Effects of Constant Chest Compression on the Mechanical and Physiologic Performance of Different Ventilators

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WITH THE TECHNICAL ASSISTANCE OF GEORGE T. MCCOY AND MIRIAM STUCKER

A mechanical and physiologic evaluation of five commercially available ventilators has been carried out in dogs. This has been accomplished by studying the response of the ventilator to the challenge of decreased total compliance produced by constant chest compression over a test period.

The mechanical performance of the ventilators followed closely the responses studied previously in a model lung. The physiologic changes, and the ability of the various ventilators to preserve pre-compression status was distinctly superior in the volume-controlled ventilators, the Emerson and Engstrom giving the best results. In all animals constant compression of the chest resulted in a decreased P02, which could not be restored to normal. The pressure limited-ventilator was the least efficient.

Previous studies of ventilator performance have been concerned with a mechanical analysis of the ventilator response to changes in resistance and compliance imposed within a model lung. While this will provide insight and information regarding the ability of the ventilator to meet a challenge under constant, controlled conditions, it cannot reveal the dynamic changes in volume, inflating pressure, peak inspiratory flow as well as the circulatory and blood-gas changes which occur in the lungs themselves.

Accordingly we have undertaken to compare five ventilators in a simulated clinical situation in dogs, in which we have measured mechanical performance. In addition we have determined changes in many of the pertinent physiologic parameters of circulation and respiration which must ultimately serve as the final criteria for ventilator performance.

Methods

Data were collected from experiments performed on 17 mongrel dogs weighing 17 to 28 kg. All dogs were anesthetized with a single dose of pentobarbital sodium (25 mg/kg). Muscle paralysis was initiated and maintained with intermittent injections of succinylcholine chloride (20 to 60 mg). The lungs were ventilated through a cuffed endotracheal tube, with the ventilators listed below. The ventilators were adjusted to maintain the end-tidal CO2 tension close to 40 mm. of mercury (range 37–45 mm. of mercury) during the control period, for all experiments. Pure oxygen or an oxygen-air mixture was used for the inspired gas. An ordinary blood pressure cuff (15 cm wide) was placed around the chest of the dog, and connected to a large plastic bottle which served as a pressure reservoir (fig. 1). Respiratory gas flow was measured between the endotracheal tube and the ventilator by means of a heated pneumotachograph (Fleisch, type 1) and a differential pressure transducer (Statham, P5-0.2D-380). Pressure in the airway at mouth level was transduced by a strain gauge (Statham, P23-4D-300). End-tidal and mean expired CO2 concentrations were measured by means of an infrared CO2 analyzer (Beckman, LB-1 with an LB-1 Linearizer), being sampled at a flow (constant
for each study) between 300 and 500 ml./minute through a catheter extending to the distal end of the endotracheal tube.

One polyethylene catheter was placed into the inferior vena cava below the diaphragm (via the left femoral vein) and another in the right atrium through the right jugular vein. These were connected to a low pressure strain gauge (Statham, P23BB) through two three-way stopcocks in line. This permitted alternate measurement of peripheral venous or right atrial pressure. A right femoral arteriovenous plastic shunt was inserted for continuous measurement of blood pH, as described previously. Mean arterial blood pressure was monitored by means of a plastic catheter placed in the left femoral artery connected to a mercury manometer. During the collection of expired gas, arterial blood samples were obtained through this catheter. Flow rate, airway pressure, venous or right atrial pressure, CO₂ concentration near the carina, and arterial pH were continuously recorded on a Sanborn Model 7720 multi-channel recorder using 350-type preamplifiers.

Expired gas was collected into a spirometer (Collins, 9-liter) without a CO₂ canister, dead space having been replaced by a waterproof paper block and the bellows counter-balanced to provide minimum resistance. Thus the spirometer served both as a collecting chamber for expired gas and as a device for measuring tidal and minute volumes. After flushing the spirometer with the expired gas at least twice, expired gas was collected for 1.0–1.5 minutes, the volume measured, and CO₂ concentration determined.

Arterial blood samples were collected in heparinized 10 ml. and 2 ml. syringes. pH, PₐO₂, and standard bicarbonate were determined immediately. The second syringe was capped and placed immediately in iced saline until PₐO₂ was measured 15 to 20 minutes later.

PₐO₂ was measured with PₐO₂ electrode (Radiometer, type E5044) calibrated with analyzed nitrogen and nitrogen-oxygen gas mixtures. Blood pH was measured with a micro-pH electrode (type E5021) and a pH-meter (Radiometer type TTT-1). PₐCO₂ was measured with a Severinghaus type CO₂-elec-
trode (Radiometer, type E5031) or determined by the method of Astrup.6 All blood 
PO2 values were converted for the temperature difference between samples and the elec-
trode.4

Our experience with dogs lying on their backs has indicated that certain of the basic 
parameters under study (i.e., PaO2) will become unstable after two hours of constant 
ventilation. Therefore, no more than two ventilators were compared in any dog.

The study was divided into three test periods. After the dog received constant ventilation 
for at least 30 minutes, arterial blood and expired gas samples were taken for control 
values (test period A). The cuff around the chest was then inflated to a pressure of 
40 mm. of mercury. The second and third samples were taken 7.5 and 15 minutes later 
(test period B). Ten minutes after the cuff was deflated a fourth sample of arterial blood 
and expired gas was obtained (test period C). Several deep inspirations and a 30-minute 
period of constant ventilation always preceded the test period.

Anatomic dead space (anat. VD) and physiologic dead space (phys. VD) were calculated 
from the data obtained, using Böhr’s equation. In the calculation of anatomic VD, errors are 
likely to be introduced in two ways: (1) by an error in the precision with which end-tidal 
CO2 approximates alveolar CO2; (2) by an absolute error in the infrared analysis of CO2 
concentration. The former was minimized by taking the mean of several end-tidal CO2 con-
centrations during the period of collection of expired gas. The variation in end-tidal CO2 
concentrations within one minute’s time was 0.15 per cent, which can cause an error of 3.3 
per cent in the calculation of anatomic dead space. The CO2 meter was calibrated using 
six CO2-in-O2 mixtures in the zero to 10 per cent range, resulting in an absolute error of 1 
mm. of mercury. Transferring this error to the Böhr equation results in a maximum abso-
late error of 7.8 per cent in calculating anatomic dead space.

Dead space of the apparatus was determined with water displacement. An appro-
priate correction for the compressed gas in the patient-circuit of the Engström and the 
Emerson Ventilators was made with the method described by Engström et al.8 This 
was not necessary for the other ventilators because the expiratory valve was very close to 
the endotracheal tube. During ventilation with pure oxygen, alveolar PO2 was calculated by 
subtracting the sum of water vapour pressure at body temperature and PaCO2 from the 
barometric pressure. An estimate of total compliance (lungs plus chest wall) was obtained 
from the ratio of tidal volume to peak airway pressure. All data was tested with Student’s 
t test and the 95 per cent confidence limit was accepted as the significant level of change. A 
97.5 per cent confidence limit was accepted as highly significant.

Table 1 describes the 5 ventilators examined and is based on our modification of the classi-
fication of them according to the method of

<table>
<thead>
<tr>
<th>Table 1. Classification of Ventilators Studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspiratory Phase</td>
</tr>
<tr>
<td>Bird (Mark-8) without Air-Mix</td>
</tr>
<tr>
<td>Bird (Mark-8) with Air-Mix</td>
</tr>
<tr>
<td>Ohio anesthesia respirator</td>
</tr>
<tr>
<td>Air-Shields respirator</td>
</tr>
<tr>
<td>Emerson postoperative ventilator</td>
</tr>
<tr>
<td>Engström respirator</td>
</tr>
</tbody>
</table>

* All of the ventilators opened to atmosphere during their expiratory phase.
Mushin et al. The last two columns in the table “Power Source” and “Power Source to Patient Coupling” have been added by us. Because the models of Emerson and Air-Shield ventilators tested have become available only recently, they will be described under “Results.” With the exception of the Bird Ventilator, all the ventilators are “volume controlled.” Only positive-pressure ventilation was employed, and expiration was passive.

Results

Mechanical Analysis of Ventilator Performance

The minute volume, respiratory rate, tidal volume, inspiratory flow time, peak inspiratory flow and airway pressure measured during each test period with the various ventilators, are listed in table 2. Total compliance, calculated from these data, is also listed in the table. During compression of the chest a decrease in total compliance was noted in all dogs and varied between 16 and 30 per cent according to the ventilator used. Ten minutes after removal of compression, the compliance rose, but failed to return to control values in any of the dogs.

Bird Mark-8 Respirator (Figure 2a and b): Use of this ventilator (with or without Air-Mix) during chest compression resulted in a highly significant decrease in tidal volume. The average decrease was 28 per cent. Since the Bird ventilator without Air-Mix is a pressure cycled, constant-flow generator, the decrease in compliance produced a reduction in inspiratory flow time, and tidal volume was consequently decreased. This reduction was statistically significant. A shortened inspiratory time resulted in an increased respiratory rate. The values of minute volume decreased because the effect of decreased tidal volume predominated over the effect of increased frequency. In the Bird with Air-Mix, a constant pressure generator, the decreased compliance produced a statistically, highly significant decrease peak inspiratory flow while the inspiratory flow time was only slightly reduced. In spite of the increased frequency, with and without Air-Mix, there was a statistically significant decrease in minute volume.

**Table 2. Ventilatory Parameters During the Three Test Periods**

<table>
<thead>
<tr>
<th>Test Period</th>
<th>Minute Volume (L/min)</th>
<th>Resp. Rate per Minute</th>
<th>Tidal Volume (ml/kg)</th>
<th>Insp. Flow Time (sec)</th>
<th>Peak Insp. Flow (l/min)</th>
<th>Airway Pressure (cm H2O)</th>
<th>Total Compliance (ml/cm H2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bird without Air-Mix (4 studies)</td>
<td>A 5.68 ± 0.55</td>
<td>20.0 ± 1.4</td>
<td>13.2 ± 1.1</td>
<td>1.20 ± 0.14</td>
<td>21.8 ± 3.2</td>
<td>7.8 ± 0.3</td>
<td>35.6 ± 4.1</td>
</tr>
<tr>
<td>B 4.51 ± 0.55</td>
<td>23.5 ± 1.8</td>
<td>9.4 ± 1.1</td>
<td>0.95 ± 0.12</td>
<td>19.5 ± 3.6</td>
<td>7.8 ± 0.3</td>
<td>25.5 ± 4.2</td>
<td></td>
</tr>
<tr>
<td>C 5.15 ± 0.66</td>
<td>20.8 ± 1.7</td>
<td>12.1 ± 0.8</td>
<td>1.05 ± 0.15</td>
<td>21.2 ± 3.5</td>
<td>7.8 ± 0.3</td>
<td>32.9 ± 6.3</td>
<td></td>
</tr>
<tr>
<td>Bird with Air-Mix (4 studies)</td>
<td>A 3.10 ± 0.41</td>
<td>13.8 ± 2.4</td>
<td>11.0 ± 0.8</td>
<td>0.87 ± 0.02</td>
<td>28.3 ± 5.0</td>
<td>8.0 ± 0.3</td>
<td>30.0 ± 3.3</td>
</tr>
<tr>
<td>B 3.32 ± 0.40</td>
<td>14.9 ± 2.8</td>
<td>11.0 ± 1.1</td>
<td>0.75 ± 0.03</td>
<td>20.9 ± 1.4</td>
<td>8.0 ± 0.3</td>
<td>21.1 ± 3.0</td>
<td></td>
</tr>
<tr>
<td>C 2.96 ± 0.41</td>
<td>13.8 ± 2.4</td>
<td>13.9 ± 0.6</td>
<td>0.85 ± 0.02</td>
<td>24.7 ± 1.3</td>
<td>8.0 ± 0.3</td>
<td>27.0 ± 3.1</td>
<td></td>
</tr>
<tr>
<td>Ohio (5 studies)</td>
<td>A 2.46 ± 0.28</td>
<td>13.0 ± 1.9</td>
<td>12.7 ± 0.8</td>
<td>0.60 ± 0.06</td>
<td>55.6 ± 2.0</td>
<td>11.0 ± 0.5</td>
<td>28.0 ± 2.1</td>
</tr>
<tr>
<td>B 2.05 ± 0.36</td>
<td>12.0 ± 1.9</td>
<td>11.5 ± 0.7</td>
<td>0.62 ± 0.07</td>
<td>50.2 ± 1.4</td>
<td>13.0 ± 0.5</td>
<td>22.6 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>C 2.35 ± 0.33</td>
<td>12.4 ± 1.9</td>
<td>12.3 ± 0.8</td>
<td>0.60 ± 0.06</td>
<td>54.4 ± 2.1</td>
<td>11.5 ± 0.6</td>
<td>28.0 ± 2.0</td>
<td></td>
</tr>
<tr>
<td>Air-Shields (4 studies)</td>
<td>A 4.32 ± 0.16</td>
<td>14.2 ± 1.3</td>
<td>21.5 ± 1.6</td>
<td>0.80 ± 0.02</td>
<td>34.9 ± 4.6</td>
<td>12.1 ± 1.1</td>
<td>26.5 ± 2.8</td>
</tr>
<tr>
<td>B 4.05 ± 0.16</td>
<td>14.2 ± 1.3</td>
<td>20.4 ± 1.6</td>
<td>0.80 ± 0.02</td>
<td>30.6 ± 4.8</td>
<td>15.8 ± 1.0</td>
<td>21.6 ± 2.1</td>
<td></td>
</tr>
<tr>
<td>C 4.22 ± 0.17</td>
<td>14.2 ± 1.3</td>
<td>21.0 ± 1.6</td>
<td>0.80 ± 0.02</td>
<td>33.5 ± 5.2</td>
<td>12.6 ± 1.3</td>
<td>24.4 ± 2.2</td>
<td></td>
</tr>
<tr>
<td>Emerson (7 studies)</td>
<td>A 4.54 ± 0.61</td>
<td>16.6 ± 1.6</td>
<td>10.7 ± 0.9</td>
<td>1.40 ± 0.13</td>
<td>22.5 ± 2.8</td>
<td>10.7 ± 0.5</td>
<td>26.3 ± 3.5</td>
</tr>
<tr>
<td>B 4.45 ± 0.62</td>
<td>16.6 ± 1.6</td>
<td>10.3 ± 1.0</td>
<td>1.40 ± 0.13</td>
<td>21.7 ± 3.0</td>
<td>12.5 ± 0.5</td>
<td>22.0 ± 3.7</td>
<td></td>
</tr>
<tr>
<td>C 4.50 ± 0.58</td>
<td>16.6 ± 1.6</td>
<td>10.8 ± 1.0</td>
<td>1.40 ± 0.13</td>
<td>22.1 ± 2.9</td>
<td>11.2 ± 0.4</td>
<td>24.6 ± 3.5</td>
<td></td>
</tr>
<tr>
<td>Engstrom (5 studies)</td>
<td>A 5.78 ± 0.75</td>
<td>18.0 ± 1.0</td>
<td>15.0 ± 0.8</td>
<td>0.84 ± 0.12</td>
<td>43.2 ± 4.3</td>
<td>9.0 ± 0.6</td>
<td>30.6 ± 3.8</td>
</tr>
<tr>
<td>B 5.78 ± 0.65</td>
<td>18.0 ± 1.0</td>
<td>16.0 ± 0.8</td>
<td>0.96 ± 0.14</td>
<td>38.2 ± 3.9</td>
<td>10.8 ± 0.6</td>
<td>30.4 ± 3.4</td>
<td></td>
</tr>
<tr>
<td>C 5.82 ± 0.75</td>
<td>18.0 ± 1.0</td>
<td>16.2 ± 0.7</td>
<td>0.84 ± 0.12</td>
<td>43.1 ± 4.5</td>
<td>9.4 ± 0.6</td>
<td>30.5 ± 4.2</td>
<td></td>
</tr>
</tbody>
</table>

* Values are the mean ± standard error.
† A = control period; B = 15 minutes after compression; C = 10 minutes after decompression.
‡ Significant at the 50 per cent level.
§ Significant at the 57.5 per cent level.
¶ With rigid tubing.
Fig. 2. Representative tracing of airway pressure and flow patterns before, and during chest compression in ventilators studied. (Solid lines—before chest compression, dashed lines—during chest compression.)

A LATOR was volume cycled, expiratory tidal volume during compression was decreased 8.6 per cent from the control. The difference between the volume delivered from the bellows and that delivered to the patient may be accounted for by the distension of non-rigid tubing and/or compressed gas remaining in the bellows at the end of inspiration. Respiratory rate was constant in two dogs and slightly diminished in three other dogs. This observation suggests that in the Ohio as a “Controller,” the respiratory rate may decrease in the face of a lowered compliance. At ten minutes after removal of compression, flow rate and tidal volume had almost returned to normal.

AIR-SHIELDS RESPIRATOR (FIGURE 2d): This ventilator is a constant flow generator with the inspiration and expiration time-cycled. The power source is an electrically driven motorblower that acts on a bellows-in-a box ventilator-patient coupler. The inspiratory flow can be adjusted and follows a square wave pattern. Tidal volume is determined by the flow setting and the inspiratory time setting. In order to maintain constant tidal volume with this ventilator, the inspiratory flow should remain constant in the face of an increased airway resistance or decreased compliance.

During compression, a decrease in inspiratory flow and minute volume took place. Tidal volume was also decreased secondary to the decrease in inspiratory flow. The mean decrease in tidal volume was 4.7 per cent. In order to determine whether the decrease in flow could be prevented or minimized by initially setting the ventilator to deliver a very high inspiratory flow rate, one experiment was performed with a 68 liters/minute flow. Under these conditions, almost the same decrease in tidal volume (4.2 per cent) and inspiratory flow was found during chest compression. Experiments in which the distensible tubing was replaced with rigid tubing resulted in almost the same decrease in tidal volume. Therefore the main cause for this reduction in tidal volume is probably the compressed gas remaining in the bellows at the end of inflation.

EMERSON POSTOPERATIVE VENTILATOR (FIGURE 2c): This ventilator is a non-constant flow generator with a variable-speed electric motor permitting independent timing of inspiration and expiration. This motor drives a piston which delivers gas directly from the piston chamber to the patient circuit. The inspiratory flow pattern is a sine wave with peak flow at mid-phase. The peak of inspiratory flow
may be altered by adjustment of the inspiration time-cycling control. During compression of the chest, the flow pattern was altered with the peak flow occurring later in the inspiratory cycle while the magnitude of peak flow remained constant. With the long, soft flexible tube provided as part of the patient circuit, there was a 14.5 per cent reduction in tidal volume during compression. In order to eliminate this loss of volume in the expandable tubing and to measure the performance of the ventilator itself, our experiments were performed with a rigid tube. With the rigid tubing the tidal volume was maintained satisfactorily during chest compression. The 1.5-liter humidifier located in the inspiratory pathway of the ventilator-patient circuit, when empty, acted as a dead-space reservoir resulting in a reduction in tidal volume which was accentuated during compression.

**Engström Respirator (Figure 2f):** This ventilator, a sine wave pattern flow generator with fixed time cycling of inspiration and expiration, produces an inspiratory/expiratory ratio of 1:2. However, the actual inspiratory flow time is determined by patient compliance and/or airway resistance and is almost always of shorter duration than the time permitted by the fixed 1:2 ratio. Therefore, at the end of the inspiratory third of the breathing cycle there is a period of no flow. The ability of the ventilator to increase inspiratory flow time into the no flow period produces a reserve of pressure, and hence of flow. Therefore, when the chest was compressed, the inspiratory flow time was prolonged while the peak inspiratory flow was decreased, and the tidal volume thus remained at the control value. The minute volume and respiratory frequency remained constant during compression.

**Physiological Analysis of Ventilator Performance**

**Anatomical and Physiological Dead Space (Anat. Vₐ and Phys. Vₐ):** Both anat. Vₐ and phys. Vₐ were reduced during compression of the chest in all dogs (table 3). These decreases in dead space were statistically significant except with the Emerson and the Engström ventilators. The decreases in dead space with the pressure-controlled ventilator (Bird) was much greater than that with any of the volume-controlled ventilators studied. The decrease in both anat. Vₐ and phys.

### Table 3. Anatomical and Physiological Dead Space (ml./kg.) During Three Test Periods

<table>
<thead>
<tr>
<th>Ventilator</th>
<th>Test Period</th>
<th>Control</th>
<th>15 Minutes After Compression</th>
<th>10 Minutes After Decompression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bird, without Air-Mix (4)</td>
<td>anat. Vₐ</td>
<td>5.0 ± 0.3</td>
<td>3.6 ± 0.2†</td>
<td>4.8 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>5.8 ± 0.4</td>
<td>3.9 ± 0.3†</td>
<td>5.4 ± 0.3</td>
</tr>
<tr>
<td>Bird, with Air-Mix (4)</td>
<td>anat. Vₐ</td>
<td>4.9 ± 0.4</td>
<td>3.4 ± 0.1†</td>
<td>4.8 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>6.3 ± 0.5</td>
<td>4.0 ± 0.2†</td>
<td>6.1 ± 0.4</td>
</tr>
<tr>
<td>Ohio (5)</td>
<td>anat. Vₐ</td>
<td>4.9 ± 0.3</td>
<td>4.1 ± 0.3*</td>
<td>4.6 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>5.9 ± 0.4</td>
<td>4.9 ± 0.3*</td>
<td>5.6 ± 0.3</td>
</tr>
<tr>
<td>Air-Shields (4)</td>
<td>anat. Vₐ</td>
<td>4.5 ± 0.2</td>
<td>3.8 ± 0.2*</td>
<td>4.3 ± 0.4</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>5.6 ± 0.2</td>
<td>5.0 ± 0.3*</td>
<td>5.6 ± 0.4</td>
</tr>
<tr>
<td>Emerson (5)</td>
<td>anat. Vₐ</td>
<td>4.5 ± 0.4</td>
<td>4.1 ± 0.4</td>
<td>4.5 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>5.7 ± 0.3</td>
<td>5.2 ± 0.4</td>
<td>5.8 ± 0.5</td>
</tr>
<tr>
<td>Engström (5)</td>
<td>anat. Vₐ</td>
<td>4.7 ± 0.4</td>
<td>4.1 ± 0.6</td>
<td>4.6 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>phys. Vₐ</td>
<td>5.5 ± 0.5</td>
<td>5.0 ± 0.5</td>
<td>5.3 ± 0.6</td>
</tr>
</tbody>
</table>

Values are expressed as the mean ± standard error.
Numbers in parentheses indicate the number of studies with each ventilator.
* Significant at the 5 per cent level.
† Significant at the 2.5 per cent level.
**Fig. 3.** Effects of Chest Compression on anatomical dead space (anat. $V_D$, ml./kg.), $V_t$ (tidal volume—apparatus dead space, ml./kg.), and alveolar ventilation ($V_A$, ml./kg./minute). The area between $V_t$ and anat. $V_D$ (shaded parts) represents alveolar tidal volume. Points plotted are the means of observations.

$V_D$ during chest compression appears to be part of an actual decrease in lung volume. This may be accentuated by those ventilators unable to maintain the preset volume in the face of chest compression.

**Alveolar Ventilation ($V_A$)**: The alveolar ventilation may be derived from the following equation: $V_A = (\text{tidal volume} - \text{anat. } V_D - \text{apparatus dead space}) \times \text{respiratory rate}$. Since both tidal volume and anat. $V_D$ vary independently, $V_A$ equals the difference between these two variable factors minus, the apparatus dead space (a constant), all multiplied by frequency. When the anat. $V_D$ is reduced and tidal volume remains the same, or the decrease in anat. $V_D$ exceeds the decrease in tidal volume, there will be an increase in alveolar ventilation. In figure 3, changes in $V_A$ are shown in relation to changes in tidal volume and anat. $V_D$ for all ventilators studied. With the Bird ventilator, the decrease in tidal volume was greater than the decrease in anat. $V_D$, and $V_A$ was markedly reduced (approximately 38.0 per cent, with or without Air-Mix). With the Ohio ventilator and chest compression, there was a decrease in $V_A$, although tidal volume and anat. $V_D$ were decreased nearly in parallel. In some studies, this was due to a decrease in respiratory frequency (table 2). With the Air-Shields ventilator, the decrease in tidal volume and anat. $V_D$ were almost parallel and therefore $V_A$ was essentially unchanged.
With the Engström ventilator, $V_A$ was increased. The mean increase of 5 measurements was 16.0 per cent after 7.5 and 15 minutes of chest compression. These changes were statistically significant. When the Emerson ventilator was used, $V_A$ also showed an increase, but this increase was statistically significant only after 7.5 minutes of chest compression.

**Arterial-Alveolar $P_{CO_2}$ Difference ($a-a$ $P_{CO_2}$ D):** In this experiment, the $a-a$ $P_{CO_2}$ D was quite variable, from 1.0 to 9.5 mm. of mercury, but averaged about 6.1 mm. of mercury during the control period. With the Bird ventilator, during compression, there was a statistically significant increase in $a-a$ $P_{CO_2}$ D. With the volume-controlled ventilators, on the other hand, this parameter remained essentially unchanged throughout the experiments (table 4).

**Arterial $P_{CO_2}$ and pH (Figure 4):** In all of the experiments, the changes in standard bicarbonate were less than 1 mEq./liter. Therefore, changes in pH were considered respiratory in origin. With the Bird respirator alveolar ventilation during the compression was decreased and a marked respiratory acidosis occurred (mean $P_{CO_2}$, 56–60 mm. of mercury), which was not reversed 10 minutes after compression was removed. In contrast, the Engström and Emerson ventilators produced an increase in alveolar ventilation with chest com-

![Fig. 4. Effect of chest compression on arterial $P_{CO_2}$ and pH.](http://anesthesiology.pubs.asahq.org/pdfsaccess.ashx?url=/data/journals/jasa/931617/)
Table 5. Values of Arterial P\textsubscript{O\textsubscript{2}} (mm. Hg) During the Three Test Periods*

<table>
<thead>
<tr>
<th>Inspired Gas</th>
<th>100% Oxygen</th>
<th>Oxygen-Air Mixture (38-62% O\textsubscript{2})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>15 Min. After Compression</td>
</tr>
<tr>
<td>Bird</td>
<td>512 (4)†</td>
<td>420 (−92)†</td>
</tr>
<tr>
<td>Ohio</td>
<td>518 (4)†</td>
<td>496 (−22)‡</td>
</tr>
<tr>
<td>Air Shields</td>
<td>515 (1)</td>
<td>498 (−17)‡</td>
</tr>
<tr>
<td>Emerson</td>
<td>520 (1)</td>
<td>500 (−20)‡</td>
</tr>
<tr>
<td>Engström</td>
<td>546 (3)</td>
<td>533 (−13)</td>
</tr>
</tbody>
</table>

* Values are the means when there was more than one study.
† Number of studies.
‡ Values in parenthesis are differences from the control.

pression and consequently a slight alkalosis resulted.

Arterial P\textsubscript{O\textsubscript{2}} and Alveolar-Arterial P\textsubscript{O\textsubscript{2}} Difference (\(\text{a-a P}_{\text{O}_2}\) D) (Table 5, Figure 4): All the animals demonstrated a fall of \(P_{\text{ao}_2}\) after chest compression. The greatest fall was with the Bird and the least fall with the Engström. To investigate the possibility of increased shunting of blood in the lung during chest compression, \(\text{a-a P}_{\text{O}_2}\) D was measured using three different ventilators (Bird, Ohio and Engström) with 100 per cent oxygen. \(\text{a-a P}_{\text{O}_2}\) D averaged 143 mm. of mercury in the control period. Assuming an a-v oxygen difference of 6 vol. per cent and no change in cardiac output, this value gives an approximate shunt of 6.9 per cent of cardiac output. With the Bird, \(\text{a-a P}_{\text{O}_2}\) D increased from 159 to 243 mm. of mercury (after 15 minutes of compression). At an a-v \(O_2\) difference of 6 vol. per cent, this represents an increase in shunt from 7.6 to 11.1 per cent of cardiac output. Ten minutes after compression was removed, \(\text{a-a P}_{\text{O}_2}\) D decreased to 199 mm. of mercury. This is significantly higher than the control. With the Ohio, \(\text{a-a P}_{\text{O}_2}\) D is increased by chest compression. The shunt value, again assuming 6 vol. per cent a-v \(O_2\) difference, is increased from 6.6 to 7.8 per cent of cardiac output. With the Engström, \(\text{a-a P}_{\text{O}_2}\) D increased after chest compression and the shunt increased from 6.2 to 6.7 per cent. At 10 minutes after removal of chest compression, the increased \(\text{a-a P}_{\text{O}_2}\) D did not return toward the control value with either the Ohio or Engström ventilators.

Changes in Circulatory Parameters: All animals had an increase in femoral venous pressure immediately after chest compression, which ranged from the mean increase of 3.0 cm. of water with the Bird without Air-Mix to 7.0 cm. of water with the Ohio. The rise in venous pressure was more distinct with the “volume-controlled” ventilators than with the “pressure-controlled” ventilator. There was no obvious difference among the ventilators of volume-controlled operation. The elevated venous pressure returned to the control level immediately after chest compression was removed. Right atrial pressure also rose 3.5–6.8 cm. of water after chest compression, and the changes were nearly in parallel to that of venous pressure.

Arterial blood pressure and pulse rate did not show any appreciable changes throughout the experiment.

Discussion

It is of interest that the results of this study which relate to mechanical factors such as respiratory volume, inspiratory flow and inflating pressure are quite comparable to the results obtained from studies in a model lung.\(^1,^2\) These findings provide confirmation in vivo of the applicability and relevance of previous studies, when these physical parameters are the center of interest. While such observations are sufficient if one is concerned only with the mechanical performance of the ventilator itself, the model lung cannot reflect any of the changes which may occur in respiration and
circulation. It is to these physiologic alterations that we draw attention.

The initial ventilation was standardized by regulating the end-tidal $P_{CO_2}$ to a predetermined control. The challenge to the ventilator was achieved by a restriction of chest expansion which reduced lung volume and total compliance. The physiological changes produced in alveolar ventilation, and arterial blood oxygenation, with the attendant alterations in acid-base balance, constituted the basis of these experiments.

The difference between pressure- and volume-controlled ventilators is graphically depicted in figures 3 and 4. The superiority of volume-controlled ventilators to automatically meet the challenge of chest compression is dramatically revealed. The pressure limited respirator may be adjusted to compensate, but this requires recognition of the changing situation and alteration in pressure and/or flow controls. At the same time, the relationship between changes in anatomical dead space and alveolar ventilation and their effect on pH and $P_{CO_2}$ is made abundantly clear. During chest compression, the a-a $P_{CO_2}$ D was essentially unchanged with the volume-controlled ventilators, but was significantly increased when the pressure-controlled ventilator was employed. This suggests that volume-controlled ventilators are better able to maintain a constant ventilation/perfusion ratio during chest compression.

Since it was apparent that a reduction in respiratory dead space could be produced by chest compression, Trendelenburg position and an inflated cuff around the abdomen were also evaluated in a few animals. Identical results were obtained (unpublished observations). This finding suggests that volume-controlled ventilators, especially of the Engström type, can indeed increase alveolar ventilation and more effectively reduce $P_{CO_2}$ under these circumstances. However, this may be offset by the degree of shunt which may be increased by chest compression. In the experiments in which animals were ventilated with 100 per cent oxygen, in spite of the increased alveolar ventilation, the a-a $P_{O_2}$ D tended to rise. While this was not significant with the volume-controlled ventilators, there appeared to be a much greater increase in shunt and fall in arterial $P_{O_2}$ with the pressure-controlled ventilator.
Attempts have been made to take advantage of decreasing lung volume in patients with emphysema. Our results, if they may be applied to man, suggest that in the emphysematous patient being artificially ventilated, only precision, volume-controlled ventilators, with moderately oxygen-enriched atmospheres, should be employed.

McIlroy et al., in conscious man, found that lowered lung compliance did not return to control values when chest compression was removed. Even after deep breaths Pao2 did not rise to the level observed in the control situation. A similar observation in anesthetized dogs has been reported by Laver et al. who stated that marked inflations accompanied by threefold increase in tidal volume were required to restore the arterial Pao2 to its precompression values. Our results support these observations because, for 10 minutes after the removal of chest compression, the slight decrease in total compliance persisted, the A-a Pao2 D remained elevated, and the arterial PCO2 levels were still abnormal, except with the Emerson and Engström ventilators. The decrease in oxygenation is best explained on the basis of poorly ventilated areas, or collapse of terminal lung units; it appears that this is minimized with a volume-controlled ventilator.

As was emphasized in a previous study, the effects as measured by a model lung do not reveal the manner in which gas is distributed in the lung itself. Unfortunately, this aspect of the problem is not directly attacked in this investigation. It is obvious from the results obtained by measuring the physiologic parameters, that such a study is essential if one is to be able to analyze more specifically the ability of a ventilator to ventilate. Partial evaluation, however, has been obtained with the measurements of Pao2 and acid-base balance. The response of arterial Pao2 to oxygen gave an indication, rough as it was, of shunting, which was more precisely determined by measuring the A-a gradient. These studies during decreased compliance strikingly reveal the difference between the two types of ventilators, and point up the quantitative differences in the various volume-controlled ventilators. It must be realized that these were short-term experiments. One can only speculate that if carried out over a longer period of time, the differences might become greater.

These experiments indicate the feasibility of utilizing the dog as a test animal for measuring ventilator performance. The advantages of such a technic in precision and in gaining insight into the manner in which respiration is affected appears to us to be advantageous. The quantitative differences among the volume-controlled ventilators studied, in response to the challenge chosen were not significant; it was our impression that the Engström respirator was able to meet altered situations with the most precise response. Studies directed toward the effects of various wave forms on distribution of inspired gas, shunting and the ventilation-perfusion ratio appears to be the most fruitful avenue for future investigation.

Summary

The mechanical and physiological performance of five commercially available ventilators was studied in the face of a lowered total compliance produced by constant chest compression in dogs. During chest compression, the dogs on the Bird ventilator showed an increase in arterial PCO2, a decrease in blood pH, and a 28 per cent decrease from the preset delivered tidal volume. There was a slight decrease in preset tidal volume with the Ohio and the Air-Shields ventilators. The Emerson and the Engström ventilators maintained the tidal volume most satisfactorily. When the chest was compressed, anatomical and physiological dead space was reduced in all dogs. With the Engström and the Emerson ventilators, alveolar ventilation was increased and arterial PCO2 decreased with chest compression. Arterial Pao2 was reduced in all the cases with chest compression, regardless of the type of ventilator employed, and the greatest fall was with the Bird ventilator. Changes in the A-a oxygen gradient also suggested that the Engström ventilator was able to provide even ventilation throughout the lungs during compression.

References


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**Respiration**

**Respiratory Failure** Physiologic dead space to tidal volume ratios from 0.38 to 0.79 and physiologic shunts from 5 to 79 per cent of the cardiac output were found in patients on long term controlled ventilation. Tidal volume changes at a constant frequency of 20 breaths per minute did not appear to influence the ratio of dead space ventilation to tidal ventilation. This ratio is thus a useful index of cardio-pulmonary function during constant frequency controlled ventilation. Tidal hyperventilation decreased the cardiac output in the patients with emphysema, but in the patients with cardio-pulmonary disease without emphysema, cardiac output was essentially unaffected by the level of tidal ventilation. The percentage of physiologic shunt was proportional to the cardiac output. Increases in output were associated with increases in the proportion of blood apparently flowing past unventilated alveoli. In patients with cardio-pulmonary disease but without emphysema, the level of tidal ventilation did not significantly affect the percentage of physiologic shunt. When established lung disease has caused large physiologic shunts and dead space to tidal volume ratios, the distribution of pulmonary blood flow appears to be influenced more by cardiac output than by tidal ventilation and intratracheal pressure. (Hedley-White, J., Pontoppidan, H., and Morris, M. J.: *The Response of Patients with Respiratory Failure and Cardiopulmonary Disease to Different Levels of Constant Volume Ventilation, J. Clin. Invest.* 45: 1543 (Oct.) 1966.)