0.26×10⁻³ versus 4.07±0.33×10⁻³, p<0.01) were significantly lower than those of normal control mice, and there was no significant difference in body weight (18.0±1.6 g versus 18.2±2.7 g, p=NS) between treated mice and normal controls. Thus, the effect of captopril in the later stage of coxsackievirus B₃ myocarditis reported by Rezkalla et al may merely reflect the drug effect itself without an effect on myocarditis. We believe that more work should be done before it can be concluded that captopril has a beneficial effect in the later stage of acute myocarditis.

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Reference

Reply
We appreciate the comments made by Matsumori et al on our article reporting the effect of captopril in acute murine viral myocarditis. While they agree that the beneficial effects are clear in the early treatment group, they express doubt concerning its beneficial effects in the late phase because it may be nonspecific. In fact, administration of captopril has been previously shown to decrease left ventricular mass in normal Sprague-Dawley rats and in patients with essential hypertension. In our study we reported a decrease in left ventricular mass when captopril was administered in the late phase; however, the study was not designed to address the mechanism(s) that might be involved. A reduction in left ventricular mass in a case of cardiomyopathy may be beneficial. Note that this reduction in this particular model was associated with a lesser degree of ventricular congestion in the late (day 30) study group. Of interest is that the reduction in heart weight in normal treated mice in the study conducted by Matsumori et al was a modest 12%, whereas in our study the reduction on days 6, 20, and 30 were 46%, 43%, and 36%, respectively. It is conceivable that the reduction observed in our study is a combination of a nonspecific effect of captopril on the heart plus a specific effect on hearts infected with coxsackievirus. We agree with Matsumori et al that this will need further studies and we did conclude in our manuscript that “further testing will be needed before a randomized human study can be considered.”

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References

Reversible Segmental Cardiac Dysfunction
In a recent issue of Circulation, van den Berg et al reported that coronary angioplasty (PTCA) improves abnormal segmental motion in patients with ischemic heart disease. The population studied consisted of 40 patients, 20 of whom had abnormal wall motion before PTCA; 13 had prior myocardial infarction, and six patients had multivessel disease. Interestingly, seven patients had unstable angina, whereas 14 patients had refractory angina. The term “refractory angina” was not defined. Wall motion was assessed semiquantitatively before and after PTCA.

Of 260 segments studied, 180 (69%) were normal before and after PTCA. Of 68 hypokinetic segments, 39 (57%) normalized after PTCA; 43% were unchanged. Of 12 akinetic segments, 10 became hypokinetic after PTCA. Data are not provided for nutritional perfusion, collateral network, or metabolism of hypokinetic and akinetic segments. Also, it is not clear what segments best reflected the diseased artery and how many regions were made up of infarcted myocardium. How were the data related to other factors contributing to abnormal wall motion, such as degree of subendocardial ischemia, percentage of myocardial scar, degree of inotropic stimulation, and transmural “tethering” to name a few, and what was the role of the functional border zone?

The discussion portion of this paper lumps together myocardial stunning and hibernation. Scientifically, there may be no basis for such grouping, because “myocardial hibernation” is still a term in search of a reality. Rahimtoola,4 who coined the word “hibernation,” has recently suggested that 1) the mechanism for hibernation is not known; 2) it is not known why severely ischemic hearts hibernate; 3) the modality that should be used to detect hibernation is not established; 4) it is not known how long the myocardium can hibernate, and 5) it is not known how soon after reperfusion is instituted the hibernating myocardium normalizes function.

At the present time, there is no experimental model to demonstrate the existence of hibernation. The experimental design of Matsuzaki et al5 of 5 hours of partial ischemia can hardly be considered a model for hibernating myocardium in the context used by Rahimtoola.4

In sum, many readers will probably wonder whether van den Berg et al can support their hypothesis of hibernating myocardium, because data are not presented to show changes in cardiac nutritional perfusion or metabolism necessary to entertain the possibility of past hibernation.

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