ABSTRACT
Objectives. We examined the relationship between partial end-tidal CO₂ (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 CO₂ with a capnograph, direct arterial pressure via a cannula, oxygen levels via pulse oximetry and body temperature.
Results. We observed significant changes in pet CO₂ (increase) after volume resuscitation and a quantitative linear relationship between pet CO₂ and mean arterial pressure.
Conclusions. Partial end-tidal CO₂ 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 CO₂ might also be a useful guide for volume resuscitation in hemorrhagic shock, especially in the pre-hospital setting.

Keywords: end-tidal CO₂, mean arterial pressure, hemorrhagic shock, relationship

Introduction
Partial end-tidal CO₂ pressure (pet CO₂) is normally determined by CO₂ production (metabolism), alveolar ventilation, pulmonary perfusion (circulation) and V/Q matching. (1) Pet CO₂ 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 CO₂ can be used for non-invasive and continuous monitoring of cardiac output in circulatory shock. (7-10) Some authors reported the use of pet CO₂ 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 CO₂ in patients with hemorrhagic shock in the presence of constant minute ventilation end tidal (Et) CO₂ monitoring be considered a technically simple, non-invasive and rapid quantitator 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 CO₂) 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 oxymetry, 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 CO₂ was continuously measured at the tip of the endotracheal tube by using a previously calibrated capnograph.
was significant lower than the average pet CO2 after adequate hemodynamic support (final value after surgical treatment: 44 +/- mmHg; p=0.02).

The average Δ Et CO2 was 5.3 ± 2.8 mmHg and the average Δ MAP was 28.8 +/-12.8 mmHg. The average difference in MAP of 61 patients was: (ΔMAP) = 5.94 (+/-0.85) X Δ pet CO2 (r=0.68; p=0.001) The average Δ BE was -3.1 +/- -1.2 and average ΔHCO3 was 1.5 +/- 0.6 (between value on admission and value after surgical treatment). Pet CO2 correlated with BE (r = 0.73; p=0.001) and HCO3 (r = 0.64; p=0.01).

Discussion

In steady state conditions alveolar CO2 elimination and therefore pet CO2 depend on CO2 production and on alveolar ventilation and pulmonary perfusion (cardiac output). (4,15) Previous studies have found that pet CO2 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 CO2 has been shown to be a prognostic indicator of outcome following resuscitation from cardiac arrest. (3,17) Other investigators have demonstrated that pet CO2 effectively tracks hemodynamic changes in low flow conditions. (8,10,18) However, little is known about pet CO2 during resuscitation from hemorrhagic shock in clinical situations. In our study we observed the significant changes in pet CO2 (increase) after volume resuscitation and the quantitative relationship between pet CO2 and MAP (linear relationship). Guzman et al. (8) observed a rapid increase in pet CO2 immediately after reinfusion of blood and restoration of oxygen delivery in an experimental study with dogs. They concluded that this increase in pet CO2 reflects CO2 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 CO2 were accompanied by decreases in Pa CO2, MAP and cardiac index during bleeding. After reinfusion of blood pet CO2, cardiac index, MAP and Pa CO2 returned to approximately baseline concentrations. Tybursky et al. (11,12) concluded that pet CO2 can be useful in predicting survival and may have utility to guide intraoperative resuscitation efforts. We concluded that pet CO2 can be used as a reliable non-invasive monitoring device in patients with hemorrhagic shock (especially in pre-hospital conditions) and correlates with HCO3, pet CO2, pO2, pH and BE. In our investigation the correlation between pet CO2, HCO3 and BE was confirmed. We find pet CO2 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 CO2 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 CO2 and arterial PCO2 may increase up to 20 mmHg or more because of a decrease in pet CO2 stemming from non-perfused areas. (19) In addition pet CO2 is typically decreased after administration of vasopressor drugs (an increase of the veno-arterial mismatch, whereby CO2 returns to the arterial circuit after shunting the alveoli).

Conclusions. The present study supports on-line pet CO2 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 CO2 for each stage of haemorrhagic shock - in the pre-hospital setting.
REFERENCES