A STUDY OF CARBON DIOXIDE TENSIONS DURING HALOTHANE ANAESTHESIA USING THE MAGILL CIRCUIT

BY
S. D. IVANOV, F. F. WADDY AND A. M. C. JENNINGS
Northampton General Hospital, England

SUMMARY
In thirteen patients capillary blood samples were obtained for measurement of pH and \( P_{CO_2} \) immediately after the induction dose of thiopentone, after 45 minutes of 2 per cent halothane breathed spontaneously (with nitrous oxide and oxygen), and 30 minutes postoperatively. A very variable rise in \( P_{CO_2} \) was observed which did not correlate well with changes in pulse rate, blood pressure, respiratory frequency, respiratory minute volume, or with the age of the patient. A single measurement of respiratory minute volume with a Wright respirometer was of no value in indicating which patients had the greatest carbon dioxide retention. The return of \( P_{CO_2} \) to normal values in the postoperative period was found to be rapid and, for practical purposes, complete in 30 minutes.

It is often suggested that when patients are breathing halothane spontaneously respiration should be assisted (at least occasionally) in order to combat the tendency to hypercarbia (Stephen, 1963; Lee and Atkinson, 1964; Graber and Markello, 1965; Wylie and Churchill-Davidson, 1966). Despite this advice halothane is commonly administered in concentrations up to 2 per cent with unassisted ventilation for periods of an hour or more, for procedures not needing deep relaxation or intubation. Probably this is due in part to the reluctance of anaesthetists to complicate a technique which owes much of its popularity to its ease and simplicity, and which gives satisfactory results without apparent signs of clinical deterioration. Many would feel that intubation was called for in nearly every case if assistance of respiration were routine during halothane anaesthesia.

The acidosis during halothane anaesthesia with unassisted ventilation has been shown to be wholly respiratory for practical purposes (Black and McKane, 1965; Marshall, 1966). The rise in \( P_{CO_2} \) found has varied considerably, being sometimes trivial, sometimes not, and it would obviously be useful if the anaesthetist could have some sort of guide as to whether the individual patient under his care was developing a significant degree of carbon dioxide retention.

The signs of carbon dioxide retention, as usually described, are modified by halothane. Stephen (1963) has denied their usefulness, saying that during halothane anaesthesia "the signs of increased carbon dioxide retention will not be apparent in the patient" and goes on to say "the importance of using a ventilation meter during halothane anesthesia is obvious; this instrument provides an objective method of determining tidal volume and hence alveolar ventilation."

Black and McKane (1965) have shown quite clearly the association between a rise in \( P_{CO_2} \) and depression of the minute volume in a sample of nine cases. However, this association, doubtless of cause and effect, will only be of use in deciding in clinical circumstances whether any one individual patient needs respiratory assistance if there is a reasonably reliable correlation between the rise in \( P_{CO_2} \) and the depression of minute volume in the same patient. If the data of Black and McKane are examined from the point of view of the individual patients, rather than as a group, it is not possible to find any such relationship. Nor do Marshall's more recent data (1966) show any. Figure 1 shows the relationship between rise in \( P_{CO_2} \) in mm Hg and the fall in minute volume, expressed as a percentage of the initial value (each symbol representing one patient), the data being derived from the two papers. No relationship is
apparent, and indeed calculation of the correlation coefficient gives negative values for the halothane data of Black and McKane (—0.43), their methoxyflurane data (—0.19) and their halothane and Marshall's halothane data combined (—0.14) suggesting that the more a patient's minute volume is depressed the less the rise in $P_{\text{CO}_2}$. This would be embarrassing if it were statistically significant but fortunately it is not (Documenta Geigy, p. 61).

The present study was prompted by a desire to see whether there is any measurement easily accessible to the anaesthetist which would enable him to determine whether a particular patient was suffering a significant rise in $P_{\text{CO}_2}$ during anaesthesia by this method. It was considered that a small series would be sufficient because if nothing useful can be found in a dozen or so cases, then anything which might be statistically significant in a large number of cases is not likely to be of any account clinically since it would not help in the management of a particular case.

**METHOD**

Three measurements of pH and $P_{\text{CO}_2}$ were made on capillary blood samples, using the interpolation method of Siggaard-Andersen and associates (1960), taken from each of thirteen adult patients. The measurements were made immediately after induction, after 2 per cent halothane had been inhaled for 45 minutes, and 30 minutes after the cessation of anaesthesia. It was not thought justifiable to draw samples of arterial blood in these patients in view of the small discrepancy found by Docrat and Kenny (1965) between arterial and capillary samples. All the measurements were made by one of us (S.D.I.). In some patients the measurements were made at the termination of surgery and when this was the case the samples were stored in a refrigerator for up to 1 hour; otherwise they were examined immediately.

The patients, whose ages ranged from 29 to 67 years, were free from known respiratory disease and were undergoing operations for which nitrous oxide, oxygen and halothane was a suitable anaesthetic. They were selected at random from the routine operating lists. Premedication consisted of papaveretum or pethidine with atropine. Blood pressures were taken by auscultation using a mercury manometer. The cuff and stethoscope end were strapped in position in the anaesthetic room and not disturbed until the observations were complete. Blood pressure, pulse and respiration rates were measured in the anaesthetic room before induction. As soon as all was ready for capillary sampling, anaesthesia was induced by means of a sleep dose of thiopentone and a capillary sample was taken immediately. Eight patients were then given suxamethonium to facilitate intubation. The patient was then settled with whatever concentration of halothane the anaesthetist thought best. Between 2 and 10 minutes later, when anaesthesia was stabilized, the Fluotec vaporizer was set to deliver 2 per cent halothane through a Magill attachment; flow rates of oxygen 3 l./min and nitrous oxide 7 l./min were used. In each case the patient's condition remained clinically satisfactory, and 45 minutes from the time the Fluotec was set at 2 per cent the second capillary sample was taken. In the 3 minutes preceding this the blood pressure, pulse rate, respiration rate and expired minute volume were measured, the latter with a Wright respirometer. Anaesthesia was then continued according to routine and the time of cessation of anaesthesia noted. The third capillary sample was taken 30 minutes later, and at the same time the blood pressure, pulse rate and respiration were each noted.

Capillary samples were taken from the lobe of the ear, vasodilatation having been achieved with Trafuril rubifacient cream. The ear was not squeezed.

The same Fluotec was used throughout and its performance was checked against a Hook and
Tucker halothane meter. When set at 2 per cent the Fluotec used in this investigation delivered 1.9 per cent.

RESULTS

The alterations of pH and Pco₂ are shown graphically in figure 2 in which the lines join sets of points belonging to individual patients.

Capillary blood pH.

The mean pre-operative pH was 7.383 (SE 0.045) and this fell to a mean value of 7.304 (SE 0.045) after 45 minutes of anaesthesia. Following anaesthesia it rose again to a mean value of 7.404 (SE 0.05). The pH during anaesthesia was significantly different from the pre- and postoperative levels (t=4.78 and 5.61 respectively, P<0.001 in each case). The slight increase in the mean postoperative pH above the mean preoperative level is not significant (t=1.23; 0.3>P>0.2).

Capillary blood Pco₂.

The mean pre-operative value for Pco₂ was 44.4 mm Hg (SE 3.7) and after 45 minutes this had risen to a mean of 57.3 mm Hg (SE 7.9). In every case there was a rise of Pco₂ (fig. 2 and table I); this varied from the trivial (cases 3 and 13) to the considerable (cases 5, 10 and 11). This rise in mean Pco₂ is significant (t=5.39, P<0.001) and so is the greater variation of Pco₂ during anaesthesia (F=4.53, P<0.01).

The mean postoperative Pco₂ was 40.4 mm Hg (SE 3.75). This is significantly lower than the mean level during anaesthesia (t=6.95, P<0.001); it is also significantly lower than the mean preoperative level (t=2.7, P<0.02). The variance is significantly less than during anaesthesia (F=4.43, P<0.01) and virtually the same as pre-operatively (F=1.02).

The comparison of Pco₂ changes with the clinical measurements.

Figures 3, 4, 5 and 6 show a complete lack of relationship between age, blood pressure change, pulse rate change and respiratory rate change from the pre-operative levels and the rise of Pco₂. The detailed results are in table I. Clearly none of these measurements assists the anaesthetist in deciding whether a patient is hypercarbic or not. A single reading with a Wright

Fig. 2

Changes in pH and Pco₂ in thirteen patients before, during and after anaesthesia with nitrous oxide, oxygen and halothane. Values obtained from capillary blood samples.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Operation</th>
<th>Pulse rate (beats/min)</th>
<th>Minute volume (l.)</th>
<th>Total volume (ml)</th>
<th>Respiratory rate (b.p.m.)</th>
<th>Blood pressure (mm Hg)</th>
<th>pH</th>
<th>Pco₂ (mm Hg)</th>
<th>Intubated or not</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>Simple mastectomy</td>
<td>72 B 63 D 84 A</td>
<td>6.0</td>
<td>182</td>
<td>16 33 16</td>
<td>120/80 100/75 140/90</td>
<td>7.39</td>
<td>46 56 33.5</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>47</td>
<td>Varicose veins</td>
<td>84 B 84 D 58 A</td>
<td>6.0</td>
<td>250</td>
<td>16 24 16</td>
<td>130/75 90/65 125/80</td>
<td>7.35</td>
<td>47 55 38.5</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>29</td>
<td>Hernia</td>
<td>84 B 104 D 92 A</td>
<td>11.5</td>
<td>360</td>
<td>16 32 20</td>
<td>140/90 120/90 130/90</td>
<td>7.38</td>
<td>46.5 49 42</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>44</td>
<td>Varicose veins</td>
<td>82 B 84 D 84 A</td>
<td>6.5</td>
<td>232</td>
<td>16 28 24</td>
<td>130/70 90/65 100/70</td>
<td>7.35</td>
<td>48 57 43.5</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>Hernia</td>
<td>68 B 88 D 88 A</td>
<td>6.5</td>
<td>406</td>
<td>20 16 12</td>
<td>130/90 110/70 120/80</td>
<td>7.43</td>
<td>41.5 68 45</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>57</td>
<td>Hernia</td>
<td>68 B 88 D 96 A</td>
<td>5.5</td>
<td>275</td>
<td>16 20 24</td>
<td>140/90 90/70 120/90</td>
<td>7.37</td>
<td>43 52 40.5</td>
<td>-</td>
</tr>
<tr>
<td>7</td>
<td>50</td>
<td>Hernia</td>
<td>80 B 88 D 84 A</td>
<td>6.0</td>
<td>177</td>
<td>20 31 20</td>
<td>165/105 105/85 125/80</td>
<td>7.36</td>
<td>47 58 43</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>67</td>
<td>Hernia</td>
<td>76 B 82 D 84 A</td>
<td>5.5</td>
<td>162</td>
<td>16 34 20</td>
<td>225/110 140/90 160/100</td>
<td>7.32</td>
<td>46.5 55 40</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>53</td>
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<td>82 B 84 D 80 A</td>
<td>4.8</td>
<td>200</td>
<td>20 24 16</td>
<td>155/90 115/80 160/90</td>
<td>7.39</td>
<td>39.5 56.5 42.5</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>52</td>
<td>Hernia</td>
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<td>6.5</td>
<td>232</td>
<td>16 28 20</td>
<td>120/80 95/75 110/80</td>
<td>7.48</td>
<td>35 72 42.5</td>
<td>-</td>
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<td>11</td>
<td>38</td>
<td>Varicose veins</td>
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<td>6.0</td>
<td>200</td>
<td>16 30 18</td>
<td>120/80 90/75 110/80</td>
<td>7.38</td>
<td>46 69.5 43.5</td>
<td>+</td>
</tr>
<tr>
<td>12</td>
<td>38</td>
<td>Varicose veins</td>
<td>82 B 84 D 88 A</td>
<td>6.0</td>
<td>173</td>
<td>20 35 16</td>
<td>120/80 110/90 130/80</td>
<td>7.39</td>
<td>45 49 38.5</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>53</td>
<td>Hernia</td>
<td>88 B 76 D 72 A</td>
<td>5.5</td>
<td>157</td>
<td>20 35 18</td>
<td>130/90 110/80 150/80</td>
<td>7.39</td>
<td>45.5 48 33</td>
<td>+</td>
</tr>
</tbody>
</table>

* B, D, A = before, during and after anaesthesia.
respirometer appears not to help either. For example, in case 5 the minute volume was 6.5 l./min and the respiratory rate 16 b.p.m. This would suggest much better alveolar ventilation than in case 13, whose minute volume was 5.5 l./min and respiratory rate 35 b.p.m. Yet in case 13 there was a trivial rise of Pco₂ of 2.5 mm Hg after 45 minutes of halothane anaesthesia whereas in case 5 there was a considerable rise of 26.5 mm Hg to a Pco₂ level of 68 mm Hg.

**DISCUSSION**

The pre-operative sample of ear capillary blood was taken immediately after the induction dose of thiopentone, as soon as the patient was asleep. The ventilatory disturbance accompanying a thiopentone induction means that these results cannot be taken as the pre-anaesthetic levels. It was thought better to accept this inaccuracy than to subject patients to ear puncture whilst awake and anticipating surgery.

Since no correction was made for temperature changes occurring during anaesthesia, the results will tend to overestimate the Pco₂ level in patients whose temperature fell during surgery, the formula $\Delta \log \text{Pco}_2/\Delta T=0.018$ (Siggaard-Andersen, 1965) indicating an error of about 8 per cent for a temperature drop of 2°C.

As the 45-minute sample was taken during operation, the respiratory stimulant effect of surgery could be expected to lead to a lesser rise in Pco₂ than would otherwise have taken place.
This could have been avoided by postponing the start of surgery but we were interested in the changes occurring in $P_{CO_2}$ during an operation carried out with this anaesthetic technique.

Like previous workers, we have found evidence of respiratory acidosis during halothane anaesthesia with spontaneous respiration. Comparing these results with those of others, there seems no reason to suggest that the Magill attachment is any worse than non-rebreathing systems in this respect. The rise of $P_{CO_2}$ was variable and, although often not serious, in three patients it rose to about 70 mm Hg. Black and Rea (1964) found that ventricular extrasystoles occurred in two children anaesthetized with 1 per cent halothane at $P_{CO_2}$ levels of 65 and 71 mm Hg. Birt and Cole (1965) showed electrocardiograms of ventricular ectopic rhythms at $P_{CO_2}$ levels of 57, 69.5, 77 and 160 mm Hg during closed circuit halothane anaesthesia and state that such arrhythmias can occur over a wide range of $P_{CO_2}$ levels. It is, therefore, a matter for concern that such a level was reached in three out of thirteen patients. It is disappointing that ordinary observation of blood pressure, pulse rate and respiration rate, or the changes thereof, was of no help in deciding which patients had a serious rise in $P_{CO_2}$. In no case was any irregularity of the pulse noticed.

The single reading with a Wright respirometer was of no help in deciding which individual patient had inadequate ventilation, as judged by the corresponding $P_{CO_2}$ value. It may be that serial measurements of minute volume would give such assistance, but the variations in surgical stimulus during operation would make interpretation of such readings difficult. Perusal of Marshall's results (table II, columns $V_T$, $f$, $V_D - V_{D_{ABG}}/V_T$) does not encourage one to seek any better indication of the alveolar ventilation than the minute volume.

On the other hand, this rise appears to be rapidly, and in most cases completely, reversed in the immediate postoperative period. Indeed, after 30 minutes the average $P_{CO_2}$ value was significantly lower than pre-operatively. This is contrary to the findings of Millar and Marshall (1965) whose patients returned slowly to normal during the first two or three postoperative hours. This may be because none of our patients had received postoperative medication at the time of the third sampling. This had not been intended but we found it was so on examining the records.

It is concluded that if 2 per cent halothane is administered via a Magill attachment to patients breathing spontaneously for 45 minutes, some, but by no means all, will have developed a degree of respiratory acidosis which many anaesthetists would wish to correct by assisting the respiration. None of the common methods of patient assessment including a single reading with a Wright respirometer, would have helped to detect the degree of carbon dioxide retention in the patients studied here.

ACKNOWLEDGEMENTS

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REFERENCES


UNE ETUDE DES TENSIONS DU BIOXYDE DE CARBONE PENDANT L'ANESTHESIE PAR L'HALOTHANE DANS LE CIRCUIT DE MAGILL

SOMMAIRE
Chez treize malades des échantillons de sang ont été prélevés pour la mesure du pH et de la Pco₂, immédiatement après administration de la dose d'induction de thiopentone, 45 minutes après inhalation de 2 pourcents d'halothane par respiration spontanée (avec protoxyde d'azote et oxygène) et 30 minutes après l'opération. Une augmentation réelle de la Pco₂, a été observée, cette augmentation ne correspondait ni avec les modifications du pouls, ni avec la tension artérielle, ni avec la fréquence respiratoire, le volume respiratoire minute ni avec l'âge du malade. Une mesure unique du volume respiratoire minute avec un respiromètre du type Wright n'avait pas de valeur pour la sélection des malades qui présentaient la rétention la plus forte de bioxyde de carbone. Le retour de la Pco₂, à une valeur normale dans la phase post-opératoire était rapide et pour des raisons pratiques complet en 30 minutes.

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