COMPARISON OF END-TIDAL AND ARTERIAL CARBON DIOXIDE MEASUREMENTS DURING ANAESTHESIA WITH THE LARYNGEAL MASK AIRWAY

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SUMMARY

We have confirmed the value of measurement of end-tidal carbon dioxide concentration as an indicator of arterial carbon dioxide tension during the use of the laryngeal mask airway in healthy patients breathing spontaneously. The mean difference between arterial and end-tidal carbon dioxide tension was 0.52 kPa (range 0-1.5 kPa), which is similar to the difference which has been reported when a tracheal tube has been used. (Br. J. Anaesth. 1993; 71: 734-735)

KEY WORDS

Amongst the advantages of the use of the laryngeal mask airway (LMA) is the ability to measure end-tidal carbon dioxide concentration, which has always been difficult when the face mask is used.

Measurement of end-tidal carbon dioxide concentration in patients breathing spontaneously with a tracheal tube has been examined in the past and the relationship between arterial and end-tidal carbon dioxide has been described [1]. In this system, there is an airtight seal between the tube and the patient, while with the LMA the orifice and cuff are opposed to the larynx and the reliability of the seal is always in doubt. If air entrainment or mixing occurs, the accuracy and reliability of the end-tidal carbon dioxide measurement must be questioned. We have therefore compared the end-tidal carbon dioxide partial pressure (PECO2) during anaesthesia with an LMA and that in arterial blood (PACO2).

METHODS AND RESULTS

After obtaining local Ethics Committee approval and informed patient consent, we studied patients undergoing routine minor operations without respiratory disease who would normally be anaesthetized using an LMA. Premedication and induction were as designated by the anaesthetist in charge, as were the inhalation agent and breathing system. A size 3 mask for females and size 4 for males was used generally. The LMA was inserted, positioned and tested as recommended in the instruction manual. The carbon dioxide analyser was attached directly to the proximal end of the LMA; a catheter mount was not used. When a reasonably steady state of anaesthesia was reached, a blood sample was obtained from the radial artery for analysis (Instrumentation Laboratory 1312 Blood Gas Manager). The analyser underwent two-point calibration before each study and had an accuracy to within 0.02 kPa of carbon dioxide. PACO2 was compared with the simultaneous end-tidal carbon dioxide partial pressure measured digitally from the plateau phase on a Hewlett-Packard capnograph (Hewlett-Packard Component Monitoring System, M1049-90001), in keeping with usual clinical practice. The capnograph (a mainstream analyser), was auto-calibrated before each study using the two standard cells containing known concentrations of carbon dioxide. Its stated accuracy is ±0.15 kPa.

Data were analysed using Statview 4.0 (Abacus Concepts Inc). Correlation was evaluated using Fisher's R-Z method and the data were assessed further using the method of Bland and Altman [2]. Multiple regression analysis (Statview 4.0) was used to determine the influence of the other factors including ASA grade, weight, age and smoking habit.

We studied 122 patients (30 male, 92 female; mean weight 67 kg (range 43-120 kg); 94 were ASA I, 23 ASA II and five ASA III; 32 known smokers).

Propofol was used as induction agent in all but seven patients and isoflurane as the inhalation agent in all patients. Analgesics used included opioids, non-steroidal anti-inflammatory agents and local anaesthetic techniques. Sixty-one patients were anaesthetized using the Magill breathing system, 41 with the Bain system and 20 with the circle system.

Mean (SD) PACO2 was 6.50 (0.9) kPa (range 4.85-9.70 kPa). Mean PECO2 was 5.96 (0.92) kPa (range 4.25-8.40 kPa). Mean arterial to end-tidal carbon dioxide difference (PACO2 - PECO2) was 0.52 (0.32) kPa (range 0-1.5 kPa).

The correlation coefficient between PECO2 and PACO2 was 0.923 (Fisher's R-Z statistic, P < 0.0001).
Using the method of Bland and Altman [2] comparing the mean and the difference of the two methods, the correlation was 0.027. When the correlation was assessed with each breathing system, no obvious difference emerged. The difference between arterial and end-tidal carbon dioxide was evaluated also by multiple regression to assess the influence of ASA grade, weight and age, but there was no obvious disparity. Smoking in these predominantly ASA I and II patients did not influence the results.

**COMMENT**

The majority of patients in whom anaesthesia with spontaneous breathing is used are ASA I and II and this study suggests that end-tidal carbon dioxide measured at the end of the LMA correlates well with arterial carbon dioxide in these patients.

In normal lungs, the end-tidal gas is considered to be in equilibrium with pulmonary capillary blood and this assumption is extrapolated to use the partial pressure of end-tidal carbon dioxide ($P_{ET\text{CO}_2}$) as an indirect measure of arterial carbon dioxide ($P_{ACO}_2$). This is not necessarily correct, and several studies have examined this during both spontaneous and artificial ventilation. In these studies, a tracheal tube has usually been used and the measurements of end-tidal carbon dioxide taken from the proximal end of the tracheal tube [1, 3]. Measurement of end-tidal carbon dioxide while a face mask is in place is feasible, but unreliable, and is not applicable in clinical practice.

Nunn and Hill studied anaesthetized patients with an intubated trachea and showed that the mean (SD) ($P_{ACO}_2 - P_{ET\text{CO}_2}$) was 0.61 (0.33) kPa and this appeared to be independent of the mode of ventilation [1]. This value was comparable to that of 0.52 (0.32) kPa found in this study, in which patients breathed spontaneously.

The results obtained by Nunn and Hill [1] have been corroborated in some studies and refuted by others [3—6]. These studies involved small numbers of predominantly ASA III patients undergoing anaesthesia, or critically ill patients in Intensive Care. This is hardly surprising, for several reasons. Measurement problems include difficulty in achieving and recognizing a genuine end-tidal carbon dioxide plateau in patients with significant lung pathology, so there is no measurable end-tidal value. This problem may be accentuated if a side-stream analyser is used, when ventilatory pattern and frequency may result in mixing which influences the ability of the analyser to determine an end-tidal value. The pathophysiology of many lung diseases is likely to alter the relationship between arterial blood-gas tension and representative capillary tensions, and the relationship between the capillary tensions and the end-tidal value. The assumption that end-tidal gas tensions represent capillary gas tensions and that these represent arterial blood-gas tensions is not valid. Data from critically ill patients demonstrate that there is no predictable relationship between arterial and end-tidal carbon dioxide tensions [5, 6] and, while it has been suggested that the trend values may be helpful, Hoffman and colleagues have shown that the use of $P_{ET\text{CO}_2}$ as a monitor of $P_{ACO}_2$ as a trend value is misleading, even after the establishment of an initial ($P_{ACO}_2 - P_{ET\text{CO}_2}$) gradient [6].

It is reasonable to assume that the end-tidal carbon dioxide values in the presence of lung disease are as unreliable with an LMA as with a tracheal tube. However, we have demonstrated that, in anaesthetized ASA grade I and II patients breathing spontaneously via an LMA, $P_{ET\text{CO}_2}$ is a reasonable indicator of $P_{ACO}_2$. Measurement of end-tidal carbon dioxide did not appear to be disturbed functionally by the absence of the airtight seal which is present when a tracheal tube is used.

**REFERENCES**