Case reports: Hyperbaric oxygen therapy for the treatment of cerebral air embolism

BRITO T[1], PITHAN N[1], MARTINS G[2], JESSEN B[2], ASSUMPÇÃO C[2], PORTO T[2], FILHO O[3], SIQUEIRA-FILHO A[4]

[1] OHB-RIO Clinica Hiperbárica, Hospital Casa de Portugal, Rio de Janeiro, Brazil
[2] Instituto Estadual de Cardiologia Aloysio de Castro, Rio de Janeiro, Brazil
[3] Hospital Naval Marcilio Dias, Rio de Janeiro, Brazil
[4] Hospital Universitário Clementino Fraga Filho, Universidade Federal do Rio de Janeiro, Brazil

CORRESPONDING AUTHOR: Tomaz Brito, M.D. – *tombrito@infolink.com.br*

ABSTRACT

Cerebral air embolism is one of the most deleterious disorders that may affect divers, but it is also a possible complication of surgeries and medical procedures. We report our experience with iatrogenic cerebral air embolism and hyperbaric oxygen treatment.

INTRODUCTION

The occurrence of arterial gas embolism (AGE) during cardiopulmonary bypass (CBP) is estimated in 0.1% of cases [1], but the incidence of AGE after CBP or in the postoperative period has not been clearly defined. It is also estimated that many occurrences of AGE during or after CBP are underdiagnosed or misdiagnosed due to several possible clinical presentations of this complication, which vary from mild headache or discrete psychological or personality changes, to motor and sensory deficits, convulsions, coma or even death. The condition is also affected by the variation of time intervals from the end of the surgery to the perception or identification of the neurological deficits [2,3,8].

There are several possible causes of AGE [3], and based on many publications and clinical evidence worldwide, whatever the cause, hyperbaric oxygen therapy (HBO_2T) is the definitive treatment for this disorder [4].

We have a small but expressive experience with four cases of AGE in cardiac surgery and CBP in which, after HBO_2T, all patients had complete recovery from their neurological acute deficits.

Case number 1

The patient was a 62-year-old white male, addicted to cigarette smoking, with a medical history of Type II hyperlipoproteinemia, coronary insufficiency and who suffered a myocardial infarction six months earlier.

The patient was considered eligible for myocardial revascularization.

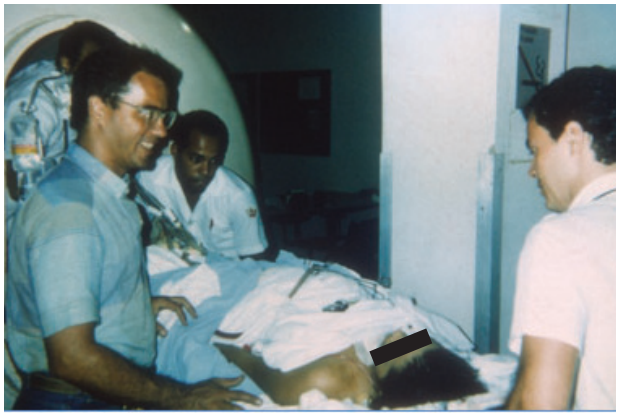
His Doppler ultrasonography showed that both carotids were free of obstructions or stenosis. According to his Doppler echocardiography his cardiac chambers' dimensions and global systolic function were normal, no hemodynamic imbalance was detected, but apex septal hypokinesia was revealed.

The patient's ECG showed left anterior hemiblock and anteroseptal inactive area. His blood tests and chest X-ray were normal.

Myocardial revascularization was performed, with bypass graft from the internal thoracic artery to the left anterior descending coronary artery and a saphenous vein graft to the right coronary artery – with no evident abnormality. Eighteen hours after the end of the surgery the patient complained of tinnitus, dizziness and left hemiparesis. Within minutes he became torporous and left-hemiplegic with facial asymmetry. At this very moment it was noted the presence of air bubbles inside the percutaneous left atrium catheter; this was considered the cause of air embolization.

The patient was referred to the hyperbaric chamber (six-patient Brazilian multiplace chamber, Marsh Engineering, 1984) of our hospital (Brazilian Navy Hospital Marcílio Dias) one hour and thirty minutes after the diagnosis of AGE. He received a total of three HBO_2 treatments (2.4 ATA for 90 minutes with regular five-minute air breaks every 30 minutes), the first treatment as soon as he was admitted at the Diving and Hyperbaric Medicine Service (DHMS) and the second and third treatments every other day.

FIGURE 1



Just after each treatment he was examined by the DHMS staff, and his neurological improvement after each HBO_2 treatment was confirmed. After the third HBO_2T he had no functional neurological deficit.

Case number 2

The patient was a 14-year-old white female who had rheumatic fever when she was 8 years old. She was a patient with mitral stenosis accompanied by mitral and aortic regurgitation and was classified as functional Class III.

Her ECG showed left axis deviation, left atrial overload and left ventricular hypertrophy. Her chest X-ray showed left heart chamber enlargement and pulmonary hypertension. The Doppler echocardiogram confirmed mitral and aortic impairment with severe aortic insufficiency and moderate mitral regurgitation but no stenosis or hemodynamic imbalance.

Aortic valve replacement was performed using a prosthetic heart valve. No other valve replacement was done. During the aortic root aspiration, there had been the accidental introduction of a considerable amount of ambient air. The patient was immediately repositioned with her head below the level of her legs, the air was aspirated through the cannulas as much as possible, and the surgery was concluded.

After her recovery from anesthesia, it became evident that she could not move her right arm or leg. Such evidence of postoperative neurological impairment guided the decision to refer the patient to our Diving and Hyperbaric Medicine Service (DHMS) at the Brazilian Navy Hospital Marcílio Dias just four hours after the surgery. In this case, the chosen treatment protocol was the U.S. Navy Table 6A (six-patient Brazilian multi-place chamber, Marsh Engineering, 1984) *(Figure 1, left)*, the first treatment immediately and another on the next day. The patient showed immediate improvement just after the first HBO_2 treatment *(Figure 2, above)* and evident complete neurologic recovery after the second hyperbaric treatment *(Figure 3, below)*. The CT performed 24 hours after this second and last treatment was normal.

FIGURE 2

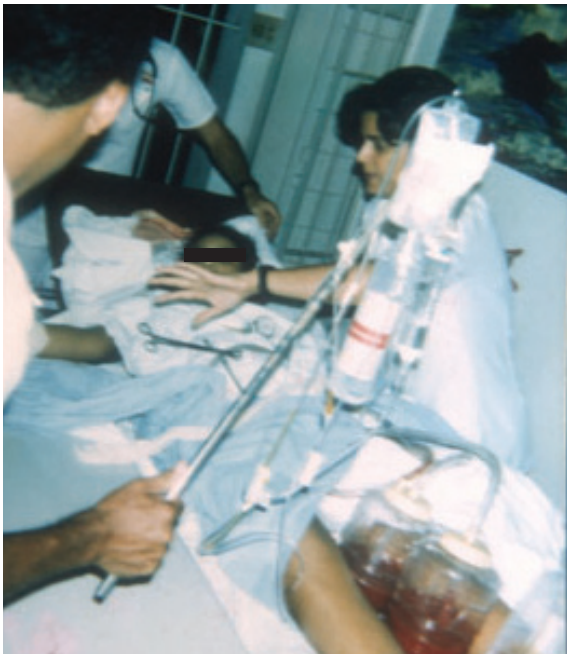


FIGURE 3

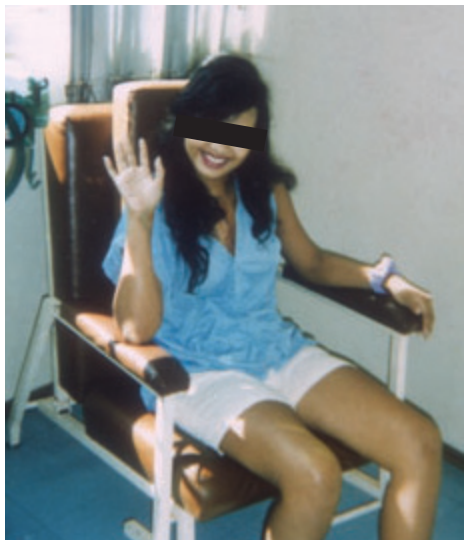


FIGURE 4 – *Case 3*

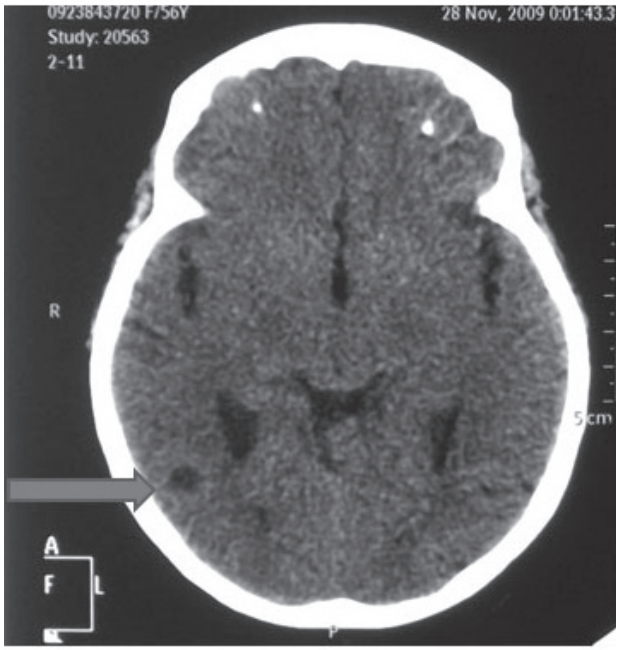
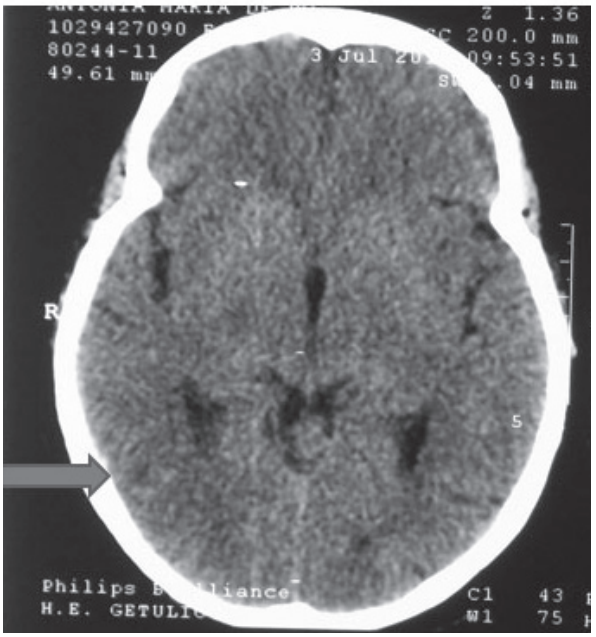


FIGURE 5 – *Case 3*



Case number 3

The patient was a 56-year-old obese white female, addicted to cigarette smoking, with a medical history of hypertension, dyslipidemia and chronic coronariopathy. Doppler ultrasonography showed that carotid artery blood flow was normal bilaterally. Her Doppler echocardiography detected normal ejection fraction, moderate mitral insufficiency, normal heart chamber dimensions and normal pulmonary artery pressure.

ECG and other diagnostic tests were normal. Myocardial revascularization was performed, with coronary artery bypass graft (CABG) from the internal thoracic artery to the left anterior descending coronary artery and with a saphenous vein graft to the right coronary artery.

Extracorporeal circulation lasted 148 minutes, and aortic clamping lasted 120 minutes. Noradrenaline (2mg/kg/minute) was infused over four hours after surgery, and endotracheal extubation was performed six hours later. On the next morning the patient was left-sided hemiplegic. Two hours later the computerized tomography scan of the brain confirmed the suspicion of cerebral arterial air embolism *(Figure 4, above)*. The patient was immediately referred to the hyperbaric chamber (monoplace Sechrist model 2500B) and treated with just one HBO_2 treatment (2.4 ATA for 90 minutes, 100% oxygen). At the end of this single treatment she had a normal neurological exam, actively moving her left arm and leg. We came to the conclusion that a second HBO_2T was not necessary. On the sixth day after the surgery, the patient was discharged from the postoperative unit with normal neurological condition and a normal CT *(Figure 5, above)*.

Case number 4

The patient was a 32-year-old male, addicted to alcohol ingestion and to cigarette smoking. He had a a medical history of chronic renal insufficiency, chronic atrial fibrillation and severe mitral regurgitation. The transesophageal echocardiogram (TEE) detected distension of left cardiac chambers, left ventricle ejection fraction of 70% and pulmonary artery pressure of 40mmHg. The TEE also detected combined mitral stenosis and regurgitation and mitral orifice area of 0.7 cm^2. A mural thrombus was detected in the left atrium. Atrial fibrillation and left ventricular overcharge were evident on ECG.

The preoperative blood tests were normal except for blood urea nitrogen and serum creatinine levels. Replacement of mitral valve with a mechanical prosthesis and thrombectomy were performed. CPB lasted 80 minutes, and aortic clamping lasted 75 minutes. Eight hours later a second surgery was necessary because of excessive postoperative bleeding, and after the second surgical intervention he was admitted to the Intensive Care Unit

FIGURE 6 – ***Case 4***

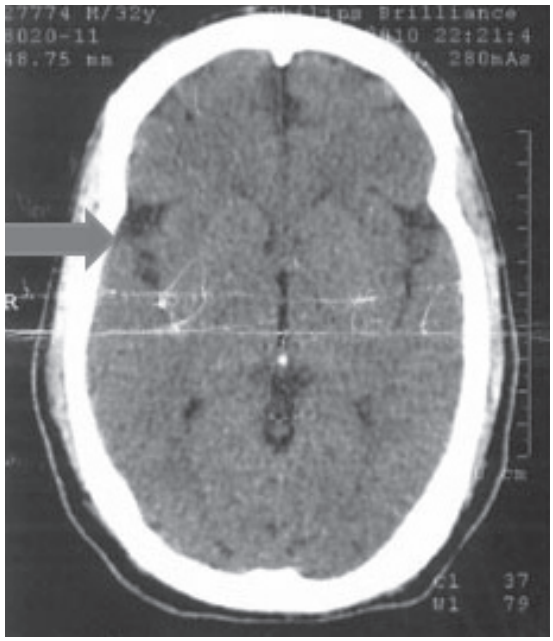
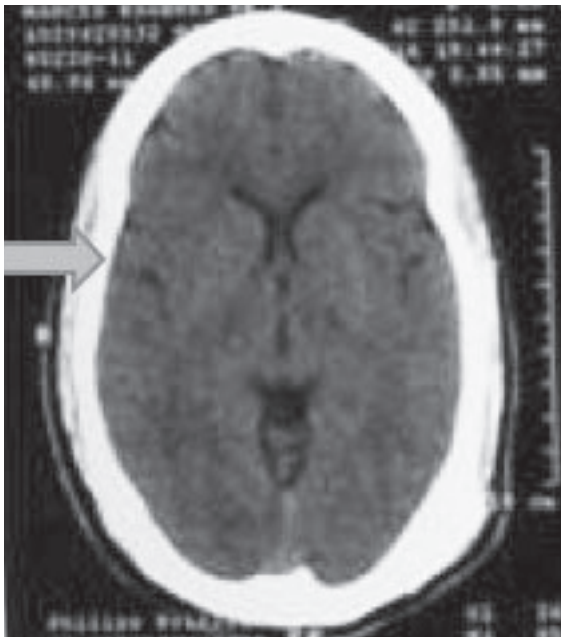


FIGURE 7 – ***Case 4***



(ICU) hemodynamically stable and maintaining sinus rhythm. Endotracheal extubation was performed six hours later. By 16:00 p.m. the next day, the patient was right-sided hemiparetic. The computerized tomography scan of the brain (CT) performed 18 hours after his neurologic deficit was observed was highly suggestive of cerebral arterial air embolism *(Figure 6, above left)*.

On the next morning, the patient was right-sided hemiplegic with left facial deviation, and ECG showed atrial fibrillation. Amiodarone infusion was initiated and the patient was referred for HBO_2T 24 hours after his neurologic deficits became evident. He was treated in a monoplace Sechrist model 2500B. Just after the first HBO_2 treatment (2.4 ATA for 90 minutes 100% oxygen) there was no evidence of his right arm motor or sensory impairment, but he still had a slight paraparesis of his right leg.

On the following day the patient was recompressed with a second HBO_2T in the same chamber and with the same protocol. He experienced total remission of any deficit of his right leg after 60 minutes of treatment in the chamber. 24 hours later – 72 hours after his first HBO_2 treatment, the patient was discharged from the ICU with no detectable neurologic deficit. A new CT performed the same day was normal *(Figure 7, above right)*.

DISCUSSION

Cerebral embolism by great volume of air is a rare complication in cardiac surgery with extracorporal circulation [6,7], but micro cerebral air embolisms with mild or temporary neurological deficits may be difficult to identify and are probably underestimated [8,2].

Air bubbles reaching the vascular endothelium cause typical foreign body reaction with inflammation, edema, vasoconstriction and perfusion disturbances [8,9,10]. When cerebral arteries are affected by air bubbles, peripheral and central neurologic deficits may be seen, *e.g.*, hemiparesis, hemiplegia, hemi-hypoesthesia, hemianopsia, aphasia, encephalopathy, seizures, coma or even death [11].

Several causes may be accounted for iatrogenic cerebral air embolism such as intra-aortic cuff rupture, arterial catheterization, cardiopulmonary resuscitation, endotracheal extubation, and cardiac ablation [20,21, 22, 23].

There are other occasions when some air can be introduced – as during central venous catheterization reaching the arterial circulation through a patent foramen ovale. This affects about 30% of the adult population of the United States. When these air bubbles reach the left side of the heart they can cause what is called paradoxical embolism [30].

The exact amount of gas necessary to cause symptoms is not known, but it has been estimated that the endovenous introduction of 50cc of air may be fatal, while 0.3cc per kg per minute was well tolerated in experiments with dogs [3,9,10].

Routinely, prophylactics measures are taken in order to avoid the entrance of air bubbles in the arterial line during CBP, *e.g.*, maintaining a safe volume of blood in the oxygenator reservoir and monitoring the arterial line filters. Unfortunately, sometimes prophylactic measures are not enough to prevent the formation and circulation of air bubbles; and even micro bubbles, independently of their amount and volume, have the potential to disturb the hematoencephalic perfusion. [8,24].

The immediate treatment for paradoxical cerebral air embolism is the Duran's maneuver: The patient is laid on the left side with the head below the level of the legs and a swan-ganz catheter shall be passed to aspirate the pulmonary artery and superior vena cava. But this and other maneuvers are not enough to deal with bubbles within the cerebral circulation.

There are several case reports and great number of publications describing the favorable results of HBO_2T in AGE affecting divers, but until now there is no established ideal protocol for iatrogenic AGE. In these four cases we have had the opportunity to try three different HBO_2T protocols, reaching the same results. This should be an indication that there is something to be learned about the relationship of AGE, time interval from the incident to the treatment and HBO_2T protocols.

In our small but very expressive experience, four patients who were seriously compromised by AGE just after major heart surgeries recovered their normal neurologic functions with no more than three, in the worse case, hyperbaric oxygen treatments.

■

REFERENCES

1. Sharkey S. Current indications for hyperbaric oxygen therapy. ADF Health. Apr00;(1-2):64-72.
2. S Kol, R Ammar, G Weisz and Y Melamed. Hyperbaric oxygenation for arterial air embolism during cardiopulmonary bypass. Ann Thorac Surg. 1993;55:401-403; Israeli Naval Hyperbaric Institute.
3. Scruggs JE, Joffe A,Wood KE. Paradoxical air embolism successfully treated with hyperbaric oxigen. J Intensive Care Med. 2008 May-Jun;23(3):204-9.
4. Calverley RK, Dodds WA, Trap WG, Jenkins LC. Hyperbaric treatment of cerebral air embolism: a report of case following cardiac catheterization. Can Anaesth Soc. J. 1971 Nov;18(6):665-74.
5. Martins GF, Felipe HS, Silva OG, Melo JCM, Pagano ER. Medicina hiperbárica no tratamento do AVE embólico no pós operatório de cirurgia cardíaca. Arq Med Naval; MB. 1992(3):48-53.
6. Avishai Z, Yochai A, Haim L, Avi S. Hyperbaric oxygen therapy for massive arterial air embolism during cardiac operations. J Thorac Cardiovasc Surg.1999;117:818-821.
7. Stefan H, Bruno R, Heinrich EM, Helfried M, Freyja M, Smolle J. Successful treatment of massive arterial air embolism during open heart surgery. Ann Thorac Surg. 2000;69:931-933.
8. Lélis RGB; Auler JOC. Lesão neurológica em cirurgia cardíaca: Aspectos fisiopatológicos. Rev Bras Anestesiol. 2004; 54(4)607-617.
9. DJ Emby, K Ho. Air embolus revisited - a diagnostic and interventional radiological perspective (bubble trouble and the dynamic Mercedez Benz sign). SA Journal of radiology Vol 10, No 1, March. 2006:3-7.
10. Muth CM, Shank ES. Gas embolism. N Engl J Med. 2000; 342:476-482.
11. Hawes J, Massey W. Neurologic injuries from scuba diving. Neurol Clin. 2008 Feb;26(1):297-308.
12. Cruz-Flores S, Diamond AL, Leira EC. Cerebral air embolism secondary to intra-aortic balloon pump rupture. Neurocrit Care. Vol 2, No 1, 2005;49-50.
13. L Dube, C Soltner, S Daenen, J Lemarieé, P Asfar, P Alquier. Gas embolism: an exceptional complication of radial arterial catheterization. Acta Anaesthesiol Scand. 2004; 48:1208-1210.
14. Yang CW, Yang BP. Massive cerebral arterial air embolism following arterial catheterization. Neuroradiology. 2005; Dec; 47(12):892-894.
15. Hwang SL, Lieu AS, Lin CL, Liu GC, Howng SL, Kuo TH. Massive cerebral air embolism after cardiopulmonary resuscitation. Journ Clin Neurosci. 2005;(12)4:468-469.
16. Turley AJ, Thambyrajah J, Clarke FL, Stewart MJ. Paradoxical embolism through a patent foramen ovale: an unexpected complication of tracheal extubation. Anaesthesia. 2005; May;60(5):501-504.
17. Hinkle DA, Raizen DM, McGarvey ML, Líu GT. Cerebral air embolism complicating cardiac ablation procedures. Neurology Maroh 2001; vol. 56 no 6:792-794.
18. Heckmann JG, Lang CJ, Kindler K, Huk W, Erbguth FJ, Neundorfer B. Neurologic manifestation of cerebral air embolism as a complication of central venous catheterization. Crit Care Med. 2000; May;28(5):1621-1625.
19. Opeskin K, Burke MP, Lynch M. Cerebral air embolism due to disconection of a central venous catheter. J Clin Neurosci.1998; Oct; 5(4):469-471.
20. Soderman M, Bystam J. Cerebral air embolism from angiography in a patient with stroke. A case report. Acta Radiol. 2001 Mar;42(2):140-143.

21. Shetty PG, Fatterpekar GM, Manohar S, Sujit V, Varsha J, Zarir U. Fatal cerebral air embolism as a complication of transbronchoscopic lung biopsy: A case report. Australasian Radiology, 2002;45:(2):215-217.

22. Rodriques RM, Saraiva AR, Embolia aérea arterial durante anestesia para marcação percutânea de nódulo pulmonar. Relato de caso. Rev Bras Anestesiol.2003;53(1): 58-62.

23. Gei AF, Vadhera RB, Hankins GD. Embolism during pregnancy: thrombus, air, and amniotic fluid. Anesthesiology Clin North America. 2003 Mar;21(1):165-182.

24. Dexter F, Hindman BJ. Recomendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absortion. Anesth Analg. 1997 Jun;84(6):1203-7.

25. Ellestad MH et al. Hyperbaric oxygen: its application in cardiology: a historical perspective and personal journey. Cardiol Rev. 2009 Nov-Dec;17(6):280-2.

26. Dunbar EM, Fox R, Watson B, Akrill P. Successful late treatment of venous air embolism with hyperbaric oxygen. Postgrad Med J. 1990; Jun;66(776):469-470.

27. Valentino R, Hilbert G, Vargas F, Gruson D. Computed tomographic scan of massive cerebral embolism. Lancet. 2003 May 31;361(9372):1848.

28. Droghetti L, Giganti M, Memmo A, Zatelli R. Air embolism: diagnosis with single-photon emission tomography and successful hyperbaric oxygen therapy. Br J Anaesth. 2002 Nov;89(5):775-8.

29. Groell R, Schaffler GJ, Rienmueller R, Kern R. Vascular air embolism: location, frequency, and cause on electron-beam CT studies of the chest. Radiology. 1997;Feb;202(2):459-62.

30. Kindwall, EP. “Gas Embolism.” Hyperbaric Medicine Practice, 2nd edition., Kindwall, EP and Whelan, HT. Flagstaff, AZ: Best Publishing Co., 1999; 489-505.

❑