Medical Education Systems, Inc.

Course 701

Respiratory Examination and Treatment

Spirometry/HBO/ECG

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Respiratory Examination and Treatments: Spirometry/HBO/ECG
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PULMONARY FUNCTIONS TESTS: SPIROMETRY

LEARNING OBJECTS

Upon completion of this continuing education unit, you should be able to:

1. Identify and discuss spirogram acceptability criteria.
2. Demonstrate how to interpret spirograms.
3. Explain how to classify obstructive ventilatory defects by severity.
4. Identify and discuss how to recognize flow-volume loops that suggest upper airway obstruction.

INTRODUCTION

Pulmonary function testing is one of the basic tools for evaluating a patient's respiratory status. In patients with suspected pulmonary disease, it is often the first diagnostic test employed in the work up. Pulmonary function tests (PFT's) are also used for pre-operative evaluation, managing patients with known pulmonary disease, and quantifying pulmonary disability. Although the fully equipped PFT laboratory utilizes a host of sophisticated equipment, simple spirometry is relatively easy to administer and standardize and has become portable enough to use at outreach clinics and industrial health centers. The increasing availability of spirometry has led to greater use by both primary care physicians and specialists, some of whom may lack formal training in interpreting results. This educational module is designed to provide an introduction to quality assessment and basic interpretation of spirometric testing.

SPIROMETRY

Spirometry with flow volume loops assesses the mechanical properties of the respiratory system by measuring expiratory volumes and flow rates. This test requires the patient to make a maximal inspiratory and expiratory effort. The patient in a sitting position breathes into a mouthpiece, and nose clips are placed to prevent air leak. To obtain interpretable results from spirometry, it is essential that the patient give full effort during testing. At least three tests of acceptable effort are performed to ensure reproducibility of results.

Flow volume loops provide a graphic illustration of a patient's spirometric efforts. Flow is plotted against volume to display a continuous loop from inspiration to expiration. The overall shape of the flow volume loop is important in interpreting spirometric results. The volume versus time curve is an alternative way of plotting spirometric results and is another useful illustration of patient performance.

Spirometry is a versatile test of pulmonary physiology. Reversibility of airways obstruction can be assessed with the use of bronchodilators. After spirometry is completed, the patient is given an inhaled bronchodilator and the test is repeated. The purpose of this is to assess whether a patient's pulmonary process is bronchodilator responsive by looking for improvement in the expired volumes and flow rates. In general, a > 12% increase in the FEV1 (an absolute improvement in FEV1 of at least 200 ml) or the FVC after inhaling a beta agonist is considered a significant response. However, the lack of an acute bronchodilator effect during spirometry does not exclude a response to long-term therapy.

Similarly, spirometry can be used to detect the bronchial hyperreactivity that characterizes asthma. By inhaling increasing concentrations of histamine or methacholine, patients with asthma will demonstrate symptoms and produce spirometric results consistent with airways obstruction at much lower threshold concentration than normals.

Definitions and Terms

- FEV1 - forced expiratory volume 1 - the volume of air that is forcefully exhaled in one second.
- FVC - forced vital capacity - the volume of air that can be maximally forcefully exhaled
- FEV1/FVC - ratio of FEV1 to FVC, expressed as a percentage
- FEF25 - 75 - forced expiratory flow - the average forced expiratory flow during the mid (25 - 75%) portion of the FVC
- PEF - peak expiratory flow rate - the peak flow rate during expiration

Spirometry is typically reported in both absolute values and as a predicted percentage of normal. Normal values vary depending on gender, race, age, and height. It is therefore not possible to interpret PFT's without such information. There is no single set of standard reference values, however, and "normal" varies with the reference value used in each laboratory. It is therefore

important to ensure that the reference formulas in a PFT lab are applicable to the patient population being tested.

REQUIREMENTS FOR GOOD PFT's

Obtaining useful information from pulmonary function tests requires both adequate equipment and reproducible performance. If these requirements are not met, the results must be interpreted with caution.

The American Thoracic Society (ATS) has published guidelines for the standardization of spirometry equipment and performance. Spirometric equipment should be selected to meet ATS recommendations, and at least daily monitoring and calibration must be done to obtain consistent and accurate PFT's.

Many physicians, however, are not involved in the selection of the equipment used for testing or its maintenance. But because spirometry is such an effort dependent test, they should examine each spirogram using performance criteria as set forth by the ATS. These include criteria for acceptability and reproducibility.

Criteria for acceptability include:

1. lack of artifact induced by coughing, glottic closure, or equipment problems (primarily leak)
2. satisfactory start to the test without hesitation
3. satisfactory exhalation with six seconds of smooth continuous exhalation and/or a plateau in the volume time curve of at least one second, or a reasonable duration of exhalation with a plateau

Criteria for reproducibility after obtaining three acceptable spirograms include:

1. largest FVC within 0.2L of next largest FVC
2. largest FEV1 within 0.2L of next largest FEV1
3. if the two above criteria have not been met, additional spirograms should be obtained

An acceptable spirogram should not be discarded even if it cannot be reproduced. Up to eight efforts may be performed in order to meet acceptability and reproducibility criteria. Beyond eight efforts, fatigue may play a role in the results, and additional efforts are not warranted. When reporting the actual values obtained from spirometry, the highest FEV1 and FVC should be reported and may be from different efforts. By contrast, FEF25-75, flow volume, and volume time curves should be reported from the best test curve. This is defined as the acceptable curve with the largest sum of FVC and FEV1.

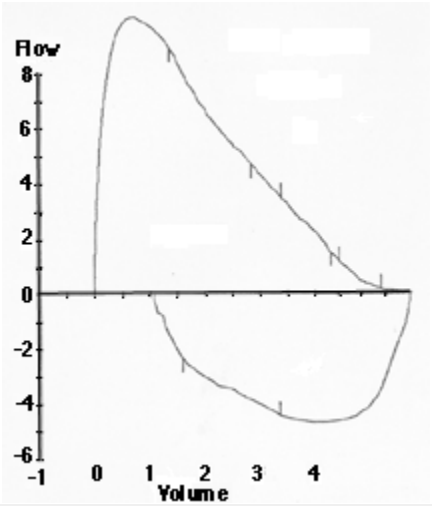
Ensuring adequate patient effort depends on the technician measuring spirometry. The patient should be well coached and instructed throughout the test while the technician evaluates both the

patient's effort and the spirograms. Meeting reproducibility criteria helps to ensure adequate effort because maximal patient effort leads to more reproducible results.

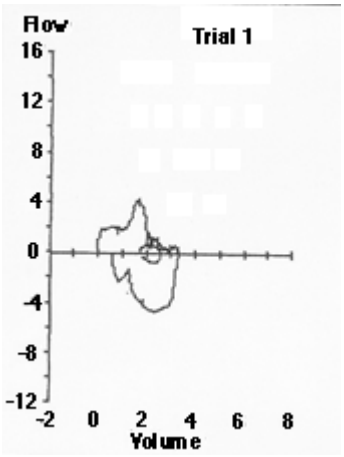
When interpreting PFT's, it is useful to keep the ATS guidelines in mind. Frequently not all the criteria are met, and this may limit the reliability of the test if the results are abnormal. If the spirometric results are normal, the test can be interpreted as normal even though all criteria may not be met.

Test acceptability is best determined by examining the flow volume loop and volume time curve. Variable effort, cough, and early glottic closure can be seen on the graphs but may not be apparent by simply looking at values for FEV1 and FVC.

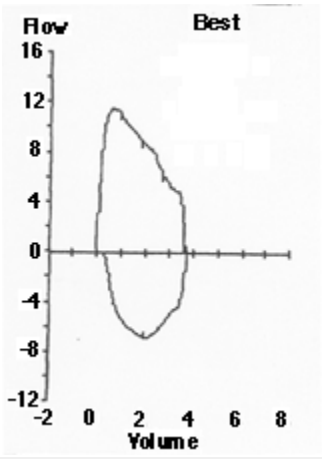
Normal



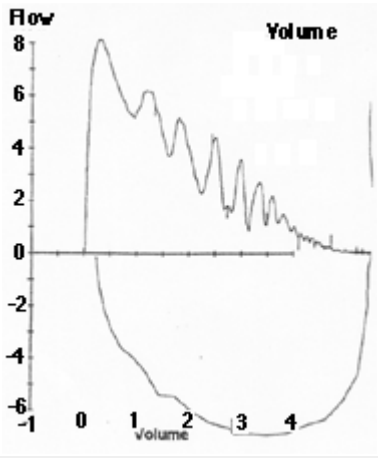
Variable Effort



Early Glottic Closure



Cough



Variable effort can be detected by a flow volume loop that fails to demonstrate the normal early peak, showing that the patient failed to expire maximally when instructed to do so. Early glottic closure is seen as an abrupt cessation of flow during expiration, visible as a sharp downslope on the expiratory flow volume curve. Coughing during spirometry appears as sudden sharp spikes in the decreasing limb of the flow volume curve.

By examining the flow volume loops, the quality of a spirogram can be assessed. In addition, the technician working with the patient should comment on the patient's effort and the session as a whole. At least three efforts must be attempted to meet reproducibility criteria.

While there is some physician-to-physician variability in the interpretation of PFT's, general principles are agreed upon. The criteria given here are the ones used at the University of Iowa Hospitals and Clinics.

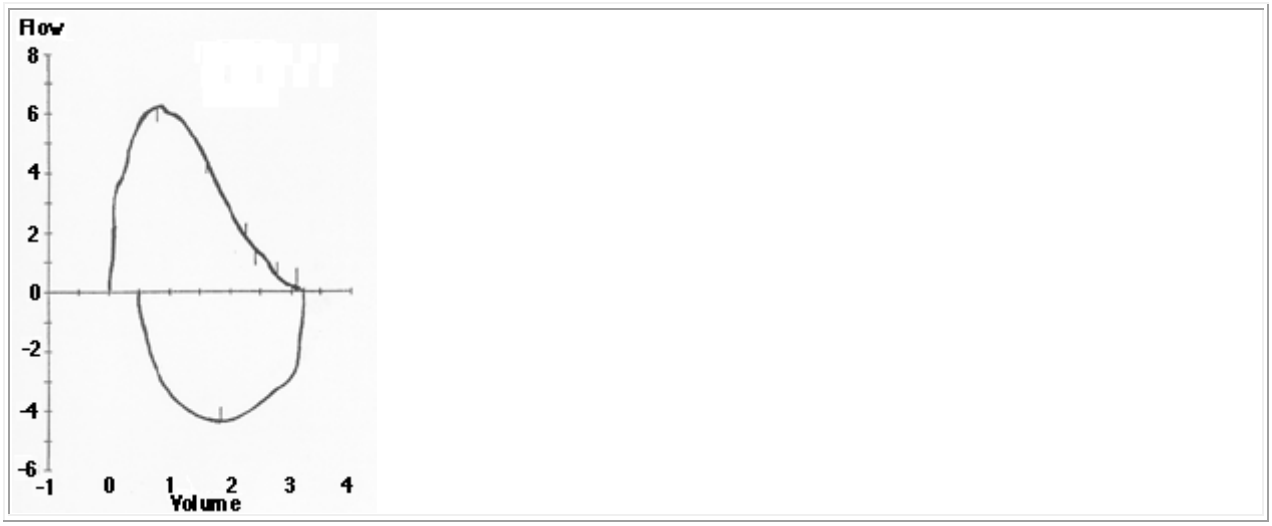
- Normal
- Obstructive Lung Disease
- Restrictive Lung Disease
- Upper Airway Obstruction
- Interpretation Algorithm

Spirometry should be interpreted using the flow volume and volume time curves as well as the absolute values for flows and volumes. The flow volume loop and volume time curve are often overlooked but provide valuable information. Certain disease states have characteristically shaped loops, so it is important to be able to recognize the different patterns.

Normal values for FEV1 and FVC are based on population studies and vary according to race, height, age, and gender. They are expressed in both absolute numbers and percent predicted of normal. Some authors have suggested that defining normal by 95% confidence intervals would be more statistically appropriate, particularly at the extremes of age. Thus, a value below the 5th percentile is defined as "below the lower limit of normal." However, many laboratory and computer software programs continue to express results as percentages of predicted normal values. A physician's interpretative strategy should be adaptable to either reporting system.

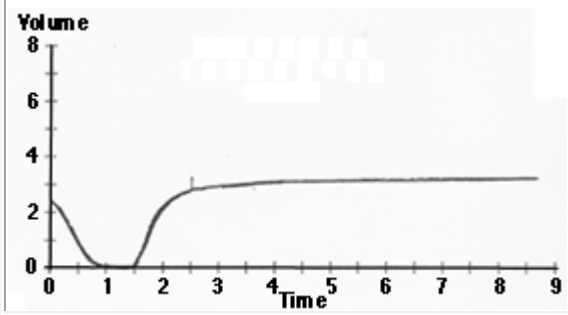
Values for FVC and FEV1 that are over 80% of predicted are defined as within the normal range. The FEV1/FVC ratio is expressed as a percentage, and a normal young individual is able to forcibly expire at least 80% of his/her vital capacity in one second. A ratio under 70% suggests underlying obstructive physiology; however, the FEV1/FVC ratio declines as a normal sequelae of aging. Thus, at advanced ages, pathologic airways obstruction is diagnosed based upon deviation from predicted FEV1/FVC values, with values below the 5th percentile best selecting patients with obstructive defects.

Normal Flow Volume Loop



A normal flow volume loop has a rapid peak expiratory flow rate with a gradual decline in flow back to zero. The inspiratory portion of the loop is a deep curve plotted on the negative portion of the flow axis. Inspiratory data is often overlooked, but the flow volume loop gives the opportunity of assessing this information as well.

Normal Volume Time Curve



The normal volume time curve has a rapid upslope and approaches a plateau soon after exhalation. The maximum volume attained represents the forced vital capacity (FVC), while the volume attained after one second represents the forced expiratory volume (FEV1).

OBSTRUCTIVE LUNG DISEASE

The primary abnormality detected by spirometry is airways obstruction. In obstructive lung diseases such as emphysema or chronic bronchitis, the FEV1 is reduced disproportionately more than the FVC resulting in an FEV1/FVC ratio less than 70 - 80%. This reduced ratio is the primary criteria for diagnosing obstructive lung disease by spirometry. At our institution, we use the following scale to grade the severity of obstruction:

FEV1	> 80% predicted	normal
	65 - 80%	mild

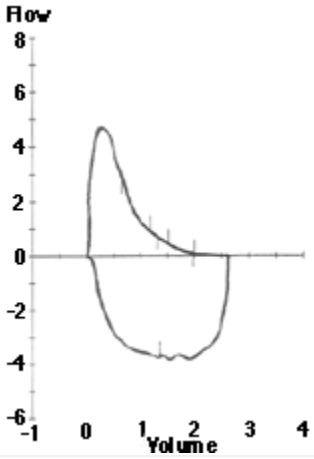
	50 - 65%	moderate
	< 50%	severe

As the obstruction becomes more severe and end expiratory air trapping develops, the forced vital capacity may be reduced as well as the FEV1; however there should continue to be a disproportionate reduction in FEV1 as evidenced by the FEV1/FVC ratio.

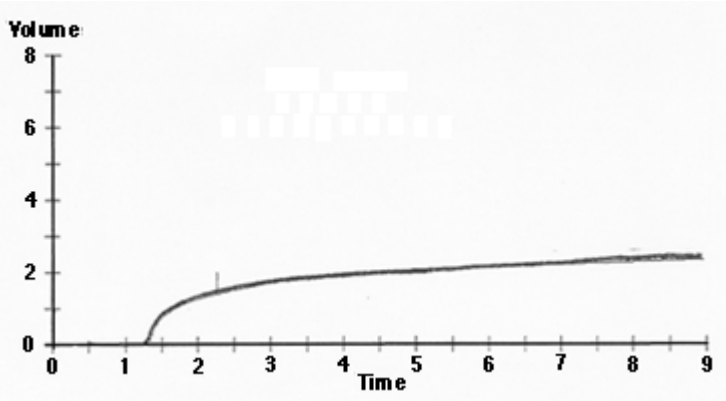
Example of spirometry results demonstrating mild obstruction:

	Measurement	Pred	%Pred
FVC	2.63	3.11	84
FEV1	1.58	2.28	69
FEV1/FVC	60	73	
FEF25-75	0.59	2.56	23
PEF	4.90	5.78	85

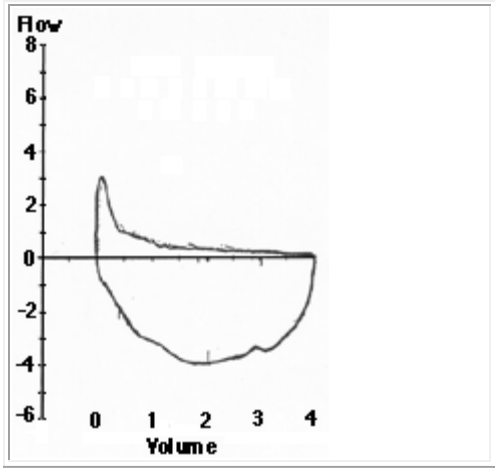
Mild Obstruction Flow Volume



Mild Obstruction Volume Time Curve



Severe Obstruction Flow Volume



Obstructive lung disease also changes the appearance of the flow volume curve. As with a normal curve, there is a rapid peak expiratory flow, but the curve descends more quickly than normal and takes on a concave shape, reflected by a marked decrease in the FEF25-75. With more severe disease, the peak becomes sharper and the expiratory flow rate drops precipitously. This results from dynamic airway collapse, which occurs as diseased conducting airways are more readily compressed during forced expiratory efforts. On the volume time curve, this is seen as a slower ascent to maximum volume, with a gradual upsloping versus the rapid rate seen in normal individuals. This equates with a prolonged forced expiratory time demonstrable on physical exam.

The ATS recommends caution in diagnosing obstruction when a patient has a reduced FEV1/FVC ratio but normal FEV1 and FVC. As mentioned above, there is a normal age-related decline in the FEV1/FVC ratio, so normal elderly patients without airway obstruction will have a ratio below 70-80%. In this case, values below the predicted FEV1/FVC ratio aid in diagnosing obstruction. The mid-range flows (FEF25-75) are always reduced in obstructive airways disease. However, some patients have normal spirometry with the exception of a reduced FEF25-75. While normal values for FEF25-75 have broader ranges than the other spirometric values, a mid-range flow less than 50% is likely to be abnormal. This is suggestive of possible small airways dysfunction and potentially early obstruction, but it should not be interpreted as meeting obstructive criteria. In the appropriate clinical setting, one may consider a trial of bronchodilators, bronchoprovocative testing to exclude asthma, or interpret this observation as a possible early indicator of smoking related lung disease.

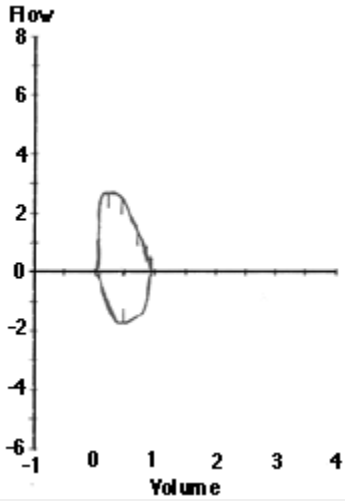
RESTRICTIVE LUNG DISEASE

In restrictive lung disease, both the FEV1 and FVC are reduced proportionately. By affecting both the FEV1 and the FVC, restrictive lung disease presents with a normal or even elevated FEV1/FVC ratio.

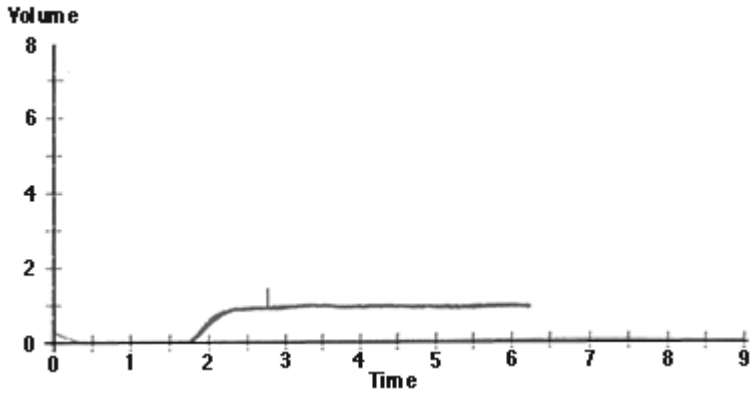
	Meas	Pred	%Pred
FVC	0.96	2.75	35
FEV1	0.94	1.90	49

FEV1/FVC	98	69	
FEF25-75	2.25	2.11	107
PEF	2.98	5.40	55

Restriction Flow Volume



Restriction Volume Time



The shape of the flow volume loop is relatively unaffected in restrictive disease, but the overall size of the curve will appear smaller when compared to normals on the same scale. Similarly, there will be a rapid upslope on the volume time curve, but such patients will reach a smaller vital capacity.

It is important to realize, however, that restrictive lung disease cannot be diagnosed by spirometry alone. With severe airways obstruction the lung volume remaining in the thorax after expiration (the functional residual capacity) may be increased to such a degree that it limits inspiratory capacity and, hence, reduces vital capacity. A reduced FEV1 and FVC are therefore only suggestive of true restrictive disease, but it is necessary to measure lung volumes to accurately diagnose restrictive physiology.

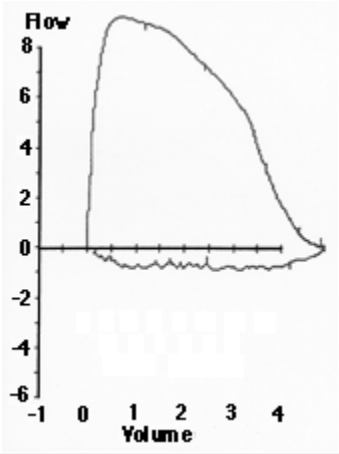
Upper Airway Obstruction

Upper airway obstruction is less common than lower airway obstruction; however it can be suggested by spirometry. Upper airway obstruction includes variable extrathoracic obstruction, variable intrathoracic obstruction, and fixed intra- or extrathoracic obstruction. These are best seen on the flow volume loops, where both inspiration and expiration can be viewed.

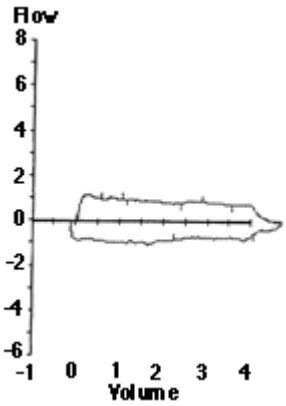
In a variable upper airway obstruction, airflow is compromised by dynamic changes in airway caliber. During normal inspiration, airways within the thorax tend to dilate as the lung inflates while airways outside of the thorax tend to collapse due to the drop in intraluminal pressure.

During expiration, the reverse is true as airways within the thorax collapse but airways outside the thorax are held open by expiratory flow.

Variable Extrathoracic Upper Airway Obstruction



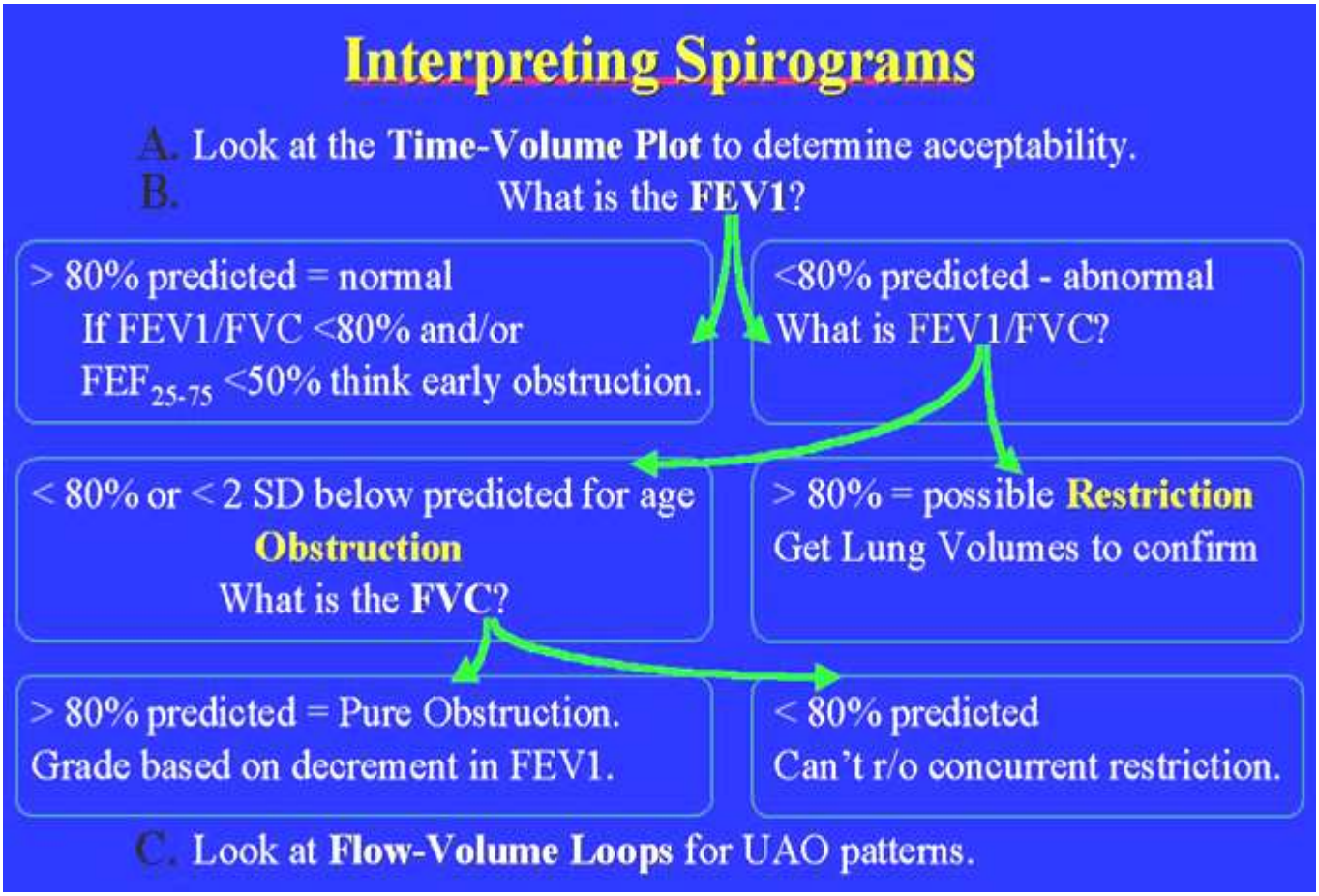
Fixed Upper Airway Obstruction



As a result, a variable extrathoracic obstruction primarily affects the inspiratory portion of the flow volume loop, viewed as a flattening of the usual deep inspiratory curve. The expiratory portion of the loop appears relatively normal. Conversely, a variable intrathoracic obstruction mainly affects the expiratory limb, again giving a flattened appearance to that aspect of the loop. This can be difficult to distinguish from the more common small to medium sized airways obstruction that characterizes bronchitis, asthma, and emphysema. Finally, a fixed intrathoracic or extrathoracic obstruction affects both inspiration and expiration, giving a flow volume loop that has an overall box-like shape as both inspiratory and expiratory limbs flatten.

Variable extrathoracic obstructions may be caused by vocal cord paralysis, thyromegaly, tracheomalacia, or neoplasm while large airways variable intrathoracic obstructions can also result from tracheomalacia or neoplasm. Examples of fixed obstruction include tracheal stenosis, foreign body, or neoplasm.

In summary, spirometry is a valuable tool for the assessment of lung disease. By ensuring proper calibration of equipment and performance of test maneuvers, one can differentiate among several different disease processes.


Interpreting Spirograms
A. Look at the Time-Volume Plot to determine acceptability.
B.
What is the FEV1?
> 80% predicted = normal
If FEV1/FVC <80% and/or
FEF25-75 <50% think early obstruction.
<80% predicted - abnormal
What is FEV1/FVC?
< 80% or < 2 SD below predicted for age
Obstruction
What is the FVC?
> 80% = possible Restriction
Get Lung Volumes to confirm
> 80% predicted = Pure Obstruction.
Grade based on decrement in FEV1.
< 80% predicted
Can't r/o concurrent restriction.
C. Look at Flow-Volume Loops for UAO patterns.

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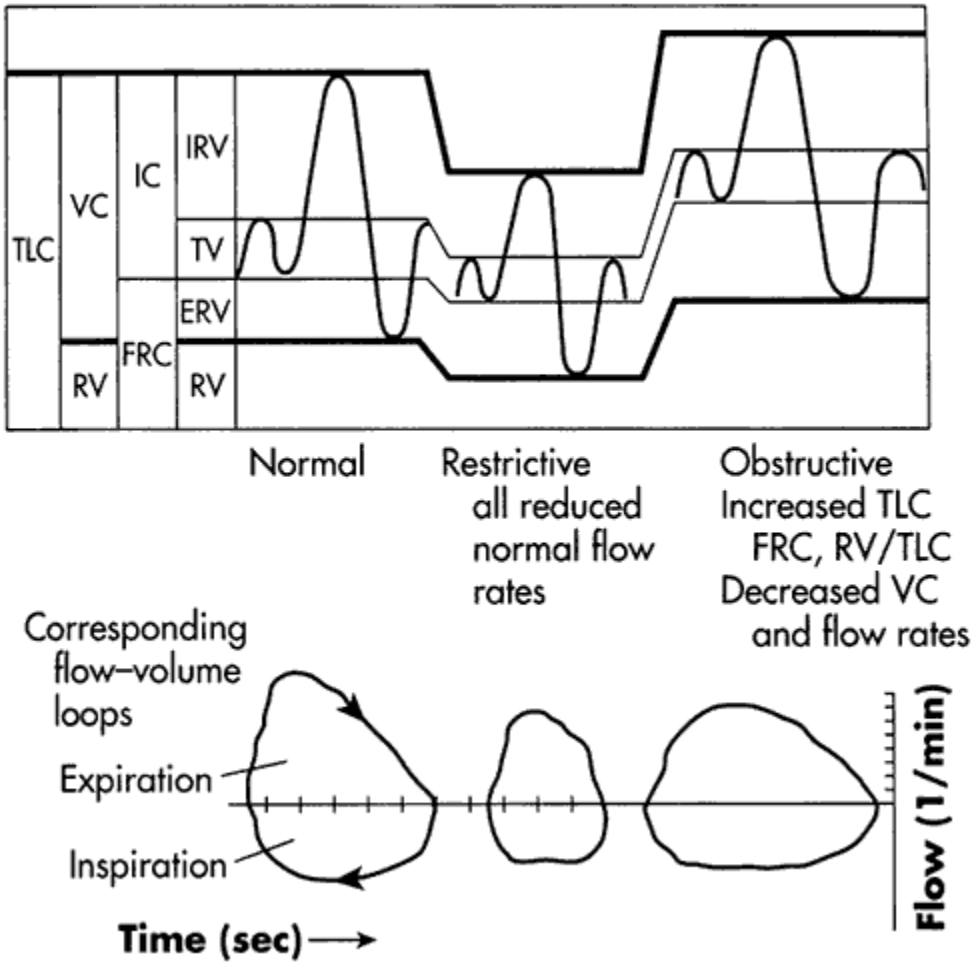
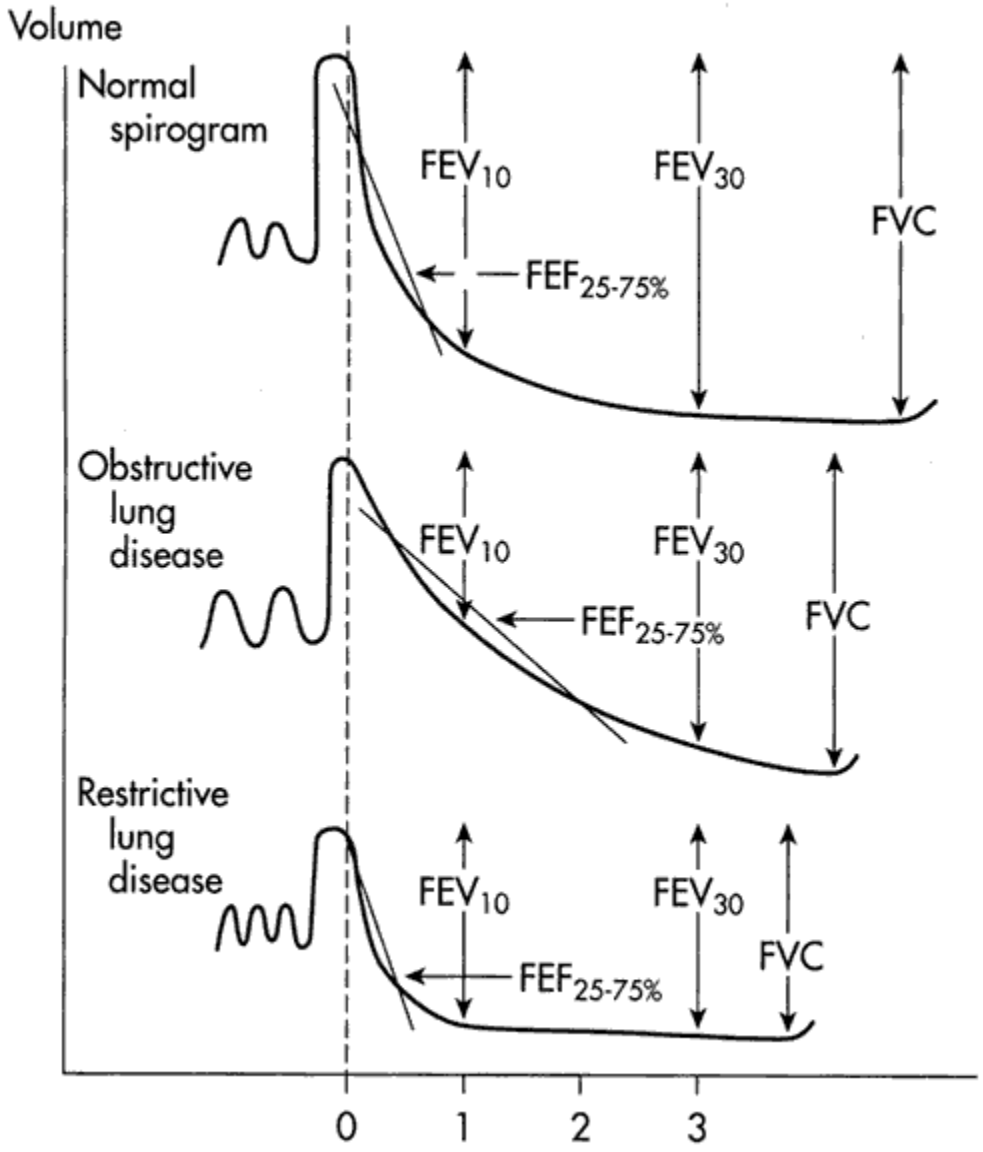
ADDENDUM:
PULMONARY FUNCTION TEST OUTLINES & REVIEW OF ASSOCITATED PULMONARY CONDITIONS



Pulmonary function tests.

I. **Spirometry** (Figure 4-2)
   A. **Forced vital capacity (FVC)** is the maximum volume of gas that can be expired forcefully after a maximum inspiration.
   B. **Forced expiratory volume in 1 second (FEV_1)** is the volume of gas expired during the first second of an FVC maneuver.
   C. **Forced expiratory flow (FEF)** 25% to 75% in middle of forced expiration.



Spirometry.

II. **Lung Volumes**

A. **Total lung capacity (TLC):** The volume in lungs at maximum inspiration.
B. **Vital capacity (VC):** The maximum volume expired after a maximum inspiration.
C. **Tidal volume (TV):** The volume inspired and expired during normal breathing.
D. **Residual volume (RV):** The volume left in the lungs after maximal expiration.

III. **Diffusion Capacity (DLCO).**
   A. DLCO measures the diffusion of carbon monoxide across the alveolar surface.
   B. Decreased by interstitial thickening (fibrosis) or decrease in alveolar surface area (emphysema, pneumonectomy, and consolidation).
   C. May be increased with significant pulmonary hemorrhage.

IV. **Average Values.**
   A. **VC** (predicted):
      I. Women 5 (21.78 - [0.101 x age in years]) x height in cm.
      II. Men 5 (27.63 - [0.112 x age in years]) x height in cm.
   B. **TV**
      I. Child: 7.5 ml/kg.
      II. Adult Female: 6.6 ml/kg.
      III. Adult Male: 7.8 ml/kg.

V. **Interpreting Patterns of Abnormal PFTs**
   A. **Obstructive Disorders.**

      1. Defined by an FEV_1/FVC of less than 0.8. Differential diagnosis includes emphysema, chronic bronchitis, and asthma.
      2. With bullous disease and air trapping, the RV and TLC will be increased, with a normal-to-decreased VC and TV.

   B. **Restrictive Disorders.**

      1. *Suggested* by a decrease in both FEV_1 and FVC, but with an FEV1/FVC ratio of 0.8 or greater. However, restrictive disease is defined by a decrease in TLC.
      2. Differential diagnosis includes interstitial lung diseases (sarcoidosis, environmental disease, pneumoconiosis, interstitial pneumonias, connective tissue disorders, pulmonary vascular diseases), fibrosis, and obesity. Also, kyphoscoliosis, status postsurgery, paralysis, ascites, pleuritis and pleural effusion.

Review of Conditions for which Pulmonary Function Tests are Ordered:

Pulmonary Medicine: Ventilators and Oxygen Therapy

I. **General.** Respiratory failure is defined as the inability of the lungs to meet the metabolic demands of the body. This can involve failure of tissue oxygenation or failure of CO_2 homeostasis, or both. Acute respiratory failure is a medical emergency requiring prompt diagnosis and management and should be suspected when a patient breathing room air has a PO_2 <60 mm Hg or a PCO_2 >50 mm Hg with a pH <7.3.

II. **Oxygen Therapy**
   A. Oxygen-delivery systems.

B. **Complications of oxygen therapy.**

1. **Pulmonary oxygen toxicity** including mucosal drying, mucociliary dysfunction, atelectasis, interstitial and alveolar edema, and alveolar hemorrhage.
2. **Decreased respiratory drive,** carbon dioxide retention, and respiratory failure in patients with chronic hypoxemia, who have a respiratory drive based on hypoxia (as in those with COPD).
3. **Retrolental fibroplasia** in neonates of low birth weight or gestational age <34 weeks.
4. **Bronchopulmonary dysplasia** in infants who require mechanical ventilation after birth.
5. **Risk of fire and explosion**.

III. **Ventilators**. Note: Initiate ventilation at 10 ml/kg tidal volume, FiO_2 of 100%, and a rate of approximately 14.

A. Indications for mechanical ventilation.

1. **Hypercapnia**. Increased PCO_2 with inability to maintain adequate alveolar ventilation. Seek treatable causes (such as narcotics). Some patients with chronic lung disease will tolerate an increased $PACO_2$, remaining awake and comfortable. However, an arterial pH below 7.1 is considered an indication for mechanical ventilation.
2. **Raised intracranial pressure**. However, hyperventilation may actually cause increased CNS ischemia and worsen brain injury.
3. **Hypoxemia.** PAO_2 will usually be improved by IPPV. Specific criteria for instituting mechanical ventilation are the following
    a. PAO_2 <40 torr on maximal inspired O_2
    b. Increasing obtundation,
    c. Rapidly progressing respiratory disease,
    d. Increased work of breathing (as with intercostal retractions during inspiration),
    e. Elevation of $PACO_2$.

B. Modes of mechanical ventilation.

1. **Volume-cycled ventilation**. A tidal volume is set, with airway pressures being increased until a set volume is delivered. Examples of volume-cycled ventilation include controlled mandatory ventilation (a fixed rate and volume are delivered), assist control (fixed minimum rate and volume are delivered with the patients respirations triggering additional breaths), and volume support. In volume-cycled ventilation, the minute ventilation is guaranteed assuring gas exchange, but the airway pressures are variable, allowing for dangerous generations of high pressures and risk of barotrauma (pneumothorax). Pressures needed depend on chest wall and lung compliance.
2. **Pressure-cycled ventilation**. An inspiratory pressure is set, with the tidal volume dependent on pressure and patient compliance. Examples include pressure-control and pressure-support ventilation. The minute ventilation can change since delivery of a set volume is not guaranteed. Hence, gas exchange can vary, making dangerous hypercapnia or alkalosis possible.

However, since inspiratory pressures are controlled, the risk of barotrauma is less. Since ventilator-associated pneumothorax may be instantly life-threatening, pressure-cycled ventilation may be preferred in patients with variable compliance (pneumonia, ARDS).

IV. **Ventilator Management**.

A. **Oxygenation**. Arterial oxygen content should be maintained at 60 mm Hg or higher, or saturations at 90% or higher. Generally, initiate mechanical ventilation with an FiO_2 of 100%, then taper 10% every 10 to 15 minutes to find the lowest FiO_2 necessary to maintain adequate oxygenation. An FiO_2 of greater than 60% for over 24 hours has been associated with lung injury. PEEP may be added to decrease the A-a gradient, allowing a lower FiO_2 while maintaining oxygenation (see below).

B. **Ventilation**. Measured by minute ventilation (= tidal volume x respiratory rate) and is reflected in the PCO_2. Increases in minute ventilation will cause a decrease of PCO_2. Goals of ventilations should be to maintain a pH (as determined by PCO_2 and underlying diseases) of 7.3-7.4.

C. **Permissive hypercapnia**. In certain situations (e.g., ARDS) it may be permissible to allow the PCO_2 to rise (permissive hypercapnia) to decrease injury from ventilation as long as the patient maintains hemodynamic stability and oxygenation. This has been shown to decrease mortality in some cases (e.g., ARDS).

D. **Minute ventilation** is the product of tidal volume and rate; it is approximately 5 to 10 L/min or 100 ml/kg/min.

E. **Tidal volume** (Vt). Initial volume is 8 to 10 ml/kg. A large Vt improves gas exchange and prevents atelectasis. However, it may decrease venous return higher volumes may increase risk of barotrauma. A smaller Vt may be required if PEEP is added.

F. **Inspiratory time and flow**. Adjust inspired flow rate to maintain a ratio of inhalation time to exhalation time of 1 to 1.5 in most patients. Patients with airway obstruction (asthma, COPD) may require additional time for exhalation. This can be accomplished by decreasing inspiratory time, or by decreasing respiratory rate.

G. **Positive end-expiratory pressure** (PEEP) may increase compliance and decrease the work of breathing by preventing atelectasis, and thereby decreasing shunting. It is usually begun at 3 to 5 cm H_2O and increased in small increments. High levels may result in decreased venous return and severe hemodynamic compromise. Other negative consequences include overventilation, barotraumas, and elevated intracranial pressure. Cardiac output should be measured if there is an indication of problems because it may increase or decrease with increased PEEP.

H. **Peak airway pressure** reflects the pressure required to overcome airway resistance and is the peak pressure during the inspiratory cycle. The alarm limit should be set 10 cm H_2O above this. If the peak inspiratory pressure increases, you need to consider obstruction in the ET tube, bronchospasm, decreased lung compliance, or a pneumothorax from barotrauma.

I. **Sedation and neuromuscular paralysis** allow the patient to rest, decrease anxiety, and ensure better compliance with the ventilator. However, periodic interruption of sedation (if tolerated) reduces the total number of days on a ventilator.

1. **Initial therapy includes** midazolam, diazepam, lorazepam and propofol. Dosages should be titrated to desired effect, with monitoring of hemodynamic and respiratory status.
2. **Neuromuscular paralysis** is occasionally necessary if sedation fails, **but patients should still be sedated.** Monitoring alarms must be functioning because ventilator malfunction is rapidly fatal if the patient is paralyzed. Immediate, short-term paralysis (3 to 7 minutes) can be achieved with succinylcholine 1 mg/kg IV. For long- term paralysis use non-depolarizing agents such as pancuronium, vecuronium, or cis-atracurium. If repeated dosing or continuous drips are necessary, consider nerve-stimulation testing to avoid over-medication. Prolonged use of these agents, especially in continuous infusions, is associated with prolonged (days to months) muscle weakness and ventilatory dependence. Use of nerve-stimulators can decrease the dose of paralyzing agents while maintaining adequate control. If necessary, a neostigmine-atropine combination can be used to reverse the non-depolarizing agents.

V. **Prevention of Respirator-Associated Complications.**

A. **Continuous subglottic aspiration** of secretions reduces the incidence of nosocomial pneumonia. A semirecumbent position in bed also will minimize the risk of ventilator-associated pneumonia. A bacteriologic diagnosis should be aggressively pursued in ventilator-associated pneumonia and will reduce mortality.

B. **Stress ulcer prophylaxis**. Sucralfate, H2 blockers, and proton-pump inhibitors have all been shown to be effective. However, sucralfate may be associated with a lower rate of ventilator associated pneumonia.

C. **DVT prophylaxis**. Heparin 5000 U SQ Q12h or LMW heparins (enox-aparin 40 mg SQ QD or 30 mg SQ Q12h) are preferred, unless contraindicated (coagulopathy, thrombocytopenia, active bleeding, recent or future surgery). Compression stockings and intermittent pneumatic devices (TEDS and Kendals) are also effective.

VI. **Withdrawal of Mechanical Ventilation.**

A. **Guidelines** for weaning from mechanical ventilation:

1. An awake, alert patient.
2. PO_2 >60 torrs, with an FiO_2 <50%.
3. PCO_2 acceptable and a pH in normal range.
4. PEEP <8 cm H_2O.
5. Minute ventilation less than 10 L/min.
6. Patient is able to generate maximum voluntary ventilation without retractions.
7. Patient is able to generate a peak negative inspiratory pressure of at least 20 cm H_2O.

B. **Weaning from the ventilator:**

1. Explain the process to the patient and encourage cooperation.
2. Begin during the daytime; allow the patient to rest at night and between trials of weaning.
3. Place the patient in an upright position.
4. Causes of weaning failure include poor respiratory or cardiac function, underlying infection, high metabolic demands, poor nutrition and energy stores, and inadequate rest.
5. Discontinue weaning if:
    a. pH <7.3, PCO_2 >50 torrs, PO_2 <60 torrs.
    b. The patient becomes anxious, fatigued, demonstrates increasing respiratory distress, or develops significant arrhythmias or hemodynamic deterioration.
6. **Methods of weaning from ventilator**
    a. Of the following three common methods, the pressure-support and T-tube methods have similar rates of success. Intermittent (IMV) has resulted in increased time to wean in some studies. However, most patients will wean successfully with any method.
    b. **Pressure-support method**. Switch from an assisted mode of breathing to pressure support, setting pressures to generate Vt similar to the assisted volumes with a ventilation rate less than 20. Gradually decrease the inspiratory pressure until 8-10 cm H_20 above expiratory pressure. If patient can maintain adequate volumes with a ventilation rate of less than 20 for 30-60 minutes, consider extubation.
    c. **T-tube method**. (A T-tube allows the patient to breathe through an endotracheal tube without assistance from the ventilator.) Have the patient use a T-tube with humidified oxygen. If the patient tolerates this for 1 to 4 hours without deterioration, discontinue mechanical ventilation. If the patient fails the attempt, resume mechanical ventilation and consider IMV method (below) for weaning.
    d. IMV method. Gradually decrease the number of assisted respirations in 1 or 2 breath increments over 30- to 90-minute intervals. Monitor ABGs and vital signs. When an assisted rate of <4 breaths/min is achieved, consider a brief T-tube trial. If the patient remains stable, discontinue mechanical ventilation. If the trial fails, increase assisted rate until patient stabilizes. Repeat attempt the following day with a more gradual decrease in the rate of assisted breaths.

Chronic Obstructive Pulmonary Disease (COPD)

I. **Definition**. A generalized increased resistance to airflow during expiration that includes chronic bronchitis, emphysema, chronic asthma, and bronchiolitis. Patients rarely have pure emphysema or chronic bronchitis. Most patients will have both processes present. COPD occurs in 10% to 15% of cigarette smokers.

II. **Chronic Bronchitis**
   - A. **Characterized** by chronic cough productive of mucus for at least 3 months in each of last 2 years with inflammatory changes in the bronchial mucosa.
   - B. **Results from** prolonged exposure to pulmonary irritants including cigarettes, allergens, pollution, and recurrent infections.

III. **Emphysema**
   - A. **Characterized by** destruction of the lung parenchyma beyond the terminal bronchioles with coalescence of alveoli.
   - B. **Divided into:**
     1. **Panlobular**, which is the result of alpha1-antitrypsinase deficiency.
     2. **Centrilobular**, which is the result of smoking and of chronic bronchitis.

IV. **Diagnosis**
   - A. **Diagnose with pulmonary function tests**, including spirometry (before and after bronchodilators), lung volumes, and DLCO.
   - B. **Spirometry** shows obstructive airflow patterns, generally nonreversible (see section on PFTs). Although degree of decrease in FEV1 does not correlate with symptoms, it a strong prognostic factor. When FEV_1 is less than 1 L, 5-year survival is 50%.
   - C. **Diffusion capacity** is decreased in emphysema; may be normal or low in chronic bronchitis.
   - D. **Lung volumes** may show hyperinflation (increased TLC) and air trapping (increased RV).
   - E. **CXR may be normal**, show evidence of bullous disease, hyperinflation, or increased interstitial markings from chronic airway inflammation.
   - F. **Blood gases** can show hypoxia and/or compensated hypercapnia (normal pH) in late stages.
   - G. **Laboratory data**
     1. **With acute exacerbations**, CBC, ABG, CXR, and ECG may be indicated, depending on the clinical situation.
     2. **Hypercapnia with acidosis** is suggestive of acute decompensation.
     3. **A high CO_2 level with a normal pH** is suggestive of a compensated chronic state.
     4. **ECG** may show multifocal atrial tachycardia, and if it is present, one should be aware of the possibility of theophylline toxicity.
     5. **Bedside peak flows** can document response to treatment.

V. **Cor pulmonale** can result from long-standing COPD. Essentially, vascular tone in the lungs increases leading to right heart failure. Patients present with leg edema, JVD, and possibly a palpable liver with elevated liver enzymes. This may respond to long-term oxygen therapy.

VI. **Acute Treatment of COPD**

A. **Treatment of COPD.**

B. **Treat any underlying infection**. (See section on pneumonia later.)

C. **Hospitalize** if clinically indicated (worsening tachypnea, falling O_2, acidosis, increasing CO2.)

D. **Beware if the patient seems too calm.** It may indicate CO_2 retention with CO_2 narcosis. This can occur as the result of giving high-flow O_2 to a patient with chronic COPD. **However, do not withhold oxygen from a symptomatic, hypoxic patient even if it means the need for intubation.**

VII. **Long-term Management of COPD**

A. **Immunization**. All patients with COPD should have an annual flu shot and Pneumovax every 5 years.

B. **Patients should be educated** about their disease and taught that if they start having difficulties with breathing, they should contact their health care professional.

C. **Pulmonary rehabilitation**, including conditioning and breathing techniques, improves lifestyle.

D. **Low-flow O_2** has been shown to be useful in reducing pulmonary arterial resistance, which if uncontrolled, leads to cor pulmonale.

1. **Use continuous oxygen** in those with either a PO_2 <55 mm Hg, an O_2 saturation of <89%, or a PO_2 <59 mm Hg with evidence of cor pulmonale (peripheral edema, HCT >55, P pulmonale on ECG).
2. **O_2 saturation should be kept above 90%** (PO2 of 60 to 80 mm Hg). This usually can be accomplished with 2 L/min O_2 but titrate to patient's needs.
3. **Survival significantly enhanced with oxygen** use 24 hours a day. Patients should be encouraged to use oxygen at least 15 hours a day.

E. **Inhaled bronchodilators**. The use of these medications is predicated on instruction of the patient in their proper use. A spacer should be used with most metered dose inhalers. Additionally patients should be given instructions about what to do if they notice a need for increased amounts of medication to remain symptom free or if their symptoms persist despite the use of normal doses of bronchodilators. Since response varies over time, even those with a poor response on post-bronchodilator PFTs should be given a long-term trial of inhaled bronchodilators.

1. **Beta-adrenergic agonists** (such as albuterol, metaproterenol) to be used PRN for symptoms.
   a. Many of those with COPD are resistant to beta-agonists and will respond better to inhaled anticholinergics (below).
   b. **Dose**. One nebulizer of albuterol (2.5 mg) is equal to about 6 to 8 puffs of an MDI via spacer. Patients can use 4 to 8 puffs every 3 to

4 hours as needed but should be instructed on what to do (such as start steroids by mouth) if their need for albuterol increases.

c. **Long-acting beta-agonists**, such as salmeterol, may be used to prevent nocturnal symptoms and to control symptoms during the day. However, salmeterol is not useful for the control of acute symptoms. Formoterol is an alternative.

2. **An anticholinergic agent** (such as ipratropium) is the bronchodilator of choice in COPD. Besides bronchodilation, it also limits secretions, which can cause mucous plugging. The normal dose is 2 puffs every 6 hours. It is also available for nebulization in higher doses. The equivalence of MDI versus nebulization is not as well worked out as it is for beta-agonists. However, it probably similar and higher doses of an MDI may be helpful. Anticholinergics have an effect that is additive to the inhaled beta-agonists. Combined beta-agonist/ anticholinergic inhalers are available (Combivent). Tiotropium, a QD anticholinergic, will soon be available in the United States.
3. **Inhaled steroids** (such as beclomethasone) should be used in patients that show some degree of reversibility to beta-agonists. Only about 15% of patients with COPD will respond to inhaled steroids. Generally, those who are responsive to inhaled beta-agonists and have acute attacks of dyspnea will be responsive to steroids.
4. **Oral steroids** (such as prednisone) are helpful in the subset of COPD patients with reversible disease (see above) but have many side effects and should be reserved for acute exacerbations and for those who do not respond to inhaled steroids.
5. **Leukotriene inhibitors** have no role in COPD.
6. **Theophylline** has an extremely limited role in COPD due to its low benefit and high potential for complications in an elderly patient population (heart disease, drug-drug interactions, increased drug clearance).
7. **Long-term antibiotics are not helpful** but should be used to treat acute purulent exacerbations. For patients with recurrent purulent exacerbations, antibiotics for 7 days each month may prevent need for hospitalization. Amoxicillin or amoxicillin/clavulanate 500 mg TID, trimethoprim-sulfamethoxazole (Septra DS) 1 PO BID, or doxycycline 100 mg PO BID are recommended.
8. **Lung reduction surgery** may be appropriate for some patients (under age 75, no longer smoking, no cor pulmonale) and improves FEV1 and symptoms. Randomized trials of survival are currently underway.

Asthma

I. **Definition**.

A. Asthma is an inflammatory disease of the airways characterized by reversible airway obstruction. Asthma, nasal polyps, and aspirin sensitivity make a common clinical triad.

II. **Diagnosis**.

A. **History**.

1. **Symptoms** include wheezing, cough, chest tightness, and shortness of breath.
2. **Exacerbating factors** include sinusitis, reflux esophagitis, viral URI, and allergens (including dust and cockroach exposure).
3. **Episodic bronchospasm may be secondary** to exacerbating factors such as exercise, cold, exposure to smoke or workplace chemicals, etc.

B. **Physical Exam**. The physical exam may be normal between exacerbations. Wheezing, prolonged expirations, depressed diaphragms (hyperinflation) may be present especially with acute exacerbations. Look for signs of sinusitis, nasal polyps, and GE reflux.

C. **Additional testing**.

1. **Pulmonary function testing** is the mainstay of asthma diagnosis. Spirometry shows airway obstruction, which reverses by 12% or greater after inhaled bronchodilators. However, spirometry may be normal between exacerbations. Chronic asthma may lead to fibrosis and irreversible airflow obstruction.
2. **Serial peak flow readings** taken at home (e.g. before and after work or school) may show airflow variability.
3. **Bronchoprovocation** with methacholine or histamine may help establish the diagnosis.
4. **Skin testing** for common allergens may be indicated for those with moderate, persistent asthma since immunotherapy may be useful.
5. **Specific testing** for sinusitis (rhinoscopy) or reflux (pH probe) to identify contributing factors may be indicated.

III. **Chronic Disease Severity**. The National Asthma Education Program (and National Heart Lung and Blood Institute) classifies chronic disease severity into four levels. Patients are classified according to the most severe class in which they have any features. Additionally, patients at any level of severity can have mild, moderate, or severe exacerbations.

A. **Mild intermittent asthma**. Symptoms occur fewer than 3 times/week, nocturnal symptoms fewer than 3 times per month, with normal FEV1, and peak flows that vary less than 20%.

B. **Mild persistent asthma**. Symptoms occur 3-6 times/week, nocturnal symptoms 3-4 times month, with normal FEV1, and peak flow varies 20%-30%.

C. **Moderate persistent asthma**. Daily symptoms, nocturnal symptoms 5 or more times per month, with FEV1 60% to 80% predicted, and peak flow varies 30% or more.

D. **Severe persistent asthma**. Continual symptoms, frequent nocturnal symptoms, FEV1 less than 60% predicted, and peak flow varies 30% or more.

IV. **Management**

A. **General Principles of Management**. Anti-inflammatory medications should be considered the mainstay of asthma therapy. Medications used for maintenance and rescue therapy are different.

B. **Written Plan. All patients should have a written plan for dealing with exacerbations (e.g. start oral steroids, increase beta-agonists, and parameters for calling their health care provider).**

C. **Inhalers**. All hand-held inhalers should be administered via the use of a spacer, or by holding the inhaler 1 inch from the patient's lips. Inhalers require initial training and periodic review of technique in order to provide adequate treatment and minimize side-effects.

D. **Monitoring**. Signs and symptoms of asthma are quite subjective. Therefore disease monitoring for those with more than mild asthma (both chronic and acute) should include objective measurement of airflow obstruction. This includes home peak flow meters, as well as intermittent formal spirometry depending on the severity of disease. See Figure 4-1 and Figure 4-2 for normals.

E. **Indicators of control**. Indicators of poor asthma control include nocturnal symptoms, urgent care visits, and increased need for rescue inhaled beta 2-agonists (daily use for a week or more than one canister per month)

V. **Chronic Medications**

A. **Anti-inflammatory drugs** are the mainstay of asthma therapy.

1. **Inhaled corticosteroids**. Indicated for all severities of asthma with the exception of mild intermittent asthma. Different formulations have different potencies; beclomethasone <triamcinolone <fluticasone. However, clinically, the effectiveness is equal in equipotent doses.
2. **Cromolyn and nedocromil**. May be used as monotherapy in children <5 years old with mild persistent asthma. However in adults, and children with moderate or severe asthma, these drugs should be an adjunct to inhaled corticosteroids to assure adequate anti-inflammatory activity.
3. **Leukotriene inhibitors**. The role of leukotriene inhibitors is not well-defined, but they are not recommended for first-line use. They are no more effective than inhaled steroids but have more potential toxicity, including Churg-Strauss syndrome (also known as "allergic granulomatosis and angiitis"). Churg-Strauss syndrome is a necrotizing angiitis. It occurs in those with preexisting asthma or allergies. Manifestations include pulmonary nodules with an abnormal CXR (25%), skin purpura and nodules, and a mononeuritis multiplex. Renal involvement is rare. Patients usually have a peripheral eosinophilia and a positive p-ANCA. The illness responds to steroids and the prognosis is good. Leukotriene inhibitors are not recommended at all for children less than 12 years of age (although montelukast can be used in those as young as 6 years old). Examples include zafirlukast (Accolate) and zileuton (Zyflo).
4. **Oral corticosteroids**. With adequate dosing of above anti-inflammatory agents, chronic systemic steroids are rarely needed. If used, attempt to wean to minimum dose or alternate day dosing to limit side effects.

B. **Long-acting beta$_2$-agonists**. Examples include salmeterol (inhaled) and extended release albuterol (systemic), as well as famoterol. May be particularly useful for nocturnal symptoms. Salmeterol has been shown to decrease the needed dose of inhaled corticosteroids, but in most cases should not be used as a replacement for

steroid inhalers. Also important to instruct patients that long-acting agents should not be used for rescue therapy.

C. **Short-acting beta$_2$-agonists**. Scheduled use of short-acting bronchodilators is generally not recommended. Control with scheduled use of beta-agonists is not as good as that achieved with PRN use. Short-acting beta2-agonists (e.g., albuterol, terbutaline, pirbuterol) should be used PRN for wheezing and symptom exacerbation, before exercise, etc. There is no advantage to levalbuterol, which is the L isomer of albuterol. The same clinical results can be obtained at a lower cost by increasing the dose of albuterol.

D. **Theophylline**. May add additional long-term control, but due to significant side-effects and low therapeutic window, theophylline is a third-line agent behind inhaled corticosteroids and long-acting beta$_2$-agonists. If used, serum drug levels should be monitored regularly and maintained at 10 to 15 μg/ml.

VI. **Quick Relief (Rescue) Medications**.

A. **Short-acting beta$_2$-agonists**. Inhaled albuterol, terbutaline, pirbuterol; available as both metered dose inhalers, as well as nebulized solutions. Inhaled bronchodilators, such as metaproterenol or epinephrine (available over the counter), should be discouraged due to potential for excessive cardiac stimulation from nonselective adrenergic properties. Patients can safely use up to 6 or 8 puffs every 2 hours but should contact their health care practitioner if increasing doses are needed.

B. **Anticholinergics. Ipratropium bromide**. Produces bronchodilation by reducing vagal tone to airways. May be particularly helpful in conjunction with beta-agonists.

C. **Methylxanthines**. Theophylline and aminophylline are not recommended for rescue medications; they provide no additional benefit over inhaled beta- agonists, and may produce adverse effects.

Bronchiectasis

I. **Definition**. Bronchiectasis is a chronic dilatation and inflammation of medium-sized bronchi. Clinically it looks similar to chronic bronchitis with chronic mucopurulent sputum production. However, sputum production is often copious and may contain *Pseudomonas* organisms. Occurs mostly in the left lower lobe followed by the lingula and the right middle lobe with hemoptysis and recurrent pneumonia in a single lobe.

II. **Predisposed** to by recurrent pneumonia, granulomatous disease, carcinoma, or any process that can lead to a sequestered lobe.

A. **CT scanning** will demonstrate areas of bronchiectasis (enlarged peripheral airways with thickened airway walls).

B. **Treat as for COPD**. A 6-month course of antibiotics (e.g., amoxicillin/clavulanate or doxycycline) initially may help eradicate underlying infections. Patients with bronchiectasis may benefit from a course of antibiotics for the first 10 days of every month (such as TMP/SMX or doxycycline).

C. **Surgical resection** of affected lobe is alternative treatment if antibiotics fail.

Pneumonia

I. **General**. An infection of the lower respiratory tract, including bacterial, viral, or fungal etiologies. Clinical manifestations include fevers, cough with or without sputum production, shortness of breath, and pleuritic chest pain. Physical exam may reveal crackles (rales) in the lungs with dullness to percussion and egophony. Infiltrates with or without pleural effusions may be seen on CXR. Patients may have few of these findings, particularly the very young or old, or those with other chronic illnesses. Additionally, these findings are nonspecific, and other diagnoses deserve consideration. **In children, tachypnea is the most sensitive physical finding and warrants an x-ray even in the absence of rales, etc.** The differential diagnosis includes heart failure, malignancy, pulmonary embolism, pulmonary vasculitis, eosinophilic pneumonia, and inflammatory lung diseases, etc.

II. **Diagnosis**

A. **Diagnosis is based** on physical exam, CXR (PA and lateral), and clinical scenario (see below for differential diagnosis of pulmonary infiltrates). However, the clinical exam for pneumonia is imprecise (even among pulmonologists) and a CXR should be done if there is any question. **The appearance on CXR (lobar versus diffuse) does not reliably differentiate between typical and atypical pneumonia. Base treatment on age, etc (see below), and not on appearance.** CBC with WBC differential may be helpful. Arterial O_2 saturation should be obtained by pulse oximetry. Send a blood gas to assess for adequacy of ventilation in those who have a low O_2 saturation or appear particularly dyspneic, cyanotic, etc.

B. **For outpatients**, attempts at microbiologic diagnosis are not time nor cost effective.

C. **For inpatients**, most feel attempts at defining the etiology are warranted, but a specific pathogen is identified in only 50% of patients even with extensive evaluation. Treatment should not be delayed awaiting results. Gram stain of sputum analysis may be helpful, but be aware of significant false negatives and false positives (especially for *Streptococcus pneumoniae*). Sputum cultures are similarly imprecise. Blood cultures should be obtained and are frequently positive in pneumococcal pneumonia. A diagnostic thoracentesis should be done if effusion is present. *Legionella* urinary antigen, *Mycoplasma* antibodies, and other pathogen-specific tests might be helpful.

D. **Immunosuppressed** patients have a high incidence of atypical pathogens not covered by standard regimens, and early bronchoscopic lavage or biopsy should be considered (see Chapter 11).

III. **Management**

A. **Antimicrobial therapy** is based upon culture results when possible. Empiric therapy is based upon patient population and regional variances. Specific populations are discussed below.

B. **Viral influenza**, type A or B, is a common cause of pneumonia, especially in winter months. See viral respiratory infections for management.

C. **Bacterial pneumonia**. Treatment should continue for 7-21 days, depending on severity and etiology. Intravenous antibiotics may be switched to oral agents 24-48 hours after defervescence.

D. **Course**. Patients may continue to be febrile 3 days into treatment, but up to 7 days for gram-negative infections and in patients with comorbid conditions. Therefore treatment changes are not indicated as long as the patient is not worsening.

E. **The chest x-ray may lag** behind clinical symptoms, but a change in therapy is not indicated if the patient is stable. X-ray resolution will be evident in 73% at 6 weeks and 94% at 24 weeks. Most patients should be followed radiographically to assure resolution.

IV. **Management of Patients Who Do Not Respond to Initial Therapy**

A. **If the patient deteriorates**, or fails to resolve, consider further testing and change in treatment.

1. **Consider resistant organisms**, such as *Staphylococcus aureus, Haemophilus influenzae*, atypical agents (*Legionella, Chlamydia*). Also viral pneumonia (influenza A and B).
2. **Consider unusual pathogens**. Histoplasmosis in Midwest/Ohio river valley, *Coccidioidomycosis* in southwestern US, psittacosis with bird exposure, Q fever with various farm animal exposures, tuberculosis, etc.
3. **Consider noninfectious diagnoses** such as the following:
    a. **Unifocal infiltrates**. Pulmonary contusion, pulmonary embolism, alveolar cell carcinoma, pulmonary hemorrhage, lymphoma, radiation pneumonitis, lipoid pneumonia, rarely lobe torsion, foreign body.
    b. **Multifocal infiltrates**. Hypersensitivity pneumonitis (see below), tuberculosis, fungal infections, alveolar cell carcinoma, Hodgkin's lymphoma, eosinophilic pneumonia, allergic granulomatosis and angiitis, collagen-vascular diseases (e.g rheumatoid arthritis, lupus, scleroderma), Wegner's granulomatosis, silicosis, black lung disease, amyloidosis, sarcoidosis.
    c. **Other illness**. This list is not inclusive.
4. **Re-image chest,** particularly looking for effusions, abscess, tumor causing a post obstructive pneumonia. Further testing with CT, bronchoscopy, or open lung biopsy may be useful.

V. **Resistant Organisms**

A. ***S. pneumoniae***. Penicillin-resistant *S. pneumoniae* (PRSP) is becoming more prevalent, but this depends on the region. Be sure to differentiate between intermediate resistance (MIC <4) and high resistance, as most beta-lactam antibiotics achieve high levels in the lung. Studies have shown that even in the face of intermediate resistance, outcomes are still good in patients treated with beta-lactam antibiotics. In cases of known high resistance or endemic areas, consider vancomycin or extended spectrum fluoroquinolone until sensitivities return.

B. ***H. influenza***. Chronic colonizer in smokers. Most have beta-lactamase, therefore beta-lactamase inhibitors needed such as clavulanate or sulbactam. Also resistant to erythromycin, but clarithromycin and azithromycin give good coverage.

C. ***Staphylococcus aureus***. Resistant to most beta-lactams, except synthetics like methicillin and nafcillin, or those with a beta-lactamase inhibitors. Where methicillin-resistant *Staphylococcus aureus* (MRSA) is prevalent, consider vancomycin.

VI. **Empiric Treatment Based on Patient Population**

A. **Neonates <5 days old**. Caused by maternal vaginal flora, including Group A and B *Streptococcus, E. coli, Chlamydia, Treponema*. Treat with ampicillin and gentamicin or third-generation cephalosporin.

B. **Neonates (5 days to 1 month)**. Group A or B *Streptococcus, S. aureus, E. coli, Chlamydia*. Treat with penicillinase-resistant penicillin (nafcillin). Consider vancomycin if MRSA is prevalent. For cough and infiltrates without a fever, consider *Chlamydia trachomatis* even in the absence of conjunctivitis. These children should be treated with a macrolide.

C. **Children (1month to 5 years)**. 80% of mild-to-moderate cases are viral. Bacterial causes include pneumococcus, H. influenzae, also chlamydia or *Mycoplasma*. Treat with extended spectrum macrolide for outpatients (e.g., clarithromycin, azithromycin). Consider third-generation cephalosporin ± aminoglycoside for inpatients.

D. **Children over 5 years**. Same as adults without comorbid factors.

E. **Adult outpatients without comorbid factors**. Most common agents are pneumococcus, *Mycoplasma, Chlamydia pneumoniae.* Treat with macrolide (tetracycline if intolerant). Must also cover *H. influenzae* in smokers, so use extended spectrum macrolide (azithromycin or clarithromycin).

F. **Adult outpatients with comorbid factors** (smoking, age >60, diabetes, emphysema, heart disease, etc). If multiple comorbid factors, consider inpatient treatment. Common etiologies same as those without comorbid factors, but increased prevalence of gram-negative rods and Moraxella. Treat with TMP/SMX + macrolide or Augmentin + macrolide. Extended spectrum macrolide or extended spectrum fluoroquinolone (e.g., levo-floxacin) may be used as monotherapy.

G. **Adult inpatients not requiring ICU**. Similar organisms but increased incidence of Legionella and gram-negative rods. Treat with third- generation cephalosporin + macrolide, beta-lactam with inhibitor + macrolide. Consider monotherapy with extended spectrum macrolide (azithromycin IV) or extended spectrum fluoroquinolone (e.g., levofloxacin).

H. **Adult inpatients requiring ICU**. Most common agents are pneumococcus, gram-negative rods, and *Legionella. Mycoplasma* in elderly. Therapy includes a macrolide plus a third-generation cephalosporin. Consider adding an aminoglycoside to cover gram-negative rods especially if the patient is hypotensive.

I. **Adult, hospital-acquired pneumonia**. As patients remain in the hospital, the oropharynx become increasingly colonized with gram-negative rods and MRSA. Therefore, for hospital acquired pneumonias that develop within the first 2-5 days

after admission, treat with a third-generation cephalosporin or fluoroquinolone. After this, aggressive gram-negative rods are common so treat with two anti-pseudomonal agents (aminoglycoside or ciprofloxacin + antipseudomonal beta-lactam like piperacillin, piperacillin-tazobactam, imipenem), consider adding vancomycin for MRSA.

VII. **Atypical Pneumonia Syndrome**

A. Cough may be nonproductive and often have associated pleuritic pain, URI symptoms, fever, headache, and myalgias. Symptoms are usually milder than with bacterial pneumonia. See also section under pneumonia for other possible diagnoses. Common in school-age children and young adults.

B. **Organisms**. Usually *Mycoplasma*, others can include TWAR (*Chlamydia pneumoniae*), Q fever, *Chlamydia psittaci* (especially in bird owners), influenza A and B, adenovirus, RSV, Legionnaire's disease, *Pneumo-cystis carinii* pneumonia (PCP) in the immunocompromised, etc, as well as differential diagnosis of infiltrates in previous pneumonia section.

C. **Treatment**. As for all pneumonia above.

Hyperbaric Oxygen Therapy

INTRODUCTION

The purpose of this CEU is to present healthcare professionals with an overview of hyperbaric oxygen therapy and the care required before and after this therapeutic intervention. There will be some repetition in the review of the topics since we have looked at HBO from several different perspectives. We hope you enjoy studying about this therapeutic intervention.

LEARNING OBJECTIVES

Upon successful completion of this course, you should be able to:

1. Identify and discuss the uses and the relative and absolute contraindications of hyperbaric oxygen (HBO) therapy.

2. Identify and explain the concepts and physiology underlying HBO therapy, as well as potential side effects.

3. List and discuss the key elements of planning and implementing care for pre- and posttreatment patients.

BRIEF HISTORY AND DEFINITION OF HBO THERAPY

The air we breathe contains 21% oxygen. Providing 100% oxygen by face mask has certain benefits but providing pure oxygen in a pressurized chamber offers distinct therapeutic benefits. Hyperbaric oxygen therapy is the administration of 100% oxygen at two to three times atmospheric pressure. The pressure of the air at sea level is considered to be one atmosphere. Each 33 feet below sea level is considered another atmosphere. For example, ifone dives 99 feet below the surface of the water, one would have dived to a pressure of four atmospheres. This pressure can be created artificially in specially designed chambers.

Hyperbaric oxygen therapy has been used for over a century, but not to any great degree until only a short time ago. Hyperbaric chambers have been in use since 1662, but have only been used clinically since the mid 1800's. Their use in treating deep sea divers with decompression sickness was discovered in the 1930's and clinical trials in the 1950's demonstrated even more benefits from HBOT.

The most widely-known application of hyperbaric oxygen therapy is in the treatment of decompression illness, also known as "the bends," which can occur when scuba diving. Today hyperbaric oxygen is used for a multitude of illnesses and conditions, but its use remains controversial. Some of the controversy surrounding the use of hyperbaric oxygen therapy is its application for "unapproved" conditions and as a "health rejuvenation tool." (More history to be presented later in this CEU)

MECHANISMS OF ACTION

Hyperbaric oxygen therapy has multiple effects on the body which include:

- **Pressure:** Any free gas trapped in the body will decrease in volume as pressure exerted on it increases (Boyle's Law). This is useful in the treatment of decompression sickness and arterial gas embolism.

- **Hyper-oxygenation:** The elevated pressure in the chamber increases the amount of oxygen present in the blood ten to thirteen times normal. The elevated level of oxygen supports compromised tissues having marginal blood flow. The flooding of the body with oxygen forces toxins like carbon monoxide from the body.

- **Vasoconstriction:** Elevated oxygen levels cause vasoconstriction, which causes reduced blood flow without affecting tissue oxygenation because of the extra oxygen in the blood. This aids in reducing edema, thus controlling compartment pressures in crush injuries, and is also effective in treating thermal burns.

- **Angiogenesis:** Hyperbaric oxygen therapy promotes the growth of new blood vessels.

- **Bactericidal:** Saturation of the tissues with oxygen slows the spread of certain toxins and is effective in killing anaerobic bacteria. Many of the body's bacterial defense mechanisms are oxygen dependent. When tissue oxygen drops, leukocyte effectiveness is decreased. Because of this, the beneficial effect of HBOT is utilized in the treatment of gas gangrene and necrotizing infections.

- **Anti-ischemic:** Hyperbaric oxygen physically dissolves extra oxygen into the plasma (Henry's Law). The quantity of oxygen carried to ischemic tissue is increased thus promoting healing.

The number of treatments and the time between treatments varies depending on the condition. Acute conditions may require treatment for ten days or less, while chronic conditions may require months of therapy. Most sessions last two hours (including compression and decompression time) and are administered once or twice a day on an outpatient basis if possible.

TYPES OF CHAMBERS

There are over 208 hyperbaric oxygen therapy centers in the United States and Canada, some of which are independent centers and others being hospital-based centers. There are three basic kinds of chambers in these centers: monoplace, dualplace, and multiplace.

Monoplace chambers accommodate one patient. They are pressurized with 100% oxygen which the patient breathes directly. The chamber is capable of providing monitoring, fluid resuscitation, and ventilatory support.

Dualplace chambers accommodate two persons: either two patients or a patient and an attendant. They are pressurized with air and the patient breathes oxygen through a built-in system. This system utilizes a large, clear hood placed over the patient's head to deliver 100% oxygen.

Multiplace chambers accommodate between four and twenty-four patients. They also are pressurized with air and the patients breathe oxygen through a similar built in system as the dualplace chamber. These chambers are large enough for stretchers, critical care patients, and medical staff.

All chambers have the capability of communication with those outside through an intercom system. Most chambers allow the patient to sleep, watch TV, or listen to music during the session.

ASSOCIATED AGENCIES

There are two primary agencies that oversee the use of hyperbaric oxygen therapy. The American Board of Hyperbaric Medicine certifies physicians as hyperbaricists. These physicians are specially trained in the use of hyperbaric oxygen therapy.

The Undersea and Hyperbaric Medicine Society (UHMS) was founded in 1967 and is the major scientific society for hyperbaric oxygen therapy in the United States. The Hyperbaric Oxygen TherapyCommittee of the UHMS, developed in 1976, reviews published data regarding hyperbaric oxygen therapy and publishes periodic reports of recommended medical indications for hyperbaric oxygen therapy. The UHMS categorizes diseases by their need for treatment with hyperbaric oxygen therapy. The criteria for each category are:

CURRENTLY ACCEPTED CONDITIONS

Disorders Involving Gases

Decompression Sickness: Decompression sickness (the bends) is the most widely known condition for which hyperbaric oxygen therapy is prescribed. This condition primarily affects scuba divers and is seen frequently in such areas as California and Florida. Decompression sickness occurs when there is production of nitrogen bubbles in the circulation. As the diver ascends to the surface, the underwater pressure decreases. Nitrogen bubbles form in the bloodstream when the pressure decreases too rapidly and these bubbles migrate to the joints causing extreme pain. These gas bubbles may embolize, travel to the brain, and cause death. By returning the diver to a simulated depth, the return to normal atmospheric pressure can be made in a slower, more controlled manner. This allows the nitrogen to be exhaled rather than accumulating and causing distress.

Embolism: Air or gas embolism is similar to decompression sickness. Gas pockets that develop in the vessels obstruct, them causing perfusion problems. Treatment theory is based on Boyles' Law. Reduction in the volume of the gas bubble allows it to pass through the circulation easier, thereby relieving any obstructions.

Carbon Monoxide Poisoning and Smoke Inhalation: Hyperbaric oxygen therapy is also used in the treatment of carbon monoxide poisoning and smoke inhalation. By flooding the body with oxygen, toxic substances are rapidly eliminated from the plasma, and oxygen replaces the carbon monoxide in the red blood cells. Most hyperbaric oxygen therapy treatment centers treat carbon monoxide poisoning when the patient presents with symptoms of carbon monoxide toxicity. Sixty-two percent of facilities, however, hold to a minimum level of carbon monoxide presence as admission criteria to their centers.

Category I: □Disorders in which HBO therapy is definitely helpful.
Category II: □Disorders with which HBO therapy is useful as an adjunct therapy.

Category III: Conditions in which HBO therapy may be helpful, but has not been proven as a satisfactory treatment.
Category IV: Conditions in which HBO therapy would be theoretically beneficial, but has not yet been studied.

WOUND MANAGEMENT

Effects of HBO Therapy

Hyperbaric oxygen therapy has many effects that aid in wound management. Oxygen is lethal to anaerobes. Because oxygen supplied under pressures greater than one atmosphere increases tissue oxygen tensions.
Hyperbaric oxygen therapy also increases oxygen content and diffusion capability, increasing tissue oxygen tensions. Hyperbaric oxygen therapy has also been shown to increase the destructive ability of leukocytes. It enhances connective tissue regeneration through the stimulation of fibroplast growth and increased collagen formation.

Further benefits of hyperbaric oxygen therapy include decreasing tissue edema and promoting revascularization. Lastly, wound management is further aided by the inhibition of toxin formation of bacteria.

***Clostridia* Myonecrosis**: *Clostridia* myonecrosis, otherwise known as gas gangrene, is a severe infection caused by gram positive bacteria of the *Clostridia* variation. This is a severe complication of trauma, fractures and open wounds. Approximately 67,000 amputations are performed each year among diabetics due to gangrenous wounds. The *Clostridia* bacteria produce exotoxins that destroy living tissues. Because oxygen supply to tissues is reduced in the presence of this bacteria, hyperbaric oxygen therapy is provided to increase tissue oxygen tensions. This inhibits toxin formation and destroys the anaerobic *Clostridia* bacterium, thereby reducing the need for amputation.

Necrotizing Soft Tissue Infections: HBO therapy slows both aerobic and anaerobic bacterial growth: it increases leukocyte effectiveness, thereby decreasing aerobic bacterial growth, and the increased oxygenation directly inhibits anaerobic growth. Use of HBO therapy can reduce mortality significantly.

Other Wounds: Other types of wounds that can be successfully treated with adjunct hyperbaric oxygen therapy include:
- Crush injuries (benefit shown in approximately 60% of cases if initiated within six hours of injury)
- Compartment syndromes
- Acute traumatic ischemias
- Refractory osteomyelitis (benefit shown in approximately 60% to 85% of cases)
- Radiation tissue damage
- Thermal burns
- Compromised skin grafts and flaps
- Diabetic wounds

ANEMIA TREATMENT

Hyperbaric oxygen therapy has been shown to be a good adjunct treatment for acute anemia cases, as well as for the maintenance of tissue oxygenation. Hyperbaric oxygen therapy increases the amount of oxygen dissolved in plasma, thereby increasing the amount of oxygen available to the body tissues. The more oxygen that is available to the body, the higher the saturation levels are in the blood.

Oxygen levels in the blood (PaO2) have been shown to increase as high as 1500 - 1800 mm/Hg (normal is 97-105 mm/Hg) when oxygen is delivered under hyperbaric conditions. There also is evidence that hyperbaric oxygen therapy increases the flexibility of red blood cells and aids patients with blood anemias (i.e., sickle cell crisis).

OTHER EFFECTS AND DISEASES TREATED

There are other beneficial effects of hyperbaric oxygen therapy that are still being investigated.

Migraine Headaches: Research indicates that hyperbaric oxygen therapy acts as an alpha-adrenergic drug, which causes vasoconstriction. This may prove to be beneficial to patients with conditions such as migraine headaches. Patients receiving normobaric oxygen feel less relief from their migraine headache than those receiving hyperbaric oxygen therapy. This difference may be associated with the vasoconstrictive effects of the therapy or it may be due to increases in the rate of energy production and neurotransmitter-related metabolic reactions. More research is needed to verify this finding.

Other diseases that have yet to be proven as receiving benefit from hyperbaric oxygen therapy, and on which studies are currently being performed, are:

- Sports injuries (related to wound management).
- Aging
- Lupus
- Scleroderma
- Multiple sclerosis
- Rheumatoid arthritis
- Closed head injury
- Sickle cell crisis (related to anemia benefits)
- Hydrogen sulfide or carbon tetrachloride poisoning
- Frostbite (wound management)
- Cerebrovascular Accident (stroke)
- Fracture healing and bone grafting.
- Spider bite (Brown Recluse & *Loxasceles reclus*a)
- Spinal cord injury

Hyperbaric oxygen therapy is also being investigated as a treatment modality to shorten the duration of colds and flu and to reduce the effects of alcohol intoxication. Further studies are also being performed regarding its use in the treatment of:

- Myocardial infarction (reduces ischemia)
- Near drowning
- Electrocution (burns)
- Chronic fatigue syndrome
- Cardiac dysfunction
- Chronic obstructive pulmonary disease
- Alzheimer's Disease
- AIDS
- Cyanide poisoning

ADVERSE EFFECTS

The most common side effect of HBO therapy is a "crackling" sound in the ears between treatments. This can be relieved by clearing the ears in the same way as in the chamber during treatments. Much less frequently, a temporary change in vision is experienced. This alteration in vision returns to its normal state within six to eight weeks after treatment has finished.

Other adverse effects associated with the administration of hyperbaric oxygen therapy are primarily due to barotraumatic responses within the body. Middle ear pain and bleeding, development of mucus plugs, and sinus discomfort are among these adverse reactions.

Medical personnel managing the dive can be either outside the chamber (as in a monoplace chamber) or inside the chamber (as in a dualplace or multiplace chamber). Personnel in the chamber are subjected to the same dive conditions as the patient and run the risk of air embolism, decompression sickness, and nitrogen narcosis. All personnel must have a recent dive physical and be cleared for chamber dives.

Contraindications

Contraindications for administration of hyperbaric oxygen therapy include:

- Pneumothorax
- Severe chronic obstructive pulmonary disease with carbon dioxide retention
- History of thoracic surgery
- Chronic sinusitis
- Pregnancy

SAFETY CONSIDERATIONS

Because of its use in an oxygen-enriched environment, most safety measures revolve around issues related specifically to oxygen: fire and static electricity. For these reasons there must be no smoking in or around the hyperbaric chambers. There are a number of preparations necessary before each treatment which include:

- No smoking. Tobacco constricts blood vessels and limits the amount of blood and oxygen that can be delivered to tissues.

- Inform the hyperbaric team of all medicines the patient is taking. Some medications change the body's response to oxygen.

- Any signs of illness such as fever, cough, sore throat, nausea, or other symptoms should be communicated to the hyperbaric team. Treatments may need to be delayed until symptoms resolve.

- Patients should be informed not to drink carbonated beverages for 4 to 6 hours prior to treatment with hyperbaric oxygen. Because of the gas laws affecting gases within the body, these beverages may be hazardous.

During treatments, the patient should be advised:
- Wear the cotton scrubs provided or all cotton clothes. No synthetic clothing or pantyhose. Cotton reduces the potential for static electricity.

- No makeup or lip balm

- No hairspray, deodorant, lotions, oils, or creams due to static electricity effects and the use of petroleum products in these items

- No contacts or glasses

- Remove dentures and partial plates

- No perfume or shaving lotion

- No metal objects including jewelry or staples in books or magazines

In brief, Hyperbaric oxygen therapy has been proven to be a beneficial first line or adjunct therapy for a multitude of diseases. Because of its minimal side effects, it is a relatively safe therapy for the patient. Its use, however, may remain controversial for many years due to its potential for abuse.

OVERVIEW OF INTERVENTIONS

John Davidson, age seven, is admitted to a general pediatric unit from an outpatient clinic for treatment of a crush injury to his right leg, which hasn't responded to traditional wound care (including antibiotic therapy and wet-to-dry dressings) at home. Assessment shows a healthy child of average weight and development, with stable vital signs. The dressing on John's right leg extends from the knee to the ankle, and foul-smelling serosanguineous drainage is evident. Capillary refill time to the toes on the right foot is six to eight seconds, and the toes are cold to the touch, with pallor and diminished voluntary movement, as compared with the toes on the left foot. Doppler assessment reveals diminished blood flow to the right leg. The physician has notified the family that a course of hyperbaric oxygen (HBO) therapy, the best option to "save" John's leg, has been ordered for a one-hour period twice daily for two weeks.

Although respiratory technicians trained in HBO therapy (certified hyperbaric technicians, or CHTs) or certified hyperbaric registered nurses (CHRNs) will operate the equipment and monitor a patient during its use, staff nurses are responsible for the patient both before and after treatment. All caregivers need to understand HBO therapy and be aware of the contraindications, potential side effects, and drug interactions, as well as the appropriate treatment interventions.

The most well-known use of HBO therapy has been in the treatment of decompression illness; when a diver ascends too rapidly, intravascular nitrogen bubbles form faster than they can be exhaled. The illness can be asymptomatic or characterized by symptoms such as vertigo and joint and limb pain, and can lead to paralysis and death. During construction of the Brooklyn Bridge in the late 1890s the condition became known as "the bends" because afflicted caisson workers walked with a stooped gait (a caisson is a watertight chamber used for underwater construction). The effectiveness of HBO therapy in treating decompression illness has been well documented,[1] but this is actually one of its rarer applications today.

The most common use of HBO therapy among pediatric patients is for carbon monoxide poisoning, for which it's the treatment of choice to prevent long-term effects on the central nervous system.[2, 3] Among adults, the most common uses include the treatment of tissue hypoxia, necrosis, gas gangrene, osteomyelitis, blood loss anemia, osteoradionecrosis, and compromised skin grafts or flaps. In both populations, typical uses also include the treatment of anaerobic infection, crush injury, thermal burns,[4] gas embolism, and Bell's palsy.[5]

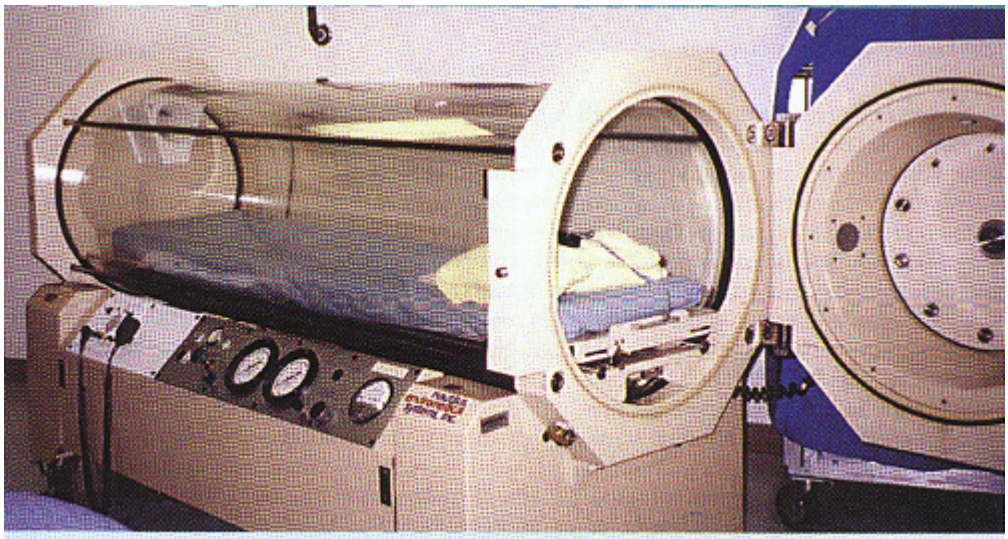
Although other uses of HBO therapy are still considered experimental, some independent outpatient clinics do offer treatment with hyperbaric oxygen to patients who can afford to pay privately. For example, increased tissue oxygenation is thought to benefit patients who have suffered a stroke or who have cerebral palsy. But the clinical evidence of the value of such uses is incomplete, and research is ongoing.

The Undersea and Hyperbaric Medical Society approves the use of hyperbaric therapy for the following conditions, which are accepted by third-party payers and Medicare[6]:

- air embolism
- thermal burns

- carbon monoxide and cyanide poisoning
- traumatic ischemias such as compartment syndrome or crush injuries
- anemia resulting from exceptional blood loss
- nonhealing wounds
- anaerobic cellulitis or gas gangrene
- necrotizing soft tissue fasciitis, including moist gangrene
- decompression illness ("the bends")
- acute peripheral arterial insufficiency
- osteomyelitis
- radiation tissue damage, including osteoradionecrosis
- actinomycosis
- intracranial abscesses
- compromised skin grafts or flaps

This course discusses primarily the use of single-patient (monoplace) chambers and focuses on the care giver's pre- and posttreatment responsibilities with both pediatric and adult patients: patient and family education; physical and psychological assessment and preparation; coordination of continuity of care; monitoring for complications; and provision of emotional support.



A monoplace HBO chamber.

HISTORY, DEFINITIONS AND PHYSICAL LAWS

History. The development of hyperbaric medicine is closely associated with the history of diving equipment. Aristotle recorded that Alexander the Great used a "glass barrel" as a diving chamber in 320 BCE. In the 1530s Guglielmo de Loreno developed the first diving bell. In 1691 Edmund

Halley improved the design by devising a way to replenish the air supply; he eventually built a diving chamber that enabled him to remain 60 feet underwater for one hour. In 1865 Benoit Rouquayrol and Auguste Denayrouse patented a compressed-air tank for underwater breathing. By adapting a car regulator in 1942 and 1943, Jacques-Yves Cousteau and Emile Gagnan invented a way to deliver compressed air automatically to a diver as he breathed; their "Aqua-Lung" was a modern forerunner of today's scuba (**s**elf-**c**ontained **u**nderwater **b**reathing **a**pparatus).

As early as 1662 experimental clinical applications of hyperbaric medicine were reportedly used with patients who had tuberculosis and other chronic diseases. In the 1830s hyperbaric chambers were used in France to improve circulation, although there was apparently no rationale for the treatment and no way to measure the partial pressure of oxygen in the blood. A French civil engineer, M. Triger, was the first to describe decompression illness symptoms in humans: in the 1840s he reported that caisson workers suffered from muscle cramps and sharp pains after prolonged exposure to increased atmospheric pressure, and noted that in such environments candles burned faster and the quality of the human voice changed. Larger chambers accommodating more than one person were subsequently built for use by patients who needed treatment from a caregiver during a session or were unable to lie down as required in a monoplace chamber. By 1879 a mobile hyperbaric operating room, originally developed for spas, was in use.

HBO therapy was introduced in the United States in 1861, primarily as treatment for "nervous" disorders and influenza and other respiratory conditions. But lack of research evidence of its effectiveness limited its use until the 1930s, when Behnke and Shaw began successfully employing it to treat decompression illness. Currently, magnetic resonance imaging and color spectrum–based computed tomography scans can be used to evaluate the effectiveness of HBO therapy, and research continues to expand and direct its use. However, the initial installment expenses are significant: HBO chambers range in cost from about $85,000 for a single-person unit to more than $500,000 for one that can accommodate two or more people, and the necessary facility modifications can run more than $200,000. But a patient's treatment costs, though they vary depending on the provider and the third-party payer, are generally not prohibitive, ranging from about $100 to $220 per treatment hour.

Definitions. *Hyperbaric oxygen therapy*, an adjunctive treatment, involves the administration of 100% oxygen at increased atmospheric pressure. Pressure can be expressed using various units, including millimeters of mercury (mmHg) and atmospheres absolute (ATA). Atmospheres absolute is the sum of atmospheric and hydrostatic pressures—in other words, the total pressure of the weight of air (and for divers, water) exerted on the body. The average atmospheric pressure exerted at sea level is 1 ATA; HBO therapy typically administers oxygen at 1.5 to 3 ATA.

Administration occurs within a *hyperbaric chamber*, an airtight enclosure capable of withstanding high internal pressures. Three types currently exist. A *monoplace* chamber accommodates one patient lying on a padded, built-in platform that glides in and out on rollers. In 1977 there were only 30 such chambers in the United States; by 1997 there were more than 300.[7] *Multiplace* (multiperson) chambers may be as large as a room, and most accommodate

between two and seven people. Patients may be either seated or supine. If necessary, a care giver can accompany the patient into the chamber; both are subject to increased atmospheric pressure, but the care giver breathes air, while the patient receives 100% oxygen through a face mask. Small, *topical* chambers are designed to fit only over the body part to be treated.

How it works. The inhalation of pure oxygen at higher than normal pressure causes the patient's plasma and hemoglobin to become supersaturated, enhancing oxygen delivery to all tissues. The higher pressure also drives oxygen directly through the skin, raising the oxygen level in all tissues by that route as well.

HBO schedules vary. Most conditions require intermittent treatment: a typical session lasts between 60 and 90 minutes, with one or two sessions prescribed daily for two to four weeks. Carbon monoxide poisoning and decompression illness are always treated with a single, longer session.

Each session consists of three phases: compression, constant pressure, and decompression. To understand what a patient is likely to experience during each phase, it will help to know the laws of physics that apply.

- *Dalton's law* states that the total pressure of a gas mixture is equal to the sum of the partial pressures of each gas in the mixture; for example, the total pressure of air equals the sum of the partial pressures of nitrogen, oxygen, and air's other component gases. Although HBO therapy typically uses 100% pure oxygen, room air may be used, or helium added (to treat nitrogen narcosis), without adversely affecting atmospheric pressure.

Compression. During this phase, atmospheric pressure inside the chamber is raised to a prescribed level above normal. This generally takes between six and 10 minutes to achieve, and sometimes as long as 30 minutes.

- *Boyle's law* states that if the temperature and mass of a gas remain constant, its volume will be inversely proportional to its pressure (as pressure increases, volume decreases). Therefore, the volume of any gas-containing body cavity will change as the pressure of the external environment increases or decreases. For example, as atmospheric pressure rises during compression, pressure will temporarily be greater outside the body than inside the eustachian tubes, and may push the tympanic membrane inward. The patient may also report feeling "weight" around him, similar to the sensation of being underwater.

- *Gay-Lussac's law* states that if the volume of a gas remains constant, its pressure will be directly proportional to its temperature. Thus, from an optimal starting temperature inside an HBO chamber of 70°F, temperature may increase to as high as 80°F during compression (the patient may report feeling warm) and decrease to as low as 65°F during decompression (the patient may report feeling cool).

Constant pressure. Atmospheric pressure is held at a prescribed level above normal for a prescribed length of time, usually between 60 and 90 (and not more than 120) minutes. Prescribed treatment pressure and duration of chamber time will be determined by the type of pathogen present at a wound site and other factors such as the degree of revascularization around the affected tissues.

Decompression. Atmospheric pressure inside the chamber is decreased to normal. Decompression generally takes between six and 10 minutes but can last as long as 30 minutes.

▲ A topical HBO chamber.
(Photo courtesy of Advanced Hyperbaric Technologies, Inc.)

Benefits of HBO

For wound healing and other approved indications, evidence has shown benefits of HBO therapy, including

increased tissue oxygen perfusion. Elevated blood oxygen levels can last for as long as four hours posttreatment, benefiting hypoxic areas such as chronically infected, wounded, or irradiated tissues.

- *Henry's law:* The degree to which a gas enters into solution in a liquid (such as plasma) is directly proportional to the partial pressure of the gas to which the liquid is exposed. Thus, the solubility of oxygen in plasma is enhanced by HBO.

enhanced wound healing. HBO therapy increases the partial pressure of arterial oxygen (PaO2), resulting in vasoconstriction, which can aid the treatment of trauma wounds by reducing edema, reducing capillary pressure, and allowing a better flow of hyper-oxygenated plasma to the tissues. Hyperoxia also enhances collagen formation, which is vital to healing.

increased neo- and revascularization. HBO therapy is intermittent: periods of hyperoxia (during treatments) alternate with periods of either normal oxygenation or hypoxia (between treatments). This pattern results in increased capillary formation and improved circulation to tissues.

inhibition of anaerobic toxins. Increased tissue oxygenation causes bacteriostasis of anaerobic bacteria, including gas gangrene toxins.

Other potential benefits of hyperbaric therapy are still being investigated.

Side Effects

Ears, nose, and throat. Gas-filled cavities such as the paranasal sinuses expand during HBO therapy, and otic or sinus barotrauma may occur. Swallowing, which temporarily increases pressure in the middle ear, can open the eustachian tubes and help equalize pressure. But an upper respiratory infection can obstruct the eustachian tubes. And because a child's eustachian tubes are shorter and straighter than an adult's, children are predisposed to otitis media, which, if present, raises the risk of otic barotrauma during HBO therapy.

Transmission of sound is altered at higher than normal atmospheric pressure, resulting in temporary changes to vocal quality and pitch. As atmospheric pressure increases, so does atmospheric density, which affects how the vocal cords vibrate and may cause the voice to sound either higher or lower than usual.

Eyes. Some patients report either better or worse vision during HBO therapy, resulting from changes to the shape of the lens, which tends to flatten under increased atmospheric pressure. This can result in temporary myopia, and it can worsen existing myopia in adolescents and adults. Changes in visual acuity are less noticeable in pediatric patients because eye tissues don't mature until after age seven. Although such changes are temporary, recovery of previous visual acuity may be slow.

Lungs. In a patient with an obstructed airway (for example, as a result of asthma or a tumor), the trapped air can result in pulmonary barotrauma when the gas expands, causing alveolar overdistention and rupture. Pneumothorax or gas embolism can also occur.

As atmospheric pressure increases, gas density increases also, making breathing more difficult. Patients who experience dyspnea or are intubated may have increased respiratory problems in a hyperbaric chamber.

Oxygen can be toxic to the lungs. After 72 hours of continuously breathing 100% oxygen at increased atmospheric pressures, the alveolar lining can be damaged.[8] However, the intermittent nature of HBO therapy means there is minimal risk to pulmonary function.[9] Signs and symptoms of pulmonary oxygen toxicity include chest discomfort, burning on inspiration, cough, and difficulty breathing.

Central nervous system. Neurologic oxygen toxicity caused by HBO therapy occurs most often when hyperbaric pressure reaches 4 ATA but can occur at any time during treatment. Signs and symptoms include tremors and seizures.

Cardiovascular system. Cardiac output is dependent on oxygen levels in the tissue. HBO therapy increases tissue oxygenation, resulting in reduced cardiac output and bradycardia. Signs and symptoms include slowed pulse when compared with baseline.

When divers ascend too rapidly, gas embolisms can occur in tissues and blood vessels. (Although gas embolism is also a slight risk during HBO therapy, the decompression rate is controlled to prevent this complication.)

Relative and Absolute Contraindications

Relative contraindications. The following conditions may place patients at higher risk during HBO therapy.

Upper respiratory infection can preclude clearing of the eustachian tubes and equalizing of internal (sinus) and external pressure. If HBO therapy is essential, patients with such infections should receive decongestants beforehand. Alternatively, the primary care physician may insert tympanostomy pressure-equalizer (PE) tubes or perform a myringotomy before HBO therapy is initiated.

Otitis media is a relative contraindication, unless PE tubes have been inserted.

Hypertension. HBO therapy causes vasoconstriction and can result in hypertensive crisis. The risk diminishes if the patient's hypertension is well controlled by medication.

Graves's disease or any thyroid disorder being treated with thyroid hormone increases the metabolic rate. In conjunction with HBO therapy, this can cause oxygen toxicity. A dosage adjustment may be necessary, and the patient must be closely monitored.

Seizure disorders (such as epilepsy). HBO therapy can disrupt the process (known as the hemoglobin–oxygen buffering mechanism) by which oxidizing free radicals are removed from tissue. This can result in the oxidation of polyunsaturated fatty acids that are normally present in cells; because central nervous system tissue has a high lipid content, it is highly vulnerable.[8] Thus, central nervous system oxygen toxicity lowers the seizure threshold in patients susceptible to seizures. Seizure disorders may necessitate a dosage adjustment or the addition of a benzodiazepine before HBO therapy is administered.

High fever can lead to convulsions in the HBO chamber. Young, febrile children who have seizure disorders may have a toxic reaction to HBO, caused by an increase in the partial pressure of carbon dioxide (PaCO2) level, and the resultant cerebral edema can provoke seizures. A lower atmospheric pressure of oxygen may be required to help normalize the PaCO2 level in a febrile patient. Hypothermia blankets and other cooling measures may be prescribed.

Diabetes mellitus types 1 and 2. HBO therapy has been shown to lower blood glucose levels.[10, 11] Special precautions must be taken, including monitoring blood glucose levels before and immediately after treatment.

Menstruation can lead to a greater risk for decompression illness in the early phase of the menstrual cycle[12]; even a slight increase in discharge at any time during the course of treatment must be brought to the HBO therapy physician's attention for evaluation.

Eye pathology, including any optic nerve or retinal disorder, necessitates evaluation by an ophthalmologist before administration of HBO therapy.

Pregnancy. There should be no adverse effects from HBO therapy if treatment isn't prolonged (no session lasting longer than 120 minutes). But even with short, intermittent treatment sessions, there is a small risk to the fetus that high oxygen levels will stimulate the muscles around the ductus arteriosus to contract, closing it and causing fetal death.

Infancy. In premature infants, high oxygen levels can be toxic to the eye vasculature and can worsen bronchopulmonary dysplasia. In neonates, retrolental fibroplasia is a prominent risk factor when the infant is placed in a high-oxygen atmosphere. The potential for oxygen toxicity (manifested by bronchopulmonary dysplasia in an infant with pulmonary surfactant deficits or in an infant receiving assisted ventilation) must be considered.

Absolute contraindications. The following conditions absolutely contraindicate the administration of HBO therapy.

Some types of congenital heart disease. Any cardiac anomaly that results in restriction of right ventricular outflow to the lungs might require the ductus arteriosus to remain patent for the patient to survive. HBO therapy can be lethal to such a patient because oxygen promotes the closure of the ductus arteriosus.

Obstructed airway or restrictive airway disease (such as asthma) can cause local trapping of air during HBO decompression. The expanding gas can lead to alveolar rupture. In the case of emphysema with carbon dioxide retention, the retention of CO2 also causes respiratory acidosis and cerebral edema, which can lead to respiratory depression, hypoxia, and death.

Recent thoracic surgery. Air trapped in the thorax can expand during HBO therapy and result in pneumothorax.

Untreated pneumothorax can become a tension pneumothorax during HBO therapy.

Unstable seizure disorders (such as unstable epilepsy). If a patient has a seizure inside a monoplace chamber, the provider can't immediately give care. (Unstable seizure disorders are not absolute contraindications to HBO therapy if the patient can be treated in a multiplace chamber.)

Pregnancy is an absolute contraindication for HBO therapy if treatment lasts more than 120 minutes in a given session. Therapy longer than 12 continuous hours greatly increases the risk of the fetal ductus arteriosus closing, resulting in fetal death. (Although 12 continuous hours of treatment is an unusual prescription, it has been used to treat decompression illness.)

Infancy. When an infant with a congenital heart defect is dependent on a patent ductus arteriosus for survival, HBO therapy is absolutely contraindicated.

PREPARATION AND PRETREATMENT

Education, including an explanation of risk, and the obtaining of informed consent constitute the first steps in preparing patients for HBO therapy. Explain to patients and their families what to expect during and after a treatment session, and give them the opportunity to talk about their concerns before therapy begins.

Assessment includes physical, psychological, and psychosocial evaluation. Record current vital signs. Note in particular the presence of upset stomach, headache, sinus conditions, cold or flu, and ear pain. Include blood glucose testing if the patient has diabetes. Note the most recent voiding times for bladder and bowel. Include a dental treatment history; temporary dental fillings can trap air, which will expand during decompression, causing severe pain.

With a patient new to HBO therapy, note risks with regard to diagnosis; and with all patients, note changes in overall status as they occur. Maintain communication with the multidisciplinary team. (For example, you may find that a woman undergoing daily HBO therapy has begun to menstruate, placing her at higher risk for decompression illness. In such a case, notify the HBO therapy physician and other members of the team. Delay of HBO therapy for a few days may be indicated.)

In conjunction with HBO therapy, the following all increase the risk of oxygen toxicity: steroid use, fever, history of seizures, vitamin E deficiency, use of vasodilators or antioxidants, acetazolamide treatment, and low pH level (acidosis). Close patient monitoring will be required; delay of HBO therapy may be indicated.

There is an inherently higher risk of a flash fire in an oxygen-rich environment under increased atmospheric pressure. All standard precautions related to oxygen therapy hazards should be posted and observed in and around the HBO unit. All materials that support combustion, items that can hold static electricity, and objects that may discharge sparks must not be permitted in or near the HBO chamber. No synthetic rubber, plastic (including Styrofoam), or metal items can be taken into the chamber.

Ensure that all removable items have been removed, including jewelry, dental spacers, ear plugs, hair clips, and canvas splints. HBO therapy is rarely given so soon postoperatively that surgical clips or staples would still be in place, but if the patient has these or orthopedic hardware, cardiac valves, or temporary dental fillings, notify the HBO therapy physician of their presence. Contact

lenses should not be worn in the HBO chamber: the increased pressure slightly changes the contour of the eye and possibly that of the contact lens also, which can result in injury. Battery-powered toys, radios, hearing aids, or other devices, as well as any metal or "friction" toys (anything that can hold static electricity) cannot be taken into the chamber. Cosmetics, hair sprays or gels, alcohol-based creams, oils, and mafenide (Sulfamylon) cream or powder must also be removed because all contain combustible ingredients. All food must be left outside the chamber.

Make sure that the patient is wearing clean, 100% cotton clothing and coverings. (Check labels; even patient gowns are sometimes made of blended materials, such as cotton and polyester.) A woman who is menstruating should wear only all-cotton sanitary pads or tampons. Infants should wear cotton diapers.

Secure dressings using only paper tape. Check extension tubing on all IV lines and make sure any attached metal items (such as clamps) have been removed. Make sure that the ends of any nasogastric tubes are secured with the correct plugs or caps. Petrolatum gauze covered by an occlusive dressing on a chest tube insertion site may be allowed in the chamber.

HBO therapy results in short-term decreased immune system response that enhances allografts, but patients must be monitored for infections.[13] Therefore, immunizations should not be given just before or during the course of therapy.

Otic barotrauma, such as effusion and otitis media, is a common complication of HBO therapy, especially in infants and young children. To help equalize internal (ear and sinus) and external pressures, infants who are not allergic to latex may be given a latex pacifier to suck on. (Most pacifiers are made of synthetic rubber, a petrochemical product, which is not safe for use inside an HBO chamber.) Young children can be taught how to yawn, swallow, or mimic chewing. Adolescents and adults may also be taught to perform gentle Valsalva maneuvers. Pretreatment with vasoconstricting nose drops or spray may be prescribed to prevent occlusion of the eustachian tubes. For patients who require repeated HBO therapy, insertion of tympanostomy PE tubes may be considered.

Before beginning HBO therapy with a patient who has an endotracheal tube, check for upper respiratory infection and ask about history of pulmonary problems such as restrictive airway disease. Endotracheal tube cuffs inflated at normal atmospheric pressure will decrease in volume as atmospheric pressure increases. Pretreatment precautions include inflating the cuff with saline instead of air, to preserve cuff integrity under pressure; this will help prevent dislodgment of the tube.

Patients are usually medicated before HBO therapy. Drugs commonly used include diazepam to reduce anxiety and the risk of oxygen convulsions; oxymetazoline nasal drops to clear nasal passages and reduce eustachian tube blockage; and vitamin E (given orally) to protect against pulmonary oxygen toxicity and reduce the risk of related convulsions.

During compression, the rise in temperature may cause the patient to feel too warm. (The temperature rise can often be controlled by venting, a process managed by the CHT or CHRN.)

Patients may use a cool, wet, cotton washcloth in the chamber to sponge themselves as needed. During constant pressure and decompression, patients will experience evaporative, convective, and conductive heat loss. Infants and young children will require special efforts to maintain normal body temperature, because they have a larger body surface area proportional to weight than do adults. Cotton blankets may be pre-warmed and kept nearby for use as needed.

Pediatric patients. Obtaining parents' informed consent should be part of the education process; telling parents and children what to expect both during and after treatment helps all involved. The better parents understand HBO therapy, the better they'll be able to help reduce their child's anxiety and increase adherence to required activities or restrictions. Note the presence of otitis media, the effects of confinement and isolation during treatment, the quality of interactions between parent and child, and the stability of vital signs.

Distraction during treatment can be invaluable. Monoplace chambers usually have acrylic windows and are equipped with a microphone and speakers to permit two-way conversation. Let parents know that they may read to their child; patients can also watch television or videotapes through the window. Few toys can be taken into a hyperbaric chamber—cotton stuffed toys and all-cotton blankets are allowed, but toys that could hold static electricity or produce sparks are not. Conscious sedation may be indicated in anxious children (in which case an advanced practice care giver or HBO therapy physician monitors the patient, in cooperation with the CHT or CHRN). More restrictive restraints usually result in increased anxiety and "fighting," which could lead to vomiting or aspiration and are not recommended for use in a monoplace chamber to which immediate access is limited.

If an infant or young child is on mechanical ventilation, a myringotomy may be necessary to prevent otic barotrauma, as any nasopharyngeal edema can obstruct the eustachian tubes.

Older adolescents and adults. As with younger patients, distraction is often effective in allaying fear. Family members can talk with the patient, or the patient can watch television or videotapes. Adolescents or adults may be given a mild sedative such as diphenhydramine before therapy begins, if needed (young children are prone to paradoxical hyperactivity reactions to this medication).

Because caffeine interferes with the absorption of oxygen by the tissues, all caffeinated beverages should be withheld 12 to 24 hours before treatment.[14] (The amount of caffeine in chocolate and most other sources is not high enough to be clinically significant.)

Smoking causes vasoconstriction and can impair tissue healing. Further, burning tobacco produces carbon monoxide, which competes with oxygen in binding to hemoglobin, thus decreasing its oxygen-carrying capacity. Encourage patients not to smoke any form of tobacco for the duration of HBO therapy. Pharmacologic assistance or social support may be necessary. Assessment for this risk factor is essential in all patients requiring HBO therapy, especially adolescents, who may already have begun smoking, usually without their parents' knowledge.

Potential Drug Interactions with Hyperbaric Oxygen Therapy	
Doxorubicin (Adriamycin)	At least one week should separate HBO therapy and doxorubicin therapy to avoid increased mortality resulting from cardiac toxicity.
Mafenide (Sulfamylon)	Frequently used for burn therapy, mafenide is contraindicated in an HBO environment. Both the powder and cream forms are hydrocarbon based and are therefore combustible.
Steroids	Steroids can cause oxygen toxicity and convulsions. If steroid treatment is essential, anticonvulsants should be prescribed and the patient should be observed closely for signs of convulsions.
Morphine or meperidine	Narcotics and high oxygen concentrations each cause respiratory depression. This results in increased blood flow to the brain to maintain tissue oxygen perfusion; but it can also precipitate oxygen toxicity, manifested by convulsions.
Anesthetics	Local anesthesia is safe in HBO chambers. General anesthesia poses safety risks (in terms of both dosages and flammability). Ketamine (Ketalar) is safe to use in the chamber but requires an anesthesiologist in attendance to deal with emergency respiratory complications.
Anticonvulsants	Anticonvulsants are often used prophylactically in HBO chambers. However, oxygen-induced damage to the central nervous system is still a risk if HBO treatment is prolonged.
Barbiturates	These drugs may be used to suppress convulsions, but they do not relieve oxygen toxicity to the tissues. Observe for respiratory depression. Diazepam (Valium) or lorazepam (Ativan) are the drugs of choice to reduce anxiety and suppress convulsions in the HBO chamber.
Insulin	The blood glucose levels of patients with diabetes may fall rapidly during HBO therapy. The care giver should check blood glucose levels pre- and post- HBO. Sliding-scale insulin dosages and orange juice should be available.
Acetazolamide (Diamox)	This drug prevents oxygen-induced vasoconstriction, thereby increasing cerebral blood flow, which in turn predisposes patients to convulsions resulting from oxygen toxicity. The drug is contraindicated with HBO therapy above 2 ATAs of pressure. To prevent convulsions, diazepam may be given with acetazolamide.
Thyroid hormone (natural or synthetic)	Administration of thyroid hormone or the presence of Grave's disease during HBO therapy increases the metabolic rate, causing oxygen toxicity. HBO therapy is not recommended for those patients receiving thyroid hormones.

Chlorpromazine (Thorazine)	This drug protects against oxygen toxicity convulsions by acting on the sympathetic nervous system and having an antiepinephrine effect. Observe for tremors or convulsions if high doses are used or carbon monoxide poisoning is involved.

Sources: McDougald JA. *The essentials of hyperbaric medicine.* Riverside (CA): Riverside General Hospital; 1991; McArthur CLI, Lockridge H. *The essentials of hyperbaric medicine. 2nd ed.* Riverside (CA): Riverside General Hospital; 2000; Kindwall EP. Contraindications and side effects of HBO treatment. In: *Hyperbaric medicine practice. 2nd ed.* Flagstaff (AZ): Best Publishing; 1995. p. 47-55; Miller BF, Keane CB. *Miller-Keane encyclopedia and dictionary of medicine, nursing and allied health. 6th ed.* Philadelphia: Saunders; 1997; Wilson BA, et al., editors. *Nurses' drug guide.* Norwalk (CT): Appleton & Lange; 2000; *Skidmore-Roth L. Mosby's 2000 nursing drug reference.* St. Louis: Mosby-Yearbook; 2000; Lininger SW, editor. *A–Z guide to drug–herb– vitamin interactions.* Rocklin (CA): Prima Health; 1999.

During the Treatment Sessions

The main adverse reactions that can occur during HBO therapy include barotrauma, gas embolism, oxygen toxicity, claustrophobia, and anxiety. Monitor and document vital signs frequently and check that the equipment necessary to handle these potential complications is readily available.

Monitor for substernal chest discomfort or pain, dyspnea, cough, pain on inspiration, and tremors or seizures, which may indicate pulmonary oxygen toxicity.

Check for signs of anxiety. Any significant vital sign changes or restlessness should be reported to the HBO therapy physician. Reassure the patient and family members, and provide diversion for the patient during treatment. Administer antianxiety medications as ordered. Note the patient's response to these interventions. Although HBO therapy cannot be terminated without adequate decompression time, the CHRN may begin decompression early if she thinks the patient isn't responding well.

Monitor the patient's level of consciousness and blood pressure pre- and posttreatment, and observe for signs of nitrogen narcosis—euphoria, followed by impaired judgment, disorientation, and decreased coordination—especially if compressed air is used instead of 100% oxygen (as it may be when treating decompression illness). Although the risk for nitrogen narcosis decreases when 100% oxygen is used, nitrogen is present in the air we breathe and in our bodies. As with decompression illness, if decompression occurs too fast, more and larger bubbles may form and can mechanically deform tissue and obstruct blood vessels, causing nitrogen narcosis. But note that high nitrogen levels produce an anesthetic effect on the brain. A patient with decompression illness or nitrogen narcosis may be asymptomatic, with no complaints of muscle or joint aches and exhibiting no muscle tremors, or seizures.

Observe closely for muscle twitching or seizures during decompression, which may indicate oxygen toxicity. If either occurs, decompression should be stopped until vital signs are stabilized.

Routine decompression and removal of the patient from the chamber can usually be accomplished within 15 minutes; these have been performed in fewer than 90 seconds in emergency situations. But rapid decompression and removal occurring in less than six minutes carry increasing risks, including that of closure of the glottis, which is life threatening. Any decision to do so must involve weighing all the risks.

Drugs are not usually administered inside a monoplace chamber because caregivers don't have immediate access to patients; thus, there's no danger of implosion of glass vials. Drugs are sometimes administered (by the CHRN, not the staff care giver) inside a multiplace chamber. The larger the glass vial, the greater the implosion risk; only 10-cc or smaller vials should be used. If drug administration is required during a treatment session, the oral or IV route (using a high-pressure iv pump) is preferred. Intravenous solutions in glass bottles should not be used unless appropriate precautions are taken to prevent a large bolus of the IV solution from being forced into the patient's circulatory system during decompression. Since vasodilation occurs immediately after therapy, an uncontrolled release of a drug that was peripherally administered during the HBO therapy may occur, which can result in a toxic response. Drugs should not be stored in an HBO chamber, as this can alter their potency.

POST-HBO

Barotrauma, causing bleeding and edema of the eustachian tubes and other structures of the middle ear and possible rupture of the tympanic membrane, may not become evident until after the treatment session ends. Patients may complain of earache. Tympanic rupture usually is evidenced by ear drainage within an hour posttreatment; other problems can manifest more slowly. Hearing loss, tinnitus, a sensation of excessive sinus pressure, tooth pain, or nystagmus may indicate a serious complication and should be reported to the HBO physician and recorded in the patient's chart.

Tinnitus, paresthesia or tingling, blurred vision, and palpitations are all signs of oxygen toxicity affecting the central nervous system. These conditions must be reported to the HBO therapy physician and closely monitored. It's essential that they be recorded in the patient's chart so that progress can be assessed.

If substernal chest pain and cough develop between treatment sessions, with no evidence of other causes, the HBO therapy and primary care physicians should be notified before the next treatment session. These can be early indicators of pulmonary oxygen toxicity.

Headache or pain over the sphenoid or frontal sinus can indicate complications involving the sinuses.

Patients who experience worsened vision should be cautioned not to seek a new prescription for corrective lenses, as most patients' eyes return to pretreatment acuity within a few months after HBO therapy has ended.

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Primer on Basic Concepts of ECG

Introduction to ECG Interpretation

Electrocardiogram interpretation is an invaluable clinical skill that is taught in many different ways at across the country. It is often informal and clinicians are expected to "pick it up" as they see patients on the wards and in clinics. There are many "courses" which can be purchased off the shelves at the bookstore -some of them too simplistic and others hopelessly detailed. In an effort to better meet the needs of ECG interpretation this course serves as a self study manual.

Differential diagnosis is emphasized to encourage thinking about the "art" of interpretation, not just a cookbook mechanistic approach. It is our sincere hope that you find this course worthwhile and a skill you will continue to use throughout your careers in medicine.

Learning Objectives

Upon successful completion of this course, you should be able to:

1. Identify and define key terms associated with ECG
2. Differentiate between “rate” and “rhythm”
3. List and discuss the criteria for “hypertrophy”
4. Explain how to identify an “infarct” on and ECG tracing

ELECTROCARDIOGRAMS

The following is a brief introduction to electrocardiograms and their interpretation:

1. P wave = depolarization of the atria.
   QRS = depolarization of the ventricle.
   T wave = repolarization of the ventricle.

Figure 1: Description of the waves on the ECG.

2. Cardiac muscle cells depolarize with a positive wave of depolarization, then repolarize to a negative charge intracellularly.
3. Skin "leads" or electrodes have a positive and negative end.
4. A positive wave form (QRS mainly above the baseline) results from the wave of depolarization moving towards the positive end of the lead. A negative waveform (QRS mainly below the baseline) is when a wave of depolarization is moving *away* from the positive electrode (towards the negative end of the lead).
5. ECG paper has 1 millimeter small squares - so height and depth of wave is measured in millimeters.

   10 mm = 1.0 m Volt
6. Horizontal axis is time.
   .04 seconds for 1 mm (1 small box).
   .2 seconds for 1 large box = 5 small boxes = 5 x .04 seconds.

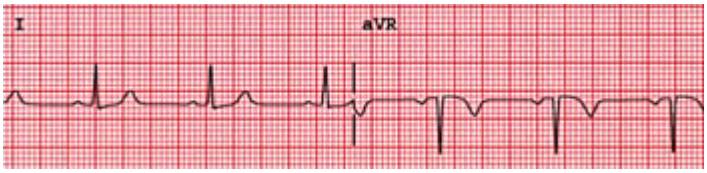


Figure 2:
Positive QRS in Lead I.
Negative QRS in Lead aVR.

R wave = 7-8 mm high in Lead I.
QRS wave = .06 seconds long in Lead I.

7. Lead nomenclature.

Limb Leads	*Chest Leads*	*Rhythm Strip*
I, II, III aVR, aVF, aVL	V1 - V6	Located on the bottom of the ECG printout. Selected to give the best relationship of the P wave to the QRS.

8.

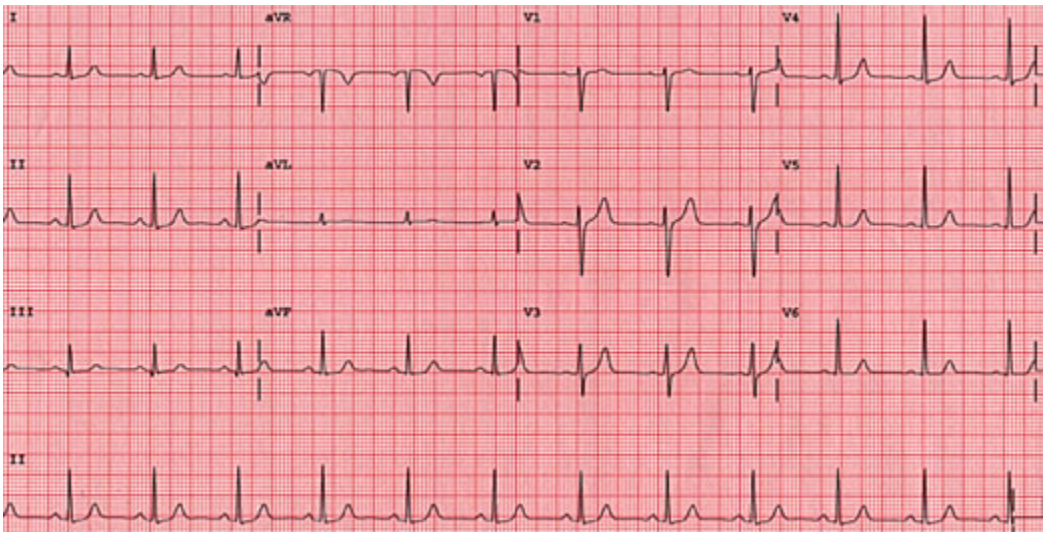
9. 

Figure 3: A normal ECG and rhythm strip.

10. ECG interpretation: look at five areas, in order, on each ECG.

Rate
Rhythm (Intervals)
Axis
Hypertrophy
Infarct

RATE

Rate is cycles or beats per minute.

Normal rate for the SA node 60-100.

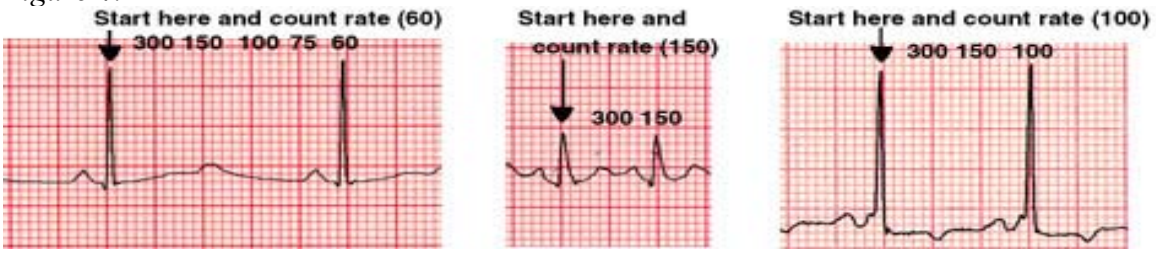
<60 bradycardia	>100 tachycardia

SA node is the usual pacemaker, other potential pacemakers (if SA node fails) are atrial pacemakers with inherent rates of 60-80, AV node (rate 40-60), or ventricular pacer (rate 20-40). In certain pathologic conditions ectopic (out of place) pacemakers can go much faster at rates 150-250 cycles/minute. There are three methods of calculating rate:

1. *Most Common Method:*
   (Most rates can be calculated this way). Find an R wave on a heavy line (large box) count off "300, 150, 100, 75, 60, 50" for each large box you land on until you reach the next R wave. Estimate the rate if the second R wave doesn't fall on a heavy black line.

Rate calculation
Memorize the number sequence:
300, 150, 100, 75, 60, 50

2. *Figure 4:*



3. Common Method.

4. *Mathematical method:*
   Use this method if there is a regular bradycardia, i.e. - rate < 50. If the distance between the two R waves is too long to use the common method, use the approach: 300/[# large boxes between two R waves].

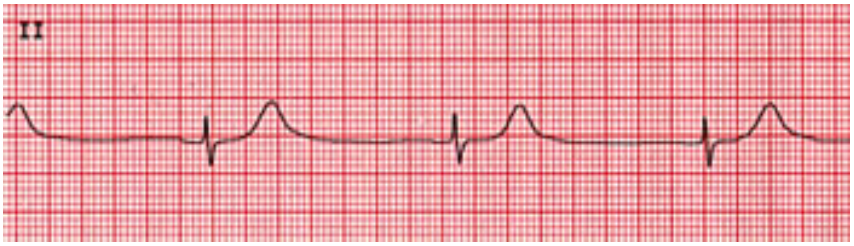


Figure 5: Count number of large boxes between first and second R waves=7.5. 300/7.5 large boxes = rate 40.

5. Six-second method:
   Count off 30 large boxes = 6 seconds (remember 1 large box = 0.2 seconds, so 30 large boxes = 6 seconds). Then, count the number of R-R intervals in six seconds and multiply by 10. This is the number of beats per minute. This is most useful if you have an irregular rhythm (like atrial fibrillation) when you want to know an average rate.

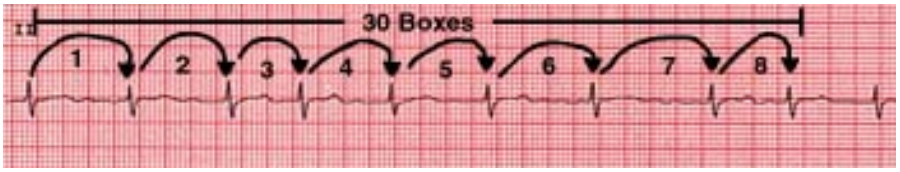


Figure 6: Count 30 large boxes, starting from the first R wave. There are 8 R-R intervals within 30 boxes. Multiply 8 x 10 = Rate 80.

RHYTHM (to include intervals)

The basic "core" of rhythms and measured "intervals" (PR, QRS, QT). Rhythms are often the most challenging aspect of ECG's.

Now for some basics - "arrhythmia" means abnormal rhythm.

The normal conduction pathway is: SA node --> AV node --> Bundle of HIS --> Bundle Branches.

Arrhythmia can be understood by realizing the existence of ectopic (out of place) foci (pacemakers) and understanding the normal conduction pathway of the heart. Very simply put, if the beat originates in the *atria or AV node* (supraventricular) the *QRS is usually narrow* (normal), because it comes from above along the normal pathway.

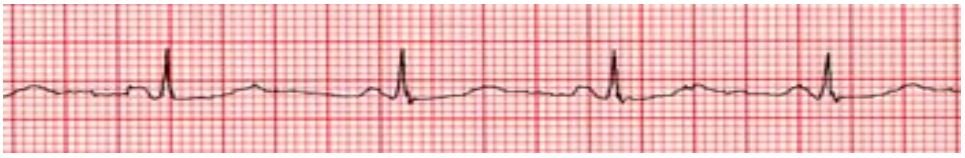


Figure 6a: QRS is narrow (normal).

If the beat is *ventricular in origin*, the *QRS is wide and bizarre* because it doesn't come down the normal pathway.

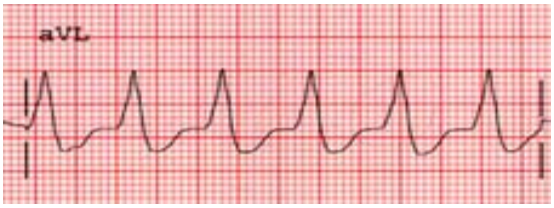


Figure 6b: QRS is wide.

Aberrancy is an exception to this rule - here it does actually follow the normal pathway (atria - AV node - ventricle) but for some reason the pathway is refractory to the beat and you get a wide QRS.

A reasonable way to group arrhythmias is in four general groups. Let us briefly review these four groups, then we will develop some common sense principles for evaluating rhythm (to include intervals).

AXIS

Direction of depolarization (vector) of the QRS complex.

1. The left ventricle is thicker so the mean QRS vector is down and to the left. (The origin of the vector is the AV node with the left ventricle being down and to the left of this).
2. The vector will point toward hypertrophy (thickened wall) and away from the infarct (electrically dead area).

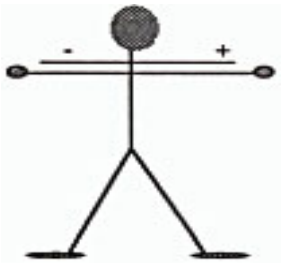
Figure 28: Axis nomenclature.

Normal axis	-30 to +90 degrees
Left axis deviation	-30 to -90 degrees

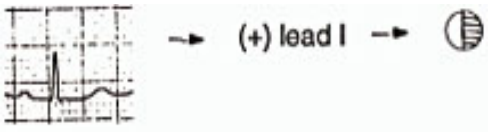
Right axis deviation +90 to +/-180 degrees
Indeterminate (extreme) axis deviation -90 to +/-180 degrees

Since lead I and aVF are perpendicular to each other, you can use those two leads to quickly determine axis.

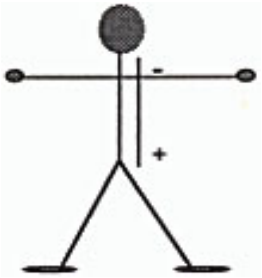
Lead I runs from right to left across a patient's body, positive at the left hand: (See figure 28).



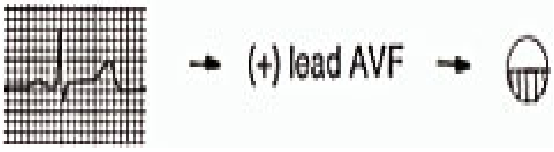
If the QRS in lead I is positive (mainly above the baseline), the direction of depolarization will be in the positive half (right half) of the circle above. You can make a diagram and shade in the positive half of the circle.



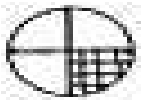
Lead aVF runs from top to bottom across a patient's body, positive at the feet:



If the QRS in lead aVF is positive (mainly above the baseline), the direction of depolarization will be in the positive half (lower half) of the circle above. You can make a diagram and shade in the positive half of the circle:



To find the axis overlap the two circles. The common shaded area is the quadrant in which the axis lies. In this example, the axis lies in the normal quadrant, which on a patient, points down and to the left.



You can repeat this process for any two leads, but I and aVF are the classic places to look. If you realize that there are two leads to consider and a positive (+) or (-) orientation for each lead, there would be four possible combinations. Memorize the following axis guidelines.

	Lead I	Lead aVF
1. Normal axis (0 to +90 degrees)	Positive	Positive
2. Left axis deviation (-30 to -90) Also check lead II. To be true left axis deviation, it should also be down in lead II. If the QRS is upright in II, the axis is still normal (0 to -30).	Positive	Negative
3. Right axis deviation (+90 to +180)	Negative	Positive
4. Indeterminate axis (-90 to -180)	Negative	Negative

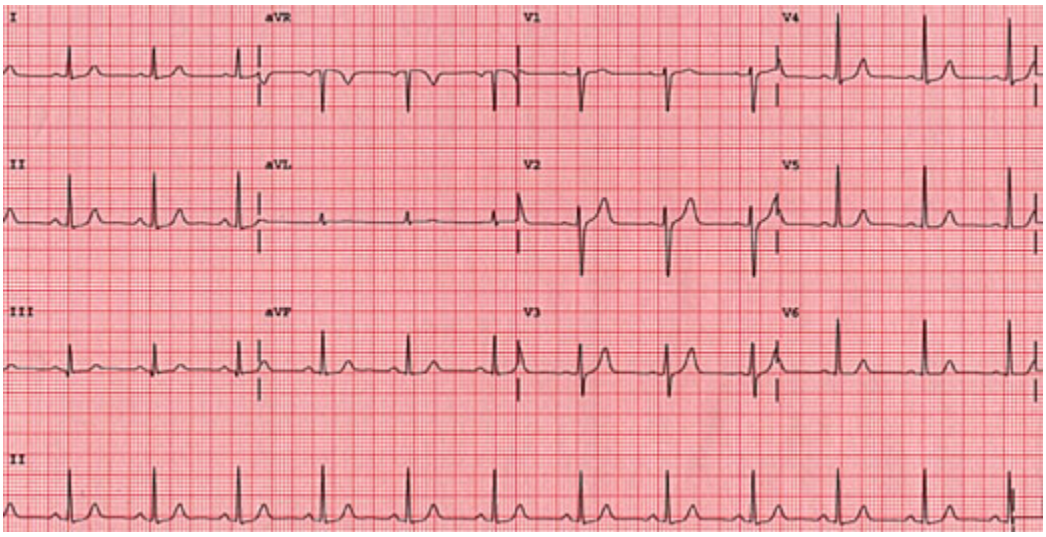


Figure 29: Normal axis.

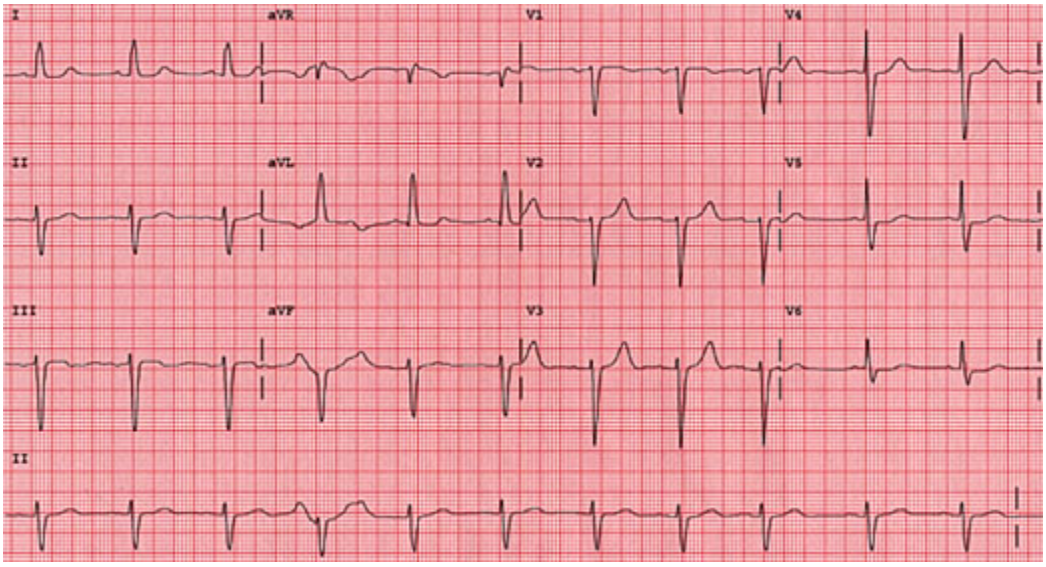


Figure 30: Left axis deviation.

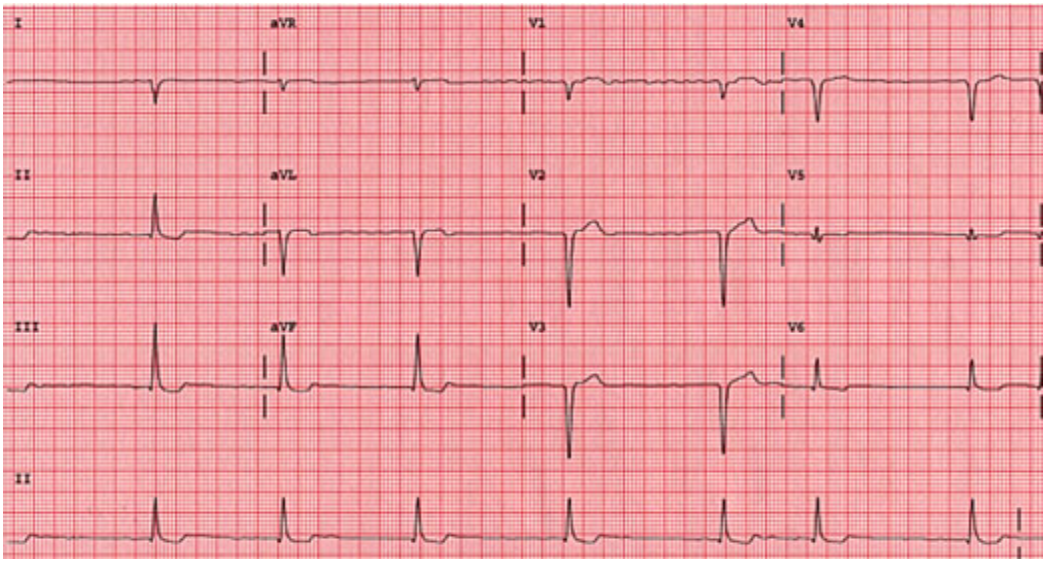


Figure 31: Right axis deviation.

The bottom line is, *if the axis is shifted out of the normal quadrant*, evaluate the reasons for this.

	Differential Diagnosis
Left axis deviation	LVH, left anterior fascicular block, inferior wall MI
Right axis deviation	RVH, left posterior fascicular block, lateral wall MI

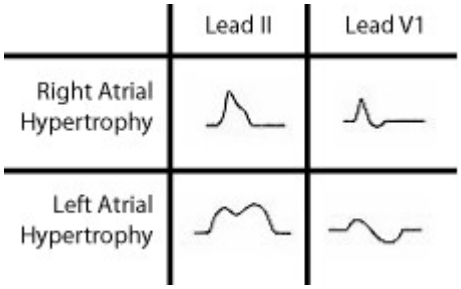
HYPERTROPHY

Hypertrophy criteria are fairly straightforward; we will be looking for enlargement of any of the four chambers.

1. LVH: (Left ventricular hypertrophy). Add the larger S wave of V1 or V2 (not both), measure in mm, to the larger R wave of V5 or V6. If the sum is > 35mm, it meets "voltage criteria" for LVH. Also consider if R wave is > 12mm in aVL. LVH is more likely with a "strain pattern" which is asymmetric T wave inversion in those leads showing LVH.

2. RVH: (Right ventricular hypertrophy). R wave > S wave in V1 and R wave decreases from V1 to V6.

3. Atrial hypertrophy: (leads II and V1). Right atrial hypertrophy - Peaked P wave in lead II > 2.5mm amplitude. V1 has increase in the initial positive deflection. Left atrial hypertrophy - Notched wide (> 3mm) P wave in lead II. V1 has increase in the terminal negative deflection.



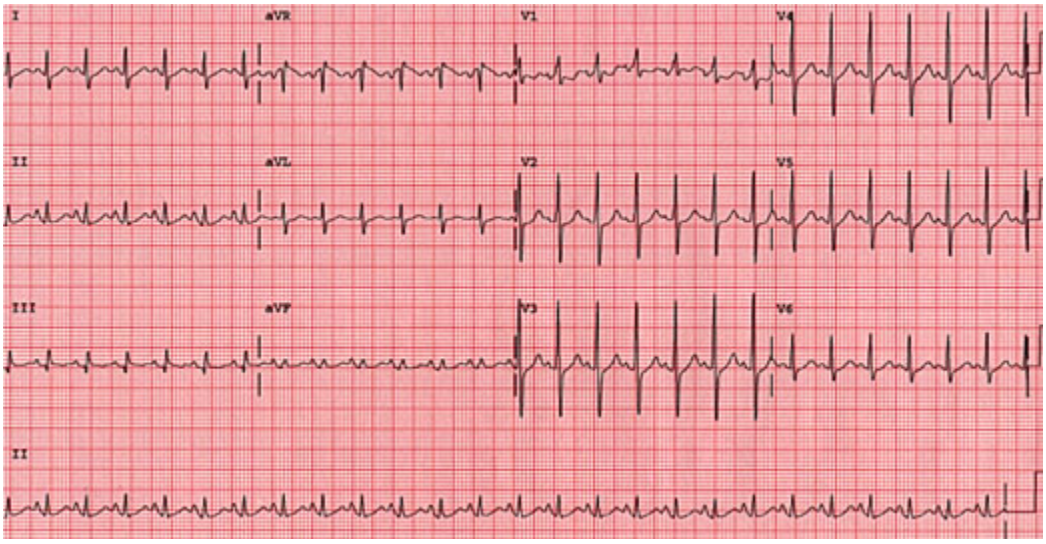


Figure 32: Right ventricular hypertrophy and right atrial enlargement.

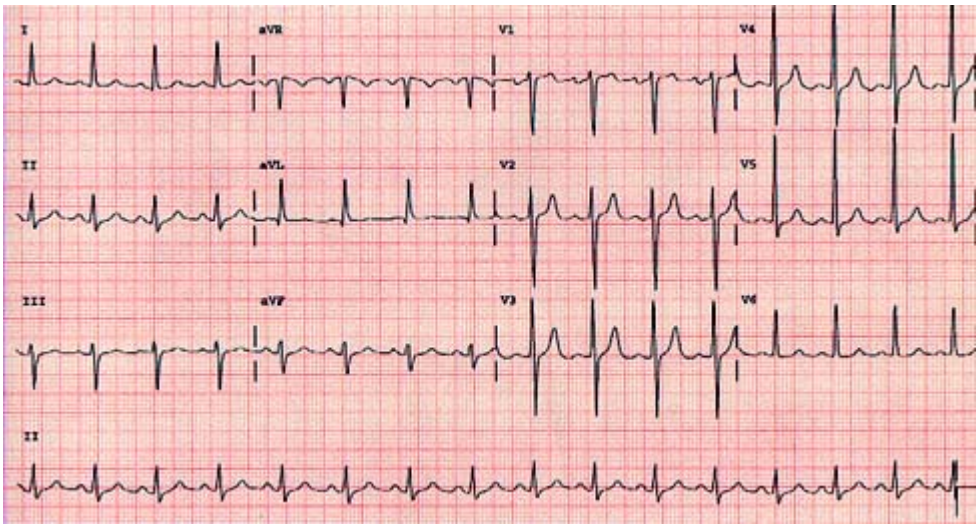


Figure 33: Left ventricular hypertrophy (S wave V2 plus R wave of V5 greater than 35mm) and left atrial enlargement (II and V1).

INFARCT

Accurate ECG interpretation in a patient with chest pain is critical. Basically, there can be three types of problems - *ischemia* is a relative lack of blood supply (not yet an infarct), *injury* is acute damage occurring right now, and finally, *infarct* is an area of dead myocardium. It is important to realize that certain leads represent certain areas of the left ventricle; by noting which leads are involved, you can localize the process. The prognosis often varies depending on which area of the left ventricle is involved (i.e. anterior wall myocardial infarct generally has a worse prognosis than an inferior wall infarct).

V1-V2	anteroseptal wall
V3-V4	anterior wall
V5-V6	anterolateral wall
II, III, aVF	inferior wall
I, aVL	lateral wall
V1-V2	posterior wall (reciprocal)

Infarct	
1. Ischemia	Represented by **symmetrical T wave inversion** (upside down). The definitive leads for ischemia are: I, II, V2 - V6.
2. Injury	Acute damage - look for **elevated ST segments**. (Pericarditis and cardiac aneurysm can also cause ST elevation; remember to correlate it with the patient.

3. Infarct	Look for significant "patholgic" **Q waves**. To be significant, a Q wave must be at least one small box wide or one-third the entire QRS height. Remember, to be a Q wave, the initial deflection must be down; even a tiny initial upward deflection makes the apparent Q wave an R wave.

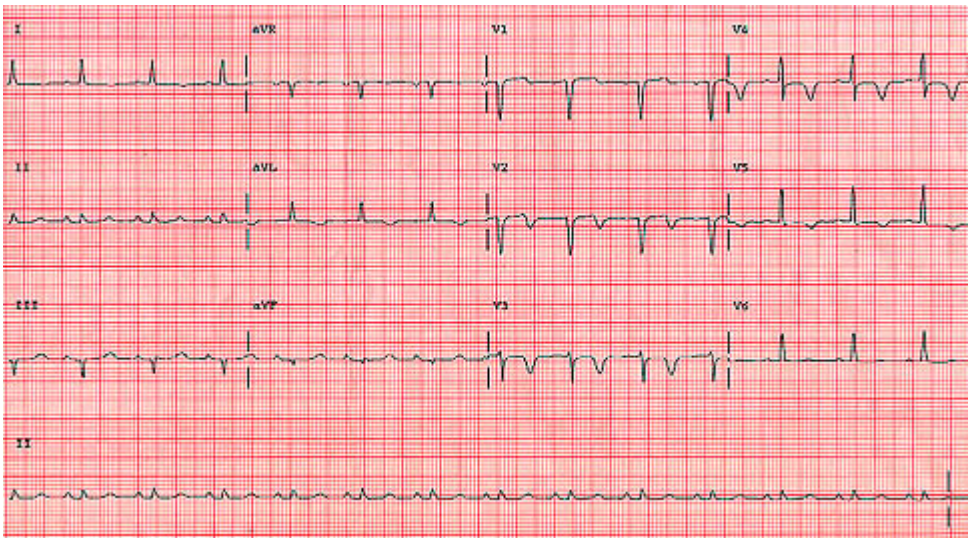


Figure 34: Ischemia: Note symmetric T wave inversions in leads I, V2-V5.

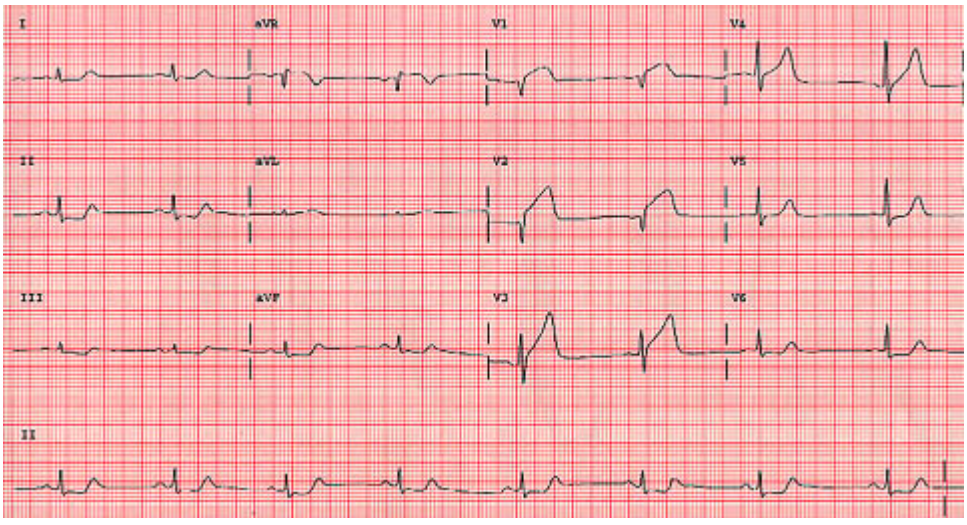


Figure 35: Injury: Note ST segment elevation in leads V2-V3 (anteroseptal/anterior wall).

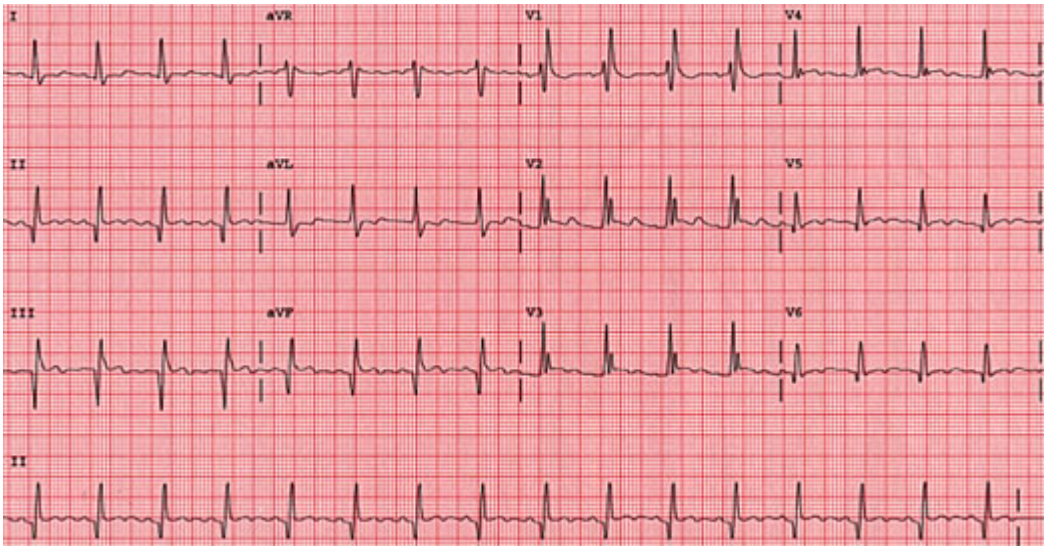


Figure 36: Infarct: Note Q waves in leads II, III, and aVF (inferior wall).

For the posterior wall, remember that vectors representing depolarization of the anterior and posterior portion of the left ventricle are in opposite directions. So, a posterior process shows up as *opposite* of an anterior process in V1. Instead of a Q wave and ST elevation, you get an R wave and ST depression in V1.

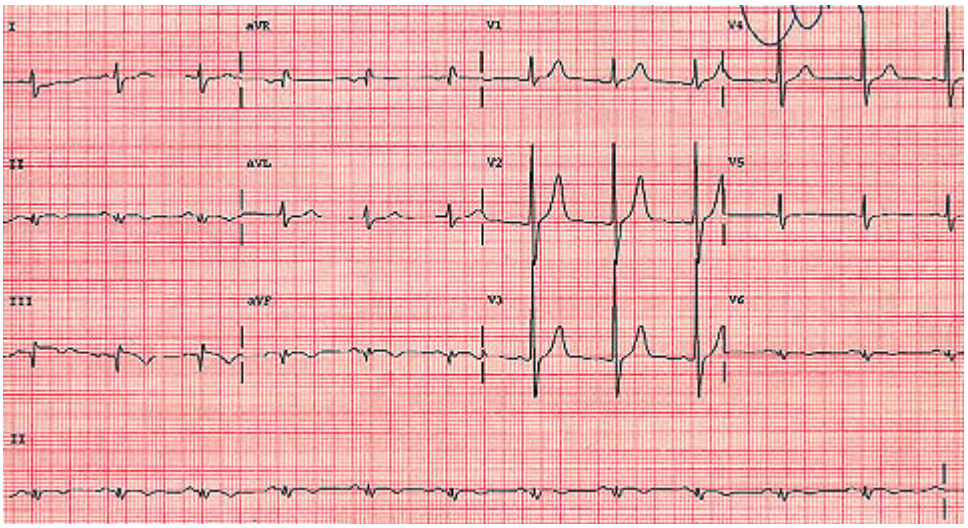


Figure 37: Posterior wall infarct. Notice tall R wave in V1. Posterior wall infarcts are often associated with inferior wall infarcts (Q waves in II, III and aVF).

Two other caveats: One is that normally the R wave gets larger as you go to V1 to V6. If there is no R wave "progression" from V1 to V6 this can also mean infarct. The second caveat is that, with a left bundle branch block, you cannot evaluate "infarct" on that ECG. In a patient with chest pain and left bundle branch block, you must rely on cardiac enzymes (blood tests) and the history.

FASCICULAR BLOCKS

Fascicular blocks are blocks of part of the left bundle, either the posterior or anterior division:

Figure 38: Divisions of the bundles.

Anterior fascicular block - the most common.

You will see left axis deviation (-30 to -90) and a small Q wave in lead I and an S in lead III (Q1S3). The QRS will be slightly prolonged (0.1 - 0.12 sec).

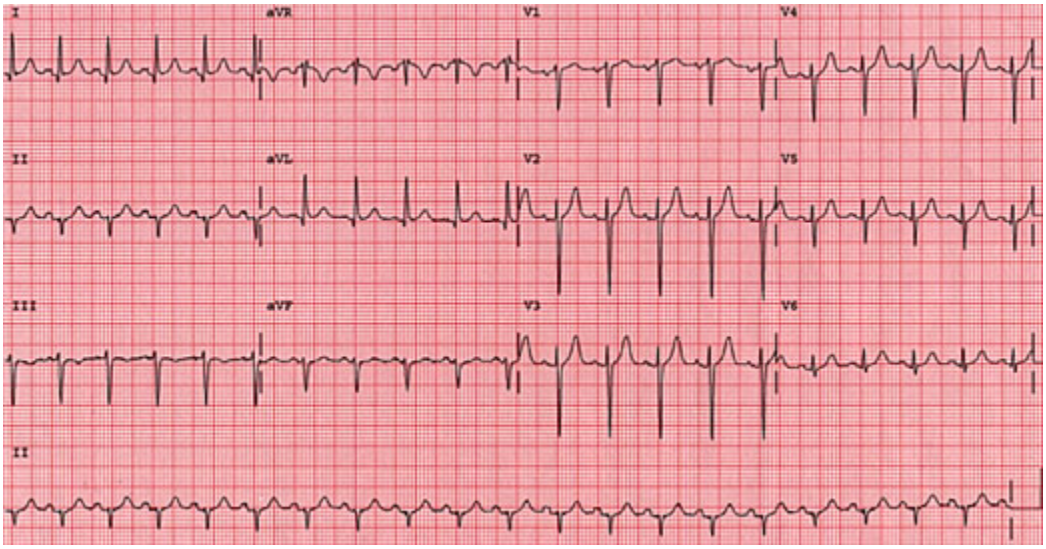


Figure 39: Anterior fascicular block.

Posterior fascicular block - less common.

You will see right axis deviation, an S in lead I and an Q in lead III (S1Q3). The QRS will be slightly prolonged (0.1 - 0.12 sec).

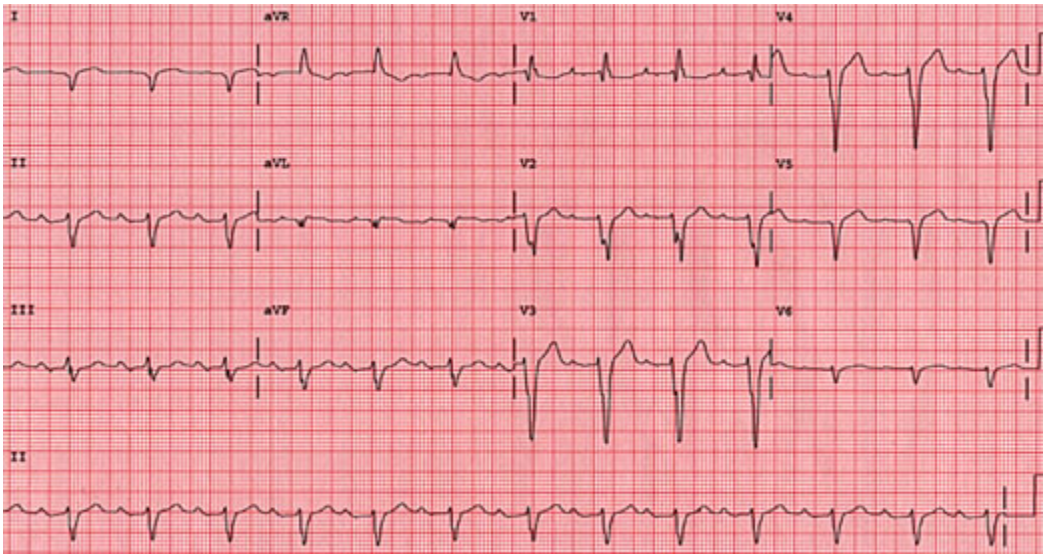


Figure 40: Posterior fascicular block.

Bifascicular block.

This means two (2) of the three (3) fascicles (in diagram) are blocked. The most important example is a right bundle branch block and a left anterior fascicular block. Watch out for this. Only one fascicle is left for conduction, and if that fascicle is intermittently blocked, the dangerous Mobitz 2 is set up!

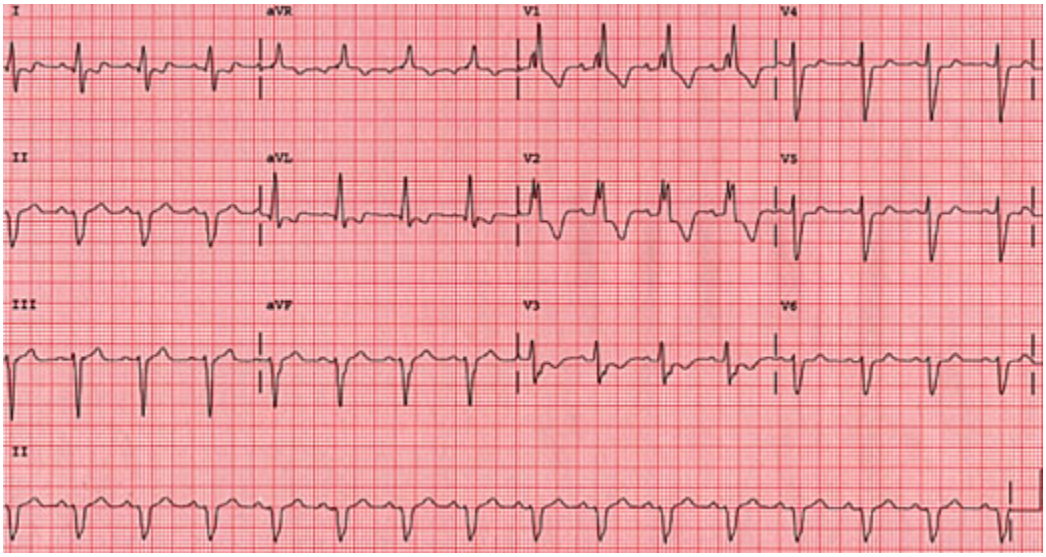


Figure 41: Right bundle branch block and left anterior fascicular block.

"Fascicular Blocks" may seem a bit complicated - simply remember that axis deviation is the clue. In your differential, consider posterior fascicular blocks with right axis deviation and consider anterior fascicular blocks with left axis deviation. Fascicular blocks cause axis deviations, like infarcts and hypertrophy. If you see a left or right axis deviation, first look for infarct or hypertrophy. If neither are present, the remaining diagnosis of fascicular block is usually correct. Review differential diagnosis of right and left axis deviation.

ONE LAST DIFFERENTIAL DIAGNOSIS

Four cases of an R wave taller than an S wave in V1 (normally R wave always < S wave in V1.

1. Right bundle branch block.
2. Right ventricular hypertrophy.
3. Posterior wall myocardial infarction.
4. Wolff-Parkinson-White.

SUGGESTIONS/SUMMARY

1. Look at each ECG for rate, rhythm, axis, hypertrophy, and infarct. The systematic interpretation guidelines below will serve as a quick reference

INTERPRETATION GUIDELINES *for Electrocardiograms*

RATE

Rate calculation
Common method: 300-150-100-75-60-50
Mathematical method: 300/# large boxes between R waves
Six-second method: # R-R intervals x10

RHYTHM

Rhythm Guidelines:
1. Check the bottom rhythm strip for regularity, i.e. - regular, regularly irregular, and irregularly irregular.
2. Check for a P wave before each QRS, QRS after each P.
3. Check PR interval (for AV blocks) and QRS (for bundle branch blocks). Check for prolonged QT.
4. Recognize "patterns" such as atrial fibrillation, PVC's, PAC's, escape beats, ventricular tachycardia, paroxysmal atrial tachycardia, AV blocks and bundle branch blocks.

AXIS

	Lead I	Lead aVF
1. Normal axis (0 to +90 degrees)	Positive	Positive
2. Left axis deviation (-30 to -90) Also check lead II. To be true left axis deviation, it should also be down in lead II.	Positive	Negative
3. Right axis deviation (+90 to +180)	Negative	Positive
4. Indeterminate axis (-90 to -180)	Negative	Negative

Left axis deviation differential: LVH, left anterior fascicular block, inferior wall MI.
Right axis deviation differential: RVH, left posterior fascicular block, lateral wall MI.

HYPERTROPHY

1. LVH -- left ventricular hypertrophy = S wave in V1 or V2 + R wave in V5 or V6 > 35mm or aVL R wave > 12mm.
2. RVH -- right ventricular hypertrophy = R wave > S wave in V1 and gets progressively smaller to left V1-V6 (normally, R wave increases from V1-V6).
3. Atrial hypertrophy (leads II and V1)

Right atrial hypertrophy -- Peaked P wave in lead II > 2.5 mm in amplitude. V1 has increase in the initial positive direction.

Left atrial hypertrophy -- Notched wide (> 3mm) P wave in II. V1 has increase in the terminal negative direction.

INFARCT

Ischemia	Represented by symmetrical T wave inversion (upside down). Look in leads I, II, V2-V6.
Injury	Acute damage -- look for elevated ST segments.
Infarct	"Pathologic" Q waves. To be significant, a Q wave must be at least one small square wide or one-third the entire QRS height.

Certain leads represent certain areas of the left ventricle:

V1-V2	anteroseptal wall	II, III, aVF	inferior wall
V3-V4	anterior wall	I, aVL	lateral wall
V5-V6	anterolateral wall	V1-V2	posterior wall (reciprocal)

.

GLOSSARY of Terms used in relation to Hyperbaric Oxygen Therapy

Aeroembolism :Obsolete term for altitude decompression sickness; also used to mean gas embolism.

Aerobic: Requiring air or free oxygen in order to live.

Air: 78% nitrogen, 21% oxygen, 1% carbon dioxide and all other gases.

Ambient : Pertaining to the surrounding environment.

Anaerobic : The ability to grow or thrive in the absence of molecular oxygen.

Angiogenesis : The development of blood vessels. Angiogenesis is a major benefit of HBO therapy.

Ascent : Movement in the direction of reduced pressure, whether simulated or due to actual elevation in water or air.

Atmospheres absolute :The sum of barometric and hydrostatic pressures. This is the most commonly used measurement when dealing with HBO therapy. The abbreviation is ATA.

Atmospheric pressure :The amount of pressure exerted by the weight of the air in our every day environment. At sea level the pressure of the atmosphere is 14.7 pounds per square inch.

Baromedicine : The area of medicine related to physiological processes that occur either from pressure changes or changes in the concentration of inhaled gases.

Barotrauma :The mechanical damage to the tissues caused by unequal pressures.

Bends : An imprecise term denoting any form of Caisson disease or decompression sickness. It is sometimes a fatal disorder that is marked by neuralgic pains (severe pain along a nerve) and paralysis, and dyspnea (difficult breathing); that is caused by the release of gas bubbles in tissue upon too rapid decrease in pressure after a stay in a compressed environment.

Bottom time :The amount of time from getting in the water at the start of a dive until the beginning of the ascent.

Chamber : A vessel designed to withstand differential pressures.

> **Double-lock** : A chamber with two compartments that can be pressurized independently.
>
> **Hyperbaric** : A chamber designed to withstand high internal pressures; used in hyperbaric experimentation, diving simulations, and medical treatment.
>
> **Monoplace**: A portable one-person hyperbaric chamber used for therapy in a hospital or clinical setting, and for transport.

Multiplace : A pressure chamber designed to be used by more than one person at a time.

Single-lock : A pressure chamber with only one pressurizable compartment.

Decompression sickness : A condition caused by too rapid a reduction in pressure, and having a variety of signs and symptoms. Synonymous with the bends, Caisson disease, or compressed air illness. The abbreviation is DCS.

Decompression :In diving, that phase in which the individual is ascending in the water, or in a chamber when the pressure is being lowered.

Embolism: Air or gas bubbles in the arterial (artery: a vessel conveying blood from the heart) system caused by gas or air passing into the pulmonary (lung) veins after rupture of the alveoli (air cells of the lung).

HBO:Common abbreviation for hyperbaric oxygen. HBO is the use of increased oxygen concentrations under greater than normal atmospheric pressure.

Hydrostatic: Relating to the pressure that liquids exert.

Hyperbaric oxygenation: Also known as hyperbaric oxygen therapy. The use of an oxygen breathing mixture where the ambient pressure is greater than 1 atmosphere. This is abbreviated as HBO or HBO_2.

Hyperbaric :Pertaining to pressure greater than one atmosphere.

Hyperbaric pressure :Pressures greater than atmospheric pressure.

Hyperoxia : An excess of oxygen in the body tissues produced by breathing a mixture in which the inspired (inhaled) oxygen pressure is greater than its partial pressure in air.

Hypoxia :Oxygen deficiency.

Ischemia : Local reduction of blood supply due to obstruction of inflow of arterial blood.

Microaerophilic :Requiring free oxygen for growth, but thriving best when the oxygen is less than the amounts in the atmosphere.

Monoplace chamber : An HBO chamber designed to hold one person, usually at a maximum pressure of 3 ATA.

Multiplace chamber : A HBO chamber designed to hold two or more persons, usually with pressures of up to 6 ATA.

Osteomyelitis :Inflammation of the marrow of the bone.

Osteonecrosis :The process of bone cells dying in mass.

> **Dysbaric**: Changes in structure of bone in which the relative density of the affected bone is increased by sclerosis (hardening). Observed changes are the result of a healing process following insult (trauma). Found in caisson workers and more recently in divers, and probably due to inadequate decompression. Synonymous with aseptic bone necrosis and avascular bone necrosis.
>
> **Juxta-articular** : Osteonecrosis occurring near the joint articulation, usually hip or shoulder. May lead to collapse of the joint, together with pain and dysfunction.
>
> **Medullary** : Osteonecrosis occurring in the shaft of the bone, usually symptomless and detected by x-ray.

Otitis: Inflammation of the ear, which may be marked by pain, fever, abnormalities of hearing, tinnitus (ringing in one or both ears), and vertigo (dizziness); a very common problem in diving.

Oxygen poisoning : Deleterious (harmful) effects caused by breathing high partial pressures of oxygen. Prolonged exposure can result in effects which become progressively more severe as the inspired partial pressure and/or the duration of exposure increases. Depending on level and length of exposure, may cause lung damage, involvement of the central nervous system causing convulsions, or early death.

Oxygen toxicity : Physical impairment that results from breathing pure oxygen for prolonged periods of time; the time to achieve toxicity is shortened as the pressure in the surrounding environment increases.

Pressure

> **Absolute** :The sum of all pressures acting on an object; in diving, the sum of the atmospheric (air) pressure and the hydrostatic (water) pressure acting on a submerged object.
>
> **Ambient** : The absolute pressure surrounding an object.
>
> **Atmospheric** : Pressure exerted by the earth's atmosphere, which varies with altitude above sea level. At sea level atmospheric pressure is equal to 760 mmHg or 1.03 kg/square centimeter, or 14.7 pounds per square inch.
>
> **Hydrostatic** : The pressure of a column of water acting upon a body immersed in the water, equal in all directions at a specific depth.

Pressurize : To increase the internal pressure of a closed vessel.

Treatment depth : The depth or pressure to which a patient is compressed for treatment.

Glossary of Terms and Abbreviations relating to Spirometry

ATPS: Ambient temperature and pressure saturated with water vapor. Volumes read directly off the volume-time spirogram are at ATPS.

Back extrapolation: In the calculation of FEV_1, a method for determining the time zero. A straight line is drawn through the steepest portion of the volume-time curve back to the baseline. Where this straight line intersects the baseline is the zero point for timing the FEV_1.

Best curve: That curve which gives the largest sum of FEV_1 and FVC. The best curve is used in the calculation of the $FEF_{25-75\%}$ and the instantaneous flow rates. In contrast, the largest FVC and the largest FEV_1 are reported for the test session, even if they are not from the same curve.

BTPS: Body temperature and pressure saturated with water vapor. All spirometric volumes and flows must be corrected to BTPS.

Calibration check: Periodic determination of a spirometer's ability to accurately measure volume. Calibration checks should be performed at least daily using a three liter syringe. The instrument should maintain an accuracy of 3% of the reading. Additional checks include checking for leaks (daily for volume spirometers), and, every 3 months, verifying the accuracy of a timed chart and checking the linearity of volume recording.

End of test: That point during the forced expiratory maneuver when a plateau at least one second long is noted on the volume-time tracing

Extrapolated volume: That volume determined by a line drawn through the zero time point perpendicular to the baseline on a volume-time curve. The extrapolated volume is read where this perpendicular line intersects the volume curve; it should be less than 5% of the FVC or 150 ml, whichever is greater.

FEV_1/FVC%: Forced expiratory volume in one second expressed as a percentage of the forced vital capacity.

Flow-measuring spirometer: Indirectly measures volume of exhaled air by measuring the rate at which air is exhaled and deriving the volume. Examples include pneumotachometer, mass flow, and turbine instruments.

Forced expiratory Volume in one second (FEV_1): Volume of air exhaled during the first second of the FVC.

Forced expiratory Volume in six second (FEV_6): Volume of air exhaled during the first six seconds of the FVC. Since it is easier for obstructed subjects to reach the FEV_6 than the FVC, there is growing interest in measuring the FEV_6 and the FEV_1/FEV_6 in screening spirometry.

Forced expiratory maneuver: Technique during spirometry where the subject takes the deepest possible inspiration from a normal breathing pattern and blows into the mouthpiece as hard, fast and completely as possible. Also known as the forced vital capacity maneuver.

Forced Vital capacity (FVC): The maximal volume of air exhaled from the point of maximal inspiration using a maximally forced expiratory effort.

Mean forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$): Average flow rate over the middle half of the expiration. Formerly called the maximal mid-expiratory flow rate (MMEF).

Predicted normal values: Expected values for various lung volumes and flow rates derived from healthy populations.

Reproducibility: In the absence of disease-related changes, the ability of a test to obtain the same result from an individual repeatedly tested over a period of time. Reproducibility of the FEV_1 and FVC within a test session should be 0.20 liters or less.

Residual Volume: Volume remaining in the lungs following a maximal expiration.

Spirogram: A graphic recording of a forced expiratory maneuver, as either a volume-time or flow-volume tracing.

Spirometer: An instrument for measuring lung volumes and flow rates.

Total Lung Capacity: Total lung volume following a maximal inspiration.

Valid Test: A spirometry test consisting of at least three acceptable forced expiratory tracings where the best FVC and the best FEV_1 are reproduced within 0.2 L.

Volume-measuring spirometer: Spirometers which directly accumulate and measure the volume of exhaled air as a function of time. Examples include water-seal, dry rolling seal and bellows instruments.

Zero time point:. In the measurement of FEV_1, the point selected as the start of the test.

Glossary of Terms and Abbreviations relating to ECG

1/f-spectral trend	Trend in which the power of the spectrum is inversely proportional to the frequency *f* according to a $1/f^A$ power law
Acrophase	see Cosinor Analysis
Akaike information criterion	Method for the order selection of autoregressive models
Aliasing	Error occurring when the sampling frequency of analog-to-digital conversion is lower than twice the highest frequency contained in the signal (Nyquist frequency)
Anacrotic pulse	A small slow-rising pulse with a notch on the ascending limb
Analytic signal	Signal with Fourier transform equal to 0 for frequencies lower than 0
Alpha index	Estimate of the sensitivity of the baroreceptor-heart rate reflex based on frequency-domain analysis of spontaneous variability of systolic blood pressure and R-R interval.
Approximate entropy	Measure of the regularity or predictability of time series
Attractor	Object to which the time series in a phase space is attracted to
Autocorrelation function	Measure of the dependence of time series values at one time on the values at another time
Autoregressive modeling	Time-series modeling based on the assumption that each value of the series depends on a weighted sum of the previous values of the same series plus "noise"
Autoregressive moving average modeling	Time-series modeling based on the assumption that each value of the series depends on a weighted sum of previous values and of the present and previous values of a different time series with the addition of "noise"
Barahona-Poon test	Test suitable to detect nonlinear dynamics in short, noisy time series
Baroreflex Effectiveness Index	Ratio between the number of systolic blood pressure ramps followed by the respective reflex pulse interval ramps and the total number of systolic blood pressure ramps observed in a given time window
Bartlett	Type of data window
Bisferiens pulse	A pulse with two peaks during systole
Bivariate autoregressive modeling	Modeling of two time-series based on the assumption that each value of the two series depends on a weighted sum of the previous values of the same series plus a second weighted sum of the present and previous values of the other series plus "noise"
Bispectrum	See higher-order spectra

Blackmann-Tukey method	Estimate of power spectral density of a time series based on the evaluation of the autocorrelation function and on the computation of its Fourier transform
Broadband spectrum	Spectrum providing spectral estimates over a frequency region wider (sometimes much wider) that the region usually explored by the standard spectral procedures employed in a given field
Broadband smoothing	Smoothing technique for power spectra in which the resulting frequency resolution and estimation variance are not constant over the frequency axis, as in conventional spectra
Cardiac events series	HRV signal derived from the ECG by considering the occurrence of each R-wave an event, and by expressing the events by infinitesimal pulses or delta-functions
Chaos	Aperiodic behaviour of a given variable of a bounded deterministic system which may appear as random behaviour
Choi-Williams distribution	Time-frequency distribution derived from the Wigner-Ville distribution
Coarse graining spectral analysis	Technique useful to separate random fractal components from a time series in the frequency domain
Coefficient of variation	measure of relative dispersion defined by: CV=100x(standard deviation/mean), used in those conditions where mean and standard deviation tend to change together
Coherence function	Degree of linear correlation between two signals as a function of the frequency
Complex demodulation	Procedure which allows to describe particular frequency components of a time series as a function of time
Cosinor analysis	Least square approximation of time series using a cosine function of known period
Correlation dimension	A measure of the fractal dimension of the time series.
Cross-correlation function	Estimates the linear correlation coefficient between two signals as a function of a time lag between the two.
Cross Mutual Information Function	see Mutual Information
Cross-terms	Oscillatory positive and negative peaks, affecting some time-frequency distributions, due to interferences between spectral components
Cubic splines	See Spline Funtion
Cumulants	Extension of the concept of autocorrelation for multiple lags, used to define higher-order spectra

Data window	A function of time that is multiplied by a data segment. It is mainly used before computing the FFT spectrum of the data segment, and its purpose is to smooth or otherwise shape the resulting spectrum
Determinism	Rule allowing to determine the evolution of a system or a variable when the causes are known. Similar causes induce similar effects.
Detrended fluctuation analysis	Method for quantifying the correlation property in nonstationary time series based on the computation of a scaling exponent *d* by means of a modified root mean square analysis of a random walk.
Diastogram	Time series derived from consecutive diastolic blood pressure values
Dicrotic wave	A pulse appearing in the blood pressure wave during the diastolic phase
Discrete time Fourier transform	See Fourier Transform
ECG-Derived Respiration	Respiratory signal derived from the ECG by assessing the fluctuations in the cardiac electric axis due to respiration.
Embedding dimension	Number of axis of a return map sufficient to describe the properties of the corresponding phase space
Energy	See Power
Entropy	Measure of uncertainty in a sequence of symbols
Ergodic process	Random process in which every time series produced is the same in statistical properties
European Data Format	Standard format for exchange and storage of multisignal biological time series
Fast Fourier transform	Family of computationally efficient algorithms for the calculation of the discrete-time Fourier transform
Filter (analog)	Device that stops specific components of a signal before A/D conversion, generally used to avoid aliasing or to arrest interferences and noises (e.g., conducted interference or muscle activity in the ECG)
Filter (digital)	System that separates specific components of a time series
Final prediction error	Method for the order selection of autoregressive models (see Akaike Information Criterion)
Fourier Series	Decomposition of a periodic signal into a series of sine and cosine waves
Fourier transform	Frequency-domain representation of a signal as a complex-valued function, characterized by a modulus function and a phase function
Fractal	Object with self-similar structure across scales, so that resembling structures appear as one zoomes in and out
Fractal dimension	Definition of the geometric dimension of an object which includes fractal objects.

Frequency resolution	Minimum difference in frequency between two sinusoids which still allows to resolve two distinct peaks in the spectrum
Hann	Type of data window
Hamming	Type of data window
Heart rate	Reciprocal of the R-R interval, expressed in beats per minute
Heart rate turbulence	The physiological response of the sinus node to premature ventricular contractions characterized by a short acceleration followed by a deceleration of the heart rate.
Heart timing signal	HRV signal used to estimate the spectrum of the modulating input *m(t)* when the IPFM model is assumed
High-Pass	Type of digital filter
Hausdorff dimension	see Fractal Dimension
Hurst exponent	Measure of the *smoothness* of fractal time series based on the asymptotic behaviour of the rescaled range of the process
Higher order spectra	Spectra obtained from the Fourier transform of cumulants
Impulse response	Response of a system when its input is an infinitly short impulse with finite energy. The behaviour of linear systems to any input can be predicted by the convolution between the input signal and the impulse response
Interpolation	Mathematical procedure used to estimate an intermediate value between samples of a time series
Instantaneous heart rate	HRV signal defined as the series of the reciprocal RR-interval durations as a function of time
Integral pulse frequency modulation model	System which transforms a continuous input signal into an event series
Interval function	HRV signal defined as the series of RR-interval durations as a function of time
Interval tachogram	HRV signal defined as the series of RR-interval durations as a function of the interval number
Iso-spectral surrogate data	Artificial time series derived from the original time series which has the same linear characteristics (same variance, same spectrum, same mean) as the original time series, but is otherwise random
Leakage	Loss of power from a frequency band to several adjacent spectral lines due to

	the finite data set over which the periodogram is estimated: leakage may be controlled by applying a data window before the spectrum computation
Levinson-Durbin algorithm	Efficient computational procedure to solve the Yule-Walker equations
Linear system	System which fulfills the principle of linear superimposition: a linear combination of the inputs will produce the same linear combination of the outputs.
Lomb periodogram	Method of spectral analysis for unevenly sampled series, such as the beat-to-beat series of cardiovascular signals
Low-pass	Type of digital filter
Low-pass filtered event series	HRV signal obtained by filtering the R-wave event series by a low-pass filter
Lyapunov exponents	Average exponential rates of divergence of initially close orbits in the phase space
Marginal conditions	Conditions, satisfied by certain time-frequency distributions, on the integrals over frequency and over time
Multivariate parametric models	See autoregressive moving average modeling and bivariate autoregressive modeling
Mutual Information	Measure of the statistical dependency between two random variables based on Shannon's entropy
Noise	Random signal with specific spectral and energy properties
Nonlinear system	a system whose time-evolution equations are non linear (i.e., the variables describing the dynamical properties of the system, such as velocity, accelaration etc. appear in equations in a nonlinear form); this implies that a linear combination of the inputs does not produce the same linear combination of the outputs (see also linear system).
Normalized Entropy	Entropy of a time-series divided by a measure of the time-series energy.
Nyquist frequency	see Aliasing
Nyquist-Shannon theorem	see Sampling theorem
P wave	First deflection of the electrocardiogram corresponding to atrial depolarization
Passband	Type of digital filter
Periodogram	Estimate of power spectral density on the basis of modulus squared Fourier

	transform
Phase resetting	Change in phase induced by an external stimulus
Phase space	Space in which each point describes the state of a dynamical system as a function of the non-constant parameters of the system
Poincaré section	Intersection between a plane and trajectories in a phase space
Polyspectra	Synonym of higher-order spectra
Power	Time average of time-series energy
Power leakage	see Leakage
Power spectral density	Amount of *power* per unit (*density*) of frequency (*spectral*) as a function of the frequency
Pulse interval	Time duration between two consecutive systolic blood pressure peaks (see also Systogram)
Q wave	First downward deflection of the electrocardiogram following the P-wave, arising from ventricular activation
R wave	First upward deflection of the electrocardiogram following the Q-wave, arising from ventricular activation
Recurrence plot	Graphic representation of the data used to reveal nonstationarities or periodicity in the phase-space orbits.
Rescaled range	Measure characterising the divergence of time series defined as the range of the sum of the deviations of data from the mean divided by the sample standard deviation
Return map	Plot of a time series as a function of the current and of the previous values.
Resampling	Procedure which derives an evenly-sampled series from a time series sampled on a beat-by-beat basis
Reverse arrangement test	Test on the stationarity of a time series
RR interval	Time duration between two consecutive R waves of the ECG
Run test	Test on the stationarity of a time series
S wave	First downward deflection of the electrocardiogram following the R-wave, arising from ventricular activation
Sampling frequency	Rate of extraction of discrete numeric data from an analog continuous signal
Sampling theorem	The sampling frequency must be higher than twice the highest frequency contained in the signal to allow the original signal to be reconstructed as accurately as desired from the sequence of samples
Sequence technique	Method for estimating the sensitivity of the baroreceptor-heart rate reflex by time-domain analysis of spontaneous variability of systolic blood pressure and

	pulse interval
Set point	Specific value of a controlled variable (eg, arterial blood pressure) that should be mantained by a given control mechanism (eg, the arterial baroreflex).
Shannon theorem	The sampling frequency of a signal must be higher than twice the highest frequency contained in th signal to allow a reconstruction of the signal without aliasing error.
Short-time Fourier transform	Method for Fourier analysis of nonstationary signals, based on a joint time-frequency signal representation
Sign Analysis	Analysis of the properties of the increments (signs) of a beat-by-beat time series
Smoothness index	Index of the homogeneity of the blood pressure reduction induced by antihypertensive treatment over the 24 hours
Spectral technique	Method for estimating the sensitivity of the baroreceptor-heart rate reflex based on the computation of the Alpha Index
Spectrogram	Method for the study of nonstationary signals based on splitting the signal in short-time sliding windows and on getting the Fourier spectrum in each window, in order to obtain a time-varying spectral description.
Spectrum	Description of a quantity as any function of frequency. See also Power Spectral Density
Spectrum of counts	Power spectrum of the cardiac event series
Spline function	Interpolating function which guarantees global smoothness up to some order derivative (e.g., cubic splines produce an interpolated function continuous up to the 2nd derivative)
Stationarity	Property of a time series in which probability distributions involving values of the time series are independent of time translations
Step response	Response of a system when its input is a step function
Surrogate data	see iso-spectral surrogate data
Symbolic dynamics	Coarse graining the data by transforming the time-series in a sequence of symbols.
Systogram	Time series derived from consecutive systolic blood pressure values
T wave	Upward deflection of the electrocardiogram following the S wave, due to ventricular repolarization
Tachogram	Time series derived from the occurrence time of the R-wave. See also cardiac events series, interval tachogram, interval function, instantaneous heart rate , heart timing signal and low-pass filtered event series
Time-Frequency Distribution	Description of energy or power of a signal as a two-dimensional funtion of both time and frequency

Toeplitz matrix	a matrix composed by elements which are constant along the diagonals
Turbulence	see Hear Rate Turbulence
Transfer function	Complex-valued function of the frequency expressing the relationship between output and input of a system
Transfer function technique	Method for estimating the sensitivity of the baroreceptor-heart rate reflex by assessing the transfer function between systolic blood pressure and R-R interval
Trigonometric regressive spectral analysis	Decomposition of beat-by-beat time series (RR intervals, peripheral blood pressure) into a small number of periodic oscillations and a non-rhythmic residual variance on the base of trigonometric regressive functions
Trough-to-peak ratio	Index of the distribution of the blood pressure reduction induced by treatment throughout the dosing interval
U wave	Small upward deflection of the electrocardiogram of uncertain origin sometimes seen following the T wave
Variance	For time-series, it is the time average of the square of deviations from the mean value (see also Power)
Warner model	Set of equations describing the heart-rate responses to the frequencies of stimulation of vagus and sympathetic efferent nerves
Wavelets analysis	Analysis which expands the signal in terms of "small waves", or wavelets: this is done analogeous to Fourier transform where the signal is expanded in sinusoids
Welch method	Periodogram estimate based on the splitting of the time series in overlapped segments multiplied by data windows, and on the ensemble average of periodograms computed in each data window
Window	see Data Window
Windowed Fourier transform	Synonym of short-time Fourier transform
Wigner-Ville distribution	Time-frequency distribution of a signal with very high time and frequency resolution
Yule-Walker equations	Set of linear equations relating the parameters of an AR model with the autocorrelation sequence
Z analysis	Statistical evaluation of the relationship between systolic blood pressure and heart rate based on the calculation of the Z coefficient
Zero-padding	Method used to artificially increase the length of a time series by adding zero-value samples before computing the Fast Fourier Transform

PFT Examination

Select the ***best*** answer to each of the following items. Mark your responses on the Answer form.

1. Pulmonary function testing is one of the basic tools for evaluating a patient's respiratory status. In patients with suspected pulmonary disease, it is often _____.

a. overlooked by the primary physician
b. the only test employed during the diagnosis
c. the first diagnostic test employed in the work up
d. postponed because of the lack of trained personnel

2. Spirometry with flow volume loops assesses the mechanical properties of the respiratory system by measuring inspiratory volumes and flow rates.

a. True
b. False

3. _____ loops provide a graphic illustration of a patient's spirometric efforts. Flow is plotted against volume to display a continuous loop from inspiration to expiration.

a. Spirogram trace
b. Broncho
c. Flow volume
d. PFT

4. In general, a > 12% increase in the FEV1 (an absolute improvement in FEV1 of at least _____ ml) or the FVC after inhaling a beta agonist is considered a significant response.

a. 10
b. 40
c. 100
d. 200

5. Similarly, spirometry can be used to detect the bronchial hyperreactivity that characterizes _____.

a. COPD
b. bronchiectasis
c. tuberculosis

d. asthma

6. After spirometry is completed, the patient is given _____ and the test is repeated.

a. a mild sedative
b. an inhaled bronchodilator
c. an hour to rest
d. treadmill exam

7. PEF is the peak expiratory flow rate, showing the peak flow rate during inspiration

a. True
b. False

8. FVC is the forced vital capacity, or the volume of air that can be maximally forcefully exhaled.

a. True
b. False

9. In terms of PFT and Spirometry results, there is no single set of standard reference values, however, and "normal" varies with the reference value used in each laboratory.

a. True
b. False

10. The _____ has (have) published guidelines for the standardization of spirometry equipment and performance.

a. American Lung Association (ALA)
b. American Chest Physicians (ACP)
c. American Thoracic Society (ATS)
d. American Medical Association (AMA)

11. Ensuring adequate patient effort depends on the technician measuring spirometry.

a. True
b. False

12. The volume versus time curve is an alternative way of plotting spirometric results and is another useful illustration of patient performance.

a. True
b. False

13. Test acceptability is best determined by examining the flow volume loop and _____. Variable effort, cough, and early glottic closure can be seen on the graphs but may not be apparent by simply looking at values for FEV1 and FVC.

a. volume time curve
b. distance variations
c. inspiratory rate
d. none of the above

14. Normal values for FEV1 and FVC are based on population studies and vary according to race, height, age, and gender. They are expressed in both absolute numbers and percent predicted of normal. Some authors have suggested that defining normal by _____ confidence intervals would be more statistically appropriate, particularly at the extremes of age.

a. 75%
b. 80%
c. 90%
d. 95%

15. The primary abnormality detected by spirometry is airways obstruction.

a. True
b. False

16. Spirometry should be interpreted using the flow volume and volume time curves as well as the absolute values for flows and volumes.

a. True
b. False

17. The primary abnormality detected by spirometry is airways obstruction.

a. True
b. False

18. In restrictive lung disease, both the FEV1 and FVC are increased proportionately.

a. True
b. False

19. Upper airway obstruction is less common than lower airway obstruction; however it can be suggested by spirometry. Upper airway obstruction includes variable extrathoracic obstruction, variable intrathoracic obstruction, and fixed intra- or extrathoracic obstruction.

a. True
b. False

20. Variable _____ may be caused by vocal cord paralysis, thyromegaly, tracheomalacia, or neoplasm while large airways variable intrathoracic obstructions can also result from tracheomalacia or neoplasm.

a. loop volumes
b. flow rates
c. extrathoracic obstructions
d. age of the patient

HBO Examination

Select the ***best*** answer to each of the following items. Mark your responses on the Answer form.

21. The air we breathe contains _____ oxygen.

a. 9.5%
b. 15%
c. 21%
d. 33%

22. Hyperbaric oxygen therapy is the administration of _____ oxygen at two to three times atmospheric pressure.

a. 40%
b. 60%
c. 75%
d. 100%

23. **Multiplace** chambers accommodate between four and _____ patients.

a. seven
b. fifteen
c. twenty-four
d. fifty five

24. The _____ was founded in 1967 and is the major scientific society for hyperbaric oxygen therapy in the United States.

a. Jacques Cousteau Society
b. Undersea and Hyperbaric Medicine Society (UHMS)
c. Hyperbaric Oxygen Therapy Committee
d. American Hyperbaric Therapy Society (AHTS)

25. Hyperbaric oxygen therapy is also used in the treatment of _____ poisoning and smoke inhalation.

a. carbon dioxide
b. carbon monoxide
c. nitric oxide
d. all of the above

26. *Clostridia* myonecrosis, otherwise known as _____, is a severe infection caused by gram positive bacteria of the *Clostridia* variation.

a. the bends
b. gas gangrene
c. gas embolism
d. fibroplast infection

27. Research indicates that hyperbaric oxygen therapy acts as an alpha-adrenergic drug, which causes vasoconstriction.

a. True
b. False

28. The most common side effect of HBO therapy is a "crackling" sound in the ears between treatments.

a. True
b. False

29. Much less frequently, a temporary change in vision is experienced. This alteration in vision returns to its normal state within six to eight days after treatment has finished.

a. True
b. False

30. Contraindications for administration of hyperbaric oxygen therapy include:

a. History of thoracic surgery
b. Pneumothorax
c. Severe chronic obstructive pulmonary disease with carbon dioxide retention
d. All of the above

31. The most common use of HBO therapy among pediatric patients is for carbon monoxide poisoning.

a. True
b. False

32. The Undersea and Hyperbaric Medical Society approves the use of hyperbaric therapy for many conditions, which are accepted by third-party payers and Medicare.

a. True
b. False

33. As early as _____ experimental clinical applications of hyperbaric medicine were reportedly used with patients who had tuberculosis and other chronic diseases.

a. 1492
b. 1662
c. 1830
d. 1867

34. _____ states that the total pressure of a gas mixture is equal to the sum of the partial pressures of each gas in the mixture; for example, the total pressure of air equals the sum of the partial pressures of nitrogen, oxygen, and air's other component gases

a. *Boyle's law*
b. *Dalton's law*
c. *Gay-Lussac's law*
d. *Henry's law*

35. HBO therapy decreases the partial pressure of arterial oxygen (PaO2), resulting in vasoconstriction, which can aid the treatment of trauma wounds by reducing edema, reducing capillary pressure, and allowing a better flow of hyper-oxygenated plasma to the tissues.

a. True
b. False

36. Oxygen can be toxic to the lungs. After ___ hours of continuously breathing 100% oxygen at increased atmospheric pressures, the alveolar lining can be damaged.

a. 8
b. 16
c. 24
d. 72

37. *Menstruation* can lead to a greater risk for decompression illness in the early phase of the menstrual cycle[12]; even a slight increase in discharge at any time during the course of treatment must be brought to the HBO therapy physician's attention for evaluation.

a. True
b. False

38. HBO therapy was introduced in the United States in _____, primarily as treatment for "nervous" disorders and influenza and other respiratory conditions.

a. 1800
b. 1830
c. 1861
d. 1907

39. The following conditions absolutely contraindicate the administration of HBO therapy:
a. some types of congenital heart disease
b. obstructed airway or restrictive airway disease (such as asthma)
c. recent thoracic surgery
d. all of the above

40. HBO therapy results in long-term decreased immune system response that enhances allografts, but patients must be monitored for infections.

a. True
b. False

ECG Examination

Select the ***best*** answer to each of the following items. Mark your responses on the Answer form.

41. In the illustration above, the "T" wave section represents which of the following:

a. repolarization of the ventricle
b. depolarization of the ventricle
c. depolarization of the atria
d. none of the above

42. In the illustration above, the "P" wave section represents which of the following

a. repolarization of the ventricle
b. depolarization of the ventricle
c. depolarization of the atria
d. none of the above

43. ECG paper has 1-millimeter small squares - so height and depth of wave is measured in _____.

a. inches
b. micrometers
c. millimeters
d. none of the above

44.In ECG terms, _____ is cycles or beats per minute.

a. Rhythm
b. Rate
c. Axis deviation
d. Pulse

45. Accurate ECG interpretation in a patient with _____ is critical.

a. chest pain
b. lung cancer
c. breathing difficulties
d. none of the above

46. In the case of an _____, ECG interpreters should look for significant "pathologic" **Q waves**. To be significant, a Q wave must be at least one small box wide or one-third the entire QRS height.

a. injury
b. ischemia
c. infarct
d. none of the above

47. "Fascicular Blocks" may seem a bit complicated - simply remember that _____ is the clue.

a. rhythm variances
b. axis deviation
c. wave deviation
d. none of the above

48. "Arrhythmia" means abnormal _____.

a. hypertrophy
b. pulse rate
c. wave length
d. rhythm

49. In ECG, direction of depolarization (vector) of the _____ complex.

a. QRS
b. AV
c. VF
d. LVH

50. In calculating "rate" by the _____ method, you count off 30 large boxes, then count the number of R-R intervals in six seconds and multiply by 10. This is the number of beats per minute.

a. mathematical
b. six-second
c. most common
d. none of the above

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