

Importance of Continuous Chest Compressions During Cardiopulmonary Resuscitation

Improved Outcome During a Simulated Single Lay-Rescuer Scenario

Karl B. Kern, MD; Ronald W. Hilwig, DVM, PhD; Robert A. Berg, MD;
Arthur B. Sanders, MD; Gordon A. Ewy, MD

Background—Interruptions to chest compression–generated blood flow during cardiopulmonary resuscitation (CPR) are detrimental. Data show that such interruptions for mouth-to-mouth ventilation require a period of “rebuilding” of coronary perfusion pressure to obtain the level achieved before the interruption. Whether such hemodynamic compromise from pausing to ventilate is enough to affect outcome is unknown.

Methods and Results—Thirty swine (weight 35 ± 2 kg) underwent 3 minutes of untreated ventricular fibrillation before 12 minutes of basic life support CPR. Animals were randomized to receive either standard airway (A), breathing (B), and compression (C) CPR with expired-gas ventilation in a 15:2 compression-to-ventilation ratio or continuous chest compression CPR. Those randomized to the standard 15:2 group had no chest compressions for a period of 16 seconds each time the 2 ventilations were delivered. Defibrillation was attempted at 15 minutes of cardiac arrest. All resuscitated animals were supported in an intensive care environment for 1 hour, then in a maintenance facility for 24 hours. The primary end point of neurologically normal 24-hour survival was significantly better in the experimental group receiving continuous chest compression CPR (12 of 15 versus 2 of 15; $P < 0.0001$).

Conclusions—Mouth-to-mouth ventilation performed by single layperson rescuers produces substantial interruptions in chest compression–supported circulation. Continuous chest compression CPR produces greater neurologically normal 24-hour survival than standard ABC CPR when performed in a clinically realistic fashion. Any technique that minimizes lengthy interruptions of chest compressions during the first 10 to 15 minutes of basic life support should be given serious consideration in future efforts to improve outcome results from cardiac arrest. (*Circulation*. 2002;105:645-649.)

Key Words: cardiopulmonary resuscitation ■ ventilation ■ heart arrest ■ fibrillation

Bystander cardiopulmonary resuscitation (CPR) is one of the major elements in the “chain of survival” for the treatment of patients in cardiac arrest. Recent reports have indicated a decrease in the number of laypersons performing CPR.^{1–3} One reason postulated for this decline in bystander CPR is lay rescuer reluctance to administer mouth-to-mouth breathing.^{4,5} The American Heart Association has called for simplification as a central educational theme in efforts to encourage more bystander participation.⁶ One dramatic step toward simplification is to eliminate mouth-to-mouth ventilation for lay rescuers. Although some continue to voice concern about this approach,⁷ others have begun to test such a strategy in preclinical cardiac arrest models and have found equivalent outcomes when standard airway (A), breathing (B), and compression (C) CPR is compared with continuous

chest compression (CCC) CPR.^{8–13} These trials have all performed ABC CPR specifically as directed in the American Heart Association guidelines using a 3- to 4-second period for delivery of the 2 ventilations.¹⁴ Two clinical trials have also shown the benefit of CCC CPR.^{15,16}

A recent report from the United Kingdom in which 495 laypersons were prospectively randomized to training in either standard ABC CPR or CCC CPR demonstrated that extensive interruptions occur when laypersons stop chest compressions to ventilate.¹⁷ The average time from stopping compressions to restarting compressions for the delivery of 2 mouth-to-mouth ventilations was 16 seconds. Circulation was interrupted for this period, resulting in no circulatory support during nearly 60% of the resuscitation time. The purpose of the present study was to compare outcome results for single-rescuer standard ABC CPR,

Received August 27, 2001; revision received November 16, 2001; accepted November 16, 2001.

From the University of Arizona Sarver Heart Center, Section of Cardiology (K.B.K., R.W.H., G.A.E.); the Steele Memorial Children’s Research Center, Department of Pediatrics (R.A.B.); and Department of Surgery (A.B.S.), University of Arizona College of Medicine, Tucson, Ariz.

Correspondence to Karl B. Kern, MD, FACC, Professor of Medicine, University of Arizona, Sarver Heart Center, 1501 N Campbell Ave, Tucson, AZ 85724. E-mail kernk@u.arizona.edu

© 2002 American Heart Association, Inc.

Circulation is available at <http://www.circulationaha.org>

with clinically realistic compression interruptions for ventilation,¹⁷ versus CCC CPR.

Methods

Animal Preparation

All trials were conducted with the approval of The University of Arizona Institutional Animal Care and Use Committee. Thirty female swine (weight 35 ± 2 kg) were anesthetized with isoflurane inhalation anesthetic administered by face mask. An endotracheal tube was placed per os, and surgical anesthesia was maintained with 1% to 2% isoflurane and room air. A rate- and volume-regulated ventilator (Narkomed 2A, North American Drager) was used to maintain partial pressure end-tidal carbon dioxide (PETCO₂) at 40 ± 3 mm Hg, measured by an in-line infrared capnograph (No. 47210A, Hewlett-Packard). Animals were placed in dorsal recumbency on the surgical table and prepared for placement of vascular introducer sheaths as described previously.^{8–12} Micromanometer-tipped catheters (5F, MPC-500, Millar Instruments) were positioned in the right atrium to measure right atrial pressure (RAP) and in the descending aorta to measure aortic pressure (AoP). A 7F fluid-filled pigtail catheter (Cordis Corp) was also inserted into the ascending aorta to collect arterial blood for blood gas analyses. A balloon-tipped catheter (No. 131HF7, Baxter Health Care Corp) was positioned in the pulmonary artery to measure pulmonary arterial pressure and wedge pressure and to collect mixed venous blood for blood gas analyses. ECG leads were attached to all 4 limbs to monitor electrical activity of the heart, and an in-line pneumotachometer (MP45-871, Validyne Engineering Corp) was placed in the airway to measure minute ventilation.

Measurements

Continuous measurements of RAP, AoP, pulmonary arterial pressure, ECG, PETCO₂, and minute ventilation were recorded on a laptop computer with data acquisition software (Windaq DI-220 PGH, Dataq Instruments Inc). Coronary artery perfusion pressure was calculated as described previously^{8–13} as the mid-diastolic AoP minus mid-diastolic RAP and also as the integrated area between the aortic and right atrial diastolic pressure curves.¹⁸ Arterial and mixed venous blood samples for blood gas analyses were drawn at baseline and at 5, 10, and 14 minutes after fibrillation.

Experimental Protocol

The primary end point was neurologically normal 24-hour survival. Secondary end points included return of spontaneous circulation, resuscitation at 30 minutes, CPR-generated hemodynamics during CPR, arterial and mixed venous blood gases, and minute ventilation.

Baseline data were collected just before the electrical induction of ventricular fibrillation (VF) cardiac arrest. Cardiac arrest was confirmed by rapid decline of AoP and the characteristic ECG waveform of VF. Ventilation was discontinued at the time fibrillation was induced. All animals underwent 3 minutes of untreated VF and were then randomly assigned to either ABC CPR or CCC CPR. The rescuer, an experienced resuscitation research technician from our laboratory, was blinded relative to the data being collected but could not be blinded to the procedure. He was instructed to “do your best CPR” beginning 3 minutes after fibrillation. Standard ABC CPR consisted of 2 ventilations using expired gases (17% oxygen plus 4% carbon dioxide plus 79% nitrogen) administered by a bag-mask device (Ambu International), followed by 15 chest compressions at a metronome-synchronized rate of 100 per minute. Basic life support (BLS) CPR continued for 12 minutes. Chest compression pauses of 16 seconds for ventilation were included, patterned after the clinical experience in Cardiff, Wales.¹⁷ The CCC technique consisted of 100 chest compressions at 100 per minute followed by 2 breaths for the rescuer (no ventilation to the subject). Defibrillation (defibrillator/monitor M1722/B, CodeMaster XL, Hewlett Packard Co) was attempted at 15 minutes of cardiac arrest. Defibrillation shocks were administered at 4, 5, and 6 J/kg (monophasic waveform) for the first 3 attempts. All subsequent attempts, if any, used the 6 J/kg dose. If

TABLE 1. Outcome Results

Survival	ABC CPR (15 Animals)	CCC CPR (15 Animals)	<i>P</i>
ROSC	6	13	0.01
30 min	5	13	0.004
2 h	5	13	0.004
24 h	4	13	0.004
24 h and “good” neuro	3	12	0.002
24 h and “normal” neuro	2	12	0.0001

ROSC indicates return of spontaneous circulation; “good” neuro, cerebral performance category of 1 or 2; and “normal” neuro, cerebral performance category of 1 only.

defibrillation was unsuccessful, epinephrine (0.02 mg/kg) was given intravenously, followed by 1 minute of CPR before another defibrillation attempt. Additional epinephrine, if needed, was given at 3-minute intervals. Ventilation was begun at the beginning of the first defibrillation attempt with 100% oxygen and was continued until successful resuscitation, after which room air was used. Return of spontaneous circulation was defined as a peak systolic pressure of at least 50 mm Hg and a pulse pressure of at least 20 mm Hg for a minimum of 1 minute. Intravenous lactated Ringer’s solution, epinephrine, or dopamine was administered, if needed, during the 1-hour intensive care period immediately after successful defibrillation. Successfully resuscitated animals were deinstrumented, allowed to recover from anesthesia, and placed in observation cages for the ensuing 24 hours. Analgesics and intravenous fluids were given, if necessary, during the observation period.

Outcome and Neurological Evaluation

Animals were evaluated at 24 hours after cardiac arrest and awarded a swine Cerebral Performance Category (CPC) score.^{12,13} The CPC evaluation uses a 5-point scale to assess neurological function (1=normal: no difficulty with standing, walking, eating, or drinking, being alert and fully responsive to environmental stimuli; 2=mild disability: able to stand but exhibiting an unsteady gait, drinking but not eating normally, and showing a slower response to environmental stimuli; 3=severe disability: unable to stand or walk without assistance, not drinking or eating, awake but failing to respond normally to noxious stimuli; 4=coma; and 5=death). All surviving animals were then killed with intravenous barbiturates.

Statistical Analysis

Data are reported as mean and SEM. Discrete variables, such as return of spontaneous circulation, resuscitation, 24-hour survival, and normal neurological function at 24 hours, were compared with Fisher’s exact testing. Continuous variables, including all hemodynamics, blood gases, and ventilatory parameters measured during the CPR period, were compared with 2-tailed Student *t* testing for unpaired data. A *P* value <0.05 was considered significant.

Results

Outcome

The primary end point of neurologically normal 24-hour survival was significantly better in the experimental group receiving CCC CPR. Outcome results are shown in Table 1. “Normal” neurological function (CPC category 1 only) and “good” neurological function, defined as either CPC category 1 or 2, were both significantly better with CCC CPR. Moreover, 24-hour survival was substantially better with CCC CPR. Seventeen animals survived 24 hours (4 in the ABC group and 13 in the CCC group). Fourteen of these survived 24 hours with normal neurological function. Each

TABLE 2. Hemodynamics During CPR

	Base	Minutes					
		4	6	8	10	12	14
AoS, mm Hg							
ABC	80±2	82±7	101±9	107±8	107±9	112±10	107±9
CCC	78±2	86±4	90±4	92±5	94±5	90±4*	87±4
AoD, mm Hg							
ABC	61±1	33±1	37±2	37±3	34±2	33±2	28±2
CCC	62±3	32±2	31±1	30±2*	30±2	31±2	29±2
RAS, mm Hg							
ABC	9±1	93±11	107±13	118±13	121±14	124±10	122±13
CCC	9±1	102±10	108±10	112±10	114±10	111±9	104±12
RAD, mm Hg							
ABC	6±1	14±1	13±1	13±1	12±1	12±1	11±1
CCC	5±1	12±1*	13±1	13±1	12±1	11±1	11±1
CPP, mm Hg							
ABC	56±2	18±1	24±2	24±2	22±2	21±3	17±2
CCC	56±3	19±2	18±2*	18±2*	18±2	20±2	17±2

* $P < 0.05$ vs standard CPR.

†AoD indicates aortic diastolic pressure; AoS, aortic systolic pressure, CPP, coronary perfusion pressure; RAD, right atrial diastolic pressure; and RAS, right atrial systolic pressure.

exhibited normal walking, eating, and drinking. All were alert and responded appropriately to their environment. Two were from the standard BLS CPR group, and 12 were from the chest compression-only CPR group. One animal was scored as CPC category 2, indicating mildly abnormal neurological function. This animal, from the standard CPR group, exhibited an unsteady gait and a slower response to environmental stimuli. Finally, 2 animals were classified as CPC category 3, which is indicative of severe cerebral disability. Neither animal could walk or stand unassisted. Neither was in full coma, but both failed to respond to any noxious environmental stimuli. One of these animals was from the standard CPR group, and 1 was from the CCC group. These differences were neither subtle nor highly subjective.

Hemodynamics

Coronary perfusion pressure measured during CPR as mid-diastolic AoP minus RAP was significantly greater with ABC CPR during the mid portion of the resuscitation period (Table 2). Average mean coronary artery perfusion pressure during this period was 21 versus 18 mm Hg. However, integrated coronary perfusion pressure was significantly greater with the CCC group throughout the entire CPR period (Table 3).

Additional hemodynamics during CPR are shown in Table 2. Aortic systolic or peak pressures were different only at minutes 12 and 13, whereas aortic diastolic pressures were greater with ABC CPR at 5, 6, 8, and 9 minutes of CPR. Right atrial systolic diastolic pressures were similar between the 2 groups. The peak (systolic) pressures in both the aorta and right atrial chambers indicate not only equal but substantial compression force applied in both groups. The ABC CPR group had an average of 496 ± 7 total chest compressions during the 12-minute BLS period compared with 1111 ± 4 for the CCC group ($P < 0.0001$).

Ventilation

Table 4 shows the results of both blood gas analysis (arterial and mixed venous) and minute ventilation. Significantly better oxygenation and ventilation are seen with ABC CPR, but CCC CPR achieved substantial oxygenation and ventilation as well.

Discussion

This is the first report of adverse outcome resulting from prolonged pauses in CPR chest compressions for the delivery of mouth-to-mouth ventilation. Previous experimental reports from our laboratory⁸⁻¹² and others¹³ have repeatedly shown that elimination of mouth-to-mouth ventilation in the early stages of bystander-provided BLS for VF cardiac arrest provides similar outcome results compared with standard CPR that includes ventilation. Careful review of those studies reveals that in each case, the 2 ventilations delivered after

TABLE 3. Coronary Perfusion Pressure as the Integrated Area

Time, min	ABC, mm ² /min	CCC, mm ² /min	<i>P</i>
4	388±27	793±77	0.0001
5	452±40	754±72	0.002
6	485±48	753±84	0.01
7	510±52	773±103	0.03
8	524±54	755±104	0.06
9	500±53	756±102	0.04
10	470±56	747±100	0.03
11	432±54	699±92	0.02
12	407±58	661±91	0.03
13	377±50	620±84	0.02
14	312±48	557±72	0.02

TABLE 4. Blood Gases and Ventilation During CPR

	Baseline	5 min	10 min	14 min
pHa				
ABC	7.43±.01	7.55±.02	7.48±.02	7.43±.03
CCC	7.45±.01	7.46±.01	7.41±.03	7.34±.04
PaO ₂ , mm Hg				
ABC	42±1	26±3	26±2	26±2
CCC	39±1	36±2*	34±4*	37±5*
PaO ₂ , mm Hg				
ABC	71±4	87±8	83±7	83±7
CCC	72±5	58±4*	64±5*	62±7*
pHmv				
ABC	7.39±.01	7.30±.02	7.21±.02	7.18±.02
CCC	7.40±.01	7.32±.01	7.23±.02	7.18±.02
PmvCO ₂ , mm Hg				
ABC	47±1	56±2	64±2	64±2
CCC	46±1	54±2	61±3	63±3
PmvO ₂ , mm Hg				
ABC	35±2	23±1	18±1	17±1
CCC	34±1	19±1*	18±1	18±2
Minute ventilation, L/min				
ABC	5.8±1.1	11.5±1.4	11.8±1.3	11.3±1.4
CCC	5.1±1.1	7.8±1.4	6.3±1.1*	6.0±1.0*

* $P < 0.05$ vs standard CPR.

pHa indicates arterial pH; pHmv, mixed venous pH; PmvCO₂, mixed venous partial pressure of carbon dioxide; and PmvO₂, mixed venous partial pressure of oxygen.

every 15 chest compressions were accomplished with minimal interruption in chest compressions. Typically, an additional rescuer stationed at the head of the animal delivered the 2 breaths immediately after the 15th chest compressions, using 1.5 to 2 seconds per breath, as recommended by the American Heart Association CPR and emergency cardiovascular care guidelines.⁶ Not until recent prospective, randomized data involving laypersons performing CPR were published was it suspected that the typical interruption for ventilation is 16 seconds.¹⁷

These improved outcome results with CCC CPR again confirm that pH, oxygen saturation, and absolute levels of minute ventilation are not the primary determinants of 24-hour neurologically normal survival. Although there are critical limits for all these parameters for successful outcome, those limits are relatively generous and are not commonly breached during the first 12 minutes of BLS effort with or without supplemental positive-pressure mouth-to-mouth breathing.

The primary determinant of the different outcomes seen here appears to be the continuity of circulatory support during prolonged VF cardiac arrest. Of particular interest in this regard are the coronary perfusion pressure data. Calculating coronary perfusion pressure in the standard fashion as the mid-diastolic AoP minus mid-diastolic RAP does not take into account the interruptions associated with layperson efforts at ventilation. However, when coronary perfusion pressure is calculated as the total time of pulsatile diastolic

pressure difference between the aortic and right atrial chambers during the entire CPR period, which does account for any interruptions in chest compressions, an entirely different result is seen. CCC CPR then is shown to produce significantly more perfusion than does the frequently interrupted standard technique. Fewer interruptions in chest compression-supported circulation during cardiac arrest result in more perfusion to the heart and central nervous system, which culminates in better outcome.

The present study examined the effect of interrupting chest compressions for mouth-to-mouth breathing. However, similar results can be predicted with such interruptions for any reason, such as prolonged search for a pulse, tracheal intubation, and even automatic external defibrillator use if the mandatory period of "do not touch the patient" while the device analyzes the rhythm occurs too frequently. Any and all such causes of chest compression interruption during BLS efforts should be minimized to maximize successful resuscitation outcome.

Study Limitations

These data reflect the period of chest compression interruption reported when laypersons perform single-rescuer ABC BLS. The length of chest compression interruption may vary under different circumstances, including multirescuer ABC BLS and professional rescuers (single or multiple). Data for these other scenarios are not yet available. The central message is nonetheless clear, namely, that interruptions of chest compression can compromise resuscitation-generated circulation and ultimately 24-hour neurological recovery after CPR.

A second potential limitation is that an experienced veterinarian who was not blinded to the experimental procedure performed the neurological evaluations. Nonetheless, the swine CPC scale used is neither complex nor subtle. Because the differences found were neither subtle nor highly subjective, it seems unlikely that such were significantly affected by the lack of blinding of the neurological examiner. The outcome differences were quite substantial whether measured as 24-hour survival, 24-hour survival with good neurological function, or 24-hour survival with normal neurological outcome.

Conclusions

CCC CPR produces superior neurologically normal 24-hour survival compared with standard CPR when performed in a clinically realistic fashion by a simulated single layperson rescuer. Mouth-to-mouth ventilation performed by single layperson rescuers produces substantial interruptions in chest compression-supported circulation. Any technique that minimizes lengthy interruptions of chest compressions during the first 10 to 15 minutes of BLS for adult victims of witnessed sudden cardiac death should be given serious consideration in future efforts to improve outcome results from cardiac arrest.

Acknowledgments

This study was supported by grants from the Desert/Mountain Affiliate of the American Heart Association and the Max and Victoria Dreyfus Foundation Inc (White Plains, NY). Special thanks

to Alice McArthur, Marc Young, and Ed Bermingham for their technical help.

References

- Schneider T, Martens PR, Paschen H, et al. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. *Circulation*. 2000;102:1780–1787.
- Plaisance P, Lurie KG, Vicaut E, et al, for the French Active Compression-Decompression Cardiopulmonary Resuscitation Study Group. A comparison of standard cardiopulmonary resuscitation and active compression-decompression resuscitation for out-of-hospital cardiac arrest. *N Engl J Med*. 1999;341:569–575.
- Kentsch M, Schlichting H, Mathes N, et al. Out-of-hospital cardiac arrest in North-East Germany: increased resuscitation efforts and improved survival. *Resuscitation*. 2000;43:177–183.
- Ornato JP, Hallagan LF, McMahan SB, et al. Attitudes of BLS instructors about mouth-to-mouth resuscitation during the AIDS epidemic. *Ann Emerg Med*. 1990;19:151–156.
- Locke CJ, Berg RA, Sanders AB, et al. Bystander cardiopulmonary resuscitation: concerns about mouth-to-mouth contact. *Arch Intern Med*. 1995;155:938–943.
- American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: international consensus on science, part 1: introduction to the international guidelines 2000 for CPR and ECC: a consensus on science. *Circulation*. 2000;102(suppl 1):I-1–I-11.
- Brennan RT, Braslow A, Kaye W. A response to “A rationale for staged teaching of basic life support.” *Resuscitation*. 2000;44:143–146. Letter.
- Berg RA, Kern KB, Sanders AB, et al. Bystander cardiopulmonary resuscitation: is ventilation necessary? *Circulation*. 1993;88:1907–1915.
- Berg RA, Wilcoxson D, Hilwig RW, et al. The need for ventilatory support during bystander CPR. *Ann Emerg Med*. 1995;26:342–350.
- Berg RA, Kern KB, Sanders AB, et al. Assisted ventilation does not improve outcome in a porcine model of single-rescuer bystander CPR. *Circulation*. 1997;95:1635–1641.
- Berg RA, Kern KB, Hilwig RW, et al. Assisted ventilation during “bystander” CPR in a swine acute myocardial infarction model does not improve outcome. *Circulation*. 1997;96:4364–4371.
- Kern KB, Hilwig RW, Berg RA, et al. Efficacy of chest-compression only BLS CPR in the presence of an occluded airway. *Resuscitation*. 1998;39:179–188.
- Noc M, Weil MH, Tang W, et al. Mechanical ventilation may not be essential for initial cardiopulmonary resuscitation. *Chest*. 1995;108:821–827.
- American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. *JAMA*. 1992;268:2212–2302.
- Bossaert L, Van Hoeyweghen RJ, and the Cerebral Resuscitation Study Group. Bystander cardiopulmonary resuscitation (CPR) in out-of-hospital cardiac arrest. *Resuscitation*. 1989;1:S55–S69.
- Hallstrom A, Cobb L, Johnson E, et al. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. *N Engl J Med*. 2000;342:1546–1553.
- Assar D, Chamberlain D, Colquhoun M, et al. Randomized controlled trials of staged teaching for basic life support, 1: skill acquisition at bronze stage. *Resuscitation*. 2000;45:7–15.
- Berg RA, Sanders AB, Kern KB, et al. Adverse effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation*. 2001;104:2465–2470.