Intracranial Pressure Responses During Hyperbaric Oxygen Therapy

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Abstract

The responses of intracranial pressure (ICP) to hyperbaric oxygen (HBO) therapy and arterial gas pressures were investigated. ICP was measured through a ventricular or spinal drainage catheter in patients with brain tumor or cerebrovascular disease. Changes in ICP, heart rate (HR), arterial blood pressure (ABP), and transcutaneous partial pressure of carbon dioxide (PtcCO₂) or oxygen (PtcO₂) were recorded continuously during air or 100% O₂ breathing at 1 and 2.5 atmospheres absolute (ATA). HR and PtcCO₂ decreased and mean ABP was unchanged during HBO inhalation. ICP was reduced at the beginning and tended to increase gradually during HBO inhalation. The change from air to O₂ without altering respiratory frequency and volume caused a gradual increase of ICP and PtcCO₂ with a transient ICP reduction in an artificially respired patient. Intentionally reduced respiration to maintain PtcCO₂ at the value at 2.5 ATA with air caused the ICP to return to near the value at 2.5 ATA with air even during HBO inhalation. These findings suggest that reduced ICP is initially due to direct cerebral vasoconstriction caused by hyperoxia and is maintained mainly by induced hypocapnia during HBO inhalation. Care is required when giving HBO therapy to patients with a high ICP and/or who are respired artificially.

Key words: blood gases, carbon dioxide, hyperbaric oxygen, intracranial pressure

Introduction

Cerebral vasoconstrictive effects of hyperbaric oxygen (HBO) have been demonstrated in both clinical and experimental investigations.^{6,9,10,12,14–16,21} HBO-induced reduction of cerebral blood flow (CBF) and increased intracranial pressure (ICP) is beneficial in various neurological conditions when brain edema is present.^{11,18–20} ICP reduction during HBO inhalation is apparently caused by vasoconstriction, but whether the effect is directly due to HBO or to hypocapnia secondary to HBO is unknown. We, therefore, continuously recorded partial pressure of both oxygen (PO₂) and carbon dioxide (PCO₂) using transcutaneous devices during 50-minute HBO therapy to investigate the effects of arterial gases on ICP.

Materials and Methods

The ICP and transcutaneous PO₂ (PtcO₂) and PCO₂ (PtcCO₂) were investigated in 11 patients with postoperative acute brain damage. Two patients presented with brain tumor, five subarachnoid hemorrhage, one cerebral hemorrhage, one ruptured arteriovenous malformation, and two cerebellar hemorrhage. The patients received external ventricular or spinal drainage and HBO therapy for postoperative neurological impairment (Table 1).

HBO therapy was performed in a multiplace hyperbaric chamber (P-1000SA, Hanyuda, Tokyo). The therapy schedule was 15-minute compression with air, 10-minute air breathing, 50-minute 100% O₂ inhalation using an O₂ mask at 2.5 atmospheres absolute (ATA), 10-minute decompression with O₂ inhalation.

The parameters monitored were PtcO₂, PtcCO₂, heart rate (HR), respiratory pattern and rate (RR). PtcO₂ was monitored by a TCM1 TC oxygen monitor (Radiometer, Copenhagen, Denmark).

Table 1 Clinical summary of the neurosurgical patients treated with HBO

Case No.	Age	Interval from operation to HBO therapy (days)	Diagnosis	Neurological status at HBO therapy	Respiration	Mean ICP at 1 ATA with air (mmHg)
1	59	6	cerebellar hemorrhage	semicoma	spontaneous intubated	8.5
2	31	13	cerebellar hemorrhage	ataxia	spontaneous	9.6
3	59	6	cerebral hemorrhage	coma	artificial (barbiturate therapy)	10.5
4	53	10	brain tumor	ataxia	spontaneous	10.9
5	16	8	brain tumor	hemiparesis	spontaneous	7.6
6	61	14	ruptured AVM	confusion	spontaneous	16.3
7	67	7	subarachnoid hemorrhage	hemiparesis	spontaneous	9.7
8	48	5	subarachnoid hemorrhage	semicoma	spontaneous	11.4
9	49	12	subarachnoid hemorrhage	somnolence	spontaneous	9.1
10	58	11	subarachnoid hemorrhage	somnolence	spontaneous	6.4
11	31	8	subarachnoid hemorrhage	confusion	spontaneous	10.2

ATA: atmosphere absolute, AVM: arteriovenous malformation, HBO: hyperbaric oxygen, ICP: intracranial pressure.

The PtcO₂ sensor was calibrated with ambient air. PtcCO₂ was monitored by a Model 634 (Kontron Medical, Zürich, Switzerland). The PtcCO₂ sensor was stable and accurate during compression to 41 bar and decompression in rats,⁴⁾ and calibrated with two gas mixtures (5% and 10% CO₂: N₂ for balance, Calibrator 344, Kontron Instruments, Ltd., Zürich, Switzerland). Arterial blood pressure (ABP) was monitored using calibrated strain-gauge P-50 transducers (Statham, U.S.A.) through an arterial catheter. The HR was determined from the electrocardiogram. The PtcCO₂ and HR were expressed as mean $\pm$ SD for each patient every 5 minutes. The respiratory pattern was monitored by the impedance method and the RR averaged every 30 seconds.

The ICP was measured through the ventricular or spinal drainage catheter with the patient in the supine position using calibrated strain-gauge transducers referenced to the external auditory meatus. The baseline ICP was measured 1 hour or more before compression, to eliminate any ICP increase due to obliteration of the external cerebrospinal fluid drainage system. All parameters including the maximum and minimum ICP values were recorded continuously by a programmable data logger (7VO7, NEC San-Ei Sokki, Tokyo) every 10 seconds before, during, and after HBO therapy (about 2 hours).

Fig. 1 Changes in mean PtcCO₂ during HBO therapy in all spontaneously respired patients (n = 10). PtcCO₂ decreased gradually during 100% O₂ inhalation at 2.5 ATA. The first significant difference is observed 5-10 minutes after O₂ inhalation. The subsequent PtcCO₂ values with 100% O₂ inhalation are significantly lower than those at 2.5 ATA with air. The lowest value is seen just before the start of decompression (45-50 minutes after O₂ inhalation). PtcCO₂ is raised gradually after decompression. *p < 0.05 and **p < 0.01 vs. 2.5 ATA with air.

The small number of patients and various illnesses prevents statistical analysis for the case studies. The *t* test was used to compare common variables such as HR or PtcCO₂ data. Data were obtained every 10 seconds and averaged over 5 minutes (30 points) or 1 minute (6 points) in each subject. The mean and SD were then calculated. The two variables were correlated by least square linear regression for each subject. Any statistical significance was then evaluated.

Results

The most consistent changes during HBO therapy were a decrease in PtcCO₂ and HR. The mean PtcCO₂ did not change at 2.5 ATA with air, but decreased gradually during HBO inhalation (Fig. 1). However, continuous measurements showed the PtcCO₂ rose transiently at the beginning of HBO inhalation and then decreased gradually during HBO inhalation. The RR did not change, but the respiratory depth increased and hyperventilation

continued during HBO inhalation. During decompression after HBO inhalation, the RR reduced transiently and the PtcCO₂ increased to the previous level (Fig. 2). The HR decreased significantly by compression to 2.5 ATA ($p < 0.01$), and the reduction continued during HBO inhalation ($p < 0.05$ compared to HR at 2.5 ATA with air).

Experiment 1 (Fig. 3): The 10 spontaneous breathing patients usually demonstrated rapidly decreased ICP at the beginning of HBO inhalation which tended to increase gradually during HBO inhalation. The PtcCO₂, however, decreased gradually during HBO inhalation. The ICP and PtcCO₂ increased during and after decompression. ICP also decreased rapidly with O₂ inhalation at 1 ATA.

Experiment 2 (Fig. 4): In the patient undergoing barbiturate therapy (Case 3), respiration was artificially controlled at 2.5 ATA with air to maintain the same PtcCO₂ as at 1 ATA. When O₂ at 2.5 ATA was supplied without any change in respiratory frequency or volume, the ICP and PtcCO₂ increased

Fig. 2 Changes in blood gases and heart (HR) and respiratory rates (RR) during HBO therapy in Case 1. HR is reduced by both compression and O₂ inhalation, and the reduction continued during HBO inhalation. RR is not changed by HBO inhalation, but respiratory depth increased. PtcCO₂ rises slightly in the first 2–5 minutes (arrow) and then decreases gradually during HBO inhalation. RR decreases during decompression.

Fig. 3 Changes in ICP and blood gases during HBO therapy in Case 1. ICP decreases rapidly with the start of O_2 inhalation at both 1 and 2.5 ATA, and rises gradually during HBO. The ICP increase is marked after HBO.

gradually with a transient ICP reduction.

Experiment 3 (Figs. 5 and 6): A cooperative patient (Case 4) was asked to reduce respiration to maintain the same $PtcCO_2$ as at 2.5 ATA with air. The decreased ICP increased gradually toward the value at 2.5 ATA with air in spite of HBO inhalation. When the intentional reduction in respiration was discontinued, both $PtcCO_2$ and ICP rapidly decreased again.

Two patients demonstrated no initial ICP response to HBO inhalation. One (Case 7) showed only a slight $PtcCO_2$ decrease when intentional hyperventilation was requested during both 1 ATA with air and HBO inhalation. In the other patient (Case 2, $PtcCO_2$ and ICP started to decrease about 5 minutes after HBO inhalation began, but no initial

Fig. 4 ICP changes during HBO therapy in Case 3, a comatose patient requiring artificial respiration. When air is changed to O_2 at 2.5 ATA without altering respiratory frequency and volume, both ICP and $PtcCO_2$ increase gradually with a transient ICP reduction.

Fig. 5 ICP changes during HBO therapy and changes in blood gases with intentionally controlled respiration in Case 4. ICP returns to the value at 2.5 ATA with air when PtcCO₂ is maintained at the value at 2.5 ATA with air (arrows). ICR: intentionally controlled respiration.

Fig. 6 The correlation between ICP and the logarithm of PtcO₂ in Case 4. ICP during intentionally induced normocapnia (○) is significantly higher than that in the HBO induced hypocapnia (●). Values in the figure denote PtcCO₂ (mmHg) in each condition.

ICP reduction was seen.

Discussion

Dautrebande and Haldane³⁾ first suggested that HBO has a direct cerebral vasoconstrictive effect. Animal experiments have also demonstrated that the CBF and/or ICP decrease during HBO inhalation at constant PCO₂.^{6,9,10)} Our first and second experiments showed initial ICP reduction at the start of HBO inhalation in most spontaneously or artificially respired patients. However, this study and others⁵⁾ also showed that the ICP then increased gradually during HBO inhalation. Therefore, the vasoconstriction directly caused by HBO may only be temporary and diminishes with continued inhalation.

Our third experiment tested ICP responses to PtcCO₂ during HBO inhalation. The ICP increased when the PtcCO₂ returned to near the value at 2.5 ATA with air controlled by intentional respiration. This suggests that induced hypocapnia maintains the reduced ICP during HBO inhalation. The decreased arterial PCO₂ during HBO inhalation is probably due to hyperventilation induced by transient CO₂

retention.^{8,12)} This CO₂ retention in the respiratory center may be due to diminished red blood cell transport capacity at considerably higher O₂ pressure¹⁾ and partly to diminished blood flow.³⁾ The present study confirmed that the PtcCO₂ decreased gradually following a transient increase and remained at a lower level throughout HBO inhalation. The changes in ICP reflecting changes in the O₂ and CO₂ pressures demonstrated by this study indicate that ICP reduction during HBO therapy is influenced by both hyperoxia and induced hypocapnia. The ICP is initially reduced by the direct vasoconstrictive effect of hyperoxia and is then maintained at a lower level mainly by induced hypocapnia during HBO inhalation.

HBO therapy is reported to promote recovery in patients with head injury or cerebral ischemia.^{7,11,13,20)} Clinically, four (Cases 7, 9–11) of our 11 patients showed neurological improvements. Animal experiments showed that HBO has protective effects in cerebral edema or ischemia,^{17–19)} and suppresses increased lactate levels in the ischemic brain.¹⁷⁾ Contreras *et al.*²⁾ also showed that HBO inhalation increased cerebral glucose metabolism in a traumatized rat brain. This indicates that HBO therapy facilitates metabolic improvement in acute brain damage.

Reduced ICP was only demonstrated during HBO inhalation and the rebound phenomenon occurred after HBO therapy in the present study. Rapid vasodilation is considered to cause the rebound phenomenon since the direct vasoconstrictive effect of HBO becomes weak and there is a rapid return to lower arterial PO₂ levels.^{5,11,15,16)} Since the effect of HBO on ICP reduction is temporary, HBO therapy should be used when improvement in a hypoxic lesion is expected, rather than to reduce ICP. Moreover, in our study, low PtcCO₂ could be achieved physiologically in spontaneously respiration patients. Normocapnia during HBO inhalation in artificially respiration patients will induce CO₂ retention and result in increased arterial PCO₂ and ICP. Therefore, HBO therapy requires care in patients with a high ICP and/or who are respiration artificially.

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