DYNAMIC BEHAVIOUR OF THE CHEST AND OF THE ABDOMEN IN ANAESTHETIZED PIGLETS DURING RAPID INSUFFLATION

M. ARBORELIUS JR AND S. LINDAHL

SUMMARY

Three piglets were ventilated with a TVM-ventilator, which releases predetermined volumes of compressed gas into the airways. The inspiratory flow depends on the pressure and volume of the compressed gas and compliance, resistance and inertia of the lungs, thorax and abdomen of the subject. The time for half filling of the chest was less than that of the abdomen and the difference increased with high driving pressure (with high acceleration). The difference was thus most probably a result of greater inertia of the abdomen. Inertia also seemed to influence the emptying of the lungs. This was so slow that increase of FRC—air trapping—was seen at a ventilatory frequency of 30 b.p.m. The practical consequences of these findings are that the abdomen (diaphragm) will take less and less part in ventilation and that a phase lag between chest and abdominal breathing should become larger with increasing ventilatory frequency. FRC will increase in regions with expiratory obstruction and lung rupture may follow.

The partition of gas between various lung regions has been shown to be governed by airway resistance and compliance of the lung, rib cage and of the abdomen (Konno and Mead, 1967; Grimby, Hedenstierna and Löfström, 1975). A thorough discussion of these factors has been given by Sybrecht and others (1976) who, in contradiction to Robertson, Anthonisen and Ross (1969), concluded that neither airway resistance nor compliance could explain why relatively less gas went to diaphragmatic lung regions during rapid as compared with slow inhalations in the supine position. A difference in intrapleural pressure was considered to be the only remaining factor that could explain the phenomenon during spontaneous breathing. Such a pressure difference must be a result of phase lag between thoracic muscles and diaphragm. A phase lag between thoracic and abdominal breathing movements at increasing ventilatory frequency has also been noted recently by Faithfull, Jones and Jordan (1979).

The purpose of this study was to observe time constants for the movement of chest and abdomen using rapid inflation of gas in animals in which complete neuromuscular block had been induced.

MATERIALS AND METHODS

Three piglets of the Swedish native breed were investigated in 12 experiments. Piglet I (weight 12 kg) took part in experiments 1, 2, 7 and 8, piglet II (7 kg) in experiments 3, 4, 9 and 10 and piglet III (8 kg) in experiments 5, 6, 11 and 12 (table I).

A combination of droperidol (Dridol) 1-2 mg kg\(^{-1}\) and ketamine (Ketalar) 30-50 mg kg\(^{-1}\) injected i.m. was used for anaesthesia. Alcuronium (Alloferin) 1-2 mg was given i.v. when necessary for the maintenance of neuromuscular block.

The piglets were studied in the supine position and the trachea intubated with a cuffed orotracheal tube (Portex no. 5), connected to a Tidal Volume Monitoring ventilator (TVM-ventilator) (Lindahl and Okmian, 1979) and the lungs ventilated with air. The TVM-ventilator (fig. 1A) was used for artificial ventilation, as it allows independent changes in driving pressure, tidal volume and ventilatory frequency. The ventilator consists of rigid chambers in which the breathing gas is compressed. When opening the inspiratory valve, air flows into the airways and lungs until a pressure equilibrium between the chamber and the airways and lungs is reached. The pressure difference \(P_i - P_s\) (fig. 1b) is adiabatic and proportional to a certain amount of gas, which depends upon the volume of the pressure chamber. In this apparatus the pressure chambers had volumes rendering compliances \(C_{vent}\) ml kPa\(^{-1}\) of 20, 40 and 80 ml kPa\(^{-1}\). The tidal volume \((V_T\) ml\) is accurately...
determined breath-by-breath (Lindahl, 1977). With this type of ventilator the ventilatory frequency can be varied without any change in driving pressure during inspiration ($P_{vent}$), end-inspiratory tracheal pressure ($P_{trachea}$) and magnitude of tidal volume. This principle offers several advantages which cannot be achieved with earlier known ventilators and was a prerequisite for this investigation.

Different ventilatory settings were used (table I) and an unchanged alveolar ventilation ($PA_{CO_2}$) was controlled by an end-tidal sampler synchronized with the ventilator’s working cycle and connected to an infra-red carbon dioxide analyser (Godart, Holland).

Frequent arterial samples were withdrawn and blood-gases analysed by conventional methods (Instrumentation Laboratories 213, Boston, USA). The correlation between end-tidal $P_{CO_2}$ and $PA_{CO_2}$ was satisfactory ($r = 0.80$) in these piglets with healthy lungs. No significant changes in $PA_{O_2}$ occurred.

Tracheal pressures and pressures in the ventilator’s pressure chamber were measured with electromanometers (Siemens–Elema, EMT 34).

Two mercury silastic tube strain gauges were used, one in the middle of the sternum and the other 5 cm distal to the xiphoid process. The generated resistance changes from these two strain gauges were recorded with the tracheal and ventilator pressures on an ink-jet recorder (Siemens–Elema, EMT 81). Changes in resistance were proportional to the volume changes in the chest and in the abdomen and were linear within the measuring range. The volume changes were expressed as a percentage of the end-inspiratory volume, since absolute volume calibration for each strain gauge was both difficult to achieve and unnecessary for the purpose of the study. Five to 10 deflections were recorded, but no measurable differences occurred between the cycles.

The time needed to reach a terminated deflection of insufflation ($t_{1,0}$) from each strain gauge was measured. In the same way the time needed to reach half this amplitude ($t_{0,5}$) was measured. In all but experiment no. 7, end-inspiratory plateaux were reached, that is no change in volume or pressure was recorded. The insufflation time ($t_{insuff}$) and the time needed to reach static end-inspiratory pressure in the ventilator pressure chamber ($t_{vent}$) were measured also (fig. 1).

All records were obtained 15 min after the setting of an adequate ventilation.
TABLE I. \( C_{\text{vent}} = \text{ventilator volume/pressure quotient}; f = \text{ventilatory frequency}; P_{\text{vent}} = \text{pre-inspiratory pressure in the ventilator pressure chamber}; P_{\text{peak}} = \text{greatest tracheal pressure}; P_{\text{el}} = \text{end-inspiratory tracheal pressure}; V_T = \text{tidal volume}; t_{\text{vent}} = \text{time needed for a terminated pressure change in the pressure chamber}; t_{\text{insuff}} = \text{the time needed for reaching end-expiratory pressure in the trachea}; t_{0.5} \text{ and } t_{1.0} = \text{time for 50% and 100% filling of chest and abdomen during inspiration.}\)

<table>
<thead>
<tr>
<th>Piglet</th>
<th>Expt</th>
<th>( C_{\text{vent}} ) (ml kPa(^{-1}))</th>
<th>( f ) (s)</th>
<th>( P_{\text{vent}} ) (kPa)</th>
<th>( P_{\text{peak}} ) (kPa)</th>
<th>( P_{\text{el}} ) (kPa)</th>
<th>( V_T ) (ml)</th>
<th>( t_{\text{vent}} ) (s)</th>
<th>( t_{\text{insuff}} ) (s)</th>
<th>( t_{0.5}(s) )</th>
<th>( t_{1.0}(s) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1</td>
<td>40</td>
<td>10</td>
<td>1.2</td>
<td>8.7</td>
<td>3.2</td>
<td>2.2</td>
<td>260</td>
<td>0.36</td>
<td>0.42</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>40</td>
<td>30</td>
<td>1.0</td>
<td>4.7</td>
<td>1.9</td>
<td>1.2</td>
<td>140</td>
<td>0.32</td>
<td>0.32</td>
<td>0.04</td>
</tr>
<tr>
<td>II</td>
<td>3</td>
<td>40</td>
<td>10</td>
<td>1.2</td>
<td>6.1</td>
<td>3.4</td>
<td>2.1</td>
<td>160</td>
<td>0.41</td>
<td>0.46</td>
<td>0.10</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>40</td>
<td>30</td>
<td>1.0</td>
<td>2.9</td>
<td>2.1</td>
<td>0.9</td>
<td>80</td>
<td>0.31</td>
<td>0.35</td>
<td>0.06</td>
</tr>
<tr>
<td>III</td>
<td>5</td>
<td>40</td>
<td>20</td>
<td>0.3</td>
<td>2.5</td>
<td>1.6</td>
<td>0.7</td>
<td>70</td>
<td>0.30</td>
<td>0.36</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>40</td>
<td>20</td>
<td>0.3</td>
<td>6.6</td>
<td>2.2</td>
<td>2.1</td>
<td>180</td>
<td>0.30</td>
<td>0.30</td>
<td>0.07</td>
</tr>
<tr>
<td>I</td>
<td>7</td>
<td>40</td>
<td>20</td>
<td>1.0</td>
<td>6.3</td>
<td>2.6</td>
<td>1.8</td>
<td>180</td>
<td>0.38</td>
<td>0.40</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>40</td>
<td>20</td>
<td>1.0</td>
<td>6.7</td>
<td>4.0</td>
<td>1.2</td>
<td>110</td>
<td>0.31</td>
<td>0.38</td>
<td>0.04</td>
</tr>
<tr>
<td>II</td>
<td>9</td>
<td>80</td>
<td>20</td>
<td>1.0</td>
<td>2.6</td>
<td>2.1</td>
<td>1.2</td>
<td>110</td>
<td>0.45</td>
<td>0.50</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>80</td>
<td>20</td>
<td>1.0</td>
<td>7.0</td>
<td>3.0</td>
<td>1.0</td>
<td>120</td>
<td>0.26</td>
<td>0.31</td>
<td>0.03</td>
</tr>
<tr>
<td>III</td>
<td>11</td>
<td>80</td>
<td>20</td>
<td>1.0</td>
<td>2.5</td>
<td>1.6</td>
<td>1.0</td>
<td>120</td>
<td>0.35</td>
<td>0.41</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>80</td>
<td>20</td>
<td>1.0</td>
<td>7.0</td>
<td>3.0</td>
<td>1.0</td>
<td>120</td>
<td>0.26</td>
<td>0.31</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Current statistical methods for mean values and SD were used. Paired significance tests (Student's \( t \) test) were applied.

RESULTS

The results are presented in table I. With the exception of experiments 6 and 12, \( t_{0.5} \) was shorter for the chest than for the abdomen (fig. 2). Experiments 6 and 12 were characterized by a low driving pressure \( (P_{\text{vent}}) \) and relatively small tidal volumes (low accelerative forces during inspiration). The difference in \( t_{0.5} \) for the chest v. the abdomen was statistically significant \( (P<0.001) \).

![Fig. 2](image2.png)

**Fig. 2.** Tracings from experiment 1. \( P_{\text{vent}} \) and \( P_{\text{el}} \) and volume changes of the chest and of the abdomen. Note time differences in both inspiratory and expiratory volume changes of the chest and of the abdomen.

![Fig. 3](image3.png)

**Fig. 3.** Tracings from experiment 7. The same plan as figure 2. Note that the inspiration is terminated at the same time in both chest and abdomen. Expiratory volume changes are identical during the first part of expiration.

![Fig. 4](image4.png)

**Fig. 4.** Tracings from experiment 11. The same plan as figures 2 and 3. Note the correspondence between volume changes of the chest and of the abdomen when high initial driving pressures were used but the delivered volume was small. \( t_{0.5} \) was, however, only 50% of \( t_{0.5} \) even if both were so brief that the visual difference was small.
The differences appeared to be greatest in experiments with high driving pressures (high accelerative forces during inspiration (nos 1, 9 and 11)). In most measurements $t_{1.0}$ was less for the chest than for the abdomen ($P<0.01$). An exception was in experiment 7, where inspiration was interrupted before the end-inspiratory volume was reached (fig. 3). A high driving pressure and moderate tidal volumes (9 and 11) resulted in an equal $t_{1.0}$ (fig. 4).

It was found that the volume change of the chest structures ($t_{1.0}$ chest) followed the evacuation of the pressure chamber ($t_{vent}$) closely, while the volume change of the abdomen ($t_{1.0}$ abd) lagged considerably in most cases (fig. 5A, B; table I). This was most obvious in experiments with high driving pressure and large tidal volumes.

It was found also that the expiratory flow was mainly affected by distention of the lung and was more rapid in early expiration at larger tidal volumes (i.e. directly proportional to the distending pressure). Furthermore, it was faster for the chest than for the abdomen (fig. 2). This difference seemed to be less pronounced when the structures were not expanded to their normal compliance at the commencement of exhalation (fig. 3). It was possible to demonstrate a substantial increase in the end-expiratory volume (air-trapping) (fig. 6A, B) even in these healthy piglets when expiration time was too short ($f = 30$ b.p.m. and 50% expiration time).

**DISCUSSION**

Sybrecht and others (1976) found that the basal parts of the lungs received less of an inhaled gas bolus with increasing speed of voluntary inhalation in supine man. They showed that different resistances in bronchi to apical and diaphragmatic areas could not explain their results. Faithfull, Jones and Jordan (1979) showed that a phase lag between thoracic and abdominal breathing movements occurred with increasing frequency of breathing as registered with strain gauges over the thorax and abdomen. The results of both these studies might be explained by slower or weaker muscle response of the diaphragm/abdomen compared with the intercostals.

In the present experiments all muscles were relaxed. Nevertheless, the chest filled more rapidly than the abdomen, a difference that was more pronounced with rapid insufflation. According to the calculations of Sybrecht and others (1976), different resistances in airways to areas of lung associated with movement of the diaphragm/abdomen and the chest is an unlikely explanation of the phenomenon. Furthermore, the thoracic volume change in our study was recorded over the middle part of the sternum. Therefore airways leading to lung regions expanding ventrally (chest) and caudally (abdomen) must, to some extent, be the same, which make flow-dependent differences in airway resistance an improbable explanation for the differences in time constants. The only remaining cause for the different pattern of movement of chest and abdomen during muscle relaxation is differences in inertia.

If the chest of the piglets is considered as a cylinder with a diameter and length of 10 cm and if it should increase its volume by 200 ml, there would be a change in diameter of about 0.6 cm. If the diaphragm, considered as a piston, should perform the same volume change it must move 2.6 cm. The acceleration of the diaphragm would thus be approximately five times greater than that of the chest wall. The chest wall moves in air, while the diaphragm must
displace water. The high inertia of the diaphragm/abdomen will counteract acceleration during the first phase of inflation and retardation during the later phase. This explains the difference in movements of the chest and abdomen shown in figure 3 and causes a small redistribution of gas at the end of inspiration, demonstrated in figure 5A and B. High inertia of the abdomen in relation to the chest, acting both during inhalation and exhalation, could easily explain the increasing phase shift at greater breathing frequencies found by Faithfull, Jones and Jordan (1979).

In our healthy piglets, using normal sizes of tracheal tube and low expiratory resistance of the circuit, the time needed for a complete expiration was so long that the normal end-expiratory level was not reached at a ventilatory frequency of 30 b.p.m. with an expiration time 50% of the whole breathing cycle. During the first ventilatory cycles FRC increased by more than 30 ml (fig. 6A, B) with a tidal volume of 140 ml. With a tidal volume of 260 ml at a ventilatory frequency of 10 b.p.m. and an expiration time of 80% of the breathing cycle FRC did not change. In obstructive lung disease expiratory time constants increase more than inspiratory time constants. High inspiratory pressures maintained throughout the inspiration will cause hyperinflation of lung regions with long expiratory time constants (Lindahl, Arborelius and Okmian, 1979) eventually leading to lung rupture (Kirkpatrick, Felman and Eitzman, 1974; Berg et al., 1975; Ogata et al., 1976) unless long expiration times are used.

With increasing ventilatory frequency the diaphragmatic parts of the lung will take a decreasing part in ventilation and with very high ventilatory frequencies (Jonzon et al., 1971) mainly chest wall ventilation is to be expected.

ACKNOWLEDGEMENTS
This investigation was supported by grants from the Swedish Medical Research Council, Project no. B76-17X-4519-02.

REFERENCES
La pratique de ces découvertes est que l'abdomen (diaphragme) peut prendre de moins en moins part à la ventilation et que le déphasage entre la respiration de la poitrine et la respiration abdominale deviendra plus important avec l'augmentation de la fréquence ventilatoire. La FRC augmentera dans les régions où il y a une obstruction respiratoire et il peut s'ensuivre une rupture des poumons.

**DYNAMISCHES VERHALTEN VON BRUTS UND UNTERLEIB BEI NARKOTISIERTEN FERKELN WAHRENDE SCHNELLER INSUFLATION**

ZUSAMMENFASSUNG

Drei Ferkel wurden mit einem TVM-Ventilator belüftet, der vorbestimmte Mengen komprimierten Gases in die Luftwege leitete. Der Einatmungsweg hing von Druck und Volumen des komprimierten Gases, sowie von Dehnbarkeit, Widerstand und Trägheit von Lunge, Brustkorb und Unterleib des Tieres ab. Die Zeit für die halbe Füllung des Brustraumes war geringer als für den Unterleib, und dieser Unterschied wurde größer bei höherem Antriebsdruck (bei mehr beschleunigter Füllung). Der Unterschied war also wahrscheinlich Resultat der größeren Trägheit des Unterleibes. Trägheit schien auch das Entleeren der Lungen zu bestimmen, das so langsam war, dass ein Anstieg von FRC—funktionelle Residualkapazität oder Luftreste—bei einer Belüftungsfrequenz von 30 b.p.m. zu sehen war. Die praktische Folge dieser Resultate ist, dass der Unterleib (Zwerchfell) weniger und weniger an der Belüftung teilnimmt, und dass der Phasenabstand zwischen Brustraum—und Unterleibsatmung bei steigender Belüftungsfrequenz größer wird. FRC wird in Regionen mit Ausatmungshindernissen ansteigen, was zu Lungenriss führen kann.

**COMPORTAMIENTO DINAMICO DEL PECHO Y DEL ABDOMEN DE PORCELETS ANESTHESIADOS DURANTE INSUFLACION RAPIDA**

SUMARIO

Mediante un ventilador-TVM, se ventiló a tres chanchitos al soltar volúmenes predeterminados de gases comprimidos en las vías respiratorias. El flujo inspiratorio depende de la presión y del volumen del gas comprimido y de la reacción, resistencia e inercia de los pulmones, del tórax y del abdomen del sujeto. La duración para llenar a medias el pecho fue inferior a la del abdomen y la diferencia aumentó cuando la presión de compresión era alta (con alta aceleración). La diferencia fue por consiguiente el resultado de una mayor inercia del abdomen probablemente. La inercia pareció influir también en el vaciamiento de los pulmones. Fue tan lento que ocurrió un aumento del FRC—retención del aire—in una frecuencia de ventilación de 30 b.p.m. La consecuencia práctica de estos resultados indican que el abdomen (diaphragma) participa cada vez menos en la ventilación y que una brecha de la fase entre la respiración torácica y abdominal se volverá mayor con una creciente frecuencia de ventilación. El FRC aumentará en áreas con obstrucción inspiratoria y puede acarrear una ruptura de los pulmones.