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Forecast *for* RESUSCITATION

EMS State of the Science Conference leads to new thinking on prehospital resuscitation practices

An editorial supplement to *JEMS*, sponsored by Laerdal Medical Corp. & Philips Medical Systems

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Forecast for Resuscitation
An editorial supplement to *JEMS*
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From the EDITOR

The U.S. Metropolitan Municipalities EMS Medical Directors, whose members are jurisdictional EMS medical authorities from the nation's largest cities, meet each February in Dallas at the annual EMS State of the Science conference to review and discuss research on EMS concepts, procedures and equipment.

The group, representing EMS systems that serve 50 million Americans, also develops member consensus statements on what emerging science indicates are effective (and ineffective) medical procedures. The EMS medical directors frequently return to their respective organizations armed with the research, scientific evidence and consensus necessary to implement changes in their systems' medical procedures.

This annual meeting is often referred to as the "Gathering of Eagles" because a media representative, commenting on the list of prominent EMS physicians participating in the group, once said, "Wow, that's some gathering of eagles." The group of medical directors then became known as the Eagles Coalition.

The 2004 conference again brought together a stellar faculty of EMS industry leaders to present cutting-edge information on EMS research, management issues and patient care innovations, usually their own. This year's program offered attendees the added opportunity to hear first-hand the results of the coalition's two-day retreat, dubbed Eagle Creek, during which many of the metropolitan medical directors, other respected cardiologists and American Heart Association representatives closely reviewed the latest results of research on patient resuscitation.

After two intense days, the physicians walked away with several consensus opinions about where EMS clinical practices are lagging behind the available science and where improvements in patient resuscitation can occur in their own systems—in some cases, immediately.

The Eagles Coalition, spearheaded by EMS pioneer Paul E. Pepe, MD, MPH, agreed to allow *JEMS* to publish this special supplement, which is sponsored by Laerdal Medical Corp. and Philips Medical Systems. The supplement reviews the status of advanced cardiac life support (ACLS) resuscitation in the United States and highlights important topics from the 2004 EMS State of the Science Conference.

This supplement affords EMS administrators, medical directors and field providers the opportunity to review some of the latest clinical research findings and procedural changes that these medical directors considered priorities to be addressed as soon as possible to improve field care and patient resuscitation practices in their jurisdictions. However, readers must be cautioned that many of the recommended changes may not be implemented quickly in their own EMS systems because of the appropriate administrative, political or medical reviews necessary to support new policies. Therefore, it is the intent of this supplement to serve as an important reference source to assist you in understanding the rationale for changes on the horizon and prepare you to implement operational and procedural changes in the future.

The first part of this report takes a close look at some of the Eagles' consensus recommendations and zeroes in on important facts that explain why certain changes should occur in current clinical practice. EMS educator David LaCombe, director of education at the National EMS Academy (operated by Acadian Ambulance Service) and an attendee at the 2004 EMS State of the Science conference, discusses important clinical information presented by Joseph P. Ornato, MD, medical director of the Richmond EMS Authority, and Paul E. Pepe, MD, MPH, medical director for the Dallas EMS system, to outline why targeted clinical changes will improve patient care and resuscitation results in the future.

COURTESY PAUL PEPE, MD

The Eagles Coalition at its 2004 Dallas conference.

The second part of this supplement presents an in-depth review of some current ACLS procedures and much of the research validating the Eagles' recommendations for procedural changes. Clifton Callaway, MD, PhD, an assistant professor at the University of Pittsburgh and an emergency physician at UPMC-Presbyterian Hospital, and David Hostler, PhD, NREMT-P, director of research at the University of Pittsburgh-affiliated Emergency Medicine Residency program, cite 43 references to illustrate key points in their article. (Visit myWebCE.com to earn CE units for this article.)

The combination of expert research, committed EMS medical leadership and well-referenced content makes this a must-read document explaining why changes must, and will, occur in your EMS system in the near future.

—A.J. Heightman

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Forecast for RESUSCITATION

Highlights from the 2004 EMS State of the Science Conference

Dallas, February 20–21, 2004

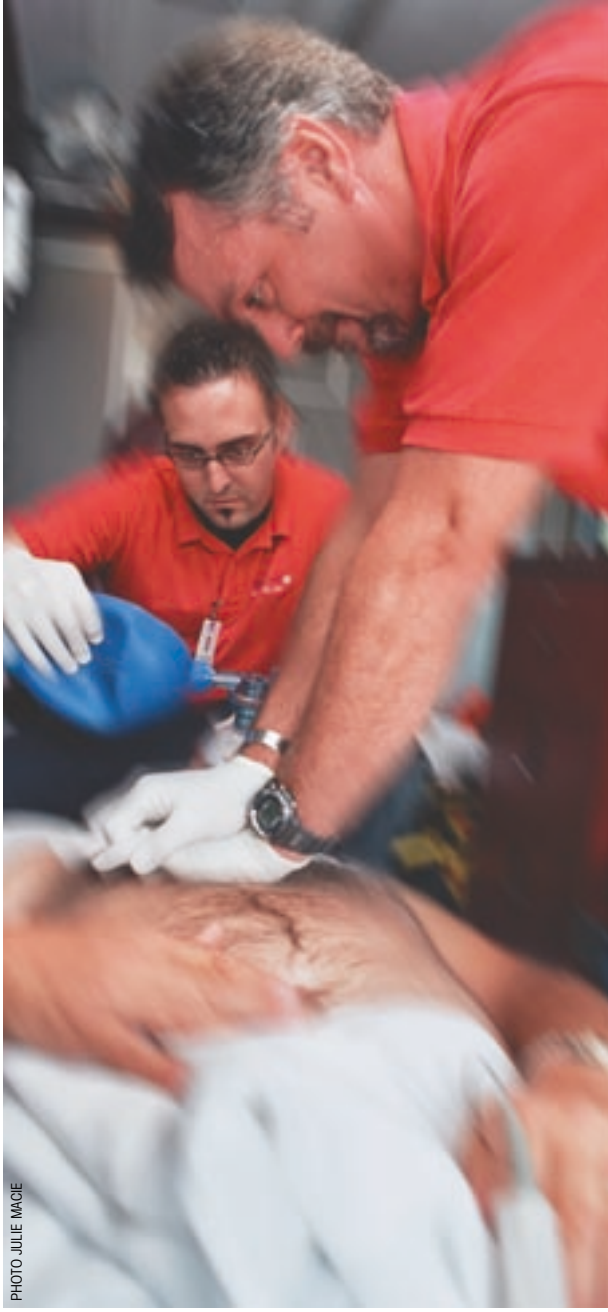

PHOTO JULIE MAGIE

Most of the EMS medical directors from the nation's largest EMS systems attended the EMS State of the Science: A Gathering of Eagles VI conference. The group presented clinical topics on key areas in EMS and results from its two-day clinical retreat (dubbed Eagle Creek) to physicians, nurses, paramedics, educators and media representatives from throughout the United States. Discussions focused on the group's latest innovations and research-based changes that the medical directors plan to make in their systems to immediately improve clinical care even while anticipating the next set of resuscitation guidelines, scheduled for release in 2005 by the American Heart Association.

The U.S. Metropolitan Municipalities EMS Medical Directors (Eagles) coalition proactively involves staff and board members from the AHA, National Association of EMS Physicians (NAEMSP) and American College of Emergency Physicians (ACEP) in its conferences and research summit. It was clear the coalition will revisit any consensus recommendations whenever such groups as the AHA, NAEMSP and ACEP publish new guidelines or position statements. In that respect, the Eagles worked hard, for example, to avoid making internal recommendations or changes that might conflict with future AHA recommendations and endorsed the AHA's comprehensive process in drafting, reviewing and publishing new guidelines.

After reviewing the current science and fellow colleagues' successes during trial studies, the Eagles decided to implement a few system changes immediately, believing that this proactive approach would save more lives, provide better care and predictably be compliant with future guidelines, but on a timetable that's months, if not years, ahead of the typical rollouts.

Although the medical directors presented more than 20 topics, the presentations on the resuscitation of cardiac arrest victims were the most compelling. In the articles that follow, we present a synopsis of the Eagles Coalition recommendations and the bottom line on why EMS systems need to make these types of changes in how they provide resuscitation.

Eagle Creek Report: Results of the 2-Day Consensus Retreat on Leading Controversies in EMS

By A.J. Heightman, MPA, EMT-P

As part of the EMS State of the Science Conference, Paul E. Pepe, MD, MPH, reported on the two-day Eagles retreat conducted to review current research and establish a working agenda on recommended changes or improvements for the group's EMS systems. Some key agenda points:

1. EMS systems need to revise clinical practice procedures to ensure that cardiac compressions aren't interrupted unless absolutely necessary. The results from automated cardiac compression devices that deliver consistent compressions are promising and could aid in this area.
2. Research indicates that agonal gasping that often occurs soon after the onset of sudden cardiac arrest may actually assist in better oxygenating and enhancing venous return than "assisted breaths" in unresponsive patients. Also, uninterrupted cardiac compressions may prolong that process, avoiding the need for early ventilation support. In fact, interrupting compressions to breathe for such gasping patients may diminish blood flow to the brain and respiratory apparatus, thus limiting the duration and effectiveness of the gasps.
3. Regarding citizen CPR involvement: a) If dispatch instructions need to be given to an untrained bystander, dispatchers should give compression-only instructions. (*Rationale:* It takes a tremendous amount of time to send someone away from the phone to open an airway and give the ventilation, and then have them return to the phone to receive additional instructions on how to compress the chest, particularly when the patient has just collapsed and may be gasping and have an oxygen reserve in the non-circulating bloodstream.) b) If a person is on scene and already performing bystander CPR, let them continue as trained; don't confuse things by having them switch to compression-only CPR.
4. Research indicates that it may be beneficial for dispatchers to instruct the public to begin the resuscitation of unwitnessed cardiac arrest victims with four cycles of 100 compressions only, then followed by only a couple of breaths. The CPR sequence would then continue at a ratio of 100:2 or 50:2. (*Note:* In a recent animal study comparing 15:2 vs. compression-only vs. 100:2, the 100:2 compression to ventilation method was the most successful.) This latter recommendation (adding some breaths after four minutes) is optional, but may be most applicable in EMS systems with prolonged response intervals.
5. Research indicates that it may be more beneficial for the first responding crews to perform a minute or two of CPR (chest compressions) *before* delivering their first defibrillation "to prime the patient's pump" and there should be virtually no time delay between cessation of compressions and countershock delivery. (*Rationale:* When the heart goes into ventricular fibrillation, its cells have only about three to four minutes of "fuel," known as adenosine triphosphate (ATP). Shocking the patient after this time period may get rid of the VFib, but typically results in asystole or pulseless electrical activity. "Priming" the heart by performing effective compressions and delivering oxygen to the cells allows the cells to build up or maintain ATP levels before the initial shock is delivered. Compressions should be continued while the defibrillator is being readied for shock delivery because several seconds of delay to shock may reduce the chances of successful resuscitation. In most EMS systems, the real time from collapse to actual first responder arrival at the patient's side exceeds the four-minute window, even with *reported* response intervals of four to five minutes [typically time from the first call to dispatch to arrival at the street address].)
6. The use of vasopressin *in concert with* epinephrine appears to be promising in the resuscitation of cardiac arrest victims, but this requires more definitive study.
7. Current research indicates that there are detrimental effects to overventilating cardiac arrest or major trauma patients and what traditionally have been referred to as "normal ventilation rates" may even be excessive, particularly in connection with hypovolemia or obstructive lung disease (e.g., asthma, COPD).
8. For non-cardiac arrest patients, the initial administration of 12 mg adenosine in patients with SVT may be more beneficial than starting with 6 mg. There is no significant downside, a potential benefit and less cost involved in this approach.

To put the cardiac arrest recommendations in perspective, let's take a look at a case that could be handled in your EMS system today.

Case study

A 56-year-old man suddenly collapses at a busy restaurant. As waiters attempt to determine if the victim is breathing, a bystander urgently calls 9-1-1. Each passing second feels like an eternity for restaurant patrons. Receiving pre-arrival instructions from dispatch, the caller yells to the waiters to open the airway and determine if the patient is at all responsive or breathing. "He looks like he's gasping for air," says one waiter. "I can't feel a pulse," says another employee.

The trained dispatcher on the phone tells the caller, "I need you to start CPR. Is there an AED in the area?"

"I don't know," replies the excited bystander. "No one here knows CPR."

The dispatcher spends a minute explaining how to open the patient's airway and administer two rescue breaths. However, the patient had vomited prior to collapsing, so the bystander hesitates before administering the breaths. The caller returns to the phone for additional

instructions. He then directs an employee to begin chest compressions and tells him how to do it.

While compressions begin, sirens become increasingly louder, and an engine company arrives at the patient's side four minutes after the initial call is placed. Firefighters instruct the bystanders to stop chest compressions so they can begin an assessment. The initial survey reveals that the patient is unconscious, apneic and pulseless. Firefighters begin to connect the bag-valve mask to oxygen and place AED electrodes on the patient's chest. After analyzing, a shock is advised. The crew delivers the shock, the body twitches and they wait for the next instruction.

The recorded voice of the AED plays the familiar statement: "Analyzing ... No shock advised. Begin CPR." The BLS first responders *then* initiate two-person CPR with a 5:1 compression/ventilation ratio a minute or more after their arrival.

Paramedics arrive moments later and initiate ALS, including ECG monitoring, intubation, IV access, medication therapy and pacing. Despite their efforts, the patient remains in asystole and is pronounced dead 30 minutes later in the emergency department.

Could anything have been done differently to improve the outcome for this patient? The answer is yes—many things. Let's take a look at the scientific evidence that proves that some changes are needed in our approach to resuscitation.

Physiology of cardiac arrest

Each day in the United States, more than 950 people experience sudden cardiac arrest. Resuscitation of those patients requires an understanding of the body's metabolism of energy, how ventilation procedures can affect coronary perfusion and how compromised blood flow affects ventilatory needs.

The critical source of energy muscles need to contract is obtained from adenosine triphosphate (ATP). These molecules are literally fuel for muscles (including the heart). Several research studies reveal that, during certain cardiac arrests, the heart consumes ATP at a rapid rate. Ordinarily, ATP levels would be continuously replenished. However, during cardiac arrest, the body's ATP reserves cannot be maintained.

The most common etiology of cardiac arrest is ventricular fibrillation. As the heart continues to fibrillate, the myocardium (heart muscle) consumes its ATP rapidly. Within about four minutes, ATP stores are significantly depleted, and the "coarse" ventricular fibrillation deteriorates to fine fibrillation and eventually asystole (standstill). So, in essence, the fibrillating heart is eating up fuel.

Though less effective after several minutes of arrest, the provision of chest compressions to pulseless patients circulates blood and oxygen, which may allow heart cells to replenish their ATP stores. When sufficient myocardial ATP levels are restored, there's increased potential for successful defibrillation to be followed by a pulse and blood pressure. Clearly, more immediate initiation of CPR is better and helps to maintain ATP levels even longer. Nevertheless, even immediate bystander CPR by family members has some potential downsides.

Unfortunately, time-motion studies and other careful observations reveal that both bystander and professional rescuers interrupt chest compressions too frequently and for time periods that are much too long. Several researchers have concluded that frequent pauses between rounds of chest compressions actually contribute to failed resuscitation efforts.

Recent studies have also indicated that the highest probability of survival exists

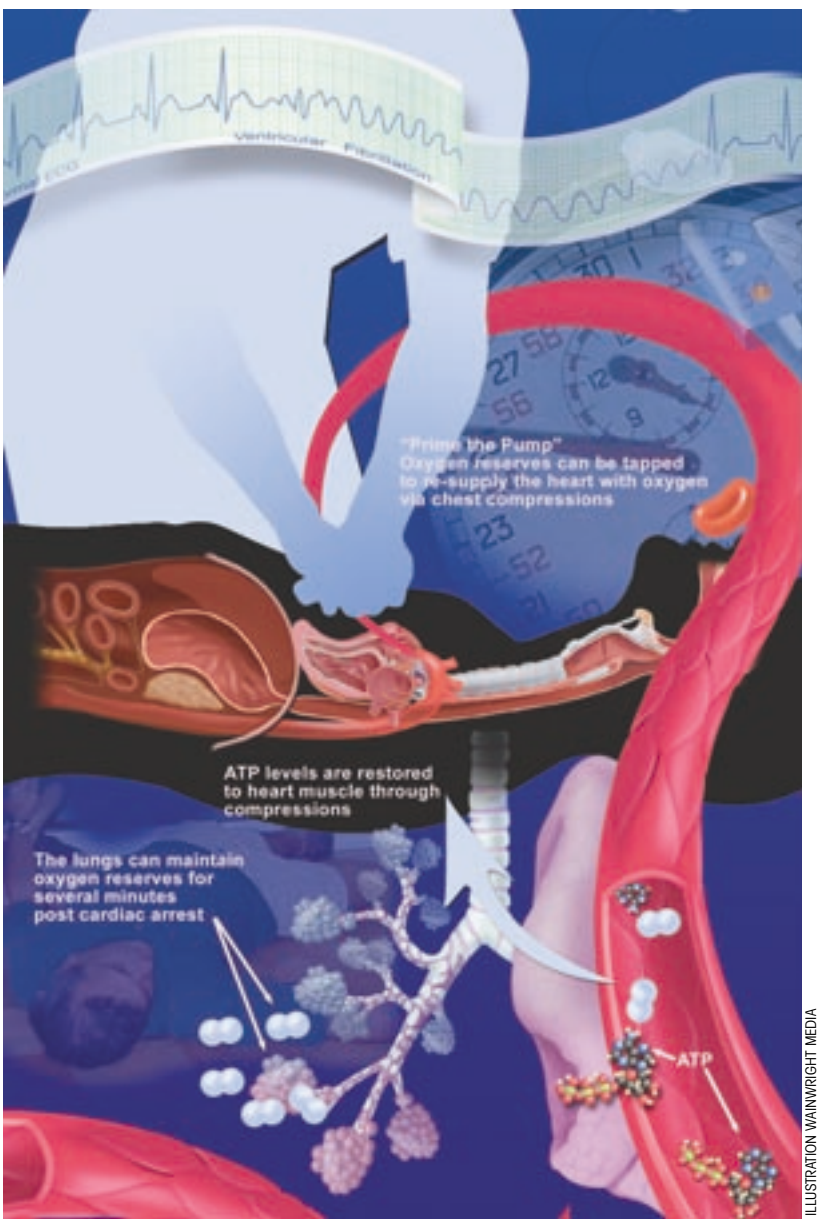

ILLUSTRATION WAINWRIGHT MEDIA

Muscles obtain energy from ATP. The provision of chest compressions to pulseless patients circulates blood and oxygen, which may restore ATP to heart cells and improve the chances for successful defibrillation.

among those patients who receive chest compressions to restore ATP levels to shockable levels *prior to any attempt to defibrillate*. Conversely, survival rates fall dramatically within a matter of seconds if there is too long a pause from the time compressions are stopped until the moment the shock is actually delivered.

The provision of CPR can be lifesaving, if applied correctly. However, in one study, researchers observed that EMS personnel perform CPR differently in the field than in the classroom by administering compressions of inconsistent depth. It's also clear to medical experts that many rescuers are very prone to excessively ventilating patients, causing increased intrathoracic pressures that markedly decrease coronary perfusion, hampering resuscitation.

Clearly, there is much work to be done to readjust our thought processes and adapt our operating procedures to conduct more effective resuscitation efforts. However, modern EMS now has the benefit of prehospital research that can serve as the beacon to guide the way to improved patient care.

Close Up: Eagles Explain the Rationale for Change

By David LaCombe, EMT-P

An audience of more than 400 physicians, nurses, EMTs, paramedics, media representatives and government leaders listened with enthusiasm as Joseph P. Ornato, MD, and Paul E. Pepe, MD, MPH, presented evidence at the 2004 EMS State of the Science Conference that some of our time-honored practices in resuscitation are in need of change. I interviewed Ornato and Pepe, two of the nation's leading medical researchers and members of the Eagles Coalition. They agreed to share some of the most important information presented about sudden cardiac arrest for this special *JEMS* supplement.

LaCombe: What was your initial reaction to the studies revealing that in many cases, providing CPR prior to defibrillation would improve outcomes in patients with out-of-hospital ventricular fibrillation?

Ornato: This is big stuff. It's starting to explain why a lot of what we do is not giving us the results we've hoped for. It's been a mystery, but it's now becoming fairly simple to understand the concept that you have fuel, and you have to replenish the fuel, and that's what it's all about. Simply put, the more you do CPR [compressions], the more you sweeten the myocardial blood flow; the more myocardial blood flow you get, the more ATP is produced and ventricular fibrillation becomes more coarse—increasing the likelihood that defibrillation will be successful if delivered within five seconds of an interruption of chest compressions.

LaCombe: During your presentation you showed video of an echocardiograph taken during the resuscitation of a patient receiving CPR with a ratio of 15 compressions to two ventilations. Could you describe for our readers the significance of what that echo revealed?

Ornato: As the sternum is compressed during CPR, blood clears the right side of the heart immediately, but it has to get through the lungs before exiting the left side of the heart. While a round of 15 compressions is delivered, the coronary perfusion pressure increases—driving blood through the coronary arteries to perfuse the heart.

The coronary perfusion drops significantly when compressions are paused for two ventilations. Then the compressions start the pump again. It takes eight to 10 beats before the blood starts to flow through the coronary arteries at a decent level. Of course, just as blood flow becomes decent, you pause again for additional ventilations, and the perfusion drops all the way back down again. It's like trying to climb to the top of the ladder: Just as you start to make real progress, you go all the way to the bottom and start all over again. That's what we're doing when we pause for more than a second or two during CPR.

Every time we stop pushing on the chest, everything comes to a halt. And when we start pushing, it takes a finite time period, eight to 10 compressions on average, before reasonable flow and pressure resume again

LaCombe: Isn't this what led to a concept referred to as the *hands-off interval*? Could you explain what this means?

Ornato: The term *hands-off interval* refers to the number of seconds it takes from the last CPR chest compression to when you fire the defibrillator. This came to light from a study in which ventricular fibrillation was electrically induced in 20 swine that were untreated for seven minutes before CPR was started [Yu, et al: *Circulation*. 106:368–372, 2002]. The researchers experimented with various hands-off intervals.

Interestingly, all the pigs that received defibrillation within three seconds of interruption of chest compression survived with good pulses and blood pressure. However, resuscitation dropped to 80% with a 10-second interruption interval, and 40% percent with a 15-second interruption interval. None of the swine survived with 20 seconds or more of interrupted chest compression.

LaCombe: So, if interrupted chest compressions are one of the culprits for failed resuscitation, what other areas have been identified for which we need to change our clinical approach to resuscitation?

Pepe: Everyone always talks about the ABCs in resuscitation. But, in my opinion, we don't talk enough about the B part of the ABCs. Obviously, breathing is lifesaving, but it can also be detrimental if we overventilate our patients—something that's easy to do in a low flow state like CPR. It comes down to understanding the unique physiology of CPR and why we often don't behave as if we understand the effects of positive pressure ventilation on resuscitation.

Ornato: I agree with Paul. It's really important to understand the physiology of the body and its preference to maintain circulation through gasping in the first minutes of cardiac arrest. For example, the case study that opens this article involves a victim who suddenly arrests in a diner. Bystanders note his gasping effort, which is a typical response of the body to maintain oxygenation and circulate some blood.

Pepe: Think about a gasp. Right now, as you breathe normally, your tidal volume is probably only around 400 or 500 mL. A gasp, however, may suck in much more air than a normal breath. The initial presence of gasping probably indicates that the body's oxygenation is still sufficient to maintain the brain's control over

the respiratory effort and the respiratory muscle contractions. Furthermore, the gasping effort enhances negative pressure in the thorax, significantly increasing venous return to the heart.

LaCombe: So the presence of gasping in the cardiac arrest patient probably means that rescuers have arrived only minutes after the onset of the arrest. What implication does this have for EMS?

Ornato: In the first five to six minutes of adult, non-traumatic cardiac arrest, providing artificial ventilation isn't as critical as we once thought, particularly for the lay public. When rescuers exhale into the lungs of a patient, they are blowing in a high volume of CO_2 and only about 17% oxygen. Every time we release pressure on the sternum following a chest compression, the lungs passively suck in some room air, which contains 21% oxygen at sea level. An adult can usually maintain oxygen saturation above 80–85% for the first five minutes after onset of a cardiac arrest. Thus, it now seems to be more important to provide uninterrupted chest compressions rather than to pause periodically for ventilations.

After the first five minutes, ventilations are needed because the amount of passive ventilation generated by each compression decreases as the "bellows effect" of the chest deteriorates when the diaphragm starts to run out of ATP.

Pepe: Most importantly, patients lose their ability to gasp after several minutes of low flow (CPR) conditions. The same is true for the heart's energy. In airport AED response and coronary care unit research, it's been shown that if we deliver that countershock within the first three to four minutes, those patients will generally come back without much of a problem. The problem generally occurs after four to five minutes of a ventricular fibrillation event. The heart begins to run out of ATP, and it becomes less susceptible to conversion to an organized rhythm with pulses after a countershock.

If you do the data dredging and break it down to those cases in which the response time interval (call to scene arrival) was an average of four minutes or less, there isn't much of a difference whether a shock is given immediately or deferred until CPR is performed for two to three minutes. Where the differences show up is when there's an excess of four minutes for EMS response. Two or three minutes of CPR first in these instances makes defibrillation more successful. The data are very compelling.

A randomized, controlled trial done in Oslo, Norway, showed a better survival to hospital discharge if they gave three minutes of standard CPR before they went to shocks.

LaCombe: The Eagles group said compressions should be administered for 90 seconds to three minutes for patients down for a long time. Some might say that three minutes is a lot.

Pepe: First of all, our recommendation was not to do CPR for three minutes, *then* hurry up and put the defibrillator on. Crews can still be starting an IV, setting up for intubation and putting the defibrillator pads on during that time period while compressions are being performed so that they're ready to shock when the time comes.

Regarding the duration of pre-shock CPR, as a group, we left an option open for our systems: If a medical director wants their system to do 90 seconds, or if another medical director wants to have it be three minutes, that's fine. So we're recommending, as a group, that our systems do five to 10 cycles of chest compression/ventilation sequences when a first responder gets on the scene [considering the heart has been deprived of oxygen for an extended period of time—beyond four minutes].

What's the exception? When you get there and the guy arrests right in front of you. No question—shock him. But if the person has been down for an extended time period, prime the pump and do five to 10 cycles of CPR [before shocking the patient] *and* with little time delay between cessation of compressions and the shock.

LaCombe: What's the latest on the use of vasopressin in resuscitation?

Pepe: In our consensus group, we talked about vasopressin. We agreed that there aren't enough data out there to say "let's switch over to vasopressin vs. epinephrine right now." We may eventually end up combining vasopressin and epinephrine somehow in our treatment regimen, but we don't have the definitive data yet. Still, we believe there's compelling enough information to look at in future studies.

Ornato: This combo effect [of vasopressin and epinephrine together] really is intriguing. Our colleagues in Europe have some pretty compelling data indicating that the epinephrine receptors become less responsive over time during resuscitation. We've known that for more than a decade now. The actual mechanism is not clear. That doesn't seem to occur to the same degree with the vasopressin receptors.

In animal models, you can show that vasopressin will continue to have a vasoconstrictor effect much longer than epinephrine during ongoing resuscitation. By giving vasopressin, then giving epinephrine on top of it, one plausible theory to explain why administering both drugs back-to-back seems to be more effective than either alone is that the vasopressin is causing vasoconstriction and a rise in the coronary perfusion pressure that actually delivers more epinephrine to its receptors. We [also] need to consider the net clinical benefit, probably one of the most difficult things that we all grapple with in medicine. When you have a therapy, whatever it is—drug, device or combination—there is an outcome that's positive. But at the same time, in another group of patients, it has a bad outcome. You're in a medical and an ethical dilemma by trying to figure out if you're doing more good for more people, even though you're causing a little more harm.

LaCombe: What about the concerns you present about the overventilation of patients?

Pepe: We have found that field crews are definitely, and inadvertently, overventilating their patients nationwide. Ventilation should not be "normalized" during CPR conditions—it's different.

Why do we ventilate? Yes, to get oxygen to the blood. But there's a difference between oxygenation and ventilation. They're intertwined in terms of respiration, but there's a key distinction. In oxygenation—pulmonary oxygenation, not tissue oxygenation—we are attempting to saturate red cells. Ventilation describes how much CO_2 we are clearing (e.g., hyperventilation or hypoventilation).

Colloquially, *hyperventilation* often means "breathe fast." However, by true

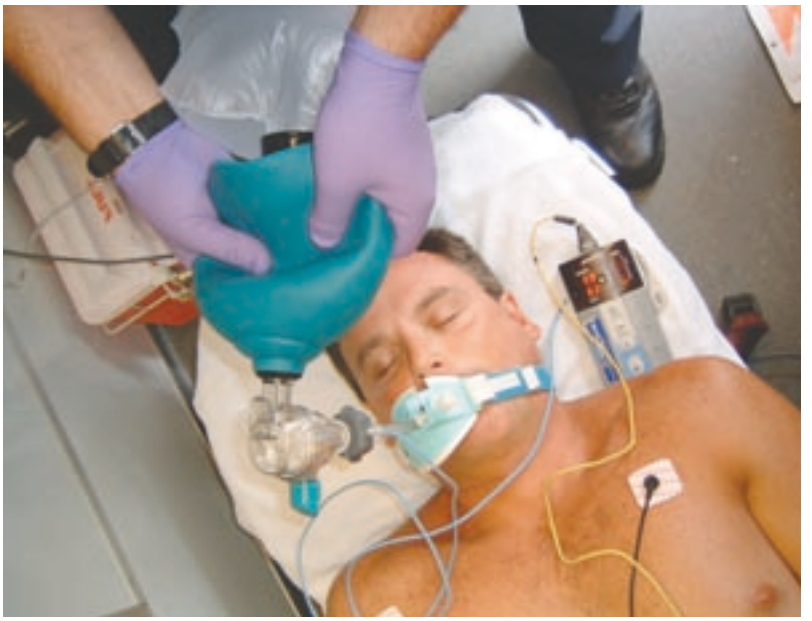

PHOTO BILLY GUTH

Field crews are definitely, and inadvertently, overventilating their patients nationwide. Ventilation should not be "normalized" during CPR conditions—it's different.

definition, it means, "low CO_2 level," and *hypoventilation* means "high CO_2 levels" in the arterial bloodstream.

When I use the term ventilation, I'm not thinking about oxygenation, I'm thinking about how much CO_2 I need to remove. One should separate these two things [oxygenation and ventilation] in terms of patient care.

When you breathe, there's a mixture of openings and closures in the lungs. In open (normal) air spaces, blood is virtually 100% saturated with oxygen. PO_2 at sea level is about 760 mmHg, take away 50 for water vapor pressure and 21% of that dry gas gets you about 150 mmHg for oxygen. So when the air enters your trachea, it's at 150 mmHg and then, displaced by CO_2 coming out, it falls to 110 mmHg in the alveoli. But when the PO_2 gets to about 110 mmHg, the saturation of red cells is still about 98–99%.

So for all intents and purposes, you don't need to put 100% oxygen into those inflated lung units; it's not going to change much. Breathing faster won't do much either. Likewise, faster rates and 100% O_2 won't affect fully closed units. Lung inflation is the key. Particularly in patients who aren't breathing, you have to reinflate their closed lung zones. So you first need to inflate enough to get most lung units closer to 100% saturation. Rate is not the important variable here.

But what's an adequate tidal volume? Normal breaths are about 5–7 mL/kg or about 400–500 mL in average person. But what we find is that air maldistributes when we provide positive pressure breaths. Unlike normal breaths that pull the diaphragm down and pull the lung zones open in a specific architecture, positive pressure breaths "push" the lungs open and maldistribute to pathways of least resistance (less "dependent" lung zones).

The irony is that oxygenation does require the "normal" number of breaths per minute if the tidal volume is adequate, and in CPR conditions, the need to remove CO_2 is extremely low and much less than normal.

First of all, when you use larger than normal tidal volumes, more CO_2 is cleared with each breath. More importantly, in CPR conditions, flow is so low that total body oxygen metabolism falls off, and even when it continues in some tissues, return of the CO_2 produced by that metabolism to the lungs is impaired by the low flow state. In turn, there's little need to ventilate frequently, at least until pulses return. In fact, positive pressure breaths inhibit venous return to the heart and, the more often they are provided, the more that cardiac output (whatever is left) falls. This effect is clinically negligible in most normovolemic persons with normal circulation. But with obstructive lung disease (e.g., asthma, COPD), hypovolemia (e.g., little preload or venous return) and severe circulatory compromise (e.g., CPR situations), the detrimental effects of positive pressure breaths are exaggerated and can even be lethal by inhibiting coronary flow altogether. This may often be an underappreciated cause of PEA and EMD.

This effect applies even to situations with spontaneous circulation. During an animal study involving moderate hemorrhage, and maintaining 10–12 mL/kg tidal volume, we switched ventilations from 12 to six, back to 20, 30, back to six. We observed variables right before the switch, but were continuously monitoring coronary perfusion pressure. At a respiratory rate of 12, the cardiac output was low (2.5 L/min.). But switching rates from 12 to six, cardiac output popped up to 2.8 L/min. More importantly, coronary perfusion pressure rose significantly while oxygenation was maintained.

When ventilation rates were raised back to 20 or 30, it pushed the coronary pressures way down. Then when the rate returned to six, there was a nice rebound effect with markedly improved coronary perfusion and maintenance of oxygenation. So there was much better oxygenation of the tissues by using slower rates. This study also pointed out that the respiratory rate, not just the hemorrhage, was a confounding variable that caused much of the low BP.

But is this issue well recognized? We went through 16 different paramedic and resuscitation training textbooks and found they are providing fairly nonspecific guidelines about ventilatory rates and the negative effects of even "normal" rates in severe shock conditions, let alone commentary about the hazards of overventilation. We found some that recommended 15–20 breaths per minute, some saying 24–28 and others 24–30. In fact, recent investigations found that, even if personnel were instructed to give just 12 breaths per minute, they were out in the field still providing more than 20 per minute.

Tom Aufderheide and his colleagues in Milwaukee directly monitored how often crews were giving breaths during CPR. They found the average was 37 per minute. Despite direct demonstration of 12 breaths per minute in follow-up classes, paramedics subsequently still averaged 22 per minute in the field. Whether it's the EMS "adrenaline effect" or whatever,

we may need to control how we ventilate beyond just training people to provide slower breaths.

LaCombe: How does the overventilation of patients negatively affect care and outcome studies?

Pepe: In looking back at the data we obtained from a multitude of studies, ventilation was not controlled and, thus, this may have been an unrecognized confounding variable that resulted in bad outcomes or blunted the positive effect of the drugs studied (e.g., high-dose epi, amiodarone, etc.).

It may very well be that such occult variables as overzealous positive pressure ventilation also led to negative studies on rapid sequence induction intubation (RSI) outcomes and traumatic circulatory arrest for which many protocols still call for rapid [ventilation] rates to "compensate for metabolic acidosis" or to "pump more oxygen in."

The low pCO_2 that showed up in the RSI outcome studies may be a surrogate variable for overzealous ventilations, too many positive pressure breaths cutting off adequate cardiac output.

Many unproven interventions for resuscitation may, in fact, work. They worked well in the lab. It just may be that we need to look more closely at overventilation in the future as a key variable that affects studies and, most importantly, survival rates.

Concluding remarks

Pepe: Corey Slovis, MD, from Vanderbilt University and the Nashville, Tenn., EMS system perhaps summed up this process of change best when he said, "As all of these data get presented, we need to be certain in our uncertainty. That is, some services may choose to give epi every three minutes, some every five [minutes]. Some [may] switch to vasopressin, some [may] alternate epi and vasopressin, and others may go to vasopressin, vasopressin, then epinephrine. We need to tell our crews, 'This is what we think is the best right now; there's going to be new information.' And we need to get away from what we've had in the past, which is a lot of dogma: 'This is how you do CPR period. You need to know how to do it *this* way.' We need to stress to our personnel that '[prehospital medicine] is continuing to evolve—even more rapidly than in the past. It's just going to continue to evolve.'"

Ornato: I'm convinced that the solution, if there is one to having the next leap in better outcome for in- or out-of-hospital resuscitation, is likely not going to be *one* drug or *one* device. If you look at what's happening physiologically to the human body, multiple bad things are happening at once.

It's awfully naïve of us as a scientific and medical community to think that one single drug is going to fix all of those physiologic derangements until the heart gets started again. Once you're beyond the electrical phase and into what's called the metabolic phase, it looks like you need multiple things.

What's so exciting is that we're now having circulatory squeeze devices that seem to at least provide better perfusion and pressure. We've got some hints that we can tweak the pharmacology to perhaps distribute the blood flow more intelligently. We've got, I think, the ability to more intelligently apply our ventilation techniques. We've got better wave forms. We've got all these things. I think the real fascinating thing to watch is what happens when you combine them all together, vs. the standard way that we've been doing it with one little tweak here and there. The combo is where we're going to wind up in the future.

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Studies Every EMS System Should Review Before Revising Resuscitation Protocols

Delaying defibrillation to give CPR

Conclusion: Patients with VFib and ambulance response intervals *longer than five minutes* had better outcomes with three minutes of CPR first before defibrillation was attempted. Performing CPR first prior to defibrillation offered no advantage (or disadvantage) in improving outcomes for patients with ambulance response times *shorter than five minutes*. Therefore, patients should get three minutes of CPR before shocks.

Reference: Wik L, Hansen TB, Fylling F, et al: "Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation." The Journal of the *American Medical Association (JAMA)*. 289(11):1389–1395, 2003.

Importance of continuous chest compressions during CPR

Conclusion: Continuous chest compression CPR produces greater neurologically normal 24-hour survival than standard ABC CPR when performed in a clinically realistic fashion.

Reference: Kern KN, Hilwig RW, Berg RA, et al: "Importance of continuous chest compression during cardiopulmonary resuscitation: improved outcome during a simulated single lay-rescuer scenario." *Circulation*. 105(5):645–649, 2002.

Lethal delays in chest compression

Conclusion: Longer delays in chest compressions with an automated external defibrillator than occur with a manual defibrillation can worsen the outcome from prolonged VFib.

Reference: Berg RA, Hilwig RW, Kern KB, et al: "Automated external defibrillation versus manual defibrillation for prolonged ventricular fibrillation: lethal delays of chest compressions before and after countershocks." *Annals of Emergency Medicine*. 42(4):458–467, 2003.

Chest compressions alone

Conclusion: The outcome after CPR with chest compressions alone is similar to that after chest compression with mouth-to-mouth ventilation. Chest compressions alone may be the preferred approach for bystanders inexperienced in CPR or receiving instructions over the phone.

Reference: 1) Ferreira D: "Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation." *Portuguese Journal of Cardiology*. 19(7–8):839–840, 2000. 2) Hallstrom A, Cobb L, Johnson E, Copass M: "Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation." *The New England Journal of Medicine*. 342(21):1546–1553, 2000.

Negative effects of overventilation

Conclusion: There is increasing evidence that current practices of rescue breathing may be detrimental in the resuscitative management of severe trauma, respiratory failure and cardiac arrest. This effect is most pronounced in patients with hypovolemia and obstructive lung disease largely because high frequencies of positive pressure ventilation can inhibit venous return and, in turn, cardiac output.

Reference: Roppolo LP, Wigginton JG, Pepe PE: "Emergency ventilatory management as a detrimental factor in resuscitation practices and clinical research efforts." *Intensive Care and Emergency Medicine*. Vincent JL (ed). Springer-Verlag: Berlin, pp. 139–151, 2004.

Hyperventilation-induced hypotension during resuscitation

Conclusion: Rescuers were observed to excessively ventilate patients during prehospital CPR. Subsequent animal studies demonstrated that similar excessive ventilation rates resulted in significantly increased intrathoracic pressure and markedly decreased coronary perfusion pressures and survival rates in both cardiac arrest and hemorrhagic models.

References: 1) Aufderheide TP, Sigurdsson G, Pirallo RG, et al: *Circulation*. 109(16):1960–1965, 2004.

2) Pepe PE, Raedler C, Lurie K, Wigginton JG: "Emergency ventilatory management in hemorrhagic states: elemental or detrimental?" *Journal of Trauma*. 54:1048–1057, 2003.

Interrupted compressions

Conclusion: Interruptions of compressions for rhythm analysis that exceed 15 seconds before each shock compromise the outcome of CPR and increase the severity of postresuscitation myocardial dysfunction.

Reference: Yu T, Weil MH, Tang W, et al: "Adverse outcomes of interrupted precordial compression during automated defibrillation." *Circulation*. 106:368–372. 2002.

The effects of biphasic waveform

Conclusion: High energy and average current are associated with increased post-shock dysfunction, while low energy and high peak current maximize survival, in an experimental comparison of biphasic waveforms designs. Maximum survival and minimum myocardial dysfunction were observed with the low capacitance 150-J waveform, which delivered higher peak current while minimizing energy and average current. The effects of biphasic truncated exponential waveform design on survival and post-resuscitation myocardial function after prolonged VFib were examined. The study examined a low-capacitance waveform typical of low-energy application (100 uF, ≤ 200 J) and a high-capacitance waveform typical of high-energy application (200 uF, ≥ 200 J) in a long downtime (seven minutes untreated VF) swine model.

Reference: Tang W, Weil MH, Sun S, et al: "The effects of biphasic waveform design on post-resuscitation myocardial function." *Journal of the American College of Cardiology*. 43(7):1228–1235, 2004.

Effects of transthoracic impedance & body weight on defibrillation

Conclusions: Data from EMS systems using low-energy, non-escalating AEDs were retrospectively analyzed with regard to the influences of transthoracic impedance and body weight on defibrillation and resuscitation. High-impedance and overweight patients were defibrillated by the biphasic waveform at high rates, with a fixed energy of 150 joules, and without energy escalation. Rapid defibrillation, rather than differences in patient impedance, accounts for resuscitation success.

References: 1) White RD, Blackwell TH, Russell JK, et al: "Transthoracic impedance does not affect defibrillation, resuscitation, or survival in patients with out-of-hospital cardiac arrest treated with a non-escalating biphasic waveform defibrillator." *Resuscitation*. 2004; in press.

2) White RD, Blackwell TH, Russell JK, et al: "Body weight does not affect defibrillation, resuscitation or survival in patients with out-of-hospital cardiac arrest treated with a non-escalating biphasic waveform defibrillator." *Critical Care Medicine*. 2004; in press.

Forecast *for* RESUSCITATION

PHOTO JONNY LAVERSKY

ACLS Update

Current & future direction for the resuscitation of cardiac arrest patients

By Clifton W. Callaway, MD, PhD, & David Hostler, PhD, NREMTP

Cardiopulmonary arrest occurs both suddenly as a consequence of heart disease and as the endpoint of chronic diseases. Sudden cardiac death is more common in males than females and affects African-Americans more than Caucasians or Asian-Americans.[1,2] Although sudden death can affect patients of all ages, most studies cite the mean age for sudden cardiac arrest between 65 and 70 years.[1,3] In the United States, currently 6–7% of patients survive after out-of-hospital cardiac arrest (OOHCA).[4]

Basic treatment priorities include rapid restoration of spontaneous circulation (ROSC) and minimizing damage to organs, primarily the brain. ROSC consists largely of mechanical and electrical rescue shocks. In contrast, brain injury involves primarily cellular and molecular events that are treated with specific and detailed intensive care.

Meaningful survival is unlikely without attention to both heart and brain. Unfortunately, the cause of cardiopulmonary arrest is frequently unknown, and treatment follows guidelines with a one-size-fits-all approach, preventing providers from directing treatment to optimize resuscitation.

A single EMS provider may work for many months without seeing a cardiac arrest victim survive, but overall about one-third of OOHCA patients have ROSC long enough to be admitted to the hospital (see Figure 1, p. 16). Of those patients admitted to the hospital after OOHCA, two-thirds die prior to discharge.[5] The most common reason for death among patients after ROSC is post-ischemic brain injury. Failure to regain consciousness leads to withdrawal of care and death for as many as 44–68% of OOHCA victims.[3,6]

Restarting the heart

There are two barriers between the OOHCA victim and a positive outcome: restarting the heart and regaining consciousness. Two activities are central to restarting the heart: 1) artificial circulation (chest compressions augmented by vasoactive medications and ventilations) to circulate oxygenated blood to the heart and brain and 2) electrical rescue shocks to terminate ventricular fibrillation (VFib) or unstable tachydysrhythmias. Rescue shocks should be used only when appropriate. It's critical to keep these priorities in mind because patients cannot be revived without excellent and near continuous chest compressions.

The contribution and recommended division of time for resuscitation activities is depicted in Figure 2 (p. 16). All other activities, including antidysrhythmic medications and advanced airway maneuvers, are designed to supplement the chest compressions or rescue shocks and may not be appropriate for all patients.

Optimal resuscitation requires minimal interruption in chest compressions. After initiating resuscitation, patient re-

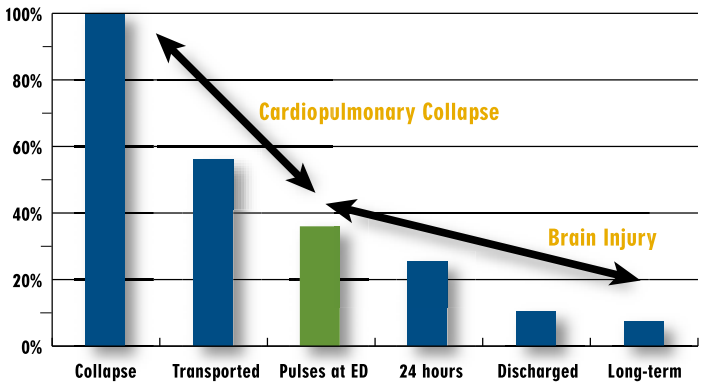

Figure 1: Early death after cardiac arrest results from cardiopulmonary collapse. Death during the first few days after hospital admission is usually related to brain injury and failure to awaken.

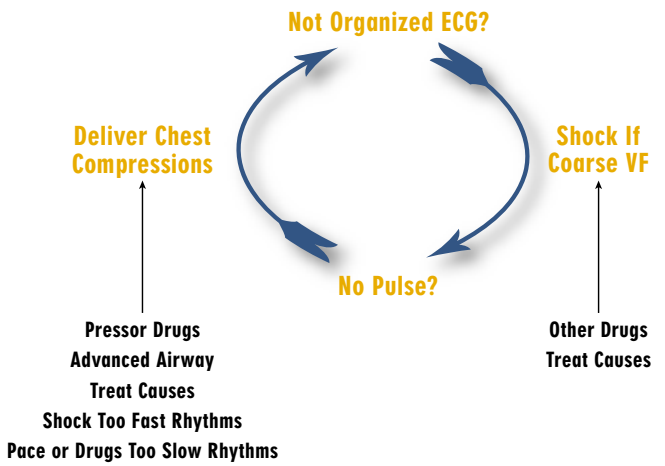

Figure 2: Prioritization of activities during cardiac resuscitation: Chest compressions should be interrupted only to provide rescue shocks when appropriate. All drugs, airway devices and other interventions are designed to augment either artificial circulation or defibrillation. None of these adjuncts should interrupt or detract from performing the two core activities.

assessment can be reduced to constant awareness of the electrocardiogram (ECG) and whether or not pulses are present. For example, there is no reason to stop compressions for a pulse check if asystole or ventricular fibrillation is noted on the ECG during ventilations.

Airway & ventilation

Agonal respirations may delay recognition of a cardiac arrest victim by lay people and EMS providers who report that the patient is having "labored breathing" or is "breathing funny" when they are actually pulseless and need CPR.

Agonal gasping is associated with survival, probably because it stops after one to two minutes and, thus, is a great time marker for recent collapse.[7,8] Agonal respirations should signal to the prehospital provider that the patient is pulseless and needs immediate chest compressions.

Advanced airway management is often not required in the initial phase of resuscitation. Excellent BLS skills are effective for oxygenating patients and do not require long pauses in chest compressions during the critical early moments of resuscitation.

During cardiac arrest, muscle tone fails, and the tongue and soft tissue occlude the upper airway. Consequently, the airway is rarely patent during cardiac arrest.[9] If uncorrected, this obstruction prevents oxygenation and ventilation, preventing resuscitation.

Simple maneuvers can open the human airway. Extension of the neck (head-tilt) and forward displacement of the mandible (chin-lift) straightens and opens the pharynx. Insertion of an oropharyngeal airway can move the tongue forward in the pharynx. With these steps, positive pressure ventilation can be provided using mouth-to-mask or bag-valve-mask ventilation.

A positive pressure breath of 10–15 mL/kg delivered over two seconds will fill the lungs. Ventilation with as little as 400 mL in adults (6–7 mL/kg) will cause the chest to rise and meets the current guidelines for artificial ventilation with supplemental oxygen for unprotected (non-intubated) patients.[10]

For CO_2 to be present in exhaled air, blood must be flowing through the lungs.[11] Therefore, capnometry (end-tidal CO_2 measurement) can be used to assess the quality of ventilations and confirm placement of an endotracheal tube. CO_2 levels measured by a capnometer may be very low (< 10 mmHg) at the onset of resuscitation. Good quality chest compressions will cause CO_2 levels to increase, and these levels may be used as feedback to improve or modify chest compressions.

An abrupt increase in end-tidal CO_2 levels, usually to levels > 35 mmHg, accompanies the ROSC and may be the first indication of successful resuscitation.

Airway devices: The self-inflating bag attached to a face mask (bag-valve mask) is the most common ventilation device used, but the bag-valve mask (BVM) has several pitfalls. For example, maintaining an airtight seal between the mask and the face of the patient is often difficult, particularly when simultaneously performing head-tilt, chin-lift maneuvers. Practice increases ventilation success by a single provider, but two providers achieve more reliable airway management. Two-provider ventilation is optimal because while one provider squeezes the bag, the second provider can use two hands to make a seal and position the head.

A second difficulty with BVM ventilation is that a significant amount of air enters the stomach if the patient is too aggressively ventilated, increasing the likelihood of vomiting.[12,13] In conscious patients, the esophagus prevents air entry into the stomach unless upper airway pressures exceed 15–20 cm H_2O.[14] However, during cardiac arrest, esophageal muscle tone declines, and air

will enter the stomach with upper airway pressures above 5–8 cm H_2O.[15] Rapidly squeezing the bag during the excitement of the resuscitation situation can result in much higher upper airway pressures. A typical adult resuscitation bag holds approximately 1,600 mL of gas. Many providers squeeze the resuscitation bag as completely as possible in the belief they are delivering better ventilation, although only 400–600 mL of oxygen is required for average-sized adults. In addition to promoting inflation of the stomach, overenthusiastic ventilation also causes positive pressure in the thorax of a patient with an unprotected airway, which further impedes venous blood returning to the heart.

A recent study examining both humans and animals suggests that hyperventilation during CPR is very common during resuscitation of cardiac arrest victims.[16]

Endotracheal intubation can secure the airway definitively by protecting it from emesis and maintaining a patent airway. However, laryngoscopy usually requires an interruption in chest compressions, and simply inserting an endotracheal tube will not restart the heart. Therefore, endotracheal intubation must be considered an adjunct to the initial resuscitation, and it should not delay or interrupt chest compressions. Obviously, any patient with coma after ROSC will require endotracheal intubation, but the interruption in artificial circulation required for this procedure should be carefully considered. With a protected airway, tidal volumes of 10 mL/kg at slower rates are preferred.

Endotracheal intubation must be considered an adjunct to the initial resuscitation, & it should not delay or interrupt chest compressions.

Alternative airway adjuncts, such as double-lumen, combination endotracheal-esophageal tubes (CombiTube) or Laryngeal Mask Airways (LMAs) can be used to manage the airway during resuscitation.[17,18] The CombiTube and LMA have an advantage in that they can be inserted blindly in seconds without laryngoscopy, thereby minimizing interruption of chest compressions.[12] One reported disadvantage of these devices is the inability to deliver medications by the endotracheal route. However, it is unclear whether endotracheal medications have any significant beneficial effect during cardiac arrest.

Artificial circulation

In the pulseless patient, circulation of blood can be accomplished by mechanical compression of the heart and chest. The critical parameter for restoring spontaneous circulation is the development of adequate pressure in the coronary arteries. Coronary perfusion pressure (CPP) is essentially the difference between pressure in the aorta and pressure inside the heart chambers. With mechanical compression of the heart and chest during resuscitation, the primary perfusion of the heart occurs during the relaxation phase (see Figure 3, right).

CPP is the best predictor of resuscitation success.[19] In humans, return of circulation requires that CPP exceed 15–20 mmHg. Below this level, it is rare for resuscitation efforts to result in ROSC. It is important to realize that chest compressions are the only intervention that will generate coronary perfusion pressure.

Vasoactive medications, such as epinephrine and vasopressin, will augment the CPP developed by chest compressions. Airway adjuncts, other drugs and monitors facilitate other aspects of the resuscitation, but they don't increase CPP. Without advanced monitors, it's impossible to directly measure CPP during resuscitation. However, increasing end-tidal CO_2 and improvement in the ECG (either from asystole to VFib or PEA, or an increase in PEA rate) provide indirect evidence that CPP is adequate.

Don't interrupt compressions

Interruptions of chest compressions must be avoided. Research in animals has shown that stopping compressions for merely four seconds can reduce CPP to zero, negating any benefit of previous chest compressions. Much like priming a pump, 10–20 chest compressions after the interruption may be necessary to build up adequate perfusion pressure.

Because CPP is so critical for resuscitation, every other intervention in OOHCA should be considered an adjunct to chest compressions and, with the exception of defibrillation, should not cause chest compressions to stop.

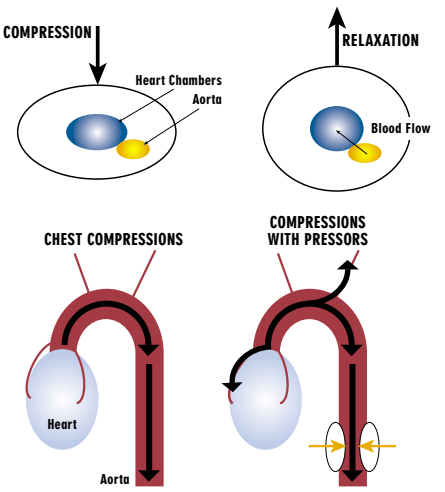

Figure 3: Chest compressions create a pressure gradient between the aorta and the inside of the heart chambers. This gradient is the coronary perfusion pressure (CPP) which drives blood flow. Chest compression (top left) increases pressure in both aorta and heart chambers. During relaxation (top right), pressure persists in the aorta, driving blood flow. During chest compressions without drugs, much of the blood in the aorta runs off into the body as well as into the heart (bottom left). Adding vasoactive drugs, such as epinephrine or vasopressin, increases the effectiveness of chest compressions by constricting the peripheral arteries, and shunting blood to the heart (bottom right).

Current research is examining whether prolonging the intervals between ventilations is beneficial (for example, increasing the interval from two breaths every 15 chest compressions to every 30 chest compressions), but these changes are still investigational.

Delivery of chest compressions is often inadequate, and uninterrupted chest compressions are critical for restoration of circulation.[20–22] Many studies have shown that medical providers at all levels of training perform inadequate chest compressions. Common mistakes include not compressing the chest deeply enough or quickly enough to generate CPP. It has also been shown that as rescuers become fatigued they become less effective. The effects of fatigue can be measured as early

as three minutes after beginning chest compressions even though the rescuer does not yet "feel tired." Therefore, rotating the provider delivering chest compression after every two to three minutes should increase the overall quality of chest compressions.

Several mechanical devices have been designed to provide more consistent and continuous chest compressions.[23] However, no device has gained widespread use because of weight and cost. Persistent interest in these devices highlights the need for strategies to improve chest compression delivery.

ECG monitoring

Continuous ECG monitoring is essential during resuscitation. ECG rhythms can be categorized as *organized* and *not organized*. Organized rhythms are supraventricular rhythms (e.g., sinus rhythm, atrial fibrillation, supraventricular tachycardia) or ventricular tachycardia. Disorganized rhythms include VFib and asystole.

Organized rhythms can support pumping of blood unless they are too slow (< 30–40 complexes per minute) or too fast (> 170–180 complexes per minute). Disorganized rhythms cannot support the pumping of blood under any circumstances. Therefore, only two actions must be based on the ECG:

1. If the rhythm isn't organized, perform an intervention to make it organized; and
2. If an organized rhythm is too fast or too slow, perform an intervention to correct the rate.

Transthoracic electrical (rescue) shocks are highly effective for converting VFib into an organized rhythm when VFib is of very brief duration (less than one to two minutes). However, repeated unsuccessful shocks may directly damage the myocardium. Further, rescue shocks are more likely to fail when cardiac arrest has lasted more than a few minutes.

In the prehospital setting, only 9% of rescue shocks restore an organized ECG if the collapse was not witnessed by the EMS provider.[24] Resuscitation is less likely after rescue shocks that convert VFib to asystole.[25]

Optimal therapy should provide rescue shocks at the lowest effective energy, while minimizing the number of unsuccessful rescue shocks. Thus, it will become important in the future to determine when a rescue shock is likely to restore electrical activity and when a shock will harm the patient by causing an unrecoverable asystole.

Improving shock success

Several parameters can improve rescue shock success. Studies show that increasing the pressure of paddles against the chest wall from 0.5 kg to 8 kg (1 to 17 lbs.) improves conduction of electricity through the chest by as much as 14%.[26,27] This advantage of paddle use must be weighed against the increased safety and convenience afforded by hands-free adhesive defibrillation pads.

In the past, multiple shocks would be delivered in rapid succession to decrease chest impedance. However, repetitive shocks decrease chest impedance by only a small amount.[26]

Biphasic shocks have been shown to be more efficacious than monophasic shocks, and biphasic rescue shocks appear to cause less cardiac dysfunction after ROSC. Finally, VFib becomes less coarse as soon as chest compressions stop, suggesting that the rescuer should strive for the briefest delay from the last chest compression to the rescue shock.

The need to prime the cardiac pump

During cardiac arrest, the appearance of VFib and the response of VFib to rescue shocks vary over time, leading researchers to suggest a phased approach to treating cardiac arrest. VFib encountered within minutes of collapse usually appears coarse and should be immediately defibrillated. However, VFib lasting longer than three to four minutes usually appears less organized, and a few minutes of chest compressions can improve rescue shock success.

Two clinical studies have found that 90 seconds to three minutes of chest compressions prior to delivery of the initial rescue shock improved resuscitation rates for subjects with VFib outside the hospital, particularly when rescuer response intervals are longer than four minutes.[28,29] Thus, defibrillation should be provided immediately for VFib shortly after a witnessed collapse, but a brief period of artificial circulation should precede any shock delivery to VFib that has lasted longer.

Providers should follow their local protocols, but be aware that the most recent research questions the traditional teaching that all VFib should receive a rescue shock as soon as recognized. In the future, providers may distinguish between "fresh," very coarse VFib that is more likely to convert with a rescue shock and "later," fine VFib that will benefit from a period of chest compressions to prime the cardiac pump with oxygenated blood.

Asystole is the extreme example of a disorganized cardiac rhythm and may be impossible to distinguish from very fine VFib. In general, the amplitude of VFib decreases over time because of the cardiac hypoxia and ischemia that develops during cardiac arrest. There's no official cutoff between very fine VFib and asystole, and both rhythms are unresponsive to rescue shocks. However, many patients in asystole will exhibit higher amplitude VFib after a few minutes of chest compressions, indicating that patients can traverse the spectrum from coarse VFib to asystole in two directions. Increasing amplitude and organization of VFib provides a marker of adequate chest compressions.

Particularly when supplemented by vasopressors, chest compressions may restore VFib to a point where rescue shocks can result in ROSC. Thus, treatment of asystole is primarily to provide adequate perfusion to the heart.

An organized rhythm without pulses is called pulseless electrical activity (PEA). The absence of perfusion in the presence of organized electrical activity may result from damage to heart muscle (as in massive myocardial infarction) or from uncoupling of electrical and mechanical activity (as in prolonged circulatory arrest).

For rhythms that are too fast, the ventricles of the heart may not fill with blood between contractions, resulting in the absence of palpable pulses during ventricular tachycardia, supraventricular tachycardia or atrial fibrillation with rapid ventricular response. These tachyarrhythmias should be corrected by immediate electrical cardioversion.

Outside of these tachyarrhythmias, the rate of complexes in PEA is related to the level of hypoxia in the heart and may be used to monitor resuscitation efforts. With increasing hypoxia/ischemia, the heart's energy stores are depleted and the rate of PEA will slow. If resuscitation is delivering

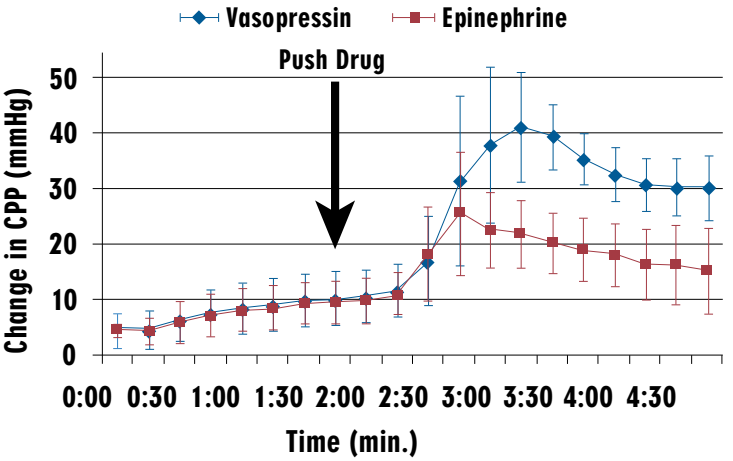

Figure 4: Vasopressin and epinephrine increase CPP produced by chest compressions in a laboratory model. CPP increases only after about 60 seconds of chest compressions. The effect of vasopressin is greater and longer lasting than that of epinephrine. Blood flow will reoxygenate and restore the heart only when CPP has been increased. Thus, when treating cardiac arrest, it is unreasonable to expect a physiological response to a pressor drug until at least 60 seconds of chest compressions, and it is more likely that the state of the heart will be changed after 120–180 seconds of drug-augmented chest compressions. (Unpublished laboratory data).

oxygen and improving the energy state of the heart, the rate of PEA will accelerate.

Narrow complexes reaching rates of 80–100 beats per minute often signal the return of pulses. Falling rates of complexes in PEA reflect poor heart perfusion, resulting in ischemia of the cardiac conduction system. Thus, the cornerstones of PEA treatment consist of cardioversion when appropriate and providing adequate perfusion of the heart.

Drug therapy

Drug therapy in cardiac arrest can be divided into three categories: vasopressor, antidysrhythmic and metabolic drugs. There's good evidence that vasopressors improve the CPP developed by chest compressions, making resuscitation more likely. Antidysrhythmic drugs are effective for preventing dysrhythmias and, therefore, have a role in stabilizing the heart after defibrillation or ROSC. The value of antidysrhythmic drugs for terminating VFib or reversing asystole is less clear. Metabolic drugs, such as bicarbonate, can be used to reverse acidosis or other electrolyte problems when they are recognized. However, there are no data to support the routine use of these drugs for all patients.

Vasopressors used during resuscitation include epinephrine and vasopressin. Both of these drugs can increase coronary perfusion pressure via actions on alpha-adrenergic (epinephrine) or vasopressin receptors[30,31] (see Figure 4, above). Epinephrine is usually administered in 1 mg (~0.015 mg/kg) increments. In laboratory studies, the pressor effects of epinephrine during cardiac arrest are brief (~5 minute).

Vasopressin is usually administered as 40 unit boluses (~0.5 units/kg) and produces a longer lasting increase in coronary perfusion pressure (~10 minutes).

In clinical studies, resuscitation rates and survival are identical overall for patients resuscitated with vasopressin and standard doses of epinephrine.[30,31] These studies found that vasopressin might be superior for patients whose first ECG rhythm is asystole and for those subjects requiring multiple doses of vasopressors. At present, use of either epinephrine or vasopressin is justified in the setting of cardiac arrest.

Future studies may examine whether combinations of epinephrine and vasopressin are more effective than either drug alone.

The role of antidysrhythmic drugs during cardiac arrest is uncertain.[32,33] Atropine may relieve bradycardia, but is ineffective if the bradycardia is caused by ischemia or hypoxia. Only case reports support the use of atropine for treatment of asystole.

Lidocaine, procainamide and bretylium have a long history of use in the treatment of VFib because these drugs are known to suppress ventricular ectopy. Once VFib is established, however, lidocaine increases the electrical energy required to defibrillate by more than 50%, making rescue shocks less effective.[34] Amiodarone, bretylium and procainamide don't have this effect. Consequently, administration of amiodarone (5 mg/kg) is superior to placebo and to lidocaine for restoration of pulses in prehospital patients with VFib that's not terminated by three rescue shocks.[35,36]

Many antidysrhythmic drugs also cause hypotension after bolus injection. That is counterproductive in OOHCA and can reduce the effects of vasopressors given to increase pressure. In summary, antidysrhythmic drugs are commonly used during resuscitation, but only amiodarone use has supporting human data.

Other drugs, such as bicarbonate, aminophylline, calcium and magnesium, have been suggested as treatments for OOHCA. However, none of these medications are supported by research and may not be useful outside very specific situations. Therefore, the pharmacological approach to treatment of cardiac arrest is fairly simple. First, the vasopressors epinephrine and vasopressin are useful for augmenting CPP generated by chest compressions. Other vasopressors could also be useful, but lack prospective data. Second, antidysrhythmic drugs are useful for maintaining organized rhythms, but not for terminating VFib. Only amiodarone has clinical data supporting its use during VFib that persists after rescue shocks. All other drug therapy should be based on the clinical situation and the response of the patient.

Minimizing brain injury

Management of the patient after ROSC affects ultimate outcome. Because two-thirds of patients admitted to the hospital after ROSC fail to emerge from coma, improvements in treatment of anoxic brain injury are essential for meaningful improvement in overall survival.

Brain injury continues to develop during the hours and days after resuscitation, and involves complex biochemical

events.[37] Unfortunately, no drug to date has demonstrated reduction of brain injury in human trials. Data do suggest that avoiding fever, hypotension and hyperglycemia improves neurological recovery after cardiac arrest.[38–40] These data are prompting efforts to provide systematic brain-oriented intensive care for the patients with ROSC who are admitted to the hospital.

Two human trials have found that induction of mild hypothermia (33–34° C) for 12–24 hours after resuscitation improves survival and neurological recovery.[41,42] Laboratory studies suggest that cooling should be accomplished as soon as possible and is probably ineffective if started more than four to six hours after resuscitation.

During resuscitation, many patients spontaneously cool to between 35° C and 35.5° C when peripheral blood equilibrates with core blood.[41–43] Thus, only a small amount of additional cooling is necessary to reach a therapeutic range. It's possible that, in the future, prehospital providers may initiate cooling in resuscitated patients using a combination of techniques, including cold IV fluids and cooling blankets.

The brain is very sensitive to low blood pressure after cardiac arrest, and low blood pressures during the first two hours after resuscitation are associated with poor neurological recovery.[39] Complicating this fact, the heart is also stunned by cardiac arrest, and resuscitated patients usually require drugs to support circulation during the first hours and days after resuscitation. Providers should anticipate that the initial high blood pressure observed moments after restoration of pulses will decline quickly, and prepare to use dopamine or other vasopressors to help maintain brain perfusion (see Figure 5, below).

Final word

Out-of-hospital cardiac arrest should not be approached with a one-size-fits-all treatment plan. Just as with other diseases, OOHCA patients will respond differently to treatments depending on the cause of the collapse and the length of time the patient has been without a pulse.

The importance of CPR and its delivery is being rediscovered. Providers must place more emphasis on external chest compressions and less on advanced airway management.

In the near future, EMS providers may be asked to determine if a patient requires immediate defibrillation or be likely to benefit from CPR and medications prior to delivering defibrillation.

Finally, as our ability to resuscitate patients improves, prehospital providers will have to provide the next segment of care by aggressively supporting blood pressure and initiating hypothermia to improve the ultimate outcome.

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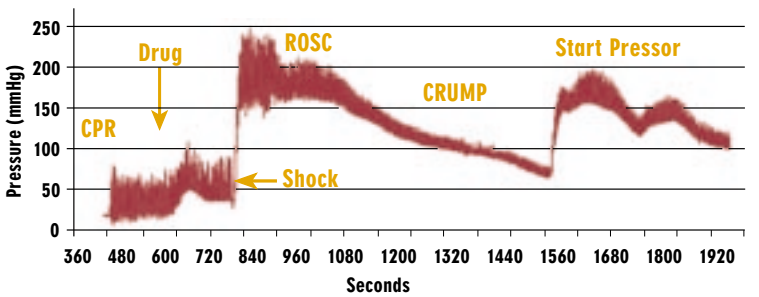

Figure 5: Medications given during resuscitation wear off shortly after the return of spontaneous circulation. In this graph of mean arterial pressure, an experimental animal resuscitated from cardiac arrest shows a high pressure initially after a rescue shock produces ROSC. However, this initial hypertension rapidly dissipates. This rapid decline is known as the "CRUMP" and will cause the patient to lose pulses again if not anticipated and countered with an infusion of some vasopressor. Note the increase in pressure produced by chest compressions (CPR) after administration of a drug. (Unpublished laboratory data).

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