PULMONARY FUNCTION TESTS IN TYPE 2 DIABETIC PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERIES UNDER GENERAL ANESTHESIA

A STUDY OF 100 CASES

DISSERTATION SUBMITTED FOR

DOCTOR OF MEDICINE

BRANCH X (ANAESTHESIOLOGY)

APRIL 2015

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

CHENNAI

TAMIL NADU

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"PULMONARY FUNCTION TESTS IN TYPE 2 DIABETIC PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERIES UNDER GENERAL ANESTHESIA"** submitted by **DR.M.KARTHIKEYAN** to the **FACULTY OF ANAESTHESIOLOGY, THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY, CHENNAI**, in partial fulfilment of the requirement in the award of the degree of M.D., degree Branch X (ANAESTHESIOLOGY) for the April 2015 examination is a bonafide research work carried out by him under my direct supervision and guidance.

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This is to certify that this dissertation entitled **"PULMONARY FUNCTION TESTS IN TYPE 2 DIABETIC PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERIES UNDER GENERAL ANESTHESIA"** is a bonafide and genuine research work done by **Dr. KARTHIKEYAN. M,** under our direct supervision and guidance, submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the degree of MD in Anaesthesiology.

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DECLARATION

I , **DR.M.KARTHIKEYAN** declare that the dissertation titled **"PULMONARY FUNCTION TESTS IN TYPE 2 DIABETIC PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERIES UNDER GENERAL ANESTHESIA"** has been prepared by me. This is submitted to the Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfilment of the requirement for the award of M.D. Degree Branch X (Anaesthesiology) Degree Examination to be held in April 2015. I also declare that this dissertation, in part or full was not submitted by me or any other to any other university or board, either in India or abroad for any award, degree or diploma.

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PULMONARY FUNCTION TESTS IN TYPE 2 DIABETIC PATIENTS UNDERGOING MAJOR ABDOMINAL SURGERIES UNDER GENERAL ANESTHESIA

ABSTRACT

Background: Diabetes mellitus is a systemic disease. Among the various organs affected by diabetes, lung is also included. Pulmonary function tests can be assessed by spirometry preoperatively which helps in better outcome of the diabetic patients postoperatively.

Aims and objectives: The aim of the study is to evaluate the pulmonary function tests in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia.

Materials and methods: Fifty type 2 diabetic patients and fifty non-diabetic patients in the age group of 40-60 yrs undergoing elective major abdominal surgeries under general anesthesia were selected for the study. General anesthesia was standardised for both the groups. Pulmonary function tests were performed 60 minutes before and 60 minutes after the end of surgery. In the diabetic group, patients with duration of diabetes of 5-15 yrs were selected for the study. The pulmonary function tests recorded were FEV1, FVC, FEV1/FVC, FEF 25%, Peak expiratory flow rate(PEFR).

Results: The pulmonary function tests were significantly reduced in diabetic patients both preoperatively and postoperatively when compared to non-diabetic patients.

Conclusion: Diabetes mellitus being a systemic disease,affects lungs causing ventilatory abnormalities probably because of glycosylation of connective tissues, reduced pulmonary elastic recoil and inflammatory changes in the lungs.

KEY WORDS: Diabetes mellitus, Pulmonary function tests, General anesthesia.

INTRODUCTION

Diabetes mellitus is a systemic disease. It affects many organ systems in the human body. Among the various systems affected, respiratory system is also included. It is both a microvascular and also a macrovascular disease. In diabetes, the alveolar capillary network in the lung may be affected by microangiopathic changes. As this network has a large reserve , these microangiopthic changes may go unrecognised clinically. Symptoms such as dyspnoea develops only in the late stages of the disease.

Pulmonary functions can be assessed by spirometry. The pulmonary function tests includes both static and dynamic tests. The pulmonary function tests can be used for diabetic patients undergoing major surgeries such as cardiothoracic and abdominal surgeries. The status of the respiratory system can be assessed preoperatively by doing this test. It helps to optimise the patients preoperatively. The preoperative optimisation includes regular breathing exercises and control of blood glucose level. This results in better postoperative outcome of the diabetic patients . In this study , we evaluate the pulmonary function tests both preoperatively and postoperatively in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia and compare them with the non diabetic patients undergoing elective major abdominal surgeries under general anesthesia.

AIM OF THE STUDY

The aim of the study is to evaluate the pulmonary function tests in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia.

ANATOMY OF RESPIRATORY SYSTEM

The respiratory tract is divided into upper and lower respiratory tract.

UPPER RESPIRATORY TRACT:

This consists of following components.

1. Nasal Passages.
2. Sinuses.
3. Pharynx.
4. Epiglottis.
5. Larynx.

The important functions of the upper airway are:

- Conducting the air to the lower airway.
- Protecting the lower airway from the foreign materials such as food or liquids soiling it.
- Warming, filtering and humidifying the inspired air for efficient gaseous exchange.

NASAL PASSAGES:

The two nasal cavities start in front from the external nares and end posteriorly in the nasopharynx. The structure of the nose with its two nasal cavities, turbinates and rich blood supply provide maximum contact between the inspired air nasal mucosa for the humidification of air. In the anterior part of the nasal fossa with its stiff hairs and spongy mucous membrane and the ciliated

epithelium provides a powerful defence against the invasion of any organism. The most important factor in the prevention of accumulation of secretions throughout the respiratory tract is the continuous activity of cilia. The absence of moisture even for a few minutes leads to ciliary activity cessation.

SINUSES:

The sinuses and paranasal sinuse play an important role in modifying the quality of the air which is breathed during normal respiration. Sinuses decreases the weight of the skull, providing mucous for the nasal cavity and also act as resonant chambers of voice. The paranasal sinuses are lined with ciliated mucous producing cells and have small pathways known as meatus which communicates with the nasal cavity.

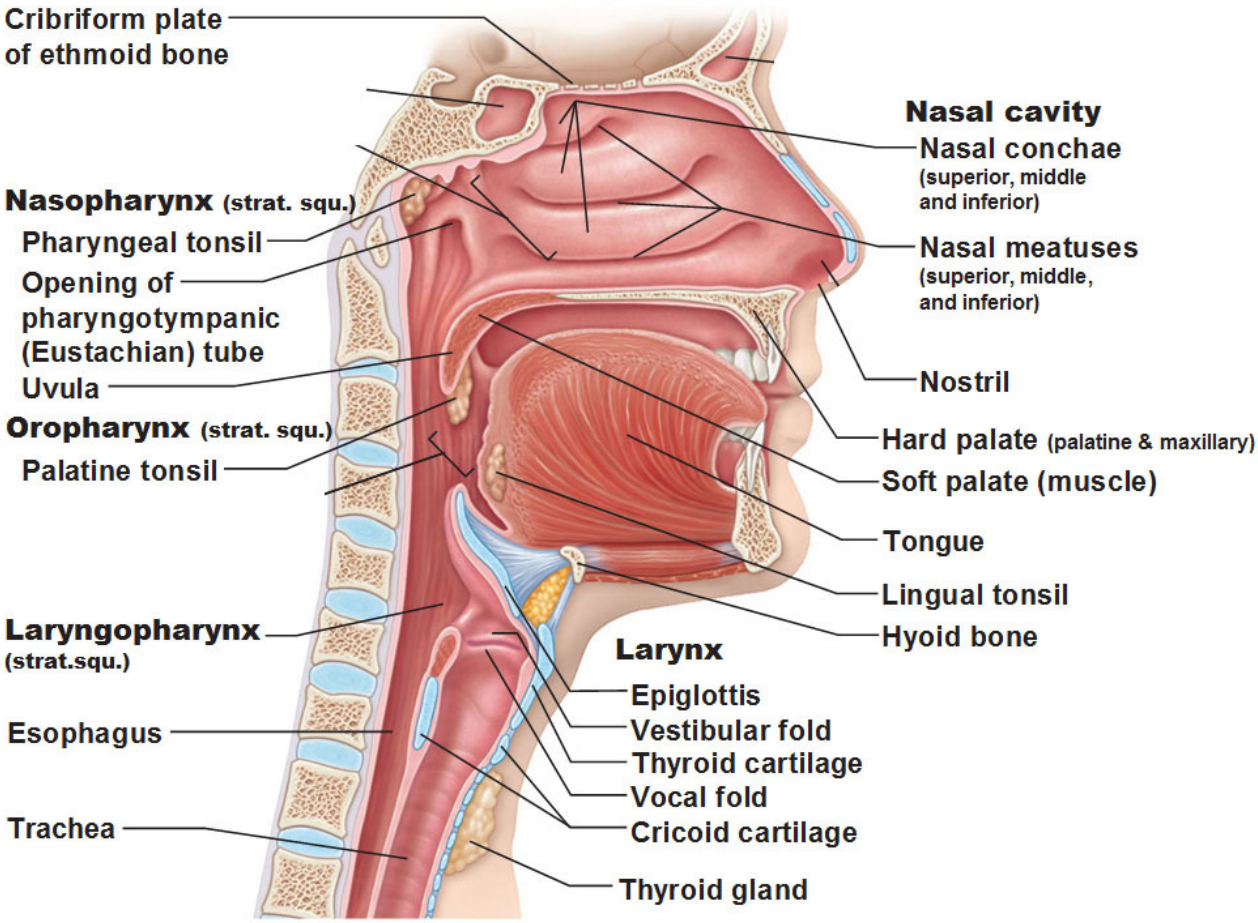
PHARYNX:

The pharynx is a common passage for food and air to pass through to take part their respective route. When a patient becomes unconscious, the area which gets obstructed is the pharynx. There are three divisions in the pharynx namely nasopharynx, oropharynx and hypopharynx. The main difficulty in maintaining a perfect airway in an unconscious patient is the tendency of the tongue to fall backwards obstructing the laryngeal opening.

For obtaining a clear airway two separate maneuvers are needed to provide perfect airway in the unconscious patient. First one is, the lower jaw must be carried forwards and upwards so that the lower incisor lie in front of the upper incisor. This is known as Prognathic attitude. The second one is, the head

is hyperextended so that the tongue is carried farther upward and forward which is away from the posterior pharyngeal wall.

The Upper Respiratory Tract



This Diagram shows various structures of the upper respiratory tract

LARYNX:

Larynx is the organ of phonation. The larynx lies at the level of 3rd to 6th cervical vertebra and consists of a number of articulated cartilages surrounding the upper end of trachea. The vocal cords in the larynx provides phonation. The larynx also provides sphincter function which prevents aspiration. The cartilages of larynx are thyroid, arytenoids and cricoid. The thyroid cartilage is the largest and protects the important structures of the larynx from any damage. The cricothyroid membrane is incised to perform Cricothyroidotomy which is an emergency procedure done for the obstruction of upper airway. The arytenoids cartilage provides attachment for vocal cord ligaments. The glottis is the space between the two vocal cords. The epiglottis is a leaf like cartilaginous structure which extends from the base of the tongue to the thyroid cartilage by ligaments. The epiglottis flaps down during swallowing to direct the swallowed material into the esophagus thus guarding the opening of the larynx.

LOWER RESPIRATORY TRACT:

The lower respiratory tract consists of the trachea which is considered as the downward continuation of the larynx. The trachea divides into right and left main bronchus which leads on to the right and left lungs. The main bronchus gives branches for the individual lobes of the lungs. The lobar bronchi branches into lobular bronchi which finally divides into smaller tube like structures resembling the branches of a tree becoming smaller and shorter. The

functions of the lower respiratory tract are to conduct air down to the alveoli to provide mucociliary defence.

TRACHEA:

The trachea is a tube made up of rings of cartilages which are incomplete posteriorly. The posterior part of the trachea is made up of smooth muscle and lies just adjacent to the esophagus. Any excessive pressure on this smooth muscle , for example, cuff of an endotracheal tube or tracheostomy tube leads to erosion causing trachea-esophageal fistula. The trachea is about 10-11 cm long extending from the lower part of the larynx opposite to the level of the 6^{th} cervical vertebra to the point where it bifurcates into right and left main bronchus at the carina at the level of upper border of 5^{th} thoracic vertebra. In children the carina is at the level of 3^{rd} costal cartilage. The trachea is lined by ciliated columnar epithelium and mucous secreting goblet cells. The upper two thirds of the trachea is supplied by inferior thyroid artery and the lower one third is supplied by the bronchial arteries. The trachea can move with respiration and changes in position with the movement of the head.

On the basis of their function lower airway is divided into

1. Conducting airways.
2. Respiratory zone.

The conducting airways is the non alveolate region and the respiratory zone is the alveolate region. The main bronchus is taken as division 1 and the subsequent divisions are numbered as 2,3 and so on. The divisions are also

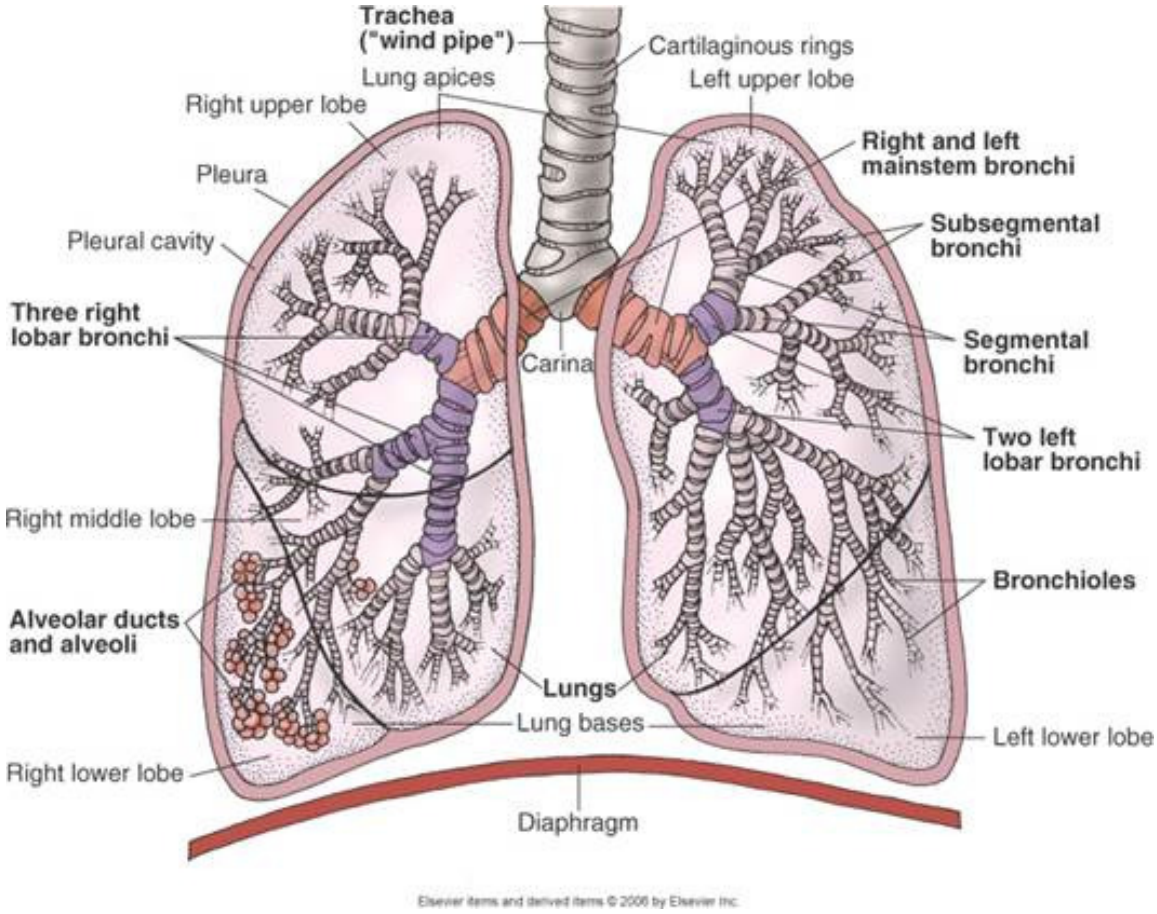
known as generation. Approximately the first 16 divisions of the tracheobronchial tree does not take part in gas exchange and so they are named as the conduction zone. The volume of air in this zone is approximately 150 ml and this is known as the anatomical dead space.

The right main bronchus starts from the trachea at an angle of 25 degrees from the vertical. This enters the right lung opposite to T5. The right upper lobe bronchus starts just 2.5 cm from the carina. This position promotes more chances for the aspiration of foreign material into the right lung. In adults, the narrowest portion is the glottis and in children the narrowest portion is the cricoid ring. After entering the lung the right main bronchus divides into 3 lobar bronchi which again divides into upper, middle and lower right lung lobes. The right upper lobe consists of three segments- apical, posterior and anterior segments. The right middle lobe consists of two segments- lateral and medial segments. The right lower lobe consists of five segments- apical, medial basal, anterior basal, lateral basal and posterior basal segments.

The left main bronchus is longer and narrower than the right main bronchus and lies more horizontal to the trachea. The length of the left main bronchus before the origin of upper lobe bronchus is 5cm. The left main bronchus leaves the trachea at an angle of about 45 degrees and enters the lung opposite to T6. The left main bronchus divides into upper and lower lobe bronchi. The left upper lobe consists of five segments – apical, posterior, anterior, superior and inferior segments. The left lower lobe consists of

3 segments- apical, anterior basal lateral basal and posterior basal segments. The lobar bronchi bifurcates into segmental bronchioles or terminal bronchioles which leads on to the lung segments.

LOWER RESPIRATORY TRACT



This Diagram shows various structures of the lower respiratory tract

RESPIRATORY ZONE:

The respiratory zone starts from the 17th zone of the bronchial tree. It consists of

- Terminal bronchiole.
- Respiratory bronchiole.
- Alveolar ducts or passages.
- Atria.
- Air sacs.
- Air cells.

The alveoli are lined with a single molecular layer of phospholipids known as phosphotidyl choline which is the major content of surfactant. The surfactant is a substance that reduces the surface tension of the alveoli.

PHYSIOLOGY OF RESPIRATORY SYSTEM:

Respiration is nothing but the gaseous exchange between an organism and its environment. Here, oxygen is absorbed and carbon dioxide is removed that involves two components. The diffusion of gases at the alveolar capillary membrane is known as external respiration and the diffusion of gases across the cell membrane is known as internal respiration. The carbon dioxide which is removed from the tissues is carried by the blood to the lungs where it is eliminated by external respiration. For adequate gaseous exchange to take place the lung has to be well perfused with deoxygenated blood from the pulmonary artery.There are three main integral parts of external respiration. They are:

- Ventilation which means moving of air in and out of lungs.
- Perfusion which is adequate blood flow to all parts of the lung.
- Diffusion which is exchange of gases across the alveolar capillary membrane.

The factors modifying the diffusion of gases across the alveolar capillary membrane are the surface area of the membrane, the thickness of the membrane, the pressure gradient between the two sides of the membrane, the diffusion coefficient of the gas. The lung has enormous surface area for gas exchange. At rest, for enough gas exchange to take place,5 healthy segments out of 19 segments is sufficient.

Increase in the thickness of the alveolar capillary membrane causes difficulty in diffusion. This can occur in chronic fibrotic lung diseases,

pulmonary edema, diabetes mellitus etc. At rest, blood passes through the capillaries in approximately 0.5 to 0.75 seconds. But it is estimated that when the blood has travelled only one fourth of the capillary distance, the gas exchange is completed. This is the reason which provides reserve time for gas exchange in conditions like disease and exercise states. In conditions like alveolar congestion, interstitial or alveolar edema, pulmonary fibrosis the diffusion distance and time may be increased.

The partial pressure of oxygen in the alveolus is 100 mm Hg and the partial pressure of oxygen in the pulmonary capillary is 40 mm Hg. Therefore, a pressure gradient of 60 mm Hg is present for oxygen to diffuse into the pulmonary blood to reach the equilibrium. Similarly, a pressure gradient of 6 mm Hg causes easy diffusion of carbon dioxide as it is 20 times more diffusible than oxygen into the alveoli.

ALVEOLAR PCO_2:

The alveolar pco_2 is expressed as $PACO_2$. At rest, the normal oxygen uptake is 250ml/minute and the normal CO_2 production is 200ml/minute. CO_2 diffuses from the pulmonary artery to the alveoli where the $PACO_2$ is determined by its rate transfer and its dilution by alveolar ventilation. $PaCO_2$ rises at a rate of 3 to 6 mm Hg during apnoea.

ALVEOLAR PO_2:

Alveolar po_2 is expressed as PAO_2.The main factor which influences the alveolar PAO_2 is the barometric pressure. The normal barometric pressure is 760 mm Hg. The normal saturated pressure of water vapour in the alveoli is 47 mm Hg. As one goes to the high altitude the barometric pressure decreases. At 63000 feet, the barometric pressure is 47mm Hg so that PIO2 becomes zero and therefore blood starts boiling. When the cardiac output decreases, the perfusion to the lung decreases which decreases the uptake of oxygen and therefore PAO_2 will tend to increase.

OXYGEN CARRIAGE IN BLOOD:

The saturation of oxygen in haemoglobin for different partial pressures of oxygen to which the oxygen is exposed is not linear. The haemoglobin is 97% saturated with oxygen at 90 to 100 mm Hg. At partial pressures of 60 mm Hg, the saturation is 90%. The partial pressure of oxygen with the percentage saturation of haemoglobin can be compared with oxygen dissociation curve. As the shape of the curve is S shaped , it is possible for the PO_2 to be reduced with only minimal effect on the saturation. But when a certain point is reached the saturation begins to fall rapidly with only a small reduction in PO_2. The shift of the curve to the left or right will have a significant effect on the availability of oxygen to the tissues. At a lower level of saturation, haemoglobin readily combines with oxygen to the tissues which is of great advantage for the delivery of oxygen to the tissues. P50 is the partial pressure of oxygen required

for 50% saturation of haemoglobin. Normally it is 26 mm Hg. Conditions where oxygen haemoglobin dissociation curve is shifted to right are acidosis, hypercarbia, hyperthermia, anemia, chronic hypoxia, stored blood. The conditions which shift the oxygen haemoglobin dissociation curve to the left are alkalosis, hypothermia, reduction in 2,3-Diphosphoglycerate levels. When fully saturated, 1 gram of haemoglobin carries 1.34 ml of oxygen. To this 0.3 ml of oxygen which is carried in the form of physical solution has to be added. When the PO2 decreases below 27 mm Hg in a normal person the consciousness is likely to be lost.

OXYGEN FLUX:

The rate of oxygen carriage in the arterial blood is known as oxygen flux. It is also defined as the amount of oxygen leaving the left ventricle per minute in the arterial blood. This can be calculated as

Cardiac output(ml/min) X SaO_2 X Hb concentration (gm/ml) X 1.34.

The normal value of oxygen flux is 1000ml/min. When this value is below 400ml/min and if it continues for long time it is dangerous. In this 1000 ml, 250 ml is used up for cellular metabolism and the remaining returns to the lungs in the mixed venous blood and therefore this blood is about 75% saturated with oxygen.

CARBON DIOXIDE TRANSPORT:

The proteins in the plasma combines with carbon dioxide to form carbamino compounds. The remaining carbondioxide is either transported in simple solution or as bicarbonate ion. Normally, 10 ml of blood carries 3 ml of carbondioxide. Carbondioxide is 20 times more diffusible than oxygen. The absolute requirement for the elimination of carbondioxide is the alveolar ventilation. Inhalation of 5% carbondioxide for a long time even though it is unpleasant, usually will not produce any ill effects but when the concentration is raised to 15%, unconsciousness occurs. At this level, muscle rigidity and tremors can occur. When the concentration of CO2 raises to 20-30% generalised convulsions can be produced.

EFFECT OF GENERAL ANESTHESIA ON RESPIRATORY SYSTEM

When a patient undergoes surgery under general anesthesia , whether the patient is on controlled ventilation or spontaneous ventilation , most of the patients suffer some degree of impairment in the arterial oxygenation. This impairment becomes more significant in elderly patients, obese patients and in smokers. In healthy young and middle aged patients undergoing surgeries under general anesthesia, venous admixture or shunt has been found to be only 10% and the scatter in ventilation to perfusion ratio (V/Q) has been found to be minimal. But in patients who has significant deterioration in the pulmonary functions preoperatively,general anesthesia causes widening in the VA/Q distribution and also the shunt.

When the depth of anesthesia is increased in patients on general anesthesia the following changes are noted in respiration. At lighter planes of anesthesia, the respiratory patterns may vary from hyperventilation to vocalisation to breath holding. As the depth of anesthesia increases and equals to that of minimum alveolar concentration, the normal respiration follows but with a larger tidal volume. With further progression in the depth of anesthesia, the regular respiration is interrupted but inspiratory pause is partial at end expiration- hitch in the inspiration followed by prolonged expiratory phase indicated by arrows. With further deepening of anesthesia, the respiration becomes faster, more regular but shallow breathing. Later becomes sine wave.

Both opioids and benzodiazepines produces respiratory depression but opioids depresses the respiratory rate and benzodiazepines decreases the tidal volume.

When halogenated agents are used, the patient is in deep anesthesia and the increased depression is manifested by rapid shallow breathing . This is known as panting. As the anesthetic depth deepens , the support provided by the intercostal muscles is lost leading to the rocking boat movement. Here, there is an out of phase depression of chestwall during inspiration and there is bellowing of the abdomen. If the patient is on spontaneous ventilation, as the anesthetic depth deepens with halothane , the increasing concentration of halothane displaces the end tidal CO2 concentration., PCO2-ventilation response curve progresses to the right shifting the apnoeic threshold to a higher end tidal CO2 concentration.

The pre-existing diseases such as pulmonary infections, lung collapse, sepsis, cardiac failure, renal failure and heavy smokers determines the effect of anesthesia on the respiratory functions. Here, the relationship between functional residual capacity(FRC) and closing capacity(CC) is important. In healthy patients FRC exceeds that of CC by 1 litre. But in obese patients , elderly and in patients with chest wall deformities , CC is 0.5-0.75 litres less than FRC. After induction of anesthesia with a 1 litre decrease in FRC, there will be no change in the relationship between FRC and CC in a healthy patient. But in patients with respiratory disturbances, a decrease in FRC will cause the

CC to increase more than FRC and causes a low ventilation perfusion ratio or an atelectatic FRC-CC relationship.

If an anesthetic drug inhibits Hypoxic pulmonary vasoconstriction (HPV) , the drug causes increased shunting in patients with pre-existing HPV than in patients without pre-existing HPV. Thus the effect of standard anesthetic produces varying degrees of respiratory changes in patients who have different degrees of pulmonary dysfunction. The position of the patients such as lithotomy, jack knife and kidney rest position may decrease the cardiac output and causes hypoventilation in spontaneously breathing patients leading on to decrease in FRC. FRC refers to the volume of the air remaining in the lungs after a normal, passive expiration. The loss of chest wall compliance or lung compliance results in reduced FRC. Anesthesia and abdominal surgeries produces a progressive upward displacement of the diaphragm . This upward displacement of diaphragm is due to loss of respiratory muscle tone which allows the abdominal content to push the diaphragm upwards. This results in reduced FRC and development of intraoperative atelectasis, intrapulmonary shunting, and hypoxemia. The head down position of the patient, type of surgery such as abdominal surgery or laparoscopy with insufflation , concurrent use of muscle relaxants can further cause the diaphragmatic shift upwards. The anatomical dead space increases because of the increase in peak airway pressure and expansion of the bronchial tree. Anesthesia cause more dangerous effects on respiratory function in obese subjects than in normal patients. For

determining the lung volume, oxygenation and respiratory mechanics body mass is important. The volume of decrease in FRC with atelectasis has been found to be related to age, weight, and size .The airway resistance is approximately twice as high in patients with severe obesity compared with those with minimal obesity. Moderate to severe hypoxemia has been found in supine obese subjects during spontaneous breathing, anesthesia and when the patient is paralysed. . Ventilation-perfusion mismatch is seen in awake, sitting and obese subjects.

If FRC reduces below closing capacity, airway closure can occur. In this case, the lung bases are well perfused, but the ventilation is poor because of airway closure and collapse of the alveoli. This causes increases ventilation-perfusion mismatch and favours formation of compression and absorption atelectasis, leading to hypoxemia.

High inspired oxygen concentrations causes significant atelectasis. There is a progressive reduction in lung compliance during anesthesia in patients with either spontaneous breathing or mechanically ventilated patients. The decrease in lung compliance is accompanied by decreasing alveolar oxygen tensions. The atmosphere is composed of 78% nitrogen and 21% oxygen. As exchange of oxygen takes place at the alveolar-capillary membrane, nitrogen is a major component contributing for the inflation of alveoli. When nitrogen in the lungs is replaced with oxygen in a larger volume, for example, when the patient is breathing 100% oxygen, the oxygen may get absorbed into the blood

thus decreasing the volume of the alveoli, which results in alveolar collapse. This is known as absorption atelectasis. Pulmonary atelectasis develops in the most dependent part of the lungs during general anesthesia in 90% of the patients with normal lung function, and this is considered as the major cause of impairment of gas exchange and reduction of lung compliance. The development of atelectasis during anesthesia is attributed to both absorption atelectasis and compression of lung tissue as a result of high inspired oxygen concentrations. In the postoperative period also, atelectasis plays an important role.

Inhalational anesthetics inhibits hypoxic pulmonary vasoconstriction (HPV). Hypoxic pulmonary vasoconstriction is a physiological phenomenon in which pulmonary arteries constrict in response to hypoxia without hypercapnia redirecting the blood flow to the alveoli with aaequate ventilation. Intravenous anesthetic agents does not inhibit hyoxic pulmonary vasoconstriction. The HPV response may be masked by concurrent changes in cardiac output, contraction of myocardium, vascular tone, blood volume distribution, blood pH and CO2 tension, and lung mechanics. The inhalational anesthetics isoflurane and halothane depress the HPV response by 50% at two times minimum alveolar concentration (MAC) without any significant decrease in cardiac output.

PULMONARY FUNCTION TESTS

HISTORICAL ASPECTS:

The concept of spirometry goes back to the period of 129-200 AD, when Claudius Galen performed volumetric experiments on human ventilation.

Giovanni Alfonso Borelli in the year 1681, measured the volume of inspired air in one breath by sucking a liquid in a cylindrical tube.

In the year 1718, Jurin J. Blew air into bladder and measured the volume of air present in the bladder by the principles of Archimedes. He measured the tidal volume of 650 ml and maximum expiration of 3610 ml.

In the year 1796, Menzies R. determined the tidal volume by using the method of body plethysmography.

In the year 1799, Pepys WHJ.found that the tidal volume to be 270 ml by using two mercury gasometers and one water gasometer.

In the early 1800's Sir Humphrey Davy was the person who measured his own vital capacity and tidal volume and found it to be 3110 ml and 210 ml with a gasometer. He also measured the residual volume by hydrogen dilution method which was found to be 590 ml.

In the year 1813, Kentish E. was the person who used a simple "pulmometer" to study ventilator volumes in disease.

It was in the year 1852, Hutchinson John, who designed the first spirometer, a water spirometer which is used till date along with few alterations.

The first measurement of Vital Capacity was made by him. He also showed the linear relationship of vital capacity to height.

Wintrich was the person who developed a modified spirometer, concluding that 3 parameters determine the vital capacity. These parmeters are height, weight and age of the individual.

Smith E. in the year 1859 developed a portable spirometer.

In the year 1866, Salter added the kymograph to the spirometer to record time and volume.

Gad J.in 1879 introduced a Pneumatograph which allowed to register the volume changes of thorax during inspiration and expiration in addition to the known parameters and he named it as Aeroplethysmograph.

Brodie T.G in 1902, used a dry bellow spirometer.

Tissot in the year 1904, introduced a close circuit spirometer.

In 1929, Knipping introduced a standardised method for spiroergometer.

Tiffnean in 1948, introduced Forced Expiratory Volume (FEV) as a useful lung function test.

Wright B.M and McKerrow C.B in 1959, introduced the peak flowmeter.

Computerised spirometer was introduced in the year 1990.

Pulmonary function tests are an important investigation in the management of patients with major respiratory diseases which are either suspected or diagnosed previously. They help us in the diagnosis, to monitor the response to treatment

and to plan for further intervention. For the interpretation of pulmonary function test, a thorough knowledge of respiratory physiology is needed.

INDICATIONS:

1. It can be done in patients whose symptoms are suggestive of respiratory disease. These symptoms include cough, cracles, wheeze, breathlessness, abnormal chest x ray.
2. It can be done in patients to monitor for the progression and response to treatment to a particular pulmonary disease. These diseases include Interstitial fibrosis, COPD, Asthma, pulmonary vascular diseases.
3. It can be done in patients having connective tissue disorders, neuromuscular disorders who may have some respiratory complications.
4. It can be done for the preoperative evaluation before Lung resection, cardiothoracic and abdominal surgeries.
5. It can be done to evaluate the patients who are at risk for lung diseases.
6. It can be done to assess for any infection, obliterative bronchiolitis and acute rejection in patients who had undergone lung transplantation.

CONTRAINDICATIONS FOR PERFORMING PULMONARY FUNCTION TESTS:

1. Pneumothorax.
2. Recent Myocardial Infarction within last one month.
3. Unstable Angina.
4. Recent thoracic surgeries.
5. Recent eye surgeries.
6. Aortic or Thoracic aneurysm.

LIMITATIONS OF PULMONARY FUNCTION TESTS:

1. As with any other tests, there is some variability in the normal predictive value.
2. Unlike any other tests, the accuracy of the pulmonary function tests depends on the well trained technician as well as cooperation of the patients. Patients should use their maximum efforts and at the same time the technician should be able to recognize submaximal efforts.
3. Pulmonary function tests must be interpreted along with proper history, clinical examination and other diagnostic tests .When this test is used alone they usually cannot distinguish among the potential causes of the abnormalities.

SPIROMETER

REQUIREMENTS FOR GOOD PULMONARY FUNCTION TESTS:

The American Thoracic Society (ATS) has published certain guidelines for the standardization of spirometry apparatus and performance that should include acceptability and reproducibility criteria. As spirometry is an effort dependent test, each and every spirogram should be examined by using the performance criteria published by the ATS.

1. There should be lack of artefact caused either by coughing, glottic closure or equipment problems.
2. The starting of the test should be satisfactory without hesitation.
3. The exhalation time should be satisfactory with 6 seconds of smooth continuous exhalation and a plateau in the time volume curve of atleast 1 second .

Criteria for reproducibility after getting 3 acceptable spirograms are:

1. Largest FVC within 200 ml of next largest FVC.
2. Largest FEV1within 200 ml of next largest FEV1.

If these above two criteria are not met, then additional spirograms have to be performed. To meet the acceptability and reproducibility criteria maximum eight efforts can be allowed. After eight efforts fatigue plays an important role in the results and interpretation which may not be reliable. If all the above criteria are not met, we should interpret the abnormal results with caution.

TECHNIQUE OF PERFORMING PULMONARY FUNCTION TESTS:

1. The patient should be advised not to smoke atleast one hour before doing the test.
2. The patient should be advised not to eat a heavy meal two hours before doing the test.
3. The patient should be advised not to wear tight clothes as this may lead on to false results.
4. False teeth if any, can be left in place unless these prevent the patient from forming an effective seal around the mouth piece.
5. The patient should be well seated with the nose clip in place.
6. It is necessary for the patient to practice the exercise before actually performing the test. The patient is asked to breathe in and out deeply several times.
7. The patient should keep their mouth completely over the mouth piece but not inside it.
8. The patient should be asked to blow out as fast and as quick as they can for atleast six seconds.
9. Once the patient has blown out as much as they can ,ask the patient to inhale deeply as much as they can.
10. The whole test can be repeated upto three times. The goal is to get a reproducible result that is consistent.

In order to get a internally valid test, the patient may be asked to repeat the test more than three times.

PHYSIOLOGICAL FACTORS AFFECTING PULMONARY FUNCTION TESTS:

Spirometry can be reported as both absolute values and as percentage predicted values of the normal. Standards for normality are based on the factors such as age, sex, height, weight and race with an abnormal value occurring if there is 20% difference from the predicted mean value.

AGE:

As age progresses, the natural elasticity of the lung is reduced resulting in gradual decrease of lung volumes and lung capacities.

GENDER:

In males, lung volumes and capacities are more when compared to females. Because of this gender difference, different normal tables should be used for males and females.

BODY HEIGHT AND WEIGHT:

Individuals with short stature will have smaller PFT result when compared to the tall individuals. Obese individuals in whom there is increased body fat to lean body mass ratio, the abdominal mass prevents downward movement of the diaphragm resulting in smaller PFT than the expected.

RACE:

PFTs of Caucasians are different from those of Blacks, Hispanics and Native Americans.

TESTS OF VENTILATORY FUNCTION:

BEDSIDE PULMONARY FUNCTION TESTS:

1. COUGH TEST:

   The patient is asked to take a deep inspiration and cough once. The test is positive if the first cough leads to recurrent coughing. This is suggestive of underlying bronchitis.

2. WHEEZE TEST:

   The patient is asked to take five deep breaths and then auscultated posteriorly between the shoulder blades to test for the presence or absence of wheezing.

3. MAXIMUM LARYNGEAL HEIGHT:

   The distance between the tip of the thyroid cartilage and the suprasternal notch at the end of expiration is measured. If this is <4cm, it is abnormal. It is an accurate sign of obstructive airway disease.

4. FORCED EXPIRATORY TIME:

   The bell of the stethoscope is placed over the trachea in the suprasternal notch and the stopwatch is set to zero. The patient is instructed to take in the deepest breath possible and then to blow it out as fast as possible as he could. When the patient begins to exhale, start the stopwatch

and stop it immediately as audible expiration is no longer heard. Value more than 6 seconds indicates severe expiratory airflow obstruction with %FEV1<50%. The test is done three times and the average is taken. This clinically measured test correlates well with the forced expiratory time measured by spirometry.

5. BREATH HOLDING TIME:

The patient is asked to take a deep breath and hold his breath as long as possible. The stethoscope is placed over the trachea to identify early expiration. A breath holding time of >30 seconds is normal. Values between 20 -30 seconds denotes compromised cardiopulmonary reserve. The value <20 seconds denotes very poor cardiopulmonary reserve.

6. SNIDER'S MATCH BLOWING TEST:

Patient's maximum breathing capacity (MBC) can be measured by this test. A lighted match stick is placed at varying distances from the patient's mouth. The patient is instructed to sit, to keep his mouth open and to blow the candle off without pursing the lips. The ability of the patient to blow the candle off at a distance of 22cm from the mouth indicates MBC >150L/min. Patients with moderate to severe COPD will find it difficult with this test. When there is need for oxygen therapy in COPD patients, this test is contraindicated.

7. SINGLE BREATH COUNTING TEST:

Ask the patient to count out loud numbers from 1 onwards after a maximum inspiration. Individuals who are able to count 50 or more have normal respiratory function. If the single breath count is <15, it indicates severe impairment of vital capacity.

STATIC TESTS:

The static tests includes lung volumes and capacities. The static lung volumes reflect the elastic properties of the lungs and the chest wall.

Lung volumes that cannot be measured by spirometry are Residual Volume, Total Lung Capacity and Functional Residual Capacity.

TIDAL VOLUME (TV):

It is the volume of air that is inhaled or exhaled with each breath when a person is breathing at rest.

INSPIRATORY RESERVE VOLUME (IRV):

It is the maximum volume of air that can be inhaled from the end of inspiration.

EXPIRATORY RESERVE VOLUME (ERV):

It is the maximum volume of air that can be exhaled from the end of expiration.

INSPIRATORY CAPACITY (IC):

It is the maximum volume of air that can be inhaled from tidal volume end expiratory level. Inspiratory capacity is Inspiratory Reserve Volume plus Tidal volume.

FUNCTIONAL RESIDUAL CAPACITY (FRC):

It is the volume of air in the lungs that follows the exhalation of a tidal volume. It is expressed as the sum of ERV+RV. It is the lung volume at which the inward elastic recoil of the lungs is balanced by the outward elastic force of the relaxed chest wall. Usually FRC is 40-50% of the Total Lung Capacity. FRC increases when the lung elasticity is reduced and decreases when the lung recoiling is increased. Posture also influences the Functional Residual Capacity. In standing position FRC is more when compared to the supine position. This is one of the reason why patients in the postoperative recovery should be kept in the head elevated position. Almost all the anesthetic drugs reduces the muscle tone thereby reducing the FRC close to the residual volume in awake state. This is the cause for sudden desaturation in case of obese patients, pregnant patients and patients with large intra-abdominal mass who are under anesthesia. Because of the loss of elastic lung tissue, FRC increases with age. The preoxygenation done prior to the induction of anesthesia is to replace the functional residual capacity with 100% oxygen that helps in delaying the desaturation of apneic patient during intubation.

CLOSING CAPACITY (CC):

Closing capacity is the lung volume at which the smaller airways in the dependent part of the lung begins to close. Closing capacity is expressed as the sum of closing volume plus residual volume.CC=CV+RV. Closing volume is the volume of gas that is expelled during phase 4 of the single breath nitrogen test. It denotes the lung volume from the starting of airway closure to the end of maximum expiration. Closing capacity in a normal young healthy adult is approximately 10% of vital capacity or 400-500 ml.

Closing volume and closing capacity increases as age increases. In patients with small airway diseases and in chronic smokers, the closing volume is increased. The normal value of closing capacity is intimately related to age and position of the patient in patients with normal lung. The lowest value of closing capacity is usually seen in late 50s . Closing capacity is progressively increased above and below this age. The factors influencing the closing capacity are obesity, early chronic bronchitis, heavy smoking, left ventricular failure, myocardial infarction and immediate postoperative period.

FRC is either independent of age in adults or may increase very minimally with increasing age. But closing capacity increases as the age increases. At the age of 66, closing capacity becomes equal to Functional residual capacity in upright position. In the supine position, at the age of 44, CC becomes equal to that of FRC. FRC is influenced mainly by postural changes. When the patient is changed from supine to upright position there is

30% change in FRC. On the other hand, CC is independent of body position. Therefore, to conclude FRC is dependent on posture and CC is dependent on age. FRC is decreased by approximately 20% with spontaneous ventilation and about 16% with artificial ventilation when the patient is anesthetised. After inducing the patient, a decrease in the cross sectional area of the rib cage occurs corresponding to decrease in the lung volume of about 200ml. In the dependent position, there will be cephalad movement of diaphragm. Previously it was thought that CC was unchanged during anesthesia but recent studies confirmed there is parallel decrease in CC along with FRC during anesthesia. The use of positive end expiratory pressure may be the probable reason why there is increase in normal PO2 by increasing FRC above CC.

Closing capacity can be measured by single breath nitrogen washout technique. This test was originally described by Flower in the year 1949. In this technique, the patient is instructed to slowly exhale to residual volume and then asked to slowly inhale a single breath of oxygen to maximum inhalation. Then the patient is asked to hold the breath for few seconds and then asked to exhale slowly and evenly. The volume of expired air and the instantaneous nitrogen concentration are recorded during this phase. A characteristic curve is obtained and this is called as single breath nitrogen curve. This curve consists of 4 phases.

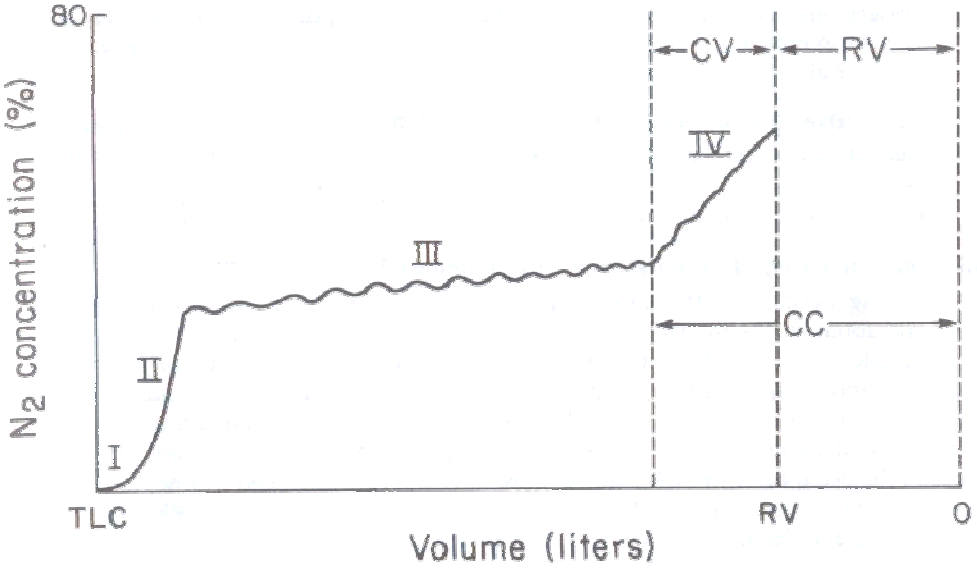
Phase 1 – dead space gas.

Phase 2 – Mixed dead space gas plus alveolar gas.

Phase 3 – Mixed alveolar gas from all the alveoli.

Phase 4 – is the phase where there is sudden increase in nitrogen concentration. Closing capacity is the volume at which phase 4 begins. During inspiration the oxygen is distributed to the smaller alveoli in the dependent parts of the lung because of the shape of the alveolar compliance curve. This results in larger change in the volume of smaller alveoli than the larger alveoli. This is the reason for more dilution of nitrogen in the smaller alveoli. During expiration, the mixed alveolar concentration from all the alveoli is measured till a point where closure of airway begins. This is the point where there is increased nitrogen concentration because the expulsion of gases from the smaller airway stops and exhalation continues from the areas of lungs where there is increased nitrogen concentration. Closing volume and closing capacity can also be measured by Bolus technique with an inert tracer gas such as helium, xenon or argon.

SINGLE BREATH NITROGEN CURVE



FORCED VITAL CAPACITY (FVC):

It is the total volume of air that can be forcibly exhaled from a maximum inspiratory effort.

VITAL CAPACITY (VC):

It is the maximum volume of air that can be exhaled from the beginning of maximum inspiration. Vital capacity is an important preoperative assessment tool because it reflects the ability of the patient to cough, to take a deep breath and to clear the excess secretion in the airways. Vital capacity is normally measured as 70 ml/kg ideal body weight. When the vital capacity is significantly reduced to <20ml/kg of the ideal body weight, it indicates that the patient is at increased risk for postoperative complications. Vital capacity helps

in evaluating the condition of the patient for weaning from a ventilator. If a patient who is on ventilator can demonstrate a vital capacity of 10-15 ml/kg of body weight, then it is generally considered that there is enough ventilatory reserve to try for weaning and extubation.

SLOW VITAL CAPACITY (SVC) TEST:

In this test, the patient is asked to blow out slowly and completely all the air from the lungs. The advantage of this test is that it eliminates the strong bronchoconstriction which usually accompanies a strong forceful exhalation effort. Therefore, after a SVC test the vital capacity of the patient may be much larger because there is little or no airway collapse during a controlled and slow exhalation effort. If after performing a SVC test, the vital capacity is increased, then it is assumed that the original small FVC is caused by the collapse of the airways and it rules out the presence of restrictive airway disease. The restrictive pathology has to be considered if the vital capacity does not improve either with the inhalation of a bronchodilator or with the administration of a SVC test.

TOTAL LUNG CAPACITY (TLC):

It is the total volume of air in the lungs at full inspiration. It is expressed as the sum of IC+FRC.

RESIDUAL VOLUME (RV):

Residual volume is the amount of air that is remaining in the lungs at the end of forceful expiration. Residual volume depends on two important factors- smaller airway collapse and limits of chest wall excursion. Residual volume is determined by the compression of the chest wall by the muscles in case of restrictive lung pathologies. In case of obstructive lung diseases, Residual volume is influenced by the collapse of the terminal airways which prevents the distal air from escaping the lungs.

METHODS TO MEASURE RESIDUAL LUNG VOLUME:

1. NITROGEN WASHOUT TECHNIQUE:

It is an open circuit gas dilution technique for measuring the Residual volume. In this technique, the patient breathes 100% oxygen at the end of a normal expiration and all the nitrogen in the lung is washed out. The exhaled volume and the concentration of nitrogen in that volume are measured. The difference in the volume of nitrogen at the initial concentration and at the final exhaled concentration allows to calculate the intrathoracic volume usually Functional Residual Capacity.

2. HELIUM DILUTION TECHNIQUE:

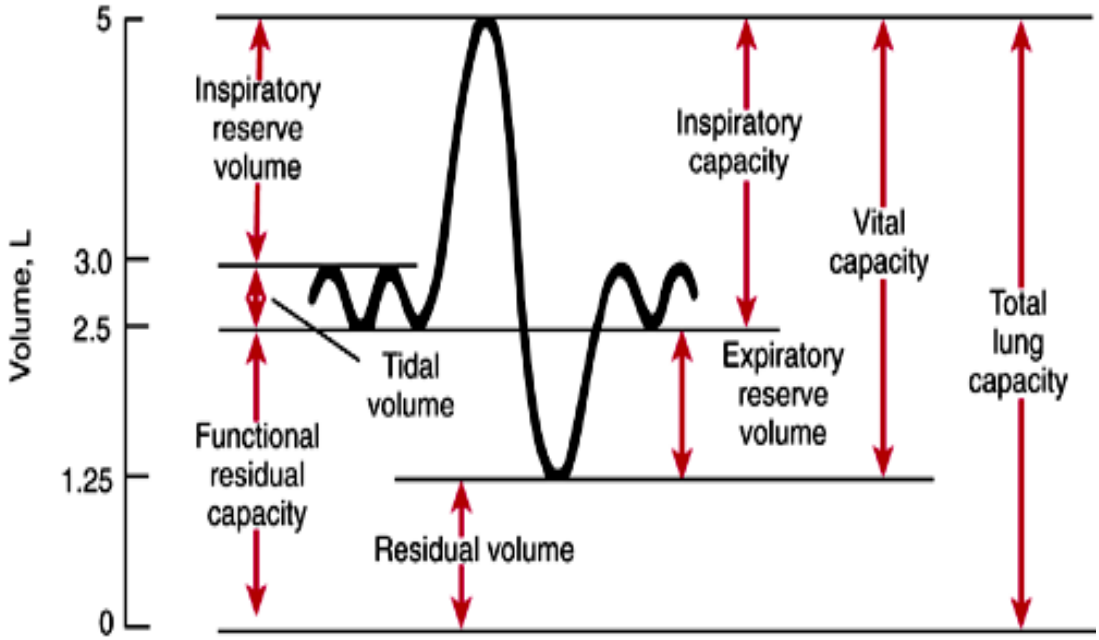
It is a closed circuit technique .It is based on the principle of inhalation of a known concentration and volume of an inert trace gas, such as helium, which is followed by equilibration of 7 to 10 minutes in the closed circuit. The concentration of helium which is finally exhaled is diluted in proportion to the

unknown volume of air in the patient's chest. Usually, the patient is connected to the spirometer at the end tidal position and therefore the lung volume which is measured is Functional Residual Capacity.

3. BODY PLETHYSMOGRAPHY:

It is based on the principle of Boyle's Law which states that the volume of gas at a constant temperature varies inversely with the pressure applied to it. In this technique, the patient is asked to sit in a closed "body box" with a known volume. The lung volumes are calculated based on the amount of air which is displaced from the box during ventilation.

PAPPENHEIMER'S CURVE



DYNAMIC TESTS:

The parameters that can be recorded by spirometry are:

1. FORCED EXPIRATORY VOLUME IN 1 SECOND(FEV1):

It is the volume of air forcibly expired from a maximum inspiratory effort in one second. The normal value is 80-120% of the predicted.

2. FORCED VITAL CAPACITY(FVC):

It is the total amount of air a person can exhale, usually measured in 6 seconds. The normal value is 80-120% of the predicted. 70-80% demonstrates mild reduction. 50-70% demonstrates moderate reduction. Less than 50% demonstrates severe reduction.

3. TIMED FEV1/FVC:

This provides an even better indication of degree of obstruction of the airway. This decreases the variability between the individuals and helps in the comparison between the individuals. This ratio is conventionally measured at 0.5, 1 and 3 seconds. This test is used for the confirmation of chronic obstructive airway disease.

4. FEV0.5/FVC:

It is used to assess the airflow at larger lung volumes and from the larger airways. Normally, it is >50%.

5. FEV1/FVC RATIO:

It is the percentage of FVC that can be expired in one second. 75-80% is normal. 60-80% demonstrates mild reduction. 50-60% demonstrates moderate reduction. Less than 50% demonstrates severe obstruction. FEV1/FVC is the most sensitive indicator of airflow obstruction than FEV1 alone. This ratio is less than 75% in case of obstructive lung diseases and it falls linearly with the progression of obstruction. FEV1/FVC reduces the variability and allows for more consistent results in assessing the dynamic airflow.

6. FEV3/FVC:

It is used to assess the function of small airways. Normally, it is >95%.

7. FORCED EXPIRATORY FLOW 200-1200:

This measures over one litre of initial part of the spirogram. This measurement starts at 200 ml to permit for the attainment of peak flow. This is

also known as Maximum Expiratory Flow Rate. Actually, this flow is lower than the true peak flow. Due to effort dependence, it has high variability. It is not useful for the assessment of small and medium sized airways or the response to treatment. The value <200litres/min in patients undergoing surgery suggests impaired cough efficiency and there is high chances for postoperative complications. This test provides a valuable tool in identifying gross pulmonary disability at the bedside because it is more pleasant and less exhaustive than FVC.

8. FEF25%-75%:

It is also known as Maximum mid-expiratory flow rate (MMEFR). It measures airflows over mid-half of vital capacity. It is a sensitive indicator of early small airway dysfunction. The normal value is 4-5 litres/sec. More than 80% is normal. 60-80% reflects mild obstruction in the small airways. 40-60% reflects moderate obstruction. Less than 40% reflects severe obstruction. This is effort independent, but it can be decreased by marked reduction in the expiratory effort and also by a subnormal inspiration before performing the maneuver.

It is a highly variable spirometric index as it depends on the absolute volume of FVC and on the changes of airway obstruction. In the detection of mild abnormalities of lung dysfunction, the lung volumes such as TLC, FVC, FEV1 are all reduced and here FEF25%-75% does not appear to be more sensitive than FEV1.

DIFFERENCE BETWEEN FEV1 AND FEF25%-75%:

It is the volume of air forcibly expired from a maximum inspiratory effort in the first second. FEF25%-75% is the average flow in the middle half portion of forceful expiration, starting when 25% of FVC is exhaled and ending when 75% FVC is completed. Even though both the values are decreased in obstructive airway diseases, FEF25%-75% reflects the calibre of airways <2mm in diameter as it is measured at lower lung volumes. The flow becomes effort independent when the lung volumes are less than approximately 70% of FVC. This effort independence is attributed to equal pressure point concept. During forceful expiration, airway pressure gradually decreases as air moves from the distal most part of the airways to the proximal airway as the diameter of the airway increases and the corresponding resistance decreases. At a particular point in the distal airways, the intraluminal pressure falls below the external pressure compressing the airways which includes the pleural pressure and the lung recoil pressure, thereby the airway collapses trapping the air. When the expiratory effort that is the pleural pressure is greater, the equal pressure point is more proximal. However the airway occlusion interrupts the airflow, the intraluminal pressure increases due to trapped air and reopens the airway. Thus, at equal pressure point, airflow is directly proportional to the elastic recoil of the lungs and inversely proportional to the resistance of the airways between alveoli and equal pressure point and not on the pleural pressure.

FORCED EXPIRATORY FLOW 75%-80%:

It is also known as Maximum end expiratory flow rate(MEEFR). It helps to assess the function of airways at lower lung volumes and is a sensitive indicator of small airway obstruction.

FORCED INSPIRATORY FLOW (FIF):

FIF is usually measured using forced spirogram and also from the flow volume loop. As it is effort dependent, it is not a useful measurement. FIF is decreased in patients with neuromuscular diseases like myasthenia gravis.

PEAK EXPIRATORY FLOW RATE (PEFR):

It is the maximum flow rate that is achieved by an individual during forced vital capacity maneuver, starting after full inspiration and ending with maximal expiration. PEFR depends on the airway caliber, patient's effort and vital capacity of the patient. PERF is measured in lit/sec or lit/min. This is measured by either pneumotachography or by a specially designed instrument such as Wright's peak flowmeter. The normal value of PEFR in males is 450-700 ml/min and in females it is 300-500ml/min.

CLINICAL APPLICATIONS OF PEFR:

1. Measurment of PEFR 3-4 times/day and a variability of >15% is highly suggestive of bronchial asthma. Thus, it helps in the diagnosis and assessment of pulmonary status of bronchial asthma at home itself.

2. It helps to assess the reversibility. PEFR is measured before and after giving bronchodilator therapy. An increase in the value of 10-15% suggests significant reversibility of the condition.
3. PEFR is used to measure exercise induced bronchospasm. PEFR is measured at rest and at 2 minutes interval during 6 minute exercise and a decrease in >15% is considered to be significant.
4. It is used to assess the severity of pulmonary disease and effectiveness of therapy.
5. PEFR is used to assess the circadian variation of the bronchial tree.
6. It is also used to monitor the response to therapy and to follow up the course of the disease.

   Ideally, PEFR measurement is done twice daily. When it is done once daily, then it should be done consistently at the same time before and after bronchodilator therapy. There is high variation between the individuals and so it should be measured objectively based on the patient's personal best conditions. The personal best condition is the highest PEFR achieved in the middle of the day after bronchodilatation.

MAXIMUM BREATHING CAPACITY (MBC):

The largest volume that can be breathed per minute by voluntary efforts is the maximum breathing capacity. This is also known as Maximum Voluntary Ventilation (MVV). It estimates the peak ventilation available to meet the physiological demands. The patient is instructed to breathe as deep and as quick as possible for 12 seconds and the measured volume is multiplied by 5 to get the volume in one minute. MBC is expressed as litres/minute. The normal value is 150-175 litres/minute. When the value is <75% of predicted, it is significant.

It reflects the respiratory muscle status, compliance of thorax –lungs complex and airway resistance.

It is the quick and easiest way to assess the strength of patient's pulmonary musculature. Poor performance of this test prior to surgery suggests that the patient may have pulmonary problems postoperatively due to muscle weakness. Since it parallels the FEV1, it can be used to test the internal consistency and to estimate patient cooperation. When the patient does not perform this test properly, the test becomes effort dependent. In this situation, it cannot be used to assess the true pulmonary muscle strength and compliance. If the value is disproportionately low in a patient who seems to be cooperating, one has to suspect for neuromuscular weakness. Most patients can generate fairly good single breathe efforts except in advanced neuromuscular diseases. Because MVV is much more demanding, it reveals decreased reserves of weak respiratory muscles. In pulmonary disorders like obstructive, restrictive

disorders or in patients with heart diseases and in very frail patients, low MVV can occur.

FLOW VOLUME LOOPS:

It is one of the way for determining the dynamic lung function by performing forced expiratory maneuver and plotting volume against flow. The change in flow volume loops of various pulmonary disorders can be predicted by this plot. Both flow and volume are plotted simultaneously on X and Y recorder when the patient fully inspires to Total lung capacity and then perform FVC maneuver.

This is followed immediately by a maximum inspiration as quick as possible back to Total lung capacity. Normally at the beginning of forced expiration, the flow rate quickly raises to a peak value at a lung volume near Total lung capacity. As expiration continues, the lung volume decreases, airway becomes narrow, resistance increases and the flow rate becomes progressively decreased. The whole inspiratory portion of the loop and expiratory cycle near TLC is highly effort dependent but the expiratory flow at 75%-25%of vital capacity is effort independent. Normally, the expiratory to inspiratory flow ratio at 50% of vital capacity is about 1.0. This ratio becomes particularly important in the identification of presence of upper airway obstruction.

It also helps to locate the site and to identify the nature of obstruction. There is no significant change in the diameter of the airways during inspiration or expiration. This results in a plateau of constant flow in the expiratory flow

over the effort dependent portion of vital capacity. There is similar plateau seen during the inspiratory flow also. As both are reduced to nearly the same extent, the mid vital capacity ratio remains approximately 1.0.

A variable obstruction is defined as a lesion whose influence changes with the phase of respiration. Variable extrathoracic obstruction occurs in case of single vocal cord paralysis, tracheomalacia or tumors invading the trachea. When a single vocal cord is paralysed, there is passive movement of this cord in accordance with the pressure gradient across the glottis. During a forceful inspiration, the paralysed vocal cord is drawn inwards resulting in reduced inspiratory flow. During a forced expiration, the paralysed vocal cord is blown aside. This results in unimpaired expired flow i.e. MIF50% FVC is <MEF 50% FVC. During normal inspiration, the airways inside the thorax becomes dilated as the lung inflates but the airways outside the thorax becomes collapsed due to negative intraluminal pressure.

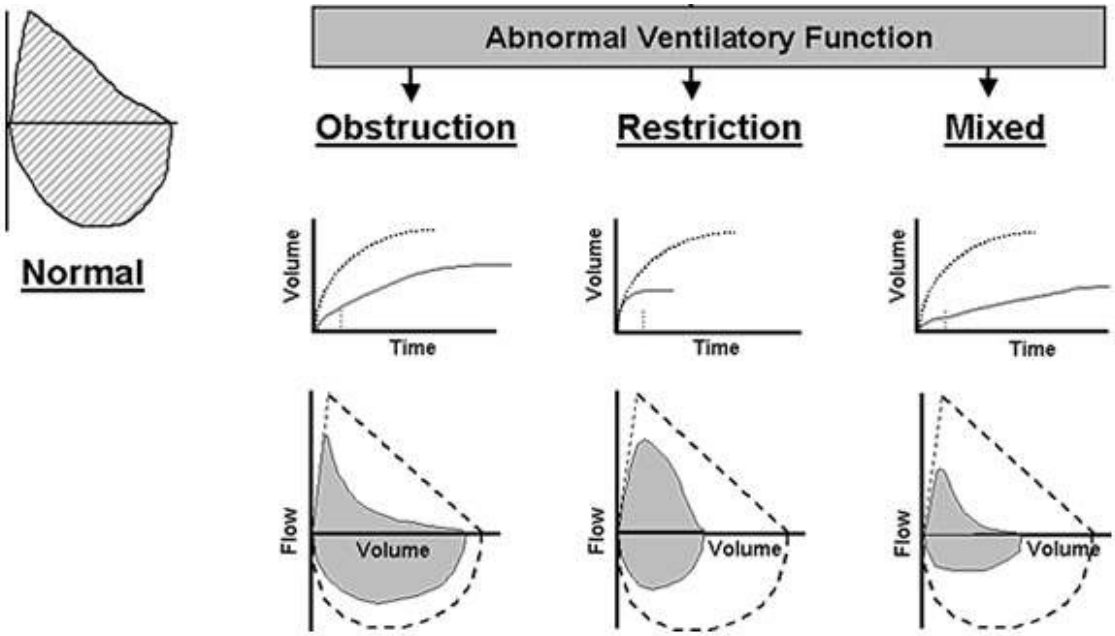
During expiration, the reverse happens as the airways inside the thorax becomes closed but the airways outside the thorax are held open by expiratory flow. As a result of this, variable extrathoracic obstruction leads to a flattened inspiratory limb with normal expiratory portion.

Variable intrathoracic obstruction occurs in cases of distal tracheomalacia and bronchomalacia. In this case, negative pleural pressure holds the floppy trachea open during a forceful inspiration. During forced expiration, there is loss of structural support resulting in narrowing of the trachea and a plateau of

decreased flow. There will be a brief period of maintained flow seen before the airway compression occurs. Therefore, a variable intrathoracic obstruction affects the expiratory limb mainly giving a flattened appearance of the expiratory portion of the loop.

Fixed upper airway obstruction is seen in bilateral vocal cord paralysis, goitre and tracheal stenosis. Here, the flow is limited by the narrowed segment rather than by the dynamic compression of the airways. This results in the equal reduction of inspiratory and expiratory flow rates. Therefore, both the top and bottom of the loop becomes flattened resulting in the rectangular configuration of the loop.

Hence, in case of fixed airflow obstruction the airflow limitation becomes equal both during inspiration and expiration.(MEF=MIF). The advantages of flow volume loop is flow at a particular point can be easily read from the curve. The adequacy of patients effort can be easily known from the overall shape of the curve .when the expiratory effort is smaller, with a long concave shape of the curve, it indicates small airway obstruction. In case of large airway obstruction the peak flow rates are typically affected resulting in the flat flow volume loop. The flattening occurs either in the expiratory limb or inspiratory limb or both which depends on the site of obstruction.



DIFFUSION CAPACITY (DL):

Diffusion capacity of the lung is defined as the rate at which a gas enters the blood divided by its driving pressure. The driving pressure is the gradient. The factors on which the diffusion capacity depends on are the character of the alveolar capillary membrane, effective surface area of gas exchange, volume of blood in alveolar capillaries, rate of combination of gas with blood and cardiac output. Diffusion capacity is also influenced by the thickness of alveolar capillary membrane. The measurement of this provides information about the amount of functional capillaries which are in contact with ventilated air spaces. The gas which is used for the measurement of diffusion capacity is carbon monoxide. It has 200 times more affinity for haemoglobin than oxygen `and therefore it does not build up rapidly in plasma. Under normal conditions the

carbon monoxide concentration in the blood is low. Pulmonary capillary tension can be assumed to be zero.

DLCO is the measurement of the ease of the transfer of CO molecules from alveolar gas to the haemoglobin of the red blood cells in the pulmonary circulation. It is also known as the transfer factor of carbon monoxide, which describes the process more accurately. It is a measure of the interaction of alveolar surface area, alveolar capillary perfusion, the physical properties of the alveolar capillary interface, capillary volume, haemoglobin concentration, and the reaction rate of carbon monoxide and haemoglobin.

The total diffusing capacity of the whole lung is the sum of the diffusing capacity of the pulmonary membrane component and the capacity of pulmonary capillary blood volume. DLCO – It is the capacity of the lungs to transfer carbon monoxide (ml/min/mmHg). DLCOc – It is the DLCO corrected for haemoglobin in ml/min/mmHg. DLVA – It is the DLCO corrected for volume in ml/min/mm Hg/lit. DLVC – It is the DLCO corrected for both volume and haemoglobin in ml/min/mm Hg/lit.

As haemoglobin concentration is a very important measurement in the interpretation of DLCO, in anemic patients DLCO may be decreased.

The level of haemoglobin present in the blood and diffusing capacity are directly related. Because of this, correction for anemic patients is used to further delineate whether a DLCO is reduced due to anemia or due to parenchymal or interface limitation. Reduction in the diffusing capacity can be

classified as mild, moderate and severe. When the reduction is less than the lower limit of normal but greater than 60% predicted, it is classified as mild. When the reduction is between 40% and 60% of the predicted value, it is classified as moderate. When the reduction is <40%, it is severe.

Different methods used for measuring DLCO are Steady state method and Single breath technique. The most commonly performed and standardised technique is Single breath technique. In this test, the patient inhales a known volume of test gas that contains 10% helium, 0.3% carbon monoxide, 21% oxygen and the remaining is nitrogen. The patient is asked to inhale the test gas and holds breath for 10 seconds. Then the patient is asked to exhale to wash out a conservative overestimate of mechanical ad anatomical dead space. After this, an alveolar sample is collected. From the total lung volume, breath holding time and the initial and final alveolar concentrations of carbon monoxide, DLCO is calculated.

The exhaled helium concentration is used for the calculation of single breath estimate of total lung capacity and the initial alveolar concentration of carbon monoxide. The patients are instructed to avoid smoking for several hours before doing the test. The patients are also instructed not to consume alcoholic beverages for atleast 8 hrs prior to the test because alcohol vapours can affect the accuracy of some fuel cell types of carbon monoxide analysers. Normal DLCO at rest is 20-30 ml/min/mm Hg. It depends upon the lung size and is a measure of lung surface area available for gas exchange. When the

patient is unable to follow the instructions, it is a contraindication to do this test. Patients should be alert, oriented and they should be able to exhale completely and inhale to total lung capacity. They should be able to maintain an airtight seal on a mouthpiece and should be able to hold a large breath for 10 seconds.

Decrease in DLCO is seen in obstructive lung diseases like emphysema, cystic fibrosis. It is also decreased in parenchymal lung diseases like interstitial lung diseases caused by fibrogenic dusts like asbestosis, biologic dusts like allergic alveolitis, drug reactions like amiodarone, bleomycin and in sarcoidosis. DLCO may also be decreased in cases of systemic lupus erythematosus, progressive systemic sclerosis, mixed connective tissue diseases, Rheumatoid arthritis and in Wegener's granulamatosis. It is also decreased in most of the cardiovascular diseases like acute myocardial infarction, mitral stenosis, pulmonary hypertension and pulmonary edema. Anemia, chronic renal failure, chronic hemodialysis , cigarette smoking and bronchitis obliterans organising pneumonia are some of the other diseases where there is decrease in DLCO.

Increase in DLCO is seen in diseases like polycythemia, pulmonary hemorrhage, left to right intracardiac shunts and exercise.

TEST FOR PULMONARY COMPLIANCE :

Compliance is defined as the change in volume per unit change in pressure. It includes three components:

- Lung compliance is the change in lung volume per unit change in alveolar to intrathoracic pressure gradient. The normal value of lung compliance is 200 ml/cm of H2O in the upright position.
- Chest wall compliance is the change in lung volume per unit change in ambient to intrathoracic pressure gradient. The normal value of chest wall compliance is 200 ml/ sq.cm.
- Total compliance is the change in lung volume per unit change in alveolar/ambient pressure gradient. The normal value of total lung compliance is 100 ml/ cm of H2O. The patient is asked to swallow a latex balloon in the esophagus and connected via a catheter to a pressure gradient and the total compliance is measured. The changes in esophageal pressure reflectsintrapleural pressure and change in lung volume is measured with a spirometer.

TEST FOR PULMONARY RESISTANCE:

It is the combination of airway resistance and pulmonary tissue resistance. 90% of the pulmonary tissue resistance is due to airway resistance (Raw). The airway resistance which is measured is used for the evaluation of airway obstruction. This technique is rapid and non invasive. The patient is asked to pant once or twice per second through a mouth piece and with the nose

clip in place. During a normal breathing, a major part of the resistance to airflow comes from the nose, pharynx and larynx. The resistance offered by the nose is bypassed by using the mouth piece. The larynx is kept dilated by using the panting maneuver. It also reduces the total airflow resistance.

The airway resistance is measured by asking the patient to sit in a constant volume body plethysmograph. This is done by recording the airflow at the mouth by means of pneumatachyograph and thereby simultaneously recording the transpulmonary pressure with latex balloon. The normal value of airway resistance is 0.5-2cm/sec. The airway resistance varies non-linearly with lung volume. The airway conductance (Gaw) is the reciprocal of airway resistance . The airway conductance varies linearly with lung volume. Specific airway conductance is the ratio of airway conduction to lung volume. This is a highly reproducible measurement which signifies the caliber of the intrapulmonary airway.

DIABETES MELLITUS AND ANESTHESIA

Diabetes mellitus is a systemic disease. It impairs many functions of the respiratory system by affecting the lungs. It reduces the lung elasticity, produces pulmonary microangiopathy and also affects the lungs with chronic inflammatory changes. The lung volumes and diffusing capacity are decreased. In patients with autonomic neuropathy, hypoxic induced ventilator drive is reduced. Due to the impaired chemoreceptor activity, diabetic individuals are more prone for the development of respiratory depression from drugs such as opioids and sedative agents. In diabetic patients the joints become stiff and rigid. The joints which are supporting the airway such as temporomandibular joint, atlanto-occipital joint and cervical joint may be involved resulting in difficulty in the management of airway. This joint rigidity is thought to be caused by the non enzymatic glycosylation of proteins and abnormality in the cross linking of collagen in joints and also in other tissues. This results in difficulty in introducing the laryngoscopy and difficulty in intubating the patient. The rigidity of the joints can be tested by Prayer sign and Palm print test. The patient is asked to oppose the palms and fingers like doing in a prayer. The diabetic patients who are having stiff joints can have a positive test as they are not able to do this. The palm print test is used to test the rigidity of fourth and fifth interphalangeal joints. The palm and fingers of the dominant hand is painted with ink. Then the patient is asked to firmly press the hand on a white paper which is placed on a hard surface.

Diabetic autonomic neuropathy affects the cardiovascular system. It alters the compensatory mechanism which includes the baroreceptor reflexes. This causes increased risk for hemodynamic lability. This hemodynamic alteration is produced mainly with the change of patient position, when positive pressure ventilation is given and when there is sympathetic blockade as in case of neuraxial blocks. In this situation, the patient should be monitored very carefully. There should be meticulous maintainence of intravascular volume. In these patients the loss of heart rate variability is a high risk factor for the development of ventricular arrhythmias and sudden death. There is high chance for the development of silent myocardial ischemia in diabetic patients with autonomic neuropathy. Respiratory arrest is also commonly seen in diabetic patients with autonomic neuropathy.

The surgery poses many perioperative problems in diabetic patients. The magnitude of stress response to surgery depends on the site of surgery, amount of tissue injured during the surgery. The hormones such as catecholamines, cortisol and growth hormone are secreted in response to stress.These hormones alters the glucose homeostasis because they have anti-insulin and hyperglycaemic effects. The process such as glycogenolysis and gluconeogenesis are stimulated. The peripheral uptake of glucose is decreased leading to hyperglycemia and ketosis. The magnitude of inflammation decides the increase in blood sugar during surgery. This results in excessive release of inflammatory cytokines, tumor necrosis factor, interleukin 1 and interleukin 6.

In addition to these factors, immobility itself is associated with decreased skeletal muscle sensitivity to insulin leading on to hyperglycemia.

In fasting patients who are undergoing elective major intra-abdominal surgeries, the blood glucose levels will increase between 126 and 180 mg/dl. In the case of cardiac surgeries, where there will be relatively more stress, the disturbance of glucose homeostasis is greater resulting in the rise of blood glucose above 270 mg/dl in patients who are not having diabetes. In patients with diabetes this value may go above 360mg/dl. The increased stress in diabetic patients may results in the development of diabetic crisis such as diabetic ketoacidosis or hyperglycaemic hyperosmolar coma. In case of gastrointestinal surgeries, there may be the need for the prolonged period of nil oral which means interruption of oral intake. This prolonged starvation can lead on to ketosis. Therefore there may be necessary for the perioperative administration of insulin for controlling the blood sugar. When the patient returns to normal oral intake, the diabetic management can be reverted back to his presurgery protocol.

Hypoglycemia poses a major problem in the perioperative period. The altered level of consciousness may mask the signs and symptoms of hypoglycaemia in the perioperative period. The anesthesia and surgery is associated with circulatory disturbances. This causes alteration in the absorption of subcutaneous insulin.Therefore, administration of intravenous insulin is preferred in the perioperative period.

yperglycemia is also with associated with increased risk of complications such as wound infection, poor wound healing and poor neurological outcome in susceptible patients. Therefore, the goals for the management of diabetic patients in the perioperative period are minimal metabolic disruption, avoidance of untoward events and return to stable glycemic control as soon as possible. When hyperglycemia is left untreated, many adverse effects can occur. There may be chance for dehydration which results from the osmotic diuretic effect of glycosuria. Because of the accumulation of ketoacids and lactic acids, academia occurs. Untreated hyperglycemia can lead on to electrolyte imbalance such as potassium and magnesium. This electrolyte imbalance poses increased risk of arrhythmias.

PRINCIPLES OF ANESTHETIC MANAGEMENT IN DIABETES MELLITUS:

1. The diabetic patients are more prone for hypoglycaemia and ketosis because of overnight fasting. Therefore, one should give preference for the diabetic patients on the operating list.
2. There may be chance for delayed gastric emptying in patients with diabetic autonomic neuropathy. There may be undiagnosed gastroparesis which results in the prolonged retention of food in the stomach. This results in the increased risk for aspiration and regurgitation. The preoperative use of oral erythromycin on gastric motility was proven to be effective. In diabetic patients, a 12 hour fasting before surgery may be beneficial.

3. The use of intravenous fluids should be meticulous in the diabetic patients in the perioperative period. When Ringer's lactate is given in larger volumes, the lactate undergoes gluconeogenesis in the liver and complicates the blood sugar control. When normal saline is used in larger volumes, it increases the risk of hyperchloremic acidosis. Therefore there is no ideal solution for use in diabetic patients and so any fluids we are using should be used judiciously.
4. In the anesthetised patient, the measurement of blood glucose level should be done frequently, rapidly and accurately. This is because the need for glucose and insulin during this period is unpredictable and hypoglycemia may go undetected.
5. Close monitoring should be done in diabetic patients during surgery. The standard monitors such as ECG, SPO2, BP, ETCO2 and temperature probe should be used. When there is presence of other comorbidities, other advanced monitorin can be used.
6. The glycosylated haemoglobin (HbA1c) provides a valuable guide for the long term glycemic control during the preoperative check up. It has no role in the intra operative or in the postoperative period.
7. In diabetic patients receiving longer acting insulin, there is high risk for hypoglycaemia in the perioperative period if glucose is not supplemented. Perioperative administration of glucose increases the postoperative glucose utilization rates.

8. Insulin supplementation may be needed in patients with absolute insulin deficiency and infections in the intraoperative period for the prevention of lipolysis and proteolysis which can lead on to ketosis.

GENERAL ANESTHESIA AND DIABETES MELLITUS:

INDUCTION:

The choice of induction agent for general anesthesia in diabetic patients depends on the severity of associated systemic diseases which includes coronary artery disease, nephropathy, neuropathy and hypertension. Supplementation of epidural analgesia may be beneficial after due consideration of autonomic neuropathy, peripheral neuropathy and ischemic heart disease. But this should be avoided in patients with sepsis. When epidural analgesia is supplemented, the dose of opioid analgesics can be reduced thereby decreasing the side effects such as respiratory depression. Epidural analgesia also helps to blunt the stress induced neurohormonal response. Rapid sequence induction should be performed in diabetic patients with gastrointestinal symptoms because of the risk of regurgitation and aspiration. In patients with anticipated difficult intubation, awake fibreoptic bronchoscopy would be the preferred technique. Patients should be carefully induced with Etomidate or Thiopentone sodium with high dose fentanyl and midazolam because in patients with autonomic neuropathy exaggerated hypotension is common. In patients with extensive peripheral neuropathy Succinyl choline should be avoided due to the increased risk of potassium release in these patients. In the presence of renal dysfunction,

atracurium and mivacurium are the preferred muscle relaxants. In case of rapid sequence induction, rocuronium may be the preferred muscle relaxant.

MAINTAINANCE OF ANESTHESIA:

Anesthesia should be maintained with inhalational agents such as isoflurane or sevoflurane with an air oxygen mixture. Nitrous oxide can be used, but it should be avoided in patients with intestinal obstruction due to the risk of bowel distension. The patients with perforative peritonitis and sepsis should be put on postoperative elective ventilation to optimise their oxygen delivery. When the bowel is very distended and when the abdominal closure is very tense, there is need for postoperative elective ventilation. For this the airway pressures should be observed observed after the abdomen is closed. In the less severe cases, the patient can be reversed and extubated at the end of surgery. Before extubation, the patient should have adequate recovery of airway reflexes.

EFFECT OF ANESTHETIC DRUGS ON DIABETES MELLITUS:

Ketamine causes significant hyperglycemia. Etomidate blocks the adrenal streroidogenesis which results in the suppression of cortisol synthesis .This decreases the hyperglycaemic response to surgery in non diabetic patients. The effect of etomidate on diabetic patients have not been established yet. Diabetic patients have decreased ability for the clearance of lipids from the circulation. Therefore, in the intensive care unit, in diabetic patient who are receiving propofol for prolonged sedation, the dose should be adjusted.

The effect of inhalational agents such as halothane, enflurane, isoflurane and sevoflurane on blood sugar control has only in vitro significance. This invitro studies shows that the inhalational agents inhibits the insulin response to glucose in a reversible and dose dependent manner. The effect of inhalational agents on blood glucose level in vivo has not yet established.

The secretion of adrenocorticotrophic hormone (ACTH) and cortisol are decreased when high dose of benzodiazepines are used. The benzodiazepines stimulates the secretion of growth hormone but reduces the sympathetic stimulation resulting in decreased glycemic response to surgery. When midazolam is given in usual sedative doses, these effects are minimal.

When opioids are used in high doses, it produces hemodynamic, hormonal and metabolic stability. The high dose opioids block the entire sympathetic system and also the hypothalamic-pituitary axis effectively. This may be probably due to the direct effect of opioids on the hypothalamus and the higher centres. But it was found that midazolam and fentanyl, by reducing the glucose clearance results in hyperglycemia.

Ganglion blocking agents which are used for hypotensive anesthesia can block the sympathetically mediated hepatic gluconeogenesis resulting in hypoglycemia. When beta blockers are used, it results in slower recovery from hypoglycemia.

MANAGEMENT OF PERIOPERATIVE BLOOD SUGAR CONTROL:

The aims of perioperative control of blood sugar are

- To reduce mortality and morbidity.
- To avoid hypoglycaemia.
- To avoid hyperglycemia.
- To avoid ketosis.

Most of the problems happens when the blood glucose level is>180mgs%. But in many patients, there is need for the maintainance of tight blood sugar control. Due to the fear of hypoglycaemia, the blood sugar is usually maintained in somewhat a higher range. But, now with the easy availability of bedside glucose tests and increasing risk of hyperglycemia, permissive hyperglycemia is no longer acceptable. Therefore, measurement of regular blood sugar is very important for the prevention of catastrophic events due to the extremes of blood glucose level. The blood sugar should be monitored prior to the induction of anesthesia and every hour thereafter when insulin infusions are used intraoperatively.

The perioperative control of blood sugar depends on whether the patient is on meal plan alone or on oral hypoglycaemic agents or on insulin. It also depends on the type of surgery whether major or minor. Patients whose blood glucose is well controlled with meal plan alone do not require any special perioperative intervention for the blood sugar control. In these patients, fasting blood sugar should be measured on the morning of surgery. If the surgery lasts

for more than 1 hour, intraoperative monitoring of blood glucose level is essential. In patients undergoing major surgeries and in patients with poorly controlled blood glucose level, an intravenous infusion of insulin and dextrose should be considered and here there is need for hourly monitoring of blood glucose level intraoperatively. In case of minor surgeries, no specific therapy is required.

If the patient is on oral hypoglycaemic drugs like sulfonylureas, it should be discontinued 1 day prior to surgery except Chlorpropamide , which should be stopped 2-3 days prior to surgery. Metformin should be stopped 1-2 days prior to surgery in case of sick patients.

In case of minor surgeries, the patient should be asked to continue the usual diabetic treatment till night prior to surgery. The dose should be with hold on the morning of surgery. Dextrose containing solutions should be avoided. Blood glucose should be monitored intraoperatively and if needed the patient should be treated with dextrose insulin infusion. In diabetic patients, the oral intake should be restarted as early as possible postoperatively. In minor surgeries if there is perioperative hyperglycemia (200mg/dl) , it should be managed with small subcutaneous dose of short acting insulin with frequent monitoring of the blood sugar level to avoid hypoglycemia. In case of major surgeries, the patient should be switch over to regular insulin if the patient is on OHAs. The optimal fasting blood sugar value preoperatively is 80- 120 mgs% for elective surgeries. If the patient is on long acting insulin it should be

switched over to a combination of short acting and intermediate acting insulins 1-2 days before elective surgeries. The blood glucose level should be monitored every hourly intraoperatively and immediately after surgery. There should be 1 hour gap between the stoppage of intravenous insulin and the restarting of subcutaneous insulin. If the diabetic patients who is on insulin are planned for elective major surgeries, the patient should be admitted 2-3 days before surgery especially if the blood sugar control is suboptimal. The obligate glucose requirement for cells such as neurons and RBCs which uses only glucose as substrate is 2mg/kg/min. This can be provided by 5% or 10% dextrose containing solutions. 5% dextrose solution can be given at a rate of 100-125 ml/hr. The initial insulin infusion rate can be calculated as between one half and three fourths of the patient's calculated hourly requirement which is patient's daily dose divided by 24. The dose of insulin requirement is higher in septic patients, obese, critically ill, patients on steroids and in patients who are undergoing cardiopulmonary bypass surgery.

The tight sugar control refers to maintaining blood sugar within a narrow range usually between 70-110 mgs%. But this approach has the significant risk of hypoglycaemia. The threshold above which complications increase is also not known. The exact limit to maintain blood sugar levels for best outcome is not clearly documented. In patients such as cardiac surgeries, neuronal injuries, burns, transplant surgeries, critically ill patients and pregnant patients the tight blood sugar control has beneficial effects. The adverse neurological outcome

due to hyperglycemia is due to intracellular acidosis and hypoxic neuronal edema. Due to hyperglycemia, the stages of inflammation such as chemotaxis, phagocytosis, polymorphonuclear neutrophils adhesion and apoptosis are impaired leading to increase in the risk of infection. In cardiac surgeries, the risk of infection is higher in patients with poor postoperative blood sugar control both in diabetics and also in non diabetics.

HYPERGLYCEMIC EMERGENCIES:

This includes Diabetic ketoacidosis (DKA) and Hyperglycemic Hyperosmolar State(HHS). The mechanism behind both these disorders is decrease in the action of circulating insulin along with increase in the counter regulatory hormones such as growth hormone, glucagon, catecholamines and cortisol. DKA is the condition where there is absolute deficiency of insulin whereas HHS is a condition with lesser degree of insulin deficiency. DKA is usually seen in type 1 diabetic patients. DKA can sometimes be the presenting feature of type 1 diabetes. In type 2 diabetic patients, DKA occurs during acute stress such as trauma, surgery and infection. Sometimes DKA occurs due to the discontinuation of antidiabetic treatment. It also occurs with the drugs that affects the carbohydrate metabolism. These drugs include steroids, thiazide diuretics, sympathomimetic drugs and some of the antipsychotic drugs. DKA is a proinflammatory state. It produces reactive oxygen species, cytokines,C-reactive protein and plasminogen activator inhibitor.

The clinical features includes polyuria, polydipsia, symptoms of acidosis and dehydration which includes respiratory distress, tachycardia, drowsiness, coma and abdominal pain. Other symptoms like vomiting, malaise and cramps can occur.

REGIONAL ANESTHESIA AND DIABETES MELLITUS

The advantages of regional anesthesia in diabetic patients are:

- Epidural anesthesia has minimal effect on the glucose metabolism when compared to general anesthesia. In pelvic and lower limb surgeries, if the surgery is done under epidural anesthesia the levels of circulating glucose, catecholamines and cortisol are not increased. In low spinal anesthesia, the insulin response to a bolus of glucose is well preserved. In case of high spinal level, due to sympathetic blockade the insulin secretion may be impaired. However, there is no clear cut evidence that either regional anesthesia or general anesthesia can have beneficial effects in terms of major complications or mortality.
- As the patient is awake in regional anesthesia , hypoglycaemia and hyperglycaemic coma can be easily detected.
- Regional anesthesia allows early return of normal oral intake and also early resumption of regular treatment regimen.
- Regional anesthesia avoids the potential problem which occurs during laryngoscopy and tracheal intubation.

- It provides excellent analgesia. It avoids the use of systemic analgesics such as NSAIDs which produces dangerous effects in patients with diabetic nephropathy. Respiratory depression caused by opioid analgesics is also avoided.

The disadvantages of regional anesthesia are:

- There is high chance for the risk of nerve damage. In diabetic patients, if epinephrine is used in regional anesthesia, there is more chance for ischemic nerve damage.
- Because of the persistence of vagal afferent input, epidural anesthesia may not be very effective in reducing stress response in case of upper abdominal and thoracic surgeries.
- In diabetic patients with autonomic neuropathy, regional anesthesia carries a high risk. Severe hypotension can occur in autonomic neuropathy because of the compromised cardiovascular compensatory mechanism. This produces deleterious effects in diabetic patients with associated comorbidities such as coronary artery disease, stroke and renovascular insufficiency.
- In diabetic patients, the risk of infection and vascular damage are increased when regional anesthesia is used.
- As diabetic patients are more prone for infections, epidural abscess can occur commonly following spinal and epidural anesthesia.

- Diabetic peripheral neuropathy which occurs after regional anesthesia may be confused with an anesthetic complication of nerve block. This results in medicolegal problems.

REVIEW OF LITERATURE

1. LUNG INDIA/VOL 30/ ISSUE 2/ APR-JUN 2013:

A study was conducted on pulmonary function tests in type 2 diabetic patients and their association with glycemic control and diabetes duration. They found that the parameters FEV1, FVC, FEF25%, PEFR were decreased significantly in patients with diabetes except FEV1/FVC. They also found out that there was no correlation between pulmonary function tests and the duration of diabetes as well as HbA1c. In this study, they concluded that diabetes mellitus which affects many systems, mainly affects lungs causing restrictive pattern of ventilatory changes. This may be due to the connective tissue glycosylation, decreased elastic recoiling and also due to inflammatory changes which occurs in the lungs.

2. LUNG INDIA/ VOL 28/ ISSUE 2/ APR-JUN 2011:

A study was conducted between January to June 2004 in 128 individuals who were non smokers and who do not have any acute or chronic respiratory diseases. They found out that the individuals with diabetes showed a significant reduction in FEV1, FVC and SVC when compared to the non diabetic individuals. They also found out that there was no significant difference in the maximum mid expiratory flow and forced expiratory ratio between the two groups. In this study, they also found out that there was a significantly increased level of triglycerides among the diabetic individuals. They concluded in this study that the diabetic patients

showed decreased lung function which is not dependent on smoking and it could be a chronic complication of diabetes mellitus.

3. INDIAN J MED RES 119, FEBRUARY 2004, PP 66-71:

A study was conducted in 29 type 2 diabetic patients and 11 healthy control subjects. They divided these patients into 3 groups. Group 1 includes type 2 diabetic patients with microangiopathic complications which includes diabetic nephropathy, diabetic neuropathy or retinopathy. Group 2 includes type 2 diabetic patients who do not have any of the complications. Group 3 includes non diabetic healthy control subjects. In this study, they did the pulmonary function test which includes strength of the respiratory muscles. They also studied the relationship between type 2 diabetes mellitus with anthropometric measurement, glycemic control and microangiopathic changes. In this study, they found out that there was a significant reduction in DLCO in type 2 diabetic patients who were complicated with microangiopathic changes. In this study, they also found out that there was no significant difference in FEV1, FVC, PEFR, maximum inspiratory and expiratory pressures among the 3 groups. They also found out that there was a significant correlation between DLCO and HbA1c, lipid profile and creatinine clearance in diabetic patients with microangiopathic changes.

4. INTERNATIONAL JOURNAL OF BIOMEDICAL AND ADVANCED RESEARCH(2012) 03 (08):

A study was conducted to evaluate the dynamic functions of the lung in individuals with type 2 diabetes mellitus. The dynamic functions of the lung such as PEFR , maximum expiratory pressure were recorded. In this study, they found out that PEFR and MEP were decreased in patients with type 2 diabetes mellitus when compared to the control group. They also found out that MEP shows a highly significant reduction. In this study, they concluded that the decrease in the dynamic functions of the lung in the study group is attributed to the respiratory muscle weakness.

5. THE FREMANTLE DIABETES STUDY/ DIABETES CARE/ MARCH 2004/ VOL 27:

A study was conducted to evaluate the relationship between diabetes mellitus, glycemic control and pulmonary function tests. They found out that airflow limitation and decreased lung volumes are mainly the chronic complications in patients with type 2 diabetes mellitus. They also found out that the severity of these complications was related to the glycemic exposure. They found out in this study that, after ruling out other risk factors, airflow limitation is an independent predictor of death in patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

Study type	:	Interventional
Design of study	:	Prospective case control study
Selection of study subjects	:	cases posted for elective major abdominal Surgeries.

1. INCLUSION CRITERIA:

   Elective major abdominal surgeries (eg. Open cholecystectomy, gastric surgeries, pancreatic surgeries).

   Duration of surgery <4 hrs.

   Both sexes.

   Age 40-60 yrs.

   ASA 1 & 2 patients.

   Non smokers.

2. EXCLUSION CRITERIA:

   Patient refusal.

   Patients with complaints of cough, sputum or dyspnoea.

   Patients with cardio-respiratory diseases or any major illness.

   Duration of surgery > 4 hrs.

   Patient requiring mechanical ventilation after surgery.

METHODOLOGY:

Fifty type 2 diabetic patients and fifty non- diabetic patients in the age group of 40-60 yrs undergoing elective major abdominal surgeries under General Anesthesia were selected for the study. General Anesthesia was standardised for both the groups. Pulmonary function tests were performed 60 minutes before and 60 minutes after the end of surgery. In the diabetic group, patients with duration of diabetes of 5-15 yrs were selected for the study.

Study group (diabetic group) - diabetic patients with duration of diabetes of 5-15 yrs in the age group of 40-60 yrs of both sexes undergoing elective major abdominal surgeries.

Control group(non-diabetic group) – non diabetic patients in the age group of 40-60 yrs of both sexes undergoing elective major abdominal surgeries.

The pulmonary function tests recorded were;

1. Forced expiratory volume in 1 second (FEV1).
2. Forced Vital Capacity (FVC).
3. FEV1/FVC ratio.
4. FEF 25%.
5. Peak Expiratory Flow Rate (PEFR).

OBSERVATIONS AND RESULTS

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,'t' value and 'p' values were calculated. 't' test was used to test the significance of difference between quantitative variables and Yate's and Fisher's chi square tests for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

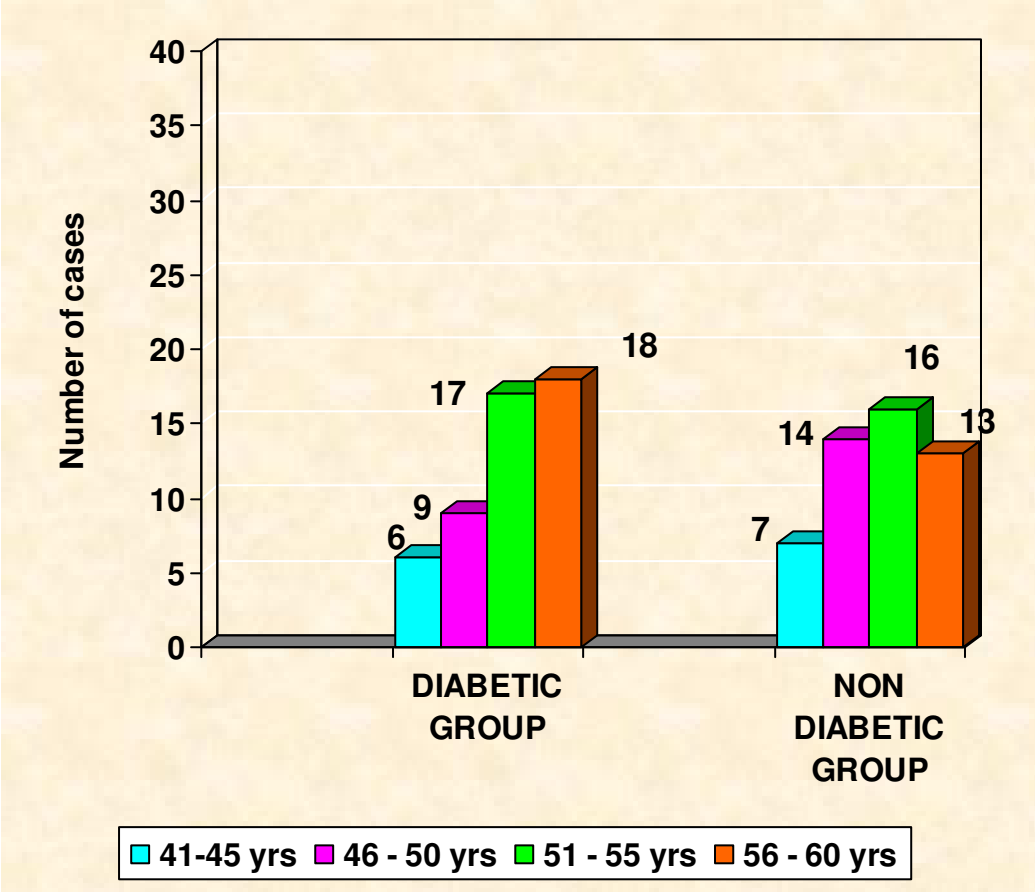
A: CHARACTERISTICS OF CASES STUDIED:

Table A1: Age Distribution

Age Group	No of Cases in			
	Diabetic Group		Non Diabetic Group	
	No	%	No	%
41 45 yrs	6	12	7	14
46 – 50 yrs	9	18	14	28
51 – 55 yrs	17	34	16	32
56 – 60 yrs	18	36	13	26
Total	**50**	**100**	**50**	**100**
Range	42 – 60yrs		42 – 60yrs	
Mean	52.9yrs		51.3 yrs	
SD	4.8yrs		4.8 yrs	
'p'	0.1036 Not significant			

The comparison of age group between diabetic group and non diabetic group shows that the 'P' value is 0.1036 which is not significant.

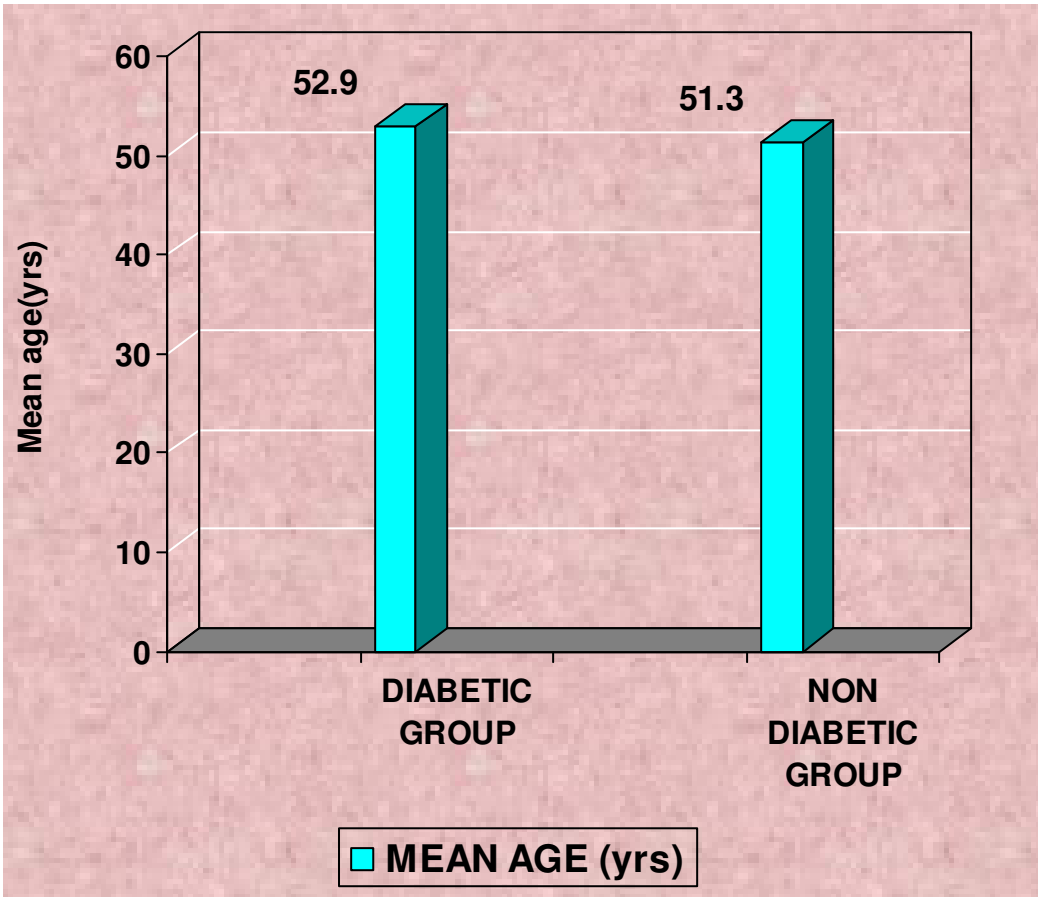
AGE DISTRIBUTION



COMPARISON OF AGE DISTRIBUTION

BETWEEN DIABETIC AND NON DIABETIC GROUP

MEAN AGE



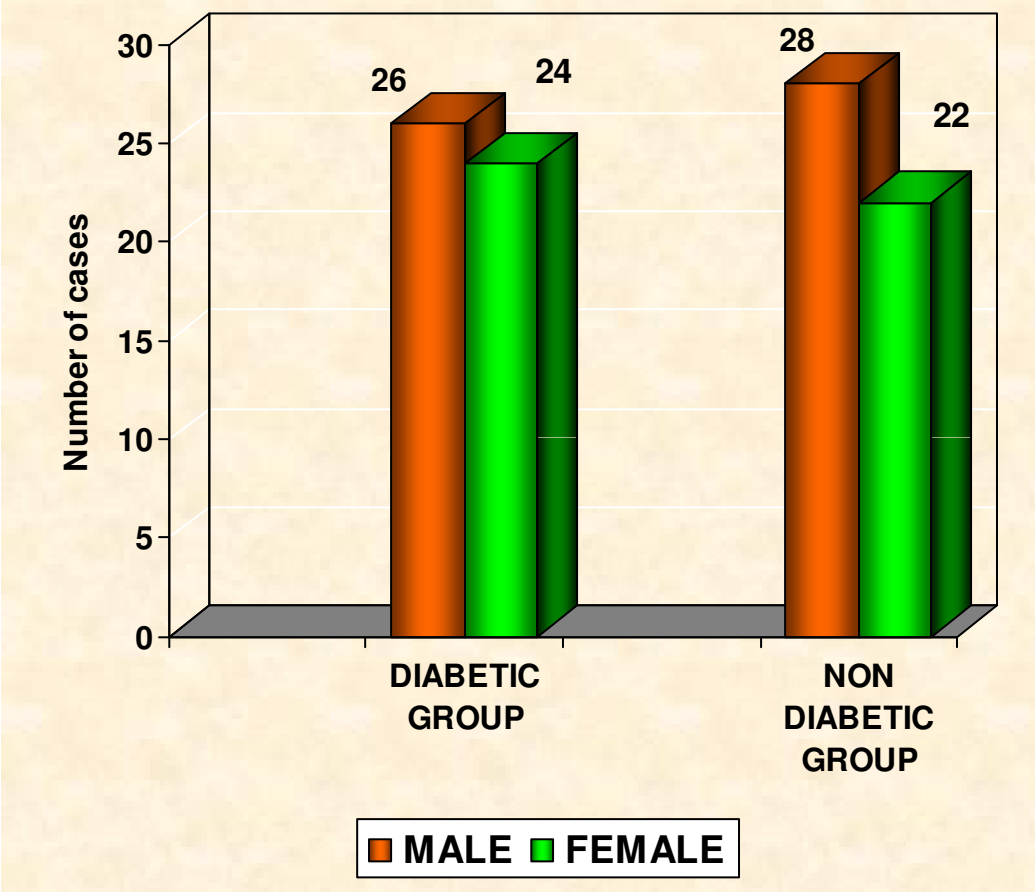
COMPARISON OF MEAN AGE BETWEEN DIABETIC AND NON DIABETIC GROUP

Table A2 : Sex Distribution

Sex	Diabetic Group		Non Diabetic Group	
	No	**%**	**No**	**%**
Male	26	52	28	56
Female	24	48	22	44
'p'	0.4206 Non Significant			

The comparison of sex distribution between diabetic group and non diabetic group shows that the 'P' value is 0.4206 which is not significant.

SEX DISTRIBUTION



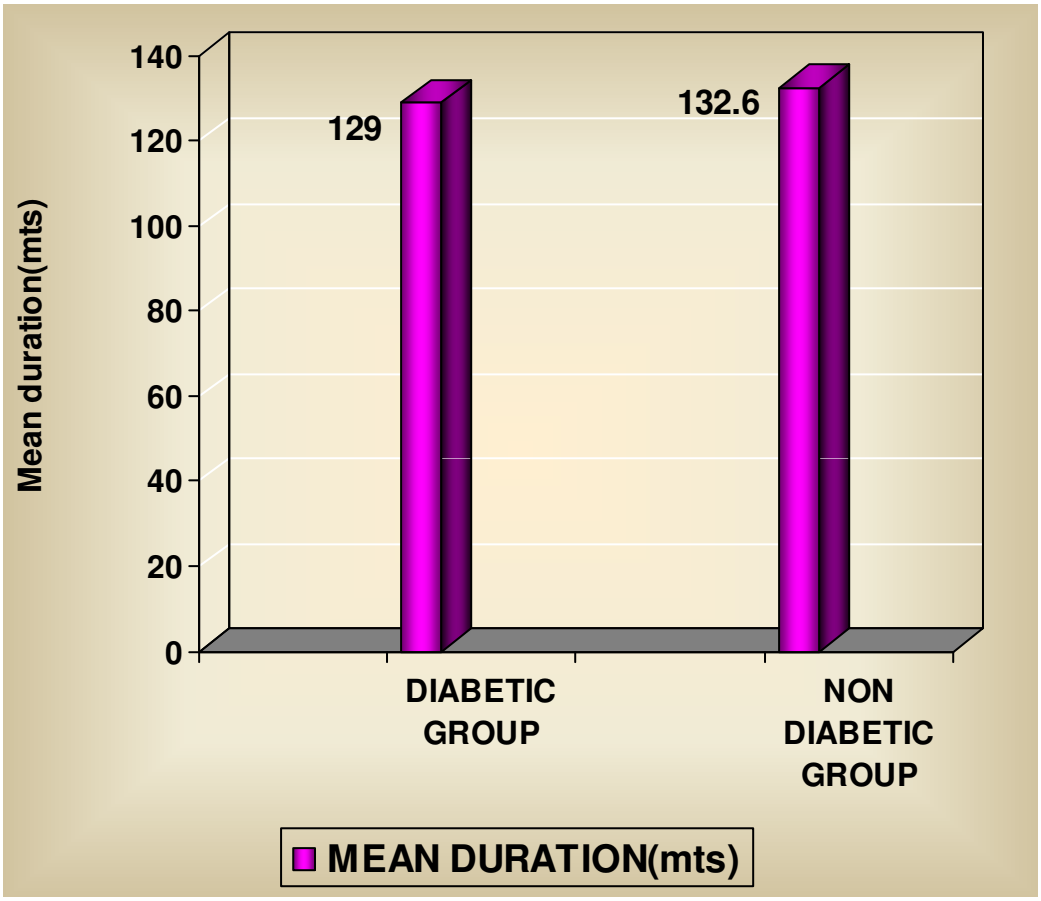
COMPARISON OF SEX DISTRIBUTION BETWEEN DIABETIC AND NON DIABETIC GROUP

Table A4: Duration of Surgery

Group	Duration of Surgery (minutes)	
	Mean	**SD**
Diabetic Group	129.0	24.2
Non Diabetic Group	132.6	20.9
'p'	0.4286 Not Significant	

The comparison of duration of surgery between diabetic group and non diabetic group shows that the 'P' value is 0.4286 which is not significant.

DURATION OF SURGERY



COMPARISON OF DURATION OF SURGERY

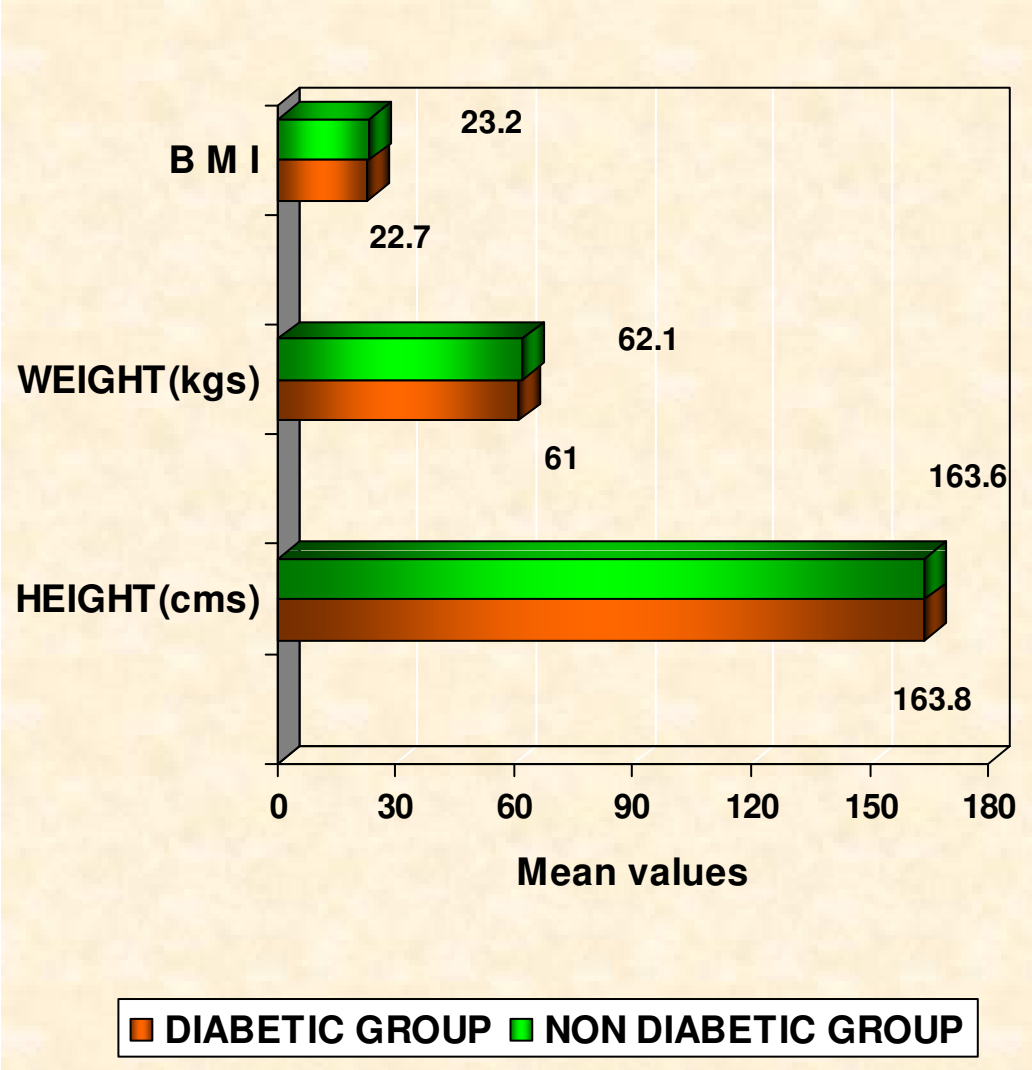
BETWEEN DIABETIC AND NON DIABETIC GROUP

TableA3 : Height / Weight / BMI

Group	Height (in cms)		Weight (in kgs)		BMI	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**
Diabetic Group	163.8	4.7	61.0	6.9	22.7	1.7
Non Diabetic Group	163.6	5.5	62.1	9.5	23.2	3.0
'p'	0.8455 Not Significant		0.5008 Not Significant		0.3109 Not Significant	

The comparison of height, weight and BMI between diabetic group and non diabetic group shows that the 'P' values are 0.8455, 0.5008 and 0.3109 respectively which are not significant.

HEIGHT/ WEIGHT/ B M I



COMPARISON OF HEIGHT, WEIGHT AND BMI BETWEEN DIABETIC AND NON DIABETIC GROUP

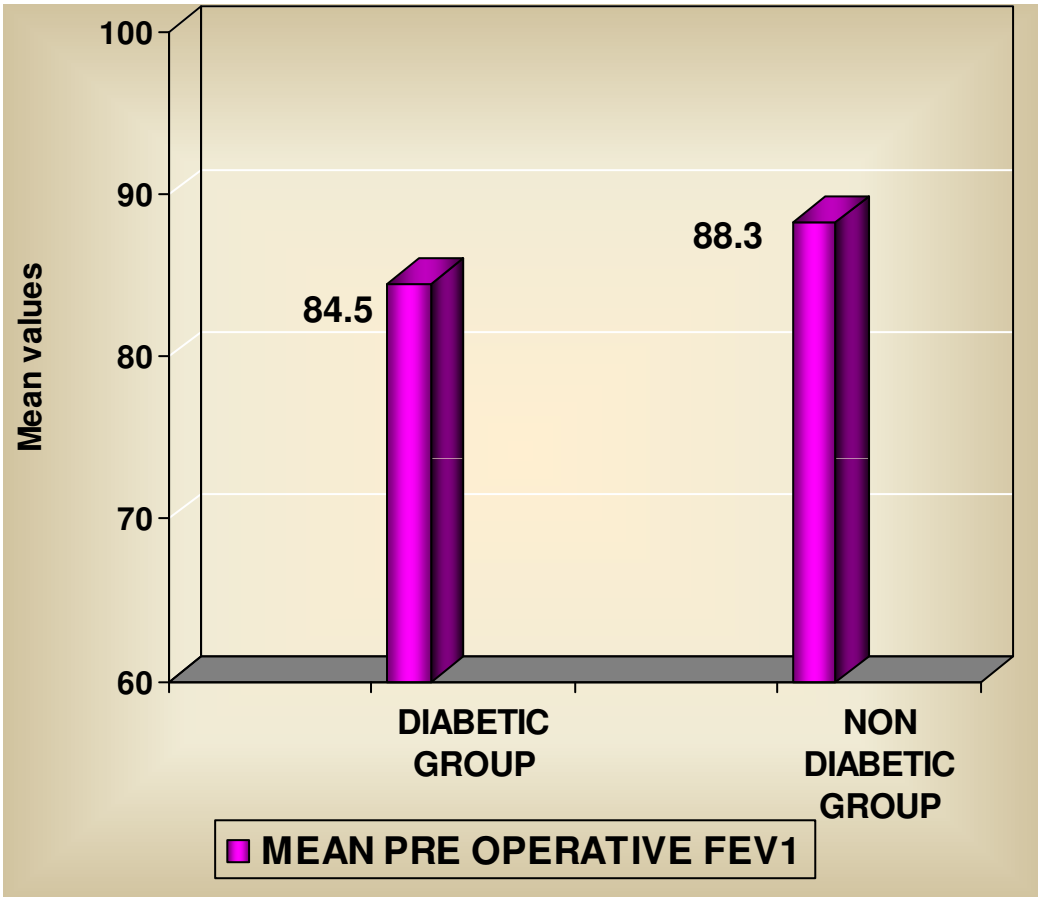
B: PRE OPERATIVE PULMONARY FUNCTION TEST VALUES IN DIABETIC AND NON DIABETIC PATIENTS

Table B1:Preoperative FEV 1

Group	**Pre Operative FEV 1**	
	Mean	**SD**
Diabetic Group	84.5	3.5
Non Diabetic Group	88.3	3.7
'p'	**<0.0001 Significant**	

The comparison of preoperative FEV1 in diabetic group and non diabetic group shows that the 'P' value is <0.0001 which is significant.

PRE OPERATIVE FEV1



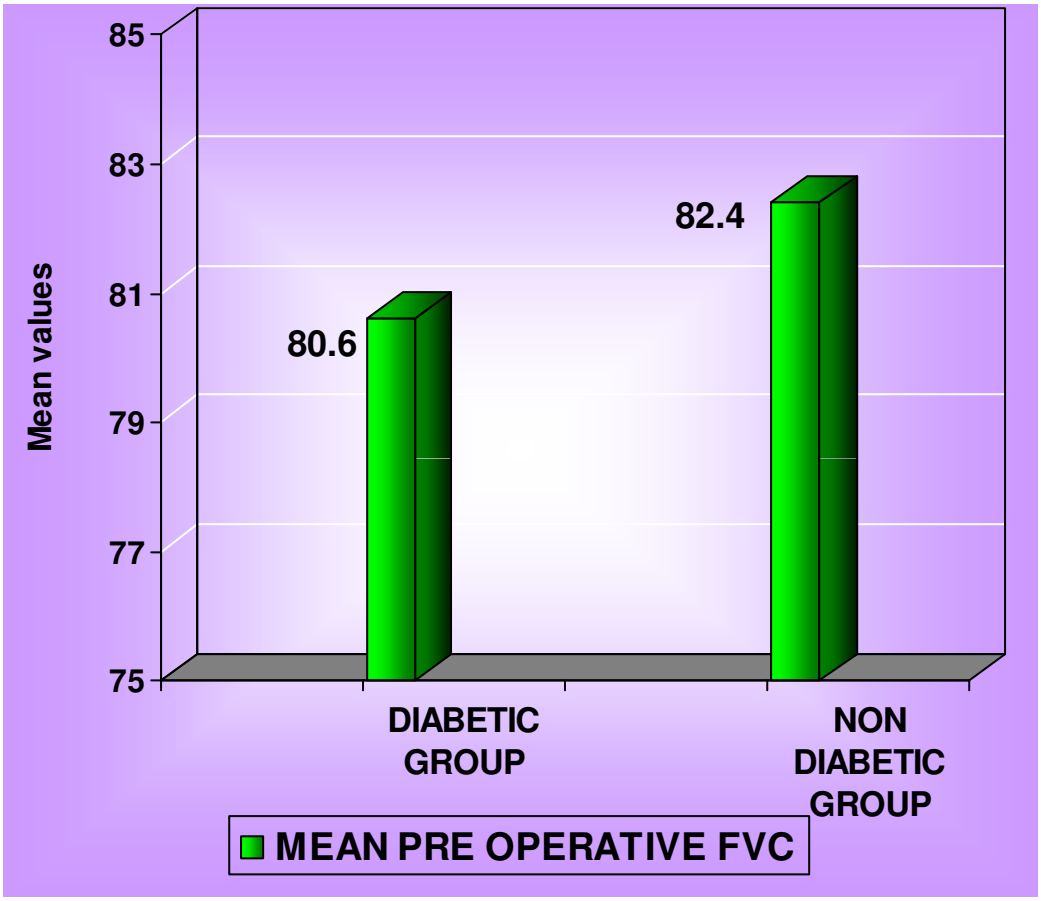
COMPARISON OF PREOPERATIVE FEV1 BETWEEN DIABETIC AND NON DIABETIC GROUP

Table B2 :Preoperative FVC

Group	Pre Operative FVC	
	Mean	**SD**
Diabetic Group	80.6	3.0
Non Diabetic Group	82.4	2.6
'p'	**0.0017 Significant**	

The comparison of preoperative FVC in diabetic group and non diabetic group shows that the 'P' value is 0.0017 which is significant.

PRE OPERATIVE FVC



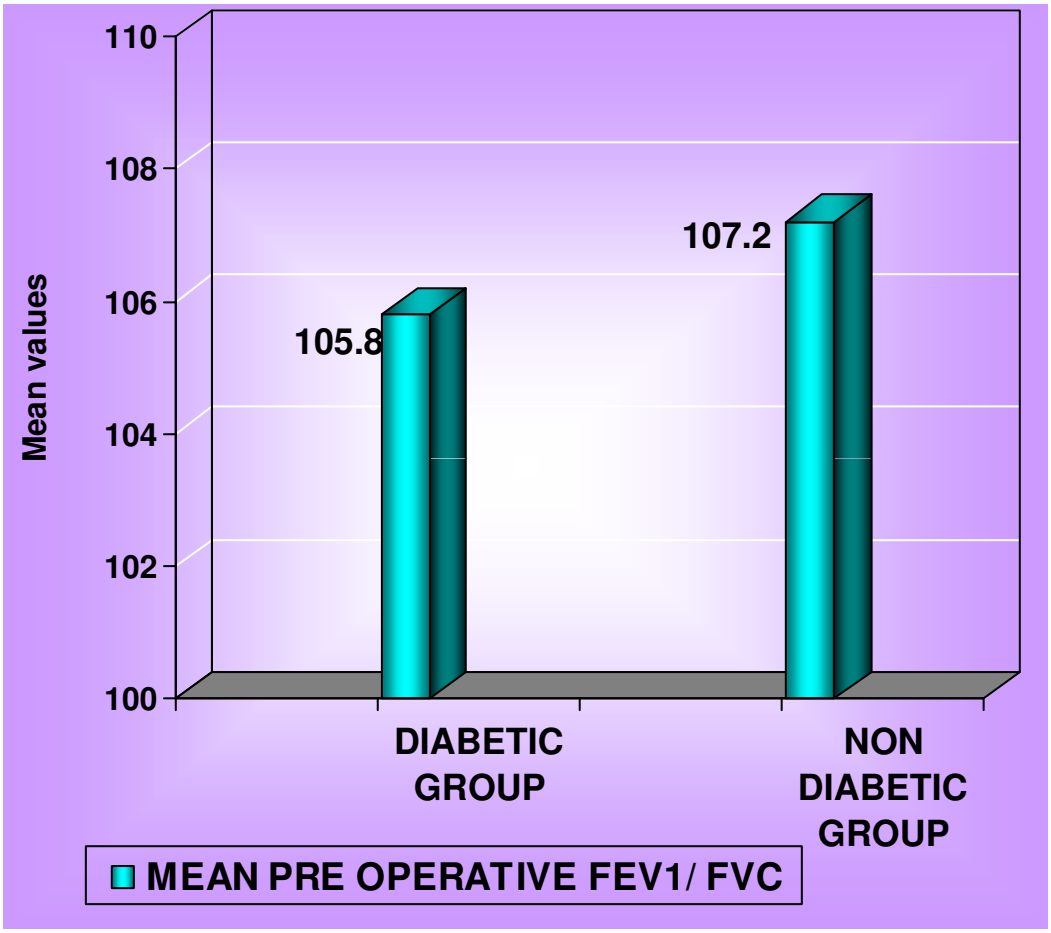
COMPARISON OF PREOPERATIVE FVC BETWEEN DIABETIC AND NON DIABETIC GROUP

Table B3 :Preoperative FEV1/FVC

Group	Pre OperativeFEV1/FVC	
	Mean	**SD**
Diabetic Group	105.8	7.0
Non Diabetic Group	107.2	4.4
'p'	0.0513 Not Significant	

The comparison of preoperative FEV1/FVC in diabetic group and non diabetic group shows that the 'P' value is 0.0513 which is not significant.

PRE OPERATIVE FEV1/ FVC



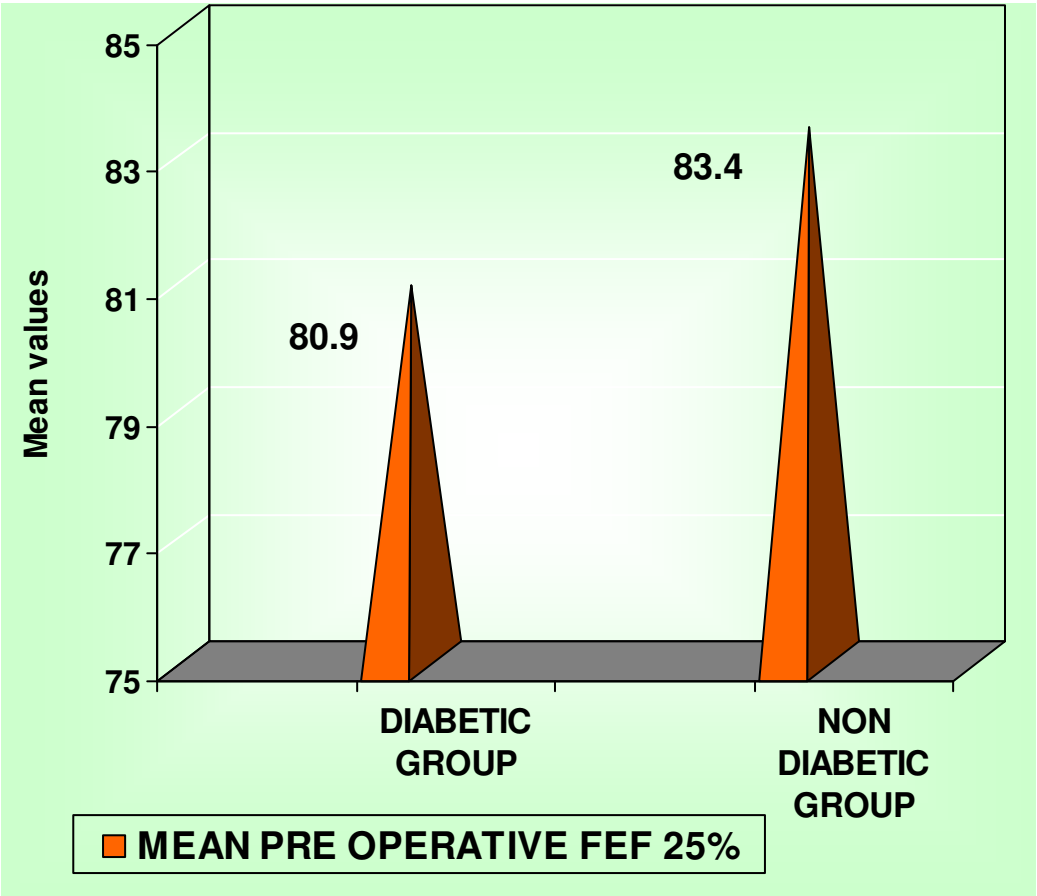
COMPARISON OF PREOPERATIVE FEV1/FVC BETWEEN DIABETIC AND NON DIABETIC GROUP

Table B4:Preoperative FEF 25%

Group	Pre Operative FEF 25%	
	Mean	**SD**
Diabetic Group	80.9	4.6
Non Diabetic Group	83.4	4.1
'p'	**<0.0053 Significant**	

The comparison of preoperative FEF 25% in diabetic group and non diabetic group shows that the 'P' value is <0.0053 which is significant.

PRE OPERATIVE FEF 25%



COMPARISON OF PREOPERATIVE FEF 25%

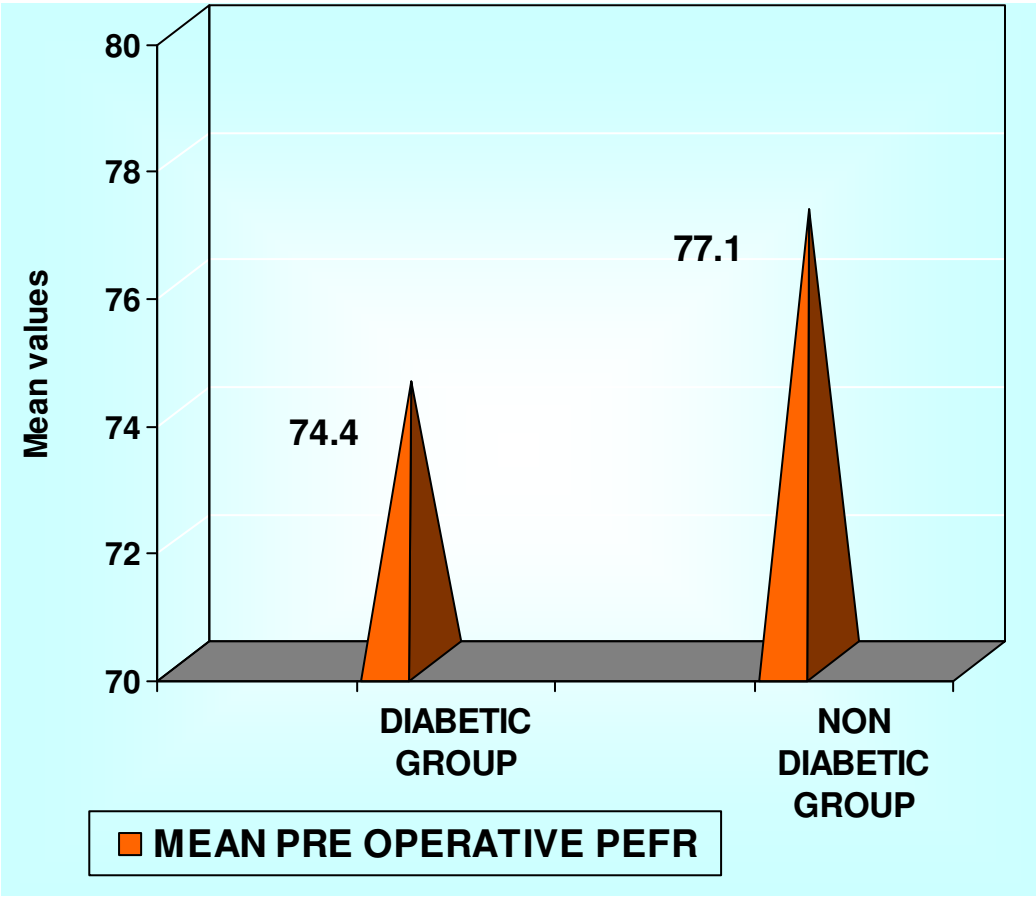
BETWEEN DIABETIC AND NON DIABETIC GROUP

Table B5 :Preoperative PEFR

Group	Pre Operative PEFR	
	Mean	**SD**
Diabetic Group	74.4	4.4
Non Diabetic Group	77.1	6.8
'p'	**0.0199 Significant**	

The comparison of preoperative PEFR in diabetic group and non diabetic group shows that the 'P' value is 0.0199 which is significant.

PRE OPERATIVE PEFR



COMPARISON OF PREOPERATIVE PEFR BETWEEN DIABETIC AND NON DIABETIC GROUP

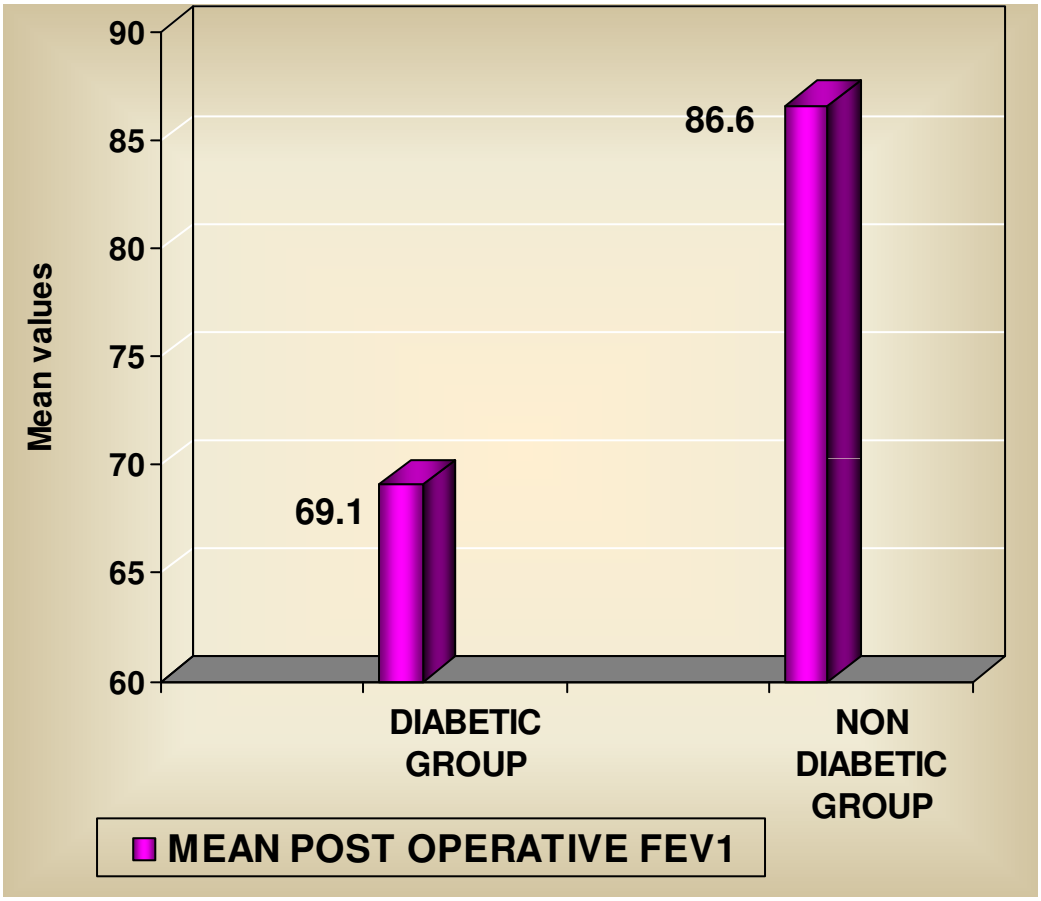
C :POST OPERATIVE PULMONARY FUNCTION TEST VALUES IN DIABETIC AND NON DIABETIC PATIENTS

Table C1 :Postoperative FEV1

Group	**Post operative FEV1**	
	Mean	**SD**
Diabetic Group	69.1	4.1
Non Diabetic Group	86.6	3.6
'p'	**<0.0001 Significant**	

The comparison of postoperative FEV1 in diabetic group and non diabetic group shows that the 'P' value is <0.0001 which is significant.

POST OPERATIVE FEV1



COMPARISON OF POSTOPERATIVE FEV1

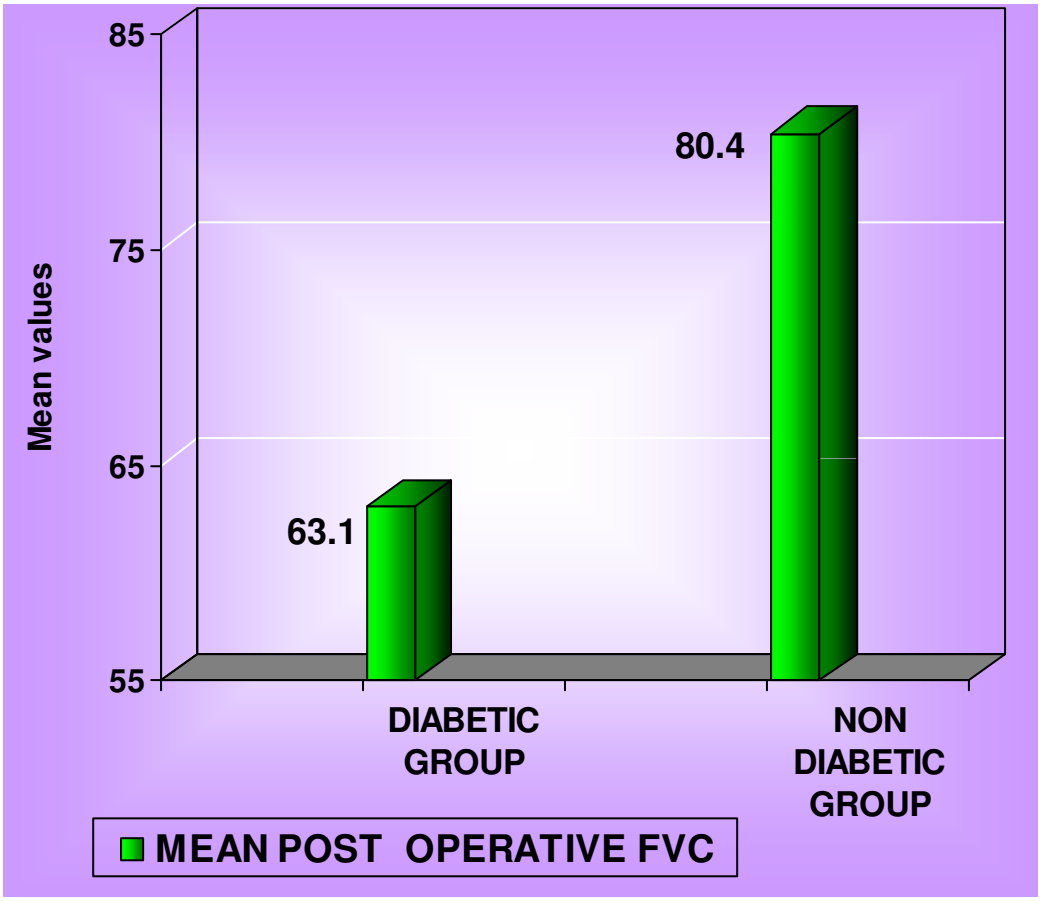
BETWEEN DIABETIC AND NON DIABETIC GROUP

Table C2 :Postoperative FVC

Group	Post operative FVC	
	Mean	**SD**
Diabetic Group	63.1	4.6
Non Diabetic Group	80.4	3.5
'p'	**<0.0001 Significant**	

The comparison of postoperative FVC in diabetic group and non diabetic group shows that the 'P' value is <0.0001 which is significant.

POST OPERATIVE FVC



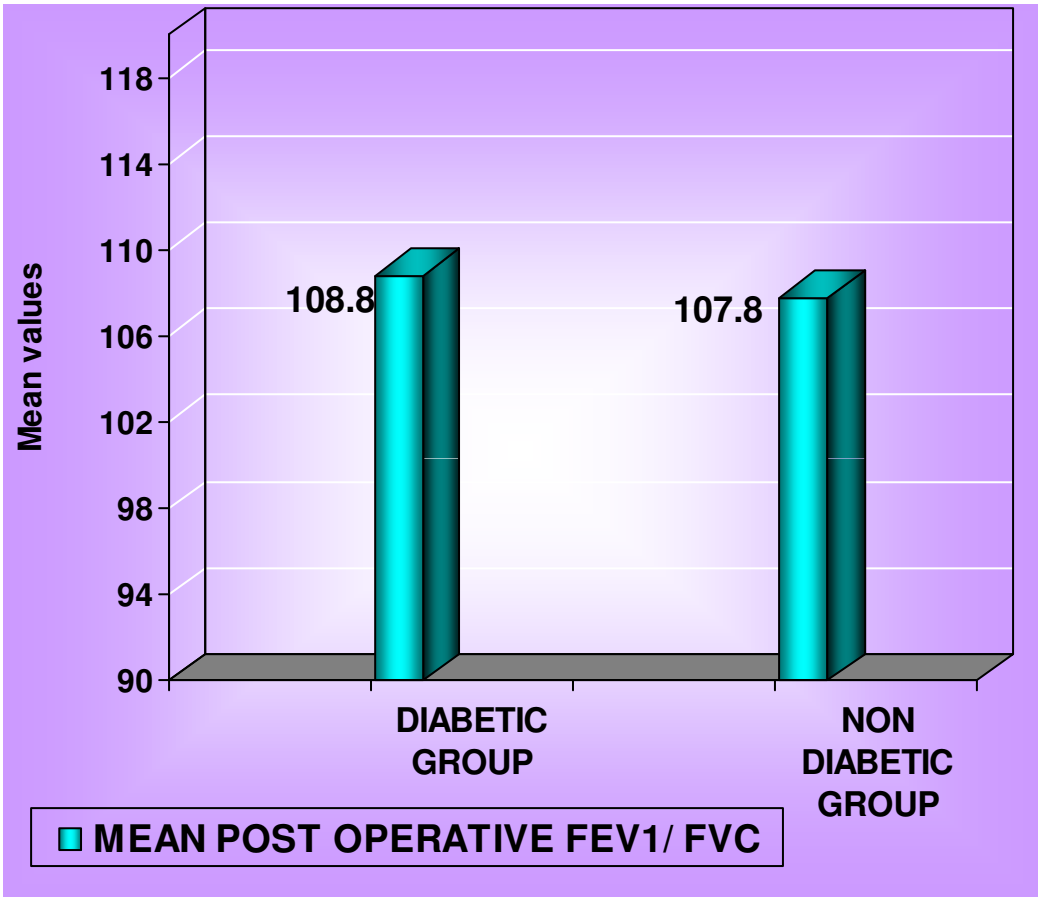
COMPARISON OF POSTOPERATIVE FVC BETWEEN DIABETIC AND NON DIABETIC GROUP

Table C3 :Postoperative FEV1/FVC

Group	Post operative FEV1/FVC	
	Mean	**SD**
Diabetic Group	108.8	6.3
Non Diabetic Group	107.8	4.9
'p'	0.2699 Not Significant	

The comparison of postoperative FEV1/FVC in diabetic group and non diabetic group shows that the 'P' value is 0.2699 which is not significant.

POST OPERATIVE FEV1/ FVC



COMPARISON OF POSTOPERATIVE FEV1/FVC

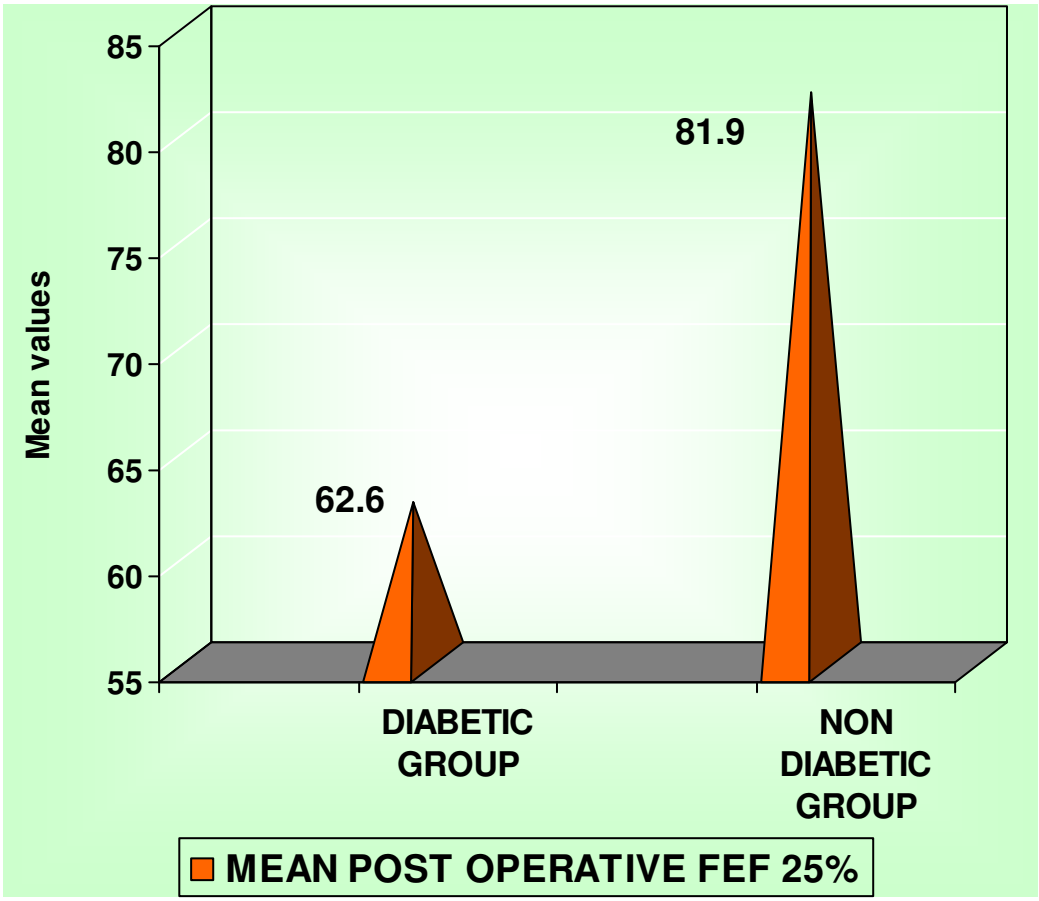
BETWEEN DIABETIC AND NON DIABETIC GROUP

Table C4 :Postoperative FEF 25%

Group	Post operative FEF 25%	
	Mean	**SD**
Diabetic Group	62.6	4.3
Non Diabetic Group	81.9	3.4
'p'	**<0.0001 Significant**	

The comparison of postoperative FEF 25% in diabetic group and non diabetic group shows that the 'P' value is <0.0001 which is significant.

POST OPERATIVE FEF 25%



COMPARISON OF POSTOPERATIVE FEF 25%

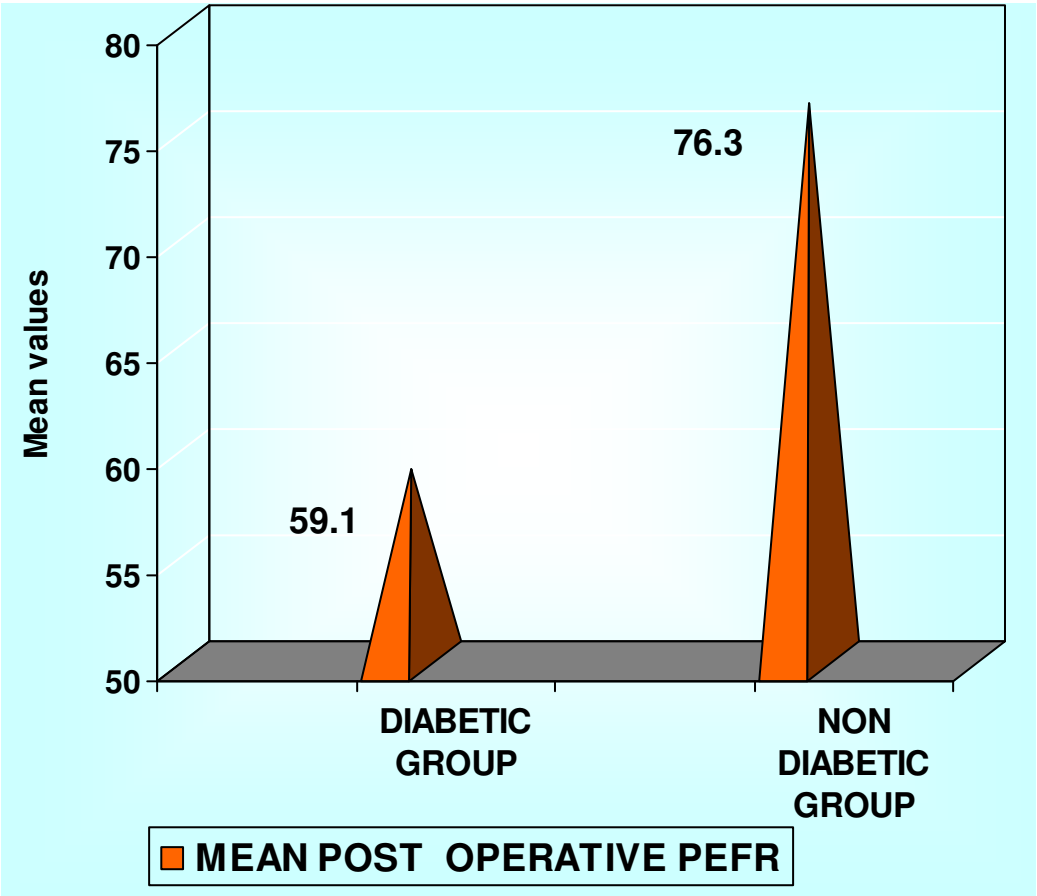
BETWEEN DIABETIC AND NON DIABETIC GROUP

Table C4:Postoperative PEFR

Group	Post operative PEFR	
	Mean	**SD**
Diabetic Group	59.1	3.4
Non Diabetic Group	76.3	5.6
'p'	**<0.0001 Significant**	

The comparison of postoperative PEFR in diabetic group and non diabetic group shows that the 'P' value is <0.0001 which is significant.

POST OPERATIVE PEFR



COMPARISON OF POSTOPERATIVE PEFR BETWEEN DIABETIC AND NON DIABETIC GROUP

D:PRE OPERATIVE AND POST OPERATIVE PULMONARY FUNCTION TEST VALUES IN DIABETIC AND NON DIABETIC PATIENTS.

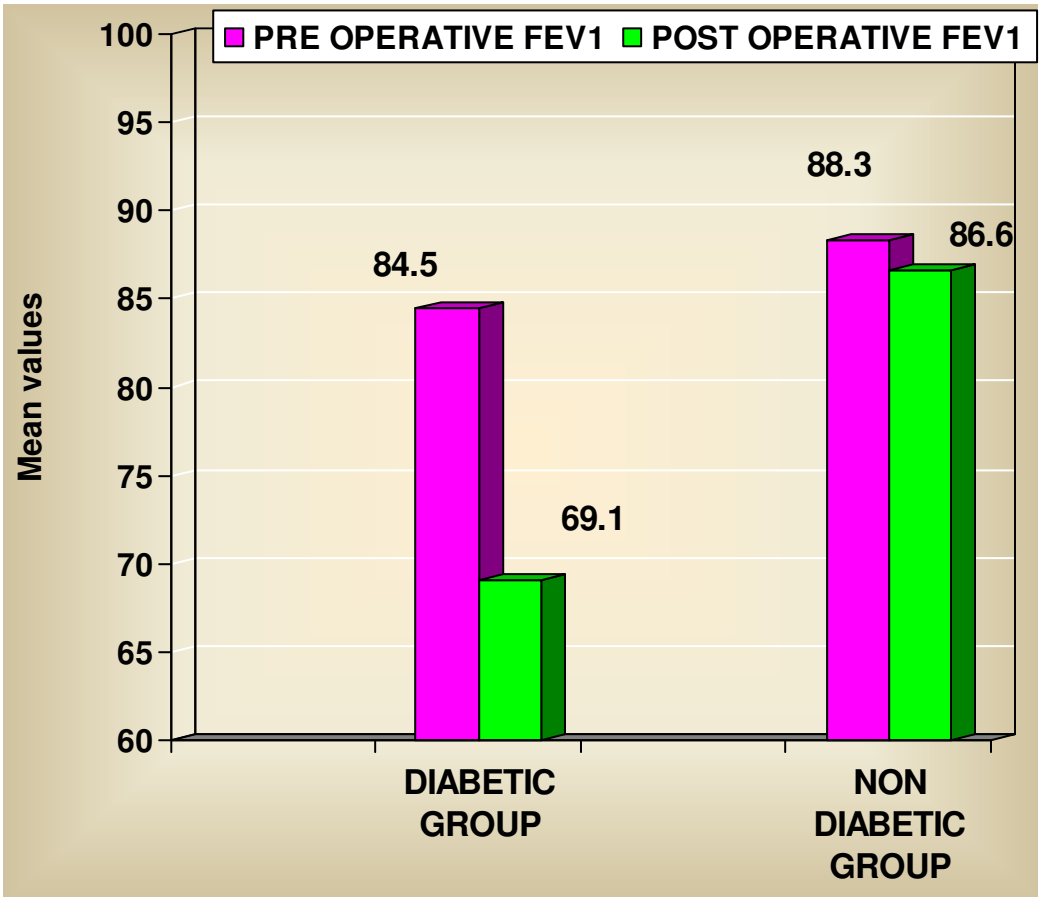
Table D1 :Preoperative and Postoperative FEV1

FEV1 Value	Diabetic Patients		Non Diabetic Patients	
	Mean	**SD**	**Mean**	**SD**
Pre operative	84.5	3.5	88.3	3.7
Post Operative	69.1	4.1	86.6	3.7
'p'	**<0.0001 Significant**		0.0703 Not significant	

The comparison of preoperative and postoperative FEV1 in diabetic patients shows that the 'P' value is <0.0001 which is significant.

The comparison of preoperative and postoperative FEV1 in non diabetic patients shows that the 'P' value is <0.0703 which is not significant.

PRE OPERATIVE &
POST OPERATIVE FEV1



COMPARISON OF PREOPERATIVE AND POSTOPERATIVE FEV1 IN DIABETIC GROUP AND NON DIABETIC GROUP

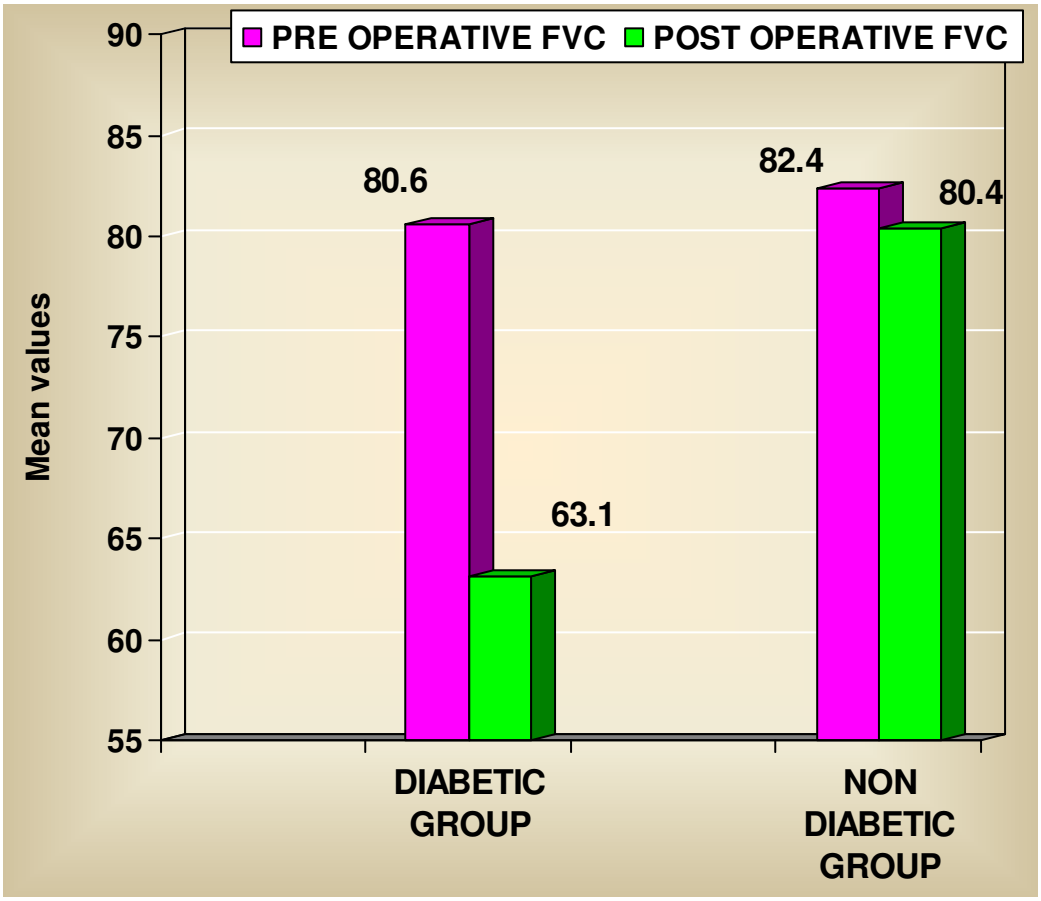
Table D2 :Preoperative and Postoperative FVC

FVC Value	Diabetic Patients		Non Diabetic Patients	
	Mean	**SD**	**Mean**	**SD**
Pre operative	80.6	3.0	82.4	4.6
Post Operative	63.1	4.6	80.4	3.5
'p'	**<0.0001 Significant**		0.1167 Not significant	

The comparison of preoperative and postoperative FVC in diabetic patients shows that the 'P' value is <0.0001 which is significant.

The comparison of preoperative and postoperative FVC in non diabetic patients shows that the 'P' value is <0.1167 which is not significant.

PRE OPERATIVE &
POST OPERATIVE FVC



COMPARISON OF PREOPERATIVE AND POSTOPERATIVE FVC IN DIABETIC GROUP AND NON DIABETIC GROUP

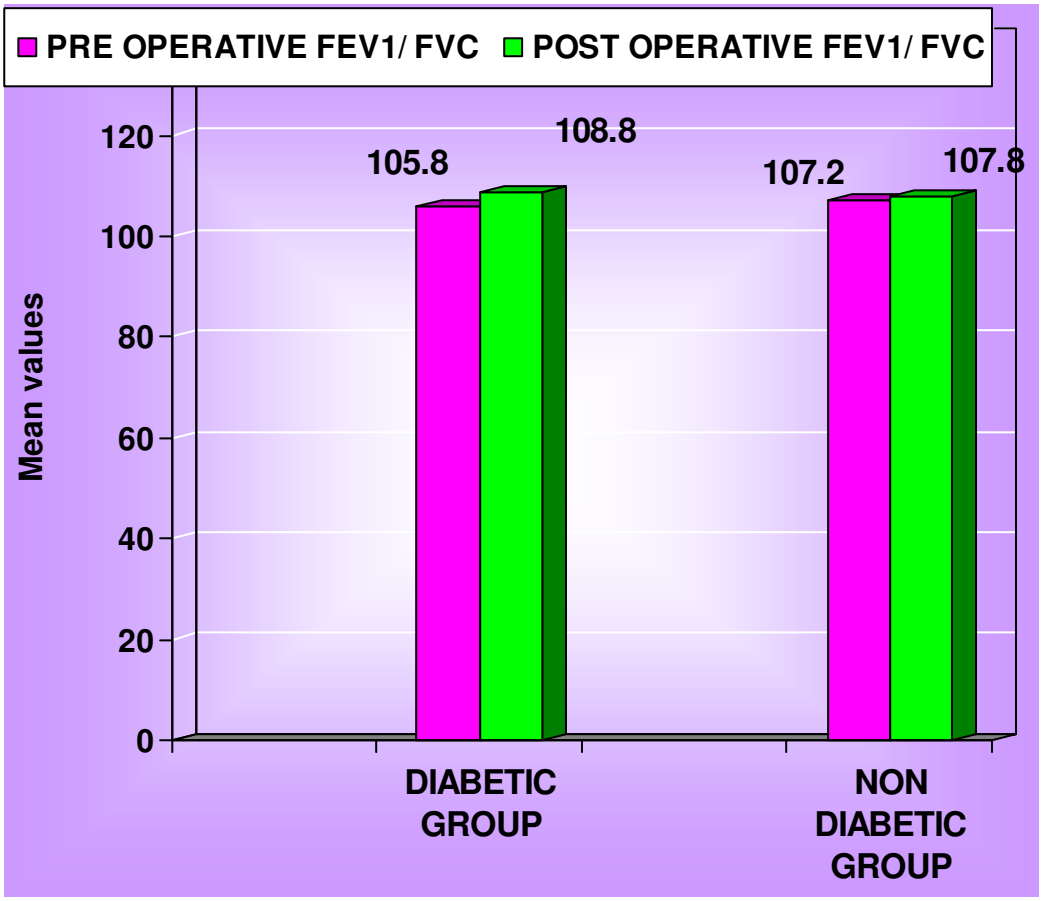
Table D3:Preoperative and Postoperative FEV1 / FVC

FEV1 / FVC Value	Diabetic Patients		Non Diabetic Patients	
	Mean	**SD**	**Mean**	**SD**
Pre operative	105.8	7.0	107.2	4.4
Post Operative	108.8	6.3	107.8	3.0
'p'	0.2946 Not significant		0.364 Not Significant	

The comparison of preoperative and postoperative FEV1/FVC in diabetic patients shows that the 'P' value is 0.2946 which is not significant.

The comparison of preoperative and postoperative FEV1/FVC in non diabetic patients shows that the 'P' value is 0.364 which is not significant.

PRE OPERATIVE &
POST OPERATIVE FEV1/ FVC



COMPARISON OF PREOPERATIVE AND POSTOPERATIVE FEV1/FVC IN DIABETIC GROUP AND NON DIABETIC GROUP

Table D4: Preoperative and Postoperative FEF 25%

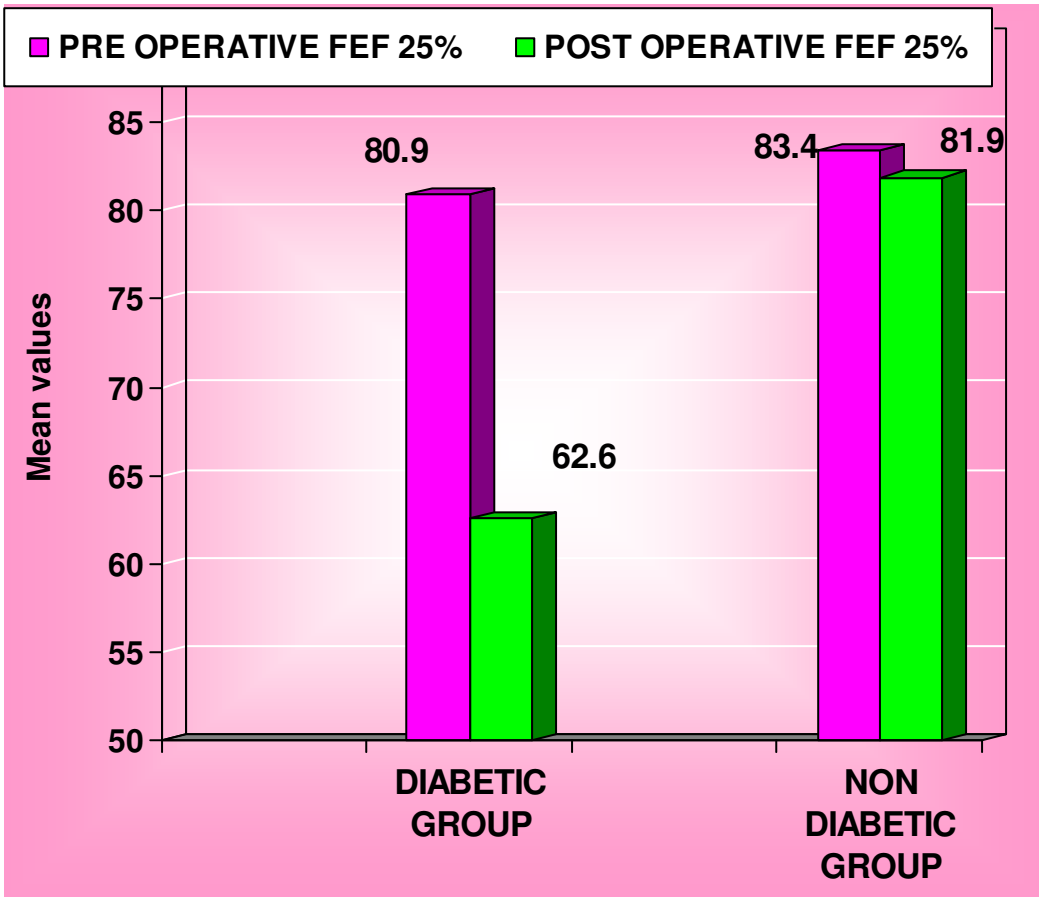
FEF 25% Value	Diabetic Patients		Non Diabetic Patients	
	Mean	**SD**	**Mean**	**SD**
Pre operative	80.9	4.6	83.4	4.1
Post Operative	62.6	4.3	81.9	3.4
'p'	**<0.0001 Significant**		0.0584 Not significant	

The comparison of preoperative and postoperative FEF 25% in diabetic patients shows that the 'P' value is <0.0001 which is significant.

The comparison of preoperative and postoperative FEF 25% in non diabetic patients shows that the 'P' value is 0.0584 which is not significant.

PRE OPERATIVE &

POST OPERATIVE FEF 25%



COMPARISON OF PREOPERATIVE AND POSTOPERATIVE FEF 25% IN DIABETIC GROUP AND NON DIABETIC GROUP

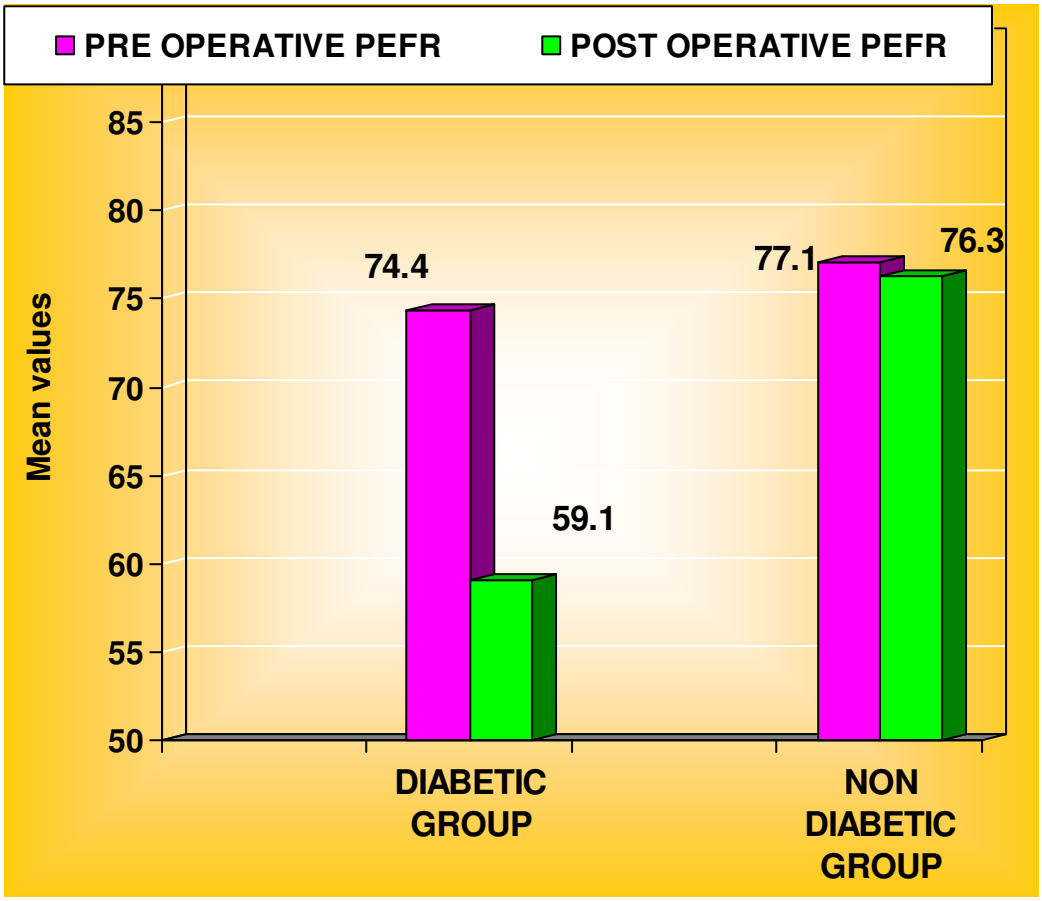
Table D5 :Preoperative and Postoperative PEFR

PEFR Value	Diabetic Patients		Non Diabetic Patients	
	Mean	**SD**	**Mean**	**SD**
Pre operative	74.4	4.4	77.1	6.8
Post Operative	59.1	3.4	76.3	5.6
'p'	**<0.0001 Significant**		0.5205 Not significant	

The comparison of preoperative and postoperative PEFR in diabetic patients shows that the 'P' value is <0.0001 which is significant.

The comparison of preoperative and postoperative PEFR in non diabetic patients shows that the 'P' value is 0.5205 which is not significant.

PRE OPERATIVE &
POST OPERATIVE PEFR



COMPARISON OF PREOPERATIVE AND POSTOPERATIVE PEFR IN DIABETIC GROUP AND NON DIABETIC GROUP

DISCUSSION

The result of this study shows that the pulmonary function test parameters such as FEV1, FVC, FEF25%, PEFR are significantly reduced except FEV1/FVC in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia when compared to the non diabetic patients. This significant difference occurs both preoperatively and postoperatively but the postoperative difference is highly significant clinically.

A similar study conducted by shah, et al. Showed that the pulmonary function tests were significantly decreased in diabetic patients when compared to the non diabetic healthy control subjects except FEV1/FVC causing a restrictive pattern of ventilator change. They also found out that there is no significant correlation between FVC and FEV1 with the duration of diabetes and glycosylated haemoglobin.

A study conducted by Van den Borst, et al. found out that the diabetes mellitus is associated with a statistically significant restrictive pattern of reduced lung function which is irrespective of body mass index, duration of diabetes and glycosylated haemoglobin.

The pathophysiology behind this is that reduced lung function in diabetic patients is the alteration in the alveolar-capillary network in the lungs producing microangiopathic changes. The alteration in the surfactant action of the alveoli, accelerated ageing process in the connective tissue cross links and the presence of non enzymatic glycosylation results in reduced lung functions. The non

enzymatic glycosylation leads to stiffening of lung parenchyma which results in restrictive pattern of lung disease. The increase in thickness of epithelium of alveoli, thickness of the capillary endothelium and basal laminae thickening are seen in the lungs of diabetic patients on electron microscopy.

The limitation of this study is that the correlation between pulmonary function tests with the duration of diabetes and glycosylated haemoglobin (HbA1c) could not be made out in this study. Another limitation is that whether the supplementation of epidural analgesia can provide improvement the pulmonary functions postoperatively could not be made out in this study.

SUMMARY

In this study, we evaluate the pulmonary function tests in type 2 diabetic patients.100 ASA 1 and ASA 2 patients of either sex were divided into two groups, 50 patients in each group.

- Group 1 includes type 2 diabetic patients who were undergoing elective major abdominal surgeries under general anesthesia.

- Group 2 includes non diabetic patients who were undergoing elective major abdominal surgeries under general anesthesia.

The study was done at Govt. Rajaji hospital and Madurai Medical College, Madurai between the period of 2012- 2014.

In our study the age of the patients, sex distribution , height, weight, BMI, duration of surgery were not statistically significant between the study group and control group.

Preoperative comparison of pulmonary function tests in diabetic and non diabetic patients shows a statistically significant decrease in FEV1, FVC, FEF 25%, PEFR except FEV1/FVC.

Postoperative comparison of pulmonary function tests in diabetic and non diabetic patients shows a statistically significant decrease in FEV1, FVC, FEF25%, PEFR except FEV1/FVC.

Preoperative and postoperative comparison of pulmonary function tests in non diabetic patients shows decrease in FEV1, FVC, FEF25%, PEFR but this is not statistically significant.

Preoperative and postoperative comparison of pulmonary function tests in diabetic patients shows a statistically significant decrease in FEV1, FVC, FEF25%, PEFR except FEV1/FVC.

CONCLUSION

From this study, it is concluded that pulmonary function tests are impaired in diabetic patients both preoperatively and postoperatively when compared to the non diabetic patients and is statistically significant.

The impairment of pulmonary function tests in diabetic patients postoperatively shows that this impairment is also significant clinically.

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PROFORMA

Name : IP. NO:

Age & sex : ASA:

Height :

Weight :

BMI :

Date of admission : Date of Discharge:

Diagnosis :

History : Smoking, duration of diabetes, cardiovascular or respiratory symptoms.

Clinical examination:

PR, BP, SPO2, CVS, RS.

Investigations :

FBS, PPBS, blood urea, serum creatinine,Urine sugar, ECG.

PREOPERATIVE PFT

(60 MINUTES BEFORE SURGERY)

PARAMETERS % PREDICTED VALUES	DIADETIC PATIENTS	NON DIABETIC PATIENTS
FEV1		
FVC		
FEV1/FVC		
FEF 25%		
PEFR		

POSTOPERATIVE PFT

(60 MINUTES AFTER THE END OF SURGERY)

PARAMETERS % PREDICTED VALUES	DIABETIC PATIENTS	NON DIABETIC PATIENTS
FEV1		
FVC		
FEV1/FVC		
FEF 25%		
PEFR		

MASTER CHART

PULMONARY FUNCTION TESTS IN NON DIABETIC PATIENTS

Sl. No.	NAME	AGE	SEX	IP. NO	GROUP	HEIGHT (CM)	WEIGHT (KG)	BMI	DURATION OF SURGERY (MINS)	PRE FEV1	PRE FVC	PRE FEV1/ FVC	PRE FEF 25%	PRE PEFR	POST FEV1	POST FVC	POST FEV1/ FVC	POST FEF 25%	POST PEFR
1	GANESAN	55	M	35834	ND	162	68	25.95	90	90	80	113	77	78	87	78	112	75	70
2	MARIMUTHU	58	M	38257	ND	158	62	24.89	175	88	84	105	86	75	88	80	110	78	76
3	RAJA	56	M	31062	ND	168	58	20.56	150	92	89	103	88	66	87	82	106	80	75
4	KUPPAMMAL	42	F	38587	ND	156	52	21.39	140	89	88	101	78	67	86	80	108	79	72
5	SHANMUGAM	54	M	37865	ND	167	59	21.22	120	87	82	106	87	88	88	82	107	85	80
6	SUGUBAR NISHA	56	F	31299	ND	157	65	26.42	145	85	80	106	78	87	82	77	106	75	70
7	THANGAM	45	F	23647	ND	154	57	24.05	130	89	80	111	79	69	85	84	101	78	67
8	SEKAR	52	M	35927	ND	169	69	24.21	125	85	82	104	87	89	84	79	106	86	75
9	KARTHIK	48	M	38918	ND	169	85	29.82	130	89	83	107	78	68	83	80	104	85	65
10	VASANTHI	52	F	40651	ND	160	57	22.26	145	90	86	105	83	71	88	82	107	80	65
11	KANDASAMY	46	M	41538	ND	167	59	21.22	130	94	85	111	86	82	92	87	106	82	84
12	PANDEESWARI	49	F	42059	ND	163	53	20	145	85	81	105	84	80	90	82	110	80	78
13	AMSAVALLI	52	F	41507	ND	158	54	21.68	135	82	82	100	84	67	88	83	106	79	80
14	RAVI	54	M	36130	ND	167	89	32.1	160	87	86	101	88	73	88	78	113	85	79
15	VELSAMY	57	M	54321	ND	167	89	32.01	180	92	88	105	89	72	92	82	112	87	73
16	KALIAMMAL	53	F	53472	ND	158	58	23.29	125	93	81	115	87	70	85	78	109	83	68
17	RAJATHI	45	F	50348	ND	159	58	23.01	110	83	83	100	87	68	91	85	107	86	65
18	AMBIGA	48	F	52360	ND	157	62	25.2	100	90	81	111	85	75	85	76	112	84	73
19	SHIVA	45	M	53683	ND	169	64	22.45	95	93	82	113	80	78	90	82	110	85	74
20	GOMATHY	53	F	53938	ND	158	65	26.1	115	92	84	110	79	83	83	84	99	76	79
21	LOORTHUMARY	57	F	55502	ND	157	62	25.2	130	85	80	106	78	82	91	83	110	84	76
22	RAGUNATHAN	52	M	54791	ND	168	68	24.11	90	84	80	105	89	76	80	75	107	88	73
23	SATHYANATHAN	46	M	54613	ND	170	78	26.98	140	89	81	110	86	67	84	78	108	84	76
24	ANNADURAI	48	M	57149	ND	169	72	25.26	150	87	83	105	83	68	80	76	105	80	77
25	RAMASAMY	46	M	56450	ND	172	82	27.79	130	88	82	107	85	89	81	79	103	81	76

MASTER CHART
PULMONARY FUNCTION TESTS IN NON DIABETIC PATIENTS

Sl.No.	NAME	AGE	SEX	IP. NO	GROUP	HEIGHT (CM)	WEIGHT (KG)	BMI	DURATION OF SURGERY (MINS)	PRE FEV1	PRE FVC	PRE FEV1/ FVC	PRE FEF 25%	PRE PEFR	POST FEV1	POST FVC	POST FEV1/ FVC	POST FEF 25%	POST PEFR
26	PANDI	46	M	56251	ND	169	57	20	115	86	81	106	81	78	88	80	110	86	82
27	KAVITHA	47	F	58311	ND	152	53	22.94	100	84	81	104	75	75	80	74	108	82	81
28	ILAYARAJA	56	M	59800	ND	166	61	22.18	115	80	80	100	72	77	84	78	108	82	84
29	DHANALAKSHMI	54	F	56438	ND	158	53	21.28	125	85	81	105	86	73	85	77	110	80	83
30	BANUPRIYA	52	F	56255	ND	156	66	27.16	135	87	82	106	84	79	80	73	110	78	85
31	VALAYUTHAM	48	M	17422	ND	167	57	20.5	170	83	83	100	89	84	90	82	110	88	80
32	KARUPPIAH	51	M	18365	ND	169	59	20.7	155	91	80	114	82	83	93	89	104	80	86
33	ESWARAN	50	M	13366	ND	168	63	22.34	160	94	89	106	85	87	85	82	104	81	80
34	IRULAMMAL	56	F	18261	ND	160	53	20.7	120	89	85	105	80	76	88	83	106	78	84
35	SARASWATHI	54	F	18891	ND	163	56	21.13	140	91	81	112	83	72	86	80	108	80	78
36	MARIAPPAN	58	M	89555	ND	168	64	22.69	130	94	82	115	89	89	89	82	109	87	85
37	VENKATESH	45	M	18475	ND	170	65	22.49	135	83	80	104	78	68	82	74	111	80	81
38	ANGUSAMY	46	M	97717	ND	168	68	24.11	120	84	82	102	86	86	91	85	107	85	78
39	PANDEESWARI	56	F	90998	ND	158	58	23.29	145	89	80	111	81	74	86	78	110	78	83
40	NEELAVATHI	53	F	97793	ND	162	55	20.99	150	85	78	109	83	70	84	78	108	80	76
41	SRINIVASAN	55	M	34201	ND	169	64	22.45	135	87	80	109	85	75	82	77	106	80	72
42	VISHNU	43	M	4067	ND	171	68	23.28	90	82	83	99	87	79	88	79	111	88	70
43	MUNIASAMY	42	M	5138	ND	169	60	21.05	145	91	81	112	88	80	87	83	105	83	77
44	SATHYA	57	F	4517	ND	159	59	23.41	125	89	80	111	82	75	88	82	107	80	70
45	PREMA	60	F	6202	ND	160	48	18.75	150	94	84	112	87	76	90	84	107	83	74
46	GOPAL	49	M	98055	ND	168	57	20.21	130	92	82	112	85	85	87	79	110	81	82
47	POTHUM PONNU	54	F	3421	ND	159	46	18.25	140	91	81	112	84	83	92	82	112	80	78
48	MURUGESWARI	58	F	7592	ND	157	51	20.73	145	89	81	110	79	80	86	76	113	85	72
49	JOSEPH	49	M	2439	ND	169	63	22.1	150	90	84	107	81	78	93	86	108	86	73
50	KUMARAN	56	M	9294	ND	168	58	20.56	120	95	86	110	85	84	90	84	107	80	74

MASTER CHART
PULMONARY FUNCTION TESTS IN DIABETIC PATIENTS

Sl. No.	NAME	AGE	SEX	IP. NO	GROUP	HEIGHT (CM)	WEIGHT (KG)	BMI	DURATION OF SURGERY (MINS)	PRE FEV1	PRE FVC	PRE FEV1/ FVC	PRE FEF 25%	PRE PEFR	POST FEV1	POST FVC	POST FEV1/ FVC	POST FEF 25%	POST PEFR
1	THIRUMALAI	54	M	5843	D	168	78	27.65	150	78	82	95	85	70	68	61	111	59	63
2	SARAVANAN	53	M	10255	D	166	72	26.18	170	85	77	110	83	77	72	70	103	66	61
3	SRIDEVI	45	F	94465	D	158	52	20.88	185	84	79	106	82	74	70	64	109	63	64
4	HEMA	47	F	2836	D	160	56	21.87	165	89	86	103	78	67	78	73	107	56	57
5	THENNARASU	45	M	6849	D	167	68	24.46	190	91	82	111	85	66	77	68	113	60	55
6	RAJENDRAN	49	M	12473	D	166	62	22.54	140	87	82	106	83	81	63	57	111	58	54
7	PANDI	57	M	3408	D	170	74	25.6	130	84	80	105	72	70	64	59	108	60	57
8	MEENA	56	F	6691	D	160	52	20.31	110	85	82	104	74	77	70	65	108	55	61
9	ANANDAVALLI	60	F	9012	D	158	54	21.68	145	86	80	108	73	79	73	63	116	58	65
10	RAJESWARI	55	F	11128	D	159	56	22.22	160	83	85	98	68	74	81	69	117	56	58
11	KASTHURI	59	F	13461	D	158	56	22.48	135	90	84	107	76	78	76	76	100	60	64
12	MURALI	53	M	13074	D	166	62	22.54	160	79	73	108	79	79	67	63	106	62	62
13	MANI	54	M	14355	D	168	56	19.85	140	87	82	106	75	81	66	59	112	58	67
14	MAHALINGAM	58	M	15301	D	167	70	25.17	125	82	84	98	76	75	64	57	112	59	56
15	LAKSHMI	44	F	13892	D	152	50	21.64	110	88	84	105	73	72	75	66	114	58	57
16	RAJAMMAL	42	F	1790	D	162	56	21.37	100	90	83	108	87	73	77	73	105	65	58
17	BALAMURUGAN	48	M	13241	D	166	66	24	140	84	81	104	90	78	68	62	110	67	55
18	GANAPATHY	56	M	18325	D	168	67	23.75	125	80	80	100	82	75	69	66	105	59	64
19	MUTHU	52	M	36297	D	170	59	20.41	160	82	78	105	84	76	66	60	110	69	58
20	KRISHNAVENI	55	F	10231	D	158	55	22.08	125	84	80	105	89	68	71	67	106	70	60
21	AMSALAKSHMI	58	F	19106	D	162	57	21.75	115	83	79	105	87	71	73	68	107	61	57
22	SARAVANAN	49	M	16530	D	170	67	23.18	90	82	77	106	81	65	67	64	105	55	56
23	ALAGU	45	F	26252	D	159	62	24.6	110	83	78	106	83	69	69	62	111	60	63
24	KALIDEVI	56	F	29631	D	163	65	24.52	150	84	80	105	83	69	66	61	108	59	60
25	KOTTASAMY	55	M	28909	D	167	69	24.82	145	84	80	105	84	74	69	63	110	60	55

MASTER CHART
PULMONARY FUNCTION TESTS IN DIABETIC PATIENTS

Sl. No.	NAME	AGE	SEX	IP. NO	GROUP	HEIGHT (CM)	WEIGHT (KG)	BMI	DURATION OF SURGERY (MINS)	PRE FEV1	PRE FVC	PRE FEV1/ FVC	PRE FEF 25%	PRE PEFR	POST FEV1	POST FVC	POST FEV1/ FVC	POST FEF 25%	POST PEFR
26	RAJA	58	M	29413	D	168	67	23.75	160	84	80	105	79	78	71	64	111	62	56
27	SAROJA	53	F	30360	D	159	56	22.22	130	83	79	105	78	76	64	57	112	58	55
28	MALAR	59	F	27241	D	157	53	21.54	120	81	78	104	80	74	72	64	113	54	59
29	HARIHARAN	52	M	30099	D	169	63	22.1	110	86	84	102	83	74	69	59	117	69	58
30	VALARMATHI	50	F	23980	D	162	54	20.61	115	85	81	105	81	73	70	65	108	68	58
31	SELVAM	49	M	29894	D	170	65	22.49	120	81	79	103	80	83	71	67	106	63	62
32	THAMARAI	44	F	29864	D	160	52	20.31	150	92	86	107	78	75	64	58	110	65	60
33	ANBURAJ	58	M	34397	D	171	68	23.28	145	94	88	107	85	71	69	57	121	66	63
34	CHITRA	51	F	32598	D	159	56	22.22	150	80	77	104	79	68	70	61	115	65	58
35	BASKARAN	48	M	37597	D	166	66	24	110	87	83	105	78	69	64	58	110	58	58
36	PANDIAN	56	M	37829	D	168	69	24.46	100	80	78	103	85	75	67	62	108	67	62
37	NAGARAJ	47	M	39713	D	168	72	25.53	115	82	83	99	87	78	72	70	103	68	58
38	MARIAMMAL	58	F	40668	D	158	55	22.08	140	80	76	105	82	87	63	60	105	63	64
39	RAMANATHAN	54	M	42500	D	167	59	21.22	95	82	78	105	87	72	64	59	108	65	58
40	MALLIKA	57	F	20354	D	160	57	22.26	110	82	78	105	84	74	70	61	115	66	59
41	CHANDRAN	46	M	51635	D	168	64	22.69	120	87	81	107	81	73	68	62	110	65	67
42	KALIAPPAN	57	M	55584	D	169	68	23.85	90	89	83	107	82	75	71	64	111	68	55
43	SHANTHI	55	F	53413	D	163	56	21.13	105	84	80	105	79	76	68	58	117	65	54
44	DEVADOSS	52	M	62395	D	166	69	25.09	110	82	78	105	76	79	64	57	112	67	57
45	NANDHINI	57	F	57894	D	159	52	20.63	105	88	84	105	79	75	69	61	113	63	58
46	VELU	53	M	52051	D	169	61	21.4	130	83	79	105	85	76	68	62	110	68	56
47	ELAVARASAN	57	M	63386	D	168	62	21.98	125	84	78	108	77	74	67	59	114	64	63
48	LAKSHMI	53	F	69878	D	160	54	21.09	115	85	79	108	83	73	65	60	108	65	56
49	VALLI	55	F	73479	D	162	55	20.99	105	88	85	104	80	77	71	70	101	68	58
50	MUNIYAMMAL	59	F	84976	D	158	57	22.89	100	82	77	106	84	76	67	63	106	66	57

INTRODUCTION

Diabetes mellitus is a systemic disease. It affects many organ systems in the human body. Among the various systems affected, respiratory system is also included. It is both a microvascular and also a macrovascular disease. In diabetes, the alveolar capillary network in the lung may be affected by microangiopathic changes. As this network has a large reserve , these microangiopthic changes may go unrecognised clinically. Symptoms such as dyspnoea develops only in the late stages of the disease.

Pulmonary functions can be assessed by spirometry. The pulmonary function tests includes both static and dynamic tests. The pulmonary function tests can be used for diabetic patients undergoing major surgeries such as cardiothoracic and abdominal surgeries. The status of the respiratory system can be assessed preoperatively by doing this test. It helps to optimise the patients preoperatively. The preoperative optimisation includes regular breathing exercises and control of blood glucose level. This results in better postoperative outcome of the diabetic patients . In this study , we evaluate the pulmonary function tests both preoperatively and postoperatively in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia and compare them with the non diabetic patients undergoing elective

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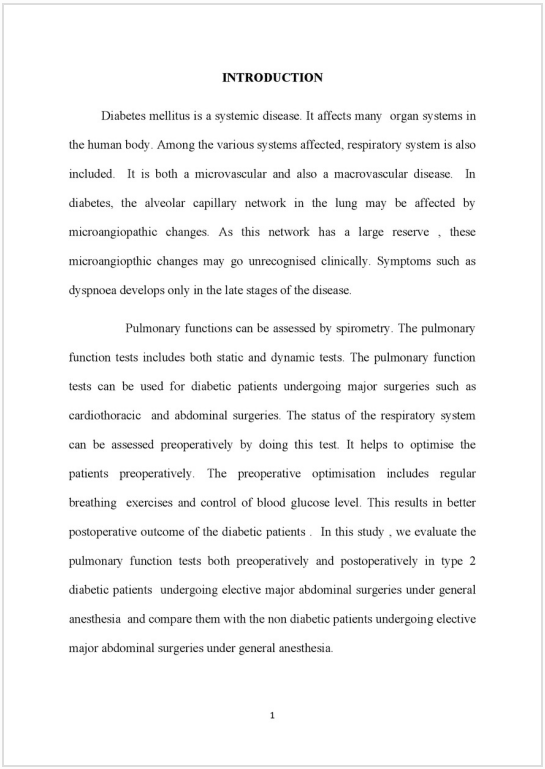

INTRODUCTION

Diabetes mellitus is a systemic disease. It affects many organ systems in the human body. Among the various systems affected, respiratory system is also included. It is both a microvascular and also a macrovascular disease. In diabetes, the alveolar capillary network in the lung may be affected by microangiopathic changes. As this network has a large reserve , these microangiopthic changes may go unrecognised clinically. Symptoms such as dyspnoea develops only in the late stages of the disease.

Pulmonary functions can be assessed by spirometry. The pulmonary function tests includes both static and dynamic tests. The pulmonary function tests can be used for diabetic patients undergoing major surgeries such as cardiothoracic and abdominal surgeries. The status of the respiratory system can be assessed preoperatively by doing this test. It helps to optimise the patients preoperatively. The preoperative optimisation includes regular breathing exercises and control of blood glucose level. This results in better postoperative outcome of the diabetic patients . In this study , we evaluate the pulmonary function tests both preoperatively and postoperatively in type 2 diabetic patients undergoing elective major abdominal surgeries under general anesthesia and compare them with the non diabetic patients undergoing elective major abdominal surgeries under general anesthesia.

1

Ref. No1864/E4/2/2014,

Govt. Rajaji Hospital,
Madurai.20. Dated: 29.03.2014

Institutional Review Board / Independent Ethics Committee.
Capt. Dr.B. Santhakumar, M.D., (F.M.,) deanmdu@gmail.com
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for March 2014
Approved list - Regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 05.03.2014, Wednesday at 10.00 am to 12.00.noon at the Auditorium, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr.V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029 nag9999@gmail.com	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology) drbkcmp@gmail.com	Professor & H.O.D of Surgical Oncology(Retired) D.No.32, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056 drparameswari@yahoo.com	Director of Pharmacology Madurai Medical College	Member
4. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048 svadivelmurugan_2007@rediffmail.com	Professor& H.O.D of Medicine Madurai Medical College	Member
5. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031 drsundarms@gmail.com	Professor & H.O.D of Surgery Madurai Medical College	Member
6. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650 lathadevadoss86@gmail.com	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
7. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127 palaramasamy2011@gmail.com	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
8. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599 pkmandco@gmail.com	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Projects was approved by the committee.

Name of P.G.	Course	Name of the Project	Remarks
Dr.M.Karthikeyan drkarthi2003@gmail.com	PG in MD (Anaesthesiology), Madurai Medical College and Government Rajaji Hospital, Madurai	Pulmonary function tests in type 2 diabetic patients undergoing major abdominal surgeries under general anaesthesia.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

29.3.14

Member Secretary **Chairman**
Ethical Committee

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

To
The above Applicant
-thro. Head of the Department concerned

19/3/14

forwarded to Dr. Karthikeyan

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
Madurai-625 020