Diving and the risk of barotrauma

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Introductory article

Risk factors for pulmonary barotrauma in divers

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Study objectives. Pulmonary barotrauma (PBT) of ascent is a feared complication in compressed air diving. Although certain respiratory conditions are thought to increase the risk of suffering PBT and thus should preclude diving, in most cases of PBT, risk factors are described as not being present. The purpose of our study was to evaluate factors that possibly cause PBT. Design. We analyzed 15 consecutive cases of PBT with respect to dive factors, clinical and radiologic features, and lung function. They were compared with 15 cases of decompression sickness without PBT, which appeared in the same period. Results. Clinical features of PBT were arterial gas embolism (n=13), mediastinal emphysema (n=1), and pneumothorax (n=1). CT of the chest (performed in 12 cases) revealed subpleural emphysematous blebs in 5 cases that were not detected in preinjury and postinjury chest radiographs. A comparison of predive lung function between groups showed significantly lower midexpiratory flow rates at 50% and 25% of vital capacity in PBT patients (p<0.05 and p<0.02, respectively). Conclusions. These results indicate that divers with preexisting small lung cysts and/or end-expiratory flow limitation may be at risk of PBT. (Chest 1997;112:654-59)

Self-contained underwater breathing apparatus (SCUBA) diving has become a worldwide recreational sport activity. The open circuit SCUBA enables the diver to breathe high pressure gas from a cylinder through a regulator, which reduces the inhaled gas to ambient pressure and allows exhalation into the water. The rapidly changing ambient pressure under water may cause a variety of physiological alterations. Given the popularity of SCUBA diving and the number of diving accidents, physicians should be aware of the specific hazards and the medical conditions that may be encountered. I shall review these first, and then consider the risks posed by pre-existing respiratory disorders, hence the importance of the introductory article.

Basic physics

Since most diving related complications are a consequence of gases under changing conditions of pressure, it is appropriate to remember the two most relevant gas laws. Boyle’s law states that at a constant temperature the volume of gas varies inversely with the pressure applied (P₁V₁=P₂V₂). The physiological consequences of this law explain the pressure related diving diseases (i.e. barotrauma). Henry’s law states that the amount of a given gas dissolved in a liquid is directly proportional to the partial pressure of that gas. This law provides the explanation of decompression sickness and nitrogen narcosis.

Diving related accidents

Drowning is reported to be the most common cause of death among divers, but arterial gas embolism as a complication of pulmonary barotrauma (PBT) ranks second, accounting for about 30% of recreational SCUBA diving fatalities. When symptoms occur because released bubbles are trapped in the non-pulmonary tissues of the diver as he ascends and is exposed to a reduction in environmental pressure (decompression), the condition is called decompression sickness (DCS).
Diving and the risk of barotrauma

Table 1 Distinction between decompression sickness and cerebral air embolism

<table>
<thead>
<tr>
<th>Decompression sickness (DCS) Type II and cerebral air embolism</th>
<th>Arterial gas embolism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diving profile Diver’s time at depth exceeds the maximum allowed time according to commonly used diving tables.</td>
<td>Diver’s time at depth exceeds the maximum allowed time at the surface where the rate of gas expansion is greatest.</td>
</tr>
<tr>
<td>Neurological symptoms Particularly spinal cord</td>
<td>Primarily cerebral</td>
</tr>
<tr>
<td>Neurological symptoms Progressive sensory and/or motor loss of the limbs</td>
<td>Unconsciousness, Paraparesis, Paraplegia</td>
</tr>
<tr>
<td>Symptoms spread from distal to proximal</td>
<td>Visual disturbances, Headache</td>
</tr>
<tr>
<td>Accompanying symptoms and signs</td>
<td>Other evidence of pulmonary barotrauma (e.g. chest pain, haemoptysis, pleural effusion, pneumothorax)</td>
</tr>
<tr>
<td>Occurrence</td>
<td>50% &lt;1 hour after the dive</td>
</tr>
<tr>
<td></td>
<td>90% &lt;6 hours after the dive</td>
</tr>
<tr>
<td>or more than 10 m ascents from greater depths.</td>
<td>Within &lt;10 minutes after the dive</td>
</tr>
</tbody>
</table>

Prevention of diving related complications

Dive profiles may influence the likelihood of DCS. The deeper the dive and the more decompressions required, the higher the incidence of decompression sickness. Repeated dives or flying within a certain time after diving may also be a source of difficulty since, after a dive, considerable quantities of nitrogen may remain in the tissues. Furthermore, a number of physiological and environmental factors are believed to increase the likelihood or severity of DCS. Most of these influence the blood supply to tissues and therefore the speed of gas uptake or release. Such factors consist of exercise, low water temperature, female gender, and obesity. The risk for DCS can be reduced if the rate of ascent is controlled by intermittent stops according to decompression tables, considering the above mentioned cofactors. Although rapid decompression will predispose to DCS, most divers who develop DCS have apparently complied with the decompression tables.

Differential diagnosis

It is not unusual that the differential diagnosis between PBT with arterial gas embolism and DCS type II causes significant problems. Contrasting the characteristic features between these two disorders tends to simplify the situation (table 1). Fortunately the same treatment—that is, recompression with intermittent breathing of 100% oxygen—may be effective for both types of complication.
Most PBT events with arterial gas embolism are caused by breath holding and/or a rapid or uncontrolled ascent in the water. The first observations illuminating a causal sequence between PBT and arterial gas embolism were made in fatal outcomes of military personnel during submarine escape training in the 1930s. Escape training is performed in a water-filled tower with a height of around 30 m. Access to the tower is by means of air locks at various levels. The subjects are instructed not to hold their breath during the ascent but to exhale at a rate adequate to prevent overpressure in the lungs, without too great a loss of buoyancy. It is conceivable that this delicate manoeuvre may fail, resulting in the development of a markedly positive instantaneous intrathoracic pressure relative to ambient pressure. This will cause PBT with leakage of air from one or more airspaces into the pulmonary parenchyma/interstitium or pleural space. Although such events have been reported not uncommonly in the more distant past, recent experience during a five-year period in a single centre led to only seven cases in 67 000 ascents, and only one fatal outcome.18

Predicting the risk for pulmonary barotrauma

From a pathogenic point of view it is reasonable to assume that certain lung diseases will exert an important influence on the risk for PBT. Most relevant will be diseases where abnormalities of pulmonary mechanics are characterised by a broad distribution of different regional time constants—for example, diseases of the airways with regional differences in the degree of obstruction and parenchymal diseases with regional differences in the level of compliance. Several groups have reported incidents of PBT which covered the entire spectrum from mere discomfort due to minor degrees of mediastinal emphysema or pneumothorax or both, to life-threatening or fatal arterial gas DCS also had a chest CT scan following the injury. Covered the entire spectrum from mere discomfort due to minor degrees of mediastinal emphysema or pneumothorax or both, to life-threatening or fatal arterial gas embolism.10,15 The authors usefully scrutinised these events for possible predisposing risk factors such as morphological or functional pulmonary abnormalities.12,13,15

Lung cysts have been observed in subjects undergoing submarine escape training12,15–19 and in tunnel labourers working under pressure19 who developed PBT with and without arterial gas embolism. In a series of 55 fatal underwater accidents involving professional divers Calder1 found 13 cases in which he could identify lung damage as the primary cause of death. In three of four cases in which the primary cause of death was pneumothorax, bullae were present on the surface of the lungs. Another series described nine cases of PBT occurring in divers during various in-water activities, even at the surface, with or without equipment mal-function.14 Bullae, presumably pre-existing, were found in two.

These observations are interesting, yet several caveats are in order while interpreting the published findings. Since pre-dive chest radiographs were available in only very few of the reported cases,17 it might be argued that in at least some of the divers the observed abnormalities might have been a consequence rather than the cause of the diving accidents. On the other hand, relevant pulmonary abnormalities detected subsequently from CT scans might have been missed on plain chest radiographs taken before the incident, since the sensitivity of a CT scan to detect morphological abnormalities is far superior. Moreover, considerable bias may have been introduced by the publication of single case records and small case series since a control population of divers differing from the index cases only by the absence of PBT is not available.

These reservations apply also to the attempts which have been made to detect abnormalities of lung function as risk indicators for PBT. Most divers who have survived PBT had no previous lung function tests. Furthermore, lung function may differ between divers and non-diver controls, irrespective of diving accidents. For example, a Norwegian study found significantly lower values for forced expired volume in one second (FEV1), FEV1/forced vital capacity (FVC) ratio, and maximal flow rates at low lung volumes in a group of 152 professional divers compared with a matched non-diver control group of 106 subjects.22 In most papers reported functional data were limited to simple spirometric tests. Detailed analysis of the expiratory flow-volume loop, which may provide important information for the assessment of obstructive lung disease, has rarely been used.

Introductory article

The introductory article by Tezillo and colleagues23 consequently makes a valuable contribution to resolving some of these uncertainties about the potential risk factors for the development of PBT. They studied 15 patients referred to their centre with PBT and compared them with 15 patients who suffered type II (neurological) DCS without PBT. The two groups did not differ in respect to age or smoking habits. The diagnosis of either PBT or DCS was made in accordance with established criteria based on history and clinical findings.17 In all patients a pre- and post-injury lung function test was available, and all patients additionally underwent post-injury chest radiography. Pre-injury chest radiographs were available for 12 of the PBT patients (taken 5–12 months before the accident) and these revealed no abnormalities. Twelve patients with PBT and four with DCS also had a chest CT scan following the injury.

Four patients in the PBT group had reported prior lung disease (two asthma, two chest surgery in early childhood) compared with none in the DCS group. All patients in both groups had normal lung function values with respect to slow inspiratory and forced expiratory vital capacity as well as FEV1. However, analysis of pre-injury lung function parameters revealed a significant difference between the groups with respect to maximum expiratory flow rates at 50% and 25% of vital capacity (MEF50, MEF25). The mean (SD) values expressed as % predicted were, respectively, 87.5 (23.3) vs 113.1 (36.7) for MEF50 and 78.5 (28.5) vs 112.8 (44.4) for MEF25.

Chest radiographs taken following the injury showed abnormalities in five of the 15 cases of PBT (two pneumothorax, two atelectasis, one pneumo-mediastinum), all of which may have been effects rather than causes of PBT, whereas the radiographs were all normal in the patients with DCS. Chest CT scanning revealed lung cysts in five of the 12 cases of PBT, none of which were detected on the plain chest radiographs, but there were no abnormalities in the four cases of DCS.

The authors concluded that flow-volume curves (and measurements of flow at low lung volumes) could identify diving candidates who are at increased risk for PBT. Furthermore, they proposed that a CT scan of the chest should be performed in the routine investigation of diving related PBT.

These observations are interesting, but the recommendations are premature. The study was retrospective in nature and comprised a relatively small
number of patients. No definitive recommendations can be justified with regard to screening sport divers with no history of previous lung disease until there is predictive information concerning the value of these tests. Even in patients with a history of bronchial asthma, which is the most prevalent lung disease in a young population, inferences concerning fitness to dive remain controversial.23–27

In order to obtain conclusive answers on the risk of a particular lung disease for the development of PBT and eventually arterial gas embolism, it would be necessary to perform experimental studies. The outcome of completely healthy persons compared with those with fully characterised functional or morphological abnormalities ought to be observed after dives with comparable parables. Such investigations are necessarily precluded by ethical considerations.24–26,28–30 Physicians therefore have to base their recommendations on observational studies, a format notorious for low levels of objective evidence. This unsatisfactory situation is relatively common in diving medicine for obvious reasons and explains why numerous topics remain controversial even among experts in the field.

No controlled clinical data are available to allow definitive conclusions about the risk of underwater diving in patients with lung diseases, particularly in patients with a history of asthma. Hence, the recommendations are based on physiological and physical inferences and on clinical information that is mostly anecdotal.24–30 Asthma is, nevertheless, the most common respiratory disorder of young people wishing to undertake recreational or professional diving, and so I shall complete this review by considering the particular problems posed by asthma.

Diving and bronchial asthma

It is commonly agreed that symptomatic asthma is a contraindication to diving because asthmatics may become limited in their exercise capacity and because mucus in the airways may trap air distally and lead to barotrauma of ascent. However, there is no unanimity about when or whether someone with asymptomatic asthma should dive, and there is wide variability among asthmatics with measured airway obstruction as to whether there are perceived symptoms.

A conservative recommendation is that any person with asthma who experiences frequent exacerbations or continuously needs medication to control symptoms should refrain from diving, and that intending divers with a past history of asthma and asthmatic symptoms within the previous five years should be advised not to dive.31–33 However, in a recently published paper which reviewed the theoretical issues underlying this advice, and which critically examined relevant accident data, the authors concluded that “… available data suggest asthmatic patients with normal airway function at rest, and with little airway reactivity in response to exercise or cold air inhalation, have a risk of pulmonary barotrauma similar to that of normal subjects.”34 Furthermore, the consensus from international experts in diving medicine is that the emphasis should shift away from an exclusionary view to one that is focused on quantifying “function”.

No data are available which support routine screening of intending recreational divers by spirometry or chest radiography. It seems reasonably satisfactory to obtain a comprehensive history (and perhaps a confirmatory signature). Every person with a history suggestive of asthma or chronic bronchitis should then undergo pulmonary function tests including a flow-volume curve. Intending divers with a past history of chest trauma, chest surgery, or major chest infection should have a chest radiograph taken, recognizing that its sensitivity is limited compared with a CT scan.
LEARNING POINTS

- The most common causes of diving fatality are drowning and arterial gas embolism from pulmonary barotrauma of ascent.
- Less common effects of pulmonary barotrauma are mediastinal emphysema and pneumothorax.
- It is not always possible to discriminate diagnostically between arterial gas embolism to the brain as a consequence of pulmonary barotrauma and type II decompression sickness affecting the spinal cord.
- Subjects with current airway obstruction or structural abnormalities of the lung parenchyma such as lung cysts or bullae are likely to be at increased risk from PBT and should be advised not to dive.
- Subjects with a history of asthma who are without symptoms during the previous few years and have normal lung function (including a normal expiratory flow volume curve) should not be prohibited from diving.