The Relationship between End Tidal Carbon Dioxide and Arterial Carbon Dioxide during Controlled Hypotensive Anaesthesia

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Key Words

Arterial blood pressure · Controlled ventilation · End tidal carbon dioxide · Hypotension · Middle ear surgery · Physiological dead space

Abstract

Objectives: To prospectively assess the magnitude of changes in the arterial-to-end tidal carbon dioxide gradient $[P(a-ET)CO_2]$ as well as in the ratio of physiological dead space to tidal volume ($V_{d_{phys}}/V_t$) during controlled hypotensive anaesthesia, and to evaluate whether or not ventilatory requirements remain unaltered during this procedure. **Subjects and Methods:** Twelve adult patients with American Society of Anesthesiologists' physical status I and II undergoing middle ear surgery were selected. A standard anaesthetic procedure was followed for all cases, using thiopental sodium, succinylcholine, fentanyl, atracurium and 60% N_2O in 40% oxygen supplemented with isoflurane. Mean arterial blood pressure (MAP) was reduced to 60 ± 5 mm Hg in all patients using a sodium nitroprusside infusion. The end tidal (ET) CO_2 , $PaCO_2$, MAP, peak airway pressure, plateau pressure and expiratory minute volume were recorded during a period of normal arterial blood pressure (time 1) and during hypotension (time 2). **Results:** A significant decrease in $PaCO_2$ (7%) and $ETCO_2$ (17%) from time 1 to time 2 ($p < 0.01$) was noted, as was a significant increase in $P(a-ET)CO_2$ (48%) and in the $V_{d_{phys}}/V_t$ ratio (41.17%) ($p < 0.01$) during the same period. **Conclusion:**

The decrease in $ETCO_2$ does not reflect the changes in $PaCO_2$. The larger decrease in $ETCO_2$ is mainly due to the increase in the $V_{d_{phys}}/V_t$ ratio. During anaesthesia, once normocapnia is achieved with normal arterial blood pressure, there is hardly any need to change the ventilation after initiation of controlled hypotension.

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Introduction

End tidal carbon dioxide ($ETCO_2$) is an indispensable monitor for ensuring safety in modern anaesthetic practice. However, it is not reliable for determining the adequacy of ventilation during low cardiac output because the arterial-to-end tidal carbon dioxide gradient $[P(a-ET)CO_2]$ changes during these conditions. The changes are due to alterations in the ratio of physiological dead space to tidal volume ($V_{d_{phys}}/V_t$) and that of ventilation to perfusion (V/Q) [1].

When cardiac output is low, as seen during controlled hypotensive anaesthesia, the $ETCO_2$ may decrease, leading to changes in $P(a-ET)CO_2$. These changes may lead to erroneous resetting of the parameters of ventilation. Neither the ventilatory requirements during controlled low-flow states like hypotensive anaesthesia nor the relationship between changes in perfusion at steady-state ventilation and $P(a-ET)CO_2$ have been evaluated. Hence, the objectives of the present study are to prospectively assess the magnitude of changes in $P(a-ET)CO_2$ and the $V_{d_{phys}}/$

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Vt ratio and to evaluate the correlation between ETCO₂ and mean arterial blood pressure (MAP) at steady-state ventilation during controlled hypotensive anaesthesia.

Subjects and Methods

Twelve patients aged 20–50 years undergoing elective middle ear surgery (7 men and 5 women) with physical status I and II as per the American Society of Anesthesiologists' classification were included in the study. The study was approved by the Scientific Subcommittee of the Council of Anaesthesia and Intensive Care, Ministry of Health, Kuwait.

A standard anaesthetic procedure was followed for all patients. They were premedicated orally with diazepam (0.15 mg/kg) on the morning of surgery. Standard monitoring included electrocardiogram, pulse oximeter, capnograph, non-invasive blood pressure, temperature and neuromuscular block with nerve stimulator (TOF guard, Organon). The Ohmeda modulus CD anaesthesia system was calibrated before the start of anaesthesia. Patients were induced with fentanyl (1 µg/kg), thiopental sodium (5–7 mg/kg) and succinylcholine (1.5 mg/kg), and endotracheal intubation was done after complete relaxation, followed by 60% N₂O in 40% O₂ supplemented with isoflurane as required. Neuromuscular blockade was achieved with atracurium (0.5 mg/kg) followed by intermittent boluses as indicated by the nerve stimulator. Patients were ventilated mechanically with a tidal volume of 7–10 ml/kg to achieve an ETCO₂ of 35–40 mm Hg. Peak airway pressure, plateau pressure and expiratory minute volume were measured. After achieving a steady-state ETCO₂ for 10 min, an arterial blood sample was acquired with a micro-sampler (time 1) and the blood gases were measured on a precalibrated (AVL Omni) blood gas analyzer. At the start of the surgery, an additional dose of fentanyl (1–2 µg/kg) was given, and hypotension was induced with sodium nitroprusside up to a maximum dose of 4 µg/kg/min to achieve a MAP of 60 mm Hg (± 5 mm Hg). When the blood pressure remained steady for 10 min, ETCO₂ was recorded and a second arterial sample was analyzed for blood gases (time 2). Throughout the procedure, the ventilator parameters of the patients were kept unchanged.

The P(a-ET)CO₂ was estimated at two different times with two different MAPs. Lung compliance was estimated and the Vd_{phys}/Vt ratio was calculated with Enghoff's modification of Bohr's equation and the changes were recorded. Delta ETCO₂ and delta MAP were measured. Data were analysed using Student's paired t test. All values were expressed as mean with standard deviation. $p < 0.01$ was considered significant.

Results

The mean age and the body weight of the patients were 34.5 ± 12.31 years and 63.83 ± 12.71 kg, respectively.

The PaCO₂ decreased from a mean of 41 ± 1.94 to 38.2 ± 2.16 mm Hg, which occurred between time 1 with normal blood pressure (MAP 83.7 ± 9.67 mm Hg) and time 2 with controlled hypotension (MAP 62.9 ± 2.02 mm Hg). The ETCO₂ decreased to a greater extent from a

Fig. 1. PaCO₂ and ETCO₂ during normal MAP (83.7 ± 9.6 mm Hg) (time 1) and during controlled hypotension (62.9 ± 2.02 mm Hg) (time 2) ($p < 0.01$).

mean of 37.8 ± 1.02 to 32.5 ± 1.83 mm Hg ($p < 0.01$) during this period (fig. 1). The P(a-ET)CO₂ increased significantly from a mean of 3.2 ± 1.7 to 6.7 ± 2.3 mm Hg ($p < 0.01$) (fig. 2).

When the MAP decreased by 25%, PaCO₂ decreased 7% and ETCO₂ decreased by 17%, while the P(a-ET)CO₂ gradient increased by 48%. This increase in P(a-ET)CO₂ gradient was statistically significant ($p < 0.01$) (fig. 3).

There was no change in lung compliance. The Vd_{phys}/Vt ratio increased significantly from 0.07 to 0.17 (41.17%) ($p < 0.01$) (table 1). No correlation between MAP and ETCO₂ ($R = 0.57$) was found.

Discussion

In this study, we investigated the impact of induced hypotension on the gradient between PaCO₂ and ETCO₂. The data showed that during MAP reduction from 83.7 ± 9.67 to 62.9 ± 2.02 mm Hg, the gradient between PaCO₂ and ETCO₂ is increased twofold. We used a moderate degree of hypotensive anaesthesia with the MAP constantly maintained between 60 and 65 mm Hg. The level of MAP produced satisfactory operating conditions. The changes in ETCO₂ in our study did not accurately predict the changes in PaCO₂. Various studies have suggested that ETCO₂ does not provide a stable reflection of PaCO₂ in certain clinical situations [2–4]. Moderate controlled hypotension under anaesthesia may cause a modest fall in cardiac output. As the pulmonary blood flow decreases due to the fall in cardiac output, it causes a loss of homeostasis at the alveolar/capillary level and a ventilation/perfusion mismatch. An increase in V/Q may cause an

Fig. 2. P(a-ET)CO₂ values for the individual patients during normal MAP (83.7 ± 9.6 mm Hg) (time 1) and during controlled hypotension (62.9 ± 2.02 mm Hg) (time 2) (p < 0.01).

Fig. 3. Changes in PaCO₂, ETCO₂ and P(a-ET)CO₂ at two different MAPs. P(a-ET)CO₂ increased significantly (p < 0.01).

Table 1. Lung compliance and Vd_{phys}/Vt ratio during general anaesthesia with normal MAP (time 1) and with controlled hypotension (time 2)

	Time 1	Time 2
MAP, mm Hg	83.7 (9.67)	62.9 (2.02)
Lung compliance, ml/cm H ₂ O	25.99 (5.07)	25.71 (4.71)
Vd _{phys} /Vt ratio	0.07 (0.04)	0.17 (0.05)

Results are shown as means with the SD in parentheses.

increase in the amount of shunted blood, which decreases ETCO₂ and increases the gradient if the arterial CO₂ does not change appreciably. Since, in our study, PaCO₂ remained in the clinical range, we did not alter the ventilator settings. A rise in the V/Q ratio, such as may occur following a fall in pulmonary blood flow, can cause an increase in physiological dead space. In this study, the Vd_{phys}/Vt ratio doubled during the controlled hypotensive period. An increase in P(a-ET)CO₂ gradient also occurs when dead space ventilation increases.

Although capnography is a standard monitoring technology, traditional monitoring of arterial blood gases is also required to prevent hypoventilation during controlled hypotensive anaesthesia, where the physiological shunt and alveolar dead space are high [5]. Nevertheless, monitoring of ETCO₂ identifies decreases in cardiac output instantly during low-flow and circulatory shock states [6]. Further studies measuring cardiac output instead of arterial blood pressure during controlled hypotensive anaesthesia are required.

Conclusion

During anaesthesia, once normocapnia is achieved with normal arterial blood pressures, there is hardly any need to decrease ventilation after induction of controlled hypotension. ETCO₂ does not reflect changes in PaCO₂, because as P(a-ET)CO₂ is increased, PaCO₂ remains in the clinically acceptable range. The larger decrease in ETCO₂ during controlled hypotension is mainly due to the increase in the Vd_{phys}/Vt and V/Q ratios.

References

- Shankar KB, Moseley H, Kumar Y: Relationship between PaCO₂-PETCO₂ gradient and physiological dead space. *Can J Anaesth* 1991; 38:1072-1074.
- Russell GB, Graybeal JM: The arterial to end-tidal carbon dioxide difference in neurosurgical patients during craniotomy. *Anesth Analg* 1995;81:806-810.
- Shankar KB, Moseley H, Kumar Y, Vemula V: Arterial to end tidal carbon dioxide tension difference during caesarean section anaesthesia. *Anaesthesia* 1986;41:698-702.
- Casati A, Salvo I, Torri G, Calderini E: Arterial to end-tidal carbon dioxide gradient and physiological dead space monitoring during general anaesthesia: Effects of patients' position. *Minerva Anestesiologica* 1997;63:177-182.
- Eckenhoff JE: Deliberate hypotension. *Anesthesiology* 1978;48:87-88.
- Jin X, Weil MH, Tang W, Povoas H, Pernat A, Xie J, Bisera J: End-tidal carbon dioxide as a noninvasive indicator of cardiac index during circulatory shock. *Crit Care Med* 2000;28: 2415-2419.