CASE REPORT

Successful therapy of cerebral air embolism with hyperbaric oxygen at 2.8 ATA

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Bove AA, Clark JM, Simon AJ, Lambertsen CJ. Successful therapy of cerebral air embolism with hyperbaric oxygen at 2.8 ATA. Undersea Biomed Res 1982; 9(1):75–80.—A 60-year-old male patient suddenly developed bilateral blindness, agitation, and disorientation 36 h after coronary bypass surgery. Onset of symptoms followed efforts to clear an air-filled radial artery cannula. Seven hours after onset of symptoms, initial compression to 2.8 ATA (60 fsw), 100% oxygen (U.S. Navy Table 6), steroids, intravenous fluids, and antiplatelet drugs were used for therapy. The patient’s agitation and disorientation dictated that we avoid initial compression to 6 ATA (165 fsw), contrary to conventional practice in therapy of air embolism, and instead immediately give oxygen at 2.8 ATA. After a second treatment with USN Table 6, given 6 h after the first, the patient’s vision and mental state returned to normal. He subsequently had an uneventful recovery from surgery and cerebral air embolism.

Cerebral air embolism from any cause is treated effectively by exposing the patient to increased ambient pressure to reduce bubble size and by hyperoxygenation to relieve tissue ischemia and enhance bubble resolution. Traditional therapy of this disorder includes an initial brief exposure to an ambient pressure of 6 ATA (165 fsw, 50 msw), followed by prolonged intermittent oxygen breathing at 2.8 ATA (60 fsw, 18 msw). This treatment scheme, designated as U.S. Navy Treatment Table 6A (1), was originally developed for treatment of acute air embolism sustained after rapid ascent during submarine escape training. In spite of its success in cases of air embolism sustained during submarine escape, this treatment scheme may not be appropriate for cases where a long delay occurs between the initial insult and the beginning of therapy. This report describes a successfully treated case of iatrogenic cerebral air embolism where start of therapy was delayed for several hours after onset of symptoms, and where the patient’s condition dictated that we treat initially at 2.8 ATA with oxygen rather than beginning at 6 ATA with air.
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The patient was a 60-year-old male who had coronary bypass surgery one day prior to onset of symptoms. Medical history obtained initially indicated that after a normal postoperative recovery for 27 h the patient was alert and responsive and was sitting in a chair, when he suddenly fell forward and became disoriented and agitated. Neurologic evaluation revealed total bilateral cortical blindness and disorientation to time and place. The remainder of his physical examination was unchanged. Hematocrit, electrolytes, arterial blood gases, and electrocardiogram were all unchanged from earlier in the same day. A CT scan of the brain showed no abnormalities, and the patient was placed on bed rest with the initial diagnosis of cerebrovascular accident. Further history revealed that the symptoms developed immediately following an attempt to clear an air-filled radial artery cannula. From this information cerebral air embolism was diagnosed, and the patient was transferred by ambulance to the Institute for Environmental Medicine for compression therapy.

On arrival at the chamber facility the patient was in acute distress, combative, disoriented, mentally confused, and blind. Blood pressure was 130/80, pulse was 75, and respirations were 14/min. Head and neck examination revealed reactive equal pupils, normal funduscopic examination, full carotid pulses bilaterally, and no neck vein distention. An intravenous line was connected to an external jugular catheter. Chest examination revealed a recent midline sternotomy, two chest drainage tubes connected to a water seal bottle, and rales with diminished breath sounds at the lung bases. Cardiac examination revealed a pulse of 90 with occasional premature beats. There were normal first and second heart sounds, a soft fourth heart sound, a 2-component friction rub anteriorly, and no murmurs. Abdominal examination showed no abnormalities. Our diagnoses were: status post-coronary bypass surgery; acute cerebral air embolism; cardiac arrhythmia; bilateral, basilar pulmonary congestion; and acute psychosis.

Seven hours after onset of symptoms the patient was placed in the hyperbaric chamber and compression was begun. Compression was stopped at 8 fsw when he complained of severe chest pain. Chest tubes were evaluated, and no irregularities were found. The chamber was returned to the surface when his chest pain persisted. Following manipulation of the chest tubes, compression was restarted at a slower rate, and pain did not recur. Because the patient was combative and uncooperative, compression to 2.8 ATA required 30 min. At this time, we decided to remain at that pressure and begin therapy with 100% oxygen. The general treatment plan of U.S. Navy Table 6 with extensions was followed. However, the patient’s disoriented reaction to application of the oxygen mask resulted in an irregular schedule of oxygen administration.

Adjunctive therapy included intravenous saline and dexamethasone, 20 mg i.v., followed by 6 mg i.v. at 4-h intervals. It was necessary to sedate the patient with multiple doses of diazepam (Valium®) and morphine throughout the therapy. The patient had also taken 600 mg aspirin and 200 mg dipyridamol within the last 12 h. Premature beats detected on initial examination remained unchanged in frequency throughout the treatment. As therapy progressed the patient became less agitated, and he was relaxed and cooperative in the final hour of therapy. At the end of the therapy he was calm, with improved orientation and less confusion, but no improvement in his vision could be confirmed.

The patient was returned 6 h later for a second treatment. He was oriented and cooperative at this time, and the chest tubes had been removed. The standard U.S. Navy Table 6 was given uneventfully. At the end of the second therapy, he could sense light but could not visualize images clearly. His cardiac and neurologic status were otherwise unchanged, and he was returned to the referring hospital for continued care. He noted a gradual return of vision
over the following 6–8 h and regained normal vision by 12 h. At that time, no abnormalities were found on detailed ophthalmologic examination. The patient subsequently had an uneventful postoperative course and was normal on follow-up examination 6 weeks after discharge.

DISCUSSION

This case of air embolism is not typical of the cases usually treated with U.S. Navy Table 6A, since there was a significant delay before treatment could be instituted, and the patient’s condition precluded an initial air exposure at 6 ATA (165 fsw). Accordingly, we used Navy Treatment Table 6, and after two treatments cerebral function returned to normal.

Pathophysiology of air embolism

There have been several case reports of air embolism resulting from injection of air into the vascular system (2–4). Pierce has reviewed the clinical features and therapy of such cases (5). The clinical syndrome, which evolves from a bolus of air entering the vascular system, differs depending on the point of entry and ultimate destination of the air. Venous air embolism produces a syndrome similar to pulmonary embolism (6). Patients with gross venous embolism die of acute cardiopulmonary decompensation due to obstruction of lung vasculature. Secondary bubble effects appear to play an important role in the pathophysiology of venous embolism. Arterial air embolism is less likely to occur during surgery, except in the case of cardiac surgery where the patient is placed on heart-lung bypass and one or more cardiac chambers may be opened.

Several studies report cases of surgically induced cerebral air embolism with significant morbidity and mortality (7–9). Tragically, in some cases of surgically induced air embolism there is no awareness of the existence of a definitive treatment: compression and hyperbaric oxygenation. The case reported herein demonstrated that air injected into the radial artery of an upright patient can be forced against the flow of blood, enter the vertebral circulation, and cause acute occlusion of the posterior cerebral circulation. Because the two vertebral arteries are conjoined at the basilar artery, bilateral symptoms occur in this process. Thus, our patient developed a bilateral occipital lobe deficit, manifested by cortical blindness, even though air entered the arterial system unilaterally.

Hyperbaric oxygen therapy of cerebral air embolism

The value of hyperbaric oxygen in treatment of cerebral air embolism is well accepted. Cerebral blood flow is known to be reduced following cerebral air embolism (10) and, because gas emboli may distribute throughout the arterial system, flow to other organs may also be compromised (11). Increasing arterial Po2 by oxygen breathing at increased ambient pressure will increase tissue oxygen tension in regions of marginal blood flow at the same time that it compresses gas bubbles and speeds their resolution. The successful use of hyperbaric oxygen in serious decompression sickness with spinal cord involvement (12) also provides evidence for its efficacy.

Cerebral air embolism has been known in diving and hyperbaric medicine for many years, and effective therapy is based on exposure to increased ambient pressure (13). When immediate recompression is possible, initial exposure to air at 6 ATA (165 fsw), followed by oxygen breathing at 2.8 ATA, has proved rapidly effective in most cases (13). Hart (14) has described a method of therapy using oxygen at 3 ATA (66 fsw, 20 msw) with a monoplace chamber. His
success rate compared favorably to that found with Navy Table 6A. He also noted reduced success when long delays preceded therapy. Leitch (15) suggests that oxygen at 18 msw (60 fsw) be used in cases where therapy is delayed for over 5 h.

Adjunctive therapeutic measures

When prolonged delay of compression therapy allows secondary effects of the embolism to become significant (16), it is advantageous to give drugs that block or reverse these secondary effects. Recent studies have demonstrated that intravascular bubbles not only occlude arteries mechanically but also trigger a variety of secondary processes that lead to platelet aggregation and thrombus formation (17), capillary leakage (18), and prolonged local ischemia (19). In order to oppose or reverse these secondary effects, steroids are used to maintain capillary endothelial integrity; antiplatelet agents are used to prevent platelet clumping in the obstructed vessels; fluid therapy is provided to avoid dehydration and hemoconcentration; and hyperbaric oxygen is used to improve oxygenation of ischemic tissues and to hasten bubble resolution. Recent trends in therapy of cerebral air embolism and decompression sickness (20) indicate that these adjunctive measures should be used in conjunction with compression therapy. The nature of secondary effects of emboli suggests that adjunctive drug therapy may become more important when compression therapy with hyperoxygenation is delayed. Although initial exposure of this patient to 6 ATA was planned, events precluded going to this depth, and the patient was treated instead with an extended U.S. Navy Table 6. Intravenous fluids, steroids, and antiplatelet agents (aspirin and dipyridamol administered as part of the postoperative therapy of coronary bypass surgery) were also considered important in his therapy.

Multiple compression therapies

Recent use of multiple hyperbaric oxygen treatments over a period of several days indicates that this concept of therapy is valid when significant residual neurologic deficit remains after the first therapy (21, 22). This approach can be justified especially when considering that cerebral edema may persist for several days and cause continued ischemia that can be ameliorated by oxygen at high pressure. Indeed, continuous prolonged administration of hyperbaric oxygen would constitute the ideal therapy if pulmonary and cerebral oxygen poisoning could be eliminated.

SUMMARY AND CONCLUSIONS

Cerebral air embolism sustained from pulmonary barotrauma related to diving is routinely treated with compression and hyperbaric oxygen. This therapy is well established and accepted by physicians involved with the care of divers. The same lesion induced by surgery has not, to date, been routinely treated with increased pressure, and the resulting mortality reflects this inadequate management.

This case demonstrates that hyperbaric oxygen therapy can be successful even when long delays occur prior to treatment. Thus, when air embolism occurs from any cause, hyperbaric oxygenation should be the first consideration in treatment. Significant preservation of cerebral function and decreased mortality should be expected with this therapy. Intravenous fluids,
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Steroids, and antiplatelet drugs are valuable adjunctive measures, especially when a long delay occurs between onset of symptoms and initiation of hyperbaric oxygen therapy.

This work was supported by USPHS-NIH Grant HL-08899 and the Office of Naval Research through Contract N00014-7-C-0048.

Dr. Bove’s work was done during tenure as an established investigator of the American Heart Association—Manuscript received for publication October 1981; revision received January 1982.

Bove AA, Clark JM, Simon AJ, Lambertsen CJ. Succès du traitement d’un aéroembolisme cérébral par l’oxygène hyperbare à 2,8 ATA. Undersea Biomed Res 1982; 9(1):75–80.—Un malade de sexe masculin, âgé de 60 ans, a présenté de façon soudaine une cécité bilatérale, de l’agitation, et un trouble de l’orientation, 36 heures après la mise en place d’un shunt coronarien. L’apparition des symptômes a suivi des tentatives effectuées pour purger une cannule de l’artère radiale, remplie d’air. Sept heures après que soient apparus les premiers symptômes on a donné comme traitement, une première compression à 2.8 ATA à 100% d’oxygène (Table 6 de l’U.S. Navy), des stéroïdes, des perfusions intraveineuses et des produits antiplaquettaires. L’agitation et le trouble de l’orientation du malade nous ont commandé d’éviter la première compression jusqu’à 6 ATA (165 pieds) comme l’aurait voulu la pratique conventionnelle pour le traitement des aéroembolismes, et de donner immédiatement à la place de l’oxygène à 2,8 ATA. Après un second traitement selon la Table 6 USN, effectué six heures après le premier, la vision et l’état mental du malade sont revenus à la normale. Il a par la suite récupéré sans incident de l’intervention chirurgicale et de l’aéroembolisme cérébral.

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References


