Recurrent pulmonary barotrauma in scuba diving and the risks of future hyperbaric exposures: a case report

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Carpenter CR. Recurrent pulmonary barotrauma in scuba diving and the risk of future hyperbaric exposures: a case report. Undersea Hyper Med 1997; 24(3):209–213.—A 27-yr-old male scuba diving student suffered two episodes of pulmonary barotrauma 6 mo. apart after 12-ft training scuba dives. In the interval between these episodes, four uneventful hyperbaric chamber dives occurred. No definite cause or risk factors were identified. A MEDLINE review of diving literature revealed few studies of recurrent pulmonary barotrauma and no studies addressed risks of recurrent pulmonary barotrauma in the hyperbaric oxygen therapy environment following scuba pulmonary barotrauma.

recurrent pulmonary barotrauma, hyperbaric oxygen therapy, mediastinal emphysema, pneumopericardium

Pulmonary barotrauma (PBT) is a well-recognized complication of compressed air diving. PBT results from the expansion of lung volume as pressure decreases on ascent. Although PBT is a rare event (1), the potential complications can be life threatening. Some studies have implicated anatomic (2), physiologic (3, 4), or procedural errors (5) in explaining the pathophysiology of PBT. Few studies have explored the risks of recurrent PBT in scuba. No studies were found on a MEDLINE search which explored the relationship between PBT resulting from scuba dives with that from the hyperbaric chamber environment. A case of recurrent scuba-related PBT and considerations for future hyperbaric exposure are presented.

CASE REPORT

A 27-yr-old male dive candidate in good health noted the onset of neck pain 90 min after surfacing from the 12-ft scuba training pool just before beginning a second training dive. The first dive was significant for two ascents to surface at rates greater than the 2 s/ft recommended by the U.S. Navy Dive Manual (6) with one ascent in 9 s and the other in 12 s. The diver entered the pool for a repeat dive with the sore neck, and immediately upon surfacing 2 h later noted severe bilateral anterior neck pain, a high pitched voice change, and odynophagia. Subsequent physical exam by a diving medical officer revealed bilateral cervical and supraclavicular subcutaneous crepitance, equal breath sounds bilaterally, positive Hamman's sign (a precordial crunching noise on auscultation), and a normal neurologic exam. An inspiratory chest x-ray revealed pneumomediastinum, pneumopericardium, and subcutaneous air over the left upper hemithorax, but no pneumothorax. The physical findings resolved spontaneously over the next 6 days.

For the next 6 mo. the diving candidate continued intensive physical training, including breath-hold diving, but no scuba dives were performed. Four hyperbaric chamber dives (Table 1) were performed without evidence of recurrent pulmonary barotrauma. When the diver returned to the scuba training environment, two error-free dives, without rapid or uncontrolled ascent, breath holding during ascent, or improper regulator purging, were performed 20 h apart, after which the diver noted recurrent cervical subcutaneous crepitance, a finding confirmed by the diving medical officer. No other signs or symptoms were noted; neurologic exam was normal and the inspiratory chest x-ray was normal without pneumothorax, pneumomediastinum, or pneumopericardium. Physical findings resolved over the next 3 days. Three weeks after the last dive a high resolution chest computerized tomography (CT) showed no scarring, granulomas, or bullae and a Xenon-133 ventilation scan demonstrated no air trapping or asymmetric ventilations. A methylcholine challenge showed no evidence of reactive airway disease (RAD).

Twelve months before the first accident the diver had a normal screening inspiratory chest x-ray and spirometry. In the 3 mo. preceding the first accident, the diver had five hyperbaric chamber dives (Table 1). He had one 12-ft scuba dive 72 h before and another 96 h before the first accident; he had no other previous diving experience. The
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*Scuba dives all occurred in 12-ft training pool.  Scuba dives followed by symptoms of pulmonary barotrauma.  Bottom time, representing total elapsed time from when divers leave surface to the time rounded to next whole minute when divers leave the bottom and make a direct ascent to the surface.
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diving candidate had been diagnosed with RAD as an infant, wheezing during an upper respiratory infection, but had never received treatment or noted symptoms of asthma. The diver's brother received therapy for asthma as a teenager and adult.

DISCUSSION

The potential etiologies of PBT can be placed into one of four categories: anatomical, physiological, procedural, and idiopathic. Each of these conditions inhibits expedient egress of air as ascent causes volume expansion in accordance with Boyle’s Law. A bulla or granuloma represents an anatomic defect, whereas RAD is a physiologic problem. Chest radiography (roentgenography or CT) may demonstrate anatomic predispositions. Methylcholine challenge and radionuclide ventilation scans can be used to identify functional defects. Procedural errors in scuba include breath holding on ascent or rapid, uncontrolled ascents, and improper regulator purging or coughing while inhaling compressed air. One study even implicated overzealous exhaling during ascent (7).

An extensive medical evaluation in this case showed no functional or anatomic etiology to explain the PBT, as in previous reports of recurrent PBT (8), thus defining this case as idiopathic PBT. Although the first incident had been attributed to two rapid ascents, the delay in symptom onset and the subsequent recurrence without procedural error do not support this explanation. Additionally, in the United Kingdom, Royal Navy divers routinely reach buoyant ascent terminal velocities of 8 ft/s from training depths as deep as 100 ft (9) with the incidence of PBT estimated at 1/19,400 dives (8). One additional possibility is that the original pulmonary lesion had not healed sufficiently to allow resumption of scuba diving or that the breath-hold diving and strenuous exertion during the 6 mo. interval had prevented proper healing. Numerous cases of breath-hold PBT and a few exercise-related PBT cases have been reported (1,10–15). The lack of symptoms, such as continued subcutaneous emphysema resulting from gas leakage through an unresolved pulmonary wound, during the interval argue against a residual injury, and no scarring or fibrosis are noted on subsequent chest films. Leitch and Green (8) noted a wide range of time intervals between episodes of PBT from 2 wk to 10 yr. The United States Navy Dive Manual (6) recommends not diving for 4 wk after arterial gas embolism with complete resolution when treated on a short oxygen table. PBT generally receives similar recommendations with resumption of diving allowed after 4 wk if symptoms have resolved. Few recurrences are noted among military divers (8). Six months, therefore, would seem to be an adequate interval for lung healing. An incompletely healed injury, however, cannot be ruled out as a possible explanation for the recurrent PBT.

Some studies have suggested clinically occult reasons why some scuba divers experience PBT despite the lack of any procedural, physiologic or anatomic irregularity. Colebatch and colleagues (3,4) noted increased pulmonary elastic recoil among divers who have suffered PBT when compared to other diving and non-diving populations. They argue that the increased elastic recoil is a predisposition to PBT not a result of the PBT. This argument is supported by the finding of decreased elastic recoil in asthmatic subjects who have an increased incidence of interstitial emphysema secondary to PBT. If the PBT of asthma caused an increased elastic recoil, these patients should have an increased, not the observed decreased, elastic recoil (16). Furthermore, these studies demonstrate normal chest x-rays and spirometry among the PBT divers. Theoretically, however, these divers may suffer PBT as a result of minor overinflation that would not manifest in an individual with more compliant lungs. The increased elastic recoil is believed to increase the stress on individual tissue fibers, an effect magnified in peribronchial alveolar tissue near total lung capacity until tissue rupture occurs (4). The majority of divers with increased elastic recoil will never suffer PBT (3), so evaluating pulmonary mechanics for all divers would probably be of little use in predicting subsequent risk of PBT.

A MEDLINE review of the last 30 yr revealed no studies comparing the PBT risk, recurrence rate, or etiologies after scuba diving with the hyperbaric chamber environment. Indeed, only two reports of chamber-related PBT were noted (9,17) and no studies were identified to evaluate risk factors specific to either diving environment. Underwater diving has several attributes not found in chambers. The pressure exerted on the diver is hydrostatic and, as Bondi and colleagues (18) and Prefaut et al. (19) have shown, may predispose to airway collapse because peribronchial vessel engorgement results from the increase in central blood volume by hydrostatic pressure and decreases elastic recoil at low lung volumes. Theoretically, a predilection for airway closure at depth, even at low lung volumes, could represent an unrecognized risk factor for PBT in water sports. In contrast, a MEDLINE search revealed no studies that evaluate the closing volumes of divers in hyperbaric chamber environments. The risk imposed by the indirect effects of hydrostatic pressure would not be present in the hyperbaric chamber.

Additionally, in scuba the regulator requires slightly greater respiratory effort to overcome the seal between the second stage and the intermediate hose. The increased
resistance to breathing could theoretically increase the transpulmonary pressure gradient. When this increased pressure gradient occurs in a diver during ascent, alveolar septal fibers, especially in peribronchial regions, may be strained to the point of rupture. The fibers in a diver with decreased pulmonary elasticity may be strained to a greater extent, increasing the risk of rupture.

The hyperbaric chamber environment, on the other hand, consists of atmospheric pressure without a substantial hydrostatic component. Whether the different source of pressure affects the closing volumes differently has not been addressed, but certainly the increased resistance to breathing is far less than in scuba, which could affect the incidence of PBT in the chamber. The larger number of reported scuba-related PBT may partially be a result of the larger number of individuals exposed to scuba compared to the hyperbaric chamber. Many scuba divers who might later be exposed to the hyperbaric chamber are excluded when their apparent predisposition to PBT is noted, often in the scuba training environment early in their diving career (1). The scuba training environment favors PBT with inexperienced divers breathing compressed air at shallow depths, whereas hyperbaric chambers are controlled by trained personnel not breathing compressed air, with rapid, uncontrolled ascents being very rare. Additionally, instructor awareness for PBT signs and symptoms are heightened in scuba training environments (20). The true incidence of PBT is impossible to measure even in strict training environments because many cases of PBT may go unrecognized or unreported (20) and diving is resumed without any sequelae as suggested by Calder’s findings on deceased divers (2).

The hyperbaric literature considers a history of spontaneous pneumothorax a relative contraindication to entering the hyperbaric environment, particularly a monoplace chamber (21), but does not address other forms of PBT (pneumomediastinum, subcutaneous emphysema) as contraindications. Current U.S. Navy medical disqualifications for diving duty include “congenital and acquired defects which may restrict pulmonary function, cause air-trapping, or affect the ventilation-perfusion balance” (22). A proposed addition to these medical disqualifications from initial dive duty is “more than two episodes” of pulmonary barotrauma (US Navy, Bureau of Medicine and Surgery, unpublished). One case of a diving medical technician, who suffered pneumomediastinum after a sur-face swim, reported a return to hyperbaric chamber duties 1 yr later, where for 18 yr he regularly performed his duties in a hyperbaric environment without a recurrence (12).

No evidence linking scuba and hyperbaric chamber PBT exists. This observation has 2-fold importance. First, hyperbaric chamber pressure testing before entering scuba training may not predict adverse pulmonary events. In the case of traumatic scuba PBT, as in this case, hyperbaric chamber trials before reentering the scuba environment may not predict adverse scuba events. Second, although recurrent PBT has been demonstrated in scuba, no such recurrence has been noted in the hyperbaric chamber environment. The relative safety of hyperbaric chamber exposure after single or multiple episodes of scuba PBT is not known. Both issues could be addressed through a retrospective review of hyperbaric chamber exposure-related PBT searching for scuba risk factors or unique risk factors. Additionally, physiologic parameters like closing volumes and elastic recoil noted to change in underwater environments should be examined in the hyperbaric chamber environment.

In conclusion, in divers without an obvious physiologic or anatomic defect on screening physical exam, including inspiratory and expiratory chest x-rays, no effective studies exist to predict who will suffer PBT or recurrent PBT. A number of physiologic pulmonary effects may be unique to the underwater environment and should receive further attention to determine the safety of future hyperbaric chamber exposure as well as the hyperbaric chamber test’s predictive value either pre- or post-scuba PBT for subsequent scuba PBT. The diving community must continue to focus attention on the occurrence, safe interval before return to diving, and rare recurrence of PBT both for optimal diver safety and to elicit risk factors for PBT that have gone unrecognized thus far.

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REFERENCES
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