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Emergency Cardiology: A Review of Recent Literature

By Amal Mattu, MD

Introduction

At the University of Maryland Emergency Medicine Residency, members of the emergency medicine faculty each review a different organ system so that the department stays familiar with current literature. I enjoy emergency cardiology, so that is the topic I review. The articles for this review come from a database of more than two dozen American and European cardiology, emergency medicine, and internal medicine journals examined weekly. In addition, with other faculty members, we use several abstract services to ensure that we do not miss important articles from other journals. Altogether, we review several hundred articles, editorials, and reviews each month, which are selected because they reflect some important change in the standard of care. For the most part, these are all original research articles. Two final criteria are relevance to emergency medicine and conclusions that are supported by the data.

I present here my most recent review, with topics grouped by common themes: acute coronary syndrome (ACS), congestive heart failure (CHF), syncope, and resuscitation.

Acute Coronary Syndrome

Risk Factors

Prognostic value of elevated biomarkers in diabetic and non-diabetic patients admitted for acute coronary syndromes[1]

Of course, if a patient has chest pain and diabetes, we take it very seriously, but there is a new understanding in the United States and Europe regarding the significance of diabetes. Diabetes, previously considered a risk factor for coronary atherosclerosis, is now considered an equivalent of coronary atherosclerotic disease; the diabetic patient should be assumed to have coronary artery disease.

Fazel et al[1] studied 1951 patients who presented with ACS: 31% had diabetes, and of those, 71% had elevated levels of cardiac biomarkers. Diabetic patients were less likely to present with ST-segment elevation and more likely to have myocardial infarctions (MIs) with non-ST-segment elevation. Prior evidence indicates that some oral hypoglycemic medications attenuate the magnitude of ST-segment elevation during MI.[2] A diabetic patient may have an ST-segment elevation of only 0.5 mm, in contrast to an elevation of 1 mm to 2 mm in patients who do not take oral hypoglycemics.

In addition, these diabetic patients were much more likely to have congestive heart failure (CHF), pulmonary edema, and renal failure while hospitalized. In-hospital mortality for diabetic patients was 90% greater than in nondiabetic patients (7.3% versus 4.0%), and six-month mortality in diabetic patients without elevated biomarkers (cardiac enzymes) was the same as or worse than that for nondiabetic patients with positive troponin test results. With diabetes now considered so significant, at the University of Maryland Medical Center we generally admit these patients to a higher level of care than for nondiabetic patients with similar presentations.

Presentation

Meta-analysis of possible external triggers of acute myocardial infarction[3]

Culic et al[3] reviewed 17 different studies involving a total of 10,000 patients who presented with ACS. Patients were asked what they were doing when their chest pain began. The majority of these patients were physically active—6.1% had been engaged in heavy physical activity and 29% in mild to moderate activity. However, 21% of the patients had been awakened from sleep by pain, and almost 7% were involved in some emotionally stressful situation when pain began. These data demonstrate the importance of avoiding the tendency to label "stress" as a benign entity—it can precipitate a heart attack.

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were eating something when their cardiac pain began. Reflux esophagitis is the most common misdiagnosis of MI, as indicated by malpractice cases against emergency medicine specialists, cardiologists, and internists. According to the cardiology literature, 20% of patients with MIs and unstable angina describe their pain as indigestion. Furthermore, 15% of patients with MI get some pain relief and 7% get complete pain relief with antacids. Never employ Maalox use as a diagnostic modality to distinguish between reflux and MI.

… acute gastroesophageal reflux may actually provoke or worsen cardiac ischemia.

The literature also indicates that almost 50% of patients with MIs report that they had an increase in belching with their cardiac pain and that gastroesophageal reflux is more common in patients with cardiac disease than in the general population. In a separate study by Dobrzycki et al,[4] 50 patients with angiographically proven cardiac disease underwent simultaneous 24-hour electrocardiography and 24-hour esophageal pH monitoring to correlate episodes of acute reflux with cardiac ischemia. Twenty-one percent of episodes of electrocardiographically proven ischemia—ST-segment depression or ST-segment elevation—were associated with acute episodes of reflux. Patients with gastroesophageal reflux had more frequent episodes of ST-segment depression, and when these patients had acute reflux, their ST segments stayed depressed for a longer period of time than did ST segments for patients without gastroesophageal reflux. Patients with gastroesophageal reflux had more frequent episodes of ischemia, worse ischemia, and more prolonged episodes of ischemia than did patients without it. The authors then treated all of the patients with proton pump inhibitors and found that not only pH levels but also the magnitude of ST-segment depression improved in all patients. The important lesson from this study and literature as far back as the 1960s is that acute gastroesophageal reflux may actually provoke or worsen cardiac ischemia.

The concept of a direct association between reflux esophagitis and acute cardiac ischemia is termed *linked angina* and was first described in 1962. When patients present with reflux symptoms, they may be having reflux, but that reflux may be inducing cardiac ischemia as well. I recently reviewed two cases of missed MIs; in both, the MIs were misdiagnosed as reflux. In one of the cases, the emergency department physician admitted the patient, who had a troponin level of 0.4 ng/mL (a minor elevation) and Wellen's sign (an electrocardiographic abnormality of biphasic T waves in the mid-precordial leads, indicative of significant proximal left anterior descending coronary artery disease). The patient was sent for endoscopy by the inpatient physicians, where reflux esophagitis and the presence of some esophageal erosions were confirmed. Because of this and a positive response to antacid therapy, the cardiologist diagnosed reflux and sent the patient home, ignoring the troponin level and the electrocardiographic change. The patient died a week later at home because of a large left anterior descending coronary artery lesion and a large anterior MI.

Evaluation of a clinical decision rule for young adult patients with chest pain[5]

Marsan et al[5] looked at 1023 patients age 24-to-39 years who presented with chest pain. Patients using cocaine were excluded. Of the 98% for whom there were 30-day follow-up data, 5.4% were found to have ACS: One of 20 patients younger than age 40 years with undifferentiated chest pain presenting to the emergency department had ACS. Also, 2.2% died because of MI or underwent emergency bypass or percutaneous coronary intervention (PCI). In the course of two or three shifts at the University of Maryland Medical Center, I see 20 people with chest pain. On the basis of the statistics of Marsan et al,[5] I can expect that one of the patients I see during those shifts is going to have ACS, and I can expect that in six shifts, one patient is going to have an adverse cardiac event (one of 50 patients younger than age 40 years dies, has an MI, or undergoes emergency bypass). From the data, Marsan et al[5] created a very low-risk protocol: If patients have no cardiac history and either no risk factors or normal electrocardiographic findings and normal cardiac enzymes, then their risk is only 0.14%. Even so, a 23-year-old woman presented to the University of Maryland Medical Center Emergency Department with chest pain and dyspnea on exertion. She was not obese and had only a single cardiac risk factor—bad luck. Her electrocardiograph showed flat T waves in lead II, inverted T waves and mild ST-segment depression in leads III, and arteriovenous fistula. My colleagues argued with the inpatient service to get her admitted despite the fact that she was only 23 years old and had no known risk factors. After MI was ruled out, she was discharged the next morning. Four days later, she presented to another hospital in Baltimore with a troponin level of 12 ng/mL; in the catheterization laboratory there, she was found to have a 100% lesion of the right coronary artery.

The take-away point is this: Do not discount cardiac concerns even in young patients. One of my colleagues

treated a 12 year old with MI who had no risk factors, did not use cocaine, and had not taken sympathomimetics or cold medicines. The patient went into his parents' bedroom in the middle of the night, diaphoretic, and they took him to the local hospital. Electrocardiography showed that the patient was having an acute ST-segment elevation MI, so he was transferred to a pediatric hospital, where the catheterization laboratory found that he had vasospasm. He will be taking beta-blockers and calcium channel blockers for the rest of his life.

Diagnosis

Single indeterminate-range troponin is associated with inpatient mortality in patients presenting to an emergency department[6]

Until 2005, my institution considered a troponin-I level greater than 1.5 ng/mL to be positive and a level less than 1.5 ng/mL to be of no concern. Increasingly, cardiology literature published since 2002 indicates that patients with "indeterminate" or "marginally elevated" troponin-I levels (> 0 ng/mL but < 1.5 ng/mL) have a higher six-month and one-year mortality than patients with negative values. The study by Waxman and Husk,[6] presented earlier in 2005, indicated that these patients also have increased inhospital mortality—5.5% for those with an indeterminate level and 8.3% for those with positive values. Patients with negative troponin values had a mortality of only 2.3%. The lesson is that troponin levels represent a continuum of risk. Every gradation of elevation of troponin puts the patient at a higher risk for short- and long-term mortality, so troponin levels not in the "positive" range cannot be discounted. Any value other than zero must be taken seriously.

Treatment

Association of intravenous morphine use and outcomes in acute coronary syndrome: Results from the CRUSADE quality improvement initiative[7]

In an article published in the *American Heart Journal* in 2005, Meine et al[7] described a nonrandomized retrospective study of patients admitted through the CRUSADE Registry—a multicenter registry of 57,039 patients from more than 443 hospitals across the US with non-ST-segment elevation and ACS—that evaluated patients' medications, interventions, inhospital outcomes, and discharge treatment. Thirty percent of patients received morphine within the first 24 hours of admission. All evidence-based therapies for the morphine group versus the nonmorphine group were identical, as were the severity of illness and electrocardiographic abnormalities. Patients who received morphine within the first 24 hours had a slightly higher risk of death (odds ratio, 1:48) and a higher likelihood of post-admission MI, CHF, and cardiogenic shock. It is not clear whether the morphine caused these problems, which could also result from a histamine effect or direct myocardial depression, which has been demonstrated before. In any case, until further notice, be cautious about giving morphine to patients for whom there is a strong suspicion of ACS, maximize nitroglycerin dosing, and send patients with intractable pain to the catheterization laboratory.

Times to treatment in transfer patients undergoing primary percutaneous coronary intervention in the United States[8]

In 2004, many studies focused on routine transfer to other hospitals for percutaneous intervention. The current American College of Cardiology–American Heart Association (ACC-AHA) guidelines recommend PCI (balloon inflation) within 90 minutes and thrombolytics after this time frame. In a 2005 study of 4278 patients from 419 hospitals undergoing interhospital transfer for primary PCI, Nallamothu et al[8] found that the median total door-to-balloon time was 180 minutes, 90 minutes longer than the national guideline. Only 4.2% of the 4278 patients were given PCI within the recommended 90 minutes. The presence of comorbid conditions, absence of chest pain, delayed presentation after symptom onset, nonspecific electrocardiographic findings, and presentation to a hospital during off-hours were associated with longer times. Nallamothu et al noted that even at US hospitals with PCI capability, the time to balloon inflation was 120 minutes.

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How long does it take from a patient's arrival in the catheterization laboratory to get the balloon inflated? Many physicians think they do a great job when they transfer patients to the laboratory within 60 minutes. But what happens once a patient gets to the laboratory? The team meets the patient, obtains a quick medical history and does a rapid physical examination, obtains informed consent, moves the patient into the procedure room, prepares the patient, drapes the patient, inserts the catheter into the patient's groin, threads the needle, injects dye, finds the infarcted artery, threads the needle down into that artery, and blows up the balloon. On average, the time from arrival at the cath-

eterization laboratory to balloon inflation is approximately 30 to 35 minutes. Therefore, in considering national 60-minute or 90-minute recommendations, these 30 minutes must be accounted for.

Transfer for primary angioplasty: The importance of time[9]

In another study looking at transfer, Hermann[9] reviewed the literature pertaining to thrombolytics and found that less than 30% of US patients have a door-to-balloon time of less than 90 minutes, even when the presenting hospital has the ability to do the catheterization itself, despite the fact that national guidelines call for a maximum of 90 minutes. This means that 70% of hospitals are not meeting that goal. This and other studies call into question the practice of routine transfer to another facility for PCI. Also, the importance of rapid reperfusion in early-presenting patients is vital, so much so that if the patient is in the emergency department within two hours of symptom onset and you cannot get him or her to a catheterization laboratory within 60 minutes, you should strongly consider giving the patient thrombolytics. The best indications for PCI are still cardiogenic shock, thrombolytic failure, long symptom-to-presentation time, and very short transfer time.

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Pharmacological facilitation of primary percutaneous coronary intervention for acute myocardial infarction[10]

Many articles now propose routine transfer of patients with an ST-segment elevation MI to the nearest site for PCI if there is not a catheterization laboratory in the local hospital. Many physicians are doing such routine transfers. Unfortunately, however, when patients are transferred for PCI, they rarely undergo PCI within the recommended 90 minutes of presentation. As described earlier in this article, even physicians in hospitals with catheterization laboratories do not usually get these patients to the laboratory within 60 to 90 minutes. Despite the delay in both situations, only a few physicians also administer thrombolytics and then transfer patients.

In an editorial in the *Journal of the American Medical Association*, Gersh et al[10] looked at multiple reports to determine the likelihood of success of either facilitated PCI (thrombolytics or G2B3A receptor antagonists followed by PCI) or transfer for PCI or thrombolytics alone. They concluded that the longer the time from the start of symptoms to intervention, the greater the benefit of PCI compared with thrombolytics. Current cardiology literature is weighted in favor of making the intervention decision on the basis of time of onset of the patient's symptoms. Gersh et al recommended that if a patient arrives within two hours of symptom onset, the physician should perform PCI only if a balloon can be inflated (open artery) in the catheterization laboratory within 60 minutes—not 90 minutes as has traditionally been specified. If this cannot be done, Gersh et al recommend administering thrombolytics because their benefit will outweigh any gained by transfer to a catheterization laboratory for PCI. Gersh et al recommend considering facilitated PCI—administering thrombolytics or abciximab and then transferring the patient—if the patient arrives two-to-three hours after symptom onset, and extending the allowable time to PCI to 90 minutes if the patient arrives more than three hours after symptom onset.

Abciximab as adjunctive therapy to reperfusion in acute ST-segment elevation myocardial infarction: A meta-analysis of randomized trials[11]

Another important study concerned facilitated PCI with abciximab (ReoPro). De Luca et al[11] reviewed randomized trials involving 27,115 patients with ST-segment elevation MI treated with abciximab. The condition worsened in patients who were given thrombolytics and abciximab, but the condition improved in patients who underwent angioplasty and received abciximab. The latter group had better outcomes and no increase in intracranial bleeding rates. The 2004 ACC-AHA guidelines and the European literature strongly support administering abciximab before performing PCI, and the ACC-AHA guidelines rank abciximab in a higher class than any of the other G2B3A inhibitors. Yet why are so many more physicians using eptifibatide? Perhaps the latter drug is more heavily marketed.

Congestive Heart Failure

Morphine for acute decompensated heart failure: valuable adjunct or a historical remnant?[12]

In the world literature on morphine in decompensated heart failure, there is practically no evidence demonstrating that morphine decreases preload in the central circulation. Where did this concept arise? Studies from the 1970s indicated that when physicians injected morphine into patients' peripheral veins, the veins vasodilated slightly. From this, it was extrapolated that morphine decreases preload. However, if a patient is short of breath, I want to know about the status of the central circulation, not what's happening in the periphery. Swan-Ganz catheter data in patients who receive morphine show no decrease in central circulation

preload, and in fact morphine may be associated with a decreased ejection fraction. Peacock et al[12] found that in 14% of 20,282 patients in the Acute Decompensated Heart Failure National Registry (ADHERE)—patients admitted with CHF—who received morphine had a five-fold increase in mortality (13% versus 2.4%), a five-fold increase in need for intubation and ventilation (39.7% versus 14.4%) and intensive care unit admission rate (15% versus 3.0%), and a more prolonged hospital stay (5.6 days versus 4.2 days). There was no difference between the morphine and nonmorphine groups in terms of age, vital signs, or comorbidity.

Risk stratification for in-hospital mortality in acutely decompensated heart failure[13]

Fonarow et al[13] analyzed clinical outcomes from the ADHERE (more than 10,000 patients from 263 hospitals) to determine which of 39 factors are predictive of inhospital mortality for patients admitted with decompensated heart failure. The three best predictors, in decreasing order, are elevated blood urea nitrogen level (>43 mg/dL), systolic blood pressure at admission <115 mm Hg, and serum creatinine level at admission >2.75 mg/dL. Patients with none of these factors had a 2% mortality rate; those with all three factors had a 22% mortality rate. Using these factors may help determine what level of care patients need. The current practice at my institution is that patients who have even one of these three factors are automatically sent to a step-down unit, an intensive care unit, or a critical care unit. One would presume that this higher level of monitoring is more beneficial than simple telemetry monitoring in patients known to be at increased risk of mortality.

Risk of worsening renal function with nesiritide in patients with acutely decompensated heart failure[14] and *Nesiritide and worsening of renal function: The emperor's new clothes?*[15]

This article by Sackner-Bernstein et al[14] and the accompanying editorial by Teerlink and Massie[15] discuss the association of nesiritide, a heavily marketed drug, with worsening of renal function in patients with decompensated heart failure. Sackner-Bernstein et al[14] reviewed five randomized trials of nesiritide in more than 1200 patients. Because complete data from the studies were not available, the authors obtained them from the US Food and Drug Administration (FDA). They concluded that patients who received nesiritide had an increased risk of worsening renal function. These patients did not have an increased need for dialysis, but 11% (versus 4% of control subjects) needed extra medical intervention—presumably renal consultation, ultrasound, urinary electrolytes—which translates into increased costs for the patients who received nesiritide. In addition, Sackner-Bernstein et al[16] conducted a study of nesiritide and its effect on mortality, again obtaining data from the FDA. They found only three randomized double-blind trials in which the authors actually reported 30-day mortality; 485 patients received nesiritide and 377 were control subjects. Sackner-Bernstein et al found that patients receiving nesiritide had a 7% mortality at 30 days versus the 4% for control subjects. We already know from the Vasodilation in the Management of Acute Congestive Heart Failure studies that these patients also have a higher 90-day mortality rate.

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The bulk of data shows that there is no indication for nesiritide in the Emergency Department for routine treatment of patients with decompensated heart failure. Aggressive use of nitrates, aggressive use of noninvasive ventilation, use of angiotensin-converting enzyme inhibitors, and reduced use of furosemide and morphine will benefit patients far more than any other type of new medication.

Syncope

Impact of the application of the American College of Emergency Physicians recommendations for the admission of patients with syncope on a retrospectively studied population presenting to the emergency department[17]

Elesber et al[17] assessed the utility of the clinical policy of the American College of Emergency Physicians (ACEP) for the evaluation and treatment of patients presenting with syncope. Specifically, they wanted to determine whether the policy would help detect (and result in admission of) all patients with a cardiogenic origin of syncope. The ACEP policy has two main levels of recommendation, levels B and C. The level B recommendation is to admit patients with syncope with any of the following: history of CHF or of ventricular arrhythmia (including premature ventricular contraction); associated chest pain or other symptoms compatible with ACS; evidence of CHF or valvular heart disease on examination; or electrocardiographic findings of ischemia, arrhythmia, prolonged QT interval, or bundle branch block. The level C recommendation is to consider admission for patients with syncope and any of the following: age >60 years, history of coronary artery disease or congential heart disease, family history of unexpected sudden death, or exertional syn-

cope in younger patients without an obvious benign cause for the syncope.

Elesber et al evaluated 200 adult patients presenting with syncope, 24 of whom had cardiac syncope. When the ACEP level B recommendation was applied to the study population, all patients who on further workup were found to have cardiac syncope would have been admitted from the Emergency Department (100% sensitivity) and 81% of patients with no cardiac syncope would have been discharged (81% specificity). The admission rate would have been 28.5%. When the level C recommendation was applied to the population, the sensitivity, specificity, and admission rates would have been 100%, 33%, and 71%, respectively. The authors essentially concluded that applying the level B recommendation would both allow all cases of cardiac syncope to be detected and reduce hospital admission rates, whereas adding the level C recommendation would increase admissions without offering any advantage. Note that routine admission of elderly patients does not seem to be warranted unless they meet level B criteria. The main drawback to the study was its small size. Larger validation studies are underway. If the validation studies also support following level B criteria only, it should change our practice with regard to our approach to patients with syncope.

Perhaps in contrast to traditional thought, chest compression rates really do matter.

Resuscitation

Chest compression rates during cardiopulmonary resuscitation are suboptimal: A prospective study during in-hospital cardiac arrest[18]

The AHA recommends chest compressions at a rate of 100 per minute. Abella et al[18] studied 97 cardiac arrests and found that the chest compression rate was <80 per minute in 37% of patients and <70 per minute in 25%. Higher compression rates were significantly correlated with initial return of spontaneous circulation: For a rate of 95 compressions per minute, there was a 75% return, and for 40 compressions per minute, a 42% return. Perhaps in contrast to traditional thought, chest compression rates really do matter.

Many recent prehospital studies have also focused on the importance of chest compressions, and many others have significantly downplayed the role of ventilation. In fact, when patients are ventilated too rapidly, this decreases venous return by increasing intrathoracic pressure and decreases cardiac output, a combination associated with worse outcomes. A study at a well-known major academic medical center recoded bagging rates for all patients in cardiac arrest. The average bagging rate was 55 compressions per minute. When bagging is that frequent, venous return and therefore cardiac output are severely compromised.

Conclusion

These are the lessons of the studies reviewed here: Beware of diabetes. It is now considered an atherosclerotic disease equivalent, not just a risk factor. Consider admitting these patients at a higher level of care. Do not discount chest pain, including pain in patients under emotional stress. Avoid making a quick diagnosis of reflux in patients with chest pain or reflux symptoms; consider the possibility of an atypical presentation. Any elevation of troponin levels should be taken seriously, and discuss with the cardiologist when the workup is going to occur. As for treatment of ACS with morphine, just say "no!" Transferring patients for PCI takes a lot longer than is immediately apparent. Remember, the average time to balloon inflation was three hours in transferred patients, not 90 minutes. Facilitated PCI is going to become more and more common to buy time for transfer to another site. The best evidence supports the use of abciximab over eptifibatide. Beware renal dysfunction in patients with CHF. Elevated blood urea nitrogen and creatinine levels are two of the three factors that most accurately predict a poor prognosis in patients with CHF who are admitted to a hospital. Be wary of using nesiritide in patients with CHF. For patients with syncope, follow level B ACA-AHA recommendations. For patients in cardiac arrest, focus on the basics: Stop hyperventilation and increase the chest compression rate. ❖

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References

1. Fazel R, Fang J, Kline-Rogers E, et al. Prognostic value of elevated biomarkers in diabetic and non-diabetic patients admitted for acute coronary syndromes. Heart 2005 Mar;91(3):388–90.
2. Huizar JF, Gonzalez LA, Alderman J, Smith HS. Sulfonylureas attenuate electrocardiographic ST-segment elevation during an acute myocardial infarction in diabetics. J Am Coll Cardiol 2003 Sep 17;42(6):1017–21.
3. Culic V, Eterovic D, Miric D. Meta-analysis of possible external triggers of acute myocardial infarction. Int J Cardiol 2005 Mar 10;99(1):1–8.
4. Dobrzycki S, Baniukiewicz A, Korecki J, et al. Does gastroesophageal reflux provoke the myocardial ischemia in patients with CAD? Int J Cardiol 2005 Sep 15;104(1):67–72.
5. Marsan RJ Jr, Shaver KJ, Sease KL, et al. Evaluation of a

clinical decision rule for young adult patients with chest pain. Acad Emerg Med 2005 Jan;12(1):26–31.

6. Waxman DA, Husk G. Single indeterminate-range troponin is associated with inpatient mortality in patients presenting to an emergency department. Acad Emerg Med 2005;12(5;suppl 1):133.
7. Meine TP, Roe MT, Chen AY, et al; CRUSADE Investigators. Association of intravenous morphine use and outcomes in acute coronary syndrome: results from the CRUSADE Quality Improvement Initiative. Am Heart J 2005 Jun;149(6):1043–9.
8. Nallamothu BK, Bates ER, Herrin J, et al; NRMI Investigators. Times to treatment in transfer patients undergoing primary percutaneous coronary intervention in the United States: National Registry of Myocardial Infarction (NRMI)—3/4 analysis. Circulation 2005 Feb;111(6):761–7.
9. Hermann HC. Transfer for primary angioplasty: the importance of time. Circulation 2005 Feb;111(6):718–20.
10. Gersh BJ, Stone GW, White HD, Holmes DR Jr. Pharmacological facilitation of primary percutaneous coronary intervention for acute myocardial infarction: is the slope of the curve the shape of the future? JAMA 2005 Feb 23;293(8):979–86.
11. De Luca G, Suryapranata H, Stone GW, et al. Abciximab as adjunctive therapy to reperfusion in acute ST-segment elevation myocardial infarction: a meta-analysis of randomized trials. JAMA 2005 Apr 13;293(14):1759-65.
12. Peacock WF, Hollander JE, Diercks DB, Fonorow G, Emerman CL. Morphine for acute decompensated heart failure: valuable adjunct or a historical remnant? Acad Emerg Med 2005;12(5; suppl 1):97-8.
13. Fonarow GC, Adams KF Jr, Abraham WT, et al; ADHERE Scientific Advisory Committee. Risk stratification for in-hospital mortality in acutely decompensated heart failure: classification and regression tree analysis. JAMA 2005 Feb 2;293(5):572–80.
14. Sackner-Bernstein JD, Skopicki HA, Aronson KD. Risk of worsening renal function with nesiritide in patients with acutely decompensated heart failure. Circulation 2005 Mar 29;111(12):1487–91.
15. Teerlink JR, Massie BM. Nesiritide and worsening of renal function: the emperor's new clothes? Circulation 2005 Mar 29;111(12):1459–61.
16. Sackner-Bernstein JD, Kowalski M, Fox M, Aaronson K. Short-term risk of death after treatment with nesiritide for decompensated heart failure: a pooled analysis of randomized controlled trials. JAMA 2005 Apr 20;293(15):1900–5.
17. Elesber AA, Decker WW, Smars PA, Hodge DO, Shen W-K. Impact of the application of the American College of Emergency Physicians Recommendations for the admission of patients with syncope on a retrospectively studied population presenting to the emergency department. Am Heart J 2005 May;149(5):826–31.
18. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation 2005 Feb 1;111(4):428–34.

More

The more that you read,

the more things you will know.

The more that you learn,

the more places you'll go.

— Dr Seuss (Theodor Seuss Geisel), 1904-1991, Pulitzer, Peabody, and Emmy Award-winning author and cartoonist