Cardiac Arrest Care:

Out with the Old CPR, In with the New

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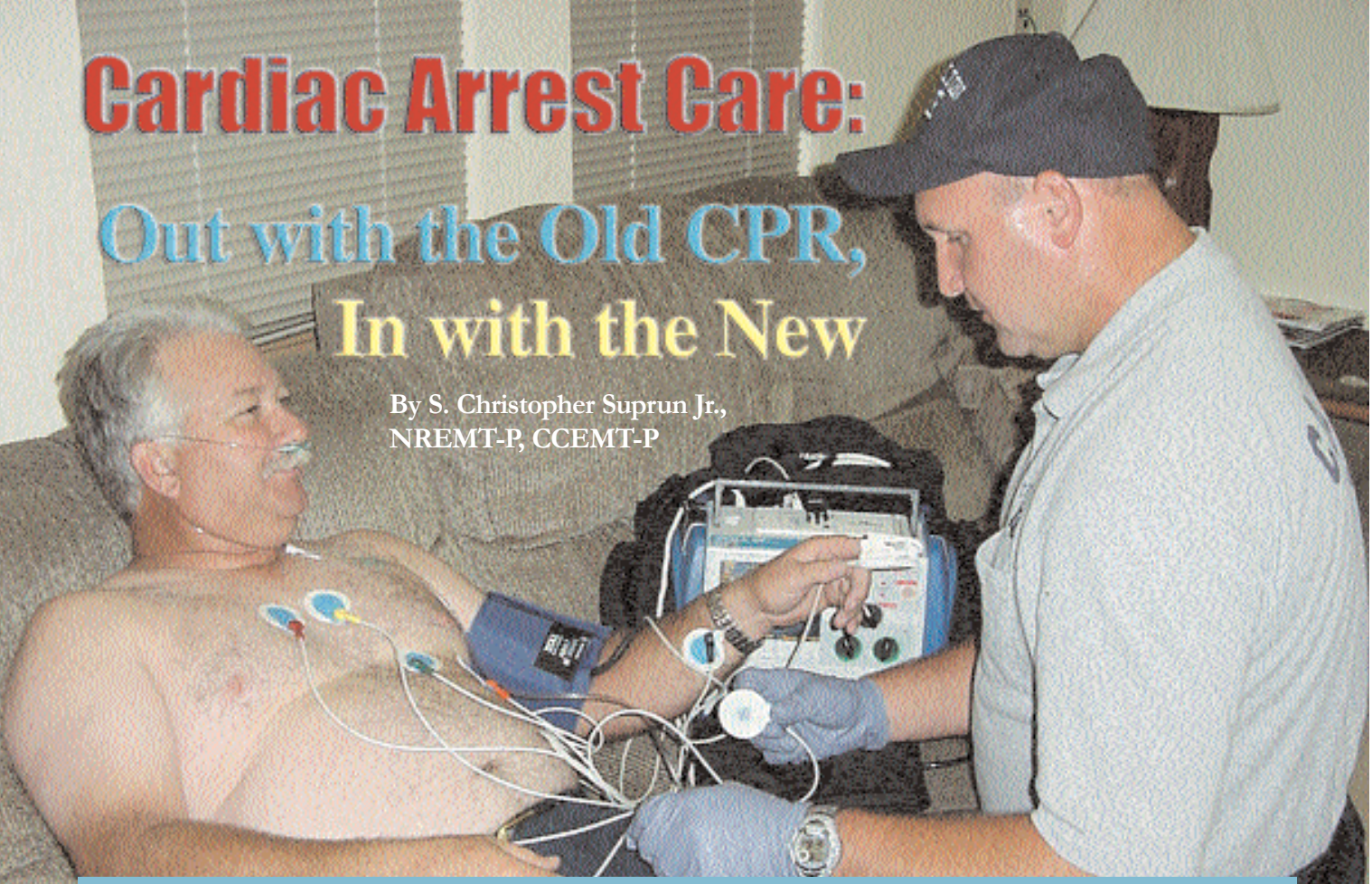

Understanding the prehospital implications of new considerations in cardiac care

For almost two decades, EMS providers have accepted 200, 300, 360 joules as the set of defibrillations delivered by either AEDs or manual defibrillators for ventricular fibrillation or pulseless ventricular tachycardia. The concept of shock early/shock often, however, may be one of many areas that changes dramatically when the American Heart Association releases its new Consensus on Science report in the December issue of the *Journal of the American Medical Association*. The report may have us changing how we think about cardiac care—saying CBA instead of ABC—and creating a whole new set of treatment options.

In some cases, these treatment options may seem more basic, like returning to the original work of Drs. George Crile, James Elam and Peter Safar, and, in reality, some are. Cardiac arrest survival, of course, has long been a losing proposition. With the science that follows, though, EMS, as the front-line response, may be able to catch up on preventable deaths by improving the effectiveness of CPR.

Circulation

In terms of cardiac arrest and cardiopulmonary resuscitation, there has always been one option only: airway. Specific to ventricular fibrillation, immediate defibrillation is the ultimate choice in care.

New science reveals that while maintaining the airway and defibrillation are important, they may both be secondary to moving blood around the body.[1,2] One study noted that while defibrillation is the "essential intervention" for ventricular fibrillation, defibrillation alone is not a cure-all.[3] The new data may send EMS in some entirely new directions in the very near future.

Chest compressions have always been considered important in cardiac arrest, but the reasons are changing, and their value may be misunderstood. For the most part, CPR has been considered a bridge to defibrillation, but new science suggests that CPR doesn't just extend the timeline until defibrillation in VF/VT events, but also may improve tissue perfusion by moving blood.

As you may remember from your initial EMS classes, the body burns ATP, or adenosine triphosphate, which is produced using oxygen and glucose stores. When the body is in cardiac arrest, however, there is no oxygen being moved to produce new ATP, and the heart uses its stores quickly because of the body's fight-or-flight response to maintain homeostasis. Without new ATP, the body starts to burn sugar without oxygen, producing lactic acidosis. By moving oxygenated blood through the heart, the myocardium can produce ATP as it normally would. It is believed that when provided its normal "food," the heart will be more inclined to benefit from defibrillation.

Science goes so far as to say that the old plan of action, which said you should use an AED early, may have been wrong. In one study, the authors noted that the time interval between using an AED versus a manual defibrillator could result in a worsened outcome,[4] and there is clearly a benefit to doing compressions before defibrillating in downtimes of four minutes or more.

Another important consideration in cardiac arrest is coronary perfusion pressure during CPR. In its 2000 standards, the American Heart Association changed the chest compression-to-ventilation ratio from 5:1 to 15:2. This change was important, because it was taking multiple compressions to build up the minimum coronary perfusion pressures to feed the heart. Typically, this number is a minimum of 20 mmHg; ideally, it will be closer to 40 mmHg. The interruption in compressions to provide ventilation or allow the AED to perform its rhythm analysis was causing loss of this minimal pressure of 20 mmHg, which, in some cases, lasted as much as 20 seconds.[5] The 15:2 ratio seems to provide better coronary perfusion pressures longer in the resuscitation.

Another study showed that continuous chest compressions produce "superior neurological outcome."[6]

To achieve good compressions, EMS providers may be able to use a new device that is having extraordinary success in its early use and study. It may be able to assist the process of continuous compressions without providers getting tired or performing poorly. The device is the AutoPulse by ZOLL Medical Corporation. Though the device is relatively new, vest CPR is not. In a 1990 study, vest CPR successfully increased aortic and coronary perfusion pressures.[7]

The AutoPulse is a portable mechanical device that has an automated load-distributed band. This is essentially a fancy definition for a short-board backboard with a band that compresses the chest. The San Francisco Fire Department has been using it, with paramedic captains implementing the device in cardiac arrests. Due to limited availability, there were cases when the device wasn't even implemented until 15 minutes into the cardiac arrest.[8]

Nonetheless, the AutoPulse has produced some surprising results, including a 74% relative increase in return of spontaneous circulation (ROSC) compared with standard CPR practices,[9] and has improved the number of nontraumatic cardiac arrest patients transported to the ED with a pulse by more than one-third.[10]

Based upon the San Francisco Fire Department's experience with the AutoPulse, its medical director, Marshal Isaacs, MD, says the "device potentially could have the most significant impact on survival from sudden cardiac arrest since the advent of CPR."

While studies are ongoing and early data are promising, it should be noted that final study results may not indicate hospital discharge with good neurologic functioning intact.

Whether using traditional CPR or a device designed to provide compressions for emergency responders, it is important to provide good compressions to the patient. The associated increased coronary perfusion pressure from continuous compressions and promoting systemic blood flow over an extended period of time appears to be important for good neurological recovery.[11] One study indicated that patients with average mean arterial blood pressure above 100 mmHg for the two-hour period following post-resuscitation were more likely to have better neurologic outcomes.[12]

Another impact on improving a patient's circulatory status in cardiac arrest is the development of biphasic waveforms in external defibrillation. Defibrillators can transmit energy either in a single direction—monophasic defibrillation—or in one direction for part of the shock and then reverse direction with biphasic defibrillation.

Biphasic defibrillation has been available in implantable defibrillators for several years, but has been used in the prehospital arena only in the last few. Some new studies indicate that biphasic may eventually overtake monophasic defibrillation as the best way to provide current through the chest.

One study, led by the Optimized Response to Cardiac Arrest (ORCA) group, found that larger patients in particular were more likely to respond to a 150-joule biphasic defibrillation and gain a return of spontaneous circulation.[13] This patient population also had better neurological outcomes than patients defibrillated with monophasic energy who were discharged from the hospital. Another study indicated that not only could successful defibrillation occur, but it could be done with 60% less current than monophasic defibrillation.[14]

One interpretation of these results may be that monophasic defibrillation is inefficient, and therefore requires more energy to get the same result. This increased energy level no doubt causes some damage to the myocardium; therefore, biphasic defibrillation may be a better avenue if similar results can be achieved without unnecessary energy. However, this is still an interpretation for discussion and study.

Breathing

For years, we have assumed that if a little oxygen is good, more must be better. Thus started our affinity for 100% oxygen concentrations all the time in patient care. Little by little we are finding that this is not the case—there are patients who don't need higher oxygen concentrations because they aren't using oxygen.

We are now learning that over-ventilation isn't just a little bit bad; it can actually cause that which we are trying to avoid—hypotension. Plus, hyperventilation is occuring on a regular basis. Although the American Heart Association recommends assisted ventilation rates of 12–15 per minute, one study found that EMS providers were ventilating patients 37 times per minute, or three times the recommended rate.[15] The researchers postponed the study until retraining on airway skills was conducted, then resumed their research. Even after retraining, EMS providers ventilated cardiac arrest patients at 22 times per minute, still almost twice the rate recommended.[16]

It is believed that these higher ventilation rates produced overall higher intrathoracic pressures. This additional pressure causes lower cardiac output by limiting venous return. This is exactly the opposite of what we want to do in compressing the chest!

One way to avoid these higher pressures is a new bag-valve mask device, which overcomes provider over-ventilation issues by product engineering. This bag-valve mask has a piston designed to fire when inflation pressures reach 20 mmH_2O—the point at which air pressure will overcome the esophageal sphincter and cause gastric inflation.[17] This piston keeps providers from overinflating the lungs and increases resistance so they cannot easily hyperventilate their patients.

Participants at an EMS conference were studied and recorded bagging a simulated patient with both the new BVM and a standard BVM device. In all categories—respiratory rate per minute, mean tidal volume, mean minute volume, gastric inflation, mean peak pressures and inspiratory-expiratory ratios—measurements were better with the new device.[18] These statistics may lead some to use a device like this for purposes of controlling ventilation.

The Future

Many people are awed by the crews that successfully resuscitate children who suffer near-drowning and survive without oxygen for half an hour or more. But the same mechanism that protects children who fall through ice into freezing water may be used in medicine to protect the neurological outcomes in patients who suffer traditional car-

diac arrest. The mechanism, known as mammalian diving reflex (MDR), slows the rate of consumption by shifting blood flow to only the brain, heart and lungs.

Two studies found that patients had better neurological outcomes when therapeutic hypothermia was introduced. In the first study, researchers found that patients who were cooled for 12 hours to a temperature of approximately 91.4° F had an increased survival and outcome, as opposed to patients who were treated in a normothermic environment.[19]

While this study only involved 77 patients, a second study with a larger number of patients yielded similar results for those who had experienced ventricular fibrillation. This study found that patients who were cooled had both better neurologic functioning and reduced mortality compared with cardiac arrest victims who were treated with standard care and a normothermic protocol.[20]

Another area of frequent debate that will likely remain undetermined with the focus on the basics of cardiac arrest resuscitation is appropriate drugs. The last ACLS update seemed to provide us with a menu for picking the right drug for the right situation.

Amiodarone was considered a good drug for patients with a history of cardiac pump issues; hence, the recurring reference to amiodarone for patients with ejection fractions less than 40%. Procainamide had a place in recurrent ventricular fibrillation. Magnesium sulfate was used for patients in torsade de pointes and might also be used initially for patients with known metabolic abnormalities. Finally, lidocaine was given consideration as a drug for patients who didn't fit into any of the above categories. But what is the correct option?

However, amiodarone does not appear to significantly alter whether or not a cardiac arrest patient will survive to hospital discharge.[21,22] Most of the antiarrhythmics mentioned above seem to have little evidence supporting their use over other drugs when looking at patients being discharged from the hospital.

However, epinephrine, a primary drug used in cardiac arrest, may be used very differently than it has been in the past. In a study of nearly 1,200 patients, when epinephrine was compared to vasopressin, they were considered to have similar effects in ventricular fibrillation, pulseless ventricular tachycardia and pulseless electrical activity. Vasopressin was shown to be superior, however, in the treatment of asystole.[23] In cases of refractory cardiac arrest, epinephrine administration followed by vasopressin appeared to be more effective.

We are now learning that over-ventilation isn't just a little bit bad; it can actually cause that which we are trying to avoid—hypotension.

While vasopressin was introduced to the discussion in a minor way in the previous ACLS update, it is possible that it will take on a much more significant role in our treatment of cardiac arrest patients.

As you can see, new cardiac care studies will likely change the way we deliver care regarding how we maintain airways or push the chest, and whether or not we add ice, pressure or other techniques to our treatment. If nothing else, it appears that more study on the part of both researchers and emergency responders is on the way. ■

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