DISCUSSION

Diabetic retinopathy is one of the common causes of blindness today. Successful treatment, in form of photoocoagulation can delay or prevent blindness in many patients of diabetic retinopathy. Detailed ophthalmoscopy and fluorescein angiography have become increasingly important to exactly pin point fundus lesions, especially, the areas of neovascularisation for effective and successful photoocoagulation.

This study comprised of twenty-five patients of diabetic retinopathy, thirteen being male and twelve female. Most of the reports show that the proportion of females to males with diabetic retinopathy is 4:3 (Duke Elder, 1967). Almost equal sex distribution in this study may not be important as total number of patients observed were small and they were selected at random.

Age of these patients varied from 20 to 74 years with an average of 51.6 years (Table 1). Maximum number of patients (56%) were in their fifth or sixth decade. This conforms to the reported incidence by Joslin et al. (1952). They reported that Diabetic retinopathy occurred in people in fifth and sixth decades of life and 50% of cases appear
Age at the onset of diabetes mellitus ranged from five years to fifty-nine years. The average age at the onset being 21.6 years in Insulin dependent diabetes mellitus patients and 45.0 years in Non-Insulin dependent diabetes mellitus patients (Table 3).

Duration of diabetes mellitus varied from 5–40 years, average duration was 16.3 years in patients with IDDM and 11.7 years in patients with NIDDM. According to Dolezaleva et al. (1981) the average interval between diagnosis of diabetes and occurrence of diabetic retinopathy was 7.3 years. Bodansky et al. (1982) reported that duration of diabetes at presentation with retinopathy was longer in IDDM. In the present study the average duration of diabetes mellitus was 13.4 years which is comparatively longer than that reported in literature. It could be due to the fact that the patients were established cases of diabetic retinopathy. Secondly the exact onset of retinopathy could not be ascertained because previous medical records were not available in most of them.
TYPE OF DIABETES MELLITUS

The patients were divided into two groups depending upon type of diabetes mellitus for comparison and analysis.

I. Insulin Dependent Diabetes Mellitus (IDDM)

II. Non-Insulin Dependent Diabetes Mellitus (NIDDM)

In the present study there were seven patients belonging to type I and eighteen to type II (Table 2).

TYPE OF DIABETIC RETINOPATHY

The cases were divided into following types of diabetic retinopathy.

1. Simple Diabetic Retinopathy (SDR)

2. Proliferative Diabetic Retinopathy (PDR)

Out of fifty eyes observed, five eyes could not be subjected to fundus examination or fluorescein angiography as four eyes had unilateral mature cataracts and one had massive vitreous haemorrhage. Amongst the remaining forty-five eyes, thirty-one eyes had changes conforming to simple diabetic retinopathy and fourteen eyes had proliferative diabetic retinopathy. The reported incidence of
proliferative diabetic retinopathy is one in every five cases of diabetic retinopathy (Root et al., 1959). Higher incidence of proliferative diabetic retinopathy (one in three) in this study could be due to selection of established cases of diabetic retinopathy with comparatively longer durations.

**Simple Diabetic Retinopathy (SDR)**

The patients who had SDR were in the age range of 22-63 years and male to female ratio was 8:7. Twenty percent of these patients had IDDM and 80% NIDDM. Age at the onset of diabetes mellitus in these patients varied from fourteen years to fifty-eight years, average being 38.5 years. The duration of diabetes mellitus in these patients ranged from five years to twenty years, average duration being ten years. The reported average duration of diabetes prior to the development of retinopathy is 7.3 years (Dolezalova et al., 1981). Average figure is higher in this study because these were established cases of diabetic retinopathy.

Quality of control of diabetes mellitus in these patients was good in 33%, moderate in 40%
and poor in 27% patients (Table 4). There was no relationship between quality of control of diabetes mellitus and occurrence of SDR in the present series.

**Proliferative Diabetic Retinopathy (PDR)**

In this study the patients having PDR were in age range of 20-74 years and male to female ratio was 1:1. 40% of these patients had IDDM and 60% had NIDDM. Age at the onset of diabetes mellitus in these patients varied from 5-59 years, average being 37 years. Duration of diabetes mellitus in these patients varied from 2-40 years average being 17-7 years. Roet et al. (1959) reported that average duration of diabetes before discovery of PDR was 17.4 years and none developed this disease in less than eight years. Oakley et al. (1974) reported this duration to be much longer thirty two years. Only one patient had shorter duration (eight years) in this study, which might be due to delay in the establishment of initial diagnosis of diabetes in this patient or poor and irregular control of diabetes.

The reported average duration of diabetes in patients presenting with PDR is sixteen years.
for NIDDM and twenty years for IDDM (Beetham, 1963). In present study these figures were fifteen years and twenty-one years respectively (Table 1A).

As per table 4, among patients with PDR, 40% had moderate quality of control of diabetes and 60% had poor control. None of these patients had good control. Burditt et al. (1963) have reported that low glycosuria percentage is associated with low frequency of retinopathy. Bedansky et al. (1982) reported that poor glycaemic control is most important factor in diabetic retinopathy. In a study of fourteen cases of PDR, Valens et al. (1978) found thirteen cases who were having poor diabetic control. Kingsley et al. (1983) reported, a long standing history of poor diabetic control in six out of nine cases of PDR in adolescents. It could be emphasized that poor control of Diabetes mellitus is an important factor in causing Diabetic retinopathy especially PDR.

**Microaneurysms**

Microaneurysms are seen on opthalmoscopy as bright red, well defined dots of a globular form with sharp rounded edges, showing a characteristic
reflex attimes. On F.A., these appear as distinct round fluorescent spots in the venous phase.
Microaneurysms were observed in 43 (96%) eyes on F.A. whereas on ophthalmoscopy only 31 (69%) eyes showed microaneurysms. Also number and extent of microaneurysms observed on F.A. was much more than that observed on ophthalmoscopy. Two eyes, where microaneurysms were not observed, had no other fundus abnormality. Other eye of these two patients showed few microaneurysms as the only retinopathic finding on F.A. All the eyes with PDR were observed to have microaneurysms. Ballantyne and Locarnstein (1943) who initially recognised microaneurysms as a significant sign of Diabetic retinopathy, stated that at first these are few in number and without other signs of retinopathy and eventually their number increased. Bresnick et al. (1977) reported that capillary microaneurysms was the earliest change on F.A. Norton and Gutman (1965) found F.A. as a more sensitive technique than colour fundus photography or ophthalmoscopy for detecting these lesions. Bresnick et al. (1977) in their clinicopathological study reported that many histologically proved microaneurysms, not seen
on colour photography, were associated with definite abnormalities on F.A. It could be concluded that the presence of microaneurysms is one of the earliest findings in diabetic retinopathy and F.A. is an invaluable technique in diagnosing early case of diabetic retinopathy as it can demonstrate even few small and early microaneurysms.

All of these microaneurysms were observed in posterior pole of fundus. Leakage of the fluorescein dye from microaneurysms was observed in majority of these eyes (Table 8). This leakage was observed in late phase of angiography. Ashton (1974) reported abnormal permeability of endothelial lining of microaneurysms which may be due to deposition of lipids or PAS positive material. This indicates that walls of the microaneurysms do not maintain the normal blood retinal barrier and hence show increased permeability.

Retinal Haeorrhages

Bremsnick (1980) reported that retinal haemorrhage in diabetic retinopathy are typically rounded and occur mainly in deep capillary plexus
from pathological capillaries and microaneurysms commonly. These may appear as small red dots or with blot appearance having fuzzy borders.

Superficial flame shaped haemorrhages may also occur. In this study retinal haemorrhages were observed in 62% of the eyes observed. Whereas all the eyes with PDR had retinal haemorrhages, only 45% of eyes with SDR had retinal haemorrhages (Table 9). According to Duke Elder (1967) haemorrhages are found in all stages of retinopathy, although they usually follow the appearance of microaneurysms.

The number of haemorrhages varied from one or two punctate spots to multiple haemorrhages. One eye had extensive subhyaloid haemorrhage. All the haemorrhages observed were behind the equator. On F.A., these haemorrhages blocked background fluorescence, some of these were stained with dye during late phase of angiography.

Superficial flame shaped haemorrhages observed in five eyes may be due to associated systemic hypertension in these cases.
Retinal Exudates

Hard retinal exudates varying from few small discrete dry exudates to large multiple exudates were observed in 60% of the eyes. All but one i.e. 93% of eyes with PDR had retinal exudates whereas 43% of eyes with SDR had retinal exudates. 56% of eyes of IDDM patients and 65% of eyes of NIDDM patients had retinal exudates (Table 10). In few eyes leakage of the fluorescein dye was observed from the retinal vessels in vicinity of hard exudates in late phase of angiography. Reker et al. (1963) however reported that there is no abnormality in retinal exudates on P.A. during passage of the dye through overlying capillary bed. But Norton and Gutman (1965) showed that exudates have no tendency to fluoresce early or late. However retinal vessels overlying them or lying between ring of exudates do leak fluorescein and stain the retina.

Cotton wool spots which were observed in six eyes can be explained by the associated presence of hypertension in all these patients.
**Venous changes**

Venous changes in the form of venous dilatation were observed in 56% of the eyes observed. 75% of eyes with PDR had venous dilatation whereas 43% of eyes with SDR had venous dilatation (Table 11). 69% of eyes of patients with NIDDM had venous dilatation whereas only 36% of eyes of patients with IDDM had venous dilatation. Though these changes were present in most of early cases retinopathy it was not a consistent feature in this study. Fluorescein angiography was more helpful in observing venous dilatation.

Venous dilatation which is however not pathognomonic of diabetic retinopathy has been reported as an early feature in Diabetic Retinopathy (Ballantyne and Michaelson, 1947). Alberts and Slesse (1957) reported distension of small venules draining paramacular area as a less frequent early phenomenon.

But Sherberg et al. (1969) showed that the venous dilatation does not precede the onset of retinopathy but may occur as one of the features of established retinopathy.
Capillary and Arteriolar Changes

Retinal capillaries show obstruction as well as dilatation in diabetic retinopathy. Capillary obstruction may be in form of focal capillary obstruction, perifoveal capillary obstruction or regional capillary obstruction (Bresnick, 1980). Earliest capillary obstruction changes are in patchy fashion (Levene et al., 1956). The areas of focal or regional capillary obstruction can be observed on F.A. as areas of non-perfusion. In the present study 40% of the eyes showed areas of capillary non-perfusion. Almost all the eyes (93%) with PDR had areas of non-perfusion whereas only 16% eyes with SDR showed these areas (Table 12). These eyes with areas of non-perfusion were associated with arteriolar changes in form of narrowing of lumen and complete obstruction of lumen of arterioles (five eyes). The latter was observed as non-filling of the arterioles and regional areas of non-perfusion in their distribution.

These areas of non-perfusion were associated with dilated patent capillaries which leaked fluorescein in late phase of angiography. Some of these dilated
vessels were very prominent and could be called shunt vessels, which were observed in twelve (27%) eyes. 71% of eyes of PDR and 6% of eyes with SDR showed shunt vessels (Table 13).

Neovascularisation

Neovascularisation is the earliest finding of PDR. It is almost always found posterior to the equator, common site being 1-3 Disc diameters around the disc, along major retinal vessels preferentially near arteriovenous crossings with a predilection for superotemporal quadrant (Taylor, 1970). In the present study new vessels were found in fourteen (31%) eyes, in ten different patients. Forty percent of these patients had IDDM and sixty percent patients had NIDDM. In a study by Beetham (1963) of 109 patients of PDR with neovascularisation, sixty-five patients had IDDM and forty-four NIDDM. Different figures in the present study as compared to the reported incidence may not be significant due to small number of patients in this study. Total number of patients with IDDM in this study was only 26% against 72% of NIDDM patients. This may be another explanation for comparatively less number of total
patients with PDR out of IDDM group.

Among these fourteen eyes with neovascularisation, nine (64%) eyes had neovascularisation at disc and five (36%) eyes had neovascularisation elsewhere in fundus. Ballantyne and Michaelson (1947) and Larsen (1960) reported that neovascular formation was usually most marked in the area around optic disc but these may occur in connection with retinal vessels anywhere between disc and periphery.

On F.A., these new vessels were observed in detail as irregular and tortuous vessels in initial angiograms but in later phase they profusely leaked the dye and their outline was obscured. Scott et al. (1963) reported that on F.A. new vessels were revealed in detail, they have a haphazard arrangement and follow a tortuous course and they showed stasis of blood and transudation of fluorescein. Yuval Yassur et al. (1980) performed F.A. to demonstrate disc neovascular leakage which occurred 1-2 minutes after the injection of fluorescein.

**Vitreous changes**

One eye was having massive vitreous haemorrhage which obscured the fundus view and F.A. was not
helpful in this case. One eye showed subhyaloid haemorrhage, new vessels could not be found in this case even on F.A., as the haemorrhage covered the central area of fundus and new vessels, the likely source of this haemorrhage, were hidden by it.

Scott et al. (1964) have demonstrated with F.A. the tendency of neovascularisation to bleed and cause recurrent vitreous haemorrhage.

Retinal detachment

Tasman (1972) reported that duration of diabetes before the retinal detachment developed was eighteen years for NIDDM and twenty-four years for IDDM. Most of retinal detachments resulting from PDR begin as neovascularogenous tractional detachments but they may become rheumatogenous by formation of holes (Bromanick, 1980). In the present series retinal detachment was not observed in any case.

Diabetic Maculopathy

Diabetic maculopathy is a condition in a case of diabetic retinopathy when aided visual acuity is less than 6/24 and there is no sufficient cause except for the changes in retina and vitreous
to explain this reduced vision. Bresnick (1980) reported that this can be due to intraretinal changes in form of macular edema and hard exudates at macula caused by increased retinal vascular permeability and macular ischemia due to occlusion of vessels supplying the macula or preretinal and vitreoretinal changes which include thickening of posterior vitreous surface and new pre-retinal membrane formation as a result of fibrous, glial and fibrovascular proliferation and tractional detachment of macula caused by subsequent shrinkage of these tissues and of the vitreous.

In the present study twenty-four eyes had aided visual acuity less than 6/24 (Table 5). Out of these twenty-four eyes, four had mature cataract and six had lenticular opacities in form of immature cataract. Therefore there were fourteen (28%) eyes with aided visual acuity less than 6/24, which could be attributed to diabetic maculopathy. Among these fourteen eyes, ten eyes belonged to patients with NIDDM and four eyes to IDDM patients. Burditt et al. (1968) reported that diabetic maculopathy
more common in NIDDM patients which they attributed to old age in these patients when macular disease in general is more common. Table 6 shows that aided visual acuity less than 6/24 is less common (29% eyes) in SDR and more common (74% eyes) in PDR. Beetham (1963) reported that overall visual prognosis in untreated PDR was poor although some patients maintained good vision for many years. He further reported that 22% of IDDM patients were legally blind after a mean duration of 3.3 years and 34% of NIDDM were legally blind after a mean duration of 3.1 years with PDR. It could be emphasized that patients with PDR had poor visual prognosis.

Side effects of Intravenous Injection of Fluorescein Dye

Hayrey (1968) reported that transient nausea and occasional vomiting may occur in 5–10% of cases but are of no serious consequences. Stein and Parke (1971) have summarised serious side effects in total of fifty-five patients reported so far, in form of urticaria, tachycardia, hypertension and cardiac arrest. In the present study only one patient had nausea followed by vomiting and patient felt relieved after little rest. Therefore, F.A. is practically quite safe diagnostic procedure.