Monitoring carbon dioxide in mechanically ventilated patients during hyperbaric treatment

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Abstract

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Background: Measurement of the arterial carbon dioxide (P_aCO_2) is an established part of the monitoring of mechanically ventilated patients. Other ways to get information about carbon dioxide in the patient are measurement of end-tidal carbon dioxide ($P_{ET}CO_2$) and transcutaneous carbon dioxide ($P_{TC}CO_2$). Carbon dioxide in the blood and cerebral tissue has great influence on vasoactivity and thereby blood volume of the brain. We have found no studies on the correlation between $P_{ET}CO_2$ or $P_{TC}CO_2$, and P_aCO_2 during hyperbaric oxygen therapy (HBOT).

Method: We studied 10 intubated and ventilatory stable patients during HBOT. End-tidal and transcutaneous measurements provided continuous data. Arterial blood samples were collected after reaching the operational pressure of 284 kPa (2.8 ATA) and analysed outside the chamber. A total of 17 paired samples of $P_{ET}CO_2$, $P_{TC}CO_2$ and P_aCO_2 were obtained.

Results: There was a good correlation between $P_{ET}CO_2$ and P_aCO_2 using linear regression ($r^2 = 0.83$). Bland-Altman analysis showed that $P_{ET}CO_2$ on average was 2.22 kPa higher than P_aCO_2 with limits of agreement (LoA) at ± 2.4 kPa. $P_{TC}CO_2$, on average, was 2.16 kPa lower than P_aCO_2 and the correlation using linear regression was poor ($r^2 = 0.24$). Bland-Altman analysis revealed LoA at ± 3.2 kPa.

Conclusion: During hyperbaric conditions we found that $P_{ET}CO_2$ as opposed to $P_{TC}CO_2$ offered the greater precision, but there was great variability among patients. Care must be taken when using $P_{ET}CO_2$ or $P_{TC}CO_2$ as an estimate of P_aCO_2.

Key words

Patient monitoring, carbon dioxide, hypercapnia, hyperbaric oxygen therapy, ventilators

Introduction

Monitoring carbon dioxide (CO_2) is vital during mechanical ventilation to prevent hypercapnia and cerebral vasodilatation. Hypercapnia is known to inhibit the normal autoregulation of cerebral blood flow resulting in vasodilatation. This increases the toxic effect of oxygen during hyperbaric oxygen therapy (HBOT) and the risk of seizures. Vasodilatation poses a specific risk in patients with cerebral oedema including carbon monoxide-poisoned patients who often have cerebral inflammation.[1–4]

The correlation between arterial carbon dioxide (P_aCO_2) and both end-tidal ($P_{ET}CO_2$), and transcutaneous carbon dioxide ($P_{TC}CO_2$) has been well established under normobaric conditions.[5–7] We have searched for similar studies performed during HBOT. However, no publications were found on the correlation between $P_{ET}CO_2$ and P_aCO_2 and only one study regarding the correlation between $P_{TC}CO_2$ and P_aCO_2.[8] The goal of this study was to determine alterations in carbon dioxide during HBOT and how $P_{ET}CO_2$ and $P_{TC}CO_2$ each correlate with P_aCO_2 under hyperbaric conditions.

Methods

SUBJECTS

This prospective observational study was performed at Rigshospitalet Copenhagen University Hospital; 10 consecutive, mechanically ventilated patients undergoing HBOT were included from January to March 2011. All data were obtained during routine clinical care. Retrospectively, the local ethics committee determined that additional informed consent was not required.

PROCEDURES

The study was conducted in a multiplace chamber equipped to handle a single intensive care patient. HBOT consisted of 90 minutes at 284 kPa (2.8 ATA). All patients were mechanically ventilated (Siaretron 1000, Siare, Italy) in volume control mode and monitored with a mainstream capnograph (M2501A, Philips, Holland). The first arterial sample was drawn a minimum of 20 minutes after reaching operational pressure and the second 30 minutes later. The samples were analysed at normobaric conditions (ABL700-series, Radiometer, Denmark) within 5 min of being drawn. The transcutaneous sensor (TCM4, Radiometer, Denmark), was placed in the mid-clavicular line, 5 cm below the right clavicle with a temperature of 44 +/-1^{O}C.

STATISTICAL ANALYSIS

Linear regression and Bland-Altman analyses were used to assess the correlation between $P_{ET}CO_2$ and P_aCO_2 and between $P_{TC}CO_2$ and P_aCO_2. In the latter, the limits of agreement are the mean difference ± 1.96 standard deviations.[9] Quantitative results were described by the mean and standard deviation (SD). MedCalc v. 11.2 was used to analyse the data.

Results

The 10 patients included were five women and five men, aged 40 to 79 years. Nine patients came from the intensive care unit and one from the trauma centre. Eight of the patients were treated for necrotising fasciitis and two for carbon monoxide poisoning. All patients were haemodynamically stabile. One patient required the use of vasopressors (noradrenaline 1 μg kg^{-1} min^{-1}). Patients were often treated with multiple sessions, of which only one was included in this study. In three of the patients, it was only possible to obtain a single arterial blood sample.

All patients were normocapnic upon arrival at the pressure chamber with P_aCO_2 at 4.94 (0.95) kPa. In one case it was necessary to increase the ventilation during treatment. In the remaining patients with unaltered respirator settings, the response to HBOT varied among patients as P_aCO_2 increased between 7% and 80% between the two arterial samples. In the 17 arterial samples obtained, P_aCO_2 was 6.2 (1.87) kPa.

During the first 10 minutes of HBOT, $P_{ET}CO_2$ increased between 62% and 151%, but after this initial increase the $P_{ET}CO_2$ remained stable until decompression. Using linear regression there was a good correlation between $P_{ET}CO_2$ and P_aCO_2 ($r^2 = 0.83$). Bland-Altman analysis showed that $P_{ET}CO_2$ overestimated P_aCO_2 by 2.22 kPa with limits of agreement (LoA) at ± 2.4 (Figure 1).

The transcutaneous readings were also stable after 10 minutes, but the response to HBOT varied among patients as $P_{TC}CO_2$ decreased in some patients and increased in others. In the Bland-Altman analysis $P_{TC}CO_2$ underestimated P_aCO_2 by 2.16 kPa with LoA at ± 3.2 (Figure 2).

Discussion

DEVELOPMENT OF HYPERCAPNIA

It seems that two mechanisms might cause hypercapnia in mechanically ventilated patients during HBOT: reduced expiratory flow and increased ventilatory dead space, V_{DS}. As pressure rises, so does the density of inspired air. This leads to an increase in airway resistance. The inspiratory flow is maintained by the ventilator by increasing the pressure, but the expiratory flow will inevitably decrease. This results in a prolonged time of expiration and favours ventilation at higher lung volumes, decreasing the compliance of the lungs, and possibly causing CO_2 retention. It has been demonstrated that V_{DS} increases by 18% at 284 kPa in a healthy subject at rest.[10] A worsening of the physiological mismatch between lung ventilation and perfusion is the most likely cause. If a corresponding increase in ventilation is not applied, CO_2 will increase. The wide variability in P_aCO_2 in patients under pressure could indicate that additional factors contribute to the hypercapnia observed during HBOT.

END-TIDAL MEASUREMENTS

That $P_{ET}CO_2$ consistently overestimated P_aCO_2 might be the result of a phenomenon termed 'cross-interference'.[11,12] The device analyses an infrared spectrum passed though the expired gas and analyses a narrow range of wavelengths, called the absorption spectrum, where the amount of light is reduced as the amount of CO_2 rises. Gases such as nitrogen, nitrogen oxide and oxygen have very similar absorption spectra and can interfere with that of CO_2. The increased ambient pressure widens the absorption spectrum of each gas causing an increased overlap with the neighboring gases, an effect called pressure broadening, which could cause

Figure 1

Bland-Altman plot of arterial carbon dioxide (P_aCO_2) and end-tidal carbon dioxide ($P_{ET}CO_2$); mean +2.2 kPa, limits of agreement ± 2.4 kPa.

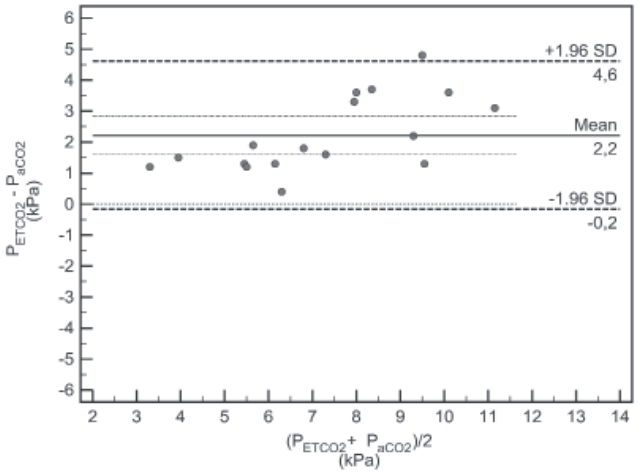
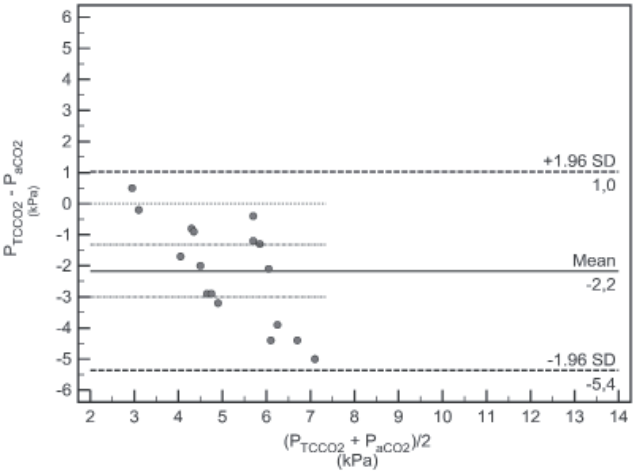


Figure 2

Bland-Altman plot of arterial carbon dioxide (P_aCO_2) and transcutaneous carbon dioxide ($P_{TC}CO_2$); mean -2.2 kPa, limits of agreement ± 3.2 kPa.



falsely elevated readings.[13] The devices used today are designed to minimise the effect of cross-interference, but are not developed specifically for hyperbaric use and therefore do not take into account the effect of pressure broadening.

TRANSCUTANEOUS MEASUREMENTS

Our findings suggest that $P_{TC}CO_2$ correlates poorly with P_aCO_2. Similar results have been reported elsewhere.[8] This is likely owing to physiological changes to the perfusion of the skin, and thereby the amount of CO_2. As an indicator of microcirculatory haemodynamics, $P_{TC}CO_2$ is an interesting parameter, but probably not one that can be expected to equal P_aCO_2, especially during HBOT.

Conclusions

Mechanically ventilated patients undergoing HBOT seem to be at risk of developing hypercapnia as P_aCO_2 increased by as much as 80% in some patients. The response, however, is highly individual, which underlines the need for precise estimates of P_aCO_2 during treatment. From the data gathered at 284 kPa, it seems that $P_{ET}CO_2$ overestimated P_aCO_2 on average by 2.22 kPa, but followed a linear regression model. $P_{TC}CO_2$ underestimated P_aCO_2 on average by 2.16 kPa and showed no linear correlation. Both methods for estimating P_aCO_2 can provide valuable information, but care must be taken when using such measurements to decide whether hypercapnia is imminent or present and changes in ventilation are required.

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