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# SELECTION CRITERIA UTILIZED FOR HYPERBARIC OXYGEN TREATMENT OF CARBON MONOXIDE POISONING

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□ Abstract – Medical directors of North American hyperbaric oxygen (HBO) facilities were surveyed to assess selection criteria applied for treatment of acute carbon monoxide (CO) poisoning within the hyperbaric medicine community. Responses were received from 85% of the 208 facilities in the United States and Canada. Among responders, 89 monoplace and 58 multiplace chamber facilities treat acute CO poisoning, managing a total of 2,636 patients in 1992. A significant majority of facilities treat CO-exposed patients with coma (98%), transient loss of consciousness (LOC) (77%), ischemic changes on electrocardiogram (91%), focal neurologic deficits (94%), or abnormal psychometric testing (91%), regardless of carboxyhemoglobin (COHb) level. Although 92% would use HBO for a patient presenting with headache, nausea, and COHb 40%, only 62% of facilities utilize a specified minimum COHb level as the sole criterion for HBO therapy of an asymptomatic patient. When COHb is used as an independent criterion to determine HBO treatment, the level utilized varies widely between institutions. Half of responding facilities place limits on the delay to treatment for patients with only transient LOC. Time limits are applied less often in cases with persistent neurologic deficits. While variability exists, majority opinions can be derived for many patient selection criteria regarding the use of HBO in acute CO poisoning.

□ Keywords – carbon monoxide; poisoning; hyperbaric oxygen therapy

## **INTRODUCTION**

Inhalation of oxygen is the primary medical therapy for the patient suffering from acute carbon monoxide (CO) poisoning. Hyperbaric oxygen administration serves to enhance the rate of clearance of CO from hemoglobin and tissue, can improve tissue oxygenation by improving peripheral oxygen delivery, and has the potential to reduce cerebral edema (1). Most physicians agree that hyperbaric oxygen (HBO) is appropriate for severe cases of acute CO poisoning. The specific definition of severe poisoning, however, remains controversial within the general medical community. As a result, some disagreement exists as to which patients should be referred for HBO (2-4).

It would be expected that one could turn to the hyperbaric medicine community for consensus recommendations regarding selection criteria for HBO treatment of patients with acute CO poisoning. The Hyperbaric Oxygen Committee of the Undersea and Hyperbaric Medical Society (UHMS) provides guidance in this area, recommending that CO-intoxicated patients with alteration in mental status, neurological signs, cardiovascular dysfunction, pulmonary edema, severe acidosis, or any interval of unconsciousness be referred for HBO therapy (5). In addi-

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RECEIVED: 1 July 1994; FINAL SUBMISSION RECEIVED: 16 September 1994; ACCEPTED: 28 September 1994 tion, the Committee notes in the same report that psychometric testing may be helpful for detection of subtle central nervous system involvement, and that efficacy of HBO is diminished when administered greater than 6 hours after the patient is discovered. Finally, the Committee suggests that treatments be performed at 2.4 to 3.0 atmospheres absolute (ATA), with daily retreatment of patients with severe neurological dysfunction until additional improvement is not seen.

Although these recommendations may appear clear, it is apparent that variation exists in clinical hyperbaric medicine practice with regard to the patient selection criteria utilized for this disease. The present study was performed in an attempt to define the degree of that variation and to determine where majority opinions regarding selection criteria may exist.

#### METHODS

Study data were obtained through a mail survey sent to medical directors of all 208 North American monoplace and multiplace hyperbaric facilities listed in the 1992 UHMS directory (6). Two repeat mailings were performed as necessary to achieve a high response rate.

Hyperbaric medical directors were initially asked whether acute carbon monoxide poisoning is treated at their facility. If so, they were asked to complete the remainder of the questionnaire. Directors were questioned regarding their facility's treatment experience for CO intoxication in calendar year 1992. Information was also collected regarding the selection criteria utilized to determine whether a patient with acute CO poisoning receives hyperbaric oxygen treatment. Survey questions addressed three general categories. First, the importance of signs or symptoms demonstrating end organ (central nervous system or heart) effect of CO was evaluated. A second group of questions focused on the patient's carboxyhemoglobin (COHb) level. The final area addressed was the issue of temporal delay, both with regard to delay to treatment in acute poisoning and the delayed onset of neurologic symptoms following acute poisoning.

Summed responses were analyzed for all treating facilities as a group. Excluded from data analysis were nonresponding facilities and those facilities that do not use their hyperbaric chamber to treat acute CO poisoning.

### RESULTS

Survey responses were received from 176 (85%) of the 208 North American hyperbaric facilities listed in the 1992 UHMS directory. Among responders, 58 multiplace and 89 monoplace facilities reported that they utilize their hyperbaric chamber for treatment of acute carbon monoxide poisoning. In the calendar year 1992, multiplace chambers were used to treat 1,398 CO-intoxicated patients, with individual facilities treating 0 to 161 patients (mean 24 patients per facility). Monoplace chambers treated a total of 1,238 patients in 1992, with individual facilities treating 0 to 112 patients (mean 14 patients per facility). Combining data from monoplace and multiplace facilities, 2,636 total patients were treated at 147 centers, averaging 18 patients per facility in that year.

With regard to selection criteria applied to determine whether a CO-poisoned individual receives HBO treatment, survey questions first addressed the patient presenting with evidence of end organ effect of CO intoxication. Summarized responses to these questions are provided in Table 1. A significant majority of facilities use HBO to treat CO-exposed patients with coma (98%), focal neurologic deficits (94%), ischemic changes on electrocardiogram (91%), abnormal psychometric testing (91%), or transient loss of consciousness (LOC) who awaken before arrival in the emergency department (77%), regardless of carboxyhemoglobin level. Half of the facilities (48%) would use HBO for an individual not experiencing loss of consciousness but presenting with COHB 9.5%, dizziness, and headache. Only a small minority (7%) would consider HBO for the same individual (no LOC, COHb 9.5%) who presented with no other signs or symptoms of poisoning.

With regard to the COHb level, 135 of 176 treating facilities (92%) would use HBO for the patient presenting with headache, nausea, and COHb level of 40% (Table 1). Only 91 facilities (62%), however, utilize a specified minimum COHb level as the sole criterion for HBO therapy of an asymptomatic patient. Among those hyperbaric physicians who utilize COHb level as an independent criterion for HBO treatment, minimum levels required for treatment range from 5 to 40%, with 20 or 25% being used by 65% of physicians (Figure 1).

Half of responding HBO medical directors place limits on the time delay allowed prior to hyperbaric treatment of patients with only transient loss of consciousness. Among those who would deny HBO in cases of delay, time limits applied vary from 1 hour to 28 days (most commonly 12 hours, Figure 2). Such limits are less consistently applied in cases with persistent neurologic deficits or coma. Only 23% of medical directors utilize time criteria to determine eligibility for HBO treatment of the patient presenting with neurological signs associated with CO poisoning. Where limits are used, intervals ranging from 6 hours to 56 days are applied (most commonly 24 hours).

#### Table 1. Importance of CO End Organ Effect in Determining Need for HBO Therapy

Would you routinely use HBO treatment for the following adult patients with CO poisoning, presenting less than 3 hours after exposure?

	Yes	No	No Response
Arrives at emergency department unconscious with COHb 9.5%	144 (98%)	2 (1%)	1 (1%)
Initially unconscious upon CO exposure, arriving at the emergency department awake and asymptomatic, with normal neurologic examination and COHb 9.5%	114 (77%)	29 ( <b>20%</b> )	4 (3%)
History of CO exposure, no loss of consciousness, COHb 9.5%, and the following:			
No symptoms or signs	10 (7%)	133 (90%)	4 (3%)
Headache and dizziness only	71 (48%)	70 (48%)	6 (4%)
ECG suggesting acute ischemia	134 (91%)	9`(6%)	4 (3%)
Focal neurologic abnormality on physical examination	138 (94%)	4 (3%)	5 (3%)
Abnormal psychometric testing	134 (̀91%)́	4 (̀3%)́	8 (5%)
No loss of consciousness, presenting with headache, nausea, COHb 40%, and normal neurologic examination, ECG, and neuropsychiatric testing	135 (̀92%́)	9 (6%)	3 (2%)

Approximately two-thirds (68%) of survey responders utilize HBO to treat patients with neurologic or neuropsychiatric sequelae developing in a delayed fashion after CO intoxication. Time limits applied to such delayed treatment were not studied.

## DISCUSSION

It has been previously reported that there is tremendous variation in the hyperbaric treatment protocols utilized for acute CO poisoning among multiplace hyperbaric chamber facilities in North America (7). A multitude of HBO protocols are in use, none of which is utilized for a majority of the patients treated. In addition to variations in the pressure applied and time of oxygen administration, there also exist differences in opinion with regard to retreatment and the number of repeat treatments given (7).

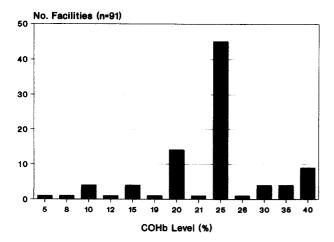


Figure 1. Carboxyhemoglobin (COHb) levels utilized to determine need for hyperbaric oxygen therapy among the 91 facilities using COHb level as an independant indication for treatment. The present study demonstrates that similar variation exists within the hyperbaric medical community with regard to some patient selection criteria used to determine the need for HBO treatment of this disease, and that consensus exists for a number of other selection criteria.

Approaches to selection criteria are most similar when dealing with the more severely poisoned patient. As noted in Table 1, a significant majority of medical directors would recommend HBO for the patient demonstrating significant end organ effect of the poisoning. Depending upon the specific condition, 77 to 98% would use HBO for the patient with coma, transient loss of consciousness, focal neurologic deficit on examination, abnormal neuropsychiatric testing, or cardiac ischemia, despite a low COHb level. Even after accounting for the 15% of facilities that did not respond to the survey, these can be considered indications for HBO by a majority of North American hyperbaric medical directors. Data

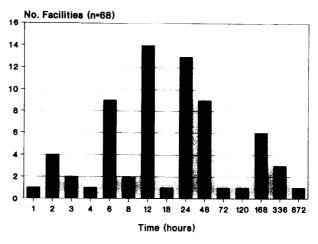


Figure 2. Limits on the time delay from CO exposure to hyperbaric treatment allowed in cases with only transient loss of consciousness (among 74 facilities applying such limits).

from published studies support the use of HBO in severe CO poisoning such as this, with lower mortality and morbidity among those who receive HBO over normobaric oxygen (8-10).

The role of the carboxyhemoglobin level in determining which cases of CO poisoning warrant hyperbaric treatment is less clear. Nearly all treating facilities would use HBO for a poisoned patient with COHb 40%, headache, and nausea. Only two-thirds, however, identify COHb level as an independent criterion for the HBO treatment of an asymptomatic patient. Therefore, although the majority of medical directors use HBO for the patient with a specified minimum COHb level regardless of apparent end organ effect, some also require symptomatic manifestation of the poisoning for HBO treatment. It would appear that manifestations of headache or nausea are considered sufficient symptoms by these physicians to administer HBO.

When the COHb level is applied as an independent indication for HBO therapy, the range of values utilized is quite wide (Figure 1). A carboxyhemoglobin level of 25% was identified most often by survey responders, but this value is used by only half of those applying COHb as a sole criterion for treatment. It is not possible to draw firm conclusions from the published clinical literature with regard to the role of COHb in determining need for HBO therapy. The UHMS Hyperbaric Oxygen Therapy Committee notes only that the "body of clinical data available suggests that functional testing, rather than a carboxyhemoglobin level, may be a more appropriate method for determining the need for HBO therapy among minimally symptomatic patients" (5).

It has been previously demonstrated that effectiveness of HBO therapy for CO poisoning decreases with duration of delay to treatment. In the classic study by Goulon, HBO treatment within 6 hours of victim discovery resulted in 13.5% mortality, while those receiving HBO more than 6 hours after being found had a 30.1% mortality rate (11). In a 1991 analysis of 187 patients treated at a North American hyperbaric facility, interval from CO exposure to HBO treatment was also found to correlate significantly with the chance for residual neurologic deficits after treatment (12).

Neither these nor other studies, however, have precisely defined time limits beyond which HBO therapy is likely to be ineffective. This is the probable explanation for the diversity in approach seen among those responding to the present survey. One-half of medical directors use a time limit to deny HBO treatment to a patient with only transient loss of consciousness. In the CO-poisoned patient presenting with focal neurologic findings, time limits are applied by only one-quarter of directors. When limits are applied in such instances, intervals ranging from 6 to 48 hours are most commonly used, but delays of 1 to 2 months are allowed by some physicians (Figure 2).

It is interesting that some who would deny HBO to the patient initially presenting with focal neurological signs because of excessive delay to treatment would utilize HBO for symptoms developing in a delayed fashion. A majority of responding medical directors reported that they utilize HBO to treat patients with delayed development of neurologic or neuropsychiatric sequelae after CO intoxication. Published data regarding the efficacy of such treatment is contradictory (8,13).

Discrepancy with regard to treatment of pregnant patients with CO poisoning has previously been reported (7). In that survey of North American multiplace hyperbaric facilities, 74% have treated or would treat pregnant patients with significant CO intoxication. One-quarter do not use HBO for pregnant CO-poisoned patients, despite a lack of data demonstrating increased risk from such treatment and recommendations from authors in both the United States and Europe that such patients be treated (14,15).

Future studies are needed to resolve the controversies that remain with regard to the hyperbaric management of the CO-poisoned patient. The variability in patient selection criteria demonstrated in the present study complicates comparison of results from different hyperbaric centers. These problems are only compounded by differences in the hyperbaric treatment profiles utilized. Randomized clinical trials, possibly multicentered, could study those categories of CO-poisoned patients for whom only a minority of hyperbaric medical directors currently apply HBO therapy in an attempt to determine whether such patients benefit from HBO treatment. In addition, HBO treatment itself could be standardized by randomizing patients treated by a majority of directors to the various HBO treatment protocols currently in use.

The variability in clinical HBO practice attests to the fact that the optimal approach to this disease is currently unknown. Through systematic study of the problem, it should be possible to define an appropriate standard of care. Until this is done, it is likely that some patients who may benefit from HBO will not be referred for treatment, and others will receive HBO therapy with little likelihood of additional benefit beyond that offered by normobaric oxygen.

#### REFERENCES

- 1. Piantadosi CA. Carbon monoxide intoxication. In: Vincent JL, ed. Update in intensive care and emergency medicine. New York: Springer Verlag NY Inc.; 1990;10:460-71.
- Olson KR. Carbon monoxide poisoning: Mechanisms, presentation, and controversies in management. J Emerg Med. 1984; 1:233-43.
- 3. Kindwall EP. Hyperbaric treatment of carbon monoxide poisoning. Ann Emerg Med. 1985;14:1233-4.
- 4. Haddad LM. Carbon monoxide poisoning: To transfer or not to transfer? Ann Emerg Med. 1986;15:1375.
- 5. Hyperbaric oxygen therapy: A committee report. Bethesda: Undersea and Hyperbaric Medical Society; 1992:12-13.
- 6. Hyperbaric Chambers, United States and Canada: A directory of hyperbaric treatment chambers. Bethesda: Undersea and Hyperbaric Medical Society; 1992.
- Hampson NB, Norkool DM, Dunford RG. Treatment of carbon monoxide poisonings in multiplace hyperbaric chambers. J Hyperbaric Med. 1992;7:165-71.
- Myers RAM, Snyder SK, Emhoff TA. Subacute sequelae of carbon monoxide poisoning. Ann Emerg Med. 1985;14: 1163-7.
- 9. Norkool DM, Kirkpatrick JN. Treatment of acute carbon monoxide poisoning with hyperbaric oxygen: A review of 115 cases. Ann Emerg Med. 1985;14:1168-71.

- Krantz T, Thisted B, Strom J, Bredgaard Sorensen M. Acute carbon monoxide poisoning. Acta Anaesthesiol Scand. 1988; 32:278-82.
- 11. Goulon M, Barois A, Rapin M, Nouailhat F, Grosbuis S, Labrousse J. Intoxication oxycarbonee at anoxie qique par inhalation de gay de charbon et d'hydrocarbures. Ann Med Interne (Paris). 1969:120:335-49. English translation: J Hyperbaric Med. 1986;1:23-41.
- Skeen MB, Massey EW, Moon RE, Shelton DL, Fawcett TA, Piantadosi CA. Immediate and long term neurological sequelae of carbon monoxide intoxication. Undersea Biomed Res. 1991;18(Suppl):36.
- Hopkins RO, Weaver LK. Does late repetitive hyperbaric oxygen improve delayed neurologic sequelae associated with carbon monoxide poisoning? Undersea Biomed Res. 1991; 18(Suppl):34.
- 14. Van Hoesen KB, Camporesi EM, Moon RE, Hage ML, Piantadosi CA. Should hyperbaric oxygen be used to treat the pregnant patient for acute carbon monoxide poisoning? JAMA. 1989;261:1039-43.
- Raphael JC, Jars-Guincestre MC, Gajdos P. Prise en charge des intoxications oxycarbonées aiguës: Oxygène normobare ou hyperbare. Rev Prat. 1993;43:604-7.