TO VENTILATE OR NOT TO VENTILATE: THAT IS THE QUESTION
Part II

To begin to answer the question of whether ventilations are needed during cardiopulmonary resuscitation (CPR), Part I of this newsletter published in September showed that ventilations during cardiac arrests are not performed according to the *American Heart Association (AHA) Guidelines*. The high rates of ventilation increase intrathoracic pressure, decrease cardiac output and coronary perfusion pressure, and are related to poor survival. Several methods were suggested to coach providers in the delivery of the correct rate of ventilations. Gasping is common early in cardiac arrest, contributes to increased coronary and cerebral perfusion pressure, and is associated with an improved survival. But the presence of gasping confuses many rescuers and leads to CPR being withheld.

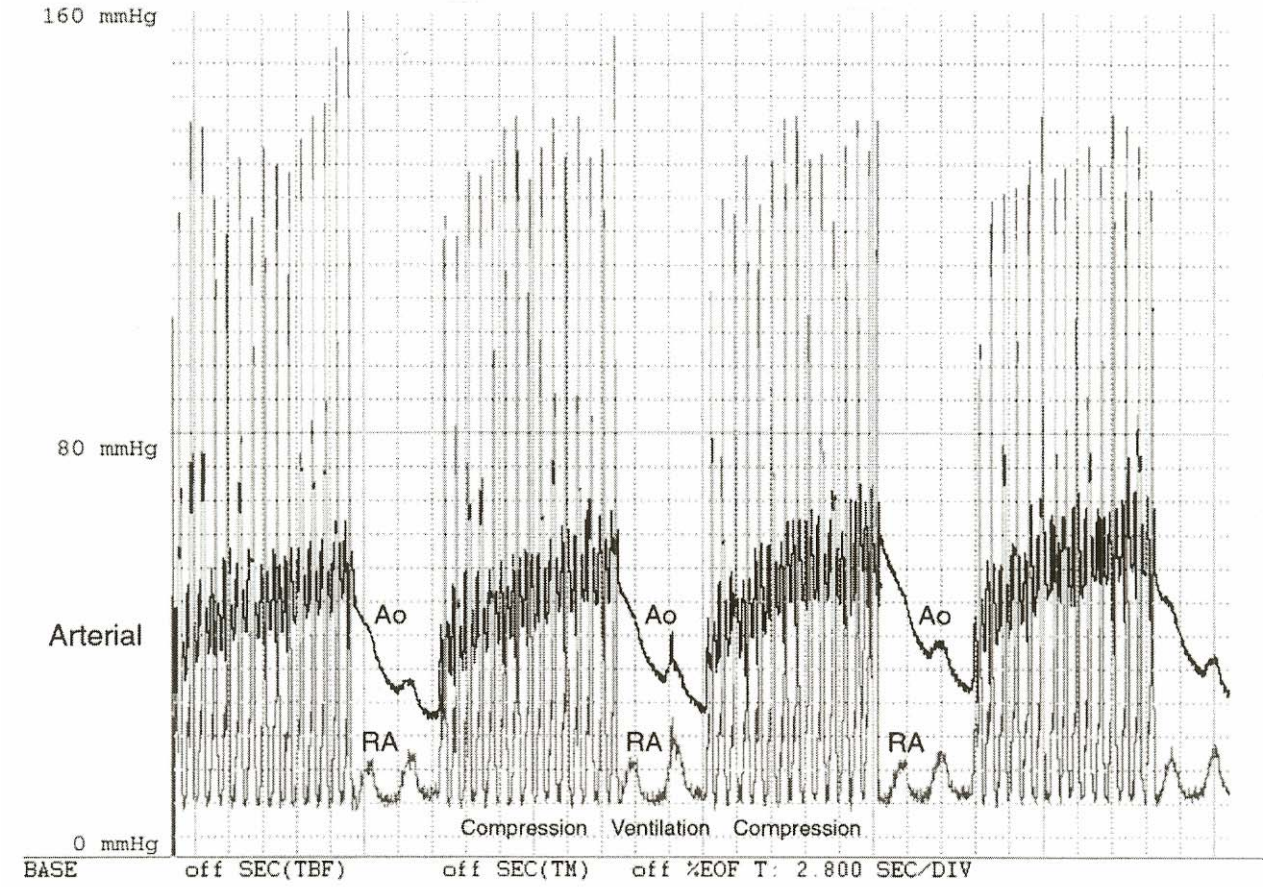
In this issue of *Code Communications* we will explore the harm produced by interruptions in compressions and the benefits/hazards of ventilation. Then we will review the research related to compressions without ventilations. Finally, we will end with a discussion of the optimal compression:ventilation ratio.

Interruption of Compressions

Wik found that in out-of-hospital (OOH) cardiac arrests, victims treated by paramedics and nurse anesthetists received chest compressions only 52% of the time.[16] In the hospital environment, Abella found that chest compressions were not being performed 24% of the resuscitation time.[4] Chest compressions are interrupted by ventilations, defibrillation, intubation, insertion of a central line, pulse checks, and inattention of the responders. Recommendations from the AHA for standard CPR assume that the pause for ventilation will take approximately 4-5 seconds, but it has been found in studies that this pause is much longer. Assar showed that a single lay bystander interrupts chest compressions for an average of 16 seconds to deliver two mouth-to-mouth ventilations.[17] This means, depending on the compression:ventilation ratio, up to 50% of the total resuscitation time is spent delivering breaths. Trained professionals do not perform much better. Higdon reported that it takes paramedics 10$\pm$1 seconds to deliver 2 breaths.[18]

What are the hemodynamic effects of interrupting chest compressions for rescue breathing during CPR? Berg addressed this question using an animal model.[19] After 3 minutes of untreated ventricular fibrillation (VF), 14 swine were randomly assigned to receive 12 minutes of compressions at a rate of 100/minute with ventilations in a 15:2 ratio for group 1 (CC+RB) vs. no ventilations for group 2 (CC). At the end of this simulated bystander CPR period, 15 minutes after VF was induced, all animals received Advanced Cardiac Life Support (ACLS) according to the AHA standards (including defibrillation), as if the paramedic unit had arrived at the scene. For the CC+RB group, the aortic relaxation ("diastolic") pressures routinely decreased during the interval of 2 rescue breaths when no compressions were provided, thereby also decreasing the coronary perfusion pressure (CPP). The mean CPP was 14$\pm$1 mmHg for the first two compressions in each compression cycle compared to 21$\pm$2 mmHg for the final two compressions – thus 50% less at the beginning of chest compressions compared to the end of the cycle. It took seven compressions to build up to a stable CPP during a cycle. See Figure 9 for hemodynamic measurements taken during the 15:2 compression-to-ventilation ratio CPR.[20]

Figure 9 Aortic and Right Atrial Pressures Measured during Standard CPR and Ventilation Using a 15:2 Compression-to-Ventilation Ratio

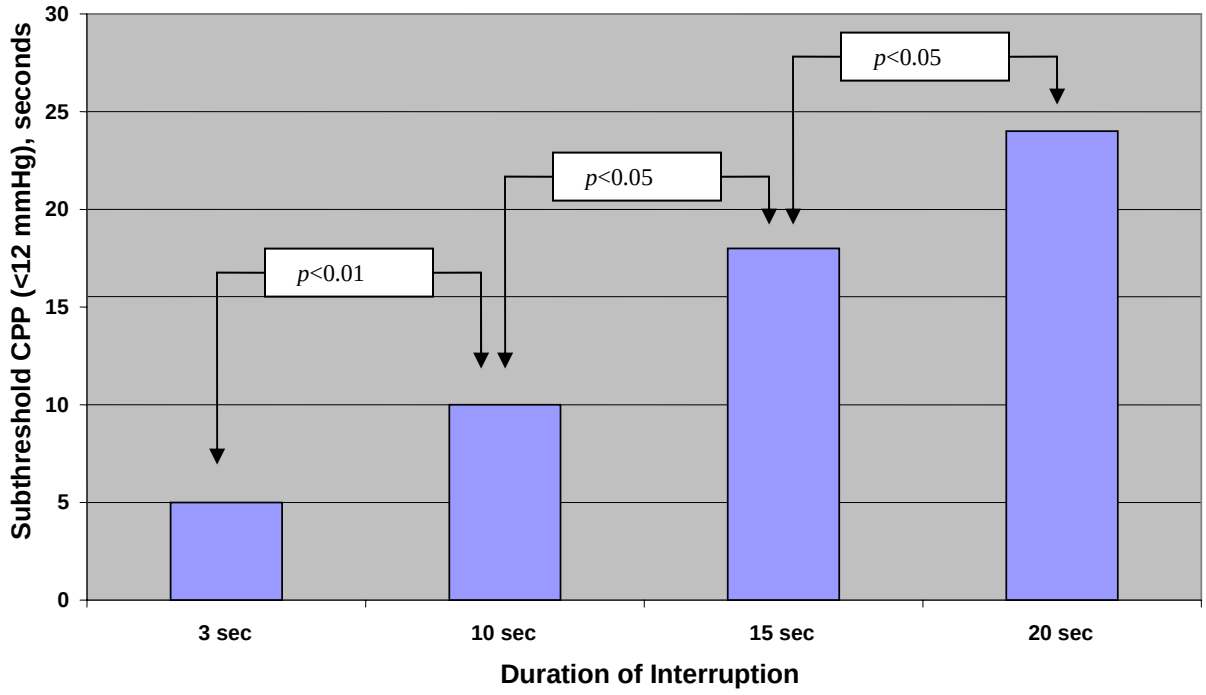


Note: The interruptions of aortic diastolic pressure (lower border of the dark band) during ventilation is easily recognized, resulting in a sub-optimal coronary perfusion pressure during that time. Right atrial diastolic pressure is seen as the most inferior border of the pressure waves. The difference is the coronary perfusion gradient. Maximal coronary perfusion occurs only a third of each compression-relaxation-ventilation cycle.

In Berg's study, the arterial oxygen saturation and pH were higher and pCO_2 lower in the CC+RB group at 5 and 15 minutes after VF (i.e., after 2 and 12 minutes of CPR). Thirteen of the 14 animals survived 24 hours with good neurological outcome. Six of the 7 CC animals, and 5 of the 7 CC+RB animals, were in cerebral performance category (CPC) 1 (i.e., normal) at 24 hours; 1 in each group was in CPC 2 (i.e., mildly abnormal); and 1 CC+RB animal was in CPC 3 (i.e., severely disabled). Berg concludes: "This investigation establishes that interrupting chest compressions for rescue breathing can adversely affect myocardial hemodynamics during CPR for VF. Compared with CC, CC+RB resulted in worse myocardial perfusion, yet better oxygen content of the blood that perfused the myocardium. The net result was no substantial difference in myocardial oxygen delivery. Not surprisingly, this study confirmed that successful resuscitation and neurological outcome are comparable after CC or CC+RB for VF cardiac arrest. More importantly, this investigation highlights the hemodynamic importance of continuous chest compressions during CPR."

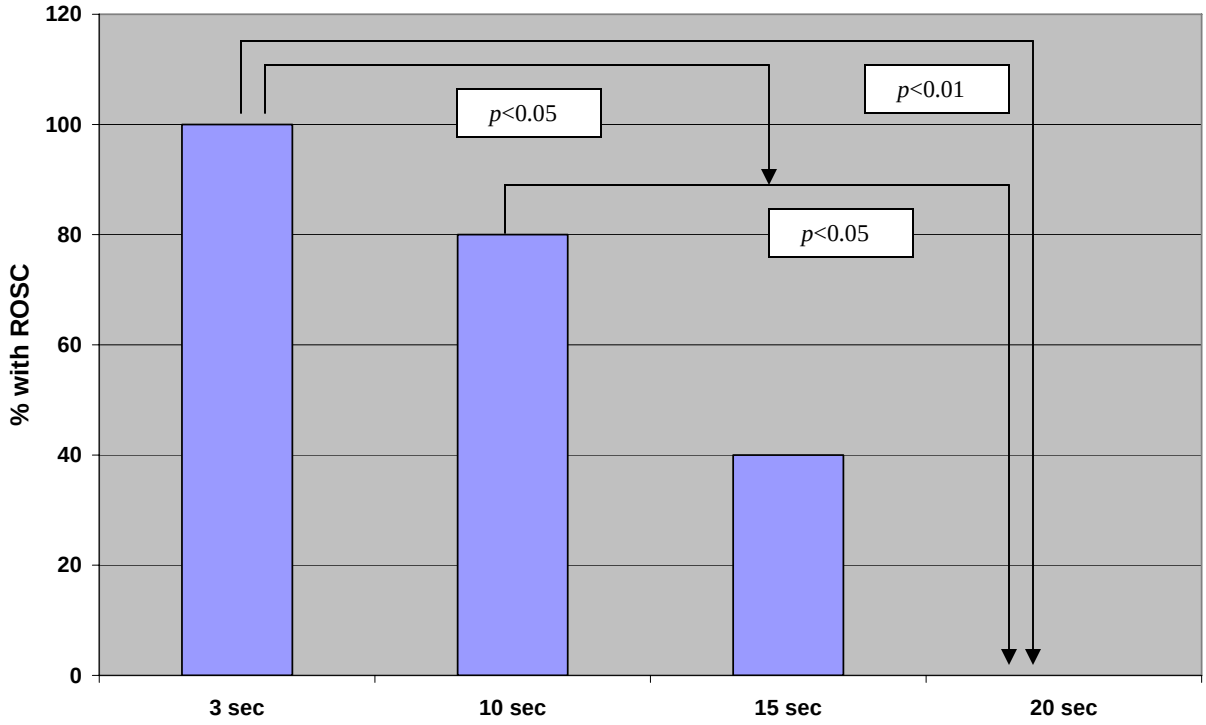
In another randomized swine study by Yu, 20 pigs in VF were left untreated for 7 minutes before CPR was started and defibrillation performed.[21] He investigated CPP and survival related to defibrillation being preceded by 3-, 10-, 15-, or 20-second interruptions in chest compressions delivered by the mechanical device, Thumper, at 100/minute. Ventilations were delivered in a synchronized fashion with compressions at a 1:5 ratio. Figure 10 shows the relationship between the duration of interruptions and sub threshold levels of CPP (i.e., <12 mmHg) during the initial 3 minutes of CPR.

Figure 10 Relationship between Duration of Interruption in Compressions and Coronary Perfusion Pressure (Yu)



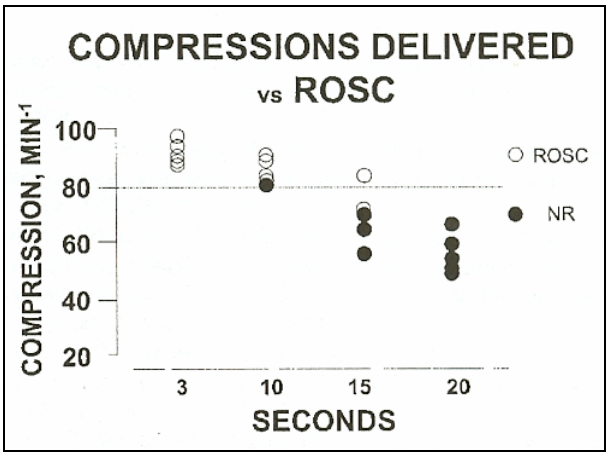
Yu found that success of CPR (i.e., return of spontaneous circulation [ROSC]) decreased with increasing durations of interrupted compressions; see Figure 11. No animals in which precordial compression was interrupted for 20 seconds were resuscitated. The ejection fraction was significantly reduced in all resuscitated animals but disproportionately so when interruptions exceeded 3 seconds. At the end of 72 hours, the ejection fraction was only 70% of baseline in animals after 15 seconds of interruption.

Figure 11 Success of CPR Related to Interruption Duration (Yu)



Yu related the number of compressions delivered over the first three minutes of CPR to either ROSC or failure of resuscitation (NR). See Figure 12. It appears that the cut-off for ROSC was 80 compressions/minute.

Figure 12 Relationship between Number of Chest Compressions and ROSC (Yu)



Yu concluded that threshold values of CPP are maintained with uninterrupted chest compression. Delays for rhythm analysis and defibrillator charging reduced the number of compressions over time and the capability to maintain threshold levels of CPP. The interruptions in chest

compression and their effect on myocardial perfusion translated into correspondingly lower success of the initial resuscitation effort and post resuscitation myocardial function. Interruptions in compressions result in these negative outcomes especially in the settings in which VF consumes disproportionately large myocardial oxygen demands.

The Physiology of Ventilation in Cardiopulmonary Resuscitation

Cardiac output and pulmonary blood flow produced by chest compression during CPR are less than 1/3 normal physiological levels. During states of low blood flow, (e.g., CPR), there is a significant reduction in total body CO_2 production and the coexisting need to eliminate it. Accordingly, smaller ventilations are required to maintain optimal ventilation/perfusion ratios. Gas exchange is induced with chest compressions, i.e., air is forced out of the chest during compressions and air is allowed back into the chest during recoil. In the swine model of VF, approximately 2-5 L/minute of mean expiratory minute ventilation has been documented with chest compression-only CPR, mostly from chest compression induced gas exchange.[22] In addition, the presence of gasping results in gas exchange.

A low compression:ventilation ratio, e.g., 5:1, produces high arterial blood oxygen levels, but results in lower oxygen delivery, compared to higher CPR ratios. This is due to the poor blood flow produced by the intermittent chest compressions. Ratios of 15:2 and 50:5 produce oxygen deliveries that are 16-17% higher than the 5:1 ratio. The lower blood oxygen levels with these ratios are more than offset by the greater blood flows generated.[23]

When the etiology of the arrest is respiratory, because pulmonary blood flow is normal (or near normal), higher ventilation rates are required to provide a ventilation/perfusion match.

Hazards of Ventilation during Cardiopulmonary Resuscitation

We know from the *AHA Guidelines* that air delivered with each rescue breath when an advanced airway is not in place can enter the stomach if pressure in the esophagus exceeds the lower esophageal sphincter opening pressure.[2] Risk of gastric inflation is increased by high proximal airway pressure and the reduced opening pressure of the lower esophageal sphincter. High pressure can be created by a short inspiratory time, large tidal volume, high-peak inspiratory pressure, incomplete airway opening, and decreased lung compliance. Recommendations to minimize the potential for gastric inflation are to deliver each breath over one second and deliver a tidal volume that is sufficient to produce a visible chest rise.

Virkkunen prospectively studied the relationship of bystander mouth-to-mouth ventilation and regurgitation during OOH cardiac arrest in three Finnish urban communities.[24] In the 390 patients who suffered a cardiac arrest prior to the arrival of EMS, the incidence of regurgitation was 28.2% (n=110). In the majority of these patients, regurgitation had occurred before the arrival of the EMS personnel (83%). Bystander CPR including mouth-to-mouth ventilation was associated with a significantly increased risk of regurgitation compared with no CPR ($p < 0.013$) and compression-only CPR ($p<0.01$).

Simons recently studied the relationship of emesis in OOH cardiac arrest to patient survival.[25] The presence or absence of emesis was documented in 1009 cases of OOH arrest between January 1, 2004 and December 31, 2005. Among victims with cardiac arrest due to heart

disease, those with emesis experienced a trend toward lower survival to hospital discharge (13%) compared to those without emesis (18%); p = 0.10. In multi-variable logistic models, emesis was associated with a reduced odds ratio of survival after adjustment for other covariates (OR = 0.50). Simons suggests that gastric insufflation is caused by ventilations. In addition, poorly located (inferiorly-placed) chest compression may increase intra-abdominal pressure. In light of the reluctance of bystanders to perform ventilations, the high incidence of regurgitation with ventilations, and a lower odds ratio for survival, both Virkkunen and Simons suggest that an approach to CPR that prevents emesis should be considered, i.e., compression-only CPR or decreasing the ventilation-to-compression ratio.

What about Compressions without Ventilations?

A meta analysis in 1991 of 17 studies showed that individuals receiving bystander CPR were 4.5 times more likely to survive.[26] Yet in OOH cardiac arrest, bystanders initiate CPR only 20-30% of the time.[27] A great impediment to initiation of bystander resuscitation efforts is the aversion to mouth-to-mouth resuscitation because of the concern for transmission of infectious disease. In addition, mouth-to-mouth ventilation is a difficult psychomotor skill to learn and retain. The current *AHA Guidelines* state that compression-only CPR should be provided if the rescuer is unable or unwilling to do ventilations.[2]

In 2002 Kern et al from the University of Arizona Sarver Heart Center reported on a study in which 30 swine underwent 3 minutes of untreated VF before 12 minutes of Basic Life Support (BLS) CPR.[28] Animals were randomized to receive either standard airway, breathing, and compression (ABC) CPR in a 15:2 ratio or continuous chest compression (CCC) CPR by an experienced resuscitation research technician. Compressions were metronome-guided at 100/minute. In the ABC group compressions were stopped while expired-gas ventilations were delivered using a bag/valve/mask device over 16 seconds – a model similar to the typical single-rescuer OOH cardiac arrest. Defibrillation shocks were initiated at 15 minutes into cardiac arrest, and ACLS protocols were followed related to medications. Outcome results are seen in the table below.

Table 3 Outcome Results Comparing ABC CPR and CCC CPR (Kern)

Survival	ABC CPR (15 animals)	CCC CPR (15 animals)	P value
ROSC	6	13	0.01
30 minutes	5	13	0.004
2 hours	5	13	0.004
24 hours	4	13	0.004
24 hours and “good” neuro	3	12	0.002
24 hours and “normal” neuro	2	12	0.0001

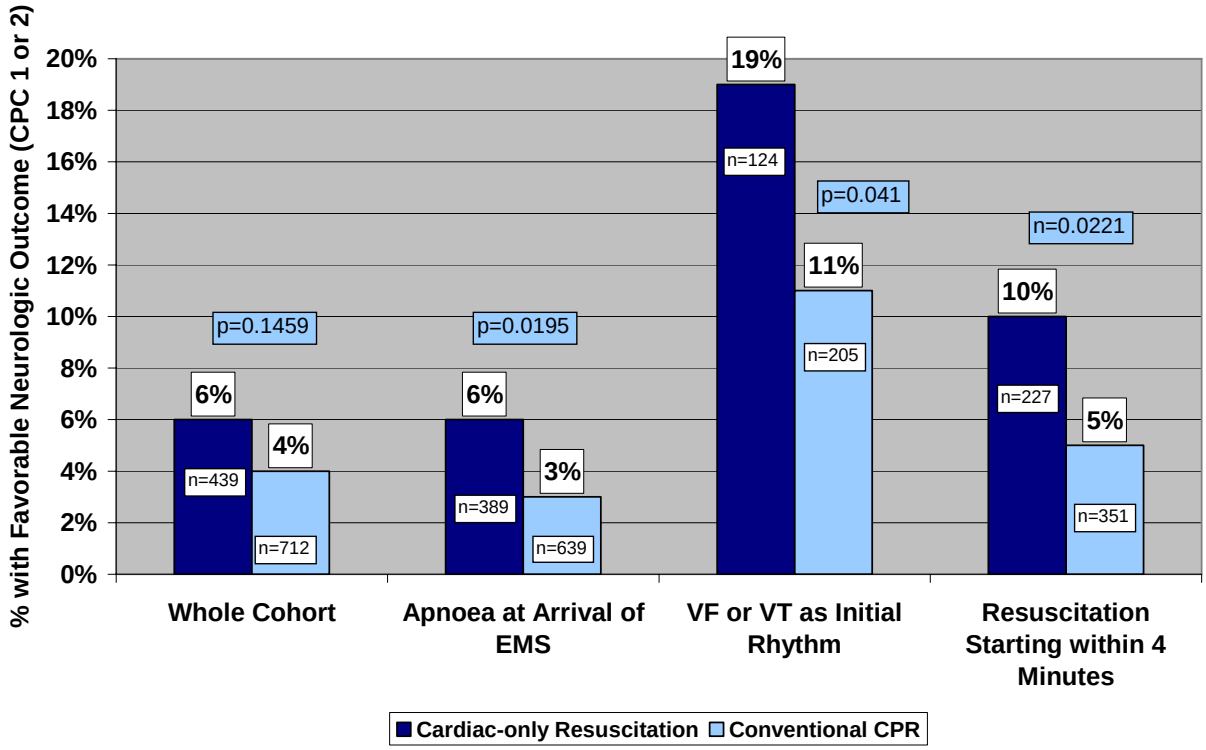
"Normal" neurologic outcome was defined as CPC score of 1 only, and "good" was defined as CPC 1 or 2. Survival and neurologic outcomes were not just significantly better, but dramatically better in the group who received CCC CPR.

Kern also reported on hemodynamics with this study. When CPP was measured during CPR in the usual fashion as the mid-diastolic aortic pressure minus right atrial pressure, it was significantly greater with ABC CPR during the mid portion of the resuscitation period (mean 21 vs. 18 mmHg). However, when CPP was 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 up to double the perfusion compared to the frequently interrupted standard technique. The ABC CPR group had an average of 496$\pm$7 total chest compressions during the 12-minute BLS period compared with 1111$\pm$4 for the CCC group. Significantly better oxygenation and ventilation were seen with ABC CPR, but CCC CPR achieved substantial oxygenation and ventilation as well. Kern concludes: "These improved outcome results with CCC CPR 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."

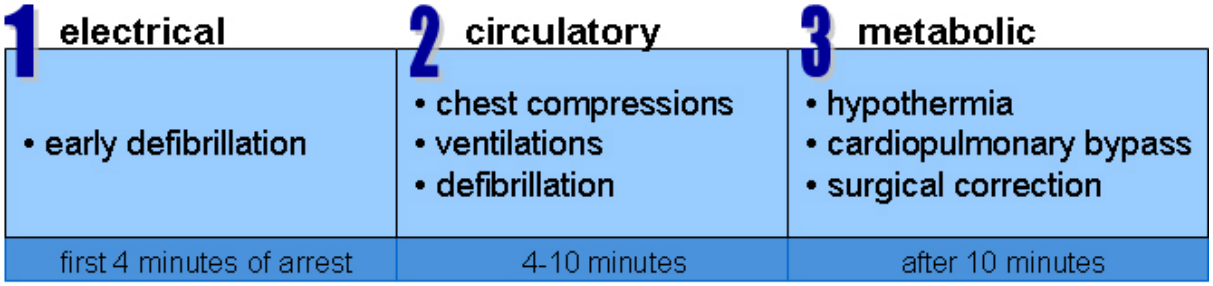
Investigators from Japan performed a prospective, multi-center, observational study designated SOS-KANTO with adult patients who had witnessed OOH cardiac arrest from September, 2002 through the end of 2003.[1] On arrival at the scene, paramedics assessed the technique of bystander resuscitation. They reported on 4068 adult patients with witnessed arrest, of whom 439 (11%) received cardiac-only resuscitation from bystanders, 712 (18%) had conventional CPR, and 2917 (72%) received no bystander CPR. CPR performed was consistent with the *2000 Guidelines*. The primary endpoint was favorable neurological outcome (defined as CPC 1 or 2) 30 days after cardiac arrest. The group who had any resuscitation attempt had significantly higher frequencies of favorable neurological outcome at 30 days than the no bystander resuscitation group (5% vs. 2%; $p<0.0001$). Although the frequency of favorable neurological outcome at 30 days did not differ between the cardiac-only resuscitation group and the conventional CPR group for the whole cohort, cardiac-only resuscitation resulted in a higher proportion of patients with favorable neurological outcomes than conventional CPR in the subgroups of patients with apnea at arrival of Emergency Medical Services (EMS), with VF or pulseless ventricular tachycardia as the initial rhythm, and when resuscitation was started within 4 minutes of discovering the victim. There was no evidence for any benefit from the addition of mouth-to-mouth ventilation in any subgroup of patients. See Figure 13. The proportion of patients *surviving* at 30 days showed no differences between the two bystander resuscitation groups, and these two groups also had similar frequencies of survival until hospital admission. This report shows that bystander cardiac-only resuscitation is equivalent or superior to conventional bystander CPR in adult patients with witnessed OOH cardiac arrest in terms of neurological benefit. The researchers suggested that interruption of chest compressions was the

main reason why conventional CPR did not result in better neurological outcome than cardiac-only resuscitation.

Figure 13 Patients with Favourable Neurological Outcome at 30 Days after Cardiac Arrest (SOS-KANTO)



The University of Arizona Sarver Heart Center CPR Research Group advocates that cardiocerebral resuscitation (CCR) replace standard resuscitation for OOH cardiac arrest.[29] Their model is built on the 3-phase time-dependent concept of cardiac arrest caused by VF that is articulated by Weisfeldt and Becker.[30]



3-Phase Time Sensitive Model of Resuscitation

The first phase, the *electrical* phase, extends from the time of arrest to approximately 4 minutes following the arrest. The most effective intervention during this phase is defibrillation. The second phase of cardiac arrest caused by VF is the *circulatory* phase, which lasts for a variable period of time, but approximately minute 4 to minute 10 of the arrest. During this time, generation of adequate cerebral and coronary perfusion pressure through CPR is critical to

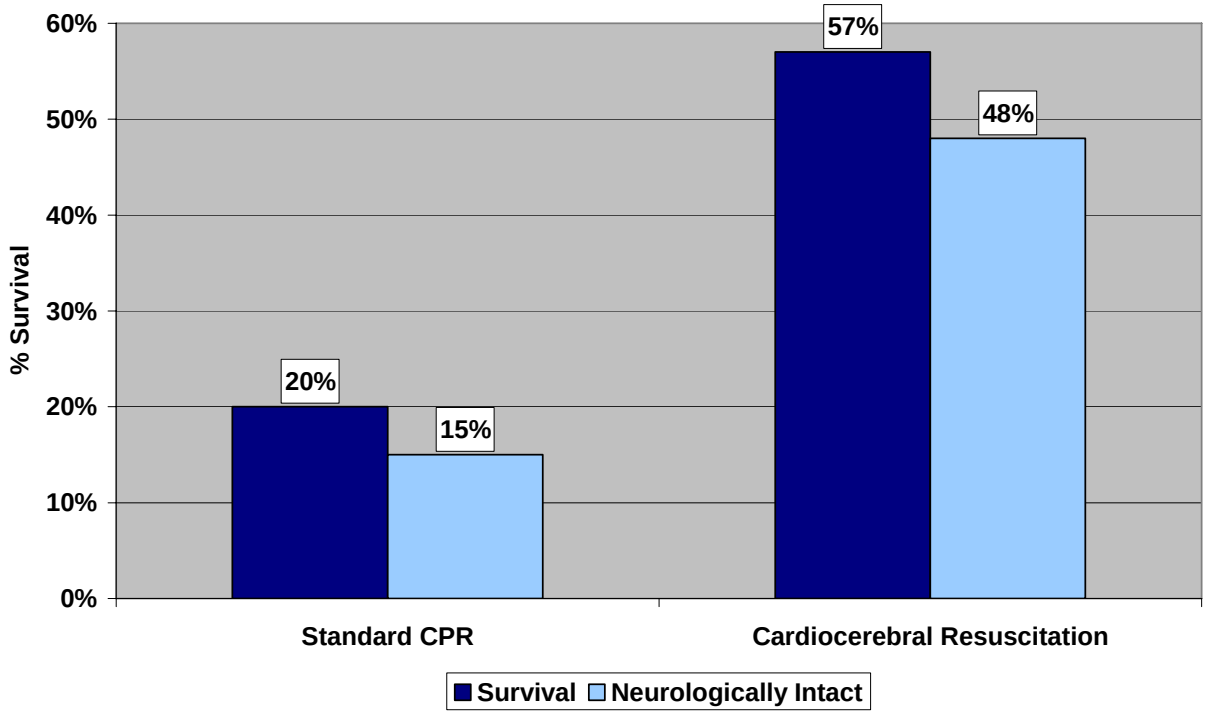
neurologically normal survival. Ironically, if an AED is the first intervention applied during this phase, survival is much less likely. It is thought that limited circulation of blood with CPR partially restores substrates, including oxygen, and washes out deleterious metabolic factors that have accumulated during ischemia. Once the heart is "primed" through CPR, it can then respond to defibrillation with restoration of a cardiac rhythm and effective pumping.

After approximately 10 minutes of cardiac arrest, in the *metabolic* phase, the effectiveness of both immediate defibrillation and CPR followed by defibrillation decreases rapidly and survival rates are poor. Tissue injury from global ischemia and from reperfusion can result in circulating metabolic factors that cause additional injury to the whole body. Interventions that may be of use during the metabolic phase include the use of hypothermia, cardiopulmonary bypass, and surgical correction of the underlying cardiac pathology.

With CCR, lay rescuers are taught to call 911 for a witnessed, unexpected collapse in an adult, and. after placing the victim on his back, to begin dispatcher-coached compressions at 100/minute without any ventilations. Once the paramedics arrive, they give 200 chest compressions prior to and after defibrillation. They open the patient's airway with an oropharyngeal device, put in place a non-rebreather mask, and administer high-flow oxygen at about 10 L/minute. Intubation is deferred until after at least 3 series of 200 compressions and 3 shocks. EMS protocols in this CCR model were also changed to minimize interruptions of chest compressions.

Kellum was the first to report survival rates in humans using the CCR model for prehospital care in two rural Wisconsin counties from early 2004 through May, 2005.[31] Dispatchers were trained to instruct 911 callers to perform compression-only CPR. When the paramedics arrived, they performed CCR as described above. If the arrest was witnessed, and down time was less then 12 minutes, rescue breaths and assisted ventilations were not performed until after the ROSC or until after 3 cycles of chest compressions followed by rhythm analysis with or without a shock were completed. Survival was compared to retrospective cases treated during the 2001 to 2003 period. During the 3-year period when standard CPR was utilized, there were 92 adult patients with witnessed cardiac arrests and an initially shockable rhythm. Eighteen of these 92 patients (20%) survived, and 14 of these 92 (15%) survived neurologically intact. After the CCR protocol was initiated, there were 33 such patients. Nineteen of these 33 patients (57%) survived, and 16 of the 33 (48%) were neurologically intact. See Figure 14. The differences in both total survival and neurologically normal survival are significant (Chi-squared = 0.0001). Among survivors, shocks were delivered within 7 minutes in 87% of patients during the standard CPR control period and in 47% of the patients during the CCR project period.

Figure 14 Survival Outcomes with Standard CPR vs. Cardiocerebral Resuscitation (Kellum)



The AHA is eager to see scientific data from programs in which compression-only CPR is prospectively promoted as part of a comprehensive community program designed to improve survival from out-of-hospital cardiac arrest.[32] It will also be helpful to see studies that evaluate outcomes from resuscitation protocols that are consistent with the more recent *2005 AHA Guidelines*.

What is the Optimal Ratio for Compressions:Ventilations?

Despite detrimental effects of ventilation, scientific evidence does not support abandoning ventilations in favor of chest compression-only CPR. Aufderheide states: "Several well established laboratories have previously demonstrated that chest compression-only CPR can be as effective as chest compressions and rescue breathing during the first 6-12 minutes of cardiac arrest in the animal model.[33] The persistence of an open airway in these models, allowing for ventilation produced by chest compressions alone or in conjunction with spontaneous gasping, may not reflect physiology in human resuscitation." Safar adds: "Rats, pigs, and dogs have straight airways which do not obstruct, even in the absence of a tracheal tube. Humans have kinked airways which tend to obstruct in comatose patients."[34]

Dorph attempted to produce a more physiologic model of CPR in VF arrest, by using an occluded airway in the pig model to simulate human airway collapse and prevent passive inspiratory airflow generated by chest compressions.[35] He then compared acid-base status and ROSC in pigs treated with chest compression-only CPR vs. a compression:ventilation ratio of 30:2. With a compression:ventilation ratio of 30:2, the arterial oxygen content stayed at 2/3 of

normal, but with compression-only CPR, the arterial blood was virtually desaturated with no arterio-venous oxygen difference within 1.5-2 minutes. The median time to ROSC was significantly shorter in the ventilated group (1.5 vs. 6.7 minutes, $p < 0.05$). These data support the conclusion that in cardiac arrest with an obstructed airway (the most common scenario in human cardiac arrest), pulmonary ventilation should still be strongly recommended.

The optimal compression:ventilation ratio has yet to be defined, but it is likely to be considerably greater compressions and fewer breaths than has been clinically practiced. Research is ongoing with swine to study various compression:ventilation ratios. For example, Sanders studied survival, neurologic outcome, and hemodynamics with 15:2, 50:5, 100:2, and chest compression-only CPR.[36] In 2003 Dorph reported on carotid blood flow, hemodynamic pressures, and blood gases with ratios of 15:2, 50:2, and 50:5 CPR.[37]

Ewy, from the University of Arizona, suggests that for a sudden, witnessed arrest, the initial rescuer do four minutes of compression-only CPR, then, if willing to do rescue breathing, perform 1 to 2 ventilations before each set of 100 compressions.[38] Clearly excessive ventilation is to be avoided, but once healthcare providers arrive are the recommended 10-12 ventilations/minute optimal? Ewy also suggests that ventilations probably can be withheld during the first 15 minutes of chest compressions as long as the patient is gasping. But these and other models need to be tested.

Are There Alternative Approaches to Ventilation during CPR?

Several other models have been suggested for ventilation during CPR. Kern proposes an AC-B sequence (airway-circulation, and much later breathing) for victims who have not had a primary respiratory arrest.[20] Anyone performing CPR should first assure an open airway by aligning the proper head and neck position so that gasping can occur. He believes that the time wasted to perform two ventilations at the beginning is not justified since patients probably have oxygenated air in their lungs, and oxygenated blood in their pulmonary veins, left atrium, left ventricle, and entire arterial system at the time of arrest. Emphasis is placed instead on compressions to circulate the oxygenated blood. In addition, prompt administration of compressions while the individual is still gasping often results in continued gasping.[38]

Recently, Hayes, in a porcine model of OOH VF cardiac arrest, managed ventilation with passive oxygen insufflation.[39] No bystander CPR was provided during the first 8 minutes of untreated VF before the simulated arrival of an EMS. Thirty-six swine were randomly assigned to one of 3 experimental groups. All groups were given 200 continuous chest compressions at a metronome-directed rate of 100/minute with the randomized ventilation regimen followed by a shock and further ACLS. Group 1 (standard ventilation) was mechanically ventilated at 10 respirations/minute (RPM) at a tidal volume (TV) of 10 ml/kg with 100% oxygen. Group II (hyperventilation) was ventilated at 35 RPM at a TV of 20 ml/kg with 100% oxygen. In Group III (insufflation) animals, a nasal cannula was placed in the oropharynx to administer oxygen continuously at 10L/minute. There were no significant differences in the 24 hour neurologically normal survival among groups (standard: 2/12, hyperventilation: 2/12, insufflation: 4/12; $p = .53$). Insufflation resulted in respiratory acidosis within 2 minutes after the initiation of CPR. As expected, the pCO_2 was significantly higher in the insufflation group. In contrast, respiratory

alkalosis developed from positive pressure ventilation. The arterial oxygen levels were lower with insufflation, but these differences in pH, pCO_2, and pO_2 did not prevent similar outcomes.

Providing oxygen insufflation during cardiac arrest has recently been reported in humans with OOH cardiac arrest. Kellum's study of cardiocerebral resuscitation in rural Wisconsin discussed on page 9 used this method for oxygenation, and showed positive outcomes related to neurologically intact survival.[31]

Perhaps care providers will be advised in the future to place an oropharyngeal airway and deliver high-flow oxygen via a non-rebreather mask during the initial minutes of resuscitation, which would avoid the deleterious effects from positive pressure ventilation and hyperventilation. The responder's emphasis could then turn to continuous compressions.

Conclusion

Most authors agree that there should be different approaches to cardiac and respiratory arrests. With asphyxial arrest, such as choking or drowning, the arterial blood becomes so desaturated that it contributes to hypotension and secondary cardiac arrest. Increased ventilation may be needed initially to clear alveolar CO_2 (or water) that accumulated during asphyxia and to increase the alveolar concentration of oxygen. Is the current rate of ventilations in the *AHA 2005 Guidelines*, i.e., 10-12 ventilations/minute, sufficient to maintain a best ventilation/perfusion match or is a higher ventilation rate needed under these circumstances?

During the initial few minutes of sudden cardiac arrest, providing blood flow to the myocardium and central nervous system is crucial to resuscitation outcome and appears more important than ensuring that additional inspired oxygen is provided via positive pressure ventilation. Will the AHA recommend compression-only CPR for the lay rescuer in hopes that bystander CPR will increase? Perhaps a first step in the CPR sequence will be assuring that there is an open airway, then immediately proceeding with compressions. When the health care provider takes over CPR, will the AHA suggest longer periods of compression before stopping for ventilation? The optimal compression: ventilation ratio has not been defined, but is likely to be considerably greater compressions and fewer breaths than has been clinically practiced. The issue is not whether to ventilate during CPR, but how to optimally match alveolar ventilation with pulmonary blood flow, i.e., the ventilation/perfusion ratio. Perhaps a 50:2 ratio will be suggested for one or two professional rescuers when compressions are provided without adjunctive devices, e.g., AutoPulse®. In this model, there would be fewer interruptions for ventilations, when the blood flow falls to zero; gastric inflation with subsequent regurgitation and aspiration would be reduced. Ongoing research is needed to show the way for the future of CPR.

What should we do at the present time? Systems to monitor the quality of CPR with real-time feedback should be implemented as part of a hospital's ongoing quality improvement programs (i.e., you cannot monitor what you cannot measure). Methods should be developed to improve education and training techniques for CPR, particularly student awareness of adherence to current guidelines and quality of CPR delivered. Use of ventilation-assist timing cues during resuscitation should be utilized. We should assure that hyperventilation does not occur, and that the airway is kept open during CPR. **Bottom line:** We should concentrate on performing the best CPR possible according to the current AHA guidelines until the recommendations change.

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