LETTERS TO THE EDITOR

Laser-Mediated Transmural Myocardial Channels Induce a Hyalin Degeneration of Neighboring Myocardium

Whittaker et al. (1 [Fig. 3]) assert that blood penetrates from laser-mediated transmural myocardial channels into neighboring interstitial tissue. One is tempted to assume that this penetration is made possible by damage to interstitial connective tissue by thermal injury and infarction. However, there are some histopathologic patterns present in Figure 3 that falsify this attractive hypothesis: 1) Some cardiomyocytes distant from the channel (e.g., in the upper left quarter) that are not thermally injured are also without birefringence, hinting at the action of still another pathologic factor. 2) A majority of the alleged red cells are not situated in the interstitium itself but are aligned along it in the space occupied previously by cardiomyocytes. This suggests that they are not genuine red cells (even if their admixture cannot be excluded) but "eosinophilic droplets" (2) produced by a fragmentation of hyalinized cardiomyocytes. 3) None of the usually recognized forms of cardiac cell death are followed by quick disappearance of necrotic tissue. In Figure 3, however, after an experiment lasting only 6 h, there are enlarged interstitial spaces and numerous adjacent cardiomyocytes fragmenting into eosinophilic droplets. In the upper left quarter, whole bundles of still recognizable cardiomyocytes without birefringence are in the process of fragmentation. Here and there, some myofibers have disappeared entirely, leaving behind "empty" spaces. All of this indicates that hyalin degeneration of myocardium induced by overload (3), able to destroy and eliminate cardiomyocytes in the course of 2 h (4), has taken place in this tissue. This conclusion is also in complete agreement with the finding of Whittaker et al. (1) that laser-mediated transmural channels do not salvage ischemic myocardium.

The similarity between eosinophilic droplets and red cells is very striking and is, without any doubt, the reason why the former have been overlooked up to now. For example, in the recent article by Wakida et al. (5 [Fig. 6]), a hemorrhage into the perimysium from hemorrhagic infarct areas is described. A careful histopathologic analysis of Figure 6 indicates, however, that the alleged hemorrhage does not concern the perimysium but the cardiomyocytes, which still manifest cross striations in their less hyalinized and not yet fragmented parts.

JIRI T. BERANEK, MD
4101 South Wappo Drive
Columbia, Missouri 65203

References

Reply

Beranek asserts that certain features of Figure 3 in our recent article (1) indicate the presence of hyalin degeneration of muscle. Specifically, he notes the presence of nonbirefringent myocytes outside the zone of thermal injury and the presence of "eosinophilic droplets" in and around the interstitial space. However, when we reexamined the section in question on the rotating stage of the polarizing microscope, we found that the myocytes, which appeared dark in the original polarized light figure (Fig. 3B), were in fact birefringent. Birefringent materials, such as cardiac muscle, appear bright when viewed with polarized light, except 1) when they have been damaged to a sufficient degree that they no longer have an anisotropic structure (as was the case in the thermally injured muscle); 2) when they are aligned parallel to the transmission axis of either of the polarizing filters (2); or 3) if they are cut in cross section. The myocytes in the left-hand corner of the original figure were aligned parallel to the transmission axis of the polarizer and thus appeared dark even though they were birefringent. When the microscope stage was rotated from the orientation shown in the figure, the dark myocytes became bright. In addition, at high magnification (×100), we observed that the "eosinophilic droplets" had the biconcave disc shape characteristic of red blood cells. These red blood cells were located in the interstitial space, which may have appeared somewhat enlarged because of edema.

Therefore, closer inspection of the tissue does not support the suggestion of hyalin degeneration of muscle proposed by Beranek.

PETER WHITTAKER, PhD
ROBERT A. KLONER, MD, PhD
KARIN PRZYKLENK, PhD
The Heart Institute, Research Hospital of the Good Samaritan
Los Angeles, California

Diagnosis of Ventricular Wall Rupture After Acute Myocardial Infarction

Rupture of the free wall of the infarcted ventricle occurs in up to 10% of patients who die of acute myocardial infarction in hospital. The course of rupture varies from sudden catastrophic massive hemorrhage into the pericardial cavity, causing immediate death, to more slowly progressing pericardial hemorrhage. In the latter case, the patient can survive for several hours, allowing clinical diagnosis and surgical repair to be performed (1). We read with interest the important study by Oliva et al. (2). Their study comprised 70 patients with myocardial rupture, 68 confirmed by autopsy and 2 by surgery, compared with 100 patients.