Welcome

Welcome to the Snowdrift Frontline Treatment Monographs. The authors welcome you to this series of monographs that aim to disseminate worldwide new knowledge about common pulmonary disorders. We offer our messages to anyone who will find them useful in the diagnosis and treatment of the many pulmonary disorders that continue to plague mankind around the world. We invite you to download these monographs and use them in your teaching and practice of medicine. We feel a fraternal connection to all practitioners who serve the suffering. We hope that we can move toward the prevention of disease as an alternative to premature morbidity and mortality.

The Authors.

Mission Statement

The Snowdrift Pulmonary Conference is a not-for-profit corporation that is dedicated to the dissemination of knowledge about the lungs and lung diseases. Composed of both private practice pulmonologists and academicians, the conferees have launched a consumer-oriented program for primary care practitioners and the patients they serve. As a result, the following concise and authoritative monographs have been written.
Books in the Frontline Series

*Frontline Treatment of COPD, 2000*
*Frontline Treatment of Asthma, 1997*
*Frontline Treatment of Common Respiratory Infections, 1998*
*Frontline Treatment of Venous Thromboembolism, 1999*
*Frontline Assessment of Common Pulmonary Presentations, 2000*
*Frontline Assessment of Lung Cancer and Occupational Pulmonary Diseases, 2001*
*Frontline Pulmonary Procedures and Interventions, 2001*
*Frontline Cardiopulmonary Topics / Dyspnea, 2001*
*Frontline Advice for COPD Patients, 2002*

* Available on the web for downloading
Frontline Pulmonary Procedures and Interventions

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Preface

This is the seventh monograph in the Frontline series. It is written for frontline physicians who encounter a wide array of common pulmonary problems in daily practice. Each Section briefly describes the indications, uses, misuses, and abuses of various techniques and procedures that may be employed by frontline physicians. In today’s economic climate, it is wise for both frontline practitioners and consultants to develop pragmatic approaches to diagnosis and treatment. Basic diagnostic techniques such as spirometry, arterial blood gases (ABG’s) and chest x-rays are appropriately ordered by all physicians involved in either primary or specialty care. But, other procedures and therapeutic interventions generally are best utilized in conjunction with a pulmonologist or other specialists.

The purpose of this monograph is to update the frontline, primary care physician on a wide variety of specialized procedures, their applications, and how best to employ them. Although medicolegal implications may be a consideration in the selection of procedures and interventions, the primary consideration is what is appropriate for the optimal care of the patient.

In keeping with the statements made in our previous monographs, we aim to provide primary care practitioners with both contemporary and concise information about pulmonary procedures that should be of value in daily practice. The Sections in this monograph were written by both academic and private practicing pulmonologists, using a consensus format. We hope that you will profit from the clinical and academic experience compiled in this monograph and that this information will assist you in the care of your patients.

The Authors
Pearls

- Pulse oximetry may overestimate the true arterial oxygen saturation when carboxyhemoglobinemia is present, including that seen in heavy cigarette smokers.

- Pulse oximetry alone is inadequate in the initial assessment of patients with acute respiratory distress due to asthma or exacerbations of chronic obstructive pulmonary disease (COPD). Arterial blood gases (ABG’s) are needed to determine adequacy of ventilation.

- Spirometry can now be performed in the primary care practitioner’s office using widely available, inexpensive, user-friendly, portable, and accurate office spirometers.

- Fully inflated normal lungs empty in six seconds or less. This is why the FEV₆ can be used as a surrogate marker of FVC.

- A pulmonary artery (PA), balloon-tipped catheter should be removed when therapeutic decisions are no longer being made on the basis of data provided by the catheter.

- Before any protocol is initiated to wean a patient from the ventilator, the practitioner should first determine whether the patient can simply be extubated. A 30-minute trial on oxygen delivered by T-piece will be helpful.

- Non-invasive ventilation techniques, such as bi-level positive airway pressure (BiPAP), may be effective in eliminating the need for intubation and mechanical ventilation in some patients with respiratory insufficiency.
Attention to the patient being weaned from mechanical ventilation is more important than the method being used.

All who snore do not have obstructive sleep apnea-hypopnea syndrome (OSAHS); but, all who have OSAHS snore.

To achieve high rates of adherence (compliance) with nasal continuous positive airway pressure (CPAP) for patients with sleep apnea, one must achieve proper fit of the nasal mask or pillows.

A routine chest radiograph should generally be obtained before specialized thoracic imaging studies are obtained.

The ventilation-perfusion (V/Q), lung scan is the initial study of choice for the diagnosis of pulmonary emboli (PE) in patients with a normal chest radiograph.

Sputum cytology studies have a high yield in patients with large, central tumors on chest x-rays. However, they also are highly useful in the early detection of bronchogenic carcinomas in patients with normal chest x-rays but smoking-induced chronic airflow obstruction.

Flexible fibrotic bronchoscopy should be performed promptly on patients with recurrent hemoptysis, localized inspiratory wheezing, and/or unresolving pulmonary infiltrates.

Community-acquired pneumonia (CAP) can be treated empirically in most adults. However, for patients with complicating diseases or special risk factors, an aggressive search for the etiologic agent is indicated.
• Interstitial lung disease may be mistaken for recurrent pneumonia or heart failure on chest radiography.

• Pneumonia involving bullous lesions in emphysema patients can mimic lung abscesses or cavities.

• e-mail is not like using the telephone. We must consider both confidentiality and the risk of misinterpretation before using e-mail to communicate with patients.

• Physicians can help their patients seek information on the Internet by being proactive “navigators” to direct them to credible, responsible sources of information.
A. Spirometry

Spirometry is one of the most useful, yet underutilized diagnostic techniques in all of medicine. Although introduced in the mid-1800’s by John Hutchinson, a surgeon, spirometry has been reluctantly accepted in the day-to-day practice of both general and specialty medicine. This hesitancy seems related to misconceptions and poor understanding of the technique, which continue to cloud its value, and the expense and complexity of previously-used instruments.

Spirometry primarily measures expiratory airflow from fully inflated lungs. Spirometry can also measure inspiratory airflow (See below).

The Basis of Pulmonary Mechanics

To take a large breath, individuals must create a negative pressure in the pleural space. This is achieved by the descent of the diaphragms and enlargement of the rib cage by the action of the thoracic and neck muscles. This negative pressure creates the force for airflow to fill the lungs to the point of maximum inflation. Expiratory airflow may be passive or, for the purposes of testing, may be initiated by forceful effort. Much of the expiratory airflow is a function of the elastic recoil of the lung tissue and the conductance of air through small and large airways. Following the initiation of forced exhalation, the alveoli empty into small airways, and small airways empty into large airways. As the air rushes out of the lungs through the mouth, the airflow is recorded by a spirometer. The forced expiratory volume in one second (FEV₁), is a flow test. The entire forced expiration (forced vital capacity or FVC) is a volume test. Recently, new terminology has been added to simple spirometry. Since normal lungs empty in six seconds, a recent convention has substituted the FEV₆ (also known as FEV₆ for the FVC) to make testing more convenient for both the patient and the spirometer technician. Normally, the patient expires approximately 70\% of his/her vital capacity in the first second. Thus, the normal ratio of the FEV₁ to FVC is...
Obstructive Lung Diseases

Table 1 lists the common diseases characterized by expiratory airflow obstruction. In obstructive diseases the FEV₁/FVC (FEV₁/FVC) is less than 70% and the absolute FEV₁ is usually less than 80% of predicted. Examples of the normal volume-time and flow-volume curves are presented in Figure 1. Both expressions present the same data, but in a different format. The advantage of the volume-time convention is that the FEV₁ and FVC can be directly visualized. The advantage of the flow-volume display is that the peak flow can be directly visualized as an indication of a good expiratory effort. Clinicians should be comfortable using either form of expiratory airflow recording. Examples of mild, moderate and severe expiratory airflow obstruction are shown in Figures 2, 3 and 4.

Restrictive Ventilatory Disorders

In restrictive ventilatory disorders, the lungs are “stiff” which results in increased elastic recoil pressure. This typically limits the amount of air drawn into the lungs, but produces higher than normal expiratory flow ratios. Thus, the FVC (or FEV₁) is less than 80% of predicted and the ratio of FEV₁/FVC is usually much higher than 70%, often 80%, 90% or even more. Examples of moderate and severe ventilatory restriction are presented in Figures 5 and 6. The common restrictive ventilatory defects are listed in Table 2.

Inspiratory Flow Disorders

Most respiratory disorders result in disturbance of expiratory airflow. By contrast, disorders such as vocal cord dysfunction, tracheal tumors or tracheal stenosis result in impaired inspiratory flow. This may not be detected on simple spirometry, but requires specialized studies. When such conditions are believed to be present, consultation with a pulmonologist and/or an otolaryngologist is advised.
Algorithm for Interpretation of Spirometry

A simple algorithm for identifying restrictive and obstructive ventilatory disorders is shown in Figure 7. Lung volume measurements may be needed to adequately evaluate restrictive pulmonary disease state (See Section B).

Spirometry and Clinical Diagnosis

It should be stressed that spirometric measurements do not make a clinical diagnosis. Rather, spirometric abnormalities indicate a physiological impairment from which the physician must derive the diagnosis. It has been traditional to label reversible airflow obstruction as asthma and irreversible airflow disorders as chronic obstructive pulmonary disease (COPD). Overlaps are common. Responses to therapy, including the use of bronchoactive drugs, i.e., bronchodilators, corticosteroids and other new agents, will help determine how to establish the diagnosis of reversibility or irreversibility. Consultation is advised in complex situations.

Prognostic Value

Spirometric abnormalities are predictive of increased risk of many diseases and all cause mortality. The major diseases associated with spirometric abnormalities are listed in Table 3. Note that not only lung diseases such as lung cancer or COPD are included, but also ischemic heart disease and cerebrovascular accidents are associated prognostically with spirometric dysfunction.

Conclusion

No doctor would prescribe antihypertensive agents without blood pressure measurements, antiarrhythmics without evidence of cardiac rhythm disturbances, insulin without measurements of blood sugar or Coumadin® without prothrombin measurements. Analogously, it is not appropriate to treat obstructive or restrictive lung diseases without spirometry. Spirometry is as fundamental to the diagnosis and management of pulmonary diseases as the sphygmomanometer is to the assessment and management of hypertension.

(continued)
Table 1  Common Obstructive Ventilatory Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
</tr>
<tr>
<td>Asthmatic bronchitis</td>
</tr>
<tr>
<td>Chronic obstructive bronchitis</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease (COPD)¹</td>
</tr>
<tr>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>Emphysema</td>
</tr>
</tbody>
</table>

¹ COPD is a generic term that includes asthmatic bronchitis, chronic bronchitis, bronchiectasis and emphysema. These states commonly overlap.


Table 2  Common Restrictive Ventilatory Disorders

<table>
<thead>
<tr>
<th>Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic fibrosing alveolitis</td>
</tr>
<tr>
<td>Interstitial pneumonitis and fibrosis¹</td>
</tr>
<tr>
<td>Fibrotic residue of disseminated granulomas (e.g., tuberculosis, histoplasmosis)</td>
</tr>
<tr>
<td>Sarcoidosis</td>
</tr>
<tr>
<td>Thoracic deformities</td>
</tr>
<tr>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
</tr>
</tbody>
</table>

¹ May be associated with drug reactions, e.g., bleomycin (Blenoxane), or occupational exposures, e.g., asbestosis, or collagen vascular diseases, e.g., rheumatoid arthritis.

Table 3  Diseases with High Risk Associated with Spirometric Abnormalities

<table>
<thead>
<tr>
<th>Disease State</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart attack</td>
<td>J Chron Dis 1987;40:849-856.</td>
</tr>
<tr>
<td>All cause mortality</td>
<td>Am J Epidemiol 1994;140:398-408.</td>
</tr>
</tbody>
</table>

Table 4  When to Perform Spirometry

In all smokers age 45 and over

In patients of any age with

- dyspnea
- cough
- wheeze
- mucus hypersecretion

Normal Flow-volume (A) and volume-time (B) curves in a normal individual. LLN indicates lower limit of normal. Notice that the expiratory time can be visualized from the volume-time curve, but the peak flow can only be visualized from the flow-volume curve. Thus, both curves are useful. Adapted from: Enright PL, Hyatt PE: Office spirometry (A practical guide to the selection and use of spirometers). Lea & Febiger, Philadelphia, 1987.

Mild Obstruction Flow-volume (A) and volume-time (B) curves in a patient with mild airflow obstruction.
**Figure 3  Moderate Obstruction**

Moderate Obstruction Flow-volume (A) and volume-time (B) curves in a patient with moderate airflow obstruction.

**Figure 4  Severe Obstruction**

Severe Obstruction Flow-volume (A) and volume-time (B) curves in a patient with advanced airflow obstruction from emphysema.
Moderate Restriction  Flow-volume (A) and volume-time (B) curves in a patient with a moderate restrictive ventilatory disorder. Notice that FEV₁/FVC is nearly 90%. A ratio this high may suggest a restrictive disorder; however, normal individuals can often empty most of the lung in one second.

Severe Restriction  Flow-volume (A) and time-volume (B) curves in a patient with a severe restrictive ventilatory disease due to idiopathic pulmonary fibrosis. Notice the FEV₁/FVC is 99%.

Figure 7 Algorithm for Interpreting Spirometry Results

<table>
<thead>
<tr>
<th>Acceptable Spirometry</th>
<th>Is FEV₁/FVC ratio low?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Obstructive Defect</td>
</tr>
<tr>
<td>No</td>
<td>Is FVC low?</td>
</tr>
<tr>
<td>Yes</td>
<td>Restrictive defect</td>
</tr>
<tr>
<td>No</td>
<td>Normal spirometry results</td>
</tr>
</tbody>
</table>

Is FVC low?

Yes

Hyper-inflation versus combined defect

Further testing (lung volume measurements)

Reversible with use of beta agonist?

Yes

Asthma

No

COPD

1. If clinical correlation is present.
2. Some COPD may have a reversible component.
According to the Spirometry Committee of the National Lung Health Education Program (NLHEP), all smokers age 45 and over, and individuals of any age with symptoms of cough, dyspnea, mucus hypersecretion or wheeze should have spirometry (See Table 4). The NLHEP aims to identify COPD and related disorders in their early stages in primary care physicians’ offices. There are fewer than 10,000 board certified pulmonologists in North America, but there are approximately 220,000 primary care providers. Every year these professionals see at least 70% of the 45,000,000 smokers in the United States for some medical problem. All smokers should be strongly advised to stop. Spirometric abnormalities may be persuasive evidence to assist in this difficult task.

Early identification of patients with spirometric abnormalities not only indicates an increased risk of the four common causes of death noted above, it also signals increases in all cause mortality. Smokers with spirometric abnormalities have an urgent need to stop. Spirometry will not only guide responses to therapy in both obstructive and restrictive disorders, but will help to predict the patient's long-term progress. ■
Early identification of patients with spirometric abnormalities not only indicates an increased risk of the four common causes of death noted in the text, it also signals increases in all cause mortality.
References

Anthonisen NR, Connett JE, Kiley JP, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV₁. The Lung Health Study. JAMA 1994;272:1497-1505. This landmark study showed an improvement in FEV₁ following stopping smoking, and a reduced rate of decline in FEV₁ over five years. Continuing smokers lost FEV₁ at a much faster rate.

Burrows B, Knudson RJ, Camilli AE, et al. The “horse-racing effect” and predicting decline in forced expiratory volume in one second from screening spirometry. Am Rev Respir Dis 1987;135:788-793. This study demonstrated that a rapid rate of decline in FEV₁ was predictive of premature mortality from COPD.


Hankinson JL, Odencrantz JR, Fedan KB. Spirometric reference values from a sample of the general U.S. Population. Am J Respir Crit Care Med 1999;159:179-187. This article provides normal spirometric values from the largest population thus far. It gives evidence that the FEV₆ is an excellent surrogate for the FVC.


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Strachan DP. Ventilatory function as a predictor of fatal stroke. BMJ 1991;302:84-87. This summary documents an increased risk of stroke in smokers with airflow obstruction.

B. Lung Volume Studies

Introduction

Measurement of lung volumes plays an integral role in the evaluation of patients with known or suspected lung disease. As discussed in the previous Section, spirometry can establish the presence or absence of obstructive pulmonary dysfunction. Spirometry may also document restrictive pulmonary dysfunction characterized by a reduction in total lung volumes, but frequently additional techniques to measure total lung volumes are needed. Restrictive dysfunction is defined as a reduction of the total lung capacity (\( \text{TLC} \)) and is associated with a wide variety of disorders involving the lung parenchyma, the thorax, the diaphragms and neuromuscular diseases (See Table 5). Lung volumes can be measured by a variety of methods. The two most commonly used are the inert gas dilution and the body plethysmography techniques, which are discussed below.

Methods of Measuring Lung Volumes

Inert Gas Dilution Method: In this method, an inert gas, usually helium, is inspired from a closed-circuit spirometer. After a period of time, the concentration of helium equilibrates between the spirometer and the lungs. Oxygen is added to the system and carbon dioxide is removed. The inert gas is neither absorbed nor metabolized. The FRC is then calculated and the RV is determined by subtracting the FRC from the ERV.

Problems with this method include the following. In patients with chronic obstructive pulmonary disease (COPD), the FRC may be underestimated due to uneven ventilation and the presence of air spaces that do not communicate with the bronchial tree (e.g., bullae). A gas leak in the system will falsely increase the RV. The inability of the patient to maintain a tight seal on the mouthpiece connected to the spirometer is a common cause of erroneously high values for the RV.

Body Plethysmography Method: In this method, the subject sits in an airtight chamber and performs maneuvers to expand and compress the lung volumes.

(continued)
## Table 5  Causes of Restrictive Lung Disease

<table>
<thead>
<tr>
<th>Pulmonary</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Idiopathic pulmonary fibrosis</td>
<td></td>
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<tr>
<td>Lung resection</td>
<td></td>
</tr>
<tr>
<td>Atelectasis</td>
<td></td>
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<tr>
<td>Pneumonia</td>
<td></td>
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<tr>
<td>Hypersensitivity pneumonia</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure (can have a restrictive and obstructive pattern)</td>
<td></td>
</tr>
<tr>
<td>Alveolar proteinosis</td>
<td></td>
</tr>
<tr>
<td>Bronchiolitis obliterans with organizing pneumonia (BOOP)</td>
<td></td>
</tr>
<tr>
<td>Pneumoconiosis (coal workers’ pneumoconiosis, asbestosis, chronic beryllium disease)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Extrapulmonary</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleural effusion</td>
<td></td>
</tr>
<tr>
<td>Pneumothorax</td>
<td></td>
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<tr>
<td>Pregnancy</td>
<td></td>
</tr>
<tr>
<td>Ascites</td>
<td></td>
</tr>
<tr>
<td>Morbid obesity</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Chest Wall</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Kyphoscoliosis</td>
<td></td>
</tr>
<tr>
<td>Ankylosing spondylitis</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Neuromuscular</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Guillain Barré syndrome</td>
<td></td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td></td>
</tr>
<tr>
<td>Amyotrophic lateral sclerosis</td>
<td></td>
</tr>
<tr>
<td>Poliomyelitis</td>
<td></td>
</tr>
<tr>
<td>Diaphragmatic paralysis</td>
<td></td>
</tr>
</tbody>
</table>

By applying Boyle’s law\(^1\) lung volumes are calculated. Body plethysmography gives more accurate measurements of lung volumes than the inert gas method in patients with COPD. Indeed, the TLC may be found to be substantially greater when measured by this technique. Another advantage of body plethysmography is that several measurements can be done over a few minutes. Occasionally, however, claustrophobic patients may not be able to tolerate the confines of the plethysmograph chamber.

### Lung Volumes Definitions

(See Figure 8)

1. **Total lung capacity (TLC):** Total volume of gas in the lungs after maximal inspiration.
2. **Vital capacity (VC):** The volume of gas expired during a maximal effort.
3. **Residual volume (RV):** The amount of gas remaining in the lungs after maximal expiration.
4. **Functional residual capacity (FRC):** The amount of gas remaining in the lungs at the end of a quiet expiration.
5. **Inspiratory capacity (IC):** The maximal amount of gas inspirable from FRC.
6. **Inspiratory reserve volume (IRV):** The maximal amount of gas inspirable after the end of a quiet inspiration.
7. **Tidal volume (TV):** The amount of gas moving in and out of the lungs with each respiratory cycle.
8. **Expiratory reserve volume (ERV):** The additional volume of gas that can be expired at the end of a quiet expiration.

### Diffusing Capacity (Transfer Factor)\(^2\)

Diffusion is the process of transferring oxygen molecules from the alveolus, through the alveolar-capillary membrane, then across the red blood cell wall.

---

\(^1\) Boyle’s law states that the product of the pressure (P) and volume (V)/(PV) of a gas is constant under constant temperature (isothermal) conditions.

\(^2\) The term carbon monoxide diffusing capacity (DL\(_{CO}\)) is used in North America. It is called the transfer factor in Europe.
Figure 8  Lung Volumes

Total Lung Capacity (TLC)
Vital Capacity (VC)
Residual Volume (RV)
Functional Residual Capacity (FRC)
Inspiratory Capacity (IC)
Inspiratory Reserve Volume (IRV)
Tidal Volume (TV)
Expiratory Reserve Volume (ERV)

where it binds to hemoglobin. Quantifying this process in the pulmonary function laboratory is an important element of evaluating a patient with known or suspected pulmonary disease, and in particular, those complaining of dyspnea. Generally, the diffusing capacity is conceptualized as a representation of the effective surface or interface between the alveoli and the capillary units of the lung. Multiple factors influence the diffusion capacity, including the number of functioning alveolar-capillary units, the volume of blood in the pulmonary capillaries, matching of ventilation to perfusion and the thickness of the alveolar-capillary membrane.

Methods of Measuring the Diffusing Capacity

In most clinical pulmonary function laboratories, the diffusing capacity is determined using the single breath carbon monoxide method. Carbon monoxide has a strong affinity for hemoglobin and the diffusing capacity for carbon monoxide (DLco) is a reliable surrogate for oxygen transfer. In this test, the subject maximally inhales a gas mixture containing a trace concentration of carbon monoxide and a suitable concentration of helium from a closed system, and holds his or her breath for 10 seconds. The gas is expired into a spirometer and is analyzed for the concentration of carbon monoxide and helium. Helium is used to calculate the lung volume. The changes in carbon monoxide concentration between inspiration and expiration estimates the amount of carbon monoxide transferred to the red blood cells. The results are expressed as ml/min/mmHg. The normal value is usually in the range of 20 ml to 30 ml/min/mmHg. Corrections are made for the subject’s hemoglobin level. Subjects with small lung volumes and normal diffusion can have a reduced DLco. Compensation for this can be made on the basis of the ratio of the diffusing capacity to alveolar volume (D/VA). In normal subjects, the TLC approximates the alveolar volume (VA) measured during the DLco measurement.
### Figure 9  Interpretation of Pulmonary Function Tests

<table>
<thead>
<tr>
<th>Condition</th>
<th>FVC</th>
<th>FEV1</th>
<th>FEV1/FVC</th>
<th>TLC</th>
<th>RV</th>
<th>RV/TLC</th>
<th>DLco</th>
<th>D/VA</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
<td>N to ↑*</td>
<td>↓</td>
<td>↓</td>
<td>N to ↑</td>
<td>↑</td>
<td>↑</td>
<td>N↓</td>
<td>N↓</td>
</tr>
<tr>
<td>Emphysema</td>
<td>N to ↑*</td>
<td>↓</td>
<td>N to ↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Asthma</td>
<td>N to ↑</td>
<td>N to ↓</td>
<td>↓</td>
<td>N to ↑</td>
<td>↑</td>
<td>↑</td>
<td>N to ↑</td>
<td>N to ↑</td>
</tr>
<tr>
<td>IPF</td>
<td>↓</td>
<td>↓</td>
<td>N to ↑</td>
<td>↓</td>
<td>↓</td>
<td>N</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>PE</td>
<td>N to ↓</td>
<td>N to ↓</td>
<td>N</td>
<td>N to ↓</td>
<td>N to ↓</td>
<td>N</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>CHF</td>
<td>N to ↓</td>
<td>N to ↓</td>
<td>N</td>
<td>↑</td>
<td>N to ↑</td>
<td>↑ or ↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N to ↑</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>N.M.</td>
<td>N to ↓</td>
<td>N to ↓</td>
<td>N</td>
<td>N to ↓</td>
<td>N to ↓</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

*Only in early stages of disease. In later stages of both, FVC decreases.

COPD=Chronic obstructive pulmonary disease
IPF=Idiopathic pulmonary fibrosis
PE=Pulmonary embolus
CHF=Congestive heart failure
N.M=Neuromuscular
N=Normal
↑=Increased
↓=Decreased

**B. Lung Volume Studies**

**Interpretation of Pulmonary Function Tests**

Normal values for the \( \text{DLCO} \) are influenced by age, size and sex. Classically, the diffusion capacity is reduced in patients with emphysema, interstitial lung disease and pulmonary vascular diseases. It is increased in polycythemia, intra-alveolar hemorrhage such as is seen in Goodpasture’s syndrome, the supine position and after exercise (See Figure 9). For a more comprehensive list of diseases affecting the diffusing capacity, refer to Table 6.

Measurement of the \( \text{DLCO} \) is especially helpful in evaluating the dyspneic patient with normal spirometry, lung volumes and normal chest x-rays. Diminishing \( \text{DLCO} \) in this setting may suggest to the clinician such conditions as primary pulmonary hypertension, recurrent pulmonary emboli (PE) or obstructive vasculopathy. The diffusing capacity is also useful in following the course and response to treatment of sarcoidosis, idiopathic pulmonary fibrosis and other interstitial diseases.

**Pharmacologic Broncho-provocation Challenge (PBC)**

In 1962, the American Thoracic Society defined asthma as a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli, and manifested by a widespread narrowing of the airways that changes spontaneously or as a result of therapy. Bronchial hyperresponsiveness is the hallmark of asthma and can be demonstrated in virtually all patients with asthma. A wide variety of stimuli can provoke bronchial constriction including exposure to cold air, allergens, smoke, chemicals, dust and exercise. In the majority of patients, the history and physical examination and pulmonary function testing provides sufficient evidence to support a diagnosis of asthma. However, in patients presenting with chronic cough or unexplained dyspnea, routine pulmonary function studies can be normal and pharmacologic bronchoprovocation challenge (PBC) can document bronchial hyperreactivity.

(continued)
Table 6  Factors Affecting the Diffusing Capacity

<table>
<thead>
<tr>
<th>Increased Diffusing Capacity</th>
<th>Decreased Diffusing Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polycythemia rubra vera</td>
<td>Emphysema</td>
</tr>
<tr>
<td>Obesity</td>
<td>Lung resection</td>
</tr>
<tr>
<td>Pregnancy (first trimester)</td>
<td>Pulmonary emboli (PE)</td>
</tr>
<tr>
<td>Intra-alveolar hemorrhage (Goodpasture’s Syndrome)</td>
<td>Primary pulmonary hypertension</td>
</tr>
<tr>
<td>Left to right intra-cardiac shunt</td>
<td>Anemia</td>
</tr>
<tr>
<td>Supine posture</td>
<td>Idiopathic pulmonary fibrosis</td>
</tr>
<tr>
<td>After exercise</td>
<td>Sarcoidosis</td>
</tr>
<tr>
<td></td>
<td>Asbestosis</td>
</tr>
<tr>
<td></td>
<td>Alveolar proteinosis</td>
</tr>
<tr>
<td></td>
<td>Hypersensitivity pneumonia</td>
</tr>
<tr>
<td></td>
<td>Congestive heart failure (CHF)</td>
</tr>
<tr>
<td></td>
<td>Obstructive vasculopathy</td>
</tr>
<tr>
<td></td>
<td>Collagen vascular diseases</td>
</tr>
<tr>
<td></td>
<td>Drug induced interstitial lung disease (methotrexate, amiodarone, nitrofurantoins, bleomycin)</td>
</tr>
<tr>
<td></td>
<td>Pregnancy, late</td>
</tr>
<tr>
<td></td>
<td>Carboxyhemoglobinemia (related to smoking)</td>
</tr>
</tbody>
</table>

Indications for Pharmacologic Broncho-provocation Testing

1. To rule in or out asthma or to determine the severity of asthma in patients with chronic cough but normal spirometry,
2. Unexplained chest tightness, dyspnea and/or decrease in exercise tolerance,
3. Recurrent episodes of bronchitis or pneumonia and/or,
4. Occupational asthma.

Contraindications for Pharmacologic Broncho-provocation Testing

1. History of unexplained urticaria or angioedema,
2. Baseline FEV₁ less than 60% of predicted,
3. Pregnancy,
4. Unstable cardiac status and/or,
5. Recent viral respiratory tract infection.

PBC Testing Methods

A number of substances and modalities have been used in PBC testing including methacholine, histamine, hyperventilation, exercise and cold air. Methacholine is a cholinergic drug that stimulates muscarinic receptors in the bronchial smooth muscles, provoking bronchoconstriction. It is the agent most commonly used in the PBC for the following reasons: safety, availability, cost, ease of administering and standardizing, is non-allergenic and non-sensitizing and it stimulates a natural asthma attack in susceptible individuals in a controlled manner.

Several protocols have been used, but all rely upon administering incremental doses of methacholine by inhalation and sequentially measuring the FEV₁. Normal saline is administered as a control. The test is considered positive if the FEV₁ falls by 20% or more after the inhalation of methacholine, compared to nebulized normal saline. The lower the concentration of methacholine that provokes this reaction, the more severe the asthma is deemed. By contrast, even “normal” persons will experience bronchoconstriction
at high concentrations (2.5 mg/ml). A fall of 16% to 19% is said to be equivocal and the challenge should be repeated. A fall of less than 16% virtually rules out asthma. The reproduction of the symptoms such as cough, wheeze or chest tightness when present, is helpful in interpretation of the test.

In two prospective studies with patients with chronic cough, the positive and negative predicative values were 60% and 100% respectively. In the evaluation of patients with unexplained dyspnea, the positive and negative predictive values were 95% and 100% respectively. Irwin and colleagues advise that a positive test must be correlated with a favorable response to therapy before concluding that the patient has asthma. False-positive responses can be seen in patients with recent viral upper or lower respiratory infections (within up to six weeks of testing), sarcoidosis, hypersensitivity pneumonia, congestive heart failure and pulmonary eosinophilia.

Safety

In two studies of over 2,500 patients tested by the PBC there were no instances of severe episodes of bronchospasm requiring hospitalization. In patients with a positive test, bronchospasm is usually reversed with inhalation of a beta agonist bronchodilator within five minutes. Other symptoms such as chest tightness, sore throat, cough, dyspnea and dizziness are usually transient and resolve within 24 hours.
References


Comroe JH, Jr., Forster RE, Dubois AB, et al. The Lung: Clinical Physiology and Pulmonary Function Tests. 2nd ed. Chicago, IL: Year Book Medical Publishers, Inc.; 1962. pp 390. This is the classic primer relating pulmonary physiology and lung function tests to various respiratory disorders.

Hyatt RE, Scanlon PD, Nakamura M. Interpretation of Pulmonary Function Tests: A Practical Guide. Philadelphia, PA: Lippincott Williams & Wilkins; 1997. pp 212. This monograph is recommended to the primary care physician who is seeking a concise and practical textbook on pulmonary function testing. The chapter of illustrative cases is particularly useful in helping the physician grasp the concepts of pulmonary physiology as it relates to clinical practice.


C. Arterial Blood Gases (ABG’s) and Pulse Oximetry

Arterial Blood Gases (ABG’s)

Arterial blood gases (ABG’s) measure what is perhaps the most important aspect of lung function, that of gas exchange and acid-base balance. Interpretation of ABG’s must be considered in perspective. In general, abnormalities in ABG’s correlate with abnormalities in other aspects of lung function. However, patients can have severe lung disease and maintain normal or near normal ABG’s. Likewise, relatively minor abnormalities in lung physiology, as measured by other pulmonary function tests such as spirometry, can be accompanied by severe abnormalities in ABG’s. One must consider the possible mechanisms causing the abnormalities to interpret their meaning and clinical importance to the individual patient.

Technical Aspects

Arterial puncture is a relatively easy procedure. Some practitioners favor infiltrating around the artery from which the blood is to be drawn with lidocaine or a similar local anesthetic agent. However, usually the blood can be drawn with a minimum of discomfort, similar to the initial skin needle puncture from injecting the lidocaine, such that a local anesthetic is unnecessary. The filling of the syringe by the arterial pressure and the color of the blood usually indicates an arterial source. Obviously, the color cannot be relied on in the presence of severe hypoxemia and oxygen desaturation.

Commonly, prepackaged syringes containing powdered heparin are used. If prepackaged syringes are not available, the syringe should be coated with heparin, but without any excess heparin solution in the syringe. The syringe containing the arterial blood should be capped and placed on ice for transport to the laboratory where the measurement should be made as soon as possible.

(continued)
The oxygen tension in the arterial blood \( (p_{aO_2}) \) reflects the end result of the complicated process of transport of oxygen from the inhaled air or gas mixture to the arterial blood. The causes of a low \( p_{aO_2} \) or hypoxemia include:

1. Breathing a gas mixture low in oxygen content,
2. Hypoventilation,
3. Ventilation-perfusion lung scan (\( \tilde{V}/\tilde{Q} \)) mismatch,
4. Diffusion abnormality,
5. Intrapulmonary or intracardiac shunt and/or,
6. Altitude.

Usually, the possibility of breathing gas low in oxygen can be ruled out (or ruled in, e.g., at altitude with a low barometric pressure that is accompanied by the inspired pressure of oxygen \( [P_{1O_2}] \) lower than that at sea level). A diffusion abnormality is rarely the sole or primary cause of hypoxemia and is almost always accompanied by one of the other mechanisms of hypoxemia, which is considerably more important as the cause of the low \( p_{aO_2} \). A low diffusing capacity, as discussed in Section B, is primarily a reflection of the amount of alveolar lung surface which is approximated by capillary blood flow. The \( DLCO \) correlates poorly with abnormalities in \( p_{aO_2} \). Therefore, for practical purposes, one can usually limit the mechanisms of hypoxemia to three: hypoventilation, \( \tilde{V}/\tilde{Q} \) mismatch and shunt.

Hypoventilation is defined by the arterial level of \( CO_2 \) \( (p_{aCO_2}) \). If the \( p_{aCO_2} \) is elevated, then alveolar hypoventilation is present by definition, regardless of the amount of minute ventilation (the total air breathed in and out in one minute measured in liters/minute). Whether the remote ventilation is found to be low, normal or high, if the \( p_{aCO_2} \) is elevated, hypoventilation is present and contributing, to some degree, to the
hypoxemia. The degree to which the hypoventilation is contributing to the hypoxemia can be determined by using the alveolar air equation.

The alveolar air equation states that the alveolar pressure of oxygen \( p_{A\text{O}_2} \) is determined by the \( p_{1\text{O}_2} \) minus the alveolar pressure of \( \text{CO}_2 \) \( p_{A\text{CO}_2} \) divided by the respiratory quotient (RQ), the difference in the rates of oxygen uptake and \( \text{CO}_2 \) production. The equation is expressed as follows: \( p_{A\text{O}_2} = p_{1\text{O}_2} - p_{A\text{CO}_2}/\text{RQ} \). The \( p_{1\text{O}_2} \) can be determined by multiplying the barometric pressure minus the water vapor pressure by the fraction of inspired oxygen \( f_{1\text{O}_2} \). Since inhaled gas is completely saturated by water by the time it reaches the alveolar level, the water vapor value is constant at 47 mm Hg. For example, at sea level breathing ambient air, the \( p_{1\text{O}_2} \) would be \( (760 \text{ to } 47 \text{ mm Hg}) \times 0.21 = 150 \text{ mm Hg} \). The \( p_{A\text{CO}_2} \) (alveolar) is assumed to be the same as \( p_{a\text{CO}_2} \) (arterial). Although the RQ can vary, it is usually assumed to be 0.8. Thus if the \( p_{a\text{CO}_2} \) is normal at 40 mm Hg, the \( p_{A\text{O}_2} = 150 - 40/0.8 = 150 - 50 = 100 \text{ mm Hg} \). Next, the alveolar \( p_{A\text{O}_2} \) is compared to the arterial pressure of oxygen \( p_{a\text{O}_2} \) and the alveolar to arterial oxygen difference, \( p_{(A-a)\text{O}_2} \), is calculated. The normal range of \( p_{A\text{O}_2} \) at sea level is approximately 75 to 90 mm Hg. The normal range of \( p_{(A-a)\text{O}_2} \) while breathing air, therefore, is 100 minus 75 to 90 = 25 mm Hg. This normal range varies with age, increasing as we get older. When hypoventilation is the sole cause of hypoxemia, the \( p_{(A-a)\text{O}_2} \) is normal. The low \( p_{a\text{O}_2} \) is caused by a reduction in \( p_{A\text{O}_2} \) associated with the hypoventilation.

If the \( p_{(A-a)\text{O}_2} \) is increased, then either \( \dot{V}/\dot{Q} \) mismatch or shunt is present and contributing to the hypoxemia. Blood flow has to be present out of proportion to a reduction in ventilation, (i.e., \( \Phi Q \)), for \( \dot{V}/\dot{Q} \) mismatch to cause a low \( p_{a\text{O}_2} \). An increase in \( \dot{V}/\dot{Q} \) ratio would result in wasted ventilation and perhaps increased work of breathing, but would not generally be accompanied
by hypoxemia. A classic example of a cause of decreased \( \dot{V}/Q \) would be an asthma attack with widespread narrowing of airways, resulting in decreased ventilation, but with continued perfusion of the hypoventilated areas.

Shunt can be thought of as an extreme form of mismatch, i.e., no ventilation, but continued perfusion. \( \dot{V}/Q \) mismatch is separated or differentiated from shunt by the response to increasing the inhaled oxygen tension (See Figure 10). If \( \dot{V}/Q \) mismatch is present, eventually the increased oxygen mixture will wash out some of the nitrogen in the alveoli, since the airways are open to some degree, and the resulting increase in \( P_{A}O_{2} \) will increase the arterial \( PO_{2} \). If a shunt is present, i.e., no ventilation, there will be no increase in oxygen in the closed or fluid-filled alveoli, and thus, no change in oxygenation will occur in the blood flowing past those alveoli. The only increase in arterial \( PO_{2} \) will occur from increasing oxygen in the non-shunt areas of lung tissue. Since the oxygen saturation in these areas is usually nearly complete already (approximately 95% or greater) and thus, oxygen content is near maximum, the addition of even 100% oxygen to these areas will result in a relatively small increase in arterial oxygen content.

The use of increased \( F_{1}O_{2} \) to differentiate between \( \dot{V}/Q \) mismatch and shunt can be stated in practical terms. If a small increment in inhaled oxygen is administered, e.g., 2 liters per minute \( O_{2} \) by nasal prongs, the \( P_{A}O_{2} \) will show a measurable and clinically important increase when \( \dot{V}/Q \) mismatch is present, but virtually no change when shunt is present. If 100% oxygen (\( F_{1}O_{2}=1.0 \)) is administered, a very large increase in \( P_{A}O_{2} \) (usually to 350 mm to 500 mm Hg) will result if \( \dot{V}/Q \) mismatch is primarily present, but only a modest increase in \( P_{A}O_{2} \) will occur if shunt exists.
Ventilation perfusion (V/Q), mismatch and intrapulmonary shunt. See text for description of how these can be differentiated by administering oxygen.
Acid-Base Balance

Arterial blood gases are necessary to determine the acid-base status of the blood. On ABG’s, the pH and PCO₂ are directly measured, and the bicarbonate (HCO₃⁻) is calculated from the pH and PCO₂. If the calculated HCO₃⁻ is significantly different (e.g., ≥ 5) from the measured serum HCO₃⁻ from the blood chemistries, a laboratory error is possible, including the arterial pH, PₐCO₂ or venous CO₂ serum (HCO₃⁻).

In interpreting the acid-base status, first look at the pH. If the pH is low (< 7.36), acidemia is present. If the pH is high (> 7.44), alkalemia is present. If acidemia is present, the etiology can be respiratory, metabolic or mixed. Next, look at the PₐCO₂. If it is elevated, then a respiratory component of the acidemia exists. An elevated PₐCO₂ will result in more dissolved HCO₃⁻ (from the Henderson–Hesselbalch equation). Next look at the HCO₃⁻. If it is elevated, the acidemia is likely entirely respiratory in etiology. However, if the HCO₃⁻ is normal or low, then a metabolic component is also likely present. If the PₐCO₂ is normal or low and the HCO₃⁻ is low, then the acidemia is caused by a metabolic acidosis. It is common for a metabolic acidosis to be partially compensated by a respiratory alkalosis (decrease in PₐCO₂). Likewise, if the pH is alkalemic, look at the PₐCO₂. If the PₐCO₂ is significantly decreased, then a respiratory cause of the alkalemia is present. If the PₐCO₂ is normal or increased, look at the HCO₃⁻. If the HCO₃⁻ is increased, then a metabolic alkalosis exists (See Tables 7 and 8).

Use of equations and formulas allow more precise calculation of the degree of an acid-base abnormality and the amount of correction necessary to result in normal or near normal acid-base balance. For practical clinical purposes, however, an understanding of the physiologic principles allow determination of the primary abnormality and the amount of compensation. Then starting in the right direction with appropriate
Table 7  Criteria of Acidemia and Alkalemia

<table>
<thead>
<tr>
<th>Acidemia</th>
<th>Alkalemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>pH</td>
</tr>
<tr>
<td>&lt; 7.36</td>
<td>&gt; 7.44</td>
</tr>
<tr>
<td>P&lt;sub&gt;2&lt;/sub&gt;CO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>&gt;45 (respiratory)</td>
</tr>
<tr>
<td>HCO&lt;sub&gt;3&lt;/sub&gt;-</td>
<td>&lt;24 (metabolic)</td>
</tr>
</tbody>
</table>

1 Compensatory or mixed acid-base patterns are common.

Table 8  Major Causes of Metabolic Acidosis

According to Mechanism and Anion Gap (AG)

<table>
<thead>
<tr>
<th>Mechanism of acidosis</th>
<th>Increased AG</th>
<th>Normal AG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased acid production</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactic acidosis</td>
<td></td>
<td></td>
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<tr>
<td>Ketoacidosis</td>
<td></td>
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<tr>
<td>Diabetes mellitus</td>
<td></td>
<td></td>
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<tr>
<td>Starvation</td>
<td></td>
<td></td>
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<tr>
<td>Alcohol-associated Ingestions</td>
<td></td>
<td></td>
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<tr>
<td>Methanol</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td></td>
<td></td>
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<tr>
<td>Aspirin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toluene (if early)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of bicarbonate or bicarbonate precursors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhea or other intestinal losses (e.g., tube drainage),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Type 2 (proximal), renal tubular acidosis (RTA), Posttreatment of ketoacidosis Toluene ingestion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ureteral diversion (e.g., ileal loop)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased renal acid excretion</td>
<td>Chronic renal failure</td>
<td>Some cases of chronic renal failure</td>
</tr>
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</tbody>
</table>

Source: Post TW, Rose BD. Approach to the patient with metabolic acidosis. In: UpTo Date, Rose BD (ed), January 8, 1999 Wellesley, MA.
therapeutic strategies and rechecking the ABG’s, is adequate for managing most cases of acid-base disturbance. Calculating the anion gap will be useful in considering the differential diagnosis of metabolic acidosis (See Table 8).

**Pulse Oximetry**

Pulse oximetry is a non-invasive method of measuring the oxygen saturation of the blood. The oxygen saturation is related to the $\text{Pao}_2$ by the oxyhemoglobin dissociation curve (See Figure 11). The oxygen saturation is calculated by shining light through tissue and measuring the light absorption on the other side of the tissue at two specific wavelengths. The tissue most commonly used is the fingertip, although the earlobe is also used. The light source consists of two light-emitting diodes (LEDs) that emit light at known wavelengths, generally 660 nm and 940 nm. These wavelengths are used because oxyhemoglobin and reduced hemoglobin have different absorption spectra at these particular wavelengths. Pulse oximetry adds a technical improvement to previously available oximetry methods by helping to correct for light other tissues can absorb in addition to the light absorbed by the blood. Pulse oximetry assumes that the only pulsatile absorption between the light source and the photo detector is that of arterial blood. Pulse oximetry corrects for absorption characteristic of substances not found in pulsatile blood by separating the pulsatile alternating current component of the absorption signal from the non-pulsatile direct current component. The direct current component represents the absorbency of the tissue bed, including venous blood, capillary blood, and non-pulsatile arterial blood. The alternating current component represents the pulsatile expansion of the arteriolar bed with arterial blood. This results in more accurate measurement of the arterial blood oxygen saturation.
The oxygen-hemoglobin (O\textsubscript{2}-Hb) dissociation curve, relating the partial pressure of oxygen in arterial blood (P\textsubscript{aO\textsubscript{2}}) to arterial O\textsubscript{2} saturation (S\textsubscript{aO\textsubscript{2}}) and to the O\textsubscript{2} content of arterial blood (Ca\textsubscript{o2}). A normal Hb concentration in milliliters per deciliter is assumed. The curve descends steeply below P\textsubscript{aO\textsubscript{2}} values of 50 mm Hg, indicating severely reduced O\textsubscript{2} -carrying capacity of Hb below this P\textsubscript{aO\textsubscript{2}}. The lower line represents O\textsubscript{2} in solution in the blood; the middle line depicts O\textsubscript{2} bound to the Hb at that P\textsubscript{aO\textsubscript{2}}; the upper line shows O\textsubscript{2} bound to Hb plus O\textsubscript{2} dissolved. Note that dissolved O\textsubscript{2} contributes little to Ca\textsubscript{o2} at a P\textsubscript{aO\textsubscript{2}} in the normal range.

C. Arterial Blood Gases (ABG’s) and Pulse Oximetry

(continued)

The Uses of Pulse Oximetry

The measurement of oxygen saturation can be useful in a variety of clinical circumstances. There are a number of limitations based upon the technical aspects of the measurement. The major conceptual limitation, however, has to do with understanding the oxyhemoglobin dissociation curve and the limitation that oxygen saturation may not reflect important changes in $p_{a}O_2$. Most oximeters claim a 95% confidence limit of ±4% for $s_aO_2$ readings above 80%. In fact, when the $s_aO_2$ is greater than 90%, the accuracy in clinical tests has been somewhat better than ±4%. However, if we use the 95% confidence limit of ±4%, an oximetry reading of 95% could present a $p_{a}O_2$ ranging between 60 mm Hg ($s_aO_2 = 91\%$) and 160 mm Hg ($s_aO_2 = 99\%$). Even if the accuracy was better than 4%, 95% confidence limit, an oxygen saturation that occurs in the flat part of the oxhemoglobin dissociation curve will not give information about relatively large changes in $p_{a}O_2$. These changes could be physiologically and clinically important. The major clinical applications of pulse oximetry are presented in Table 9.

Limitations of Pulse Oximetry

In addition to the issue of accuracy which was discussed above, there are several clinical conditions listed in Table 10 which may affect the accuracy of pulse oximetry measurements. Abnormal hemoglobins can give false readings. The two most common clinical anomalous hemoglobins which affect pulse oximetry readings when present in significant amounts are carboxyhemoglobin (COHb), and methemoglobin (MetHb). The presence of carboxyhemoglobin (such as might occur in accidental intoxication or even in heavy smokers) causes consistent overestimation of the true $s_aO_2$. Methemoglobin causes the pulse oximetry to falsely read approximately 85%. The treatment of methemoglobinemia is the administration of methylene blue. Although methylene blue reverses the clinical signs of methemoglobinemia, including cyanosis, it also causes falsely low pulse oximetry readings, and can cause the reading to go as low as zero.
Table 9  Major Clinical Applications of Pulse Oximetry

1. Continuous monitoring of oxygen saturation in the clinic as either a screen for lung disease that might cause oxygen desaturation or monitoring patients with known lung disease.

2. Determining the oxygenation response to exercise in an outpatient setting. Walking the patient in the clinic and determining if oxygen desaturation occurs, gives extremely important clinical information.

3. Continuous monitoring of hospitalized patients for episodes of oxygen desaturation. This can be useful in a variety of pulmonary conditions, including a screening evaluation for patients with significant sleep apnea.

4. Titration of oxygen therapy in the hospitalized patient or outpatient setting. In patients receiving mechanical ventilation, F\textsubscript{1}\textsubscript{O}\textsubscript{2} can be reduced as the patient improves. By following pulse oximetry, the need for multiple ABG measurements can be reduced.

Table 10  Limitations of Pulse Oximetry

<table>
<thead>
<tr>
<th>Limitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Limited information on ( \text{PaO}_2 ) changes on the flat portion of the oxyhemoglobin dissociation curve.</td>
</tr>
<tr>
<td>No information on acid-base status</td>
</tr>
<tr>
<td>Spurious data with anomalous hemoglobins</td>
</tr>
<tr>
<td>COHb causes overestimation of ( \text{SaO}_2 )</td>
</tr>
<tr>
<td>MetHb causes ( \text{SaO}_2 ) reading of ( \sim 85% )</td>
</tr>
<tr>
<td>Sickle cell anemia with venoocclusive crisis causes</td>
</tr>
<tr>
<td>underestimation of ( \text{SaO}_2 )</td>
</tr>
<tr>
<td>Blue, green and black nail polishes cause falsely low readings</td>
</tr>
<tr>
<td>Dark pigmented skin may cause technical problems with inaccurate readings</td>
</tr>
<tr>
<td>Low perfusion state may cause difficulty in obtaining an interpretable signal</td>
</tr>
</tbody>
</table>
Anemia theoretically might affect the accuracy of pulse oximetry readings. While a study of patients with severe anemia but with normal oxygenation found pulse oximetry to be accurate in this situation, the combined effect of anemia and hypoxemia has not been studied. One study suggested that pulse oximetry underestimates oxygen saturation in patients with sickle cell anemia presenting with a venoocclusive crisis. Pulse oximetry has been shown to be generally accurate in patients with hyperbilirubinemia.

Some nail polishes can cause inaccurate readings. Blue, green and black nail polish have been shown to cause falsely low pulse oximetry readings, although no distortions were seen with red and purple nail polishes. This problem can be alleviated by mounting the oximetry probe side-to-side on the finger.

Darkly pigmented skin is a possible cause of an inaccurate reading. One study showed that technical problems in obtaining an interpretable reading occurred much more frequently in black patients than in whites.

Low perfusion states can make it difficult for the oximetry sensor to distinguish the true signal from background noise and can limit the usefulness of pulse oximetry in critically ill patients.

Finally, pulse oximetry alone, without an internal ABG measurement, might give the clinicians false reassurance in some critically ill patients (such as those with acute respiratory failure and COPD) when oxygen saturation might be satisfactory, but abnormalities of pH and paco₂ might be present. When any doubt exists about the accuracy of oximetry, an ABG is needed.


D. Chest X-rays

The chest x-ray is over a hundred years old and yet remains the cornerstone of chest imaging. The presence of abundant air in the lungs provides many air-fluid interfaces in the chest that allow reasonably precise definition of many important thoracic structures, and provides an opportunity to examine the alveolar tissue of the lung for infiltrative and nodular abnormalities. The chest x-ray initially provided a much-needed breakthrough in tuberculosis management. The chest x-ray remains the initial study of choice in anyone presenting with significant chest symptoms and should always be obtained before considering more advanced chest imaging such as computed tomography (CT) scanning.

Reading abnormalities on chest x-rays requires a basic knowledge of normal thoracic structures and the basic principles of chest radiography. An air-fluid interface defines a border or contour, such as a heart border, or hemidiaphragm contour. On a normal chest x-ray, one sees two sharply defined hemidiaphragms extending centrally nearly to the spine, left and right heart borders, the aortic knob on the left and the hilar structures. Absence of the normal contours occurs as a result of a fluid density such as effusion, infiltrate or atelectasis obliterating a normal contour, the so-called “silhouette sign.” Absence of lung markings occurs as a result of bullous lesions or pneumothorax. Infiltrates in the lungs are the result of fluid density (e.g., white cells, edema fluid, blood or fibrosis) in or around the alveoli. Alveolar infiltrates are characterized by air bronchograms and the tendency of the infiltrates to coalesce and silhouette out adjacent structures. Interstitial infiltrates are characterized by fine lines and dots (reticulo-nodular pattern) and often accentuate borders of adjacent structures. Patterns of infiltrates and evolution of infiltrates over time are important in differential diagnosis (See Table 11).

(continued)
Table 11 Diagnosis From Patterns and Evolution of Infiltrates

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Pattern</th>
<th>Resolution Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Edema</td>
<td>Diffuse alveolar/interstitial</td>
<td>Hours to days</td>
</tr>
<tr>
<td>Alveolar Hemorrhage</td>
<td>Diffuse alveolar</td>
<td>3 to 5 days</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>Segmental, lobar, or whole lung</td>
<td>Hours to days</td>
</tr>
<tr>
<td>Infectious Pneumonia</td>
<td>Segmental to diffuse alveolar</td>
<td>10 to 30 days</td>
</tr>
<tr>
<td>Pulmonary Fibrosis</td>
<td>Diffuse interstitia</td>
<td>Persists indefinitely</td>
</tr>
</tbody>
</table>

D. Chest X-rays (continued)

Chest X-ray Screening

The “routine” or “screening” chest x-ray was an important part of tuberculosis surveillance and control in the first sixty years of the twentieth century. More recently, we have rejected the concept and practice of “routine” procedures and try to logically select appropriate indications and intervals for chest radiographs. There are no systematic data about the utility of “routine” chest x-rays in American adults. Arguably, the best practice is to obtain such studies only on the basis of cardiorespiratory symptoms or special risk factors. Preoperative chest x-rays prove useful as a baseline comparison in patients who might develop postoperative respiratory problems. Preoperative chest x-rays should be obtained in older patients, those with significant cardiopulmonary problems and those undergoing extensive abdominal procedures.

Studies of the utility of chest x-rays for lung cancer screening fail to support a yearly chest x-ray in smokers. However, we recommend a yearly chest x-ray in high-risk patients with 30 pack-years of smoking, as a chest x-ray is our only widely employed lung cancer screening tool. Virtually all early-stage, asymptomatic lung cancers are discovered on “routine” chest x-ray or serendipitously during x-ray investigation of another problem. Early diagnosis and more successful treatment of lung cancer, the most common fatal cancer, remains one of our greatest challenges and will require selective application of newer technologies (CT scanning or sputum cytology) in high-risk patients to achieve success.
Is it Pneumonia or Bronchitis?

Clinicians are faced with patients with acute respiratory symptoms on a daily basis. A decision whether to obtain a chest x-ray must be made in every case. COPD patients with acute bronchitis are at higher risk for serious sequelae. They are generally sicker than are bronchitis patients without chronic lung disease. Pneumonia rather than bronchitis is more probable with increasing numbers and severity of respiratory symptoms. Temperature above a hundred degrees Fahrenheit, new or increased shortness of breath, purulent sputum production, pleuritic chest pain and shaking chills can be signals of pneumonia. COPD patients with milder exacerbations may not require a chest x-ray, but an x-ray is indicated for those requiring hospital admission, both to exclude the presence of pneumonia, congestive heart failure and pneumothorax, and to provide a baseline if improvement is not rapid. Patients with normal lung function and clinical signs and symptoms of pneumonia should have a chest x-ray to define the absence or presence of pneumonia and its extent, if present. Patients with more extensive pneumonia (i.e. greater than one lobe involved) either require admission or close follow-up to assure that progression to generalized involvement with respiratory failure does not occur in an uncontrolled setting.

Since the chest x-ray may suggest the presence of cancer as a cause of the pneumonia, the x-ray should be carefully examined for mass lesions, and hilar or mediastinal adenopathy. Bronchoalveolar cell carcinoma usually presents as a chronic non-resolving pulmonary infiltrate and can mimic infectious pneumonia.

Chest x-rays may reveal complications of pneumonia such as pleural effusion or lung abscess. Significant pleural effusion occurs in at least 20% of pneumonias. Signs of effusion can include silhouetting out of a hemidiaphragm, ground glass opacity at the lung base
and fluid density with meniscus in the lower lung zone. Thoracentesis is indicated for large effusions, if response to therapy is not rapid, and particularly if the effusion increases in size with therapy. Examination and culture of pleural fluid can yield specific microbiology and cytology information pertinent to the etiology of the pneumonia and effusion. Pleural ultrasound examination is more useful than a lateral decubitus x-ray in determining the presence, size and location of a pleural effusion, and is the preferred procedure when available. Early drainage of complicated parapneumonic effusion can prevent formation of loculated empyema.

Lung abscess should always be followed to resolution in order to assure that the abscess is not a cavitary carcinoma. Most patients with lung abscess should have a chest CT scan to size the cavity and to look for other signs of malignancy. Bronchoscopy may be indicated in patients with lung abscess to exclude malignancy and foreign body. Since simple infectious lung abscesses usually result from aspiration of periodontal debris in the edentulous patient, the appearance of a cavitary process with an air-fluid level may well represent a bronchogenic carcinoma with central necrosis.

The chest x-ray is abnormal in as many as 40% of older patients presenting with isolated dyspnea, and is an important part of the initial evaluation of all patients with significant shortness of breath. Significant findings include hyperinflation and bullous changes, interstitial infiltrates, pleural effusion, cardiomegaly and congestive heart failure. A normal study often suggests the need for more specialized testing, such as detailed pulmonary function testing, thromboembolic work-up or cardiology evaluation.
Pulmonary Embolus (PE)

The chest x-ray typically reveals subtle, non-specific abnormalities in patients with PE. Non-specific findings include atelectasis, subtle elevation of a hemidiaphragm, pleural effusion and cardiomegaly. More specific findings include segmental or lobar oligemia (Westermark’s Sign) and peripheral wedge-shaped infiltrate with rounded apex (Hampton’s Hump). A normal chest x-ray in the dyspneic patient also should raise the possibility of PE. However, these findings are rarely distinctive enough to be clinically helpful. They often are recognized only in retrospect after more specific testing enables a diagnosis of PE.

Cardiovascular Abnormalities

The chest x-ray may reveal an enlarged cardiac silhouette due to cardiac dilation or pericardial effusion. A prominent, muscular-appearing left ventricular contour suggests hypertrophy due to hypertension or aortic valvular stenosis. Mitral stenosis is suggested by straightening of the left heart border due to enlargement of the left atrial appendage, by a normal to small heart size, and by pulmonary vascular engorgement with upper zone redistribution of flow. Marked non-specific cardiomegaly is typical of end stage cardiomyopathy, regardless of its cause. Enlargement of the central pulmonary vasculature suggests primary or secondary pulmonary hypertension. Kerley-B lines indicate interstitial edema due to heart failure or lymphatic engorgement from lymphangitic tumor spread to the lung. Ectasia of the thoracic aorta is common in the elderly, but focal dilation suggests atherosclerotic aneurysm or dissection of the aorta. Interstitial fibrosis can be easily confused with congestive heart failure. Often more detailed testing is required to differentiate between the two.

(continued)
### Anterior mediastinum
- Thymoma
- Germ cell tumors
- Lymphoma
- Thyroid/parathyroid tumors
- Mesenchymal tumors

### Middle mediastinum
- Benign and malignant lymphadenopathy
- Lymphoma
- Developmental cysts
- Vascular aneurysms
- Hiatal hernia

### Posterior mediastinum
- Neurogenic tumors
- Esophageal carcinoma
- Esophageal diverticulum

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**Table 12  Mediastinal Masses by Location**

Pneumothorax Versus Bullous Lung Disease

The diagnosis of pneumothorax is made reliably with standard x-rays in patients without intrinsic lung disease. In patients with extensive bullous lung disease and acute dyspnea, it may be difficult to differentiate pneumothorax from large intraparenchymal bullous lesions. Placement of a chest tube into a bullous lesion can be catastrophic. Caution is advised in the evaluation of possible pneumothorax in COPD patients. Chest CT scan may be required for a certain diagnosis.

Mediastinal and Hilar Adenopathy and Masses

Asymptomatic bilateral hilar adenopathy is usually due to sarcoidosis. Unilateral hilar adenopathy is due more often to tuberculosis or benign or malignant tumors. The presence of unilateral hilar and mediastinal adenopathy suggests unresectability in lung cancer patients. However, further staging with CT imaging and biopsy is required in most such patients. Other mediastinal masses are classified according to their location in the anterior, middle or posterior mediastinum (See Table 12).
References


Kerr IH, Simon G, Sutton GC. The value of the plain radiograph in acute massive pulmonary embolism. Br J Radiol 1971;44:751-757. X-ray abnormalities are common in massive PE, but are non-specific and often apparent only in retrospect.

Muller NL. Imaging of the pleura. Radiology 1993;186:297-309. This paper offers a good discussion of the x-ray diagnosis of pleural effusion.


Multiple imaging techniques to better assess pulmonary anatomy, physiology and pathology are now available. Before any of these more advanced techniques are used, a posterior-anterior (PA) and lateral chest x-ray should be obtained to give an overview and to direct the order in which other studies should be obtained. Imaging techniques to be discussed include:

1. Computed tomography (CT)
   a. CT (with and without contrast)
   b. High resolution CT (HRCT)
   c. Spiral CT
   d. Low radiation-dose CT
2. Ventilation-perfusion lung scanning (V/Q scan)
3. Diagnostic ultrasound
4. Positron emission tomography (PET)
5. Magnetic resonance imaging (MRI)
6. Pulmonary angiography

Physicians need to be aware of the local radiology department’s areas of expertise as this too will influence the type and sequence of the additional studies chosen. A summary of clinical indications for each study is presented in Table 13.

Computed Tomography (CT)

The conventional CT scan gives an excellent anatomical picture of the thorax and its structures. The addition of intravenous contrast allows blood vessels and other vascular structures to be differentiated from non-vascular structures such as mediastinal nodes, masses and soft tissue densities. Serial coronal images are taken every 7 mm to 10 mm from the apices to the bases of the lungs. This may be visualized as slicing the lungs as a loaf of bread. Each cross-sectional image is then laid out for inspection. Nodules, masses, infiltrates, atelectasis, pleural effusions and lymphadenopathy are identified in much greater detail than with the chest x-ray. When more specialized versions of the CT scan, (See below), are employed without the basic CT scan, the above
Table 13  Imaging Techniques

<table>
<thead>
<tr>
<th>Imaging Technique</th>
<th>Clinical Indication for Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest x-ray</td>
<td>All patients should have a chest x-ray prior to other imaging techniques.</td>
</tr>
<tr>
<td>CT with contrast</td>
<td>Evaluate lung masses, nodules, hilar and mediastinal structures (including aorta), pleural disease and pulmonary infiltrates.</td>
</tr>
<tr>
<td>CT without contrast</td>
<td>Use if patient is allergic to contrast and for follow-up of lung nodules.</td>
</tr>
<tr>
<td>High resolution CT</td>
<td>Evaluate parenchymal lung disease such as interstitial lung disease, bronchiectasis and emphysema.</td>
</tr>
<tr>
<td>Spiral CT with contrast</td>
<td>Evaluate for acute and chronic thromboembolic disease in patients with non-diagnostic lung scans, abnormal chest x-ray or chronic lung disease.</td>
</tr>
<tr>
<td>Low radiation-dose CT</td>
<td>Screen high-risk patients (smokers with airflow obstruction), for lung cancer.</td>
</tr>
<tr>
<td>Ventilation-perfusion lung scan (V/Q scan)</td>
<td>Current standard for evaluating patients with normal chest x-ray for thromboembolic disease</td>
</tr>
<tr>
<td>Diagnostic ultrasound</td>
<td>Evaluate and localize pleural effusions. Evaluate lower extremities for deep vein thrombosis.</td>
</tr>
<tr>
<td>Positron emission tomography (PET)</td>
<td>Consider after chest x-ray and CT with contrast to determine likelihood of malignancy in a mass or nodule, to stage lung cancer, to evaluate cancer therapy and recurrence of malignancy.</td>
</tr>
<tr>
<td>Magnetic resonance imaging (MRI)</td>
<td>Rarely used for imaging the thorax.</td>
</tr>
<tr>
<td>Pulmonary angiogram</td>
<td>Traditional standard for diagnosis of PE when other studies are non-diagnostic.</td>
</tr>
</tbody>
</table>
abnormalities can be missed. Once an abnormality such as a solitary pulmonary nodule is identified, 1 mm to 2 mm sections through the lesion should be obtained. Abnormalities can be evaluated for calcification, density, contrast enhancement, cavitation and vascular supply.

**High Resolution CT Scan (HRCT):** This technique provides extreme detail of the lung parenchyma and is particularly helpful in diagnosing interstitial lung disease and emphysema. While this technique gives an excellent anatomical evaluation of the lung, it does not replace the need for lung biopsy in most patients with interstitial lung disease.

**Spiral CT Scan:** The spiral (or helical) CT technology allows for direct visualization of the pulmonary vasculature and is an excellent tool for assessing patients for suspected acute and chronic thromboembolic disease. In contrast to conventional CT scans which produce a single axial image, the spiral technique results in a helical volume of data over the entire thorax, in seconds. This allows for images to be reconstructed at overlapping intervals to further improve resolution. Rather than viewing static images, radiologists are able to scan the software computer images in a dynamic fashion. Thus, they may identify emboli or thrombi at the segmental level of the lung. Patients with suspected pulmonary emboli (PE) and abnormal chest x-rays should have a spiral CT instead of a V/Q lung scan. However, this technique has been found to lack sensitivity for small subsegmental emboli. Nonetheless, with recent improvements in equipment and experience, this technique may become the imaging technique of choice for all patients suspected of having acute thromboembolism.

**Low Radiation-dose CT Scan:** This method is deemed most appropriate for serial studies to screen for bronchogenic carcinoma. Individuals at high risk of
developing lung cancer (smokers with airflow obstruction) are excellent candidates for this study. The spiral (helical) CT technique is used. Thin sliced images through the thorax are obtained. Nodules and other abnormalities are identified much sooner than with chest x-rays, and appropriate management (observation or biopsy) can be initiated. This technique results in lower doses of radiation exposure compared to other CT studies. Guidelines for its use are under current development.

**Ventilation-Perfusion Lung Scan (\(\dot{V}/\dot{Q}\) Scan):**

For years, \(\dot{V}/\dot{Q}\) scans have been the most commonly used initial study to diagnose pulmonary embolism (PE). But, unless the lung scan is normal (excluding PE), or high probability in conjunction with high clinical suspicion, the studies are regarded as equivocal or non-diagnostic. In the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study, 65% to 70% of patients had non-diagnostic scans. Combining \(\dot{V}/\dot{Q}\) scans with lower extremity ultrasound or impedance plethysmography may improve the yield. But, until spiral CT was available, pulmonary angiography was necessary to confirm PE. Individuals with chronic lung disease and those with abnormal chest x-rays will have abnormal lung scans, usually non-diagnostic, limiting the usefulness of \(\dot{V}/\dot{Q}\) scanning in these patients. Thus, the diagnostic algorithm for PE seems to be changing. Spiral CT imaging (when available) is becoming the diagnostic study of choice in PE suspects with abnormal chest x-rays or chronic lung disease.

Quantitative \(\dot{V}/\dot{Q}\) scanning is helpful to determine differential lung function and is frequently used in patients with severe underlying lung disease requiring pulmonary resection. This provides data on the percentage of blood flow and ventilation to each lung, helping to stage for operability.
**Diagnostic Ultrasound**

The best pulmonary use of ultrasound is to evaluate pleural effusions and to localize optimal location for thoracentesis or chest tube placement. Because of its portability, the study can be done at the bedside.

Lower extremity ultrasound with color Doppler allows for the assessment of acute deep venous thrombosis, flow abnormalities and chronic venous insufficiency. This non-invasive technique is extremely helpful in the evaluation of lower extremity pain and swelling.

**Positron Emission Tomography (PET)**

Positron emission tomography (PET) is a metabolic imaging technique that uses amino acids or glucose to which positron emitting radionuclides are tagged. For thoracic PET scanning, the radiopharmaceutical, F-2-deoxy-D-glucose (FDG) is used and is labeled with fluorine-18. Because malignant cells metabolize glucose at a much higher rate than non-malignant cells, FDG injected intravenously is preferentially accumulated in malignant cells. This is new technology, and guidelines have not been firmly established for its use. When combined with other imaging techniques, we anticipate that it may be helpful in evaluating localized pulmonary lesions, characterizing hilar and mediastinal adenopathy, staging of lung cancer, evaluating recurrence of disease and measuring treatment response. False-positives may be seen with active granulomatous infection, such as seen in the Midwest (histoplasmosis) and Southwest (coccidiodomycosis).

**Magnetic Resonance Imaging (MRI)**

While MRI has revolutionized the diagnostic approach in the orthopedic and neurologic specialties, its use in the thorax remains limited. Because of the continuous cardiac and respiratory motion, images are not as clear as in CT or x-rays. In general, the chest x-ray and CT scan provide more useful data. Magnetic resonance imaging (MRI), of the thoracic aorta is an accurate method for diagnosing aortic dissection, but spiral CT is
Pulmonary angiography remains the final definition test for the diagnosis of PE. Numerous diagnostic algorithms exist for diagnosing this disorder. But, when \( V/Q \) scanning, spiral CT, venous ultrasound or metabolic markers such as D-dimer fail to confirm a diagnosis, pulmonary angiography is utilized. Because it is more invasive and inherently hazardous, pulmonary angiography should not be used first in the evaluation for PE (See \( V/Q \) scanning and spiral CT, above).
References


F. Ultrasound Procedures

The Echocardiogram

The echocardiogram is one of the most widely used and useful techniques in the evaluation of the patient with cardiopulmonary symptoms. It is non-invasive. A normal echocardiogram excludes a broad range of potential cardiac problems. Newer techniques allow direct imaging of cardiac structures, generally accurate determination of various cardiac pressures and more sensitive imaging of valvular abnormalities. Unfortunately, lung interference often limits the quality of the echocardiogram in patients with COPD. Table 14 summarizes available widely used echocardiographic techniques.

Evaluation of Cardiac Murmurs

The echocardiogram is helpful in the diagnosis of cardiac murmurs and their severity and is performed as the initial evaluation in most patients felt to have a significant murmur. Abnormalities in aortic stenosis include bicuspid valve, thickening of valve leaflets, decreased leaflet excursion and increased pressure gradient across the valve. In aortic regurgitation, the echocardiogram provides an estimate of regurgitant flow, and may show premature closure of the mitral valve in severe aortic valve regurgitation. In mitral stenosis, the echocardiogram shows thickening and decreased excursion of the valve leaflets, left atrial enlargement and provides an estimate of the valve area. Findings in mitral insufficiency include regurgitant jet into left atrium, abnormalities of the chordae and papillary muscle, and mitral valve prolapse.

Evaluation of Cardiac Failure

The echocardiogram provides an accurate estimate of left ventricular volume, contractility and ejection fraction in most patients. It can reveal segmental wall motion abnormalities that correspond to coronary anatomy and suggest ischemic cardiomyopathy. Diffuse hypokinesis is seen in non-ischemic congestive cardiomyopathy. Interventricular and interatrial septal defects can be visualized and their diagnosis is enhanced with bubble contrast and transesophageal techniques. The echocardiogram may reveal surprisingly good ventricular function and define unexpected valvular
### Table 14  Echocardiographic Techniques and Their Advantages

<table>
<thead>
<tr>
<th>Echocardiographic Technique</th>
<th>Characteristics</th>
<th>Advantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>M-Mode (Motion-mode)</td>
<td>Visualizes motion of cardiac structures by plotting one dimensional “ice-pick” view of heart against time.</td>
<td>Good visualization of mitral and aortic valve pliability and motion; accurate sizing of left atrial and ventricular dimensions.</td>
</tr>
<tr>
<td>Two-Dimensional (2-D)</td>
<td>Ultrasound beam continuously sweeps a pie-shaped sector.</td>
<td>Direct, spatially correct visualization of cardiac structures and motion.</td>
</tr>
<tr>
<td>Doppler</td>
<td>Uses Doppler shift principle to determine velocity of blood flow and estimation of pressures.</td>
<td>Good estimation of valvular gradients and intracardiac pressures.</td>
</tr>
<tr>
<td>Color flow Doppler</td>
<td>Gated Doppler displays spatially correct, colored images of moving blood superimposed on 2-D images.</td>
<td>Dynamic, spatially correct visualization of abnormal intracardiac blood flow.</td>
</tr>
<tr>
<td>Transesophageal</td>
<td>2-D probe passed into the esophagus, allows placement of transducer in close proximity to heart.</td>
<td>Better visualization of valvular structures/vegetations and central, massive VTE. Excellent visualization of thoracic aortic disease and cardiac shunt defects.</td>
</tr>
</tbody>
</table>
abnormalities that explain cardiac dysfunction. Stress echocardiography demonstrates inducible ventricular contraction abnormalities in patients with active ischemia. The echocardiogram is essential to exclude pericardial effusion in patients with non-specific “cardiomegaly” on chest x-ray.

Findings in right ventricular dysfunction are similar to those in left ventricular dysfunction, but Doppler estimates of right ventricular and pulmonary artery pressure are often inaccurate, and right heart catheterization is frequently required to precisely define pressures in patients with suspected primary pulmonary hypertension. Echocardiography is useful in evolving pericardial effusions and with evidence of tamponade or pericardial restriction.

Frontline practitioners often see patients with dyspnea that remains unexplained after initial evaluation including physical examination, chest x-ray, pulmonary function testing and the electrocardiogram. The echocardiogram may be an appropriate next test to search for unexpected left or right heart disorders.

Diastolic dysfunction is a form of “functional” inflow obstruction to the left ventricle, with the stiff left ventricle causing decreased passive diastolic filling ("E"-wave) and an increased atrial wave ("A"-wave) seen in the mitral inflow velocity wave forms. Diastolic left ventricular dysfunction can be worsened by such stresses as volume overload, exercise-induced ischemia and worsening of pulmonary status in patients with chronic lung diseases. Examination of mitral inflow velocity patterns is an important part of the echocardiographic evaluation of any patient with unexplained dyspnea.

Unexpected right ventricular hypertrophy, dilation, tricuspid valve regurgitation and suggestion of pulmonary hypertension may be found on examination of the right heart.
A normal echocardiogram study excludes most major cardiac dysfunction. Further evaluation including stress testing or catheterization may be required to exclude myocardial ischemia as a cause of dyspnea.

In most patients suspected of PE, other studies enumerated above are performed for diagnosis. However, for patients who are too unstable to be transported to radiology or as an emergent screening technique while awaiting other studies, the transthoracic echocardiogram may be suggestive of PE, showing right ventricular dilation and pulmonary hypertension. Rarely, clot in the right ventricle can be visualized. Paradoxical ventricular septal motion is reportedly a sign of massive emboli with acute right ventricular pressure overload. Occasionally, the technique allows direct imaging of large clots in the central pulmonary arteries.

Patients presenting with shock and no obvious source such as ruptured abdominal aneurysm or gastrointestinal sepsis, often benefit from echocardiography. Transthoracic echocardiography may reveal severe left ventricular dysfunction, pericardial tamponade, acute mitral or aortic regurgitation, hyperdynamic ventricle due to hypovolemia or sepsis or findings suggestive of massive PE. Transesophageal echocardiography is a more sensitive test for thoracic aortic dissection, posterior mitral valve papillary muscle rupture, thoracic aortic dissection, valvular vegetations from endocarditis, central PE and prosthetic valve dysfunction.

Duplex ultrasound is widely used in the diagnosis of venous thrombosis. The technique uses B-mode scanning to evaluate the veins of the legs for patency, blood flow and the presence of thrombi. Thrombi are most reliably diagnosed using the criteria of non-compressibility. Another criterion is abnormal or
absent flow sounds. Echogenicity of clot is less reliable as acute clots are frequently non-echogenic.

Duplex ultrasonography is sensitive (~93%) and specific (~95%) for the diagnosis of proximal leg vein thrombosis (popliteal and femoral veins) in symptomatic patients. The technique is less sensitive for the diagnosis of calf vein thrombosis (~70%) and proximal vein thrombosis in asymptomatic patients (~50%). Serial studies are useful to detect proximal extension of calf vein thrombosis.

Duplex ultrasonography is often the first test in patients with symptoms and signs of thrombophlebitis in the legs. The procedure is also useful in patients with suspected PE, as it increases the likelihood of PE, when positive.

Pleural Ultrasound Examination

Pleural ultrasound is not necessary for thoracentesis of large volume pleural effusions. However, it allows precise localization of small effusions, differentiates extra- and intrapulmonary mass lesions from pleural fluid, helps define loculations and guides optimal location for catheter drainage of complicated effusions. For aspiration of pleural collections, it is preferred that the thoracentesis be performed at the time of ultrasonography since skin marks and fluid can shift with change in patient position and result in an unsuccessful procedure.
References


Gelernt MD, Mogtader A, Hahn RT. Transesophageal echocardiography to diagnose and demonstrate resolution of an acute massive pulmonary embolus. Chest 1992;102:297-299. Case report and review of transesophageal echocardiographic diagnosis of massive PE. In addition to findings of pulmonary hypertension, the technique can provide direct visualization of a central clot.


G. Cardiac Catheterization

Cardiac catheterization is a procedure, or, more accurately, a group of procedures, in which catheters are introduced into the heart through veins or arteries to provide information about cardiovascular anatomy and function. Although cardiac catheterization is relatively safe, it does carry some risks and expense. Therefore, as with any procedure, the risk/benefit ratio must be considered before recommending it. The benefit usually consists of important information which will guide a therapeutic decision. The question, “How will the obtained data change the therapeutic plan?” is always appropriate in considering whether or not to recommend cardiac catheterization.

Indications

Cardiac catheterization can confirm the presence of a clinically suspected condition, define its anatomic and physiologic severity and determine whether important associated conditions are present. It is usually indicated when the patient is experiencing increasing or significant symptoms of cardiac dysfunction or when other studies indicate a high risk for development of a significant complication such as a myocardial infarction. Cardiac catheterization is usually indicated to define the anatomy when a cardiac surgical procedure is being considered. Exceptions include when decisions regarding cardiac surgery can be made by clinical examination and non-invasive studies such as echocardiography (See Section F). Clinical examples might include uncomplicated patent ductus arteriosus or atrial septal defect.

Complications and Relative Contraindications

The mortality rate associated with diagnostic cardiac catheterization is quoted as being 0.1% or 1 in 1,000. Complications include myocardial infarction, perforation of the heart and great vessels, ventricular tachycardia or fibrillation, atrial arrhythmias or A–V block, thromboembolic complications including stroke, bleeding, infection and hypersensitivity reaction to contrast agents.
Relative contraindications include uncontrolled ventricular irritability, hypokalemia, digitalis toxicity, uncorrected hypertension, decompensated heart failure (especially acute pulmonary edema), anticoagulated state with an elevated prothrombin time, known severe allergy to radiographic contrast agents and severe renal insufficiency. The risk of complications with several of these relative contraindications can be reduced by treatment. In addition to correction of metabolic and physiologic abnormalities, this can include changing from an oral anticoagulant to heparin to facilitate rapid reversal, if necessary, or dialysis to remove fluid and lessen the risk of radiographic contrast load in the presence of renal insufficiency.

Catheters can be introduced through a brachial artery or vein, the subclavian or internal jugular veins, or a femoral artery or vein. Catheterization of either the right or the left heart can be accomplished depending on the suspected condition and the desired information. With the catheter in the heart or great vessels, pressure measurements as well as measurements of flow (cardiac output), can be obtained. Cardiac output can be calculated or measured using the Fick principle:

\[
\text{Cardiac output} = \frac{\dot{V}_O_2}{\text{Arterial O}_2 \text{ content} - \text{mixed venous O}_2 \text{ content}}
\]

Cardiac output can also be measured using indicator–dilution techniques or angiography. Angiography using contrast agents can define the anatomy of the coronary arteries, left ventricle or aorta. Angiography can also confirm valvular regurgitation and the ventricular ejection fraction. Pressure measurements, including determination of pressure gradients across valves, can help define valvular stenosis and regurgitation, pulmonary hypertension, hypertrophic obstructive
cardiomyopathy, other cardiomyopathies and cardiac tamponade. Cardiac catheterization is usually performed with the patient awake, but sedated (conscious sedation).

Pulmonary Artery (PA) Catheterization

Pulmonary artery (PA) catheterization using a balloon-tipped flow-guided catheter (Swan-Ganz catheterization, named for the cardiologists who developed this technique), deserves special comment. This procedure is generally used in critically ill patients in the Intensive Care Unit (ICU). It may either be done for a short period of time to obtain diagnostic information, or continued for a period of days for the purpose of hemodynamic monitoring and guidance of therapy over time.

By inflating the balloon with the catheter in the pulmonary artery, a segment of the pulmonary vascular system is changed to a state without flow, allowing measurement of an approximation of the pressure in the left atrium, the balloon occlusion PA wedge pressure. The pressure that is actually measured is that at the most proximal point of the occluded pulmonary vascular system where flow is present (See Figure 12) — a value which is very close to left atrial pressure.

A recent large retrospective study suggested that the use of PA catheterization may be associated with increased mortality, even after attempting to control for severity of illness (a difficult task, especially in a retrospective study). This has heightened the controversy over the role of PA catheterization. Currently, a number of studies are underway in North America and Europe investigating the use of PA catheterization and its effect on outcome in a variety of specific clinical circumstances. Pending the results of those studies, a prudent approach is to be particularly critical about the indications for placing such a catheter, the quality of the data being obtained, the clinical therapeutic response to those data and the length of time the catheter remains in
Figure 12  Measuring Pulmonary Artery (PA) Pressure

place. Regarding the last issue, it is useful to ask on a daily basis whether therapeutic decisions are being made on the data being collected. If not, the catheter should be discontinued.

**Indications**

The primary monitoring uses of PA catheterization include the following:

1. To assess the adequacy of intravascular volume. This may be warranted in patients with hypotension, oliguria or the high-risk surgical patient.
2. To assess the effect of changes in the pulmonary wedge pressure on pulmonary edema.
3. To assess therapy for shock. This includes:
   a. vasodilator and inotrope therapy in cardiogenic shock,
   b. volume, vasopressor and inotrope therapy in septic shock and
   c. volume therapy in hypovolemic shock.
4. To assess the effects of positive end-expiratory pressure (PEEP) on cardiac output in adult respiratory distress syndrome (ARDS).

Ensuring high quality data requires a knowledge of the technical aspects of obtaining the data and an understanding of normal physiology and pathophysiology. This subject is beyond the scope of this Section. The interested reader is referred to the reference list which follows. ■
References

Connors AF, Jr., Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. JAMA 1996;276:889-897. This article summarizes the SUPPORT Study paper reporting a higher mortality in critically ill patients receiving a PA catheter.


Sputum analysis is performed to aid with the diagnosis of a variety of respiratory disorders. Broadly speaking, the diseases for which sputum studies have been employed can be classified as infectious, neoplastic or other inflammatory conditions. Appropriate and efficient utilization of sputum studies requires a clear understanding of the indications and limitations of these tests.

Sputum tests have historically been a primary diagnostic tool in pneumonia. Specific methods are noted below.

The Gram Stain: Learned as a medical student, this test has been a sentimental favorite of physicians for years, but its validity in identifying etiologic agents involved in bacterial pneumonia has been challenged. Indeed, recent guidelines of the American Thoracic Society (ATS) and Canadian Thoracic Society (CTS)/Canadian Infectious Diseases Society (CIDS) have emphasized “empiric” treatment for most cases of community-acquired pneumonia (CAP) based on epidemiological and clinical features of the patient. By contrast, the new version of the Infectious Diseases Society of America (IDSA) places more emphasis on sputum Gram stain. All three of the current guidelines stratify the evaluation of CAP by the facilities in which they are to be treated. For patients who are diagnosed in the office or outpatient department and are to be treated at home, the ATS and CTS/CIDS guidelines both advocate empirical therapy without bacteriological studies. However, the IDSA statement makes sputum stain and culture optional. All groups advocate sputum studies for patients whose status requires hospital-based treatment.

To be a valid predictor, the test must be done on a “good specimen” and read by a competent observer. A “good” sputum specimen is denoted by a high number of neutrophils (> 25 per low power field), and a low number of epithelial cells (< 10 per low power field),
with a consistently prominent pathogen (e.g., Gram-positive diplococci, Gram-negative rods, etc.). One might regard sputum Gram stain results in the following manner: a clearly “positive” good specimen is useful in establishing the probability of various bacterial pneumonias, but a negative study does not have strong predictive value.

Special stains may be helpful in detecting “exotic” pneumonias due to mycobacteria, fungi, nocardia, legionella or pneumocystis. Thus, if clinical features, radiographic findings, epidemiological profiles and/or failure to respond to conventional therapy suggest the possibility of an unusual pathogen, “special stains” should be requested. This is particularly relevant to patients with immunosuppression.

Most patients can spontaneously expectorate lower respiratory tract secretions on request. However, for various reasons, some are unable to do so. “Induction” of cough and sputum by inhalation of heated or hypertonic saline via nebulizer may facilitate retrieval of useful specimens in these cases.

**Culture Techniques:** Cultivation of respiratory secretions has variable utility in the diagnosis of pneumonia. Particularly problematic is disease associated with microbes which are part of the normal respiratory flora, i.e., pneumococcus, haemophilus, moraxella, etc. Nonetheless, cultivation may be appropriate to both confirm the presence of the suspected pathogen and to study *in vitro* susceptibility. The combined findings of a Gram stain demonstrating dominant organisms and a culture positive for such organisms are especially useful.

As with microscopy, cultivation of exotic organisms may require specialized methods including selective media, enrichments or incubating practices. Thus, if exotic agents are suspected, specified cultures for
mycobacteria, nocardia, fungi, legionella or chlamydia may be ordered. Routine sputum cultures for anaerobic bacteria are useless due to the inevitable exposure to oxygen in the upper airway.

**Other Sputum Tests for Pneumonia:** In addition to microscopy and culture, a variety of novel methodologies are being used to identify pathogens in sputum. For *M. tuberculosis* and other mycobacteria such as *M. avium* complex or *M. kansasii*, nucleic acid amplification (NAA) techniques have proven to be rapid (24 to 48 hours), and reliable (85% to 95% sensitive, 95% to 100% specific), but are currently recommended only in specimens which are positive on microscopy.

Direct fluorescent antibody tests have proven quite specific (± 90%), but relatively insensitive (± 50%) for *Legionella pneumophila*. Urinary antigen is more sensitive for *L. pneumophila* but is limited to the most common serovar, type 1. Preliminary data suggest a possible role for urinary antigen tests for invasive disease due to pneumococci.

Empirical treatment of CAP has become an endorsed and widely accepted practice in the United States and Canada. This is based on:

1. A high-probability of efficacy with agents such as the newer fluoroquinolones, macrolides or azolides,
2. Mistrust of the accuracy of sputum studies,
3. The observation that even aggressive microbiological and serological testing identifies an etiologic agent in less than 50% of cases, and
4. The desire to curtail expenditures for testing.
The counter arguments, as framed by the IDSA, include this logic in support of microscopy, cultivation or other means to establish a specific etiologic agent:

1. It permits selection of an “optimal” agent against the specific pathogen,
2. Selection of a specific antibiotic will limit costs, the risk of acquired drug resistance and drug toxicity, and
3. There is epidemiological utility in identifying pathogens such as legionella, Hantavirus or penicillin-resistant pneumococci,
   a. careful/improved studies should increase the yield to above 50%,
   b. inability to see/recover typical pathogens should trigger earlier pursuit of atypical pathogens,
   c. the absence of particularly ominous pathogens such as Staphylococcus aureus or Gram-negative rods is reassuring to the clinician and
   d. the average cost of standard microbiological studies is < 1% of the average hospital bill!

Overall, we conclude that empiric choice of antibiotics for CAP in a general adult population to be treated at home is a practical and appropriate strategy. While the arguments posed in the IDSA guidelines are rational, there is no persuasive evidence documenting improved outcomes, lessened drug resistance or cost savings from this approach. However, clinicians should be more aggressive in obtaining respiratory secretions for microscopy, cultivation ± other special studies for patients whose pneumonia is severe enough to merit hospitalization, or those with special risk factors. These might include recent hospitalization, nursing home residency, very poor dental hygiene, exotic exposures (birds, rabbits, rural rodents or travel to the southwest United States), underlying structural abnormalities of the lungs or immunodeficiency status.
Respiratory Tract Neoplasms and Sputum Studies

Cytologic examination of cells exfoliated from the tracheobronchial tree and lung parenchyma has proven highly useful in the diagnosis of cancer involving these tissues.

Sputum cytology is an appropriate test for individuals with significant risk factors for respiratory tract cancer including smoking (≥ 30 pack-years), or exposure to carcinogens including passive tobacco smoke, asbestos or uranium. However, the overall return for screening such populations is too low to be justified. But, if we select for persons with chronic airflow obstruction on simple spirometry, the yield of positive findings may reach 1% to 2%. Such a process appears to result in early diagnosis and improved survival.

Other indications for sputum cytology studies among individuals with other risk factors include an unexplained protracted cough, hemoptysis or suspicious findings on chest radiography. Sputum cytology is more likely to yield positive results among patients with larger or centrally located tumors.

Positive cytology for malignancy should be combined with bronchoscopy (See Section I), computed tomography, as well as careful history and physical examination and other laboratory studies to localize and stage the neoplasm.

Overall, a single cytology is positive in roughly 50% of patients with bronchogenic cancers. Three sputum cytology examinations increase the yield modestly and should be done in high-risk subjects.

Negative cytology results are common in patients with smaller peripheral nodules. One might order cytology studies in such patients hoping that “positive” findings will preclude more invasive studies. If such tests are negative, one must proceed to more aggressive studies.
References


Saccomanno G. Diagnostic Pulmonary Cytology. 2nd ed. Chicago, IL: American Society of Clinical Pathologists Press; 1986. pp 211. A beautiful collection of 90 color plates which illustrate normal and many abnormal cytologic and histologic studies with emphasis on various types of primary and secondary cancers of the lung.
I. Bronchoscopy

Endoscopy of airways is a widely practiced, safe and effective modality of diagnosis and, less often, treatment. The original technique employed a metal, open-tube device, the rigid bronchoscope (RB). Over the past 30 years, this procedure has largely been supplanted by the flexible fiberoptic (FFB) method. The indications for bronchoscopy among patients seen in frontline practice are delineated below and in Table 15.

Indications

Generally, bronchoscopy is performed by pulmonologists or, less frequently, thoracic or general surgeons. The major advantages of the flexible endoscopy include ease of performance under topical anesthesia, ability to directly examine more remote airways and the capacity to pass biopsy forceps or culture brushes into the distal airways and lung parenchyma. The rigid, open-tube device is the instrument of choice for the extraction of larger foreign bodies, evaluation and management of massive hemoptysis, dilation/stenting of stenotic or collapsible airways and for laser or brachytherapy of endobronchial neoplasms.

Complications and Contraindications

The risks of bronchoscopy can be compartmentalized into adverse reactions to the premedications or topical anesthetic agent, mechanical trauma to the airways or lungs, bleeding and infectious or inflammatory sequelae of passage of the instrument and/or instillation of lavage fluids.

Overall, the experience with the FFB technique in the United States has been quite benign. One survey indicated minor complications in 0.2%, and major problems in 0.08% of patients undergoing the procedure. It should be noted though, that these were retrospective, self-reported events. An earlier 1976 series described 12 deaths (0.02%), and “life
### Table 15 Overall Indications for Bronchoscopy

<table>
<thead>
<tr>
<th>Condition/Problem</th>
<th>Instrument Of Choice</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest x-ray suspicious for lung cancer</td>
<td>FFB</td>
<td>Bronchoscopy indicated all lesions except very small, peripheral nodules which cannot be reached.</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>FFB or RB</td>
<td>For minor hemoptysis the FFB allows more thorough visualization of airways, but, for massive bleeding, the RB permits more effective aspiration of blood and airway control.</td>
</tr>
<tr>
<td>Diffuse interstitial or fibronodular lung disorders</td>
<td>FFB</td>
<td>Bronchoscopy with transbronchial lung biopsy and bronchoalveolar lavage is often the first step in the diagnosis of diffuse lung disorders such as sarcoidosis, idiopathic pulmonary fibrosis, etc.</td>
</tr>
<tr>
<td>Cough</td>
<td>FFB</td>
<td>Endoscopy done for chronic cough but a normal chest x-ray has a very low yield. Evaluation to rule out more common causes of cough (sinusitis, esophageal reflux), is indicated prior to bronchoscopy.</td>
</tr>
<tr>
<td>Wheezing/stridor</td>
<td>FFB</td>
<td>“All that wheezes is not asthma”…but most is! Once asthma has been excluded, other entities should be considered, (See Section A). Refractory, chronic localized wheezing should lead to endoscopy. One may either perform serial laryngoscopy, then bronchoscopy, or — if the operator is skilled at assessing both laryngeal and lower airway anatomy and function — a single endoscopy may be adequate to rule out tumors, strictures or foreign bodies.</td>
</tr>
<tr>
<td>Undiagnosed pneumonia, refractory or progressive</td>
<td>FFB</td>
<td>Treatment of community-acquired pneumonia (CAP), is most often empiric, (See Section H). In cases when the patient does not respond to therapy as expected, FFB may be indicated to rule out foreign body or tumor, obtain diagnostic material and, occasionally, to remove inspissated secretions from airways.</td>
</tr>
<tr>
<td>Foreign body</td>
<td>RB</td>
<td>For patients observed or suspected to have aspirated a foreign object into their airways, examination and retrieval is indicated. If the object is large enough or placed in a critical site where airway patency is seriously compromised (wheezing, choking, hypoxia, largospasm), emergent retrieval is indicated. The RB is clearly a superior device in terms of removal of the object and control of ventilation. For small, more remote objects, the FFB may be adequate in the hands of a skilled practitioner.</td>
</tr>
</tbody>
</table>

FFB = Flexible fiberoptic bronchoscopy, R = Rigid bronchoscopy
threatening” cardiovascular events (0.5%) in 48,000 endoscopies. A 1978 report noted major complications in 1.7% of patients and a mortality rate of 0.12%.

The few absolute contraindications to FFB are hemodynamic instability, life threatening arrhythmias and severe hypoxia which in the non-intubated patient, if worsened, might be life threatening. A number of features increase the risks of morbidity and mortality. They include extensive bullous emphysema, severe hypercapnia ($\text{PaCO}_2 > 60$), unstable angina, recent seizure activity, labile asthma, significant coagulopathy/thrombocytopenia and advanced airway narrowing. Obviously, the competency of the operator, the capabilities of support personnel and the adequacy of the facilities used, all influence outcomes. The more ominous sequelae of bronchoscopy include:

1. **Premedications**: hypoventilation, hypotension,
2. Topical anesthetics: laryngobronchospasm, seizures, arrhythmias,
3. **Endoscopy/biopsy/brushing**: bleeding, bronchospasm, hypoxia, arrhythmias, fever, pneumonia and pneumothorax,
4. **Bronchoalveolar lavage (BAL)**: Fever associated with release of cytokines due to perturbation of alveolar macrophages may occur in a substantial portion of those undergoing BAL. Careful observation is required to distinguish this reaction from true infectious pneumonia since BAL typically results in retention of fluid in the air spaces and new shadows on chest x-ray.

**Summary**

Bronchoscopy is a useful and safe tool for diagnosis and management of a variety of airway and lung parenchymal disorders. Optimally, it should be performed by a well-trained endoscopist with adequate support staff and facilities. A full appreciation of the limitations and risks is essential to selecting patients for this procedure.
References


Nasal continuous positive airway pressure (CPAP), is the most common and effective therapy available for sleep apnea. The air pressure is adjusted so that it is just enough to prevent airway collapse during sleep. Apneas will recur if this modality is not in use, therefore 100% adherence to therapy is optimum.
Sleep apnea, first described in 1965, is a serious and potentially life threatening disorder which affects an estimated 18 million Americans of both sexes in all age groups. It is a breathing disorder characterized by repeated collapse of the upper airway during sleep, culminating in apnea. It is more prevalent in the obese individuals, males, the elderly, ethnic minorities and those with systemic hypertension. A familial tendency for sleep disordered breathing (SDB) has been reported, raising the question of a genetic link. It is estimated that 4% of middle-aged men and 2% of middle-aged women meet minimal criteria for sleep apnea syndrome. A detailed history, physical examination and standardized diagnostic testing together can establish abnormalities of SDB. An American Academy of Sleep Medicine Task Force recently outlined the diagnostic criteria and available measurement techniques necessary to establish the diagnosis of the following SDB abnormalities: Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS), Central Sleep Apnea-Hypopnea Syndrome (CSAHS), Cheyne-Stokes Breathing Syndrome (CSBS), and Sleep Hypoventilation Syndrome (SHVS).

OSAHS, by far the most common sleep disorder, occurs when airflow is blocked in the upper airway by physical constraints while efforts to breath continue. The throat, tongue and/or soft palate relax, fall back in the throat and block the airway. Obese patients have redundant posterior pharyngeal tissue which may contribute to this airway blockage. Apneas result and lead to oxygen desaturation, hypercapnia and frequent arousals that prevent the patient from entering restorative deep sleep. CSAHS occurs when the brain fails to send the appropriate signals to respiratory muscles to initiate breathing. CSBS occurs in patients with cardiac dysfunction, most commonly in association with severe congestive heart failure and central nervous system disorders. SHVS is defined as an abnormal increase in $\text{PaCO}_2$ during sleep, which results in hypoxemia, erythocytosis, pulmonary hypertension and/or
respiratory failure. An overlap syndrome of COPD and OSAHS is also common with a prevalence of 10% to 20%, higher than that expected from multiplying the incidence of COPD and OSAHS. This distinction is of clinical importance. It has been demonstrated that sleep-related hypoxemia is more pronounced in this overlap syndrome than in patients with either COPD or OSAHS alone.

**Clinical Presentation**

Recognition of signs and symptoms of SDB in the primary care setting in conjunction with diagnostic testing by physicians with specialized training in sleep disorders leads to early diagnosis of SDB and prevention of the serious sequellae of these disorders. Sleep apnea is often characterized by habitual snoring associated with snorting, gasping and choking sensations. Some 40% to 60% of all adults report snoring. Of note, all patients who snore do not have sleep apnea. Spouses or relatives are usually the first to recognize snoring and nocturnal apneas. Witnessed apneas are highly predictive of OSAHS. Patients are often surprised or in denial of these symptoms. In fact, self-reported awakenings are often not indicative of sleep apnea, but rather a sign of chronic heart failure, gastroesophageal reflux, nocturnal asthma, nocturia or panic disorder. Patients with sleep apnea may also report early morning headaches and dry mouth, excessive daytime sleepiness, decreased daytime concentration and performance/productivity, depression, irritability, sexual dysfunction, learning and memory dysfunction and falling asleep at work, on the phone—or of great concern—while driving, (See Table 16).

A history of excessive daytime sleepiness should raise a suspicion for sleep apnea. The Epworth Sleepiness Scale and the Stanford Sleepiness Scale are surveys widely used for patient self-reporting of sleepiness, which aid in the diagnosis of SDB. The Epworth scale has been validated in clinical studies and correlates with objective
Table 16  Patients at Risk for Sleep Apnea

<table>
<thead>
<tr>
<th>Symptoms</th>
<th></th>
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<tbody>
<tr>
<td>Chronic, loud snoring</td>
<td></td>
</tr>
<tr>
<td>Gasping or choking episodes during sleep</td>
<td></td>
</tr>
<tr>
<td>Excessive daytime sleepiness (especially drowsy driving)</td>
<td></td>
</tr>
<tr>
<td>Automobile- or work-related accidents due to fatigue</td>
<td></td>
</tr>
<tr>
<td>Personality changes or cognitive difficulties related to fatigue</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity, especially nuchal obesity (neck size ≥17 inches in males and</td>
<td></td>
</tr>
<tr>
<td>16 inches in females), &gt;120% ideal body weight, or body mass index (BMI),</td>
<td></td>
</tr>
<tr>
<td>≥30 kg/m²</td>
<td></td>
</tr>
<tr>
<td>Systemic hypertension</td>
<td></td>
</tr>
<tr>
<td>Nasopharyngeal narrowing</td>
<td></td>
</tr>
<tr>
<td>Pulmonary hypertension or cor pulmonale (rarely)</td>
<td></td>
</tr>
</tbody>
</table>

Table 17  The Epworth Sleepiness Scale

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose the most appropriate numbers for each situation.

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of Dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
</tr>
<tr>
<td>Watching TV</td>
<td></td>
</tr>
<tr>
<td>Sitting, inactive in a public place</td>
<td></td>
</tr>
<tr>
<td>As a passenger in a car for an hour</td>
<td></td>
</tr>
<tr>
<td>Lying down in the afternoon</td>
<td></td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td></td>
</tr>
<tr>
<td>Sitting quietly after a lunch without alcohol</td>
<td></td>
</tr>
<tr>
<td>In a car, while stopped for a few minutes in traffic</td>
<td></td>
</tr>
</tbody>
</table>

A score of 10 or greater is highly suggestive of daytime sleepiness.

measurements of sleepiness. A score of 10 or greater is highly suggestive of daytime sleepiness (See Table 17), but is not specific for sleep apnea.

Although the exact link is unknown, the prevalence of systemic hypertension in patients with SDB is very high. Fifty percent of patients with sleep apnea have systemic hypertension. Sleep apnea can also be associated with irregular heart beats, heart attacks and strokes.

Certain abnormalities in the physical examination can also be suggestive of SDB (See Table 18). The physical finding which is most predictive of OSAHS is central obesity. Sleep apnea occurs in individuals who are overweight (Body Mass Index [BMI], > 30 kg/m²). Forty percent of individuals with a BMI ≥ 40 kg/m² and 50% of individuals with a BMI ≥ 50 kg/m² have significant SDB. Men and women with a neck circumference of 17 or 16 inches respectively have a higher incidence of sleep apnea. Patients with anatomic abnormalities of the nose, throat or other areas of the upper airway also have an increased incidence of OSAHS.

**Diagnostic Tests**

**Overnight PSG:** is the gold standard for diagnosis of OSAHS. An overnight PSG includes recordings of airflow, ventilatory effort, oxygen saturation, electrocardiogram, body position, electrooculography (EOG), electromyography (EMG) and electroencephalography (EEG). In standard, laboratory-based PSG, a technician is present for the entire overnight study. Patients with severe OSAHS can have as many as 20 to 30 apneas per hour of sleep. Accordingly, the diagnosis of OSAHS can be made early in the study, and the second half of the evening can be used to titrate the treatment (nasal continuous positive airway pressure [CPAP]). When diagnosis and CPAP titration are done in a single night, the PSG is called a “split night” study.
The EEG is the core measurement of PSG, defining relaxed wakefulness, stage 1 through 4 of non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. Standard lead placement and interpretation is of paramount importance. The identification of characteristic EEG patterns can help diagnose specific sleep disorders: narcolepsy (occurrence of REM within 15 minutes of sleep onset), sleep apnea (increased frequency of arousals and body movements, K complexes), alpha-delta sleep (alpha intrusion into REM sleep in patients with psychiatric disorders) and transient arousals (daytime sleepiness or old age in the absence of apneas). A summary of the amount of time spent in each stage of sleep (REM and the 4 stages of NREM) is generated to evaluate sleep architecture, sleep latencies (elapsed time from lights out until 3 consecutive stage 1 events or one event of any other stage occurs), cycles (NREM-REM descriptions) and the timing of events (apneas, hypopneas, oxygen desaturation, arrhythmias, etc.) with REM or NREM sleep.

The EOG records the cardinal sign of REM sleep, the phasic bursts of rapid eye movements. This measurement is essential for sleep stage scoring criterion. The EMG measures activity in the mentalis and submentalis muscles located under the chin (used as a criterion for REM sleep), in the anterior tibialis (important in evaluating patients with periodic limb movements in sleep, a.k.a., periodic limb movement disorder [PLMD]), in the masseter (to monitor bruxism) and occasionally in the intercostals (to monitor respiratory effort). Airflow is measured with a thermocouple, thermistor, pneumotachometers or capnography (infrared analysis of inhaled/exhaled CO₂). Ventilatory effort is monitored by string gauges, plethysmography, magnetometers, EMG or esophageal pressure.

(continued)
The Apnea-Hypopnea Index (ahi) is the most commonly used criterion derived from PSG testing to establish the diagnosis of OSAHS. The ahi is defined as the sum of apnea and hypopnea events divided by the hours of sleep. In adults, apnea is defined as the cessation or near cessation of airflow for 10 seconds or longer which occurs despite continued ventilatory efforts. The strict definition of hypopnea is more controversial. It is often described as a disturbance which causes a 50% reduction in airflow or a reduction in airflow that causes a physiologic abnormality i.e. a 3% fall in oxygen saturation or an arousal. There is no consensus on what constitutes a “normal” ahi. Adults may experience up to five events (apneas or hypopneas), per hour of sleep and be asymptomatic. It is clear, however, that an increase in ahi closely correlates with the severity of symptoms. In general, the literature supports that the ahi is diagnostic of sleep apnea when the index is ≥ 10, or if the ahi is ≥ 5 in conjunction with the self reporting of hypersomulence or other symptoms of SDB. Another PSG measurement is the Respiratory Effort-Related Arousal (RERA) where an event is defined as a sequence of breaths characterized by increasing respiratory effort leading to an arousal from sleep, but which does not meet criteria for an apnea or hypopnea. Another consistent index generated from PSG testing is the oxygen desaturation index 4% (ODI4), the number of 4% drops in oxygen saturation per hour. Further classifications of the definitions and technology available can be found in a summary statement of the American Academy of Sleep Medicine (See Table 18).

**Multiple Sleep Latency Test (MSLT):** Physiological sleepiness can be thought of as the underlying biological drive to sleep. The primary index of sleepiness is the speed with which an individual falls asleep. This speed is therefore a measure of the intensity of one’s drive to sleep. The MSLT measures the speed with which an individual falls asleep. A MSLT is a series
Table 18  Diagnostic Criteria for the Obstructive Sleep Apnea-Hypopnea Syndrome

The individual must fulfill criterion A or B, plus criterion C

A. Excessive daytime sleepiness that is not better explained by other factors;

B. Two or more of the following that are not better explained by other factors;
   - Choking or gasping during sleep,
   - Recurrent awakenings from sleep,
   - Unrefreshing sleep,
   - Daytime fatigue,
   - Impaired concentration, and/or

C. Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep. These events may include any combination of obstructive apneas/hypopneas or respiratory effort related arousals.

of “nap” opportunities presented at two-hour intervals beginning two hours after initial (morning) awakening. The patient is placed into an environment conducive to sleep. They are instructed to allow themselves to fall asleep and not to resist falling asleep. The time it takes the individual to fall asleep is monitored. EEG, EOG, submentalis EMG, airflow and sound (snoring) parameters are also monitored. Normal individuals will take between 10 and 20 minutes to fall asleep. If the time is 5 minutes or less, the patient is likely to have a sleep disorder which requires treatment. Other measures of sleepiness include: pupillography, electroencephalography, Maintenance of Wakefulness Test, Vigilance Tests, Profile of Mood States and the two Sleepiness Scales (Epworth and Stanford) mentioned above. MSLT is not generally indicated in the work-up of SDB.

**Home Sleep Studies:** Given the prevalence of SDB in adults and the fact that PSG may not be readily available for all patients via a formal laboratory-based sleep center, considerable interest exists to develop inexpensive, yet reliable portable screening devices for sleep disorders which can be used in the home. To date, these devices, which can measure airflow, ventilatory effort, heart rate, oxygen saturation and sleep parameters, have not been shown to save time or cost in the diagnosis of SDG. An excellent review of available devices was recently published. While use of oximetry alone has been proposed to diagnose OSAHS, its sensitivity and specificity are controversial.

**Treatment**

The goal for sleep apnea treatment includes both physiologic (eliminate sleep fragmentation, apneas, hypopneas and oxygen desaturation) and symptomatic (eliminate snoring and sleepiness, improving quality of life and reducing comorbidities) components (See Table 19). Therapy is individualized based on medical history, physical examination and results of PSG. (continued)
**Table 19  Treatment of Sleep Apnea**

<table>
<thead>
<tr>
<th>Modification of Behavioral Factors</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss (including exercise program)</td>
<td></td>
</tr>
<tr>
<td>Avoidance of alcohol and sedatives before sleep</td>
<td></td>
</tr>
<tr>
<td>Avoidance of supine sleep position</td>
<td></td>
</tr>
</tbody>
</table>

**Nasal CPAP**
- Non-invasive
- Very effective
- Patient adherence variable

**Oral/Dental Devices**
- May be useful in mild to moderate cases
- Not uniformly effective

**Surgical Procedures**
(UPPP, Tonsillectomy, LAUP, Nasal and Maxillofacial Surgery)
- Invasive
- Not uniformly effective
- May carry risk
- Repeat sleep study is necessary after each procedure

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Behavioral modification is essential: weight loss, avoidance of alcohol, tobacco, sleeping pills and sedatives and perhaps repositioning during sleep (if apneas occur preferentially while patients lie in one position). Patients with sleep apnea should be advised not to drive if untreated.

Nasal CPAP is the most common and effective therapy available. The air pressure is adjusted so that it is just enough to prevent airway collapse during sleep. Apneas will recur if this modality is not in use, therefore 100% adherence to therapy is optimum. However, many patients use CPAP intermittently. Some CPAP use is better than none at all. Various devices and accessories are utilized to minimize the side effects of nasal irritation and drying, fascial skin irritation, abdominal bloating, mask leaks, sore eyes, rhinorrhea or nasal congestion and headaches. In certain cases, dental appliances can aid in repositioning of the tongue and jaw for patients who snore but do not have apneas.

Several surgical procedures have been developed to increase the size of the upper airway, to reduce the redundancy of upper airway soft tissue and to reconstruct deformities of the nose and/or lower jaw. Some of the more common procedures include: removal of the adenoids and tonsils, uvulopalatopharyngoplasty (UPPP), laser-assisted uvulopalatoplasty (LAUP), tracheostomy and surgical procedures to treat obesity. UPPP success is in the range of 15% to 20%. LAUP, like UPPP, may decrease or eliminate snoring, but not sleep apnea itself. Tracheostomy is definitive therapy and highly effective, bypassing the area of airway collapse. But it is an extreme invasive measure with known complications that are poorly tolerated by the majority of patients.

Administration of supplemental oxygen may improve nocturnal desaturation, but does not primarily treat sleep disruption or daytime sleepiness. Oxygen should
be used in those patients with the “overlap syndrome”, OSAHS and COPD, who have nocturnal desaturations and indications for oxygen therapy based on conventional criteria.

Once effective therapy has been initiated, all patients should be periodically reevaluated for reoccurrence of symptoms such as snoring, daytime sleepiness and comorbidities. The primary caregiver plays a key role in determining if the patient is adhering to treatment and in monitoring concurrent problems such as systemic hypertension (which may require less treatment if sleep apnea is controlled). If patients are adherent to therapy and symptoms of sleepiness persist, the patient should be reevaluated and consideration of diagnoses of other sleep disorders should be entertained. ■

(continued)
References


Johns MW. Reliability and factor analysis of Epworth Sleepiness Scale. Sleep 1992;15:376-381. This article presents a commonly used grading system for daytime sleepiness.


The National Heart, Lung, and Blood Institute Working Group on Sleep Apnea. Sleep Apnea: Is your patient at risk? NIH Publication 95-3803, September, 1995, pp 1-10. This monograph from NHLBI reviews the definition, prevalence and co-morbidities of sleep apnea. It summarizes how to identify patients, make the diagnosis and treatment options for sleep apnea.
The authors realize that while most frontline physicians are not involved in the ventilatory management of their patients with respiratory failure, they may closely follow this aspect of their medical care. The purpose of this Section is to familiarize primary care physicians with concepts of mechanical ventilation, not to provide specifics of ventilatory management.

Mechanical ventilator therapy is a central component of critical care for patients with impaired respiratory capacity due to various acute and chronic disorders. Ventilator support is needed when patients are not capable of maintaining adequate oxygenation (low $\text{PaO}_2$) or providing adequate ventilation (high $\text{PCO}_2$). Multiple situations apply including ventilatory support of patients with acute and chronic lung disease, congestive heart failure, neuromuscular disease, acute trauma and postoperative status. Because of the wide array of conditions requiring mechanical ventilator support and the different pathologic and physiologic processes that are involved, a comprehensive understanding of types of ventilator support is essential for physicians managing these complex patients. More recently, non-invasive mechanical ventilatory techniques have offered a means to support patients’ respiratory status without intubation and standard ventilation.

This discussion will cover the following modes of ventilatory support:

1. Volume ventilation
   a. Assist-control (AC) and continuous mechanical ventilation (CMV)
   b. Intermittent mandatory ventilation (IMV) and synchronized intermittent mandatory ventilation (SIMV)
2. Pressure ventilation
   a. Pressure control ventilation (PCV)
   b. Pressure support ventilation (PSV)
3. Positive end-expiratory pressure (PEEP) and continuous positive airway pressure (CPAP)
4. Non-invasive ventilatory support
   a. Continuous positive airway pressure (CPAP)
   b. Bi-level positive airway pressure (BiPAP)
   c. Negative pressure chest wall devices, (e.g., cuirass)

<table>
<thead>
<tr>
<th>Volume Ventilation</th>
<th>Assist-Control (AC), and Continuous Mechanical Ventilation (CMV)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With both AC and CMV modes tidal volumes (TV’s) of 6 to 12 cc/kg and respiratory rates (RR’s) of 10 to 14 breaths per minute are selected. With CMV, the patient will only receive the number of breaths and the volume chosen. The ventilator generates whatever pressure is necessary to deliver the set TV. If the patient has a more rapid rate or pulls increased negative pressure, then the ventilator is overridden and there is poor patient-ventilator interface. In contrast, the AC mode allows for the patient to add as many breaths per minute as wanted. Each breath delivers the preset TV. In critical patients who are heavily sedated, the AC mode allows for fine control of minute ventilation (MV = RR TV) while allowing the patient to add extra breaths as desired. Typical settings for a 70-kg man would be a TV of 500–600 cc with a RR of 12.</td>
</tr>
</tbody>
</table>

| Intermittent Mandatory Ventilation (IMV) and Synchronized Intermittent Mandatory Ventilation (SIMV) | As with the AC and CMV modes, TV’s of 6 to 8 cc/kg and RRS of 6 to 14 breaths per minute are selected. The key difference between IMV-SIMV and CMV-AC is that when the patient takes additional breaths, the TV is based on the negative pressure generated by the patient, not by the preset volume of the ventilator. Thus, with IMV, the patient has a preset rate which delivers a set TV on a regular basis (e.g., for a rate of 10, one breath is given every 6 seconds). Because breaths with preset TVs are given without regard to the patient’s spontaneous respirations, the patient could receive a much higher TV (the patient’s breath plus that of the ventilator’s), potentially resulting in excessive volumes. SIMV, on the other hand, not only allows the patient to breathe spontaneously between mechanical |
breaths, but also synchronizes any delivered mechanical breaths with the next spontaneous breath. In recent years, SIMV has replaced IMV in most hospitals. SIMV weaning consists of gradually reducing the RR over minutes to hours. When the SIMV rate is 0 to 2 and the patient supports ventilation, extubation is performed (See Weaning From Mechanical Ventilation).

In patients with no spontaneous respirations who have the same RR and TV settings, the AC and SIMV modes function in identical manners.

**Pressure Ventilation**

As with volume ventilation modes, a RR must be selected. In contrast to AC and CMV, a peak inspiratory pressure is chosen. The volume delivered is dependent on lung compliance (volume/pressure). Following acute injury, lungs become stiff and high pressures (>50 cm H$_2$O) may result from delivery of the usual TV’s, 6 to 8 cc/kg, when employing AC or CMV modes. Because high mean airway pressures may cause barotrauma with additional lung injury, it is sometimes necessary to limit peak pressures. Peak and mean airway pressures can be limited by use of a pressure control mode (PCV). Because delivered TV’s are lower, the RR is increased to provide adequate MV. With this mode of ventilation, other more complicated ventilator adjustments are frequently required. Such patients usually require heavy sedation and neuromuscular blockers.

**Pressure Control Ventilation (PCV)**

This form of ventilation is used in conjunction with SIMV or as additional support to traditional weaning. A set pressure is selected and delivered with every spontaneous breath. Because the patient must initiate a breath to receive the pressure support, this should never be used alone in patients with significant apneas. As with PCV, the TV delivered depends on lung compliance (i.e., the stiffer the lung, the lower the TV for any given pressure). The use of PSV and SIMV allows for the patient to receive a set TV from the SIMV setting and pressure support variable volumes with spontaneous respirations (See Weaning From Mechanical Ventilation).
Both PEEP and CPAP provide continuous airway pressure and are used in conjunction with either volume or pressure modes of ventilation. The purpose is to improve oxygenation not ventilation. This is accomplished by increasing the functional residual capacity (See Section B) and recruiting atelectatic alveoli opening previously collapsed lung units. Generally, PEEP is the term used to describe the positive airway pressure associated with both volume and pressure ventilation, and CPAP is used in association with weaning (spontaneous breathing) and non-invasive ventilation. The physiologic effect is identical since there is positive airway pressure at all times including end-expiration.

CPAP has become an extremely important therapy for patients with sleep disordered breathing (SDB), especially obstructive sleep apnea. A mask or nasal pillow device is placed over either the nose alone or both the nose and mouth. The key component to success is a good fit of this facial device. CPAP is applied, usually in the range of 5 cm to 15 cm H₂O pressure. More recently, this technique has been used in patients with acute pulmonary decompensation (e.g., pneumonia and COPD) to prevent intubation and mechanical ventilation.

BiPAP represents a refinement of CPAP. BiPAP not only provides continuous positive airways pressure (CPAP) at end-expiration, but it also provides a higher peak pressure which improves oxygenation and ventilation during inspiration. BiPAP not only provides positive airway pressure during inspiration, improving ventilation, it also provides continuous positive airway pressure (CPAP) at end-expiration, improving oxygenation. As with CPAP, a new family of improved masks that either cover the nose alone or both the mouth and nose must be carefully fitted. Typical initial settings might be an inspiratory positive airway pressure (IPAP) of 10 cm H₂O and an expiratory positive
airway pressure (EPAP) of 4 cm H\textsubscript{2}O. BiPAP can be administered with a spontaneous setting where the patient controls the RR, a timed setting where the RR is predetermined, or both. The latter is similar to SIMV and CPAP ventilation. As this form of respiratory support has become more available, it has generally replaced CPAP as the preferred non-invasive positive pressure ventilation mode in treatment of acute and chronic respiratory insufficiency. Patients with severe neuromuscular diseases who do not want a tracheotomy with volume ventilation support now have BiPAP as an option.

**Negative Pressure Chest Wall Devices**

The development of these devices dates back to the polio era and the use of the iron lung. Patients were placed in a metal tube with a rubber seal around the neck and negative pressure was generated to expand the chest wall. Passive exhalation occurred and the cycle was repeated. Because these were large, bulky, non-portable devices, their use was limited. Currently, many refinements on this concept have produced small, portable devices that fit over the thorax. The original cuirasses were made of firm plastic, which sometimes made a tight seal on the chest wall difficult. New models employ more flexible materials allowing for better comfort. Since BiPAP has become available, these devices are used less frequently.

**Complications**

The use of mechanical ventilation with an endotracheal tube or tracheostomy may be associated with many unforeseen complications. These are briefly summarized in Table 20, based on a prospective study in a large number of patients.

(continued)
A. Complications attributable to intubation and extubation
   1. Prolonged intubation attempt
   2. Intubation of right mainstem bronchus
   3. Premature extubation
   4. Self extubation
B. Complications associated with endotracheal/tracheostomy tubes
   1. Tube malfunction
   2. Nasal necrosis
C. Complications attributable to operation of the ventilator
   1. Machine failure
   2. Alarm failure
   3. Alarm found off
   4. Inadequate nebulization or humidification
   5. Overloading of inspired air
D. Medical complications occurring during assisted ventilation
   1. Alveolar hypoventilation
   2. Alveolar hyperventilation
   3. Massive gastric distention
   4. Pneumothorax
   5. Atelectasis
   6. Pneumonia
   7. Hypotension

Weaning From Mechanical Ventilation

Initial Evaluation of the Patient Prior to Weaning

The choice of a weaning modality remains one of the most controversial areas in pulmonary and critical care medicine. There are many variations on the two common weaning methods. However, before considering “weaning,” the physician should determine if the patient continues to require mechanical ventilation. If support is no longer necessary, then the patient should be extubated without a weaning process.

The first step in determining whether continued mechanical ventilation is necessary is measuring the patient’s spontaneous ventilation parameters. These include:

- Respiratory rate (RR)
- Tidal volume (TV)
- Minute ventilation (MV)
- Negative inspiratory force (NIF)
- Vital capacity (VC)

Arterial blood gases identify the patient’s current oxygenation (\(\text{PaO}_2\) and \(\text{SaO}_2\)) and ventilation (\(\text{PaCO}_2\) and pH, See Section C). Frequently, though, measurements of \(\text{O}_2\) saturation and end-tidal \(\text{CO}_2\) are sufficient. When most of the following guidelines are met, the patient can usually be safely extubated:

- \(\text{RR} = 8\) to 25 breaths per minute
- \(\text{TV} = 5\) to 15 cc/kg
- \(\text{MV} = 5\) to 15 liter per minute
- \(\text{NIF} \geq -25 \text{ cm H}_2\text{O}\)
- \(\text{VC} > 1 \text{ liter}\)
- \(\text{PaO}_2 \geq 55 \text{ mm Hg}\) with \(\text{F}_1\text{O}_2 \leq 60\%\)
- \(\text{SaO}_2 \geq 90\%\) with \(\text{F}_1\text{O}_2 \leq 60\%\)
- \(\text{PaCO}_2 \leq 45 \text{ mm Hg}\) (without chronic \(\text{CO}_2\) retention)
- \(\text{pH} \geq 7.35\)

If the patient does not satisfy most of the above criteria, then continued ventilator support is presumably needed. Otherwise, the weaning process is initiated.
Weaning Strategies

The two most common forms of weaning are the T-piece or 0-cPAP mode and the simv mode. Considerable controversy exists over which modality is superior and the underlying reason for respiratory failure (e.g., hypoventilation versus high oxygen requirements).

T-Piece or 0-cPAP Weaning Mode

The intubated patient may either be disconnected from the ventilator and placed on a T-piece or the ventilator may be set in the 0-cPAP mode which gives no additional pressure or volume support to the patient’s spontaneous effort. At time 0 and after 30 to 60 minutes of this weaning process, the patient is transiently removed from ventilatory support and the above spontaneous parameters are measured. Once the above-noted extubation criteria are met, the endotracheal tube is removed. Patients who still require ventilatory support are again transitionally removed, initially for five to 60 minutes, three to four times per day. The duration and frequency of these weaning periods may be increased until extubation criteria are met.

There are two additional support modes which can be added to 0-cPAP weaning. These will not work with T-piece weaning since the T-piece is strictly a flow-by oxygen delivery system. The first is the addition of cPAP, usually in the 5 to 10 cm H₂O range, which may improve oxygenation and allows for reduction in F₁O₂, or pressure support (ps) may be added to deliver increased TVs and to improve ventilation. The initial pressure selected should deliver an adequate TV of 6 to 10 cc/kg. This pressure is then decreased over hours to days until the patient may be extubated. When ps and cPAP are used together, the ventilatory mode is identical to biPAP. However, the patient is intubated rather than using a facial or nasal mask. When patients achieve settings of ps ≤ 10 cm H₂O and/or cPAP ≤ 5 cm H₂O, most are ready for extubation.

(continued)
SIMV Weaning

Traditional SIMV weaning involves decreasing the RR from 12 to 14 breaths per minute to 0 to 2 breaths per minute over the course of hours to days. The concept is to support ventilation with an adequate number of volume-delivered breaths until the patient’s improved strength and medical condition allow for adequate spontaneous breathing. One potential problem with this method is prolongation of mechanical ventilation, since some patients may meet extubation criteria as they continue to be weaned with this model.

As with 0-cPAP weaning, PS and CPAP may be added to the SIMV mode. When PS is added, the patient still has a set number of volume breaths, but when spontaneous breaths are taken, they are pressure augmented to give higher TV’s. In this system the patient receives both volume and pressure ventilation. Usually the SIMV is reduced to low levels of 0 to 4 breaths per minute before reducing PS. Initial PS settings should be high enough to give TV’s of 6 to 10 cc/kg. In patients where oxygenation is still a problem, CPAP may be added to increase functional residual capacity and to allow for a lower F1O2.

Pressure Support Ventilation (PSV)

In patients with adequate respiratory drive (e.g., no apneas), but marginal spontaneous respirations, the addition of PS may allow them to breathe spontaneously. The extra pressure gives just enough support to prevent the patient from fatiguing. Over several hours to a few days strength may improve allowing the patient to be extubated.

Regardless of the mode of weaning, periodic measurement of the patient’s spontaneous ventilatory parameters should be performed. When adequate values are obtained, weaning ceases, and the patient is extubated.
References

Butler R, Keenan SP, Inman KJ, et al. Is there a preferred technique for weaning the difficult-to-wean patient? A systematic review of the literature. Crit Care Med 1999;27:2331-2336. Concludes that no one technique is superior to the other. How each technique is used is probably more important than the technique itself.


Tobin MJ. Donald F. Egan Scientific Lecture. Weaning from mechanical ventilation: What have we learned? Respir Care 2000;45:417-431. A scholarly review about the evolution of the techniques of weaning from mechanical ventilation and the pros and cons of each.

Zwilich CW, Pierson DJ, Creagh CE, Sutton FD, Schatz E, Petty TL. Complications of assisted ventilation. A prospective study of 354 consecutive episodes. Am J Med 1974;57:161-170. This study was done in a single Intensive Respiratory Care Unit a quarter of a century ago. Similar complications are encountered in the present era.
For many primary care physicians and specialists, stepping into an examination room to find a patient with a stack of printouts of information gleaned from the Internet has become a frequent occurrence. The Internet is changing all healthcare relationships, including those between physicians and their patients. This Section discusses how the Internet evolved, how healthcare consumers and physicians are using it, and concludes with a brief vision of future Internet applications.

The roots of the Internet trace back to a Defense Department program originally designed to provide military organizations with secure and reliable sharing of computer resources and information exchange particularly in the event of nuclear warfare. For an excellent, although detailed, history of the Internet, see www.isoc.org/internet-history/brief.html#origins.

J.C.R. Licklider of Massachusetts Institute of Technology is widely regarded as “The Father of the Internet.” In 1962, he envisioned a globally connected network of computers through which data and programs could be easily accessed from any site. He termed this, “The Galactic Network.” As the system evolved, it was called “DARPA Network” after the sponsoring governmental agency (Defense Advanced Research Projects Agency). It was later renamed the “Internet” to recognize the concept of an open architecture of multiple computer networks. Table 21 summarizes key events in early Internet history. The development of the Internet has generated new terms to describe its structure and function. Some important terms are defined in Table 22.

In less than half a century, the Internet has become a sprawling collection of interconnected government, academic, military, commercial and private individual computers. The term “World Wide Web” (or “web”) is derived from a specialized management of electronic text called “hypertext” which provides a method to
create, access and transfer online documents. The web has grown because browser programs such as Netscape Navigator and Microsoft Explorer make it possible to navigate the web by means of point-and-click graphic interfaces.

The Internet is regarded as having the fastest, most widespread adoption of any new technology. For instance, the telephone was invented by Alexander Graham Bell in 1876 and first commercialized a few years later. However, it did not become a common utility until after World War I. In contrast, it has taken less than three decades for an estimated 300 million people worldwide to have easy access to the Internet.

“Cyberchondriacs”: A recent Harris Poll found that 98 million people accessed the Internet in 1999 seeking healthcare information. This is an increase of 44 million since 1998. The proportion of those who used the web to look for health or medical information increased during the same period from 71% to 86%. Humphrey Taylor of the Harris Poll has termed these people, “Cyberchondriacs.” Healthcare information is not the most frequent reason people access the web, but it is among the most important. The rise of “consumer empowerment” in healthcare has been fed by the ease of access to information afforded by the Internet.

Consumers are using the Internet to find information they say they are not getting from the healthcare system.

People are becoming more actively involved in determining their care: Only 5% of Americans over age 18 years, surveyed in 2000, report they want doctors to make their healthcare decisions for them. The remainder either want to actively share in the decision-making process or to make all the decisions themselves.
Table 21 Important Events in Early Internet Development

<table>
<thead>
<tr>
<th>Year</th>
<th>Event</th>
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<tbody>
<tr>
<td>1961</td>
<td>First paper on packet switching theory (necessary for the Internet to work).</td>
</tr>
<tr>
<td>1962</td>
<td>First recorded descriptions of implications of the Internet (“Galactic Network”), by J.C.R. Licklider.</td>
</tr>
<tr>
<td>1965</td>
<td>First wide area computer network established allowing computers to talk together.</td>
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<tr>
<td>1972</td>
<td>First public demonstration of ARPANET technology to the public.</td>
</tr>
<tr>
<td>1972</td>
<td>Introduction of e-mail.</td>
</tr>
<tr>
<td>1972</td>
<td>Concept of “open-architecture” developed by Bob Kahn, lead to the development of Transmission Control Protocol/Internet Protocol (TCP/IP), allowing widespread networking of computers.</td>
</tr>
<tr>
<td>1982</td>
<td>Required that all host computers convert simultaneously to the TCP/IP standard.</td>
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<tr>
<td>1992</td>
<td>Internet Society founded.</td>
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</tbody>
</table>
Table 22  Glossary of Internet Terms

| **Bandwidth** | The range of frequencies that can be transmitted over a communications channel. The larger the bandwidth, the greater the amount of information that can be transmitted. |
| **Baud** | The rate at which data travels over telephone lines. |
| **BBS** | Bulletin Board System, that can provide online services, such as e-mails, chat groups and access to data bases. |
| **Browser** | Software, which allows navigation to various websites through a point-and-click graphic system. |
| **Cashe** | Storage, as for memory or web pages. |
| **Chat** | Online conferencing in real time. |
| **Chat group** | Online meetings where people log on to the same room at the same time to exchange information. |
| **Domain** | A subset of the Internet. Domains are identified by .gov for government, .org for non-profit organizations, .edu for educational institutions, and .com for commercial ventures. New generic suffixes (e.g., .news, .info, .arts, .shop) are being proposed to increase the number of available web sites. |
| **E-mail address** | An Internet address where people can receive e-mails. |
| **FTP** | File Transfer Protocol, an Internet protocol for transferring files. |
| **Flame** | An angry, critical message sent from one person to another over the Internet. This process is called flaming and is often very verbally abusive. |
| **Gopher** | An Internet search and retrieval system. |
| **Hacker** | A person who specializes in unauthorized access to Internet information and systems. This is a term of honor for some, and disdain for most. |
| **Home page** | The opening screen of a World Wide Web resource, such as documents. |
| **Host** | A computer on the Internet. |
| **HTML** | Hypertext Markup Language is the codes, which are used to construct web pages. |
| **http** | Hypertext Transfer Protocol, the software standards that are necessary for functionality of the World Wide Web. |
| **Internet** | A global information system, linked by specific protocols and standards, that makes high level services based upon computer networked interaction. |
| **ISP** | An Internet Service Provider. |
| **Lurking** | Logging on to a forum, but only as an observer. |
| **MeSH** | The Medical Subject Headings indexing thesaurus of the National Library of Medicine, MEDLARS system. |
| **Moderated** | A forum, mail list or other electronic information exchange whose postings are reviewed and controlled to keep content relevant and to encourage cyber civility. |
| **Mosaic** | A pioneer web browser that spawned Navigator and Explorer. |
| **Newsgroups** | The bulletin board discussions groups of USENET and the Internet. |
| **Posting** | A public accessible written message, usually to a forum, bulletin board, newsgroup or mail list. |
| **Search engine** | Software, which allows searching on the web for particular information. |
| **Server** | A computer that provides programs, archives or other resources to other computers on the network. |
| **Shareware** | Software distributed online with no initial charge. You pay, on the honor system, only if you decide to keep the program. |
| **TCP/IP** | Transmission Control Protocol/Internet Protocol, the computer communications program of the Internet. |
| **Thread** | A series of messages or postings on a single topic. |
| **URL** | Universal Record Locator, the Internet address necessary to find a particular web page. |
| **USENET** | A system of distributed computer bulletin boards on the Internet. |
| **UserID** | The code name or account name assigned to an online user. |
| **Virus** | A computer program that travels from computer to computer and destroys data. At best, it can be a nuisance; at worst highly destructive and disruptive. |
| **World Wide Web** | The hypertext-based protocol that allows access to various resources on the Internet. |
The Internet is a frequent source of consumer healthcare information: Empowered by the Internet, consumers—whether patients or patients’ friends and loved ones—are going outside the traditional healthcare system to obtain the information that they want. Seventy percent of persons searching for health and medical information believe that the Internet empowers them by providing information before and after visiting a doctor. Table 23 lists frequently visited consumer healthcare websites. Table 24 lists some websites that are related to pulmonary disease.

Quality of consumer healthcare information on the web is a concern: It is estimated that there are over 15,000 healthcare-related sites on the web. The quality of information on the Internet varies dramatically. The Internet has been termed, “The world’s largest medical garbage dump.” While this may be an overstatement, it does underscore the variable quality of information on the web.

Consumers commonly visit healthcare portals. Portals are websites that aggregate many sources of electronic information that is then presented primarily in text form. There are non-profit sites, such as that supported by the Mayo Clinic Foundation (www.mayohealth.org), and commercial sites, such as www.medscape.com. The commercial sites have not been financially successful because of the unwillingness of consumers to pay for content information and the inability to generate sufficient advertising and sponsorship revenues.

Where consumers prefer to go for healthcare information is important: Some commercial sites have also developed companion consumer-oriented sites. Such sites have been failures because consumers have virtually ignored them. They prefer to visit physician-oriented sites. Consumers and physicians rely upon the same online sources of clinical and research information, such as the National Library of Medicine’s Medline service (www.nlm.nih.gov). It is clear that
### Table 23  Frequently Visited Consumer Healthcare Websites

<table>
<thead>
<tr>
<th>(AOL) Health Channel</th>
<th>(America on Line users only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drkoop.com</td>
<td><a href="http://www.drkoop.com">www.drkoop.com</a></td>
</tr>
<tr>
<td>HealthAnswers</td>
<td><a href="http://www.healthanswers.com">www.healthanswers.com</a></td>
</tr>
<tr>
<td>IntelliHealth</td>
<td><a href="http://www.intelihealth.com">www.intelihealth.com</a></td>
</tr>
<tr>
<td>Mayo Clinic Health Oasis</td>
<td><a href="http://www.mayohealth.org">www.mayohealth.org</a></td>
</tr>
<tr>
<td>Mediconsult</td>
<td><a href="http://www.mediconsult.com">www.mediconsult.com</a></td>
</tr>
<tr>
<td>Medscape</td>
<td><a href="http://www.medscape.com">www.medscape.com</a></td>
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<tr>
<td>Thriveonline</td>
<td><a href="http://www.thriveonline.com">www.thriveonline.com</a></td>
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</tbody>
</table>
### Table 24  Consumer-oriented Pulmonary Websites

<table>
<thead>
<tr>
<th>Topic</th>
<th>Organization</th>
<th>Web Address</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma</td>
<td>Asthma, Allergy &amp; Pulmonary News on the Net</td>
<td><a href="http://www.asthmanews.com">www.asthmanews.com</a></td>
</tr>
<tr>
<td>Bronchiolitis obliterans and sarcoidosis</td>
<td>Dynamics in Healthcare</td>
<td><a href="http://www.epler.com">www.epler.com</a></td>
</tr>
<tr>
<td>COPD and lung cancer</td>
<td>National Lung Health Education Program</td>
<td><a href="http://www.nlhep.org">www.nlhep.org</a></td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>The Cystic Fibrosis Foundation</td>
<td><a href="http://www.cff.org">www.cff.org</a></td>
</tr>
<tr>
<td>Emphysema and COPD</td>
<td>The National Emphysema Foundation</td>
<td><a href="http://www.emphysemafoundation.org">www.emphysemafoundation.org</a></td>
</tr>
<tr>
<td>Lung diseases</td>
<td>American Lung Association</td>
<td><a href="http://www.lungusa.org">www.lungusa.org</a></td>
</tr>
<tr>
<td>Pulmonary hypertension</td>
<td>The Pulmonary Hypertension Association</td>
<td><a href="http://www.phassociation.org">www.phassociation.org</a></td>
</tr>
<tr>
<td>Sleep disorders</td>
<td>National Sleep Foundation</td>
<td><a href="http://www.sleepfoundation.org">www.sleepfoundation.org</a></td>
</tr>
</tbody>
</table>
consumers want to read the same information their doctors are reading. Physicians should remember this when referring patients and family members to healthcare information on the web.

Other sources of consumer information on the Internet:
Other places consumers are going for information on the web include Internet mailing lists such as CancerNet, sponsored by the National Cancer Institute. They may also seek information from e-commerce sites, such as Internet drug stores and sites that sell products for specific disease conditions (e.g. www.gazoontite.com, an asthma and allergy products site). Internet newsgroups or chat rooms are electronic “places” where people with similar interests can exchange messages or meet online. There are healthcare specific areas of large websites such as Yahoo/health (www.yahoo.com). Computer-based, electronic linkages such as Comprehensive Health Enhancement Support Systems (CHESS http://chess.chsra.wisc.edu/Chess/), are Internet-enabled interactive systems to assist patients with chronic diseases such as AIDS, HIV infection and breast cancer.

Search engines are commonly used, but have issues:
Particular information can be sought on the web by the use of search engines. Eighty-five percent of web users rely upon search engines to find information. Recent studies have shown that no single search engine covered more than 16% of the web content. Some engines merely covered 2.2%. Since it is estimated that in 1999 the web encompassed three million servers hosting 800 billion pages, an immense amount of information is not accessed by individual search engines. Furthermore, the combined 11 popular search engines which were analyzed covered less than half of the web. Search engines may also be a source of bias. New search technologies, such as Google and DirectHit, use measures of “popularity” to rank relevance of pages that they index.
If the growth of the Internet slows, then search engines may “catch up.” Meanwhile, to conduct exhaustive studies, software tools like MetaCrawler that use several search engines should be used.

Consumer use of the web to access information is going to continue to increase: While it is certain that use of the Internet for consumer healthcare information will increase, so too will the volume and complexity of information. This predictability will result in misinterpretation and frustration. Consumers will be faced with sorting out information which is irrelevant, inaccurate and misleading. Less than one-third of medical sites on the Internet studied have been subject to peer review. At least 6% of medical sites contain erroneous information. Attempts are underway to monitor the quality of healthcare information on the web. However, much of the current information which consumers are receiving is unstructured, unreviewed and unregulated. Table 25 lists some questions to ask when evaluating Internet healthcare information sources.

**Information specialists are emerging to assist consumers:** To help consumers, “navigators” who provide human-based assistance are starting to emerge. To illustrate, one of the problems in electronic commerce on the web is frustrated shoppers abandoning their virtual shopping carts. In response to this problem, commercial Internet companies are giving consumers the opportunity to access a customer service representative online to assist them. Such “navigators” may emerge to help healthcare consumers find the suitable information on the Internet. Primary health providers can also serve this function for patients by helping direct patients to websites which they believe contain responsible information that is pertinent to their condition.
Table 25  Questions to Ask When Evaluating Internet Healthcare Sites

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Is it sponsored by a trusted source?</td>
<td>Most academic, professional and some commercial sites receive a high quality peer-review.</td>
</tr>
<tr>
<td>Are forums, chat rooms and bulletin boards moderated?</td>
<td>Some sites allow anyone to post or to participate, rather than have a moderator who monitors content and quality.</td>
</tr>
<tr>
<td>How recently has the site been updated?</td>
<td>Good sites give both dates of posting, electronic publication and when the site was last updated. As with other forms of information, Internet information becomes outdated.</td>
</tr>
<tr>
<td>Are “experts” identified and verifiable?</td>
<td>Early in the history of the Internet there was an infamous case of a 14-year-old giving advice over the Internet to breast cancer patients.</td>
</tr>
<tr>
<td>Is there an issue with language?</td>
<td>Since the Internet allows global participation, the possibility exists for errors and misinterpretations to occur when information is translated from one language to another.</td>
</tr>
<tr>
<td>What are the other potential sources of bias?</td>
<td>This can include commercial advertising, sponsorship groups, institutional viewpoints, religious beliefs and alternative approaches to healthcare.</td>
</tr>
<tr>
<td>Is there any risk of your confidentiality being compromised?</td>
<td>Anytime you are asked to give personal information or you volunteer such information, there is the risk that such information might fall into the wrong hands.</td>
</tr>
</tbody>
</table>
### Table 26  Pulmonary Websites for Physicians

<table>
<thead>
<tr>
<th>Website</th>
<th>URL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha 1-Antitrypsin Deficiency Association</td>
<td><a href="http://www.alpha1.org">www.alpha1.org</a></td>
</tr>
<tr>
<td>American Academy of Sleep Medicine</td>
<td><a href="http://www.asda.org">www.asda.org</a></td>
</tr>
<tr>
<td>American Association for Respiratory Care</td>
<td><a href="http://www.aarc.org">www.aarc.org</a></td>
</tr>
<tr>
<td>Am. Assoc. of Cardiovascular &amp; Pulmonary Rehabilitation</td>
<td><a href="http://www.aacvpr.org">www.aacvpr.org</a></td>
</tr>
<tr>
<td>American College of Chest Physicians</td>
<td><a href="http://www.chestnet.org">www.chestnet.org</a></td>
</tr>
<tr>
<td>American Lung Association</td>
<td><a href="http://www.lungusa.org">www.lungusa.org</a></td>
</tr>
<tr>
<td>American Thoracic Society</td>
<td><a href="http://www.thoracic.org">www.thoracic.org</a></td>
</tr>
<tr>
<td>MDlinx.com</td>
<td><a href="http://www.mdlinx.com">www.mdlinx.com</a></td>
</tr>
<tr>
<td>Medscape; a commercial site with well-reviewed content</td>
<td><a href="http://www.medscape.com">www.medscape.com</a></td>
</tr>
<tr>
<td>National Heart, Lung, and Blood Institute (NHLBI)</td>
<td><a href="http://www.nhlbi.nih.gov">www.nhlbi.nih.gov</a></td>
</tr>
<tr>
<td>NHLBI Asthma Management Model System</td>
<td><a href="http://www/nhlbisupport.com/asthma/">www/nhlbisupport.com/asthma/</a></td>
</tr>
<tr>
<td>National Jewish Medical &amp; Research Center (many topics/services on pulmonary disease)</td>
<td><a href="http://www.nationaljewish.org">www.nationaljewish.org</a></td>
</tr>
<tr>
<td>National Lung Health Education Program (NLHEP)</td>
<td><a href="http://www.nlhep.org">www.nlhep.org</a></td>
</tr>
<tr>
<td>Resource for clinical trials involving pulmonary patients</td>
<td><a href="http://www.clinicaltrials.com">www.clinicaltrials.com</a></td>
</tr>
</tbody>
</table>
Doctors have embraced the Internet and web: Like consumers, physicians have become major users of the Internet. A 2001 survey of 834 physicians conducted by Harris Interactive found that overall, 93% of physicians use the Internet with usage at their clinics (40%) and offices (56%) being considerably lower than at home (87%). The average physician usage was 6 hours per week. However, the majority of time (61%) was for personal use such as e-mail, shopping, financial tracking and accessing general information, such as news. Other uses were for administration and management of their practices.

The Internet has yet to establish a compelling value for doctors in their professional lives: Doctors are quick to grasp a new technology when it has a benefit for both themselves and their patients. Advances in diagnostic imaging and new medical devices are examples. In the case of the Internet though, a persuasive reason for use of the Internet in patient care has yet to be developed. While over 80% of doctors use cellular phones as part of their practice, only 8% of physicians use the Internet as a way to access clinical information. Just as accuracy of information is an issue for consumers accessing information on the web, so too is it for physicians. Table 26 lists some sites that physicians will find relevant to pulmonary disease topics.

It is becoming increasingly popular for physicians to have websites: One rapidly growing Internet area for physicians is establishing websites to support their practices. Forty-two percent of physicians surveyed work in practices with their own websites. This figure is projected to continue to increase. Therefore, practicing physicians should plan to develop websites as a fundamental part of their practice.

e-mail is infrequently used for doctor-patient communication: The use of e-mail between physicians and patients has both promise and problems. Fewer
than 5% of adults who have access to the Internet report using it to communicate with their doctors. Only 13% of physicians report using e-mail to interact with their patients and some doctors who have attempted it have stopped doing so. This is despite studies that have shown that patients would strongly prefer doctors willing to communicate with them using e-mail. The desire of patients to use e-mail involves convenience, but also reflects the decrease in both the frequency and time for face-to-face encounters with their physicians.

**Doctor-patient use of e-mail is not analogous to using the telephone:** When the telephone first became widely available, some doctors expressed concern about being overwhelmed by patients seeking over-the-telephone care. They worried that the telephone would have an adverse effect on the traditional doctor-patient encounter. Similar issues are being raised with the use of e-mail. A critical difference between the telephone and e-mail is that the telephone is a synchronous form of communication occurring in real-time, while e-mail is asynchronous, meaning patients and physicians are not communicating in real-time. Consequently, an urgent e-mail might go unread for a critical period of time.

**There are important issues with the use of e-mail in clinical practice:** While e-mail may be an efficient way to schedule appointments and request refills of prescriptions (many states are now allowing e-mail prescriptions by physicians), serious issues exist around privacy, identifying appropriate and inappropriate uses of the technology and compensation of physicians for the time spent answering e-mails.

Privacy and confidentiality are important issues. The primary care physician using e-mail to communicate with patients must avoid using computers and e-mail accounts that could be accessed by others, including
patients’ family members. Similarly, patients should be advised that if they are sharing resources and e-mail with spouses and family, their medical information may be accessible to them. The best solution is a medical account address that is distinct from personal and professional accounts. Encryption using “public-private key” procedures might also protect against privacy violations. However, such technology is not available for widespread use.

The medicolegal issues of the use of e-mail are also murky. At the least, medical e-mails should be linked to or otherwise included in the patient’s medical record. This represents a logistical challenge that will best be resolved with the widespread availability of an electronic medical record. Unfortunately the electronic medical record has been described as a constantly emerging technology.

Perhaps the most vexing issue regarding e-mail is that of compensating physicians for their time. Until physicians receive fair payment for this form of healthcare delivery, all the anticipated benefits will not be realized. Some payers have begun to appreciate that allowing patients to ask simple or important questions of their physicians via e-mail might well decrease costly office or Emergency Room visits. Some are now experimenting with paying physicians for providing such services. A few physicians are also beginning to offer subscription services, whereby, for a monthly fee, they will respond to their patients’ e-mails. Table 27 lists some things for primary care physicians to consider when contemplating an e-mail system of communication with their patients.
Table 27  Issues to Consider in Using e-mail With Patients

| Is the information secure for both the physician and the patient? |
| Can others access the sending machine and/or the e-mail account? |
| Are all e-mail addresses of patients treated as highly confidential, and only made available on a “need to know” basis? Most violations of the confidentiality of electronic data are committed by authenticated persons. |
| Providing patients with guidelines, and perhaps a template, for communication may decrease length and help triage messages received at a physician’s office. Categories could include prescription refill requests, appointment scheduling and questions that could be answered by an office nurse. |
| Consider having “office hours” during which time you will be online to answer e-mails. |
| Regulatory issues may limit your ability to request payment for e-mail and other online interactions with patients. |
| Electronic communication should become a part of the patient’s permanent medical record. |
| The use of e-mail has complex medicolegal implications. Electronic receive-and-read receipts will help assure that critical messages have been read. Documentation with e-mails is superior to telephone calls; but it may protect against or increase physician liability. |

The Internet and World Wide Web are at a nascent stage of development: Management guru, Peter Drucker, points out the first uses of new technology are to enhance existing systems. It is not until later that truly revolutionary applications are developed. For instance, James Watt’s improved steam engine, when applied to the spinning of cotton, dramatically improved the output and decreased the cost of producing cloth. But, a novel product, the steamboat, invented by Robert Fulton in 1807, had minimal initial impact. Through the end of the 19th century, most freight on the world’s oceans was carried by sailing ships. However, when the steam locomotive gave rise to railroads, a new, revolutionary product was born that forever changed the economy and society.

The Internet and the web were totally unanticipated and unprecedented: The Internet’s marvelous ability to share information was first utilized by the military and academic institutions and only later by the public. The Internet is still in the stage of enhancing the pre-existing systems, such as selling goods and communicating text information and simple images. The Internet has yet to reach the point of revolution where it changes how decisions are to be made, and, like the early use of steam, is only beginning to change some parts of the economy and society. In healthcare, the use of the Internet for accessing information has become commonplace for consumers and doctors. Healthcare information has been democratized. Suppliers of healthcare supply chain products and services are now using the Internet to conduct their business, and as a consequence, introduce efficiencies and cost saving into their products and services.
**Emerging Uses of the Internet**

Other uses for the web, including on-line counseling and second opinions, are in early stage use by both commercial and non-commercial organizations. There are also for-profit “cyber-physician” services that will provide consultation and in some cases even prescribe medications for patients, based upon only online interactions. Such services raise both ethical and legal issues.

New ventures are in early stages to use the Internet for remote monitoring of patients with chronic conditions, such as congestive heart failure, diabetes, COPD and asthma.

**Many new Internet applications in healthcare must await technical advancements:** Other applications, such as use of the Internet to transmit radiographic images, pathology images, and ultrasound studies or for telemedicine, await improvement in Internet technology. These images are electronically dense and require both increased transmission speeds as well as bandwidth carrying capacity. There is currently a national initiative to develop a second generation Internet, termed “Internet-2” or “The Big Pipe.” Information about this project is available at [www.ccic.gov/ngi](http://www.ccic.gov/ngi) and [www.ucaid.org](http://www.ucaid.org). This medium will allow full-motion video and 3-dimensional virtual reality for medical education or training, and for patient education. However, who ultimately will pay for these products and services enabled by the Internet is the question most are reluctant to ask.

**The future is easy to imagine, but difficult to deliver:** The biggest vision, as well as the biggest challenge, for the use of the Internet is to link together the wildly disparate systems for information exchange within the healthcare system. This includes clinical as well as “referral, authorization, claims, eligibility and reporting” (RACER) transactions. While the Internet may be seen as a form of elixir that can somehow
work to provide the miraculous infrastructure linking patients, providers and others with a stake in the healthcare process, there may be similarities to early visions of a space station. It was easy for scientists to imagine the benefits and for engineers to produce the design. In reality, making it happen has taken an immensely long period of time, proven to be significantly more complex than originally imagined and considerably more costly. Companies proposing to use the Internet to widely “wire together healthcare” initially excited the investment community. More recently, enthusiasm has cooled as people have realized the enormity of the task and the fiscal constraints of the healthcare system. The practitioner, faced daily with the realities of a complex, cumbersome and often unresponsive healthcare system, should not expect technological salvation for a number of years.

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Stoll C. Silicon Snake Oil: Second Thoughts on the Information Highway. New York, NY: Doubleday Books; 1995. pp 247. A “classic” (in Internet time) perspective on both the promise, as well as hype about the Internet. The author has been involved in computer networks since their inception. Stoll tracked down a German spy ring working over the Internet and wrote about it in his best-selling book, “The Cuckoo’s Egg.”

A common cause of medical malpractice claims is failure to follow-up on chest x-ray abnormalities which later are found to represent lung cancer. The delays involved may result in the evolution of advanced disease, including locally invasive or metastatic cancer. Uncalcified solitary pulmonary nodules are more common than most primary care practitioners or specialists realize. All non-calcified solitary nodules need to be studied further unless a previous chest x-ray shows no change over two or more years. New technologies, such as isotope imaging (i.e., PET scans) are changing the approach to differentiating benign from malignant lesions. Needle biopsies and video-assisted thorascopic biopsies have reduced the need for exploratory thoracotomy.

Pulmonary infiltrates which persist after acute pneumonic processes, require assessment. Bronchoscopy should be ordered for an acute infiltrate that does not resolve within three months. However, it is important to note that resolution of pneumonic infiltrates may take longer in patients with advanced emphysema. In some patients with very severe COPD one may take a watchful rather than an aggressive stance in evaluating these residual infiltrates. This is based on the relatively high risks for diagnostic procedures (such as bronchoscopy or fine-needle aspiration), the likelihood that the COPD—not the potential neoplasm—will be the life-limiting condition, and the fact that persistent non-neoplastic densities are more apt to occur in such patients.

Failure to provide prophylaxis for patients at high risk of pulmonary thromboembolism has increasingly become a cause for bringing malpractice actions. The fact that anticoagulation can reduce the incidence of PE and mortality in various orthopedic procedures, including hip and knee replacements, is now well-established on the basis of controlled clinical trials. There have not been randomized, controlled, clinical
trials in abdominal operations such as appendectomy, cholecystectomy and bowel resection, but anticoagulation is strongly suggested for high-risk individuals including those for whom immediate ambulation is not possible. Clinical trials are pending for patients undergoing surgical procedures such as radical resection for carcinoma of the prostate. Until results from these studies are available or announced, we believe that it is appropriate that low-dose heparin (5,000 u.s.q. units twice daily or employing a low molecular weight heparin—LMWH) be given to all patients (except those with the contraindication noted below) with major surgical procedures including cancer operations. This is particularly true when the period of immobility may be more than just a few days. Contraindications to this practice include patients undergoing central nervous system or spinal surgery (in whom a hematoma might jeopardize vital neurological function), those with a recent history of pathological bleeding, or those with low platelet counts. Of course, non-pharmacologic therapies such as TED hose or compression stockings carry essentially no risk and probably should be used in all surgical patients. Continuing prophylactic therapy until the patient is fully ambulatory seems reasonable.

Another major reason for malpractice claims is failure to make the diagnosis of acute pulmonary embolism (PE) which subsequently results in a fatal event. The most common symptom of a major PE is sudden dyspnea. Thus, for those complaining of abrupt onset of shortness of breath (particularly those with risk factors including extended travel, recent lower extremity imaging or trauma, women receiving oral contraceptives or persons with a family history of VTE) studies to prove or to disprove PE are strongly advised.
Failure to Do Spirometry

It is amazing that every year pulmonary experts are asked to defend physicians in malpractice actions because of complications of steroid therapy for “asthma” when there is no documentation on the medical record of airflow obstruction. Such examples may be individuals who enter the Emergency Room with classical manifestations of asthma, such as acute wheezing and dyspnea, for whom steroids are deemed necessary. Unfortunately, if the patient continues to receive corticosteroids and a complication such as aseptic necrosis of the femoral head occurs, a lawsuit frequently follows. If there is no documentation of airflow obstruction, the clinician may have substantial legal exposure. The advent of new simple, accurate and inexpensive office spirometers under the National Lung Health Education Program (NLHEP) today should make spirometer use as ubiquitous as blood pressure in physicians offices, clinics and at the workplace.

Spirometry and Lung Cancer Screening

It is now well-established that heavy smokers with airflow obstruction have 4 to 6 times the prevalence of lung cancer compared with those with normal airflow, but with equal smoking, occupational and family histories. Efforts are underway to encourage spirometry testing in all smokers over the age of 45, and anyone with chronic cough, excessive dyspnea on exertion, mucus hypersecretion or wheeze. Patients with 30 pack-years of smoking and airflow obstruction have an approximately 2% prevalence of lung cancer at the time of initial screening, and 3% to 4% more lung cancer in the five years that follow. The authors advise that this group of patients should be screened for lung cancer with spiral CT scans and sputum cytology.

Failure to Refer to Specialists

Today, the public expects that all symptoms and signs of disease will ultimately result in a diagnosis. When troubling symptoms or signs like dyspnea, cough, chest pain, fever, weight loss or anemia cannot be readily diagnosed or managed, the primary care physician should initiate referral to an appropriate specialist.
Although insuring agencies or managed care systems may place impediments in the way of such referrals, we must not lose sight of our ethical responsibility to our patients.

A definitive diagnosis should be expected in all but self-limited symptoms and signs that present to the primary care physician.

One of the most common problems that result in malpractice action is failure to accurately document the medical record. “If it is not written down, it hasn’t been done,” has become a legal dictum. Sadly, this fails to recognize that a physician may be too busy to write down everything done or considered in the differential diagnosis. But a clearly written note, whether or not it follows the style of the problem-oriented record, should be entered into every hospitalized patient’s chart each day, no matter how busy the practice. Records must be dated, timed and signed. The same is true for each office visit.

One of the most common reasons for a lawsuit is when the family feels that they have been badly treated or ignored. Detailed, honest communication is the fiduciary responsibility of every managing and consulting physician. Covering up mistakes, altering the medical record or misrepresentations are predictable causes for adverse medical malpractice judgments placing the physician in great peril. Always tell the truth. Communicate clearly with the family regularly. Document every visit.

Failure to respond to calls from medical personnel such as nurses who are monitoring patients on the clinical wards or in the ICU are a common cause of unfavorable malpractice judgments. The need to respond within a reasonable time is one of the clearest precepts in medicine. Coverage by another medical professional must be provided if you are unable to respond to calls, both on and off of your regular duties.
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A new national healthcare initiative known as the National Lung Health Education Program (NLHEP) is directed at COPD and related disorders, including lung cancer, heart attack and stroke. Thus, this is a new initiative for improved general health in America. The key role of the primary care physician and respiratory care practitioners is stressed by the NLHEP. The NLHEP is directed to all primary care practitioners, the public, third party payers and healthcare administrators. The NLHEP urges that all primary care physicians and other healthcare providers obtain simple, accurate, handheld spirometers for office and clinic use. Only the FEV₁ and the FVC are required to assess patients with COPD in incipient or advanced stages of diseases. “Test Your Lungs, Know Your Numbers” is the battle cry of the NLHEP.

The NLHEP recommends that all smokers age 45 and over and individuals of any age with symptoms of cough, dyspnea, mucus hypersecretion or wheeze should be tested by spirometry. Early identification of patients with spirometric abnormalities indicates an increased risk of all-cause mortality and will guide early diagnosis, therapy and a more positive prognosis. You are invited to view the NLHEP web page at www.nlhep.org.
The Snowdrift Pulmonary Conference, Inc. is a not-for-profit corporation that is dedicated to the dissemination of knowledge about the lungs and lung diseases. Both private practice pulmonologists and academicians have launched a program for primary care practitioners and the patients they serve. This is a consumer-oriented program. Already, concise and authoritative monographs for the frontline practitioner have been written on:

- *Frontline Assessment of Lung Cancer and Occupational Pulmonary Diseases* (2001)
- *Frontline Pulmonary Procedures and Interventions* (2001)

All of the Frontline series are being periodically updated. They are available in hard copy or in a cd-rom format. They can be viewed on the Internet website: www.FrontlinePulmonology.org.
Lung cancer, the most common fatal malignancy of men and women, presents the most challenging frontier. To help disseminate the new knowledge about the role of early identification and intervention in lung cancer as a major new initiative, the Snowdrift Pulmonary Conference has produced a newsletter, *Lung Cancer Frontiers* (*LCF*). *LCF* now reaches the nearly 10,000 board certified pulmonologists in North America. *LCF* is available on the Internet at: www.LungCancerFrontiers.org.

We believe that an enlightened and informed public working with their personal physician and consultants can help guide the future of medicine as we aim to prevent, cure and diminish the number and severity of lung diseases which impact the health of our nation.
The evolution of technology in medicine for diagnostic purposes began with the microscope, which was so critical to understanding the pathology of lung diseases. It was key to the diagnosis of infectious diseases, such as tuberculosis. The first instrument for the evaluation of diseases of the chest was the stethoscope, introduced by Laënnec in 1819. Shortly thereafter, John Hutchinson, a surgeon, introduced the concept of spirometry into medicine. Hutchinson coined the term, “vital capacity,” i.e., the capacity to live. He recognized that a reduced vital capacity was related to all-cause mortality. He was particularly interested in the effects of tuberculosis and coal mining on the vital capacity.

Oxygen was introduced by Priestly in 1774 and finally found application in clinical medicine, beginning with Barach’s use of oxygen in the treatment of pneumonia. Oxygen has revolutionized the management of advanced states of chronic respiratory insufficiency. Evolving mechanical ventilators offered new approaches to the management of acute respiratory failure.

Roentgen discovered x-rays in 1895—chest imaging has evolved and is the cornerstone for many pulmonary diagnoses.

The cuff sphygmomanometer was invented by Italian physician, Scipione Riva-Rocci, in 1896. This simple device caught the eye of U.S. surgeon, Harvey Cushing, who believed he could use it in the measurement of blood pressure, which would be useful in his ongoing studies of cerebral perfusion. Cushing introduced this instrument at Johns Hopkins Hospital. Early supporters of this new method of blood pressure measurements were Theodore Janeway in New York City and George Crile in Cleveland, Ohio. After only two years of experience on the wards of Johns Hopkins, Cushing and his surgical house officers set out to promote wider
use of the cuff. This was the foundation for the use of blood pressure measurements in epidemiological studies and for controlled clinical trials of antihypertensive agents, which have so dramatically reduced the socioeconomic impact of heart attack and stroke in the last 25 years. Perhaps the same may still happen with spirometry, but progress has been painfully slow.

The EKG was introduced by Einthoven in 1903. Rigid tube bronchoscopy was originally introduced by Killian in 1898 and evolved into a flexible fiberoptic bronchoscope, by Ikeda, in 1964. Cardiac catheterization was first developed and studied by Forssmann, a resident surgeon, in 1928. Courand and Richards made a slight modification of Forssmann’s venous catheter and introduced this technique into medicine in 1941. The three pioneers shared the Nobel prize for this momentous advance in 1956. The flow-directed catheter was later introduced by Swan and Ganz. Video-assisted thoracotomy added a new approach to making a tissue diagnosis.

During the past century, numerous blood tests, ultrasonic imaging, isotope scans, various forms of computed tomography and magnetic resonance imaging have continued to aid clinicians in diagnosing a host of disorders of the respiratory and cardiovascular systems. It took not only the invention and introduction of new technologies into medicine, but also an evolving understanding of the advantages and disadvantages of each new technique and treatment to be able to truly benefit from these advances. How each new technology can be applied in a systematic and cost-effective manner, remains a challenge to medicine. No technology, however, can replace the history and physical examination in creating a concept that will lead to a diagnosis, which then can be confirmed by appropriate studies. Systematic treatment requires the interface of new technologies with the judgment and skill of experienced physicians.
References


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Mike Iseman grew up in Fremont, Nebraska. He received his undergraduate degree from Princeton University where he majored in history and played football. He attended Columbia University’s College of Physicians and Surgeons, receiving his MD in 1965. He received his training in internal medicine and pulmonary medicine in New York City between 1965 and 1972.

Joining the faculty of the University of Colorado in 1972, he spent ten years at Denver General Hospital. Then he moved to National Jewish Hospital in 1982 as Head of the Clinical Mycobacterial Diseases program. His primary research interests relate to drug-resistant tuberculosis and disease due to the “atypical mycobacteria.” He currently is Professor of Medicine in the Division of Pulmonary Medicine and Infectious Diseases. He is also Editor-in-chief of the International Journal of Tuberculosis and Lung Diseases.

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Thomas L. Petty received his MD at the University of Colorado in 1958. He interned at Philadelphia General Hospital and received his residency training at the University of Michigan and the University of Colorado. His pulmonary training was at the University of Colorado. He is a pulmonologist and Professor of Medicine at the University of Colorado Health Sciences Center in Denver and at Rush University in Chicago. He was previously Head of the Division of Pulmonary Sciences at the University of Colorado and Director of the Fellowship Training Program. Dr. Petty was founding President of the Association of Pulmonary Program Directors (APPD) and has served as President of the American College of Chest Physicians. He is a former member of the Board of Governors of the American Board of Internal Medicine.

Dr. Petty received the Distinguished Service Award of the American Thoracic Society (1995), was elected to the Colorado Pulmonary Physicians’ “Hall of Fame” (1995) and received the annual award for excellence by the American Association for Respiratory and Cardiovascular Rehabilitation (1995) and the designation of FAARC in 1999. He was elected to Master Fellow of the American College of Chest Physicians (1995). He also received the Master Award of the American College of Physicians in 1996. Dr. Petty has been named Chairman of the National Lung Health Education Program (NLHEP). Its goal is the early diagnosis of COPD and lung cancer. He is also Editor-in-chief of the newsletter, Lung Cancer Frontiers.

Today, Dr. Petty also remains active in teaching, patient care and research. He enjoys fishing, small game hunting and playing with his three “kids” and eight grandchildren.
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Dr. Doherty is Professor of Medicine and Chief of the Division of Pulmonary and Critical Care Medicine and the Medical Director of Respiratory Care Services at the University of Kentucky, Chandler Medical Center, and the Lexington Veterans Administration Medical Center. He completed his medical school and internal medicine residency training at the Ohio State College of Medicine in Columbus, Ohio between 1977 and 1983. His pulmonary and critical care fellowship was received from the University of Colorado Health Sciences Center in Denver, Colorado, from 1983 to 1986, where he remained on the faculty for eleven years.

In 1996, Dr. Doherty relocated to the University of Kentucky to serve as Chief of Pulmonary and Critical Care Medicine. He has been principal investigator on over 35 basic science and clinical grants and has published numerous articles and chapters on the subjects of acute and chronic lung inflammation, obstructive lung disease and pulmonary fibrosis. He serves as editor for three pulmonary journals (The Journal of COPD Management, Pulmonary Perspectives, for physicians, and BreatheWell, for COPD patients) and two medical internet sites (airwaves.com and medscape.com, critical care section). He is the Co-chairman of the National Lung Health Education Program, a Fellow of the ACCP, and is President of the Kentucky Thoracic Society.

Dr. Doherty is an avid home winemaker, a former national champion in handball and enjoys spending time with his wife, Kim, and two children, Erin and Collin.
J. Roy Duke, Jr., MD
Dr. Duke was born in Ocala, Florida and attended Tulane University School of Medicine in New Orleans, Louisiana, obtaining his medical degree in 1960. After a two-year stint in the U.S. Air Force, he completed his postgraduate training in pulmonary medicine at Tulane in 1967.

Dr. Duke joined the Palm Beach Medical Group in West Palm Beach, Florida in 1967 and has practiced pulmonary medicine and internal medicine there to the present. He has served as Chief of Medicine and Chief of Staff of Good Samaritan Hospital in West Palm Beach.

He has an interest in hyperbaric medicine, which is an extension of his hobbies of scuba diving and underwater photography. He is also an avid fly fisherman and fly tier.

Dr. Duke is married to Bobbye Craig Duke and has two children, Denise and Christopher.

(continued)
James T. Good, Jr., MD
Dr. Good received his MD degree from the University of Kansas and completed a medical internship, residency and chief medical residency at the University of Kansas. He then completed a three-year pulmonary and critical care medicine fellowship at the University of Colorado. The next four years he remained on the faculty at the University of Colorado as an Assistant Professor of Medicine and was Medical Director of both the Respiratory Therapy Department and the Critical Care Unit at Denver General Hospital. His scientific interests include management of critical patients with acute respiratory failure, pleural diseases and asthma. He is a fellow of the American College of Physicians and the American College of Chest Physicians (ACCP) and served as the Governor for the states of Colorado and Wyoming for the ACCP from 1988 to 1994.

He currently is in the private practice of pulmonary and critical care medicine in south Denver and is Medical Director of the Swedish/Columbia Critical Care Unit. He remains actively involved in clinical research, teaching medical students and residents and in continuing medical education programs.
Leonard D. Hudson, MD

Dr. Hudson received his B.S. from Washington State University in Pullman, Washington and his M.D. from the University of Washington in Seattle. He did his internship at Bellevue Hospital Center located in New York City, and his residency at New York Hospital, Cornell Medical Center (New York City), and at the University of Washington, in Seattle. He did his pulmonary/critical care fellowship at the University of Colorado and stayed on the faculty there from 1971 to 1973. In 1973, he moved to Seattle’s Harborview Medical Center and the University of Washington.

In 1985, Dr. Hudson became Head of Pulmonary and Critical Care Medicine at the University of Washington. Since 1982, he has been a Professor of Medicine at the University of Washington, Seattle.

Dr. Hudson’s honors include Outstanding Resident, Harborview Medical Center; American Thoracic Society Fellowship in Pulmonary Diseases; Chair, Pulmonary Disease Subspecialty Board, American Board of Internal Medicine; and Chair, Critical Care Medicine Test Committee, American Board of Internal Medicine. He was President of the American Thoracic Society from 1995 to 1996.

Hudson has four children, Sean, Sherry, Meg and Kevin. His hobbies include wood-fired pottery, listening to jazz and reading poetry. He is an amateur fly fisherman and plays soccer for the Seattle Flounders, older men’s division.

(continued)
Charles H. Scoggin, MD

Dr. Scoggin received his medical degree from the University of Colorado, training in internal medicine at Duke University and pulmonary medicine and critical care in the Division of Pulmonary Sciences at the University of Colorado in Denver, Colorado. He also trained in molecular and cellular biology at the University of Colorado and Eleanor Roosevelt Institute.

He has held the positions of Professor of Medicine, Clinical Director of the Department of Medicine, Director of the House Staff Training Program of the Department of Medicine and Head of the Adult Human Genetics Section, all at the University of Colorado. He was also Senior Scientist and Vice President of the Eleanor Roosevelt Institute.

During his academic career, Dr. Scoggin received numerous awards and recognitions. He was Teaching and Research Scholar of the American College of Physicians, a Fellow of the American College of Physicians, Research Scholar of the American Lung Association and Fellow of the American College of Chest Physicians.

Dr. Scoggin is currently Chairman, President and Chief Executive Officer of Medrock, Inc., a Boulder, Colorado and Cambridge, Massachusetts-based company focused on providing assistance to family members and loved-ones of patients experiencing a medical crisis. He enjoys fishing, hunting, his family and good friends.
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