

## Treatment Experiences of Pulmonary Barotrauma with a Fatal Case Report

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### Abstract

Pulmonary barotrauma (PBT) is caused by overinflation of the lungs and the intrapulmonary gas enters into the surrounding tissues and blood vessels leading to mediastinal and subcutaneous emphysema, pneumothorax and Cerebral Arterial Gas Embolism (CAGE). Due to its specific prerequisite conditions, PBT is rare in comparison with other traumas and most physicians are inexperienced in its treatment. In this report, a fatal case of PBT complicated with CAGE and pneumothorax was presented and some experiences in treatment were introduced.

**Keywords:** Pulmonary barotrauma; Cerebral arterial gas embolism; Pneumothorax; Recompression therapy

### Introduction

Pulmonary barotrauma (PBT) results from overexpansion of lung alveoli during a fast reduction of ambient pressure. In diving, PBT is mostly caused by uncontrollable rapid ascent ("blow up") or voluntary emergency ascent due to accidental incidents (such as interrupted air supply). If the intra-pulmonary gas be exhaled insufficiently, lung over inflation and rupture of the alveoli occur. Localized pulmonary obstructions that can cause air trapping, such as asthma or cysts, are other causes [1]. The free gas may enter the surrounding tissues leading to mediastinal emphysema, subcutaneous emphysema in the neck, or pneumothorax. The gas may also enter into arterial circulation to cause Cerebral Arterial Gas Embolism (CAGE) [2-4]. CAGE and severe pneumothorax are both life threatening.

Due to its specific prerequisite conditions, PBT is rare in comparison with other traumas and most physicians are inexperienced in its treatment [5]. In this report, a fatal case of PBT complicated by CAGE and pneumothorax was presented and the experiences of treatment were discussed.

### Case Report

A 30-year-old male diver with eight years of diving experience performed a surface supplied dive to 35 msw (meter sea water). The dive was aborted at about 10 min underwater with a rapid ascent due to an interrupted gas supply caused by a rupture of the air hose. Immediately after surfacing, chest pain exacerbated by coughing, unconsciousness and paralysis were present. The diver was transported to our unit one hour later. On examination, he was mentally clear with facial pallor, tachypnea and tachycardia. His blood pressure was 70/40 mmHg. Tenderness was found on the chest but without obvious rales. Symptomatic therapy was performed with fluid and drugs including antitussives, glucocorticoids, hemostatics and antibiotics. When vital signs stabilized, recompression was administered with the No. 4 schedule developed in our department, which lasts for 31 h 10 min (Table 1) [6]. Symptoms were markedly improved during the recompression, and the patient could sit and stand when the pressure reached 50 msw. He felt 'completely recovered' at 70 msw, and was required to lie down and reduce activity. According to the decompression schedule, the pressure was successfully reduced to 12 msw, where the patient complained of dyspnea and chest pain. The chamber was recompressed to 18 msw and the symptoms resolved. Oxygen was additionally administered for two cycles (20 min of oxygen and 5 min of air) at 18 msw and decompression was resumed with more oxygen breathing. Dyspnea recurred on arrival at 10 msw, and subcutaneous emphysema was found around the neck

and upper chest. Tympany to percussion was noted on the patient's left chest. Thoracentesis was performed to remove the air from the pleural space. Dyspnea was improved but consciousness blurred progressively. The chamber was pressurized again but failed to effect the patient's deterioration. Death was confirmed five minutes later.

### Discussion

This case was diagnosed as CAGE secondary to PBT. The early phase of treatment was successful, but CAGE recurred and pneumothorax was developed during the decompression, which finally resulted in sudden death. In our previously published report [6], PBT accounted for 54.5% (18/33) of all fatalities, of whom 5 died of rapidly progressed pneumothorax or mediastinal emphysema and 13 died of CAGE. As mentioned in the report, there were nearly 300 deaths before hospitalization during the past 16 years [6]. Among which, approximately half died of PBT. Due to lack of the proper breathing technique during the rapid ascending for the victims, the pulmonary wounds were usually severe, and the treatments were extremely difficult.

This was one of the early PBT cases treated at our hyperbaric unit. With the increase in the number of cases treated, we gained some experience and also learned lessons. The rate of successful treatment increased with the team's experience.

Usually, the mediastinal and subcutaneous emphysema secondary to PBT need no recompression unless the symptoms are severe, but are evidences for the possible existence of pneumothorax and CAGE. Mild, simple pneumothorax also requires no specific treatment. However, for severe or tension pneumothorax, gas aspiration should be performed. For CAGE, recompression should be administered immediately [2-4]. Favorable therapeutic efficacy can be obtained as long as no gas enters through the ruptures during the treatment. If the ruptures are severe, or forced breathing or excessive activities expand the pre-existing wound or create additional wounds nearby, gases may re-enter during the

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No	Depth (m)	Bottom time (min)	Decompression stops (m) and the stop times (min)																	Treatment time (min)					
			54	51	48	45	42	39	36	33	30	27	24	21	18	16	14	12	10		8	6	4	2	
1	30	20												3	10-20	10-20	10-20	10-20	10-20	20-30	30-60	30-90	60-90	233-413	
2	40	30										2	5	5	10	20-30	20-30	20-30	20-30	20-30	30-90	30-90	30-90	60-90	323-583
3	50	30						1	4	7	7	13	13	14	20-30	20-40	20-40	40-60	40-60	60-120	90-120	90-120	90-120	590-840	
4	70	30	3	3	3	3	5	5	10	15	20	25	40	60	70	110	160	180	190	210	220	240	270	1870	

Note: Descent rate: 10-20 m/min; Ascent rate from bottom to first stop: 2 m/min; Travel time between stops: 1 min. Oxygen breathing begins on arrival at 18 m for 20-30 min periods separated by 5-10 min air breaks. See in Discussion for detail. SMMU: The Second Military Medical University in China.

**Table 1:** Recompression schedules developed in SMMU.

decompression period of the recompression treatment. This is a key challenge in the treatment of cases of CAGE caused by PBT.

Generally, the treatment of CAGE due to PBT is considered the same as severe DCS, and differentiation between them is not necessary in order not to delay the recompression [5]. However, we believe the diagnosis of PBT is critical for the special considerations during the treatment. Most of the PBT cases are difficult to be discerned merely depending on clinical manifestations. CAGE should be suspected if a diver has a new onset of altered consciousness, confusion, aberrant mood, focal cortical signs, seizure or other neurological abnormalities during ascent or within a few minutes after surfacing. If a symptomatic diver performed “blow-up”, rapid ascent or breath-holding during ascending, CAGE is highly suspected. Subcutaneous emphysema around the neck warrants prompt suspicion of the coexistence of CAGE or pneumothorax. Despite an atypical history or manifestation, PBT cannot be excluded once there is possibility, especially for DCS divers coexisted with PBT [7].

Symptoms of CAGE usually respond well to recompression (similar to that of brain DCS), and it is curable provided that no recurrence occurs during the decompression [2,3,8]. However, pneumothorax and/or CAGE may recur during decompression (usually at the pressure shallower than 14 m). If treated inappropriately, the outcome could be fatal. We propose the following recommendations in the treatment of CAGE caused by PBT:

(1) Patients should be placed on high-flow rate pure oxygen promptly, and fluid supplementation, glucocorticoids and dehydrants (where necessary) should be administered to mitigate brain injury.

(2) Recompression should be commenced as soon as possible after necessary preparations. Of note, a percentage of CAGE patients would undergo a spontaneous clinical recovery, but the improvement was not always sustained [2]. Thus, patients who experience spontaneous resolution following presumed CAGE should also be promptly recompressed. For patients with severe pulmonary injuries, oxygen breathing at 18 m should be first administered and then continue the recompression schedule adopted. If higher pressure must be applied, moderate treatment schedules, such as our Schedule 1, 2, or 3 are recommended with prolonged oxygen breathing where necessary (Table 1) [6]. Schedules with higher pressure (more than 50 m) are not recommended because the treatment time is significantly prolonged and nitrogen narcosis may developed, which may pose additional difficulties during the treatment and increase the risk of recurrence [4,9].

(3) The pulmonary injury should be treated aggressively using drugs including hemostatics, antitussives, analgesics, spasmolytics and antibiotics [3].

(4) The patients should be required to breath gently and smoothly especially during the decompression phase to avoid significant

fluctuation of intrapulmonary pressure. The supply of oxygen from the mask should be adjusted to minimize the respiratory resistance.

(5) The decompression rates should be slow, limited even to 0.1-0.3 m/min.

(6) Sensible precautions and regular evaluation of the patient’s condition should be taken during decompression. If tension pneumothorax or CAGE is suspected, recompress to depth of relief. The recompression profile is based on CAGE, but before decompression, a chest tube must be inserted.

It is critical to commence emergency recompression for CAGE. However, gas may re-enter through the rupture into pleural cavity and/or arterial circulation during decompression, especially in patients with severe lung injury. Improper treatment may be fatal. The development of efficacious therapeutic strategies for this condition is warranted in future clinical studies.

**Consent**

The informed consent was obtained from the victim’s relative (wife) only. The Ethic Committee of the Second Military Medical University approved to report the case.

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