

Chapter one: Introduction

Chapter one

Introduction

1-1 Introduction :

The optic nerve it is the sensory nerve of the retina. Its fibres originate in the ganglion layer and converge on the posterior part of the eye ball. The nerve passes backwards through the orbit and optic canal into the middle cranial fossa where it unites with the nerve of opposite side of the optic chiasma.(Longman , 2003)

Diabetes is a common cause of damage to the optic nerve. Diabetes is a condition where the body's incapable of correctly processing and using sugar. The body's incapable of metabolizing sugars properly, resulting in high levels of glucose within the blood. When left untreated for prolonged periods, blindness can result from damage to the optic nerve. Blurred vision usually occurs prior to complete vision loss in diabetes patients. (Diabetes Programme World Health Organization. Retrieved 22 April 2014.)

MRI has great value in measurements of the optic nerve diabetic Patient .

1-2 Research problem :

It is well known that diabetes affects the optic nerve, but this is not supported by measurements, and here lies the importance of research it gives measurements to see the extent of the damage caused by diabetes, which helps to reduce the increase.

1-3 General Objective:

- ❖ To chaitlize the optic nerve diameter in diabetic patient.

1-4 Specific Objective:

- 1- To measure the optic nerve diameter in coronal and sagittal MRI Images.
- 2- To compare the findings with control group.
- 3- To correlate the findings with patient age, gender and duration of diabetes.
- 4- To correlate the findings with diabetes type.

1-5 Significance:

My study is important because it is measuring the effectiveness of magnetic resonance imaging device to measure the damage of the optic nerve damage due to diabetes to resolve this problem.

1-6The scope if the study:

The study contains five chapters, chapter one consisted of introduction that contain an idea about the eye and the optic nerve in addition to Research problem, objectives and Significance of the study. Chapter two includes the literature review and previous studies .Chapter three describes the material and methods. Chapter four includes the result, and lastly chapter five includes the discussion, conclusion and recommendation.

Chapter two: Literature review

Chapter two

Literature review

(Anatomy, Physiology and Pathology)

2-1-1 Anatomy of the Eye:

The Eye

When you look at a person's eye you see only a small part of the whole eye. Three layers of tissue form the eye ball: the sclera, the choroids, and the retina. The outer layer of sclera consists of tough fibrous tissue. The white of the eye is part of the front surface of the sclera. The other part of the front surface of the sclera is called the cornea and is sometimes spoken of as the window of the eye because of its transparency. At casual glance, however, it does not look transparent but appears blue, brown, gray, or green because it lies over the iris, the colored part of the eye. A mucous membrane known as the conjunctiva lines the eyelids and covers the sclera in front. The conjunctiva is kept moist by tears formed in the lacrimal gland located in the upper lateral portion of the orbit. .(Longman , 2003)

The middle layer of the eyeball, the choroid, contains a dark pigment to prevent the scattering of incoming light rays. Two involuntary muscles make up the front part of the choroids. One is the iris, the colored structure seen through the cornea, and the other is the ciliary muscle . The black center of the iris is really a hole in this doughnut-shape muscle; it is pupil of the eye. Some of the fibers of the iris are arranged like spokes in a wheel. When they contract the pupils dilate, letting in more light rays. Other fibers are circular. When they contract, the pupils

constrict, letting fewer light rays. Normally, the pupils constrict in bright light and dilate in dim light. When we look at distant objects, the ciliary muscle is relaxed, and the lens has only a slightly curved shape. To focus on near objects, however, the ciliary muscle contract. As it contracts, it pulls the choroids coat forward toward the lens, thus causing the lens to bulge and curve even more. Most of us become more farsighted as we grow older and lose the ability to focus on close objects because our lenses lose their elasticity and can no longer bulge enough to bring near objects into focus. Presbyopia or oldightedness is the name for this condition. The retina or innermost layer of the eyeball contains microscopic receptor cells, called rods and cones because of their shapes. Dim light can stimulate the rods, but fairly bright light is necessary to stimulate the cones. In other words, rods are the receptors for night vision and cones for daytime vision. There are three kinds of cones; each is sensitive to a different color: red, green, or blue. Scattered throughout the central portion of the retina, these three types of cones allow us to distinguish between different colors. .(Longman , 2003)

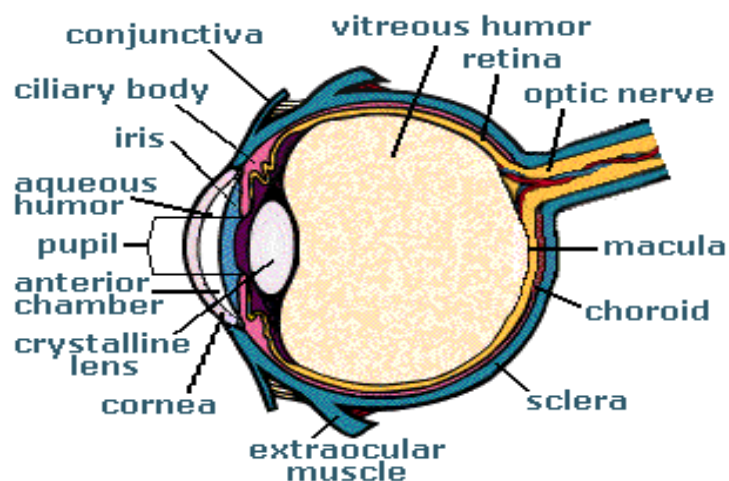


Fig number 2-1 shows the Anatomy of the Eye.(Longman , 2003)

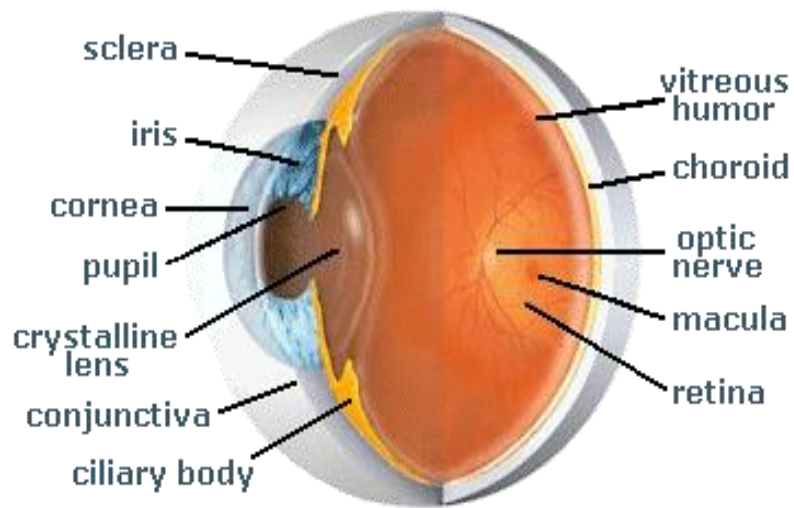


Fig number 2-2 shows the internal eye anatomy .(Longman , 2003)

2-1-2 The Optic Nerve:

The **optic nerve** it is the sensory nerve of the retina. Its fibres originate in the ganglion layer and converge on the posterior part of the eye ball. The nerve passes backwards through the orbit and optic canal into the middle cranial fossa where it unites with the nerve of opposite side of the optic chiasma. (Longman, 2003)

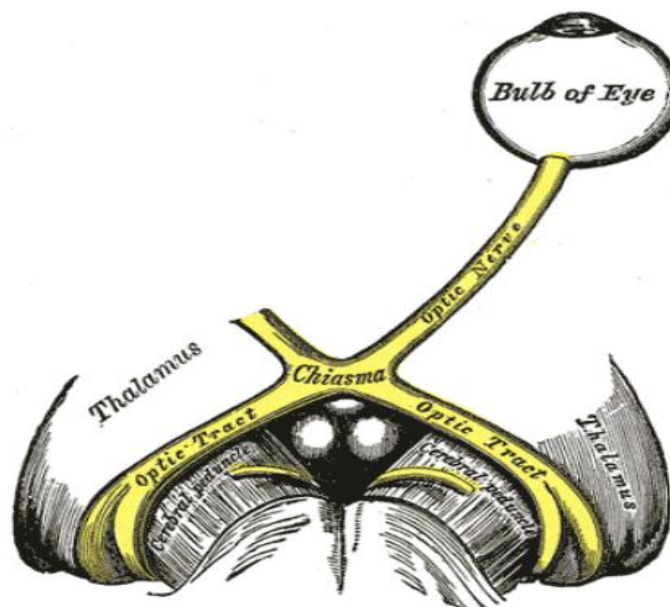


Fig number 2-3 show the optic nerve.(Longman , 2003)

2-1-3 Visual pathway:

Light is the stimulus that results in vision (that is our ability to see objects as they exist in our environment). Light enters the eye through the pupil and is refracted or bent so that it is focused on the retina.

Refraction occurs as light passes through the cornea, the aqueous humor, the lens, and the vitreous humor on its way to the retina. The innermost layer of the retina contains the rods and cones, which are the photoreceptor cells of the eye. (Longman, 2003).

They respond to a light stimulus by producing a nervous impulse. The rod and cone photoreceptor cells synapse with neurons in the bipolar and ganglionic layers of the retina. Nervous signals eventually leave the retina and exit the eye through the optic nerve on the posterior surface of the eyeball. No rods or cones are present in the area of the retina where the optic nerve fibers exit. The result is a "blind spot" known as the optic disc. (Longman, 2003).

After leaving the eye, the optic nerves enter the brain and travel to the visual cortex of the occipital lobe. In this area of the brain, visual interpretation of the nervous impulses that were generated by light stimuli in the rods and cones of the retina result in "seeing". (Longman, 2003).

2-1-4 Blind spot:

The beginning of the optic nerve in the retina is called the optic nerve head or optic disc. Since there are no photoreceptors (cones and rods) in the optic nerve head, this area of the retina cannot respond to light stimulation. As a result, it is known as the "blind spot," and everybody has one in each eye. The reason we normally do not notice our blind spots is because, when both eyes are open, the blind spot of one eye corresponds to retina that is seeing properly in the other eye.

(Montgomery, 1998).

2-1-5 The Retina:

The retina is the innermost layer of the eye (the tunica intima or internal tunic) and is comparable to the film inside of a camera. It is composed of nerve tissue which senses the light entering the eye. (Montgomery,1998).

This complex system of nerves sends impulses through the optic nerve back to the brain, which translates these messages into images that we see. That is, we “see” with our brains; our eyes merely collect the information to do so. The retina is composed of 10 layers, from the outside (nearest the blood vessel enriched choroid) to the inside (nearest the gelatinous vitreous humor): pigmented epithelium, photoreceptors, bacillary layer (outer and inner segments of cone and rod photoreceptors), external (outer) limiting membrane, outer nuclear (cell bodies of cones and rods), outer plexiform (cone and rod axons, horizontal cell dendrites, bipolar dendrites), inner nuclear (nuclei of horizontal cells, bipolar cells, amacrine cells, and Müller cells), inner plexiform (axons of bipolar cells and amacrine cells, dendrites of ganglion cells), ganglion cells (nuclei of ganglion cells and displaced amacrine cells), nerve fiber layer (axons from ganglion cells traversing the retina to leave the eye at the optic disc), and internal limiting membrane (separates the retina from the vitreous). (Montgomery,1998).

Beneath the pigmented epithelium of the retina are these 4 layers, from the outside (furthest from the retina) to the inside (closest to the retina): sclera (white part of the eye) , large choroidal blood vessels, choriocapilaris , Bruch’s membrane (separates the pigmented epithelium of the retina from the choroid) (Montgomery,1998).

Light entering the eye is converged first by the cornea, then by the crystalline lens. This focusing system is so powerful that the light rays intersect at a point just behind the lens (inside the vitreous humor) and diverge from that point back to the retina. (Montgomery,1998).

This diverging light passes through 9 (clear) layers of the retina and, ideally, is brought into focus in an upside-down image on the first (outermost) retinal layer (pigmented epithelium). Then, amazingly, the image is reflected back onto the adjacent second layer, where the rods and cones are located.

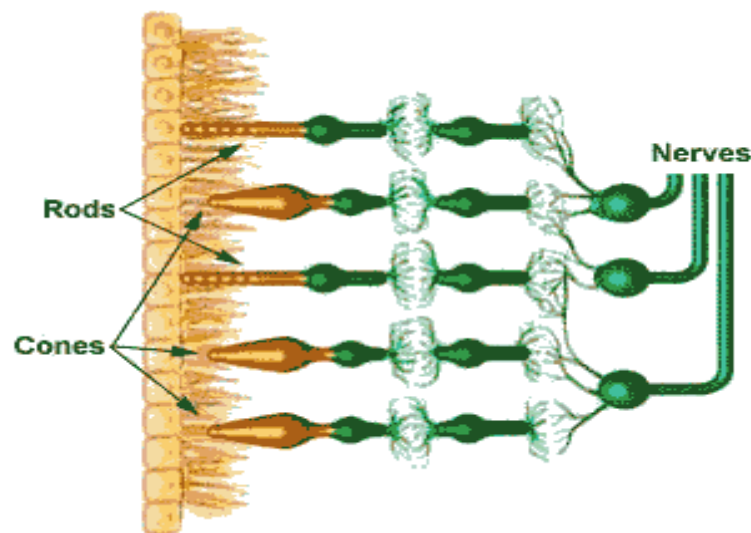


Fig number 2-4 show the photoreceptors (Montgomery,1998).

Cones and rods actually face away from incoming light, which passes by these photoreceptors before being reflected back into them. Light causes a chemical reaction with photopigments: “iodopsin” in cones (activated in photopic or bright conditions) and “rhodopsin” in rods (activated in scotopic or dark conditions). This begins the visual process. (Montgomery,1998).

2-2 Physiology of the Eye:

Process of vision:

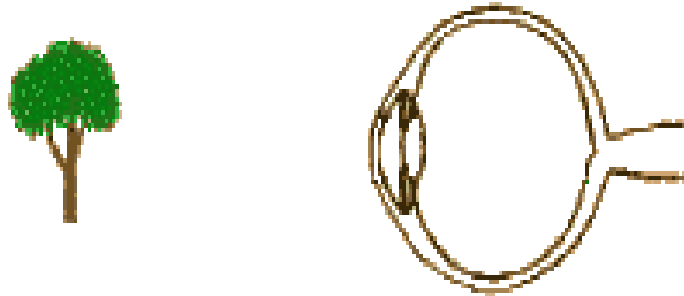


Fig number 2-5 show Process of vision(Montgomery,1998).

light waves from an object (such as a tree) enter the eye first through the cornea, which is the clear dome at the front of the eye. It is like a window that allows light to enter the eye. The light then progresses through the pupil, the circular opening in the center of the colored iris. (Montgomery,1998).

Fluctuations in the intensity of incoming light change the size of the eye's pupil. As the light entering the eye becomes brighter, the pupil will constrict (get smaller), due to the pupillary light response. As the entering light becomes dimmer, the pupil will dilate (get larger). (Montgomery,1998).

Initially, the light waves are bent or converged first by the cornea, and then further by the crystalline lens (located immediately behind the iris and the pupil), to a nodal point (N) located immediately behind the back surface of the lens. At that point, the image becomes reversed (turned backwards) and inverted (turned upside-down).

(Montgomery,1998).

The light continues through the vitreous humor, the clear gel that makes up about 80% of the eye's volume, and then, ideally, back to a clear focus on the retina, behind the vitreous. The small central area of the retina is the macula, which provides the best vision of any location in the retina. If the eye is considered to be a type of camera (albeit, an extremely complex one), the retina is equivalent to the film inside of the camera, registering the tiny photons of light interacting with it. (Montgomery,1998).

Within the layers of the retina, light impulses are changed into electrical signals. Then they are sent through the optic nerve, along the visual pathway, to the occipital cortex at the posterior (back) of the brain. Here, the electrical signals are interpreted or “seen” by the brain as a visual image. (Montgomery,1998).

2-3 Pathology of the Eye:

2-3-1 Optic atrophy:

Optic atrophy of the optic disc (visible to an eye doctor looking inside the eye) is the result of degeneration of the nerve fibers of the optic nerve and optic tract. It can be congenital (usually hereditary) or acquired. (Montgomery,1998).

If acquired, it can be due to vascular disturbances (occlusions of the central retinal vein or artery or arteriosclerotic changes within the optic nerve itself), may be secondary to degenerative retinal disease (e.g., optic neuritis or papilledema), may be a result of pressure against the optic nerve, or may be related to metabolic diseases (e.g., diabetes), trauma, glaucoma, or toxicity (to alcohol, tobacco, or other poisons). (Montgomery,1998).

Loss of vision is the only symptom. A pale optic disc and loss of pupillary reaction are usually proportional to the visual loss.

Degeneration and atrophy of optic nerve fibers is irreversible, although in some cases, intravenous steroid injections have been seen to slow down the process. (Montgomery,1998).

2-3-2 Optic neuritis:

Optic neuritis is an inflammation of the optic nerve. It may affect the part of the nerve and disc within the eyeball (papillitis) or the portion behind the eyeball (retrobulbar optic neuritis, causing pain with eye movement). It also includes degeneration or demyelination of the optic nerve. There will be no visible changes in the optic nerve head (disc) unless some optic atrophy has occurred. (Montgomery,1998).

This condition can be caused by any of the following:

demyelinating diseases (e.g., multiple sclerosis, postinfectious encephalomyelitis), systemic infections (viral or bacterial), nutritional and metabolic diseases (e.g., diabetes, pernicious anemia, hyperthyroidism), Leber's Hereditary Optic Neuropathy (a rare form of inherited optic neuropathy which mainly affects young men, causing them to lose central vision), secondary complications of inflammatory diseases (e.g., sinusitis, meningitis, tuberculosis, syphilis, chorioretinitis, orbital inflammation), toxic reactions (to tobacco, methanol, quinine, arsenic, salicylates, lead); and trauma. (Montgomery,1998).

The condition is unilateral rather than bilateral. If the nerve head is involved, it is slightly elevated, and pupillary response in that eye is sluggish. There usually is a marked but temporary decrease in vision for

several days or weeks, and there is pain in the eye when it is moved. Single episodes generally do not result in optic atrophy nor in permanent vision loss; however, multiple episodes can result in both. (Montgomery,1998).

2-3-3 Papilledema:

Papilledema is edema or swelling of the optic disc (papilla), most commonly due to an increase in intracranial pressure (often from a tumor), malignant hypertension, or thrombosis of the central retinal vein. The condition usually is bilateral, the nerve head is very elevated and swollen, and pupil response typically is normal. (Montgomery,1998).

Vision is not affected initially (although there is an enlargement of the blind spot), and there is no pain upon eye movement. Secondary optic atrophy and permanent vision loss can occur if the primary cause of the papilledema is left untreated. (Montgomery,1998).

2-3-4 Ischemic optic neuropathy:

Ischemic optic neuropathy is a severely blinding disease resulting from loss of the arterial blood supply to the optic nerve (usually in one eye), as a result of occlusive disorders of the nutrient arteries. Optic neuropathy is divided into anterior, which causes a pale edema of the optic disc, and posterior, in which the optic disc is not swollen and the abnormality occurs between the eyeball and the optic chiasm. (Montgomery,1998).

Ischemic anterior optic neuropathy usually causes a loss of vision that may be sudden or occur over several days. Ischemic posterior optic neuropathy is uncommon, and the diagnosis depends largely upon exclusion of other causes, chiefly stroke and brain tumor. (Montgomery,1998).

2-3-5 Glaucoma:

Glaucoma is an insidious disease which damages the optic nerve, typically because the “intraocular pressure” (IOP) is higher than the retinal ganglion cells can tolerate. This eventually results in the death of the ganglion cells and their axons, which comprise the optic nerve. Thus, less visual impulses are able to reach the brain. (Montgomery,1998).

In advanced glaucoma, the visual field in the peripheral retina is decreased or lost, leaving vision in the central retina (macular area) intact. This results in “tunnel vision.” Elevated eye pressure occurs when too much aqueous fluid enters the eye and not enough of the aqueous fluid is leaving the eye. Eye pressure can be measured by performing a “tonometry” test. (Montgomery,1998).

Normally, fluid enters the eye by seeping out of the blood vessels in the ciliary body. This fluid eventually makes its way past the crystalline lens, through the pupil (the central opening in the iris), and into the irido-corneal angle, the anatomical angle formed where the iris and the cornea come together. Then the fluid passes through the trabecular meshwork in the angle and leaves the eye, via the canal of Schlemm. (Montgomery,1998).

If the rate of aqueous fluid is entering the eye is too great, or if the trabecular meshwork “drain” gets clogged (for instance, with debris or cells) so that the fluid is not leaving the eye quickly enough, the pressure builds up in what is known as “open angle glaucoma.” It is more common with increasing age. (Montgomery,1998).

Open angle glaucoma, which tends to be a chronic and painless condition, also can be caused when the posterior portion of the iris,

surrounding the pupil, somehow adheres to the anterior surface of the lens (creating a “pupillary block”). This can prevent intraocular fluid from passing through the pupil into the anterior chamber. (Montgomery,1998).

On the other hand, if the angle between iris and the cornea is too narrow, or is even closed, then the fluid backs up because it cannot flow out of the eye properly. This causes an increased intraocular pressure in what is known as “closed angle glaucoma.” Typically, there is an acute (sudden), painful onset. It can be accompanied by the appearance of rainbow-colored rings around white lights. (Montgomery,1998).

An internal pressure more than that which the eye can tolerate can deform the lamina cribrosa, the small cartilaginous section of the sclera at the back of the eye through which the optic nerve passes. A deformed lamina cribrosa seems to “pinch” nerve fibers passing through it, eventually causing axon death. Untreated glaucoma eventually leads to optic atrophy and blindness. (Montgomery,1998).

Eye pressure is measured by using a “tonometer” (with the test being called “tonometry”), and the standard tonometer generally is considered to be the “Goldmann tonometer.” The normal range of intraocular pressure (IOP) is 10 mm Hg to 21 mm Hg, with an average of about 16 mm Hg. Typically, eyes with intraocular pressure measurements of 21 mm Hg or higher, using a Goldmann tonometer, are considered to be “ocular hypertensive” and are suspect for glaucoma. (Montgomery,1998).

However, although glaucoma typically is associated with elevated IOP, the amount of pressure which will cause glaucoma varies

from eye to eye and person to person. Many people with glaucoma actually have IOP's in the normal range ("low tension" glaucoma), possibly indicating that their lamina cribrosas are too weak to withstand even normal amounts of pressure. Conversely, many people with IOP's which would be considered high have no evidence of glaucomatous damage. (Montgomery,1998).

Glaucomatous changes in the optic disk (optic nerve head) usually can be detected over time. If the optic cup within the optic disk increases in size over a period of months or years, if notching is observed anywhere around the nerve head rim, and/or if an asymmetry is observed between the optic cups of the two eyes, then that person may be considered to be a "glaucoma suspect." In glaucoma, optic nerve rim atrophy and/or notching, with a corresponding visual field decrease, usually will occur in this order:

Table number 2-1 Optic Nerve Quadrant and their Visual Field Loss

Optic Nerve Quadrant	Visual Field Loss
1. Inferior Quadrant	Superior Field
2. Superior Quadrant	Inferior Field
3. Temporal Quadrant	Nasal Field
4. Nasal Quadrant	Temporal Field

(Montgomery,1998).

Visual field loss, caused by optic nerve damage, is measured by using a "visual field analyzer" or "perimeter," especially by measuring and comparing changes over time. The procedure is known as

“perimetry.” Most field loss due to glaucoma usually is not even measurable until 25% to 40% of the optic nerve’s axons have been destroyed. (Montgomery,1998).

Studies seem to show that the first fibers to die are the larger fibers, which primarily carry form and motion information, rather than the smaller fibers, which primarily detect light. Therefore, pattern discrimination perimetry (PDP), which requires detection of both form and motion, may be a better test for early glaucoma than conventional perimetry, which requires detection of spots of light. (Montgomery,1998).

In pattern discrimination perimetry (PDP), various locations of the retina are stimulated with a checkerboard pattern on a background of randomly moving dots. The more random the dot movements, the more difficult it is to continue to perceive the checkerboard pattern. Even a normal eye eventually will not be able to see the checkerboard when the dot movement is random enough. (Montgomery,1998).

The more advanced the stage of glaucomatous nerve damage, the less “noisy” the dots need to be for the checkerboard pattern to be indistinguishable from the background of moving dots. In effect, the PDP seems to be more sensitive than a standard perimeter in detecting early glaucomatous visual field losses. (Montgomery,1998).

Typically, the elevated pressure in open angle glaucoma can be controlled using glaucoma medications, which either decrease the production of aqueous fluid or else increase its outflow from the eye. However, closed angle glaucoma often requires surgical intervention to be controlled. (Montgomery,1998).

2-3-6 Cataract:

Normally, all the layers of the crystalline lens are clear, and light passes through it unobstructed. However, with age or due to certain systemic diseases, as well as with a cumulative absorption of ultraviolet radiation over many years, the lens material can become cloudy, yellow, brown, and even opaque. Anything in the lens which obstructs entering light is referred to as a “cataract.” (Montgomery,1998).

More than 50% of people over the age of 60 have some form of a cataract. It has been said that if one lives long enough, he/she will develop a cataract. Even some infants are born with a “congenital” cataract which, if left untreated, can cause permanent visual impairment or blindness, even if the cataract is removed years later. (Montgomery,1998).

It is not possible to remove a primary cataract without irreparably damaging the crystalline lens within which the cataract is contained. A laser cannot be used successfully to remove a cataract, except as described later (in the case of a secondary cataract). Therefore, cataract surgery involves removing most or all of the lens of the eye and replacing it with an artificial “intraocular lens” or “lens implant,” made of a hard plastic (polymethyl methacrylate or PMMA), silicone, acrylic, or hydrogel material.(Montgomery,1998).

An “extracapsular” cataract extraction (ECCE) is the routine type of cataract removal. In an ECCE procedure, an opening is made in the front of the lens capsule. Through this opening, the lens nucleus is removed, either as a whole or by dissolving it into tiny pieces and vacuuming out the pieces, a procedure called “phacoemulsification.” Next, the lens cortex also is sucked out, leaving the lens capsule in place,

and into the lens capsule is inserted the artificial lens implant. (Montgomery,1998).

Prior to the 1980's, the entire crystalline lens was removed in a cataract surgery, called an "intracapsular" cataract extraction (ICCE). Usually, this was performed using "cryoextraction," where a cryoprobe froze the entire lens, permitting its complete removal. Now, in the unusual case of an intracapsular lens extraction, or ICCE, the implant lens is placed in front of the iris, rather than behind it, because there is no lens capsule to hold the implant in place. Rarely is this procedure done anymore. (Montgomery,1998).

Approximately 1-2% of post-cataract extraction patients develop swelling in the area of the retina responsible for central vision (the macula). This swelling occurs in cystoid spaces, and is referred to as cystoid macular edema. After an initial improvement following surgery, these patients subsequently will describe blurred vision. Cystoid macular edema can occur as early as days, or as late as several years, following surgery. Treatment options include observation, topical therapy, periocular injections, and surgery. (Montgomery,1998).

Naturally occurring carotenoids in the crystalline lens—lutein and zeaxanthin (molecular cousins of beta carotene and vitamin A)—have been shown to reduce the risk of cataracts. These pigments act as antioxidants within the lens, inhibiting the formation of free radicals, which can damage lenticular material and contribute to the development of cataracts. (Montgomery,1998).

Thus, it may be that the greater the amount of antioxidants such as lutein and zeaxanthin in the system, the less the risk of cataract formation. These two antioxidants are found particularly in yellow fruits and in green leafy vegetables (especially xanthophyll-rich vegetables

such as spinach, kale, collard greens, and broccoli), in eggs, and as nutritional supplements. (Montgomery,1998).

2-3-7 Secondary cataract:

Not uncommonly, following an “extracapsular” cataract extraction (ECCE), a few cells of the crystalline lens cortex remain adhered to the inner surface of the posterior lens capsule. After a few weeks or months, these cells can become opaque, resulting in a secondary cataract. Fortunately, the eye does not have to be reopened for this simple cataract to be removed. (Montgomery,1998).

Rather, a YAG (yttrium aluminum garnet) laser is used, in a procedure taking only a few minutes, to fire through the clear cornea and pupil and to obliterate the secondary cataract (and a small portion of the capsule behind it). This enables light to pass into the eye again, unobstructed. If this laser procedure is successful, a cataract never again should pose a problem for that eye. (Montgomery,1998).

2-3-8 Retinal detachment (RD):

Normally, with age, the vitreous gel collapses and detaches from the retina—an event known as a posterior vitreous detachment. Occasionally, however, the vitreous membrane pulls on and creates a tear in the retina. Vitreous fluid can seep into or beneath the retina, detaching it from the pigmented epithelium underneath. (Montgomery,1998).

When a retinal detachment occurs, a shower of floaters may be observed by the person experiencing the detachment. These are thousands of blood cells being liberated from a tiny blood vessel which has been broken due to the retinal tear or detachment. Sometimes the floaters are described as being like a “shower of pepper” before the eyes. (Montgomery,1998).

Sudden flashes of light, as well as a “web” or “veil” in front or else in the periphery of the eye, also may appear in conjunction with the onset of floaters. The retinal tear and subsequent detachment must be repaired as soon as possible, usually with one of these procedures:

sealing it using an argon laser (“photocoagulation”), freezing it (“cryotherapy” or “cryopexy”), securing it, after cryotherapy, with a tiny belt around the equator of the eye (“scleral buckle” surgery), injecting a gas bubble into the eye (in conjunction with photocoagulation or cryopexy) so that the bubble rests against the hole or tear (“pneumatic retinopexy”), requiring the person to keep his/her head in the same position for several days, or removing the vitreous gel and filling the eye with a gas bubble or silicon oil (“vitrectomy”). (Montgomery,1998).

If the tear and detachment are not repaired, permanent vision loss can result. The worst vision loss occurs if the macula becomes detached. (Montgomery,1998).

2-3-9 Retinitis pigmentosa (RP):

One of the most devastating conditions affecting the rods is “retinitis pigmentosa,” an inherited disorder in which the rods gradually degenerate. With time, night vision is severely affected. Eventually, all peripheral vision will continue to be destroyed, to the point where only central or “tunnel” vision remains. (Montgomery,1998).

There is no known treatment. However, since blue and ultraviolet light may make aggravate the condition, amber-colored glasses with an ultraviolet absorption coating, worn during the day, may slow down the disease process. (Montgomery,1998).

Studies have shown that retinitis pigmentosa is caused by mutations in the rhodopsin gene, the peripherin gene, and possibly in other genes within the rod. Mutations in the peripherin gene also may be the cause of another devastating retinal disorder: “macular dystrophy.” (Montgomery,1998).

2-3-10 Diabetes mellitus:

Diabetes is a condition where the amount of glucose in your blood is too high because the body cannot use it properly. (Kronenberg , 2011)

This is because your pancreas doesn't produce any insulin, or not enough insulin, to help glucose enter your body's cells – or the insulin that is produced does not work properly (known as insulin resistance).

Insulin is the hormone produced by the pancreas that allows glucose to enter the body's cells, where it is used as fuel for energy so we can work, play and generally live our lives. It is vital for life.

Glucose comes from digesting carbohydrate and is also produced by the liver. (Kronenberg , 2011)

If you have diabetes, your body cannot make proper use of this glucose so it builds up in the blood and can't be used as fuel.

There are three main types of diabetes mellitus:

Type **1** DM results from the body's failure to produce insulin. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes".

Type **2** DM results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes also with an absolute insulin

deficiency. This form was previously referred to as non insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes".

Gestational diabetes, is the third main form and occurs when pregnant women without a previous diagnosis of diabetes develop a high blood glucose level . (Kronenberg , 2011)

2-3-10 -1The Causes :

Table number 2-1 The Causes Feature Comparison of type 1 and 2 diabetes . (Kronenberg , 2011)

Comparison of type 1 and 2 diabetes		
Feature	Type 1 diabetes	Type 2 diabetes
Onset	Sudden	Gradual
Age at onset	Mostly in children	Mostly in adults
Body size	Thin or normal	Often obese
Ketoacidosis	Common	Rare
Autoantibodies	Usually present	Absent
Endogenous insulin	Low or absent	Normal, decreased or increased
Concordance in identical twins	50 %	90 %
Prevalence	~10 %	~90 %

2-3-10 -2 Signs and symptoms :



Fig number 2-6 shows the most significant symptoms of diabetes(Kronenberg , 2011)

The classic symptoms of untreated diabetes are weight loss, polyuria (frequent urination), polydipsia (increased thirst), and polyphagia (increased hunger). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes. (Kronenberg , 2011)

Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. Blurred vision is a common complaint leading to a diabetes diagnosis. A number of skin rashes that can occur in diabetes are collectively known as diabetic_dermadromes. (Kronenberg , 2011)

2-3-10 -3 Complications of diabetes mellitus

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10-20), but may be the first symptom in those who have otherwise not received a diagnosis before that time. (Kronenberg , 2011)

The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease and about 75% of deaths in diabetics are due to coronary artery disease. Other "macrovascular" diseases are stroke, and peripheral vascular disease. The primary microvascular complications of diabetes include damage to the eyes, kidneys, and nerves. Damage to the eyes, known as diabetic retinopathy, is caused by damage to the blood vessels in the retina of the eye, and can result in gradual vision loss and potentially blindness. Damage to the kidneys, known as diabetic nephropathy, can lead to tissue scarring, urine protein loss, and eventually chronic kidney disease, sometimes requiring dialysis or kidney transplant. Damage to the nerves of the body, known as diabetic neuropathy, is the most common complication of diabetes. The symptoms can include numbness, tingling, pain, and altered pain sensation, which can lead to damage to the skin. Diabetes-related foot problems (such as diabetic foot ulcers) may occur, and can be difficult to treat, occasionally requiring amputation. Additionally, proximal diabetic neuropathy causes painful muscle wasting and weakness. (Kronenberg , 2011)

There is a link between cognitive deficit and diabetes. Compared to those without diabetes, those with the disease have a 1.2 to 1.5 fold greater rate of decline in cognitive function. (Kronenberg , 2011)

2-3-10 -4 The effect of diabetes on eyes:

The National Eye Institute outlines the damage that diabetes causes in the eyes. Retinopathy, the most common disorder, occurs when the vessels in the eye do not adequately provide nourishment to the retina. Sometimes new vessels form on the retina to compensate, but these can interfere with vision. Diabetics are also at increased risk for cataracts and also for glaucoma, in which eye pressure increases as the eye fails to drain properly. (Kronenberg , 2011)

2-3-10 -5The effect of diabetes on optic nerve :

The increased pressure damages the optic nerve and can cause blindness. Additionally, nerve damage associated with diabetes. (Kronenberg , 2011)

Diabetes is a common cause of damage to the optic nerve. Diabetes is a condition where the body's incapable of correctly processing and using sugar. The body's incapable of metabolizing sugars properly, resulting in high levels of glucose within the blood. When left untreated for prolonged periods, blindness can result from damage to the optic nerve. Blurred vision usually occurs prior to complete vision loss in diabetes patients. (Kronenberg , 2011)

2-4 Orbit Imaging technique :

In order to imaging the optic nerve we usually use Computed Tomography (CT) or Magnetic Resonance Imaging (MRI)

2-4-1 Orbit C.T technique :

2-4-1-1 Indications:

Structural diseases of the orbits and orbital contents, trauma and foreign body.

2-4-1-2 Image criteria and Slice thickness:

Visualization of entire orbits, osseous walls.

Volume of investigation: from 0.5 cm below to 0.5 cm above the orbital cavity.(Henwood, 1999).

- 2-5 mm.

2-4-1-3 Patient position:

Supine for axial scan; supine or prone for coronal scans.

Lateral scout view is obtained.

Gantry tilt: 6-10 degree from OM or parallel to optic nerve for axial scan ; according to the patient position for coronal scanning or x-ray beam parallel to the IOML.

Axial scan should cover all orbital region. (Henwood, 1999).

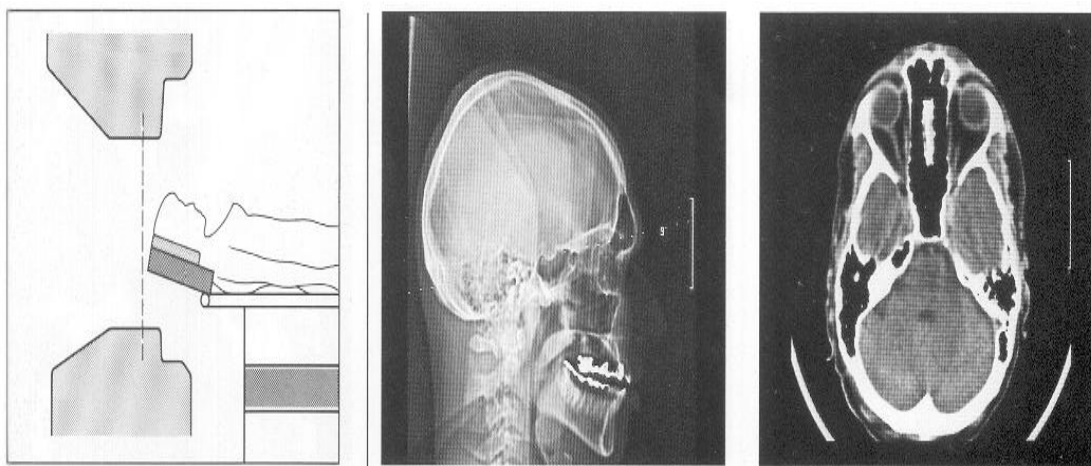


Fig number 2-7 shows the Axial C.T scan.(Henwood, 1999).

Slice thickness 2-5 mm . Feed 2-5 mm

Some institutions prefer to used 1-mm axial slices with reformatting to produce coronal images. This technique is useful when coronal images can not be obtained; however, reformatted images tend to be of poor quality. (Henwood, 1999).

2-4-1-5 coronal scan

Patients supine, head rest in coronal head holder, with neck hyper extended.

Lateral scout view is obtained.

coronal scan should cover all orbital region.

Slice thickness 2-5 mm, spacing 2-5mm. (Henwood, 1999).

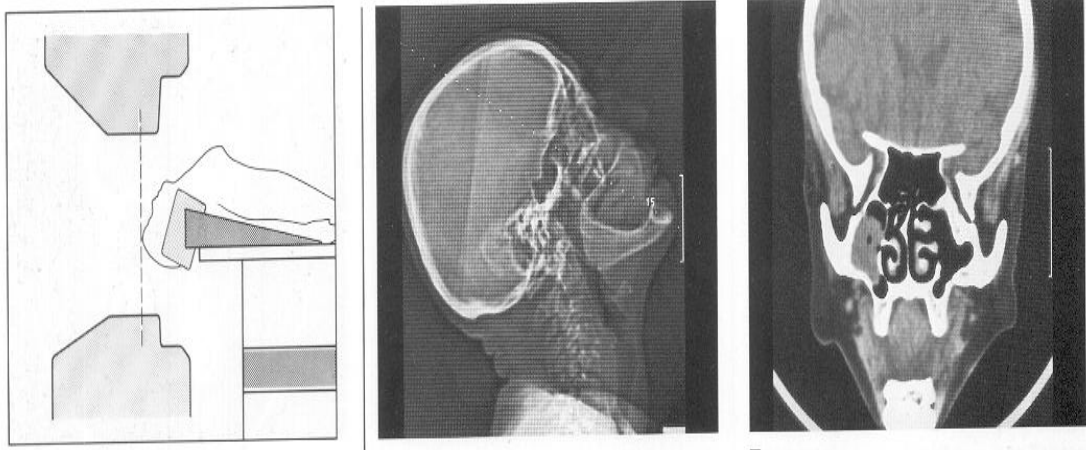


Fig number 2-8 shows the C.T coronal scan supine.(Henwood, 1999).

Slice thickness 2-5mm. Feed 2-5mm

Coronal prone

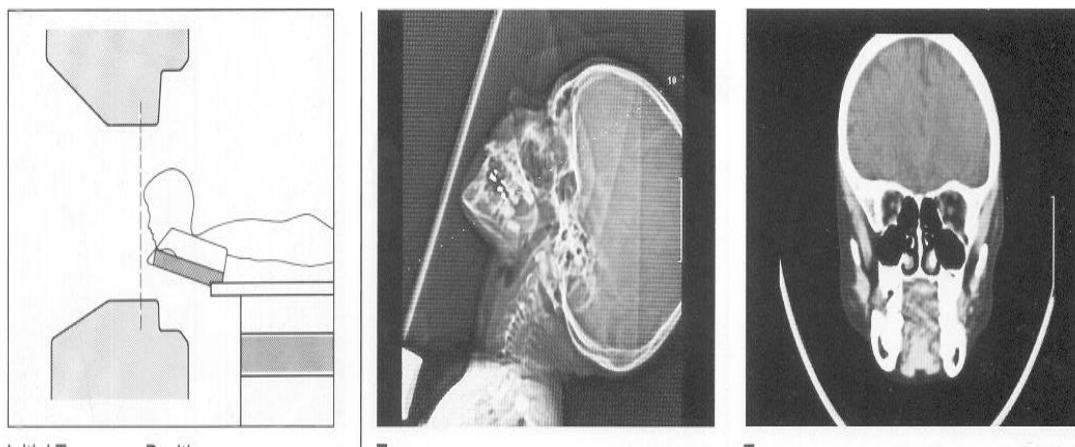


Fig number 2-9 shows the C.T coronal scan prone.(Henwood, 1999).

Slice thickness 2-5mm. Feed 2-5mm

2-4-2 Orbit MRI technique :

2-4-2-1 Common indications:

Proptosis .

Visual disturbance.

Evaluation of orbital or ocular mass lesion.

2-4-2-2Equipment :

Small surface coil for globe and orbit ,Quadrature head coil for orbital apex, chiasm and intra-cranial optic pathways, Immobilization straps and foam pads. (Westbrook, 2008).

2-4-2-3Patient positioning

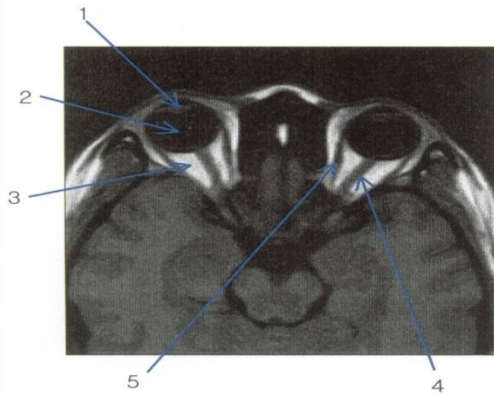
The patient lies supine on the examination couch . Both orbits are usually examined at the same time . If surface coils are used , these are placed over-each orbit but should not touch the patient . Special holder are often provided by the manufactures to enable the coils to be placed anteriorally over the eyes . Ensure that the receiving side of the coil.

Is faces the orbits ,i.e. toward the table . the patient assumes a fixed gaze , straight ahead , with the eyes open . This enables the patient to focus and keeps the eyes still . Thereby reducing motion artifact . Any eye make – up is removed prior the examination as this causes image artifact and patient discomfort especially if it contains metal . (Westbrook, 2008).

The patient is positioned so that the longitudinal alignment light lies in the midline , and the horizontal alignment light passes through the orbits . If surface coils are used , this corresponds to the centre of the coils .Straps and foam pads are used for immobilization . (Westbrook, 2008).

Orbit

1. Anatomical call-off :



- 1. Lens
- 2. Globe
- 3. Peri-orbital fat
- 4. Optic nerve
- 5. Medial rectus muscle



- 2. Coil : Head coil
- 3. Center : Glabella (The head is adjusted so that the inter-papillary line is parallel to the couch and head is straight)

Fig number 2-10 shows the orbit anatomy and head coil(Westbrook, 2008).

2-4-2—4 Suggested protocol:

Axial SE T1,

Sagittal SE T1,

Coronal SE T1.

Note, if optic neuritis is suspected scan the brain.

(Westbrook, 2008).

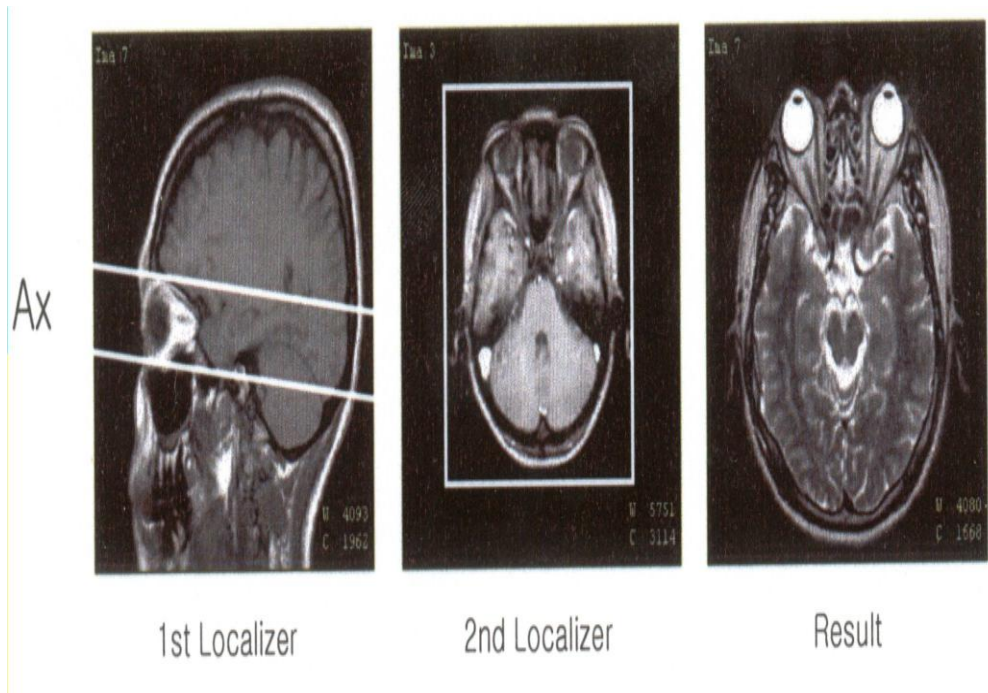


Fig number 2-11 shows the Axial scan (Westbrook, 2008).

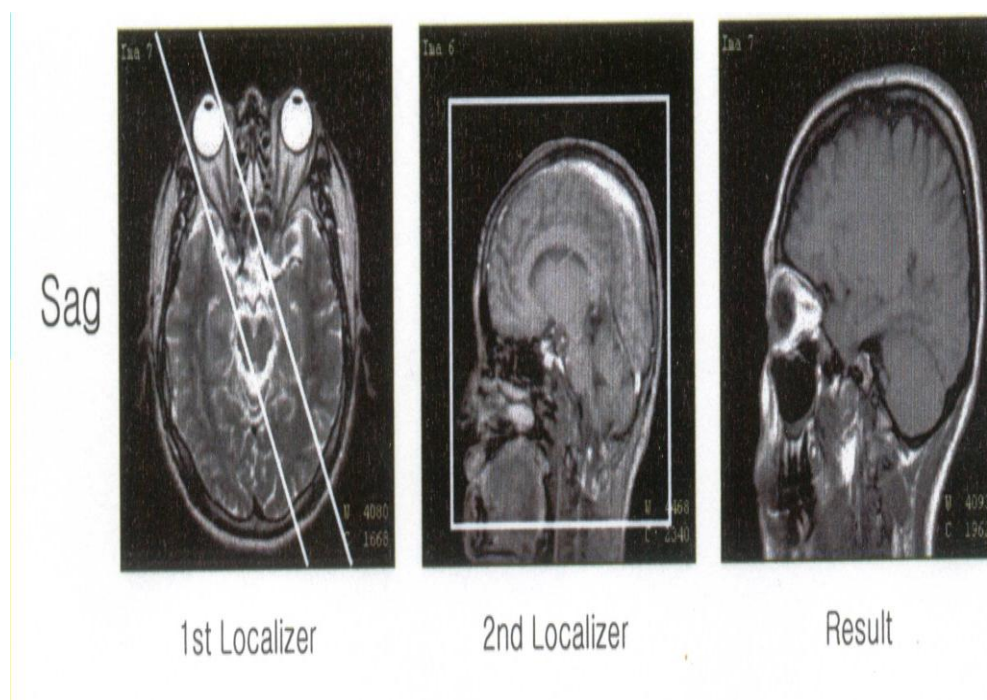


Fig number 2-12 shows the Sagittal scan(Westbrook, 2008).

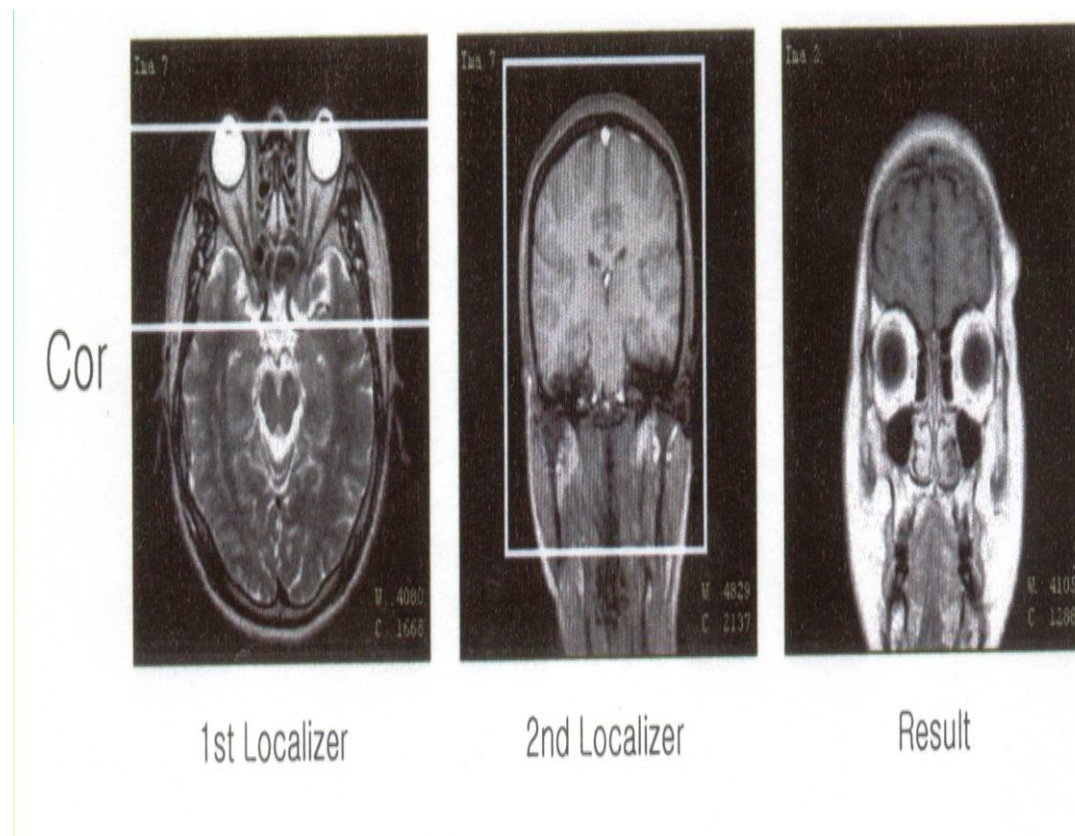


Fig number 2-12 shows the Coronal scan (Westbrook, 2008).

2-5 Previous studies:

Benevento , etal 2011 studied Optic Nerve Measurements in Normal Human Eyes by MRI and they used Coronal MRI imaging of normal human eyes it's showed an average ONSD range of 4.0 – 6.0 with SD 0.5mm, and an average OND range of 2.6 – 4.0 with SD 0.3mm. there range is consistent with published data on the ONSD. However, we are not aware of any published data on the OND.

Newcombe, etal 2008 Used T2-weighted magnetic resonance imaging of the optic nerve sheath to detect raised intracranial and they

results that the Measurement of ONSD was possible in 95% of cases. The ONSD was significantly greater in TBI patients with raised ICP (>20 mmHg; ± 6.31 , 0.50 mm, 19 measures) than in those with ICP of 20 mmHg or less (± 5.29 , 0.48 mm, 26 measures; $P < 0.0001$) or in healthy volunteers (± 5.08 , 0.52 mm; $P < 0.0001$). There was a significant relationship between ONSD and ICP ($r = 0.71$, $P < 0.0001$). Enlarged ONSD was a robust predictor of raised ICP (area under the receiver operating characteristic curve = 0.94), with a best cut-off of 5.82 mm, corresponding to a negative predictive value of 92% , and to a value of 100% when ONSD was less than 5.30 mm.

Brex , etal 2011 describe an MRI technique for quantifying optic nerve atrophy resulting from a single episode of unilateral optic neuritis. They imaged 17 patients, with a median time since onset of optic neuritis of 21months (range 3– 81months), using a coronal-oblique fat-saturated short-echo fast fluid-attenuated inversion-recovery (sTE fFLAIR) sequence. The mean cross-sectional area of the intraorbital portion of the optic nerves was calculated by a blinded observer from five consecutive 3 mm slices from the orbital apex forwards using a semiautomated contouring technique and compared with data from 17controls. The mean optic nerve area was 11.2mm^2 in the affected eye of the patients, 12.9mm^2 in the contralateral eye ($P = 0.006$ compared to the affected eye) and 12.8mm^2 in controls ($P = 0.03$ compared to the affected eyes). There was a significant negative correlation between disease duration and the size of the affected optic nerve ($r = -0.59$, $P = 0.012$). The measurement coefficient of variation was 4.8% . The sTE fFLAIR sequence enables measurement of optic nerve area with sufficient reproducibility to show optic nerve atrophy following a single episode of unilateral optic neuritis. The correlation of increasing optic nerve atrophy with disease duration would be consistent with ongoing axonal loss in a

persistently demyelinated lesion, or Wallerian degeneration following axonal damage during the acute inflammatory phase

Hickman, et al 2009 investigate optic neuritis as a model for atrophy in multiple sclerosis (MS) lesion they performed serial magnetic resonance imaging (MRI) on 10 patients with a history of optic neuritis using a fat saturated short-echo fast fluid-attenuated inversion recovery (sTE fFLAIR) sequence. The first study was performed a median of 19.5 months after the onset of optic neuritis and the second 1 year later. Using a computer-assisted contouring technique, a blinded observer calculated the mean area of the intra-orbital optic nerves. The mean area of affected optic nerves decreased over 1 year by 0.9 mm² from 11.1 to 10.2 mm² (p=0.01). Poor visual acuity and decreased visual-evoked potential (VEP) amplitude were associated with atrophy. These findings suggest that atrophy is a feature of focal demyelinating lesions, it may evolve over several years, and may have functional significance. Optic neuritis provides a model to study the effect of inflammatory demyelination through the ability to accurately measure visual function and to visualize and measure the optic nerves using magnetic resonance imaging.

Newman, et al 2009 evaluate the utility of measuring the optic nerve sheath diameter in children with shunted hydrocephalus, suspected of having raised intracranial pressure used 23 children with shunted hydrocephalus were examined, six had well controlled ICP, 17 however manifested symptoms suggestive of intracranial hypertension. A clinical history was taken from all patients and their parents or carers. The shunt valve was examined clinically, and signs of raised intracranial pressure were sought. Ultrasound examination was performed in both eyes to measure the optic nerve sheath diameters 3 mm behind the globe. These measurements were compared with control data obtained from 102

children who attended the radiology department for unrelated renal ultrasound examination. Control data are the upper limit of normal for optic nerve sheath diameter is 4.5mm (measured 3mm behind the globe) in patients over 1year of age, and 4.0mm in children less than 1year of age. Those patients with functioning ventriculoperitoneal shunts had a mean optic nerve sheath diameter of 2.9(SD 0.5) mm; those with raised intracranial pressure had a mean optic nerve sheath diameter of 5.6(0.6) mm ($p<0.0001$). These results confirm that optic nerve sheath diameters in excess of the control data are strongly suggestive of raised intracranial pressure.

Chapter three: Materials and methods

Chapter three

Materials and methods

3-1 Material :

3-1-1 Study population :

The total sample of patients were **50** (**30** of them had diabetes and **20** as control group in both genders) their ages between 30 – 90 years old all were under went MRI orbits .

3-1-2 Area , duration of study and data analysis :

The study has been carried out during the period from June to November **2014** at Garash international hospital. The data was analyzed using Excel program and SPSS version 16 .

3-2 Methods:

The patients lies supine on the examination couch . Both orbits are examined . used head coil , these are placed over-each orbit but should not touch the patient . The patients are positioned so that the longitudinal alignment light lies in the midline , and the horizontal alignment light passes through the orbits . Straps and foam pads are used for immobilization .

3-3 Used Machine and Methods of measurements :

Toshiba 1.5 Tesla .

We measured the length of the optic nerve (the area from the posterior part of the eye ball to the optic chiasma) and the area between the two border of optic canal (widths of the optic nerve) .

3-4 Used protocol :

Sagittal oblique T2 Tr 5000 Te 105 for the length .

Coronal T2 Tr 5000 Te 105 for the widths .

Chapter Four: The Results

Chapter Four

Results

The following tables and figures presented the data obtained from 30 patients who were examined for sagittal and coronal MRI for orbits, the optic nerve width and length was measured , patients age ,gender ,diabetis duration have also been examined. The data was analyzed using Excel program and SPSS version 16 for significances of tests was used. Frequency tables mean and standard deviations were presented.

Table 4 - 1 the diabetic patient classification according to gender

Gender	Frequency	Percentages%
Male	15	50%
Female	15	50%
Total	30	100%

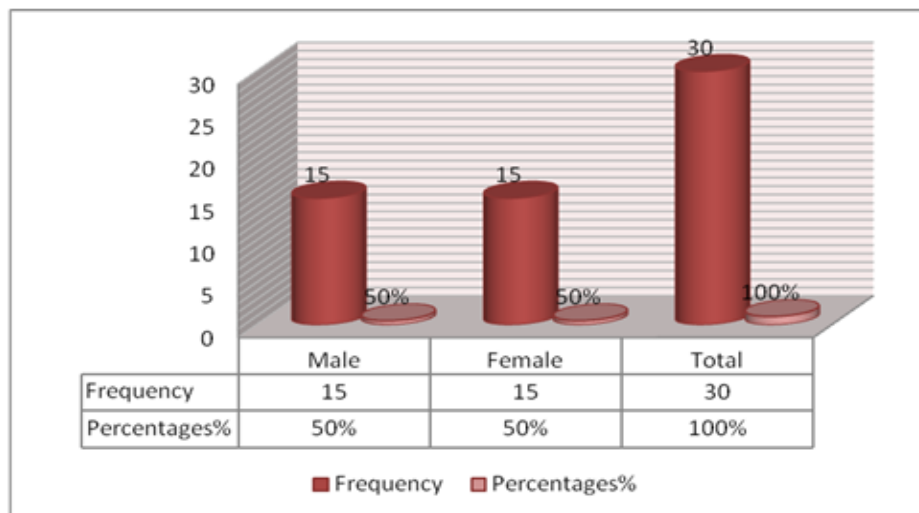


Figure 4 - 1 Gender Distribution

Table 4 - 2 Classification of Diabetes Type

Type	Diabetes Type1	Diabetes Type2
Frequency	20	10
Percentage	66.7%	33.3%

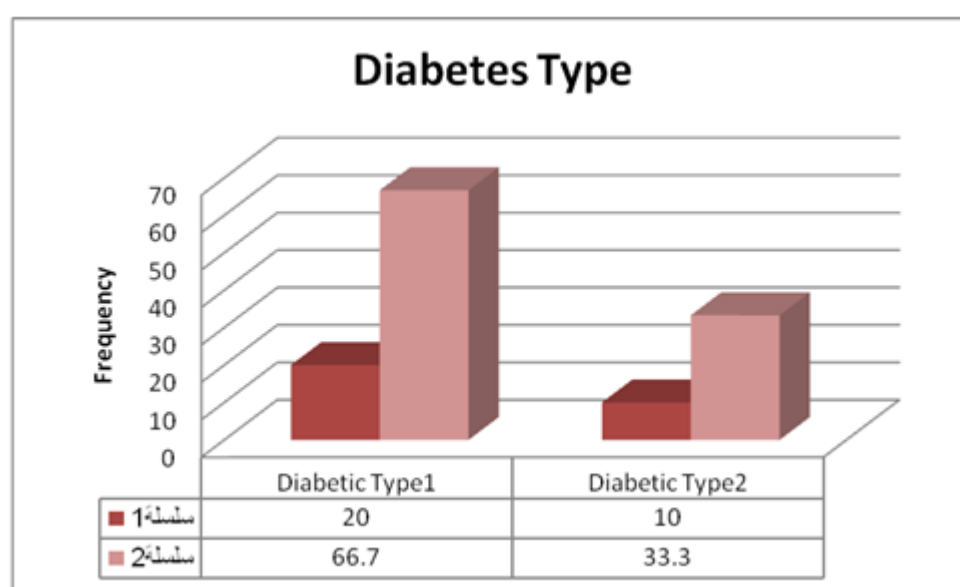


Figure 4 - 2 Diabetes type Distribution

Table 4 - 3 The mean and standard deviation of the variables for control group

Age	Coronal Measurements	Sagittal Measurements
52.8	0.49	3.27
± 15.51	± 0.08	± 0.22

Table 4 – 4 The mean and standard deviation of the variables for diabetic patients group

Age	Coronal Measurement	Sagittal Measurement
62.67	0.3	2.88
± 15.1	± 0.04	± 0.1

Table 4 - 5 P value (degree of significance between the control group and diabetic patient group)

Test	Age	Coronal Measurement	Sagittal Measurement
P-value	0.000	0.032	0.002

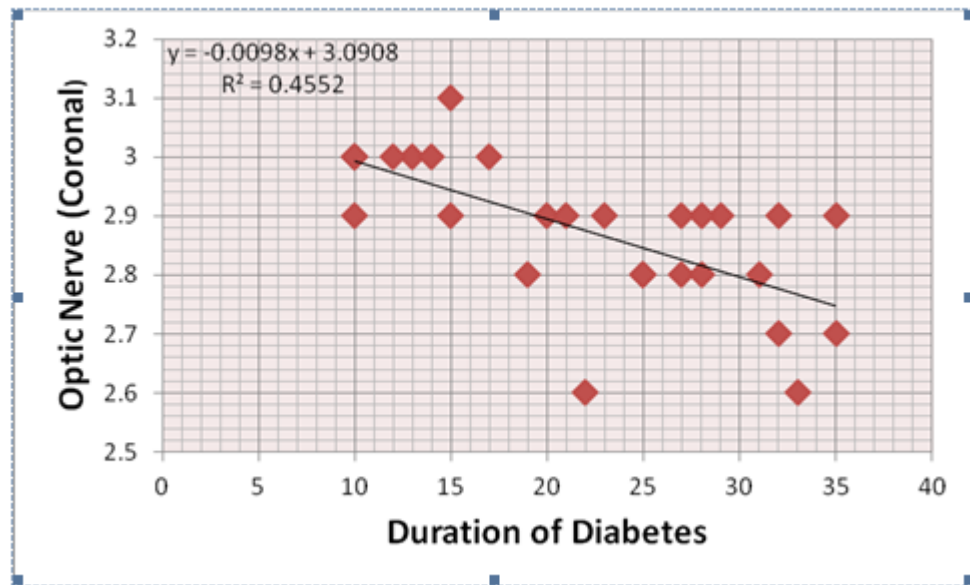


Figure 4-3 Scatter plot diagramme shows the linear relationship between the duration of diabetes and optic nerve coronal measurement ,when the duration of diabetic increased the measurements decreased by 0.009 starting from 3.09 $r^2=0.455$

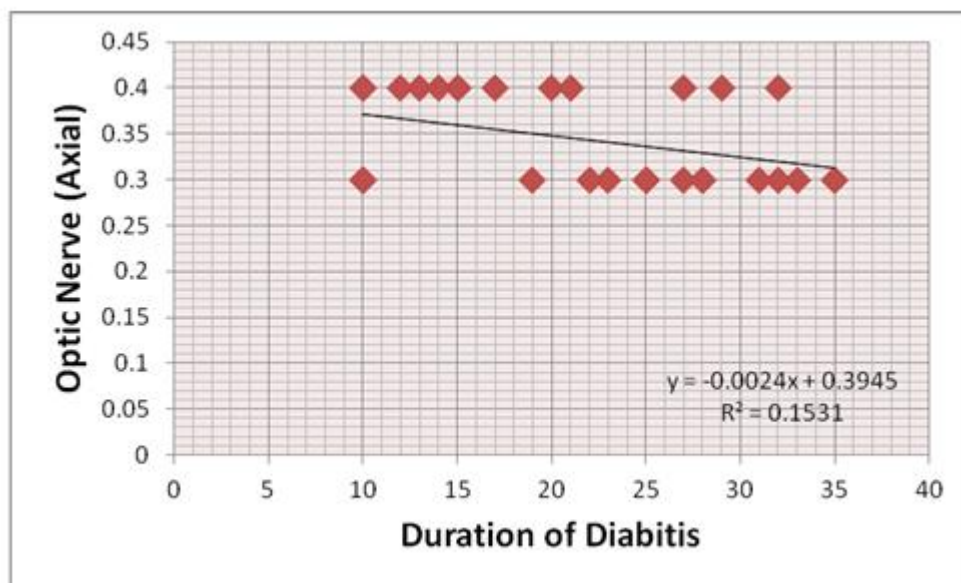


Figure 4-4 Scatter plot diagramme shows the linear relationship between the duration of diabetes and optic nerve sagittal measurement ,when the duration of diabetic increased the measurements decreased by 0.002 starting from 3.94 $r^2=0.153$

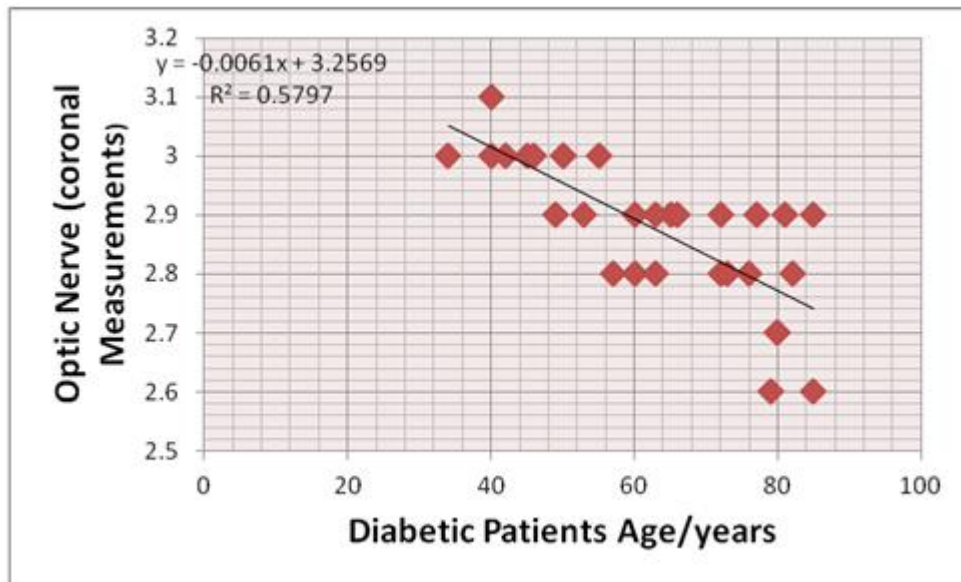


Figure 4-5 Scatter plot diagramme shows the linear relationship between the Patients age and optic nerve coronal measurement ,when the age increased the measurements decreased by 0.006 starting from 3.256 $r^2=0.579$

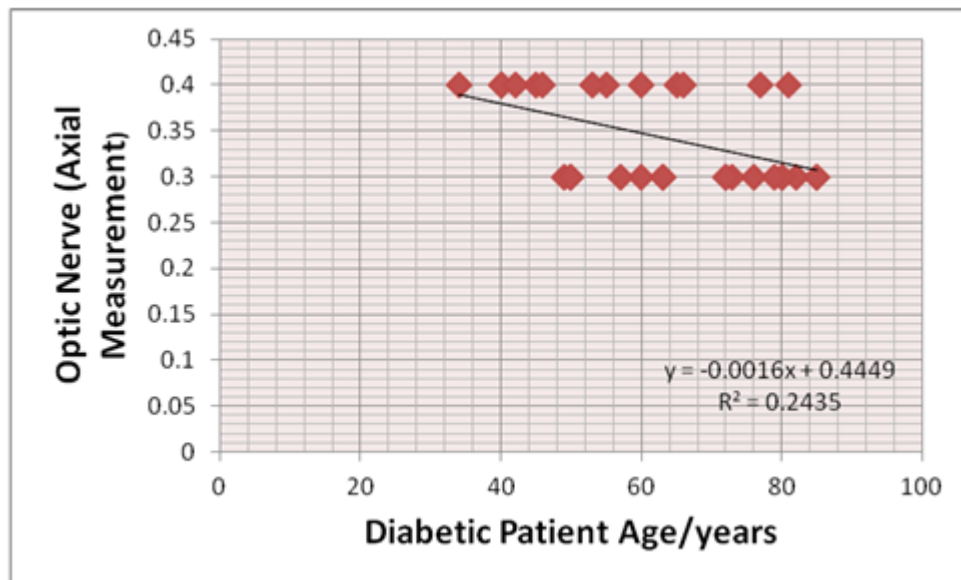


Figure 4 - 6 Scatter plot diagramme shows the linear relationship between the Patients age and optic nerve sagittal measurement ,when the age increased the measurements decreased by 0.001starting from 0.4 $r^2=0.243$

Chapter five: The discussion, conclusion and Recommendations

Chapter five

The discussion, Conclusion and Recommendations

5-1 The Discussion:

In order to assess the The effect of diabetes on optic nerve we analyzed 30 diabetic patients . was established in 20 cases.

Compared to controls, statistical significance was demonstrated regarding.

Table 4.1 showed the diabetic patient classification according to gender to 50% Male and 50% Female .

Table 4.2 showed the Classification of Diabetes Type in the sample Diabetes Type 1 20 patient that mean 66.7% and Diabetes Type2 10 patient that mean 33.3% .

Table 4.3 showed The mean of the Age for control group is 52.8 and the standard deviation is ± 15.51 , The mean of the Coronal Measurements for control group is 0.49 and the standard deviation is ± 0.08 and The mean of the sagittal Measurements for control group is 3.27 and the standard deviation is ± 0.22 near to J.D. Benevento , etal 2011 study of Optic Nerve Measurements in Normal Human Eyes by MRI and they used Coronal MRI imaging of normal human eyes it's showed an average ONSD range of 4.0 – 6.0 with SD 0.5mm, and an average OND range of 2.6 – 4.0 with SD 0.3mm.

Table 4.4 showed The mean of the Age for diabetic patients group is 62.67 and standard deviation is ± 15.1 , The mean of the Coronal Measurements for diabetic patients group is 0.3 and the standard deviation is ± 0.04 and The mean of the sagittal Measurements for diabetic patients group is 2.88 and the standard deviation is ± 0.1 near to FJ Newcombe, etal 2013 Used T2-weighted magnetic resonance imaging of the optic nerve sheath to detect raised intracranial (>20

mmHg; ± 6.31 ,0.50 mm, 19 measures) than in those with ICP of 20 mmHg or less (± 5.29 , 0.48 mm, 26 measures; $P < 0.0001$)

Table 4.5 showed The P value (degree of significance between the control group and diabetic patient group) of the Age is 0.000 , The P value of the Coronal Measurements is 0.032 and The P value of the Axial Measurements is 0.002 .

Figure 4.3 Scatter plot diagramme shows the linear relationship between the duration of diabetes and optic nerve coronal measurement ,when the duration of diabetic increased the measurements decreased by 0.009 starting from 3.09 $r^2 = 0.455$.

Figure 4.4 Scatter plot diagramme shows the linear relationship between the duration of diabetes and optic nerve sagittal measurement ,when the duration of diabetic increased the measurements decreased by 0.002 starting from 3.94 $r^2 = 0.153$.

Figure 4.5 Scatter plot diagramme shows the linear relationship between the Patients age and optic nerve coronal measurement ,when the age increased the measurements decreased by 0.006 starting from 3.256 $r^2 = 0.579$.

Figure 4.6 Scatter plot diagramme shows the linear relationship between the Patients age and optic nerve sagittal measurement ,when the age increased the measurements decreased by 0.001 starting from 0. 4 $r^2 = 0.243$.

5-2 The Conclusion :

The P value (degree of significance between the control group and diabetic patient group) of the Age is 0.000 , The P value of the Coronal Measurements is 0.032 and The P value of the sagittal Measurements is 0.002 and that is mean the diabetes effect the optic nerve diameter in length and widths .

From the coronal measurement of the optic nerve in diabetic patient group when the age increased the measurements decreased by 0.006 starting from 3.256 $r^2=0.579$ and From the sagittal measurement of the optic nerve in diabetic patient group when the age increased the measurements decreased by 0.001 starting from 0.4 $r^2=0.243$ **with is mean the patient age effect the optic nerve diameter in length and widths.**

The gender has no effect on the optic nerve diameter in diabetic patient cause they have equal rates .

From the coronal measurement of the optic nerve in diabetic patient group when the duration of diabetic increased the measurements decreased by 0.009 starting from 3.09 $r^2 = 0.455$ and From the sagittal measurement of the optic nerve in diabetic patient group when the duration of diabetic increased the measurements of the optic nerve decreased by 0.002 starting from 3.94 $r^2 = 0.153$ **and that is mean when the duration of diabetic increased the measurements of the optic nerve decreased .**

Globally, as of 2013 , an estimated 382 million people have diabetes worldwide, with type 2 diabetes making up about 90 % of the cases. This is equal to 3.3 % of the population, with equal rates in both women and men. In 2011 diabetes resulted in 1.4 million deaths worldwide, making it the 8 th leading cause of death. The number of people with diabetes is expected to rise to 592 million by 2035 .

5-3 The Recommendations:

1- Prevention is better than cure well all know it but I hope to apply it in our life by being a normal body weight, physical exercise, and following a healthy diet . Dietary changes known to be effective in helping to prevent diabetes include a diet rich in whole grains and fiber, and choosing good fats, such as polyunsaturated fats found in nuts, vegetable oils, and fish. Limiting sugary beverages and eating less red meat and other sources of saturated fat can also help in the prevention of diabetes. Active smoking is also associated with an increased risk of diabetes, so smoking cessation can be an important preventive measure as well .

2- Diabetes management concentrates on keeping blood sugar levels as close to normal ("euglycemia") as possible without causing hypoglycemia . This can usually be accomplished with diet, exercise, and use of appropriate medications (insulin in the case of type 1 diabetes; oral medications, as well as possibly insulin, in type 2 diabetes

3- Learning about the disease and actively participating in the treatment is vital for people with diabetes, since the complications of diabetes are far less common and less severe in people who have well-managed blood sugar levels .

4- Attention is also paid to other health problems that may accelerate the deleterious effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise.

5- **Anti-diabetic medication Metformin** is generally recommended as a first line treatment for type 2 diabetes, as there is good evidence that it decreases mortality. **Routine use of aspirin**, however, has not been found to **improve outcomes in uncomplicated diabetes**.

6- More precise measurements may be possible using dedicated multi-plane high resolution MRI images from larger sample size normal volunteers.

7- More measurements MRI images from larger sample size or diabetes patients .

5-4The References:

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5-5 The Appendix:

Sudan University of Science and Technology

Faculty of Graduation Studies

MSc Diagnostic Radiologic Technology

Research About:

Evaluation of The Optic Nerve Diabetic Patient Using MRI

The data collection questioner

No.	name	gender	age	measurement		Type of diabetes	Duration of diabetes	Effect on retina	Other finding
				Sagittal	coronal				
1									
2									
3									
4									
5									
6									
7									
8									
9									
10									
11									
12									
13									
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17									
18									
19									
20									
21									
22									
23									
24									
25									

26									
27									
28									
29									
30									

The control group questioner

No.	Name	Age	Gender	Measurements	
				Coronal	Sagittal
1					
2					
3					
4					
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					

From my measurement

