

Middle-ear barotrauma after hyperbaric oxygen therapy

JACQUES BESSEREAU ^{1,2}, ALEXIS TABAH ^{2,3}, NICOLAS GENOTELLE ², ADRIEN FRANÇAIS ³,
MATHIEU COULANGE ¹, DJILLALI ANNANE ²

¹ *Hyperbaric Medicine Centre, Pôle RUSH, Sainte-Marguerite Hospital, Marseille, France;*

² *Intensive Care Unit and Hyperbaric Medicine, Raymond Poincaré Hospital, Garches, France;*

³ *INSERM U823; university Grenoble 1 –Albert Bonniot Institute, Grenoble, France*

CORRESPONDING AUTHOR: Dr. Alexis Tabah – atabah@chu-grenoble.fr

ABSTRACT

Background: Middle-ear barotrauma (MEB) is one of the most common side effects of hyperbaric oxygen therapy (HBO₂). The incidence of MEB has been shown to vary between treatment centers and patients. This study was aimed to determine which patients are at high risk of MEB.

Materials and methods: Prospective study including all the patients treated in a multiplace HBO₂ chamber between January and December 2005. Scoring of MEB before and after HBO₂ by otoscopy was performed using the Haines and Harris classification.

Results: We included 130 patients: 53 Males, 37.5 ± 20.5 years old; 76% were treated for CO poisoning, 11% for iatrogenic gas embolism, 12% for decompression sickness and 4% for necrotizing soft tissue infection. 13% were intubated. MEB occurred in 13.6% of the patients (12.4% of the conscious and 24.4% of the intubated patients, *p*=0.26). Risk factors for MEB were: repetitive treatments and difficulties with pressure equalization. There was no influence of age, sex or mechanical ventilation on the occurrence of MEB.

Conclusions: MEB induced by HBO₂ occurred in 13.6% of the patients. There is no difference in incidence when comparing intubated and non-intubated patients. In non-comatose patients, difficulties with equalizing pressure were predictive of MEB.

INTRODUCTION

Hyperbaric oxygenation therapy (HBO₂) is proposed as a treatment for various diseases such as iatrogenic gas embolism (IGE), decompression sickness (DCS), carbon monoxide poisoning, necrotizing fasciitis or wound healing (1). Our HBO₂ facility is located within the medical intensive care unit (ICU) and treats only acute conditions. It is open for emergencies 24 hours a day, seven day a week.

Middle ear barotrauma (MEB) is a known complication of HBO₂ (2). As ICU sedated and ventilated patients are unable to perform a Valsalva maneuver to prevent MEB, they might be at higher risk of MEB. We conducted this study

to know the incidence of MEB in this particular “acute-only” setting and to determine whether sedation and mechanical ventilation are risk factors for MEB.

MATERIALS AND METHODS

This monocentric prospective study included all the patients treated with HBO₂ at our facility from January 2005 to December 2005.

Study treatment

We used a multiplace hyperbaric chamber (CxPRO, COMEX[®], Marseille, France) pressurized with compressed air. HBO₂ was performed as usual by using local dive tables derived from the French

FIGURE 1

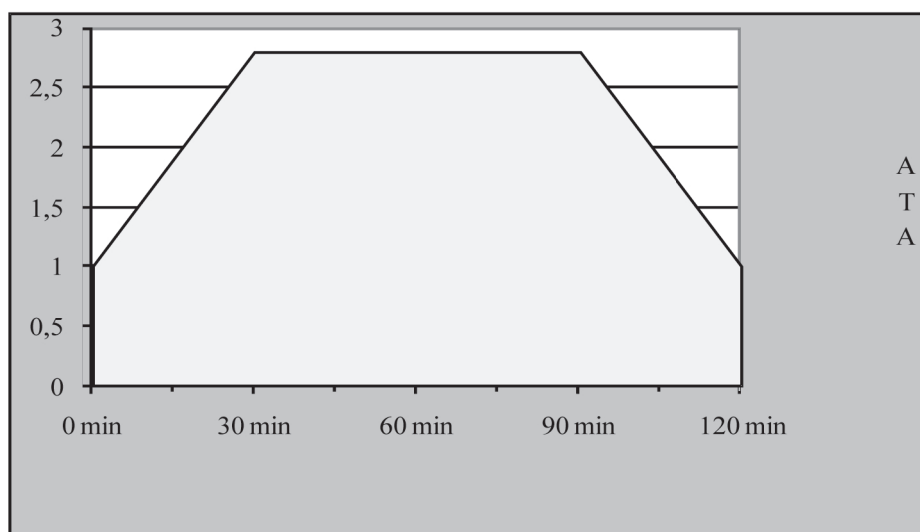


Figure 1. Protocol for DCS and soft tissue infection

FIGURE 2

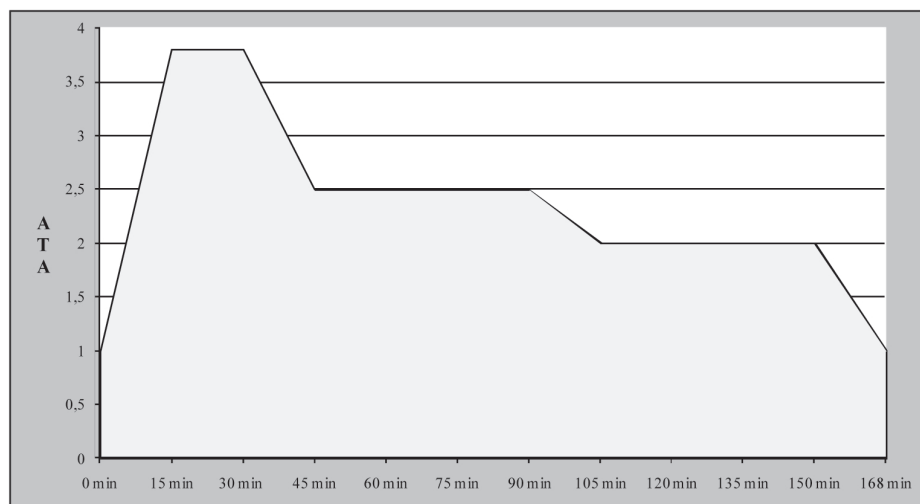


Figure 2. Protocol for iatrogenic gas embolism

COMEX 12, 18 and 30 tables, ratified by a locally recognized hyperbaric engineer. Treatment protocol and depth were determined by the treated condition. We used maximal pressures of 2.0 ATA (202.65 kPa) for carbon monoxide (CO) poisoning, 2.8 ATA for DCS and soft tissue infection (283.71 kPa) and 3.8 ATA (385.03 kPa) for IGE (Figures 1-3, above and facing page).

Accordingly, compression rates were from 3.3 to 18.7 kPa.min⁻¹ (Figures 1-3). We usually perform

one or two HBO₂ treatments for IGE, DCS and CO poisoning, and six to 10 treatments for soft tissue infection. When required, mechanical ventilation with 100% oxygen was achieved using a dedicated pressure-capable ventilator (RCH LAMA[®]). Spontaneously breathing patients received 100 percent oxygen during the entire session via a tight-fitting face mask.

Chemoprophylaxis for hyperoxic seizures was done with diazepam (10mg parenterally) in awake

FIGURE 3

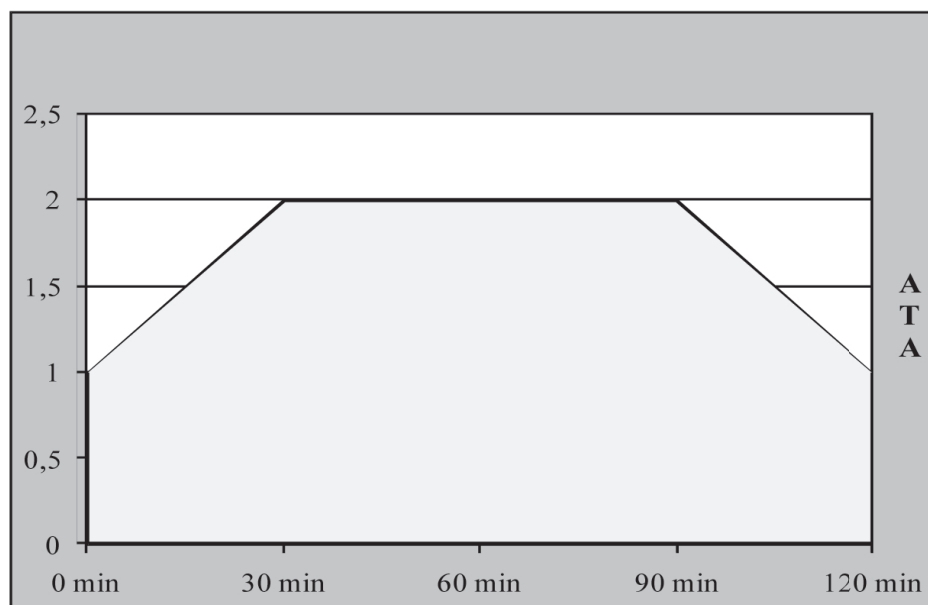


Figure 3. Protocol for carbon monoxide poisoning

patients and midazolam in ventilated patients. All patients were accompanied by a nurse or physician in the chamber for the entire treatment duration.

Conscious patients received a brief education on potential side effects of HBO₂, particularly MEB and the importance of pressure equalization; they were instructed to perform repetitive Valsalva maneuvers during compression and to alert in case of ear pain or discomfort. The attending physician or nurse instructed the patient to repeat such maneuvers at appropriate moments and watched the patient for potential side effects during the entire treatment.

Data collection

Before HBO₂, history of hearing or tympanic-related trouble was recorded. Bilateral otoscopy was performed just before and after each HBO₂ session by a physician trained in grading tympanic lesions with the six grades of the Haines and Harris classification:

- **Grade 0:** normal aspect of the tympanic membrane;

- **Grade 1:** injection of the tympanic membrane, especially along the handle of the malleus;
- **Grade 2:** injection plus slight hemorrhage within the substance of the tympanic membrane;
- **Grade 3:** gross hemorrhage within the substance of the tympanic membrane;
- **Grade 4:** hemorrhage and free blood in the middle ear;
- **Grade 5:** perforation of the tympanic membrane.

MEB was defined as a higher grade of otoscopic lesions after compared with the state of the ear before HBO₂. We defined difficulty clearing ears as any ear pain or discomfort during the compression phase, regardless of the successfulness of the Valsalva maneuvers.

Statistical analysis

All the analyses were done using the SAS Software 9.1.3 (Cary, North Carolina). The relation between potential risk factors and MEB was analyzed using a Chi square test for qualitative variables and a Wilcoxon Rank Sum test for quantitative variables. Results are reported as *n* (%) or mean (standard deviation) when applicable.

TABLE 1

	no MEB	MEB	<i>p</i> -value
ALL 130 PATIENTS	<i>n</i>=112	<i>n</i>=18	
Male sex <i>n</i> (%)	48 (42.9)	5 (27.8)	0.2269
Age, years (+/-SD)	37.2 (19.4)	39.6 (27.2)	0.9785
Mechanical ventilation <i>n</i> (%)	13 (11.6)	4 (22.6)	0.26
Number of repetitive treatments <i>n</i> (+/-SD)	1.3 (1.8)	2.1 (2.6)	0.245
113 CONSCIOUS PATIENTS	<i>n</i>=99	<i>n</i>=14	
Performed a Valsalva successfully <i>n</i> (%)	90 (90.9)	13 (92.9)	0.81
Difficulties with equalizing pressure <i>n</i> (%)	15 (15.1)	8 (57.1)	0.0002

Table 1: Risk factors for MEB in the study population and in the conscious patients group

RESULTS

We included 130 patients: 53 (40.8%) males, mean age 37.5±20.5 years. HBO₂ was performed for carbon monoxide poisoning in 100 patients (76.9%), DCS in 12 (9.2%), necrotizing fasciitis in four (3.1%) and gas embolism in 14 (10.8%). Seventeen (13.1%) required mechanical ventilation at the time of HBO₂; all of them were intubated orotracheally.

Of the 113 conscious patients, 22 (19.6%) reported difficulties for equalizing pressure, of whom eight (36.3%) presented with MEB. MEB was found in 18 (13.6%) patients: in 14 (12.4%) of the conscious patients and in four (23.4%) of the ventilated patients (*Table 1*, above). Of the 14 conscious patients with MEB, six (43%) were asymptomatic. Risk factors for MEB were repetitive treatments and pressure equalization problems (*Table 1*). We found no influence of age, sex or mechanical ventilation on the occurrence of MEB.

DISCUSSION

Middle ear barotrauma is one of the most common side effects of HBO₂ (3); reported incidences range from 8-68.7% and up to 91% in patients unable to auto-inflate their middle ear (4-7).

Known risk factors for MEB include female sex, older age (2), artificial airways (intubation) (8) and history of Eustachian tube dysfunction (9) or the inability to auto-inflate the middle ear (7).

Slower compression (10) is a known protective factor for MEB; in our center, compression rates are below 7 kPa.min⁻¹ (except for IGE), and slowed in the case of pain or discomfort.

Treatments were performed in a multiplace chamber, and patients were accompanied by a chaperone who explained the potential complications of HBO₂. This is reported to reduce the risk of MEB (2). The successful equalization of pressure by inflating the middle ear has been reported to be protective (7), whereas the prophylactic use of nasal decongestants has not (11).

In our study, MEB incidence was 13.6%; all the patients were treated for an emergency, but always received explanations and education on HBO₂ and were taught how to perform a Valsalva maneuver. They were informed about the risks for MEB and told to report to the chaperone any symptoms of ear pain or discomfort. In cases of difficulties with pressure equalization, compression was slowed down and the patient assisted by the chaperone. We believe that it contributed to helping reduce the patient's anxiety and improved his ability to

perform effective pressure equalization. Moreover, the dose of benzodiazepine, which is used to help prevent oxygen toxicity, might have helped in relieving anxiety.

In contrast with previous findings, we found no association between age or gender and MEB. Difficulties with equalizing pressure were associated with MEB, while patients who did or did not perform a Valsalva had the same MEB incidence. This could be explained by a number of patients performing ineffective Valsalvas, who would be at higher risk for MEB; this is on par with previous findings on Eustachian tube function (9) and higher risk of MEB in patients unable to auto-inflate the middle ear (7).

Repetitive treatments increased the risk for MEB, whereas the treated condition (which determined treatment depth) did not influence MEB incidence.

In 1994 Presswood reported that 94% of the intubated patients developed a middle ear complication, and 61% required placement of tympanostomy tubes (8). We found that mechanical ventilation was not associated with an increased incidence of MEB, which might be explained by deep sedation of the patients while HBO₂ was performed, as it may have helped peristaphylin muscle relaxation and unconscious pressure equalization. Other factors explaining this difference might be that the Presswood series included patients treated for head and neck surgical and radiation side effects, whereas our series included only emergency conditions without any patient specifically treated for a head and neck condition.

CONCLUSION

Difficulties in pressure equalization and repetitive treatments were the only risk factors for MEB. We believe that slower compression rates, better patient education and follow-up during the treatment to quickly detect any pressure equalization problem can be effective in reducing the incidence of MEB.

Acknowledgments

We want to thank the following persons for their participation in the study:

Martine Dhalluin-Brame, Katrine Pecout, Christian Dubois, Michel Le Bescont, Michel Roustan, Pascal Wawrzyniak (nurses) and Charles Devere (M.D.).

REFERENCES

1. Tibbles, P.M. and J.S. Edelsberg. Hyperbaric-oxygen therapy. *N Engl J Med*, 1996. 334(25): p. 1642-8.
2. Fitzpatrick, D.T., B.A. Franck, K.T. Mason, and S.G. Shannon. Risk factors for symptomatic otic and sinus barotrauma in a multiplace hyperbaric chamber. *Undersea Hyperb Med*, 1999. 26(4): p. 243-7.
3. Plafki, C., P. Peters, M. Almeling, W. Welslau, and R. Busch. Complications and side effects of hyperbaric oxygen therapy. *Aviat Space Environ Med*, 2000. 71(2): p. 119-24.
4. Blanshard, J., A. Toma, P. Bryson, and P. Williamson. Middle ear barotrauma in patients undergoing hyperbaric oxygen therapy. *Clin Otolaryngol Allied Sci*, 1996. 21(5): p. 400-3.
5. Karahatay, S., Y.F. Yilmaz, H. Birkent, H. Ay, and B. Satar. Middle ear barotrauma with hyperbaric oxygen therapy: incidence and the predictive value of the nine-step inflation/deflation test and otoscopy. *Ear Nose Throat J*, 2008. 87(12): p. 684-8.
6. Igarashi, Y., Y. Watanabe, and K. Mizukoshi. Middle ear barotrauma associated with hyperbaric oxygenation treatment. *Acta Otolaryngol Suppl*, 1993. 504: p. 143-5.
7. Beuerlein, M., R.N. Nelson, and D.B. Welling. Inner and middle ear hyperbaric oxygen-induced barotrauma. *Laryngoscope*, 1997. 107(10): p. 1350-6.
8. Presswood, G., W.A. Zamboni, L.L. Stephenson, and P.M. Santos. Effect of artificial airway on ear complications from hyperbaric oxygen. *Laryngoscope*, 1994. 104(11 Pt 1): p. 1383-4.

9. Fernau, J.L., B.E. Hirsch, C. Derkay, S. Ramasastry, and S.E. Schaefer. Hyperbaric oxygen therapy: effect on middle ear and eustachian tube function. *Laryngoscope*, 1992. 102(1): p. 48-52.
10. Vahidova, D., P. Sen, M. Papesch, M.P. Zein-Sanchez, and P.H. Mueller. Does the slow compression technique of hyperbaric oxygen therapy decrease the incidence of middle-ear barotrauma? *J Laryngol Otol*, 2006. 120(6): p. 446-9.
11. Carlson, S., J. Jones, M. Brown, and C. Hess. Prevention of hyperbaric-associated middle ear barotrauma. *Ann Emerg Med*, 1992. 21(12): p. 1468-71.

