malities of the mitral valve apparatus. Second, in such patients aggressive treatment with vasodilators should be avoided because they may precipitate or worsen the dynamic obstruction. In contrast, beta-blockers should blunt the hyperdynamic contraction and thus, in addition to the reduction of arrhythmias and work load, prevent the fatal cardiac rupture (4). We believe that discussion of these mechanisms could stimulate other investigators to look carefully at LVOT dynamics in patients with anterior myocardial infarction to determine whether compensatory hyperdynamic contractions of the basis of the heart may indeed be deleterious in these patients.

Jozef Bartunek, MD, PHD
Marc Vanderheyden, MD
Bernard De Bruyne, MD, PHD
Cardiovascular Center
Moorselbaan 164
9300 Aalst,
Belgium
E-mail: jozef.bartunek@olvz.aalst.be

PII S0735-1097(99)00479-9

REFERENCES

1. Becker RC, Hochman JS, Cannon CP, et al. Fatal cardiac rupture among patients treated with thrombolytic agents and adjunctive thrombin antagonists: observations from the Thrombolysis and Thrombin Inhibition in Myocardial Infarction 9 study. J Am Coll Cardiol 1999;33:479–88.
2. Oliva PB, Hammil SC, Edwards WD. Cardiac rupture, a clinically predictive complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993;22:720–6.
3. Bartunek J, Vanderheyden M, De Bruyne B. Dynamic left ventricular outflow tract obstruction after anterior myocardial infarction: a potential mechanism of myocardial rupture. Eur Heart J 1995;16:1439–42.
4. International Study of Infarct Survival (ISIS-1) Collaborative Group. Randomised trial of intravenous atenolol among 16,027 cases of suspected acute myocardial infarction: ISIS-1. Lancet 1986;2:57–66.

REPLY

The work of Bartunek et al. (1) is well known and respected in the field of echocardiography, and their elegant work concerning the mechanism of outflow tract obstruction after aortic valve replacement for aortic stenosis represents the definitive study on this subject. We are greatly interested in their more recent work concerning cardiac rupture in two elderly women with anterior myocardial infarction, dynamic outflow tract gradient and a documented ventricular septal defect in one patient and possible left ventricular free wall rupture in the other.

Their hypothesis of increased wall stress, occasioned by dynamic outflow obstruction due to hyperdynamic basal wall contraction as a possible trigger to cardiac rupture, is novel and plausible. We have recently observed an instance where outflow tract obstruction, as demonstrated by transesophageal echocardiography, was the cause for profound hypotension in a patient who had a large anteroapical myocardial infarction.

The pressure theory of cardiac rupture is widely quoted (2) and, in combination with a *vulnerable* myocardium, provides the required substrate for an often fatal event. Although our study did not have premorbid echocardiographic information (3), data derived from the Late Assessment of Thrombolytic Efficacy (LATE) study (4), National Registry of Myocardial Infarction (5) and Thrombolysis and Thrombin Inhibition in Myocardial Infarction (TIMI-9) experience (3) support Bartunek et al.'s hypothesis in the following ways. First, an anterior site of infarction was present in >50% of patients. Second, although a previous myocardial infarction was an independent risk factor for cardiac death, it was not common in patients with a fatal cardiac rupture. This observation suggests that preservation of left ventricular performance in the noninfarct zone is a prerequisite for rupture and is supported by the relatively low proportion of patients with cardiac rupture initially classified as Killip class III or higher (4). Lastly, the protective effect of beta-blockers is consistent with an intracavity pressure trigger for cardiac rupture. Despite the fact that angiotensin-converting enzyme (ACE) inhibitors and other vasodilators could potentially worsen a dynamic obstruction, similar to large-scale trials (6), we observed an inverse relation between ACE inhibitor use and the occurrence of cardiac rupture (odds ratio 0.27, $p < 0.0001$). Clearly, several mechanisms contribute to cardiac rupture.

We agree with Bartunek et al. that future investigations carried out among patients with myocardial infarction must include studies of dynamic change in ventricular cavity performance as a means to better understand the conditions required for cardiac rupture and, more importantly, its prevention.

Richard C. Becker, MD
Gerard Aurigemma, MD
University of Massachusetts Medical School
55 Lake Avenue
Worcester, Massachusetts 01655

PII S0735-1097(99)00482-9

REFERENCES

1. Bartunek J, Vanderheyden M, De Bruyne B. Dynamic left ventricular outflow tract obstruction after anterior myocardial infarction: a potential mechanism of myocardial rupture. Eur Heart J 1995;16:1439–42.
2. Becker RC, Pezzella AT. Myocardial rupture. In: Topol EJ, editor. Acute Coronary Syndromes. New York: Marcel Dekker, 1998:269–326.
3. Becker RC, Hochman JS, Cannon CP, et al. Fatal cardiac rupture among patients treated with thrombolytic agents and adjunctive thrombin antagonists. J Am Coll Cardiol 1999;33:479–87.
4. Becker RC, Charlesworth A, Wilcox RG. Cardiac rupture associated with thrombolytic therapy: impact of time to treatment in the Late Assessment of Thrombolytic Efficacy (LATE) study. J Am Coll Cardiol 1995;25:1063–8.
5. Becker RC, Gore JM, Lambrew C, et al. A composite view of cardiac rupture in the United States National Registry of Myocardial Infarction. J Am Coll Cardiol 1996;27:1321–6.
6. Ambrosioni E, Borghi C, Magnann B, for the Survival of Myocardial Infarction Long-term Evaluation (SMILE) Study Investigators. The effect of the ACE-inhibitor zofenopril on mortality and morbidity after anterior myocardial infarction. N Engl J Med 1994;332:80–5.