INTRODUCTION
Diabetes mellitus is the most common of the serious metabolic diseases of humans. True frequency of the general population is difficult to ascertain, because of differing standards of diagnosis, probably is somewhere around 1%. In United Kingdom 1.2% of the population have diabetes about half are known to have diabetes and the rest can be estimated by population studies. Estimates for Insulin dependent diabetes mellitus are much more reliable than non-Insulin dependent Diabetes Mellitus, since most young patients are diagnosed after the appearance of symptoms. In England prevalence of Type I illness has been estimated to be 0.22% by age 10 year and in United States a study in Alleghen country, pennsylvania suggested a prevalence of 0.23% by age 20. Insulin dependent (Type I) diabetes is due to damage to the Beta cells of pancreatic islet of Langerhans. It is not directly inherited, although individuals may inherit a predisposition associated with certain HLA types. The peak incidence is 10-20 years, although elderly patients can also be insulin dependent. Non Insulin dependent (Type II) diabetes has no known cause, although in many cases there is a strong genetic
component, unrelated to the HLA system. It is most prevalent after middle age and occurs most frequently between the ages of 50 and 70 year, although there is a certain amount of overlap between the two types of diabetes.

The disease is characterised by series of hormone induced metabolic abnormalities; by long term complications involving the eyes, kidneys and blood vessels. Diabetes, besides its other ocular manifestations, also affects the intraocular pressure. Diabetics are more prone to have primary open angle glaucoma, Fallemer, in a comprehensive review states that chronic simple glaucoma doesn't occur more frequently in diabetics than in general population and that it is generally known that routine tonometry gives a lower average tension in diabetics, whereas Armstrong et al. (1960) reported evidence of 4.1% of chronic simple glaucoma in diabetics, was almost two times higher than reported in most of the studies of general population.

The risk of blindness is about 25 times greater in diabetics than in non-diabetics. Diabetic Retinopathy is a leading cause of blindness in United States between the ages of 20 and 65 years. The frequency of retinopathy
appears to vary with age of onset as well as with duration of disease. Approximately 85% of patients eventually develop retinopathy but some never develop ophthalmoscopically visible lesions even upto 30 year of disease. Absolute figures vary, however, depending upon the method of patient selection. In the recent study of prevalence of diabetic retinopathy among the patients at Joslin clinic, a sample of records of patients attending the clinic throughout a six month period was reviewed. The prevalence of Diabetic retinopathy was 25% in this total diabetic population, 7% in patients with diabetes for less than 10 year; 26% in patients with diabetes for 10-14 year; and 63% in patients with diabetes for 15 year or more.

Diabetic retinopathy can be broadly divided into two forms. In the majority of cases the lesion consists of Microneneurysms, haemorrhages, lipid exudates and retinal oedema; this may be termed as Simple Diabetic Retinopathy. Superimposed on this form is another more virulent type, PROLIFERATIVE DIABETIC RETINOPATHY, characterized by formation of new vessels in the retina and proliferating into the vitreous. It has been estimated that 10-15% of patients with simple retinopathy progress
to proliferative disease in a 10 year period.

The evidence of specific changes in the fundus of sufferers from Diabetes Mellitus was first described by Von Jaeger (1855) and at a later date many manifestations which had been observed were fully elaborated in a classical paper by Hirschberg (1990-91). The fundus abnormalities seen in diabetic retinopathy consists of changes within the retina, in front of retina and within the vitreous cavity. The intraretinal changes compose the non-proliferative or simple phase of the disease, while the preretinal and the vitreous alterations make up the proliferative or malignant phase. Non proliferative changes are first to occur and they may or may not develop into the proliferative phase.

Diabetes besides its other ocular manifestation also affects its intraocular pressure. The mean intraocular pressure in maturity onset diabetes (MODY) is 19.26 mmHg which is higher than the normal limits of intraocular pressure reported in general population i.e. 16.1 mmHg (Decker and Schaffer). The tonometer is a device that measures the pressure through the eye ball. The first practical tonometer was that of Nahlakoff (1935).
The second applanator tonometer Fick (1931) employed a fixed area of flattening produced by an adjustable force. The modern version of this instrument, Goldmann (1954) is a very accurate clinical tonometer. An applanation tonometer that has been proved to be useful in the face of corneal edema or scarring as well as in healthy cornea is the MacKay-Harg tonometer. Krakar has recently developed a tonometer that senses vibration, characteristic of corneal indentation tonometer's, have been in use over longer than applanation tonometers. Most widely accepted indentation tonometer is the one devised by Schiötz.

The present study is undertaken to evaluate the intraocular pressure in normal and diabetic patient and also to assess the changes in different grades of diabetic retinopathy.