JACC Vol. 52, No. 4, 2008
July 22, 2008:315–8

Reply

We appreciate the comments from Drs. Roik and Opolski on our long-term observations in patients with cardiogenic shock who were enrolled in the GUSTO (Global Utilization of Streptokinase and Tissue-Type Plasminogen Activator for Occluded Coronary Arteries)-I trial (1). One of the important advantages of long-term datasets is that long-term data are available—along with the advantage comes the disadvantage that they are old data on patients followed up for a long time. That must be kept in mind because the GUSTO-I trial enrolled patients presenting with their index event between 1990 and 1993 from around the world, including Warsaw, Poland. Many things have changed since then in the field of acute myocardial infarction and cardiogenic shock. Just to name a few, we no longer use streptokinase; in fact, it is no longer even available in the U.S. for use during acute myocardial infarction. We now routinely use stents, sometimes even drug-eluting stents, and IIb/IIa inhibitors are now commonly given. Thus treatment strategies have changed dramatically.

The patients included in our long-term follow-up were all randomized because they had presented with acute infarction as part of the 41,021-patient cohort. We note that there are many subsets of patients with shock, but at the time of the initial GUSTO shock publication, this shock substudy was the largest in the literature. Undoubtedly some subsets have a worse prognosis than others, and work continues to optimize identification of higher-risk patients as well as to optimize their outcomes.

As previously documented in the GUSTO-I experience (2), 89% of these patients developed shock after admission using a definition of shock of "a systolic blood pressure <90 mm Hg for at least 1 h, not responsive to fluid administration, thought to be secondary to cardiac dysfunction, and associated with signs of hypoperfusion." The fact that the in-hospital mortality was 56% identifies that this indeed was a very-high-risk group of patients.

The most important points of our follow-up study are that: 1) we need better strategies for optimizing early outcomes of cardiogenic shock; and 2) as Drs. Hochman and Apolito pointed out in their accompanying editorial (3), after the initial storm, there is surprising calm.

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doi:10.1016/j.jacc.2008.03.056

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Driving and Ventricular Arrhythmia: A Historical Perspective

Albert et al. (1), in their important study on the risk of defibrillator shocks during driving, suggest that their study is "the first to examine the association between driving a car and the onset of ventricular arrhythmias." There were, in fact, a series of publications, albeit including only a small number of patients, published some 40 years ago and performed in patients with a spectrum of cardiac diseases driving in British traffic (2–4). These studies documented a remarkably high prevalence of ventricular arrhythmias and ST-segment shifts despite the lack of symptoms and even included 1 patient who developed sustained ventricular tachycardia (clearly illustrated in the article but misrepresented as sinus tachycardia) associated with the development of pulmonary edema requiring a hospital stay (4).

The apparent discrepancy between the high prevalence of driving-induced arrhythmia in the early studies and a low frequency of implantable cardioverter-defibrillator shocks in the high-risk population enrolled in the current study is striking. On the assumption that city driving in the U.S. in the 21st century is as least as stressful as it was in central London in the 1960s, one wonders whether the antiadrenergic effect of beta-blockade might account for these differences. However, reference to the authors' Table 2 indicates this not to be the case, at least in terms of malignant ventricular arrhythmias. The early studies had a high proportion of patients with active angina, whereas nowadays revascularization is the norm in ischemic heart disease. This difference in driving-related arrhythmia points to the powerful role of ischemia in triggering arrhythmias in susceptible patients, highlights how far we have come in the past 40 years, and underscores the enduring value of now-historic studies for gaining insight into potential mechanisms of disease. To paraphrase a well-known saying: "She who knows history may be destined to improve it."

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doi:10.1016/j.jacc.2008.01.075

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