

A study of partial pressure of arterial carbon dioxide and end-tidal carbon dioxide correlation in intraoperative and postoperative period in neurosurgical patients

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ABSTRACT

Background and Aim: Monitoring carbon dioxide (CO₂) is of utmost importance in neurosurgical patients. It is measured by partial pressure of arterial CO₂ (PaCO₂) and end-tidal CO₂ (ETCO₂). We aimed to study the correlation between PaCO₂ and ETCO₂ in neurosurgical patients in the intraoperative and postoperative period on mechanical ventilation in Postanesthesia Care Unit (PACU).

Methodology: This was prospective observational study done at tertiary care teaching public hospital over a period of 1 year. We studied 30 patients undergoing elective craniotomy intraoperatively and in the postoperative period on mechanical ventilation for 24 h. Serial measurement of ETCO₂ and PaCO₂ at baseline, hourly intraoperatively and every 6 hourly in the PACU were studied. Data analysis was done using SPSS software version 20.

Results: The mean PaCO₂-ETCO₂ gradient intraoperatively over 4 h is 3.331 ± 2.856 and postoperatively over 24 h is 2.779 ± 2.932 and lies in 95% confidence interval. There was statistically significant correlation between PaCO₂ and ETCO₂ intraoperatively baseline, 1 h, 2 h, 3 h, and 4 h with Pearson's correlation coefficients of 0.799, 0.522, 0.582, 0.439, and 0.547, respectively ($P < 0.05$). In PACU at baseline, 6 h, 12 h, 18 h, and 24 h Pearson's correlation coefficients were. 534, -0.032, 0.522, 0.242, 0.592, and 0.547, respectively, which are highly significant at three instances ($P < 0.01$).

Conclusion: ETCO₂ correlates PaCO₂ with acceptable accuracy in neurosurgical patients in the intraoperative and postoperative period on mechanical ventilation in Intensive Care Unit. Thus, continuous and noninvasive ETCO₂ can be used as a reliable guide to estimate arterial PCO₂ during neurosurgical procedures and in PACU.

Key words: End-tidal carbondioxide, neurosurgery, partial pressure of arterial carbon dioxide, postoperative care unit

Introduction

In neurosurgeries monitoring of arterial partial pressure of carbon dioxide (PaCO₂) is most vital as it affects intracranial pressure (ICP), cerebral blood flow, volume and cerebral

perfusion pressure (CPP). Increase in PaCO₂ increases ICP thereby decreasing CPP. Raised ICP can be reduced through therapeutic hyperventilation; however, excessive hyperventilation (<20 mmHg) could result in regional cerebral hypoxia.^[1,2] Hence, continuous monitoring of CO₂ is of utmost importance. End-tidal CO₂ (ETCO₂) is another method to estimate CO₂ continuously and noninvasively. A good alveolar ventilation-perfusion matching results in an ETCO₂ that closely

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correlates with PaCO₂; hence in patients without significant cardiopulmonary disorders, PaCO₂ may be estimated by using actual ETCO₂ measurements. The difference between PaCO₂ and ETCO₂ (P(a-ET) CO₂ gradient) is reported to be 3.6–4.6 mmHg in healthy awake patients. However, in literature various studies mention substantial variability in patients undergoing craniotomy in different positions and mechanically ventilated neurosurgical Intensive Care Unit (ICU) patients.^[3-6] In addition, in diseased lungs, impaired cardiac function, increased dead space ventilation, ventilation-perfusion (V/Q) mismatch, sampling line error, and critical illness may widen the above gradient.^[7,8] Nevertheless, monitoring ETCO₂ has many advantages such as it reduces the need for invasive arterial blood gas (ABG) sampling, allowing safe, comfortable, and continuous monitoring. A sudden change in ETCO₂ can prompt the clinician to measure PaCO₂ via an ABG sample thus before the patient is compromised, early intervention is guaranteed.^[9] This also has important implication in Postanesthesia Care Unit (PACU) for more cautious postoperative care. There is lot of contradiction in the recent literature regarding ETCO₂ and PaCO₂ correlation in neurosurgical patients.^[1,3-7,10,11] Hence, we decided to do the present study in Indian population.

The aim was to study the correlation between PaCO₂ and ETCO₂ in patients undergoing neurosurgery in the intraoperative as well as in the postoperative period on mechanical ventilation in PACU.

Methodology

This was prospective observational study done at a tertiary care teaching public hospital in neurosurgery operation theater and PACU after approval from the Institutional Ethics Committee and written informed valid consent. The study was conducted over a period of 1 year from June 2014 to June 2015. We studied 30 patients aged between 18 and 60 years, belonging to the American Society of Anesthesiologists (ASA) Grade 1/2 undergoing elective craniotomy (surgical duration of 4–5 h) in the supine position. In the postoperative period, we included only those patients who required mechanical ventilation for minimum 24 h period. We excluded patients with lung disease and hemodynamically unstable patients.

After adequate preoxygenation and premedication, induction was done with intravenous (IV) fentanyl 2 µg/kg and thiopentone 5 mg/kg. Vecuronium 0.08 mg/kg was used to facilitate tracheal intubation. After intubation with an appropriate-sized cuffed endotracheal tube, intermittent positive pressure ventilation was given using a volume-controlled mode with a tidal volume of 7–10 ml/kg and a respiratory rate of 10–12 breaths per minute and continuous ETCO₂ was monitored using a side-stream capnometer (Patient Monitor 9000 Express side-stream CO₂, Penlon Limited, Abington, Oxon). Postinduction radial artery cannulated and baseline ABG were collected. Anesthesia was maintained with oxygen (40–50%), air and desflurane (minimum alveolar

concentration 0.8–1.0). The first postinduction measurement of ETCO₂ and PaCO₂ was taken as a baseline and then repeated for every 1 h until the end of surgery. In the postoperative period in PACU, patients were maintained on volume-synchronized intermittent mandatory ventilation (volume SIMV) with inspired oxygen fraction (FiO₂) 40–50% and adequate sedation and analgesia with titrated doses of IV midazolam and fentanyl. Continuous ETCO₂ was recorded by using a sidestream capnometer which was connected by angle piece connector in between the endotracheal tube and breathing circuit. After stabilizing the patient in PACU, a baseline measurement of ETCO₂ and PaCO₂ was recorded and thereafter every 6 hourly. Simultaneous measurement of blood pressure, heart rate, respiratory rate, central venous pressure, tidal volume, and FiO₂, peak inspiratory pressure were recorded at each sampling time. Standard calibration of sidestream CO₂ of patient monitor 9000 express was done with the same gas mixture before induction of each case as per the specifications of manufacturer.^[12]

Statistical analysis

We calculated sample size with reference to Husaini and Choy, 2008, by taking into consideration Pearson's correlation between PaCO₂ and ETCO₂, with Type I error of 0.05 and Type II error of 0.20, with power equal to 0.80, which came to be 21.^[1]

We decided to go ahead with a sample size of 30, which was appropriate for the study design and institutional settings. Quantitative data are presented with the help of mean, median, standard deviation (SD), interquartile range (IQR), minimum and maximum values. Qualitative data are presented with the help of frequency and percentage table. Data were initially analyzed using Pearson's correlation to assess the relationship between PaCO₂ and ETCO₂ at different stages of the operation. $P < 0.05$ was considered statistically significant with 95% confidence interval (CI).

Data analysis is done with the help of IBM Corp. Released 2011. IBM SPSS Statistics for windows, Version 20.0. Armonk, NY:IBM Corp.

Results

We analyzed 30 patients in the age group of 18–60 years with youngest being 23 years old and oldest 59 years and 11 of them belonged to the age group of 31–40. Among these 60% (18) were male and rest 40% (12) female with ASA Grade 1 as 40% (12) and ASA Grade 2 60% (18). There was no significant correlation of PaCO₂ and ETCO₂ values and demographic data, ASA grades. The various neurosurgeries included in the study with percentage distribution are depicted in Figure 1 with no significant correlation between diagnosis and correlation between PaCO₂ and ETCO₂. The parameters ETCO₂ and PaCO₂ and P(a-ET) CO₂ gradient at regular intervals were recorded with mean, SD, median, IQR, minimum and maximum values as depicted in Table 1. The mean P(a-ET) CO₂ gradient at each time interval in both intraoperative and the postoperative period is represented in Table 1, Figures 2 and 3. The mean of

P(a-ET) CO₂ gradient intraoperatively over 4 h is found to be 3.331 ± 2.856 and postoperatively over 24 h 2.779 ± 2.932 and lies in 95% CI. Correlations between PaCO₂ and ETCO₂ at different intervals during intraoperative period and in PACU are depicted in Tables 2 and 3. Data are analyzed by using

Pearson's correlation to study the relationship between PaCO₂ and ETCO₂ at regular intervals. Table 2 shows correlation between PaCO₂ and ETCO₂ intraoperatively with statistically significant Pearson's correlation coefficients. Table 3 shows the correlation between PaCO₂ and ETCO₂ postoperatively in PACU, displaying highly significant correlation at three occasions however not significant at two occasions.

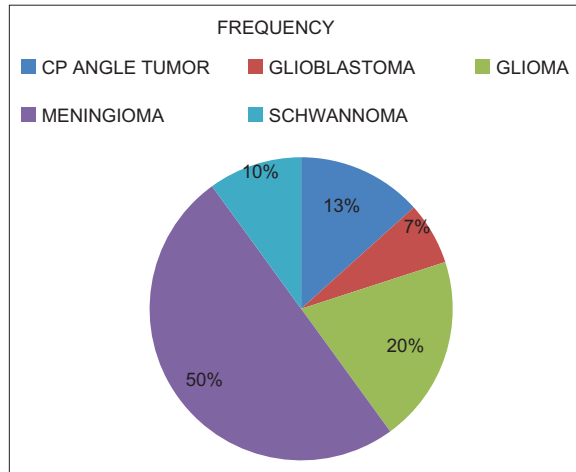


Figure 1: Distribution of study group as per diagnosis

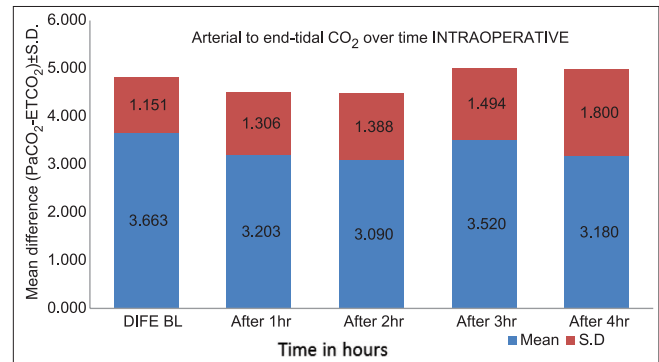


Figure 2: The arterial to end-tidal carbon dioxide differences over time for intraoperative period (mean ± standard deviation)

Table 1: Arterial and end-tidal carbon dioxide values in intraoperative and postoperative period

Parameters (in general)	n	Mean±SD	Median	IQR	Minimum	Maximum
PaCO ₂ baseline/intraoperatively	30	37.130±1.847	37.600	1.775	32.800	39.800
PaCO ₂ after 1 h	30	36.737±1.490	36.800	2.375	34.500	39.900
PaCO ₂ after 2 h	30	36.490±1.608	36.800	1.675	32.100	38.900
PaCO ₂ after 3 h	30	36.620±1.555	36.600	2.900	33.800	39.700
PaCO ₂ after 4 h	30	36.613±1.814	36.800	2.450	32.700	39.900
PaCO ₂ (PACU) baseline	30	37.337±0.989	37.400	1.775	35.400	38.800
PaCO ₂ (PACU) 6 h	30	38.223±1.188	38.200	1.050	36.400	42.400
PaCO ₂ (PACU) 12 h	30	38.517±1.562	38.800	2.350	34.200	41.200
PaCO ₂ (PACU) 18 h	30	38.073±1.847	37.600	2.650	35.300	42.400
PaCO ₂ (PACU) 24 h	30	37.110±1.325	36.600	1.800	35.400	39.900
ETCO ₂ baseline/intraoperatively	30	33.467±1.776	34.000	3.000	29.000	37.000
ETCO ₂ after 1 h	30	33.533±1.074	34.000	1.250	32.000	35.000
ETCO ₂ after 2 h	30	33.400±1.404	33.500	1.250	31.000	36.000
ETCO ₂ after 3 h	30	33.100±1.213	33.000	2.000	31.000	35.000
ETCO ₂ after 4 h	30	33.433±1.960	34.000	2.000	30.000	38.000
ETCO ₂ PACU baseline	30	35.067±1.484	36.000	2.000	32.000	37.000
ETCO ₂ after 6 h	30	35.500±0.938	35.000	1.000	34.000	38.000
ETCO ₂ after 12 h	30	35.400±1.499	36.000	2.250	32.000	38.000
ETCO ₂ after 18 h	30	34.867±1.306	35.000	1.000	33.000	38.000
ETCO ₂ after 24 h	30	34.533±1.137	34.000	2.000	33.000	37.000
Difference baseline	30	3.663±1.151	3.550	1.255	1.500	6.800
After 1 h	30	3.203±1.306	3.650	1.425	-0.400	5.600
After 2 h	30	3.090±1.388	2.800	1.300	0.900	6.600
After 3 h	30	3.520±1.494	3.800	1.950	0.600	5.800
After 4 h	30	3.180±1.800	3.200	1.650	-0.300	8.100
Difference PACU/baseline	30	2.270±1.214	2.200	1.325	0.300	6.200
After 6 h	30	2.723±1.473	2.600	2.050	0.600	7.400
After 12 h	30	3.117±1.490	2.800	2.025	0.800	6.700
After 18 h	30	3.207±1.976	2.600	1.975	0.400	9.400
After 24 h	30	2.577±1.179	2.400	1.775	0.400	5.300

PaCO₂ – Partial pressure of arterial carbon dioxide; ETCO₂ – End-tidal carbon dioxide; PACU – Postanesthesia Care Unit; SD – Standard deviation; IQR – Interquartile range

Table 2: Correlation between partial pressures of arterial and end-tidal carbon dioxide during craniotomy

Parameters (in general)	ETCO ₂ baseline	ETCO ₂ after 1 h	ETCO ₂ after 2 h	ETCO ₂ after 3 h	ETCO ₂ after 4 h
PaCO ₂ baseline					
Pearson's correlation	0.799	0.247	-0.062	0.43	0.235
Significant (two-tailed)	0.000**	0.188	0.745	0.018	0.212
<i>n</i>	Significant	30	30	30	30
PaCO ₂ after 1 h					
Pearson's correlation	0.217	0.522	0.006	-0.166	-0.239
Significant (two-tailed)	0.248	0.003**	0.975	0.38	0.203
<i>n</i>	30	Significant	30	30	30
PaCO ₂ after 2 h					
Pearson's correlation	-0.146	-0.015	0.582	0.015	0.031
Significant (two-tailed)	0.442	0.938	0.001**	0.939	0.871
<i>n</i>	30	30	Significant	30	30
PaCO ₂ after 3 h					
Pearson's correlation	0.238	0.018	0.298	0.439	0.355
Significant (two-tailed)	0.206	0.924	0.11	0.015*	0.054
<i>n</i>	30	30	30	Significant	30
PaCO ₂ after 4 h					
Pearson's correlation	-0.079	-0.299	0.29	0.269	0.547
Significant (two-tailed)	0.678	0.108	0.12	0.151	0.002**
<i>n</i>	30	30	30	30	Significant

*Correlation is significant at the 0.05 level (two-tailed); **Correlation is significant at the 0.01 level (two-tailed). PaCO₂ – Partial pressure of arterial carbon dioxide; ETCO₂ – End-tidal carbon dioxide

Table 3: Correlation between partial pressures of arterial and end-tidal carbon dioxide in Postanesthesia Care Unit

Parameters (in general)	ETCO ₂				
	PACU BL	After 6 h	After 12 h	After 18 h	After 24 h
PaCO ₂ (PACU) baseline					
Pearson's correlation	0.581	-0.024	-0.199	0.236	0.050
Significant (two-tailed)	0.001**	0.899	0.293	0.209	0.795
<i>n</i>	Significant	30	30	30	30
PaCO ₂ (PACU) 6 h					
Pearson's correlation	0.226	0.054	0.144	0.122	0.057
Significant (two-tailed)	0.230	0.776	0.449	0.521	0.765
<i>n</i>	30	Not significant	30	30	30
PaCO ₂ (PACU) 12 h					
Pearson's correlation	0.046	0.279	0.527	0.343	0.395
Significant (two-tailed)	0.811	0.135	0.003**	0.064	0.031
<i>n</i>	30	30	Significant	30	30
PaCO ₂ (PACU) 18 h					
Pearson's correlation	0.287	0.265	0.152	0.251	0.390
Significant (two-tailed)	0.123	0.157	0.422	0.180	0.033
<i>n</i>	30	30	30	Not significant	30
PaCO ₂ (PACU) 24 h					
Pearson's correlation	0.368	0.312	0.255	0.300	0.550
Significant (two-tailed)	0.045	0.093	0.174	0.108	0.002**
<i>n</i>	30	30	30	30	Significant

**Correlation is significant at the 0.01 level (two-tailed). PaCO₂ – Partial pressure of arterial carbon dioxide; ETCO₂ – End-tidal carbon dioxide; PACU – Postanesthesia Care Unit

Figures 4–13 show correlation between two methods of CO₂ measurement at given point of time by plotting a scatter diagram with *R* as the correlation coefficient between each set of values.

Discussion

ETCO₂ monitoring is considered the standard of care during general anesthesia and ICU care. The monitoring of PaCO₂ and

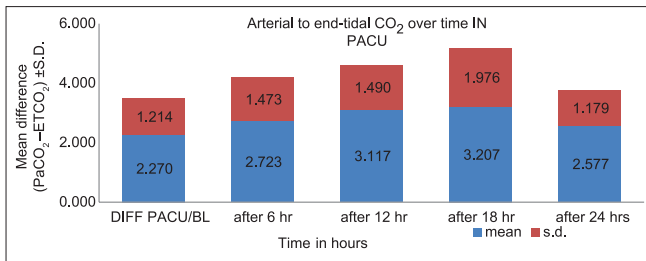


Figure 3: The arterial to end-tidal carbon dioxide differences over time for postoperative period (mean ± standard deviation)

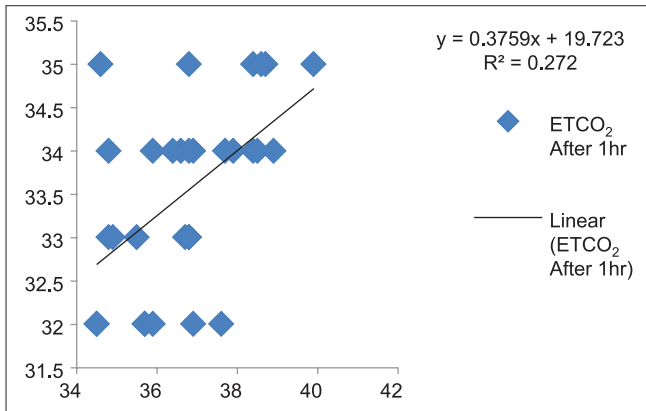


Figure 5: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 1 h ($P < 0.05$)

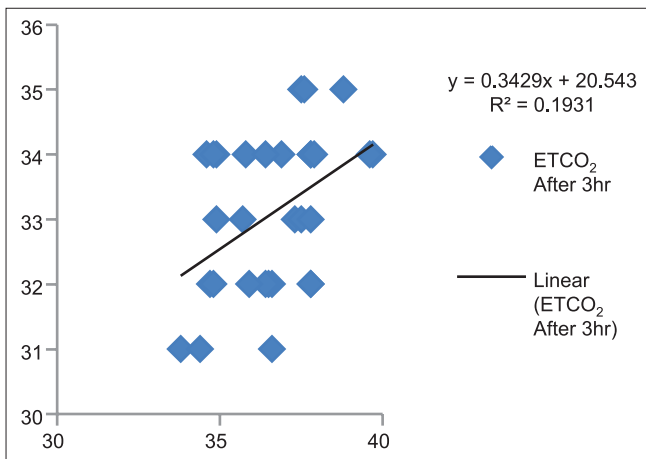


Figure 7: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 3 h ($P < 0.05$)

control in a narrow range is necessary during neurosurgical procedures as this affects and ICP dynamics and CPP.

ABG measurement of PaCO₂ is considered gold standard for monitoring changes in CO₂, which is invasive, expensive, and provides only intermittent measures of PaCO₂. ETCO₂ which is continuous respiratory measure of CO₂ can also reflect an indirect quantity of PaCO₂.^[1] The ETCO₂ may be used as a surrogate marker for monitoring PaCO₂ in neurosurgical and ICU patients and thus reducing repetitive invasive ABG sampling. However, various studies have shown inconsistent results regarding this

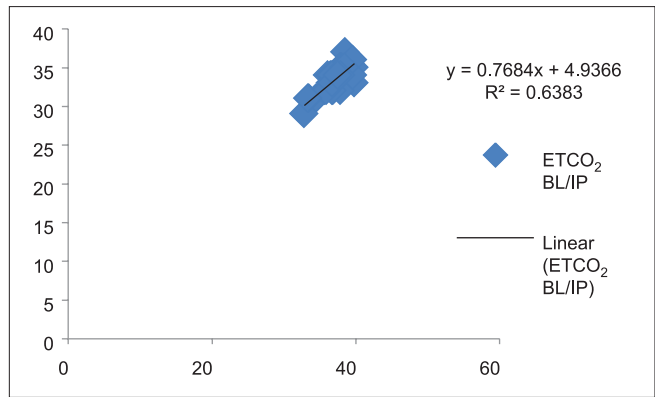


Figure 4: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide ($P < 0.05$)

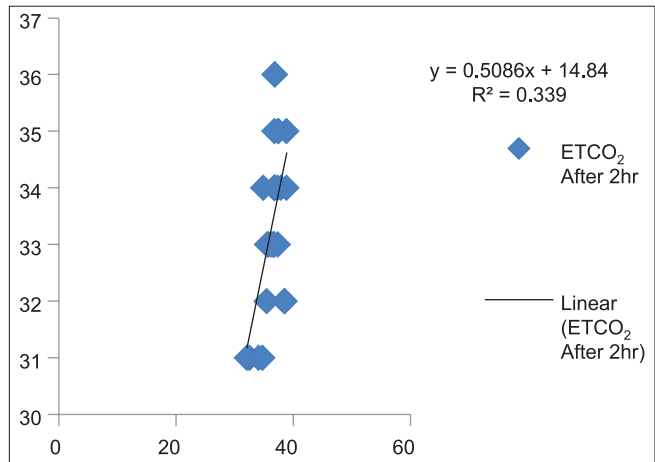


Figure 6: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 2 h ($P < 0.05$)

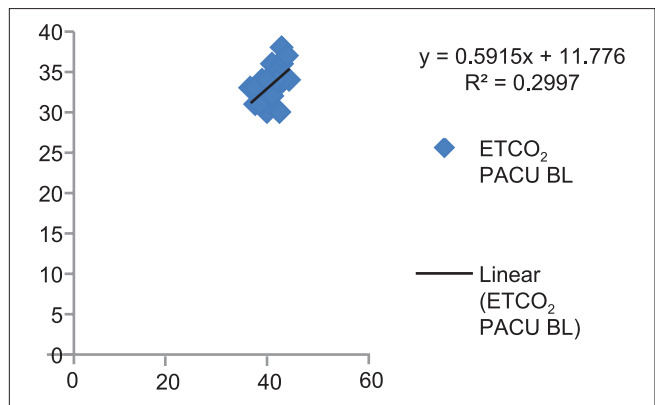


Figure 8: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 4 h ($P < 0.05$)

correlation during intraoperative period and in ICU patients. Hence, we conducted this study to evaluate the correlations in patients undergoing neurosurgeries who are also requiring postoperative ventilatory support for at least 24 h. Hence, this is the first initiative to assess the correlation of CO₂ level through invasive and noninvasive methods in the intraoperative period as well as in the postoperative period in the same set of patients.

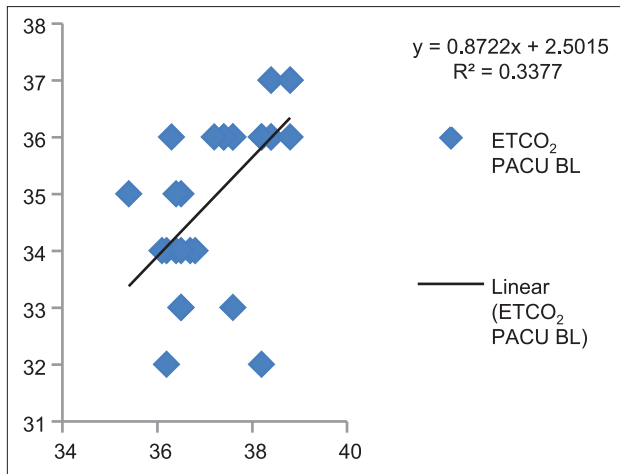


Figure 9: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide baseline in Postanesthesia Care Unit ($P < 0.05$)

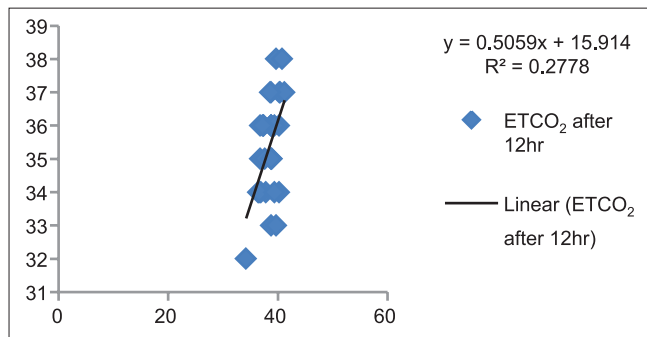


Figure 11: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 12 h in Postanesthesia Care Unit ($P < 0.05$)

Thirty patients aged between 18 and 60 years, undergoing elective craniotomy and those who required postoperative mechanical ventilation for minimum 24 h period, were studied. We found no significant correlation between PaCO₂ and ETCO₂ with respect to demographic data, ASA grading, and diagnosis. Kenichi Satoh 2015 concluded that partial pressure gradient of PaCO₂ to ETCO₂ increases with increasing age in patients undergoing surgeries in general anesthesia in supine position however we did not observe similar finding.^[8]

During intraoperative period mean difference between PaCO₂ and ETCO₂ was found to be 3.31 ± 2.856 with 95% CI [Table 4 and Figure 2]. In our study, PaCO₂ values always exceeded ETCO₂ and at any point of time it never went in opposite directions. P(a-ET) CO₂ gradient lies between 3.6 and 4.6 mmHg in healthy awake patients. This gradient mainly depends on the degree of alveolar dead space.^[12] Under stable physiologic conditions, with completely accurate monitoring, P(a-ET) CO₂ gradient should be close to zero, thus PaCO₂ values can be implicated accurately and constantly from ETCO₂ values. The various causes of widened gradient are V/Q mismatch and poor sampling of gas at patients end, impaired cardiac

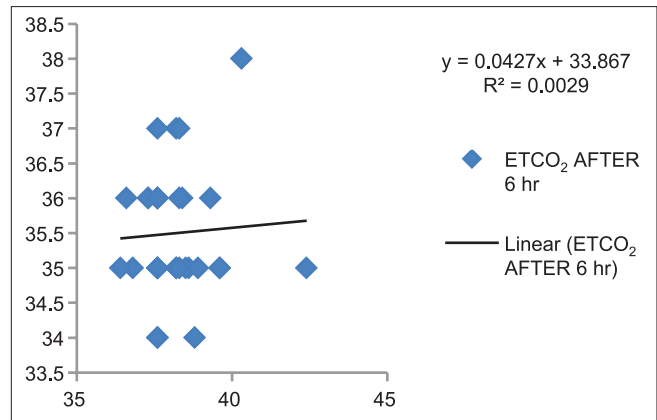


Figure 10: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 6 h in Postanesthesia Care Unit ($P > 0.05$)

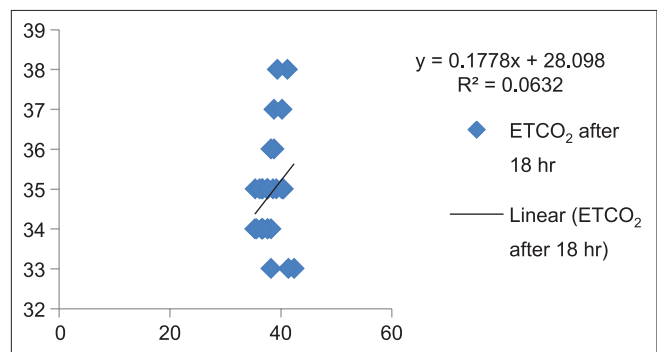


Figure 12: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 18 h in Postanesthesia Care Unit ($P > 0.05$)

Table 4: Mean of gradient of partial pressures of arterial carbon dioxide and end-tidal carbon dioxide during intraoperative period

P (a-ET) CO ₂	Mean±SD
Difference baseline	3.663±1.151
After 1 h	3.203±1.306
After 2 h	3.090±1.388
After 3 h	3.520±1.494
After 4 h	3.180±1.800
Total mean	3.331±1.428

SD – Standard deviation; P (a-ET) CO₂ – Partial pressure of arterial carbon dioxide and end-tidal carbon dioxide

function, and critical illness.^[3] Khan *et al.* 2007, Sharma *et al.* 1995, Hemmati *et al.* 2012 showed similar mean values which remained satisfactorily persistent with ETCO₂ for predicting PaCO₂ under anesthesia.^[3,6,11] However, above positive gradient was not consistently found in all the patients in the study of Russell and Graybeal 1995.^[4]

During the postoperative period, the mean of P(a-ET) CO₂ gradient was found to be 2.78 ± 2.932 with 95% CI [Table 5 and Figure 3]. In these values also, there was a

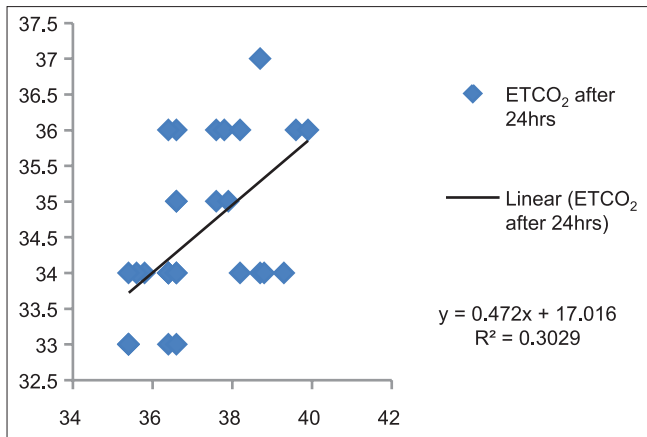


Figure 13: Correlation between partial pressure of arterial carbon dioxide and end-tidal carbon dioxide after 24 h in Postanesthesia Care Unit ($P < 0.05$)

positive gradient in all values with PaCO₂ exceeding ETCO₂ values. According to Razi *et al.* 2012, in healthy subjects there are close correlation between PaCO₂ and ETCO₂, and it is commonly accepted that PaCO₂ measurements vary approximately 2–5 mmHg above ETCO₂ values.^[7] Russell and Graybeal *et al.* 1992 found a significant correlation between the gradients in total study population but not in individual patients. The direction of PaCO₂ change was also inaccurately predicted by ETCO₂ changes. ETCO₂ does not provide a stable reflection of PaCO₂ in all neuro-intensive care patients.^[5]

We also calculated the correlation between PaCO₂ and ETCO₂ at given time and found out the positive correlation with each value, which was statistically significant ($P < 0.05$). During intraoperative period monitoring of each hourly value of PaCO₂ and ETCO₂, a positive significant correlation was found [Table 2]. These results are consistent with Khan *et al.* 2007, Husaini and Choy 2008, Hemmati *et al.* 2012, which showed a positive correlation at each time interval,^[1,3,11] whereas Russell and Graybeal 1995 did not show positive correlation in all patients undergoing craniotomy.^[4]

In the same patients in the postoperative period in ICU on volume SIMV mode for 24 h, we obtained a highly significant positive correlation ($P < 0.01$) at three occasions, however not significant at two [Table 3]. Razi *et al.* 2012 assessed in neurological patients admitted in intensive care in various modes of ventilation at a given point of time. They have found a positive correlation in each mode of ventilation (volume SIMV, continuous positive airway pressure, T-piece).^[7] Kerr *et al.* 1996 studied the relationship between PaCO₂ and ETCO₂ in mechanically ventilated adults with severe head trauma and also observed ETCO₂ monitoring correlating well with PaCO₂ in patients without respiratory complications or without spontaneous breathing.^[10] Russell and Graybeal 1992 did not obtain positive correlation in all ICU patients.^[5] The reasons for this variability in the above gradient in our study and other

Table 5: Mean of gradient of partial pressure of arterial carbon dioxide and end-tidal carbon dioxide in Postanesthesia Care Unit

P (a-ET) CO ₂	Mean±SD
Difference PACU/baseline	2.270±1.214
After 6 h	2.723±1.473
After 12 h	3.117±1.490
After 18 h	3.207±1.976
After 24 h	2.577±1.179
Total mean	2.779±1.466

PACU – Postanesthesia Care Unit; P (a-ET) CO₂ – Partial pressure of arterial carbon dioxide and end-tidal carbon dioxide; SD – Standard deviation

studies as well are explained by various factors such as dead space fraction, ventilation-perfusion mismatch, the site of sampling, and nonuniform alveoli CO₂ emptying patterns.^[7,9] Cheifetz and Myers *et al.* 2007 have emphasized capnography as the standard of care in all respects right from operation theater to ICUs during mechanical ventilation.^[9]

The present study was not without limitations as we did not study patients with major hemodynamic changes, severe lung disease, or positions other than supine. In healthy lungs and hemodynamically stable patients due to good alveolar ventilation and perfusion matching, ETCO₂ closely correlates with PaCO₂ but in above set of patients it may not correlate well, hence it is practical to verify with ABG analysis. Hence, future research should be directed in including all these patients. In addition, we used sidestream capnography intraoperatively as well as postoperatively, as mainstream was unavailable. In the PACU, we did not compare different modes of ventilation with respect to ETCO₂ and PaCO₂ correlation. Hence, above factors must be considered when generalizing the results.

Conclusions

From this study, we conclude that ETCO₂ reflects PaCO₂ with acceptable accuracy. In addition, ETCO₂ correlates PaCO₂ in patients undergoing neurosurgery in the intraoperative as well as in the postoperative period on mechanical ventilation (SIMV mode) in PACU. The above correlation is perfect in patients who are hemodynamically stable and with healthy lungs. Thus, simple, continuous, and noninvasive ETCO₂ can be used as a reliable guide to estimate PaCO₂ during neurosurgical procedures and in PACU.

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Conflicts of interest

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