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USE OF END TIDAL CO_2 AS AN ALTERNATIVE TO PARTIAL PRESSURE OF CO_2 IN PATIENTS UNDERGOING CRANIOTOMY

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ABSTRACT

OBJECTIVE: To determine the relationship between partial pressure of CO_2 ($PaCO_2$) and end tidal carbon dioxide ($ETCO_2$) during craniotomies in supine position.

METHODS: This prospective observational study included adult patient above 18 years of age. Samples of ABG for $PaCO_2$ were taken at 10 minutes after induction of general anesthesia, after craniotomy and prior to dural incision, beginning of dural closure and before extubation and $ETCO_2$ was recorded simultaneously. Correlation and agreement between $ETCO_2$ and $PaCO_2$ were analysed and Bland Altman plot was constructed. $P < 0.05$ was considered significant.

RESULTS: Seventy patients were included with mean age of 44.2±11.9 years. Mean $ETCO_2$ measured was lower than the corresponding $PaCO_2$. A significant (p-0.000) positive correlation exists between the $PaCO_2$ and $ETCO_2$. Bland Altman plot showed good agreement between $PaCO_2$ and $ETCO_2$ values.

CONCLUSION: We conclude that $ETCO_2$ can be used as an alternative of $PaCO_2$ for neurosurgical procedures in healthy patients.

KEYWORDS : End Tidal Carbon Dioxide, Partial Pressure Of Co_2, Craniotomy, Neuroanaesthesia.

INTRODUCTION

The ability to control partial pressure of CO_2 ($PaCO_2$) is vital during neurosurgical procedures as this affects intracranial pressure (ICP) dynamics. Increased $PaCO_2$ could cause an increase in cerebral blood volume with resulting intracranial hypertension and decreased cerebral perfusion pressure (CPP). [1] Therapeutic hyperventilation ($PaCO_2$ of 25-30 mmHg) is often used to lower intracranial pressure during craniotomies before the dura is opened. Hyperventilation can effectively reduce ICP by reducing cerebral blood flow and volume. However regional cerebral tissue hypoxia could result if hyperventilation decreases $PaCO_2$ to 20 mmHg or less which makes measurement of $PaCO_2$ inevitably important during neurosurgery. Measurement of $PaCO_2$ by Arterial blood gas (ABG) analysis has been considered as the gold standard. [2] However measurement of $PaCO_2$ by ABG is an invasive method and not easily available in the operating room. Also continuous monitoring of $PaCO_2$ is not feasible.

Capnography is based on the measurement of end tidal carbon dioxide ($ETCO_2$). $ETCO_2$ refers to the partial pressure of carbon dioxide at the end of expiration and closely reflects $PaCO_2$. [3-5] The usual reported difference between $PaCO_2$ and $ETCO_2$ is approximately 2.0 to 5.0 mmHg in a healthy adult with the latter being lower. [3] If the gradient between arterial and end-tidal carbon dioxide partial pressure is within clinical limits, then $ETCO_2$ could be used reliably to assist in the titration of hyperventilation therapy. Nunn and Hill have suggested that during anaesthesia in healthy subjects the difference of $PaCO_2$ and $ETCO_2$ is sufficiently constant and $ETCO_2$ can to be used as an alternative method for assessment of arterial CO_2.[6]

The aim of this study is to determine the relationship between $PaCO_2$ and $ETCO_2$ during craniotomies, and to determine if $ETCO_2$ can be used as an alternative to $PaCO_2$.

METHODS

This prospective observational study was conducted in a neurosciences department of a tertiary care hospital after obtaining the approval of the institutional ethical committee. Adult patient above 18 years of age with American society of anaesthesiology grade I & II posted for elective craniotomy for resection of cerebral tumours under general anaesthesia from Oct 2015 to June 2016 were enrolled in the study after obtaining an informed consent. Patient with history of previous craniotomy, PEEP more than 5, blood loss more than 500 ml, operation duration more than 5 hours and operating position other than supine were excluded from the study.

Pre-anaesthetic evaluation was done on the day before surgery. Vitals were recorded on the real time monitors including electrocardiogram, pulse oximetry, capnography, non- invasive blood pressure & temperature. Pre-medication was done with an intravenous Glycopyrolate, Midazolam, Ondansetron, Hydrocortisone and Dexamethasone. Analgesia was provided with intravenous Fentanyl and Induction was done with intravenous Thiopentone sodium (2.5%) titrated to the loss of eyelash reflex and intravenous Atracurium bromide. Mask ventilation was carried out till adequate relaxation was achieved. Orotracheal intubation was done with an appropriate size endotracheal tube and intermittent positive pressure was instituted using volume control mode with tidal volume of 8-10 ml/kg and a respiratory rate of 10-14 breath per minute. All patients were positioned supine and following induction of anaesthesia, radial arterial cannulation was done for invasive arterial pressure monitoring. All patients were maintained on inhalational anaesthetic agent Sevoflurane (0.8 - 1% MAC), N_2O:O_2 (60:40), intravenous infusion of Propofol and Atracurium bromide infusion to achieve good anaesthetic plane. At the end of surgery, patients were extubated after the reversal of residual neuromuscular blockade.

Four samples of arterial blood gas were taken: at 10 minutes after induction of general anesthesia, after craniotomy and prior to dural incision, beginning of dural closure and before extubation. Values of $PaCO_2$ were measured from arterial blood gas analyser (RAPIDLab*1200, SIEMENS) corrected to a temperature of 37 degree celsius. $ETCO_2$ was recorded simultaneously at the time of arterial blood gas sampling using side stream capnometer (capnometry module, PHILIPS, IntelliVue GS-M1019A). The difference between $PaCO_2$ and $ETCO_2$ was calculated for each arterial blood gas sample.

The average elective craniotomies performed at our center in the last 3 years was 84. By using convenience sampling method, we proposed to conduct active data collection for our study (duration of 9 months). Considering enrollment of 7 patients every month with 10% consent refusal/revoke cases, the sample size was calculated for the study was 70.

Statistical analyses were done using statistical software (STATA version 12). The qualitative data were expressed in proportion and percentages and the quantitative data were expressed as mean and standard deviations. Pearson's correlation coefficient was used to find the correlation between the $ETCO_2$ and $PaCO_2$. Bland Altman analysis was done to demonstrate the agreement between the two different measures of CO_2 ($PaCO_2$ and $ETCO_2$). Bland Altman plot were also constructed displaying 95% agreement limits. $P < 0.05$ was considered significant.

RESULTS

Seventy patients were included in the study with mean age of 44.2±11.9 years (range 20-60 years) including 37 (52.8%) male and 33 (47.2%) female. Mean BMI of the study population was 24.86±3.32 kg/m^2 and majority were graded in ASA Grade II. All the patients had normal pre-operative vital parameters.

None of the patients experienced any hemodynamic abnormality during the procedure. A statistically significant ($p<0.001$) positive

correlation exists between the $PaCO_2$ and $ETCO_2$ at all the study point. (table 1) Correlation was strongest before extubation (0.86) and slightly lower at 10 min after induction (0.75). Mean $ETCO_2$ measured was lower than the corresponding $PaCO_2$ at all the study time point. (table 1) Although the difference of $PaCO_2$ and $ETCO_2$ of individual measurements were positive, negative difference were found in 1 of the patient at 10 minute after induction and start of dural closure. No significant difference exits between the mean difference between $PaCO_2$ and $ETCO_2$ [P(a–ET) CO_2] at any study time point. (table 2)

Bland Altman plot was constructed for the difference between $PaCO_2$ and $ETCO_2$ (Y-axis) and average value of $PaCO_2$ and $ETCO_2$ (X-axis) depicting mean and 95% agreement limit (figure 1). The mean and the upper and lower agreement limit of the difference between $PaCO_2$ and $ETCO_2$ was 4.19 ± 3.07 (10.23,-1.85) for values at 10 minutes of induction; 4.51 ± 2.68 (9.76, -0.74) after cranium opening prior to dural incision; 4.02 ± 2.47 (8.86, -0.82) at the start of dural closure and 3.93 ± 1.74 (7.34, 0.52) before extubation.

DISCUSSION

$ETCO_2$ measurement is currently the standard of care where general anesthesia is administered. The end-tidal CO_2 or $ETCO_2$ closely approximates the arterial PCO_2 in normal lungs. In the current study we found the measured value of $ETCO_2$ to be lower than the $PaCO_2$ with maximum and minimum difference of 4.51 ± 2.68 and 3.93 ±1.74 in hemodynamic stable adult patients posted for elective craniotomy. The usual reported difference between $PaCO_2$ and $ETCO_2$ is approximately 2.0 to 5.0 mmHg in a healthy adult with the latter being lower which is consistent with the reports of the current study. [3] The measured $ETCO_2$ is lower than the 'ideal' alveolar PCO_2, because the CO_2 free gas from the anatomical dead space dilutes and lower the $ETCO_2$. Other factors include sampling error, calibration error and leak or occlusion in sampling lines. Nunn and hill noted mean difference of 4.6 ± 2.5 mmHg between $PaCO_2$ and $ETCO_2$ which remained sufficiently constant whereas Collier et al observed it to be 0.9 ± 1.8 mmHg. [6-7] Kerr ME et al found a mean difference of 6 ± 6 mm of Hg in mechanically ventilated patients whereas Russell GB et al found it to be 7 ± 3 mm of Hg in neurosurgical patients in the operating room and 7±4 mm of Hg in ICU. [8]

In the current study we did not found any significant difference between P(a–ET)CO_2 at any of the study point with the mean values being fairly constant. Sharma SK et al. examined the stability of the P(a–ET)CO_2 in 21 patients undergoing elective craniotomies lasting greater than four hours and found no significant difference with time suggesting a constant relationship between the arterial and end-tidal PCO_2 measurements over time. [9] Askrog et al. saw a progressive increase in the difference over time in seven patients undergoing major general surgery when deliberate hypotension was induced; which they attributed to increase in dead space. [10] Yamanaka MK et al. compared the difference between $ETCO_2$ and $PaCO_2$ in 17 patients undergoing mechanical ventilation. Large differences were found between $PaCO_2$ and $ETCO_2$ in individual patients; P(a-ET)CO_2 correlated closely with the ratio of dead space ventilation to tidal ventilation. [11] The fairly constant P(a–ET)CO_2 value in the current study could be due to the absence of any hemodynamic abnormality and normal lung dynamics in our patients.

In the current study positive P(a–ET)CO_2 difference exits at all the measurements except in one patients where negative difference was noted at two point of measurement. Slow emptying of the alveoli and longer time constant has been hypothesized as the probable mechanism in earlier studies for this negative difference. Russell GB et al reported negative differences in 8% of patients after coronary artery bypass surgery while Shankar et al reported 37% in early pregnancy and 50% in pregnant patients during Caesarean section. [12-14]

Takki et al reported $PaCO_2$ and $ETCO_2$ difference of 3.5 ± 2.0 mmHg in 24 stable patients with excellent correlation between mean values of $PaCO_2$ and $ETCO_2$ in group of patient without or with lung disease during controlled ventilation using different respiratory rates and tidal volumes. [15] A statistically significant ($p<0.001$) positive correlation exists between the $PaCO_2$ and $ETCO_2$ at all the study point with maximum correlation of 0.86. Weinger MB et al. found the correlation coefficient of 0.768 ($p<0.001$) with gradient of 4.24 ± 4.42 mm Hg. [16] Grenier B et al showed a poor correlation during neurosurgical procedures and recommended arterial blood gas in addition to capnography.[17]

On further assessment using Bland Altman plot showed good agreement between $PaCO_2$ and $ETCO_2$ with a mean difference close to 4 at each study time point with $PaCO_2$ being the higher. Hussaini et al also found a good agreement between $PaCO_2$ and $ETCO_2$ in anaesthetized patients during craniotomy. The greatest mean difference observed was just prior to dural incision (4.85) and the lowest was at 10 minutes after induction (3.84). [18] In the current study the greatest difference was observed after Cranium opening prior to dural incision (4.51) and the lowest before extubation (3.94).

CONCLUSION

This study showed a good correlation and agreement between $PaCO_2$ and $ETCO_2$ values. We conclude that $ETCO_2$ can be used as an alternative of $PaCO_2$ for neurosurgical procedures in healthy patients.

TABLES

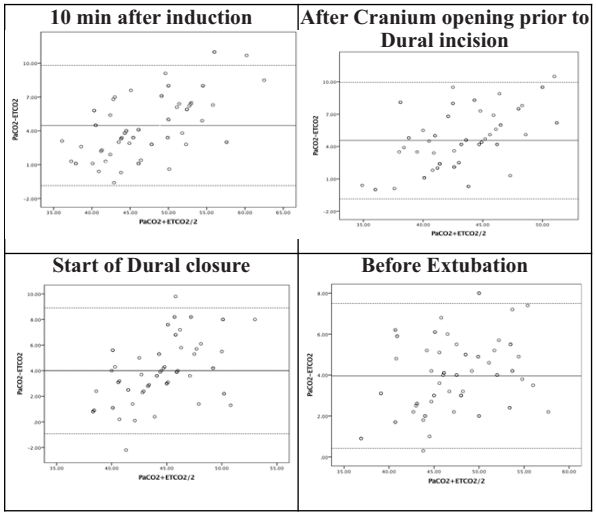
Table 1: Correlation between Mean $PaCO_2$ and $ETCO_2$ and different time points.

Time points	$PaCO_2$ (mmHg)	$ETCO_2$ (mmHg)	Correlation Coefficient	P value
10 min after induction	32.94±4.61	28.74±3.18	0.75	< 0.001
After Cranium opening prior to dural incision	30.27±3.06	25.76±2.06	0.51	< 0.001
Start of dural closure	30.96±2.81	26.94±2.04	0.52	< 0.001
Before extubation	32.72±3.45	28.79±3.07	0.86	< 0.001

Table 2: Comparison of the $PaCO_2$ – $ETCO_2$ at the different time point of surgery.

Variable	Mean difference ± SD	P value
10 in after induction vs After Cranium opening prior to dural incision	-0.320 ± 2.431	0.275
10 min after induction vs Start of dural closure	0.174 ± 2.948	0.622
10 min after induction vs Before extubation	0.260 ± 2.989	0.469
After Cranium opening prior to dural incision vs Start of dural closure	0.494 ± 2.719	0.133
After Cranium opening prior to dural incision vs Before extubation	0.580 ± 2.393	0.066
Start of dural closure vs Before extubation	0.086 ± 2.381	0.764

Figure 1: Bland Altman plots to demonstrate the agreement between the two different measures of CO_2($PaCO_2$ and $ETCO_2$) at different time point.



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