A DISSERTATION ON

# "RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA"

Submitted to

**THE TAMIL NADU DR. M. G. R. MEDICAL UNIVERSITY**

In partial fulfilment of the requirements

For the award of degree of

**M.S. (Branch III) --- OPHTHALMOLOGY**

**MAY 2020**

# CERTIFICATE

This is to certify that the study entitled **"RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA"** is the result of original work carried out by **DR.A.REVATHY**, under my supervision and guidance at **GOVERNMENT 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, course from 2017 to 2020 at Government Stanley Medical College, Chennai.

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## CERTIFICATE FROM GUIDE

This is to certify that the dissertation entitled, **"RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA"**is a bonafide record of Research work done by **DR.A.REVATHY**, Post Graduate Resident in Department Of Ophthalmology, Government Stanley Medical College Chennai.

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# DECLARATION

I hereby declare that this dissertation entitled "**RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA**" is a bonafide and genuine research work carried out by me under the guidance of **PROF. DR. THANGERANI RAAJASEHARAN M.S.D.O.,** Head of the Department, Department of Ophthalmology, Government Stanley Medical college and Hospital, Chennai –01.

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| Title of the Work | : | "RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA" |
| Principal Investigator | : | Dr. A. Revathy, |
| Designation | : | Postgraduate in Ophthalmology (2017-2020), Department of Ophthalmology, Govt. Stanley medical college & hospital. Chennai. |

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 17.02.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

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This is to certify that this dissertation work titled "**RELATIONSHIP BETWEEN SYSTEMIC HYPERTENSION, OCULAR PERFUSION PRESSURE AND GLAUCOMA"** of the candidate **DR.A.REVATHY** with registration number **221713054** for the award of **M.S DEGREE** in the branch of **Ophthalmology**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and the result shows 8% of plagiarism in the dissertation.

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# ABBREVIATIONS

| | | |
|---|---|---|
| **IOP** | **–** | Intraocular pressure |
| **JNC** | **-** | Joint National Committee |
| **ONH** | – | Optic Nerve Head |
| **BP** | - | Blood Pressure |
| **OPP** | - | Ocular Perfusion Pressure |
| **RGC** | – | Retinal ganglion cell |
| **BF** | – | Blood Flow |
| **SBP** | – | Systolic blood pressure |
| **DBP** | – | Diastolic blood pressure |
| **MAP** | – | Mean Arterial pressure |
| **MOPP** | – | Mean Ocular Perfusion Pressure |
| **SPP** | – | Systolic Perfusion Pressure |
| **DPP** | – | Diastolic Perfusion Pressure |
| **GAT** | – | Goldmann Appalanation Tonometry |
| **NTG** | - | Normal Tension Glaucoma |
| **AR Blockers** | - | Angiotensin Receptor blockers |
| **CC Blockers** | - | Calcium Channel blockers |
| **ACE** | – | Angiotensin Converting Enzyme |

# PART I

## INTRODUCTION

- The prevalence of hypertension is estimated to range from 12-17% among rural adults and 20-40% in urban adults.(1)

- Hypertensive retinopathy refers to changes in the retinal vasculature associated with elevated blood pressure.

- Among the different retinal signs observed, focal arteriolar narrowing and arteriovenous nicking accounts for 7% to 12% in hypertensive people, respectively and the most common retinopathy lesions observed are isolated retinal hemorrhages (3%-17%)and cottonwool spots being (0.3%) (2)

- Glaucoma is a family of multifactorial optical neuropathies characterized by loss of retinal ganglion cells (RGCs) leading to typical optic nerve head (ONH) damage and distinctive visual field defects.

- As an Ophthalmologist we can record Intraocular Pressure easily.

- It has been hypothesized that a vascular component is involved in glaucoma pathophysiology.

- In order to better assess glaucoma risk, several markers can be used one of which is **OCULAR PERFUSION PRESSURE**, a potential marker of Optic Nerve Head Perfusion and the major culprit mechanism in causing glaucoma.

- Several reports have demonstrated an association between Hypertension and Ocular perfusion pressure.

Thus our study aims to determine the association between Systemic Hypertension, Ocular Perfusion Pressure and Glaucoma.

## NEED FOR STUDY:

Hypertension is one of the leading causes of morbidity in developing country like India. Glaucoma being one of the leading cause of blindness worldwide, is a multifactorial disease. Intraocular pressure (IOP) is one entity that decides development and progression of glaucoma as well as plays a role in ocular perfusion .Blood pressure also plays a major role in determining the ocular perfusion pressure. So it is prudent to screen the high risk population and prevent complication of both the diseases at the earliest by recording IOP.

Retinopathy is a well known complication of hypertension and is routinely screened nowadays. Thus as an ophthalmologist, it is our responsibility to move one step further and screen all hypertensive patients for other complications due to hypertension .By simply measuring intraocular pressure and recording blood pressure we can calculate OCULAR PERFUSION PRESSURE and it enables us to identify patients at risk for progressing to glaucoma.

Thus our study was done to find the actual association between SYSTEMIC HYPERTENSION , OCULAR PERFUSION PRESSURE and GLAUCOMA.

## HYPERTENSION:

Hypertension - one of the most common vascular diseases in the developing world is characterized by a persistent pathologic elevation of arterial pressure and increased total peripheral resistance leading to vascular complications in the brain, heart, kidneys and eyes.

The prevalence of hypertension is 35-40% of the general population worldwide.Overall prevalence for hypertension in India is 29.8%.

The pooled prevalence of hypertension for the rural and urban south Indian population is 21.1% and 31.8% respectively.(1)

The increasing prevalence of hypertension due to a stressful and unhealthy lifestyle can pose a great public health burden.

**JNC 8 (JOINT NATIONAL COMMITTEE) guidelines**(3)**:**

**Figure 1**

| BP CLASSIFICATION | SBP* MMHG | DBP* MMHG |
|---|---|---|
| NORMAL | <120 | and <80 |
| PREHYPERTENSION | 120–139 | or 80–89 |
| STAGE 1 HYPERTENSION | 140–159 | or 90–99 |
| STAGE 2 HYPERTENSION | ≥160 | or ≥100 |

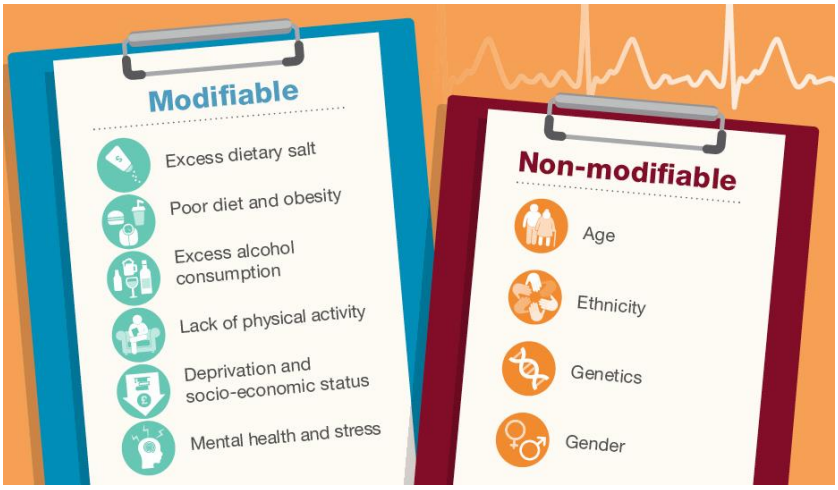
DBP = Diastolic Blood Pressure, SBP = Systolic Blood Pressure
*Treatment should be determined by the highest blood pressure
‡Treat patients with chronic kidney disease or diabetes to BP goal of <130/80mmHg

The JNC on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure lists **retinopathy** as one of several markers of target-organ damage in hypertension.

On the basis of the JNC criteria, the presence of **retinopathy** may be an indication for initiating anti**hypertensive** treatment, even in people with stage 1 hypertension (blood pressure, 140 to 159/90 to 99 mm Hg) who have no other evidence of target-organ damage.

## RISK FACTORS OF HYPERTENSION:

**Figure 2**




## TYPES OF HYPERTENSION:

The two main types of hypertension are

1. **Primary (Essential)** – most common type and has no cause
2. **Secondary** – caused by disorders like renal disease, Cushing's syndrome, pheochromocytoma, aldosterone excess etc

OTHER CLINICAL TYPES:

ACCELERATED HYPERTENSION

- Clinically identified by severe retinopathy (without papilloedema) – characterised by exudates, hemorrhages, arteriolar narrowing, and spasm.

MALIGNANT HYPERTENSION

- Considered a deterioration of the accelerated form and is distinguished by papilloedema.

Both the accelerated and malignant forms of hypertension are associated with severe vascular injury to the kidney and other target organs.

RESISTANT HYPERTENSION

- Patients with systolic blood pressure persistently >/= 140mmHg despite taking three or more anti-hypertensive medications including a diuretic in a reasonable combination and at full doses.
- More common in patients > 60 years
- Related to "pseudo-resistance" – higher office and lower home blood pressures, non adherence to therapy, obesity, excess alcohol intake
- Also due to sclerotic arteries – inability to measure blood pressure accurately in older patients: suggested if the radial pulse remains palpable despite brachial artery occlusion by the cuff (Osler manoeuvre)

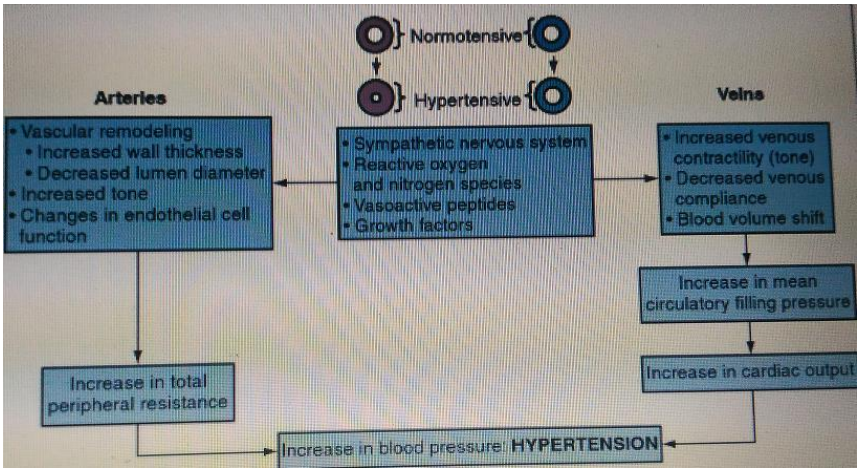
## CLINICAL TYPES OF HYPERTENSION:

- **HYPERTENSION WITH INVOLUTIONARY SCLEROSIS →** In older patients
- **HYPERTENSION WITHOUT SCLEROSIS →** in young individuals

  Exposed to increased BP for a short duration
- **HYPERTENSION WITH COMPENSATORY ARTERIOLAR SCLEROSIS →** In young patients exposed to increased BP for a longer duration, associated with benign nephrosclerosis (RENAL RETINOPATHY)
- **MALIGNANT HYPERTENSION →** Rapidly progressive, severe fundus changes (GRADE 4 retinopathy changes)

## PATHOGENESIS OF HYPERTENSION:

**Figure 3**



## CLINICAL FEATURES IN HYPERTENSION:

- Acute malignant hypertension – blurring of vision, hypertensive headache which typically occurs in the morning and in the occipital region.
- Chronic hypertension with arteriosclerotic changes – generally asymptomatic

## DIAGNOSIS OF HYPERTENSION:

Based on

RELEVANT HISTORY:

1. Medication history (as some drugs may have increased BP as side effect)
2. Dietary and psychosocial history
3. Family history of hypertension and cardiovascular disease
4. History of dyslipidemia, smoking, diabetes, physical inactivity
5. Evidence of secondary hypertension: history of renal disease, muscle weakness, sweating, palpitations, tremors, sleep disturbances, hypo / hyperthyroidism
6. Evidence of target organ damage: history of TIA, stroke, transient blindness, , myocardial infarction, congestive heart failure
7. History of other co-morbidities

BLOOD PRESSURE RECORDING: (3)

(1) Blood pressure should be measured after a 5 min period of rest with feet resting on the floor, in a comfortable room temperature[23]

(2) A sphygmomanometer that has been validated, well maintained and calibrated to be used, atleast 2 readings should be taken

**Figure 4**

(3) Centre of the cuff to be at heart level

(4) Width of the cuff should be atleast 40% of the arm circumference[23]

(5) Length of the cuff should encircle atleast 80% of arm circumference

(6) Rate of deflation to be 2mmHg/s[23]

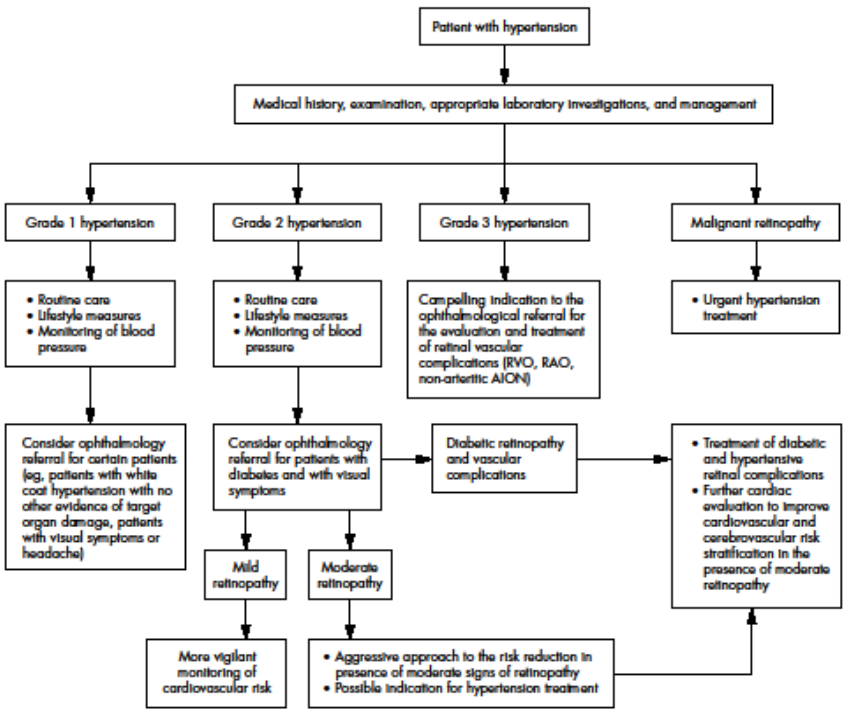
(7) Systolic BP – first of atleast 2 regular tapping Korotkoff sounds, Diastolic BP – point at which the last regular Korotkoff sound heard

## TREATMENT OF HYPERTENSION:

## LIFESTYLE MODIFICATIONS

| | |
|---|---|
| WEIGHT REDUCTION | Attain and maintain BMI < 25kg/ square metre, Waist circumference < 102 cm in men, < 88 cm women |
| DIETARY SALT RESTRICTION | < 6g/day |
| ADAPT DASH-TYPE DIET PLAN<br>(DASH – dietary approach to stop hypertension) | Diet rich in fruits , vegetables, low fat- diary products with reduced content of saturated and total fat |
| MODERATION OF ALCOHOL CONSUMPTION | </= 2 drinks/ day in men<br></=1 drink/ day in women |
| SMOKING CESSATION | |
| PHYSICAL ACTIVITY | Regular aerobic activity – brisk walking for 30 min/day, 6-7 days/ week |

**Figure 5**



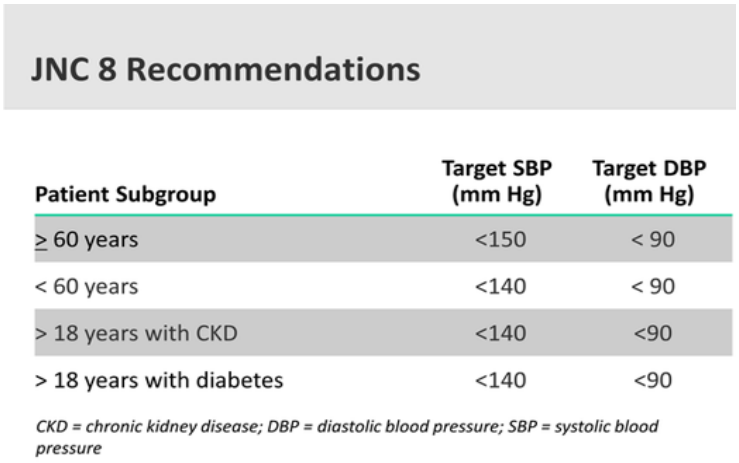
**Management of hypertension**

In malignant hypertension (HYPERTENSIVE EMERGENCY)

Hypertensive emergencies should preferably be managed in an INTENSIVE CARE UNIT to allow for continuous monitoring of the general hemodynamic status of the patient.

- The initial goal for BP reduction is to achieve a progressive, controlled reduction in BP to minimize the risk of hypoperfusion in cerebral, coronary, and renovascular beds.
- Under normal conditions, blood flow to these organs remains relatively constant despite wide fluctuations in BP. In the presence of severe hypertension, the autoregulatory range is shifted upward so that higher levels of BP are tolerated, hence sudden BP reduction can put the organ circulation at risk.
- Hence it is suggested that initial reduction in mean arterial pressure should not exceed 20% to 25% below the pre-treatment BP. A reasonable goal for most hypertensive emergencies is to lower the diastolic BP to 100 mm Hg over a period of minutes to hours in MALIGNANT HYPERTENSION WITH ENCEPHALOPATHY
- In MALIGNANT HYPERTENSION WITHOUT ENCEPHALOPATHY Can be reduced over a period of hours.

**Figure 6**




## Classification of Anti-Hypertensives

| | |
|---|---|
| **Diuretics** | Thiazides - Hydrochlorothiazide,Chlorthalidone<br>High Ceiling - Frusemide<br>K+ Sparing - Spironolactone,Triamterene,Amiloride |
| **ACE Inhibitors** | Captopril,Lisinopril,Enalapril,Ramipril,Fosinopril |
| **ACE Receptor Blockers** | Losartan,Candesartan,Telmesartan,Valsartan |
| **Direct Renin Inhibitor** | Aliskrien |
| **Calcium channel blockers** | Verapamil,Diltiazem,Nifedipine,Amlodipine |
| **Beta adrenergic blockers** | Non selective - Propanolol<br>Cardio-selective – Atenolol,Metaprolol |
| **Beta & alpha adrenergic blockers** | Labetalol,Carvedilol |
| **Alpha adrenergic blockers** | Prazosin,Terazosin,Doxazosin,Phentolamine |
| **Centrally Acting** | Clonidine,Methyldopa |

## Parenteral Agents for Hypertensive Emergencies

**Figure 7**

| Parenteral Drug | Mechanism of action | Onset of action | Duration of action | Infusion rate |
|---|---|---|---|---|
| Sodium Nitroprusside | Direct arterial and venous dilator | Less than 2 minutes | 1-10 minutes | Initial dose is 0.5 µg/kg/minute and the flow rate is increased in increments of 1 µg/kg/minute every 2 to 3 minutes |
| Fenoldopam | Dopamine agonist causing decreased SVR | Within 10 minutes | Up to 1 hour | Initial dose is 0.1 µg/kg/minute, the increments must not exceed 0.1 µg/kg/minute at 20-minute intervals |
| Labetolol | Non selective β- and α1-adrenergic blocker | 5-10 minutes | 2.5-6.5 hours | Initial dose of 20 mg bolus over 2 minutes with 20 mg increments every 10 minutes interval to maximum of 300 mg |
| Esmolol | Cardioselective β-blocker resulting in decreased cardiac output | Within 60 seconds | 10-20 minutes | 500-1000 µg/kg loading dose over 1 minute, followed by an infusion starting at 50 µg/kg/minute and increasing up to 300 µg/kg/minute |
| Clevidipine | Calcium channel blocker causing decreased SVR | 2-4 minutes | 5-15 minutes | Initial dose is 1 to 2 mg/h and then titrated (usually doubled every 90 seconds) |
| Nicardipine | Calcium channel blocker causing decreased SVR | 5-10 minutes | 4-6 hours | Initial infusion rate of 5 mg/hour, increasing by 2.5 mg/hour every 5 minutes to a maximum of 15 mg/hour |
| Nitroglycerine | Venodilator | 2-5 minutes | 10-15 minutes | 5 mcg/minute, increase by 5 mcg/minute every 3-5 minutes upto 200 mcg/minute. |
| Phentolamine | Nonselective α-adrenergic blocker | 2-3 minutes | 15-30 minutes | Initial dose is 1 mg bolus with subsequent boluses of 1 to 5 mg up to a total of dose of 20 to 30 mg |

## HYPERTENSIVE RETINOPATHY

It is a bilateral, symmetrical, small blood vessel disease characterized by a spectrum of retinal vascular signs in people with elevated BP, and is an end organ manifestation.

Hypertensive retinopathy prevalence is high in both males and females. Incidence increases with duration of the disease.

Elevation of systemic BP causes both focal and generalised retinal arteriolar constriction mediated by autoregulation. This may be associated with breakdown of inner blood- retinal barrier leading to pathological changes of hypertensive retinopathy.

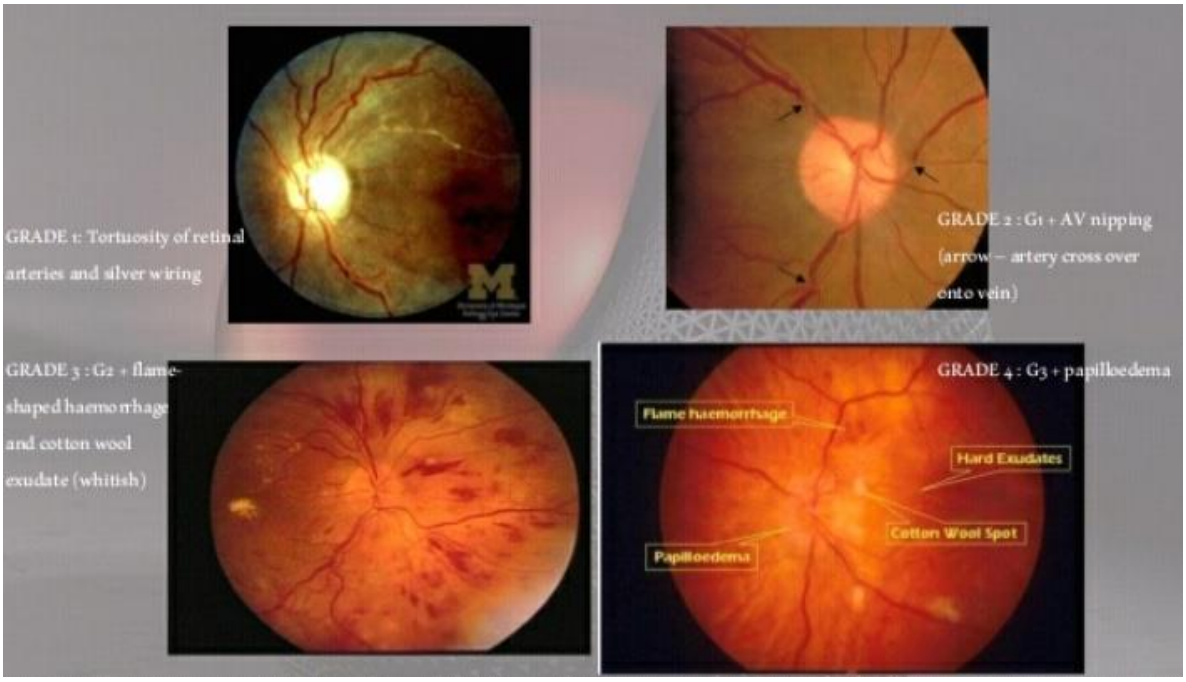
## CLASSIFICATION OF HYPERTENSIVE RETINOPATHY:

### KEITH- WAGNER- BARKER CLASSIFICATION (1939)

| | | |
|---|---|---|
| Grade 1 | Minimal constriction of retinal arterioles with some tortuosity | No symptoms |
| Grade 2 | Moderate to marked arteriolar narrowing, Arteriovenous crossing changes, minimal or no systemic involvement | Asymptomatic |
| Grade 3 | Moderate to marked arteriolar narrowing, Arteriovenous crossing changes<br>+ retinal edema, haemorrhages, cotton wool spots<br>Identifiable cardiac, renal or cerebral dysfunction | Symptomatic |
| Grade 4 | Grade 3 + papilloedema<br>Severe cerebral, cardiac, renal dysfunction | Reduced survival |

**Grading of hypertensive retinopathy:**

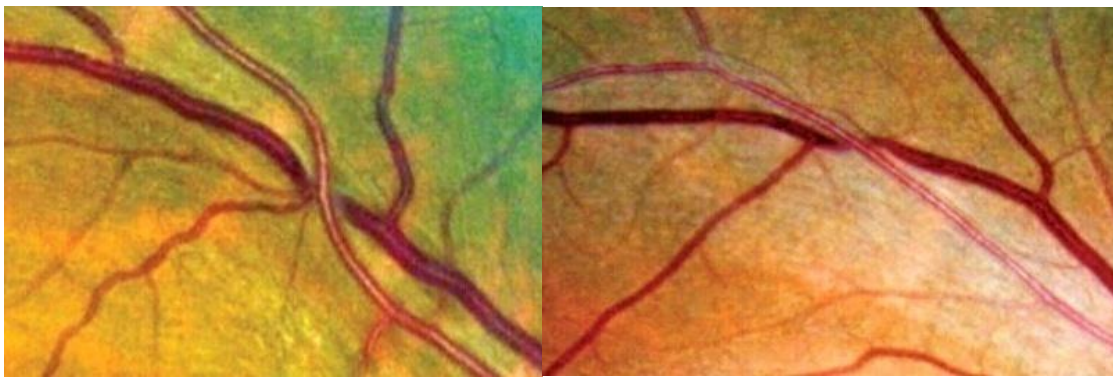
**Figure 8**




**AV CROSSING CHANGES:**

**Gunn's sign:** arteriolovenous nicking, tapering of veins on either side of arteriovenous crossing

**Figure 9**



**Salu's sign** – deflection of venule at arteriovenous crossing

**Bonnet sign** – banking of veins distal to arteriovenous crossing

## EFFECTS OF SYSTEMIC HYPERTENSION ON THE POSTERIOR SEGMENT OF THE EYE:

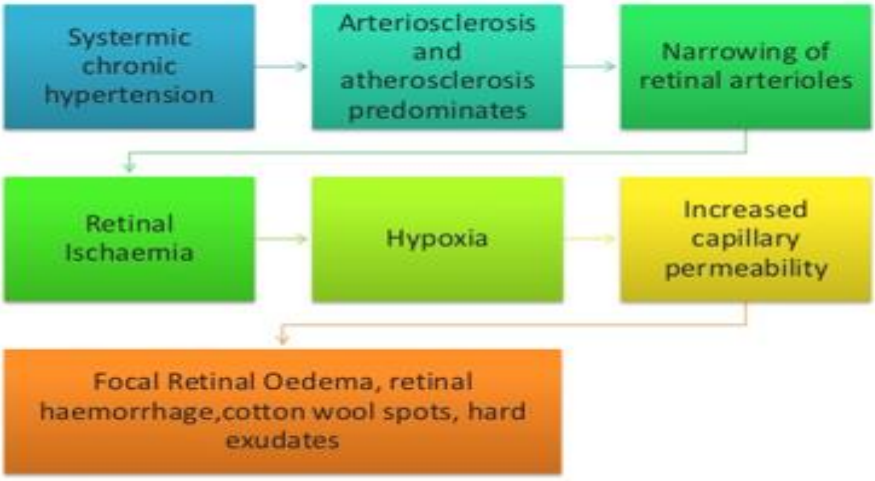
1) HYPERTENSIVE RETINOPATHY, HYPERTENSIVE CHOROIDOPATHY
2) OPTIC NERVE ISCHEMIA
3) RETINAL VENOUS, ARTERIAL OCCLUSIONS, ARTERIOLAR EMBOLI
4) RETINAL MACROANEURYSM

## RELATIONSHIP OF HYPERTENSIVE RETINOPATHY WITH BLOOD PRESSURE:

- ✓ The patterns of specific retinal vascular changes vary with current and past blood pressure levels. Generalized retinal arteriolar narrowing and arteriovenous nicking usually appear in patients with long-term hypertension and are independently associated with past blood pressure levels measured up to 10 years before retinal assessment.[25]

- ✓ In contrast, focal arteriolar narrowing and retinopathy lesions (retinal hemorrhages, microaneurysms, and cotton wool spots) may indicate more transitory blood pressure changes and are related only to concurrently measured blood pressure.

**Figure 10**


Systermic chronic hypertension
Arteriosclerosis and atherosclerosis predominates
Narrowing of retinal arterioles
Retinal Ischaemia
Hypoxia
Increased capillary permeability
Focal Retinal Oedema, retinal haemorrhage,cotton wool spots, hard exudates

# GLAUCOMA

Glaucoma is not a single disease process but a group of disorders characterized by a progressive optic neuropathy resulting in a characterstic appearance of the optic disc and a specific pattern of irreversible visual field defects that are associated frequently but not invariably with raised intraocular pressure (IOP). Thus, IOP is the most common risk factor but not the only risk factor for development of glaucoma.

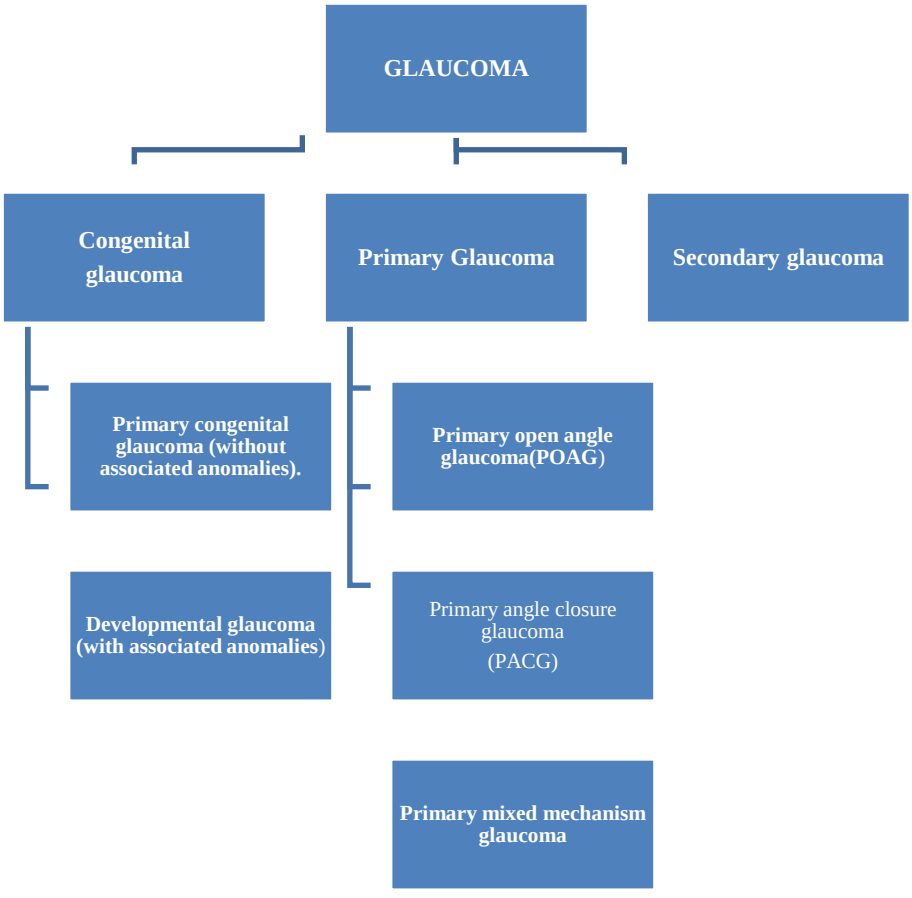
**Ocular hypertension** - cases having constantly raised IOP without any associated glaucomatous damage.

**Normal or low tension glaucoma (NTG/LTG) -** typical cupping of the disc and/or visual field defects associated with a normal or low IOP.

## Classification

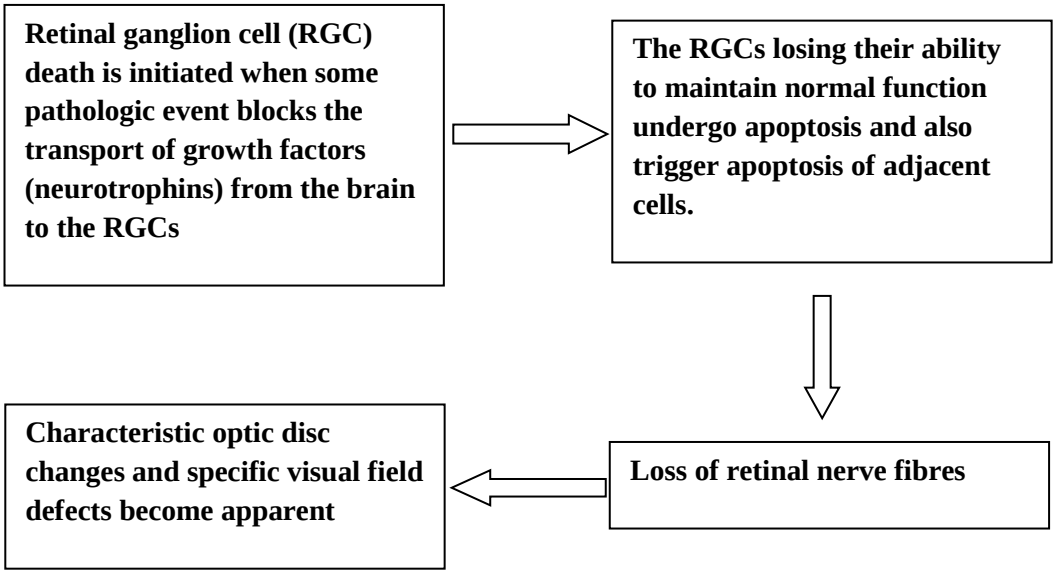
Clinico-etiologically glaucoma may be classified as follows:

**Figure 11**



## PATHOGENESIS OF GLAUCOMATOUS OCULAR DAMAGE

All glaucomas are characterized by a progressive optic neuropathy. Progressive optic neuropathy results from the death of retinal ganglion cells (RGCs) in a typical pattern which results in characteristic optic disc appearance and specific visual field defects. .




**Factors involved in the etiology of retinal ganglion cell death and thus in the etiology of glaucomatous optic neuropathy can be grouped as (7):**

**A. Primary insults –**

**1. Raised intraocular pressure (Mechanical theory).**

Raised intraocular pressure causes mechanical stretch on the lamina cribrosa leading to axonal deformation and ischaemia by altering capillary blood flow. As a result of this, neurotrophins (growth factors) are not able to reach the retinal ganglion cell bodies in sufficient amount needed for their survival.

**2. Pressure independent factors (Vascular insufficiency theory)**(4):

Factors affecting vascular perfusion of optic nerve head in the absence of raised IOP have been implicated in the glaucomatous optic neuropathy in patients with normal tension glaucoma (NTG). However, these may be the additional factors in cases of raised IOP as well.

These factors include:

i. **Failure of autoregulatory mechanism of blood flow**.

The retina and optic nerve share a peculiar mechanism of autoregulation of blood flow with rest of the central nervous system. Once the autoregulatory mechanisms are compromised, blood flow may not be adequate beyond some critical range of IOP (which may be raised or in normal range).

ii. **Vasospasm**

It is another mechanism affecting vascular perfusion of optic nerve head. This hypothesis gets credence from the convincing association between NTG and vasospastic disorders (migranous headache and Raynaud's phenomenon).

iii. **Systemic hypotension**

It is particularly nocturnal dips in patients with night time administration of antihypertensive drugs has been implicated for low vascular perfusion of optic nerve head resulting in NTG

iv. **Other factors** such as acute blood loss and abnormal coagulability profile have also been associated with NTG.

**Pathophysiology of glaucoma revolves around the aqueous humour dynamics.**

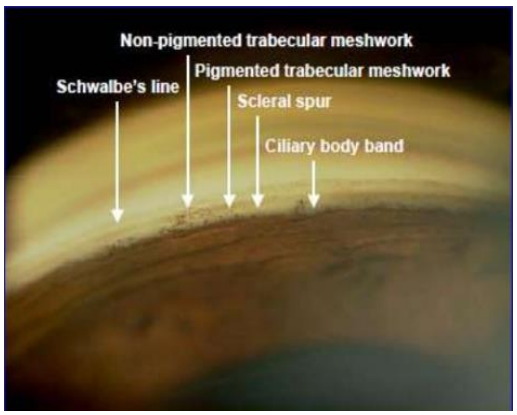
The principal ocular structures concerned with it are ciliary body, angle of anterior chamber and the aqueous outflow system. (5)

**Ciliary body** - It is the seat of aqueous production.

**Angle of anterior chamber** - important role in the process of aqueous drainage

Angle of anterior chamber is formed by: root of iris, anterior-most part of ciliary body, scleral spur, trabecular meshwork Schwalbe's line (prominent end of Descemet's membrane of cornea)

**Figure 12**




| Grade | Angle width | configuration | Chances of closure | Structure visible on gonioscopy |
|---|---|---|---|---|
| IV | 35-45° | Wide open | Nil | Schwalbes line to ciliary body |
| III | 20-35° | Open | Nil | Schwalbes line to scleral spur |
| II | 20° | Moderately narrow | Closure possible | Schwalbes line to TM |
| I | 10° | Very narrow | High | Schwalbes line only |
| 0 | 0° | Closed | closed | none |

The angle width varies in different individuals and plays a vital role in the pathomechanism of different types of glaucoma. Clinically the angle structures can be visualised by gonioscopic examination

**Gonioscopic grading of the angle width:** Various systems have been suggested to grade angle width.

The most commonly used **Shaffer's system of grading** the angle is given in Aqueous outflow system.

It includes the trabecular meshwork, Schlemm's canal, collector channels, aqueous veins and the episcleral veins.

**1. Trabecular meshwork** : It is a sieve-like structure through which aqueous humour leaves the eye. It consists of three portions.

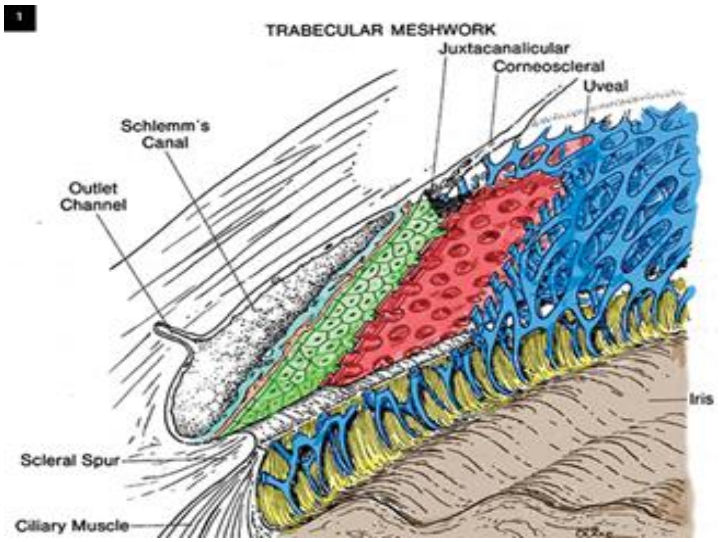
i. **Uveal meshwork**

ii. **Corneoscleral meshwork**:

iii. **Juxtacanalicular (endothelial) meshwork**:.

This part of trabecular meshwork mainly offers the normal resistance to aqueous outflow.

**Figure 13**



**2. Schlemm's canal:** This is an endothelial lined oval channel present circumferentially in the scleral sulcus. The endothelial cells of its inner wall are irregular, spindle-shaped and contain giant vacuoles. The outer wall of the canal is lined by smooth flat cells and contains the openings of collector channels.

**3. Collector channels:** These, also called intrascleral aqueous vessels, are about 25-35 in number and leave the Schlemm's canal at oblique angles to terminate into episcleral veins in a laminated fashion. These intrascleral aqueous vessels can be divided into two systems (Fig. 9.3A). The larger vessels (aqueous veins) run a short intrascleral course and terminate directly into episcleral veins (direct system). Many smaller collector channels form an intrascleral plexus before eventually going into episcleral veins (indirect system).

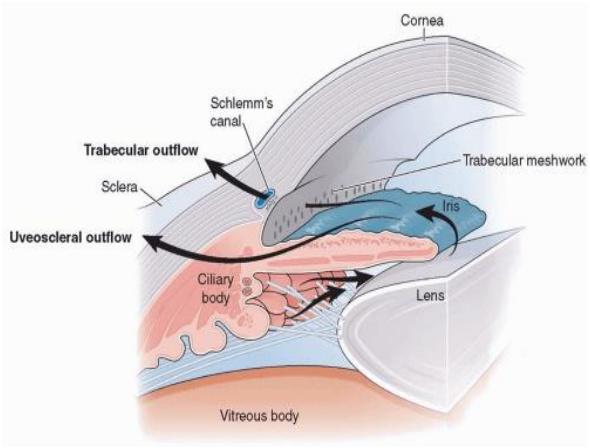
**Drainage of aqueous humour** Aqueous humour flows from the posterior chamber into the anterior chamber through the pupil against slight physiologic resistance. From the anterior chamber the aqueous is drained out by two routes:

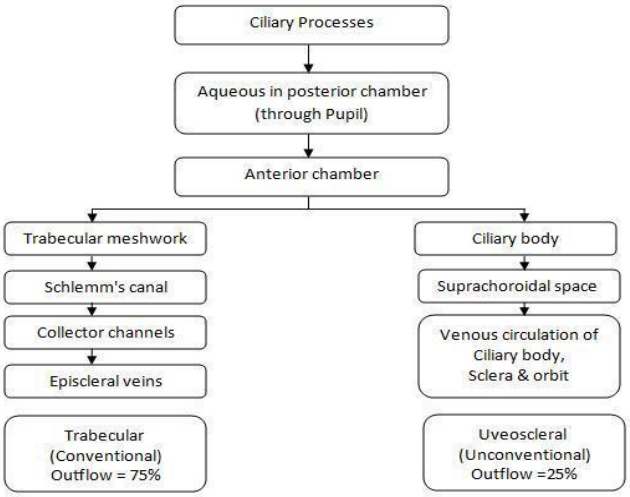
**1. Trabecular (conventional) outflow**: Trabecular meshwork is the main outlet for aqueous from the anterior chamber. Approximately 90 percent of the total aqueous is drained out via this route. Free flow of aqueous occurs from trabecular meshwork up to inner wall of Schlemm's canal which appears to provide some resistance to outflow.

## Mechanism of aqueous transport across inner wall of Schlemm's canal

Vacuolation theory is the most accepted view. According to it, transcellular spaces exist in the endothelial cells forming inner wall of Schlemm's canal. These open as a system of vacuoles and pores, primarily in response to pressure, and transport the aqueous from the juxtacanalicular connective tissue to Schlemm's canal. From Schlemm's canal the aqueous is transported via 25-35 external collector channels into the episcleral veins by direct and indirect systems A pressure gradient between intraocular pressure and intrascleral venous pressure (about 10 mm of Hg) is responsible for unidirectional flow of aqueous.

**Figure 14**



**2. Uveoscleral (unconventional) outlow:** It is responsible for about 10 percent of the total aqueous outflow. Aqueous passes across the ciliary body into the suprachoroidal space and is drained by the venous circulation in the ciliary body, choroid and sclera.

## RECORDING OF IOP:

### Types of tonometry

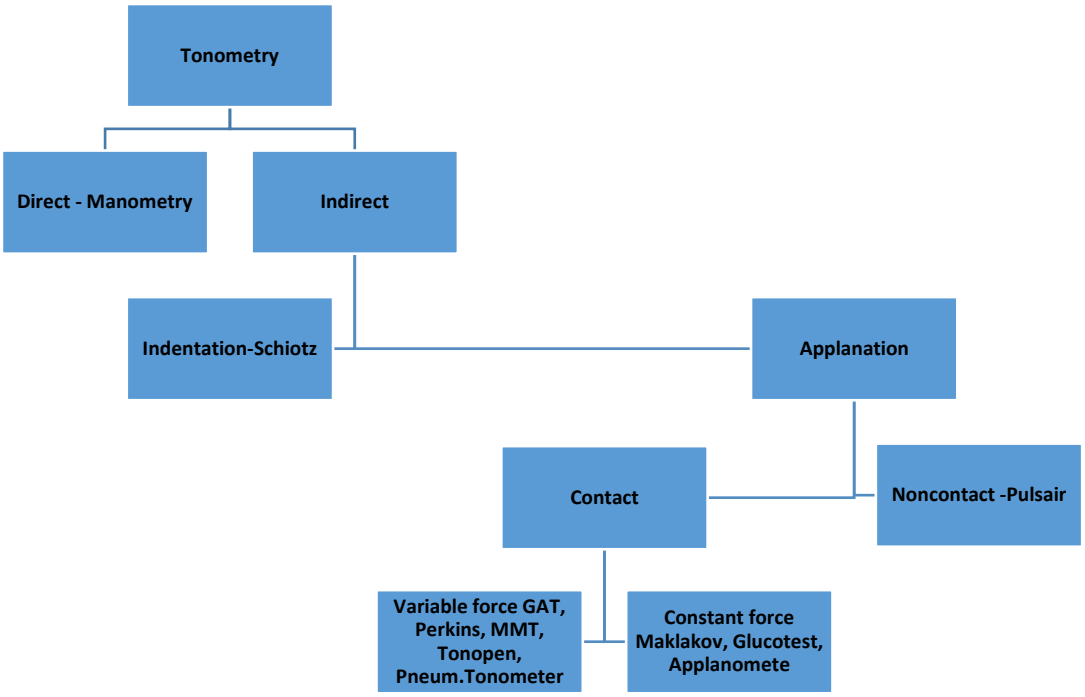


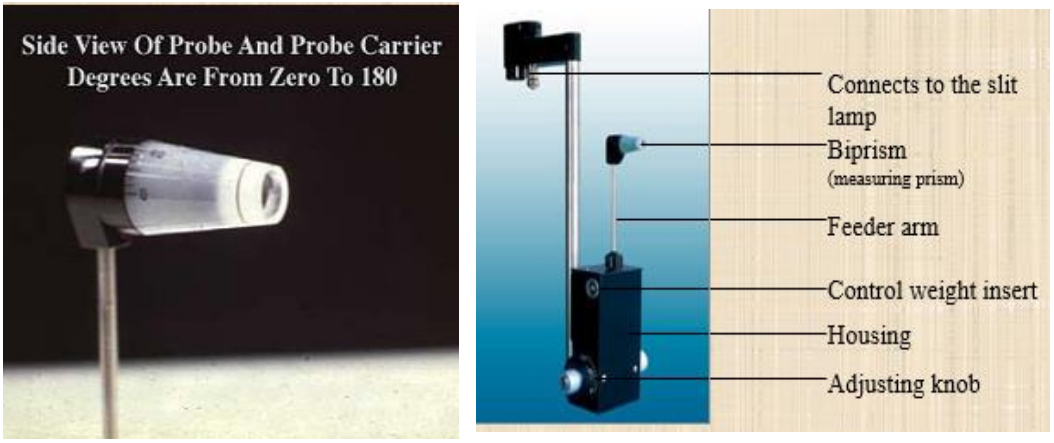

**Figure 15**

## Principles

Goldmann applanation tonometry (GAT) is based on the Imbert–Fick principle, which states that for a dry thin-walled sphere, the pressure (*P*) inside the sphere equals the force (*F*) necessary to flatten its surface divided by the area (*A*) of flattening (i.e. $P = F/A$).

Theoretically, average corneal rigidity (taken as 520 μm for GAT) and the capillary attraction of the tear meniscus cancel each other out when the flattened area has the 3.06 mm diameter contact surface of the Goldmann prism, which is applied to the cornea using the Goldmann tonometer with a measurable amount of force from which the IOP is deduced.(6)

**Figure 16**

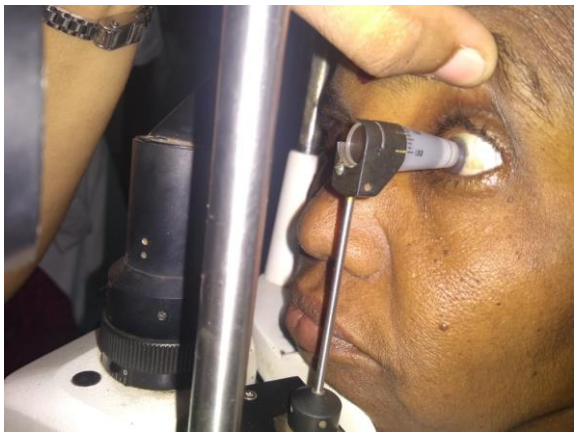


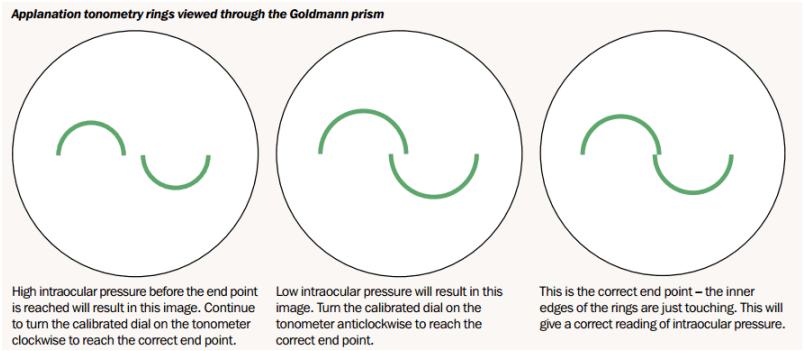
**Technique**

- Instil the local anaesthetic drops and then the fluorescein. Only a very small amount of fluorescein is needed
- For measuring the IOP in the right eye, make sure the slit beam is shining onto the tonometer head from the patient's right side; for the left eye, the beam should come from the patient's left side
- Move the filters so that the blue filter is used to produce a blue beam
- Make sure the beam of light is as wide as possible, and that the light is as bright as possible. This makes visualising the fluorescein rings easier (with the slit diaphragm fully open)
- Ask the patient to look straight ahead, open both eyes wide, fix his or her gaze and keep perfectly still
- With the thumb, gently hold up the patient's top eyelid, taking care not to put any pressure on the eye
- Direct the blue light from the slit lamp or the Perkins tonometer onto the prism head
- Make sure that the tonometer head is perpendicular to the eye
- Move the tonometer forward slowly until the prism rests gently on the centre of the patient's cornea
- With the other hand, turn the calibrated dial on the tonometer clockwise until the two fluorescein semi-circles in the prism head are seen to meet and form a horizontal ‘S’ shape. (Note: the correct end point is when the inner edges of the two fluorescein semi-circle images just touch)

- The dial on the tonometer is rotated to vary the applied force; the inner margins of the semicircles align when a circular area of diameter precisely 3.06 mm is flattened.
- The reading on the dial, multiplied by 10, gives the IOP;
- Note the reading on the dial and record it in the notes
- Withdraw the prism from the corneal surface and wipe its tip
- Repeat the procedure for the other eye
- Wipe the prism with a clean, dry swab and replace it in the receptacle containing the disinfectant

**Figure 17**





**SOURCES OF ERROR**:

**Falsely low IOP**

- ✓ too thin cornea
- ✓ corneal edema
- ✓ prolonged contact
- ✓ Repeated tonometry
- ✓ little flourescein

**Falsely high IOP**

- ✓ too much fluorescein
- ✓ thick cornea
- ✓ steep cornea
- ✓ wider meniscus
- ✓ against the rule astigmatism
- ✓ Widening the lid fissure excessively

**Maintenance of intraocular pressure:**

The intraocular pressure (IOP) refers to the pressure exerted by intraocular fluids on the coats of the eyeball. The **normal IOP varies between 10 and 21 mm of Hg (mean 16 ± 2.5 mm of Hg).** The normal level of IOP is essentially maintained by a dynamic equilibrium between the formation and outflow of the aqueous humour.

Various factors influencing intraocular pressure can be grouped as under:

**(A) Local factors**

| | | |
|---|---|---|
| **1.** | **Rate of aqueous formation** | permeability of ciliary capillaries and osmotic pressure of the blood. |
| **2.** | **Resistance to aqueous outflow (drainage)** | Most of the resistance to aqueous outflow is at the level of trabecular meshwork. |
| **3.** | **Increased episcleral venous pressure** | Valsalva manoeuvre causes temporary increase in episcleral venous pressure and rise in IOP |
| **4.** | **Dilatation of pupil in patients with narrow anterior chamber angler** | relative obstruction of the aqueous drainage by the iris. |

**(B) General factors:**

| | | |
|---|---|---|
| 1. | **Heredity** | Multifactorial |
| 2**.** | **Age** | IOP is equal between the sexes in ages 20- 40 years. In older age groups increase in mean IOP with age is greater in females. |
| 3. | **Sex** | after the age of 40 years, possibly due to reduced facility of aqueous outflow |
| 4. | **Diurnal variation** | tendency of higher IOP in the morning and lower in the evening<br>**Normal eyes have a smaller fluctuation (< 5 mm of Hg) than glaucomatous eyes (> 8 mm of Hg).**<br>related to diurnal variation in the levels of plasma cortisol |
| 5. | **Postural variations** | IOP increases when changing from the sitting to the supine position |
| **6.** | **Blood pressure** | prevalence of glaucoma is marginally more in hypertensives than the normotensives. |
| 7. | **Plasma osmolarity** | **increase in plasma osmolarity**<br>fall in IOP- occurs after intravenous mannitol, oral glycerol<br>**reduction in plasma osmolarity**<br>rise in IOP -water drinking provocative tests |
| 8. | **General anaesthetics and other drugs** | alcohol lowers IOP, tobacco smoking,<br>caffeine and steroids may cause. antiglaucoma drugs which lower IOP. |

## OCULAR PERFUSION PRESSURE:

Ocular blood flow refers to the distribution of oxygenated blood throughout the vasculature in the eye. Ocular perfusion pressure (OPP) refers to the pressure available to drive blood through the intraocular vasculature, with the degree of perfusion being influenced by the resistance to flow, which is a function of the vessel calibre or the vessel tone.

Ocular blood flow exhibits autoregulation, which is characterized 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.

**Perfusion pressure** of an isolated organ is defined as the pressure difference between its arterial and venous blood supply.

**Mean ocular perfusion pressure** is the net pressure gradient causing the blood to flow to the eye. It's the difference in pressure between the arterial and venous parts of vascular bed throughout the eye including optic nerve head.

The potential consequences of abnormal low levels of OPP include optic nerve head and retinal ischaemia as well as reperfusion injury leading to development of open angle glaucoma and it's progression.(7) Hence, OPP changes occur when there is a change in either in BP/IOP or both. When blood pressure decreases or IOP raises, ocular perfusion pressure becomes low. This is maintained by the presence of autoregulation mechanism.

## Autoregulation

**Autoregulation** is the ability of a vascular bed to maintain its BF despite changes in perfusion pressure. Autoregulation impairment means the eye is less able to cope up with the continuous episodes of low ocular perfusion pressure and with overtime produces a cumulative effect producing progressive retinal ganglion cell loss in turn causing nerve fibre defect ultimately progressing to field defect.

In the retina and the ONH it appears that autoregulation is strongly dependent on myogenic and metabolic mechanisms. In the choroid the rich parasympathetic, sympathetic and sensory innervation as well as intrinsic choroidal neurons plays a key role in BF regulation in face of changes in OPP (8)Hence, OPP is termed recently as '**Risk Factor' (or) 'Progression Factor'** in the development of open angle glaucoma.

Alterations in autoregulation in glaucoma may also arise from a phenomenon called **primary vascular dysregulation.** Several observations indicate that endothelial dysfunction is associated with glaucoma .In addition, both endothelin and nitric oxide (NO) are key regulators of ONH and choroidal BF at baseline and during isometric exercise(9) (11)(10).

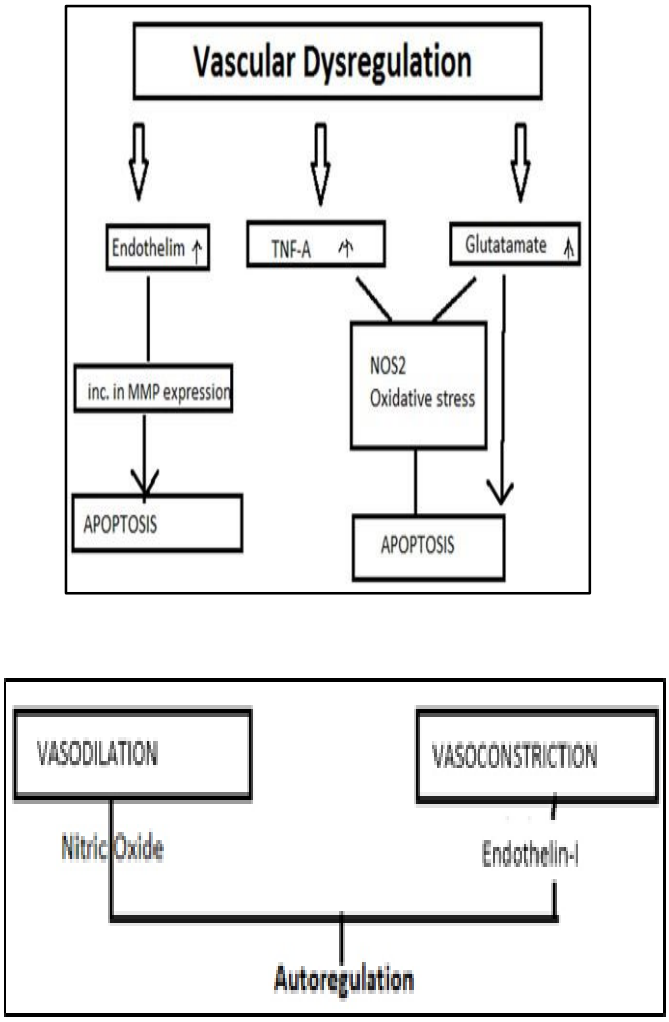



**Figure 18**

In the ONH there is evidence that **glial cells** play a role in autoregulatory processes. This may be related to loss of autoregulation in glaucoma, because astrocytes are considered to play a key role in tissue remodeling of the ONH. It is, however, unclear how early this activation of astrocytes occurs although there is evidence that release of substances such as glutamate and tumor necrosis factor α from astrocytes is involved in RGC death . In the ONH astrocytes are involved in autoregulation during an increase in IOP, because the gliotoxic agent L-2-aminoadipic acid modifies the BF response during the decrease in OPP .

**Neurovascular coupling**

In the brain and the retina BF increases when neurons get active, a response called functional hyperemia . This phenomenon called neurovascular coupling (7). An abnormal BF response to neuronal stimulation causes cell death caused by inadequate nutrient supply.

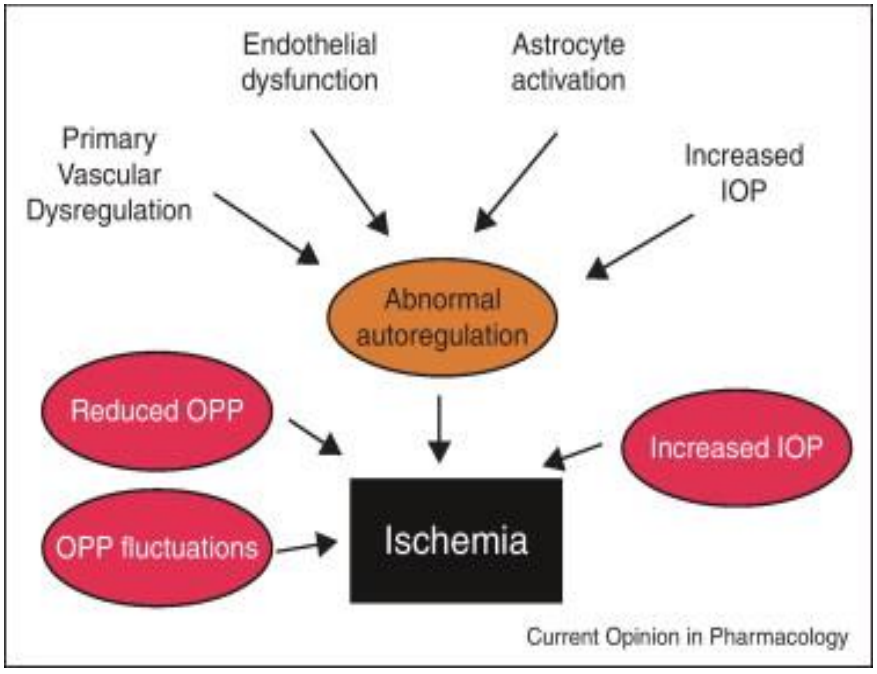
Astrocytes play a key role in mediating the vasodilator response associated with neural activity.

Synaptically released glutamate ⟹ activates N-methyl-D-aspartate receptors and **metabotropic glutamate receptors** in neurons and astrocytes ⟹ Increase in intracellular $Ca^{2+}$ ⟹ activating **arachidonic acid pathway** ⟹ synthesis of **vasodilators** such as **prostaglandins** and **epoxyeicosatrienoic acids** and **vasoconstrictors** such as **20-hydroxy-eicosatetraenoic acid.**

In addition NO synthesized from NO synthase-1 in neurons may play a role in the vasodilator response. Indeed, NO synthase inhibition blunts the retinal hyperemic response to flicker stimulation in humans . Generally it is, however, believed that NO has a modulatory rather than a mediating role in the human retinal neurovascular coupling, because the activity of the enzymes in the arachidonic acid pathways depend on the level of NO . As such the hyperemic response may also depend on endothelial NO related to flow-mediated mechanisms.

In glaucoma the response of retinal and ONH BF to flicker stimulation is reduced .

**Figure 19**



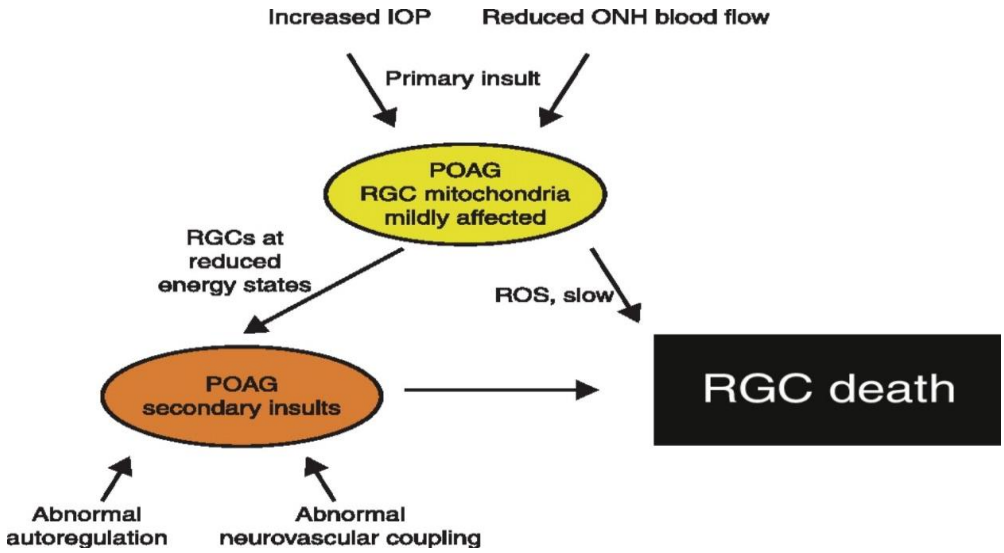

**Pathogenesis of glaucomatous damage due to decreased ocular perfusion pressure** :

a. **Optic nerve head ischaemia**, which reduce nutrients to retinal ganglion cell axons.

b. Once primary insult has occurred at the level of optic nerve head, **retinal ganglion cells appear to function at reduced energy levels** with affected mitochondria.

c. **Oxidative stress** associated with extensive production of reactive oxygen species, free radicals, hydrogen peroxide.(7)

**Figure 20**

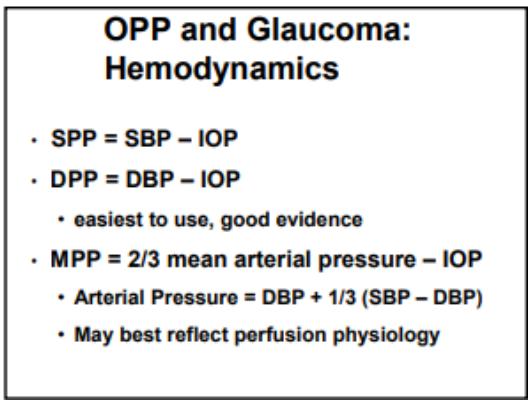


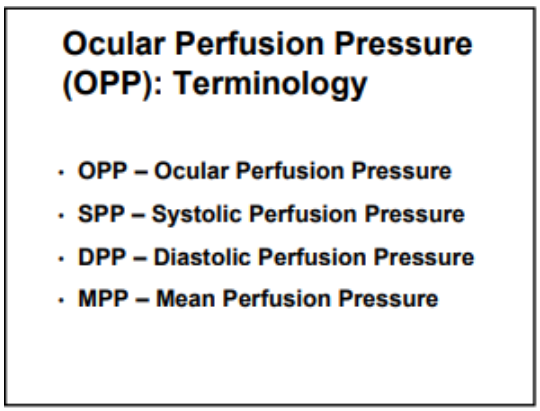

- **Mechanical theory** which is currently prevailed, postulates that raised IOP alters the architecture so much that axons get compressed and die.
- **Vascular theory**, starring OPP, suggests that when enough blood does not reach the optic nerve when perfusion pressure gets too low.
- **Fluctuation theory** contends that single pressure readings taken months apart are far less concerning for optic nerve insults than dramatic, diurnal fluctuations.
- **Neurogenic theory** suggests that individuals have very different thresholds of vulnerability to all pressures, and true damage to the optic nerve occurs far in advance of the appearance of visual field defects, even in people with "normal" pressures.

Even in the presence of autoregulation, low perfusion pressure predisposes for ischaemic periods, by reducing the range of the autoregulatory reserve.

Mean OPP (MOPP) can be calculated as 2/3 of the mean arterial BP – IOP; where mean arterial pressure = diastolic BP + 1/3 (systolic BP – diastolic BP). The factor 2/3 accounts for the drop in blood pressure between the brachial and ophthalmic artery when the subject is seated (Riva et al. 1986) and the fact that the orbital arteries are further downstream.

Systolic ocular perfusion pressure (SOPP) is defined as the difference between systemic systolic BP and IOP, whereas diastolic ocular perfusion pressure (DOPP) equals systemic diastolic BP – IOP. DOPP is especially useful in displaying the lowest OPP values.



**Factors that Influence the Calculation of OPP**

OPP is a complex variable that may be affected by one or more of the components that are used to calculate its value. For example, the OPP is decreased when systemic BP is low or when IOP is high. Each of these components can be modified by antihypertensive and antiglaucoma medications respectively. Moreover, the maintenance of OPP depends on a complex regulation process that balances BP and IOP to guarantee adequate perfusion of ocular tissues (Costa et al. 2009; Leske 2009; Topouzis & Founti 2009).

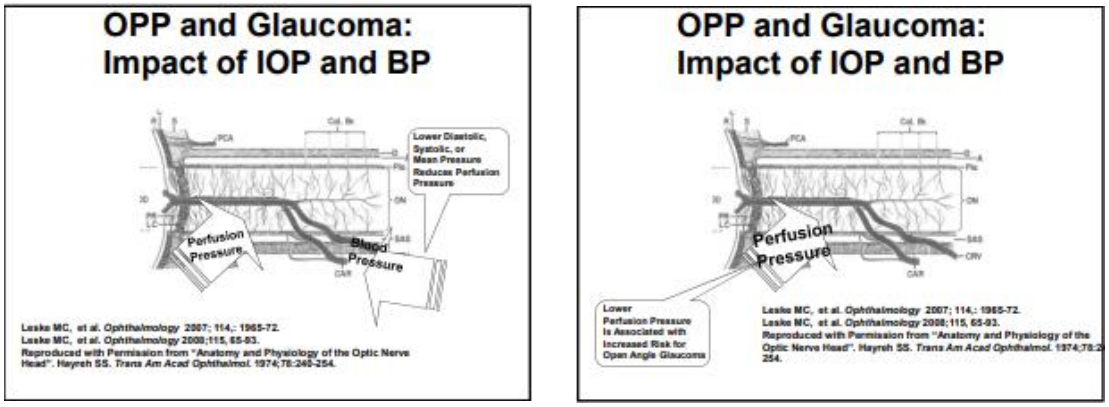
In clinical studies, the **brachial arterial pressure** has often been considered representative of systemic blood pressure and is used as the basis for calculating the ophthalmic arterial pressure in the calculation of OPP. However, the pressure in the brachial artery is not a precise predictor of the pressure in the ophthalmic artery. When an individual is **sitting or standing**, due to the higher position of the head from the heart, the ocular arterial blood pressure is lower than the brachial arterial pressure because of the hydrostatic column effect.

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(11,12). IOP, the other variable determining OPP, has also been shown to follow a pattern of circadian change. It has been shown in healthy subjects that nocturnal IOP is significantly higher than diurnal IOP, with peak IOP occurring at the end of the night just before awakening(12). Part of the nocturnal increase in IOP has been attributed to a change from sitting to supine

position with sleep, but this circadian IOP elevation can be detected even without a postural change (Liu et al. 2003a)

In clinical practice, IOP is mostly measured in the upright position. However, higher readings are often found in the supine position, probably as a result of an increase in the episcleral venous pressure(13) (No€el et al. 2001; Liu et al. 2003a,c).

**Figure 21**




It has been reported that during sleep (supine), 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)

Wider diurnal fluctuations in IOP have been associated with increased risk for glaucomatous visual field loss

**Effects of Medications on OPP**

Recent clinical trials have underlined the importance of lowering BP to to American Heart Association (AHA) released a scientific statement in 2007 that recommends more aggressive control of BP (reduction to <130/80mmhg) among those at high risk of coronary artery disease, including patients with diabetes and others conditions, and even lower (reduction to <120/80mmhg) in patients with left ventricular dysfunction.

Overtreatment of systemic hypertension could worsen glaucoma damage. Besides the contribution of a low blood pressure to glaucoma risk, it is necessary to remember that patients suffering from systemic hypertension usually have increased peripheral vascular resistance and a compromised vascular system, which can also influence glaucoma damage. This may be due to the inability of the eye to maintain consistent and adequate perfusion during pharmacologically lowered blood pressure in susceptible individuals.

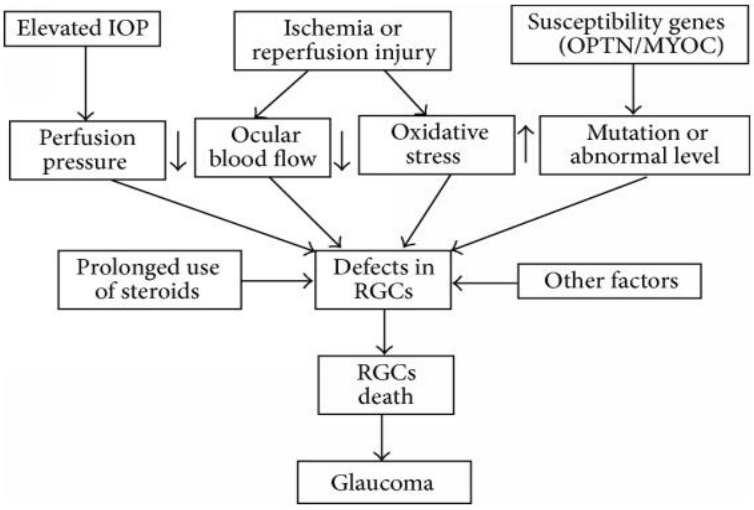



**Figure 22**

## PRIMARY OPEN ANGLE GLAUCOMA

As the name implies, it is a type of primary glaucoma, where there is **no obvious systemic or ocular cause of rise in the intraocular pressure.** It occurs in eyes with open angle of the anterior chamber. Primary open angle glaucoma (POAG) also known as chronic simple glaucoma of adult onset and is typically characterised by slowly progressive raised intraocular pressure (>21 mmHg recorded on at least a few occasions) associated with characteristic optic disc cupping and specific visual field defects.

**1. Raised intraocular pressure (Mechanical theory).**

**2. Pressure independent factors (Vascular insufficiency theory)**

**B. Secondary insults (Excitotoxicity theory)**

### (A) Predisposing and risk factors:

- polygenic inheritance,10% risk in the siblings and 4% in the offspring of patients with POAG
- Risk increases with increasing age, more commonly between 5th and 7th decades.
- More common, develops earlier and more severe in black people than in white.
- **Myopes** -more predisposed than the normals.
- Diabetics higher prevalence of POAG than non-diabetics.
- Cigarette smoking - increase its risk.

High blood pressure is not the cause of rise in IOP, prevalence of POAG is more in hypertensives than the normotensives.

- Thyrotoxicosis is also not the cause of rise in IOP, but prevalence of POAG is more in patients suffering from Graves' ophthalmic disease than the normals.

**(B) Pathogenesis of rise in IOP**

It is certain that rise in IOP occurs due to decrease in the aqueous outflow facility due to increased resistance to aqueous outflow caused by age-related thickening and sclerosis of the trabeculae and an absence of giant vacuoles in the cells lining the canal of Schlemm.

**(C) Corticosteroid responsiveness:** Patients with POAG and their offspring and sibilings are more likely to respond to six weeks topical steroid therapy with a significant rise of IOP.

**INCIDENCE OF POAG** It varies in different populations. In general, it affects about 1 in 100 of the general population (of either sex) above the age of 40 years. It forms about onethird cases of all glaucomas.

**Symptoms**

1. The disease is insidious and usually asymptomatic
2. mild headache and eyeache.
3. Occasionally, a defect in the visual field.

4. Frequent changes in presbyopic glasses.Reading and close work often present increasing difficulties owing to accommodative failure due to constant pressure on the ciliary muscle and its nerve supply.
5. Delayed dark adaptation

**Signs**

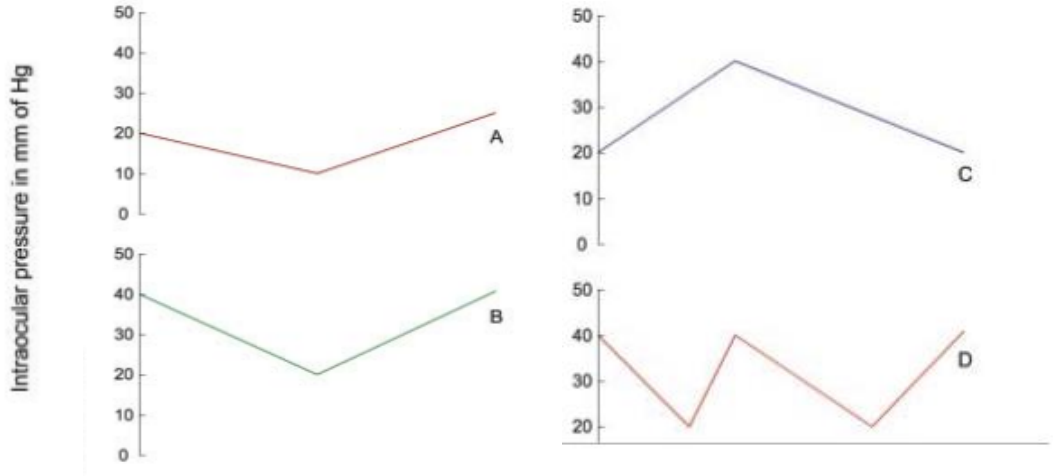
1. **Anterior segment signs:** Ocular examination including slit-lamp biomicroscopy may reveal normal anterior segment. In late stages pupil reflex becomes sluggish and cornea may show slight haze.

2. **IOP changes**:In the initial stages the IOP may not be raised permanently, but there is an exaggeration of the normal diurnal variation. Therefore, repeated observations of IOP (every 3-4 hour), for 24 hours is required during this stage (Diurnal variation test). In most patients IOP falls during the evening, contrary to what happens in closed angle glaucoma.

3. **Patterns of diurnal variation of IOP** : A variation in IOP of over 5 mm Hg (Schiotz) is suspicious and over 8 mm of Hg is diagnostic of glaucoma. In later stages, IOP is permanently raised above 21 mm of Hg and ranges between 30 and 45 mm of Hg.

**Patterns of diurnal variation of IOP:**

**Figure 23**




A, normal slight morning rise; B, morning rise seen in 20% cases of POAG;

C, afternoon rise seen in 25% cases of POAG;

D, biphasic variation seen in 55% cases of POAG.

I. **Optic disc changes**: Optic disc changes, usually observed on routine fundus examination, provide an important clue for suspecting POAG. These are typically **progressive, asymmetric** and present a variety of characteristic clinical patterns. It is essential, therefore, to record the appearance of the nerve head in such a way that will accurately reveal subtle glaucomatous changes over the course of follow-up evaluation.

**Examination techniques:**

Careful assessment of disc changes can be made by direct ophthalmoscopy, slitlamp biomicroscopy using a + 90D lens, Hruby lens or Goldmann contact lens and indirect ophthalmoscopy.

The recording and documentation techniques include serial drawings, photography and photogrammetry. Confocal scanning laser topography (CSLT) i.e., Heidelberg retinal tomograph (HRT) is an accurate and sensitive method for this purpose. Other advanced imaging techniques include optical coherence tomography (OCT) and scanning laser polarimetry i.e., Nerve fibre analyser (NFA).

**Glaucomatous changes in the optic disc can be described as early changes, advanced changes and glaucomatous optic atrophy.**

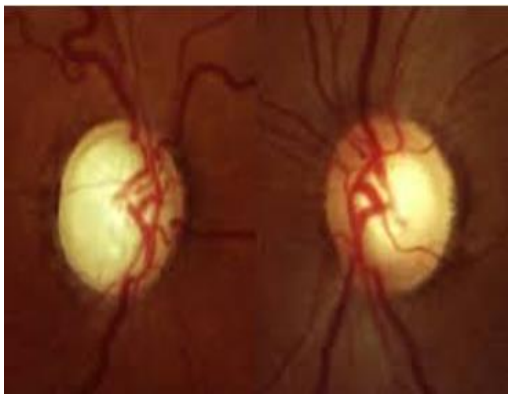
**(a) It should be suspected to exist if fundus examination reveals one or more of the following signs(14):**

1. **Vertically oval cup** due to selective loss of neural rim tissue in the inferior and superior poles.
2. **Asymmetry of the cups**. A difference of more than 0.2 between two eyes is significant.
3. **Large cup** i.e., 0.6 or more (normal cup size is 0.3 to 0.4) may occur due to concentric expansion.
4. **Splinter haemorrhages** present on or near the optic disc margin.
5. **Pallor areas on the disc**.
6. **Atrophy of retinal nerve fibre layer** which may be seen with red free light.

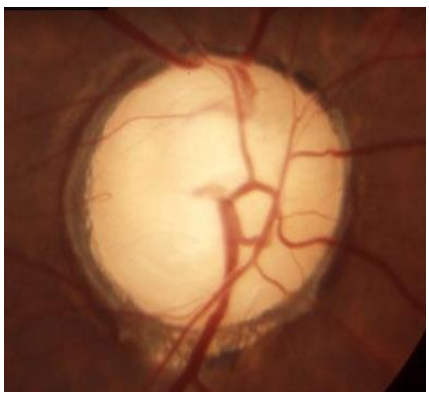
**(b) Advanced glaucomatous changes in the optic disc:**

1. **Marked cupping (cup size 0.7 to 0.9),** excavation may even reach the disc margin, the sides are steep and not shelving (c.f. deep physiological cup).

**Figure 24**



Nasalisation of vessels

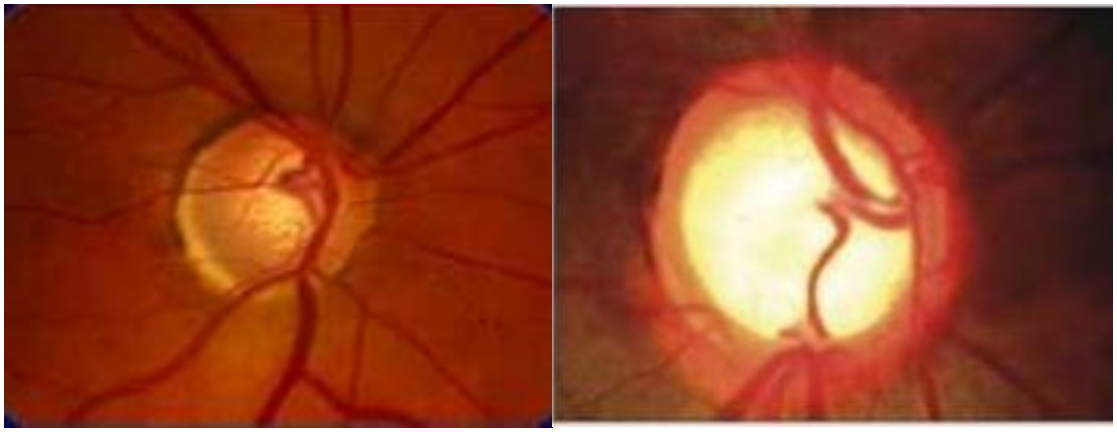


Bayonetting

2. **Thinning of neuroretinal rim** which occurs in advanced cases is seen as a crescentric shadow adjacent to the disc margin.
3. **Nasal shifting of retinal ve**ssels which have the appearance of being broken off at the margin is an important sign (Bayonetting sign). When the edges overhang, the course of the vessels as they climb the sides of the cup is hidden.
4. **Pulsations of the retinal arterioles** may be seen at the disc margin (a pathognomic sign of glaucoma), when IOP is very high.
5. **Lamellar dot sign** the pores in the lamina cribrosa are slit-shaped and are visible up to the margin of the disc.

**(c) Glaucomatous optic atrophy**: As the damage progresses, all the neural tissue of the disc is destroyed and the optic nerve head appears white and deeply excavated

**Figure 25**



Laminar Dot Sign Glaucomatous optic atrophy

**Pathophysiology of disc changes:**

Both mechanical and vascular factors play a role in the cupping of the disc.

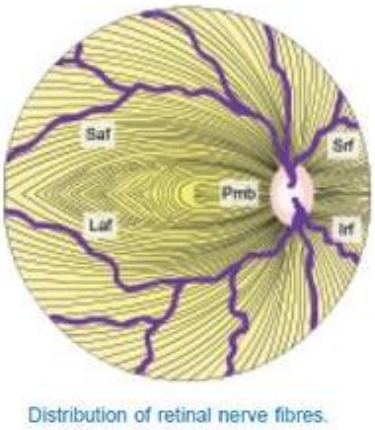
**Mechanical effect** of raised IOP forces the lamina cribrosa backwards and squeezes the nerve fibres within its meshes to disturb axoplasmic flow.(15)

**Vascular factors** contribute in ischaemic atrophy of the nerve fibres without corresponding increase of supporting glial tissue. As a result, large caverns or lacunae are formed (cavernous optic atrophy).

**II. Visual field defects:** Visual field defects usually run parallel to the changes at the optic nerve head and continue to progress if IOP is not controlled. These can be described as early and late field defects. Anatomical basis of field defects. For better understanding of the actual field defects, it is mandatory to have a knowledge of their anatomical basis.

**(A) Distribution of retinal nerve fibres**

1. Fibres from nasal half of the retina come directly to the optic disc as superior and inferior radiating fibres (srf and irf).
2. Those from the macular area come horizontally as papillomacular bundle (pmb).
3. Fibres from the temporal retina arch above and below the macula and papillomacular bundle as superior and inferior arcuate fibres with a horizontal raphe in between (saf and iaf )



Distribution of retinal nerve fibres.

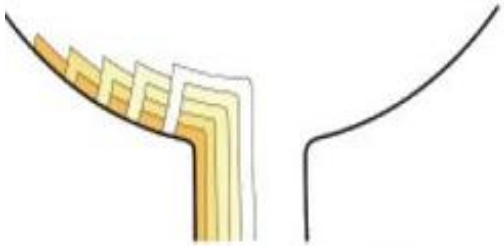

Arrangement of nerve fibres within optic nerve head.

**Figure 26**

**(B) Arrangement of nerve fibres within optic nerve head:**

Those from the peripheral part of the retina lie deep in the retina but occupy the most peripheral (superficial) part of the optic disc. While fibres originating closer to the nerve head lie superficially in the retina and occupy a more central (deep) portion of the disc.

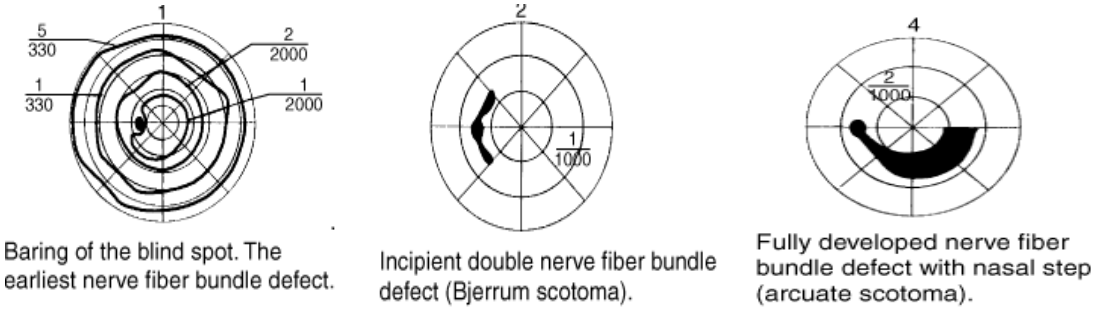
The arcuate nerve fibres occupy the superior and inferior temporal portions of optic nerve head and are most sensitive to glaucomatous damage; accounting for the early loss in the corresponding regions of the visual field. Macular fibres are most resistant to the glaucomatous damage and explain the retention of the central vision till end.

**Progression of field defects.** Visual field defects in glaucoma are initially observed in Bjerrum's area (10- 25 degree from fixation) and correlate with optic disc changes(16). The natural history of the progressive glaucomatous field loss, more or less, takes the following sequence:

1. **Isopter contraction:** It refers to mild generalised constriction of central as well as peripheral field. It is the earliest visual field defect occurring in A C glaucoma.
2. **Baring of blind spot:** It is also considered to be an early glaucomatous change, but is very non-specific and thus of limited diagnostic value. Baring of the blind spot means exclusion of the blind spot from the central field due to inward curve of the outer boundary of 30° central field

3. **Small wing-shaped paracentral scotoma** : It is the earliest clinically significant field defect. It may appear either below or above the blind spot in Bjerrum's area (an arcuate area extending above and below the blind spot to between 10o and 20o of fixation point).

4. **Seidel's scotoma:** With the passage of time paracentral scotoma joins the blind spot to form a sickle shaped scotoma known as Seidel's scotoma .



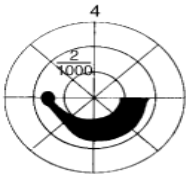

Baring of the blind spot. The earliest nerve fiber bundle defect.

Incipient double nerve fiber bundle defect (Bjerrum scotoma).

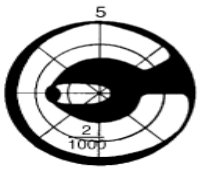
Fully developed nerve fiber bundle defect with nasal step (arcuate scotoma).

**Figure 27**

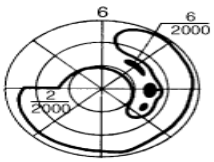
5. **Arcuate or Bjerrum's scotoma:** It is formed at a later stage by the extension of Seidel's scotoma in an area either above or below the fixation point to reach the horizontal line (Fig. 9.13D). Damage to the adjacent fibres causes a peripheral breakthrough.
6. **Ring or double arcuate scotoma**: It develops when the two arcuate scotomas join together
7. **Roenne's central nasal step:** It is created when the two arcuate scotomas run in different arcs and meet to form a sharp right-angled defect at the horizontal meridian.




Fully developed nerve fiber bundle defect with nasal step (arcuate scotoma).



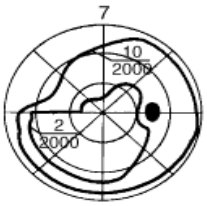

Double arcuate scotoma with peripheral breakthrough and nasal step.




Seidel scotoma. Islands of greater visual loss within a nerve fiber bundle defect.

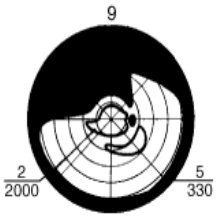
**Figure 28**

8. **Peripheral field defects:** These appear sometimes at an early stage and sometimes only late in the disease. The peripheral nasal step of Roenne's results from unequal contraction of the peripheral isopter.

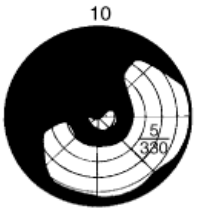



Peripheral breakthrough of large nerve fiber bundle defect with well-developed nasal step.


Nasal depression connected with arcuate scotoma. Nasal step of Rönne.




Peripheral depression with double nerve fiber bundle defect. Isolation of central field.




End stages in glaucoma field loss. Remnant of central field still shows nasal step.

**Figure 29**

9. **Advanced glaucomatous field defects:** The visual field loss gradually spreads centrally as well as peripherally, and eventually only a small island of central vision (tubular vision) and an accompanying temporal island are left. With the continued damage, these islands of vision also progressively diminish in size until the tiny central island is totally extinguished. The temporal island of the vision is more resistant and is lost in the end leaving the patient with no light perception

## REVIEW OF LITERATURE

**BLOOD PRESSURE, PERFUSION PRESSURE, AND OPEN-ANGLE GLAUCOMA: THE LOS ANGELES LATINO EYE STUDY**(17)

**Farnaz Memardazeh Mei Ying-Lai; Jessica Chung; Stanley P. Azen; Rohit Varma; Los Angeles Latino Eye Study Group**

The study included 6130 subjects ,aged 40 years and older w ho underwent an interviewer-administered questionnaire and complete ocular examination. Among the paticipants, higher prevalence of Open angle glaucoma was found in subjects with Low systolic, diastolic and mean perfusion pressures and low diastolic blood pressure . Higher prevalence of OAG was positively correlated with higher systolic BP and MAP.

**OPEN-ANGLE GLAUCOMA AND SYSTEMIC HYPERTENSION: THE BLUE MOUNTAINS EYE STUDY**

**Mitchell, Paul MD, PhD, FRANZCO; Lee, Anne J. MBBS; Rochtchina, Elena MApplStat; Wang, Jie Jin MMed, PhD,Journal of Glaucoma: August 2004 - Volume 13 - Issue 4 - p 319-326**

The Study examined 3654 subjects aged 49 to 97 years. Hypertension was diagnosed from systolic (BP) ≥160 mm Hg or diastolic BP ≥95 mm Hg and from history in treated subjects . OAG was diagnosed from visual field loss, without reference to intraocular pressure (IOP) level. It was found significantly associated with OAG. Strong corelation was found in participants with poorly controlled treated hypertension compared with normotensive subjects independent of IOP.

**INTRAOCULAR PRESSURE AND SYSTEMIC BLOOD PRESSURE: LONGITUDINAL PERSPECTIVE: THE BEAVER DAM EYE STUDY** (18)

**Klein BEK, Klein R, Knudtson MD,British Journal of Ophthalmology 2005;89:284-287**

It was a study of people aged 43– 86 years old living in Beaver Dam. History of use of BP medications was recorded Measurements at baseline (1988–90) and 5 year follow up of systemic BP, IOP, and. Changes in systemic BP and changes in IOP was correlated and it was statistically significant. IOP for a 10 mm Hg increase in SBP was associated with a 0.21mm Hg rise in IOP and 10 mm Hg increase in diastolic BP caused a 0.43 mm Hg rise in IOP. Decreased systolic or diastolic BP of more than 10 mm Hg over 5 years were found to be significantly associated with decreased IOP.

**PRIMARY OPEN-ANGLE GLAUCOMA, INTRAOCULAR PRESSURE, AND SYSTEMIC BLOOD PRESSURE IN THE GENERAL ELDERLY POPULATION: THE ROTTERDAM STUDY (5)**

**IdaDielemansMD[12]JohannesR.VingerlingMD[12]DouweAlgraMSc[2]AlbertHofmanMD, PhD[2]Diederick E.GrobbeeMD, PhD[2]Paulus T.V.M.de JongMD, PhD[1]**

In the Rotterdam Study 4187 subjects, 55 years of age and older were examined. The relation between BP and hypertension with IOP, POAG, high-tension glaucoma, and NTG was studied. A SBP or DBP that was 10 mmHg higher was associated with an IOP average, 0.23 mmHg. The presence of hypertension was associated with a higher mean IOP of 0.66 mmHg.

**DISTRIBUTION OF OCULAR PERFUSION PRESSURE AND ITS RELATIONSHIP WITH OPEN-ANGLE GLAUCOMA STUDY(5):**

**Yingfeng Zheng,1,2 Tien Y. Wong,1,3,4 Paul Mitchell,5 David S. Friedman,6 Mingguang He,2 and Tin Aung1,4**

This was a population-based,cross-sectional study comprising 3280 ethnic Malays. IOP was measured with GAT. SBP and DBP was measured with a digital automatic blood pressure monitor. Independent risk factors for OAG was found as Low DBP, low MOPP, and low DPP and it provided further evidence of a vascular mechanism in glaucoma pathogenesis.

**ASSOCIATION OF BLOOD PRESSURE STATUS WITH THE OPTIC DISK STRUCTURE IN NON-GLAUCOMA SUBJECTS: THE THESSALONIKI EYE STUDY**(19)

**FotisTopouzisMD [a]Anne L.ColemanMD,PhD[b] AlonHarrisPhD[c] ChristianJonescu-CuypersMD[d]FeiYuPhD[b] LeonidasMavroudisMD[a] ArchimidisKoskosasMD[a]M. American Journal of Ophthalmology,Volume 142, Issue 1, July 2006, Pages 144-145**

The association of BP status on the optic disk structure as measured with the Heidelberg Retina Tomograph (HRT) in people without glaucoma.

Rim area was significantly different among groups when DBP was considered as the criterion to classify subjects. Increased cupping and decreased rim area of the optic disk was observed in patients without glaucoma, the DBP was <90 mm Hg that has resulted from antihypertensive treatment.

# PART II

## AIM OF THE STUDY:

- To study mean ocular perfusion pressure in hypertensive patients.
- To study the influence of anti-hypertensive medications on ocular perfusion pressure

## MATERIALS AND METHODS:

This was a cross sectional study carried out in 150 patients patients who were diagnosed cases of systemic hypertension and attended the outpatient department of ophthalmology.

The period of study was from January 2018 – August 2019.

This study was done in accordance with the ethical committee guidelines.

All the patients were informed about the purpose of study and an informed consent was obtained.

A thorough clinical history regarding co-morbidities and treatment history – concerning drugs was taken, and cases were selected here based on inclusion and exclusion criteria.

- Sitting posture BP was recorded in the selected patients after a 5 minute period of rest.
- BCVA and slit lamp examination
- IOP by Goldman Applanation Tonometry
- Gonioscopy for both the eyes using Goldmann 3 mirror lens

- Dilated fundus examination with a direct ophthalmoscope, +90D and Indirect ophthalmoscopy (in cases of media haziness) was done in these patients. Pupillary dilatation was done using 1% tropicamide eye drops
- Visual field examination

Mean ocular perfusion pressure was calculated using standard formula:

**MOPP=2/3*[MAP-IOP]**

**MAP=DBP+1/3 [SBP-DBP]**

**SPP = SBP – IOP**

**DPP = DBP – IOP**

**MOPP**-Mean ocular perfusion pressure

**MAP** - Mean arterial pressure

**SBP** -Systolic blood pressure

**DBP** - Diastolic blood pressure

**SPP** - Systolic perfusion pressure

**DPP** - Diastolic perfusion pressure

**SAMPLE SIZE: 150 HYPERTENSIVE PATIENTS**

## INCLUSION CRITERIA:

Patients with systemic hypertertension of all ages and both sexes on meal plan and anti-hypertensive medications

## EXCLUSION CRITERIA:

Exclusion criteria Patients with hypertension due to secondary causes (kidney disease, endocrine, steroid induced)

## STATISTICAL ANALYSIS:

- **The collected data were analysed with IBM.SPSS statistics software 23.0 Version.**
- **To describe about the data - descriptive statistics, frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.**
- **To find the significance in categorical data Pearson Coefficient test was used. In all the above statistical tools the probability value 0.05 is considered as significant level.**

## OBSERVATIONS AND RESULTS

During this Period of study from January 2018 to August 2019, 150 systemic hypertensive patients reported to ophthalmology Department. Among the 150 subjects, 128 patients were under medications which included single and double drug regimen of anti-hypertensives. 22 subjects were on strict meal plan.

**Table 1**

| | FREQUENCY | PERCENTAGE |
|---|---|---|
| MEAL PLAN | 22 | 14.66% |
| TOTAL PATIENTS ON ANTI-HYPERTENSIVE DRUGS | 128 | 85.33% |
| SINGLE DRUG REGIMEN | 109 | 85.15% |
| DOUBLE DRUG REGIMEN | 19 | 14.84% |
| TOTAL NO OF PATIENTS | 150 | 100% |

Among the 128 subjects on anti-hypertensive drugs,109 patients were on single drug regimen and 19 patients were on double drug regimen.

## DISTRIBUTION OF STUDY POPULATION

**FIGURE 30**

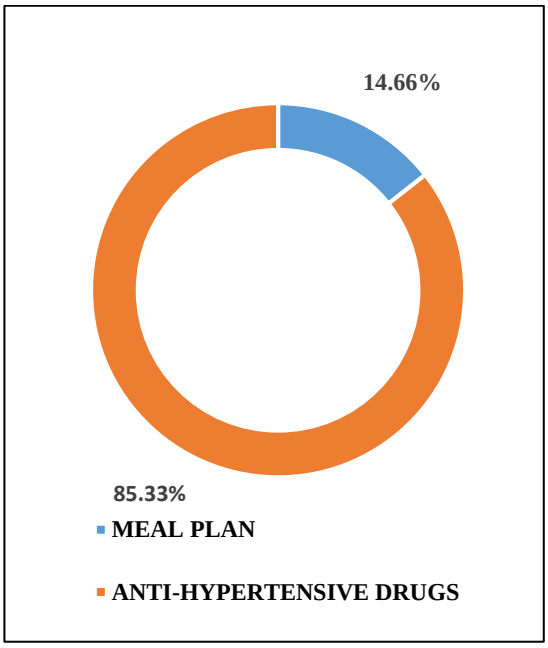
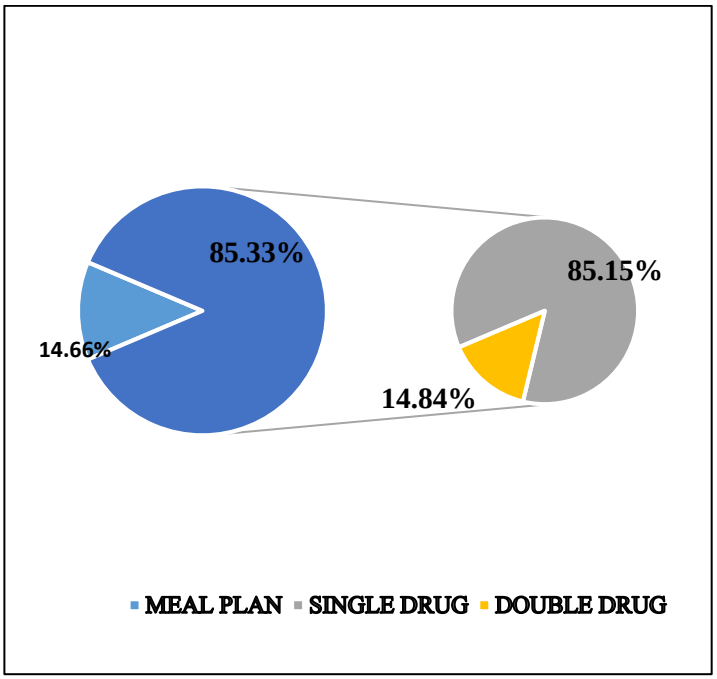



**FIGURE 31**



## AGE & SEX DISTRIBUTION:

Among the 150 patients, there were 94 males (62.66%) and 56 females(37.33%)

The average age of male subjects in our study were 57.5 years and female subjects were 56.8yrs.

**TABLE 2**

| | | |
|---|---|---|
| **TOTAL NO OF PATIENTS** | **150** | |
| **MALE** | **94** | **62.66%** |
| **FEMALES** | **56%** | **37.33%** |

**FIGURE 32**

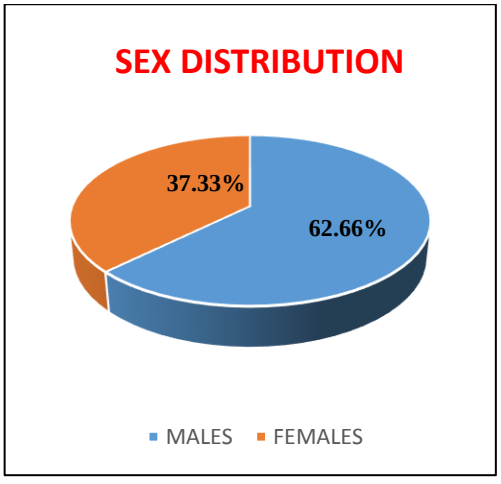
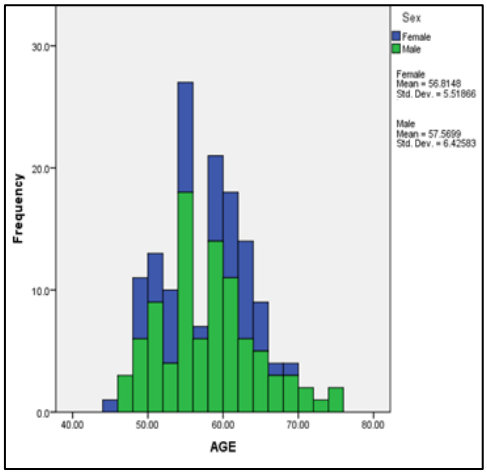



**FIGURE 33**



## INTRAOCULAR PRESSURE:

**Table 3**

| | Mean | Minimum | Maximum | Standard Deviation |
|---|---|---|---|---|
| **IOP (mm Hg)** | 16.52 | 10.00 | 22.00 | **3.06** |

IOP was measured by Goldman Applanation Tonometry. The mean IOP among the subjects were **16.52 ± 3.06 mm Hg.**

## HYPERTENSIVE RETINOPATHY GRADING:

**FIGURE 34**

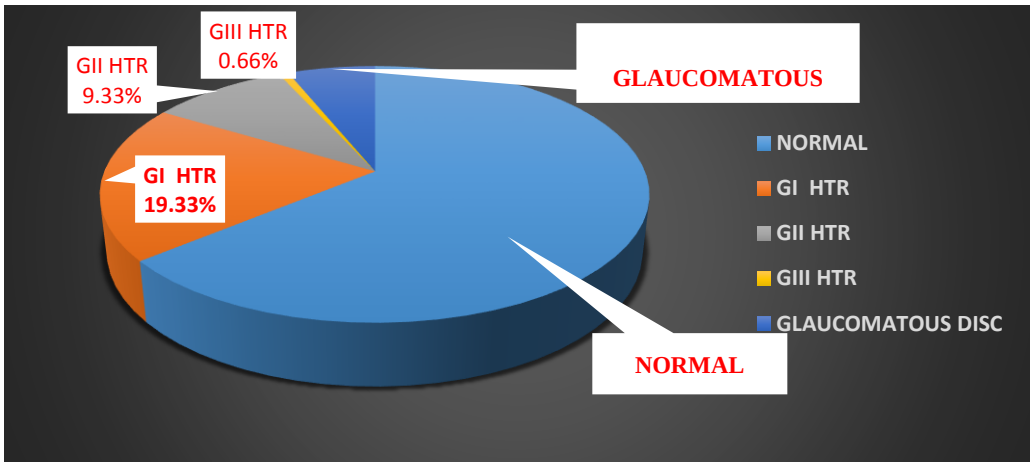



**Table 4**

| GRADE OF RETINOPATHY | FREQUENCY | PERCENTAGE |
|---|---|---|
| **NORMAL** | 97 | 64.66% |
| **GI HTR** | 29 | 19.33% |
| **GII HTR** | 14 | 9.33% |
| **GIII HTR** | 1 | 0.66% |
| **GLAUCOMATOUS DISC** | 9 | **6.00%** |

On examining the fundus of hypertensive patients,glaucomatous changes was obseved in 9 patients,which accounted for 6.20% of study population.The lower prevalence of higher grades of retinopathy can be explained by the fact that we have excluded secondary causes of hypertension

## SYSTOLIC & DIASTOLIC BLOOD PRESSURE:

**FIGURE 35**

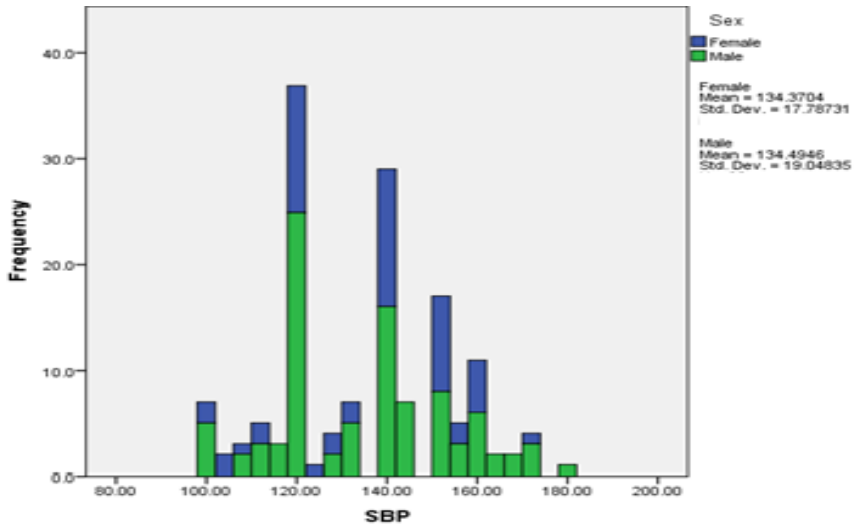



**FIGURE 36**

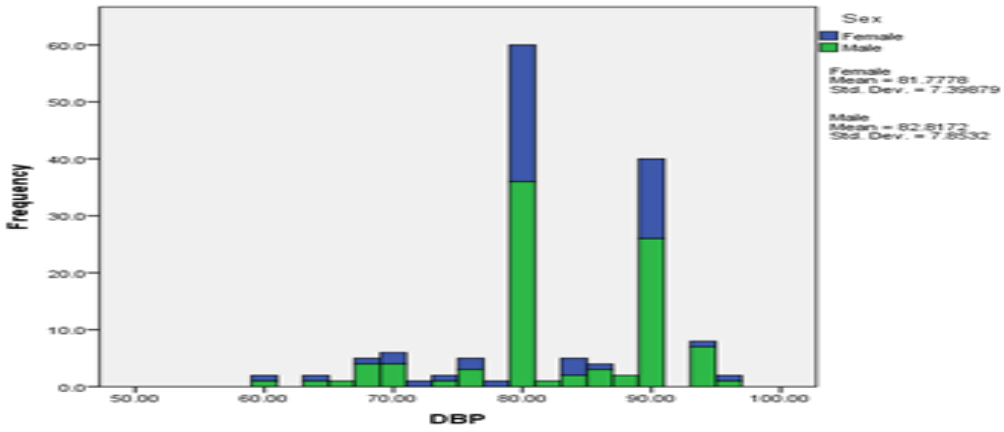



**Table 5**

| | **Mean** | **Minimum** | **Maximum** | **Standard Deviation** |
|---|---|---|---|---|
| **SBP (mm Hg)** | 134.45 | 100.00 | 180.00 | **18.53** |
| **DBP (mm Hg)** | 82.44 | 60.00 | 96.00 | **7.68** |

In our study the mean SBP was around 134 mm Hg and mean DBP wasaround 82mm Hg.The minimum systolic BP recorded was 100 mmHg and diastolic BP recorded was 60 mm Hg.

## CALCULATION OF OPP:

The quartiles were based on MOPP calculated using the below formulas:

| | |
|---|---|
| **MOPP=2/3*[MAP-IOP]**<br><br>**MAP=DBP+1/3 [SBP-DBP**<br><br>**SPP = SBP – IOP**<br><br>**DPP = DBP – IOP** | **MOPP**-Mean ocular perfusion pressure<br>**MAP** - Mean arterial pressure<br><br>**SBP** - Systolic blood pressure<br>**DBP** - Diastolic blood pressure<br><br>**SPP** - Systolic perfusion pressure<br>**DPP** - Diastolic perfusion pressure |

The 150 patients who were included in the study were grouped into 4 Quartiles based on the range of MOPP observed among the study group.

**Table 6**

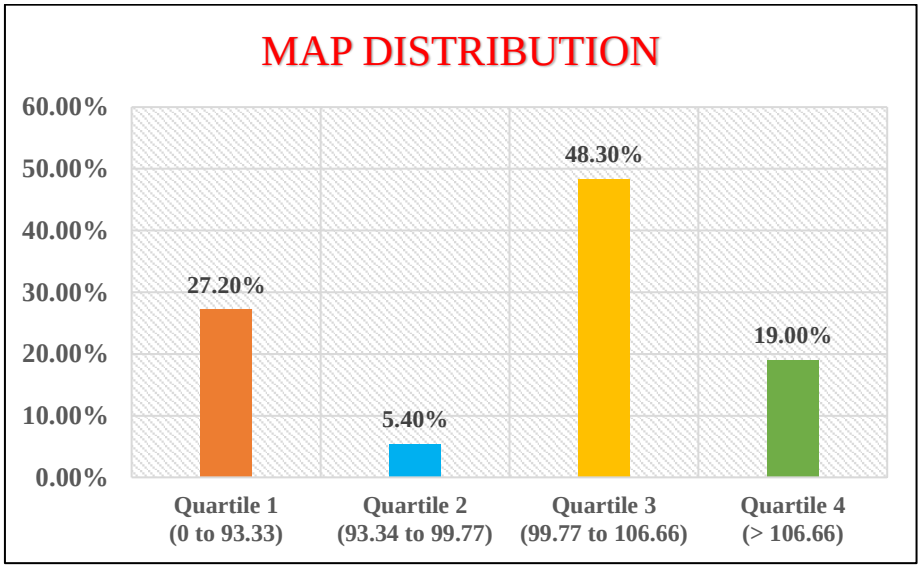
| Quartile | Mean Ocular Perfusion Pressure | Total No of cases at risk | Percentage of cases at risk |
|---|---|---|---|
| **Q1** | **< 53.33** | **52** | **34.66%** |
| **Q2** | **53.33- 55.72** | **25** | **16.6%** |
| **Q3** | **55.72-59.55** | **40** | **26.6%** |
| **Q4** | **>59.55** | **33** | **22.0%** |

## MEAN ARTERIAL BLOOD PRESSURE:

**MAP=DBP+1/3 [SBP-DBP]**
**Where, MAP** - Mean arterial pressure
**SBP** - Systolic blood pressure
**DBP** - Diastolic blood pressure

Mean arterial blood pressure was calculated using the below formula:

**FIGURE 37**




Among the study group, Q1 patients had MAP < 93.33mm Hg,, Q2 had MAP between 93.34% Hg and 99.77mm Hg, Q3 patients between 99.77 to 106.66mm Hg and >106 mmHg.

**Table 7**

| | Mean | Minimum | Maximum | Standard Deviation |
|---|---|---|---|---|
| **MAP (mmHg)** | 99.77 | 73.33 | 116.00 | **9.22** |

## DISTRIBUTION OF OCULAR PERFUSION PRESSURE:

**FIGURE 38**

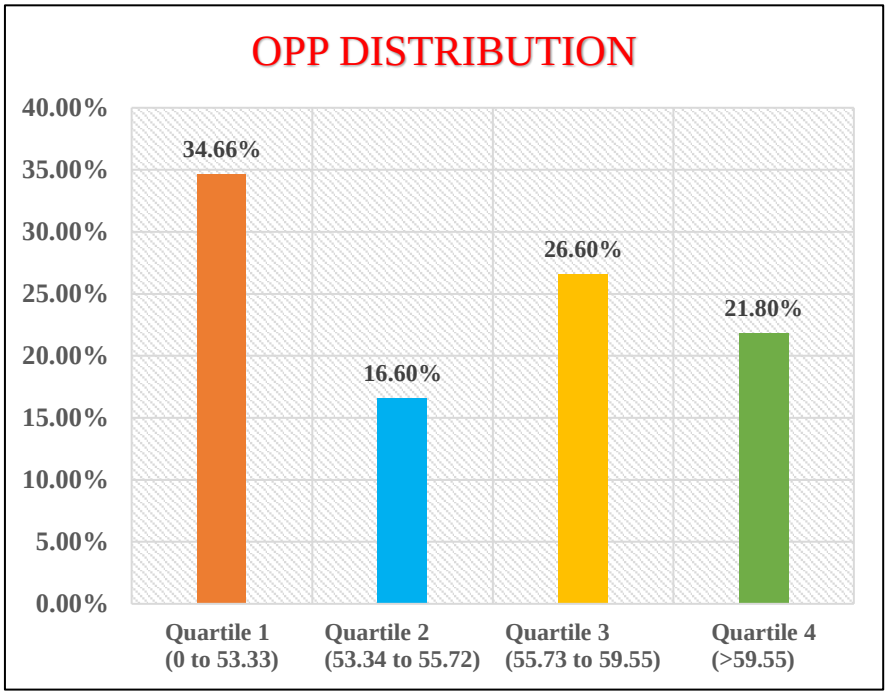



**Table 8**

| | Mean | Minimum | Maximum | Standard Deviation |
|---|---|---|---|---|
| **OPP (mmHg)** | 55.34 | 37.78 | 66.67 | **5.58** |

Among the subjects, Q1 patients had OPP < 53.33 mmHg, Q2 patients had OPP between 53.34 mmHg and 55.72 mmHg, Q3 patients between 55.73 mmHg and 59.55mmHg and Q4 patients had OPP >59.5 mm Hg.

## INCIDENCE OF GLAUCOMA:

Among the study subjects, totally 9 cases of glaucoma were diagnosed. The diagnosis of glaucoma was based after examination of the patients IOP measurement, fundus examination and visual field examination.

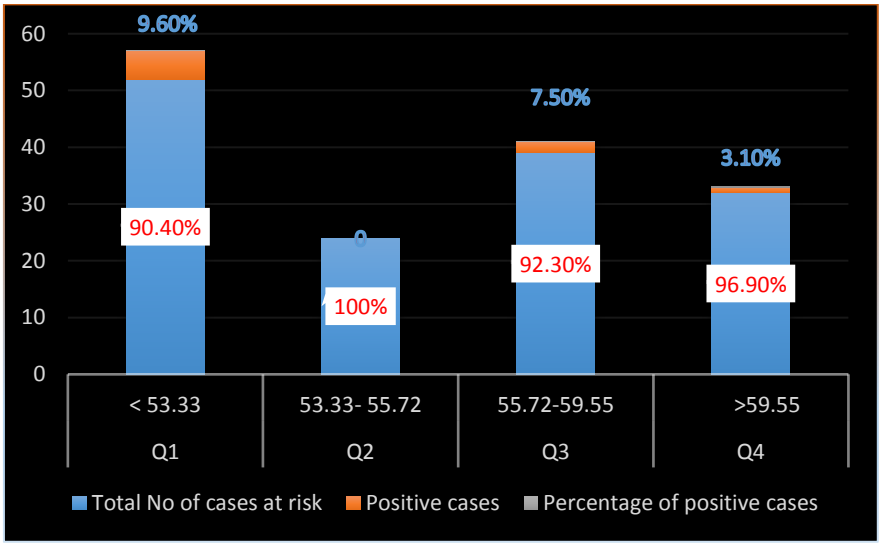
**Table 9**

| Quartile | Mean Ocular Perfusion Pressure | Total No of cases at risk | Positive cases | Percentage of positive cases |
|---|---|---|---|---|
| **Q1** | **< 53.33** | **52** | **5** | **9.6%** |
| **Q2** | **53.33- 55.72** | **25** | **0** | **0** |
| **Q3** | **55.72-59.55** | **40** | **2** | **7.5%** |
| **Q4** | **>59.55** | **33** | **1** | **3.1%** |
| | | **150** | **9** | **6.00%** |

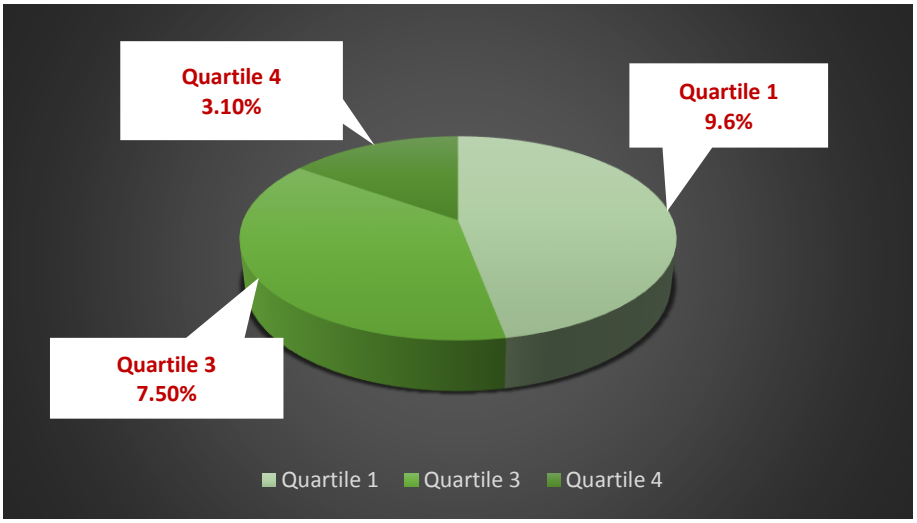
Table 9 explains the incidence of glaucoma among study subjects.In our study of 150 subjects,the number of diagnosed glaucoma cases was 9,which accounts for 6% of study population/. Among the 9 patients, 5 patients belonged to 1$^{st}$ quartile, 3 patients belonged to 2nd quartile and 1 patient belonged to 3$^{rd}$ quartile. The highest incidence of glaucoma in 1$^{st}$ quartile can be attributed to the lower OPP of the subjects.

# INCIDENCE OF GLAUCOMA

## FIGURE 39

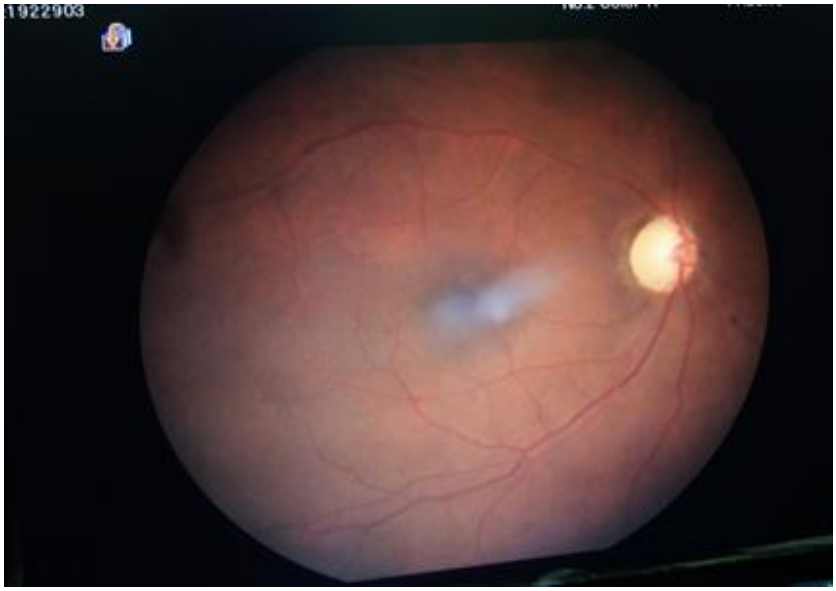



## FIGURE 40

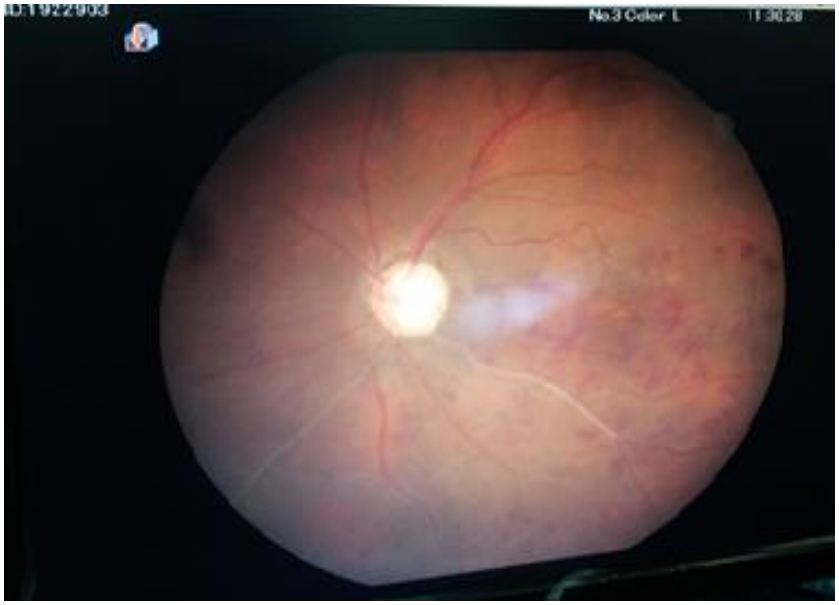


The following fundus picture is one of our diagnosed cases of Glaucoma Mr.Balaji who was a known systemic hypertensive on combined drug regimen of Beta blocker and calcium channel blocker for 4 years.

RIGHT EYE



LEFT EYE



He was diagnosed as a case of,

BE POAG WITH LE GLAUCOMATOUS OPTIC ATROPHY with INFEROTEMPORAL BRVO AND GI HTR.

## CORELATION BETWEEN IOP AND OPP:

**Table 10**

| | Mean | Standard Deviation | Pearson Correlation | Significance (2-tailed) |
|---|---|---|---|---|
| **IOP (mm Hg)** | 16.52 | 3.06 | .203 | **0.05 level** |
| **OPP (mm Hg)** | 55.34 | 5.58 | | |

Significant correlation was noted between IOP and OPP which was statistically proved using Pearsons Corelation at 0.05 level.

## CORELATION BETWEEN SBP,DBP AND OPP:

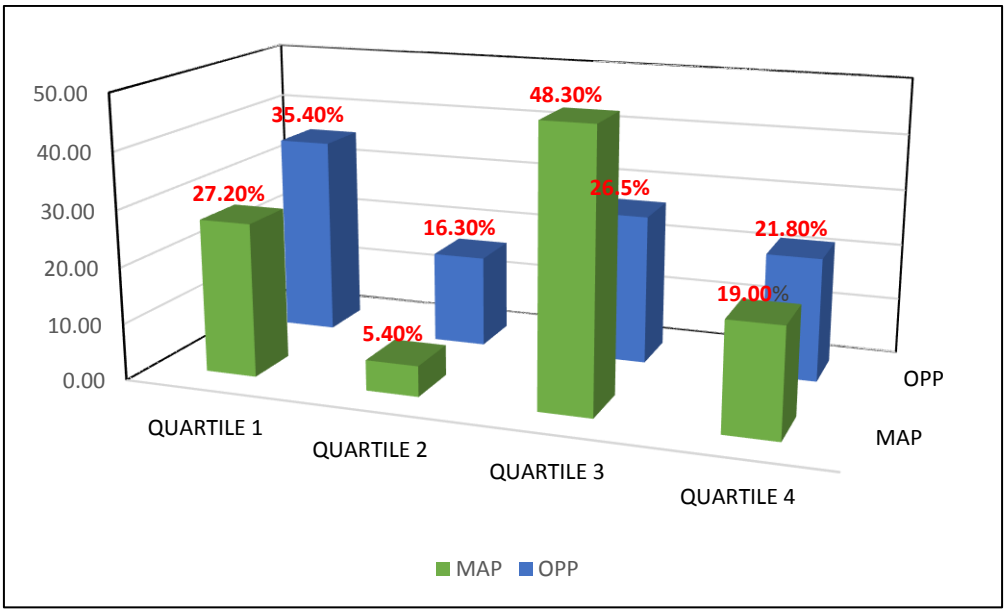
**Table 11**

| | Mean | Standard Deviation | Pearson Correlation | Significance (2-tailed) |
|---|---|---|---|---|
| **SBP (mm Hg)** | 134.45 | 18.53 | .756 | **0.01 level** |
| **DBP (mm Hg)** | 82.44 | 7.68 | .778 | **0.01 level** |
| **OPP (mm Hg)** | 55.34 | 5.58 | | |

Significant correlation noted between SBP,DBP and OPP which was statistically proved using Pearsons Corelation at 0.01 level.

## CORELATION BETWEEN MAP AND OPP:

**FIGURE 41**



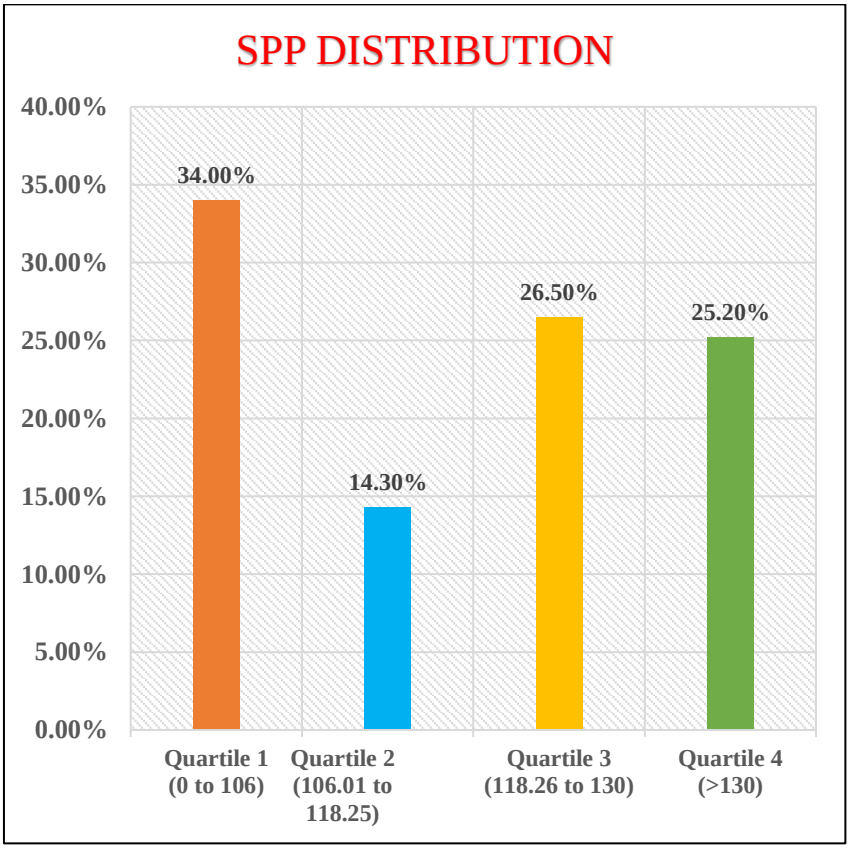

The higher incidence of glaucoma cases in quartile 1 when compared to the others can be explained by the fact that these patients had lower MAP as well as lower OPP than the rest of the study population. The **correlation between MAP and OPP was statistically significant** at 0.01 level

**Table 12**

| | **Mean** | **Standard Deviation** | **Pearson Correlation** | **Significance (2-tailed)** |
|---|---|---|---|---|
| **MAP (mm Hg)** | 99.77 | 9.22 | .939 | **0.01 level** |
| **OPP (mm Hg)** | 55.34 | 5.58 | | |

## DISTRIBUTION OF SYSTOLIC PERFUSION PRESSURE:

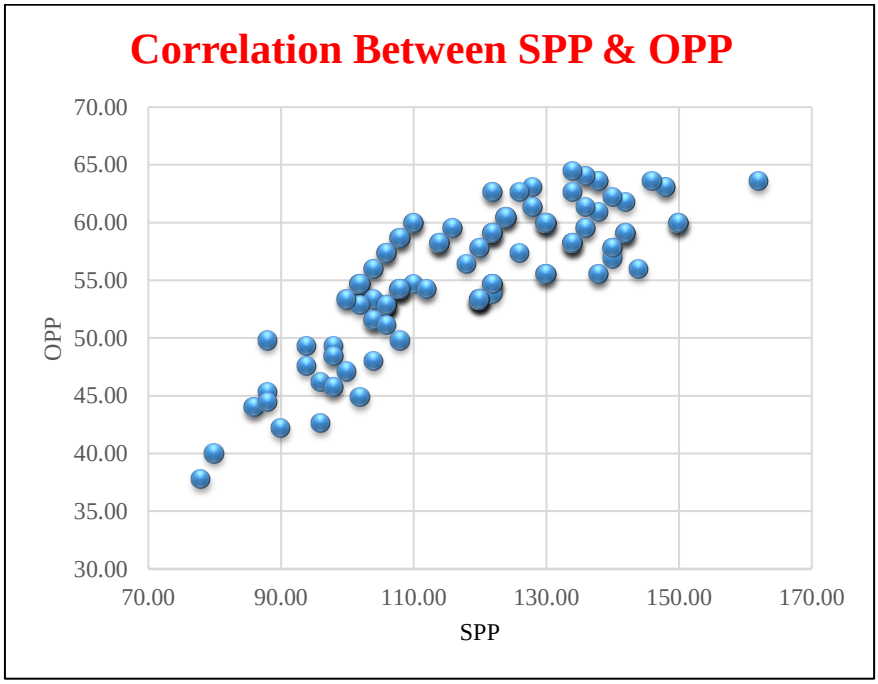
**Figure 42**




The quartile1 patients had a lower SPP <106 mmHg ,quartile 2 between 106-118.25 mmHg, quartile 3 between 118.26mm Hg -130mm Hg and quartile 4 had SPP <106mm Hg .

## CORELATION BETWEEN SYSTOLIC PERFUSION PRESSURE AND OCULAR PERFUSION PRESSURE:

**Figure 43**



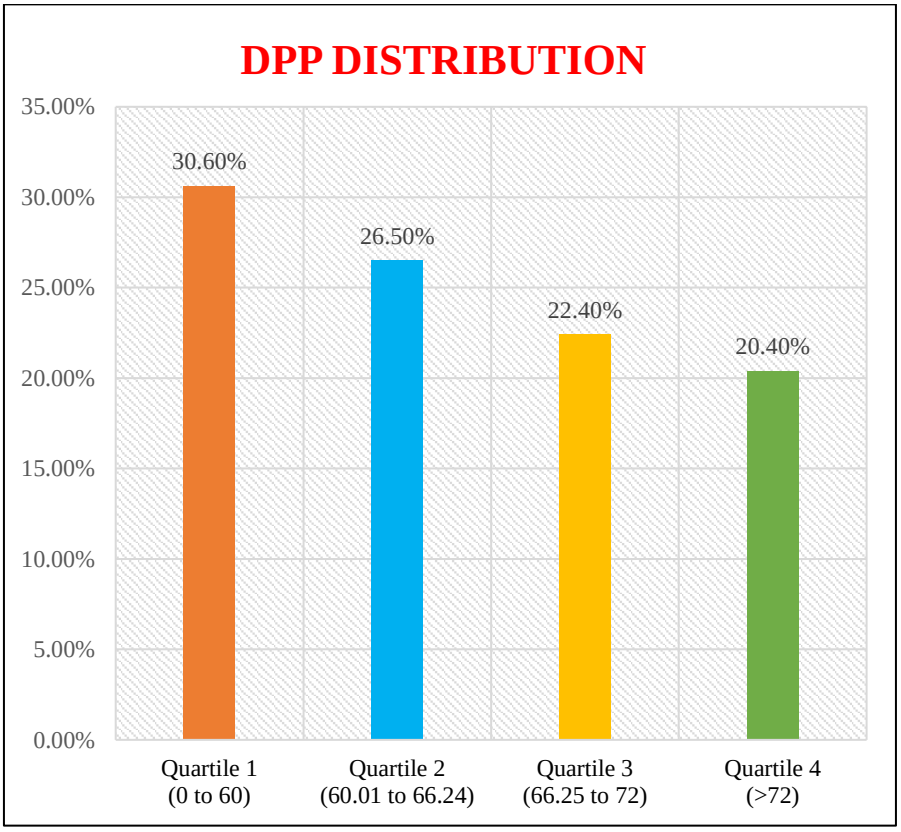

**SPP and OPP showed a statistically significant correlation at 0.01 level,** and this explains for the lower SPP and lower OPP in quartile 1 and hence higher number of glaucoma cases in that quartile.

**Table 13**

| | **Mean** | **Min** | **Max** | **Standard Deviation** | **Pearson Correlation** | **Significance (2-tailed)** |
|---|---|---|---|---|---|---|
| **SPP (mmHg)** | 118.26 | 78.00 | 162.00 | **16.95** | **.778** | **0.01 level** |
| **OPP (mm Hg)** | 55.34 | 37.78 | 66.67 | **5.58** | | |

## DISTRIBUTION OF DIASTOLIC PERFUSION PRESSURE:

**Figure 44**




The quartile1 patients had a lower DPP <60 mmHg ,quartile 2 between 60 mmHg - 66.24mm Hg, quartile 3 between 66.25 mm Hg –72 mmHg and quartile 4 had DPP >72mm Hg .

## CORELATION BETWEEN DIASTOLIC PERFUSION PRESSURE AND OCULAR PERFUSION PRESSURE:

**Figure 45**

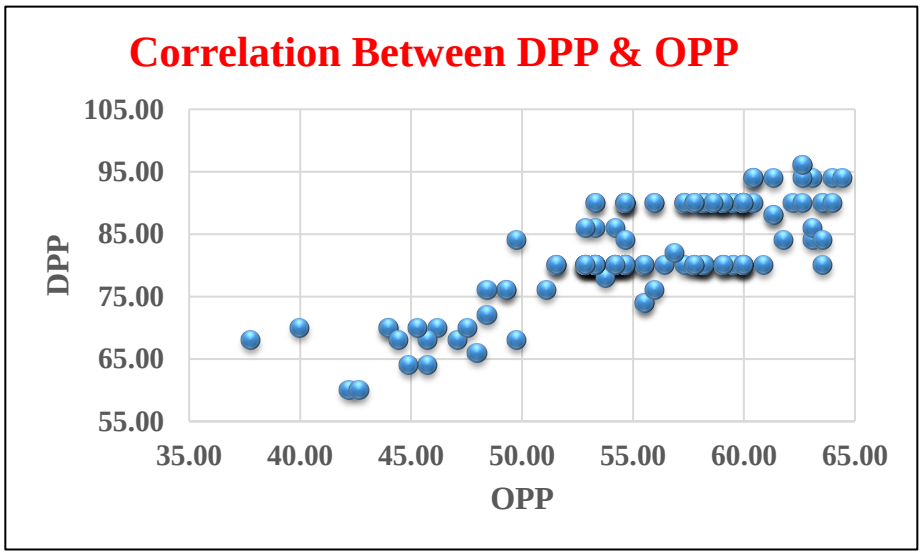



**Table 14**

| | Mean | Min | Max | Standard Deviation | Pearson Correlation | Significance (2-tailed) |
|---|---|---|---|---|---|---|
| **DPP (mmHg)** | 66.24 | 46.00 | 80.00 | **7.45** | **.714** | **0.01 level** |
| **OPP (mm Hg)** | 55.34 | 37.78 | 66.67 | **5.58** | | |

A significant **positive corelation can be observed between DPP and MOPP** in our study which is also **significant statistically** and this explains for the lower DPP and lower OPP in quartile 1 and a higher incidence of glaucoma cases in that quartile.

## COMPARISON BETWEEN MAP ,MOPP ,SPP AND DPP:

**Table 15**

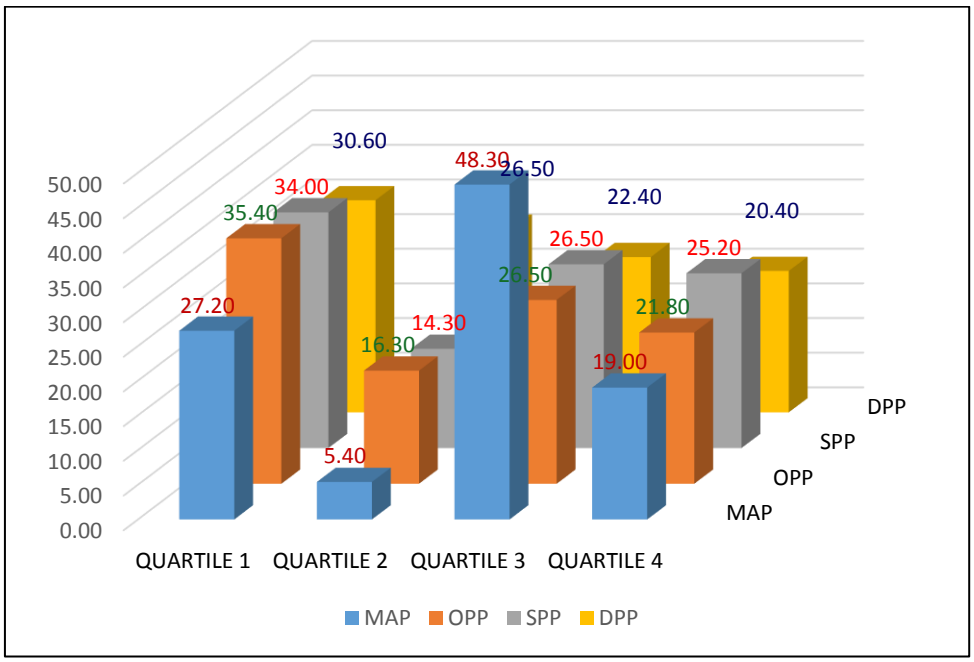



**Figure 46**

## CORELATION BETWEEN MAP ,MOPP ,SPP AND DPP:

**Table 16**

| | **Mean** | **Standard Deviation** | **Pearson Correlation** | **Significance (2-tailed)** |
|---|---|---|---|---|
| **MAP (mm Hg)** | 99.77 | 9.22 | .939 | **0.01 level** |
| **SPP (mm Hg)** | 118.26 | 16.95 | .778 | **0.01 level** |
| **DPP (mm Hg)** | 66.24 | 7.45 | .714 | **0.01 level** |

Our study showed a significant corelation between MAP,SPP,DPP and OPP which was statistically proven using Pearsons Coefficient at 0.01 level.

**DISTRIBUTION OF STUDY POPULATION:**

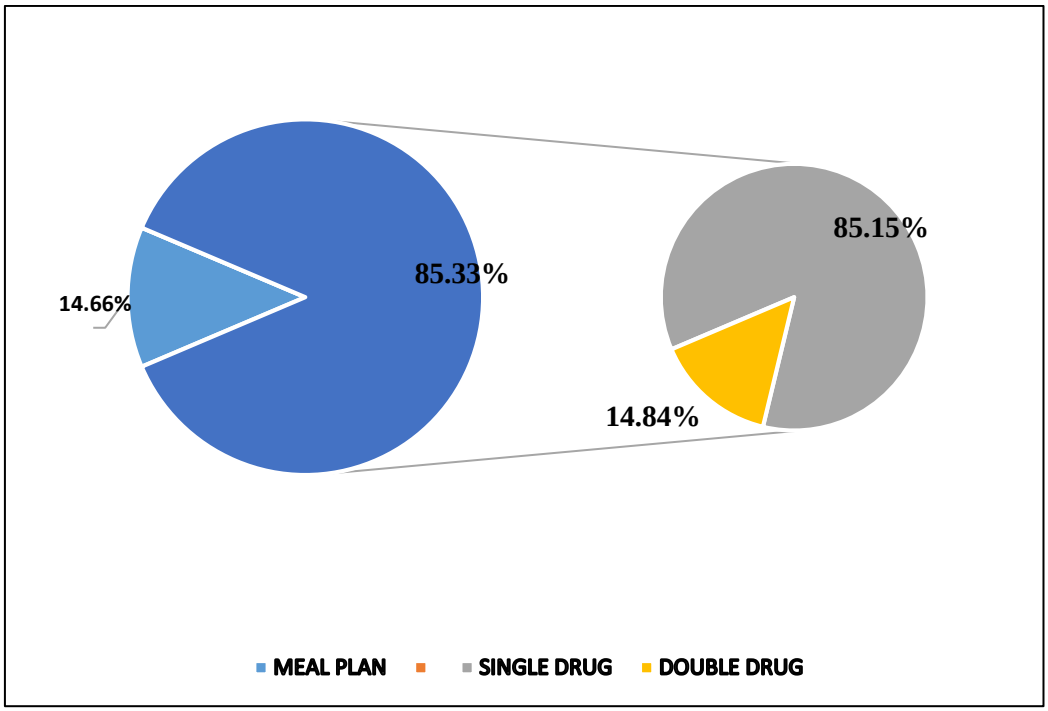



**Figure 47**

Among the study subjects of 150, 22 patients(14.66%) were on plan and 128 patients(85.33%) were on antihypertensive drugs. Among the 85.33%, the majority of 85.15% patients were on single drug regimen and 14.84% patients were on double drug regimen.

## DISTRIBUTIONOF MEAL PLAN PATIENTS:

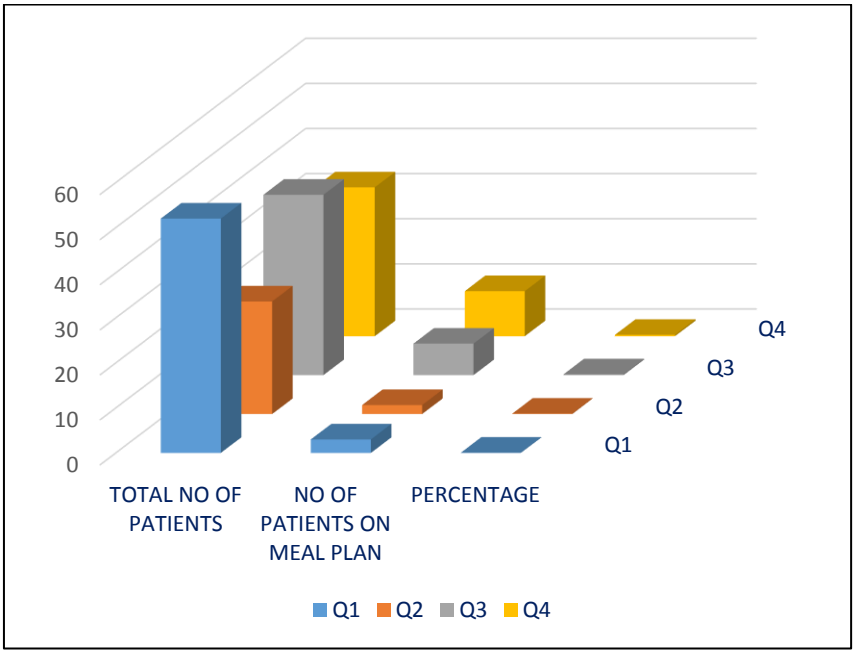



**Figure 48**

**Table 17**

| QUARTILE | Q1 | Q2 | Q3 | Q4 |
|---|---|---|---|---|
| TOTAL NO OF PATIENTS | 52 | 25 | 40 | 33 |
| NO OF PATIENTS ON MEAL PLAN | 3 | 2 | 7 | 10 |
| PERCENTAGE | **5.76%** | **8%** | **17.50%** | **30.30%** |

The majority of Meal plan patients belonged to 4th quartile accounting for 30.30% followed by 17.50% in 3rd quartile ,8% in 2nd quartile and 5.76% in 1st quartile.

**DISTRIBUTION OF SINGLE DRUG REGIMEN :**

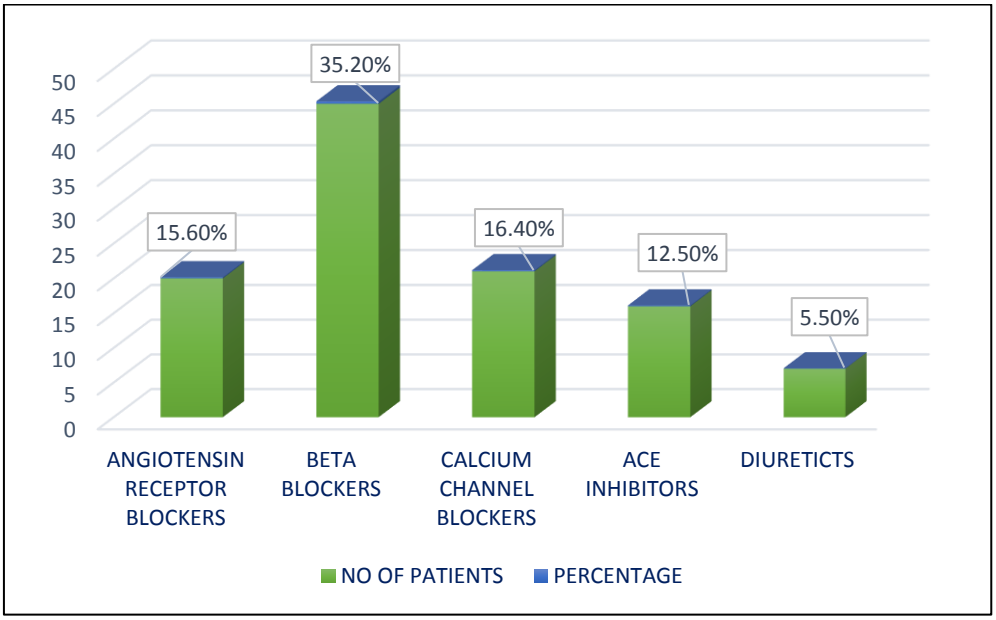



**Figure 49**

**Table 18**

| ANTI-HYPERTENSIVE DRUGS | NO OF PATIENTS | % |
|---|---|---|
| **ANGIOTENSIN RECEPTOR BLOCKERS** | 20 | **15.60%** |
| **BETA BLOCKERS** | 45 | **35.20%** |
| **CALCIUM CHANNEL BLOCKERS** | 21 | **16.40%** |
| **ACE INHIBITORS** | 16 | **12.50%** |
| **DIURETICTS** | 7 | **5.50%** |

Beta blockers was the most commonly used drug in our study subjects (35.2%), followed by CC blockers 16.4%,AR Blockers 15.60%,ACE Inhibitors 12,5% and Diuretics 5.5%.

## DISTRIBUTION OFDOUBLE DRUG REGIMEN:

The total number of patients on double drug includes 19.The following table depicts details of drugs use in the double drug regimen.

**Table 19**

| | NO OF PATIENTS | % |
|---|---|---|
| **BETA BLOCKERS** | 19 | **100.00%** |
| **CALCIUM CHANNEL BLOCKERS** | 11 | **57.90%** |
| **ACE INHIBITORS** | 7 | **36.80%** |
| **DIURETICTS** | 1 | **5.30%** |

**DOUBLE DRUG REGIMEN DISTRIBUTION**

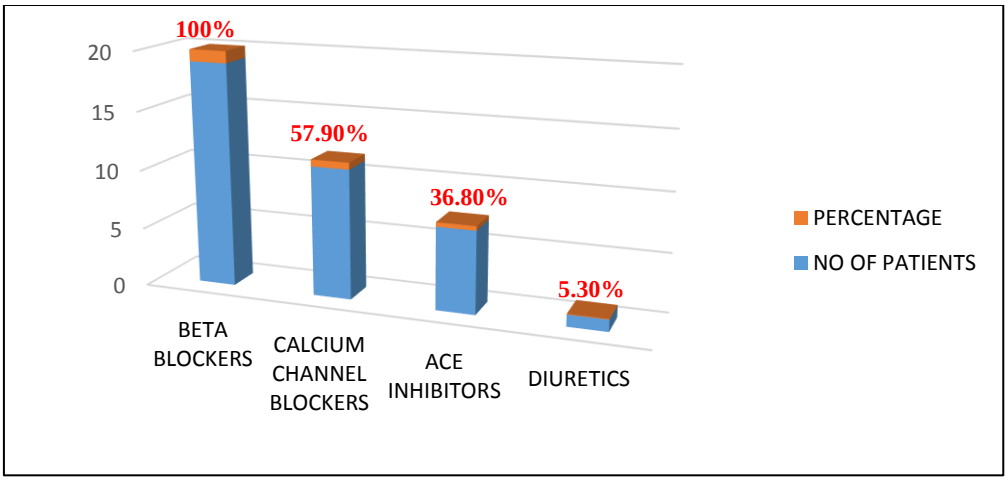



**Figure 50**

In our study subjects, beta blockers was most frequently used in combination with other group of antihypertensives. Almost 57.9% patients have been found to use CC blockers .ACE inhibitors use was found in 36.8% and the least combined drug in our study was diuretics which accounted for 5.3%,which could be explained by the fact that we have excluded patients with hypertension from secondary hypertension from our study.

## QUARTILE WISE ANTI-HYPERTENSIVE DISTRIBUTION:

**Figure 51**

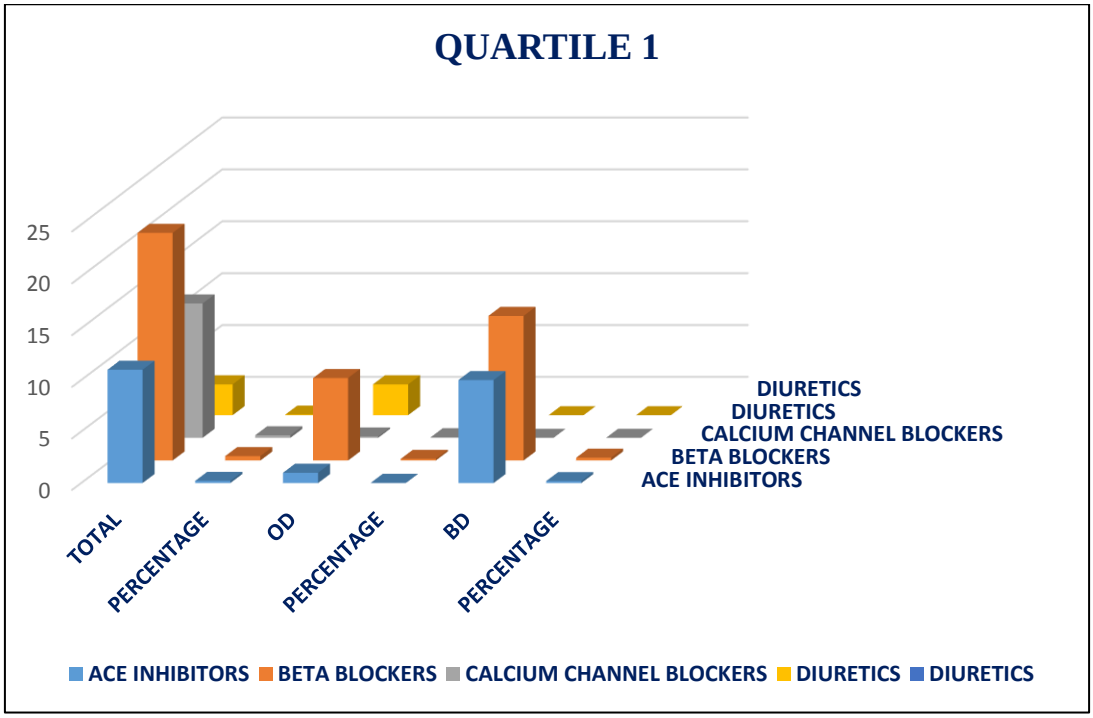



**Table 20**

| | AR BLOCKERS | ACE INHIBITORS | BETA BLOCKERS | CALCIUM CHANNEL BLOCKERS | DIURETICS |
|---|---|---|---|---|---|
| TOTAL | 10 | 11 | 22 | 13 | 3 |
| PERCENTAGE | 19.20% | 21.20% | 42.30% | 25.00% | 5.80% |
| OD | 10 | 1 | 8 | 13.00% | 3 |
| PERCENTAGE | 19.20% | 1.90% | 15.40% | 5.80% | 5.80% |
| BD | 0 | 10 | 14 | 0 | 0 |
| PERCENTAGE | 0.00% | 19.20% | 26.90% | 0.00% | 0.00% |

In the quartile 1 group,Betablockers and ACE inhibitors were used at a BD dosage,accounting for 42.3% and 21.2% respectively.The above drugs were found to used in combination and this explains the lower OPP in this group further accounting for the higher number of glaucoma cases in this group. Other drugs in the group were used only in OD dosage.

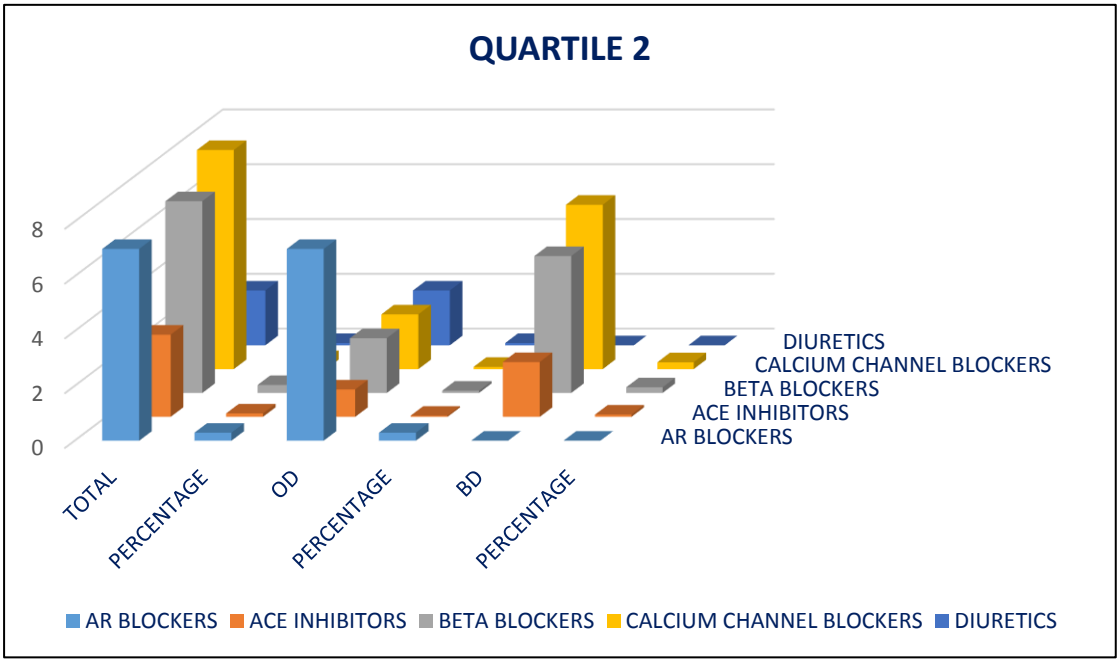



**Figure 52**

**Table 21**

| | AR BLOCKERS | ACE INHIBITORS | BETA BLOCKERS | CALCIUM CHANNEL BLOCKERS | DIURETICS |
|---|---|---|---|---|---|
| **TOTAL** | **7** | **3** | **7** | **8** | **2** |
| **PERCENTAGE** | **29.20%** | **12.50%** | **29.20%** | **33.30%** | **8.30%** |
| **OD** | **7** | **1** | **2** | **2** | **2** |
| **PERCENTAGE** | **29.20%** | **4.20%** | **8.30%** | **8.30%** | **8.30%** |
| **BD** | **0** | **2** | **5** | **6** | **0** |
| **PERCENTAGE** | **0.00%** | **8.30%** | **20.80%** | **25.00%** | **0.00%** |

Among the quartile 2 patients,majority of the patients were found to use CC blockers in BD dosage at 33.3% followed by AR Blockers and Beta blockers at BD Dosage accounting for 29.2%.The least common group among the drugs was diuretics at 8.3% at OD dosage.

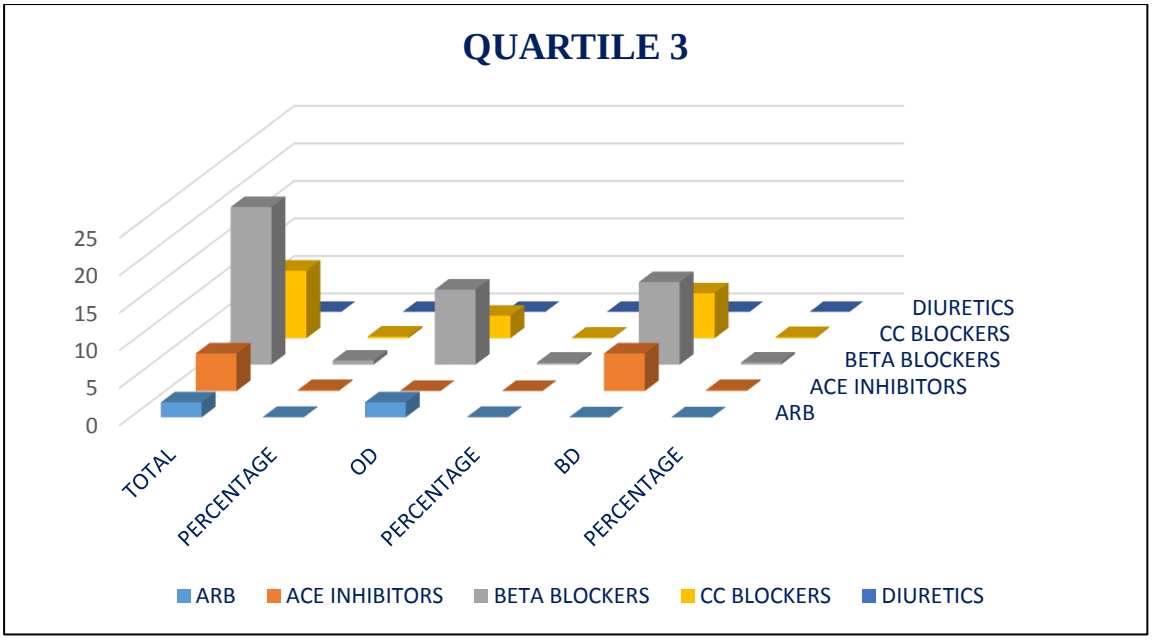



**Figure 53**

**Table 22**

| | AR BLOCKERS | ACE INHIBITORS | BETA BLOCKERS | CALCIUM CHANNEL BLOCKERS | DIURETICS |
|---|---|---|---|---|---|
| **TOTAL** | **2** | **5** | **21** | **9** | **0** |
| **PERCENTAGE** | **5.10%** | **12.80%** | **53.80%** | **23.10%** | **0.00%** |
| **OD** | **2** | **0** | **10** | **3** | **0** |
| **PERCENTAGE** | **5.10%** | **0.00%** | **25.60%** | **7.70%** | **0.00%** |
| **BD** | **0** | **5** | **11** | **6** | **0** |
| **PERCENTAGE** | **0.00%** | **12.80%** | **28.20%** | **15.40%** | **0.00%** |

In Quartile 3,the major drugs used was beta blockers followed by CC Blockers in BD dosage percentage of 53.8 and 23.1 respectively.AR Blockers was found to be used in OD dosage at 5.10%.

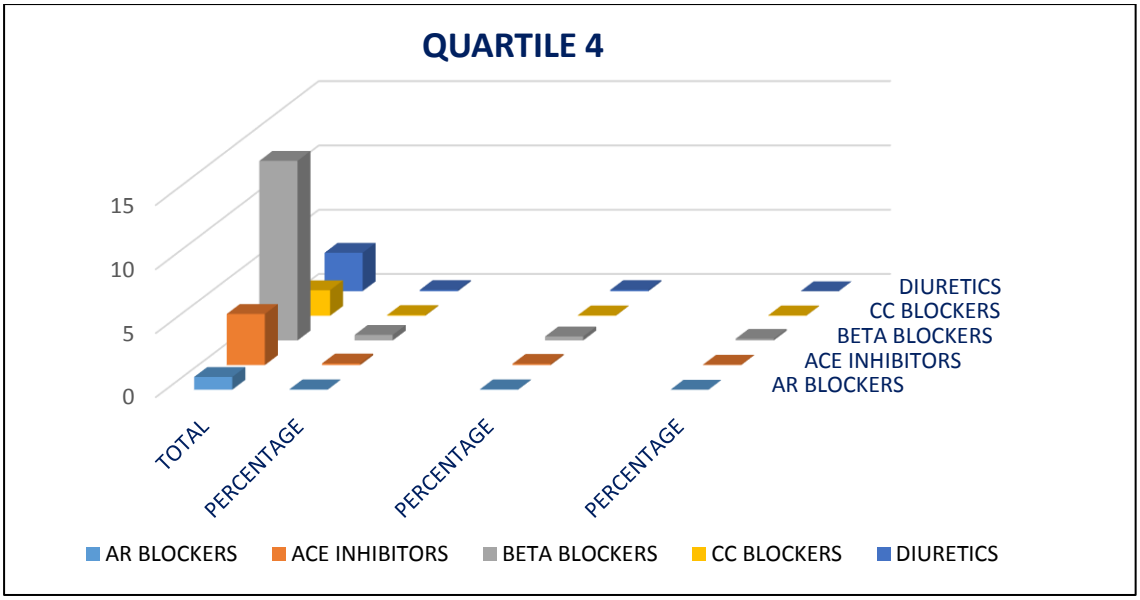



**Figure 54**

**Table 23**

| | AR BLOCKERS | ACE INHIBITORS | BETA BLOCKERS | CALCIUM CHANNEL BLOCKERS | DIURETICS |
|---|---|---|---|---|---|
| **TOTAL** | **1** | **4** | **14** | **2** | **3** |
| **PERCENTAGE** | **3.10%** | **12.50%** | **43.80%** | **6.30%** | **9.40%** |
| **OD** | **1** | **3** | **10** | **1** | **3** |
| **PERCENTAGE** | **3.10%** | **9.40%** | **31.30%** | **3.10%** | **9.40%** |
| **BD** | **0** | **1** | **4** | **1** | **0** |
| **PERCENTAGE** | **0.00%** | **3.10%** | **12.50%** | **3.10%** | **0.00%** |

Beta blockers was the most common drug used in this quartile as at 43.80%.The next common drug used was ACE inhibitors at 12.5%.Mostly the drugs were used at a OD dosage in this group.

## OVERALL DRUG DISTRIBUTION:

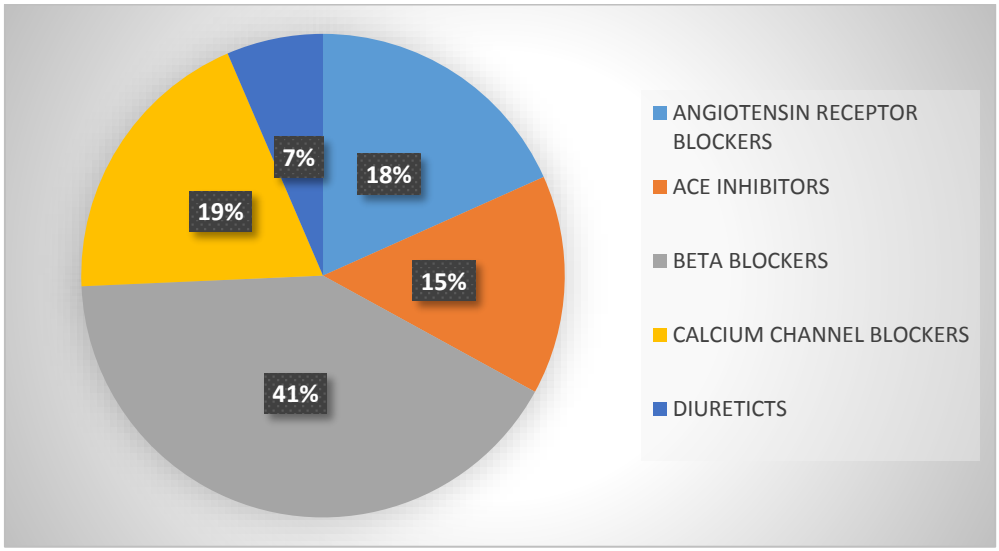



**Figure 55**

**Table 24**

| ANTI-HYPER-TENSIVE DRUG | ANGIOTENSIN RECEPTOR BLOCKERS | ACE INHIBITORS | BETA BLOCKERS | CALCIUM CHANNEL BLOCKERS | DIURE-TICS |
|---|---|---|---|---|---|
| PERCENTAGE | 18% | 15% | 41% | 19% | 7% |

Beta blockers was the most commonly used drug among the study population followed by calcium channel blockers. Diuretics was the less common drug used by our study population.

## DISCUSSION

The present study was designed to ascertain the relationship between systemic hypertension and ocular perfusion pressure among hypertensive patients who presented to our hospital . Our study included 150 patients with systemic hypertension on meal plan and anti-hypertensive drugs. The study revealed a male preponderance with Male : female ratio 2:1(20) since the incidence of hypertension as such was more in males.

The mean age group among males were 57.5yrs years and females were 56.8 years and the **most common age group** was 55-60yrs, with median **57.29 ± 6.10 years**. This is comparable to the **Beaver Dam study** which reported similar results.

The **mean IOP** in our study was **16.52 ± 3.06 mm Hg** which is comparable with the mean age of distribution of 15.3 ± 3.5mm Hg (median 15.0 mm Hg) reported by Zheng et tal seen in Singapore Malay Eye Study(21). The **mean SBP** in our study was **134.45 ± 18.45 mm Hg** and **mean DBP** was **82.44±7.68mm Hg**.

The **mean OPP** in our study was **55.34 ± 5.58 mm Hg, mean SPP value 118.26± 16.95mm Hg** and **mean DPP value 66.24 ± 7.45 mmHg**(21)**.** There were 9 cases of **glaucoma** in our study,7 cases of POAG and 2 cases of NTG which totally accounts for **6%** of study population.(21)

In our study, presence of systemic hypertension or elevation of IOP alone did not lead to increased likelihood of developing glaucoma. The frequency of occurrence of open angle glaucoma was found to be more in a smaller group, which had a lower perfusion pressure due to lower extremes of blood pressure (Averaging around <134/82 mmHg)

Hence, from the analysis, we come to know that patients who are on rigorous antihypertensive treatment or due to nocturnal hypotension have low OPP are those who are at increased risk for development of glaucoma. A direct and clear relationship between BP and glaucomatous damage has however not been established clearly till now(22) Association between systemic hypertension and POAG has been evaluated by various population-based studies.

In our study POAG was found mostly in subjects with low DPP <60mmHg and decreased MOPP <53 mmHg , **mean DPP value of 66.24 ± 7.45 mm Hg** . This in accordance with other studies such as Rotterdam eye study,Baltimore eye study and Egna-Neumarket Study(23,24)

**Rotterdam Eye Study** which was done to study prevalence of OAG in patients receiving antihypertensives. , DOPP < 50mmHg had increased risk of OAG.[12] (24)

**Baltimore Eye Survey** reported that cases who had OPP <30 mmHg, there was 6 fold higher risk of development of OAG.(20)

**Egna-Neumarket Study** reported increased risk of OAG was associated with DBP <50 mmHg.(25)

**Proyecto VER study**, 3-fold higher risk was observed among subjects with DBP <45 mmHg of glaucoma was decreased DBP, decreased SBP, decreased MOPP (<40 mmHg).(23)

In our study subjects ,Quartile 1 patients who had a systolic perfusion pressure <106mm Hg had a lower MOPP among all.There was a significant positive corelation between SPP AND MOPP(Pearson Coeff) in our study.Therefore **Lower the SPP,Lower is the MOPP**. Hence subjects with **lower SPP are at a higher risk for glaucoma.** This is similar with the results from Early Manifest Glaucoma Trail, in which patients with **lower SPP at baseline progressed faster than their counterparts** and had->50% higher risk.(20)

The **Thessaloniki Eye Study**,also found increased disc cupping and decreased rim in those on antihypertensives making possible explanation that marked lowering of BP in susceptible individuals may lead to optic disc changes. (19)

In **Los Angeles Latino Study**, also increased prevalence of OAG was found with lower MOPP, DOPP, SOPP(17) .From these studies, it is understood that people on antihypertensive medications have an increased risk, which may be related to the bedtime dosing or reduction in nocturnal BP.

The **Singapore Malay Eye Study** has reported that low DBP, low MOPP and low DOPP were independent risk factors for OAG. However, only a small proportion of the OAG cases had the lowest levels of MOPP (≤46 mmHg) and DOPP (≤56 mmHg) (32.1 and 35.1% respectively)(21)

Similar findings of increased risk of developing glaucoma with lower diastolic, systolic or mean perfusion pressures have been reported in various other population based studies (18)(26,27)

**Pache and Flammer**(28) reported hypotension and in particular, a nocturnal drop in BP as an important risk factor for OAG.

Despite the challenge in comparing different studies because of the great variability in defining and classifying 'dips' and the wide range in the number of blood pressure measurements as well as the time interval that are used to define day and night, most reports suggest a correlation between nocturnal hypotension and glaucoma and also nocturnal fluctuations and glaucoma (11,12,21)(Hayreh et al. 1994; Detry et al. 1996; Graham & Drance 1999; Orgul € et al. 1999; Flammer et al. 2002)

## ANTI-HYPERTENSIVES AND GLAUCOMA

In our study the Beta blockers was the most used drug at 41 % followed by CC Blockers at 19%, AR Blockers at 18 %, ,ACE inhibitors at 15 %, and Diuretics at 7 %.

In quarile 1 the majority of patients were using combination of beta blockers along with ACE inhibitors or AR Blockers at BD dosage which caused a lower OPP when compared to the others. The next commonly used drug in our study population was CC Blockers at 19%

The incidence of glaucoma was higher in patients of Quartile 1 using Double drug regimen at BD dosage which could have caused nocturnal hypotension and predisposing to glaucoma. Therefore Q 1 constituter the high risk group who need need ophthalmic evaluation,Q2 and Q3 the at risk group who must be placed under periodic ophthalmic surveillance and Q4,the safer group among the others .

Specific antihypertensive agents could have varying effects on OAG. Recent incidence data from the **Rotterdam Eye Study** revealed an **increased risk of OAG in users of calcium channel antagonists** after 6.5 years of follow-up (RR=1.9 (95% CI: 1.1, 3.3)).[15] These agents decrease BP without affecting IOP, thus reducing ocular perfusion pressure, which could explain the findings. On the other hand, the same study found a non-significant trend toward a reduced risk in persons using beta-blockers.(29)

According to **Deb A tal** (19) and **Musken et al**(29) found that subjects on antihypertensive medications had two- to three-fold increased likelihood of having glaucoma or glaucoma suspect. One potential reason may be related to the bedtime dosing of the antihypertensive medications which cause a drop in nocturnal BP and subsequent reduction in ONH perfusion.lower the MOPP, greater the risk of developing glaucoma.

According to **Langman et tal** (30) differential risk of glaucoma was found in in takers of ACE inhibitors and calcium channel blocking agents, from those taking beta blocking agents

In The Los Angeles Latino Eye Study, the authors had found a strong association between OAG prevalence low OPP and. Moreover, the study showed that both low DBP and high SBP (DBP ≤ 60 mmHg and SBP = 161–170 mmHg and >170 mmHg respectively) were associated with an increased prevalence of OAG (OR = 1.9; 2.0; 2.1 respectively), indicating that patients at both extremes of the blood pressure spectrum are at greater risk it is understood that people on antihypertensive medications have an increased risk, which may be related to the bedtime dosing or reduction in nocturnal BP.(17)

The significant correlation of retinal hemodynamics to mean arterial blood pressure and ocular perfusion pressure might reflect impaired autoregulation in glaucoma. This is in good agreement with other studies indicating that an abnormal association exists between blood pressure and ocular blood flow in glaucoma .

It may be hypothesized that

(i) patients with low DBP suffer from low OPP at the ONH
(ii) those with high SBP may develop a decrease in vessel diameter,

which, over time may cause arteriosclerosis that could compromised vascular autoregulation, as well as impair nutrient exchange in the capillary beds at the ONH. These hypotheses not only provide a possible explanation for the discrepancy among the epidemiological studies but also indicate that low OPP can occur secondary to high IOP, low or high blood pressure or atherosclerosis.

## SUMMARY

- **150** patients with systemic hypertension were studied.
- Study included 94 males and 56 females
- The mean age group in males were **57.5 yrs** and in females were **56.8 yrs**
- The most common age range was 55 -60 yrs
- Among 150 patients,22 were on Meal Plan and 128 were on anti-hypertensive drugs
- Average BP 134/82mm Hg, Lowest BP recorded was 100/60 mmHg
- Patients were divided into 4 quartiles based on MOPP
- Totally **9 cases of glaucoma** were diagnosed among the study subjects
- Among the 4 Quartiles, Subjects in Q1 had highest number of glaucoma cases
- Subjects in Q1 had lower MOPP,SPP and DPP when compared to others and comprised the at risk group
- Majority of **the diagnosed glaucoma cases** were using **Double drug regimen in BD dosage** in Quartile 1
- Patients with **SPP < 106mm Hg , DPP < 60mm Hg** were found to be at a higher risk for glaucoma.
- **Lower OPP, Lower SPP and Lower DPP are associated with a higher risk of glaucoma which is statistically proven.**
- **DPP** is **better predictor of perfusion status of optic nerve head** and **DPP < 60 mm Hg signifies higher risk to the patient.**

- **Beta blockers** was most commonly used drug among our subjects **(41%)** followed by **Calcium channel blockers(19%), Angiotensin Receptor blockers(18%)**, **ACE Inhibitors(15%)** and **Diuretics(7%)**
- Beta blockers was found to be frequently combined with other anti-hypertensive drugs such as ACE Inhibitors and Angiotensin Receptor Blockers in our study

## LIMITATIONS OF STUDY

One of the main limitations of our study regarding OPP is that blood pressure and IOPs measurements are generally performed only once and during daytime. A single reading often does not represent a patient's state at all times and there may also be a systematic error related to the patient's alerting reaction to the blood pressure and IOP measurement procedures.

Moreover, any circadian influence on blood pressure or on IOP will be overlooked, as will be the influence of the fluctuation of both parameter

The real physiological status of ocular perfusion may not be reflected by the calculation of MOPP which is done using theoretical formula. Direct measurement of ocular blood flow could result in different outcomes. (31)

# CONCLUSION

The results of the present study suggest that MOPP plays a potential role in the pathogenesis of glaucoma in subjects on antihypertensive drugs. Retinal optic nerve head blood flow may be difficult, Doppler OCT may be needed to measure the same. This difficulty of measuring optic nerve head flow is the main reason why the relationship between ocular perfusion and glaucoma is not clinically utilised.

Its not possible to increase ocular perfusion pressure as a part of glaucoma treatment. In exception, it maybe used to decrease rigorous antihypertensive treatment and to avoid the night dosage in patient with systemic hypertension to prevent very low OPPs. This maintains the autoregulatory mechanism by maintaining not only constant blood flow (vascular reserve), but also preserves neuronal functions (functional reserve).

**Diastolic perfusion pressure is better predictor of lower ocular perfusion** when compared to OPP and SPP**. DPP < 60 mm Hg signifies higher glaucoma risk to the patient.**

Moreover patients on Combined regimen of Beta blockers along with calcium channel blockers/ACE Inhibitors though attain adequate BP control, they are **at risk for glaucoma because of lower OPP values.**

In our study, OPP measurement was done as a noninvasive, easy to obtain, inexpensive clinical data. It will be useful in rural settings where more number of patients are seen regularly on a daily basis, it will be a cost efficient, easy to screen, and to obtain data, which can be used as a monitoring tool for patients who are on antihypertensive treatment.

OPP can be a potential clinical screening tool to identify those patients on overdosage with antihypertensive medications. It can find out the risky patients among those with nocturnal hypotension, who may develop glaucoma or progress after a period of hypoperperfusion of optic nerve head during night.

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31. Quaranta L, Katsanos A, Russo A, Riva I. 24-hour Intraocular Pressure and Ocular Perfusion Pressure in Glaucoma. Surv Ophthalmol. 2013 Jan;58(1):26–41.

## PROFORMA

Serial no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

History of disease :

Treatment history :

Duration of treatment :

Drug history :

Ass. Systemic illness :

Family history :

**BASELINE CLINICAL EXAMINATION:**

Blood Pressure ( Average of 3 readings) :

**BASELINE OCULAR EXAMINATION**

| | **RE** | **LE** |
|---|---|---|
| Vision | | |
| Eyelids and lashes | | |
| Extraocular movements | | |
| *Slit lamp examination* | | |
| Conjunctiva | | |
| Cornea | | |
| Anterior chamber | | |
| Iris | | |
| Pupil | | |
| Lens | | |
| Colour vision | | |
| Fields | | |
| Gonioscopy | | |
| IOP | | |
| Fundus Examination | | |
| Fundus Photography | | |
| Pachymetry(whenever necessary) | | |
| Optical Coherence Tomography(whenever necessary) | | |

**DIAGNOSIS:**

**<u>சுய ஒப்புதல் படிவம</u>**

ஆராய்ச்சி நிலையம் மருத்துவமனை : அரசு ஸ்டான்லி மருத்துவ கல்லூரி

பங்கு பெறுபவரின் பெயர் :

பஙகு பெறுபவரின் எண் :

பங்கு பெறுபவர் இதனை ( ) குறிக்கவும்

நாள் பட்ட இரத்தகொதிப்பு நோய் உள்ள நோயாளிகளுக்கு இந்நோயினால் கண் அழுத்தத்தில் ஏற்படும் பாதிப்பை முன்னமே கண்டறிய ஓர் ஆய்வு.

ஆய்வு பற்றிய விபரங்கள் எனக்கு விளக்கப்பட்டன. என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பு அளிக்கப்பட்டது.

இந்த ஆராய்ச்சியின் விபரங்களும், அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

மேற்கண்ட பரிசோதனையின் பொது ஏற்பட கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு

மனமார சம்மதிக்கிறேன்.

பங்கு பெறுபவரின் கையொப்பம்

*ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உரிமைகள்*

இந்த ஆய்வில் தங்களின் பங்கேற்பு தன்னிச்சையானது. மற்ற காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்வில் இருந்து எந்த நேரத்திலும் விலகிக் கொள்ளலாம்.

எந்த ஒரு நேரத்திலும் உங்களுக்கு திருப்தி இல்லை என்று உணர்ந்தாலோ அல்லது வேறு ஏதேனும் உடல் நல குறைவு உண்டானாலோ உங்களை கவனித்து வரும் மருத்துவரிடம் உடனடியாக தெரிவிக்கவும். உங்களுக்கு சிகிச்சை பொருத்தமாக இருக்காது என தோன்றினால் உடனடியாக நிறுத்தப்படும்.

வேறு ஏதேனும் கேள்விகள் பிரச்சனைகள் பற்றி நீங்கள் கேட்க விரும்பினால் கீழ்கண்ட நபரை தொடர்பு கொள்ளவும்.

மருத்துவர் .அ. ரேவதி
முதுநிலை மருத்துவ மாணவர்,
கண் இயல் துறை,
அரசு ஸ்டான்லி மருத்துவ கல்லூரி மருத்துவமனை,
சென்னை.
தொலைபேசி எண்: 9444969409

# ANNEXURES

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## PROFORMA

Serial no. :

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

History of disease :

Treatment history :

Duration of treatment :

Drug history :

Ass. Systemic illness :

Family history :

**BASELINE CLINICAL EXAMINATION:**

Blood Pressure ( Average of 3 readings) :

**BASELINE OCULAR EXAMINATION**

| | **RE** | **LE** |
|---|---|---|
| Vision | | |
| Eyelids and lashes | | |
| Extraocular movements | | |
| *Slit lamp examination* | | |
| Conjunctiva | | |
| Cornea | | |
| Anterior chamber | | |
| Iris | | |
| Pupil | | |
| Lens | | |
| Colour vision | | |
| Fields | | |
| Gonioscopy | | |
| IOP | | |
| Fundus Examination | | |
| Fundus Photography | | |
| Pachymetry(whenever necessary) | | |
| Optical Coherence Tomography(whenever necessary) | | |

**DIAGNOSIS:**

**<u>சுய ஒப்புதல் படிவம</u>**

ஆராய்ச்சி நிலையம் மருத்துவமனை : அரசு ஸ்டான்லி மருத்துவ கல்லூரி

பங்கு பெறுபவரின் பெயர் :

பஙகு பெறுபவரின் எண் :

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இந்த ஆராய்ச்சியின் விபரங்களும், அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

மேற்கண்ட பரிசோதனையின் பொது ஏற்பட கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு

மனமார சம்மதிக்கிறேன்.

பங்கு பெறுபவரின் கையொப்பம்

*ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உரிமைகள்*

இந்த ஆய்வில் தங்களின் பங்கேற்பு தன்னிச்சையானது. மற்ற காரணங்கள் எதையும் கூறாமலேயே நீங்கள் இந்த ஆய்வில் இருந்து எந்த நேரத்திலும் விலகிக் கொள்ளலாம்.

எந்த ஒரு நேரத்திலும் உங்களுக்கு திருப்தி இல்லை என்று உணர்ந்தாலோ அல்லது வேறு ஏதேனும் உடல் நல குறைவு உண்டானாலோ உங்களை கவனித்து வரும் மருத்துவரிடம் உடனடியாக தெரிவிக்கவும். உங்களுக்கு சிகிச்சை பொருத்தமாக இருக்காது என தோன்றினால் உடனடியாக நிறுத்தப்படும்.

வேறு ஏதேனும் கேள்விகள் பிரச்சனைகள் பற்றி நீங்கள் கேட்க விரும்பினால் கீழ்கண்ட நபரை தொடர்பு கொள்ளவும்.

மருத்துவர் .அ. ரேவதி
முதுநிலை மருத்துவ மாணவர்,
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சென்னை.
தொலைபேசி எண்: 9444969409

## KEY TO MASTER CHART

**M** - Male

**F** - Female

**RE** - Right Eye

**LE** - Left Eye

**IOP** - Intraocular pressure

**BP** - Blood Pressure

**OPP** - Ocular Perfusion Pressure

**SBP** - Systolic blood pressure

**DBP** - Diastolic blood pressure

**MAP** - Mean Arterial pressure

**MOPP** - Mean Ocular Perfusion Pressure

**SPP** - Systolic Perfusion Pressure

**DPP** - Diastolic Perfusion Pressure

| | NAME | AGE | sex | COMORBIDITY | MEAL PLAN/ORAL DRUG | Angio receptor blockers | dosage | T.ATEN | DOSAGE | T.AMLO | DOSAGE | S | DOSAGE | DIURETICTS | DOSAGE | RE FUNDUS | IOP RE | SBP | DBP | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) | LE FUNDUS | LE | SBP | | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Baskar | 50 | M | | N | | | Y | 2BD | Y | 2BD | | | | | GLAUC I | 20 | 100 | 70 | 30 | 80.00 | 80 | 50 | 40.00 | GLAUC DI | 22 | 100 | 70 | 30 | 80.00 | 78 | 48 | 38.67 |
| 2 | Balaji | 55 | M | | N | | | Y | 2BD | | | Y | 2BD | | | RE GI DI | 22 | 100 | 68 | 32 | 78.67 | 78 | 46 | 37.78 | LE GL OPT | 22 | 100 | 68 | 32 | 78.67 | 78 | 46 | 37.78 |
| 3 | Rajeswari | 62 | F | | N | | | | | Y | bd | | | | | GII | 10 | 108 | 64 | 44 | 78.67 | 98 | 54 | 45.78 | GII | 12 | 108 | 64 | 44 | 78.67 | 96 | 52 | 44.44 |
| 4 | Sabarish | 48 | M | | N | | | | | Y | bd | | | | | GI | 14 | 100 | 70 | 30 | 80.00 | 86 | 56 | 44.00 | GI | 14 | 100 | 70 | 30 | 80.00 | 86 | 56 | 44.00 |
| 5 | Palani | 60 | M | | N | | | y | od | Y | bd | | | | | GII | 12 | 112 | 68 | 44 | 82.67 | 100 | 56 | 47.11 | GII | 10 | 112 | 68 | 44 | 82.67 | 102 | 58 | 48.44 |
| 6 | Prakasam | 52 | M | | N | | | y | od | Y | bd | | | | | G1 | 16 | 114 | 76 | 38 | 88.67 | 98 | 60 | 48.44 | GI | 14 | 114 | 76 | 38 | 88.67 | 100 | 62 | 49.78 |
| 7 | Rajathi | 62 | F | CAD | N | | | | | Y | bd | | | | | GI | 12 | 100 | 70 | 30 | 80.00 | 88 | 58 | 45.33 | GI | 12 | 100 | 70 | 30 | 80.00 | 88 | 58 | 45.33 |
| 8 | Sugunathan | 48 | M | | N | | | | | Y | bd | | | | | GII | 14 | 116 | 64 | 52 | 81.33 | 102 | 50 | 44.89 | GII | 12 | 114 | 60 | 54 | 78.00 | 102 | 48 | 44.00 |
| 9 | Samshudeen | 55 | M | | N | | | | | | | Y | BD | | | GII | 12 | 108 | 60 | 48 | 76.00 | 96 | 48 | 42.67 | GII | 14 | 108 | 60 | 48 | 76.00 | 94 | 46 | 41.33 |
| 10 | Mohideen | 54 | M | | N | | | | | | | Y | BD | | | GI | 14 | 100 | 70 | 30 | 80.00 | 86 | 56 | 44.00 | GI | 14 | 100 | 70 | 30 | 80.00 | 86 | 56 | 44.00 |
| 11 | James | 68 | M | | N | | | | | | | Y | BD | | | GI | 12 | 100 | 68 | 32 | 78.67 | 88 | 56 | 44.44 | GI | 16 | 100 | 68 | 32 | 78.67 | 84 | 52 | 41.78 |
| 12 | Sarika | 58 | F | | N | | | | | | | Y | BD | | | GI | 10 | 104 | 70 | 34 | 81.33 | 94 | 60 | 47.56 | GI | 14 | 104 | 70 | 34 | 81.33 | 90 | 56 | 44.89 |
| 13 | Jasmine | 52 | F | | N | | | | | | | Y | BD | | | N | 12 | 110 | 72 | 38 | 84.67 | 98 | 60 | 48.44 | N | 14 | 110 | 74 | 36 | 86.00 | 96 | 60 | 48.00 |
| 14 | Rafi | 55 | M | | N | Y | OD | | | | | | | | | GII | 14 | 112 | 68 | 44 | 82.67 | 98 | 54 | 45.78 | GIII HTR | 16 | 112 | 68 | 44 | 82.67 | 96 | 52 | 44.44 |
| 15 | Kuruvathai | 63 | F | | N | Y | OD | | | | | | | | | N | 10 | 118 | 68 | 50 | 84.67 | 108 | 58 | 49.78 | N | 10 | 118 | 68 | 50 | 84.67 | 108 | 58 | 49.78 |
| 16 | Subramani | 65 | M | | N | Y | OD | | | | | | | | | N | 14 | 110 | 70 | 40 | 83.33 | 96 | 56 | 46.22 | N | 16 | 110 | 70 | 40 | 83.33 | 94 | 54 | 44.89 |
| 17 | Janani | 45 | F | | N | Y | OD | | | | | | | | | N | 10 | 100 | 60 | 40 | 73.33 | 90 | 50 | 42.22 | N | 10 | 100 | 60 | 40 | 73.33 | 90 | 50 | 42.22 |
| 18 | Ravichandran | 47 | M | | N | Y | OD | | | | | | | | | GI | 12 | 106 | 76 | 30 | 86.00 | 94 | 64 | 49.33 | GII | 14 | 106 | 76 | 30 | 86.00 | 92 | 62 | 48.00 |
| 19 | Annamalai | 59 | M | | N | Y | OD | | | | | | | | | GII | 10 | 114 | 66 | 48 | 82.00 | 104 | 56 | 48.00 | GIII HTR | 12 | 114 | 66 | 48 | 82.00 | 102 | 54 | 46.67 |
| 20 | Kushal Bee | 60 | F | | N | Y | OD | | | | | | | | | N | 16 | 104 | 84 | 20 | 90.67 | 88 | 68 | 49.78 | N | 18 | 104 | 84 | 20 | 90.67 | 86 | 66 | 48.44 |
| 21 | Komala | 62 | F | CAD | N | Y | OD | | | | | | | | | GI | 14 | 112 | 76 | 36 | 88.00 | 98 | 62 | 49.33 | GI | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 |
| 22 | Vinayagavelu | 60 | M | | N | | | y | bd | | | Y | 2BD | | | | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 |
| 23 | Senbagam | 54 | F | | N | | | y | bd | | | Y | 2BD | | | | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 | | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 24 | Kanagaraj | 67 | M | | N | | | y | bd | | | Y | 2BD | | | | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | | 20 | 120 | 90 | 30 | 100.00 | 100 | 70 | 53.33 |
| 25 | Mohd Sulthan | 49 | M | | N | | | | | | | Y | BD | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 |
| 26 | Suryanarayanan | 51 | M | | N | | | | | | | Y | BD | | | N | 18 | 126 | 86 | 40 | 99.33 | 108 | 68 | 54.22 | N | 16 | 126 | 86 | 40 | 99.33 | 110 | 70 | 55.56 |
| 27 | Chandraprakash | 59 | M | | N | | | | | | | Y | BD | | | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 28 | Mukesh | 55 | M | | N | | | y | bd | Y | 2BD | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 29 | Sabarish | 48 | M | | N | | | Y | bd | Y | 2BD | | | | | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 30 | Maharani | 52 | F | | N | | | y | bd | Y | 2BD | | | | | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 31 | Parameshwaran | 60 | M | | N | | | | | Y | BD | | | | | N | 20 | 120 | 90 | 30 | 100.00 | 100 | 70 | 53.33 | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 |
| 32 | Saroja | 62 | F | | N | | | | | Y | BD | | | | | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 33 | Kamala | 64 | F | | N | | | | | Y | BD | | | | | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 34 | Sharadha | 66 | F | | N | | | | | Y | BD | | | | | N | 18 | 122 | 86 | 36 | 98.00 | 104 | 68 | 53.33 | N | 16 | 122 | 86 | 36 | 98.00 | 106 | 70 | 54.67 |
| 35 | Pushpa | 48 | F | | N | | | | | Y | BD | | | | | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 |
| 36 | Pandiraj | 59 | M | | N | | | y | 2BD | Y | OD | | | | | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 37 | Purushotaman | 70 | M | | N | | | | | Y | OD | | | | | GI | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 | GI | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 38 | Subhashri | 60 | F | | Y | | | | | | | | | | | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 39 | Susairaj | 58 | M | | N | | | | | Y | OD | | | | | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 40 | Surendran | 54 | M | | N | | | | | Y | OD | | | | | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 41 | Geetham | 49 | F | | N | Y | OD | | | | | | | | | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |

| | NAME | AGE | sex | COMORBIDITY | MEAL PLAN/ORAL DRUG | Angio receptor blockers | dosage | T.ATEN | DOSAGE | T.AMLO | DOSAGE | S | DOSAGE | DIURETICTS | DOSAGE | RE FUNDUS | IOP RE | SBP | DBP | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) | LE FUNDUS | LE | SBP | | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 42 | Lalitha | 60 | F | | N | Y | OD | | | | | | | | | N | 16 | 126 | 84 | 42 | 98.00 | 110 | 68 | 54.67 | N | 16 | 126 | 84 | 42 | 98.00 | 110 | 68 | 54.67 |
| 43 | Vijayakannan | 54 | M | | N | Y | OD | | | | | | | | | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 |
| 44 | Mary | 59 | F | | N | Y | OD | | | | | | | | | N | 14 | 126 | 80 | 46 | 95.33 | 112 | 66 | 54.22 | N | 14 | 126 | 80 | 46 | 95.33 | 112 | 66 | 54.22 |
| 45 | Kasinathan | 58 | M | | N | Y | OD | | | | | | | | | N | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 46 | Ganeshan | 57 | M | | N | Y | OD | | | | | | | | | N | 20 | 120 | 90 | 30 | 100.00 | 100 | 70 | 53.33 | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 |
| 47 | Chandru | 50 | M | | N | Y | OD | | | | | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 48 | Ganga | 56 | F | | N | Y | OD | | | | | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 |
| 49 | John Paul | 51 | M | | N | Y | OD | | | | | | | | | GI | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | GI | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 |
| 50 | Parameshwaran | 62 | M | | N | | | y | 2od | | | Y | OD | | | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 51 | Udhayamoorthy | 64 | M | | N | | | | | Y | OD | | | | | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 52 | Chellamma | 69 | F | | N | | | | | | | Y | OD | | | N | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 | N | 12 | 120 | 80 | 40 | 93.33 | 108 | 68 | 54.22 |
| 53 | Suguvanam | 56 | M | CAD | N | | | y | od | | | | | Y | OD | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 54 | Madhavan | 54 | M | CAD | N | | | | | | | | | Y | OD | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 55 | Sundaramoorthy | 59 | M | CAD | N | | | | | | | | | Y | OD | N | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 56 | Venugopal | 54 | M | CAD | N | | | | | | | | | Y | OD | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | N | 20 | 120 | 90 | 30 | 100.00 | 100 | 70 | 53.33 |
| 57 | Mohammed Yusu | 60 | M | | N | | | Y | BD | | | | | | | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | N | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 |
| 58 | Pushparaj | 69 | M | | N | | | Y | BD | | | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 |
| 59 | Pitchumani | 64 | M | | N | | | Y | BD | | | | | | | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | N | 20 | 120 | 90 | 30 | 100.00 | 100 | 70 | 53.33 |
| 60 | Sreenevasan | 62 | M | | N | | | Y | BD | | | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 61 | Praveena Mary | 52 | F | | N | | | Y | BD | | | | | | | GI | 20 | 140 | 80 | 60 | 100.00 | 120 | 60 | 53.33 | GI | 18 | 140 | 80 | 60 | 100.00 | 122 | 62 | 54.67 |
| 62 | Karupaiyah | 59 | M | | N | | | Y | BD | | | | | | | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 63 | Rosy | 54 | F | | y | | | | | | | | | | | N | 14 | 120 | 76 | 44 | 90.67 | 106 | 62 | 51.11 | N | 14 | 120 | 76 | 44 | 90.67 | 106 | 62 | 51.11 |
| 64 | Kumaran | 60 | M | | N | | | Y | BD | | | | | | | N | 18 | 120 | 86 | 34 | 97.33 | 102 | 68 | 52.89 | N | 20 | 120 | 86 | 34 | 97.33 | 100 | 66 | 51.56 |
| 65 | Celina | 55 | F | | y | | | | | | | | | | | N | 18 | 140 | 78 | 62 | 98.67 | 122 | 60 | 53.78 | N | 20 | 140 | 78 | 62 | 98.67 | 120 | 58 | 52.44 |
| 66 | Mohammed Ansa | 56 | M | | y | | | | | | | | | | | N | 14 | 126 | 80 | 46 | 95.33 | 112 | 66 | 54.22 | N | 14 | 126 | 80 | 46 | 95.33 | 112 | 66 | 54.22 |
| 67 | Karpagam | 52 | F | | | | | Y | 2OD | | | | | | | N | 16 | 120 | 80 | 40 | 93.33 | 104 | 64 | 51.56 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 68 | Arumugam | 50 | M | | N | | | Y | BD | Y | BD | | | | | GI ,GLAU | 22 | 166 | 76 | 90 | 106.00 | 144 | 54 | 56.00 | GI ,GLAUC | 20 | 166 | 76 | 90 | 106.00 | 146 | 56 | 57.33 |
| 69 | Jagadeeswari | 54 | F | | N | | | | | Y | BD | | | | | N | 14 | 130 | 90 | 40 | 103.33 | 116 | 76 | 59.56 | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 |
| 70 | Krishna Prasad | 59 | M | | N | | | Y | BD | Y | BD | | | | | GII | 12 | 130 | 80 | 50 | 96.67 | 118 | 68 | 56.44 | GII | 14 | 130 | 80 | 50 | 96.67 | 116 | 66 | 55.11 |
| 71 | Selvi | 54 | F | | N | | | Y | BD | | | Y | BD | | | GII,GLA | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 | GII | 22 | 160 | 80 | 80 | 106.67 | 138 | 58 | 56.44 |
| 72 | Muniyammal | 58 | F | | N | | | Y | BD | Y | BD | | | | | GIII | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 | GIII | 18 | 150 | 80 | 70 | 103.33 | 132 | 62 | 56.89 |
| 73 | Joseph raj | 56 | M | | N | | | Y | BD | Y | BD | | | | | GII | 12 | 120 | 90 | 30 | 100.00 | 108 | 78 | 58.67 | GII | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 |
| 74 | Punitha Mary | 50 | F | | N | | | | | Y | BD | | | | | GI | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 | GI | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 |
| 75 | Jagadeeswari | 51 | F | | Y | | | | | | | | | | | N | 10 | 140 | 80 | 60 | 100.00 | 130 | 70 | 60.00 | N | 12 | 140 | 80 | 60 | 100.00 | 128 | 68 | 58.67 |
| 76 | Manoharan | 54 | M | | N | | | Y | BD | | | Y | BD | | | GII | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 | GII | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 |
| 77 | Ramalingam | 60 | M | | N | | | | | | | Y | BD | | | GII | 18 | 160 | 80 | 80 | 106.67 | 142 | 62 | 59.11 | GII | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 |
| 78 | Sathyakumar | 50 | M | | N | | | | | Y | BD | | | | | GI,GLAU | 24 | 164 | 82 | 82 | 109.33 | 140 | 58 | 56.89 | GI,GLAUC | 22 | 164 | 82 | 82 | 109.33 | 142 | 60 | 58.22 |
| 79 | Lakshmi | 59 | F | | N | Y | OD | | | | | | | | | N | 14 | 150 | 80 | 70 | 103.33 | 136 | 66 | 59.56 | N | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 |
| 80 | Amsa | 64 | F | | N | | | | | | | Y | BD | | | GI | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 | GI | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 |
| 81 | Sumathy | 53 | F | CAD | N | | | | | | | Y | BD | | | N | 12 | 120 | 90 | 30 | 100.00 | 108 | 78 | 58.67 | N | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 |
| 82 | Ramu | 53 | M | | N | | | | | | | Y | BD | | | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 | N | 14 | 130 | 90 | 40 | 103.33 | 116 | 76 | 59.56 |

| | NAME | AGE | sex | COMORBIDITY | MEAL PLAN/ORAL DRUG | Angio receptor blockers | dosage | T.ATEN | DOSAGE | T.AMLO | DOSAGE | S | DOSAGE | DIURETICTS | DOSAGE | RE FUNDUS | IOP RE | SBP | DBP | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) | LE FUNDUS | LE | SBP | | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 83 | Sarala | 55 | F | | N | Y | OD | | | | | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 84 | Govind | 59 | M | | N | | | | | Y | BD | | | | | GII | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 | GII | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 |
| 85 | Ranjitham | 60 | M | | N | | | Y | BD | | | | | | | N | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 | N | 20 | 150 | 80 | 70 | 103.33 | 130 | 60 | 55.56 |
| 86 | Ravichandran | 62 | M | | N | | | Y | BD | | | | | | | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 | N | 14 | 130 | 90 | 40 | 103.33 | 116 | 76 | 59.56 |
| 87 | Asokan | 60 | M | | N | | | Y | BD | | | | | | | GI | 18 | 160 | 80 | 80 | 106.67 | 142 | 62 | 59.11 | GI | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 |
| 88 | Charulatha | 54 | F | | N | | | Y | BD | | | | | | | N | 18 | 160 | 80 | 80 | 106.67 | 142 | 62 | 59.11 | N | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 |
| 89 | Saravanamoorthy | 59 | M | | N | | | Y | BD | | | | | | | N | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 | N | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 |
| 90 | Moorthy | 60 | M | CAD | N | | | | | | | | | Y | OD | GI | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 | GI | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 |
| 91 | Rathi meena | 62 | F | CAD | N | | | | | | | | | Y | OD | N | 20 | 150 | 80 | 70 | 103.33 | 130 | 60 | 55.56 | N | 20 | 150 | 70 | 80 | 96.67 | 130 | 50 | 51.11 |
| 92 | Pushpalatha | 59 | F | | N | | | Y | BD | | | | | | | N | 16 | 120 | 90 | 30 | 100.00 | 104 | 74 | 56.00 | N | 16 | 120 | 90 | 30 | 100.00 | 104 | 74 | 56.00 |
| 93 | Raghavan | 60 | M | | N | | | | | Y | OD | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 94 | Joseph | 62 | M | | N | | | | | Y | OD | | | | | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 |
| 95 | Raziya Begum | 63 | F | | N | | | | | Y | OD | | | | | GI | 14 | 150 | 80 | 70 | 103.33 | 136 | 66 | 59.56 | GI | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 |
| 96 | Kannammal | 60 | F | | Y | | | | | | | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 97 | Pushparaj | 58 | M | | N | | | Y | BD | | | | | | | N | 14 | 130 | 90 | 40 | 103.33 | 116 | 76 | 59.56 | N | 14 | 130 | 90 | 40 | 103.33 | 116 | 76 | 59.56 |
| 98 | Komala | 64 | F | | N | | | Y | BD | | | | | | | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 |
| 99 | Chellamma | 55 | F | | Y | | | | | | | | | | | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 | N | 16 | 130 | 90 | 40 | 103.33 | 114 | 74 | 58.22 |
| 100 | Venugopal | 60 | M | | N | Y | OD | | | | | | | | | GI | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 | GI | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 |
| 101 | Jaffer Khan | 54 | M | | N | | | Y | BD | | | | | | | GI | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 | GI | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 |
| 102 | Rafi | 55 | M | | N | | | Y | BD | | | | | | | N | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 | N | 18 | 150 | 70 | 80 | 96.67 | 132 | 52 | 52.44 |
| 103 | Gurunathan | 54 | M | | N | | | Y | BD | | | | | | | N | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 | N | 16 | 120 | 90 | 30 | 100.00 | 104 | 74 | 56.00 |
| 104 | Jones | 59 | M | | N | | | Y | BD | | | | | | | N | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 | N | 20 | 150 | 90 | 60 | 110.00 | 130 | 70 | 60.00 |
| 105 | Dhatchayini | 60 | F | | N | | | Y | OD | | | | | | | N | 20 | 150 | 80 | 70 | 103.33 | 130 | 60 | 55.56 | N | 18 | 150 | 70 | 80 | 96.67 | 132 | 52 | 52.44 |
| 106 | Dharaneeswaran | 65 | M | | N | | | Y | OD | | | | | | | N | 18 | 156 | 74 | 82 | 101.33 | 138 | 56 | 55.56 | N | 20 | 156 | 74 | 82 | 101.33 | 136 | 54 | 54.22 |
| 107 | Sumathi | 51 | F | | N | | | Y | OD | | | | | | | N | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 | N | 18 | 150 | 70 | 80 | 96.67 | 132 | 52 | 52.44 |
| 108 | Sravana Kumar | 62 | M | | Y | | | | | | | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 109 | Dharmamani | 55 | M | | N | | | Y | OD | | | | | | | GI | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 | GI | 18 | 160 | 80 | 80 | 106.67 | 142 | 62 | 59.11 |
| 110 | Menaga | 60 | F | | N | | | Y | OD | | | | | | | GII | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 | GII | 20 | 170 | 80 | 90 | 110.00 | 150 | 60 | 60.00 |
| 111 | Mukesh | 64 | M | | N | | | Y | OD | | | | | | | GII | 18 | 160 | 80 | 80 | 106.67 | 142 | 62 | 59.11 | GII | 20 | 160 | 80 | 80 | 106.67 | 140 | 60 | 57.78 |
| 112 | Mukundan | 69 | M | | N | | | Y | OD | | | | | | | N | 14 | 140 | 80 | 60 | 100.00 | 126 | 66 | 57.33 | N | 16 | 140 | 80 | 60 | 100.00 | 124 | 64 | 56.00 |
| 113 | Sundaraj | 63 | M | | N | | | Y | OD | | | | | | | N | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 | N | 12 | 120 | 90 | 30 | 100.00 | 108 | 78 | 58.67 |
| 114 | Vasanthan | 70 | M | | N | | | Y | OD | | | | | | | N | 16 | 120 | 90 | 30 | 100.00 | 104 | 74 | 56.00 | N | 18 | 120 | 80 | 40 | 93.33 | 102 | 62 | 50.22 |
| 115 | Sivanesh | 72 | M | | N | | | Y | OD | | | | | | | GI | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 | GI | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 116 | Shanmugam | 74 | M | | N | | | Y | OD | | | | | | | N | 12 | 120 | 90 | 30 | 100.00 | 108 | 78 | 58.67 | N | 14 | 120 | 80 | 40 | 93.33 | 106 | 66 | 52.89 |
| 117 | Murugan | 75 | M | | N | | | Y | OD | | | | | | | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 |
| 118 | Damodharan | 67 | M | | | | | Y | OD | | | | | | | N | 20 | 150 | 80 | 70 | 103.33 | 130 | 60 | 55.56 | N | 18 | 150 | 80 | 70 | 103.33 | 132 | 62 | 56.89 |
| 119 | Gengaiammal | 64 | F | | | | | Y | OD | | | | | | | N | 18 | 156 | 74 | 82 | 101.33 | 138 | 56 | 55.56 | N | 16 | 156 | 74 | 82 | 101.33 | 140 | 58 | 56.89 |
| 120 | Gracy | 50 | F | | | | | Y | OD | | | | | | | N | 16 | 150 | 80 | 70 | 103.33 | 134 | 64 | 58.22 | N | 18 | 150 | 80 | 70 | 103.33 | 132 | 62 | 56.89 |
| 121 | Moihdeen Bee | 49 | F | | | | | Y | OD | | | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 |
| 122 | Indrani | 55 | F | | Y | | | | | | | | | | | N | 10 | 140 | 80 | 60 | 100.00 | 130 | 70 | 60.00 | N | 10 | 140 | 80 | 60 | 100.00 | 130 | 70 | 60.00 |
| 123 | Nirmala | 52 | F | CAD | N | | | | | Y | OD | | | | | GII HTR | 24 | 160 | 94 | 66 | 116.00 | 136 | 70 | 61.33 | GII HTR,P | 22 | 160 | 94 | 66 | 116.00 | 138 | 72 | 62.67 |

| | NAME | AGE | sex | COMORBIDITY | MEAL PLAN/ORAL DRUG | Angio receptor blockers | dosage | T.ATEN | DOSAGE | T.AMLO | DOSAGE | S | DOSAGE | DIURETICTS | DOSAGE | RE FUNDUS | IOP RE | SBP | DBP | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) | LE FUNDUS | LE | SBP | | SBP- DBP | MAP=DBP+1/3{SBP-DBP} | SPP=SBP-IOP | DPP = DBP – IOP | OPP=2/3*(MAP-IOP) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 124 | Vinayagam | 59 | M | | N | | | | | | | Y | OD | | | N | 14 | 150 | 90 | 60 | 110.00 | 136 | 76 | 64.00 | N | 16 | 150 | 90 | 60 | 110.00 | 134 | 74 | 62.67 |
| 125 | Pavithra | 48 | F | CAD | N | | | | | | | Y | OD | | | N | 16 | 150 | 90 | 60 | 110.00 | 134 | 74 | 62.67 | N | 18 | 150 | 90 | 60 | 110.00 | 132 | 72 | 61.33 |
| 126 | Kamal | 50 | M | | Y | | | | | | | | | | | N | 16 | 140 | 90 | 50 | 106.67 | 124 | 74 | 60.44 | N | 14 | 120 | 90 | 30 | 100.00 | 106 | 76 | 57.33 |
| 127 | Rajan | 52 | M | | N | | | | | | | Y | OD | | | GII | 18 | 180 | 80 | 100 | 113.33 | 162 | 62 | 63.56 | GII | 20 | 180 | 80 | 100 | 113.33 | 160 | 60 | 62.22 |
| 128 | Ponnambalam | 54 | M | | N | | | | | | | | | Y | OD | GI | 16 | 150 | 94 | 56 | 112.67 | 134 | 78 | 64.44 | GI | 14 | 150 | 96 | 54 | 114.00 | 136 | 82 | 66.67 |
| 129 | Manjula | 46 | M | | N | | | | | | | | | Y | OD | N | 14 | 160 | 84 | 76 | 109.33 | 146 | 70 | 63.56 | N | 16 | 164 | 84 | 80 | 110.67 | 148 | 68 | 63.11 |
| 130 | Padmavathy | 49 | M | | Y | | | | | | | | | | | N | 16 | 144 | 94 | 50 | 110.67 | 128 | 78 | 63.11 | N | 16 | 146 | 96 | 50 | 112.67 | 130 | 80 | 64.44 |
| 131 | Kalavathi | 50 | M | | N | | | Y | OD | | | | | | | N | 16 | 164 | 84 | 80 | 110.67 | 148 | 68 | 63.11 | N | 14 | 160 | 84 | 76 | 109.33 | 146 | 70 | 63.56 |
| 132 | Pushpa | 54 | M | | Y | | | | | | | | | | | N | 14 | 142 | 88 | 54 | 106.00 | 128 | 74 | 61.33 | N | 16 | 140 | 86 | 54 | 104.00 | 124 | 70 | 58.67 |
| 133 | Gurunathan | 59 | M | | N | | | Y | OD | | | | | | | N | 18 | 154 | 94 | 60 | 114.00 | 136 | 76 | 64.00 | N | 16 | 148 | 90 | 58 | 109.33 | 132 | 74 | 62.22 |
| 134 | Kasthuri | 62 | F | | Y | | | | | | | | | | | N | 16 | 138 | 96 | 42 | 110.00 | 122 | 80 | 62.67 | N | 18 | 136 | 96 | 40 | 109.33 | 118 | 78 | 60.89 |
| 135 | Boopalan | 55 | M | | N | | | Y | OD | | | | | | | N | 18 | 166 | 86 | 80 | 112.67 | 148 | 68 | 63.11 | N | 20 | 160 | 96 | 64 | 117.33 | 140 | 76 | 64.89 |
| 136 | Rajkumaran | 54 | M | | Y | | | | | | | | | | | N | 16 | 142 | 94 | 48 | 110.00 | 126 | 78 | 62.67 | N | 16 | 140 | 96 | 44 | 110.67 | 124 | 80 | 63.11 |
| 137 | Mohanraj | 56 | M | | Y | | | | | | | | | | | N | 20 | 144 | 94 | 50 | 110.67 | 124 | 74 | 60.44 | N | 18 | 146 | 96 | 50 | 112.67 | 128 | 78 | 63.11 |
| 138 | Vasantha | 59 | F | | N | | | Y | OD | | | | | | | N | 16 | 154 | 90 | 64 | 111.33 | 138 | 74 | 63.56 | N | 16 | 152 | 96 | 56 | 114.67 | 136 | 80 | 65.78 |
| 139 | Amudha | 48 | F | | N | | | Y | OD | | | | | | | N | 16 | 158 | 84 | 74 | 108.67 | 142 | 68 | 61.78 | N | 14 | 156 | 86 | 70 | 109.33 | 142 | 72 | 63.56 |
| 140 | Chandrasekar | 47 | M | | Y | | | | | | | | | | | GI | 20 | 144 | 94 | 50 | 110.67 | 124 | 74 | 60.44 | GI | 18 | 140 | 96 | 44 | 110.67 | 122 | 78 | 61.78 |
| 141 | Mukesh | 49 | M | | Y | | | | | | | | | | | N | 14 | 142 | 88 | 54 | 106.00 | 128 | 74 | 61.33 | N | 14 | 142 | 88 | 54 | 106.00 | 128 | 74 | 61.33 |
| 142 | Vennila | 59 | F | | N | | | Y | OD | | | | | | | N | 12 | 150 | 80 | 70 | 103.33 | 138 | 68 | 60.89 | N | 14 | 150 | 80 | 70 | 103.33 | 136 | 66 | 59.56 |
| 143 | Malliga | 60 | F | | N | | | Y | OD | | | | | | | N | 20 | 160 | 90 | 70 | 113.33 | 140 | 70 | 62.22 | N | 18 | 160 | 90 | 70 | 113.33 | 142 | 72 | 63.56 |
| 144 | Gurumoorthy | 56 | M | | Y | | | | | | | | | | | N | 16 | 138 | 96 | 42 | 110.00 | 122 | 80 | 62.67 | N | 14 | 138 | 96 | 42 | 110.00 | 124 | 82 | 64.00 |
| 145 | Rajendiran | 51 | M | | N | | | Y | OD | | | | | | | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 | N | 10 | 120 | 90 | 30 | 100.00 | 110 | 80 | 60.00 |
| 146 | Arr Uman | 52 | M | | Y | | | | | | | | | | | GI | 20 | 144 | 94 | 50 | 110.67 | 124 | 74 | 60.44 | GI | 18 | 144 | 94 | 50 | 110.67 | 126 | 76 | 61.78 |
| 147 | Sengan | 66 | M | | N | | | Y | OD | | | | | | | N | 16 | 154 | 90 | 64 | 111.33 | 138 | 74 | 63.56 | N | 16 | 154 | 90 | 64 | 111.33 | 138 | 74 | 63.56 |
| 148 | Ramatha | 65 | F | | Y | | | | | | | | | | | N | 18 | 140 | 90 | 50 | 106.67 | 122 | 72 | 59.11 | N | 20 | 140 | 90 | 50 | 106.67 | 120 | 70 | 57.78 |
| 149 | Senthuralingam | 50 | M | | Y | | | | | | | | | | | N | 16 | 138 | 96 | 42 | 110.00 | 122 | 80 | 62.67 | N | 18 | 136 | 96 | 40 | 109.33 | 118 | 78 | 60.89 |
| 150 | Parveen Banu | | F | | Y | | | | | | | | | | | N | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 | GI | 18 | 120 | 90 | 30 | 100.00 | 102 | 72 | 54.67 |