Effect of Ventilation on Resuscitation in an Animal Model of Cardiac Arrest

Ahamed H. Idris, MD, FACEP; Lance B. Becker, MD; Ronnie S. Fuerst, MD; Volker Wenzel; William J. Rush, BA; Richard J. Melker, MD, PhD; David J. Orban, MD, FACEP

Background The need for ventilation during the initial management of cardiac arrest is an important public health problem that is being debated. The present study was designed to determine whether ventilation affects return of spontaneous circulation from cardiac arrest in a swine model with an interval of untreated ventricular fibrillation of 6 minutes, as reported in witnessed out-of-hospital human cardiac arrest.

Methods and Results Twenty-four animals were randomly assigned to two groups: one that received ventilation during the first 10 minutes of chest compression and one that did not. Coronary perfusion pressure and minute ventilation were continuously recorded. Arterial and mixed venous blood gases were measured at intervals. Return of spontaneous circulation was defined prospectively as an aortic systolic blood pressure of >80 mm Hg for >5 minutes and was the primary outcome variable. All animals were anesthetized, paralyzed, and intubated. Ventricular fibrillation was induced and persisted for 6 minutes without chest compression, followed by mechanical chest compression for 10 minutes and then attempted defibrilation. Animals without return of spontaneous circulation were given epinephrine, ventilation, and chest compression for an additional 3 minutes. Defibrillation was again attempted, and animals were assessed for return of spontaneous circulation.

Recently, an expert panel on cardiopulmonary resuscitation (CPR) questioned the need for ventilation during the initial management of human cardiac arrest.1 This is an important issue to be resolved because many people, including health professionals, have become fearful of giving mouth-to-mouth ventilation because of the threat of transmissible diseases such as the human immunodeficiency virus and tuberculosis.2,3

The importance of ventilation during CPR is supported by studies showing that survival from shock and cardiac arrest is influenced by blood acid-base balance and oxygenation.4-15 Both hypoxia and hypercarbic acidosis profoundly decrease the force of contraction of the myocardium, raise the threshold for defibrillation, and adversely affect prognosis.16-22 A recent laboratory study compared the effects of normal ventilation, hyperventilation, and hypventilation on arterial and mixed venous blood gases during low blood flow rates comparable to those that occur during shock and cardiac arrest. During the lowest blood flow rates, hypventilation adversely affected mixed venous pH (P<.05), cP02, and PCO2, whereas hyperventilation improved mixed venous hypercarbic acidosis.23

Few investigations have focused on the effects of ventilation on resuscitation from cardiac arrest, and those that have did not control or measure ventilation during CPR.24-27 The objective of the present study was to compare the effectiveness of chest compression with or without ventilation in producing return of spontaneous circulation following cardiac arrest. The study used a swine model in which spontaneous ventilation was prevented and minute ventilation was measured continuously. In addition, we used a prolonged period of untreated ventricular fibrillation (6 minutes). Although this model did not attempt to simulate either basic or advanced life support CPR, the interval of untreated ventricular fibrillation of 6 minutes used in this study is similar to the mean interval from collapse to the start of CPR reported in witnessed human out-of-hospital cardiac arrest.28

Conclusions In this animal model of cardiac arrest, ventilation was important for resuscitation. The importance of ventilation could be related to the prolonged duration of untreated ventricular fibrillation and the significantly greater hypoxia and hypercarbic acidosis found in the nonventilated animals. (Circulation. 1994;90:3063-3069.)

Key Words • carbon dioxide • cardiopulmonary resuscitation • hypoxia • respiratory acidosis • ventilation

Received March 22, 1994; revision accepted June 27, 1994.

From the Departments of Surgery (Division of Emergency Medicine [A.H.I., R.S.F., V.W., R.J.M., D.J.O.]), Internal Medicine (A.H.I.), Anesthesiology (W.J.R., R.J.M.), and Pediatrics (R.S.F., R.J.M.), University of Florida College of Medicine (Gainesville); and Section of Emergency Medicine (L.B.B.), Department of Medicine, University of Chicago, Ill.

Presented in part at the 66th Annual Scientific Sessions of the American Heart Association, Atlanta, Ga, November 1993.

Correspondence to Ahamed H. Idris, MD, Division of Emergency Medicine, PO Box 100392, University of Florida College of Medicine, Gainesville, FL 32610-0392.

© 1994 American Heart Association, Inc.
four domestic swine weighing between 23 and 61 kg were sedated with ketamine (20 mg/kg IM). Each animal was placed on a U-shaped board in the dorsal recumbent position on an external warming blanket. An ear vein was cannulated with a 20-gauge catheter and a thiamylal bolus (6 mg/kg IV) was given, followed by an infusion at a rate of 9 to 16 mg·kg⁻¹·h⁻¹ to maintain adequate anesthesia as judged by heart rate, blood pressure, spontaneous respiration, and the canthal reflex. A midline incision was made in the anterior neck, and an 8-mm ID endotracheal tube was secured in the trachea. The animal’s lungs were ventilated with a time-cycled volume-controlled ventilator (model 3-PV with model 3V-GB IMV control, J.H. Emerson Co). Initial tidal volume was 12 mL/kg with a respiratory rate of 10 breaths/min and an inspired O₂ fraction (Fio₂) of 0.4. Tidal volume was adjusted to maintain arterial partial pressure of CO₂ (Paco₂) at 40±5 mm Hg. Expiratory flow rate and expiratory time were measured with a pneumotachometer (model 2, A. Fleish), and the signal was electronically integrated to determine ventilation. End-tidal carbon dioxide was measured with a rapid response mainstream capnograph (model 6000, Novametrix Medical Systems Inc). All pulmonary parameters were recorded continuously on a polygraph (model 7D, Grass Medical Instruments).

A bipolar pacing lead was advanced through the left jugular vein into the right ventricle in preparation for inducing ventricular fibrillation. A catheter was placed in the pulmonary artery via the right jugular vein, connected to pressure transducers (model P25, Gould-Statham, Inc) via fluid-filled lines, and right atrial and pulmonary artery pressures were recorded continuously; core temperature was also monitored. Catheter position was verified by the presence of typical pressure waves. To measure mixed venous pH, PO₂, PCO₂, and HCO₃⁻, blood was sampled from the distal port of the pulmonary artery catheter. Cardiac output was measured in triplicate before the induction of ventricular fibrillation. The left common carotid artery was dissected from supporting tissues, and an 8F sheath was advanced into the aorta to record arterial pressure continuously. Blood samples for measuring arterial pH, PO₂, PCO₂, and HCO₃⁻ were drawn from the aortic catheter and measured with a pH/blood gas analyzer (model 1312, Instrumentation Laboratory, Inc). A compression cylinder (Thumper, Michigan Instruments) was positioned on the sternum, and the compression pad was sutured onto the skin overlying the middle of the sternum.

Experimental Procedure

Ten minutes before induction of ventricular fibrillation, the ventilation gas Fio₂ was decreased to 0.21 and baseline prearrest blood gases were measured. Pancuronium (0.2 mg/kg) was administered to prevent spontaneous ventilation during cardiac arrest. Maintenance thiamyal infusion was stopped immediately before induction of ventricular fibrillation, which was induced with an alternating current of 5 to 10 V applied through the right ventricular pacing wire. When ventricular fibrillation was confirmed by ECG and the presence of profound aortic hypotension, ventilation was stopped and the ventilator was disconnected from the endotracheal tube. All animals remained in ventricular fibrillation without ventilation or chest compressions for 6 minutes.

Experimental Groups

Twenty-four animals were assigned to either a ventilated (n=12) or nonventilated (n=12) group. After the initial 6 minutes of untreated ventricular fibrillation, each animal in the ventilated group was reconnected to the ventilator and received tidal volume and minute ventilation identical to prearrest values, except that Fio₂ was increased to 0.85, which is a setting commonly used in laboratory models of CPR.²³⁻³² The animals in the nonventilated group remained disconnected from the ventilator. All animals received mechanical external chest compressions with sufficient force to produce a coronary perfusion pressure of 25 mm Hg (aortic end-diastolic minus right atrial end-diastolic pressure) if possible and a depth of compression of 1.5 to 2 in. Compressions were performed at a rate of 100 min⁻¹ with a 60% duty cycle and were unsynchronized with ventilation. Previous studies have shown a 60% duty cycle to be associated with improved arterial blood pressure and blood flow.³⁴⁻³⁵ Chest compression was performed for 10 minutes (total time from induction of ventricular fibrillation was 16 minutes), after which chest compression was stopped.

Animals that remained in ventricular fibrillation received as many as three consecutive defibrillation attempts at 3, 4, and 6 J/kg body wt. Animals that had electromechanical dissociation or that failed to have return of spontaneous circulation after attempted defibrillation were given 0.05 mg/kg epinephrine through the distal port of the pulmonary artery catheter and ventilated with gas with an Fio₂ of 0.85; chest compression was resumed for 3 additional minutes. The dose of epinephrine used in this study is commonly used in laboratory models of cardiac arrest and results in maximum coronary perfusion pressure and return of spontaneous circulation in swine and canine models of CPR.²¹⁻²³,³³⁻³⁶⁻³⁹ Chest compression was then stopped, and animals in ventricular fibrillation again received as many as three defibrillation attempts at 3, 4, and 6 J/kg body wt, which concluded the resuscitation protocol.

Animals were evaluated to determine whether return of spontaneous circulation had occurred (Fig 1) and then were killed and assessed for the presence of a pneumothorax and location of catheters. Return of spontaneous circulation was defined prospectively as an aortic systolic blood pressure of >80 mm Hg sustained for 5 consecutive minutes. Arterial and mixed venous pH, PO₂, PCO₂, and HCO₃⁻ were measured immediately before ventricular fibrillation, after 5 minutes of untreated ventricular fibrillation, and after 15 minutes of total experimental time (6 minutes of untreated ventricular fibrillation plus 9 minutes of chest compression with or without ventilation). Exhaled tidal volume caused by chest compressions was measured continuously in both groups, and exhaled tidal volume caused by mechanical ventilation was measured continuously in the ventilated group.

Data Analysis

Group return of spontaneous circulation rates were compared with Fisher’s exact test. Values for arterial and mixed venous blood gases, hemodynamic, and ventilation data are given as mean (±SD) and were compared with the Mann-Whitney U test; coronary perfusion pressure was compared with repeated-measures ANOVA and Schéffe’s multiple-comparison procedure; and α was set at .05 for statistical significance.

Results

Except for coronary perfusion pressure, there were no significant differences between the two groups in baseline hemodynamic and blood gas values measured before induction of ventricular fibrillation (Table 1). After 5 minutes of untreated ventricular fibrillation, arterial PO₂ was significantly lower in the nonventilated group (Table 2). However, after 9 minutes of chest compression, significant differences were noted with arterial and mixed venous blood gases between the ventilated and nonventilated groups (Table 3). When compared with the ventilated group, the nonventilated group had significantly greater arterial acidemia (pH 7.29±0.06 U compared with 7.14±0.11 U), hypoxia (PO₂ 216±104 mm Hg compared with 38±17 mm Hg), and hypercarbia (PCO₂ 35±8 mm Hg compared with 62±16 mm Hg) and mixed venous acidemia (pH 7.15±0.04 U).
compared with 7.07±0.09 U), hypoxia (\(\text{PO}_2\) 32±7 mm Hg compared with 15±7 mm Hg), and hypercarbia (\(\text{PCO}_2\) 60±7 mm Hg compared with 74±13 mm Hg). The ventilated group had a significantly lower arterial bicarbonate concentration (\(\text{HCO}_3^-\) 17±2 mEq/L compared with 21±3 mEq/L), but the mixed venous bicarbonate concentration was similar in both groups (\(\text{HCO}_3^-\) 21±2 mEq/L compared with 21±2 mEq/L).

Although the nonventilated group received no mechanical ventilation during the first 10 minutes of chest compression, the group did have substantial ventilation produced by chest compressions alone (mean minute ventilation was 3.8±1.5 L/min). The ventilated group received mechanical ventilation (mechanical mean minute ventilation was 4.8±1.6 L/min) in addition to ventilation produced by chest compressions (mean minute ventilation of 2.3±1.3 L/min). During the first 10 minutes of chest compression, the mean total minute ventilation was 7.1±1.8 L/min and 3.8±1.5 L/min (\(P<.01\)) for the ventilated and nonventilated group, respectively.

For the first 10 minutes of chest compression, mean coronary perfusion pressure was 16±10 mm Hg for the ventilated group and 19±14 mm Hg for the nonventilated group (\(P=.46\)). For the ventilated group, coronary perfusion pressure increased significantly after epinephrine when compared with coronary perfusion pressure immediately before administration of epinephrine.

### Table 1. Baseline Values Before Cardiac Arrest

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ventilated Group (n=12)</th>
<th>Nonventilated Group (n=12)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight, kg</td>
<td>38±10</td>
<td>34±6</td>
<td>.26</td>
</tr>
<tr>
<td>Temperature, °C</td>
<td>38.8±1.1</td>
<td>39.1±0.8</td>
<td>.62</td>
</tr>
<tr>
<td>(V_c), mL·min(^{-1}·kg(^{-1})</td>
<td>132±17</td>
<td>143±23</td>
<td>.68</td>
</tr>
<tr>
<td>CI, mL·min(^{-1}·kg(^{-1})</td>
<td>128±23</td>
<td>124±43</td>
<td>.86</td>
</tr>
<tr>
<td>CPP, mm Hg</td>
<td>98±18</td>
<td>116±21</td>
<td>.02</td>
</tr>
<tr>
<td>Arterial blood</td>
<td>7.44±0.05</td>
<td>7.46±0.03</td>
<td>.24</td>
</tr>
<tr>
<td>(\text{PO}_2), mm Hg</td>
<td>76±10</td>
<td>74±16</td>
<td>.98</td>
</tr>
<tr>
<td>(\text{PCO}_2), mm Hg</td>
<td>38±2</td>
<td>39±4</td>
<td>.43</td>
</tr>
<tr>
<td>(\text{HCO}_3^-), mEq/L</td>
<td>26±2</td>
<td>27±2</td>
<td>.09</td>
</tr>
<tr>
<td>Mixed venous blood</td>
<td>7.40±0.04</td>
<td>7.42±0.03</td>
<td>.42</td>
</tr>
<tr>
<td>(\text{PO}_2), mm Hg</td>
<td>39±5</td>
<td>39±9</td>
<td>.77</td>
</tr>
<tr>
<td>(\text{PCO}_2), mm Hg</td>
<td>43±3</td>
<td>42±3</td>
<td>.45</td>
</tr>
<tr>
<td>(\text{HCO}_3^-), mEq/L</td>
<td>27±2</td>
<td>28±2</td>
<td>.71</td>
</tr>
</tbody>
</table>

\(V_c\) indicates minute ventilation given by a mechanical time-cycled volume-controlled ventilator; CI, cardiac index; CPP, coronary perfusion pressure; \(\text{PO}_2\), partial pressure of oxygen; \(\text{PCO}_2\), partial pressure of carbon dioxide; \(\text{HCO}_3^-\), bicarbonate concentration. Group values (mean±SD) were compared with the Mann-Whitney U test.

### Table 2. Arterial and Mixed Venous Blood Gas Measurements After 5 Minutes of Untreated Ventricular Fibrillation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Ventilated Group (n=12)</th>
<th>Nonventilated Group (n=12)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood</td>
<td>7.49±0.20</td>
<td>7.45±0.13</td>
<td>.77</td>
</tr>
<tr>
<td>(\text{PO}_2), mm Hg</td>
<td>76±19</td>
<td>59±17</td>
<td>.04</td>
</tr>
<tr>
<td>(\text{PCO}_2), mm Hg</td>
<td>35±13</td>
<td>39±10</td>
<td>.49</td>
</tr>
<tr>
<td>(\text{HCO}_3^-), mEq/L</td>
<td>25±2</td>
<td>26±2</td>
<td>.13</td>
</tr>
<tr>
<td>Mixed venous blood</td>
<td>7.30±0.06</td>
<td>7.36±0.13</td>
<td>.20</td>
</tr>
<tr>
<td>(\text{PO}_2), mm Hg</td>
<td>37±10</td>
<td>36±12</td>
<td>.47</td>
</tr>
<tr>
<td>(\text{PCO}_2), mm Hg</td>
<td>51±8</td>
<td>48±15</td>
<td>.47</td>
</tr>
<tr>
<td>(\text{HCO}_3^-), mEq/L</td>
<td>25±2</td>
<td>26±2</td>
<td>.91</td>
</tr>
</tbody>
</table>

\(\text{PO}_2\) indicates partial pressure of oxygen; \(\text{PCO}_2\), partial pressure of carbon dioxide; \(\text{HCO}_3^-\), bicarbonate concentration. Group values (mean±SD) were compared with the Mann-Whitney U test.
TABLE 3. Arterial and Mixed Venous Blood Gas Measurements After 9 Minutes of Chest Compression With and Without Ventilation

| Variable | Ventilated Group (n=12) | Nonventilated Group (n=12) | P  
|----------|------------------------|---------------------------|---
| Arterial blood | | | |
| pH       | 7.29±0.06              | 7.14±0.1                  | .002 |
| PO₂, mm Hg | 216±104               | 38±17                     | .0002 |
| PCO₂, mm Hg | 35±8                  | 62±16                     | .0003 |
| HCO₃⁻, mEq/L | 17±2                  | 21±3                      | .004 |
| Mixed venous blood | | | |
| pH       | 7.15±0.04              | 7.07±0.09                 | .04 |
| PO₂, mm Hg | 32±8                  | 15±7                      | .0002 |
| PCO₂, mm Hg | 60±7                  | 74±13                     | .009 |
| HCO₃⁻, mEq/L | 21±2                  | 21±2                      | .45 |

PO₂ indicates partial pressure of oxygen; PCO₂, partial pressure of carbon dioxide; and HCO₃⁻, bicarbonate concentration. Group values (mean±SD) were compared with the Mann-Whitney U test.

However, for the nonventilated group, coronary perfusion pressure did not increase significantly following epinephrine (P=.4) (Fig 2).

The cardiac rhythms after the first and second sets of defibrillation attempts are shown in Fig 3. Nine of 12 (75%) of the ventilated animals and only 1 of 12 (8%) of the nonventilated animals had return of spontaneous circulation after cardiac arrest (P<.002).

Discussion

The necessity of ventilation during CPR is being studied and debated. An expert task force on the future of CPR suggested that ventilation may be unnecessary during the first few minutes of CPR, based on the premise that advanced cardiac life support personnel will arrive quickly. Some investigators have suggested that the standard order of resuscitation “ABC” (airway, breathing, and circulation) could be changed to “CAB” and ventilation delayed for 30 seconds because the reservoir of oxygenated blood present in the large arteries and heart could be brought into circulation with chest compressions alone. In addition, a CPR technique without ventilation would be easier for lay rescuers to master. With the public’s concern about transmission of infectious disease during mouth-to-mouth ventilation, it seems reasonable to assess the importance of ventilation during cardiac arrest. The present study showed that ventilation during 10 minutes of chest compression after 6 minutes of untreated ventricular fibrillation was associated with a significantly higher rate of resuscitation compared with a nonventilated group. These findings suggest that ventilation may be an important component of human CPR and successful treatment of cardiac arrest, especially if advanced cardiac life support is delayed by more than 6 minutes. Although a 6-minute interval of untreated ventricular fibrillation was needed to demonstrate the importance of ventilation in swine, this interval may not be as long in human cardiac arrest.

In contrast with our results, a similar study found no difference in rates of return of spontaneous circulation in ventilated and nonventilated swine receiving chest compressions. In that study, all animals that received chest compression survived regardless of whether they were ventilated. However, there are a number of substantial differences in the methods used in that study compared with the present study that could account for important differences in results. In their study, ventilation with 100% O₂ was given before induction of ventricular fibrillation, there was a short duration of untreated ventricular fibrillation (30 seconds), and they allowed spontaneous ventilation, which was unmeasured. All of these factors favored improved outcome in the experimental nonventilated group. The model used in the present study had a prolonged duration of ventricular fibrillation (6 minutes), used room air for ventilation before inducing ventricular fibrillation, and prevented spontaneous ventilation with pancuronium during cardiac arrest. All of these factors would be expected to decrease the rate of survival, especially in the nonventilated group.
Ventilation with room air before cardiac arrest and inhibition of spontaneous ventilation during cardiac arrest are appropriate for most basic life support models of cardiac arrest. Although agonal respiration is known to occur during human cardiac arrest and CPR, it is unpredictable and does not provide a reliable source of ventilation since upper airway obstruction is common.41-46 In contrast, spontaneous ventilation occurs frequently in unparalyzed swine during CPR, and the rate and depth of gasping have been shown to correlate with coronary perfusion pressure and rate of return of spontaneous circulation.47 Because all of the animals in the present study were intubated, our model does not simulate bystander CPR. Although an endotracheal tube could have impaired gas exchange by increasing dead space by a small amount, it also maintained an open airway in the nonventilated animals and permitted a small but measurable minute ventilation caused by compressions of the sternum to occur. Thus, gas exchange in the nonventilated group was more likely to be enhanced than impaired by intubation.

A multicenter study of time to therapeutic interventions in witnessed cardiac arrest found that the mean interval from collapse of the victim to the start of bystander CPR was 4 to 5 minutes and the mean interval from collapse to intubation was 17 minutes.28 Although our model did not attempt to simulate either basic or advanced life support CPR, the nonintervention interval of 6 minutes used in this study is similar to the interval of cardiac arrest before CPR that occurs in human cardiac arrest. In addition, in our model the interval from onset of ventricular fibrillation to ventilation with intubation in the nonventilated group was 17 minutes, which is also similar to reported out-of-hospital treatment intervals.

What is the mechanism accounting for the difference in the rate of return of spontaneous circulation between the ventilated and nonventilated groups? Our model was designed to control for and to measure ventilation continuously. Although we found substantial minute ventilation caused by chest compression alone, it was insufficient for effective gas exchange. Minute ventilation and gas exchange were significantly greater in the group that received mechanical ventilation. Coronary perfusion pressure is known to be one of the most important determinants of successful resuscitation from cardiac arrest.48-50 Yet, in the present study, the two groups had similar mean coronary perfusion pressures sufficient to permit successful resuscitation, and all other interventions were identical except for ventilation. Although coronary perfusion pressure during the first 10 minutes of chest compression was similar for the two groups, the response of coronary perfusion pressure to epinephrine was significantly greater in the ventilated animals than in the nonventilated animals (P<.01) (Fig 2). The decreased return of spontaneous circulation rate in the nonventilated group may be due to less coronary blood flow caused by peripheral vasodilation associated with increased levels of hypercarbic acidosis and a blunted vasoconstriction response to exogenous epinephrine, as seen in the nonventilated group.51-56 Although many studies have shown a relationship between coronary perfusion pressure and return of spontaneous circulation, ventilation may play an important role in regulating coronary perfusion pressure and return of spontaneous circulation through the vascular response to catecholamines.

The present study was not designed to distinguish independently the effects of hypoxia, hypercarbic acidosis, and acidemia, all of which may influence the efficacy of drugs and defibrillation used for resuscitation. Because all three were favorably affected by ventilation, any combination of them may be important for improved outcome. A study of human resuscitation with uncontrolled ventilation demonstrated greater return of spontaneous circulation in patients with better central venous PO2, PCO2, and pH,10 as seen in our study. That study also did not distinguish between the effects of each of these factors on return of spontaneous circulation. Another study demonstrated that hypoxia and hypercarbia profusely decreased the myocardial force of contraction.22 Other studies of the effects of pH, PCO2, and PO2 on the success of defibrillation have reported results similar to ours: animals with greater hypercarbic acidosis or hypoxia required the highest dose of electrical energy for defibrillation and more frequently failed resuscitation.13,15,21

In the present study, although the ventilated group had lower arterial pH and bicarbonate concentration than the nonventilated group, this did not have an adverse effect on resuscitation. Improvement of metabolic acidosis with sodium bicarbonate has not yet been shown to improve human resuscitation,57 and most studies have failed to show any improvement in outcome for animals treated with bicarbonate.58-60 Therefore, differences in return of spontaneous circulation may be due to differences in hypoxia and/or hypercarbic acidosis but seem unlikely to be due to metabolic acidosis. Studies designed to clarify the independent effects of hypoxia and hypercarbic acidosis on outcome from cardiac arrest are being performed in our laboratory.67

Acknowledgments

This work was supported by grant 91GIA/721 from the American Heart Association, Florida Affiliate. The authors wish to thank Michael J. Banner, PhD; Joachim S. Gravenstein, MD; Nikolaus Gravenstein, MD; and Kathryn Rosemeier, PharmD; for their ideas, support, and encouragement. We would also like to thank Susan Lorash and Suzanne White for their skillful editorial assistance in preparing this manuscript and Diane Pettry for graphics.

References


Effect of ventilation on resuscitation in an animal model of cardiac arrest.

Circulation. 1994;90:3063-3069
doi: 10.1161/01.CIR.90.6.3063
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1994 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circ.ahajournals.org/content/90/6/3063

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published
in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial
Office. Once the online version of the published article for which permission is being requested is located,
click Request Permissions in the middle column of the Web page under Services. Further information about
this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/