INTRODUCTION AND AIM OF THE WORK

Although diabetic maculopathy was once considered as an overlooked complication of diabetic retinopathy, it has been now recognized as the leading cause of visual impairment in diabetic population. So it requires evaluation and management specific to the maculopathy rather than being considered a non specific component of generalized retinopathy.

Fluorescein angiography is very useful method in detecting retinal microvascular changes, and it has been used in this study to evaluate diabetic microvascular changes, and to compare these changes in relation to functional and clinical findings.

The macular region is a specilized region of the retina that is approximately 5.5 mm in diameter **(Hamming and Apple, 1980)**. It is a shallow oval depression whose center lies 3.5 mm lateral to the edge of optic disc and about l mm inferior to its center **(Wolff, 1976)**.

The macular region consists, from its center outward of the foveola (0.35 mm), the fovea (l.9 mm), and the peripheral macula (5.5 mm). The peripheral macula is somtimes divided into an inner parafoveal area and outer perifoveal area **(Wolff, 1976)**.

The anatomic macula starts from the temporal edge of the disc extending temporally for a distance of 4-5 dis diameters and almost completely between the superior and the inferior vascular arcades. Clinically, the anatomic macula is commonly referred to as the posterior pole.

The anatomic fovea: can be defined as that part of the retina where there is a sudden change in its internal surface so that a slope or cilvus directed internally, then flat, then externally forms an elongated narrow based pit. The diameter across the pit is (l.5 mm) approximately. Clinically the start of the slope causes a change in the reflex from the retinal surface appearing as a ring. The anatomic fovea is commonly referred to clinically as the macula **(Yanoff, 1979)**.

The anatomic foveola: is the floor of the fovea and measures approximately 0.35 mm, it is slightly smaller than and contained within the retinal avascular zone. The shape of the foveola, much like a concave

mirror, often produces a reflected spot of light overlying it in the vitreous. This is called clinically the foveal light reflex. The size, shape and intensity of the light reflex depends on the slope and shape of the foveola. Clinically the anatomic foveola is referred to as the fovea **(Yanoff, 1979).**

* *Pigment epithelium: The cells of macular retinal pigment epithelium are narrow, tall, regular and contain pigment granules (Pure lipofuscin granules and a mixture of lipofuscin and melanin granules), throughout their cytoplasm. The extramacular retinal pigment epithelium are wide, low, irregular and contain melanin granules which concentrated mainly within their apical cytoplasm. The configuration of pigment epithelium in the macular area together with the type and the distribution of pigment granules within the cells, and the fact that its center is devoid of blood vessels, as well as the presence of yellow pigment (xanthophyll) in the external plexiform layer are the cause for its hypofluorescence in fluorescein angiography* **(Yanoff, 1979).**

* *Muller's supporting fibers: are long complicated structures that traverse the entire thickness of the retina from the internal limiting to the external limiting membrane. The floor of the fovea is abundant in Muller's fibers processes* **(Hamming and Apple, 1980).**

* *Henle's Layer: in order to optimize light transmission to the foveal cone all retinal elements have to be displaced laterally out of the light path. The nerve fibers in the outer plexiform layer, therefore have to run almost parallel with the retinal surface before reaching their*

points of synapse. However this lateral displacement of the retinal layers also disturbs the normal reticular architecture of the supporting mullers cells and consequently, the retina in this region loses its compact nature and becomes very susceptible to deposition of large amounts of extracellular fluids., Exudates within Henle's fiber layer typically assume a star shape configuration corresponding to the radial arrangement of the fibers as they diverge from the center of the fovea **(Kanski, 1989).**

The importance of the macular region for visual function is emphasized by the fact that at least 1/3 of all the fibers which enter the optic nerve originate in this region. These macular fibers enter the optic nerve at its temporal edge but immediately coarse into the central part **(Hamming** and **Apple, 1980).**

* Blood supply: The macular region is supplied by tiwgs from the superior and inferior temporal vessels, but the fovea itself, over an area about 0.5 mm in diameter entirely free of vessels. The superior and inferior macular branches supply the perifoveal capillaries almost equally, so that a horizontal division exists through the fovea, which accounts for the horizontal division of the fixation area frequently seen in obstruction of an arterial branch **Wolff, 1976).**

A cilioretinal artery can be seen with the ophthalmoscope as a vessel with hooked shaped origin, running from just within the temporal edge of the disc toward the macula. It is present in about 15-20% of persons and derived from the ciliary circulation. Occasionally a large cilioretinal artery may supply the entire macular region.

The peripheral macular region is richly vascularized by three layers of capillaries all situated within the inner half of the retina. Two superficial capillary plexuses, one entirely in the nerve fiber layer and the other at the inner boundary of the inner nuclear layer. A deep capillary plexus lying at the outer boundary of the inner nuclear layer. The three capillary plexuses are not however independent, anastomotic capillaries run from one layer to the other, and the same capillary may run for part of its course in one layer and then change to another (**Wolff, 1976**).

Histologically, the retinal capillaries consists of two distinct cell type: the endothelial cells and the pericyte. The endothelial cells are closely bound together by intercellular junctions of the zonulae occludens type. These junctions normally prohibit a free flow of fluid and solutes from the vascular lumen into the retinal interestitum, thus creating a blood retinal barrier. The pericytes form a layer around the outer aspect of the endothelial cell basement membrane and probably serve to provide structural integrity to the vessel wall (**Hamming and Apple, 1980**).

Leakage of the superficial capillary plexus is not too important because of the rich surrounding capillary bed which reabsorb the excess fluid rapidly, leakage however from the deep plexus as occurs in diabetic retinopathy "spills over" into the avascular outer retinal layers where reabsorption takes place slowly. Hence if leakage is significant, fluid collects in the outer retinal layers in the form of microcystoid spaces (**Yanoff, 1979**).

The ratio of pericytes to endothelial cells in retinal capillaries of a normal young person approximately l: l. In normal aging process, relative decrease in capillary endothelial cells occurs, however in diabetic retinopathy the reverse takes place. Pericytes gradually lost **(Yanoff, 1969).**

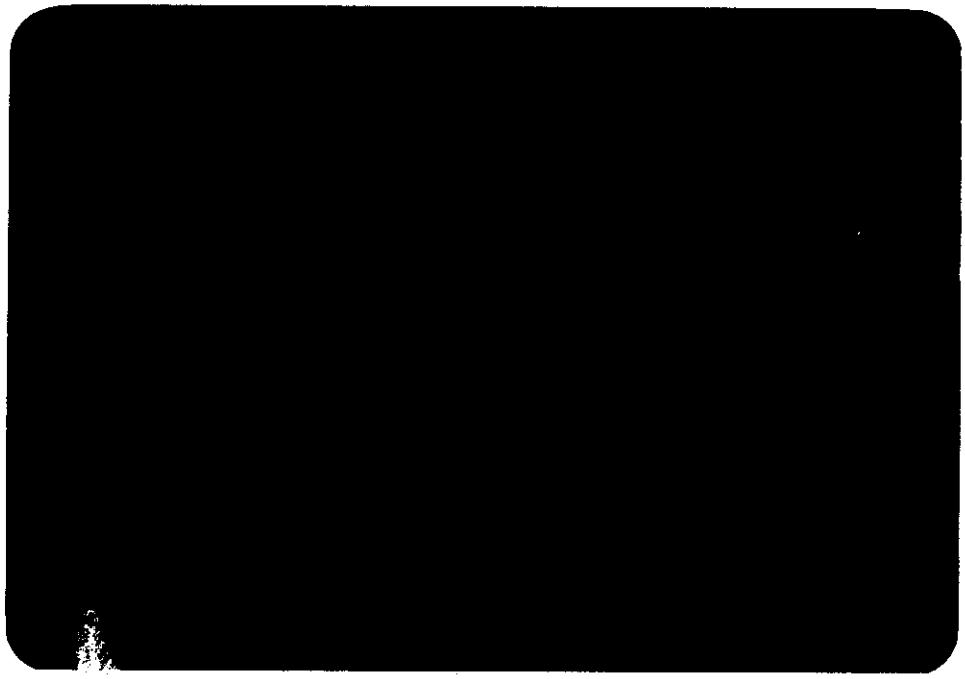

Fig. (l)

Left eye: Coloured fundus photograph,
Normal macular area.

Diabetic maculopathy is now a Technical Term more precise than a sick macula in a diabetic and may be defined as "visual loss due to macular oedema or hard exudates" **(Kohner et al., 1982).**

The pathologic changes in diabetic maculopathy can be divided on an anatomic basis into two broad categories:

I. Intraretinal changes.

II. Preretinal and vitreoretinal changes.

The intraretinal changes include macular oedema, hard exudates, and macular ischaemia. The preretinal and vitreoretinal changes include: Thickening of the posterior vitreous surface, preretinal membrane formation, tractional detachment of the macula, and macular ectopic **(Bresnick, 1980).**

(I) INTRARETINAL MACULOPATHY

A. Macular oedema:

Patz et al. (1973) were one of the first to call the attension of the ophthalmic community to the fact that although complications of proliferative diabetic retinopathy cause the most disastrous losses of vision (from hand movement to no light perception) the most common cause of mild to moderate visual loss is macular oedema. It occurs in approximately 10% of diabetic population. For those patients with 20 or more years duration of disease, the prevalence increase to approximately 25% **(Klein et al., 1984).**