A Dissertation on

A CLIICAL STUDY ON VISUAL EVOKED POTETIAL CHANGES AND VISUAL FIELD DEFECTS IN COPD PATIENTS – A CROSS SECTIONAL STUDY

Submitted to the

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the Regulations

for the Award of the Degree of

M.S. (BRANCH - III)

OPHTHALMOLOGY

GOVT. STANLEY MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 001

MAY 2020

CERTIFICATE

This is to certify that the study entitled "**A CLIICAL STUDY ON VISUAL EVOKED POTETIAL CHANGES AND VISUAL FIELD DEFECTS IN COPD PATIENTS – A CROSS SECTIONAL STUDY**" is the result of original work carried out by **Dr. A. KAOWSHALYAA**, under my supervision and guidance at **GOVT. STANLEY MEDICAL COLLEGE, CHENNAI.** The thesis is submitted by the candidate in partial fulfilment of the requirements for the award of **M.S Degree in Ophthalmology**, a course from **2017 to 2020** at Govt. Stanley Medical College, Chennai.

Prof. Dr.R.Shanthi Malar M.D.,D.A.,
The Dean
Govt. Stanley Medical College,
Chennai-600 001

Prof.Dr.Thangerani Raajaseharan M.S.,D.O.,
Unit chief & Head of the Dept.,
Govt. Stanley Medical College,
Chennai-600 001

CERTIFICATE FROM GUIDE

This is to certify that the dissertation entitled **A CLINICAL STUDY ON VISUAL EVOKED POTENTIAL CHANGES AND VISUAL FIELD DEFECTS IN COPD PATIENTS – A CROSS SECTIONAL STUDY** is a bonafide record of research work done by **Dr.A.KAOWSHALYAA,** Post Graduate Resident in Dept of ophthalmology, Government Stanley medical college, Chennai

Prof .Dr.Thangerani Raajaseharan M.S.,D.O.,
HOD & Professor of ophthalmology
Govt Stanley medical college
Chennai -01

DECLARATION

I hereby declare that this dissertation entitled "**A CLINICAL STUDY ON VISUAL EVOKED POTENTIAL CHANGES AND VISUAL FIELD DEFECTS IN COPD PATIENTS – A CROSS SECTIONAL STUDY**" is a bonafide and genuine research work carried out by me under the guidance of **Prof.Dr.THANGERANI RAAJASEHARAN M.S.,D.O.,** HOD, Department of Ophthalmology, Government Stanley Medical College and Hospital, Chennai - 600 001.

Date:

Place: Chennai

Signature

(Dr. A. KAOWSHALYAA)

ACKNOWLEDGEMENT

I express my immense gratitude to the Dean, **Prof. Dr. R. SHANTHI MALAR, M.D.,D.A.,** Government Stanley Medical College**,** Chennai for giving me the opportunity to work on the study.

With overwhelming respect and gratitude, I wish to express my heartfelt and profound indebtness to my teacher **Prof. Dr.THANGERANI RAAJASEHARAN M.S.,D.O.,** Head of Department, Department of Ophthalmology Government Stanley Medical College**,** Chennai-01**.** for here valuable advice, and guidance, in this endeavour. Her kind attitude and encouragement have been a source of inspiration throughout this study, which helped me to do my best in this effort.

I would like to thank **Prof. Dr. K. VINAYAGAMURTHY M.S.,** for his vauable guidance and suggestions for the study.

I am very grateful to **Prof. Dr. M. S. GOKILA M.S.,D.O.,** for her timely support and guidance.

I would like to thank **Prof. Dr. K.S.T.LATHA M.S.,** for her timely suggestions and guidance.

I am thankful to **Prof Dr. NANCY GLORY M.D.,** Department of CHEST MEDICINE, **Prof. Dr. S. ARUNAN M.D.,DM**., Department of NEUROMEDICINE, Government Stanley Medical College, Chennai, for their support and guidance without which this would not have been possible.

My heartfelt thanks to my teachers and Assistant Professorl to **Dr. S. VENKATESH M.S., Dr .K. VINODHINI M.S.,D.O., Dr. S. HEMAPRIYA DO.,DNB.,** for rendering their valuable suggestions and supervision throughout the progress of work.

I specially thank Assistant Professor **Dr. K. SIVAKUMAR M.S.,** for his supervision and assistance throughout my thesis work.

I am thankful to all my colleagues and friends for their support.

I am deeply indebted to all my patients for their sincere cooperation for completion of this study.

Finally, my heartfelt thanks to my sister and parents for their encouragement, motivation and timely help during the course of this study.

Date:

Place: Chennai

(Dr. A. KAOWSHALYAA)

GOVERNMENT STANLEY MEDICAL COLLEGE& HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

Title of the Work : "A CLINICAL STUDY ON VISUAL EVOKED POTENTIAL CHANGES AND VISUAL FIELD DEFECTS IN COPD PATIENTS-A CROSS SECTIONAL STUDY"

Principal Investigator : DR. A. KAOWSHALYAA,

Designation : Postgraduate in Ophthalmology (2017-2020),
Department of Ophthalmology,
Govt. Stanley medical college & hospital.
Chennai.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 21.12.2017 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator, investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY
Stanley Medical College

PLAGIARISM CERTIFICATE

URKUND

Urkund Analysis Result

Analysed Document:	thesis COPD.docx (D57293938)
Submitted:	10/20/2019 3:20:00 AM
Submitted By:	drkaow29@gmail.com
Significance:	12 %

Sources included in the report:

Dr Anitcheady K.docx (D31029009)
JP THESIS .docx (D31197251)
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2954376/
https://www.researchgate.net/publication/282382027_Evaluation_of_visual_evoked_potential_VEP_in_patients_with_chronic_obstructive_pulmonary_disease_COPD
https://respiratory-research.biomedcentral.com/articles/10.1186/s12931-019-1053-7
https://www.ncbi.nlm.nih.gov/pubmed/22489568
https://www.ncbi.nlm.nih.gov/books/NBK107218/
2db69769-d2d8-427a-aa07-8401fccf4970
https://www.dovepress.com/evaluation-of-central-and-peripheral-neuropathy-in-patients-with-chron-peer-reviewed-fulltext-article-COPD
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4332314/
https://www.researchgate.net/publication/6303879_The_evaluation_of_cognitive_functions_with_P300_test_for_chronic_obstructive_pulmonary_disease_patients_in_attack_and_stable_period

Instances where selected sources appear:

39

CERTIFICATE II

This is to certify that this dissertation work titled **“A CLINICAL STUDY ON VISUAL EVOKED POTETIAL CHANGES AND VISUAL FIELD DEFECTSIN COPD PATIENTS – A CROSS SECTIONAL STUDY** of the candidate **Dr. A. KAOWSHALYAA** with registration number **221713051** for the award of **M.S OPHTHALMOLOGY**. I personally verified that the urkund.com website for the purpose of checking plagiarism. I found that the uploaded thesis file contains contents from introduction to conclusion and result shows **12%** of plagiarism in the dissertation.

Guide & supervisor sign with Seal.

Place: Chennai
Date:

Prof .Dr.THANGERANI RAAJASEHARAN
M.S.,D.O.,
HOD & Professor of ophthalmology
Govt Stanley medical college
Chennai -01

PART – I

PART – II

ANNEXURES:

ABBREVIATIONS

1. COPD	–	CHRONIC OBSTRUCTIVE PULMONARY DISEASE
2. GOLD	–	GLOBAL INITIATIVE FOR CHRONIC OBSTRUCTIVE LUNG DISEASE
3. PAO2	–	ARTERIAL PARTIAL PRESSURE OXYGEN
4. VP	–	VENTILATION PERFUSION
5. VEP	–	VISUAL EVOKED PERFUSION
6. CNS	–	CENTRAL NERVOUS SYSTEM
7. ATS	–	AMERICAN THORACIC SOCIETY
8. ETS	–	ENVIRONMENTAL TOBCCO SMOKE
9. AATD	–	ALPHA 1 ANTITRYPSIN DEFFICIENCY
10.CD8+	-	CLUSTER OF DIFFERENTIATION 8+
11.CD4+	-	CLUSTER OF DIFFERENTION 4+
12.PRVEP	–	PATTERN REVERSAL VISUAL EVOKED POTENTIAL
13.EP	–	EVOKED POTENTIAL
14. EEG	–	ELECTROENCEPHALOGRAPHY
15.MS	–	MULTIPLE SCLEROSIS
16.MRI	–	MAGNETIC RESONANCE IMAGING
17.VF	–	VISUAL FIELD
18.FEV	–	FORCED EXPIRATORY VOLUME
19.FVC	–	FORCED VITAL CAPACITY

PART-I

INTRODUCTION

The COPD was referred by names such as “chronic airflow obstruction” and “chronic obstructive lung disease.”It is defined by the Global initiative for Chronic Obstructive Lung Disease (GOLD) as “A preventable and treatable disease with some signficant extrapulmonary effects that may contribute to the severity in the individual patient”.[1]Airflow are restricted which is fully not reversible. The airflow restrcition is usually progressive and inflammatory response is associated with noxious particles or gases.[1]Pontomedullary portion of brain is believed to be affected by smoking, COPD, airway obstruction by altering blood gases causing hypoxemia, hypercapnia, and respiratory acidosis.[1]Patients with mild stages of COPD may have significant cardiovascular physiologic impairments. Individuals with COPD cannot achieve maximum oxygen uptake or a maximum heart rate. Due to their physical de-conditioning they get exhausted easily and their excercise performance is compromised even in patients with only mild degrees of airflow obstruction and without hypoxemia.[1]There is a need now to recognize COPD as a systemic disease and start to understand the metabolic and musculoskeletal implications of this generalized process. COPD has been recognized as a risk factor for cardiovascular morbidity and mortality. When arterial partial oxygen pressure (PaO2) falls below 60mmhg hypoxia occurs and this causes systemic effects.COPD is a progressive disease in which hypoxemia occurs due to VP

(ventilation perfusion) mismatch.Many studies have shown peripheral neuropathy as an extra pulmonary manifestation of COPD.Association of COPD patients with peripheral neuropathyhas been reported in previous studies.

Visual evoked potentials (VEP), the electrical potentialdifference generated in response to visual stimuli, provide a functional measurement of optical pathway.[2]VEP waveform is generated in striate, peristriate occipital cortex and thalamocortical volleys.[2]It indicates functional aspects of optic nerve, optic chiasm and tracts, lateral geniculate bodies and geniculocalcarine projection to visual cortex.The normal visual evoked potential (VEP) reflects the functional integrity of the visual pathways from retina to occipital striate area.Visual receptors are sensitive to hypoxia.Moreover, this evaluation can provide valuable evidence of optic nerve involvement in the primary stages of ophthalmic disease.[2]The VEP in an adult individual consists of three negative and three positive waves within a span of 350msec after the application of the stimulus.Out of first three waves, N1 (N75), P1(P100), and N2(N145) of NPN complex, the latency and amplitude of P1 is clinically important.[2]In nueroophthalmologic disorders visual field assessment also is one of the major diagnostic tools.Retinal sensitivity is reduced and are associated with local ischemia/hypoxia due to COPD related vascular changes, low oxygen saturation and incrseased oxidative stress.With this background the study was undertaken to correlate VEP changes in stable COPD patients and to evaluate the effect on the visual field in patients with COPD.

NEED FOR STUDY

Chronic obstructive pulmonary disease (COPD), is a public health problem seen worldwide being the fourth largest international cause of death and second largest cause of death in India.It has been identified to have multisystem involvement with significant extra pulmonary manifestations including central and peripheral neuropathy.Several studies have been conducted regarding peripheral neuropathy, however studies regarding its effect on CNS and visual pathway is limited.Considering its severity in causing visual pathway impairment, our study was carried out to find out functional integrity of the visual pathway through the visually evoked potentials (VEP) in COPD patients and to evaluate the effect on the visual field in patients with COPD.

REVIEW OF LITERATURE

HISTORY OF COPD

In earlier days patients suffering from these symptoms (chronic bronchitis, chronic asthma) were categorized by either pathological changes (emphysema) or by physiological correlates (pink puffers, blue bloaters). Recognizing these entities were overlapping in nature and often coexisted leading them to the term COPD.[2]

In current scenario it has been realized that COPD coexisted with a number of co-morbidities, e.g. ischemic heart disease, hypertension, diabetes, heart failure and cancer, suggesting that there was a generalized systemic inflammatory process. Higgins in 1959 organized the clear relationship between "smoking and persistent cough and sputum production.[3]COPD is probably not going to be new condition. Within the past, several physicians have used completely different terminologies for what we all know these days as COPD. In 1846, John Hutchinson innovated the spirometer as measuring instrument whch was essential in the diagnosis and treatment of COPD. It took almost hundred years for Tiffeneau to adjoin the concept of timed vital capacity as a measure of airflow, for spirometry to become complete as a diagnostic instrument (Tiffeneau and Pinelli 1947).[5] Gaensler found the notion of the air velocity index afterwards on the forced vital capacity, supported on Tiffeneau's work; which became the basis of the FEV_1 and FEV_1/FVC percent (Gaensler 1950).[6]

In 1956 Barach and Bickerman altered the primary comprehensive text book, pulmonary emphysema, that demonstrates the treatment of the era. Contributors to the current book enclosed Dayman, the primary to recognize the spirometric and flow volume designs designative of dynamic expiratory airway collapse in emphysema.[9]COPD may involve one or more system that is repeatedly associated with significant extra pulmonary manifestations.[2]

COPD - Definition

COPD was mentioned by names like “chronic airflow obstruction” and “chronic obstructive lung disease.” Dr. William Briscoe assumed to be the first person to use the term “chronic obstructive pulmonary disorder” at the Ninth Aspen respiratory disorder Conference in June of 1965.[2]In 1959, a gathering of medical professionals known as the Ciba Guest Symposium helped outline the components that make up the definition and management of COPD as we all know it in recent times.[2]Some physicins jointly outlined the definition of chronic bronchitis by The American Thoracic Society (ATS) incorporating chronic cough lasting at least three months for at least two years. By contrast, the society defined emphysema in biological terms of enlarged alveolar spaces and loss of alveolar walls.[10]Asthmatic bronchitis was thought carefully an overlapping condition (Committee on Diagnostic Standards for Nontuberculous Respiratory Diseases 1962).[10]

COPD (CHRONIC OBSTRUCTIVE PULMONARY DISEASE):

COPD is a public health problem seen worldwide.Chronic Obstructive Pulmonary Disease is a progressive disease drawing attention all over the world, causing major health care burden. It is quite common and is the fourth- leading international cause of death and is one of the major causes of chronic respiratory failure. Routinely occuring respiratory symptoms include dyspnea, cough and/or sputum production. It is elucidated by the Global Initiative for Chronic Obstructive Lung Disease(GOLD), as "A preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in the individual patient.[19] It is mainly caused by hazardous dusts and particles, particularly smoking, and other environmental and personal risk factors, and characterized by airflow limitation, with co-existence of chronic bronchitis and emphysema, and not fully reversible.[19]

RISK FACTORS OF COPD:[19]

Tobacco smoke - additionally cigarette, pipe, cigar, water-pipe and other types of tobacco smoking common in many countries, and also environmental tobacco smoke (ETS). Air pollution (Indoors) - from biomass fuel used for cooking could be poteniatl hazard and heating in poorly vented dwellings, which could be a risk factor that particularly affecting the women in developing countries. Occupational exposures – comprising organic and inorganic dusts, chemical agents and fumes, are considered risk factors for COPD. Air pollution (outdoors) conjointly bestowed to the lungs' total burden of

inhaled particles, although it materialize to have a relatively small effect in causing COPD. Genetic factors can influence, for example severe hereditary deficiency of alpha-1 antitrypsin (AATD) is present. Old age group and female gender are at highest risk of developing COPD. Socioeconomic status - there is strong evidence that the risk of developing COPD is indirectly related to socioeconomic status. However, whether this pattern could reflect the exposures to indoor and outdoor air pollutants, crowding, poor nutrition, infections, or other factors related to low socioeconomic status are under surveillance. Asthma and airway hyper-reactivity - asthma can contribute as a risk factor for the evolution of airflow limitation and COPD. Chronic bronchitis - can expand the frequency of total and severe exacerbations.

Pathological changes in COPD

Proximal airways (trachea, bronchi> 2 mm internal diameter)

- Inflammatorycell:IncreasedMacrophages,$CD8^+$(Cytotoxic)T lymphocytes, few neutrophils or eosinophils.
- Structural changes: Increased Goblet cells, enlarged submucosal glands (both leading to mucus hypersecretion), squamous metaplasia of epithelium.

Peripheral airways (bronchioles<2mm internal diameter)

- Inflammatory cells: IncreasedMacrophages,TLymphocytes (CD8+>$CD4^+$), Blymphocytes, lymphoid follicles, fibroblasts, few neutrophils or eosinophils.

- Structural changes: Airway wall thickening, peribronchial fibrosis, luminal inflammatory exudates, airway narrowing (obstructive bronchiolitis) increased inflammatory response and exudates correlated with disease severity.

Lung parenchyma (respiratory bronchioles and alveoli)

- Inflammatory cells: Increased Macrophages, $CD8^+$ T lymphocytes
- Structural Changes: Alveolar wall destruction, apoptosis of epithelial and endothelial cells.

Centrilobular emphysema: dilatation and destruction of respiratory bronchioles; most commonly seen in smokers. Panacinar emphysema: destruction of alveolar sacs as well as respiratory bronchioles; most commonly seen in alpha – 1 antitrypsin deficiency.

Pulmonary Vasculature

- Inflammatory cells: increased Macrophages,T lymphocytes.
- Structural Changes: Thickening of intima, endothelial cell dysfunction, smooth muscle cell proliferation leads to pulmonary hypertension.

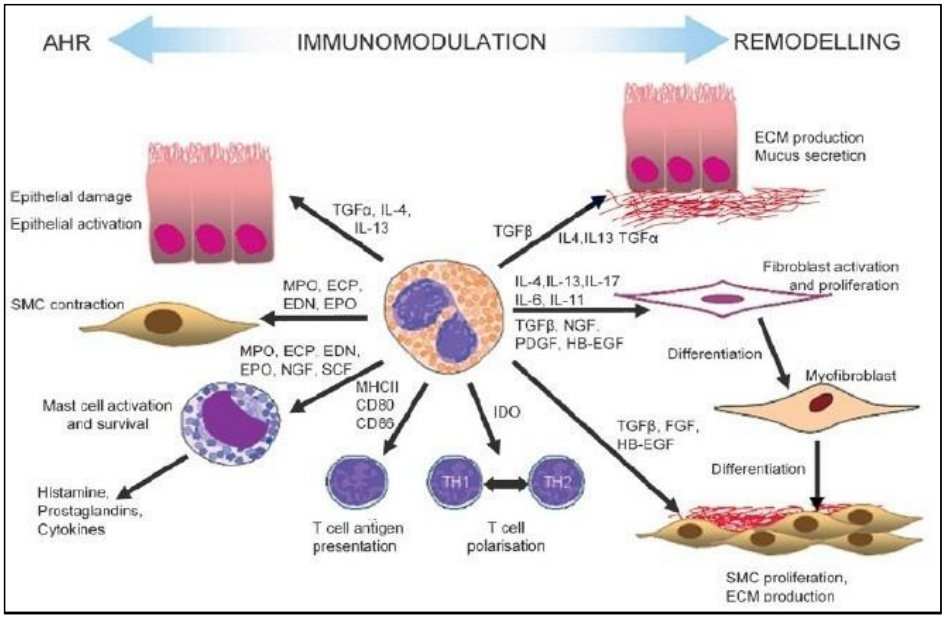


Fig 1: Mechanisms of Small Airway Obstruction in COPD

Small airways are the major sites of airflow limitation. Small airways show a variety of lesions narrowing their lumina, including goblet cell hyperplasia, mucosal and submuosalinflammatory cell, edema, peribronchial fibrosis, intra luminal mucus plugs and increased smooth muscle.[21]

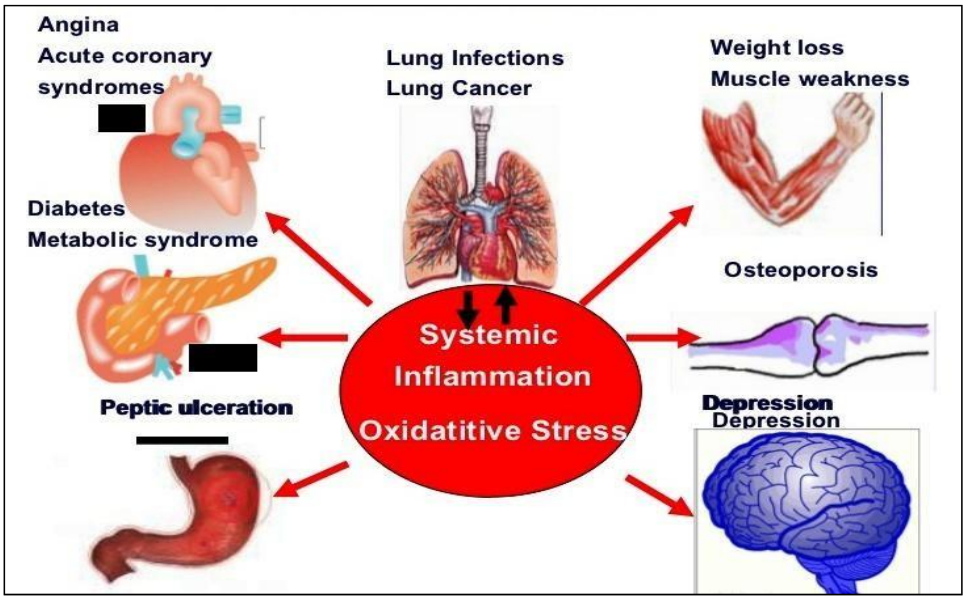


FIG 2: Systemic Effects of COPD Inflammation

CLASSIFICATION OF COPD

CLASSIFICATION OF AIRFLOW LIMITATION SEVERITY IN COPD

(Based on post-bronchodilator FEV1)

In patients with FEV1/FVC < 0.70

GOLD 1:	Mild	FEV1≥ 80% predicted
GOLD 2:	Moderate	50% ≤ FEV1< 80% predicted
GOLD 3:	Severe	30% ≤FEV1< 50% predicted
GOLD 4:	Very Severe	FEV1 < 30% Predicted

VISUAL EVOKED POTENTIALS (VEP):

Visual evoked potentials **(VEP),** the electrical voltage generated in response to visual stimuli, offers a useful measurement of optical pathway. Moreover, this analysiswill offer valuable evidence of optic nerve involvement in the early stages of ophthalmic illness.[13]

HISTORY OF VEP

VEPs initiated by scientific instrument flash were noticed in the early years of clinical encephalography in the 1930s.[13]Visual evoked potientials will usually be seen in the background encephalography recorded from the occipital scalp suceeding a flash of light.[13]Evoked potentials, whether or not auditory,

visual or somatosensory, are extracted from the encephalogrpahy by a simple program. This capability of extracting a signal from random noise is one of the ancient applications of computer technology.[14]Dawsonearlier demonstrated a signal-averaging device in 1951 and signal-averaging computers are available since the early 1960s.[14] The computer programs shave an outlined time frame of encephalography activities following a visual stimulus, which are repeated over and over adding the signals together. The random graph activity averages away, deed the visually evoked potential. Counting on the signal to noise ratio, associated evoked potential will be seen forming solely when only a few stimuli such as flashes of light.

ELECTRODE LOCATIONS ON THE SCALP

Visually evoked potentials evoked by flash stimuli can be recorded from numerous scalp spots in humans. Visual stimuli stimulate each of the primary visual cortices and secondary areas. Clinical VEPs areas are basically recorded from occipital scalp overlying the calcarine fissure. This is the nearest spot to primary visual cortex (Brodmann's area 17).[15]A common system for placing electrodes is the "10-20 International System" which is ascertained on measurements of head size.[15]Another set of locations is the "Queen Square system"[16]

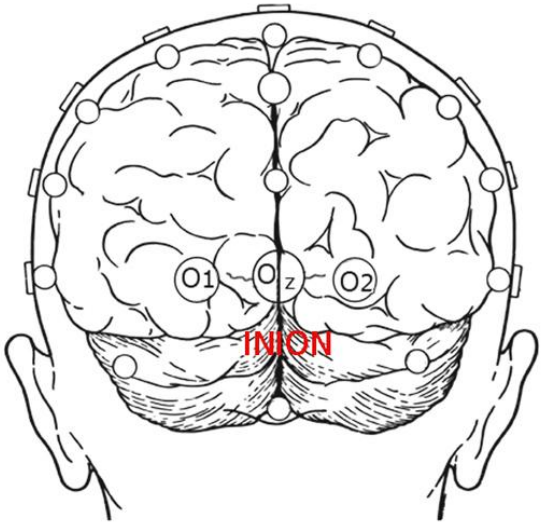


Fig 3:Occipital scalp electrode locations using 10-20 International System

The INION is the skull location at the position shown. The Nasion is on the bridge of the nose, between the eyes.[17]

Sources of visual evoked potentials

Most of primary visual cortex in humans is spotted on fissures, not on the cortical surface of the occipital pole. [18]

VEP Recording strategies

A reference conductor is usually placed on the earlobe, on the midplane on top of the head or on the forehead.[14]A ground conductor can be placed anywhere such as, mastoid, scalp or earlobe.The time taken to interpret the results usually takes between 200 and 500 milliseconds following onset of each visual stimulus.[14]When testing young infants, analysis time ought to be 300 msec

or longer as a result of the components of the VEPs may have long peak time during early maturation.[15] Most of the children and adults take the tested in a time frame of 250 msec or less.[15]The most frequently used amplifier bandpass frequency limits are 1 Hz and 100 Hz.[15]Pattern reversal is a desired stimulus as there are more inter-subject visual-evoked potentials reliability than with flash or pattern onset stimuli.[16]

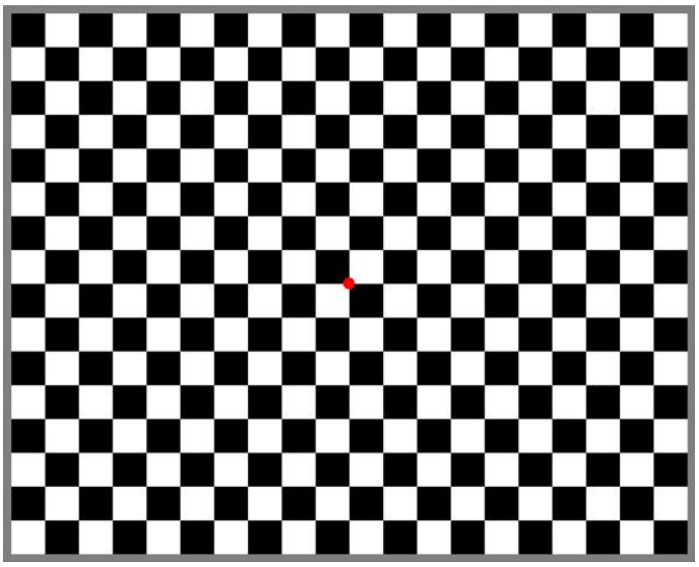


Fig 4. Checkerboard pattern with red fixation point.[16]

Representative normal pattern reversal VEP recorded from mid-occipital scalp using 50′ checkerboard pattern stimuli.[17]

VISUAL EVOKED POTENTIALS (VEP) AND COPD

The visual system in human being ishighly sensitive to hypoxia that its loss leads to incapacitate the life of a person. Visual Evoked Potential (VEP) is more sensitive than enchephalography and psychometry in detecting clinically silent and unrecognized atypical cases.[20] It is more sensitive and less costly when

compared to magnetic resonance imaging (MRI) in detecting the lesions affecting the visual pathway in front of the optic chiasma. The changes in the latency and amplitude of VEP waves, viz. N1 (N75), P1 (P100) and N2 (N145) reflect the degeneration in the quality ofsight.[20] Further, information on COPD with reference to cranial nerve involvement are limited in India which makes difficult to assess the magnitude of the problem nationwide.

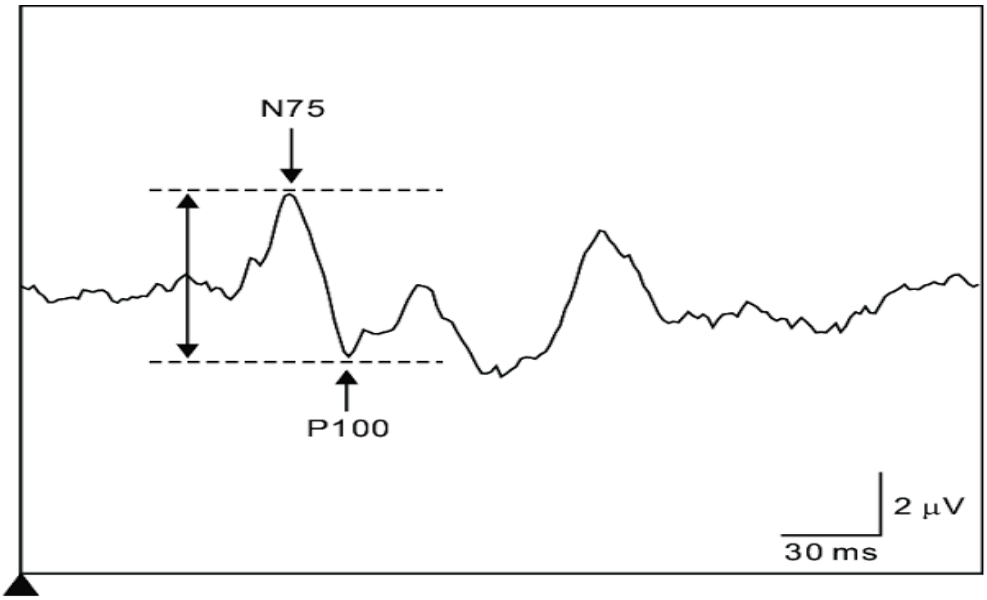


Fig 5. VEP in a normal individual

With these facts in mind, the present study was aimed at finding out the functional integrity of the visual pathway; determining the factors influencing the condition; and assessing the impact of **COPD on VEP**changes. The visual evoked potential (VEP) is essentially a comparatively large, positive polarity wave generated in the occipital cortex in response to visual stimulation. [21] It measures the conduction time of neuronal activity from the retina to the occipital cortex and is used clinically as a measure of the integrity and function of that pathway. [21]

The foremost structure examined was optic nerve. The visual evoked potential is of large enough voltage can be easily seen on a routine encephalography as an occipital waveform within the first one hundred and fifty ms after a single photic stimulus.[21] The standard visual evoked potential averages many such waveforms, time-locked to the stimulus. Foremost interest is the latency of the positive wave generated at a midline occipital EEG electrode, usually at approx 100 ms after stimulation, called the P100. This P100 peak is generally easy to recognize and measure.[21] VEP provide a qualitative and quantitative measure of the optical pathway, as they indicate the functional aspects of the optic nerve, optic chiasm and tracts, lateral geniculate bodies and geniculocalcarine projection to visual cortex. The normal visual evoked potential (VEP) reflects the functional integrity of the visual pathways from retinal to occipital striate area.[22] The VEP in an adult individual consists of three negative and three positive waves within a span of 350 msec after the application of the stimulus. [22] Out of the first three waves, N1 (N75), P1 (P100) and N2 (N145) of NPN complex, the latency and amplitude of P1 is clinically important Visual Eps

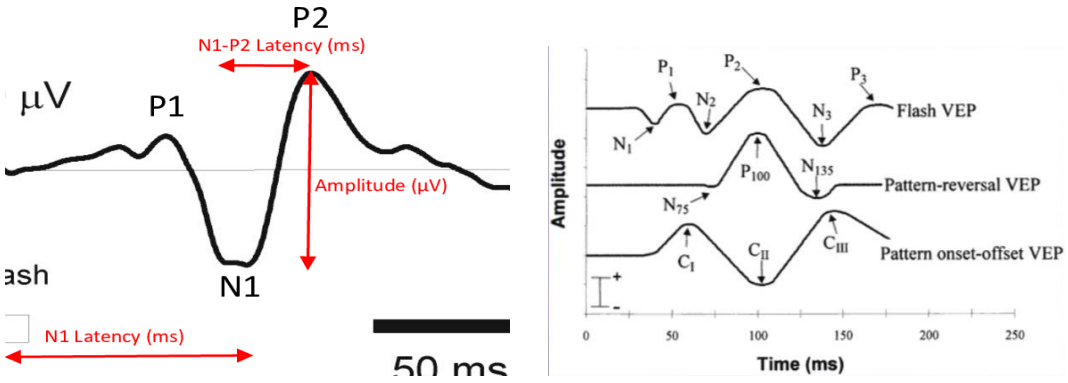


Fig 6. VEP Parameters and VEP types

Visual evoked potential records the electrical potentials that are generated using flashing light or changing motions in a monitor from the occipital cortex with surface electrodes and amplifications. [20] The most distinguished component in visual evoked potential examination is the positive peak wave (P100) that generates at the hundreth ms and is calculated as a 100-millisecond average in normal individuals.[20] The utomst detection demonstrating optic nerve demyelination is elongation of P100 latency.[20] An malformation in visual evoked potential response is seen in many diseases involving the optic nerve, such as multiple sclerosis, systemic lupus erythematosus, sarcoidosis, neurosyphilis, spinocerebellar degeneration, and vitamin B12deficiency.[23] Moreover studies comprising both peripheral and central involvement in patients with COPD at the same time are quite extinct. Visual receptors are influenced from hypoxia. The visual system in human including perceived brightness, acuity and dark adaptation is sensitive to hypoxia.[22] An increase in visual evoked potential latency clinically means a degradation in the quality of sight. Visual evoked potential values were assessed in various diseases.Visual evoked potential waveform is generated in striate, peristriateoccipital cortex and thalamocortical volleys. The VEP is a measure of physiologic function rather than primarily reflecting neuroanatomic lesions. [22]

VEPs are particularly helpful clinically in determining a physiologic abnormality where the neurologic and ophthalmologic examinations are normal. The primary measurement of interest clinically is the P100 latency after pattern

reversal stimulation.[22] Most subjects have much less variability, and the results are often remarkably precise and reproducible.

RECORDING THE VEP

TYPES OF VEP

FLASH VEP, PATTERN REVERSAL VEP, PATTERN ONSET/ OFFSET VEP

FLASH VEP –[24]

Diffusely flashing light stimulus that subtends a visual field of 20 degrees. A flashing light in a strobe sequence (flash VEPs) or even alternating intensities or luminances of light can be the stimuli.[24] They are used typically when a subject is unable to cooperate, for instance, neonates or patients with impaired mental status.

Fixation is not required, and the eyes may be closed. Flash VEPs assess the integrity of the visual system at least through the lateral geniculate nucleus and can help to determine whether the opticnerve is intact.[24] Flash potentials have greater latency variability and less sensitivity to visual conduction defects.

PATTERN REVERSAL VEP

The customary stimulus for Visual evoked potentials is a pattern of checkerboard where the squares alternate from black to white—the pattern reversal VEP (PRVEP). Dark squares become light and viceversa, in the absence

of any change in the overall luminance of the display. [21] Usually, the pattern is reversed 100 or 128 times at 1 to 2 Hz, and the results are then constituted by adding together and divinding it. Usually a repeattrial of averaged stimuli is also recorded.[21] Pattern reversal visual evoked potentails require maintaining visual fixation on the center of the pattern. The occipitalcortex is particularly sensitive to the perception of edges, and a sharp-bordered checkerboardproduces a strong and measurable response.[21]

PRVEPs are remarkably precise and constant fora given subject (who has no clinical change) and are very sensitive to dysfunction in thevisual conducting system.[24] Eyes are tested one at a time, with the other eye covered by a patch. Simultaneousbinocular testing could not localize an abnormality to one optic nerve or the other.

Check Size:[21]

One degree (or **1/360th of a circle**) is divided into **60 min (60′).** An individual checkerboard square usually has a visual angle of 30′, with 8° for the entire stimulus or video screen. Smaller checks are more sensitive in detecting visual system defects, but visual acuity can be a problem. Peripheral vision is stimulated better by larger checks.

Contrast:[21]

Contrast is the difference in luminance (or brightness) of the dark and light areas dividedby the sum of their luminances. Low contrast decreases the P100

amplitude and increases thelatency. Greater luminance decreases the P100 latency and increases the amplitude

Repetition Frequency:[21]

The pattern reversal rate is usually approximately two per second. Repetition faster than four per second can produce overlap (interference of one potential with the next) and distort the waveform; it may also increase the P100 latency. Slower repetition rates prolong testing and might produce a varied response because of diminished attention.

Averaging:[21]

The time-locked voltage signals are averaged over 100 to 200 trials, usually with aduration of 500 ms each. A signal sampling rate of 1000 samples in500 ms (2000 samplesper second) is high enough to avoid distortion of the waveform. VEPs are usually amplifiedby a factor of 50,000. VEPs have a relatively high signal-to-noise ratio, and a larger numberof trials is not required.Averaging the waveforms eliminates the variation unrelated to the stimulus, distinguishing the VEP from theEEG background. The two separate trials (each following averaging) should be nearly superimposable.

The recording computer usually includes artifact-rejecting programs; many provide an additional smoothing function.

Hardware:[21]

The recording EEG electrodes are placed 5 cm above the occipital midline (OZ) position and 5 cm to the right and left of this electrode—designated MO, RO, and LO. Usuallyfour channels are recorded. The first three channels consist of electrodes MO, RO, and LO,each referenced to a midfrontal electrode (MF). The fourth channel records MF referencedto an ear electrode and shows the active component of the MF electrode at approx 100 ms(with a negative polarity in contrast to that of the P100) and helps to explain some P100distortions. MO–MF is the primary channel used for most readings. Noncephalic references generally contain too much artifact to be useful.

Filters:

The **low-frequency filter** is usually set at **1 Hz** and the high-frequency filter at 100 to 300 Hz(the shape of the **standard P100** has a frequency of approx **15–20 Hz**). A high-frequencyfilter may cause an apparent increase in P100 latency.

Patient Factors:[24]

The patient should be alert and comfortable. No noise should accompany the stimulus; thiscould cause artifact. It is important to be sure that the stimulus can be seen clearly. Visual acuitymust be tested. Usual eyeglasses are used to optimize visual acuity. There should be no pharmacological pupillary dilation. One eye is tested at a time. The technologist should ascertain that the patient is

actually focusing on the center of the target throughout the test. This is particularly difficult for children and infants (note that fixation is not required with flash stimuli; they can beused even with comatose patients).It should be determined before testing whether the patient has a significant visual fieldabnormality. If so, the distribution of the P100 field may be distorted and the latency prolongedsomewhat. It may need to be recorded with more laterally placed electrodes. Low-amplitude VEPs may arise from inattention to the stimulus. The technologistshould ask whether the stimulus is seen clearly. If visual acuity is a problem, increasing check size may help. Technical problems often eliminate all potentials, so the finding of some normal and someabnormal potentials suggests that there is a true clinical deficit.

INTERPRETATION:

ANTERIOR PATHWAYS [21]

The primary role of VEPs is to assess the anterior visual pathway on each side. Monocular,full visual field stimulation restricts interpretation of an individual potential to the visualpathway anterior to the chiasm. A normal P100 latency indicates normal conduction from theretina to the occipital cortex. A delayed potential after stimulation of one eye implies a defect in conduction in the optic pathwayanterior to the chiasm on that side.If a P100 is absent, it must be determined whether this is caused by technical factors before it can be considered abnormal. If not a technical artifact, complete absence of the VEP is generally caused by severeocularor optic nerve disease, such as complete

blindness in an eye or interruption of the optic nerve.On both the eyes delay in latencies after stimulation of each eye separately could be caused by bilateral optic nerve lesions, but they could also be caused by chiasmatic or widespread posteriorlesions.Without hemifield stimulation, posteriorvisual pathways cannot be evaluated.Most abnormally prolonged P100 latencies are caused by disease affecting the optic nerve, especially in demyelinating disorders, compression, and other optic neuropathies. In clinical practice,VEPs are used primarily to detect lesions when they are not easily demonstrable by clinicalexamination or indicated by history.

POSTERIOR PATHWAYS:[21]

Full-field stimulation of either eye activates both occipital cortices. The occipital cortexon each side generates a response large enough to be recorded over a wide area, includingthe opposite occipital region. Since cortical activity in either occipital lobe produces a P100 wave with a standard latency at the occipital midline, and because the signals from each occipital lobe cannot be separated, full-field stimulation can produce a normal VEP even ifthere is a large lesion in posterior optic pathways.Hemifield stimulation is required to assess posterior pathways. This is more difficult technically. The patient focuses on the center of the screen. A randomization program may be used to present standardized stimuli alternately to either visual fieldat random times to maintain fixation on the central target. Stimulation is typically with 35′ checks, but halffield testing often uses larger check sizes. With hemifield stimulation there

may be no VEP produced or recordable in the midline. Lesions in the chiasm itself can interfere with crossing of conduction from the retina to theopposite hemisphere (from the nasal retina, i.e., temporal visual field).

Retrochiasmal lesions should produce VEPs of prolonged latency in the affected field afterstimulation of each eye separately. An absent VEP or prolonged VEP latency after hemifield stimulation of one eye alone suggests a partial lesion in that eye or the optic nerve on the side affected. In summary, it is only very large posterior pathway lesions that disrupt the VEP significantly.

CLINICAL APPLICATIONS:

The most common and important VEP abnormality is a delay in the P100 latency after full-field stimulation of a single eye. This indicates a defect in the optic conducting system anterior to the chiasm on that side—usually optic nerve disease. Chiasmal and moreposterior lesions, or widespread brain dysfunction can cause bilateral delay of the P100, usually with similar delays on testing of each eye. Optic neuropathies can occur on both sides. Bilateral demyelinating lesions typically produce asymmetric and abnormal latencies on the two sides. Metabolic and degenerative disorders typically cause delays that are similar on the two sides. Also, P100 latency prolongationscaused by retrochiasmal lesions are usually similar after stimulation of each eye separately.

Optic Neuritis:

Optic neuritis is the primary concerns in the assessment of the optic nerve. Roughly 90% of patients with a definite history of optic neuritis have delayed P100s. Normal Visual evoked potential makes the diagnosis of optic neuritis very impropable, especially with severe clinical symptoms. An abnormal visual evoked potential in an eye that appears normal clinically suggests an old optic neuritis with clinical recovery.

Multiple Sclerosis:

A clinically silent optic neuropathy found by VEP can be an aid in making the diagnosisof MS (although the diagnosis is often made now with greater reliance on MRI scans). In MS with optic nerve involvement, there is typically a marked delay (e.g., 20–30 ms),but not complete absence of the P100.They are abnormal in approx90% of patients with definite MS and in up to 25% of patients with normal MRI results whoare being evaluated for the possibility of MS. For study of the optic nerve alone, VEPshave sensitivity comparable to that of MRI.

Traumatic Visual Loss:

After blindness caused by trauma, a normal VEP offers a relatively good prognosis forrecovery of vision, and an absent VEP is a very poor prognostic sign.

High-Pressure States:

Pseudotumor cerebri usually does not affect the VEP substantially, although the high pressure can cause visual loss. In pseudotumor, VEP abnormalities may precede visual loss, butthis is not reliable, and VEP changes may occur too late to be of clinical aid. Similarly, a mildlatency change may be seen with glaucoma.

Neurodegenerative Diseases:

Alzheimer's disease typically leaves the PRVEP unaffected. A minority of Parkinson disease patients has prolonged P100s, even without evident neuropathological changes in theoptic conducting system. Leukodystrophies, such as adrenoleukodystrophy, delay the P100. Most patients with Friedreich's ataxia have bilateral but relatively mild P100 delays. Abnormal VEPs are relatively common in spinocerebellar degenerations. Leber's hereditary optic neuropathy increases the P100 latency substantially, but alsoreduces the amplitude and eventually abolishes the VEP.

Operative Monitoring:

PRVEPs require focus on a target and are, thus, not feasible during general anesthesia orcoma. As noted earlier, flash VEPs may help to establish the integrity of the optic nerve or itssevere disruption by trauma. They may indicate problems resulting from surgery (e.g., for anoptic glioma) or other masses near the optic nerve or chiasm. Many patients with significantdisease in these areas

may have retained normal VEPs, however, so they cannot be used asscreening tests.

Chiasmal Lesions: Chiasmal lesions often produce dramatic and unusual visual field defects, best detectedclinically. They may produce bilaterally delayed P100 latencies, and, at times, hemifield stimulation may be abnormal. In most cases, however, the visual field examination and MRI scanare of greater use in detecting chiasm area lesions.

Retrochiasmal Lesions: Retrochiasmal lesions are rarely detectable by full-field stimulation. Bilateral posteriorpathway lesions may delay the P100 bilaterally, typically in a symmetric fashion. Hemifieldstimulation is necessary to detect unilateral posterior pathway lesions.

Ocular Problems: Ocular problems diminishing luminance at the retina tend to lower P100 amplitude, butany increase in VEP latency should be minimal. This can occur with eye closure, ptosis,corneal opacities, cataracts, severe miosis, or hemorrhages and foreign bodies in the corneaor liquid chambers of the eye.

Psychiatric Blindness: Psychiatric blindness can be evaluated to some degree with VEPs. Most activities leadingto an intentional diminution of VEP recording (such as poor fixation or eye closure) can beseen by the technologist, and they typically will not affect latency anyway.

HISTORY OF VISUAL FIELD:[18]

MD Ptolemy in B.C. 150, used some kind of perimetric device to estimate the extend of visual field. Primary clinical investigation of visual field defect – Hippocrates in 5th century, hemianopic field defect.Ultimately in 1604 Kepler described the principle of sight in term of an inverted retinal image.Maroiotte in 1666, uncovered the physiological blind spot.Young in 1801, stated that the normal extension of visual field of an eye.Von Graefe spotted out blind spot, central scotomas, construction of isopter.Until 1869, Foerester discovered arc perimeter, till visual field plotted on flat surface.Bjerrum in 1880, framed Tangent screen.Sloan in 1939, reported static perimetry.Goldmann in 1945described perimeter.Tubinger in 1960 – manual testing of both static and kinetic parimeter.

VISUAL FIELD TESTING METHODS/TOOLS

CENTRAL:

- Amsler grid: 20 degree
- Tangent (Bjerrum screen) : 30 degree
- Goldmann
- Automated (Octopus/ Humphrey): 30 degree

PERIPHERAL:

- Confrontation
- Goldmann
- Automated 90 degree programme

SIGNIFICANCE OF VISUAL FIELD TESTING:

Find out the extent of visual field.To diagnose and detect diseases as well as extent of damage caused in VF by the disease.To locate the possible lesion in neurological disorder.To find out the progression of diseases.

VISUAL FIELDS IN COPD PATIENTS:[25]

Visual field assessment is vital diagnostic tools in neuro ophthalmologic disorders.Perimetryanalysis may show few involved areas in the visual field which even though the patients central vision has been conserved.Perimetry may be convenient in determiningthe ischemic and hypoxic retinal disorders.The retinal ganglion cells are sensitive to mild hypoxemiain chronic obstructive pulmonary diseases and observed that ganglion cell function is restricted with decreased arterial blood oxygen.The neural function during hypoxia is influenced as a result of metabolic changes and cannot be compensated properly by vascular regulation of inner retina.The position of reactive oxygen species and related vascular endothelium damage in most respiratory events is because of desaturation and reoxygenation sequence.

VISUAL FIELD TESTING USES [26]:

We need to find out the extent of visual field inorder to diagnose and look for diseases as well as extent of damage caused by visual field by the disease. We need to locate possible lesion in the neurological disorder to find out the progression of diseases.

OCTOPUS PERIMETRY

Fig 7. OCTOPUS PERIMETRY

RELATIONSHIP AMONG OCTOPUS VISUAL FIELD REPRESENTATIONS[26]

If we see most of the visual field representations on octopus perimeters are based on the following three key representations.

a) Values

b) Comparisons

c) Corrected comparisons

VALUES[26]

It actually represents a set of raw data of visual field testing and is a 2-D numerical map of a patient's hill of vision.Sensitivity thresholds are displayed in dB and absolute defects are displayed using a"■"symbol.

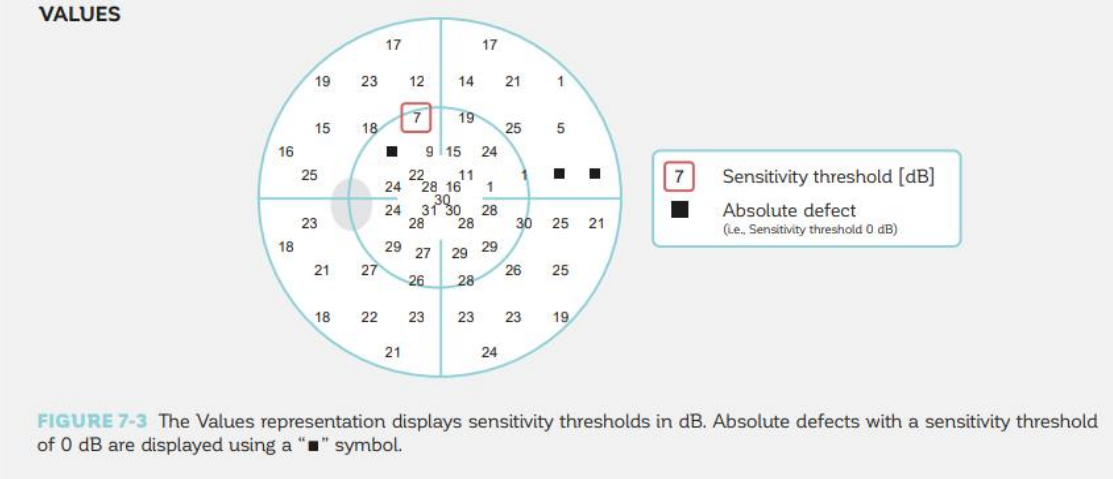


FIGURE 7-3 The Values representation displays sensitivity thresholds in dB. Absolute defects with a sensitivity threshold of 0 dB are displayed using a "■" symbol.

Fig 8. Values Representation in visual filed

GRAYSCALE OF VALUES

Each color represents sensitivity thresholds within a range of 5 dB. White represents sensitivities of 36 to40 dB, while black represents the other end of the scale, showing sensitivity thresholds of 0 dB.

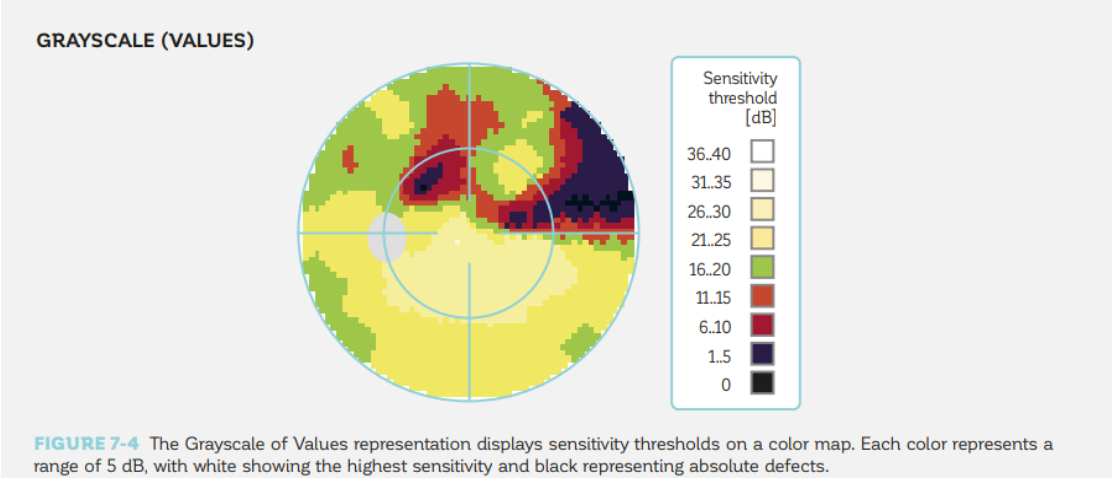


FIGURE 7-4 The Grayscale of Values representation displays sensitivity thresholds on a color map. Each color represents a range of 5 dB, with white showing the highest sensitivity and black representing absolute defects.

Fig 9. Grayscale of values field

COMPARISON[26]

The Comparisons representation allows direct assessment of the shape and magnitude of diseaserelated change in sensitivity. The Comparisons represe

ntation is defined as the individual deviationfrom the average normal visual field of the respective age group.

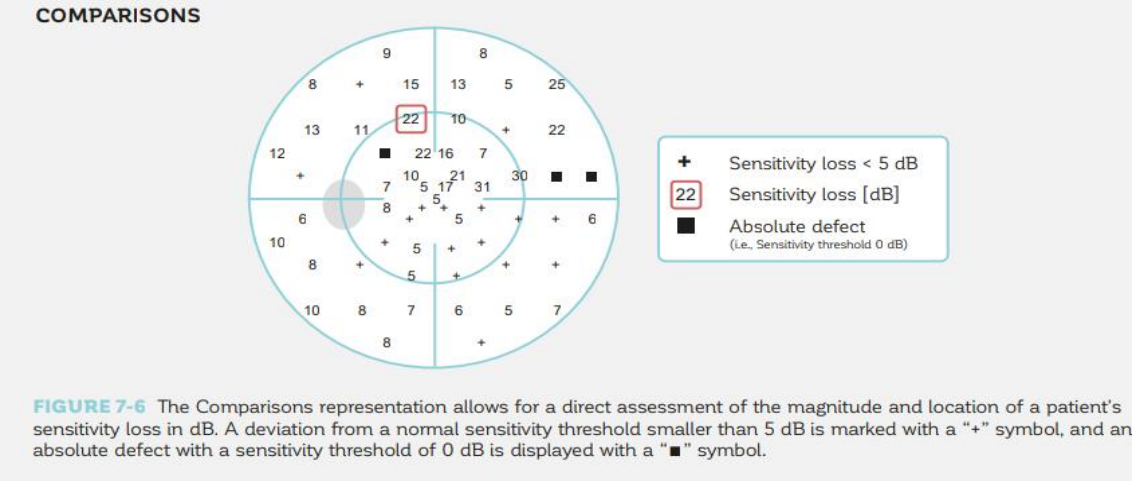


FIGURE 7-6 The Comparisons representation allows for a direct assessment of the magnitude and location of a patient's sensitivity loss in dB. A deviation from a normal sensitivity threshold smaller than 5 dB is marked with a "+" symbol, and an absolute defect with a sensitivity threshold of 0 dB is displayed with a "■" symbol.

Fig 10. Comparisons representation in visual field

GRAYSCALE OF COMPARISONS

The Grayscale of Comparisons is used clinically to intuitively assess the magnit ude and shape of sensitivity loss.

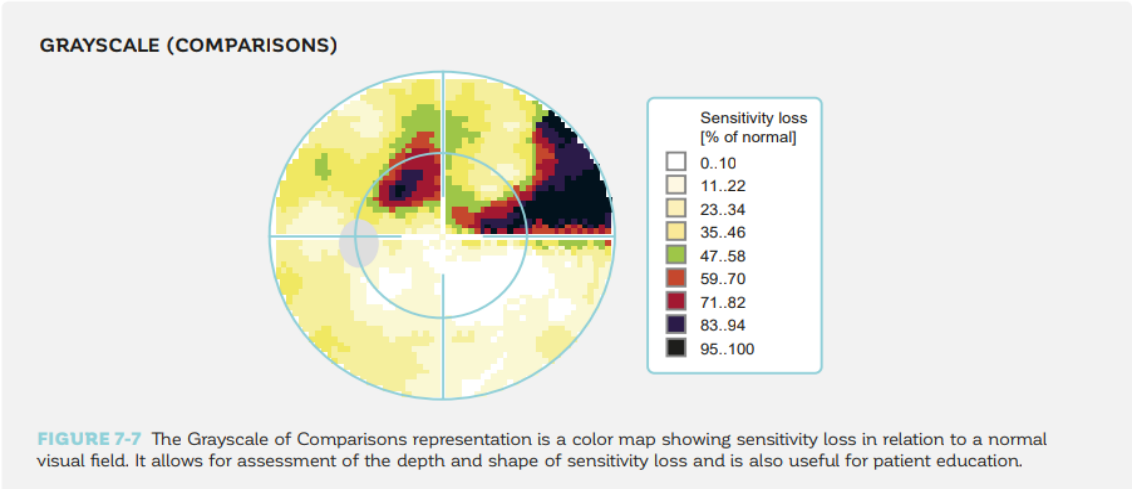


FIGURE 7-7 The Grayscale of Comparisons representation is a color map showing sensitivity loss in relation to a normal visual field. It allows for assessment of the depth and shape of sensitivity loss and is also useful for patient education.

Fig 11. Grayscale of Comparisons representation in visual field

3

PROBABILITIES[26]

The Probabilities representation is used clinically to distinguish between normal and abnormal visual field locations.

(p > 5%): there is a high probability that a person with a normal visual field would show this sensitivity loss.

(p < 5%): there is a smaller than5% (and larger than 2%) chance that a person with a normal visual field would show this sensitivity loss.

(p < 2%): there is a smaller than2% (and larger than 1%) chance that a person with a normal visual field would show this sensitivity loss.

(p < 1%): there is a smaller than 1% (and larger than 0.5%) chance that a person with a normal visual field would show this sensitivity loss.

(p < 0.5%): there isa smallerthan 0.5% chance that a person with a normal visual field would show this sensitivity loss.

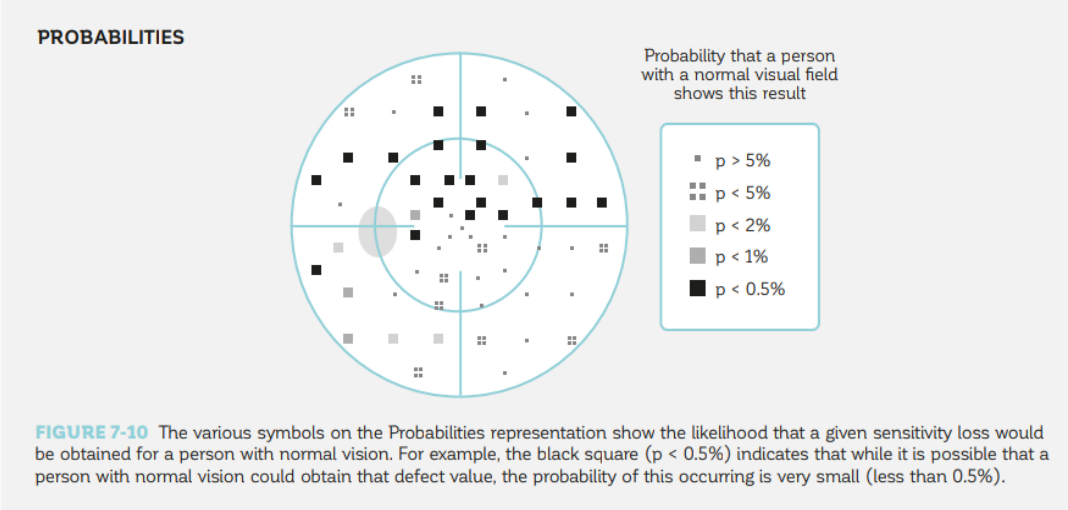


FIGURE 7-10 The various symbols on the Probabilities representation show the likelihood that a given sensitivity loss would be obtained for a person with normal vision. For example, the black square (p < 0.5%) indicates that while it is possible that a person with normal vision could obtain that defect value, the probability of this occurring is very small (less than 0.5%).

Fig 12. Probabilities representation in visual field

DEFECT CURVE[26]

The graphical representation of the defect curve also called as Bebie curve alerts the clinician to the presence of diffuce defects. It gives as a summary of the VF and it makes it possible to distinguish between diffuse and local defects at a glance.

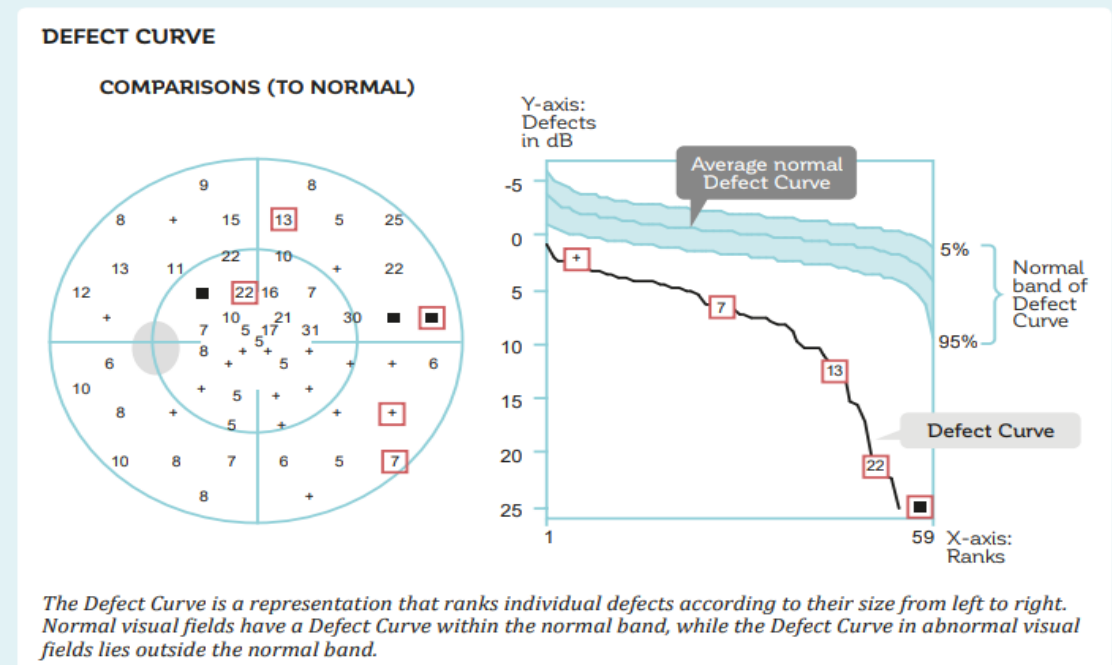


Fig 13. Defect curve in visual field

CLUSTER ANALYSIS

Cluster Analysis has been designed specifically for glaucoma and is very sen sitive to detection of subtle glaucomatous defects. It capitalizes on the fact that typical glaucomatous defects consist of cluster of adjacent defective visual field locations that correspond to path followed by the retinal nerve fiber bundles in the retina.

POLAR ANALYSIS[26]

It is a specific analysis especially designed for glaucoma. It provides adequate information about the structural defects that to be expected at the optic disc by displaying VF resulting in structural coordinates.

CORRECTED COMPARISONS

It is useful to analyze localized visual field defects independently of diffusedefects.

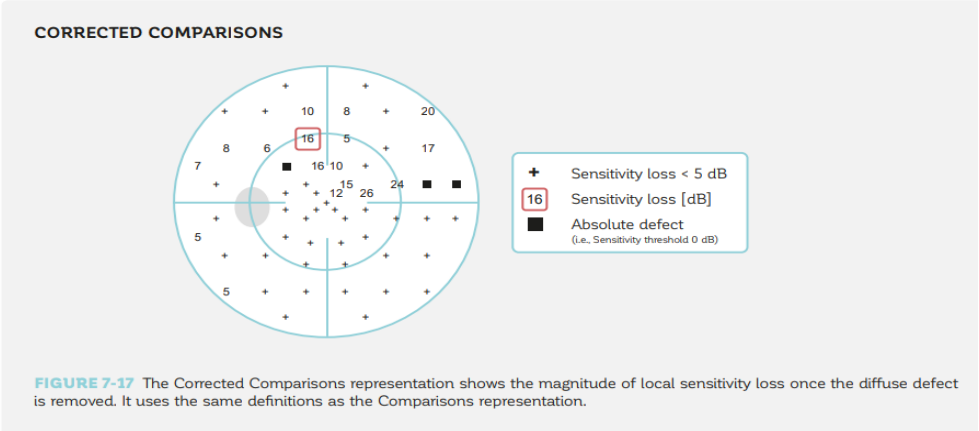


Fig 14. Corrected Comparisons of visual field

GRAYSCALE OF CORRECTED COMPARISONS

It displays sensitivity loss as a color map without diffuse defect.

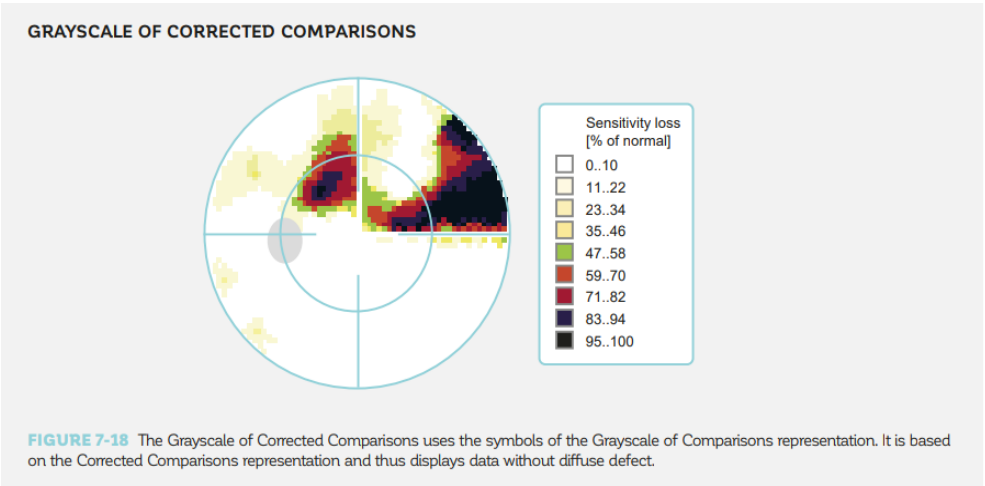


Fig 15. Grayscale of corrected comparisons

CORRECTED PROBABILITIES [26]

The Corrected Probabilities representation is very similar to the Probabilities representation and shows the probability that a person with normal visual field shows this corrected sensitivity loss at various significance levels.

GLOBAL INDICES

It provides a summary of the status of the visual field, 2) They are useful to objectively assess the severity of visual field loss and 3) It is also helpful in the evaluation of change over time.

MEAN SENSITIVITY (MS)

The Mean Sensitivity (MS) is the arithmetic mean of the sensitivity thresholds displayed in the Values representation. It represents a patient's average sensitivity to light with respect to the locations that are tested. MS is based on the Values and its diagnostic value is therefore limited because it is dependent on patient age and on the spatial distribution of the test locations.

MEAN DEFECT (MD)

The Mean Defect (MD) is the arithmetic mean of the sensitivity loss displayed in the Comparisons representation. It represents the average visual field loss of a patient derived from the locations that are tested and is thus often used to assess visual field severity.

SQUARE ROOT OF LOSS VARIANCE (sLV)

The square root of Loss Variance (sLV) represents the standard deviation of the individual defects at all visual field locations and provides a measure of variability across the visual field.

PART-II

AIM AND OBJECTIVES

AIM OF THE STUDY:

The main aim of the study was

A. To study the **visual evoked potential** (VEP) changes in **chronic obstructive pulmonary disease (COPD)** patients with no clinical visual impairment.

B. To study the **visual field defects** in **chronic obstructive pulmonary disease (COPD)** patients with no clinical visual impairment.

OBJECTIVES OF THE STUDY

- To study the association between **severity of chronic obstructive pulmonary disease (COPD)**and **VEP changes.**
- To study the association between **severity of chronic obstructive pulmonary disease (COPD)** and visual **field changes**.

MATERIALS AND METHODS

Study design: Cross sectional hospital based observational study

Setting: Department of Ophthalmology in Government Stanley Medical College Hospital.

Study participants: The COPD patients attending the chest medicine OPD in Government Stanley Medical College Hospital.

Source of data:

COPD patients presented to the chest medicine OPD in Government Stanley Medical College Hospital were included and graded according to GOLD guidelines based on spirometry indices.

Inclusion Criteria:

Patients diagnosed as COPD and classified by Global Initiative for Chronic Obstructive Lung Disease (GOLD)criteria.

Exclusion criteria:

- Patients with diabetes Mellitus, hypertension, tuberculosis, bronchial Asthma, already
- Preexisting Optic Neuropathy of other causes, patients with disc anomalies, hereditary disorders involving peripheral nerves and history of intake of any neurotoxic drugs.

Sample size: Sample size was calculated to be 96 using Master software version 2.0 taking into consideration, as 91.7 % from previous study [10] with 5% absolute precision, 95%confidence interval and 10% non-response rate.

The confidence interval or margin of error is estimated at +/-5

Assuming p%=35.8% and q%=64.2%

$n = p\% \times q\% \times [z/e\%]^2$

$n = 35.8 \times 64.2 \times [1.96/5]^2$

$n = 96$

Study duration:

Jan 2018 to Jan 2019

The study was carried out at the Department of Ophthalmology, Govt. Medical College with assistance from the Department of Chest medicine and Department of Neuromedicine, Govt. Stanley Medical College and Hospital in accordance with the ethical committee guidelines.

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

Sampling Method

Using simple random sampling.

Brief Procedure:

PULMONARY FUNCTION TEST: Were performed using computerized spirometer (INTEX 17) digital suga monitor model No:17 – 173SB, spiroexcel – MEDICAID. The following indices were recorded FVC, FEV1&FEV1/FVC.

PULMONARY FUNCTION TEST (PFT):

Recorded using computerized spirometry. These days computerized multifunctional spirometers are available which allow highquality made virtually breath. These spirometers display to highresolution graphic display as well as the predicted curves. The generated reports may be seen on the display or can be printed. All pulmonary function test parameters with actual Predicted and percentage predicted values, as well as normal range with the option of interpretation and lung age can also be obtained. Moreover, all tests performed are presented with both the selected test highlighted and the percentage variation frombest. The subject is made to sit comfortable in a stool facing the spirometer, nose is clipped and the mouthpiece is inserted between the teeth and the lips.The subject is then instructed to breathe in with maximum effort form the end of resting expiration and subsequently to breathe out completely with maximum effort. He is beforehand instructed not to breathe in while he is breathing out. At least three such forced vital capacity (FVC) curves are obtained and the maximum (best performance) of three values is taken for calculation purposes,

and other parameters FEV1, FEV1/FVC etc. will be displayed with graphic display.VEPs were recorded in a dark room 100cm away from the monitor. A chess board pattern reversal method on a 12 inch screen. Stimulation frequency at speed of 2Hz.

VEP Was carried out using digital four channel polygraph intex monitor, MRI GNAINI – 15”/17’ INSTUMENT MODEL IT - 173SB.

STIMULUS: The standard stimulus for VEPs is a checker board pattern in which the squares alternate from black to white – the pattern reversal VEP (PRVEP) Dark square become light and vice versa, without a change in the overall Luminance of the display. Typically, the pattern is reversed 100 or 128 times at 1 to 2 Hz, and the results are then averaged. Usually a repeat trial of averaged stimuli & also recorded.

Check size: 8^{o} x 8^{o} for the entire stimulus or video screen

Contrast: Contrast is the difference in luminance (or brightness) of the dark and light areas divide by the sum of their luminances.

Repetition frequency: The pattern reversal rate is usually two per second.

Averaging: Voltage signals are averaged over 100 trials, usually with a duration of 500-ms each. VEPs have a relatively high signal to noise ratio, and a larger number of trails is not required.

Filter: The Low – frequency filter is usually set at 1 Hz and the high frequency filter at 100 – 300 Hz. (The shape of the standard P100 has a frequency of approx 15 - 20Hz)

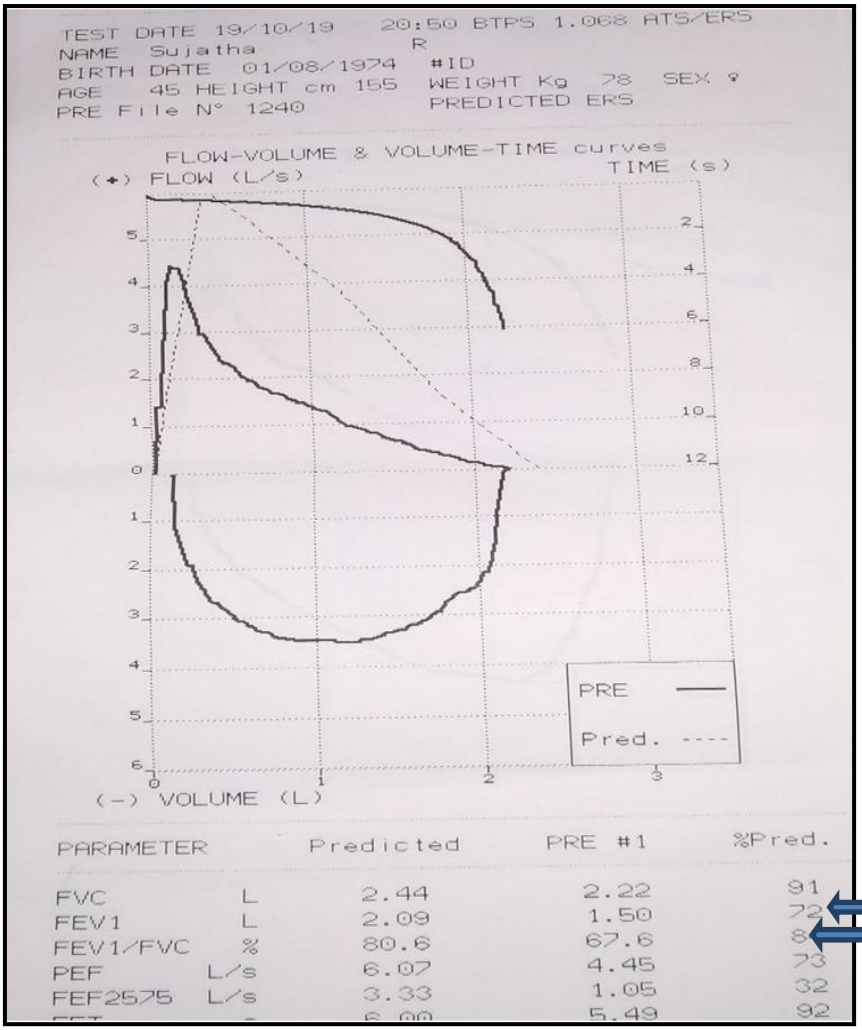
Sites where electrodes place were cleaned with 75% alcohol. Electrodes were smeared with conductive paste, recording electrode was positioned 1.5cm above the occipital bone reference on middle forehead, ground electrode the vertex.

VISUAL FIELD ANALYSISwas done using Haag- Streit Octopus 300 perimeter. During the examination, the patient concentrates on the fixation target displayed in centre of field of view. At various locations in the field of view, stimuli (flashes of light) of certain duration and intensity were shown. The patient was asked to press on the patient response button if the stimulus is perceived. Duration of the examination can range from roughly 3-15 min. Global indices (mean sensitivity and mean defect) were evaluated.

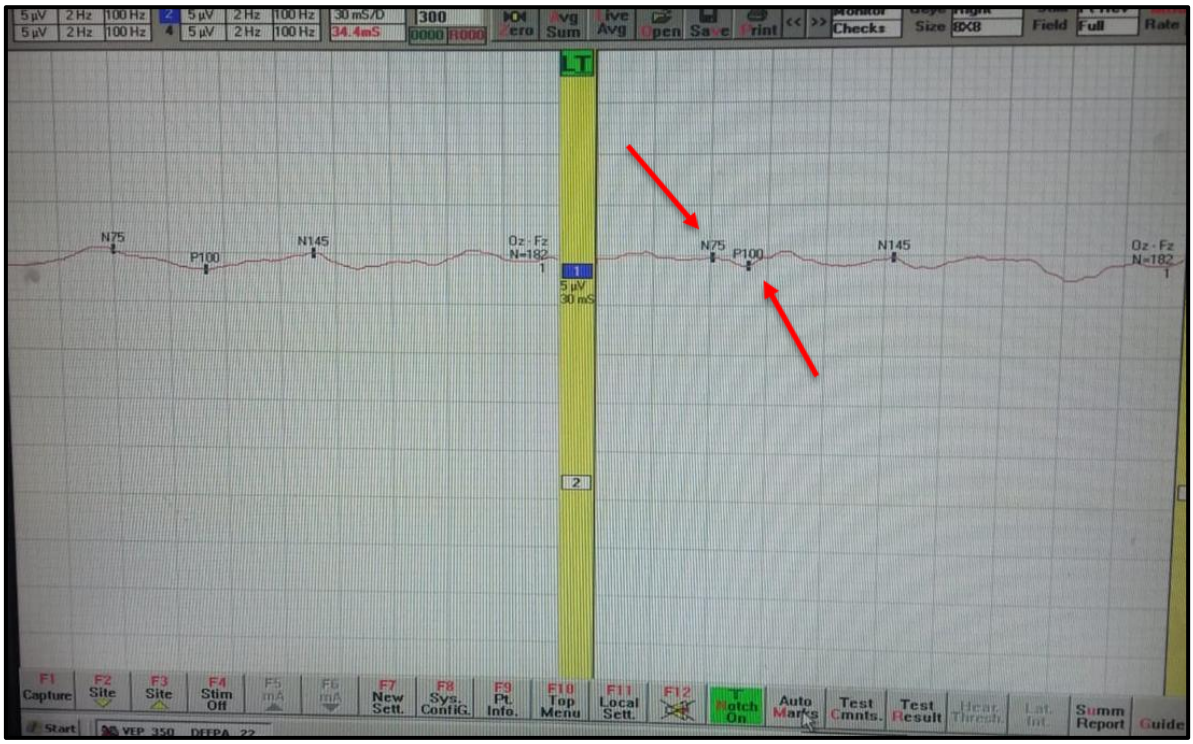
Statistical analysis:

Data were entered in the software Epi info version 7.2.1.0 and analysed using software SPSS version 24.0. Description of categorical variables was mentioned in percentage. Data related to continuous variable like age, were described in terms of mean and standard deviation. Pearson's (linear) correlation coefficient was used to find the degree of association. One-way ANOVA was used to find whether there are any statistically significant differences between the means of two or more independent (unrelated) groups. Scatter plot was

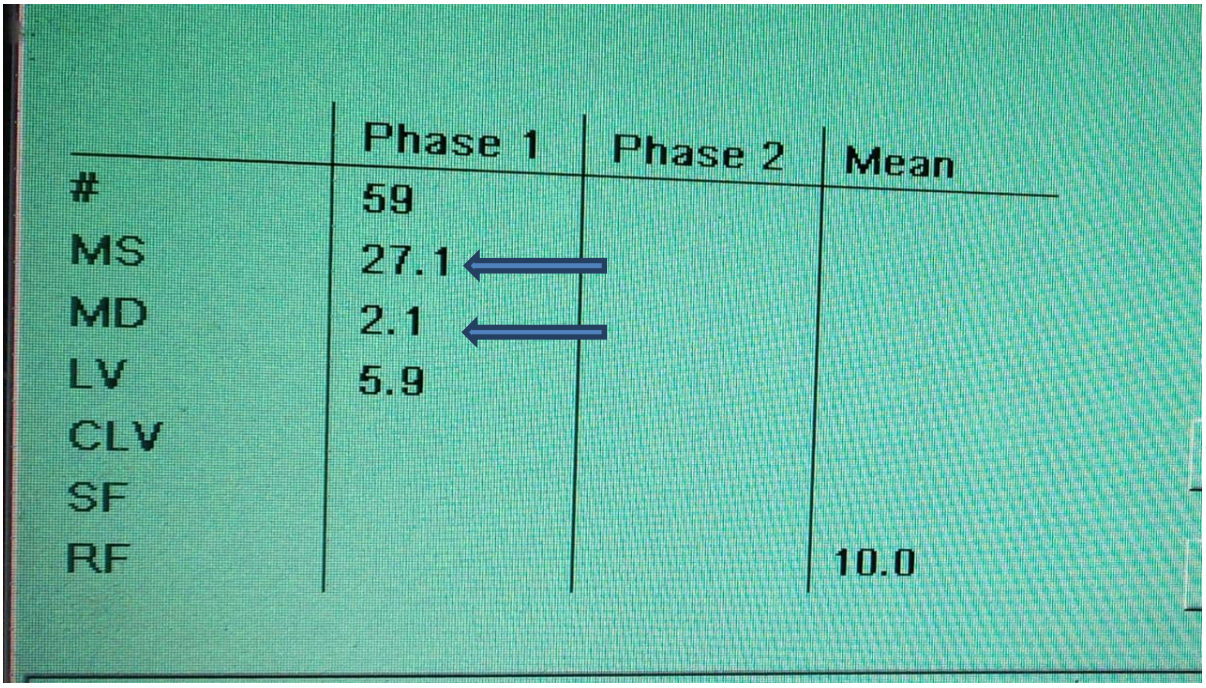
constructed to show the strength of correlation across VEP and FEV1, FEV1/FVC. All statistical test will be two-tailed and statistical significance will be set at P <0.05.



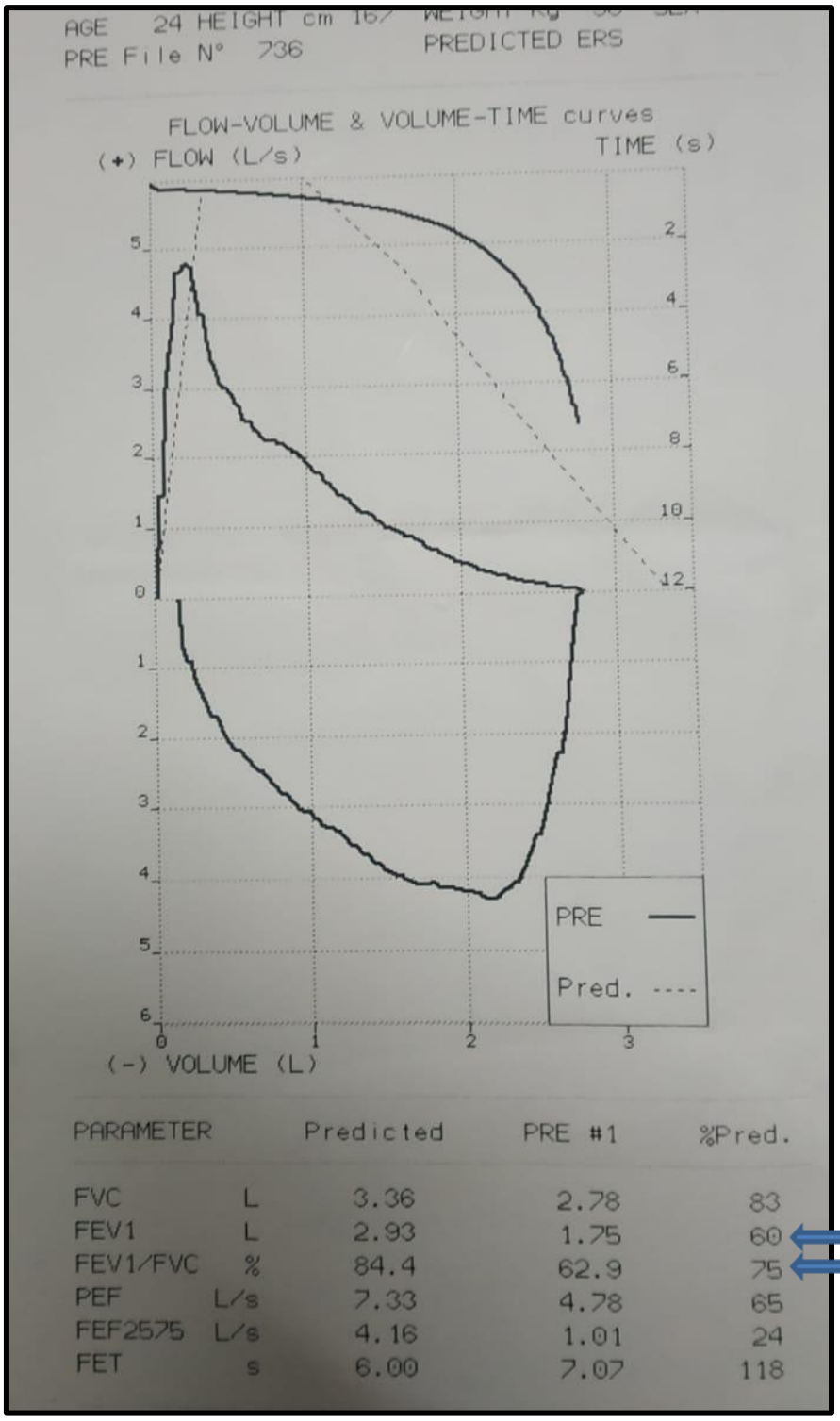
Picture 1 : Spirometry indices of patient showing FEV1 : 72%, FEV1/FVC : 80%



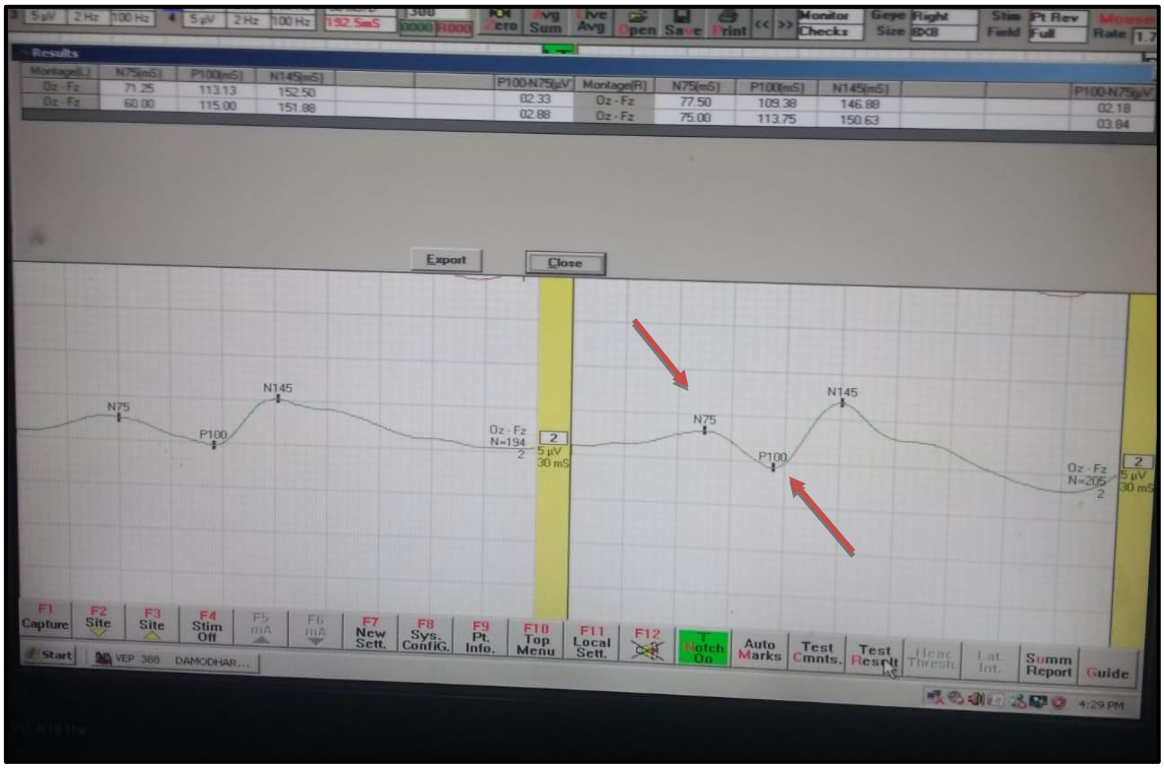
Picture 2 :VEP of patient showing N75 (ms) : 76.06, P100 (ms) : 104.64, P100 (µv) : 5.67



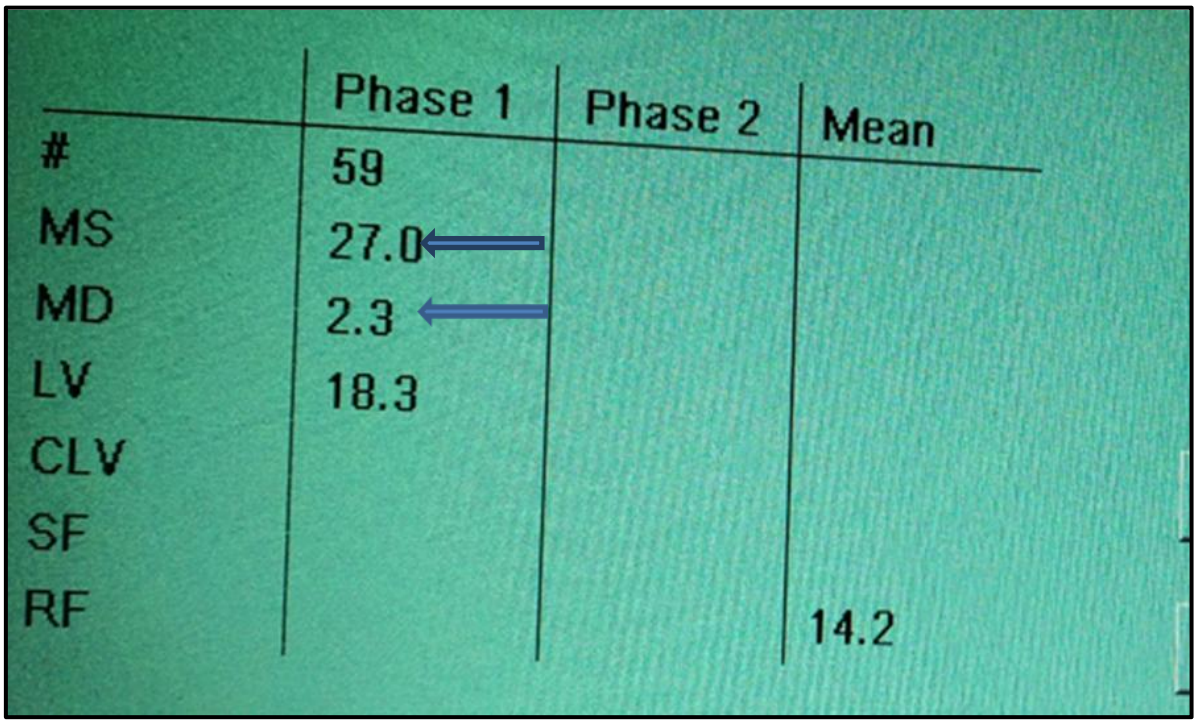
Picture 3 :Visual field of patient with MS : 27.1 dB and MD : 2.1dB



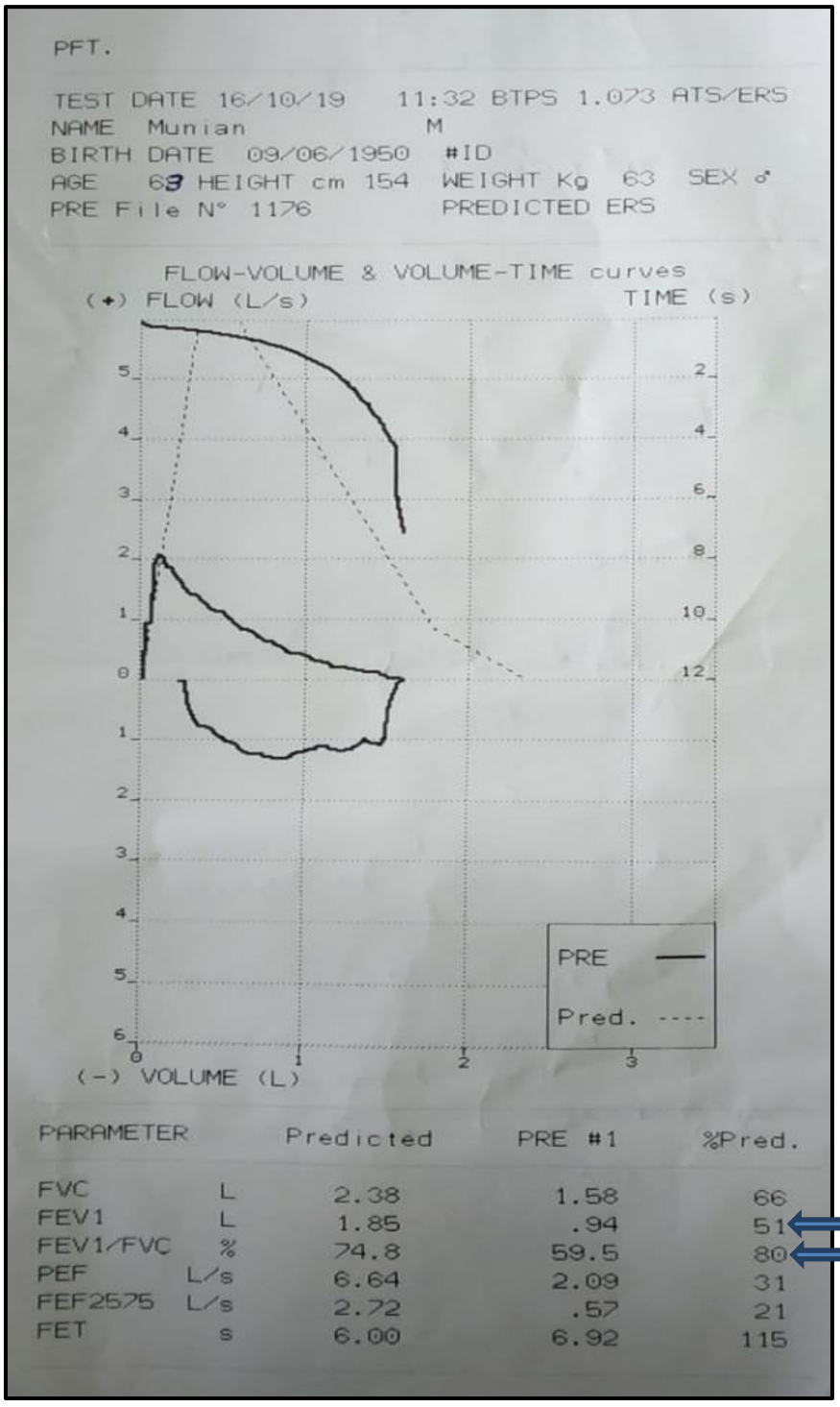
Picture 4 :Spirometry indices of patient showing FEV1 :60%, FEV1/FVC :75%



Picture 5 : VEP of a patient showing N75 (ms) : 77.50, P100 (ms) : 113.75, P100 (μV) : 3.04



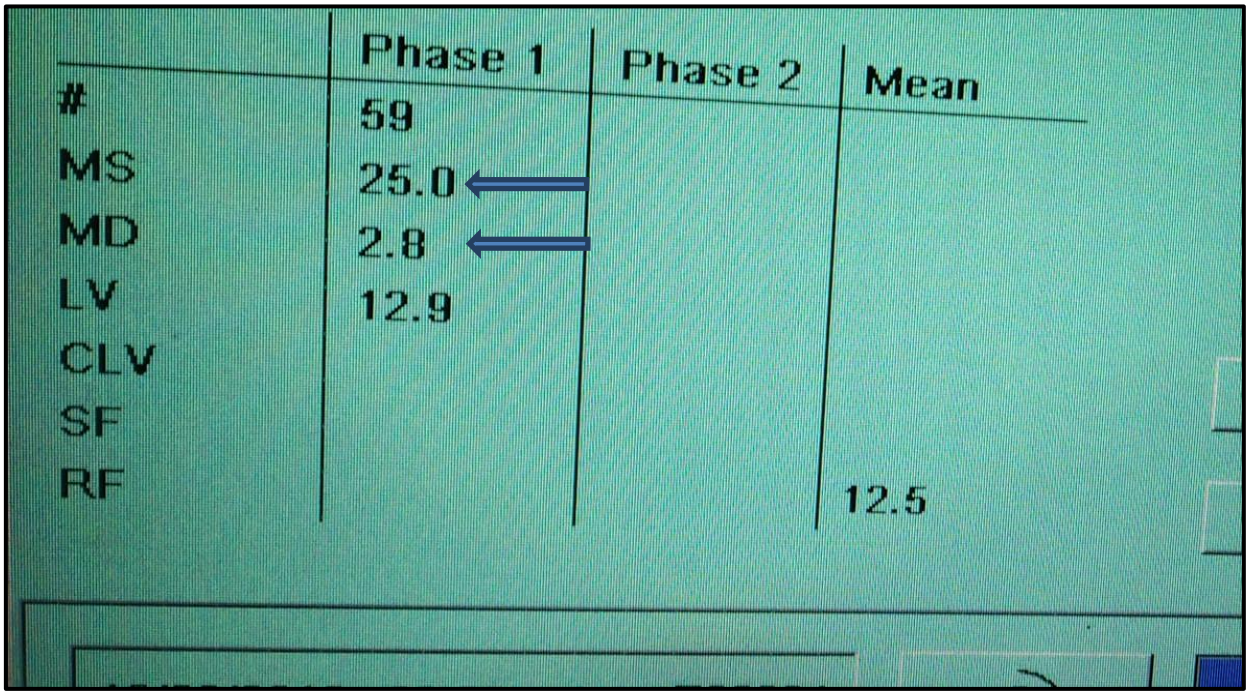
Picture 6 : Visual field of patient with MS : 27.0 dB and MD : 2.3dB



Picture 7 :Spirometry indices of patient showing FEV1 : 51%,

FEV1/FVC:80%

Picture 8 : VEP of a patient showing N75 (ms) : 76.77, P100 (ms) 114.38, P100 (µV) : 3.41



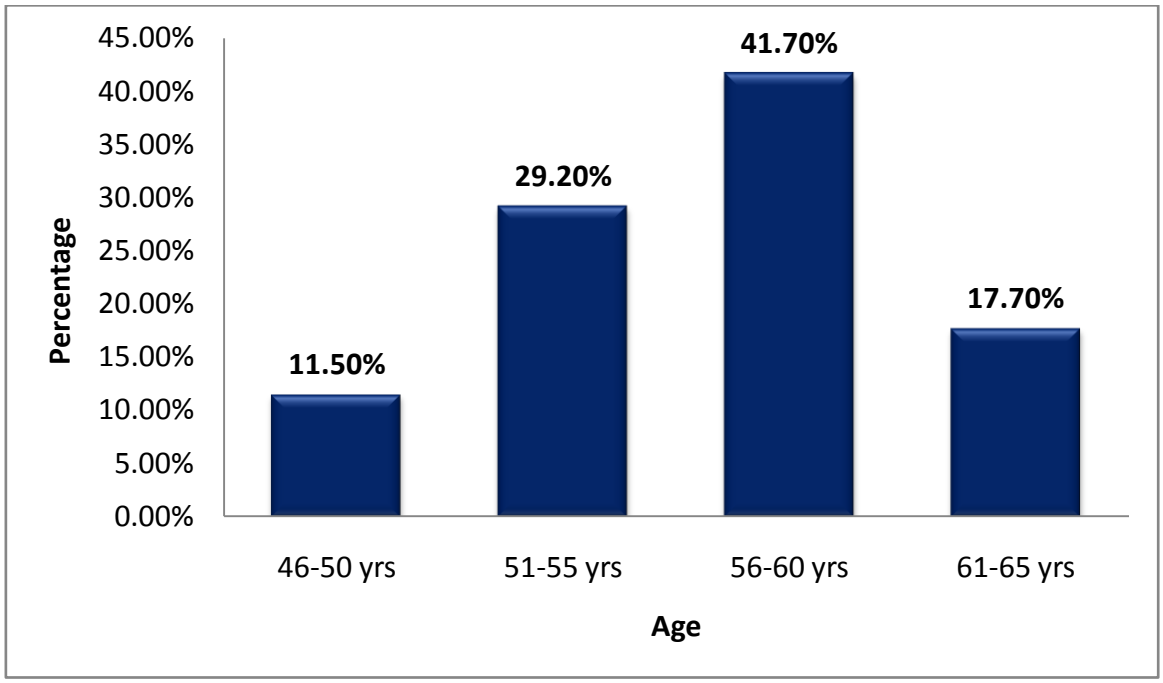
Picture 9 : Visual field of patient with MS : 27.0 dB and MD : 2.8dB

RESULTS

Table 1: Distribution of study participants according to their age (N=96)

Slno	Age	Frequency	Percentage
1	46-50 yrs	11	11.5
2	51-55 yrs	28	29.2
3	56-60 yrs	40	41.7
4	61-65 yrs	17	17.7
Mean=56.44±4.80	**Total**	**96**	**100**

Graph 1: Distribution of study participants according to their age (N=96)

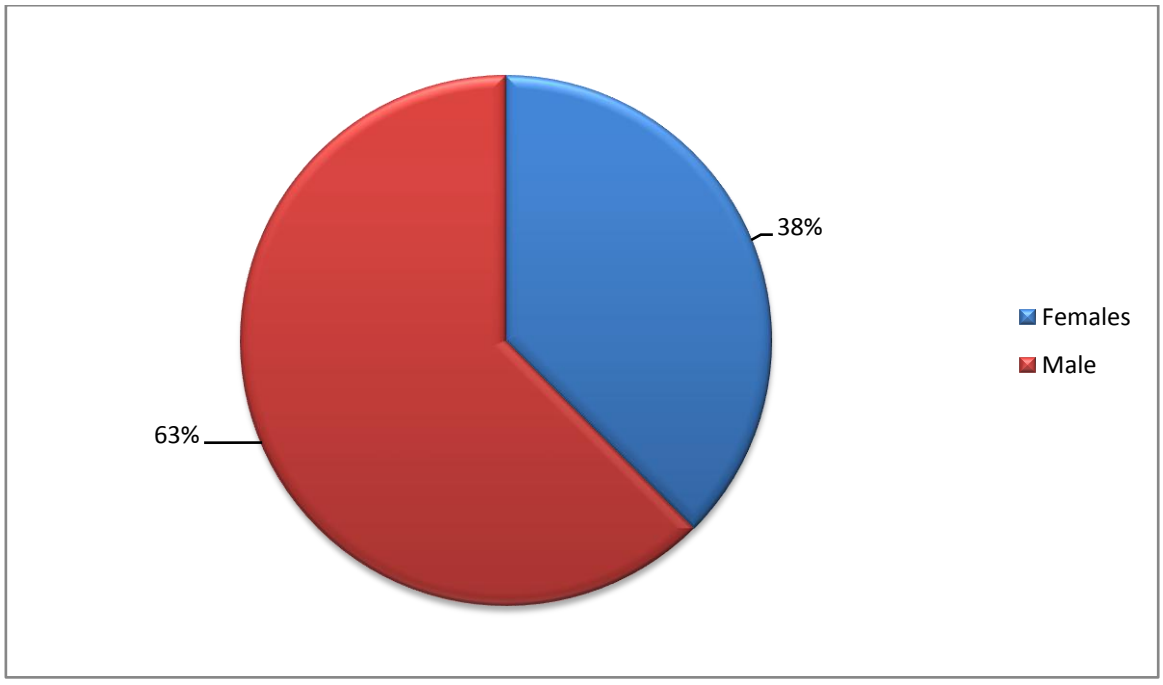


Majority of Patients in our study was in age group 56-60 years (41.7%) and 51-55 years (29.2%), followed by 61-65 years (17.7%), and 46-50 years (11.5%). Mean age of patient was 56 years**.**

Table 2: Distribution of study participants according to their gender (N=96)

Slno	Gender	Frequency	Percentage
1	Females	36	37.5
2	Males	60	62.5
	Total	**96**	**100**

Graph 2:Distribution of study participants according to their gender (N=96)

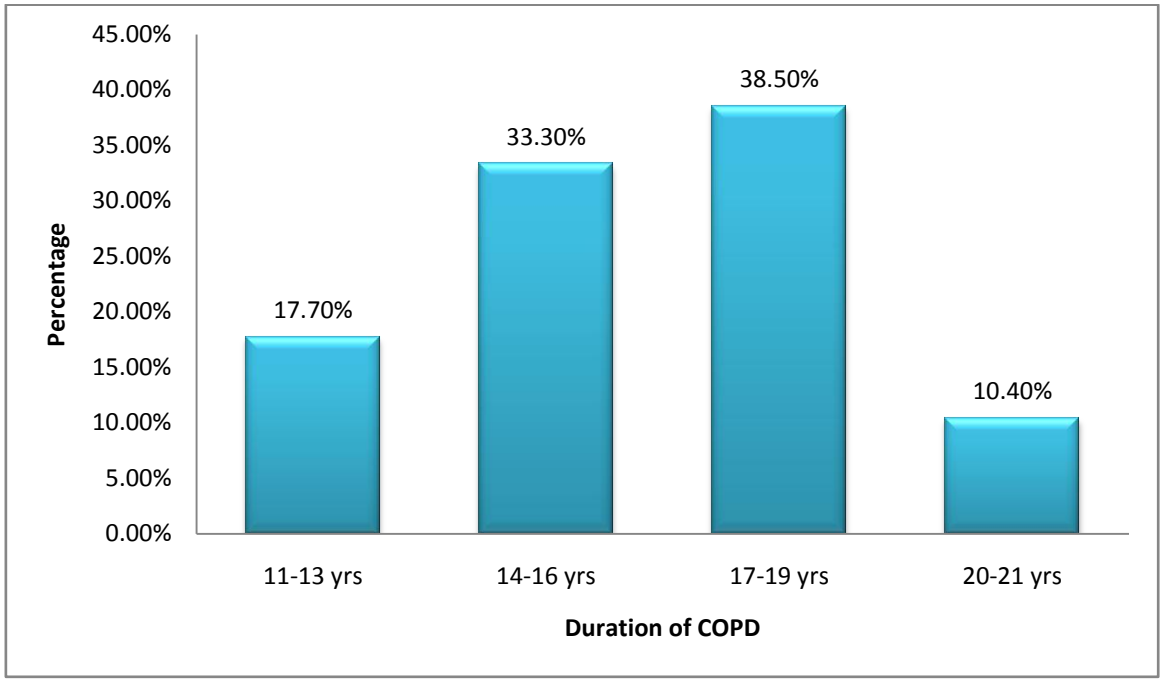


Male preponderance was observed in our study with 63% patients being males.

Table 3: Distribution of patients according to their duration of disease (N=96)

Slno	Duration	Frequency	Percentage
1	11-13 yrs	17	17.7
2	14-16 yrs	32	33.3
3	17-19 yrs	37	38.5
4	20-21 yrs	10	10.4
Mean =16.13±2.62	**Total**	**96**	**100**

Graph 3: Distribution of patients according to their duration of disease (N=96)

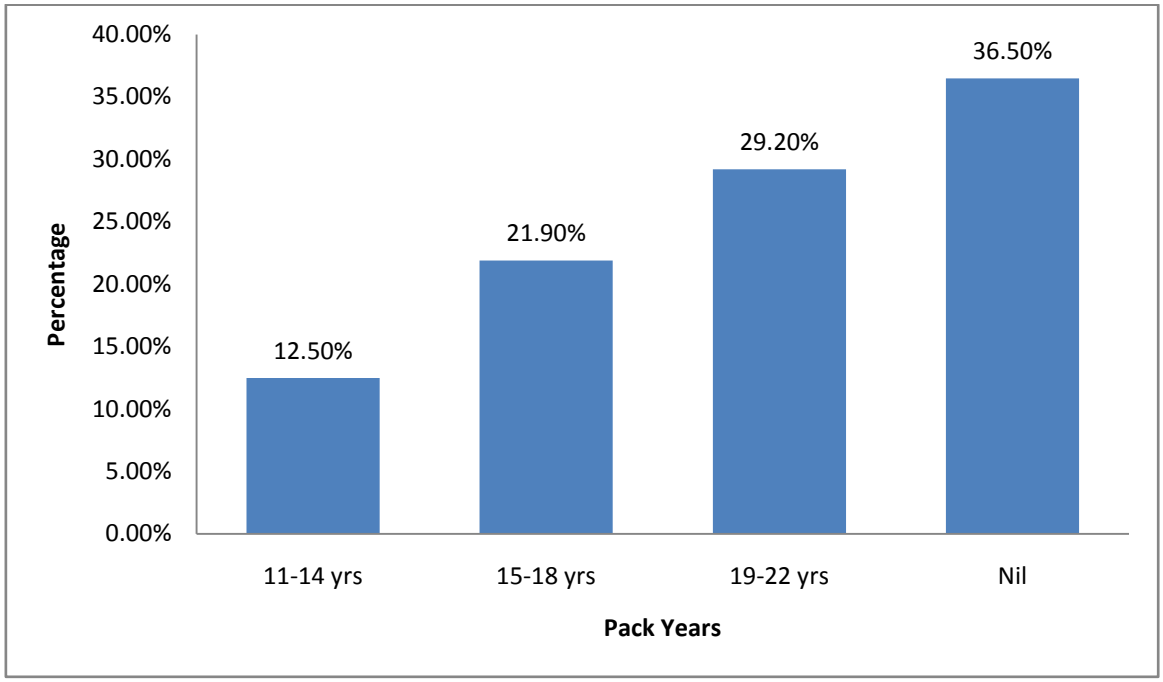


Majority of Patients in our study are having duration of COPD for 17-19 yrs (38%), 14-16 yrs (33.3%) and 11-13 years (17.7%), and 20-21 years (10.4%). Mean duration of COPD among patients were 16 years**.**

Table 4: Distribution of study participants according to their pack years (N=96)

Slno	Pack Years	Frequency	Percentage
1	11-14 yrs	12	12.5
2	15-18 yrs	21	21.9
3	19-22 yrs	28	29.2
4	Nil	35	36.5
Mean=17.54±2.97	**Total**	**96**	**100**

Graph 4: Distribution of study participants according to their pack years (N=96)



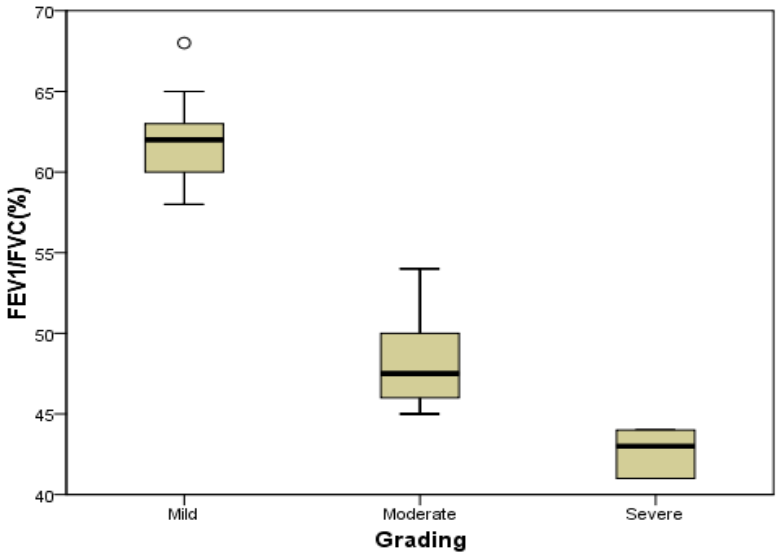
Majority of Patients in our study are having pack years between 19-20 yrs (29.2%), followed by 15-18 years (21.9%), and 11-14 years (12.5%). Mean pack years of patient was 17 years**.**

Table 5: Distribution of study participants according to their FEV1/FVC (%) (N=96)

Gender	COPD Categories	Number of patients	FEV1/FVC% Mean±SD	p value (One-way ANOVA)
Females	Mild	21	61.95±2.31	<0.001
	Moderate	10	48.10±2.76	
	Severe	5	42.60±1.51	
Males	Mild	37	61.08±2.91	<0.001
	Moderate	18	49.67±3.48	
	Severe	5	42.00±1.41	

There was a statistically significant difference between groups as determined by one-way ANOVA . A Tukey post hoc test revealed that the FEV1/FVC (%) was statistically significantly lower among the patients as the disease progresses.

Graph 5A: Distribution of study participants according to their FEV1/FVC (%) among females patients (n=36)



Graph 5B: Distribution of study participants according to their FEV1/FVC (%) among male patients (n=60)

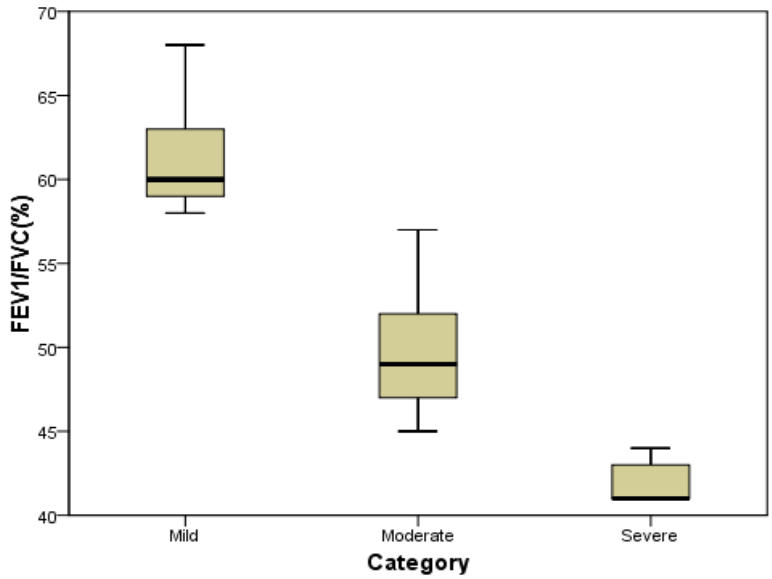
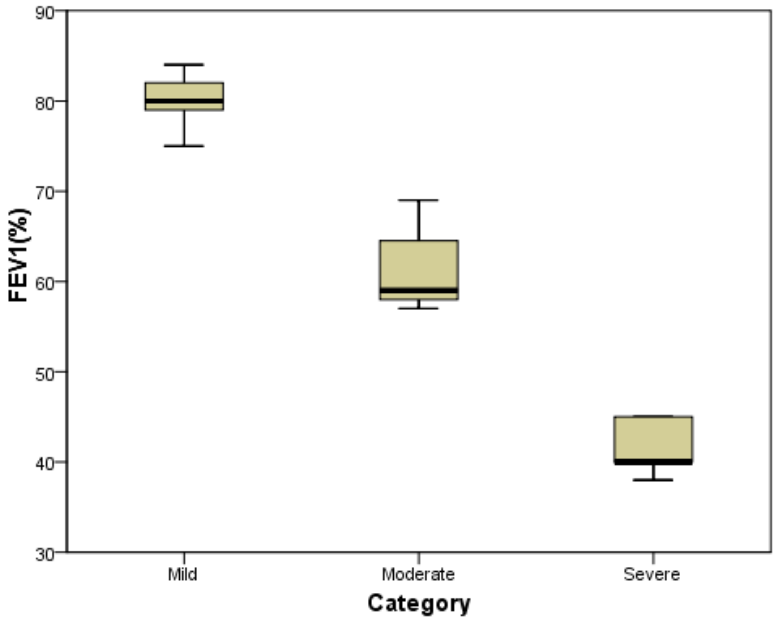


Table 6: Distribution of study participants according to their FEV1(%) (N=96)

Gender	COPD Categories	Number of patients	FEV1% Mean±SD	p value (One-way ANOVA)
Females	Mild	18	80.00±2.56	<0.001
	Moderate	12	61.00±4.45	
	Severe	6	41.33±2.94	
Males	Mild	29	78.10±2.84	<0.001
	Moderate	24	60.88±3.63	
	Severe	7	40.86±2.67	

There was a statistically significant difference between groups as determined by one-way ANOVA . A Tukey post hoc test revealed that the FEV1 (%) was statistically significantly lower among the patients as the disease progresses among both the genders.

Graph 6 A: Distribution of study participants according to their FEV1(%) among females (n=36)



Graph 6B:Distribution of study participants according to their FEV1(%) among males (n=60)

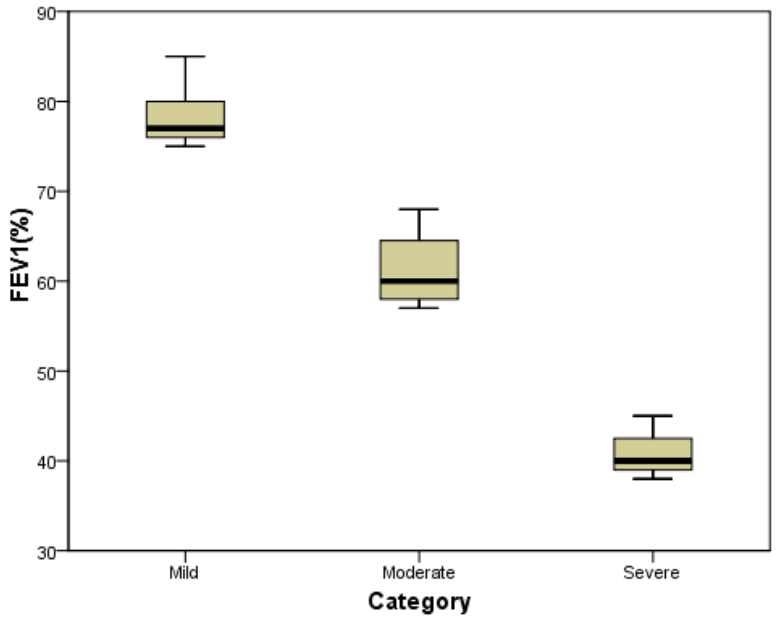


Table 7: Distribution of study participants according to their N75 VEP (ms) levels on right eye (N=96)

Gender	COPD category	Number of patients	N75 VEP (ms) Mean±SD	p value (One-way ANOVA)
Females	Mild	18	75.35±0.99	<0.001
	Moderate	12	76.75±0.33	
	Severe	6	77.92±0.00	
Males	Mild	28	75.18±0.94	<0.001
	Moderate	22	76.75±0.32	
	Severe	10	77.72±0.08	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **N75 VEP (ms)** was statistically significantly higher among the patients on right eye as the disease progresses, and similar results were seen among both the genders.

Table 8: Distribution of study participants according to their N75 VEP (ms) levels on left eye (N=96)

Gender	COPD Category	Number of patients	N75 VEP (ms) Mean±SD	p value (One-way ANOVA)
Females	Mild	21	75.64±0.85	0.008
	Moderate	9	76.72±0.17	
	Severe	6	76.68±1.68	
Males	Mild	23	75.32±0.95	<0.001
	Moderate	30	76.71±0.24	
	Severe	7	77.25±0.27	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **N75 VEP (ms)** was statistically significantly higher among the patients on left eye as the disease progresses, and similar results were seen among both the genders.

Table 9: Distribution of study participants according to their P100 VEP (ms) levels on right eye (N=96)

Gender	COPD Category	Number of patients	P100 VEP(ms) Mean±SD	p value (One-way ANOVA)
Females	Mild	18	104.27±4.11	<0.001
	Moderate	13	112.69±1.09	
	Severe	5	113.96±1.02	
Males	Mild	40	106.50±4.47	<0.001
	Moderate	13	112.67±1.06	
	Severe	7	113.96±0.59	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **P100 VEP (ms)** was statistically significantly higher among the patients on right eye as the disease progresses, and similar results were seen among both the genders.

Table 10: Distribution of study participants according to their P100 VEP (ms) levels on left eye (N=96)

Gender	COPD Category	Number of patients	P100 VEP(ms) Mean±SD	p value (One-way ANOVA)
Females	Mild	24	106.26±5.01	0.003
	Moderate	7	112.95±1.12	
	Severe	4	113.96±0.00	
Males	Mild	42	106.67±4.55	<0.001
	Moderate	12	112.73±1.28	
	Severe	6	113.66±0.30	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **P100 VEP (ms)** was statistically significantly higher among the patients on left eye as the disease progresses, and similar results were seen among both the genders.

Table 11: Distribution of study participants according to their N145 VEP (ms) levels (N=96)

Gender	N145 VEP (ms) Right eye	N145 VEP (ms) Left eye	p value
Females	147.56±1.87	147.39±1.35	0.539
Males	147.78±1.67	147.62±1.98	

There was no statistically significant difference between the groups for N145 VEP (ms) (p = .539).

Table 12: Distribution of study participants according to their P100 VEP (µv) levels on right eye (N=96)

Gender	COPD Category	Number of patients	P100 VEP (µv) Mean±SD	p value (One-way ANOVA)
Females	Mild	18	6.17±1.10	<0.001
	Moderate	13	3.80±0.74	
	Severe	5	2.50±0.20	
Males	Mild	27	6.20±0.86	<0.001
	Moderate	28	3.67±0.58	
	Severe	5	2.55±0.16	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **P100 VEP (µv)** was statistically significantly lower among the patients on right eye as the disease progresses, and similar results were seen among both the genders.

Table 13: Distribution of study participants according to their P100 VEP (µv) levels on left eye (N=96)

Gender	COPD Category	Number of patients	P100 VEP (µv) Mean±SD	p value (One-way ANOVA)
Females	Mild	18	6.21±1.10	<0.001
	Moderate	12	3.88±0.68	
	Severe	6	2.52±0.12	
Males	Mild	29	6.10±0.90	<0.001
	Moderate	28	3.49±0.52	
	Severe	3	2.56±0.07	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that the **P100 VEP (µv)** was statistically significantly lower among the patients on left eye as the disease progresses, and similar results were seen among both the genders.

Table 14: Distribution of study participants according to their visual field mean sensitivity levels on right eye (N=96)

Gender	COPD Category	Number of patients	Mean±SD	p value (One-way ANOVA)
Females	Mild	19	26.00±0.66	<0.001
	Moderate	13	22.36±0.49	
	Severe	4	21.00±0.00	
Males	Mild	32	25.88±0.60	<0.001
	Moderate	24	22.50±0.51	
	Severe	4	21.00±0.00	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that visual field sensitivitywas statistically significantly lower among the patients on right eye as the disease progresses, and similar results were seen among both the genders.

Table 15: Distribution of study participants according to their visual field mean sensitivity levels on left eye (N=96)

Gender	COPD Category	Number of patients	Mean±SD	p value (One-way ANOVA)
Females	Mild	18	26.11±0.32	<0.001
	Moderate	15	22.40±0.50	
	Severe	3	20.95±0.08	
Males	Mild	32	26.00±0.67	<0.001
	Moderate	23	22.43±0.50	
	Severe	5	21.00±0.00	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that visual field sensitivitywas statistically significantly lower among the patients on left eye as the disease progresses, and similar results were seen among both the genders.

Table 16: Distribution of study participants according to their visual field mean deviation levels on right eye (N=96)

Gender	COPD Category	Number of patients	Mean±SD	p value (One-way ANOVA)
Females	Mild	17	-1.71±0.49	<0.001
	Moderate	13	2.28±0.60	
	Severe	6	2.48±0.05	
Males	Mild	23	-0.22±1.69	<0.001
	Moderate	27	2.25±0.04	
	Severe	10	2.48±0.00	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that visual field mean deviation was statistically significantly higher among the patients on right eye as the disease progresses, and similar results were seen among both the genders. Patients with mild COPD had average visual field mean deviation levels of -1.71±0.49 for females and -0.22±1.69 for males.

Table 17: Distribution of study participants according to their visual field mean deviation levels on left eye (N=96)

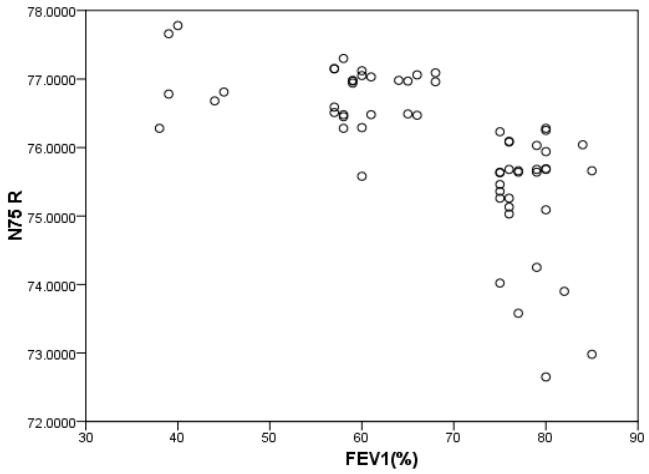
Gender	COPD Category	Number of patients	Mean±SD	p value (One-way ANOVA)
Females	Mild	10	-1.65±0.48	<0.001
	Moderate	8	2.29±0.06	
	Severe	18	2.40±0.50	
Males	Mild	17	-0.30±1.65	<0.001
	Moderate	13	2.27±0.05	
	Severe	30	2.50±0.13	

There was a statistically significant difference between groups as determined by one-way ANOVA. A Tukey post hoc test revealed that visual field mean deviation was statistically significantly higher among the patients on left eye as the disease progresses, and similar results were seen among both the genders. Patients with mild COPD had average visual field mean deviation levels of -1.65±0.48 for females and -0.30±1.65 for males.

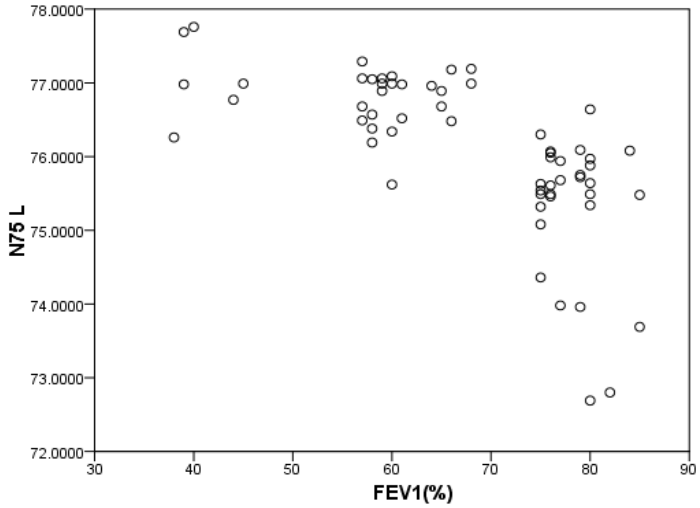
Table 18: Correlation of study participants according to their FEV1(%) and FEV1/FVC (%) with visual evoked potentials among females (n=36)

EYE	VEP	FEV1 (%)	FEV1/FVC (%)
Left	**P100(ms)**	r=-0.571 p=<0.001	r=-0.719 p=<0.001
	N75(ms)	r=-0593 p=<0.001	r=-0.660 p=<0.001
	N145(ms)	r=-0.138 p=0.421	r=-0.282 p=0.096
	P100(µv)	r=0.792 p=<0.001	r=0.712 p=<0.001
Right	**P100(ms)**	r=-0.690 p=<0.001	r=-0.787 p=<0.001
	N75(ms)	r=-0.364 p=0.029	r=-0.472 p=0.004
	N145(ms)	r=-059 p=0.734	r=-0.306 p=0.070
	P100(µv)	r=0.789 p=<0.001	r=0.679 p=<0.001

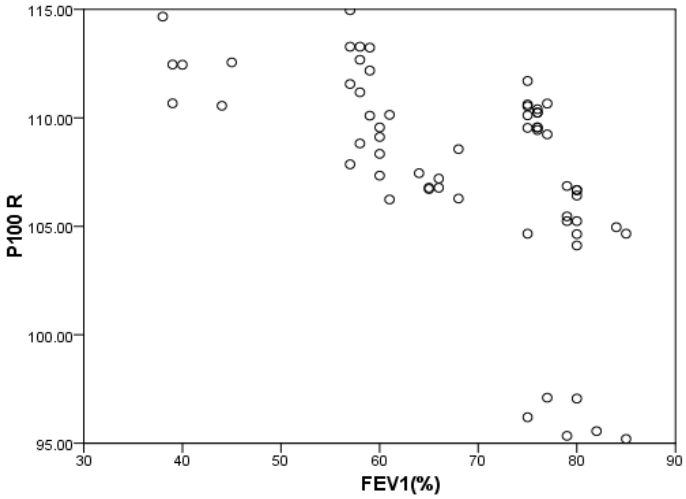
The correlation between the VEP variables and the severity of COPD revealed that the latency P100, N75 of both right eye and left eye werecorrelated negatively with the spirometric indices. All showed significant correlation except N145 amongfemales reported no statistical significance.The p100 amplitude of both right and left eyes was correlated positively and significant with the spirometric indices.



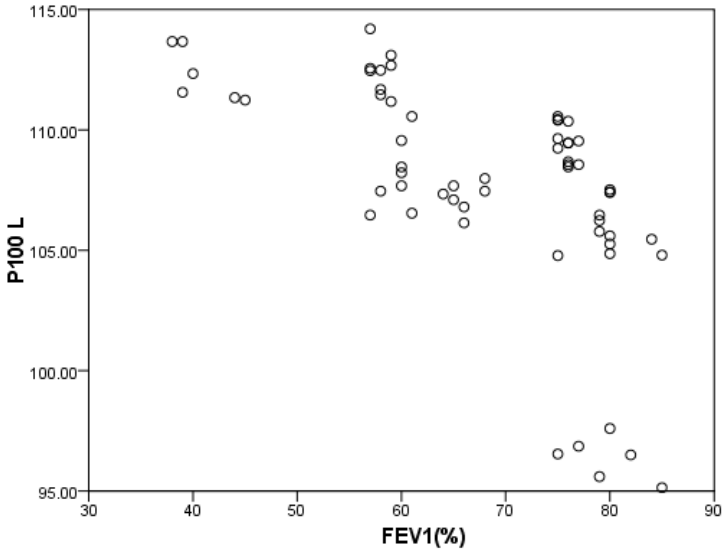
Graph 7: Scatter plot between N75 VEP on right eye Vs FEV1 (%) among females (n=36)



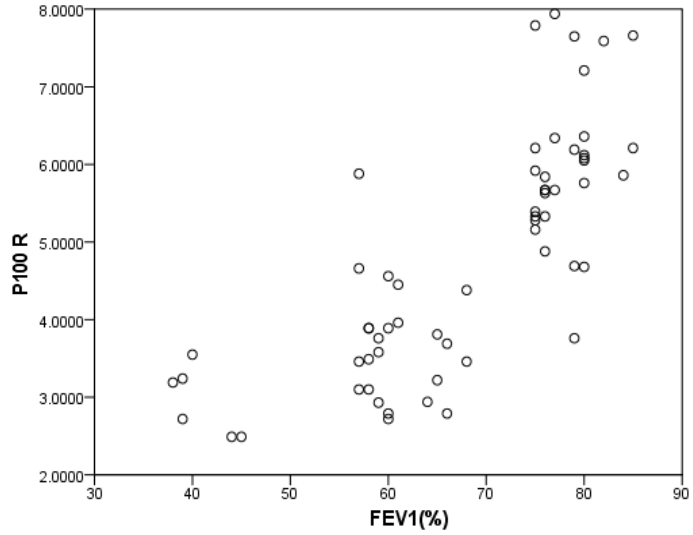
Graph 8: Scatter plot between N75 (ms) VEP on left eye Vs FEV1 (%) among females (n=36)



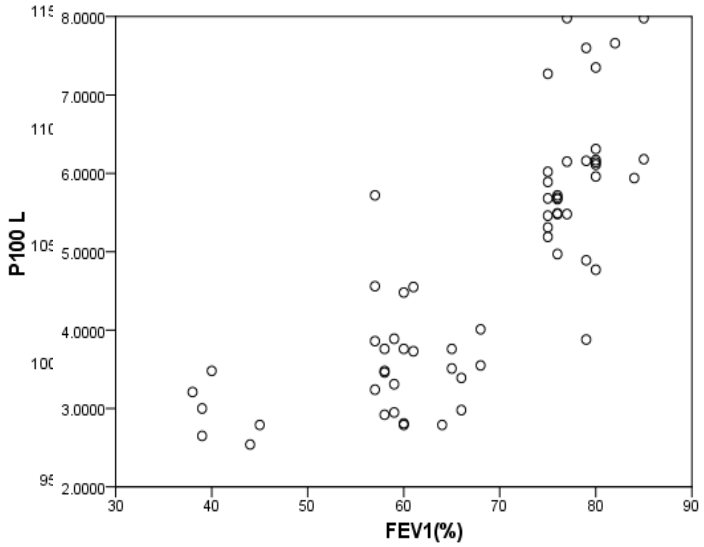
Graph 9: Scatter plot between P100 (ms) VEP on right eye Vs FEV1 (%) among females (n=36)



Graph 10: Scatter plot between P100 (ms) VEP on left eye Vs FEV1 (%) among females (n=36)



Graph 11: Scatter plot between P100 (µv) amplitude on right eye Vs FEV1 (%) among females (n=36)

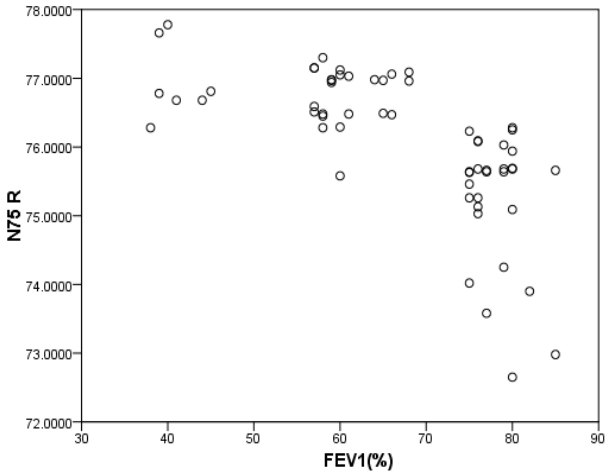


Graph 12: Scatter plot between P100 (µv) amplitude on left eye Vs FEV1 (%) among females (n=36)

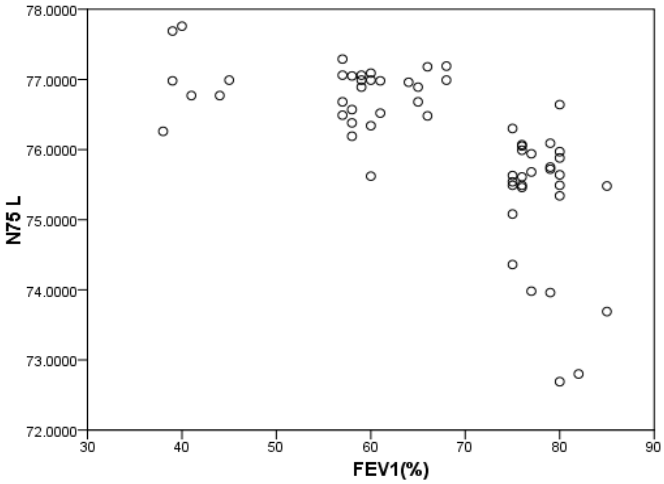
Table 19: Correlation of study participants according to their FEV1 (%) and FEV1/FVC (%) with visual evoked potentials among males (n=60)

EYE	VEP	FEV1 (%)	FEV1/FVC (%)
Left	**P100(ms)**	r=-0.701 p=<0.001	r=-0.629 p=<0.001
	N75(ms)	r=-0.668 p=<0.001	r=-0.636 p=<0.001
	N145(ms)	r=-0.299 p=0.020	r=-0.199 p=0.128
	P100(µv)	r=0.792 p=<0.001	r=0.884 p=<0.001
Right	**P100(ms)**	r=-0.629 p=<0.001	r=-0.701 p=<0.001
	N75(ms)	r=-0.667 p=<0.001	r=-0.636 p=<0.001
	N145(ms)	r=-0.122 p=0.354	r=-0.260 p=0.045
	P100(µv)	r=0.778 p=<0.001	r=0.674 p=<0.001

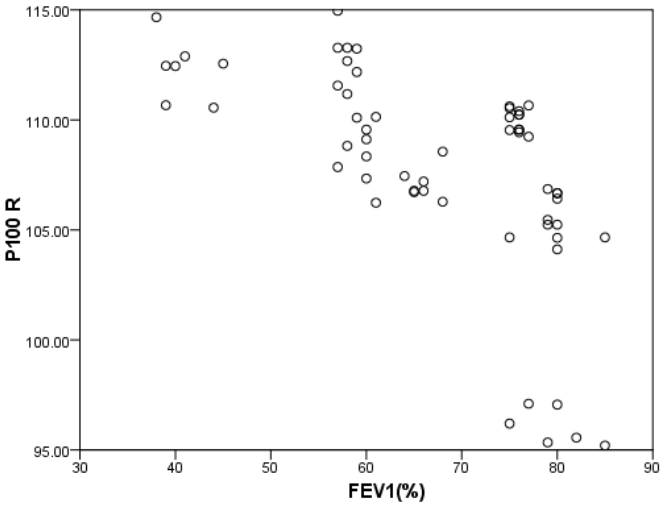
The correlation between the VEP variables and the severity of COPD revealed that the latency P100, N75 of both right eye and left eye werecorrelated negatively with the spirometric indices. All showed significant correlation except N145 among males reported no statistical significance.The P100 amplitude of both right and left eyes was correlated positively and significant with thespirometric indices.



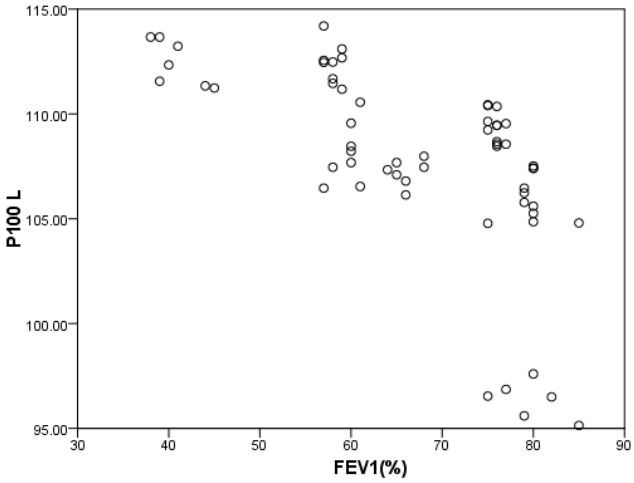
Graph 13: Scatter plot between N75 (ms) VEP on right eye Vs FEV1 (%) among Males (n=60)



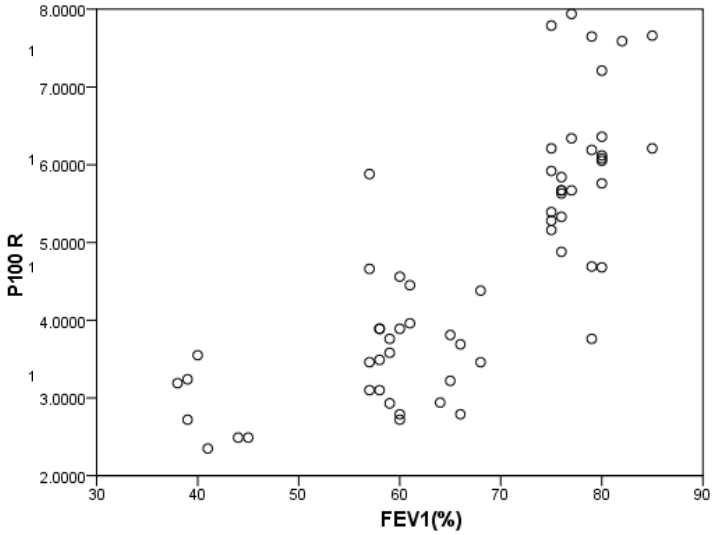
Graph 14: Scatter plot between N75 (ms) VEP on left eye Vs FEV1 (%) among Males (n=60)



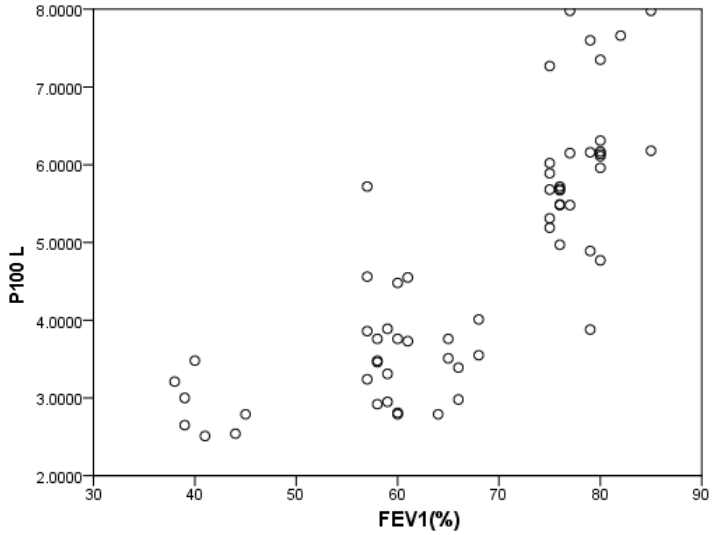
Graph 15: Scatter plot between P100 (ms) VEP on right eye Vs FEV1 (%) among Males (n=60)



Graph 16: Scatter plot between P100 (ms) VEP on left eye Vs FEV1 (%) among Males (n=60)



Graph 17: Scatter plot between P100(µv) VEP on right eye Vs FEV1 (%) among Males (n=60)

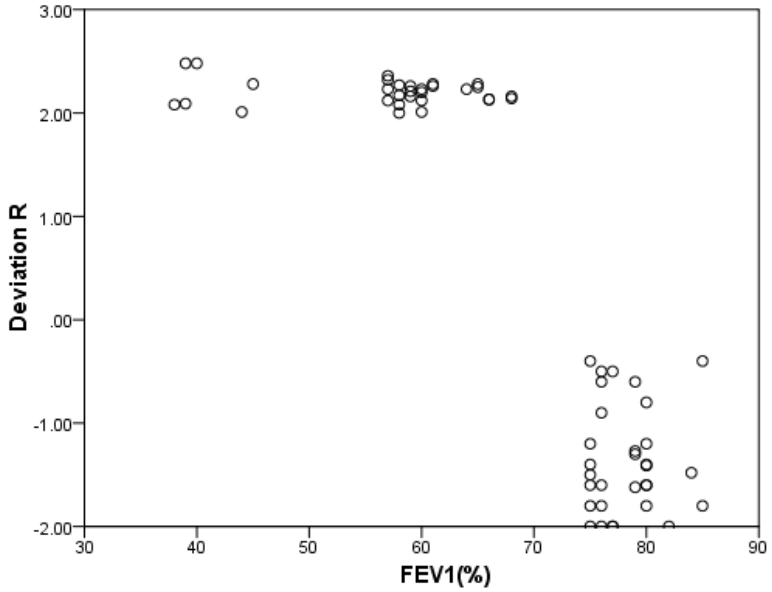


Graph 18: Scatter plot between P100(µv) VEP on left eye Vs FEV1 (%) among Males (n=60)

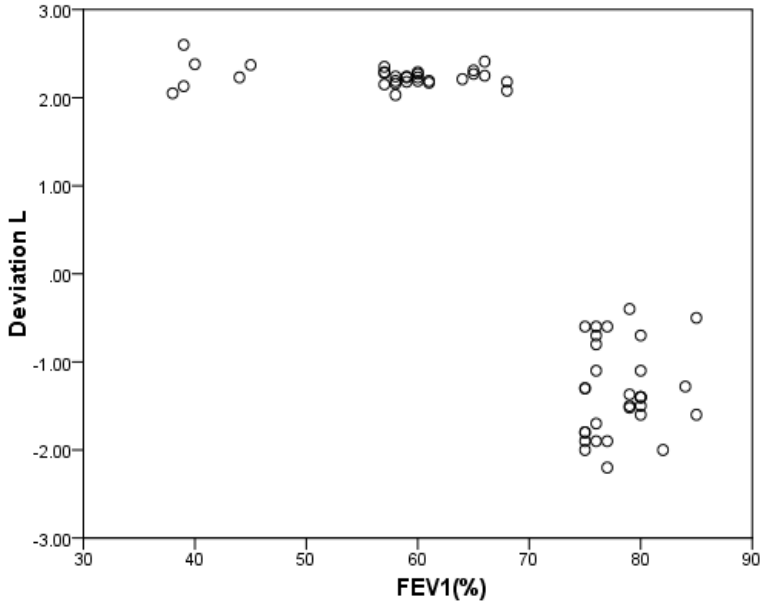
Table 20: Correlation of study participants according to their FEV1 (%) and FEV1/FVC (%) with visual fields on females (n=36)

EYE	Visual fields	FEV1 (%)	FEV1/FVC (%)
Left	**Mean sensitivity**	r=-0.833 p=<0.001	r=-0.697 p=<0.001
	Mean deviation	r=-0.829 p=<0.001	r=-0.776 p=<0.001
Right	**Mean sensitivity**	r=-0.871 p=<0.001	r=-0.719 p=<0.001
	Mean deviation	r=-0.832 p=<0.001	r=-0.781 p=<0.001

The correlation of the visual field and mean deviation among the COPD patients revealed that on both right eye and left eye were correlated negatively with the spirometric indices among Females. All showed significance



Graph 19: Scatter plot between visual field mean deviation on right eye Vs FEV1 (%) among females (n=36)

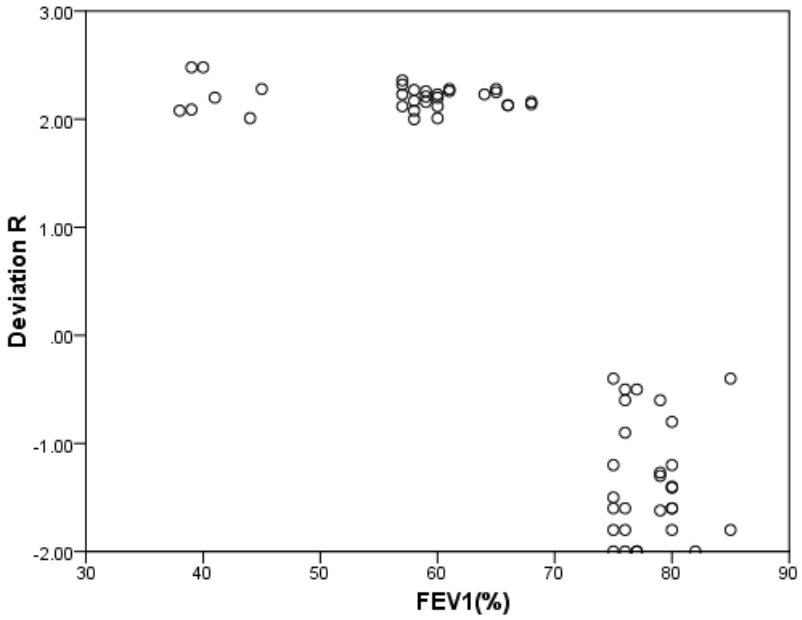


Graph 20: Scatter plot between visual field meandeviation on left eye Vs FEV1 (%) among females (n=36)

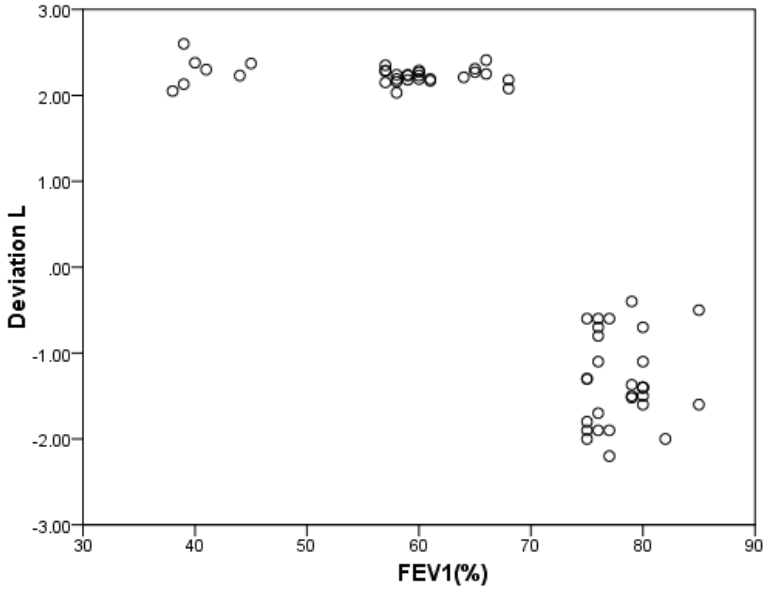
Table 21: Correlation of study participants according to their FEV1 (%) and FEV1/FVC (%) with visual fields on males (n=60)

EYE	Visual fields	FEV1 (%)	FEV1/FVC (%)
Left	**Mean sensitivity**	r=-0.845 p=<0.001	r=-0.642 p=<0.001
	Mean deviation	r=-0.829 p=<0.001	r=-0.713 p=<0.001
Right	**Mean sensitivity**	r=-0.834 p=<0.001	r=-0.621 p=<0.001
	Mean deviation	r=-0.811 p=<0.001	r=-0.684 p=<0.001

The correlation of the visual field and mean deviation among the COPD patients revealed that on both right eye and left eye were correlated negatively with the spirometric indices among males. All showed significance.



Graph 21: Scatter plot between mean deviation visual field on right eye Vs FEV1 (%) among Males (n=60)



Graph 22: Scatter plot between mean deviation visual field on left eye Vs FEV1 (%) among Males (n=60)

DISCUSSION

COPD is a disorder which involves multisystem and is frequently associated with significant extra pulmonary manifestations[26.] Even though COPD affects the lung, it also produces significant systemic consequences. The consequences can be detected clinically and appear to be associated with the presence of systemic inflammatory markers.All the patients included in our study were stable COPD patients without any clinical evidence of complications. The mean age of COPD patients observed in the present study was 56.44 years ranging from 46 to 65 years. A study conducted by **Atis et al**[28] reported the participants mean age to be 64 ± 6.5 years (range 46–72 years).**Bedar etal**[29] in his study also reported similar age group of patients. Few more studies were also carried out in older patients (with the mean age of 59.4±9.4 and 62.1±9.9 respectively). [30,31]Therefore, it could be confirmed in the present study that the COPD is also affecting the middle age group which is due to the disease and not age related. Male preponderance was observed in our study with 63% being males with COPD. However, according to GOLD, there is an increasing trend of COPD in females though it has plateaued in males.

Our study reported mean pack years of 17.54±2.97 years. The mean cigarette consumption was 24.59 ± 21.21 pack-years in a study done at turkey[27]. Similar findings were reported in few more studies.[29,30]The mean duration of patients suffering from COPD reported in our study was 16.13±2.62 yrs. Similar

results were found in a study by **Karthikeyan et al**[32] where the mean duration of disease was 15.03±5.15 years. The mean duration of COPD was 8 ± 2.82 years in a study done by **Atis et al**[28].

Our study results showed the mean latency of P100 wave in the right as well as left eye in COPD group was statistically prolonged with the stages of disease among both the genders($P < 0.001$). In a study done by **Gupta et al**[27] reported the mean latency of P100 wave in the right and left eye in COPD group was statistically prolonged as compared with that of the corresponding eye in healthy volunteers group ($P < 0.001$) and the mean amplitudes of P100 wavein both eyes of COPD patients were significantly decreased ($P < 0.001$) when compared with that in the eyes of the healthy volunteers group. **Sezer et al**[31] also showed that P100 value was altered in COPD patient and further hypothesized that the elevations in latencies were brought about by the hypoxia, hypercapnea and acidosis resulting from COPD.

In our study P100 VEP (ms), N75 (ms) was statistically significantly higher among the patients on both the eye as the disease progresses, and similar results were seen among both the genders. There was no statistically significant difference between the groups for N145 VEP (ms) ($p = .539$).P100 VEP (μv) was statistically significantly lower among the patients on both eyes as the disease progresses, and similar results were seen among both the genders. In a study done by **Ogze et al**[30] evaluated optic nerve involvement with severe COPD and

they observed VEP abnormalities. In a study done by **kanmani etal**[31] showed the correlation between the VEP variables and the characteristics of COPD patients revealed that the amplitude P100 of both eyes were correlated positively with the spirometric indices. **Sezeret al**[30]showed that P100 value was altered in COPD patients and further hypothesized that the elevations in latencies of both N and P waves were brought about by the hypoxia, hypercapnea and acidosis, resulting from COPD. Since, N145 wave is generated from extra-striate visual cortex, the insignificant result obtained in our study confirms the irrelevance of N145 wave getting affected by the COPD.

In the present study the correlation between the VEP variables and the characteristics of COPD patients revealed that the latency P100, N75 and N145 of both right and left eyes were correlated negatively with the spirometric indices. All showed significant correlation except N145 among males reported no statistical significance.The amplitude of P100 of both right and left eyes was correlated positively and significant with the spirometric indices.

Similar findings were seen in **Kanmani et al**[31] study where there was positive significant ($P<0.05$) relationships observed between P100 latency and spirometric indices (FEV1/FVC and FEV1 percent). However, the correlation coefficients for the spirometric indices and the amplitude (P100) were found to be positive and highly significant ($P<0.01$; Table III). This correlation was

further strengthened by ANOVA which showed highly significant differences existing among mild, moderate and severe COPD patients.

In our study the correlation between the visual field mean sensitivity as well as mean deviation among the COPD patients revealed that on both right eye and left eye were correlated negatively and are highly significant with the spirometric indices among both genders. **Helin at al**[33] study revealed mean deviation (MD), pattern standard deviation (PSD) and corrected pattern standard deviation (CPSD) were significantly different between their patients and control groups as for both standard achromatic perimetry (SAP) and short-wavelength automated perimetry (SWAP).

SUMMARY

A total of 96 patients were enrolled in the study. All the selected patients were within the inclusion criteria mentioned.

- Mean age of patient was 56 years**.**
- Male preponderance was observed in our study with 63% patients being males and 37% being females.
- Mean duration of COPD among patients were 16 years**.**
- Mean pack years of patient was 17 years**.**
- As per GOLD guidelines, 46 patients belonged to mild category, 34 belonged to moderate category and 16 belonged to severe category.
- According to statistical reports, FEV1/FVC % and FEV1% was significantly lower in both men and women as the disease progressed among them.
- N75 latency (ms) and P100 latency (ms) were higher on both eyes in men and women as the disease progressed among them.
- P100 amplitude (µV) was significantly lower among the patients in both eyes as the disease progressed and similar results were observed among both the genders.
- There was no statistically significant difference between the groups for N145 VEP (ms) (p = .539).
- Visual field mean sensitivity was significantly lower in both eyes in men and women as the disease progressed.

- Visual field mean deviation was significantly higher in both eyes in both genders as the disease progressed.

CONCLUSION

1. Our study included stable COPD patients with moderate airflow restriction having no clinical features suggesting neuropathy or visual impairment.
2. Our study identified the presence of impaired VEP and visual field parameter abnormalities noted in COPD patients with no clinical visual impairment.
3. In conclusion
    a. A significant decrease in P100 amplitude (μV)
    b. A significant increase in N75 latency (ms)
    c. A significant increase in P100 latency (ms)
    d. A significant decrease in visual field mean sensitivity (MS)
    e. A significant increase in visual field mean deviation (MD) were observed in both eyes in men and women as the disease progressed among them.
4. By identifying the presence of subclinical abnormalities of VEP waveforms and changes in visual field parameters, it can be useful when planning treatment strategies for the COPD patients.
5. By adopting spirometry in primary health care centers, we are able to categorize the COPD patients early and prompt referral to tertiary hospitals for further evaluation can be made.

    From the above data, there is a clear correlation between VEP abnormalities and visual field changes in COPD patients with nil

significant visual impairment, with a positive correlation with the grades of severity. Hence it can be concluded that VEP and visual field parameters are subclinical indicators of integrity of visual field pathway and is an effective screening tool to identify the same much earlier than visual symptoms develop.

LIMITATIONS OF OUR STUDY

1. In our study we didn't find any geographical difference between the prevalence of COPD and its severity correlation with VEP and visual field.
2. Most of our patients in our study were stable COPD patients, we excluded very severe COPD patients with acute respiratory symptoms and we did not find out their correlation with VEP and visual field parameters.
3. Various factors such as smoking tobacco, higher smoking pack years, malnutrition, that are individually associated with COPD have been known to cause neuropathy.
4. In our study, patients presented with longer duration of disease and higher smoking pack years didn't show any significant hypoxemia. Hence, whether severity of hypoxemia alone with chronicity of illness or with higher smoking pack years causing these abnormalities need to be further evaluated in future.

BIBLIOGRAPHY

1. Available from https://www.sleepapnea.org/when-sleep-apnea-and-copd-meet-overlap-syndrome/. Last acessed on 30th sep, 2019.
2. Petty TL. The history of COPD. International journal of chronic obstructive pulmonary disease. 2006 Mar;1(1):3.
3. Laënnec RTH. In: A treatise on the diseases of the chest (English translation from the French) Forbes J, editor. London: T and G Underwood; 1821
4. Hutchinson J. On the capacity of the lungs, and on the respiratory functions, with a view of establishing a precise and easy method of detecting disease by the spirometer. Medico-chirurgical transactions. 1846;29:137.
5. Benfante A, Sorino C, Scichilone N. The asthma-COPD overlap syndrome (ACOS): hype or reality? That is, a curiosity for the media or an opportunity for physicians?. Shortness of Breath. 2014;3(4):165-74.
6. Gaensler EA. Air velocity index: A numerical expression of the functionally effective portion of ventilation. American review of tuberculosis. 1950 Jul;62(1):17-28.
7. Christie RV. Emphysema of the Lungs—II. British medical journal. 1944 Jan 29;1(4334):143.
8. Oswald N, Harold J, Martin WJ. Clinical pattern of chronic bronchitis. The Lancet. 1953 Sep 26;262(6787):639-43.

9. Barach AL, Bickerman HA. Pulmonary emphysema. Baltimore: Williams and Wilkins; 1956.

10. Committee on Diagnostic Standards for Nontuberculous Respiratory Diseases, American Thoracic Society. Definitions and classification of chronic bronchitis, asthma, and pulmonary emphysema. Am Rev Respir Dis. 1962;85:762–9.

11. Carter R, Nicotra B, Blevins W, et al. Altered exercise gas exchange and cardiac function in patients with mild chronic obstructive pulmonary disease. Chest. 1993;103:745–50.

12. Sin DD, Man SF. Inhaled corticosteroids and survival in chronic obstructive pulmonary disease: does the dose matter? Eur Respir J. 2003;21:260–6.

13. Blumhardt LD, Barrett G, Halliday AM, Kriss A. The asymmetrical visual evoked potential to pattern reversal in one half field and its significance for the analysis of visual field effects. Br. J. Ophthalmol. 1977;61: 454-61.

14. Di Russo F, Martínez A, Sereno MI, Pitzalis S, Hillyard SA. Cortical sources of the early components of the visual evoked potential. Hum Brain Mapp. 2002;15(2):95-111.

15. Emmerson-Hanover R, Shearer DE, Creel DJ and Dustman RE: Pattern reversal evoked potentials: Gender differences and age related changes in amplitude and latency. Electroenceph clin Neurophysiol1994;92:93-101

16. Fishman GA, Sokol S. Electrophysiologic testing in disorders of the retina, optic nerve, and visual pathway. 1990. San Francisco: American Academy of Ophthalmology, 1990.

17. Creel DJ. Visually evoked potentials by Donnell J. Creel. Webvision: The Organization of the Retina and Visual System [Internet]. 2016:1-21.

18. Availabale from https://www.slideshare.net/hiranathdahal/visual-field-testing-and-interpretation-80589191. Last accessed on 30th sep, 2019.

19. Rabe KF. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. GOLD executive summary. Am J Respir Crit Care Med. 2007;176:532-55.

20. Aras YG, Aydemir Y, Güngen BD, Güngen AC. Evaluation of central and peripheral neuropathy in patients with chronic obstructive pulmonary disease. International journal of chronic obstructive pulmonary disease. 2018;13:1857.

21. Drislane FW. Visual evoked potentials. InThe Clinical Neurophysiology Primer 2007 (pp. 461-473). Humana Press.

22. Creel D. Visually Evoked Potentials. 2012 Mar 1. In: Kolb H, Fernandez E, Nelson R, editors. Webvision: The Organization of the Retina and Visual System [Internet]. Salt Lake City (UT): University of Utah Health Sciences Center; 1995.

[Available from: https://www.ncbi.nlm.nih.gov/books/NBK107218/]. Last accessed on Oct1st 2019.

23. Grecescu M. Optical coherence tomography versus visual evoked potentials in detecting subclinical visual impairment in multiple sclerosis. J Med Life. 2014;7(4):538–541.

24. Blum AS, Rutkove SB, editors. The clinical neurophysiology primer. Springer Science & Business Media; 2007 Sep 26.

25. Demir HD, İnönü H, Kurt S, Doruk S, Aydın E, Etikan İ. Evaluation of visual field parameters in patients with chronic obstructive pulmonary disease. Acta ophthalmologica. 2012 Aug;90(5):e349-54.

26. Racette L, Fischer M, Bebie H, Holló G, Johnson CA, Matsumoto C. Visual field digest. A guide to perimetry and the Ocotpus perimeter. 6th Edition. Haag-Streit AG, Koniz, Switzerland. 2016.

27. Gupta PP, Sood S, Atreja A, Agarwal D. Assessment of visual evoked potentials in stable COPD patients with no visual impairment. Ann Throac Med 2010; 5: 222–227.

28. Atiº S, Özge A, Sevim S. The brainstem auditory evoked potential abnormalities in severe chronic obstructive pulmonary disease. Respirol2001; 6: 225–229.

29. Kayacan O, Beder S, Deda G, Karnak D. Neurophysiological changes in COPD patients with chronic respiratory insufficiency. Acta Neurol Belg2001; 101: 160–165.

30. Ozge C, Ozge A, Yilmaz A, Yalçinkaya DE, Calikoðlu M. Cranial optic nerve involvements in patients with severe COPD. *Respirol*2005; 10: 666–672.

31. Sezer M, Yaman M, Oruc S, Fidan F, Unlu M. Visual evoked potential changes in chronic obstructive pulmonary disease. *Eur J Gen Med* 2007; 4: 115–118.

32. Karthikkeyan K, Padma K, Rao BV. Evaluation of visual evoked potential (VEP) in patients with chronic obstructive pulmonary disease (COPD). Indian J PhysiolPharmacol. 2015;59(2):182-8.

33. Demir HD, İnönü H, Kurt S, Doruk S, Aydın E, Etikan İ. Evaluation of visual field parameters in patients with chronic obstructive pulmonary disease. Acta ophthalmologica. 2012 Aug;90(5):e349-54.

PROFORMA

Serial number:

Name :

Age :

Sex :

Occupation :

Address :

Ocular complaints :

Associated systemic illness:

Duration of COPD :

Treatment :

Smoking pack years :

Family history :

Spirometry indices:

FEV 1/FVC :

FEV 1 :

COPD classification :

(By gold criteria)

BASELINE OCULAR EXAMINATION

	RE	**LE**
Vision		
Eyelid and eyelashes		
Extra ocular movements		

SLIT LAMP EXAMINATION

Conjunctiva		
Cornea		
Anterior chamber		
Iris		
Pupil		
Lens		
EOM		
Refraction		

VISUAL FIELDS USING HAAG-STREIT

OCTOPUS 300 PERIMETER

Mean sensitivity[ms]:

Mean defect[MD]

[dB]

Colourvision :

IOP :

Fundus examination :

Pattern VEP :

Diagnosis :

சுய ஒப்புதல் படிவம்

ஆராய்ச்சி நிலையம் மருத்துவமனை : அரசு ஸ்டான்லி மருத்துவ கல்லூரி

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

பங்கு பெறுபவர் இதனை ( )குறிக்கவும்

நாள் பட்ட நுரைஈரல் அடைப்பு நோய் உள்ள நோயாளிகளுக்கு இந்நோயினால் கண் நரம்பில் ஏற்படும் பாதிப்பை முன்னமே கண்டறிய ஓர் ஆய்வு.

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

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

மேற்கண்ட பரிசோதனையின் பொது ஏற்பட கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

பங்கு பெறுபவரின் கையொப்பம்

ஆய்வில் உங்கள் பங்கேற்பு மற்றும் உரிமைகள்

இந்த ஆய்வில் தங்களின் பங்கேற்பு தன்னிச்சையானது. மற்ற காரணங்கள் எதை யும் கூறாமலேயே நீங்கள் இந்த ஆய்வில் இருந்து எந்த நேரத்திலும் விலகிக் கொள்ளலாம் , எந்த ஒரு நேரத்திலும் உங்களுக்கு திருப்தி இல்லை என்று உணர்ந்தாலோ அல்லது வேறு ஏதேனும் உடல் நல குறைவு உண்டானாலோ உங்களை கவனித்து வரும் மருத்துரிடம் உடனடியாக தெரிவிக்கவும். உங்களுக்கு சிகிச்சை பொருத்தமாக இருக்காது என தோன்றினால் உடனடியாக நிறுத்தப்படும்.

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

மருத்துவர் கவுசல்யா. அ,
முதுநிலை முருத்துவ மாணவர்,
கண் இயல் துறை,
அரசு ஸ்டான்லி மருத்துவ கல்லூரி மருத்துவமனை
சென்னை
தொலைபேசி எண் : 9790464696

KEY TO MASTER CHART:

FEV – Forced expiratory volume

FVC – Forced vital capacity

MS – Mean sensitivity

MD – Mean deviation

ms – millisecond

µv – microvolt

MASTER CHART

S.NO	PATIENT NAME	AGE	SEX	DURATION OF DISEASE	QUANTUM OF SMOKING	FEV1/FVC(%)	FEV1	N75 (MILLISECONDS)		P100(MILLISECONDS)		N145 (Millisecond)		p100 (microvolt)		FIELDS (MEAN SENSITIVITY)		MEAN DEVIATION	
					(PACK YEARS)		(%)	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE	RE	LE
1	Mathew	46	male	12	13	68	80	72.65	72.69	97.06	97.6	145	143	7.21	7.35	25.5	25.9	-1.41	-1.5
2	umayal	46	female	12	NIL	68	79	73.86	73.71	97.67	97.32	140	142	7.51	7.56	26.01	26.06	-1.8	-1.9
3	sundaram	47	male	11	12	67	79	74.25	73.96	95.34	95.6	147	146	7.65	7.6	25.14	25.23	-1.62	-1.52
4	sujatha	45	female	12	nil	84	72	76.03	76.06	105.24	104.64	146	147	5.67	5.48	27.1	27	2.1	2.1
5	rajarajan	48	male	11	12	66	85	72.98	73.69	95.2	95.14	145	147	7.66	7.98	26.32	25.99	-1.8	-1.6
6	hameed	46	male	11	10	65	82	73.9	72.8	95.56	96.5	147	146	7.59	7.66	26.01	26.31	-2	-2
7	michael	48	male	12	12	64	77	73.58	73.98	97.1	96.86	145	143	7.94	7.98	26.39	26.24	-2	-1.9
8	anjalai	50	female	11	nil	65	77	73.66	73.79	97.1	97.24	148	147	7.88	7.96	25.4	25.56	-1.7	-1.9
9	raji	47	female	12	NIL	64	78	73.69	72.97	94.38	94.56	147	147	7.95	7.91	26.18	26.22	-1.4	-1.2
10	lakshmanan	50	male	12	13	65	80	75.09	75.34	104.64	105.26	146	147	6.36	6.17	26.31	26.54	-0.8	-0.7
11	laila	50	female	12	nil	64	79	75.66	76.05	97.26	97.74	149	149	7.93	7.95	26.19	26.2	-1.2	-1.2
12	somasundaram	52	male	13	14	63	79	76.03	76.09	105.24	105.78	148	147	4.69	4.89	25.71	26.78	-1.3	-1.5
13	mani	51	male	14	17	62	77	75.66	75.94	110.66	109.54	146	143	5.67	5.48	26.47	26.33	-2	-2.2
14	kathirvel	55	male	15	16	58	75	75.64	75.63	110.54	109.64	149	148	5.28	5.31	25.79	25.88	-1.8	-1.9
15	vijaya	51	female	15	nil	62	84	75.03	75.33	104.64	104.86	150	149	4.98	4.86	26.57	26.64	-1	-1.1
16	mutharasi	52	female	14	nil	62	80	76	76.06	104.76	105.46	146	147	7.86	7.93	26.44	26.48	-1.5	-1.8
17	masthan	54	male	13	17	59	75	75.63	75.54	109.54	109.24	148	150	5.39	5.68	25.99	25.68	-1.5	-1.3
18	kathiravan	53	male	15	14	59	76	75.03	75.61	110.4	110.36	146	148	5.67	5.69	26.13	26.25	-1.8	-1.7
19	muthan	55	male	14	15	58	76	75.13	75.99	109.44	108.46	152	150	4.88	4.97	26.35	26.44	-2	-1.9
20	karunanithi	53	male	15	16	60	80	76.25	75.97	106.42	107.4	149	148	5.76	5.96	25.98	26.91	-1.2	-1.4
21	solai	55	male	14	15	58	75	76.23	76.3	110.62	110.4	147	146	6.21	6.02	25.47	25.69	-0.4	-0.6
22	muthu	52	male	15	16	59	77	75.64	75.68	109.24	108.56	148	149	6.34	6.15	26.55	26.49	-0.5	-0.6
23	maniyan	54	male	13	16	62	80	75.94	75.88	104.12	104.86	147	147	6.05	6.11	25.98	25.88	-1.6	-1.4
24	munniyammal	55	female	15	nil	63	80	76.09	76.21	105.24	105.42	149	148	5.98	6	26.28	26.32	-0.6	-0.8
25	govindammal	54	female	14	nil	62	80	76.18	76.25	104.8	105.6	148	147	5.69	5.67	26.38	26.49	-1.8	-1.8
26	nandan	55	male	15	14	63	80	75.69	75.49	106.66	107.52	149	148	6.12	6.31	26.03	26.15	-1.8	-1.6
27	gopal	54	male	13	16	62	79	75.64	75.72	105.46	106.24	147	146	6.19	6.16	25.68	25.81	-0.6	-0.4
28	kanaga	53	female	16	17	61	84	76.04	76.08	104.96	105.46	148	149	5.86	5.94	26.24	26.01	-1.48	-1.28
29	karpagam	55	female	14	16	58	75	75.36	75.32	111.7	110.56	148	147	5.33	5.46	25.87	25.73	-1.4	-1.8
30	saba	51	male	14	16	60	76	76.08	76.05	109.56	108.68	148	148	5.84	5.72	26.35	26.42	-0.5	-0.6
31	vaithi	52	male	15	16	59	75	75.26	75.08	104.66	104.78	147	146	5.16	5.19	26.15	26.31	-1.6	-1.8
32	vasudevan	54	male	14	14	59	79	75.68	75.75	106.86	106.46	149	148	3.76	3.88	26.26	26.48	-1.27	-1.37
33	ramani	54	female	15	nil	60	82	75.89	75.94	105.46	105.44	148	148	5.91	5.83	25.81	25.98	-1.8	-0.9
34	ragu	53	female	13	NIL	60	80	75.96	75.76	106.46	106.24	147	146	5.72	5.76	26.32	26.15	-1.1	-0.8
35	nandagopal	54	male	14	16	58	76	76.09	76.07	110.24	109.46	148	147	5.63	5.67	25.72	25.79	-1.6	-1.1

36	kalavathy	52	female	15	nil	61	81	76.31	76.28	105.12	105.26	148	147	5.99	6.01	25.64	25.85	-0.9	-1.2
37	kumari	51	female	15	nil	59	76	75.96	75.49	111.62	110.78	148	147	5.84	5.62	26.82	26.69	-0.4	-0.5
38	ravi	51	male	13	14	63	85	75.66	75.48	104.66	104.8	149	150	6.21	6.18	26.15	26.03	-0.4	-0.5
39	krishnadas	53	male	15	17	61	80	75.68	75.64	106.68	107.42	146	145	6.08	6.14	26.19	26.28	-1.4	-1.1
40	sekar	57	male	18	21	58	76	75.26	75.46	109.56	108.56	150	149	5.33	5.49	25.97	25.99	-0.6	-0.8
41	rani	58	female	17	nil	59	79	75.64	75.67	109.46	109.24	149	148	4.86	4.98	26.14	26.2	-1.4	-1.8
42	thirumalai	56	male	15	18	59	80	76.28	76.64	105.24	105.6	148	146	4.68	4.77	26.38	26.84	-1.6	-1.4
43	vedanayagam	56	male	17	20	58	76	75.68	75.49	110.24	109.46	147	148	5.67	5.48	26.66	26.73	-0.9	-0.7
44	sivagami	56	female	18	nil	63	83	76.02	76.08	105.1	104.18	145	146	5.42	5.61	26.62	26.44	-1.9	-1.8
45	valarmathy	57	female	16	nil	62	83	75.98	75.87	104.26	105.24	148	147	5.16	5.14	25.94	25.72	-0.4	-0.2
46	pachamuthu	57	male	16	18	58	75	75.46	75.49	110.12	110.44	149	148	5.92	5.89	25.77	25.81	-1.2	-1.3
47	malliga	56	female	18	nil	62	80	76.07	76.13	105.68	105.62	147	148	5.34	5.67	25.83	25.64	-0.32	-0.6
48	murugan	59	male	17	20	49	57	76.59	76.68	114.96	114.2	148	149	5.88	5.72	26.34	26.03	2.12	2.15
49	lingam	58	male	16	18	52	65	76.97	76.89	106.72	107.68	150	151	3.22	3.51	21.58	21.63	2.25	2.27
50	pichandi	56	male	15	18	63	68	77.09	77.19	108.56	107.46	149	148	4.38	4.01	22.68	22.75	2.16	2.08
51	alex	59	male	17	20	45	57	77.15	77.29	113.28	112.46	150	149	4.66	4.56	21.79	21.84	2.23	2.29
52	sampoornam	57	female	16	nil	54	62	76.34	76.28	108.46	109.78	149	148	4.48	4.36	22.66	22.63	2.18	2.14
53	padma	56	female	17	nil	60	67	77.25	77.31	106.6	106.46	145	146	2.82	2.97	21.57	21.62	2.23	2.26
54	jothimani	60	male	18	21	46	57	77.15	77.06	111.56	112.56	149	148	3.1	3.24	23.77	23.88	2.32	2.35
55	dasaradan	60	male	16	19	47	58	76.48	76.57	113.28	112.48	147	146	3.89	3.76	23.67	23.94	2.27	2.24
56	radha	59	female	18	nil	48	59	76.75	76.98	111.24	110.26	149	149	2.59	2.38	22.09	22.18	2.15	2.18
57	lakshmi	59	female	18	nil	50	60	76.38	76.29	109.26	108.48	150	150	4.22	4.05	21.64	21.57	2.12	2.35
58	sarala	60	female	18	nil	46	58	77.15	77.24	113.96	114.96	149	148	4.06	4.19	22.66	22.48	2.38	2.34
59	ramasamy	59	male	17	20	49	59	76.98	77.06	113.24	112.68	150	152	2.93	2.95	21.16	21.06	2.21	2.23
60	kubendran	59	male	17	19	50	60	75.58	75.62	108.34	107.68	148	147	3.89	3.76	22.27	22.34	2.12	2.19
61	jayavel	58	male	18	19	60	68	76.96	76.99	106.28	107.98	146	146	3.46	3.55	22.61	22.82	2.14	2.18
62	sankari	60	female	18	nil	47	59	77.29	77.34	113.98	111.46	145	147	4.26	4.18	21.67	21.48	2.29	2.31
63	kuppan	60	male	18	21	48	57	76.51	76.49	107.86	106.46	150	149	3.46	3.86	22.56	22.49	2.36	2.28
64	devi	60	female	18	nil	47	58	76.67	76.58	106.44	107.24	148	147	4.09	4.46	21.91	21.82	2.25	2.23
65	anthony	60	male	18	21	46	58	76.45	76.38	108.82	107.46	145	144	3.49	3.48	21.58	21.68	2.17	2.19
66	mohammed basha	57	male	17	19	54	61	76.48	76.52	106.24	106.54	149	149	3.96	3.73	22.75	22.94	2.26	2.17
67	manikandan	58	male	17	19	52	60	77.12	77.09	107.34	108.46	145	146	2.72	2.79	21.67	21.78	2.2	2.23
68	pandirajan	60	male	18	19	49	59	76.97	76.99	110.1	111.18	151	151	3.58	3.31	22.62	22.49	2.26	2.24
69	anthonyammal	60	female	17	nil	45	58	76.68	76.67	111.7	113.78	147	148	4.25	4.17	21.49	21.68	2.14	2.23
70	backiyanathan	60	male	18	21	49	59	76.94	76.89	112.18	113.1	149	147	3.76	3.89	22.55	22.71	2.16	2.18
71	manikavel	59	male	18	20	50	60	77.05	76.99	109.12	108.22	148	149	4.56	4.48	22.67	22.84	2.23	2.27
72	vennila	56	female	16	nil	64	68	76.39	76.42	106.6	106.78	146	147	2.77	2.94	21.67	21.49	2.18	2.15
73	santhanam	58	male	17	19	57	65	76.49	76.68	106.78	107.1	146	148	3.81	3.76	22.37	22.48	2.28	2.31
74	gopalan	56	male	15	18	60	66	76.47	76.48	107.2	106.14	149	148	3.69	3.39	21.94	21.89	2.13	2.41

75	velayutham	57	male	16	18	59	61	77.03	76.98	110.14	110.56	147	149	4.45	4.55	21.72	21.85	2.28	2.19
76	marimuthu	59	male	17	20	56	66	77.06	77.18	106.78	106.8	145	146	2.79	2.98	22.67	22.61	2.13	2.25
77	chandira	59	female	17	nil	49	57	76.91	76.68	113.12	112.46	149	147	4.64	4.54	22.79	22.98	2.31	2.11
78	mageshwari	61	female	19	nil	45	57	77.21	77.06	112.6	111.8	149	148	3.17	3.23	23.89	23.47	2.17	2.18
79	sabapathi	60	male	18	21	45	58	76.28	76.19	112.68	111.46	150	152	3.89	3.46	22.57	22.67	2.08	2.03
80	kuttyammal	64	female	20	nil	41	69	76.98	76.78	113.12	111.45	149	147	4.76	4.24	23.15	23.38	2.16	2.19
81	manjula	63	female	20	nil	50	45	76.32	76.21	112.43	99.37	148	147	2.58	2.62	22.72	22.68	2.22	2.21
82	kovindammal	62	female	19	nil	44	40	77.92	77.87	114.6	113.9	147	149	2.15	2.46	21.94	21.57	2.12	2.09
83	selvarasan	59	male	17	21	75	60	77.5	77.5	113.13	113.75	148	149	3.01	3.04	27	27	2.3	2.29
84	srinivasan	61	male	19	19	60	58	77.3	77.05	111.18	111.68	147	148	3.1	2.92	23.18	23.28	2	2.16
85	vashmuni	63	male	20	20	61	64	76.98	76.96	107.45	107.34	150	150	2.94	2.79	21.97	21.88	2.23	2.21
86	joseph	65	male	21	22	41	38	76.28	76.26	114.67	113.67	149	147	3.19	3.21	22.64	22.84	2.08	2.05
87	ramesh	64	male	20	nil	43	41	76.68	76.77	112.89	113.23	148	147	2.35	2.51	22.03	21.85	2.2	2.3
88	mangalam	63	female	19	nil	62	45	76.82	76.76	111.67	111.45	146	147	3.02	3.25	20.67	20.86	2.12	2.08
89	kuppusamy	62	male	20	20	59	44	76.68	76.77	110.56	111.34	147	146	2.49	2.54	21.98	21.88	2.01	2.23
90	darmendra	61	male	19	20	60	45	76.81	76.99	112.56	111.24	149	149	2.49	2.79	20.88	20.76	2.28	2.37
91	danam	65	female	21	nil	41	38	76.75	76.68	113.56	113.2	148	147	2.89	2.56	21.88	21.79	2.19	2.27
92	munian	69	male	19	20	80	51	76.81	76.77	114.38	113.75	145	145	3.41	3.47	25	25	2.8	2.8
93	moorthy	64	male	21	22	41	39	77.66	77.69	110.67	111.56	148	149	3.24	3	21.68	21.49	2.09	2.13
94	pasupathi	65	male	21	22	41	39	76.78	76.98	112.46	113.67	147	149	2.72	2.65	20.88	20.67	2.48	2.6
95	selvi	64	female	20	nil	43	40	73.29	77.49	111.45	111.65	146	146	2.64	2.68	21.49	21.66	2.48	2.39
96	asha	63	female	19	nil	44	40	76.59	76.94	113.65	112.96	149	149	2.58	2.46	22.38	22.75	2.15	2.37