CONGENITAL HEART MALFORMATIONS AND VENTILATORY EFFICIENCY IN CHILDREN

Effects of Lung Perfusion During Halothane Anaesthesia and Spontaneous Breathing

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Lung compliance is decreased in awake, spontaneously breathing children with congenital heart disease who present with increased pulmonary blood flow (Wallgren, Geubelle and Koch, 1960; Howlett, 1972) whereas minute ventilation, alveolar ventilation and the arterial-to-end tidal carbon dioxide difference are similar to the values obtained in children who have a normal cardiopulmonary function (Lees, Way and Ross, 1967). The opposite is true in awake children whose heart disease results in a decrease in pulmonary blood flow (Lees et al., 1968; Howlett, 1972).

Anaesthesia alters the mechanics of the lung (Westbrook et al., 1973; Gilmour, Burnham and Craig, 1976; Hedenstierna and Santesson, 1979), and gives rise to mismatching of ventilation-perfusion (West, Dollery and Naimark, 1964) which impairs gas exchange and could, conceivably, worsen already compromised lung function in children with congenital cardiac malformations. Since such children often need repeated anaesthetics for open-heart surgery and various minor surgical and investigational procedures, investigation of their gas exchange, during anaesthesia and spontaneous ventilation, is of interest.

PATIENTS AND METHODS

Tidal volume (VT), ventilation rate (f), durations of inspiration (Ti) and expiration (Te) and of the whole ventilatory cycle (Ttot), airway occlusion pressure (Paw) and timing during occlusions (T0), end-tidal carbon dioxide tension (PETCO2), mixed expired carbon dioxide fraction (FECO2), arterial carbon dioxide tension (Paco2), and airway (Paw) and oesophageal (Poe) pressures were measured in 18 spontaneously breathing infants and children during halothane anaesthesia before cardiac surgery. Minute ventilation (VE), carbon dioxide elimination (VECO2), alveolar ventilation (VA), deadspace minute ventilation (VD), VD/VT and VE/VECO2 ratios, dynamic compliance (Cdyn)
Patients

All the children had congenital cardiac malformations and were scheduled for surgical correction of their anomaly(s). They were divided into two groups according to physical examination, chest x-ray, findings on cardiac catheterization, echocardiography, haemoglobin (Hb) concentration and haematocrit (Hct). There were 10 children with a normal Hb concentration, Hct below 40% and signs of a left-to-right shunt resulting in pulmonary hyperperfusion (group LR). Their body weights ranged from 3.7 to 16 kg, and their ages from 2 months to almost 5 yr. In the other group, in which there was right-to-left intracardiac shunting, all the children (n = 8) had a high Hb concentration, a Hct above 43% and showed signs of pulmonary hypoperfusion (group RL). These children weighed between 3.4 and 12 kg and were aged between 1 day and almost 3 yr. Further patient data are presented in table I. Parents gave their informed verbal consent to the study which was approved by the Hospital Ethics Committee.

Anaesthesia

The children were fasted for at least 4 h and those younger than 1 yr arrived in theatre unpremedicated. Older children were premedicated with a combination of diazepam 0.5 mg kg\(^{-1}\), morphine 0.15 mg kg\(^{-1}\) and hyoscine 0.01 mg kg\(^{-1}\) given rectally 45-60 min before the induction of anaesthesia. Anaesthesia was induced with nitrous oxide and halothane in oxygen (\(F_{IO}\), 0.5) via an anaesthetic face mask. In seven patients (one in group LR, six in group RL) thiopentone 2-3 mg kg\(^{-1}\) i.v. was given in addition. After induction a cannula was inserted to a peripheral vein and atropine 0.02 mg kg\(^{-1}\) and suxamethonium 1-1.5 mg kg\(^{-1}\) were given.
were injected. Nasotracheal intubation was performed (cuffed tracheal tubes: Mallinkrodt). Once spontaneous ventilation had resumed, anaesthesia was maintained with nitrous oxide and 0.5–1 % halothane in oxygen ($F_{102} 0.5$). A Malseon D system was used and fresh gas flows set high enough to avoid rebreathing. Radial artery and central venous cannulae were inserted.

**Measuring apparatus**

An in-line infra-red capnograph (Hewlett-Packard, 14360 A), a pneumotachograph (Fleisch No. 00 below 8 kg, Fleisch No. 0 above 8 kg) and connections for an airway occluder were placed in the apparatus deadspace. Airway pressures were measured at the tracheal tube connection (fig. 1).

![Diagram](https://example.com/diagram.png)

**Fig. 1.** Schematic presentation of recorded variables. The change in volume ($\Delta V_T$) between points of zero flow was used for calculation of dynamic compliance and the flow ($\Delta V$) between midvolume points was chosen for the calculation of total pulmonary resistance. Typical tracings for carbon dioxide concentration, airway ($P_{aw}$) and oesophageal ($P_{oe}$) pressures are shown. One occlusion test is illustrated.

The deadspace of the system was 6 ml with the Fleisch No. 00 and 8 ml with Fleisch No. 0 (measured by water displacement). The inspiratory and expiratory resistances of the measuring apparatus were 1.6 kPa litre$^{-1}$ s$^{-1}$ for Fleisch No. 00 and 1.8 kPa litre$^{-1}$ s$^{-1}$ for Fleisch No. 0 at a flow rate of 8 litre min$^{-1}$. No measurements were made until the halothane concentration and the level of anaesthesia were considered to be stable and at least 25 min had elapsed since induction. The investigation delayed the start of surgery by approximately 15–20 min during which time, however, the bladder catheter and the venous cannulae were inserted, and routine clinical monitoring established. All measurements were performed before surgery during spontaneous ventilation—which is the normal practice for these patients at our institution until venous access has been secured and the trachea intubated.

$V_E$ was measured by electrical integration of the flow signal from the heated pneumotachograph and a differential pressure manometer (Validyne, MP 45-1-871, range ±2 cm H$_2$O). The Fleisch No. 00 was linear for flow rates up to 8 litre min$^{-1}$ and Fleisch No. 0 up to flow rates of 16 litre min$^{-1}$. The capnograph response time to a complete deflection was 0.05 s and a well identified end-tidal plateau was observed in all patients. $P_{aw}$ and $P_{oe}$ were measured by Druck pressure transducers (PDCR-75). For the measurements of oesophageal pressures fluid-filled catheters were used (Olsson and Lindahl, 1985). Flow, volume, pressures and $P_{E_{CO2}}$ were recorded on an ink-jet recorder (Siemens-Elema, EM 81). Flow and volume were calibrated with an accurate pump, the capnograph with a known carbon dioxide tension (corrections for nitrous oxide being made) and the pressure transducers at −2 kPa.

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 5 min) of a measured volume of gas could be made into a Douglas bag. The mean exhaled fraction of carbon dioxide ($F_{E_{CO2}}$) in the bag was then immediately measured by the capnograph. Arterial blood (0.5 ml) was sampled in heparinized syringes and analysed within 3 min (Radiometer, Copenhagen, Denmark). The blood-gas machine was calibrated daily.

After the collection of the gas and measurements of the indices of ventilation, occlusion tests during two to three breaths were performed. The airway occluder was controlled electrically by the
pneumotachograph so that occlusions occurred precisely when expiration switched to inspiration, that is at the functional residual capacity (FRC). The response time of the occluder was 0.03 s.

Calculations

\[ \dot{V}_E, \dot{V}_A, \dot{V}_T, \dot{V}_D, \text{ and } \dot{V}_{CO_2} \] are presented at body temperature and pressure saturated (BTPS). The following formulae were used:

\[ \dot{V}_{CO_2}(\text{ml min}^{-1}) = \text{gas collection } \dot{V}_E \times P_{ECO_2} \]

\[ \dot{V}_A(\text{ml min}^{-1}) = \frac{\dot{V}_{CO_2} \times P_B}{P_{ACO_2}} \]

\[ \dot{V}_{A\text{Bohr}}(\text{ml min}^{-1}) = \frac{\dot{V}_{CO_2} \times P_B}{P_{ECO_2}} \]

\[ \dot{V}_D = \dot{V}_E - \dot{V}_A; \dot{V}_{D\text{Bohr}} = \dot{V}_E - \dot{V}_{A\text{Bohr}} \]

\[ C_{dyn}(\text{ml kPa}^{-1}) = \frac{\dot{V}_T}{P_E - P_I} \]

\[ TPR (\text{kPa litre}^{-1} \text{ s}^{-1}) = \frac{P_E - P_I}{\dot{V}} \]

where \( P_E \) and \( P_I \) are the mean of eight transpulmonary pressures calculated during zero flow after inspiration (\( P_I \)) and expiration (\( P_E \)) for calculations of \( C_{dyn} \), and at mid-volume points during inspiration and expiration for calculations of total pulmonary resistance (TPR) (see also figure 1). \( P_B \) is atmospheric pressure in kPa. To use transpulmonary pressures for the calculation of \( C_{dyn} \) and TPR a ratio of 0.9:1.1 between oesophageal (\( P_{oe\text{ o}} \)) and airway pressure (\( P_{aw\text{ o}} \)) during airway occlusions was required. This was achieved in nine patients in group LR and in five patients in group RL. At occlusion the decreases in airway pressure during 0.1 s (\( \Delta P/\Delta t \)) of the fast and of the slow phases were also calculated.

Statistics

Mean values, standard deviation (SD) and standard error of the mean (SEM) were calculated. Mean values and SEM are presented. Paired and unpaired two-sided \( t \) tests were carried out. Probability values less than 0.05 were considered to indicate statistical significance.

RESULTS

Arterial pressures (mean arterial pressure (MAP): 64±4 mm Hg group LR; 57±5 mm Hg group RL), heart rates (143±5 beat min\(^{-1}\) group LR; 139±6 beat min\(^{-1}\) group RL) and halothane concentrations (0.73±0.06% group LR; 0.81±0.08% group RL) were similar in the two groups during the measurements of ventilation and gas exchange.

Pulmonary ventilation

Minute ventilation was 35% greater in children with oligaemic lungs (256±22 ml min\(^{-1}\) kg\(^{-1}\)) compared with those who had overperfused lungs (189±14 ml min\(^{-1}\) kg\(^{-1}\)) (\( P < 0.05 \)) (fig. 2). Rates of ventilation were similar in the two groups (fig. 2) and so were the \( T_I/T^{tot} \) ratios (0.44±0.01 in group LR and 0.43±0.01 in group RL). \( V_T \) was 4.5±0.3 ml kg\(^{-1}\) in group LR and 5.7±0.3 ml kg\(^{-1}\) in group RL (\( P < 0.05 \)) (fig. 2). \( P_{ACO_2} \) was lower
Pa,

**FIG. 3.** Mean values (+ SEM) of arterial oxygen (P_{\text{aO}_2}) and carbon dioxide (P_{\text{aCO}_2}) tensions and of pH in groups LR (shaded bars) and RL (open bars). *P < 0.05; **P < 0.001 for the differences between the two groups.

<table>
<thead>
<tr>
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<th>Left to right shunts (group LR)</th>
<th>Right to left shunts (group RL)</th>
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<tbody>
<tr>
<td>C_{\text{dyn}} (ml kPa^{-1} kg^{-1})</td>
<td>10.4 ± 0.8</td>
<td>8.1 ± 1.5</td>
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<tr>
<td>TPR (kPa litre^{-1} s^{-1})</td>
<td>3.9 ± 0.6</td>
<td>5.8 ± 1.4</td>
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5.4 ± 0.1 kPa) in group RL than in group LR (6.2 ± 0.3 kPa) (P < 0.05) (fig. 3). Base excess was similar in the two groups, while pH was greater in the children with pulmonary hypoperfusion (7.32 ± 0.02 v. 7.26 ± 0.01; P < 0.05). The P_{\text{aO}_2} values in the cyanotic group (RL) were lower than in the acyanotic group (LR) (P < 0.001) (fig. 3). The lowest P_{\text{aO}_2} measured in group RL was 5.4 kPa while no subnormal values were found in the other group. C_{\text{dyn}} and TPR were similar in the two groups (table II).

**Gas exchange**

Mean carbon dioxide elimination (\dot{V}_{\text{CO}_2}) was slightly but not significantly lower in children with hypoperfused lungs. \dot{V}_{\text{A}} was similar in the two groups (fig. 4, table IV). Physiological deadspace ventilation (\dot{V}_{\text{D}}) (ml min^{-1} kg^{-1}) was 91% greater in group RL (145 ± 21 ml min^{-1} kg^{-1}) than in

**FIG. 4.** Mean values (+ SEM) of alveolar ventilation (\dot{V}_{\text{A}}), deadspace ventilation (\dot{V}_{\text{D}}) and carbon dioxide elimination (\dot{V}_{\text{CO}_2}) in groups LR (shaded bars) and RL (open bars). **P < 0.01 for the difference between the two groups.
group LR (76 ± 12 ml min⁻¹ kg⁻¹) (P < 0.01) (fig. 4). $\frac{V_D}{V_T}$ was 0.39 ± 0.03 in group LR and 0.55 ± 0.05 in group RL (P < 0.01) (fig. 5) and corresponding values for $\frac{V_E}{V_{CO_2}}$ ratio were 28 ± 2 and 45 ± 7, respectively (P < 0.05) (fig. 5).

There was no difference between arterial (6.2 ± 0.3 kPa) and end-tidal (6.2 ± 0.2 kPa) carbon dioxide tensions in children with hyperperfused lungs, while those with hypoperfused lungs had a $P_{aCO_2}$ of 5.4 ± 0.1 kPa and a $P_E^{CO_2}$ of 4.5 ± 0.2 kPa—a difference of 0.9 kPa (P < 0.01).

In group LR alveolar ventilation ($V_A^{Bohr}$) and $V_D^{Bohr}/V_T$ ratio were similar to $V_A$ and $V_D/V_T$—that is, values calculated from $P_{aCO_2}$ (fig. 6). In group RL, however, the deviations between $V_A^{Bohr}$ and $V_A$ (P < 0.01) as well as between $V_D^{Bohr}/V_T$ and $V_D/V_T$ (P < 0.01) were greater and individual relations between these variables are presented in figure 6.

**Ventilatory drive**

The mean inspiratory flow ($V_T/T_I$) (ml kg⁻¹ s⁻¹) was less in group LR (7.1 ± 0.4 ml kg⁻¹ s⁻¹) than in group RL (9.8 ± 0.7 ml kg⁻¹ s⁻¹) (P < 0.01). Airway occlusion pressure curves were biphasic, with an initial fast phase followed by a slower one. $P_{aw}^{fast}$ was more pronounced in children with oligaemic than in those with overperfused lungs (P < 0.05) (fig. 7). The duration of the fast phase was similar in the two groups (table III). The maximal occluded airway pressure ($P_{aw}^{max}$) was more pronounced in group RL than in group LR (P < 0.05) (fig. 7) and the duration from the start of the occluded breath to the maximal occluded intra-airway pressure ($T^{max}_w$) was longer in group RL than in group LR (P < 0.05) (fig. 7) (table III). The $\Delta P/\Delta t$ ratio of the initial fast phase was...
Fig. 7. Airway occlusion pressure curves in the two groups drawn schematically from mean values of pressure and time. The pressure differences of $P_{aw}\text{~fast}$ and of $P_{aw}\text{~max}$ between the two groups are indicated as well as the differences in time to reach $P_{aw}\text{~max}$ in the two groups. *$P < 0.05$ for the differences between the groups.

Table III. Mean values (±SEM) of airway occlusion pressures after the initial fast phase ($P_{aw}\text{~fast}$) and the maximal occluded pressure ($P_{aw}\text{~max}$) and of corresponding times ($T_{aw}\text{~fast}$ and $T_{aw}\text{~max}$) as well as the duration of an occluded breath ($T_{aw\text{~tot}}$) and the $T_{aw\text{~max}}/T_{aw\text{~tot}}$ ratio. *$P < 0.05$ for the difference between groups LR and RL.

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<thead>
<tr>
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<th>$P_{aw\text{~fast}}$ (kPa)</th>
<th>$P_{aw\text{~max}}$ (kPa)</th>
<th>$T_{aw\text{~fast}}$ (s)</th>
<th>$T_{aw\text{~max}}$ (s)</th>
<th>$T_{aw\text{~tot}}$ (s)</th>
<th>$T_{aw\text{~max}}/T_{aw\text{~tot}}$</th>
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<tr>
<td>Group LR</td>
<td>1.0±0.1</td>
<td>1.5±0.1</td>
<td>0.24±0.02</td>
<td>0.56±0.04</td>
<td>1.38±0.09</td>
<td>0.40±0.01</td>
</tr>
<tr>
<td>Group RL</td>
<td>1.3±0.1</td>
<td>2.0±0.2</td>
<td>0.29±0.03</td>
<td>0.71±0.05</td>
<td>1.68±0.17</td>
<td>0.43±0.02</td>
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5.2±0.6 kPa s$^{-1}$ in group LR and 6.2±0.7 kPa s$^{-1}$ in group RL (ns) and of the slower phase 2.2±0.3 kPa s$^{-1}$ and 3.6±0.6 kPa s$^{-1}$ ($P < 0.05$), respectively (fig. 7). The $T_{aw\text{~max}}/T_{aw\text{~tot}}$ ratio was similar in the two groups (table III). In group LR the $T_{aw\text{~max}}/T_{aw\text{~tot}}$ ratio was lower than the $T_{t}/T_{tot}$ ratio at unoccluded breathing ($P < 0.01$) which, however, was not the case in group RL.

DISCUSSION

In this study, no measurements were performed until at least 25 min had elapsed after the induction of anaesthesia. Recently, Gallagher and Black (1985) showed that after 30 min the alveolar-to-inspired halothane ratio was greater than 0.7 in children with normal cardiopulmonary function. This was most probably also the case in the present study for children with over-perfused lungs, while a somewhat lower ratio might exist in children with hypoperfused lungs. Insertion of bladder catheters, venous cannulae and other preparatory preoperative non-surgical procedures were not thought to have any influence on the actual measurements obtained.

In the group of children with increased pulmonary blood flow as a result of a left-to-right shunt, cardiac failure was treated in three, but none had any signs of cardiac decompensation before anaesthesia and surgery. In those with a diminished pulmonary blood flow, patients number 11 and 18 (table I) had the smallest ($Pa_{CO_2} - Pe'_{CO_2}$) (fig. 6). Patient No. 11, who was investigated during his first day of life, was deeply
Table IV. Mean values (± 1SD) of \( \bar{V}_E \), \( V_T \) and \( \bar{V}_A \) from this study compared with those of spontaneously breathing healthy children anaesthetized with halothane (Olsson and Lindahl, 1986) and of awake children (Lees, Way and Ross, 1967; Lees et al., 1968). \( \bar{V}_A \) calculated from end-tidal carbon dioxide tensions (\( V_A^{\text{end-tidal}} \)). *P < 0.05 for the differences between group LR and RL; †P < 0.05 for the difference between data from Olsson and Lindahl (1986) and group LR in this study; \( \dagger\dagger\dagger \)P < 0.001 for the difference between data from Olsson and Lindahl (1986) and group RL in this study.

<table>
<thead>
<tr>
<th>Anaesthetized</th>
<th>This study</th>
<th>Olsson and Lindahl (1986)</th>
<th>Awake</th>
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<tr>
<td></td>
<td>Group LR</td>
<td>Group RL</td>
<td>Normal lung perfusion</td>
</tr>
<tr>
<td>( \bar{V}_E ) (ml min(^{-1}) kg(^{-1}))</td>
<td>189 ± 45*</td>
<td>256 ± 61</td>
<td>228 ± 28†</td>
</tr>
<tr>
<td>( V_T ) (ml kg(^{-1}))</td>
<td>4.5 ± 0.8*</td>
<td>5.7 ± 1.0</td>
<td>4.1 ± 0.7( \dagger\dagger\dagger )</td>
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<tr>
<td>( \bar{V}_A ) (ml min(^{-1}) kg(^{-1}))</td>
<td>113 ± 21</td>
<td>111 ± 27</td>
<td>115 ± 25§</td>
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Cyanotic as a result of pulmonary atresia and needed a continuous prostaglandin infusion to keep the ductus arteriosus open during preparation for surgery (Freed et al., 1981). Although his \( P_{A,O_2} \) was low and Hct was 70% (high, even at this early age) and he was cyanotic, \((P_{A,O_2} - P_{E'O_2})\) was only −0.4 kPa. This was unexpected and might be explained by the prostaglandin infusion which has a vasodilatory effect on pulmonary arteries (Lewis et al., 1981) and could have improved the ventilation–perfusion relationship. The other child in group RL (No. 18) with a small \((P_{A,O_2} - P_{E'O_2})\) had previously required a subclavian flap because of a preductal coarctation of the aorta, and pulmonary banding had been performed because of a large ventricular septal defect. About 1 month after the first operation, signs of pulmonary hypoperfusion developed and by the time debanding was performed, the patient was being treated for cardiac failure and was thought to have a slow pulmonary circulation. Since pulmonary congestion increases pulmonary blood volume, more blood comes to the alveoli, increasing the alveolar carbon dioxide tension and this may have contributed to the small \((P_{A,O_2} - P_{E'O_2})\) in this patient, in spite of low lung perfusion.

Children with oligoemic lungs had larger tidal volumes than children with overperfused lungs, while ventilatory rates were similar. Tidal volumes in group RL were also greater \((P < 0.001)\) than in children with normal cardiopulmonary function (Olsson and Lindahl, 1986) (table IV). Anaesthetized children with normal and increased lung perfusion have similar tidal volumes as awake children (Lees, Way and Ross, 1967; table IV). Furthermore, the significantly greater tidal volumes found in group RL, compared with group LR, agree with the findings of Lees and colleagues (1968) in awake patients (table IV).

To achieve adequate alveolar ventilation, the children with oligoemic lungs required larger minute ventilations compared with those who had overperfused lungs \((P < 0.05)\). Since \( \bar{V}_A \) was similar in the two groups, there must have been a much larger proportion of wasted ventilation in group RL (fig. 4)—that is, greater \( V_D/V_T \) ratios \((P < 0.01)\) (fig. 5). However, this seems to be contrary to the results obtained by Lees and colleagues (1968). They found that \( \bar{V}_E \) (mean ± 1SD) was 217 ± 26 ml min\(^{-1}\) kg\(^{-1}\) in awake children with hypoperfused lungs and 265 ± 71 ml min\(^{-1}\) kg\(^{-1}\) in children with hypoperfused lungs, that is 22% greater. Simultaneously, they found that the corresponding value of alveolar ventilation was 38% greater (table IV). Therefore, in awake children ventilation seemed to be more efficient in children with oligoemic lungs. However, the alveolar ventilation calculated by Lees and colleagues was based on end-tidal carbon dioxide tensions \( (\bar{V}_A^{\text{end-tidal}}) \) and, since their patients with hypoperfusion had a large \((P_{A,O_2} - P_{E'O_2})\) it
was an over estimate. Assuming that in patients with oligaemic lungs $V_A^{Bohr}$ exceeded $V_A$ by 22% as was the case in the present study, their value of 207 ml min$^{-1}$ kg$^{-1}$ becomes 170 ml min$^{-1}$ kg$^{-1}$, and brings their data into conformity with the findings in the present study.

Children with diminished pulmonary blood flow require an enhanced ventilatory drive to maintain carbon dioxide homeostasis. Since ventilatory rates and $Tl/T_{tot}$ ratios were similar in the two groups the increase in $Vt/Tl$ ratio in group RL was attributable to larger tidal volumes, indicating increased ventilatory motor activity. This was also supported by the greater airway occlusion pressures in group RL. During occlusion, the airway pressures in both the fast and slow phases were more negative in group LR. This indicates that both the neuronal output of the intended breath from the ventilatory motor centre, shown by the early phase (Cherniack et al., 1976), and the total number of nerve impulses to the ventilatory muscles, reflected by the slower phase and maximal occlusion pressure (Kay, 1979; Lindahl and Olsson, 1986), were increased.

It is concluded that adequate gas exchange was achieved during the short period of time (up to 45 min) that elapsed in this study. In children with cardiac anomalies which resulted in an increase in pulmonary blood flow gas exchange was efficient; tidal volumes were similar to those obtained in the normal child. Invasive and non-invasive calculations of gas exchange were similar. In children with diminished pulmonary blood flow, however, ventilation was inefficient with high $V_E/V_{CO_2}$ and $V_D/V_T$ ratios and increases in ventilatory drive. In these, invasive and non-invasive calculations of gas exchange differed greatly because of the large value of $(P_{aCO_2} - P_{E_{CO_2}})$.

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