Case report

Influence of scuba diving on asymptomatic isolated pulmonary bullae

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Key words
Pulmonary barotrauma, arterial gas embolism, fitness to dive, risk assessment, medical conditions and problems, case reports

Abstract

(Germonpré P, Balestra C, Pieters T. Influence of scuba diving on asymptomatic isolated pulmonary bullae. *Diving and Hyperbaric Medicine*. 2008; 38: 206-211.)
Pulmonary blebs and bullae are generally considered an absolute contra-indication for scuba diving, because of a high estimated risk of pulmonary overpressure syndrome due to air-trapping inside the bulla. This is primarily based on a number of retrospective studies and case reports; formal prospective evidence of a higher risk is lacking. We present three cases where a pulmonary bulla was radiographically shown to increase in diameter, seemingly related to scuba diving activity, and causing ultimately a barotraumatic diving accident. These cases provide pathophysiological clues as to how even an isolated, non-ventilated bulla can be the cause of pulmonary barotrauma. The most likely mechanism for this phenomenon is a 'stretching' of the bulla upon ascent from the dive: after a period of compression (Boyle's Law), there is a gradual diffusion of air through the bulla wall, with restoration to its initial size by the end of the dive. Upon ascent, the air diffuses only slowly out of the bulla, causing a temporary increase in diameter and stretching of the bulla wall. This repeated stretching causes the bulla to grow gradually. At one point, the cyst wall may become critically thin and rupture during the ascent.

Introduction

Scuba diving has gained enormous popularity over the past two decades. With the development of reliable, comfortable diving breathing apparatus, diving has become accessible for people of all ages and physical fitness. The usefulness of a medical examination prior to engaging in recreational scuba diving is little disputed; however, the exact extent of such an examination is subject to much discussion. With regard to pulmonary function tests, there is evidence that abnormal flow-volume loops and compliance represent an increased risk for pulmonary barotrauma (PBT).[1] With regard to pulmonary blebs or bullae, the necessity to perform high-resolution imaging tests for detection has to be weighed against the estimated risk that such a bulla presents.

Asymptomatic pulmonary blebs can be found in a large number of persons, but no large series have been published to establish prevalence in a normal population.[2] Likewise, the prevalence of large bullae in a normal population is unknown. This makes a risk estimation practically impossible, since the denominator in the risk equation is missing. As PBT in recreational diving is fortunately a rare event (estimated between 1:19,800 and 1:34,000 dives),[3] the suggestion has been made that it is not justified to use CT scan screening for blebs in recreational or even professional divers.[2]

Retrospectively in divers with PBT, functional or anatomical abnormalities of the lung (scarring, emphysematous bullae or blebs) can be demonstrated in a number of cases using high-resolution (spiral) CT scanning.[4] These lesions are often undetectable on plain chest radiographs. Therefore, even if no large series exist that compare divers with PBT with controls, the presence of such structural anomalies is generally considered a contra-indication to diving, because of the risk of air trapping on ascent.[1]

In (large or small) ventilated bullae, air trapping can happen either on the basis of a one-way valve mechanism ('real' air trapping) or by a volume increase upon ascent because of a narrow inlet-outlet opening ('virtual' air-trapping). The risk of isolated, non-ventilated bullae is in most cases considered similar, although no plausible explanation is given. Some authors express their doubts on the presumed risk of isolated bullae, since pressure-volume mechanistic theory (Boyle's Law) would predict that these will never expand to greater than their initial volume.[5]

We describe three patients in whom isolated, non-ventilated pulmonary bullae were observed to increase in size during a period of three years of intense scuba diving. In two cases, this led to an episode of pulmonary overpressure syndrome with cerebral arterial gas embolism (CAGE). In one diver, the condition was followed up and during the next seven years of not diving, the bulla remained unchanged in size.

Case 1

Case 1 was a male born in 1942 with a 20-pack-year history of smoking (he stopped smoking in 1973). He

started recreational scuba diving in 1981, and performed approximately 1,200 uneventful dives over 16 years (about 75 dives per year in mostly cold water, to depths of 40–50 metres' sea water (msw)). In 1991, during a routine chest X-ray, a 35 mm diameter, thin-walled, asymptomatic bulla was discovered in the lower lobe of the left lung (Figure 1). On spiral CT scanning, there was no apparent ventilation orifice of this lung cyst. Ventilation-perfusion scanning was not performed. Routine pulmonary function testing, including flow-volume curve, was normal, and alpha-1-antitrypsin serum level was normal. He continued diving.

In April 1997, after an uneventful dive, he suddenly experienced severe general fatigue, headache, paraesthesia and mild paresis of both lower limbs. These symptoms recovered with normobaric oxygen administration; no hyperbaric treatment was given. Emergency chest X-ray upon arrival in hospital revealed a large bulla – approximately 100 mm in diameter. A repeat chest X-ray and CT scan six months later showed a reduction in the diameter of the cyst: 50 mm, thin-walled (Figure 1). He discontinued diving.

In 2002, because he wished to resume diving, a new chest X-ray and CT-scan were done, revealing a bulla diameter of 51 mm (Figure 1). A ventilation-perfusion scan showed an isolated, non-ventilated bulla. After discussion with a diving medical officer (DMO), he decided against resuming his diving hobby.

Summary of case 1: Asymptomatic bulla in left lower lung; increased diameter after six years of diving; hyperinflation of bulla immediately after diving accident with symptoms of CAGE; stable diameter after seven years of not diving.

Case 2

Case 2 was a female born in 1950, non-smoker, who had annual chest X-rays as part of occupational medicine checkups (she worked as a nurse in a respiratory ward). These were always classified as "normal" by the reviewing radiologist. She had received prophylactic anti-tuberculosis treatment after a positive Mantoux test in 1982.

She took up diving in 1988 and performed more than 100 dives per year for the next three years. In December 1991, after an uneventful dive, she experienced the following symptoms upon surfacing: mild chest discomfort, moderate dyspnoea, general fatigue, rigidity of neck and jaw, dysarthria, and marked coordination disturbances of the upper limbs with uncontrolled jerking when attempting to perform fine movements. These symptoms were initially attributed to cold, and no specific treatment other than

Figure 1
Case 1; serial chest X-rays and comparable CT slices taken over 11 years on the dates shown (see text for details)

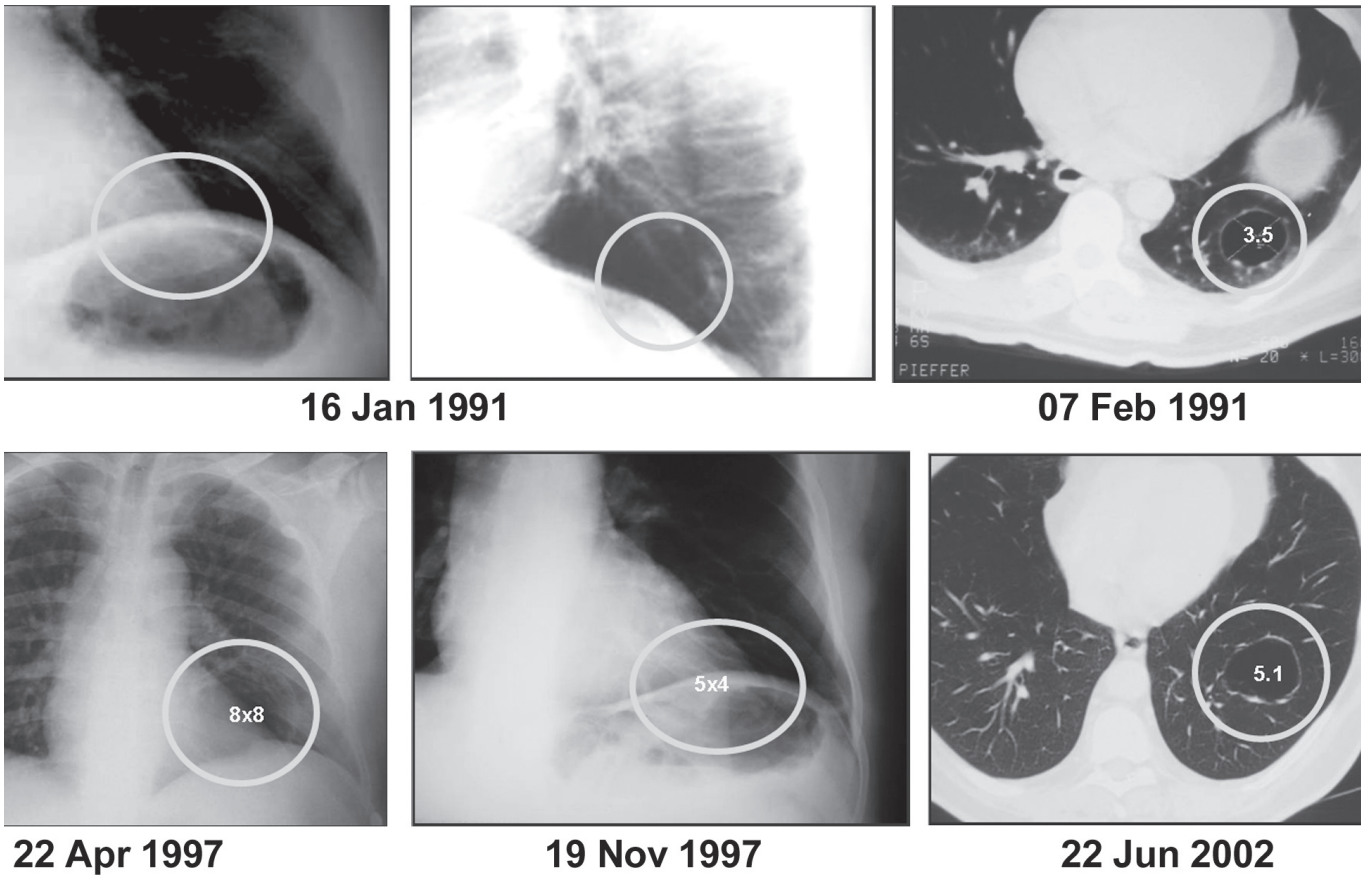
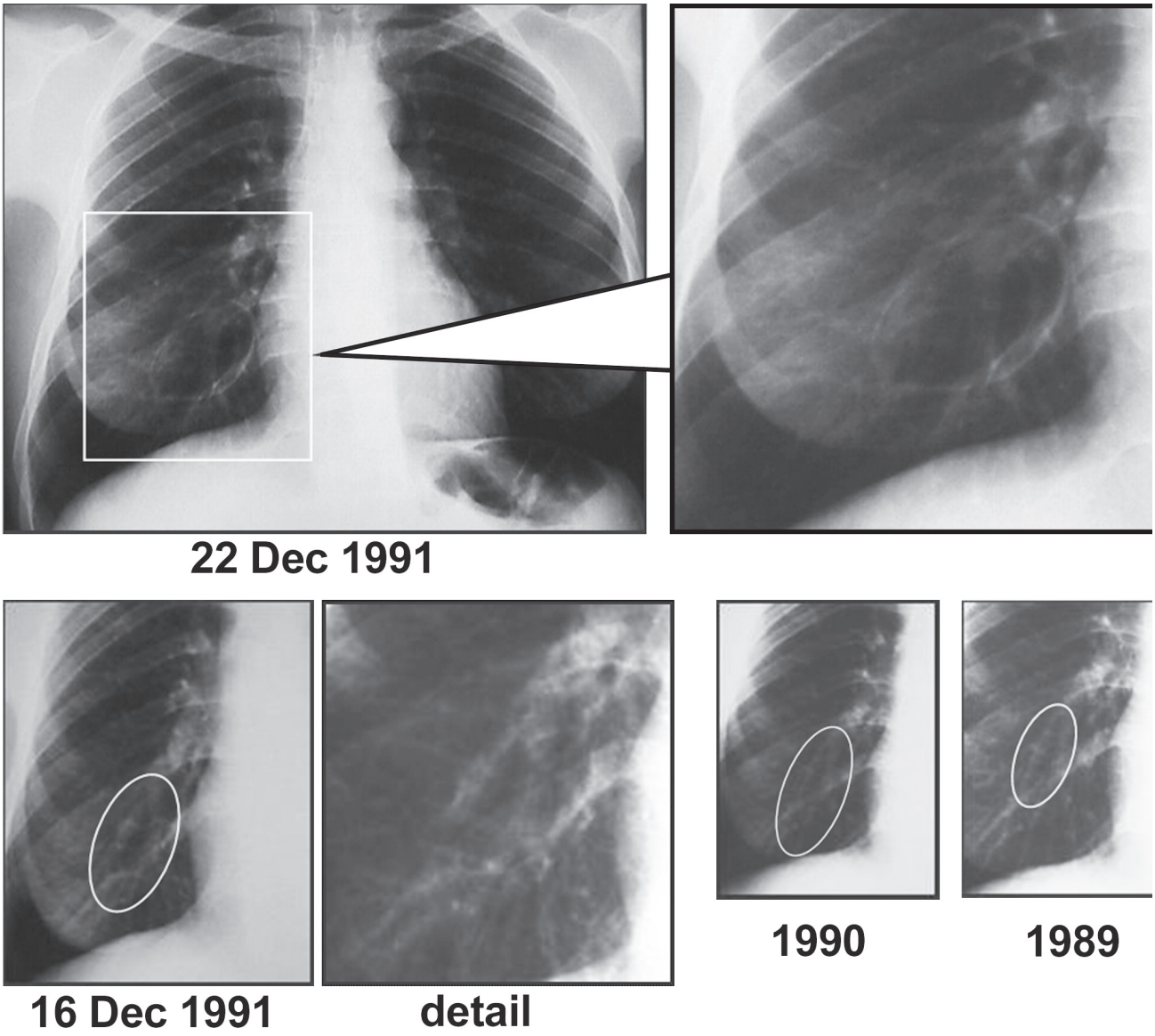


Figure 2
Case 2; serial chest X-rays taken over two years on the dates shown (see text for details)



rewarming was performed. She presented at the hospital 18 hours after surfacing, because these symptoms had taken longer than expected to resolve. She did not have major residual symptoms upon admission.

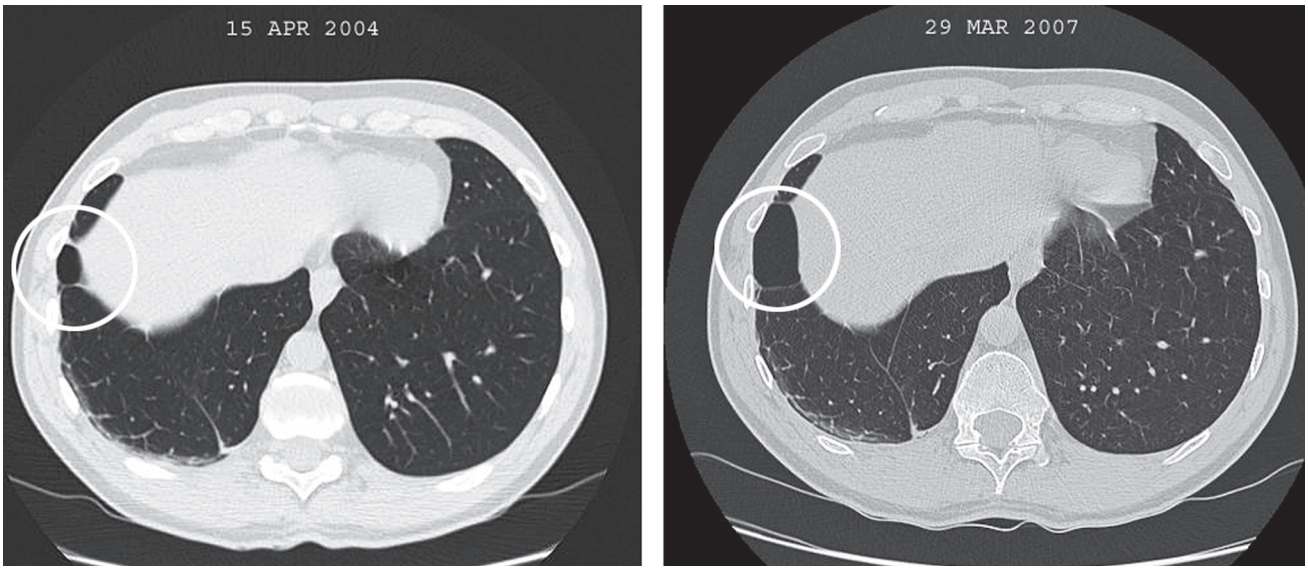
A chest X-ray revealed a large, spherical, thin-walled bulla in the right lower lung with a diameter of 70 mm (Figure 2). CT scan confirmed this to be a thin-walled cyst with no apparent ventilation orifice. A ventilation-perfusion scan showed no ventilation. She was not treated with hyperbaric oxygen, because of the absence of residual symptoms. A control chest X-ray one week after the incident showed a decreased diameter of the bulla, which was now partly collapsed and ellipsoid, measuring 6 by 3 cm. Serum alpha-1-antitrypsin level was normal. Routine pulmonary function testing, including flow-volume curve, was normal. A review of previous chest X-rays was undertaken (Figure 2):

- **16 December 1991** (one week prior to the accident): described as "normal". Upon careful review, the lung cyst can be seen as a faintly visible, flattened, ellipsoid measuring 60 by 30 mm.
- **1990**: classified "normal". Upon review the lung cyst is visible, but is notably smaller: 40 by 20 mm.
- **1989**: as above, 30 by 15 mm
- **1988**: as above, 20 by 10 mm

Following the DMO's advice, she stopped diving. A control chest X-ray three months later showed the bulla to be unchanged in size. She was then lost to follow up.

Summary of case 2: Asymptomatic bulla in right lower lobe; progressive increase documented with yearly chest

Figure 3
Case 3; comparable CT slices taken three years apart (see text for details)



X-rays over three years of diving; hyperinflation of bulla immediately after diving accident with symptoms of CAGE; all previous chest X-rays classified "normal".

Case 3

Case 3 was a non-smoking male born in 1959, who at the age of 20 had two episodes of spontaneous right-sided pneumothorax, the second episode of which was treated with unilateral chemical pleurodesis. He started diving in 1998, and made 160 uneventful dives. In April 2004, after a dive to 41 msw, he suffered from inner ear decompression sickness, and was treated with repeated hyperbaric oxygen sessions. As he was found to have a large patent foramen ovale (PFO), the decompression sickness was attributed to paradoxical embolisation of nitrogen bubbles through this PFO.

During the diagnostic work up, a high resolution CT scan of the lungs showed an ellipsoid pleural air space in the lower right lobe, with diameters 18 by 14 by 20 mm (Figure 3). Extensive pulmonary function tests, including flow-volume loops and methacholine provocation testing, were normal. He was advised by the DMO to stop diving; however, he chose to ignore this advice, as two independent respiratory physicians stated that, because the air space was isolated, there should be no problem for diving.

Over the next three years, he made a further 100 dives, during two of which he suffered from "unexplained vertigo after the dive". He was extensively reviewed in March 2007, with contrast echocardiography and high-resolution CT scan of the lungs (Figure 3). With regard to the PFO, this was found to be widely patent, as before. The lower right pleural bulla, however, was found to have substantially increased in size, measuring now 4.2 by 2.7 cm and extending well into the costo-diaphragmatic angle, to a total vertical length of 3.7 cm. Volume calculation (assuming the bulla has a roughly ellipsoid shape) shows an increase from 2.6 ml to 22 ml over these three years, or an 800% increase. He was again advised not to dive.

Summary of case 3: Asymptomatic isolated pleural bulla after pleurodesis 25 years previously; marked increase in size over the course of three years of regular diving.

Discussion

Pulmonary barotrauma is a major concern in compressed-air diving because of the high possibility of life-threatening complications such as tension pneumothorax and CAGE.[6] Although the occurrence of PBT is often associated with inappropriate ascent procedures (failing to exhale during ascent will induce a rapid increase in the pulmonary volume), many cases are reported where no such risk behaviour was observed or apparent.[4,7,8]

Alveolar rupture has been shown to occur with pulmonary overpressures of only 80 mmHg (equivalent to a water depth of only 65 cm). Proposed mechanisms by which such a slight increase of transpulmonary pressure might cause PBT include:

- *Decreased pulmonary elasticity* ('stiff' lungs). In a study of 14 young men who had suffered pulmonary barotrauma, it was shown that these individuals had less distensible lungs and airways than healthy divers and non-smoking non-divers.[9] The authors suggested that the relative stiffness of the airways increased the elastic stresses in the peribronchial alveolar tissue leading to an increased risk of alveolar rupture. It has been suggested that the increase in thoracic blood volume

during immersion may compound this problem by further reducing lung distensibility.[10]

- *Local or regional air trapping*. Classically, asthma (defined as a history of wheezing and abnormal pulmonary function tests) would disqualify a person from scuba diving, on the presumption that allergic or exercise-induced bronchospasm of the small airways may likely cause local zones of inefficient air exhalation, resulting in local overexpansion of pulmonary tissue.[11] This is supported by a number of case reports, where asthma was considered to be the only risk factor present.[4,12] In recent years, and considering that many asthmatic subjects apparently do dive without a high rate of pulmonary barotrauma, these guidelines have been somewhat relaxed, excluding now only those individuals with active, exercise- or emotion-induced asthma.[1] The remaining individuals are allowed to dive but warned of the potential risks. The value of pulmonary function tests as the sole criterion for detection of divers at risk for pulmonary barotrauma has been challenged because of low specificity.[13]
- *The presence of pulmonary blebs or bullae*. Numerous case reports are available in which divers with PBT were subsequently shown to have one or more (smaller) blebs or (larger) bullae, sometimes not visible on standard chest X-rays.[14] The same observation has been made in patients with spontaneous (non-traumatic) pneumothorax or recurrent spontaneous pneumothorax.[15,16] It is generally hypothesized that bullae predispose to pressure-reduction barotrauma either by a one-way valve mechanism at their 'entrance' (allowing entry of air upon descent but blocking outflow of air during ascent from a dive), or by an insufficient outflow capacity through a small orifice during rapid ascents.[14]

A direct causal relationship between the presence of bullous structures and lung tissue overpressure has not been established. Indeed, as imaging technology improves, asymptomatic pulmonary blebs can be found in a large number of persons. Although the exact prevalence is not known, it is reported that radiologists in a major hospital do not even mention the presence of small blebs as "*they are so common as to be considered normal findings in the patient population seen by a major hospital radiology department*".[2] Similarly, the prevalence of large bullae in a normal population is essentially unknown. Moreover, the causal relationship between the presence of bullae or blebs on chest CT and the occurrence of a first pneumothorax on the contralateral lung or recurrent spontaneous pneumothorax on the ipsilateral lung is still heavily disputed.[16,17]

When a diver is found to have pulmonary function test abnormalities after a pulmonary barotraumatic incident, few people would question the statement that these were already present before the dive. This, of course, lends credibility to a possible causal relationship. Only recently it has been suggested that diving by itself may induce changes in pulmonary function.[18] Experienced sports divers were shown to have a significantly reduced maximal expiratory flow at 25% (MEF25) and at 50% (MEF50) of vital capacity ($P < 0.01$ and 0.05 respectively) compared to non-divers.[19] There was a higher prevalence of cold-air hyper-reactivity in divers and there appeared to be a relationship with diving experience. The same observations have been made in professional saturation divers.[20]

When, after a pulmonary barotrauma, a diver is found to have pulmonary bullae on high-resolution CT scanning, the question may arise whether these bullae were pre-existent or whether they are the consequence of the barotrauma. As pre-barotrauma high-resolution pulmonary CT scans are inevitably lacking, it is impossible to ascertain the pre-existent nature of any bulla observed, making it impossible to state with certainty a significant causal relationship. Factors suggesting these bullae to be pre-existent and not caused by the diving accident could be: thick bulla wall, absence of liquid level and the presence of multiple non-ventilated spaces in the pulmonary parenchyma in combination with only localised barotrauma. Also, pulmonary over-distension bullae that have been acutely caused by PBT tend to resolve spontaneously within a few months.[21] A control CT scan some four months after the accident should therefore be performed in all cases.

Although divers are usually excluded from further pressure exposure after a PBT and/or detection of lung cysts or bullae, there is controversy as to whether a diver with an isolated, asymptomatic bulla should be excluded from diving. The discussion focuses on the possible mechanisms for such a bulla to rupture in the course of a dive. A common position suggests that according to Boyle's Law, such an air-filled structure will be compressed proportionally as the environmental pressure rises; then, when the diver ascends, the bulla will return to its initial, but not a greater volume. How then, would this promote rupture?

We hypothesize that during the dive, even if there is no direct ventilation orifice, a gradual diffusion of nitrogen through the bulla wall can take place, driven by the pressure gradient. As the elasticity of the lung tissue counteracts the volume reduction by Boyle's Law, air will diffuse from the nearby lung tissue into the bulla cavity, causing it to gradually re-expand, while still remaining in pressure equilibrium with the surrounding lung tissue. Then, as the pressure is reduced, and the air is 'trapped' inside the bulla, the bulla will grow beyond its initial volume. The elasticity of the surrounding lung tissue will now exert a concentric pressure on the bulla wall, and the resulting slight overpressure will make air (or nitrogen) diffuse out of the cavity into the surrounding tissue; the bulla will gradually return to its initial volume again. When this cycle happens in a repetitive manner, the bulla wall will get progressively stretched and the 'resting volume' of the bulla will increase. It is probable that the wall

gets thinner as this happens. At one point, the acute stretching during an ascent from a dive will cause an air leak from the bulla wall and may cause symptoms of PBT.

Conclusions

To our knowledge this is the first documented case series of non-ventilated pulmonary bullae where an increase in size can be attributed to scuba diving activity. Although a causal relationship cannot be demonstrated by the existing literature, these case reports suggest strongly that at least some pre-existing bullae can and will increase in size over the course of a few years' intensive diving. Because of the low sensitivity of plain chest X-rays, it may be advisable to obtain high-resolution CT scans of the chest in candidate divers where personal medical history leads one to suspect possible pulmonary parenchymal damage. If a diver with an isolated bulla were to be allowed to dive, it would then be advisable to perform serial follow-up CT scans after a number of years or exposures (dives), in order to observe a possible size increase in a timely manner.

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