Winter and Cardiovascular Mortality

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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 located at an equivalent latitude as São Paulo, but 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 thrombolysis 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 anticoagulation 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 pathophysiologic 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-