Summary and Conclusion

Our study include 61 male diabetic patients, all have type II diabetes. Thirty one patients have autonomic neuropathy while thirty patients are without autonomic neuropathy. The study also, include 21 healthy controls of matched age and sex.

All patients have no primary chest, hepatic or renal disease, no hypertension, no clinical evidence of any cardiac disease, no history or resting E.C.G. evidence of ischaemic heart disease. They were subjected to : hisory and clinical examination, fasting and 2- hours post prandial blood glucose level, resting E.C.G., autonomic function tests, echocardiography and exercise ECG. Test (Bruce Protocol).

The pulse rate is signifiantly higher in diabetics with autonomic neuropathy when compared with control group or with diabetics without autonomic neuropathy but there is no significant difference between diabetics without autonomic neuropathy and healtly controls.

Systolic blood pressure at rest is significantly lower in diabetics with autonomic neuropathy when compared with those without autonomic neuropathy.

Non significant difference was found in diastolic blood pressure on comparing diabetics with autonomic neuropathy with those without autonomic neuropatly or with healtly controls.

There is significant increase of septal wall thickness, posterior wall thickness, aortic root diameter and right ventricular dimension in diabetics with autonomic neuropathy when compared with control group and in diabetics without autonomic neuropathy when compared with control group but non significant difference was detected in diabetics with versus diabetics without autonomic neuropathy.

The left ventricular end systolic dimension was significantly higher in diabetics with autonomic neuropathy when compared with diabetics without autonomic neuropathy or with control group but non significant difference was found between diabetics without autonomic neuropathy and control group.

The left ventricular end diastolic dimension was significantly higher in both diabetic groups when compared with control group but insignificant difference was found between the two diabetic groups.

Non significant change was found in ejection fraction and percentage fractional shortening on comparing the two diabetic groups with control group or with each other.

Resting E.C.G. parameters as P-R interval, R-wave, QTc show non significant difference between diabetics without autonomic neuropathy and control group, however QTc was significantly longer in diabetics with autonomic neuropathy when compared with diabetics without autonomic neuropathy and control group.

There is also significant prolongation of QTc after exercise in diabetics with autonomic neuropathy versus the control group and versus diabetics without autonomic neuropathy but non significant change was detected on comparing diabetic patients without autonomic neuropathy versus the control group.

R-Wave amplitude during exercise tends to decrease in the three groups i.e. no abnormal increase could be detected.

The incidence of S-T segment depression was 32.3% (10/31) in diabetics with autonomic neuropathy, 10% (3/30) in diabetics without autonomic neuropathy, and 4.8% (1/21) in the control group. Nine of the ten diabetics with autonomic neuropathy who showed S-T segment depression have no chest pain i.e. silent ischaemic episodes while in the other two groups, the S-T segment depression was always associated with chest pain.

Non significant difference in the degree of S-T segment depression was detected between the three groups.

The duration of S-T segment depression was significantly longer in diabetics with autonomic neuropathy versus diabetics without and versus control group but the difference was found to be statistically insignificant on comparing diabetic patients without autonomic neuropathy versus the control group.

Highly significant decrease in the exercise duration was found in both diabetic groups when compared with control group meanwhile non significant difference between two diabetic groups was detected.

The heart rate at maximal stress was significantly lower in diabetics with autonomic neuropathy than diabetics without autonomic neuropathy.

The exercise systolic blood pressure was significantly lower in diabetics with autonomic neuropathy compared with control group but non significant difference was detected when compared with diabetics without autonomic neuropathy.

Also non significant difference between the diabetics without autonomic neuropathy and control group was detected.

An overall conclusion of the results of our study is that the incidence of slient ischaemia is high in diabetics with autonomic neuropathy and there is an overall decrease in exercise performance in diabetics as evidenced by short exercise duration and abnormal hemodynamic responses. This abnormal performance may be due to an effect of diabetic state itself on ventricular performance (Fein et al., 1981) and also due to autonomic neuropathy with reduction of adrenergic support of the heart causing decrease of myocardial contractility and blunted cardiac acceleration which contribute to impairement of cardiac output.

The exercise E.C.G. may be valuable non-invasve technique for identification of heart diseases in diabetic patients with few complications and for screening of certain high risk population for detecting coronary artery disease because of the possibility of silent myocardial ischaemia.

A future study including exercise E.C.G., stress echocardiography together with coronary angiography is needed for better evaluation and diagnosis of the problem of silent ischaemia in diabetic patients.