SUMMARY AND CONCLUSION

Epidemiological evidence has led to the identification of a consistent marker of cardiovascular risk in non diabetic populations, i.e. Plasma insulin levels. Subjects with hyperinsulinemia and normal blood glucose levels exhibit a state of resistance to insulin whether this is as a cause or a consequence of hyperinsulinemia is still unresolved.

Experimental evidence ascribes to insulin resistance. Several clinical and metabolic abnormalities usually mild, e. g elevated blood pressure, a disturbed lipid profile, glucose intolerance. The most natural biological marker of insulin resistance i.e. hyperinsulinemia as an independent predictor of CHD mortality risk, supports the hypothesis that a cluster of mild abnormalities can lead to significant increased arterial damage.

This cluster of mild abnormalities is probably present long before a susceptible patient develops frank hyperglycemia and eventually type II diabetes. Indeed, type II diabetes appears to develop essentially in individuals who suffer from syndrome X. This is evidenced by the fact that high levels of insulinemia predict future type II diabetes in glucose tolerant subjects. Type II diabetes has a strong hereditary component and children who have one or both parents with type II diabetes are susceptible for developing the disease later in life. At an early age, although their glucose tolerance is normal, these children have some degree of insulin resistance and significantly higher levels of plasma

insulin than age-matched children with no parental susceptibility to diabetes.

This evidence suggest that the development of arterial damage or conditions for cardiovascular complications, probably start long before diabetes is diagnosed by chronic hyperglycemia.

Pancreas transplantation unlike heart or liver transplantation, is not an immediate life saving procedure. The objective of a pancreas transplantation is to improve the quality of life and to favorably influence the secondary complications of diabetes that would otherwise arise several years hence. Pancreas transplantation is similar to kidney transplantation, in that if kidney fails the patient can resume dialysis. Rejection, or other causes of pancreatic graft failure should be followed by a return to exogenous insulin therapy and resumption of a life style no different than that achieved pretransplant. Pancreatic transplantation has contributed to the understanding of diabetes in several respects, including defining its autoimmune nature. Many other fundamental questions related to the nature of diabetes mellitus, such as etiology or its association with microvascular and other complications may also be forthcoming from observation of the pancreas transplant recipients.

The mounting body of evidence suggest that pancreas transplantation does confer benefits to the patient make it a worthwhile procedure though the true benefits while not be known until long term follow-up in clinical trials has been established in a significant number of patients. Should islet cell transplantation become a realistic alternative then whole organ pancreas transplantation will undoubtedly

become obsolete. Until then it remains the new gold standard against which other treatments must be compared.

In conclusion, this canine model of hyperinsulinemia demonstrates that elevated insulin levels may be associated with significant disturbances in blood pressure, lipoprotein profile and atherosclerosis.

We can also implicate that portal-enteric transplantation of the pancreas leads to lower insulin levels. Therefore, this approach may be important for the prevention of atherosclerotic vascular disease in transplant recipients.