"RELATIONSHIP BETWEEN CONTRAST SENSITIVITY AND METABOLIC CONTROL IN DIABETICS WITHOUT RETINOPATHY"

By

DR.DEVI SINDHUJA.S, **M.B.B.S**

Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, CENTRE, TAMAKA, KOLAR

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OPHTHALMOLOGY

Under the guidance of

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RELATIONSHIP BETWEEN CONTRAST SENSITIVITY AND METABOLIC CONTROL IN DIABETICS WITHOUT RETINOPATHY

ABSTRACT BACKGROUND: Contrast sensitivity is the capacity to detect minor changes in luminance between regions that do not have distinct borders.However, visual acuity evaluates the sharpness of one's eyes at a specific distance. High contrast optotypes, which consist of black text on a white backdrop, are used to evaluate this. The fact that contrast sensitivity, a measure of visual quality, often decreases at an earlier age than visual acuity does is generally acknowledged. The current diabetes epidemic in India is largely attributable to diabetic retinopathy, a vision- threatening complication of diabetes mellitus. Research suggests that retinal neurodegeneration occurs in the early stages of diabetic retinopathy, despite the fact that the condition is characterised as a microvascular disease.Visual impairments such as reduced contrast sensitivity, changed colour perception, and altered temporal perception can be caused by retinal neurodegeneration. Such deficiencies might manifest prior to alterations in visual acuity and vascular architecture .Detecting early visual functional alterations in lower contrast situations may be beyond the capabilities of the current screening techniques for diabetic retinopathy, which primarily evaluate the morphologic integrity of the retina and retinal circulation. Therefore, the purpose of this study is to ascertain whether metabolic control and contrast sensitivity are associated in Type 2 diabetics who do not have retinopathy. METHODS: From August 2022 to December 2023, a cross-sectional study was carried out on a minimum of 53 patients who met the inclusion criteria at R. L. Jalappa Hospital and Research in Kolar. The study was approved by the Institutional Ethical Committee of Sri Devaraj Urs Medical College, and the subjects were provided written informed consent. The period of diabetes and diabetic control were recorded after collecting a brief history of the patient's eyes and overall health, as well as their treatment for the condition. Visual acuity, anterior and posterior segment evaluation, and demographic data were all part of the clinical examination that each patient underwent. A thorough evaluation of the retina was carried out in order to exclude the possibility of diabetic retinopathy. Exclusion criteria for participation in the study include the presence of intra retinal haemorrhage or microaneurysms, the first sign of diabetic retinopathy seen through ophthalmology.Estimations were made for fasting blood sugar, postprandial blood sugar, and glycated haemoglobin (HbA1C). Using the identical lighting conditions, we measured contrast sensitivity at a distance of 1 metre using a Pelli-Robson chart. Log contrast units are used to quantify contrast sensitivity. The contrast sensitivity is negatively correlated with the score. RESULTS: The present study enrolled 53 patients and their mean age was 60.16±7.80 years and ranged from 40 to 71 years.The mean fasting blood glucose was 127.28±40.75 mg/dl and the post prandial blood glucose was 174.37±59.52 mg/dl. The three months glycemic control marker HbA1c was assessed and the mean was 7.72±1.85.The mean duration of diabetes was 7.21±4.37 years The mean contrast sensitivity for right eye was 1.57±0.37 which indicated some visual impairment. Similarly the left eye

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ACKNOWLEDGEMET

First and foremost, I would like to thank the almighty for giving me the strength and abilityto carry out this study.

I take this opportunity to express my gratitude to my teacher and guide, **DR. B.O. HANUMANTHAPPA,** Professor, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar, for his invaluable guidance, constant support and encouragement during the course of study and completion of dissertation.

I would also like to thank **DR. SANGEETHA.T** Professor and Head of the Department of Ophthalmology, SriDevaraj Urs Medical College, Tamaka, Kolar for the constant guidance and advice. I would like to express my deepest gratitude to my Professors,

DR. MANJULA T R, DR. USHA B R, DR. RASHMI G; my Associate Professors**, DR. INCHARA.N, DR. CHAITRA MC,** and my Assisstant Professors

DR.ASAM NAVEENA,DR.APOORVA.N and Senior Resident DR.NARENDRAN B.S.,DR.ATHIRA.K,DR.SHWETHA.V Sri Devaraj Urs Medical College Tamaka, Kolar, for their encouragement and suggestions during the course of this study and post-graduation course. My gratitude and thanks to **DR.K.PRABHAKAR**, Principal, Sri Devaraj Urs Medical College Tamaka, Kolar, for letting me use the college and hospital facilities and resources.I would also like to thank my batchmates for all their help during this study and making my journey through it smooth and all my friends for their help and support. I would like to thank my parents, **DR.A.PARMIALA, DR.K.SHANTHA KUMAR** and whose countless sacrifices and blessings have made me who I am today. Thank you for always being with me and giving me strength at every step of my life.

I would like to express my heartfelt gratitude to my late Grandparents **MRS.KRISHNAVENI, MRS.LEKSHMY, MR.KULANDAIVELU, MRS.BAKKIYAM**. I feel your presence and blessings in every step I take. I am grateful to my grandfather **MR.K.ARUMUGAM** for his immense love and blessings. My sincere gratitude to my Uncles **K.RAJENDRAN, DR.A.HELENKUMAR, DR.PADMANABAN,** Aunts **A.VIMALA DEVI, DR.S.ARULJOTHI,** my brother **MR.S.ARUNAN**, my Cousin**s MRS.R.POORANI PRIYA, R.SRIHARISHGOPI, KARTHIK, ABINANDHAN** and other family members for their constant support, patience and understanding. for being my support in all the tough times. Last but not the least, I thank all my **PATIENTS** involved in this study, without whose cooperation, this dissertation would have never materialized. I sincerely thank my institute Sri Devaraj Urs Medical College, Tamaka, Kolar for giving me a wonderful foundation and forum of knowledge in the field of Ophthalmology, which will stand with me for the rest of my life

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LIST OF ABBREVIATIONS

CS	Contrast Sensitivity
CSF	Contrast Sensitivity Function
DCCT	Diabetes Control and Complications Trial
DERG	Digital Electroretinography
DM	Diabetes Mellitus
DR	Diabetic Retinopathy
ERG	Electroretinogram
FACT	Functional Acuity Contrast Test
HbA1c	Glycosylated Haemoglobin
IDDM	Insulin Dependent Diabetes Mellitus
IDF	International Diabetes Federation
MODY	Maturity Onset Diabetes of the Young
NIDDM	Non-Insulin-Dependent Diabetes
OCT	Optical Coherence Tomography
OGTT	Oral Glucose Tolerance Test
OP	Oscillatory Potentials
PERG	Pattern Electroretinogram
STZ	Streptozotocin
SWCT	Sine-Wave Contrast Test

T2DM	Type 2 Diabetes Mellitus
T1DM	Type 1 Diabetes Mellitus
UKPDS	UK Prospective Diabetes Study
VA	Visual Acuity
VCTS	Vision Contrast Test System
VEP	Visual Evoked Potentials
WHO	World Health Organization

ABSTRACT

BACKGROUND:

Contrast sensitivity is the capacity to detect minor changes in luminance between regions that do not have distinct borders.However, visual acuity evaluates the sharpness of one's eyes at a specific distance. High contrast optotypes, which consist of black text on a white backdrop, are used to evaluate this. The fact that contrast sensitivity, a measure of visual quality, often decreases at an earlier age than visual acuity does is generally acknowledged.

The current diabetes epidemic in India is largely attributable to diabetic retinopathy, a vision-threatening complication of diabetes mellitus. Research suggests that retinal neurodegeneration occurs in the early stages of diabetic retinopathy, despite the fact that the condition is characterised as a microvascular disease.Visual impairments such as reduced contrast sensitivity, changed colour perception, and altered temporal perception can be caused by retinal neurodegeneration. Such deficiencies might manifest prior to alterations in visual acuity and vascular architecture .Detecting early visual functional alterations in lower contrast situations may be beyond the capabilities of the current screening techniques for diabetic retinopathy, which primarily evaluate the morphologic integrity of the retina and retinal circulation.

Therefore, the purpose of this study is to ascertain whether metabolic control and contrast sensitivity are associated in Type 2 diabetics who do not have retinopathy.

METHODS:

From September 2022 to December 2023, a cross-sectional study was carried out on a minimum of 53 patients who met the inclusion criteria at R. L. Jalappa Hospital and

Research in Kolar. The study was approved by the Institutional Ethical Committee of Sri Devaraj Urs Medical College, and the subjects were provided written informed consent.

The period of diabetes and diabetic control were recorded after collecting a brief history of the patient's eyes and overall health, as well as their treatment for the condition. Visual acuity, anterior and posterior segment evaluation, and demographic data were all part of the clinical examination that each patient underwent. A thorough evaluation of the retina was carried out in order to exclude the possibility of diabetic retinopathy.

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RESULTS:

The present study enrolled 53 patients and their mean age was 60.16±7.80 years and ranged from 40 to 70years.The mean fasting blood glucose was 127.28±40.75 mg/dl and the post prandial blood glucose was 174.37±59.52 mg/dl. The three months glycemic control marker HbA1c was assessed and the mean was 7.72±1.85.The mean duration of diabetes was 7.21±4.37 years

The mean contrast sensitivity for right eye was 1.57±0.37 which indicated some visual impairment. Similarly the left eye also showed the same (1.56±0.37). The negative

correlation was observed in the present study which indicated that as the duration of diabetes increased with poor glycemic control, the contrast sensitivity decreased on both right eye (r=0.7097; P<0.0001) and left eye (r=-0.6990; P<0.0001) .

CONCLUSION:

In conclusion, our study highlights the critical interplay between diabetes duration, glycemic control, and visual impairment. As the duration of diabetes increased; contrast sensitivity decreased on both eyes significantly (P<0.0001).Similarly, both eyes contrast sensitivity had a negative correlation with HBA1c levels, which revealed that as the glycemic control progressed to poor control, the contrast sensitivity progressed to visual impairment. The significant correlations found in this study should encourage healthcare providers to focus on comprehensive diabetes management strategies to prevent or delay the onset of visual disabilities in diabetic patients.

Keywords: Contrast sensitivity,Glycemic control,Diabetes mellitus,Diabetic Retinopathy

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF GRAPHS

LIST OF PHOTOS

INTRODUCTION

INTRODUCTION

The long-term effects of diabetes mellitus (DM), a metabolic illness defined by consistently high blood glucose levels, can be devastating to many bodily systems, including the cardiovascular system, eyes, kidneys, nerves, and blood vessels.[1]The International Diabetes Federation (IDF) and the World Health Organization (WHO) both agree that diabetes mellitus (DM) is quickly becoming a major public health issue in the modern era.[2,3]

During the progression of diabetes mellitus, various structures may be impacted, including alterations in blood vessels[4] and neuropatathy[5] which commonly result in diabetic foot, renal disease, or ocular disorders,[6] particularly diabetic retinopathy. That being stated, it is understood that in the retina, even when detected early, microangiopathy can be noted in small retinal capillaries, resulting in increased vascular permeability, ocular haemorrhage, and lipid exudates.[4] Hence, the significance of scrutinizing indicators in preclinical diabetes facilitates the potential for prompt detection.

Reducing diabetic complications and improving the long-term prognosis of type 2 diabetes mellitus(T2DM) can be achieved by the rigorous regulation of blood glucose levels and blood pressure. For instance, the UK Prospective Diabetes Study (UKPDS) demonstrated the critical need of tightly regulating blood sugar levels using anti-diabetic medications to forestall the long-term complications associated with type 2 diabetes. A lower risk of diabetes-related death, slower progression of diabetic retinopathy, and prevention of blindness were all outcomes of rigorous blood pressure control in people with Hypertension and Type 2 diabetes[4]

Examining visual function in the preclinical period of DM involves studying visual abnormalities using non-invasive examinations. This can be done by analysing measurements of visual acuity (VA) or contrast sensitivity function (CSF). VA refers to the capacity to

distinguish between distinct points and recognize shapes. Contrast sensitivity (CS) is the ability to perceive minor differences or distinguish between two closely located points. Contrast sensitivity function (CSF) is a measure of the threshold for detecting contrast, determined by evaluating the ability to discern varying spatial frequencies in four or more circles with bands. In contrast to the usual Snellen test, which only assesses letter size and high contrast (black-on-white), the CS assessment evaluates the ability to identify low-contrast objects of different sizes and analyse the capacity to resolve fine detail. Consequently, the CSF values provide more comprehensive qualitative information about visual function compared to VA. It is frequently seen that individuals may successfully identify the smallest letter on a visual acuity test card, yet still experience visual impairment, which is commonly associated with a decrease in contrast sensitivity.[9] In addition, CSF is influenced by variations in ambient brightness.

In general, increased ambient illumination results in improved CSF. Nevertheless, it is crucial to take into account the constriction of the pupil in brighter lighting situations. This decreases distortions and also reduces the amount of light that reaches the centre of the retina.[10] Therefore, in order to accurately assess a patient's visual function, it is necessary to take into account various lighting and viewing situations.[11] CSF values can be obtained using several techniques and tests, including measurements utilizing variable contrast optotypes such as Pelli-Robson Test, Rabin Test, or Mars Letter Test,[12] as well as sine-wave gratings with varying spatial frequencies like the CSV-1000 test or OPTIC 6500 test.[13,14] The CSF values may vary depending on age and the administration of many medications.[15]

Cognitive decline in diabetics has been the subject of several studies. Multiple investigations on CS in DM patients, some of whom had DR and others who did not, were included in a meta-analysis of psychophysical assessments of DR. In order to detect early

changes in visual function and signs of neurodegeneration in individuals with diabetes, the review found that CS is a better test than VA.[16]

Having said that, a number of research have looked into CS and diabetes control with and without DR.[17-19] Glycosylated hemoglobin (HbA1c), visual acuity, age, length of diabetes, and the existence of diabetic retinopathy (DR) are all variables that have been associated to decreased CS in diabetes.[17, 18] Because of differences in approach, the reports are incoherent. Some studies included people with Type1 diabetes, whereas others included people with Type 2 diabetes, and various methods were used to assess contrast sensitivity. Furthermore, while some research included participants with varying degrees of DR, others did not. Little is known about the prevalence of diabetes in the Indian population or the role of abnormal CS in the development of vascular and metabolic complications.

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AIMS AND OBJECTIVES

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AIM:

To measure the contrast sensitivity in patients with Type 2 Diabetes Mellitus without retinopathy and to correlate it with the duration of diabetes and glycaemic control (HbA1c).

OBJECTIVES:

- To evaluate contrast sensitivity and metabolic control in diabetic patients without retinopathy
- To determine the correlation between contrast sensitivity and metabolic control
- To assess whether the duration of diabetes influences contrast sensitivity in diabetic patients without retinopathy

REVIEW OF LITERATURE

REVIEW OF LITERATURE

AFFERENT VISUAL PATHWAY:

The visual system's light processing begins in the retina. The light that passes through the optical elements of the eye is focused onto the fovea, where the light-sensitive components transform the incoming light rays into electrical signals. The obtained information is conveyed along the visual pathway to the occipital brain, where further processing of the received information takes place. The visual pathway denotes the neuronal linkage connecting the retina to the cortical visual center situated in the occipital lobe. Considering the light-sensitive elements of the retina as sensory receptors, the visual circuit comprises three neurons. The nuclei of the first two neurons are located on the retina, namely on the retinal bipolar and ganglion cells. The axons of the retinal ganglion cells leave the optic bulb as the optic nerve. The optic nerve partially crosses at the chiasm before ending in the corpus geniculatum laterale. At this location, it establishes connections with the primary visual centers. The axonal projections of the third neuron develop connections with the visual center in the occipital lobe, creating visual radiation.[20]

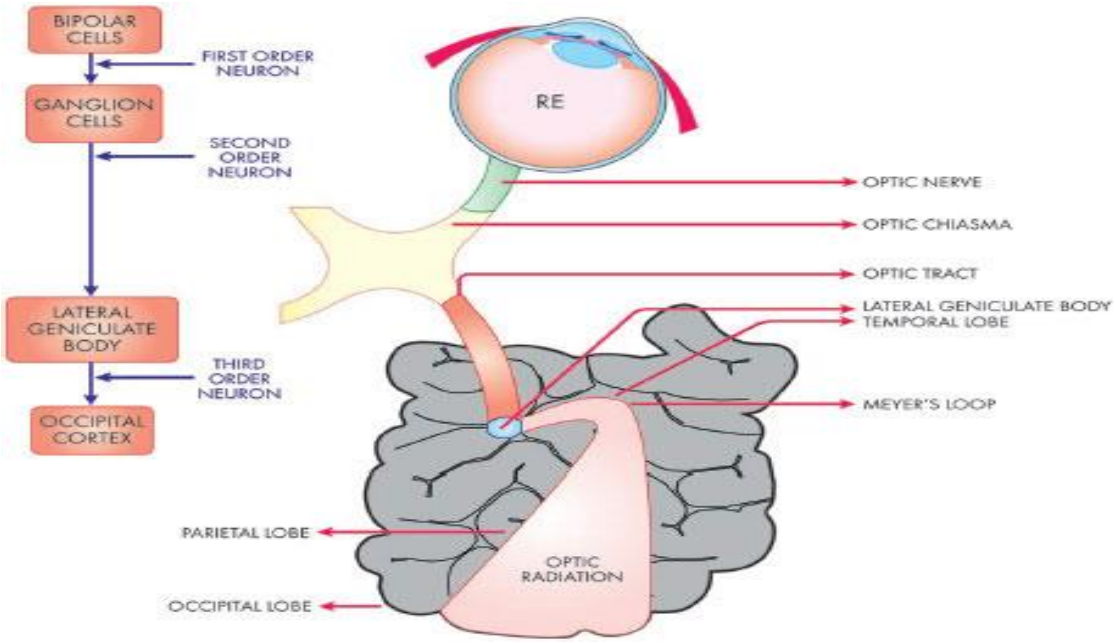


Figure 1: Order of neurons in visual pathway

ANATOMY OF MACULA:

Photoreceptors are derived from the innermost layer of the eyeball and are therefore situated in the deepest layer. At this layer, they make contact with the pigment epithelium, which improves the quality of the retinal image, and with the choriocapillaries, which guarantees that the high energy requirements of these neuroepithelia are fulfilled. The retina has approximately 130 million rod cells and 7 million cone cells. The clinical posterior pole refers to the center region of the posterior retina, which has a diameter of roughly 5 to 6 mm. It is located between the two temporal retinal arteries. The area centralis or macula is the term used to describe it. The clinical macula, also known as the anatomic fovea, refers to the central region that is roughly 1.5 mm in diameter within the area centralis. The anatomic fovea contains a central depression that measures roughly 0.35 mm in diameter. The specific name for this area is the foveola. It is situated in the capillary-free region, which has an average diameter of around 0.4 mm in the majority of individuals. The photoreceptors in this area are exclusively composed of cones. The central point of the pit located within the foveola is referred to as the umbo. Each cone within the macula is associated with its own bipolar and ganglion cell. Each cone in the eye is associated with its own individual fiber in the optic nerve. This is essential for the exceptional amount of detail found in the central retinal fovea. In the outermost area of the retina, known as the peripheral region, there is a continuous connection between many rods and a single bipolar cell. This region is responsible for monochromatic night vision. The presence of several receptors in the eye reduces its ability to distinguish, but at the same time, it increases the total sensitivity of the peripheral vision. In addition to the vertical chain of neurons in the retina, there are also components with horizontal connections that contribute to the processing of the signal transmitted by the retina to the visual pathway.[20]

The axons of ganglion cells arising from the entire macula, including both the nasal and temporal halves, come together at the papilla. The arc surrounds the fovea in both the upper and lower directions, taking up a substantial section of its temporal sector. The optic fibers extend directly from the entire nasal region to the target. Conversely, fibers that come from the outer edges of the non-central part of the retina take a curved route around the region where the optic nerve and the macula meet in order to reach the intended visual destination.[20]

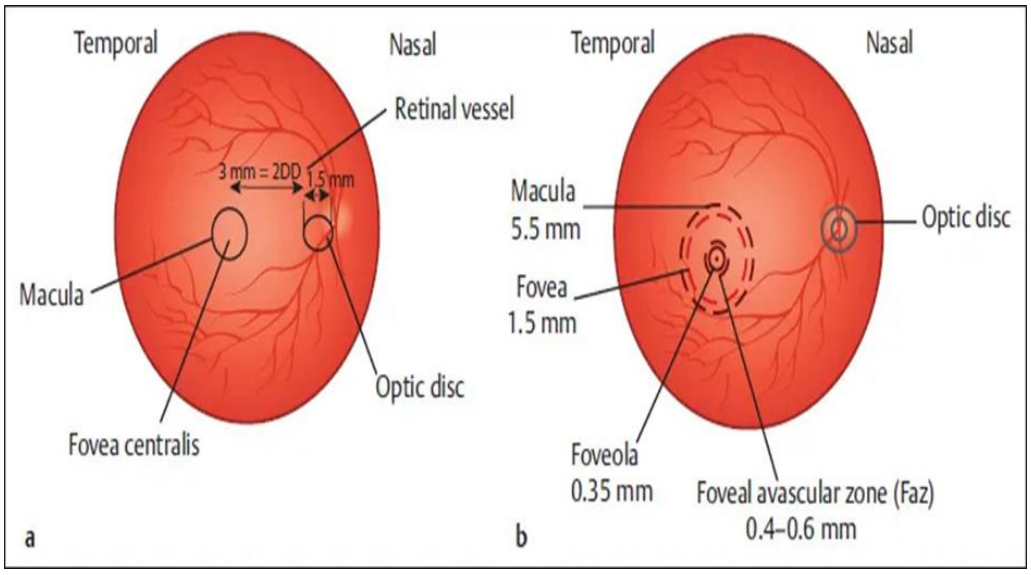


Figure 2: A:Posterior pole of retina Figure 2:B:Macula lutea

LAYERS OF LATERAL GENICULATE BODY:

The Lateral geniculate body consists of three discrete layers, each containing cells with unique physiological characteristics. These cells project to specific layers of the primary visual cortex, creating three separate paths for visual processing. The layers present are the magnocellular (MC), parvocellular (PC), and koniocellular (KC) layers. The thickness of each layer ranges from 4 to 10 cells.[21]

Parvocellular (PC) cells: PC cells exhibit optimal responsiveness to chromatic stimuli with excellent spatial resolution and relatively slow motion.

Magnocellular (MC)cells have a more rapid reaction to alterations in stimulus contrast and possess greater receptiveness to stimuli with high temporal frequency and low spatial frequency.

Koniocellular (KC)cells receive input from many types of retinal ganglion cells, including bistratified Retinal ganglion cells. Certain individuals exhibit binocular reflexes. In addition, they can also demonstrate orientation and direction selectivity.[21]

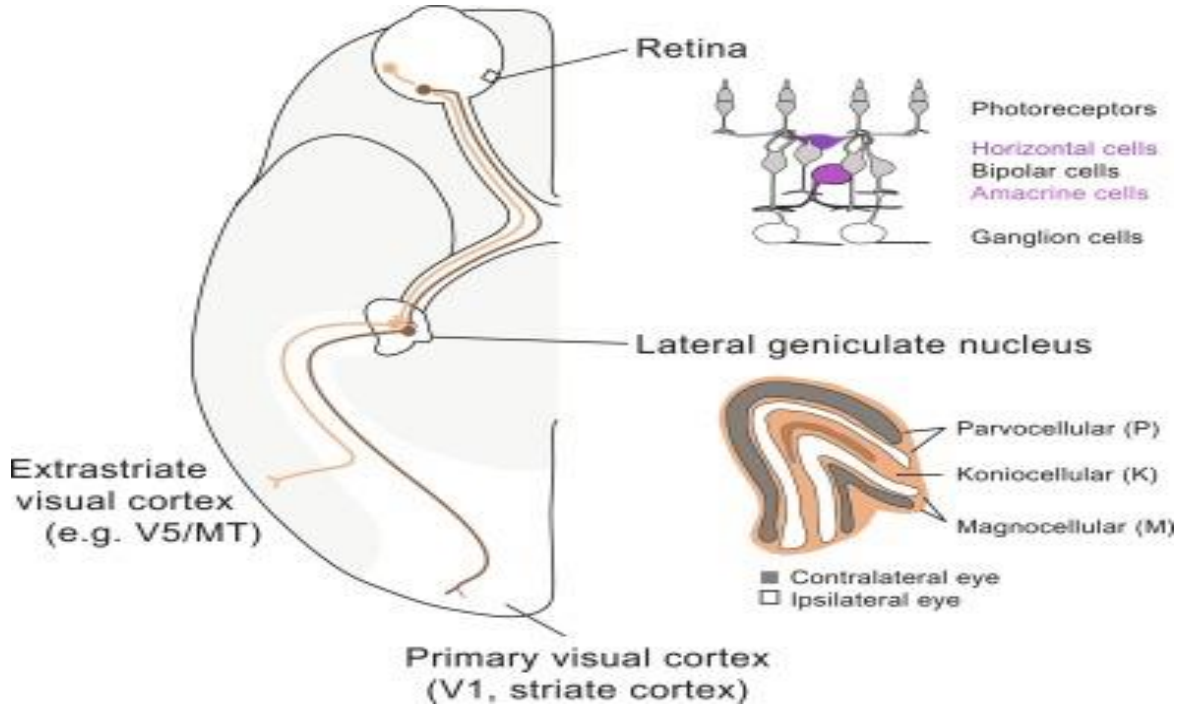


Figure 3: Subcortical visual pathways

DIABETES MELLITUS-CLASSIFICATION AND EPIDEMIOLOGY:

Diabetes mellitus (DM) cause abnormally high blood sugar levels over an extended period of time because of insulin resistance or inadequate insulin production. Hyperglycemia, which if left unchecked, can gradually harm many bodily systems and organs.[22] For the purposes of the American Diabetes Association, there are mainly four ways to classify DM.

Diabetes mellitus type 1, also called T1DM: This type of diabetes, which accounts for 5–10% of cases, is defined by an absence of insulin.

In 90% of cases, the type of diabetes is type 2 diabetes mellitus (T2DM). Opposition to insulin's peripheral actions is the main commonality.

Dementia during pregnancy: During the second or third trimester of pregnancy, a woman is usually diagnosed with type 2 gestational diabetes mellitus. Both genetic predisposition and the hormonal shifts experienced by pregnant women might set off this disorder.

Various forms of diabetes A number of subtypes of diabetes mellitus fall under this umbrella. These include monogenic diabetes, Maturity Onset Diabetes of the Young(MODY), exocrine pancreas disorders (like cystic fibrosis), and diabetes caused by immunosuppressants and glucocorticoids. Eight percent of the global population has diabetes mellitus (DM), which translates to over 425 million people. According to projections, this figure will soar to more than 629 million by 2045.[22]According to estimates, type 2 diabetes is prevalent in

high-income nations at 87-91% and type 1 diabetes between 2-7% of the overall population. In Europe, 6.8% of the population has diabetes when adjusted for age and sex.[23] Complications from diabetes contribute significantly to premature death. According to the World Health Organization, 1.5 million people died as a result of diabetes in 2012, with another 2.2 million dying from complications such as heart disease[24.]

Up to half of all persons with diabetes go undetected since the disease can occasionally exist asymptomatically for long periods of time. This is the main epidemiological difficulty in type 2 diabetes. As a result, there are now precise guidelines for the diagnosis and screening of the illness.

A chronic, crippling, and financially burdensome disease with serious consequences, diabetes poses serious risks to families, nations, and the world at large, according to a 2006 official description by the United Nations General Assembly.[25] Significant financial losses are caused by diabetes and its complications, including lost income and productivity at work and high medical bills.[23,24]

ETIOPATHOGENESIS AND RISK FACTORS OF DIABETES MELLITUS:

An autoimmune reaction in which the body assaults the insulin-producing beta cells in the pancreas causes T1DM, also known as insulin-dependent diabetes. Because of this, the hormone is rendered ineffective, and the patient must undergo daily external delivery in order to maintain normal blood glucose levels.

This study focuses on type 2 diabetes, which is marked by a slow but steady decrease in insulin release by beta pancreatic cells. When insulin resistance sets established, blood sugar levels drop, but the pancreas responds by pumping out more insulin. However, over time, this compensatory mechanism fails, resulting in a progressive loss of insulin secretion. In some cases, the pancreas may not produce enough insulin to regulate blood glucose levels, resulting in elevated levels even when there are no noticeable symptoms. In such instances, there exists a concealed clinical presentation that could remain undetected for an extended period of time, and difficulties may arise upon the identification of the condition.[26]

Diabetes mellitus type 2 (T2DM) has a complex origin, involving multiple factors such as a strong genetic predisposition, a lack of physical activity, and being overweight or obese, which might potentially initiate the condition. This type of DM was commonly identified in individuals over the age of 30. The rise in children and youth obesity is leading to its occurrence at progressively earlier ages. The subsequent classical risk factors[27] have been identified for the development of T2DM:

- Previously diagnosed with pre-diabetes.
- Obesity is caused by dietary choices, insufficient physical exercise, and several variables such as individual and genetic susceptibility.
- There is a familial history of type 2 diabetes mellitus in the immediate family.
- Dyslipidemia refers to an abnormal level of lipids (such as cholesterol and triglycerides) in the blood.
- Hypertension.
- Pertaining to ethnicities with a higher susceptibility to danger (African American, Hispanic American, Native American, Asian American, and Pacific Islander).
- Previous occurrence of gestational diabetes or giving birth to a baby weighing more than 4 kg.

LONG TERM COMPLICATIONS OF DIABETES:

Damage to several organs and systems occurs as a result of persistently high blood glucose levels in people with diabetes mellitus (DM), leading to complications such as: Diabetic retinopathy (DR), diabetic nephropathy, and diabetic neuropathy are microvascular diseases that cause problems with small blood arteries.

There has been a huge surge in the prevalence of type 2 diabetes mellitus (T2DM) in the last 20 years, putting a lot of people at risk for retinal alterations that could cause vision loss. One of the most difficult conditions to treat is diabetic retinopathy, which affects around 40% of diabetics and is the leading cause of vision loss in individuals of working age. With the progression of the condition, the likelihood of getting diabetic retinopathy and eventually losing vision rises.[28] Research shows that diabetes-related vision impairment and blindness can be mitigated with prompt diagnosis and treatment.[29] It is rather surprising that typical visual acuity tests performed during routine visual screenings in clinics do not reveal any symptoms or indications of impaired vision in around one-third of patients with diabetic retinopathy, including some with late stages.[30] This means these patients might not get the follow-up care they need because their risk factors were not recognized. Hence, better, more targeted ways to identify diabetic eye function deficits at their earlier stages are required.

METABOLIC REGULATION OF DIABETES:

The potential influence of successful metabolic regulation on the postponement or avoidance of chronic microvascular complications in individuals with diabetes has not yet been confirmed in human subjects, despite the substantial amount of evidence that supports this viewpoint.[31,32] Presently, there is an insufficiency of precise techniques to quantify the extent of glucose regulation over an extended duration. Nevertheless, the recent progress in the glycosylated haemoglobin (HbA1c) assay,[33] which offers a thorough assessment of blood glucose levels during the previous weeks to months, could partially resolve this concern. An additional obstacle that has emerged relates to the lack of dependable and accurate techniques for assessing diabetes complications with quantitative precision in the early stages. However, there is progress being made in this area.[34]

Additionally, it has been extremely difficult to maintain a prolonged state of normal blood sugar levels, which is critical for preventing the development of diabetic problems,

using traditional insulin injection therapy. There are now many ways being actively pursued to improve blood glucose regulation. The effective enhancement of metabolic regulation in multiple patients can be ascribed to a collaborative initiative involving physicians, nutritionists, and nurses who have undergone training in diabetes treatment. This cooperative endeavor frequently involves the use of a home glucose monitoring equipment. The process of home glucose monitoring is placing a small amount of blood, obtained by pricking the finger, onto a glucose oxidase reagent strip.

VISUAL DYSFUNCTION IN EARLY DIABETES:

Impaired contrast sensitivity and visual acuity, or the capacity to distinguish between foreground and background objects, as well as the ability to discern fine details, have been associated with early diabetes. In humans, these are typically assessed by verbal feedback; however, in animal models, the challenges are more substantial. One way rats can gauge their contrast sensitivity and visual acuity is by measuring their optokinetic response. To track the movement of bars, animals in this experiment turn their heads in a specific direction.[35] Animals' sensitivity to very minor changes in contrast and bar size is tested by researchers by varying these parameters. The ability to see fine details and respond to changes in contrast is tested in diabetic animal models.

To investigate how early-stage diabetes impacts eyesight, researchers have employed a number of animal models.[36] Of these versions, the STZ model is by far the most popular. The existing model relies on injecting STZ into the pancreatic beta cells to increase its absorption by glucose transporters. The model is suitable for studying type 1 diabetes in different species since it begins with beta cell death and then leads to hyperglycemia.[37] You can control the timing in this model precisely because animals given streptozotocin (STZ) usually show signs of hyperglycemia soon after injection. Insulin buildup in the endoplasmic

reticulum of pancreatic beta cells is modeled in the widely used Ins2Akita mouse line, which has a mutation in the insulin2 gene. As a result, beta cells eventually die off and blood sugar levels gradually rise, starting about 4 weeks of age.[38] Subjects lacking diabetic retinopathy but with type I or type II diabetes showed diminished contrast sensitivity. Glycemic control, as measured by elevated levels of the glucose-binding protein HbA1c, an indicator of blood sugar levels during the past few days, is directly correlated with reduced contrast sensitivity.[41]

When light sensitivity was intermediate (mediated by rods and cones), contrast discrimination was good. When light sensitivity was low, contrast discrimination was poor (mediated by cones).[40] While noticeable shifts in visual clarity sometimes occur, they are less often reported. Alterations in visual evoked potentials (VEPs), which assess activity in the main visual cortex, become apparent within six months following a diabetes diagnosis.[42,43] Less contrast sensitivity and visual acuity can be observed as early as four weeks following the introduction of diabetes in rodent models.[44-46] The results indicate that the inner retina, particularly the layers responsible for contrast sensitivity—the inner nuclear layer, the inner plexiform layer, and the ganglion cell layer—is impacted by early diabetes. Human diabetic patients' visual changes are similar to those seen in animal models of the disease.Impact of diabetes on retinal function tests for color vision, contrast sensitivity, and electroretinogram (ERG) can detect early abnormalities in diabetic retinopathy (DR) before a diagnosis is made There are telltale changes in electroretinograph (ERG) recordings in diabetes patients and animal models, including reduced voltage amplitude and delayed onset of oscillatory potentials. The presence of microvascular change is not necessary for the observation of these alterations.[47,48] A recent study included 400 patients with type 2 diabetes (DM2), and it found that 58% of those patients had abnormal ERGs without the presence of vascular lesions that would indicate diabetic retinopathy (DR).[49] In addition, electrical changes in the

retina of a diabetic rat model are detected prior to the development of retinal vascular lesions and cognitive deficits in this disease using multifocal electroretinography (ERG).[50,51] There appears to have been neurodegeneration before changes in the retina and the systemic blood vessels, according to these results. The use of visual evoked potentials in electrophysiological studies has also shown early changes in people who do not have diabetic retinopathy (DR).[52,53] Among newly diagnosed type 1 and type 2 diabetic patients with apparently normal retinas, Lee et al. recently found that the P100 wave delay lengthened.[52] Unlike patients with type 1 diabetes (T1DM), patients with type 2 diabetes mellitus (T2DM) exhibited unusually elevated levels of glycosylated hemoglobin (HbA1c), which could explain the longer latency. In addition, this electrophysiological study has shown changes in the retinal time delay in both children born to fat moms and those with gestational diabetes.[54] Prior to the development of clinical or established DR, which may be detected using fundoscopy, other visual function evaluations in DM patients, such as colour perception and contrast sensitivity, have revealed anomalies. To interpret visual data, the brain uses a network of conduction channels that function at various frequencies. However, the mechanisms that are most impacted by DM remain unknown. This is why it's crucial to assess their contrast sensitivity at different spatial frequencies. No discernible reduction of contrast sensitivity has been shown in prior research including type 2 diabetics who did not have DR.[57,58] On the other hand, separate studies in T2DM patients without retinopathy have shown alterations impacting contrast sensitivity across all frequencies.[59] Contrast sensitivity seems to decrease in the context of established DR,[57] and this decline may occur independently of visual acuity reduction.[55]Nevertheless, the exact timing of when this decrease in contrast sensitivity starts is still up for debate. According to others, parvocellular dysfunction—which affects 80% of the retinal ganglion cells—is indicated by a specific decrease of contrast sensitivity for higher frequencies.[60] Publications of correlation research

are also available. Evidence from studies indicates that contrast sensitivity is worse in patients whose disease has progressed for more than ten years or who have poor metabolic control.[57-59] Researchers have shown that people with diabetes may experience abnormalities in their color vision, particularly along the blue-yellow axis, as well as DR.[61] The likelihood of developing diabetes and changes in retinal blood vessel function due to high blood sugar have been linked to this change in color vision, which has also been seen in diabetic patients who do not have DR.[62.63] Since the cones are primarily responsible for color vision, these results are consistent with previous research that has shown a reduction in the retina's photoreceptor layer even when microvascular changes are not visible.[64.65] It appears that dyschromatopsia is a symptom that these people experience before any neurological problems become serious. Diabetics have also been the subjects of perimetry studies. Typical DR changes may not appear in patients with T1DM until after they have already had visual field abnormalities.[66,67] Similarly, it has been proposed that individuals with type 2 diabetes may experience visual field involvement independently of DR, and that this involvement becomes increasingly noticeable as the abnormalities in the retinal vasculature progress.[68] Although these alterations are associated with retinal vasculopathy, several investigations have either failed to identify them in T2DM patients without DR or have failed to find an association between them and the condition.[69-71] Keep in mind that these studies used a variety of perimetry equipment and enrolled individuals having diabetes with and without retinopathy . Maybe this is why we got different results. Confounding variables in optical coherence tomography (OCT) include, but are not limited to, axial length, gender, age, and cardiovascular risk factors (such as smoking, hyperlipidemia, obesity, etc.) There is still no clear answer as to whether vascular or neuropathic mechanisms produce DR in visual function investigations of DM patients. It is possible that early-stage neurodegeneration is supported by the alterations seen in visual function tests (ERG, contrast sensitivity, color

vision, and visual field scores) in diabetic individuals without DR or with minimal changes.[72,73] Risk factors for diabetic neuropathy include dyslipidemia, insufficient metabolic control, and oligoalbuminuria, all of which contribute to the condition's macrovascular and microvascular complications. Animal models and human biopsies have shown that endoneural hypoxia is a hallmark of diabetic neuropathy, which is defined by poor microvascularization of nerves. Because of the shared pathophysiology between subclinical retinal perfusion dysfunction and extraocular consequences of diabetes, it is reasonable to assume that the two disorders may coexist. Limitations in peripheral vision, contrast, color perception, clarity of vision, and oxygen delivery to the retinal nerve structures may occur in the early stages of vascular injury.

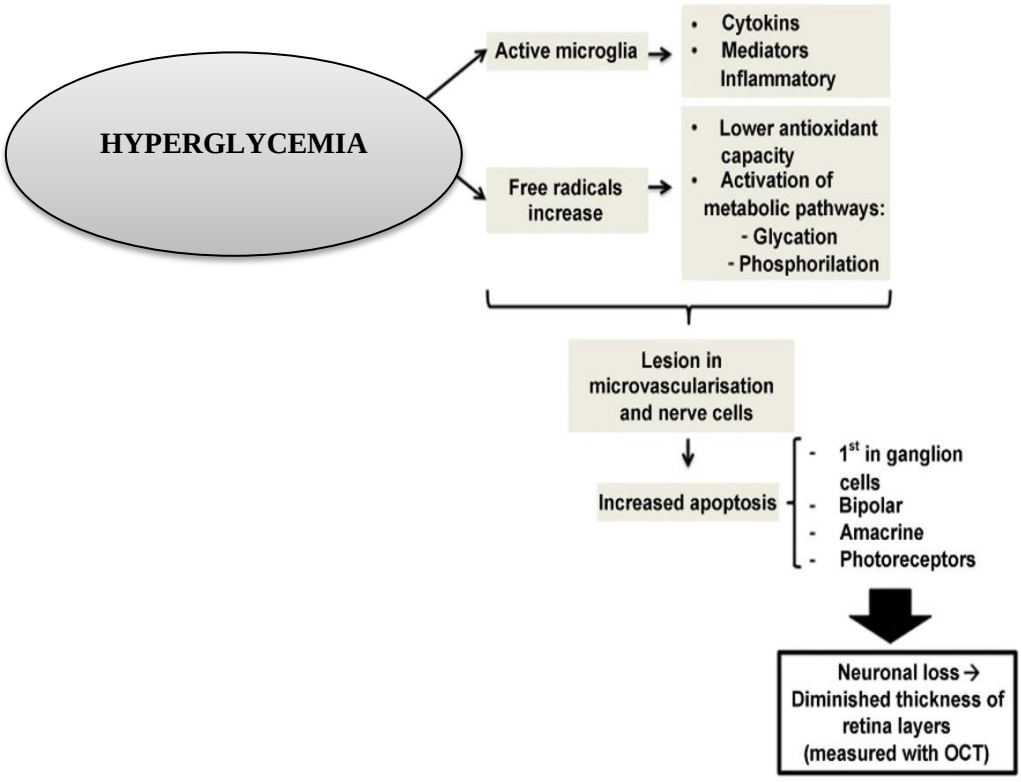


Figure 4: Pathophysiology of neurodegenerative retinal damage in patients with type 2 diabetes mellitus

FUNCTIONAL TESTS FOR DIABETICS IN OPHTHALOMOLOGY:

Examining the fundus is a well-accepted clinical method for identifying diabetic retinopathy in people with diabetes. However, before the pathological changes by diabetes appear in the retina, the patient's visual ability may decrease. Diabetic individuals who do not develop diabetic retinopathy experience a decrease in visual acuity, contrast sensitivity, and retinal sensitivity in the visual field due to damage to the light-sensitive components. In addition, there are changes in color perception and an overall reduction in the retina's sensitivity to light-induced irritation.

EFFECT OF DIABETES ON COLOR PERCEPTION:

The severity of color vision impairment is directly related to the level of damage to the retina. In cases of advanced diabetic retinopathy, color vision is mainly affected in the blue range of the electromagnetic spectrum. Photocoagulation laser therapy has a potential danger to the patient's ability to see colors, since it can lead to additional damage to the light-sensitive cells in the eye known as photoreceptors.[75,76]

COLOR VISION EXAMINATION:

The application of color perception examination approach provides benefits not only in assessing and measuring inherent color vision deficiencies, but also as a diagnostic tool for evaluating acquired disorders caused by toxic or degenerative damage to the retina and optic nerve. Pseudoisochromatic tables are frequently used in clinical settings as a method to assess color sensitivity. The examination consists of a series of dots that display different shades and levels of brightness. The colored dots in the test symbolize distinct numerical values, alphabetical characters, or geometric coordinates. An individual with color vision deficiency will struggle to accurately recognize the displayed character because of the choice of colors used for both the character and its background.

Table tests are mostly used to evaluate congenital problems associated with color perception. The Farnsworth-Munsell 100-hue test is employed to acquire a more accurate evaluation of color sensitivity. This test consists of 85 color targets that maintain a uniform level of brightness and saturation. The color transitions smoothly from red to blue. The tested patient arranges the mixed targets in the proper sequence, with the goal of minimizing the color contrast between adjacent objects. The evaluation of success is carried out utilizing a circular methodology, in which the error score for each target can be obtained. The Panel D-15 test is an alternate method that consists of 15 targets and is available in both saturated and desaturated versions. Furthermore, this testing methodology can be used to diagnose acquired color vision impairments.[77]

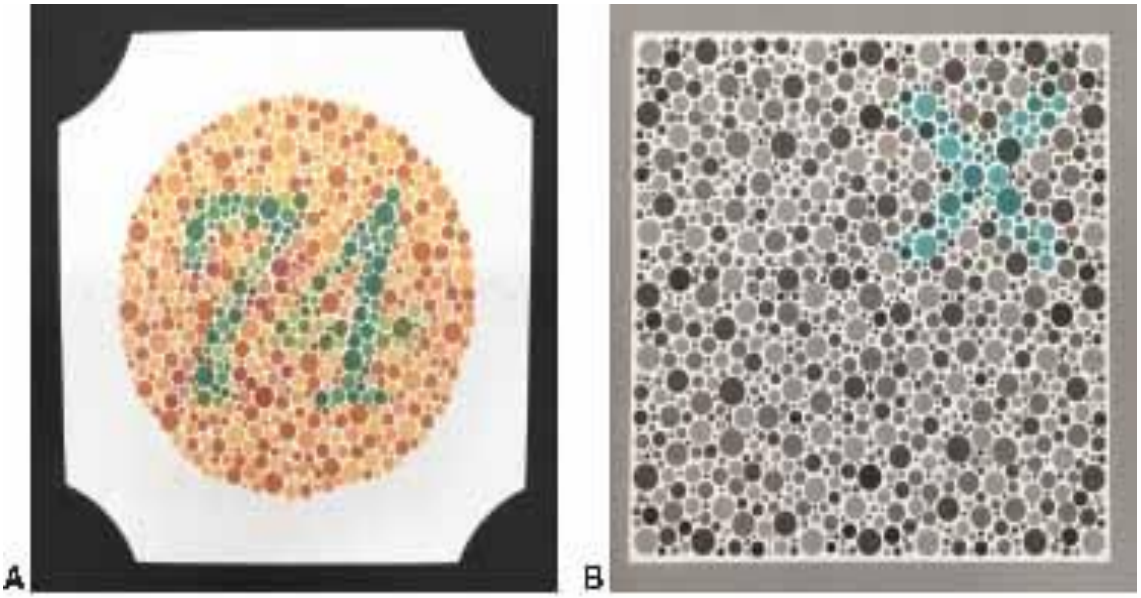


Figure 5: Ishihara chart

THE EFFECT OF DIABETES ON ELECTRORETINOGRAM:

Electroretinography (ERG) is primarily important in diabetic patients because it can detect the likelihood of developing diabetic retinopathy before any changes in the eye's background structure occur. The electrooculogram (EOG) of a diabetic patient shows notable differences compared to the ERG of a healthy population, particularly in the oscillatory potentials.[78]

ELECTRORETINOGRAM(ERG):

An electroretinogram is a device that records the electrical potentials produced by the retina in response to light stimulation. The electroretinogram (ERG) is used to diagnose retinal disorders by employing two different types of stimulation. There are two types of measurement methods used in this context. The first one is called a digital electroretinograph (DERG), which uses a single flash for stimulation. The second method is known as a structured electroretinogram(ERG), which involves repeated flashes. The examination is performed using monocular vision, incorporating both modalities. The electroretinogram measures the direct current (DC) potential between the cornea and the retina by positioning examination electrodes on the cornea and the outer canthus.

The generation of this potential is a result of the regulation of ion movement from the choroid to the photoreceptors. The resting potential established between the retina and the choroid remains unchanged by bulb movement, but experiences modifications in response to changes in the level of adaptation. The most frequently detected components of the ERG signal are waves a, b, c, and d. Size,form and latency of ERG waves depend on the intensity, duration, and color of the light stimulus, as well as the level of retinal adaptation. This phenomena exhibits a range of values that extend across several hundred microvolts. When the stimulus intensity is decreased, the subsequent waveform only shows a positive wave ‘b’. However, with greater intensities, a negative wave ‘a’ follows. During this diagnostic process, the pigment epithelium produces electrical potentials. Photoreceptors correspond to wave ‘a’, but bipolar cells correspond to wave ‘b’, while ganglion cells do not. Abnormality is a term used to describe a disorder that is marked by abnormal changes in the retina, such as degeneration, memory loss, or inadequate blood supply. These changes specifically affect the first and second tiers of retinal neurons.

The electroretinogram (ERG) is formed in response to a patterned checkerboard stimulus, specifically known as "pattern-reversal" (pERG). This physiological event is defined by the existence of a small negative deflection near 35ms,positive deflection near 50ms (P50) and a negative deflection near 95ms(N95). The ganglion cells, which are the third retinal neuron, generate these waves. Anomalies in this waveform indicate malfunction in ganglion cells. Individuals who have high intraocular pressure, optic nerve atrophy, or glaucoma experience reduced amplitudes of the pERG wave. The indicated lesions mainly relate to eye disorders, including demyelinating, repressive, or traumatic injuries.[79-81]

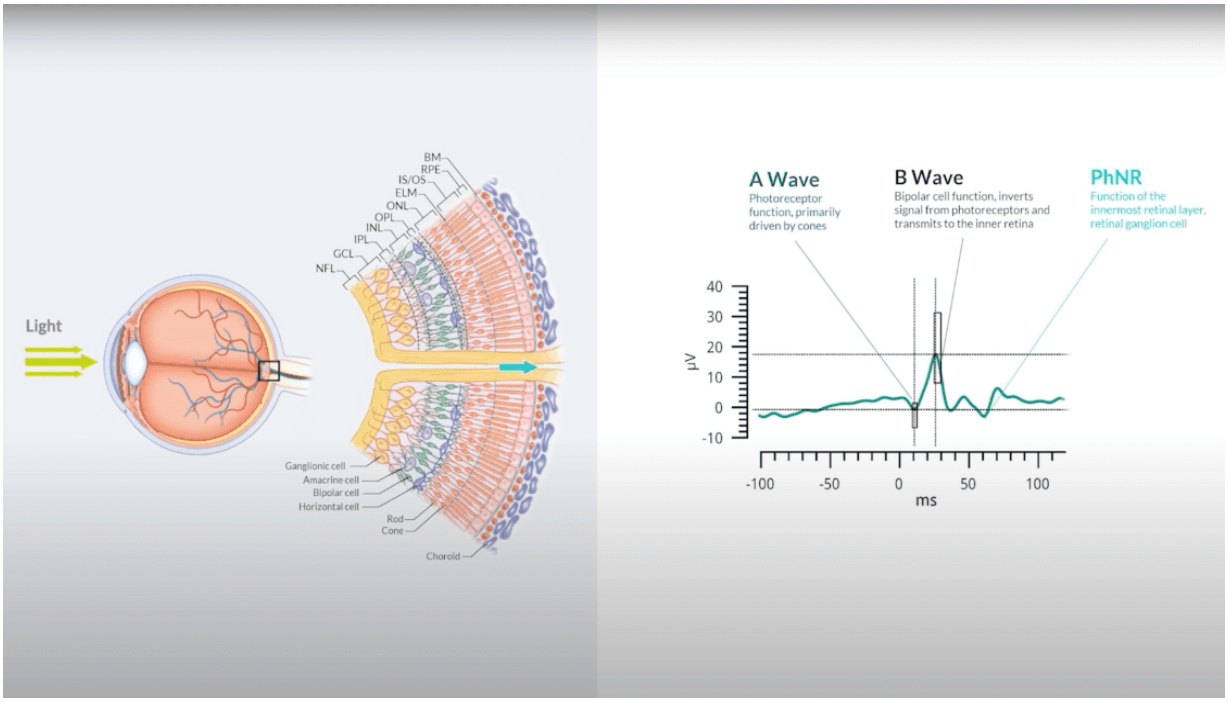


Figure 6: Electroretinography (ERG)

Several studies have evaluated flash ERG, pERG, or both in individuals with diabetes. Diabetes patients can have electroretinogram (ERG) abnormalities long before retinopathy develops, according to strong evidence. When comparing diabetic persons without retinopathy to nondiabetic controls, most studies that focus on flash ERG have found anomalies, most notably a reduction in OP amplitude. [82- 88] Research on pERG is still in its

early stages, and results are inconclusive. Some groups have seen abnormalities in diabetics, whereas others haven't noticed a thing. The results of the three cohorts that tested both approaches in the same patient populations were mixed.[89-91] Despite changes in oscillatory potentials (OP) amplitudes, Coupland[91] and Arden et al.[89] showed that the pattern electroretinogram (pERG) stayed the same in diabetic patients with retinopathy. Because Wanger and Persson[90] did not see any changes in PERG or OP, they concluded that ERG was not very useful for assessing diabetic early-stage retinal damage. When compared to other electrophysiological tests, ERG has shown conflicting results in a number of studies. [92,93] A study analyzing flash ERG in a group of individuals recently diagnosed with IDDM was carried out by Uccioli et al.[92.] Visual evoked potentials (VEPs), and more especially the P100 component, were compared to the findings. There were no statistically significant differences in flash ERG between the diabetic and non-diabetic groups. P100 delay, however, was significantly higher in the diabetes group. Papakostopoulos et al.[93,] on the other hand, discovered significant changes to the "b"wave's amplitude. There was no statistically significant relationship between the two variables, even though this group also had changes in P100 delay. Across all spatial frequencies and contrast levels, Martinelli et al.[94] shown that persons with IDDM had inferior average pERG results. Additionally, they looked at the P100 latencies and found that people with reduced pERG amplitude had significantly longer P100 latencies than people with normal pERG. The results imply that changes in pERG amplitude are among the first electrophysiological abnormalities that can be detected in the optic pathway.

ASSOCIATION BETWEEN DIABETES TYPES AND ERG:

Individuals diagnosed with IDDM have been the sole subjects of most ERG studies. People with NIDDM (non-insulin-dependent diabetes) have minimally studied ERG alterations. Bresnick and Palta[95] looked at a small sample size of 7 patients with NIDDM and retinopathy

in their investigation. Nevertheless, no study or observations were provided indicating possible differences between this subgroup and the larger group of IDDM participants, which included 78 patients. Although no statistical analysis was provided, Boschi et al.[96] found that pERG amplitudes were lower in insulin-dependent diabetes mellitus (IDDM) patients compared to non-insulin-dependent diabetes mellitus (NIDDM) patients. Vingolo et al.[97] looked at diabetic pregnant women and found that they had different flash ERG responses. The decline in the b2/b1 ratio was more pronounced in the group with NIDDM or gestational diabetes mellitus compared to the IDDM patients or nondiabetic controls.

THE IMPACT OF DIABETES DURATION AND GLYCEMIC CONTROL ON ERG:

We looked at five studies worth of data to see whether we could find any correlation between the length of time people have had diabetes and changes in electroretinograms (ERGs).The results have been mixed, with just two groups showing a clear trend of decreasing ERG amplitude with longer diabetes duration.[98-100]

Greco et al.[100] examined the relationship between pERG anomalies and long-term glycemic management in children with diabetes, as measured by glycated haemoglobin (HbA1c). They did not find any statistically significant link. No other study has discovered an association between HbA1c and ERG abnormalities; however, this link may not have been investigated prior to the Diabetes Control and problems Trial (DCCT), which demonstrated the significance of rigorous glycemic control in preventing problems associated with diabetes.[101]

INFLUENCE OF AGE ON ERG:

A common misconception is that prepubescent diabetic children are less prone to develop microvascular complications like retinopathy.[102] By analyzing ERG, researchers have looked for signs of possible electrical changes in the retina that could happen before visible retinopathy develops.[103] Adolescents (12–20 years old) with IDDM mirror the changes in 'b' wave amplitude seen in adults with the disease. However, the changes were only observed in the left eye, even though blue flash ERG tests were conducted on both eyes.[103]Twenty children with IDDM who were not yet in their teens and had normal results on fluoroscein angiography were reviewed for macular function using focal electroretinography (FERG). The findings demonstrated that the 2F or 2P component's ERG amplitude decreased significantly. According to medical experts, these anomalies are linked to the degeneration of macular neurons. Nearly half (45%) of the diabetic patients exhibited some abnormality in neuroretinal function. Psychophysical abnormalities manifest at the same time as electrical changes linked to diabetes and vision. The majority of these issues centre on the fovea, the area of the eye that provides the sharpest centre vision. Colour vision and contrast sensitivity testing are both simple and painless. Degenerative diseases affecting the retina, the primary organ responsible for macular function, can impair color recognition. [104]

It was in 1953 that the first reports of diabetic eye problems were made.[105] It is unclear what causes visual pathway errors like this one; instead of microvascular disease, it may be associated with metabolic abnormalities in the retina.[106] The findings of colour vision tests, which come in several forms, can be impacted by lens opacities or colour blindness.[107] The Farnsworth-Munsell 100-Hue Test is a popular one.[108] In a study of young individuals with

IDDM, this test demonstrated superior sensitivity and specificity compared to flash and pERG tests in diagnosing visual pathway impairment.[109]

DIABETES EFFECT ON COLOR VISION:

Several experimental investigations have been conducted over the past 25 years to formally assess the association between diabetes and changes in color vision. These research followed early reports of these effects in people with diabetes.[110 to 114] Excluding one study, all the results show that those with diabetes who do not have retinopathy have significantly worse color vision (as measured mostly by the 100-Hue Test) compared to those who do not have diabetes. Spectrum deficiencies in yellow-blue discrimination, specifically known as tritanopia, have been documented in persons with diabetes. The prevalence of tritanopia varies; 30% of the population has mild to moderate cases, while 70%[104]and 80%[107] have severe cases.

Tritanopia is a very rare congenital defect. There may be a connection between neuronal hypoxia and other clinical circumstances, such as diabetes, where it has been seen. Opacity and other lens alterations can cause red-green spectral losses, but tritanopia is distinct from that. This points to retinal abnormalities, not lens issues, as the source of the reported yellow-blue color deficiencies.[104] The significance of this anomaly's occurrence in interpreting visual strips for blood glucose levels is substantial.Patients with diabetes are more likely to incorrectly interpret blood glucose strips than healthy controls, according to research by Lombrail et al. [111]. Therefore, the authors recommend that diabetes patients who self-monitor their blood glucose levels should be more frequently administered tests that measure color discrimination.

ASSOCIATION BETWEEN COLOR VISION AND HYPOGLYCEMIA:

Lakowski et al.[104] were the first to record changes in color as a result of hypoglycemia in their investigation. One of the research subjects had an episode of hypoglycemia while doing the study, which led to this discovery. However, a formal evaluation of color vision revealed a decrease in performance, even though the subject was unaware of any visual loss during the episode. A retest showed that the drop had been reversed once normal blood sugar levels were restored. Initially, color perception under low blood sugar levels was empirically evaluated by Harrad et al. [117.] During severe hypoglycemia, the 100-Hue Test showed that both diabetics and non-diabetics performed worse. Keep in mind that the severity of the hypoglycemia that could occur as a result of intravenous insulin injections varied widely (average venous blood glucose 1.9 60.4 mmol/liter). Abnormalities in cognitive function can influence how well a person does on the 100-Hue Test, which could explain any abnormalities in color vision. The time, focus, and ability to make decisions needed to complete this test are substantial.

CONTRAST SENSTIVITY:

The edge-to-edge shift from light to dark that characterizes an object or pattern in a picture is a physical dimension known as spatial contrast. A location's contrast can be described as the ratio of its difference in luminance to the absolute value of its lowest or highest luminance value. As far as visual science is concerned, there are two standard ways to describe contrast. The Michelson contrast, often applied to periodic patterns like as sine-wave gratings, is the maximum brightness minus the luminance of the least dimmest area, divided by their total.[118,119] Contrast is traditionally defined as the difference between the background's luminance and the letter's luminance, divided by the background's luminance, and used to nonperiodic patterns like letters on charts with spatially protracted white backgrounds.[120]

From zero to one hundred percent, you can get any contrast measure you want. A contrast of 0 indicates the absence of any pattern or border between adjacent sections. For any value above zero, an edge exists; however, the ability of the detector to analyze images determines whether or not the edge is visible. A target may be seen at a certain contrast level, which is called the contrast threshold. Find out how much contrast is required to make visual decisions with activities including detecting, discriminating, recognizing, and identifying. As a general rule, the target detection threshold is lower than the contrast threshold; nonetheless, there are cases where the two thresholds are somewhat close. One way that contrast threshold is expressed in clinical research and patient care is as contrast sensitivity, which is the inverse of threshold. There is a strong correlation between low thresholds and low sensitivity, and vice versa. In the field of visual sensory science, the sensitivity and contrast threshold are measured using logarithmic scales.[121] As a result, the logarithm of the contrast threshold, sensitivity, and contrast at a level of 0.01 (1% of the total).[122] Contrast sensitivity in space and visual acuity are sometimes mistaken for one another. In a highly contrasted environment (at least 85%), with varying sized targets shown at the same contrast, acuity is measured.[120] To determine the lowest contrast threshold required to see an object, contrast sensitivity testing involves varying the contrast.

MEASURING CONTRAST SENSIVITY:

Using computer-generated visual images as test targets and a software-controlled threshold measuring process, it was one of the first types of contrast sensitivity testing. Clinical practice and research were the initial settings for its utilisation. The subjects of the experiment were distinguished by their vertically aligned sine-wave gratings, which exhibited a sinusoidal brightness. When staring through bar gratings, the test subject noticed an ocular angle ranging from half a degree to sixteen cycles per degree (c/d). Each test

grating's contrast sensitivity was shown as a logarithmic plot. The contrast-sensitivity function (CSF) plot showed that for normally sighted observers, sensitivity peaked at mid-spatial frequencies (3-6 c/d), with a steep decline at higher and lower frequencies, respectively. In photopic conditions, a normally-sighted young to middle-aged adult's contrast-sensitivity function is 1.

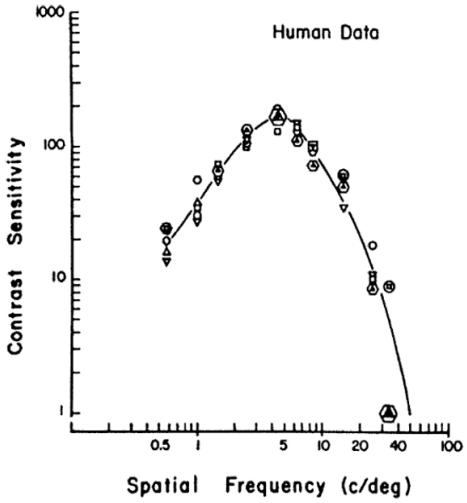


Figure 7: Example of the spatial contrast-sensitivity function under photopic conditions for normally sighted human observers in the young to middle adult age range

Acuity is defined as the highest spatial frequency perceptible at 100% contrast in CSF. So, sine-wave gratings are good candidates for testing because of two traditions. When testing image-processing hardware with bar gratings of varying spatial frequencies, the modulation transfer function is utilised to describe the hardware, especially optical systems.[122,123] According to studies conducted since the 1960s, the CSF is derived from the arrangement of many neuronal spatial filters inside the human visual system, each of which possesses unique spatial-tuning properties.[123-127] When studies showed that ocular and neurologic conditions could perturb the CSF shape,[128-130] investigators and clinicians were

excited about its clinical relevance. The processes of the underlying illness or ailment may be revealed in this way.

With this theoretical and practical foundation, CSF measurement to characterise spatial contrast sensitivity quickly gained popularity as a method to evaluate normal or disordered pattern vision abilities in the human visual system. It is not feasible to conduct clinical patient evaluations and research using computer-based systems for a number of reasons. There is a lack of normative data, the equipment are costly, the procedures and image calibration are difficult to standardise across clinics and sites, and testing regimens typically last more than 5 minutes. After realising that computer-controlled tests weren't going to cut it in clinical settings and patient-centered research, researchers started working on chart-based contrast sensitivity tests in the 1980s. As far as clinic-friendly printed contrast-sensitivity tests go, the Arden plates were pioneers. We will discuss these before delving into the two main tests that are currently used to determine contrast sensitivity in charts.[128] The seven plates that make up the Arden set each have a different contrasting pattern, but they all have one sine-wave grating printed on them. Grating diameters vary between 0.4 and 6.4 c/d at 57 cm. In order for the patient to view the bars, the examiner meticulously lifts each plate and moves them from low to high contrast. At the plate uncovered threshold, a score between one and twenty is assigned at random.

A total contrast-sensitivity score can be obtained by adding the scores on each plate. Even in those who had the right reading glasses, several studies conducted in the 1970s and 1980s used Arden plates to show that different eye disorders had a negative effect on contrast sensitivity.[135–140] It was shown that contrast sensitivity could screen for eye illness in early computer-generated grating experiments.[136-138] There are issues with the current test techniques for Arden plates. Threshold can be affected by environmental factors, patient

decision-making criteria, the rate at which the plates are shown, the amount of false positives, and the lighting. Despite their rarity and lack of commercial availability, Arden plates are occasionally employed in conjunction with specialized scoring techniques.[139] There are grating and letter tests that are used nowadays to measure contrast sensitivity. The visually comparative test system (VCTS; formerly known as the VisTech chart) was the first of its kind and was created by Ginsburg.[140] A VCTS consists of six rows of sine-wave grating patches with spatial frequencies ranging from 1 to 24 c/d. Above the threshold is the initial grating sample patch. The contrasts in these eight patches range from zero to slightly above or below the threshold. Out of the three possible orientations for gratings, the one with the highest threshold is shown at the very bottom of the figure. Each patch might have a grating in one orientation or be blank; it's up to the patient to decide. The test procedure typically lasts around six minutes. Similar to the VCTS, another grating chart test is the Sine-Wave Contrast Test (SWCT). In contrast to the VCTS, Ginsburg's Functional Acuity Contrast Test (FACT) (Vision Science Research Corporation, [141] uses a bigger patch size, smooths off patch edges, and uses a step size of 0.15 log, to measure contrast. You can also find view-in or orthorater wall charts in the VCTS, SWCT, and FACT data sets. The test-retest dependability of the VCTS chart can be a hindrance to change screening and monitoring. Reliability coefficients ranging from 0.3 to 0.6, depending on spatial frequency, are observed in study samples that are not restricted to individuals with normal eye health. Within the studied range, coefficients are significantly reduced for low spatial frequencies and high spatial frequencies.[142-144]

A comparison of test-retest reliability is not made public by the FACT, the more recent VCTS. So, it's not a good idea to use the VCTS or anything similar to measure contrast sensitivity in clinical studies or trials, according to a number of authors. These and other issues with clinical research's use of contrast-sensitivity grating tests are detailed in

other articles. [143–148] The visual contrast sensitivity test (VCTS) and its variations may not be the most applicable assessments because the majority of clinical research protocols already assess visual acuity. Since the acuity test uses the VCTS to measure high spatial frequency rows, this method effectively duplicates the results. To save money, researchers should use a middle (or peak) contrast-sensitivity function measure. Among the grating charts is CSV-100, often known as Vector Vision. Clinical experiments for contrast sensitivity have made use of its built-in retro-illumination system, which allows it to be wall-or stand-mounted. [149–151]

For some studies, letter charts aren't as useful as grating transmission-sensitivity tests like the VCTS, SWCT, FACT, or CSV-100.[152-153] The broad-contrast-sensitivity function reveals the many visual filters, ranging from low to high spatial frequencies; it has been proposed that clinical investigations should examine this function. One well-known contrast sensitivity letter chart is the Pelli-Robson chart, which is an adaptation of the Haag-Streit chart.[154] The wall-mounted chart has eight rows, and in each row there are two groups of three letters. At a distance of 1 metre, the viewing angle for each letter sub is 2.86. As can be observed from the graph, the contrast between test letters drops from around 100% at the top to less than 1% at the bottom, and the sensitivity increases by 0.15 log units for each triplet. Thanks to its double-sided design, the chart comes with its own set of letters to represent each eye. The patient is required to repeat the letters on top of the chart until they are unable to do so anymore. If two out of three letters are read correctly, the first scoring method grants 0.15 credit per triplet.[154]

The revised scoring system assigns 0.05 points to every letter.[155] One can find log contrast sensitivity ratings between zero and two and a half on the Pelli-Robson chart. Pelli-Robson scores are used to estimate the peaks of the contrast-sensitivity function.[156,157] Using

the letter-by-letter scoring method, the chart's test-retest reliability is even higher at 0.98 for patients[158], while the original scoring system has great test-retest reliability at 0.86.[154] Results from tests conducted at distances ranging from 0.25 to 4 meters and with a degree of defocus of 2 diopters are unaffected by the chart's scores, thanks to the large font.[159]Due to its relative immunity to different test settings, good test-retest reliability, convenience of administration (3-5 minutes), and availability of normative data, the chart is utilised in numerous epidemiologic research.[160-163]

The correlation between visual acuity and Pelli-Robson contrast sensitivity has been found to be weak in several studies.[163-164] For example, a square wave cycle of 0.4 c/d is used to quantify the sensitivity to contrast at the sharp edge of a brightness profile.[165] The creators of this test opted to employ edges as their focal point due to their ecological validity and the prevalence of edges in our visual environment. While they don't offer any validation data for their edge test, they do mention that the contrast-sensitivity function peak is substantially connected with edge detection .Our knowledge does not extend to newborn and kid contrast-sensitivity testing; nonetheless, a wonderful description was published by the National Research Council.[166]

While most charts measure contrast sensitivity, others assess low-contrast acuity. Less contrasty ones, similar to acuity charts, include smaller fonts. Low contrast, rather than 85% or higher contrast, is used for the letters. Although the two concepts are not exact, low contrast acuity and contrast sensitivity are associated (correlation range, 0.3-0.5).[167,168] were Because different charts use different methods to evaluate different types of visual contrasts, it's important to choose a chart according to your spatial resolution or contrast threshold. Charts by Regan, [168, 169] Bailey-Lovie, [170] and the reduced brightness Low contrast acuity charts from SKILL card[171] have high test-retest reliability.According to studies conducted by

Rosa and Aleci (2022)[172,] there are clinical tools that can measure contrast sensitivity within a specific frequency range. This approach works wonders in healthcare settings. It must be noted, however, that only considering a small subset of spatial frequencies can lead to insufficient information on the patient's visual system and visual function. The capacity to perceive contrast in various spatial frequency ranges is also affected by a number of eye diseases, which usually reveal individual variances. The use of more sophisticated medical equipment would be necessary to obtain the complete CSF, yet doing so would be advantageous. As an added complication, there is a dearth of consistently reliable clinical procedures. Visual acuity is the most easily grasped notion, whereas explaining topics like CSF, contrast sensitivity, and contrast threshold can be challenging for patients. Correct assessment of contrast sensitivity and CSF may necessitate additional education for doctors and patients alike.

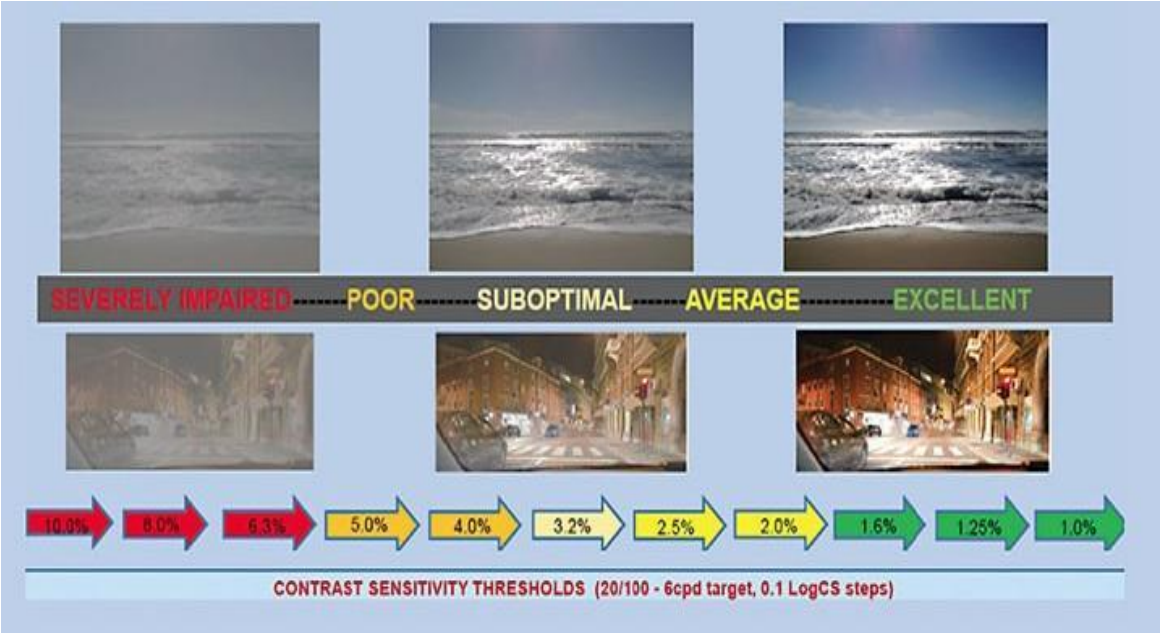


Figure 8: Pictures showing incremental contrast levels from from left to right

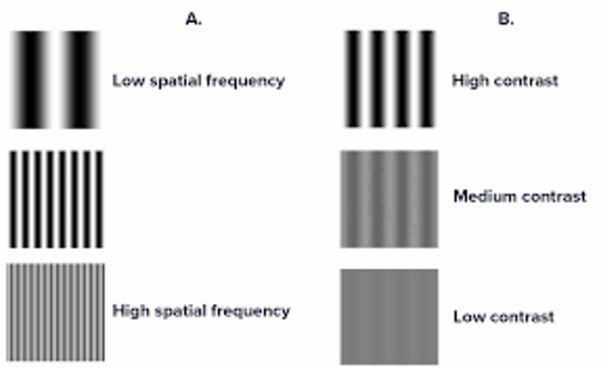


Figure 9: To measure contrast sensitivity, gratings with black and white stripes of different widths (known as spatial frequency, image A). For each frequency, they determined the minimum amount of contrast (separation from black and white, image B) that would enable the subjects to still see the grating pattern

THE EFFECT OF DIABETES ON CONTRAST SENSITIVITY:

Patient with IDDM who showed no signs of retinopathy exhibited notable declines in contrast sensitivity compared to individuals without diabetes,[173,174] notably in spatial frequencies in the mid to high range.[174] Di Leo et al. (1996) also showed that similar changes happen in both dynamic and static modes, with the latter being more responsive to early modifications. In contrast, Sokol and colleagues[175] found that patients with IDDM who had no retinopathy exhibited normal contrast sensitivity.

ASSOCIATION BETWEEN DIABETES TYPES AND CONTRAST SENSITIVITY:

Multiple studies have included both insulin-dependent and non-insulin-dependent diabetic patients in their cohorts.[173] Sokol et al. (1997)[175] found that contrast sensitivity changes noticeably at a rate of 22.8 cycles per degree. The results were out of the ordinary in the group comprising individuals with NIDDM, while they were within the usual range in the group comprising individuals with IDDM ($P < 0.01$). On the other hand, Trick et al. (1994),[176] found no differences between the IDDM and NIDDM patients they studied.

THE IMPACT OF DIABETES DURATION AND GLYCEMIC CONTROL ON COTRAST SENSITIVITY:

A significant decrease in contrast sensitivity was associated with short-term IDDM at all spatial frequencies (with the exception of the highest one). Additionally, two separate studies have demonstrated a strong inverse relationship between contrast sensitivity and the length of diabetes, in both insulin-dependent diabetes mellitus (IDDM) and non-insulin-dependent diabetic mellitus (NIDDM) patients with longer duration of disease.[175,176] A separate study by Buckingham and Young, however, did not find this correlation.[177]

The correlation between deteriorating contrast sensitivity and insufficient glycaemic management, as assessed by HbA1c, has been demonstrated in two separate studies. Banford et al. [179] found a significant correlation (r 520.142) for 6 and 12 cycles/degree of spatial frequency, whereas Di Leo et al. [178] found positive associations (r 5 0.34 ±0.51) at different spatial frequencies. Researchers, on the other hand, failed to detect any link between variations in contrast sensitivity and changes in haemoglobin A1c levels.[176]

INFLUENCE OF AGE ON CONTRAST SENSITIVITY:

Contrast sensitivity in adults with diabetes has been the primary focus of most studies reported in the literature. Furthermore, a particular analysis of the results was carried out for the children's subset of the research population.[175] Significant differences in contrast sensitivity ratings were seen at two spatial frequencies between the non-diabetic control group and the children with diabetes in the study.

CONTRAST SENSITIVITY AND ADAPTATION:

The human eye exhibits remarkable adaptability to function effectively across a broad range of lighting conditions. Humans have the ability to navigate in various lighting conditions, such as a forest illuminated solely by stars, where each rod in the eye is exposed to around one photon each minute. Additionally, humans can navigate in the bright sunlight on a beach, when the cones in the eye absorb thousands of photons every second. The average disparity in brightness between a darkly lit woodland and the midday sun can be up to a hundred million times. Nevertheless, the broad range of brightness levels poses a computational challenge for the retina, as ganglion cells are only capable of producing 20 action potential impulses within the roughly 100 ms integration period of a postsynaptic neuron. Ganglion cells must constantly adapt their sensitivity to transform a broad range of light intensities (about 8 log units) into a more limited range of the original signal (roughly 1-2 log units).[180-184]

The retina employs light adaptation mechanisms to continuously regulate its sensitivity based on the average illumination intensity, thereby compensating for discrepancies between input and output impulses. The mechanisms of light adaptation encompass the following: the transition between rods (used for night vision) and cones (used for day vision), the inherent capacity of individual photoreceptors to adjust their sensitivity to the average light intensity of the day, and the processing that occurs in the retinal circuit after the photoreceptors. The ultimate objective of these mechanisms is to modify the ganglion response in such a way that it no longer conveys information regarding the exact intensity of the detected light, but instead focuses on the contrast and the relative deviation from the typical light intensity.[184]

The contrast of a visual stimuli is a stronger characteristic of the visual impulse compared to information on the exact brightness level. The relative reflectance of an object

to the background is a constant quality, unlike absolute reflectance, which depends on the intensity of illumination. Contrast sensitivity testing cannot fully substitute visual acuity testing, as previously believed, but it can offer essential and objective insights on the challenges faced by individuals in their daily activities.[184]

CAUSES OF REDUCED CONTARST SENSTIVITY:

Optical causes:

Refractive errors can cause a decrease in contrast sensitivity for low and medium spatial frequency values, especially when the refractive errors are more severe. Conversely, lower refractive errors result in reduced ability to distinguish fine details at higher spatial frequencies.

In the early stages of keratoconus, there is a decline in visual acuity before a decrease in contrast sensitivity, particularly in lower spatial frequencies. As the disease advances, the decrease is also seen in higher spatial frequencies.

Cataracts, particularly posterior subcapsular cataracts, predominantly result in a reduction in contrast sensitivity at higher spatial frequencies. Nevertheless, there will be a marginal reduction in sensitivity observed in mid and low spatial frequencies. Advanced cataract is linked to a reduction in the ability to distinguish contrasts at all levels of spatial frequency.

Corneal and intraocular refractive and cataract surgery can result in a reduction in contrast sensitivity, which becomes apparent when there is inadequate correction of refractive errors. The operation may cause additional complications that can impact the patient's sensitivity to contrast. The primary factors contributing to these conditions are the development of higher-order aberrations, the accumulation of neocollagen in the stroma, corneal edema, and the presence of surface corneal opacity following excimer laser surgery.[185]

A multifocal intraocular lens differs from a monofocal intraocular lens in that it results in a reduction in contrast sensitivity at low spatial frequencies. Secondary cataract, also known as posterior capsule opacification, results in a reduction in the ability to distinguish contrasts, particularly at higher and middle spatial frequencies. Even after undergoing Nd YAG capsulotomy, some decrease in contrast sensitivity persists. Uncorrected astigmatism in soft contact lenses leads to a reduction in higher spatial frequencies. Deposits on contact lenses can lead to a reduction in medium and upper spatial frequencies, unlike new contact lenses.[186]

Retinal involvement:

Age-related macular degeneration leads to a reduction in the ability to perceive differences in contrast across all spatial frequencies. Patients with the incipient type experience a reduction in higher spatial frequencies and a decrease in the apex of the contrast sensitivity curve. The disease's progression is accompanied by a continued decline in the contrast sensitivity curve. Cystoid macular edema mostly results in a reduction of higher spatial frequencies.

Diabetic retinopathy is characterized by a reduction in the ability to respond to changes in visual stimuli across all spatial frequencies. Tapetoretinal degeneration and central serous chorioretinopathy also lead to a change in contrast sensitivity.[185,186]

Optic nerve dysfunction:

Optic neuritis: there is a reduction in different spatial frequencies. Glaucoma can lead to a decrease, particularly in intermediate or all spatial frequencies. The decline happens prior to the onset of visual impairments. The changes in contrast sensitivity during this phase are a result of the enlargement of the excavation of the optic nerve and a reduction in visual acuity.

The contrast sensitivity reduces even more as the excavation expands and the field loss progresses along the perimeter.[186]

Others:

Amblyopia: Reduced ability to perceive differences in contrast is present in all types of spatial frequencies.

Disorders related to metabolism:

Exposure to toxic substances can have specific effects on the human body. Alcohol consumption can lead to a reduction in all spatial frequencies, while organic solvents can cause a decrease in contrast sensitivity specifically in the middle spatial frequencies. Neurological disorders include Parkinson's disease, Alzheimer's dementia, cortical vision impairment, and multiple sclerosis.[186]

CONTRAST SENSITIVITY TESTING:

In clinical practice, there are two main types of contrast sensitivity tests that are classified by the manner of study. There will be both letter-based and sine-grid-based tests here. To determine contrast sensitivity, we use the Weber's law–based contrast definition in our letter tests. In this sense, contrast is defined as the ratio of the letter's brightness to the backdrop's brightness, divided by the background's brightness. The following is the mathematical expression for this notion of contrast:

$$K = \frac{(L_p - L_o)}{L_p}$$

where: K – contrast

–background brightness

–letter brightness

The second technique involves measuring contrast sensitivity by utilizing a sine grating. Contrast is determined based on Michaelson's definition, which calculates the difference between the greatest and lowest observed brightness and divides it by their sum. The mathematical expression can be represented as;[187]

$$K = \frac{(L_{max-L_{min}})}{(L_{max} + L_{min})}$$

where: K – contrast

–maximum contrast

minimum brightness[

QUANTIFICATION OF CONTRAST SENSITIVITY USING SINE STRIP TECHNIUQE:

The standard method for evaluating contrast sensitivity entails using a sine grating, which is a pattern consisting of alternating light and dark stripes. To comprehensively evaluate contrast sensitivity at all spatial frequencies, one can use sine stripes of different lengths and contrasts for measurement purposes. The wide range of computed spatial frequencies enables a more precise investigation of certain visual pathways. The clinical efficacy of sine bar tests in determining contrast sensitivity surpasses that of contrast sensitivity letter boards. AP Ginsburg developed one of the early studies for contrast sensitivity using the sine strip technique in 1984. The board utilized by Ginsburg features a thorough set of 45 targets, each incorporating sine strips. These targets are grouped into five main classes based on the spatial frequency employed. The spatial frequency used includes measurements of 1, 5, 3, 6, 12, and 18 cycles per degree, which are divided into five separate rows.The assessment of contrast sensitivity entails a nine-step procedure, in which different

levels of contrast are classified into nine columns, each displaying uneven increments of contrast decrease. The average discrepancy between individual contrasts is 0.25 logarithmic units. The patient is recommended to consciously distinguish the alignment of the stripes, namely whether they are positioned vertically, to the right, or to the left. The patient's ultimate precise response is recorded for each spatial frequency. The contrast sensitivity curve is formed by combining the measured data. The deviation of the contrast sensitivity curve can be evaluated in numerous cases. An aberration from the standard distribution of the curve is seen through a universal decrease in contrast sensitivity across all spatial frequencies. An anomaly can be detected if there is a discrepancy of over 2 logKC between the right and left eye in a single spatial frequency, or if there is a difference of one logKC unit in two or more spatial frequencies. To evaluate contrast sensitivity, researchers created boards that could be used at both close and far distances. Ginsburg's boards served as a catalyst for the creation of innovative models designed to measure contrast sensitivity. This category includes two devices: the lighted Multivision Contrast Tester MCT8000 container and the VCTS6500 wall board (Visual Contrast Test System). The VCTS6500 wall board consists of nine columns of test marks. The sine test marks exhibit varying spatial frequencies across many columns, and their contrast decreases with each row. The contrast reduction rate demonstrates non-constant behavior, with an average decrease of 0.25 logKC. The sine grid in the targets can be oriented either vertically or at a 15° angle to the right or left. It is the individual's responsibility to determine the alignment of each objective.[187-189]

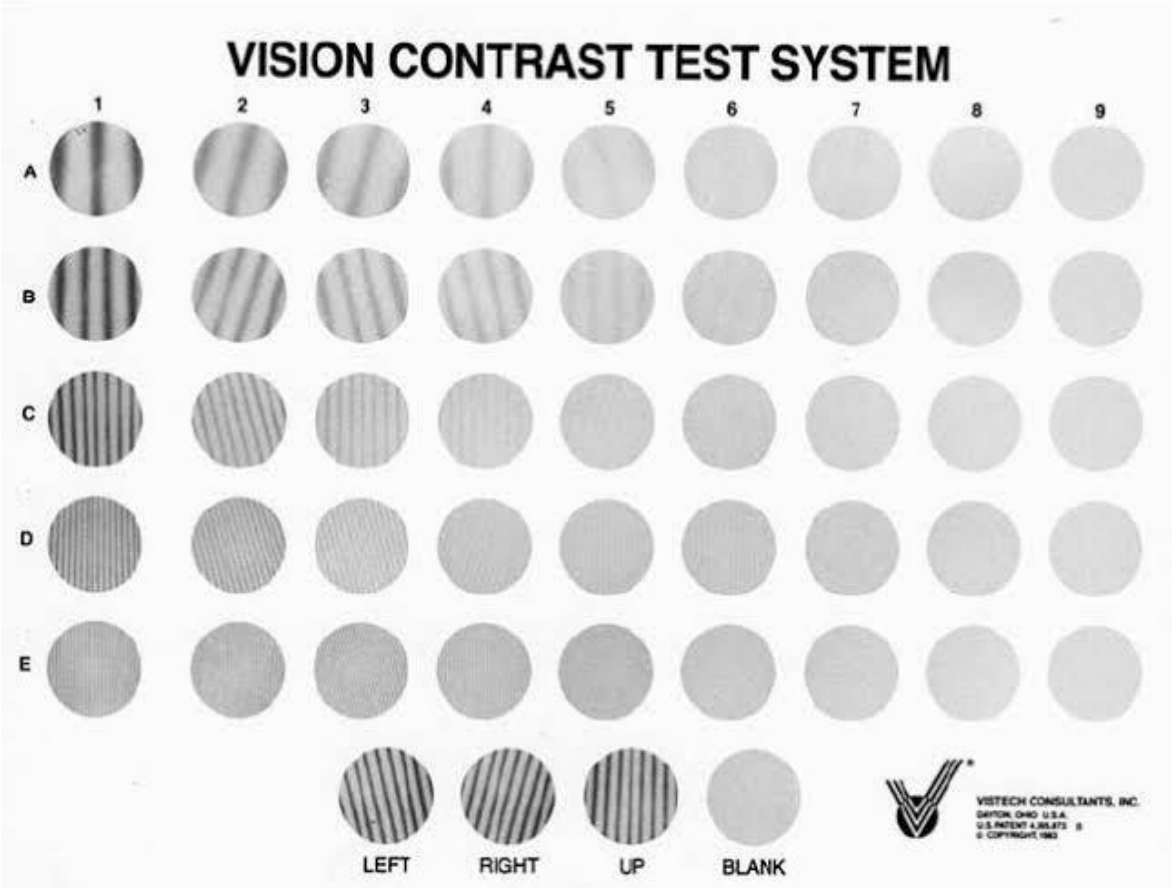


Figure 10: Contrast sensitivity testing system

The second version of tests measuring the sensitivity to differences in sine waves is represented by the nine-scale FACT (Functional Acuity Contrast). This test, renowned for its improved ability to be replicated, consistently decreases the difference in brightness between individual targets by a lower amount of 0.15 logarithmic units. Nevertheless, this decrease in contrast sensitivity leads to a more limited spectrum of detectable contrast sensitivity for an equivalent number of targets. Currently, FACT boards, coupled with ETDRS boards, serve as the cornerstone for the computer-based methodology of measuring contrast sensitivity. The CST1800 Digital software, in conjunction with the online CST vision screener and FVTB, provides functional analysis capabilities for information technology EYEVIEW. These tools include quantified scores and visual representations of contrast sensitivity and visual acuity.

They also include a visual perception display that compares it to the functional vision test battery. In addition, the software include a program that simulates night driving and also contains assessments for color and spatial vision.[188,189]

The CSV-1000E board is widely used worldwide and consists of four rows of targets with sine stripes at four different spatial frequencies (3, 6, 12, 18 cycles per degree). The board is being observed at a distance of 2.5 meters. The CSV-1000RS board possesses a unique characteristic in the form of a singular configuration of sine bar targets, functioning at a spatial frequency of 12 cycles per degree. Furthermore, it includes an ETDRS optotype that allows for visual acuity ranging from 0.2 to 2.0. If the contrast sensitivity is below the average range, it is recommended to undergo a CSV-1000E examination. The CSV-1000S board was created by combining two sets of targets with sine stripes. The spatial frequency of these stripes are 6 and 12 cycles per degree, respectively. A 0.1–1.33 micron ETDTS optotype is also included on the board.

The test also has 5 real-life scenarios that vividly demonstrate the different degrees of visual perception quality that arise when visual functions deteriorate. These scenes might clarify the change in perception that is experienced by either the patient or a family member. In addition, there are contrast sensitivity tests that make use of alternative optotypes. An example that demonstrates this is CSV – 1000SlanC, which shares similarities with CSV – 1000S when including Landolt rings. The CSV-1000CVA board is used to assess contrast sensitivity and visual acuity at low contrasts. The test consists of two rows of targets decorated with sine stripes, with one row measuring 6 cycles per degree and the other measuring 12 cycles per degree. Additionally, the test includes ETDRS optotypes with a contrast of 3 degrees.

Wachler and Krueger created the methodology called normalized contrast sensitivity. This approach entails creating a scoring system with values that range from 0.1 to 1.35

logKC, which align with the objectives of the CSV-1000 boards. The scoring scale is derived from the examination of results collected from a population that follows a normal distribution. The board was designed to simplify and streamline the evaluation of contrast sensitivity, eliminating the need to create curves. Four cycles per degree is the spatial frequency at which the Cambridge Low Contrast Chart is tested. Eleven plastic sheets are laid out in a timetable-like arrangement for the test. In each set of two sheets, you'll find a standard grid on one side and a similar pattern with vertical light stripes on the other. The initial pair displays a notable disparity. The contrast decreases gradually with each subsequent pair. The patient reports perceiving horizontal light stripes on the exhibited boards.[187-189]

LETTER CONTRAST SENSITIVITY:

Another often employed approach in clinical practice is letter contrast sensitivity, which entails assessing the ability to discern the contrast of letter optotypes. The authors of the Pelli-Robson table have linked the increase in the number of techniques used to measure letter contrast sensitivity to many variables. 1) Letter contrast sensitivity tests evaluate the patient's ability to perceive different spatial frequencies when their natural contrast sensitivity is at its highest level. The apex of the contrast sensitivity curve is established based on the collected data, while the far end of the curve shows the vision's ability to perceive contrast in high spatial frequencies. Usually, these two data points are sufficient for determining the entire trajectory of the contrast sensitivity curve. 2) The use of tests to measure letter contrast sensitivity is simple and effective, and it is somewhat different from evaluating visual acuity using optotypes.[188,189]

The procedure for quantifying letter contrast sensitivity is evaluating the visual acuity of the patient necessary to distinguish between relatively large letters of the same dimensions. Multiple PelliRobson boards of different types can be affixed to the wall. The

test consists of 16 sets of letters, arranged in 8 rows, with each row including two sets of letters. When standing 1 meter away, each line may be seen at an angle of 2.8 degrees. Each triplet has an equal contrast value, whereas the letter contrast lowers by 0.15 logarithmic units for each succeeding triplet. The patient exhibits the behavior of reading from left to right and from top to bottom. The patient's contrast sensitivity logarithm is determined by subtracting the last triplet from the scoring sheet in which the patient correctly read two out of the three letters. The contrast sensitivity is assessed on a logarithmic scale ranging from 0.00 to 2.25 units. A limitation of the test is the need for a large wall space and the demand for high-quality and consistent lighting. Other disadvantages include the possibility of damage to the plastic boards and a potential change in the legibility of individual letters due to the degradation of the material caused by continuous exposure to light.[188,189]

Figure 11: Pelli-Robson Chart for measuring contrast sensitivity

Arditi created the Mars letter contrast sensitivity test, which is derived from the Pelli-Robson board principle and employs ETDRS optotypes, consisting of sets of three letters with different levels of contrast. This exam has the following advantages:

The test shows that by using much smaller contrast reduction steps (0.04 log units) between triplets, there is a 28% improvement in measurement accuracy. This technique allows for the use of the same normative data as that used for Pelli-Robson boards. The gadget has a small size of 22.8x35.6 mm, which makes it easier to achieve even lighting.
The instrument is portable and ideal for evaluating contrast sensitivity at a distance of 50 cm. Storing the plates separately during measurements is more convenient, leading to a longer lifespan of the test plates.

The test's repeatability is improved by using three different types of boards, which makes it more difficult to remember the optotype letters. Regan's charts, also called the Regen Low Contrast Letter Acuity Chart, allow the measurement of contrast sensitivity on Snell optotypes, unlike previous charts used to assess letter contrast sensitivity. They consist of a collection of four optotype boards offering various contrasts. Regan boards have the limitation of only allowing testing in high spatial frequencies. The Sloan boards, similar to the ETDRS boards, have the advantage of standardization and are derived from the ETDRS boards. The Multi Distance Testing Low Contrast Set is a commercially available collection of Sloan boards that can be used to assess the ability to detect differences in letter contrast at different distances. The test consists of seven sheets that display different levels of contrast, namely 100%, 25%, 10%, 5%, 2.5%, 1.25%, and 0.6%. The investigation was carried out at two distinct distances, namely 1.6 meters and 1.0 meters. Boards that are equipped with E-hooks are specifically developed for children to improve their ability to detect contrasts. The spatial frequencies of these boards are 3 and 6 cycles per degree. The LEA low contrast

symbols and the Hiding Heidi test are suitable for young children who have low contrast sensitivity.[187-189]

Figure 12: LEA test for measuring visual acuity in low contrast

LITERATURE FROM PREVIOUS STUDIES:

A study was carried out by Arend et al. to assess the relationship between contrast sensitivity function, foveal microcirculation, and early diabetes mellitus. Measurements of contrast sensitivity in diabetic patients can help detect ischemic diabetic maculopathy in its early stages, according to the study. There may be more clinical data that can help with the diagnosis if this is done.[190] Examining the contrast sensitivity of diabetic adolescents and young adults, retinopathy included or not, was the primary goal of the research. The goals were to examine the correlation between metabolic control and the severity of retinopathy, measure central vision, and reassess contrast sensitivity after a considerable improvement in metabolic control. The research included a group of 40 diabetics with varying degrees of

retinopathy, as well as a group of 20 diabetic adolescents and young adults who never developed the condition. The sample consisted of twenty healthy adults who were age and sex matched. With spatial frequencies of 3, 6, 12, and 18 cycles per degree, the CSV-1000 contrast sensitivity was evaluated. Contrast sensitivity varied somewhat but noticeably at 18 cycles per degree (cpd) among diabetics who did not have retinopathy ($p = 0.04$). However, at 12 and 18 cpd, subject whose retinopathy was present at baseline showed a statistically significant ($p<0.001$) decline in contrast sensitivity. Contrast sensitivity is drastically lower in patients with preproliferative or proliferative retinopathy compared to controls at all frequencies. Significant improvement in metabolic management was followed by patient assessment. Patients with mild to moderate retinopathy and those with preexisting retinopathy showed an improvement in contrast sensitivity, however those with advanced retinopathy showed no effect. Diabetic adolescents and young adults saw a decline in contrast sensitivity regardless of whether fluorescein angiography revealed retinopathy. Individuals with preproliferative/proliferative retinopathy showed a more noticeable decline. This long-term study is the first to show that people with diabetes can improve their contrast perception, regardless of whether they had retinopathy before the study starting or not.[191]

Sotirios et al. compared the contrast sensitivity of healthy volunteers with that of people who had impaired oral glucose tolerance test (OGTT). Twelve healthy controls and sixteen patients with impaired oral glucose tolerance test (OGTT) were evaluated for contrast sensitivity. Our spatial frequency measurements were taken four times independently. Glucose intolerance was properly characterised in 1985 by the World Health Organisation (WHO). Both datasets shared commonalities in terms of age, visual acuity, refractive correction, and lens opacities. Reduced contrast sensitivity was found to be significantly associated with substandard oral glucose tolerance test (OGTT) results across all spatial

frequencies ($p < 0.001$). Patients who meet both the World Health Organization's 1985 criteria for impaired glucose tolerance and the American Diabetes Association's 1997 criteria for functional visual loss are considered to have diabetes.[192]Three groups of patients were examined in an observational cross-sectional study at Sumatera Utara University Hospital: those without diabetes, those with diabetes for less than five years, and those with diabetes for more than five years. Fifteen people out of forty-five (90 eyes) observed a decline in contrast sensitivity, most likely as a result of diabetes for over five years. In terms of group characteristics, there was no statistically significant relationship between subjects' ages ($p > 0.05$). Given that $p > 0.05$, we may say that age has no bearing on contrast sensitivity. The results demonstrated a correlation between the contrast sensitivity ratings and the patient group's features ($p < 0.05$). There were notable disparities in the contrast sensitivity values across the patient groups with diabetes, those without diabetes, and those with diabetes who were less than 5 years old (p value <0.05).[193]In a recent study, researchers looked at people with Type 2 Diabetes Mellitus (T2DM) who did not have diabetic retinopathy (DR) to see if there was a connection between glycosylated haemoglobin (HbA1c) and contrast sensitivity (CS). The 120 participants in this cross-sectional study had normal eyesight (6/6 in both eyes), type 2 diabetes mellitus (T2DM), and no diabetic retinopathy (DR). The investigation was carried out by the endocrinology department of a tertiary hospital. All of the people there were in their thirties and forties. Lea utilised a discrete symbol size of 10M on the chart for the purpose of CS analysis. We used linear regression analysis to find out whether there was a connection between HbA1c and CS. In a study of 120 patients without diabetic retinopathy (DR) and type 2 diabetes mellitus (T2DM), 83 (or 69.2%) were female. Of the total participants, 64 (or almost 50%) were in the age bracket of 36 to 40. The typical number of years someone has had diabetes was 3.3±1.65 years. The average HbA1c value was 10.46±1.48%, and it was determined to be above 8% in 75% of the people. The average CT

values at 1 metre, 2 metres, 3 metres, and 4 metres were 164.75±21.12, 122.0±45.08, 93.0±45.37, and 58.67±20.04, respectively. A whopping 94.2% of the 113 subjects had typical 1 metre CS, with 170 subjects exhibiting 0.6% contrast. Nearly half (53.3%), or 40 individuals, showed diminished contrast sensitivity at 4 metres, with a much more limited range of 2.5% contrast. A negative association between CS at 3 metres and both the duration of diabetes ($r=-0.855$, $p<0.001$; $R2=0.731$) and HbA1c levels ($r=-0.865$, $p<0.001$; $R2=0.747$) was discovered by the researchers. Consequently, CS declines and HbA1c levels increase as diabetes persists. People with type 2 diabetes mellitus (T2DM) who did not have any issues with their visual acuity or diabetic retinopathy (DR) were the main focus of the study[194]

MATERIALS AND METHODS

MATERIALS AND METHODS

STUDY AREA:

R.L.Jalappa Hospital and Research Centre attached to Sri Devaraj URS Medical College.

STUDY POPULATION:

The source of the population was all participants attending Outpatient Department of Ophthalmology, R. L. Jalappa Hospital and Research, Kolar and the target population who fulfil the inclusion criteria and diagnosed as diabetes mellitus without retinopathy was tested for contrast sensitivity. 53 patients were recruited in the present study.

STUDY DESIGN: Cross sectional study

SAMPLE SIZE CALCULATION:

Sample size was calculated based on the Mean Contrast Sensitivity at (1.119 ± 0.29) among diabetic subjects without retinopathy from the study by Shaili Mishra et al.2 Considering these values at 5% alpha error, and 80% power, and null hypothesis at 1.0, sample size of 48 was obtained. Considering 10% Nonresponse a sample size of 48 + 4.8 ≈ 53 subjects will be included in the study.

Formula used: Sample size (N) = Z1-α/22 SD2 / d2 (16)

Z1-α/2 = Is standard normal variate (at 5% type 1 error (P<0.05) it is 1.96.

SD = Standard deviation of variable. Value of standard deviation can be taken from previously done study or through pilot study.

d = Absolute error or precision

TIME FRAME TO ADDRESS THE STUDY: September 2022 to December 2023.

INCLUSION CRITERIA:

- All patients of either sex from 40 -70 years with Type 2 Diabetes Mellitus without retinopathy

EXCLSUION CRITERIA:

- Presence of congenital colour vision defects
- Spherical power > -6 dioptres or cylinder power > -4 dioptres
- Presence of other retinal or ocular disorders
- Clinical history or evidence of ocular or neurological diseases not caused by diabetes, including glaucoma, trauma, multiple sclerosis, stroke, Parkinson's disease and Alzheimer's disease
- Medications include ethambutol, amiodarone, corticosteroids, and vigabatrin that affect eye function as part of treatment
- Mentally challenged patients
- Recent Photocoagulation.

METHODOLOGY:

This cross-sectional study includes 53 patients who met the inclusion criteria. Diabetes duration, medication, and diabetic control have been recorded following a brief ocular and systemic history and therapy for the same.

Each patient has assessed clinically by the following methods:

1. Visual acuity assessment by using Snellen chart for distant vision.

2. Near vision by using near vision charts.

3. Slit lamp biomicroscopy for evaluation of anterior segment.

4. Evaluation of the posterior segment is carried out following pupil dilation using indirect ophthalmoscopy and +90D biomicroscopy. A thorough evaluation of the retina was carried out in order to exclude the possibility of diabetic retinopathy. Exclusion criteria for the study were the presence of intraretinal haemorrhage or microaneurysms, the first signs of diabetic retinopathy that can be seen through ophthalmology.

5. Laboratory tests were performed to evaluate fasting blood sugar, postprandial blood sugar, and glycated haemoglobin.

6. Contrast sensitivity evaluation in the same consultation room using a Pelli-Robson chart held at a distance of 1 metre and under identical lighting circumstances. The test was ended when the participant failed to properly identify two out of three letters in the triplet using the same contrast, and each letter has a score of 0.05 log units when read correctly. Log contrast units are used to quantify contrast sensitivity. The contrast sensitivity is negatively correlated with the score:

- A score of 2.0 indicates normal contrast sensitivity.
- A score of less than 1.5 is consistent with visual impairment (moderate loss).
- A score of less than 1.0 represents visual disability (severe loss).

Data Processing and Statistical Analysis:

For further processing, the data was exported from an Excel data sheet into SPSS 22. The mean ± standard deviation was used to represent continuous variables, whereas percentages were used for all categorical variables. To find out whether the continuous variables were

significantly related, we conducted one-way ANOVA. We used Pearson's correlation analysis to look for a relationship between contrast sensitivity, diabetes duration, and HbA1c. For categorical variables, the chi-square test was employed for significance. Statistical significance was determined when $P < 0.05$.

Ethical Consideration:

Participants were informed that their participation was entirely voluntary and could be terminated at any moment without affecting their medical care, and they were asked to sign a consent form.The institutional ethics committee at Sri Devaraj Urs Medical College gave their approval to the research plan.

RESULTS

RESULTS:

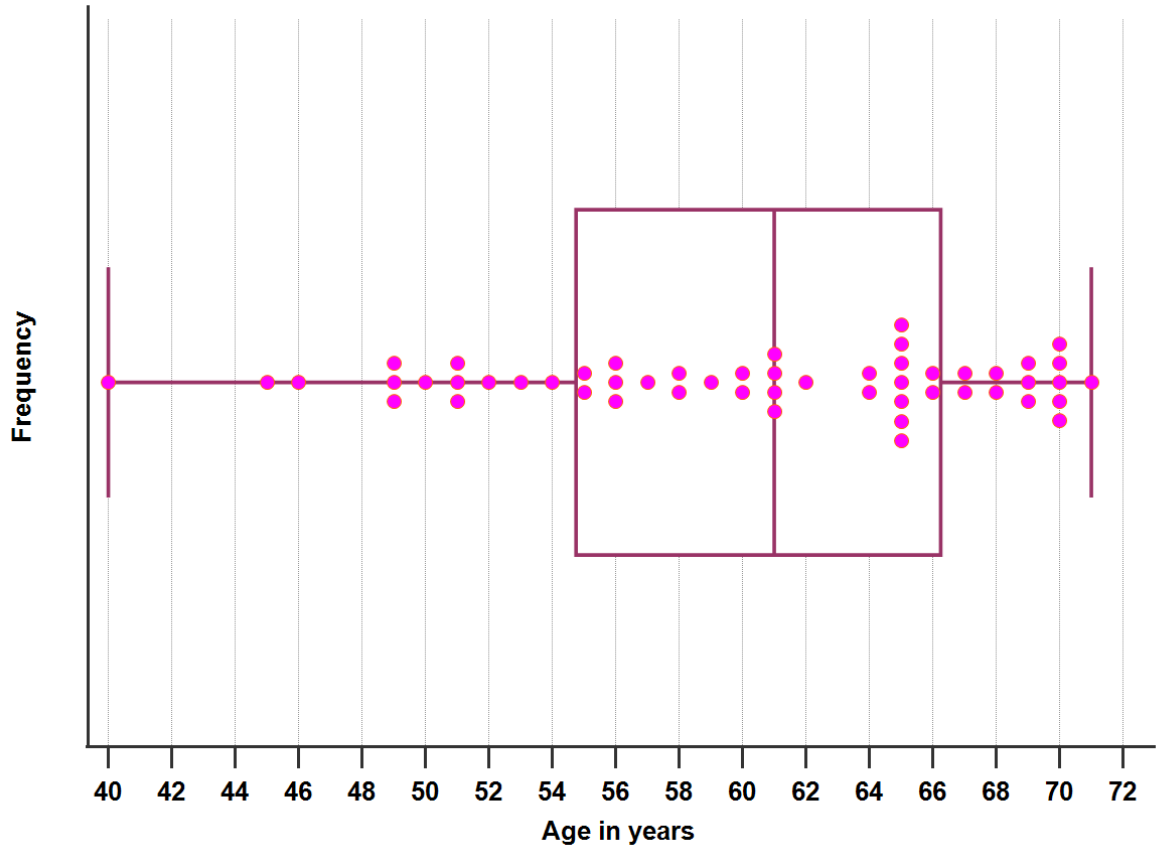
Age

The present study enrolled 53 patients and their mean age was 60.16±7.80 years and ranged from 40 to 70 years (Table 1 &Graph1).

Table 1: Age distribution

Variable	Mean ± SD	95% CI
Age in years	60.16±7.80	58.0186 to 62.3210

Graph 1: Age distribution



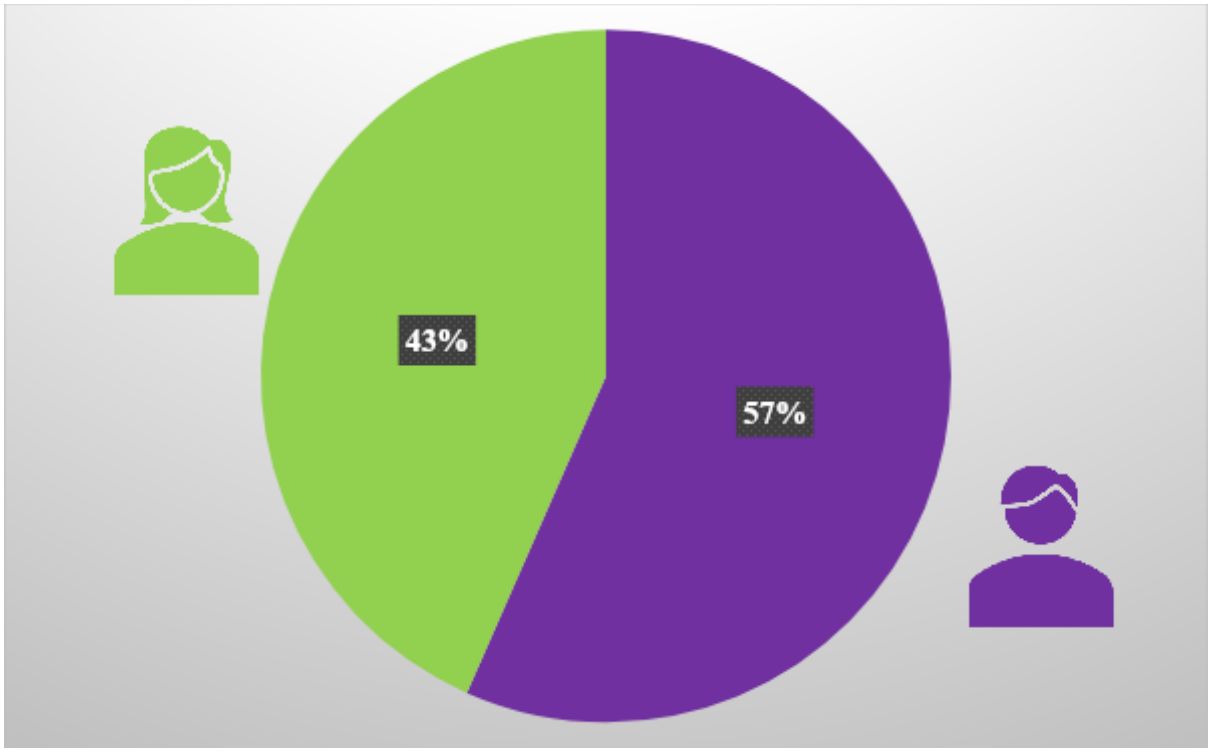
Gender:

The gender of the study population was observed and the results were displayed in table 2. The male patients were predominant (56.6%) and 43.4% of female patients (Graph2).

Table 2: Gender profile

Gender	Frequency	%
Male	30	56.60
Female	23	43.39

Graph2: Gender profile



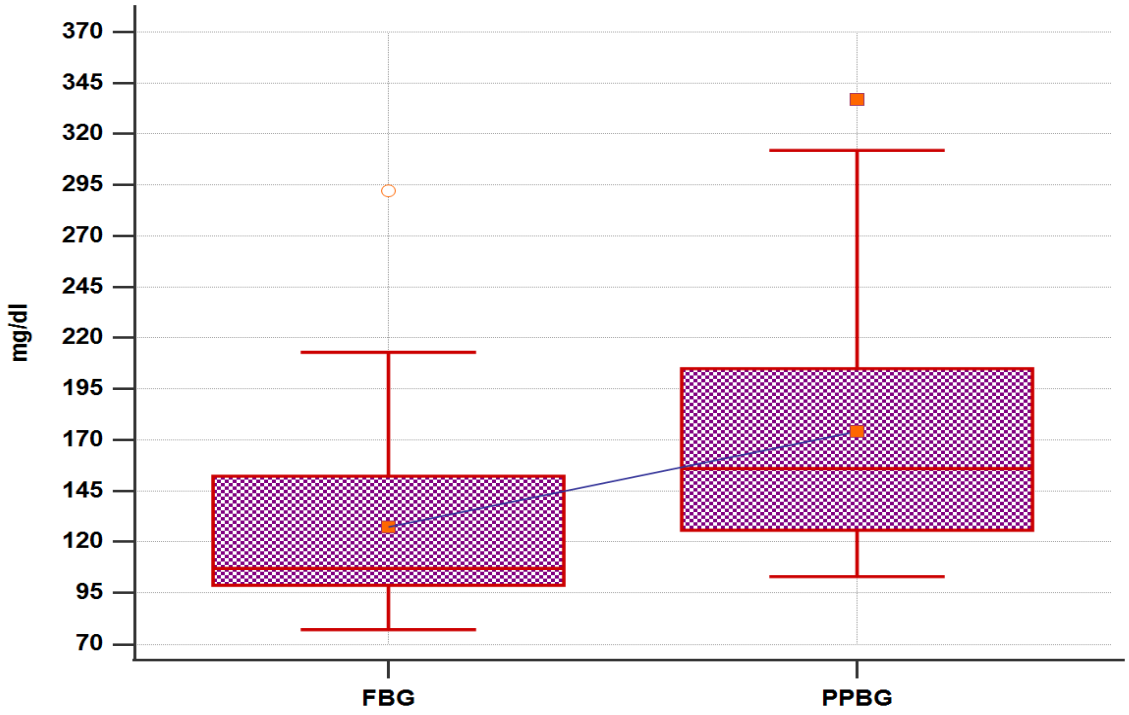
Glycemic profile:

The glycemic profile of the study population was evaluated and the outcome was displayed in table 3. The mean fasting blood glucose was 127.28±40.75 mg/dl and the post prandial blood glucose was 174.37±59.52 mg/dl (graph3). The three months glycemic control marker HbA1c was assessed and the mean was 7.72±1.85 (graph4).

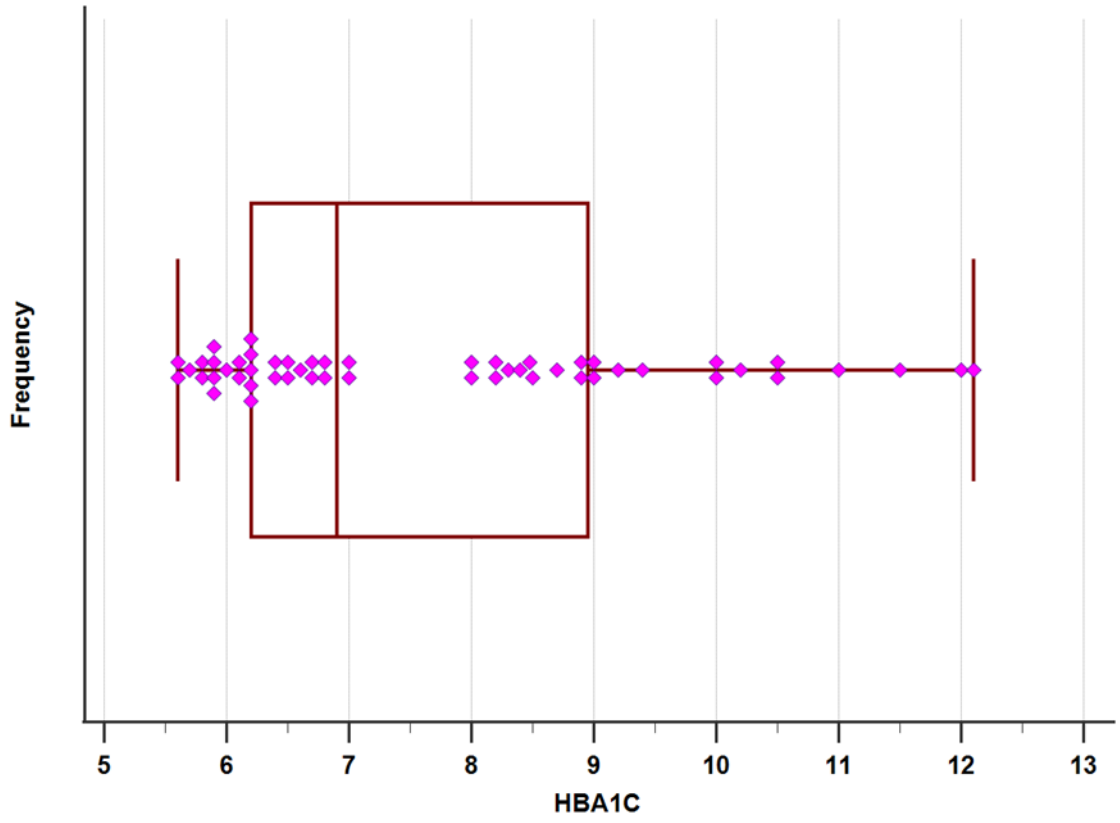
Table 3: Glycemic profile

Glycemic parameters	Mean±SD	95% CI
Fasting Blood Glucose mg/dl	127.28±40.75	116.0499 to 138.5161
Post-Prandial Glucose mg/dl	174.37±59.52	157.9715 to 190.7832
HBA1c	7.72±1.85	7.2087 to 8.2406

Graph 3: Profile of fasting and post prandial blood glucose



Graph 4: Profile of HBA1c levels



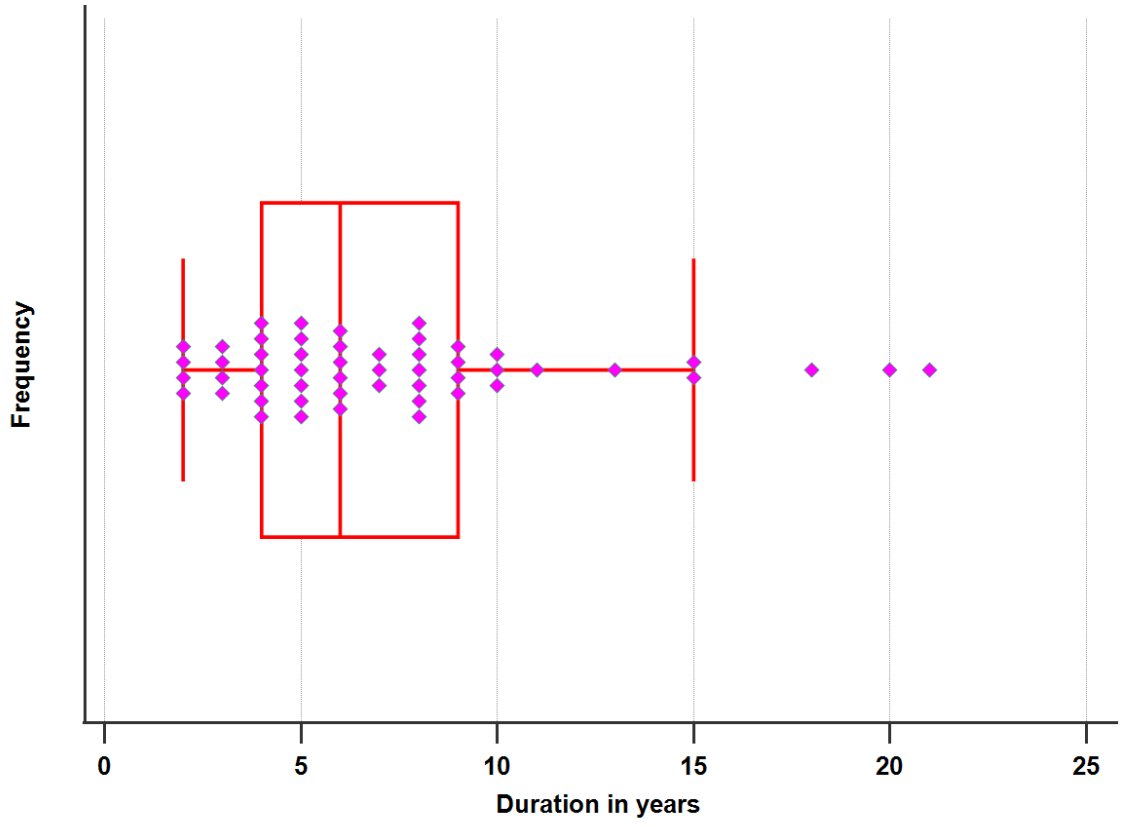
Duration of Diabetes:

In the present study, duration of diabetes was also observed and the mean duration was 7.21±4.37 years (Table 4& graph5)

Table 4: Profile of Duration of Diabetes

Variable	Mean±SD	95% CI
Duration in years	7.21±4.37	5.9945 to 8.4286

Graph 5: Profile of Duration of Diabetes



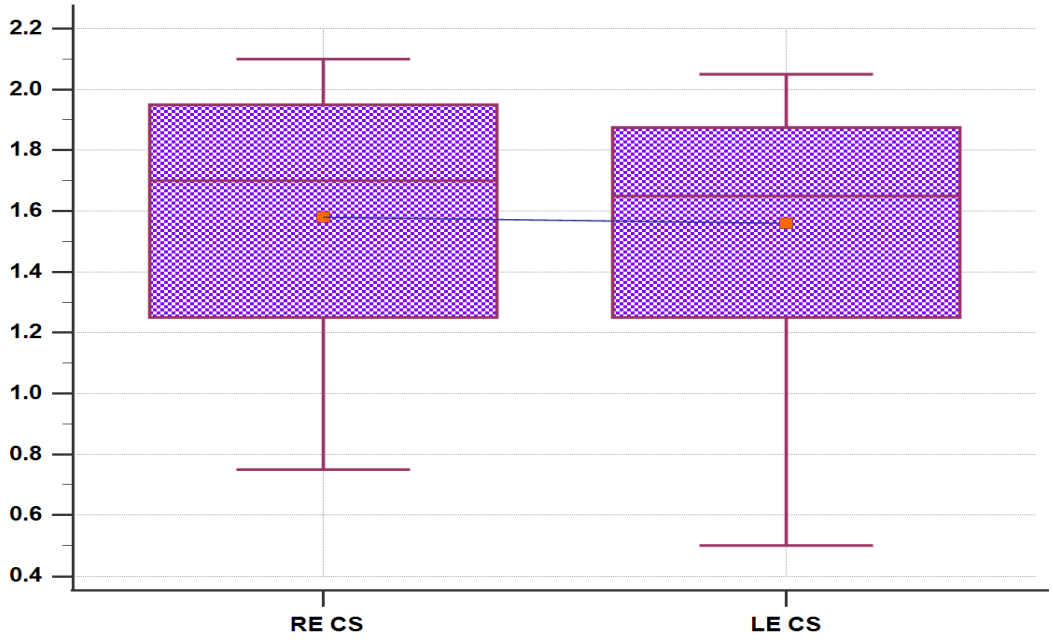
Contrast sensitivity:

The contrast sensitivity testing was used to detect the changes in vision that are hidden by visual acuity. In the present study, the out come of contrast sensitivity was described in table 5. The mean contrast sensitivity for right eye was 1.57±0.37 which indicated some visual impairment. Similarly in the left eye also showed the same (1.56±0.37) (graph6).

Table 5: Profile of contrast sensitivity

Contrast Sensitivity	Mean±SD	95% CI
RE CS	1.57±0.37	1.4737 to 1.6840
LE CS	1.56±0.37	1.4541 to 1.6652

Graph 6: Contrast Sensitivity Profile



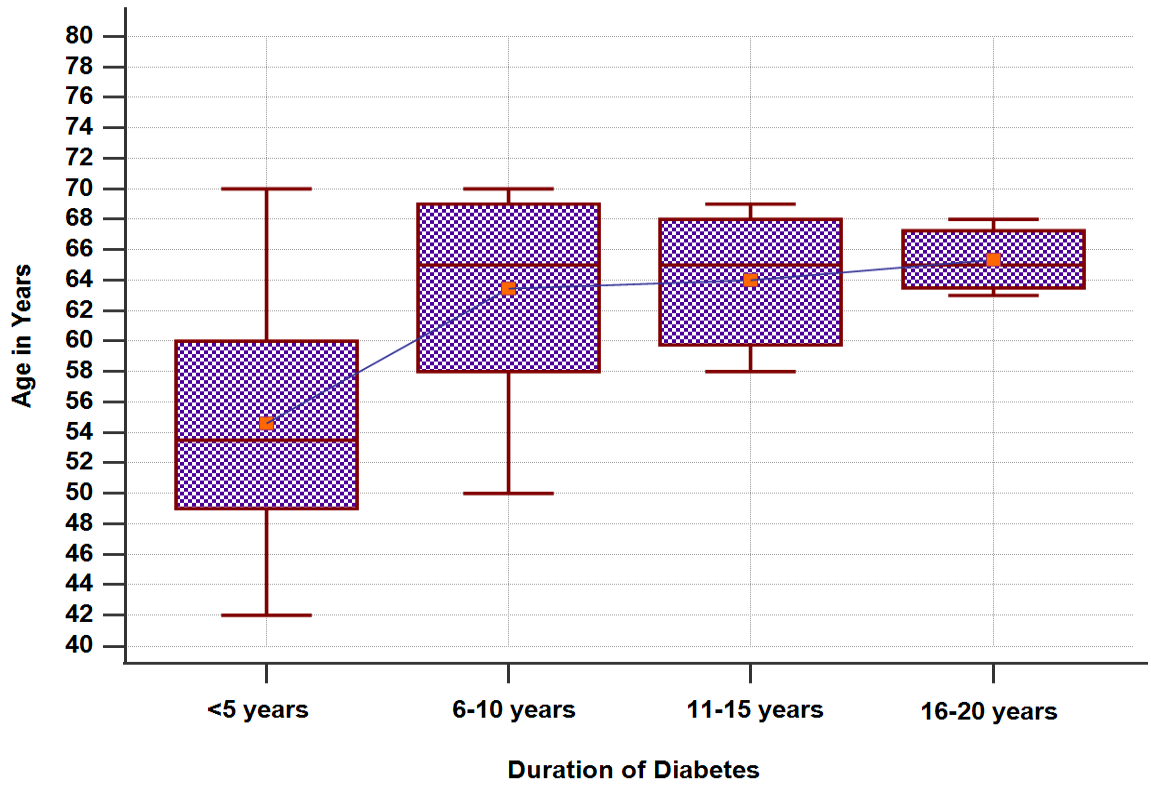
Duration of Diabetes:

Table 6 displays the results of the study's categorization of the population according to the length of diabetes mellitus.Graph1 shows that there was a statistically significant relationship between the duration of diabetes and the average age of the research participants (P<0.001). No statistically significant difference was found between the sexes (P=0.5057) (graph2). The longer a person has had diabetes, the higher their fasting glucose level will be. The disparity was found to be statistically significant (P<0.001), as seen in graph3. The same holds true for the post-prandial blood glucose levels; they grew significantly (P<0.001) as the duration of diabetes did. (graph3). The three-month glycemic marker HbA1c was shown significant difference.The glycemic control was poor when duration of diabetes increased. The difference was statistically significant (P<0.001) (graph4). The duration of diabetes (in years) has shown statistical significance (P<0.001) (graph5). Both right and left eye showed the significant difference in contrast sensitivity which was decreased when the duration of diabetes (in years) increased (P<0.001) (graph6).

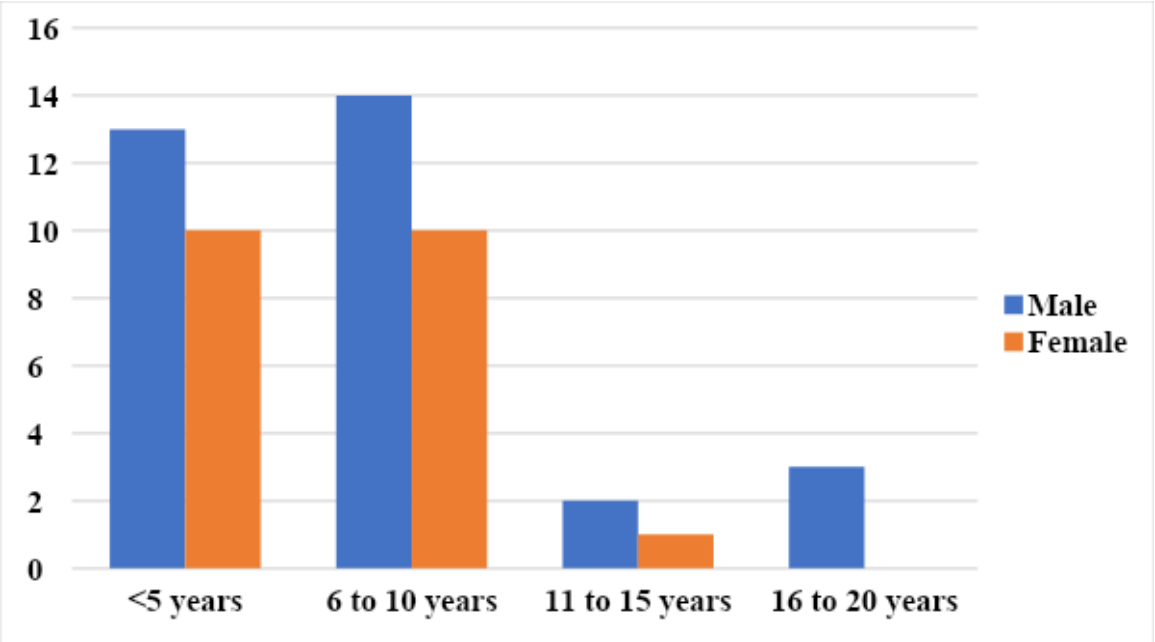
Table 6: Comparison of study variables based on Duration of Diabetes (in years)

Variables	≤5 years N=23	6-10 years N=24	11-15 years N=03	16-20 years N=03	P value
Age in years	54.59±7.50	63.45±6.21	64±5.56	65.33±2.51	<0.001
Gender (M:F)	13:10	14:10	2:1	3:0	0.5057
FBG mg/dl	102.90±19.55	133±33.03	156.33±11.93	185.66±24	<0.001
PPG mg/dl	138.68±38.52	185.90±50.64	213.66±27.20	272.33±53.87	<0.001
HBA1c	6.22±0.54	8.14±1.31	10.3±1.08	11.7±0.60	<0.001
Duration in years	3.77±1.10	7.68±1.32	13.66±2.30	19.66±1.52	<0.001
RE CS	1.85±0.21	1.47±0.33	1.23±0.07	0.91±0.20	<0.001
LE CS	1.84±0.21	1.45±0.33	1.28±0.14	0.9±0.18	<0.001

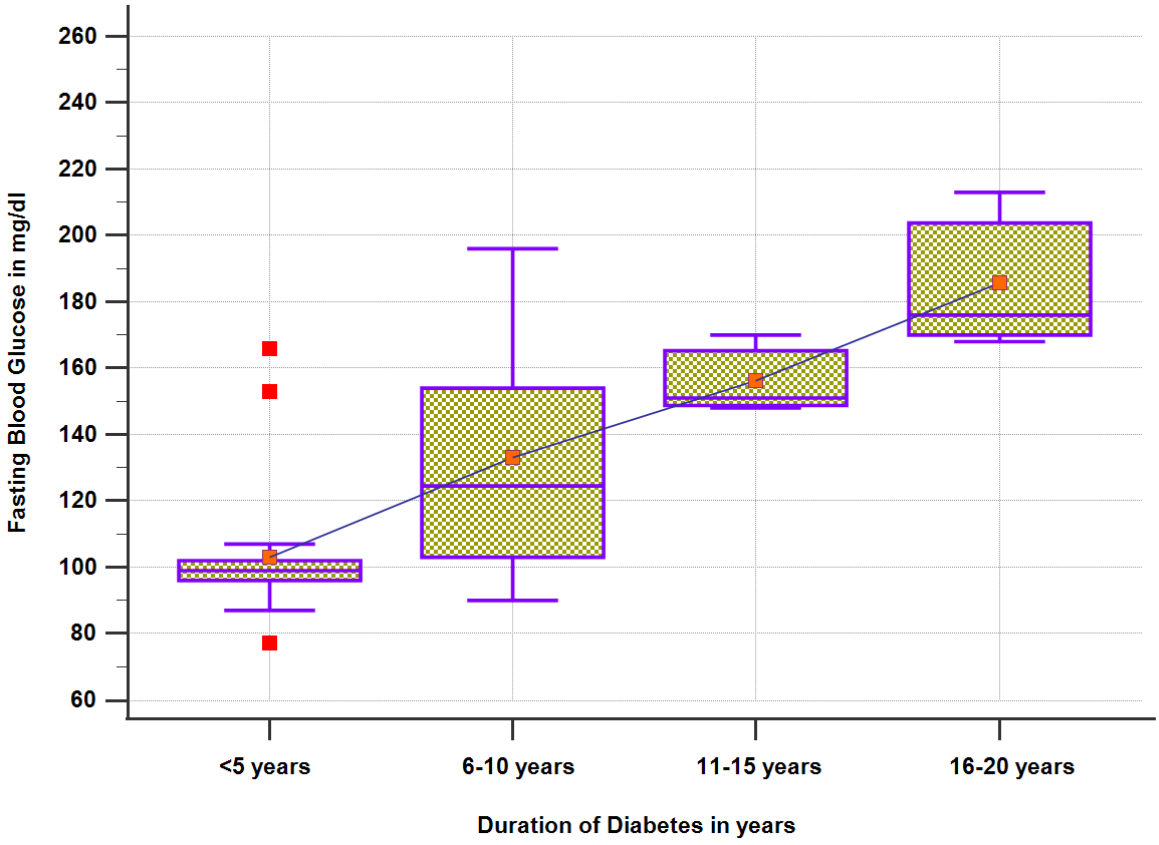
Graph 7: Comparison of Age



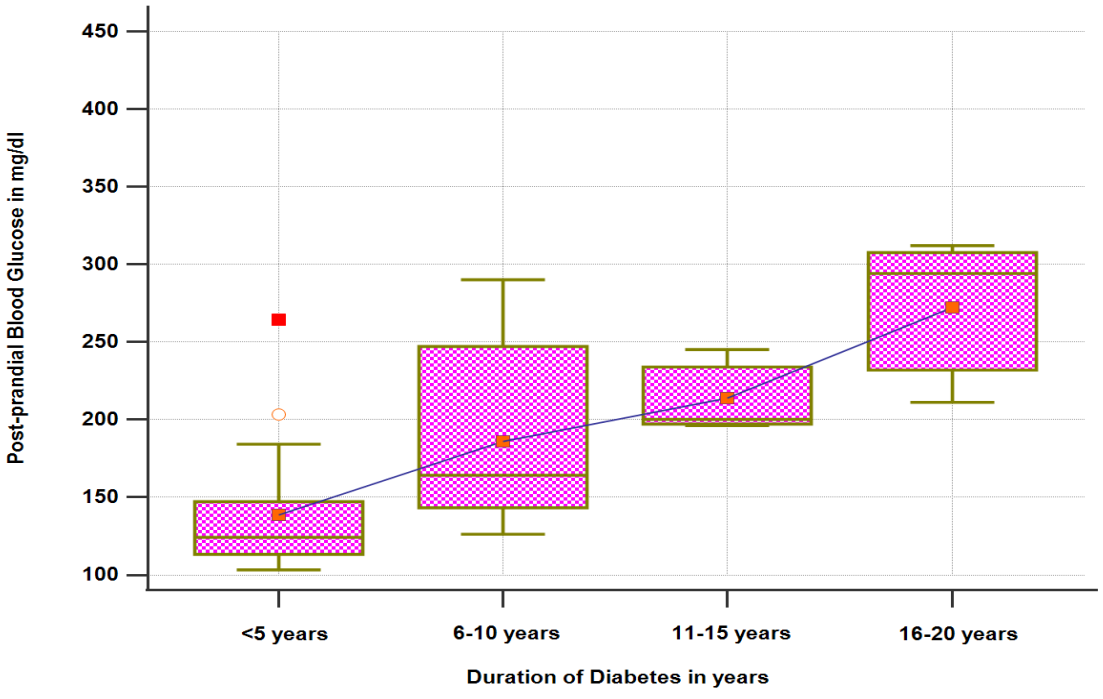
Graph8: Comparison of Gender



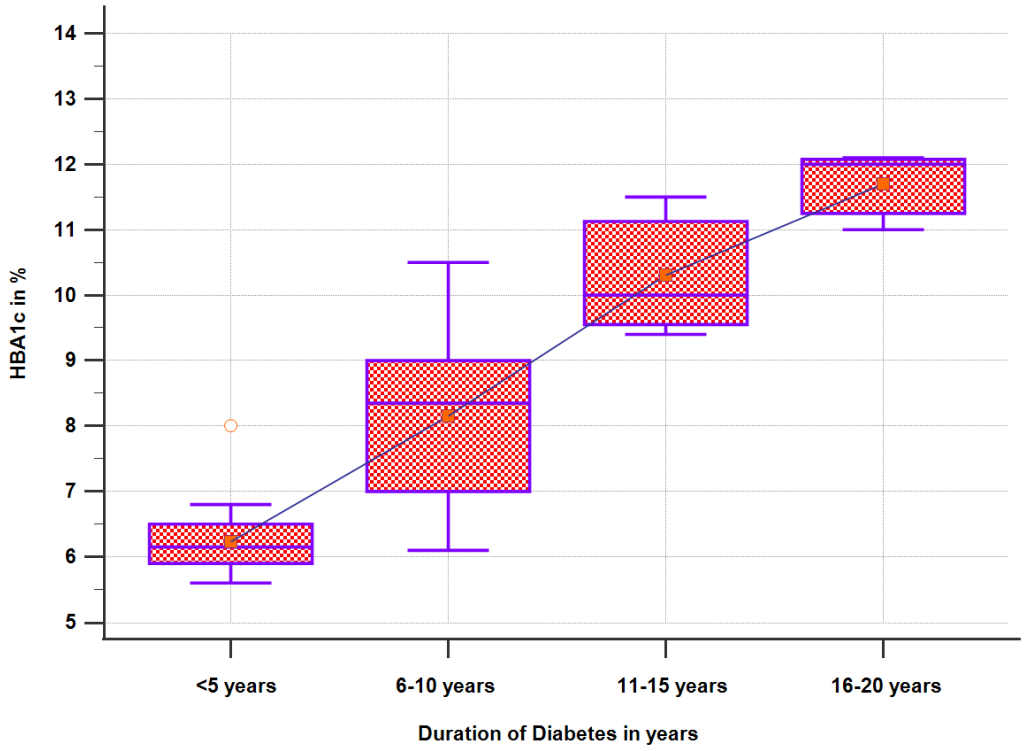
Graph9: Comparison of Fasting Blood Glucose



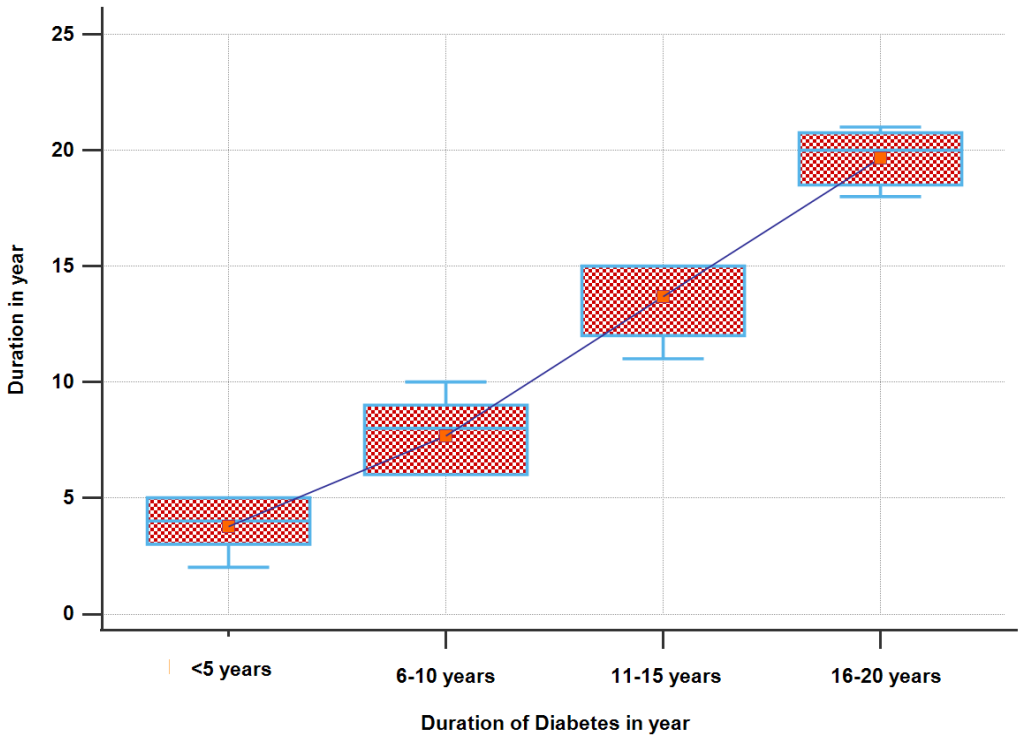
Graph10: Comparison of Post-prandial Blood Glucose



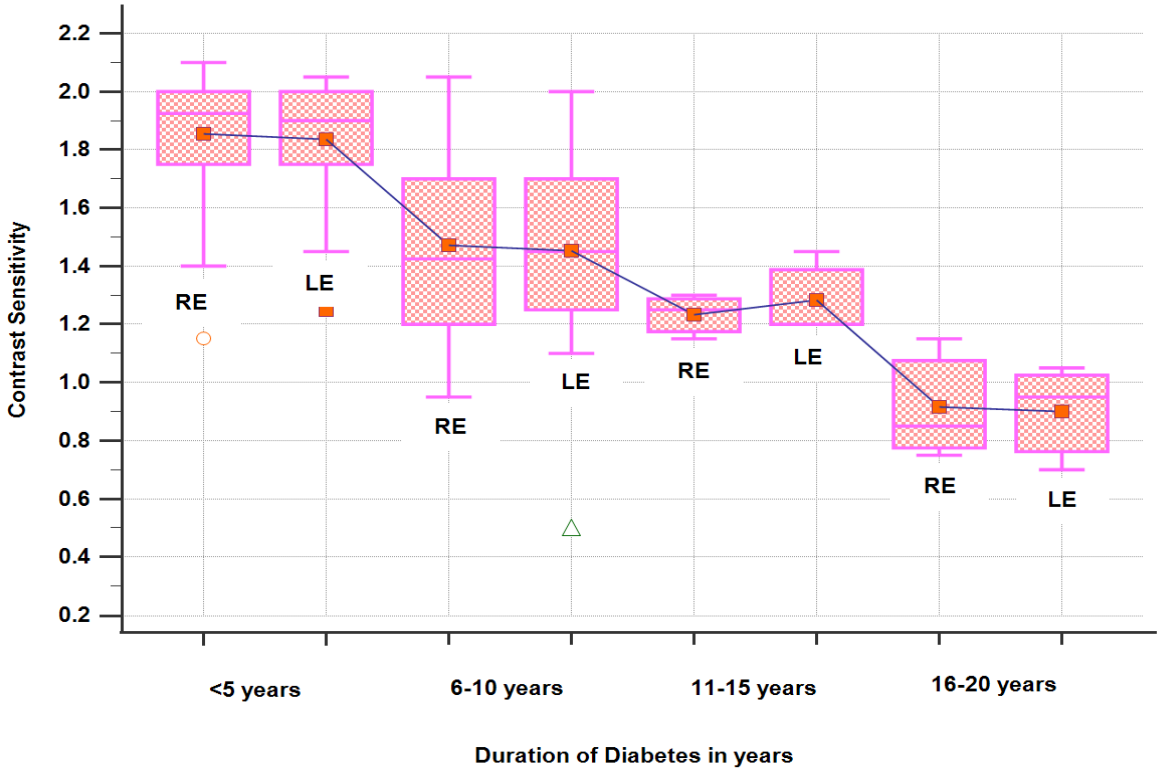
Graph11: Comparison of HBA1c



Graph12: Comparison of Duration of Diabetes



Graph13: Comparison of Contrast Sensitivity



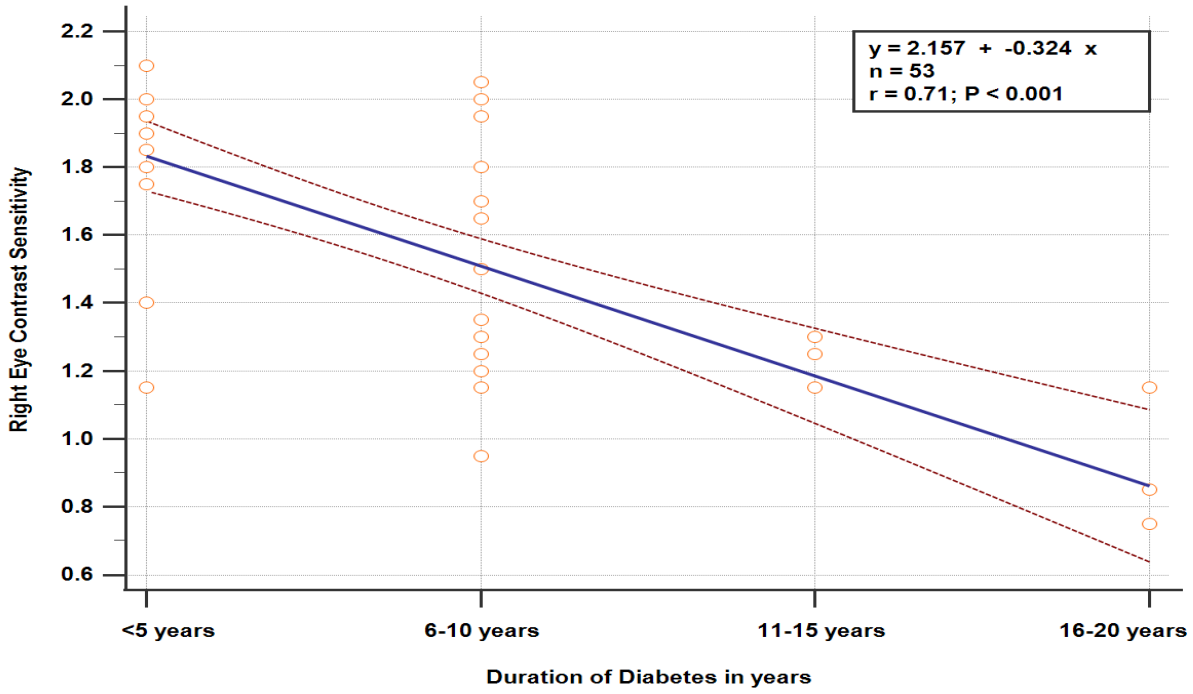
Correlation of contrast senstivity with duration of DM :

The contrast sensitivity was correlated strongly with the diabetes duration (in years) on both the eyes (Table 7). The negative correlation was observed in the present study which indicated that as duration of diabetes increased, the contrast sensitivity decreased on both right eye (r=0.7097; P<0.0001) and left eye (r=-0.6990; P<0.0001) (graph14&15)

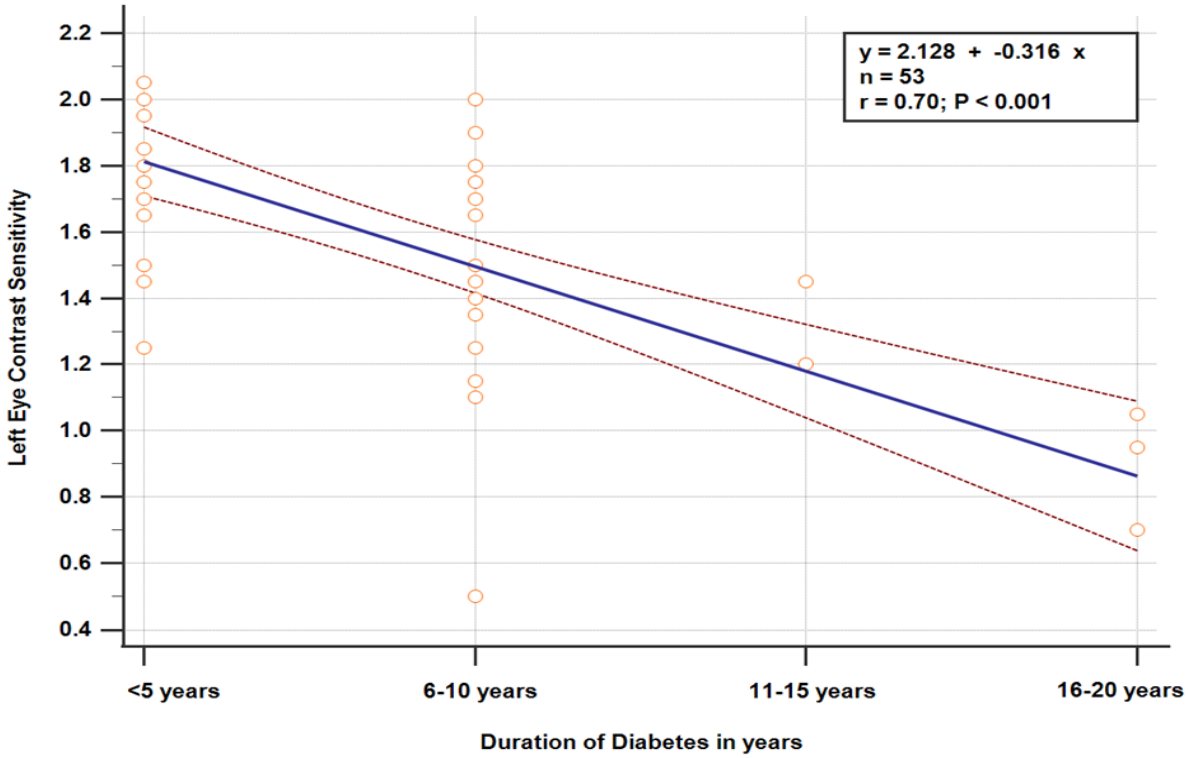
Table 7: Correlation of Contrast Sensitivity and duration of diabetes

Variable	Correlation coefficient (r)	P value
Right Eye Contrast Sensitivity	-0.7097	<0.0001
Left Eye Contrast Sensitivity	-0.6990	<0.0001

Graph14: Correlation of Right eye contrast sensitivity with duration of diabetes



Graph15: Correlation of Left eye contrast sensi tivity with duration of diabetes



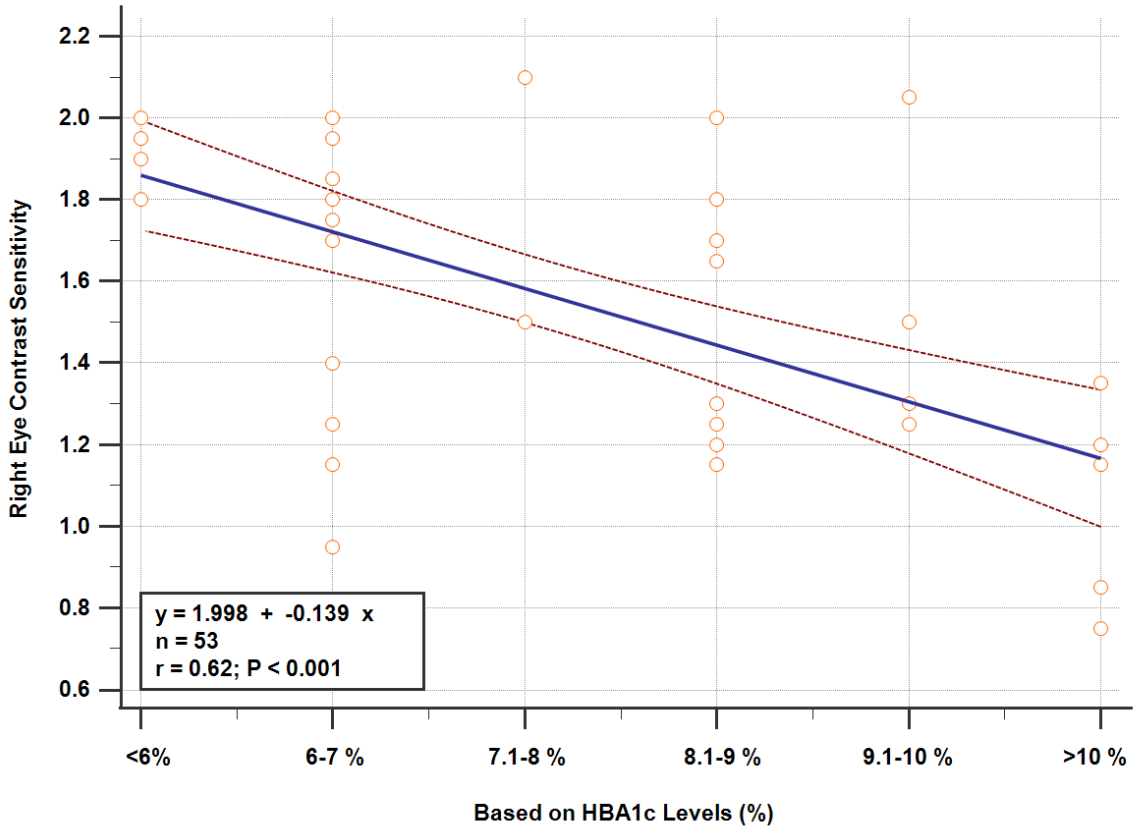
Correlation of contrast sensitivity with HBA1c:

The correlation analysis between contrast sensitivity and HBA1c levels was done and the results were displayed in table 8. The right eye showed negative correlation with HBA1c levels which was statistically significant (r=-0.6216; P<0.0001).Similarly left eye contrast sensitivity also had negative correlation with HBA1c levels (r=-0.7562; P<0.0001) which revealed that as the glycemic control progress to poor control, the contrast sensitivity progress to visual impairment and disability.(graph16&17).

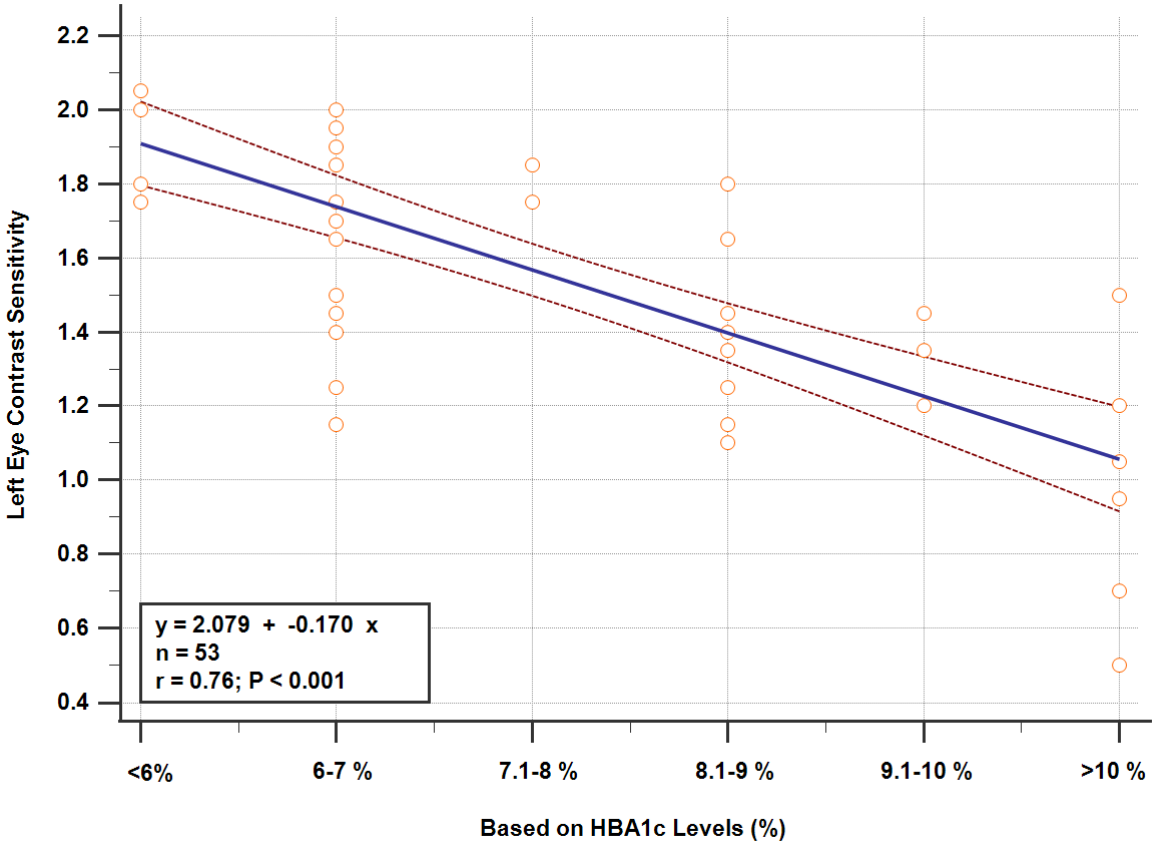
Table 8: Correlation of Contrast Sensitivity and HBA1c Levels

Variable	Correlation coefficient (r)	P value
Right Eye Contrast Sensitivity	-0.6216	<0.0001
Left Eye Contrast Sensitivity	-0.7567	<0.0001

Graph16: Correlation of Right eye contrast sensitivity with HBA1c Levels



Graph17: Correlation of Left eye contrast sensitivity with HBA1c Levels



DISCUSSION

DISCUSSION

The present study provides valuable insights into the relationship between diabetes duration, glycemic control, and visual impairment among the study participants. Our findings align with existing literature, highlighting the significant impact of prolonged diabetes on various health parameters.

The mean age of the participants was 60.16±7.80 years, with a range from 40 to 70 years, indicating a predominantly older population affected by diabetes. This aligns with the general trend observed in diabetes epidemiology, where the prevalence increases with age due to the cumulative exposure to risk factors over time.[195] The male patients were predominant, accounting for 60.37% of the sample, which is consistent with previous studies showing a higher prevalence of diabetes in men compared to women.[196] This gender disparity might be attributed to differences in lifestyle, hormonal factors, and genetic predispositions.[197]

The mean fasting blood glucose was 127.28±40.75 mg/dl, and the postprandial blood glucose was 174.37±59.52 mg/dl. These levels indicate poor glycemic control among the participants, as evidenced by the mean HbA1c value of 7.72±1.85, which is above the recommended target for diabetic patients.[195] Elevated HbA1c levels are indicative of chronic hyperglycemia, which is known to increase the risk of microvascular and macrovascular complications.[198] This is in line with findings from other studies that have reported similar mean HbA1c levels in populations with suboptimal diabetes management.[199 -201]

On average, 7.21±4.37 years elapsed over the course of diabetes. The correlation between the average age of participants and the length of time they had diabetes was found to be statistically significant ($P<0.001$) in our study. The likelihood of problems is higher in the elderly because they have presumably suffered from diabetes for a longer period of time.[202]

Using contrast sensitivity as a measure of visual impairment, there were noticeable variations between the two eyes. In terms of average contrast sensitivity, the right eye measured 1.57±0.37 and the left eye 1.56±0.37. As the duration of diabetes grew, there was a statistically significant decrease in contrast sensitivity in both eyes ($P<0.001$).[203] This finding is supported by previous research that has demonstrated a decline in visual function with prolonged diabetes duration.[204] Diabetic retinopathy and other diabetes-related ocular conditions, such as macular edema, can impair contrast sensitivity, impacting patients' quality of life.[205]

Our study identified a negative correlation between the duration of diabetes and contrast sensitivity in both the right eye ($r=-0.7097$; $P<0.0001$) and the left eye ($r=-0.6990$; $P<0.0001$). This indicates that longer diabetes duration is associated with greater visual impairment. Similarly, the right eye contrast sensitivity had a negative correlation with HbA1c levels ($r=-0.6216$; $P<0.0001$)and the left eye ($r=-0.7562$; $P<0.0001$) also showed statistically significant negative correlation as well.These correlations suggest that poor glycemic control exacerbates visual impairment, leading to increased disability.[206] Previous studies have shown that intensive glycemic control can reduce the incidence and progression of diabetic retinopathy.[207]

A normal level of visual acuity is not necessarily indicative of normal contrast sensitivity. Ten age- and sex-matched control individuals (20 eyes each) and twenty-two diabetic patients (22 eyes total) were tested for contrast sensitivity using fluorescein angiography. Twenty-two eyes of the patients did not have retinopathy, whereas sixteen eyes of the controls had background retinopathy. People with diabetes who did not have retinopathy had noticeably reduced contrast sensitivity ($p = 0.033$). In their view, primary care clinics could benefit from using the test to screen diabetic patients for retinopathy..[206] Similar findings were also observed in an additional investigation. More than two standard deviations below

the norm for age-matched controls were observed in the test scores of fifteen diabetic patients (6/20 with retinopathy and 9/22 without).[207]

Comparing diabetics with and without retinopathy, as well as examining the correlation between metabolic management and retinal severity and existence, Verotti A et al. performed a series of contrast sensitivity tests. Contrast sensitivity was lower in all groups, including those without retinopathy, compared to controls. Contrast perception and glycemic management were also positively correlated.[208] Contrast sensitivity was shown to be reduced in insulin resistant obese individuals and retinopathic diabetic patients in an evaluation by Dosso AA et al. It appears that early neurosensory impairment can happen even in the absence of retinal abnormalities.[209]

Contrast sensitivity was also found to be significantly associated with uncontrolled diabetes in our study ($P< 0.001$). There was a statistically significant correlation (P value less than 0.05) between the state of diabetes (managed or uncontrolled) and contrast sensitivity, as demonstrated in the current investigation and in the work by Vaibhavee and Manisha.[210] The results showed that while blood glucose levels fluctuated, contrast sensitivity function was impaired similar to the study done by Virotti et Al[208]

The observed relationships between glycemic control, duration of diabetes, and visual impairment underscore the importance of stringent diabetes management. Effective glycemic control can potentially mitigate the progression of diabetic retinopathy and other visual impairments.[211] These findings emphasize the need for regular monitoring and appropriate interventions to manage blood glucose levels effectively and reduce the risk of complications. Regular ophthalmic examinations and early detection of visual impairment are crucial for preventing severe visual disability in diabetic patients.[212]

CONCLUSION

CONCLUSION

Our study highlights the critical interplay between diabetes duration, glycemic control, and visual impairment.As the duration of diabetes increased,the contrast sensitivity decreased on both eyes significantly ($P<0.0001$). Both eyes contrast sensitivity had negative correlation with HBA1c levels ($P<0.0001$)which revealed that as the glycemic control progressed to poor control, the contrast sensitivity progressed to visual impairment. The significant correlations found in this study should encourage healthcare providers to focus on comprehensive diabetes management strategies to prevent or delay the onset of visual disabilities in diabetic patients. Future research should explore the potential benefits of emerging treatments and technologies in improving glycemic control and preventing diabetes-related complications.

SUMMARY

SUMMARY

The present study enrolled 53 patients and their mean age was 60.16±7.80 years and ranged from 40 to 70 years.The male patients were predominant (56.6%) and 43.4% of female patients.The mean fasting blood glucose was 127.28±40.75 mg/dl and the post prandial blood glucose was 174.37±59.52 mg/dl. The three months glycemic control marker HbA1c was assessed and the mean was 7.72±1.85

On average, 7.21±4.37 years elapsed over the course of diabetes.Some visual impairment was revealed by the mean contrast sensitivity for the right eye, which was 1.57±0.37. The left eye likewise displayed the same value (1.56±0.37).

There was a statistically significant relationship ($P<0.001$) between the duration of diabetes and the average age of the study participants.The longer a person has had diabetes, the higher their fasting glucose level will be. The statistical significance of this difference was high ($P<0.001$). Likewise, there was a statistically significant increase ($P<0.001$) in post-prandial blood glucose levels as the duration of diabetes continued. As diabetes lasted longer, glycemic control became worse. $P<0.001$ indicates that the difference is statistically significant.

Both the the right eye's contrast sensitivity ($r=0.7097$; $P<0.0001$) and left eye's contrast sensitivity ($r=-0.6990$; $P<0.0001$) declined as the duration of diabetes increased, according to the present study's negative correlation

The right eye contrast sensitivity showed negative correlation with HBA1c levels which was statistically significant ($r=-0.6216$; $P<0.0001$).Similarly left eye contrast sensitivity also had negative correlation with HBA1c levels ($r=-0.7562$; $P<0.0001$).This revealed that as the glycemic control progressed to poor control, the contrast sensitivity progressed to visual impairment and disability.

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ANNEXURES

ANNEXURE 1

CASE PROFORMA	
Name:	Case No:
Age:	Date:
Sex:	1P No:
Occupation:	DOE:
Address:	

Chief complaints:

Past history:

Duration of DM / HTN / BA / Epilepsy

Family history:

Personal history:

Appetite – Sleep – Bowel –

Diet – Habits – Bladder –

GPE:

Pallor / Edema /Icterus / Cyanosis / Clubbing / Lymphadenopathy

Vital signs:

a. Pulse – c) RR –

b. BP – d) Temp –

Systemic examination:

a. CVS – c. RS –

b. PA – d. CNS –

OCULAR EXAMINATION		
	OD	**OS**
1. Head posture **2. Ocular posture** **3. Facial symmetry**		
4. Ocular movements		
5. Visual acuity: Distant Near Refraction		
6. Anterior Segment		
7. Fundus (IDO & Slit Lamp +90D)		
8. Contrast Sensitivity		
9. Laboratory Report a. FBS b. PPBS c. HbA1c		

ANNEXURE 2

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case no:

IP no:

TITLE: RELATIONSHIP BETWEEN CONTRAST SENSITIVITY AND METABOLIC CONTROL IN DIABETICS WITHOUT RETINOPATHY

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of personal information as outlined in this consent form.

I understand the purpose of this study, the risks and benefits of the technique and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw the participation from this study at any time and this will not change the future care.

Participation in this study does not involve any extra cost to me.

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ, ಟಮಕ, ಕೋಲಾರ - 563101

ತಿಳಿವಳಿ ಕೆಸಮ್ಮತಿ ನಮೂನೆ

ಶೀರ್ಷಿಕೆ : "ರೆಟಿನೋಪತಿ ಇಲ್ಲದೆ ಮಧುಮೇಹಿಗಳಲ್ಲಿ ಕಾಂಟ್ರಾಸ್ಟ್ ಸೆನ್ಸಿಟಿವಿಟಿ ಮತ್ತು ಮೆಟಬಾಲಿಕ್ ನಿಯಂತ್ರಣದ ನಡುವಿನ ಸಂಬಂಧ "

ಈ ಸಂಶೋಧನೆಗೆ ರೋಗಿಯ ಗುರುತಿನ ಸಂಖ್ಯೆ:

ಐಪಿ ಸಂಖ್ಯೆ:

ಅಂಗೀಕರಿಸಿದ ನಾನು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ಈ ಸಮ್ಮತಿಯ ರೂಪದಲ್ಲಿ ವಿವರಿಸಿರುವಂತೆ ನನ್ನ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ದೃಢೀಕರಿಸುತ್ತೇನೆ.

ನಾನು ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ತಂತ್ರಗಳ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರಯೋಜನಗಳನ್ನು ಮತ್ತು ಅಧ್ಯಯನದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವ ಮಾಹಿತಿಯ ಗೌಪ್ಯತೆಗೆ ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳನ್ನು ಕುರಿತು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಮತ್ತು ನನ್ನ ತೃಪ್ತಿಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರ ನೀಡಲಾಗಿದೆ.

ಈ ಸಂಶೋಧನೆಯಿಂದ ಹೊರಬರುವ ಮಾಹಿತಿಯನ್ನು ವೈದ್ಯರು ಯಾವುದೇ ಜರ್ನಲ್ನಲ್ಲಿ ಅಥವಾ ಕಾನ್ಫೆರೆನ್ಸ್ನಲ್ಲಿ ಪ್ರಕಟಿಸಲು ಅನುಮತಿ ಸೂಚಿಸಿರುತ್ತೇನೆ

ನಾನು ಈ ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ಮುಕ್ತವಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಮುಂದಿನ ಕಾಳಜಿಯನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯ ಭಾಗವಹಿಸುವಿಕೆ ನನಗೆ ಯಾವುದೇ ಹಣಕಾಸಿನ ಹೊರೆ ಒಳಗೊಂಡಿರುವುದಿಲ್ಲ.

ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿಯ:			
ಸಾಕ್ಷಿ 1:			
ಸಾಕ್ಷಿ 2:			
ಪ್ರಾಥಮಿಕ ತನಿಖೆದಾರ / ಡಾಕ್ಟರ್:			

ANNEXURE 3

PATIENT INFORMATION SHEET

This information is to help you understand the purpose of the study "**RELATIONSHIP BETWEEN CONTRAST SENSITIVITY AND METABOLIC CONTROL IN DIABETICS WITHOUT RETINOPATHY"**You are invited to take part voluntarily in this research study, it is important that you read and understand purpose, procedure, benefits and discomforts of the study.To find the relationship between visual outcome, contrast sensitivity and blood sugar levels in patients with Diabetes mellitus.There are no risks associated with the various investigations to be done which includes detailed examination of the eye and blood sugar levels.Participation in this research study may not change the final outcome of your eye condition. However, patients in the future may benefit as a result of knowledge gained from this study.You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary.You may refuse to take part in the study or you may stop your participation in the study at any time, without any penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY:

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available Your original records may be reviewed by your doctor ethics review board For further information./clarification please contact the below mentioned doctor. SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA KOLAR 563101 to

Dr .DEVI SINDHUJA.S or DR.B.O.HANUMANTHAPPA Contact no: 9500414877 or 9448322889.

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ, ಟಮಕ, ಕೋಲಾರ - 563101

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಶೀರ್ಷಿಕೆ: " ರೆಟಿನೋಪತಿ ಇಲ್ಲದೆ ಮಧುಮೇಹಿಗಳಲ್ಲಿ ಕಾಂಟ್ರಾಸ್ಟ್ ಸೆನ್ಸಿಟಿವಿಟಿ ಮತ್ತು ಮೆಟಬಾಲಿಕ್ ನಿಯಂತ್ರಣದ ನಡುವಿನ ಸಂಬಂಧ "

ಈ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳಲು ನಿಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಭಾಗವಹಿಸಲು ನಿಮ್ಮನ್ನು ಆಹ್ವಾನಿಸಲಾಗಿದೆ. ನೀವು ಹೇಳಿದ ಮತ್ತು ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳುವುದು ಬಹಳ ಮುಖ್ಯ.

ಕಾಂಟ್ರಾಸ್ಟ್ ಸೆನ್ಸಿಟಿವಿಟಿಯನ್ನು ಅಳೆಯುವುದು ದೃಷ್ಟಿಯಷ್ಟೇ ಮುಖ್ಯವಾಗಿದೆ. ಮತ್ತು ದೃಷ್ಟಿಯ ಗುಣಮಟ್ಟವನ್ನು ಪ್ರತಿಬಿಂಬಿಸುವುದರಿಂದ ಮತ್ತು ಅನೇಕ ಸಂದರ್ಭಗಳಲ್ಲಿ ಮುಂಚೆಯೇ ಕ್ಷೀಣಿಸುತ್ತದೆ, ಆದರೆ ದೃಷ್ಟಿ ಸಾಮಾನ್ಯವಾಗಿರುತ್ತದೆ. ರೋಗಿಗಳಲ್ಲಿ ದೃಶ್ಯ ಫಲಿತಾಂಶ, ಕಾಂಟ್ರಾಸ್ಟ್ ಸೆನ್ಸಿಟಿವಿಟಿ ಮತ್ತು ರಕ್ತದ ಸಕ್ಕರೆಯ ಮಟ್ಟಗಳ ನಡುವಿನ ಸಂಬಂಧವನ್ನು ಕಂಡುಹಿಡಿಯಲು.ಅಂತಹ ತೊಡಕುಗಳನ್ನು ಅವನು ಗುರುತಿಸುವುದು ಅಥವಾ ಅಭಿವೃದ್ಧಿ ಹೊಂದುವ ಅಪಾಯವು ಅದರ ಸಂಭವವನ್ನು ಕಡಿಮೆ ಮಾಡಲು ಬೇಕಾದ ಬದಲಾವಣೆಗಳ ನಿರ್ಣಯದಲ್ಲಿ ಮಹತ್ವದ್ದಾಗಿರುತ್ತದೆ, ಹೀಗಾಗಿ ತೀವ್ರವಾದ ಆಕ್ಯುಲರ್ ಆವಿಷ್ಕಾರದ ಹೊರೆಯನ್ನು ಕಡಿಮೆ ಮಾಡುತ್ತದೆ ನಮ್ಮ ವೀಕ್ಷಣೆ ಸಹ ಪ್ರಾಮುಖ್ಯತೆಯನ್ನು ಹೊಂದಿರಬಹುದು

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನೀವು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದಕ್ಕಿಂತ ಮೊದಲು ನೀವುಯಾವುದೇಅರ್ಹತೆಗೆಯಾವುದೇದಂಡವಿಲ್ಲದೆಅಥವಾನಷ್ಟವಿಲ್ಲದೆಯೇಯಾವುದೇಸಮಯದಲ್ಲಿಯಾವುದೇಸಮಯದಲ್ಲಿಭಾಗವಹಿಸುವಿಕೆಯನ್ನುನಿಲ್ಲಿಸಬಹುದು. ಈ ಸಂಶೋಧನೆಯಿಂದ ಹೊರಬರುವ ಮಾಹಿತಿಯನ್ನು ವೈದ್ಯರು ಯಾವುದೇ ಕಾನ್ಫರೆನ್ಸ್ನಲ್ಲಿ ಪ್ರಕಟಿಸಲು ಅನುಮತಿ ಸೂಚಿಸಿರುತ್ತೇನೆ

ಗೌಪ್ಯತೆ

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನದ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡಲಾಗುವುದು ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಿರುವುದಿಲ್ಲ. ನಿಮ್ಮ ಮೂಲದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ನೈತಿಕ ವಿಮರ್ಶೆ ಮಂಡಳಿ ಪರಿಶೀಲಿಸಬಹುದು.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ

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ANNEXURE-IV

Photography 1:Anterior segment evaluation using Slit lamp examination

Photography2:Fundoscopy using Indirect Ophthalmoscope

Photography3:Right eye contrast sensitivity assessment using Pelli Robson Chart

Photography4:Left eye contrast sensitivity assessment using Pelli Robson Chart

KEY TO MASTER CHART

RE CS -right eye contrast sensitivity

LE CS -Left eye contast sensitivity

FBS -Fasting blood sugar

PPBS -Post prandial blood sugar

HbA1C -Glycated Haemoglobin

MASTERCHART

UHID	AGE	SEX	RE CS	LE CS	FBS	PPBS	HBA1(	DURATION
214373	65	F	0.95	1.4	138	247	7	6
181816	68	M	0.85	1.05	168	211	12.1	20
223176	70	M	1.95	2	77	184	6.2	2
208846	56	F	2	1.85	104	158	6.5	4
217658	50	M	1.25	1.1	154	182	8.7	6
409512	49	F	2	1.8	166	203	5.9	5
213789	65	F	1.15	1.2	148	200	11.5	15
135767	69	M	1.15	1.05	292	337	10.5	13
343886	64	F	1.65	1.1	186	290	9	10
362204	51	F	2.1	1.85	153	264	8	5
252974	65	M	1.15	0.7	176	294	12	20
347597	62	F	1.2	0.5	196	258	10.2	9
257923	70	M	1.2	1.45	120	156	8.2	8
230367	56	M	1.75	2	98	136	6.2	3
219065	51	F	1.75	1.5	99	113	6.8	5
217758	53	M	2.05	1.35	105	126	9.2	8
230367	51	M	1.95	2.05	102	118	5.9	4
135671	65	F	1.15	1.8	191	254	8.4	9
199772	67	M	1.85	1.7	92	175	6.6	5
409503	46	F	1.9	2	98	134	5.8	2
338977	69	M	0.95	1.7	103	164	6.2	8
125291	61	F	2	1.75	99	124	5.9	4
231752	56	M	1.3	1.25	140	254	8.48	7
81994	40	M	2.1	2	96	118	5.6	2
135798	65	F	1.15	1.65	106	145	6.4	5
227760	61	M	1.2	1.25	152	215	9	7
149591	65	F	1.35	1.5	176	198	10.5	9
311704	59	M	1.8	2	96	122	5.9	4
352480	70	F	1.25	1.15	118	247	7	8
223565	67	M	0.75	0.95	213	312	11	18
249762	58	M	1.3	1.45	170	245	9.4	11
144158	61	M	1.95	2	90	164	6.7	6
160930	49	F	2	2.05	87	125	5.8	4
431090	55	F	1.7	1.65	96	136	6.1	6
307744	70	M	1.25	0.7	151	196	10	15
431093	55	M	1.4	1.25	102	110	6.7	5
409458	69	F	1.3	1.15	128	144	8.9	11
135797	60	F	1.7	1.45	98	138	6.5	6
154565	49	M	2	1.75	107	147	6	2
347611	71	F	1.8	1.9	99	140	6.2	8
351802	61	F	1.95	2	89	109	5.6	3
351803	66	M	1.5	1.45	164	185	10	9
135749	54	M	1.75	1.45	96	103	6.8	4
370066	70	M	1.25	1.35	126	152	8.9	8
143577	52	M	1.75	2	100	124	6.2	5
351809	64	M	1.85	2	98	109	6.1	3
404953	66	F	1.8	1.5	134	146	8.5	6
144874	45	F	1.95	1.8	99	110	5.7	3
336350	68	M	2	1.4	103	143	8.2	7
351806	57	M	1.85	1.95	100	120	6.4	4
167357	58	F	1.45	1.75	114	137	8	10
403985	65	M	1.5	1.6	123	158	8.3	8
310482	60	M	1.8	1.5	118	148	7	3