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Missed MI: Costly, deadly, and sometimes unpreventable

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Malpractice litigation targeting emergency departments (EDs) and ED physicians is dominated by the problem of ischemic heart disease (IHD), with its myriad convolutions and expressions, including sudden death and congestive heart failure (CHF).[1-4] IHD is a fearsome problem because the consequences of a failed diagnostic effort or a wrong choice can mean infarction, arrhythmia with death, or severe injury. Moreover, the problem presents in adult males and females with dependents and careers; therefore, losses and awards in cases of negligence are significant. Actuarial studies have shown high average awards.[5,6]

If a cab driver who brings a patient to the hospital knows that chest pain can mean heart trouble, why do emergency physicians still miss as many as 10% of myocardial infarctions (MIs) and many cases of unstable or new angina?[7-10] The answer is that the diagnosis of IHD is not easy and requires a high index of suspicion, even in atypical situations. Exercising extreme care in cases of chest pain and unexplained respiratory and circulatory complaints can help reduce the risk. Some hospital staffs reduce the worry of missing IHD by admitting all chest pain cases, but that cannot be the standard of care, since a high percentage of chest pain is not caused by IHD. This issue of *ED Legal Letter* will discuss a reasonable standard of care that ED physicians should meet when evaluating patients with possible IHD. This discussion is intended to reduce the chance that the emergency physician will hear the ominous inquiry, "Remember that guy with chest pain you saw two nights ago?"

IHD was addressed in *ED Legal Letter* a number of years ago, and deserves a regular vetting. Work done by medical societies, government work groups, and risk management experts, along with the tremendous clinical studies and papers from expert physicians inside and outside of emergency medicine, continue to contribute valuable new information on the subject. Cardiology and IHD diagnostic and management research is a very active area. This is not an all-inclusive review, but focuses on standards of care for evaluation and management

that most frequently are the focus targets of malpractice claims against emergency physicians.

The American College of Emergency Physicians (ACEP) chose nontraumatic chest pain as its first clinical guideline, published in 1990. ACEP updated its clinical practice guidance on IHD in 1995, and published a revised clinical policy in 2000.[11] This focused attention far exceeds ACEP's clinical policy development on any other entity, for good reason. IHD is a major cause of death and morbidity in the United States, and certainly is a major responsibility for ED physicians. Research in cardiology is extraordinary, and developments in diagnosis and treatment of IHD create an avalanche of information for all practitioners. A review of all three ACEP policies on IHD shows that diagnostic guidance is driven by history, physical, and electrocardiogram (ECG) evaluation. The treatment and management of IHD is driven by lab, ECG, and imaging results. The first two ACEP policies focused on the complaint-driven work-up of nontraumatic chest pain, and the third focused on the cardiology diagnostic and management standards for IHD.

Research from the field of clinical cardiology on early identification and management of candidates for thrombolytics has become part of the emergency medicine lexicon, along with widespread promotion of clinical practice guidelines for use of aspirin, beta blockers, anticoagulation, antiplatelet drugs; and interventional and surgical therapies; and the success rates of each.[12-17] New developments in anticoagulation therapy and antiplatelet therapy create additional problems for emergency physicians who try to keep up with the rapidly changing recommendations on management.

Our Success Creates Problems

Today, complicated heart patients are kept alive longer, and some diagnostic and therapeutic problems are created by these successes. Patients who would have died a few years ago are now getting second, third, or sixth interventions. Their psychiatric, emotional, and family conditions affect their presentations to the ED. They know what the medical process is. They come in saying their pain is "6.5 over 10," that they have diaphoresis and dyspnea, and that they know their cardiologists' phone numbers by heart. Nothing comes easy in such situations. So many are saved that we have a whole population of people with advanced heart and vessel disease who have personal cardiologists, electrophysiologists, and cardiovascular surgeons. In those situations, ED physicians are temporary interveners who are faced with a critical decision — is this latest presentation important, different, and ominous? The joke might go, "Who gets sued for not managing the patient well?" "The last physician to see the patient before the catastrophe."

Patients with proven or suspected heart disease—and their families—have high expectations for success. Patients with coronary artery bypass scars who smoke and don't exercise still want to live to see their grandkids off to college.

ED physicians must consider atypical and occult IHD as typical,[18] silent MI and IHD as common,[19] and missed MI as more common than the clinical studies report. They also must accept that patients of advanced age with chronic illness or other problems that mask ischemic presentations are part of the ED patient population. The reports in the literature show that silent

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ischemia is just as prone as known ischemia to major catastrophic complications like sudden death.[20]

Heart disease is important for the physician and patient as a potential cause of catastrophe and disability. In terms of impact on malpractice litigation, nothing else comes close. All the closed claims information and risk management literature put IHD ahead of cancer, infection, and trauma in terms of magnitude of high-dollar claims. Judgments and settlements for missed MI and IHD with sudden death or other complications average as much as $250,000 in awards, and in claims studies are exceeded only by brain and spinal cord injury cases.[21] Medical advances and successes create new problems, including lawsuits about therapy and loss of functioning myocardium. Typical would be a claim that failure to give heparin caused damaged myocardial tissue and loss of ejection fraction. Such claims stretch the science of clinical studies in IHD that have patency or mortality as their endpoints.[22] Now, malpractice claims are filed because of claims of incomplete, delayed, or inadequate therapy. For example, experts might appear to say that a patient would have been better if heparin had been started earlier or aspirin had been given. Sometimes, the science simply is not there to support the opinion.

Another problem is the timing of thrombolytics and other interventions from the point of "the event." In studies, the effects on mortality are time-related, but that assumes a specific time of event, which is not always known. Stuttering events are common, so close analysis of each case is necessary. Sudden death is one thing, but cardiac damage is another. The plaintiff in a malpractice case must establish that, more likely than not, the omission or negligence caused a measurable injury — not a speculative consequence, but one supported by current science. Thrombolytics are known to reduce mortality — when given in a timely manner — from about 14% to 7%, but the effect of thrombolytics on prevention of sudden death or CHF is not known. The non-Q wave infarction is not even considered in the thrombolytic research, and the research on IHD management and outcomes is not definitive because matching patients and endpoints is very difficult. Broad assertions in a malpractice litigation should be subjected to careful scientific scrutiny.

What Is Ischemic Heart Disease?

A short definition of IHD is a mismatch of oxygen with heart muscle and conduction or pacer tissue caused by narrowing, spasm, inflammation, obstruction, stress, demand, abnormal vessels, valvular and or structural abnormalities. The clinical expression of the disease is in heart failure, arrhythmias, and resultant risk to life or quality of life.

The most common catastrophic manifestation of IHD that comes to mind is sudden death. Too often, without thinking, physicians write on death certificates or in the record that death is due to "massive heart attack." Well, it *must* have been massive, right? But all it takes is a few minutes of a nonviable rhythm for death to result. Many patients die without a new infarction or occlusion. Moreover, IHD can present for the first time as fatal arrhythmia, but it also increasingly is a problem in patients who develop chronic heart disease and cardiomyopathy.

The biggest problem in the area of IHD litigation continues to be failure to recognize abnormalities of ischemia in the 12-lead ECG. Some studies show that retroanalysis by cardiologists picks up a 20% or greater miss rate by noncardiologists.[9] There also is a standard of care in the history, physical, and other diagnostic options available in evaluating patients for possible IHD; the initial management of suspected IHD; and reasonable decision-making on disposition.

Fatal Cardiac Arrhythmia. Electrolyte problems, drugs, hypertension, dilated cardiomyopathy, alcohol or alcohol withdrawal, caffeine, prescription medications, and even antiarrhythmics can cause fatal cardiac arrhythmias, so now the standard of care to be considered is how to evaluate risk of fatal arrhythmia, and when to install a defibrillator. The standard of care being discussed is automatic cardioverters for dilated cardiomyopathy, because of the greater than 10% annual rate of sudden deaths in the group of patients with fixed dilated myocardopathies and decreased ejection fractions.[23] The job of the ED physician is becoming formidable — keeping track of new recommendations and considering the ominous threat of IHD, all amidst the pressures and limits of ED resources and time.

Unstable or Stable Angina. Conditions that ED physicians constantly must keep in mind as they consider ischemia and its presentation are unstable angina and stable angina. Many of the malpractice claims might come under the label "missed MI," but they aren't. More often, they are sudden death with coronary artery disease. What is unstable angina? It can be angina that—for a particular patient—is new,

changing, more persistent, more severe, different, associated with near syncope or syncope, longer lasting, or not as responsive to nitroglycerine.

New onset, low-risk (normal ECG), suspected angina was considered during the past decade by a federal agency blue ribbon panel as being a case that could be worked up in the ambulatory setting.[24,25] This is something most ED physicians would find anxiety-provoking. In the ED, IHD is always top priority, but how many patients across the country get ambulatory evaluations by cardiologists, internists, or family practitioners when the complaint is new-onset chest pain? What is the standard of care? There is some clinical dissonance in the medical community about IHD. An emergency physician rarely feels comfortable discharging a chest pain patient with a new prescription for nitroglycerine.

Is there such a thing as stable angina that presents to the ED? Both denial and anxiety can bring a patient with chronic angina into the ED. Or, is the angina unstable by definition, since a patient with stable angina probably would not come to the ED for just another episode of stable angina? The challenge for ED physicians involves recognition of the risks, negotiating with the patient, and reaching a reasonably safe strategy and disposition, knowing that anyone with proven coronary artery disease and angina is at risk for MI and sudden, deadly arrhythmia. Discussion with the patient about the treatment plan and the related uncertainties is one step to consider.

The Framingham study showed evidence of occult or undiscovered MI in one-third of study patients who had MI. Those occult MIs affect quality of life, even if they aren't as likely to cause sudden death as known MIs. [26,27] Silent MI or silent IHD is not benign — it increases mortality and morbidity.[28] The silent IHD and MI reports from the clinical studies don't address occult or silent MI that leaves no scar or relic of some kind on the ECG. The Framingham study does not report the nature of the miss, or whether it was caused by the physician, patient, or an unknown factor. The study leaves out the MIs that do not leave scars, non-Q wave MIs, creatine phosphokinase myocardial band (CPK-MB), and troponin injuries that were treated as flu or bronchitis.

Both older and more recent studies on failure to diagnose inpatient MI premortem showed that physicians miss inpatient MI almost half the time. The small and non-clinical infarctions are missed all the time, resulting in a clinical entity that is much more pervasive than it might appear.[29] ED physicians and primary care physicians wade through an increasingly older patient population, trying to meet the expectations of the public and comply with a changing and demanding standard of care. Everyone knows that there are diabetics, the elderly, and just unlucky people who have silent ischemia. When someone presents weak and dizzy, think of IHD and consider the need to identify those atypical cases. When the patient presents with something other than chest pain, such as epigastric burning, dyspnea, near syncope, orthopnea, and weakness, at least think about IHD, particularly in the diabetic or the elderly. Finally, IHD must should be considered even in those patients with proven pulmonary or gastrointestinal disease. **(See Tables 1 and 2.)**

Table 1. Handling IHD in the ED

- Obtain ECG and CPK-MB tests when patient has a history and physical that at least imitate the computer protocols.
- If acute MI is found, with ST segment elevations of a diagnostic nature and no contraindications, then thrombolytics, streptokinase, and tissue plasminogen activator (tPA) are equally effective; or, intervention in cardiac catheterization lab, if available immediately.[17]
- Repeat ECGs and enzymes in rule-out angina or MI cases, with constant ST monitoring if available.
- Give aspirin, heparin, beta blockers, and ACE inhibitors (or now, angiotensin II inhibitors) for MI.
- In the work-up, if the ECG was normal or non-diagnostic and clinical suspicion was present, repeat ECG and labs to rule out injury, followed by studies like technesium-99 or stress testing.
- Resting and stress technesium-99 study is the best non-invasive study to find IHD and its location.

What We Know About Diagnosing IHD

The Heart Attack Alert Program Working Group (Working Group), representing the American Colleges of Cardiology, two groups of cardiologists and ED physicians, and some 40 other organizations, has produced and published a comprehensive and valuable assessment of diagnostic tools for IHD in the ED or ambulatory care setting.[30] The Working Group evaluated the reliability and sensitivity of tests commonly used to diagnose IHD, and rated them for practical clinical usefulness. They studied lab, imaging, electrocardiographic, stress, and computer-

assisted diagnostic tools, and rated them on a scale of effectiveness, sensitivity, and specificity, and then organized their advice in an orderly and comprehensible monograph.

History and Physical Basics. The Working Group rated the most important historical elements that suggest IHD as follows:

- location, severity, circumstances, and duration of pain;
- radiation; associated symptoms like syncope, palpitations, diaphoresis, nausea, and vomiting;
- age, sex, body habitus, history of smoking, diabetes, hypertension, and family history; and
- abnormalities of lipids, cholesterol, and heart anatomy.

Physical findings that make a difference are evidence of heart arrhythmia, heart failure, and new murmur, but frequently there are no physical findings that are convincing or helpful.

ECG. The ECG is evaluated for ST segment and T wave changes, new arrhythmias, new axis, or conduction defects. Critical ST segment elevations (measured in two contiguous leads) are 1 mm in the limb leads or 2 mm in the chest leads, indicating possible injury justifying thrombolytics. Other changes, including new Q waves, ST segment depressions, flipped T and U waves, straightened ST segments, and elevations of the J point, may suggest early and important changes of IHD.

We know that the best tool in the armamentarium for diagnosing IHD in the ED is the 12-lead ECG — or is it? One-time, 12-lead ECG is easy to do, accessible, and low-tech. However, it has a low sensitivity in diagnosing ischemia. The ECG can be normal in acute MI. ECG also is non-specific to a high degree. It is good for identifying high-risk patients and in diagnosing injury that would justify thrombolytics if other criteria were met, and when the ECG is abnormal, the physician is well well advised to be alert. However, when the ECG is silent, the dilemma of occult or clinical IHD still is real. Ischemic changes are important because, when present, they justify and encourage intervention. There are so many interventions now available — aspirin, heparin, platelet inhibitors, catheters, rotor blades, lasers, stents, and surgeons.

However, clinical suspicion is important in patients with suspect history and normal ECG. Right-sided ECG leads, additional posterior leads, and body-mapping strategies, along with continuous ST segment monitoring, may be helpful. Continuous ECG 12-lead, ST segment monitoring certainly can be enlightening, and can be the equivalent or better of repeated ECGs. The inability to recognize subtle ECG changes of ischemia frequently can be a telling thing in malpractice litigation, so emergency physicians increasingly must raise their skill levels and competency in electrocardiography. Minor ST and T wave changes may not be diagnostic, but provide support for clinical suspicion of IHD.

Don't be afraid to repeat ECGs. In the case of new left bundle-branch block (LBBB), assume MI or ischemic event. ST interpretation in LBBB is best left to cardiologists, although ST segment elevation on a concordant T wave and extreme ST change on a discordant T wave are considered signs of acute injury. **(See Table 3, "ECG Hints of Subtle IHD Changes.")**

Stress Testing. The stress test is becoming a tool that is included in the ED diagnostic armory, but stress tests on the treadmill are not sensitive and have low specificity, particularly in women. The stress test prior to discharge to rule out IHD in patients is assessed by the Working Group as low on a list of useful tools with low sensitivity and fairly high specificity. Young women, in particular, are

Table 2. Risky Practices in the ED

- Ignoring past pain if patient is pain-free when evaluated, and ignoring long-lasting, severe, autonomic symptom-accompanied, radiating chest pain with little or no musculoskeletal elements
- Failing to get a good pain history for location, duration, and severity with associated symptoms like sweating, dyspnea, palpitations, syncope, and sense of doom
- Ignoring epigastric and upper abdominal pain as signs of inferior cardiac ischemia
- Relying too heavily on maneuvers like palpation to determine the diagnosis (6% in one study of chest wall tenderness had MI)[37]
- Using antacid therapy or lack of pain in the ED to rule out IHD. Patients can become pain-free for various reasons[38]
- Failing to review old ECGs if the new one has any abnormalities
- Excess use of the phrases "non-diagnostic" or "nonspecific ST and T wave changes" without considering that those changes might be non-diagnostic but could be ominous
- Inaccurate or ill-advised use of the term "early repolarization variant." Too often, early repolarization is ischemia in a patient who never had early repolarization before
- Misinterpretation of subtle ECG changes

Table 3. ECG Hints of Subtle IHD Changes

(Listed in order of importance)

- J point elevations of any degree
- Straightened ST segments or a loss of their normal concavity. (Be aware that leads V2 and V3 frequently have ST segment shaping changes that are difficult to evaluate.)
- T wave discordance with the vector of the QRS and peaked or hyperacute-appearing T waves
- ST depressions as ischemia (flattened is particularly indicative of possible ischemia) or ST depressions as early signs of a reciprocal injury
- Discordant U waves (the small wave after the T that probably is His-Purkinje repolarization) RR in the anterior leads (V1, V2, V3) with ST segment depression as an indicator of posterior MI
- The problem of the septal small Q that appears in II, III, and AVF and may be meaningless

prone to false positives. Sensitivity and specificity of stress testing is operator-dependent and must be evaluated in the context of the clinical situation.

Computer Logic and Risk Assessment Tools for IHD. Does risk profiling just add to the theoretical problems? Lee Goldman, MD, expert on chest pain evaluation and author of the Goldman Chest Pain Protocol, once said that risk profiling for IHD means nothing, clinically, because we treat people, not profiles.[31] Being an American adult is enough to create risk for IHD.

Clinical risk assessment and diagnostic predictive, computer-driven instruments named such as the ACI (acute cardiac ischemia) program,[32] ACI-TIPI (same ACI instrument modified to be a time insensitive predictive instrument),[33] the computer decision tool by Goldman (Goldman Chest Pain Protocol),[31] and other probability analysis tools and instruments for evaluation of IHD that computerize and systematize the process of risk assessment in IHD are useful and effective. A good history and physical combined with intelligent interpretation of lab and ECG are the keys to these risk assessment tools, along with the software scoring backup that is the equivalent of having a senior cardiologist standing at the bedside. All the tools studied by the Working Group were considered highly accurate, for good reason, because they used widely recognized criteria and were developed by excellent clinical groups. The clinical impact is considered good, pending further work and wider use.

ACI, ACI-TIPI, and the Goldman Protocol are not used widely in a formal way, but the elements of these clinical tools are used in one way or another in many hospitals. Considering that the first two are 20 and 10 years old, respectively, their popularity is not great, but they may help formalize and structure the clinical decision-making process.

CPK-MB. When used in series over several hours, CPK-MB is the gold standard for assessing cardiac injury. However, CPK-MB is insensitive for ischemia and rises only after 2-4 hours have elapsed following injury. Index and ratio-to-total CPK occasionally are important in definitive clinical use; however, they are affected by kidney disease, rhabdomyolisis, shock, and other conditions that can cause false positive results.

Myoglobin. Myoglobin is sensitive, early, but very nonspecific, and is of limited usefulness, since myoglobin needs to be measured for rise and can come from other muscle. It is useful only in a series of two.

Troponin T and I. Troponin increasingly is useful as a marker of cardiac injury from any cause. It remains detectable in the body for up to seven days and is very specific for cardiac muscle. It is very sensitive, accurate, and important clinically, but is delayed in appearance after acute injury. Troponin is helpful for delayed presentations in non-Q wave or non-ST segment ischemic events or MI. Troponin is now an area of increased interest because of the obvious clinical importance of minor premonitory injury.[34-36] Results can be elevated falsely by renal dysfunction.

Echo Resting and Stress. The results of echo and stress tests are operator-dependent, insensitive for IHD, non-specific for acute injury, and unreliable overall. They are great for evaluating complicated MI —for example, ruptured papillary muscle, mitral or other valvular dysfunction, tamponade, and dissection.

Thallium. Thallium scanning yields good information, but it is technically difficult for ED use because of timing of the injection, cost, and availability of isotope. It produces fuzzy pictures with less resolution and display than technesium-99. A unique property of thallium is that it redistributes to help identify stunned myocardium.The scan is performed as stress by adenosine or exercise, then a second picture is taken on delay, at rest, for redistribution to cells. A second injection sometimes is used for better pictures, but limits evaluation for stunned myocardium.

Technesium-99. Accurate, sensitive (95%+), specific (95%+), extremely useful, and anatomic. Technesium-99 is available in the resting or stress modes, and doesn't require injection during pain. Very high sensi-

tivity and specificity and pictures that are less operator-dependent make this test the best available non-invasive diagnostic test for IHD. It even may be more clinically useful than angiograms, except that angiograms are needed for intervention and surgical evaluation.

Case No. 1

Beard v. Kaiser Foundation Hospitals.[39] On March 19, 1985, a female patient, Mary Henry, presented to the ED of Kaiser Hospital and complained of right-side chest pain and numbness in her right arm. She was diabetic, had a previous history of previous MI and CHF, and was taking digoxin. She had an increased respiratory rate and an irregular pulse. Dr. Gruzdys, the ED physician who attended Ms. Henry, ordered only a urine test for sugar and acetone. He diagnosed Ms. Henry's condition as costochondritis, an inflammation of the rib cartilage, and prescribed an anti-inflammatory agent. One week later, Ms. Henry died of an acute MI.

Discussion. Right-sided chest pain is just as important as left-sided pain. A history of diabetes and previous MI always is ominous. In this case, the plaintiff's expert argued that admission and observation, not ECG or lab, were the standard of care. The treating physician, Dr. Gruzdys, who lost at trial and was found negligent, appealed because he said that the plaintiff's expert failed to establish the standard of care for an ED physician and that there was a breach of that standard. In this case, whether the ECG and lab were not diagnostic or were negative makes no difference, because admission to rule out ischemia in this setting is reasonable. IHD can even escape detection by even ECG and laboratory testing.

*(**Author's note:** This was an appeal on a directed verdict, so the appellant physician was attempting to show that there was adequate testimony to get to the jury. Right-sided pain is as ominous as left left-sided pain. In a patient with chest pain, don't foreclose an opportunity to prevent a catastrophe — get the ECG and labs.)*

Case No. 2

Campbell v. Hospital Service District No. 1. [40] On May 21, 1995, 78-year-old Eugene Campbell arrived at the ED of Citizen's Medical Center at approximately 8:15 p.m., complaining of chest pains that radiated into his jaws and arms. He also was sweating and was short of breath. Dr. Nguyen, a contract physician who was placed into the hospital on weekends by the Gould Group, attended to Campbell, diagnosing him with "unstable angina, rule out myocardial infarction." After reading what he believed to be an unremarkable ECG taken at 8:32 p.m., Dr. Nguyen admitted Campbell to the intensive care unit (ICU) for observation at approximately 9 p.m., after the administration of nitrates alleviated his chest pains. Contemporaneous nurses' notes upon his admission to the ICU noted that Campbell's skin remained cold and clammy and that his skin color was ashen. Dr. Nguyen, who was an ear, nose, and throat resident when not working as a contract ED doctor, did not consult with a cardiologist or call his attending physician. Dr. Nguyen advised Campbell's wife and daughter that they could return home, stating that he had angina, was being kept overnight for observation, and that transfer to a larger hospital in Monroe was unnecessary. Shortly thereafter, at 10:05 p.m., Campbell went into cardiac arrest and was resuscitated. A second ECG was then taken, which indicated damage to Campbell's heart. Campbell was transferred to St. Francis Medical Center in Monroe the next day, where a subsequent examination by a cardiologist determined that Campbell suffered an acute anteroseptal infarction complicated by cardiac arrest with mild CHF. Campbell also suffered brain damage due to oxygen deprivation during his cardiac arrest. After several weeks of hospitalization, Campbell was transferred to a nursing home, because he was unable to care for himself or walk without assistance, and was unaware of his surroundings. He died on March 19, 1996, never having returned home after his trip to Citizen's Medical Center.

An expert cardiologist opined that applicable clinical guidelines at the time mandated that a patient such as Campbell should have received aspirin and anticoagulant therapy immediately upon admission. Furthermore, the 8:32 p.m. ECG showed a subtle abnormality, which, although perhaps not as discernable to a noncardiology resident, should have resulted in better management.

Plaintiffs appealed the trial court's grant of summary judgment to the defendant medical center. The appellate court reversed and remanded for a new trial.

Discussion. Frequently, the attending or the admitting physician has to come in. That is particularly true in IHD cases. Management now is just as important in IHD cases as diagnosis. In this case, the

failure to adequately treat and appropriately consult resulted in a less-qualified physician undertreating the patient. Failure to push treatment to the aggressive side can be bad strategy.

Unfortunately, in many states, defendant ED physicians will be subject to the criticisms of cardiologists and internists as testifying experts**.**

Case No. 3

Taylor v. Decker.[41] James Taylor, a 38-year-old man, woke with chest pain at 4 a.m. on Oct. 6, 1983. Still experiencing chest pains at 10 a.m., Taylor began driving the car to the hospital, but his chest pains became so severe that he was forced to pull off the road. Shortly after 11:30 a.m., Taylor was taken into the Bethesda North Hospital ED. The admission sheet stated that at 4 a.m., Taylor began experiencing chest pain, which radiated to both shoulders. The pain was accompanied by shortness of breath and sweating. He was taking two medicines for high blood pressure, he smoked, and he was overweight. Dr. Maur was the ED physician who treated Taylor. Certain tests were administered to Taylor, including an ECG.

At approximately 1:55 p.m., Taylor was discharged from the ED. He was given a card containing instructions with respect to his chest pain. Taylor arrived home at approximately 3 p.m. and went into his living room. Suddenly, he screamed, his eyes rolled back, and he jerked violently. He was rushed back to the Bethesda North Hospital ED under full cardiopulmonary resuscitation, and he was pronounced dead upon arrival at the hospital.

Lewis Seeder, MD, a physician practicing emergency medicine, physical medicine, and rehabilitation, testified that the failure to admit Taylor to the hospital was negligent, below the standard of care of physicians, and the proximate cause of his death. Further, Dr. Seeder testified that, in his opinion, based upon the patient's history and the diagnostic tests administered, Taylor should have been admitted to the hospital for observation and monitoring of his cardiac rhythm. Dr. Seeder stated that, had Taylor been admitted to the hospital, the greatest medical probability was that he would have survived the MI and the hospitalization following the heart attack, as certain treatments would have been available in the hospital. In fact, Dr. Seeder estimated that there was an 85-90% probability that Taylor would have survived the heart attack had he been hospitalized. However, Dr. Seeder was unable to say specifically how long Taylor would have lived following his heart attack. Finally, Dr. Seeder stated that in his opinion, the direct and proximate cause of Taylor's death was the failure of Dr. Maur to admit Taylor to the hospital.

The trial court opinion was that, as a matter of law, even if Dr. Maur was negligent and failed to use that standard as recognized in the community for ED physicians, and even if he had admitted the decedent into the hospital's coronary care unit, there was no medical evidence and no expert evidence to indicate the life expectancy of the decedent. The appellate court determined that the plaintiff was not required to submit evidence as to the specific period of time that Taylor would have lived following his heart attack. It is sufficient that appellant presented evidence that the medical probability was that Taylor would have survived the heart attack and the hospitalization. The appellate court found the trial court erred in directing a verdict in favor of the defendants. The case was reversed and cause remanded for a new trial.

Discussion. In this case, the trial court was reversed on a flimsy summary judgment based on the failure to show certain damages by not providing evidence of Taylor's life expectancy. The reader can see that the negligence issues are very strongly in favor of the plaintiff. The history was classic, the patient had numerous risk factors for IHD, and should have been admitted, regardless of the ECG. Severe ischemic pain with diaphoresis and dyspnea is enough for admission, even if the pain occurs outside the ED and is gone by the time the patient arrives at the ED.

Case No. 4

McKain v. Bisson.[42] At 11 p.m. on June 1, 1989, McKain, a truck driver, left his home near Louisville, KY, to drive his truck to Michigan. At 6 a.m., he began experiencing double vision and chest pains, so he stopped his truck in Angola, IN. He was taken by ambulance to a hospital in Angola, where he was admitted to the ED at 9:20 a.m. The ED record indicates that around 6 a.m., McKain began suffering from chest pain that radiated into both arms, with shortness of breath, and

sweatiness. The fact that McKain had undergone triple bypass surgery in 1985 was also noted on the ED record. Shortly after McKain presented to the ED, Dr. Bisson, the treating emergency physician, ordered that McKain be given nitroglycerin. The nitroglycerin gave McKain some relief, and he slept.

McKain was discharged at 11:20 that morning, and taken by police car to a hotel. (Angola does not have taxi service.) McKain's daughter, son, and daughter-in-law picked him up at the hotel around 5 p.m. They took McKain to Indianapolis, where his son lived. McKain continued to experience chest pains. At one point during the drive to Indianapolis, his son almost exited the highway to take his father to a hospital in Fort Wayne, IN, but McKain insisted on seeing his own doctor in Louisville. Upon reaching Louisville, McKain was admitted to a hospital at 1:29 a.m. on June 3. Testing indicated that he had suffered a heart attack at some point on June 2.

McKain had coronary heart disease. Out of 12 siblings, all but one (a half-sister who had a different mother) had heart problems. McKain had two more heart attacks in the summer of 1992, and again underwent bypass surgery. Pursuant to Indiana law, McKain filed a complaint with the Indiana Department of Insurance prior to filing suit in court. A medical review panel rendered an advisory opinion, which was entered into evidence at trial. The panel found that Dr. Bisson had "failed to comply with the appropriate standard of care" when he misdiagnosed McKain's condition. The panel was unable to determine whether McKain suffered any damages as a result of the misdiagnosis.

Discussion. In this case, the physician won because the plaintiff failed to put together a lucid case, and there were some delays in care that were because of the plaintiff. However, you would not want to defend this case for failure to take an adequate history, family history, and failure to play it safe. Loss of chance is an accepted claim for recovery in 30 of the 50 states, even though not accepted in Indiana, and that was one reason for the appeal of this case after the jury found for the defendant. Be extremely sensitive to morning chest pain, since the circadian rhythms produce more MIs in the morning**.** Chest pain that awakens may be ominous.

Case No. 5

Boburka v. Adcock.[43] In 1984, Theodore T. Boburka was a 46-year-old Florida resident with a history of heart trouble. Boburka was a construction engineer, and his job required him to travel to oversee projects. Boburka's physicians recommended that while traveling, he keep copies of his ECGs with him and make contact with a physician in each location where he was assigned.

In July 1984, Boburka was assigned to Memphis, and his daughter Heather accompanied him to that city. While in Memphis, he had occasion to visit Dr. William Burrow, to whom he had been referred by a medical referral service. Boburka related to Dr. Burrow his history of heart trouble and described the medication he was presently taking. On Aug. 15, 1984, while traveling, Boburka exerted himself by running through an airport and lifting heavy luggage. He experienced severe pain. The next day, Aug. 16, he again visited Dr. Burrow, who performed an ECG and diagnosed muscle strain.

Very early on the morning of Aug. 18, Boburka woke up with severe pain, and his daughter drove him to the Methodist Hospital South ED. A nurse took his vital signs, and the defendant, Dr. Frank Adcock, examined him. Dr. Adcock's examination, according to Boburka, consisted of poking him on his left shoulder blade a few times. Dr. Adcock was told of Boburka's history of muscle strain and was advised that a recent ECG had been taken, but he did not order a new ECG or a chest x-ray. Dr. Adcock diagnosed Boburka's condition as muscle strain and prescribed a small dose of Demerol. After returning home, Boburka continued to suffer severe pain. He returned to the hospital ED at 6:45 a.m. and was again seen by Dr. Adcock, who gave Boburka another dose of the same pain medication. Boburka then returned home and fell asleep.

Later that same day, Boburka visited a Dr. Mellor, a physician in Dr. Burrow's clinic. Dr. Mellor received Boburka's history and diagnosed muscle strain. Boburka visited Dr. Burrow, who also diagnosed muscle strain. On Aug. 24, Boburka began suffering severe pain and had difficulty breathing. He returned to the Methodist Hospital South ED, where Dr. Adcock saw him briefly. After Boburka passed out, another physician completed the exam-

ination. Boburka was diagnosed as suffering from congestive heart failure and was sent to Methodist Hospital Central where, several days later, successful bypass surgery was performed. The surgery, however, did not alleviate the effect of the earlier heart attack, which Boburka's expert testified had been occurring during Boburka's second visit to the emergency room on Aug. 18, and which caused significant injury to Boburka's heart muscle. Trial testimony revealed that this injury significantly reduced Boburka's longevity and endurance.

At the close of the plaintiff's case, Dr. Adcock moved for a directed verdict. The motion was denied. After presenting his own case, Dr. Adcock again moved for a directed verdict and the motion was again denied. The district court then instructed the jury and provided it with a special verdict form. After several days of deliberation, which included a supplemental instruction, the jury returned a verdict for Boburka in the amount of $600,000.

The appellate court concluded that Dr. Adcock was entitled to a directed verdict. The case was reversed and remanded with an entry of judgment for the defendant.

Discussion. Here again is a case where the physician defendant won the case because the plaintiff expert didn't give the case the elements needed and was not specific or definitive on the damages. However, when you look at how many times this patient with known IHD, who is carrying around his own ECG, came to the ED, the diagnosis of chest wall pain is not acceptable practice. The patient's diagnosis of chest wall pain was diagnosed made without an ECG, in spite of known history of IHD. A second visit for chest pain always should raise your level of concern.

Conclusion

In each of these legal cases, the failure to anticipate and play safe was the key, not the failure to obtain any particular study, since there is no magic bullet and all cases of IHD can have many confusing presentations. The clinical skills and sagacity of the emergency physician are essential to avoiding missed diagnosis of IHD.

Consider IHD in certain patient populations, always rule out IHD in chest pain cases, and think about atypical IHD as typical. Play it safe with chest pain. Everybody looks smart in the morning.

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CME Questions

1. Ischemic heart disease is:
   A. a mismatch of oxygen with heart muscle and conduction or pacer tissue.
   B. caused by a number of factors, including narrowing, obstruction, stress, and/or abnormal vessels.
   C. is clinically expressed as heart failure, arrhythmia, and risk to life.
   D. All of the above

2. The 12-lead ECG, when used for diagnosing IHD in the ED:
   A. has low sensitivity for IHD.
   B. is rarely non-specific.
   C. is difficult to perform.
   D. always detects occult IHD.

3. The gold standard for assessing cardiac injury is:
   A. echo resting and stress.
   B. serial CPK-MBs.
   C. thallium.
   D. myoglobin.

4. If antacid therapy relieves chest pain in the ED, IHD safely can be ruled out.
   A. True
   B. False

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Proposed Regulations Change Face of EMTALA

In May, the Centers for Medicare and Medicaid Services released proposed changes to The Emergency Treatment and Labor Act (EMTALA). Since its inception, many questions and concerns have been raised regarding EMTALA's changing practice patters, confusion resulting from inconsistent enforcement, and variation between regulatory and case law interpretation.

To clear up these inconsistencies for health care professionals, American Health Consultants offers a compact disc of EMTALA Update 2002, its successful audio conference presented by Charlotte Yeh, MD, FACEP, and Nancy Brent, RN, MS, JD, nationally recognized speakers on EMTALA. The anticipated date for the new rules to go into effect is Oct. 1. Don't miss out on important information to bring your facility into compliance. The cost of the CD is $249, which includes 1 nursing contact hour or 1 AMA Category 1 CME credit for each member of your staff. To order this invaluable teaching tool, please call American Health Consultants' customer service department at (800) 688-2421, or order online at www.ahcpub.com.

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