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Arterial to end-tidal carbon dioxide difference in neurosurgical patients undergoing craniotomy: a review of practice

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Original Article

Arterial to End-Tidal Carbon Dioxide Difference in Neurosurgical Patients undergoing Craniotomy: A Review of Practice

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Abstract

Objective: To see if $PETCO_2$ reflects $PaCO_2$ with acceptable accuracy.

Methods: In this audit the anaesthetic chart of fifty consecutive patients, age 12 years and above undergoing craniotomy for intracranial pathology, were reviewed.

Results: The difference between end tidal carbon dioxide ($ETCO_2$) value corresponding to the time of taking the arterial sample and the $PaCO_2$ was calculated. The mean end tidal CO_2 was 29.3 ± 2.8 and the mean $PaCO_2$ was 32.63 ± 4.5. The mean difference between the two values was calculated as 4.09 ± 3.0. The regression coefficient was 0.496, which showed a moderate association. A wide variability was observed in the results.

Conclusion: Based on our results we recommend that arterial samples should be taken to determine PaCO2 in neurosurgical patients where maintenance of cerebral blood flow is crucial e.g. cerebral aneurysm surgery (JPMA 57:446:2007).

Introduction

Both $PaCO_2$ and $PETCO_2$ are indicators of ventilatory adequacy. It is important to know the $PaCO_2$ in neuroanaesthesia because of its effect on cerebral blood flow. $PETCO_2$ has been used as a non-invasive estimate of $PaCO_2$.[1] The usual reported difference between $PaCO_2$ and $PETCO_2$ in healthy awake patients is 3.6 to 4.6 mm of Hg[2] but a significant variability has been observed in mechanically ventilated neurosurgical ICU patients[3] and in patients undergoing craniotomy in different positions.[4] There has also been some controversy in recent anaesthetic literature whether end tidal CO_2 ($PETCO_2$) is an accurate reflection of $PaCO_2$.[3,5]

This audit was undertaken to review our routine practice of obtaining $PaCO_2$ during craniotomy procedures and comparing it with the $PETCO_2$ at the same time. The objective was to see whether $PETCO_2$ reflected the $PaCO_2$ with acceptable accuracy.

Methods

Since the last three years our routine practice in neuroanaesthesia is to take an arterial blood sample from the arterial line one hour after start of surgery in patients undergoing craniotomy and to note the end tidal carbon dioxide concentration simultaneously. The readings for $ETCO_2$ are routinely noted on the patient perioperative charts.

In craniotomy patients the $ETCO_2$ value corresponding to the time of taking the arterial sample is specifically noted on our anaesthetic chart. A file note is also made on ventilatory and haemodynamic parameters at the time of obtaining the sample. The $PETCO_2$ is taken as the maximal terminal value at end expiration. The charts of the last 50 consecutive patient's age 12 years and above, undergoing craniotomy were reviewed to look for the difference between the two values and to see whether the $PETCO_2$ value could be substituted for $PaCO_2$. The difference between the two value P (a-ET) CO_2 was calculated manually for each sample. The other values routinely noted at the time of sampling were heart rate, systolic and mean arterial pressure (SAP, MAP), respiratory rate, tidal volume and inspired oxygen. These patients were undergoing craniotomy for different pathologies.

The institutional ethical committee was consulted and no permission was required for this review of practice.

All these patients had undergone craniotomy in supine position and had no significant lung disease. The patient's lungs were ventilated on Aestiva/5 machine ventilator. Volume controlled ventilation was used and the initial ventilatory setting was to achieve a $PETCO_2$ of 30-32mm of Hg. Carbon dioxide sampling was done on a side stream sample with the Datex AS/3 monitors. Arterial blood gas samples were taken from the radial artery catheter, which is standard monitoring in a craniotomy at our institution. The sample was sent to the laboratory immediately and was analyzed on NOVA stat profile Ultra (Nova Biomedical International, USA). These results were not corrected for patient's temperature.

Values were entered on the SPSS 13.0 statistical program. Data is presented as mean SD and range of values. Confidence intervals for mean difference between $ETCO_2$ and $PaCO_2$ was also calculated. Regression analysis between $PaCO_2$ and $PETCO_2$ and its standard error was also calculated.

Results

All patients underwent craniotomy in supine position. The mean age of the patients was 36.05 $\pm$ 18.0 years. The median age was 33.5 years. Age range was 12-71 years. 95 CI were 30.2 to 41.8. The mean weight was 62.6 $\pm$ 17.0 kgs.

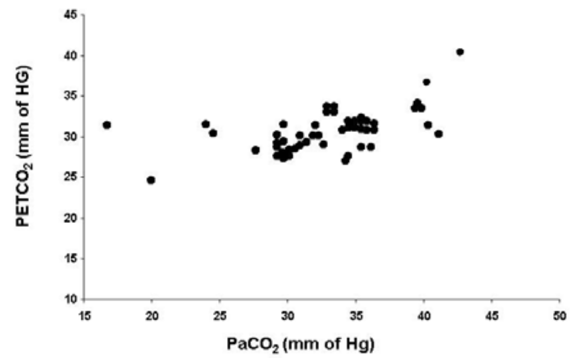

Figure 1. Regression analysis comparing PaCo2 to End-Tidal PCO2 (PETCO2) n = 50
Each ? represents PETCO2 and PaCO2 difference in individual patients.

There were 30 males and 20 females. The mean ETCO2 was 29.30 $\pm$ 2.8 (range 23-40 mm of Hg). The mean PaCO2 was 32.63 $\pm$ 4.5 mm of Hg (range 16.8 - 42.5). The mean difference between ETCO2 and PaCO2 was 4.09 $\pm$ 3.08 with 95% CI = 3.21-4.97 (range 2.7 to 15.2). The regression coefficient was calculated as: 0.496, SE = 3.96. The regression analysis between PaCO2 and ETCO2 is shown in Figure 1.

The values differed by less than 6 mm of Hg in 41 patients (82%). The difference was more than 6 mm of Hg in nine (18%) patients. In two patients (4%) a negative correlation was observed.

Discussion

Capnometry is the measurement of the concentration or partial pressure of carbon dioxide at the patient's airway during the entire ventilatory cycle. It provides a numerical measurement of inspired and end tidal CO_2.[6] The end-tidal CO_2 or $PETCO_2$ closely approximates the arterial PCO_2 in normal lungs. The two common causes of more than normal difference are ventilation/perfusion mismatch and poor sampling of gas at patients end. End tidal CO_2 measurement is currently the standard of care where general anaesthesia is administered.

The average P (a-ET) CO_2 gradient was determined as 4-6 mm of Hg in patients with normal lungs by Nunn et al.[7] Other investigators have come up with similar figures: Takki et al, 3-5 mm of Hg[8], Weingner 4$\pm$2 mm of Hg[9], and Ashrog, 5 $\pm$2 mm of Hg.[10] No correlation was found with age and systolic arterial pressure. In respiratory failure patients, the average P (a-ET) CO_2 gradient was found to be 18 mm of Hg.[11]

Several other factors have been shown to affect this relationship e.g. positioning[12], site of measurement i.e.

main stream versus side stream measurement[13], and pregnancy.[14] The relationship appears to be unaffected by temperature or oxygenation.[15]

The measurement of arterial $PaCO_2$ is of special importance in neuroanaesthesia because of its effect on cerebral circulation.[15] In order to have a noninvasive measure of $PaCO_2$ several authors have investigated this relationship between $PaCO_2$ and end tidal CO_2 in neuroanaesthesia with mixed results. Sharma[16] found this mean difference to be 5 $\pm$ 2mm of Hg, but with large individual variations between -14 to 10 $\pm$ 19 mm of Hg. Kerr looked at head injured patients and found a mean difference of 6 $\pm$ 6 mm of Hg.[17] Russell and Greybell[3] found the mean difference to be 7 $\pm$ 3 mm of Hg in neurosurgical patients in the operating room and 7 $\pm$ 4 mm of Hg in ICU. They found a correlation coefficient of 0.63. Isert[4] found this difference to be 4 $\pm$ 4 mm of Hg and observed that end tidal CO_2 correlated with $PaCO_2$ in 82% cases. Excellent correlation was shown by Mackersie et al who looked at data of 36 patients with known or suspected severe head injury.[5] Grenier et al[12] showed a poor correlation and recommended arterial blood gas in addition to capnography. Ferber et al[18] also showed poor correlation in patients undergoing craniotomy for head injury.

In our data the mean difference was 4.09 ($\pm$ 3.08) CI. These results are very similar to those shown by Isert et al.[4] We also found a wide variability with the lowest value as -2.7 and the highest as +15.2. We found an r value (regression coefficient of 0.496). This only shows a moderate association and not a strong one.[19]

Eighty two percent of our values differed by less than 6 mm of Hg. Negative correlation was observed in only 2 patients (4%). Negative values have also been reported by other investigators in neuroanaesthesia[3,4], and slow emptying of alveoli with long time constant has been hypothesized as the mechanism[12] but this concept has been challenged. A higher incidence of negative values have been reported in children, in laparoscopic surgery or after cardiac surgery,[2,20,21] and in pregnancy.[14] Negative correlation was seen in 20% of patients undergoing sub-arachoid haemorrhage surgery.[18] Both our patients in whom negative values were reported had a history of hypertension but did not have any respiratory disease. The haemodynamic parameters were stable at the time of sampling.

In summary we observed that a relationship between $PaCO_2$ and end tidal CO_2 was predictable in only 49% of patients. A wide variability was seen in the results. Hence based on this audit we would recommend that arterial blood samples should be taken to determine $PaCO_2$ in neurosurgical patients undergoing craniotomy where maintenance of cerebral blood flow is crucial e.g. during cerebral aneurysm surgery or patients with large intracranial tumours. In neurosurgical patients undergoing surgery for small superficial tumours with minimal disruption of cerebral blood flow and intracranial pressure, the cost benefit analysis may not favour arterial sampling.

References

1. Benallal H, Busso T. Analysis of end-tidal and arterial PCO2 gradients using a breathing model. Eur J Appl Physiol 2000; 83:402-8.
2. Nunn JF: In Nunn JF Applied Respiratory Physiology. 3rd edition London: Butterworths, 1987, pp 207-34.
3. Russell GB, Graybeal JM. End-tidal carbon dioxide as an indicator of arterial carbon dioxide in neurointensive care patients. J Neurosurg Anesth 1992; 4:245-9.
4. Isert PR. Arterial to end-tidal CO2 difference during neurosurgical procedures. Can J Anesth 1996; 43:196-7.
5. Mackersie RC, Karagianes TG. Use of end-tidal carbon dioxide tension for monitoring induced hypocapnia in head-injured patients. Critical Care Medicine 1990; 18: 764-5.
6. Swedlow DB, Irwing SM. Monitoring and patient's safety. Monitoring in Anesthesia and Critical Care Medicine. Churchill Livingstone, Second Edition Ed Casey D Blitt, New York 1990; pp 33-63.
7. Nunn JF, Hill DW. Respiratory dead space and arterial to end-tidal CO2 tension difference in anesthetized man. J Appl Physiol 1960; 15:383-9.
8. Takki S, Aromaa U, Kauste A. The validity and usefulness of the end-tidal Pco2 during anaesthesia. Ann Clin Res 1972; 4:278-84.
9. Weinger MB, Brumin JE. End-tidal carbon dioxide as a measure of arterial carbon dioxide during intermittent mandatory ventilation. J Clin Monit 1987; 3:73-9.
10. Askrog V. Changes in (a-A) Co2 difference and pulmonary artery pressure in anaesthetized man. J App Physiol 1966; 21:1299-305.
11. Yamanaka MK, Sue Dy. Comparison of arterial-end-tidal PCO2 difference and dead space/tidal volume ratio in respiratory failure. Chest 1987; 92:832-5.
12. Grenier B, Verchere E, Mesli Abdelghani, Dubrevil M, Su OD, Vandendriessche M et al. Capnography monitoring during neurosurgery. Reliability in relation to various intraoperative positions. Anesth Analg 1999; 88:43-8.
13. Chan KL, Chan MT, Gin T. Main stream vs. sidestream capnometry tension during supine craniotomy. Anaesthesia 2003; 58:149-55.
14. Shanker KB, Moseley H, Kumar Y, Vemula V. Arterial to end-tidal carbon dioxide tension difference during Caesarean section anaesthesia. Anaesthesia 1986; 41:698-702.
15. Sakabe T, Nakakimura K. Effects of Anesthetic agents and other drugs on cerebral flow, metabolism and ICP. In: Cottrell JE, Smith DS, eds. Anesthesia and Neurosurgery. 4th ed. St Louis: Mosby, 2001, pp 129-43.
16. Sharma SK, McGuire GP, Cruise CJ. Stability of the arterial to end-tidal carbon dioxide difference during anaesthesia for prolonged neurosurgical procedures. Can J Anaesth 1995; 42:498-503.
17. Kear ME, Zempsky J, Sereika S Orndoff P, Rudy EB. Relationship between arterial carbon dioxide and end-tidal carbon dioxide in mechanically ventilated adults with severe head trauma. Crit Care Med 1996; 24:785-90.
18. Ferber J, Juniewicz HM, Lechowicz-Glogowska EB, Pierick R, Wroriski J. Arterial to end tidal carbon dioxide difference during craniotomy in severely head injured patients. Folia Med Cracov 2001; 42:141-52.
19. Regression and Correlation. In: Statistical methods of Anaesthesia and Intensive Care. Myles PS and Gin T, eds. 1st ed. Butterworth, Heinemann. Oxford 2000; pp 78-93..
20. Rich GF, Sconzo JM. Continuous end-tidal CO2 sampling within the proximal endotracheal tube estimates arterial CO2 tension in infants. Can J Anaesth 1991; 38:201-3.
21. Wahba RWM, Mamazza J. Ventilatory requirements during laparoscopic cholecystectomy. Can J Anaesth 1993; 40:206-10.