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Improved Neurological Outcome With Continuous Chest Compressions Compared With 30:2 Compressions-to-Ventilations Cardiopulmonary Resuscitation in a Realistic Swine Model of Out-of-Hospital Cardiac Arrest

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Background—The 2005 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care changed the previous ventilations-to-chest-compression algorithm for bystander cardiopulmonary resuscitation (CPR) from 2 ventilations before each 15 chest compressions (2:15 CPR) to 30 chest compressions before 2 ventilations (30:2 CPR). It was acknowledged in the guidelines that the change was based on a consensus rather than clear evidence. This study was designed to compare 24-hour neurologically normal survival between the initial applications of continuous chest compressions without assisted ventilations with 30:2 CPR in a swine model of witnessed out-of-hospital ventricular fibrillation cardiac arrest.

Methods and Results—Sixty-four animals underwent 12 minutes of ventricular fibrillation before defibrillation attempts. They were divided into 4 groups, each with increasing durations (3, 4, 5, and 6 minutes, respectively) of untreated ventricular fibrillation before the initiation of bystander resuscitation consisting of either continuous chest compression or 30:2 CPR. After the various untreated ventricular durations plus bystander resuscitation durations, all animals were given the first defibrillation attempt 12 minutes after the induction of ventricular fibrillation, followed by the 2005 guideline–recommended advanced cardiac life support. Neurologically normal survival at 24 hours after resuscitation was observed in 23 of 33 (70%) of the animals in the continuous chest compression groups but in only 13 of 31 (42%) of the 30:2 CPR groups ($P=0.025$).

Conclusions—In a realistic model of out-of-hospital ventricular fibrillation cardiac arrest, initial bystander administration of continuous chest compressions without assisted ventilations resulted in significantly better 24-hour postresuscitation neurologically normal survival than did the initial bystander administration of 2005 guideline–recommended 30:2 CPR. (***Circulation***. 2007;116:2525-2530.)

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

T he 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, hereafter referred to as the 2005 guidelines, changed the recommendations for single-rescuer cardiopulmonary resuscitation (CPR) from 2:15 ventilations-to-chest compressions (2:15 CPR) to 30:2 chest compressions-to-ventilations (30:2 CPR).[1] The rationale for these changes was to provide more chest compressions per minute.[1] It was stated that the new compression-to-ventilation ratio of 30:2 was selected on the basis of a consensus rather than clear evidence.[1]

In a realistic nonparalyzed swine model of prolonged ventricular fibrillation (VF) arrest, previous studies from our resuscitation research laboratory showed that continuous chest compression (CCC) without assisted ventilations was as effective as 2:15 CPR when the 2 ventilations interrupted chest compressions for only 4 seconds.[2–6] Although the previous guidelines recommended that each of the 2 breaths be delivered over a 2-second time interval,[7] manikin studies have demonstrated that lay individuals interrupt chest com-

pressions for an average of 16 seconds to deliver the 2 breaths.[8] When realistic 16-second interruptions of chest compressions were applied to our swine model comparing the then-standard ventilation-to-compression ratio (2:15 CPR) with CCC, CCC resulted in significantly better neurologically normal survival.[9]

The purpose of the study reported here was to determine whether the 2005 guideline–recommended single-bystander 30:2 CPR results in equivalent or different 24-hour postresuscitation normal neurological function compared with CCC in a swine model of out-of-hospital VF cardiac arrest when realistic 16-second interruptions of chest compressions were used to provide the 2 breaths.

Methods

Outcomes

The primary outcome was 24-hour survival with normal neurological function. Secondary end points included return of spontaneous circulation (ROSC), the presence of a perfusing rhythm after the first defibrillation shock, and overall 24-hour survival. The purpose of studying progressively longer intervals before the initiation of resuscitation efforts was to determine whether the duration of untreated VF had an influence on the need for ventilation during basic cardiac life support.

Experimental Preparation and Design

This study was conducted with the approval of the University of Arizona Institutional Animal Care and Use Committee in accordance with the guidelines set forth in the *Position of the American Heart Association on Research Animal Use*. Sixty-four domestic swine of either sex weighing 28 ± 4 kg were anesthetized with 5% isoflurane inhalation anesthetic in oxygen administered by nose cone. An endotracheal tube was placed per os, and anesthesia was maintained using 1.5% to 3% isoflurane in room air until induction of VF. The ventral neck area was prepared in standard fashion for sterile cutdown procedures to place vascular introducer sheaths (5F to 7F, Cordis Corp, Miami, Fla) into selected vessels. ECG leads were placed on the limbs to continuously monitor heart rate and rhythm. An infrared capnometer (47210A, Hewlett Packard Co, Palo Alto, Calif) and a pneumotachometer (MP45–871, Validyne Engineering Corp, Northridge, Calif) were placed in the airway to measure the end-tidal partial pressure of carbon dioxide (P_{ETCO_2}) and tidal volume, respectively. Ventilation was provided by a rate- and volume-regulated ventilator/anesthesia machine (Narkomed 2A, North American Drager, Telford, Pa). The initial ventilation rate was 12 rpm, and tidal volume was 15 mL/kg. Rate and/or volume were altered to maintain P_{ETCO_2} at 40 ± 3 mm Hg. Solid-state pressure transducers (MPC-500, Millar Instruments, Houston, Tex) were placed, one via a common carotid artery sheath into the descending aorta to measure its pressure, and the other via an external jugular vein into the right atrium to measure its pressure. The animals were placed in a dorsal recumbent position in a V tray, and adhesive defibrillator pads (Quik-Combo, Medtronic, PhysioControl, Redmond, Wash) were adhered to the chest.

Data Acquisition

Data, consisting of aortic pressure, right atrial pressure, tidal volume, ECG, and Peak end-tidal CO_2 (P_{ETCO_2}), were continuously displayed on a physiological recorder (Gould Ponemah Physiology Platform, model P3 Plus, LDS Life Science, Valley View, Ohio) and stored on a laptop computer for later analyses. Coronary perfusion pressure during CPR and CCC administration was calculated as the difference between aortic pressure and right atrial pressure in the midrelaxation phase of chest compressions. All animals had normal sinus rhythm, arterial blood gases, oxygen saturations, and blood pressures before induction of VF.
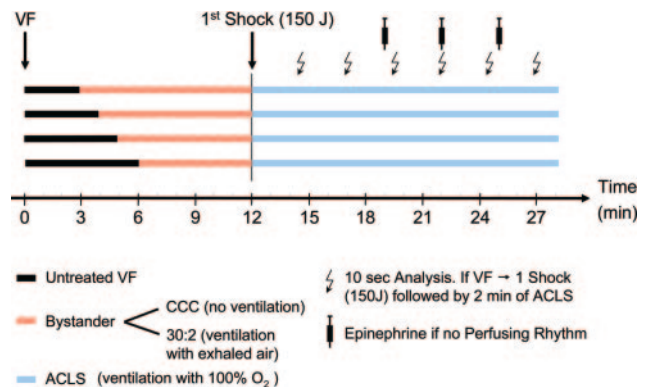

Figure 1. Experimental protocol timeline. The initial biphasic defibrillator shock of 150 J was given after 12 minutes of VF. The crooked arrows indicate the onset of a 10-second pause in chest compressions for rhythm analysis and a single 150-J shock if indicated. The syringe indicates the administration of epinephrine if a perfusing rhythm was not present. Black bars indicate the duration of untreated VF; hatched red bars, CCC or 30:2 CPR bystander resuscitation; light blue bars, ACLS according to the 2005 guidelines.

Baseline data were collected; then, VF was induced with a pacing electrode temporarily placed in the right ventricle. The presence of VF was confirmed by the characteristic ECG waveform and the precipitous fall in aortic pressure. Assisted ventilation was discontinued, and the animals underwent a period of untreated VF to simulate the time it might take a bystander to recognize the problem, try to arouse the victim, perhaps call for help, phone the emergency medical service, and begin resuscitation efforts.

Experimental Protocol

The animals were assigned to 1 of 4 experimental groups relative to the time before simulated bystander resuscitation was begun randomly with either CCC without assisted ventilations or 30:2 CPR. Groups 1 through 4 sustained untreated VF for periods of 3, 4, 5, and 6 minutes, respectively, before initiation of the resuscitation protocols (Figure 1). Within each group, the animals were randomly assigned to receive either CCC without assisted ventilations or 30:2 CPR using exhaled gas for 2 equally spaced mouth-to-endotracheal tube ventilations of 2 seconds each during the 16-second interruption of chest compressions. All chest compressions were administered manually at a metronome-directed rate of 100 per minute. Care was taken to ensure that the hands of the rescuer were completely elevated from the chest during the relaxation phase of compressions to allow recoil of the chest. These resuscitation efforts continued until simulated emergency medical services arrival at 12 minutes after arrest. A single biphasic defibrillation shock of 150 J was delivered at this time (LifePak 12, Medtronic Emergency Response Systems, Redmond, Wash), and advanced cardiac life support (ACLS) per the 2005 guidelines was begun immediately after the shock with no pause for automated external defibrillator rhythm analysis.[1] Ventilations with 100% oxygen were delivered manually by Ambu bag. After each 2 minutes of standard CPR, a 10-second pause was interjected to simulate automated external defibrillator rhythm analysis. If after the defibrillation shock a perfusing rhythm (peak aortic systolic pressure >50 mm Hg) was achieved, the animals were connected to the mechanical ventilator and given 100% oxygen at an initial rate of 12 breaths per minute and a tidal volume of 15 mL/kg. Rate and/or tidal volumes were subsequently adjusted as needed to return P_{ETCO_2} to normal values. Isoflurane was added if and when the animals began to stir. No further chest compressions were administered unless the animals had recurrent VF.

ROSC was defined as a peak aortic pressure of >50 mm Hg and pulse pressures of >20 mm Hg sustained for 1 minute. If either VF or pulseless electrical activity was present after the defibrillation shock, an additional 2 minutes of ACLS was given before the next

Table 1. Baseline Data

	CCC	30:2 CPR	*P*
n	33	31	
Gender, F/M	19/14	10/21	0.042
Weights, kg	27±3	28±4	0.159
Heart rate, bpm	101±24	106±18	0.714
Mean systolic BP, mm Hg	80±16	81±14	0.607
Diastolic BP, mm Hg	54±12	55±10	0.430
Cardiac output, L/min	2.5±0.4	2.6±0.7	0.304
Arterial Po_2, mm Hg	75±12	78±22	0.920
Arterial saturation, %	40±2	39±2	0.463
Arterial Pco_2, mm Hg	92±2	92±4	0.651

BP indicates blood pressure.

Table 2. Outcomes

	CCC	30:2 CPR	*P*
n	33	31	
Normal 24-h neurological score	23/33	13/31	0.025
24-h Survival	24/33	18/31	0.217
ROSC	26/33	19/31	0.126
Perfusing rhythm after first shock	21/33	9/31	0.006

10-second pause to simulate automated external defibrillator rhythm analysis. This procedure was continued until a perfusing rhythm was attained or until 19 minutes after arrest, when epinephrine (0.02 mg/kg IV) was given. If required, epinephrine administration was repeated at 3-minute intervals, and ACLS was continued as previously until successful ROSC or 3 doses of epinephrine were administered. If VF or pulseless electrical activity was still present at 28 minutes after arrest (3 minutes after the last epinephrine dose), resuscitation efforts were discontinued.

Animals that had a positive ROSC were reconnected to the ventilator/anesthesia machine and underwent a 1-hour intensive care period during which they were given intravenous fluids to restore third-space fluid losses. With the exception of 1 jugular vein sheath that was used for fluid administration, all other vascular sheaths were removed. The animals were allowed to recover from anesthesia, placed in observation cages, and monitored over the ensuing hours until 24 hours after resuscitation, when a neurological examination was performed as previously described.[3,5,10] Briefly, a score of 1 is normal, 2 is abnormal (eg, not eating or drinking normally, unsteady gait, or slight resistance to restraint), 3 is severely abnormal (the animal is recumbent, unable to stand, and only partially responsive to stimuli), 4 is comatose, and 5 is dead. After the neurological examination, the animals were humanely euthanized by intravenous injection of a commercial euthanasia solution (Fatal+, Vortech Pharmaceuticals, Dearborn, Mich).

Data Analysis

The data were transported into SPSS 15.0 for Windows for statistical analysis (SPSS, Inc, Chicago, Ill). Continuous variables are presented as mean±SD and were analyzed by Student's *t* test or the Mann–Whitney *U* test for nonnormal distribution. The primary and secondary outcomes in different groups were compared with the χ^2 test or Fisher's exact test. A logistic regression analysis was used to determine the odds of 24-hour postresuscitation normal neurological function in the CCC group compared with the 30:2 CPR group with adjustment for differences in the time lags.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

The 2 major groups were comparable at baseline (Table 1). Because of a randomization error, 33 swine were in the CCC groups and 31 swine were in the 30:2 CPR groups. The number of neurologically normal survivors 24 hours after resuscitation was significantly greater in the CCC groups (23 of 33, 70%) compared with the 30:2 CPR groups (13 of 31, 42%; *P*=0.025; Table 2 and Figure 2). With longer duration of untreated VF before the initiation of resuscitation efforts,

fewer animals survived with good neurological outcomes, and more animals had significant neurological deficits (Figure 2). In the combined 4, 5, and 6 minutes of untreated VF groups, 16 of 17 in the CCC groups and 6 of 11 in the 30:2 CPR groups survived with normal neurological function (*P*=0.022, Fisher exact test). In the combined 5 and 6 minutes of untreated VF groups, 10 of 10 in the CCC groups but only 4 of 9 in the 30:2 CPR groups survived with normal neurological function (*P*=0.011, Fisher exact test). The odds of neurologically normal 24-hour postresuscitation survival was significantly greater for the CCC groups (odds ratio, 3.7; 95% confidence interval, 1.2 to 11.3) compared with the 30:2 CPR groups with adjustment for differences in the time lag. The odds ratios for ROSC and 24-hour survival were not significant between the 2 groups.

A significant difference was found between the 2 resuscitation techniques relative to the cardiac rhythm following the first defibrillation shock. A perfusing rhythm followed the first defibrillation in 21 of 33 animals (64%) in the CCC groups and in 9 of 31 animals (29%) in the 30:2 CPR groups (*P*=0.006; Table 2). This outcome contributed to a significant difference between groups in the greater need for epinephrine administration during ACLS resuscitation efforts (12 of 33 in the CCC groups versus 20 of 31 in the 30:2 CPR groups; *P*=0.024; Table 3).

The hemodynamic data during resuscitation are shown in Table 3. The mean integrated coronary perfusion pressure during basic life support was 20±10 mm Hg with CCC and 14±10 mm Hg with 30:2 CPR (*P*=0.028). The mean integrated coronary perfusion pressure in the 24-hour survivors with normal neurological function was 23±8 mm Hg but only 10±7 mm Hg in animals with severe neurological deficits (*P*=0.001). There was no difference in the mean integrated coronary perfusion pressure between the survivors with severe neurological deficits and nonsurvivors. The mean number of chest compressions delivered during the bystander resuscitation period was 746±112 in the CCC groups and 385±63 in the 30:2 CPR groups (*P*=0.001; Table 3). The mean arterial blood oxygen saturation after 12 minutes of VF, just before the first defibrillation shock, was 79±29% for the CCC group versus 88±7% for the 2:30 CPR group (*P*=0.288; Table 3). No significant differences existed in arterial Po_2 or Pco_2 between groups at the completion of the bystander resuscitation period (eg, after 12 minutes of VF) (Table 3).

Discussion

This is the first reported study comparing CCC without assisted ventilations with the 2005 American Heart Associa-
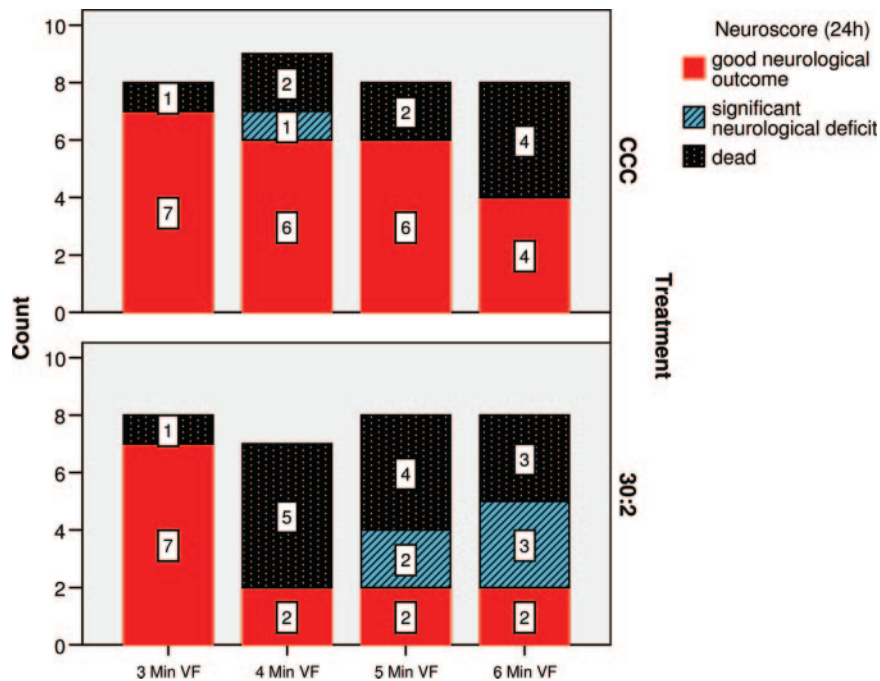
Figure 2. Outcomes of all 64 animals in the experiments. Top, Outcomes of the animals receiving CCC without assisted ventilations. Bottom, Outcomes of the animals given 30:2 CPR. The individual columns represent durations in minutes of untreated VF. Red bars indicate normal neurological outcomes at 24 hours; hatched blue bars, 24-hour survival with significant neurological deficits; and dotted black bars, animals that did not survive.

tion guideline recommendations for basic cardiac life support of 30 chest compression before each 2 ventilations during simulated single-bystander resuscitation for out-of-hospital VF arrest. When all untreated VF times were combined, there was a significant difference favoring CCC over 30:2 CPR for 24-hour neurologically normal survival (23 of 33 [70%] versus 13 of 31 [42%]; $P=0.025$). When the duration of untreated VF exceeded 3 minutes before the initiation of simulated bystander resuscitation, 16 of 17 of the surviving animals in the CCC groups were neurologically normal at 24 hours after resuscitation compared with 6 of 11 in the 30:2 CPR groups ($P=0.022$; Figure 2).

It has been shown in experimental models that a major determinant of survival after cardiac arrest is coronary per-

Table 3.   Intraresuscitation Parameters

	CCC	30:2 CPR	P
n	33	31	...
Epinephrine administration,	12/33	20/31	0.045
Chest compressions delivered, n	746±112	385±63	0.0001
Mean CPP, mm Hg	20±10	14±10	0.028
Mean aortic "systolic" pressures, mm Hg	95±31	93±40	0.368
Mean aortic "diastolic" pressures, mm Hg	29±11	24±11	0.0001
Mean RA "systolic" pressures, mm Hg	89±44	90±36	0.323
Mean RA "diastolic" pressures, mm Hg	11±5	11±4	0.064
Mean arterial Po_2 at 12 min of VF, mm Hg	59±24	60±13	0.829
Mean arterial Pco_2 at 12 min of VF, mm Hg	32±18	34±11	0.477
Mean arterial So_2 at 12 min VF, %	79±29	88±7	0.384

CPP indicates coronary perfusion pressure; RA, right atrial.

fusion pressure (ie, the difference between the aortic and right atrial pressures during the release phase of chest compression).[11] Uninterrupted chest compressions produced higher integrated coronary perfusions pressures relative to the 30:2 CPR groups because of the required pauses in compressions in the latter group to provide breaths (Table 3). Furthermore, CCC generated more consistent arterial systolic pressures, which provided cerebral perfusion and presumably contributed to the better neurologically normal survival in this group (Figure 3).

Typically, out-of-hospital resuscitation efforts are delayed for some time after collapse. The present study suggests that perfusion should take precedence over ventilations for postresuscitation neurologically normal survival when resuscitation efforts are not initiated within 4 minutes after collapse. Although ventilation might become essential during the treatment of very prolonged VF, this study indicates that assisted ventilations apparently are not necessary during the first 12 minutes of VF even if initiation of bystander resuscitation is delayed for as long as 6 minutes after the onset of VF.

The change in the 2005 guidelines from 2:15 CPR to 30:2 CPR was based in part on the fact that the revised ratio would produce more chest compressions per unit of time and theoretically would improve perfusion.[1] The increased number of chest compressions delivered using 30:2 CPR has been validated.[12] However, that experimental study comparing 2:15 CPR with 30:2 CPR did not evaluate 24-hour survival or neurological outcome.[12] A before-and-after clinical study among municipal firefighters who originally used 15:2 CPR and then were retrained to perform 30:2 CPR was reported from Pittsburgh.[13] No differences in any outcome measures were found.[13]

Our study has several potential limitations. The conclusions of the study may not apply to bystander CPR performed by 2 rescuers when the interruptions for chest compressions
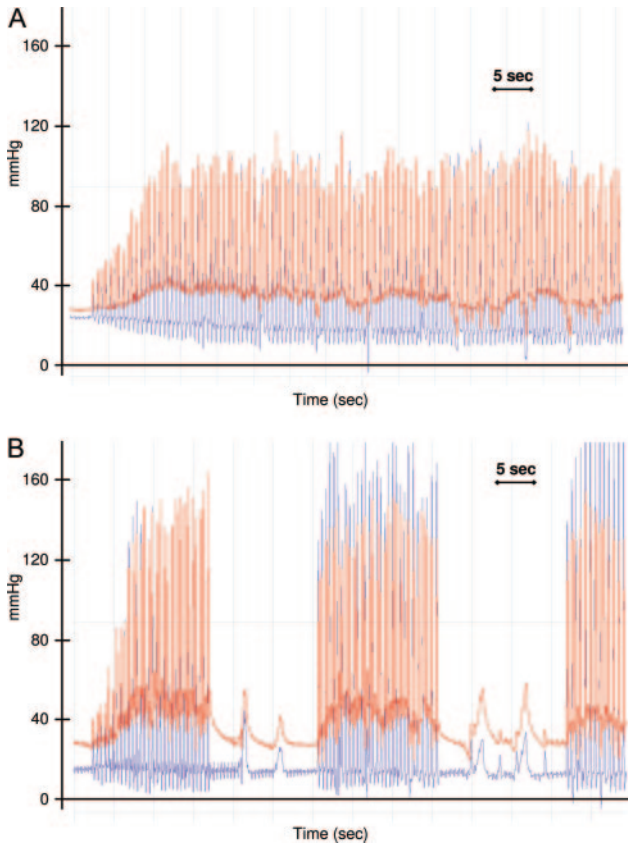
Figure 3. A, Initiation of CCC during VF. B, Initiation of 30:2 CPR during VF. Aortic pressure is in red; right atrial pressure is in blue.

to deliver the 2 recommended ventilations after each 30 compressions would not be as long.

The nature of this study precluded the investigators from being blinded to the procedures. To ensure that bias did not influence the depth or force of manual chest compressions, the mean arterial systolic blood pressures were measured during the last 10 seconds of the first 4 compressions cycles during 30:2 CPR and compared with analogous time frames during CCC. Table 3 shows that these means were not different. This study was done in young healthy swine with compliant chests and without discernible coronary artery disease. The neurological examinations were conducted by experienced study personnel, including a veterinarian with substantial experience evaluating swine neurological status. Observer bias was minimized by the clearly defined and easily assessed swine cerebral performance categories.

Another potential limitation is that this study was done with an endotracheal tube in place. In previous animal studies, we have demonstrated substantial passive gas exchange during chest compressions even when the endotracheal tube was removed before the provision of chest compressions.[4,5,10] We also have shown that animals were effectively resuscitated by chest compressions alone with clamped endotracheal tubes.[6] Moreover, many adults have gasping breaths before and during resuscitation efforts.[14] Finally, humans without endotracheal tubes can have better outcomes after bystander chest compressions alone compared with chest compressions with rescue breathing.[15]

For a variety of reasons, the question of the relevance of swine studies to human resuscitation is always an issue. However, our previous finding[9] of improved survival in swine with CCC versus 2:15 CPR has recently been confirmed by an observational study in humans (Cardiopulmonary Resuscitation by Bystanders With Chest Compression Only [SOS-KANTO]).[15,16]

Conclusions

In a realistic swine model of single-bystander out-of-hospital VF cardiac arrest in which defibrillation was first attempted at 12 minutes of the arrest, CCC resulted in more 24-hour neurologically normal survivors than did the 2005 guideline–recommended 30:2 CPR.

Disclosures

None.

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CLINICAL PERSPECTIVE

In the absence of early defibrillation, prompt initiation of resuscitation by bystanders is a major determinant of neurologically intact survival after out-of-hospital sudden cardiac arrest. Unfortunately, some bystanders do not initiate resuscitation because of an aversion to mouth-to-mouth ventilation. This study assessed neurological outcomes in a porcine model of cardiac arrest treated with continuous chest compressions without interruption for ventilation compared with chest compressions interrupted for ventilation. Neurological outcomes were better with chest compressions only. Although this study was conducted in a nonparalyzed swine model, recent human observations support the applicability of this model to humans with important implications. In contrast to secondary cardiac arrest resulting from severe hypoxia, "rescue breathing" may be unnecessary in patients with primary cardiac arrest. During chest compressions, perfusion is marginal, so that stopping compressions even briefly for ventilation (except for prompt defibrillation) is potentially harmful. Furthermore, positive pressure ventilation decreases venous return to the chest and subsequently perfusion of the heart and the brain. The findings of this study support continuous chest compressions as a new bystander approach to saving patients with cardiac arrest.