Autopsy and experimental observations on factors leading to barotrauma in man

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Calder IM. Autopsy and experimental observations on factors leading to barotrauma in man. Undersea Biomed Res 1985; 12(1):165–182.—Morbid anatomical data have been examined in detail from 13 fatal diving accidents in which the primary cause of death was due to pulmonary damage. In addition, the observations on 6 cases of experimental barotrauma on cadavers are recorded. The information is supplemented by the incidental findings in the respiratory system of 19 professional divers killed by causes other than barotrauma, and 97 healthy, age-matched controls who died suddenly or unnaturally. In cases of pneumothorax resulting both from diving and experiment the constant feature was the presence of pleural adhesions and lung bullae, some of which were involved in causing tissue damage. A high proportion (up to 26%) of lungs not involved in barotrauma also showed pleural adhesions. In cases of barotrauma, the parenchymal damage is not consistently related to scars or fibrous tissue. The observations suggest that a high incidence of pleural lesions are not detected or detectable on routine medical examination which do not compromise the lung. However once a pneumothorax has occurred it is highly probable that a further incident may rupture another bulla and these appear to be multiple. The converse appears to be true with parenchymal barotrauma that is not consistently related to scar tissue. This would therefore suggest that in a proven and recovered case of barotrauma it should not necessarily debar further diving activity.

barotrauma
pneumothorax
experiments
lungs
diving accidents

Pulmonary barotrauma may result from pressure differentials as low as 50 mmHg (70 cm of water) according to Kidd (1). However, many lungs are excessively stressed without giving rise to overt clinical signs but nevertheless unexplained barotrauma occurs, and Colebatch et al. (2) found survivors to have reduced lung compliance. Mead et al. (3) examined the elasticity of lung parenchyma and suggested that normally compliant lung could be overstressed by adjacent, unusually stiff lung during ascent from depth. Clarke and Rogers (4) found in experimental animals that isolated compliance measurements were inadequate as an index of barotrauma susceptibility.
The purpose of this paper is to examine available human data to establish a pattern of findings that could be used as predictors or indicators of barotrauma risk factors and whether these could be determined by clinical examination.

It is first necessary to consider the morbid anatomical features predisposing to barotrauma and then leading to gas embolism or pneumothorax. These can be classified as either intrinsic or promoted. Intrinsic is regarded as inbuilt, structural abnormalities that are essentially compliance factors. Promoted is considered to result from stresses in a lung already compromised by earlier insult, with tearing of alveolar walls at the peripheries of lungs giving rise to bullae (Fig. 1). The formation of a bulla establishes a physical weakness in lung parenchyma, which may increase in size due to local effects of pressure differential and is an increasingly self-potentiating lesion until rupture and pneumothorax occur. Mathematically the development of such a lesion can be regarded as analogous to the pressure effects on the surface of a hemisphere, which is represented by the formula $2\sqrt{3}\pi r^2$. Thus, once such a weakness has developed the potential distention of a bulla will be related to the square of the area, and rapid distention and rupture are brought about by small pressure changes. From the practical aspect it is necessary to accurately but simply differentiate and define.

MATERIALS AND METHODS

Materials from these sources were used in this investigation, and are summarized as:

1. Autopsies on underwater deaths resulting from lung damage;

Fig. 1. Periphery of lung showing dilation of terminal airways and destruction of alveolar walls to form bullae. Hematoxylin and eosin $\times 120$. 
BAROTRAUMA IN MAN

2. experimental barotrauma on cadavers;
3. examination of routine autopsies on a series of sudden deaths to establish the incidence of lung lesions in a "normal population."

Of primary importance was the accurate interpretation of the findings to establish a definitive diagnosis. All cases were subjected to radiographs of the chest before dissection. In addition lesions, which included blood-gas interfaces, were subjected to histological examination to establish whether a vital reaction was present. This was of value to eliminate artifacts produced by handling of the cadaver or resulting from attempts at resuscitation. The overall clinicopathological discussion of each case resulted in an accurate appraisal of the differential diagnosis with special reference to pneumothorax and barotrauma. The former was recognized by the significant presence of gas in the pleural cavity. From the morbid anatomical aspect barotrauma was more difficult to establish. For the purpose of this investigation parenchymal hemorrhage associated with significant antemortem arterial gas embolism.

Autopsy data

In a series of 55 fatal underwater accidents involving professional divers between 1969 and 1976, Calder (5) found 13 to which he could attribute lung damage as the primary cause of death. Tables 1 and 2. All cases had been subjected to routine medical examination within a year of death, which included chest x-ray and vitalograph studies, and revealed no abnormality. Of these deaths, 9 were due to barotrauma and 4 to pneumothorax, all the lungs were routinely fixed with 10% formal saline by insufflation using the technique described by Heard (6). Of the remaining 42 cases in which lung damage was not a feature, 9 were suitable for application of this method of fixation and could be regarded as representative of a population of divers. All lungs were routinely sliced at 0.5–1.0 cm by the method described by Kleinerman and Cowdrey (7).

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, yr</th>
<th>History</th>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25</td>
<td>Rapid ascent from 40 m</td>
<td>Escape of air from surface of left upper lobe</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>Controlled ascent from 10 m</td>
<td>Rupture of blebs on margin of right lower lobe</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>Ascent from 20 m</td>
<td>No lesion detected</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
<td>Free ascent from 30 m</td>
<td>Generalized surface rupture of both lungs</td>
</tr>
</tbody>
</table>
### Table 2

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, yr</th>
<th>History</th>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>23</td>
<td>Ascent from 65 m in saturation</td>
<td>Hemorrhage at hilum TB focus in left upper lobe</td>
</tr>
<tr>
<td>6</td>
<td>26</td>
<td>Breath holding to 10 m</td>
<td>Hemorrhage in upper lobes of lungs</td>
</tr>
<tr>
<td>7</td>
<td>21</td>
<td>Uncontrolled ascent from 30 m</td>
<td>Interstitial emphysema Diffuse hemorrhage</td>
</tr>
<tr>
<td>8</td>
<td>33</td>
<td>Ascent by open bell from 80 m saturation</td>
<td>Diffuse hemorrhage Interstitial emphysema</td>
</tr>
<tr>
<td>9</td>
<td>22</td>
<td>Free ascent from 33 m following failure in gas supply</td>
<td>Blebs in parenchyma of lungs TB focus in left upper left apex.</td>
</tr>
<tr>
<td>10</td>
<td>28</td>
<td>Drop in gas supply pressure at 95 m</td>
<td>Discreet hemorrhage in upper and lower lobes Adhesions at bases</td>
</tr>
<tr>
<td>11</td>
<td>22</td>
<td>Panic ascent from 30 m</td>
<td>Hemorrhage at margin of lungs</td>
</tr>
<tr>
<td>12</td>
<td>27</td>
<td>Failure of bell at 80 m saturation</td>
<td>Diffuse hemorrhage Adhesions at bases</td>
</tr>
<tr>
<td>13</td>
<td>34</td>
<td>Fall in bell pressure at 70 m saturation</td>
<td>Discreet hemorrhage in parenchyma</td>
</tr>
</tbody>
</table>

**Experimental barotrauma**

Seven fresh young cadavers were subjected to a modified technique described by Malhotra and Wright (8) to induce lung damage, which could then be confirmed by dissection. None had been divers or compressed air workers.

A diagram of the apparatus used is shown in Fig. 2. The trachea was exposed in the neck by mid-line incision and blunt dissection, and blood vessels were ligated. The trachea was divided below the first ring of the larynx and an airway of suitable size inserted. The trachea was bound by two layers of 2.5 cm "Elastoplast" to prevent the ligature cutting into the soft tissues. The airway was connected to the inflation apparatus by pressure tubing.

Air was fed into the 10 liter desiccator, bottle A, from an electric pump causing water to flow into a similar but empty desiccator, bottle B. This then displaced air at room temperature (20°C), which was forced into the trachea. The intratracheal pressure was measured by a continuous recording on a kymograph and mercury sphygmomanometer connected into the circuit. Air was injected in volumes of 250 ml measured by a scale on bottle B. After each injection, the kymograph tracing was
Fig. 2. Diagram of apparatus used for raising the intrapulmonary pressure.

Fig. 3. Diagrams of arrangement of many-tailed bandages and position of sandbags.

observed to ascertain whether there was a sudden or gradual pressure fall that would indicate pulmonary tissue breakdown. The intrapulmonary pressure was then reduced to atmospheric.

In 3 of the cadavers the chest and abdomen were bound with many-tailed bandages before experiment. In 2 cadavers the abdomen only was bound and in another 2, no binding was performed but two sandbags were placed on the abdomen to prevent downward displacement of abdominal organs, Fig. 3. Air in the pleural cavities was detected by making water traps in the lateral sides of the chest, before opening the thoracic cavity, using the technique described by Gresham and Turner (9).
Routine autopsy observations

In an attempt to assess the incidence of pulmonary lesions in normal males within the age group 19–40, the autopsy data on 97 sudden or unnatural deaths between 1951 and 1974 investigated at the Department of Forensic Medicine of St. George Hospital Medical School were examined. In this series both personally examined cases and those of other workers were included, but those with a known antemortem respiratory problem were excluded. The lesions were classified into broad groups of adhesions on lung surface and lesions within parenchyma, which are shown in Table 3. Such observations could be regarded as not detectable on routine clinical examination but being sufficient to predispose to barotrauma if the lung were put under stress.

RESULTS

The results are considered under the headings from which the data were derived.

Autopsy data from diving accidents

Thirteen cases formed this group in which the primary cause of death was the result of lung damage, shown in Tables 1 and 2. Four deaths showed the pneumothoraces, which can be caused as part of the spectrum of barotrauma. The remaining 9 cases died as a result of barotrauma.

Pneumothorax

In 3 of the 4 cases the site of rupture could be demonstrated convincingly (Table 1). Two showed rupture through lung parenchyma to visceral pleurae with adjacent hemorrhage on the lateral aspect of the left upper lobes. One case had identifiable rupture through a bulla at the apex of the left lung (Fig. 4). Adhesion obliterating the pleural cavity caused technical problems during removal of lungs in the remaining case, which did not allow definitive identification of a point of rupture.

Barotrauma

In 9 cases the immediate cause of death was due to barotrauma. Of these, 5 were in accidents at depths greater than 50 m, and were considered nonsurvivable on current physiological principles of treatment at the time. The remaining 4 were exposed to pressure at depths from 10 to 30 m. All the lungs were suitable for detailed examination by the thin section technique of Kleinerman and Cowdrey (7). Although considerable parenchymal damage could be seen (Fig. 5) no focus could be identified as the primary source of tearing or damage.

Case 9 had both clinical and histological (Fig. 6) evidence of acute bronchiolitis leading to air trapping and parenchymal rupture. There was no reliable evidence to support lung infection during the previous year which could be implicated in the causation of pleural adhesion. The damage to lung parenchyma was distributed in the parenchyma (Table 2), and 7 cases were not related to fixed structures but showed
<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Cause of Death</th>
<th>Left Lung</th>
<th>Right Lung</th>
</tr>
</thead>
<tbody>
<tr>
<td>39</td>
<td>Road traffic accident, head injury</td>
<td>Adhesions to Chest Wall: -  Adhesions in Fissures: -  Other: Focus of apical TB</td>
<td>Adhesions to Chest Wall: -  Adhesions in Fissures: -  Other: -</td>
</tr>
<tr>
<td>22</td>
<td>Acute alcoholic poisoning</td>
<td>-</td>
<td>Few  Apical blebs, no bronchitis</td>
</tr>
<tr>
<td>25</td>
<td>Drug overdose</td>
<td>Base  Focus of old apical TB</td>
<td>Base  -</td>
</tr>
<tr>
<td>31</td>
<td>Asphyxia</td>
<td>Upper lobe  Obliteration of pleural cavity: -  Scarring and tethering to base: -</td>
<td>Upper lobe  Lateral wall, upper lobe: -</td>
</tr>
<tr>
<td>36</td>
<td>Carbon monoxide poisoning</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>38</td>
<td>Aortic rupture</td>
<td>Scarring and tethering to base</td>
<td>Few  -</td>
</tr>
<tr>
<td>27</td>
<td>Head injury</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>28</td>
<td>Fracture of cervical spine</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>20</td>
<td>Hemorrhage into cerebral tumor</td>
<td>Few  Tethering apex, old TB</td>
<td>Few  -</td>
</tr>
<tr>
<td>40</td>
<td>Subarachnoid hemorrhage</td>
<td>Few  -</td>
<td>Few  Scarring lower lobe</td>
</tr>
<tr>
<td>24</td>
<td>Head injury</td>
<td>Few  Obliteration</td>
<td>Few  -</td>
</tr>
</tbody>
</table>

**TABLE 3**

**DISTRIBUTION OF LUNG LESION IN RANDOM NORMAL POPULATION**
evidence of interstitial emphysema. In cases 10 and 12, minute holes were found on the lung surface, related to small bands of adhesions between lung and pleural cavity at the bases.

Experimental

Examples of the tracings of pressure changes during inflation of the lungs are shown in Fig. 7. A similar pattern was present in all six tracings in that after each volume of air had been injected there was a slow fall in pressure for a few seconds until a constant level was reached, but as the intratracheal pressure was raised it took longer to settle down. If the drop in intratracheal pressure continued, it was assumed that injury had occurred in the lungs and no more air was injected. Dissection showed evidence of barotrauma in all 6 of the completed cases (Table 4).

Case 1: male age 27 (chest and abdomen bound)

The maximum intrapulmonary pressure was 210 mmHg and the total volume of air injected was 8.7 liters. There was suffusion of the neck and face, with interstitial emphysema over the upper thorax. No air was detected in the pleural cavities. The lungs were voluminous with scattered petechial hemorrhage on the exposed surfaces and bullae on the free borders. When lungs were examined under saline, bubbles of air escaped at points where there were adhesions between visceral and parietal pleura.
Fig. 5. Damaged area of lung due to barotrauma. Reticulin. x 125.

Case 2: male age 37 (chest and abdomen bound)

The maximum intrapulmonary pressure was 195 mmHg and the total volume of air injected was 9.2 liters. Air was not detected in the pleural cavities. There was conspicuous interstitial emphysema of the neck. Dissection of the thorax revealed a large volume of air trapped in the anterior mediastinum and adjacent connective tissue. Both lungs were voluminous, and the left lower lobe adhered to the lateral chest wall by fine adhesions.

Case 3: male 33 (chest and abdomen bound)

Rejected due to technical failure.

Case 4: female age 28 (abdomen bound)

The intrapulmonary pressure rose to 95 mmHg after 5.9 liters of air had been injected. Air was detected in the right pleural cavity. Dissection showed bullae on the margins of voluminous lungs. Submersion in saline showed numerous small leaks of air from fine adhesions on the left base and from bullae. In addition, there was diffuse interstitial emphysema.
Fig. 6. Bronchiole lumens obstructed by muco-pus. Hematoxylin and eosin ×140.

Fig. 7. Examples of intratracheal pressure tracings on cadavers.
## Table 4
Observations on Cadavers Subjected to Intrapulmonary Pressure

<table>
<thead>
<tr>
<th>Binding</th>
<th>Case No.</th>
<th>Age, yr</th>
<th>Pressure, mmHg</th>
<th>Volume, liters</th>
<th>Cause of Death</th>
<th>Observations of Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest or Abdomen</td>
<td>I</td>
<td>27</td>
<td>210</td>
<td>8.7</td>
<td>Drug overdose</td>
<td>Voluminous lungs, interstitial emphysema in neck, bulla on edges, no air leakage</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>37</td>
<td>195</td>
<td>9.2</td>
<td>Coronary artery disease</td>
<td>Voluminous lung, interstitial emphysema</td>
</tr>
<tr>
<td></td>
<td>III</td>
<td>33 Male</td>
<td>Technical failure</td>
<td></td>
<td>Head injury</td>
<td></td>
</tr>
<tr>
<td>Abdomen</td>
<td>IV</td>
<td>28 Female</td>
<td>95</td>
<td>6.1</td>
<td>Subarachnoid hemorrhage</td>
<td>Pneumothorax on right peripheral bullae with rupture, interstitial emphysema</td>
</tr>
<tr>
<td></td>
<td>V</td>
<td>39 Male</td>
<td>85</td>
<td>5.9</td>
<td>Drug overdose</td>
<td>Peripheral bullae on upper lobes, rupture at left base between parenchyma and adherions with pneumothorax</td>
</tr>
<tr>
<td>None</td>
<td>VI</td>
<td>25 Male</td>
<td>110</td>
<td>6.4</td>
<td>Head injury</td>
<td>Adhesions and leaks</td>
</tr>
<tr>
<td></td>
<td>VII</td>
<td>30 Male</td>
<td>80</td>
<td>5.7</td>
<td>Cerebral tumor</td>
<td>Rupture of lung at left base, bullae on edges of lungs</td>
</tr>
</tbody>
</table>
Case 5: male age 39 (abdomen bound)

The intrapulmonary pressure rose to 85 mmHg after the injection of 5.9 liters of air, followed by a rapid fall. A left pneumothorax was detected by a water trap. Dissection showed emphysema of the neck. The lungs were voluminous, and air was trapped in the connective tissue of the anterior mediastinum. Fine adhesions were present on the lateral side of the left lower lobe. Immersion in saline showed escape of gas from minute ruptures adjacent to the adhesions.

Case 6: male age 25 (no binding)

The intratracheal pressure rose to 110 mmHg after the injection of 6.4 liters of air. Water traps detected air in both pleural cavities. Dissection showed the lungs filled the pleural cavities with bullae on the free edges. Numerous fine adhesions were found on the diaphragmatic surfaces of both lower lobes. Immersion in saline showed escape of air from numerous small leaks on these surfaces.

Case 7: male age 30 (no binding)

The intratracheal pressure rose to 80 mmHg after the injection of 5.7 liters of air, and then fell rapidly. Air was detected in the left pleural cavity. Dissection showed air in the subcutaneous tissues of the neck. The lungs were voluminous and there were bullae on the free margin of the upper lobes. The mediastinal connective tissue showed the presence of trapped air. At the left costophrenic angle were a few fine adhesions between visceral and parietal pleurae. Immersion of the lungs under saline showed leakage of air from small holes adjacent to these adhesions.

Routine autopsy observations

The data in a consecutive series of 97 sudden deaths between the age of 19 and 40 showed that lung lesions were observed in 11 cases (Table 3). There is obviously considerable observer error in such a group. The two cases with apical tuberculosis (TB) could have been excluded by routine radiography, which leaves 9 otherwise healthy males who clinically would not show evidence of lung lesions but could be considered potentially at risk from lung trauma when exposed to a diving environment. Thus, if this is considered an average population, 10% can be considered to have some latent lesion.

However, additional control data were obtained from 29 autopsies of the series by Calder (5) in which the primary cause of death was not pulmonary damage. Of these, 19 were suitable for examination by the thin section technique, and the findings are shown in Table 5. Ages ranged from 19 to 41 yr (mean 26.4 yr). The detailed autopsies showed evidence of lung lesions in 7 cases (13, 15, 19, 22, 24, 27, 29), and all were minimal. In 2 cases (15 and 27) there was evidence of old healed tuberculous foci in upper lobes. Five cases showed evidence of adhesions between visceral and parietal pleura, which were very thin and ruptured easily during dissection. In 2 cases no evidence of recent medical examination was available, but the other 17 together with the 13 who suffered pneumothorax and barotrauma had been certified medically fit within a year before death. This therefore suggests that in a highly selected population
of divers subjected to critical autopsy examination there is a 26% incidence of clinically occult lung lesions.

**DISCUSSION**

The primary anatomical factor involved in lung rupture is pulmonary compliance, which may result in pneumothorax or parenchymal barotrauma. Compliance is the direct relationship between volume changes in lungs and transpleural pressure. Lung tissue over a large volume obeys the principle of Hooke's Law, which states that strain is related to stress by the bulk modulus. The reciprocal of such modulus is termed compliance, whenever transpulmonary pressure is the stress applied to the lung and strain is the resulting volume change of the tissue. Hooke's Law is no longer relevant when the elastic limit of the lung is reached and large changes in transpulmonary pressure result in little change in volume. When the stress applied to the lung reaches the terminal strength of the parenchyma, tearing occurs. The ultimate strength of the lung is related in a complex manner to the unidimensional tensile strength of lung tissue. Experimental work by Pierce (10) found this to be approximately 1.6 kg/mm² for pleural strips and 1.1 kg/mm² for parenchymal strips of dried human lung tissue. This would suggest on basic mechanical principles that lung parenchyma is more vulnerable to mechanical stress than that of pleura. However, the mechanism of arriving at a situation of barotrauma is a complex of stress compromising the mechanical strength of lung.

The sizes and shapes of lungs or chest walls have been suggested by Forgacs (11) and Withers et al. (12) as influencing the development of a spontaneous pneumothorax in young adults, and this has been substantiated by further clinical investigation by Peters et al. (13). These findings have been appraised in a retrospective study analyzing radiographic data by Goad et al. (14). Based on this data, it is postulated that long, narrow chests might expose increased stress on the apices of lungs, and the result of this, under the right conditions, can give rise to bullae by pulsion and traction. However, in this investigation no quantitative morbid anatomical data were obtained and radiographs of suitable technical quality were not available to give discrimination suitable for Hotelling's T² Test.

In 3 of the 4 cases in which the primary cause of death was due to pneumothorax, bullae were present on the surfaces, although not confined to the apices. This may be explained by the fact that this was major trauma due to more violent pressure changes. However, in one case (Fig. 1) there was evidence of preexisting bullae supported by the presence of connective tissue in the wall revealed by special stains (Masson trichrome). No adhesions were present on lung surfaces, and thus the bullae can be considered to be due to pulsion, most likely as the result of air trapping in the past.

The separation of barotrauma from pneumothorax poses a difficult differential diagnostic problem. In the main it is dependent on reasoned analysis of the clinicopathological data. Nevertheless, from the 9 cases in Table 2 the dominant observation was hemorrhage into the parenchyma, which may be associated with the formation of lung blebs.

Examination of these lungs, applying the technique similar to that of Gough and Wentworth (15) for thin, whole lung sections, allowed detailed examination of areas
of suspected damage, which were identified in the parenchyma by hemorrhage. In
these there was no morphological evidence to relate the damaged areas to fixed
structures (vessels and airways) or scar tissue in the case of healed primary tuber-
culos. The presence of the blebs associated with parenchymal damage from baro-
trauma must be considered a general reflection of the general pressure stresses
imposed.

Consideration of results obtained from experimental barotrauma, however, reflects
in a controlled way the findings in the series of autopsy cases from diving deaths,
and ages are comparable. Much of the observable damage was at the peripheries of
the lungs, in addition to which there was interstitial emphysema. A dominant factor
was the relationship of peripheral damage to lungs, to areas of pleural adhesions, and
to resultant pneumothorax. Again there was no definite relationship of parenchymal
damage to fixed structures or scar tissue.

Cases in which there was no binding brought about rupture at the site of the
adhesions at the base of the lung. As a result, these were stretched and produced
shearing forces resulting in rupture. When the abdomen was supported by the mul-
titailed bandage, the downward expansion of the lungs was restricted and thus stretch-
ing of basal adhesions was limited and insufficient to damage the visceral pleura. In
this situation however, the pressure is transmitted to the parenchyma with develop-
ment of interstitial emphysema. The development of interstitial emphysema, how-
ever, occurred at about the same pressure, both in the cases with abdominal binding
and without support. This was caused by the alveoli rupturing into the pulmonary
interstitial planes because the expansion of the chest was not limited by the tension
of intercostal and thoracic muscles. This observation is analogous to the experimental
finding in dogs by Joannides and Tsoulos (16) where it was found that in anesthetized
animals interstitial emphysema developed at inflation pressures ranging from 40 to
60 mmHg, whereas in those unanesthetized the pressure to cause barotrauma was
from 60 to 100 mmHg.

Binding of thorax and abdomen in the experimental cadavers allowed higher pres-
sures to be applied before damage become apparent. In this situation, neither pneu-
mothorax nor lung rupture occurred although adhesions were present. Damage was
confined to interstitial emphysema.

A significant observation is that damage was not related to fixed points such as
airways, vessels, or scar tissue within the parenchyma which could be considered as
anchoring points to cause stress. These findings would suggest that over-expansion
of lungs is primarily responsible for barotrauma, and when adhesions were present
mainly at the bases there was rupture of visceral pleura at lower pressures with less
distention, giving rise to pneumothorax. Therefore, if the chest and abdomen are
supported to prevent overstretching the risk of barotrauma is diminished. This is in
agreement with the experimental work of Malhotra and Wright (17) using unanesthet-
etized rabbits with abdominal binding, which showed the incidence of pulmonary
barotrauma to be reduced. This reflects that the living, fit male has a relatively strong
rib cage supported by muscle tone that protects the upper lobes of the lungs, while
the lower lobes require support from the diaphragm and this can be enhanced by the
use of an abdominal binder.

From the foregoing data the significant observation from autopsies and experiments
was the positive relationship of adhesions to pleural damage and the negative rela-
tionship of parenchymal barotrauma to fixed structure and scars. These lesions may
be difficult to identify clinically, and this fact is emphasized by finding some 26% in
apopulation of divers who had been subjected to rigorous medical examination. The
figure of 10% of lung lesions identified from routine autopsy observations may be
lower due to the observer not being conscious of the significance of such lesions.

Nevertheless it must be considered that the presence of such lesions may, in a
situation of decompression, be sufficient to compromise the lung and cause significant
trauma. This is emphasized by the results of the experimental work. Conversely
many of the 19 cases examined must have been subject to lung stress at some time
during their diving careers, without obvious clinical lung damage. This is supported
by the fact that in 7 (36%) of these cases lesions were present but had not caused
lung trauma. It is not possible based on available data to determine whether such
lesions result from previous barotrauma or infection, but none of these cases had
clinical records of lung damage.

The factors thus involved in causing lung damage must be more than the simple
inter-relationship of pressure and compliance to scar tissue, and the effects must be
regarded at present as unpredictable.

CONCLUSION

Consideration of the morbid anatomical data shows significant findings which could
not have been identified in the course of routine medical examination, and may be
summarized:

1. Pneumothorax is essentially related to adhesions and pleural damage. Never-
theless a significant number of cases were examined where adhesions were
present but did not cause visceral pleural damage.
2. Scar tissue and fixed structures in the lung parenchyma were not consistently
related to areas of barotrauma.

From the foregoing it may be reasoned that lung trauma in some cases is a chance
incident, with lung parenchyma rupturing under minimal stress. The prime agent is
a pressure differential on an already compromised lung parenchyma. This is an aspect
that cannot readily be identified on routine diving medical examinations.

However, on the data obtained from autopsies and experiments it is apparent that
parenchymal barotrauma is not constantly related to fixed structures such as major
blood vessels and scar tissue. This would lend support to a view that an incident of
pulmonary barotrauma once it has undergone healing does not necessarily predispose
to further damage and should not preclude further diving activities per se.

The evidence relating to pneumothorax is somewhat contentious in that from
observations on the available data, multiple bullae develop. However, the rupture of
one produces a pneumothorax, which when healed leaves a residuum of bullae on the
lung surface. Nevertheless, from the overall examination of many lungs the presence
of bullae is quite common and the cause of no clinical problems. Some of these bullae
as seen in Fig. 1 have walls that are thick and possess relatively high tensile strength.
A healed rupture of a bulla could also be considered to be scar tissue and be relatively
more resistant to further trauma. These multiple bullae if they can be identified remain
to compromise the lung should further insults of pressure differentials occur. This
must therefore remain a contraindication to further diving. It cannot be reasonably
argued that the weakest part of the lung has ruptured and therefore after healing the
### TABLE 5
**Distribution of Incidental Lung Findings by Diving Population Killed by Other Than Pulmonary Damage**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, yr</th>
<th>Cause of Death</th>
<th>Observation on Lungs</th>
<th>Time Elapsed Between Last Medical and Death, months</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>33</td>
<td>Hypothermia</td>
<td>2 cm focus of emphysema adjacent to hilum</td>
<td>8</td>
</tr>
<tr>
<td>14</td>
<td>27</td>
<td>Acute anoxia</td>
<td>NIL</td>
<td>9</td>
</tr>
<tr>
<td>15</td>
<td>41</td>
<td>Coronary artery occlusion</td>
<td>TB focus in left upper lobe, no adhesions</td>
<td>3</td>
</tr>
<tr>
<td>16</td>
<td>23</td>
<td>Asphyxia due to drowning</td>
<td>NIL</td>
<td>11</td>
</tr>
<tr>
<td>17</td>
<td>26</td>
<td>Asphyxia due to drowning</td>
<td>NIL</td>
<td>No medical traced</td>
</tr>
<tr>
<td>18</td>
<td>19</td>
<td>Acute anoxia</td>
<td>NIL</td>
<td>4</td>
</tr>
<tr>
<td>19</td>
<td>24</td>
<td>Epileptic fit and drowning</td>
<td>Fine adhesions on left upper lobe</td>
<td>10</td>
</tr>
<tr>
<td>20</td>
<td>28</td>
<td>Acute anoxia</td>
<td>NIL</td>
<td>4</td>
</tr>
<tr>
<td>21</td>
<td>23</td>
<td>Cardiomyopathy</td>
<td>NIL</td>
<td>3</td>
</tr>
<tr>
<td>22</td>
<td>21</td>
<td>Drowning after nitrogen narcosis</td>
<td>Fine adhesions between right lower lobe and parietal pleura</td>
<td>8</td>
</tr>
<tr>
<td>23</td>
<td>29</td>
<td>Hyperthermia</td>
<td>NIL</td>
<td>9</td>
</tr>
<tr>
<td>24</td>
<td>31</td>
<td>Asphyxia due to drowning</td>
<td>Scenting adhesions between right middle lobe and parietal pleura</td>
<td>4</td>
</tr>
<tr>
<td>25</td>
<td>25</td>
<td>Hypothermia</td>
<td>NIL</td>
<td>6</td>
</tr>
<tr>
<td>26</td>
<td>24</td>
<td>Asphyxia due to drowning</td>
<td>NIL</td>
<td>5</td>
</tr>
<tr>
<td>27</td>
<td>37</td>
<td>Asphyxia due to drowning</td>
<td>TB focus in left upper lobe, no adhesions</td>
<td>11</td>
</tr>
<tr>
<td>28</td>
<td>22</td>
<td>Hyperthermia</td>
<td>NIL</td>
<td>2</td>
</tr>
<tr>
<td>29</td>
<td>35</td>
<td>Hyperthermia</td>
<td>Fine adhesions between visceral and parietal pleura, left lower lobe</td>
<td>No medical received</td>
</tr>
<tr>
<td>30</td>
<td>20</td>
<td>Undetermined</td>
<td>NIL</td>
<td>4</td>
</tr>
<tr>
<td>31</td>
<td>25</td>
<td>Asphyxia due to drowning</td>
<td>NIL</td>
<td>8</td>
</tr>
</tbody>
</table>
 overall resistance of the lung to further damage must be increased because the weakest part has become scarred and nonfunctional.

The salient fact remains that there is both within the medically screened divers and the population in general a high incidence of occult minor lung abnormalities (up to 26%). It is difficult to evaluate the significance of these for the purposes of operational diving activities. However, the fact remains that scarring in parenchyma and fibrous bands between lung and pleura have not, in these cases, shown in Table 5, caused clinical problems although many must undoubtedly have suffered pulmonary stress during the diving career.

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Calder IM. Autopsie et observations expérimentales sur les facteurs conduisant au barotraumatisme chez l'homme. Undersea Biomed Res 1985; 12(2):165-181.—Les données anatomiques morbides de 13 accidents mortels de plongée, dans lesquels les dommages pulmonaires étaient la cause primaire de la mort, ont été étudiées en détail. De plus, les observations sont faites sur le cas de barotraumatisme expérimental chez six cadavres. Cette information est supplémentée par des découvertes fortuites faites dans le système respiratoire de 19 plongeurs professionnels tués par des causes autres que le barotraumatisme, et 97 témoins en bonne santé et d'âge correspondant qui décédèrent soudainement ou de manière anormale. Dans les cas de pneumothorax résultant tous les deux de la plongée et du test expérimental, la caractéristique constante fut la présence d'adhérences pleurales et bulles pulmonaires dont quelques-unes causé- rent des dommages tissulaires. Des adhérences pleurales furent observées aussi dans une proportion élevée (jusqu'à 26%) des poumons n'ayant aucun rapport au barotraumatisme. Dans les cas de barotraumatisme, le dommage parenchymal n'est pas constamment relié aux cicatrices ou tissu conjonctif. Ces observations suggèrent qu'il existe une incidence élevée de lésions pleurales qui ne sont pas détectées ou décelables lors d'un examen médical systématique et qui ne compromettent pas le poumon. Cependant, une fois qu'un pneumothorax est produit, il est fort probable qu'un autre incident pourrait faire éclater une autre bulle et celles-ci paraissent être multiples. Le contraire semble être vrai pour le barotraumatisme parenchymal qui n'est pas constamment lié au tissu cicatriciel. Ceci tend donc à suggérer qu'un cas de barotraumatisme confirmé et bien guéri ne devrait pas nécessairement constituer une interdiction participer à des activités ultérieures de plongée.

barotraumatisme accidents de plongée pneumothorax poumons expériences

REFERENCES