Pulmonary Barotrauma and Related Events in Divers

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Although pulmonary barotrauma (PBT) is a well-known clinical entity, its recognition in divers is sometimes delayed and its implications for future diving often are unappreciated. The pulmonary complications of diving activities range from mere discomfort from mediastinal emphysema or pneumothorax, or both, to life-threatening gas embolization. In nine cases described here, only minor manifestations were associated with PBT which occurred at or close to the surface, but three of these four divers were found to have abnormal pulmonary function. More serious manifestations resulted from PBT which took place at depths of 16 to 120 ft. Even minor forms of PBT should be considered a contraindication to further diving, since they are prone to recur. Such recurrences—even at shallow depths—may cause serious complications. *(CHEST 1995; 107:1648-52)*

ATA=atmosphere absolute pressure; MMEFR=maximum mid-expiratory flow rate; PBT=pulmonary barotrauma; Scuba=self-contained underwater breathing apparatus

Key words: barotrauma; diving; gas embolism; subcutaneous emphysema; obstructive lung disease; pneumomediastinum; pneumothorax

Pulmonary barotrauma (PBT) is a frequent complication of mechanical ventilation. As pointed out by Marcy[1] in a recent review, the risk of PBT is increased both by the degree of pressure elevation applied by the ventilator to central airways and by the extent of lung disease.[2,3] Pulmonary barotrauma also has been reported in divers.[4,5] In this situation, a different means of external ventilation is used. That is, the diver breathes gas from a high-pressure tank by means of a two-stage regulator which ensures that the supplied gas is at a pressure only slightly higher than ambient pressure, regardless of the dive depth. Unlike the lungs of most ventilator patients, the diver's lungs usually are healthy or are presumed to be so.[6] In divers, PBT is a much-feared event nevertheless. If it occurs at any significant depth, a number of life-threatening consequences may ensue.[7,8] These complications develop as the extraalveolar zones of escaped breathing gas expand according to Boyle's law, in direct proportion to the rate and extent of the diver's decompression profile.

Consider, for example, the case of a diver who develops PBT at a depth of 165 ft, causing a 500-mL pneumothorax (Fig 1). The absolute pressure at this depth is 6.0 atmosphere absolute pressure (ATA); that is, the conversion factor is 1 ATA pressure per 33 ft of sea water; thus 165 ft divided by 33=5.0 ATA, plus 1.0 ATA of sea level barometric pressure. If this diver then ascends to the surface, the volume of the pneumothorax would increase from 0.5 to 3 L due to the sixfold fall in ambient pressure. Such an expansion would create a tension pneumothorax. In addition, the expanding gas might force its way into pulmonary venous structures, making serious gas embolization likely, as occurred in some of the cases presented in this report.

This report describes nine diver patients with PBT (Table 1). Some (cases 1 to 4) developed this condition at or near the water surface. Since they sustained relatively minor consequences of PBT,[9] they are termed minor cases. They occurred either spontaneously in the presence of increased lung volume and pressure (Valsalva maneuver with hyperinflation), with vigorous in-water activity, or because of equipment malfunction. In these instances, the immediate sequelae were uncomfortable but not serious. However, three of the four such individuals were later found to have abnormal pulmonary function after experiencing PBT. This finding raises the issue of whether such abnormalities could have caused the episode or might have resulted from it.

Five other cases (divers 5 to 9) developed PBT while submerged to depths of 16 to 120 ft (1.5 to 4.6 ATA), thus making the Boyle's law consequences of the resulting pneumothorax more serious. These are thus referred to as major cases. Since each case illustrates an important feature, each will be described individually.

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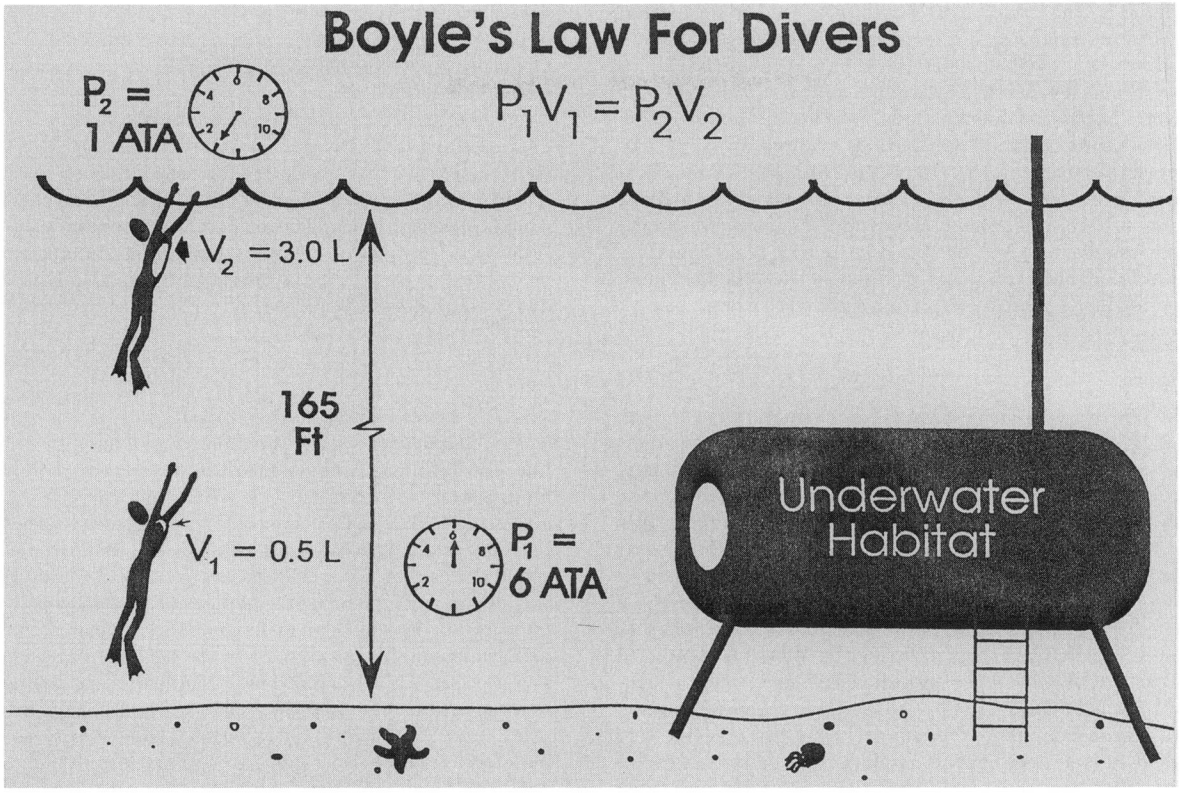

FIGURE 1. Schematic representation of a diver who develops a 0.5-L apical pneumothorax *(small arrow)* while submerged at 165 ft of sea water (pressure P_1=6.0 ATA) and then ascends to the surface (pressure P_2=1.0 ATA). In accordance with Boyle's law, this sixfold reduction in ambient pressure would result in a tension pneumothorax of about 3.0 L *(large arrow)* and possibly other complications of gas embolization.

METHODS

The subjects described in this report were cared for directly or referred for consultation or evaluation by other physicians. All were nonsmokers.

All pulmonary function measurements were made in a pulmonary laboratory at sea level, with subjects in the seated position. Spirometry testing was done with a Stead-Wells device (Warren E. Collins, Braintree, Mass), using the best of three successive efforts. The subject's results were compared with the normal values reported by Knudson and coworkers.[10] Other pulmonary function measurements were made using standard methods and normal values.[11-14]

REPORT OF CASES

Minor Cases, Occurring at Water Surface

CASE 1

A 19-year-old white male diving medical technician was being trained in "drown-proofing," a technique to prolong in water survival[15] (breath-holding for 15-s periods at total lung capacity is alternated with rapid vital capacity breaths). After one such maneuver, the training supervisor noted that the voice of diver 1 had changed, taking on a "tinny" nasal quality, soon followed by neck pain and difficulty swallowing. Examination showed a

Table 1—*Pulmonary Barotrauma in Divers, Nondiving vs Hyperbaric*

Case	Age, Gender	Activity Preceding PBT	Prior Lung Disease?	Complications of PBT
Minor (Surface) Cases				
1	19, M	Drown-proofing, on surface	None	None
2	23, M	Buddy-breathing, 0 to 5 ft	None	Small airway dysfunction
3	27, M	1,000-yard swim, surface	None	Small airway dysfunction, decreased CO diffusing capacity
4	35, M	Scuba malfunction, surface	None	Pneumothorax, mild COPD
Major (Hyperbaric) Cases				
5	18, M	Hull repair, 18 ft	None	Tension pneumothorax, tuberculous pleurisy
6	23, F	Sport diving, 40 ft	Bullae	Pyopneumothorax
7	23, M	Salvage dive, 120 ft	None	Cerebral emboli, death
8	35, M	Sport diving, 40 ft	None	Cerebral ischemia
9	43, F	Sport diving, 16 ft	Bullae	Pyopneumothorax

bulging neck with crepitus extending over the supraclavicular areas. Scattered crackles were heard over both lungs. Radiographs showed cervical subcutaneous emphysema and pneumomediastinum outlining the heart. All findings cleared without specific treatment. Pulmonary function tests 3 weeks later were normal.

Comment: As is true of spontaneous pneumothorax, persons who develop subcutaneous emphysema from pneumomediastinum have an increased risk of a recurrence as noted by Parker et al.[9] This probably is because the conditions share a common pathogenesis, *ie,* marginal alveolar rupture with dissection of released gas along the bronchovascular bundle back into the hilum. He was advised against future diving since a recurrence of PBT under water could have devastating consequences.

Case 2

A 23-year-old white male diving technician practiced "buddy breathing" from self-contained underwater breathing apparatus (scuba) equipment with a double-hose regulator. This technique sometimes is used when one diver has run out of breathing gas. The divers then take turns breathing from one mouthpiece and its gas supply, while coming carefully to the surface. In the case of diver 2, the buddy breathing was being done in a shallow training pool. Because the air tanks became buoyant during the 2-h practice, he thought he might have come to the surface without completely exhaling, at least once. When he completed the practice session, he noted that his voice had taken on a high-pitched "Donald Duck" quality. In the clinic the next morning, he had a swollen, tender, crepitant neck, and fine crackles were audible over both lung fields. X-ray films showed pneumomediastinum, subcutaneous emphysema in the neck, and a thin-walled cyst in the azygous lobe of the right lung. Pulmonary function tests 8 weeks after the episode of PBT showed a functional residual capacity of 141 percent of predicted. The maximal midexpiratory flow rate (MMEFR) and carbon monoxide transfer factor were 73 and 78 percent of predicted. The alveolar-arterial oxygen pressure difference ($P[A\text{-}a]O_2$), 17 mm Hg, and closing capacity, 134%, also were mildly abnormal.

Comment: Depending on the relative positions of the diver's mouthpiece and the regulator during buddy-breathing, the gas supply from older double-hose regulators are able to "free-flow," supplying air at a pressure above ambient.[16,17] The diver's airway can thus be overpressurized by as much as 50 cm H_2O, perhaps contributing to the PBT in diver 2. Alternatively, he may have caused his PBT by breath-holding during ascent in the pool. This seems more likely since he had not noticed any "free-flow" of his breathing gear. The pulmonary function results suggest small airway dysfunction. Without prior measurements, it is unclear whether the abnormalities were the result of PBT or were preexisting and perhaps causally related to the episode. Had they, or the paratracheal lung cyst, been identified prior to his entry into diving, it is uncertain whether they would have been considered contraindications to future diving. Given the risk of recurrence of PBT, diver 2 was so advised.

Case 3

A 27-year-old white male diving technician was walking home after a 1,000-yard training swim when he noticed a sharp substernal pain and neck stiffness. He tried to eat lunch, but could not swallow because of pain. When examined the next day, his neck and supraclavicular tissues were sore and crepitant. Radiographs showed subcutaneous emphysema and pneumomediastinum. Pulmonary function tests 1 month after his episode showed mild hyperinflation and a reduced MMEFR. The closing capacity was increased, consistent with small airways dysfunction. The carbon monoxide transfer factor was 75% of predicted. Exercise testing showed a widened $P(A\text{-}a)O_2$ due to ventilation-perfusion mismatch, without shunting. His pulmonary function and blood gas values improved over the next year, and he resumed a vigorous physical fitness program, running 4 to 6 miles a day. He was medically cleared for hyperbaric chamber duties 2 years after his pneumomediastinum.

Comment: The case of diver 3 exemplifies pneumomediastinum resulting from vigorous in-water exertion, without the unusual maneuvers involved in the first two instances. Because his job was that of a research technician at a hyperbaric chamber in a medical center, it was believed that he could be cleared to perform such work with the understanding that he might require recompression therapy in the event of a recurrence. His chamber duties were sedentary and he experienced no such recurrence over the ensuing 18 years.

Case 4

A 35-year-old white male mechanical engineer was evaluating a closed-circuit underwater breathing apparatus with a tight-fitting oronasal mask. The device malfunctioned and briefly exposed his upper airway to a mixture of 80% helium and 20% oxygen at a pressure of 1.24 ATA (about 207 mm Hg above atmospheric pressure). He momentarily lost consciousness but had no seizure activity or cardiac abnormalities. At the hospital 10 min later, he was alert and complained only of pleuritic substernal pain. Chest radiographs showed bilateral pneumothorax, pneumomediastinum, subcutaneous emphysema in the neck, and gastric distension. Because his condition rapidly stabilized, and the inert gas was helium, no chest tubes were placed. Nasal oxygen was given at 3 L/min. Repeat chest x-ray films 3 h later showed near-complete resolution. He soon resumed his normal duties but developed a chronic cough with clear sputum and was referred for evaluation with regard to continued diving. Wheezing was heard over the left lung base. Pulmonary function tests (which were supranormal pre-PBT) showed a MMEFR of 72% of predicted, improving to 88% with a bronchodilator aerosol. Although his abnormalities were mild, and apparently caused by the unusual equipment malfunction, it was thought that continued diving posed an undue risk of further PBT. His cough continued and he eventually developed recurrent wheezing. Pulmonary function test results remained abnormal.

Comment: This diver developed mild symptomatic airway obstruction after PBT due to equipment malfunction. Whether the latter event played a causal role cannot be concluded with certainty. The very rapid reexpansion of bilateral helium pneumothorax with oxygen as the only form of treatment[18,19] is an unusual sidelight of the case.

Major Cases, Occurring at Depths of 16 to 120 ft

Case 5

An 18-year-old white male diving technician was making hull repairs at a depth of 18 ft. He came to the surface unconscious, and was taken immediately to the nearby hospital. A tension pneumothorax was treated by chest tube with complete reexpansion of the collapsed lung. Upper-lobe shadows suggested active tuberculosis, and the chest tube drainage was positive for *Mycobacterium tuberculosis*. He made an uneventful recovery.

Comment: Since prior radiographs could not be obtained, it is possible that a subapical focus of infection may have been overlooked in this case, thus setting the stage for the development of PBT which might otherwise not have occurred. Because the diver-patient did not recall circumstances immediately preceding his loss of consciousness, the actual sequence of events remains unknown. He was advised against further diving.

Case 6

A 23-year-old white female developed chest pain soon after she

surfaced from scuba diving. She had no history of lung disease. Chest x-ray films in the Emergency Room showed a right tension pneumothorax. A chest tube was inserted, but lung reexpansion was incomplete. Bronchoscopy showed no foreign body or other endobronchial abnormality. A second chest tube was placed, but expansion remained incomplete; therefore, right thoracotomy was performed, and bullous disease was disclosed. A small pleural effusion grew Klebsiella and enterococcal species; ampicillin and gentamicin were administered. The patient was discharged with a small residual pneumothorax and instructions to avoid future diving.

Comment: This young woman's preexisting bullae—clearly a contraindication to diving—might have been identified by screening radiographs and spirometry tests, even though they escaped detection by physical examination. When tested a year after her episode of PBT, she showed evidence of moderate airway obstruction.

CASE 7

A 23-year-old white male salvage diver was working in scuba gear at 120 ft from a Boston Whaler. He ran out of air and "buddy-breathed" from his diving partner's mouthpiece until they reached a depth of 20 ft. He abandoned the buddy technique and immediately surfaced, apparently holding his breath for the rest of the ascent. He rapidly lost consciousness. Recompression facilities were unavailable. He developed hypotension and agonal respirations while being transferred by aircraft in a "monoplace hyperbaric chamber" and was dead on arrival at a hyperbaric treatment facility. Autopsy showed massive air embolism with severe cerebral and pulmonary edema.[8]

Comment: This case illustrates that breath-holding during any ascent must be avoided by SCUBA divers, since it can lead to cerebral air embolism which most likely developed from PBT during the panic-driven ascent of this diver. Had facilities been readily available for prompt recompression treatment, his death may have been avoided.

CASE 8

A 35-year-old white male executive was sport diving on vacation. His dive sequence was as follows: in the morning, 115 ft for 25 min, with 3-min decompression stop at 10 ft; in the afternoon, 40 ft for 60 min, with no decompression stop.

The interval between the two dives was "a couple of hours." The applicable US Navy Diving Table would have called for longer decompression, but whether following the US Navy Diving Table would have avoided the events described later is uncertain. Two hours after the second dive, he noted the acute onset of sharp mid-chest pain and dyspnea. He did not seek medical help, but during dinner an hour later, his wife noted several episodes of transient unresponsiveness during which "he went blank and was glassy-eyed." There was no motor, sensory or speech deficit, seizure activity, or cardiac irregularity. Diagnostic facilities on the island were limited; no chest radiographs could be done. On the basis of his symptoms, he was treated with recompression for the presumptive diagnosis of cerebral air embolism and had no further difficulty. When evaluated by a pulmonary physician 2 weeks later, his physical examination and chest radiographs were normal. He was advised against further diving.

Comment: This case should probably be considered PBT only in the generic sense. That is, the chest pain may have reflected a form of decompression sickness known as "chokes." This is thought to be due to pulmonary air embolism, related to inadequate decompression. His neurologic symptoms were interpreted by the attending physician as evidence of cerebral air embolism, for which recompression treatment seems clearly to have been indicated, even though the delayed presentation was atypical. Whether a more conservative repetitive-diving decompression schedule would have avoided this episode is unknown.

CASE 9

A 43-year-old white woman asked medical advice about her fitness to dive. She had a chronic cough and radiographic evidence of extensive bullous lung disease. Alpha-1-antitrypsin deficiency, cystic fibrosis, and foreign bodies were excluded. Against medical advice, she went on a diving vacation. While surfacing from 16 ft, she noted left shoulder pain. A diagnosis of pleurisy was made by history and physical examination, and ampicillin was administered. Two weeks later, she felt well enough to fly home. When seen 4 weeks after the initial symptoms, she had a pyopneumothorax which required tube thoracostomy and additional antibiotic therapy.

Comment: This patient was fortunate in that a risk of further PBT was posed during her 8-h flight home, since she probably already had a 2-week-old pneumothorax. She ultimately required decortication because of persistent infection.

DISCUSSION

This series of cases has a simple message: a variety of in-water activities can cause divers to develop pneumothorax or mediastinal and subcutaneous emphysema, or all, even at the surface with or without equipment malfunction. Those cases which occurred at or near the surface caused relatively minor discomfort in divers 1 to 4, but they raise the possibility—given continued diving—that recurrences[9] might develop at substantial depths. Recurrences are likely, as is emphasized by the evidence of airway obstruction in divers 2 to 4. If so, these later episodes might well involve more major complications,[7,8] such as those which affected divers 5 to 9. It is important that physicians who see such patients pursue the diagnosis of PBT with a detailed history, appropriate physical examination, and radiographic and laboratory investigation. Because its clinical manifestations are not dramatic, minor PBT does not receive much attention in reviews of diving medicine.[5] Our experience and that of Parker et al[9] suggest that it is not, however, uncommon. Both military and civilian divers comprise the aforementioned cases, but since the population from which they are derived is undefined, there is no way to estimate the incidence of PBT. These cases were referred for evaluation over a 6-year period. However, they may represent the unusual case which any physician may see in a general or pulmonary practice or which a specialist in hyperbaric medicine may see in consultation.

Why was major PBT (cases 5 to 9) associated with significant depths? At least two factors seem likely to contribute to this event. First, higher transpulmonary pressures may be generated by the diver under hyperbaric conditions than at the surface. For example, we have measured much higher values of transpulmonary pressure during both maximum voluntary ventilation and flow-volume maneuvers, when the divers breathed a gas mixture which was eight times

the density of air at normal pressure.[20] Such high values of transpulmonary pressure make it more likely that a vulnerable area of lung parenchyma may undergo rupture, setting the stage for PBT to occur. This is an incomplete explanation for the hyperbaric cases in this short series, however, since they occurred at the modest depths of 16 to 120 ft. Once such a rupture occurs, however, the Boyle's law consequences of gas expansion (Fig 1) during a diver's ascent work to magnify the effects of extrapulmonary gas escape and increase the likelihood of major gas embolization. The greater the depth excursion, and the more rapid the rate of decompression, the higher the risk of serious complications. At the same time, it is important to recognize that the relative volumetric expansion for a given absolute pressure decrement is greatest in shallower depths. This latter explanation better fits with the observations in divers 5 to 9.

The three diver-patients who were found to have abnormal post-PBT pulmonary function tests raise a more puzzling issue. Did such abnormalities cause PBT, or were they the result of it? Only in case 4 do we have a hint that the latter is true, since he is the only individual whose pre-PBT pulmonary function had been measured. His spirometry, dilution lung volumes, and carbon monoxide transfer factor all exceeded their predicted values. Thus, it seems likely that his post-PBT abnormalities resulted from the event rather than being its cause. The same may be true of the pulmonary function abnormalities in divers 2 and 3, since the abnormalities in the latter case improved over 18 months after his episode.

In a larger series of Australian divers studied post-PBT, Colebatch et al[21] found evidence that the conductive properties of the divers' lungs were normal but that their lungs were stiffer than those of control divers who had never sustained PBT. They suggested that measurements of elastic recoil to screen out divers at risk of PBT would have limited predictive value, however, since the majority of those divers with stiff lungs, *eg*, about 58 of 500, had not suffered an episode of PBT. A second study confirmed their initial findings as well as disclosed that abnormal airway stiffness was present in the divers studied more recently,[22] in addition to decreased lung distensibility. Their studies were not designed to address directly the possible value of screening spirometry in identifying divers who are destined to sustain PBT. However, their finding that the pulmonary conductance in PBT divers was nonsignificantly lower than in control divers suggests that spirometry would lack the needed sensitivity and predictive value to make it useful. At present, we know of no readily applicable technique to screen healthy diver candidates except to exclude those who have a history of PBT.

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