VENTILATORY RESPONSES OF CHILDREN TO CHANGES IN DEADSPACE VOLUME

Studies Using the T-Piece (Mapleson F) System

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Alveolar deadspace is greater during anaesthesia than in the conscious state (Nunn and Hill, 1960), and during spontaneous ventilation it increases with depth of anaesthesia (Kain, Panday and Nunn, 1969). However, although the additional influence of apparatus deadspace was investigated by Kain, Panday and Nunn (1969), few studies have considered the effects of apparatus deadspace on anaesthetized children, although these are relatively more important in smaller patients. In one study in children anaesthetized with halothane (Lindahl, Hulse and Hatch, 1984), smaller children were shown to have a higher percentage deadspace ventilation as a result of their higher respiratory rates and of a higher total deadspace to tidal volume ratio, while net deadspace (apparatus deadspace excluded) to tidal volume ratio did not differ with rate.

In this study, the ventilatory responses to two different apparatus deadspaces (2 and 16 ml) were compared in spontaneously breathing children during halothane anaesthesia, and the efficiency of ventilation in younger and older children evaluated.

PATIENTS AND METHODS

Twelve patients, aged between 3 weeks and 6 yr, and weighing between 4.3 and 25.3 kg (mean ± SD: 11 kg ± 5.6) were studied. All were in-patients, free from cardiorespiratory disease, undergoing a variety of minor surgical procedures such as lens aspiration, corrections of hypospadias, repair of cleft palate and orchidopexy. Seven children younger than 1 year of age and weighing less that 10 kg (mean ± SD: 7.6 ± 2.1 kg) received atropine 0.2-0.4 mg i.m. for premedication. Of the other five patients (weight > 10 kg; mean ± SD: 15.8 ± 6.2 kg), three weighing between 10 and 15 kg were premedicated with pethidine compound 0.07 ml kg$^{-1}$ and atropine 0.2–0.4 mg i.m. about 1.5 h before the operation. (Pethidine compound 1 ml contains pethidine 25 mg, chlorpromazine 6.25 mg and promethazine 6.25 mg.) The other two patients (> 15 kg) received papaveretum 0.4 mg kg$^{-1}$ and hyoscine 0.008 mg kg$^{-1}$ i.m. 1.5 h before surgery.

SUMMARY

Twelve patients (4.3–25.3 kg) undergoing minor surgical procedures were investigated during halothane anaesthesia with spontaneous breathing through a modified T-piece (Mapleson F) with an apparatus deadspace that could be changed from 2 ml ($V_D$small) to 16 ml ($V_D$large). Immediately following the switch from $V_D$small to $V_D$large, $ETCO_2$ (mean ± SD) increased from 6.89 ± 1.09% to 7.61 ± 1.14% (ns) then gradually decreased during a 10-min period. The initial plateau of $PFCO_2$ (mean ± 1 SD) with $V_D$large was 0.74 ± 0.34%, but gradually decreased to 0.63 ± 0.25% after 10 min. This was achieved by an increase in $VE$ (P < 0.05 by 2 min). After 10 min $VE$ had increased by more than 40% (P < 0.01) as a result of an increase in $VT$ (mean ± 1 SD) of 14.6 ± 6.5 ml. After 10 min of $V_D$large ventilation, $VA$ and $VCO_2$ were maintained at $V_D$small values. The adequate ventilatory response to the large deadspace was seen in all patients, but the ventilatory efficiency, as judged by $V_D/VT$ and $VE/VCO_2$ ratios, was reduced significantly in the children weighing less than 10 kg.
Anaesthesia

Anaesthesia was induced with cyclopropane in oxygen ($F_{CO_2}$ 0.5). The trachea was intubated in all patients after the injection of suxamethonium 1–1.5 mg kg$^{-1}$ i.v. Ventilation was spontaneous throughout, and anaesthesia was maintained with nitrous oxide and 0.5–2% halothane in oxygen ($F_{CO_2}$ 0.5). In two patients undergoing the correction of hypospadias, a caudal block was established after induction using 0.25% bupivacaine 0.5 ml kg$^{-1}$.

The anaesthetic system was a modified T-piece (Mapleson F system) (fig. 1). To avoid rebreathing, the fresh gas flow was set according to the formula: $FGF = 3(1000 + 100)$ kg (Froese and Rose, 1982). The patient limb of the small deadspace system had a volume of 2 ml ($VD_{small}$) consisting of the airway adaptor to a Hewlett-Packard in-line capnograph (14360 A) and a modified Y-piece.

The patient limb of the large deadspace system had a volume of 16 ml ($VD_{large}$) as a result of the addition of a Fleisch No. 0 pneumotachograph and a T-piece connection. Minute ventilation ($VE$) was derived by electrical integration of the flow signal from a differential pressure manometer (Validyne MP 45-1-871, range ± 2 cm H$_2$O). Flow volume and carbon dioxide signals were recorded by a u.v. recorder (S.E. Labs (EMI) Ltd, S.E. 3006). Volume was calibrated with a 50-ml syringe, and the capnograph by using certified gas mixtures containing oxygen–nitrogen–carbon dioxide within the measuring range. All carbon dioxide values were corrected by a factor of 0.95 because of the collision broadening effect of nitrous oxide (Kennel, Andrews and Wollman, 1973). The resistance at 7 litre min$^{-1}$ was 3.5 cm H$_2$O litre$^{-1}$ s$^{-1}$ in the $VD_{small}$ system and 7.0 cm H$_2$O litre$^{-1}$ s$^{-1}$ in the $VD_{large}$ system.

Measurements

No measurements were made until at least 20 min after the induction of anaesthesia. During breathing through the $VD_{small}$ system, end-tidal carbon dioxide (ETC$_{O_2}$) was measured and a 3–5-min collection of expired gas made through a dry gas meter (AB Nordgas, Stockholm, Sweden) into a Douglas bag for determination of expired carbon dioxide fraction ($FE_{CO_2}$). The fresh gas flow (FGF) was then redirected through the $VD_{large}$ system. Tidal volume ($VT$), respiratory rate ($f$), ETC$_{O_2}$, and inspired carbon dioxide concentration measured from the plateau phase (plateau $F_{CO_2}$) (fig. 3), were measured immediately (i.e. breaths 5–10, time 0) and then every minute for up to 5 min, following which a timed collection (3–5 min) of expiratory gas was obtained. FGF was then redirected to the $VD_{small}$ and ETC$_{O_2}$ recorded. After 5 min a further timed collection of expiratory gas was obtained.

Calculations

$VE$, $VA$, $VT$, deadspace volume ($VD$), deadspace ventilation ($VD$) and carbon dioxide output ($VCO_2$) are presented at body temperature and pressure saturated (BTPS).

The following formulae were used:

\[
\begin{align*}
VCO_2 (\text{ml min}^{-1}) &= \frac{\text{gas collection } \dot{V}E \cdot F_{CO_2}}{100} \\
VA (\text{ml min}^{-1}) &= \frac{\dot{V}CO_2 \cdot 100}{ETCO_2} \\
VD (\text{ml min}^{-1}) &= \dot{V}E - \dot{V}A \\
VD (\text{ml}) &= \frac{\dot{V}D}{f}
\end{align*}
\]
Statistics

Mean values and standard deviation (SD) were calculated. The results were evaluated by the use of paired Student’s t test.

RESULTS

During measurements, using the small and large deadspaces, mean values (± 1 SD) of systolic and diastolic arterial pressure and heart rate were virtually unchanged (table 1).

### TABLE I. Mean values (± SEM) in all 12 patients for arterial pressure (AP) and heart rate (HR) with small (VDsmall) and large (VDlarge) deadspace ventilation

<table>
<thead>
<tr>
<th>VDsmall</th>
<th>VDlarge</th>
</tr>
</thead>
<tbody>
<tr>
<td>AP (mm Hg)</td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>98±4</td>
</tr>
<tr>
<td>Diastolic</td>
<td>50±2</td>
</tr>
<tr>
<td>HR (beat min⁻¹)</td>
<td>143±10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>VDsmall</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>AP (mm Hg)</td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>95±5</td>
</tr>
<tr>
<td>Diastolic</td>
<td>55±6</td>
</tr>
<tr>
<td>HR (beat min⁻¹)</td>
<td>139±11</td>
</tr>
</tbody>
</table>

End-tidal and inspired CO₂ concentration

With the small deadspace, the mean value (± 1 SD) of ETCO₂ concentration was 6.89 ± 1.09%. Immediately on changing to the large deadspace, ETCO₂ increased to a mean value (± 1 SD) of 7.61 ± 1.14%. During the period of observation, ETCO₂ gradually decreased to a mean value of 7.0 ± 1.30% after 10 min (fig. 2). One minute after changing to the small deadspace again, ETCO₂ decreased to a mean (± 1 SD) of 6.50 ± 1.34% (fig. 2) and continued to decrease over 5 min to 6.33 ± 1.36%. At 10 min ETCO₂ had stabilized at a mean value (± 1 SD) of 6.50 ± 1.34%.

Mean value (± 1 SD) of inspired carbon dioxide plateau (plateau \(F_{CO2}\)) was also greatest (0.74 ± 0.34%) immediately after changing to the large deadspace. It decreased to 0.65 ± 0.23% at 5 min and was 0.63 ± 0.25% after 10 min (fig. 2). The mean values (± 1 SD) of the end-tidal and inspired carbon dioxide concentrations and a typical carbon dioxide curve, are demonstrated in figure 3.

Minute ventilation, tidal volume and respiratory rate

Within a few breaths of large deadspace ventilation, minute ventilation increased. Measurements showed, for the first 5–10 breaths, a minute ventilation (mean ± 1 SD) of 1594 ± 430 ml min⁻¹, at 1 min 1900 ± 536 ml min⁻¹ (ns) and at 2 min 2068 ± 520 ml min⁻¹ (P < 0.05) (fig. 4). After 10 min, VT had increased by more than 40% to 2303 ± 792 ml min⁻¹ (P < 0.01). Respiratory rates were unchanged throughout the 10 min of VDlarge ventilation and the ventilatory response was the result of larger tidal volumes (fig. 4). During the first 5–10 breaths, VT was 44 ± 22 ml and increased by 16% to 51 ± 23 ml (ns) 1 min later. After 2 min, VT increased further, to 55 ± 24 ml (ns), and after 10 min it was 59 ± 26 ml (ns). The overall mean increase of VT (± 1 SD) was 14.6 ± 6.5 ml.

Alveolar ventilation and carbon dioxide elimination

During VDsmall ventilation, the alveolar ventilation (mean ± 1 SD) was 929 ± 343 ml min⁻¹. After 10 min of large deadspace ventilation, it was slightly increased to 984 ± 465 ml min⁻¹. It increased to 1087 ± 447 ml min⁻¹ when VDlarge was in use again. These differences were not statistically significant. Corresponding to the unaltered or slightly increased VA, carbon dioxide elimination was also virtually unchanged throughout.

Age variations

In the seven patients weighing less than 10 kg, the mean value (± 1 SD) of VT increased by 49% from 1540 ± 500 to 2296 ± 796 ml min⁻¹. In the remain-
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8.0-
7.0-
6.0-
5.0-
4.0-
3.0-
2.0-
1.0-
1 0 1 2 3 4 5 6 7 8
Duration of large deadspace ventilation (min)

fig. 2. Mean values ± 1 SD of end tidal carbon dioxide concentration (open columns) and of inspired carbon dioxide concentration (plateau FlCO2) (filled columns) before (-1), during 10 min (0-10) and 1 min after large deadspace ventilation (+1).

Deadspace ventilation (VD) at VDlarge was, however, more than 250% higher in the younger patients, with a mean value (± 1 SD) of 199 ± 87 ml min⁻¹ kg⁻¹ below and 76 ± 35 ml min⁻¹ kg⁻¹ above 10 kg (P < 0.05) (fig. 6). This was also reflected by the VD/VT ratio, which was greater in the smaller patients (P < 0.05) (fig. 6). The inefficient ventilation in the younger patients was also demonstrated by the higher VE/ VCO₂ ratio (P < 0.05) (fig. 6) - a difference that was not seen in the VA/VCO₂ ratio.

DISCUSSION

It was not possible to design a system of low deadspace and still incorporate a pneumotachograph. Thus, minute ventilation and tidal volume could not be measured before and after large deadspace ventilation and VT calculated from breaths number 5-10 of VDlarge ventilation were used as a basis for the evaluation of the ventilatory response to increased deadspace. At a flow rate of 7 litre min⁻¹, which was the highest met with in this study, the resistance of the system was 3.5 cm H₂O litre⁻¹ s⁻¹ for VDsmall and 7.0 cm H₂O litre⁻¹ s⁻¹ for VDlarge. Related to the normal nasal airway resistance in neonates of about 18–20 cm H₂O litre⁻¹ s⁻¹, and to the total airway resistance of a 10-kg child, the difference in the system resistances was thought to be too small to affect the results. In a technical assessment of the paediatric and adult airway adaptor of the capnograph used in this study, it was observed that minute ventilation was increased when the airway resistance was decreased by changing from the paediatric to the adult adaptor (Lindahl, 1984). Furthermore, a subsequent study of the effect of changing the airway resistance in a comparable group of children has shown that an increase in resistance of this order of magnitude does not produce the response seen in this study (Lindahl and colleagues, in preparation).

In the present study, the influence of biological variation was minimized by comparing the responses of each child to changes in deadspace volume. No measurements were made until a clini-
cally stable level of anaesthesia and surgery had been reached. This was confirmed by the stability of arterial pressure and heart rate throughout the actual measurements. The reduction in inspired alveolar halothane concentrations caused by increased apparatus deadspace did not appear to be of clinical

FIG. 4. Mean values (+ 1 SD) of minute ventilation (VE, filled columns), tidal volume VT, unfilled columns) and respiratory rate (f, cross-hatched columns) during 10 min of large deadspace ventilation. *P < 0.05; **P < 0.01.

FIG. 5. VE and ET\textsubscript{CO\textsubscript{2}} (mean ± 1 SD) for children less than (open circles) and greater than (closed circles) 10 kg during small (VD\textsubscript{small}) and large (VD\textsubscript{large}) deadspace ventilation.
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In all children the ET\textsubscript{CO\textsubscript{2}} increased when the large deadspace was added. This change was almost instantaneous and so could not have a metabolic cause. Higher ET\textsubscript{CO\textsubscript{2}} concentrations could also be caused by the presence of carbon dioxide in inspired gas. Inspired carbon dioxide was characterized by an initially high concentration early in inspiration, which then rapidly declined to a well identified short plateau phase (fig. 3). This could have been explained by a low fresh gas flow and thus represent rebreathing, not only from the added deadspace, but also as a result of low FGF; but rebreathing caused by inadequate fresh gas flows occurs late in inspiration (Lindahl, Charlton and Hatch, 1985) and was not seen in any patient in the present series. Besides, the formula used for setting the FGF was found to give an adequate FGF (Lindahl, Charlton and Hatch, 1984). Hence, rebreathing as a result of a low fresh gas flow was unlikely. The presence of a short plateau phase may be a reflection of a gas-mixing interface between alveolar gas and fresh gas from the T-piece. Simultaneously with the increase in mean ET\textsubscript{CO\textsubscript{2}} of 0.72% immediately after the start of \textit{VD}\textsubscript{large} ventilation, the mean plateau of inspired carbon dioxide was 0.74%. This makes the value of the inspired carbon dioxide concentration a likely explanation for the increased ET\textsubscript{CO\textsubscript{2}} concentrations. During the period of \textit{VD}\textsubscript{large} ventilation, inspired plateau concentrations of carbon dioxide and ET\textsubscript{CO\textsubscript{2}} decreased gradually as a result of compensatory ventilatory mechanisms.

All patients increased their minute ventilation in response to the larger deadspace by increasing tidal volume; respiratory rate did not change. At 10 min, ET\textsubscript{CO\textsubscript{2}} had been restored to the values found during \textit{VD}\textsubscript{small} ventilation. Hypoxia is an unlikely explanation for this response since, although the inspired oxygen tension is reduced in early inspiration, the small size of the added deadspace in relation to the tidal volume, and the high $F_\text{O}_2$ (0.5), would have preserved a high alveolar oxygen tension. Furthermore, hypoxic drive is reduced during halothane anaesthesia (Hirshman et al., 1977). A more likely causal factor is carbon dioxide rebreathing. However, studies of carbon dioxide rebreathing during halothane anaesthesia in children produced by both low fresh gas flow (Byrick, 1980; Lindahl, Charlton and Hatch, 1985) and the addition of 2% and 4% carbon dioxide to an adequate FGF (Olsson and Lindahl, 1985) have failed to show the uniform response demonstrated in this study. The pattern of exposure to carbon dioxide differs in these three types of carbon dioxide rebreathing. In the large deadspace situation the first part of the inspired gas is almost identical to alveolar gas. An attractive but hypothetical explanation for the response to increased deadspace is that pulmonary reflexes, sensitive to oxygen or carbon dioxide tensions, are terminating inspiration at a higher volume because of the continued presence of alveolar gas tensions in the airways during the early part of inspiration. This

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**Fig. 6.** Mean values at \textit{VD}\textsubscript{large} (+ SD) of deadspace ventilation (\textit{VD}) related to body weight, and of the ratios \textit{VD}/VT, \textit{VE}/\textit{VCO\textsubscript{2}}, and \textit{VA}/\textit{VCO\textsubscript{2}} for children < 10 kg and > 10 kg. *P < 0.05.
would explain both the uniformity of response and the fact that the increase in mean tidal volume of 14.6 ml was almost equal to the extra deadspace added.

Although the ventilatory response to the large deadspace was adequate in the smaller patients, ventilatory efficiency was decreased significantly. The mean deadspace ventilation (ml min$^{-1}$ kg$^{-1}$) was more than 250% higher than in the children weighing more than 10 kg. VD/VT ratios were also higher in the smaller patients and the ventilatory efficiency expressed by the VE/VCO$_2$ ratio was significantly reduced (fig. 6). Thus, apparatus deadspace is of relatively greater importance in younger children since proportionately higher volumes of ventilation are required to eliminate carbon dioxide.

It was concluded that, when the apparatus deadspace was increased from 2 to 16 ml in spontaneously breathing children, the ventilatory response was adequate in all patients as far as minute ventilation, end-tidal carbon dioxide concentration, alveolar ventilation and carbon dioxide elimination were concerned. The ventilatory efficiency was, however, significantly worse in the patients weighing less than 10 kg, emphasizing the need to minimize apparatus deadspace during spontaneous breathing in children. Furthermore, the results suggest the liberal use of controlled ventilation during anaesthesia and surgery on these smaller children.

ACKNOWLEDGEMENTS

We thank the anaesthetists and surgeons at the Hospital for Sick Children, Great Ormond Street, London, for their cooperation with the study. S.G.E.L. was supported by grants from the Swedish Society of Medical Sciences, the Swedish Medical Research Council and by the Sven Jerring-Bo Vahlqvist grant.

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