Large myocardial infarction (MI) in the left ventricle (LV) leads not only to expansion of the necrotic infarct zone, but also to compensatory remodeling throughout the remainder of the LV. This is shown in the Figure (left), which was acquired from a 73-year-old man who presented with NYHA class II congestive heart failure 7 years after an anterior MI. Cardiac MRI, as described in the legend, revealed a thin-walled pseudoaneurysm of the apical anterior wall and severe systolic dysfunction in the rest of the myocardium. These findings were confirmed at surgery (pseudoaneurysm repair and 2 coronary artery bypass grafts).

The genetic mechanisms underlying LV remodeling as a result of a large MI can be identified by studying transgenic and knockout mice. However, studies of chronic MI in genetically manipulated mice have been impeded by the technical challenges of inducing myocardial infarction in small animals (<35 g) and assessing cardiac structure and function at such rapid heart rates (>500 bpm). To address these problems, 6 C57BL/6N mice (28 to 30 g) were anesthetized with pentobarbital and intubated for a reperfused 2-hour occlusion of the major left anterior descending coronary artery. Four weeks later, each mouse was anesthetized, and cardiac MRI was performed. As shown in the Figure (right) and in the accompanying cine images (found at www.circulationaha.org), the LV remodeling and aneurysm formation characteristic of clinical heart failure after MI can be replicated with a high degree of fidelity in mice using these techniques. Thus, the combination of cardiac MRI and murine models of chronic MI should prove valuable in elucidating the role of specific genes in the pathophysiology of LV remodeling after MI.
Of Mice and Men . . . and Broken Hearts
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Circulation. 2001;104:e110
doi: 10.1161/01.HC4601.098067
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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