TO STUDY THE CORRELATION BETWEEN THE PRESSURE-TO-CORNEA INDEX AND BOTH STRUCTURAL AND FUNCTIONAL MEASURES OF GLAUCOMA

BY

DR. AAKASH PATEL

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SBKS MEDICAL INSTITUTE & RESEARCH CENTRE
SUMANDEEP VIDYAPEETH, PIPARIA, VADODARA

IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF

M.S.

IN

OPHTHALMOLOGY

UNDER THE GUIDANCE OF

DR. R.N.KOTHARI,

PROFESSOR,

DEPARTMENT OF OPHTHALMOLOGY
SBKS MEDICAL INSTITUTE & RESEARCH CENTRE,
PIPARIA, VADODARA - 391760
YEAR 2015-2018

Sumandeep Vidyapeeth
Institutional Ethics Committee (SVIEC)

Declerad as deemed to be university u/s 3 of UGC act of 1956
At & Po Pipariya,Ta. Waghodia,
Dist. Vadodara-391760 (Gujarat) India , Phone :+02668-245262/64/66
E-Mail : rd.sumandeep@gmail.com | www.sumandeepuniversity.co.in

Dr. Aakash Patel (1st Yr Resident)
Department of Ophthalmology
SBKS MI&RC, DGH,
Sumandeep Vidyapeeth,
Piparia, Waghodia Road,
Vadodara-391760
Gujarat.

Date: 22nd Apr 2016

SUMANDEEP VIDYAPEETH
INSTITUTIONAL ETHICS COMMITTEE
OUTWARD: SVIEC/ON/MED/ BNPG15/
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SIGN.:

Ref: Your study synopsis entitled "To study correlation between the pressure-to-cornea index and structural and functional measures of glaucoma." Submitted to the SV IEC for approval.

Sub: Approval for conducting the referenced study

Dear Dr. Aakash,

The Sumandeep Vidyapeeth Institutional Ethics Committee (SV IEC) is in receipt of your above mentioned study document and as the research study classifies in the minimal risk category; as recommended by HRRP SBKS MI&RC. The SV IEC approves your study to be conducted in the presented form.

The approval remains valid for a period of 1 year. In case the study is not initiated within one year, the Ethics Committee expects to be informed about the reason for the same and a fresh approval will have to be obtained subsequently.

The Sumandeep Vidyapeeth Institutional Ethics Committee expects to be informed about the progress of the study (every 6 months), any Serious Adverse Event (SAE) occurring in the course of the study, and if any changes are made in the protocol or patient information/informed consent the SVIEC needs to be informed about this in advance and an additional permission is required to be taken. The SV IEC also requires you to submit a copy of the final study report.

Dr. Niraj Pandit
Member Secretary
SV Institutional Ethics committee

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INSTITUTIONAL ETHICS COMMITTEE
At & PO. PIPARIA, TAL. WAGHODIYA,
Dist. VADODARA-391760.

HRRP
S.B.K.S.M.I.R.C.
Outward No.: 524
Date: 23.4.2016
Sign.:

Sumandeep Vidyapeeth
Institutional Ethics Committee (SVIEC)

Declared as deemed to be university u/s 3 of UGC act of 1956
At & Po Piparia, Ta. Waghodia
Dist. Vadodara-391760(Gujarat), India, Phone: +2668-245262/64/66
E-mail: rd.sumandeep@gmail.com www.sumandeepuniversity.co.in

SUMANDEEP VIDYAPEETH
INSTITUTIONAL ETHICS COMMITTEE
OUTWARD : SVIEC/Ophthalmology/161
DATE : 25/09/17
SIGN. :
017059

Date: 25th Sep 2017

STUDY COMPLETION CERTIFICATE

This is to certify that your study entitled: "To Study Correlation between the Pressure-to-Cornea index and Structural and functional Measures of Glaucoma." Research Project was done by **"Dr. Aakash Patel"**(PG Student, Dept of Ophthalmology, S.B.K.S MI & RC, Dhiraj Hospital, Piparia, Waghodiya road, Vadodara-391760, Gujarat) and it was conducted to the satisfaction of the Sumandeep Vidyapeeth Institutional Ethics committee.

Dr. Niraj Pandit
Member Secretary
SV Institutional Ethics committee

SUMANDEEP VIDYAPEETH
INSTITUTIONAL ETHICS COMMITTEE
At. & Po. Piparia, Ta. Waghodia.
Dist. Vadodara-391760.

Patel
HRRP

HRRP
S.B.K.S.M.I.R.C.
Outward No.: 942
Date: 26/09/2017
Sign.:

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I hereby declare that the dissertation entitled **"TO STUDY THE CORRELATION BETWEEN THE PRESSURE-TO-CORNEA INDEX AND BOTH STRUCTURAL AND FUNCTIONAL MEASURES OF GLAUCOMA"** is a bonafide and genuine research work carried out by me under the guidance of DR. R.N.Kothari, Professor, Department of OPHTHALMOLOGY, SBKS Medical Institute & Research Centre, Piparia, Vadodara.

Date:
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Dr. AAKASH PATEL

SUMANDEEP VIDYAPEETH

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Date:
Place:PIPARIA

Signature of the Guide

DR. R.N.KOTHARI
Professor,
Department of Ophthalmology
SBKS MI & RC, Piparia.

SUMANDEEP VIDYAPEETH

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Seal & Signature of the HOD

DR. R. N. KOTHARI
Professor& HOD,
Department of Ophthalmology.
Date:
Place: PIPARIA

Seal & Signature of the Dean

DR. G. V. SHAH
Dean,
SBKS MI & RC
Date:
Place: PIPARIA

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Dr. AAKASH PATEL

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Dr. Aakash Patel

ABSTRACT

Introduction

In an attempt to integrate IOP and CCT into a unified risk factor, rather than simply attempting to correct for IOP measurement inaccuracy, **Iliev *et al***. have proposed a new glaucoma index, the *PRESSURE TO CORNEA INDEX (*PCI). The authors believed that PCI could better reflect the individual susceptibility to glaucomatous damage than either IOP alone or CCT by itself. We in our study tried to explore possible use of PCI as a parameter for disease severity. In an attempt to do so, we compared PCI to the structural (C/D ratio) and functional (MD and PSD) measures of glaucoma.

Methods

In this cross-sectional study, Pressure-to-cornea index was calculated for 100 eyes of 53 patients (ocular hypertension, primary open angle glaucoma, normal tension glaucoma and controls). Cup-to-disc (C/D) ratio, mean deviation (MD) and pattern standard deviation (PSD) as recorded by Humphrey automated perimetry (SITA 24-2) were correlated with PCI.

Results

The difference in the value of PCI among different groups was statistically significant. (p = 0.000) There was positive correlation between PCI and C/D (*P* = 0.000); negative correlation between PCI and MD (*P* = 0.000); and positive correlation between PCI and PSD (*P* = 0.106).

Conclusion:

We conclude that PCI can be used as a unified risk factor. Also we have found statistical significant correlation between structural and functional measures of glaucoma to Pressure to cornea index (PCI) and hence, we conclude that it can be used in glaucoma severity as well.

Keywords : Glaucoma, Central Corneal thickness, Intraocular pressure, Pressure to Cornea Index.

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INTRODUCTION

Glaucoma is the leading cause of irreversible blindness worldwide and is also the second leading cause of overall blindness as such.[1] It has been estimated that by 2020 there will be approximately 80 million people with glaucoma, an increase of about 20 million since 2010. It is assumed that at present over 8 million people are bilaterally blind due to glaucoma, which may rise to over 11 million by 2020 with the increasing prevalence, unless improved screening and effective treatment strategies are implemented. In India glaucoma is the third leading cause of blindness with 12 million people affected accounting for 12.8% of the country's blindness. Population based studies report a prevalence between 2 to 13 %.[3]

Glaucoma is chronic, progressive, multifactorial optic neuropathy caused by a group of ocular conditions which lead to damage of optic nerve with loss of visual field[4]. There are different types of glaucoma. Primary open angle glaucoma is glaucoma [POAG] which occur in the presence of open anterior chamber angles with high intraocular pressure[5].Normal tension glaucoma [NTG] is a form of glaucoma in which the intraocular pressure remains within the normal range but optic nerve is damaged[6].Ocular hypertension [OHT] is increase in pressure above 21 mm Hg but there is no optic nerve damage.[7]

Many risk factors have been identified for primary open angle glaucoma of which elevated IOP is the most significant along with family history, race, age older than 40 years, and myopia, diabetes mellitus and hypertension[5].IOP is also one of the modifiable risk factor.

The role of intraocular pressure (IOP) as a major causative risk factor in glaucoma has been confirmed in several large multicentre, randomised controlled clinical trials[8,9,10].

Intraocular pressure [IOP] refers to the pressure exerted by intraocular contents on the coats of the eyeball. Normal IOP varies between 10.5 and 20.5 mm Hg with a mean pressure of 15.5 ±2.57 mmHg. The mean amplitude of daily fluctuation is usually less than 5 mmHg in normal individual[11]. Gender, CCT, the presence of DM, and refractive error are known factors affecting IOP measurement[12].

There are different methods of measuring IOP of which some are goldmann applanation tonometry, schiotz tonometry and non contact tonometry. Goldmann applanation tonometry is considered gold standard. Gender, central corneal thickness (CCT), the presence of Diabetes mellitus (DM), and refractive error are known factors affecting IOP measurement[12].

Central corneal thickness (CCT) has influence on the measurements of intraocular pressure. Ocular hypertensive subjects have thicker cornea when compared to normal subjects[13,14]. Central corneal thickness is 0.5-0.6 mm & peripheral corneal thickness is 0.6 to 0.8 mm in normal population[4].

Measurement of central corneal thickness [CCT] is an important component for patients being evaluated for the risk of developing glaucoma. A thin central cornea may explain loss of visual field in an eye despite normal measurements of intraocular pressure [IOP] because measurements do not reflect a higher true IOP. In patients being evaluated for the risk of developing glaucoma for which CCT measurement must be considered[15].

Many studies have also pointed out the importance of central corneal thickness (CCT) as a parameter influencing the accuracy of tonometric readings as well as our decision-making in the management of glaucoma[16,17,18,19]. The influence of CCT has been demonstrated to affect IOP measurement by various tonometers, and particularly the Goldmann applanation tonometer, with thin corneas leading to an underestimation and thick corneas to an overestimation of the true IOP[20,21,22,23,24].

To correct for this variable, several conversion tables or formulae have been suggested in the literature. However, these formulae deviate considerably from one another, and there is no formula with proven superiority at the present time[25,26,27,28,29]. In addition, the relationship between applanation IOP and CCT may not be linear[22]. To find an alternative indication IOP and CCT have been integrated into single risk factor by some workers which is termed as Pressure to Cornea Index.[30]

AIM AND OBJECTIVES

AIM

To study the efficacy of pressure to cornea index as risk factor in case of open angle glaucoma.

OBJECTIVES

1) To find that in addition to serve as a single-risk factor, can Pressure to Cornea Index be used to stage glaucoma severity as well.

2) To correlate functional damage due to glaucoma with the pressure to cornea index as compared to Intra Ocular Pressure.

3) To find the possible role of pressure to cornea index (PCI) in determining he line of management.

REVIEW OF LITERATURE

Glaucoma represents a group of diseases defined by a characteristic optic neuropathy that is consistent with excavation and undermining of the neural and connective tissue elements of the optic disc and by the eventual development of distinctive patterns of visual dysfunction.

Traditionally, glaucoma has been classified as open angle or closed angle and as primary or secondary.[31]

Although elevated *intraocular pressure (IOP)* is one of the primary risk factors, its presence or absence does not have a role in the definition of the disease. In most individuals with glaucoma, the optic nerve and visual field changes seen with this disease are determined by both the level of the IOP and the resistance of the optic nerve to damage. Although progressive changes in the visual field and optic nerve are often related to elevated IOP, in some glaucoma patients the IOP remains within statistically normal range, and is called Normal Tension Glaucoma (NTG)[32]

However, in some individuals IOP remains more than arbitrary cut off levels with no disc damage, retinal nerve fiber layer damage or visual field defect and it is defined as Ocular Hypertension (OHT). [33]

As glaucoma is defined as chronic, progressive, multifactorial optic neuropathy caused by a group of ocular conditions which lead to damage of optic nerve with loss of visual field[4], IOP being the major risk factor, knowledge regarding **optic disc**, **visual field** and **intraocular pressure** is necessary in understanding of glaucoma.

GLAUCOMATOUS OPTIC NEUROPATHY

Histologically, there occurs loss of axons, blood vessels, and glial cells in glaucomatous optic neuropathy. The loss appears to start at the level of the lamina cribrosa. The loss is more evident at the superior and inferior pole of the optic disc as the superior and the inferior rim is the thickest. Structural loss may at times appear before functional loss. In advanced glaucoma central visual pathway is also affected. IOP has a major role in the glaucoma and is the most significant modifiable risk factor.[34] Two theories explains the pathophysiology of the glaucoma.

1. *Mechanical :* Compression of the axonal fibers against the lamina cribrosa plate may contribute to RGC death.[35]

2. *Vascular:* There is decrease in optic nerve head perfusion and disturbance in vascular autoregulation which may be responsible for optic nerve damage in glaucoma.[36,37]

Few points suggesting glaucomatous cupping are as follows which helps in differentiating from physiological cupping-

Generalized	Focal	Less specific
Large optic cup Asymmetry of the cups Progressive enlargement of the optic cup	Narrowing of the rim Vertical elongation of the cup Cupping of the rim margin Nerve fiber layer haemorrhage Nerve fiber layer loss	Exposed lamina cribrosa Nasal displacement of the vessels Peripapillary crescent

Enlargement of the cup is the earliest change in glaucoma which occurs due to RGC damage and NFL loss. The normal vertical C/D ratio is 0.1 to 0.4. C/D ratio is suggestive of glaucomatous change although 5% of the population without glaucoma may have C/D ratio of more than or equal to 0.6. Comparison of C/D ratio of two eyes is also important as difference of 0.2 between two non-glaucomatous eyes is seen only in 1% of the population.[39]

Enlargement of the cup results in narrowing or notching of the rim. To identify thinning of the neuroretinal rim (NRR) in the early stage of glaucoma, ISNT rule may be helpful. Generally, Inferior NRR is the thickest followed by superior, nasal and temporal. However violation of the ISNT rule is not highly specific for glaucoma.[39]

In advanced glaucoma, neural atrophy results in visualization of the laminar pores of the underlying lamina cribrosa. As the cup advances nasalization of the central retinal artery and central retinal vein is seen. 33% of the glaucomatous patient develops disc hemorrhage once during the entire course of the disease, which takes weeks to month's time to clear. They occur in nerve fiber layer and appear as linear red streak near the disc surface. Notching of the NRR and visual field loss generally follows hemorrhage. Patients diagnosed as Normal Tension Glaucoma (NTG) are more likely to develop disc hemorrhage. Optic disc hemorrhage is important in assessing progression of visual field loss and is an important prognostic sign. Other causes of disc hemorrhages include posterior vitreous detachments, diabetes mellitus, branch retinal vein occlusions, and anticoagulation therapy.[40]

Nerve fiber layer is best seen with red free illumination. It appears as fine striations created by the arrangement of the nerve fiber layer. Nerve fiber layer has a refractile appearance. As the glaucomatous optic neuropathy progresses, RNFL gets

thinner and becomes less visible. The RNFL loss may be diffuse or localized. Focal abnormalities can be slit like or wedge shaped. Diffuse loss is more common than the focal loss but is more difficult to observe. The combination of wide slit beam, red-free filter, and posterior pole lens at the slit lamp affords the best view.[41]

Peripapillary atrophy of two types occurs:

1. Alpha-zone peripapillary atrophy[42]

   Typical temporal cresent often seen in myopia with areas of hyper and hypo pigmentation and has no known impact on glaucoma.

2. Beta-zone peripapillary atrophy[43]

   It represents loss of choriocapillaris and retinal pigment epithelium resulting in characteristic white appearance of the underlying choroidal vessel and the sclera. It is seen more extensively in glaucomatous eyes.

Other, less specific signs of glaucomatous damage include

- Nasal displacement of the vessels
- Narrowing of peripapillary retinal vessels
- Baring of the circumlinear vessels.

VISUAL FIELD

Visual field assessment is very important in the evaluation and management of glaucoma. Visual field testing, termed as perimetry, can be performed by various methods, static perimetry (Humphrey, Octopus etc) being the most common. Perimetry refers to the systemic measurement of the visual field. It is used to determine the extent and the progression of the glaucoma. All the perimeters have age matched normative data of sensitivity to stimuli at all the locations of the visual field. Perimeter compares it and determines field damage.

Advances in science and technology have facilitated more sensitive and reproducible visual field loss detection, helping to detect glaucoma earlier in its course and to monitor quantitatively loss over time.

Characteristics of visual field loss in glaucoma

The visual field defect in glaucoma is in coherence with the location and distribution of the RNFL loss. Therefore in advanced glaucoma it is common to have central or temporal island of vision as nasal fibers and the maculopapular bundle are typically spared until late. Damage is always nasal to the blind spot, and is often in the form of a "step" in the nasal aspect of the visual field.[44, 45] Damage to the arcuate fibers can result in an arcuate or Bjerrum scotoma. As the arcuate fibers do not cross the horizontal midline, most glaucomatous visual field loss respects (i.e. doesn't cross) the horizontal midline. Finally, while glaucomatous visual field loss can occur anywhere in the visual field, most patients with visual field loss have some detectable field loss within the central 24-30^{o}.[46]

Testing Algorithms

Visual field tests employ either threshold or suprathreshold algorithms. In suprathreshold tests, an intensity of pre-determined brightness is employed at each test location. In threshold testing, an attempt is made to measure the intensity of the dimmest stimulus which can be detected 50% of the time.

- Swedish Interactive Threshold Algorithm (SITA) is the most commonly employed algorithm. It is developed for Humphrey perimeter. The test time is roughly half as long as full threshold test. It has similar reproducibility and is available as SITA STANDARD (SS) and SITA FAST (SF)

Summary measures of visual field performance

Mean deviation

The average of deviations across all test locations is referred to as the Mean Deviation (MD). Subjects seeing dimmer stimuli than others of similar age and race will have positive values for their MD, while subjects requiring brighter stimuli to be perceived will have negative MD values. MD values range from +2 dB to -30 dB. [47]

Pattern Standard Deviation

Pattern standard deviation (PSD) measures irregularity by summing absolute value of the difference between the threshold value for each point and the average visual field sensitivity at each point. Visual fields with the age-normal sensitivity at each point will have a PSD of 0, as will visual fields in which each point is uniformly depressed from the age-normal value. Thus, the largest PSD will be registered for focal, deep visual field defects. [47]

Glaucoma Hemifield Test (GHT)

The GHT, devised for the Humphrey Field Analyzer, compares 24-2 visual fields of 5 inferior regions representing mirror images of 5 corresponding superior regions. Differences between corresponding superior and inferior zones are compared with the differences present in the population of normal controls.[48] Possible test outcomes are:

- Outside normal limits
- Borderline
- General reduction of sensitivity
- Abnormally high sensitivity
- Within normal limits

INTRAOCULAR PRESSURE

The pressure generated by the aqueous humour on the coats of the eyeball is called Intra ocular pressure (IOP). Large, population-based epidemiologic studies have revealed a mean IOP of 15.5 mm Hg, with a standard deviation of 2.6 mm Hg. This led to the definition of "normal" IOP as 2 standard deviations above and below the mean IOP, or approximately 10–21 mm Hg. The procedure to determine IOP is called tonometry.

Factors affecting IOP [49]

- *Demographic*
    - *Age*: Mean IOP increases with increasing age
    - *Sex:* IOP is higher in women
    - *Race*: Higher IOP among blacks
    - *Heredity*: Higher IOP is inherited

- *Systemic*
    - *Diurnal variation*: Most people have a diurnal pattern of IOP
    - *Seasonal variation:* In winter months IOP is higher
    - *Blood pressure:* IOP increases with increasing blood pressure
    - *Obesity:* IOP is higher in obese people
    - *Posture:* IOP increases from sitting to inverted position
    - *Exercise:* Strenuous exercise generally lowers IOP slightly
    - *Neural:* Cholinergic and adrenergic input alters IOP
    - *Hormones:* Corticosteroids raise IOP; diabetes is also associated with raised IOP
    - *Drugs:* Multiple drugs alter IOP
- *Ocular*
    - *Refractive error*: Myopic individuals have higher IOP
    - *Eye movements:* IOP increases if eye moves against resistance
    - *Eyelid closure:* IOP increases with forcible closure
    - *Inflammation:* IOP decreases unless aqueous humour outflow affected more than inflow
    - *Surgery*: IOP generally decreases unless aqueous humour outflow affected more than inflow

TONOMETRY:

❖ Direct Method

This is the measurement of IOP directly in a living eye using a manometric technique. A needle is inserted into anterior chamber via paracentesis site and is connected to fluid filled tubing. The height of fluid in the tube corresponds to IOP. The needle can also be connected to fluid filled reservoir with a pressure sensitive membrane. Movement of membrane recorded optically or electronically is a measure of IOP. This method is not applicable clinically.

❖ **Indirect Method**

- Based on the response of the eye to an externally applied force
- IOP measurement is performed by deforming the globe and correlating the force responsible for it to the pressure in the eye.

❖ **Digital Method:**

IOP estimated by response of eye to pressure applied by finger pulp (indents easily / firm to touch). The patient must look downwards during palpation and the fingers should be placed on the eyelid superior to the tarsal plate and IOP estimated.

CLASSFICATION OF TONOMETERS:

The basic principle by which all tonometers measure IOP is by relating the deformation of the globe to the force responsible for the deformation. On the basis of the shape and magnitude of the deformation induced, tonometers are classified into two types:

❖ Indentation tonometers

❖ Applanation (flattening) tonometers.

❖ Others (DCT)

Indentation Tonometers

The shape of the deformation with this type of tonometer is a truncated cone (fig 1A). However, the precise shape is variable and unpredictable. The volume of aqueous displacement is relatively large with these tonometers and this underscores

the need for conversion tables based on empirical data from in vitro and in vivo studies to estimate the IOP. The prototype of this group is the *Schiotz Tonometer*.

Applanation Tonometers [50]

The shape of the deformation with these tonometers is a simple flattening (fig 1B), and because the shape is constant, its relationship to the IOP can usually be derived from mathematical calculations. The applanation tonometers are further differentiated on the basis of the variable that is measured. i.e. variable force and variable area tonometers.

- Variable Force

Variable force tonometers measure the force that is required to applanate (flatten) a standard area of the corneal surface. The prototype of this class is the *Goldmann applanation tonometer*. Others include:

  - Hand-held Goldmann type tonometers – eg. Perkins, Draegers
  - Mackay-Marg tonometer
  - Tonopen
  - Pneumatic tonometer.

- Variable area

These tonometers measure the area of cornea that is flattened by a known force (weight). The prototype in this group is the *Maklakov tonometer*.

Others Include:

- Applanometer
- Halberg tonometer
- Tonomat
- Glaucotest

The distinction between indentation and applanation tonometers, however, does not correlate entirely with the magnitude of intraocular volume displacement. Goldmann type tonometers have minimal displacement, whereas that with Maklakov type variable area applanation tonometer is sufficiently large so as to require conversion tables.

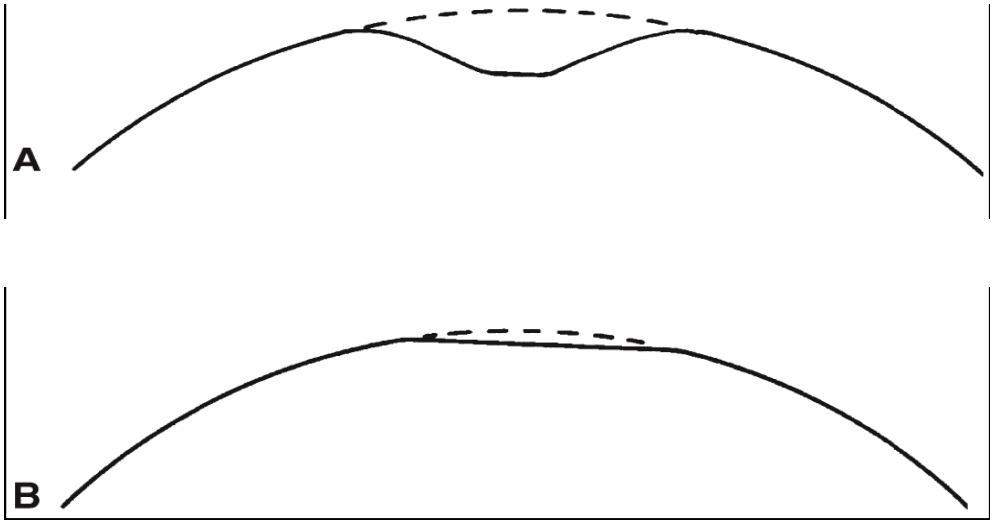


Fig. 1: Corneal deformation by (A) Indentation tonometers (a truncated cone) (B) Applanation tonometers (simple flattening)

Non-Contact Tonometer

Non-contact tonometer uses a puff of air to deform the cornea and measures either the time or force of the air puff that is required to create a standard amount of

corneal deformation. The prototype was introduced by Grolman in 1972. The Pulsair tonometer is a modern non-contact tonometer.

Pascal Dynamic Contour Tonometer

The Pascal Dynamic Tonometer (Zeimer Ophthalmic systems AG, Port, Switzerland) utilizes a piezoelectric sensor embedded in the tip of the tonometer to measure the dynamic pulsatile fluctuations in IOP. Measurements with the DCT are less affected by corneal thickness, corneal curvature and rigidity. Ocular pulse amplitude can also be measured by DCT. Disposable covers are used for each measurement and the digital display provides a Q-value which assesses the quality of the measurements. [51]

GOLDMANN APPLANATION TONOMETRY[50]

Basic Concept

Goldmann applanation tonometry is considered the international clinical standard for measuring intraocular pressure. Goldmann based his concept of tonometry on a modification of the Imbert-Fick law.[52,53]

Imbert-Fick's law *states that an external force (W) against a sphere equals the pressure inside the sphere (Pt) times the area (A) flattened (applanated) by the external force*.

$W = Pt \times A$

But this law holds good only when the sphere is:

a) Perfectly spherical

b) Perfectly flexible

c) Dry

d) Infinitely thin

The cornea fails to satisfy any of these requirements, as it is aspherical and wet, and neither perfectly flexible nor infinitely thin. The moisture on the cornea creates a surface tension (S), and the lack of flexibility requires a force to bend the cornea (B), which is independent of the internal pressure. In addition because the cornea has a central thickness of approximately 520μm, the outer area of flattening (A) is not the same as the inner area (A1). Therefore, the Imbert-Fick law was modified as below, taking these characteristics of the cornea into consideration.

$W + S = (Pt \text{ x } A1) + B$

When 'A1' = 7.35 mm^2, 'S' balances 'B' and 'W = Pt x A1'. This internal area of applanation is obtained when the diameter of the external area of corneal applanation is 3.06 mm, which is used in the standard instrument. The volume of displacement produced by applanating an area with a diameter of 3.06 mm is approximately 0.50 mm so that 'Pt' is very close to 'P0' and ocular rigidity does not significantly influence the measurement.

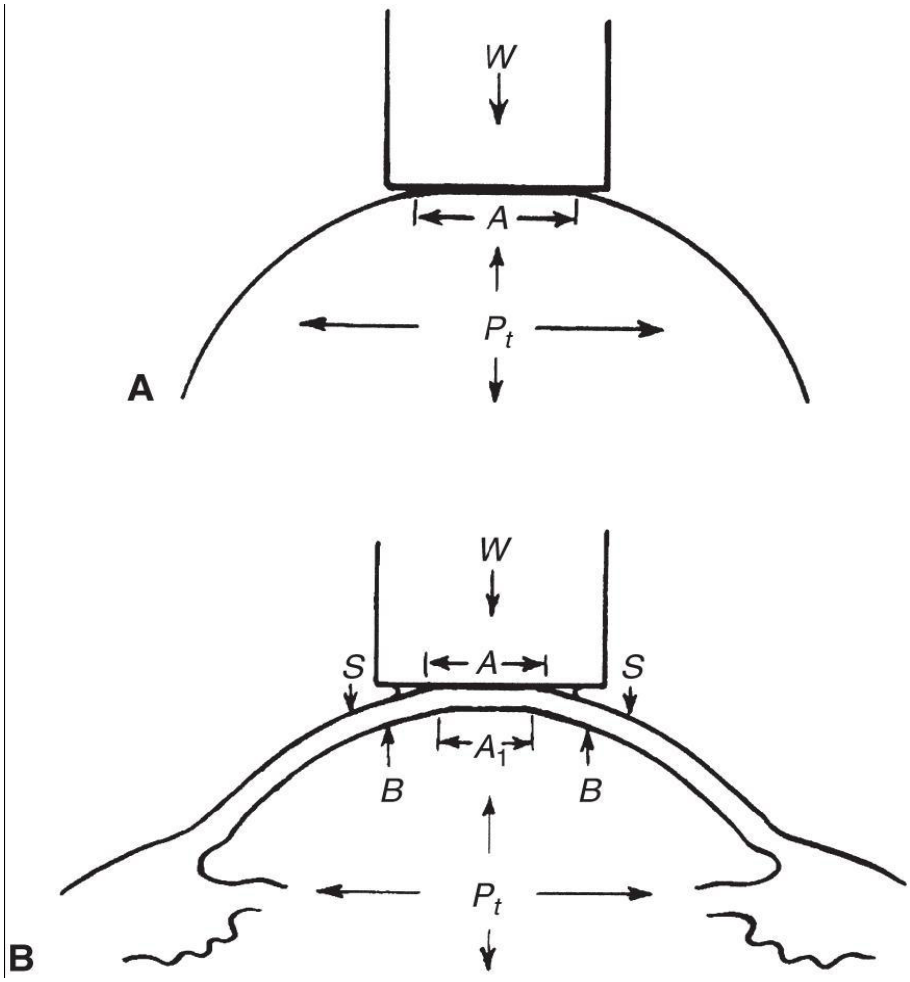


Fig. 2: Principle of Goldmann applanation tonometry

A : The Imbert–Fick law (W = Pt x A)

B : Modified Imbert-Fick law for cornea (W+S = Pt xA1+ B)

Description of the Goldmann Applanation Tonometer[50]

The instrument is mounted on a standard slit-lamp such that the examiner's view is directed through the centre of a plastic biprism, which is used to applanate the cornea. Two beam-splitting prisms within the applanating unit optically convert the circular area of corneal contact into two semicircles. The prisms are adjusted so that the inner margins of the semicircles overlap when 3.06 mm of cornea is applanated.

The biprism is attached by a metal rod to a housing, which contains a coil spring and series of levers that are used to adjust the force of the biprism against the cornea (Fig 3).

Technique[50]

Corneal anaesthesia is achieved by topical anaesthetics, and the tear film is stained with sodium fluorescein. The tonometer tip is cleaned with a sterilizing solution and the tip and prism are set in correct position on the slit lamp. The tension knob is set at 1g. The '0' graduation mark of the prism is set at the white line on the prism holder. Cobalt blue filter is used and the slit beam is maximally opened. The illumination arm is set at 60 degrees to the microscope.

The biprism is brought into gentle contact with the apex of cornea. Through a monocular view through the biprism, the clinician observes the applanation under low power. A central applanated zone and the surrounding fluorescein stained tear film is seen. The two semicircles touch when a 3.06 mm diameter circular area is applanated. Using the control stick the observer centres the assembly until two equal semicircles are seen in the centre of the field of view. The thickness of the fluorescein rings should be about one-tenth the diameter of the flattened area (0.25 to 0.35 mm). If the rings are too thin, IOP is underestimated and the patient should blink two or three times to replenish the fluorescein. Additional fluorescein may be instilled if necessary. If the fluorescein rings are too thick, the IOP is overestimated. The tension knob is rotated until the inner borders of the fluorescein rings touch each other. The reading obtained in grams is multiplied by 10 to give the IOP in millimeters of mercury. The value is recorded along with the date and time of day.

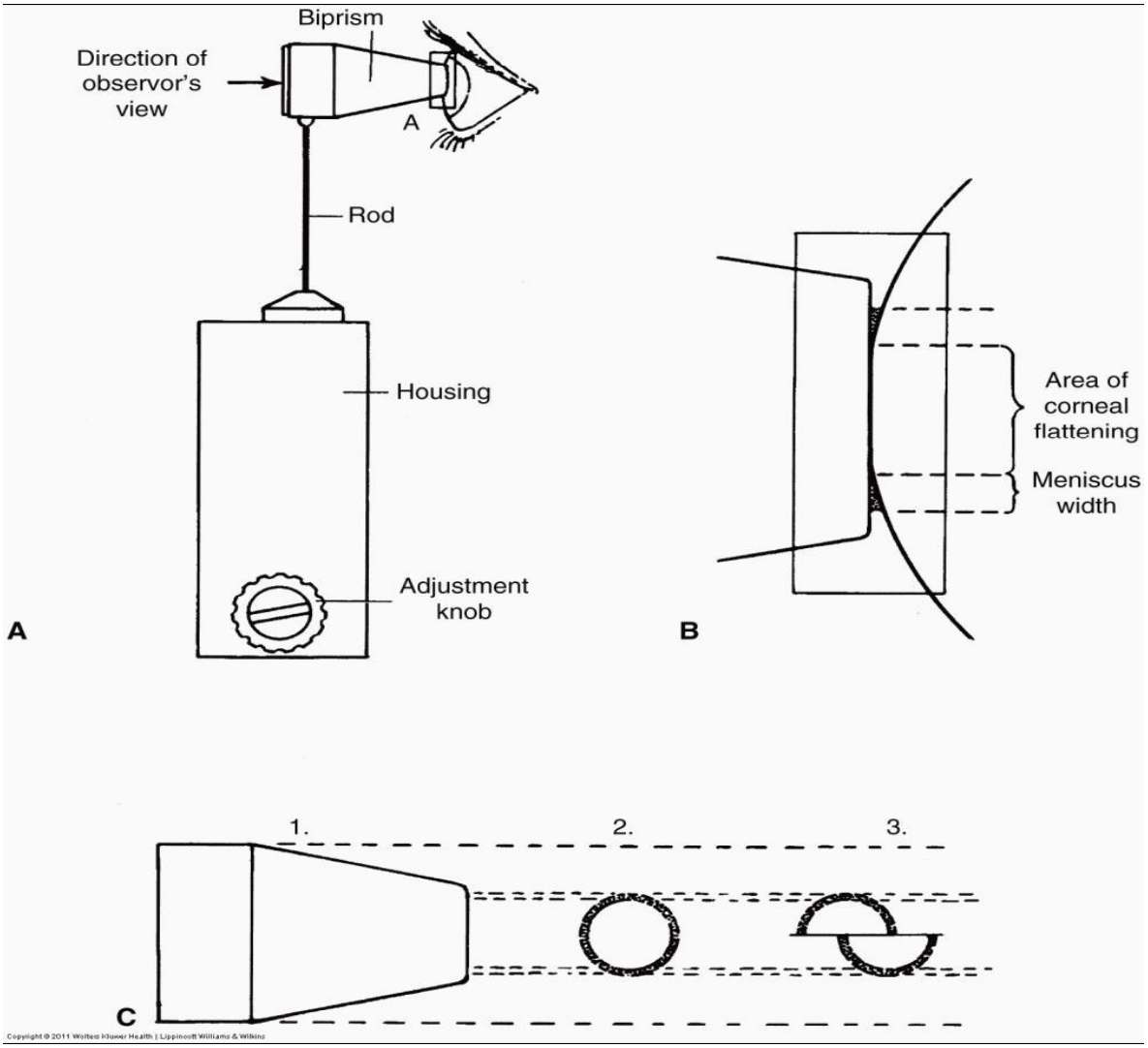


Fig 3 Goldmann-type applanation tonometer.

A. Goldmann tonometer shown in contact with patients cornea

B. Enlarged view of the tear film meniscus created by contact of biprism and cornea

C. View through the biprism: reveals circular meniscus.

Sources of Error with Goldmann Tonometry

1. The alignment and thickness of the mires affects the reading as thick mires overestimate IOP and thin mires underestimate IOP.
2. Inadequate fluorescein staining gives false low IOP reading.
3. Repeated tonometry reduces IOP, causing an underestimation of the true level. This effect is greatest between the first and second readings.
4. Widening the lid fissure excessively also causes an over estimation of IOP.
5. IOP measurement in a scarred and irregular cornea is difficult as mires get distorted.
6. The thickness of the cornea affects IOP readings. If the cornea is thick because of edema, IOP is underestimated. If the cornea is thick because of additional tissue, IOP is overestimated. [25, 53].
7. Decreased corneal thickness leads to underestimation of the IOP. This is seen following excimer laser ablation (LASIK, PRK etc).
8. Corneal astigmatism greater than 3 diopters affects IOP measurement.

Reasons for falsely high and falsely low IOP are:

➢ Falsely Low IOP	➢ Falsely High IOP
• Thin cornea	• Thick cornea
• Too little fluorescein	• Too much fluorescein
• Corneal oedema	• Steep cornea
• 1 mm Hg per 3 dioptres of with-the-rule astigmatism.	• 1 mm Hg per 3 dioptres of against-the-rule astigmatism

Goldmann applanation Tonometry is a gold standard in measurement of IOP as its results are accurate and are easily reproducible. It is easy to use. Demerits of GAT are that it needs slit lamp and is non portable. Corneal contact related complication like epithelial damage and infection may set in.

CENTRAL CORNEAL THICKNESS

Measurement of the corneal thickness is very important for the screening, diagnosis, and management of glaucoma.[54] Many studies[55, 56, 57] have shown that CCT significantly influences the measured IOP. Thin corneas may result in underestimation of the true IOP, while thick corneas may result in overestimation of IOP.[58] However, this factor alone is not sufficient to explain the increased susceptibility of those with thinner corneas to glaucoma.[58]

Technique

Topical anesthetic agent is instilled in the chosen eye. The patient is made to seat erect and is instructed to maintain a forward gaze and fixate at a target placed three meters away. With the patient in this position, measurement is taken from the centre of the cornea with the probe held perpendicular to the corneal surface.

Factors Affecting Corneal Thickness

Corneal thickness is more at the periphery than at the centre. It is affected by age, gender, osmolality of tears, epithelial and endothelial integrity, intraocular pressure, disease, drugs, axial length, refractive state, refractive surgeries and anthropometric factors.

Age: Several studies[55, 59, 60] have found significantly lower CCT with age. Hahn *et al* [59] suggested that there is a decrease in the density of keratocytes with age which could explain the reduction in CCT with age.

Gender: Several authors[61, 62, 63] compared the CCT between male and female groups and concluded that gender influences CCT values, while other studies [57, 60] found that gender had no significant effect on CCT. Shimmyo *et al* [61], Hahn *et al* [59] and Garcia-Medina *et al* [63] found thicker corneas in males than in females. Hahn *et al* [59] concluded that this difference between the CCT in men and women was statistically but not clinically significant.

Race and Ethnicity: The influence of race on CCT has been reported in multiple studies [59, 61]. Shimmyo *et al* [61] showed that African Americans and other populations of African descent have thinner CCT than other races. In the Los Angeles Latino Eye Study, the CCT of Hispanic patients had values intermediate between those found in their African-American and Caucasians populations.[59]

Intraocular pressure: When IOP exceeds the swelling pressure (SP) of the corneal stroma, epithelial oedema will occur, causing an increase in CCT. This correlates well with the occurrence of clinically detectable corneal edema when the IOP is raised above 50 mmHg. Stromal pressure is the pressure exerted by the stromal glycosaminoglycans which acts like a sponge. Normally it is around 60mm Hg. The electrostatic repulsion of the negative or anionic charge on the glycosaminoglycan molecules expands the tissue, sucking in the fluid with equal but negative pressure called imbibition pressure (IP). The values of IP are equal to the swelling pressure in vitro, but in vivo the IP is reduced by values equal to the IOP. The swelling pressure

along with the endothelial pump mechanism is critical factors in maintaining corneal thickness and transparency.

Drugs: It has been suggested that drugs like anti-glaucomatous medication (prostaglandin analogues, carbonic anhydrase inhibitors and beta blockers) may have an effect on corneal thickness. Schrems *et al* studied the effect of various topical antiglaucomatous medications, monotherapy and combined therapy.[64] They found a statistically significant decrease in central corneal thickness for eyes treated with prostaglandin monotherapy, and combined therapy with prostaglandin analogues, carbonic anhydrase inhibitors and β-blockers. The decrease in corneal thickness was found to be maximum after two years, with no or little decrease thereafter. In another study, PG analogue use was associated with significantly decreased corneal thickness and keratocyte densities, which could explain the decrease in corneal thickness.[65] Although considered safe in normal eyes[66], topical carbonic anhydrase inhibitors increase the corneal thickness in patients with low endothelial functional reserve[67]. In patients with endothelial dysfunction due to corneal guttata[68] or Fuchs corneal dystrophy or surgically induced endothelial failure or post-penetrating keratoplasty, the use of topical CAIs can permanently compromise endothelial function leading to an increase in corneal thickness.

Epithelial and Endothelial Integrity: Play a role in the maintenance of the corneal thickness by preventing the entry of water from the tears and therefore any compromise in the integrity of the epithelium or the endothelium will result in influx of fluid into the corneal stroma and increase the corneal thickness.

Corneal Disease: Like keratoconus, corneal degenerations, keratitis, etc may cause significant variation in the corneal thickness.

Axial length: Chang *et al* [69] conducted a study correlating axial length to CCT and found thinner CCT in longer eyes.

Refractive Error: Mohammed *et al* [70] found that CCT correlates with refractive error, and myopes have the thinnest CCT, followed by emmetropes and hyperopes. Price *et al* [71]suggested that thin CCT associated with myopic eyes may help explain their increased susceptibility to glaucoma.

Refractive Surgeries: Laser photoablation procedures such as LASIK and PRK reduce corneal thickness, thereby providing falsely low IOP. One study in subjects who underwent LASIK, concluded that the reduction of IOP readings after corneal refractive surgery is a linear function of the amount of refractive correction.[72]

Anthropometry: In a study correlating CCT and anthropometric factors which included height and weight, there was a positive association between CCT and height.[73] Central Corneal *Thickness*: Role in Applanation Tonometry because the mathematical calculation for Goldmann applanation tonometry is based on a presumed average central corneal thickness (520μm), variations in this parameter can lead to errors in this measurement.

Ehlers observed that corneal edema caused an underestimation of true IOP although the corneal thickness is increased, whereas variations of CCT in normal corneas can lead to falsely higher readings with thicker corneas and falsely lower with thinner corneas.[25] The clinical importance of the latter observations has subsequently been highlighted by numerous authors. Appropriate correction formula that should be used to determine the adjusted IOP when the CCT deviates from the mean has to be determined.

In their modification of the Imbert-Fick Law, Goldmann and Schmidt assigned an average central corneal thickness of 520μm. Subsequent studies have found slightly higher mean values of 537-554μm in normal subjects. As discussed earlier, multiple factors may contribute to variations in the CCT even among normal population eg. age, race, etc. Of even greater clinical importance is the observation that individuals with ocular hypertension have significantly thicker CCT whereas patients with normal tension glaucoma have thinner mean CCT.

Measurement of Corneal Thickness

Corneal Pachymetry is the technique of measuring the thickness of the cornea. Some common indications for pachymetry include glaucoma, Ocular hypertension, prior to and after refractive surgery (LASIK), ecstatic dystrophies, diabetes mellitus, dry eye and contact lens related complications. As stated earlier corneal thickness is shown to influence the accuracy of applanation tonometry.[26] The normal cornea has a central thickness of about 0.52 mm and becomes thicker in the paracentral zone (from about 0.52 mm inferiorly to 0.57 mm superiorly) and peripheral zone (from 0.63 mm inferiorly to 0.67 mm superiorly). The thinnest zone is about 1.5 mm temporal to the anatomic centre. Currently, the most common approaches to corneal thickness measurement include optical and ultrasound pachymetry.

Methods of Pachymetry

- **Ultrasound Pachymetry**

In 1980, **Kremer** introduced the first ultrasound pachymeter.[74] Ultrasound pachymetry uses high-frequency sound waves to detect the epithelial and endothelial layers, both of which are highly reflective surfaces. Knowing the velocity of sound in

corneal tissue, the distance between the two reflecting surfaces can be calculated by detecting the time lapse between the reflected sound waves from the two surfaces. It is an efficient, accurate and relatively inexpensive way to measure corneal thickness. This method requires corneal anaesthesia since the pachymeter probe comes in contact with the cornea. The applanating tip is held perpendicular to the corneal surface because tilting induces errors. The machine produces an audible beep once the measurement is recorded. Traditionally, optical pachymetry had been performed using the Haag-Streit pachymeter, whose measurements are reported to be less reproducible and less reliable than the ultrasound pachymeter.[75] Ultrasound pachymetry measurements have demonstrated high intra-observer reproducibility.[76] However results among observers vary significantly.[77]

Disadvantages

1. Requirement of physical contact with the cornea

2. Technician errors and inter-observer variability.

A new high frequency ultrasound technique (Artemis-2) is non –invasive method and can measure both epithelial and corneal thickness with precision. The velocity of sound used here is 1640m/sec.

Fig. 4: (A) PACSCAN 300p – Pachymetry instrument.

(B) Pachymetry probe

Other methods of pachymetry are

- **Ultrasound Biomicroscopy (UBM)**
- **Slit Lamp Optical Pachymetry**[78]
- **Specular Microscopy**
- **The Scanning Slit Topography System**[79]
- **Anterior Segment Optical coherence Tomography (AS-OCT)**
- **Pentacam**

THE IMPORTANCE OF CENTRAL CORNEAL THICKNESS IN GLAUCOMA

The diagnosis of glaucoma relies on a combination of factors including IOP, optic disc and nerve fiber layer damage and specific field defects. In everyday practice, IOP is the most important modifiable factor in the diagnosis, assessing the progression and response to treatment of glaucoma. Goldmann applanation tonometry is the gold standard for the measurement of IOP[78] and is based on the Imbert-Fick law. As mentioned earlier when an area of 7.35mm^2 was applanated, the surface tension due to the tear film counter-balanced the resistance to indentation of the cornea, thus nullifying the effects of rigidity of the globe and the surface tension of the tear film in applanation tonometry[80]. More recent evidence indicates that these, as well as a number of other factors (e.g. corneal curvature, significant astigmatism) do affect the accuracy of applanation tonometry.

The resistance offered by the cornea to the indentation, changes with variations in the CCT, so that it is not longer balanced exactly by the tear film surface tension. This may affect the accuracy of the IOP measurement. A thinner cornea will require less force to applanate it, leading to underestimation of true IOP. On the contrary, a thicker cornea will require more force thus giving a falsely high IOP reading. So the clinician should be alert when faced with patient with thin cornea and high-normal IOP and to rule out glaucoma. A positive correlation between increased corneal thickness and IOP has been reported earlier[25, 53, 81]. Studies done in eyes with manometrically controlled IOP have reported a significant disparity between the true IOP and simultaneous applanation tonometry readings. This was attributed to the variation in the CCT. It was observed that the underestimation of IOP in thin corneas

was as much as 4.9 mmHg, while an overestimation of 6.8 mm Hg was produced with thick corneas.[82, 83, 84] In view of these findings, it was suggested that measurement of corneal thickness is mandatory for the accurate interpretation of applanation tonometry.

Formulae for IOP Correction:

Several formulae have been proposed for the adjustment of the applanation IOP for deviation from normal mean CCT. There is a lack of agreement regarding the correction factor that should be used for adjusting the IOP, measured by Goldmann tonometry, when the CCT deviates from the normal. While most authors argue about which correction formula is best, there are some who believe that the use of correction formulae for GAT IOP are unsuitable to clinically approximate to true IOP values.[85] Another study showed that individual risk for developing POAG in ocular hypertensive individuals is simpler and equally accurate using IOP and CCT as measured, rather than applying an adjustment formula to correct IOP for CCT.[86]

- ***Ehlers*** proposed a correction factor for IOP, to adjust for CCT measurements that differ from his assumed normal CCT of 520µm.[25]

Corrected IOP = IOPGAT – [5mmHg × (Measured CCT - Mean normal CCT)/ 70µm] = IOPGAT – [5mmHg × (measured CCT – 520)/70 µm]

Accordingly it had been calculated that applanation tonometry over/underestimated IOP by 5mm Hg for every 70µm corneal thickness, i.e. 1mmHg change of IOP for every 14µm change in CCT, which has been supported by others. Other studies, however, have revealed smaller errors of 0.2mm Hg per 10µm, which is consistent with a direct cannulation study.

- According to ***Whitacre's formula***[26], a 10μm change in CCT from normal 520μm, resulted in only 0.2mm Hg change in the applanation IOP.

Corrected IOP = IOPGAT - [2mmHg × (measured CCT – Mean normal CCT)/100 μm] = IOPGAT - [2mmHg × (measured CCT – 520)/100 μm]

Other Formulae:

- ***Doughty's formula*** [27]*:* considered normal CCT of 535μm

Corrected IOP = IOPGAT – [2.5mmHg × (measured CCT – Mean normal CCT/50μm)] = IOPGAT – [2.5mmHg × (measured CCT – 525/50μm)]

- ***Kolhaas's formula*** [29]*: Corrected IOP = IOPGAT + (23.28 – 0.0423 × CCT)* The role of CCT in glaucoma is still confounding and is under scrutiny. CCT is important in glaucoma by two means:

  1) By altering the accuracy of applanation tonometry readings and
  2) As a predictive factor in the development of POAG as shown by the OHT

PRESSURE TO CORNEA INDEX

In an attempt to integrate IOP and CCT into a unified risk factor, rather than simply attempting to correct for IOP measurement inaccuracy, **Iliev *et al***.[30] have proposed a new glaucoma index, the *PRESSURE TO CORNEA INDEX (*PCI). The authors believed that PCI could better reflect the individual susceptibility to glaucomatous damage than either IOP alone or CCT by itself.

The basic pressure-to-cornea index was defined as IOP (Highest recorded) in mm Hg, divided by the CCT in mm [PCI_{basic} = IOP/CCT in mm]. In an attempt to reduce the relative role of IOP and accentuate the relative role of CCT in the formula,

they included "amplified" index versions: IOP/CCT^2, IOP/CCT^3 and IOP/CCT^4. Since IOP/CCT^3 differentiated between the groups best, it was suggested as pressure to cornea index ($PCI = IOP/CCT^3$)

PCI (IOP/CCT^3) better differentiated glaucoma from non-glaucoma than each of the individual parameters alone and when compared with the corrected IOP according to three published correction formulae. PCI may therefore may advantageous as it does not replace the measured IOP, but rather adds a new component. It is also easier to measure.

A PCI range of 120–140 was proposed as normal. 120 being the cut-off value for eyes with untreated pressures $\leqslant$21 mm Hg, 140 when untreated pressure $\geqslant$22 mm Hg. They proposed that PCI may reflect individual susceptibility to a given IOP level, and thus represent a glaucoma risk factor.

Franco et al. [87] performed a study to correlate (C/D ratio) and functional measures (Mean Deviation and Pattern Standard Deviation) of glaucoma and concluded that PCI can be used to assess glaucoma severity as well. Their results revealed linear correlation between **Cup to Disc ratio** and PCI. Patients with higher C/D ratios presented with higher PCI values. The C/D ratio is a subjective, qualitative method to assess the optic nerve head in glaucoma patients.

Since C/D ratio is dependent on the size of the disc, it is not a precise indicator of glaucomatous optic disc damage unless relative disc size, area, quantitative assessment of neural rim width and area is done They found correlation between the PCI and the **Mean Deviation** (MD) value of automated perimetry. Patients with lower

MD presented with higher PCI values. The PCI showed a statistically significant negative correlation with MD.

The PSD value is the standard deviation of the difference between the threshold value at each test location and expected value and as an indicator of localized defects it reflects the roughness of the visual field. Higher PSD indicates more damaged visual field. Hence a positive correlation is expected between PSD and PCI. However, the correlation between PCI and PSD revealed a trend toward a negative correlation in their study.

MATERIALS AND METHOD

- Study design – Cross-sectional and comparative study
- Sample size - 100 eyes of 53 patients
- Study period - From April 2016 to August 2017,

Inclusion Criteria

- Open angle on Gonioscopy
- Newly diagnosed cases of POAG, OHT and NTG.
- Patient willing to participate.

Exclusion Criteria

- Secondary glaucoma
- Closed angle on Gonioscopy
- History of ocular surgery (Glaucoma surgery, Lasik, Cataract)
- Extended Contact lens use
- Patient with infective corneal pathology
- Chronic ocular surface diseases
- Pregnant and lactating mothers Secondary glaucoma
- Closed angle on Gonioscopy
- History of ocular surgery (Glaucoma surgery, Lasik, Cataract)
- Extended Contact lens use
- Patient with infective corneal pathology

Method

Patient coming to the OPD of Ophthalmology department, Dhiraj Hospital, SBKS MI&RC were enrolled in the study after taking informed and written consent. A detailed careful history and complete ophthalmic examination was done. Uncorrected visual acuity (UCVA) and Best corrected visual acuity (BCVA) was checked using Snellen's chart. Anterior segment evaluation was done by slit lamp. IOP was

determined with Goldmann applanation tonometer (AATM-5001). There consecutive readings were taken and the mean of those were considered. Angle of anterior chamber was assessed by performing gonioscopy with Zeiss four mirror gonio lens. Dilated fundus examination was done with slit lamp biomicroscopy (using 90D and 78D Zeiss lens). Visual field was done using Humphrey's automated static Perimetry using SITA standard algorithm and 24-2 program. Perimetry results with reliable indices only were taken into consideration**.** CCT was measured by regularly calibrated ultrasonic pachymeter i.e. Ultrasound system SONOMED PACSCAN 300P. The procedure was repeated thrice by a single observer. The average of the three readings was taken. The subjects were divided into four groups i.e., Group 1 – ocular hypertension, Group 2 - primary open angle glaucoma, Group 3 – normal tension glaucoma and Group 4 – controls (Normal). All patients were aged between 37 and 71 years. A total of 25 ocular hypertensives eyes, 25 primary open angle glaucoma eyes, 25 eyes of normal tension glaucoma and 25 control eyes were enrolled in the study.

Group 1: Ocular hypertensive subjects

- IOP > 21 mm Hg with GAT
- Healthy optic discs with no glaucomatous features
- No visual field defects and

Group 2: Primary open angle glaucoma subjects

- IOP > 21mm Hg with GAT
- Glaucomatous disc changes with or without nerve fiber layer defects
- Glaucomatous visual field defects.

Group 3: Normal Tension Glaucoma

- IOP < 21mm Hg with GAT
- Glaucomatous disc changes with or without nerve fiber layer defects
- Glaucomatous visual field defects.

Group 4: Normal subjects (Control group)

- IOP < 21 mm Hg with GAT
- Normal optic disc
- No family history of glaucoma, no suspicion of any form of glaucoma, or any other eye disease.

Fig 5: Visual Field Testing by automated Perimetry ZEISS HFA

Fig 6 : IOP checking by GAT

Fig 7 : Measurement of Central Corneal Thickness using US pachymetry

Statistical Analysis

The statistical analysis for this study was performed using IBM SPSS version 20 for windows. Categorical variables were analysed with frequencies and percentages. For continuous variables, mean and standard deviation were calculated and the Student's unpaired 't' test was used to for comparison between two groups whereas repeated measures ANOVA was applied for comparison between more than two groups. When ANOVA was applied, Bonferroni's Post Hoc multiple comparison has been done to know the one-to-one relation. To know the relation between two variables, Pearson's correlation coefficient was applied. 'p' value < 0.05 was considered to be statistically significant.

OBSERVATIONS AND RESULTS

Table 1a: Sex distribution of subjects in all group

Group	Number of eyes : Male	Number of eyes: Male (in %)	Number of eyes : Female	Number of eyes: Female (in %)	Total
Normal	10	40 %	15	60 %	25
OHT	19	76 %	6	24 %	25
POAG	13	52 %	12	48 %	25
NTG	23	92 %	2	8 %	25
Total	**65**	**65 %**	**35**	**35 %**	**100**

Table 1a shows distribution of males and females in different groups. There were 65 males (65%) and 35 females (35%). In normal group there were 10 (40%) males and 15 (60%) females. In OHT group there were 19 (76%) males and 6 (24%) females. In POAG group there were 13 (52%) males and 12 (48%) females. In NTG group there were 23 (92%) males and 2 (8%) females.

Graph 1a: Sex distribution of subjects in all group

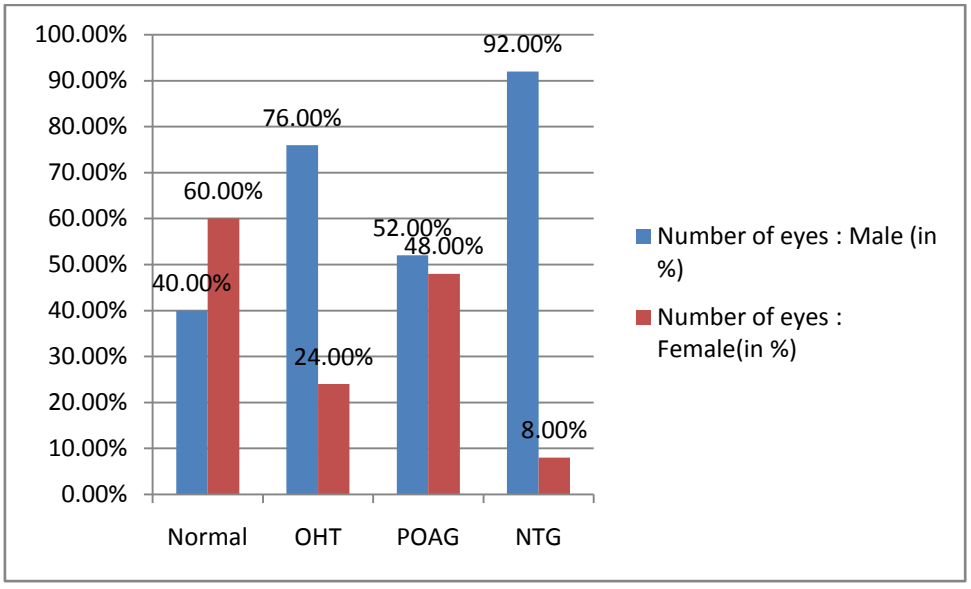
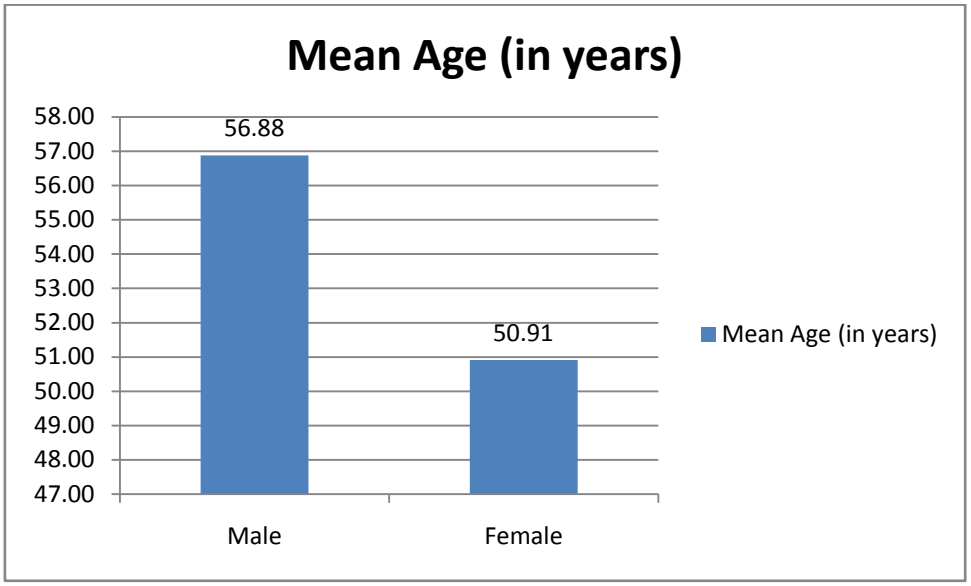


Table 1b: Mean age of male and female

Age		
	Mean Age (in years)	SD
Male	56.88	8.07486
Female	50.91	11.16816

The mean age of 65 males included in the study was 56.88 ± 8.07 and that of 35 females was 50.91 ± 11.16.

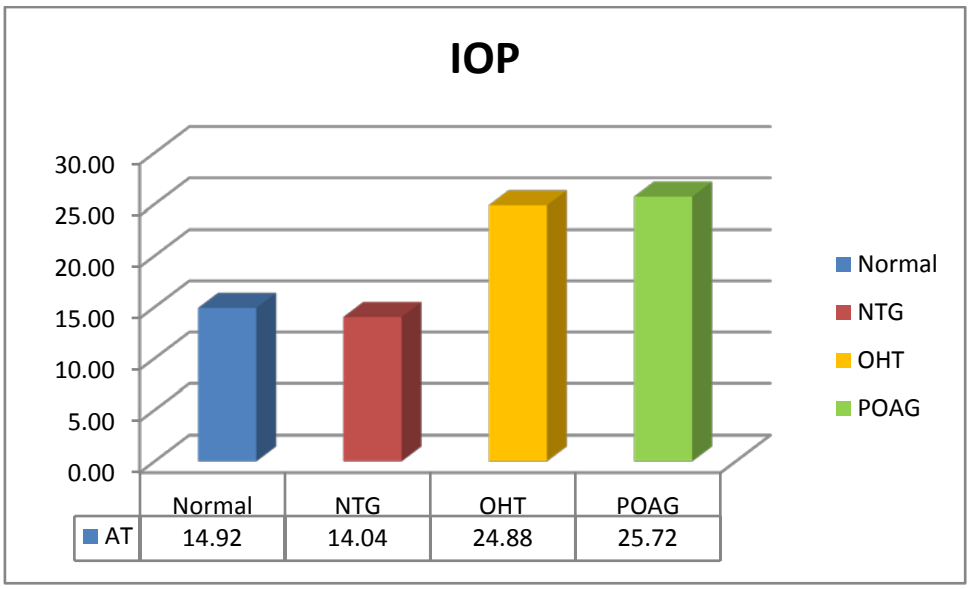


Graph 1b: Mean age of male and female

Table 2a: Mean IOP with SD by AT of subjects in all group

IOP				p value
	N	Mean	SD	
Normal	25	14.92	2.414	**0.000**
NTG	25	14.04	1.513	
OHT	25	24.88	1.943	
POAG	25	25.72	3.530	
Total	**100**	**19.89**	**5.971**	

The value IOP measured by GAT is shown in table 2a. On comparison of IOP values of four groups, the difference is statistically significant. The mean IOP measured by GAT in normal group was 14.92 ± 2.41, NTG group was 14.04 ± 1.51, OHT group was 24.88 ± 1.94 and POAG group was 25.72 ± 3.53. The values of IOP in Controls and NTG group is within normal limits i.e. <21 mm hg and that of OHT and POAG groups is >21 mm hg.



Graph 2: Mean IOP with SD by AT of subjects in all group

On multiple comparisons done by ANNOVA there is no statistical difference between IOP values of NTG group and controls and POAG and OHT. But when NTG and controls are matched to POAG and OHT group, the difference is statistically significant.

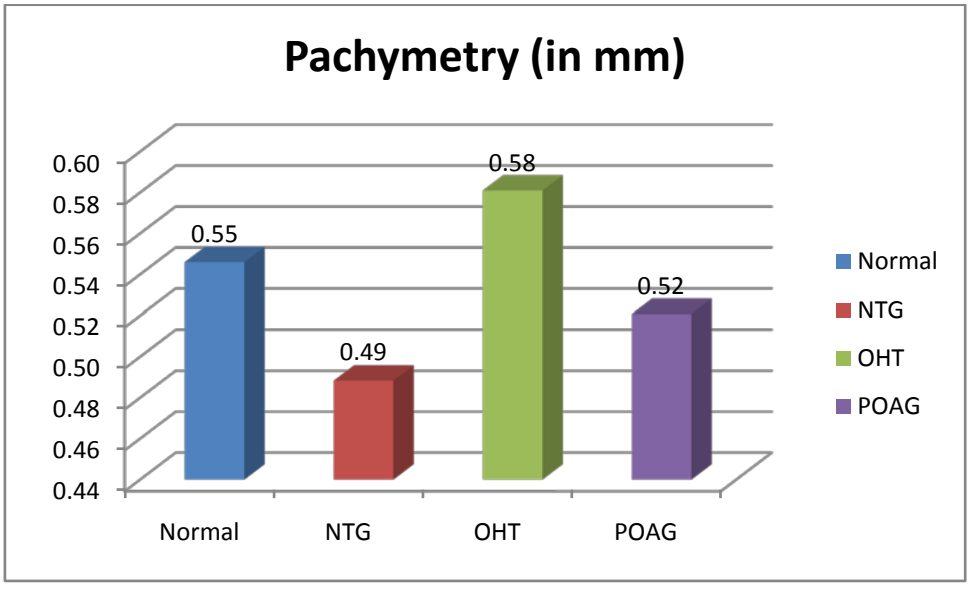
Table 2b: Mean IOP with SD by AT of subjects in all group with multiple comparisons

Multiple Comparisons					
(I) Code		Std. Error	p value	95% Confidence Interval	
				Lower Bound	Upper Bound
Normal	NTG	0.698	1.000	-1.00	2.76
	OHT	0.698	0.000	-11.84	-8.08
	POAG	0.698	0.000	-12.68	-8.92
NTG	Normal	0.698	1.000	-2.76	1.00
	OHT	0.698	0.000	-12.72	-8.96
	POAG	0.698	0.000	-13.56	-9.80
OHT	Normal	0.698	0.000	8.08	11.84
	NTG	0.698	0.000	8.96	12.72
	POAG	0.698	1.000	-2.72	1.04
POAG	Normal	0.698	0.000	8.92	12.68
	NTG	0.698	0.000	9.80	13.56
	OHT	0.698	1.000	-1.04	2.72

Table 3a: Mean pachymetry with SD of subjects in all groups

Descriptives – CCT (in mm)							p value
		N	Mean (mm)	SD	95% Confidence Interval for Mean		
					Lower Bound	Upper Bound	
Pachymetry	Normal	25	0.55	0.03	0.535	0.557	**0.000**
	NTG	25	0.49	0.02	0.478	0.499	
	OHT	25	0.58	0.02	0.574	0.588	
	POAG	25	0.52	0.03	0.508	0.534	
	Total	100	0.53	0.04	0.526	0.542	

CCT is shown in table 3a. On comparison of CCT values of four groups, the difference is statistically significant. The mean CCT in normal group was 0.55 ± 0.03, NTG group was 0.49 ± 0.02, OHT group was 0.58 ± 0.02 and POAG group was 0.52 ± 0.04.



Graph 3: Mean pachymetry with SD of subjects in all groups

On multiple comparisons done by ANNOVA there is statistical difference between IOP values of all groups when compared separately. Though the significance of difference in CCT values of POAG group and Normal group is less than difference of significance of other groups are compared, still it is statistically significant. (p value 0.005)

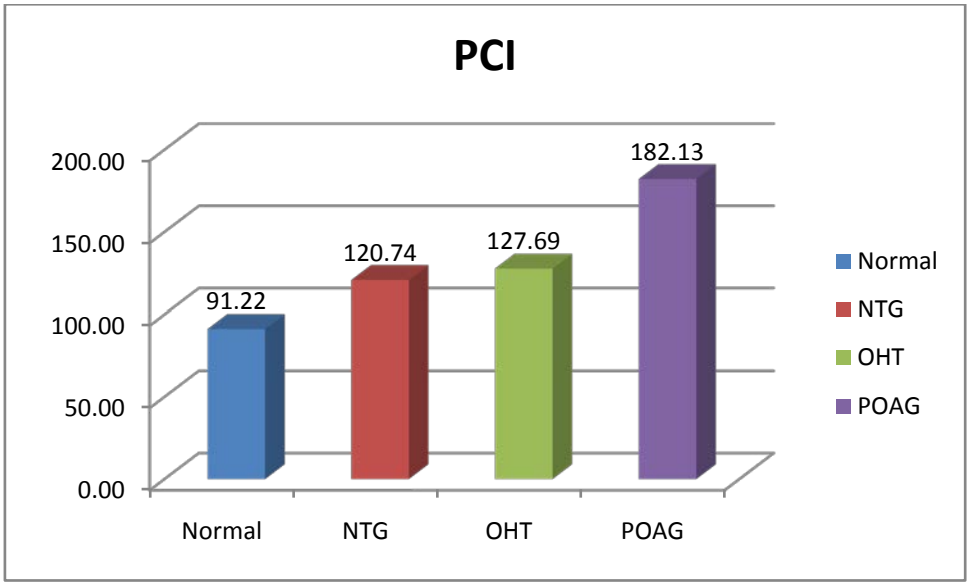
Table 3b: Mean pachymetry with SD of subjects in all groups with multiple comparisons

Dependent Variable			p value	95% Confidence Interval	
				Lower Bound	Upper Bound
Pachymetry	Normal	NTG	0.000	0.04	0.08
		OHT	0.000	-0.05	-0.02
		POAG	0.005	0.01	0.04
	NTG	Normal	0.000	-0.08	-0.04
		OHT	0.000	-0.11	-0.07
		POAG	0.000	-0.05	-0.01
	OHT	Normal	0.000	0.02	0.05
		NTG	0.000	0.07	0.11
		POAG	0.000	0.04	0.08
	POAG	Normal	0.005	-0.04	-0.01
		NTG	0.000	0.01	0.05
		OHT	0.000	-0.08	-0.04

Table 4a: Mean PCI value with SD of subjects in all groups

Descriptives							p value
		N	Mean	SD	95% Confidence Interval for Mean		
					Lower Bound	Upper Bound	
PCI	Normal	25	91.22	8.46	87.725	94.705	**0.000**
	NTG	25	120.74	10.18	116.534	124.940	
	OHT	25	127.69	16.85	120.737	134.648	
	POAG	25	182.13	16.71	175.228	189.025	
	Total	100	130.44	35.61	123.378	137.508	

PCI is shown in table 4a. On comparison of PCI values of four groups, the difference is statistically significant. The mean PCI in normal group was 91.22 ± 8.46, NTG group was 120.74 ± 10.18, OHT group was 127.69 ± 16.85 and POAG group was 182.13 ± 35.61.



Graph 4: Mean PCI value with SD of subjects in all groups

On multiple comparisons done by ANNOVA there is no statistical difference between PCI values of NTG group and OHT group. Other comparisons are statistically significant.

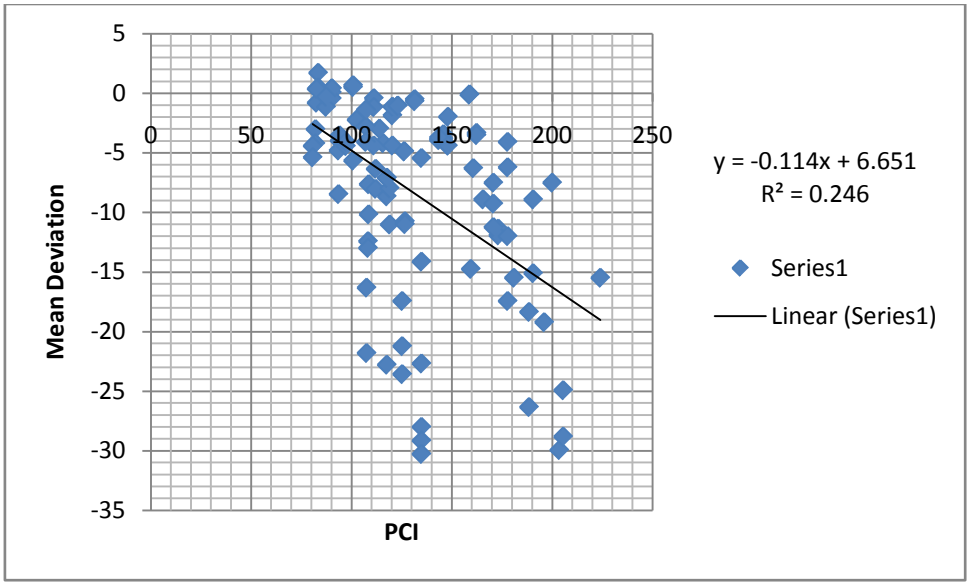
Table 4b: Mean value of PCI with SD of subjects in all groups with multiple comparisons

Multiple Comparisons					
Dependent Variable			p value	95% Confidence Interval	
				Lower Bound	Upper Bound
PCI	Normal	NTG	0.000	-39.87	-19.17
		OHT	0.000	-46.83	-26.12
		POAG	0.000	-101.26	-80.56
	NTG	Normal	0.000	19.17	39.87
		OHT	0.441	-17.31	3.40
		POAG	0.000	-71.74	-51.04
	OHT	Normal	0.000	26.12	46.83
		NTG	0.441	-3.40	17.31
		POAG	0.000	-64.79	-44.08
	POAG	Normal	0.000	80.56	101.26
		NTG	0.000	51.04	71.74
		OHT	0.000	44.08	64.79

Table 5: Correlation between the pressure-to-cornea index (PCI) and mean deviation (MD)

Coefficient					
	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
(Constant)	32.720	58.108		0.563	0.575
Mean Deviation	-2.196	0.524	-0.507	-4.189	0.000

The mean value of MD was -8.37 ± 8.45. The PCI showed a statistically significant negative correlation with MD (*P* = 0.000)

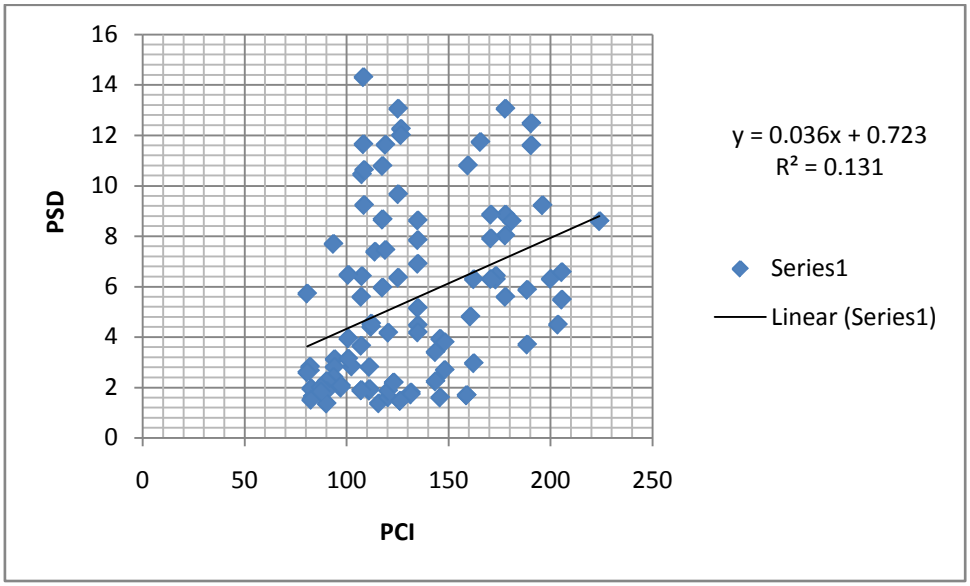


Graph 5: Correlation between the pressure-to-cornea index (PCI) and mean deviation (MD)

Table 6: Correlation between the pressure-to-cornea index (PCI) and pattern standard deviation (PSD)

Coefficient					
	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
(Constant)	32.720	58.108		0.563	0.575
Pattern Standard Deviation	1.758	1.077	0.175	1.632	0.106

The mean value of PSD was 5.41 ± 3.41. The PCI showed positive correlation with PSD but the correlation is statistically not significant (*P* = 0.106)

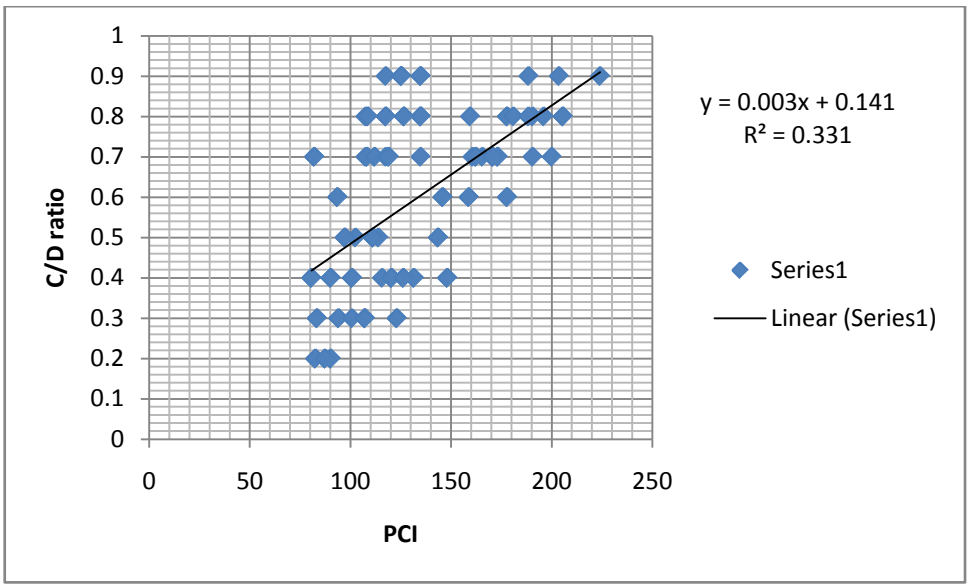


Graph 6: Correlation between the pressure-to-cornea index (PCI) and pattern standard deviation (PSD)

Table 7: Correlation between the pressure-to-cornea index (PCI) and Cup to disc ratio (C/D ratio)

Coefficients[a]						
Model		Unstandardized Coefficients		Standardized Coefficients	t	p value
		B	SE	Beta		
1	(Constant)	107.442	4.603		23.342	.000
	Disc (CDR)	39.657	6.044	.552	6.561	.000

The median C/D ratio was 0.7 (Range = 0.2 to 0.9). The PCI showed positive correlation with C/D ratio and the correlation is statistically significant (*P* = 0.000)



Graph 7: Correlation between the pressure-to-cornea index (PCI) and Cup to disc ratio (C/D ratio)

DISCUSSION

Several studies have pointed out the importance of central corneal thickness (CCT) as a parameter influencing the accuracy of tonometric readings as well as our decision-making in the management of glaucoma[16,17,18,19]. The influence of CCT has been demonstrated to affect IOP measurement by various tonometers, and particularly the Goldmann applanation tonometer, with thin corneas leading to an underestimation and thick corneas to an overestimation of the true IOP[20,21,22,23,24].

Central Corneal Thickness

CCT values have shown difference in individuals with NTG, POAG and OHT. Anupama C. Shetgar[88] et al. performed a study to compare the Central Corneal Thickness (CCT) of Normal Tension Glaucoma (NTG) with those of Primary Open Angle Glaucoma (POAG) and Ocular Hypertension (OHT). They concluded that the central corneal thickness was significantly lower in the normal tension glaucoma patients as compared to those in the controls and in the primary open angle glaucoma patients, whereas the ocular hypertension patients had significantly higher central corneal thicknesses than the controls and the primary open angle glaucoma patients. No significant difference was found between the primary open angle patients and the controls. Copt RP[89] et al. performed a similar study and found that here was no significant difference in CCT between controls and patients with POAG, but the CCT in the group with NTG was significantly lower than that in the control group or the group with POAG ($P < .001$), and the CCT in the group with OHT was significantly higher than in controls or patients with POAG ($P < .001$).

In our study we found similar significant difference between the CCT among individuals with NTG, OHT and POAG. CCT in NTG group (0.49 mm ± 0.02) was significantly lower than POAG (0.52 mm ± 0.03), OHT (0.58 mm ± 0.02) and normal individuals (0.55 mm ± 0.03). Also the CCT in OHT group was significantly higher than POAG, NTG and normal individuals. (P value 0.0000). However unlike above studies we found significant difference between CCT in POAG and normal group. The CCT in POAG group was significantly lower than that in normal group. (p value 0.005). We were unable to find the reason for this disparity. Our results are similar to a study by Aghaian E[60] et al. who found that Glaucoma suspects and patients with normal tension glaucoma (NTG), primary open-angle glaucoma (POAG), pseudoexfoliation glaucoma (PEX), and chronic angle-closure glaucoma (CACG) had corneas significantly thinner than those of normal participants ($P < \text{or} = 0.004$), whereas ocular hypertensives had significantly thicker corneas ($P < 0.0001$) in Asian (Chinese, Japanese, and Filipino), Caucasian, Hispanic, and African American patients.

Several studies have also shown the importance of central corneal thickness (CCT) as a parameter influencing the accuracy of tonometric readings. Various tonometers, and particularly the Goldmann applanation tonometer have shown underestimation of IOP in thin corneas and vice versa.

Ko YC[22] et al. in an attempt to study varying effects of corneal thickness on intraocular pressure measurements with different tonometers found that pressure readings with the GAT, NCT, and Ocular Blood Flow Tonometer are all affected by CCT, with the NCT being the one most affected and the GAT the least. Their findings suggest that CCT is an essential variable to consider in interpreting IOP readings,

Several conversion tables or formulae have been suggested to correct for this variable but none has shown superiority over the other. Moreover it may not be appropriate to simply correct for the measurement inaccuracy as importance of CCT in glaucomatous process is not fully addressed. Also the relation of CCT and IOP may not be linear. Furthermore there are reasons to believe that corneal thickness may represent an independent risk factor for the development and progression of glaucoma.

In OHTS study, Central corneal thickness was found to be a powerful predictor for the development of POAG.[90] Herndon LW[91] et al. found that lower CCT was significantly associated with worsened Advanced Glaucoma Intervention Study score, worsened mean deviation of visual field, and increased vertical and horizontal cup-disc ratios. Kim JW[92] et al. in a study to investigate the association between corneal pachymetry and visual field progression in patients with chronic open-angle glaucoma found that visual field progression in patients with open-angle glaucoma was significantly associated with thinner CCT.

Hence in order to integrate IOP and CCT into unified risk factor Ilive[30] et al. proposed a new index called Pressure to Cornea Index. PCI (IOP/CCT^3) was defined as the ratio between untreated IOP and CCT^3 in mm. PCI distribution in 220 normal controls, 53 patients with normal tension glaucoma (NTG), 76 with ocular hypertension (OHT), and 89 with primary open-angle glaucoma (POAG) was investigated. Mean PCI values were: Controls 92.0 (SD 24.8), NTG 129.1 (SD 25.8), OHT 134.0 (SD 26.5), and POAG 173.6 (SD 40.9).

They concluded that a PCI range of 120–140 as the upper limit of "normality", 120 being the cut-off value for eyes with untreated pressures $\leqslant$21 mm Hg, 140 when

untreated pressure ⩾22 mm Hg. They also proposed that PCI may reflect individual susceptibility to a given IOP level, and thus represent a glaucoma risk factor.

Our study also showed similar results. Mean PCI values in our study were: Controls 91.22 (SD 8.46), NTG 120.74 (SD 10.18), OHT 127.69 (SD 16.85), and POAG 182.13 (SD 16.71). The difference among the group was statistically significant except that the difference between NTG group and OHT group was not significant. (p value 0.441) This result is also in accordance with the parent study. Such significance was least desired between OHT and NTG group as clinically, after the untreated pressure has been established, differential diagnosis is usually made between NTG and normality, and between POAG and OHT.

André Omgbwa Eballe[93] et al. in a study aiming to determine the profile of central corneal thickness (CCT) in the Cameroonian non glaucomatous black population and its relationship with intraocular pressure (IOP) studied four hundred and eighty-five patients (970 eyes) found that Pressure-to-cornea index (PCI) in the right eye was 88.50 ± 23.06 and 89.78 ± 23.31 in the left eye ($P > 0.05$); in both eyes (right and left combined) PCI was 89.14 ± 23.19. Since the study was performed on a large scale, though taking into consideration only non glaucomatous individuals i.e. normals, PCI values for this population was close to the cutoff proposed by Iliev *et al*.

Moving a step ahead we in our study tried to explore possible use of PCI as a parameter for disease severity. In an attempt to do so, we compared PCI to the structural (C/D ratio) and functional (MD and PSD) measures of glaucoma.

Franco et al.[87] in their study correlated PCI with C/D ratio, MD and PSD. 72 eyes of 36 patients were included. All the patients included had raised IOP (Either POAG or OHT). We have included controls as well as NTG group in our study. We

evaluated for the possible use of PCI for both, as a glaucoma risk factor as well as a parameter for disease severity. Moreover we believe that by considering all four groups, the probability of PCI being used as a parameter for disease severity is enhanced.

Our results revealed good linear correlation between the PCI and the C/D ratio. Patients with higher C/D ratios presented with higher PCI values. The C/D ratio is a subjective, qualitative method to assess the optic nerve head in glaucoma patients. It is widely used in clinical practice, and it gives an appraisal of the cup diameter in relation to the optic disc size; on a decimal scale, it ranges from zero (no cupping) to one (optic nerve head completely excavated). Franco et al.[87] also observed significant correlation between C/D ratio and PCI.

Patients with lower MD presented with higher PCI values. The MD value of automated perimetry is a weighted average decibel deviation from age normal database; the lower the MD value, the more damaged the visual function is. Nevertheless, the MD can be affected by media opacity such as cataract and uncorrected refractive error. We found statistically significant correlation when MD and PCI were compared. Similar result was also seen in study done by Franco et al.[87]

The PSD value is the standard deviation of the difference between the threshold value at each test location and expected value and as an indicator of localized defects it reflects the roughness of the visual field. It is calculated by summing the absolute value of the difference between the threshold value for each point and the average visual field sensitivity at each point. As higher PSD indicates more damaged visual fields, and assuming that PSD has a positive correlation with PCI, one would expect that the higher the PCI value, the higher the PSD. We found a

positive correlation between PCI and PSD but it was statistically insignificant. We attribute the insignificance to selection of the patient. As PSD is not expected to rise in very advanced cases of glaucoma where there is generalized depression of the field, it may not reflect the severity of glaucoma in such cases. However in the study done by Franco et al.[87], the correlation between PCI and PSD revealed a negative correlation, though not statistical significant. They were unsure about the result and attributed it to the sample size or any selection bias.

There are some limitations in our study. As a structural damage of glaucoma we had considered only C/D ratio. However, it does not take into account disk hemorrhages, localized defects of the neural rim or the posterior bowing of the lamina cribrosa. Besides, glaucoma patients with small optic discs will have proportionally small C/D ratios, giving a falsely impression of healthy looking optic disk. Conversely, normal subjects with large disc will present with large C/D ratios giving a false impression of damaged optic disk. Hence, the C/D ratio is not a precise surrogate of glaucomatous optic disc damage without consideration of the relative disc size, area, and the quantitative assessment of neural rim width and area. Using this structural measure is a major shortcoming and quantitative measures of the optic disk structure as provided by new technologies should have been a better choice for correlation studies.

Another shortcoming of the study is the use of both eyes of the same individual. Doing so for the measurement of an attribute or variable, rather than selecting one eye at random or the more severe affected eye for analysis tend to overestimate variability, artifactually influencing *P* value and decreasing chances of

observing a significant effect, decreasing statistical power and increasing chances of type II error. We decided to use both eyes of the same patient to avoid waste of data.

Automated Perimetry, as a psychophysical test, is subject to patient cooperation and individual cognitive function causing imprecision of the measurements. We have tried to minimize this imprecision by selecting only automated perimetry exams with good reliable indices. However, we had taken patients with early cataract and that might have had influenced the value of MD.

Our study is a cross sectional study. Further longitudinal studies are warranted on the subject to explore other possible uses of PCI and strengthen its role as a unified risk factor and indicator of glaucoma severity. Other possible uses the can be explored are its use in glaucoma progression and thereafter in decision making of target pressure on the basis of PCI.

CONCLUSION AND SUMMARY

We found statistical significant difference between the values of Pressure to Cornea Index (PCI) among all the four groups. Hence we conclude that PCI can be used as a unified risk factor clinically in the management of glaucoma. Also we have found statistical significant correlation between structural and functional measures of glaucoma to Pressure to cornea index (PCI) and hence, we conclude that it can be used in assessing glaucoma severity as well.

PCI is a simple method and both the values of IOP and CCT are given importance. It can be easily calculated and does not require complex calculating formulas. It is an inexpensive method and does not require additional procedure

There is a lot of scope for further studies and work that can be done on this topic and explore other possible uses of PCI which can be beneficial in diagnosis and treatment of glaucoma.

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ANNEXURE

1. Annexure 1: Abbreviations
2. Annexure 2: Patient information sheet (English)
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ANNEXURE 1

LIST OF ABBREVIATIONS

IOP	Intraocular pressure
CCT	Central corneal thickness
PCI	Pressure to cornea index
GAT	Goldmann applanation tonometer
OHT	Ocular hypertension
NTG	Normal Tension Glaucoma
POAG	Primary Open Angle Glaucoma
MD	Mean deviation
PSD	Pattern standard deviation
C/D ratio	Cup to disc ratio
RNFL	Retinal nerve fiber layer
NRR	Neuro retinal rim

ANNEXURE 2

PARTICIPANT INFORMATION SHEET

TITLE: To study the correlation between the pressure-to-cornea index and both structural and functional measures of glaucoma

DATE:

1. Introduction:

You are being cordially invited to participate in the above titled study. The proposed study is a cross-sectional observational study to know clinical profile of person with POAG, NTG, OHT and normal individual.

2. What is the purpose of this study?

Purpose isTo analyse and study the pressure to cornea index in person with POAG, NTG, OHT and normal individual.

3. Why have I been chosen?

Your participation in this study is voluntary and at your free will.

4. Do I have to take part?

You can refuse to participate in the study. Moreover, you are also free to withdraw at any time without having to give a reason. Despite this, you will continue to receive your standard medical care and treatment.

5. How long will the study last?

This study will last for a period of 1 year and 11 months.

6. What will happen to me if I take part?

Complete examination and analysis of your reports.

7. What do I have to do?

Your cooperation in required in acquiring reports and for complete ophthalmic examination.

8. What is the drug being tested?

This study does not test efficacy of any drug.

9. What are the benefits of the study?

Outcome of the study may help in improving patient care in future.

10. What are the alternatives for treatment?

The design of the study does not involve any treatment procedure.

11. What are the side effects of the treatment received during the study?

Not applicable

12. What if new information becomes available?

Not applicable

13. What happens when the study stops?

It would not affect the group of patients as this study does not involve any treatment procedure

14. What if something goes wrong?

This study is an investigative study so it would not affect the participant involved in the study.

15. Will my taking part be kept confidential?

Yes

16. What else should I know?

Not applicable

17. Additional Precautions

Not required

18. Who to call with questions?

Dr Aakash Patel - 9913234486

ANNEXURE 3

સહભાગી માહિતી શીટ

શીર્ષક: દબાણ-થી-કોર્નીયા ઇન્ડેક્સ અને ઝામર બંને માળખાકીય અને વિધેયાત્મક પગલાં વચ્ચે સંબંધ અભ્યાસ

તારીખ:

1. પરિચય:

તમે અંતઃકરણપૂર્વક ઉપર શીર્ષક અભ્યાસમાં ભાગ લેવા માટે આમંત્રિત કરવામાં આવે છે. સૂચિત અભ્યાસ poag, NTG, oht અને સામાન્ય વ્યક્તિ સાથે વ્યક્તિ નૈદાનિક પ્રોફાઇલ જાણવા માટે ક્રોસ વિભાગીય નિરીક્ષણ અભ્યાસ છે.

2. આ અભ્યાસ હેતુ શું છે?

વિશ્લેષણ અને poag, NTG, oht અને સામાન્ય વ્યક્તિ સાથે વ્યક્તિ કોર્નીયા ઇન્ડેક્સ દબાણ અભ્યાસ કરવા માટે.

3. હું શા માટે પસંદ કરવામાં આવી છે?

આ અભ્યાસમાં તમારી ભાગીદારી સ્વૈચ્છિક અને તમારા મફત ઇચ્છા છે.

4. મારે ભાગ લેવા માટે હોય છે?

તમે અભ્યાસ ભાગ ઇન્કાર કરી શકો છો. વધુમાં, તમે પણ એક કારણ આપી કર્યા વગર કોઈપણ સમયે પાછી ખેંચી માટે મુક્ત છે. આમ છતાં, તમે તમારા પ્રમાણભૂત તબીબી સંભાળ અને સારવાર મેળવવા માટે ચાલુ રહેશે.

5. લાંબા કેવી રીતે અભ્યાસ ચાલશે?

આ અભ્યાસ 1 વર્ષ અને 11 મહિના સુધી ચાલશે.

6. હું ભાગ લેવા હોય તો મને શું થશે?

તમારી રિપોર્ટ્સ વિશ્લેષણ.

7. હું શું છે?

અમને સહકાર

8. ડ્રગ શું પરીક્ષણ કરવામાં આવી રહી છે?

આ અભ્યાસ કોઇ દવાની અસરકારકતા ચકાસવા નથી.

9. અભ્યાસ શું ફાયદા છે?

દર્દીઓ પરિણામ સુધારવા.

10. સારવાર માટે આ વિકલ્પો શું છે?

આ અભ્યાસના ડિઝાઇન કોઈપણ સારવાર પ્રક્રિયા સમાવેશ કરતું નથી.

11. અભ્યાસ દરમિયાન પ્રાપ્ત સારવાર ની આડઅસરો શું છે?

લાગુ નથી

12. નવી માહિતી ઉપલબ્ધ બને તો શું?

લાગુ નથી

13 .અભ્યાસ અટકે છે જ્યારે 13 શું થાય છે?

આ અભ્યાસમાં કોઇ સારવાર પ્રક્રિયા સમાવેશ કરતું નથી, કે દર્દીઓની જૂથ અસર કરતું નથી

14. શું કંઈક ખોટું થાય તો?

આ અભ્યાસ તપાસ અભ્યાસ તેથી તે અભ્યાસમાં સામેલ સહભાગી અસર કરતું નથી છે.

15. મારા ભાગ લેવા ગુપ્ત રાખવામાં આવશે?

હા

16. હું બીજું શું જાણવું જોઈએ?

લાગુ નથી

17. વધારાની સાવચેતી

જરૂરી નથી

18 પ્રશ્નો સાથે કોને કૉલ કરવા માટે?

ડૉ આકાશ પટેલ (9913234486)

ANNEXURE 4

Informed Consent Form (ICF) for Participants in

Research Programmes involving studies on human beings

Study Title: - To study the correlation between the pressure-to-cornea index and both structural and functional measures of glaucoma

Please initial box (Subject)

1.	I confirm that I have read and understood the information sheet dated ……....….for the above study and have had the opportunity to ask questions.	☐
(ii)	I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.	☐
(iii)	I understand that the Sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published.	☐
(iv)	I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s)	☐
(v)	I agree to take part in the above study.	☐

Signature (or thumb impression) of the subject/ LAR: _____

Date:____/

Signatory's Name:_____

Signature of the Investigator:_____

Date:____/____/_____

Study Investigator's Name:_____

Signature of the Witness_____

Date:____/____/_____

Name of the Witness:

ANNEXURE 5

સહભગી માહિતી પત્રક

<u>અભ્યાસમાં ભાગ લેવા માટે સમજી વિચારીને આપેલી પરવાનગીનુ સમ્મતિ-પત્રક</u>

અભ્યાસનુનામ: " દબાણ-થી-કોર્નીયા ઇન્ડેક્સ અને ઝામર બંને માળખાકીય અને વિધેયાત્મક પગલાં વચ્ચે સંબંધ અભ્યાસ"

અભ્યાસ ક્રમાંક:_____ તારીખ:_____

સહભાગીનુ
પુરુંનામ:_____

સહભાગીનુ
ટુંકુનામ:_____

૧. હું ખાત્રી આપું છું કે મે ઉપરોક્ત અભ્યાસની (તા: / /) માહિતીવાંચી છે અને સમજી છે અને તે અંગેના મુંઝવતા પ્રશ્નો પુછવાની મને તક આપવામા આવી છે.
૨. હું જાણું છું કે આ અભ્યાસમાં ભાગ લેવો મારા માટે મરજીયાત છે અને, કોઈપણ જાતનુ કારણ આપ્ય વગર, તે માથી ગમે ત્યાર ખસી જવાની મને છૂટ છે, અને આમ કરવાથી મારી તબીબી સારવાર કેકાયદેસરના હક્કોને કોઈ અસર નહીં થાય.
૩. હું જાણુ છું કે આ અભ્યાસના તપાસ કર્તા, તેમના મદદનીશો, એથિકલટીમ અને તેના ઉપર દેખરેખ રાખતા અધિકારીઓને મારા સ્વાસ્થયની કોઈ પણ જાતની માહિતી, સદર અભ્યાસને લગતી કે તેસિવાયની, મેળવવા માટે મારી પરવાનગીની જરૂરરહેશેનહીં, ભલે પછી હું અભ્યાસમાંથી ખસી જાઉં. હું જાણું છું કે મારી આ પ્રકારની માહિતી અન્યકોઈને જાણ કે પ્રસિધ્ધ નહીં કરવામાંઆવે.
૪.આ અભ્યાસ દરમ્યાન, અથવા તેના અંતે પ્રાપ્ત થતી માહિતી: કોઈ પણ જાતની વૈજ્ઞાનિકશોધ માટે ઉપયોગ કરવા માટે હું સ્વૈચ્છિક રીતે છુટ આપું છું.
૫.હું આ અભ્યાસમાં ભાગ લેવા / જોડાવા માટે સહમતિ આપું છું.

અભ્યાસમાંભાગલેનારનીસહિઅથવાઅંગુઠાનુનિશાન:_____ તારીખ:_____

કાયદેસરનાસ્વિકૃતતપાસકર્તાનીસહી: _____ તારીખ:_____

તપાસકર્તાનુનામ: _____

તટસ્થસાહેદ / ગવાહનીસહી: _____
તારીખ:_____

તટસ્થસાહેદ / ગવાહનુનામ:
_____

ANNEXURE 6

PROFORMA FOR THESIS

Topic : To study the correlation between the pressure-to-cornea index and both structural and functional index of glaucoma.

Sr. No:
Date:

Name of Patient:-

OPD number:-

Date of birth:-

Age/Sex/Occupation:-

Address/Contact no.:-

Chief Complaints:

Negative history:

Past history - H/O ocular surgery/trauma/ocular disease

H/O any systemic illness:

Family history:

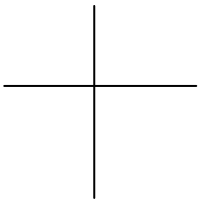
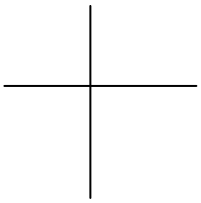
Personal history:

Drug history:

Topic : To study the correlation between the pressure-to-cornea index and both structural and functional index of glaucoma.

	R.E	L.E
Uncorrected visual acuity		
Vision with pinhole		
Best corrected visual acuity		
Present spectacle power		
Vision with present spectacle		

R.E L.E

Retinoscopy  

-Head Posture:-

-Eye position:-

-Ocular Motility:-

Slit lamp examination:-

Fundus

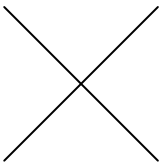
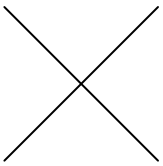
Perimetry:

Topic : To study the correlation between the pressure-to-cornea index and both structural and functional index of glaucoma.

Tonometry:

- NCT :
- APLLANATION :

Pachymetry:

Gonioscopy:  

Pressure to cornea index:-

ANNEXURE 7

Key to Master chart

OD – Right eye

OS – Left eye

OU – Both eyes

M – Male

F– Female

Sr.No. – Serial number

DOE- Date of examination

DOB – Date of birth

H/O –History of

DM – Diabetes mellitus

HTN – Hypertension

COPD- Chronic obstructive pulmonary disease

IOP- Intraocular pressure

NAD- No abnormality detected

WNL – Within normal limit

NS – Nuclear sclerosis (LOCS grading)

Sr. No	Name	Age	Sex	Eye	UCVA	BCVA	Anterior Segment	Disc (CDR)	Posterior segment	NCT	AT	Pachymetry	PCI	Diagnosis	Code	Mean Deviation	Pattern Standard Deviation
1	Madhukar Birhade	60	M	R	6/18	6/6	Wnl	0.4	wnl	23	24	0.575	126.24	OHT	3	-4.89	1.47
2	Madhukar Birhade	60	M	L	6/18	6/6	Wnl	0.4	wnl	22	22	0.575	115.72	OHT	3	-4.20	1.37
3	Kantibhai Patel	58	M	R	6/36	6/18	lens - NS II	0.7	wnl	28	30	0.540	190.52	POAG	4	-8.93	12.47
4	Kantibhai Patel	58	M	L	6/36	6/18	lens - NS II	0.8	wnl	28	30	0.540	190.52	POAG	4	-15.12	11.60
5	Jentibhai Patel	60	M	R	6/60	6/12	lens - NS II	0.8	wnl	27	28	0.560	159.44	POAG	4	-14.76	10.80
6	Jayantibhai Patel	62	M	R	6/6	6/6	Wnl	0.6	wnl	23	27	0.570	145.79	OHT	3	-3.83	3.93
7	Jayantibhai Patel	62	M	L	6/6	6/6	Wnl	0.6	wnl	25	27	0.570	145.79	OHT	3	-3.45	1.60
8	Nirmala Parihar	53	F	R	6/18	6/6	Wnl	0.3	wnl	14	14	0.530	94.04	N	1	-4.00	3.12
9	Nirmala Parihar	53	F	L	6/12	6/6	Wnl	0.3	wnl	14	14	0.530	94.04	N	1	-3.55	2.81
10	Saheda Vohra	62	F	R	6/9(p)	6/6(p)	lens - NS II	0.5	wnl	18	20	0.560	113.88	N	1	-2.98	7.38
11	Saheda Vohra	62	F	L	6/12	6/6(p)	lens - NS II	0.5	wnl	18	18	0.560	102.50	N	1	-2.24	2.83
12	yakub Patel	60	M	R	Cf4m	6/12	lens - NS II	0.9	wnl	15	13	0.480	117.55	NTG	2	-22.80	10.78
13	Indira Joshi	71	F	R	6/36	6/24	lens - NS II	0.7	wnl	28	33	0.590	160.68	POAG	4	-6.30	4.81
14	Indira Joshi	71	F	L	6/36	6/24	lens - NS II	0.7	wnl	28	34	0.590	165.55	POAG	4	-8.92	11.73
15	Haribhai Patel	62	M	R	6/6	6/6	Wnl	0.8	wnl	13	16	0.530	107.47	NTG	2	-21.82	6.41
16	Haribhai Patel	62	M	L	6/6	6/6	Wnl	0.7	wnl	14	16	0.530	107.47	NTG	2	-16.33	10.45
17	Jamila taiyabali	54	F	R	6/36	6/9	Wnl	0.6	wnl	13	11	0.490	93.50	N	1	-4.82	2.41
18	Jamila taiyabali	54	F	L	6/60	6/12	Wnl	0.6	wnl	13	11	0.490	93.50	N	1	-8.48	7.69
19	Devraj Kathariya	65	M	R	6/18	6/9	Wnl	0.8	wnl	13	14	0.470	134.84	NTG	2	-28.02	4.47
20	Devraj Kathariya	65	M	L	6/18	6/9	Wnl	0.8	wnl	13	14	0.470	134.84	NTG	2	-29.17	4.18
21	Madhu Gupta	58	F	R	6/9	6/6	Wnl	0.4	wnl	14	15	0.550	90.16	N	1	0.05	2.20
22	Madhu Gupta	58	F	L	6/9	6/6	Wnl	0.4	wnl	14	15	0.550	90.16	N	1	-0.46	2.25
23	Amulakh Kumar	60	M	R	6/6	6/6	Wnl	0.5	wnl	18	18	0.570	97.20	N	1	-4.45	1.99
24	Amulakh Kumar	60	M	L	6/6	6/6	Wnl	0.5	wnl	18	18	0.570	97.20	N	1	-4.10	2.07
25	Dinesh desai	54	M	R	6/6	6/6	Wnl	0.3	wnl	17	18	0.600	83.33	N	1	1.70	1.64
26	Dinesh desai	54	M	L	6/6	6/6	Wnl	0.3	wnl	17	18	0.600	83.33	N	1	0.47	1.64
27	Gokulsing rajput	60	M	R	6/12	6/6	Wnl	0.7	wnl	20	18	0.550	108.19	NTG	2	-12.43	14.30
28	Gokulsing rajput	60	M	L	6/12	6/6	Wnl	0.7	wnl	20	18	0.550	108.19	NTG	2	-13.00	11.64
29	Saryuben soni	60	F	R	6/9	6/6	Wnl	0.3	wnl	15	15	0.530	100.75	N	1	-5.67	6.46
30	Gopalsing Mateda	60	M	R	6/6	6/6	Wnl	0.4	wnl	22	26	0.600	120.37	OHT	3	-1.87	4.17
31	Gopalsing Mateda	60	M	L	6/6	6/6	Wnl	0.4	wnl	22	26	0.600	120.37	OHT	3	-1.14	1.61
32	Pushpaben Thakor	45	F	R	6/6	6/6	Wnl	0.7	wnl	15	16	0.580	82.00	N	1	-3.03	2.66
33	Pushpaben Thakor	45	F	L	6/6	6/6	Wnl	0.7	wnl	15	16	0.580	82.00	N	1	-4.23	2.82
34	Savita Patel	50	F	R	6/60	6/6	Wnl	0.4	wnl	13	12	0.530	80.60	N	1	-4.43	2.60
35	Savita Patel	50	F	L	6/60	6/6	Wnl	0.4	wnl	13	12	0.530	80.60	N	1	-5.39	5.72
36	Jatin parmar	52	M	R	6/6	6/6	Wnl	0.7	wnl	22	27	0.550	162.28	POAG	4	-3.34	6.29
37	Jatin parmar	52	M	L	6/6	6/6	Wnl	0.7	wnl	22	27	0.550	162.28	POAG	4	-3.53	2.96
38	Badrilal Dhakad	68	M	R	6/18	6/12	Wnl	0.9	wnl	13	14	0.470	134.84	NTG	2	-22.70	8.62
39	Badrilal Dhakad	68	M	L	6/18	6/12	Wnl	0.9	wnl	13	14	0.470	134.84	NTG	2	-30.29	5.15
40	Takuben Bhavsar	47	F	R	6/9	6/6	Wnl	0.7	wnl	22	21	0.495	173.14	POAG	4	-11.38	6.28
41	Takuben Bhavsar	47	F	L	6/9	6/6	Wnl	0.7	wnl	23	21	0.495	173.14	POAG	4	-11.94	6.42
42	Warney Wilfred	65	M	R	6/60	6/24	lens - NS III	0.8	wnl	12	12	0.480	108.51	NTG	2	-10.18	9.23
43	Warney Wilfred	65	M	L	6/60	6/18	lens - NS III	0.8	wnl	12	12	0.480	108.51	NTG	2	-7.65	10.62
44	Reena Radadiya	20	F	R	6/6	6/6	Wnl	0.4	wnl	14	15	0.530	100.75	N	1	0.50	3.94
45	Reena Radadiya	20	F	L	6/6	6/6	Wnl	0.4	wnl	14	15	0.530	100.75	N	1	0.66	3.14
46	Malik kansharba	50	M	R	6/12	6/6	lens - NS I	0.8	wnl	24	25	0.520	177.80	POAG	4	-12.01	8.85
47	Malik kansharba	50	M	L	6/12	6/6	lens - NS I	0.8	wnl	24	25	0.520	177.80	POAG	4	-17.45	13.05
48	Jisham Rathwa	61	M	R	6/18	6/18	lens - NS II	0.4	wnl	24	26	0.600	120.37	OHT	3	-4.40	1.87
49	Khanvir Chandrasinh	61	M	R	6/24	6/24	lens - NS III	0.7	wnl	12	14	0.500	112.00	NTG	2	-6.36	4.40
50	Khanvir Chandrasinh	61	M	L	6/24	6/24	lens - NS III	0.7	wnl	12	14	0.500	112.00	NTG	2	-8.04	4.53

51	Anwar Mirza	60	M	R	6/24	6/24	lens - NS II	0.8	wnl	24	26	0.510	196.00	POAG	4	-19.23	9.22
52	Anwar Mirza	60	M	L	6/36	6/36	lens - NSIII	0.9	wnl	24	27	0.510	203.54	POAG	4	-29.96	4.49
53	Alka Sinha	56	F	R	6/12	6/12	lens - NS I	0.7	wnl	12	13	0.480	117.55	NTG	2	-8.67	5.96
54	Alka Sinha	56	F	L	6/12	6/12	lens - NS I	0.9	wnl	12	13	0.470	125.21	NTG	2	-17.45	13.05
55	Rajesh Sinha	58	M	L	6/12	6/12	lens - NS II	0.5	wnl	14	24	0.600	111.11	OHT	3	-4.40	1.87
56	Patel Abdulsalam	61	M	R	6/24	6/24	lens - NS II	0.7	wnl	12	14	0.490	119.00	NTG	2	-11.03	11.62
57	Patel Abdulsalam	61	M	L	6/36	6/36	lens - NS III	0.7	wnl	12	14	0.490	119.00	NTG	2	-7.98	7.45
58	Jahir Pathan	58	M	R	6/24	6/24	lens - NS III	0.8	wnl	14	14	0.470	134.84	NTG	2	-14.15	7.84
59	Jahir Pathan	58	M	L	6/24	6/24	lens - NS III	0.7	wnl	14	14	0.470	134.84	NTG	2	-5.45	6.90
60	Vimlaben Patel	59	F	R	6/18	6/12	lens - NS II	0.7	wnl	26	25	0.500	200.00	POAG	4	-7.52	6.30
61	Vimlaben Patel	59	F	L	6/18	6/12	lens - NS II	0.9	wnl	26	28	0.500	224.00	POAG	4	-15.50	8.59
62	Rama Bhil	42	M	R	6/6	6/6	Wnl	0.3	wnl	22	24	0.580	123.01	OHT	3	-1.06	2.19
63	Rama Bhil	42	M	L	6/6	6/6	Wnl	0.3	wnl	23	24	0.580	123.01	OHT	3	-1.06	2.19
64	Anshulkumar Roy	24	M	R	6/9	6/6	Wnl	0.5	wnl	28	28	0.580	143.51	OHT	3	-3.95	2.24
65	Anshulkumar Roy	24	M	L	6/9	6/6	Wnl	0.5	wnl	28	28	0.580	143.51	OHT	3	-3.73	3.40
66	Pamuben Bharvad	62	F	R	6/12	6/9	lens - NS II	0.8	wnl	20	20	0.460	205.47	POAG	4	-24.94	6.57
67	Pamuben Bharvad	62	F	L	6/12	6/9	lens - NS II	0.8	wnl	20	20	0.460	205.47	POAG	4	-28.83	5.48
68	Kantibhai Patel	53	M	R	6/6	6/6	Wnl	0.6	wnl	21	25	0.520	177.80	POAG	4	-6.23	8.02
69	Kantibhai Patel	53	M	L	6/6	6/6	Wnl	0.6	wnl	26	25	0.520	177.80	POAG	4	-4.10	5.59
70	Padma Soni	45	F	R	6/6	6/6	Wnl	0.7	wnl	22	24	0.520	170.69	POAG	4	-11.25	8.85
71	Padma Soni	45	F	L	6/6	6/6	Wnl	0.7	wnl	22	24	0.520	170.69	POAG	4	-9.28	7.91
72	Ramesh More	46	M	R	6/6	6/6	Wnl	0.3	wnl	24	22	0.590	107.12	OHT	3	-2.78	3.65
73	Ramesh More	46	M	L	6/6	6/6	Wnl	0.3	wnl	24	22	0.590	107.12	OHT	3	-4.10	5.59
74	Riddhi Modi	48	F	R	6/6	6/6	Wnl	0.7	wnl	23	24	0.520	170.69	POAG	4	-7.52	6.30
75	Riddhi Modi	48	F	L	6/6	6/6	Wnl	0.8	wnl	23	24	0.510	180.93	POAG	4	-15.50	8.59
76	Anwarhusen Shaikh	59	M	R	6/24	6/24	lens - NS II	0.8	wnl	23	25	0.510	188.46	POAG	4	-18.37	3.70
77	Anwarhusen Shaikh	59	M	L	6/36	6/36	lens - NS III	0.9	wnl	25	25	0.510	188.46	POAG	4	-26.35	5.87
78	Shankarlal	59	M	R	6/12	6/12	lens - NS II	0.6	wnl	25	25	0.540	158.77	OHT	3	-0.12	1.69
79	Shankarlal	59	M	L	6/12	6/12	lens - NS II	0.6	wnl	25	25	0.540	158.77	OHT	3	-0.12	1.69
80	Khukhar singh	58	M	R	6/12	6/12	lens - NS II	0.5	wnl	24	24	0.600	111.11	OHT	3	-0.43	1.94
81	Khukhar singh	58	M	L	6/12	6/12	lens - NS II	0.5	wnl	24	24	0.600	111.11	OHT	3	-1.18	2.81
82	Zaltana	47	F	R	6/9	6/9	lens - NS I	0.4	wnl	24	27	0.590	131.46	OHT	3	-0.51	1.80
83	Zaltana	47	F	L	6/9	6/9	lens - NS I	0.4	wnl	24	27	0.590	131.46	OHT	3	-0.69	1.72
84	Ramaben	47	F	R	6/6	6/6	Wnl	0.4	wnl	25	26	0.560	148.05	OHT	3	-4.42	3.79
85	Ramaben	47	F	L	6/6	6/6	Wnl	0.4	wnl	25	26	0.560	148.05	OHT	3	-1.99	2.69
86	Akhilesh Yadav	52	M	R	6/6	6/6	Wnl	0.2	wnl	15	15	0.550	90.16	N	1	0.40	1.85
87	Akhilesh Yadav	52	M	L	6/6	6/6	Wnl	0.2	wnl	15	15	0.550	90.16	N	1	0.38	1.36
88	Khunkhar	62	M	R	6/9	6/9	lens - NS II	0.2	wnl	12	13	0.540	82.56	N	1	-0.81	1.96
89	Khunkhar	62	M	L	6/9	6/9	lens - NS II	0.2	wnl	12	13	0.540	82.56	N	1	0.36	1.51
90	Shahrukh pathan	61	M	R	6/9	6/9	lens - NS II	0.2	wnl	12	13	0.530	87.32	N	1	-1.16	2.01
91	Shahrukh pathan	61	M	L	6/9	6/9	lens - NS II	0.2	wnl	12	13	0.530	87.32	N	1	-0.30	1.83
92	Premkumar prajapati	55	M	R	6/24	6/24	lens - NS III	0.9	wnl	24	13	0.470	125.21	NTG	2	-21.24	9.66
93	Premkumar prajapati	55	M	L	6/24	6/24	lens - NS III	0.9	wnl	12	13	0.470	125.21	NTG	2	-23.59	6.35
94	Jayantibhai Patel	62	M	R	6/24	6/24	lens - NS III	0.8	wnl	12	13	0.480	117.55	NTG	2	-7.11	8.66
95	Jayantibhai Patel	62	M	R	6/24	6/24	lens - NS III	0.8	wnl	12	13	0.480	117.55	NTG	2	-7.11	8.66
96	kanubhai kachela	50	M	R	6/12	6/12	lens - NS II	0.8	wnl	12	14	0.480	126.59	NTG	2	-10.76	12.25
97	kanubhai kachela	50	M	L	6/12	6/12	lens - NS II	0.8	wnl	12	14	0.480	126.59	NTG	2	-11.00	12.00
98	Lilly	37	F	R	6/6	6/6	Wnl	0.3	wnl	21	22	0.590	107.12	OHT	3	-1.41	1.89
99	Lilly	37	F	L	6/6	6/6	Wnl	0.3	wnl	21	22	0.590	107.12	OHT	3	-1.41	1.89
100	Devilal	60	M	R	6/18	6/6	Wnl	0.4	wnl	23	24	0.575	126.24	OHT	3	-4.89	1.47