences. The five cases reported in the following articles, and a later letter to the Editor regarding retrograde venous gas embolism have been put together to remind clinicians to always be on the lookout for these complications. I have myself managed breath-hold divers who have taken a breath from a scuba regulator at depth, leading to AGE, and iatrogenic cases similar to the one reported by Janisch et al.

Pneumomediastinum or lung damage in breath-hold divers from different mechanisms: a report of three cases

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Abstract

(Toklu AS, Erelel M, Arslan A. Pneumomediastinum or lung damage in breath-hold divers from different mechanisms: a report of three cases. *Diving and Hyperbaric Medicine*. 2013 December;43(4):232-235.)
Normally pulmonary over-inflation is not an issue during breath-hold diving, in contrast to lung squeeze. Compared with compressed air diving, pulmonary barotrauma is rare in breath-hold diving. Several mechanisms can lead to an increase in intrathoracic pressure in breath-hold diving that may cause alveolar rupture. Here we report three cases of pulmonary barotrauma in breath-hold diving. Using high-resolution chest tomography, bullous damage in Case 1, and pneumomediastinum in Cases 2 and 3 were detected. Transient neurological symptoms in Cases 1 and 2 suggested cerebral arterial gas embolism. The mechanisms that caused intrapulmonary overpressure were, respectively, lung packing ('buccal pumping'), considerable effort and straining at depth, and breathing compressed air at depth and ascending without exhaling. All three cases recovered without specific treatment such as recompression.

Key words
Breath-hold diving, freediving, pulmonary barotrauma, arterial gas embolism, cerebral arterial gas embolism (CAGE), buccal pumping, case reports

Introduction

Breath-hold diving is associated with a variety of physiological responses induced by immersion, apnea and lung compression.[1] Normally pulmonary over-inflation is not an issue during breath-hold diving, in contrast to lung squeeze.[2] Compared with compressed air diving, pulmonary barotrauma (PBT) is rare in breath-hold diving. Nevertheless, several mechanisms can lead to an increase in intrathoracic pressure.[2] Alveolar rupture can cause pneumothorax, pneumomediastinum, surgical emphysema and arterial gas embolism (AGE) and may occur when intrapulmonary pressure is higher than environmental pressure, such as in divers and aviators or airline passengers and mechanically ventilated patients.[3,4] PBT may also be caused by repeated bearing down to increase abdominal pressure, sneezing, vomiting and oesophageal rupture. PBT is especially a risk for divers using scuba or surface-supply breathing apparatus (SSBA). Gas may escape from ruptured alveoli to the interstitial space, tracking along perivascular sheaths or entering the pulmonary circulation via torn pulmonary vessels to reach the left heart, leading to AGE.[5] Cerebral (CAGE) or coronary AGE are the most dangerous complications of pulmonary barotrauma and CAGE is a major cause of mortality in diving.[6]

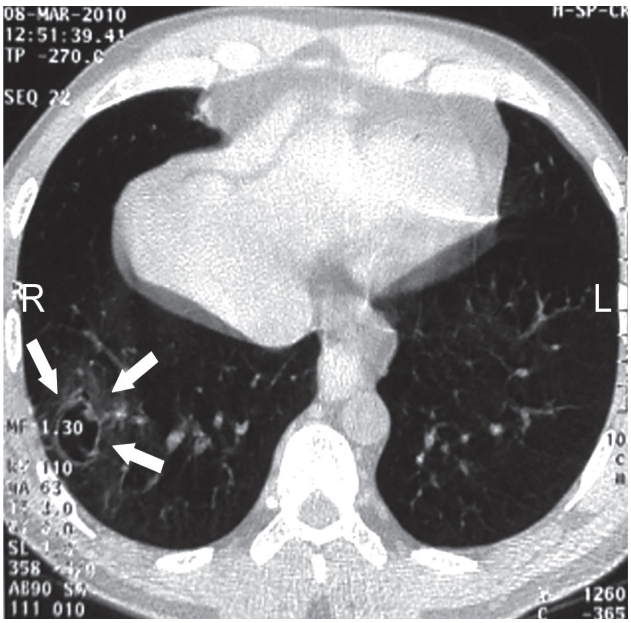
We report three cases of breath-hold (BH) divers who suffered PBT from three different but well-documented mechanisms and presented between 2010 and 2012. Patients gave their verbal consent for their cases to be reported.

Case reports

CASE 1

A 40-year-old, otherwise healthy, male, competitive breath-hold diver, who was in the habit of lung packing (glossopharyngeal insufflation or 'buccal pumping') prior to breath-holding, made a dive in a swimming pool to a depth of approximately 1.5–2 metres of water for dynamic apnea training. He performed lung packing for 20 seconds prior to the dive. He felt dizziness, nausea and numbness all over his body 10 seconds after his dive started, and was unable to control the right side of his body and had visual disturbances

Figure 1
Case 1: a single HRCT cut showing bullous damage in the right lung



when he surfaced. After about an hour, all symptoms had resolved spontaneously. On presentation two days after the incident, physical examination was unremarkable except for rales at the right lung base. High-resolution chest tomography (HRCT) revealed bullous damage in the right lower lobe (Figure 1). Cerebral magnetic resonance imaging (MRI) was normal. He was advised not to dive until a repeat HRCT three months after the incident, which revealed no pathology. He was advised not to use manoeuvres such as lung packing that increase transpulmonary pressure.

CASE 2

An 18-year-old, male breath-hold diver made four dives to about 18 metres' sea water (msw) depth, each lasting 45–60 seconds, with 1–2 minute surface intervals. Then after about an hour, he made another ten dives to 6–7 msw depths to release a rope that was stuck underneath a rock at the bottom. This involved considerable effort and straining. He did not perform lung packing before any of the dives. He had pain and a sensation of pressure in his chest for 30 minutes after his last dive and noticed swelling around his neck that was crepitant on palpation. He was diagnosed as having a pneumomediastinum, confirmed by HRCT (Figure 2). The symptoms disappeared within two days without treatment. Repeat HRCT 20 days after the incident revealed no pathology.

CASE 3

An otherwise healthy, 36-year-old man made three breath-hold dives to 3–5 msw without any problems. He made a final dive to 9 msw, during which he breathed

Figure 2
Case 2: a single HRCT cut showing mediastinal and cervical emphysema

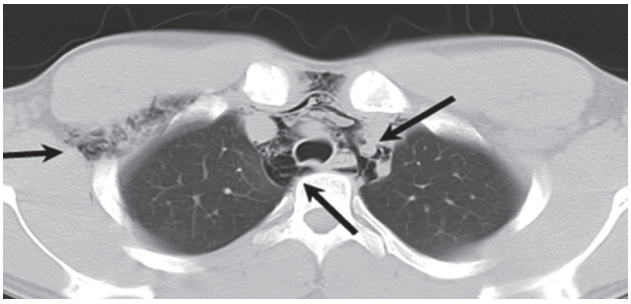
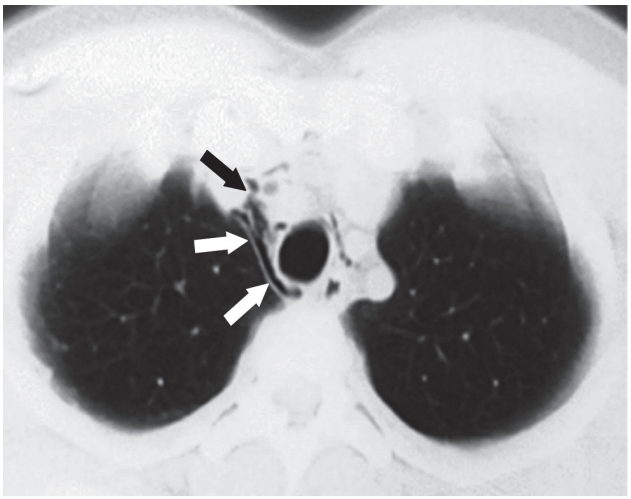


Figure 3
Case 3: a single HRCT cut showing mediastinal emphysema



from a friend's scuba 'octopus' regulator before ascending to the surface. During the ascent, he did not exhale and he felt pressure in his chest at 4–5 msw. Some air escaped from his mouth involuntarily when he was about to surface. He felt retrosternal pain and jerking and abnormal sensation over the right side of his torso and leg when he reached the surface. He was lifted into the boat with difficulty, where his right leg felt weak and he was dysarthric, which subsided within about 10 minutes. His symptoms, which clearly indicated CAGE, almost completely disappeared within 30 minutes except for the chest pain. Thirty minutes after the incident, oxygen via nasal prongs and an intravenous infusion of electrolytes were commenced and he was given dexamethasone 8 mg intramuscularly. The patient reported that his retrosternal pain decreased within 30 minutes following the initiation of this treatment.

A diving physician recommended by phone that he have HRCT and be transferred on 100% oxygen for recompression treatment on the presumptive diagnosis of PBT/CAGE. HRCT was normal. He was discharged the same day to present the next day to the

Department of Underwater and Hyperbaric Medicine, İstanbul Faculty of Medicine, since he still had slight chest pain while breathing. There were no pathological findings on physical examination. However, mediastinal emphysema was detected on his HRCT (Figure 3). Repeat HRCT three weeks later was normal. He was advised not to resume breath-hold diving for six months.

Discussion

The first diver was a sport breath-hold diver who competed in the discipline of dynamic apnea (underwater swim in a swimming pool with or without fins for as great a distance as possible on a single breath-hold). For this, breath-hold time is critical, and some athletes perform various manoeuvres, such as hyperventilation or lung packing, to extend their breath-hold time. Vital capacity can be increased by more than 30% by lung packing, which can increase transpulmonary pressures by up to 80 cm H_2O, and lung rupture may occur.[7] There are several reports of lung damage after lung packing.[7–9] In our first case, the initial symptoms were neurological and it is likely this was the result of CAGE from air entrainment into the pulmonary veins from an area of ruptured alveoli. CAGE may be fatal, so it makes sense to discourage BH divers from performing lung packing before diving. The bullous damage in the lung healed within three months with no bulla being detected on HRCT. There are a few case reports showing spontaneous collapse of bullae in non-divers in long-term follow up.[10,11] On the other hand, the cystic lesions did not resolve in long-term follow up in divers with pulmonary barotrauma in another study in which the authors assumed that the lung cysts or blebs were pre-existing conditions which had led to the pulmonary barotrauma.[12] In another series of three cases, bullae were seen to increase in diameter, seemingly related to diving.[13] In our case, the bullous lesion was likely owing to trauma from overpressure.

In the second case, the cause of the diver's lung injury was probably straining against a closed glottis during his efforts to free the jammed rope.[14] Underwater tasks that require physical effort and straining should not be done by breath-hold diving but rather using scuba or SSBA.

Lung over-inflation is not normally expected in breath-hold diving, but the third diver practised something completely outside of BH diving rules in breathing from scuba equipment at depth. Having done so, if the diver continues to hold his breath, the decrease in ambient pressure during ascent results in an increase in intrathoracic gas volume and lung rupture, which may be fatal. This diver was not a scuba diver and, therefore, he was not aware of the risk of breathing compressed air at depth and then ascending whilst still holding his breath.

For the same reason, free emergency ascent was abandoned as part of scuba training about 20–25 years ago.[15] Free ascent from 18 msw is still part of submariner training in the controlled environment of a submarine escape training tank. The risks associated with this training are well documented.[16,17]

In PBT cases, 100% oxygen administration is an important component of first aid. However, nasal prongs are an unsuitable mode of delivery as they provide considerably less than 100% oxygen. Oxygen should be given by mask with reservoir bag or other 100% oxygen delivery system in such cases. Many PBT cases recover spontaneously, as in all three of these divers.[18] However, it is important that, where there is a suspicion of PBT, a recompression facility should be sought since the diver's clinical condition may deteriorate secondarily and sufficiently to be life-threatening. Consideration should be given to recompression treatment for all PBT cases with neurological symptoms or signs, however transient, as in Cases 1 and 3. At the very least, the diver should be monitored carefully.

Symptoms and signs of PBT usually appear within minutes to an hour after the dive.[4] While symptoms like retrosternal pain and coughing may point to pneumomediastinum, and CAGE may cause neurological symptoms, sometimes PBT cases may be asymptomatic. HRCT is a valuable tool in determining the extent and presence of mediastinal emphysema, as plain chest radiographs may miss a small pneumomediastinum.[19]

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