VENTILATION AND GAS EXCHANGE DURING ANAESTHESIA AND SURGERY IN SPONTANEOUSLY BREATHING INFANTS AND CHILDREN

S. G. E. LINDAHL, M. G. HULSE AND D. J. HATCH

SUMMARY

Minute ventilation (VE) (ml min⁻¹), respiratory frequency (f), mixed expired carbon dioxide fraction (FE\textsubscript{CO2}) and end-tidal carbon dioxide concentration (E′CO\textsubscript{2}) (%) were measured, and alveolar ventilation (VA), deadspace (VD), deadspace/tidal volume ratio (VD/VT) and carbon dioxide output (VCO\textsubscript{2}) calculated in 58 anaesthetized, spontaneously breathing infants and children weighing 2.8–20.5 kg. Although minute volumes varied, tidal volume correlated well with weight (r = 0.83), with a mean tidal volume (± 1SD) of 5.2 ± 1.2 ml kg⁻¹. It was concluded that, by the use of mean VT + 1SD (approximated to 6 ml kg⁻¹) the fresh gas flow in ml min⁻¹ should be set at 2.5 × 6 ml kg × f/(15 × kg × f) to avoid rebreathing in various T-piece systems in anaesthetized, intubated and spontaneously breathing infants up to a body weight of 20 kg. End-tidal carbon dioxide concentration was lower in younger patients who were premedicated with atropine alone than in the older ones who received opioid premedication also. Respiratory frequency, VD/Vf and total Vd per minute were higher in the younger age group, which explained the finding of a high VE in relation to VCO₂ for these patients. This inefficiency of ventilation emphasizes the need to minimize apparatus deadspace in breathing systems used for small infants.

The place of anaesthesia with spontaneous ventilation in infants and young children has been a subject of debate for some years. There have been relatively few studies of the effects of anaesthesia on ventilation and gas exchange in paediatric patients and those published have been devoted to specific aspects of the subject (Wilson and Harrison, 1964; Lunn, 1968; Bain and Spoerel, 1977; Lindahl, Olsson and Thomson, 1981).

Mapleson D and E systems are used widely in paediatric anaesthesia and, to avoid rebreathing of expired carbon dioxide, fresh gas flows of two-and-a-half to three times the minute volume have been recommended (Conway, Seeley and Barnes, 1977; Byrick, 1980). However, there is little information on minute volume in anaesthetized children.

The purpose of this study was to measure tidal volume, frequency of respiration, minute volume, and end-tidal carbon dioxide concentration, and to calculate deadspace, alveolar ventilation and carbon dioxide output in anaesthetized infants and young children. These data should help to calculate the fresh gas flow required to avoid rebreathing with these systems. In addition, these normal data should enhance the value of end-tidal carbon dioxide measurements which are used increasingly during anaesthesia.

PATIENTS AND METHODS

Minute ventilation (VE), respiratory frequency (f), end-tidal carbon dioxide concentration (E′CO\textsubscript{2}) (%) and mixed expired carbon dioxide fraction (FE\textsubscript{CO2}) were measured during surgery in 58 anaesthetized, spontaneously breathing infants and children (38 male). Alveolar ventilation (VA), deadspace minute ventilation (VD), deadspace volume (VD), VD/Vf ratio and carbon dioxide output (VCO₂) were calculated. The ages of the patients ranged from 1 month to 6 yr and their body weights (bw) from 2.8 to 20.5 kg. All were free from cardiorespiratory disease. Surgery consisted of elective abdominal, genito-urinary and ophthalmic procedures. No patient had significant blood loss at the time of the study.

All patients fasted for 4–5 h before surgery. Twenty-two patients (mean bw 6.9 kg) received atropine 0.2–0.4 mg i.m.; 16 were younger than 1 year of age and 19 were less than 10 kg in weight. Thirty-six patients (mean bw 13.9 kg) received premedication which included an opioid analgesic. All were older than 1 year and all but three weighed more than 10 kg. Most children between 10 and 15 kg bw received Pethidine Compound 0.07 ml kg⁻¹ (1 ml contains: pethidine 25 mg, pro-
methazine 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.

Anaesthesia was induced with cyclopropane in oxygen (F₁O₂ 0.5). The trachea was intubated in all patients after the injection of suxamethonium 1–1.5 mg kg⁻¹ i.v. Once spontaneous ventilation had resumed anaesthesia was maintained with nitrous oxide and 0.5–2% halothane in oxygen (F₁O₂ 0.5). In 14 patients undergoing circumcision, epispadias or hypospadias repair, caudal analgesia using 0.25% bupivacaine 0.5 ml kg⁻¹ was established after the induction of general anaesthesia.

The non-rebreathing anaesthetic system was derived from the AMBU Paedi-Anaesthesia system (fig. 1). The inspiratory limb, which was separated from the expiratory limb by a low resistance non-rebreathing valve, included a reservoir bag and a variable pressure relief valve. \( V_E \) was measured by electrical integration of the flow signal from a heated pneumotachograph (Fleisch No. 0) with a differential pressure transducer (Validyne MP 45-1-871, range ± 2 cm H₂O). This was placed in the deadspace of the system as closely as possible to the tracheal tube connector. Flow and volume signals were monitored on an oscilloscope (Tektronix 5112) and stored on a tape recorder (Racal Store 4) for eventual transfer to a u.v. paper record (S.E. Labs (EMI) Ltd, S.E. 3006). Flow calibration of the pneumotachograph was carried out using accurately calibrated rotameters (Rotameter Mfg Co., series 1100) and volume calibration using a syringe. A 50% nitrous oxide in oxygen mixture was used for the calibrations.

\( E'C_{O₂} \) (%) was monitored continuously by an in-line infra-red capnograph (Siemens—Elema 130). This is a non-gas sampling capnograph which measures directly the carbon dioxide content of gas passing between two windows in a small cuvette (internal volume 2.5 ml) placed in the system deadspace. The carbon dioxide wave form was recorded on an ink-jet recorder (Mingograph, Siemens—Elema EM41). The presence of an end-tidal plateau was verified for all patients included in the study. The deadspace of the pneumotachograph, capnograph cuvette, non-rebreathing valve and the rubber connections up to, but not including, the tracheal tube connector was 9 ml (measured by water displacement).

The inspiratory and expiratory resistance of the non-rebreathing valve, pneumotachograph and capnograph cuvette was 15.0 cm H₂O litre⁻¹ s⁻¹. This value was constant up to a flow rate of 10 litre min⁻¹, which was the highest obtained in this study.

Expired gas passed to a dry gas meter (Standard gas meter, AB, Nordgas, Stockholm, Sweden) and then to a three-way valve from which a timed collection, over 3–5 min, of a measured volume of gas could be made into a Douglas bag. The \( FECO₂ \) of this gas was measured using an infra-red capnograph (Gould Godart MK II). Both capnographs were calibrated daily with certified carbon dioxide—nitrogen—oxygen gas mixtures (British Oxygen Company) within the measuring range. Because of dispersion of the infra-red spectrum by nitrous oxide in oxygen mixtures, all carbon dioxide values obtained were corrected by a factor of 0.95.

Fresh gas flow was set slightly in excess of the patient's minute volume, so that the reservoir bag remained well filled but not tense. In five patients the competence of the valve to prevent any leakage of expired gas into the inspiratory limb was assessed by sampling gas from the inspiratory limb just upstream of the valve with the Gould capnograph. Carbon dioxide was not detected.

The anaesthetist in charge of the case, who was not involved in the study, was at liberty to control the depth of anaesthesia as he saw fit. No measurements were made until anaesthesia was considered

![Fig. 1. The non-rebreathing system and measuring apparatus.](image-url)
stable clinically, the halothane concentration was constant and at least 20 min had elapsed since induction (Salanitre and Rackow, 1976).

**Calculations**

Deadspace values were divided into total deadspace \( (V_{DT}) \) (ml) including all apparatus deadspace and net deadspace \( (V_{DN}) \) (ml) by subtraction of 9 ml for apparatus deadspace. \( VE, VA, VT, VD \) and \( VD \) were corrected to body temperature and pressure saturated (BTPS) and \( VCO_2 \) values to ambient temperature and pressure saturated (ATPS).

The following formulae were used:

\[
VCO_2 (ml \text{ min}^{-1}) = \frac{\text{gas collection} \times V_E \times F_{ECO_2}}{100}
\]

\[
VA (ml \text{ min}^{-1}) = \frac{VCO_2 \times 100}{F_{ECO_2}}
\]

\[
VT (ml \text{ min}^{-1}) = V_E - VA
\]

\[
VD (ml) = \frac{VD}{f}
\]

**Statistics**

Mean values and standard deviation (SD) were calculated. Linear regressions and covariance analyses were performed and unpaired Student’s \( t \) test applied to the mean data.

**RESULTS**

**Minute ventilation**

The relationship between \( VE \) and body weight (kg) in all 58 patients is shown in figure 2. The relationship for patients premedicated with atropine alone was: \( VE = 144 \times kg + 940, \ r = 0.70. \) For patients after opioid premedication, the regression equation was: \( VE = 107 \times kg + 680, \ r = 0.51. \) The slopes of these regression lines did not differ significantly. However, the difference between the mean values of the two regressions was significant \( (P<0.01). \)

**Tidal volume.** The linear relationship between \( VT \) and body weight is shown in figure 3. From data divided on the basis of premedication, no significant differences between the regressions were obtained. A ratio standard was derived from these data and gave a mean value \( \pm 1 \)SD for \( VT \) in all children studied of \( 5.2 \pm 1.2 \) ml kg\(^{-1}.\)

**Respiratory frequency.** A wide range (18–96 b.p.m.) was encountered in respiratory frequency. The mean frequency in the atropine group was \( 61 \pm 19 \) b.p.m. and in the opioid group \( 32 \pm 9 \) b.p.m. The difference between these mean values was significant \( (P<0.001). \)
There was a tendency for respiratory frequency and minute ventilation to be lower in patients who received caudal analgesia. However, the inclusion of these patients did not significantly affect the above results.

End-tidal carbon dioxide concentration and alveolar ventilation

The range of E′CO₂ values for the whole group was 3.5–8.5% with a mean value (±SD) of 6.2±1.1%. The mean E′CO₂ for infants premedicated with atropine alone was 5.5±1.1% and for those whose premedication included a narcotic analgesic, 6.6±0.9%, the difference between these mean values was significant (fig. 5) (P<0.001).

A linear relationship was shown to exist between VA and body weight, described by the regression equation: VA = 70.8 x kg + 420, r = 0.62. Regression equations for the atropine group (VA = 111 x kg + 199, r = 0.71) and opioid group (VA = 70 x kg + 392, r = 0.42) were not significantly different. Mean values of VA (ml min⁻¹ kg⁻¹) were lower for those receiving opioid premedication (fig. 5) (P<0.01).

Deadspace

VΔ and VΔ were linearly correlated with body weight and were unaffected by the type of premedication (VΔ = 1.26 x kg + 7.6, r = 0.81; VΔ = 1.27 x kg – 1.5, r = 0.81). VΔ/VT ratio ranged from 0.16 to 0.75 and was higher in the rapidly breathing non-narcotized infants (fig. 4) (P<0.001). There was a negative correlation with body weight (r = 0.53) and a close positive correlation (r = 0.63) (fig. 6A) with respiratory frequency. VΔ/VT ratio showed no such correlation (fig. 6B).

Carbon dioxide output

Linear regression of VCO₂ on body weight, body surface area and body weight each showed r values of 0.81. Body length gave an r value of 0.78 and age an r value of 0.70.

The relationship between VCO₂ and kg in all 58 patients is shown in figure 7. The regression equation for patients premedicated with atropine only was: VCO₂ = 6.6 x kg + 2.8, r = 0.81 and for those receiving opioid premedication: VCO₂ = 4.9 x kg + 14.1, r = 0.62. No statistically significant difference was revealed between these two regression lines.

The ratio of VE to VCO₂ for each patient was linearly and negatively correlated to body weight in the atropine group following the equation: V/E/VCO₂ = −2.5 x kg + 62.7, r = 0.57 (P<0.01).
This relationship did not correlate in the narcotic group \( \frac{\dot{V}E}{\dot{V}CO_2} = -0.5 \times \text{kg} + 34.3, \; r = 0.26, \; P < 0.1 \). The two regression slopes were significantly different \( P < 0.025 \).

**DISCUSSION**

In order to obtain accurate \( \dot{V}CO_2 \) measurements, a non-rebreathing system was used. This system had a slightly increased resistance compared with a conventional T-piece, which at the flow rates seen in the study was equivalent to the total airway resistance of a 10-kg child or to the nasal resistance of a neonate or small infant. This slight increase in resistance was not thought to affect the results significantly. As shown previously (Lindahl, Nordström and Olsson, 1981), it is possible for excess fresh gas flow to leak directly across the non-rebreathing valve and dilute the expired gas. This gas does not influence \( \dot{V}CO_2 \) estimation, but measurements of \( \dot{V}E \) must be obtained by other means. This was obtained in the present study from a pneumotachograph placed in the deadspace. The deadspace of the system, including pneumotachograph and capnograph cuvette, was 9 ml, which was similar to that of a conventional T-piece circuit with a standard catheter mount.

**Minute ventilation**

Minute ventilation is not measured easily in anaesthetized infants and children, and is affected by factors such as the depth of anaesthesia and surgical stimulation. Published results show considerable variability (Wilson and Harrison, 1964; Lunn, 1968). Studies in sedated infants and small children have also yielded variable results (Krieger, 1963; Phelan and Williams, 1969; Doershuk et al., 1970). Minute ventilation has been measured in the awake state in neonates (Cook et al., 1955; Nelson et al., 1962) but extrapolation from the neonate to the older child may not be justified.

Results obtained in this study were compared with the pooled data from the three studies on sedated children mentioned above, which included a total of 82 patients weighing between 2 and 14 kg. There was no significant difference in \( \dot{V}E \) between our atropine group and the pooled data, although mean \( \dot{V}T \) was decreased by approximately 30% during anaesthesia, and mean respiratory frequency was doubled. In the opioid group, \( \dot{V}T \) was decreased similarly, but respiratory frequency remained the same, producing a significant decrease in \( \dot{V}E \) \( P < 0.001 \). This could be a direct effect of the opioid or reflect the fact that the relatively higher apparatus deadspace in the younger group was causing an increase in respiratory frequency.

The behaviour of the commonly-used Mapleson D and E systems during spontaneous ventilation in children has been studied extensively (Ayre, 1937; Inkster, 1956; Onchi, Hayashi and Ueyama, 1957; Mapleson, 1958; Bain and Spoerel, 1972; Willis, Pender and Mapleson, 1975; Henville and Adams, 1976; Barnes et al., 1976; Thomsen, 1980). It is accepted widely that, if the volume of the expiratory limb is greater than the tidal volume, a fresh gas flow of two-and-a-half to three times the minute volume is required to prevent rebreathing (Willis, Pender and Mapleson, 1975; Conway, Seeley and Barnes, 1977; Byrick, 1980). Thus, the value of a simple, clinically useful estimate of minute volume is clear. Unfortunately, the linear regression of minute volume on weight does not pass close to the origin of the axes, but has a large positive intercept with the ordinate, making inappropriate the use of a simple standard ratio derived from mean values. The dangers of using such inappropriate standard ratios in respect of individuals whose weights are significantly different from the mean weight of the group was commented upon by Tanner (1949). In this case the predicted \( \dot{V}E \) would be underestimated seriously for small infants. This problem was recognized by Froese and Rose (1982), who suggested fresh gas flows of 3 \( (1000 \text{ ml min}^{-1} + 100 \text{ ml kg}^{-1}) \) in children of 10–30 kg, with an endotracheal tube in place, during spontaneous ventilation. The change from atropine to narcotic premedication occurred in the present study at approximately 10 kg. Accepting
that fresh gas flows of two-and-a-half to three times the minute ventilation should be used with spontaneous breathing, the requirements calculated from our regression data would be 3(140 x kg + 940) below and 3(107 x kg + 680) above 10 kg. However, these formulae are difficult to memorize.

The wide scatter of \( VE \) values, caused largely by variations in frequency means that, if these data are used to avoid rebreathing in every patient, unnecessarily high flows will be used frequently. On the other hand, there was a good correlation between \( VT \) and body weight (fig. 3) with a regression line which did pass close to the origin of both axes. \( VT \) could therefore be predicted with acceptable accuracy at 5.2 ± 1.2 ml kg\(^{-1}\). This compares well with the figure of 5.5 ml kg\(^{-1}\) quoted by Wilson and Harrison (1964), during halothane anaesthesia. Since respiratory frequency can be counted easily, it seems reasonable to estimate \( VE \) from the formula:

\[
VE = VT \times f \times kg.
\]

Applying the fresh gas flow recommendations of Froese and Rose (1982) to the minute volumes recorded in the present series (fig. 2) suggests that they are a little too low to avoid rebreathing in all instances. Their recommendations give fresh gas flows similar to those based on 2.5 times \( VE \) estimated from the expression 5.2 x kg x f. Hence, the mean \( VT \) of 5.2 ml kg\(^{-1}\) found in this study was increased by 1.2 ml kg\(^{-1}\) (that is + 1SD) and for clinical purposes approximated to 6 ml kg\(^{-1}\). Therefore, we would recommend fresh gas flows in ml min\(^{-1}\) of 2.5 x 6 x kg x f (15 x kg x f) for clinical use from birth to 20 kg. Above this weight, fresh gas flows become uneconomical and a more appropriate system should be used.

End-tidal carbon dioxide concentration and alveolar ventilation

This study showed that anaesthesia with spontaneous ventilation in infants premedicated with atropine alone was not accompanied by a significant increase in \( E'\text{CO}_2 \), and alveolar ventilation was well maintained. Our values for \( VA \) were similar to those reported by Evans, Hogg and Rosen (1977), Nelson and co-workers (1962) and Cook and colleagues (1955), for awake neonates. In children older than 1 year of age, where sedative premedication was considered desirable and an opioid analgesic was used, \( E'\text{CO}_2 \) values during anaesthesia were greater. This was presumably a result of the significant decrease in alveolar ventilation in this group (fig. 5).

Deadspace and carbon dioxide output

The calculations of deadspace were made from an estimation of alveolar carbon dioxide concentration using \( E'\text{CO}_2 \). During anaesthesia in healthy adults there is an arterial—end-tidal carbon dioxide tension difference (\( P_{\text{A}CO_2} - P_{E\text{CO}_2} \)) as a result of an increase in alveolar deadspace. Ramwell (1958) and Nunn and Hill (1960), found (\( P_{\text{A}CO_2} - P_{E\text{CO}_2} \)) during anaesthesia in healthy adults to be 0.7 kPa (5 mm Hg). Thus, the values obtained in this study underestimate physiological deadspace and approximate to anatomical deadspace. The difference between these two values cannot be assessed without analyses of arterial blood-gas tensions.

Anaesthetic systems always contain some apparatus deadspace, although tracheal intubation decreases deadspace by more than 50% in adults (Nunn, Campbell and Peckett, 1959). The deadspace of the system used in the present study was increased by the volume of the pneumotachograph head and the cuvette for end-tidal carbon dioxide measurements. However, the extra deadspace of 9 ml was similar to that of a standard T-piece with elastomeric tracheal tube adaptor. As in the clinical situation, this apparatus deadspace was constant throughout the age range studied, but formed a relatively higher percentage of the total deadspace in the smaller patients. Thus, the ratio \( VD^{inf}/VT \) was greater in younger patients, although when apparatus deadspace was subtracted, the ratio \( VD^{expr}/VT \) was found to be constant at a mean value of 0.22 ± 0.09. \( VD^{expr} \) calculated from the regression equation presented resulted in a deadspace value of approximately 0.8 ml kg\(^{-1}\) for a 3-kg infant. Values published by Nelson and co-workers (1962) and by Cook and colleagues (1955), for awake, non-intubated infants of similar weight were almost exactly double this — 1.7 ml kg\(^{-1}\).

The infants in this series premedicated with atropine alone were able to maintain a normal end-tidal carbon dioxide concentration despite increases in respiratory frequency and high values for total deadspace ventilation per minute which were as high as those seen in the older children. While this appeared to be acceptable clinically in these infants, it may not be so well tolerated by those with cardiorespiratory disease. In such patients, either a decrease in apparatus deadspace or the use of controlled ventilation might be considered.

Comparison of the values for carbon dioxide output between this and other published series is shown in table I. There is little difference between pub-
TABLE I. Published data on carbon dioxide output in anaesthetized and awake infants and children. All data at ATPS except when otherwise stated. *Spontaneous ventilation before surgery; **controlled ventilation, values at A TPD; ***controlled and spontaneous ventilation; †calculated from basal metabolic rate measurements; ‡neonates

<table>
<thead>
<tr>
<th>Body weight (kg)</th>
<th>Anaesthetized</th>
<th>Awake</th>
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<tr>
<td>This study</td>
<td>Lindahl, Olsson and Thomson (1981)*</td>
<td>Nightingale and Lambert (1978)**</td>
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<tr>
<td>5</td>
<td>37.2</td>
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<td>10</td>
<td>63.0</td>
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<td>15</td>
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<td>20</td>
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published values for awake and anaesthetized children. The higher values in the present study compared with the results published by Lindahl, Olsson and Thomson (1981) using the same technique, may be explained by the fact that in the latter series measurements were made before surgery. Other reasons for differences in carbon dioxide output include depth of anaesthesia, mode of ventilation, preoperative fasting, premedication and type of surgical procedure.

The ratio of \( \text{VE} \) to \( \text{VCO}_2 \) was much greater (mean 46) and showed a negative correlation with body weight in the atropine group whereas in the opioid group it was lower (mean 27) and showed no such correlation. This higher minute ventilation per unit carbon dioxide output of the younger subjects is most probably explained by the high deadspace ventilation, since it was not seen in the ratio of alveolar ventilation to carbon dioxide output. Therefore ventilation and gas exchange were less efficient in the small infant premedicated with atropine alone than in the larger child whose premedication included opioid analgesia. Apparatus deadspace made a relatively large contribution to this inefficiency in the very young.

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VÉNULATION ET ÉCHANGES GAZEUX AU COURS DE L’ANESTHÉSIE ET DE LA CHIRURGIE CHEZ DES NOUVEAU-NES ET DES ENFANTS EN VENTILATION SPONTANÉE

**RESUME**

La ventilation — minute (VE)(ml min⁻¹), la fréquence respiratoire (f), la fraction expirée mêlée de dioxyde de carbone (FICO₂) et la concentration télé-expiratoire de dioxyde de carbone (FICO₂) (%) ont été mesurées et la ventilation alvéolaire (VA), l’espace mort physiologique (Vd), le rapport de l’espace mort au volume courant (Vd/VT) et le débit de CO₂ expiré (VCO₂) ont été calculés chez 58 nouveau-nés et enfants pesant de 2,8 à 20,5 kg anesthésiés, ventilant spontanément. Malgré des ventilations variables, le volume courant était bien corréllé au poids (r = 0,83) avec un volume courant moyen (±1DS) de 5,2 ± 1,2 ml kg⁻¹. Par conséquent, en utilisant la valeur moyenne de VE + 1DS (approchée à 5 ml kg⁻¹), il faudrait fixer le débit de gaz frais en ml min⁻¹ à 2,5 x 6 x kg x f = 15 x kg x f pour éviter tout rebreathing dans les différents systèmes comportant des pièces en t chez des nourrissons intubés en ventilation spontanée et pesant jusqu’à 20 kg. La concentration télé-expiratoire de CO₂ était plus basse chez les enfants les plus jeunes, dont la prémédicamtion ne comportait que de l’atropine, que chez les plus âgés qui recevaient aussi des opiacés. La fréquence respiratoire, le Vd/VT et le Ve total par minute étaient plus élevés chez les plus jeunes ce qui explique la découverte d’une VE élevée par rapport au VCO₂ chez ces enfants. Ce manque d’efficacité ventilatoire souligne la nécessité de minimiser l’espace mort dû à l’appareillage dans les circuits utilisés chez les jeunes enfants.

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VENTILACIÓN E INTERCAMBIO DE GAS DURANTE LA ANESTESIA Y LA CIRUGÍA EN NIÑOS Y CRIATURAS QUE RESPIRAN ESPONTÁNEAMENTE

**SUMARIO**

Se midieron la ventilación-minuto (VE) ml min⁻¹), la frecuencia respiratoria (f), la fracción mezclada de anhídrido carbónico expirado (FICO₂) y la concentración de anhídrido carbónico respiratorio-terminal (FICO₂), y se calcularon la ventilación alveolar (VA), el espacio muerto (Vb), la relación espacio muerto/volume respiratorio (Vd/Vt) así como el rendimiento de anhídrido carbónico (VCO₂) en 58 niños y criaturas bajo respiración espontánea cuyo peso variaba de 2,8 a 20,5 kg. Aunque el volumen-minuto varió, el volumen respiratorio se encon-
traba bien relacionado con el peso \( (r = 0.83) \) con un volumen respiratorio medio \( (\pm 1\text{SD}) \) de \( 5.2 \pm 1.2 \text{ ml kg}^{-1} \). Se concluyó que, mediante el uso del \( V_t + 1\text{SD} \) promedio (reondeado a \( 6 \text{ ml kg}^{-1} \)), la corriente de gas nuevo en \( \text{ml min}^{-1} \) debería establecerse en \( 2,5 \times 6 \times \text{kg x f} \) \( (15 \times \text{kg x f}) \) para evitar la re- respiración en varios sistemas de tubos en T de las criaturas anestesiadas, intubadas y respirando espontáneamente hasta un peso corporal de 20 kg. La concentración respiratoría-terminal de anhídrido carbónico era más baja en los pacientes menores que habían recibido una premedicación con atropina sola que en los mayores que habían recibido premedicación opiáceas también. La frecuencia respiratoria, el \( V_d/V_t \) y el \( V_b \) total por minuto eran más altos en el grupo de pacientes más jóvenes, lo que explica que el \( V_t \) era alto en relación con el \( V_{CO_2} \) de dichos pacientes. Esta ineficacia de ventilación subraya la necesidad de reducir al mínimo el espacio muerto de los aparatos en los sistemas respiratorios usados con pequeñas criaturas.