To the Editor:

Dr. Carpenter (Undersea & Hyperbaric Medicine, Vol. 24 No. 3, 1997) described an interesting case of recurrent pulmonary barotrauma (PBT) in a previously healthy male scuba diver who suffered from repeated pneumomediastinum after shallow-water scuba dives. In the interval between the incidents, the diver performed four dry hyperbaric chamber dives without complications. Having reviewed the pertinent literature, Carpenter concludes that hyperbaric exposures in dry chambers rarely lead to PBT when compared to open-water diving and thus risk factors for PBT may be unique to the underwater environment.

We are aware of more cases (MEDLINE) than those cited of documented PBT in subjects performing hyperbaric chamber dives. Radermacher et al. (1) described a case of severe cerebral arterial gas embolism (AGE) following a 50-msw dry chamber dive. Collins (2) presented a case of possibly recurrent AGE, the initial incident appearing after a dry chamber dive to 112 ft. The latest case of PBT occurring at our institute in October 1997 was a severe AGE after a 50-msw dry chamber dive in an asymptomatic male sport diver being certified fit for diving. Previously unknown pulmonary sarcoidosis could be diagnosed as the possible precipitating mechanism in this diver, who had performed more than 500 scuba dives and never experienced decompression illness before. We know of more clinical incidents that support PBT. Commonly, immediate recompression therapy is performed in such cases with mostly spontaneous recovery of symptoms; this may be the reason so few cases are published.

Second, in the case presented, a lack of functional or anatomic pathology is stated. However, we are not given detailed information on lung function except a normal methacholine challenge. Thus, possible risk factors like small airways disease (3) or exercise-induced asthma (which may be present without a significant response to clinical bronchoconstrictors) are not excluded. Childhood asthma and a family history of atopy should focus attention on sophisticated lung function assessment. Moreover, it is stated that high resolution computed tomography (HR-CT) revealed no bullae. Since HR-CT normally is not performed using a continuous table feed rate producing contiguous slices as Spiral-CT does, bullae cannot be excluded (4,5).

In most cases of PBT reported in the literature, it is obvious that risk factors such as airway obstruction or lung bullae may emerge in clinical investigations, but subjects often have performed uncomplicated dives before diagnosis without being injured. It may be assumed that a pathological lung anatomy or airway obstruction may increase the risk of PBT, but other factors (e.g., procedural) must exist to induce lung injury. In conclusion, one may agree with Dr. Carpenter that occurrence and possibly recurrence of PBT should focus our attention to elicit presently unknown risk factors for PBT. However, lung anatomy and respiratory function should be carefully investigated using appropriate methods to evaluate such factors.

K TETZLAFF, M.D.
Naval Medical Institute of the
Federal German Navy
Kronshagen, Germany
M. REUTER, M.D.
University Department of
Diagnostic Radiology
Kiel, Germany

REFERENCES

The author responds:

Doctors Tetzlaff and Reuter make several worthwhile observations regarding recurrent pulmonary barotrauma (PBT). The additional dry chamber PBT cases they reference fell outside the range of my MEDLINE search parameters. The third dive in Collins’ case report (1) is consistent with dry chamber PBT, but the first two dives have atypical histories for PBT. In addition, the cases noted by Radermacher et al. (2), Tetzlaff and Reuter have dive profiles more consistent with decompression sickness than with
Table 1: Pulmonary Function Tests

<table>
<thead>
<tr>
<th></th>
<th>20 Sep '94 Dive Screen, % Predicted</th>
<th>19 Apr '96 Baseline, % Predicted</th>
<th>19 Apr '96 Methacholine, % Predicted</th>
<th>19 Apr '96 Reversal, % Predicted</th>
</tr>
</thead>
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<tr>
<td>FVC</td>
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<td>102</td>
<td>101</td>
<td>102</td>
</tr>
<tr>
<td>FEV&lt;sub&gt;1.0&lt;/sub&gt;</td>
<td>105</td>
<td>106</td>
<td>100</td>
<td>102</td>
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<tr>
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<td>122</td>
<td>119</td>
<td>95</td>
<td>103</td>
</tr>
<tr>
<td>FEF&lt;sub&gt;50&lt;/sub&gt;</td>
<td>120</td>
<td>124</td>
<td>98</td>
<td>105</td>
</tr>
</tbody>
</table>

PBT. No literature could be found suggesting unreported dry chamber PBT or immediate therapy within the chamber to induce spontaneous recovery. In contrast, Cianci conducted an informal telephone survey of hyperbaric specialists in which he concluded the incidence of dry chamber PBT at 1:50,000 (3).

The pulmonary function tests omitted from the original case report are included in Table 1. Although these tests are relatively insensitive for detecting certain subsets of small airway obstruction, they remained the gold standard for the diagnosis of asthma and other small airways disease (4). An exercise challenge test, however, would be a worthwhile study in this case to better evaluate for exercise-induced asthma (EIA) (5). Interestingly, if present, EIA would seem to have been exacerbated more in the underwater environment than in the dry chamber because each dive (dry and scuba) was preceded by 1 h of intense cardiovascular exercise, yet the recognized PBT occurred during two of six scuba dives rather than in any of the nine dry chamber dives. As for the history of infantile wheezing, the pediatric literature suggests an overdiagnosis of wheezing as asthma and the patient had noted no manifestations of asthma through an active childhood and adult athletic life (6,7).

Finally, Drs. Tetzlaff and Reuter make the observation that pathological anatomy or physiology alone may not be sufficient to induce symptomatic pulmonary barotrauma, but that additional factors must act concurrently. The margin for error in scuba is greater than in hyperbaric chamber dives, as I have noted. Whether this would explain the higher incidence of PBT in scuba remains to be shown, but several physiologic attributes may be unique to the underwater environment and should be the focus of future research. Additionally, the use of promising new imaging modalities should be researched in cases of underwater and dry chamber PBT to identify otherwise unrecognized pathology. Hyperbaric professionals should be encouraged to report all cases of PBT, treated or not, to obtain more precise estimates of dry chamber PBT and populations of divers susceptible to scuba PBT, but not to dry chamber PBT.

C. R. CARPENTER, M.D.
Marine Aviation Logistics Squadron - 13
Marine Air Group - 13
Box 99190
Yuma, Arizona 85369

Address for Dr. Carpenter after 1 June 1998: Allegheny General Hospital, Department of Emergency Medicine, Pittsburgh, PA.

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Unusual Causes of Convulsions in a Hyperbaric Chamber

To the Editor

We describe two cases of convulsions occurring under hyperbaric conditions which have been reported previously. *Case 1* is a 36-yr-old woman who was an experienced diver doing a commercial diving course. On Day 1 she did a 50-m chamber dive and decompressed following DCIEM tables, without incident. On Day 2 she did two 7-m Hookah dives. Bottom time was 30 min on both occasions, with a 3-h surface interval. The next day she developed a headache,
fatigue, poor concentration, and aching in both knees and both elbows with fleeting pain in her knuckles. Past history included chronic back pain and mosquito-borne viral illness resulting in joint pains. She had no history of epilepsy or head injury, and she was on no medication.

On examination she was afebrile with no neck stiffness or papilledema. Neurologic examination was normal, sharpened Romberg's was 60 s, and the remainder of the examination was unremarkable.

The differential diagnosis was viral illness or decompression illness, and it was decided that a trial of pressure was indicated. Recompression was commenced using a modified Royal Navy 62 table. No oxygen had been administered before recompression. Sixteen minutes into the first O₂ period at 2.8 atm abs she had a grand mal convulsion preceded by an auditory aura. O₂ was ceased and the convulsion terminated spontaneously with a total duration of less than 1 min. As the convulsion occurred so early in the treatment, intravenous diazepam was administered prophylactically to a total dose of 6 mg. O₂ was recommended 25 min after the convulsion. Fifteen minutes into the third O₂ period, the auditory stimuli reoccurred, so the O₂ was again stopped. Despite this, she had another grand mal convulsion lasting less than 1 min. She was decompressed to 2.3 atm abs and the table completed without further incident.

On review at completion of treatment her original symptoms were unchanged. Further neurologic investigation was undertaken because the convulsions were so unexpected. A computerized tomography scan of the brain with contrast was normal and a neurology review was requested. An electroencephalogram was abnormal with generalized epilepsy.

Case 2 was a 28-yr-old woman who was compressed to 2.0 atm abs for treatment of a traumatic compartment syndrome of her right arm. After 7 min at 2 atm abs she suffered a grand mal convulsion. Oxygen was stopped and the convulsion resolved spontaneously in less than 1 min. Subsequent investigation revealed that the patient had been administered 1,300 mg of pethidine over a 28-h period. Although not well reported, the proconvulsive effects of pethidine's major metabolite (norpethidine) are well known. Although pethidine has a short half life, norpethidine has a half life of 14–21 h and up to 35 h in patients with renal failure (1).

In case 1, hyperbaric oxygen (HBO₂) unmasked latent epilepsy. The patient is at high risk for further convulsions particularly with increased partial pressures of oxygen during diving. She has therefore been advised not to dive again. In patients who have unusual convulsions under hyperbaric conditions, alternative diagnoses should be excluded.

In case 2, HBO₂ precipitated convulsions caused by norpethidine. Previously described risk factors for the development of pethidine-related convulsions include renal failure, high doses of pethidine, and co-administration of hepatic enzyme-inducing medications or phenothiazines (2). HBO₂ should now be added to this list.

G. M. Emerson, MBChB, Senior Registrar
H. F. Oxer, MA, MB, B.Chir, Director
Fremantle Hospital Hyperbaric Medicine Unit
Western Australia 6000

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