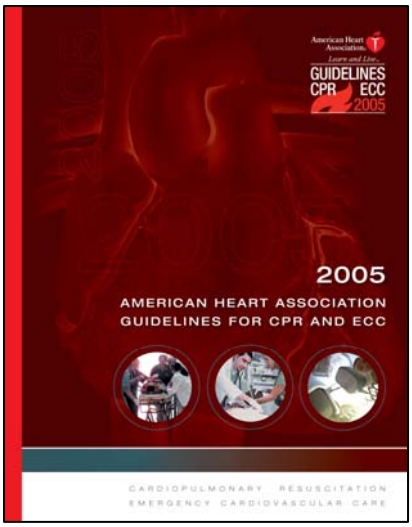
TO VENTILATE OR NOT TO VENTILATE: THAT IS THE QUESTION!
Part I

Over the past several years healthcare practitioners have heard it suggested that perhaps ventilations are not needed in the cardiopulmonary resuscitation (CPR) sequence. The public is even questioning the need for emergency breathing with the recent news headlines from the SOS-KANTO group in Japan who reported that victims receiving compression-only CPR by bystanders for out-of-hospital (OOH) cardiac arrest had a higher survival rate compared to those receiving standard CPR.[1] The *American Heart Association (AHA) Guidelines for CPR and Emergency Cardiovascular Care* have seemingly put less emphasis on the ventilation component of CPR with the compression:ventilation ratio increasing from 5:1 to 15:2 in 2000 to 30:2 in 2005 for initial rescuers until a secured airway is in place. In the August and September issues of the *Code Communications* newsletters we will explore the ventilation issue by answering the following questions:

a. What are the current *AHA Guidelines* related to ventilation?
b. Do rescuers perform ventilation according to the *AHA Guidelines*?
c. What are the consequences of hyperventilation?
d. What methods could help us perform ventilations correctly?
e. How often does gasping occur and what are its benefits?
f. How well do rescuers assess breathing at an arrest?
g. How do interruptions for ventilations affect blood flow during CPR?
h. What are the physiologic gains from ventilation during resuscitation?
i. What harm can result from ventilations during resuscitation?
j. What does research show us about providing compressions without ventilations?
k. What is the optimal compression: ventilation ratio?

2005 American Heart Association Guidelines Related to Ventilation

In the *2005 AHA Guidelines* a universal compression:ventilation ratio of 30:2 is recommended for lay rescuers of infant, child, and adult victims until healthcare providers take over.[2] Healthcare providers should use a 30:2 ratio for all 1-rescuer and all adult CPR and a 15:2 ratio for infant and child 2-rescuer CPR. Once the airway is secured with an endotracheal tube (or other advanced airway), compressions should continue at 100/minute without interruption while ventilations are given unsynchronized at 8 to 10 breaths/minute for the adult (i.e., 1 breath about every 6 to 8 seconds) and a rate of 12 to 20 breaths/minute for the infant and child (i.e., 1 breath every 3 to 5 seconds).



When the rescuer is alone, the sequence of initial actions varies for the layperson based on the *age* of the victim and for the healthcare provider based on the *etiology* of the arrest. If the collapse of a victim of any age is sudden and witnessed, the healthcare provider should first phone the emergency number and get an automated external defibrillator (AED) if available. S/he should then begin CPR and use the AED as appropriate. When an asphyxial cause of arrest (e.g., drowning, injury, drug overdose) is presumed, the healthcare provider should deliver 2 minutes of CPR before leaving the victim to phone the emergency number and get the AED. On the other hand, the

lay rescuer responding to an adult victim should telephone and retrieve an AED prior to beginning CPR and using the AED. When the victim is a child, the lay rescuer is taught to provide 2 minutes of compressions and ventilations before leaving to telephone and get an AED.

Emergency Medical System (EMS) medical directors may consider implementing a protocol that would allow EMS responders to initially provide about 2 minutes of CPR before attempting defibrillation when the EMS system call-to-response interval is greater than 4 to 5 minutes.

For the patient with a respiratory arrest and a perfusing rhythm, healthcare workers are taught to deliver rescue breaths without compressions at a rate of about 10 to 12 breaths/minute (i.e., 1 breath every 5 or 6 seconds) for the adult, a rate of about 12 to 20 breaths/minute for the infant and child, and a rate of 40 to 60 breaths/minute for newborn infants. Teaching of rescue breathing without chest compressions is no longer taught to lay rescuers as a means to "make CPR easier for all rescuers to learn, remember, and perform."

When lay rescuers check breathing in the unresponsive adult victim, they should look for *normal* breathing. When they check for breathing in the infant or child, the lay rescuer should look for the presence or absence of breathing. The basic healthcare worker should continue to assess for *adequate* breathing in the adult. Assessment of breathing should take 5 to 10 seconds, but no more than 10 seconds.

Each rescue breath should be delivered in 1 second and should produce visible chest rise. Lay rescuers are now taught to perform another head tilt-chin lift if the first breath does not make the chest rise. Healthcare providers should take particular care to provide *effective* breaths in infants and children even if this means attempting to open the airway several times. The rescuer should take a normal – not a deep – breath before giving a rescue breath. When using an adult ventilating bag, a 1-L bag should be compressed about halfway, and a 2-L bag about one third, to produce a visible chest rise.

Interruptions in compressions should be minimized. Thus, insertion of an advanced airway may be deferred for several minutes as long as adequate bag/valve/mask ventilation with oxygen is being delivered. The choice of advanced airway varies based on provider experience, EMS or healthcare system characteristics, and patient condition. Confirmation of endotracheal tube placement requires both clinical assessment and use of a device (e.g., exhaled CO_2 detector, esophageal detector device). Use of a device is part of primary confirmation and is no longer considered secondary confirmation.

Quality of Ventilations Delivered During Resuscitations

Do rescuers perform the skill of ventilation according to the *AHA Guidelines*? In 2004 Aufderheide and Lurie reported on a clinical observation study of ventilations performed by EMS providers in the Milwaukee area during OOH cardiac arrest.[3] A team of researchers used a Propaq monitoring system to record ventilation frequency and airway pressures from the endotracheal tube of 13 consecutive adults undergoing CPR. The average ventilation rate for the 7 patients in group 1 was 37 $\pm$ 4 breaths/minute (range, 19 to 49 breaths/minute). After

recognizing that rescuers were consistently hyperventilating patients in cardiac arrest, investigators immediately retrained all EMS personnel to provide ventilations at a rate of 12 breaths/minute during CPR after establishment of a secured airway. After retraining, 3 of the 6 patients in group 2 still had ventilation rates of $\geq$ 26 breaths/minute. The average ventilation rate for these six patients was significantly slower at 22 $\pm$ 3 breaths/minute than group 1 patients. Although ventilation rates were slower after retraining in group 2, average ventilation duration was significantly longer than group 1 patients (1.18 $\pm$ 0.06 vs. 0.85 $\pm$ 0.07 seconds/breath). As a result, the average percentage of time in which a positive pressure was recorded in the lungs was similar in group 2 and group 1 patients (44.5 $\pm$ 8.2% vs. 50 $\pm$ 4%, respectively; p = not significant). Combining groups 1 and 2, the average ventilation rate for all 13 patients was 30 breaths/minute – more than twice that recommended by the AHA. This was the first time that ventilations had been objectively and electronically recorded during CPR that was performed by professionals at OOH cardiac arrests.

An in-hospital observational study was performed by Abella and colleagues at the University of Chicago Hospitals.[4] His group observed 67 patients undergoing CPR performed by nurses and medical students certified in Basic Life Support (BLS) or by physicians certified in Advanced Cardiac Life Support (ACLS) from December 2002 to April 2004. Ventilation data were obtained using impedance measurements captured from defibrillator pads. Analysis of the first 5 minutes of resuscitation by 30-second segments showed that during 60.9% of the segments, ventilations were performed at a rate greater than 20/minute. Ventilation volumes did not appear to deviate greatly from physiological ranges and were not reported.

O'Neill observed detailed ventilation variables prospectively during manual ventilation of 12 OOH cardiac arrest patients treated in the emergency department of a United Kingdom general hospital.[5] Resuscitation attempts were made according to the European Resuscitation Council 2000 guidelines that were in place at the time of the study. All patients were intubated and ventilated using a bag/valve/mask device by a member of the resuscitation team, usually a senior emergency physician. The respiratory rate was found to be at least double that recommended in 9 of the 12 patients (75%) while the tidal volume was no higher than the recommended 10 ml/kg in 9 of the 12 patients (75%). This is the first study to report human in vivo tidal volumes during cardiopulmonary resuscitation. In 11 of the 12 patients (91.7%), the airway pressure remained positive for more than 90% of the time. This finding of high airway pressure for a more prolonged period of time than in Aufderheide's study may be due to the fact that a LUCAS® automatic device was used to deliver compressions in O'Neill's study.

Not only are victims hyperventilated during resuscitation, but the rescuer also shows signs of hyperventilation. Theirbach asked 20 healthy volunteers (medical students and health care providers) to perform two-rescuer CPR using mouth-to-mouth ventilations at a ventilation:compression ratio of 2:15 for 5 minutes according to the *2000 Guidelines* in a lung model connected to a BLS-manikin.[6] Remember that rescuers at that time were taught to take a deep inspiration before every breath. Fifteen of the 20 participants complained of hyperventilation-associated symptoms while performing ventilations, e.g., paresthesia, dizziness, carpopedal spasms. These symptoms most often occurred during the first three minutes of CPR. None of the participants stopped their ventilations due to symptoms. During the ventilation period the median value for $EtpCO_2$ decreased significantly from 38 to 24 mmHg over the total

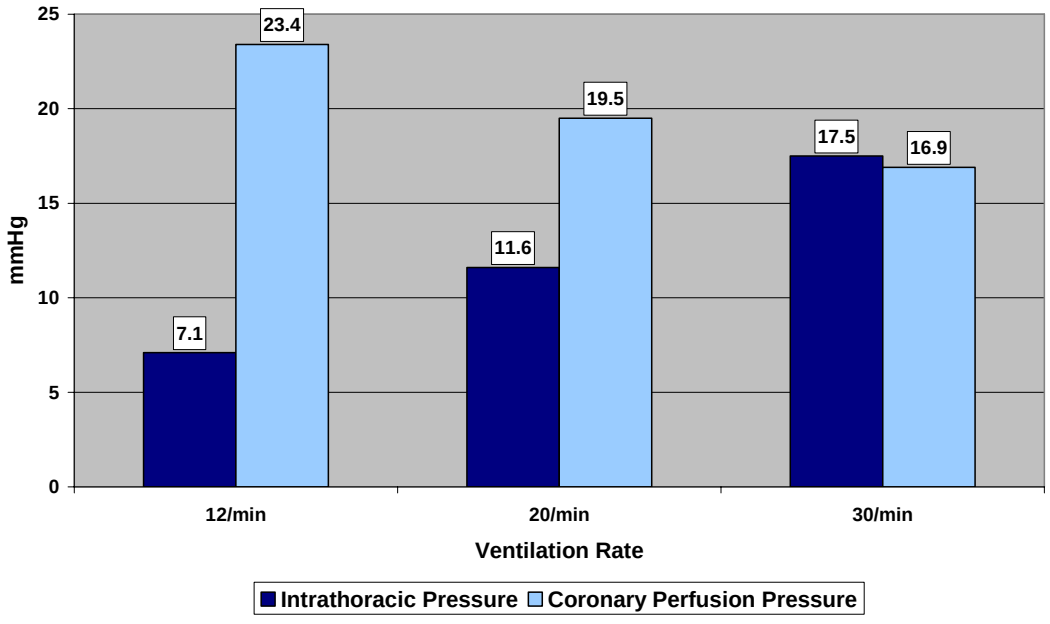
measurement period, as well as between the individual measurement points during the first 3 minutes (p = 0.0001). Values as low as 20 mmHg were detected in 3 participants. These alterations of the CO_2 pressure were confirmed by blood gas analysis. Thus, revisions in the *2005 Guidelines* for the rescuers to change positions every two minutes may assist the person performing ventilations as well as the one performing compressions. The recommendation to take a deep breath prior to delivering mouth-to-mouth ventilation was eliminated from the *Guidelines* in 2005.

In conclusion, these scientific studies show us that rescuers are not performing ventilations according to the *AHA Guidelines*. Hyperventilation of the victim during cardiopulmonary arrest is occurring due to a high rate of ventilations, rather than a high tidal volume, and positive pressure is evident in the lungs for greater than 50% of the time. Even after manikin retraining, healthcare providers still provide ventilations at a higher rate than recommended. It continues to be a challenge to translate theory and manikin-based training into practice.

Consequences of Hyperventilation

Aufderheide brilliantly describes the consequences of hyperventilation in his research report entitled "Death by hyperventilation: A common and life-threatening problem during cardiopulmonary resuscitation."[3] In protocol 1, 9 pigs were placed in ventricular fibrillation (VF) cardiac arrest and left untreated for 6 minutes. Compressions were then delivered by an automatic piston device (CPR Controller) at a rate of 100/minute. After the initial 2 minutes of CPR, each animal received ventilations delivered asynchronously (each over 1 second) in a random order at 12, 20 and 30 breaths/minute with each phase lasting 2 minutes, and physiologic variables were measured. Note in Figure 1 that with increasing ventilation rate, the mean intrathoracic pressure increases (p<0.0001) and the coronary perfusion pressure (CPP) drops (p=0.03).

Figure 1 Mean Intrathoracic Pressure and Coronary Perfusion Pressure Related to Ventilation Rate (Aufderheide)



Arterial blood gas results for Aufderheide's study are shown in Table 1. Increased ventilation rate was associated with significantly higher arterial pH, but no significant change in PaO_2.

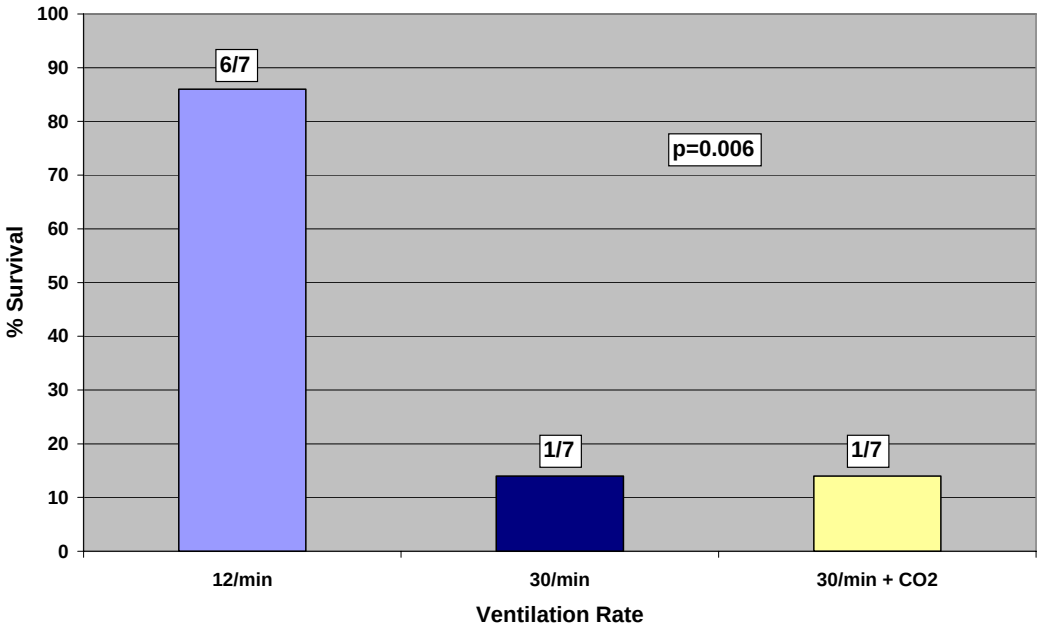
Table 1 Changes in Arterial Blood Gases with Three Different Ventilation Rates (Aufderheide)

Arterial blood gases	Ventilation Rate, Breaths/Minute			P Value
	12	20	30	
pH	7.34+0/02	7.45 + 0.03	7.52 +0.03	0.0006
PaCO2, mmHg	22.7 + 2.7	15.6 + 2.2	11.6 + 1.5	0.005
PaO2, mmHg	340.9 + 40.7	403.3 + 47.0	403.7 +48.0	0.59

Aufderheide speculates that hyperventilation and prolonged ventilation intervals result in persistently positive intrathoracic pressure during the decompression phase of CPR, thereby decreasing cardiac preload and cardiac output and impeding right ventricular function. The intrathoracic pressure never went below 0 mmHg when the ventilation rate was 30/minute in this study. There was never enough time to allow for the development of negative intrathoracic pressure between compressions.

The purpose of protocol 2 in Aufderheide's study was to evaluate whether excessive ventilation rates were associated with increased mortality rates. Twenty-one pigs in VF cardiac arrest were left untreated for 6 minutes, followed by CPR administered with a 5:1 ratio for 2 minutes, stopping compressions for the ventilations. Then the animals were randomized into 3 groups to receive ventilations delivered asynchronously at either 12 breaths/minute with 100% oxygen, 30 breaths/minute with 100% oxygen, or 30 breaths/minute with 5% CO_2/95% oxygen. At the end of each protocol the animals were shocked up to 3 times, and survival was assessed. Figure 2 shows that the excessive ventilation rates were associated with decreased survival rates in this animal model of cardiac arrest. Survival was defined as a stable blood-perfusing rhythm generating a measurable blood pressure over the first hour of observation after resuscitation. Thus, unrecognized and inadvertent hyperventilation could be contributing to the current dismal survival rates in cardiac arrest.

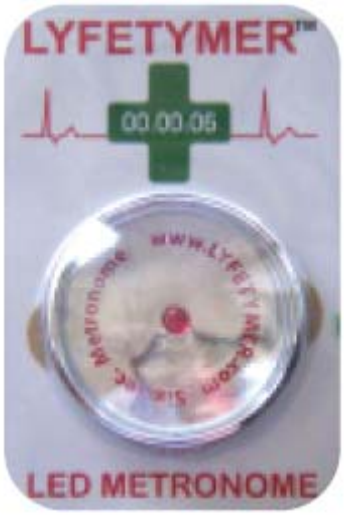
Figure 2 Survival Study (Aufderheide)



What Methods Help Rescuers Keep Ventilation Rates Low?

Keeping ventilation rates low during a code is difficult because the high-adrenalin state of rescuers alters time perception, and the rapidly refilling bag/ventilation system sets up a reflex in which rescuers deliver breaths as soon as the bag refills. So how can we help rescuers slow their ventilation rate and thus improve survival? We should certainly inform providers during training courses of the deadly nature of hyperventilation and coach them to ventilate at the proper rate and duration. But Aufderheide reported that when EMS responders were found to ventilate during OOH arrests at a rate of 37/minute and underwent retraining, their rates dropped only to an average of 22/minute.[3]

Figure 3 Lyfetymer™



So what devices are available that provide cues for timing of ventilations? The disposable 3.5 cm. diameter Lyfetymer™ metronome blinks every 6 seconds; see Figure 3. It quickly adheres "anywhere in your sight line with 3M adhesive". Customers are known to place the device on the bag/valve device. Information can be found at: http://www.lyfetymer.com/index.php. The Lyfetymer is available @$5 for a single device (and quantity discounts) from Tri-anim.

Another one-time use device is the ResQPOD, which is placed between a bag/valve device and the resuscitation mask or between the bag/valve device and endotracheal tube; see Figure 4. It has a switch to activate the light, which blinks at 10/minute in order to guide ventilation rate. The device was originally designed as a circulatory aid, selectively impeding inspiration gases from coming into the lungs during the release phase of CPR. This action increases the negative intrathoracic pressure and creates a greater vacuum in the chest, resulting in greater venous return. On the subsequent compression phase of CPR, cardiac output is increased. Studies have shown that blood flow back to the heart is doubled, and 70% of the normal blood flow is provided to the brain when using the ResQPOD. The device is manufactured by Advanced Circulatory Systems, and sold by Tri-anim. Information about the ResQPOD can be found at: http://www.advancedcirculatory.com/cet/resqproduct.htm.

Figure 4 ResQPOD

The PocketCPR™ device, manufactured by ZOLL Medical Corporation, is available for training currently, but FDA clearance is pending for clinical application; see Figure 5. The puck is placed onto the manikin's (or patient's) sternum, and compressions are performed over it. The device provides verbal instructions for the initial rescuer sequence, which includes cues for when to ventilate. Coaching of the rescuer is accomplished through voice prompts, metronome beeps, and light cues to achieve the optimal rate and

Figure 5 PocketCPR™

depth of compressions. The PocketCPR training device is currently being sold by Channing Bete Company. Information can be found at:
http://store.channing-bete.com/onlinestore/storeitem.html?vid=20050104002&iid=175031&lang=0&pos=4&icode=2019-PCPR&pcode=&item=The+PocketCPR%28TM%29

Figure 6 AutoPulse®

If the AutoPulse® is being used to provide compressions with the load-distributing LifeBand®, audio tones are available to cue ventilations. See Figure 6. When the AutoPulse is set up in a 30:2 ratio, audio tones sound on the 28th, 29th, and 30th compressions, just prior to the pause which allows the rescuer to administer ventilations. Once the mode is set to Continuous, an audio tone will sound 8 times/minute to cue ventilations. For more information about the AutoPulse, go to the ZOLL web site: http://www.zoll.com/autopulse.

It is very important for the quality of CPR – including ventilations – to be monitored in real time during codes. The leader of the resuscitation team, who stands back and does not perform interventions, should be guiding the responders with actions to take and monitoring their care.

Figure 7 Q-CPR Display on Philips HeartStart MRx

Objective measurement of ventilations is available with Philip's Q-CPR™ technology on their HeartStart MRx monitor/defibrillator. Ventilation data is collected via the same chest electrodes used for defibrillation. These pads detect changes in chest impedance, which are interpreted by the MRx and then displayed as lung volume and ventilation rate on the monitor screen; see Figure 7. Note the ventilation indicator showing lung volume alongside the calculated ventilations/minute (vpm) value. If either the volume or rate falls outside target range, the MRx provides on-screen signals and audible feedback. Both Philips and Medtronic Physio-Control now provide printed feedback on ventilation in their defibrillator data management software packages, so the quality of care can be reviewed after the code is over.

Incidence and Significance of Gasping During CPR

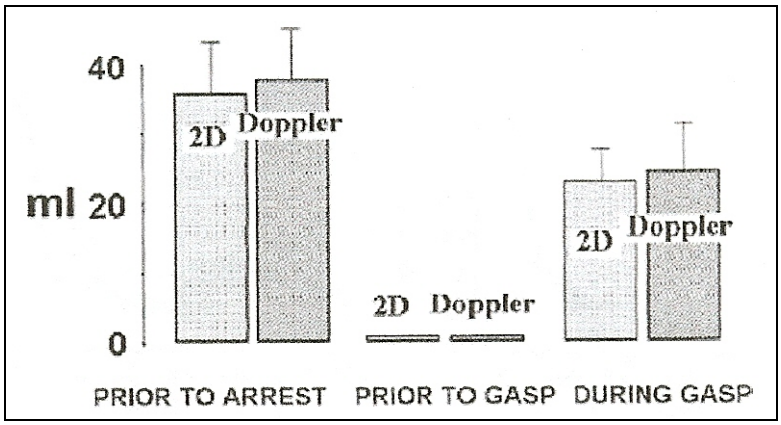
Agonal respirations, occurring at the time of or shortly before death, are characterized as being on a continuum from slow, shallow respirations seen in respiratory demise to ineffective, gasping respirations seen in sudden cardiac arrest. This discussion will focus on agonal respirations associated with sudden cardiac arrest. Eisenberg writes about a study in which researchers listened to 445 calls reporting a cardiac arrest to the emergency dispatch centers.[7] For witnessed cardiac arrest, agonal respirations occurred in 55% compared with 16% of unwitnessed arrests. The identification of agonal breathing came from the callers' descriptions and actually hearing the agonal sound in some recordings. Of the patients in VF, 56% had agonal respirations compared with 34% of patients without VF. It was estimated that agonal respirations last approximately

four minutes in at least one-third of cases. 27% of patients with agonal respirations were discharged alive compared with 9% of patients without agonal respirations.

In 1923 Lumsden described the location of the "gasping center" as the brainstem.[8] Gasping is a common mammalian terminal phenomenon that occurs secondary to medullary ischemia or hypoxia. Animal studies have shown that gasping occurs in all animals during VF. In a rodent model of VF cardiac arrest, von Planta demonstrated that during the inspiratory phase of gasping there is a decrease in intrathoracic pressure, which creates a pressure gradient between the right side of the heart and vessels so that venous return is promoted.[9] During the expiratory phase of gasping, intrathoracic pressure increases along with aortic pressure. This results in a pressure gradient between the aorta and the right atrium, which favors coronary perfusion.

Gasping-mediated blood flow augmentation was recently reported by Xie in a study of pigs with induced VF.[10] Gasping generated 23 $\pm$ 6 ml of blood flow volume during the inspiration phase, which represented 60% of the stroke volumes of the spontaneously beating heart; see Figure 8.

Figure 8 Stroke Volumes Related to Gasping (Xie)



The increase in stroke volume that follows increased inspiratory filling of the heart with gasping increases the forward blood flow, which in turn increases CPP and coronary blood flow during cardiac arrest, which favors successful resuscitation. With cardiac arrest there is an initial decrease in cardiac output, then with gasping there is a transient partial increase in cardiac output recovery. Once gasping ceases, the cardiac output falls to zero.

Through animal studies additional benefits from gasping have been found. Srinivasan showed in a pig study that spontaneous gasping during VF arrest decreased intracranial pressure and increased cerebral perfusion pressure.[11] He concluded that this potential beneficial effect of gasping on cerebral perfusion may underlie the observed survival benefit. Gasping is also associated with improved upper airway patency[12] and improved pulmonary gas exchange during resuscitation. A sharp increase in arterial oxygen saturation and a decrease in arterial CO_2 tension typically follow the gasp.[13] Thus, gasping improves both circulation and ventilation.

How Well is Breathing Assessed in Cardiac Arrest?

In actual cardiac arrests rescuers are often confused by the presence of gasping. Bystanders at OOH cardiac arrests are frequently lulled into thinking the person is still breathing, thus identification of cardiac arrest may be missed by the dispatcher and CPR is not begun. Dispatchers should be trained to specifically ask all callers, "Is the person conscious?" If the answer is no or there is uncertainty, the dispatcher should ask, "Is the person breathing *normally*?"

Healthcare providers should do better assessment of breathing in the hospital setting, right? Perkins lead a study in which six video clips were made of an experienced anesthetist lying on the floor, simulating unconsciousness and demonstrating six different breathing patterns as follows: 1) normal breathing (normal tidal volume at a rate of 12/minute), 2) no breathing, 3) shallow breathing (reduced tidal volume at a rate of 12/minute), 4) rapid breathing (tidal ventilation at a rate of 30/minute), 5) agonal breathing with an obstructed airway, and 6) agonal breathing with a clear airway.[14] The 10 second video clips (reflects the maximum time recommended in current *Guidelines* for assessment of breathing) were shown in random order to 48 second year medical students from the University of Birmingham, U.K., who were all trained in BLS. For each clip the observer was told: "You have found this patient collapsed and unconscious." Then the observer was asked, "Is the patient breathing?" (Answers: yes, normal; yes, abnormal; or no) and "What action would you take?" (Answers: place in recovery position or perform rescue breathing). Their answers are shown in Table 2 below.

Table 2 Answers to Assessment and Treatment of Breathing Questions (Perkins)

Breathing Pattern	Breathing Present?			Treatment	
	Yes - Normal	Yes- Abnormal	No	Rescue Breathing	Recovery Position
Normal	29 (61%)	16 (33%)	3 (6%)	6 (14%)	42 (86%)
Rapid	20 (42%)	28 (58%)	0 (0%)	1 (2%)	47 (98%)
Shallow	21 (44%)	11 (23%)	16 (33%)	22 (46%)	26 (54%)
Apneic	4 (8%)	3 (6%)	41 (86%)	42 (86%)	6 (14%)
Agonal gasp (obstructed)	0 (0%)	45 (94%)	3 (6%)	15 (45%)	33 (55%)
Agonal gasp (unobstructed)	15 (31%)	33 (69%)	0 (0%)	4 (8%)	44 (92%)

Based on the recommendations of their expert group, Perkins defined the normal breathing video as "normal" breathing, apneic as "absent" breathing and agonal, rapid and shallow breathing as "abnormal" breathing. They defined the correct treatment for normal, rapid or shallow breathing as "recovery position" and for absent or agonal breathing as "rescue breathing". This study demonstrated that BLS-trained medical students were unable to reliably differentiate normal breathing from abnormal breathing shown on video clips, resulting in a high number of

inappropriate, potentially harmful actions or omissions. The diagnostic accuracy for recognizing normal breathing and realizing when it was appropriate to perform rescue breathing was only marginally better than chance alone. The sensitivity in this study was 42%, meaning that 58% of the time the victim was falsely diagnosed as not requiring rescue breathing.

These same investigators than went on to conduct a randomized, controlled trial in order to determine if specific instruction on agonal breathing improves the accuracy of checking for signs of circulation as a diagnostic test for cardiac arrest.[15] Sixty-four first year medical students were randomized to a control group who were taught the standard CPR course or to an intervention group who received standard CPR training plus specific instruction (using a slide and DVD clip) on the characteristics of agonal breathing. Two weeks after initial training, the students' ability to recognize cardiac arrest was tested using a simulated cardiac arrest victim demonstrating normal, absent or agonal breathing. The students were asked to assess an unconscious victim for signs of circulation and then state as soon as they were certain (given up to 20 seconds), how they would treat the simulated victim. The intervention group had greater diagnostic accuracy for cardiac arrest compared to the control group (90% vs. 78%, $p = 0.03$). The intervention group was more likely to recognize cardiac arrest correctly and initiate CPR than the control group (sensitivity 90% vs. 78%, $p = 0.02$). The improved results were predominantly due to recognition that agonal breathing is a sign of cardiac arrest (75% intervention group vs. 43% control group; $p = 0.01$). The principle finding of this study is that "checking for signs of circulation" as currently taught has a high false negative rate (31%) when used as a diagnostic test in this human simulation model of cardiac arrest. This implies that an erroneous decision to withhold CPR may occur once in every three times a victim in cardiac arrest is encountered. Diagnostic accuracy can be improved and the risk of false negative errors reduced by emphasizing to CPR providers that agonal breathing is a sign of cardiac arrest.

In summary, gasping occurs early in cardiac arrest, especially when the initial rhythm is VF, and is associated with augmented coronary and cerebral perfusion, gas exchange and successful resuscitation. But rescuers are confused by the presence of gasping, thinking it to be a sign of life, leading to CPR being withheld. Dispatchers should be taught to recognize when gasping may be present so they can coach lay rescuers in CPR. BLS courses should include specific content on assessment of agonal breathing and actions to take. The bottom line according to the AHA: Rescuers should be taught to treat gasping as *no* breathing.

Conclusion to Part I

We have seen that ventilations during cardiac arrests are not performed according to the *AHA Guidelines*. The increased rates of ventilation increase intrathoracic pressure, decrease cardiac output and coronary perfusion pressure, and are related to poor survival. Methods are available to coach providers to deliver the correct rate of ventilations. Gasping is common early in cardiac arrest, contributes to increased coronary perfusion pressure, and is associated with an improved survival. In next month's issue of *Code Communications* we will explore the benefits of ventilation during resuscitation and the harm produced by the resultant interruptions in compressions. This should help us answer the question of whether compressions can be performed without ventilations.

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