Observations of Ventilation During Resuscitation in a Canine Model

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Background Fear of infection limits the willingness of laymen to do cardiopulmonary resuscitation (CPR). This study assessed the time course of change in arterial blood gases during resuscitation with only chest compression (no ventilation) in an effort to identify the time for which ventilation could be deferred.

Methods and Results Aortic pressures and arterial blood gases were monitored in seven 20- to 30-kg dogs in ventricular fibrillation (VF) at 2-minute intervals during chest compression alone (no ventilation) at 80 to 100 compressions per minute. Before the induction of ventricular fibrillation, all animals were intubated and ventilated with room air, 10 mL/kg. The endotracheal tube was removed when VF was induced. Pre-VF arterial pH, Pco2, and O2 saturation were (mean±SEM) 7.39±0.02, 27.0±1.5 mm Hg, and 97.5±0.5%, respectively, with aortic pressures being 143.2±5.7/116.2±4.6 mm Hg. At 4 minutes of chest compression alone, the corresponding values were 7.39±0.03, 24.3±3.1 mm Hg, and 93.9±3.0%, with an arterial pressure of 48.1±7.7/22.6±3.9 mm Hg. Mean minute ventilation during the fourth minute of CPR, measured with a face mask-pneumotachometer, was 5.2±1.1 L/min.

Conclusions These data suggest that in the dog model of witnessed arrest, chest compression alone during CPR can maintain adequate gas exchange to sustain O2 saturation >90% for >4 minutes. The need for immediate ventilation during witnessed arrest should be reexamined. (Circulation. 1994;90:3070-3075.)

Key Words • ventilation • cardiopulmonary resuscitation

The traditional teaching of cardiopulmonary resuscitation (CPR) in the United States and many other countries is based on the “A-B-C” sequence, i.e., the prompt initiation of Airway patency, Breathing, and Circulation (chest compression), a sequence that reflects the historical development of these techniques. However, an alternative approach is that of C-A-B: Chest compression, Airway, Breathing, in which airway manipulation is deferred for several seconds. In dogs with cardiac arrest, Meursing et al delayed CPR 5 minutes after arrest and then initiated chest compression but withheld ventilation for 2 minutes and demonstrated no significant fall in arterial Pco2 or rise in Pco2 for 30 seconds. At 45 seconds after the initiation of chest compression alone, arterial Pco2 was lower, but still 52.3 mm Hg, which probably reflected an arterial hemoglobin saturation of 70% to 80%. Based on these and other data in the Netherlands, clinical CPR does not employ the “A-B-C” rule; rather, the approach is “C-A-B” (Circulation-chest compression, Airway, Breathing), with ventilation being delayed and chest compression being initiated as soon as possible. Using the “C-A-B” approach, survival data from the Netherlands suggest resuscitation rates comparable to those seen in the best paramedic response centers in the United States. The actual delay between chest compression and ventilation has never been reported, but it is probably in the range of several seconds and possibly >1 minute. No study has addressed the issue of when ventilation must be initiated during CPR to ensure favorable results. This becomes particularly applicable in the 1990s because fear of infection has prompted many laymen and medical personnel to avoid performing mouth-to-mouth ventilation in unknown victims of cardiac arrest. Laymen would probably be willing to perform chest compression alone and activate emergency medical system personnel, who on arrival could then initiate ventilation with the necessary protective equipment. These issues become particularly important to explore because clinical studies have previously shown that prompt bystander CPR after arrest improves outcome.

During CPR, of the various indexes of respiratory function, arterial oxygen saturation and content are of significant importance because they, along with blood flow, determine the delivery of oxygen to tissue such as the brain and heart. In most clinical situations, tissue oxygenation is adequate if arterial oxygen saturation is maintained >90%. The present recommendations for ventilation during CPR are based on studies that were conducted in the 1960s and are founded on the premise that the ventilatory requirements during CPR are comparable to those during normal circulation. However, substantially less ventilation may be sufficient to maintain arterial oxygenation and CO2 clearance during CPR, because venous return and cardiac output are only 10% to 15% of normal during manual chest compression. The goal of ventilation during CPR is to provide adequate oxygenation and CO2 removal. What is “adequate” has never been established. Data from Bishop et al suggest that an arterial Pco2 <35 mm Hg during CPR is effective in correcting the metabolic

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Acidosis of hypoperfusion during CPR. Also, a lower PCO2 (in the range of 25 to 35 mm Hg) may limit cerebral edema. From these observations, it would appear that ventilation sufficient to produce a Paco2 <35 mm Hg and an arterial O2 saturation >90% would probably be sufficient and meet bodily needs during the initial phases of resuscitation.

The specific goal of this study was to measure in dogs, during resuscitation with chest compression alone, the time course of rise in arterial PCO2 and fall in arterial pH and O2 saturation. We reasoned that the time course of fall in oxygen saturation and pH would help identify the time when ventilation must be initiated during CPR.

Methods
This study was approved by the Animal Care and Use Committee of The Johns Hopkins University. This committee conforms strictly to the guiding principles of the American Physiological Society. Seven mongrel dogs weighing 20 to 30 kg were anesthetized with intravenous pentobarbital, intubated, and positioned supine in a special cradle as previously described. Additional anesthetic was given as needed. All animals were ventilated with a Harvard ventilator (model 607), 10 mL/kg tidal volume, at a frequency of 10 to 12 breaths per minute. Millar micromanometer-tipped catheters (model PC-470) were inserted retrograde into the right atrium and ascending aorta via femoral cutdowns, and a pacing catheter was positioned in the right ventricle. The neck of the dog was carefully maintained in a neutral position, avoiding extension or flexion. The head of the animal was not specifically positioned in any holding device. A special face mask was constructed for purposes of this study and is depicted in Fig 1. This conical mask was fitted over the snout, glued into position, and then taped to provide an airtight seal. This mask had at its apex a removable stopper with an opening that could be connected to a pneumotachometer (Fleisch model 3). Before intervention, the endotracheal tube exited through this opening. When the cone was positioned, care was taken not to compress subpharyngeal structures. Baseline hemodynamics were recorded on a Gould 4 channel recorder (model 2406), and baseline arterial blood gases were drawn. All blood gases were measured on a Radiometer ABL3 electrode and analyzer system that was calibrated and set to the animal's body temperature. The animal's temperature was recorded with a rectal mercury thermometer before arrest and twice during the period of chest compression. After the induction of ventricular fibrillation (via the right ventricular pacing electrode), the endotracheal tube was removed, and the face mask stopper was replaced and connected to a pneumotachometer.

To minimize dead space during CPR, the pneumotachometer was connected to the exit port for measurements of air flow for only 5 to 10 seconds every 60 seconds. Hemodynamic and air flow data were digitized at a rate of 200 Hz and recorded on magnetic disk and also on a Gould 4 channel recorder. Tidal volumes were obtained by integration of the inspiratory air flow. Chest compression was initiated, and chest compression force was maintained constant at 400 N for 14 minutes by use of a hand-held device (previously described). Chest compression force was chosen to be 375 to 400 N on the basis of prior studies that have shown that vital organ perfusion is optimal and trauma less likely at this compression force. Arterial blood gases were measured at 2-minute intervals.

Data are reported as mean±SEM. Arterial PCO2, PO2, and pH were analyzed as a function of time with custom-written software. For vascular pressures (aortic and right atrial), 10 beats were analyzed before arrest and at 2, 4, and 6 minutes after the initiation of ventricular fibrillation. Means and SEM were calculated for each animal for each period in time and then the mean was developed for the overall group for each of the time intervals indicated. Tidal volume was similarly calculated by analysis of five representative beats. Minute ventilation was calculated by multiplying the tidal volume by the chest compression rate.

Results
Fig 2 is a representative tracing of a dog in the fourth minute of chest compression alone. With chest compression, aortic and right atrial pressures increase by a similar amount and air leaves the chest. On chest compression release, marked by a fall in chest compression force, air enters the upper airway. The tidal volume for Fig 2 was 103 mL per breath, with the minute
ventilation during the fourth minute of resuscitation being 8.7 L/min.

Fig 3, upper left, shows pooled data from seven dogs and depicts the change in \( O_2 \) saturation during chest compression without ventilation over a 14-minute time period. At 4 minutes after the onset of ventricular fibrillation with chest compression alone, oxygen saturation was still approximately 94\% (Table). The range of \( O_2 \) saturation at 4 minutes was from 77\% to 97.8\%, with six of seven dogs being >90\% (Fig 3, lower right). At 6 minutes, it had fallen to 87\%.

**Ventilation During Only Chest Compression**

<table>
<thead>
<tr>
<th>Time (2-Min Intervals During Resuscitation)</th>
<th>Prearrest</th>
<th>2 Min</th>
<th>4 Min</th>
<th>6 Min</th>
<th>8 Min</th>
<th>10 Min</th>
<th>12 Min</th>
<th>14 Min</th>
</tr>
</thead>
<tbody>
<tr>
<td>AO sys, mm Hg</td>
<td>143.2±5.7</td>
<td>49.6±5</td>
<td>48.1±7.7</td>
<td>52.2±10.2</td>
<td>36.1±7.8</td>
<td>33.6±4.7</td>
<td>34.4±4.8</td>
<td>33.7±4.1</td>
</tr>
<tr>
<td>AO dia, mm Hg</td>
<td>116.2±4.6</td>
<td>25.4±3</td>
<td>22.6±3.9</td>
<td>24.6±6.0</td>
<td>21.8±4.8</td>
<td>19.5±3.3</td>
<td>19.3±3.2</td>
<td>18.1±2.4</td>
</tr>
<tr>
<td>RA mean, mm Hg</td>
<td>5.3±1.4</td>
<td>17.1±1</td>
<td>14.8±0.8</td>
<td>12.9±0.7</td>
<td>13.4±0.5</td>
<td>12.2±0.7</td>
<td>11.3±1.0</td>
<td>11.2±0.9</td>
</tr>
<tr>
<td>Min vent, L/min</td>
<td>...</td>
<td>4.9±0.9</td>
<td>5.2±1.1</td>
<td>6.1±1.2</td>
<td>6.7±1.3</td>
<td>5.6±1.1</td>
<td>5.7±1.1</td>
<td>5.1±0.8</td>
</tr>
<tr>
<td>Tidal vol, mL/breath</td>
<td>...</td>
<td>70.2±17.9</td>
<td>73.3±17.7</td>
<td>81.7±16.1</td>
<td>89.5±16.1</td>
<td>78.1</td>
<td>79.5±12.2</td>
<td>69.7±12.8</td>
</tr>
<tr>
<td>pH</td>
<td>7.39±0.02</td>
<td>7.39±0.02</td>
<td>7.39±0.03</td>
<td>7.36±0.05</td>
<td>7.29±0.05</td>
<td>7.19±0.04</td>
<td>7.19±0.05</td>
<td>7.15±0.04</td>
</tr>
<tr>
<td>( P_{CO_2} )</td>
<td>27±1.5</td>
<td>25.7±3.1</td>
<td>24.3±3.1</td>
<td>26.3±5.3</td>
<td>34.3±4.9</td>
<td>38.8±6.4</td>
<td>46.9±7.3</td>
<td>48.5±6.8</td>
</tr>
<tr>
<td>( P_O_2 )</td>
<td>108.9±7.5</td>
<td>101.6±7.9</td>
<td>95.8±11.7</td>
<td>84.3±14.6</td>
<td>57.3±9.7</td>
<td>42.0±9.4</td>
<td>40.9±7.5</td>
<td>36.1±5.7</td>
</tr>
<tr>
<td>% ( O_2 ) sat</td>
<td>97.5±0.5</td>
<td>96.44±1.2</td>
<td>93.9±2.8</td>
<td>87.5±6.8</td>
<td>75.2±1.0</td>
<td>70.4±10.6</td>
<td>58.8±10.5</td>
<td>50.7±9.9</td>
</tr>
</tbody>
</table>

AO sys indicates aortic systolic pressure; Ao dia, aortic diastolic pressure; RA mean, right atrial mean pressure; Min vent, minute ventilation; and tidal vol, tidal volume. Arterial pH, \( P_{CO_2} \), \( P_O_2 \), and % \( O_2 \) saturation values are also noted.
These time intervals are chosen for display because they are clinically meaningful and span the time periods when arterial O₂ saturation falls below 90%. Vascular pressures are low, since epinephrine was not administered, but are similar to those that have been reported previously during conventional CPR without epinephrine.15 Despite a fall in pH and Po₂, there is no substantial hemodynamic effect and specifically no decrease in arterial pressure observed for >6 minutes. Tidal volume during chest compression alone also remains fairly constant over time. The range of tidal volume in the dogs studied was 21 to 127 mL per breath.

**Discussion**

Resuscitation research over the past 20 years has been broad based and has encompassed the study of the epidemiology of arrest, the hemodynamics of CPR, and the role of early defibrillation and vasopressors in resuscitation.9,12,14,17-21 Based on the results of these studies, recommendations have been developed that have formed the basis for the changes in the American Heart Association Standards and Guidelines for CPR.1 Despite this growth in knowledge, little is known of the role of ventilation during CPR. The 1990s have ushered in an era in which a perceived risk of disease transmission may undermine the significant public education effort of bystander CPR that marked the 1970s and 1980s.7,8 Although often considered, transmission of HIV during CPR has never been documented.22 Nevertheless, the risk of transmission of other diseases during CPR, such as herpes simplex, has been documented, and transmission of drug-resistant tuberculosis is also a matter of concern.23 Although most cardiac arrest victims collapse at home and hence are attended to by relatives and friends, several surveys and studies have identified the lack of willingness of most laymen and medical personnel to initiate CPR in unknown victims of cardiac arrest.7,8

When should ventilation be initiated during CPR in adults? What is the tidal volume necessary during CPR to maintain adequate gas exchange? There is little in the literature that gives concrete answers to these basic questions about ventilation during resuscitation. This preliminary study was designed to initiate the process of reevaluating ventilation during CPR. The pioneering work of Safar and Elam in the 1950s established the superiority of the mouth-to-mouth technique of ventilation compared with the manual techniques of ventilation that did not require any oral contact between rescuers and victim. Safar et al2 compared tidal volumes during mouth-to-mouth or mouth-to-airway respiration with manual ventilation in anesthetized and curarized apneic adults studied with natural airways and an artificial oral pharyngeal airway. These studies suggested that of all the manual techniques of ventilation, the Holger-Nielsen method, although inferior to the mouth-to-mouth technique, was probably the most effective in maintaining tidal volume. On the basis of these and other studies in 1961, these investigators recommended the preferential use of mouth-to-mouth ventilation during CPR. It was further recommended that mouth-to-mouth ventilation be initiated immediately and interposed after every five chest compressions, since chest compression alone moved relatively small volumes of air.11 Whether ventilation could be delayed in relation to chest compression was never assessed, and the studies of ventilation frequency during CPR were also limited in scope.11 These earlier studies of manual and mouth-to-mouth ventilation cannot be readily extrapolated to the human cardiac arrest situation. In most of these studies, the patients were alive, with nearly normal cardiac output.2 In addition, these studies presumed that the goal of ventilation during CPR was to achieve nearly normal tidal volumes and minute ventilation; hence, the emphasis in all of these studies was the prompt institution of ventilation with normal or higher than normal tidal volumes.

Our data show that in a canine model of witnessed cardiac arrest when chest compressions were started within 1 minute of arrest, arterial oxygen saturation remained >90% for 4 minutes after initiation of chest compressions without ventilation. Changes in arterial pH and Po₂ that occurred were not of sufficient magnitude to affect vascular tone, as judged by the lack of change in aortic diastolic pressure. Our observations are supported by data from Berg et al,24 who demonstrated no change in survival in dogs with witnessed cardiac arrest in whom only chest compressions were performed before defibrillation (compared with conventional CPR).

Weil et al25 demonstrated that during CPR in humans (in-hospital arrest victims), arterial pH, Pco₂, and bicarbonate change little from prearrest values. This observation can probably be explained by the reduced cardiac output of CPR and adequate alveolar ventilation. On the basis of these observations and studies, we hypothesize that the recommended parameters for tidal volume and minute ventilation may be unnecessarily high for CPR in most clinical situations. In addition, because of the reduced cardiac output of CPR, if cardiac arrest occurs with the blood relatively well oxygenated, ventilation could well be briefly deferred if chest compression is initiated promptly. CPR Registry data from Belgium further support the concept of deferred ventilation.26 Using an observational database of 2971 victims of cardiac arrest, Bossaert et al26 examined 21-day survival in patients undergoing out-of-hospital resuscitation. Of those who had no bystander CPR, 6% survived (123 of 2055 patients). Of those who had external chest compression and mouth-to-mouth ventilation (probably initiated in an A-B-C sequence, since this is the preferred technique in Belgium), 12% survived (67 of 561 patients) (P<.001). However, 9% (24 of 258) of those who had external chest compression alone and no ventilation survived (P<.05 compared with no bystander CPR). This compares with only 5 of 97 patients who survived from those who had only mouth-to-mouth ventilation (P=NS versus no bystander CPR).26 Since these data represent an observational database, ie, a registry, it is unknown whether the groups were balanced in terms of downtime, accuracy of diagnosis, etc. Nevertheless, by virtue of the large numbers and the fact that cardiac arrest was confirmed in all patients by physicians (who were part of the Emergency Medical Response System in Belgium), these observations are intriguing and support the hypothesis that ventilation may be delayed if chest compressions are initiated immediately.

The applicability of animal data to the human cardiac arrest situation can obviously be questioned, and the use
of the dog to model ventilation during CPR in humans may be regarded by some as a limitation. Although the dog has been used extensively as the standard model to study circulation and the hemodynamics of CPR, there is relatively little published information regarding the similarity of canine and human respiratory function during resuscitation. However, dog models have proved extremely useful in the study of various pathological and physiological states such as the adult respiratory distress syndrome,27,28 pulmonary embolism,29 pneumonia,30 pulmonary fibrosis, and emphysema.31 Moreover, despite anatomic differences in chest configuration, intrathoracic vascular pressures during chest compression in humans and large dogs have been shown to be similar.17 Thus, it is probable that the transmission of chest compression forces to the lungs is also similar. We recently demonstrated that cineradiographic images of medium-sized bronchi during chest compression in dogs are similar to those obtained during forced expiratory efforts in humans.32

Another limitation of this study is that the mean PaCO2 before cardiac arrest was approximately 27 mm Hg. This low PaCO2 probably reflects respiratory compensation for tissue acidosis that occurred from femoral artery ligation for catheter placement. The low initial PaCO2 values could potentially cause a longer period of time for PaCO2 to rise to values considered unacceptable. However, PaCO2 initially decreased during the 4 to 6 minutes after cardiac arrest, indicating that the level of ventilation that occurred with chest compression only was more than sufficient to clear the relatively small amount of CO2 delivered to the lungs during this phase. From 6 to 14 minutes after cardiac arrest, PaCO2 increased from approximately 26 to approximately 48 mm Hg (Fig 3, lower left). The rate of rise in PaCO2, even when starting from a level of 30 mm Hg, indicates that it took at least 3 to 4 minutes for the PCO2 to rise by 5 mm Hg (Fig 3, lower left).

Unfortunately, there are no data on actual ventilation during chest compression alone in patients with true cardiac arrest. The data that exist were obtained in patients with beating hearts. Their relevance to human CPR, in which cardiac output is reduced and upper airway obstruction is common, is unclear. Upper airway obstruction can be overcome by positioning the head and neck as shown by Elam et al35 in 1958, but an in-depth knowledge of ventilation during resuscitation is lacking. Our study was designed to initiate the process of reevaluating ventilation during CPR because of the increasing concern of even health care professionals to initiate mouth-to-mouth ventilation.7

Our data suggest that in the animal model, ventilation can be deferred for several minutes in a witnessed arrest and arterial saturation maintained >90%. These data are clearly not sufficient to warrant any change in the performance of human CPR, nor do they represent a call to cease ventilation during resuscitation. They should be regarded only as a stimulant to study ventilation during resuscitation more intensely. The prompt institution of ventilation during CPR is undoubtedly ideal but is causing great concern in our present health care environment. The preliminary Belgian Registry data are intriguing and suggest that brief periods of chest compression alone are clearly superior to no CPR at all and nearly as effective as conventional CPR (chest compression plus ventilation) in terms of survival. In light of our present health care environment, these data should prompt a reexamination of the recommendations for ventilation and specifically the need for immediate ventilation during witnessed arrest. Ventilatory requirements in victims of unwitnessed arrest may be quite different, to judge by preliminary animal data.34 Reluctant rescuers should be encouraged to activate the Emergency Medical Service system and at least open the airway and initiate chest compressions, pending the arrival of trained and equipped rescuers/paramedics. Further detailed studies to better identify the timing of ventilation and tidal volumes during resuscitation in humans are clearly needed.

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References


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