Diabetic patients with cardiovascular disease (CVD) have a worse prognosis than non-diabetic individuals who develop CVD. Although the mechanism(s) responsible for this is (are) unknown, two leading candidates include hyperglycemia and insulin resistance. To completely separate the effects of hyperglycemia from insulin resistance, cardiac-selective insulin receptor knockout mice (i.e., CIRKO) mice were developed. These mice have normal glucose concentrations but lack insulin signaling in the cardiac myocyte. As such, the contribution from impaired insulin signaling per se can be evaluated. In a previous study we produced left-ventricular hypertrophy (LVH) in CIRKO and wild type (WT) mice by placing a restriction around the transverse aorta. This restriction produces a constant pressure-overload on the heart. Hearts from CIRKO mice exhibited greater left-ventricular dysfunction and subendocardial fibrosis compared to WT hearts. Increased subendocardial fibrosis could result from impaired coronary blood flow in CIRKO vs. WT mice. As such, we tested the hypothesis that coronary vascular function is impaired in CIRKO vs. WT mice in response to LVH. Fourteen days after restricting the aorta to cause LVH we assessed coronary vascular function using wire myography. Our main end-point of vascular function is the ability of the vessel to vasorelax, or increase its diameter. Contrary to our hypothesis, we observed that endothelium-dependent vasorelaxation was abolished in WT animals, but remained intact in CIRKO mice. Furthermore, the extent of subendocardial fibrosis was similar between groups. Since coronary function was intact in CIRKO mice, but subendocardial fibrosis persisted, dysfunction of the relatively large epicardial coronary vessels evaluated in the present study is not responsible. Instead, subendocardial fibrosis in CIRKO mice likely results from microvascular dysfunction.