

EDITORIAL

Detection of Acute Myocardial Infarcts by Infarct-Avid Imaging

In this issue of the *Journal*, Orlandi et al. demonstrate that technetium ^{99m}Tc -glucaric acid (^{99m}Tc -glucaric acid) may be used to detect acute myocardial infarcts in canine models with temporary coronary artery occlusion followed by reperfusion (1). In these studies, ^{99m}Tc -glucaric acid showed increased affinity for necrotic myocardial tissue as detected by triphenyltetrachloride staining and by gross inspection of the myocardium in animals in whom several days of reperfusion had been allowed. An apparent advantage of ^{99m}Tc -glucaric acid imaging was the lack of marked liver and osseous structure uptake by the infarct-avid imaging agent. Thus, ^{99m}Tc -glucaric acid joins several other previously demonstrated useful compounds for infarct-avid myocardial imaging and the detection of acute myocardial necrosis (2-12).

Some question the need for additional means to detect myocardial infarction (MI) in humans. Selected physicians argue that the availability of electrocardiographic methods and enzyme measurements in addition to careful clinical histories allow the identification of virtually all MIs. Others suggest that for those MIs small enough not to be detected by the traditional methods mentioned above, one can simply proceed with more aggressive evaluation at the correct time, including coronary arteriography or some form of stress testing with myocardial perfusion, metabolic or functional assessments using scintigraphic, echocardiographic, rapid

speed CT, or, perhaps, magnetic resonance imaging or spectroscopy methods in the future.

After two decades of providing care for patients with coronary heart disease, we remain convinced of the need to have several alternative means for infarct detection in patients. Many patients with transmural or Q-wave MIs are easily detected by their history and associated typical electrocardiographic changes and enzyme alterations. In patients with nontransmural (generally non-Q-wave MIs), the electrocardiogram is never specific for the presence of MI, but the clinical history and assessment of enzyme alterations often allows a diagnosis of MI to be made or excluded. However, there are patients in whom traditional techniques simply do not suffice for infarct detection and in whom a diagnosis is uncertain. These patients include the following: (a) patients with left bundle branch block; (b) patients with several previous MIs so that the electrocardiogram shows infarct patterns in several different regions; (c) immediately following open heart surgery, including those with coronary artery bypass procedures; (d) patients who delay their hospital admission for hours to days after the onset of symptoms suggestive of possible MI; and (e) patients who are resuscitated following sudden death. In these individuals, the electrocardiogram is sometimes unable to provide the necessary information and/or enzyme alterations are nonspecific or do not occur because of temporal limitations related to the timing of MI. However, the ability to make an accurate diagnosis is important and directly influences the timing of future diagnostic evaluations, including stress testing, de-

veloping a plan for limiting physical activity following a potential MI, the patient's ability to acquire insurance, timing the return to work, and generally addressing patient concerns. In these and other similar circumstances, the availability of sensitive, specific, and relatively noninvasive means to detect, localize, and estimate the size of acute MIs provides an important diagnostic and prognostic advantage.

The animal model used by Orlandi et al. was one with a temporary coronary artery occlusion followed by reperfusion. Permanent coronary artery occlusion with persistent reductions in coronary blood flow are a more stringent test of the potential clinical utility of an infarct-avid imaging agent since the delivery and accumulation of compounds with affinity for irreversibly injured myocytes occurs more slowly, often requiring hours to days (4,6). It is important to demonstrate the utility of any compound proposed as a myocardial imaging agent for the detection of myocardial necrosis with and without reperfusion since many acute infarcts occur with coronary artery occlusion without reperfusion. Thus, the ultimate utility of an agent proposed for clinical use in MI detection needs to be established in experimental animal models with permanent coronary artery occlusions as well as those with temporary coronary artery occlusions followed by reperfusion. It will be important to learn more about the ability of ^{99m}Tc -glucaric acid to concentrate in areas of irreversible cellular injury with permanent coronary artery occlusion in relevant animal models in the near future.

At least two myocardial scintigraphic techniques have proven use-

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