CORRESPONDENCE

Coronary–Coronary Bypass with a Segment of Internal Thoracic Artery to Revascularize the Right Coronary Artery

To the Editor:

We read with great interest the paper by Korkmaz and colleagues,[1] in which the authors describe right coronary artery (RCA) revascularization with internal thoracic artery (ITA) segments, using the coronary–coronary bypass grafting (CCBG) technique. We congratulate them for achieving the 2nd-greatest reported series of CCBG surgery (after Nottin and associates[2]), as well as for their meticulous search of the literature on CCBG.

The selection of an appropriate conduit and surgical technique to bypass a low-grade lesion (50%–60%) of the RCA remains a challenge for cardiac surgeons. We ourselves have considered,[3] in theory, the use of the CCBG technique in such circumstances. It was therefore a great pleasure to read that Korkmaz and co-authors have used this approach in 6 patients with low-grade lesions (their Group III). However, we do not understand why, in 4 of those patients, the bypassed lesions were nonsignificant (as little as 20%–25% stenosis). The authors' follow-up arteriograms revealed free ITA grafts that were patent (if slightly narrowed); yet we wonder if Korkmaz and colleagues are truly advocating the revision of guidelines for coronary revascularization, in regard to the grade of coronary artery stenosis that should be bypassed.

Further, there are very few articles in the medical literature that discuss the mid-term angiographic patency of arterial coronary–coronary conduits (CCC). Barboso and Rusticali[4] have reported follow-up angiograms in 2 patients (at 3 and 8 years after surgery), which showed patent arterial CCC (free left ITA segments) over distal lesions on the left anterior descending coronary artery (LAD). Both LADs had presented with proximal (grafted with in situ remnants of the left ITA) and distal lesions. We have recently reported a case in which we achieved perfect angiographic patency of a CCC (free left ITA segment) over a single, distal LAD lesion, 3.5 years after surgery.[5]

Korkmaz and associates calculate their mean time to follow-up angiography for 24 patients (50% of all cases) to be 16.5 ± 7.8 months (7 days to 2 years). This would be a nice contribution to the literature on mid-term angiographic patency follow-up of coronary–coronary arterial conduits incorporated into the right coronary artery system, were these data not misleading. The authors have reported that 14 of their patients underwent follow-up coronary angiography during the 1st postoperative week (7 days = 0.23 month) and that 2 others underwent follow-up angiography in the 2nd postoperative year (these angiograms were performed 13 to 24 months postoperatively). One other patient underwent follow-up angiography at 4 months, and the remaining 7 patients did so during the 1st postoperative year (these angiograms were performed 7 days to 12 months postoperatively). So the properly calculated mean time to follow-up angiography now appears to be only 5.8 ± 7.7 months (or even less). Thus, overall, this report presents only results that show short-term angiographic patency of coronary–coronary ITA conduits used to revascularize the right coronary system.

Because the progression of coronary artery disease at the site of proximal anastomosis of the CCC is of major concern, the authors have chosen the ostium or 1st segment of the RCA as their proximal anastomotic site in 38 patients (79.2% of cases): they have correctly observed that at those sites there is almost no progression of the disease in the natural course of arteriosclerosis. In their report of long-term angiographic follow-up data on that topic (maximum follow-up, 7 years), Nottin and associates[2] never observed the progression of coronary disease on a grafted RCA at the site of proximal CCC anastomosis. However, in the subsequent discussion of the Nottin and colleagues[2] article, Mills made the interesting point that Nottin's patients who had undergone follow-up angiography after 10 years were found to have RCA disease that had progressed to the ostia in nearly half of those cases (7 out of 15). It is very difficult to decide in retrospect whether these lesions are the consequence of natural progression of the disease or of medial reactivity and thickening promoted by arteriotomy and manipulation at the site of the proximal CCC anastomosis.

In conclusion, we would like to congratulate Dr. Korkmaz's team for their results and for their efforts to include the CCBG procedure in the armamentarium of cardiac surgeons.

Dusko Nezic, MD, PhD, FETCS,
Aleksandar Knezevic, MD, BcS,
Petar Milacic, MD, and
Miomir Jovic, MD, PhD,
"Dedinje" Cardiovascular Institute,
Belgrade, Serbia

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This letter was referred to Dr. Korkmaz, who replies in this manner:

When the 4 patients with nonsignificant lesions (20%–25% stenosis) were inspected during the operation, their lesions were seen to be yellowish, soft plaque with partial thrombus, which we judged to be prone to rupture and embolization. To eliminate the occlusion risk, we performed CCBG with a segment of ITA. Although CCBG was performed on vessels with nonsignificant stenosis, the postoperative angiograms revealed patent grafts.

Bruschke and colleagues[1] state that RCA atherosclerosis progresses chiefly in the first segment of the RCA and in the mid-RCA. Our experience supports this view. Therefore, when performing the proximal CCBG anastomosis in the atherosclerotic RCA, the surgeon should first choose the ostium (if it is free of calcification), and then make the distal anastomosis beyond the crux of the RCA. By performing the proximal anastomosis in the ostium, one can avoid the risk of atherosclerotic progression in the proximal RCA. If the patient has less extensive atherosclerosis in the RCA and the first segment is seen to be free of lesions, upon angiographic and intraoperative inspection, then CCBG anastomosis can be performed in any segment of the RCA. The distal RCA anastomosis site is also very important for long-term patency, which is why our preferred site is beyond the crux.

We thank Dr. Nezic and his colleagues for their keen attention. In our calculations, all our data were based on weeks, but in the text the mean duration of angiographic follow-up was mistyped as 16.5 months. We sincerely apologize for this mistake and agree with the correction.

Askin Ali Korkmaz, MD,
Department of Cardiovascular Surgery,
Florence Nightingale Hospital,
Istanbul, Turkey

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Mortality in Friedreich's Ataxia

To the Editor:

We read with interest the report "Friedreich's ataxia as a cause of premature coronary artery disease" by Giugliano and Sethi.[1] The article provides a review of many of the diverse cardiologic manifestations of Friedreich's ataxia (FA) and the overall heterogeneity of this disorder. In particular, the issue of coronary artery disease in FA has been understated in the neurological literature, and it provides one explanation for the occasional precipitous decline in cardiac function that we have noted in a few of the roughly 100 patients with FA whom we have evaluated over the past decade.

However, it is important to put the cardiac issues in FA within the context of the modern prognosis for the disorder. Although the exact mortality rate for patients with FA has not been investigated recently, we disagree with the authors' contention that most patients with FA die in the 3rd or 4th decade. Life expectancy in FA is affected by a variety of factors. First, better medical therapy has led to longer survival and improved quality of life for patients with FA. Second, patients with less-severe phenotypes have been identified since the advent of genetic testing. Third, we now recognize that a large number of patients with FA, even among those who carry more severe genetic abnormalities,[2-4] do not have significant cardiomyopathy. Because patients with FA generally do not develop dementia, they usually attend college, are employed as professionals and paraprofessionals, and lead lives that are successful and productive.

Over the next few years, new therapies for FA are likely to improve survival and quality of life even further. With such improvements, there will be opportunities for more aggressive therapies directed at repair of the cardiac dysfunction. Patients with FA have already received cardiac transplants on occasion and have had ventricular assist devices implanted chronically.[5,6] Whether to pursue such therapies in a specific patient will always be a decision best made by the individual patient and his or her physicians. Therefore, it is important for cardiologists to be able to provide advice on such decisions within the context of the modern medical prognosis for patients with FA.

David R. Lynch, MD, PhD, and
Jennifer M. Farmer, MS, CGC,
Departments of Neurology and Pediatrics,
University of Pennsylvania School of Medicine and The Children's Hospital of Philadelphia;
and
Robert B. Wilson, MD, PhD,
Department of Pathology and Laboratory Medicine,
University of Pennsylvania School of Medicine;
Philadelphia, Pennsylvania

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This letter was referred to Drs. Giugliano and Sethi, who reply in this manner:

We would like to thank Dr. Lynch and colleagues for their interest in our paper. They highlight several interesting issues with regard to patients with Friedreich's ataxia (FA).

First, we agree that coronary artery disease in FA may be under-recognized, but, more important, it is probably under-reported in the literature. Friedreich's ataxia is a rare disorder, and we encourage those with extensive experience with such patients to publish their findings, in order to expand physician awareness and to counter misconceptions about this disease.

Second, with regard to the mortality rate in FA, our findings were drawn from the available, peer-reviewed literature. Although there may be anecdotal evidence of improved mortality using a wide array of therapies in patients with FA and in selected subsets of FA clinics, the literature does not support any significant change in survival since the report in 2000 by Delatycki and colleagues,[1] in which the average age of death was at 37.5 years (range, 5–71 years). The wide range of life expectancy highlights the limited data and also the genotypic variability of this disorder.[1-4] Certainly, patients with less-severe phenotypes will likely outlive the more severely impaired patients. While there have been several advances in medical therapy that improve survival in patients with coronary artery disease and heart failure, it is unclear if this survival benefit will be realized in patients with FA. We agree that advances in medical therapy, most notably in the area of antioxidants, may lead to some improvement in neurologic function,[5] and to a delay in disease progression,[6] but there are still no definitive data on the mortality benefit. We could not identify any quality-of-life studies in patients with FA and cardiomyopathy, although it is conceivable that better management of heart failure and angina could translate into improved quality of life. The assertion by Dr. Lynch and colleagues that patients with FA "usually attend college, are employed as professionals and paraprofessionals, and lead lives that are successful and productive" may be the consequence of selection bias, tilted towards those patients who survived longer than the mean and had less-severe phenotypes, without cardiac compromise.

We recognize that some patients with FA have received cardiac transplants and left ventricular assist devices (LVADs), although these aggressive treatments may not be warranted. Because we do not understand the predictors of survival in patients with FA, it is difficult to justify at this time the allocation of limited and expensive resources such as cardiac transplantation, LVADs, and defibrillators. After all, it has only recently been demonstrated that LVADs improve survival in patients with congestive heart failure.[7]

It is true that if the prognosis and quality of life in patients with FA improve with innovative medical and gene therapy, the cardiology community will need to become proactive in the early diagnosis and management of the cardiovascular manifestations. To that end, further research needs to be directed towards identifying predictors or markers of survival, so that patients may be appropriately selected for destination therapy and for primary prevention of sudden cardiac death with automated implantable cardioverter-defibrillator therapy. Until that time comes, let us not lose sight of the simple and relatively inexpensive therapies, such as aspirin, statins, β-blockers, and angiotensin-converting enzyme inhibitors, which will improve heart failure and symptomatic angina.

Gregory R. Giugliano, MD, SM, and
Prabhdeep S. Sethi, MD, MPh,
Division of Cardiology,
Baystate Medical Center,
Springfield, Massachusetts

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The Amazing Adventures of a Heart Surgeon

To the Editor:

Having read Dr. Domingo Liotta's recently published book titled *Amazing Adventures of a Heart Surgeon. The Artificial Heart: The Frontiers of Human Life*, I was most delighted to read Dr. Haller's letter to the editor[1] and Dr. Cooley's postscript.[2] I wish to make some additional comments, not only because the book represents a major work of science and humanities in combination, but also because Dr. Liotta is a dear old friend of mine.

Dr. Liotta certainly has enjoyed a remarkable career, with many well-deserved honors and much recognition. He has eloquently recounted his story in this book. His recollections of the development of a left ventricular assist device with Dr. Michael E. DeBakey (Chapter 24) and of a total artificial heart with Dr. Denton A. Cooley (Chapter 25), both in Houston, Texas, are so thorough and unique that they should serve as extremely valuable historical records.

The chapter on Chou En-lai, the Chinese premier under Chairman Mao Tse-tung (Chapter 30), contains so many historically important details that they should be of interest to people outside the medical community as well.

The publication of Dr. Liotta's book came at an appropriate time, right after the February 2007 inauguration of the Liotta-Cooley artificial heart display at the Smithsonian's National Museum of American History in Washington, DC (Fig. 1). The display is a part of the museum's "Treasures of American History" exhibit. On 14 February 2007, the Smithsonian held a noon talk, "A Cure for the Broken Hearted: Artificial Hearts in America—An American Heart Month Event," that included a history of the Liotta-Cooley heart and of other artificial heart devices. For more information, visit the museum's Web site at http://americanhistory.si.edu or visit the marvelous exhibit itself, which establishes the Liotta-Cooley artificial heart as a worthy part of human history. Whether or not your readers are able to travel to Washington, DC, to visit the Smithsonian, I recommend Dr. Liotta's book to all physicians and surgeons who have an interest in the heart—real or artificial, broken or intact.

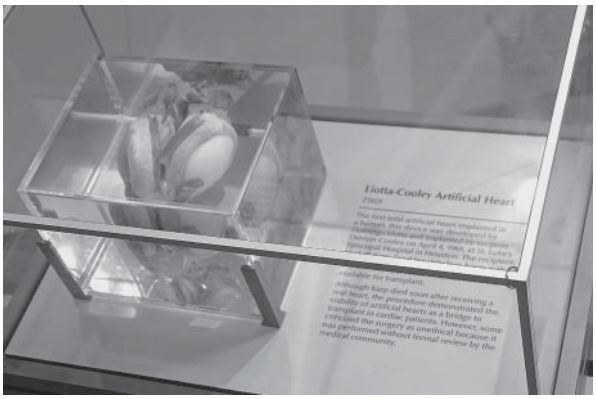

Fig. 1 *Two views of the Liotta-Cooley artificial heart, as displayed at the National Museum of American History, Washington, DC.*

(Photographs courtesy of J.M. Chelnick, Smithsonian Institution)

Tsung O. Cheng, MD,
Professor of Medicine,
The George Washington University,
Washington, DC

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