This study aimed to analyze morphological changes of postinfarction tissue during the restorative period after acute myocardial infarction (AMI), which, in our opinion, can change the perception of AMI in terms of its biological significance.

**Methods.** Objects of the study were cardiac biopsy specimens from 27 AMI patients who died in the hospital within 7 to 34 days after the onset of AMI, as well as heart biopsy specimens from 8 people aged 45 to 61 years old who deceased due to an unnatural cause (car accident) and whose autopsies revealed large foci of postinfarction tissue. The deceased patients had history of coronary heart disease (CHD) and arterial hypertension (AH).

**Results.** During the autopsy we diagnosed extensive intramural myocardial infarctions localized in the anterior-lateral walls of the left ventricle with the infarction zone spreading to the apex and the anterior part of the interventricular septum. All deceased patients had a severe atherosclerotic lesion in left anterior descending artery; thrombotic masses were revealed in the upper third of coronary artery in 19 deceased patients (11 men and 8 women). Postinfarction fibrous tissue was detected at the infarction site on average by 29 to 30 days after the onset of AMI. The special feature was that its fibers were oriented parallel to the preserved muscle fibers, and the remains of the preserved muscle tissue fibers, breaking

on the border with the infarction site, were continued by the fibers of newly formed postinfarction connective tissue. There was a small amount of glycogen and oxidation-reduction enzymes present in the postinfarction tissue, which were also present in the preserved cardiomyocytes.

**Conclusion.** Thus, according to the results obtained, AMI is a compensatory-adaptive process aimed at correcting the anatomical-physiological mismatch between hypertrophied myocardium and the state of coronary vessels affected by atherosclerotic process in postnatal (often late) ontogenesis.