

# Arterial Helium Embolism from a Ruptured Intraaortic Balloon

James W. Frederiksen, M.D., Jacqueline Smith, R.N., M.S., Paul Brown, M.D., and Claude Zinetti, M.D.

**ABSTRACT** A 67-year-old man sustained a sudden global neurological deficit immediately following rupture of an intraaortic balloon catheter. Considerable improvement in his neurological function occurred during and after three hyperbaric oxygen treatments.

Arterial helium embolization consequential to rupture of an intraaortic balloon catheter may cause ischemic injury to the brain, heart, and other less critical organs. The following case report suggests that a patient who sustains severe neurological injury after balloon rupture may recover substantial neurological function during and after treatment with hyperbaric oxygen.

A 67-year-old man was admitted to an outside hospital because of severe chest pain. Emergency coronary arteriography showed occlusion of the proximal right coronary artery and 90% obstruction of the proximal left anterior descending coronary artery. Patency of the right coronary artery was restored with intracoronary infusion of streptokinase. Because of subsequent hemodynamic instability, a 10.5F intraaortic balloon catheter (Datascope, Percor Stat D. L.; balloon volume, 40 ml) was inserted into the right femoral artery and advanced into the proximal descending thoracic aorta under fluoroscopic guidance. The balloon tip was positioned just distal to the orifice of the left subclavian artery. The patient was then transferred to Northwestern Memorial Hospital.

Six days later, the patient underwent double coronary artery bypass grafting. By 12:30 P.M., one hour following completion of the operation, the patient was awake, moving all four extremities, and responding appropriately to simple commands. At 6:15 P.M., the patient suddenly became unresponsive and lost all voluntary neurological function. Blood was also noted in the balloon catheter of that time. The balloon was removed immediately, the inspired oxygen concentration increased to 100%, and 12 mg of dexamethasone given intravenously. Following balloon removal, the patient suffered slow, intermittent focal seizures for approximately three minutes. Thereafter he became totally flac-

cid. Inspection of the inflated balloon revealed the presence of a gas leak through a pinhole opening in the balloon tip (Figure).

The patient was transferred by ambulance to Edgewater Hospital, which is equipped with a multiple-occupancy, 6-atmosphere hyperbaric chamber. He was treated for gas embolism according to the 5-hour compression-decompression protocol outlined in Table 6A of the *United States Navy Diving Manual* [1]. Compression to 6 atmospheres pressure absolute while the patient was ventilated with a 50% oxygen-50% nitrogen mixture (inspired oxygen tension [PiO<sub>2</sub>], 2280 mm Hg) was achieved at 8:55 P.M. After decompression to 2.8 atmospheres, the patient was ventilated with 100% oxygen (PiO<sub>2</sub>, 2180 mm Hg). By 9:15 P.M., the patient had opened his eyes and begun moving his left arm and leg to commands. His right side remained flaccid, however, and by the end of the decompression period, his responsiveness and left-sided improvement had failed to persist.

A second compression-decompression treatment was performed at 11:00 A.M. on the first postoperative day and a third at 9:30 A.M. on the second postoperative day. These two treatments were performed according to the 4½-hour protocol outlined in Table 6 of the *United States Navy Diving Manual* [1], which calls for 100% oxygen breathing at an initial pressure of 2.8, instead of 6, atmospheres pressure absolute. During the second treatment the patient's responsiveness and his left-sided strength returned and remained following decompression. He also regained transient function of his right leg. During the third treatment, his right leg and arm function returned and remained following decompression. He was extubated on the following day and began ambulating independently two days later. Aphasia did not develop, although a magnetic resonance imaging (MRI) study of the brain showed multiple regions of cortical infarct involving the left hemisphere. The patient was discharged with only mild residual weakness on the eleventh postoperative day. During an office visit seven months after operation the patient felt well and had no evident motor weakness or dysfunction.

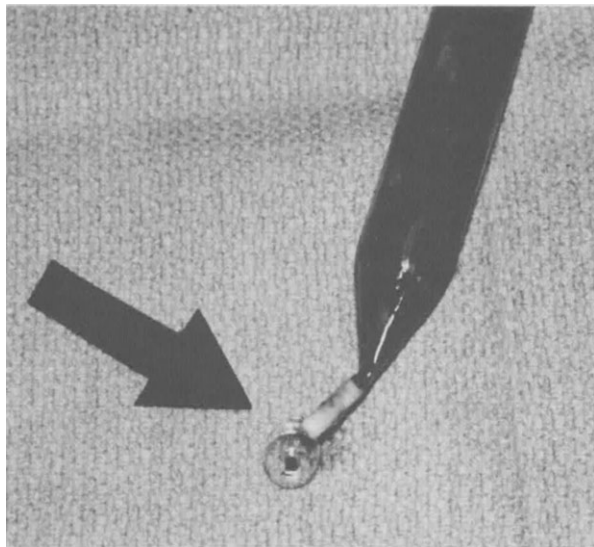
## Comment

This patient is considered to have had a cerebral helium embolism, since the onset of his global neurological deficit and the leak in the tip of the balloon catheter occurred simultaneously. The fact that his left-sided motor function began returning before his right-sided function suggests that a greater volume of helium entered the left side of the cerebral circulation than the

From the Department of Surgery, Division of Cardiothoracic Surgery, Northwestern University Medical School, Northwestern Memorial Hospital and the Department of Medicine, Edgewater Hospital, Chicago, IL.

Accepted for publication July 28, 1988.

Address reprint requests to Dr. Frederiksen, Northwestern University Medical School, 303 East Chicago Ave, Ward 9-105, Chicago, IL 60611.



Blood-filled balloon catheter inflated to demonstrate the leak at the tip.

right side. The position of the balloon tip and the MRI findings support this hypothesis.

The danger of arterial gas embolism from rupture of an intraaortic balloon catheter has been recognized since 1962 when Mouloupoulos and colleagues [2] first demonstrated the feasibility of intraaortic balloon pumping. Emphasizing this danger, Feola and associates [3] warned in 1971 that "accelerated fatigue testing of the balloon is no guarantee that in actual application in a human a pinhole leak, with the resulting development of massive [gas] embolism, would not occur." Furman and co-workers [4] studied the pathological sequelae of helium embolism after injecting measured volumes of this gas into the proximal descending aortas of anesthetized dogs. They found that substantial cerebral and coronary artery obstruction occurred following an injection of 0.5 to 1.0 ml per kilogram of body weight, and that death usually occurred within minutes following injection of a larger volume. In 1973, Scheidt and co-workers [5] reported that a patient in their series had died as the result of a balloon rupture, although they did not state whether evidence of gas embolism was found on postmortem examination.

Early hyperbaric oxygen treatment is the only specific therapy for gas embolism [6]. It has been shown to reduce the volume of intravascular gas bubbles [6, 7], reduce hypoxia by increasing the distance oxygen will diffuse [6-8], reduce cerebral edema by lowering intracranial pressure [8, 9], and reduce the inflammatory response due to blood-bubble interaction [7]. Should neurological symptoms persist after the initial hyperbaric treatment, as was the case in our patient, Kindwall [10] recommends additional treatments.

Fortunately, balloon rupture and serious complications from rupture have occurred very rarely. McEnany and colleagues [11] reported only one leak in their series

of 729 cases of balloon catheters. Kantrowitz and associates [12], in a more recent study, reported only 14 leaks among over 800 cases of balloon catheters. No patient in either series showed clinical evidence of gas embolism. Several other authors who have reported isolated cases of balloon rupture also have observed no untoward effects [13-15].

The incidence of apparent complications following balloon rupture may be low for several reasons. First, most pumps are designed so that the balloon volume is slightly larger than the total volume of shuttle gas used to achieve inflation [16]. Even at the time of peak inflation, therefore, the pressure inside the balloon equals the pressure in the aorta, the balloon membrane remains flaccid, and no gas escapes if the leak is small enough. Second, surface tension at the leak site helps to impede the egress of shuttle gas [17]. Third, the presence of blood in the catheter usually prompts early removal of the balloon.

The patient's neurological deficit might have been less severe if carbon dioxide had been the shuttle gas, since the solubility of carbon dioxide in blood greatly exceeds that of helium [18]. However, inflation and deflation can be achieved more rapidly with helium, especially through small-diameter balloon catheters designed for percutaneous insertion [19]. For this reason, all balloon pumps currently marketed in the United States\* use helium as the shuttle gas [19]. Clinicians using balloon pumps should be aware that helium embolism, while rare, can produce devastating ischemic complications that early hyperbaric oxygen can help to reverse.

## References

1. Department of the Navy. United States Navy Diving Manual. Washington, D.C., U.S. Government Printing Office, 1978, pp 8-24; 8-25. Cited in Strauss RH: Diving Medicine. New York, Grune & Stratton, 1976, pp 359-360
2. Mouloupoulos SD, Topaz S, Kolff WJ: Diastolic balloon pumping (with carbon dioxide) in the aorta—a mechanical assistance to the failing circulation. *Am Heart J* 63:669, 1962
3. Feola M, Adachi M, Akers WW, et al: Intra-aortic balloon pumping in the experimental animal: effects and problems. *Am J Cardiol* 27:129, 1971
4. Furman S, Vijaynagar R, Rosenbaum R, et al: Lethal sequelae of intraaortic balloon rupture. *Surgery* 69:121, 1971
5. Scheidt S, Wilner G, Mueller H, et al: Intra-aortic balloon pumping in cardiogenic shock. *N Engl J Med* 288:979, 1973
6. Pierce EC: Specific therapy for arterial air embolism (editorial). *Ann Thorac Surg* 29:300, 1980
7. Murphy BP, Harford FJ, Cramer FS: Cerebral air embolism resulting from invasive medical procedures. Treatment with hyperbaric oxygen. *Ann Surg* 201:242, 1984
8. Sukoff MH, Hollin SA, Espinosa OE, Jacobson JH: The protective effect of hyperbaric oxygenation in experimental cerebral edema. *J Neurosurg* 29:236, 1968

\*The four companies currently marketing balloon pumps are Aries Medical, Datascope Corporation, Kontron, and Mansfield Scientific, Incorporated.

9. Miller JD, Ledingham IM: Reduction of increased intracranial pressure. *Arch Neurol* 24:210, 1971
10. Kindwall EP: Hyperbaric and ancillary treatment of decompression sickness, air embolism, and related disorders. In Strauss RH (ed): *Diving Medicine*. New York, Grune & Stratton, 1976, pp 83-96
11. McEnany MT, Kay HR, Buckley MJ, et al: Clinical experience with balloon pump support in 728 patients. *Circulation* 58(Suppl I):I-124, 1978
12. Kantrowitz A, Tarik W, Freed P, et al: Intra-aortic balloon pumping 1967 through 1982. Analysis of complications in 733 patients. *Am J Cardiol* 57:976, 1986
13. Rajami R, Keon WJ, Bedard P: Rupture of an intra-aortic balloon. *J Thorac Cardiovasc Surg* 79:301, 1980
14. Mayerhofer KE, Billhardt RA, Conдини MA: Delayed abrasion perforation of two intra-aortic balloons. *Am Heart J* 108:1361, 1984
15. Alimo EB, Foster JM, Cashman JN, Casson WR. Balloon perforation. *Anesthesiol Rev* 13:49, 1986
16. Ohley WJ: Mechanical support of the failing circulation. In Callaghan JC, Wortak J (eds): *Open Heart Surgery, Theory and Practice*. New York, Praeger, 1986, pp 127-142
17. Ohley WJ, Spraker TE, Hamilton R: Characteristics of leaks through holes in intra-aortic balloons. In *Proceedings of the Association for the Advancement of Medical Instrumentation, 17th Annual Conference, 1982*, p 92
18. Weast RC: *Handbook of Chemistry and Physics*. Boca Raton, Chemical Rubber Company Press, 1982, pp B-82, B-93
19. Intra-aortic balloon pumps. *Health Devices* 16:135, 1987