The ultimate goal of all cardiovascular clinical research should be to reduce the morbidity and mortality from cardiovascular diseases at an acceptable cost. In no area of medicine has clinical research led to a greater change in medical care than in the management of acute myocardial infarction (AMI). We now try to accomplish early coronary reperfusion in all ST-elevation MIs and administer a combination of drugs in the postinfarction period to halt or slow the progressive remodeling process that leads to heart failure and shortened survival. Clinical trials have documented the dramatic efficacy of these therapeutic interventions on morbidity and mortality.

The old nontherapeutic approach was not so long ago. During my internship in Boston in 1956, my first admission was a middle-aged man with an acute transmural anterior wall MI. After ministering to him for his 4-week hospital stay, which was mandatory in those days, he went home stable, only to return in 6 months with fulminant heart failure that took his life. What happened to him in that 6 months was mysterious to me, as well as to my Harvard attending physicians, who viewed the patient’s course as a rather uninteresting response to heart damage. It was 30 years before the concept of structural remodeling became recognized and effectively treated.

Chin et al in this issue of Circulation explore changes in the course of patients like mine as a consequence of our new aggressive therapeutic approach. They used a complete national sample of AMI hospitalizations in nearly 3 million Medicare fee-for-service beneficiaries, with their focus on hospitalization for heart failure within 1 year after the index infarction and the mortality in the year after this heart failure hospitalization. My patient had an adverse outcome on both endpoints. Certainly with our new insights and tools for intervention, we must have greatly reduced the morbidity and mortality in this group of patients.

However, clinical trials are not clinical practice. The patients may differ, the drugs may not be administered as they were in the trials, and the patients may stop the medication in the absence of the oversight of study personnel. The unanswered question is whether the benefits found in these trials are reflected in the real world of health care. If not, we should spend more time and effort resolving the paradox than in doing more clinical trials.

The good news is that Chin et al demonstrated a progressive decline in the readmission rate for heart failure. The bad news is that the decline was precisely small, not nearly the magnitude that we might have expected from the clinical trial data, and the mortality within 1 year after rehospitalization was not reduced over this time period. On first look, these data would appear to suggest that the real world of clinical practice has not benefited from our mechanism-based interventions nearly to the extent that we would have expected.

However, the design of the study and its metric for measuring efficacy may be partially at fault. The baseline for the study was 1998, more than a decade into the era of reperfusion therapy and antiremodeling pharmacotherapy. These aggressive approaches to management certainly have become more widely used in the past 15 years, but perhaps the 1998 data were already heavily influenced by efforts to salvage myocardium and inhibit structural remodeling.

There are other problems with interpreting the data. The diagnosis of AMI in the Medicare database may not distinguish between ST-elevation, coronary occlusion MIs, and nonocclusive non-ST-elevation MIs. Our therapeutic revolution has been in ST-elevation, coronary occlusion MIs, which respond to reperfusion and antiremodeling therapy. Changes in the course of non-ST-elevation MIs may be less dramatic, and they may represent a substantial fraction of the population evaluated.

The metric used by the investigators was hospitalization for heart failure within 1 year after their hospitalization for AMI. Hospitalization is an increasingly attractive endpoint because it is not only costly to the healthcare system, but it reduces quality of life and is associated with a higher subsequent mortality. It is becoming a metric of choice for hospitals and health care systems. However, hospitalizations compete with death as an endpoint. Death in the first year after the infarct, which was not distinguished in this study, would preclude future hospitalization. Furthermore, enhanced survival likely preserves a population with more cardiac damage and more advanced atherosclerotic disease that places them at enhanced risk for subsequent heart failure. This later likelihood is borne out by the Medicare database, which reveals the increasing age and worsening renal function in the hospitalized cohort over time. Thus, the heart failure hospitalization metric may not be a useful guide to the...
effectiveness of contemporary therapy and the mortality after this hospitalization not an adequate test of our improved management of heart failure over the past 15 years.

What the data do demonstrate is that, once a patient suffers from an MI, he or she is likely eventually to succumb to their cardiovascular disease. Under these conditions, healthcare costs will continue to rise because prevention of early death will lead to enhanced downstream costs. The expensive interventional programs to deal with the acute phase of MI do not necessarily prevent subsequent progression of disease nor the population risk for heart failure (Figure).

The message from these data should be that prevention of MI should receive at least as much attention as treatment for MI. Hospitals and interventional cardiologists generate robust revenue from efforts at early reperfusion. No one would advocate for a return to the days of passive management that characterized my training in Boston in 1956, but not enough attention has been devoted to prevention. Admittedly, it does not generate much revenue, for either doctors or hospitals, but appropriately targeted pharmacopreventive therapy saves lives. By evaluating individuals for early functional and structural abnormalities of the cardiovascular system, it is possible to identify the phenotype of early disease likely to lead to morbid events, such as AMI. Intervention with statins, inhibitors of the renin–angiotensin system, aspirin, and other preventive therapies in such patients would prevent the AMIs that lead to such a dismal prognosis. It is sad that so many individuals who are at clear risk remain untreated with these effective agents.

Chin et al have made it clear that our contemporary management strategies for AMI have not solved the real-world problem of mounting morbidity, mortality, and costs of cardiovascular disease. Prevention of AMI is possible. Whether by a polypill to be administered to every adult or an evaluation process to identify early disease to intervene with individualized management, the agenda should be clear to any thoughtful observer. We should continue to use our best methods for treatment once people get sick, but we must mount a new and more effective effort at identifying risk and intervening to keep people free of progressive symptomatic disease.

Disclosures
Dr Cohn has an equity position in Cardiovascular Centers, whose goal is to establish prevention centers elsewhere, and is the principal investigator on a research grant funded by Forest Research Institute.

References


Key Words: Editorials ■ heart failure ■ hospitalization ■ myocardial infarction
The Message Is Clear: Prevent as Well as Treat Acute Myocardial Infarction
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In the article by Cohn, “The Message is Clear: Prevent as Well as Treat Acute Myocardial Infarction,” which published in the December 17, 2013 issue of the journal (Circulation. 2013;128:2554–2556), three corrections are needed:

1. On page 2554, the sentence, “The diagnosis of AMI in the Medicare database may not distinguish between ST-elevation, coronary occlusion MIs, and nonocclusive non-ST-elevation, coronary occlusion MIs” should read, “The diagnosis of AMI in the Medicare database may not distinguish between ST-elevation, coronary occlusion MIs, and nonocclusive non-ST-elevation MIs.”
2. On page 2554, the sentence, “Changes in the course of non-ST-elevation, coronary occlusion MIs may be less dramatic, and they may represent a substantial fraction of the population evaluated” should read, “Changes in the course of non-ST-elevation MIs may be less dramatic, and they may represent a substantial fraction of the population evaluated.”
3. On page 2555, the sentence, “It is sad that so few individuals who are at clear risk…” should read, “It is sad that so many individuals who are at clear risk…”.

The current online version of the article has been corrected.