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Hyperbaric Treatment of Cerebral Air Embolism Sustained During an Open-Heart Surgical Procedure

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A case of cerebral air embolism sustained during replacement of the mitral valve resulted in postoperative coma and seizures. Hyperbaric treatment, begun 30 hours after the occurrence of the air embolism, resulted in good immediate and long-term recovery. Mild deficits of the left hemisphere were present at follow-up 53 days after the embolus was sustained, and lesser, minimal residua were present at 14-month follow-up. Hyperbaric treatment is the definitive therapy for cerebral air embolism. Although it is most effective when administered early, the outcome may be excellent even with late treatment.

When air enters the arterial circulation, it forms emboli, which become symptomatic if they lodge in organs that serve essential functions. If they reach the brain, they may cause diffuse and focal neurologic deficits.¹⁻³ The incidence of recognized air embolism as a complication of openheart operations performed with current techniques of extracorporeal circulation has been reported as 1.2 to 1.4 per 1,000 patients.⁴⁻⁶ Such emboli result in severe neurologic sequelae or death in approximately a third of the cases.^{4,7} Hyperbaric treatment is the definitive therapy for cerebral air embolism.^{8,9} Although prompt intervention is associated with the best outcome,^{1,2,9} successful treatment has been reported in isolated cases even after a delay of 29 hours.^{10,11} Herein we report an additional case of excellent outcome after delayed treatment of cerebral air embolism.

REPORT OF CASE

A 50-year-old left-handed woman with progressive mitral stenosis underwent an initial procedure for replacement of the mitral valve in 1981.

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Between November 1986 and November 1987, she had an occlusion of a branch of the left ophthalmic artery, an episode of right amaurosis fugax, and two transient episodes of dysarthria, light-headedness, and weakness, greater on the left than on the right side, despite treatment with warfarin and dipyridamole. Results of a neurologic examination were normal. Α small residual paracentral scotoma on the left, improved from the year before, was noted on formal testing of visual fields. Computed tomography of the head without use of contrast medium disclosed two zones of infarction in the anterior left basal ganglia. Although cardiovascular examination, including echocardiography, showed a normally functioning valve, the prosthetic mitral valve was thought to be the embolic source.

In December 1987, the patient underwent replacement of the valve. Shortly after initiation of cardiopulmonary bypass, during insertion of a left atrial vent, approximately 100 to 200 ml of air entered the atrium, passed to the ventricle, and was ejected into the ascending aorta. The aorta was cross clamped, the patient was placed in the Trendelenburg position, 400 ml of cardioplegia solution was administered through the aortic root, and air was removed from the system at normothermia. Circulatory arrest for 6 minutes was necessary to accomplish these maneuvers, during which time thiopental sodium (500 mg) and dexamethasone (25 mg) were administered intravenously. Ventilation was resumed at a fractional concentration of oxygen in inspired gas of 100%. After satisfactory resumption of extracorporeal circulation, the mitral valve was replaced without further complication. The total time of cardiopulmonary bypass was 115 minutes, the mean perfusion pressure was 60 mm Hg, and the patient was cooled to 20°C. She was weaned from bypass without vasopressors and left the operating room in a hemodynamically stable condition.

After the operation, the patient remained unresponsive, opening her eyes spontaneously and making occasional decerebrate movements. At 18 hours after operation, status epilepticus developed, clinically manifested as twitching of the right upper extremity and both eyebrows. The seizures were stopped by intravenous administration of 500 mg of phenytoin. She then opened her eyes spontaneously but did not respond to threats, noises, or commands. She withdrew the left lower extremity to pain but had poor withdrawal on the right. The upper extremities did not respond to painful stimuli. A bilateral grasp reflex was present. Deep tendon reflexes were brisk and symmetric, and plantar responses were flexor. The pupils were equal, 1.5 mm in diameter, and reactive to light. Conjugate horizontal and vertical eve movements occurred in response to the doll's eyes maneuver. Funduscopic examination through dilated pupils showed blanched, nonfilling retinal arteries bilaterally, with partial filling only of the right inferior temporal and the left superior temporal branches. Streaming air bubbles were not seen. No spontaneous respirations occurred when the patient was off the ventilator for up to 30 seconds. A computed tomographic scan of the head (Fig. 1) showed no change from the preoperative computed tomographic findings. Electroencephalographic examination showed a reactive theta coma pattern, decreased in amplitude on the left.

A loading dose of 1,000 mg of phenytoin was administered intravenously; the resultant serum level was $23.4 \,\mu$ g/ml. The medical management thus far had consisted of replacement of blood loss with colloids, ventilatory support with hyperventilation to maintain an arterial oxygen tension of more than 80 mm Hg and an arterial carbon dioxide tension of less than 30 mm Hg, and infusion of dopamine at 2 μ g/kg per min.

Probable cerebral air embolism was diagnosed, and the patient was transferred in a hemodynamically stable condition by a low-flying (152m) helicopter on 100% oxygen to the Multiplace Hyperbaric Unit at the Hennepin County Medical Center for hyperbaric treatment.

Hyperbaric treatment was initiated in accordance with the US Navy Dive Table 6A for air embolism¹² 26 hours after the end of operation and 30 hours after the occurrence of the air embolus. Treatment was rendered in a cylindri-



Fig. 1. A and B, Computed tomograms of head without use of contrast medium obtained on first day after patient underwent replacement of a prosthetic mitral valve. Two zones of infarction were evident in anterior left basal ganglia (unchanged from preoperative scan).

cal multiplace chamber (2.7 by 4.9 m), which was equipped with locks that allowed entry and exit of equipment and personnel while the chamber was pressurized.

At the start of treatment, the patient had random spontaneous eye movements but did not attend or follow commands. Corneal and gag reflexes were intact, and shallow, irregular respirations occurred without the ventilator. The patient had spontaneous nonpurposeful movement of all extremities, lower greater than upper and left greater than right, and nonpurposeful withdrawal of all extremities to painful stimuli. Hyperreflexia, greater on the right, sustained ankle clonus, and extensor plantar reflexes were noted bilaterally.

After 25 minutes of treatment, the patient appeared more awake and opened her eyes reliably to vocal stimuli. Lower extremity clonus was no longer elicitable, and plantar responses were flexor. She did not, however, follow simple commands. Because her condition was improving, the treatment time was extended, and a US Navy Table 4, with modifications, was followed thereafter.

At 4.5 hours into the "dive," the patient was breathing well without the ventilator and appeared to be making minor purposeful movements. Five hours into treatment, she exhibited twitching of the right wrist, which was readily controlled by administration of lorazepam. During the last hours of her 38-hour hyperbaric treatment, she opened her eyes to vocal stimuli, looked at the attendants' faces, closed her eyes on command, and extended her legs from the preferred flexed position on command.

The patient's condition temporarily worsened 6 hours after completion of hyperbaric treatment. The next morning, an electroencephalogram showed some left midtemporal spikes. An episode of tremor of the right hand had no correlated electroencephalographic abnormality. Shortly afterward, the patient awoke and responded appropriately and consistently to her husband and to the examiner. The next day, she followed two-step commands, but her right upper extremity appeared paretic and she had aphasia. That night she was able to feed herself (although she was "somewhat sloppy," according to her husband), and she could walk. The next morning, she began expressing words, initially "I...," then "I want...," and subsequently "I want ice" and "I want juice." We elected not to give additional hyperbaric treatments because the patient showed signs of mild pulmonary oxygen toxicity, and she was improving neurologically. She was transferred back to a Mayoaffiliated hospital 7 days after the mitral valve replacement procedure and 4 days after completion of the hyperbaric treatment.

On examination at that time, the patient had right-sided upper motor neuron weakness of the upper extremity and face and absent sensation to pinprick, right-sided neglect to bilateral simultaneous visual and tactile stimuli, and apraxia in the performance of skilled and alternate movements of both upper extremities, the tongue, and the eyes. A moderate to severe aphasic language deficit that encompassed all linguistic modalities, involving both comprehension and expression, was noted in conjunction with difficulty in naming and repetition, speech apraxia, and frequent use of clichés. She was unable to read words but could name some letters correctly. Although she was left-handed, she was unable to write, draw, or copy with her left hand, and her graphic repertoire was limited to D-shaped and circular forms. She followed simple one-step commands, recognized her husband, and correctly identified the pictures of two presidents. She was unable to perform a task that involved matching shapes and colors. An electroencephalogram showed minimal slowing of the background activity. A magnetic resonance imaging study of the head (Fig. 2) disclosed an area of increased T2 signal involving the left lenticular nucleus and extending into the periventricular white matter, consistent with infarction, a small infarct involving the left thalamus, and mild generalized cerebral atrophy. She underwent intensive physical, occupational, and speech therapy and had gradual improvement of her condition. Mild deficits of the left hemisphere were present at follow-up examination 53 days after the embolus was sustained, and lesser, minimal residua were present at 14-month follow-up.

DISCUSSION

The pathologic features of cerebral air emboli are due to arrested gas emboli in the arterioles, which cause obstruction and decreased blood flow, immediate vasoconstriction followed by prolonged vasodilation, and dysfunction of the blood-brain barrier in association with extravasation of fluid and electrolytes and subsequent cerebral edema. Lesions are multifocal, but the gray matter is relatively more affected than the white matter; lesions have a tendency to occur in the boundary zones between the cortical areas supplied by the three principal cerebral arteries. On microscopic examination, the lesions are similar to acute ischemic insults of other origin.^{1,13}

The clinical manifestations of cerebral air embolism include general symptoms such as coma, convulsions, cardiorespiratory collapse, stupor, and confusion as well as localized symptoms such as bilateral or unilateral sensory or motor changes, isolated cranial nerve dysfunction, and localized brain-stem findings.¹ Most commonly, the symptoms of cerebral air emboli reflect multifocal cerebral and brain-stem insults rather than deficits confined to a single cerebral arterial supply,^{1,2} regardless of the cause of the air embolism.^{3,4,6,14-18}

A specific sign of arterial air embolism is air in retinal vessels. Other signs, noted rarely, are sharply defined areas of pallor in the tongue (Liebermeister's sign), marbling of the skin, or air bleeding from a small skin incision made in the most superior portion of the body. Radiographic demonstration of air in the cerebral vessels is uncommon.¹⁹ The absence of any or all of these signs does not exclude the diagnosis.

The initial treatment of cerebral air embolism includes 100% oxygen to counteract the cerebral hypoxic insult of the emboli and the Trendelenburg position to minimize further distribu-



Fig. 2. A and B, Magnetic resonance images of head (same patient as in Figure 1) obtained on 14th postoperative day. Area of increased T2 signal involved left lenticular nucleus and extended into periventricular white matter, consistent with infarction.

tion of gas to the cerebral circulation. The maintenance of adequate tissue perfusion with fluid support and the use of glucocorticoids to decrease vasogenic cerebral edema are widely accepted adjunctive measures.¹ The definitive therapy, however, is hyperbaric treatment, which should be undertaken as soon as possible.^{1,8,20}

The rationale for hyperbaric treatment is threefold. First, compression to 6 atmospheres (165 feet seawater), which is the initial step in most therapeutic regimens for arterial gas embolism, reduces the diameter of air bubbles by 45%. Bubbles that occlude arteries or arterioles may be able to advance in the arterial tree, and blood flow to previously compromised tissue is immediately restored. The tiniest bubbles, which occlude end-arterioles in critical structures such as the brain stem, will be able to advance to the venous circulation and will no longer cause critical focal ischemia. Second, hyperbaric oxygen treatment facilitates resorption of bubbles by increasing the bubble surface area-to-volume ratio and producing a large diffusion gradient, which favor absorption of the nitrogen within the bubble by the hyperoxic surrounding plasma. Third, the high arterial oxygen tension provides an increased plasmato-tissue oxygen diffusion gradient to support tissues compromised by poor blood flow.

The desirability of instituting recompression therapy as soon as possible in cases of arterial air emboli is uncontested because the outcome is better with prompt intervention.^{1,2,9} At what point after embolism hyperbaric treatment ceases to be of value has not been established. The decision to treat the current patient despite the 30-hour delay after embolization was based on several factors. First, the possibility that intravascular air might still be present was considered because some nonfilling retinal arterioles continued to be evident. Cerebral bubbles have been found in pathologic studies as long as 48 hours after embolization.²¹ In that setting, some neurologic dysfunction might still arise from an ischemic penumbra, resulting from a decrease in local blood flow to less than a critical value into a range where it can maintain structurally intact neurons but is insufficient to support their function.²² We expected this dysfunction to be reversible with restoration of flow to more than that critical value as a result of treatment. Second, the patient's reactive electroencephalographic pattern suggested that ischemic changes might still be reversed with the reduction of cerebral edema²³ by the hyperoxygenation of the recompression treatment. Third, successful hyperbaric treatment of cerebral air embolism has been reported previously after a delay of 29 hours.^{10,11}

CONCLUSION

Although we have no proof that hyperbaric treatment contributed to our patient's recovery, the immediate improvement of her neurologic status on pressurization is highly suggestive, as were her gradual improvement during treatment and temporary regression after it was completed. Hyperbaric treatment is the definitive therapy for cerebral air embolism. Diagnosis and treatment depend on awareness of its occurrence and familiarity with its manifestations. Even late treatment may be associated with a satisfactory outcome.

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REFERENCES

1. Pearson RR: Diagnosis and treatment of gas embolism. In The Physician's Guide to Diving Machine. Edited by CW Shilling, CB Carlston, RA Mathias. New York, Plenum Press, 1984, pp 333-367

- 2. Peirce EC II: Cerebral gas embolism (arterial) with special reference to iatrogenic accidents. Hyperbaric Oxygen Rev 1:161-184, 1980
- 3. Gillen HW: Symptomatology of cerebral gas embolism. Neurology 18:507-512, 1968
- Stoney WS, Alford WC Jr, Burrus GR, Glassford DM Jr, Thomas CS Jr: Air embolism and other accidents using pump oxygenators. Ann Thorac Surg 29:336-340, 1980
- 5. Spampinato N, Stassano P, Gagliardi C, Tufano R, Iorio D: Massive air embolism during cardiopulmonary bypass: successful treatment with immediate hypothermia and circulatory support. Ann Thorac Surg 32:602-603, 1981
- Fishman NH, Carlsson E, Roe BB: The importance of the pulmonary veins in systemic air embolism following open-heart surgery. Surgery 66:655-662, 1969
- Cuetter AC, Ferrans VJ: Neurological complications of cardiovascular therapy. Curr Probl Cardiol 12: 153-211, 1987
- 8. Davis DL, Elliott DH: Treatment of the decompression disorders. *In* The Physiology and Medicine of Diving. Edited by PB Bennett, DH Elliott. Third edition. London, Baillière Tindall, 1982, pp 473-487
- Leitch DR, Greenbaum LJ Jr, Hallenbeck JM: Cerebral arterial air embolism. I. Is there benefit in beginning HBO treatment at 6 bar? II. Effect of pressure and time on cortical evoked potential recovery. III. Cerebral blood flow after decompression from various pressure treatments. IV. Failure to recover with treatment, and secondary deterioration. Undersea Biomed Res 11:221-235; 237-248; 249-263; 265-274, 1984
- 10. Murphy BP, Harford FJ, Cramer FS: Cerebral air embolism resulting from invasive medical procedures: treatment with hyperbaric oxygen. Ann Surg 201:242-245, 1985
- 11. Mader JT, Hulet WH: Delayed hyperbaric treatment of cerebral air embolism: report of a case. Arch Neurol 36:504-505, 1979
- US Navy Diving Manual (NAVSEA 0994-LP-001-9010). Vol 1: Air Diving. Revision 1. Washington, DC, US Government Printing Office, June 1985, pp 8-37
- 13. Garcia JH, Klatzo I, Archer T, Lossinsky AS: Arterial air embolism: structural effects on the gerbil brain. Stroke 12:414-421, 1981
- Thomas AN, Stephens BG: Air embolism: a cause of morbidity and death after penetrating chest trauma. J Trauma 14:633-637, 1974
- Halpern P, Greenstein A, Melamed Y, Taitelman U, Sznajder I, Zveibil F: Arterial air embolism after penetrating lung injury. Crit Care Med 11:392-393, 1983
- Peters EJ, Boyars MC: Arterial air embolism after a percutaneous needle lung aspiration. J Hyperbaric Med 1:151-155, 1986
- 17. Winter PM, Alvis HJ, Gage AA: Hyperbaric treatment of cerebral air embolism during cardiopulmonary bypass. JAMA 215:1786-1788, 1971

- Takita H, Olszewski W, Schimert G, Lanphier EH: Hyperbaric treatment of cerebral air embolism as a result of open-heart surgery: report of a case. J Thorac Cardiovasc Surg 55:682-685, 1968
- Durant TM, Oppenheimer MJ, Webster MR, Long J: Arterial air embolism. Am Heart J 38:481-500, 1949
- 20. Heimbach RD, Sheffield PJ: Decompression sickness and pulmonary overpressure accidents. *In* Fundamentals of Aerospace Medicine. Edited by RL DeHart. Philadelphia, Lea & Febiger, 1985, pp 132-161
- 21. Fries CC, Levowitz B, Adler S, Cook AW, Karlson KE, Dennis C: Experimental cerebral gas embolism. Ann Surg 145:461-470, 1957
- Astrup J, Symon L, Branston NM, Lassen NA: Cortical evoked potential and extracellular K⁺ and H⁺ at critical levels of brain ischemia. Stroke 8:51-57, 1977
- 23. Sukoff MH, Hollin SA, Jacobson JH II: The protective effect of hyperbaric oxygenation in experimentally produced cerebral edema and compression. Surgery 62:40-45, 1967

