

The End of Cardiology and the Curing of Medicare?

J. Philip Smith, M.D.
Karen Cosper, R.N.

A revolution in cardiac care is developing that may save Medicare hundreds of billions of dollars.

Recent advances in imaging technology and combination pharmacologic interventions are changing our concepts of cardiovascular disease and improving our therapies.^{1,2} These advances portend a significant improvement in quality of care and reduction of costs.

Surgically Driven Technology

Historically, the primary cardiovascular therapy has been surgical, based on the development of cardiac angiography and its images of discrete luminal incursions with limitations to blood flow.³ Focal discrete lesions can be bypassed or stented. Since Dr. DeBakey performed the first bypass in the 1960s, about half a million coronary artery bypass grafts are now performed each year in the U.S. The idea of a progressive, relatively focal lesion impinging on the lumen seemed very amenable to anatomical correction.³

Over the last decade a different model of the disease has emerged.⁴ Arterial disease is now seen as a diffuse inflammatory process of the arterial wall that is mediated by T-cells and macrophages. Modern thought believes the process occurs throughout the arterial system, and that its pathology resembles that of rheumatoid arthritis in its method of destruction of the connective tissue in the endothelium.⁴ The wall gradually weakens, and then suddenly collapses into the lumen, generating thrombus and occlusion (see Figure 1).⁴

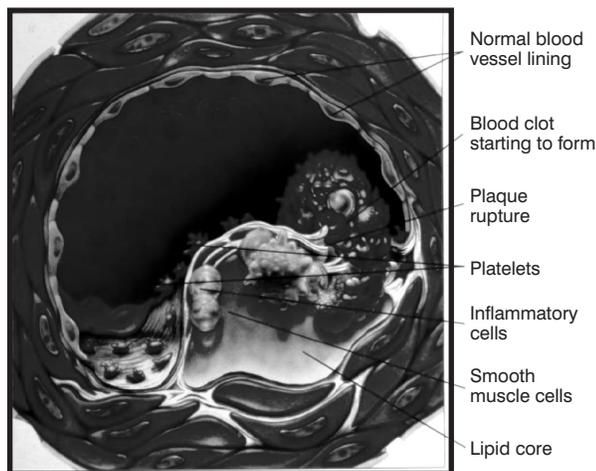


Figure 1. Most heart attacks occur as a result of small, unstable plaques that break open and lead to the formation of blood clots that suddenly block circulation to the heart.²⁻⁴

Plaque is the build-up of lipids (fat), cell debris, smooth muscle cells, and collagen within the artery wall.

Our understanding of this process, while still incomplete, has been improved by the new technique of intravascular ultrasound, an ultrasound catheter inserted into the coronary artery that gives a two-dimensional real-time view of the arterial wall.⁵ The atheroma, or area of endothelial injury, can be examined directly and reinvestigated as necessary. Thus, an active real-time biological marker allows us to measure our medical interventions. Previously, no technique was available to study the arterial wall in vivo.³ We relied on inference, guess, and hope that risk factor reduction would result in better outcomes.

Atherosclerotic disease and its treatments were discovered only by indirect methods: stress test, nuclear imaging, and arteriography. These tests are costly, open to subjective interpretation, and often wrong, with a high level of false positives and negatives.³

Previously, our therapeutic interventions in coronary artery disease could only be investigated by “outcome studies” requiring large numbers of participants and many years of follow-up. A positive result to a therapy often took five years to prove with statistical methods.⁶ This limitation has been a major impediment to our understanding of best practices. Outcome studies are time-consuming and expensive to perform. Often only one or two key questions can be addressed with each study.

Cost-Reducing Technology

Today, the ability to measure the disease directly is improving our knowledge base and may soon start to reduce the cost of the disease significantly.⁷ Rather than a fixed, inexorably progressive process, the acute inflammation of the arterial wall now appears to be mutable and amenable to therapeutic alteration.^{5,8,9} In one recently published study, regression of the lesions could be demonstrated within six weeks of using pharmacologic therapies.⁵

New, noninvasive imaging techniques are also emerging.^{10,11} Both computerized tomography (CT) and magnetic resonance imaging (MRI) may soon be able to delineate the interior structural elements of the arterial wall noninvasively and in real time (see Figure 2). It is hoped that these technologies will reduce the cost of

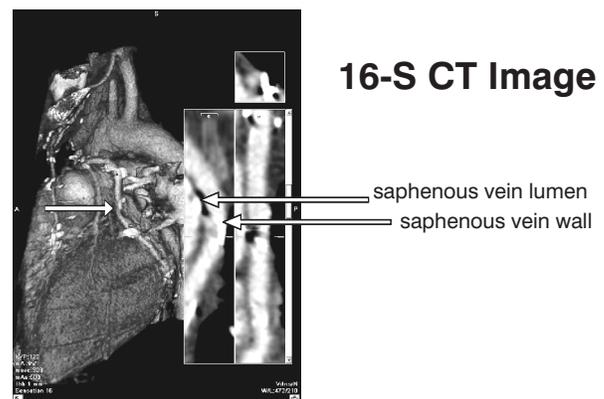


Figure 2. The saphenous vein graft to the circumflex artery is highlighted in this 16-s CT image of the author’s myocardium. In the images to the right, the lumen and walls of the saphenous vein graft are demonstrated.

cardiovascular care and improve results. In the future, expensive interventional and indirect inferential measurements will seldom be necessary.

Directly screening, diagnosing, and effectively treating the arterial wall early in the disease will make our current model of cardiovascular care obsolete. In most cases, medical therapy may completely supplant surgical intervention,¹¹ thus greatly reducing costs for a better outcome.

Effective cure will trump limited care. In the 1950s, tuberculosis was a surgical disease.¹² An elaborate hospital system with highly specialized care had been established in the developed world for TB therapy. Patients were placed in special hospitals, not just to prevent the spread of the disease, but for surgical care of diseased lungs. Many operations were developed to collapse the diseased portion of the lung.¹² Surgical techniques improved healing with a 70 percent cure rate, as opposed to 35 percent untreated.¹³

In the early 1950s, antibiotics were proposed to cure TB. For a decade these pharmacologic efforts failed until triple antibiotic therapy, which prevented the development of resistant organisms, achieved a 99 percent cure rate.¹³ By the mid-1960s an entire hospital system was eliminated.¹⁴ The pharmaceutical interventions cured the disease and collapsed the costs.

Arterial disease appears to be following a similar pattern. Lifestyle interventions with exercise and dietary changes are both effective and inexpensive.¹⁵ Cholesterol lowering with statins provides significant, sustainable, safe reductions in serum lipid levels. Moderate reductions in LDL cholesterol levels have been associated with a 25 to 35 percent reduction in relative risk of vascular events such as stroke and heart attack.⁶ Aggressive lowering of LDL cholesterol reduces the risk even further.^{1,5,8,9}

Platelet disaggregation has also shown relative risk reductions of 25 percent or greater.¹⁶ Beta blockade during and after myocardial infarction reduces mortality by close to 25 percent,¹⁷ and recently angiotensin converting enzyme (ACE) inhibitors have been reported to add an additional risk reduction of close to 25 percent.¹⁸

These interventions, when combined, are synergistic; risk reduction rates of 80 percent are achievable.² Thus, multi-interventional pharmacologic therapy reduces disease occurrences by more than 80 percent by interrupting the inflammatory destruction of the arterial wall at many points. The cost of the pharmaceutical care is much less than current interventional therapies.¹⁹ Additionally, pharmacologic expense will only decline, as many drugs are about to lose their patents. Identifying those patients at risk and directly targeting a cure may result in an outcome similar to the historical example of TB.

Medicare's cost crisis is largely the result of suboptimal, expensive care. The costs are illustrative. The total cost of cardiovascular disease in 2000 was \$326 billion, and pharmacologic costs are only 9 percent of the total.^{19,20} The difference was essentially the institutional costs of care. Medicare takes the current costs and extrapolates them into the future, factoring in demographic data. This approach allows no calculation for technological advancements or cures. It assumes the status quo and guarantees it, if mechanisms for technological improvements are discouraged.

Limitations on inducements to invest in new technologies may preserve the costly status quo and limit new cures. Cost controls on pharmaceuticals, and restricting investigative techniques by third parties or government, slow the progress to better outcomes.

Curing disease should be our policy goal. We have achieved this in the past, and it is our only solution for the future.

J. Philip Smith, M.D., is a pulmonologist in private practice. Address: 670 Rio Lindo Ave., Suite 300, Chico, CA 95926. Telephone: (530) 899-7120. E-mail: jpsmith670@cchcgroup.com. **Karen Cosper, R.N.**, is a diabetes educator and cardiovascular specialist.

REFERENCES

- 1 Topol EJ. Intensive statin therapy—a sea change in cardiovascular prevention. *N Engl J Med* 2004;350:1562-1564.
- 2 Yusuf S. Two decades of progress in preventing vascular disease. *Lancet* 2002;360:2-3.
- 3 Topol EJ, Nissen SE. Our preoccupation with coronary luminology—the dissociation between clinical and angiographic findings in ischemic heart disease. *Circulation* 1995;10:92:2333-2342.
- 4 Virmani R, Kolodgie FD, Burke AP, Farb A, Schwartz SM. Lessons from sudden coronary death—a comprehensive morphological classification scheme for atherosclerotic lesions. *Arterioscler Thromb Vasc Biol* 2000;5:1262-1275.
- 5 Nissen SE, Tsunoda T, Tuzcu EM, et al. Effect of recombinant ApoA-1 milano on coronary atherosclerosis in patients with acute coronary syndromes—a randomized controlled trial. *JAMA* 2003;11:2292-2300.
- 6 Heart Protection Study Collaborative Group. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high risk individuals: a randomized placebo-controlled trial. *Lancet* 2002;360:7-22.
- 7 Nissen SE, Yock P. Intravascular ultrasound: novel pathophysiological insights and current clinical applications. *Circulation* 2001;103:604-616.
- 8 Cannon CP, Braunwald E, McCabe CH, et al. Intensive versus moderate lipid lowering with statins after acute coronary syndromes. *N Engl J Med* 2004;350:1495-1504.
- 9 Nissen SE, Tuzcu EM, Schoenhagen P, et al. Effect of intensive compared with moderate lipid-lowering therapy on progression of coronary atherosclerosis—a randomized controlled trial. *JAMA* 2004;291:1071-1080.
- 10 Schoenhagen P, Tuzcu EM, Stillman AE, et al. Non-invasive assessment of plaque morphology and remodeling in mildly stenotic coronary segments: comparison of 16-slice computed tomography and intravascular ultrasound. *Coron Artery Dis* 2003;5:14:459-462.
- 11 Rehwald WG, Chen EL, Kim RJ, Judd RM. Noninvasive cineangiography by magnetic resonance global coherent free precession. *Nat Med* 2004;10:545-549.
- 12 Fraser RG, Paré JAP, Paré PD, Fraser RS, Genereux GP. Infectious disease of the lungs. In: Fraser RG, Paré JA. *Diagnosis of Diseases of the Chest*. Vol. II. Philadelphia, Pa: W.B. Saunders; 1989.
- 13 Crofton JW, Douglas A. *Respiratory Diseases*. Oxford, England: Blackwell Scientific Publications; 1969.
- 14 Texas Department of Health. History of TB. Available at: <http://www.tdh.state.tx.us/TB/history.htm>. Accessed Aug. 4, 2004.
- 15 Sesso HD, Paffenbarger RS Jr., Lee I. Physical activity and coronary heart disease in men: the Harvard alumni health study. *Circulation* 2000;8:102:975-980.
- 16 Anti-thrombotic Trialists' Collaboration. Collaborative metaanalysis of randomised trials of anti-platelet therapy for prevention of death, myocardial infarction, and stroke in high-risk patients. *BMJ* 2002;1:324:71-86.
- 17 Yusuf S, Peto R, Lewis J, Collins R, Sleight P. Beta blockade during and after myocardial infarction: an overview of the randomized trials. *Prog Cardiovasc Dis* 1985;27:335-371.
- 18 Heart Outcomes Prevention Evaluation Study Investigators. Effects of an angiotensin-converting enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. *N Engl J Med* 2000;342: 142-153.
- 19 Jensen T. Proofs of prototypes for sale: the tales of university licensing. In: NIH Response to the Conference Report Request for a Plan to Ensure Taxpayers' Interests are Protected; 2001:7.
- 20 American Heart Association. Heart and Stroke Statistical Update 2000.