FRESH GAS FLOW REQUIREMENTS USING THE ADE ANAESTHETIC SYSTEM DURING LATE PREGNANCY

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Humphrey (1983) described a new simple, "universal" anaesthetic system which had the capability of changing from Mapleson A to D or E configurations at the flick of two levers. He showed that, in the E configuration, the breathing system functioned as a Bain system (Bain and Spoerel, 1972) when used for intermittent positive pressure ventilation (IPPV).

The production of carbon dioxide is increased by 35% in pregnant women at term (Templeton and Kelman, 1976), and the partial pressure of arterial carbon dioxide ($P_{\text{aCO}_2}$) can be predicted ($P_{\text{aCO}_2} = k \times V_{\text{CO}_2}/V_{\text{FGF}}$ (where $k$ is a constant, $V_{\text{CO}_2}$ equals the volume of carbon dioxide production and $V_{\text{FGF}}$ is the fresh gas flow)) when using Mapleson E systems during IPPV (Baraka, 1969; Spoerel, 1980). Therefore, in pregnant women at term, a 35% increase in fresh gas flow should be necessary to maintain $P_{\text{aCO}_2}$ at preanaesthetic values when using such apparatus.

We have compared the maternal $P_{\text{aCO}_2}$ and $P_{\text{ETCO}_2}$ values attained with FGF of 70 and 100 ml kg$^{-1}$ min$^{-1}$ administered via the ADE anaesthetic system set in the E mode in 32 women requiring elective Caesarean section.

PATIENTS AND METHODS

Thirty-two pregnant patients at term undergoing elective Caesarean section were ventilated with a non-co-axial ADE anaesthetic system (E mode) supplied with fresh gas flows (FGF) of either 70 or 100 ml kg$^{-1}$ min$^{-1}$, on a random basis. Ventilation with an FGF of 70 ml kg$^{-1}$ min$^{-1}$ produced mean $P_{\text{aCO}_2}$ and $P_{\text{ETCO}_2}$ values of 6.48 ± 1.15 kPa and 6.41 ± 0.76 kPa, respectively. Patients were thus hypercapnic, which contrasts with the normocapnia achieved using an FGF of 100 ml kg$^{-1}$ min$^{-1}$ via the ADE system (E mode) ($P_{\text{aCO}_2}$ 5.07 ± 0.7 kPa; $P_{\text{ETCO}_2}$ 4.83 ± 0.46 kPa) (mean values ±SD). The latter FGF is therefore recommended for the pregnant patient at term when using a Mapleson E system such as the Humphrey ADE apparatus.

Patients with evidence of cardiopulmonary disease, hypertension or gross obesity (> 110 kg) were excluded. Informed patient consent and institutional approval were obtained.

The patients were premedicated with 30 ml of sodium citrate 0.3 mol litre$^{-1}$ given orally 1-1.5 h before operation. They were placed on the operating table in the left lateral tilt position. An i.v. infusion of 1 litre of lactated balanced salt solution was commenced.

The anaesthetic technique was standardized and comprised domperidone 10 mg i.v.; preoxygenation for 5-10 min with the ADE system in the A configuration; and the rapid sequence induction of anaesthesia (thiopentone 3.5 mg kg$^{-1}$ and suxamethonium 100 mg) while cricoid pressure was applied. After intubation of the trachea, and confirmation of the correct placement of the tracheal tube (by auscultation), the ADE system was changed to the E configuration and mechan-
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Tidal volumes were set at 10 ml kg\(^{-1}\) and verified using a Wright's respirometer.

A self-calibrated Hewlett-Packard capnometer with nitrous oxide compensation, was connected between the tracheal tube and the catheter mount. Nitrous oxide and oxygen in a 1:1 ratio with 0.5% halothane were administered at flow rates of either 70 or 100 ml kg\(^{-1}\) min\(^{-1}\) from vertically mounted, freely rotating flow meters.

Neuromuscular blockade was maintained with an initial dose of alcuronium 12.5 mg and increments of 2.5 mg, when necessary. At delivery, syntocinon 5 units was given i.v. and 15 units added to the i.v. infusion. Papaveretum 15–20 mg was administered i.v., the halothane discontinued and the ratio of nitrous oxide to oxygen was changed to 2:1 without alteration in the flow rates.

Heart rate was recorded continuously (ECG), and arterial pressure measured before induction and then every 5 min thereafter (automated oscillometer (Sentry)). End-tidal carbon dioxide \((P_{\text{ET}} CO)\) was recorded immediately after intubation, and every 5 min thereafter.

Arterial blood was sampled at 10–15 min after the induction of anaesthesia and \(P_{\text{ET}} CO\) noted simultaneously. Blood samples were stored on ice while being transferred for analysis (Radiometer ABL2 automated blood-gas analyser).

The study was terminated once the neuromuscular blockade had been antagonized. Results were subjected to Student's \(t\) test.

RESULTS

Two patients were excluded from the study since they became hypotensive—with accompanying decreases in \(P_{\text{ET}} CO\).

Two patients in the 70-ml group were changed to higher flow rates when a high \(P_{\text{ET}} CO\) was associated with clinical signs of hypercarbia. These signs abated as the \(P_{\text{ET}} CO\) decreased. The results of these two patients are included in the study, but the end point was taken as the time at which the gas flows were increased.

Of the 30 patients whose results are included, the two groups were similar in weight. Mean weight (SD) in the 70-ml group was 74.4 (13.4) kg, and 80.8 (15.7) kg in the 100-ml group. No statistical difference existed between the two groups in respect of initial \(P_{\text{ET}} CO\), or arterial to end-tidal carbon dioxide partial pressure difference \((P_{\text{ACO}} - P_{\text{ET}} CO)\).

\(P_{\text{ACO}}\) did not correlate well with \(P_{\text{ET}} CO\), the two groups having almost identical \(r\) values, but failing to reach statistical significance. \((P_{\text{ACO}} - P_{\text{ET}} CO)\) was extremely variable with negative and positive values (table I).

Following the methods of Whitesell and colleagues (1981), we used \((P_{\text{ACO}} - P_{\text{ET}} CO)\) to predict the \(P_{\text{ACO}}\). Similarly, we used the ratio of \(P_{\text{ACO}}\) to \(P_{\text{ET}} CO\) in line with the findings of Perrin and co-workers (1983) (table II). In the 70-ml group, the final \(P_{\text{ET}} CO\) was higher than the initial value in all but one patient. The mean (SD) increase in \(P_{\text{ET}} CO\) was 1.14(0.73) kPa. In 80% of these patients, the \(P_{\text{ET}} CO\) was still increasing at the end of the study. In contrast, in the 100-ml group, only 33% of patients had a final \(P_{\text{ET}} CO\) which was higher than the initial measurement and then only minimally, so the mean (SD) decrease in \(P_{\text{ET}} CO\) was 0.16(0.38) kPa (\(P < 0.001\)).

In the 70-ml group there was a significant (\(P < 0.05\)) negative correlation between the patient’s weight and both the final \(P_{\text{ET}} CO\), and the increase from initial to final \(P_{\text{ET}} CO\) values. Such a correlation did not exist in the other group.

DISCUSSION

The Bain circuit is a lightweight anaesthetic breathing system of Mapleson D configuration originally designed for use during head and neck surgery (Bain and Spoerel, 1972). When used for IPPV with the ventilator attached to the bag mount, it becomes a Mapleson E system. When used in this way, the system functions as a partial rebreathing system (Bain and Spoerel, 1973; Spoerel, 1980) where \(P_{\text{ACO}}\) varies predictably with the FGF supplied to the system, provided the minute ventilation \((\dot{V})\) is greater than the \(\dot{V}_{\text{FGF}}\) (Bain and Spoerel, 1972; Spoerel, 1980). Further-

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**TABLE I. Partial pressure (kPa) of arterial carbon dioxide minus end-tidal carbon dioxide**

<table>
<thead>
<tr>
<th>Fresh gas flow</th>
<th>100 ml kg(^{-1}) min(^{-1})</th>
<th>70 ml kg(^{-1}) min(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.24</td>
<td>0.05</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>0.67</td>
<td>0.94</td>
</tr>
<tr>
<td>Minimum</td>
<td>-0.53</td>
<td>-1.24</td>
</tr>
<tr>
<td>Maximum</td>
<td>1.97</td>
<td>2.13</td>
</tr>
<tr>
<td>Range</td>
<td>2.5</td>
<td>3.37</td>
</tr>
</tbody>
</table>

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more, at an FGF of 70 ml kg\(^{-1}\) min\(^{-1}\), normocarbia is achieved (Bain and Spoerel, 1973; Henville and Adams, 1976).

The concentration of alveolar carbon dioxide is equal to \(\frac{\dot{V}_{CO_2}}{\dot{V}_{FGF}}\), providing there is complete mixing of fresh gas with alveolar gas (Baraka, 1969; Bain and Spoerel, 1973, 1975; Spoerel, 1980). Therefore, it can be predicted that, when carbon dioxide production is increased, \(\dot{V}_{FGF}\) should be increased proportionally to maintain normocarbia.

Pregnancy is a condition in which the production of carbon dioxide is increased. Templeton and Kelman (1976) showed that \(\dot{V}_{CO_2}\) increased by 35\% in pregnant women at term. Studies of women in labour have shown increases in \(\dot{V}_{CO_2}\) of up to 75\% above values in non-pregnant individuals (Fisher and Prys-Roberts, 1968). Thus we decided to confine our study to women at term who were not in labour.

Although \(\dot{V}_{CO_2}\) is increased, awake pregnant women hyperventilate to a greater extent than is necessary to maintain \(P_{a\text{CO}_2}\) at normal values. Minute volume is increased by 42\% above non-pregnant values (Cugell et al., 1953) and, usually, results in a compensated respiratory alkalosis (Stenger et al., 1964; Blechner et al., 1968; Templeton and Kelman, 1976; Chappell, 1978). This hyperventilation is attributed to the action of progesterone (Boutourline-Young and Boutourline-Young, 1956).

The increase in FGF to 100 ml kg\(^{-1}\) min\(^{-1}\) was a 43\% increase over that normally recommended to maintain normocarbia when the Bain system is used during IPPV. This increase in FGF matches the increase in ventilation at term, so it might be predicted that the mild hypocarbia of pregnancy would be maintained during IPPV by this increase in FGF.

Humphrey (1983) described a new anaesthetic breathing system which could be changed readily from a Mapleson A to a Mapleson D or E configuration. He showed that, during IPPV in the E configuration, the system behaved as a Bain breathing system and maintained normocarbia with an FGF of 70 ml kg\(^{-1}\) min\(^{-1}\). The use of this breathing system in pregnant women had not been investigated.

The measurement of \(P_{E'\text{CO}_2}\) by capnography is a recognized technique for monitoring \(P_{a\text{CO}_2}\) indirectly during anaesthesia (Nunn and Hill, 1960; Takki, Aromaa and Kauste, 1972). Different values for mean \(\langle P_{a\text{CO}_2} - P_{E'\text{CO}_2} \rangle\) have been published: 0.61 kPa (Nunn and Hill, 1960), 0.46 kPa (Takki, Aromaa and Kauste, 1972), and 0.11 kPa (Whitesell et al., 1981).

In the absence of arterial hypotension, Whitesell and colleagues (1981) found that \(\langle P_{a\text{CO}_2} - P_{E'\text{CO}_2} \rangle\) remained constant during anaesthesia, and suggested that this difference could be quantified by one measurement of \(P_{a\text{CO}_2}\), this value being used to correct subsequent \(P_{E'\text{CO}_2}\) readings.

Perrin and co-workers (1983) showed that, while \(\langle P_{a\text{CO}_2} - P_{E'\text{CO}_2} \rangle\) may vary, for each patient the ratio \(P_{a\text{CO}_2}\) to \(P_{E'\text{CO}_2}\) was constant. Thus, the use of the ratio \(\frac{P_{a\text{CO}_2}}{P_{E'\text{CO}_2}}\) could be used to derive...
$P_aCO_2$ from measurements of $P_eCO_2$. We applied both these methods to produce our indirect measurements of $P_aCO_2$.

While this study was in progress, Kneeshaw, Harvey and Thomas (1984) reported an investigation of the use of an FGF of 120 ml kg$^{-1}$ min$^{-1}$ to maintain $P_aCO_2$ at 4.1–4.4 kPa may have been influenced by the fact that they included patients undergoing an emergency Caesarean section in whom carbon dioxide production is higher. Moreover, they did not measure $P_aCO_2$ directly.

Undoubtedly 70 ml kg$^{-1}$ min$^{-1}$ FGF was too low for patients undergoing elective Caesarean section. However, our prediction that preoperative values of $P_aCO_2$ could be maintained by increasing the FGF to 100 ml kg$^{-1}$min$^{-1}$ was not fulfilled. Although $P_aCO_2$ was not measured before operation, it is unlikely that our study group would have had higher than the “normal” $P_aCO_2$ in pregnancy as no depressant premedication was administered. It is possible that the $P_aCO_2$ increased during the period of apnoea between induction and the commencement of IPPV. This period may have been unduly prolonged as elective lists are used for training our less experienced anaesthetists.

In the 100-ml kg$^{-1}$ min$^{-1}$ FGF group, the fact that the means of the final $P_eCO_2$ and the derived $P_aCO_2$ altered little from initial values supports our view that the increase in FGF to 100 ml kg$^{-1}$ min$^{-1}$ is correct as far as carbon dioxide homeostasis is concerned. Perhaps a short period of manual hyperventilation after intubation would have reduced the $P_aCO_2$ to “normal”, which would then have been maintained by an FGF of 100 ml kg$^{-1}$ min$^{-1}$.

For absolute accuracy in estimating $P_aCO_2$ in pregnant women at term undergoing elective surgery, and being ventilated with a Mapleson E circuit, repeated blood-gas analyses are probably required. In clinical practice an FGF of 100 ml kg$^{-1}$ min$^{-1}$ will effectively avoid excessive hypo- or hypercarbia in most patients.

REFERENCES


