PULMONARY FUNCTION TEST ANALYSIS IN SMOKERS

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CERTIFICATE

This is to certify that **Dr. T. MUTHU ,** Post - Graduate Student (July 2003 to September 2006) in the Department of Internal Medicine, Kilpauk Medical College, Chennai- 600 010, has done this dissertation on **"PULMONARY FUNCTION TEST ANALYSIS IN SMOKERS"** under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in September 2006.

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CONTENTS

ABBREVIATIONS

FVC	-	Forced vital capacity
VC	-	Vital capacity
FEV_1	-	Forced expiratory volume in 1s
FEV_1/FVC	-	Ratio of FEV_1 to FVC
$FEF_{25-75\%}$	-	Forced expiratory flow rate between 25 and 75% of the VC
TLC	-	Total lung capacity
RV	-	Residual volume
MMFR	-	Maximal midexpiratory flow rate
MIP	-	Maximal inspiratory pressure
MEP	-	Maximal expiratory pressure

INTRODUCTION

Pulmonary Function Tests involves the measure of airflow rates, lung volumes, and the ability of the lung to transfer gas across the alveolar capillary membrane. Pulmonary function parameters are very useful to diagnose obstructive lung disease. Smoking is the most important risk factor for chronic obstructive pulmonary disease.

SMOKING

Smoking is major public health problem. Cigarette smoke contains more than 4000[1] compounds of which a few toxins have been studied in detail. Among the most prevalent diseases with which cigarette smoking has been implicated are cancers, chronic obstructive lung disease and atherosclerotic cardiovascular disease.

In the respiratory tract smoking predisposes to malignant conditions like lung cancer, laryngeal cancer and non-malignant conditions like COPD, sleep apnea, eosinophilic granuloma of lung, respiratory bronchiolitis, Goodpature syndrome and pneumothorax.

Smoking is a major risk factor for COPD. The risk of COPD is increased 30-fold in smokers. Nearly all patients with clinically significant emphysema are smokers.

Smoking is the major risk factor for the development of chronic bronchitis.

SPIROMETRY

Spirometry is a simple method for studying the pulmonary function by recording the volume of air movement into and out of the lungs. In this test, the subject inhales maximally to total lung capacity (TCL) and then exhales as rapidly and forcefully as possible into the turbine of spirometer, which calculates the flow rates and volume measurements. The flow rates can be calculated from the 'spirogram' which is a plot of volume versus time, and the volume can be calculated from the ' Flow – volume tracings' which is a plot of airflow versus the expired or inspired lung volume.

FLOW RATES

Forced vital capacity is the maximal volume of gas which can be expired from the lungs during a forced expiration from a position of full inspiration. The FVC can be subdivided into forced expiratory volume in first second (FEV_1), in the second second (FEV_2), and in the third second (FEV_3). So, FEV_1, represents the integrated flow over first second of expiration and reflects airway narrowing during expiration. It is effort independent and normally 70% - 80% of the FVC is expired in the first second. This is a useful method to analyze

the airway obstruction that occurs in varied pathological conditions of lung.

A more sensitive means of evaluating airway obstruction is, to express the forced expired volume as percent of vital capacity, abbreviated as FEV_1/FVC. This ratio is relatively independent of patients restriction of lung volumes. Normally it is 70% or greater.

Another way of assessing airflow obstruction is to measure specific flow rates. Peak expiratory flow rate (PEFR) is defined as the maximum flow achievable from a forced expiration starting at full inspiration with an open glottis. It measures the maximum expiratory flow rate over the first ten milliseconds. PEFR is reduced in large airway narrowing due to asthma, COPD, vocal cord palsy and expiratory muscle weakness etc.

Flow from 25 to 75 percent of the total FVC, termed $FEF_{25\%-75\%}$ is another measurement which was originally called as Maximal mid-expiratory flow rate (MMER). This is effort – independent and is a very sensitive to airflow obstruction in peripheral, small airways, where disease of chronic airflow obstruction are thought to begin. $FEF_{25\%-75\%}$ is independent of FVC, in otherwords, it is the average flow rate during the middle two quarters of the FVC.

FLOW VOLUMES

Flow volume curve, a recording during spirometry, of the expiratory flow plotted against expired volume, instead of time, resembles a triangular shaped envelope. At the point where 25% of vital capacity has been exhaled, this flow rate is termed Vmax25 or $FEF_{25\%}$, when 50% of the vital capacity has been exhaled is is termed $Vmax_{50}$ or $FEF_{50\%}$ and at 75% of vital capacity it is $Vmax_{75}$ or $FEF_{75\%}$

The inspiratory portion of the curve is helpful in distinguishing large airway obstruction which occurs above the level of the thoracic inlet from obstruction that occur below this level. Large airway obstruction above the thoracic inlet results in a plateau of the flow rate on the inspiratory portion of the curve, while the expiratory portion in affected when the flow limiting portion is within the thoracic cavity.

$FEF_{75\%}$ is thought to be very sensitive to detect early small airway obstruction. In early small airway disease, the only abnormality detected may be reduced $FEF_{75\%}$ and $FEF_{50\%}$ with normal PEFR and FEV_1.

PATTERNS OF ABNORMAL VENTILATORY CAPACITY

	FEV_1	FVC	FEV_1 /FVC

Obstructive	↓↓	↓/ N	↓
Restrictive	↓	↓↓	↑/ N

Smoking being the most important risk factor for COPD, this study was undertaken to assess the pulmonary dysfunction in asymptomatic smokers, so that early intervention by smoking cessation would reduce the disease burden of COPD.

AIM OF STUDY

1. To evaluate the pulmonary function test parameters in asymptomatic smokers.
2. To compare the spirometric findings in asymptomatic smokers to their expected values.
3. To identify the asymptomatic COPD among smokers, so that cessation of smoking would halt their progression.
4. To identify the degrees of deterioration in PFT.
5. To correlate the dysfunction with the quantum of smoking.
6. To look into gender differences in the progression of disease.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

Chronic obstructive pulmonary disease (COPD) has been defined by the Global Initiative for Chronic Obstructive Lung Disease (GOLD) as a disease state characterized by airflow limitation that is not fully reversible.

By 1964, the Advisory committee to the surgeon general of the unitedstates concluded that cigarette smoking was a major risk factor for mortality from COPD. Subsequent longitudinal studies have shown accelerated decline in the volume of air exhaled within first second of the forced expiratory maneuver (FEV_1) in a dose dependent manner to the intensity of cigarette smoking, which is typically expressed in pack years (average number of packs of cigarette smoked per day multiplied by total number of years of smoking).

This dose – response relationship between reduced pulmonary function and smoking intensity accounts for higher prevalence rate of COPD with increasing age. The historical higher rate of smoking among male is the likely explanation for higher prevalence of COPD among male. However the prevalence rate of COPD among women is

increasing as the gender gap in smoking rates is diminishing globally in the past 50 years.

COPD includes all of the following clinical labels namely,[13]

1. Chronic bronchitis
2. Emphysema
3. Chronic airway obstruction
4. Chronic non-specific lung disease
5. Non-reversible obstructive airways disease
6. Small airway disease(obliterative bronchiolitis)
7. Cases of chronic asthma with fixed airway obstruction

INDIAN SCENARIO

In India COPD is the second most common lung disorder, the first being pulmonary tuberculosis. COPD most commonly affects people of middle age. It is rare in patients < 35 years of age. In India male are affected more than the female. This could be due to difference in smoking habits among the Indian population. The prevalence of the disease in equal in both urban and rural population. In rural areas disease prevalence is mainly due to unchecked smoking practices among the uneducated people, especially smoking

beedies. In urban area, smoking with superimposed exposure to dust, fumes and industrial particles predispose to COPD.

DEFINITION

The American Thoracic society defines chronic obstructive pulmonary disease as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; Airway obstruction is generally progressive, may be accompanied by airflow hyperreactivity, and may be viewed as partially reversible.

COPD is usually diagnosed by demonstration of irreversible airflow limitation. Most groups such as the American Thoracic Society (1987), and the European Respiratory Society (1995), specifically excluded asthma. However American Thoracic Society (1995) guidelines included asthma with an irreversible component within COPD. Some studies mean COPD as smoking related chronic airway disorder[2,3]. While others include all disorders causing chronic airway obstruction (eg. Bronchiectasis and obliterative bronchiolitis).[4,5]

Recent data has suggested that COPD is a complex and heterogenous disorder with a number of different

pathological processes leading to recognition of subgroups that may have their own characterisations and natural history.[6]

The Dutch hypothesis originally put forward by Orie et al proposed a unitary hypothesis for all chronic airway disorders which included chronic asthma and COPD were due to constitutional disturbance, airway hyper-responsiveness (AHR) and atopy.[7] However, as most workers considered asthma and COPD are different entities and COPD was considered to be due to an exogenous factor i.e. smoking, Dutch hypothesis was not accepted.

Although the attributable risk of tobacco smoking to COPD is about 85-90%[8], only 15% of smokers develop COPD. Polymorphism for the genes controlling xenobiotic metabolism, hence oxidant-antioxidant balance may explain this susceptibility.[9,10] About 10-15% of COPD have been thought due to other risk factors like environmental tobacco smoke (ETS), occupational exposures and genetic factors. Causal association between COPD and passive smoking requires additional evidence, as the magnitude of association is small.[8]

Recently COPD is defined more clearly using the following clinical diagnostic criteria.[11]

1. History of chronic progressive symptoms like cough and / or wheeze and /or breathlessness.

2. Objective evidence of airway obstruction i.e. FEV1 <80% predicted and FEV1/FVC <70% which is irreversible.
3. Usually a smoking history of more than 20 pack years.

Milder forms of chronic bronchitis may be relatively asymptomatic with only cough due to mucus production and normal airflow called "chronic simple bronchitis".[12] Most patients with chronic bronchitis do not have airflow obstruction and hence do not have COPD. However 10-15% of smokers are susceptible to more rapid decline in airflow than normal.

Emphysema is defined in anatomic terms – abnormal permanent enlargement of gas exchanging units of the lung in association with destruction of alveolar walls and without obvious fibrosis. The predominant physiological consequences of these anatomical abnormality is a decrease in elastic recoil of the lung – which in turn causes outward displacement of the chest wall, flattening of the diaphragm, hyper inflation of the lungs and increased resistance to airflow due to circumferential traction on the small airways by the over distended lung.

As a rule clinicians believe that they can identify most patients with atopic or extrinsic asthma that begins in childhood and those with intrinsic asthma of middle

age. The characteristic features are reversibility of airflow obstruction and hyperresponsiveness of the airways to diverse inciting influences. In asthma the larger bronchi are predominantly affected, goblet cells undergo hyperplasia, bronchial glands hypertrophy, the basement membrane thickens, airway smooth muscle hypertrophies and the walls and lumina are invaded by eosinophils.

Localized bronchiectasis is often present in patients with COPD. As a rule, however, bronchiectasis is characterized by dilatation of airways rather than narrowing. Bronchiectasis does not contribute to obstruction of airflow. Characteristically, proximal airways are dilated and distal airways are obstructed by mucopurulent exudate and by narrowed obliterated terminal airways.

In a recent study O'Brien and colleagues evaluated cases admitted as acute exacerbation of COPD with full lung function and high resolution CT after they were clinically stable. An interesting finding of the study was that evidence of bronchiectasis was present in 29% of the cases suggesting that CT scanning may play an important role in defining the exact nature and subgroup of COPD.

Obliterative bronchiolitis or constrictive bronchiolitis is a specific cause of chronic airway limitation and is due to small airway obstruction. The term small airway

disease was coined by Hogg et al [14] and consists of involvement of the peripheral airways. Small airways are those less than 3 mms in diameter most of which are respiratory bronchioles. They have a large cross sectional area and hence contribute very little to airways resistance, also called the silent zone of the lung.

A recent epidemiological study[13] on prevalence of COPD in spain used spirometric criteria for diagnosis. Irreversible airway obstruction was seen in majority of the cases (74 .1%), though 21.7% of cases had good reversibility of airway obstruction after bronchodilator test. The prevalence is reported as 15% in smokers, 12.8% in ex-smokers and 4 .1% in non-smokers.

RISK FACTORS

There are numerous risk factors for COPD. It includes, increasing age[27], male gender, smoking habits, environmental pollution, socioeconomic status, genetic factors like alpha1 antitrypsin defieciency, birth weight and childhood respiratory illness, recurrent bronchopulmonary infections. Of these smoking is associated with 85-90% of COPD cases. Yet three points about disease occurrence is to be stressed. First, with exception of smoking and alpha1 antitrypsin deficiency, the mechanism by which the risk factors cause COPD is not at all clear. Second, even with smoking which carries the highest

risk, it is not possible to predict which person will actually develop COPD. Third, even though smoking and alpha1 antitrypsin deficiency dominate as risk factors, there are definitely other risk factors for developing COPD. Low socioeconomic status is one such risk factor[22].

EPIDEMIOLOGY

COPD occurs worldwide, but is a major health problem principally in societies where cigarette smoking is common and average lifespan extend into the fifth decade and beyond. In western population it is the fourth most common cause of death and the death percent accounted for about 4.5% of all deaths.

In 1995, in the developed countries 2 million death were due to tobacco and in developing countries it could be 1 million. By 2020 it is likely to increase to 3 million and 7 million in developed and developing countries respectively [24].

In a meta analysis by Halbert RJ et al[30,] the prevalence of COPD was 7.5% , the prevalence of chronic bronchitis was 6.4% and that of emphysema was 1.8%. prevalence of COPD by spirometric estimates was 8.9%.

SMOKING AND DISEASE PATTERN

Smoking usually begins for psychological reasons, such as curiosity, parental smoking, peer pressure, rebelliousness, and assertion of independence.

Pattern of smoking is different in developing and developed countries.

In developing countries over 50% men smoke while only 10% of women smoke, compared to developed countries where 25-30% of both men and women smoke.

SMOKING AND DISEASE ASSOCIATIONS

Smoking increases the absolute number of deaths from lung cancer, cancer of other respiratory sites, chronic bronchitis/ emphysema and cor pulmonale. Death from lung cancer is 8-25 times more common among smokers with a clear dose-response relationship

Death from IHD and cerebrovascular disease are advanced by smoking without an increase in absolute number.

. Smoking is also associated with gastro intestinal diseases like peptic ulcer disease, gastro esophageal reflux, chronic pancreatitis, crohns disease, colonic adenomas.

It can cause reproductive disease like ovarian failure, decreased sperm quality, pregnancy related events like spontaneous abortion, prematurity, fetal effects like low birth weight, SIDS, impaired lung growth, atopy and asthma.

It can also be related to renal diseases like glomerulonephritis, and others such as benign prostatic hypertrophy, cataract and peripheral vascular disease.

SMOKING AND COPD

Nearly all patients with clinically significant emphysema are smokers. Smoking is also a major risk factor for chronic bronchitis. Heavy smokers are at greater risk of developing COPD, than moderate smokers. Low exposures to cigarette smoke as encountered during passive smoking also seem to be harmful.

Fletcher and Peto[16] showed that smoking was associated with irreversible obstructive changes in airways in some subjects while not in others.

Sandvik et al[17] described the long term effects of smoking on pulmonary function through his longitudinal

study in 1393 middle aged Norwegian males and followed up the patients for about 7 years. The study stated that smoking is definitely associated with increased risk of COPD. But other factors also determine its predisposition.

Marcus et al[18] described that smoking causes decline in pulmonary function in dose dependent manner and cessation of smoking improves the obstructive pattern.

On an average, the rate of expiratory airflow in smokers decrease twice as fast in smokers (40 ml per year) as in non- smokers(20ml per year). [19]

Nonetheless, most smokers do not develop symptomatic COPD, probably because of the large physiological reserve of the lung. However some smokers (15%) do experience accelerated loss of lung function and develop symptomatic disease. The basis of the heightened susceptibility in this group is not known.

The degree of cough, phlegm and wheeze are directly related to number of cigarette smoked daily. The tar yield in the cigarette is related to the volume of sputum production.[20]

Smoking in early stages apart from causing cough and wheeze, reduces the exercise tolerance. Reduction in

exercise tolerance is aggravated by high levels of carboxyhemoglobin [21]

PATHOPHYSIOLOGY

Many pulmonary function abnormalities occur in COPD. Increases in residual volume, and the residual volume to lung capacity ratio, non uniform distribution of ventilation and ventilation perfusion mismatch are also typical findings but, persistent reduction in forced expiratory flow rates is the most typical finding. Reduction in FEV1 and FEV1/FVC are the characteristic physiological abnormalities of COPD.

Airflow during forced exhalation is the result of balance between the elastic recoil of the lungs promoting the flow and the resistance of the airways limiting the flow. The relative contribution of diminished elastic recoil and increased resistance in reducing maximal expiratory airflow can be quantified from the flow – pressure curves. With decreased elastic recoil, the curve has a normal slope, but it terminates prematurely. In contrast, with increased airway resistance the slope becomes less steep, reflecting the necessity for increased driving pressure for any level of airflow.

Though there is considerable variability in the relationship between the fev1 and other physiological

abnormalities in COPD, certain generalisations can be made. The arterial Po_2 (Pao_2), usually remains near normal until the FEV1 is decreased to about half of the predicted value. Even a much lower FEV1 may be associated with normal Pao_2, atleast at rest. An elevation of Pa_{co2} is not expected in COPD until the FEV1 is less than one-fourth of the predicted value.

PATHOGENESIS

COPD is characterized by mild chronic inflammation throughout the airways, lung parenchyma and pulmonary vasculature.[22]

Various proposed hypothesis for COPD are

1. Inflammatory repair hypothesis
2. Proteinase anti-proteinase hypothesis

INFLAMMATORY REPAIR HYPOTHESIS

Cigarette smoke is made up of 92% vapourised chemicals and 8% particulate materials**.** These chemicals and particulate materials, attract the various inflammatory cells that play an important role in the pathogenesis**.**

Neutrophils, macrophages and CD8+ lymphocytes are the major cells involved in the inflammatory response in COPD patients. Smoking plays a major role in producing such inflammatory reaction in the airways and lung parenchyma.

Smoking recruits macrophages, neutrophils and lymphocytes and these cells are increased in various parts of the lung. The inflammatory cells get activated and initiate the inflammatory process through various mediators.

Activated inflammatory cells release a variety of mediators including Leukotriene B4, IL-8, TNF and others capable of damaging lung structure or sustaining neutrophil inflammation.

Any stimulus that increases either the number of leukocytes (neutrophils, macrophages) in lung or release their elastase containing granules, increase the elastolytic activity thereby triggering a chronic inflammatory response in the lung.

Stimulated neutrophils also release oxygen free radicals that inhibit alpha1 antitrypsin. With low levels of alpha1AT, the elastic tissue destruction is unchecked leading to emphysema, a form of chronic obstructive pulmonary disease.

In smokers, neutrophils and macrophages accumulate in the alveoli and pathogenesis may involves the following processes.

- Direct chemoattractant effects of nicotine and also due to the effects reactive oxygen species in smoke

- Accumalated neutrophils are activated and release their granules rich in variety of their cellular proteases (Neutrophil elastase, Proteinase 3, Cathepsin G).

- Various cellular proteinases released cause tissue destruction.

- Smoking enhance the elastase activity of the macrophages.

- Macrophage elastases are not inhibited by alpha 1 AT and hence the elastase activity is unchecked causing tissue destruction.

- Matrix metalloproteinases from neutrophils and macrophages also play an important role in tissue destruction.

- Oxidant and anti-oxidant balance is essential and smoking disturbs it causing an imbalance, once again predisposing to tissue destruction.

- Normal lung contain a lot of anti-oxidants like SOD, Glutathione that keep oxidative damage at minimum.

- Tobacco smoke contain an abundant reactive oxygen species (Free radicals) that deplete these anti-oxidant mechanism thereby inciting tissue damage.

- Secondary consequence of oxidative injury is inactivation of native anti - proteases resulting in functional alpha1 anti- trypsin deficiency even in patients without enzyme deficiency.

PROTEINASE ANTI-PROTEINASE HYPOTHESIS

According to this theory there is steady and episodic release of proteolytic enzymes into the lung parenchyma. Normally, plasma proteinase inhibitors, especially alpha1 anti-trypsin, permeate lung tissue and prevent the proteolytic enzymes from digesting the structural proteins of the lung. The uninhibited activity of proteinases cause destruction of the Elastin in the lung parenchyma which is the key event in the development of emphysema. Elastin is the principal component of elastic fibers, which are prominent part of lung's extra cellular matrix.

Elastin is secreted as a soluble protein of 60 to 70 kDa called tropoelastin, encoded by gene on chromosome 7. Tropoelastin molecules are deposited into the extra cellular space and aligned on a scaffolding of microfilaments consisting of number of proteins like fibrillin, microfibril associated

protein. Elastin synthesis in the lung begins in the late fetal life and stops in adult life. Destruction of the lung elastin plays an important role in the pathogenesis of emphysema.

COMPLICATIONS

- Secondary erythrocytosis
- Recurrent acute exacerbations
- Pneumothorax
- Chronic and acute on chronic respiratory failure
- Pulmonary hypertension
- Chronic Cor Pulmonale
- Right ventricular failure
- Weight loss

TREATMENT FOR ACUTE EXACERBATION

1. Oxygen therapy

2. Bronchodilator- nebulised salbutamol (5mg) or terbutaline (10mg) + ipratropium 500µg

3. Corticosteroids – inhaled, in severe cases parenteral steroids.

4. Diuretics

5. Antibiotics

6. Respiratory stimulants like Doxapram

7. Chest physiotherapy

8. Mechanical ventilator – Non invasive positive pressure ventilation is the procedure of choice

TREATMENT FOR CHRONIC COPD

1. Smoking cessation

2. Avoid dusty environment

3. Bronchodilator

4. Steroids

5. Theophylline or aminophylline

6. Cilomilast[31] is an orally active phospho diesterase inhibitor-4 and is effective in reducing the exacerbations and maintaining lung function.

SMOKING CESSATION

Nicotine is the active psychopharmaceutical drug contained in the leaves of tobacco plant. Nicotine is a potent euphoriant. On a molar basis nicotine is more active than

euphoria inducing drugs such as cocaine, amphetamine and morphine.

Nicotine has number of other effects on CNS. It may improve task performance and attention time, measurably in non habituated subjects. It may also alleviate anxiety and depression and induce a sense of well being, while causing a state of arousal. Unfortunately nicotine is also an addicting substance.

Smoking is a major public health problem. Health hazards attributable to smoking parallel smoking prevalence. Hence smoking cessation is very important for reducing the disease burden of COPD which is the fourth most common cause of death worldwide. Smoking cessation can be attained by

1. **Non pharmacological methods**
2. **Pharmacological**

Non pharmacological methods involve various behavioral techniques.

PATIENT EDUCATION – If smokers could be educated about health hazards of cigarette smoking, they could theoretically become motivated and psychologically empowered to quit. Unfortunately, the anticipated benefits were not obtained. 80% smokers indicate that they want to quit but could not.

HYPNOSIS is to enable the smokers to achieve an altered state of consciousness that enhances the ability to quit. However, it is of low reliability with published qui rates between 0 and 88%.

ADVERSIVE CONDITIONING is extinguishing smoking by associating it with negative sensation like electric shock, nausea inducing drugs, hot, smoky air treatment. It is of limited value.

GROUP COUNSELING offered by several commercial and voluntary health organizations. It typically include lectures, group interaction, some form of tapering method like quit a day development of coping skills. This method is largely limited to cities and one year success rate ranges between 15-35%.

PHARMACOLOGICAL METHODS

It includes tranquilizers, antidepressants, topical anaesthetics, anti anxiety agents, anti cholenergics, clonidine and nicotine replacement therapy.

Lobeline sulphate is pharmacologically similar to nicotine and was selected as stop-smoking drug. Interest in it has declined but recently sublingual formulation are being tested.

Amphetamine is similar to nicotine and cause euphoria. Once thought of promising drug, now proved to be of little value.

Bupropion, originally developed as anti depressent is very useful in nicotine deaddiction.

Nicotine replacement therapies

Nicotine replacement therapy dates back to 1950s in the form of lozenges. Today there are several forms of nicotine replacement therapy available, including nicotine polacrilex, Transdermal nicotine, nicotine nasal spray, nicotine vapours and aerosols, and nicotine toothpicks. Two widely adopted therapies are

1. Nicotine polacrilex - Nicotine is bound to a resin and is available in two doses of 2mg and 4mg. Blood nicotine levels are less than 40% associated with customary smoking. It is proved to be effective in reducing the nicotine withdrawal symptoms.
2. Transdermal nicotine – primary advantage of transdermal delivery system is ease of use and controlled drug delivery. They reduce withdrawal symptoms and studies have proved that abstinence associated with transdermal patch is double that of placebo.[26]

GUIDELINES FOR ENDOTRACHEAL INTUBATION

1. Persistent hypoxemia with failure to maintain Pao2 55-60mm of Hg or oxygen saturation <88-90% despite maximum medical treatment
2. Worsening respiratory acidosis
3. Signs of progressive respiratory muscle fatigue
4. Deterioration of mental status
5. Inability to protect airway
6. Inability to clear copious secretions.

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PATIENTS AND METHODS

PLACE OF STUDY

This study was conducted in the general medical ward of Government Kilpauk medical college Hospital, Chennai.

PERIOD OF STUDY

From April 2004 to February 2005.

DESIGN

Observational prospective cohort study of patients who are smokers admitted in the hospital for complaints other than respiratory symptoms. A total of 100 patients were included in the study.

METHODOLOGY

A. Subject selection

1. Inclusion criteria

a. Subjects randomly selected from medical ward in age group 20 -60 years.

b. with h/o smoking of atleast one pack year(ie.. > 10 cigarettes or 15 beedies per day for atleast one year).

2. Exclusion criteria

a. Patients who had symptoms as per American Thoracic Society scale for dyspnoea

b. Known case of cardiac disease.

c. Known diabetic patients.

d. Patients with spinal deformity.

e. Age > 60 years

All patients included in the study were subject to thorough clinical examination. All were subject to laboratory investigation as per the proforma.

After the investigations, patients were subjected to spirometry in medical department, kilpauk medical college, Chennai by using **FUKUDA SANGYO** spirometer and results were recorded and analyzed.

FUKUDA SANGYO

The spirometer used was FUKUDA SANGYO. This spirometer can measure and analyze the pulmonary function by means of Fleish type flow sensor and variable reluctance type transducer.

The readings were calculated according to Indian population by a formula devised by Udwadia et al ., which is as follows:

UDWADIA'S FORMULA

VARIABLES	MALE	FEMALE
FEV_1(L)	Adults <30years 0.039×H-0.010 × A-3.266 Adults >30years 0.037×H-0.022× A-2.650	Adults <30years 0.025× H-0.011 × A-1.424 Adults >30years 0.032×H-0.012×A-2.580
FVC(L)	Adults <30years 0.055×H+0.019×A-6.058 Adults >30years 0.054×H-0.018×A-4.832	Adults <30years 0.030×H+0.006×A-2.284 Adults >30years 0.043×H-0.010×A-3.755
PEF	0.085×H-0.0187×A-6.2083	0.0497×H-0.0336×A-0.1399
$FEF_{25-75\%}$	0.0173×H-0.0407×A+1.6108	0.0245×H-0.0336×A0.1399
FEF_{50}	0.0195×H-0.0365×A+1.7383	0.0272×H-0.279×A-0.2704
FEF_{75}	0.0088×H-0.0301×A+1.0402	0.0113×H-0.0288×A+0.5012

A - Age in years

H - Height in cms

Interpretation

The spirometric readings are useful in the interpretation of pattern of pulmonary dysfunction

Various abnormalities are

1. Obstructive pattern

$$\frac{\text{FEV1}}{\text{FVC}} < 70\%$$

2. Restrictive pattern

$$\frac{\text{FEV1}}{\text{FVC}} > 80\%$$

In normal subjects a full VC may be expired in 3 seconds and a slow VC equals FVC. So %FVC is taken for grading restriction.

Normal > 80%

Mild restriction ≥66 - 80%

Moderate restriction ≥50 - 65%

Severe restriction <50%

3. Mixed pattern

When both FEV_1/ FVC < 70% and %FVC< 80% are present.

All datas obtained were statistically analyzed and their significance studied.

However, patients cooperation, standardization of the equipment and the procedure are crucial for any meaningful interpretation. It is also important to calculate the percent predicted values based on regression equations drawn from the population to which the patient belongs. From tropical countries like India there are large number of reports on normal spirometric values which can be used for prediction.

A value less than the lower limit of normal can be considered normal provided the technique is standardized and the measurement well judged.

The technician is required to calibrate the spirometer to a given standard daily before subjecting any patient to spirometric test. Each patient's anthropometric data in terms of age, sex, height, and weight entered in the computer. The machine is corrected for ambient temperature and pressure.

The patient is then connected to the spirometric tubing using a mouthpiece and all leaks around the mouthpiece are seen to be occluded.

Nasal leaks are prevented by occluding the nose with nasal clips.

The patient is then asked to breathe normally in the spirometer circuit for a couple of breathes and then patient is asked to take maximum inspiration and stop. Then patient is instructed to expire forcefully through the mouthpiece.

The computer analyzes these flows and volumes in flow-volume and volume-timed formats to give results comparing them with the normal predicted values. Spirometry can find VC, FVC, FEV_1, FEV_1/FVC, $FEF_{25-75\%}$.

Clinical spirometry detects obstructive lung disease by measuring dynamic flow rates. It identifies whether flow obstruction is present or not, and characterizes whether the obstruction is inspiratory, expiratory or both.

It provides an objective record of the degree of obstruction to aid the prognosis and therapy. It

grantifies the stage of the disease with broad limits of mild, moderate and severe.

General rule, expiratory flow rate < 70% of predicted normal value of FEV_1/FVC ratio is suggestive obstructive lung disease. All the values obtained were statistically analyzed.

GOLD classification of COPD

STAGE	CHARACTERISTICS
0 : at risk	Normal spirometry
I : mild COPD	FEV_1/FVC <70%
	$FEV_1 \geq 80\%$ predicted
II : moderate COPD	FEV_1/FVC <70%
	$30\% \leq FEV_1 < 80\%$ predicted
III: severe COPD	FEV_1/FVC <70%
	FEV_1 < 30% predicted OR presence of respiratory failure OR signs of RV failure.

RESULTS AND ANALYSIS

In this study, 100 patients admitted for other complaints were selected according to inclusion criteria and studied.

DETAILS OF STUDY POPULATION

Total Number of Patients - 100

TABLE –1

Age Distribution

AGE GROUP (YRS)	NUMBER	PERCENTAGE
20 – 29	17	17%
30 – 39	24	24%
40 – 49	29	29%
50 – 60	30	30%
TOTAL	100	

Maximum number of cases studied in age group 50 – 60 (30%) Youngest patient in study group was 20 yrs and the oldest was 60yrs.

Age Distribution

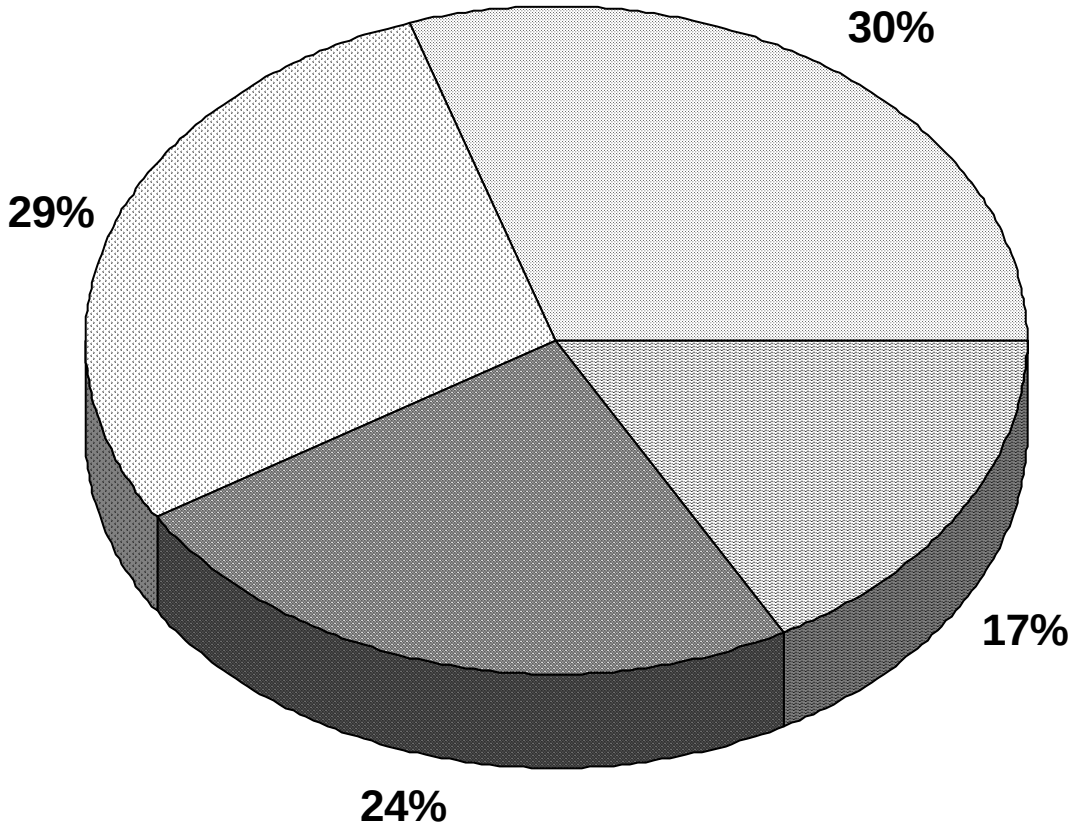
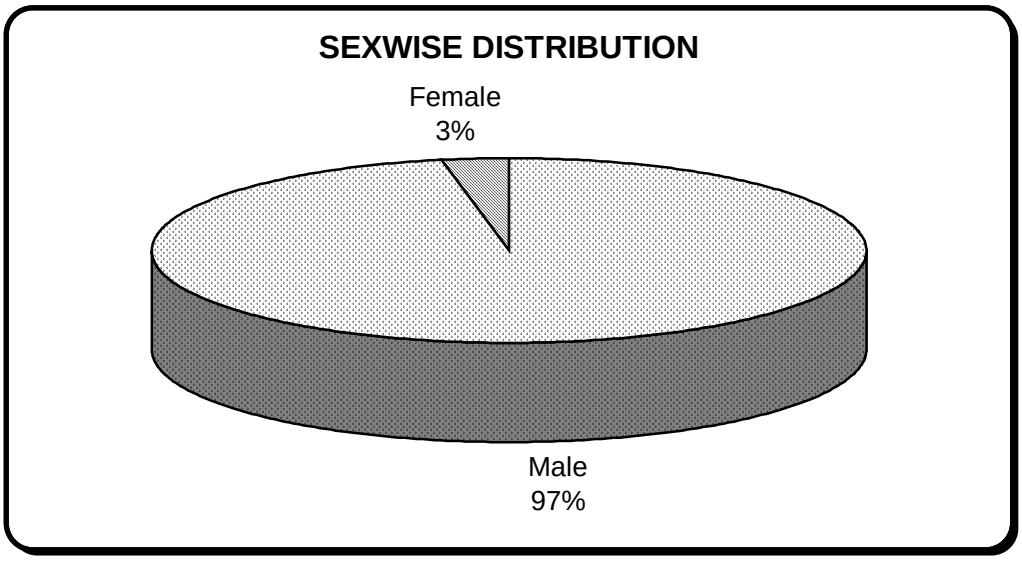


TABLE – 2

SEXWISE DISTRIBUTION

S.No.	Sex	Number	Percentage
1.	Male	97	97%
2.	Female	3	3%

Among the patients 97 were male and remaining were female

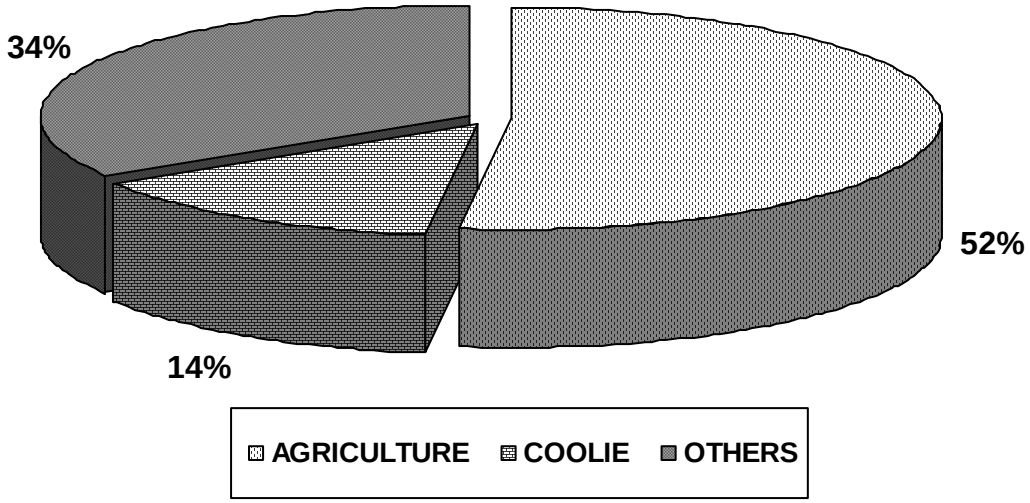


OCCUPATION

Patients of varied occupation were studied . Among them most of them belonged to the group of agricultural workers

TABLE -3

	CASES	**PERCENT**
FARM WORKERS	52	52
MANUAL LABOURERS	14	14
OTHERS	34	34



The remaining (others group) patients comprised of factory workers, mansons, butchers etc.,

TABLE – 4

DURATION OF SMOKING

DURATION IN PACK YRS	CASES	%
0 -5	6	6
5 – 10	38	38
10 – 15	20	20
>15	36	36

Most of the patients (38%) belonged to 5-10 pack years of smoking. Another 36% belonged to > 15 pack years of smoking.

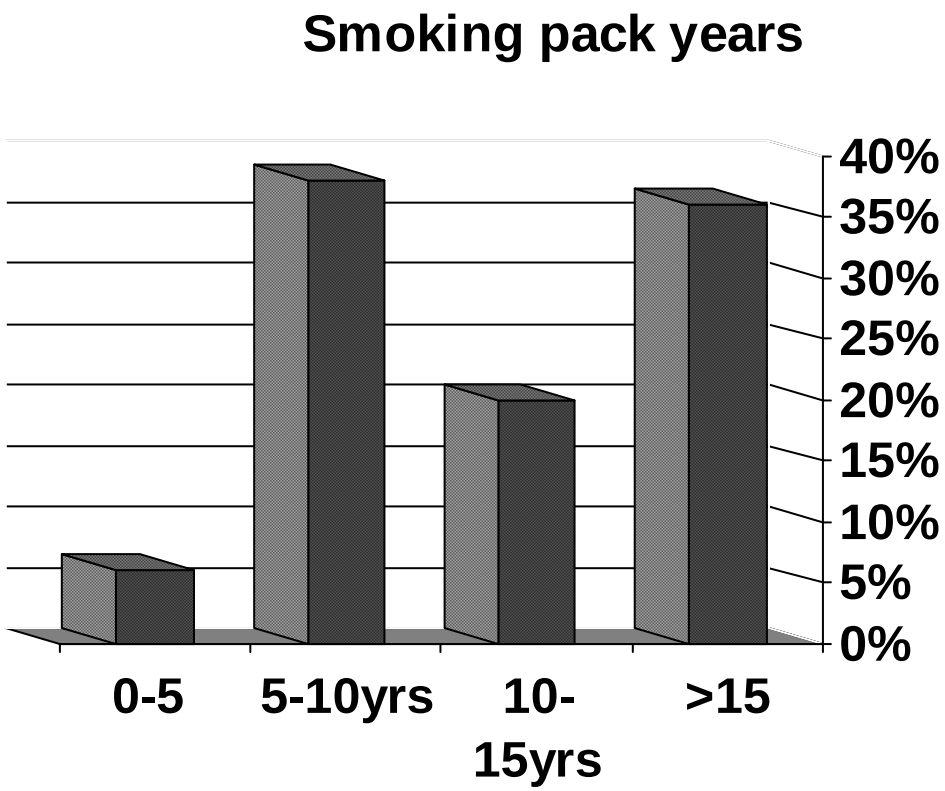

Smoking pack years
40%
35%
30%
25%
20%
15%
10%
5%
0%
0-5
5-10yrs
10-15yrs
>15

TABLE – 5

DISTRIBUTION OF SMOKERS

TYPE	CASES	%
CIGARETTE	10	10
BEEDI	72	72
BOTH	18	18

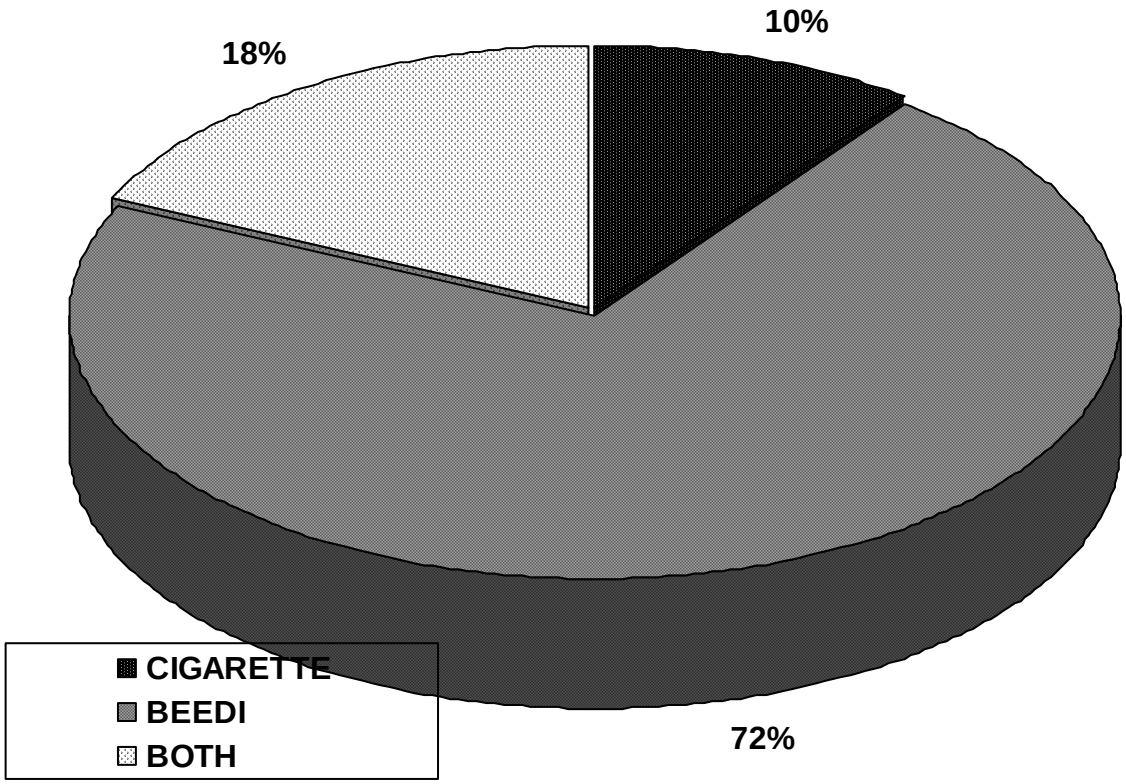
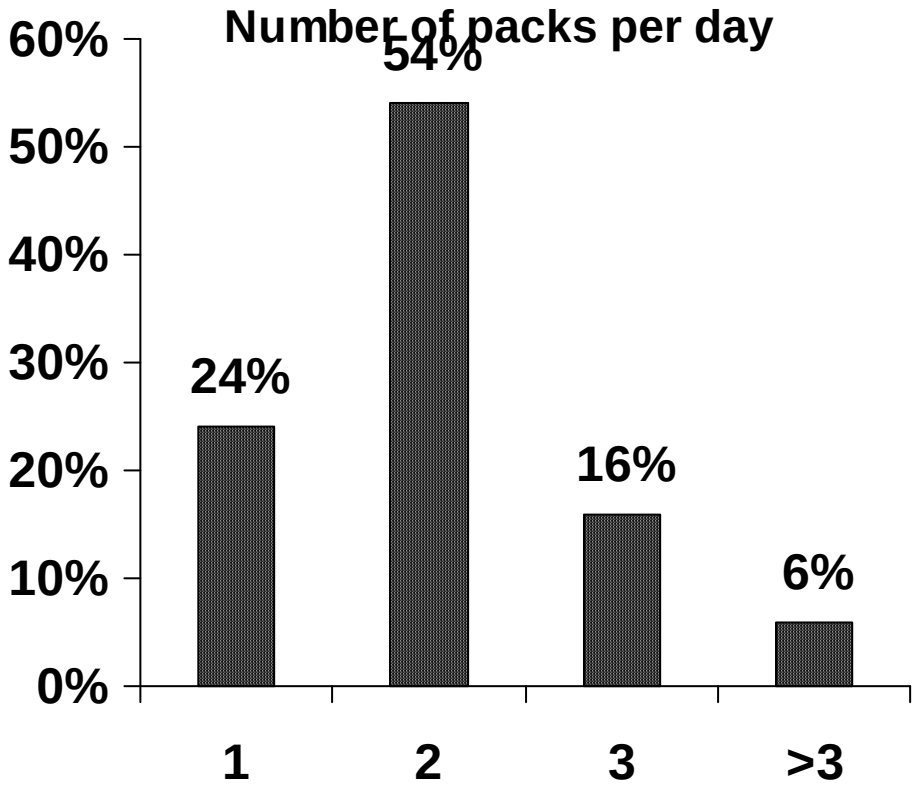


TABLE – 6

BEEDIES CONSUMPTION

NUMBER OF PACKS PER DAY	CASES	%
1	24	24
2	54	54
3	16	16
>3	6	6



Other habits

74% were alcoholic, usually combined with smoking beedies

GENERAL EXAMINATION

The general examination of the patients showed the following abnormalities. Of the 100 cases studied, 38 patients had pallor. Ten patients had clubbing.

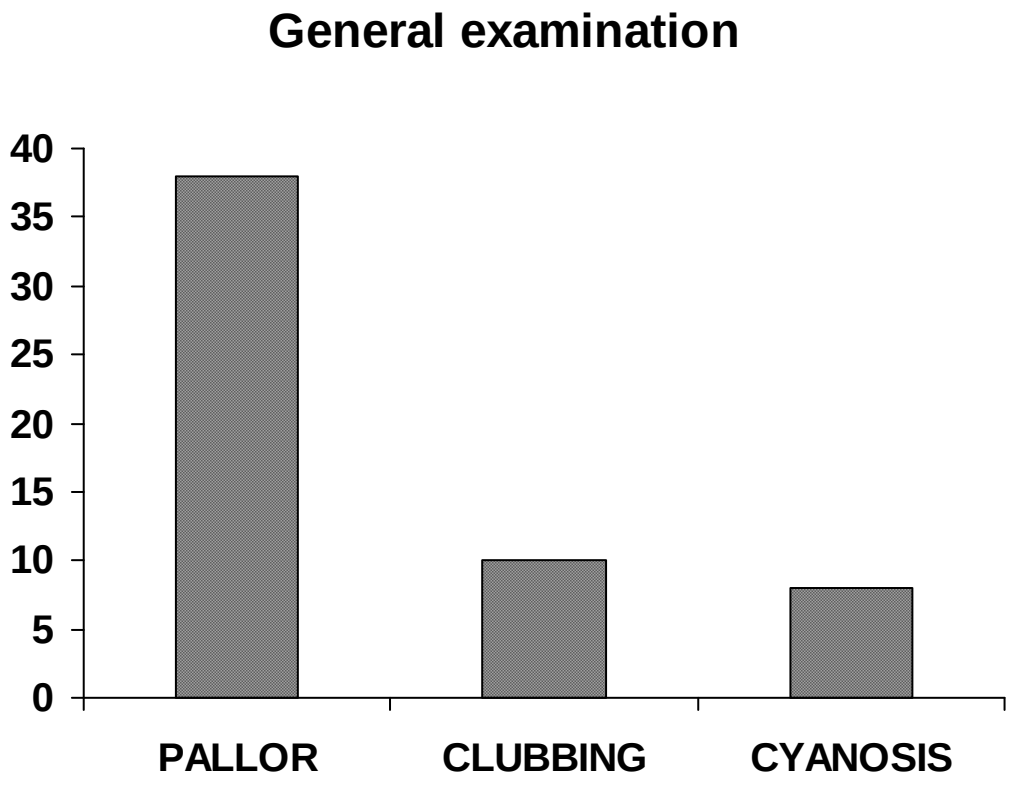


TABLE - 7

RESPIRATORY RATE RANGE

RESPIRATORY RATE	CASES	PERCENT
14 – 18	36	36
19 -22	20	20
>22	24	24

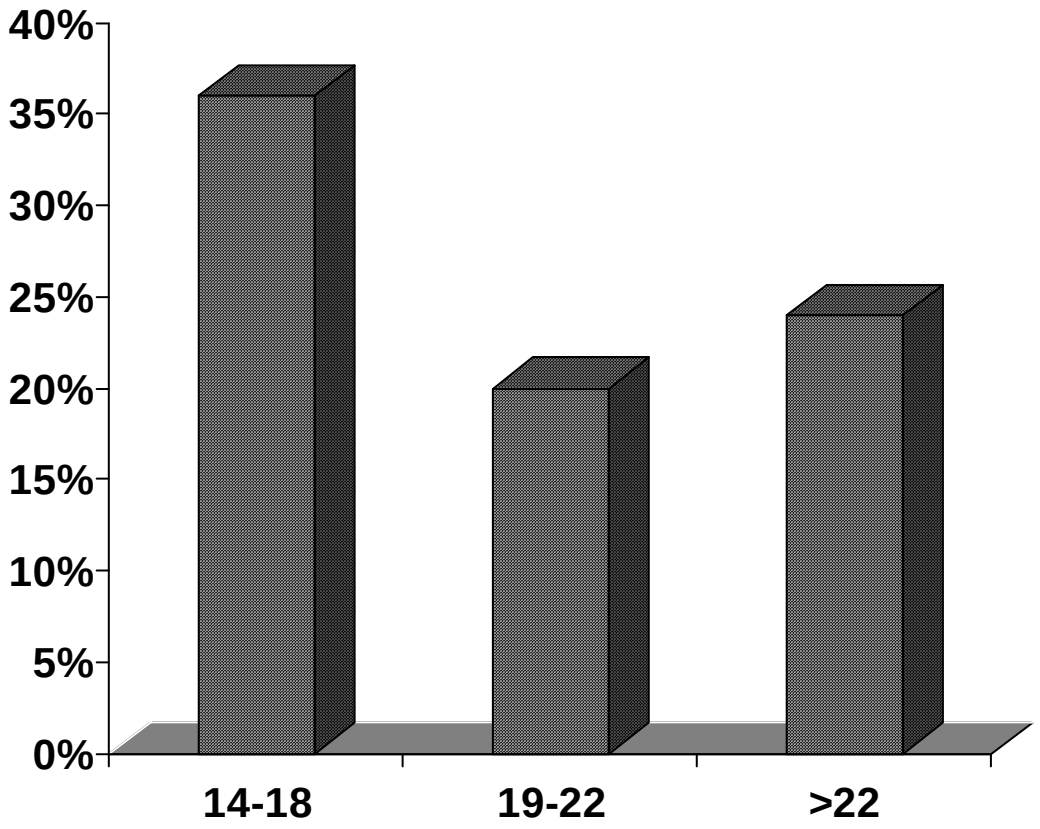


TABLE - 8

RESPIRATORY SYSTEM EXAMINATION

	CASES	**PERCENT**
BARREL SHAPED CHEST	8	8
↓ CHEST MOVEMENT	6	6
↓ BREATH SOUNDS	18	18
RHONCHI	16	16
CREPTS	18	18

TABLE – 9

CHEST CIRCUMFERENCE

CHEST EXPANSION	CASES	PERCENT
2.5 -3.5	32	32
3.5 – 5	58	58
>5	10	10

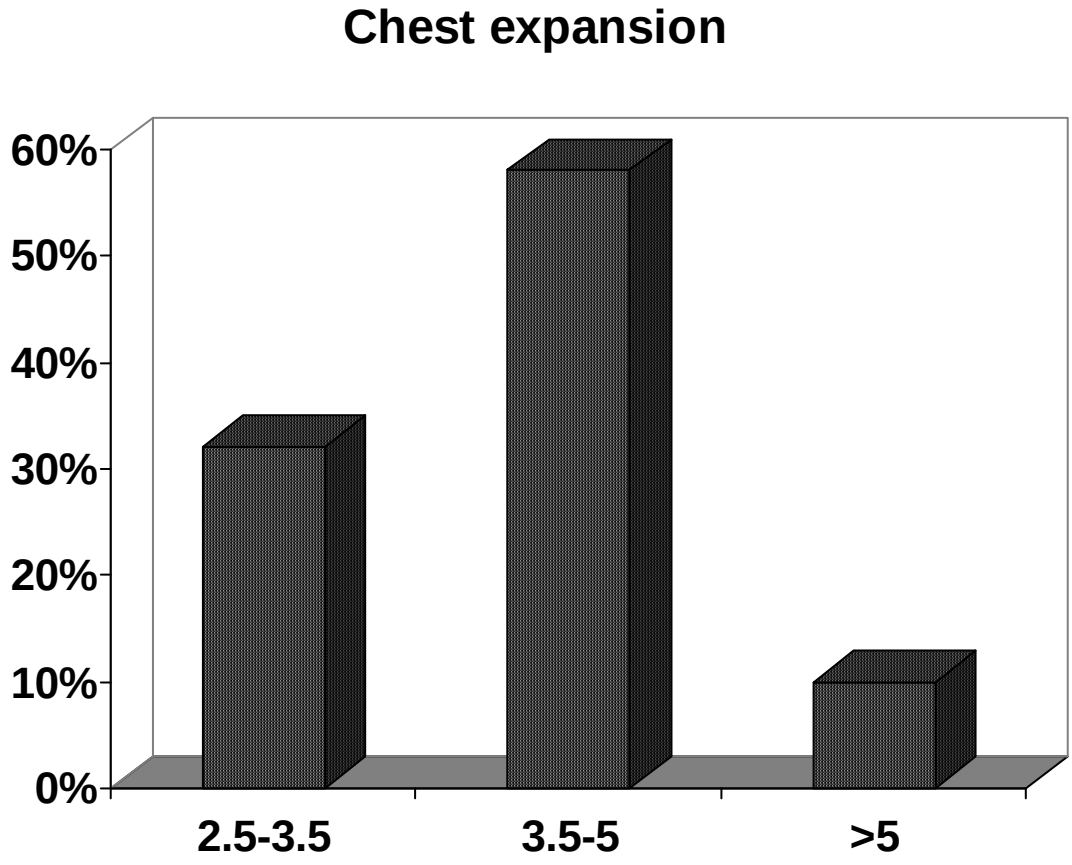


TABLE - 10

HAEMOGLOBIN

Hb%	cases	PERCENT
<10	6	12
10 – 14	40	80
> 14	4	8

TABLE -11

LEUKOCYTE COUNT

TC	CASES	PERCENT
< 6000	1	5
6000 – 10000	60	60
10001 – 14000	36	36
>14000	2	2

ESR VALUES

Out of the 100 cases studied , 42 had ESR 6-10/ hr

58 had 11- 30/ hr

Random blood sugar, blood cholesterol, and serum creatinine were in normal range.

PCV values – none had polycythemia.

CHEST X-RAY

It was normal in most of the patients (94%).

6 patients had early emphysematous changes.

NORMAL EXPECTED SPIROMETRIC VALUES IN INDIA (MEAN + SD)

Male	**15-19**	**20-24**	**25-34**	**35-44**	**45-54**	**>55**
FVC	3.49±0.57	3.65±0.53	3.59±0.54	3.31±0.62	3.14±0.65	2.98±0.67
FEV1	3.04±0.52	3.09±0.51	2.9±0.47	2.6±0.53	2.42±0.42	2.35±0.51
FEV1/FEC%	87.1±11	84.6±7	80.6±9	78.4±10	72.4±10	68.9±9
PEFR	530±72	553±72	559±87	512±98	471±91	451±102

Female	**15-19**	**20-24**	**25-34**	**35-44**	**45-54**	**>55**
FVC	2.46±0.47	2.42±0.52	2.31±0.42	2.24±0.4	1.98±0.44	1.4±0.28
FEV1	2.08±0.42	2.07±0.45	1.85±0.41	1.75±0.54	1.64±0.3	1.23±0.28
FEV1/FEC%	85.4±15	85±17	84.5±12	82.9±12	84±10	87±23
PEFR	450±53	394±62	364±64	360±60	328±67	282±32

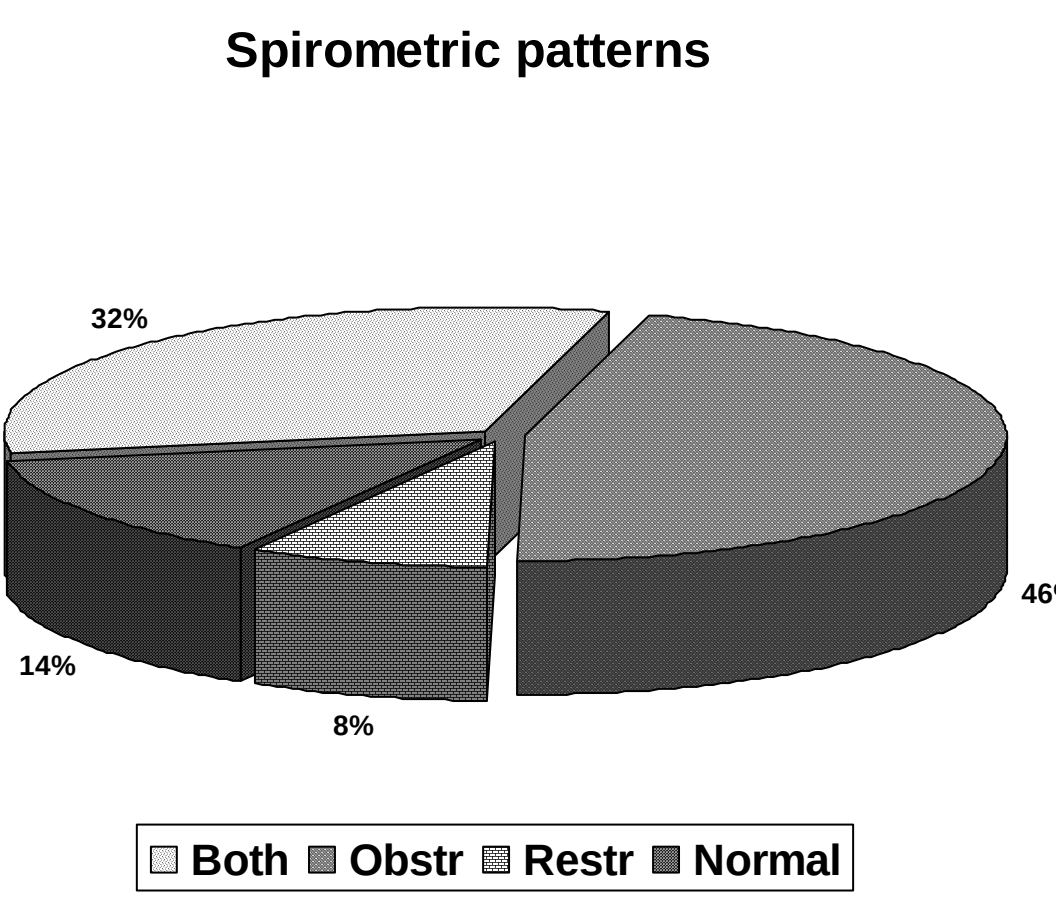
SPIROMETRY

32 Patients showed combined defect (both restrictive and obstructive)

46 Cases showed obstructive pattern

8 cases showed restrictive pattern

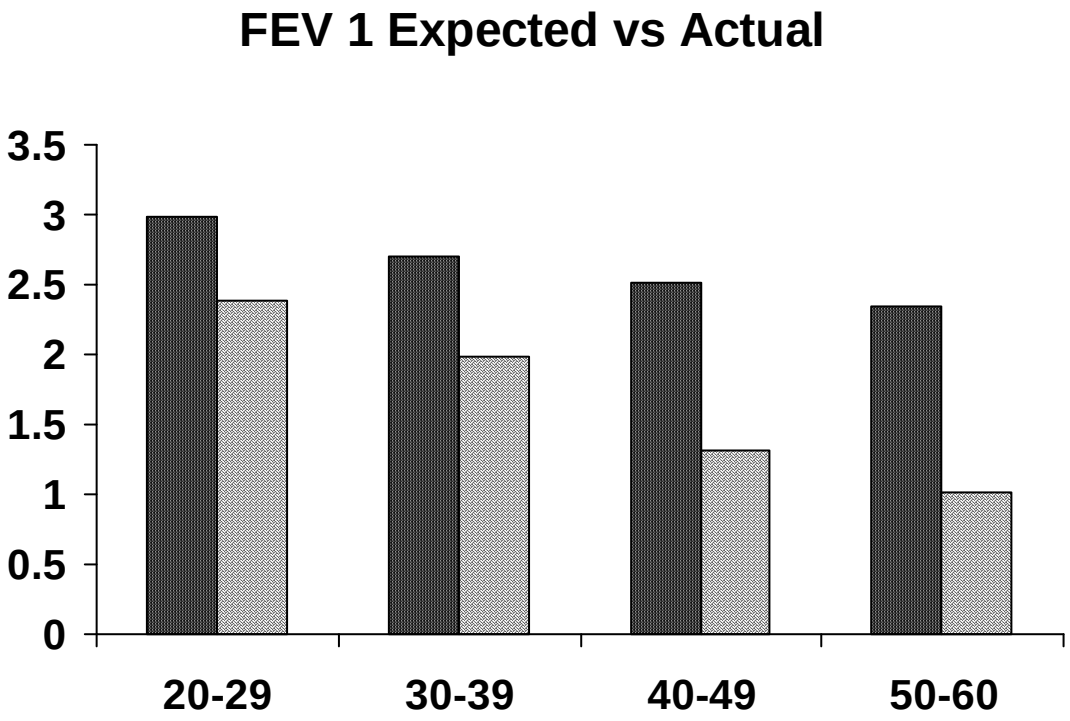
14 showed normal study.



FEV_1 EXPECTED VS ACTUAL

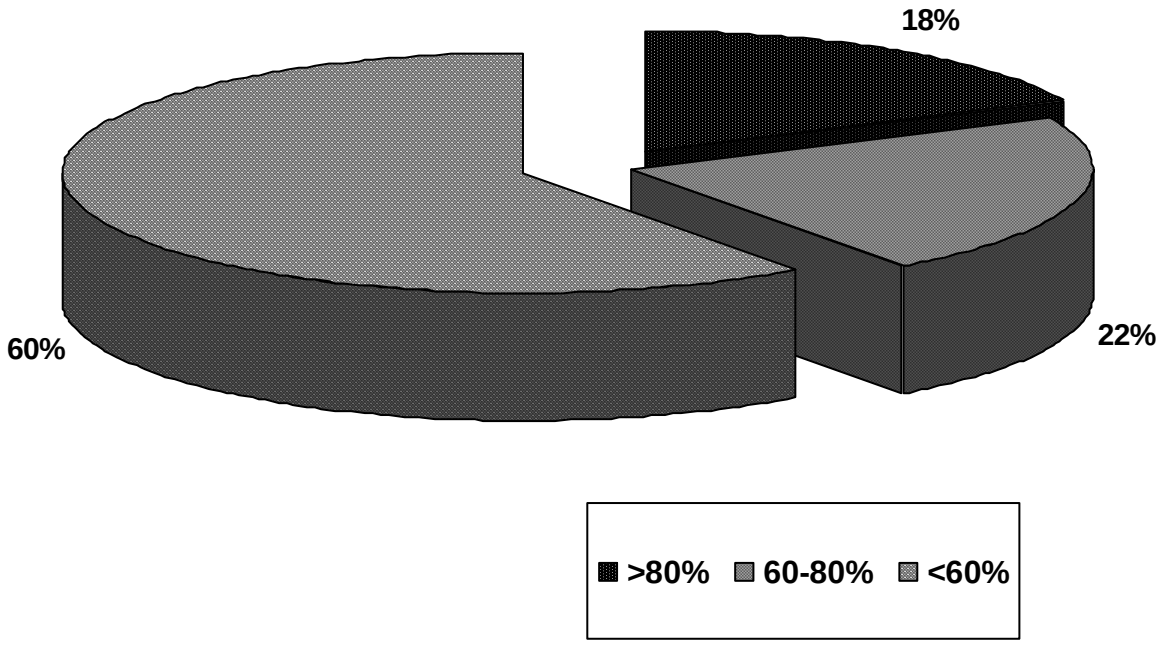
AGE GROUP (YRS)	EXPECTED	ACTUAL	PERCENTAGE
20 – 29	2.99±0.49	2.38	79%
30 – 39	2.7±0.47	1.99	73%
40 – 49	2.51±0.51	1.32	53%
50 – 60	2.34±0.49	1.01	43%
MEAN	2.56	1.67	

The expected FEV_1 among subjects studied was 2.51± 0.42 L, however, the actual mean FEV_1 was 1.67± 0.5 L, 65.2% of the expected value.



FEV_1 %

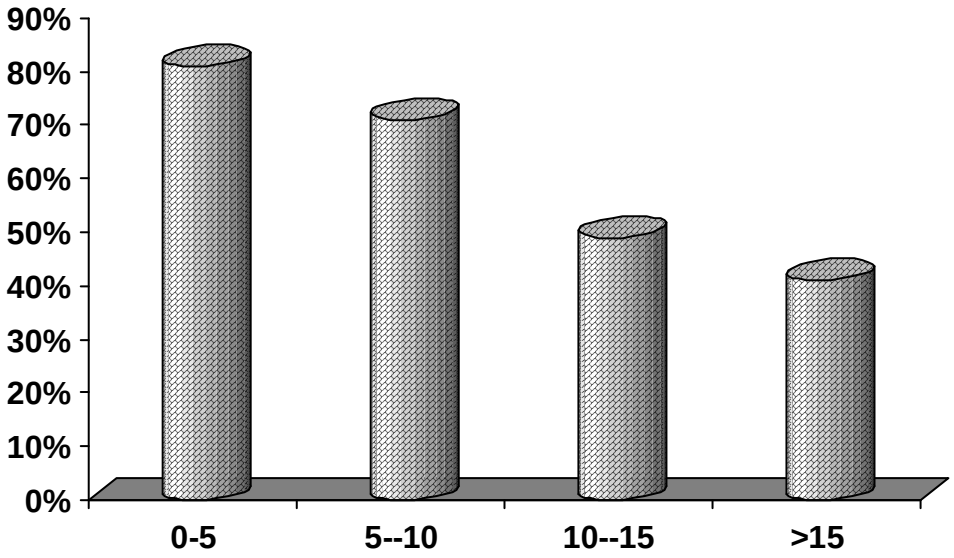
STAGES OF FEV1	CASES	PERCENT
> 80%	18	18
60-80%	22	22
<60%	60	60



FEV_1 % AND PACK YEARS

PACK YEARS	FEV1%
0-5	81%
5-10	71%
10-15	49%

>15	41%

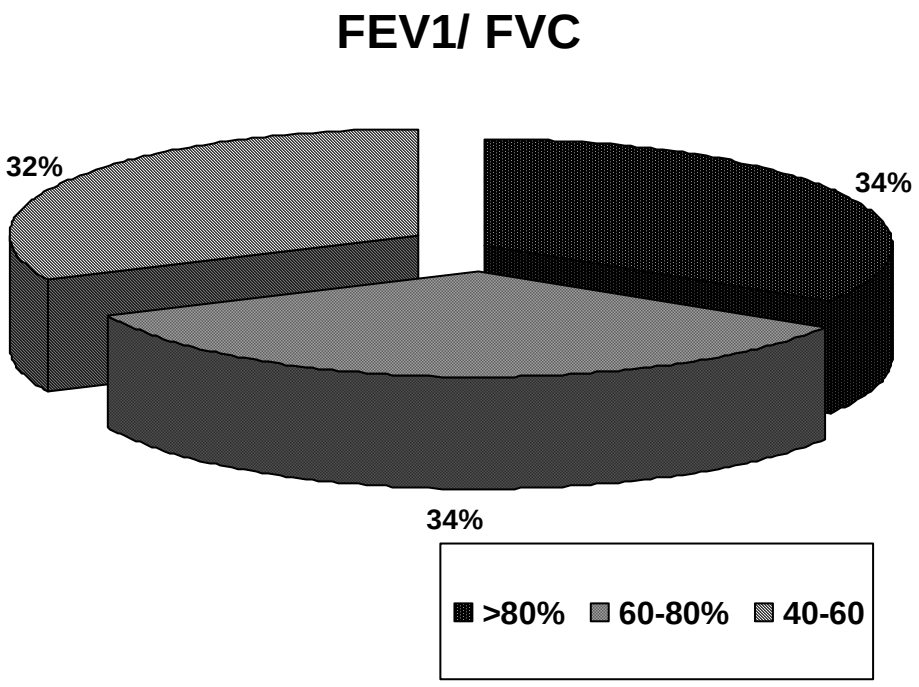


DISTRIBUTION OF CASES ACCORDING TO FEV $_1$/FVC%

FEV1/FVC	CASES	PERCENT
>80	34	34
60 – 80	34	34

40 – 60	32	32
< 40	0	0

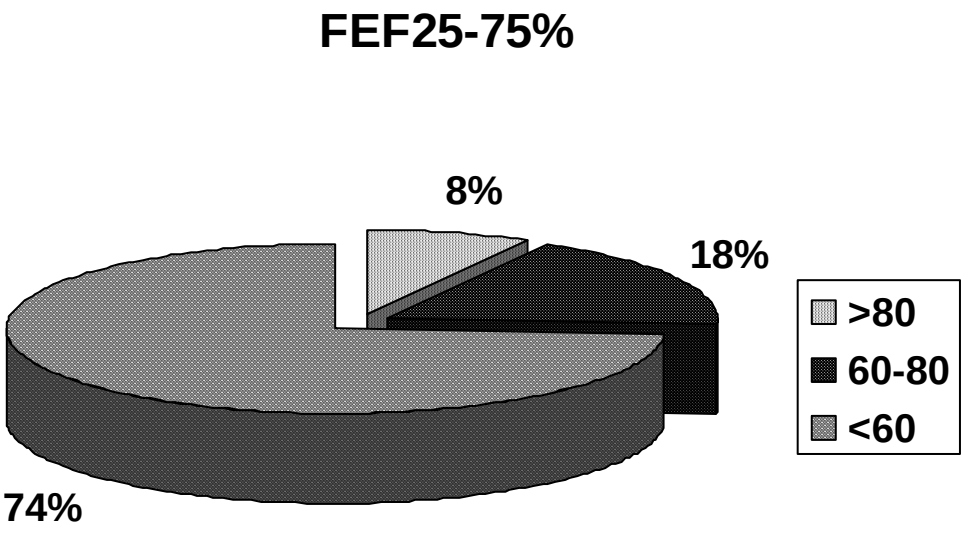
Actual mean FEV1/FVC was 70.49% of the expected value.



DISTRIBUTION OF CASES ACCORDING TO $FEF_{25-75\%}$

VALUES	CASES	PERCENT
> 80	8	8

60 -80	18	18
<60	74	74



$FEF_{25-75\%}$ is a good indicator of small airway disease.

The mean value is 49.04%

GENDER DIFFERENCE

	MALE	**FEMALE**
FEV_1	1.01(39%)	0.92(43%)
FEV_1/FVC	43%	41%

DISCUSSION

In this study, a major group of patients 46% showed obstructive pattern, 32% showed mixed pattern, 8% showed restrictive pattern and 14% had normal lung function.

In a similar study by Zielensky.J et al[27]., spirometric analysis, airway limitation was observed in 20.3% patients and restrictive pattern was observed in 8.3% patients comparable to this study.

In this study, though patients did not complain subjective respiratory symptoms, 10 patients had clubbing. In the present study, decreased chest movement was seen in 12% patients, decreased breath sounds in 18% and barrel shaped chest was seen in 8% subjects. In a similar study by K.V.Thiruvengadam et al., decreased chest movement, decreased breath sound, and barrel shaped chest were seen in 50%, 43.3% and 16.7% patients.

An important finding in this study is that 88% patients had reduced $FEF_{25-75\%}$ and 74% showed significant fall in $FEF_{25-75\%} < 60\%$. $FEF_{25-75\%}$ is a very good indicator of small airway disease. Statistically the percentage of reduction occurred in 88% of patients which is significant.

Hogg et al[14] made a finding of major importance that airways 2 mm or less in diameter contribute only a minor part of the total airway resistance normally,

but that these airways become the principal sites of increased resistance in COPD. This study gave rise to the concept that COPD is a small airway disease.

In male, the reduction in forced expiratory volume reduction in 1s (FEV_1) per year above the normal decline in adults for each pack-year of smoking is 9 ml; in female the excess rate of decline is about 6 ml. Based on these rates of decline, a man who has smoked one pack per year for the past 20 years will have FEV_1 that is 180 ml less than it would have been if the person had not smoked. In this study it is observed that patients with longer duration of smoking had much reduced FEV_1 than those with shorter duration of smoking.

In the present study, there was significant reduction in FEV_1. The mean FEV_1 was 1.67±0.05 L against the expected 2.56±0.49L which was 65% of the expected for the age. Reduction in FEV_1 was maximum in the age group of 50-60 years - 43% of the expected and minimum in 20-29 yrs -79%.

In a study by Fletcher et al.,[16] 800 working men both smokers and non smokers were studied,

the rate of decline of FEV_1 that normally begins by 25 years of age, was markedly increased in 10-15 % of susceptible smokers. Clinical disability did not occur until late in the disease when FEV_1 was reduced by about 70% of the expected value. This study results may help to explain why many patients even when FEV_1 was decreased to 60% did not have respiratory symptoms like breathlessness. The findings of Fletcher's study have been confirmed by the Lung Health Study.[23]

The Lung Health Study revealed that among middle- aged smokers with FEV_1 between 55 and 90% of predicted, differences of several hundred ml in FEV_1 developed within five years between those who quit and those who did not quit smoking. Such deleterious effect of smoking was seen in 10-15% of the susceptible smokers.

The mean FEV_1 in this study was 1.675 L and the expected mean FEV_1 was 2.56± 0.42 L, which is 65.5% of the expected value. In a similar study by Vaidya PR et al the observed FEV_1 was 1.28 ±0.66L.

The mean expected FVC was 3.25 ±0.48L , the actual mean was 1.94 ± 0.64 L which was about 60% of the expected value. In Vaidya et al., study observed value was 1.97±0.66 L.

On an average, the rate of expiratory airflow in smokers decrease twice as fast in smokers (40ml per year) as in non smokers(20ml per year). [19]

The small sample of 3% female patients were matched for age and smoking habits and it was found that FEV_1 was 0.92L (39%) compared to 1.01 (43%) in male subjects.

Though definitive conclusion could not be drawn due to small sample size, the accelerated decline in fev1 was noted in women than men.

In a study by Gan WQ et al[28], it was concluded that women as they age, are at increased risk of accelerated decline in FEV_1. In another study by Johannessen et al[29]., sex and residential area did not influence the incidence of COPD.

Thus spirometry is very useful to detect obstructive lung disease by measuring dynamic flow rates. It identifies whether flow obstruction is present or not, and characterizes whether the obstruction is inspiratory, expiratory or both. It thus helps in identifying COPD in early stages so that smoking cessation will reduce the disease burden to the society.

CONCLUSION

- In this study, 46% patients showed obstructive pattern, 32% showed mixed pattern, 8% showed restrictive pattern and 14% subjects had normal lung function.

- Eighty eight percent patients had small airway disease as evidenced by decreased FEF25-75.

- Progressive decline of FEV_1 (more than that of age related decline) was observed in asymptomatic smokers as the f was 79%, 73%, 53%,and 43% in the age groups of 20-29, 30-39, 40-49 and 50-60 respectively.

- Age, increased quantum of smoking, female gender all are compounding factor in obstructive lung disease as evidenced by more FEV_1 reduction with increasing age, increased pack years of smoking. Mean FEV_1 was 65.5% of the expected normal value.

- Though definitive conclusion could not be drawn, due to small sample size, the accelerated decline of FEV_1 was noted in women than men, as age matched FEV_1 was 39% in women as compared to 43% in men of same age.

- Routine Pulmonary function test in asymptomatic smokers is an important investigation to identify early obstructive/mixed/restrictive lung disease, lest such identified persons could be enrolled for nicotine deaddiction. Early nicotine deaddiction if contemplated, might play an important role in primary prevention of obstructive lung disease.

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PROFORMA

Case No.

Name	:	Age	:	Sex :
IP No.	:	DOA	:	DOD:
Address	:			Occupation :

FINAL DIAGNOSIS:

H/o Hypertension

H/o diabetes mellitus

Past History

IHD ☐ TB ☐ RHD ☐ BA ☐

Personal History

TOBOCCO use: Smoking Beedi/ Cigarette/ pipe / cigar

Quantity

Period Other forms

Alcohol

GENERAL EXAMINATION

Height Weight BMI

PR : RHYTHM : CHARECTOR :

Temperature: BP:

Respiratory rate: Type:

Pallor: Cyanosis:
Lymph node: Clubbing:
JVP:

RESPIRATORY SYSTEM

UPPER RESP.TRACT NONE
PNS
THROAT

INSPECTION

Shape of chest: B/L Symmetrical / Not
Trachea position: R / Central / L
Any spinal deformity: Scoliosis / kyphosis / Normal
Any chest deformity: Hollowing / Bulging / Normal
Movement of chest: Equal / Not
Any dilated veins/pulsations: Present / Not
Any scar / sinuses : Present / Not
Use of accessory muscles: Yes / No
Intercostal retraction: Yes / No
Apical impulse: Visualized / Not

PALPATION
Tracheal position: R / Central / L
Movement of chest: Equal / Not
Apical impulse: Normal / Not

MEASUREMENTS
AP Diameter:
Transverse diameter:
Chest circumference:
Expansion:
Palpable rales/ ronchi: Yes / No
Site

Percussion
Anteriorly
- Kronig isthmus
- Infraclavicular
- Mammory
- Liver dullness 5th ICS Yes / No
- Traubes area Tympanic / Not
- Tidal percussion:

Posteriorly
- Suprascapular:
- Infrascapular:
- Interscapular:
- Axillary:
- Infra axillary:

AUSCULTATION
Breath sounds - normal / diminished
Type - Vesicular / Bronchovesicular / Bronchial
Added sounds - rales / rhonchi
Site :
VR Equal / Not

CVS
ABDOMEN
CNS
Investigations :

Blood Sugar : URINE :

Serum Creatinine :

CXR PA :

ECG :

Echo :

Spirometry :

Others

Bronchoscopy

HRCT

MASTER CHART								
S. No.	**IP No.**	**Age**	**Sex**	**occupation**	**Duration of smoking**	**FEV1%**	**FEC1/FVC**	**FEF25-75%**
1	107402	23	1	2	1	1	1	1
2	107569	30	1	1	2	1	1	2
3	107600	26	1	1	2	1	1	2
4	107771	38	1	3	2	1	1	1
5	107772	57	1	1	4	3	3	3
6	107974	24	1	1	1	1	1	1
7	108064	58	1	2	4	3	3	3
8	107984	46	1	1	3	3	2	3
9	108411	49	1	3	3	3	3	3
10	108481	39	1	1	2	2	1	3
11	108492	60	1	1	4	3	3	3
12	108596	31	1	2	2	1	1	1
13	108730	40	1	3	2	2	1	3
14	109025	59	1	1	4	3	2	3
15	109026	54	1	1	3	3	2	3
16	109137	59	1	2	4	3	3	3
17	109146	41	1	1	3	3	2	3
18	109289	42	1	1	3	3	3	3
19	109349	44	1	3	3	3	2	3
20	109360	53	1	2	4	3	3	3
21	109681	51	1	1	4	3	2	3
22	109779	59	1	1	4	3	3	3
23	109980	42	1	2	2	2	1	3
24	109984	25	1	1	1	1	1	1
25	110026	33	1	3	2	2	1	2
26	110139	57	1	1	4	3	2	3
27	110163	35	1	3	2	2	1	2
28	110176	50	1	3	4	3	2	3

29	110260	41	1	1	2	3	1	3
30	110288	58	1	3	4	3	2	3
31	110575	36	1	1	2	2	1	2
32	110585	55	1	3	4	3	3	3
33	110684	48	1	1	3	3	2	3
34	110857	53	1	2	4	3	3	3
35	110870	44	1	1	3	3	2	3
36	111042	59	2	3	4	3	3	3
37	111138	35	1	1	2	2	2	3
38	111542	40	1	3	2	2	2	3
39	111630	51	1	3	4	3	3	3
40	111748	28	1	1	2	1	1	3
41	112044	60	2	3	4	3	3	3
42	112104	59	1	1	4	3	3	3
43	112402	49	1	3	3	3	2	3
44	112455	35	1	3	2	2	1	2
45	112459	39	1	1	2	2	2	2
46	112582	29	1	3	2	1	1	1
47	112586	36	1	1	2	3	3	3
48	112661	51	1	3	3	3	2	3
49	112786	56	2	1	4	3	3	3
50	112911	42	1	1	2	2	2	3
51	112961	58	1	1	4	3	2	3
52	113053	31	1	1	2	1	1	1
53	113211	52	1	1	4	3	2	3
54	113146	44	1	1	3	3	2	3
55	113204	58	1	1	4	3	3	3
56	113284	40	1	1	2	2	1	2
57	113361	42	1	2	2	2	1	2
58	113529	37	1	1	2	2	1	2
59	113619	56	1	3	4	3	3	3
60	113696	49	1	1	3	3	2	3

61	113800	52	1	2	4	3	3	3
62	113820	59	1	1	4	3	3	3
63	113815	43	1	1	3	3	3	3
64	113828	60	1	2	4	3	3	3
65	114140	36	1	1	2	2	2	2
66	114165	27	1	1	2	1	1	1
67	114263	51	1	3	4	3	2	3
68	114301	59	1	1	4	3	3	3
69	114364	40	1	1	2	2	2	3
70	114424	49	1	3	3	3	3	3
71	114624	24	1	2	1	1	1	1
72	114648	51	1	3	4	3	3	3
73	114653	48	1	3	3	3	2	3
74	114686	29	1	3	2	1	1	2
75	115074	60	1	1	4	3	3	3
76	115092	25	1	1	1	1	1	1
77	115205	60	1	1	4	3	3	3
78	115094	45	1	3	3	3	2	3
79	115291	35	1	1	2	3	3	3
80	115319	59	1	2	4	3	3	3
81	115332	26	1	1	1	1	1	1
82	115555	34	1	3	2	2	1	2
83	115706	58	1	3	4	3	3	3
84	115782	41	1	1	2	2	2	3
85	115783	53	1	3	3	3	2	3
86	115813	34	1	3	2	2	1	3
87	115936	53	1	1	3	3	2	3
88	116013	59	1	3	4	3	3	3
89	116031	40	1	1	2	3	1	3
90	116242	30	1	2	2	1	1	3
91	116463	57	1	3	4	3	2	3
92	116942	41	1	3	2	2	2	3

93	116977	27	1	1	2	1	1	1
94	117082	56	1	1	4	3	2	3
95	117131	39	1	3	2	1	1	2
96	117135	34	1	3	2	2	1	3
97	117194	51	1	2	4	3	3	3
98	117200	41	1	3	2	2	1	3
99	117218	47	1	1	3	3	2	3
100	117615	40	1	1	3	3	2	3

KEY

SEX: MALE-1; FEMALE-2

OCCUPATION: FARM WORKER-1; MANUAL LABOURER-2; OTHERS-3

DURATION: 0-5YRS-1; 5-10YRS-2; 10-15YRS-3; >15YRS-4.

FEV1 : >80%-1 ; 60-80%-2 ; <60%-3

FEV1/FVC : >80%-1 ; 60-80%-2 ; 40-60%-3 ; <40%- 4

FEF25-75 : >80-1 ; 60-80-2 ; <60-3.