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Review

The cardiac literature 2010

Amal Mattu MD[a,*], Michael C. Bond MD[a], Semhar Z. Tewelde MD[a], William J. Brady MD[b]

[a]*Department of Emergency Medicine, University of Maryland School of Medicine, Baltimore, MD, USA*
[b]*Department of Emergency Medicine, University of Virginia, Charlottesville, VA, USA*

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1. Cardiac arrest

A. Pokorna M, Necas E, Kratochvil J, et al. A sudden increase in partial pressure end-tidal carbon dioxide ($P_{ET}CO_2$) at the moment of return of spontaneous circulation. J Emerg Med 2010;38:614-621.

During the past decade, there has been a significantly increased focus on optimal performance of high-quality chest compressions for patients in cardiac arrest. Maintenance of proper rate and depth of compressions is critical. Another critical element concerns continuous chest compressions; the interruptions that occur for pulse checks and other interventions are detrimental to maintenance of perfusion and the success of the resuscitation. The use of continuous end-tidal carbon dioxide ($P_{ET}CO_2$) monitoring for patients during chest compressions appears to hold promise for obviating the need for pulse checks.

The authors of this study evaluated 108 patients with cardiac arrest who were receiving advance life support measures and who had continuous $P_{ET}CO_2$ monitoring. The authors retrospectively compared the $P_{ET}CO_2$ of 59 patients who had return of spontaneous circulation (ROSC) followed by stable spontaneous circulation vs that of 49 patients who had no ROSC. The mean initial $P_{ET}CO_2$ of all patients before ROSC (during compressions) was 26.6 ± 12 mm Hg. The mean $P_{ET}CO_2$ after ROSC in the survivors was 36.6 ± 12 mm Hg ($P < .0001$). The mean increase in $P_{ET}CO_2$ after ROSC was 10.0 mm Hg ($P < .001$), and the 95% confidence interval (CI) of the difference was 6.5-13.5 mm Hg. Evaluation of the $P_{ET}CO_2$ waveform from all 59 patients with ROSC demonstrated that the significant increase occurred at the moment of ROSC.

The authors caution against use of absolute levels of $P_{ET}CO_2$ because 6 of 59 survivors started with very low levels (<10 mm Hg), and also 4 of 49 patients without ROSC started with high levels (>41 mm Hg). An abrupt, absolute increase in $P_{ET}CO_2$ during compressions of at least 10 mm Hg appears to be a reliable indicator of ROSC.

Using this information, it would seem advisable to perform continuous compressions without interruptions for pulse checks until either resuscitative efforts are discontinued or an abrupt increase of $P_{ET}CO_2$ ≥10 mm occurs. This study, compromised of a small sample of patients, suggests that $P_{ET}CO_2$ monitoring can be used as a means of determining the ROSC without interrupting chest compressions—further data and additional clinical experience with this recommendation likely are needed before widespread implementation.

B. Simpson PM, Goodger MS, Bendall JC. Delayed versus immediate defibrillation for out-of-hospital cardiac arrest due to ventricular fibrillation: a systematic review and meta-analysis of randomized controlled trials. Resuscitation 2010;81:925-931.

Immediate defibrillation has long been considered to be the most important therapy for patients with out-of-hospital cardiac arrest (OOHCA) due to ventricular fibrillation. The 2005 international resuscitation guidelines, however, suggested that for patients with an unknown downtime or if the downtime was known to be greater than 4 to 5 minutes without bystander intervention (ie, chest compressions), outcomes might improve if defibrillation was preceded by a short period of cardiopulmonary resuscitation (CPR). This suggestion was based on animal as well as human studies [1,2]. Two subsequent prehospital studies, however, demonstrated no benefit in delayed defibrillation [3,4]. Simpson et al, therefore, decided to perform a systematic review and meta-analysis of randomized controlled trials to clarify whether patients with ventricular fibrillation (VF) and delayed defibrillation benefit from a brief period of CPR before defibrillation.

The researchers were able to find only 3 randomized controlled trials that addressed the question at hand. The pooled results demonstrated no benefit to providing CPR before defibrillation vs immediate defibrillation, even when ambulance response times less than 5 minutes and greater than 5 minutes were compared. They also found no harm to performance of CPR before defibrillation. The authors concluded that Emergency Medicine Services (EMS) jurisdictions are justified using either defibrillation strategy. The recently published 2010 American Heart Association (AHA) guidelines [5] also acknowledge that the literature does not support a CPR-first approach, and they have backed away from their prior recommendation.

C. Yu T, Ristagno G, Yongqin L, et al. The resuscitation blanket: a useful tool for "hands-on" defibrillation. Resuscitation 2010;81:230-235.

High-quality chest compressions are clearly considered to be one of the most important interventions for optimizing the chances for survival in

* Corresponding author.
E-mail address: amattu@smail.umaryland.edu (A. Mattu).

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cardiac arrest victims. Elements of "high quality" include the usage of appropriate rate and depth of compressions and also minimization of interruptions of compressions. Intermittent interruptions in compressions, as little as 10 seconds, have been associated with worsened outcomes because of significant reductions in coronary perfusion pressure (CPP) and delays in restoring the threshold values of CPP [6,7]. Even interruptions in compressions for as little as 5 seconds produce decreases in CPP that require as many as 7 chest compressions to restore preinterruption pressures [8,9]. Longer delays are more common during pauses for rhythm interpretation and defibrillations, and these are associated with even longer levels of suboptimal CPPs.

Recently, researchers demonstrated that interruptions in compressions during defibrillation while rescuers "clear" might be unnecessary [10]. In this study assessing "hands-on defibrillation," researchers discovered that rescuers did not perceive the shocks at all if they wore standard examination gloves while performing full-force chest compressions *during* biphasic defibrillation via standard self-adhesive defibrillation electrodes. Unfortunately, there still remains a fear among rescuers of the possibility of electrical injury if contact is made with the patient during defibrillation, although that fear is based on reports in which older monophasic defibrillators were still being used [11].

Yu et al studied the safety and efficacy of a special "resuscitation blanket" to protect a rescuer performing compressions during a defibrillation by attenuating the current flow away from the rescuer's hands. The blanket is made from lightweight insulating materials and is placed over the victim's thorax and chest electrodes. The rescuer performing compressions is in contact with the blanket. The researchers induced VF in pigs and performed a total of 259 biphasic defibrillations using 150, 200, and 360 J. Precordial compressions were performed simultaneously with the defibrillations. In *none* of the defibrillations did the rescuers perceive any abnormal neurologic sensations, discomfort, or skin damage. The researchers also measured CPPs and found that they were maintained successfully during and after the hands-on defibrillations. In a separate series where compressions were interrupted for defibrillations, there was an expected drop in CPP.

The researchers also measured the amount of voltage leak into the rescuer during the defibrillations and found it to be less than 4 μA on average, with a maximal leakage current of 31.9 μA. In the study by Lloyd et al [10], the average and maximal current leakages were 280 and 900 μA, respectively. In either case, these currents are far less than the occupational and medical electrical safety standards for medical equipment, and it is uncertain whether the differences noted between the Lloyd study as compared with the Yu study (with the blanket) produce any difference clinically (for the patient or for the rescuer); the reduced current leakage with the blanket, however, might provide rescuers a greater measure of mental comfort in performing hands-on defibrillations. Most importantly, this study and future studies will hopefully emphasize the potential importance and safety of hands-on defibrillations when biphasic defibrillation is used and thus remove another reason for interruptions in chest compressions.

D. Edelson DP, Robertson-Dick BJ, Yuen TC, et al. Safety and efficacy of defibrillator charging during ongoing chest compressions: a multi-center study. Resuscitation 2010;1521-1526.

Despite studies such as the Yu et al article discussed above, many rescuers will continue to feel uncomfortable about hands-on defibrillation. Defibrillator charging during ongoing compressions is the next best method to minimize hands-off time when patients require defibrillation. Typically, when a patient requires defibrillation, the hands-off time is approximately 15 seconds: approximately 5 seconds for rhythm analysis followed by approximately 10 seconds for charging the defibrillator and delivering the shock. Clinical studies have confirmed the adverse effects of prolonged interruptions for defibrillation and the potential benefit of reducing those interruptions: Eftest et al [12] reported that a 10-second hands-off period before defibrillation decreases the chance of ROSC by approximately half; and Edelson et al [13] reported that the chances of successful defibrillation approximately doubles for every 5-second reduction in the preshock interruption in compressions.

Edelson et al evaluated the safety, efficacy, and hands-off time when compressions were continued during the charging of the defibrillator. Interestingly, the AHA had already made this recommendation in 2005 [14]. Specifically, the AHA recommends that when VF or pulseless ventricular tachycardia (VT) (VF/VT) is diagnosed, chest compressions should resume while the defibrillator is charging until it is time to deliver the shock. As these authors point out, however, there is only a single mention in the guidelines about this recommendation, and the technique is not emphasized in the training materials. Furthermore, the European Resuscitation Council has not made this recommendation. Many rescuers worldwide still withhold chest compressions during the charging.

These researchers analyzed 562 defibrillations from 244 cardiac arrests. In 345 cases (61.4%), the defibrillator was charged during ongoing chest compressions. Two methods of charging during compressions were used. The first was the method recommended by the AHA ("AHA method"): chest compressions were briefly interrupted for rhythm analysis. If VF/VT was diagnosed, compressions were immediately resumed while the defibrillator was charged. As soon as charging was completed, compressions were again briefly interrupted, the patient received the shock, and then compressions were resumed.

In the second method of charging during compressions, the defibrillator was charged during compressions even before rhythm analysis in anticipation of defibrillation ("anticipatory method"). When compressions were interrupted for rhythm analysis, if VF/VT was diagnosed, the patient was immediately defibrillated, and compressions were then resumed. If a nonshockable rhythm was diagnosed instead, the defibrillator was simply disarmed, either manually or automatically after 30 seconds. This method was used in 67 cases.

These 2 methods were compared with the "traditional method" of pausing for rhythm analysis, followed by charging and subsequent defibrillation. The researchers found that total hands-off time during the 30 seconds preceding the shock were as follows: patients receiving the traditional method averaged 14.8 seconds of hands-off time, patients receiving the AHA method averaged 11.5 seconds of hands-off time, and patients receiving the anticipatory method averaged 3.9 seconds of hands-off time. There was no significant difference in the number of patients receiving inappropriate shocks. There was one instance of a shock delivered during compressions, but the rescuer was unaffected by the shock.

Given the importance of minimizing interruptions in compressions for patients in cardiac arrest, rescuers should continue compressions while charging the defibrillator rather than pausing for charging. The technique is feasible, safe, and produces significant reductions in hands-off time. Charging in anticipation of a potentially shockable rhythm decreases hands-off time even more, and it may result in more successful resuscitations.

E. Mosier J, Itty A, Sanders A, et al. Cardiocerebral resuscitation is associated with improved survival and neurologic outcome from out-of-hospital cardiac arrest in elders. Acad Emerg Med 2010;17:269-275.

In 2003, researchers from the University of Arizona published a new protocol for the early management of primary cardiac arrest called cardiocerebral resuscitation (CCR) [15]. This protocol emphasized good quality, minimally interrupted chest compressions, rapid defibrillation for shockable rhythms, early epinephrine, and delayed positive pressure ventilation (bag-valve-mask ventilation, endotracheal intubation) during the first 5 to 10 minutes after primary cardiac arrest. Subsequent studies [16-19] confirmed significant improvements in survival and neurologic recovery for patients in cardiac arrest. Recent reviews and editorials [20-22] have endorsed CCR as an important advance, and the AHA has now also taken a step away from recommending early airway intervention, changing the traditional "mantra" of resuscitation from "A-B-C" (Airway-Breathing-Circulation) to "C-A-B" for cardiac arrest [5].

Mosier et al were interested in finding whether the CCR would be effective in elderly patients, who often have lower oxygen reserve and more comorbidities and might, therefore, not do as well with a protocol that

incorporates delayed airway intervention. They retrospectively evaluated 3515 adult OOHCAs that were presumed to be due to cardiac events (ie, "primary cardiac arrest") in Arizona. Among these patients, 2491 (71%) received standard advanced cardiac life support (ACLS) and 1024 (29%) received CCR. The patients receiving CCR in this study protocol received no positive pressure ventilation (endotracheal intubation) until after 3 cycles (6 minutes) of chest compressions. The primary outcome measures were survival to hospital discharge and cerebral performance category (neurologic outcome). The authors also broke the groups down into age ranges for comparison as well. The authors note that during the study period, 2005 to 2008, very few of the hospitals in the database had protocols for postarrest therapeutic hypothermia.

The overall survival was 5.8%. Patients receiving CCR fared significantly better. The patients younger than 40 years had the most dramatic increase in survival with CCR vs ACLS (18.75% vs 3.74%), but patients in the older groups all fared better with CCR as well: age 60 to 69 years, 10.24% vs 5.99%; age 70 to 79 years, 6.28% vs 4.24%; and age older than 80 years, 4.59% vs 1.85%. Overall, the patients older than 65 years had an odds ratio (OR) for survival benefit of 1.5 when CCR was used, and when adjusting specifically for witnessed VF/VT arrest, the OR was 1.9. The study was too small to assess neurologic outcomes across all age groups, but, overall, the patients who received CCR fared much better. After adjusting for witnessed VF/VT arrest, the CCR group had a 6-fold greater likelihood of a favorable neurologic outcome (OR, 6.54).

This study adds to the growing literature demonstrating the effectiveness of CCR for survival and improvements in neurologic outcome from primary cardiac arrest. Although the most dramatic benefits of CCR occur in younger patients experiencing witnessed arrest and an initial rhythm of VF/VT, the researchers here have demonstrated benefit even in older patients with cardiac arrest and undifferentiated initial rhythms. Acute care providers should be encouraged to adopt CCR in cases of presumed primary cardiac arrest.

F. Larsson IM, Wallin E, Rubertsson S. Cold saline infusion and ice packs alone are effective in inducing and maintaining therapeutic hypothermia after cardiac arrest. Resuscitation 2010;81:15-19.

Therapeutic hypothermia (TH) is currently recommended for resuscitated victims of VF/VT cardiac arrest who remain unconscious, and it should be considered in patients resuscitated from cardiac arrest associated with other initial rhythms as well [23]. In fact, TH was first incorporated into the AHA guidelines in 2005 [14], but many hospitals have been slow to adopt protocols. One possible reason that TH has not been widely adopted is because many protocols for cooling are relatively complicated, involving endovascular catheters, cooling blankets, cooling helmets, or other devices that are expensive and not widely available. Surface cooling with cooling blankets is easier, less expensive, and more widely available. The cooling process can actually be even simpler, however, using nothing more than ice and cool intravenous (IV) fluids.

The authors of this study evaluated induction and maintenance of TH using these simple measures. They prospectively evaluated 38 adult victims of medical cardiac arrest (regardless of initial rhythm) who were resuscitated but had a Glasgow Coma Scale less than 8 upon admission to the intensive care unit. Before cooling, all patients were intubated and sedated (propofol and fentanyl infusions). Patients were cooled using 30 mL/kg of a 4°C IV saline infusion at a rate of 100 mL/min via 2 peripheral IV lines. Ice packs were simultaneously applied to the groin, axillae, and along the neck of the patients. A core body temperature of 32°C to 34°C was the goal, and researchers also planned to maintain this temperature for 24 hours. In cases in which the temperature fell too far, ice packs were removed. If the temperature rose above goal limits, ice packs were replaced. If shivering occurred, sedation was increased. If this was unsuccessful, rocuronium was administered for paralysis. Sedation was not terminated until rewarming was completed.

Using these simple measures, the researchers were able to achieve the goal temperature and maintain TH for the full period. The goal temperature was achieved in an average of 216 minutes. The lowest temperature to which any patient fell was 31.3°C. No patients exceeded the upper limit of 34°C. Passive rewarming was initiated after the 24-hour period of TH. Rewarming was easily and successfully performed. Further details of the rewarming process are beyond the scope of this discussion. From the standpoint of emergency medicine practice, however, the study makes a very important point: expensive equipment and invasive methods are unnecessary for inducing TH in post–cardiac arrest survivors. The simple use of cool IV fluids and ice is feasible, effective, and inexpensive.

G. Dumas F, Cariou A, Manzo-Bilberman S, et al. Immediate percutaneous coronary intervention is associated with better survival after out-of-hospital cardiac arrest: insights from the PROCAT (Parisian Region Out of Hospital Cardiac Arrest) Registry. Cardiovasc Interv 2010;3:200-207.

In recent years there has been increasing literature focusing on the utility of rapid coronary angiography and potential percutaneous coronary intervention (PCI) for patients with ROSC after cardiac arrest. Most these studies have focused specifically on patients with electrocardiographic (ECG) evidence of ST-segment elevation (STE) myocardial infarction (STEMI), and they have demonstrated very good rates of survival to hospital discharge, regardless of initial neurologic status at the time of PCI [24-26]. In fact, the AHA issued a policy statement in February 2010 [27] supporting the use of immediate catheterization in survivors of cardiac arrest who demonstrate evidence of STEMI on ECG: "Patients resuscitated from OOHCA with STEMI should undergo immediate angiography and receive PCI as needed" and when necessary, EMS providers "…should bypass referral hospitals and go directly to a cardiac resuscitation receiving hospital so that these patients can receive angiography within 90 minutes." Many victims of cardiac arrest, however, do not demonstrate STE despite the presence of acute coronary occlusion. In fact, the absence of STE after ROSC does not reliably rule out the presence of a coronary occlusion on acute angiography [27,28]. There has been increasing support in the critical care literature [29] as well as in the cardiology literature [20] for early angiography even in these patients without STE.

The study by Dumas et al provides further support for rapid coronary angiography in survivors of cardiac arrest with or without ECG STE. The authors evaluated 714 victims of OOHCA in Paris who survived to hospital admission. They excluded 279 patients who were considered to have extracardiac causes of cardiac arrest (most often, respiratory etiologies). The remaining 435 patients (61%) underwent coronary angiography. Of these patients, 134 (31%) had STE on ECG and 301 (69%) had no STE. Overall, at least one significant coronary artery lesion was found in 304 patients (70%). When separating these patients based on ECG, the researchers found that 128 (96%) of the 134 patients *with STE* had at least one coronary lesion; 74% of these patients had successful PCI and a resulting survival rate of 54%. On the other hand, 176 (58%) of the 301 patients *without STE* had at least one coronary lesion. Percutaneous coronary intervention was attempted in only half of these patients but was successful in 85% of these patients; 47% of these patients survived. The overall survival rate in the study was 51% if PCI was successfully performed compared with 31% if PCI failed or was not attempted. Successful PCI was found to be an independent predictor of survival regardless of the ECG finding, similar to another recent study [30]. Is it time to provide immediate coronary angiography—and coronary intervention—for survivors of "cardiac-caused" cardiac arrest?

H. Batista LM, Lima FO, Januzzi JL, et al. Feasibility and safety of combined percutaneous coronary intervention and therapeutic hypothermia following cardiac arrest. Resuscitation 2010;81:398-403.

There is little doubt that TH is an important intervention for survivors of cardiac arrest. If studies such as the Dumas et al investigation above are true,

however, it would appear that patients should also be treated rapidly with coronary angiography. The combined use of angiography with TH has been studied and found to be feasible, effective, and safe, [10,30] producing improvements in both long-term mortality and neurologic status with no increase in door-to-balloon time. Despite these early findings, however, concern regarding the safety of the combined approach remains. Hypothermia is known to increase the risk of dysrhythmias, coagulopathy, and infection [31-34]; and it is also known that reperfusing injured myocardium can lead to dysrhythmias as well [35]. Batista et al sought to provide further evidence regarding whether the combination therapy is safe.

Ninety patients within 6 hours of ROSC after cardiac arrest were included in the study. Twenty patients underwent PCI during TH and were compared with a control group of 70 patients receiving TH without PCI. The primary end point was rate of dysrhythmias; and the secondary end points were other adverse events (including hypotension, infection, coagulopathy) and mortality. The study included asystolic and pulseless electrical activity (PEA) patients (comprising 53% of the total patients), unlike many prior studies that only included patients with VF. The researchers found that there was no significant difference between the combination group as compared with the TH-alone group in any parameter. There was no difference in mortality between the 2 groups, unlike aforementioned studies that demonstrated a mortality benefit when combination therapy was used; however, as the researchers speculated, this may have been because of the inclusion of large numbers of asystole and PEA patients, who are known to have poor outcomes, regardless of interventions provided. The researchers concluded that combination therapy of PCI with TH is feasible and safe in cardiac arrest. Further large studies will be needed to confirm whether combination therapy is associated with improvements in mortality and in which subgroups this may be found.

I. Field JM, Hazinski MF, Sayre MR, et al. Part I: executive summary 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122:S640-S656.

In November 2010, the newest set of guidelines pertaining to CPR and emergency cardiovascular care were published by the AHA in *Circulation*. The guidelines consist of 16 parts. They address not only cardiac arrest but also postarrest care, dysrhythmias, acute coronary syndromes (ACS), stroke, cardiac arrest in special situations (eg, in pregnancy, pulmonary embolism, and other), pediatric considerations, and ethics. Part I is a summary statement of the major changes in cardiac arrest and emergency cardiovascular care since the last set of guidelines, which were published in 2005. The highlights of this "executive summary" follow. For purposes of brevity, we include only information pertaining to adult patients with acute cardiac conditions (cardiac arrest and dysrhythmias), excluding ACS, stroke, and pediatric considerations. The reader should note that the bulk of guideline recommendations, such as in past years, are focused on victims of primary *cardiac* arrest and are not necessarily relevant to victims of *pulmonary* arrest (eg, drowning, drug overdose, and other).

1.1. Change from A-B-C to C-A-B

A major change in basic life support (BLS) is a step away from the traditional approach of Airway-Breathing-Chest compressions (A-B-C) to the initiation of good chest compressions first (C-A-B). There are several reasons for this change.

- Most survivors of adult cardiac arrest are patients with an initial rhythm of VF or pulseless VT, and these patients are best managed with initial chest compressions and early defibrillation rather than airway management.
- Airway management, whether mouth-to-mouth breathing, bag valve mask, or endotracheal intubation, often results in a delay of initiation of good chest compressions. Airway management is no longer recommended until after the first cycle of chest compressions—30 compressions in 18 seconds. The 30 compressions are now recommended to precede the 2 ventilations, which previous guidelines had recommended at initiation of the resuscitation.
- Only a minority of victims of cardiac arrest receive bystander CPR. It is believed that one of the obstacles to bystanders performing CPR is their fear of doing mouth-to-mouth breathing. By changing the initial focus of resuscitation to chest compressions rather than airway maneuvers, it is thought that more patients will receive important bystander intervention, even if only chest compressions.

1.2. Basic life support

The traditional recommendation of "Look, Listen, and Feel" has been removed from the BLS algorithm because the steps tended to be time-consuming and inconsistently useful. Hands-only CPR (compressions only, no ventilations) is recommended for the untrained layrescuer to obviate their fears of mouth-to-mouth ventilations and to prevent delays or interruptions in compressions.

Pulse checks by layrescuers should not be attempted because of the frequency of false-positive findings. Instead, it is recommended that layrescuers should just assume that an adult who suddenly collapses, is unresponsive and not breathing normally (eg, gasping), has cardiac arrest; activate the emergency response system; and begin compressions. Pulse checks by health care providers have been de-emphasized in importance. They are often inaccurate and produce prolonged interruptions in compressions. If pulse checks are performed, health care providers should take no longer than 10 seconds to determine if pulses are present. If no pulse is found within 10 seconds, compressions should resume immediately.

The use of end-tidal CO_2 ($ETCO_2$) monitoring is a valuable adjunct here. When patients have absence of spontaneous circulation, the $ETCO_2$ is generally <10 mm Hg. However, when spontaneous circulation returns, $ETCO_2$ levels are expected to abruptly increase to at least 35 to 40 mm Hg. By monitoring these levels, interruptions in compressions for pulse checks become unnecessary.

1.3. Cardiopulmonary resuscitation devices

Several devices have been studied in recent years, including the impedance threshold device and load-distributing band CPR. No improvements in survival to hospital discharge or neurologic outcome have been proven with any of these devices when compared with standard conventional CPR.

1.4. Electrical therapies

Patients with VF/VT should receive chest compressions until a defibrillator is ready. The defibrillation should then be performed immediately. Chest compressions for 1.5 to 3 minutes before defibrillation in patients with cardiac arrest >4 to 5 minutes have been recommended in the past, but recent data have not demonstrated improvements in outcome. Transcutaneous pacing of patients with asystole has not been found to be effective and is no longer recommended.

1.5. Advanced cardiac life support

Adequate BLS, including high-quality chest compressions and rapid defibrillation of shockable rhythms, is again emphasized as the foundation for successful ACLS.

The recommendations for airway management have undergone 2 major changes: (1) the use of quantitative waveform capnography for confirmation and monitoring of endotracheal tube placement is now a class I recommendation in adults and (2) the routine use of cricoid pressure during airway management is no longer recommended.

As they did in 2005, the AHA concedes once again that as of 2010 "there [are] still insufficient data to demonstrate that any drugs improve long-term

outcome after cardiac arrest." Several important changes in recommendations for dysrhythmia management have occurred.

- For symptomatic or unstable bradydysrhythmias, IV infusion of chronotropic agents (eg, dopamine, epinephrine) is now recommended as an equally effective alternative therapy to transcutaneous pacing when atropine fails.
- As noted above, transcutaneous pacing for asystole is no longer recommended.
- Atropine is no longer recommended for routine use in patients with PEA or asystole.

1.6. Postcardiac arrest care

Postcardiac arrest care has received a great deal of focus in the current guidelines and is probably the most important new area of emphasis. There are several key highlights of postarrest care:

- Induced hypothermia, although best studied in survivors of VF/VT arrest, is generally recommended in adult survivors of cardiac arrest who remain unconscious, regardless of presenting rhythm. Hypothermia should be initiated as soon as possible after ROSC with a target temperature of 32° to 34°C.
- Urgent cardiac catheterization and PCI is recommended for survivors of cardiac arrest who demonstrate ECG evidence of STEMI regardless of neurologic status. There is also increasing support for patients without STE on ECG who are suspected of having ACS to receive urgent cardiac catheterization.
- Hemodynamic optimization to maintain vital organ perfusion, avoidance of hyperventilation, and maintenance of euglycemia are also critical elements in postarrest care.

The AHA 2010 guidelines represent an important advance in the care of victims of cardiac arrest. Most important is the stronger emphasis on postcardiac arrest care. Induced hypothermia is more strongly emphasized, and perhaps, the most important advance is the recommendation for urgent PCI in survivors of cardiac arrest. The wealth of data, thus, far indicates that postarrest PCI may be the most important advance toward improving survival and neurologic function since defibrillation was first introduced decades ago. Optimal management of cardiac arrest in the current decade can be summarized simply by "the 4 *C*s": Cardiovert/defibrillate, Compressions, Cooling, and Catheterization.

The AHA hesitated in adopting the full concepts of CCR (discussed above). Although CCR also promotes the C-A-B approach to resuscitation, it promotes even further delays in airway intervention—withholding any form of positive pressure ventilations, in favor of persistent chest compressions, for as many as 5 to 10 minutes after the cardiac arrest. The current guidelines only recommend withholding positive pressure ventilation for a mere 18 seconds. As supporting evidence for full CCR continues to mount, it is anticipated (and hoped) that the AHA will finally endorse and promote this form of BLS.

2. Acute coronary syndromes

A. Body R, Carley S, Wibberley C, et al. The value of symptoms and signs in the emergent diagnosis of acute coronary syndromes. Resuscitation 2010;81:281-286.

Classic teaching regarding the presenting features of acute myocardial infarction (AMI) includes midsternal chest pressure beginning with exertion or, of even greater concern, having onset at rest; the discomfort radiates to the left arm, neck, or jaw; and it is associated with dyspnea, diaphoresis, nausea, and vomiting. The experienced clinician, however, knows that such presentations are not the norm and, in fact, are not always the most common. The authors of this study sought to evaluate the utility of classic as compared with atypical symptoms in patients with proven AMI.

The study evaluated 796 patients older than 25 years presenting to an emergency department (ED) in the United Kingdom with symptoms concerning for ACS. The primary outcome that was evaluated was a diagnosis of AMI, and the secondary outcome was the occurrence of adverse cardiac events (death, AMI, or need for urgent coronary revascularization) at 6 months. Presenting symptoms and signs consisting of 35 variables (including vital signs and elements of the history of present illness) were assessed and correlated with the primary and secondary outcomes.

Acute myocardial infarction was diagnosed in 148 patients (18.6%) based on troponin testing. Not surprisingly, ischemic findings on the ECG had the highest association with the primary and secondary outcomes. Among the symptoms and signs associated with AMI, observed diaphoresis had the highest OR (5.18), followed by vomiting (OR, 3.50), central location of chest pain (OR, 3.29), radiation to both arms (OR, 2.69), and radiation to the right arm (OR, 2.23). On the other hand, many of the "classic" descriptors and associated symptoms typically described with infarction did *not* alter the probability of AMI, including rest pain, pain radiating to the left arm or to the neck/jaw/throat, pain reported to be similar to a patient's prior myocardial infarction, heaviness or pressurelike in nature, tightness/squeezing pain, or tachycardia. All of the above findings were similarly correlated with the 6-month secondary outcome as well.

Also of interest was the finding that typical "negative predictors" of AMI, such as pleuritic chest pain, burning/indigestion–like pain, sharp/stabbing pain, chest wall tenderness, and abdominal tenderness, did *not* decrease the probability of AMI. The only factor that was found to decrease the probability of AMI was left anterior location of pain (OR, 0.25), although readers should be wary of making conclusions based on this finding. This is likely because of the larger number of patients with noncardiac disease who tend to present with left-sided chest pain (eg, gastric causes) and also laypersons with even mild pain tend to seek medical attention more frequently when the pain is left sided because of the common thought that the heart is on the left side of the chest.

The takeaway point here is very simple: "atypical" symptoms should not necessarily be considered benign and that the classic textbook descriptions are less reliable in "real" patients. We certainly should not abandon the use of the history in the evaluation of the patient with chest pain suspected of ACS. Yet, we should "have an open mind" when we approach the patient with chest pain and not "rule out" the possibility of the diagnosis based upon single elements of the history. Remember that medical decision-making includes an analysis of all features of the presentation, including the history of the present illness.

B. Walker J, Galuska M, Vega D. Coronary disease in emergency department chest pain patients with recent negative stress testing. West J Emerg Med 2010;11:384-388.

Cardiac stress testing is an important element in risk stratification of patients and prediction of future cardiac events. However, the utility of a recent negative stress test (ST) is limited when it is used to determine the risk of a patient presenting to the ED with anginal symptoms. Almost every experienced emergency physician has cared for patients with true ACS or even primary cardiac arrest despite having a recent negative ST. Unfortunately, over-reliance on negative tests, especially STs, is a common reason for misdiagnosis or delays in diagnosis in patients with ACS. Walker et al have provided us with a nice reminder that negative STs are certainly not a guarantee of cardiac health.

The authors performed a retrospective chart review of adult patients presenting to their community-based teaching hospital with a chief complaint of chest pain, with a negative or inconclusive recent ST within the preceding 3 years. Various types of STs were included (eg, treadmill ECG study, treadmill echocardiogram, treadmill nuclear study, pharmacologic echocardiogram, or pharmacologic nuclear study). Patients were excluded if they had a cardiac catheterization or coronary artery bypass graft surgery between the time of their most recent ST and the ED visit. Patients

were then evaluated for significant coronary artery disease (CAD) within 30 days of the ED visit. *Significant CAD* was defined as an AMI with positive cardiac biomarkers, subsequent positive ST (of any type), cardiac catheterization requiring intervention, coronary artery bypass graft surgery, or death due to medical cardiac arrest.

A total of 164 patients were evaluated, of which 34 (20.7%) ruled in for CAD. There was no significant difference between the patients with true negative STs vs inconclusive STs: 122 patients had negative STs, of which 25 (20.5%) ruled in for CAD; and 42 patients had inconclusive STs, of which 9 (21.4%) ruled in for CAD. Of the 34 patients who ruled in for CAD, 16 (47.0%) had their most recent ST within 6 months of admission, and 8 (23.5%) had their most recent ST *within 1 month* of admission.

The key takeaway point here is very simple and provides confirmation of the anecdotal experience of seasoned emergency physicians: recent negative STs do not exclude ACS in patients presenting with anginal symptoms. It is critical to remember that cardiac tests (eg, ECGs, troponins, STs, and even coronary angiography) are useful for risk stratification, but no test is capable of stratifying a patient's risk to zero—such stratification is not possible.

3. Acute cardiogenic pulmonary edema

A. Ferrari G, Milan A, Groff P, et al. Continuous positive airway pressure vs. pressure support ventilation in acute cardiogenic pulmonary edema: a randomized trial. J Emerg Med 2010;39:676-684.

Acute cardiogenic pulmonary edema (ACPE) is a common emergency in EDs around the country. This presentation is likely to increase in the coming years as the number of patients with chronic congestive heart failure increases. The mainstay of therapy includes vasodilators, diuretics, morphine, and supplemental oxygen. Despite these standard therapies, some patients are not able to improve gas exchange and require airway support. In recent years, noninvasive ventilation (NIV) has gained support in the common management of patients with ACPE. The 2 forms of NIV that are used are continuous positive airway pressure (CPAP) and pressure support ventilation (PSV; also commonly referred to as bilevel positive airway pressure [BiPAP]).

The authors conducted a prospective randomized trial to evaluate and compare the 2 modalities in their effects on endotracheal intubation (ETI) rates, mortality, improvement in gas exchange especially in those with hypercapnea, duration of ventilation, and hospital length of stay. Acute cardiogenic pulmonary edema was defined by dyspnea at rest, bilateral infiltrates on chest radiograph, Pao_2/Fio_2 less than 200, use of accessory muscles, and respiratory rate greater than 30. Patients with any confounders such as hemodynamic or respiratory instability, AMI, pulmonary embolism, pneumonia, chronic obstructive pulmonary disease, and altered mental status were excluded from the study. Patients were all treated with oxygen (Fio_2, 60%), furosemide 60-mg IV, morphine 2-mg IV, nitrate infusion titrated until resolution of dyspnea or followed by a sodium nitroprusside infusion if not responding to nitrates, and either CPAP or PSV. After 1 hour of therapy, all patients were evaluated for the need for ETI; criteria included Pao_2/Fio_2 less than 100, inability to improve respiratory rate or arterial blood gas, psychomotor agitation and mask intolerance, hemodynamic instability, and inability to handle secretions. A total of 80 patients were evaluated (40 CPAP and 40 PSV), of which 0 patients in the CPAP group and 3 patients (7.5%) in the PSV group required ETI. There was no statistical difference in these ETI rates. There was also no significant difference between the 2 groups in improvements in gas exchange (including patients with hypercapnea), duration of assisted ventilation, and hospital length of stay. There was a trend toward decreased mortality in the CPAP group (2 [5%]) vs the PSV group (7 [17.5%]), but the difference was not statistically significant.

The study supports several other recent meta-analyses (please refer to next article) that demonstrated equivalence, effectiveness, and safety of either form of NIV [36-38]. The authors, however, suggest that CPAP should be considered the preferred first-line treatment because of ease of use and lower cost.

B. Weng CL, Zhao YT, Liu QH, et al. Meta-analysis: noninvasive ventilation in acute cardiogenic pulmonary edema. Ann Int Med 2010;152:509-600.

As noted above, meta-analyses evaluating CPAP and BiPAP have been fairly consistent in touting the benefits of NIV in patients presenting with ACPE [36-38]. The studies demonstrate that NIV is effective in preventing ETI, hospital and intensive care unit length of stay, hospital costs, and even mortality. There remains one large well-publicized study, however, that opposes these data. Gray et al [39] evaluated CPAP and BiPAP vs standard oxygen therapy in patients admitted with ACPE, and they found that although patients' symptoms and acidosis resolved more quickly with NIV, there was no difference in 7-day intubation rates or mortality. This study has been widely criticized for the following reasons: patients included in the study had a very high acuity, with an average of pH 7.2, indicating that patients were already on the verge of respiratory arrest; and the mortality rate was extraordinarily high—10%. There was no indication of how soon NIV was used, when NIV was used, and it was used for only a minimum of 2 hours, yet outcomes were evaluated 7 days later. In addition, 80% of patients receiving standard therapy later received NIV, which makes a proper comparison between the groups unreliable.

Weng et al chose to perform their own meta-analysis of randomized trials from 1966 to 2009 that compared standard medical therapy, CPAP, and BiPAP in efficacy in lowering incidence of ETI, mortality, and AMI. A total of 31 articles were evaluated: 11 compared CPAP with BiPAP, 10 compared CPAP with standard medical therapy, 5 compared BiPAP with standard medical therapy, and 5 compared all 3 groups. They found that all studies using CPAP reported reduced need for ETI and mortality with no effect on incidence of AMI. Moreover, the decrease in mortality was even greater for those in whom ACPE was caused by AMI or ischemia. Studies using BiPAP reported decreased ETI, with no effect on mortality or incidence of AMI. Studies comparing CPAP with BiPAP reported no significant difference in ETI, mortality, or incidence of AMI.

The authors of this meta-analysis conclude that despite the study by Gray et al [39], overwhelming evidence favors NIV in safety and efficacy for patients with ACPE. Both forms of NIV reduce the need for ETI, and CPAP reduces mortality, especially in patients with ACS.

4. Dysrhythmias

A. Stiell IG, Clement CM, Perry JJ, et al. Association of the Ottawa Aggressive Protocol with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. Can J Emerg Med 2010;12:181-191.

What is the most appropriate approach to atrial fibrillation (AF) in the ED? This question has been asked—and answered—numerous times, and yet there is no firm consensus on the subject today. The 3 primary issues for the emergency physician with respect to AF include the management of the unstable patient, ventricular rate control, and the consideration of rhythm conversion.

This article by Stiell et al addresses the third issue here—rhythm conversion of the ED patient with AF. Numerous studies suggest that a significant portion of patients with new onset AF will spontaneously convert to sinus rhythm within 24 hours of onset and evaluation [40-44]. This very high rate of spontaneous conversion coupled with the results of numerous AF trials demonstrating that rate control is similar to rhythm control in several key end points [45,46]. For instance, the AFFIRM and RACE trials demonstrated no significant difference in the occurrence of study end points representing quality of life issues, control of symptoms, and the occurrence of adverse events between the rate and rhythm control groups [45,46].

In an effort to expedite ED care and reduce hospital admissions, the authors sought to investigate the effectiveness and safety of the Ottawa

Aggressive Protocol in the management of patients with these AF and atrial flutter (AFL). Briefly, the Ottawa Aggressive Protocol consists of a loading dose of IV procainamide (1 g administered over 1 hour); if the patient remains in AF, electrical cardioversion follows in the ED. If clinically appropriate after completion of the protocol-driven therapy, the patient is discharged from the ED with outpatient cardiology follow-up.

The study included 660 patient visits (95% AF and 5% AFL) with a mean age of 65 years. Ninety-seven percent of patients were discharged to home after ED care; of those patients discharged, 93% were in sinus rhythm. Intravenous procainamide converted 383 patients (58%) to sinus rhythm. A total of 243 patients (37%) underwent subsequent electrical cardioversion with a 92% successful conversion rate to sinus rhythm. Thirty-four patients (5%) did not convert to sinus rhythm. Adverse events occurred in 8% of cases, including hypotension (7%) and bradycardia (0.3%); no instances of polymorphic VT, stroke, or death occurred. Recurrence occurred in 9% of patients at 7 days. The ED length of stay was approximately 5 hours in all study patients. For patients converting with procainamide therapy, the length of stay was approximately 4 hours; understandably, those patients requiring electrical cardioversion after failed procainamide infusion remained in the ED for a longer period, averaging 6.5 hours. The authors concluded that the Ottawa Aggressive Protocol is effective, safe, and rapid; furthermore, this protocol has the potential to significantly reduce hospital admissions and expedite ED care.

The Ottawa Aggressive Protocol for the management of AF and AFL does appear to be effective and safe, as outlined by the authors. The emergency physician must also consider the nursing and physician time required to appropriately manage these patients during both the chemical and electrical conversion portions of the protocol. Furthermore, the ED bed space required to execute this protocol must be taken into account; an adequately staffed observation unit is likely the most appropriate ED space to use. Lastly, agreement with the local cardiologists regarding protocol support and timely follow-up is needed. Thus, if one elects to pursue this approach to the patient with AF, time, space, and support are required beyond the relatively simple medicine of the issue.

B. Stiell IG, Dickinson G, Butterfield NN, et al. Vernakalant hydrochloride: a novel atrial-selective agent for the cardioversion of recent-onset atrial fibrillation in the emergency department. Acad Emerg Med 2010;17:1175-1182.

Vernakalant hydrochloride (VH) is an atrial-selective antiarrhythmic agent useful in the immediate treatment of new onset AF. Vernakalant hydrochloride is effective in terminating recent-onset AF; it has been shown to successfully convert AF to normal sinus rhythm in patients with arrhythmia onset less than 7 days before presentation. Vernakalant hydrochloride is not useful in the management of chronic AF or AFL. Vernakalant acts mainly in the atria via blockade of atrial potassium channels, thereby prolonging repolarization; sodium channels are also blocked. The specific potassium channel, which is blocked, is the cardiac transient outward potassium current; this blockade increases as the heart rate increases, a useful feature for the management of AF. Because of its potassium blockade, the QT interval is also prolonged, yet the drug is relatively free of proarrhythmic effect.

The authors of this study evaluated both the efficacy and safety of vernakalant for patients with recent-onset AF. The study was a multicenter, ED-based, retrospective analysis of patients with recent-onset AF, defined as less than 48-hour duration, who were enrolled in the double-blind, placebo-controlled ACT I and the open-label ACT IV trials [47]. Patients received a 10-minute IV infusion of vernakalant or placebo, followed by an additional infusion if AF persisted. Efficacy was measured by the conversion to sinus rhythm within 90 minutes as well as the median time to conversion; safety was determined by an evaluation of the ECG and the occurrence of adverse events. Two hundred ninety patients (with a mean age of 59 years) were entered into the study with 229 receiving vernakalant and the remainder placebo. Sixty percent of the VH patients converted to sinus rhythm within 90 minutes, whereas only 5% of placebo patients converted. Of those VH patients who converted to sinus rhythm, the median time to conversion was 12 minutes. Adverse effect was very rare; clinically significant bradycardia and hypotension were uncommon, and no cases of torsade de pointes or VF occurred. In this study, VH safely and effectively converted a significant number of patients to sinus rhythm.

Although a 60% conversion rate is impressive, 40% of patients remain in AF, requiring additional therapy. This conversion rate is not dissimilar from other agents used in similar circumstances for AF in the ED. The primary benefits of vernakalant appear to be its rapid effect and relatively safe use. This drug should be watched closely. If subsequent studies support the early investigations into this agent, particularly in rapid effect and safe use, vernakalant will become an important tool in the management of recent-onset AF in the ED.

5. Syncope

A. Thiruganasambandamoorthy V, Hess EP, Alreesi A, Perry JJ, Wells GA, Stiell IG. External validation of the San Francisco syncope rule in the Canadian setting. Ann Emerg Med 2010;55(5):464-472.

Syncope, a transient loss of consciousness, is estimated to be responsible for 1.4% of ED visits and 0.6% of hospital admissions with an estimated cost of $2.4 billion annually [48-51]. Numerous clinical decision rules (CDR) have been made in an attempt to identify patients who are at risk for a serious outcome [50,52-55]. One of the most commonly discussed rules is the San Francisco Syncope Rule (SFSR) that attempts to identify patients at risk for a serious outcome within 30 days of their ED visit [50]. The SFSR defined a serious outcome as death, myocardial infarction, arrhythmia, pulmonary embolism, stroke, subarachnoid hemorrhage, significant bleeding at any site, any procedural intervention to treat a related cause of syncope, or a condition causing or likely to cause a return ED visit, or hospitalization, for a related event within 30 days. During its validation trial, it had a sensitivity of 98% and specificity of 56% in identifying patients at risk for a serious outcome if they had any of the following: history of congestive heart failure, hematocrit less than 30%, abnormal ECG, history of shortness of breath, or a triage systolic blood pressure less than 90 mm Hg. In additional external validation tests, however, the SFSR has not fared as well. In this study, Thiruganasambandamoorthy et al attempted to validate the SFSR in the Canadian setting.

The authors conducted a single-center retrospective review of patients 16 years and older who presented to their tertiary care center with syncope. The authors only included local residents to aid in their ability to verify records from local hospitals and the coroner. They excluded patients who were transferred from another hospital; who were felt to have syncope that was due to alcohol, illicit drug use, or a seizure; who had head or significant trauma; or if the patient had a change in their mental status from baseline when they regained consciousness. Ultimately, 505 patient visits by 490 patients were included in their analysis. Serious outcomes were noted in 49 (9.7%) of the patient visits with 27 outcomes (5.3%) occurring after ED discharge (either as an inpatient or at their place of residence).

There were 5 deaths with 3 occurring after ED discharge. The most common serious outcome was a procedural intervention in 31 patients (63.3%), followed by arrhythmias in 21 patients (42.9%). The analysis showed that the SFSR had a sensitivity of 90% (95% CI, 79%-96%), predicting 44 of the 49 serious outcomes, and a specificity of 33% (95% CI, 32%-34%). The SFSR did not identify 5 serious outcomes—4 occurred in the ED and 1 outside the ED. Three of 4 that occurred in the ED were arrhythmias that were noted at 34, 45, and 395 minutes after arrival to the ED. Of the 3 deaths that occurred after leaving the ED, the SFSR would have predicted all of them.

The most interesting finding is that, if the authors had implemented the SFSR in their population, they would have admitted 69.5% of the patients—in stark contrast to their present admission rate of 12.3%. Ultimately, the SFSR in the Canadian population would have predicted the 3 deaths that were discharged from the ED, although it missed 5 serious outcomes and would have increased their admissions by a rather large 565%. Most hospitals have a historic admission rate of 30% to 40% for syncope;

therefore, although not as drastic, the SFSR would still cause an approximately 2-fold increase in the rate of admissions [56,57].

This study and the other external validation studies of the SFSR have shown that it can not reliably predict all serious outcomes while still reducing admissions. Missing 10% of serious outcomes is not acceptable.

B. Gabayan GZ, Derose SF, Asch SM, Chiu VY, Glenn SC, Mangione CM, Sun BC. Predictors of short-term (seven-day) cardiac outcomes after emergency department visit for syncope. Am J Cardiol 2010;105:82-86.

The SFSR attempted to identify patients at risk for a serious outcome within 30 days of discharge. Because most patients should be able to follow up with their primary care provider within 7 days, the authors of this study attempted to identify historical, physical examination, or diagnostic test results that could predict an adverse outcome within 7 days.

The authors conducted a retrospective cohort study of patients enrolled in an integrated health system (HMO) that consists of 11 Southern California EDs and over 100 outpatient clinics. The authors reviewed the charts of any patient older than 18 years who had one or more ED visits for syncope from 2002 to 2005. Emergency department visits within and external to the health system were included. Syncope was identified by *International Classification of Disease*, *Ninth Edition* (*ICD-9*) code 780.2 on the patient's chart. Identification of other comorbidities and arrhythmias was also done solely by *ICD-9* code extraction.

The primary outcome for this study was 7-day cardiac outcomes occurring after an ED visit for syncope. Cardiac outcomes included cardiac death, hospitalization or procedures for arrhythmia, ischemic heart disease, and valvular heart disease. Mortality and cause of death data were identified through linked California vital statistics files. Over the 4-year observation period, the authors identified 35 330 patients who accounted for 39 943 ED visits for syncope. There were 893 (2.5%) 7-day cardiac outcomes. The most common cardiac outcome was an arrhythmia (63%), followed by ischemic heart disease (26%), cardiac death (15%), and valvular heart disease (3%).

The authors found that the risk of an adverse outcome was positively associated with age older than 60 years (OR, 3.8; 95% CI, 2.0-5.0), male sex (OR, 1.5; 95% CI, 1.3-1.7), congestive heart failure (OR, 2.0; 95% CI, 1.1-3.5), and ischemic heart disease (OR, 3.7; 95% CI, 2.1-6.5). They also found that younger patients (18-60 years) with an arrhythmia or valvular heart disease were at increased risk for an adverse outcome when compared with patients older than 60 years. The authors also found that dementia, pacemaker, coronary revascularization, and cerebrovascular disease were associated with a decreased risk of adverse outcome.

This study is limited by the fact that it is a retrospective chart review and that the authors relied on how the patient's charts were coded with *ICD-9* codes. Unfortunately, this study does not offer any groundbreaking information that will improve outcomes in the ED, although it reinforces what we already know. Patients with underlying cardiac disease are at increased risk for an adverse cardiac outcome after they have syncope. However, it is interesting that the authors noted a higher rate of adverse outcomes in younger patients with underlying cardiac disease and that a history of coronary revascularization, dementia, and cerebrovascular disease is "protective."

C. Reed MJ, Newby DE, Coull AJ, et al. The ROSE (risk stratification of syncope in the emergency department) study. J Am Coll Cardiol 2010;55:713-721.

D. Benditt DG, Can I. Initial evaluation of "syncope and collapse" the need for a risk stratification consensus. J Am Coll Cardiol 2010;55:722-724.

The SFSR attempted to predict risk of serious outcomes in patients who had syncope by considering easily obtained historical elements and diagnostic tests; external validation studies, however, have not shown promise in this CDR. With the failings of the SFSR, the authors of this study attempted to validate a new CDR (risk stratification of syncope [ROSE]) rule to predict 1-month serious outcomes and all-cause death in patients presenting with syncope to the ED. The study was also designed to assess whether biochemical markers (ie, B-type natriuretic peptide [BNP]) would be useful in determining risk.

The authors conducted a single-center, prospective, observational derivation and validation cohort study at the Royal Infirmary of Edinburgh in Scotland. They reviewed the charts of all patients older than 16 years who presented with syncope. Patients were excluded if they could not provide consent; had a persistent neurologic deficit suggestive of stroke; had been previously recruited into the study; had collapse due to alcohol consumption, hypoglycemia, or trauma; or had seizure activity with a greater than 15-minute witness-reported postictal phase. Serious outcomes that were measured were life-threatening arrhythmia (VF, sustained VT, ventricular pause greater than 3 seconds, ventricular standstill, or asystole), AMI, implantation of a pacemaker or defibrillator within 1 month of index collapse, pulmonary embolus (confirmed by diagnostic study), cerebrovascular injury, intracranial hemorrhage, subarachnoid hemorrhage, hemorrhage requiring a blood transfusion greater than 2 U, or an acute surgical or endoscopic procedure.

The authors conducted their derivation trial in 2007 for 8 consecutive months, enrolling 550 patients. Twenty-one patients were excluded (2) or lost to follow-up (19); 529 patients were analyzed. Multiple logistic regression analysis identified the following independent predictors of a serious outcome: BNP concentration greater than 300 pg/mL (OR, 7.3), a rectal examination showing occult blood (OR, 13.6), hemoglobin level less than 9 g/dL (OR, 6.7), Q waves present in any lead on ECG except for lead III (OR, 2.8), left bundle branch block (4.8), male sex (OR, 2.6), oxygen saturation less than 94% on room air (OR, 3.0), albumin less than 3.7 g/dL (OR, 3.2), and white blood count greater than 14×10^9 cells per liter (OR, 2.4). Further analysis showed that chest pain with syncope or bradycardia (heart rate less than 50 beats per minute) identified another 5 patients. For the validation study, the authors removed male sex, white blood count, and albumin. The final derived ROSE rule indicated that patients with any of the following should be admitted to the hospital:

- BNP greater than 300 pg/mL
- Bradycardia less than 50 beats per minute in the ED or prehospital
- Rectal examination showing fecal occult blood (note that the authors do not recommend a rectal examination on all syncope patients but only on those that have a suspected gastrointestinal bleed; in this study, they performed a rectal examination on 13% of their patients)
- Anemia—hemoglobin level less than 9 g/dL
- Chest pain associated with syncope
- ECG showing Q wave (not in lead III) and/or
- Saturation ≤94% on room air.

For the derivation trial, the ROSE rule, which can be remembered with the acronym BRACES, had a sensitivity of 92.5%, specificity of 73.8%, and positive and negative predictive values of 22.4% and 99.2%, respectively. In the derivation cohort, the ROSE rule would have missed 3 patients compared with 5 patients with serious outcomes that were discharged from the ED (4 of whom would be identified by the ROSE rule).

The validation study was completed in another 8-month period from 2007 to 2008. The authors enrolled 550 patients, but 10 were lost to follow-up, and one was previously enrolled, leaving 538 for analysis. Thirty-nine patients in this cohort had a primary outcome. The calculated sensitivity, specificity, and positive and negative predictive values for this cohort were 87.2%, 65.5%, 16.5%, and 98.5%, respectfully. Five patients were missed who had the following serious outcomes: myocardial infarction, subarachnoid hemorrhage, basal ganglia hemorrhage on day 29, VT in the ED, and a gastric ulcer found on endoscopy that did not undergo a rectal examination in the ED. Two patients who had an intracranial hemorrhage, basal ganglia hemorrhage, and subarachnoid hemorrhage were discharged from the ED and would not have been identified by the ROSE rule. One additional patient who was discharged would have been identified. The authors estimate that implementation of the

ROSE rule would identify 85% of the patients who have a subsequent serious outcome or death that may not be readily apparent in the ED and that the rule would decrease the rate of admissions by 30%.

Although the authors state that BNP is an excellent predictor of serious outcomes, it was only elevated in 40 (7%) of the patients in the validation cohort. The mean age of those with a raised BNP was 82 ± 8 years, and these patients often had a history of hypertension, ischemic heart disease, previous myocardial infarction, known cardiac failure, or signs of cardiac failure on their clinical examination. As alluded to in the editorial that accompanied this article, could historical elements equally predict a patient's increased risk of a serious outcome without the need for another laboratory test? Unless externally validated to show that it is beneficial in younger patients, especially those without any significant cardiac history, routine BNP levels are unlikely to help diagnostically and will only increase the cost of care.

Although the authors predict that they could decrease admissions by 30%, the ROSE rule cannot be recommended for routine use. The validation trial missed 15% of serious outcomes, and if the SFSR is a predictor of this decision rule, one might predict that external validation trials will show an even higher miss rate. It is also difficult to justify the additional cost of routine BNP testing in younger patients without any history of cardiac disease.

E. Serrano LA, Hess EP, Bellolio MF, et al. Accuracy and quality of clinical decision rules for syncope in the emergency department: a systematic review and meta-analysis. Ann Emerg Med 2010;56:362-373 e361.

The authors of this article conducted a systematic review and meta-analysis of syncope CDRs to assess their quality and overall accuracy in identifying syncope patients in the ED that are at risk for an adverse event. The authors conducted a search of the medical literature using 6 electronic databases to identify prospective or retrospective derived or validation CDR or risk score studies that predict subsequent adverse events in patients with syncope. Articles were included if they enrolled patients who presented with a compliant of syncope or near-syncope to an ED; were based on original research; and used 3 or more variables from the history, physical examination, or basic diagnostic tests. Studies were not excluded based on their timing of the adverse outcome assessment.

Of the 388 records that were identified by the electronic search, only 18 studies meet the inclusion criteria, which represented 9 different CDRs. The authors were only able to obtain sufficient data for a quantitative analysis for 12 studies that represented 5 different CDRs. These CDRs were the Boston Syncope Rule (1 study), the Syncope Risk Score (1 study), the ROSE (1 study), the Osservatorio Epidemiologico sulla Sincope nel Lazio (OESIL) risk score (3 studies), and the SFSR (9 studies). Two studies provided quantitative information for more than one CDR.

The meta-analysis showed that the SFSR had an overall sensitivity of 86% (95% CI, 83%-89%) and specificity of 49% (95% CI, 48%-51%), whereas the OESIL rule had an overall sensitivity of 95% (95% CI, 88%-98%) and specificity of 31% (95% CI, 29%-34%). The variability seen in the SFSR rule was felt to be due to difference in study design and ECG determination. Prospective studies had a 4-fold higher diagnostic OR over a retrospective study, and ECG determination made by the treating physician had a diagnostic OR of 25.5 (95% CI, 4.41-148) vs a diagnostic OR of 4 (95% CI, 2.15-7.55) if made by a researcher or cardiologist. The higher diagnostic OR for the treating physician making the ECG determination is probably because the treating physician is interpreting the ECG in context of the patient's clinical condition.

In the end, this meta-analysis highlights that the current CDRs for syncope are limited and should not be relied on solely in determining a patient's risk for an adverse outcome. The 98% sensitivity of the OESIL rule is excellent, although its specificity of 31% results in a lot of patients being admitted who will not have an adverse event. The SFSR's lower sensitivity increases the risk of missing patients who will have an adverse event, and its low specificity has the potential to increase admissions as seen in the study by Thiruganasambandamoorthy et al [58].

The approach to a patient with syncope should consist of a comprehensive history and physical examination specifically looking for risk factors for gastrointestinal bleeding, cardiac disease, or intracerebral bleeding. The physical examination should include postural blood pressure measurements along with a 12-lead ECG. Any additional laboratory tests should be obtained as needed and as indicated by historical or physical examination findings. A blanket policy that all patients with syncope require any specific laboratory tests results in unnecessary testing that is often low yield and increases the cost of care. The CDRs do highlight risk factors for an adverse outcome, and individuals with these risk factors should have a more thorough evaluation while in the ED. Ultimately, the current syncope CDRs cannot replace sound clinical judgment.

6. Pericarditis

A. Imazio M, Spodick DH, Brucato A, et al. Controversial issues in the management of pericardial diseases. Circulation 2010;121:916-928.

Acute pericarditis is a condition that is considered frequently in the ED. Although the diagnosis is not necessarily confirmed everyday, it must be included in the differential diagnosis of any patient with chest pain and in any ECG that demonstrates STE. The authors of this publication have surveyed the literature to address some of the areas of controversy and confusion regarding management of patients with pericardial disease. Regarding etiology, infectious (mostly viral) and idiopathic represent approximately two thirds of all cases of pericarditis seen in the United States. The major specific causes that need to be considered and ruled out (not only during the ED stay but also during hospitalization) are tuberculous (TB) pericarditis, neoplastic pericarditis, and pericarditis associated with a systemic disease. Importantly, TB pericarditis has a high incidence in sub-Saharan Africa (ie, 70%-80%) and in association with HIV infection (greater than 90%); it may also be found in developed countries, especially among immigrants from areas with a high prevalence of TB and HIV infection.

The basic diagnostic evaluation in the ED should include a focused history and physical examination, 12-lead ECG, select laboratory blood tests (serum chemistry studies, C-reactive protein, erythrocyte sedimentation rate, and troponin), and chest radiograph. A transthoracic echocardiogram should be considered in all such patients, particularly those with significant or ongoing hemodynamic compromise. Importantly, the authors do not indicate specifically if the echocardiogram is an emergent test. Pericardial effusion is a common finding on echocardiography; approximately 90% of cases of moderate-to-large effusions are associated with a specific cause, including neoplasms, TB, and myxedema.

Regarding admission decisions, no specific decision tool or risk scoring model has been proposed to identify patients requiring admission. Five factors have been validated as predicting a poor prognosis: fever greater than 38°C, subacute onset of the syndrome, large pericardial effusion, cardiac tamponade, and lack of response to aspirin or nonsteroidal anti-inflammatory drugs (NSAID) after at least 1 week of therapy. Four additional factors can be predictive of poor outcome, although they have not yet been validated: myopericarditis, immunosuppression, trauma, and oral anticoagulant therapy. Patients with any of these factors should be admitted for further management and evaluation. Importantly, the absence of these factors does not equate with stability for discharge.

The authors review the concept of *myopericarditis*. The widespread STE that is normally attributed to acute pericarditis is, in reality, due to concomitant superficial epicardial involvement; note that the pericardium is actually considered electrocardiographically silent. Myocardial involvement will often produce troponin elevations. Myocarditis and pericarditis share similar etiologic agents, and the 2 entities often coexist with varying degrees of myocardial and pericardial involvement. Cases where pericarditis predominates are often referred to as myopericarditis, cases where myocarditis predominates are often referred to as perimyocarditis, and at both ends of the spectrum exist cases of "pure" pericarditis and "pure" myocarditis. Myopericarditis is generally associated with fairly preserved left ventricular function and has a good prognosis without progression to heart failure, recurrence, and other.

Regarding therapy, aspirin or NSAIDs are the mainstay of treatment of acute pericarditis. Treatment failure is often because of using dosages that are too low or using treatment courses that are too short. Examples of recommended starting dosages are aspirin 2 to 4 g daily, indomethacin 75 to 150 mg daily, or ibuprofen 1600 to 3200 mg daily; treatment should continue until CRP or erythrocyte sedimentation rate normalizes. Aspirin is the preferred agent in patients with atherosclerotic heart disease. Colchicine is recommended as adjunctive treatment of recurrent pericarditis (in preference to steroids); it also is considered useful in acute pericarditis as well. Colchicine use is associated with a greater than 50% reduction in recurrence rate and a marked decrease in symptoms at 72 hours. The typical dose is 0.6 mg twice a day for 3 to 6 months, and in cases of severe recurrences, the duration may be extended to 12 to 24 months. Colchicine is best used as adjunctive treatment (in addition to aspirin or NSAIDs) rather than as monotherapy. Corticosteroids are recognized as a risk factor for pericarditis recurrence, probably because of impaired viral clearance, and they should generally not be initiated in the ED.

New data continue to accumulate that assists the emergency physician in the management of patients with pericardial disease. The emergency physician should be familiar with presentations in which patients can be safely discharged home, the conditions under which patients *must* be admitted for a workup, and the proper acute treatment modalities for these patients. This comprehensive review will assist with these important areas of management.

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