REPLY

We would like to thank Karamanoukian, Donias and Bergslund for their remarks regarding our randomized trial of percutaneous coronary intervention (PCI) versus coronary artery bypass graft (CABG) in patients with medically refractory myocardial ischemia and risk factors for early mortality with CABG. They raise two specific issues about our surgeons’ CABG methods: 1) variability in myocardial protection, and 2) use of “beating heart” or “off-pump” techniques.

Our trial was an attempt to answer a real-world clinical question that often seems to arise on nights and weekends: “What is the best means of revascularizing this high-risk patient who desperately needs more myocardial blood flow?” To make the trial clinically relevant in an era when techniques are evolving and strong differences of opinion exist, we settled on a “strategy” study. That is, both our surgeons and interventionists could use whatever tools and methods they believed would yield the best long-term outcome for their individual patients. Accordingly, the means of myocardial protection varied from center to center and even operator to operator. We obtained prospective data regarding such issues as antegrade versus retrograde, cold versus warm and crystalloid versus blood, specifically so that we could look at both temporal and spatial variations in technique and attempt to determine what, if any, influence those variations had on outcome. From the overall perspective, an in-hospital mortality of 4% for the oldest (mean age 67 years; >5 years older than any other revascularization trial), and sickest (mean left ventricular ejection fraction = 0.45; >0.12 lower than any other revascularization trial and ~1/3 patients within seven days of a myocardial infarction and ~1/3 patients with prior CABG: both high-risk exclusions from previous trials), suggests that we had excellent myocardial protection.

Regarding the issue of “off-pump,” although Karamanoukian and colleagues state that 1995 is when the method was popularized, their citation is dated 1997, and most of their references are electronic abstracts, dated 2000 and 2001, after AWESOME completed enrollment. This timing explains why we did not even collect data on the use of this method. Several of our study surgeons are among the planners of a proposed Veterans Affairs cooperative study, which we are told would randomly allocate patients between conventional and off-pump surgery. We look forward to that type of data. In the meantime, we must continue to make the difficult and often nocturnal decisions, in part, influenced by the awareness of which techniques each of us is most comfortable using.

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Perpetuation of the Myth of the Q-Wave Versus the Non-Q-Wave Myocardial Infarction

In a recently published study, Furman et al. (1) perpetuate the myth that the non-Q-wave myocardial infarction (MI) is a “distinctive pathophysiological entity from non-Q-wave MI (NQWMI).” The results and conclusions of that study deserve careful scrutiny.

The classification of Q-wave versus NQWMI was based on the presence or absence of Q waves 24 h after hospital admission. It has been documented that at least 10% of patients will develop Q waves between 3 to 11 days after MI (2). This error could well affect the results and conclusion. Even though the same definition was used throughout the study, it is not clear whether the potential error was unchanged throughout the study.

In addition, limiting the electrocardiogram (ECG) definition of Q-wave MI (QWMI) to a Q-wave of ≥0.04 s and an amplitude ≥25% of the R-wave in that lead (in addition to evolutionary ST- and T-wave changes) is a perpetuation of an older and now discarded definition of the pathologic Q-wave. In a recent consensus document (3) the ECG criteria for establishing an MI are “any Q-wave in leads V1 through V3, Q-wave ≥30 msec in leads II, III, aVL, V4, V5, or V6 (the Q-wave changes must be present in any two continuous leads), and be ≥1 mm in depth.” In addition, it was noted that criteria for Q-wave depth requires more research. Inevitably, many infarcts presenting with significant wide but shallow pathologic Q waves would be erroneously lumped by Furman et al. (1) in the NQWMI category. Thus, the criteria selected in the Furman et al. (1) study would tend to overestimate the number of patients with NQWMI. These problems in classification illustrate one of the difficulties in attempting to classify patients as Q-wave versus NQWMI.

Furman et al. (1) quote the criteria for an adequate study as outlined in our report (4) in this Journal in 1997 and they claim they corrected for the variables we listed. However, they failed to correct for one of the most important variables, and this omission can alter the results and conclusions. They recognized only the negative deflection in the first 40-ms vectors, or classic Q-wave, completely overlooking other depolarization abnormalities or “Q-wave equivalents” that have been well documented to correlate with MI. For example, ECGs that present the onset of tall R waves in leads V1 and V2 representing the “Q-wave” of posterior infarction would be classified as an NQWMI by Furman et al. (1). This error has plagued most studies in this field and is worth emphasizing. We quote from our previous publication in the Journal: “The process of infarction alters depolarization by a number of mechanisms, e.g., dispersion, slow conduction and localized block. These forces change the surface QRS in a number of ways: to suppose that they only cause negative deflections, or Q waves, is electrocardiographically naive. It is equally naive to confine attention to the initial 40 msec vectors since at least 8% to 10% of all infarcts involve the basal myocardium, which is depolarized during the middle or terminal vectors of the QRS. A tall R in the right precordium and localized R-wave diminution in the mid precordium are two obvious and accepted ‘Q-wave equivalents’ but there are others. QRS alterations correlated with infarction in a number of studies include R/S changes, acute frontal-plane right axis deviation, new left axis deviation, low voltage and QRS notching, precordial QRS notching, initial and terminal QRS notching, high-frequency notching in orthogonal leads and abnormally narrow precordial R waves. . . . Summing up