

Myocyte contracture, vascular resistance, and vascular permeability after global ischemia in isolated hearts from alloxan-induced diabetic rabbits.

R G Tilton, A Daugherty, S P Sutera, K B Larson, M P Land, D L Rateri, C Kilo and J R Williamson

[+](#) Author Affiliations

Abstract

Coronary vascular hemodynamics, albumin permeation, and myocyte contractility were assessed in isolated hearts from 6-mo alloxan-induced diabetic (ALX-D) rabbits during 3 h of reperfusion after 40 min of global no-flow ischemia. Residue-detection data, generated during the single passage of a bolus of 125I-labeled bovine serum albumin (125I-BSA) through the coronary vasculature, were used to estimate indices of vascular function, including the mean transit time of 125I-BSA, the fractional rate of intravascular clearance of 125I-BSA, and 125I-BSA permeation of coronary vessels. During reflow after ischemia in hearts from control rabbits, vascular resistance increased approximately three times that at baseline, left ventricular end-diastolic pressure (LVEDP) increased 8–10 times, and maximum +dP/dt recovered 0.4 times baseline, whereas the fractional rate of washout of intravascular 125I-BSA decreased to less than one-half of baseline values (was prolonged 2-fold), and albumin permeation and mean-transit time were increased 3 and 5 times baseline, respectively. In hearts from diabetic rabbits, vascular resistance was similar to the control group before ischemia but increased only one-third as much during reflow after ischemia. Increases in LVEDP during reflow were approximately 50% lower than controls, and +dP/dt recovered approximately 2.5 times more than in control hearts. 125I-BSA permeation in diabetics was similar to controls before ischemia, but during reflow increased 6 times (approximately 2 times controls). Washout of intravascular 125I-BSA was prolonged approximately 20% versus baseline during 3 h of reflow in hearts from diabetic rabbits. Thus, ALX-D in the rabbit delayed ischemia-reperfusion injury to myocytes and vascular smooth muscle cells while increasing vascular albumin permeation.