Editorial Comment

Nuclear Magnetic Resonance (NMR) Imaging in Ischemic Heart Disease*

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Identification of infarcted myocardium by nuclear magnetic resonance (NMR) imaging. Early in vitro studies (1,2) showed that the NMR relaxation times of infarcted tissue were altered, and images of excised hearts demonstrated that infarcted tissue could be discriminated from normal myocardium (2). Electrocardiographically gated NMR images of the intact heart in animals also depicted regions of increased signal intensity corresponding to the site of acute myocardial infarction (3-5). The increased signal intensity could be detected as early as 30 min after coronary occlusion in some animals and was uniformly recognized by 3 to 4 h after occlusion. The appearance of high signal intensity on spin-spin (T2)-weighted images and the prolongation of the T2 relaxation time occurred even earlier when the ischemically injured region was reperfused (6).

The identification of acute myocardial infarctions by NMR imaging in patients was reported in 1985 (7). Several subsequent studies (8,9) confirmed these observations, although one report (10) suggested that the finding of regions of increased signal was nonspecific and might not be distinguishable from motion-induced artifacts. Recent observations (11) indicate that both sensitivity and specificity for the identification of the site of acute infarction are improved by strongly T2-weighted NMR images. The report in the current issue of Journal of the American College of Cardiology (12) found that, by using specific objective criteria, the regions of infarction could be identified in all 20 patients with complete NMR studies.

The present study: accuracy of NMR imaging for measuring infarct size. The study by Johns et al. (12) indicates that the size of the infarcted region defined from measurement of the volume of the infarcted region of the myocardium on NMR images showed a reasonably good correlation with the volume of the infarct, as calculated from the area of the left ventricle displaying abnormal wall motion on contract ventriculography. It should be noted that the NMR method defined the morphologic volume of the infarct, whereas left ventriculography estimated the functional extent of the acute infarction. Prior studies (13) in animals using two-dimensional echocardiography have revealed that the functional extent of the infarct is larger than the actual infarct size at postmortem examination. Studies in animals (14,15) have revealed a very close correlation between the infarct size determined from the high signal region of the myocardium on NMR images and the actual volume of the infarct directly measured at postmortem examination. The estimation of infarct size, as defined by electrocardiographically gated NMR imaging was accurate for both 1 week and 3 week old infarcts (15). In that study (15), the standard error of the estimate for NMR imaging compared with postmortem measurements of infarct size was approximately 1.6 g. The animal studies, in which actual infarct volume can be measured directly, suggest that the NMR morphologic estimation of the infarct size is very precise when compared with actual infarct size as defined by postmortem examination. Consequently, the study of Johns et al. (12) may have underestimated the precision of NMR imaging for quantitating myocardial infarction by using a reference standard that depends on functional assessment of infarct size.

Clinical application of infarct sizing. It is now possible to estimate infarct size by a variety of imaging techniques including thallium scintigraphy, two-dimensional echocardiography, computed tomography and NMR imaging. This capability has been in existence for several years, but has not been clinically utilized to any extent. The lack of clinical application of infarct sizing suggests that 1) quantitation of acute infarctions is not recognized to have critical importance in the management of acute myocardial infarction, or 2) the imaging procedure or the measurements are too cumbersome and, therefore, unrealistic in patients with acute infarction. The latter consideration is real because NMR imagers are usually located far from coronary care units and not readily available for emergent studies. In addressing the former consideration, the measurement of infarct size in patients could be generically useful as a method to evaluate interventions directed toward salvaging myocardium. To employ a noninvasive technique for this purpose requires that the technique must not only measure morphologic infarct size, but also define the volume of myocardium jeopardized by an acute coronary occlusion. The ratio of infarct volume to jeopardized volume can be used to assess the effectiveness of therapeutic interventions. The accurate measurement of the jeopardized volume has not been readily achieved in patients by the several nonin-
vasive imaging techniques. Recently, the jeopardized area after acute coronary occlusion in animals was demonstrated in studies using both paramagnetic and magnetic susceptibility contrast agents with NMR imaging (15–17).

Future directions. The effectiveness of NMR imaging in ischemic myocardial disease will probably depend on the development and adaptation of magnetic resonance contrast media effective in defining regional deficiency in myocardial perfusion and the volume of myocardium jeopardized by an acute coronary occlusion. Several NMR contrast media have been shown (16–18) to distinctly demarcate the jeopardized region after acute coronary occlusion in experimental animals. Depending on the contrast medium applied, the jeopardized zone is shown to be a region of low intensity (negative image) compared with normal myocardium (16,17) or to have greater intensity (positive image) compared with normal myocardium (18). The former effect is produced by paramagnetic contrast agents, and the latter effect by magnetic susceptibility agents. Studies in animals have also indicated the capability of NMR contrast media to 1) distinguish between occlusive and reperfused myocardial infarctions (19); 2) discriminate between reversible and irreversible myocardial injury (unpublished observations); and 3) detect regions of reduced myocardial flow when used with dipyridamole.

It seems likely that the role of NMR imaging in ischemia will be advanced by the clinical introduction of contrast media with specific attributes favorable for myocardial imaging. The report of Johns et al. (12) should provide impetus to the evaluation of acute infarction with NMR imaging, but widespread utilization of such imaging will probably depend on the ability of the technique to detect regional ischemia and document reperfusion of infarction. These latter capabilities will require NMR contrast media.

References


