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Understanding of Pulmonary Function Tests in View of Respiratory Physiology

Physiology

KEYWORDS:

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ABSTRACT Pulmonary function tests permit an accurate assessment of the functional state of respiratory system with quantification of the severity of the disease, if any. Particularly these tests provide important information relating to the process of ventilation and diffusion of gases across the alveolar membrane. The construal of pulmonary functions tests require concept of respiratory physiology as the ventilation is dependent to a large extent on the activity of respiratory muscle especially diaphragm and external intercostal muscle in relation to intra pulmonary and intrapleural pressures, lung compliance, alveolar surface tension and large and small airways resistance. Diffusion of gases across the alveolar membrane is principally determined by the surface area, integrity of the alveolar membrane and the pulmonary vascular bed. However, to conclude the respiratory status of any person the patient's clinical history, presentation and other basic investigations are also imperative. Although, profound amount of in depth information is available on all the aspects of PFTs, this review will be very helpful to understand these tests in a simple way.

Introduction

Pulmonary function tests (PFTs) provide reproducible assessment of the functional state of respiratory system with quantification of the severity of the disease, if any. Particularly these tests provide important information relating to the large and small airways, the pulmonary parenchyma and the size and integrity of the pulmonary capillary bed. Although they do not provide a diagnosis per se, but different patterns of abnormalities are seen in various respiratory diseases, and reflected in PFTs which helps to establish a diagnosis. Pulmonary function tests cannot by themselves distinguish among the probable causes of abnormalities, and must be interpreted in light of the patient's history, physical examination, and other supplementary investigations. Once a pattern is recognized, the diagnosis usually follows.

Although, profound amount of in depth information is available on all the aspects of PFTs, this review will be very helpful to understand these tests in a simple way especially by relating them to physiological component of respiration. Indications and contraindication for doing PFTs are as follows (1):

Indication

- To identify the severity of pulmonary impairment in related symptomatic cases.
- To monitor the effectiveness and fine tuning of therapy in the patients with known pulmonary disease.
- To monitor the effectiveness and fine tuning of therapeutic intervention in the patients with known pulmonary disease for progression and response to treatment e.g. ° Interstitial fibrosis ° COPD ° Asthma ° Pulmonary vascular disease.
- To investigate patients with diseases that may have a respiratorycomplications e.g. ° Connective tissue disorders ° Neuromuscular diseases.
- To screen patients with a risk of lung diseases e.g.° Exposure to pulmonary toxins such as radiation, environmental or occupational exposure, smoking and certain medication (eg, amiodarone, beryllium)
- To evaluate patients preoperatively.
- To evaluate the fitness and predicting safety in case taking part in strenuous exercises or ascending to high altitude.
- To evaluate the patient's condition for weaning from a ventilator.

Contraindications to perform

Recent Myocardial infarction
Unstable angina
Recent thoraco-abdominal surgery
Recent ophthalmic surgery
Thoracic or abdominal aneurysm
Current pneumothorax

Classification of PFTs, based on Lung Functions (2)

Respiratory assessment encompasses following functions of lungs-
1. Tests for assessing Ventilation function 2. Tests for assessing Gas Exchange function

Ventilation (3,4)

Ventilation refers to exchange of the gases between the external environment and the alveoli of the lungs. During ventilation, oxygen is carried to the alveoli from the environment, and carbon dioxide is carried away from the alveoli.

To understand ventilation, the following key areas are important in relation to a breathing cycle -

1.'Active' component -

It includes sequential respiratory muscle especially of diaphragm and external intercostal muscle action in relation to intra pulmonary and intrapleural pressures.

2. 'Passive' component -

a. Static Factor: Compliance
b. Dynamic Factor: Respiratory Resistance

1. Active component –

Air pressure differences and activity in respiratory muscles causes air flow during inhalation and exhalation and normal ventilation or breathing cycle is as follows-

Brainstem neuronal inputs to motor neurons → Inspiratory muscle contract (Diaphragm and External intercostals) → Thoracic cavity volume increases (Chest wall expands) → Intrapleural pressure (Pip) decreases to – 6 cm of H_2o from – 4 cm of H_2o (pleural space is between visceral and parietal pleurae and contain pleural fluid that exerts surface tension and due to attachment of visceral pleura to lungs and parietal pleura to chest wall a negative pressure is generated here due to the elastic forces of the lung [tending to contract] and the chest wall [tending to expand] under normal conditions. This negative intrapleural pressure helps keep airways open) → This causes stretch/expansion of lung of lungs (that adhere tightly to the thoracic wall due to surface tension of fluid in pleural space) → Intrapulmonary volume increases → Intra pulmonary pressure (intra alveolar pressure) decreases (atmospheric pressure 0 → &1 cm H_2O) → Air flows into lungs down its pressure gradient from ext environment until intrapulmonary pressure is 0 (atm pressure) → This then followed by expiration passively as inspiratory muscles stop contracting and all the pressure changes just get reversed (Fig-1 & Fig-2)

Fiaure 1- Luna and Chestwall forces

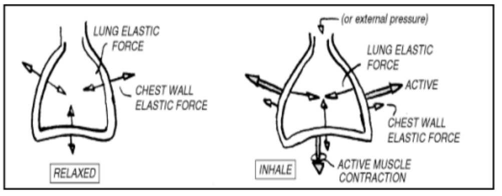
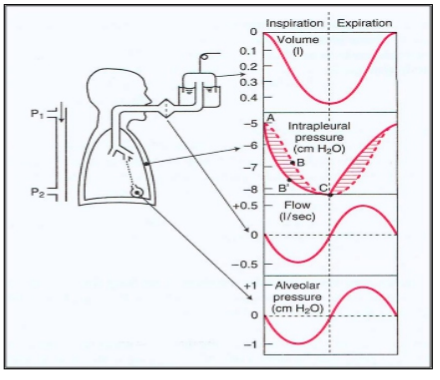


Figure 2 -various pressures in various parts of lungs



Conditions or diseases affecting ventilation, influencing active component (5)

A. Inadequate muscle contraction-

a) The central nervous system may fail to stimulate the respiratory muscles effectively. Examples – poliomyelitis, the virus seldom attacks respiratory-control centers in the brainstem. b) In amyotrophic lateral sclerosis (ALS or "Lou Gehrig's disease"), the disease process leads to the destruction of premotor and motor neurons, including those to the muscles of respiration.

c) Certain drug like barbiturate, opiates and all anaesthetic agents overdoses may transiently inhibit respiratory-control centers in the brainstem. d) impaired medullary blood flow - pressure, trauma, neoplasia e) The pain that accompanies chest surgery or other injuries to the chest can also severely limit the ability to ventilate.

B. Rigidity of chest wall

Despite normal neuro-muscular performance, chest-wall rigidity makes it difficult to increase thoracic volume –

a) Ankylosing spondylitis is an inflammatory disorder of the axial skeleton that may reduce the bucket-handle rotation of the ribs during quiet inspirations and the flexion and extension of the trunk during forced inspirations and expirations.

b) Kyphoscoliosis (angulation and rotation of the spine), deformation of the vertebrae and ribs may reduce ventilation.

C. Flaccidity of chest wall

Local paralysis of the external intercostal muscles and broken ribs (flail chest)

allow the enhanced intrathoracic vacuum to suck in intercostal tissues during inspiration. This paradoxical movement reduces the efficiency of inspiration

2.a. Pulmonary Compliance (4):

Compliance refers to how much effort is required to stretch the lungs and chest wall, thus high compliance means that they expand easily at any trans pulmonary pressure; low compliance means that they oppose expansion and harder to inflate or lungs would need a greater-than-average change in intrapleural pressure to change the volume of the lungs. By comparison, a thin balloon that is easy to inflate is said to have high compliance, and a heavy and rigid balloon that takes a lot of effort to inflate has low compliance.

Pulmonary compliance is calculated using the following equation, where ΔV is the change in volume, and ΔP is the change in pleural pressure: C=V/P

For example, if a patient inhales 500 mL of air from a spirometer with an intrapleural pressure before inspiration of – 4 cm H_2O and &6 cm H_2O at the end of inspiration. Then: C=0.5 L / - 4 - (-6) = 0.25 L / cm H_2O. Since compliance is determined by V/P, the P-V loop curve provides useful information on the characteristics of a patient's compliance. There are 2 different curves according to different phases of respiration- °Inspiratory compliance curve and °Expiratory compliance curve. The compliance curves of the lungs demonstrate hysteresis that is; the compliance is different on inspiration and expiration for identical volumes (Fig-3 and Fig-4). Compliance is highest at moderate lung volumes and much lower at volumes which are very low or very high. The total work of breathing of the cycle is the area contained in the loop.

Causes of low compliance of lung:

*Fibrosis of lungs (A stiff lung parenchyma i.e. pulmonary fibrosis gives rise to restrictive lung diseases decrease lung compliance, and thus require more effort (work) to overcome increased elastic recoil. Patients compensate by making rapid but shallow inspirations)

*Severe restrictive disorders
*Reduced Surfactant
*Hydrothorax and Pneumothorax
*High standing of a diaphragm
*Supine posture

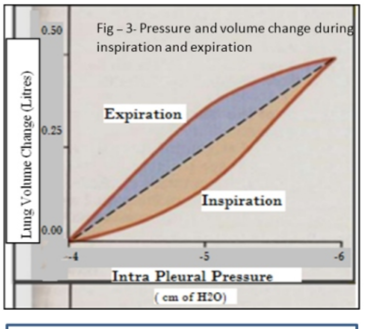
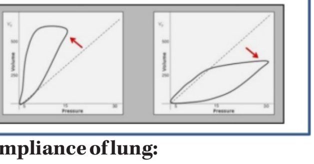


Fig – 3- Pressure and volume change during inspiration and expiration

Fig 4-A slope is drawn from the start point dividing the expiratory and inspiratory limbs. A shift of the slope towards the pressure axis signifies a decrease in compliance whereas shift towards the volume axis indicates an increase in compliance.



Causes of high compliance of lung:

*Emphysema, the elastic tissue is damaged by enzymes results in poor elastic recoil. These enzymes are secreted by leukocytes in response to a variety of inhaled irritants, such as cigarette smoke. They have extreme difficulty exhaling air. In this condition extra work is required to get air out of the lungs.
*Increasing age

Compliance is related to two principal factors:

Elasticity or elastic recoil forces (More elastic → less compliant or stiff) contribute 1/3rd of compliance this phenomenon occurs because of the elastic fibers in the connective tissue of the lungs.

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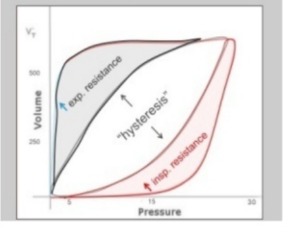
Surface tension (6):
It is the attractive force exerted by like molecules of the fluid lining alveoli causing them to collapse to their smallest size to resist the force that tends to increase the area (Greater surface tension → less compliant, P=2T/R; T = surface tension (If surface tension increases, the pressure to inflate the bubble increases); P- distending pressure, R = radius (If the radius decreases, the distending pressure to open the alveoli increases). contribute to 2/3rd of the compliance pulmonary surfactant increases compliance by decreasing the surface tension of water, covering internal surface of alveoli. presence of surfactant (alveoli are lined with a surface-tension lowering agent-surfactant - chemically Dipalmitoyl lecithin Phosphatidyl glycine produced by alveolar type II cells.) in this fluid breaks up the surface tension of water, making it less likely that the alveoli can collapse inward. If the alveoli were to collapse, a great force would be required to open it

Disorders altering or destroying surfactant causing decrease compliance
*Prematurity
*Respiratory distress syndrome (RDS)
*Oxygen toxicity
*Acidosis
*Hypoxia
*Atelectasis
*Pulmonary vascular congestion
*Pulmonary edema
*Pulmonary embolism
*Pneumonia
*Excessive pulmonary lavage or hydration
*Drowning

2.b. Respiratory Resistance (4,7):
Airway resistance is present due to opposition to flow caused by the forces of friction between the air passage and moving column of air. It occurs when the system is in motion and air is moving (Fig -5).

Fig -5- As airway resistance increases, the volume-pressure loop will become wider. An increase in expiratory resistance is more commonly seen.



i) Tissue Resistance (20%, mainly due to movement of pleural layers between chest wall and lungs during inspiration and expiration); Causes that increase tissue resistance: Obesity, Fibrosis, Abdominal distention.

ii) Airway Resistance (80% of total resistance)
Major airway resistance is contributed by upper airway and only 10% is in small airways (peripheral airway < 2 mm; less due to more cross section, less length slower & laminar flow)

It is also defined as the ratio of driving pressure to the rate of air flow. So-

Airway resistance (R_{aw})= ΔP / $Flow_{air}$ = Atmospheric pressure - Alveolar pressure / $Flow_{air}$
= - (-1) cm of H_2O / 500ml /sec

Normal airway resistance = 2 cm of H_2O per L per sec

Factors that control airway resistance:
during laminar flow may be calculated via a rearrangement of

Poiseuille's Law:
R=8 l η / πr4, where length of respiratory tract=l, gas viscosity= and radius of the different segments of respiratory tract tubes=r

The most important variable here is the radius, which, by virtue of its elevation to the fourth power, has a tremendous impact on the resistance. Thus, if the diameter of a tube is doubled, resistance will drop by a factor of sixteen.

*While a single small airway provides more resistance than a single large airway, resistance to air flow depends on the number of parallel pathways present. For this reason, the large and particularly the medium-sized airways actually provide greater resistance to flow than do the more numerous small airways.

*Airway resistance decreases as lung volume increases (during inspiration) because the airways distend as the lungs inflate, and wider airways have lower resistance.

*Airway diameter is also regulated by the degree of contraction or relaxation of smooth muscle in the walls of the airways. Signals from the sympathetic division of the autonomic nervous system cause relaxation of this smooth muscle, which results in bronchodilation and decreased resistance.

*Any condition that narrows or obstructs the airways increases resistance, so that more pressure is required to maintain the same airflow. The hallmark of asthma or chronic obstructive pulmonary disease (COPD) is increased airway resistance due to obstruction or collapse of airways.

Causes of alteration in airway Resistance
*Increase tissue resistance-
Pulmonary edema
Pulmonary fibrosis
Pneumonia
*Increase airway resistance-
Airway collapse & edema
Bronchospasm (Asthma)
Excessive secretion (COPD- Bronchitis, Bronchiectasis)
Mechanical obstruction (Tumors, Endotracheal intubation, Foreign body)
Infection (bronchiolitis, epiglossitis)
Due to increase in airway resistance, there is increased residual volume and functional residual capacity due to air trap giving rise to dyspnoea and decreased respiratory rate.
*Decrease airway resistance-
Bronchodilators
Anti-inflammatory agents
*Work of breathing is highly increased in case of low compliance and Increased airway resistance

1) Tests for assessing Ventilation function
A) Simple Bed Side PFTs
B) Spirometry

A) Simple Bed Side PFTs (8,9)
a. Respiratory Rate - Essential yet frequently undervalued component of PFT - Imp evaluator of presence of acute respiratory problem, in weaning & extubation protocols (Increase RR - muscle fatigue - workload - weaning fails).

b. Peak Expiratory Flow Rate
Patient self-monitoring with a peak flow meter or similar device is now an accepted part of the day-to-day management of asthma. Peak expiratory flow rate (PEFR) is a person's maximum speed of expiration, as measured with a peak flow meter, a small, hand-held device used to monitor a person's ability to breathe out air. It measures the airflow through the bronchi and thus the degree of obstruction in the airways.

The peak flow rate in normal adults varies depending on age and height. Normal : 450 -700 l/min in males and 300 - 500 l/min in females.

Clinical significance – values of <200 l/min means impaired function

c.Sabrasez Breath Holding test: Ask the patient to take a full but not too deep breath & hold it as long as possible.
5 - 10 sec - 1500 ml VC
10 - 15 sec - 2000 ml VC
15 - 20 sec - 2500 ml VC
20 – 25 sec - 3000 ml VC
25- 30 sec - 3500 ml VC
° >25 sec - normal Cardio-Pulmonary Reserve (CPR)
° 15-25 sec - limited CPR
° <15 sec - very poor CPR

d. Single breath count: After deep breath, hold it and start counting till the next breath. Normally 30 - 40 counts indicate normal vital capacity.

e. Schneider's match blowing test: It measures Maximum Breathing Capacity. Ask to blow a match stick from a distance of 15 cm with mouth wide open, chin supported, no head movement, no air movement in the room and mouth & match at the same level. The inferences are as follows:

Not able to blow out a match – MBC < 60 L/min – FEV_1 < 1.6L
Able to blow out a match – MBC > 60 L/min – FEV_1 > 1.6L

f. Modified Match Test: With the same above method but at different distances, the inferences derive out: Distance 9"> MBC-150 L/min; 6"> 60 L/min; 3"> 40 L/min

g. Forced Expiratory Time (FET): After deep breath, exhale maximally and forcefully after keeping stethoscope over trachea & listen.

Normal FET– 3 – 5 sec; Obstructive lung disease > 6 sec; Restrictive lung disease < 3 sec

SPIROMETRY (10,11):
Spirometric evaluation of respiratory system is the cornerstone of all PFTs

John Hutchinson invented spirometer in 1846
Spirometry is a medical test that that is designed to measure changes in volume and can only measure lung volume compartments that exchange gas with the atmosphere. But it cannot measure - Functional Residual Capacity, Residual Volume, and Total Lung Capacity. Spirometers with electronic signal outputs (pneumotachs) also measure flow (volume per unit of time). Thus there are following types of spirometer.

º Computerized Spirometer
º Portable Digital Spirometer
º Benedicts Roth Spirometer

Prediction of normal values
To interpret ventilatory function tests in any individual, the results should be compared with reference values of normal subjects matched for gender, age, height and ethnic origin and using similar test protocols; and carefully calibrated and validated instruments. Normal predicted values for ventilatory function generally vary as follows:

Age: As a person ages, the natural elasticity of the lungs decreases after the age of 20-25 years. This translates into smaller and smaller lung volumes and capacities as we age.

Gender: Usually the lung volumes and capacities of males are larger than the lung volumes and capacities of females. Even when males and females are matched for height and weight, males have larger lungs than females.

Body Height & Size: A small man will have a smaller PFT result than a man of the same age who is much larger. Sometimes as people age they begin to increase their body mass by increasing their body fat to lean body mass ratio. If they become too obese, the abdominal mass prevents the diaphragm from descending as far as it could and the PFT results will demonstrate a smaller measured PFT outcome then expected

Race: Race affects PFT values. Caucasians have the largest FEV_1 and FVC of the various ethnic groups. Chinese have been found to have an FVC about 20% lower and Indians about 10% lower than matched Caucasians Other factors such as environmental factors and altitude may have an affect on PFT results but the degree of effect on PFT is not clearly understood at this time

Spirometric Parameters:
1. Static lung volumes & capacities (Time fraction is not considered)
2. Dynamic spirometric parameters (Time fraction is considered)

Static lung Volumes & Capacities (12, 13, 14) (Fig-6,7)
- Lung Volumes-Tidal Volume, Inspiratory Reserve Volume, Expiratory Reserve vol
- Lung Capacities - Inspiratory Capacity, Vital capacity, (Addition of 2 or more volumes comprise a capacity).

Fig – 6 – Spirogram, also showing RV, FRC and TLC

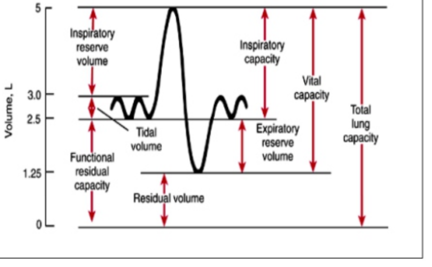
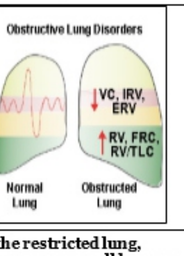
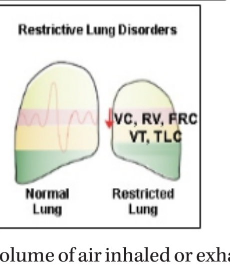


Fig 7- Changes in Static lung volumes and capacity in obstructive and obstructive lung diseases

In obstructive lung disease RV FRC are increased as respiration ends prematurely



In the restricted lung, volumes are small because inspiration is limited due to reduced compliance.



Tidal Volume (TV): volume of air inhaled or exhaled with each breath during quiet breathing (6 – 8 ml/kg) - 500 ml

Inspiratory Reserve Volume (IRV): maximum volume of air inhaled from the end inspiratory tidal position - 3000 ml

Expiratory Reserve Volume (ERV): maximum volume of air that can be exhaled from resting end-expiratory tidal Position - 1500 ml

Vital Capacity (VC): maximum volume of air exhaled from maximal inspiratory level- 5000ml (60-70 ml/kg)

Inspiratory Capacity (IC): Sum of IRV and TV or the maximum volume of air that can be inhaled from the end-expiratory tidal position. (2400-3800ml).

Dynamic Spirometric Parameters (15)
These tests are for assessing Pulmonary Mechanics i.e. ability of the lungs to move large volume of air through the airways that is time dependent

Instructions for spirometry:
Prior to testing, the patient's condition should be stable (ideally six weeks since the last exacerbation of the respiratory condition.

Standing is not mandatory but may provide better results. Sitting is safer for the elderly and infirm; if sitting, then the patient should sit straight up, with their head slightly extended.

Ask the patient to breathe in maximally, grip the mouthpiece between the teeth and then apply the lips for an airtight seal. Breathe out as hard and as fast as possible maintaining for 6 sec at least. Then patient should aim for maximal flow at the moment expiration starts. Keep breathing out until the lungs cannot be emptied any longer.

Limit the total number of attempts (practice and recording) to eight.

Three satisfactory blows should be performed and best values taken for interpretation. Criteria for satisfactory blows are: The blow should continue until a volume plateau is reached - this may take more than 12 seconds in severe COPD.

The expiratory volume-time graph should be smooth and free from irregularities.

Reversibility testing
An increase of >400 ml from baseline in FEV1 is suggestive of asthma. Smaller increases are less discriminatory, so perform baseline spirometry first and then

bronchodilator reversibility testing should be done. Before undertaking bronchodilator testing, the patient should stop short-acting beta 2 agonists for 6 hours, long-acting bronchodilators for 12 hours and Theophyllines for 24 hours. Administer bronchodilator (at least 400 micrograms Salbutamol) and repeat spirometry after 15 minutes.

A steroid reversibility testing can also be undertaken by a steroid trial (30 mg prednisolone daily for 2 weeks or 200 micrograms beclomethasone or equivalent inhaled corticosteroid for 6-8 weeks).

Dynamic Spirometric Parameters are:
1) FVC (Forced Vital Capacity)
2) FEV_1 (Forced Expiratory Volume in first second, Timed Vital Capacity) &
3) FEV_1/FVC ratio
4) MMEF or $FEF_{25-75\%}$ (Maximum Mid Expiratory Flow Rate, Forced mid Expiratory Flow)
5) MVV (Maximum Voluntary Ventilation) or Maximum Breathing Capacity (MBC)

1. Forced Vital Capacity (10, 16, 17,)
*The most important spirometric test is the FVC.
*To measure FVC, the patient inhales maximally, then exhales as rapidly, forcibly and as completely as possible (At TLC is on full inspiration followed by complete expiration to RV).

*Normal lungs commonly can empty more than 80 percent of their volume in six seconds or less.
*Indirectly reflects flow resistance property of airways and clearly distinguish between Obstructive and Restrictive Lung Disease

Causes of Obstructive Lung Diseases
° Bronchial Asthma (Bronchospasm)
° COPD (chronic bronchitis-increased secretions, emphysema-decreased elastic recoil of lungs)
° Extensive bronchiectasis
° Lung tumours

In an obstructive lung disease, there is airflow limitation due to airway obstruction may be caused by Airway inflammation, Airway fibrosis, luminal plugs and Increased airway resistance of large and small airways and on rapid breathing, greater pressure is needed to overcome the resistance to flow, and the volume of each breath gets smaller.

Asthma
Asthma is a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.

COPD
COPD is a common preventable and treatable disease, characterized by persistent airflow limitation that is usually progressive and associated with enhanced chronic inflammatory responses in the airways and the lungs to noxious particles or gases. Exacerbations and co morbidities contribute to the overall severity in individual patients.

Causes of Restrictive Lung Disease
Demonstrate reduced lung Volumes, the compliance (distensibility of lungs) of the lung is reduced, which increases the stiffness of the lung and limits expansion. In these cases, a greater pressure (P) than normal is required to give the same increase in volume (V).

Examples:
°Sarcoidosis, °Pneumoconiosis, °Interstitial lung disease(Fibrosis of lung tissue or thickening of respiratory membrane)
°Kyphoscoliosis , °Scoliosis, Obesity (Mechanical Obstruction to lungs)
°Atelectasis (Partial or total alveolar collapse)
°Pleural Effusion (Increased fluid in pleural spaces)
°Pneumonia, °Pulmonary edema, °Alveolitis (Consolidation of parts of lung parenchyma, alveolar /noninflammatory transduates/ inflammatory exudates)
°Lung Resection (lung tissue loss)
°Neuromuscular diseases- Myasthenia gravis, muscular dystrophies (Reduced generation of respiratory effort)

Assessment by FVC-
Mild to moderate reduction occurs in obstructive lung diseases and moderate to severe in restrictive lung diseases
>80% Normal (> 4 L ; Nr: 5 L)
70%-79% Mild reduction
50%-69% Moderate reduction
<50% Severe reduction

2. FEV(Forced Expiratory Volume in 1st second/ Timed Vital Capacity) (18, 19, 20, 21, 22)
Measurements obtained from the FVC Curve. This is the Forced Expiratory Volume in first second (FEV_1) of the FVC maneuver.
Normal is 3 - 4.5 L in first 1 sec.

Assesemt:

- FEV1 is **much decreased in obstructive** but in restrictive lung disorders it may be normal or higher.

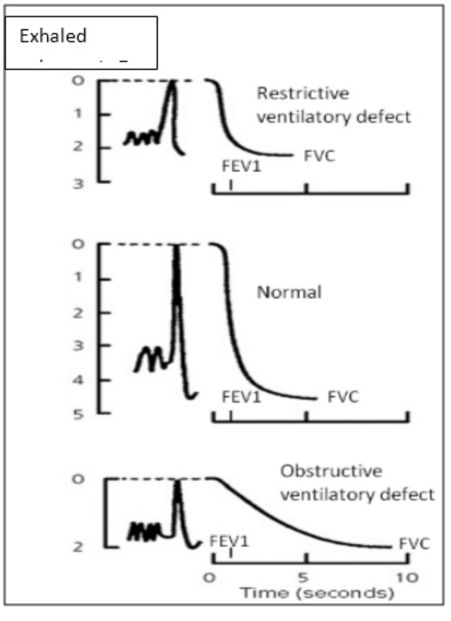
- Measures the general severity of the airway obstruction (Staging) by FEV1 as a percentage of predicted-

Stage 1 - mild: 80% or above (symptoms should be present to diagnose COPD in people with mild airflow obstruction).
Stage 2 - moderate: 50-79%.
Stage 3 - severe: 30-49%.
Stage 4 - very severe: below 30% (or FEV1 less than 50% but with respiratory failure).

3. FEV_1/FVC Ratio Percentage (Fig 8)

FEV_1/FVC ratio will be much lower than normal in obstructive disorders (FVC is smaller but FEV_1 is much smaller than normal), for example 40% as opposed to 80% due to the increase in airway resistance, quick expiration is difficult.

Fig – 8- Status of curves of FVC and FEV1 in restrictive, normal and obstructive lung diseases



In the restricted lung, the FEV_1/FVC ratio can be normal or higher than normal (the FVC is smaller than normal, but the FEV_1 is relatively large), for example 90% as opposed to 80%. This is because it is easy for a person with a restricted lung to breathe out quickly, because of the high elastic recoil of the stiff lungs.

Interpretation of % predicted:

>70% Normal
60%-70% Mild obstruction
50-59% Moderate obstruction
<49% Severe obstruction

5-Step Approach to Spirometry Interpretation -

Step 1: Begin by looking at the forced vital capacity (FVC) to determine if it's within a normal range.
Step 2: Next, look at the forced expiratory volume in one second (FEV1) to see if it's within normal limits.
Step 3: If the FVC and the FEV1 are both normal, stop at this step — the spirometry test is normal.
Step 4: If the FVC and/or the FEV1 are decreased, there is a strong possibility of lung disease.
Step 5: If Step 4 suggests the presence of lung disease, look closely at the % predicted for FEV1/FVC. If % predicted for FEV1/FVC is 70% or less (less than 0.70 according to the Global Initiative for Obstructive Lung Disease), an obstructive lung disease is highly likely. A value of 85% or greater is suggestive of restrictive lung disease.

A normal flow-volume loop (23, 24) (Fig-9)

A normal Flow-Volume loop begins on the X-axis (Volume axis): at the start of the test, person is ready with the maximum inspiration (TLC) but flow is equal to zero. After the starting point the curve rapidly mounts to a peak: Peak (Expiratory) Flow.

After the PEF the curve descends (the flow decreases) as more air is expired. A normal, F/V loop will descend in a straight or a convex line from top (PEF) to bottom (FVC).

The forced inspiration that follows the forced expiration has roughly the same morphology, but the PIF (Peak Inspiratory Flow) is not as distinct as PEF.

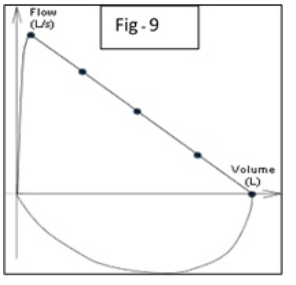
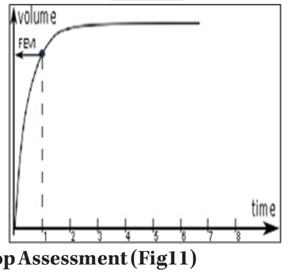


Fig - 9

A normal volume-time curve (Fig - 10)

Another way of demonstrating the spirometry test is by the volume-time graph. The start is at coordinates 0-0 (at time 0, flow is 0). Since most air is expired at the beginning, when the patient empties his large airways, the graph rapidly rises. About 80% of total volume is expired in the first second. As the lungs are emptied the rise in expired volume gets lesser and to end in a horizontal level.

Fig - 10



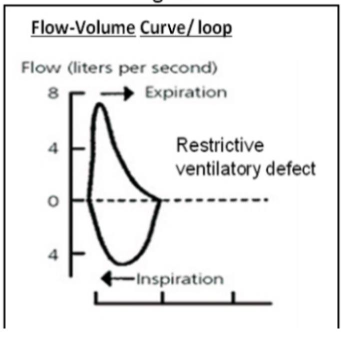
Flow volume loop Assessment (Fig11)

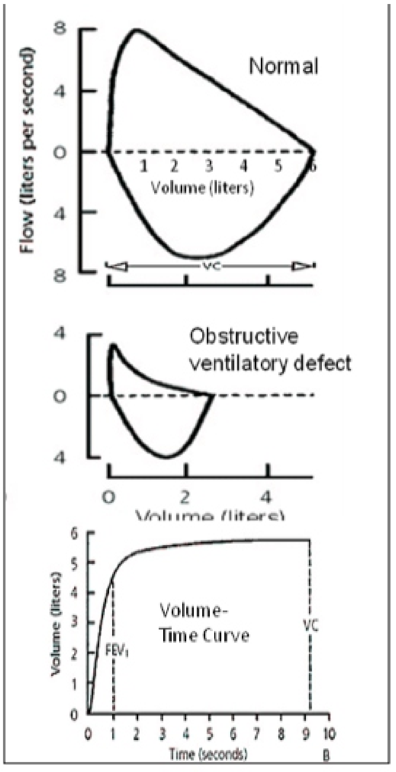
Normal - on exhalation there is a rapid rise to the maximal expiratory flow, followed by a steady uniform decline until exhalation is complete.

Obstructive lung diseases - usually the curve is a smooth concave shape as airway obstruction is relatively constant throughout expiration and may present before the change in FEV1 or FEV1/FVC. COPD - classically the curve is angled or 'kinked' as COPD lungs collapse with forced expiration.

Restrictive disease - the curve is typically a normal height but with a very steep gradient as the lung volume is diminished.

Fig - 11





4. Maximum Mid Expiratory Flow or Forced Expiratory Flow $_{25-75\%}$ (MMEF, FEF$_{25-75\%}$) (Fig 12)

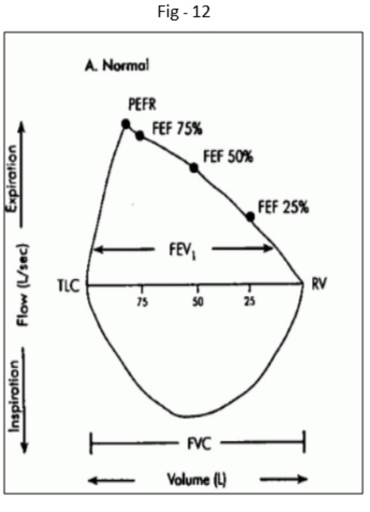
The MMEF is the average expiratory flow over the middle half of the FVC maneuver.

The MMEF is in use as an index of airway obstruction, every disorder that affects the FVC will also influence the MMEF.

It follows that the index is highly dependent on the validity of the FVC measurement and the level of expiratory effort. Normal Value is 4.5-5 l/sec or 300 l/min

Interpretation of % predicted:
>60% Normal
40%-60% mild obstruction
<40% Moderate to severe obstruction

Fig - 12



Max. Voluntary Ventilation (MVV) or Max Breathing Capacity (MBC, Fig 13) (25)

Maximum volume of air that can be breathed in and out of the lungs in 1 minute by maximum voluntary effort. The subject is asked to breathe as quickly and as deeply as possible for 12 secs and the measured volume is extrapolated to 1min (Periods longer than 15 seconds should not be allowed because prolonged hyperventilation leads to fainting due to excessive lowering of arterial pCO2 and H^+).
Normal : 150 - 175 L/min

Assessment

MVV measures speed and efficiency of filling & emptying of the lungs during increased respiratory effort and thus it is markedly decreased in patients with airway obstruction and restrictive lung diseases especially with poor respiratory muscle strength.

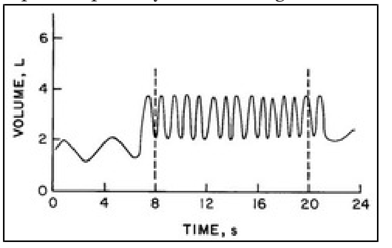


Fig 13-MVV

2)Tests for assessing Gas Exchange function (26,27, 28,29)
A. Diffusion capacity of lungs
B. Measurement of blood gases

A. Diffusion capacity of lungs

Diffusion is the process by which oxygen and carbon dioxide gases are exchanged at the air–blood interface i.e. alveolar–capillary membrane. In the normal healthy adult, oxygen and carbon dioxide pass across the alveolar–capillary membrane without difficulty owing to its thinness and large surface area and differences in gas concentrations in the alveoli and capillaries.

The measurement of diffusion capacity of the lung for carbon mono oxide (DLCO also known as transfer factor) gives important information regarding the diffusion of gases across the alveolar membrane in relation to surface area, integrity of the alveolar membrane and the pulmonary vascular bed. DLCO generally measures the overall gas-exchange function of the lung.

It is measured by a single breath technique where 10% helium and 0.3% carbon monoxide are rapidly inspired, held for 10 seconds and then expired with the measurement of the remaining carbon monoxide. Thus determination of the partial pressure difference between inspired and expired carbon monoxide with the corrected factors i.e. atmospheric pressure (altitude), hemoglobin concentration, age, sex etc. is done as recommended by the American Thoracic Society. The normal value of DLCO is approximately 20-30 ml/min/mm Hg. It depends on strong affinity and binding capacity of hemoglobin for carbon monoxide.

Causes of alteration in DLCO:

Decrease- D_{LCO} is decreased in any condition which affects the valuable alveolar surface area, and capillary bed:
*Disorder of the alveolar wall. e.g. fibrosis, alveolitis, vasculitis
*Decrease of total lung area, e.g. restrictive lung disease
*COPD (bronchitis, emphysema)- decreased alveolar surface area and damage to capillary bed.
*Uneven spread of air in lungs, e.g. emphysema
*Pulmonary embolism
*Cardiac insufficiency
*Pulmonary hypertension
*Heart failure
*Anemia
*Amiodarone high cumulative dose

Increase
*Polycythemia
*Asthma (sometimes normal D_{LCO})
*Increased pulmonary blood volume - exercise

*Left to right intra-cardiac shunting
*Alveolar hemorrhage

B. Measurement of blood gases

Arterial blood gases sample to measure the arterial oxygen tension (PaO_2) and arterial carbon dioxide tension ($PaCO_2$) offers substantial information on gas exchange and oxygen release to the tissues. Normal arterial oxygen partial pressure (P_aO_2) is 11–13 kPa or 75–100 mmHg and arterial carbon dioxide partial pressure (P_aCO_2) is 4.7–6.0 kPa or 35–45 mmHg.

*Findings of lesser partial pressure of oxygen (Hypoxia, PaO_2) i.e.< 8 kPa and normal partial pressure of carbon dioxide ($PaCO_2$ - Normal value= 5.3 kPa) are usually due to type 1 respiratory failure and the causes may be pneumonia and pulmonary embolism.

*Findings of lesser partial pressure of oxygen (Hypoxia) i.e.< 8 kPa and higher partial pressure of carbon dioxide i.e. > 6.5 kPa (Hypercapnia) are included in type 2 respiratory failure due to ventilatory failure and the causes may be respiratory muscle weakness, COPD and advanced type 1 respiratory failure as they tire and develop ventilator failure. Such patients may entail ventilatory support in the form of non-invasive or invasive ventilation.

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