ORIGINAL ARTICLE

End-Tidal to Arterial Carbon Dioxide Partial Pressure Difference During Craniotomy in Anaesthetised Patients

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SUMMARY

This study to evaluate the relationship between end-tidal carbon dioxide pressure ($ETCO_2$) and arterial partial pressure of carbon dioxide ($PaCO_2$) included 35 patients between the ages of 18 and 65 years, ASA grade 1 and 2, who had elective craniotomies. Measurements of $PaCO_2$ and $ETCO_2$ were taken simultaneously: 1) 10 minutes after induction of general anaesthesia, 2) after cranium opening prior to dural incision, 3) start of dural closure. There was significant correlation between $ETCO_2$ and $PaCO_2$ (correlation coefficient: 0.571, 0.559 and 0.629 respectively). The mean (SD) difference for $PaC0_2$ and $ETCO_2$ were: 3.84 (2.13), 4.85 (5.78) and 3.91 (2.33) mmHg respectively. Although there was agreement, the bias is of significant clinical importance. In conclusion, we find that $ETCO_2$ consistently underestimated the value of $PaCO_2$ during craniotomy.

KEY WORDS:
End-tidal carbon dioxide pressure, Arterial partial pressure of carbon dioxide, Craniotomy

INTRODUCTION

Capnography, which is based on the measurement of end tidal carbon dioxide ($ETCO_2$), is a well-established method for intraoperative monitoring of respiratory function during routine anaesthesia[1]. $ETCO_2$ refers to the partial pressure of carbon dioxide at the end of expiration and reflects arterial carbon dioxide tension ($PaCO_2$) [1–3]. The usual reported difference between PaCO2 and $ETCO_2$ is approximately 2.0 to 5.0 mmHg in a healthy adult with the latter being lower [1].

The ability to control $PaCO_2$ is vital during neurosurgical procedures as this affects intracranial pressure dynamics. Increased $PaCO_2$ could cause an increase in cerebral blood volume with resulting intracranial hypertension and decreased cerebral perfusion pressure[4]. Therapeutic hyperventilation is often used to lower intracranial pressure during craniotomies before the dura is opened. Regional cerebral tissue hypoxia could result if hyperventilation decreases $PaCO_2$ to 20 mmHg or less[4]. If the gradient between arterial and end-tidal carbon dioxide partial pressure is within clinical limits, then $ETCO_2$ could be used reliably to follow respiratory acid-base status and assist in the titration of hyperventilation therapy, as well as decrease the expense and time involved in frequent arterial blood sampling.

During craniotomies, there is a doubt if capnography alone is adequate to monitor pulmonary ventilation to achieve targeted $PaCO_2$. The difficulty lies in not knowing how much the expected gradient between $ETCO_2$ and $PaCO_2$ is. Russell reported that in mechanically ventilated neuro-intensive care patients, there is significant variability in the relationship between $PaCO_2$ and $ETCO_2$ [6]. The $P(a\text{-}ET)CO_2$ was reported on average to be 6.9 ± 4.4 mmHg. Russell studied neurosurgical patients undergoing craniotomies and found that $ETCO_2$ did not provide a statistically stable estimation of $PaCO_2$ in mechanically ventilated patients[6]. However, another study which determined $P(a\text{-}ET)CO_2$ in head injury patients, found that $ETCO_2$ was an accurate reflection of $PaCO_2$ [7]. Most studies suggested that $ETCO_2$ will not show consistent results if used to estimate $PaCO_2$ for neurosurgical patients. The objective of this study was to determine the relationship between the arterial partial pressure of carbon dioxide ($PaCO_2$) and end-tidal carbon dioxide partial pressure ($ETCO_2$) measured in neurosurgical patients during craniotomy, and to determine if $ETCO_2$ could be used to estimate $PaCO_2$ during craniotomy.

MATERIALS AND METHODS

This was a prospective study carried out in Hospital Universiti Kebangsaan Malaysia over one year after obtaining approval from the Dissertation / Ethics Committee of the Department of Anaesthesiology and Intensive Care, Faculty of Medicine, Universiti Kebangsaan Malaysia.

Thirty five patients, after giving informed consent were enrolled in this study. Inclusion criteria were: ASA grade 1 and 2, ages from 18 to 65 years, and scheduled for elective craniotomies in the supine position. Exclusion criteria were: patients with suspected difficult airway and a history of heavy smoking. Standard monitoring included: electrocardiography, pulse oximetry, central venous pressure and invasive arterial blood pressure.

Anaesthesia was induced with intravenous fentanyl 2 $\mu g.kg^{-1}$ and propofol 2 $mg.kg^{-1}$. Rocuronium 0.6 $mg.kg^{-1}$ was used to facilitate tracheal intubation. After adequate jaw relaxation was achieved, an appropriate sized cuffed tracheal tube was inserted and intermittent positive pressure ventilation was instituted using a volume-controlled mode with a tidal volume of 7-10 $ml.kg^{-1}$and a respiratory rate of 10-12 breaths per minute. General anaesthesia was maintained with oxygen (40-50%), air and sevoflurane (MAC 0.8-1.0).

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Table I: Patient Demographic data. Values are mean (SD)

	n=35
Age: years	45.9 (11.5)
Weight: kg	63.4 (9.1)
Height: m	160.9 (6.5)
Sex: M : F	20 : 15
Race: Malay : Chinese : Indian	23 : 10 : 2
ASA grade: 1 : 2	17 : 18

Table II: Cardio-respiratory variables during craniotomy and surgical details. Data are mean (SD)

	10 min after induction	After cranium opening prior to dural incision	Start of dural closure
PaO_2; mmHg	182 (38)	147 (41)	146 (42)
Mean arterial pressure; mmHg	87 (8)	83 (12)	81 (7)
Heart rate; beat $min.^{-1}$	77 (17)	84 (12)	85 (11)
Respiratory rate; breath $min.^{-1}$	10 (1)	11 (1)	10 (1)
Peak airway pressure; mmHg	18 (4)	26 (4)	18 (5)
Expired tidal volume; ml	464 (86)	465 (86)	462 (86)
Temperature; ° C	35.9(1.3)	35.5 (0.6)	35.6 (0.6)
Duration of operation; hour		4.14 (1.7)	
Estimated blood loss; ml		742 (405.9)	

Table III: Arterial and end-tidal carbon dioxide values, average and difference values are mean (SD).

	Ten minutes after induction n=35	After craniotomy before dural incision n=35	Start of dural closure n=35
$PaCO_2$; mmHg	34.6 (3.2)	36.3 (3.0)	36.9 (2.5)
$ETCO_2$; mmHg	31.0 (3.4)	32.7 (3.2)	33.0 (3.4)
Average of two methods; mmHg	32.9 (2.9)	34.6 (3.0)	34.0 (6.0)
($PaCO_2$ - $ETCO_2$); mmHg	3.8 (2.1)	4.9 (5.8)	3.9 (2.3)

Table IV: Correlation between $PaCO_2$ and $ETCO_2$ during craniotomy

$PaCO_2$ / $ETCO_2$	Correlation coefficient	P value
10 min after induction	0.571	< 0.01
After craniotomy before dural incision	0.559	< 0.01
Start of dural closure	0.629	< 0.01

Following induction of anaesthesia, a 20 G cannula was inserted for invasive arterial pressure monitoring. Three samples of arterial blood gases were taken at 10 minutes after induction of general anaesthesia and tracheal intubation (baseline), after craniotomy prior to dural incision and at the beginning of dural closure. Additional analyses of arterial blood gases were performed at the discretion of the attending anaesthesiologist when clinically indicated.

The $PaCO_2$ was measured from arterial blood sample using the blood gases analyser (ABL Radiometer Copenhagen) and corrected to a temperature of 37º C. The $ETCO_2$ was recorded simultaneously at the time of each arterial blood gas sampling using a side-stream capnometer (capnometry module, Kion, M-CAIOV.01). The P(a-ET)CO_2 was calculated for each arterial blood gas sample.

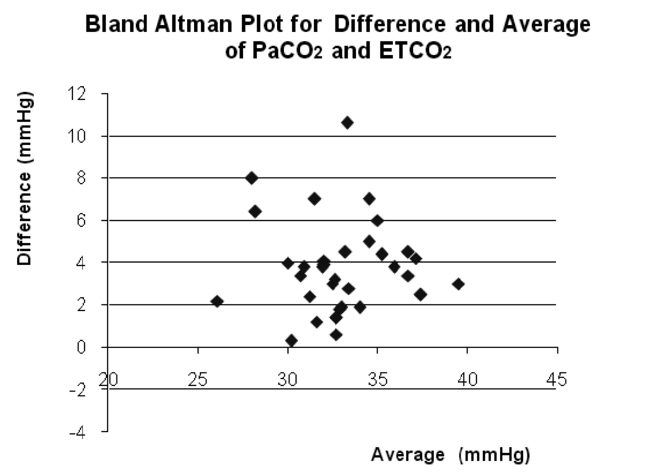
Heart rate, blood pressure, respiratory rate, tidal volume, peak inspiratory pressure and PaO_2 were recorded at each sampling time.

Data were initially analyzed using Pearson's Correlation to see the relationship between $PaCO_2$ and $ETCO_2$ at different stages of the operation. A P value of < 0.05 was considered significant. The agreement between the measures of CO_2 was assessed using Bland-Altman method, where mean difference and average between $PaCO_2$ and $ETCO_2$ were calculated. The 95% limits of agreement were also displayed.

RESULTS

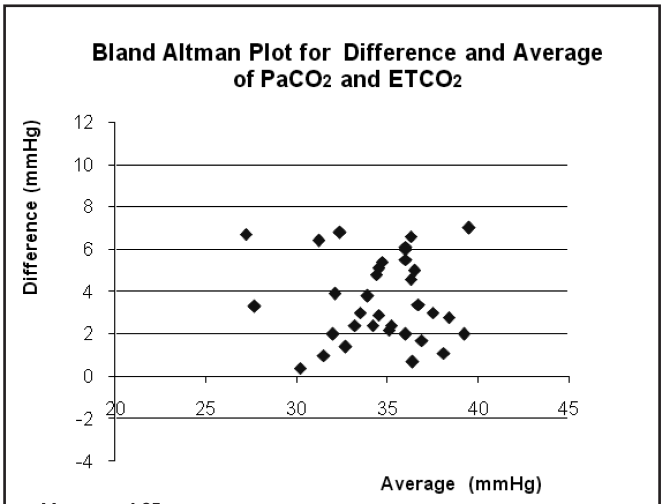
A total of thirty five patients were studied. Demographic data of patients are shown in Table I. Cardio-respiratory physiological variables are summarized in Table II. Table III shows the end-tidal to arterial carbon dioxide values. Table IV shows significant correlation between $PaCO_2$ and $ETCO_2$ at all three sampling times.

Figures 1-3, show Bland-Altman plots to demonstrate agreement between the two methods of measuring carbon dioxide partial pressure. The calculated mean difference, standard deviation and 95% limits of agreement are presented. The greatest mean difference occurred just prior to dural incision and was 4.85 mmHg. The 95% agreement was from - 1.8 to 12 mmHg. The lowest mean difference was at 10 minutes after induction and was 3.84 mmHg. The 95% agreement was from - 0.34 to 8.02 mmHg.



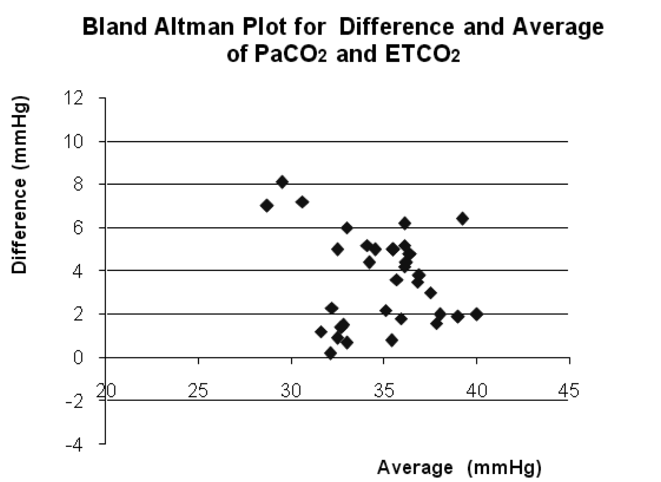
Mean difference : 3.84
SD : ± 2.13
Upper limit of agreement: 8.02
Mean + 1.96 (SD)
Lower limit of agreement:-0.34
Mean - 1.96 (SD)

Fig. 1: Bland-Altman plot for differences and average of $PaCO_2$ and $ETCO_2$ a 10 minutes after induction.



Mean : 4.85
SD :± 5.82
Upper limit of Agreement : 12.00
(Mean +1.96 (SD))
Lower limit of Agreement :-1.80
(Mean -1.96 (SD))

Fig. 2: Bland-Altman Plot for the difference and average of $PaCO_2$ and $ETCO_2$ after craniotomy before dural incision.



Mean : 3.91
SD : ±2.33
Upper limit of Agreement : 8.48
(Mean +1.96 (SD))
Lower limit of Agreement :-0.66
(Mean -1.96 (SD))

Fig. 3: Bland-Altman plot for the differences and average of $PaCO_2$ and $ETCO_2$ during start of dural closure.

DISCUSSION

Although arterial blood gas measurement of $PaCO_2$ is the gold standard for monitoring changes in CO_2, it is invasive, expensive and provides only intermittent measures of $PaCO_2$. $ETCO_2$ provides continuous respiratory monitoring. Many anaesthetists rely on $ETCO_2$ to predict the $PaCO_2$ during craniotomy in order to minimize arterial blood sampling. However, the ability of $ETCO_2$ changes to predict the direction of changes in $PaCO_2$ is questionable, since previous studies showed inconsistency in their findings [6-8].

In this study, values of $PaCO_2$ and $ETCO_2$ were shown to be significantly correlated throughout the craniotomy operation. $PaCO_2$ always exceeded $ETCO_2$ and there were no difference that went in opposite directions. These results are in contrast with those observed by Russell during craniotomy[6]. Interestingly, a study done by Kerr in severe head injury patients showed that $ETCO_2$ correlated well with $PaCO_2$ only in patients without respiratory complications or without spontaneous breathing[8]. Russell had studied neuro-intensive care patients and found consistent relationship between $PaCO_2$ and $ETCO_2$[6]. However, Russell was doubtful, and among many discrepancies he found the $P(a\text{-}ET)CO_2$ differences became bigger at higher average of $PaCO_2$ and $ETCO_2$. In this study, the correlation of $PaCO_2$ with $ETCO_2$ was significant at different times during the operation. In contrast to these results, Grainier reported that $P(a\text{-}ET)CO_2$ was unstable over time, when the procedure took more than three hours[9].

Further assessment using Bland Altman method showed agreement between those two methods of measuring carbon dioxide partial pressure. Although there was agreement, the authors feel that, the bias is of significant clinical importance. We did not combine the repeated measures of $PaCO_2$ and $ETCO_2$ for analysis as such a technique would result in invalid conclusions[5]. During craniotomies, Russell found $P(a\text{-}ET)CO_2$ to be 7.2 ± 3.3 mmHg[4]. He found $P(a\text{-}ET)CO_2$ to be 5.47 ± 5.51 mmHg in post cardiac surgery, 11 and 6.9 ± 4.4 mmHg in mechanically ventilated neurointensive care patients[6]. It may be difficult to relate these results which are higher compared with results for neurosurgical patients in this study. Instability of intra operative cardiac and pulmonary status might explain these inconsistent results.

Other researchers in various clinical situations also demonstrated inconsistent results. Shanker recorded $P(a\text{-}ET)CO_2$ to be 1.9 to 2.4 mmHg during laparoscopic surgery in pregnancy at various stages during the operation[12]. During caesarean section under general anaesthesia, Shanker[13] found the P(a-ET)CO_2 was 0.03 to 0.78 mmHg and Rudolph *et al*[14] found P(a-ET)CO_2 to be 5.5 to 6.9 mmHg at different phases during early recovery from general anaesthesia.

Significant differences between $PaCO_2$ and $ETCO_2$ were seen at all stages in this study, which may not be clinically acceptable. These findings are consistent with the results from previous studies [6-8]. The difference can be explained with the theories of dead space, shunt and ventilation-perfusion mismatch (V/Q mismatch). Even in the healthy lung, the $PaCO_2$ and $ETCO_2$ difference is not zero. V/Q mismatching can occur in patients who are given general anaesthesia or have lung diseases. Fresh gas entrainment during side-stream gas sampling tends to dilute expired carbon dioxide tension and contributes further to errors during measurement of $ETCO_2$.

Other reasons for the variation in the difference could be attributed to equipment error, such as calibration of the equipment and the temperature at which the blood gases were analyzed, as these factors may affect the accuracy of the readings. Chan found that main-stream capnometry provided a more accurate estimation of $PaCO_2$ compared with side-stream capnometry[15]. Sitzwoh *et al* observed increased mean difference of 2.5 fold from 4.1 to 10.4 mmHg during mild to moderate hypothermia when CO_2 determinations were not temperature corrected [16]. Grenier et al found lateral positioning increases the mean difference compared with supine, prone and sitting positions[9].

As a result of different clinical scenarios, many variations and differences between $PaCO_2$ and $ETCO_2$ are seen during anaesthesia. Although many studies showed that we can rely on capnography to estimate $PaCO_2$ values, we recommend ABG measurement in conjunction with capnography. Furthermore, electrolytes, glucose and lactate levels can be measured in the blood samples. This contributes to safe clinical management of patients.

In conclusion, significant correlation was found between $PaCO_2$ and $ETCO_2$ at various stages of craniotomy and both methods were in agreement with each other throughout the operation. Although there was agreement, the authors feel that, the bias is of significant clinical importance. $ETCO_2$ consistently underestimates the value of $PaCO_2$ during craniotomy.

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