SUMMARY
The early microvascular abnormality of retinal and glomerular capillaries suggest a common background for non-specific changes characteristic of diabetic microangiopathy. The presence of the endothelial cell, loss of mural cells in the retinal capillaries and associated changes in the glomerular basement membrane suggest a correlated underlying mechanism play an important role in the patho-physiology of microangiopathy in alloxan diabetic animals. The concurrence of similar observations in diabetic patients naturally show a same common feature in both the experimental induced diabetes and idiopathic and secondary human diabetes. The earliest microvascular manifestations suggest that local chemical events in the vessel wall, prior to frank carbohydrate intolerance cause dilatation of the small blood vessels before there is an impairment of blood flow. Thus microangiopathy seems to be an integrated component of the diabetic syndrome which may be genetically linked the molecular basis of sufficient but ineffective insulin circulating in the blood in the early stages of diabetes.