## Managing the Patient With Possible Acute Myocardial Infarction

Gordon A. Ewy, MD Tucson, Arizona

T he evaluation of patients with actual or suspected acute myocardial infarction is assuming increasing importance since prompt intervention to limit myocardial infarction size by increasing myocardial blood supply,<sup>1-5</sup> decreasing the myocardial metabolic demand,<sup>6-9</sup> or both have been shown to decrease not only the mortality but also the complication rate of patients with acute myocardial infarction. Patients with acute myocardial infarction need to be identified promptly and accurately if these potentially valuable interventions are to be safely utilized.

Of equal concern is the increasing cost of caring for the patient with chest pain of possible cardiac origin-the "rule-out" myocardial infarction patient.<sup>10-12</sup> Two decision-support tools are evaluated by Green and Smith in this issue of The Journal.<sup>12</sup> This study was retrospective, using chart review, and contained a relatively small number of patients. Acknowledging these limitations, the study may help to validate the value of Pozen's heart disease predictive instrument (HDPI) in a rural setting. A major concern is that this report may be used inappropriately by decision makers who control hospital and physician reimbursement to conclude that all "rule-out" myocardial infarction patients can be managed in a hospital setting without electrocardiographic monitoring and without skilled nursing supervision, as such an approach would have obvious economic advantages. As Green and Smith indicate, decision-support tools are not to be confused with decision-making algorithms. The appropriately trained physician's clinical judgment cannot be replaced by a set of written guidelines.

A major problem with this and similar studies is the evolving role of the coronary care unit. Coronary care units were initially established in the 1960s so that patients at high risk of sudden cardiac death were located in a confined area. In some hospitals these early coronary care units were run by the anesthesiologist, as the major func-

tion was to facilitate the resuscitation of patients with cardiac arrest. To help with the early recognition of ventricular fibrillation, electrocardiographic monitoring was provided. Monitoring patients with acute myocardial infarction led to the conclusion that "death while sudden, is not unannounced." In the 1970s the emphasis of the coronary care unit was directed toward prophylactic therapy of warning arrhythmias, the prevention of arrhythmic deaths, and the treatment of the complications of acute myocardial infarction. In the late 1970s it was realized that almost all complications of acute myocardial infarctions were related to infarct size, and attention was focused on attempts to limit myocardial infarction size. These efforts were of limited success until the 1980s, when studies of intravenous  $\beta$ -adrenergic blocking drugs and of acute thrombolytic therapy were reported.<sup>1-9</sup> In the late 1980s the emergency room and the cardiac catheterization laboratory have become important adjuncts to the coronary care unit, for if infarction is prevented or aborted, there are few complications to treat.<sup>1-9</sup> In the late 1980s it is not always acceptable to watch the natural history of coronary occlusion and to treat the complications that result therefrom. Many hospitals do not have coronary care units-they have intensive care unit beds that are used as coronary care units and for rule-out myocardial infarction patients; this environment is all too often, as Green and Smith state, "noisy, not conducive to sleep, tends to induce disorientation (and results in sedation or restraint), and lacks privacy."12

A potential problem with the retrospective study reported here by Green and Smith is that it did not appear to consider the very different management of the patient with unstable angina. It is now clear that unstable angina is not the result of the slow progression of atherosclerosis of the coronary arteries; rather, it is most often the result of ulceration or rupture of the fibrous cap of such lesions, resulting in platelet thrombi and intermittent coronary occlusion.<sup>13–16</sup> Patients with unstable angina treated with antiplatelet agents (aspirin), heparin anticoagulation, or thrombolytic agents are less likely to develop thrombosis, infarction, and complications.<sup>17–20</sup> Any plan for managing the rule-out myocardial infarction patient, therefore, must

From the Department of Medicine, Section of Cardiology, University of Arizona College of Medicine, Tucson, Arizona. Requests for reprints should be addressed Io Dr. Gordon A. Ewy, Department of Medicine, Section of Cardiology, University of Arizona College of Medicine, Arizona Health Sciences Center, 1501 N. Campbell Ave, Tucson, AZ 85724.

take into consideration the subset of patients with unstable or preinfarction angina so that appropriate therapy can be instituted. In many of these patients the only finding on admission is a history suggestive of coronary disease. The patient may not have an infarction during hospitalization, but unless correctly identified (for example, with immediate electrocardiograms when discomfort recurs in the middle of the night), he may be discharged only to have infarction or sudden death.

The study by Green and Smith points out a weakness in the heart disease predictive instrument (HDPI) should this instrument be used to determine the probability of complications of the rule-out myocardial infarction patient. This weakness is the lack of appropriate consideration given to the presence of congestive heart failure. Patients without prior clinical heart disease will usually do well following a small- to moderate-sized myocardial infarction, especially when no other area of the myocardium is in jeopardy, and might be safely managed out of the coronary care setting. Yet that same amount of myocardial damage to the patient with congestive heart failure might result in the progression to cardiogenic shock,<sup>21</sup> a situation in which intensive care is needed.

Coronary care units are expensive but have life-saving capabilities. Because of the potentially fatal nature of myocardial infarction, physicians have tended to err on the side of admitting patients with suspected infarctions. The litigious nature of our society may perpetuate this tendency. Our challenge is to develop a system for handling the rule-out myocardial infarction patient in a setting that is not so costly and labor intensive as the intensive care setting, yet in an atmosphere where optimal diagnostic and therapeutic interventions can be applied quickly. Studies such as the one by Green and Smith may help in this effort.

## References

- Kennedy JW, Ritchie JL, Davis KB, et al: Western Washington randomized trial of intracoronary streptokinase in acute myocardial infarction. N Engl J Med 1983, 309:1477
- Sheehan FH, Mathey DG, Schofer J, et al: Effect of intervention in salvaging left ventricular function in acute myocardial infarction: A study of intracoronary streptokinase. Am J Cardiol 1983; 52: 431–438
- Verstraete M, Bernard R, Borg M, et al: Randomized trial of intravenous recombinant tissue-type plasminogen activator vs in-

travenous streptokinase in acute myocardial infarction. Lancet 1985; 1:842-847

- GISSI Trial. Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986; 1:397–401
- White HD, Norris RM, Brown MA, et al: Effect of intravenous streptokinase on left ventricular function and early survival after acute myocardial infarction. N Engl J Med 1987; 317:850–855
- Hjalmarson A, Elmfeldt D, Herlitz J, et al: Effects on mortality of metoprolol in acute myocardial infarction: A double blind randomized trial. Lancet 1981; 2:823–827
- The International Collaborative Study Group: Reduction of infarct size with the early use of timolol in acute myocardial infarction. N Engl J Med 1984; 310:9
- Gold HK, Leinbach RC, Maroko PR: Propranolol-induced reduction of signs of ischemic injury during acute infarction. Am J Cardiol 1976; 38:689
- Gold HK, Leinbach RC, Harper RW: Usefulness of intravenous propranolol in predicting left anterior descending blood flow during anterior myocardial infarction. Am J Cardiol 1984; 54:264
- Pozen MW, D'Agostino RB, Selker HP, et al: A predictive instrument to improve coronary care unit admissions practices in acute ischemic heart disease. N Engl J Med 1984; 310:1273
- Fineberg HV, Scadden D, Goldman L: Care of patients with a low probability of acute myocardial infarction: Cost effectiveness of alternatives to coronary care unit admission. N Engl J Med 1984; 310:1301–1307
- Green L, Smith M: Evaluation of two acute cardiac ischemia decision-support tools in a rural family practice. J Fam Pract 1988; 26:627–632
- Davies MJ, Woolf N, Robertson WB: Pathology of acute myocardial infarction with particular reference to occlusive coronary thrombi. Br Heart J 1976; 38:659
- Bresnahan DR, Davis JL, Holmes DR, Smith HC: Angiographic occurrence and clinical correlates of intraluminal coronary artery thrombus; role of unstable angina. J Am Coll Cardiol 1985; 6:285
- Capone G, Wolf NM, Meyer B, Meister SG: Frequency of intracoronary filling defects by angiography in angina pectoris at rest. Am J Cardiol 1985; 56:403
- Sherman CT, Litvack F, Grundfest W, et al: Coronary angioscopy in patients with unstable angina pectoris. N Engl J Med 1986; 315:913
- Telford AM, Wilson C: Trial of heparin vs atenolol in prevention of myocardial infarction in intermediate coronary syndrome. Lancet 1981; 1:1225
- Lewis HD, David JW, Archibald DG, et al: Protective effects of aspirin against acute myocardial infarction and death in men with unstable angina. Results of a Veterans Administration Cooperative Study. N Engl J Med 1983; 309:396–403
- Cairns JA, Gent M, Singer J, et al: Aspirin, sulfinpyrazone, or both in unstable angina. N Engl J Med 1985; 313:1369–1375
- Gold HK, Johns JA, Linbach RC, et al: A randomized, blinded, placebo-controlled trial of recombinant human tissue-type plasminogen activator in patients with unstable angina pectoris. Circulation 1987; 75:1192–1199
- Page DL, Caulfield JB, Kastor JA, et al: Myocardial changes as sociated with cardiogenic shock. N Engl J Med 1971; 285:133-137