

# Relationship between mean arterial pressure and end-tidal partial pressure of carbon dioxide during hemorrhagic shock and volume resuscitation

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## ABSTRACT

**Objectives.** We examined the relationship between partial end-tidal CO<sub>2</sub> (pet) and mean arterial pressure in patients with traumatic hemorrhagic shock, who were receiving constant minute ventilation.

**Methods.** In 61 patients we continuously measured pet CO<sub>2</sub> with a capnograph, direct arterial pressure via a cannula, oxygen levels via pulse oximetry and body temperature.

**Results.** We observed significant changes in pet CO<sub>2</sub> (increase) after volume resuscitation and a quantitative linear relationship between pet CO<sub>2</sub> and mean arterial pressure.

**Conclusions.** Partial end-tidal CO<sub>2</sub> can be used as a reliable non-invasive monitoring device in patients with hemorrhagic shock when minute ventilation is relatively constant. The monitoring of pet CO<sub>2</sub> might also be a useful guide for volume resuscitation in hemorrhagic shock, especially in the pre-hospital setting.

**Keywords:** end-tidal CO<sub>2</sub>, mean arterial pressure, hemorrhagic shock, relationship

## Introduction

Partial end-tidal CO<sub>2</sub> pressure (pet CO<sub>2</sub>) is normally determined by CO<sub>2</sub> production (metabolism), alveolar ventilation, pulmonary perfusion (circulation) and V/Q matching. (1)

Pet CO<sub>2</sub> has been shown to be a reliable and non-invasive prognostic indicator of the success of cardiac resuscitation, (2,3) and highly correlates with cardiac index. (4-6) Additionally, investigators have confirmed on animal models that pet CO<sub>2</sub> can be used for non-invasive and continuous monitoring of cardiac output in circulatory shock. (7-10) Some authors reported the use of pet CO<sub>2</sub> as a predictor of mortality and as a useful intraoperative tool for assessing

the physiological conditions of the patient. (11,12) The aim of this study was to examine the relationship between mean arterial pressure (MAP) and pet CO<sub>2</sub> in patients with hemorrhagic shock in the presence of constant minute ventilation end tidal (Et) CO<sub>2</sub> monitoring be considered a technically simple, non-invasive and rapid quantifier of severity of circulatory shock states.

## Patients and methods

Intraoperative data were acquired in 61 patients who underwent emergency surgery for massive haemorrhage. Data were gathered prospectively (16 measurements of MAP and pet CO<sub>2</sub>) in the Department of anesthesiology, intensive care medicine and for the treatment of pain, University Clinical Centre Maribor from June to December 2006. The operative procedures were:

21 laparotomies, 19 explorations of the extremities and/or major fractures, 12 craniotomies, 9 thoracotomies and 1 neck exploration. On admission we calculated for all patients (with the standard protocol) the value of two trauma scores: Injury Severity Score (ISS) and Trauma Score (TS). (13,14) Patients with lung trauma were excluded by CT scan and CT angiography. Routine intraoperative monitoring (continuous electrocardiography, pulse oximetry, direct arterial pressure via cannula and body temperature measured in the bladder) was performed. Systemic vascular resistance was measured with a Swan Ganz catheter. We used intermittent positive pressure ventilation (with sedation, analgesia and relaxation). Pet CO<sub>2</sub> was continuously measured at the tip of the endotracheal tube by using a previously calibrated capnograph

(Propaq encore – Model 202 EL, Protocol Systems INC, Beaverton, Oregon, USA). MAP was measured using the invasive method (Schiller Physiogard, TM 910, France). Arterial blood gasses were obtained every 30 – 60 minutes during the procedure. For fluid resuscitation we used 0,9% saline, Ringer solution, plasma expanders (6% HES and Voluven), fresh frozen plasma and concentrated erythrocytes. We compared initial data on admission to the operating room with those collected after adequate hemodynamical stability for each patient was achieved. Unless otherwise indicated, summary values are expressed as the mean +/- SD (standard deviation). The measurements of MAP and pet CO<sub>2</sub> were distributed normally. Categorical variables were compared using the Fisher exact test and continuous variables using the Wilcoxon rank sum test. To quantify the association between two variables (relationship between MAP and pet CO<sub>2</sub> and between pet CO<sub>2</sub>, HCO<sub>3</sub><sup>-</sup> and base excess (BE) we determined the Pearson correlation coefficient (r). We considered p < 0.05 to be significant.

Statistical calculations were performed using SAS version 8.2 (SAS Institute, Cary, NC) Systemic vascular resistance wasn't determined in the early stage of treatment.

The study was approved by the Ethical Committee of the Ministry of Health in Slovenia.

## Results

A total of 216 simultaneous measurements were analyzed (61 patients; 45 male patients). The mean ISS (see methods section) was 24,3 +/- 11,3, and mean Trauma Score (see methods section) was 8,6 +/- 2,2. The average MAP on admission (95 +/-16 mmHg) was significantly lower than the average MAP after adequate hemodynamic stability was achieved (final value after surgical treatment: 131 +/- 18 mmHg; p=0.03. The average partial pressure of pet CO<sub>2</sub> on admission (31 +/- 5 mmHg)

was significant lower than the average pet CO<sub>2</sub> after adequate hemodynamic support (final value after surgical treatment: 44 +/- mmHg; p=0.02).

The average  $\Delta$  Et CO<sub>2</sub> was 5,3 ± 2,8 mmHg and the average  $\Delta$  MAP was 28,8 +/-12,8 mmHg.. The average difference in MAP of 61 patients was: ( $\Delta$ MAP) = 5.94 (+/-0.85 ) X  $\Delta$  pet CO<sub>2</sub> (r=0.68; p=0.001) The average  $\Delta$  BE was -3,1 +/- -1,2 and average  $\Delta$ HCO<sub>3</sub><sup>-</sup> was 1,5 +/- 0,6 (between value on admission and value after surgical treatment). Pet CO<sub>2</sub> correlated with BE (r = 0,73; p=0.001) and HCO<sub>3</sub><sup>-</sup> (r = 0,64; p=0.01).

## Discussion

In steady state conditions alveolar CO<sub>2</sub> elimination and therefore pet CO<sub>2</sub> depend on CO<sub>2</sub> production and on alveolar ventilation and pulmonary perfusion (cardiac output). (4,15) Previous studies have found that pet CO<sub>2</sub> correlates with cardiac output. (4-7) In the setting of cardiac arrest it can aid identifying restoration of spontaneous circulation. (2,16) In addition pet CO<sub>2</sub> has been shown to be a prognostic indicator of outcome following resuscitation from cardiac arrest. (3,17) Other investigators have demonstrated that pet CO<sub>2</sub> effectively tracks hemodynamic changes in low flow conditions. (8,10,18) However, little is known about pet CO<sub>2</sub> during resuscitation from hemorrhagic shock in clinical situations. In our study we observed the significant changes in pet CO<sub>2</sub> (increase) after volume resuscitation and the quantitative relationship between pet CO<sub>2</sub> and MAP (linear relationship). Guzman et al. (8) observed a rapid increase in pet CO<sub>2</sub> immediately after reinfusion of blood and restoration of oxygen delivery in an experimental study with dogs. They concluded that this increase in pet CO<sub>2</sub> reflects CO<sub>2</sub> washout and repayment of oxygen debt in addition to re-establishment of normal aerobic metabolism following restoration of systemic perfusion.

Jin et al. (7) in animal models of hemorrhagic shock, concluded that decreases in pet CO<sub>2</sub> were accompanied by decreases in Pa CO<sub>2</sub>, MAP and cardiac

index during bleeding. After reinfusion of blood pet CO<sub>2</sub>, cardiac index, MAP and Pa CO<sub>2</sub> returned to approximately baseline concentrations. Tybursky et al. (11,12) concluded that pet CO<sub>2</sub> can be useful in predicting survival and may have utility to guide intraoperative resuscitation efforts. We concluded that pet CO<sub>2</sub> can be used as a reliable non-invasive monitoring device in patients with hemorrhagic shock (especially in pre-hospital conditions) and correlates with HCO<sub>3</sub><sup>-</sup>, pet CO<sub>2</sub>, pO<sub>2</sub>, pH and BE. In our investigation the correlation between pet CO<sub>2</sub>, HCO<sub>3</sub><sup>-</sup> and BE was confirmed. We find pet CO<sub>2</sub> an important adjuvant in monitoring patients with hemorrhagic shock. In addition to conventional monitoring of heart rate, blood pressure, respiratory rate, body temperature and blood oxygen saturation we suggest pet CO<sub>2</sub> as a vital sign that should be monitored. On the basis of the results of these small pilot observational studies in hospital, we have started a large prospective study in the pre-hospital setting.

Limitations. Changes in alveolar ventilation can affect results. In patients with altered ventilation/perfusion ratios (chronic obstructive pulmonary disease, atelectasis, chronic congestive heart failure, respiratory distress syndrome) the difference between pet CO<sub>2</sub> and arterial pCO<sub>2</sub> may increase up to 20 mmHg or more because of a decrease in pet CO<sub>2</sub> stemming from non-perfused areas. (19) In addition pet CO<sub>2</sub> is typically decreased after administration of vasopressor drugs (an increase of the veno-arterial mismatch, whereby CO<sub>2</sub> returns to the arterial circuit after shunting the alveoli).

Conclusions. The present study supports on-line pet CO<sub>2</sub> monitoring as a reliable, non-invasive and continuous methods for measuring perfusion failure in the setting of hemorrhage (when minute ventilation is relatively constant). In the future, research should be done to determine accurate parameters – the values of pet CO<sub>2</sub> for each stage of haemorrhagic shock -in the pre-hospital setting.

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