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Study on Oxygen Dynamics and Metabolism under Hyperbaric Oxygenation

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To investigate the mechanism of the hyperbaric oxygenation (HBO) therapy efficacy, we assessed oxygen dynamics and metabolism during HBO therapy (60 hours at 2 ATA) by near infrared spectroscopy (NIRS) and percutaneous blood gas partial pressure monitoring in 21 men and 2 women aged 20～67 years (mean: 40.7 ± 15.4). Oxyhemoglobin (Oxy-Hb), deoxyhemoglobin (Deoxy-Hb), total hemoglobin (Total-Hb), and cytochrome aa3 (Cyt.Oxi.) were recorded at intervals of 10 seconds from the rest period preceding HBO therapy until the measurements stabilized during rest after decompression. Percutaneous blood gas partial pressure ($PtcO_2$), carbon dioxide partial pressure ($PtcCO_2$), oxygen partial pressure (PaO_2), carbon dioxide partial pressure ($PaCO_2$), pH, bicarbonate ion concentration (HCO_3^-), and base excess (BE) were also determined. The Oxy-Hb, Total-Hb, and Cyt.Oxi. levels were significantly elevated and the Deoxy-Hb levels were significantly reduced during the stable period preceding HBO therapy and the peak stable time during therapy. $PtcO_2$ significantly increased and $PtcCO_2$ significantly decreased during the stable period before HBO therapy, the peak stable period during therapy, and immediately after the end of decompression. PaO_2 and pH were significantly elevated and $PaCO_2$ was significantly reduced immediately before the start of HBO therapy and immediately after the end of decompression. There were no significant differences in BE or HCO_3^- at any of the times measured. In conclusion, the oxidized Cyt.Oxi. levels increased during HBO, substantiating a report on the dominance of reduced Cyt.Oxi. under homeostatic conditions. The elevated Oxy-Hb and Total-Hb levels appear to reflect enhancement of oxygen metabolism at the tissue level. HBO therapy appears to have little effect on acid-base equibrium, although it markedly increased the PaO_2 levels and decreased the $PaCO_2$ levels.

Introduction

Hyperbaric oxygenation (HBO) is a therapeutic modality by which a patient inhales 100% oxygen under a hyperbaric environment. Boerema et al[1] reported on the efficacy of HBO in an experiment using piglets and proved that life can be sustained by perfusion with a fluid without hemoglobin (life without blood) under a hyperbaric oxygen environment of 3 absolute atmosphere (ATA) for a limited duration. In Japan, HBO has been practiced for the past 35 years[2)～4)], during which efforts have been made in areas such as basic research, an expansion of the categories of diseases to which the treatment can be applied, examination of therapeutic results, and improvement of the therapeutic devices used.

For the physiological action mechanisms through which this therapeutic modality manifests its efficacy, the followings have been investigated: ① improvement in hypoxia by administering a large quantity of dissolved oxygen; ② physical effect of dissolved oxygen and pressure; and ③ bactericidal effect brought about by oxygen toxicity. A number of studies have been conducted to verify the action mechanism involved in HBO but because of the many restrictions placed on studies under unique conditions associated with the HBO apparatus, there are many unanswered questions.

The present study was conducted to examine the mechanism that is responsible for the efficacy of HBO. A specific method was employed: near infrared spectroscopy (NIRS). Since a report by Jobsis[5] in 1977, this method has been attracting attention as a way to determine oxygen dynamics and metabolism: it permits sequential determination of oxyhemoglobin, deoxyhemoglobin, and the redox states of mitochondrial cytochrome aa3[6)~9)]. Because of its low invasiveness, its area of clinical application is being expanded[10)~14)]. To determine the systemic oxygen dynamics simultaneously, a percutaneous gas partial pressure monitor, which has been used mainly in neonatology, was employed. This method enables one to make continuous measurements; and for making indirect measurements of arterial blood oxygen and carbon dioxide partial pressures.

An instrument was developed that can be used under HBO[15)~17)]. Until now, it was not possible to conduct NIRS under HBO. The authors added innovations to the probes so that oxygen dynamics can be examined by employing NIRS under HBO. This represents a pioneering work for evaluating oxygen dynamics and metabolism by using NIRS while conducting HBO. The authors examined oxygen dynamics and metabolism under this condition from a new viewpoint and compared the results with those of earlier studies.

Subjects and Methods

1. Subject

The subjects were 23 adults between 20 and 67 years (There were 21 men and 2 women, mean age: 40.7 ± 15.4 years). None has a history of cardiac or respiratory diseases and all were devoid of lesions or vascular disturbances at the sites where test readings were to be made. The group included 5 patients with digestive system diseases, 8 with orthopedic diseases, 2 with central nervous system diseases, 2 with diseases related to oral surgery, and 6 healthy adults. A complete explanation was given to the patients and their consents were obtained before starting the study.

2. Methods

For HBO, a T Model 2500B by Sechris was used. Oxygen dynamics and metabolism were determined during the treatment by employing NIRS and the percutaneous gas partial pressure method. With NIRS, the tissue levels of oxy- and deoxy-hemoglobins (Oxy-Hb and Deoxy-Hb) and the oxidative state of mitochondrial cytochrome aa3 (Cyt.Oxi.) were continuously monitored transcutaneously, based on the differences in absorbance of infrared rays (700 to 1,000 nm). Living tissue includes components that absorb and random reflect light; and changes in the oxygen concentration alter the photoabsorptive characteristics in some but not in others. In NIRS, the former constitutes the subject of determination. Specifically, Hb and Cyt.Oxi. are these components.

A living system contains other substances that also undergo changes in photic absorbance that depend on the oxygen concentration; but this absorbance does not occur within the wavelengths of near infrared rays. This is the starting point for the development of the present method. In the format adapted for the present method, those with a constant photic absorbancy are subtracted

as a fixed offset so that they will not affect the absorbance measurement in determining the changes in the concentration of the desired components. By determining the amount of this "light out", the changes in the concentrations of Hb and Cyt. Oxi. can be computed.

When a clear test sample is irradiated by light with a wavelength of λ_1, the concentration, C, is expressed by formula (1). This is known as the Beer-Lambert Law, in which the quantity of transmitted light is expressed as 1:

$$C = 1/(\varepsilon d) \cdot \log (I/I_0) \quad (1)$$

where I_0: amount of radiation, ε: absorption coefficient, and d: light path length. The Beer-Lambert Law applies only when the light is unscattered and uniform but it can apply for approximation for nonhomogenous system such as a living organism.

$$C = 1/(\varepsilon L) \cdot \log (I/I_0) + X \quad (2)$$

X in formula (2) represents a constant but unknown quantity that escapes without detection due to light scattering. In general, it is not possible to measure X. However, because it does not vary, it can be subtracted, e.g., $\Delta C = C(t) - C(t_0)$ when the change from time t to t_0 is to be computed. L is the light path length and represents the actual distance the light travels while being scattered through tissue from the point of radiation to that of detection[18)~20)].

For a near infrared spectrometric monitor of the present study, a Niro 500 from Hamamatsu Photonics was used. The probes were modified for use in a hyperbaric oxygen chamber. Rubber packing was used at the feed through terminal of the opening and closing door of the chamber to maintain the airtightness of the chamber.

A probe was attached to the palmer side of the right radius, 5 cm from the elbow joint, for near infrared monitoring and the entire apparatus was covered by a piece of cloth that was taped to the skin to form a shield from the external light. The distance between the light emitting and light receiving points at the site where the measurements were to be made was fixed at 4.5 cm, the path length at 16.2 cm (3.6 times the actual distance[19)]). The Oxy-Hb, Deoxy-Hb, total hemoglobin (Total-Hb), and Cyt.Oxi. contents were recorded continuously at 10 sec intervals from the time of resting before the start of HBO to the second rest period after completion of decompression when the readings became stabilized. To measure transcutaneous oxygen and carbon dioxide partial pressures, a method that was developed by Huch et al[21)] when the heating element of a transcutaneous electrode raises the temperature immediately below it was used. This method is based on the fact that dermal capillary blood flow increases, raising the O_2 and CO_2 partial pressures and augmenting the gas diffusion and permeability through the skin when the heating element of a transcutaneous electrode raises the temperature immediately below it[15)16)]. A TCM-3 by Radiometer was used to analyze and record the transcutaneous gas partial pressures.

A probe for transcutaneous monitoring of oxygen and carbon dioxide partial pressures was placed at the left edge of the sternum at the left second rib, a site that is close to the heart, relatively free of adipose tissue, and believed to yield relatively stable readings and generally reflect systemic conditions. In the probe, the $PtcO_2$ electrodes are Clark-type oxygen electrodes that are composed of an anode (silver) and a cathode (platinum). For the $PtcCO_2$ electrodes, Severinghause-type pH electrodes were used. The probe temperature was set at 43 ℃, which is well within a measurable range and is not likely to injure the skin. The transcutaneous oxygen and carbon dioxide partial pressures were also continuously recorded from the time of rest before the start of HBO until the rest time after the completion of decompression when the readings became stabi-

lized. Immediately before the start of pressurization and immediately after the completion of decompression, blood samples were collected from the left femoral artery to determine oxygen and carbon dioxide partial pressures, pH, bicarbonate ion (HCO_3^-), and the base excess (BE) and evaluate the systemic gas and acid-base dynamics. When both instruments were stabilized, recording was started and after 5 min or more of recording at rest at ambient, continuous determination during HBO was initiated (The duration of treatment was set at 60 min at 2 ATA. Compression and decompression were applied for approximately 10 min each.). After the reading had been stabilized for more than 10 min following decompression, the recording was completed. The changes in oxygen dynamics and metabolism were examined from the readings taken by each instrument before, during, and after HBO (Fig. 1).

The near infrared ray readings were expressed by the change (Δ μmol/l) from base value 0, which is the reading made immediately after the instrument was attached. The readings made at the stable period before the start of HBO were designated as group a and those made at the peak stable period during treatment at 2 ATA (immediately before decompression) as group b. The means of 30 serial readings (5 min) were compared. For the transcutaneous gas partial pressure measurements, the readings during the stable period before the start of HBO were designated as group c, those at the peak stable period (immediately before decompression) as group d, and those immediately after completion of decompression as group e. For the arterial blood analyses, the reading taken at the stable period before the start of HBO were designated as group f and those made immediately after the completion of decompression as group g. The means for these groups were used for compari-

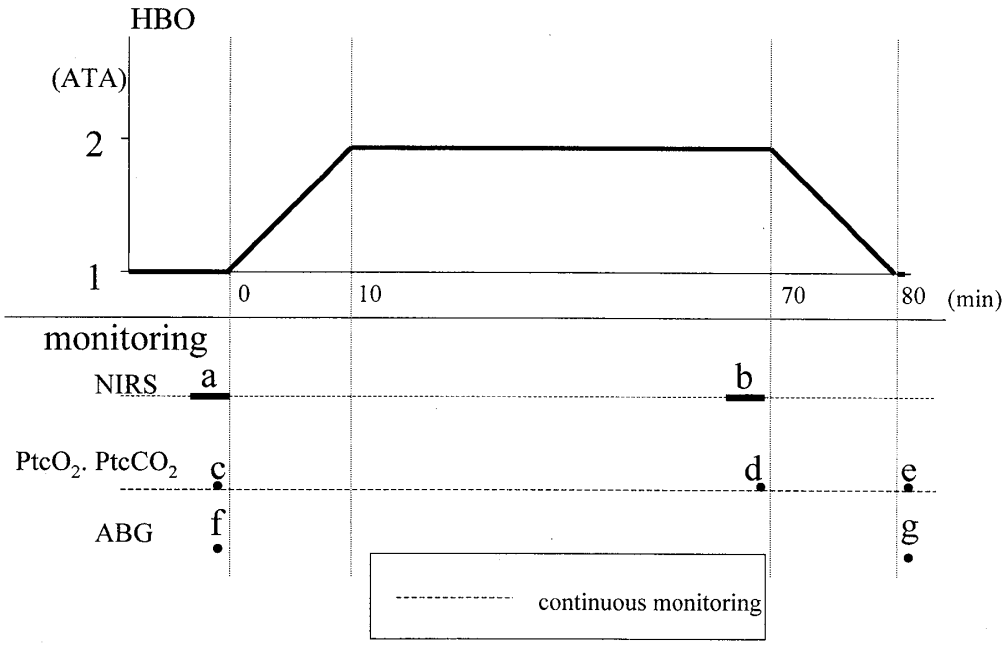

Fig. 1 Schedules for data collection for comparison under hyperbaric oxygenation (HBO)
NIRS : near infrared spectroscopy, $PtcO_2$: transcutaneous tissue oxygen partial pressure, $PtcCO_2$: transcutaneous tissue carbon dioxide partial pressure, ABG : arterial blood gas.

sons. For statistical analyses, all values were expressed as the mean ± SD. Mean were compared by *t* test (paired *t* test), one-way analysis of variance (ANOVA) followed by Fisher's Protected Least Significant Difference test. The level of significance was set at less than 5%.

Results

1. Determination by NIRS (Figs. 2 and 3)

1) Oxyhemoglobin (Oxy-Hb)

Twenty of the 23 subjects exhibited increases immediately after the start of HBO and reached peaks. Immediately after decompression, the level began to decrease, approaching the level before treatment, where it was stabilized. Group a: mean − 3.20 ± 6.88 Δμmol/*l*, group b: mean 5.04 ± 12.54 Δμmol/*l*. The difference between the two groups was 8.24 Δμmol/*l*. The increase showed statistical significance ($p<0.01$).

2) Deoxyhemoglobin (Deoxy-Hb)

In 18 of these 23 subjects, the level began to decline immediately after the start of HBO, shortly after which it reached a peak. The level began to rise immediately after decompression, approached the pretreatment level, then stabilized. Group a: mean −1.77 ± 0.50 Δμmol/*l*, group b: mean −5.37 ± −4.94 Δμmol/*l*. The mean difference between the two groups was 3.60 Δμmol/*l*. The level showed a significant reduction ($p<0.01$).

3) Total hemoglobin (Total-Hb)

Among 23 subjects, 17 showed increases and 6 showed reductions. Group a: mean − 5.47 ± 11.62 Δμmol/*l*, group b: mean 0.67 ± 14.98 Δμmol/*l*. The intergroup difference was 5.64 Δμmol/*l*, showing an increase with statistical significance ($p<0.05$).

4) Cytochrome aa3 (Cyt.Oxi.)

In 20 of the 23 subjects, the level increased im-

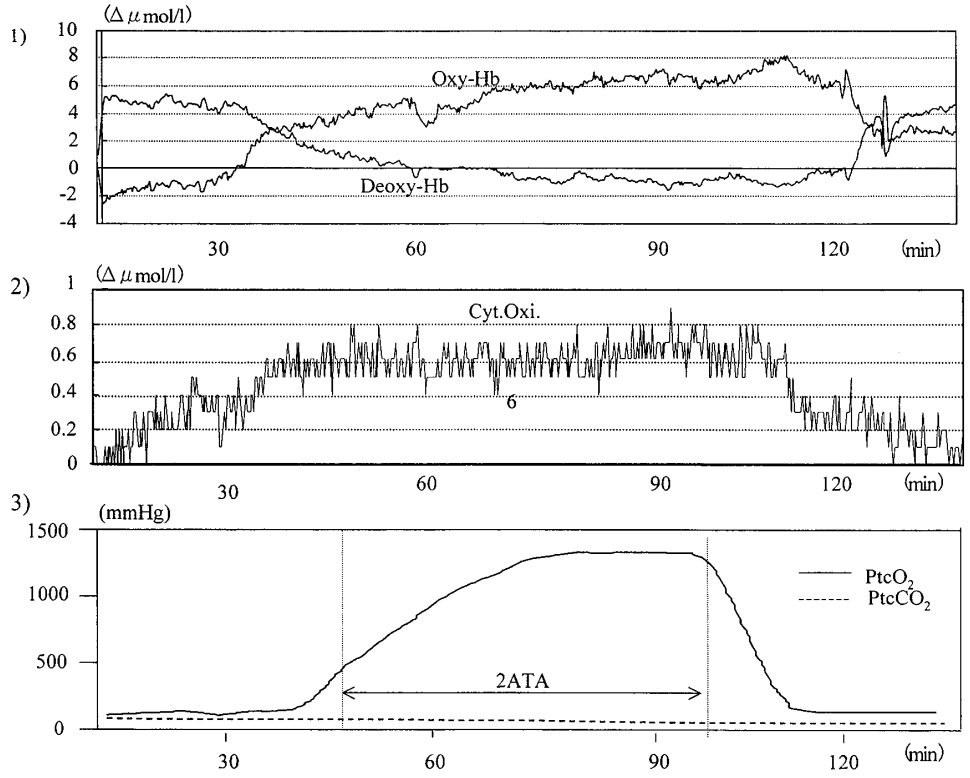

Fig. 2 A 57-year-old male

1), 2): Changes in the oxy-, deoxy-hemoglobin (Oxy-HB and Deoxy-Hb) and the cytochrome aa3 (Cyt.Oxi.) concentration levels under HBO determination by NIRS.

3): Transcutaneous gas partial pressure ($PtcO_2$ and $PtcCO_2$) under HBO.

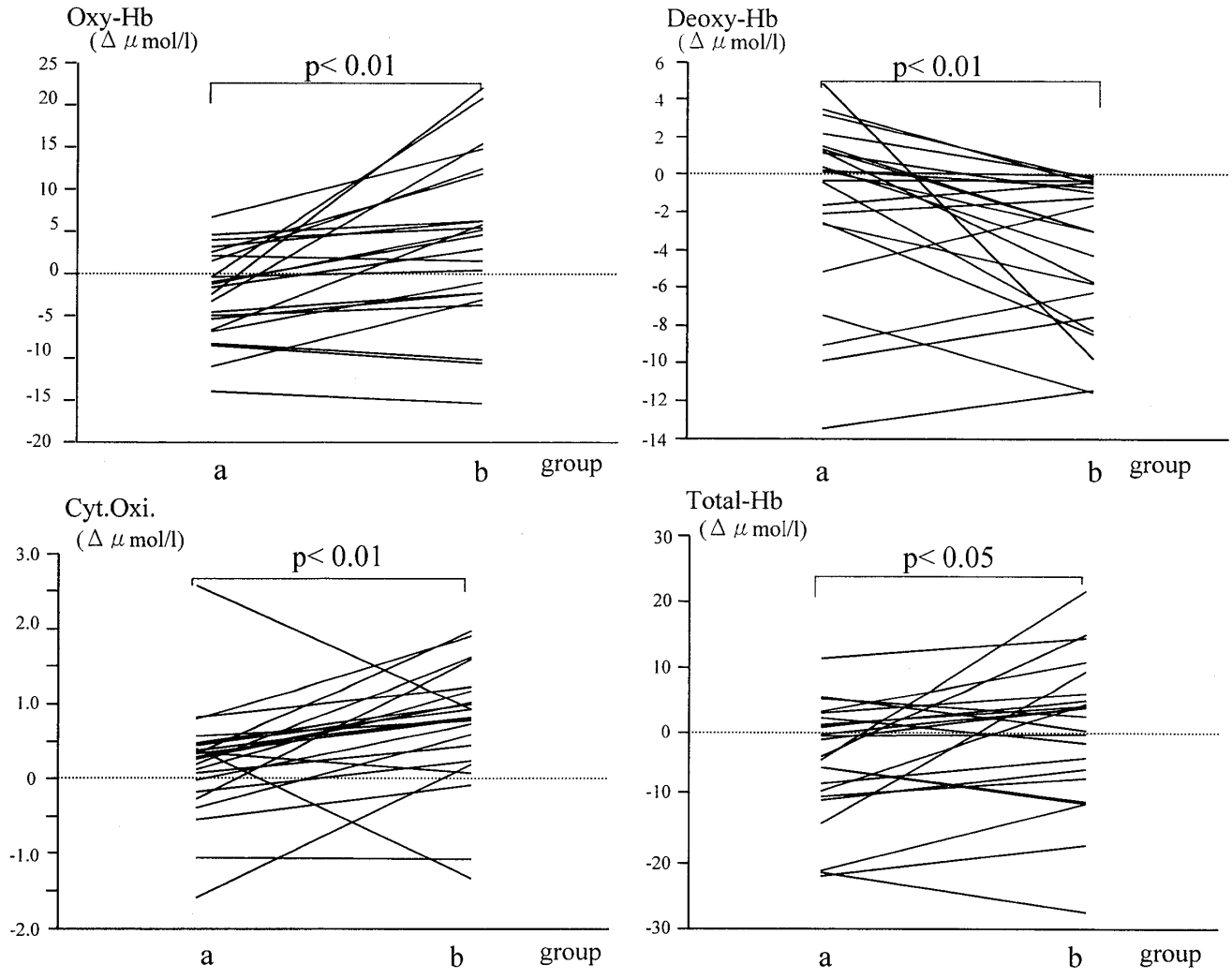

Fig. 3 Oxi-Hb, Deoxy-Hb, Total-Hb and Cyt.Oxi. concentration determined by NIRS
group a: during rest period before the start of HBO, group b: at the peak during HBO.

mediately after the start of HBO, peaked, then decreased immediately after decompression, approached the pretreatment level, and became stabilized. Group a: mean 0.28 ± 1.03 Δμmol/*l*, group b: mean 1.00 ± 1.09 Δμmol/*l*. The mean difference was 0.72 Δμmol/*l*. The increase showed a statistically significant difference ($p<0.01$).

2. Transcutaneous gas monitoring determination (Figs. 2 and 4)

In all subjects, the $PtcO_2$ began to increase immediately after the start of compression, reaching a plateau in about 45 min. At the start of decompression, the level began to decline, almost reaching the pretreatment level; then it stabilized within about 45 to 60 min. During HBO, the $PtcCO_2$ level came down gradually and at a fixed rate.

1) Transcutaneous oxygen partial pressure ($PtcO_2$)

Group c: mean 83.3 ± 12.8 mmHg, group d: mean 1329.2 ± 121.3 mmHg, group e: mean 312.5 ± 157.1 mmHg. Significant differences were noted between groups (groups c : d $p<0.01$, groups c : e $p<0.01$, groups d : e $p<0.01$).

2) Transcutaneous carbon dioxide partial pressure ($PtcCO_2$)

Group c: mean 34.4 ± 4.2 mmHg, group d: mean 29.8 ± 4.9 mmHg, group e: mean 29.9 ± 4.6 mmHg.

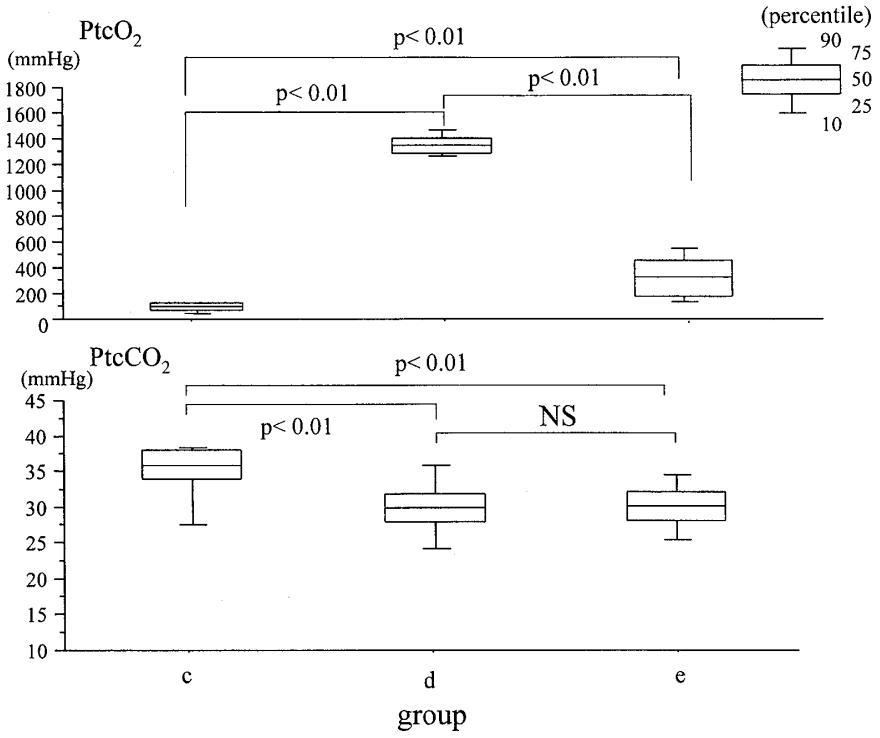

Fig. 4 Transcutaneous tissue oxygen partial pressure ($PtcO_2$) and carbon dioxide partial pressure ($PtcCO_2$)
group c: before HBO, group d: at the peak, group e: immediately after completion.

A significant difference was noted between groups c and d and c and e (groups c : d $p<0.01$, groups c : e $p<0.01$).

3. Arterial blood gases (ABG) (Figs. 5 and 6)

1) Arterial blood oxygen partial pressure (PaO_2)

Group f: mean 92.7 ± 13.6 mmHg, group g: mean 128.5 ± 44.0 mmHg. A significant increase ($p< 0.01$) was noted.

2) Arterial blood carbon dioxide partial pressure ($PaCO_2$)

Group f: mean 38.6 ± 13.6 mmHg, group g: mean 36.7 ± 4.4 mmHg. A reduction with a significant difference ($p < 0.05$) was noted between the groups.

3) pH

Group f: mean 7.46 ± 0.04, group g: mean 7.48 ± 0.04. An increase with a significant difference ($p <0.01$) was noted between the groups.

4) Base excess (BE)

Group f: mean 4.17 ± 2.53 mmol/*l*, group g: mean 4.35 ± 2.51 mmol/*l*. No significant difference was found between the groups.

5) HCO_3^-

Group f: mean 27.6 ± 2.9 mmol/*l*, group g: mean 27.8 ± 2.8 mmol/*l*. No significant difference was found between the groups.

Discussion

In this study, oxygen metabolism was determined by using NIRS. The Oxy-Hb content that can be determined by this method was found to increase under the following conditions: ① an increse in blood oxygen saturation, ② an increase in the blood flow volume in association with vasodilation and a rise in blood pressure, ③ an increase in the volume of oxygen being transported by Hb, ④ a reduction in oxygen consumption by tissue. The Deoxy-Hb content reacts to changes either in O_2 saturation or circulation. Under conditions such as a reduction in O_2 rsaturation, venous

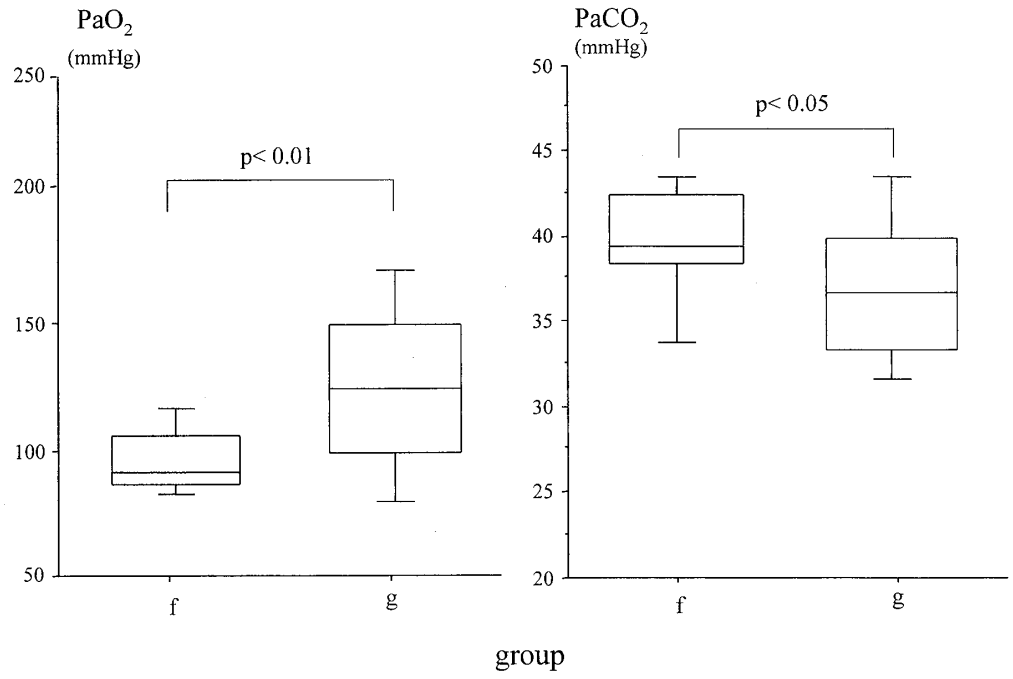

Fig. 5 Arterial blood oxygen partial pressure (PaO_2) and carbon dioxide partial pressure ($PaCO_2$) sample collected from the femoral artery
group f: immediately before the start of HBO, group g: immediately after the completion of decompression.

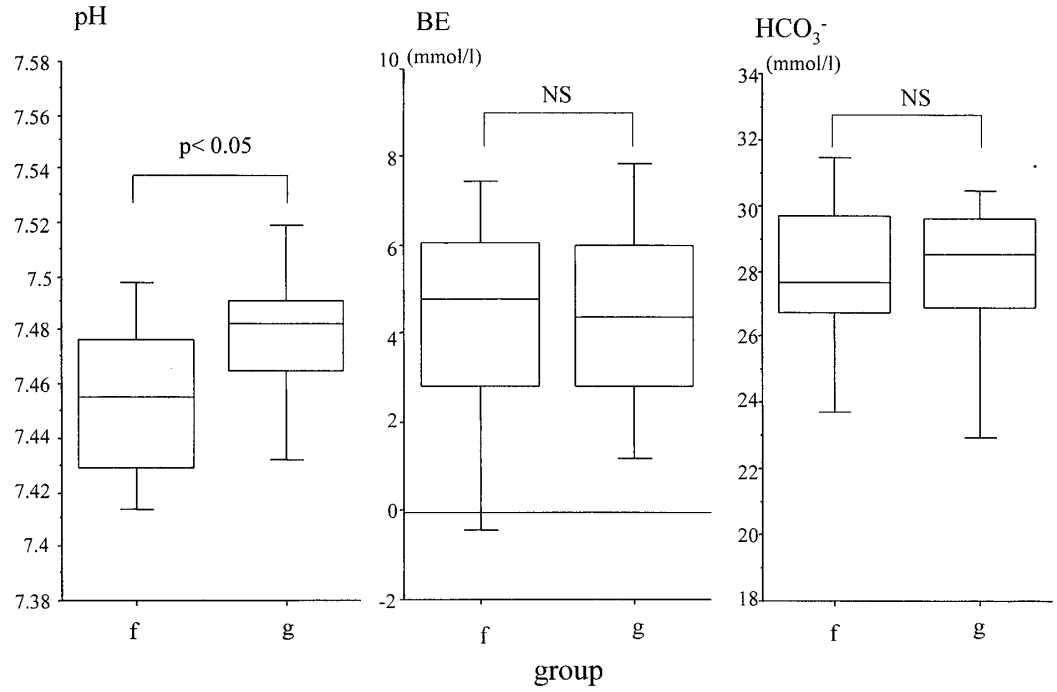

Fig. 6 Femoral arterial blood pH, base excess (BE) and bicarbonate ion (HCO_3^-)

occlusion, and flow of blood with a low oxygen saturation into tissue (hypoxia), the responses of the Oxy-Hb and Deoxy-Hb levels are diametrically opposed: i.e., with a drop in the oxygen satu-

ration level, Oxy-Hb is reduced while Deoxy-Hb increases.

In NIRS, the blood flow volume is computed as the total Hb content (Oxy-Hb + Deoxy-Hb). When this blood flow volume remains unchanged, the Oxy-Hb and Deoxy-Hb contents change in opposite directions but at the same amplitude while the Total-Hb remains the same. If there is a difference between the changes in the Oxy-Hb and Deoxy-Hb contents or the two undergo changes in the same direction (increase or reduction), the blood flow volume will be altered[9)22)23)]. If sudden changes in the hemoglobin concentrations are added to this (such as from a blood transfusion or hemorrhage), the test results will be seriously affected, requiring special attention.

In the present study, however, no patients required blood transfusion or suffered from a hemorrhage during the observation period so special provisions were not needed. According to the results from the observation of tissue hemoglobin dynamics determined by NIRS at the right forearm, the Oxy-Hb and Total-Hb contents showed an increase and the Deoxy-Hb level underwent a reduction under HBO (both with a statistical significance). These findings indicate that during HBO, the content of hemoglobin with highly saturated oxygen increases in tissue while the Deoxy-Hb content is reduced. A significant increase in the Total-Hb content (the quantity of total hemoglobin), an indicator for the blood flow volume in tissue, was noted when compared with the readings before and during HBO, suggesting a possible increase in tissue blood flow during this treatment modality.

According to the literature on this subject, HBO causes the cerebral vasculature to contract and reduces the blood flow volume by 1.3% at 1 ATA (Kety[24)]), 21% at 2 ATA (Jacobson[25)]), and 25% at 3.5 ATA (Lanbertsen[26)]). From the finding obtained by transcranial Doppler sonography, the blood flow velocity at the middle cerebral artery is 43 cm/sec at 1 ATAair, which is reduced to 41 cm/sec at 1 ATA O_2 and 30 cm/sec at 2 ATA HBO, indicating an approximately 30% reduction for each ATAair[25)]. For the effects of HBO on the cardiovascular system, it has been reported that the cardiac output is reduced by 25% at 4 ATA for 90 min[27)]. It was suggested that the procedure may even provoke bradycardia[28)]. Because no changes have been detected in the arterial and venous pressures, increases in peripheral vascular resistance are possible and contraction of peripheral vasculature has been suggested[27)]. To explain this phenomenon, a biological mechanism to adjust to the exposure to excessive oxygen (oxygen toxicity) has been visualized.

As noted above, in a number of studies reductions in the peripheral blood flow under HBO have been described. Dysfunctions of vascular autoregulation[29)] and increases in blood flow to the ischemic lesions under HBO have also been reported in relation to cerebrovascular circulation in cerebral ischemic states such as cerebral infarctions[30)]. In practice, however, this therapeutic modality has been applied to treat various types of ischemic diseases and many have reported successful outcomes[31)32)]. In this study, tissue blood flow (Total-Hb) showed significant increases, suggesting a mechanism to explain this therapeutic modality. This differs from the results of earlier studies, in which the blood volume was determined through direct observation of blood vessels[25)26)33)].

NIRS is a method to determine changes in the Total-Hb concentrations in tissue as a whole unit. The discrepancy from prior studies may be explained by the fact that the blood flow volume increases in microcirculation (that was not possible to detect in the past) through mechanisms such as an improvement in erythrocyte transport velocity and a reduction in platelet aggregation[34)]

under HBO. To confirm the efficacy of this therapeutic efficacy, further evaluation of tissue perfusion for each organ, anatomical site, or pathophysiological condition is necessary.

The extent of oxidation of Cyt.Oxi. at a normal state is still the subject of controversy. In 1955, Chance et al [35] reported that in an *in vitro* study, most of the mitochondrial Cyt.Oxi. is in an oxidized state in a normal condition and the reduced form is found in a very hypoxic environment. In 1985, Jobsis et al[36] reported in their *in vivo* study that 50% of Cyt.Oxi. is in an oxidized state under normal conditions but the enhanced tissue oxygen metabolism increases the oxidized type while a hypoxic state causes an increase in the reduced form. A report by Hoshi et al[37][38] contradicted these findings: in an experiment using rats, the oxidative state of Cyt.Oxi. remained constant in spite of a rise in inhaled oxygen concentration from 21% to 100%; and its reduction was recognized only when the oxygen concentration was reduced to 8%. Jobsis[36] contradicted these findings by citing the possibility of overlapping effects of changes in the Hb content to those of Cyt.Oxi. or the hypoxic state of the tissue.

In 1992, Okada et al[23] postulated that a manipulation to reduce the substrate content might result in a reduced supply of substrate electrons to the respiratory chain, resulting in a failure of electrons to reach Cyt.Oxi. on the respiratory chain and exaggerated oxidation of the Cyt.Oxi. When one examines changes in the Cyt.Oxi. content in the hypoglycemic model that was used by Bryan and Jobsis[39], it is found that a sustained extreme hypoglycemic state results in a shift of Cyt.Oxi. to a more oxidized state, suggesting the presence of a reduced state in a normal condition[23]. As described above, no consensus has been reached on the state of Cyt.Oxi. under normal conditions. Under HBO (2 ATA) of the present study, changes in the concentrations of Cyt.Oxi. undergoing redox increased, showing a significant difference between before and during this therapeutic procedure. This finding indicates that in a normal condition, Cyt.Oxi. is not altogether in an oxidized state but does exist in a reduced state. According to the report by Hoshi et al [37] cited above, inhalation of 100% oxygen did not cause any changes; the changes that were observed in this study may be unique to HBO. It is also possible that under HBO, oxygen metabolism is enhanced at the cellular level.

Because it is difficult to collect specimens by an invasive method for blood gas analysis under HBO, application of the method as a transcutaneous gas monitor during this therapeutic modality has started. There have been several reports on this subject[15]~[17]. In this study, the mean $PtcO_2$ was 1329.2 mmHg under HBO at 2 ATA, which approximated the theoretical value of 1433 mmHg[40] for blood oxygen partial pressure under HBO at 2 ATA. It proved that $PtcO_2$ faithfully reflects the blood oxygen partial pressure; and the method is effective as a blood gas monitor under high atmospheric pressure. When compared against the results of arterial blood gas analysis immediately after decompression, an evident difference was noted between $PtcO_2$ and PaO_2, indicating a tendency for a delay in the reduction of $PtcO_2$ after a rapid reduction in the arterial blood oxygen partial pressure. This finding appears to correspond to data in a report by Boerema et al[1], in which it was stated that even after HBO, the tissue oxygen partial pressure remains high for 2 to 4 hours.

The $PtcCO_2$ content before HBO was compared against those during the procedure and immediately after decompression. A tendency toward a significant reduction is probably due to transient retention of CO_2, which triggers a hyperventilatory state and a subsequent reduction in the $PtcCO_2$ content[27]~[41]. There have been occa-

sional reports on arterial blood gas contents under HBO. The current study was expanded to include pH, BE, and HCO_3 before and after HBO. The findings on $PaCO_2$ in the literature are generally divided into two groups—those describing reductions[27)41)] and those that report on no significant changes or increases[30)42)]. In this study, a trend for significant reductions was noted together with a significant rise in pH. As described earlier, it appears that the physiological mechanism under HBO is responsible for this reduction in $PaCO_2$ and a rise in pH. However, there are reports that note only a slight rise in the CO_2 content in the central nervous system and a drop in pH[42)43)], indicating a lack of uniformity among organs within a body. A need to evaluate the effects on each organ and anatomical site is indicated. No significant changes were noted in BE and HCO_3^-. The findings were interpreted to mean that the effect of HBO at 2 ATA for one hour is minimal on acid-base equilibrium in the body.

Conclusion

Peripheral (at the right forearm) oxygen metabolism and dynamics under HBO was examined percutaneously by using NIRS. Percutaneous oxygen and carbon dioxide partial pressures were measured to find *in vivo* oxygen dynamics and metabolism under HBO. The findings were :

1. Under HBO at 2 ATA, a significant increase was recognized in the Oxy-, Total-Hb. The finding reveals the state of tissue blood flow: it is believed that it shows a mechanism attesting to the efficacy of HBO.

2. Under HBO, the oxidized cytochrome level also increased, which supports the reports that describe the reduced state of cytochrome in a normal state. It was believed that under this therapeutic condition, oxygen metabolism is augmented at the tissue level.

3. A slight rise in pH is recognized in response to acid-base equilibrium in the body. The procedure appears to have little effect or cause changes in BE or HCO_3^-.

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高気圧酸素治療下の酸素動態，代謝に関する検討

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高気圧酸素治療(HBO)の有効性の機序を検討することを目的とし，近赤外線法(near infrared spectroscopy: NIRS)を利用し，同時に全身的酸素動態の測定には経皮的ガス分圧モニターを利用した．著者らは新たにプローベを改造しHBO下のNIRSによる酸素動態の測定を可能とした．従って近赤外線法によるHBO下の酸素動態，代謝の検討は本研究が嚆矢となるものである．対象は20～67（平均40.7±15.4）歳，男性21例，女性2例で治療時間は2気圧で60分間である．治療開始前よりNIRSで右前腕部組織中の酸化ヘモグロビン(Oxy-Hb)，還元型ヘモグロビン(Deoxy-Hb)，総ヘモグロビン(Total-Hb)，チトクローム aa3 (Cyt.Oxi.)を測定し，同時に経皮酸素分圧 ($PtcO_2$)，二酸化炭素分圧 ($PtcCO_2$) も測定した．治療前後に左大腿動脈より血液を採取し酸素分圧 (PaO_2)，二酸化炭素分圧 ($PaCO_2$)，pH，重炭酸イオン (HCO_3^-)，Base excess (BE) を検討した．治療開始前，治療中，治療後の各測定機器の測定値より酸素動態，代謝の変化を検討した．NIRSは開始前をa群，ピーク時をb群とし，経皮ガス分圧測定法はHBO開始前をc群，ピーク時をd群，減圧終了直後をe群とし，動脈血分析は開始直前をf群と減圧終了直後をg群とし検討した．結果，a群，b群間でOxy-Hb，Total-Hb, Cyt.Oxi.は有意に増加，Deoxy-Hbは有意に減少，$PtcO_2$はc群とd群，e群間で有意に増加，$PtcCO_2$はc群とd群，e群間で有意に減少，f群，g群間でPaO_2，pHは有意に増加，$PaCO_2$は有意に減少，BE，HCO_3^-は有意差を認めなかった．

HBO下では酸化型チトクローム aa3の増加を認めた．これは，恒常状態で還元型チトクローム aa3が存在するという報告を支持するものであり同治療下ではOxy-Hb，Total-Hbも増加しており組織レベルで酸素代謝が亢進していると考えられた．また同治療中にPaO_2の著明な上昇と$PaCO_2$の低下を認めるが酸塩基平衡には影響の少ない治療法であった．