Winter and Cardiovascular Mortality

I read with interest the report by Sheth et al. (1) on seasonal variations of coronary heart disease and stroke mortality, and I would like to present a new analysis of data culled from an old Brazilian study performed in São Paulo, a city situated at the Tropic of Capricorn.

Between January 1932 and December 1941, Chiaverini and Rey (2) studied all deaths from congestive heart failure (CHF) confirmed by the city’s unique autopsy service. They concluded that most of the CHF deaths occurred during the winter, which in São Paulo runs from June to August. I examined the mortality data during a shorter period (July 1939 to December 1941), because there was better weather data, and confirmed Chiaverini and Rey’s results. In my analysis, CHF deaths were 28.8% higher during the winter months as compared with the summer months (December to February). Minimal monthly temperature (annual range 7.7 to 18.1°C) had a persistent association with CHF deaths ($r = 0.31$, $p = 0.0456$) after multiple linear regression analysis for humidity, hours of sunlight, temperature range and CHF mortality. These results from historic data are in concordance with a recent report analyzing coronary artery disease mortality in Hawaii (3), which is in the Northern Hemisphere. However, the most relevant finding from Chiaverini and Rey’s study is that most of the CHF cases were categorized as “nonatherosclerotic diseases.” During the 1940s in São Paulo (4), the etiology of CHF was rheumatic (23.6%), hypertensive heart disease (23.1%), atherosclerosis (20.6%), Chagas’ disease (10.8%) and syphilis (7.1%). Given these data, it is plausible to speculate that seasonal variations in cardiovascular mortality may be due to factors directly associated with impairment of myocardial contractility, such as arrhythmias and increasing blood pressure, rather than to factors more specifically related to rupture of coronary atherosclerotic plaque.

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Dynamic Left Ventricular Outflow Tract Obstruction as a Potential Mechanism of Myocardial Rupture After Acute Myocardial Infarction

We read with great interest the study of Becker et al. (1), who investigated the incidence and predictive factors of fatal cardiac rupture in patients treated with thrombolyis enrolled in the Thrombolysis and Thrombin Inhibition in Myocardial Infarction (TIMI-9) study. The authors reported female gender and age as independent risk factors for fatal cardiac rupture. Of note, they did not observe a significant relation between antiaggregation therapy and cardiac rupture. In contrast, the incidence of rupture was higher in patients with anterior myocardial infarction not receiving early angiotensin-converting enzyme inhibition and beta-blockers. Although the study did not address directly pathophysiological mechanisms of the rupture, the authors speculated that changes in collagen matrix associated with aging and, potentially, with female gender may underlie the higher risk for mortality. This sounds plausible, but nevertheless, the authors do not report some clinically relevant information such as the extent of coronary artery disease and overall or regional left ventricular (LV) function. Likewise, they do not address triggers that may precipitate the occurrence of the rupture. However, Oliva et al. (2) reported that myocardial rupture is often preceded by particular signs—namely emesis, restlessness, pericarditis, alterations of the T waves and abrupt episodes of bradycardia or hypotension. The latter signs deserve closer attention by clinicians. Hypotension or bradycardia is often present owing to the activation of LV mechanoreceptors in patients with LV outflow tract (LVOT) obstruction. We have recently reported a novel observation of a dynamic LVOT obstruction with systolic anterior motion of the mitral leaflets in patients after acute anterior myocardial infarction that preceded a cardiac rupture or intraventricular septal defect (3). The LVOT obstruction was observed in women with nonhypertrophied ventricles and calcified posterior mitral annulus and/or thickened mitral leaflets who presented with hyperdynamic contraction of the noninfarct-related artery segments. Since this publication (3), we observed a third myocardial rupture also preceded by LVOT obstruction in a 57-year-old man. Based on our observations, we postulated that the presence of the LVOT obstruction led to an increase in the end-systolic wall stress of the infarct segments, which may represent a direct mechanical insult to a weakened necrotic tissue. This observation has several potential clinical implications. First, it implies that in patients with a large anterior infarction and hyperkinetic noninfarct segments, particular attention should be paid to LVOT flow dynamics, especially in women with abnor-
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.

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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.

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