

Arterial to end-tidal carbon dioxide tension difference during anaesthesia in early pregnancy

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Sixteen patients requiring general anaesthesia for termination of pregnancy by dilatation and evacuation of the uterus were studied. Arterial and end-tidal carbon dioxide tensions were determined during anaesthesia. The mean arterial to end-tidal carbon dioxide tension difference was 0.07 kPa (-0.02-0.16, 5-95 per cent confidence limits). These results were similar to those observed during Caesarean section and those during anaesthesia for post-delivery tubal ligations. The physiological changes such as increased cardiac output, haemodilution, and increased blood volume which manifest by 12 weeks of gestation probably result in a reduced (a-E')PCO₂ value.

It has been observed that during Caesarean section the arterial to end-tidal carbon dioxide tension difference, (a-E')PCO₂ was decreased and that arterial carbon dioxide tension (PaCO₂) was closer to end-tidal carbon dioxide tension (Pe'CO₂) than in non-pregnant subjects.^{1,2} It has also been observed that, the (a-E')PCO₂ values return to normal non-pregnant values by 8 days following delivery of the baby.³ We were unable to find a study which had evaluated this relationship during general anaesthesia in early pregnancy. The study was planned to evaluate the (a-E')PCO₂ difference during general anaesthesia in early pregnancy.

Methods

The study was approved by the hospital ethics committee and informed consent was obtained from all the patients. The study comprised 16 patients scheduled for termination of pregnancy by dilatation and evacuation of the uterus. All patients were nonsmokers and had no pre-

Key words

ANAESTHESIA: obstetric, general; MEASUREMENT TECHNIQUE: capnography.

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vious respiratory or cardiac abnormalities. None of the patients was obese according to weight and height indices ($W/H^2 < 25$).⁴

All patients were unpremedicated. Anaesthesia was induced with thiopentone 5 mg · kg⁻¹ and tracheal intubation performed following succinylcholine 1.5 mg · kg⁻¹. Anaesthesia was maintained with 50 per cent oxygen in nitrous oxide and one per cent halothane. Succinylcholine infusion (60 µgm · kg⁻¹ · min⁻¹) was used to maintain muscle relaxation. The lungs were ventilated with a Seimens Elema Servo ventilator (Model 900 B). Patients received a minute volume of 80 ml · kg⁻¹ · min⁻¹ at a frequency of 12 breaths · min⁻¹ (tidal volume 6.67 ml · kg⁻¹).

The end-tidal carbon dioxide concentration was measured continuously at the proximal end of the tracheal tube using a Seimens Elema 930 CO₂ analyser. The analyser was calibrated before each case using a known concentration of carbon dioxide. Arterial blood was sampled from the left radial artery 15 minutes after induction of anaesthesia. The sample was analysed immediately for PaCO₂ in a Corning blood gas analyser (Model 165/2) after two-point calibration before each measurement, at 37°C. The nasopharyngeal temperature was monitored by a calibrated thermometer in all patients and the blood gas results were corrected to body temperature using the nomograms of Kelman and Nunn.⁵ The end-tidal carbon dioxide concentrations recorded at the time of arterial sampling, were corrected for the presence of 50 per cent nitrous oxide.⁶ They were also corrected to the body temperature.⁶ Surgery commenced after the arterial sampling to avoid the haemodynamic disturbances due to surgical stimulation and blood loss.

The fraction (F) of end-tidal gas from unperfused alveoli (representing alveolar dead space) was calculated using the equation of Nunn and Hill.⁷

$$F = (\text{PaCO}_2 - \text{Pe}'\text{CO}_2) / \text{PaCO}_2$$

A paired t test was used to evaluate the statistical difference between mean PaCO₂ and mean Pe'CO₂ (the data was normally distributed). Linear regression analy-

TABLE I Mean values with 5–95 per cent confidence limits for age, duration of pregnancy, PaCO₂, PE'CO₂ and (a-E')PCO₂

	Age (years)	Duration of pregnancy (weeks)	PaCO ₂ (kPa)	PE'CO ₂ (kPa)	(a-E')PCO ₂ (kPa)
Mean	25.19	12.44	4.19	4.12	0.07
5–95% confidence limits	22.12–28.26	11.69–13.19	4.02–4.36	3.97–4.27	–0.02–0.16

TABLE II Results of linear regression analysis

Independent variable (x)	Dependent variable (y)	Correlation coefficient (r)	Regression equation	Confidence interval of b (at P = 0.05)
PaCO ₂	PE'CO ₂	0.88	$y = 0.86 + 0.78 x$	0.54–1.02

sis was done between PaCO₂ (independent variable, x) and PE'CO₂ (dependent variable, y) and the variables of the regression equation and confidence limits were determined.

Results

The mean weight \pm SEM of the subjects in the group studied was 59.41 kg \pm 3.10. Table I shows mean age, PaCO₂, PE'CO₂ and (a-E')PCO₂ values together with 5–95 per cent confidence limits. The mean duration of pregnancy was 12.44 weeks (11.69–13.19, 5–95 per cent confidence limits). The difference between the mean PaCO₂ and PE'CO₂ was not statistically significant. There was a positive correlation ($r = 0.88$, $P < 0.001$) between PaCO₂ and PE'CO₂ (Table II). The fraction of tidal gas from unperfused alveoli was 0.02 ± 0.01 . The mean tidal volume used was 379.80 ml \pm 19.57.

Discussion

The mean arterial to end-tidal carbon dioxide tension difference during anaesthesia in the pregnant women studied, was 0.07 kPa (0.02–0.16).^{*} This is lower than the values reported in non-pregnant anaesthetized (artificial ventilation) patients by Nunn *et al.*⁷ 0.63 kPa (0.42–0.84),^{*} Fletcher *et al.*⁸ 0.6 kPa (0.20–1.40)[†], Takki *et al.*⁹ 0.47 kPa (0.37–0.57),^{*} Askrog,¹⁰ 0.71 kPa (0.46–0.96),^{*} Askrog *et al.*¹¹ 0.67 kPa (0.57–0.77),^{*} and Raemer *et al.*¹² 0.54 kPa (0.41–0.67).^{*} They were also lower than the non-pregnant values of 0.70 kPa (0.61–0.79)^{*} observed by us using similar methodology.¹ There was no statistically significant difference between PaCO₂ and PE'CO₂ and hence the latter might

reflect PaCO₂. It was observed previously by us that (a-E')PCO₂ value was 0.00 kPa (0.17–0.17)^{*} during Caesarean section, and 0.08 kPa (0.02–0.18)^{*} during post-delivery tubal ligations. The patient population and methodology used in these studies were similar to those of the present study. The (a-E')PCO₂ values observed in the present study did not differ significantly from the (a-E')PCO₂ values observed during Caesarean section,¹ and during anaesthesia for post-delivery tubal ligations³ (Student's *t* test).

Our results show that (a-E')PCO₂ is small and PE'CO₂ approaches PaCO₂ at the end of first trimester. This is probably due to the many physiological changes associated with pregnancy which are significant by 12 weeks of gestation.^{13–16} There is a 25–30 per cent increase in the cardiac output at 12 weeks of pregnancy over the non-pregnant levels.^{13,14} It has been shown that cardiac output rises to a plateau of about 1.5 L \cdot min⁻¹ above the mean post-partum level well before the end of the first trimester.¹⁵ An increase in blood volume is seen as early as four to six weeks after conception and rises to 15–20 per cent over the non-pregnant level at 12 weeks of pregnancy.^{13,14,16} The plasma volume expands steadily from the first month of pregnancy.¹⁶ Following an initial drop, the red cell volume also increases progressively during pregnancy. However, the plasma volume expands proportionately more than the red cell volume resulting in haemodilution.¹⁶ The increased cardiac output, haemodilution and increased blood volume probably result in better perfusion of the alveoli and improved gaseous exchange. This would account for low (a-E')PCO₂ values observed in our study. The fraction of the end-tidal gas from the unperfused spaces (representing alveolar dead space) was calculated as 0.02 ± 0.01 in this study. This is similar to the 0.01 ± 0.02 observed during

*5–95 per cent confidence limits with mean.

†Median with 5th and 95th percentiles.

Caesarean section and the 0.02 ± 0.01 observed during anaesthesia for post-delivery tubal ligation.^{1,3} However, this fraction was significantly less (Student's *t* test, $P < 0.001$) when compared to the 0.19 ± 0.01 observed in non-pregnant patients under anaesthesia indicating improved alveolar perfusion in early pregnancy.¹⁷

The (a-E')PCO₂ values in 37 per cent of our patients were negative. Negative or zero (a-E')PCO₂ values were observed in 12 per cent of non-pregnant patients under anaesthesia/IPPV with large tidal volumes and low frequencies.⁸ Increases in tidal volume and decrease in frequency result in the following. (i) Gas emptying from slow alveoli can reach the mouth, whereas it would have remained in the airways with small tidal volumes. (ii) Relatively better ventilation of dependent well perfused alveoli. Under these circumstances the low \dot{V}/\dot{Q} compartments (alveoli with higher CO₂ concentration) make a more substantial contribution to gas exchange. Normally, there are differences in the emptying patterns between various compartments in the lung. Therefore, more gas with an above average alveolar PCO₂, can now reach the airway opening from "late" or "slow" compartments. This results in end-tidal CO₂ exceeding the mean arterial PCO₂.¹⁸⁻²⁰ The increased cardiac output associated with pregnancy increases the number of alveoli with low \dot{V}/\dot{Q} ratio (alveoli with high CO₂ concentration).¹⁷ This therefore increases the likelihood of the occurrence of negative (a-E')PCO₂ values in the pregnant women. As pregnancy advances, reduced functional residual capacity and increased CO₂ production result in further increase in the alveolar PCO₂. This leads to an increase in the incidence of negative (a-E')PCO₂ values (50 per cent) as were observed during Caesarean section anaesthesia.¹

Acknowledgements

We gratefully acknowledge Prof. E. R. Walrond, FRCS, Professor of surgery and Dean, faculty of Medical Sciences, University of West Indies, Barbados, for his advice and review of the manuscript, Dr. P Seshagiri Rao, PhD for his assistance in statistical evaluation of the results. We wish to thank Mr. Sam McCaskie, Mr. R. King and Mr. Gibson, technical assistants for assisting in calibrating the equipment and blood gas analyser.

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Résumé

Seize patientes requérant une anesthésie générale pour avortement par dilatation et évacuation de l'utérus ont été étudiées. Lors de l'anesthésie, la différence entre la PCO₂ artérielle et la PCO₂ en fin d'expiration fut déterminée. La différence moyenne de la PCO₂ artérielle et de la PCO₂ en fin d'expiration était de 0.07 kPa (-0.02-0.16, 5-95 pour cent de limite de confiance). Ces résultats sont similaires à ceux observés lors d'une césarienne et lors d'une anesthésie après accouchement pour ligature tubaire. Les altérations physiologiques comme l'augmentation du débit cardiaque, l'hémodilution et l'augmentation du volume sanguin qui se manifestent 12 semaines après la gestation, amènent probablement une réduction des valeurs de (a-E') PCO₂.