Bystander Cardiopulmonary Resuscitation

Is Ventilation Necessary?

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Background. **Prompt initiation of bystander cardiopulmonary resuscitation (CPR) improves survival. Basic life support with mouth-to-mouth ventilation and chest compressions is intimidating, difficult to remember, and difficult to perform. Chest compressions alone can be easily taught, easily remembered, easily performed, adequately taught by dispatcher-delivered telephone instruction, and more readily accepted by the public. The principal objective of this study was to evaluate the need for ventilation during CPR in a clinically relevant swine model of prehospital witnessed cardiac arrest.**

Methods and Results. **Thirty seconds after ventricular fibrillation, swine were randomly assigned to 12 minutes of chest compressions plus mechanical ventilation (group A), chest compressions only (group B), or no CPR (group C). Standard advanced cardiac life support was then provided. Animals successfully resuscitated were supported for 2 hours in an intensive care setting, and then observed for 24 hours. All 16 swine in groups A and B were successfully resuscitated and neurologically normal at 24 hours, whereas only 2 of 8 group C animals survived for 24 hours ($P<.001$, Fisher's exact test). One of the 2 group C survivors was comatose and unresponsive.**

Conclusions. **In this swine model of witnessed prehospital cardiac arrest, the survival and neurological outcome data establish that prompt initiation of chest compressions alone appears to be as effective as chest compressions plus ventilation and that both techniques of bystander CPR markedly improve outcome compared with no bystander CPR. (*Circulation*. 1993;88[part 1]:1907-1915.)**

KEY WORDS • **cardiac arrest** • **ventilation** • **nervous system** • **cardiopulmonary resuscitation** • **fibrillation**

"Why is it every time I press on his chest he opens his eyes, and every time I stop to breathe for him he goes back to sleep?"

—Anonymous bystander during telephone-directed CPR

Most cardiac arrests occur outside the hospital in the presence of family, friends, or other bystanders.[1-5] Prompt initiation of effective cardiopulmonary resuscitation (CPR) by bystanders substantially improves the chances of surviving out-of-hospital cardiac arrest.[1,2,5-14]

Basic life support as recommended by the American Heart Association includes mouth-to-mouth ventilation and chest compressions.[1] This is a complex psychomotor task that may be intimidating, difficult to remember, and difficult to perform.[1,2,15-21] In addition, there are increasing concerns that citizens may withhold potentially lifesaving CPR because of perceived risks of contracting an infectious disease such as AIDS.[1,22]

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The principal objective of this study was to evaluate the need for ventilation during CPR in a clinically relevant swine model of prehospital witnessed cardiac arrest. Preliminary experimental data suggest that chest compressions alone result in physiological benefit comparable to chest compressions and ventilation.[23-25] Our hypothesis was that initial treatment of cardiac arrest by chest compressions, with or without ventilation, would result in comparable rates of successful resuscitation, 24-hour survival, and good 24-hour neurological outcome.

Methods

CPR Model

An important limitation of studies involving cardiac resuscitation has been the development of an adequate model to test the experimental question. Swine were used in the present study because they more closely reflect the human thoracic and coronary anatomy than do canine preparations.[26-29] To simulate bystander-initiated CPR in an out-of-hospital cardiac arrest, we used a short period of ventricular fibrillation (30 seconds) before initiating basic life support, followed by a relatively long period (12 minutes) of closed-chest compressions before defibrillation. When a patient collapses in an observed cardiac arrest, a short downtime follows as the observer realizes the gravity of the situation and calls for help in accordance with the 1992 adult basic life support guidelines.[1] The average paramedic response

times for out-of-hospital cardiac arrest ranged from 4 to 9 minutes in 16 different programs.[10] Time from collapse to defibrillation may be 2 to 8 minutes longer.[30,31] A 12-minute experimental CPR period is therefore realistic. Defibrillation after more than 8 to 12 minutes of cardiac arrest is less effective.[10-12,32] This model includes a short downtime and a CPR period that is short enough to permit excellent outcomes in some animals to determine the importance of standard CPR versus chest compressions alone versus no CPR while awaiting definitive advanced cardiac life support.

Preparation

Experimental protocols were approved by the institutional animal review board and followed the guidelines of the American Physiological Society. All experiments were performed on healthy domestic swine, ranging in weight from 14 to 20 kg. After an overnight fast, the pigs underwent mask induction of anesthesia with isoflurane followed by oral endotracheal intubation. They were mechanically ventilated with a volume-cycled Harvard ventilator connected to a mixture of 100% oxygen and titrated isoflurane (approximately 1%). The tidal volume was initially set at 15 mL/kg, ventilator rate at 16 breaths per minute, and ventilator settings adjusted to maintain end-tidal carbon dioxide between 30 and 40 mm Hg. After a surgical plane of anesthesia was obtained, a calibrated micromanometer-tipped catheter (Millar Instruments, Houston, Tex) was inserted into the femoral artery by cutdown technique. The catheter tip was advanced into the thoracic aorta. The right external jugular vein was then isolated by cutdown and cannulated with a 7F introducer sheath, and a 5F balloon-tipped pulmonary artery flotation catheter was placed in the main pulmonary artery under pressure tracing guidance. The left external jugular vein was then isolated by cutdown technique and cannulated with a 7F introducer sheath, and a 4F bipolar pacing catheter was advanced through the sheath into the right ventricle under electrocardiographic guidance. After ventricular fibrillation was induced, the pacing catheter was removed, and a calibrated micromanometer-tipped catheter (Millar Instruments) was advanced through the introducer into the right atrium.

Measurements

Right atrial and thoracic aortic pressure waveforms, ECG, and end-tidal carbon dioxide measurements were continuously monitored and recorded on a four-channel Gould ES 1000 recorder throughout the experiment until the 2-hour simulated intensive care unit (ICU) period ended. End-tidal carbon dioxide was measured with an infrared capnometer (model 47210A, Hewlett Packard, Waltham, Mass) through a sensor attached to the ventilator circuit at the end of the endotracheal tube. Coronary perfusion pressure was calculated by subtracting right atrial middiastolic pressure from simultaneous aortic middiastolic pressure at a single point during three consecutive compression-relaxation cycles. Arterial blood gas specimens were obtained from the thoracic aorta, and mixed venous blood gas specimens were obtained from the main pulmonary artery. Oxygen saturation, P_{CO_2}, P_{O_2}, and pH were measured directly with a blood gas analyzer (IL-1306 with model 482 co-oximeter, Instrumentation Laboratories, Lexington, Mass), and plasma bicarbonate concentrations were calculated.

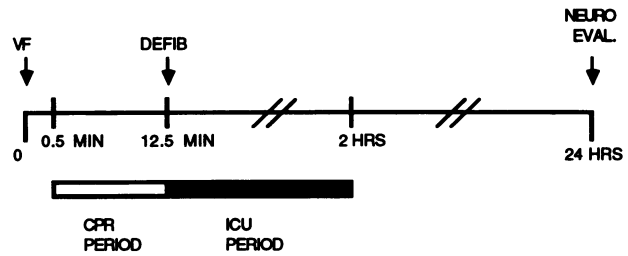

FIG 1. *Graphic representation of the experimental protocol. VF indicates ventricular fibrillation, induced at time 0; Defib, defibrillation attempt at the end of the CPR period; Neuro eval, neurological evaluation; CPR period, cardiopulmonary resuscitation period from 0.5 minutes after VF until 12.5 minutes after VF; ICU period, 2-hour intensive care unit period after defibrillation.*

Experimental Protocol

After completion of all baseline data acquisition, isoflurane was discontinued, and cardiac arrest was induced by applying a 60-cycle AC to the endocardium through the pacing electrode. Ventricular fibrillation was confirmed by the typical ECG rhythm and precipitous decrease in arterial pressure. Mechanical ventilation was discontinued after ventricular fibrillation was noted. After 30 seconds of ventricular fibrillation without intervention ("downtime"), a 12-minute "CPR" period followed. Animals were randomly assigned to one of three groups. For group A, 12 minutes of chest compressions and mechanical ventilation were provided in accordance with the American Heart Association basic life support standards. Chest compressions were delivered manually at 80 to 100 compressions per minute. Mechanical ventilation was administered at the rate and tidal volume that resulted in end-tidal carbon dioxide partial pressures between 30 and 40 mm Hg before ventricular fibrillation. The research technician providing the chest compressions was blinded to end-tidal carbon dioxide and vascular pressure measurements during CPR. Group B animals had their endotracheal tubes removed and then received 12 minutes of chest compressions only. Group C (control group) received no ventilation or compressions during the 12-minute basic life support period. See Fig 1.

At the end of the CPR period (12½ minutes after the induction of ventricular fibrillation), all animals received advanced cardiac life support beginning with defibrillation according to the American Heart Association algorithms for ventricular fibrillation.[2] For initial defibrillation, we used 50 J, followed by 100 J on the second attempt and 200 J on the third and any subsequent defibrillation attempts. Group B animals were reintubated during the minute immediately preceding the first defibrillation attempt, and ventilation was resumed after that attempt. In group C, ventilations and chest compressions (if necessary) were started after the first defibrillation attempt. After three unsuccessful attempts at defibrillation, intravenous epinephrine (0.02 mg/kg) was administered, CPR was continued for 30 to

60 seconds, and defibrillation was again attempted. If spontaneous circulation was not restored after the defibrillation attempt following the third epinephrine dose, resuscitative efforts were discontinued.

Restoration of spontaneous circulation was defined as unassisted pulse with a systolic arterial pressure of at least 50 mm Hg and pulse pressure of at least 20 mm Hg lasting for at least 1 minute.

Arterial and mixed venous blood gas specimens were obtained at baseline before ventricular fibrillation was induced, 10 minutes after ventricular fibrillation was induced, and 30 minutes after ventricular fibrillation was induced (after initial resuscitation).

Intensive Care

All animals with successful restoration of spontaneous circulation were supported for 2 hours in an ICU setting. Systolic blood pressure was maintained >80 mm Hg with dopamine and/or volume resuscitation as clinically indicated. All pigs surviving the intensive care period received 40 mL/kg of normal saline during the intensive care period. Ventricular dysrhythmias were treated with lidocaine as necessary. Mechanical ventilation was continued with 100% oxygen and adjusted to obtain an end-tidal carbon dioxide partial pressure of 30 to 40 mm Hg. Recurrent cardiac arrest was treated with standard CPR and advanced life support in accordance with the American Heart Association algorithms. At the end of 2 hours, all animals were weaned off pharmacological and ventilatory support. Before ventilatory support was discontinued, cutdown sites were anesthetized with 0.25% bupivicaine, and incisions were closed with silk sutures. Throughout the intensive care period, titrated doses of isoflurane were administered to maintain adequate analgesia and anesthesia.

Overnight Care

Animals surviving the intensive care period were transferred to overnight holding cages.

Neurological Evaluation

Neurological evaluation was performed at 4 and 24 hours after the initial cardiac arrest. To provide objective neurological evaluation, the canine neurological deficit score and cerebral performance categories developed at the University of Pittsburgh were adapted for use in swine, as previously reported.[33-35] Briefly, the neurological deficit score assigns values for deficits in neurological function so that a score of 0 is normal and a score of 400 is brain death. Cerebral performance category is a more global assessment of neurological function, with category 1 being normal and category 5 being brain death. After the 24-hour evaluation, survivors were killed by infusion of Euthanol.

Data Analysis

Systolic and diastolic aortic pressures, central venous pressure, and end-tidal carbon dioxide partial pressure were collected from the graphic records at (1) prearrest baseline, (2) during chest compressions at 4, 6, 8, 10, and 12 minutes after induction of ventricular fibrillation, and (3) after resuscitation at 15, 20, 30, 60, and 90 minutes after induction of ventricular fibrillation.

Comparisons of blood pressures, coronary perfusion pressures, end-tidal carbon dioxide partial pressures, swine neurological deficit scores, and swine cerebral performance categories among the three groups were evaluated by ANOVA. For all significant variables, differences between group means were evaluated with Scheffé's tests. During basic life support, comparisons of blood pressures, coronary perfusion pressures, and end-tidal carbon dioxide partial pressures between group A (chest compressions and mechanical ventilation) and group B (chest compressions alone) were evaluated by Student's t test. Continuous variables are described as mean±SD. A value of $P<.05$ was considered significant.

Comparisons of rate of restoration of spontaneous circulation, 2-hour ICU survival, 24-hour survival, epinephrine administration during immediate resuscitation, administration of lidocaine bolus, administration of lidocaine continuous infusion during the ICU period, and administration of dopamine continuous infusion during the ICU period between each group were evaluated by Fisher's exact test. Comparison of total joules administered during resuscitation among the three groups was analyzed by ANOVA. Differences between group means were evaluated by Scheffé's tests. Assuming 16 animals in groups A and B and 100% survival in group A, the experiment was designed to have a statistical power of 0.80 to detect a 30% difference in survival, with an α-error of 5%.

Results

All 16 swine in groups A and B attained restoration of spontaneous circulation and survived for 24 hours. In contrast, the outcome for group C (control group) was significantly worse than either group A or group B. All 8 animals in group C had restoration of spontaneous circulation, but 2 died during the ICU period. Only 2 of 8 animals survived for 24 hours ($P<.001$), and only one was neurologically normal; the other was comatose and unresponsive. The 95% confidence interval for 24-hour survival is 79% to 100% in groups A and B but only 0.3% to 53% in group C.

All animals in groups A and B were in cerebral performance category 1 (normal neurological examination) at 24 hours. Of the two 24-hour survivors in group C, the mean cerebral performance category was 2.5±2.1, which was significantly different from groups A and B ($P<.0001$). In addition, the mean neurological deficit score was 0±0 in group A and 1.9±7.5 in group B. Both differed significantly from the score of 123±173 in group C ($P<.0001$).

Baseline data before fibrillation did not differ significantly among the three groups (Table 1). Blood pressures obtained during CPR in groups A and B were generally comparable, although the aortic diastolic pressures at 4 and 6 minutes of CPR and the coronary perfusion pressure at 6 minutes were higher in group B. The coronary perfusion pressure for group A at 6 minutes was 23±4 mm Hg versus 27±8 mm Hg in group B ($P<.05$), a difference unlikely to be hemodynamically important.

After resuscitation, aortic and right atrial blood pressures, coronary perfusion pressures, and end-tidal carbon dioxide did not differ among the three groups. However, successfully resuscitated group C animals had

TABLE 1. Hemodynamics During Cardiopulmonary Resuscitation

	CPP, mm Hg		AoD, mm Hg		RAD, mm Hg	
Time, min	A	B	A	B	A	B
Base	56±7	54±6	60±7	59±7	4±4	5±4
4	22±4	26±11	29±4**	36±8**	7±3	11±7
6	23±4*	27±8*	29±4**	37±7**	6±4	9±4
8	21±5	24±9	28±4	32±9	8±3	9±5
10	21±4	24±9	28±5	33±10	7±4	10±4
12	27±25	27±13	29±6	36±13	9±3	11±4

Time indicates minutes after induction of ventricular fibrillation; CPP, coronary perfusion pressure; AoD, aortic diastolic pressure; RAD, right atrial diastolic pressure; A and B, groups A and B, respectively; Base, baseline, before ventricular fibrillation. Data are mean±SD.
*$P<.05$; **$P<.01$.

higher heart rates than either group A or group B throughout the ICU period (Table 2).

Difficulty with resuscitation and intensive care management was further estimated by comparing the three groups in terms of total joules required, need for epinephrine during initial resuscitation, administration of atropine or lidocaine boluses, and continuous infusions of dopamine or lidocaine (Table 3). Epinephrine was required after failure to respond to the initial three defibrillation attempts significantly more frequently in the control group C compared with groups A and B. There was a trend toward increased need of epinephrine in group B compared with group A ($P=.098$, Fisher's exact test). Group C also received significantly more joules than groups A or B. There were trends toward increased usage of lidocaine infusions and lidocaine boluses in the control group C compared with group A ($P<.10$).

Arterial and mixed venous P_{O_2} and oxygen saturation did not differ among the three groups at baseline and at 30 minutes after initial cardiac arrest (ie, approximately 15 minutes after resuscitation) (Tables 4 and 5). During CPR, the arterial P_{O_2} and oxygen saturation were higher in group A (compressions plus ventilation) than in group B (compressions only). At the same time, mixed venous P_{O_2} and oxygen saturation remained high in group C (no CPR) but were markedly reduced in both other groups ($P<.0001$).

Arterial and mixed venous pH, P_{CO_2}, and bicarbonate did not differ among groups before cardiac arrest (Tables 4 and 5, Fig 2). During the CPR period (10 minutes after fibrillation), arterial pH was higher and P_{CO_2} lower in group A (compressions plus ventilation) than the other two groups. However, group B (compressions only) had a lower mixed venous pH because of a higher mixed venous P_{CO_2} than the other two groups. In addition, the arterial bicarbonate in the control group C (no CPR) was higher than the bicarbonate in either experimental group (group C, 28±6 mmol/L versus group A, 23±4 mmol/L and group B, 24±4 mmol/L, $P=.02$). A few animals in each group had rare, episodic, agonal respiratory efforts, but none had rhythmic, sustained respirations.

Fifteen minutes after resuscitation, arterial and mixed venous pH was lower in control group C than the other groups ($P<.001$). This was primarily because of higher arterial and mixed venous P_{CO_2} in group C, although there was also a small decrease in arterial bicarbonate in this group. After resuscitation, there were no differences in arterial and mixed venous pH and P_{CO_2} between groups A and B.

Discussion

In this swine model of a witnessed prehospital cardiac arrest, the survival and neurological outcomes were the same when basic life support included prompt initiation

TABLE 2. Hemodynamics After Resuscitation

	AoS, mm Hg			AoD, mm Hg			RA, mm Hg			HR, bpm		
Time, min	A	B	C	A	B	C	A	B	C	A	B	C
Base	88±2	85±2	84±4	60±2	59±2	56±3	4±1	5±1	5±1	117±5	116±4	119±5
15	131±11	133±10	95±20	92±8	88±9	59±16	4±1	6±1	6±1	152±9	147±9	187
20	121±7	115±7	108±16	91±6	83±6	65±17	4±1	5±1	7±2	140±8	146±8	200±21*
30	95±3	90±5	111±19	71±3	65±5	79±14	3±1	5±1	5±1	138±8	147±7	190±19†
60	94±3	90±5	109±6	68±3	66±4	79±4	5±1	5±1	8±1	128±8	143±7	177±17‡
90	89±3	84±5	93±9	63±3	61±4	67±8	5±1	4±1	6±1	122±7	131±7	182±23*

AoS indicates aortic systolic pressure; AoD, aortic diastolic pressure; RA, right atrial pressure; HR, heart rate; bpm, beats per minute; A, B, and C, groups A, B, and C, respectively; Base, baseline.
*Group C different from groups A and B, $P<.01$.
†Group C different from groups A and B, $P<.05$.
‡Group C different from group A, $P<.05$.

TABLE 3. Interventions During Resuscitation and ICU Management

	Group A (n=16)	Group B (n=16)	Group C (n=8)
Total joules	103±126*	116±135*	713±169
Epinephrine	12.5%†	43.8%‡	100%
Dopamine	0%	25%	25%
Lidocaine drip	0%	6.3%	37.5%
Lidocaine bolus	0%	25%	37.5%
Atropine	0%	6.3%	0%

% refers to percent of animals in each group receiving therapy.
*Different from group C, $P<.001$.
†Different from group C, $P<.01$.
‡Different from group C, $P<.05$.

of chest compressions plus ventilation or chest compressions alone. If confirmed by additional studies, the clinical importance of these findings is clear: (1) people are often reluctant to provide mouth-to-mouth ventilation[1,2,22] and (2) chest compressions plus ventilation is a complex psychomotor task that is difficult to teach, learn, remember, and perform.[12,15-21]

A recent survey suggested that the vast majority of basic cardiac life support instructors in Virginia would withhold or hesitate to perform mouth-to-mouth resuscitation on most adult strangers.[22] Since committing oneself to a complex, fearful task requires more than a positive inclination on a survey, it is likely that even more of these survey respondents would actually withhold mouth-to-mouth resuscitation and chest compressions.

Dispatcher-delivered telephone instruction in CPR increases the rates of bystander CPR in cases of out-of-hospital cardiac arrest.[36] Chest compressions without ventilation are more amenable to dispatcher-delivered telephone instruction.[37] Provision of adequate chest compressions was comparable by simulated telephone-instructed volunteers and traditional basic cardiac life support–trained volunteers. However, adequate ventilations were provided 50% more often by traditional basic cardiac life support–trained volunteers than simulated telephone-instructed volunteers.

Few previous studies have compared chest compressions with and without ventilation. In a rat model of fibrillatory cardiac arrest, preliminary data suggest that arterial oxygenation, resuscitation rates, and survival were comparable after 5 minutes of precordial compressions alone versus compressions plus mechanical ventilation.[25] However, arterial P_{CO_2} was 36±5 mm Hg in the ventilated group versus 68±6 mm Hg in the nonventilated group. Airway patency apparently was ensured with an endotracheal tube in both groups. Preliminary data in a dog model of fibrillatory cardiac arrest suggest that chest compressions without additional ventilatory assistance provided adequate ventilation, arterial oxygenation, and arterial acid-base balance for >4 minutes.[24] Presence of an endotracheal tube during compressions permits more effective ventilation because it prevents airway obstruction by the larynx and pharyngeal soft tissues. In the present study, despite prior removal of the endotracheal tube, direct observations and blood gas analyses suggested that some ventilation had occurred in the chest compressions only group. Similarly, remarkable ventilation has been documented in four unintubated humans who received 1 minute of active compression-decompression CPR with a hand-held suction device and no other ventilatory support.[38]

In the present study, the outcomes for animals that received chest compressions with or without ventilation were excellent. Whereas only 1 of 8 control animals survived for 24 hours neurologically intact, all 32 animals in the two experimental groups were alive and

TABLE 4. Arterial Blood Gas Analyses

	P_{O_2}, mm Hg	S_{O_2}, %	P_{CO_2}, mm Hg	pH	HCO_3, mmol/L
Baseline					
Group A	196±97	96±2	42±5	7.45±0.05	30±2
Group B	204±79	97±1	43±5	7.44±0.05	29±1
Group C	137±72	95±2	40±4	7.46±0.02	29±3
CPR					
Group A	124±5*†	94±4*‡	29±8*‡	7.50±0.10*‡	23±4§‖
Group B	58±23	71±28	48±22	7.33±0.16	24±4§‖
Group C	74±34	81±20	45±12	7.39±0.10	28±6
After resuscitation					
Group A	208±77	97±2	43±6§‖	7.35±0.05‖†	24±3*§‖
Group B	215±76	97±1	44±5§‖	7.29±0.08‖†	21±3
Group C	173±48	97±1	52±10	7.19±0.11	20±1

Baseline refers to immediately before ventricular fibrillation; CPR, cardiopulmonary resuscitation 10 minutes after induction of ventricular fibrillation; after resuscitation, 20 minutes after induction of ventricular fibrillation.
*Different from group B.
†$P<.001$.
‡$P<.01$.
§$P<.05$.
‖Different from group C.

TABLE 5. Mixed Venous Blood Gas Analyses

	Po_2, mm Hg	So_2, %	Pco_2, mm Hg	pH	HCO_3, mmol/L
Baseline					
Group A	46±5	67±9	49±7	7.40±0.05	31±2
Group B	50±12	71±10	48±6	7.40±0.05	30±2
Group C	46±5	64±9	46±5	7.42±0.02	30±3
CPR					
Group A	21±4*†	17±7*†	54±11‡§	7.31±0.08‡§	27±3
Group B	22±6*†	19±17*†	71±16	7.20±0.11	27±2
Group C	47±20	47±10	58±11	7.28±0.07	26±6
After resuscitation					
Group A	52±6	70±9	49±6*§	7.31±0.05*†	25±3
Group B	53±8	67±9	52±7*§	7.25±0.08*†	23±3
Group C	60±8	71±14	61±4	7.11±0.03	24±7

Baseline refers to immediately before ventricular fibrillation; CPR, cardiopulmonary resuscitation 10 minutes after induction of ventricular fibrillation; after resuscitation, 20 minutes after induction of ventricular fibrillation.
*Different from group C.
†$P<.001$.
‡Different from group B.
§$P<.01$.

apparently neurologically normal at 24 hours. They were eating, drinking, alert, active, observant, distrusting swine that were rooting around and difficult to catch. To our examination, they did not differ from their littermates in the next cage who were awaiting their day of experimentation. Ease of resuscitation and subsequent ICU management were generally comparable for the chest compressions only and chest compressions plus ventilation groups. However, there was a trend toward greater need for epinephrine during initial resuscitation in the chest compressions only group.

Arterial blood gas measurements tend to remain stable during cardiac arrest without resuscitation.[39-42] Essentially, oxygen extraction and carbon dioxide production are minimal in large arteries, and the static blood in the large arteries is not exchanging oxygen or carbon dioxide in the lung or peripheral tissues to any great degree. Numerous investigators have demonstrated that mixed venous blood gas determinations more sensitively reflect inadequate perfusion in low-flow states than arterial determinations.[43-50] Mixed venous hypoxemia occurs because of increased oxygen

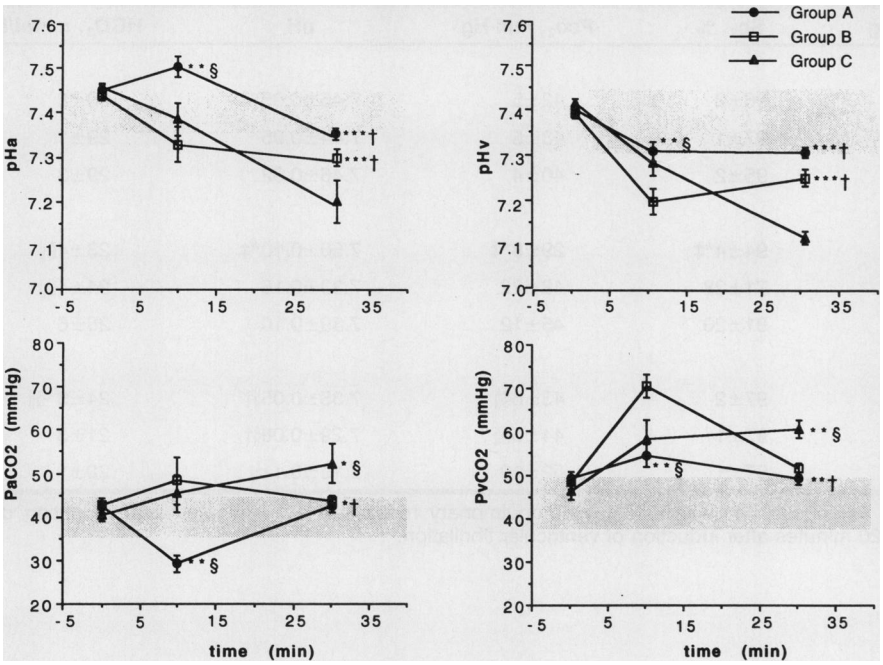

FIG 2. *Graphs showing arterial pH (pHa) and Pco_2 (PaCO2) and mixed venous pH (pHv) and Pco_2 (PvCO2) during the experiment. Time 0 is immediately before fibrillation, 10 minutes is during cardiopulmonary resuscitation, and 30 minutes is after resuscitation. §Different from group B, †different from group C, *P<.05, **P<.01, and ***P<.001. Shaded area indicates normal range.*

extraction during inadequate tissue oxygen delivery. Mixed venous hypercarbia occurs because of inadequate removal of carbon dioxide from the periphery and elimination through the lungs. Mixed venous acidosis results from mixed venous hypercarbia as well as lactic acidosis caused by anaerobic metabolism. Analysis of blood gas determinations in our control group is consistent with previous data: minimal changes 10 minutes after untreated cardiac arrest, followed by arterial and mixed venous acidosis and hypercarbia after successful restoration of spontaneous circulation. Ten minutes after cardiac arrest, both experimental groups developed mixed venous hypercarbia and acidosis. However, the arterial hypocarbia and alkalemia during CPR was demonstrable only in the ventilated animals and certainly contributed to the lesser mixed venous hypercarbia and acidemia in the ventilated group versus the chest compressions only group. The mixed venous-to-arterial P_{CO_2} difference is the same in the two experimental groups. Maintaining the prearrest minute ventilation during CPR (when pulmonary blood flow is diminished) resulted in relatively excessive removal of carbon dioxide per volume of blood. It is not clear whether the arterial alkalemia is potentially dangerous because of leftward shift of the oxyhemoglobin dissociation curve, increased arrythmogenicity potential, or decreased cerebral blood flow. Presumably, the pH is lower at the tissue level. Arterial and mixed venous oxygen concentrations 10 minutes after cardiac arrest were essentially unchanged in the control group but decreased in both experimental groups, especially the chest compressions only group. During chest compressions, circulation is reestablished, and oxygen consumption results in mixed venous and arterial hypoxemia. Lack of ventilation in the chest compressions only group probably led to greater decreases in functional residual capacity and more atelectatic areas, resulting in more intrapulmonary shunting and venous admixture. Within 20 minutes of defibrillation, there were no differences in arterial and mixed venous blood gas determinations between the two experimental groups.

There are several limitations to this study. By its very nature, this experiment could not be blinded. Nevertheless, the resuscitation and postresuscitation protocols were standardized and adhered to strictly. Chest compressions resulted in comparable coronary perfusion pressures in both experimental groups (Table 1). Most humans do not have fibrillatory cardiac arrests with normal coronary arteries. Myocardial blood flow during CPR is adversely affected by coronary artery lesions.[51] However, coronary artery pathology would not be expected to differentially influence the efficacy of chest compressions versus chest compressions plus ventilation. It is likely that myocardial oxygen delivery is even more dependent on coronary perfusion pressure than ventilation in the setting of coronary artery pathology. Alternatively, it is possible that the greater oxygen content of blood that results from ventilation during CPR could be important in the extremely low flow conditions that exist distal to coronary stenoses. Another potentially confounding aspect of the cardiac arrest model was the use of nearly 100% inspired oxygen before fibrillation. Whether provision of high oxygen concentrations differentially influenced the excellent outcomes in the unventilated versus ventilated group is not clear. High oxygen concentrations are commonly used in CPR studies.[25,26,41,42,51-57] The downtime of 30 seconds is shorter than most published prehospital cardiac arrest downtimes. However, prehospital cardiac resuscitation is provided to a wide spectrum of patients, including those with prolonged downtimes, inadequate basic life support, and dismal outcomes. We chose a best-case scenario of a witnessed arrest with prompt initiation of basic life support followed by an average paramedic response time to test our hypothesis in the prehospital circumstance most likely to be successful. We felt that using prolonged downtimes and prolonged basic life support would test our hypothesis only in a situation that is likely to be hopeless. On the other hand, the 12-minute CPR period was necessary to adequately test our hypothesis. If the CPR period were too brief, even the control group might have attained excellent outcomes.

A further limitation is that both groups received excellent CPR. It is highly unlikely that excellent chest compressions and mouth-to-mouth ventilation can be provided by a single rescuer in the field. Even during two-rescuer CPR, attention to ventilation may be counterproductive with respect to optimal chest compressions. The animals that were ventilated during basic life support in this study benefited from mechanical ventilation and optimal airway management, with an endotracheal tube placed before the cardiac arrest. Mouth-to-mouth resuscitation in a prehospital setting is unlikely to be nearly as controlled, effective, or safe. Rescue breathing before endotracheal intubation frequently causes gastric distension, which can lead to aspiration of gastric contents.[1]

Many clinical studies have documented benefit from bystander CPR.[5-9,11-14] However, the only two controlled animal experiments evaluating the effectiveness of bystander CPR in clinically relevant models did not demonstrate improved survival.[3,58] In a swine CPR model with a 2-minute fibrillatory downtime and 8 minutes of CPR, none of the animals were successfully resuscitated.[3] In a canine study with a 5-minute fibrillatory downtime and 5 minutes of CPR, most dogs in both groups were successfully resuscitated. However, simulated bystander CPR resulted in improved neurological outcome and greater ease of resuscitation.[58] In contrast, the present study is the first controlled animal experiment demonstrating that bystander CPR results in improved 24-hour survival and neurological outcome compared with the control group that did not receive bystander CPR. Mean coronary perfusion pressures were >25 mm Hg immediately before defibrillation in both bystander CPR groups in the present study but were approximately 1 mm Hg in the earlier swine experiment that failed to demonstrate any benefit from bystander CPR. Clearly, bystander CPR is beneficial only if the CPR provides adequate coronary perfusion.

In conclusion, prompt initiation of chest compressions alone, a much simpler technique of basic life support, was as effective as chest compressions plus ventilatory assistance in this swine model of bystander CPR. Standard basic life support is a complex, intimidating task that is difficult to teach, learn, remember, and perform. If chest compressions alone were similarly

effective during initial resuscitation for human cardiac arrests, the clinical implications would be important. Chest compressions alone can be easily taught, easily remembered, easily performed, adequately taught by dispatcher-delivered telephone instruction, and more readily accepted by the public. These benefits are especially important in the setting of single-rescuer bystander CPR. Further evaluation of simpler CPR techniques, such as chest compressions alone, is warranted.

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