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PULMONARY BAROTRAUMA A NEW LOOK AT MECHANISMS

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Abstract

A review is presented of pulmonary barotrauma (PBT) of ascent in the context of the more general condition of pulmonary over-inflation and lung rupture. Having identified some caveats with respect to the diagnosis of the condition and discussed the routes which air may take having left the intra-pulmonary air spaces, the epidemiology of the condition in the contexts of diving and submarine escape is discussed, noting that gas embolism appears to be a frequent consequence of PBT. Predisposing factors such as inexperience, rapid ascents and pulmonary disease are discussed before comparing the presentation of PBT in diving with that arising in aviators, with mechanical ventilation of the lung and spontaneously. Of particular interest is the observation that gas embolism is a rare complication of PBT in circumstances where the victim is surrounded by air rather than water. Having briefly discussed some relevant pulmonary mechanics, possible mechanisms whereby lung rupture in divers appears to differ from other circumstances are presented. A common breathing pattern of divers (skip-breathing) and the increase in thoracic blood volume which occurs with immersion and negative-pressure breathing are considered to be possible explanations.

Key Words

Physiology, pulmonary barotrauma.

Introduction

It is frequently difficult to establish an accurate history and firm diagnosis in people who have suffered accidents underwater. In some, barotrauma has only become obvious many hours after the last dive when precise memories of critical details have faded. In others, events have been so dramatic and rapid that patients have been unable to recall exactly what happened during their ascent. Furthermore, a rapidly evolving neurological syndrome after diving has all too frequently been blamed on PBT and arterial gas embolism even where there has been no evidence of lung injury and where assessment of the dive profile indicates that the condition is just as likely to have been a consequence of a dissolved-gas disease process. As a result, there is an aura of mystery surrounding PBT and diving. In this presentation I will review a number of aspects of pulmonary barotrauma in order to identify why it is that the lung may rupture in certain circumstances. In doing so I would like to acknowledge the substantial contribution of Professor David Denison to this work. He and I have spent many a long hour debating these issues. This paper and the more substantial book chapter from which it is drawn[1] represent a synthesis of our experiences and thoughts.

Definitions

Pulmonary barotrauma (PBT) is a collective term for two different insults to the lung. The first may occur during exposure to increasing ambient pressure (PBT of descent, or "lung squeeze") and the second during exposure to reducing pressure (PBT of ascent). The former condition is extraordinarily rare to the point that PBT has come to be synonymous with PBT of ascent. Its rarity is of interest. During the descent phase of a breath-hold dive, the thoracic gas volume (TGV) will decrease in accordance with Boyle's Law. Assuming that the lung was filled to total lung capacity (TLC) immediately prior to the dive, the depth at which this will be reduced to residual volume (RV) can be expressed as 10*(TLC/RV -1) msw. For most people this would occur at a depth of between 30 and 50 m (of sea water). If this depth were exceeded, the compliance of the lung and thoracic wall would increase, resulting in the generation of a relatively negative pressure in the airways and barotrauma of descent would result.

However, the world record breath-hold dive of 127 m, currently (April 1997) held by Pipin Ferreras indicates that, unless he has a TLC/RV ratio of 13.7 (which is unlikely), the assumptions behind such a calculation are wrong. It has been suggested that the thoracic blood volume (TBV) increases during breath hold dives and that, by effectively reducing the residual volume, this effect would increase the theoretical maximum depth of such a dive.[2] Evidence to support this idea was provided by Schaefer et al.,[3] who carefully studied a series of breathhold dives by Robert Croft to a maximum depth of 73 m. With a TLC of 9.1l and a RV of 1.3 l, his theoretical maximum depth was 69.5 m. They found that, depending upon the depth of the dive, the TBV increased by between 850 and 1,047 ml. In the same report, they calculated that for Jacques Mayol (TLC 7.2l; RV 1.88l) to perform his (then) record dive to 70 m, an increased TBV of 980 ml would have been required. It seems that this effect explains the extraordinary depths to which breathhold dives can be conducted without pulmonary injury and that factors other than lung squeeze limit the maximum depth which can be achieved by divers with normal lungs.

I will focus on PBT of ascent. Vertical movement through a column of water is associated with substantial

changes in pressure. Since the liquid and solid tissues of the body are effectively incompressible, such pressure changes are evenly distributed between these tissues and there is little alteration in their function. Indeed, very high pressures can be tolerated before there is any resultant tissue dysfunction.[4]

The situation is different for tissues which contain, or are adjacent to, a gas phase because this is liable to the constraints of Boyle's Law. For some organ systems, such as the bowel, the contraction and expansion of its gaseous contents during increases and decreases in depth are normally of no consequence. This is because the bowel wall is highly compliant and, provided that no gas is introduced at depth, such changes in volume can normally be accommodated with an insignificant pressure differential being generated across its wall. However, where tissue compliance is limited, alterations in the volume of gas will be accompanied by the development of a more substantial pressure differential between the tissue and its adjacent gaseous phase. Furthermore, if gas is introduced into the space at depth, it will expand during ascent. Unless this gas-containing space is allowed to vent freely, a pressure differential will develop between it and the surrounding tissue. This force may result in tissue injury which is known as barotrauma of ascent.[5]

With respect to the lung, there is evidence that the degree of overpressure required to cause tissue injury is dependent on the extent to which the lung is splinted by its surrounding structures.[6-8] More strictly, therefore, the tissue injury of PBT is caused by over-stretching of the lung by a transmural pressure change rather than the change in volume of the intra-thoracic gas.

Distribution of extra-pulmonary gas

Once the lung has ruptured, gas is free to escape from the airways into surrounding tissues. Although the presence of aberrant air in the pleural, mediastinal or adjacent subcutaneous spaces, or in systemic arterial blood, is usually accepted as evidence of lung rupture, this is not necessarily so.

a Firstly, extensive ruptures of the lung interior from blast injury do not lead to the escape of much air unless the victim survives to make several to many respiratory efforts.[9-14]

b Secondly, gas of non-pulmonary origin can appear in peri-pulmonary tissues (e.g the pleural, pericardial, mediastinal, peritoneal and retro-peritoneal spaces, the soft tissues of the neck and chest wall and the pulmonary and systemic vasculature) because:
   i it has been generated *in situ* by infecting organisms,
   ii swallowed air has been forcibly expelled from the GI tract, as in the ruptured oesophagus syndrome described by Boerhaave[15] and Agarwal and Miller.[16]
   iii it has escaped from an extra-pulmonary airway in the mouth, pharynx, larynx, trachea or extra-pulmonary bronchi, and may have extended downwards to the mediastinum, the pericardium, burst through the thin hilar pleura to the pleural space or carried on down below the diaphragm.[17,18]

c Thirdly, some penetrating injuries of the chest wall and much cardiothoracic surgery lead to an open traumatic pneumothorax with considerable or complete collapse of an intact lung. Closed heart bypass surgery can lead to the accidental introduction of gas emboli via bypass lines or via oxygenators (bubble- more than membrane-oxygenators).[19-21]

d Of particular importance to the investigation of diving accidents, gas absorbed during the hyperbaric exposure which preceded the ascent to the surface may leave solution and form pockets of gas in almost any location. This can occur post-mortem (Calder IM, personal communication).

Lastly, as a diagnostic feature of PBT haemoptysis may be unreliable. Blood in the airways or in sputum often comes from broken vessels in the upper respiratory tract, especially the nose and the peri-nasal sinuses. It is helpful to keep these reservations in mind when reading reports of presumed lung rupture.

Common forms of lung rupture occur when pulmonary air:

a has ruptured into the pleural space via the visceral pleura, from whence it may extend into the chest wall, particularly if an inadequate drain is in place (spontaneous or closed traumatic pneumothorax).

b has been forced through the wall of a proximal airway, travelling centripetally along the bronchovascular bundles, to reach the mediastinum (spontaneous pneumomediastinum, mediastinal emphysema) from where it can travel up into the neck (cervical emphysema), out along superficial fascial planes (subcutaneous emphysema), down into the pericardium or sub-diaphragmatic spaces (pneumopericardium, pneumoretroperitoneum, pneumoperitoneum), or burst through the thin walls of the pulmonary arteries or veins, (pulmonary and/or systemic gas embolism).

c has escaped though the walls of, usually many, distal airways into the small bronchovascular bundles, splinting the lung, as it travels centrally (pulmonary interstitial emphysema or PIE). In this condition it is increasingly difficult to breathe, or to ventilate the lung artificially, as the air-filled splints extend. A striking account of this condition, developing during spontaneous breathing, is given by Torry and Grosh who cared for

and reviewed a vast number of U.S. soldiers affected by the virulent influenza epidemic of 1918.[22] They observed that many of these patients developed PIE, seen radiographically, that progressed to pneumomediastinum and cervical emphysema which often blocked venous return. Relief of the cervical obstruction was life-saving, (see also Coelho[23]).

Following lung rupture, the escaped gas may track in a number of directions as shown in Figure 1. In the case of hyperbaric decompression, it is difficult to assess the true frequency of the various manifestations of PBT for a number of reasons:

a Not all cases are symptomatic. Even where gas embolism of the brain has occurred, the manifestations may be minor, transient and go unreported.

b In all but the most trivial of hyperbaric exposures it is impossible, in most cases, to determine with confidence whether neurological manifestations have arisen as a result of arterial gas embolism from PBT or as a result of bubbles formed in tissues supersaturated with gas.

c In many parts of the world the collection of diving accident data is haphazard and therefore incomplete.

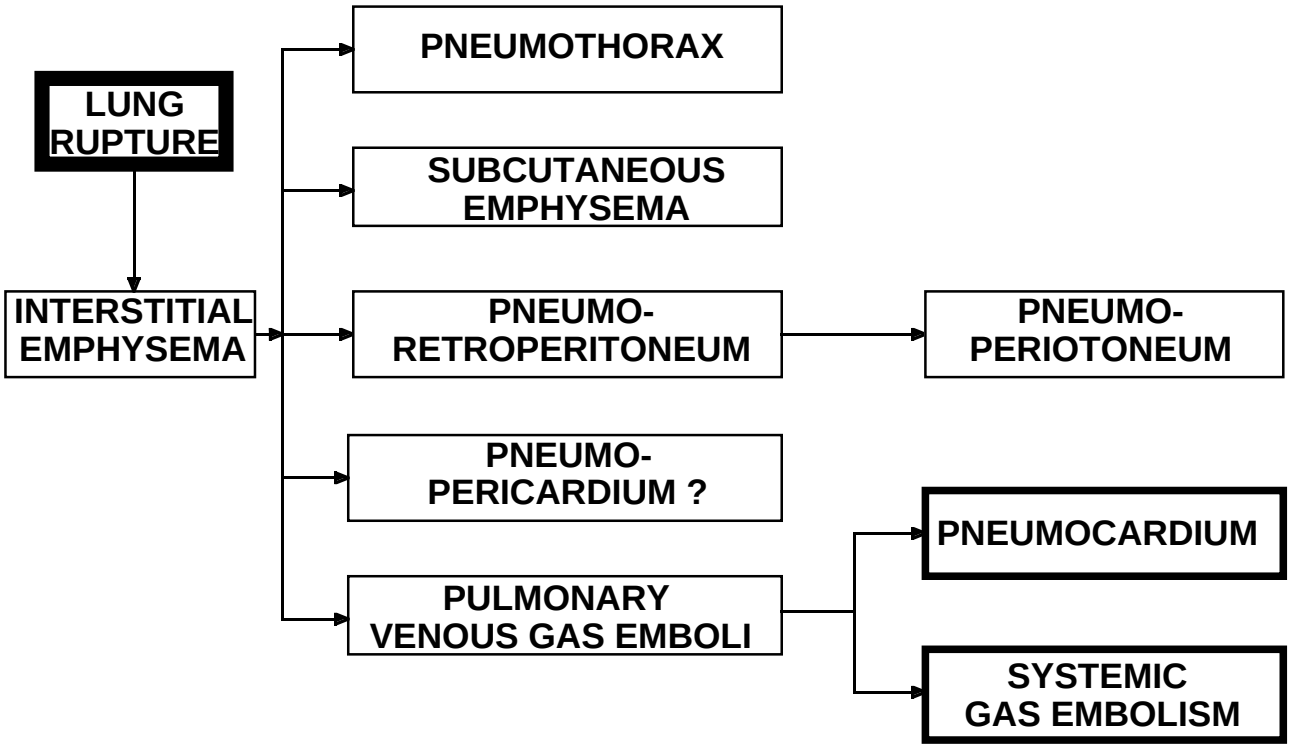

Figure 1. Possible routes for extra-pulmonary gas following pulmonary barotrauma.

TABLE 1

INCIDENCE OF PULMONARY BAROTRAUMA AND SYMPTOMS

Diagnosis	Benton[24]		Leitch[25]		Gorman[26]	
PBT only	8	9%	8	10%	42	66%
Neurological DCI	63	69%	60	77%	21	33%
Both	20	22%	10	13%	1	1%
Totals	**91**		**78**		**64**	

Under each investigator's name the first column gives the number of subjects and the next column gives the percentage of subjects with the diagnoses in the left hand column.

One specific circumstance where the latter two constraints do not apply is submarine escape training. Analysis of a series of 91 cases in which the diagnosis could be established with reasonable confidence[24] showed that pulmonary barotrauma alone occurred in eight, neurological DCI in 63 and pulmonary barotrauma with neurological DCI in 20 . PBT in this series was defined as cases in which there was x-ray or clinical evidence of a pneumothorax or extra-alveolar gas in the lung parenchyma, mediastinum or other site to which gas may track.

In a study of 140 cases of PBT in divers reported to the Institute of Naval Medicine over a period of 20 years, and which suffers from all of the above constraints, Leitch and Green described 78 cases which met similar diagnostic criteria.[25] . Gorman reported a similarly constrained study of 64 diving casualties in Australia.[26] The diagnoses of the

cases in these three studies is shown in Table 1. It can be seen that neurological manifestations, arising from the distribution of the escaped gas to the pulmonary venous circulation and subsequent cerebral arterial gas embolism, predominate. Within the non-neurological categories of PBT, pneumothorax is uncommon, present in only 5% of cases in submarine escape trainees.[27] In fatal cases, the finding of a pneumothorax at autopsy needs to be interpreted with caution as, in certain circumstances, it may be a consequence of post mortem events. In the remainder, following the escape of gas into the interstitial tissues of the lung, its migration to the mediastinum may be continued, where sufficient gas has escaped, cephalad to the subcutaneous tissues of the neck and head, or caudally in the retropleural and retroperitoneal planes to form collections beneath the diaphragm, around the great vessels of the abdomen and in the pelvis.[28] Occasionally the radiological features may suggest a pneumopericardium although close examination of the films commonly shows that such gas lies between the parietal pleura and pericardium.[5] In particularly severe cases, a pneumocardium may be seen.[29] It is assumed that gas found in the left side of the heart has travelled along the pulmonary veins and that gas in the right side of the heart has travelled against the normal flow in the pulmonary arterial tree.

Predisposing factors

A high proportion of cases of PBT occurs amongst inexperienced divers.[30-33] In a large study of accidents arising from submarine escape training, the incidence of PBT amongst initial trainees is almost double that of requalifiers.[24] In the diving setting, it is well recognised that rapid ascent is a predisposing factor for PBT.[34] This may occur as a result of panic following, for example, the loss or failure of a piece of equipment or running out of air. Should such panic result in involuntary closure of the glottis, PBT will be the consequence for all but the shallowest of ascents. Such crises are largely avoidable and panic is less likely to arise in experienced divers.[35] A potential problem during the very rapid ascents of submarine escape training is that only a brief interruption to a controlled exhalation caused by, for example, coughing, sneezing or hiccupping, may be sufficient to provoke PBT. This is particularly so during the last few metres of an ascent when the rate of increase of thoracic gas volume is at its greatest.

To avoid pulmonary over-inflation it is important to breathe out during a rapid ascent. This is not an intuitive action, particularly if the diver believes that he or she is out of air, and has to be learned. It is therefore not surprising that inexperienced divers are at greater risk of PBT than those who are more experienced. The reason why trainee submariners appear to be at greater risk than requalifiers is less clear. All Royal Navy submariners undertaking escape training are carefully instructed and supervised in the water, with particular attention being paid to adequate exhalation during ascent. There is no obvious reason why this should be less effective in initial trainees than requalifiers particularly since such training is infrequent, meaning that on each occasion the lessons need to be relearned.

It is also apparent from submarine escape training that PBT may occur in personnel who have been witnessed to exhale, apparently adequately, throughout their ascent.[25,36] Therefore, there are likely be other factors which may predispose an individual to PBT.

It has been argued for many years that conditions which cause airways obstruction predispose to PBT and such arguments can be persuasive.[37,38] Over-distension of the lung distal to the obstruction may occur as the diver ascends. In the case of reversible airways disease, support of this hypothesis has been provided by alarming anecdotal reports of asthmatics who have suffered from PBT.[39] Whilst there is little dispute that asthmatics who are symptomatic should not dive, both on the grounds of the risk of PBT and their reduced exercise tolerance, the situation with respect to asthmatics in remission is controversial.[40]

As Edmonds observes,[39] even if an asthmatic diver enters the water asymptomatic, the underwater environment provides a number of potential triggers for an acute asthmatic attack:

a Exertion (from overweighting, equipment drag, swimming against tides etc.);

b Inhalation of cold, dry air (adiabatic expansion of dehumidified, compressed air);

c Inhalation of hypertonic saline through a faulty regulator;

d Breathing against a resistance (increased gas density, faulty regulator, low air supply).

As a result of such arguments, asthmatics have been excluded from military and commercial diving for many years and discouraged from sports diving. However, such exclusion is unenforceable in most jurisdictions. The consequence of this approach has been that there is no substantial data set from which an objective assessment of the risk of PBT in asthmatics, compared with non-asthmatics, can be determined.[41,42] Recent attempts to gather such data have been criticised for poor study design[43-45] or are limited to small pilot studies.[46] Nonetheless, they indicate that the risk of asthma provoking PBT may have been overstated in the past and, until high quality data are available, this issue will remain controversial.[47]

The evidence for an association between chronic obstructive pulmonary disease (COPD) and PBT is also

scant. This is for much the same reasoning as above and also because COPD, unlike asthma, is unusual in the generally young, fit population from which submariners and military, commercial and the great majority of sports divers is drawn. There is radiological and autopsy evidence, albeit anecdotal, that bullous disease may predispose to PBT.[36,48-50] However, small bullae and apical blebs are common and may avoid detection by routine radiography. Furtermore there is anecdotal evidence that uneventful diving may be possible with encysted pockets of intra-thoracic, extra pulmonary gas.[51]

The autopsy evidence from cases of fatal PBT needs to be interpreted with caution since pulmonary over-inflation can cause blebs and careful histological examination is required to distinguish these from pre-existing disease.[49] Nonetheless, it can be argued intuitively that poorly- communicating, gas-filled spaces in the lung are liable to be vulnerable to barotrauma of ascent, particularly if the ascent is rapid. It is for this reason that Jenkins et al[52] have recommended that patients with conditions which may predispose to bullae or subpleural blebs, such as Marfan's and Ehlers-Danlos syndromes, should be considered unfit to dive.

The complete obstruction of a small airway is unlikely to predispose to PBT since the distal lung is likely to be atelectic and, even if it were not, the process of descent and ascent through a column of water is unlikely to result in a net increase in the volume of trapped gas in a similar manner to the bowel. However, incomplete obstruction could theoretically do so, in a similar manner to bullous disease, by limiting the rate at which gas distal to the obstruction can leave the lung during ascent. Despite the attractiveness of this hypothesis, there is a dearth of compelling evidence to support it. Liebow et al. reported a case, which has been frequently quoted, of fatal PBT in a submariner undergoing escape training.[48] At autopsy, an irregular calcified mass was found in the left main bronchus and a second in a superior segmental bronchus of the left lower lobe. The latter was associated with bronchiectasis, from which it can be implied that there was substantial chronic airway obstruction. Of interest, however, is the fact that the extent of the PBT in this case was not confined to the left lower lobe or even the left lung, since a pneumothorax and substantial interstitial emphysema were found on the right side. Although the finding of interstitial emphysema associated with the bronchiectasis may be evidence that localised airway obstruction is a predisposing factor for PBT, the question is left open as to whether the affected lung is more vulnerable than that served by normal airways.

Unsworth described a case of tension pneumothorax which arose following a session of hyperbaric oxygen therapy.[53] The patient was subsequently found to have a neoplastic mass in the right middle lobe. The pneumothorax was effectively treated and there were consequently no acute autopsy findings. The attribution of the pneumothorax to airway obstruction due to the neoplasm has, therefore, to be speculative. The same argument applies to the case reported by Calder in which localised airway obstruction was implied from the finding of clinical and histological evidence of acute bronchiolitis.[49]

To further confound the role of involuntary airways obstruction, it now appears that indices of obstruction do not correlate with the risk of sustaining PBT. Benton et al. studied the screening spirometry data of a population of healthy Royal Navy submarine escape trainees who had suffered unequivocal PBT.[24] They confirmed the observation of Brooks et al[54] that a lower than predicted forced vital capacity (FVC) was associated with PBT rather than any measured index of obstruction.

There can be little doubt that pleural scarring can predispose to PBT, particularly pneumothorax.[8,48,49] However, this is again a common condition and liable, if limited in extent, to be missed on a routine chest x-ray, which in any case may not be required for sports divers. Since it is likely that many thousands of uneventful dives have been conducted by personnel with pleural scarring, the magnitude of the risk of PBT associated with this is likely to be small.

Lung rupture in other circumstances

Before going on to discuss how the lung ruptures, it is worthwhile studying circumstances other than diving in which the lung may rupture. Rapid decompression to altitude is an obvious place to start. Although the early work of Robert Boyle,[55] Paul Bert[56] and others identified hypoxia and decompression illness as limitations to hypobaric decompression, it was not until the early 20th century that strategies to overcome these problems, other than the provision of oxygen at ambient pressure, were developed. Haldane realised that the maximum altitude to which men could go when breathing pure oxygen at ambient pressure would be about 40,000 ft.[57] He also calculated that delivering oxygen at an overpressure of about 15 mm Hg would enable them to fly a few thousand feet higher.

Towards the end of World War II, the pressurised aircraft cabin was developed. This obviated the need for prolonged positive-pressure breathing and allowed aircraft to climb much higher. However it introduced two new problems, those of rapid decompression and of the brief but profound positive-pressure breathing regimes that would be required to bring aircrew back safely, if they lost cabin pressures suddenly at altitudes well above 45,000 ft. Thus air forces throughout the world embarked on a series of theoretical, animal and human studies on rapid decompression and brief but severe positive pressure breathing.

In essence these studies have shown that the human lung can be decompressed very rapidly indeed without coming to harm, providing that the glottis is kept open during the decompression. Heath reported a series of 86,916 decompression runs to altitude that were without significant adverse effects.[58] These included 771 rapid decompressions. Hitchcock et al. exposed 150 subjects to a total of 550 "explosive" decompressions.[59] Holstrom reported the safe outcomes of a large series of decompressions from an initial altitude of 10,000 ft to a final altitude of 40,000 ft in 1.6-2.3 sec.[60] Bryan and Leach did likewise for a series from 8,000 ft to 40,000 ft (565 mm Hg to 141 mm Hg) in 2.5 sec.[61] Ernsting et al. safely completed 6 decompressions on each of 3 subjects, from 8,000 ft to 40,000 ft in 1.6 seconds.[62] Holness et al. successfully decompressed two subjects from 22,500 ft to 60,000 ft and from 45,000 ft to 80,000 ft.[63] The rate of decompression in these studies works out as roughly twice the rate achieved in the last 10 m of a submarine escape training run.

Meanwhile actual decompressions in flight were providing more evidence that the human lung could survive quite rapid decompressions. For example Achiary et al[64] reported satisfactory outcomes to 15 abrupt decompressions with time constants of 0.1 to 3 sec, in French high performance aircraft, and Brooks[65] did likewise with 29 incidents of massive/explosive decompression in Canadian fighter aircraft over the 20 year period 1962-1982. In almost every case the crew were able to land their aircraft safely.

Although millions of passengers, many of whom have respiratory disease, travel commercially each year, PBT is rare in flight or in decompression chambers. In Cran and Rumball's series of 994 spontaneous pneumothoraces occurring in RAF personnel over a 22 year period, only 8 occurred in flight.[66] This incidence was judged to be proportionate to the fraction of time spent in the air and was not thought to have been caused by the flights. Nonetheless, dramatic exceptions have been described in cases of pre-existing lung disease.[67,68] In contrast to decompression from depth, decompression to altitude rarely causes arterial gas embolism and only a single case report has been found.[69]

Another circumstance in which PBT can occur is with mechanical ventilation. There are several forms of mechanical ventilation. In one, gas is supplied to the respiratory tract at higher than ambient pressure, via an oronasal mask or an endotracheal tube (positive pressure ventilation). In a second, all of the body but for the head and neck is surrounded by a rigid vessel (or iron lung) which is cyclically decompressed whilst the airway is exposed to ambient air pressure, (negative pressure ventilation). In all but very minor details, these two forms of artificial ventilation have identical effects upon the lung and circulation. Both inflate the lungs but obstruct the circulation.[70]

In a third form, the chest wall alone is surrounded by a rigid jerkin which is cyclically decompressed whilst the rest of the body remains exposed to ambient pressure. This form of ventilation inflates the lung and promotes the circulation.[71]

Experiments on positive pressure inflation of fresh human cadavers show that lungs expanding in an unsupported thorax burst at about 70 mm Hg but, if the thorax is confined, they rupture in a different manner at pressures of about 110 mm Hg.[7,8] Many animal experiments have confirmed this.[72,73] In the early days of mechanical ventilation (with the Drinker negative-pressure iron lung) the technique was applied successfully for long periods of time at relatively low transthoracic pressures, about 30 cm of water, in patients with neurological disorders (usually polio) but healthy lungs. Since then, positive-pressure ventilation, often delivered by endotracheal tube, has been applied to very many people with damaged lungs (most frequently neonatal or adult respiratory distress syndromes). Not surprisingly, studies of such cases have shown that diseased lungs are more fragile than healthy ones. Diseased lungs often become stiff because of consolidation, loss of surfactant, fibrosis and/or pulmonary interstitial emphysema. Stiff lungs need higher pressures to ventilate them.[74] The pressure-volume curve of the diseased lung reflects the properties of the ventilateable part of the lung as has been demonstrated by the use of computed tomography.[75] Mechanical ventilation leads to pulmonary capillary rupture and to pulmonary oedema. These complications appear to be a function of lung volume rather than inflation pressure, so much so that Dreyfus et al. have proposed the term "barotrauma" should be replaced by "volutrauma".[76,77] Positive pressure ventilation opposes systemic venous return, causing pulmonary vessels to collapse, increasing the transmural stress on neighbouring alveoli.[78] Systemic hypovolaemia compounds this and may increase the risk of lung rupture.[79] Often a rise in pulmonary arterial pressure, i.e upstream of the compressed intra-pulmonary vessels, precedes a pneumomediastinum. Once the gas has escaped into the soft peri-pulmonary tissues (surgical emphysema) it can block cerebral venous return at the base of the neck.[23] It can also escape from the soft tissues into systemic veins leading to paradoxical systemic venous gas embolism.[80]

The commonest sequences observed in mechanically ventilated patients on Intensive Care Units are pulmonary interstitial emphysema (PIE) leading centripetally to pneumomediastinum and cervical emphysema and centrifugally to the appearance of sub-pleural pulmonary blebs. The appearance of blebs and/or surgical emphysema are ominous signs that a pneumothorax will soon follow. Sick patients tolerate pneumothoraces badly because they have little pulmonary reserve. In addition most pneumothoraces on mechanically ventilated patients proceed to tension pneumothoraces.[81,82]

In summary, many studies of lung damage during mechanical ventilation support the idea that the risk of rupture is rare at inflation pressures of 40 mm Hg or less and substantial at pressures of 70 mm Hg or more. Some show that respiratory gas can escape into the pulmonary arteries or capillaries and travel against the flow to fill the right heart with foam, or it can break into the pulmonary veins and travel with the flow to the left heart and to the coronary, carotid and cerebral circulations.

Finally, two common manifestations of PBT occur spontaneously. Spontaneous pneumothoraces have a trimodal distribution with patients' age, affecting neonates with respiratory distress, young adults with no other apparent lung disorder and patients over the age of 40 years who commonly have extensive, pre-existing lung disease. It is the middle group, of primary spontaneous pneumothoraces in young adults, who are of interest to diving medicine. Up to the early 1930's they were always attributed, without proof, to tuberculosis, but then Kjaergaard showed that they were rarely accompanied by any underlying lung disease.[83] Such primary spontaneous pneumothoraces may be caused by sudden, but not necessarily forceful, distortions of the chest wall, as in twisting to attach or find a seat-belt, but most develop when subjects are at rest. Sixty percent are noticed in the first three hours of the waking day.[84] They are much more frequent in young men than young women (in the ratio of 6 or 7 to 1) and they are very much more common in smokers than non-smokers. In young men "light" smoking increases the risk seven fold, moderate smoking raises it some twenty fold and heavy smoking about one hundred fold.[85,86] Almost all are unilateral and affect the right and left lungs with equal frequency, but some 2% are bilateral, probably betraying a defect in the mesothelial barrier separating the two pleural spaces in the antero-superior mediastinum. They rarely occur after the age of 40 years. Recurrence rates after recovery from a first spontaneous pneumothorax are high (about 50%), are more commonly ipsilateral than contralateral but are very infrequent after intervals of two years or more.[85,87,88]

Primary spontaneous pneumothoraces are believed to be due to the rupture of apical subpleural blebs that have probably filled with alveolar air dissecting from splits in local small airways. The apices are the sites of stress concentrations in the upright lung and it is thought this explains the higher incidence in tall, young men with tall, thin chests.[89] Such blebs are also found at the sharp edged apices of the lower lobes. The gravity-dependent density gradients seen in CT scans of healthy lobes suggest that, in the supine and prone postures at least, the major fissures act as shelves on which the uppermost lobe rests and from which the lowermost lobe hangs, so it is possible that there are important stress concentrations at these sites also.[90,91]

Some spontaneous pneumothoraces are associated with forced inspiratory manoeuvres such as hiccupping or the completion of tests of total lung capacity or peak inspiratory pressures.[92,93] Such cases suggest that voluntary high inflations can stretch some parts of healthy lungs beyond their elastic limits

Other clinical experiences at sea-level show that sudden and forceful expiratory efforts (e.g Valsalva manoeuvres as in child-birth, weight-lifting, playing the trumpet, violent coughing against an expiratory resistance or repeated attempts at producing a maximal expiratory pressure as a test of lung function can force air through the walls of the respiratory tract, most probably at extra-pulmonary or at extra-thoracic levels.[94-97]

Spontaneous pneumomediastinums are probably about 10 times rarer than spontaneous pneumothoraces. However, as many are asymptomatic and only half of them are visible on a straight postero-anterior radiograph of the chest, their true incidence is unknown. They are said to be present in 5% of all children X-rayed for asthma.[98,99] Often they are only suspected once dysphonia or cervical subcutaneous emphysema become obvious.[100] Their natural history and management are reviewed extensively by Pierson.[101] Recurrences are infrequent. The commonest presenting symptoms are: retrosternal pain (88%), dyspnoea (60%) and dysphagia (40%). Sixty percent also have cervical subcutaneous emphysema and one in eight have a concurrent pneumothorax.[102] In one retrospective study of 17 patients aged 12 years or more, 12 of the 17 gave a history of performing Valsalva manoeuvres during inhaled drug abuse.[103] Recently Fujiwara has reported spontaneous pneumomediastinum as a complication in 15% of patients with fibrotic lung disease.[104]

Summary of the circumstances for and consequences of lung rupture

The diving population, being predominantly male and aged between the late teens and early forties, is vulnerable to spontaneous lung rupture in the absence of any significant mechanical stress. When this happens it results in a primary pneumothorax or, more rarely, a pneumomediastinum. From a diving viewpoint, primary spontaneous pneumothoraces in the under 40 age group are unlikely to recur after an interval of two years. In the over 40 group, they are associated with pre-existing lung disease and are more likely to proceed to a tension pneumothorax and to recur. Since the diving population is generally fit, secondary spontaneous pneumothoraces in the over 40 age group is not a common problem. Traumatic pneumothoraces, in the absence of other lung disease, heal naturally and do not recur.

We know from experiments and experience with ventilators that normal lungs will rupture at an inflation pressure above about 70 mm Hg and that diseased lungs

will do so at a lower pressure. Again, the common presentations are of pneumomediastinum and pneumothorax while gas embolism is rare. Healthy lungs can be decompressed very rapidly to altitude over a pressure range of up to 0.5 bar without injury. If the lung is damaged, pneumothorax and pneumomediastinum are common and gas embolism is very rare.

In diving, the situation is different. Although very rapid ascents from great depth can be achieved without pulmonary injury, lung rupture can occur from a dive as shallow as 1 msw[105] in which the overpressure generated, assuming a breath-holding ascent at TLC, cannot be much greater than about 70 mm Hg. Although quantification is difficult, PBT in divers and submarine escape trainees appears to be more common and results in gas embolism far more frequently than in other circumstances, with pneumothorax being rare. In order to address why this may be it is necessary to review a little pulmonary anatomy and physiology.

Some relevant pulmonary anatomy and physiology

The pulmonary arterial tree lies directly alongside the bronchial tree, sharing a common capsule. The pulmonary venous tree lies between, but separated from, the arterio-bronchial bundles. As the trees approach the hilum the veins and airways come closer together. Most alveoli abut on each other but some lie directly alongside the broncho-arterial bundles that penetrate the lung. These marginal alveoli may be critical in some forms of lung rupture.[78] The healthy pleural cavity used to be thought of as a potential, rather than actual, space, but recent electron microscopic studies have shown it to be a continuous liquid film about 1 μm thick with a volume of a few millilitres.[106]

In water, the variations in ambient pressure between the uppermost and lowermost parts of a submerged water-filled sac, such as a lung-less human body, are considerable but balanced by almost equal hydrostatic gradients within.[107] In a gas-filled sac (Figure 2), such as the thorax, submerged to the same depth, the gas pressure within is determined by the net force pressing on the asymmetrically shaped vessel from without. It is that pressure which would exist at the sac's centroid of pressure (C_p), were it to be filled with water continuous with the water outside. Many experiments have shown that, for a typical adult male thorax, this point lies in the mid-sagittal plane some 19 cm below and some 4 cm behind the sternal notch, i.e. somewhere in the middle of the right atrium (Figure 3).[108] Gas delivered to the lung at this pressure, when the chest wall is relaxed, will inflate it to its normal functional residual capacity. This eupnoeic pressure is that at which the work of breathing underwater is minimised. By holding a regulator in his mouth, a diver who is vertical in the water is breathes at a relatively negative pressure.

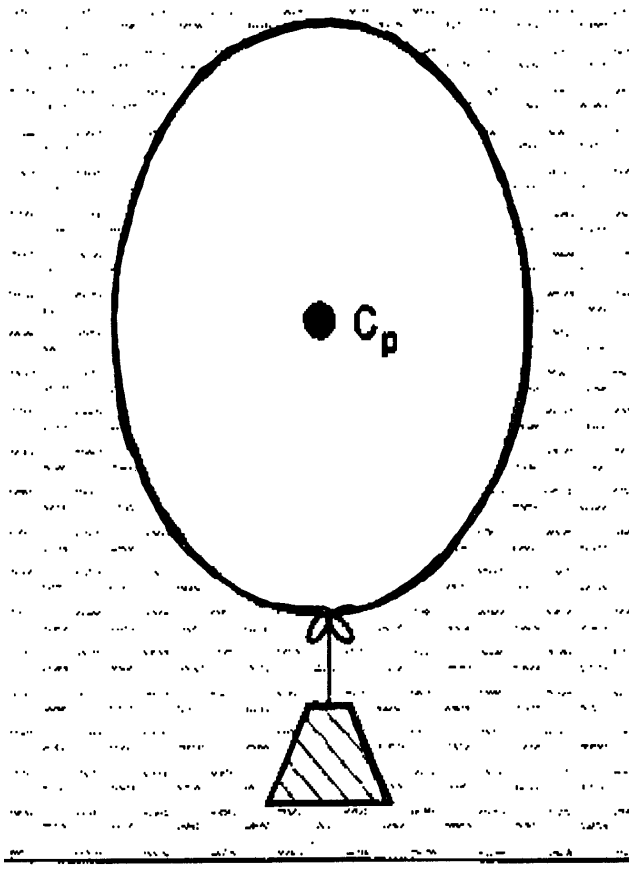

Figure 2. The centroid of pressure (C_p) of a weighted balloon immersed in water.

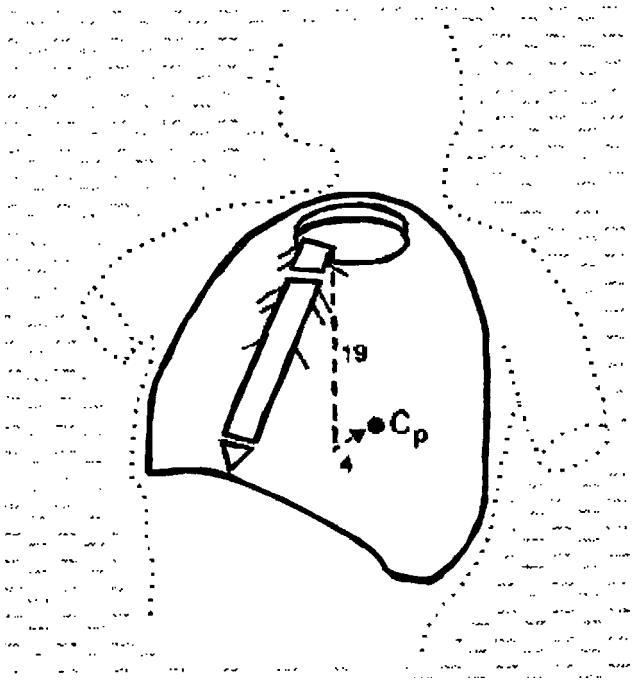

Figure 3. The centroid of pressure (C_p) of the human chest is located approximately 19 cm below and 4 cm behind the sternal notch.

Figure 4 shows an elastic, gas-filled balloon, modelling the lung, that empties through a floppy tube, modelling the airways, when they are compressed by the syringe or thorax. As the plunger is advanced the pressure immediately outside the balloon and tube rises. The pressure within the balloon is augmented by the elastic recoil pressure of the balloon at its volume at any instant. The pressure along the tube falls from the sum of $P_{syringe}$ and P_{recoil} at one end to $P_{ambient}$ at the other. It follows that at some point along the tube the internal pressure will equal $P_{syringe}$. Proximal to that **equal pressure point** the floppy tube will be inflated, distal to that point it will be collapsed (unless the airway at that point is rigid). The higher the value of $P_{balloon}$, the more completely the balloon will empty. At RV in adults, each airway will be closed at some point along its length. If this system is decompressed, the equal pressure point will move to the right as $P_{ambient}$ is reduced and the balloon will empty.

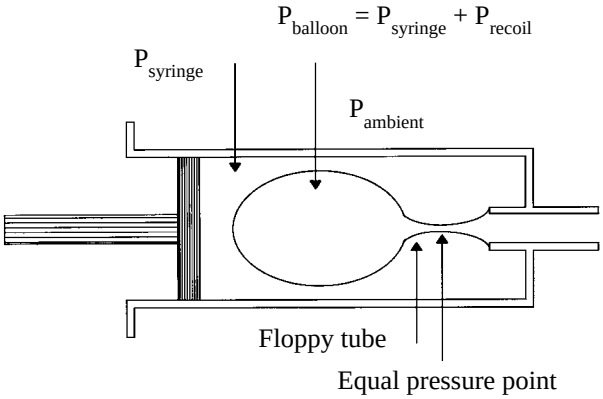

Figure 4. A balloon-in-syringe model of the lung showing airway collapse at the equal pressure point (see text).

The chest wall has a tendency to spring outwards and the lung has a tendency to collapse inwards. The elastin fibres of the broncho-alveolar tree are the prime determinants of the stretchability or compliance of the lung, i.e the instantaneous slope of its volume/pressure curve. This curve is sigmoid and eventually almost flat, indicating that its maximum compliance is in its mid-volume range. It becomes more rigid as it approaches residual volume and stiffens markedly as it approaches its maximum volume. The collagen fibres of the tree and pleura set the upper limit to its expansion.[109-111]

The inspiratory power of the chest wall is maximum at minimum lung volume and least at maximum lung volume. The reverse is true for its expiratory power. The maximum gas volume of the lung that can be achieved by a voluntary inspiration is described as Total Lung Capacity (TLC). It is reached when the diminishing inspiratory power of the expanding chest wall is balanced by the increasing elastic recoil pressure of the enlarging lung. Note that the lung itself can expand by a further 15% or so before it reaches its elastic limit. The minimum gas volume of the lung that can be achieved by voluntary expiration is described as its residual volume (RV). In children and adolescents it is set by the power of the expiratory muscles (mainly the abdominal wall) but if their chest walls are squeezed when they are at RV, more air can be heard to leave. In adults, RV is set by the collapsibility of the bronchial tree as bronchial closure proceeds reaching the elastic limit of the chest wall. When their chests are squeezed at RV no more air comes out although some 1.5 litres are still within.

In essence the lung hangs from the uppermost parietal pleural surface and sometimes rests on the lowermost parietal pleural surface, that is to say that the weight of the whole lung is concentrated at the apex of its upper surface (in any posture). The stress concentration that results is believed to favour the development of sub-pleural blebs of air that have escaped from alveoli which have fractured locally.[89,112]

On inspiration the expanding chest wall increases the negative pressure in the pleural film expanding the intra- and extra-pulmonary airways in the thorax, but the negative pressure conveyed to the gas in the airways is then transmitted to the extra-thoracic airways, which tend to collapse. On expiration, the compressing chest wall raises the pleural pressure causing the extra-pulmonary, intra-thoracic airways to collapse immediately (pressure-dependent collapse) and causing the intra-pulmonary airways to collapse progressively as emptying proceeds (volume-dependent collapse) until, at RV, all routes to the mouth are closed somewhere along their length.

In a forced expiration, peak flows rise to twice the FVC/second. After one second, about 75% of the vital capacity has been exhaled. This volume known as the forced expired volume in 1 second, or FEV_1, is frequently compared with the FVC, and this FEV_1/FVC ratio is used as an index of airway obstruction but, in relation to pulmonary barotrauma, this is misleading. When the positively pressurised lung empties during sustained decompression it does so from a near-maximal volume to which the peak expiratory flow (PEF) is more relevant. The PEF indicates that the normal lung at TLC will vent a volume equal to one vital capacity in about one-fifth of a second, i.e. the fully inflated respiratory system behaves as if it had an unimpeded time-constant of emptying of no more than 0.3 sec. The experimental work of Haber and Clamann suggests that this figure will be almost independent of depth.[113] The irrelevance of the FEV_1/FVC ratio in predicting those vulnerable to PBT in diving or submarine escape has been shown at the Institute of Naval Medicine over the past 15 years. Following a series of accidents in the Submarine Escape Training Tank in the early 1970s, the routine spirometric testing of submariners and divers was introduced. At entry into their relevant service they had to achieve an FEV_1/FVC ratio of 75% and subsequently 70%. This resulted in approximately 12% of new entrants being

rejected on spirometric grounds, an unacceptable wastage rate with the manpower reductions in the Navy of the early 1980s. To address this issue it was agreed in 1983 that, provided other aspects of lung function (notably transfer factor and lung volumes) were within normal limits and there was no evidence of gas trapping, candidates who failed their Vitalograph would be permitted to undertake submarine escape training or diving. The rate of rejection of candidates fell from 12% to less than 2%, with no increase in PBT rates for either group.

Proposed mechanisms for lung rupture in divers

We know that divers who fail to exhale during ascent, or do so inadequately, rupture their lungs. Although very occasionally cases are reported in the literature where a pulmonary lesion may have been the cause of the problem, there remains a group of divers and submarine escape trainees for whom, despite extensive investigation, no explanation for their PBT can be found. This highlights, amongst other things, the limitations of even the most sophisticated investigative tools which are frequently disadvantaged by being employed some time after the accident. However, since only a very small lung defect is required for it to rupture, it is considered unlikely that even investigation shortly after the incident would prove more fruitful. The following ideas are presented as possible explanations for these cases.

1 It is important to appreciate that the lung ruptures when it is stretched beyond its elastic limit. In some people, this can occur when a deep breath is taken and held. In normal life at an ambient pressure of about 1 bar (ATA), when the lung ruptures in these circumstances, it would appear that the gas escapes into the mediastinum. The tears in these instances must have occurred in the lung interior, otherwise they would have led to pneumothoraces and they must have been close to broncho-vascular bundles, otherwise gas would not have been able to reach the mediastinum. In diving, it is considered that this is more likely to occur and that the condition will be symptomatic. It is more likely to occur because divers commonly skip breathe in which the normal sequence of: inhale - exhale - pause, is changed to: inhale - pause - exhale. It can be seen that this technique exposes the lung to a greater period of time during which it is at, or close to, its elastic limit, thereby increasing the opportunity for it to rupture. Gas escaping from the lung at depth will expand in accordance with Boyle's Law during ascent to the surface. By effectively increasing the volume of gas which has escaped from the lung, this will increase the probability that symptoms will arise.

2 Why, as Colebatch and Brooks and Pethybridge proposed,[54,114,115] is rupture of the lung during diving more likely to occur in small, stiff lungs than in large obstructed ones? The Macklins' elegant ideas on the particular vulnerability of marginal alveoli, close to bronchovascular bundles, and the possible compounding effect of hypovolaemia should be noted.[78] This, they argued, could increase the stress on the marginal alveoli if the cardiac output could not increase, thereby allowing the bronchovascular bundles to expand at a similar rate to the gas-containing elements of the lung. However, David Denison and I wish to add two additional proposals:

a Figure 5 shows an imaginary spherical lung in which bronchovascular bundles and their associated collagen and elastin fibres radiate outwards from the centre of the sphere. As the normal lung is inflated, it expands isotropically and stress is evenly distributed along each bundle with no one more liable to rupture than the others. Now introduce fibrosis into the middle third of one element and inflate the lung again. The fibrosed part is stronger than the rest of the lung and therefore less liable to tear. But, because it also can not stretch, it puts additional stress on the normal elements in series with it, making them more liable than the rest of the bundles to rupture. This may explain Calder's observation that he could find no consistent association between the sites of lung rupture and pulmonary scarring.[49]

b A feature of the submarine escape trainees and divers, which differs from all the other circumstances in which PBT may occur discussed above, is that they are immersed in water. In the case of submarine escape trainees using either a submarine escape and immersion suit (SEIS) or a Steinke hood, they are invariably head-up and vertical in the water column and therefore subjected to negative-pressure breathing because their

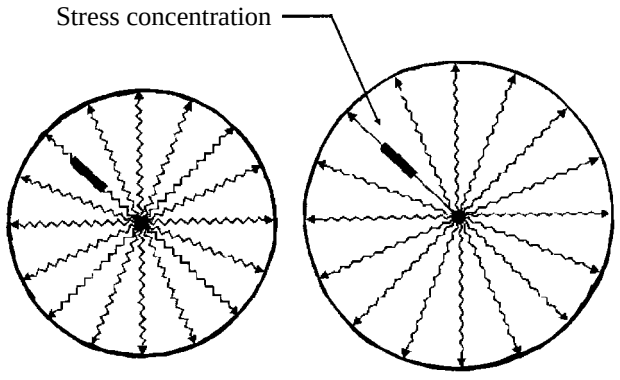

Figure 5. A model of the lung showing, on the left, a representation of the radial arrangement of the collagen and elastin fibres at functional residual capacity (FRC). The middle third of one of these is fibrosed and incapable of stretching. At total lung capacity (TLC), shown on the right, the stress is concentrated at the fibers which are in series with the fibrosis and it is here that they may rupture.

mouth is above the centrum of the lungs. For most divers (saturation divers being a notable exception), the same applies during surfacing and, in addition to the negative pressure breathing, additional negative pressure is commonly required each breathing cycle to trigger the regulator. Although the extent of the increase in TBV (discussed under lung squeeze in breath-hold divers) is less when breathing apparatus is worn underwater, there is still a displacement of blood into the thorax which occurs with submersion, particularly during negative pressure breathing.[116,117] This causes a reduction in compliance of the lung which is liable to make it more susceptible to tearing.

We have seen that, as the lung reaches TLC, its compliance is reduced and, in a setting of potential pulmonary over-inflation, it becomes more likely to rupture. It may be, therefore, that lungs of normal compliance, but which are smaller than predicted for an individual, are at a greater risk of tearing than normally-sized lungs because they are more readily overfilled. This may be an explanation for the findings of Brooks and Pethybridge et al. that a low FVC is associated with PBT rather than any spirometric index of obstruction.[24,54]

In most circumstances where the lung is exposed to an over-pressure, such as in mechanical ventilation, the over-pressure causes the pulmonary vasculature to empty. This may serve as a protective mechanism to prevent escaping gas from entering the pulmonary circulation. Immersion in water and negative-pressure breathing oppose this process, such that the TBV of divers and submarine escape trainees is liable to be greater at the point of lung rupture than in most other circumstances and most notably the military pilot subjected to positive pressure breathing during explosive decompression. Could this be an explanation for the far higher prevalence of apparent gas embolism arising from in-water PBT than from other causes?

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DIVING AND THE LUNG

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Key Words
Physiology, pulmonary barotrauma.

Introduction

The respiratory system is affected by diving via a number of mechanisms. The increased flow resistance