Accumulating evidence has shown that insulin has a relevant effect on cardiovascular function and insulin-resistance has been linked to the development of essential hypertension. Macro- and microvascular complications are also common complications in diabetes. Insulin is known to stimulate sympathetic nerves, enhance Na+ renal reabsorption, alter Ca++ and Mg+ ion movement and increase norepinephrine-induced vasoconstriction. In contrast, vasorelaxing effects, attenuation of vasopressor response to angiotensin and epinephrine and reduction of vascular resistance have been demonstrated for insulin. Balance between previous effects markedly contributes to either the normality of BP or setting the hypertensive conditions. Present study compares the regional blood flow in four arterial resistance beds (carotid, renal, mesenteric and hindquarter) in control versus streptozotocin-induced diabetic rat groups and investigates the vascular reactivity of these beds to pressor and dilator substances in both groups. DRCs of vascular conductance were constructed in the 4 vascular beds after the administration of phenypherine, clonidine, isoprenaline, ATII, Na+ nitroprusside and indomethacin. The results demonstrated that in streptozotocin induced diabetic rat, regional blood flow was markedly reduced especially in the renal and hindquarters vascular beds. An enhanced response to the VC effect of the α2-adrenoceptors agonist, clonidine, angiotensin and the nitric oxide synthetase inhibitor, L-NAME was observed. No change was observed in α1-adrenoceptors-mediated responses. An attenuated response to the VD effect of the β adrenoceptors agonist, isoprenaline and the cyclo-oxygenase inhibitor, indomethacin was determined. These results suggest an impaired vascular responses in diabetes that possibly involve endothelium, prostglandins, α2 and β adrenoceptors.