Pulmonary Function Testing for the Cardiologist

By

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This monograph will consists of two portions.

The first part will deal with underlying principles essential to the understanding of the pulmonary function test.

This will be followed in the second part by description of alteration of its results in the different diseases of Cardiovascular system.

The main function of respiration is arterialisation of venous blood perfusing the millions of the alveoli in the lung. This objective is achieved through three distinct process taking place within the lung at the same time.

The Control of breathing however is extra pulmonary being regulated by the metabolic demands.

Ventilation implying aeration of the alveoli is brought about by reduction of the intra thoracic pressure, through the active contraction of the inspiratory muscles to subatmospheric level. Perfusion of the aerated alveoli with adequate quantity of blood depends on the competency of right ventricle as a pump and patency of the pulmonary vasculature. Diffusion of gases across the normal alveoleo-capillary wall continues uninhibited until equibilirium is achieved.

Briefly thus the process of respiration involves, the transfer of oxygen enriched atmospheric air to the alveolus, which is surrounded by capillaries containing deoxygenated blood conveyed to it by the pulmonary arteries for gaseous deffusion across the alveoleo-capillary membrane.

No single maneouver can evaluate all these three components of the respiratory process. This can be done only by employing series of test which are classified in table I according to their utility. Only those tests which have any practical value will be dealt here.

Both the Lungs and chest wall are distensible indicating that when the force distending them is removed they recoil back to their respective resting volume. The volume of air necessary to distend the Lung can be measured by spirometry, which involves breathing in and out through simple gas volume recorder called spirometer. It consists of a double wall drum fitted with a bell, which in turn is attached by a pulley to a pen that writes on a second rotating drum. During expiration with the entry of air in it the drum rises, lowering the pen because of pulley, with the opposite effect during inspiration.

* **From the National Institute of Cardiovascular** Diseases (Pakistan), Karachi.

Table I. The most important "Routine and Specialized" Tests of Respiratory Function.

Screening	*Routine*	*Specialized*
Spirometry	Lung volumes	Lung compliance
Vital capacity	Vital capacity	Airways resistance
Expiratory flow rates	Functional residual capacity	Exercise studies
	Residual volume	O2 uptake
	Expiratory flow rates	Wasted ventilation
	Single-breath O2 (closing volume)	Cardiac output
	Arterial blood composition	Pulmonary arterial and
	Po2	"Wedge" pressures
	Pco2	CO2—response curves
	pH	Hypoxia response curves
	Bicarbonate	Ventilation perfusion lung scans.

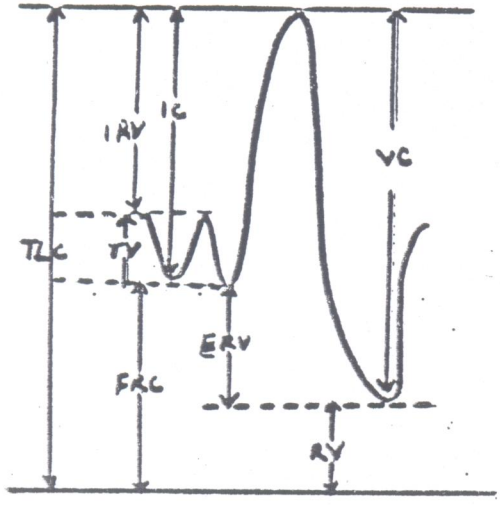

Fig. 1 Normal Lung Volumes

Vital Capacity (VC) the Volumes of Gas that can be Exhaled after Maximal Inspiration in a slow non Explosive Manner. Normal 100±25% of Predicted.

Residual Volume (RV) The Volume of Gas Remaining in the Lungs at the end of a Maximal Expiration. Normal 100±25% of Predicted

Total Lung Capacity (TLC) the Volume of Gas Contained in the Lungs at the end of a Maximal Inspiration. Normal 100±25% of Predicted.

Functional Residual Capacity (FRC) the Volume of Gas Remaining in the Lungs at Resting end Expiratory Position of the Lungs and Chest Wall. Normal 100±25% of Predicted is made of Two Component RV+ERV.

ERV (25% of VC) is the Volume of Air that can be Exhaled following Normal Expiration.

Inspiratory Capacity (75% of Vital Capacity) is the Maximal Volume of Air that can be Inhaled from FRC and is made up of two Subdivision T.V. (Tidal Volume) and Inspiratory Reserve Volume.

Inspiratory Reserve Volume is the Maximum Volume of Gas that can be Inspired from the end Inspiratory Position.

Tidal Volume (TV) the Volume of Gas Inspired or Exhaled during each Breath. Normal 100±25% of Predicted.

The various lung volumes obtained by spirometry are illustrated and defined in figure II. Due to difference in distensibility of the Lung and the chest wall, there is always a certain amount of air remaining inside the chest, even at the completion of full expiration, called the residual volume (R.V.). This and other capacities of which it constitutes a part can be measured only by specialised tests like "the Helium dilution tests" and "plethysmography". Spirometry also does not provide direct assessment of resistance to breathing (flow sensitive properties of the Lung) which can be evaluated by plotting "a volume time curve". Here the excursion of reservoir in which the patients breath, are recorded as volume change on vertical axis, against seconds on horizontal axis.

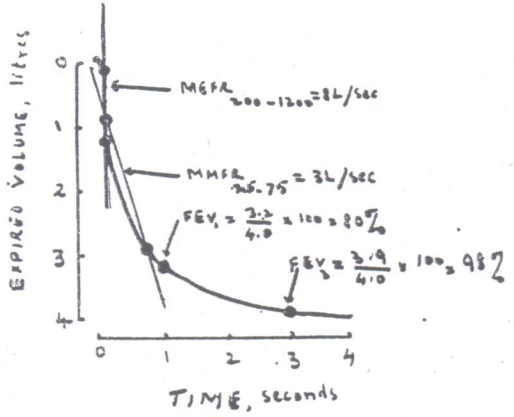

Fig. 2. Flow Rate Curve

Forced Vital Capacity (F.V.C.) is the Volume of Air Exhaled in a rapid and Explosive Manner after Maximal Inspiration FVC/VC Ratio can be taken as Magnitude of Trapping.

Following Derivative of FVC can be used to Evaluate Air way Obstruction Further.

FEV1 & FEV3 are the Volume Exhaled in one and Three Seconds Respectively also Expressed as Percentage of F.V.C. (Normal FEV1/FVC 83%, FEV3/F.V.C. 97%).

MEFR, 200-1200 ML or FEF 2-12 (Maximal Expiratory Flow Rate Indicates that the Flow Rate was Measured between Expired Volume of 200-1200 ML).

MMFR 25-75% or FEF 25-75 (Maximal MID Expiratory Flow) Rate Indicates that the Rate was Measured between Expired Volume of 25% to 75% of FVC.

MEFR & FEV1 Reflect the Airway Resistance offered by the Larger Bronchi (Affected in Bronchial Asthma) MMFR Reflects Airflow in Small Bronchi Affected in Emphysema.

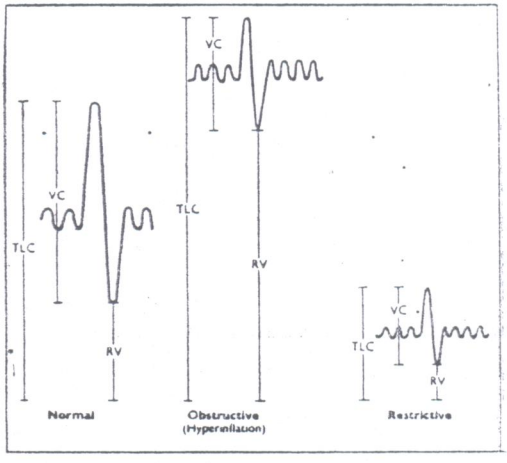

Fig. 3. Normal and Abnormal Lung Volume.

Figure III outlines such a curve and defines the various measurement which can be obtained from it.

Table II.

TEST	RESTRICTIVE	OBSTRUCTIVE
Vital Capacity	Decreased	Decreased or normal
Residual volume	Decreased	Increased
Total lung capacity	Decreased	Normal or Increased
RV/TLC	Normal	Increased
FEV/FVC	Normal or increased	Decreased
MMFR	Normal or decreased	Decreased
Single-breath O2	Normal or increased	Increased

On the basis of the results of the above tests it is possible to classify patients of ventilatory repiratory diseases into two broad groups, "obstructive" and "restrictive" as tabled in Fig. IV. In restrictive pulmonary disease all the Lung volumes are propotionately reduced where as in obstructive pulmonary disease there is selective increase in residual volume, total Lung volume and forced expiratory volume due to reduction in the rate of air flow resulting from obstruction of the airways. FEV1 can be used as a parameter, of severity by comparing the results obtained to that of the predicted values:-

Mild	20 % reduction from predicted value	
Moderate	35 %	-do-
Severe	50 %	-do-
Very severe	65 %	-do-

"FEV1" can also be used as Criteria of reversibility which is measured by the degree of improvement after the use of bronchodilator based on the difference between the prebronchodilator and post bronchodilator figures.

Slightly reversible: 25 % or less improvement, over prebronchodilator figure. Moderately responsive 55 % or less improvement over prebronchodilator. Markedly reversible 75 % improvement over prebronchodilator figures. When the diagnosis of obstructive airway disease has been made then the following two tests may be used further to locate the actual site of obstruction which may be central in asthma or peripheral in chronic bronchitis and emphysema.

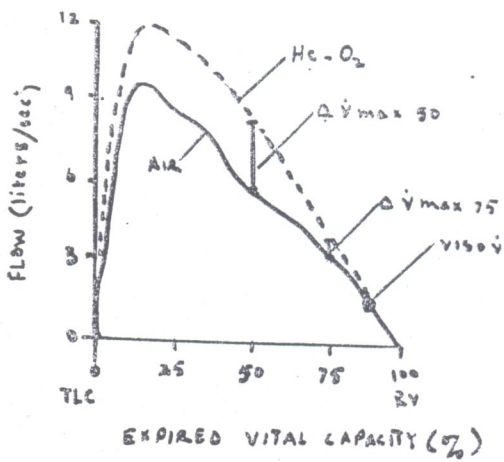

Fig. 4. Maximum Expiratory flow—Volume

Table III: Points to Remember iu Pulmonary Function Testing:

Measurements are made of Maximal Rates of Expiratory Air Flow at 25% (V MAX 25) 50% (V MAX 50) or 75% (V MAX 75) of Vital Capacity

A Maximal Expiratory Flow Volume Curve can also be Recorded after few Breaths of (HE-02) 79% Helium and 21% Oxygen.

The Flow Rate with HEO2 is Higher (Being Less Viscous and More Dense is Less Turbulent Than Air) in Trachea and Larger Bronchi—V Max 50—V Max 75.

Later in Smaller Bronchi when Slower Laminar Flow Develops the HEO2 Curve is Identical to Room Air Curve V150.

In the Presence of Narrowing to Peripheral Airways Turbulence are Less Prominent the HEO2 Curve is Closer to Room Air Curve (V MAX 50 V150) Comparison of HEO2 and Room Air Maximal Expiratory Flow Curve is the most Sensitive Test for Evaluating Small Airway Disease.

In maximum expiratory flow volume curve (Fig. V) F.V.C. is represented in a different way to flow time curve, displaying the rate of airflow against the volume of air expelled from the Lung. Here the flow rate in liters per sec. is recorded in vertical axis against volume as percentage of vital capacity in the horizontal axis.

Measurements are made of maximal rate of expiratory air flow at 25% (VMAX 25), 50% (VMAX 50) and 75% (VMAX 75) of vital capacity and reported as percentage of vital capacity.

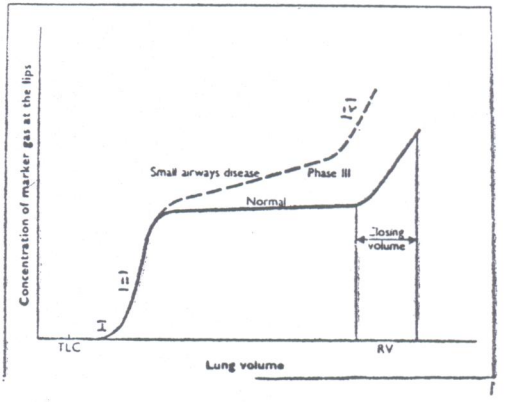

Fig. 5. The Distribution of Ventilation and Volume at which Airways at the Lung Bases begin to close can be Assessed by the Single-Breath Nitrogen Washout and Closing Volume Test.

The impeded rate of flow is also represented by alteration in shape of curve and by comparison of the rate of flow of air, with helium the site of obstruction can be established as explained in the text of the figure.

(2) The single breath nitrogen washout and closing volume test locates the site of obstruction and the even-ness of ventilation.

The underlying principle is that the first gas inhaled is the last gas exhaled, which can be identified either by 'Labelling' it with a tracer gas "zenon" or by its varying nitrogen content. The nitrogen concentration of the exhaled air is measured at the mouth and plotted against expired lung volume from total lung capacity to residual volume, following a single full inspiration of 100% of oxygen. The curve so obtained as illustrated in Fig.VI can be demarcated into four phases depending on the concentration of nitrogen.

PHASE I is the air from the dead space containing no nitrogen, as all of it is replaced by non-nitrogenous air inhaled prior to expiration.

PHASE II is the portion of the curve, where the nitrogen concentration keeps on rising sharply, as it is now being derived in increasing proporation from alveolei containing normal air, (having oxygen and nitrogen in the usual concentration) unlike the nitrogen free air exhaled from the dead space in Phase I.

PHASE III is the plateau containing air of uniform nitrogen concentation as all of its is expired from the alveolei of the lower lobe where there has been homogenous distribution of inahled air. If however the ventilation is uneven the plateu will not be a straight line but slanting upwards as a portion of the expired air though derived from the lower lobe will be containing higher nitrogen concentration because they have not all been ventilated by 100 % oxygenated air.

PHASE IV represents the air exhaled from the upper lobe, being originally derived from the dead space prior to inhalation of air containing 100 % oxygen. It contains highest concentration of nitrogen. The volume at which this increase in slope (begining of Phase IV) occurs is referred as the closing volume because the pleural pressure at this point is

Table V. **Differential Diagnosis of Anoxia**

	02	CO2	*PH*
NORMAL	*Normal* 95 *mmHg* (*Range* 75-100 *mg*)	40 *mg* (36-44 *mg*)	7.4 (7.35-7.45)
LOW ATMOSPHERIC O2	↓	↓	
NEUROMUSCULAR DISEASE	↓	↑ (Hypo Ventilation)	(No change)
PULMONARY DISEASE OBSTRUCTIVE	↓	↑	↓
UNEVEN DISTRIBUTION	↓	(Over ventilation) ↓	
IMPAIRMENT OF DIFFUSION	↓ Normal	↓ (Over ventilation)	Normal
VENO ARTERIAL SHUNT	↓	↑ (if shunt is more than 50 %) (not completely corrected by 100 % O2 inhalation	

Note: The level of arterial PCO2 is more reliable parameter of ventilation because it is not affected by the concentration of oxygen in the inhaled air or changes in the rate of diffusion across the alveolo cappilary membrane.

Table VI: Arterial Blood Gas Analysis

	BLOOD PH		BLOOD CO2	HCO3	
	Acute	Chronic		Acute	Chronic
ACIDOSIS (RESPIRATORY)	↓	↓ or (normal compensated)	↑	Normal No Change	↑
ACIDOSIS (METABOLIC)	↓	↓ or (normal compensated)	N	↓	
ALKALOSIS (RESP)	↑	↑	CO2 (WASHED OUT)	No Change	
(METABOLIC)	↑	↑	N	↑	

DIFFERENTIAL DIAGNOSIS OF ALTERATION IN P.H.

$pH = PK + \text{Log } \frac{HCO3}{CO2}$ or $\frac{56.8 \text{ (CO2 as Bicarbonate}}{2.8 \text{ (Disolved CO2 in blood)}} = \frac{\text{Renal Regulation}}{\text{Pulmonary ,,}}$

$pH = 6.10 + \text{Log } HCO3/0.0301/$ $PCO2 = 6.1 + 1.30 = 7.40$

In Hypoventilation pH falls because of the following reaction $CO2 + H20 \rightarrow H2\ CO3 \rightarrow H^+ + CHOCO3^-$ H lowers the pH because HCO3 is a weaker base though it raises the ratio of $\frac{HCO3}{PCO2}$

In Hyperventilation there is decrease in H^+ Ion and alkalosis occurs due to reversal of the above reaction.

In chronic cases the alteration in pH may be minimal (within normal range) due to compensaory mechanism.

The compensation through alteration of PCO2 (by adjustment of ventilatory rate) in metabolic acidosis or Alkalosis may be unnecessary if the renal adjustment of CO3 has been sufficient. The compensatory alteration in HCO3 n Hypo or Hyperventilation (Respiratory Acidosis or Alkalosis) may not be present initialy in acute, stage.

enough to compress the volume of air present in the lower lobe and close its airways. The changes in chronic bronchitis and emphysema promote premature airways closure of the bronchi of less than 2 mm (small airways) at much larger volume than normal.

M.V.V. (Maximum Volume Ventilation) and M.V. (Minute Ventilation) are "multificated tests", useful for asessment of ventilatory reserve. They are used mainly for the assesment of the progress of disease process rather than confirmation of provisional diagnosis or for evaluation of fitness for general anaesthesia prior to surgery particularly thoracic.

For assesment of M.V.V. patient is instructed to breathe deeply (Tidal volume 50% of vital capacity) and rapidly (40-70/mt) for twelve seconds which is then multiplied by five to get the maximum minute ventilation/mt.

It is exhausting procedure and known patient of ischaemic heart should not be subjected to this test. The minute ventilation (M.V) is measured by asking the patient to breath quietly in the reservoir for 60 sec. If the volume of dead space is available than alveoler ventilation can be easily calculated from it. Like all the other parameters the normal range is (100±25%) of predicted volume but M.V.V. figures below 40 litres is taken as absolute contraindication to surgery.

Fig. VII shows, the differentiating features of the various causes of Anoxia. Thus from the analysis of Blood O2 and CO2 and pH changes the underlying causative factor of anoxia can be ascertained.

Fig. VIII deals with the mechanism of alteration in pH secondary to changes in blood gases due to respiratory cause and how it can be differentiated from that of metabolic origin.

Changes in pulmonary function test in Cardiac Diseases.

No. 1. **Myocardial Infarction.**

(1) Disturbance of diffusion.

(A) Hypoxemia — Arterial oxygen tension varies inversely with the Pulmonary artery diastolic pressure which is mediated through the following process:-.

i) Rise in pulmonary capillary pressure leads to interstitial oedema & Compression of arteries and bronchus.

ii) Intra pulmonary shunting due to left ventricular failure.

iii) Affinity of Hb for oxygen is reduced due to increased level of (2, 3-DPG).

(B) Hypocapnia and respiratory alkalosis due to hyperventilation and restlessness.

(2) Reduction of airway conduction, manifested by increase in pulmonary compliance, forced expiratory volume (Fev) and Mid expiratory flow rate and closing volume.

(a) When the left atrial pressure is under 15 mm then the increase in closing volume may be present only. It is related to the widespread closure of small dependent airways due to competition of space, between the arteries and airways in the bronchovascular sheath.

(b) At higher left arterial pressure, increase in airways resistance is secondary to interstitial, alveolar and peribronchial oedema.

(3) Alteration between ventilation and perfusion ratio. Rise in pulmonary venous pressure results in redistribution of blood from bases to apices, (Encephalisation) where the air entery is comparatively less than the bases.

II Left ventricular failure leads to a form of restrictive Lung disease with a tendency towards moderate arterial Hypoxemia

(1) Reduction of vital capacity and total lung capacity.

(2) Moderate increase in resistance to airflow.

(3) Hypoxemia underlying causes of which are same as those mentioned before in Myocardial Infarction.

(4) Hypocapnia (Low blood Co2 Level) may result from Hyperventilation due to restlessness.

III Pulmonary Hypertension.

(A) Hypoxemia due to reduction in diffusion capacity resulting from increase in capillary to alveoler distance secondary to hypertrophy of endothelial cells.

(B) Hypocapnia due to hyperventilation.

(C) Rise in residual volume and fall in Maximum voluntary ventilation (M.V.V.) may result from increase in compliance.

IV Preoperative Assesment for cases undergoing Cardiac Surgery.

Values showing reduction upto 80% of "Predicted values" is of no consequence.

Values showing below 50% of Normal (Predicted) increases the risk of complication and death.

IV Pregnancy with Cardiac Disease.

Pulmonary function test (Volume Studies) remains unaltered during pregnancy.

In cases of valvular heart disease, reduction in lung volume during the course of pregnancy indicates Cardiac failure.

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