Emergency Cardiology 2010: The Articles You've Gotta Know!

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Cardiac Arrest

Olasveengen TM, Sunde K, Brunborg C, et al. Intravenous drug administration during out-of-hospital cardiac arrest. JAMA 2009;302:2222-2229.

Despite the traditional use of intravenous medications such as vasopressors and antiarrhythmics for victims of cardiac arrest, there is actually very little evidence to support these therapies. On the contrary, recent multicenter center stuies demonstrated that the use of intravenous medications that are advocated in standard advanced cardiac life support (ACLS) guidelines was ineffective at improving survival of patients with out-of-hospital cardiac arrest. Olasveengen and colleagues now add further support to the contention that the use of intravenous medications in victims of non-traumatic cardiac arrest is not associated with improvements in meaningful outcomes.

The authors performed a prospective randomized trial of consecutive adults with non-traumatic cardiac arrest that were treated within their emergency medical services (EMS) system in Oslo between 2003-2008. Patients were randomized to either receive standard ACLS therapies with intravenous drug administration (IV group) or ACLS therapies without any intravenous drugs (no IV group). A total of 851 patients were included in the study, 418 patients in the IV group and 433 in the no IV group.

The researchers found there was an increase in survival to hospital admission with return of spontaneous circulation in the IV group vs. the no IV group (32% vs. 21%, P < 0.001). However, there was no difference between the IV group vs. the no IV group in terms of survival to hospital discharge (10.5% vs. 9.2%, P = 0.61), survival with favorable neurological outcome (9.8% vs. 8.1%, P = 0.45), or survival at 1 year (10% vs. 8%, P = 0.53). The results demonstrate that with the use of IV ACLS medications, patients simply die in the hospital rather than in the ED. Practically speaking, this amounts to increased intensive care unit bed utilization, hospital resource utilization, and expenses; but without any increase in meaningful survival. In this era of ED and hospital overcrowding and the increasing demand for cost-effectiveness in medical therapies, Stiell's and Olasveengen's studies should force us to consider that the use of IV medications for patients in cardiac arrest should be the exception rather than the rule…or guideline.

Recent Advances in Cardiopulmonary Resuscitation: Cardiocerebral Resuscitation
Ewy GA, Kern KB. J Am Coll Cardiol 2009;53:149-157.

If you ask many older physicians in emergency medicine what "CCR" stands for, they might talk to you about a rock band that was popular in the 1960s and 1970s that still gets some play on the "oldies" radio stations. However, emergency physicians that have a particular interest in prehospital care or cardiac arrest know that "CCR" now also represents the most important advance in care of cardiac arrest patients since CPR was first described: cardiocerebral resuscitation. Some people refer to *this* CCR as the new form of CPR, while others refer to this simply as the *replacement* for traditional CPR. Either way, cardiocerebral resuscitation is something that everyone practicing emergency medical care — emergency medical technicians, ED nurses, emergency physicians, cardiologists, and intensive care physicians — needs to know.

Sudden cardiac death (SCD) is one of the leading causes of mortality in the U.S. and other developed nations. Unfortunately, survival rates from SCD are abysmal despite the promulgation of lay-person and first-responder CPR courses as well as Advanced Cardiac Life Support courses over the past few decades. Critics of traditional CPR have stated that the **emphasis on early ventilation (either mouth-to-mouth ventilaiton, bagging, or endotracheal intubation) is misguided for several reasons.** First, these airway interventions are well-known to take time away from performing adequate chest compressions at 100/minute. Second, these airway interventions all produced an increase in intrathoracic pressure, which decreases venous return and thus cardiac output…not a good thing in a patient in cardiac arrest. Third, the majority of patients with sudden cardiac arrest are not hypoxic at the time of onset of the arrest, and so the blood in the central circulation is not oxygen-deficient. Ventilations, therefore, are not immediately needed to improve oxygenation. While these statements are certainly not true for patients suffering a pulmonary arrest, the clear majority of patients with SCD are victims of ventricular fibrillation (cardiac) arrest rather than pulmonary arrest.

Recent literature has also **separated ventricular fibrillation arrest into time-sensitive phases**. During the first phase (the first 4-5 minutes), the heart is “amenable” to prompt defibrillation. This is termed the **“electrical phase”** of cardiac arrest. During the second phase, the **“circulatory phase,”** the “fibrillating myocardium has used up much of its energy stores” and chest compressions are necessary to perfuse, or “prime” the heart, before defibrillation will likely be successful. This second phase is usually the phase during which time prehospital care providers arrive.

Based on this information, researchers at the University of Arizona in 2003 broke from the traditional guidelines for CPR and created a new protocol for management of out-of-hospital cardiac arrest that they termed “Cardiocerebral Resuscitation.” CCR consists of three major parts: **(1) continuous chest compressions with no early ventilations pre- and post-shock; (2) delayed intubation; and (3) early use of epinephrine (EPI).** A recent study (3) demonstrated that when CCR was compared to standard CPR in patients with shockable rhythms, both survival (47.2% vs. 19.6%) and percentage of survivors with good neurological outcome (83.3% vs. 77.8%) were significantly improved.

Ewy and Kern, both leaders in the field of cardiac resuscitation, review CCR in this manuscript and take this one step beyond by describing ideal post-resuscitation care as well. They describe the three “pillars” of CCR, which are listed in the article and reproduced here:

1. Compression-only CPR by anyone who witnessed the event.
2. CCR by emergency medical service personnel, assumed to be arriving > 5 minutes post-arrest.
    a. 200 chest compressions (at 100/minute), delay intubation, second person applies defibrillation pads and initiates *passive* oxygen insufflation (e.g. 100% oxygen via facemask)

b. Single shock if indicated followed immediately by 200 more chest compressions (no pulse check after shock)
c. Check for pulse and rhythm; note that this pulse check occurs 4 minutes after the CCR has begun
d. EPI IV or IO as soon as possible in order to improve central circulation, coronary circulation, and diastolic blood pressure
e. Repeat (b) and (c) 3 times; intubate if no return of spontaneous circulation after 3 cycles; **note that neither bag-valve-mask ventilation nor intubation occurs until *10 minutes after the CCR has begun***
f. Continue resuscitation efforts with minimal interruptions of chest compressions until successful or pronounced dead

3. Post-resuscitation care to include mild hypothermia and urgent cardiac catheterization and percutaneous coronary intervention unless contraindicated

Ewy and Kern's "pillars" include post-resuscitation care…it certainly seems sensible to do this. Induced hypothermia has become fairly-well accepted, but they also add on the recommendation that patients should be treated with coronary angiography and **percutaneous intervention (PCI) regardless of the ECG.** They reference data (4) in support of PCI in conjunction with induced hypothermia for cardiac arrest patients, regardless of ECG findings, noting that in the study this **reperfusion therapy was "the most influential factor on survival, with an odds ratio of > 27."** If further studies support this recommendation, there will be a need to make profound changes in prehospital protocols whereby **all patients in cardiac arrest will be taken to medical centers with resources for immediate PCI should the patient survive.** Stay tuned…

In summary, the traditional mantra in emergency medicine of "A-B-C" has been turned upside-down by CCR. Aggressive management of the airway in victims of cardiac arrest is being relegated to a far lower priority. Good chest compressions and early EPI are the most important interventions when ventricular fibrillation is present in the "circulation phase" of cardiac arrest. Future studies will need to evaluate whether these concepts are applicable to non-shockable rhythms as well, though intuitively this seems reasonable. Finally, post-arrest survivors should be treated with induced hypothermia, and pending more studies they may benefit from early coronary angiography and PCI as well.

Garza AG, Gratton MC, Salomone JA, et al. Improved patients survival using a modified resuscitation protocol for out-of-hospital cardiac arrest. Circulation 2009;119:2597-2605.

Ewy GA. Do modifications of the American Heart Association Guidelines improve survival of patients with out-of-hospital cardiac arrest? Circulation 2009;2542-2544. (editorial accompanying above article)

Sudden cardiac death (SCD) is one of the leading causes of mortality in the U.S. and other developed nations. Unfortunately, survival rates from SCD are abysmal despite the promulgation of lay-person and first-responder CPR courses as well as ACLS courses over the past few decades. Critics of traditional CPR have stated that the emphasis on early ventilation (either mouth-to-mouth ventilation, bagging, or endotracheal intubation) is misguided for several reasons. First, these airway interventions are well-known to take time away from performing adequate chest compressions at 100/minute (see article by Wang, et al, below). Second, these airway interventions all produced an increase in intrathoracic pressure, which decreases venous return and thus cardiac output; and hyperventilation can also decrease cerebral perfusion…certainly harmful effects in cardiac arrest patients. Third, the majority of patients with sudden cardiac arrest are not hypoxic at the time of onset of the arrest, and so the blood in the central circulation is not oxygen-deficient. Ventilations, therefore, are not immediately needed to improve oxygenation. While these statements may not be true for patients suffering a pulmonary arrest, the clear majority of patients with SCD are victims of ventricular fibrillation (primary cardiac) arrest rather than pulmonary arrest.

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Garza and colleagues this year provide further evidence to support CCR as the optimal form of management for patients with cardiac arrest. They evaluated patients that were victims of witnessed ventricular fibrillation arrest that were presumed to be cardiac in cause (i.e., not victims of drowning, overdose, etc.). They compared 1097 patients that were treated under traditional ACLS protocols in the Kansas City, Missouri EMS system vs. 339 patients that received CCR, which was initiated in their system after 2005. Specifically, CCR in this study consisted of the following, to be instituted by EMS personnel:

1. 200 chest compressions (at 100/minute), at a 50:2 ratio of compressions with “gentle” ventilations,” second person applies defibrillation pads and initiates 100% oxygen via facemask; if “adequate” chest compressions had been instituted prior to paramedic arrival, the initial 200 compressions could be skipped
2. Single shock if indicated followed immediately by 200 more chest compressions, with 50:2 compression:ventilation ratio
3. Check for pulse and rhythm; repeat step (2) if still shockable rhythm and no pulse
4. EPI IV or IO every 3 minutes beginning after the second shock in order to improve central circulation, coronary circulation, and diastolic blood pressure
5. Repeat (2) and (3) 2 times; intubate if no return of spontaneous circulation after 3 cycles; note that intubation is not performed during the first 6 minutes; instead,

minimally-interrupted CPR with a 50:2 compression:ventilation ratio is performed during that time

6. Continue resuscitation efforts with minimal interruptions of chest compressions until successful or pronounced dead

It's notabale that the protocol employed by Garza included a 50:2 compression:ventilation ratio, whereas that recommended by Ewy and employed by Kellum incorporated nothing more than passive oxygenation (100% facemask oxygen without any bag-valve-mask ventilation) during the delay-time before intubation. In the accompanying editorial by Ewy he writes that he had emailed Garza questioning why they incorporated the 50:2 ratio for ventilations instead of simply passive oxygenation, Garza responded that "their group did not think the paramedics would accept not intubating and not ventilating their patients." Nevertheless, this slightly revised form of CCR resulted in an improvement in survival to discharge from the hospital from 22% to 44%, with 88% of survivors having good neurologic outcome.

The authors note that the typical limitations of a retrospective study. Another concern that was raised in Ewy's editorial is that CCR in this study was essentially compared with the 2000 and not with the 2005 guidelines. However, recent studies from Norway and King County, Washington comparing resuscitation rates using the 2005 guidelines vs. the 2000 guidelines showed non-significant improvements. Cardiocerebral resuscitation appears to be a significant advance in care of the patient with primary cardiac arrest.

Survival and Neurologic Recovery in Patients With ST-Segment Elevation Myocardial Infarction Resuscitated From Cardiac Arrest

Hosmane VR, Mustafa NG, Reddy VK, et al. J Am Coll Cardiol 2009;53:409-415.

Emergency Percutaneous Coronary Intervention in Patients With ST-Elevation Myocardial Infarction Complicated by Out-of-Hospital Cardiac Arrest: Early and Medium-Term Outcome

Lettieri C, Savonitto S, De Servi S, et al. Am Heart J 2009;157:569-575.

- Patients with evidence of STEMI before or after resuscitation from out-of-hospital cardiac arrest fare better with emergent catheterization and PCI, regardless of neurologic status after resuscitation.

Reynold JC, Callaway, CW, El Khoudary SR, et al. Coronary angiography predicts improved outcome following cardiac arrest: propensity-adjusted analysis. J Int Care Med 2009;24:179-186.

In recent decades, numerous therapies have been promoted as great advances in primary cardiac arrest: epinephrine, high-dose epinephrine, vasopressin, lidocaine, amiodarone, electrical defibrillation, biphasic defibrillators, induced hypothermia, and so on. The *initial* literature on these "miracle cures" has always looked promising. However, when attempts at validating the early studies are made or when meaningful outcomes — hospital discharge with good neurological function — are evaluated, most of these therapies have fallen short and eventually lost favor in the literature, as noted in the Olasveengen article discussed above. Currently it appears that only three therapies have emerged as truly beneficial in terms of meaningful outcomes: rapid defibrillation of VF/VT, good chest compressions (with less emphasis on early airway interventions and minimizing interruptions), and induced hypothermia.

Given that the majority of cases of primary cardiac arrest are associated with acute coronary syndromes, it would seem reasonable to assume that urgent coronary angiography and percutaneous coronary intervention (PCI) in patients with return of spontaneous circulation (ROSC) after cardiac arrest would be associated with improved outcomes. Recent studies have demonstrated this to be true for patients with ECG evidence of ST-segment elevation myocardial infarction (STEMI) either before or after resuscitation. However, it is well-known that the ECG is far from perfect at demonstrating evidence of acute MI, and so use of the ECG to determine which patients should benefit from urgent PCI might potentially lead to many patients missing out on a beneficial therapy. Can coronary angiography and PCI improve the outcomes of resuscitated patients *without* definite evidence of STEMI as well? This is the question that Reynolds, et al., attempted to answer.

The authors performed a chart review of resuscitated cardiac arrest patients between 2005-2007, evaluating an assortment of parameters including acute ischemic ECG changes (new left bundle branch block or STEMI), presenting rhythm, neurologic status, and outcome. A good outcome was defined as discharge home or to an acute rehabilitation facility. Of the 241 patients they reviewed, 40% received coronary angiography. Significant disease (defined as $\geq$ 70% stenosis in at least one coronary artery) was identified in 69% of patients including *57% of patients without any ischemic changes on ECG*. Of the patients that received coronary angiography and PCI, 54% experienced a good clinical outcome compared to 25% of patients not receiving coronary angiography. The authors used a propensity-adjusted analysis to account for the non-randomized nature of the study and determined that improved survival and good outcome were associated with coronary angiography regardless of the presence of new left bundle branch block or STEMI, and also regardless of presenting rhythm or neurologic status immediately after resuscitation.

Reynolds' study provides further support to previously published reports encouraging urgent catheterization for survivors of cardiac arrest regardless of ECG evidence of STEMI. Recent publications also show that therapeutic hypothermia can be safely employed in these patients during and after PCI as well without producing delays in time to balloon inflation, hemodynamic instability, malignant arrhythmias, or increases in bleeding complications.

The significance of this new literature cannot be overstated. If further studies confirm these findings, it would strongly argue for enormous changes in prehospital systems of care to recommend that all survivors of primary cardiac arrest should be immediately transported to hospitals that have the capability of performing urgent PCI in conjunction with therapeutic hypothermia. Based on the current literature, it certainly seems advisable that emergency health care practitioners that care for resuscitated victims of primary cardiac arrest should engage in conversations with cardiology consultants and urge them to take an aggressive approach to PCI in these patients.

Mild Therapeutic Hypothermia in Patients After Out-of-Hospital Cardiac Arrest Due to Acute ST-Segment Elevation Myocardial Infarction Undergoing Immediate Percutaneous Coronary Intervention
Wolfrum S, Pierau C, Radke PW, et al. Crit Care Med 2008;36:1780-1786.

- The combination of mild therapeutic hypothermia plus PCI is safe and effective in patients with STEMI who are resuscitated after cardiac arrest

Pericarditis vs. Acute MI, Electrocardiography

Salisbury AC, Olalla-Gomez C, Rihals CS, et al. Frequency and predictors of urgent coronary angiography in patients with acute pericarditis. Mayo Clin Proc 2009;84:11-15.

Bainey KR, Bhatt DL. Acute pericarditis: appendicitis of the heart? Mayo Clin Proc 2009;84:5-6. (editorial accompanying above article)

The distinction between acute pericarditis and acute myocardial infarction (AMI) is a difficult one. Clinically, both presentations cause chest pain that is often described as severe in nature, and "classic, textbook" descriptions (e.g., pleuritic, positional pain radiating to the trapezius ridge in pericarditis; substernal pressure radiating to the left jaw/neck/arm in AMI) of the conditions are often absent in real patients. Electrocardiogram (ECG) findings are often similar — both conditions are well-known to produce ST-segment elevation. The overall result is that many patients with acute pericarditis are mistakenly diagnosed as having ST-segment myocardial infarction (STEMI) and sent for primary percutaneous coronary intervention (PCI; i.e., angioplasty or stent placement). Larson et al recently reported that 18.7% of patients that were sent for primary PCI for presumed STEMI and found to have no culprit lesion were later diagnosed with pericarditis or myocarditis. Complicating matters further is the fact that anticoagulation or thrombolysis for presumed STEMI can be dangerous in patients with acute pericarditis[2] because these medications increase the chance that a pericardial effusion may develop into a hemorrhagic tamponade. During my career I know of two patients with acute pericarditis that developed pulseless electrical activity and died from hemorrhagic tamponade after they were treated for presumed STEMI with thrombolytics. Clearly, a greater understanding of both the overlapping as well as the distinguishing features of acute pericarditis vs. AMI is needed.

This study and the accompanying editorial together present important information that helps us compare and contrast acute pericarditis and AMI. Salisbury et al set out to determine the frequency of urgent PCI in patients with acute pericarditis; in other words, to evaluate patients with pericarditis that were mistakenly diagnosed with AMI and sent for urgent PCI. They also evaluated the clinical characteristics of those patients that were referred for PCI. They retrospectively identified 238 patients between January 2000 and December 2006 with a *final* diagnosis of acute pericarditis, 40 (16.8%) of who were referred for PCI. Of the total group, 146 patients (61.3%) had ST-segment elevation (STE) and 92 (38.7%) did not. Not surprisingly, PCI was performed in many more of the STE group (24.7%) than in the non-STE group (4.3%). Statistical analysis revealed that the factors associated with a higher likelihood for urgent PCI were typical anginal-type description of chest pain, STE on ECG, history of previous PCI, elevated troponin (TN), diaphoresis, and male sex.

In comparing the groups that had STE vs. no STE, the researchers revealed some interesting results, many of which are highlighted in the accompanying editorial.

1. In terms of clinical features, 49.6% of patients reported that their pain was positional, and 68.9% reported that their pain was pleuritic. These numbers are clearly less than what we are commonly taught. Complicating matters further, 12.2% of patients complained of pain that was consistent with typical angina.
2. There appear to be significant gender differences with acute pericarditis. Of the total number of patients there were almost twice as many men diagnosed with pericarditis than women (157 vs. 81). Men constituted 81.5% of the patients with STE, whereas women accounted for 58.7% of the non-STE group. Looking at the numbers another way, men diagnosed with pericarditis demonstrated STE in 76% of cases, whereas women diagnosed with pericarditis demonstrated STE in only 33% of cases. One must wonder if there is a physiological reason why women are less likely to develop STE in acute pericarditis.
3. Patients in the STE group were less likely than patients in the non-STE group to report a recent history of viral illness (32.2% vs. 46.7%) and less likely to have a friction rub (12.3% vs. 27.2%). For both of these factors, notice how uncommon these "classic" features are.
4. Elevated TN levels were found in 15.8% of the STE group and 7.6% of the non-STE group (12.6% of patients overall). This difference was not statistically significant, but it is important to be aware that acute pericarditis is often associated with elevated TN levels, further complicating diagnostic certainty. Other studies have actually demonstrated even higher rates of positive TN at admission, including one that reported a 23.7% incidence of positive TN levels at the time of admission.[3]
5. Pericardial effusion was common: 38.1% of patients in the STE group and 73.5% of the patients in the non-STE group had effusions. This difference between the STE and the non-STE group may not be a huge surprise when one considers that larger effusions tend to decrease voltage, including the magnitude of STE. Although the authors didn't comment on the size of the effusions, the editorial discusses a study in which 60% of patients with acute pericarditis presented with effusions, with 80% being mild, 10% being moderate, and 10% being severe in size.[4]
6. What about that classic "pulsus paradoxus" (PP) that the inpatient physicians always ask us about? The researchers noted a positive PP in only one of the 146 patients in the STE group and in only 3 of the 92 patients in the non-STE group (and remember that a LOT of the patients in this latter group did have pericardial effusions).

The researchers remind us of the limitations of a retrospective study, not the least of which is that the "definition of acute pericarditis relied heavily on the opinion and diagnosis of the treating physician." Nevertheless, this study serves as an excellent reminder to all of us that (1) acute pericarditis must remain in the differential diagnosis for any patient with chest pain, with or without STE; (2) neither patients with STEMI nor patients with acute pericarditis can be relied upon to present "classically;" and (3) given the frequency with which patients with acute pericarditis present with pericardial effusions, the use of bedside ultrasound in *any* patient with chest pain may be enormously helpful. Finally, just as there is an appropriate "false-laparotomy rate" for patients with presumed appendicitis, it may be appropriate to accept a "false-catheterization rate" for patients with acute pericarditis, even despite our best efforts and knowledge.

Jayroe JB, Spodick DH, Nikus K, et al. Differentiating ST elevation myocardial infarction and nonischemic causes of ST elevation by analyzing the presenting electrocardiogram. Am J Cardiol 2009;103:301-306.

In recent years emergency physicians have borne the brunt of criticism regarding ECG misinterpretation in patients with ACS and the patient outcomes related to these miscues (1-3). However, ECG interpretation is by no means straightforward in all cases. ST-segment elevation (STE), for example, can be associated with acute myocardial infarction (AMI) but it can also be associated with other conditions in which routine treatment for AMI can be harmful or at the very least unnecessary. Some of those conditions include acute myopericarditis, benign early repolarization, ventricular aneurysm, left bundle branch block, Brugada syndrome, and hyperkalemia to name just a few. In contrast to emergency physicians, cardiologists are often considered a local gold-standard at most hospitals for ECG interpretation, but how reliable are they in interpreting ECGs with STE? Jayroe and colleagues studied just that question.

Fifteen experienced cardiologists from North America (7), Europe (7), and Israel (1) were asked to analyze 116 consecutive ECGs demonstrating STE ≥ 1 mm at the J-point in ≥ 2 contiguous leads. The ECGs were obtained from patients ≥ 18 years of age in various medical settings, including the ED, intensive care units and other inpatient beds, as well as outpatient settings. Patients were excluded if they were within 1 month of cardiothoracic surgery or if they had a bundle branch block, ventricular rhythm, or paced rhythm. The cardiologists had no direct contact with the patients, simulating the conditions of wireless transmission. They were simply told that the patients had "compatible symptoms." They were then asked to determine if they would send the patient for primary PCI for suspected STE AMI (STEMI). If the cardiologist did not believe that PCI was indicated, he was asked to determine a reason why (e.g. identify an alternative diagnosis). The patient charts, subsequent ECGs, biomarkers, and in some cases angiograms and echocardiograms were assessed to determine which of the patients had true STEMI.

Of the 116 patients, only 8 patients (7%) had true STEMI, all of whom underwent PCI. There were large variations amongst the cardiologists as to how many of the ECGs were judged suitable for primary PCI (7.8% to 33%). The sensitivities amongst the cardiologists for detecting the true STEMIs ranged from 50% to 100% (average 75%), and the specificities ranged from 71% to 97% (average 85%). Note that on average, the cardiologists missed 25% of the true STEMIs. There were no significant differences between the North American cardiologists vs. those from other countries in any of these measures. Additionally, there was no consistency in terms of the reasons why the cardiologists misdiagnosed the STEMIs as non-STEMIs.

The authors, many of who are well-known cardiologists, expressed in the discussion section that the findings were very unexpected: "We hypothesized that these electrocardiographers would be both highly accurate and show consistency among individual readers. We found neither to be true." They further state that, "If experienced readers, using the current criteria and guidelines, cannot accurately and consistently distinguish between STEMI and [non-STEMI], less-experienced readers cannot be expected to do so." Although it's debatable whether emergency physicians should fall into that category of "less-experienced readers," this study certainly does lend support to emergency physicians that are criticized by cardiologists for over- or under-diagnosing STEMI. More importantly, this study should serve as a reminder that cardiologists are not a reliable gold-standard for ECG interpretation and that the onus is on us emergency physicians to commit ourselves to improving our own ECG interpretation skills so that we don't need to rely on any other specialists to provide the interpretations.

New LBBB and AMI

Chang AM, Shofer FS, Tabas JA, et al. Lack of association between left bundle-branch block and acute myocardial infarction in symptomatic ED patients. Am J Emerg Med 2009;27:916-921.

The traditional teaching and practice guideline for emergency physicians caring for patients with chest pain is that if a patient's ECG demonstrates a new or presumed-new left bundle branch block (LBBB) pattern, then that patient should be treated similarly to a patient with ST-segment elevation myocardial infarction (STEMI). Therefore, that patient is a candidate for acute reperfusion therapy (either IV fibrinolytics or immediate percutaneous coronary intervention, PCI). However, skeptics of the current guidelines suggests that many of these patients actually rule-out for acute myocardial infarction (AMI) and have no evidence of acute coronary occlusions during cardiac catheterization. "Inappropriate" reperfusion therapy has significant drawbacks, including major bleeding risks from fibrinolytics, anticoagulants, and anti-platelet medications; potential complications related to cardiac catheterization; and increased costs. We certainly don't want to miss opportunities to treat patients with acute coronary syndromes aggressively, but also must remember the dangers of unnecessary treatment as well.

The authors of this study sought to determine whether patients with ischemic-type symptoms presenting with new or presumed-new LBBB do, in fact, have a higher likelihood of AMI. They evaluated 7937 patients > 30 years of age (avg. age 54 years of age) presenting with either chest pain or other potential ischemic symptoms. The majority (7746) had no ECG evidence of LBBB, but 55 (0.6%) had a new or presumed new LBBB and 136 (1.7%) had LBBB that was known to be old. The authors did not address whether any patients demonstrated the Sgarbossa criteria, which have been previously described as highly specific for AMI in the presence of LBBB. Thirty-day follow-up was obtained on the patients and the main outcome was AMI. Secondary outcomes included revascularization (defined as PCI or coronary artery bypass grafting) within 30 days and presence of coronary artery disease (defined as at least one vessel with > 70% stenosis or documented AMI).

The investigators found no significant difference in the rate of AMI between the three groups (7.3% for patients with new/presumed-new LBBB, 5.2% for patients with old LBBB, 6.1% of patients with no LBBB; P = 0.75). There was, however, a higher rate of revascularization within 30 days in patients with new/presumed-new LBBB (7.8%) vs.

patients with old LBBB (5.2%) and patients without LBBB (4.3%) (P = 0.04). Documented coronary artery disease was also more common in patients with new/presumed-new LBBB vs. the other two groups (19.2% vs. 11.9% vs. 10.1%, P = 0.0004).

What should we take away from this study? First, based on the more frequent need for revascularization and the greater presence of coronary artery disease in patients with new/presumed-new LBBB, it certainly does seem reasonable to take a more aggressive approach to treating these patients when they are admitted to the hospital. However, given that these patients do not appear to be at a higher risk for AMI on first presentation, the more aggressive approach to these patients should not extend to the use of acute reperfusion therapies in the ED. The use of potent anticoagulant, anti-platelet, and especially fibrinolytic medications in the ED would appear to pose greater harm than benefit. Emergent activation of the catheterization lab, while safer than fibrinolysis, would also appear to be unjustified and pose greater costs than benefits if done purely on the basis of finding a new or presumed-new LBBB. Non-emergent PCI would appear to be the better choice in patients with concerning symptoms that are relieved with standard anti-anginal medications (e.g. nitroglycerin). Emergent PCI may be best reserved for these patients if they demonstrate Sgarbossa criteria on their ECG or for intractable ischemic symptoms (as we would do with any other patients) or patients that demonstrate evolving ECG changes. In other words, the study suggests that we should treat the presumed-*new* LBBB as a presumed-*old* LBBB in the ED.

Syncope in the Elderly

Mendu ML, McAvay G, Lampert R, et al. Yield of diagnostic tests in evaluating syncopal episodes in older patients. Arch Intern Med 2009;169:1299-1305.

Syncope is a common presenting complaint amongst ED patients. The differential diagnosis for this complaint is large, and as a result physicians tend to be liberal in ordering tests. A recent Clinical Policy from the American College of Emergency Physicians (1) provided rational guidelines regarding reasonable use of laboratory testing and ED imaging in most patients. However, most physicians still tend to order many tests during the workup of elderly patients presenting with syncope.

The authors attempted to evaluate the yield (i.e., whether the tests helped establish the etiology of the syncopal episode or affected diagnosis or management) of many of these tests that are routinely ordered in elderly patients. They reviewed 2106 consecutive patients $\geq$ 65 years of age who were admitted following a syncopal episode.

The authors found that the most frequently ordered tests were ECG (99% of admissions), telemetry (95%), and cardiac enzymes (95%). The majority of ECGs and cases of telemetry monitoring revealed minor abnormalities; telemetry helped determine an etiology in only 5% of cases. Only 5% of patients had positive cardiac enzymes, defined as any elevation of troponin I. The most frequently abnormal test was the echocardiogram ordered in 39% of cases and abnormal in 63% of these, but the majority were mild abnormalities and only 2% of echocardiograms revealed findings (e.g., aortic stenosis) that were thought to contribute to the syncope. Postural blood pressure (BP) was recorded in 38% of patients and was considered positive if the systolic BP dropped by 20 mm Hg or the diastolic BP dropped by 10 mm Hg. Postural BP had the highest yield in terms of affecting the presumed diagnosis (18%) and management (25%). However, significant opportunity for bias exists — the presence of the abnormal test may have led the clinicians to assume that that was the cause of the patient's syncope. Therefore, it's difficult to truly know the yield of postural BP testing.

The tests with the lowest likelihood of affecting diagnosis or management decisions were head CT, carotid ultrasound, electroencephalogram (EEG), and cardiac enzyme testing. In 8 out of the 9 admissions in which cardiac enzymes were thought to be helpful, the ECG was also noted to be abnormal. Head CT, ordered in 63% of cases, affected diagnosis or management in only 2%. In 25/28 of these cases, neurologic disease was suspected on clinical grounds already. The authors also evaluated costs per test affecting diagnosis or management. The EEG cost $32,973 for every test ordered that affected management. Other expensive tests per diagnosis or management were head CT ($24,881) and cardiac enzyme tests ($22,397). Postural BP recording had the lowest cost ($17-20), with the limitation of this test noted above. Telemetry monitoring ($710) and ECG testing ($1020) also had relatively low cost per test affecting diagnosis or management.

This study provides further evidence that the workup for syncope, even in elderly patients, can be very focused and should be based on a good history and physical exam. The routine use of laboratory testing and radiographic imaging is rarely helpful. Postural BP testing, telemetry monitoring, and ECG testing appear to be relatively cost-effective.