"ASSESSMENT OF PULMONARY FUNCTION TESTS IN TYPE 2 DM (SPIROMETRY BASED)"

Dissertation submitted in partial fulfillment of the

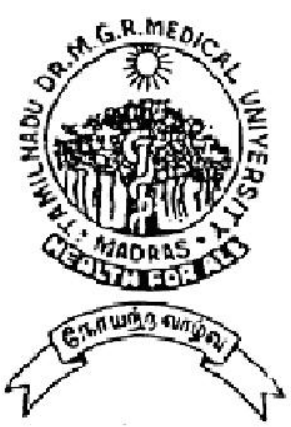
Requirement for the award of the Degree

of

DOCTOR OF MEDICINE

BRANCH I - GENERAL MEDICINE

APRIL 2012



THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled **"ASSESSMENT OF PULMONARY FUNCTION TESTS IN TYPE 2 DM (SPIROMETRY BASED)"** is a bonafide work of **Dr.A.THANGADHURAI,** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D General Medicine Branch I examination to be held in April 2012.

Dr. MOSES.K.DANIEL M.D
Professor and HOD,
Department of General Medicine,
Government Rajaji Hospital,
Madurai Medical College,
Madurai.

Dr.M.NATARAJAN M.D
Professor,
Department of General Medicine,
Government Rajaji Hospital,
Madurai Medical College,
Madurai.

DECLARATION

I, **Dr. A.THANGADHURAI ,** solemnly declare that, this dissertation **"ASSESSMENT OF PULMONARY FUNCTION TESTS IN TYPE 2 DM (SPIROMETRY BASED)"** is a bonafide record of work done by me at the Department of General Medicine, Government Rajaji Hospital, Madurai, under the guidance of **Dr.M.NATARAJAN M.D.,** Professor, Department of General Medicine, Madurai Medical college, Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Degree of Doctor of Medicine (M.D.), General Medicine Branch-I, examination to be held in April 2012.

Place: Madurai

Date:

Dr.A.THANGADHURAI

ACKNOWLEDGEMENT

I would like to thank **Dr.EDWIN JOE, M.D.,** Dean, Madurai Medical College, for permitting me to utilise the hospital facilities for the dissertation.

I also extend my sincere thanks to **Prof.Dr.MOSES .K.DANIEL M.D,** Head of the Department and Professor of Medicine for his constant support during the study.

I would like to express my deep sense of gratitude and thanks to my Unit Chief, my guide and Professor of Medicine,**Dr.M.NATARAJAN M.D.,** for his valuable suggestions and excellent guidance during the study.

I express my sincere thanks to **Dr.RAMESH, M.D.(TB&Chest diseases)** Professor & HOD,Dept.of Pulmonology for permitting me to utilise the facilities of the Department for the purpose of this study and guiding me with enthusiasm through out the study period.

I thank the Assistant Professors of my Unit **Dr.G.SELVARANI M.D., D.H.Sc (Echo)** and **Dr.K.MURALIDHARAN, M.D.** and Assistant Professor of Department of Pulmonology **Dr.S.VIVEKANANDHAN, M.D,D.T.C.D.,** for their valid comments, guidance and suggestions.

I wish to acknowledge all those, including my Post graduate colleagues, my parents and my wife who have directly or indirectly helped me complete this work with great success.

Last but definitely not the least, I thank all the patients who participated in this study for their extreme patience and co-operation.

CONTENTS

S.NO.	TITLE PAGE	PAGE.NO.
1.	NEED FOR THE STUDY	1
2.	INTRODUCTION	4
3.	REVIEW OF LITERATURE	28
4.	AIM OF THE STUDY	39
5.	MATERIALS AND METHODS	40
6.	OBSERVATIONS AND RESULTS	44
6.	DISCUSSION	70
7.	CONCLUSION	73
8.	LIMITATIONS OF THE STUDY	75

BIBLIOGRAPHY

PROFORMA

MASTER CHART

ABBREVATIONS

ETHICAL COMMITTEE APPROVAL FORM

LIST OF TABLES

TABLE NO	TABLES	PAGE NO
1	Age distribution	44
2	Sex distribution	46
3	Physiological variables	47
4	Blood Sugar levels	49
5	Duration of Diabetic cases	49
6	Observed Spirometric results	51
7	% Predicted values of PFT	52
8	Duration and Observed Spirometric results	54
9	Duration and% Predicted values of PFT	55
10	FBS Level and Observed Spirometric results	58
11	FBS level and% Predicted values of PFT	59
12	PPBS Level and Observed Spirometric results	62
13	PPBS level and% Predicted values of PFT	63
14	HbA1c Level and Observed Spirometric results	66
15	HbA1c level and% Predicted values of PFT	67

LIST OF FIGURES

FIG. NO	FIGURES	PAGE NO
1	Mechanism of hyperglycemia in type 2 DM	5
2	Normal flow-volume loop of spirometry	20
3	Flow-volume loop (Obstructive pattern)	20
4	Flow-volume loop (Restrictive pattern)	21
5	Lung volumes	25
6	Age distribution	45
7	Sex distribution	46
8	Physiological variables	48
9	Mean blood sugar values between cases and controls	50
10	Duration of Diabetic cases	50
11	Comparison of spiromtric values among Diabetics and Non Diabetics	53
12	Duration % Predicted values of PFT	56
13	Comparison of %Predicted FEV1/FVC with duration of Diabetics	57
14	FBS level and% Predicted values of PFT	60
15	Comparison of FEV1/FVC ratio with FBS among Diabetics	61
16	PPBS level and% Predicted values of PFT	64
17	Comparison of %Predicted FEV1/FVC with PPBS among Diabetics	65
18	HbA1c level and% Predicted values of PFT	68
19	Comparison of %Predicted FEV1/FVC with HbA1c among Diabetics	69

NEED FOR THE STUDY

We are today witnessing an epidemic of diabetes mellitus(DM), globally nationally. DM and its complications have become the most important contemporary and challenging health problem. There are 170 million diabetics worldwide. In india more than 5 crores have been affected. India has become the diabetic capital of world.

Practically every system is affected by complications of DM .Attention is usually paid to micro and macro angiopathy,retinopathy and nephropathy,but one of the system most neglected in DM is the respiratory system,except for the recognition of increased infection prevalence like tuberculosis.

Normal lung function has three components,which contribute to gas exchange

- Ventilation – Movements of gas in and out of lung.
- Perfusion – The perfusion of venous blood from right ventricle to ventilated alveoli.
- Diffusion – The diffusion of gases across the alveolar capillary membrane.

Ventilation has two processes Inspiration and Expiration. Inspiration is an active process occurring on contraction of intercostal muscles and diaphragm normally, Expiration is essentially passive due to elastic recoil of chest wall and lungs.

Several changes occur in DM, including:

1. Non-enzymatic glycosylation of connective tissue,especially collagen,which might be responsible for end organ damage causing diabetic neuropathy, diabetic nephropathy, diabetic retinopathy and lung changes. (diabetic pulmonopathy)
2. Diabetic myopathy
3. Micro vascular angiopathy

These changes could lead to

- Loss of elasticity.
- Altered perfusion characteristics.
- Weakness of the respiratory muscles responsible for ventilation.

Ventilation may be affected by myopathy and altered elastic recoil of lung tissue. Perfusion may be affected by changes in basement membrane and micro vascular angiopathy.

All may contribute to altered lung function, there are studies, which showed changes in lung function in DM, but the study number is not large.

This study will help add to the growing literature on changes in lung function in diabetes mellitus.

There is increasing interest in this area and few publications. However the total number of studies with respect to lung function in type 2 DM is still very small and involved very small patients. Hence this study was performed to add to the experience in this area.

INTRODUCTION

Diabetes mellitus is a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia resulting from defects in insulin secretion, action or both. Based on etiopathogenic categories, it is classified as Type-1 and Type-2 diabetes mellitus. In Type-1 there is absolute deficiency of insulin secretion. In Type-2 there is a combination of resistance to insulin action and inadequate compensatory insulin secretory response. Diabetes mellitus is accompanied by wide spread biochemical, morphological and functional abnormalities which may precipitate certain complications that affect the renal, cardio-vascular, neural systems and also skin, liver, collagen and elastic fibres. Thus diabetes is a multisystem disorder that affect many organs of the body.[1]

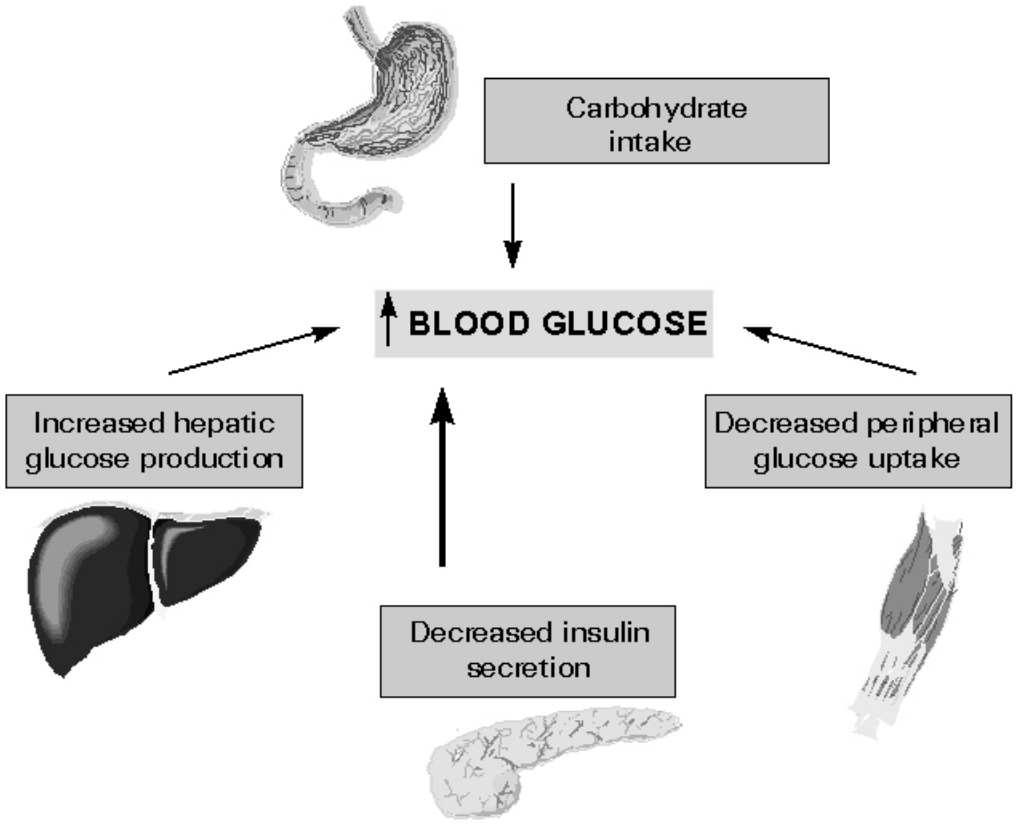
ADA-Criteria for the Diagnosis of Diabetes Mellitus.[2]

- Symptoms of diabetes plus random blood glucose concentration -11.1 mmol/L (200 mg/dL)[a] *or*
- Fasting plasma glucose -7.0 mmol/L (126 mg/dL)[b] *or*
- Two-hour plasma glucose -11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[c]

TYPE 2 DIABETES MELLITUS

Type 2 diabetes is characterized by the combination of peripheral insulin resistance and inadequate insulin secretion by pancreatic beta cells. Insulin resistance, which has been attributed to elevated levels of free fatty acids in plasma,[3] leads to decreased glucose transport into muscle cells, elevated hepatic glucose production, and increased breakdown of fat.

Fig 1: Mechanism of hyperglycemia in type 2 DM



For type 2 diabetes mellitus to occur, both defects must exist. For example, all overweight individuals have insulin resistance, but diabetes develops only in

those who cannot increase insulin secretion sufficiently to compensate for their insulin resistance. Their insulin concentrations may be high, yet inappropriately low for the level of glycemia.

Beta cell dysfunction is a major factor across the spectrum of pre-diabetes to diabetes. A study of obese adolescents by Bacha et al confirms what is increasingly being stressed in adults as well: Beta cell function happens early in the pathological process and does not necessarily follow stage of insulin resistance.[4] Singular focus on insulin resistance as the "be all and end all" is gradually shifting, and hopefully better treatment options that focus on the beta cell pathology will emerge to treat the disorder early.

In the progression from normal glucose tolerance to abnormal glucose tolerance, postprandial blood glucose levels increase first; eventually, fasting hyperglycemia develops as suppression of hepatic gluconeogenesis fails.

During the induction of insulin resistance, such as is seen after high-calorie diet, steroid administration, or physical inactivity, increased glucagon levels and increased glucose-dependent insulinotropic polypeptide (GIP) levels accompany glucose intolerance; however, postprandial glucagonlike peptide-1 (GLP-1) response is unaltered.[5] This has physiologic implications; for example, if the GLP-1 level is unaltered, GLP-1 may be a target of therapy in now a days.

Major risk factors for type 2 diabetes mellitus are the following:

- Age greater than 45 years (though, as noted above, type 2 diabetes mellitus is occurring with increasing frequency in young individuals)
- Weight greater than 120% of desirable body weight
- Family history of type 2 diabetes in a first-degree relative (eg, parent or sibling)
- Hispanic, Native American, African American, Asian American, or Pacific Islander descent
- History of previous impaired glucose tolerance (IGT) or impaired fasting glucose (IFG)
- Hypertension (>140/90 mm Hg) or dyslipidemia (high-density lipoprotein [HDL] cholesterol level < 40 mg/dL or triglyceride level >150 mg/dL) .[8]
- History of gestational diabetes mellitus or of delivering a baby with a birth weight of >9 lb
- Polycystic ovarian syndrome (which results in insulin resistance)

Type 2 DM has a strong genetic component. The concordance of type 2 DM in identical twins is between 70 and 90%. Individuals with a parent with type 2 DM have an increased risk of diabetes; if both parents have type 2 DM, the risk approaches 40%. Insulin resistance, as demonstrated by reduced

glucose utilization in skeletal muscle, is present in many nondiabetic, first-degree relatives of individuals with type 2 DM. The disease is polygenic and multifactorial since in addition to genetic susceptibility, environmental factors (such as obesity, nutrition, and physical activity) modulate the phenotype. The genes that predispose to type 2 DM are incompletely identified, but recent genome-wide association studies have identified several genes that convey a relatively small risk for type 2 DM (relative risk of 1.1-1.5).

Most prominent is a variant of the transcription factor 7-like 2 *(TCF7L2)* gene that has been associated with type 2 diabetes in several populations and with impaired glucose tolerance in one population at high risk for diabetes. *TCF7L2* is a transcription factor and key component of the Wnt signaling pathway, and it is involved in the development of a wide variety of cell lineages and organs.[9] Potential mechanisms through which *TCF7L2* variants influence type 2 diabetes include its role in adipogenesis, myogenesis, and pancreatic islet development, as well as in beta-cell survival and insulin secretory granule function.[10,11] It is also involved in the transcriptional regulation of the genes for proglucagon and the glucagon-like peptides GLP-1 and GLP-2; these peptides play a role in postprandial insulin secretion.[12]

Finally, *TCF7L2* polymorphisms have been associated with impaired insulin secretion, glucose production, and glucose tolerance via direct effects

on pancreatic islet beta cells.[13,14] Indeed, dysregulation of glucose metabolism, decreased processing of proinsulin, and elevated levels of gastric inhibitory peptide and glycated haemoglobin (HbA1c) can be observed in normoglycemic individuals with *TCF7L2* polymorphisms before the onset of type 2 diabetes.[15,16] Thus, while the specific mechanism driving the development of type 2 diabetes remains unclear, there is sufficient evidence to demonstrate that *TCF7L2* variants strongly predict the development of type 2diabetes and/or the progression to diabetes from impaired glucose tolerance.[14, 17]

Although genetic tests for *TCF7L2* could help predict the incidence and the rate of onset of type 2 diabetes,[18] the strongest predictors continue to be positive family history, increased body mass index, increased blood pressure, and increased serum levels of triglycerides, apolipoprotein A-1, and liver enzymes, all of which precede inception of metabolic syndrome.[19] In fact, the predictive power of *TCF7L2* variants disappears with lifestyle modifications or metformin treatment, while the improved insulin sensitivity resulting from these changes directly oppose the pathological influence of *TCF7L2* variants.[20] These data suggest that genetic susceptibility to type 2 diabetes as

determined by *TCF7L2* variants might prove an actionable indicator for early intervention and disease prevention.[17]

The incidence of Type-2 diabetes has been steadily increasing in urban areas to 8.4% the rapid urbanization, change in the lifestyle coupled with ethnic susceptibility has increased the incidence of diabetes mellitus. This globally important condition needs to be understood with a proper perspective to deliver effective strategies to the individual and also the population.

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Chronic complications can be divided into vascular and nonvascular complications . The vascular complications of DM are further subdivided into microvascular (retinopathy, neuropathy, nephropathy) and macrovascular complications [coronary artery disease (CAD), peripheral arterial disease (PAD), cerebrovascular disease]. Nonvascular complications include problems such as gastro paresis, infections, and skin changes. Long-standing diabetes may be associated with hearing loss.[41] Whether type 2 DM in elderly individuals is associated with impaired mental function is not clear.

PATHOGENESIS OF COMPLICATIONS

Although chronic hyperglycemia is an important etiologic factor leading to complications of DM, the mechanism(s) by which it leads to such

diverse cellular and organ dysfunction is unknown. Four prominent theories, which are not mutually exclusive, have been proposed to explain how hyperglycemia might lead to the chronic complications of DM.[33]

One theory is that increased intracellular glucose leads to the formation of advanced glycosylation end products (AGEs) via the nonenzymatic glycosylation of intra- and extracellular proteins. Nonenzymatic glycosylation results from the interaction of glucose with amino groups on proteins. AGEs have been shown to cross-link proteins (e.g., collagen, extracellular matrix proteins), accelerate atherosclerosis, promote glomerular dysfunction, reduce nitric oxide synthesis, induce endothelial dysfunction, and alter extracellular matrix composition and structure. The serum level of AGEs correlates with the level of glycemia, and these products accumulate as glomerular filtration rate declines.[44]

A second theory is based on the observation that hyperglycemia increases glucose metabolism via the sorbitol pathway. Intracellular glucose is predominantly metabolized by phosphorylation and subsequent glycolysis, but when increased, some glucose is converted to sorbitol by the enzyme aldose reductase. Increased sorbitol concentration alters redox potential, increases cellular osmolality, generates reactive oxygen species, and likely leads to other types of cellular dysfunction. However, testing of this theory in humans, using

aldose reductase inhibitors, has not demonstrated significant beneficial effects on clinical endpoints of retinopathy, neuropathy, or nephropathy.

Third hypothesis proposes that hyperglycemia increases the formation of diacylglycerol leading to activation of protein kinase C (PKC).[34]

Among other actions, PKC alters the transcription of genes for fibronectin, type IV collagen, contractile proteins, and extracellular matrix proteins in endothelial cells and neurons. Inhibitors of PKC are being studied in clinical trials.

And the fourth theory proposes that hyperglycemia increases the flux through the hexosamine pathway, which generates fructose-6-phosphate, a substrate for O-linked glycosylation and proteoglycan production. The hexosamine pathway may alter function by glycosylation of proteins such as endothelial nitric oxide synthase or by changes in gene expression of transforming growth factor (TGF-β) or plasminogen activator inhibitor-1 (PAI - 1).

Growth factors appear to play an important role in DM-related complications, and their production is increased by most of these proposed pathways. Vascular endothelial growth factor A (VEGF-A) is increased locally in diabetic proliferative retinopathy and decreases after laser photocoagulation. TGF- β is increased in diabetic nephropathy and stimulates basement

membrane production of collagen and fibronectin by mesangial cells. Other growth factors, such as platelet-derived growth factor, epidermal growth factor, insulin-like growth factor I, growth hormone, basic fibroblast growth factor, and even insulin, have been suggested to play a role in DM-related complications.[43]

The micro vascular complications appear early, within 5 to 10yrs and macro vascular complications appear within 15 to 20yrs from the onset of diabetes. If diabetes is detected early and adequate steps are taken,it may be possible to significantly delay the occurrence of complications and thereafter the progression.

DIABETES AND LUNG

There are histopathological changes seen in lungs of diabetics such as thickened alveolar epithelial and pulmonary capillary basal lamina leading to reduced pulmonary elastic recoil and lung volume. There is impaired diffusion due to reduced pulmonary capillary blood volume and thickening of the basement membrane. The underlying mechanism seem to be microangiopathy brought in by the nonenzymatic glycosylation of various scleroproteins in lungs and elsewhere. Since collagen is the most abundant tissue protein in major bronchi, vessels and interstitium, the alterations in pulmonary functions occur as a rule. These alterations are reversible to start with & can be delayed

by keeping the blood sugar levels in the normal range. Similar changes have been observed with advancing age though progression & intensity of changes are less marked than seen in patients with DM .[21] Non-enzymatic glycoslyation induced alteration of lung connective tissue is the most likely mechanism underlying the mechanical pulmonary dysfunction in diabetic subjects. This suggests that lung should also be considered as target organ.[22]

The review of research work in this field shows two conflicting schools of thought, each one either expounding involvement or non-involvement of respiratory system in Type-2 DM. The pulmonary function in diabetes of this cross section of population is not extensively documented. The present study is undertaken to evaluate the impact of Type-2 DM and pulmonary function in this cross section of population and thereby resolve the conflict between two schools of thought.

RESPIRATORY FUNCTION

The principal function of the lung is to efficiently exchange oxygen in the distal air spaces with carbon dioxide in the blood. Ventilation-perfusion matching is accomplished by structural attributes that create an enormous capillary surface area and exceedingly thin diffusion barrier for gas. The airways, forming the connection between the outside world and the terminal respiratory units, are of central importance to our understanding of lung

function in health and disease. Intrapulmonary airways are divided into three major groups: bronchi, membranous bronchioles and respiratory bronchioles/gasexchange ducts . Bronchi, by definition, have cartilage in their wall. Respiratory bronchioles serve a dual function as airways and as part of the alveolar volume (gas exchange).[42]

Secondary functions of the lung also are important, such as surfactant synthesis, secretion, and recycling, mucociliary clearance, immunomodulation, neuroendocrine signaling, and synthesis and secretion of a myriad of molecules by its epithelial and endothelial cells. The diversity of secondary functions emphasizes the importance of the lung in homeostasis.

SPIROMETRY

Spirometry is the most basic and frequently performed pulmonary function test. Pulmonary function testing measures how well you are breathing. There are different types of breathing tests that can be done during pulmonary function testing. These tests include spirometry, Peak flow meter, lung volumes and diffusing capacity. Lung volumes measure different parts of the breath to determine how much air you can breathe in and out. Some of the volumes are: total lung capacity, vital capacity and functional residual capacity. The diffusing capacity of the lungs (DLCO) measures how well gases such as oxygen move from the lungs into the blood.[23,24] The most common way to

measure the DLCO is the ten second single-breath-hold technique. Spirometry is a simple test to measure how much (volume) and how fast (flow) you can move air into and out of your lungs. Spirometry is a non-invasive and completely painless procedure, like the measurement of blood pressure it is a useful screen of general health.[42]

NEED FOR SPIROMETRY

Diagnostic

• To evaluate symptoms, signs or abnormal laboratory tests: Symptoms: shortness of breath, wheezing, cough, phlegm. Signs: diminished breath sounds, cyanosis.

• To screen individuals at risk of having pulmonary disease: Smokers, occupational exposure

• Assess preoperative risk

• Assess prognosis (lung transplants)

Monitoring

• To assess therapeutic interventions: Bronchodilator therapy Steroid treatment for asthmatics, interstitial lung disease, etc Management of congestive heart failure, Other (antibiotics in cystic fibrosis)

• To describe the course of diseases affecting lung function: Pulmonary diseases (Obstructive or interstitial diseases) Cardiac diseases (congestive heart failure) Neuromuscular disease

• To monitor persons in occupations with exposure to injurious agents

Disability/Impairment Evaluations

• To assess patients as part of a rehabilitation program: Medical, Industrial,Vocational

• To assess risks as part of an insurance evaluation

Contraindications

• Hemoptysis of unknown origin

• Pneumothorax

• Unstable cardiovascular status

• Thoracic/abdominal/cerebral aneurysm

• Recent thoracic surgery

• Nausea or vomiting

Types of Spirometers

Spirometers use different methods of measuring patient's lung values. The most common type of device is called a "pneumotach". This device has been the "gold standard" of spirometry for many years. A pneumotach measures air

flow where particles of gas are accelerated, creating a pressure gradient to measure flow and calculate a volume of gas (essentially it measures a drop in pressure).

Anemonometer, this type of flowmeter consists of a thin platinum wire, electrically heated to constant temperature, and centrally located in a tube. As gas passes through the meter the wire cools off requiring electrical energy to maintain its temperature. The extra electrical energy is a measure of energy. This system is very vulnerable to damage and needs to be handled with extra care. The flowmeters are also not reliable due to the inability to know the direction of the patient's flow to measure inspiration and expiration.

Ultrasonic spirometers utilize transducers located on either side of the flow sensor and receive sound in alternating directions. When the gas flow moves through the tube, the pulse that travels against the flow is slowed down and takes a longer time to reach the opposite transducer. The pulse traveling with the flow is sped up and takes a shorter time to reach the opposite transducer. The gas flow in the flow sensor is then calculated from upstream and downstream times. This calculation is independent of gas composition, pressure, temperature and humidity.

Turbine spirometers use an infrared light to measure inspiratory and expiratory flows. The harder the patient blows, the faster the turbine rotates.

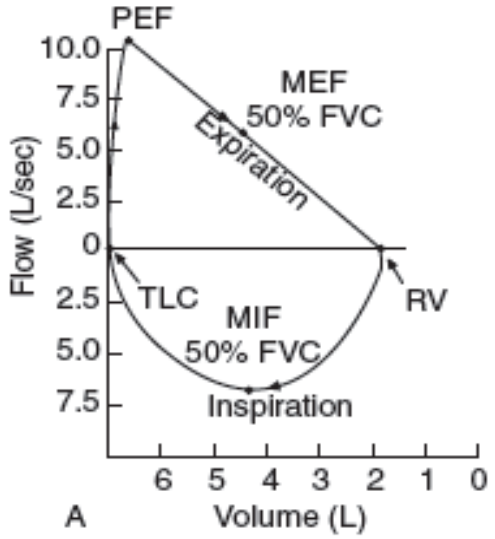
These rotations are measured by an infrared beam. They do not need to be calibrated or corrected for BTPS and are not influenced by pressure changes or air temperature.

Flow Volume Loop

The Flow Volume Loop is the most important curve in spirometry. It shows the relationship between flow (how fast air moves) and volume (how big of a breath). The normal flow volume is the graphic display of the FVC and is used to evaluate the patient's effort and test technique. A normal flow volume loop starts with a hard expiration to the peak expiratory flow (PEF). After the PEF the patient's flow decreases as more air is expired.[46] The Forced Expiratory Flow (FEF 25) measures the point were 25% of the volume is expired. The (FEF 50) and (FEF 75) are where 50% and 75% of the volume is expired. The (FEF 25-75) is the mean flow between the FEF 25 and FEF 75. This measurement is used in the diagnoses of many respiratory diseases.

(A) Normal. Inspiratory limb of loop is symmetric and convex. Expiratory limb is linear. Flow rates at the midpoint of the inspiratory and expiratory capacity are often measured. Maximal inspiratory flow at 50% of forced vital capacity (MIF 50%FVC) is greater than maximal expiratory flow at 50% FVC (MEF 50%FVC) because dynamic compression of the airways occurs during exhalation.

Fig 2: Normal flow-volume loop of spirometry



(B) Obstructive disease (eg, emphysema, asthma). Although all flow rates are diminished, expiratory prolongation predominates, and MEF < MIF. Peak expiratory flow is sometimes used to estimate degree of airway obstruction but is dependent on patient effort.[50]

Fig 3:Flow-volume loop (Obstructive pattern)

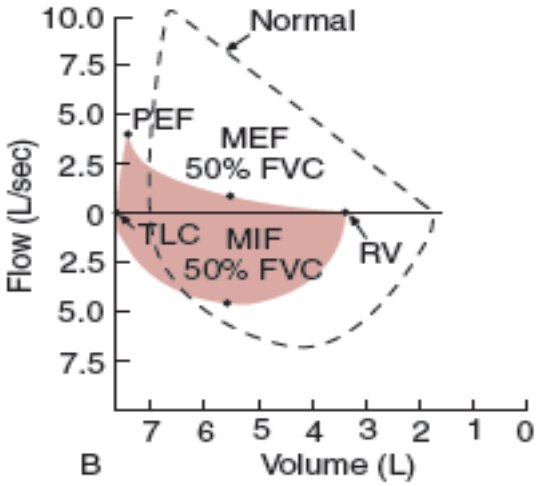
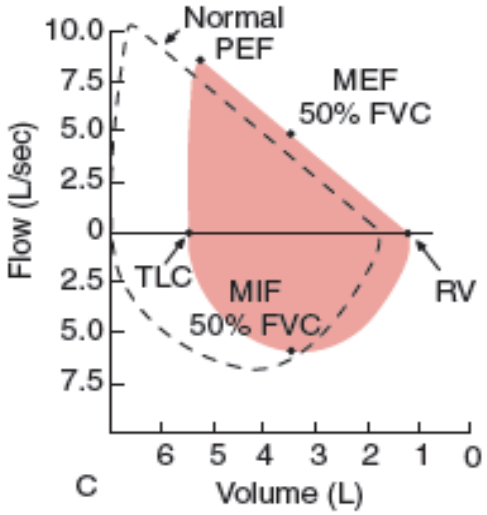


Fig 4: Flow-volume loop (Restrictive pattern)



(C) Restrictive disease (eg, interstitial lung disease, kyphoscoliosis). The loop is narrowed because of diminished lung volumes, but the shape is generally the same as in normal volume. Flow rates are greater than normal at comparable lung volume because the increased elastic recoil of lungs holds the airways open.

Interpretation

Interpretation of spirometry results should begin with an assessment of test quality. Failure to meet performance standards can result in unreliable test results. The American Thoracic Society (ATS) defines acceptable spirometry as an expiratory effort that shows (1) minimal hesitation at the start of the forced expiration (extrapolated volume (EV) £5% of the FVC or 0.15 L, whichever is larger), (2) no cough in the first second of forced exhalation, and

(3) meets 1 of 3 criteria that define a valid end-of-test: (a) smooth curvilinear rise of the volume-time tracing to a plateau of at least 1-second duration; (b) if a test fails to exhibit an expiratory plateau, a forced expiratory time (FET) of 15 seconds; or (c) when the patient cannot or should not continue forced exhalation for valid medical reasons.[25]

In patients that have significant loss of lung elastic recoil (pulmonary emphysema), spirometry may show "negative effort dependence of forced expiratory flow." In other words, the effort that has the highest peak expiratory effort may produce a lower FEV_1 because of dynamic compression of the larger airways. In this circumstance, the effort with the highest FEV_1produced by a submaximal effort should not be reported. Although not yet a standard, it appears that selecting only efforts that have a time to peak flow (T_{PEF}) less than or equal to 0.12 seconds helps eliminate this effect.[46,47]

Additionally, the 2 largest values for FVC and the 2 largest values for FEV_1 in the same testing session should vary by no more than 0.15 L (0.1 L if the largest value is < 1 L). A recent study has shown start-of-test problems (affecting FEV_1 measurements) to be relatively uncommon (2% prevalence in one series) and end-of-test problems (affecting FVC quality) being very common (61-84% prevalence). Allowing the patient to relax and push gently

after 3-4 seconds of forced exhalation has been shown to greatly enhance the ability of patients with airflow obstruction to satisfy end-of-test criteria.[51]

Inspection of the volume-time tracing aids in identification of early termination of expiration by evaluating the presence of an expiratory plateau. In the absence of an expiratory plateau, a 12- to 15-second expiratory time ensures the quality of the FVC. Inspection of the start of the volume-time tracing can identify a hesitant start, which can result in a falsely low FEV_1. Reproducibility of the FVC and the FEV_1 helps ensure that the results truly represent the patient's lung function. Attention should be focused on 3 key parameters: FVC, FEV_1, and the FEV_1 -to-FVC ratio.[45]

In the United States, normal values and lower limits of normal defined by Hankinson et al.[26] (the National Health and Nutrition Examination Survey [NHANES] III predicted set) should be used. These provide specific equations for whites, African Americans and Mexican Americans. If the patient belongs to another ethnic group, the predicted values and lower limits of normal provided for whites by Hankinson et al should be reduced by 12% by multiplying the predicted value by 0.88 before comparison with the patient's results.[48,49]

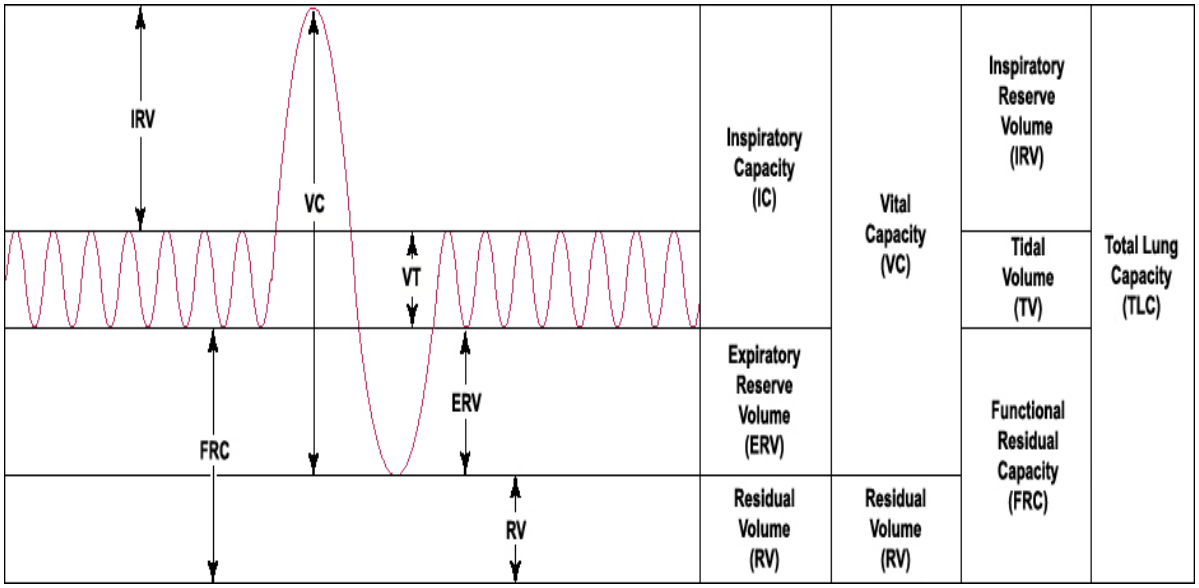
Disproportionate reduction in the FEV_1 as compared to the FVC (and therefore the FEV_1 -to-FVC ratio) is the hallmark of obstructive lung diseases. This physiologic category of lung diseases includes but is not limited to asthma, acute and chronic bronchitis, emphysema, bronchiectasis, cystic fibrosis, pneumonia, alpha1-antitrypsin deficiency, and bronchiolitis. The expiratory flow at any given expiratory volume is reduced. The mechanism responsible for the reduction in airflow can be bronchial spasm, airway inflammation, increased intraluminal secretions, and/or reduction in parenchymal support of the airways due to loss of lung elastic recoil.[52]

The use of a fixed lower limit of normal for the FEV_1/FVC ratio as proposed by the Global Initiative for Obstructive Lung Disease (GOLD) lacks a scientific basis and results in misclassifying patients at either end of the age spectrum. Young patients are classified as "normal" when airflow obstruction is present, and older patients are classified as showing obstruction when no airflow obstruction is present. The use of the GOLD threshold in clinical practice should be discouraged.[39]

Reduction in the FVC with a normal or elevated FEV_1 -to-FVC ratio should trigger further diagnostic workup to rule out restrictive lung disease. Because the FEV_1 is a fraction of the FVC, it also is reduced, but the FEV_1 -to-FVC ratio is preserved at a normal or elevated level.

LUNG VOLUMES

Fig 5 :Lung volumes



FVC

Forced vital capacity, volume change between a full inspiration to total lung capacity and a maximal expiration to residual volume.

FEV1

Forced expiratory volume at 1 second, volume of air that can be expired in 1 second after a maximal inspiration.

FEV1/FVC

Ratio between FEV1 and FVC.

FEV1/FEV6

Ratio between FEV1 and FEV6. This is a test that is used in the primary care or physicians office setting because it is easier to perform and obtain from the patient.

FEF 25-75%

The average expired flow over the middle half of the FVC and is regarded as a more sensitive measure of small airways narrowing than the FEV1.

PEF

Peak expiratory flow, maximum flow generated during expiration performed with maximal force and started after a full inspiration.

FRC

Functional Residual CapacitJy, volume of gas contained in the lung after a normal expiration.

IVC

Inspiratory Vital Capacity, the volume change between a maximal expiration to residual volume and a full inspiration to total lung capacity.

MVV

Maximal Voluntary Ventilation, a measure of the maximum amount of air that can be inhaled and exhaled in one minute, measured in liters/minute.

RV

Residual Volume, volume that remains in the lungs after a maximal expiration, cannot be measured by spirometry.

TLC

Total Lung Capacity, the volume of gas contained in the lung after a full inhalation, cannot be performed by spirometry because it includes the residual volume.

VC (Vital Capacity)

The volume change between a full inspiration and a maximal expiration.

REVIEW OF LITERATURE

1) Hiroshi Mori et al examined the possible association between the vascular complications of diabetes and changes in pulmonary function, they performed pulmonary function tests including assessment of the diffusing capacity (%DLco) in 80 patients with non-insulin dependent diabetes mellitus (45 males and 35 females) without overt lung or heart disease was performed. The mean age of the subjects was 57.9 years and the mean duration of diabetes was 10.8 years. The %DLco decresed significantly as the duration of diabetes increased ($r=-0.38$, $p<0.01$), and the same relationship was also observed in non-smoking individuals (n=37) ,The reduction in %DLco was greater in patients with diabetic microangiopathy (especially nephropathy) and in those treated with insulin. Other spirometric functions showed no relationship to the duration of DM, the degree of microangiopathy or the type of treatment. These results suggest that diabetic microangiopathy play on important role in the decrease of %DLco.[36]

In this study, %DLco was found to correlate negatively with duration of diabetes. Asanuma et al, and Sandler at al have reported that as the duration of diabetes increase, the pulmonary diffusion capacity is decreased. Uchida et al in a study of ventilation-perfusion scintigrams in diabetics, observed a decreased pulmonary diffusing capacity in patients with perfusion

defect. As these patients had a longer duration of diabetes and a higher incidence of retinopathy, pulmonary microangiopathy was suggested.[36]

2) Walter.E.Robert, Alexa Beiser, Rachel J, Givelber,George T, O'Connor, et al. studied "Association between glycemic state and lung function".

This study was conducted to analyze the relationship of diabetes and of fasting blood sugar to level of Pulmonary function test by Spirometric assessment in 87 members of Framingham and pulmonary function FEV1, FVC & FEV1/FVC ratio was detorating Collins survey 11 Spirometer and the predicted pulmonary function test was determined by the coefficients of regression of pulmonary function test on age, sex and body habitus.

Results :

The diabetes and a higher levels of FBS were associated with lower pulmonary function-The FVC was lower by 109 ml, FEV1 was lower by 27 ml and FEV1/FVC% was higher by 1.5% in the diabetics compared to non diabetics.[27]

3) Lange P. et al Studied- the possible association between diabetes mellitus, plasma glucose, forced vital capacity and forced

expiratory volume in 1 second in 11,763 subjects of 20yrs or older 284 of the participants were with diabetes mellitus.

Results:

There was a slight impairment of lung function it was more prominent in subjects treated with insulin than those taking oral hypoglycemic agents and/or diet, FVC was reduced by 334ml and FEV1 239ml in subjects treated with insulin and FVC was reduced by 184ml and FEV1 117ml in subjects treated with oral hypoglycemic agents and/or diet compared to controls.[28]

4) SCHNAPF et al found that there was reduction of lung volumes in IDDM patients when the patients also had decreased joint mobility. Consequently, it has been suggested that non-enzymatic glycosylation of connective tissue, especially the collagen, might be responsible for both lung and jont abnormalities. Their findings of an association between raised values of plasma glucose and lung function impairment are in accordance with this hypothesis.[38]

The prevalence of self reported DM in their study sample was 2.5%, which is slightly higher than the estimated prevalence of DM of 1-1.5% in the Danish population. This is due to age stratified sampling used in the present study

resulting in the bulk of the participants in the age group being between 40-70 years, in whom the prevalence of NIDDM can be as high as 4.5%. However the age distribution and the treatment regimes strongly suggests that the great majority of the diabetic subjects investigated had NIDDM. This means that some of the subjects in the DM1 group, especially the ones older than 60 years of age were suffering from NIDDM in spite of treatment with insulin.

Due to small number of diabetic subjects in many of the subgroups, the study does not allow detailed analysis of the impact of DM on ventilator function in the different age group. Even so, the slight lung function impairment in the diabetic subjects was present in all age groups and there was no significant interaction between age and DM in the regression analysis.

Many confounding factors might lead to reduction of both FEV1 and FVC in diabetic subjects. Two of them are obesity and cardiac failure. As many subjects with NIDDM are obese, the reduction of FEV1 and FVC in NIDDM might therefore be the result of being overweight rather than the result of NIDDM. However since they included BMI in the regression model, obesity was unlikely to be an explanation for observed lung function impairment. In addition, the most pronounced lung function impairment was in the DM1 group. Although none of the diabetic subjects had manifest heart failure during the examination, it was not possible to exclude that a mild pulmonary

congestion was present in some of the diabetic subjects, as ischemic heart disease is more prevalent in subjects with DM than in normals.[38]

5) Davis A Wendy, Matthew Knuiman, Peter Kendell, Valerie Grange, Timothy M.E.Davis et al Studied- Glycemic exposure is associated with reduced pulmonary function test in Type 2 diabetes.

In this study 495 patients with Type-2 DM who had no history of pulmonary disease was studied between 1993 and 1994 by community –based cohort study and 125 patient was restudied 7 years later for FVC, FEV1, VC & PEF corrected for BTPS and were expressed as absolute terms or as percentage predicted for age, sex and height.

Results:

There was a decrease in mean percentage-predicted values of each Spirometric measure to 10% in the whole cohort study at baseline and absolute measures continued to decline at an annual rate of 68ml, 71ml and 84ml/year and 17 l/min for FVC, FEV1, VC and PEF[29] respectively in the study group

6) Malcolm sander et al Studied-Is lung a 'target organ' in diabetes mellitus.

There is a histopathological evidence of lung involvement in subjects with diabetes mellitus by thickened alveolar epithelial and pulmonary capillary basal laminae suggestive of pulmonary microangiopathy. Abnormal pulmonary function has been detected in diabetic patients such as reduced lung volumes,

reduced pulmonary elastic recoil in diabetes, impaired diffusion due to a reduced pulmonary capillary blood volume and nonenzymatic glycosylation-induced alteration in the lung connective tissue is the most likely pathogenesis mechanism underlying the mechanical pulmonary dysfunction[21] in diabetic subjects.

7) Spomenka Ljubic et al study showed a reduction of diffusion capacity for carbon monoxide in DM patients. Diabetes can cause the development of pulmonary complications due to collagen and elastin changes, as well as microangiopathy. This study demonstrates the relationship between pulmonary complications and other chronic complications of diabetes. 27 patients with diabetes, aged 21 to 62 years, who had the disease from 3-32 years, were included in this study. The protein excretion rate(PER) and the diffusion capacity of the lung for carbon monoxide (DLco) were included as parameters of the severity of complications. PER was determined by Biuret method. DLco was measured by single breath method and was corrected by the measurement of alveolar volume (V/A).[23] The values of DLco as corrected by V/A (DLco/VA) were included in the statistical evaluation of the results. The variations of age, duration of DM, and complication parameters were included in a multiple regression model with forward, stepwise selection to asses their value in predicting DLco/VA. The variables were found to be significant

predictors of DLco/VA (R2=0.46, R2=0.32, p<0.022). However, proteinuria was the only significant independent predictor of DLco / VA. This finding indicates that both renal and pulmonary complications of diabetes share a similar microangiopathic background.

8) Davis Timothy M. E, Matthew Knuiman, Peter Kendell, Hien Vu, Wendy A. Davis et al Studied – Reduced pulmonary function is associated in Type-2-Diabetes mellitus.

The study was conducted on 421 subjects. Detailed demographic and diabetic specific-data were collected, spirometry was performed and FVC, FEV1, VC and PEF were measured and were expressed as percentage of prediction values for age, sex and height the means of all Spirometric measures were reduced by 9.5%. HbA1c was not associated with any measure of lung function but the diabetes duration was significantly associated with FEV1% prediction and PEF % prediction had borderline associations with FVC% prediction and VC% prediction.[30] Pulmonary function is reduced in type 2 DM and diabetes duration has more influence on pulmonary function than the glycemic control.

9) Sreeja C.K, Elzabeth Samuel, C.Kesava chandran, Shankar Shashidaran. et al Studied- Pulmonary function in patients with Diabetes mellitus.[40]

Lung function tests were carried out in20 Type2Diabetes mellitus and 20 Type1 Diabetes mellitus and 40 subjects as controls. There was a significant reduction in FEV1/FVC% in both diabetes mellitus groups compared with controls. The decrease in FEV1/FVC% in both the groups may be related to the poor mechanical properties of lung. Viz lung compliance and elastic recoil of lungs, TLC was lower in diabetes mellitus group because of alteration in collagen and elastin by loss of elastic recoil and low lung volumes and abnormal pressure volume relationship may be due to the respiratory muscle weakness.[31,40]

10) SK Rajan et al study of spirometric evaluation of type 1 DM, was a cross sectional study of 30 patients (group1) who are on insulin therapy. Group 2 consisted of age matched, non-smoking healthy volunteers who acted as controls. Spirometric revealed normal findings in 10 patients (33%), and abnormal findings in 20 patients (67%). Among these 20 patients with abnormal findings obstructive pattern in 8 patients, restrictive pattern was present in 9 patients and mixed pattern was observed in 7 patients. This study showed that the lung has to be considered as one of the target organs in type 1 DM. In view of the possibility pneumopathy in asymptomatic type1DM, all persons with long –standing type1DM should undergo regular assessment of pulmonary function.[37]

DM is associated with widespread hormonal, metabolic and micro vascular abnormality, as well as with disturbances of the function of many organ systems. The kidneys. Eyes, cardio vascular system and respiratory system can be damaged. The aim of the study was to investigate lung function in DM and renal complications. The development of these complications could be explained by the biochemical alteration of connective tissue constituents, particularly collagen and elastin, as well as micro angiopathy due to a non-enzymatic glycosylation of proteins induced by chronic hyperglycemia.

Collagen is an abundant structural protein found in the previously mentioned organic systems, so the disturbance of the function of those systems can be expected. The kidneys, eyes and lungs of patients with diabetes are affected consequently, and the patient develops obstructive and restrictive disorders. As a result of alveolar capillary membrane thickenings due to collagen and elastin alterations and microangiopathy, the capacity for the diffusion of carbon monoxide is reduced.

An altered rate urinary protein excretion due to glomerular capillary injury also can be found. The aim of this study was to assess the presence of pulmonary complications. The values the proteins excretion rate and the diffusion capacity for carbon monoxide (DLco) corrected by alveolar volume were compared for this purpose. Also, they investigated the development of

retinopathy and its connection with the previously mentioned renal and pulmonary complications, because it is believed that they share a similar etiopathogenic mechanism.

11) Benbassat Carlos A, Ervin Stern, Mordechai Kramer, Joseph Lebzelter, Ilana Blum, Gershon Fink et al Studied -pulmonary function in patients with Diabetes mellitus.

Pulmonary complications of diabetes mellitus have been poorly characterized. Some have reported normal pulmonary function test others have found abnormal lung volumes, pulmonary mechanics and diffusing capacity.

The study was conducted in patients with DM using a combined cardiopulmonary exercise test in 27 patients with DM aged 48 ± 13 years. The FVC, FEV1 and FEF, MEF were within the predicted values, but the residual volumes and TLC ratio was slightly elevated. Comparison by diabetes type showed no significant differences in FEV1 and FEF, MEF, residual volumes and TLC ratio was significantly elevated in Type-1 DM compared with Type2-Diabetes mellitus. There was no correlation between the pulmonary function test and duration of disease, presence of microangiopathy or glycemic control.[32]

12) Dr.Mohankumar and Dr.S.Arulmozhi et al Studied –Pulmonary complications in elderly diabetics.In diabetes mellitus the total lung capacity,

lung volume and lung compliance are reduced, the central and peripheral airflows are reduced, and acceleration of aging process in pulmonary connective tissue are seen. There is interference with connective tissue cross-links and the presence of increased non-enzymatic glycosylation, and modification of alveolar surfactant action. The diffusing capacity for carbon monoxide is reduced because of pulmonary microangiopathy in diabetes mellitus.[44,7]

AIM OF THE STUDY

- ✓ To study the ventilatory function of individuals with type 2 diabetes mellitus by performing spirometry.
- ✓ To record the pulmonary function test in Type-2 diabetes mellitus and control group.
- ✓ To evaluate the impact of Type-2 DM on pulmonary functions by comparing with control groups.
- ✓ To correlate the spirometric values and variables(duration,FBS, PPBS,HbA1c) of diabetes.

MATERIALS AND METHODS

A case-control study, descriptive, prospective study of the lung function of diabetics compared with age and sex-matched non-diabetic controls.

Sample size:

The sample used in this study consisted of 100 subjects – 50 Diabetics, 50 Healthy Non-Diabetics.

Sampling procedure:

50 Diabetic individuals were recruited from those attending outpatient departments of Government Rajaji Hospital, Madurai. 50 Healthy Non-Diabetic individuals from the general population were taken as controls.

Ethical clearance:

Ethical clearance was obtained from Government Rajaji Hospital ethical committee for human research to conduct the study.

Inclusion criteria:

- ✓ Type 2 diabetes mellitus of more than 5 years duration
- ✓ able to give informed consent.

Exclusion criteria:

- × Smokers

- Present or past history of respiratory illness that might affect lung function such as asthms, COPD, tuberculosis, bronchiectasis, interstitial lung disease.
- History of occupational exposure to any substance that could affect lung function.
- Individuals with current or recent upper respiratory or lower respiratory infection, that could predispose to heightened airway reactivity.
- Individuals with unacceptable spirometric technique. An unacceptable spirometry was that in which FEV1 or FVC could not be correctly measured due to
    - ☒ Cough
    - ☒ Obstruction of teeth or toungue
    - ☒ Sub-maximal effort
    - ☒ Air escape
    - ☒ Effort sustained for less than 6 seconds duration
    - ☒ Lack of understanding of the procedure
    - ☒ Recent thoracic and abdominal surgery

Materials

Micro medical spirometer, weighing scale, stadiometer, Microsoft excel.

Methodology

Diabetics and controls were selected as per the criteria laid down. Their written consent was taken. The screening of diabetic subjects and control group was done for exclusion criteria. The history was elicited. Age, height, weight, BMI were recorded. Each subject was instructed to visit cardio respiratory laboratory with 6 hrs of fasting on a specific date, the blood samples [3ml volume] was drawn for estimation of FBS and glycated hemoglobin.

Performance of PFT by a patient

The performance of the pulmonary function test was demonstrated. The subjects and controls were made to undergo pulmonary function test using the

MICRO MEDICAL computerized Spirometer, for three times at every 15 minutes interval. The FVC, FEV1, PEF, FEV1/FVC% and FEF25-75% were recorded. And the best of the three was taken into account. The subject was asked to take breakfast and blood sample was drawn 2 hrs later for PPBS estimation.

The anthropometric, respiratory, blood glucose parameters and glycated Hb levels were recorded in their respective proforma.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2010)** developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATION AND RESULTS

A total number of 100 subjects were suitable for analysis. There were 50 diabetics (CASES) and 50 non-diabetic group (CONTROL)

A : PROFILE OF CASES STUDIED

Table 1 : Age distribution

Age group	Cases group		Control group	
	No	%	No	%
Upto 40 years	2	4	6	12
41-50 years	35	70	28	56
51-60 years	13	26	16	32
Total	50	100	50	100
Range	39-58 years		36-57 years	
Mean	46.6 years		47.48 years	
SD	5.01 years		5.21 years	
'p'	0.6123 Not significant			

The Diabetes group had an age of 46.6 +5.01 years and the Control group 47.48 +5.21 years. There was no statistically significant difference.

Fig 6 : Age distribution

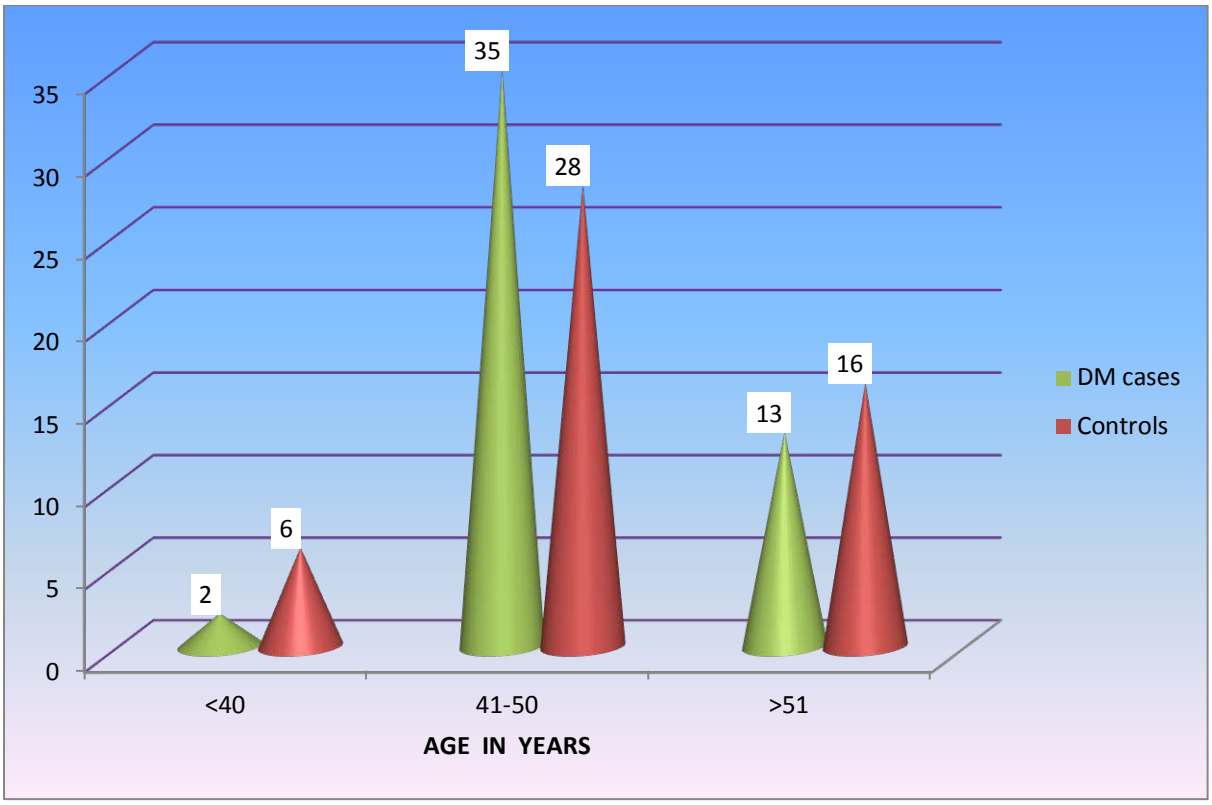


Table 2 : Sex distribution

Sex	DM Cases group		Control group	
	No	%	No	%
Male	27	54	28	56
Female	23	46	22	44
Total	50	100	40	100
'p'	0.9193			
	Not significant			

Sex distribution of the study group and control group did not have any significant difference (p = 0.9193).

Fig 7 : Sex distribution

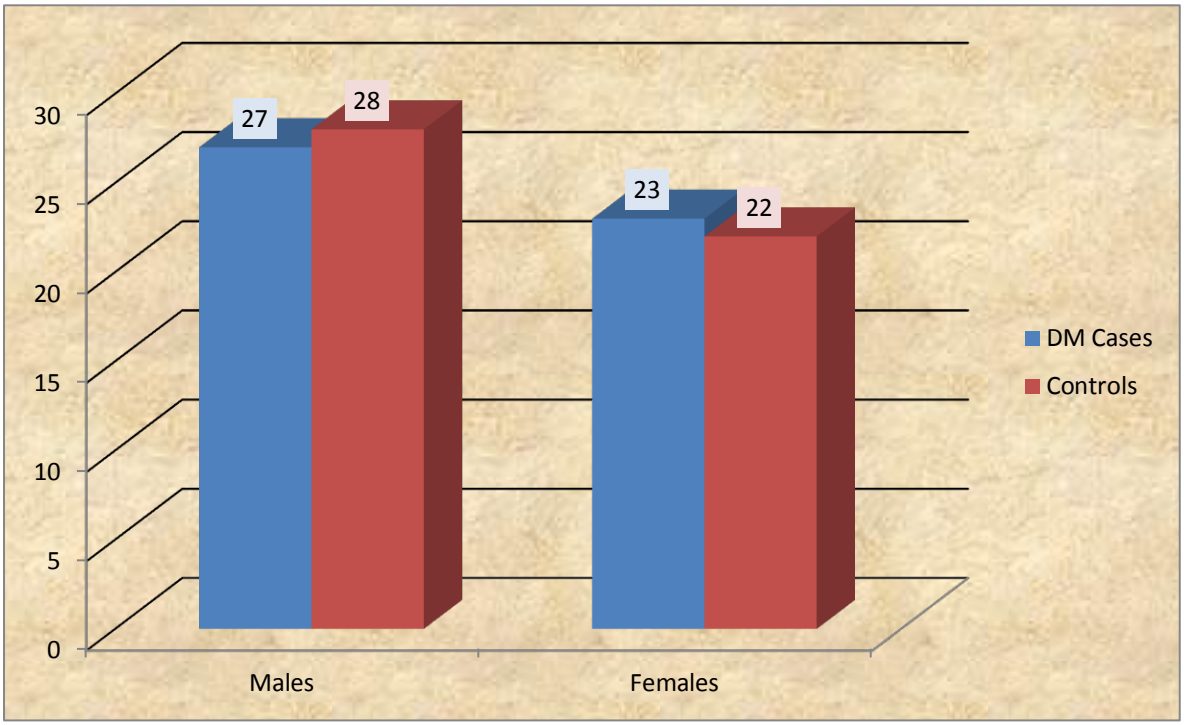


Table 3 : Physiological variables

Variable	DM Cases group		Control group		'p'
	Mean	**SD**	**Mean**	**SD**	
Height (in cms)	164.92	6.04	162.34	7.85	0.6642 Not significant
Weight (in kgs)	66.30	8.70	67.14	10.51	0.646 Not significant
BMI	24.30	2.2	25.69	4.9	0.762 Not significant

Height ,weight and BMI of the control and Diabetic cases studied did not have any significant difference ($p > 0.05$).

Fig 8 : Physiological variables

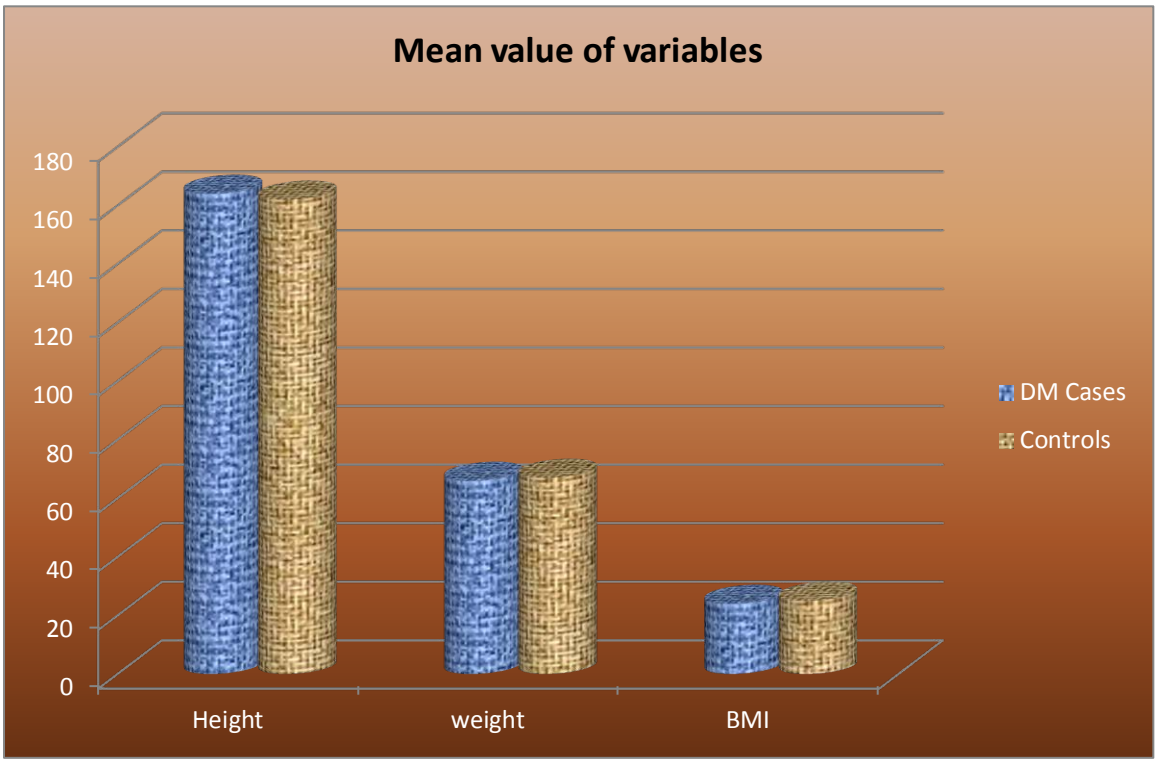


Table 4 : Blood Sugar levels

Blood Sugar	DM Cases group		Control group		'p'
	Mean	SD	Mean	SD	
Fasting	193.12	53.70	88.00	10.12	**0.0001 Significant**
Post prandial	267.16	63.28	125.74	11.31	**0.0001 Significant**

The P value for the basic characters FBS and PPBS is <0.0001 is significant.

Table 5 : Duration of Diabetic cases

Duration in years	No.of cases	% of cases
5-6	14	28%
7-8	17	34%
9-10	14	28%
>10	5	10%

Fig 9 : Mean blood sugar values between cases and controls

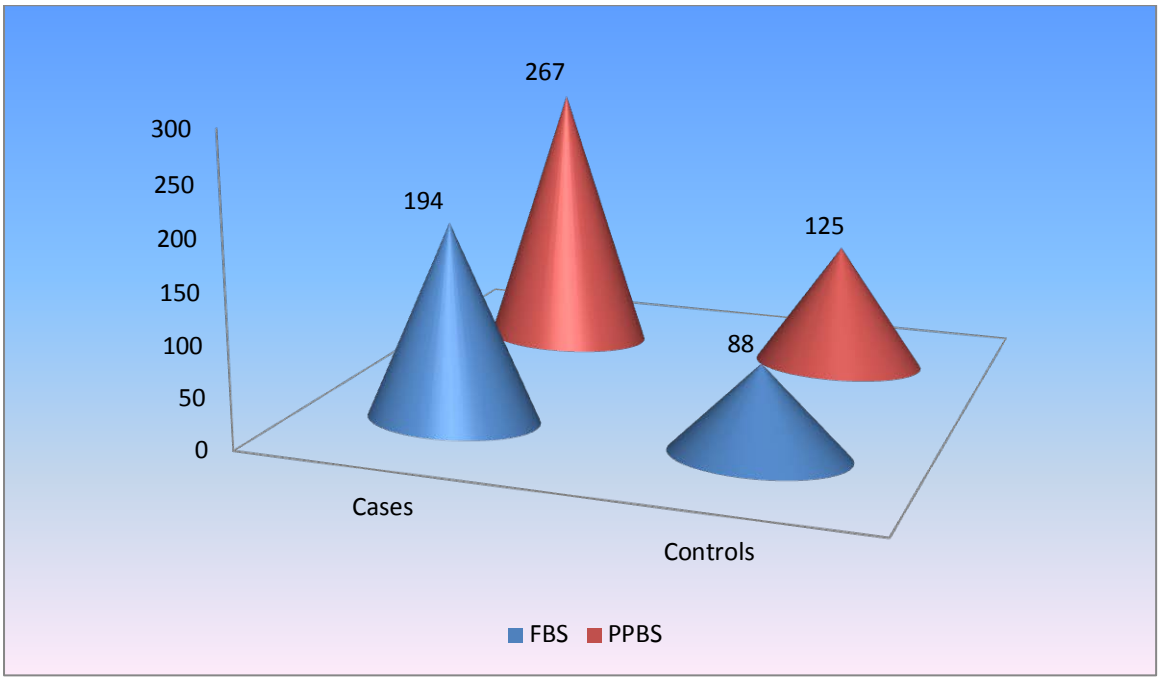
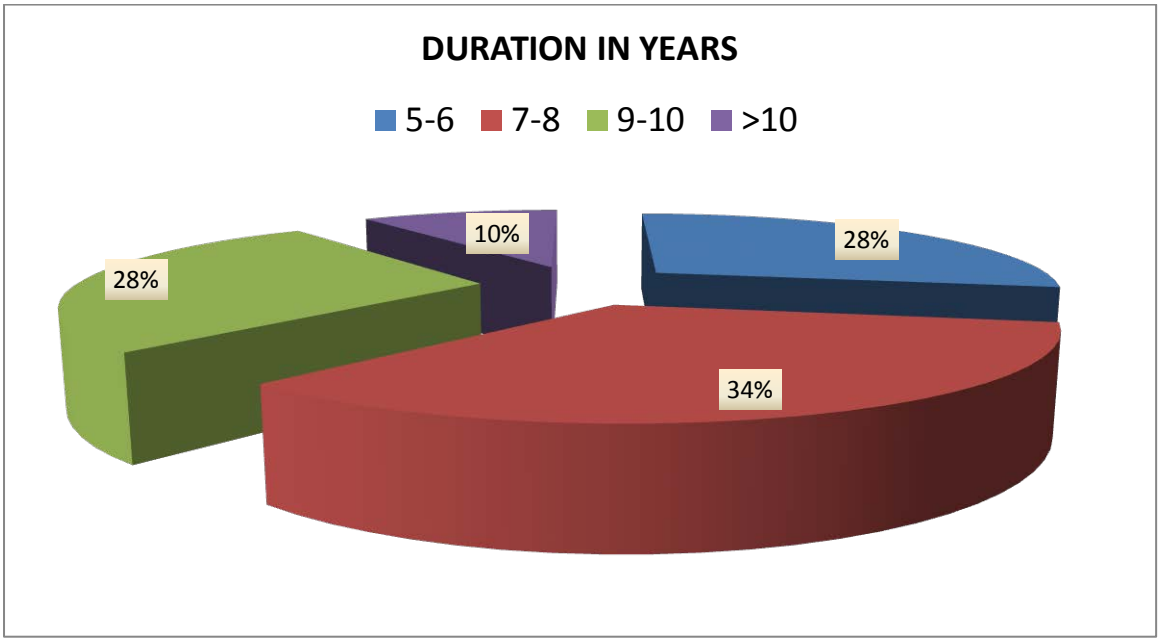


Fig 10 : Duration of Diabetic cases



The pulmonary function test was conducted on 50 Type-2 diabetics with a history of diabetes for more than 5 yrs duration and 50 healthy individuals. An attempt was made to evaluate the effect of Type-2 DM on pulmonary functions. The effect of extent of diabetes status as reflected by FBS, PPBS, HB1Ac and duration on pulmonary function was also evaluated. The pulmonary function tests recorded were FVC, FEV1, FEV1/FVC%, PEF and FEF25%-75%.

The basic character's Age, sex, height, weight, BMI, FBS and PPBS of cases and controls, are shown in the table 3&4. The P value for the basic characters FBS and PPBS is <0.05 which is significant, however the basic character's age, sex, ht, wt, BMI and P value is >0.05 which is not significant.

B : Effect of Type-2 diabetes mellitus on pulmonary function test

Table 6: Observed Spirometric results

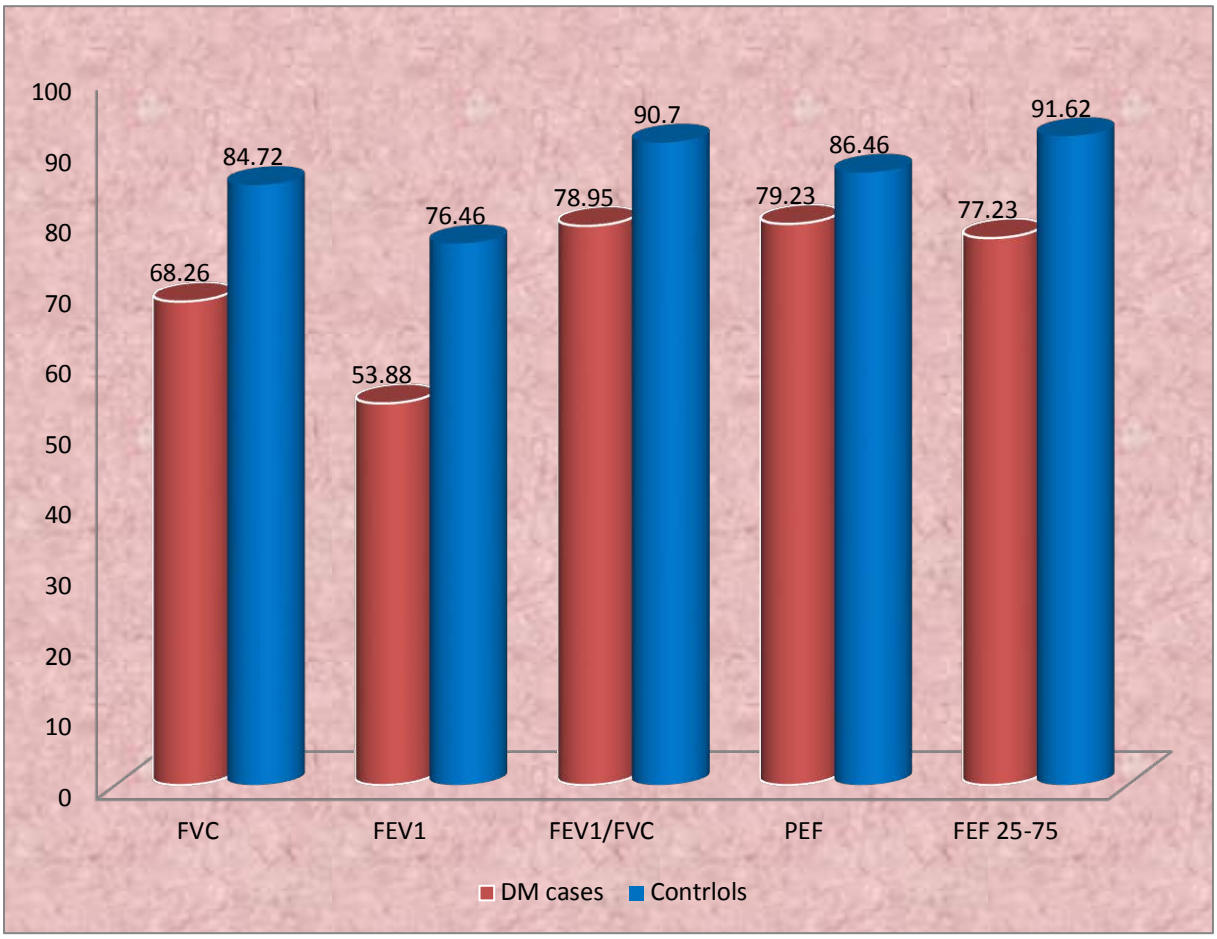
Pulmonary function test	DM Cases group (n=50)		Control group (n=50)		P Value	Significance
	Mean	SD	Mean	SD		
FVC	2.67	0.48	3.25	0.24	0.0001	**Significant**
FEV1	1.68	0.37	2.32	0.38	0.0001	**Significant**
FEV1/FVC Ratio	62.75	6.9	72.02	11.01	0.0001	**Significant**
PEF	400.52	118.92	448.94	105.40	0.0336	**Significant**
FEF 25-75	2.34	0.55	2.69	0.53	0.0022	**Significant**

Table 7 : % Predicted values of PFT

Pulmonary function test	DM Cases group (n=50)		Control group (n=50)		P Value	Significance
	Mean	SD	Mean	SD		
FVC	68.26	16.7	84.72	13.4	0.0001	**Significant**
FEV1	53.88	15.03	76.46	13.84	0.0001	**Significant**
FEV1/FVC Ratio	78.95	8.2	90.7	13.21	0.0001	**Significant**
PEF	79.23	17.69	86.46	10.78	0.0155	**Significant**
FEF 25-75	77.23	19.73	91.62	20.13	0.005	**Significant**

Spirometric values were consistently lower in Diabetic case groups than control groups. However, the differences were statistically highly significant for FVC,FEV1 &FEV1/FVC ratio (p=0.0001) and moderately significant for PEF & FEF 25-75%.(p=0.0155,p=0.005)

Fig11:Comparison of spiromtric values among Diabetics and Non Diabetics



Mean %predicted spirometric values among Diabetics and Non-Diabetics

C: Relationship between spirometric values and other variables in Diabetics

PFT and DURATION

Table 8 : Duration and Observed Spirometric results

Pulmonary function test	Duration of DM group in years (Mean ± SD)				P Value	Significance
	5-6 (n=14)	7-8 (n=17)	9-10 (n=14)	>10 (n=5)		
FVC	2.85±0.63	2.68±0.38	2.28±0.34	2.02±0.42	0.001	**Significant**
FEV1	1.94±0.44	1.76±0.39	1.57±0.22	1.28±0.12	0.003	**Significant**
FEV1/FVC	78.86±5.75	73.83±4.92	74.69±5.36	71.86±4.71	0.003	**Significant**
PEF	484.99±121.91	417.41±93.8	326.36±84.9	314.4±107.86	0.001	**Significant**
FEF25-75	2.60±0.53	2.50±0.6	2.30±0.44	1.84±0.54	0.004	**Significant**

Observed spirometric values were inversely related to the duration of diabetes. as the duration increases the spirometric values were consistenly decreased, which is highly significant for all parameters. ($p<0.005$)

Table 9 : Duration and% Predicted values of PFT

Pulmonary function test	Duration of DM group in years (Mean ± SD)				P Value	Significance
	5-6 (n=14)	7-8 (n=17)	9-10 (n=14)	>10 (n=5)		
FVC	68.66±18.19	68.14±17.68	65.09±11.26	52.09±5.75	0.019	**Significant**
FEV1	57.78±15.15	55.44±16.5	52.74±11.25	56.78±16.06	0.05	**Significant**
FEV1/FVC	78.86±5.75	75.26±5.19	73.84±4.92	71.86±4.71	0.032	**Significant**
PEF	88.45±13.48	77.64±17.62	69.7±16.36	63.94±9.08	0.006	**Significant**
FEF25-75	71.66±21.44	81.15±19.68	73.84±4.92	71.86±4.71	0.059	**Not Significant**

There was a negative correlation between the spirometric parameters and the duration of diabetes, which is statistically significant for all parameters except FEF 25-75%.

Fig 12 : Duration and% Predicted values of PFT

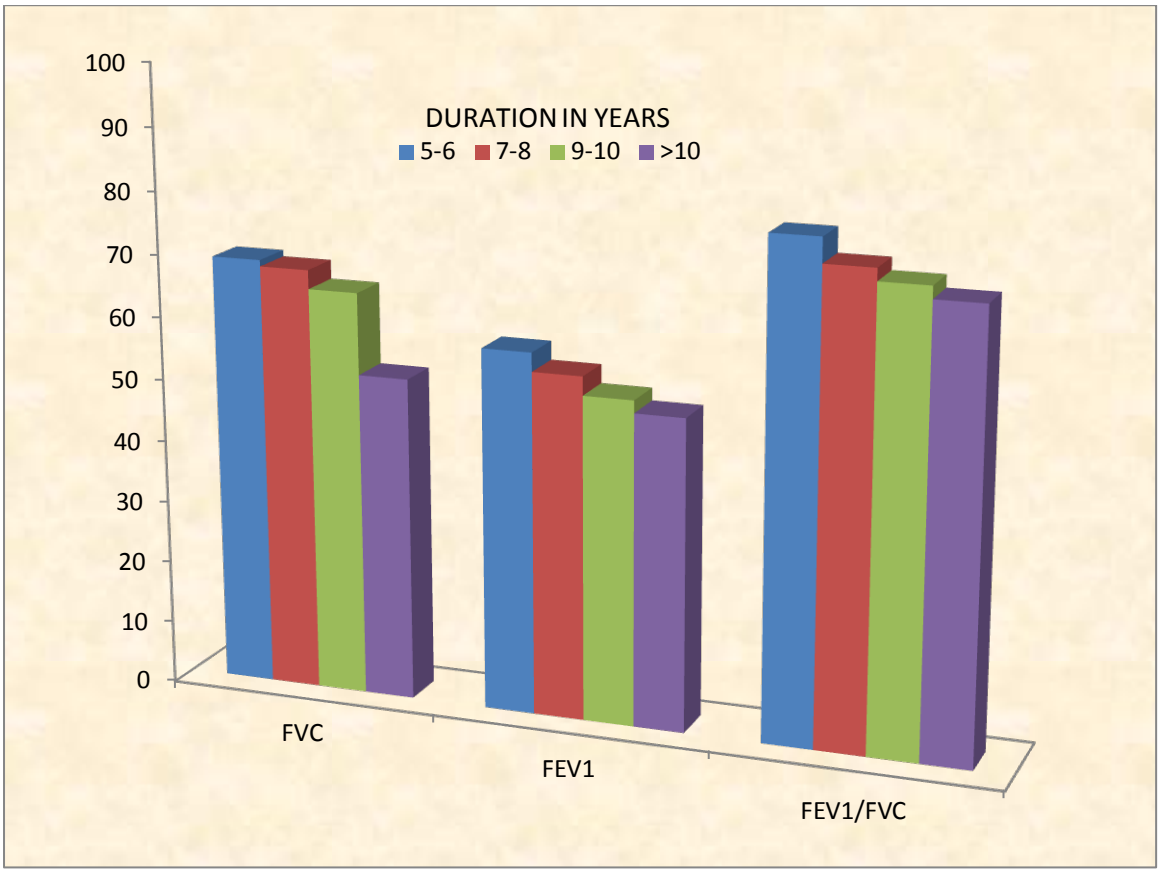
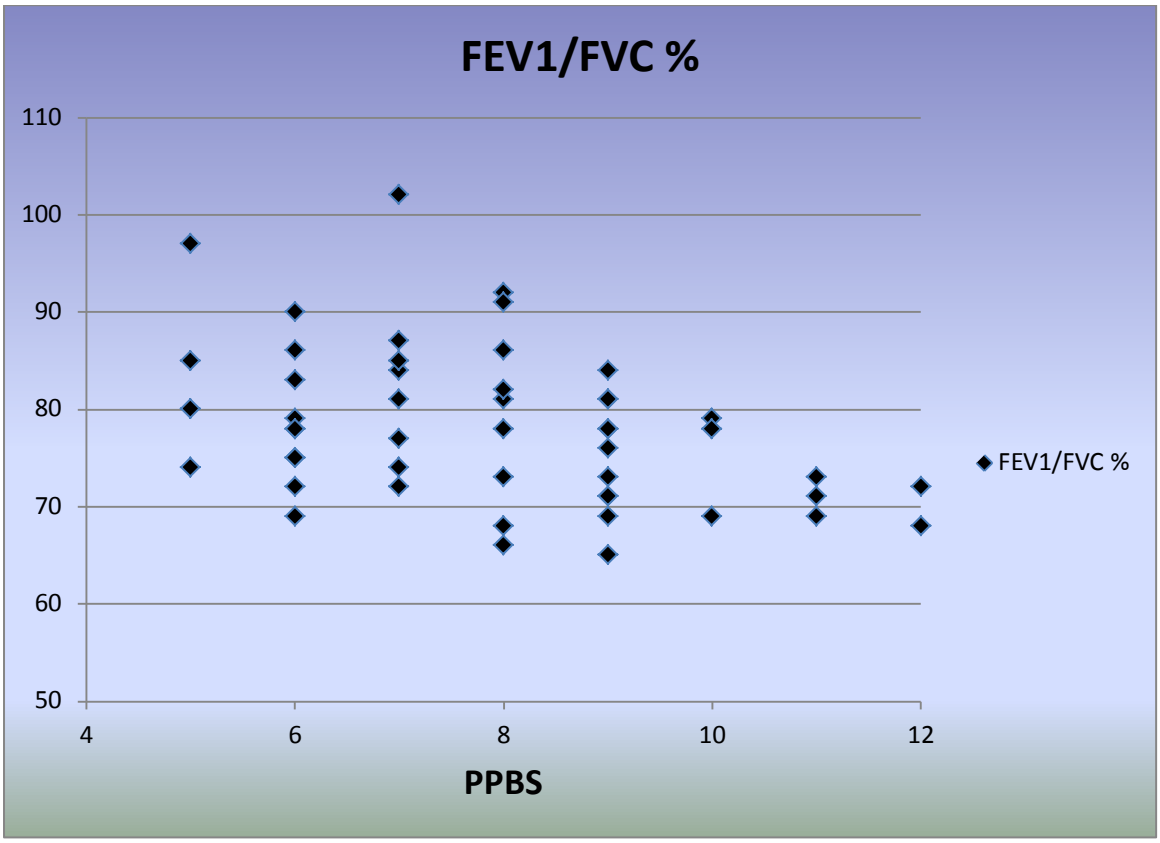


Fig 13 : Comparison of % Predicted FEV1/FVC with duration of Diabetics



PFT AND FASTING BLOOD SUGAR LEVEL

Table 10 : FBS Level and Observed Spirometric results

Pulmonary function test	FBS (mg/dl) in DM group (Mean ± SD)			P Value	Significance
	<130 (n=8)	131-200 (n=23)	201-300 (n=19)		
FVC	3.24±0.84	2.64±0.66	2.22±0.58	0.011	**Significant**
FEV1	2.53±1.29	1.81±0.44	1.68±0.29	0.012	**Significant**
FEV1/FVC	84.85±1.76	76.11±5.75	73.8±4.59	0.001	**Significant**
PEF	467.25±140.49	415.26±120.2	355±96.11	0.046	**Significant**
FEF25-75	2.94±1.42	2.43±0.62	2.33±0.51	0.25	**Not Significant**

Observed spirometric values were inversely related to the Fasting blood sugar level of diabetes. as the blood sugar level increases the spirometric values were consistenly decreased, which is statistically significant.

Table 11 : FBS level and% Predicted values of PFT

Pulmonary function test	FBS (mg/dl) in DM group (Mean ± SD)			P Value	Significance
	<130 (n=8)	131-200 (n=23)	201-300 (n=19)		
FVC	70.2±4.28	69±18.27	63.74±12.86	0.005	**Significant**
FEV1	57.29±2.7	56.11±15.22	55.81±13.56	0.009	**Significant**
FEV1/FVC	78.9±1.7	76.11±5.74	74.85±6.04	0.04	**Significant**
PEF	76.27±21.8	80.73±18.28	72.35±14.85	0.279	**Not Significant**
FEF25-75	61.7±19.9	76.53±19.79	82.54±18.91	0.148	**Not Significant**

Poor glycemic control were reflected in Pulmonary function test as fasting blood sugar level increases the spirometric values were decreased with significant p value for FVC,FEV1 and FEV1/FVC ratio.

Fig 14 : FBS level and% Predicted values of PFT

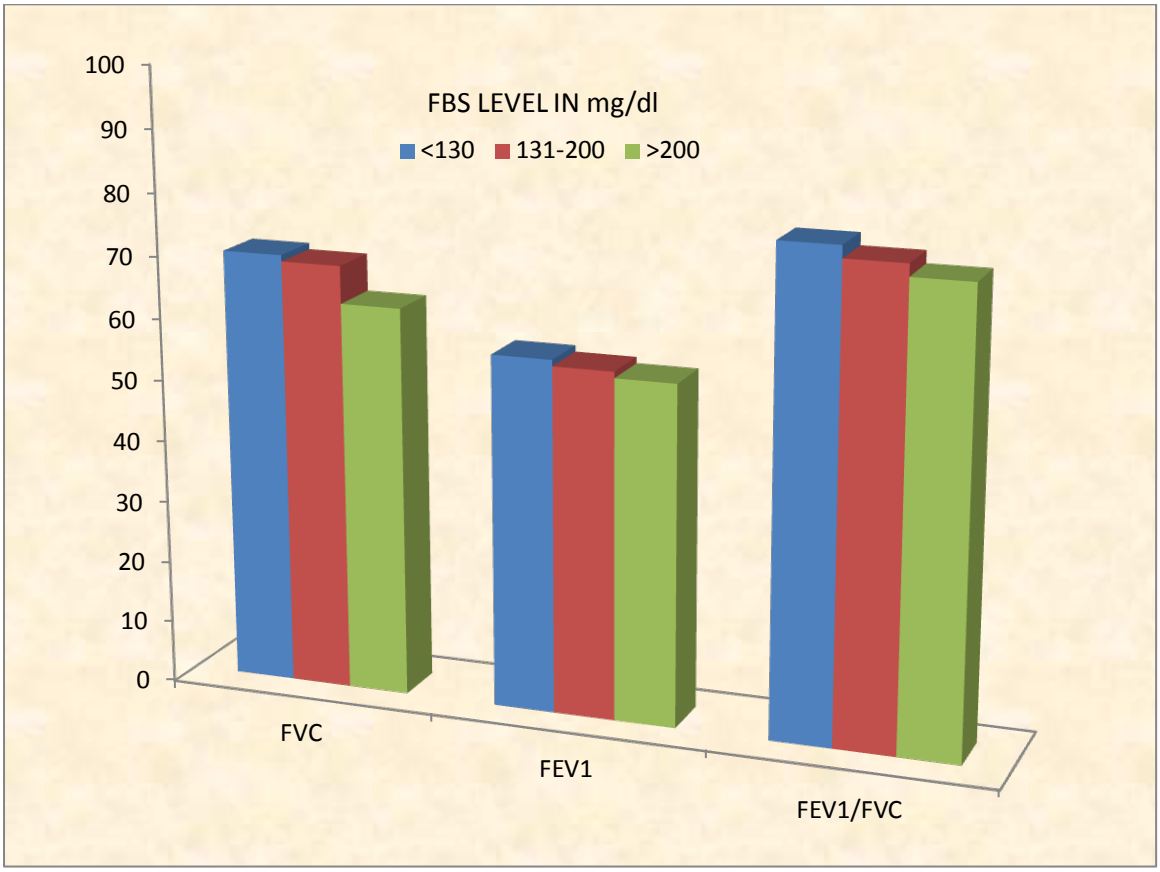
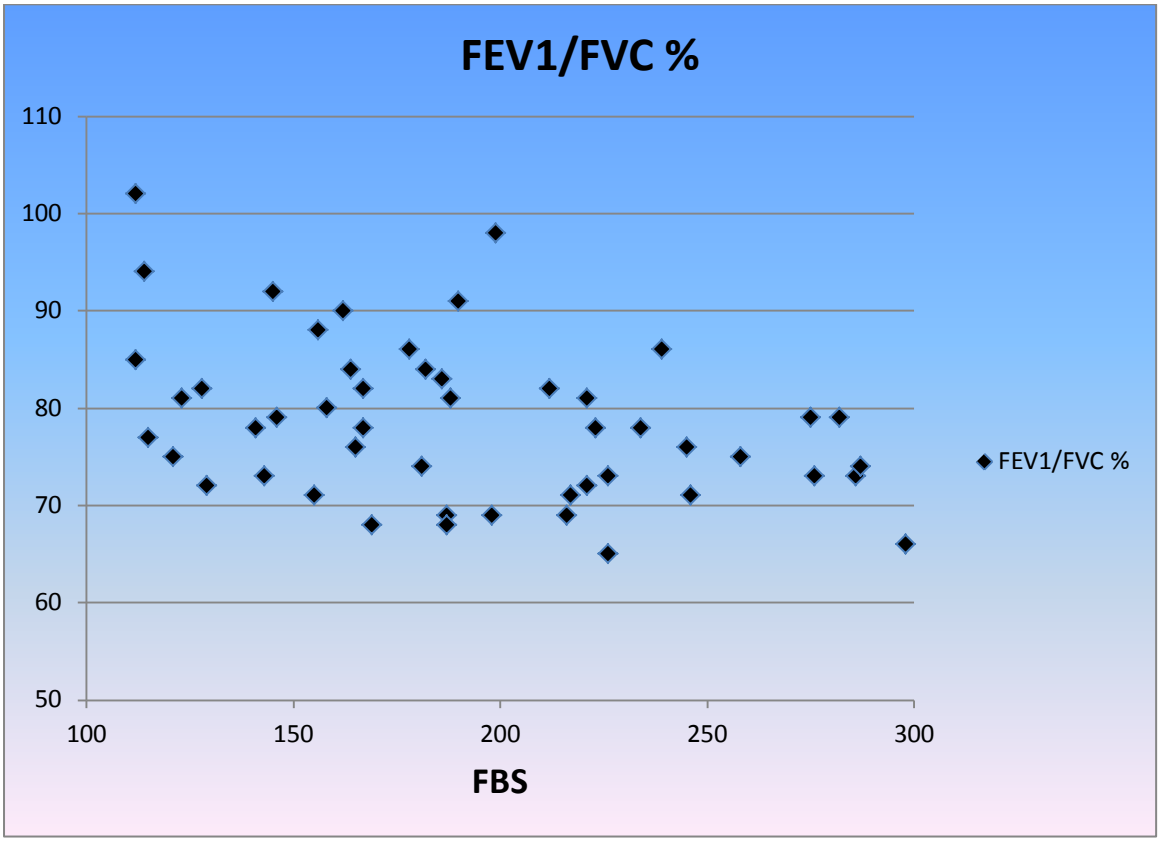


Fig 15 : Comparison of FEV1/FVC ratio with FBS among Diabetics



PFT AND POSTPRANDIAL BLOOD SUGAR LEVEL

Table 12 : PPBS Level and Observed Spirometric results

Pulmonary function test	PPBS (mg/dl) in DM group (Mean ± SD)			P Value	Significance
	<200 (n=10)	201-300 (n=26)	>300 (n=14)		
FVC	2.7±0.83	2.55±0.57	2.36±0.41	0.003	**Significant**
FEV1	2.23±0.59	1.77±0.39	1.57±0.36	0.002	**Significant**
FEV1/FVC	80.1±5.63	76.11±5.03	74.04±4.87	0.002	**Significant**
PEF	444.69±140.03	362.1±113.72	317.36±98.8	0.036	**Significant**
FEF25-75	2.63±0.55	2.21±0.43	2.11±0.34	0.014	**Significant**

High post prandial blood sugar levels were also associated with Low spirometric values, which is highly significant for all parameters of spirometry with the p value of <0.05.

Table 13 : PPBS level and% Predicted values of PFT

Pulmonary function test	PPBS (mg/dl) in DM group (Mean ± SD)			P Value	Significance
	<200 (n=10)	201-300 (n=26)	>300 (n=14)		
FVC	67.77±14.48	65.67±15.7	63.65±18.97	0.0180	**Significant**
FEV1	60.63±12.66	54.97±14.04	49.55±15.70	0.0178	**Significant**
FEV1/FVC	75.10±3.88	73.73±5.6	71.47±6.91	0.028	**Significant**
PEF	73.19±21.59	80.65±16.74	73.61±15.24	0.349	**Not Significant**
FEF25-75	67.39±13.85	74.45±17.9	90.84±21.16	0.006	**Significant**

The % predicted values of spirometry and postprandial blood sugar levels were statistically significant for FVC,FEV1,FEV1/FVC with $p<0.05$, which indicate poor glycemic control were reflected in pulmonary functions with the decline of spirometric parameters.

Fig 16 : PPBS level and% Predicted values of PFT

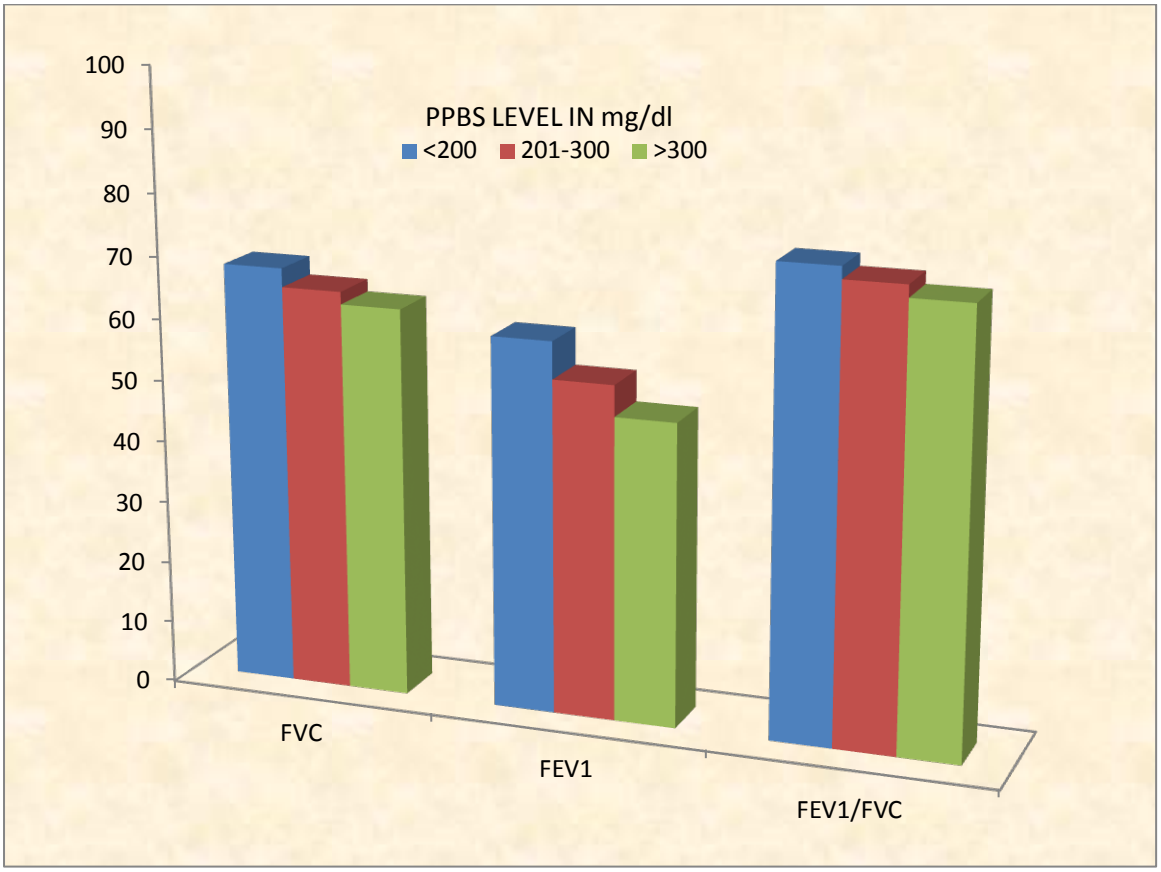
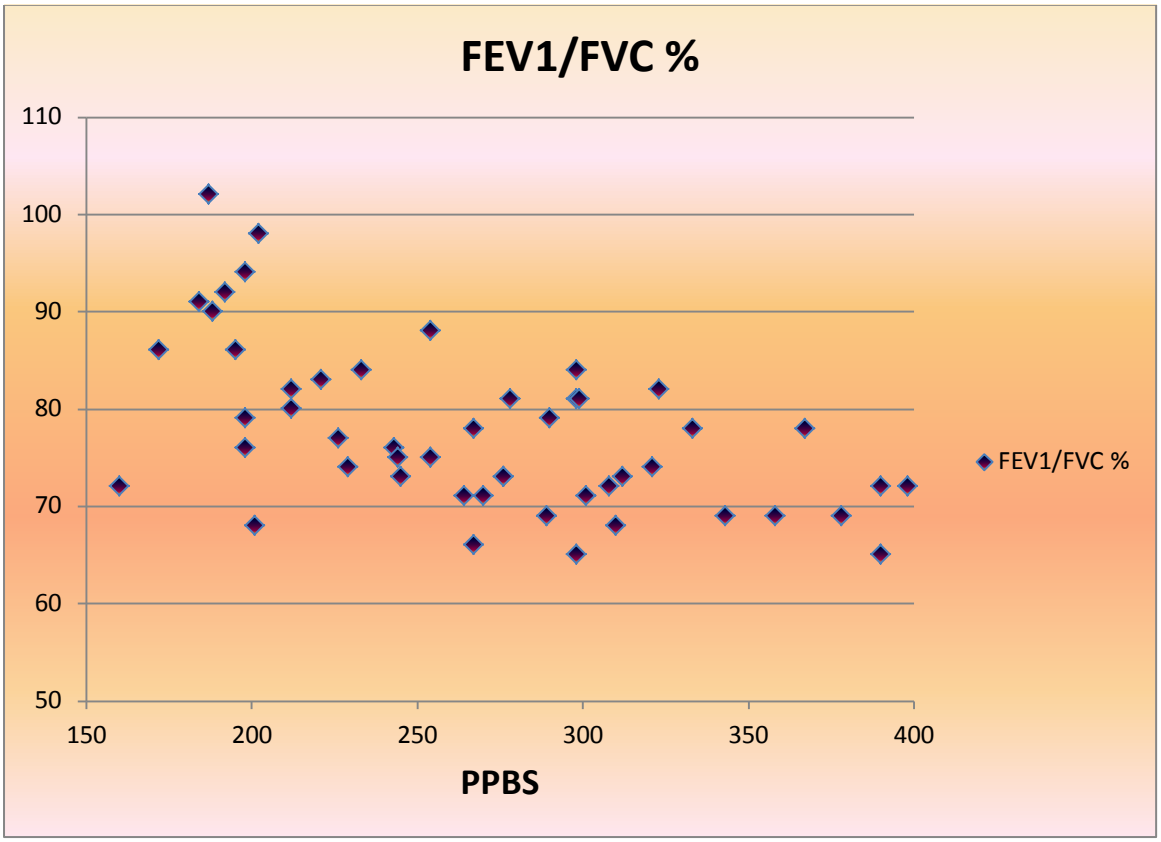


Fig 17 : Comparison of %Predicted FEV1/FVC with PPBS among Diabetics



PFT AND HbA1c LEVEL

Table 14 : HbA1c Level and Observed Spirometric results

Pulmonary function test	**HbA1c (mg/dl) in DM group (Mean ± SD)**			**P Value**	**Significance**
	5-7(n=18)	**7.1-9(n=24)**	**>9(n=8)**		
FVC	2.75±0.60	2.49±0.48	1.81±0.66	0.001	**Significant**
FEV1	1.98±0.47	1.76±0.33	1.53±0.20	0.019	**Significant**
FEV1/FVC	64.35±5.28	61.08±5.54	58.67±3.4	0.028	**Significant**
PEF	458.83±140.81	371.54±92.43	356.25±94.28	0.029	**Significant**
FEF25-75	2.57±0.54	2.37±0.53	2.14±0.59	0.59	**Not Significant**

Observed spirometric results were negatively correlated with HbA1c levels with significant p (<0.05) value for FVC, FEV1, FEV1/FVC,PEF. Which indicates poor glycemic control was associated with pulmonary functions.

Table 15 : HbA1c level and% Predicted values of PFT

Pulmonary function test	HbA1c (mg/dl) in DM group (Mean ± SD)			P Value	Significance
	5-7(n=18)	7.1-9(n=24)	>9(n=8)		
FVC	66.16±16.85	59±16.21	59.81±13.9	0.034	**Significant**
FEV1	58.67±15.15	53.29±15.07	51.06±10.49	0.039	**Significant**
FEV1/FVC	76.86±5.76	75.08±5.96	73.9±3.85	0.040	**Significant**
PEF	84.83±19.32	74.21±14.23	68.91±17.15	0.046	**Significant**
FEF25-75	75.93±20.98	79.92±19.68	75.45±19.84	0.78	**Not Significant**

Poor glycemic control were also reflected in Pulmonary function test as HbA1c level increases the spirometric values were decreased with significant p value for FVC,FEV1 and FEV1/FVC ratio.

Fig 18 : HbA1c level and% Predicted values of PFT

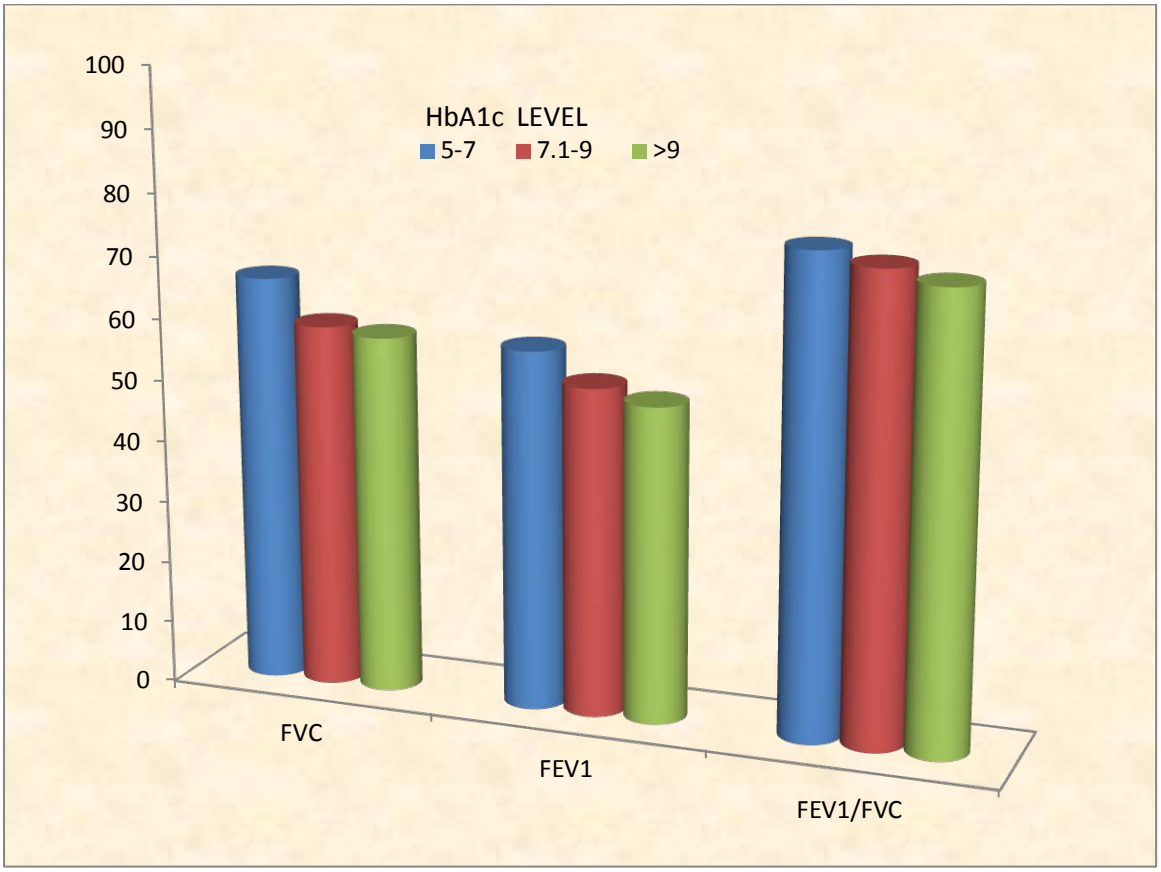
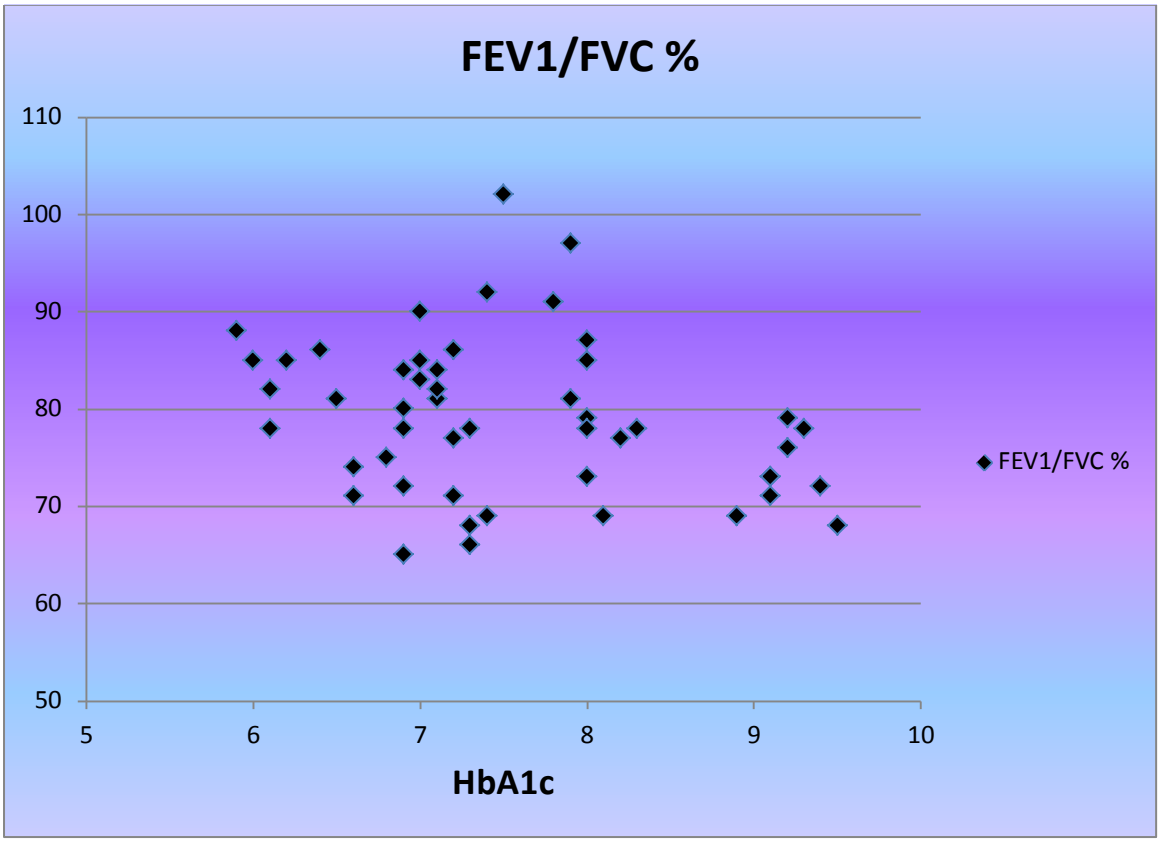


Fig 19 : Comparison of %Predicted FEV1/FVC with HbA1c among Diabetics



DISCUSSION

This study was undertaken to assess the pulmonary function of type 2 diabetes mellitus patients, and to compare it with those of non-diabetic healthy subjects. Few studies have focused on the relationship between pulmonary function and diabetes.

The different groups i.e., cases and controls were comparable in terms of age, height, weight and BMI. These being the major determinants of the spirometric values, the main determinant of spirometry were likely to be the presence or absence of diabetes.

The groups were also homogeneous in respect of having no known respiratory disease, and all being non-smokers. In the study of Hirishi Mori et al, smokers were included in the analysis, and this was therefore an additional confounding variable.

The effect of diabetes in pulmonary function tests were observed and the Spirometric values were consistently lower in Diabetic case groups than control groups.

The present study is in agreement with Walter.E.Robert et al who studied the relationship between diabetes mellitus and pulmonary function and showed a decrease in FVC by 109ml and FEV1 by 27ml in diabetes mellitus.[27] Davis M.E. Timothy studied the pulmonary function and its association with Type-2 diabetes mellitus and showed an average decrease of 9.5% in FVC and FEV1 of diabetics.[29] His study was also showed a 1.5% increase in the FEV1/FVC %[27] with a P value <0.05% which is statically significant. Suggesting a restrictive pattern of ventilatory impairment.

In SK Rajan's study, spirometric readings of study group patients revealed that 60% showed an obstructive pattern, 30% showed a restrictive pattern, and 10% showed a mixed pattern.[37]

In our study, we found a predominantly restrictive pattern with a FVC & FEV1 were <80% of the predicted value and FEV1/FVC ratio were >70% of the predicted.

Duration and other parameters were compared with all values of spirometry, during which the following results were observed:

1) There was tendency for all parameters to fall with longer duration of diabetes. However, a multiple regression analysis showed that this was

not significant. Clearly, those with a longer duration of diabetes also were older, and the effect of decling in lung function with age was a greater contributing factor.

2) Poor diabetic control was associated with poorer lung function. There was a association between greater declines in FVC,FEV1 &FEV1/FVC and higher values of FBS and PPBS.
3) A similar inverse association was noted between higher HbA1c levels and lower spirometric values.

In the study of Marco Guzzi et al, absolute values and percentage of predicted normal values of FEV1, MVV, vital capacity and total lung capacity were reduced in NDDIM patient group. DLco showed a step wise highly significant reduction from normal to hyperglycaemic.[35]

In Hiroshi Mori study people says %DLco was negatively correlated with duration of diabetics, but other PFTs like %VC, FEV1 or PaO2 did not show such a negative correlation.[36]

In P Lange's study, the diabetic subjects had slightly smaller height adjusted FEV1 & FVC compare values of non-diabetic subjects, their regression analysis also, showed association between raised values of plasma glucose and reduction of the lung function was highly significant.[28]

CONCLUSION

The present study was undertaken to resolve conflict between two schools of thought, one expounding impact of Type-2 diabetes mellitus on respiratory system and another non-impact. Pulmonary functions in Type-2 diabetes mellitus and controls were statistically compared to resolve this. The intra diabetic subgroups- FBS wise, PPBS wise, duration wise and HbA1c were correlated to pulmonary functions to find out the impact of Type-2 diabetes mellitus on respiratory system. This study confirms the following features.

1) The pulmonary functions FVC, FEV1, PEF and FEF25%-75% are decreased in Type-2 diabetes mellitus compared to controls. FEV1/FVC% increased in Type-2 diabetes mellitus, which is indicative of restrictive disorder of the lung.
2) There were negative correlation between FBS levels and pulmonary functions FVC, FEV1 and FEV1/FVC. Linear relationship exists between increasing FBS and FEV1/FVC%, which is indicative of restrictive disorder of the lung.
3) Poor glycemic control were reflected in pulmonary functions with the decline of spirometric parameters associated with high PPBS levels.

Linear relationship exists between increasing PPBS and FEV1/FVC%, which is indicative of restrictive disorder of the lung.

4) There were negative correlation between duration of diabetes mellitus and pulmonary functions FVC, FEV1, PEF and FEF25%-75%. Linear relationship exists between increasing duration and FEV1/FVC%, which is indicative of restrictive disorder of the lung.

5) Poor glycemic control were also reflected in Pulmonary function test as HbA1c level increases the spirometric values FVC, FEV1, FEV1/FVC, were consistently decreased.

The above mentioned effects of Type-2 diabetes mellitus on pulmonary functions are all due to the alterations in pulmonary connective tissue, thickening of basement membrane of capillary and alveolus, modification of surfactant, decreased recoiling tendency of lung and decreased muscle endurance.

LIMITATIONS OF THE STUDY

- The study population was small
- Subjects declaration of being non-smokers was accepted at face value, we were not able to confidently exclude previous smoking or other irritant fume exposure, unless declared by our subjects.
- Cardiac failure was excluded only by history and examination,and not by echocardiography. It is known that mild pulmonary congestion can cause spirometric abnormalities.
- Diffusion studies were not performed on our subjects.this was one of the main parameters affected according to other studies on the same subjects.

BIBLIOGRAPHY

1. Larsen, Kronenberg, Melmed, Polonsky, Williams textbook endocrinology.10th ed. Pennsylvinia: Elsevier India Publisher; 2003.
2. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. Jan 2010;33 Suppl 1:S62-9.
3. Boden G. Fatty acids and insulin resistance. *Diabetes Care*. Apr 1996;19(4):394-5.
4. Bacha F, Lee S, Gungor N, Arslanian SA. From pre-diabetes to type 2 diabetes in obese youth: pathophysiological characteristics along the spectrum of glucose dysregulation. *Diabetes Care*. Oct 2010;33(10):2225-31.
5. Hansen KB, Vilsbøll T, Bagger JI, Holst JJ, Knop FK. Increased postprandial GIP and glucagon responses, but unaltered GLP-1 response after intervention with steroid hormone, relative physical inactivity, and high-calorie diet in healthy subjects. *J Clin Endocrinol Metab*. Feb 2011;96(2):447-53.

6. Chiefari E, Tanyolaç S, Paonessa F, Pullinger CR, Capula C, Iiritano S, et al. Functional variants of the HMGA1 gene and type 2 diabetes mellitus. *JAMA.* Mar 2 2011;305(9):903-12.
7. Dr.Mohankumar, Dr.S.Arulmozhi. Diabetes in elderly: Pulmonary complications in elderly diabetics. Micro labs Ltd.Bangalore:2005;119-125.
8. Wei GS, Coady SA, Goff DC Jr, et al. Blood Pressure and the Risk of Developing Diabetes in African Americans and Whites: ARIC, CARDIA, and the Framingham Heart Study. *Diabetes Care.* Apr 2011;34(4):873-9.
9. Stern MP. Do non-insulin-dependent diabetes mellitus and cardiovascular disease share common antecedents?. *Ann Intern Med*. Jan 1 1996;124(1 Pt 2):110-6.
10. Haffner SM, D'Agostino R Jr, Mykkänen L, Tracy R, Howard B, Rewers M, et al. Insulin sensitivity in subjects with type 2 diabetes. Relationship to cardiovascular risk factors: the Insulin Resistance Atherosclerosis Study. *Diabetes Care*. Apr 1999;22(4):562-8.
11. Philippe MF, Benabadji S, Barbot-Trystram L, et al. Pancreatic volume and endocrine and exocrine functions in patients with diabetes. *Pancreas*. Apr 2011;40(3):359-63.

12. Murphy HR, Steel SA, Roland JM, et al. Obstetric and perinatal outcomes in pregnancies complicated by Type 1 and Type 2 diabetes: influences of glycaemic control, obesity and social disadvantage. *Diabet Med*. Sep 2011;28(9):1060-7.
13. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. Jan 10 2004;363(9403):157-63.
14. Wei GS, Coady SA, Goff DC Jr, et al. Blood Pressure and the Risk of Developing Diabetes in African Americans and Whites: ARIC, CARDIA, and the Framingham Heart Study. *Diabetes Care*. Apr 2011;34(4):873-9.
15. Dabelea D, Pettitt DJ, Hanson RL, Imperatore G, Bennett PH, Knowler WC. Birth weight, type 2 diabetes, and insulin resistance in Pima Indian children and young adults. *Diabetes Care*. Jun 1999;22(6):944-50.
16. Yarbrough DE, Barrett-Connor E, Kritz-Silverstein D, Wingard DL. Birth weight, adult weight, and girth as predictors of the metabolic syndrome in postmenopausal women: the Rancho Bernardo Study. *Diabetes Care*. Oct 1998;21(10):1652-8.
17. [Best Evidence] Wang J, Luben R, Khaw KT, Bingham S, Wareham NJ, Forouhi NG. Dietary energy density predicts the risk of incident type 2 diabetes: the European Prospective Investigation of Cancer (EPIC)-Norfolk Study. *Diabetes Care*. Nov 2008;31(11):2120-5.

18. Hectors TL, Vanparys C, van der Ven K, et al. Environmental pollutants and type 2 diabetes: a review of mechanisms that can disrupt beta cell function. *Diabetologia*. Jun 2011;54(6):1273-90.

19. Kilpelainen TO, Zillikens MC, Stancakova A, et al. Genetic variation near IRS1 associates with reduced adiposity and an impaired metabolic profile. *Nat Genet*. Jun 26 2011;43(8):753-60.

20. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, 2011. National diabetes fact sheet: national estimates and general information on diabetes and prediabetes in the United States, 2011.

21. Sandler Malcom.Is the Lung a 'Target Organ' in Diabetes Mellitus. Arch Intern Med.1990;150:1385-1388.

22. Wallace O Fenn, Hermann Rahn. Hand book of Phsiology.3rd ed; 45-48.

23. Johnson DC. Importance of adjusting carbon monoxide diffusing capacity (DLCO) and carbon monoxide transfer coefficient (KCO) for alveolar volume. *Respir Med.* Jan 2000;94(1):28-37.

24. Macintyre N, Crapo RO, Viegi G,Johnson DC,van der Grinten CP, Brusasco V, et al. Standardisation of the single-breath determination of carbon monoxide uptake in the lung. *Eur RespirJ.*Oct 2005;26(4):720-35.

25. American Thoracic Society/European Respiratory Society. ATS/ERS Statement on respiratory muscle testing. *Am J Respir Crit Care Med*. Aug 15 2002;166(4):518-624.

26. Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. population. *Am J Respir Crit Care Med*. Jan 1999;159(1):179-87.

27. Walter.E.Robert, Alexa Beiser, Rachel J, Givelber, George T, O'Connor, et al. Association between glycemic state and lung function. Am J Respir Crit Care Med 2003;167:911-916.

28. lange P.Diabetes mellitus, plasma glucose and lung function in a cross sectional population study.Eur Respir J 1989:2:14-19

29. Davis A Wendy, Matthew Knuiman, Peter Kendell, Valerie Grange, Timothy M.E.Davis.Glycemic Exposure is Associated with Reduced Pulmonary Function in Type-2 Diabetes.Diabetes care 2004:27:752-757.

30. Davis Timothy M. E, Matthew Knuiman, Peter Kendell, Hien Vu, Wendy A. Davis. "Reduced pulmonary function and its associations in type 2 diabetes: Diabetes research and Clinical practice 2000; 50:152-159.

31. Sreeja C.K, Elzabeth Samuel, C.Kesava chandran, Shankar Shashidaran. Pulmonary function in patients with diabetes mellitus. IJPP 2003; 47(1): 87-93.

32.Benbassat Carlos A, Ervin Stern, Mordechai Kramer, Joseph Lebzelter, Ilana Blum, Gershon Fink. Pulmonary function in patients with Diabetes Mellitus. The Am J Med Sci 2001; 322(3):127-132.

33.Robert B Tattersall. History of diabetes mellitus, Picket and Williams Text book of diabetes mellitus: Blackwell Scientific; 3rd ed (1):1-20;2003.

34.Vasudevan .D.M, Sreekumari.S. Textbook of biochemistry. Jaypee publications 9th ed.INDIA:Jaypee publisher:81-114.

35. Marco Guazzi MD, Gianluco Pantane MD, Piegiuseppe Agastani, MD, PhD Effect of NIDDM on pulmonary function and Exercise tolerance in chronic congestive heart failure. Amer Jour Card Vol.89.Jan2002

36. Hiroshi Mori, Masamichi Okuba, Midori Qkamura, Kiminori Yamane, Seijiro Kado, Genshi Egusa, Abnormalities of pulmonary function in patients with NIDDM Internal Medicine 31,2,189-193.

37. SK Rajan, P Rajesh Prabhu, Madhu Sasidharan, Spirometric evaluation of type1DM Abstract of paper presented at APICON 2003, JAPI Vol 50,1529 Dec,2002.

38. Schnaph BM et al. Pulmonary function in IDDM with limited joint mobility. Chest 90; 670. 1986.

39. Asanuma Y, Fujiya S, Ide H, Agishi Y.Characteristics of pulmonary functions in diabetes mellitus. *Diab Res Clin Pract* 1985; 1: 95–101.

40. Sreeja CK, Samual E, Keshavachandran, Shashidhar S. Pulmonary functions in patients with diabetes mellitus. *Ind J Physiol Pharmacol* 2003;47(1): 87–93.

41. Sandler M, Bunn AE, Stewart RI. Cross-section study of pulmonary function in patients with insulin dependent diabetes mellitus. *Am Rev Respir Dis* 1987; *135* : 223-9.

42. Maccioni FJ, Colebatch HJ. Lung volume and distensibility in insulin dependent diabetes mellitus. *Am Rev Respir Dis* 1991;*143* : 1253-6.

43. Marvisi M,Bartolini L,del Borrello P,Brianti M, MaraniG, Guariglia A, *et al*. Pulmonary function in non-insulin-dependent diabetes mellitus. *Respiration* 2001; *68* : 268-72.

44. Isotani H, Nakamura Y, Kameoka K, Tanaka K, Furukakawa K, Kitaoka H, *et al*. Pulmonary diffusion capacity, serum angiotensin Converting enzyme activity and angiotensin-converting enzyme gene in Japanese non insulin-dependent diabetes mellitus patients. *Diabetes Res Clin Pract* 1999; *43* : 173-7.

45. American Thoracic Society Statement, Snowbird workshop on standardization of spirometry. *Am Rev Respir Dis* 1979; *119* : 831-8.

46. Pedersen OF, Pedersen TF, Miller MR. Gas compression in lungs decreases peak expiratory flow depending on resistance of peak flow meter. J Appl Physiol 1997; 83:1517–1521.

47. Kano S, Burton DL, Lanteri CJ, Sly PD. Determination of peak expiratory flow. Eur Respir J 1993; 6: 1347–1352.

48. Gardner RM, Hankinson JL, West BJ. Evaluating commercially available spirometers. Am Rev Respir Dis 1986; 134:626–627.

49. Hankinson JL, Reynolds JS, Das MK, Viola JO. Method to produce American Thoracic Society flow–time waveforms using a mechanical pump. Eur Respir J 1997; 10: 690–694.

50.Chronic Obstructive Pulmonary Disease: National clinical guideline for management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax* 2004; **59 (Suppl 1):** 1–232

51. Guidelines for the measurement of respiratory function. Recommendations of the British Thoracic Society and the Association of Respiratory Technicians and Physiologists. Respir Med 1994; 88: 165–194.

52. Wanke T, Formanek D, Auinger M, Popp W, Zwick H, Irsigler K. Inspiratory muscle performance and pulmonary function changes in insulin-dependent diabetes mellitus. *Am Rev Respir Dis* 1991; *143* : 97-100.

PROFORMA

PULMONARY FUNCTION TESTS IN TYPE-2 DIABETES MELLITUS

NAME:	AGE:	SEX:
Study ID NO:	Spirometry ID NO:	OPD / IP NO:
Height (cms):	Weight (kgs):	BMI :
Education:	Occupation:	

Address:-

QUESTIONNARIE

History of Diabetes Mellitus: Yes /No

Symptoms:

-Polyuria Yes / No

-Polydpsia Yes / No

-Polyphagia Yes / No

-Unexplained weight loss Yes / No

Duration of Diabetes Mellitus:

Was Medications taken regulary: Yes / No

Drug	Dose	Duration taken

Complications Of Diabetes mellitus: Present /Absent
(if present) –

History of smoking/Hypertension/ tabacoo chewing habitt: Yes / No

History of alcohol consumption: Yes / No

History of chronic respiratory disease: Yes / No

History of unstable angina pectoris / M.I / C.H.F: Yes / No

Hospitalised for last 6 mts for respiratory illness: Yes / No

Clinical Examination:

General physical examination:

Temp: ^{0}F	Pulse: /min	Respiratory rate:/min	BP: mm of Hg

R.S-

C.V.S-

P / A-

C.N.S-

INVESTIGATIONS:

FBS : mg / dl
PPBS: mg / dl
HB1Ac: %

Pulmonary function test	Observed	Predicted	% Prediction
FVC			
FEV1			
PEF			
FEF 25 % - 75%			
FEV1/FVC%			

Master chart showing basic characters of control group

Sl no	Study ID No. of controls	age	sex	ht in cms	wt in kgs	BMI	FBS mg/dl	PPBS mg/dl
51	2011051	51	Male	175	69	22.53	101	136
52	2011052	55	Male	159	85	33.62	96	132
53	2011053	52	Male	148	70	31.96	85	128
54	2011054	49	Female	154	60	25.30	76	128
55	2011055	54	Male	150	82	36.44	86	121
56	2011056	40	Male	166	56	20.32	84	135
57	2011057	41	Female	157	56	22.72	94	128
58	2011058	47	Male	175	77	25.14	80	135
59	2011059	44	Female	170	74	25.61	82	120
60	2011060	44	Male	176	67	21.63	98	126
61	2011061	39	Female	167	50	17.93	104	132
62	2011062	36	Male	154	88	37.11	78	116
63	2011063	41	Female	164	71	26.40	86	102
64	2011064	44	Female	164	66	24.54	76	121
65	2011065	56	Female	167	55	19.72	85	125
66	2011066	44	Male	171	57	19.49	94	134
67	2011067	41	Male	154	55	23.19	94	138
68	2011068	51	Female	152	68	29.43	102	118
69	2011069	50	Male	152	60	25.97	78	132
70	2011070	50	Female	160	80	31.25	95	129
71	2011071	55	Male	158	65	26.04	74	136
72	2011072	51	Male	151	73	32.02	84	135
73	2011073	50	Female	159	70	27.69	100	140
74	2011074	52	Male	154	71	29.94	85	125
75	2011075	51	Male	152	76	32.89	94	134

Master chart showing basic characters of control group

Sl no	Study ID No. of controls	age	sex	ht in cms	wt in kgs	BMI	FBS mg/dl	PPBS mg/dl
76	2011076	47	Female	152	77	33.33	74	132
77	2011077	54	Male	160	68	26.56	82	128
78	2011078	49	Female	158	55	22.03	91	121
79	2011079	53	Male	151	57	25.00	84	106
80	2011080	38	Male	168	55	19.49	111	144
81	2011081	44	Female	168	68	24.09	82	128
82	2011082	39	Male	165	60	22.04	91	121
83	2011083	40	Female	161	80	30.86	84	106
84	2011084	47	Male	167	50	17.93	98	112
85	2011085	42	Male	167	82	29.40	112	136
86	2011086	49	Female	150	56	24.89	86	114
87	2011087	44	Male	166	56	20.32	94	138
88	2011088	46	Male	170	57	19.72	102	118
89	2011089	49	Female	167	56	20.08	76	118
90	2011090	51	Male	170	86	29.76	80	136
91	2011091	50	Female	154	70	29.52	72	132
92	2011092	51	Female	164	69	25.65	91	138
93	2011093	47	Male	164	85	31.60	82	128
94	2011094	49	Male	172	70	23.66	101	136
95	2011095	52	Female	172	60	20.28	96	132
96	2011096	57	Male	164	82	30.49	84	134
97	2011097	41	Female	169	56	19.61	76	114
98	2011098	47	Male	168	56	19.84	72	98
99	2011099	49	Female	171	70	23.94	78	116
100	2011100	50	Female	170	73	25.26	89	129

Master chart showing spirometric values of control group

Sl no	Study ID No. of controls	Rec FVC	Pred FVC	% Pred FVC	Rec FEV1	Pred FEV1	% Pred FEV1	Rec PEF	Pred PEF	% Pred PEF	Rec FEF25-75	Pred FEF25-75	% Pred FEF25-75	Rec FEV1/FVC	Pred FEV1/FVC	% Pred FEV1/FVC
51	2011051	3.27	4.85	67.40	2.19	3.76	58.30	576	604	95.36	2.58	3.32	77.70	67	77.5	86.40
52	2011052	3.68	3.74	98.40	2.73	2.88	94.80	484	555	87.21	2.92	2.57	113.70	74.2	76.7	96.70
53	2011053	3.19	3.2	99.80	2.12	2.5	84.80	448	540	82.96	2.6	2.37	109.70	66.5	77.3	85.90
54	2011054	3.61	3.16	114.40	2.06	2.52	81.80	332	406	81.77	2.73	2.61	104.60	57.1	80.4	71.00
55	2011055	3.22	3.25	99.00	1.52	2.52	60.30	475	537	88.45	2.9	2.3	124.40	47.2	76.9	61.40
56	2011056	3.17	4.54	69.80	2.39	3.64	65.60	606	613	98.86	2.96	3.55	83.30	75.4	79.8	94.50
57	2011057	3.3	3.42	96.50	2.76	2.79	98.80	376	425	88.47	3.62	2.97	121.80	83.6	82.1	101.90
58	2011058	3.56	4.95	71.90	2.63	3.88	67.80	543	618	87.86	2.45	3.52	69.60	73.9	78.4	94.30
59	2011059	3.32	4.01	82.80	2.73	3.22	84.80	454	436	104.13	2.83	3.16	89.60	82.2	81.5	100.90
60	2011060	3.53	5.09	69.40	2.47	4.01	61.50	578	628	92.04	2.75	3.71	74.20	70	79	88.60
61	2011061	3.25	3.92	82.80	2.68	3.2	87.30	467	441	105.90	2.73	3.27	83.60	82.5	82.5	99.90
62	2011062	3.4	3.9	87.10	2.78	3.21	86.70	511	587	87.05	2.95	3.36	87.90	81.8	80.6	101.40
63	2011063	2.68	3.75	71.40	2.58	3.05	84.60	413	429	96.27	2.96	3.1	94.60	96.3	82.1	117.00
64	2011064	3.52	3.71	94.80	2.78	2.99	93.00	404	429	94.17	2.85	3.02	94.40	79	81.5	97.00
65	2011065	2.47	3.63	68.10	1.78	2.83	62.90	299	404	74.01	2	2.62	76.30	72.1	78.9	91.30
66	2011066	2.94	4.77	61.70	2.42	3.77	64.20	632	617	102.43	2.38	3.53	67.50	82.3	79	104.80
67	2011067	2.55	3.8	67.10	2.11	3.07	68.60	601	583	103.09	2.13	3.11	68.60	82.7	79.6	104.00
68	2011068	3.11	3.03	102.70	1.9	2.4	79.10	267	399	66.92	3.35	2.49	134.60	61.1	80	76.40
69	2011069	3.76	3.47	108.30	2.78	2.73	101.90	421	556	75.72	2.23	2.59	86.00	73.9	77.7	95.10
70	2011070	2.91	3.42	85.20	1.85	2.71	68.20	287	411	69.83	3.59	2.7	132.80	63.6	80.2	79.30
71	2011071	3.19	3.68	86.60	2.5	2.84	88.20	433	552	78.44	3.6	2.54	142.00	78.4	76.7	102.20
72	2011072	3.63	3.39	107.10	2.21	2.66	83.20	467	550	84.91	2.9	2.51	115.40	60.9	77.7	78.50
73	2011073	3.17	3.37	94.10	2.21	2.67	82.60	334	410	81.46	1.55	2.68	57.80	69.7	80.2	86.90
74	2011074	3.32	3.53	94.00	1.52	2.75	55.20	475	554	85.74	1.32	2.56	51.60	45.8	77.3	59.20
75	2011075	3.17	3.45	92.00	1.85	2.7	68.50	429	553	77.58	2.04	2.54	80.20	58.4	77.5	75.00

Master chart showing spirometric values of control group

Sl no	Study ID No. of controls	Rec FVC	Pred FVC	% Pred FVC	Rec FEV1	Pred FEV1	% Pred FEV1	Rec PEF	Pred PEF	% Pred PEF	Rec FEF25-75	Pred FEF25-75	% Pred FEF25-75	Rec FEV1/FVC	Pred FEV1/FVC	% Pred FEV1/FVC
76	2011076	3.22	3.1	103.80	2.78	2.49	111.60	349	408	85.54	2.84	2.64	107.40	86.3	80.8	106.80
77	2011077	3.22	3.83	84.10	1.98	2.96	66.90	398	561	70.94	1.94	2.65	73.20	61.5	76.9	80.00
78	2011078	3.25	3.34	97.30	2.39	2.66	89.80	387	411	94.16	2.52	2.7	93.40	73.5	80.4	91.50
79	2011079	3.43	3.34	102.80	2.21	2.59	85.20	411	543	75.69	2.65	2.41	109.90	64.4	77.1	83.60
80	2011080	3.22	4.7	68.50	2.89	3.79	76.30	643	620	103.71	2.52	3.72	67.70	89.8	80.2	111.90
81	2011081	3.56	3.91	91.10	2.78	3.14	88.40	369	434	85.02	2.63	3.11	84.50	78.1	81.5	95.90
82	2011082	3.17	4.5	70.50	2.76	3.62	76.20	651	612	106.37	3.52	3.57	98.60	87.1	80	108.80
83	2011083	3.3	3.62	91.10	2.63	2.96	88.90	421	432	97.45	2.63	3.1	85.00	79.7	82.3	96.80
84	2011084	3.17	4.44	71.40	2.4	3.49	69.60	586	600	97.67	2.58	3.24	79.70	76.7	78.4	97.80
85	2011085	3.32	4.56	72.80	2.34	3.63	64.80	609	612	99.51	2.67	3.49	76.60	70.5	79.4	88.80
86	2011086	3.17	2.98	106.50	2.39	2.38	100.50	356	401	88.78	1.85	2.52	73.30	75.4	80.4	93.80
87	2011087	3.22	4.45	72.30	2.42	3.53	68.50	543	606	89.60	2.58	3.35	76.90	75.2	79	95.20
88	2011088	3.12	4.65	67.00	2.68	3.66	73.10	576	610	94.43	2.92	3.39	86.10	85.9	78.6	109.30
89	2011089	3.37	3.77	89.30	2.81	3	93.80	377	422	89.34	3.6	2.9	124.10	83.4	80.4	103.70
90	2011090	3.45	4.53	76.20	2.7	3.52	76.80	432	594	72.73	2.75	3.14	87.50	78	77.4	100.90
91	2011091	2.79	3.14	88.90	1.9	2.49	76.20	311	404	76.98	2.73	2.57	106.20	68.1	80.2	84.90
92	2011092	3.22	3.59	89.70	2.18	2.84	76.90	301	413	72.88	2.95	2.75	107.10	67.7	80	84.70
93	2011093	3.25	4.26	76.40	1.85	3.35	55.20	485	594	81.65	3.52	3.14	112.30	56.9	78.4	72.60
94	2011094	3.4	4.71	72.20	2.5	3.67	68.10	499	605	82.48	2.63	3.31	79.40	73.5	77.9	94.30
95	2011095	3.01	3.97	75.90	1.82	3.12	58.30	355	420	84.52	2.58	2.9	88.90	60.5	79.8	75.80
96	2011096	2.91	3.98	73.10	2.06	3.04	67.70	391	557	70.20	2.34	2.64	88.80	70.8	76.3	92.80
97	2011097	3.19	4	79.80	1.52	3.24	46.90	457	441	103.63	3.35	3.24	103.30	47.6	82.1	58.00
98	2011098	3.63	4.5	80.60	2.39	3.54	67.50	584	603	96.85	3.23	3.27	98.70	65.8	78.4	84.00
99	2011099	3.31	3.98	83.30	2.41	3.15	76.50	345	426	80.99	2.63	3	87.80	72.8	80.4	90.60
100	2011100	3.09	3.91	79.10	2.06	3.09	66.90	299	423	70.69	1.58	2.93	53.90	66.7	80.2	83.10

Master Chart showing basic characters of case group

Sl no	Study ID No. of cases	age	sex	ht in cms	wt in kgs	BMI	Duration of Type-2DM	Rx	BP	complications	fundus	FBS mg/dl	PPBS mg/dl	Hb1Ac %
1	2011001	41	Male	158	52	20.83	8	OHA	130/90	-	-	188	278	7.1
2	2011002	44	Female	151	56	24.56	6	OHA	142/96	-	-	145	290	8
3	2011003	42	Male	153	44	18.80	8	OHA	120/70	-	-	226	276	9.1
4	2011004	44	Female	162	67	25.53	6	OHA	144/88	-	-	178	321	6.4
5	2011005	44	Male	165	71	26.08	5	OHA	136/84	-	-	258	390	8
6	2011006	57	Female	158	53	21.23	11	Ins	160/94	N,DN	PDR	287	398	8.9
7	2011007	48	Female	155	59	24.56	9	OHA	130/80	DN	NPDR	199	298	7.3
8	2011008	49	Female	157	50	20.28	9	OHA	120/80	-	-	286	378	8.1
9	2011009	47	Male	159	68	26.90	8	OHA	148/88	-	NPDR	282	333	7.4
10	2011010	55	Female	157	65	26.37	9	Ins	154/86	DN	PDR	226	298	6.9
11	2011011	53	Male	166	78	28.31	11	OHA	148/78	-	-	245	398	9.1
12	2011012	58	Female	163	53	19.95	12	Ins	158/86	-	-	212	323	9.5
13	2011013	47	Male	171	80	27.36	7	OHA	140/90	-	-	146	276	5.9
14	2011014	41	Female	169	69	24.16	9	Ins	130/80	DN	PDR	123	187	6.5
15	2011015	42	Male	169	73	25.56	8	OHA	134/88	DN	NPDR	167	212	7.1
16	2011016	47	Male	172	79	26.70	7	OHA	138/80	-	-	164	202	6.9
17	2011017	42	Female	169	69	24.16	6	OHA	116/74	-	-	187	289	7.4
18	2011018	44	Male	171	74	25.31	7	OHA	160/90	-	-	217	264	8
19	2011019	39	Male	165	69	25.34	5	OHA	128/82	-	-	114	172	6.6
20	2011020	41	Female	169	64	22.41	6	OHA	126/80	-	-	162	254	7
21	2011021	46	Male	168	76	26.93	9	OHA	130/90	-	-	234	267	6.9
22	2011022	52	Female	162	68	25.91	9	OHA	138/88	-	-	165	243	9.2
23	2011023	49	Male	172	82	27.72	8	OHA	140/90	-	NPDR	239	308	7.2
24	2011024	44	Male	177	73	23.30	6	OHA	144/84	-	-	112	188	6.8
25	2011025	45	Female	169	61	21.36	7	OHA	130/80	-	-	141	198	7

Master Chart showing basic characters of case group

Sl no	Study ID No. of cases	age	sex	ht in cms	wt in kgs	BMI	Duration of Type-2DM	Rx	BP	complications	fundus	FBS mg/dl	PPBS mg/dl	Hb1Ac %
26	2011026	49	Male	178	81	25.56	5	OHA	150/90	-	-	115	226	7.9
27	2011027	48	Male	166	66	23.95	6	OHA	140/94	-	-	223	367	8.3
28	2011028	49	Female	154	57	24.03	8	OHA	130/90	-	NPDR	187	310	8
29	2011029	45	Male	158	61	24.44	6	OHA	132/90	-	-	167	192	6.1
30	2011030	57	Female	162	59	22.48	12	Ins	150/94	N,DN	PDR	275	358	9.4
31	2011031	49	Female	169	70	24.51	10	Ins	166/100	DN	PDR	216	198	7.2
32	2011032	51	Male	173	81	27.06	7	OHA	146/90	-	-	198	343	8.2
33	2011033	44	Female	166	65	23.59	9	OHA	154/90	-	-	246	270	6.6
34	2011034	54	Male	169	67	23.46	7	OHA	126/78	-	-	112	195	6.9
35	2011035	51	Male	168	78	27.64	10	Ins	170/90	N,DN	PDR	128	198	9.2
36	2011036	47	Female	169	68	23.81	9	OHA	116/70	-	-	155	184	7.2
37	2011037	49	Male	163	66	24.84	7	OHA	142/90	-	-	181	229	6
38	2011038	55	Female	161	54	20.83	9	OHA	158/94	-	-	182	233	7.1
39	2011039	53	Male	165	73	26.81	8	OHA	154/90	-	-	169	201	7.3
40	2011040	51	Female	168	69	24.45	6	OHA	130/90	-	-	129	160	6.1
41	2011041	48	Male	166	70	25.40	11	OHA	126/78	-	-	143	245	9.1
42	2011042	41	Male	168	59	20.90	5	OHA	120/80	-	-	158	212	6.9
43	2011043	48	Female	161	60	23.15	8	OHA	130/90	-	NPDR	190	301	7.8
44	2011044	51	Male	169	66	23.11	9	Ins	150/88	N,DN	PDR	221	299	7.9
45	2011045	41	Male	165	61	22.41	6	OHA	128/86	-	-	186	221	7
46	2011046	41	Female	159	60	23.73	7	OHA	110/76	-	-	221	298	7.5
47	2011047	44	Male	169	70	24.51	6	OHA	124/80	-	-	121	244	6.2
48	2011048	41	Female	160	67	26.17	9	Ins	130/80	DN	NPDR	276	312	8
49	2011049	49	Female	163	64	24.09	8	OHA	140/90	DN	NPDR	298	267	7.3
50	2011050	51	Male	171	70	23.94	10	Ins	144/88	N,DN	NPDR	156	254	9.3

Master Chart showing spirometric values of case group

Sl no	Study ID No. of cases	Rec FVC	Pred FVC	% Pred FVC	Rec FEV1	Pred FEV1	% Pred FEV1	Rec PEF	Pred PEF	% Pred PEF	Rec FEF25-75	Pred FEF25-75	% Pred FEF25-75	Rec FEV1/FVC	Pred FEV1/FVC	% Pred FEV1/FVC
1	2011001	2.92	4.03	97.20	1.88	3.25	57.90	377	593	63.58	1.74	3.24	53.80	64.4	79.6	80.90
2	2011002	2.57	3.11	82.80	1.66	2.52	65.90	312	412	75.73	2.13	2.73	77.90	64.6	81.5	79.00
3	2011003	2.67	3.72	85.10	1.56	3	51.90	433	579	74.78	1.54	3.02	54.20	58.4	79.4	73.60
4	2011004	3.63	3.62	100.40	2.55	2.92	87.50	354	426	83.10	3.83	2.97	128.80	70.2	81.5	86.20
5	2011005	2.7	4.39	61.50	1.83	3.49	52.50	456	604	75.50	3.13	3.32	94.30	67.8	79	85.80
6	2011006	1.24	3.17	57.00	1.85	2.47	75.00	256	443	57.79	1.7	2.7	113.70	54	78.7	64.00
7	2011007	2.24	3.22	69.50	1.78	2.57	69.10	335	409	81.91	2.04	2.37	76.40	67.5	80.6	78.60
8	2011008	2.3	3.44	66.80	1.51	2.83	53.30	284	428	66.36	2.04	3.04	67.10	65.7	82.5	77.60
9	2011009	2.68	3.96	67.80	1.95	3.13	62.40	579	582	99.48	3.45	2.97	116.20	62.8	78.4	72.90
10	2011010	2.32	3.17	84.80	1.72	2.48	69.30	205	395	51.90	2.62	2.44	107.60	51.8	79.1	65.50
11	2011011	1.45	4.22	48.00	1.45	3.26	50.50	442	578	76.47	2.07	2.9	78.20	59.2	77.1	76.70
12	2011012	1.12	3.38	52.20	1.58	2.63	60.20	230	394	58.38	2.04	2.44	83.40	58.2	78.5	72.50
13	2011013	2.48	4.69	52.80	1.43	3.68	38.80	558	609	91.63	2.27	3.38	67.20	57.7	78.4	73.60
14	2011014	2.78	4	69.50	1.85	3.24	57.10	256	440	58.18	2.86	3.24	88.20	66.5	82.1	81.10
15	2011015	3.09	4.68	66.00	2.01	3.7	53.90	461	617	74.72	3.11	3.56	87.40	65	79.4	81.90
16	2011016	3.14	4.76	66.00	2.08	3.73	55.70	577	607	95.06	3.68	3.41	107.80	60.2	78.4	74.50
17	2011017	2.08	3.99	52.20	2.18	3.22	66.60	424	439	96.58	2.65	3.21	82.60	56.7	81.9	69.30
18	2011018	2.58	4.77	54.10	1.46	3.77	38.70	324	617	52.51	2.7	3.53	76.50	56.6	79	71.70
19	2011019	2.55	4.5	76.40	1.52	3.62	52.00	609	612	99.51	2.7	3.57	75.70	59.7	80	74.60
20	2011020	2.44	4	61.00	1.8	3.24	55.50	435	440	98.86	3.04	3.24	62.90	73.8	82.1	89.90
21	2011021	2.03	4.53	44.80	1.25	3.57	35.00	499	605	82.48	2.45	3.32	73.70	61.6	78.6	78.40
22	2011022	2.74	3.64	75.20	1.72	2.96	58.20	451	430	104.88	2.99	3.05	98.10	62.8	81.9	76.70
23	2011023	2.44	4.71	51.80	1.65	3.67	44.90	357	605	59.01	1.87	3.31	56.40	67.6	77.9	86.80
24	2011024	3.01	5.15	70.40	1.78	4.06	53.80	458	630	72.70	2.56	3.74	41.70	59.1	79	78.90
25	2011025	2.34	3.94	59.30	1.48	3.16	46.80	487	433	112.47	2.11	3.1	68.10	63.2	81.2	77.80

Master Chart showing spirometric values of case group

Sl no	Study ID No. of cases	Rec FVC	Pred FVC	% Pred FVC	Rec FEV1	Pred FEV1	% Pred FEV1	Rec PEF	Pred PEF	% Pred PEF	Rec FEF25-75	Pred FEF25-75	% Pred FEF25-75	Rec FEV1/FVC	Pred FEV1/FVC	% Pred FEV1/FVC
26	2011026	2.48	5.1	68.30	1.49	3.97	57.50	524	617	84.93	2.68	3.53	47.60	60.1	77.9	77.10
27	2011027	2.77	4.35	63.60	1.7	3.42	49.80	542	595	91.09	2.42	3.15	76.70	61.4	78.1	78.50
28	2011028	3.27	3.16	103.60	1.8	2.52	71.50	387	406	95.32	2.67	2.61	102.30	55	80.4	68.50
29	2011029	4.02	3.94	101.90	2.48	3.14	79.00	558	585	95.38	2.11	3.04	69.50	61.7	78.8	78.30
30	2011030	1.33	3.36	58.30	1.71	2.61	65.40	223	396	56.31	2.19	2.46	88.90	60.5	78.7	72.60
31	2011031	1.73	3.87	67.00	1.88	3.07	61.20	261	424	61.56	1.63	2.95	55.30	55.8	80.4	69.40
32	2011032	2.24	4.72	47.50	1.18	3.66	32.20	329	600	54.83	2.15	3.25	66.20	52.7	77.5	69.70
33	2011033	2.35	3.81	61.70	1.36	3.07	44.30	398	431	92.34	2.47	3.07	80.60	57.9	81.5	71.00
34	2011034	2.41	4.38	65.70	1.35	3.37	60.00	278	580	47.93	2.42	2.96	81.80	56	76.9	77.80
35	2011035	2.33	4.4	52.90	1.43	3.42	41.80	334	589	56.71	2.31	3.07	75.20	61.4	77.5	79.20
36	2011036	2.04	3.91	54.20	1.17	3	39.00	286	429	66.67	1.65	2.95	55.90	57.4	80.8	71.00
37	2011037	2.38	4.15	57.40	1.38	3.25	42.50	470	585	80.34	2.03	3	67.60	58	77.9	74.40
38	2011038	2.53	3.36	75.40	1.69	2.63	64.30	215	400	53.75	1.94	2.52	76.80	63.8	79.1	74.40
39	2011039	2.1	4.16	50.50	1.9	3.22	64.20	440	576	76.39	2.13	2.87	74.20	52.4	77.1	67.90
40	2011040	2.33	3.79	61.60	1.34	2.99	44.80	254	418	60.77	1.78	2.85	62.50	57.5	80	71.90
41	2011041	1.25	4.35	44.80	1.12	3.42	32.80	421	595	70.76	1.2	3.15	38.10	57.4	78.1	73.50
42	2011042	2.88	4.64	62.00	1.84	3.71	49.60	603	616	97.89	2.28	3.57	63.80	63.9	79.6	80.30
43	2011043	3.3	3.5	94.20	2.44	2.79	87.40	312	417	74.82	3.03	2.8	108.10	65.9	80.6	71.70
44	2011044	2.3	4.46	51.50	1.45	3.57	41.80	377	585	64.44	1.94	3.11	62.40	63	77.5	71.30
45	2011045	4.1	4.46	92.00	2.7	3.57	75.70	672	609	110.34	3	3.47	57.70	65.9	79.6	82.70
46	2011046	3.13	3.51	89.10	2.62	2.87	91.40	359	428	83.88	2.74	3.02	90.90	73.1	82.1	82.00
47	2011047	2.38	4.64	51.30	2.42	3.67	58.70	588	613	95.92	2.13	3.46	61.60	59.7	79	75.50
48	2011048	2.63	3.45	76.10	1.55	2.76	56.20	352	429	82.05	2.54	2.78	91.40	58.9	80.6	73.10
49	2011049	2.5	3.58	69.80	1.77	2.84	62.20	368	417	88.25	2.83	2.81	100.80	65.8	80.4	78.80
50	2011050	1.62	4.59	61.90	1.7	3.56	47.70	316	596	53.02	2.78	3.18	87.50	51.5	77.5	66.40

LIST OF ABBREVIATIONS

DM	Diabetes Mellitus
ADA	American Diabetes Association
FVC	Forced vital capacity
FEV1	Forced expiratory volume in 1 seconds
FEV1/FVC%	FEV1 as percentage of FVC
FEF25-75%	Forced mid-expiratory flow
FBS	Fasting blood glucose level
HbA1c	Glycated hemoglobin
PEF	Peak expiratory flow
PPBS	Post prandial blood glucose level
PFT	Pulmonary function tests
IDDM.	Insulin dependent diabetes mellitus
NIDDM	Non-Insulin dependent diabetes mellitus
BMI	Body mass index
SD	Standard deviation
BTPS	Body temperature pressure saturated