The J-Point Revisited

To the Editor:

Oh, the diabolical J-curve! Will we ever resolve this issue? Recently, you published a posthoc analysis1 of data from theValsartan in Acute Myocardial Infarction Trial that showed, inter alia, that, after a myocardial infarction (MI), a sustained low blood pressure (BP; systolic BP <100 mm Hg on 2 of the post-MI visits at 1, 3, and 6 months) was a marker for bad cardiovascular outcomes. We were also told, incidentally, that this subset of patients had a significantly higher incidence of anterior MI and Q-wave MI and significantly higher maximum creatine kinase values during the MI than those who had normal BPsa t 1, 3, and 6 months. In my view, these differences are consistent with the idea of a larger volume of infarcted myocardium, which will do 2 things: increase the risk of cardiovascular death and also produce a greater degree of functional impairment with a lower systolic BP. It is reasonable for the authors to say, therefore, that low BP may be associated with adverse events, but the wrong impression may be generated that somehow the lower BP caused cardiovascular deaths.

The editorialists2 are right in saying that very low diastolic BPs may theoretically be associated with increased adverse events as follows: (1) because of ischemia from decreased myocardial perfusion; (2) because large artery stiffness is associated with increased pulse pressure and low diastolic pressures; or (3) as an epiphenomenon, related to an underlying illness, causing increased morbidity or mortality for other reasons. However, there is as yet no good evidence that the first or second events are practical problems in the management of hypertension in patients with ischemic heart disease. The posthoc analysis from the International Verapamil-Trandolapril Study3 is cited in both the analysis by Thune et al1 and the accompanying editorial2 as support for the existence of a J-curve and a warning against “excessive” lowering of the BP. However, what was not mentioned was that patients in that trial who had a BP <120/70 mm Hg (the level below which the risk of adverse outcomes seemed to rise) were older and had a history of more MI, coronary artery bypass grafting or percutaneous coronary intervention, stroke or transient ischemic attack, diabetes, heart failure, and cancer, a classic example of reverse causality. The comorbidities probably caused the low BP, not the other way around.

It is true that there is a lower limit to coronary autoregulation, and if the diastolic BP, which is the coronary perfusion pressure, goes below that limit, myocardial ischemia may occur. However, we do not have any idea what that lower limit of diastolic BP is in the human coronary circulation, with or without coronary artery disease. Both the epidemiological and clinical trial data, although indirect, have encouraged the American Heart Association to endorse more aggressive BP lowering to <130/80 mm Hg in patients with, or at high risk of developing, ischemic heart disease.4 The common sense caveat is that, in patients with an elevated diastolic BP and coronary artery disease with evidence of myocardial ischemia, the BP should be lowered slowly, and caution was advised in inducing falls of diastolic BP <60 mm Hg if the patient has diabetes or is over the age of 60 years. In older hypertensive individuals with wide pulse pressures, lowering systolic BP may cause very low diastolic BP values (<60 mm Hg). This should alert the clinician to assess carefully any untoward signs or symptoms, especially those because of myocardial ischemia.

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Disclosures
None.

References
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