Pulmonary Function Testing

A Special Exhibit from the Section on Diseases of the Chest of the American Medical Association*

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DEFINITION OF OBJECTIVES

Practical Pulmonary Function Testing

Planning a pulmonary function testing program:

There is as much need in the modern hospital for a lung station as there is for a heart station and one can be staffed and equipped at no greater expense than the other. A clear definition of objectives is essential. Consideration must be given to what tests of function can and cannot do and the real professional, technical, physical, and financial resources available.

The common denominator among patients referred for pulmonary function testing is that they have or are suspected of having chronic pulmonary disease. The general purpose of pulmonary function testing is to detect impairment and if present, to characterize it and estimate its severity. The specific purpose derives from the nature of the problem which the particular patient presents to his doctors or to interested third parties such as insurance companies, employers or compensation boards. A routine suitable for one purpose may not serve another although certain tests should probably be employed in every case.

There is often a lack of awareness that some of the outstanding laboratories from which emanate a large fraction of the important publications in the field are primarily research organizations which, despite a large staff, a heavy investment in equipment and a generous operating budget can actually work up only a few patients each week. In a laboratory organized to provide a clinical service, close attention must be given to the versatility of each instrument, the level of technical skill required to operate it and the amount of time involved in each test. In the long run, personnel becomes the major budgetary item and efficiency is at a premium. Often a larger investment in equipment is justified by the rapidity or facility with which individual tests can be performed.

Real resources available:

The traditional approach to the problem of pulmonary function testing is to develop a schema of the respiratory processes and then review methods available for testing each component. However desirable this may be for teaching purposes, it does not answer the practical question. Laboratory activities are circumscribed by instrumental resources and the skill and available time of the medical and technical staff. Tests of pulmonary function can be classified horizontally by purpose and vertically by degree of complexity, equipment required and extent of experience needed to perform them. It is logical, therefore, to approach the problem by reviewing the instruments which have been found generally useful and analyzing what can be accomplished with them. Then, with the particular objectives clearly outlined, it should be possible to select the equipment which most nearly fits the requirement, set up the desired methods and operate an effective lung station within the framework of the available real resources.

There is now fairly general agreement on “basic” equipment and methods. The irreducible minimum procedure which can be termed a test of pulmonary function is the determination of timed vital capacity, i.e., one second, three second and total vital capacity and total vital capacity time. Relatively simple equipment suffices and the procedure is suitable for office work as well as in the small hospital.

*Chairman, General Committee, Special Exhibit on Pulmonary Function Testing, American Medical Association.

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or clinic. Closed circuit respirometry with a well constructed low resistance two speed recording respirometer with ventilograph is certainly the next step. This versatile instrument is also practical for office work by men particularly interested in chest disease. Next in order is determination of arterial oxygen saturation and carbon dioxide content. The basic method today employs the Courmand needle, syringes sample storage and analysis with the manometric Van Slyke apparatus. Other methods are promising but have not stood the test of time.

When this procedure is available, it is highly desirable to add arterial pH determination with a modern simple direct glass electrode based pH meter. Next, determination of the lung volume and its fractions is widely accepted as the best measure of the degree of pulmonary distention present and of special value in the appraisal of pulmonary emphysema, but agreement is less general about methodology. All of the methods in current use give results which are useful clinically. Whether bronchospirometry is included in the "basic" repertoire will depend upon the specific purpose of the program and is most generally useful in pre- and post-operative evaluation of thoracic surgery patients.

There are a large number of procedures which are "on the way" from the research laboratory to the clinic. The extent to which the basic repertoire may be expanded is limitless but contemplated additions should be considered carefully in terms of their established value and the instrumental and time investment required.

This presentation is primarily confined to actual tests of pulmonary function but three important collateral matters must be considered. First, fluoroscopy is of such importance in all aspects of chronic pulmonary disease that it must be assumed a fluoroscope is available and that the doctor responsible for function testing personally fluoroscopes each patient as an integral part of the routine. Second, close integration of the clinical cardiac and respiratory laboratory program is always desirable and particularly so if advanced procedures are to be employed because there is a large overlap of methodology. Efficient and economical utilization of skilled personnel and elaborate equipment dictates adjacent physical location or even actual combination if wasteful duplication of equipment and technical skills is to be avoided. There are, of course, circumstances which may require separate establishments but in any event, cooperation and close integration are invaluable. Third, the training and experience of the doctor responsible for the testing program may dictate selection of particular methods because of personal familiarity with them and confidence in them.

**What pulmonary function tests can do:**

1. Provide the information needed for an objective appraisal of pulmonary impairment, insufficiency or disability when interpreted in relation to the other findings in the patient. Extensive involvement of the lungs seen in x-ray shadows may be associated with little or no pulmonary impairment while others with little roentgenological evidence of disease may have true pulmonary disability.

2. Useful in ruling out pulmonary disease in non-organic types of dyspnea such as psychoneurosis, neurocirculatory asthenia, etc.

3. May provide a quantitative measure of pulmonary function which can aid in the evaluation of candidates for permanent forms of collapse therapy or surgical removal of lung tissue, both so far as immediate risk and ultimate useful existence are concerned.

4. May aid in following objectively certain aspects of the course of pulmonary disease.

5. May permit evaluation of therapeutic measures, medical or surgical.

6. Aid in differentiating cardiac from pulmonary disease.

7. Aid in differentiating primary from secondary types of polycythemia.

8. Occasionally permit detection of pulmonary abnormalities in patients in whom physical examination and x-ray studies cannot disclose certain diseases of the lung.

9. May be useful in pre-employment evaluation.

10. Aid in evaluation disability claims in insurance or industrial practice.

11. Pulmonary function studies indicate the specific function of the lung that has been impaired by disease and give the physician a clearer concept of the disease process in each patient.

**What pulmonary function tests cannot do:**

1. Cannot provide etiological or anatomical diagnoses but only physiological diagnoses, estimates of functional capacity and, sometimes, information of prognostic import.

2. Cannot localize a process geographically: thus certain tests might reveal the existence of a venous-arterial shunt but in themselves fail to identify it as intracardiac or intra-pulmonic.

3. Cannot distinguish between processes...
Producing certain effects; for example, impaired diffusion across the alveolo-capillary membrane might be demonstrated but without distinguishing between alveolar edema and interstitial edema.

4. Cannot reveal pulmonary disease unless function is impaired and then only when impairment is of sufficient degree that present tests can recognize with certainty the deviation from normal values; thus slight reduction in function cannot be detected nor involvement of small areas only.

5. Localized disease produces changes only when so much space is occupied or the lesion is strategically situated so that function is affected. As with other parenchymatous organs, diffuse disease is far more likely to impair function than localized lesions.

6. Cannot supplant careful analysis of the history, physical examination, fluoroscopic and other radiological examinations, bacteriological or pathological studies.

7. No single test now known is sufficient for evaluation of all aspects of pulmonary function.

8. At present sufficient data have not been obtained upon large enough groups of healthy persons to determine with certainty what constitutes normal values for pulmonary function in men and women of all age groups and what values represent an irreducible minimum below which patients cannot live in comfort.

**Methodology**

*Resources from the instrumental point of view:*

**A. Closed circuit equipment**

1. **Vital capacity spirometer with timing device:**
   - Timed VC, total VC, total VC time.

2. **Two speed respirometer with ventilograph:**
   - The above plus: f, TV, ERV, IC and MV at rest, during exercise, and maximal voluntary effort, patterns of the respiratory tracing, O₂ consumption (during steady states only), ORR, BMR, BR and BR/MBC.

3. **Respirometer as in 2. with helium analyser added:**
   - The above plus: FRC, RV, TC and RV/TC. If circuit with blower is used, intrapulmonary mixing can be measured.

**B. Open circuit equipment:**

1. **Simplest version:** low resistance breathing valves, Douglas bag and valve, gas meter and stop watch.
   - F, TV and MV at rest, during exercise and maximal, BR, BR/MBC.

2. **Tissot spirometer** (used direct or to measure volumes transferred from bags. A kymograph is useful. More precise than gas meter.) Observations same as 1.

3. **Either 1. or 2. with provision for gas analysis, switching to 100% O₂ and collecting Haldane-Freely "alveolar air" samples.**
   - Same as 1. and 2. plus O₂ consumption, ORR, FRC, IIM. (For lung volume, ERV, IC, etc. must be determined with a closed circuit respirometer.)

**C. Van Slyke manometric apparatus:**

Analysis of arterial blood samples for O₂ saturation and CO₂ content (and many other analyses).

**D. Glass electrode blood pH meter:**

Arterial pH, (and, with CO₂ content known, arterial CO₂ tension).

**DEFINITIONS RELATING TO PULMONARY FUNCTION**

"Standardization of Definitions and Symbols in Respiratory Physiology" (Abridged from Federation Proceedings, Vol. 9, Pages 602-605, 1950)

"On April 19, 1950, a group of physiologists met in Atlantic City to discuss the possibility of establishing a systematic set of symbols for use in teaching and research publications relating to respiratory physiology. The need for such a set of symbols is great . . . ."

"The following definitions and symbols represent the fruit of these discussions and they are presented here with the hope that authors and editors will use them as a reference source to achieve uniformity and clarity in terminology in the field of respiratory physiology . . . ."
Definitions of lung volume are shown diagrammatically above. The "volume" subdivisions shown at right apply to all levels of respiratory effort and contain no overlapping volumes. The "capacity" terms at left each include two or more primary "volumes" and are specially convenient for clinical applications where gasometric measures are most simply made from the expiratory position of the resting patient.

**Usual Abbreviations and Definitions of Terms:** (The Pappenheimer committee recommendations are followed as closely as possible for clinical purposes.)

**Lung Volume:**

- **TV:** Tidal Volume: volume, BTPS, of each breath.
- **ERV:** Expiratory Reserve Volume: volume, BTPS, expelled by maximal effort beginning at end of normal expiration.
- **IC:** Inspiratory Capacity: volume, BTPS, inspired by maximal effort beginning at end of normal expiration.
- **VC:** Vital Capacity: 
  a) one stage: volume, BTPS, expelled by maximal expiratory effort beginning at end of maximal inspiratory effort.
  b) two stages: sum of IC plus ERV determined separately.
- **RV:** Residual Volume: volume, BTPS, remaining in lung at end of maximal expiration.
- **FRC:** Functional Residual Capacity: volume, BTPS, in lung at end of normal inspiration.
- **TC:** Total Lung Capacity: volume, VTPS, in lung at end of maximal inspiration.

**Essential Information**

**State of the Patient:** i.e., during an attack of asthma, etc.

**Position of the Patient:** i.e., supine, etc.

**Activity of the Patient:** i.e., resting, exercise, etc.

**Corrections Applied:** Observations are usually corrected to some standard condition:

- **STPD:** Standard conditions of temperature and pressure, dry, (0° centigrade, 760 mm. Hg. barometric pressure)
- **BTPS:** Normal body temperature, ambient pressure, saturated with water.
- **ATPS:** Ambient temperature and pressure, saturated with water.

**Relation of Observations to Normal Standards:**

"Normal" values for a particular patient are often predicted from tables, graphs or equations based upon height, weight, age, sex, etc. An observation may be expressed as a percent of the *Predicted value* (% Pred.) or as a percent Difference (% Diff.) from the predicted value.
Respiration: The gaseous exchange between an organism and its environment.

Internal Respiration: The exchange of gases between tissue cells and the fluid bathing these cells.

External Respiration: The exchange of gases between the blood and air entering the lung.

Mechanisms of External Respiration:
4. Perfusion (Circulation): The quantity and distribution of blood flow through the lungs.

Impairment: Significant deviation from normal.

Insufficiency: Failure to meet normal requirements for ordinary activity or abnormal compensation to meet normal requirements.

Disability: The individual is rendered incapable of meeting the requirements of ordinary activity.

Ventilation Studies:

f: Respiratory frequency (rate).
M: Maximal and per Minute.
MV: Minute Ventilation.

MMV = MBC: Maximal Minute Ventilation = Maximal Breathing Capacity.

BR: Breathing Reserve; (VR: Ventilatory Reserve:) MBC minus resting MV.

BR/MBC x 100: Ratio of Breathing Reserve to Maximum Breathing Capacity, per cent.


VE: Ventilatory Equivalent: liters ventilated per 100 ml. O₂ removed.
(Note that VE equals 100 divided by ORR.)

AVI: Air Velocity Index: % Pred. MBC/ % Pred. VC.

IIM: Index of Intrapulmonary Mixing: % conc. of Nitrogen in alveolar air after 7 minutes 100% oxygen breathing.

Blood Gases:

C₀₂: Arterial O₂ Content: i.e., concentration of oxygen in arterial blood.
C.CO₂: Arterial CO₂ Content: i.e., concentration of carbon dioxide in arterial blood.

S.O₂: Arterial O₂ Saturation: C.O₂ x 100
Arterial O₂ Capacity is the maximum amount of oxygen which can combine with a sample of arterial blood.

P.O₂: Partial pressure of O₂ in arterial blood.

P.CO₂: Partial pressure of CO₂ in arterial blood.

Plasma CO₂ C.P.: Plasma carbon dioxide combining power.

Art. pH: Arterial hydrogen ion concentration in pH units.

Respiratory Gases:

I: Inspired Gas: The gas mixture inspired.
E: Expired Gas: The gas mixture expired.

V.O₂: Volume of O₂ inspired.

V.CO₂: Volume of CO₂ expired.

C.O₂: Concentration of O₂ in alveolar gas.
P.CO₂: Partial pressure of CO₂ in inspired gas.
Timing of the Vital Capacity

Measuring the speed of performance of VC is as important as the measure of the volume of the total VC. It is one of the simplest methods for differentiating obstructive and restrictive ventilatory insufficiency. There are many methods for introducing the element of time into the vital capacity measurement.

"Timed Vitalometer": An electronic timer (with selection for the desired time interval) actuates a solenoid which stops the recording of the VC at a pre-determined time interval.

Timed VC Measured with a Recording Spirometer: The desired time intervals are measured on the record of the vital capacity effort.

Mid-Half VC Time: Whereas the other two methods measure the volume of the VC exhaled in a given time, the mid-half VC time measures the time required for exhaling the middle half of the vital capacity. Normally this time interval is less than 4 seconds.

Restrictive ventilatory insufficiency.

Restrictive Ventilatory Insufficiency

J. C. D.—36 year old male, oleo-pneumothorax: VC=3.776 L. (89% of predicted); 1 Sec. VC=2.108 L. (56% of total VC); 2 Sec. VC=3.425 L. (91% of total VC); 3 Sec. VC=3.645 L. (96% of total VC); MBC=155 L./Min. (117% of predicted).

Obstructive Ventilatory Insufficiency

H. L.—56 year old male, pulmonary emphysema, cor pulmonale: VC=2.664 L. (70% of predicted); 1 Sec. VC=0.755 L. (28% of total VC); 2 Sec. VC=1.155 L. (43% of total VC); 3 Sec. VC=1.421 L. (53% of total VC); MBC=29 L./Min. (31% of predicted).
CLOSED CIRCUIT RESPIROMETRY

Spirograms are recorded data with the advantage that the qualitative aspects of breathing can be studied in addition to the measure of volume. They are useful for comparative purposes in which changes can be more completely evaluated. Spirograms in normals from day to day and week to week are uniform for the individual although they do vary from person to person.

There are two commonly used spirometers for closed circuit respirometer. They differ in size (9L and 13.5L). The larger spirometer offers the advantage of larger tubing and therefore lower resistance. Recording spirometers are usually equipped with two writing pens. The "spirograph" pen is a "direct" writing pen that records both inspiration and expiration. The "ventilograph" pen records the expiratory volumes only in a fixed ratio (1.10 for the 13.5L model) to the spirograph pen. This greatly facilitates measurement of ventilation volumes. The Kymograph runs at two speeds: 0.6 mm./sec. and 2.5 mm./sec.

Vital Capacity: Recorded at 2.5 mm./sec. for accuracy of measurement. The form of the inspiratory and expiratory curves can be seen readily. Total VC time and timed VCs can be measured. Two Stage Vital Capacity: obtained by adding the separate values for IC and ERV. Air Trapping: best demonstrated by successive determinations of the VC. If the VC becomes progressively smaller, trapping is present.

Maximum Breathing Capacity: Recorded at 2.5 mm./sec. The ventilation volume is measured from the "ventilograph" tracing. The normal MBC is performed below and above the baseline for tidal volume.

The accompanying spiograms were recorded on a Collins 13.5L Spirometer.
PULMONARY FUNCTION TESTING

Closed Circuit Respirometry

I. Kymograph run at two speeds: ½ mm./sec.; 2½ mm./sec.
II. The ventilograph pen records the total of Expiratory Volumes in a fixed ratio (1:10) to the spiograph pen.
III. Spiromgrams are recorded data that can be compared with future spiromgrams and progress and changes more easily evaluated.
IV. Spiromgrams in normals from day to day and week to week are uniform for the individual though they vary from person to person.

Breathing Patterns

I. With CO₂ absorber in place, O₂ consumption can be measured from the slope of the base line.
II. Ventilation is recorded by the ventilograph.
III. Tracings may help in evaluating the patient's cooperative effort and his general breathing patterns:
   a) Hyperventilation
   b) Irregular breathing patterns
IV. Paper speed ¼ mm./sec. for O₂ consumption. Paper speed 2½ mm./sec. to study patterns.

Vital Capacity

I. Run at 2½ mm./sec. for accuracy of measurement.
II. An Expiratory VC followed by an Inspiratory VC. The two should be equal.
III. Not only total volume but total VC time and 1 sec. and 3 sec. VC can be measured and the form readily seen.
IV. In obstruction—all patients should improve after adrenalin. (Increasing total volume with improved 1 and 3 sec. VC and shortened total VC time.)

Trapping

Air trapping is best demonstrated by three successive determinations of VC. VC becomes increasingly smaller where trapping is prominent. If this is due to obstruction which can be relieved, adrenalin will change the pattern toward normal.

Inspiratory Capacity and Expiratory Reserve Volume

I. Inspiratory Capacity and Expiratory Reserve Volume show changes in obstruction of acute nature, emphysema, fibrosis, and many other states including extrapulmonary lesions such as of cervical cord, etc.
II. Two state VC obtained by adding separate values for IC and ERV is equal to the one stage VC in the normal, but larger than the one stage VC in emphysema by a significant amount, and slightly larger in obstruction.
III. If Inspiratory Capacity is low and Expiratory Reserve Volume large due to obstruction that can be relieved, adrenalin should increase Inspiratory Capacity and reduce Expiratory Reserve Volume.

Maximum Breathing Capacity

I. Volume per minute can be measured from the ventilograph tracing.
II. Rate can be measured by counting.
III. Normal MBC is performed above and below the base line of the resting breathing pattern.
IV. Obstruction and loss of elasticity cause the MBC to be performed at high level to overcome obstruction and use what elasticity is still available.
V. If obstruction is temporary as in asthma, adrenalin will relieve it, increasing the MBC and lowering the level at which it is performed.
Restrictive Ventilatory Insufficiency

Spirographic Studies Characteristically Reveal:

Loss of total lung volume with decreased vital capacity.
Absence of significant slowing in expiratory VC.
3 sec. VC = 90-100% of total VC.

MBC normal or decreased, if reduced the reduction is disproportionately less than the decrease in VC.
MBC baseline level is not elevated.

C. C.—58 year old male.
Chief Complaint—Cough, weakness and 20 pound weight loss for 5 months.

Additional Pulmonary Function Data —
RV/TLC x 100: 8%.
S,O: 94.7%.
C,CO: 82.5 vol. %.

J. C.—24 year old male.
Chief Complaint—Mild chronic cough with hemoptyses for 6 months.

Additional Pulmonary Function Data —
RV/TLC x 100: 21%.
PULMONARY GRANULOMATOSIS († Boeck's Sarcoi): A. S.—30 year old male.
Chief Complaint — Dyspnea on exertion, weakness and 20 pound weight loss for 2 years.
Additional Pulmonary Function Data — RV/TLC x 100: 26%.

Obstructive Ventilatory Insufficiency

Spirographic Studies Characteristically Reveal:
Total VC Normal or Decreased, Generally Decreased.
Prolongation of the Expiratory VC time. 3 sec. VC less than 90% of the total VC.
Two stage VC Greater than one stage VC. MBC decreased and performed at an elevated baseline level.

PULMONARY EMPHYSEMA—PULMONARY FIBROSIS: J. A.—58 year old male.
Chief Complaint — Progressive exertional dyspnea and cough for nine years.
Additional Pulmonary Function Data — RV/TLC x 100: 48%.

Chief Complaint — Chronic cough with mucopurulent sputum for four years. Progressive exertional dyspnea for one year.
Additional Pulmonary Function Data — RV/TLC x 100: 89%. S.O.: 89%. C.C.O. 52.6 vol. %.
PULMONARY EMPHYSEMA—BRONCHIAL ASTHMA

R. R.—60 year old male; Chief Complaint—Asthma for twelve years. Progressive dyspnea on exertion for eight years.

A. before therapy—Additional pulmonary function data:
RV/TLC x 100: 78%.

B. after bronchodilators, antibiotics and pneumoperitoneum—Additional pulmonary function data:
RV/TLC x 100: 61%.
S\textsubscript{O}_2: 94%.
C\textsubscript{O}_2: 46 vol. %.

Procedure

1. Rinse spiro with room air.
2. Bring bell to “zero” volume and close valve.
3. Admit 1.0-1.5 liters helium and inscribe “He. Line.”
4. Add enough O_2 for 7-10 minutes breathing.
5. Start kymograph and then connect subject.
6. Read helium meter at the time the respiratory baseline crosses the “He. Line.”
7. Record bell temperature for correction of volumes to BTPS.
8. Inspiratory capacity and expiratory reserve volume are then measured for the lung volume calculations on the same spirometer with subject in same position as for FRC, usually semi-recumbent.
Reproducibility of the Functional Residual Capacity:
(based on difference between first and second of 42 serial duplicate estimations with P = 0.05)

For all values of FRC (n = 42, o = 121 ml.):
Difference should not exceed 245 ml.
“Error” of Mean ±86 ml.

For FRC less than 5 liters (n = 29, o = 75 ml.):
Difference should not exceed 153 ml.
“Error” of Mean ±54 ml.

For FRC more than 5 liters (n = 13, o = 176 ml.):
Difference should not exceed 385 ml.
“Error” of Mean ±136 ml.

*This volume must be corrected to the observed baseline in the respiratory tracing, otherwise errors up to 600 ml. or more may result.
OPEN CIRCUIT

Functional Residual Capacity, Oxygen Dilution Method of Darling, Cournand and Richards*

Modified from: Chronic Pulmonary Emphysema. Physiopathology and Treatment (1953) by M. S. Segal, M.D. and M. J. Dulfano, M.D.

with permission of the publisher, Grune and Stratton, Inc., New York City

Procedure

1. Rinse Douglas bag completely with "100% O₂", and empty.
2. With valves A and B turned to outside air, connect patient with mouthpiece. (Fig. 1)
3. Allow tidal volume to become constant.
4. At the beginning of an inspiration turn valves A and B so that patient inspires "100% O₂" and expires into the Douglas bag. (Fig. 2)
5. Seven and one-half minutes later, at the beginning of an expiration, return valves to original position. At the end of the expiration collect the "terminal" expiratory air sample at point C.

Collect a sample of gas from the bag and measure the total volume of the bag.
7. Analyze the "terminal" expired air sample and the Douglas bag sample for O₂ and CO₂ content.

A refinement of the open circuit method of measuring the FRC is the incorporation of a monitoring spirometer with "bag in box" to monitor the point in the vital capacity at which the patient is turned into the circuit for inspiration of oxygen and expiration into the Douglas bag. In this way, any error in starting the test at the beginning of an inspiration can be corrected. The monitoring spirometer is pictured in the illustration of an actual test.
Calculations

\[
\text{FRC (dry)} = \frac{(V) (b-a) - C}{\text{Alv. } N_e - \text{Alv. } N_a}
\]

where \text{FRC (dry)} = \text{functional residual capacity (cc.)}

- \( V \) = volume (cc.) in Douglas bag after 7½ min. oxygen.
- \( b \) = per cent nitrogen in Douglas bag after 7½ min. oxygen.
- \( a \) = per cent nitrogen in oxygen tank.
- \( \text{Alv. } N_e \) = per cent nitrogen in alveolar air at beginning of procedure (average, 81.00%).
- \( \text{Alv. } N_a \) = per cent nitrogen in alveolar air at end of 7½ min. oxygen (normal, 2.50%).
- \( C \) = correction (cc.) for nitrogen excreted from body during 7½ min.
- \( \text{oxygen: } C = (\text{B. S. m}^3) (96.5) + 35. \)
- \( \text{DS} \) = dead space (70 cc.).

Example: \( \text{FRC (dry)} = \frac{(8090) (.0537-.00352)-181=4073}{.8109 - 1026} = 5710 \text{ cc.} \)

- \( \text{FRC (dry)} - \text{DS} \times 1.066 = \text{FRC (wet)} \)
- \( \text{FRC (wet)} - \text{Exp. Res. Vol.} = \text{RV} \)
- \( \text{RV} - \text{VC} = \text{TLC} \)
- \( \text{Ratio} = \frac{\text{RV}}{\text{TLC}} \times 100 \)

<table>
<thead>
<tr>
<th></th>
<th>Normal Patient</th>
<th>Emphysema Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Index IP mixing (Alv. ( N_e ))</td>
<td>1.03%</td>
<td>10.26%</td>
</tr>
<tr>
<td>FRC (wet) cc.</td>
<td>3976</td>
<td>6012</td>
</tr>
<tr>
<td>Exp. Res. Vol. cc.</td>
<td>2150</td>
<td>1595</td>
</tr>
<tr>
<td>RV cc.</td>
<td>1826</td>
<td>4417</td>
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<tr>
<td>VC cc.</td>
<td>5609</td>
<td>3199</td>
</tr>
<tr>
<td>TLC cc.</td>
<td>7435</td>
<td>7683</td>
</tr>
<tr>
<td>Pred. TCC cc.</td>
<td>6719</td>
<td>5328</td>
</tr>
</tbody>
</table>

| RV                  |               |                   |
| TLC                 | 25%           | 58%               |
ARTERIAL OXYGEN SATURATION IN PULMONARY DISEASE

Anoxemia is reduced arterial oxygen saturation. In pulmonary disease, anoxemia may result from impaired ventilation, intrapulmonary gas mixing, diffusion or perfusion occurring singly or, more commonly, in combination.

Normal

1. Lack of airway obstruction.
2. Good pulmonary elasticity
3. Normal size of alveolus.

Impaired Ventilation

1. Depressed respiratory center.
2. Impaired thoracic cage mechanics.
3. Impaired diaphragmatic function.
4. Other causes of restriction of mobility of the lung.
5. Obstruction of respiratory passages.
6. Impaired pulmonary elasticity.
7. Pulmonary distention.

Impaired Mixing

1. The factors listed under impaired ventilation also apply to impaired intrapulmonary gas mixing.
2. Emphysematous bleb formation.
3. Air cysta.
5. In chronic pulmonary disease these factors are generally progressive.
Impaired Diffusion  Impaired Perfusion (Shunting)

1. Fluid in alveolus.
2. Interstitial edema, fibrosis and inflammation.
3. Thickened pulmonary capillary.

Congenital heart disease with a “right to left shunt” of blood may produce changes essentially identical to those seen with perfusion of non-aerated pulmonary tissue.

Normal

Determination of the arterial oxygen saturation is useful in the evaluation of patients with pulmonary disease. The primary factors which govern the arterial oxygen saturation and likewise, the factors which may be evaluated by a determination of the arterial oxygen saturation are: ventilation, intrapulmonary gas mixing, diffusion, and perfusion. Measurement of the arterial oxygen saturation is useful as an overall test of the four factors listed above but will not delineate specific impairment of these functions. Combined with other studies it is helpful in the separate evaluation of the four factors. More elaborate blood gas studies are necessary for specific delineation, especially of diffusion and perfusion. Although the arterial oxygen saturation is useful in assessing the ability of the lung to oxygenate blood, it is not the most sensitive measure of this function. More elaborate blood gas studies may reveal impaired diffusion in some patients who have normal arterial oxygen saturation.
In the usual case of pulmonary emphysema with arterial oxygen unsaturation no single function is wholly responsible for the impairment of oxygenation. Usually, ventilation, intrapulmonary gas mixing, diffusion, and perfusion are all impaired and it is not possible to quantitate impairment of any one function. Determination of the arterial oxygen saturation in pulmonary emphysema is frequently used in combination with the measurement of arterial carbon dioxide content for classification of the severity of the disease. This is especially true when the measurements are made during exercise as well as at rest. In addition, serial determinations of arterial oxygen saturation in any given patient with pulmonary emphysema are helpful in determining the type of therapy and evaluating its effectiveness.
Pulmonary Fibrosis

In pulmonary fibrosis, there may be much variation in impairment of pulmonary function. Thus, the arterial oxygen saturation in patients with different types of pulmonary fibrosis is highly variable. In these cases of pulmonary fibrosis which lead to airway obstruction, pulmonary emphysema is frequently found.

The arterial oxygen saturation will be influenced not only by the basic lesion but also by the factors discussed under pulmonary emphysema. In some patients with pulmonary fibrosis, diffusion of gases across the alveolar-capillary membrane is the only significant lesion. (The alveolocapillary block syndrome.) Measurement of the arterial oxygen saturation may be most helpful here for it may be the only commonly determined measurement which is significantly impaired.

In addition to the specific classes of pulmonary fibrosis discussed above, there are cases of pulmonary fibrosis with no impairment of oxygenation of the blood. Thus, a normal arterial oxygen saturation may be helpful.
Controlled carbon dioxide elimination is as important a function of respiration as oxygenation. When carbon dioxide is released from body cells into the blood it forms \( \text{H}_2\text{CO}_3 \), an acid. This acid combines to some extent with the base of blood. Depending upon the amount of base and upon the amount of carbon dioxide, there will be more or less "free" dissolved carbon dioxide. Carbon dioxide tension (partial pressure of \( \text{CO}_2 \)) is the measure of "free" carbon dioxide, \( \text{pH} \), the measure of relative acidity or alkalinity of the blood, is dependent upon the amount of base and the amount of carbon dioxide in the blood at the moment.

Since carbon dioxide is highly diffusible, the carbon dioxide tension of arterialized blood leaving an alveolar capillary network is almost exactly the same as the carbon dioxide tension of the gas in that alveolus. This remains true even when alveolar-capillary oxygen diffusion is severely impaired because carbon dioxide is so much more highly diffusible than oxygen. Alveolar carbon dioxide tension therefore "controls"
arterial carbon dioxide tension, even in advanced pulmonary impairment. Alveolar carbon dioxide tension in turn depends upon the quantity and quality of ventilation and intrapulmonary gas mixing.

The amount of base in the blood is controlled principally by the kidney. It normally retains extra base (principally sodium) when pH shifts toward the acid side. This compensatory renal process is slow (days) compared to the pulmonary regulation of alveolar CO₂ (seconds). The pH of the blood is directly dependent upon the relative amount of CO₂ and base present. The retention or excretion of base by the kidney may be regarded as the "fundamental" factor in acid-base balance, but pulmonary regulation of alveolar CO₂ tension is the "immediate" factor since it can alter alveolar CO₂ tension almost instantaneously. From moment to moment, pulmonary regulation of arterial CO₂ tension is the major factor in maintaining blood pH. Serious consequences rapidly result if blood pH is not held virtually constant at 7.45.

Therefore it is not enough for the lungs merely to eliminate CO₂ from the body; they must do it in such a manner that blood pH is controlled within narrow limits. There are many methods to measure arterial CO₂ tension and arterial pH. For the pulmonary physiologist the easiest adequate method depends upon certain relations which have been found to exist between pH, CO₂ content and CO₂ tension. The CO₂ content of an arterial blood sample can be measured with the Van Slyke manometric apparatus and its pH measured directly with a glass electrode pH meter. With this information arterial CO₂ tension can be read from a graph.

In pulmonary function testing, arterial pH determination is of importance largely because when it is known and when arterial CO₂ content is known, arterial CO₂ tension may be derived. This latter is closely related to alveolar CO₂ tension which in turn is an excellent measure of the adequacy of ventilation and intrapulmonary gas mixing.

With normal ventilation and intrapulmonary gas mixing, the partial pressure of CO₂ in the alveoli is usually 40 mm. Hg. Normally there is no significant alveolar-arterial blood CO₂ gradient. Thus, for blood leaving any given pulmonary capillary the partial pressure of carbon dioxide in that capillary will be the same as the partial pressure of CO₂ in that alveolus. With normal alveoli and pulmonary capillaries throughout the lung, the partial pressure of carbon dioxide in arterial blood is 40 mm. Hg. With a normal blood base, the pH will be normal.

Inadequate ventilation for any reason results in an increase in the partial pressure of CO₂ in the alveoli. Since the arterial blood PCO₂ is in equilibrium with the alveolar PCO₂, there will be an increase in arterial PCO₂. Acutely this CO₂ retention causes lowering of the pH, and is known as respiratory acidosis. If CO₂ retention persists renal retention of base raises pH to normal compensating for the high CO₂ tension. This condition is then compensated respiratory acidosis.
Emotion, drugs, central nervous system disorders, high altitude and other factors may cause hyperventilation. With hyperventilation excessive CO₂ is "blown off" and the alveolar PÇO₂ is lowered. Acutely, this results in a decreased arterial PÇO₂ and increased pH. This situation is known as respiratory alkalosis. With prolonged hyperventilation renal loss of base raises the pH and leads to a compensated respiratory alkalosis. Prolonged hyperventilation rarely occurs as a result of pulmonary disease, but is of importance in understanding pulmonary function in certain situations (i.e., respirator patients).

In chronic pulmonary disease the usual abnormality of arterial CO₂ and pH is that which accompanies hypoventilation. Thus, there is usually a respiratory acidosis which may or may not be compensated. In addition by the time CO₂ retention and acidosis occur, anoxia will also be present. In these patients, anoxia is the main stimulus to ventilation because the respiratory center has become insensitive to CO₂. Oxygen therapy may abolish the anoxia and further depress respiration. Further retention of CO₂ may occur rapidly and lead to CO₂ narcosis and even death if steps are not taken to insure adequate ventilation.
BRONCHOSPIROMETRY

Indications:
For collapse therapy, surgery, following unilateral progressive disease, etc., where is it necessary to know how much each lung is contributing.

Contraindications:
1. Ulcerative tracheobronchitis.
2. Recent hemoptysis in tuberculosis (within two weeks).
3. Tracheobronchial deformities.
4. Stricture of the left main stem bronchus makes it necessary to put the catheter in the right main stem bronchus which alters results because resistance is greater on the left.
5. In bronchiectasis daily sputum must be well controlled before attempting procedure.
6. Dyspneic patients may have difficulty.
7. Uncontrolled cough and secretions.
Procedures Done

1. Differential O₂ uptakes
2. Differential vital capacities
3. Differential resting ventilation
4. Differential Functional Residual capacity
5. Differential intrapulmonary mixing
6. Differential studies with exercise

Precautions during bronchospirometry assuring reliable results:

1. Adequate topical anesthesia to trachea and bronchi.
2. If cough or uncontrollable secretions occur, procedure should be abandoned and repeated another day.
3. Catheter should be placed at least 2 cm. and not over 4 cm. into the left main stem bronchus.
4. A tracing should be made with catheter in place and observations of position made before and after. This serves as a good control for later tracings, and to insure catheter being well placed.
5. Some catheters develop leaks from one main lumen to the other.
FLUOROSCOPIC ESTIMATE OF PULMONARY FUNCTION

Fluoroscopy is a supplement to volume measurements and allows one to see time factors. It does not tell how much air is breathed, but it makes visible:

1. The SPEED with which air can be drawn in and forced out.
2. The MECHANICS of breathing:
3. AERATION — relative lucency during inspiration and expiration in:
   a. Apex; b. Base.
4. Abnormal MOTILITY or FIXATION:
5. Differences between the lungs and segments of a lung with respect to the above.
6. Evidence of the nature of CYSTS:
   a. Freely communicating; b. Ball-valve; c. Closed.

The Diaphragm

Functionally there are two diaphragms, right and left.

1. Note POSITION and CONTOUR.
2. Note MOTION — degree and equality on quiet breathing, deep breathing and sniff.
   a. Are descent and ascent similar in duration, and equal right and left? b. Is direction of motion normal or paradoxical? c. Is quiet expiration normal and deep expiration prolonged — unilateral or bilateral? d. One portion of one leaf of diaphragm may be restricted, fixed, or paradoxical, and may give rise to a “hinge” type of motion. 1. Examine in A-P and oblique.
3. Does suprapubic fist pressure alter the excursion of the diaphragm?
4. Look for DELAYED AND PROLONGED ASCENT of one or both diaphragms (EXPIRATORY TRAPPING)
   a. On deep breathing? b. On rapid, forced breathing?
The Midline Structures

1. Open screen to full vertical aperture and narrow horizontally to OBSERVE DURING QUIET AND DEEP BREATHING:
   a. Heart
   b. Mediastinum
   c. Hila
   d. Trachea
2. ON DEEP INSPIRATION, heart, trachea and hila normally elongate and remain in midline position.
   a. Inferior hilar markings normally elongate more than superior ones.
3. ON FORCED EXPIRATION should see:
   a. Midline structures return to normal
   b. Freely compressible hilar marking
   c. Heart may normally rotate, according to habitus
4. SHIFT of midline structures is due to INEQUALITY OF INTRATHORACIC PRESSURES.
5. If structures shift to one side:
   a. Note DIRECTION and AMOUNT of shift, and PHASE OF BREATHING during which it occurs.
   b. Note if structures return to midline or swing to opposite side during opposite phase of breathing.
   c. Note in SEQUENCE the following in relation to the shift to help explain its cause:
      1. Diaphragmatic motion
      2. Intercostal motion
      3. Relative width of each hemithorax
      4. Differences in lucency of right and left lower and upper lung fields
      5. Differences in motility or fixation of peripheral and mid-lung marking

The Hila

1. Observe RIGHT and LEFT hilus separately
2. Each hilus has SUPERIOR and INFERIOR DIVISION
   a. Greater motility of inferior division during deep breathing
3. REDUCED MOTION of hilar markings caused by:
   a. Restricted diaphragmatic motion
   b. Pulmonary fibrosis
   c. Inflammatory or neoplastic fixation
   d. Apical and subapical pleural synechias (superior hilus)
4. COMPRESSION OF HILUM may be seen during expiration when expiratory trapping is marked (emphysema)
5. ROTATIONAL MOVEMENT of hilum due to:
   a. Relative fixation of hilus centrally, periphery free
   b. Segmental peripheral fixation of hilus, center free
   c. Marked differences in intrapulmonary pressures between adjacent segments (tension cyst, segmental atelectasis)

Intercostal Movement and Peripheral Lung Marking

Breathing may be largely intercostal in some people.

1. Note that intercostal motion is seen best during deep breathing.
   a. Examine in A-P and P-A.
2. Note that intercostal motion is normally less over upper lung field than lower.
3. Note any inequalities of costal movement, comparing right and left sides of the upper, middle and lower lung fields.
   a. Use narrow vertical screen aperture and full screen horizontally.
4. Local reduction of costal movement or reduced mobility of peripheral lung markings may be due to:
   a. Old or recent pleural involvement
   b. Inflammatory or neoplastic disease.
Special Exhibit on Pulmonary Function Testing

The Special Exhibit on Pulmonary Function Testing is presented by the Section on Diseases of the Chest of the American Medical Association. It has been developed and continued with the help of many individuals, under the auspices of the following General Committee and representatives of Government Services:

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The Picker X-Ray Corp. Fluoroscope was provided for the 1964 exhibit by Sicular X-Ray Corp., 1420 Sutter St., San Francisco, California
Arthur H. Thomas Co., 230 South Seventh St., Philadelphia 5, Pennsylvania
PULMONARY FUNCTION TESTING

A Special Exhibit from the Section on Diseases of the Chest

Acknowledgments

These panels were prepared for a special exhibit from the Section on Diseases of the Chest of the American Medical Association with the cooperation of the Joint Committee on Occupational Diseases of the Chest of the American College of Chest Physicians, the Industrial Medical Association, and the American Trudeau Society; the Committee on Occupational Diseases of the Chest and the Committee on Physiologic Treatment, Council on Research, American College of Chest Physicians and the Veterans Administration Committee on the Cooperative Study of Pulmonary Function Testing.

Many individuals participated in the preparation of this exhibit. Particular thanks are due to Dr. Maurice Segal, Boston, who provided the material for the panel devoted to the open method for F.R.C. of Darling, Courmand and Richards (J.C.I. 19: 609, 1940); to Dr. Julius Comroe, Jr., Philadelphia, for some of the material presented in Definition of Objectives and for other helpful suggestions; to Dr. Edwin Gaensler, Boston, for the material concerning Bronchospirometry; to Dr. John Seabury, New Orleans, for the material concerning Fluoroscopy and to all others who generously helped in many ways. The arrangements for the exhibit were made by Dr. Edwin R. Levine, Chicago, Representative of the Section on Diseases of the Chest to the Scientific Exhibition of the American Medical Association. The other officers of the Section on Diseases of the Chest have, by their individual and collective cooperation, contributed in a major way to this project. For the participation of The American College of Chest Physicians, of the officers of the College and of its Executive Administration, particularly for the tireless and effective assistance of Mr. Murray Kornfeld and his staff, the physicians concerned with this project give special thanks.

Those who have worked on this Exhibit feel a deep sense of gratitude to Dr. Thomas G. Hull, Secretary, Council on Scientific Assembly, American Medical Association, to Mrs. Hull and to Miss Frances Nyberg, Secretary to Dr. Hull, for all their help and guidance.

The material for the panels was assembled in the Radioisotope Service and Research Laboratory of Thayer Veterans Administration Hospital, Nashville, Tennessee, and prepared for presentation by Dr. Donald S. Tysinger, Jr., Dr. James J. Callaway, Dr. Joseph M. Merrill, and Mrs. Con O. T. Ball.

The Fluoroscopy posters were prepared in the Department of Medical Illustration of Louisiana State University, Mr. Gerald Hodge, Director. The other panels were prepared and the whole exhibit completed in the Medical Illustration Laboratory, Thayer Veterans Administration Hospital, Nashville, Tennessee, by Mr. Homer Jones, Chief, and Mrs. Ann Marshall Rees, Medical Artist.

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