Troponin I Degradation and Myocardial Stunning
To the Editor:
The article by Feng et al.1 caused a revolution in current thinking regarding the pathomechanism involved in myocardial stunning, in that it irrefutably proved that there is no causal link between troponin I (TnI) degradation and the transiently depressed contractile function after a brief ischemic episode. However, does this fact imply that there is no answer to the question of why the myocardial force is depressed and its Ca\(^{2+}\) sensitivity is reduced?

Certainly, the concept of TnI degradation provided an attractive explanation for myocardial stunning.2 The existence of a link between truncated TnI molecules, altered myofibrillar Ca\(^{2+}\) reactivity, and overall myocardial function was firmly established on the basis of experiments with transgenic animals.3 TnI modulates myofibrillar Ca\(^{2+}\) sensitivity and is a substrate for Ca\(^{2+}\)-dependent proteases such as \(\mu\)-calpain. The idea of increased end-diastolic pressure and not ischemia/reperfusion as a trigger for TnI degradation provides new insight into the prevention of stunning.1 What remains is that an intracellular Ca\(^{2+}\) overload evokes stunning because calpastatin preserves the contractile function, irrespective of the origin of the Ca\(^{2+}\) overload.1 Accordingly, the proteolytic basis of myocardial stunning is preserved.

Perhaps proteins other than TnI have more to do with stunning. It is important to note that intracellular calpains damage a broad spectrum of muscle proteins and that regulatory and structural changes (e.g., desmin degradation) may reduce the apparent Ca\(^{2+}\) responsiveness of the contractile filaments.4 Thus, myocardial stunning might be a consequence of the intricate protein alterations5 that are encountered during an ischemic/reperfusion insult. If so, the task is to find the most significant ones and to disentangle them.

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