htpe rinsulinenia and atheroslerotic vascular disease

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Epidemiological evidence has led to the identification of aconsistent marker of cardiovascular risk in non diabetic populations,i.e. Plasma insulin levels. Subjects with hyperinsulinemia and normalblood glucose levels exhibit a state of resistance to insulin whether thisis as a cause or a consequence of hyperinsulinemia is still unresolved.Experimental evidence ascribes to insulin resistance. Severalclinical and metabolic abnormalities usually mild, e. g elevated bloodpressure, a disturbed lipid profile, glucose intolerance. The mostnatural biological marker of insulin resistance i.e. hyperinsulinemia asan independent predictor of CHD mortality risk, supports thehypothesis that a cluster of mild abnormalities can lead to significantincreased arterial damage.This cluster of mild abnormalities is probably present long before asusceptible patient develops frank hyperglycemia and eventually type IIdiabetes. Indeed, type II diabetes appears to develop essentially inindividuals who suffer from syndrome X. This is evidenced by the factthat high levels of insulinemia predict future type II diabetes in glucosetolerant subjects. Type II diabetes has a strong hereditary componentand children who have one or both parents with type II diabetes aresusceptible for developing the disease later in life. At an early age,although their glucose tolerance is normal, these children have somedegree of insulin resistance and significantly higher levels of plasmainsulin than age-matched children with no parental susceptibility todiabetes.This evidence suggest that the development of arterial damage orconditions for cardiovascular complications, probably start long beforediabetes is diagnosed by chronic hyperglycemia.Pancreas transplantation unlike heart or liver transplantation, is notan immediate life saving procedure. The objective of a pancreastransplantation is to improve the quality of life and to favorablyinfluence the secondary complications of diabetes that would otherwisearise several years hence. Pancreas transplantation is similar to kidneytransplantation, in that if kidney fails the patient can resume dialysis.Rejection, or other causes of pancreatic graft failure should befollowed by a return to exogenous insulin therapy and resumption of alife style no different than that achieved pre transplant. Pancreatictransplantation has contributed to the understanding of diabetes inseveral respects, including defining its autoimmune nature. Manyother fundamental questions related to the nature of diabetes mellitus,such as etiology or its association with microvascular and othercomplications may also be forthcoming from observation of thepancreas transplant recipients.The mounting body of evidence suggest that

pancreastransplantation does confer benefits to the patient make it a worthwhileprocedure though the true benefits while not be known until long termfollow-up in clinical trials has been established in a significant numberof patients. Should islet cell transplantation become a realisticalternative then whole organ pancreas transplantation will undoubtedlybecome obsolete. Until then it remains the new gold standard againstwhich other treatments must be compared.In conclusion, this canine model of hyperinsulinemia demonstratesthat elevated insulin levels may be associated with significantdisturbances in blood pressure, lipoprotein profile and atherosclerosis.We can also implicate that portal-enteric transplantation of thepancreas leads to lower insulin levels. Therefore, this approach maybe important for the prevention of atherosclerotic vascular disease intransplant recipients.