
Introduction

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WE HAVE SEEN IMPRESSIVE ADVANCES in the field of heart disease in recent years. In terms of prevention, we now have a much clearer picture of the pathogenesis and growth of atherosclerotic plaques. And a host of predisposing genetic and environmental factors have been identified. These include not only the well-known risk factors—diabetes, hypertension, smoking, stress, sedentary lifestyle and the disorders of lipid metabolism—but also elevated levels of lipoprotein (a), homocysteine and C-reactive protein.

The vulnerable plaque is now a focus of many investigators. These soft plaques appear to be smoldering, volcano-like excrescences that can rupture and release thromboplastic mediators that lead to clot formation and that may occlude the vessel lumen. Our new knowledge has led to the extensive and successful use of antiplatelet as well as anticoagulant therapies for the patient with an acute cardiac ischemic syndrome. It has also led to new theories about the role of inflammation and perhaps occult infection in the process of plaque softening and rupture.

Recent studies have consistently favored the use of angioplasty over thrombolysis for acute myocardial infarction. This is consistent with prior findings of the lack of evidence for a role for thrombolysis in non-ST elevation infarction and other ischemic variants. Consequently, there is now a strong tendency to provide the services of the catheterization laboratory for these patients. As this process has evolved, some hospitals without cardiac surgical programs have had to offer invasive cardiologic services for the patient with acute ischemic cardiac disease. Several trials have shown that these services can be safely provided at these sites as long as they have a coordinating center that offers emergency cardiac surgery as a backup.

Although the last update to the emergency care guidelines for basic life support (BLS) and advanced cardiac life support (ACLS) was issued in 2000, many providers are still struggling to interpret and implement the new material. The new ACLS medications that were added are arginine vasopressin and amiodarone. And vasopressin either replaces or is used with epinephrine for cardiac arrest. Amiodarone is used for unconvertible and recurrent ventricular fibrillation and for treating ventricular tachycardia, for which it supersedes procaine amide.

For the first time, the BCLS guidelines include the bystander application of closed chest compression without ventilation. Some evidence shows that during the early phase of resuscitation, ventilation is not entirely necessary. It has also been shown that early chest compression cardiopulmonary resuscitation (CPR) is vastly better than delayed perfusion. (This guideline was also a response to the understandable but unfortunate reluctance of both professional and lay rescuers to perform mouth-to-mouth ventilation.)

Recently we have participated in the patient access defibrillation (PAD) trials, in which residents of large apartment complexes and people in other institutions received CPR training with or without an automated external defibrillator program (AED). Patient survival was the dependent variable. Unfortunately, there was only a small increase in survival in the studied populations. So the presence of an AED in these settings was not effective as a public health measure. But the demographics of these patients help explain the lack of efficacy; in our study, the victims of sudden death in the community were mainly the elderly living alone.

There has also been a great deal of progress in our understanding and treatment of the patient with heart failure. We now have a more complete understanding of the remodeling process that the heart undergoes prior to and during heart failure. It is clear that many of the

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homeostatic mechanisms affecting the cardiovascular system are in themselves ultimately destructive to normal functioning. The remodeling of the heart's fabric has a major impact on electrical-mechanical coordination of contraction. The hypertrophy and fibrosis that is associated with remodeling also decreases the compliance of the heart. The decreased compliance in turn interferes with left ventricular diastolic filling and, in the case of hypertrophic cardiomyopathy, can also obstruct left ventricular ejection.

Neurohormonal responses of the heart in failure are both homeostatic and pathophysiologic factors presenting major new foci for management. The first medication that seems to counter the vasoconstriction and salt and water retention associated with chronic and acute heart failure is brain natriuretic peptide, a natural vasodilator and diuretic. Whether this parenterally administered agent has a long-term effect in management still remains to be determined. But it is clear that the issue of unloading the heart is a key management strategy that can be fulfilled pharmacologically.

The use of elective left ventricular pacing to "resynchronize" the heart is another recent treatment modality for managing congestive heart failure (CHF). Initially, this procedure was used for hypertrophic cardiomyopathies, but it has been found that many patients with cardiac hypertrophy have abnormal excitation pathways that reduce the efficacy of ejection. Some but not all of these patients have a widened QRS pattern or frank bundle branch blocks. A number of studies have now shown that resynchronization therapy can increase the ejection fraction and cardiac indices of many patients with severely depressed cardiac function.

While death from acute myocardial infarction has been reduced to approximately 8% of hospitalized patients, the incidence of sudden death in the community has not changed significantly. Some of these deaths are due to intracranial hemorrhage, pulmonary embolism and cardiomyopathies, but the vast majority are caused by dysrhythmic events due to ischemic heart disease. However, the use of antidysrhythmic medications has diminished be-

cause a majority of these drugs, while decreasing ectopic rhythms, have not led to improved outcomes and may be proarrhythmic. Amiodarone is the only notable exception to this statement, as its use has accelerated for both atrial and ventricular rhythm disturbances. Radio wave ablation and the use of implantable defibrillators have replaced antidysrhythmic medications, with significantly better outcomes.

Because of the rapid growth of our knowledge base and therapeutic capabilities, this is an exciting time in the field of emergency cardiac care. The emergency department is one of the clinical hubs where these advances are made available to patients. These patients may be critically ill on arrival or at risk to deteriorate and become unstable if proactive interventions are not instituted. Emergency physicians must be trained and able to provide emergency cardiac care to this diverse patient population, which includes individuals with acute myocardial infarction and the more common presentation of acute coronary syndrome. Physicians must be prepared to treat patients with acute or chronic heart failure and a host of acute and chronic rhythm disturbances. They must know how to help patients with problems related to implanted devices. They may also need to assess patients with valvular heart disease presenting with both preoperative and postoperative problems. They will be seeing more patients with heart transplants, who will be presenting with problems related both to graft rejection and intrinsic recurrent vascular disease in the transplanted heart.

As emergency physicians, we can best help and support these acutely ill and complex patients by maintaining our cardiac knowledge base at a very high level and by working closely with our colleagues, the cardiologists and cardiothoracic surgeons. It is our intention that this issue of the *Journal* will present a critical update of those recent advances in cardiology that have been introduced into emergency departments. These are some of the advances about which we will need an in-depth understanding in order to evaluate and treat our patients and communicate effectively with our colleagues.