Special Feature: Management of Acute Myocardial Infarction: Introduction

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**Introduction**

Acute myocardial infarction (MI) is a major health care problem in the United States, with an estimated 1.5 million cases each year.

In the 1960s, patients with suspected acute MI were hospitalized in general ward and placed at bed rest for approximately three weeks. At that time, therapy was aimed toward healing and prevention of cardiac rupture.

In the 1970s, coronary care units (CCUs) were introduced, with the main goal to monitor and treat arrhythmias and mechanical complications within the first few days postinfarction. This appeared to reduce the in-hospital mortality by approximately 50%, from 30% to 15%. However, during those years, little was done to limit the infarct size or to prevent its recurrence.

In 1980, DeWood et al (1) demonstrated conclusively that an occlusive thrombus was present in most patients with an acute MI who presented 4 to 6 hours after the onset of symptoms. This finding supported the “ulceration-thrombosis” theory which can explain the acute coronary syndromes.

Experimental and clinical data showed that acute MI can be aborted or the damage significantly reduced by reperfusion of the infarct-related artery segments if done within hours after the onset of infarction. During the 1980s, large-scale trials (2) demonstrated conclusively that medical interventions and thrombolytic agents in particular can reduce the infarct size and, more importantly, decrease mortality as much as 40% in a selected group of patients presenting with acute MI. However, the success of these interventions in limiting infarct size and improving prognosis was critically dependent upon the time from the onset of symptoms to intervention. Although thrombolytic agents are extremely effective in reducing mortality in patients presenting with an acute MI, this form of therapy is underutilized (3). Less than 25% of patients with an acute MI receive this therapy (4). According to Muller and Topol (5), “The full potential of thrombolytic therapy to alter the natural history of acute myocardial infarction can be realized only through the continued evaluation of selection criteria and the identification and treatment of the greatest possible number of eligible patients.” A recent editorial (6) in the *Journal of the American College of Cardiology* states, “Perhaps the two greatest impediments to rapid reperfusion therapy are the patient’s misinterpretation or denial of symptoms that prevents him or her from seeking help and the delay from hospital admission to initiation of treatment.” It is also clear that the delay could be greatly reduced if therapies were instituted by paramedics in the field. Findings from the Prehospital Administration of t-PA Study (PATS) suggest that this is feasible, safe, and effective when compared to conventional protocols (personal communication, Dr. D. O. Williams). Clearly, a team approach including the patient, paramedic, and Emergency Department (ED) nurse and physician, with backup by the cardiologist and cardiac catheterization laboratory and input from CCU nurses, pharmacy, and laboratory is the only way to streamline care and contribute to the successful and optimal management of a large percentage of patients with an acute MI.

This issue of the *Journal* on the management of acute MI is a reflection of the team approach at Henry Ford Hospital in treating the acute MI patient. Many of the contributors to this issue are members of the recently created Management of Acute Myocardial Infarction (MAMI) group. This group is comprised of nurses (ED, CCU) and physicians (ED, CCU, catheterization laboratory) from within the Henry Ford Health System who come into contact with the acute MI patient. Through periodic meetings, the MAMI group has the goals of improving access, communication, and education and providing optimal care for patients with acute MI.

In this issue, Drs. A. B. Levine and T. B. Levine explore an interesting and relatively new pathophysiologic topic, i.e., the neuroendocrine responses to an acute MI, suggesting that blunting of excessive neurohormonal activity with angiotensin-converting enzyme inhibitors and β-adrenergic blocking agents may decrease complications (i.e., infarct expansion) and improve survival.

Dr. H. N. Sabbah and colleagues suggest that significant recovery of regional and global left ventricular function may occur soon after acute MI. The rate at which this recovery takes place is related to the amount of stunned myocardium that occurs during the early phases of acute MI. This is an important aspect, because various therapies or interventions (7) may facilitate this recovery and result in better stratification and improved prognosis.

Dr. C. C. Foreback acknowledges that the creatine kinase (CK) MB assay is the most sensitive and specific laboratory test available for diagnosing acute MI. The newer CK-MM and CK-MB isoforms will probably allow us to diagnose acute MI in the ED setting and assess its size and ascertain if reperfusion occurred spontaneously or with thrombolytic agents.
The role of echocardiography is addressed by Dr. M. Alam, who stresses the importance of this tool in selecting therapy, particularly in complicated acute MI patients. For example, when heart failure complicates the acute MI, echocardiography will identify the pathophysiological mechanism for heart failure (i.e., mitral regurgitation, ventricular septal defect, right ventricular infarction, large scar, etc.).

Dr. A. R. Gokli and colleagues review a very important topic: the prehospital care of the acute MI patient. They are exploring the feasibility, safety, and efficacy of administering thrombolytic agents in the field. Given the prospects of the development of newer thrombolytic agents that may be more effective and safe when compared to available agents, it is likely that in the not too distant future the aggressive management to limit the infarct size will be initiated by paramedics in the field or in the patient's home.

Psychological factors relevant to the prehospital and in-hospital phases of acute MI are reviewed by Dr. L. W. Kenyon and associates. The authors conclude that psychological factors are strongly associated with prognosis during each phase of acute MI. More importantly, they suggest that the evaluation of patients at risk to develop an acute MI could allow early identification and appropriate intervention for those at risk from a psychological point-of-view of excessive delay in responding to symptoms of acute MI. Their work was recently published in Circulation (8) and received great attention by the American Heart Association.

Dr. B. L. Walters examines the issues confronting the ED physician in using thrombolytic agents. Without a well-organized and efficient emergency care system, any attempt to introduce a program of early thrombolytic therapy will be ineffective and possibly unsuccessful.

Dr. S. M. Jafri reviews the role of antiplatelet therapy and intravenous, subcutaneous, and oral anticoagulant therapy in the acute and long-term management of acute MI patients. Newer drugs currently under study may be better than aspirin or warfarin in interfering with platelet adhesion to injured vessels.

Dr. S. Goldstein discusses the pivotal role of β-adrenergic blocking agents in the current therapy of patients with acute and chronic coronary heart disease. This topic is particularly important, for despite the proven benefits of β-adrenergic blocking agents, these agents are underutilized in favor of therapies of uncertain value, such as calcium channel blockers.

Dr. S. Borzak reviews the role of intravenous nitroglycerin in acute MI, concluding that this therapy is safe and well tolerated. However, he acknowledges that its prophylactic use in all patients with uncomplicated infarctions remains controversial.

In the article on calcium channel blockers, I suggest that dihydropyridines, such as nifedipine or nicardipine, should not be used during the acute phase of MI for secondary prophylaxis. Diltiazem or verapamil may be used for secondary prevention when there are contraindications for β-adrenergic blocker therapy. However, all calcium channel blockers are contraindicated when the postinfarction course is complicated by significant systolic dysfunction.

Dr. J. E. Tisdale concludes that, based on currently available data, the disadvantages of routine prophylactic administration of lidocaine appear to outweigh potential advantages when used in acute MI patients.

Dr. C. R. Webb provides a rationale for the management of tachyarrhythmias during the acute and postinfarction phases, stressing the importance of precipitating or exacerbating noncardiovascular factors for arrhythmias. He acknowledges that there is no proof that chronic suppression of even complex ventricular arrhythmias with antiarrhythmic agents prolongs survival in the postinfarction patient. On the contrary, effective suppression of ventricular ectopy in postinfarction patients by type IC antiarrhythmic drugs is associated with increased mortality.

The Henry Ford Hospital experience with percutaneous transluminal coronary angioplasty (PTCA) is reviewed by Dr. S. K. Sharma and colleagues. The authors conclude that the main indication for PTCA is residual ischemia in the recovery phase of the acute MI, or primary PTCA in patients who have a contraindication for thrombolytic therapy.

The role of revascularization in patients presenting or developing cardiogenic shock is reviewed by Dr. A. R. Moosvi and associates. Based on the Henry Ford Hospital experience as well as that of others (9), acute mechanical revascularization should be considered in patients with acute MI who present with severe heart failure or shock, as no apparent benefit has been demonstrated from thrombolytic agents in this group.

Drs. A. R. Dresdale and G. Paone suggest that emergency revascularization for postinfarction angina, evolving acute MI, cardiogenic shock, and failed PTCA can be performed in selected patients with good results. In addition, they stress that surgical treatment of mechanical complications in acute MI, such as free wall rupture, ischemic mitral insufficiency, and ventricular septal defect, should be undertaken on an emergency basis.

D. L. Crimaldi Adams and S. R. Perez review the nursing care aspects of acute MI. The nurse plays a key role in implementation of therapies in limiting infarct size and in the secondary prevention. The authors offer practical suggestions for quality patient care delivery in the CCU.

Drs. S. Borzak and H. S. Rosman review the pathophysiology, natural history, risk assessment, and treatment of the non-Q wave MI patient. Given the heterogeneity of the non-Q wave MI patient population, firm recommendations for therapy must await future trials.

In our article on incomplete versus complete MI, Dr. S. Goldstein and I stress the importance of recognizing patients with an incomplete infarction based on clinical criteria. In the era of thrombolytic therapy, electrocardiograms may not be sufficient in identification of patients with an incomplete acute MI.

Dr. M. Lesch addresses the issue of risk stratification, suggesting that "the challenge of the future is not only to prolong the life of the patient with poor left ventricular function and arrhythmia but also to identify why and who of the ostensibly healthy post-MI patients will experience early morbidity or mortality."

Collectively, these 20 articles clearly demonstrate that our group at Henry Ford Hospital is committed to providing optimal care for all acute MI patients in the era of early intervention. The
A team approach is the key component to the successful management of these patients.

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References