COMPARISON OF VENTILATION AND GAS EXCHANGE IN ANAESTHETIZED INFANTS AND CHILDREN DURING SPONTANEOUS AND ARTIFICIAL VENTILATION

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Measurements of minute and alveolar ventilation ($V_E$ and $V_A$), respiratory frequency, end-tidal carbon dioxide concentration ($E'TCO_2$), deadspace ($V_b$) and carbon dioxide output ($VO_2$) were made in 22 anaesthetized infants and young children during spontaneous (SV) and intermittent positive pressure ventilation (IPPV). In the children who had been given an opioid premedication, $E'TCO_2$ concentrations were significantly greater during SV than the predetermined value set for IPPV. In infants premedicated with atropine alone, $E'TCO_2$ during SV was only slightly greater than during IPPV, and $V_A$ was not changed. A mean tidal volume ($V_T$) of $9.8 \pm 2.5$ ml kg$^{-1}$, and a mean $V_E$ of between 225 and 250 ml min$^{-1}$ kg$^{-1}$, were required to produce $E'TCO_2$ 4.5% during IPPV. Despite a decrease in respiratory frequency, $V_b/V_T$ and $V_b$ per minute were both decreased by IPPV in infants. $VCO_2$ was unchanged in both groups. The decrease in wasted ventilation seen during IPPV in infants supports its use in clinical practice.

Opinions are divided about the relative merits of spontaneous and artificial ventilation during anaesthesia in infants and young children. Opponents of artificial ventilation cite the unnecessary complexity of the technique, whilst its advocates suggest that it ensures adequate ventilation and increases functional residual capacity.

This paper compares measurements of ventilation and gas exchange during spontaneous ventilation in a group of infants and young children with measurements taken subsequently in the same children during artificial ventilation.

PATIENTS AND METHODS

Minute ventilation ($V_E$), respiratory frequency ($f$), end-tidal carbon dioxide concentration ($E'TCO_2$) and mixed expired carbon dioxide fraction ($FE'CO_2$) were measured during surgery in 22 anaesthetized infants and children during spontaneous ventilation (SV), and then during intermittent positive pressure ventilation (IPPV). Alveolar ventilation ($V_A$), deadspace minute ventilation ($V_D$), deadspace/tidal volume ratio ($VD/V_T$), carbon dioxide output ($VO_2$) were calculated. The ages of the patients ranged from 4 weeks to 5 yr and their body weights ($bw$) from 3.7 to 17.9 kg. All patients were free from cardiorespiratory disease. Surgery consisted of elective abdominal, genito-urinary and ophthalmic procedures. No patient had lost a significant quantity of blood at the time of the study.

All patients fasted for 4 - 5 h before surgery. Thirteen patients (mean $bw$ 6.9 ± 3.3 kg) were premedicated with atropine 0.2-0.4 mg i.m. alone; 11 were younger than 1 yr of age and all were less than 10 kg in weight. Nine patients (mean $bw$ 14.5 ± 2.8 kg) received premedication which included an opioid analgesic; all were older than 1 year and all weighed more than 10 kg. Children between 10 and 15 kg $bw$ received Pethidine Compound 0.07 ml kg$^{-1}$ (1 ml contained: pethidine 25 mg, promethazine 6.25 mg and chlorpromazine 6.25 mg) and atropine i.m. 1 h before surgery. Children heavier than 15 kg received papaveretum 0.4 mg kg$^{-1}$ and hyoscine 0.008 mg kg$^{-1}$ i.m. 1.5 h before surgery.

Anaesthesia was induced with cyclopropane in oxygen ($FIO_2$ 0.5). Suxamethonium 1-1.5 mg kg$^{-1}$ was administered i.v. and the trachea intubated. Spontaneous breathing was resumed and anaesthesia maintained with nitrous oxide and 0.5-2% halothane in oxygen ($FIO_2$ 0.5).

The characteristics of the non-rebreathing anaesthetic system (derived from the AMBU Paedi-
Anaesthesia system, the measurement and recording systems and their calibration for $\dot{V}E$, $VT$, $f$, $\dot{E}'CO_2$ and $VCO_2$ have been described in the preceding paper (Lindahl, Hulse and Hatch, 1984). During intermittent positive pressure ventilation at a peak airway pressure of $15 \text{ cm H}_2\text{O}$, the measurement of tidal volume was decreased by less than 5% when measured against volumes determined plethysmographically.

The anaesthetist in charge of the patient was not involved in the study and was at liberty to control the depth of anaesthesia as he saw fit. No measurements were made until anaesthesia was considered stable clinically, the halothane concentration was constant and at least 20 min had elapsed since induction of anaesthesia (Salanitre and Rachow, 1976). After the initial series of measurements had been obtained, tubocurarine $0.4 \text{ mg kg}^{-1}$ was administered, and IPPV commenced by connecting a Nufield 200 ventilator to the system in place of the reservoir bag. The $I:E$ ratio was set at $1:1.5$, giving a frequency of $24 \text{ b.p.m.}$ and the tidal volume was adjusted to achieve $E'CO_2$ $4.5\%$. Measurements were repeated 20 min after the change to controlled ventilation. In eight patients, heart rate and arterial pressure were recorded at 1-min intervals throughout the study (Dinamap 847).

Calculations

Deadspace values were divided into total deadspace ($V_D^{\text{tot}}$) (ml) (including all apparatus deadspace) and net deadspace ($V_D^{\text{net}}$) (ml) by subtraction of 9 ml for apparatus deadspace. $\dot{V}E$, $\dot{V}A$, $VT$, $V_D$, and $\dot{V}D$ were corrected to body temperature and pressure saturated (BTPS), $\dot{V}CO_2$, $VCO_2$ values to ambient temperature and pressure saturated (ATPS). The following formulae were used:

$$\dot{V}CO_2 \text{ (ml min}^{-1}) = \frac{\text{gas collection } \dot{V}E \times P\dot{E}CO_2}{100}$$

$$\dot{V}A \text{ (ml min}^{-1}) = \frac{\dot{V}CO_2 \times 100}{E'CO_2}$$

$$\dot{V}D \text{ (ml min}^{-1}) = \dot{V}E - \dot{V}A$$

$$V_D \text{ (ml)} = \frac{\dot{V}D}{f}$$

Statistics

Mean values and standard deviation (SD) were calculated. Linear regressions and covariance analysis were performed and paired Student's $t$ tests applied to the mean data.

RESULTS

End-tidal carbon dioxide concentration and alveolar ventilation

In the younger patients premedicated with atropine alone, $E'CO_2$ (mean ± 1SD) decreased from $5.4±1.2\%$ during SV to $4.7±0.5\%$ during IPPV ($P<0.05$) (fig. 1). In the older patients receiving an opioid premedication, the corresponding decrease in $E'CO_2$ was from $6.9±1.0\%$ to $4.5±0.4\%$ ($P<0.001$) (fig. 1).

The younger patients premedicated with atropine alone had $\dot{V}A$ of the same magnitude during spontaneous and controlled ventilation (fig. 1). In the opioid group the decreased $E'CO_2$ was reached at a
greater alveolar ventilation \( (P < 0.01) \) (fig. 1). The regression equation for \( VA \) during IPPV for all patients was \( VA = 151 \times kg^{-20}, r = 0.84. \) The mean \( \pm 1 SD \) for \( VA \) was: 148 ± 43 ml min\(^{-1}\) kg\(^{-1}\).

**Minute ventilation**

In the atropine group, the mean value \( \pm 1 SD \) for \( VE \) was decreased from 1980 ± 608 ml min\(^{-1}\) during SV to 1658 ± 855 ml min\(^{-1}\) during IPPV (n.s.) (fig. 2). In those patients who received opioid premedication, the mean value \( \pm 1 SD \) for \( VE \) was increased from 2154 ± 513 ml min\(^{-1}\) during SV to 2917 ± 927 ml min\(^{-1}\) with IPPV \( (P < 0.05) \) (fig. 2).

The relationship between \( VE \) and body weight for all patients during IPPV followed the equation: \( VE = 210 \times kg + 170, r = 0.92. \) The mean value \( \pm 1 SD \) for \( VE \) was 224 ± 52 ml min\(^{-1}\) kg\(^{-1}\).

**Respiratory rate and tidal volume**

In both groups, the respiratory rate during SV was significantly faster than that set for IPPV \( (P < 0.001) \) (fig. 3). In both groups \( VT \) was smaller during SV than IPPV \( (P < 0.001) \) (fig. 3).

The regression equation to the relationship between \( VT \) and body weight (kg) was \( VT = 9.1 \times kg + 5.1, r = 0.86 \) during IPPV. The mean value \( \pm 1 SD \) for \( VT \) was 9.8 ± 2.5 ml kg\(^{-1}\).

**Carbon dioxide output**

The elimination of carbon dioxide per minute was similar during SV and IPPV (fig. 2). The relationship between \( V\text{CO}_2 \) and body weight during IPPV \( (n = 22) \) was expressed by the equation:

\[
\dot{V}\text{CO}_2 = 6.2 \times kg + 0.70, \quad r = 0.86.
\]

There was an almost direct proportionality between \( \dot{V}\text{CO}_2 \) and body weight during IPPV with a mean value \( \pm 1 SD \) of 6.3 ± 1.5 ml min\(^{-1}\) kg\(^{-1}\).

**Deadspace**

In the younger patients (premedicated with atropine alone) deadspace ventilation per minute \( (Vd^\text{tot}) \) was decreased during IPPV \( (P < 0.05) \), while it was unchanged between the two modes of ventilation in the opioid group (fig. 4). During IPPV, deadspace per breath \( (Vd^\text{breath}) \) was greater than during SV (fig. 4) in the atropine group \( (P < 0.01) \) and the opioid group, although in the latter group the difference was not significant. The mean ratio of \( Vd^\text{tot} \) to \( VT \) \( \pm 1 SD \) in the atropine group decreased from 0.49 ± 0.14 during SV to 0.38 ± 0.10 during IPPV \( (P < 0.05) \) (fig. 4). In the opioid group, this ratio decreased from 0.44 ± 0.05 during SV to 0.28 ± 0.11 during IPPV \( (P < 0.001) \) (fig. 4).
Heart rate and arterial pressure
There were no significant differences in heart rate or arterial pressure during spontaneous ventilation compared with those obtained during IPPV in the eight patients in whom the Dinamap recorder was used.

DISCUSSION
End-tidal carbon dioxide concentration and alveolar ventilation
In the younger patients premedicated with atropine, $E'CO_2$ during spontaneous ventilation was low and only had to be decreased by an average of 0.7% to reach the desired value during IPPV. It is unlikely that the low end-tidal carbon dioxide concentration during SV was the result of inaccurate measurements at the high respiratory rates sometimes found since, in all patients, an end-tidal carbon dioxide plateau phase was reached, suggesting that the response time of the carbon dioxide meter was adequate at respiratory rates up to 80 b.p.m. As might be expected from the small difference in $E'CO_2$ between SV and IPPV in the younger patients, $VA$ was similar with the two forms of ventilation.

The higher end-tidal carbon dioxide concentration during SV in the older patients was probably a result of the opioid premedication used in this age group, and has been discussed previously (Lindahl, Wallgren and Wahlin, 1966). $E'CO_2$ was decreased by an average of 2.4% to reach the value desired during IPPV, a decrease which was achieved at an increased alveolar ventilation ($P<0.001$) (fig. 1).

The high correlations between $E'CO_2$ and $VA$ illustrated by these measurements in both groups could be expected, as $VA$ is known to be related to the arterial carbon dioxide tension and recent work (Valentin, Lomholt and Thorup, 1982) demonstrated a systematic difference of about 0.6 kPa between arterial and end-tidal carbon dioxide tensions for anaesthetized patients with normal lungs.

Minute ventilation
In a previous study the average minute ventilation, measured in anaesthetized adult patients, was increased by approximately 130% during mechanical ventilation as compared with spontaneous ventilation, resulting in a decrease in arterial carbon dioxide tension by 0.7 kPa (Bindslev et al., 1981). In the present study $VE$ was higher during IPPV in the older patients receiving opioid premedication and the average minute volume required to decrease $E'CO_2$ by 2.4% was 135% higher than that during SV ($P<0.05$) (fig. 2). However, in the younger age group, the decrease in mean $E'CO_2$ by 0.7% during IPPV was achieved at a minute volume which was not significantly lower than that during SV. This suggests that there was considerable wasted ventilation during spontaneous breathing in these younger patients, possibly because of the relatively high apparatus deadspace in this group, and high respiratory frequency during SV.

The tidal volumes reported during IPPV compare well with previously published standards for artificial ventilation in infants and children. Okmian, Wallgren and Wahlin (1966) found that a tidal volume of 10 ml kg$^{-1}$ at a rate of 20 b.p.m., resulted in a capillary carbon dioxide tension of 4.7 kPa, and Lindahl, Okmian and Thomson (1979) used a tidal volume of 12 ml kg$^{-1}$ at a rate of 20 b.p.m. to obtain a mean arterial carbon dioxide tension of 3.7 kPa.

Minute volumes of between 225 and 250 ml min$^{-1}$ kg$^{-1}$ were required to produce $E'CO_2$ 4.5%, but it should be remembered that this will depend on the compressible volume of the circuit used.

Carbon dioxide output
Carbon dioxide production for the whole group was of the same magnitude during SV and IPPV as found in a previous study by Bain and Spoerel (1976). The value of $6.3 \pm 1.5$ ml min$^{-1}$ kg$^{-1}$ for carbon dioxide output during controlled ventilation in children was in agreement with that published by Nightingale and Lambert in 1978.

The similarity in carbon dioxide production suggests that the metabolic state was similar during SV and IPPV. A higher metabolic rate might have been expected in the 13 younger patients premedicated with atropine alone than in the nine who received a narcotic. In fact, in 10 of the 13 patients in the atropine group, $VCO_2$ was lower during IPPV than SV, although the difference between the two groups just failed to reach significance.

Deadspace ventilation
The high deadspace ventilation per minute during SV in small children was most probably attributable to their high respiratory frequencies; it was significantly decreased by controlled ventilation, although deadspace per breath was increased. In the older patients $VD$ was unchanged between the two modes of ventilation (fig. 4).

The importance of respiratory frequency for ven-
VENTILATION IN ANAESTHETIZED CHILDREN


REFERENCES

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It is concluded that in younger patients, the rapid, shallow spontaneous breathing and apparatus deadspace which is inevitably relatively large, increased the amount of wasted ventilation. This resulted in a high minute ventilation without a significant effect on carbon dioxide output. Although the choice between spontaneous and artificial ventilation depends on many factors, the improved ventilatory efficiency of artificial ventilation, seen particularly in the infants, justifies the frequent use of IPPV during anaesthesia in this age group.

REFERENCES


