The Composition of Gas Given by Mouth-to-Mouth Ventilation During CPR*

Volkert Wenzel; Ahamed H. Idris, MD; Michael J. Banner, PhD; Ronnie S. Fuerst, MD; and Kelly J. Tucker, MD

Study objective: To compare the concentration of a rescuer’s exhaled O2 and CO2 during mouth-to-mouth ventilation with or without chest compression.

Design: Prospective repeated measures study. Simulated one- and two-rescuer cardiopulmonary resuscitation (CPR) was performed as recommended by the American Heart Association.

Setting: University laboratory.

Participants: Fifty-five healthcare professionals certified in basic and advanced cardiac life support volunteered as rescuers in this study.

Measurements and results: Thirty-three volunteers performed one-rescuer CPR, and 22 volunteers performed two-rescuer CPR. Minute ventilation for both groups increased 50% to 130% during CPR (p<0.05). During the performance of CPR, the concentration of exhaled O2 increased from 16.4 ± 0.7% to 16.9 ± 0.5% in the one-rescuer CPR group and from 16.5 ± 0.9% to 17.8 ± 0.6% in the two-rescuer CPR group (p<0.05). The concentration of exhaled CO2 in the one-rescuer CPR group did not change significantly throughout the entire experiment, but decreased in the two-rescuer CPR group from a baseline measurement of 4.0 ± 0.4% to 3.5 ± 0.4% (p<0.05). During CPR, the concentration of exhaled CO2 was 4.0 ± 0.4% in the one-rescuer CPR group compared with 3.5 ± 0.4% in the two-rescuer CPR group (p<0.05).

Conclusions: The gas given by mouth-to-mouth ventilation is a hypercarbic and hypoxic mixture compared with room air. Mouth-to-mouth ventilation is the only circumstance in which a hypercarbic and hypoxic gas is given as therapy. Further laboratory and clinical studies are necessary to determine the effect of mouth-to-mouth ventilation during CPR. (Chest 1994; 106:1806-10)

CPR=cardiopulmonary resuscitation

Key words: cardiac arrest; CO2; heart-arrest-therapy; respiration-artificial; resuscitation

The value of early mouth-to-mouth ventilation during cardiopulmonary resuscitation (CPR) has been well accepted since the early 1960s, but is currently being reassessed.1 Recent studies in laboratory models of CPR have shown no clear benefit to early ventilation in cardiac arrest.2-4 We have recently completed studies in a swine model showing that inhalation of a gas mixture containing 5% CO2 and 95% O2 has an adverse effect on resuscitation from cardiac arrest.5 We have also shown that CO2 has striking negative inotropic and chronotropic effects on isolated myocytes.6

The composition of gas exhaled by a person giving mouth-to-mouth ventilation has been studied in human models of respiratory arrest,7-10 but not during CPR. The purpose of the present study was to compare the concentration of a rescuer’s exhaled O2 and CO2 during mouth-to-mouth ventilation with or without chest compression.

METHODS AND MATERIALS

The experimental protocol of this study was approved by the institutional review board of our institution. Fifty-five health care professionals (nurses, paramedics, medical students, physicians) certified in basic life support volunteered as rescuers in this study and gave informed consent to participate.

The exhaled gas of a volunteer was collected by having the person exhale into a mouthpiece placed on an airway filter (Respirgard II, Bacterial/Viral filters, Marquest Medical Products Inc, Englewood, Colo) to prevent transmission of infectious agents. The airway filter was connected to a one-way valve to ensure that only exhaled gas was collected, and this was attached to a spirometer with a three-way piece (Haloscale MX, Wright Respiriometer In-line model, Ohio Medical Products, Madison, Wis) to measure minute ventilation. The infusion line of an intravenous catheter was attached to a stopcock placed in a cork that sealed one three-way-piece outlet. This equipment was connected to a Douglas-type gas collection bag (30 L maximum capacity; Warren E. Collins Inc, Braintree, Mass) to store the exhaled gas. This setup caused a gas flow resistance of 5 cm H2O/L/s, which approximates the resistance of a normal human airway.

Before measurement of baseline values, an approximate upper limit of normal minute ventilation was calculated for each

*From the Departments of Anesthesiology (Dr. Banner), Medicine (Drs. Idris and Tucker), Pediatrics (Dr. Fuerst), Physiology (Dr. Banner), and Surgery (Division of Emergency Medicine [Drs. Wenzel, Idris, and Fuerst]), University of Florida College of Medicine, Gainesville.


Manuscript received March 24, 1994; revision accepted June 2.
volunteer using the following formula: weight (kg) × 15 (mL tidal volume) × 12 (ventilation rate/min). The volunteers were asked to maintain their ventilation within the estimated minute ventilation limit. A noseclip was used so that all exhaled gas entered the collection bag. One investigator observed ventilation and coached the volunteers continuously during baseline measurements to prevent hyperventilation. The use of a noseclip, irritation of the buccal mucosa, and a decrease in air-flow resistance caused by breathing through the mouthpiece may induce changes in breathing patterns.11,12 The volunteers sat in a chair and were instructed to inhale room air through the mouth and to exhale into the gas-collecting equipment for 2.5 min as described above.

After baseline measurements were completed, the volunteers were assigned to a group that performed either one-rescuer CPR (n=33) or two-rescuer CPR (n=22), both following the guidelines of the American Heart Association.10 The volunteers in the one-rescuer CPR group performed simulated CPR for 5 min using a CPR manikin (Skillmeter Resusci-Anne, Laerdal Medical Corporation, Armonk, NY). However, instead of ventilating the manikin, the rescuers exhaled into the gas collection equipment, which was attached near the manikin’s head. Chest compressions were administered at a minimum rate of 60/min, with a depth of 3.75 to 5 cm and a force of 27 to 36 kg, which was displayed on a control unit. Ventilation during two-rescuer CPR was simulated without performing chest compressions. After 2.5 min had elapsed, the collection bag was changed to collect the exhaled gas during the next 2.5 min separately. Immediately after completion of simulated CPR, the collected gas was measured in a gas analyzer (model 1312, Instrumentation Laboratory, Lexington, Mass).

**Data Analysis**

Baseline spirometry data, chest compression rate, depth, and force, and the collected mouth-to-mouth gas composition values are expressed as mean (±SD). Comparisons were made with one-factor and repeated-measures analysis of variance and with Scheffe’s multiple comparison procedure; alpha was set at 0.05 for statistical significance.

**RESULTS**

Thirty-three volunteers (13 women [40%]), 20 men [60%] performed one-rescuer CPR, and 22 volunteers (9 women [41%], 13 men [59%]) performed two-rescuer CPR. When compared with the baseline minute ventilation of the volunteers measured at rest, applied mouth-to-mouth minute ventilation for both groups increased significantly during CPR (Fig 1). Minute ventilation was significantly greater for the two-rescuer CPR group during both the first CPR interval (12.1 ± 0.83 compared with 9.8 ± 1.1 L/min) and the second CPR interval (11.6 ± 0.95 compared with 9.9 ± 1.1 L/min).

**FIGURE 1.** Mean (±SD) minute ventilation before and during simulated CPR with mouth-to-mouth ventilation.

**FIGURE 2.** Mean (±SD) fraction of exhaled oxygen concentration before and during simulated CPR with mouth-to-mouth ventilation.
The concentration of exhaled $O_2$ for both groups increased significantly from baseline during the performance of CPR (Fig 2). During CPR, the concentration of exhaled $O_2$ was consistently higher in the two-rescuer CPR group ($p<0.05$).

The concentration of exhaled $CO_2$ in the one-rescuer CPR group did not change significantly throughout the entire experiment (Fig 3). The concentration of exhaled $CO_2$ in the two-rescuer CPR group decreased from a baseline measurement of $4.0 \pm 0.6\%$ to $3.7 \pm 0.4\%$ for the first CPR interval and $3.5 \pm 0.4\%$ for the second ($p<0.05$). During CPR, the concentration of exhaled $CO_2$ was significantly higher in the one-rescuer CPR group than in the two-rescuer CPR group.

The concentration of $O_2$ in exhaled gas is significantly lower than that in room air (20.9%) and the concentration of $CO_2$ in exhaled gas is significantly higher than that in room air (0.03%).

**DISCUSSION**

We found that exhaled gas given by mouth-to-mouth ventilation during one- or two-rescuer CPR had a significantly higher concentration of $CO_2$ and a significantly lower concentration of $O_2$ than room air. The one-rescuer CPR group exhaled more $CO_2$ and less $O_2$ than the two-rescuer CPR group. This may have been because the applied mouth-to-mouth minute ventilation of the rescuers in the two-rescuer CPR group was greater than that of the one-rescuer CPR group during CPR, resulting in greater dilution of exhaled gas with fresh air. Another explanation may be increased metabolism in the one-rescuer CPR group caused by the work of performing chest compression. The volunteers in the one-rescuer CPR group compressed the manikin $358 \pm 39$ times in 5 min (range, 301 to 475), while the rescuers in the two-rescuer CPR group did not perform chest compression at all.

Although the volunteers in the one-rescuer CPR group were experienced health care professionals, it was difficult for them to achieve a chest compression rate of 80 to 100/min as recommended by the American Heart Association. Despite continuous coaching of the rescuers during CPR, the mean chest compression rate was only $72 \pm 8$/min (range, 60 to 95). Only 15% of the volunteers achieved a rate of 80 to 100/min.

Early examples of mouth-to-mouth ventilation are described throughout history. References to mouth-to-mouth ventilation have been found in the Bible, in a description of the resuscitation of a coal miner in 1744, and in an experiment in 1796 demonstrating that exhaled gas was safe for breathing. In 1954, Elam and colleagues described artificial respiration with the exhaled gas of a resucer using a mouth-to-mask ventilation method.

In the 1950s and early 1960s, other alternative methods of ventilation were investigated in addition to mouth-to-mouth ventilation. A number of studies showed that the application of external pressure to the chest during manual maneuvers in normal volunteers caused substantial tidal volumes that ranged from 50 to 1,114 mL. These studies also noted that tidal volumes generated by actively expanding the chest with arm-lift or hip-lift techniques were 20 to 40% greater when compared with passive chest expansion. Techniques that relied exclusively on passive chest expansion were ineffective for adequate ventilation and resulted in a mean arterial $O_2$ saturation of 67% in normal subjects. Manual techniques that included active chest expansion produced a mean arterial $O_2$ saturation of 93% in human subjects, and thus were able to maintain acceptable gas exchange without positive pressure ventilation. In contrast to chest pressure manual techniques, pressure applied directly over the sternum of curarized, intubated volunteers produced a mean tidal volume...
of only 156 mL. Furthermore, without intubation, sternal pressure produced no tidal exchange because of airway obstruction by the tongue.  

During the time that ventilation techniques were being studied, mouth-to-mouth ventilation was also studied extensively. Several research projects were funded by the US Department of the Army because of the problem of resuscitation for victims of nerve gas poisoning. In one study, 29 volunteers were paralyzed with curare and mouth-to-mask resuscitation was started before the onset of cyanosis. The arterial O\textsubscript{2} saturation of the volunteers receiving mouth-to-mouth ventilation was never below 85% and the mean O\textsubscript{2} saturation was 94%. Alveolar CO\textsubscript{2} tension, measured in 21 patients, was maintained at or below 50 mm Hg. The mean alveolar CO\textsubscript{2} concentration was 5.6% before resuscitation and 3.9% during resuscitation. Expired gas resuscitation produced a decrease in alveolar CO\textsubscript{2} concentration in 12 patients. The authors concluded that with mild hyperventilation, the rescuer’s exhaled gas was readily converted to a suitable resuscitation gas. These experiments were designed to simulate a respiratory arrest and only healthy volunteers were studied. Other studies found similar results in models of respiratory arrest in which cardiac output was normal. Whether exhaled gas would benefit a patient who suffers cardiac arrest was not considered and has never been investigated (to our knowledge).

Since exhaled gas contains CO\textsubscript{2}, it may have adverse cardiovascular effects during CPR, but few investigations of this issue have been done. A study of the effect of ventilation on resuscitation during CPR using an animal model showed that both hypoxia and hypercarbia independently have an adverse effect on outcome from cardiac arrest. Swine were ventilated with experimental gas mixtures consisting of 85% O\textsubscript{2} in a control group, 95% O\textsubscript{2} and 5% CO\textsubscript{2} in a hypercarbic group, and 10% O\textsubscript{2} and 90% N\textsubscript{2} in a hypoxic group. The study succeeded in producing isolated hypoxia without hypercarbia and isolated hypercarbia without hypoxia. Only one of six animals could be resuscitated in both of the hypercarbic and hypoxic groups, while seven of eight were resuscitated in the control group. The present study shows that the rescuers in both groups exhaled a concentration of CO\textsubscript{2} that was similar to the concentration shown to be lethal during CPR in the above-cited study. Other studies of the effects of hypercarbia on the myocardium during CPR have shown that hypercarbia raises the threshold of defibrillation and decreases the force of myocardial contraction.

A study of the isolated effect of CO\textsubscript{2} on spontaneously contracting isolated chick myocytes showed that myocytes perfused with 4.6% and 9.6% CO\textsubscript{2} had inhibition of both rate and force of contraction. The model demonstrated a rapid and profound effect of CO\textsubscript{2} independent of pH, PO\textsubscript{2}, vascular tone, neuroendocrine factors, or inflammatory mediators.

Future studies are needed to address whether mouth-to-mouth ventilation is any better than chest compression without ventilation during cardiac arrest. The cardiovascular effects of mouth-to-mouth ventilation during CPR should be investigated to determine if there are adverse effects of this form of ventilation during low blood flow states. Manual techniques of ventilation have a number of advantages over mouth-to-mouth ventilation, including safety from transmission of infectious diseases and a superior ventilation gas (i.e., room air). If obstruction of the airway could be prevented, manual ventilation could be a useful alternative and should be studied. Chest compression alone can provide some ventilation, and recent studies in our laboratory show that tidal volume and minute ventilation are enhanced by active chest compression-decompression, a new method of CPR.

**Conclusion**

The gas given by mouth-to-mouth ventilation contains 3.5 to 4% CO\textsubscript{2} and 16.4 to 17.8% O\textsubscript{2}, a hypercarbic and hypoxic gas mixture. Mouth-to-mouth ventilation is the only circumstance in which a hypercarbic and hypoxic gas is given as therapy. Mouth-to-mouth ventilation may contribute to hypercarbic acidosis and possibly to an adverse outcome in a victim of cardiopulmonary arrest, regardless of the number of rescuers. Further laboratory and clinical studies are necessary to determine the exact effect of mouth-to-mouth ventilation during CPR.

**Acknowledgments:** The authors wish to thank Richard J. Melker MD, PhD, Lance B. Becker, MD, Kathryn A. Rosemeyer, PharmD, and David J. Orban, MD, for their ideas, support, and encouragement. We would also like to thank Susan Lorash and Suzanne White for their skillful editorial assistance in preparing the manuscript. Diane Pethey for graphics, and the volunteers of the University of Florida Health Sciences Center, who donated their time and effort to make this study possible.

**References**


CHEST / 106 / 6 / DECEMBER, 1994 1809
cardiomyocyte contractions by carbon dioxide. Circulation. 1993; 88(suppl):I-225


8 Elam JO, Greene DC, Brown ES, Clements JA. Oxygen and carbon dioxide exchange and energy cost of expired air resuscitation. JAMA 1958; 167:328-34


13 Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care: II. adult basic life support. JAMA 1992; 268:2184-98


24 Safar P. Failure of manual respiration. J Appl Physiol 1959; 14:54-8


