

A Dissertation on
**CORRELATION OF OCULAR PERFUSION PRESSURE AND
INTRAOCULAR PRESSURE CHANGES DURING
HEMODIALYSIS IN END-STAGE RENAL DISEASE – AN
OBSERVATIONAL STUDY**

Submitted to the
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

*In partial fulfillment of the Regulations
for the Award of the Degree of*
**M.S. (BRANCH - III)
OPHTHALMOLOGY**



**GOVT. STANLEY MEDICAL COLLEGE AND HOSPITAL
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600 001.**

APRIL 2017

CERTIFICATE

This is to certify that the study entitled “**CORRELATION OF OCULAR PERFUSION PRESSURE AND INTRAOCULAR PRESSURE CHANGES DURING HEMODIALYSIS IN END-STAGE RENAL DISEASE – AN OBSERVATIONAL STUDY**” is the result of original work carried out by **DR. NITHYA .G** , under my supervision and guidance at **STANLEY MEDICAL COLLEGE, CHENNAI**. The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in ophthalmology**, a course from 2014 to 2017 at the Stanley medical college, Chennai.

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DECLARATION

I hereby declare that this dissertation entitled “**CORRELATION OF OCULAR PERFUSION PRESSURE AND INTRAOCULAR PRESSURE CHANGES DURING HEMODIALYSIS IN END-STAGE RENAL DISEASE – AN OBSERVATIONAL STUDY**” is a bonafide and genuine research work carried out by me under the guidance of **Prof.Dr.K.BASKER, M.S.,D.O.**, HOD, Department of ophthalmology, Government Stanley medical college and hospital, Chennai-600001.

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INSTITUTIONAL ETHICAL COMMITTEE,
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Title of the Work : Correlation of ocular perfusion pressure and intracular pressure during hemodialysis in end-stage renal disease.

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.01.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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PART- I

INTRODUCTION

Chronic kidney disease (CKD) is a gradual progressive irreversible loss of renal function over a period of months or years. About 10% of the global population may be affected by CKD. The rise in chronic kidney disease patients is primarily due to ageing population, diabetes, hypertension, glomerulonephropathy.

CKD and its treatment Hemodialysis (HD) may lead to ocular complaints or exacerbation of underlying ocular disease like elevated Intraocular pressure (IOP), glaucoma, macular edema, age-related macular degeneration, ischemic optic neuropathy.

Glaucoma is a chronic progressive optic neuropathy caused by a group of ocular conditions which lead to damage of optic nerve with visual field defects. Increase in intraocular pressure which is the most common risk factor and an only treatable parameter of glaucoma.

During hemodialysis changes in haemoconcentration, plasma colloid-osmotic pressure, plasma osmolarity during HD could be registered. It is important to reveal their influence on ocular perfusion pressure and intraocular pressure.

Elevated intraocular pressure and *decreased ocular perfusion pressure* are the known risk factors for glaucoma development and progression. Unrecognised significant intraocular pressure elevation or ocular perfusion pressure reduction during hemodialysis could lead to glaucomatous optic nerve damage and subsequent irreversible visual loss.

- Hemodialysis has been known to induce changes in the eye by an increase in IOP, when serum osmolality decreases during hemodialysis, the resulting imbalance in osmolality between the ocular chamber and the blood causes an influx of volume into the posterior chamber via the ciliary body.
- Adequate oxygenation of ocular tissues depends on maintenance of ocular perfusion pressure through systemic regulation of blood pressure and local regulation of IOP, It has been proposed that vascular dysregulation leads to abnormal ocular perfusion and thus optic nerve ischemia, serving as an underlying cause of glaucomatous damage

HD potentially produces only a transient change in OPP. Low OPP is a risk factor for glaucoma development and progression when the abnormal ocular perfusion pressure is under issue, examining IOP and OPP changes occurring during individual HD sessions has been done and further investigated whether OPP levels reached thresholds associated with optic nerve damage.

NEED FOR STUDY

Chronic kidney disease patients on Hemodialysis are found to have a transient increase in intraocular pressure and decrease in ocular perfusion pressure during hemodialysis. Hence it necessitates to monitor IOP & OPP for these patients as a routine during hemodialysis session and also during a routine examination of CKD patients who are on hemodialysis. This may help in early detection of elevation and fluctuation of IOP & OPP and also in early detection of glaucomatous optic nerve damage and subsequent irreversible visual loss. It also helps in early intervention in order to prevent the progression of the disease.

This study was done among patients attending hemodialysis session in Nephrology Department, Govt. Stanley medical college & hospital, Chennai.

REVIEW OF LITERATURE

Dr. Drance (1973) provided for the first time the definition of glaucoma as a disease of the optic nerve (an optic neuropathy) caused by numerous factors, called risk factors³⁵.

In 1826, **William Bowman** was recommending digital palpation as part of the routine examination of the eye.

Von Graefe developed the first instrument for measuring intraocular pressure in 1865.

The first applanation tonometer was introduced in 1867 by **Adolph Weber** but was not generally accepted.

Imbert (1885) and Fick (1888) developed the principle on which modern applanation tonometers are based.

Maklakoff applanation tonometer was the first reasonably accurate instrument for measuring the intraocular pressure of the late 19th century until relatively recently³⁴.

Schiötz developed an indentation tonometer that was widely used throughout the world during the first two-thirds of the 20th century³⁴.

Goldmann's applanation tonometer of 1950 began the era of truly accurate intraocular pressure measurement.

Sitprija et al observed a significant increase in intra-ocular pressure in uraemic dogs, during hemodialysis.

Watson & Greenwood' found a rise in intraocular pressure following hemodialysis in human beings.

Los Angeles Latino Eye Study (LALES), a population-based study of over 6,000 adult Latinos, which recently reported a correlation between both high and low systemic blood pressures and the prevalence of OAG.

The **Baltimore Eye Survey** found that those with DOPP <30 mmHg had a six-times higher risk of disease development than those with DOPP >50 mmHg.

The **Barbados Eye Study** showed that individuals with the lowest 20% of DOPP were 3.3-times more likely to develop glaucoma. In a subgroup of patients from the Barbados Eye Study followed for 9 years, lower OPPs and lower systolic BPs were again identified as risk factors.

AQUEOUS HUMOR INFLOW AND OUTFLOW –ANATOMY

Aqueous inflow anatomy (i.e. site of aqueous formation) - Ciliary body

Aqueous outflow pathway (i.e. drainage) anatomy located at the angle of anterior chamber

AQUEOUS INFLOW ANATOMY:

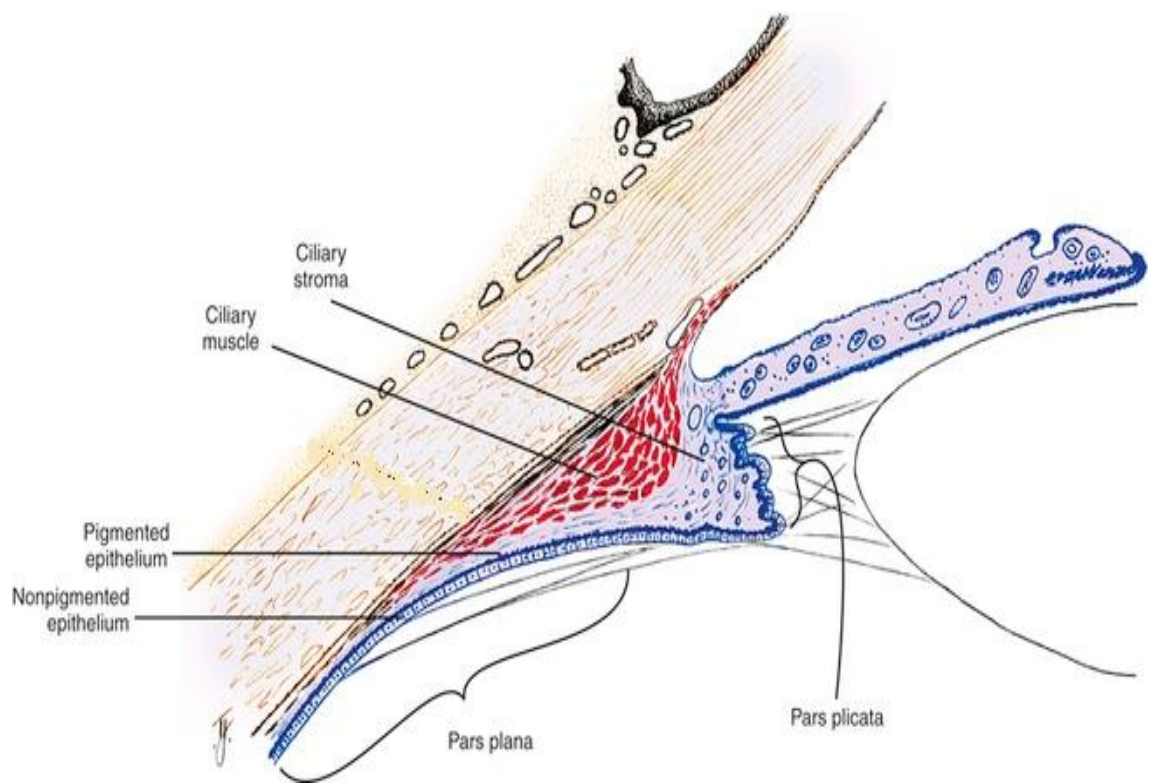
CILIARY BODY AND THE CILIARY PROCESSES:

The ciliary body is a part of the uveal tract. It is a triangular structure comprising of

1. Ciliary body muscle
2. Vessels
3. Epithelia lining the ciliary processes
4. Nerve terminals from the autonomic nervous system

1. Ciliary body muscle: It is a non-striated muscle. It consists of two main portions: Longitudinal fibres & Circular fibres

2. Ciliary processes: The functional unit responsible for aqueous humor secretion is the ciliary process. It is a whitish finger-like projection from the pars plicata part of the ciliary body



AQUEOUS INFLOW ANATOMY

AQUEOUS OUTFLOW PATHWAY – ANATOMY

Most of the aqueous drainage is through the anterior chamber angle.

Anatomy:

Aqueous outflow system includes:

1. Trabecular meshwork
 - i. Uveal meshwork
 - ii. Juxtacanalicular (endothelial) meshwork
 - iii. Corneoscleral meshwork
2. Schlemm's canal
3. Collector channel
4. Episcleral veins

1. Trabecular meshwork:

The trabecular meshwork is a sieve-like structure bridging scleral sulcus and converts it into a tube - Schlemm's canal . It consists of endogenous glycosaminoglycan – hyaluronic acid, chondroitin, heparin, and dermatan and keratin sulphates². The glycoproteins and GAG composition of the trabecular meshwork are not constant throughout life. Changes happen in composition as age advances, which have been implicated in the pathogenesis of primary open angle glaucoma. It consists of 3 portions

- a) Uveal meshwork
- b) Corneoscleral meshwork
- c) Juxtacanalicular meshwork

2. Schlemm's canal:

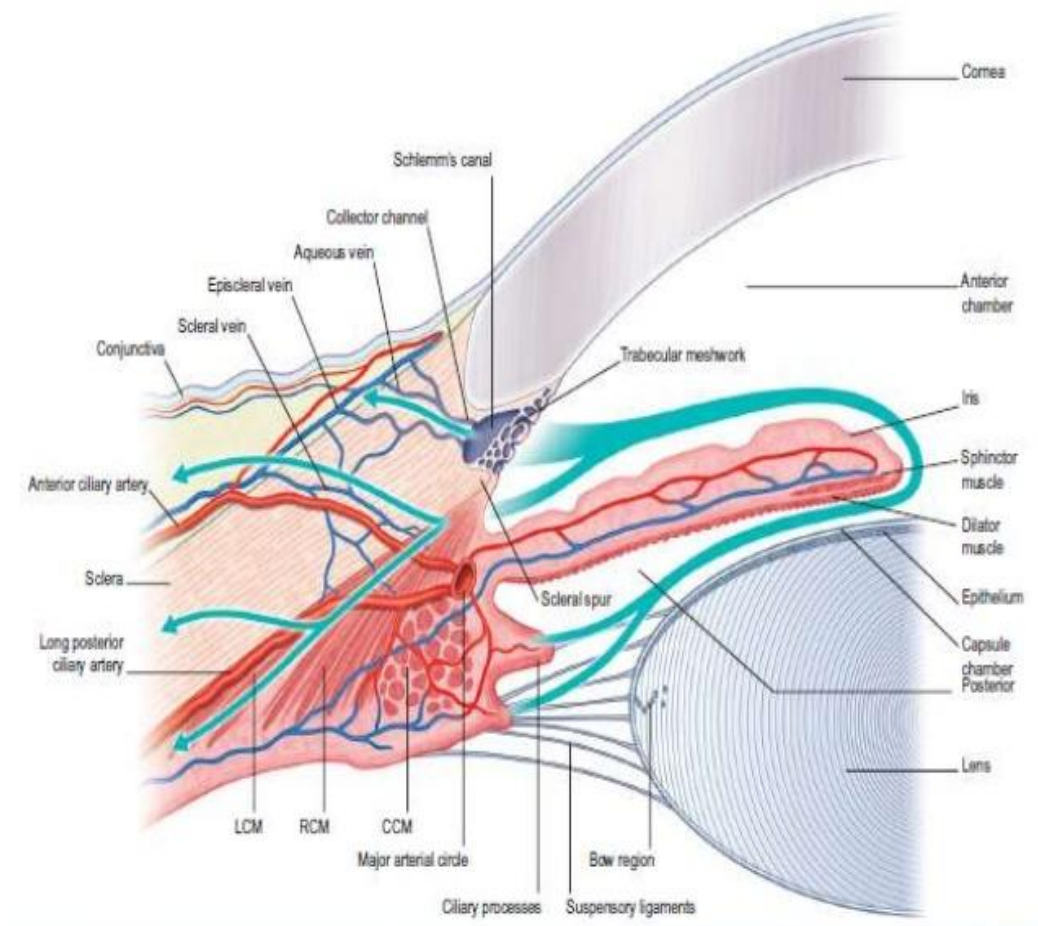
It is an oval channel embedded in the scleral sulcus. Its outer wall is lined by endothelial cells, which contains numerous openings of the collector channels. Torus or lip like thickening and septa are present to keep the canal open.

3. Collector channels:

The outer wall of schlemm's canal has 25 to 35 opening for the collector channels. They connect the schlemm's canal to the episcleral and conjunctival veins of the limbal region.

4. Episcleral veins:

Most of the aqueous vessels drain into the episcleral veins, which ultimately drain into cavernous sinus via the anterior ciliary and superior ophthalmic veins.



AQUEOUS HUMOR INFLOW AND OUTFLOW –ANATOMY

AQUEOUS HUMOR

Aqueous humor is an intraocular fluid derived from the plasma within the capillary network of ciliary processes. The circulating aqueous humor enters the posterior chamber and flows around the lens and through the pupil into the anterior chamber. Within the anterior chamber, a temperature gradient (cooler toward the cornea) creates a convection flow pattern.

Formation of aqueous humor:

The constituents of the aqueous humor traverse the three tissue layers of ciliary processes; the capillary wall, the stroma, and the two layers of the epithelium. They undergo the following process as they pass through these layers.

1. Diffusion :

It is a biophysical process. **Lipid -soluble substances** are transported through the lipid portions of the membrane proportional to a **concentration gradient** across the membrane (i.e.) Molecules of gas or solution distribute themselves uniformly throughout the space they are contained, by the motion of its particles.

2. **Ultrafiltration**

It is the transport of **water and water soluble substance** across the cell membrane through the protein micropores along the **osmotic gradient and hydrostatic pressure**

3. **Active Secretion:**

It is responsible for 90% of total aqueous production active transport of charged substances against the concentration gradient. This mechanism is believed to be mediated by globular proteins in the membrane. Energy is consumed in this process. The energy is derived by hydrolysis of adenosine triphosphate (ATP)

Composition of aqueous humor

- 99.9% Water
- Proteins concentration is 5-16mg/100ml
- Glucose – 75% of the plasma concentration.
- Electrolytes:
 - Sodium ion is similar in plasma and aqueous.
 - Bicarbonate ion Concentration is increased in the posterior chamber.
 - chloride ion concentration is increased in aqueous than plasma and phosphate concentration is decreased than plasma.
- Ascorbic acid concentration is very high in aqueous than plasma.

INTRAOCULAR PRESSURE

Intraocular pressure (IOP) is determined by the balance between the rate of aqueous production and its outflow. In individuals who are susceptible to glaucoma, “normal” intraocular pressure (IOP) may be defined as that pressure which does not lead to glaucomatous damage of the optic nerve head. The average IOP in the general population is around a range of about 11-21mmHg- two standard deviations either side of average 16 mmHg.

FACTORS INFLUENCING INTRAOCULAR PRESSURE:

1. Heredity:

Relatives of patients with POAG, tend to have increased IOP.

2. Age:

- Increase in IOP with age.
- Related to reduced facility of aqueous outflow and uveoscleral outflow because aqueous production also decreases with increasing age.

3. Gender:

- IOP is equal between the sexes in the age group 20-40 years.

- In older age- apparent increase in mean IOP with age is higher in women and coincides with the onset of menopause.

4. Ethnicity:

- In the United States, blacks have higher IOPs than whites¹. Recent studies using regression analysis of multiple covariates found that black race is not an independent risk factor
- Full blood Indians in New Mexican tribe – found to have slight lower mean IOP

5. Refractive error:

- Myopes tend to have slight higher IOP

6. Arterial blood pressure:

IOP is generally not affected by physiological changes in arterial blood pressure; however, sudden large swings may affect the IOP accordingly.

7. Systemic venous pressure (SVP):

Changes in SVP can cause a profound effect on IOP by affecting ipsilateral venous pressure, for about 1 mmHg rise in episcleral venous pressure raises the IOP by 0.8 mmHg.

8. Plasma osmolarity:

It affects the IOP profoundly. When the concentration of solutes in plasma is lower than the ocular fluids, the water will enter the eye from plasma and raise the IOP. This forms the basis of water drinking test used as a provocative test for glaucoma.

When the total concentration of the solute molecule in the blood exceeds, the water from the eye (vitreous & aqueous) is withdrawn, lowering the IOP. This effect is used clinically to lower the IOP by use of hyperosmotic agents like mannitol.

9. Blood pH:

Systemic acidosis lowers the IOP. Metabolic acidosis induced by carbonic anhydrase inhibition that is responsible for their pressure lowering effect.

10. Seasonal variation:

IOP has been recorded as highest during winter and lowest during summer.

11. Diurnal variation in IOP:

The IOP is subject to cyclic fluctuations throughout the day. The IOP was highest during the early morning and lowest in the late evening. A diurnal variation in IOP of more than 8mmHg is considered pathognomic. The physiologic mechanisms that regulate diurnal IOP variation are complex. The IOP is regulated in part by adrenocortical steroids and catecholamines.

12. Postural Variation:

Postural variation in IOP showed the consistent elevation of IOP at night time. It is physiologically relevant because sleep occurs in the supine position.

TONOMETRY

Measurement of IOP is known as tonometry.

CLASSIFICATION OF TONOMETERS:

All clinical tonometers measure the IOP by relating a deformation of the globe to the force responsible for the deformation. The basic types of tonometers differ according to the shape of the deformation.

1. Indentation.
2. Applanation (flattening).
3. Rebound tonometry

Schiotz tonometer:

Schiotz developed the first device that quantified intraocular pressure. It's based on the principles of indentation tonometry.

Principle

Indentation tonometry measures amount of deformation of the anterior surface of the cornea by a fixed amount of force. The weight of tonometer on the eye increases the actual IOP (P_o) to a higher level (P_t).

The change in pressure from P_o to P_t is an expression of the resistance of the eye (scleral rigidity) to the displacement of fluid.

IOP with Tonometer in position (P_t) = Actual IOP (P_o) + Scleral Rigidity (E)

Determination of P_o from a scale reading, P_t requires conversion which is done according to Friedenwald conversion tables. Friedenwald generated a formula for the linear relationship between the log function of IOP and the ocular distension.

$$P_t = \log P_o + C \Delta V$$

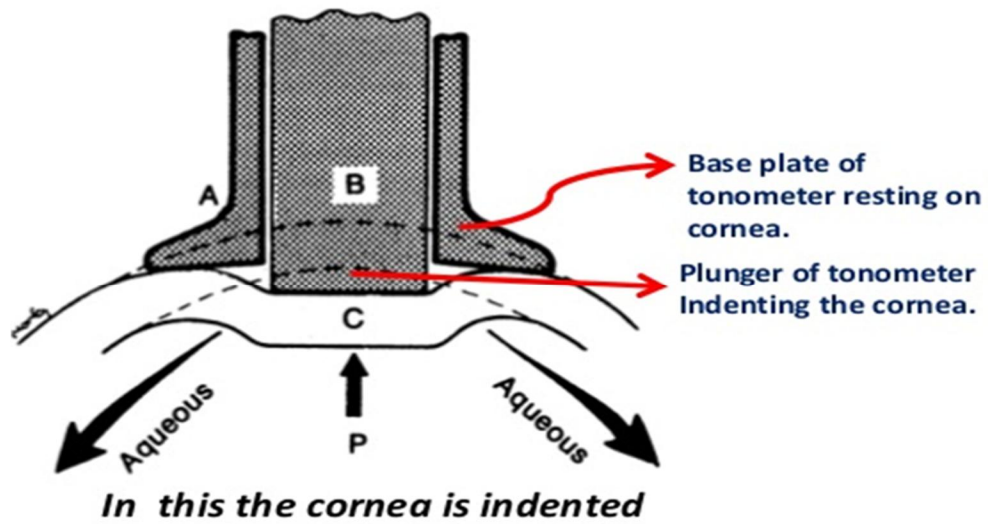
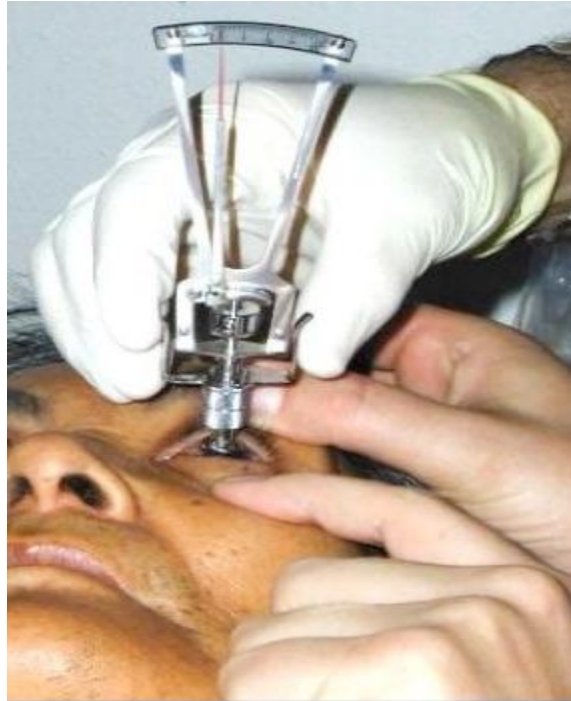
C a numerical constant, coefficient of ocular rigidity= 0.025

ΔV is the change in volume

Technique:

Explain the patient about the procedure and ask him to lie supine, fixing in primary position. Topical anaesthetic drops are instilled in the eyes. The foot plate of tonometer is kept on the cornea and the reading on the scale is read. It is then correlated with the Friedenwald conversion table provided with a tonometer.

SCHIOTZ TONOMETER



Goldmann Applanation Tonometer:

Goldmann applanation tonometer consists of a bi-prism mounted on a standard slit lamp. The prism applanates the cornea in an area of 3.06mm diameter. It is the most accurate tonometer.

Principle:

The principle is based on IMBERT- FICK LAW. The pressure (P) within a sphere is roughly equal to the external force (F) needed to flatten a portion of the sphere divided by the area (A) of the sphere that is flattened.

$$P = F/A$$

Technique:

Local anaesthetic is administered. Fluorescein dye impregnated paper (0.25%) is administered into the lower fornix. The two semicircles are viewed through the slit lamp under cobalt blue light at 60⁰. The fluorescein stained tear meniscus facilitates visualisation at the margin of the contact between cornea and bi-prism. A central blue circle, which is the flattened cornea, surrounded by 2 yellow semi-circles which are the

tear meniscus is seen. Force against the cornea is adjusted until the inner edges overlap by turning measuring dial.

Potential Errors:

Overestimated readings

1. Wide tear meniscus
2. Unsatisfactory vertical alignment of vertical semicircles
3. Thick cornea due to increased collagen content of stroma
4. Increased corneal curvature
5. Against the rule astigmatism

Underestimated readings

1. Measurement without use of fluorescein
2. Inadequate fluorescein concentration
3. Decreased corneal curvature
4. With the rule astigmatism

GOLDMANN APPLANATION TONOMETER

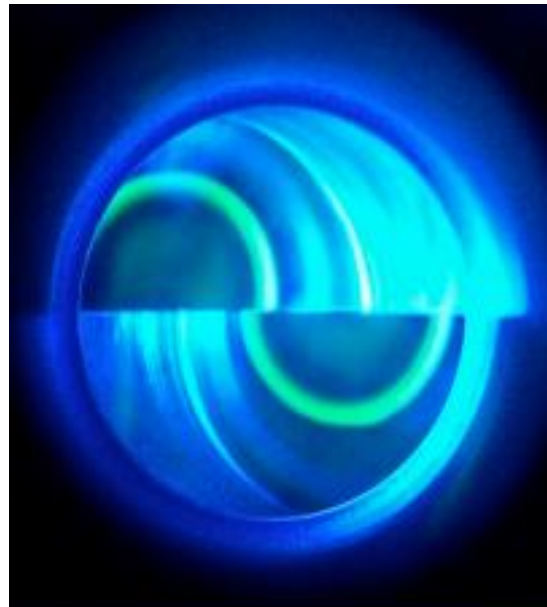
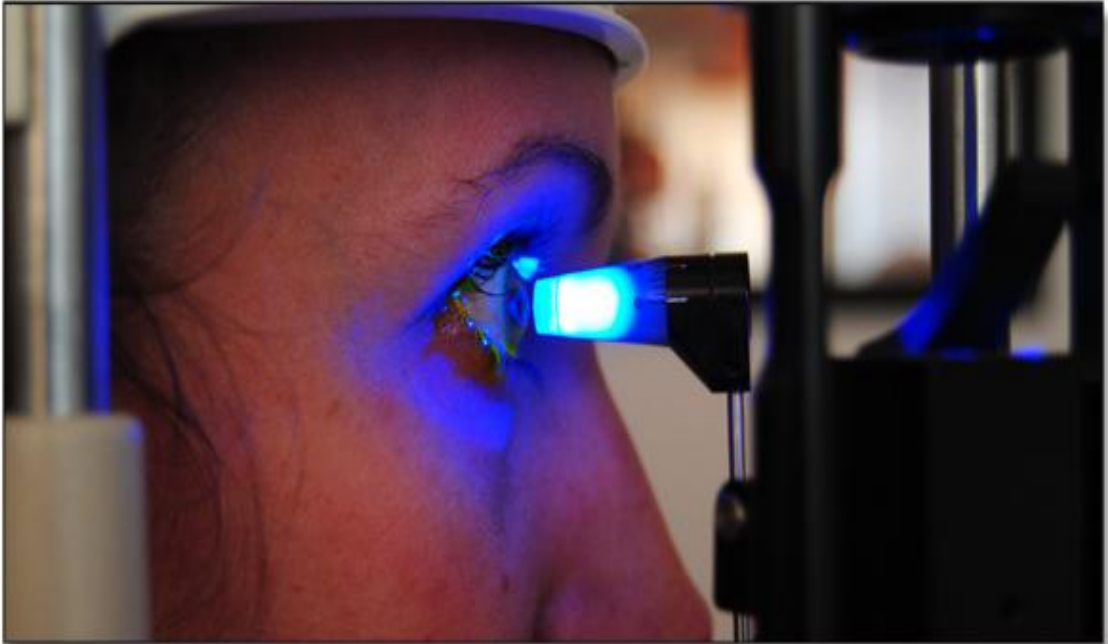
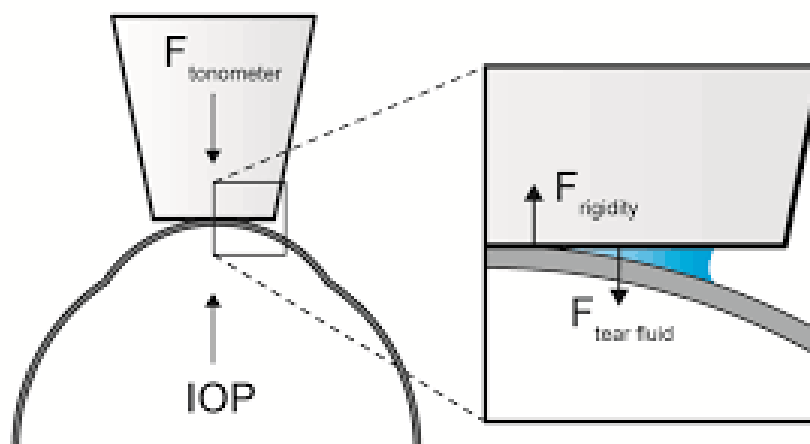
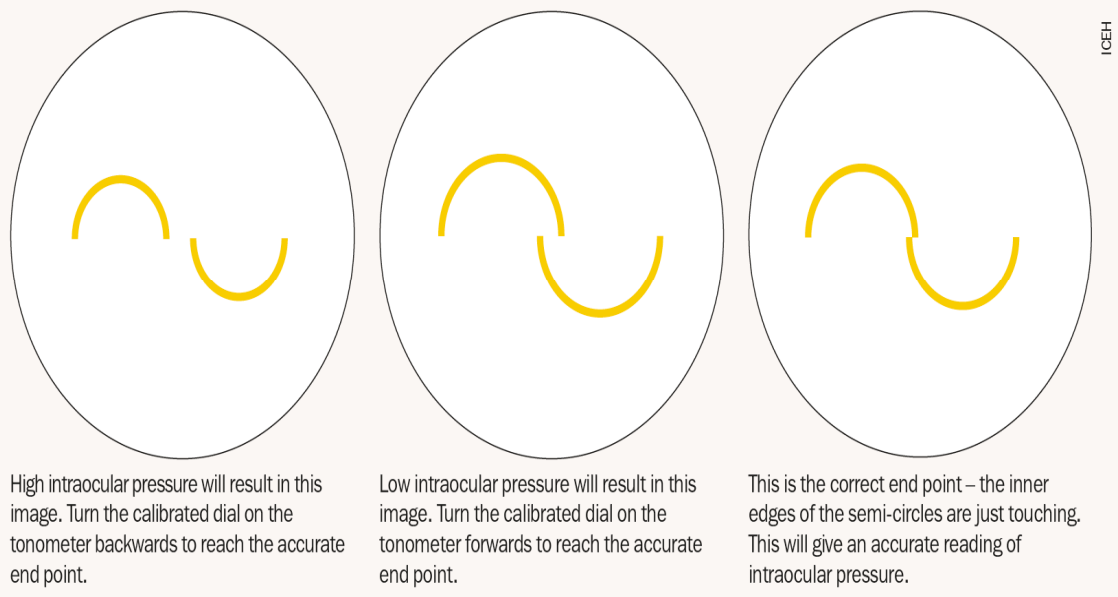


Figure 1. Applanation tonometry semi-circles viewed through the Goldmann prism



Other tonometer types:

1. Perkins applanation tonometer
2. MacKay Marg tonometer
3. Tono-Pen tonometer
4. Pneumatic tonometer
5. Non-contact airpuff tonometer
6. Optical response analyser
7. Dynamic contact tonometer (pascal)
8. Rebound tonometer

OCULAR PERFUSION PRESSURE

Ocular blood flow refers to the distribution of oxygenated blood throughout the ocular vasculature.

Ocular perfusion pressure (OPP) is defined as the difference between arterial blood pressure and intraocular pressure. It refers to the pressure available to drive blood through the intraocular vasculature, and the amount of perfusion being influenced by the resistance to flow, which is a function of the vessel tone or the vessel calibre. This can be further described as systolic perfusion pressure and diastolic perfusion pressure.

The following formulas are used to calculate³⁶

- Mean arterial pressure (MAP)

$$\text{MAP} = \text{diastolic BP} + \frac{1}{3}(\text{systolic BP} - \text{diastolic BP})$$

- Ocular perfusion pressure (OPP)
 - $\text{OPP} = \text{MAP} - \text{IOP}$
 - $\text{Systolic SOPP} = \text{systolic BP} - \text{IOP}$
 - $\text{Diastolic DOPP} = \text{diastolic BP} - \text{IOP}$
 - $\text{Mean MOPP} = \frac{2}{3}(\text{MAP} - \text{IOP})$

Ocular blood flow exhibits autoregulation, which is characterised by local vascular constriction or dilatation causing an increase or decrease in vascular resistance, thereby maintaining a constant nutrient supply in response to perfusion pressure changes. Ocular perfusion pressure is directly proportional to ocular blood flow

$$\circ \text{ Ocular blood flow} = \frac{\text{ocular perfusion pressure}}{\text{Vascular resistance}}$$

Ocular blood flow and ischemic injury to the optic nerve head is dependent on ocular perfusion pressure and vascular resistance. Any changes in ocular perfusion pressure ((i.e.) fluctuation in IOP or blood pressure) or vascular resistance (i.e. autoregulatory dysfunction) affect ocular blood flow.

Lower ocular perfusion pressure is associated with an increased risk of the development of open-angle glaucoma as well as its progression. Several major studies, including the *Baltimore Eye Survey*, the *Barbados Eye Study*, the *Egna-Neumarkt Study*, and *Projecto Ver*, all studies identified low OPP as a risk factor for developing glaucoma. “In the Baltimore Eye Survey, glaucoma was six-fold more common in eyes with low versus high diastolic perfusion pressure.”

According to the data from the on-going Los Angeles Latino Eye Study (LALES), this is evaluating the relationship between OPP and the

risk of having open-angle glaucoma. This is a cross-sectional, population-based study of 6,130 adult Latinos in Los Angeles. All subjects underwent a comprehensive eye examination as well as blood pressure measurement. Overall, 1,770 subjects had systemic hypertension. It has been found that low systolic, diastolic, and mean OPP all led to increased risk of having open-angle glaucoma. Of these, the relationship between glaucoma risk and diastolic perfusion pressure was the strongest. The risk of glaucoma increases significantly as diastolic perfusion pressure falls.

In the LALES data, it has also been reported that high systemic blood pressure was also related to an increased risk of glaucoma. It's possible for both systemic hypertension and systemic hypotension to contribute to the risk of open-angle glaucoma. This is due to disruption of autoregulation of blood flow.

GLAUCOMA

The word *glaucoma* meant ‘*clouded*’ in Greek. Glaucoma is the second leading cause of blindness worldwide, with more than 50 million people affected. Glaucoma is a multifactorial disease and its precise pathogenesis, despite extensive research, remains unknown.

The term does not refer to a single disease entity, but rather to a group of diseases share certain features. It is characterised as a multifactorial optic neuropathy with a characteristic acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons, and manifesting characteristic visual field abnormalities.

Intraocular pressure (IOP) is considered the main risk factor for the development and progression of glaucoma. At present, decreasing IOP is the only proven means halting the development and/or progression OAG

Other risk factors for developing the disease

OCULAR RISK FACTORS	QUALITY OF EVIDENCE
1. Intraocular pressure	Excellent – Most Important
2. Thinner central corneal thickness	Excellent
3. Myopia	Excellent
4. Disc haemorrhage	Good

5.Increased cup/disc ratio	Equivocal
6.Asymmetric cupping	Equivocal

NON OCULAR RISK FACTORS	QUALITY OF EVIDENCE
--------------------------------	----------------------------

1. Age	Excellent
2. Race (e.g. African or Hispanic descent)	Excellent
3. Family History	Excellent
4. Adult onset diabetes	Equivocal
5.Diastolic perfusion pressure	Excellent
6.Migraine and peripheral vasospasm	Equivocal

There is increasing evidence that the cardiovascular system (e.g., altered blood flow) may play a major role in the pathogenesis OAG. The major cause of this reduction in ocular blood flow is thought to be secondary to vascular dysregulation in susceptible patients, resulting from abnormal/insufficient autoregulation.

CLINICAL STAGING OF GLAUCOMA:

1. Glaucoma suspects (at risk)
 - Optic disc asymmetry
 - Large physiological cup

- Low intraocular pressure in early to mid-twenties
- Pigment dispersion syndrome
- Positive family history of visual loss from glaucoma
- Myopia
- Hypermetropia
- Presence of a gene known to be associated with glaucoma

2. Pre glaucoma

- IOP above 30mmHg
- Abnormal disc appearance
- Narrow occludable anterior chamber angle
- Abnormal visual field without evidence of definite ocular damage

3. Glaucoma

- Asymptomatic glaucoma
- Definite glaucoma damage but without symptoms
- Symptomatic glaucoma

Pathogenesis of neuronal damage in glaucoma

Progressive optic neuropathy results from the death of retinal ganglion cells in a typical pattern which results in characteristic optic disc changes and visual field defects.

A) Primary insults

1. Mechanical theory

- Elevated IOP led to direct compression and death of the neurons.
- The entrance of optic nerve is the weakest part of eyeball increase in IOP this part pushed out first.
- Increase in intraocular pressure → backward bowing of lamina cribrosa → axonal deformation → axonal transport both orthograde and retrograde flow interrupted by compression & also leads to ischemia by altering capillary blood flow → neurotrophin deprivation → lead to apoptosis of retinal ganglion cells.

2. Vascular insufficiency theory (Pressure independent factors):

Factors affecting vascular perfusion pressure of optic nerve head are also implicated in glaucomatous optic neuropathy which includes

- Defective autoregulatory mechanism of blood flow
- Systemic hypotension - nocturnal hypotension , patients on anti-hypertensive medications
- Vasospasm

B) Secondary insults (excitotoxicity theory):

- Retinal ganglion cell apoptosis due to glutamate-mediated toxicity
- Glutamate is excitatory neurotransmitter → under hypoxic or ischemic conditions → glutamate accumulates and reaches toxicity concentration in the inter-synaptic junction.

GLAUCOMATOUS OPTIC DISC CHANGES:

A. Definitely pathogenic signs

- Documented diffuse thinning of neuroretinal rim
- Acquired pit of optic nerve (pseudo pit)
- Concentric enlargement of cup

B. Highly suggestive signs

- Asymmetric cup between eyes >0.2
- Vertical extension of cup
- Notching focal loss of neuroretinal rim
- Localized haemorrhage crossing the disc
- Bayoneting sign

C. Moderate to mildly suggestive signs

- CD ratio >0.6
- Localized pallor of optic disc
- Baring of circumlinear vessels

- Progressive vascular loops on the optic disc
- Narrowing of retinal vessels
- Dilated retinal veins
- Peripapillary area beta zone atrophy

D. Nonspecific signs

- Nasal shift of disc vessels
- Alteration of lamina cribrosa

VISUAL FIELD LOSS IN GLAUCOMA

Early defects:

- Generalized depression
- Nasal step or depression
- Temporal wedge or depression
- Enlargement and baring of blind spot
- Isolated paracentral scotoma

Late defects:

- Arcuate defects

- Annular scotoma

- end-stage field, with a small central island and a larger temporal crescent remaining.

ROLE OF OCULAR PERFUSION PRESSURE IN GLAUCOMA

It is well established that the main risk factor for glaucoma is elevated intraocular pressure (IOP). Reducing IOP is effective in slowing down the progression of the disease but some patients still progress despite adequately controlled IOP.

Besides the mechanical effect of raised intraocular pressure (IOP) on optic nerve head (ONH), several vascular risk factors such as systemic hypertension, atherosclerosis, vasospasm etc., have also been implicated as potential factors capable of increasing the risk of open-angle glaucoma (OAG).

The vascular hypothesis of OAG states that a low blood pressure (BP) relative to IOP can lead to low mean ocular perfusion pressure (MOPP), thus impairing perfusion of the ONH with resultant glaucomatous cupping and visual field loss.

Several studies implicated vascular risk factors in the pathogenesis of glaucoma. Blood pressure (BP) and ocular perfusion pressure (OPP) being the most studied. This vascular hypothesis is based on abnormal

perfusion and the subsequent ischemia of the ONH play a major role in the glaucomatous damage.

It has been reported that Low DBP, low MOPP and low DOPP were independent risk factors for OAG¹⁴. Of which Low DOPP has the strongest correlation with the development of glaucoma. The Baltimore Eye Survey found that those with DOPP <30 mmHg had a six-times higher risk of disease development than those with DOPP >50 mmHg³⁸. The Egna-Neumarkt Study reported a 4.5% increase in glaucoma prevalence in patients with DOPPs <50 mmHg compared with patients with DOPPs ≥66 mmHg. According to Leske MC et al study, low mean ocular perfusion pressure (<42 mmHg), systolic perfusion pressure (<101 mmHg) and DOPP (<55 mmHg) were all shown to be risk factors for the development of glaucoma, with relative risks of 3.1, 2.6 and 3.2, respectively.

Baseline for OPP³⁷:

- **Systolic OPP (SOPP) less than 101 mm Hg**
- **Diastolic OPP (DOPP) less than 55 mm Hg**
- **Mean OPP (MOPP) less than 42 mm Hg**

More likely to have open angle glaucoma.

Pathogenesis of Ocular Perfusion Pressure and Glaucoma:

Impaired autoregulation:

Blood flow to many organs and tissue beds is autoregulated. The goal of autoregulation is to maintain a relatively constant blood flow, capillary pressure, and nutrient supply in spite of changes in perfusion pressure. Vascular bed in the ONH and retina maintains autoregulatory capacity over a wide range of perfusion pressures.

Autoregulation of blood flow is due to alteration in resistance to blood flow in the terminal arterioles, i.e. they dilate to increase the blood flow when the perfusion pressure falls, and constrict to reduce the blood flow in arterial hypertension.

In an autoregulated vascular bed, changes in perfusion pressure do not translate into a change in blood flow, as long as the perfusion pressure is modified within the autoregulation range. However, in a non-autoregulated vascular bed, small changes in perfusion pressure may lead to changes in blood flow.

Breakdown of blood flow autoregulation in the ONH:

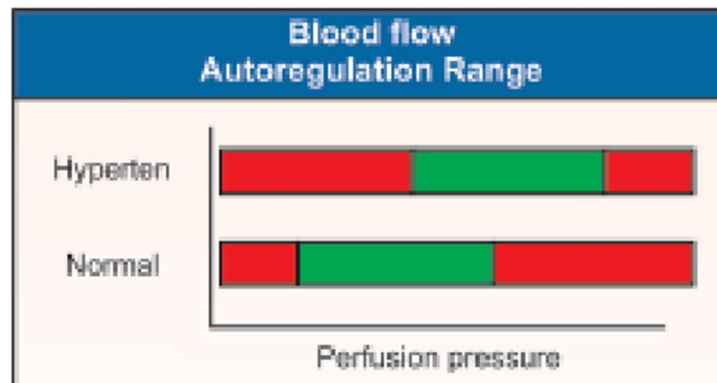
Many systemic and local factors can cause this. The systemic causes include aging, arterial hypertension, diabetes mellitus, marked arterial

hypotension from any cause, arteriosclerosis, atherosclerosis, hypercholesterolemia, vasospasm and probably regional vascular endothelial disorders³⁰⁻³³. It is also possible that many other, as yet unknown, causes can derange the autoregulation. And also some persons are born with defective autoregulation, for instance, those who suffer from Orthostatic arterial hypotension for no apparent reason.

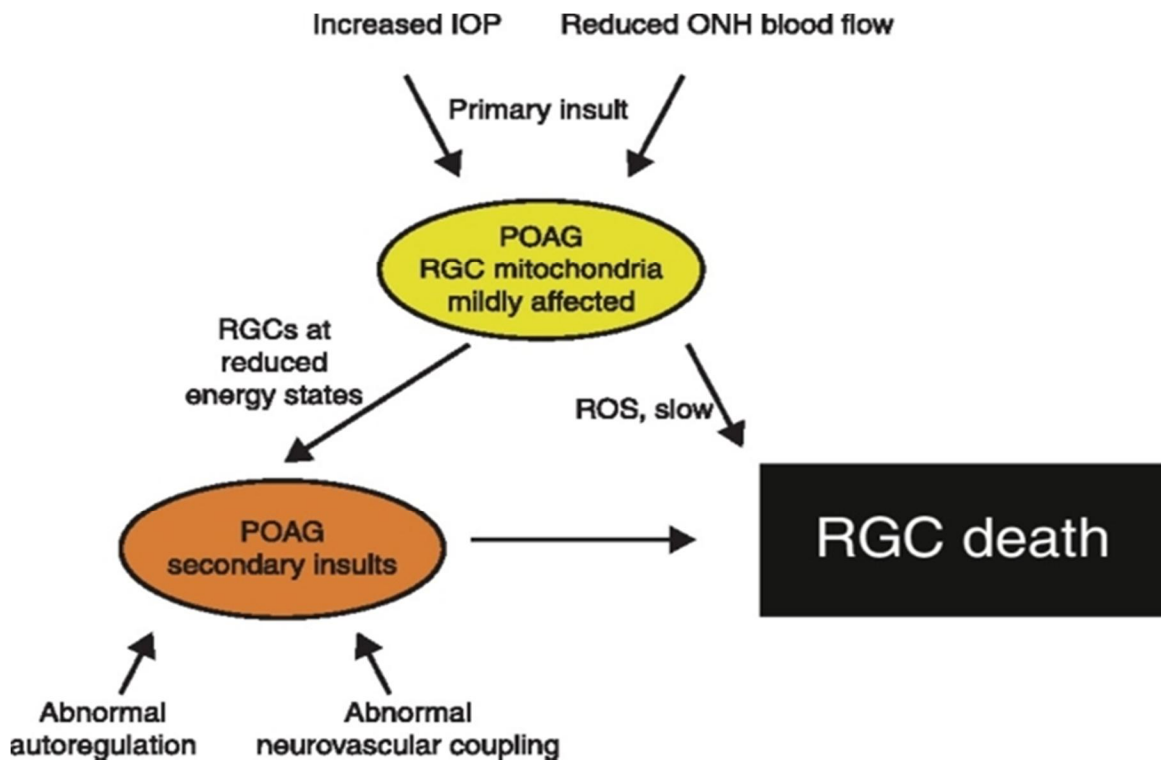
According to Liang et al. (2009) when OPP is challenged beyond the autoregulatory range, reduction of ocular blood flow occurs (ONH blood flow measured with laser speckle phenomenon¹³).

- When IOP was increased and mean BP was normal, no significant change was observed in ONH blood flow, indicating that autoregulation was adequate.
- When IOP was increased and mean arterial blood pressure was reduced down to 56 mmHg, significant reductions in ONH blood flow were observed.

Suggesting that autoregulation was unable to maintain blood flow at extremely low OPP.



A diagrammatic representation of range of blood flow autoregulation at different perfusion pressures in normal persons and in hypertensives (Hyperten). Green denotes range of presence of autoregulation and red its absence (Reproduced from Hayreh⁴⁸)



Vascular dysregulation is hypothesized to be involved in at least some Cases of glaucoma, especially in normal tension glaucoma and could lead not only to chronically low perfusion but too unstable perfusion with wide fluctuations.

Vascular Endothelial Vasoactive Agents:

Vascular Endothelial Vasoactive Agents (vasoconstrictor & vasodilator) are formed by vascular endothelium. They play an important role in modulating the local vascular tone and most probably also in blood flow autoregulation. Production of these agents is impaired in many systemic and local conditions, and that would secondarily interfere with the ONH circulation.

Fluctuation of BP and OPP:

Some studies have indicated that glaucoma patients show abnormal autoregulation especially in response to acute changes in OPP (Evans et al. 1999; Galambos et al. 2006; Feke & Palquale 2008; Portmann et al. 2011).

Any increase in IOP or reduction in blood pressure may reduce the perfusion pressure of intraocular beds, which represents a challenge to the ocular circulation.

Nagel et al. (2001) compared the reaction of retinal vessels to a short-term increase of IOP to supra-systolic values by the suction cup method, using a retinal Vessel analyser (RVA). A short-term rise in IOP leads to a reduced retinal vessel reaction (change in vessel diameter) in POAG patients and that this might represent an impaired autoregulation.

Circadian Variations in Ocular Perfusion Pressure

The term circadian rhythm was introduced in 1959 by Halberg¹⁶. It is defined as a biological cycle with a period of approximately 24 hr, representing an important biological regulator in every organism.

Among various circulatory circadian responses in humans, the most prominent is the blood pressure dip at night during sleep, due to a decrease in sympathetic output. IOP has also been shown to follow a pattern of circadian change. IOP and BP are the determining variables show circadian change.

It has been reported that during sleep, a reduction in both ocular perfusion pressure (OPP) and ocular blood flow may occur due to the combined effects of a rise in IOP and a dip of systemic BP (Trew & Smith 1991a,b; Perlman et al. 2007).

Low nocturnal OPP might result in further damage in glaucoma patients, especially when autoregulation is impaired (Okuno et al. 2004).

It has also been found that both anatomical (retinal nerve fibre layer thickness) and functional (visual field) outcome variables were significantly worse in glaucoma patients with wider circadian OPP fluctuation.

Choi et al. (2007) identified circadian OPP fluctuation as the most consistent clinical risk factor for glaucoma severity

The Evidence - Epidemiologic Studies Linking Diastolic Perfusion Pressure and Glaucoma⁸⁻¹²

S.NO	STUDY	DESIGN	Glaucoma Risk From Low DOPP vs. Normal DOPP
1.	Baltimore Eye Survey	Cross-sectional	6-fold
2.	Egna-Neumarkt Study	Cross-sectional	2.5-fold
3.	Proyecto VER	Cross-sectional	4-fold
4.	Los Angeles Latino Eye Study	Cross-sectional	1.9-fold
5.	Barbados Eye Study	Longitudinal	3.2-fold (4 years)

Patient Subgroups- To Consider the Value of Assessing Ocular

Perfusion Pressure:

- Normal-tension glaucoma
- Eyes with optic disc haemorrhage
- Patients with progression at low IOP
- History of low BP, multiple systemic antihypertensives, symptoms of orthostasis
- Patients with nocturnal hypotension

EFFECT OF INTRAOCULAR PRESSURE ON HEMODIALYSIS

Intraocular pressure (IOP) is a major risk factor for development and progression of the glaucomatous disease, and transient increases in IOP have been reported during HD in patients with and without glaucoma.

Systemic variables that may affect IOP are changes in blood volume, plasma osmolarity, and colloidal osmotic pressure. Hemodialysis affects all of these parameters, and given the significant number of patients on dialysis, this could be an important factor in IOP control.

Mechanism

1. Rapid reduction in total serum osmolarity²⁵⁻²⁸

- Most aqueous is produced by active transport mechanisms.
- The ultrafiltration component of aqueous production is significant.

It is this component that is affected by dialysis

Urea has established an osmotic ocular gradient. During the dialytic process, solutes urea toxins, in particular, are removed from the blood. Delayed transport of urea across blood-aqueous barrier has been demonstrated. Rapid removal of urea from plasma by hemodialysis will

thus not the significantly lower concentration of urea in aqueous and the serum osmolarity of the blood is reduced. This reduction in plasma or serum osmolarity favours net water movement in the direction of aqueous production. As the osmolar gradient intensifies, water flows into the aqueous fluid compartment and intraocular pressure rises.

Thus a rise in intraocular pressure could thus result from the osmotic movement of water from the plasma into aqueous. This effect of a decrease in blood urea on intraocular pressure as a result of a change in osmolarity can be counteracted by adding glucose to dialysate / by combining hemodialysis with ultrafiltration.

2. Colloid osmotic pressure²⁹

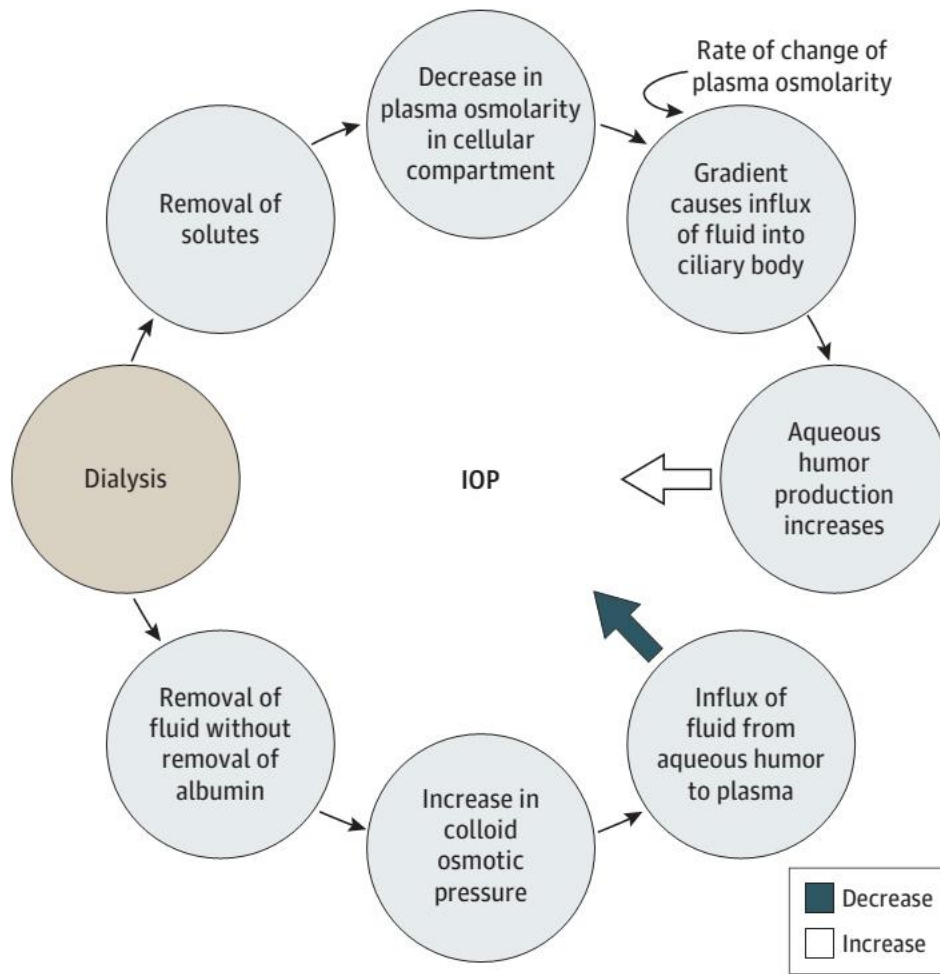
Fluid removal during ultrafiltration without concomitant albumin removal increases colloid osmotic pressure, leading to a fluid shift from the aqueous humor to the plasma and a decrease in IOP.

3. Postdialysis urea rebound (PDUR) and Haematocrit

PDUR, defined as urea measured 1 hr after dialysis subtracted from urea measured at the end of dialysis, was studied by Tovbin et al.

4. Compromised aqueous outflow facility²⁴

5. Narrow angles by gonioscopy²¹⁻²³



OCULAR FINDINGS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

Chronic kidney disease (CKD) is a progressive loss of renal function over a period of months or years. The global rise of CKD is primarily due not only to an increased ageing population, but also an association with other chronic conditions, including cardiovascular disease, diabetes (DM), and cancer.

Etiology of chronic kidney disease:

1. Diabetic nephropathy.
2. Hypertensive nephropathy.
3. Glomerulonephritis (inflammation of the kidney).
4. Pyelonephritis (infection of the kidney).
5. Systemic lupus erythematosus.
6. Polycystic kidney disease.
7. Long-term, regular use of medicines, such as non-steroidal anti-inflammatory drugs (NSAIDs)

Ocular findings in chronic kidney disease:

1. *Conjunctival calcium deposits*⁴⁰ - tiny, discrete, amorphous dull white deposits, usually in the interpalpebral area.
2. *Conjunctival erythema*, termed the red eyes of uremia, may be noted when high plasma phosphate levels induce corneal and conjunctival precipitation of calcium pyrophosphate.
3. *Corneal calcium deposits.*
4. *Band keratopathy.*
5. *Raised intraocular pressure.*
6. *Open angle glaucoma.*
7. *Hypertensive retinopathy.*
8. *Diabetic retinopathy.*
9. *Age-related macular degeneration.*
10. *Alport syndrome.*

PART- II

AIM

1. To assess the correlation of intraocular pressure changes during hemodialysis
2. To assess the correlation of ocular perfusion pressure changes during hemodialysis.
3. To determine that the screening for intraocular pressure and characteristic optic nerve head changes and visual field changes of glaucoma in end-stage renal disease patients under hemodialysis is reasonable and justifiable.

INCLUSION CRITERIA:

Criteria for selection of patient for study was:

1. Chronic kidney disease patients who are receiving hemodialysis therapy for \geq 1month at Dialysis Unit, Nephrology Department, Government Stanley Medical College, Chennai.

EXCLUSION CRITERIA:

1. Patients with pre-existing corneal abnormality.
2. Patients with ocular infection.
3. Patients with secondary glaucoma, narrow angles.
4. Patients with high myopia.
5. Patients with other ocular diseases, dense cataract, opaque media.

MATERIALS AND METHODS

This study was performed on 100 Chronic Kidney Disease (stage V) patients under Hemodialysis treatment, who are attending our Dialysis unit in Nephrology Department, Govt. Stanley Medical College, Chennai for more than one month were included in this study.

This study was done in accordance with the rules of ethical committee. All patients were informed about the purpose of the study and an informed consent was obtained.

Methods:

Patients falling inside the inclusion criteria were included in the study. A total of 100 patients were selected for this study.

A detailed Medical, Hemodialysis and Ocular history was collected from all the participants and they underwent a thorough physical examination, relevant laboratory tests regarding chronic kidney disease and complete ocular examinations.

All participants are subjected to Intraocular pressure measurement by using schiottz tonometry and Blood pressure measurement using sphygmomanometer on the upper arm over the brachial artery at three

different times during Hemodialysis session. Dialysis was performed with session durations of 3 to 5 hours. High-performance dialyzers (polysulfone) were used at a blood flow rate of 250 to 400 mL/min, and the systemic circulation was accessed through an arteriovenous fistula.

T1 → 15 minutes before initiation of hemodialysis

T2 → 2 hours after initiation of hemodialysis

T3 → 15 minutes after ending hemodialysis

Mean Arterial Pressure (MAP), Ocular Perfusion Pressure (OPP), Systolic Ocular Perfusion Pressure (SOPP), Diastolic Ocular Perfusion Pressure (DOPP) and Mean Ocular Perfusion Pressure (MOPP) were calculated.

The following formulas will be used to calculate at each time point:

- Mean arterial pressure (MAP)

$$\text{MAP} = \text{Diastolic BP} + 1/3 (\text{Systolic BP} - \text{Diastolic BP})$$

- Ocular perfusion pressure (OPP)

$$\text{OPP} = \text{MAP} - \text{IOP}$$

$$\text{SOPP} = \text{Systolic BP} - \text{IOP}$$

$$\text{DOPP} = \text{Diastolic BP} - \text{IOP}$$

$$\text{MOPP} = 2/3(\text{MAP} - \text{IOP})$$

Baseline for OPP³⁷:

- **Systolic OPP (SOPP) less than 101 mm Hg**
- **Diastolic OPP (DOPP) less than 55 mm Hg**
- **Mean OPP (MOPP) less than 42 mm Hg**

Each patient underwent detailed

Clinical ophthalmic examination:

- Best corrected visual acuity.
- Slit lamp examination of the anterior segment.
- Intraocular pressure measurement with schiottz tonometry and Goldman applanation tonometry.
- Goldman gonioscopy of anterior chamber angle.
- Visual fields by octopus field analyser.
- Detailed fundus and optic nerve evaluation by direct ophthalmoscope , 90D and indirect ophthalmoscope
- Fundus documentation using fundus camera.

Patients with MOPP, DOPP, SOPP below baseline and significant variation in IOP were reviewed every 3 months. The patients were subjected to all needed glaucoma screening investigations at each visit.

A maximum of 3 review visits was done in this study period. Patient's data at each visit were recorded and compared at each follow-up visits.

The data was analysed using **SPSS PACKAGE (statistical analysis of social science) version 16 and Microsoft excel 2007.**

The following statistical analyses are used in this study

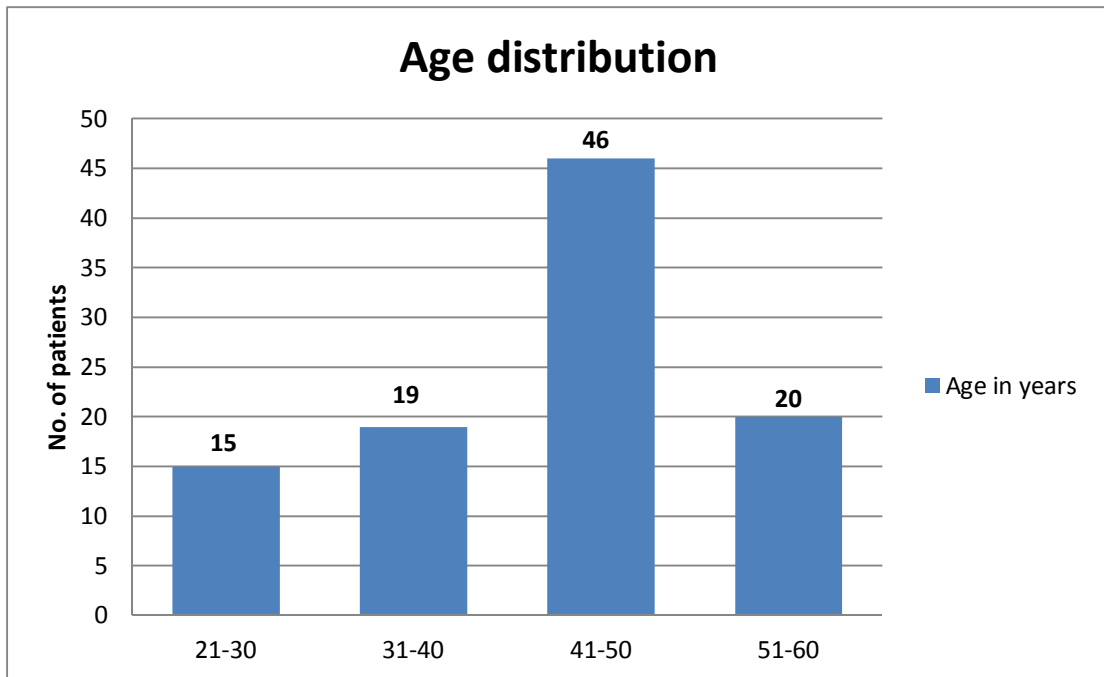
- a. Percentage analysis was calculated to find the exact percentage of respondents
- b. The mean score was calculated to find the average responses from the respondents.
- c. Standard deviations were also calculated to find the level of deviations among the respondents.
- d. Paired sample 't' test was used to find the significance difference between variables.

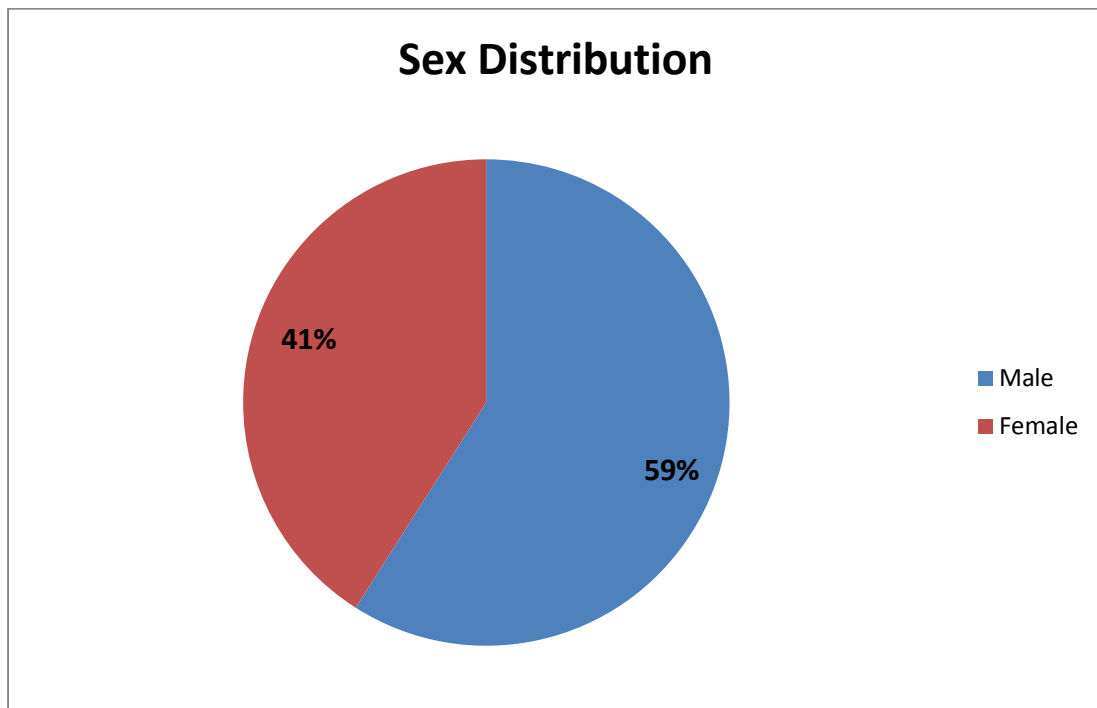
This helped in interpretation and interpreted data was summarized in percentage. Diagrams and tables were also used to highlight the interpretation.

OBSERVATIONS

Age and sex distribution

S.No	Age in years	Male	Female	Total
1	21-30	11	4	15
2	31-40	9	10	19
3	41-50	27	19	46
4	51-60	12	8	20
Total		59	41	100
Percentage		59	41	



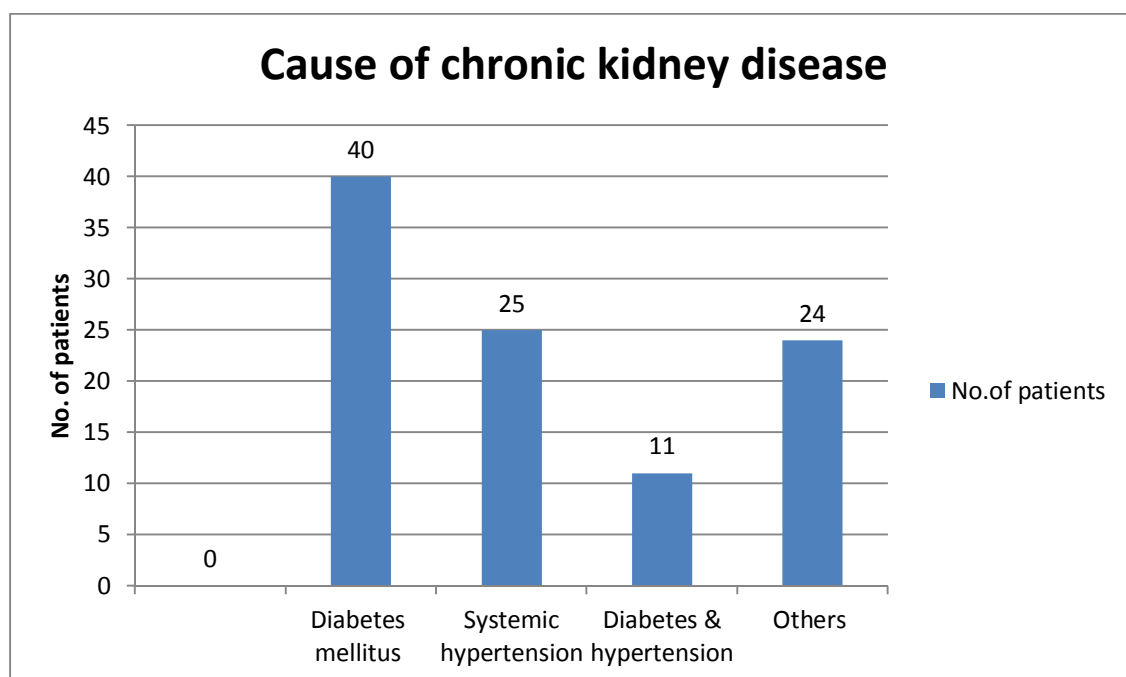


Maximum number of subjects were in the age group of 41-50 years

Maximum number of subjects were males (59%)

Cause of Chronic Kidney Disease

S.No	Cause	No.of patients (n=100)	Percentage (100%)
1.	Diabetes mellitus	40	40.0
2.	Systemic hypertension	25	25.0
3.	Diabetes & hypertension	11	11.0
4.	Others	24	24.0



Systolic blood pressure

Systolic blood pressure	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	126.70	116.90	115.60
SD	10.736	10.702	8.327

Mean change of systolic blood pressure during time interval

Time interval	Mean change of systolic blood pressure	SD	P-value paired t test
T1 to T2	-9.80	1.407	0.000
T2 to T3	-1.30	7.338	0.080
T1 to T3	-11.10	7.507	0.000

Mean systolic blood pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **11.10 mm of Hg**.

It was found to be statistically significant (p- value: 0.000)

Diastolic blood pressure

Diastolic blood pressure	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	82.50	74.30	74.00
SD	7.833	6.705	4.924

Mean change of diastolic blood pressure during time interval

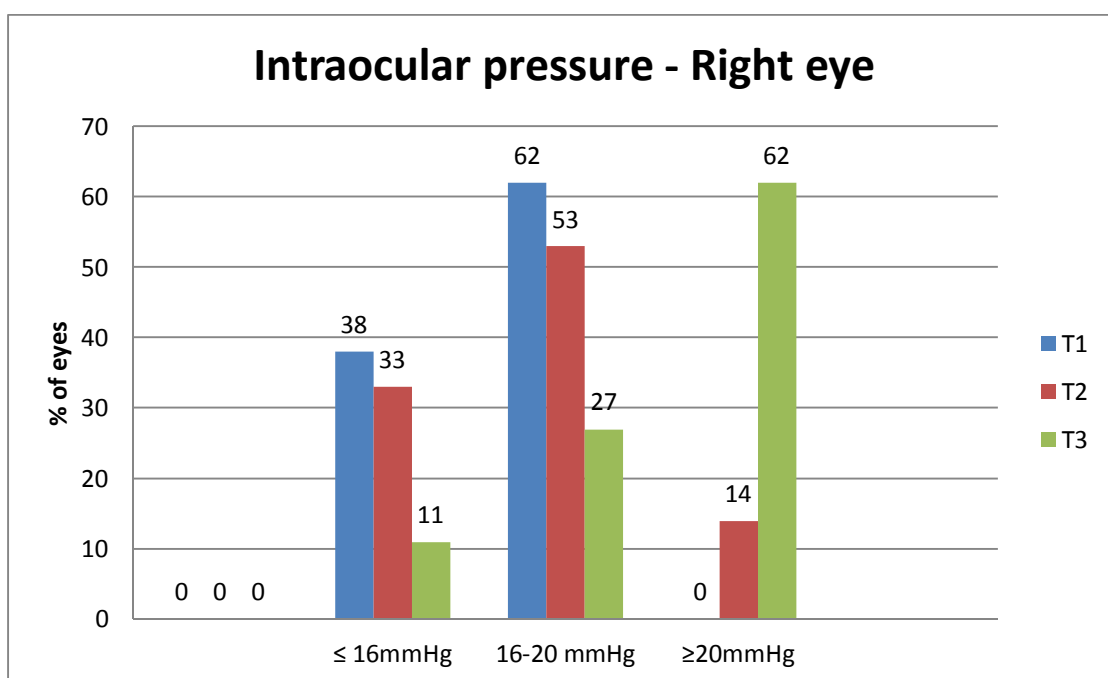
Time interval	Mean change of diastolic blood pressure	SD	P-value paired t test
T1 to T2	-8.20	3.861	0.000
T2 to T3	-0.30	5.588	0.593
T1 to T3	-8.50	6.872	0.000

Mean diastolic blood pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **8.50 mm of Hg**.

It was found to be statistically significant (p- value: 0.000)

Intraocular pressure(IOP) – Right eye

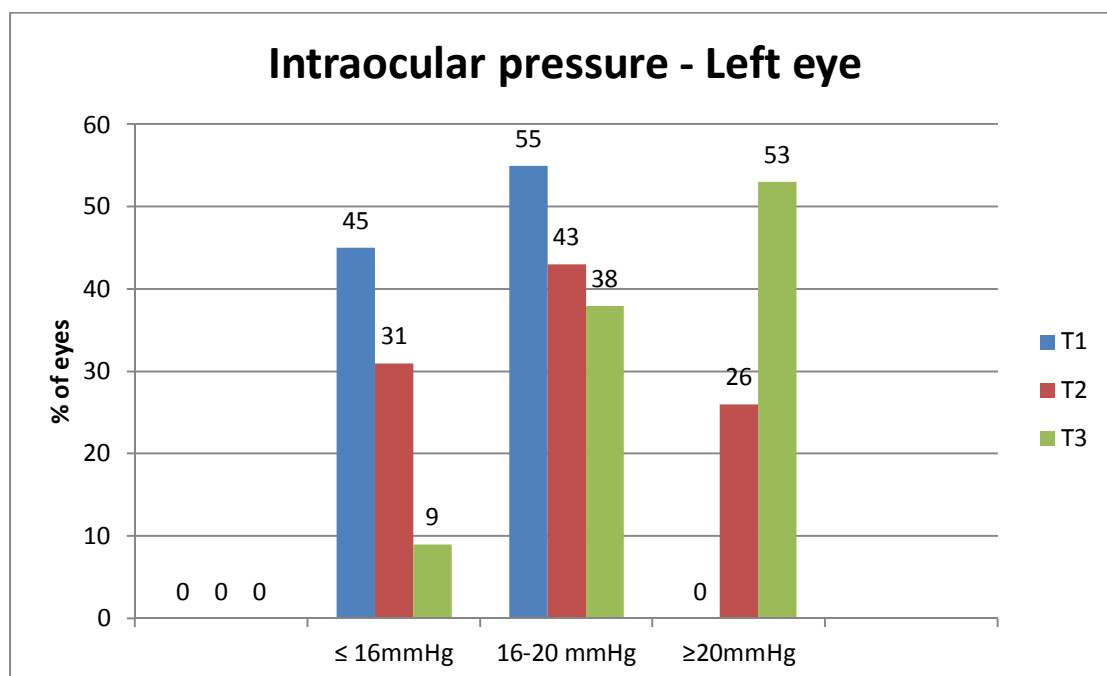
Intraocular pressure – Right eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 16mmHg	38.0	33.0	11.0
16-20 mmHg	62.0	53.0	27.0
≥20mmHg	0	14.0	62.0
Total	100	100.0	100.0



Intraocular pressure – Right eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	16.637	18.07	19.35
SD	1.5546	1.81	2.06

Intraocular pressure – Left eye

Intraocular pressure – Left eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 16mmHg	45.0	31.0	9.0
16-20 mmHg	55.0	43.0	38.0
≥20mmHg	0	26.0	53.0
Total	100.0	100.0	100.0



Intraocular pressure – left eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	16.784	18.203	19.284
SD	1.8886	2.3205	2.2060

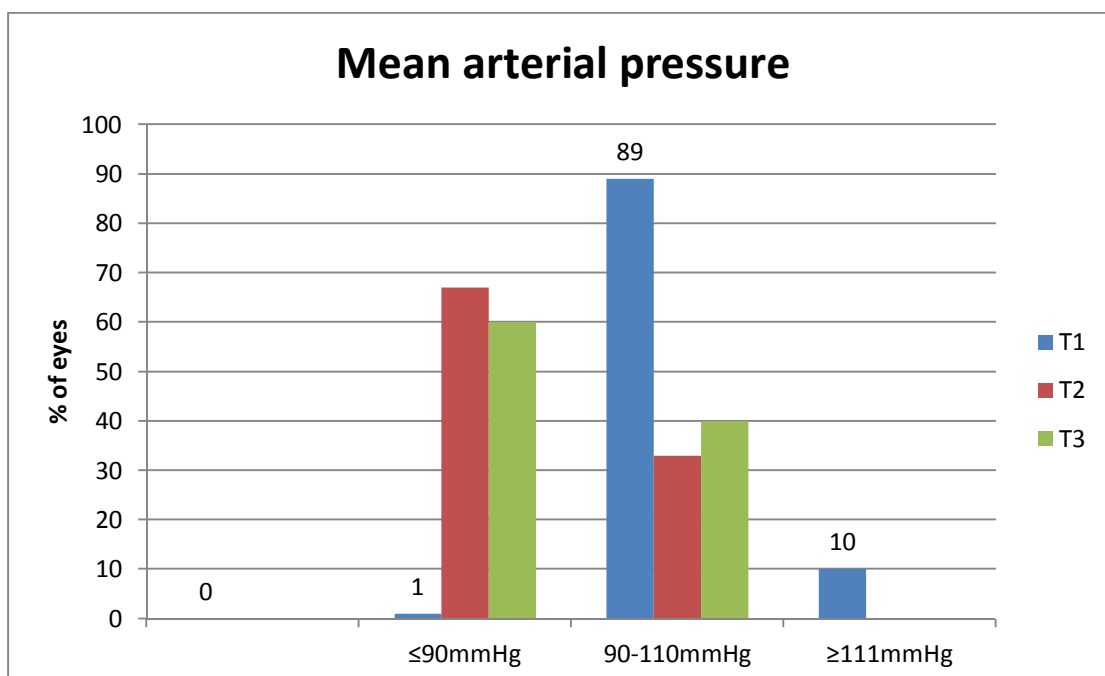
Mean change of Intraocular pressure during time interval

Time interval	Right eye			Left eye		
	Mean change of IOP	SD	P-value paired t test	Mean change of IOP	SD	P-value Paired t test
T1 to T2	1.435	.4527	0.000	1.419	.9005	0.000
T2 to T3	1.285	.5615	0.000	1.081	.8394	0.000
T1 to T3	2.720	.8828	0.000	2.500	1.0477	0.000

Mean IOP from T1 to T3 (from the initiation to the end of hemodialysis) was found to be increased by **2.7mm of Hg** in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

Mean arterial pressure (MAP)

Mean arterial pressure (MAP)	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤90mmHg	1.0	67.0	60.0
90-110mmHg	89.0	33.0	40.0
≥111mmHg	10.0	0	0
Total	100.0	100	100



Mean arterial pressure (MAP)	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	97.656	88.236	87.522
SD	8.0791	8.0950	5.8831

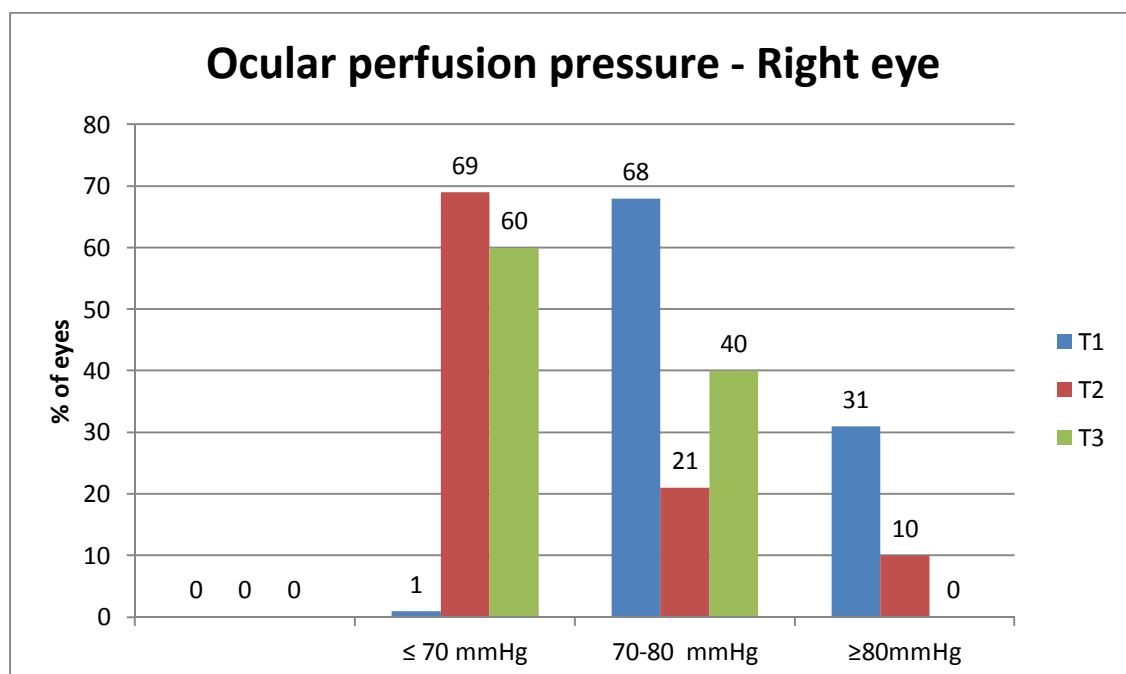
Mean change of Mean arterial pressure (MAP) during time interval

Time interval	Mean change of MAP	SD	P-value paired t test
T1 to T2	-9.420	2.0280	0.000
T2 to T3	-0.714	6.1106	0.245
T1 to T3	-10.134	6.5030	0.000

Mean arterial pressure (MAP) from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **10.13 mm of Hg**. It was found to be statistically significant (p- value: 0.000)

Ocular perfusion pressure (OPP) – Right eye

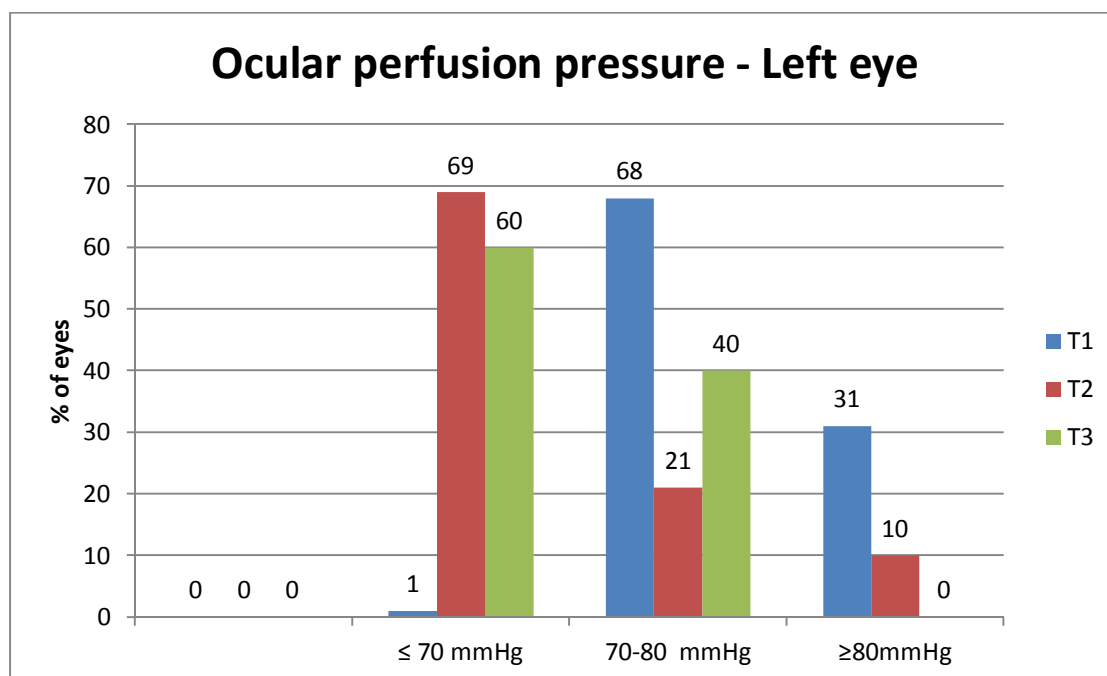
Ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 70 mmHg	1.0	69.0	60.0
70-80 mmHg	68.0	21.0	40.0
≥80mmHg	31.0	10.0	0
Total	100.0	100.0	100.0



Ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	81.045	69.990	68.201
SD	7.8347	7.8999	6.7689

Ocular perfusion pressure – Left eye

Ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 70 mmHg	1.0	69.0	60.0
70-80 mmHg	68.0	21.0	40.0
≥80mmHg	31.0	10.0	0
Total	100.0	100.0	100



Ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	80.900	69.851	68.186
SD	7.1442	7.8713	6.7353

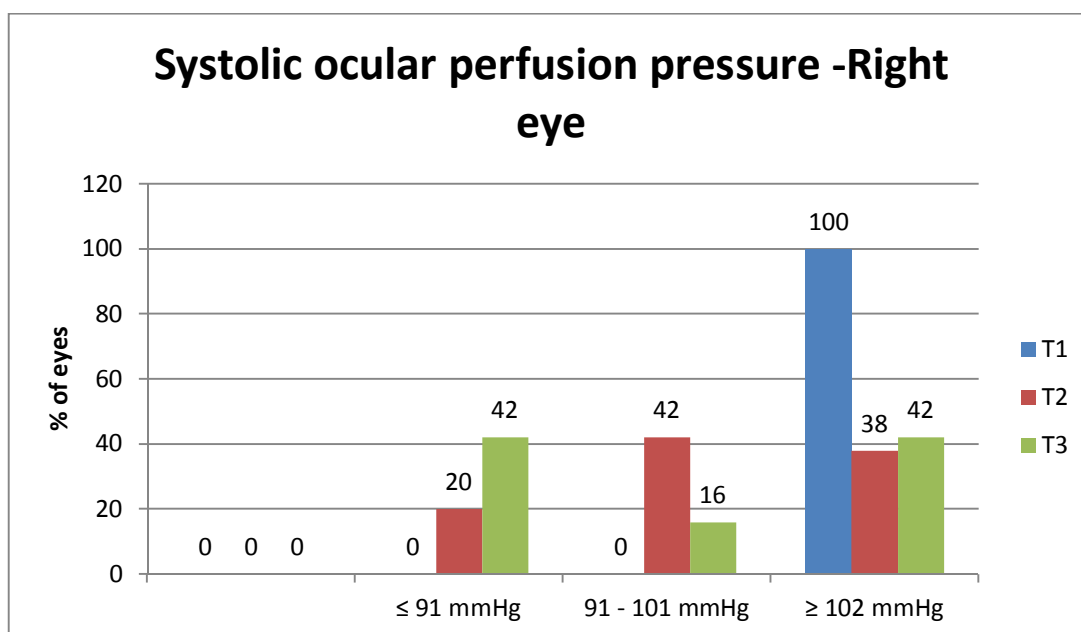
Mean change of Ocular perfusion pressure (OPP) during time interval

Time interval	Right eye			Left eye		
	Mean change of OPP	SD	P-value paired t test	Mean change of OPP	SD	P-value Paired t test
T1 to T2	-11.055	1.5728	0.000	-11.049	2.0055	0.000
T2 to T3	-1.789	6.5732	0.008	-1.665	6.5306	0.012
T1 to T3	-12.844	7.0538	0.000	-12.714	6.8398	0.000

Ocular perfusion pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **12.84 mm Hg** in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

Systolic ocular perfusion pressure (SOPP) – Right eye

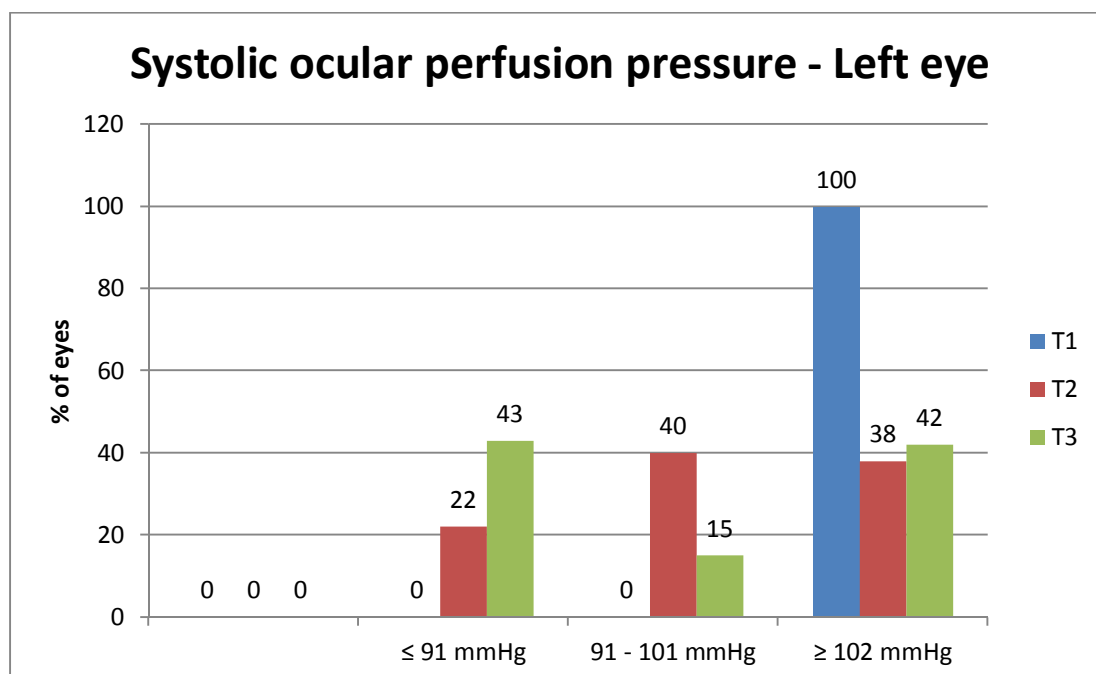
Systolic ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 91 mmHg	0	20.0	42.0
91 - 101 mmHg	0	42.0	16.0
≥ 102 mmHg	100.0	38.0	42.0
Total	100.0	100.0	100.0



Systolic ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	110.489	99.524	96.573
SD	10.6946	10.6343	8.8659

Systolic ocular perfusion pressure – Left eye

Systolic ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 91 mmHg	0	22.0	43.0
91 - 101 mmHg	0	40.0	15.0
≥ 102 mmHg	100.0	38.0	42.0
Total	100.0	100.0	100.0



Systolic ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	110.328	99.415	96.616
SD	9.9877	10.5267	8.7909

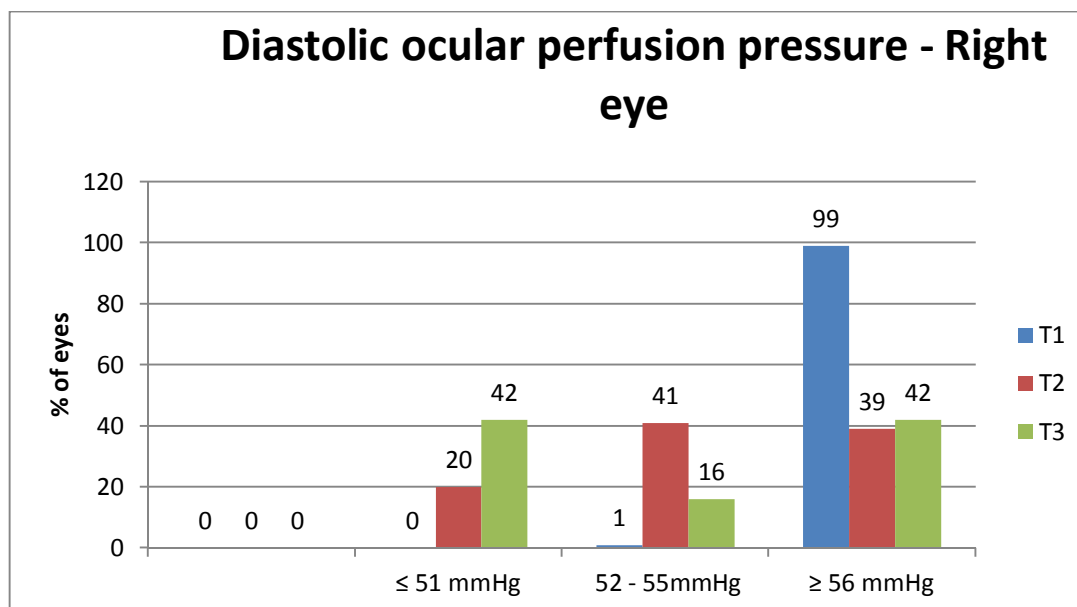
Mean change of Systolic Ocular perfusion pressure (SOPP) during time interval

Time interval	Right eye			Left eye		
	Mean change	SD	P-value paired t test	Mean change	SD	P-value Paired t test
T1 to T2	-10.965	2.2154	0.000	-10.913	2.3815	0.000
T2 to T3	-2.951	7.3428	0.000	-2.799	7.3481	0.000
T1 to T3	-13.916	7.9550	0.000	-13.712	7.7918	0.000

Systolic ocular perfusion pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **13.92 mm Hg** in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

Diastolic ocular perfusion pressure (DOPP) – Right eye

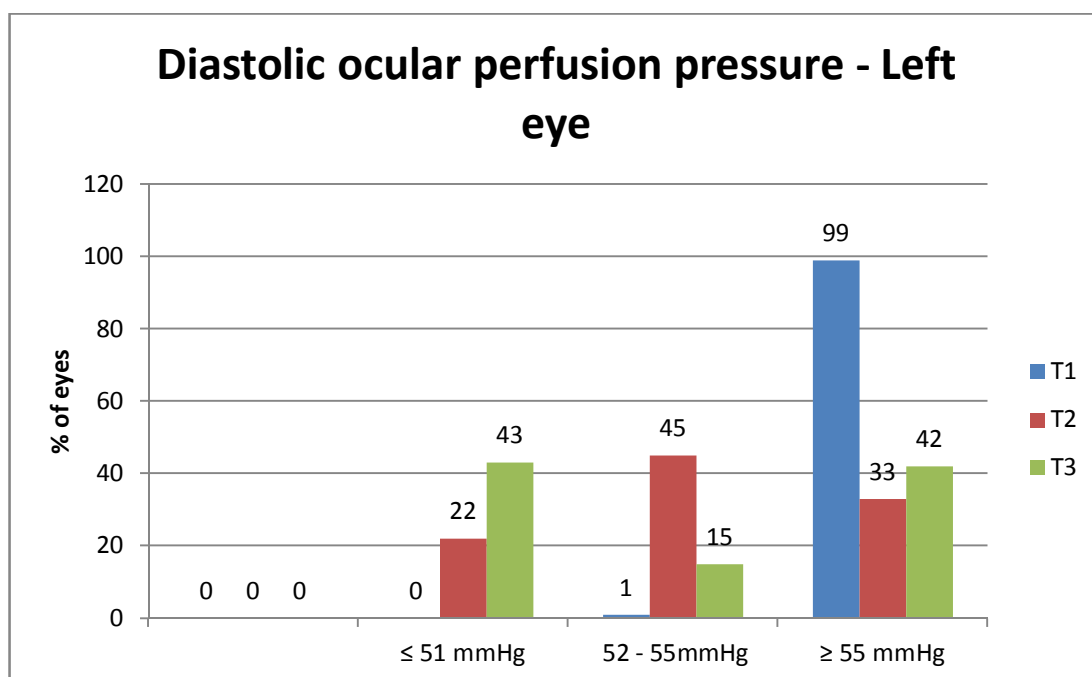
Diastolic ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 51 mmHg	0	20.0	42.0
52 - 55mmHg	1.0	41.0	16.0
≥ 56 mmHg	99.0	39.0	42.0
Total	100.0	100.0	100.0



Diastolic ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	66.889	56.354	54.873
SD	6.4941	6.7739	6.1247

Diastolic ocular perfusion pressure – Left eye

Diastolic ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 51 mmHg	0	22.0	43.0
52 - 55mmHg	1.0	45.0	15.0
≥ 55 mmHg	99.0	33.0	42.0
Total	100.0	100.0	100.0



Diastolic ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	66.744	56.235	54.916
SD	5.8260	6.8524	6.1358

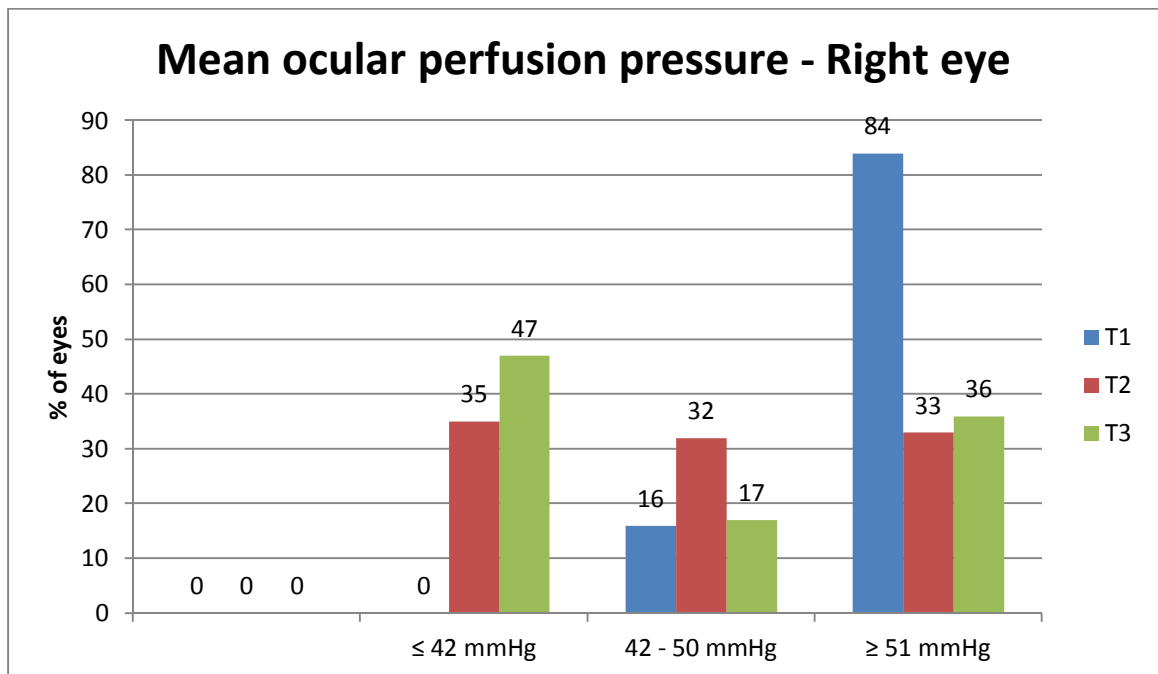
Mean change of Diastolic ocular perfusion pressure (DOPP) during time interval

Time interval	Right eye			Left eye		
	Mean change	SD	P-value paired t test	Mean change	SD	P-value Paired t test
T1 to T2	-10.535	3.2280	0.000	-10.509	3.5680	0.000
T2 to T3	-1.481	6.1205	0.017	-1.319	6.1056	0.033
T1 to T3	-12.016	6.9660	0.000	-11.828	6.8300	0.000

Diastolic ocular perfusion pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **12.016 mm Hg** in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

Mean ocular perfusion pressure (MOPP) – Right eye

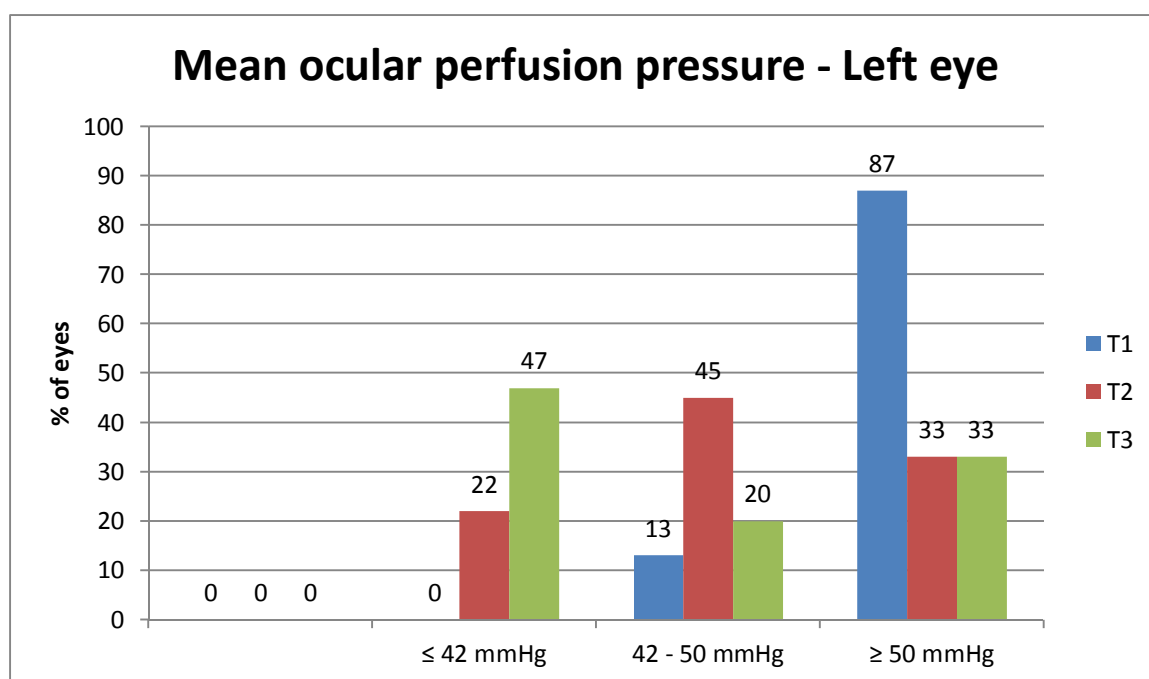
Mean ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 42 mmHg	0	35.0	47.0
42 - 50 mmHg	16.0	32.0	17.0
≥ 51 mmHg	84.0	33.0	36.0
Total	100.0	100.0	100.0



Mean ocular perfusion pressure – Right eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	53.918	46.634	45.328
SD	5.1937	5.4039	4.5408

Mean ocular perfusion pressure – Left eye

Mean ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis) % of eyes	T2 (2 hours after initiation of hemodialysis) % of eyes	T3 (15mins after ending hemodialysis) % of eyes
≤ 42 mmHg	0	22.0	47.0
42 - 50 mmHg	13.0	45.0	20.0
≥ 50 mmHg	87.0	33.0	33.0
Total	100.0	100.0	100.0



Mean ocular perfusion pressure – Left eye	T1 (15mins before hemodialysis)	T2 (2 hours after initiation of hemodialysis)	T3 (15mins after ending hemodialysis)
N	100	100	100
Mean	53.892	46.647	45.302
SD	4.7592	5.2583	4.5284

Mean change of Mean ocular perfusion pressure (MOPP) during time interval

Time interval	Right eye			Left eye		
	Mean change	SD	P-value paired t test	Mean change	SD	P-value Paired t test
T1 to T2	-7.284	1.6074	0.000	-7.245	1.7009	0.000
T2 to T3	-1.306	4.2474	0.003	-1.345	4.2267	0.002
T1 to T3	-8.590	4.7445	0.000	-8.590	4.5734	0.000

Mean ocular perfusion pressure from T1 to T3 (from the initiation to the end of hemodialysis) was found to be decreased by **8.590 mm Hg** in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

Applying the thresholds used in the Barbados eye studies for evaluating relative risk of open angle glaucoma

**Percentage of Ocular perfusion pressure changes below threshold
(Total 100 patients)**

	T1 (15mins before hemodialysis) % of eyes		T2 (2 hours after initiation of hemodialysis) % of eyes		T3 (15mins after ending hemodialysis) % of eyes	
	Right eye	Left eye	Right eye	Left eye	Right eye	Left eye
Mean ocular perfusion pressure $\leq 42\text{mmHg}$	0	0	36	31	48	48
Diastolic ocular perfusion pressure $\leq 55\text{mmHg}$	0	0	63	66	58	58
Systolic ocular perfusion pressure $\leq 101\text{mmHg}$	0	0	63	60	58	58

RESULTS

A total of 200 eyes of 100 subjects were recruited in our study. They were in the age group ranging from 25 to 62 years.

All the recruited patients were within inclusion criteria of our study.

The following were the results of our study:

1. Mean IOP from the initiation to the end of hemodialysis was found to be increased by 2.7mm Hg in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)
2. Mean arterial pressure (MAP) from the initiation to the end of hemodialysis was found to be decreased by 10.13 mm Hg. It was found to be statistically significant (p- value: 0.000)
3. Ocular perfusion pressure from the initiation to the end of hemodialysis was found to be decreased by 12.84 mm Hg in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)
4. Systolic ocular perfusion pressure from the initiation to the end of hemodialysis was found to be decreased by 13.92 mm Hg in both

eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

5. Diastolic ocular perfusion pressure from the initiation to the end of hemodialysis was found to be decreased by 12.016 mm Hg in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

6. Mean ocular perfusion pressure from the initiation to the end of hemodialysis was found to be decreased by 8.59 mm Hg in both eyes. It was found to be statistically significant (RE p- value: 0.000, LE p-value 0.000)

7. Applying the thresholds used in the Barbados eye studies for evaluating relative risk of open angle glaucoma development and progression:

a. 58 % of both eyes had a Systolic ocular perfusion pressure of \leq 101mmHg

b. 58 % of both eyes had a Diastolic ocular perfusion pressure of \leq 55mmHg

c. 48% both eyes had a Mean ocular perfusion pressure of \leq 42mmHg.

Patients were asked for follow-up based on their findings.

Among the 100 patients, 58 patients had Ocular perfusion pressure (OPP, MOPP, SOPP, DOPP) below baseline at their first visit examination. Hence they were followed up every 3 months.

Among 58 patients advised follow-up, 46 attended regular follow-up of maximum 3 visits every 3 months during our study period.

They underwent the needed ocular and systemic examinations in all the 3 visits and the results were compared. 12 Patients did not attend complete follow-up as per advice. In these 12 patients **2 patients** had early field changes and optic nerve head changes but we lost follow up of them in following visits.

At the end of this 2 year period, **4 of the 46 patients** were found to develop early glaucomatous field defects and early optic nerve head changes in both eyes at follow-up visits compared to normal fields and optic nerve head in their initial visits. They were also found to have increased in intraocular pressure (≥ 21 mm Hg in both eyes) compared to the initial reading. (FOLLOW UP CHART IN ANNEXURE)

GLAUCOMATOUS FIELD DEFECTS

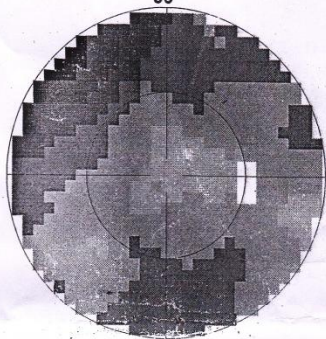
HAAG-STREIT
Seven-in-One

OCTOPUS 300Series

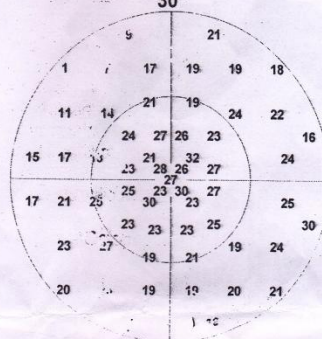
V 6.07f

Name:	LAKSHMANAN	Eye / Pupil[mm]:	Right(OD) / 3
First name:	G	Date / Time:	09/02/2016 09:23 AM
ID #:	107423	Test duration:	2:47
Birthdate:	09/01/1984	Program / Code:	G1
Age:	32	# Stages / Phases:	/ 1
Sex:	male	Strategy / Method:	TOP / Normal
Refr. S / C / A:	/ /	Test target / duration:	III / 100 ms
Acuity:	6/6	Background:	10 cd/m ²
IOP:	18	# Questions / Repetitions:	72 / 0
Diagnostics:		# Catch trials:	pos 0 / 4, neg 0 / 4
Patient file:		C:\Program Files\Octopus\ExDat\data z.PVD	

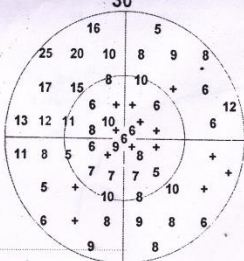
Greyscale of values
30°



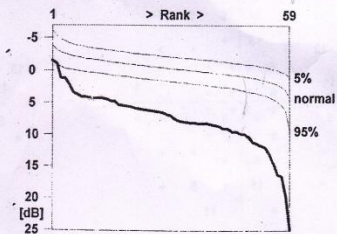
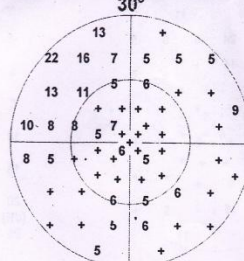
Values
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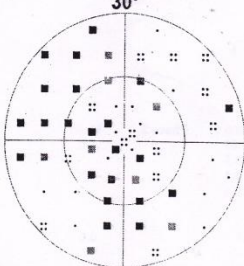
Comparisons
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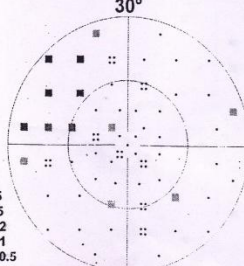
Corrected comparisons
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Probability
30°



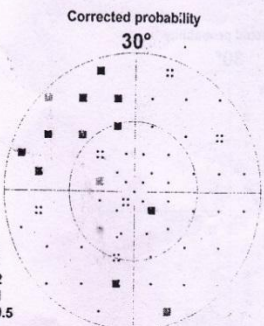
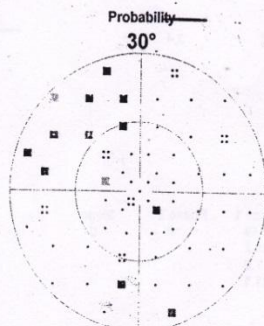
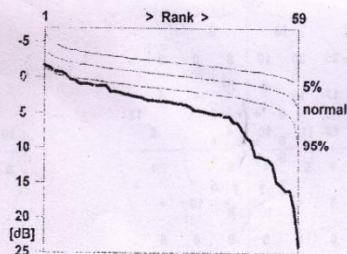
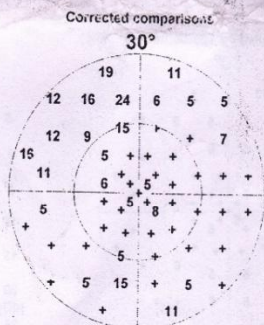
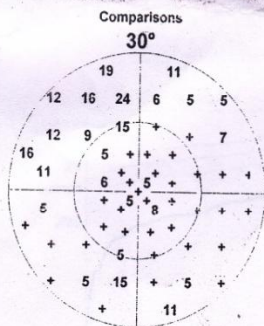
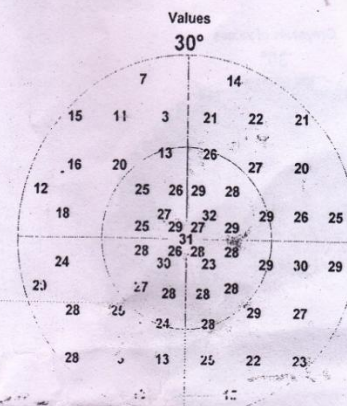
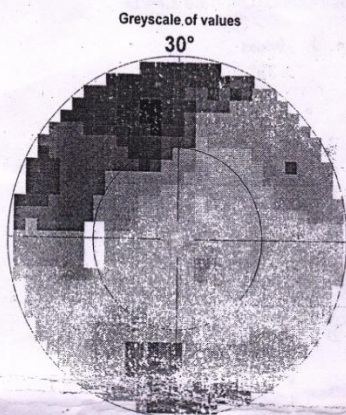
Corrected probability
30°



Deviation [dB] 3.4

#	Phase 1	Phase 2	Mean
MS	59	0	0
MD	21.2		
LV	7.6		
CLV	21.1		
SF			
RF			0.0

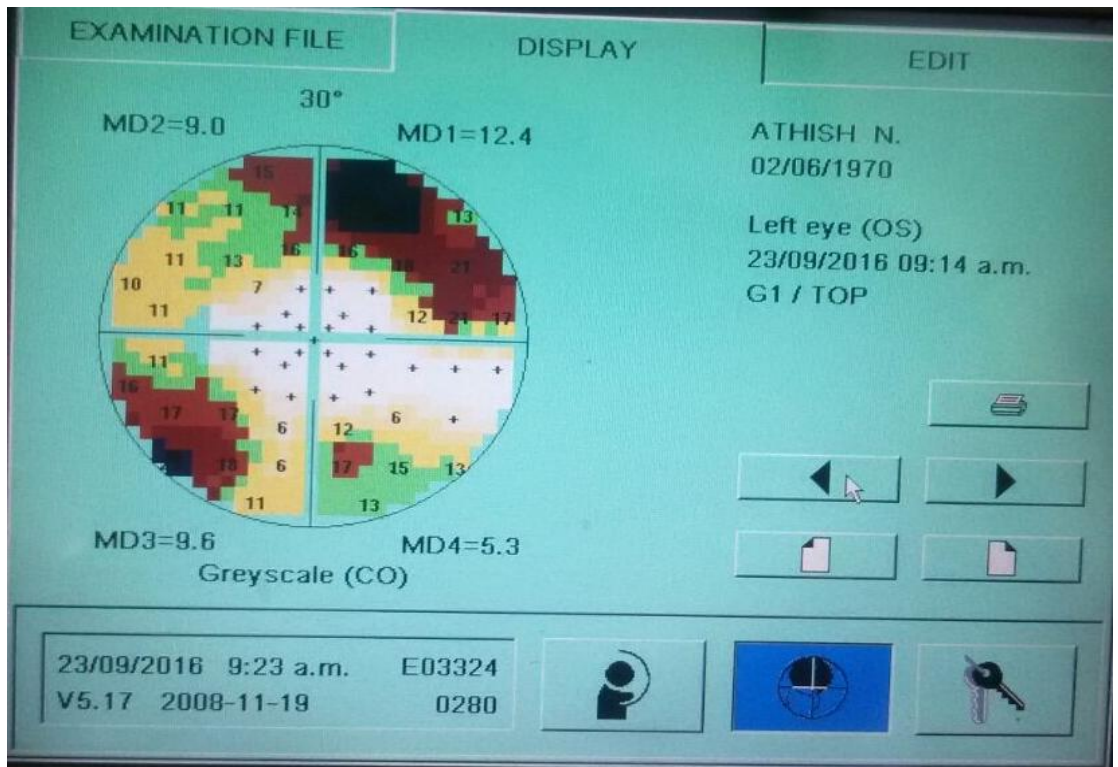
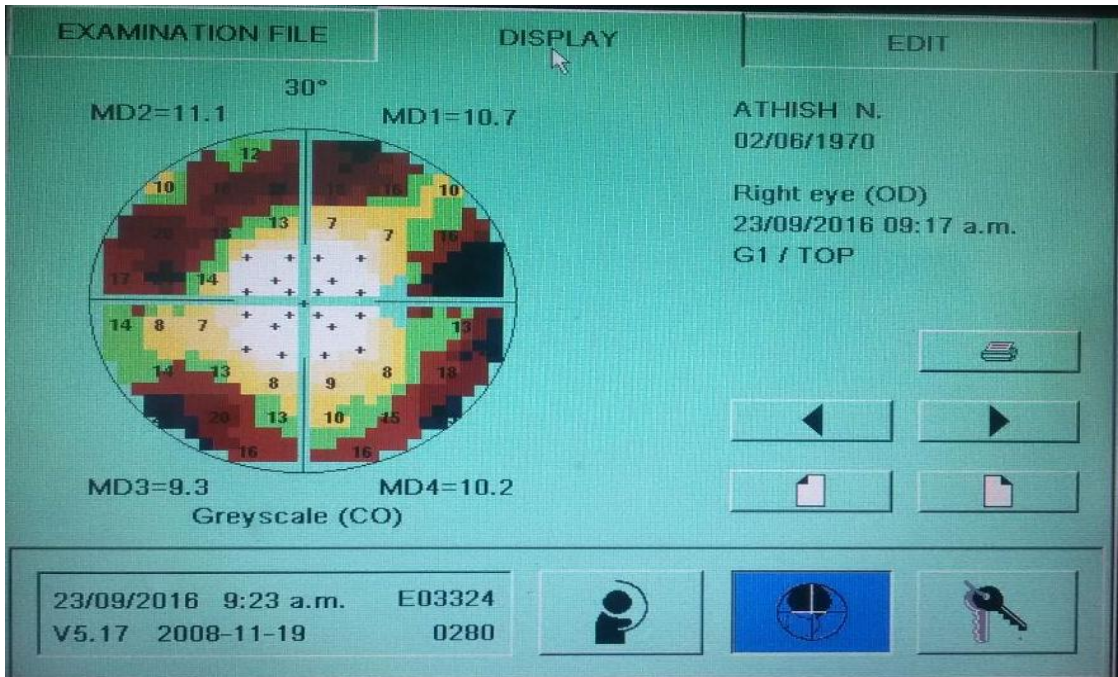
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 First name: G Date / Time: 09/02/2016 09:28 AM
 ID #: 107423 Test duration: 2:44
 Birthdate: 09/01/1984 Program / Code: G1
 Age: 32 # Stages / Phases: / 1
 Sex: male Strategy / Method: TOP / Normal
 Refr. S / C / A: / / Test target / duration: III / 100 ms
 Acuity: 6/6 Background: 10 cd/m²
 IOP: 18 # Questions / Repetitions: 71 / 0
 Diagnostics: # Catch trials: pos 0 / 3, neg 0 / 4
 Patient file: C:\Program Files\Ocupus\ExDat\data z.PVD



· P>5
 :: P<5
 ■ P<2
 ■ P<1
 ■ P<0.5

Deviation [dB] 0.0

#	Phase 1	Phase 2	Mean
MS	59	0	0
MD	23.7		
LV	5.1		
CLV	29.1		
SF			
RF			0.0



GLAUCOMATOUS OPTIC NERVE HEAD CHANGES



DISCUSSION

Our study reveals statistically significant increase in intraocular pressure during hemodialysis.

Our study also reveals that there is statistically significant decrease in ocular perfusion pressure, diastolic ocular perfusion pressure, systolic ocular perfusion pressure and mean ocular perfusion pressure during hemodialysis.

Similar results were also given by other related studies:

Burn RA et al²⁵ in 1973 observed an IOP rise in one-third of the patients treated with long-term HD.

Jennifer Hu et al.¹⁸ study in 2013 have also reported that statistically significant rise in IOP by 3.1mmHg during Hemodialysis session. Our study also concludes the same that there is a significant increase in intraocular pressure by 2.7 mmHg during a hemodialysis session.

	Jennifer Hu et al. study 2013		Chhabra S C et al. study 1988	OUR STUDY	
	RE	LE	MEAN	RE	LE
OBSERVATION	IOP before HD 19.1 mmHg	IOP before HD 17.7 mmHg	Mean IOP before HD 16.8 ± 0.29 mmHg	IOP 15 mins before HD 16.6 mmHg	IOP 15 mins before HD 16.8 mmHg
	IOP at 2 hours after starting HD 21.0mmHg	IOP at 2 hours after starting HD 19.2mmHg	Mean IOP at 2 hours of HD 16.9 ± 0.34 mm Hg	IOP at 2 hours after starting HD 18.07mmHg	IOP at 2 hours after starting HD 18.2mmHg
	IOP after HD 22.2 mmHg	IOP after HD 20.8 mmHg	Mean IOP at end of HD 16.7 ± 0.33 mmHg	IOP 15 mins after ending HD 19.35mmHg	IOP 15 mins after ending HD 19.28mmHg
INFERENCE	IOP significantly increased by 3.1mmHg in both eyes (p<0.001)		No statistically significant difference in IOP. (P<0.05)	IOP significantly increased by 2.7mmHg in both eyes (p<0.001)	

Chhabra S C et al.⁴¹ study in 1988 reported that there was no statistically significant difference in IOP ($P < 0.05$). But in one patient with a borderline intraocular pressure in one eye & raised intraocular pressure in the other eye, there was a considerable rise (10.1 mm Hg in one eye & 7.7 mm Hg in the other eye) in intraocular pressure at 2 hours & comparatively smaller rise (5.6 mm Hg in one eye & 3.7 mm Hg in the other eye) at the end of dialysis.

The reason behind raise in IOP during hemodialysis has been explained as an effect secondary to a rapid decrease in plasma osmolarity and a relative increase in intracellular compared with extracellular urea concentration. This rapid change results in a gradient between plasma and ocular compartments, inducing a shift of extracellular fluid from the blood to the anterior chamber. And also increase in IOP is more pronounced in eyes with compromised aqueous outflow.

The evidence is increasing that IOP and BP instability may be associated with glaucoma. Variations in ocular perfusion pressure due to IOP and BP fluctuations may play a role in glaucoma development or progression.

Fluctuation of ocular perfusion pressure is one of the known risk factors of development and progression of Normal tension glaucoma.

Jennifer Hu et al in 2013 have reported that significant decrease in OPP, MOPP, DOPP, SOPP occur during the active process of Hemodialysis. Our study also concluded that ocular perfusion pressure is significantly reduced during a hemodialysis session.

		Jennifer Hu et al. study 2013		OUR STUDY		
OBSERVATION		Mean change from start to end of HD session (mmHg)	p-value	Mean change from start to end of HD session (mmHg)	p- value	
	OPP	RE	-8.9	0.002	-12.8	<0.001
		LE	-8.7	0.004	-12.7	<0.001
	SOPP	RE	-11.4	0.01	-13.9	<0.001
		LE	-11.0	0.02	-13.7	<0.001
	DOPP	RE	-7.7	0.001	-12.01	<0.001
		LE	-7.5	0.002	-11.8	<0.001
	MOPP	RE	-7.0	<0.0013	-8.6	<0.001
		LE	-6.8	0.001	-8.6	<0.001
INFERENCE		All OPP measures significantly decreased ($p \leq 0.02$)		All OPP measures significantly decreased ($p < 0.001$)		

According to the thresholds used in the Barbados eye studies for evaluating the relative risk of open angle glaucoma :

		Jennifer Hu et al. study 2013		OUR STUDY	
OBSERVATION		Percentage of eyes below baseline threshold (%)		Percentage of eyes below baseline threshold (%)	
		Right eye	Left eye	Right eye	Left eye
	SOPP	53	46	58	58
	DOPP	71	73	58	58
	MOPP	63	65	51	51

Reasons of ocular perfusion pressure fluctuation during hemodialysis session are as follows :

- Autoregulation, the ability of vascular bed to change the vascular resistance in response to the perfusion pressure changes to maintain a relatively constant blood flow- plays a crucial role if undergoes impairment.

- Fluctuation changes of BP secondary to fluid shifts during active process of hemodialysis.

Our study found that there is statistically significant fluctuation in ocular perfusion pressure during a hemodialysis session. Accordingly, hemodialysis patients might have frequent IOP and OPP fluctuations during long-term, frequent sessions of hemodialysis, each lasting several hours may subsequently increase patients risk for glaucoma development and progression.

Survival rates of hemodialysis patients have been studied which showed that there is an improvement in life expectancy of end-stage renal disease patients after hemodialysis initiation⁴⁴.

Barbados eye study demonstrated that increased risk of glaucoma development at 4 to 9 years in patients with low baseline OPP.

Our study found that when patients with ocular perfusion pressure below baseline were regularly followed up over the study period of 2 years, 6 patients of the 58 patients developed early glaucomatous field defects and early optic nerve head changes in both eyes at follow-up visits compared to normal fields and optic nerve head in their initial visits.

According to Barbados eye study risk of glaucoma development was demonstrated as 4-9 years following low baseline OPP. Even though in our study we followed up patients only for 2 years post hemodialysis, 6 out of 58 patients developed early glaucoma field changes and optic nerve head changes. Which comes around 10%

Hence our study reveals the importance of screening and monitoring of intraocular pressure and characteristic early optic nerve head changes and early visual field changes of glaucoma in end-stage renal disease patients who are on hemodialysis.

CONCLUSION

Glaucoma is one of the preventable causes of irreversible visual impairment, on diagnosis and treatment at early stages. Intraocular pressure and Ocular perfusion pressure has been shown to be known risk factors for open angle glaucoma.

The evidence is increasing that IOP , BP and ocular perfusion pressure instability may be associated with glaucoma. Variations in ocular perfusion pressure due to IOP and BP fluctuations may play a role in Normo-tension glaucoma development or progression.

Transient changes in intraocular pressure and ocular perfusion pressure during hemodialysis has been studied only by few studies

Our study found that there is transient increase in intraocular pressure to around 3mmHg during hemodialysis and transient decrease in ocular perfusion pressure during hemodialysis. At the end of this 2 year study period, 6 of the 58 patients were found to develop early glaucomatous field defects and early optic nerve head changes in both eyes at follow-up visits compared to normal fields and optic nerve head in their initial visits. Which comes around 10%

This study will help in better management of known glaucoma patients when they require hemodialysis. That way the impending ischemic insult of the optic nerve head can be avoided.

Chronic kidney disease patients with diabetes mellitus, systemic hypertension, glaucoma suspects, family history of glaucoma may benefit from intraocular pressure and blood pressure monitoring during a hemodialysis session.

Hence our study reveals the importance of screening and monitoring of intraocular pressure and characteristic early optic nerve head changes and early visual field changes of glaucoma in end-stage renal disease patients who are on hemodialysis. So doing a routine screening of IOP and glaucoma changes before initiation of hemodialysis therapy is reasonable and justifiable.

ANNEXURE

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PROFORMA

NAME:

AGE/SEX:

DATE:

IP/OP NO:

ADDRESS:

COMPLAINTS:

H/O CKD-

Duration :

stage:

GFR:

Treatment :

H/O other co-morbidities:

Diabetes milletus:

Hypertension:

Glaucoma :

OTHERS:

SYSTEMIC EXAMINATION:

Bp :

OCULAR EXAMINATION:

RE

LE

Vn

Vn PH

CONJUCTIVA:

CORNEA:

ANTERIOR CHAMBER

IRIS

PUPIL

LENS

FUNDUS

RE

LE

MEDIA

DISC

VESSELS

MACULA

BACKGROUND

DIAGNOSTIC TEST:

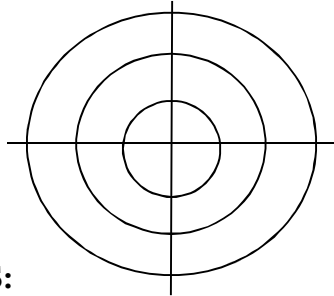
RE

LE

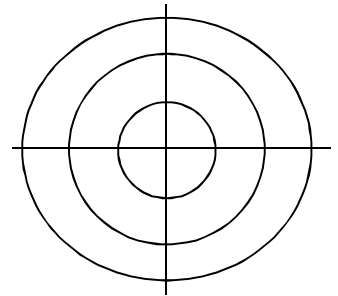
IOP

GONIO

PERIMETRY **LE**



RE



INVESTIGATIONS:

RBS:

Sr.urea:

Sr. electrolytes:

Sr.creatinine:

Total protein:

Sr.albumin

S.NO		T1	T2	T3
1.	IOP			
2.	BP			
3.	MAP			
4.	OPP			
5.	SOPP			
6.	DOPP			
7.	MOPP			

INFERENCE:

சுய ஒப்புதல் படிவம்.

தலைப்பு: இறுதி சிறுநீரக செயலிழப்பு நோய் இரத்த ஊடு பிரித்தல் போது விழி
செலுத்தல் அழுத்தம் மற்றும் கண்ணின் நீர் அழுத்தத்திற்கும் உடன் தொடர்பு
-ஓர் ஆய்வு

பங்கு பெறுபவரின் பெயர் -

பங்கு பெறுபவரின் எண் -

பங்கு பெறுபவர் இதனை (✓) குறிக்கவும் :

மேலே குறிப்பிடப்பட்டுள்ள இறுதி சிறுநீரக செயலிழப்பு நோய் இரத்த ஊடு பிரித்தல் போது விழியின் வழி செலுத்தல் அழுத்தம் மற்றும் கண்ணின் நீர் அழுத்தத்திற்கும் உடன் தொடர்பு கண்டறியும் ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வை சார்ந்து மேலும் ஆய்வுகள் மேற்கொள்ளப்படும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய் அறிகுறி தென்பட்டாலோ உடனே அதை மருத்துவ அணிக்கு தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்.

ABBREVIATIONS

S.NO	-	Serial Number
M	-	Male
F	-	Female
RE	-	Right Eye
LE	-	Left Eye
MMHG	-	Millimeters of mercury
CKD	-	Chronic Kidney Disease
HD	-	Hemodialysis
IOP	-	Intraocular Pressure
BP	-	Blood Pressure
DBP	-	Diastolic Blood Pressure
SBP	-	Systolic Blood Pressure
MAP	-	Mean Arterial Pressure
OPP	-	Ocular Perfusion Pressure

SOPP	-	Systolic Ocular Perfusion Pressure
DOPP	-	Diastolic Ocular Perfusion Pressure
MOPP	-	Mean Ocular Perfusion Pressure
ONH	-	Optic Nerve Head
POAG	-	Primary Open Angle Glaucoma
OAG	-	Open Angle Glaucoma
ARMD	-	Age Related Macular Degeneration
DM	-	Diabetes Mellitus
HT	-	Hypertension
PDUR	-	Post Dialysis Urea Rebound
RVA	-	Retinal Vessel Analyser
NSAIDs	-	Non- Steroidal Anti- Inflammatory Drugs
GAG	-	Glycosoaminoglycans
ATP	-	Adenosine Tri Phosphate
SVP	-	Systemic Venous Pressure
LALES	-	Los Angeles Latino Eye Study

MASTER CHART - I

S.NO	NAME	AGE /SEX	SYS. ILL	BP (mmHg)			IOP (mmHg)						MAP (mmHg)			MOPP (mmHg)						OPTIC NERVEHEAD		VISUAL FIELD	
							T1		T2		T3					T1		T2		T3					
				T1	T2	T3	RE	LE	RE	LE	RE	LE	T1	T2	T3	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
1.	Venkatesh	47/M	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	20.9	106.6	96.6	96.6	59.5	58.4	51.8	50.6	50.7	49.8	0.3	0.3	N	N
2.	VargheseThomas	34/M	-	130/80	120/80	120/80	14.6	14.3	15.9	14.3	15.9	14.3	96.6	93	93	54.6	54.8	51.4	52.4	51.1	52.1	0.3	0.3	N	N
3.	Kamala	51/F	SHT/DM2	140/90	130/80	130/80	18.9	17.3	20.6	18.9	21.9	20.6	106.6	96.6	96.6	54.8	59.5	50.6	51.8	49.8	50.7	0.3	0.3	N	N
4.	Sasikumar	30/M	-	120/80	110/70	110/70	18.9	18.9	20.6	20.9	20.9	20.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
5.	SanthoshKumar	35/M	-	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
6.	Vinothraj	45/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
7.	Samuvel Raja	50/M	DM2	120/80	110/70	100/70	18.9	17.3	19.6	18.9	20.1	20.1	93	83	80	49.4	50.4	42.0	42.7	39.9	39.9	0.3	0.3	N	N
8.	Jayalakshmi	46/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
9.	Rajamuthaiah	49/M	DM2/SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
10.	Gnanaprakasan	50/M	DM2	120/80	110/70	110/70	17.3	15.9	19.6	17.3	20.1	18.9	93	83	83	50.4	51.4	42.0	43.8	41.6	41.3	0.3	0.3	N	N
11.	Chinnapaiyan	50/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
12.	Kotti	45/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
13.	Vasanthi	47/F	DM2	130/80	120/80	120/80	14.6	14.3	15.9	14.3	15.9	14.3	96.6	93	93	54.6	54.8	51.4	52.4	51.1	52.1	0.3	0.3	N	N
14.	Silambarasan	43/M	-	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
15.	Kotteshwaran	46/M	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	20.9	106.6	96.6	96.6	59.5	58.4	51.8	50.6	50.7	49.8	0.3	0.3	N	N
16.	Sekar	40/M	-	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	20.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
17.	Lakshmanan	47/M	SHT	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	20.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
18.	Parthiban	24/M	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
19.	Savarimuthu	40/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
20.	Kannnan	44/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
21.	Murugan	46/M	DM2/SHT	120/80	110/70	120/80	14.6	15.9	14.6	15.9	14.6	15.9	93	83	93	52.2	51.4	45.6	44.7	52.2	51.4	0.3	0.3	N	N
22.	Thenmozhi	49/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
23.	Menaka	57/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
24.	Ganesh kumar	27/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
25.	Eswariya	55/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
26.	Dinesh	21/M	-	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
27.	Maruthai	50/M	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
28.	Narayanan	28/M	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
29.	Mohana	47/F	-	120/80	110/70	110/70	15.9	15.6	17.3	17.0	18.9	19.6	93	83	83	51.4	51.6	43.8	44.0	41.6	41.9	0.3	0.3	N	N
30.	Valli	50/F	-	120/80	120/80	120/80	15.9	17.3	15.9	17.3	15.9	17.3	93	93	93	51.4	50.4	51.4	50.4	51.4	50.4	0.3	0.3	N	N
31.	Anbu	49/M	-	120/80	110/70	110/70	17.3	15.6	18.9	17.0	20.6	19.6	93	83	83	50.4	51.6	41.9	44.0	41.3	41.9	0.3	0.3	N	N
32.	Mahalingam	43/M	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
33.	Ganesh	55/M	DM2/SHT	120/80	110/70	120/80	14.6	15.9	14.6	15.9	14.6	15.9	93	83	93	52.2	51.4	45.6	44.7	52.2	51.4	0.3	0.3	N	N
34.	Ramesh	30/M	-	120/80	110/70	110/70	17.3	15.9	19.6	17.3	20.1	18.9	93	83	83	50.4	51.4	42.0	43.8	41.6	41.3	0.3	0.3	N	N

S.NO	NAME	AGE /SEX	SYS. ILL	BP (mmHg)			IOP (mmHg)						MAP (mmHg)			MOPP (mmHg)						OPTIC NERVEHEAD		VISUAL FIELD	
				T1	T2	T3	T1		T2		T3		T1	T2	T3	T1		T2		T3		RE	LE	RE	LE
							RE	LE	RE	LE	RE	LE				RE	LE	RE	LE	RE	LE				
35.	Amelarpara mary	57/F	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
36.	Chandrasekar	25/M	-	130/80	120/80	120/80	14.6	14.3	15.9	14.3	15.9	14.3	96.6	93	93	54.6	54.8	51.4	52.4	51.1	52.1	0.3	0.3	N	N
37.	Mala	30/F	-	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
38.	Periyandi	62/M	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
39.	Chalopathy	40/M	DM2	120/80	110/70	110/70	15.9	15.6	17.3	17.0	18.9	19.6	93	83	83	51.4	51.6	43.8	44.0	41.6	41.9	0.3	0.3	N	N
40.	Sankaraselvi	40/F	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	96.6	59.5	58.4	51.8	50.6	50.7	49.8	0.3	0.3	N	N
41.	Padmavathi	42/F	DM2	120/80	110/70	120/80	14.6	15.9	14.6	15.9	14.6	15.9	93	83	93	52.2	51.4	45.6	44.7	52.2	51.4	0.3	0.3	N	N
42.	Banu	48/F	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
43.	Murugan	46/M	DM2/SHT	120/80	110/70	110/70	17.3	15.6	18.9	17.0	20.6	19.6	93	83	83	50.4	51.6	41.9	44.0	41.3	41.9	0.3	0.3	N	N
44.	MohamedImbran	57/M	DM2	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
45.	George joshva	39/M	DM2	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.4	0.4	N	N
46.	Savitha	30/F	-	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
47.	Narayanan	46/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
48.	Latha	30/F	-	120/80	120/80	120/80	15.9	17.3	15.9	17.3	15.9	17.3	93	93	93	51.4	50.4	51.4	50.4	51.4	50.4	0.3	0.3	N	N
49.	Murugan	45/M	DM2	120/80	110/70	110/70	15.9	15.6	17.3	17.0	18.9	19.6	93	83	83	51.4	51.6	43.8	44.0	41.6	41.9	0.3	0.3	N	N
50.	Ramesh	28/M	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	96.6	59.5	58.4	51.8	50.6	50.7	49.8	0.3	0.3	N	N
51.	Krishnamoorthy	52/M	DM2	120/80	110/70	110/70	17.3	15.6	18.9	17.0	20.6	19.6	93	83	83	50.4	51.6	41.9	44.0	41.3	41.9	0.3	0.3	N	N
52.	Shylaja	30/F	-	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
53.	Govindasamy	40/M	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
54.	Babitha dass	49/F	-	120/80	110/70	110/70	17.3	15.6	18.9	17.0	20.6	19.6	93	83	83	50.4	51.6	41.9	44.0	41.3	41.9	0.3	0.3	N	N
55.	Dhandapani	47/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
56.	Paramasivan	28/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
57.	Punitha	50/F	-	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
58.	Rubashree	21/F	DM/SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
59.	Athish	30/M	-	120/80	110/70	110/70	17.3	15.9	19.6	17.3	20.1	18.9	93	83	83	50.4	51.4	42.0	43.8	41.6	41.3	0.3	0.3	N	N
60.	Varalakshmi	42/F	DM2	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	96.6	59.5	58.4	51.8	50.6	50.7	49.8	0.3	0.3	N	N
61.	Vasuki	48/F	SHT	130/80	120/80	120/80	14.6	14.3	15.9	14.3	15.9	14.3	96.6	93	93	54.6	54.8	51.4	52.4	51.1	52.1	0.3	0.3	N	N
62.	Gomathi	43/F	-	140/90	130/80	130/80	18.9	17.3	20.6	18.9	21.9	20.6	106.6	96.6	96.6	54.8	59.5	50.6	51.8	49.8	50.7	0.3	0.3	N	N
63.	Ramu	32/M	SHT	120/80	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
64.	Kumar	47/M	-	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
65.	Manogaran	57/M	-	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
66.	Manikkam	57/M	SHT	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
67.	Saidhani	42/F	-	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
68.	Babarao	49/M	DM2	120/80	110/70	100/70	18.9	17.3	19.6	18.9	20.1	20.1	93	83	80	49.4	50.4	42.0	42.7	39.9	39.9	0.3	0.3	N	N
69.	Prakasam	50/m		120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
70.	Menaka	45/F	-	120/70	110/70	110/70	17.3	17.3	19.6	18.9	20.6	20.1	87	83	83	46.4	46.4	42.0	42.7	41.6	39.9	0.3	0.3	N	N
71.	Thenmozhi	50/F	-	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
72.	Vasanthi	45/F	SHT	120/80	110/70	110/70	15.9	15.6	17.3	17.0	18.9	19.6	93	83	83	51.4	51.6	43.8	44.0	41.6	41.9	0.3	0.3	N	N

S.NO	NAME	AGE /SEX	SYS. ILL	BP (mmHg)			IOP (mmHg)						MAP (mmHg)			MOPP (mmHg)						OPTIC NERVEHEAD		VISUAL FIELD	
				T1	T2	T3	T1		T2		T3		T1	T2	T3	T1		T2		T3		RE	LE	RE	LE
							RE	LE	RE	LE	RE	LE				RE	LE	RE	LE	RE	LE				
73.	Selvi	40/F	-	120/80	110/70	100/70	18.9	17.3	19.6	18.9	20.1	20.1	93	83	80	49.4	50.4	42.0	42.7	39.9	39.9	0.3	0.3	N	N
74.	Kannan	31/M	-	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
75.	Velu	46/M	DM2	120/80	110/70	110/70	17.3	15.6	18.9	17.0	20.6	19.6	93	83	83	50.4	51.6	41.9	44.0	41.3	41.9	0.3	0.3	N	N
76.	Annammal	52/F	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	93	59.5	58.4	51.8	50.6	48.2	47.4	0.3	0.3	N	N
77.	Jayalakshmi	55/F	DM2	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
78.	Williams Durai	38/M	DM2	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
79.	Vijaya	40/F	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
80.	Ramesh	40/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
81.	Forook	50/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
82.	Shenbagavalli	60/F	DM2/SHT	120/80	110/70	120/80	14.6	15.9	14.6	15.9	14.6	15.9	93	83	93	52.2	51.4	45.6	44.7	52.2	51.4	0.3	0.3	N	N
83.	Elizabeth	53/F	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
84.	Jothi	40/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
85.	Joseph	50/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
86.	Parveen	32/F	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
87.	Mohd. roshan	56/M	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
88.	Balaraman	48/M	SHT	120/80	110/70	110/70	15.9	15.6	17.3	17.0	18.9	19.6	93	83	83	51.4	51.6	43.8	44.0	41.6	41.9	0.3	0.3	N	N
89.	Lakshmiammal	53/F	DM/SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
90.	Punitha	50/F	DM2	120/80	110/70	110/70	17.3	15.9	19.6	17.3	20.1	18.9	93	83	83	50.4	51.4	42.0	43.8	41.6	41.3	0.3	0.3	N	N
91.	Kanagaraj	45/M	DM2	120/80	110/70	110/70	14.6	14.3	15.9	15.6	17.3	17.0	93	83	83	52.2	52.4	44.7	44.9	43.8	44	0.3	0.3	N	N
92.	Shankar	42/M	SHT	140/90	130/80	110/70	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	83	59.5	58.4	51.8	50.6	41.6	40.7	0.3	0.3	N	N
93.	Chindhanaiselvi	56/F	DM2	130/80	120/80	120/80	14.6	14.3	15.9	14.3	15.9	14.3	96.6	93	93	54.6	54.8	51.4	52.4	51.1	52.1	0.3	0.3	N	N
94.	Ramu	40/M	DM2	120/80	110/70	110/70	17.3	17.3	18.9	19.6	20.6	20.1	93	83	83	50.4	50.4	41.9	42.0	41.3	41.6	0.3	0.3	N	N
95.	Bhavani	46/F	SHT	140/90	130/80	130/80	17.3	18.9	18.9	20.6	20.6	21.9	106.6	96.6	93	59.5	58.4	51.8	50.6	48.2	47.4	0.3	0.3	N	N
96.	Jaikumar	53/M	DM2	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
97.	Marimuthu	57/M	DM2	120/70	110/70	110/70	18.9	18.9	20.6	21.9	21.9	21.9	93	83	83	49.4	49.4	41.6	40.7	40.7	40.7	0.3	0.3	N	N
98.	Gajalakshmi	45/F	SHT	150/100	140/90	130/80	17.3	18.9	18.9	18.9	20.6	20.6	116.6	106.6	96.6	66.2	65.1	58.4	58.4	50.6	50.6	0.3	0.3	N	N
99.	Malarkodi	51/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N
100.	Sudha	40/F	DM2	120/80	110/70	120/80	14.6	14.3	15.9	15.6	17.3	17.0	93	83	93	52.2	52.4	44.7	44.9	50.6	50.7	0.3	0.3	N	N

MASTER CHART - 2

S.N O	NAME	AGE /SEX	VISION		OPP (mmHg)						DOPP (mmHg)						SOPP (mmHg)					
					T1		T2		T3		T1		T2		T3		T1		T2		T3	
			RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
1.	Venkatesh	47/M	6/24	6/24	89.3	87.7	77.7	76	76	74.7	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
2.	VargheseThomas	34/M	6/24	6/24	82	82.3	77.1	78.7	77.1	78.7	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
3.	Kamala	51/F	6/36	6/18	87.7	89.3	76	77.7	74.7	76	71.1	72.7	59.4	61.1	58.1	59.4	121.1	122.7	109.4	111.1	108.1	109.4
4.	Sasikumar	30/M	6/12	6/12	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
5.	SanthoshKumar	35/M	6/6	6/6	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
6.	Vinothraj	45/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
7.	Samuvel Raja	50/M	6/12	6/12	74.1	75.7	63.4	64.1	59.9	59.9	61.1	62.7	50.4	51.1	49.9	49.9	101.1	102.7	90.4	91.1	79.9	79.9
8.	Jayalakshmi	46/F	6/24	6/24	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
9.	Rajamuthaiah	49/M	6/9	6/9	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
10.	Gnanaprakasan	50/M	6/12	6/12	75.7	77.1	63.4	65.7	62.9	64.1	62.7	64.1	50.4	52.7	49.9	51.1	102.7	104.1	90.4	92.7	89.9	91.1
11.	Chinnapaiyan	50/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
12.	Kotti	45/M	6/24	6/9	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
13.	Vasanthi	47/F	6/24	6/36	82	82.3	77.1	78.7	77.1	78.7	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
14.	Silambarasan	43/M	6/9	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
15.	Kotteshwaran	46/M	6/18	6/12	89.3	87.7	77.7	76	76	74.7	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
16	Sekar	40/M	6/9	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
17.	Lakshmanan	47/M	6/24	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
18.	Parthiban	24/M	6/18	6/12	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
19.	Savarimuthu	40/M	6/9	6/9	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
20.	Kannan	44/M	6/18	6/18	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
21.	Murugan	46/M	6/24	6/12	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	95.4	94.1	105.4	104.1
22.	Thenmozhi	49/F	6/24	6/24	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
23.	Menaka	57/F	6/12	6/18	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
24.	Ganesh kumar	27/M	6/6	6/9	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
25.	Eswaraiya	55/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
26.	Dinesh	21/M	6/9	6/9	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
27.	Maruthai	50/M	6/9	6/6	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
28.	Narayanan	28/M	6/18	6/12	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
29.	Mohana	47/F	6/18	6/18	77.1	77.4	65.7	66.0	64.1	63.4	64.1	64.4	52.7	53	51.1	50.4	104.1	104.4	92.7	93	91.1	90.4
30.	Valli	50/F	6/36	6/12	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	95.4	94.1	105.4	104.1
31.	Anbu	49/M	6/12	6/12	75.7	77.4	64.1	66.0	62.4	63.4	62.7	64.4	51.1	53	49.4	50.4	102.7	104.4	91.1	93	89.4	90.4
32.	Mahalingam	43/M	6/24	6/18	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
33.	Ganesh	55/M	6/24	6/24	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	95.4	94.1	105.4	104.1
34.	Ramesh	30/M	6/9	6/12	75.7	77.1	63.4	65.7	62.9	64.1	62.7	64.1	50.4	52.7	49.9	51.1	102.7	104.1	90.4	92.7	89.9	91.1

S.N O	NAME	AGE /SEX	VISION		OPP (mmHg)						DOPP (mmHg)						SOPP (mmHg)					
					T1		T2		T3		T1		T2		T3		T1		T2		T3	
			RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
35.	Amelarpara mary	57/F	6/12	6/9	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
36.	Chandrasekar	25/M	6/9	6/9	82	82.3	77.1	78.7	77.1	78.7	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
37.	Mala	30/F	6/6	6/6	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
38	Periyandi	62/M	6/9	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
39	Chalapathy	40/M	6/36	6/24	77.1	77.4	65.7	66.0	64.1	63.4	64.1	64.4	52.7	53	51.1	50.4	104.1	104.4	92.7	93	91.1	90.4
40.	Sankaraselvi	40/F	6/24	6/24	89.3	87.7	77.7	76	76	74.7	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
41.	Padmavathi	42/F	6/12	6/12	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	95.4	94.1	105.4	104.1
42.	Banu	48/F	6/12	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
43.	Murugan	46/M	6/12	6/12	75.7	77.4	64.1	66.0	62.4	63.4	62.7	64.4	51.1	53	49.4	50.4	102.7	104.4	91.1	93	89.4	90.4
44.	MohamedImbran	57/M	6/24	6/24	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
45.	George joshva	39/M	6/12	6/12	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
46	Savitha	30/F	6/6	6/6	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
47.	Narayanan	46/M	6/6	6/6	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
48.	Latha	30/F	6/12	6/12	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	95.4	94.1	105.4	104.1
49.	Murugan	45/M	6/36	6/24	77.1	77.4	65.7	66.0	64.1	63.4	64.1	64.4	52.7	53	51.1	50.4	104.1	104.4	92.7	93	91.1	90.4
50.	Ramesh	28/M	6/24	6/24	89.3	87.7	77.7	76	76	74.7	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
51.	Krishnamoorthy	52/M	6/12	6/12	75.7	77.4	64.1	66.0	62.4	63.4	62.7	64.4	51.1	53	49.4	50.4	102.7	104.4	91.1	93	89.4	90.4
52.	Shylaja	30/F	6/6	6/6	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
53.	Govindasamy	40/M	6/12	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
54.	Babitha dass	49/F	6/12	6/12	75.7	77.4	64.1	66.0	62.4	63.4	62.7	64.4	51.1	53	49.4	50.4	102.7	104.4	91.1	93	89.4	90.4
55.	Dhandapani	47/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
56.	Paramasivan	28/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
57.	Punitha	50/F	6/24	6/24	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	104.1	104.4	102.7	103
58.	Rubashree	21/F	6/9	6/9	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
59.	Athish	30/M	6/12	6/12	75.7	77.1	63.4	65.7	62.9	64.1	62.7	64.1	50.4	52.7	49.9	51.1	102.7	104.1	90.4	92.7	89.9	91.1
60.	Varalakshmi	42/F	6/24	6/24	89.3	87.7	77.7	76	76	74.7	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
61.	Vasuki	48/F	6/24	6/24	82	82.3	77.1	78.7	77.1	78.7	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
62.	Gomathi	43/F	6/36	6/18	87.7	89.3	76	77.7	74.7	76	71.1	72.7	59.4	61.1	58.1	59.4	121.1	122.7	109.4	111.1	108.1	109.4
63.	Ramu	32/M	6/12	6/12	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
64.	Kumar	47/M	6/6	6/6	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
65.	Manogaran	57/M	6/9	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
66.	Manikkam	57/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
67.	Saidhani	42/F	6/24	6/24	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	94.1	94.4	102.7	103
68.	Babarao	49/M	6/12	6/12	74.1	75.7	63.4	64.1	59.9	59.9	61.1	62.7	50.4	51.1	49.9	49.9	101.1	102.7	90.4	91.1	79.9	79.9
69.	Prakasam	50/m	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
70.	Menaka	45/F	6/9	6/9	69.7	69.7	63.4	64.1	62.4	59.9	52.7	52.7	50.4	51.1	49.4	49.9	102.7	102.7	90.4	91.1	89.4	89.9
71.	Thenmozhi	50/F	6/24	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
72.	Vasanthi	45/F	6/9	6/9	77.1	77.4	65.7	66.0	64.1	63.4	64.1	64.4	52.7	53	51.1	50.4	104.1	104.4	92.7	93	91.1	90.4

S.N O	NAME	AGE /SEX	VISION		OPP (mmHg)						DOPP (mmHg)						SOPP (mmHg)					
					T1		T2		T3		T1		T2		T3		T1		T2		T3	
			RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
73.	Selvi	40/F	6/12	6/12	74.1	75.7	63.4	64.1	59.9	59.9	61.1	62.7	50.4	51.1	49.9	49.9	101.1	102.7	90.4	91.1	79.9	79.9
74.	Kannan	31/M	6/12	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
75.	Velu	46/M	6/12	6/12	75.7	77.4	64.1	66.0	62.4	63.4	62.7	64.4	51.1	53	49.4	50.4	102.7	104.4	91.1	93	89.4	90.4
76.	Annammal	52/F	6/18	6/12	89.3	87.7	77.7	76	72.4	71.1	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
77.	Jayalakshmi	55/F	6/9	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
78.	Williams Durai	38/M	6/24	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
79.	Vijaya	40/F	6/18	6/12	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
80.	Ramesh	40/M	6/9	6/9	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
81.	Forook	50/M	6/18	6/18	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
82.	Shenbagavalli	60/F	6/24	6/12	78.4	77.1	68.4	67.1	78.4	77.1	65.4	64.1	55.4	54.1	65.4	64.1	105.4	104.1	105.4	104.1	105.4	104.1
83.	Elizabeth	53/F	6/12	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
84.	Jothi	40/F	6/12	6/18	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	104.1	104.4	102.7	103
85.	Joseph	50/M	6/6	6/9	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
86.	Parveen	32/F	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
87.	Mohd. roshan	56/M	6/9	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
88.	Balaraman	48/M	6/9	6/9	77.1	77.4	65.7	66.0	64.1	63.4	64.1	64.4	52.7	53	51.1	50.4	104.1	104.4	92.7	93	91.1	90.4
89.	Lakshmiammal	53/F	6/36	6/18	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
90.	Punitha	50/F	6/12	6/12	75.7	77.1	63.4	65.7	62.9	64.1	62.7	64.1	50.4	52.7	49.9	51.1	102.7	104.1	90.4	92.7	89.9	91.1
91.	Kanagaraj	45/M	6/24	6/36	78.4	78.7	67.1	67.4	65.7	66	65.4	65.7	54.1	54.4	52.7	53	105.4	105.7	94.1	94.4	92.7	93
92.	Shankar	42/M	6/24	6/9	89.3	87.7	77.7	76	62.4	61.1	72.7	71.1	61.1	59.4	49.4	48.1	122.7	121.1	111.1	109.4	89.4	88.1
93.	Chindhanaiselvi	56/F	6/24	6/36	82	82.3	77.1	78.7	77.1	78.7	65.4	65.7	64.1	65.7	64.1	65.7	115.4	115.7	104.1	105.7	104.1	105.7
94.	Ramu	40/M	6/9	6/12	75.7	75.7	64.1	63.4	62.4	62.9	62.7	62.7	51.1	50.4	49.4	49.9	102.7	102.7	91.1	90.4	89.4	89.9
95.	Bhavani	46/F	6/18	6/12	89.3	87.7	77.7	76	72.4	71.1	72.7	71.1	61.1	59.4	59.4	58.1	122.7	121.1	111.1	109.4	109.4	108.1
96.	Jaikumar	53/M	6/9	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
97.	Marimuthu	57/M	6/24	6/18	74.1	74.1	62.4	61.1	61.1	61.1	61.1	61.1	49.4	48.1	48.1	48.1	101.1	101.1	89.4	88.1	88.1	88.1
98.	Gajalakshmi	45/F	6/18	6/12	99.3	97.7	87.7	87.7	76	76	82.7	81.1	71.1	71.1	59.4	59.4	132.7	131.1	121.1	121.1	109.4	109.4
99.	Malarkodi	51/F	6/24	6/24	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	104.1	104.4	102.7	103
100.	Sudha	40/F	6/12	6/18	78.4	78.7	67.1	67.4	75.9	76	65.4	65.7	54.1	54.4	62.7	63.0	105.4	105.7	101.1	102.4	102.7	103

