Pneumothorax as a complication of recompression therapy for cerebral arterial gas embolism

J. R. BROOME and D. J. SMITH

Undersea Medicine Division, Institute of Naval Medicine, Alverstoke, Hampshire, UK

Broome JR, Smith DJ. Pneumothorax as a complication of recompression therapy for cerebral arterial gas embolism. Undersea Biomed Res 1992; 19(6):447–455—The danger from pneumothorax in patients who undergo compression chamber treatment for cerebral arterial gas embolism (CAGE) following pulmonary barotrauma is frequently emphasized. Two cases of CAGE treated by recompression after submarine escape tank training (SETT) accidents are described. Both were complicated by bilateral pneumothoraces but the first case, treated on an air table, required thoracentesis in the chamber, whereas the second case, treated on an oxygen table, escaped the need for in-chamber thoracentesis despite large pneumothoraces. Review of similar Royal Navy and United States Navy SETT accidents suggests that the danger from pneumothorax during recompression treatment of CAGE victims may be overstated. Modern management on oxygen-based therapeutic recompression tables may significantly reduce the risk. Thoracentesis while under pressure should be reserved for cases developing symptoms or signs of tension pneumothorax. Treatment options for these cases are discussed and a decision algorithm is proposed.

pneumothorax  submarine escape
decompression  diving
cerebral arterial gas embolism  recompression therapy

Pulmonary overinflation, occurring in divers or submarine escape trainees, may result in potentially fatal cerebral arterial gas embolism (CAGE). Immediate recompression treatment offers the best chance of avoiding death or disability (1). Pneumothorax is another sequel to pulmonary overinflation and may coexist with CAGE. When detected during compression chamber treatment for CAGE, traditional teaching advocates prompt thoracentesis because of the potential for intrapleural gas, expanding during decompression, to emulate tension pneumothorax (2–4).

Recognizing that formal surgical thoracentesis in the confines of a compression chamber may be technically difficult and carries a potentially high risk of infection, a conservative approach has been proposed which relies on the theoretical efficacy of oxygen under pressure to aid the reabsorption of intrapleural air (5, 6). We describe two cases of bilateral pneumothorax accompanying life-threatening CAGE after submarine escape training tank accidents. These cases illustrate the contrasting results
obtained by compression treatment on the older air-based therapeutic tables compared with modern oxygen-based tables. Subsequent analysis of similar accidents suggests that pneumothorax need not cause major modifications to therapeutic decompression profiles, particularly if cases are treated on oxygen-based tables, and formal surgical thoracentesis in a chamber environment is largely avoidable.

CASE REPORTS

Each case involved a prospective submariner undergoing training at the Royal Naval Submarine Escape Training Tank (SETT) in Gosport. Training is performed in a tower of water 30 m high where each trainee passes through pressurized locks to enter the water column at different depths. The trainees then make buoyant ascents through the water to the surface to simulate escaping from a sunken submarine. All initial SETT trainees are subject to stringent medical examination and screening, including spirometry and inspiratory and expiratory chest x-rays, to exclude detectable pulmonary disease. (The units of depth referred to in both reports are those used in the historical record with alternatives in brackets. [1 fsw = 3.063 kPa; 1 msw = 10.000 kPa].)

Case 1

In early 1958, a trainee was making his first ascent from 100 ft (30 m). On reaching the surface, he was seen to be distressed and coughing and while being removed from the water he lost consciousness. He was immediately placed in the tank top recompression chamber and compressed to 165 feet of seawater (fsw) pressure (50 m). During the compression he was noted to be limp, apneic, and cyanosed. Artificial respiration was administered.

Within 1 min of reaching 165 fsw (50 m), the patient had recommenced spontaneous breathing and regained consciousness. He coughed up small quantities of frothy, bright red sputum, his respiratory rate was 28 breaths/min, and his color had reportedly improved. No abnormal signs were detected on chest examination, although there was pronounced tenderness with guarding in the epigastrium and over both subcostal regions. Examination of all other systems was reported as normal, and after 28 min at 165 fsw (50 m) it was decided to decompress on RN therapeutic table III (an 18-h air table) (7). At depths of 140 fsw (42 m) and 100 fsw (30 m) during the subsequent decompression, the patient complained of upper abdominal pain and vomited small amounts of altered blood mixed with food, but his general condition improved. Specifically, no chest signs were detected at these depths.

At 60 fsw (18 m), signs of a right pneumothorax were detected with diminished expansion, hyperresonance with absence of liver dullness, and reduced breath sounds on the right. A small area of bronchial breathing was audible at the right costal margin. As neither the trachea nor apex beat were displaced and the patient was not distressed, treatment was continued. However, on decompression to 30 fsw (9 m), the patient’s condition markedly deteriorated with increased pain and pronounced respiratory embarrassment. Recompression to 40 fsw (12 m) produced rapid relief of symptoms. After some delay, full surgical thoracentesis was performed through the second right intercostal space, anteriorly.
MANAGEMENT OF PNEUMOTHORAX DURING RECOMPRESSION

Therapeutic table III was abandoned in favor of a less prolonged schedule, and decompression was recommenced. At 10 ft (3 m) the patient's condition again deteriorated with signs of a left pneumothorax, accompanied by a rise in pulse rate to 130 beats/min, but after half an hour the symptoms had stabilized and the chamber was surfaced. After transport to a hospital, chest radiography confirmed the additional presence of a left pneumothorax which was subsequently drained. The therapeutic air table treatment had lasted 5 h 47 min.

Case 2

In late 1991, a SETT trainee had made two uneventful ascents from 9 m (30 ft) and was making his first ascent from 18 m (60 ft). The ascent seemed to proceed normally but on reaching the surface the trainee emitted an expiratory gasp, became unconscious, and convulsed as he was pulled from the water.

Mask ventilation with oxygen was commenced while, apneic and cyanosed, he was placed in the adjacent recompression chamber with a physician in attendance. The chamber was pressurized to 50 meters of seawater (msw) (165 ft) depth equivalent and within 4 min the patient had regained consciousness and was breathing spontaneously. Neurologic examination remained normal although a further convolution occurred later in the treatment. Subsequent decompression of the patient took over 5 h on RN therapeutic table 63 (USN 6A) (8). While at 50 msw (165 ft) pressure the patient breathed a mixture of 67.5% nitrogen:32.5% oxygen. At 18 msw (60 ft) and shallower, 100% oxygen was breathed with periodic short air breaks.

On examination at 50 msw (165 ft), the patient's respiratory rate was about 28 breaths/min with a prominent abdominal component to his breathing. He complained of retrosternal discomfort on inspiration but he denied subjective respiratory distress. Subcutaneous emphysema was palpable in his neck and supraclavicular fossae. On chest examination the trachea was central and the apex beat palpable. Expansion was symmetrical though apparently reduced by inspiratory discomfort. Percussion was equally resonant bilaterally, and breath sounds, although faint, were symmetrical and vesicular.

Pneumothorax was suspected clinically because of the faint breath sounds and abdominal breathing, although lateralization was difficult. The attending physician, mindful of the risks, documented frequent, careful observations of the patient's condition, especially during the decompression phases of the treatment from 50 to 18 msw (165–60 fsw), 18 to 9 msw (60–30 fsw), and 9 msw (30 fsw) to the surface, but no signs of developing cardiovascular or respiratory embarrassment were observed.

On completion of the treatment, the patient walked from the chamber apparently undistressed. Previously noted chest signs persisted and, additionally, a small area of bronchial breathing was distinctly audible at the right lung base, posteriorly. On admission to a hospital, the chest x-ray revealed bilateral pneumothoraces estimated (in the posteroanterior plane) at 70% on the right and 30% on the left. Gross mediastinal and subcutaneous emphysema was evident with tracking into retroperitoneal tissues. Free gas was also present under the diaphragm.

The right pneumothorax was treated by formal thoracentesis with underwater drainage, while the smaller, left-sided pneumothorax was aspirated. The mediastinal and retroperitoneal gas resolved with intermittent normobaric oxygen treatment. Within 4 wk all symptoms and signs had resolved and the patient returned to duty.
Review of pneumothorax cases in SETT incidents

The case records and reported experience for both the Royal Naval and United States Naval SETT accidents were reviewed. The case notes of all 187 RN SETT training incidents recorded since 1954, when training began, were scrutinized. Of these, 126 cases were treated by recompression for a presumptive diagnosis of CAGE. Additionally, a total of 113 incidents treated by recompression for possible CAGE were identified from USN sources for the period 1928–1977. The USN information was collated from available case records, two published reviews, (9, 10), and a computer search of the U.S. Naval Safety Center database.

Fifteen cases (10 RN and 5 USN) treated by recompression for CAGE had associated pneumothorax (6 bilateral). Of the 15 cases, 12 were present during air-only therapeutic tables and 3 were treated on oxygen treatment tables. In 4 further cases (all treated on air tables), signs such as change in percussion note or reduced breath sounds, associated with ipsilateral chest discomfort, led the attending physician to the confident clinical diagnosis of pneumothorax while in the chamber, but subsequent hospital chest radiography was normal. Of the 15 confirmed cases, 3 underwent thoracentesis in the chamber whereas 12 did not. All 3 of the instrumented cases were treated using air-only therapeutic tables before the introduction of oxygen-based therapeutic tables for the treatment of SETT accidents.

DISCUSSION

The case reports described typify the presentation of CAGE in the context of SETT. In addition, both patients suffered pulmonary overinflation injury with the release of substantial volumes of air into the pleural spaces and mediastinum. Both cases underwent recompression treatment of similar initial depth and length and both cases had clinically evident pneumothorax at 18 msw (60 fsw). In case 2, the initial volume of extra-pulmonary air was undoubtedly large, as indicated by the presence of obvious cervical subcutaneous emphysema at 50 msw (165 fsw) and the wide distribution of gas demonstrated radiographically posttreatment. Despite this, decompression was achieved without modifying the treatment table and without the development of significant cardiorespiratory compromise. This contrasts with case 1 where no subcutaneous emphysema was evident but marked deterioration in the patient’s clinical condition occurred during the later stages of decompression, presumably due to the expansion of intrapleural gas.

Although the initial sizes of pneumothoraces in the respective cases can never be known with certainty, both cases had comparable signs at 18 msw (60 fsw), suggesting there was no marked difference between them. It could be hypothesized that case 1 experienced a slow continuous leak exacerbating the clinical presentation, whereas case 2 did not. This seems unlikely because, had this been so, an earlier presentation would be expected. Thus, with the exception of the gas breathed during treatment, there seem to be no major clinical differences between the 2 cases that might explain their differing outcome.

An immediate clinical benefit of hyperbaric oxygen (HBO) in the treatment of pneumothorax is that, even where substantial lung collapse persists, an increased inspired partial pressure of oxygen will tend to counteract the hypoxic respiratory
distress that might otherwise occur if the patient were breathing air. Additionally, Daugherty (5) recently outlined the theoretical case for using HBO in preference to surgical thoracentesis in the treatment of pneumothorax diagnosed while under pressure in compression chambers. Breathing gas containing an increased percentage of oxygen greatly enhances the rate of resorption of intrapleural and mediastinal air by promoting the elimination of the nitrogen in the trapped air due to an enhanced partial pressure gradient. Daugherty cites Van Liew’s assertion that breathing 100% oxygen at 2.8 atm abs may “increase the speed of resorption of extrapleural air by a factor of 30 compared to air breathing at 1 ATA.” This implies that all but large pneumothoraces or those with a continuing leak may resorb during the 4 or more hours of HBO administered during a modern oxygen-based therapeutic recompression table (e.g., RN 62 or 63, USN 6 or 6A). Small pneumothoraces may resolve undetected.

Overall, our review of the experience from SETT cases casts doubt on any general requirement for thoracentesis before decompression and lends support to the efficacy of HBO in the treatment of pneumothoraces identified during recompression treatment. Pneumothorax complicating the compression treatment of CAGE victims is a rare entity, occurring in only 15 of 239 reviewed cases (6.3%). Even where pneumothorax is clinically evident, 80% or more (12 out of 15) patients may experience no symptoms of significant cardiorespiratory compromise during the decompression phase of therapeutic recompression. Therefore, in the context of SETT, approximately 1 case of CAGE in 75 (3 from 239) may have a pneumothorax that causes problems in the chamber. If Daugherty’s (5) reasoning holds true, this proportion is likely to decrease further if modern oxygen-based therapeutic recompression tables are used.

In the absence of any suggestion that the clinicopathologic features of pneumothorax after pulmonary overinflation in open-water diving accidents differs from those arising from SETT, these observations should be referable to the problem of pneumothorax complicating recompression treatment in general.

Chamber management of tension pneumothorax

Despite its rarity and despite the potential benefits of oxygen breathing during recompression treatment, cases will occasionally occur where symptoms or signs of tension pneumothorax develop, perhaps aggravated by a continued leak combined with expansion of extrapulmonary gas during decompression. These cases will exhibit signs of cardiorespiratory embarrassment which should alert the vigilant attendant to the problem and enable prompt remedial action. Decision making in these circumstances should be influenced by three basic principles:

1. The diagnosis and treatment of pneumothorax should result in minimal modification of the treatment schedule selected for the underlying decompression illness. Saturation denies a patient rapid access to full-scale medical care. Treatment of the underlying CAGE may rarely require saturation treatment, but generally a short therapeutic table should not become a saturation decompression simply because a pneumothorax is diagnosed. Such extension is unnecessary to treat a simple pneumothorax and potentially hazardous if not indicated for the underlying condition. Unplanned saturation treatments are also expensive and a drain on logistic support.

2. Invasive procedures in the chamber should be the simplest compatible with achievement of the aim.
3. If the planned decompression is expected to be prolonged (e.g., from saturation), invasive procedures should be kept to a minimum to reduce the risk of infection. Cutaneous infection is a potential problem in saturation under normal circumstances. If thoracentesis is required for tension pneumothorax, the smaller the skin wound and the less any instrument or sterile equipment is handled, the more likely it is that infection will be avoided.

Thus, bearing in mind the principles above, if a pneumothorax is diagnosed during decompression, the first management decision is based on whether there is evidence of cardiopulmonary embarrassment suggesting a tension pneumothorax. If not, decompression can proceed with the patient under careful observation. However, if tension pneumothorax develops, decompression should be temporarily suspended. If cardiopulmonary symptoms are life threatening, prompt recompression should be carried out to depth of relief. Two basic treatment options are then proposed, depending on the planned decompression in progress:

**Saturation decompression**

If treatment is on a saturation or prolonged decompression schedule (i.e., days rather than hours), the initial approach should be to administer an enhanced partial pressure of oxygen intermittently to the patient to optimize resorption of intrapleural gas. If recurrence of tension pneumothorax is detected, the rate of decompression should be slowed. This strategy is likely to be successful in the vast majority of cases.

However, where the above measures are not possible or fail to prevent the development of tension pneumothorax, in/out needle or i.v. cannula thoracentesis may be performed as described below. In recurrent cases where a continued leak is suspected, the plastic i.v. cannula (without the needle) may be left in situ and a one-way valve fashioned. If possible, this should be avoided in saturation conditions due to the risk of empyema (5).

**Nonsaturation decompression**

During a nonsaturation therapeutic recompression table where treatment is expected to be completed within a few hours, the diagnosis of pneumothorax should result in minimal modification of the planned decompression schedule. On a modern oxygen-based table, the development of a tension pneumothorax is probably less likely. If it does develop, the risk of infection after thoracentesis while in the chamber is less of a consideration because of the relative brevity of the treatment. What is of concern, however, is that a planned, short oxygen table does not undergo unnecessary transformation into a saturation decompression because of delays imposed by the treatment of a pneumothorax.

If symptoms of tension pneumothorax develop and progress while the chamber is at constant pressure, a continued leak is the likely cause and there is little option but to perform urgent thoracentesis. Much more likely is the development of symptoms during the decompression phase of a treatment due to expansion of intrapleural gas. In either case, decompression should be temporarily suspended and if the condition of the victim is sufficiently severe, recompression to depth of relief is indicated. The medical attendant may then opt either for maximal extension of oxygen-breathing
periods within the limits of the table to allow optimum resorption of intrapleural air before further decompression is attempted, or, for prompt in/out thoracentesis with resumption of the decompression. If tension pneumothorax recurs, thoracentesis can be repeated as necessary or the cannula left in situ with a one-way valve arrangement fitted.

Fig. 1. A decision algorithm for chamber management of pneumothorax.
Thoracentesis with needle or i.v. cannula

The reader should consult a standard text (11) for full details of this procedure. However, in the chamber environment, if relief of a tension pneumothorax is required, a needle or i.v. cannula thoracentesis is recommended as the treatment of choice. This is a quick and relatively simple procedure well within the capabilities of a trained paramedic or non-specialist physician. A variety of specialized instruments are available commercially, but the insertion of an i.v. cannula of any common gauge (ideally 14 or 16 gauge) or even use of a standard 21-gauge syringe needle (or a spinal or cardiac needle) will achieve prompt relief from a tension pneumothorax in an emergency, after which the needle or cannula may be removed. Full expansion of the lung will not occur, but relief of the tension may be all that is required in a patient well oxygenated by HBO.

As it is air rather than fluid that requires drainage, the anterior approach in the mid-clavicular line via the second intercostal space (at the level of the sternal angle) is classically the preferred site (11). Being further from the axilla, it is generally easier to achieve and maintain asepsis at this site in a chamber environment. However, the choice of site may be influenced by factors such as the experience of the medical attendant. A lateral approach, in the mid-axillary line via the 5th or 6th intercostal space (approximately level with the nipples in the male) may also be satisfactory. (Both approaches have hazards: Anteriorly the operator should be aware the internal mammary vessels lie 2–3 cm lateral to the sternal edge. Laterally, the presence of adhesions or the temptation to enter the chest too low may result in damage to the lung itself or risk piercing abdominal organs.)

Whatever approach is chosen, the procedure is often easier if the needle or cannula is mounted on a 5- or 10-ml syringe (if available). The skin should be stretched as the needle is introduced and then released before the needle is advanced through the intercostal muscles and pleura. This avoids a straight needle track and reduces the infection risk. The needle should be kept close to the superior surface of the lower rib to avoid the intercostal neurovascular bundle. If it is intended to leave the cannula in situ, insertion at a slight angle to the chest wall may avoid kinking of the cannula as the lung expands. Entry to the pleural space will be signaled by a satisfying hiss of escaping air or expulsion of air into the syringe.

Asepsis for this quick procedure described above is relatively simple to achieve and analgesia is unnecessary in an emergency (although, if time permits, local anesthetic can be introduced by syringe as the needle or cannula is advanced). The risk of infection can be minimized by immediate removal of the needle after release of the tension.

During both short or saturation decompressions, the in/out thoracentesis can easily be repeated if signs of a tension pneumothorax recur. Alternatively, if the planned decompression is short and if frequent deterioration occurs, or if a continuing leak is suspected during a saturation decompression, then an i.v. cannula, once inserted, can be secured in situ. With the needle withdrawn, a plastic cannula will not damage the expanding lung, although contact between the cannula and visceral pleura may cause the patient to cough. Provided the cannula remains patent, a tension pneumothorax will not recur during decompression and a patient well-oxygenated by HBO should experience minimal distress. In the absence of specific equipment, a one-way valve may be fashioned from the finger of a surgical glove secured to the cannula.
with thread or tape. If available, a three-way tap system with one-way valve and syringe allows aspiration of intrapleural air if desired.

CONCLUSION

Patients may require formal chest tube thoracentesis under controlled conditions once they have completed recompression treatment, but we propose conservative management on modern oxygen-based therapeutic recompression tables as the initial treatment of choice for pneumothorax diagnosed while under pressure. We suggest that classical surgical thoracentesis with chest-tube drainage has no place in modern chamber management of pneumothorax. Needle or i.v. cannula thoracentesis should be reserved for cases developing symptoms or signs of tension pneumothorax where HBO treatment is inappropriate or has failed.

Finally, we suggest that the danger from pneumothorax during recompression treatment of CAGE victims is often overstated and potentially the legacy from the days when air-based therapeutic recompression tables were used routinely. The treatment algorithm proposed in Fig. 1 incorporates the points and principles detailed above and may assist in the chamber management of this uncommon condition.

The authors acknowledge the medical officers and staff of the submarine escape tank in HMS Dolphin, Gosport, for their contribution to the treatment of the cases described, and thank Surgeon Commanders J.J.W. Sykes and T.J.R. Francis for their helpful criticism of the manuscript.

The opinions or assertions herein are the private ones of the authors and are not to be construed as official or reflecting the views of the Royal Navy or the United States Navy.—Manuscript received June 1992; accepted September 1992.

REFERENCES
