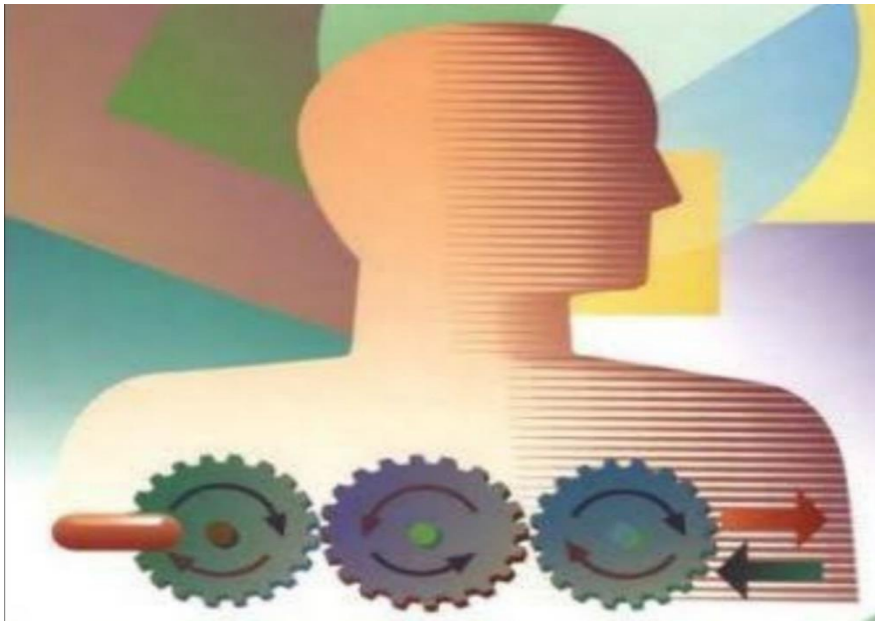
Physical Therapy and Rehabilitation for Cardiothoracic Diseases and Surgery



(Book 1)

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2024-2025

Content

Introduction to cardiac physical therapy

Heart: Heart Pumps Blood into Two Circuits in Sequence

- Pulmonary circuit to and from the lungs.
- Systemic circuit to and from the body.

Two Sets of Pumping Chambers in Heart

- **Right atrium:** Receives systemic blood
- **Right ventricle**: Pumps blood to lungs (pulmonary)
- **Left atrium**: Receives blood from lungs
- **Left ventricle**: Pumps blood to organ systems (systemic)

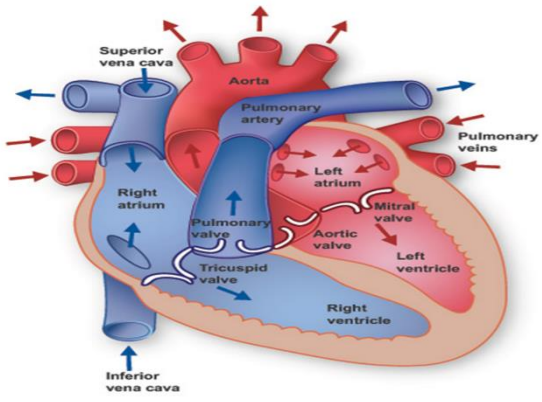
Heart Valves: Four valves regulate blood flow through your heart:

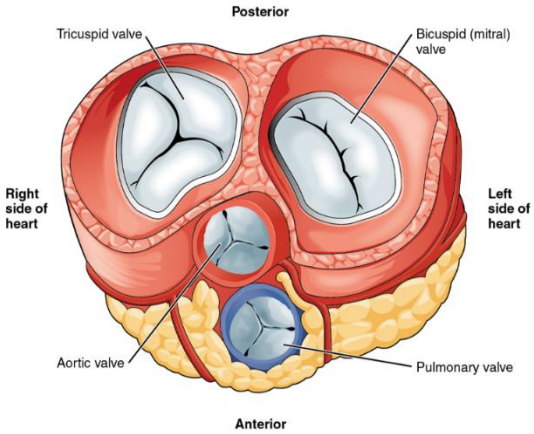
— **Tricuspid valve** right atrium and right ventricle.

— **Pulmonary valve** right ventricle into pulmonary arteries

— **Mitral valve** left atrium into the left ventricle.

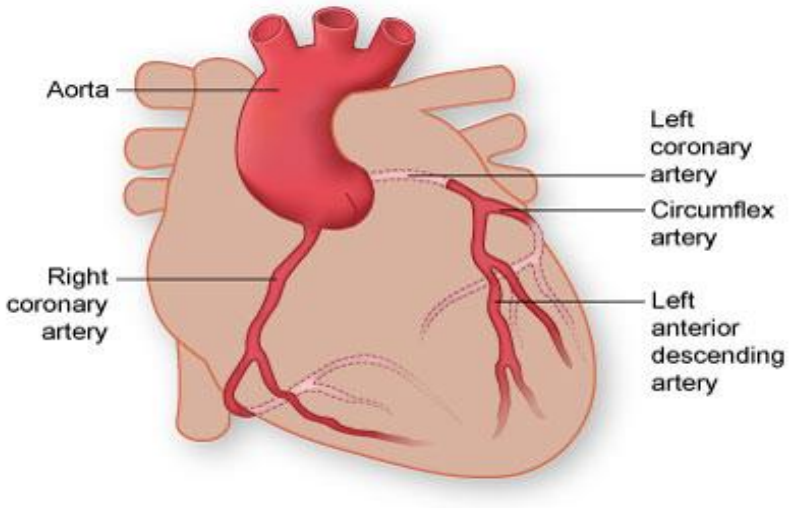
— **Aortic valve** left ventricle into the aorta





Coronary artery:

- **Left main coronary artery:** supplies blood to the left side of the heart muscle (left ventricle and left atrium).
  - — ***Left anterior descending artery*** supplies blood to the front of the left side of the heart.
  - — ***The circumflex artery*** supplies blood to the outer side and back of the heart.
- **Right coronary artery:** supplies blood to the right ventricle, the right atrium, SA and AV nodes
  - — ***Right posterior descending artery***
  - — ***Marginal artery***
- **Left anterior descending artery and right coronary artery helps supply blood to the middle or septum of the heart.**



Coronary circulation: circulation of blood in the blood vessels of the heart muscle myocardium known as coronary arteries. The vessels that remove the deoxygenated blood from the heart muscle are known as cardiac veins.

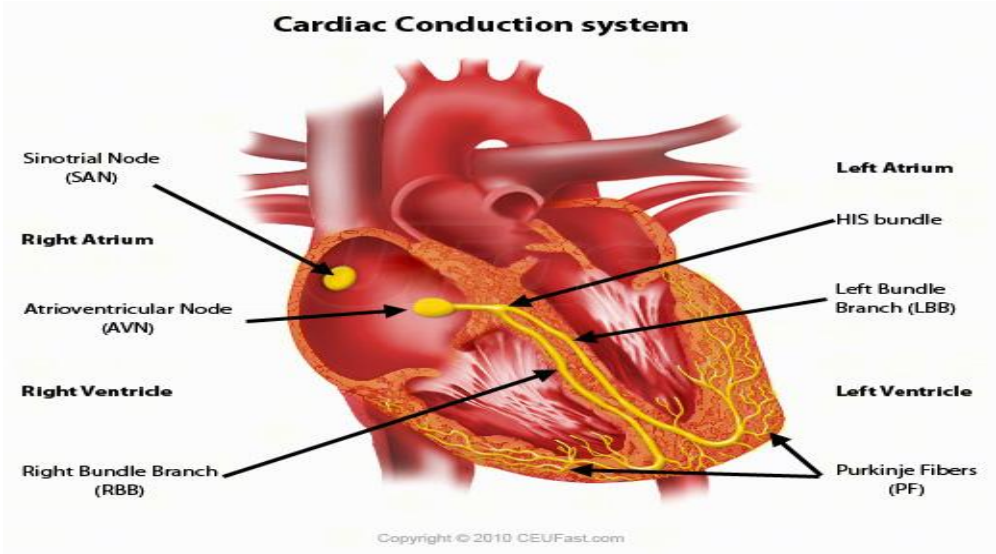
Cardiac veins: great cardiac vein, middle cardiac vein and small cardiac vein and the anterior cardiac veins.

Coronary circulation refers to the circulation of blood in blood vessels of the human heart. It is an essential process that delivers oxygen-rich blood to the coronary arteries. In addition to

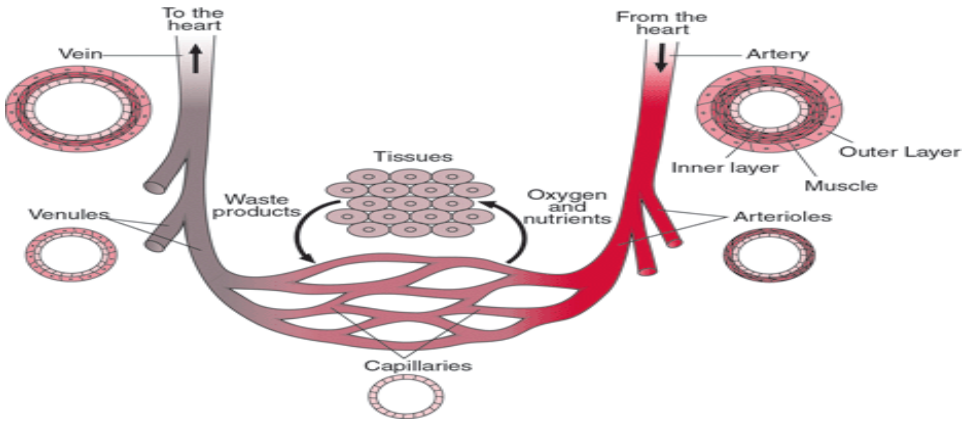
supplying the heart with blood, coronary circulation provides drainage systems to remove deoxygenated blood.

Cardiac conduction system:

The cardiac conduction system is a group of specialized cardiac muscle cells in the walls of the heart that send signals to the heart muscle causing it to contract. The main components of the cardiac conduction system are the SA node, AV node, bundle of His, bundle branches, and Purkinje fibers



Blood vessel: a tubular structure carrying blood through the tissues and organs; a vein, artery, or capillary.



Cardiac output:

CO L/min= SV L/beat x HR beats/min

Cardiac output: is amount of blood pumped by the heart per minute.
= 5 to 6 liters per minute.

Heart rate: is the number of beats per minute = 60 to 100 beats/min

Stroke volume: is amount pumped per beat = 60-70 ml/heartbeat.

Tachycardia: is a resting heart rate more than 100 beats per minute.

— This number can vary as smaller people and children have faster heart rates than average adults.

— ***Physiological conditions where tachycardia occurs:***

1. Exercise
2. Pregnancy
3. Emotional conditions such as anxiety or stress.

— ***Pathological conditions where tachycardia occurs:***

1. Sepsis
2. Fever
3. Anemia
4. Hypoxia
5. Hyperthyroidism
6. Cardiomyopathy
7. Valvular heart diseases

Bradycardia: heart rate less than 60 beats per minute

Conditions where Bradycardia occurs:

- Changes in the heart that are the result of aging.
- Diseases that damage the heart's electrical system. These include coronary artery disease, heart attack, and infections such as endocarditis and myocarditis.
- Conditions that can slow electrical impulses through the heart. Examples include having a low thyroid level
- Some medicines for treating heart problems or high blood pressure, such as beta-blockers, antiarrhythmic, and digoxin.

Symptoms of cardiac diseases

1. Symptoms of lung congestion
2. Symptoms of systemic congestion
3. Pain (cardiac pain)
4. Low cardiac output system
5. Palpitation
6. Mediastinal compression syndrome

Symptoms of lung congestion:

- Dyspnea or difficulty breathing
- Haemoptysis: coughing up blood
- Excessive sweating, anxiety and pale skin.
- Orthopnea: Difficulty breathing when lying down
- Paroxysmal nocturnal dyspnea (episodes of severe sudden breathlessness at night).
- Raised jugular venous pressure.

Symptoms of systemic congestion:

- Congested pulsated neck vein.
- Congested pulsated enlarged tender liver.
- Ascites.
- Peripheral edema (swelling of the legs, in general, of the "pitting" variety, wherein the skin is slow to return to normal when pressed upon).

- Dyspepsia sense of pain or discomfort after pain.

Pain (cardiac pain)

- Onset
- Site of pain
- Reference of pain
- Character of pain
- Provoking factors
- Relieving factors

- Duration of pain
- Associating factors

Onset: Acute: angina and myocardial infarction

Site of pain: Retrosternal: angina, myocardial infarction, pericarditis, dissecting aortic aneurysm.

Reference of pain: Retrosternal pain radiating to the right side of neck, left shoulder and inner side of left arm …… ischemic heart diseases.

Character of pain: Compressing, chocking, crushing, pressing or suffocating …… ischemic heart diseases.

Tearing ……dissecting aortic aneurysm.

Duration of pain

Minutes …… anginal pains.

Hours ……. myocardial infarction pain.

Provoking (Aggravating) factors

Exercise and emotions …….. ischemic heart disease.

Relieving factors:

Rest and nitroglycerine …… anginal pains.

Sitting up ……. Pericarditis.

Associating factors

Sweating and palpitation …… angina.

Causes of chest pain

- Cardiac neurosis.
- Angina pectoris.
- Acute myocardial infarction*.
- Acute pericarditis.
- Acute pulmonary embolism.

- Dissecting aortic aneurysm*.
- Aortalgia.

Symptoms of Decreased cardiac output

- Chest pain
- Fatigue
- Exercise intolerance
- Abnormal heart rhythm
- Rapid breathing
- Dizziness and Fainting
- Reduced urine output
- Anxiety
- Cold clammy skin
- Reduced peripheral pulse

Mediastinal syndrome: Compression of mediastinal structures by any tumors gives rise to a group of symptoms known as mediastinal syndrome.

Common causes are:

- Bronchogenic carcinoma
- Hodgkin's disease causing enlargement of the mediastinal lymph nodes
- Aneurysm or dilatation of aorta

Common symptoms of mediastinal syndrome

- Pressure over the trachea causes dyspnea and cough
- Pressure on the esophagus causes dysphagia
- Pressure on the left recurrent laryngeal nerve gives rise to hoarseness of voice

- Pressure on the intercostal nerve gives rise of pain in the area supplied by them. It is called intercostal neuralgia.
- Pressure on the phrenic nerve causes paralysis of the diaphragm on that side

Ischemic heart disease: is a condition of recurring chest pain or discomfort that occurs when a part of the heart does not receive enough blood.

— This condition occurs most often during exertion or excitement, when the heart requires greater blood flow.

— Coronary heart disease (CHD) is a disease in which a waxy substance called plaque builds up inside the coronary arteries. These arteries supply oxygen-rich blood to your heart muscle.

— When plaque builds up in the arteries, the condition is called atherosclerosis. The buildup of plaque occurs over many years.

Ischemic Heart Disease:

- ✓ Myocardial infarction (MI)
- ✓ Sudden cardiac death
- ✓ Angina pectoris (stable angina, unstable angina)
- ✓ Chronic IHD with heart failure

Risk factors:

- High blood cholesterol
- High blood pressure
- Smoking
- Obesity
- Diabetes

- stress
- Lack of physical activity

Diagnosis:

- Chest X Ray
- Blood Tests: check the levels of certain fats, cholesterol, sugar, and proteins in your blood. electrocardiography (ECG)
- Exercise ECG – Stress test
- Echocardiography (including stress echocardiography)
- Coronary angiography
- Magnetic resonance imaging (MRI)

Prevention:

- Diet
- Lifestyle changes that have been shown to be effective to this goal include:
- Weight control
- Smoking cessation
- Avoiding the consumption of trans fats (in partially hydrogenated oils)
- Decrease psychosocial stress.
- Exercise. Aerobic exercise

Management:

- Lifestyle changes
- Medical treatment – drugs (e.g., cholesterol lowering medications, beta-blockers, nitroglycerin, calcium antagonists, etc.);
- Coronary interventions as angioplasty and coronary stent;
- Coronary artery bypass grafting (CABG)

Myocardial infarction: is an ischemic necrosis of the myocardium, caused by occlusion of coronary artery and prolonged myocardial ischemia.

— A heart attack happens if the flow of oxygen-rich blood to a section of heart muscle suddenly becomes blocked and the heart can't get oxygen. Most heart attacks occur as a result of coronary heart disease.

Risk Factors:

- Smoking
- High blood pressure
- High blood cholesterol
- Overweight and obesity
- An unhealthy diet (for example, a diet high in saturated fat, trans fat, cholesterol, and sodium)
- Lack of routine physical activity
- Diabetes

Causes:

- Plaque builds up in the arteries, atherosclerosis.
- Coronary artery spasm
- Taking certain drugs, such as cocaine
- Emotional stress or pain
- Exposure to extreme cold and cigarette smoking

Most Common Symptoms:

Chest pain or discomfort: Discomfort in the center or left side of the chest. The discomfort usually lasts for more than a few minutes or goes away and comes back. It can feel like pressure, squeezing, fullness, or pain. It also can feel like heartburn or indigestion. The feeling can be mild or severe.

Upper body discomfort: You may feel pain or discomfort in one or both arms, the back, shoulders, neck, jaw, or upper part of the stomach (above the belly button).

Shortness of breath: This may be your only symptom, or it may occur before or along with chest pain or discomfort. It can occur when you are resting or doing a little bit of physical activity.

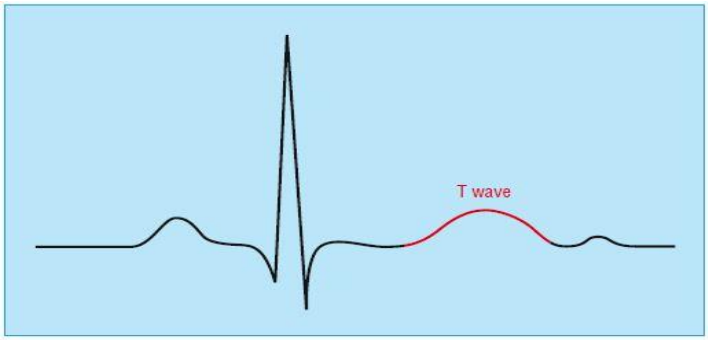
Laboratory Tests:

- Creatine Kinase: CK-MB appears in the serum in 6 to 12 hours, peaks between 12 and 28 hours, and returns to normal levels in about 72 to 96 hours.

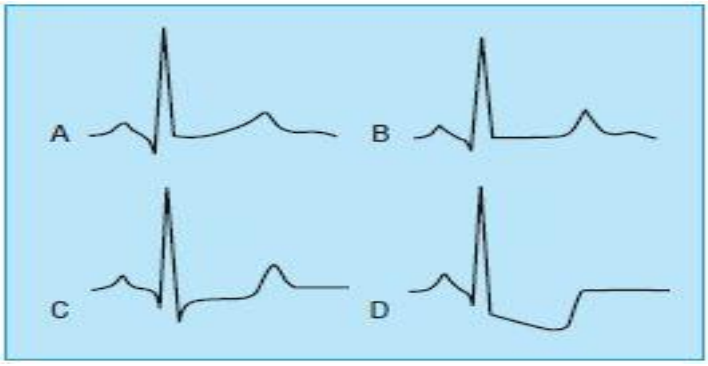
- Serial samplings are performed every 4 to 6 hours for the first 24 to 48 hours after the onset of symptoms
- Myoglobin: The myoglobin level can elevate within 1 to 2 hours of acute MI and peaks within 3 to 15 hours.
- Troponin. (troponin T and troponin I)
    - Troponin I levels rise in about 3 hours, peak at 14 to 18 hours, and remain elevated for 5 to 7 days.
    - Troponin T levels rise in 3 to 5 hours and remain elevated for 10 to 14 days
- Coronary Angiography

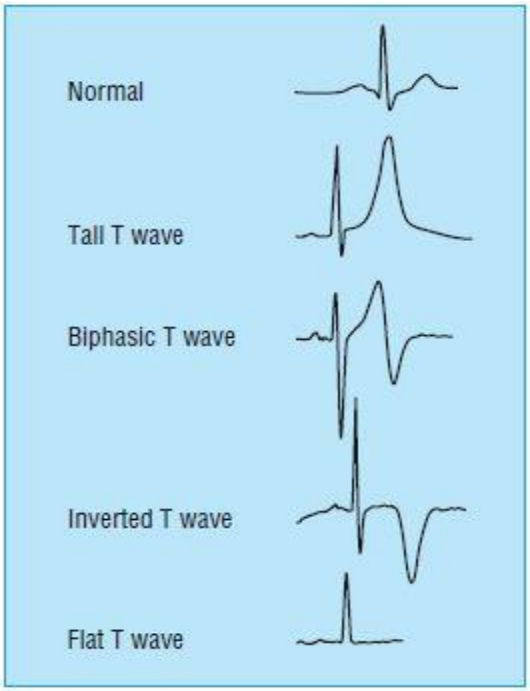
ECG:

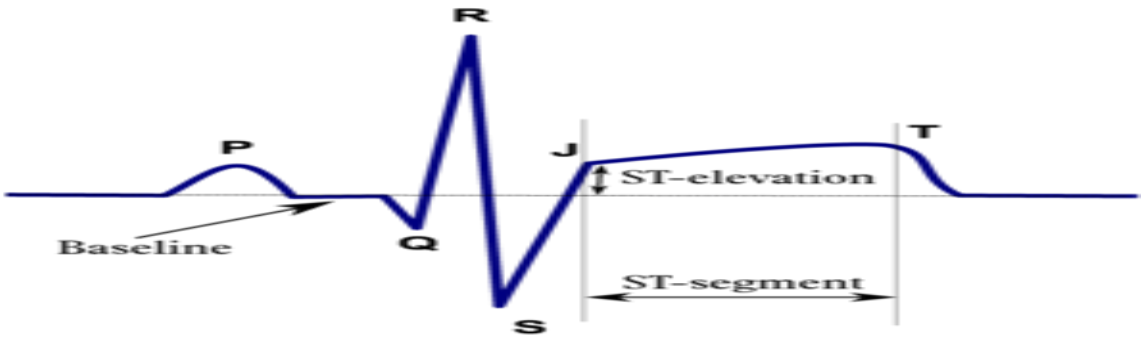
- During the earliest stage of MI, known as the hyper acute phase, the **T waves become tall and narrow**. This configuration is referred to as **hyper acute or peaked T waves**.
- **Within a few hours, these hyper acute T waves invert.**
- Next, the **ST segments elevate**, a pattern that usually lasts from several hours to several days.



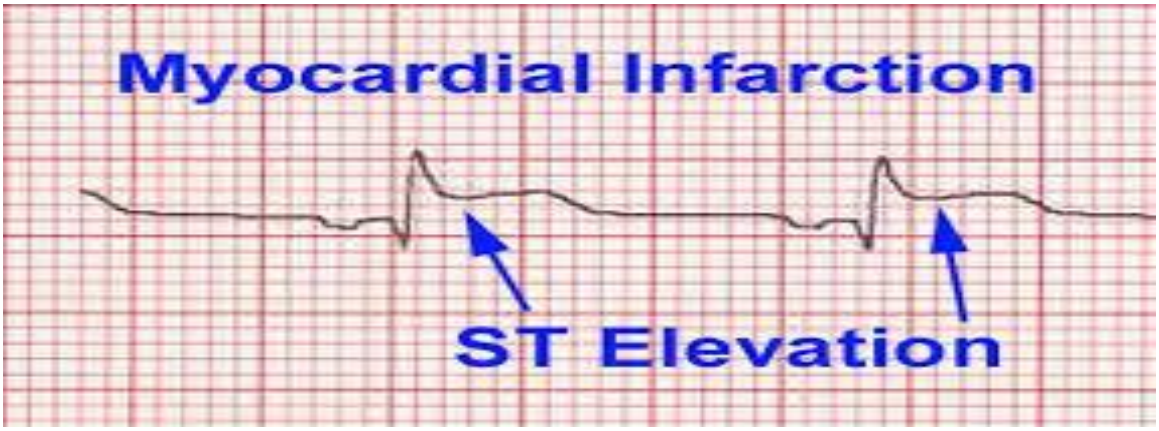
Complex showing T wave highlighted







How to measure ST elevation?



Management:

— The symptoms of angina can be similar to the symptoms of a heart attack. Angina is chest pain that occurs in people who have coronary heart disease, usually when they're active. Angina pain usually lasts for only a few minutes and goes away with rest.

— Chest pain or discomfort that doesn't go away or changes from its usual pattern (for example, occurs more often or while you're resting) can be a sign of a heart attack.

Prevention:

- ✓ Aspirin primary prevention
- ✓ Heart-healthy eating
- ✓ Physically active
- ✓ Quitting smoking
- ✓ Managing stress
- ✓ Managing your weight
- ✓ Lowering High blood pressure
- ✓ Lowering High blood cholesterol
- ✓ Diabetes

Early management

- Administer aspirin, 160 to 325 mg chewed.
- Place the patient on a cardiac monitor and obtain serial ECGs.
- Give oxygen by nasal cannula.
- Administer sublingual nitroglycerin (unless the systolic blood pressure is less than 90 mm Hg or the heart rate is less than 50 or greater than 100 beats/minute.
- Provide adequate analgesia with morphine sulfate. Provide adequate analgesia with morphine sulfate.

Other Treatments for Heart Attack

- Medicines

- ACE inhibitors: ACE inhibitors lower blood pressure.
- Anticlotting medicines: aspirin.
- Anticoagulants. warfarin
- Beta blockers. Beta blockers decrease your heart's workload.
- Statin medicines: Statins control or lower your blood cholesterol Atorvastatin

- Medical procedures: Coronary artery bypass grafting
- Heart-healthy lifestyle changes
- Cardiac rehabilitation

Heart-healthy lifestyle changes:

- ✓ Heart-healthy eating
- ✓ Aiming for healthy weight
- ✓ Managing stress
- ✓ Physical activity
- ✓ Quitting smoking

Atherosclerotic cardiovascular disease (ASCVD)
Prevention Guideline

Atherosclerotic cardiovascular disease is caused by plaque buildup in arterial walls and refers to the following conditions:

- Coronary heart disease (CHD), such as myocardial infarction (MI), angina, and coronary artery stenosis
- Cerebrovascular disease, such as transient ischemic attack, ischemic stroke, and carotid artery stenosis
- Peripheral artery disease, such as claudication.
- Aortic atherosclerotic disease, such as abdominal aortic aneurysm

Atherosclerotic cardiovascular disease:

- **Primary prevention:** refers to the effort to prevent or delay the onset of ASCVD.
- **Secondary prevention:** refers to the effort to treat known, clinically significant ASCVD, and to prevent or delay the onset of disease manifestations.

High-sensitivity C - reactive protein:

- Hs-CRP considers for patients at moderate risk for patients at moderate risk, consider testing hs-CRP.
- This test provides information that is helpful in confirming elevated risk when making the decision whether or not to recommend statin therapy for these Patients

— The American Heart Association and U.S. Centers for Disease Control and Prevention have defined risk groups as follows:
  - Low risk: less than 1.0 mg/L
  - Average risk: 1.0 to 3.0 mg/L
  - High risk: above 3.0 mg/L

Lifestyle Modifications for all Patients:

- ✓ Tobacco cessation
- ✓ Healthy diet
- ✓ Moderation of alcohol consumption
- ✓ Physical activity

- ✓ Weight management
- ✓ Blood pressure management
- ✓ Dietary Supplements

Tobacco cessation:

- Ask patients about tobacco use at every office visit.
- Advise tobacco users to quit.
- Advise patients at every office visit to avoid exposure to environmental tobacco smoke at home, work and in public places.

Healthy diet:

All patients should strive to:

- Make smart choices from every food group to meet caloric needs

Get the most and best nutrition from the calories consumed.

There is strong evidence that adhering to a Mediterranean- style eating plan reduces the incidence of major cardiovascular events in people at high risk for CVD.

Adhering to a DASH eating plan can be an alternative.

Both eating plans provide similar key elements: an emphasis on plant foods (fruits, vegetables, whole-grain breads or other forms of cereals, beans, nuts, and seeds)

There is some evidence that consuming an average of 2 fish servings weekly may reduce CHD mortality.

Physical activity:

The American Heart Association recommends the following physical activity goals:

- At least 30 minutes of moderate-intensity aerobic activity 5 or more days per week.
- Muscle strengthening activity 2 or more days per week
- For patients who have been inactive for a while, recommend that they start slowly and work up to at least 30 minutes per day at a pace that is comfortable.

If they are unable to be active for 30 minutes at one time, suggest accumulating activity over the course of the day in 10- to 15-minute sessions.

Weight management:

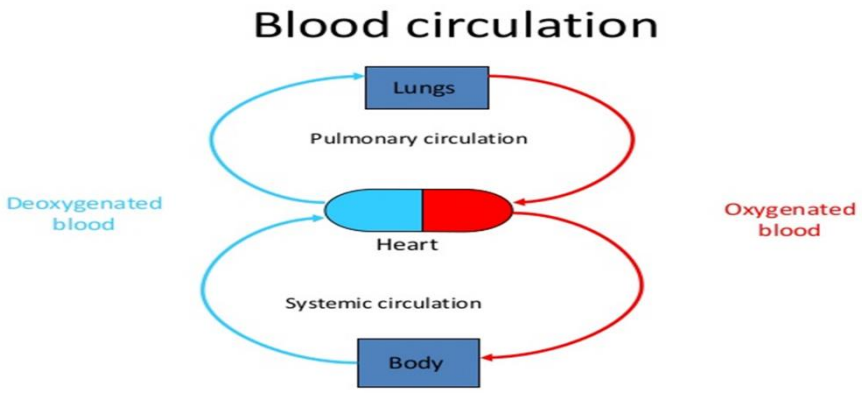
Encourage getting to or maintaining a healthy weight through an appropriate balance of caloric intake and physical activity.

Blood pressure management:

- For patients aged 79 or younger, the blood pressure goal is < 140/90 mm Hg.
- For patients age 80 or older, the blood pressure goal is < 150/90 mm Hg.

Dietary Supplements: Calcium and vitamin D

- If a patient is taking a calcium supplement for the prevention of osteoporosis, recommend that it be taken in combination with vitamin D and that its dose not exceed 1,200 mg per day.
- There is some evidence that calcium supplementation may be associated with increased risk of cardiovascular events, particularly myocardial infarction.
- The co-administration of vitamin D with the calcium supplement may weaken the observed adverse effects of calcium supplementation.
- The literature indicates that intake of calcium from whole foods is not associated with an increased CVD risk.



Pulmonary circulation and systemic circulation

Heart Failure

Heart failure (HF) may be defined as a condition characterized by a reduction in cardiac output that is often insufficient to meet the demands of vital organs and physiological systems.

The etiology of heart failure is caused by the impaired ability of the heart to either pump or accept blood. Inadequate delivery of blood to specific areas may be associated with a variety of physiological sequelae. This may result in fatigue or dyspnea on exertion progressing to dyspnea at rest.

The inability to perform exercise without discomfort may be one of the first symptoms experienced by patients with heart failure and is often the principal reason for seeking medical care.

It is apparent that the person with heart failure may have some compensatory mechanisms to maintain an adequate ejection of blood from the heart. However, these mechanisms may improve or maintain heart function for only a temporary period, after which heart failure will progress.

Heart failure patients are among those who at highest risk of further cardiac events during exercise.

Symptoms of heart failure

- shortness of breath - when you are being active or at rest
- swelling - of your feet, ankles, stomach and lower back areas
- fatigue

Left-sided failure

- Failure of the left side of the heart causes blood to back up (be congested) into the lungs, causing respiratory symptoms as well as fatigue due to insufficient

supply of oxygenated blood. Common respiratory signs are increased rate of breathing and increased work of breathing

Right-sided failure

- difficulties of the pulmonary circulation
- Pitting peripheral edema, ascites, and liver enlargement.
- Jugular venous pressure

Causes

- Coronary artery disease and heart attack.
- high blood pressure
- Cardiomyopathy (diseases of the heart muscle. Sometimes these are inherited from your family and sometimes they are caused by other things, such as viral infections).

Risk factors

- Congenital heart defects
- Valvular heart disease
- Viruses. A viral infection may have damaged your heart muscle.
- Alcohol use.
- Tobacco use.
- Obesity.
- Irregular heartbeats.

Complications

- Kidney damage or failure. Heart failure can reduce the blood flow to your kidneys, which can eventually cause kidney failure if left untreated. Kidney damage from heart failure can require dialysis for treatment.

- Heart valve problems. The valves of your heart, which keep blood flowing in the proper direction through your heart, may not function properly if your heart is enlarged or if the pressure in your heart is very high due to heart failure.
- Heart rhythm problems. Heart rhythm problems (arrhythmias) can be a potential complication of heart failure.

- Liver damage. Heart failure can lead to a buildup of fluid that puts too much pressure on the liver. This fluid backup can lead to scarring, which makes it more difficult for your liver to function properly.

Diagnosis

- medical history
- A blood test to check for a chemical called N-terminal pro-B-type natriuretic peptide (NT-proBNP) may help in diagnosing heart failure if the diagnosis isn't certain when used in addition to other tests.
- Chest X-ray.
- Electrocardiogram (ECG).
- Echocardiogram.
- Stress test.

Lifestyle

- Stop smoking.
- Maintain a healthy weight
- Eat a healthy diet. Aim to eat a diet that includes fruits and vegetables, whole grains, fat-free or low-fat dairy products, and lean proteins.
- Restrict salt in your diet.
- Limit alcohol

- Exercise
- Reduce stress.

Prevention

Lifestyle changes you can make to help prevent heart failure include:

- Not smoking
- Controlling certain conditions, such as high blood pressure and diabetes
- Staying physically active
- Eating healthy foods
- Maintaining a healthy weight
- Reducing and managing stress

Chronic heart failure (CHF) is characterized by an intolerance to exercise, with this group of patients often experiencing early fatigue and shortness of breath. Such symptoms impact upon one's ability to perform activities of daily living, thus significantly contributing to reduced participation and poor quality of life.

Aerobic Exercise and Chronic Heart Failure

- Improved aerobic metabolism
- Improved autonomic regulation
- Improved peripheral perfusion
- Decreased local inflammation
- Improved ventilatory control
- Improved quality of life
- Decreased hospital readmissions and mortality

- Aerobic training and CHF, it has often been noted that functional capacity improves
- The ventilation to carbon dioxide production slope (VE/CO2) is a marker of CHF severity and has also been shown to consistently improve with exercise training
- Improved oxidative capacity and decreased pulmonary vascular resistance
- Aerobic training may have a positive benefit on central sleep apnoea
- Changes in cardiac output (CO) at peak effort may be the result of a change in stroke volume (SV), heart rate (HR), or both. Significant improvement has been noted in the majority of studies
- Improvement in VO2 peak.
- The 6 minute walk test (6MWT) improve in CHF patients post aerobic training
- aerobic training improve total peripheral vascular resistance (TPR) and peripheral perfusion in patients with CHF
- aerobic training corrects endothelial dysfunction in trained extremities, possibly through an upregulation of nitric oxide (NO) synthase locally.

F.I.T.T Principle

- Frequency
- Intensity
- Time
- Type

Frequency

- Current recommendations suggest a minimum frequency of 3-5 days per week

- Twice weekly exercise may be sufficient to maintain functional improvement gained following a center based training program

Time

- Patients should start at short durations (10-20 minutes) and progress to longer session times (30-40 minutes).
- decreased physical activity and a loss of the positive benefits achieved with formal exercise training Such functional decline occurs within 3 weeks post cessation of activity

Intensity

- Ranging from 40-85% VO2peak
- some researchers hypothesize myocardial wall stress is more likely to be minimized at the lower intensities. 50-70% VO2 peak or 60-80% heart rate reserve (HRR) is the intensity usually recommended for rehabilitation program

Determine intensity target heart rate (THR)

- THR = (MHR - RHR) x %TI + RHR
- MHR = 220 – age
- Maximum heart rate MHR
- Training intensity (TI)
- Resting heart rate (RHR)
- MHR – RHR = heart rate reserve (HRR)

Rating of perceived exertion (RPE)

- Patients should also exercise at a rating of perceived exertion between 9-14 on the 6-20 RPE scale
-

Absolute and relative contraindications to resistance training in CHF patients

Absolute	Relative
NYHA classification IV	Unstable angina pectoris
Obstruction of left ventricular outflow	New onset of atrial fibrillation
Decompensated CHF	Severe pulmonary hypertension
Threatening arrhythmias	Complex ventricular arrhythmias at rest
Exercise capacity of ≤3 METs	Arrhythmias which increase in severity/frequency with exercise
Moderate to severe aortic stenosis	Significant exercise-induced ischemia (>3 mm ST segment depression)
Uncontrolled diabetes	

RATE OF PERCEIVED EXERTION (RPE)

BORG RPE	MODIFIED RPE	BREATHING	TRAINING ZONE	% of MHR*	EXERCISE TYPE
6	0	No Exertion	1	50%-60%	Warm up
7		Very Light			
8	1				
9					
10	2	Deeper but comfortable breathing. Able to hold a conversation.	2	60%-70%	Recovery
11					
12	3				
13		Aware that breathing is harder; able to talk but difficult to hold conversation	3	70%-80%	Aerobic
14	4				
15	5	Starting to breathe hard and getting uncomfortable	4	80%-90%	Anaerobic
16	6				
17	7	Deep and forceful breathing. Uncomfortable and not wanting to talk	5	90-100%	VO^2 Max
18	8				
19	9	Extremely hard			
20	10	Maximum exertion			

* % of maximum heart rate

Criteria for termination of exercise program

Symptoms

- Sever chest pain
- Sever dyspnea
- Leg pain, sever cramp and heaviness of limb
- Palpitation and arrhythmia
- Blurred vision an dizziness
- Excessive fatigue

Signs of patient

- Pallor
- Sever sweeting
- Cyanosis
- Confusion
- ataxia

Monitor

- Inappropriate bradycardia: drop of heart rate more than 10 beat\min with increase or no change in exercise intensity.
- Hypertensive response to exercise: Systolic blood pressure raise about 50 mmHg from resting
- Diastolic blood pressure raise 15 mmHg from resting more than that 20 mmHg is consider critical
- Exercise induced hypotension (drop of systolic blood pressure more than 20 mmHg
- Marked S-T segment depression or elevation
- Exercise induced 2^{nd} or 3^{rd} gedree of A-V block.

Normal response of HR and blood pressure (BP) to exercise

- HR gradual increase up to 10-20 only in phase I
- HR return normal after stopping of exercise within 3-4 min
- Systolic and diastolic blood pressure increase gradual within limit (10-15)

Physiological Benefits of Resistance Training

- increased 6 minute walk distance and increased VO2 peak
- Increased muscle strength
- increased muscle endurance
- increased forearm blood flow
- increased mitochondrial ATP production rate
- increased oxidative capacity
- relative increase in area of type I fibre type distribution

Type of Exercise

- Isometric exercise is not recommended for patients with CHF due to the pressure load that it places upon the heart. During this form of exercise, the increased intramuscular pressure compromises blood flow and oxygen delivery and thus energy requirements are met by anaerobic metabolism.
- During isometric exercise, systolic blood pressure increases to maintain perfusion and may have a negative influence upon central hemodynamics.
- When combined with a Valsalva maneuver, these hemodynamic changes become increasingly dangerous
- dynamic resistance training has been established as safe in this population.
- Isokinetic resistance training, which requires specific equipment, has been utilized with positive physiological benefits.

Resistance Training Recommendations for Patients with CHF

Parameter	NYHA class I	NYHA class II–III
Warm up and warm down	10 mins each	10 mins each
Intensity	50–60% 1RM	40–50% 1RM
Frequency	2–3 days/week	1–2 days/week
Duration of session	15–30 min	12–15 min
number of exercise stations	4–9	3–4
Number of sets per station	2–3	1–2
repetitions per set	6–15	4–10

Management of Heart Failure

Heart failure is usually a chronic condition that has taken years to develop and worsen. Treatment for heart failure is generally designed for three purposes:

Goals:

- To improve any symptoms
- To slow progression of the heart failure
- To prolong survival.

In general, management of heart failure should focus on the following:

- **Rest**:
  It should be physical and mental to decrease the cardiac load imposed on the heart. However, prolonged rest may lead to detrimental effects.

- **Diet:**
  It should be salt free, easily digestible, and should be taken in small amounts and at frequent intervals.

- **Medical treatment:** as

***Digitalis*:** Increasing force of myocardial contractility and decreased heart rate

Diuretic*s*: Stimulating kidney to loose excess sodium in urine.

- **Physical therapy**

Some degree of cognitive dysfunction is commonly associated with heart failure. Cognitive deficits are caused by cerebral hypoperfusion. These changes may affect patient physical activity and obeying the therapist orders during exercises.

Exercise prescription in heart failure

Exercise should always include adequate warm up and cool down periods. The warm up period should include 5 to 10 minutes of intermittent or continuous low intensity aerobic activity at approximately 25 to 50 % of the patient's functional capacity.

This phase can also include reducing the risk of musculoskeletal injuries, especially in a deconditioned population. After the warm up, exercise can be progressed to the goal intensity level. The cool down should be similar to the warm up. This will enhance venous return and decrease the risk of post exercise hypotension which may affect greatly patients with heart failure.

Patients with heart failure, who do not adequately cool down are also thought to be at increased risk of arrhythmias because of myocardial irritability from the large volume of blood returned to the heart when exercise stops suddenly.

N.B: Patients who develop exhaustion after training may need a day of rest between sessions. Supplemental walking should be encouraged on the nontraining days.

Walking exercise in heart failure

Walking can be performed by a wide range of patients with heart failure because its speed can be easily varied. Patients with a documented peak Vo2 of at least 13 ml / kg /min can generally

tolerate treadmill speeds as high as 3.7 mph. while less fit patients (peak Vo2 of 8 ml/kg /min) generally tolerating speeds of 1.9 mph or less.

Brisk walking could elicit an adequate training effect in patients with heart failure. While Jogging is generally not advised because even a slow jogging speed of 3 mph is only tolerated in patients with Vo2 of 20 ml/kg/min or greater which is not recommended in patients with heart failure.

The importance of interval training in heart failure:

Interval training consists of short bouts of exercise followed by periods of rest applied in a repeated sequence.

Interval training allows the muscles to work harder than they could with steady state exercise without excessive stresses on cardio vascular system.

Interval training with 30 seconds of exercise followed by 60 seconds of rest at an intensity of 75 % of peak Vo2 produced lower rate of pressure, lower rate of perceived exertion, and lower plasma catecholamine levels than steady state exercise at the same intensity.

Interval training may be performed using a stationary bicycle or a treadmill, and a variety of exercise / rest ratio may be used, including 15 seconds / 60 seconds, 10 seconds / 60 seconds, or 30 seconds /60 seconds for a total of 15 to 30 minutes.

Interval training may be gradually progressed in intensity by lengthening the exercise period or shortening the rest periods.

Exercise progression in heart failure

Progression of exercise will depend on the patient's baseline functional status, activity tolerance, vital sign response, and subjective complaints. Patients with a low

initial exercise capacity will generally make faster initial progress than those who start at a high functional capacity. Training can be considered as occurring in three stages. Progression from one stage

to another is based on the patient attaining specific intensity and duration goals. These three stages are outlined in the further table:

Stage	Intensity	Duration
Initial	Low level (From 40 % to 50 % of peak Vo 2)	10 : 15 min.
improvement	Primary goal to increase intensity (From 50 % to 70 %of peak Vo2)	15 : 30 min.
maintenance	Primary goal to maintain intensity (From 50 % to 70 % of peak Vo2)	30 : 35 min.

Resistance exercises in heart failure:

Resistance training reduces symptoms of heart failure when doing the normal activities of daily living and increases nitric oxide production, which relaxes arteries, and cause vasodilatory response.

A program of combined aerobic by cycling ergometer for 20 minutes and resistance training administered as a circuit weight training program can increase voluntary contractile strength and short term gains in left ventricular function in heart failure patients in addition to increasing

peak $\dot{V}O_2$ significantly. Also, an increase in strength and endurance is commonly associated with a lowering in O2 consumption at submaximal workloads.

Patients with heart failure performed resistance exercise consisting of three sets of 10 repetitions at 60 % of one repetition maximum, of leg extensions, leg curls, seated arm press, pull downs, rowing, and lateral arm abductions.

N.B: Isometric resistance exercise should be avoided in patients with heart failure, because it increases after load on the heart to a great degree.

Myocardial Infarction

Myocardial infarction (MI) is a necrotic portion of myocardium as a result of prolonged or permanent occlusion of its blood supply followed by inflammatory cell infiltration and eventual fibrous repair.

The classical cause of infarction is atheromatous plaque with thrombosis and vasospasm leading to complete occlusion of the lumen of coronary arteries.

The infarction occurs during activity increased metabolic demands on the heart. There are three levels of damage may occur after each other: ischemia, injury and infarct.

The ischemia is reversible, the injured area usually heals but normal structure and function may not return whereas the infracted area does not recover. During acute phase, the permeability of cardiac cell membrane to vital electrolytes is altered and the contractility of myocardium and cardiac output is depressed.

ST Segment elevation on electrocardiograph and raised levels of serum proteins released from disrupted myocardial cells are important indicators for myocardial infarction. Isoenzyme creatine kinase (CK) is raised from about 4 hours to 72 hours following infarction. Troponin T and I are found only in myocardial cells and considered as the gold standard diagnostic test for MI.

Clinical picture:

1. Severe chest pain at rest (which may be silent), lasts several hours with sudden shortness of breath.
2. Autonomic responses such as sweating, nausea and vomiting.
3. Pallor and an anxiety.

Types of myocardial infarction:

1. ***Uncomplicated****:*

It is happened as a small infarct without complication after full recovery in coronary unit for 2-3 days with increasing physical activity and education of patient and family in risk factor reduction. Research studies have found that patients without

complications during the first 4 days after infarction can be discharged from the hospital at 7 days without increasing their mortality during the next 6 months.

Uncomplicated patients therefore can be progressed more rapidly in phase I than the complicated patients. Those patients may be out of bed performing self-care activities and ward ambulation as early as the third day after the infarction.

2. ***Complicated****:*
Patient has one or combination of all four conditions as arrhysmia, heart failure, thrombosis and damage of heart structure. Complicated patients are not referred to cardiac rehabilitation to avoid catastrophic event (i.e., repeated infarction, cardiac arrest) that may be increased with physical activity. Those patients may be up as early as the fifth day after the infarction.

Submaximal exercise stress test with careful assessment may be conducted through physical therapist to provide the basis for an exercises program. Good monitoring should be performed through hemodynamic responses, perceived exertion recorded from Borg scale, and electrocardiogram recordings.

Physical Therapy post MI:

Physical Therapy program following MI is divided into four phases:

Acute phase

where patient is gradually progressed from bed rest in cardiac care unit to daily living activity with gradual increase in exercise intensity and mobilization in bed.

Convalescent phase:

This may last 6 weeks from the onset of infarction with an increase in endurance for exercise through walking or bicycling using heart rate.

Training phase:

This is an actual training program; it begins with a symptom limited exercise tolerance test. Maximum heart rate achieved in the test then limited end point such

as fatigue, musculoskeletal pain, angina, arrythmias, and achievement of target heart rate must be taken into consideration.

The following components may be used:

Mode of exercises:

Treadmill, upper or lower extremity ergometers, rowing machine, walking, and running.

Frequency*:*

Three sessions per week for 6- 8 weeks.

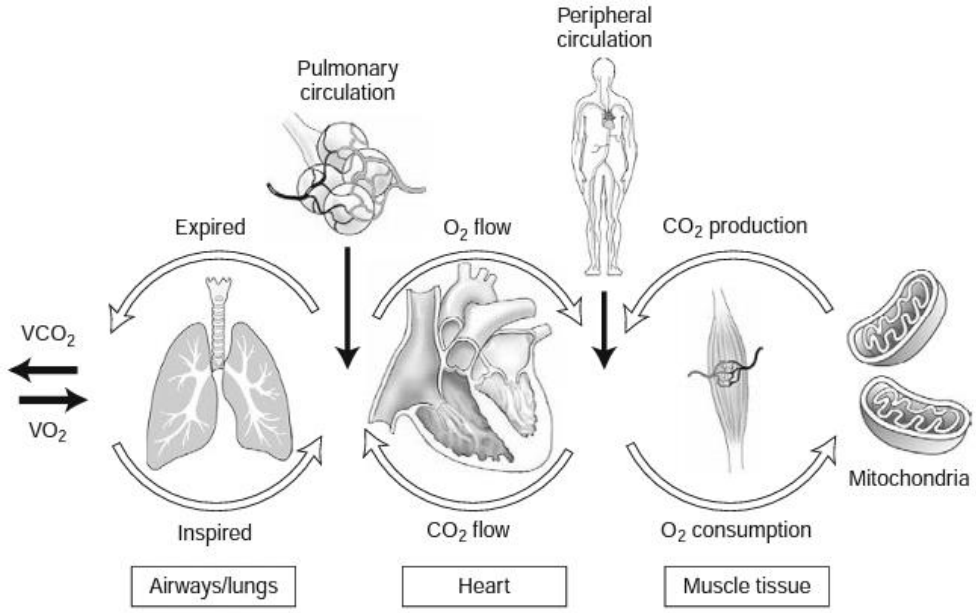
Intensity*:*

Each session composed of warm up training in the form of stretching followed by an aerobic program and ended by cool down period.

Maintenance phase:

This phase is used to ensure the benefits of training phase. The minimum requirement in this phase is exercise at least twice a week for at least 30 minutes and electrocardiogram monitoring is not necessary during this phase.

The oxygen transport system



Why the heart and lungs are important to physical therapists

The physical therapists are concerned with the prevention, diagnosis, and treatment of movement impairments and the enhancement of physical health and functional abilities. Diseases affecting the pulmonary and cardiovascular systems result in movement impairment because of their fundamental roles in the oxygen transport system, through which energy is provided for movement, as shown in figure above .When an individual wants to perform an activity, the central nervous system stimulates the appropriate muscles, and, if both systems are intact, the desired movements are produced.

However, for activity to continue for more than a few minutes without local discomfort or shortness of breath, the muscles must receive adequate blood supply carrying enough oxygen to produce the energy required to sustain the activity. Under normal circumstances this oxygen is readily available in the air that we breathe;

through the process of ventilation, it is inhaled through active contraction of the inspiratory muscles and flows through progressively smaller airways to the most distal units of the lungs, the alveoli.

The oxygen then diffuses from the alveoli into the surrounding pulmonary capillaries, which are perfused by blood flow coming from the right ventricle via the pulmonary arteries. Most of the oxygen is bound to hemoglobin, and the oxygen-rich blood returns to the left atrium via the pulmonary veins and is pumped by the left ventricle to all the tissues of the body, including the contracting muscles.

In the final steps of the oxygen transport system, the oxygen dissociates from arterial hemoglobin and diffuses across the capillary membrane into the muscle cells, where it enters the mitochondria to participate in the oxidative metabolic processes, which ultimately produce adenosine triphosphate, ATP, for energy.

Then the oxygen transport pathway proceeds in the reverse direction to eliminate metabolic by-products, particularly carbon dioxide, which diffuse from the muscle cells into the capillaries and are transported back to the heart via the systemic venous system. The right ventricle pumps the venous blood to the lungs, where carbon dioxide diffuses from the capillaries into the alveoli and, given adequate ventilation, is exhaled from the lungs.

Individuals with a number of lung pathologies, such as pneumonia, pulmonary edema, and pulmonary fibrosis, may have difficulty not only with delivering enough air to the alveoli but also with diffusion of oxygen from the alveoli into the bloodstream, particularly during activity.

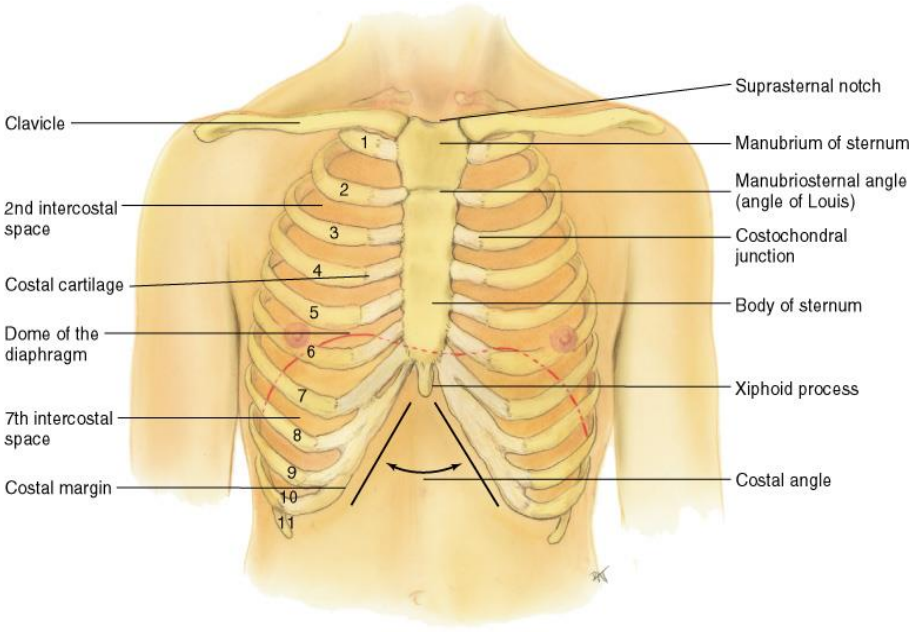
Thus, disorders of the pulmonary system ultimately increase the work of breathing and interfere with gas exchange, while abnormalities of the cardiovascular system limit the amount of blood that can be pumped and delivered to the skeletal muscles. The result is manifested as exercise intolerance, which has direct implications for physical therapy interventions. Because all of our clients depend on adequate cardiovascular and pulmonary function to participate effectively in rehabilitation activities, and diseases involving these systems are so prevalent in our society, assessment of the cardiovascular and pulmonary systems should be an essential component of every physical therapy evaluation.

Anatomy of the respiratory system

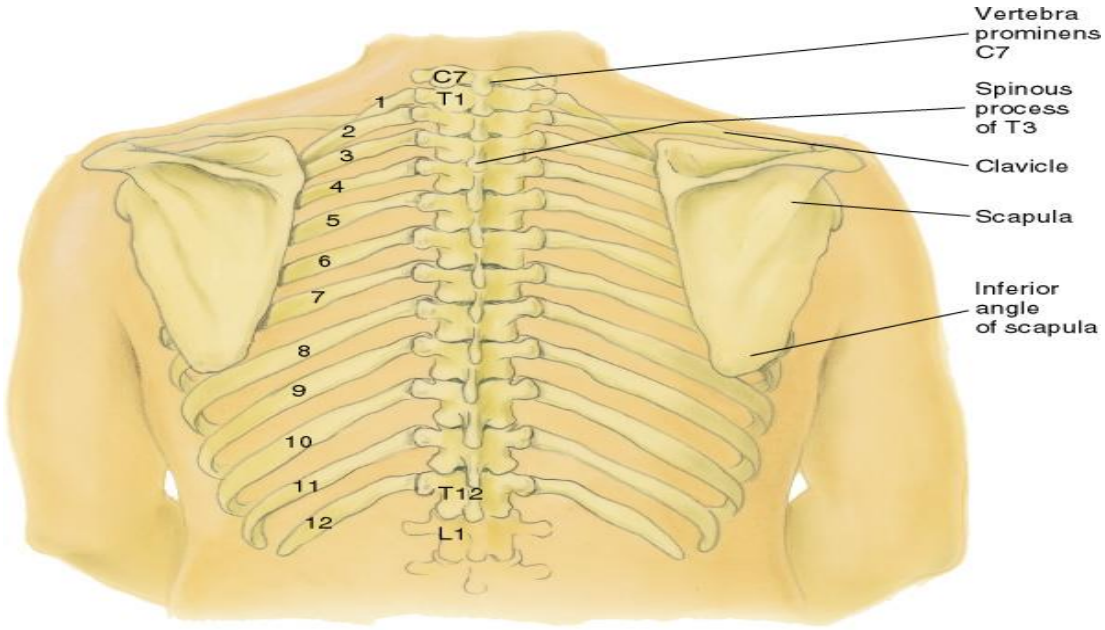
The thoracic cage consists of 12 thoracic vertebrae, 12 ribs, the sternum, and costal cartilage as presented in figure. The respiratory passages, depicted in figure 3, consist of the upper airways, including the nose, pharynx, and larynx; and the lower airways, referred to as the tracheobronchial tree, containing

(a) the non respiratory conducting airways, or the anatomic dead space (i.e., the trachea, bronchi, and bronchioles), that channels inspired air to the gas exchange areas,

(b) the respiratory units, or acini, where gas exchange takes place.



A) Anterior view

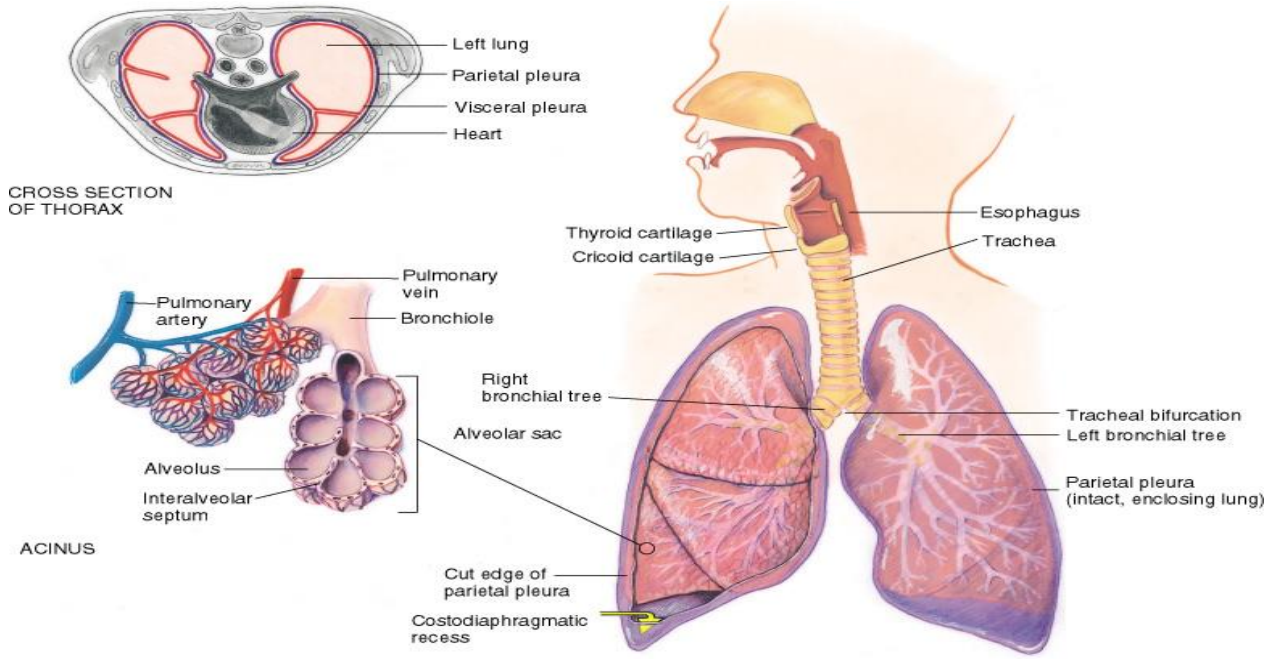


B) Posterior view

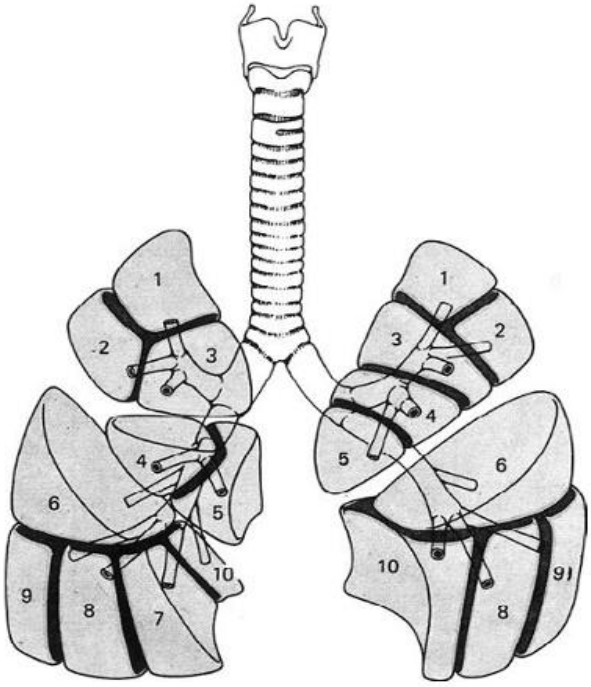
The two lungs with their various lobes and segments are illustrated in Figure 4. The major airways from the trachea through the 10 generations of bronchi have decreasing amounts of cartilaginous support surrounded by smooth muscle and elastic fibers; they have goblet cells for mucus production and are lined with ciliated columnar epithelium to facilitate secretion clearance.

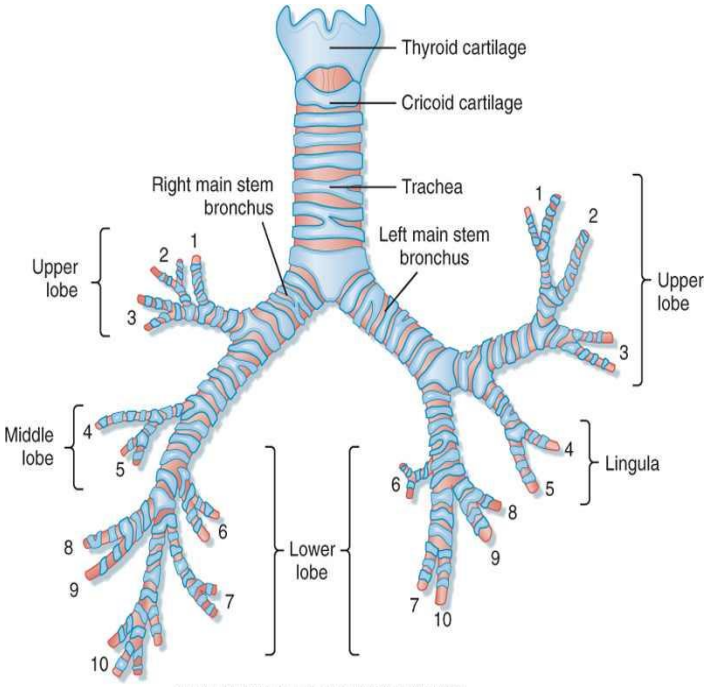
The five generations of bronchioles have no cartilage or goblet cells, but still have elastic tissue and smooth muscle fibers; they are lined with ciliated cuboidal epithelium.

The functional unit of the lungs is the acinus, which participates in gas exchange. It includes the respiratory bronchioles, alveolar ducts and sacs, and the alveoli, whose walls consist of a thin epithelial layer over a connective tissue sublayer.



The respiratory passages, including gas exchange at the alveolus and pulmonary capillary.

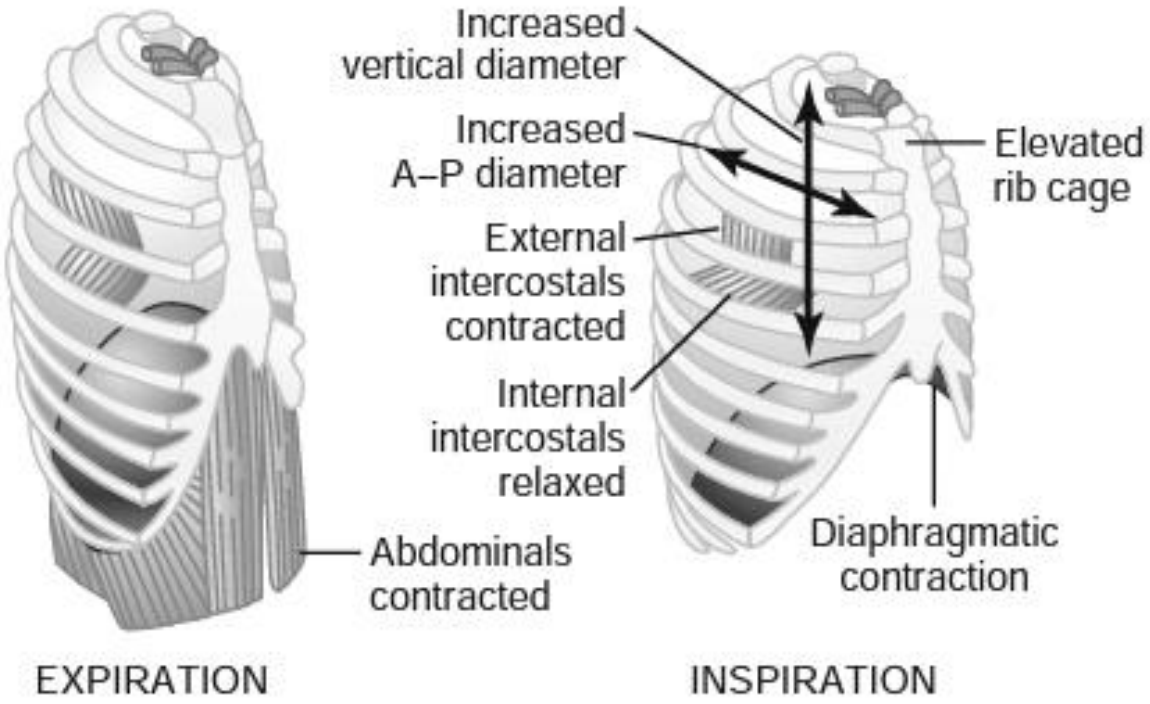




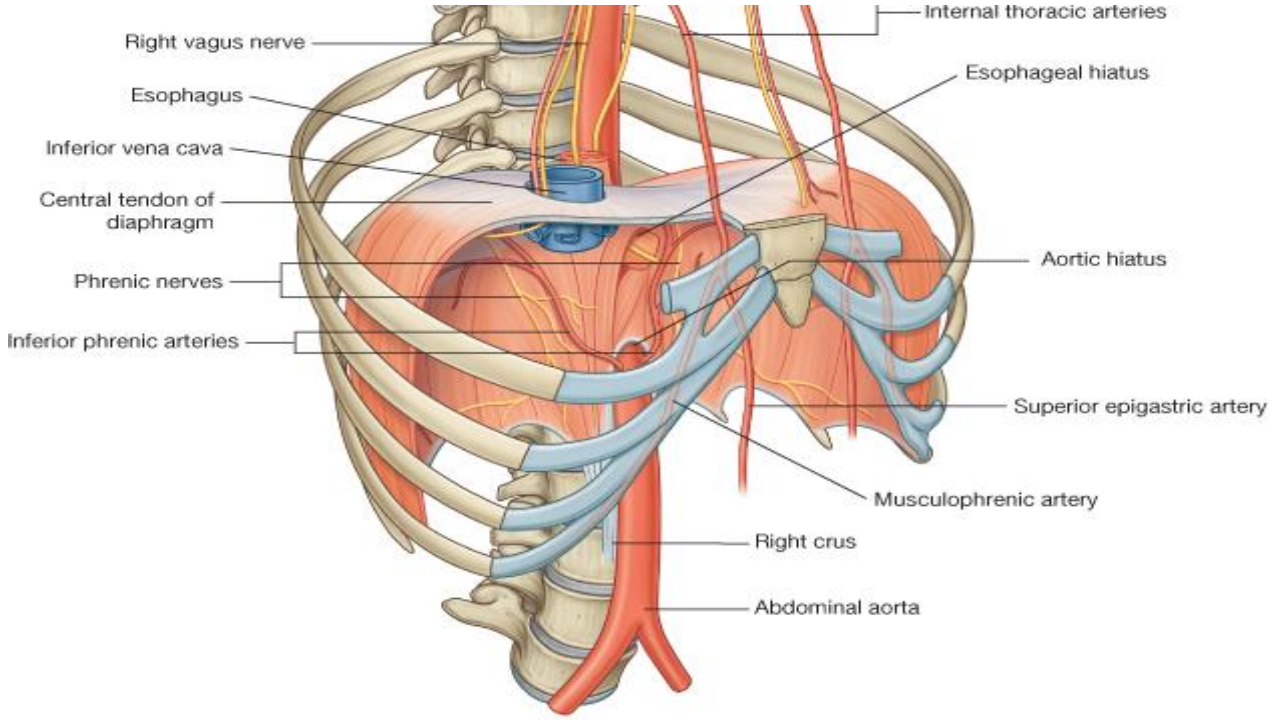
The bronchopulmonary segments. Left and right upper lobes: 1, apical; 2, posterior; and 3, anterior segments. Left upper lobe: 4, superior lingular and 5, inferior linguinal segments. Right middle lobe: 4, lateral and 5, medial segments. Lower lobes: 6, superior; 7, medial basal (no medial basal segment in the left lung); 8, anterior basal; 9, lateral basal; and 10, posterior basal segments.

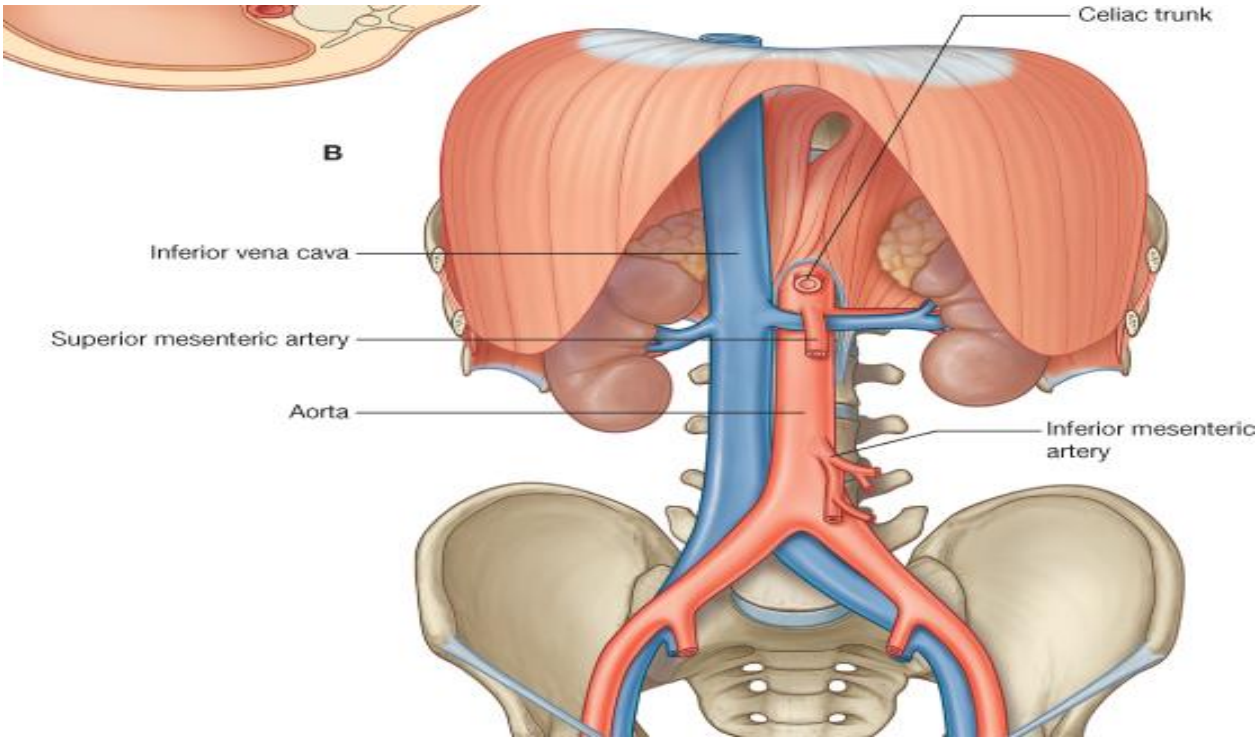
Muscles of Respiration

The respiratory muscles, their innervations, and their functions are listed in Table 1. The primary muscles of inspiration are the diaphragm, external intercostal muscles, and parasternal intercostals.



Contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating contraction of the abdominals and depression of the rib cage that take place during active expiration and diaphragmatic and external intercostal muscle contraction (with relaxation of the internal intercostals), elevation of the rib cage, and increased vertical diameter that occur during inspiration. Not shown are the parasternal intercostals (which form the ventral part of the internal intercostal layer from the sternum and between the costal cartilages) and the scalenes, both of which also contract during normal quiet inspiration to produce elevation and expansion of the upper rib cage.


Internal thoracic arteries
Right vagus nerve
Esophageal hiatus
Esophagus
Inferior vena cava
Central tendon of diaphragm
Aortic hiatus
Phrenic nerves
Inferior phrenic arteries
Superior epigastric artery
Musculophrenic artery
Right crus
Abdominal aorta



The diaphragm

During deep or labored breathing, the accessory muscles of inspiration are recruited. At rest, expiration is a passive process, occurring as the inspiratory muscles relax and lung elastic recoil takes over. During forced expiration and coughing, the abdominal and internal intercostal muscles are activated. Respiratory muscle weakness and limited endurance can impair gas exchange and lead to respiratory insufficiency or failure, especially when the mechanics of breathing are altered by hyperinflation of the chest (e.g., emphysema, chronic bronchitis, and acute asthma attack).

Table 1: The respiratory muscles

Muscle (Innervation)	Functions
Primary Inspiratory Muscles	
Diaphragm (C_{3-5})	Expands thorax vertically and horizontally; essential for normal vital capacity and effective cough
Scalenes (C_{2-7})	When neck is fixed, elevate first two ribs to expand chest superiorly
Parasternal intercostals (T_{1-11})	Elevate ribs to expand upper half of rib cage
External intercostals (T_{1-11})	Anterior and lateral expansion of upper and lower chest
Accessory Inspiratory Muscles	
Sternocleidomastoid (cranial nerve XI and C_{2-3})	When head is fixed, elevates sternum to expand chest superiorly and anteriorly
Serratus anterior (C_{5-7})	When scapulae are fixed, elevates first eight or nine ribs to provide posterior expansion of thorax
Pectoralis major (C_5-T_1)	When arms are fixed, elevates true ribs to expand the chest anteriorly
Pectoralis minor (C_{6-8})	When scapulae are fixed, elevates third, fourth, and fifth ribs to expand the chest laterally
Trapezius (cranial nerve XI and C_{3-4})	Stabilizes scapulae to assist the serratus anterior and pectoralis minor in elevating the ribs
Erector spinae (C_1 down)	Extend the vertebral column to allow further rib elevation
Expiratory Muscles	
Abdominals (T_{7-12} + L_1 for some)	Help force diaphragm back to resting position and depress and compress lower thorax leading to ↑ intrathoracic pressure, which is essential for effective cough
Internal intercostals (T_{1-11})	Depress third, fourth, and fifth ribs to aid in forceful expiration

Nervous Control

The lungs and airways are innervated by the pulmonary plexus (located at the root of each lung), which is formed from branches of the sympathetic trunk and vagus nerve. Sympathetic nervous system stimulation results in bronchodilation and slight

vasoconstriction, whereas parasympathetic nervous system stimulation causes bronchoconstriction and indirect vasodilation.

The function of the lungs is controlled through complex interactions of specialized peripheral and central chemoreceptors, as well as the respiratory center with groups of neurons located in the medulla oblongata and pons, as illustrated in the figure.

The medulla oblongata is the primary respiratory control center. Its main function is to send signals to the muscles that control respiration to cause breathing to occur.

- There are two regions in the medulla that control respiration:
    - The dorsal respiratory group (DRG) stimulates inspiratory movements.
    - The ventral respiratory group (VRG) stimulates expiratory movements.

• The respiratory center in the medulla contain **central chemoreceptors** that respond to changes in carbon dioxide levels (PCO_2) and hydrogen ion (H^+) concentration, while other areas receive input from the peripheral chemoreceptors, baroreceptors, and several types of receptors in the lungs. They control inspiration and respiratory rhythm both at rest and during exercise.

• **The pneumotaxic center** in the pons limits the duration of inspiration and increases the respiratory rate.

• **The apneustic center** in the pons sends signals for inspiration for long and deep breaths.

• Peripheral receptors provide input to the respiratory center:

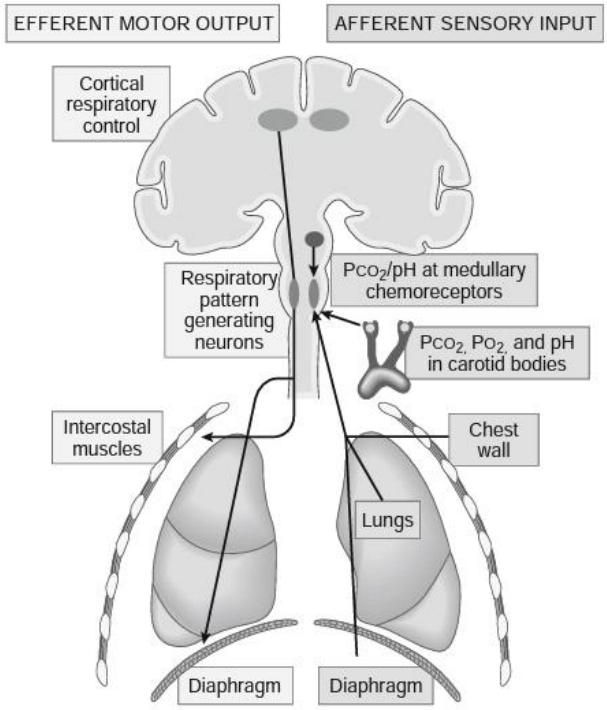
- **Stretch receptors** in the lungs act to prevent over inflation.

- **Peripheral chemoreceptors** located in the carotid and aortic bodies respond to hypoxemia ($\downarrow PO_2$) and, to a lesser extent, to rising PCO_2 and Hb concentration.
- **Proprioceptors** in the joints and muscles excite the respiratory centers in the medulla to increase ventilation.

• Higher centers in the motor cortex are responsible for voluntary control of breathing (e.g., voluntary breath holding or hyperventilation) and often stimulate respiration in anticipation of exercise.

• It is now recognized that the distribution of neural drive is a major determinant of which regions of the respiratory muscles are selectively activated and in what manner under various resting and exercise conditions, and thus of the actions they produce.

• Disturbances in the control of breathing will result in abnormal blood gas values.



A simplified diagram of respiratory integration and control, showing the principal efferent (left) and afferent (right) pathways. The respiratory areas, as well as the central nervous system links to them, are shown using a section through the brain, brainstem, and spinal cord.

Blood Supply to the lungs

The bronchial arteries arising from the descending aorta provide blood supply to the non respiratory airways, pleurae, and connective tissue, while the pulmonary arteries supply the respiratory units (acini) and participate in gas exchange. Numerous

pulmonary vasoactive substances can induce vasoconstriction or vasodilation of the pulmonary arterioles.

Defense of the lungs

The lungs have a number of structures that serve to protect the lungs from inhaled organisms and particles, and they are assisted by several different types of cells that reside within the lungs.

• Nasal mucosa and hairs warm and humidify inhaled air and filter out particles.

• Goblet cells and bronchial mucous glands produce mucus, which contains immunoglobulin A, to protect underlying tissue and trap organisms and particles. Mucus production is increased by inflammation (e.g., asthma and bronchitis) and its composition may be altered by various diseases (e.g., asthma and cystic fibrosis).

• Cilia are hair like structures that wave mucus up to the carina and throat (mucociliary transport). Mucociliary transport is impaired by inhalation of toxic gases (e.g., cigarette smoke and air pollution), acute inflammation, infection, and other disease processes.

• Type II pneumocytes produce surfactant, which protects underlying tissue and repairs damaged alveolar epithelium. Type I pneumocytes Provide a very thin membrane for gas exchange.

• Alveolar macrophages roam the surface of the terminal airways and engulf foreign matter and bacteria. They also kill bacteria in situ by means of lysozymes. Their activity can be impeded by cigarette smoke, air pollution, alveolar hypoxia, radiation, corticosteroid therapy, and the ingestion of alcohol.

• B lymphocytes produce gamma globulin for the production of antibodies to combat lung infections, and T lymphocytes release a substance that attracts macrophages to the site of an infection.

• Polymorphonuclear leukocytes engulf and kill blood-borne gram-negative organisms.

• Mast cells, which are more numerous in distal airways, release mediators of the inflammatory response to alter epithelial and vascular permeability. Smokers and persons with asthma have greater numbers of mast cells.

Respiratory physiology

It is important for physical therapists to understand the factors that contribute to normal functioning of the respiratory system in order to appreciate normal versus abnormal physiological indicators, both at rest and during exercise, as well as the implications for physical therapy interventions.

Basic functions of the respiratory system

The basic functions of the respiratory system include:

- Oxygenation of the blood
- Removal of carbon dioxide
- Control of acid–base balance
- Production of vocalization.

Mechanics of breathing

Respiratory gas exchange requires the movement of sufficient volumes of air into the terminal airways to meet the oxygen needs of the body, whether at rest or during exercise. This occurs through active contraction of the inspiratory muscles with enough force to override the elastic recoil of the lungs and the resistance to airflow offered by the airways. Thus, the respiratory cycle consists of:

• Inspiration, during which active muscle contraction results in expansion of the thorax and the lungs, a fall in alveolar pressure, and airflow into the lungs. At rest, inspiration is accomplished primarily by the diaphragm with some assistance from

the parasternal and external intercostals and scalenes (the parasternal intercostals and scalenes act to lift the ribs and expand the upper half of the rib cage, which is important to counteract the inward motion of the upper chest that would result from an unopposed decrease in intrapleural pressure produced by diaphragmatic descent). During exercise the accessory muscles of inspiration are recruited to increase tidal volume (see Table 2), which is assisted by passive relaxation of the expiratory muscles that are also activated. The drop in intrathoracic pressure during inspiration also facilitates venous return to the heart.

• Expiration, during which passive relaxation of the inspiratory muscles to their resting positions and elastic recoil of the lungs cause alveolar pressure to rise, resulting in airflow out of the lungs. During exertion, forced expiration, and coughing, active contraction of the

expiratory muscles (plus closure of the glottis during coughing) causes a marked rise in intrathoracic pressure so that expiration occurs more rapidly and completely; in addition, passive relaxation of these muscles at end-expiration promotes descent of the diaphragm and induces an increase in lung volume toward its neutral resting position.

A number of factors determine respiratory function and are described as the following:

• **Ventilation (V):** The process by which air moves into and out of the lungs.

• **Airway resistance (R_{aw}):** The resistance to airflow through the airways; increased airway resistance can limit airflow, which is most noticeable during expiration when the airways are narrower.

• **Pulmonary compliance (C):** The ease with which the lungs expand during inspiration; normal lungs are very compliant and easily expand during inspiration, according to the specific compliances of both the lungs and the chest wall and their elastic properties, as well as the adequacy of thoracic pump function.

• **Diffusion:** The movement of gases into and out of the blood; because CO2 is more readily diffusible than oxygen, diffusion abnormalities will result in hypoxemia long before hypercapnia develops.

• **Perfusion (Q):** The blood flow through the pulmonary circulation that is available for gas exchange; hypoxic vasoconstriction is stimulated to reduce blood flow to alveoli that are not being ventilated (i.e., alveolar dead space).

• **Ventilation–perfusion (V/Q) matching:** The degree of physical correspondence between ventilated and perfused areas of the lungs; the optimal V/Q ratio is 0.8 (4 parts ventilation to 5 parts perfusion) to maintain normal gas exchange.

• **Oxygen–hemoglobin (O_2–Hb) binding:** The level of oxygen saturation of the arterial blood, normal arterial oxygen saturation (SaO_2) is 95% or more.

Lung Volumes and Capacities

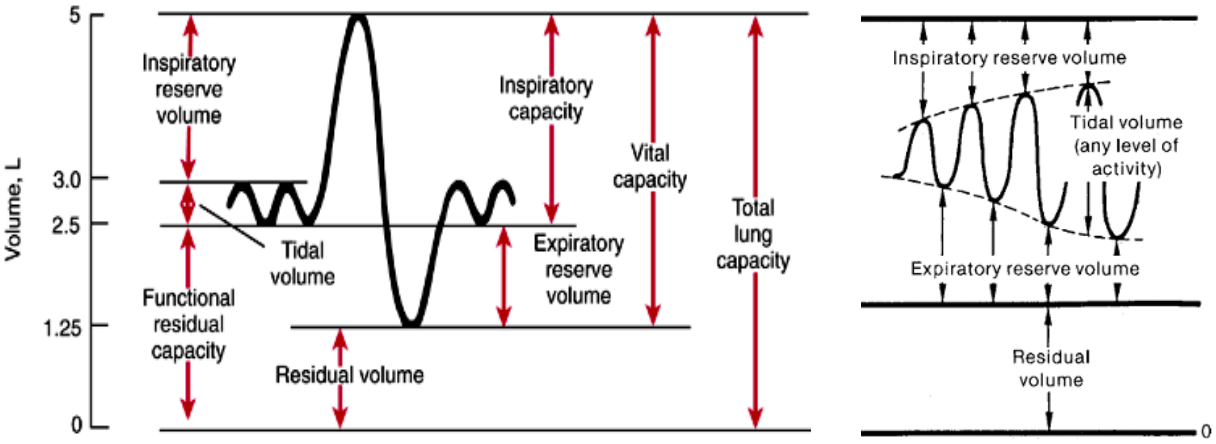
As illustrated in the figure, it is possible to determine various lung volumes and capacities, which are described in table 2.

• Tidal volume (V_T) represents the most efficient breathing pattern and volume and includes both dead space and alveolar volumes.

• Functional residual capacity (FRC) reflects the balance of the elastic forces exerted by the chest wall and the lungs and is the neutral resting volume of the respiratory system after a normal expiration.

• At total lung capacity (TLC), the elastic forces of the lungs are balanced by the maximal inspiratory muscle forces during a very deep breath.

• At residual volume (RV), the elastic forces of the chest wall are balanced by the maximal expiratory muscle forces at the end of a forced expiration.



A spirogram showing the lung volumes and capacities during normal breathing and during maximal inspiration and expiration. To the right are the changes in primary lung volumes that occur during progressively more intense activity.

Table 2: Lung Volumes and Capacities

Volume or Capacity	Description
Tidal volume (V_T)	Amount of air inspired or expired during normal breathing
Inspiratory reserve volume (IRV)	Extra volume inspired over and above the tidal volume
Expiratory reserve volume (ERV)	Extra amount of air forcefully expired after the end of a normal tidal expiration

Residual volume (RV)	Volume of air still remaining in the lungs (i.e., in the anatomic dead space and the acini) after a maximal forced expiration
Inspiratory capacity (IC)	Maximal volume of air that can be inspired after a normal tidal expiration = TV + IRV
Functional residual capacity (FRC)	Amount of air remaining in the lungs after a normal tidal expiration = RV + ERV
Vital capacity (VC)	Maximal volume of air that a person can forcefully expire after taking in a maximal inspiration = IRV +TV + ERV
Total lung capacity (TLC)	Maximal volume of air that the lungs can contain following a maximal inspiration = RV + ERV + TV + IRV

Exercise Physiology

During exercise the respiratory system must increase the volume of air (oxygen) that is ventilated by the lungs (i.e., minute ventilation, V, which is usually measured during expiration and thus referred to as (V_E) and diffused into the blood for delivery to the exercising muscles. There are a number of factors that affect respiratory function and the ability of the respiratory system to meet the oxygen demands of the exercising muscles during vigorous exertion.

• At rest, V_E is usually 5 to 10 L/min and it often increases 15 to 20-fold during maximal exercise.

• At the onset of mild to moderate exercise, V_E typically increases via increasing tidal volume.
• During more strenuous exercise, rising respiratory rates further augment V_E.
• At rest, the energy cost of breathing is 1% to 4% of total body V_{O2}, and at maximal exercise it increases to 8% to 11% of total V_{O2} in healthy individuals and as high as 40% in those with severe pulmonary disease.
• Ventilation is not normally a limiting factor to aerobic capacity.

Effects of Aging

Many of the changes that were once attributed to the normal aging process are now known to be effects of deconditioning and can be retarded or reversed with exercise conditioning. However, there are a number of physical and functional changes that do occur with normal aging that affect pulmonary function and increase the work of breathing.

• The thoracic cage becomes less compliant due to increased stiffness of the costovertebral joints and chest wall, so thoracic expansion is reduced.
• A decrease in the number and thickness of elastic fibers impairs elastic recoil during expiration, which increases RV and FRC.
• The diaphragm assumes a lower and less mechanically efficient position in the chest, causing a reduction in its force-generating ability.
• Airway resistance increases, so expiratory time is prolonged.
• Diffusing capacity decreases as a result of reductions in alveolar surface area (the alveoli and alveolar ducts become enlarged) and the number of pulmonary capillaries, which reduces the efficiency of gas exchange.
• Recruitment of the accessory muscles of respiration occurs more often and at lower levels of exertion, leading to an increase in the work of breathing.
• The peripheral and central chemoreceptors become less sensitive to increasing levels of carbon dioxide and hypoxemia, so they are less likely to stimulate ventilation to compensate for abnormalities.

Pulmonary function tests

Pulmonary function tests (PFTs) consist of a series of inspiratory and expiratory maneuvers designed to assess the integrity and function of the respiratory system. The information provided by PFTs is helpful to the therapist in establishing realistic treatment goals and an appropriate treatment plan according to the patient's current pulmonary problems and degree of impairment.

PFTs include measurements of lung volume and capacity and ventilation.

Lung volumes and capacities

• The various lung volumes and capacities.

• Normal values vary depending on age, gender, height, and ethnicity.

• Total lung capacity is not measured directly during PFTs but is extrapolated from other measurements. It may be decreased in disease processes with space-occupying lesions (such as edema, atelectasis, tumors, and fibrosis) and in pleural effusion, pneumothorax, and thoracic deformity. It may be normal or increased in obstructive lung diseases, being elevated in hyperinflation.

• Vital capacity (VC) is reduced if there is a loss of distensible lung tissue (such as in atelectasis, pneumonia, pulmonary fibrosis, pulmonary congestion or edema, bronchial obstruction, carcinoma, and surgical excision) or impairment of thoracic pump function. It is also affected by patient effort and motivation.

• Residual volume (RV) and functional residual capacity (FRC) are reduced in restrictive lung dysfunction, when there is interference with either lung or thoracic expansion. They are increased (>120% of predicted normal) in chronic obstructive lung disease, indicating air trapping.

Ventilation

The parameters that describe ventilatory function include the following:

• Tidal volume (V_T)

- Values should always be assessed within the context of respiratory rate and minute ventilation.
- Values of 400 to 700 mL are typical, although there is considerable variation.
- Values may be decreased in severe restrictive lung dysfunction and respiratory center depression, in which case a greater proportion of the volume serves as dead space and less volume reaches the acini for participation in gas exchange; values are increased during exertion and at rest in some patients with pulmonary disease.

• Respiratory rate (RR)

- Normally, RR =12 to 20 breaths/min in adults. Values are increased with exertion, hypoxia, hypercapnia, acidosis, increased dead space volume, and decreased lung compliance; they are often decreased in central nervous system depression and carbon dioxide narcosis.
- RR is often considered to be a good indicator of the stimulus to breathe and of normal versus abnormal ventilatory status.

• Minute ventilation, expired (V_E)

- $V_E = V_T$ x RR and is usually between 5 and 10 L/min. V_E will be increased (>20 L/min) in hypoxia, hypercapnia, acidosis, increased dead space volume, anxiety, and exercise and will be decreased in hypocapnia, alkalemia, respiratory center depression, and neuromuscular disorders with ventilatory muscle involvement.
- V_E is the primary index of ventilation when used in conjunction with arterial blood gases.

- Hypoventilation is defined as inadequate ventilation to eliminate normal levels of carbon dioxide (CO_2), resulting in hypercapnia and respiratory acidosis.
- Hyperventilation is ventilation in excess of that needed to maintain adequate CO_2 removal, and produces hypocapnia and respiratory alkalosis.

• Dead space (V_D)

- V_D is the volume of lungs that is ventilated but not perfused by pulmonary capillary blood flow, and is usually 125 to 175 mL.
- V_D can be divided into the volume in the non respiratory conducting airways (the anatomic dead space), and that in the non perfused alveoli (the alveolar dead space). Anatomic dead space is increased in larger individuals and in bronchiectasis and emphysema and is decreased in asthma, bronchial obstruction, and mucous plugging. V_D is increased during normal exercise and in pulmonary embolism and pulmonary hypertension.

• Ratio of dead space to tidal volume (V_D/V_T)

- Normally, the derived value is 0.2 to 0.4.
- V_D/V_T decreases in normal individuals during exercise because of increased cardiac output and enhanced perfusion of the alveoli at the lung apices (despite an absolute increase in V_D) and increases in pulmonary embolism and pulmonary hypertension. The failure of V_D/V_T to decrease during exercise may be an early sign of pulmonary vascular disease.

• Alveolar ventilation (V_A)

- V_A is the volume of air that participates in gas exchange. $V_A = (V_T - V_D)$ and is usually about 4 to 5 L at rest, with large variations in healthy individuals. Decreased V_A can result from absolute increases in dead space, as well as decreases in V_E.
- V_A is one of the major factors determining gas exchange, the adequacy of which can be measured only through determination of arterial blood gases.

Arterial blood gases

Arterial blood is often analyzed to obtain information related to a patient's oxygenation, ventilatory, and acid–base status. Values can be obtained by analysis of an arterial blood sample or by direct-reading electrodes after arterial cannulation.

• The data obtained from arterial blood gases (ABGs) and normal values are listed in table 3.

• Sudden acute changes in the levels of $PaCO_2$ and pH are more dangerous than gradual chronic changes and tend to be associated with more serious clinical manifestations.

• It is important for physical therapists to appreciate normal versus abnormal values for ABGs and how abnormalities affect an individual's ability to perform exercise and rehabilitation activities.

• Patients with chronic pulmonary disease and low oxygen saturations often benefit from the use of supplemental oxygen, especially during exertion.

Table 3: Parameters measured in arterial blood gas analysis

Partial pressure of oxygen (PaO_2)	97 mm Hg (80-100)
Partial pressure of carbon dioxide ($PaCO_2$)	40 mm Hg (35-45)
Hydrogen ion concentration (pH)	7.40 (7.35-7.45)

Arterial oxygen saturation (SaO_2)	>95%
Bicarbonate level (HCO_3^-)	24 mmol/L (22–26)
Base excess/deficit (BE)	0 (-2 to +2)

PaO_2: partial pressure of oxygen in arterial blood, i.e. oxygen dissolved in plasma.
SaO_2: extent to which haemoglobin in arterial blood is saturated with oxygen, i.e. capacity of blood to carry oxygen.
Oxygen content: total amount of oxygen in blood, i.e. oxygen in both plasma and haemoglobin.
$PaC0_2$: partial pressure of CO_2 in arterial blood.
Hypoxaemia: reduced oxygen in arterial blood.

$Pa0_2$ < (60 mmHg) or Sa O_2 <90%.
Hypoxia: reduced oxygen at tissue level.
Hypocapnia/hypocarbia: reduced CO_2 in arterial blood.
Hypercapnia/hypercarhia: increased CO_2 in arterial blood.

Acid-Base Regulation

The proper balance of acids and bases in the body is essential to life. This balance is very complex and must be kept within the narrow parameters of a pH of 7.35 to 7.45 in the extracellular fluid. This number (or pH value) represents the hydrogen ion concentration in body fluid.

A reading of less than 7.35 is considered acidosis, and a reading greater than 7.45 is called alkalosis. Life cannot be sustained if the pH values are less than 7 or greater than 7.8.

Living human cells are extremely sensitive to alterations in body fluid pH (hydrogen ion concentration); thus various mechanisms are in operation to keep the pH at a relatively constant level.

Acid-base regulatory mechanisms include chemical buffer systems, the respiratory system, and the renal system. These systems interact very closely to maintain a normal acid-base ratio of 20 parts of bicarbonate to 1 part of carbonic acid and thus to maintain normal body fluid pH.

The blood test used most often to measure the effectiveness of ventilation and oxygen transport is the arterial blood gas test (table 3). The measurement of arterial blood gases is important in the diagnosis and treatment of ventilation, oxygen transport, and acid-base problems.

The ABG test measures the amount of dissolved oxygen and carbon dioxide in arterial blood and indicates acid-base status by measurement of the arterial blood pH. In simple terms a low pH reflects increased acid buildup, and a high pH reflects an increased base buildup.

Acid buildup occurs when there is an ineffective removal of carbon dioxide from the lungs or when there is excess acid production from the tissues of the body. These problems are corrected by adjusting the ventilation or buffering the acid with bicarbonate.

Respiratory Acidosis

Any condition that decreases pulmonary ventilation increases the retention and concentration of carbon dioxide (CO_2), hydrogen, and carbonic acid; this results in an increase in the amount of circulating hydrogen and is called respiratory acidosis. If ventilation is severely compromised, CO_2 levels become extremely high and respiration is depressed even further, causing hypoxia as well.

During respiratory acidosis, potassium moves out of cells into the extracellular fluid to exchange with circulating hydrogen. This results in hyperkalemia (abnormally high potassium concentration in the blood) and cardiac changes that can cause cardiac arrest.

Respiratory acidosis can result from pathologic conditions that decrease the efficiency of the respiratory system. These pathologies can include damage to the medulla, which controls respiration, obstruction of airways (e.g., neoplasm, foreign bodies, pulmonary disease such as COPD, pneumonia), loss of lung surface ventilation (e.g., pneumothorax, pulmonary fibrosis), weakness of respiratory muscles (e.g., poliomyelitis, spinal cord injury, Guillain-Barré syndrome), or overdose of respiratory depressant drugs.

As hypoxia becomes more severe, diaphoresis, shallow rapid breathing, restlessness, and cyanosis may appear. Cardiac arrhythmias may also be present as the potassium level in the blood serum rises.

Treatment is directed at restoration of efficient ventilation. If the respiratory depression and acidosis are severe, injection of intravenous sodium bicarbonate and use of a mechanical ventilator may be necessary. Any client with symptoms of inadequate ventilation or CO_2 retention needs immediate medical referral.

Clinical signs and symptoms of respiratory acidosis

- Decreased ventilation
- Confusion
- Sleepiness and unconsciousness
- Diaphoresis

- Shallow, rapid breathing
- Restlessness
- Cyanosis

Respiratory alkalosis

Increased respiratory rate and depth decrease the amount of available CO_2 and hydrogen and create a condition of increased pH, or alkalosis. When pulmonary ventilation is increased, CO_2 and hydrogen are eliminated from the body too quickly and are not available to buffer the increasingly alkaline environment.

Respiratory alkalosis is usually due to hyperventilation. Rapid, deep respirations are often caused by neurogenic or psychogenic problems, including anxiety, pain, and cerebral trauma or lesions. Other causes can be related to conditions that greatly increase metabolism (e.g., hyperthyroidism) or overventilation of clients who are using a mechanical ventilator.

If the alkalosis becomes more severe, muscular tetany and convulsions can occur. Cardiac arrhythmias caused by serum potassium loss through the kidneys may also occur. The kidneys keep hydrogen in exchange for potassium.

Treatment of respiratory alkalosis includes reassurance, assistance in slowing breathing and facilitating relaxation, sedation, pain control, CO_2 administration, and use of a rebreathing device such as a rebreathing mask or paper bag. A rebreathing device allows the client to inhale and "rebreathe" the exhaled CO_2.

Respiratory alkalosis related to hyperventilation is a relatively common condition and might be present more often in the physical therapy setting than respiratory acidosis. Pain and anxiety are common causes of hyperventilation, and treatment needs to be focused toward reduction of both of these interrelated elements. If hyperventilation continues in the absence of pain or anxiety, serious systemic problems may be the cause, and immediate physician referral is necessary.

If either respiratory acidosis or alkalosis persists for hours to days in a chronic and not life-threatening manner, the kidneys then begin to assist in the restoration of normal body fluid pH by selective excretion or retention of hydrogen ions or bicarbonate. This process is called renal compensation. When the kidneys compensate effectively, blood pH values are within normal

limits (7.35 to 7.45) even though the underlying problem may still cause the respiratory imbalance.

Clinical signs and symptoms of respiratory alkalosis

- Hyperventilation
- Lightheadedness
- Dizziness
- Numbness and tingling of the face, fingers, and toes
- Syncope (fainting)

Chronic obstructive pulmonary diseases

Chronic obstructive lung diseases (COPD) include, chronic bronchitis, emphysema, asthma, bronchiectasis, and cystic fibrosis, among others, which are characterized by airflow limitation that is particularly noticeable during forced expiration. Airflow limitation can result from impediments within the airways (e.g., excessive secretions, edema fluid, or foreign material), airway narrowing (e.g., bronchoconstriction, mucous gland hypertrophy, or inflammation), or peribronchial abnormalities (e.g., destruction of lung parenchyma, as in emphysema, or compression by enlarged lymph nodes or tumor).

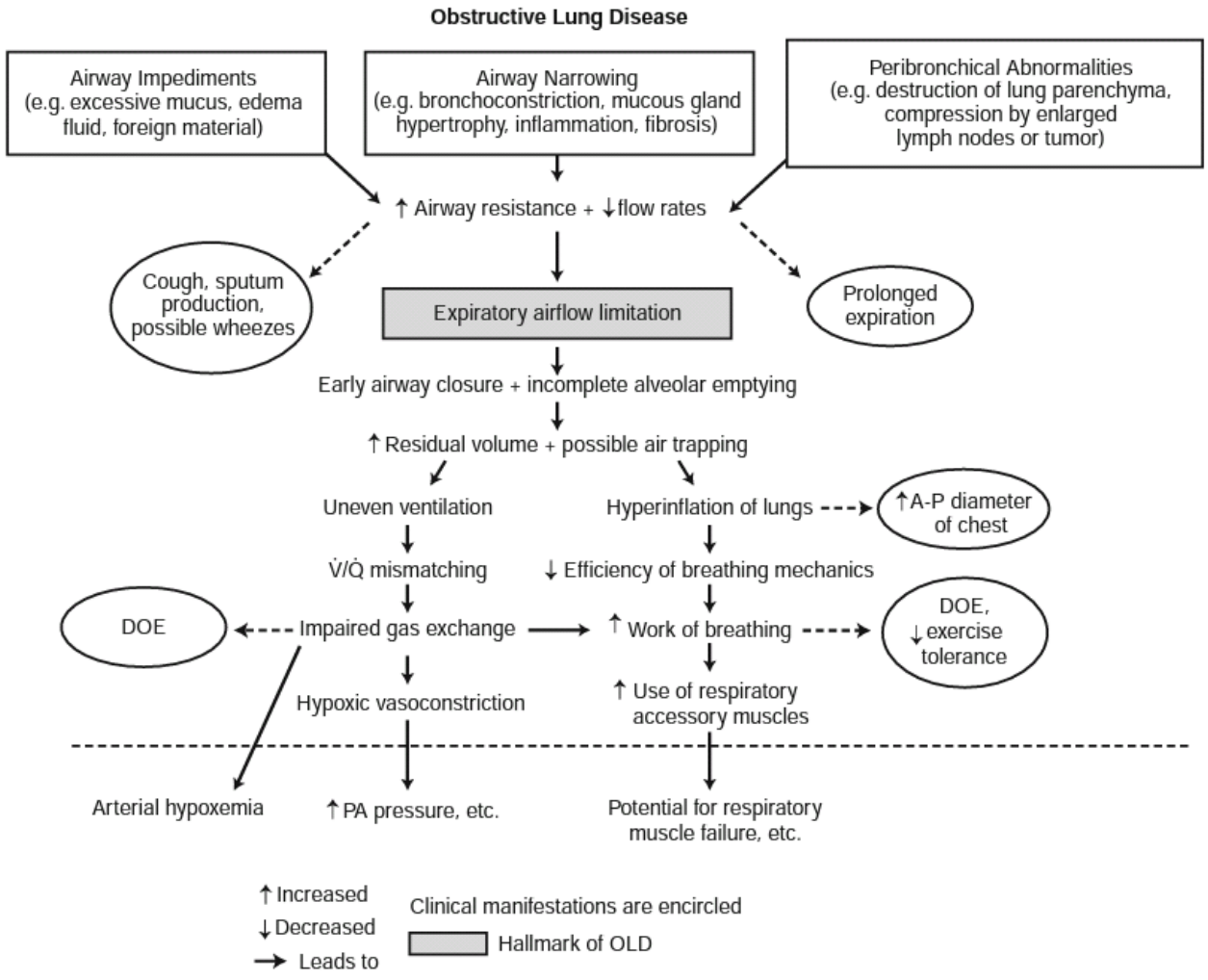
Because there is a great deal of overlap in the clinical and pathophysiological features of asthma, chronic bronchitis, and emphysema and many patients have features of more than one of these diseases, the generic diagnosis of COPD is frequently used (sometimes called chronic obstructive lung disease or chronic obstructive airway disease [COLD or COAD, respectively]). Common to all obstructive pathologies is chronic inflammation of the airways and parenchymal and vascular destruction, which occur in highly variable combinations. In most cases, COPD is slowly progressive and only partially reversible with treatment.

Smoking is the principal cause of COPD, with many of its chemical substances inducing proinflammatory, cytotoxic, and carcinogenic effects. In addition, inhibition of ciliary function is associated with recurrent episodes of bronchitis and lower respiratory tract infections. However, only a small minority of smokers actually develop COPD; more commonly they experience serious cardiovascular, cerebrovascular, and peripheral vascular diseases.

A general description of COPD is presented here, followed by the specific characteristics of the more common types of COPD.

Pathophysiology

The general pathophysiological effects of COPD are illustrated.



General pathophysiology of obstructive lung diseases leading to increased ventilation–perfusion mismatching (V/Q) and increased work of breathing; the resultant clinical manifestations are encircled. A-P, antero-posterior; DOE, dyspnea on exertion; V/Q, ventilation - perfusion.

• Chronic inflammation of the airways leads to mucosal edema, increased mucus production, ciliary dysfunction, and sometimes bronchoconstriction, all of which increase airway resistance, resulting in expiratory flow limitation. Initially, the disease is asymptomatic, but

once the forced expiratory volume in 1 second (FEV_1) falls to approximately 50% of predicted normal, symptoms usually appear.

• As COPD progresses, obstruction of the small airways occurs because of airway thickening and accumulation of inflammatory exudates induced by mucociliary dysfunction. Thus, in moderate to severe disease, early airway closure and incomplete alveolar emptying give rise to increased residual volume, air trapping, and lung hyperinflation, which are usually exacerbated by exercise.

• Destruction of alveoli decreases the surface area for gas exchange, while bronchial obstruction and expiratory air trapping produce uneven ventilation. Thus, there is V/Q mismatching, interference with gas exchange, and increased work of breathing.

• In addition, increased residual volume and air trapping producing lung hyperinflation result in a barrel-shaped chest (figure 9), which alters the mechanics of breathing. The efficiency of the primary respiratory muscles is reduced as the diaphragm becomes flattened (so its pull becomes more horizontal and inhibits chest expansion) and the accessory muscles of respiration are recruited, further increasing the work of breathing.

• Acute exacerbations, which are usually precipitated by respiratory infection or environmental pollutants, occur with greater frequency and severity as the disease processes progress and can be life-threatening.

• Airway resistance suddenly increases because of bronchospasm, mucosal edema, and intensified sputum production, which aggravate expiratory flow limitation and dynamic hyperinflation.

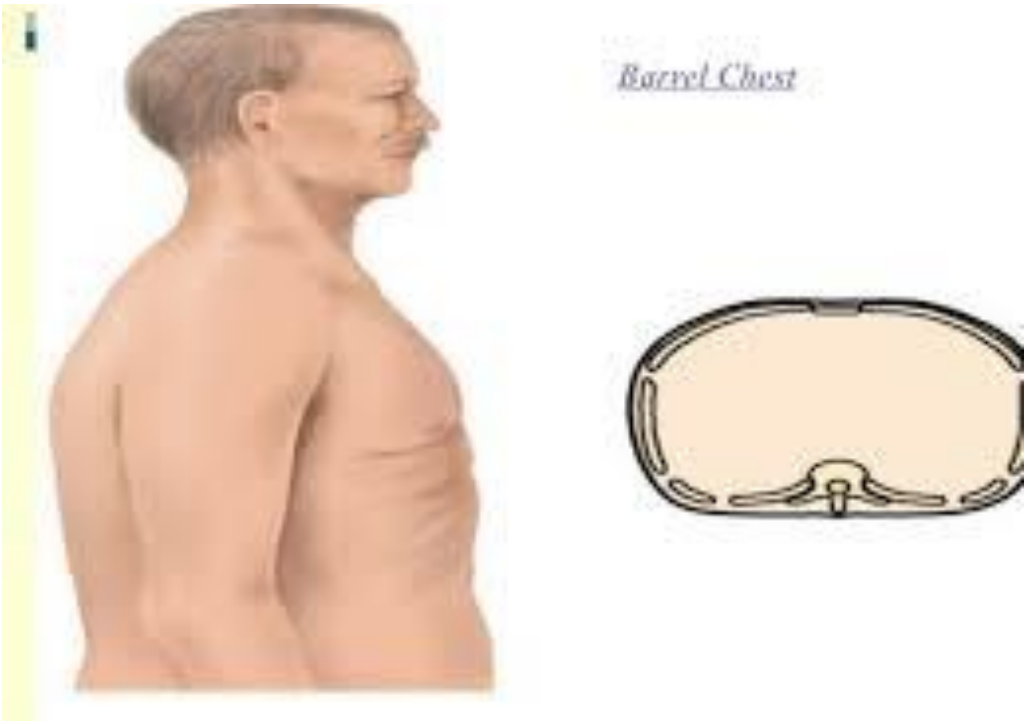
• The subsequent escalation of V/Q mismatching and work of breathing increases the potential for ventilatory failure and/or right ventricle (RV) failure and cor pulmonale.

• During exercise, pulmonary dysfunction increases and is associated with cardiovascular limitations. Exercise intolerance is the chief clinical complaint and is caused by a number of complex interdependent factors:

- Ventilatory limitation develops as a result of expiratory flow limitation, abnormal breathing mechanics, impaired gas exchange, and increased work of breathing.

- Excessively increased pulmonary vascular pressures at low workloads, even in individuals with normal pressures and little or no hypoxemia at rest, often result in pulmonary hypertension even without exercise-induced hypoxemia.
- RV pump dysfunction induced by pulmonary hypertension leads to reduced stroke volume and higher submaximal heart rates.
- Impaired skeletal muscle function develops as a result of structural and functional changes arising from hypoxemia, disuse atrophy, cachexia, corticosteroid therapy.

• The prognosis for individuals with mild COPD, especially if smoking cessation is achieved, is good, whereas that for patients with severe disease, especially if there is hypercapnia or cor pulmonale, is poor, with a mortality rate of 30% at 1 year and 95% at 10 years.



Barrel shaped chest

General clinical manifestations of COPD

• Dyspnea, especially on exertion

• Cough, often productive

• Reduced exercise tolerance

• Use of pursed-lip breathing for relief of dyspnea

• Prolonged expiration, so inspiratory-to-expiratory (I:E) ratio increases to 1:3 or 1:4 (normal, 1:2). With tachypnea; expiration is shortened, so I:E ratio decreases to 1:1, leading to increased air trapping in COPD

• Abnormal pulmonary function test (PFT) results with increased total lung capacity (TLC), residual volume, and functional residual capacity (FRC) and normal or decreased vital capacity (VC); reduced flow rates at all lung volumes ; and impaired diffusing capacity.

Classification of severity of COPD on the basis of spirometric data:
For educational reasons, a simple classification of disease severity into four stages (table 4).
Table 4. Classification of COPD by Severity

Stage	Characteristics
0 **At Risk**	- normal spirometry - chronic symptoms (cough, sputum production)
I **Mild COPD**	- $FEV_1/FVC < 70\%$. - $FEV_1 \geq 80\%$ predicted. with or without chronic symptoms (cough. sputum production)
II **Moderate COPD**	- $FEV_1/FVC < 70\%$ - $30\% \leq FEV_1 < 80\%$ predicted (IIA: $50\% \leq FEV_1 < 80\%$ predicted), (IIB: $30\% \leq FEV_1 < 50\%$ predicted) with or without chronic symptoms (cough, sputum production, dyspnea)
III **Severe COPD**	- $FEV_1/FVC < 70\%$. - $FEV_1 < 30\%$ predicted.

FEV_1: forced expiratory volume in one second; FVC: forced vital capacity.

• The term "**blue bloaters**" is often used to describe patients who have a stocky body build, bloated abdomen, and cyanosis (most common in chronic bronchitis), and the term "**pink puffers**" is used to describe patients who increase their work of breathing in order to maintain relatively normal oxygenation (most common in emphysema).

Respiratory muscles and COPD

1. Diaphragm only contributes 30% (compared with its usual 65%) of the inspiratory force, while the accessory muscles play an increased role.
2. The respiratory muscles may become fatigued and lung becomes hyper inflated.
3. There is increased resistance of their airways and the hyperinflation. The hyperinflation of the lung flattens the diaphragm, shortens the inspiratory muscles and places them at a mechanical disadvantage. In addition to the reduced efficiency of the inspiratory muscles, large amount of pressure work are required to overcome the high airway resistance.
4. During maximal exercise, the respiratory muscles may utilize 35-40% (normal 10-15%) of whole body oxygen consumption. More respiratory work is performed during inspiration.
5. About 25% of COPD patients are unable to maintain their nutritional status, as evidenced by weight loss. This nutritional depletion will increase mechanical and gas exchange impairment. In addition, loss of protein and lean body mass leads to skeletal muscle and diaphragmatic weakness.

Physiotherapy of COPD

COPD patients suffer from the following Problems:

1. Dyspnea: Due to dysfunctional pulmonary mechanics, weak ventilatory muscles, poor diaphragmatic positioning for length – tension functioning, increased airway resistance and inadequate gas exchange.
2. Accumulation of secretions.
3. Decreased exercise tolerance: Due to general muscle weakness, poor endurance and inadequate nutritional status.

Aims:

1. Relief of dyspnea.
2. Remove secretions.

3. Improve exercise tolerance.

Methods:

Relief of dyspnea:

1. **Relaxed positions:**

The first step towards self-help is positioning. It is an effective technique to reduce both the symptoms of breathlessness and the work of breathing.

2. **Breathing retraining exercises:**

Breathing exercises relieve dyspnea and improve gas exchange. The techniques most commonly taught are diaphragmatic breathing and pursed lips breathing or a combination of both.

a. Diaphragmatic breathing exercise:

Diaphragmatic breathing exercise increases the force of the diaphragm as an inspiratory muscle. It improves ventilation of small airways and bases of the lungs. In addition, it is often used in combination with pursed lips breathing and relaxation techniques.

b. Pursed lips breathing (PLB):

Pursed lips breathing exercise prevents collapse of airways during expiration due to maintain positive pressure in airways during expiration. In addition, prolonged expiration leads to decrease air trapping and residual volume. In addition, it recruits more alveolar units at the lung base.

3. **Breathing control techniques:**

Breathing control techniques encourage deep breathing and to control dyspnea (shallow rapid breathing).

Timing the breathing to steps works very well when walking or climbing stairs, e.g. one step to breathe in and two steps to breathe out, or one for each, or any rhythm or pattern that suits that particular individual. In addition, breathing control can be performed via diaphragmatic and pursed lips breathing exercise, which encourage deep breathing and control the dyspnea.

4. **Biofeedback and respiratory muscle training:**

Biofeedback teaches self-control over physiological functions and as a result, Ventilatory muscle training builds strength and endurance in the respiratory muscles.

Applications:

A. Incentive spirometry: The Goal of it's application is to encourage patient to take deep breathing which leads to reduction of breathlessness.
B. Peak expiratory flow meter: which encourage patient to do full expiration in each succeeding trial of expiration.
C. The oximetry biofeedback augmented pursed lips breathing training: patients can use pulse oximetry as a biofeedback guide to teach them to increase their oxygen saturation during performance of pursed lips breathing which relieves dyspnea and improves gas exchange, which result in improvement of oxygen saturation.

5. **Secretion clearance:**

A. Coughing:

Patients are trained and encouraged to cough and clear secretions effectively. As an alternative, the "huff" consists of a slow inspiration to total lung capacity, followed by huffs with the glottis open and may be effective. The multiple huffs are thought to minimize collapse of small airways, bronchospasm and fatigue.

B. Chest physiotherapy:

Postural drainage, percussion and chest wall vibration are clinically effective.

6. **Exercise:**

Muscle weakness both in skeletal and ventilatory muscles is common in COPD patients. Strength training in specific muscle groups has enabled patients to more comfortably and confidently perform their ADL. Hence, strength training may be adjunctive to endurance training.

Guidelines for exercise prescription for patients with COPD

A. Flexibility exercise:

Stretching of the major muscle groups of both upper and lower extremities. Flexibility/stretching considered as a part of the warm up before aerobic training and as part of the cool down after aerobic training.

B. Aerobic Exercises:

1. **Mode:** Should incorporate large muscle groups in the body. Types of exercise include walking, cycling, rowing, swimming etc.
2. **Frequency:** Recommended minimal frequency of training is three to five times per week.
3. **Intensity:** Minimal intensity 50% of peak VO_2. Another approach is to exercise at maximum limits tolerated by symptoms.
4. **Duration:** Minimal recommended duration is 20 to 30 min. of continuous exercise.

Chronic Bronchitis

Chronic bronchitis is characterized by a chronic cough with excessive mucus production that is not due to known specific causes, and is present for most days of at least 3 months of the year for two or more consecutive years.

Pathophysiology

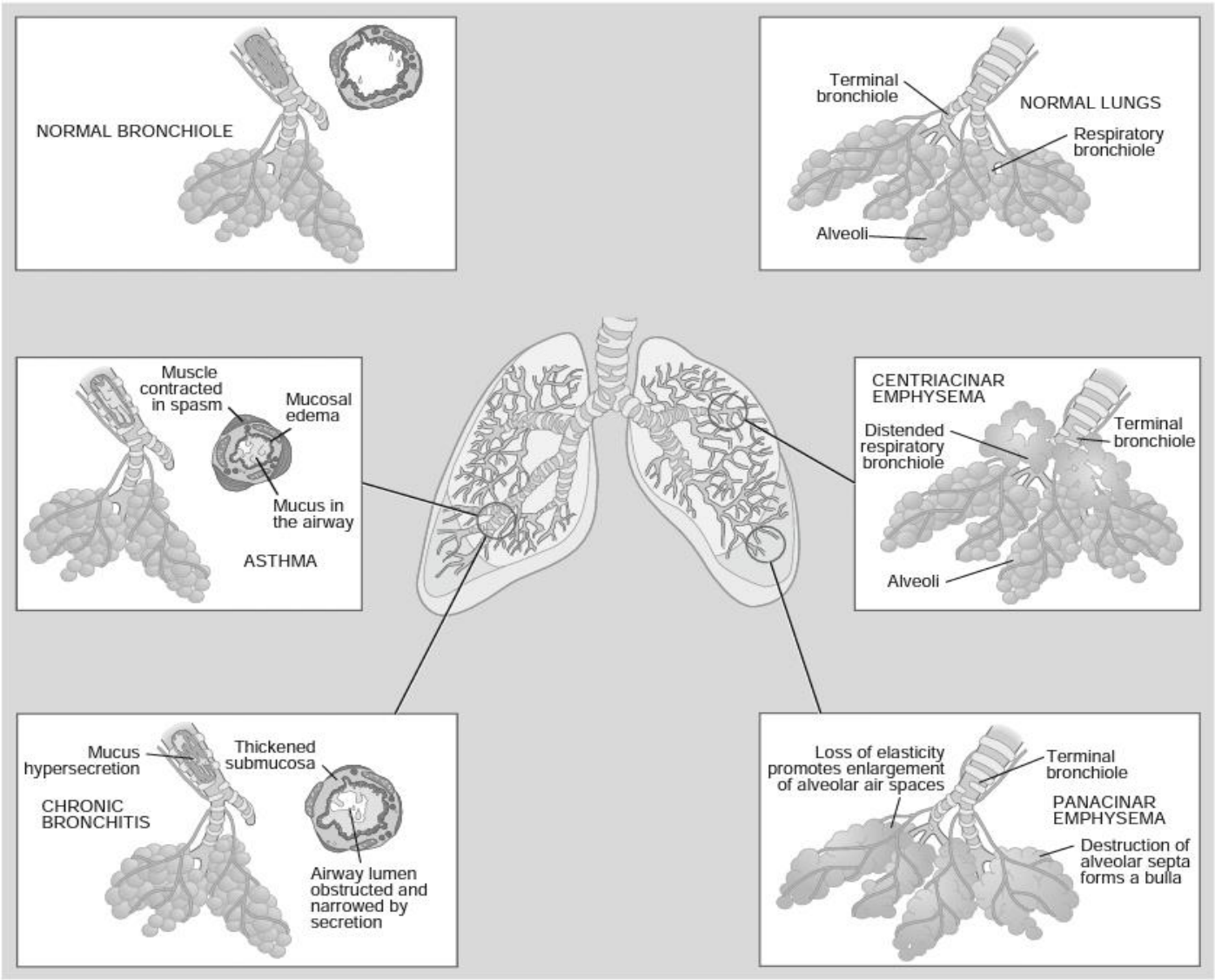
Chronic irritation of the airways (due to smoking, air pollution, occupational exposure, or bronchial infection) provokes an inflammatory response that stimulates pathological change in the bronchial walls, and leads to a chronic productive cough and recurrent pulmonary infections.

• Chronic inflammation of the airways induces mucosal edema, hypersecretion of mucus, and destruction of cilia, all of which increase airway resistance and produce expiratory flow limitation. Airway obstruction and pulmonary dysfunction develop.

• In addition, irritation of the airways can cause bronchoconstriction, which further increases airway resistance and expiratory flow limitation.

• During acute exacerbations, sputum production escalates and more secretions are retained, resulting in further aggravation of V/Q mismatching and increased work of breathing.

• With severe disease, the excessive work of breathing and deterioration of gas exchange, along with polycythemia, lead to ventilatory failure, cor pulmonale, or both.



Comparisons of normal airways and those affected by asthma, chronic bronchitis, and emphysema, both centriacinar and panacinar.

Clinical manifestations

• Insidious onset of cough, which progresses to chronic productive cough

• Progressive exertional dyspnea, with possible respiratory distress late in the disease

• Increased respiratory symptoms due to irritants, cold, damp or foggy weather, and acute pulmonary infections

• Normal breath sounds with prolonged expiratory time and possible crackles or wheezing.

• With more advanced disease:

  - Hypoxemia and increased arterial carbon dioxide ($PaCO_2$) (i.e., hypercapnia), respiratory acidosis.
  - Clinical manifestations of cor pulmonale, respiratory failure, or both.

Specific treatment:

• Medications include the following:

  - Bronchodilators.
  - Inhaled corticosteroids.

• Supplemental oxygen.

Emphysema

Emphysema consists of several diseases that ultimately result in permanent over distension of the air spaces distal to the terminal non respiratory bronchioles accompanied by destruction of the alveolar walls and without obvious fibrosis. Abnormal air spaces, called bullae and blebs, may be seen on chest radiograph when the disease is moderately advanced. The most common causes of emphysema include cigarette smoking and occupational exposure, which typically produce centriacinar destruction (see figure 10). A rarer cause is α1-protease inhibitor deficiency (α1-PI; also called α1-antitrypsin deficiency), which tends to cause a diffuse panacinar emphysema.

Pathophysiology

Repeated inflammation of the airways produces an imbalance between endogenous proteinases and antiproteinases, which causes progressive lung destruction.

• Loss of elastic recoil due to tissue destruction leads to expiratory collapse of distal, poorly supported airways, resulting in air trapping and alveolar overdistension. The alveolar walls fragment and attenuate so that several alveoli coalesce to form bullae, which can become quite large and compress adjacent lung tissue.

• In addition, the loss of large portions of lung parenchyma reduces the pulmonary vascular bed, which further increases pulmonary vascular resistance and accelerates pulmonary hypertension.

• As the disease progresses, pulmonary hyperinflation decreases the efficiency of the respiratory muscles and increases the work of breathing.

• Loss of functional alveoli results in gross V/Q mismatching throughout the lungs, hyperventilation of well-ventilated, well-perfused alveoli in order to maintain normal blood gases, increased work of breathing, and greater risk of ventilatory muscle fatigue and failure.

• Also, increased V/Q mismatching with more advanced disease interferes with gas exchange, leading to pulmonary hypertension, RV failure, and cor pulmonale.

Clinical Manifestations:

• Progressive dyspnea, especially on exertion

• Cough, often severe, with variable degrees of productiveness

• Increased symptoms with acute respiratory infections

• Decreased breath sounds with prolonged expiration, possible end-expiratory wheeze on forced expiration

• With more advanced disease:

- Diminished nutritional status, weight loss from greater energy used for breathing
- Hypoxemia and hypercapnia, respiratory acidosis
- Possible signs and symptoms of cor pulmonale, ventilatory failure, or both when end-stage disease develops

Specific Treatment:

• Medications:

- Bronchodilators
- Inhaled corticosteroids
- Supplemental oxygen

• Surgical interventions

- Bullectomy
- Lung volume reduction
- Lung transplantation, particularly for a1-PI deficiency

Clinical problems of chronic bronchitis and emphysema summarized:

a. An increase in the amount and viscosity of mucus production.
b. A chronic often productive cough.
c. Attacks of shortness of breath (dyspnea).
d. An abnormal breathing pattern with the most difficulty experienced during expiration which results in:

1. Use of accessory musculature.
2. Upper chest breathing.
3. Poor exchange of air in the lower lobes.

e. Changes in pulmonary function:
   1. Increased residual volume.
   2. Decreased vital capacity.
f. Decreased mobility of the chest wall – a barrel chest deformity develops.
g. Abnormal posture-forward head and rounded shoulders.
h. Decrease in general endurance, during daily activities.

Physical therapy treatment goals and plan of care:

Treatment Goals	Plan of Care
a. Decrease the amount and viscosity of secretions.	a. Administration of bronchodilators, antibiotics, and humidification therapy. If the patient smokes, he should be strongly encouraged to stop.
b. Remove or prevent the accumulation of secretions, (this is an important goal if emphysema is associated with chronic bronchitis or if there is an acute respiratory infection).	b. Deep and effective cough, postural drainage to areas where secretions are identified. **Note:** Drainage positions may need to be modified if the patient is dyspneic in the head-down position.
c. Promote relaxation of the accessory muscles of	c. Promote for relaxation

inspiration to decrease reliance on upper chest breathing and decrease tenseness associated with dyspnea.	relaxed head – up position in bed; trunk, arms, and head are well supported. -Sitting-leaning forward, resting forearms on thighs. -Sitting-leaning forward against pillows on a table. -Standing – leaning forward on an object, with hands on thighs or leaning backward against a wall. -Relaxation exercises for shoulder musculature. Active shoulder shrugging followed by relaxation. Shoulder and arm circles. Horizontal abduction and adduction of the shoulders.
d. Improve the patient's breathing pattern and ventilation. Emphasize relaxed expiration; decrease the rate of respiration and the use of accessory muscles. Carry over controlled breathing to functional activities.	d. Breathing exercises Relaxed diaphragmatic breathing with minimal upper chest movement. Lateral costal breathing. Pursed lip breathing (careful to avoid forced expiration). Practice controlled breathing during standing, walking, climbing stairs, etc.
e. Minimize attacks of shortness of breath.	e. Have the patient assume a relaxed position, so the upper chest is relaxed and the lower chest is as mobile as possible. Emphasize relaxed diaphragmatic breathing.

	Have the patient breathe out as rapidly as possible without forcing expiration (Note: Initially, the rate of respiration will be rapid and shallow. As the patient gets control of his breathing, he will slow down the rate). Administer supplemental oxygen in a severe attack, if needed.
f. Improve the mobility of the lower thorax.	f. Exercises for chest mobility emphasizing movement of the lower rib cage.
g. Improve posture	g. Exercises to decrease forward head and rounded shoulders.
h. Increase exercise tolerance	h. Graded endurance and conditioning exercises.

Asthma

Asthma is a chronic inflammatory condition characterized by airway hyperreactivity to various external and internal stimuli and manifested as recurrent episodes of intermittent reversible airway obstruction.

• When the provocative stimuli are immunologic in origin, sensitized mast cells exposed to specific antigens release bronchoactive mediators, causing asthma, which is most commonly seen in children.

• When the cause is not clearly related to allergy, as in adult onset asthma, sometimes it is due to sensitivity to aspirin or non steroidal anti-inflammatory drugs (NSAIDs), nasal polyposis, or sinusitis.

• Factors that may precipitate acute asthmatic attacks:

- Inhaled irritants (e.g., Pollens, smoke, dusts, pollution, sprays)
- Weather (e.g., high humidity, cold air, fog)
- Respiratory infections (e.g., common cold, bronchitis)
- Drugs (e.g., aspirin, other analgesics)
- Emotions (e.g., stress)
- Exercise

Pathophysiology

• Hyperreactivity of the airways to various stimuli provokes bronchial smooth muscle contraction and hypertrophy, mucosal edema, and overproduction of viscous mucus.

• Over time, bronchial airway remodeling occurs with increased thickness of all layers of the airways and leads to chronic stable asthma with intermittent acute exacerbations.

• Because ventilation is significantly reduced in numerous alveoli and perfusion is preserved or even increased by increased cardiac output, V/Q matching is poor to very poor, which impedes gas exchange and increases the work of breathing.

• Usually the individual hyperventilates in order to maintain near-normal oxygenation, resulting in hypocapnia and respiratory alkalosis; however, prolonged or severe attacks may lead to respiratory muscle fatigue with hypercapnia and respiratory acidosis, necessitating ventilatory support.

• If respiratory distress continues without response to treatment, status asthmaticus is present and hospitalization is required.

• Occupational asthma develops as a result of toxic exposure to various substances in the work place.

• In exercise-induced asthma, acute bronchoconstriction during exercise is thought to be provoked by hyperventilation, change in airway temperature, or water loss. The resultant increase in osmolality is proposed to stimulate activation of mast cells or afferent nerve endings, leading to bronchoconstriction.

Clinical Manifestations

• Recurrent paroxysmal attacks of cough, chest tightness, and difficult breathing, often accompanied by audible wheezing

• Thick sputum, which may be difficult to expectorate

• Symptom-free between attacks versus chronic state of mild asthma, with symptoms particularly noticeable during periods of exertion or emotional excitement

• Distant breath sounds, prolonged expiration; high-pitched expiratory and possibly inspiratory wheezes throughout both lungs.

• Tachypnea, possible signs of respiratory distress (e.g., increased use of accessory muscles, intercostal retractions, nasal flaring)

• Markedly reduced FEV_1, forced expiratory flow from 25% to 75% of vital capacity (FEF25%-75%), and maximal expiratory flow rate at all lung volumes and increased total lung capacity (TLC) during acute attacks; increased airway resistance and reduced maximal expiratory flow rates during remissions (patients often monitor their status via daily peak expiratory flowmeter readings).

• During acute attacks, hypoxemia with hypocapnia is common; normal or increased $PaCO_2$ is a serious sign indicating ventilatory insufficiency and possible impending respiratory failure.

Specific Treatment:

- Medications
  - Short-acting ß$_2$-agonists as rescue therapy
  - Inhaled corticosteroids
  - If unsuccessful, long-acting bronchodilators, leukotriene modifiers, mast cell stabilizers, or anticholinergic drugs
- Avoidance of asthma triggers

Physical therapy treatment goals and plan of care:

Treatment Goals	**Plan of Care**
a. Decrease bronchospasm.	a. Removal of allergen (s); bronchodilators with intermittent positive pressure breathing (IPPB).
b. Minimize attacks of shortness of breath and gain control of breathing.	b. Relaxation of upper chest and accessory muscles by positioning Diaphragmatic breathing, emphasizing relaxed expiration.
c. Mobilize and remove secretions after attack of shortness of breath.	c. Humidification of secretions with aerosol therapy. Effective coughing. Postural drainage (after, not during, the asthmatic attack, as it may increase bronchospasm).
d. Correct posture to decrease rounded shoulders and forward head.	d. Postural training.

e. Gradually increase exercise tolerance and endurance.	e. Avoid prolonged, vigorous physical activities. Encourage mild to moderate activities for short periods, followed by rest. Use controlled breathing during exertion.

Bronchiectasis

Bronchiectasis is characterized by progressive dilation and destruction of bronchi and bronchioles. It is usually localized to a few lung segments or an entire lobe of one lung, although bilateral diffuse involvement is also common. Before antibiotic therapy and immunizations, bronchiectasis usually developed as a sequela of a chronic necrotizing pulmonary infection, but now it occurs most often in patients with underlying systemic disorders on which airway infection is superimposed, such as primary ciliary dyskinesia, cystic fibrosis, and immune deficiency. Although bronchiectasis can be classified according to the resultant airway deformities (e.g., cylindrical, varicose, or saccular/cystic), there is little clinical or pathophysiological difference between the types. The hallmark of bronchiectasis is a chronic productive cough, often with copious amounts of mucopurulent sputum.

Pathophysiology

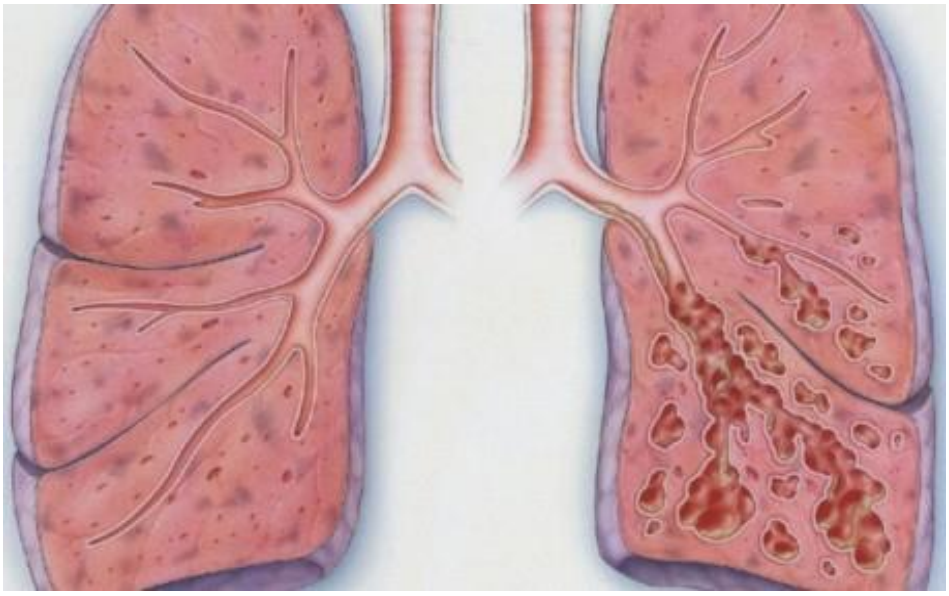
• Inflammation of the bronchial walls, due to acute or chronic infection or possibly defective regulation of the inflammatory response caused by congenital syndromes, immune deficiencies, and many other disorders, results in mucociliary clearance dysfunction, which leads to a vicious cycle of persistent bacterial colonization, chronic mucosal inflammation, and progressive tissue destruction.

• Bronchial dilation and distortion are caused by destruction of the elastic and muscular airway components and hypertrophy and hyperplasia of the surrounding undamaged musculature.

• With long-standing disease there is damage to the peribronchial alveolar tissue by inflammation followed by fibrosis, squamous metaplasia of the bronchial epithelium with loss of ciliated cells, obliteration of the distal bronchi and bronchioles, and hypertrophy of the bronchial arteries with anastomosis and sometimes considerable shunting of blood to the pulmonary arteries.

• During exacerbations, there is increased obstruction and additional secretion production, causing greater V/Q mismatching, which results in impaired gas exchange and increased work of breathing.

• In rare cases in which disease is severe, increased V/Q mismatching can activate the events that ultimately lead to respiratory muscle fatigue or failure and cor pulmonale.



Bronchiectasis

Clinical Manifestations

• Persistent or intermittent productive cough with variable amounts of purulent sputum (often copious)

• Dyspnea, particularly if both lungs are extensively involved; tachypnea

• Possible pleuritic chest pain

• Fever, loss of appetite, weakness, weight loss

• May be asymptomatic between episodes of acute infection

• Possible blood-streaked sputum or frank hemoptysis

• Adventitious breath sounds, including crackles and often wheezes

• Possible clubbing of digits

• Expiratory air flow limitation on PFTs, which correlates with the severity of the disease and may be reversible with bronchodilators

• Possible hypoxemia even in mild disease; severe hypoxemia with hypercapnia and acidemia if diffuse disease

• If severe disease, signs and symptoms of pulmonary hypertension and possibly cor pulmonale

clubbing of fingers

Specific treatment

• Medications:

- Antibiotics for acute exacerbations
- Inhaled corticosteroids
- Mucolytics
- Possible long-acting ß$_2$-agonists, leukotriene modifiers, oral steroids

• Surgical resection if localized disease

Physical therapy treatment goals and plan of care:

Treatment Goals	Plan of Care
a. Clear the airways of secretions.	a. Effective, controlled cough postural drainage during acute episodes
b. Prevent recurrent infections	b. Home program of postural drainage to be carried on throughout life.

1. **Precautions:**
   a. If mild hemoptysis (blood – streaked sputum) occurs, continue postural drainage, but omit percussion for at least 24 hours.
   b. If severe hemoptysis (hemorrhage) occurs, discontinue postural drainage until further notice.

Cystic fibrosis

Cystic fibrosis is a genetically based disease (autosomal recessive) which involves malfunction of the exocrine glands, leading to abnormal secretions in the body. The disease is characterized by a very high concentration of sodium in the sweat, diffuse lung disease, and malfunction of the pancreas. The disease must be managed throughout life with diet, medication, and preventive chest physical therapy as soon as any symptoms are noted in the young child.

1. **Clinical picture:**
   a. These children are usually small for age because of mal-absorption of foods.
   b. The exocrine gland dysfunction leads to increased production of viscous mucus, which obstructs the airways. Chronic obstruction of the airways and pooling of secretions leave the child vulnerable to pulmonary infection.
   c. Prognosis for survival has improved in the past 20 years. The average patient now survives into the late 20s or early 30s. The digestive involvement can be managed by diet; pulmonary complications are eventually the cause of death.

2. **Clinical problems of cystic fibrosis summarized:**
   a. Increased production of viscous mucus throughout the lungs.
   b. Periodic pulmonary infections.
   c. Possible problems of compliance with a life – long regimen of home postural drainage and prevention of lung infections.

Physical therapy treatment goals and plan of care:

Treatment Goals	Plan of Care
a. Prevent accumulation of secretions and pulmonary infection.	a. Daily home program of postural drainage, usually twice per day, if no acute pulmonary problems exist.

b. Decrease viscosity of secretions.	b. Humidification therapy with mist tent or IPPB.
c. Prevent use of accessory muscles of respiration.	c. Diaphragmatic breathing and lateral costal expansion. Daily practice and use of deep breathing during postural drainage is important. Emphasize relaxed expiration so bronchospasm and air trapping does not occur.
d. Removal of secretions during an acute infection.	d. Postural drainage for longer periods, as needed. Appropriate use of antibiotics.

Note: The key to successful preventive treatment of cystic fibrosis over many years is a consistent home program of postural drainage. This requires a supportive and cooperative family atmosphere.

Restrictive lung disease

An abnormal reduction in lung expansion, and therefore pulmonary ventilation, can be caused by a number of lung parenchymal diseases that interfere with lung expansion, pleural abnormalities, like Pleural effusion, Pleural fibrosis Pneumothorax, and hemothorax and result in restrictive lung dysfunction (RLD). Chest x-ray findings are extremely variable in RLD because of the diverse nature of the abnormalities that produce it. Pulmonary function testing typically reveals reductions in almost all volumes and capacities with relatively normal flow rates, as well as reduced diffusing capacity when RLD is due to lung disease.

Pathophysiology

• RLD results in limited lung expansion.

- If a lung is less compliant, greater transpulmonary pressure is required to expand it to any given volume, which is usually less than normal.
- If pleural abnormalities compress the lung, normal expansion is inhibited.
- If ventilatory pump dysfunction (e.g respiratory muscle weakness and thoracic deformity) impairs expansion of the thoracic cage in any direction, lung expansion will be reduced even though lung compliance may be normal.

• Thus, decreased pulmonary compliance results in greater resistance to lung expansion, increased effort of the inspiratory muscles, especially the diaphragm, with recruitment of the accessory muscles of inspiration, and higher respiratory rates; thus the work of breathing is markedly increased.

• The greater the severity of restriction, the more dependent the individual is on respiratory rate as the only means to increase minute ventilation to meet higher demands for oxygen, and the greater the ventilatory limitation.

• In addition, reduced pulmonary compliance produces ventilation– perfusion (V/Q) mismatching, which further increases the work of breathing, creating the potential for ventilatory muscle fatigue and ventilatory failure as the disease progresses in severity.

• Furthermore, chronic hypoventilation seen in moderately severe disease results in hypoxemia with resultant hypoxic vasoconstriction and increased pulmonary artery pressures (especially during exertion), as well as polycythemia; this further increases the workload of the right ventricle, leading to right ventricular hypertrophy and possible eventual cor pulmonale.

Clinical Manifestations

• Dyspnea, especially on exertion

• Reduced exercise tolerance

• Cough, usually dry non productive

• Tachypnea

• Possible weight loss

• Possible hypoxemia

• Abnormal PFTs with decreased VC, inspiratory capacity (IC), and TLC, and possible impaired diffusing capacity, but relatively normal expiratory flow rates

• Abnormal breath sounds

• Abnormal chest x-ray findings according to specific disorder

General Treatment

•Specific therapeutic or corrective measures (e.g., appropriate anti microbial therapy for pneumonia, treatment of pulmonary edema, reversal of drug-induced central nervous system depression)

• Supportive measures:

- Supplemental oxygen
- Nutritional support
- Ventilatory support

• Physical therapy interventions:

General Goals:

1. Relief dyspnea.
2. Increase chest mobility and expansion.
3. Correct postural defects.
4. Relieve pain.
5. Improve exercise tolerance.

This can be achieved by:

1. Respiratory exercises: nose ex, localized breathing exercise, deep breathing exercise, exercise connected with respiration.

   Using of some devices as incentive spirometer, inspiratory muscle trainer → inspiratory resistance exercise.
2. Mobilizing exercises: active free through full ROM.
   - Swinging exercise.
   - Gym ex. using shoulder wheel, raw machine, parallel bar.
3. Stretching exercises: Pectoralis muscle and hip flexors stretching exercises.
4. Pain relief modalities: any source of heat especially moist heat, infrared, massage, TENS, diadynamic currents or laser.
5. Endurance exercises treadmill training, walking, bicyle ergometer and swimming exercises.

Specific restrictive pulmonary conditions

Dry pleurisy

Definition:

Inflammation of the pleura, of one or both sides with no detectable free exudates.

Aetiology:

1. Pneumonia the commonest cause.
2. Pulmonary infarction.
3. Bronchial carcinoma.
4. Lung abscess.
5. Pulmonary tuberculosis.
6. Extension from a sub diaphragmatic abscess.

Pathological changes:

1. Pleural membrane becomes hyperaemic and red.
2. Fibrin deposited on the inflamed membrane.
3. Adhesions formed between both pleural layers.
4. So respiratory movement is restricted causing pain.

Clinical Features:

Symptoms:

1. Pleuritic pain: Pain that is maximal at the end of inspiration, it is worsened by deep breathing and coughing. It may be local or referred to the anterior chest wall or – in the presence of diaphragmatic pleurisy – to the front of shoulder, or to the anterior chest wall.
2. Difficulty of breathing.
3. Dry cough.
4. Bending toward the painful side or lying down on affected side (auto splinting).

Signs:

1. Rapid and shallow breathing pattern.
2. Asymmetric breathing: limitation of chest movement on the affected side in cases of diaphragmatic pleurisy.
3. On palpation of chest wall: there is tenderness over the area of pleurisy.
4. Pleural friction rub: which stimulates crepitations, yet is unaltered by coughing.
5. Decreased the tactile vocal frimitius: due to limited air volume.
6. On auscultation: there is a decreased breathing sound over the affected side.

Treatment

Medical:
Antibiotics, anti-inflammatory, antipyretics and analgesics.

Physical therapy:

Aims:
1. To relax the patient and improve respiration.
2. To relieve the pain.
3. To prevent the postural deformity.

Methods:
1. Rest in bed in proper supported alignment.
2. Application of a moist heat.
3. Bandage or strapping of the painful sides.
4. Positioning of affected side to prevent deformity.

Pleural Effusion

Accumulation of fluid in the pleural cavity as a result transudation or exudation from the pleural surfaces.

Aetiology:

Transudates (hydrothorax): as in congestive heart failure, constrictive pericarditis and myxoedema.

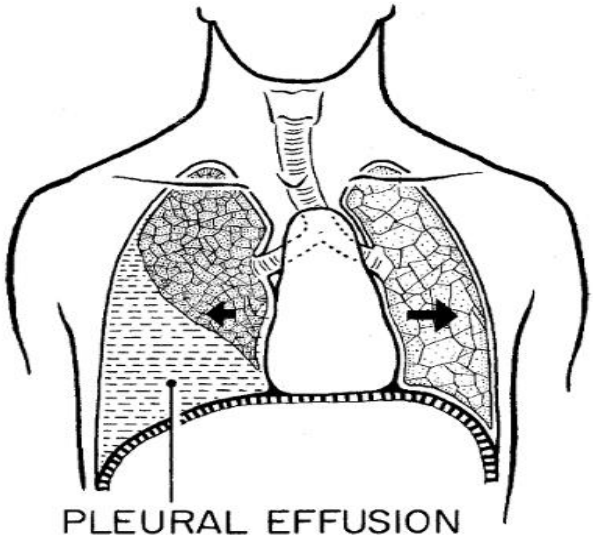
Exudates: fluid with a high protein content of > 3 gm/100mL accumulates in the pleural space; it may occur due to bacterial pneumonia, pleural malignancy and T.B and collagen diseases as rheumatic fever, rheumatoid arthritis.

Clinical Feature:

Symptoms:
Acute symptoms onset: high fever, fatigue, dyspnea. Gradual, onset: toxemia, dull aching pain.

Signs:
1- Signs of the primary disease.
2- Signs of the fluid in the pleural space:
   - Decreased or absent ribs movement on affected side.
   - Displacement “shifting” of apex beat and usually trachea to opposite side (in large effusion).

- Stony dull percussion.
- Distant breath sounds: High-pitched bronchial breathing may be heard over upper margin of effusion.
- Pleural rub may be heard above fluid.
- Vocal resonance decreased or absent fremitus.



A diagram showing a large right pleural effusion with marked compressive atelectasis and mediastinal shift away from the affected side.

Treatment:

1- Treatment of the primary cause.

2- Build up the body resistance by proper diet.

3- Aspiration of the excess pleural fluid to reduce dyspnea. (Thoracentesis: the insertion of a needle into the pleural space allows for the removal of pleural fluid or acquisition of a pleural biopsy).

4- Physical therapy treatment:

• Positioning: on the normal side to improve ventilation/ perfusion ratio, also it helps the movement on the affected side and subsequently helps the drainage.

• Breathing exercises: diaphragmatic and localized breathing exercises.

• Postural exercises: to maintain good posture and avoid chest wall unilateral contracture.

• Aerobic exercises: as walking and up and down stairs to maintain physical endurance and fitness.

Empyema

Definition:

Empyema is the presence of pus in the pleural cavity.

Aetiology:

1- Extension of infection from the lung as in T.B, Pneumonia, cancer or lung abscess.
2- Extension of infection from the mediastinum or chest wall.
3- Sub diaphragmatic abscess.
4- General as septicemia.

Clinical Feature:

Symptoms:

1- Those of the primary disease, usually pneumonia.
2- Fever, rigors, pleuritic pain and later loss of weight.
3- Toxemia with swinging temperature.
4- Insomnia.
5- Chest pain.
6- Sudden coughing of a large amount of sputum (pus), which may be blood stained indicates the occurrence of a bronchopleural fistula.

Signs:

1- Clubbing fingers, developing is 2-3 weeks.
2- Deformity of the chest wall.
3- Restricted movement of the chest on the affected side.
4- Scoliosis to the affected side.

Treatment:

Aim of treatment:

1- Control of infection.

2- Removal of pus.

3- Obliteration of empyema space.

Medical treatment:

- Appropriate antibiotics and analgesics.

Surgical treatment:

- Repeated aspiration in case of thin pus.
- Thoracoplasty.

Physical therapy treatment:

Aims:

- To re-expand the lung after aspiration.
- To prevent the deformity.
- To maintain adequate range of motion in the upper limbs and trunk.
- To relieve pain and anxiety.
- To reduce dyspnea and respiratory rate.

Post-operative aims:

- To prevent pulmonary complications.
- To prevent circulatory complications.
- To prevent chest wall contracture and deformity.
- To improve lung expansion.
- To improve physical fitness.

Physical therapy methods:

- Respiratory exercises.
- Circulatory exercises and early ambulation.
- Postural exercises.
- Endurance exercises.
- Heat application.

Atelectasis

Atelectasis is a restrictive lung dysfunction in which lobes or segments of a lobe have been collapsed.

Clinical picture

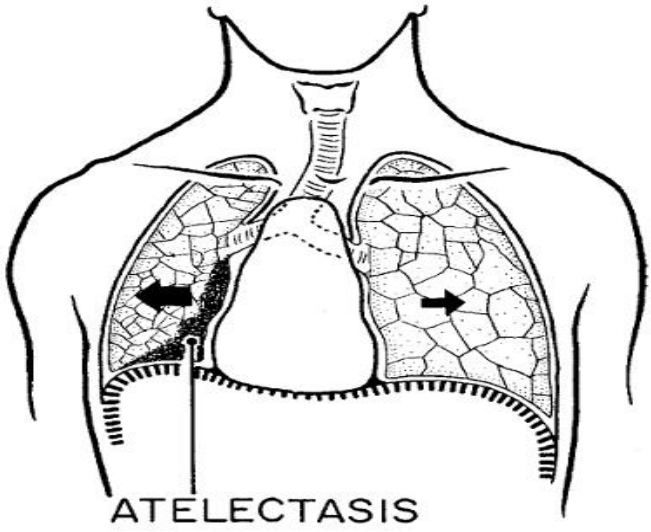
1- Absent breathing sounds over the collapsed lung area.
2- Tachycardia and cyanosis.
3- Decreased chest movement over the affected area.

Treatment Goals:

1- Re inflate collapsed areas of the lung
2- Increase inspiratory capacity.

Methods

1- Postural drainage with percussion and vibration.
2- Effective cough.
3- Segmental breathing with emphasis over collapsed areas.



A diagram showing atelectasis with elevated hemidiaphragm and mediastinal shift toward the affected side.

Other forms of pleural disorders:

Hemothorax: Hemorrhage into the pleural space.

Pneumothorax: The presence of air in the pleural cavity.

Breathing exercises

A number of breathing retraining techniques are used to relieve and control dyspnea in patients with COPD and asthma. PTs often teach diaphragmatic breathing and pursed-lip breathing to improve ventilation, ventilatory efficiency, and gas exchange, and mobilize secretions.

Diaphragmatic breathing exercises

Diaphragmatic breathing exercises are performed to improve ventilation, decrease the work of breathing, alleviate dyspnea, normalize breathing pattern, and reduce the incidence of postoperative pulmonary complications.

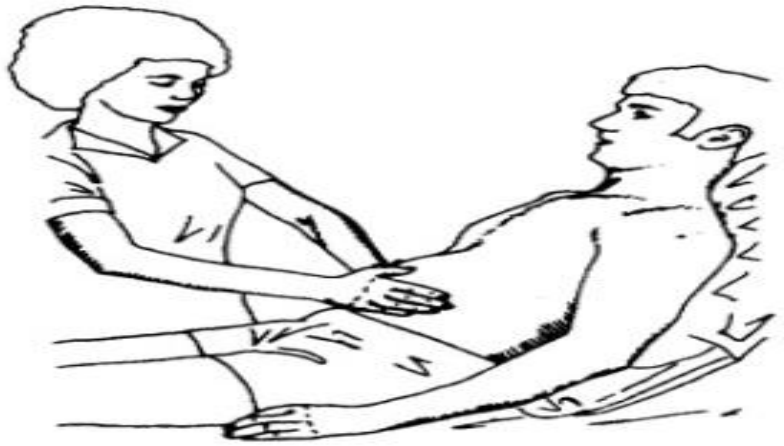
- Have the patient assume a comfortable position as sitting supported or semi-fowler position. In patients with COPD with marked hyperinflation of the lungs and a paradoxical breathing pattern, sitting with trunk flexion is recommended.
- Put one hand on the patient's abdomen above the umbilicus and the other on the upper chest while asking the patient to breathe slowly and comfortably.
- Slowly breathe in through your nose and feel your belly rise into your hand.
- Slowly breathe out through pursed lips and feel your belly fall away from your hand.
- Keep your shoulders relaxed - not hunched up.
- Repeat 5 times.

Diaphragmatic strengthening exercises:

- Resistance can be applied with weights, manual pressure, positioning, or incentive spirometry.
- Weights are applied over the epigastric area with the patient in the supine position, thus providing resistance to diaphragmatic descent.
- Cuff weights tend to be the most comfortable and have less chance of falling off the patient with the breathing movement.

- Manual resistance by therapist hand can be applied in a similar manner over the epigastric area to resist the diaphragmatic descent.
- The patient perform several series of three to five slow sustained deep diaphragmatic breaths with interposed rest periods.

- The proper starting weight should permit full diaphragmatic excursion (i.e., full epigastric rise) using a coordinated, unaltered breathing pattern (no signs of accessory muscle contraction) for 15 minutes.
- As strength improves, additional weight can be added until the patient demonstrates normal strength.



Diaphragmatic breathing

Segmental Breathing Exercises

Segmental breathing exercises, also called localized expansion breathing exercises or thoracic expansion exercises are used to increase localized lung expansion. The goals are to increase and redistribute ventilation, improve gas exchange, aid in re-expansion of air spaces, mobilize the thoracic cage, and increase the strength, endurance, and efficiency of the respiratory muscles.

Segmental breathing exercises use manual pressure as proprioceptive input to encourage expansion of specific areas of the chest. Hand placements are unilateral or bilateral.

- Identifying the area of treatment.
- The therapist's hand(s) are placed on the appropriate part of the patient's chest.

- The therapist instructs the patient to take a slow, deep breath in through the nose "to fill my [the therapist's] hand with air, or push my hand up by your chest wall" while applying gradually diminishing resistance to permit full range of motion.
- Maximal inspiration is sustained for 2 to 3 seconds and then the patient exhales.
- The therapist apply manual pressure at the end of expiration.

The patient can perform the exercise with her/his own hands or a towel or belt. The patient places a towel/belt over the area of treatment, grasps the ends with her/his hands, and applies firm pressure at the end of expiration. As the patient is taking a slow, deep breath in through the nose, s/he is applying gradually diminishing resistance through the towel/belt, adjusting to allow full ROM. Maximal inspiration is sustained for 2 to 3 seconds and then the patient exhales, which may be assisted manually or with the towel/belt.

Types of segmental breathing exercises:

A. Lateral costal expansion

1. This is sometimes called lateral basal expansion and may be done unilaterally or bilaterally.
2. The patient may be in sitting position.
3. Place your hands along the lateral aspect of the lower ribs to fix the patient's attention to the areas which movement is to occur.
4. Ask the patient to breathe out, and feel the rib cage move downward and inward.
5. As the patient breathes out, place firm downward pressure into the ribs with the palms of your hands.
6. Just prior to inspiration, apply a quick downward and inward stretch to the chest. This places a quick stretch on the external intercostals to facilitate their contraction. These muscles move the ribs outward and upward during inspiration.
7. Tell the patient to expand the lower ribs against your hand as he or she breathes in.
8. Apply gentle manual resistance to the lower rib area to increase sensory awareness as the patient breathes in and the chest expands and ribs flare.
9. Then, again, as the patient breathes out, assist by gently squeezing the rib cage in a down ward and inward direction.

10. The patient may then be taught to perform the maneuver independently. He or She may place the hand (s) over the ribs or apply resistance using a belt.

B. Posterior basal expansion

1. Have the patient sit and lean forward on a pillow, slightly bending the hips.
2. Place your hands over the posterior aspect of the lower ribs.
3. Follow the same procedure as described above.
4. This form of segmental breathing is important for the post surgical patient who is confined to bed in a semi upright position for an extended period of time. Secretions often accumulate in the posterior segments of the lower lobes.

C. Right middle lobe or lingula expansion

1. Patient is sitting.
2. Place your hands at either the right or the left side of the patient's chest, just below the axilla.
3. Follow the same procedure as described for lateral basal expansion.

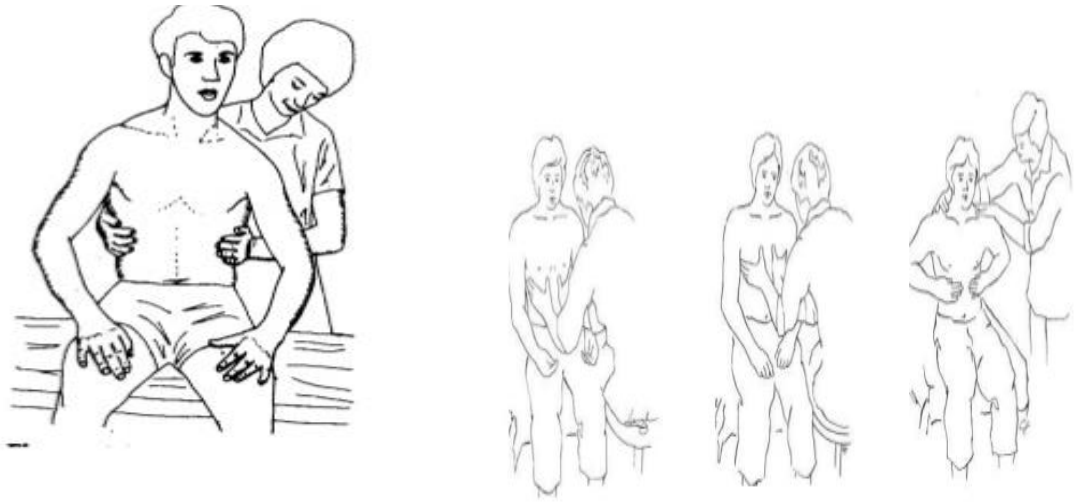
D. Apical expansion

1. Patient in sitting position.
2. Apply pressure (usually unilaterally) below the clavicle with the fingertips.
3. This pattern is appropriate in an apical pneumothorax after a lobectomy.

Hand placement in segmental breathing exercise:

Hand location of the therapist	Local area of lung being augmented
Subclavicular areas	Upper lobes.
Anterior midchest	Right middle lobe and lingual.
Anterior lower ribs	Anterior basal segments of the lower lobes.
Lower lateral costal area	Lateral basal segments of the lower lobes.

Posterior lower chest	Posterior basal segments of the lower lobes.
Posterior midchest (with scapulae abducted)	Superior segments of the lower lobes



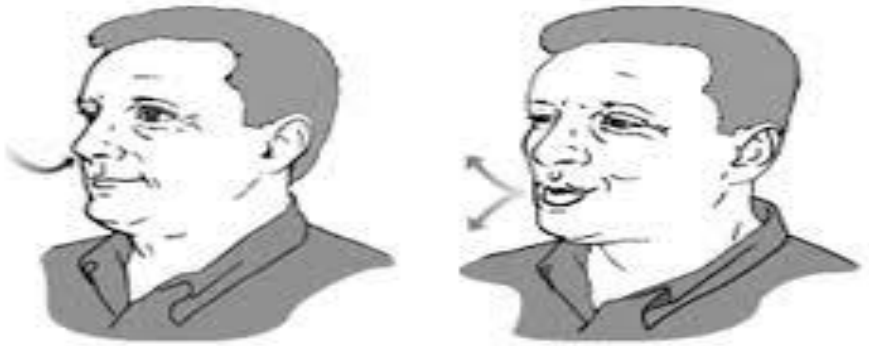
Costal breathing exercises

Pursed-lips breathing

Pursed-lips breathing aims to slow the rate of expiration, increase the volume of expired air, and limit dynamic hyperinflation during periods of increased ventilatory demand, including daily activities and exercise. It has been shown to decrease the respiratory rate, improve gas exchange (both oxygen and carbon dioxide), increase tidal volume, and reduce the work of breathing, thus relieving dyspnea and increasing exercise tolerance.

- The patient should assume a comfortable position.

- With a hand on the patient's mid abdominal muscles, the therapist instructs the patient to inhale slowly through the nose.
- The patient is then told to let the air escape gently through the pursed lips, avoiding excessive use of the abdominal muscles.
- Giving the patient a verbal cue, such as "imagine you want to make the flame flicker on a candle that is being held at arm's length from you.
- The patient is directed to stop exhaling when abdominal contraction is detected.



Pursed-lips breathing

Incentive Spirometry

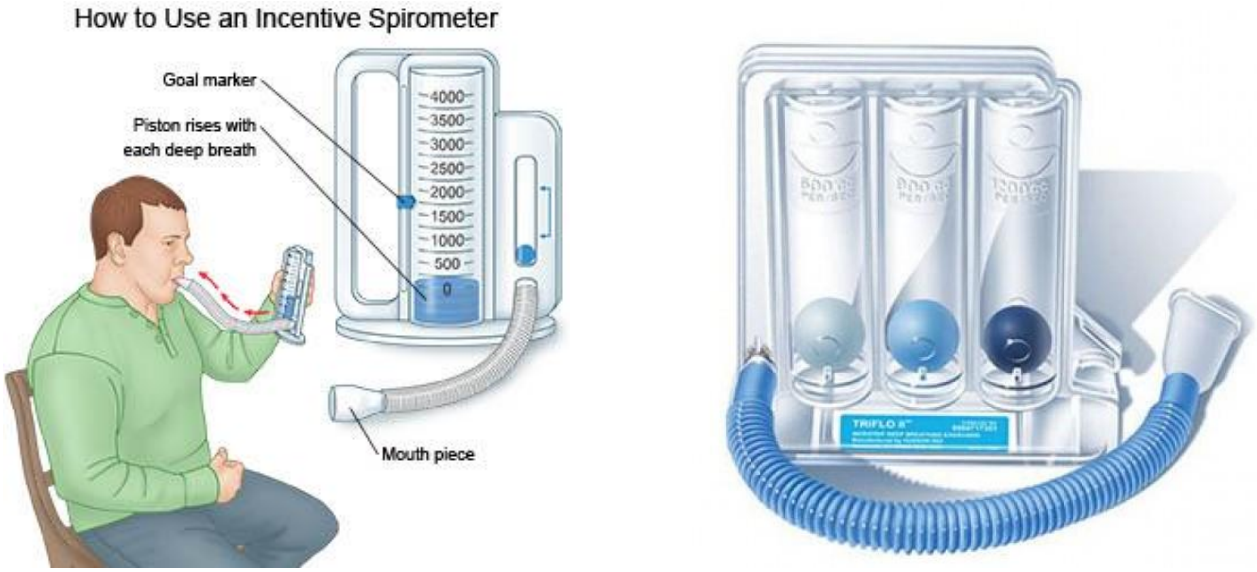
Incentive spirometry (IS) involves the use of a visual or auditory feedback device to encourage reproducible slow, deep inspirations. IS is commonly performed by postoperative patients in order to reduce the incidence of pulmonary complications associated with surgery-related shallow breathing, bed rest, diaphragmatic dysfunction, pain, and impaired mucociliary clearance.

Two types of incentive spirometers are commonly used:

- A flow IS typically consists of one or more chambers containing small balls that are elevated by sufficient inspiratory air flow.
- A volume IS consists of bellows or pistons that register the volume inspired and can indicate whether a preset volume goal is achieved.

Instruction in proper technique is critical to the effectiveness of IS, especially with flow devices: a rapid inspiratory rate will elevate the balls to the top of the chambers with a relatively low-volume breath; or a deep breath at a slow flow rate may achieve a high volume without moving the balls.

- The patient is in sitting, semi-fowler, or supine position (with the head of the bed raised 45 degrees, to relieve diaphragmatic resistance applied by the abdominal contents.
- Put the mouthpiece in the mouth, forming a tight seal with the lips, and then performs a slow, deep diaphragmatic breath, minimizing any upper chest movement.
- The inspiration is sustained 5 seconds to allow collateral ventilation of the well-aerated alveoli with those that are poorly ventilated.
- The mouthpiece is removed and the patient relaxes to allow expiration.
- This procedure is performed 10 times every hour.



Incentive spirometer

Breathing exercise connected with posture

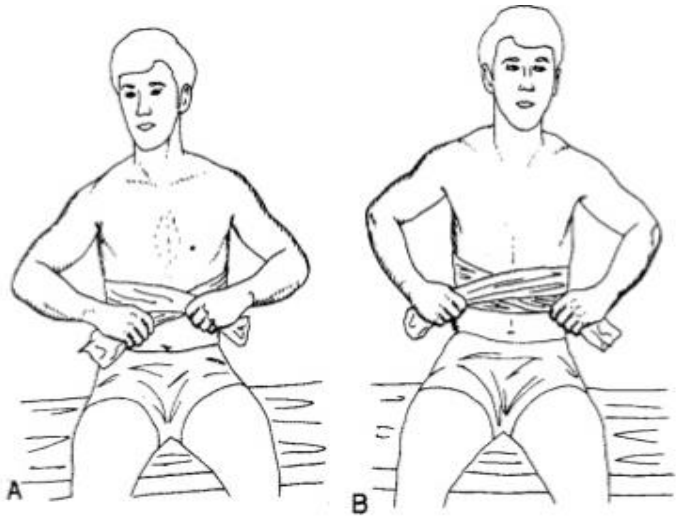
- This can give mobilizing, strengthening and stretching exercise connected with respiration.
- Any movement away from the chest is connected with inspiration and toward the chest is connected with expiration.

Belt exercise

Length = 2 meter Width = 30 cm

Advantages:

- After operations as it make equal pressure in all points and takes a large area so its pressure become less painful.
- Easy to be used by the patient himself.



Exercises to mobilize the chest

A. Definition

Chest mobility exercises are any exercises that combine active movements of the trunk or extremities with deep breathing.

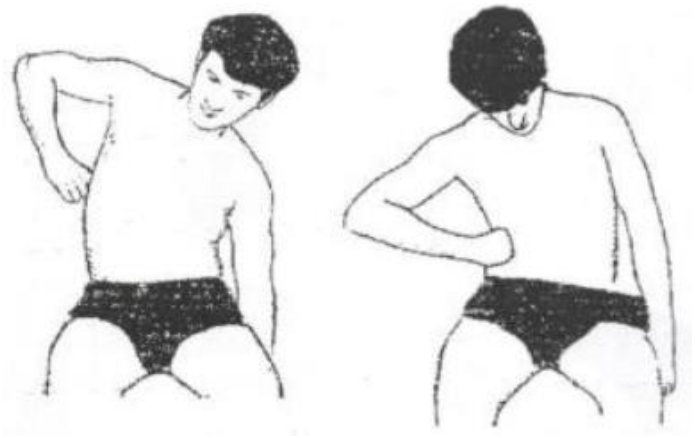
B. Goals

Maintain or improve mobility of the chest wall trunk and shoulders when it affects respiration.
A patient with tightness of the trunk muscles on one side of the body will not expand that part of the chest fully during inspiration. Exercises that combine stretching of these muscles with deep breathing will improve ventilation on that side of the chest.
Reinforce or emphasize the depth of inspiration or controlled expiration. For example, a patient can improve expiration by leaning forward at the hips or flexing the spine as he or she breathes out. This pushes the viscera superiorly into the diaphragm and further reinforces expiration.

C. Specific Exercises:

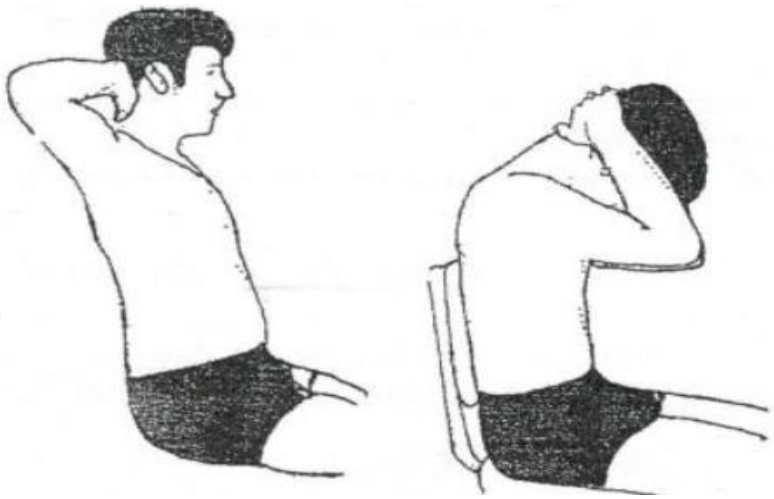
1. To mobilize one side of the chest.

While sitting, have the patient bend away from the tight side to lengthen tight structures and expand that side of the chest during inspiration.
Then, have the patient push the fisted hand into the lateral aspect of the chest, as he or she bends toward the tight side and breathes out.
Progress by having he patient raise the arm on the tight side of the chest over the head and side bend away from the tight side, This will place an additional stretch on the tight tissues.



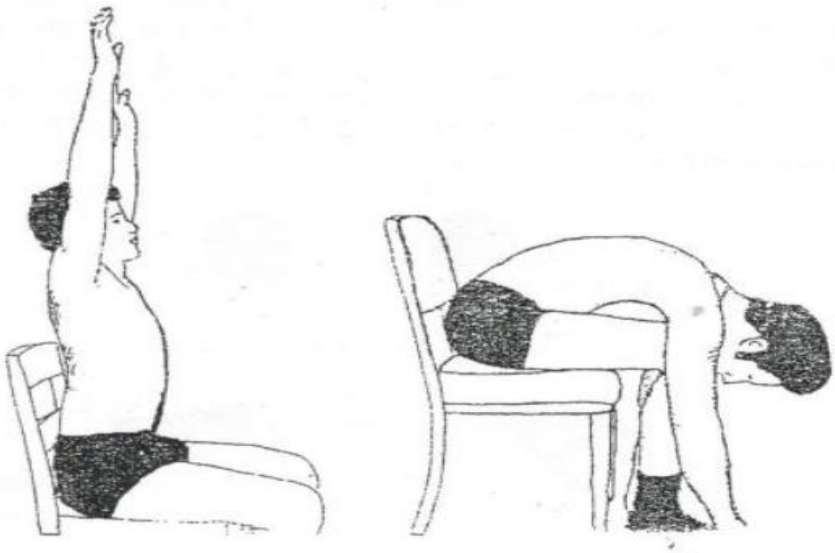
2. To mobilize the upper chest and stretch the pectoralis muscles.

While the patient is sitting in a chair with hands clasped behind the head, have him or her horizontally abduct the arms (elongating the pectoralis muscles) during a deep inspiration.



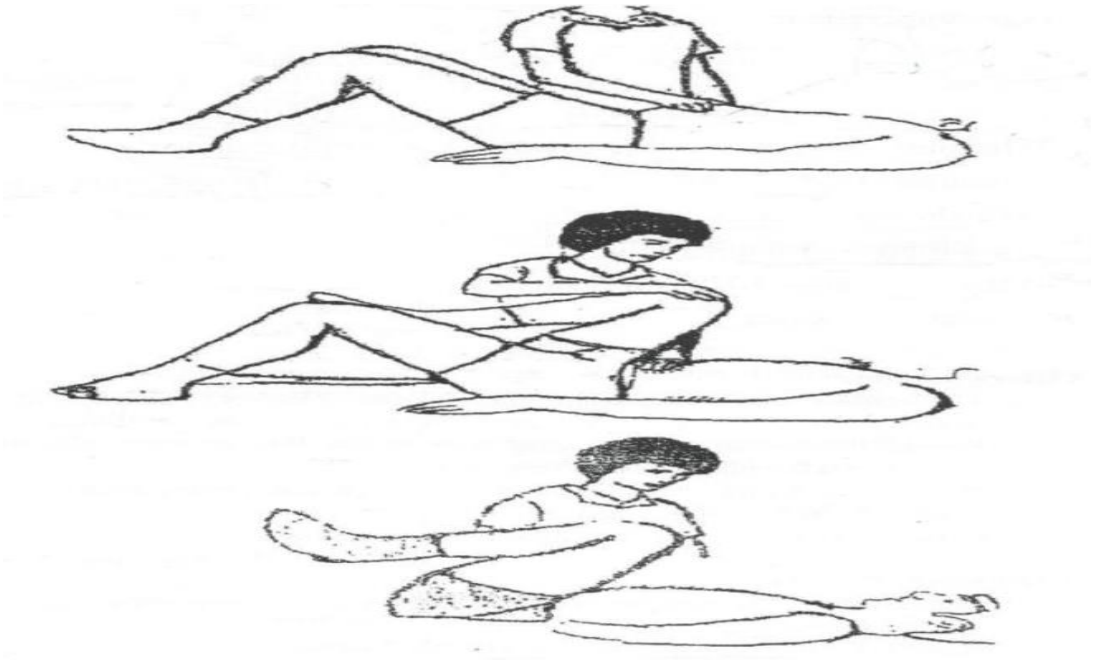
3. To mobilize the upper chest and shoulders.

With patient sitting in a chair, have him or her reach with both arms over head (180 degrees bilateral shoulder flexion and slight abduction) during inspiration. Then have the patient bend forward at the hips and reach for the floor during expiration.



4. To increase expiration during deep breathing.

Have the patient breathe in while in a hook lying position (hips and knees are slightly flexed). Then instruct the patient to pull both knees to the chest (one at a time to protect the low back) during expiration (picture). This pushes the abdominal contents superiorly into the diaphragm to assist with expiration.



Airway clearance techniques

Cough

Definition:

A cough is defense mechanism of the lungs and the primary means of clearing the first six to seven generations of airways of excess secretions and foreign material.

Coughs can be initiated voluntarily or can occur spontaneously, being triggered reflexively by mechanical stimulation of the larynx or by increased mucociliary stimulation by excessive secretions.

An effective cough:

Consists of a deep inspiration followed by closure of the glottis and a momentary hold during which contraction of the abdominal muscles increases intrathoracic and intra-abdominal pressures. The glottis then opens and forceful abdominal contraction produces expulsion of the trapped air.

Huffing, also called forced expiration, is an effective alternative for cough but is done with the same manner of cough without closure of glottis.

Common causes of ineffective cough:

Include respiratory muscle weakness or paralysis, uncoordinated muscular effort, abnormal thoracic configuration reducing mechanical efficiency (e.g., COPD and thoracic deformity), depression of the central nervous system, pain, and fear. An impaired cough results in retained secretions and bronchial obstruction which can lead to atelectasis or pneumonia and other problems.

Cough reflex:

- **Receptor** → vagus + trigeminal + glossopharangeal
- **Center** → medulla
- **Efferent** → pherenic + intercostal
-

A variety of techniques and devices can be used to assist with the mobilization and expulsion of bronchial secretions. Patients most likely to benefit from secretion clearance methods are those with objective signs of secretion retention.

Conventional chest physical therapy treatments consisting of postural drainage, percussion, and vibration or shaking.

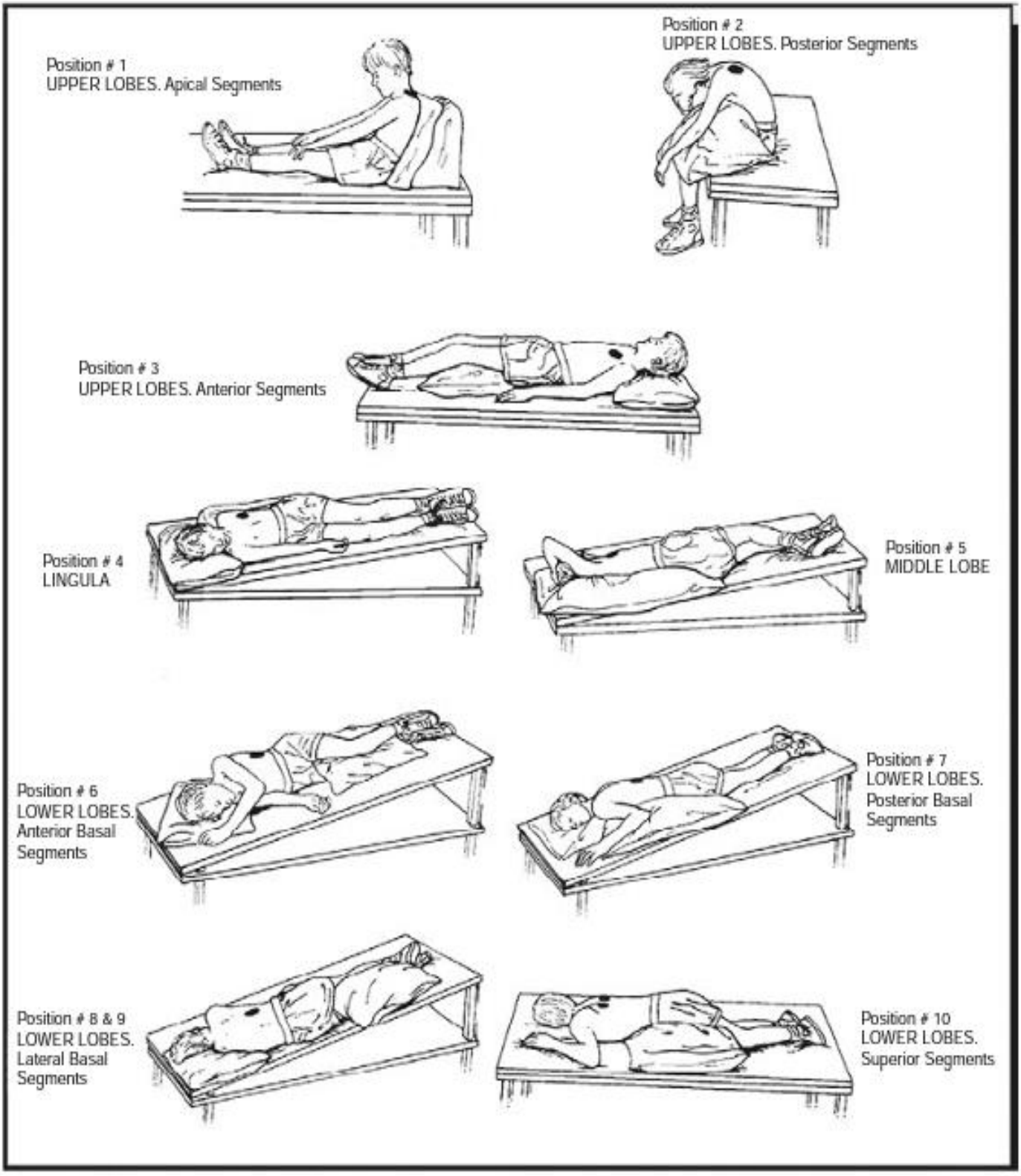
Postural drainage

Postural, drainage consists of positioning the patient according to bronchopulmonary anatomy so that each lung segment is placed upside down with its bronchus perpendicular to be drained by effect of gravity. The goal is to facilitate drainage of secretions into the segmental bronchus, from which they can be removed by coughing or suctioning.

Precautions and contraindications related to postural drainage:

1. Hemorrhage (severe hemoptysis)
    - Copious amounts of blood in the sputum.
    - Note: This is different from lightly blood–streaked sputum.
2. Untreated acute conditions
    - Severe pulmonary edema.
    - Congestive heart failure.
    - Large pleural effusion.
    - Pulmonary embolism.
    - Pneumothorax.
3. Cardiovascular instability
    - Cardiac arrhythmia, unstable angina.
    - Severe hypertension or hypotension.
    - Recent myocardial infarction.
4. Recent neurosurgery.

    The head-down position increases intracranial pressure.



Bronchial drainage positions with treatment areas marked for percussion

The procedures:

- Explain the treatment to the patient and ask the patient to loosen any tight or binding clothing.
- Observe any tubes or other equipment connected to the patient, making sure everything has enough slack to allow the positional change without pulling taut or dislodging. Make any required adjustments.
- Check the pulse, BP, oxygen saturation, respiratory rate, and pain level, as able, before positioning any patient who is critically ill or possibly unstable. Monitor the patient during treatment.
- If the patient has excessive secretions, have her/him cough or perform suctioning before positioning. Simply moving the patient into the postural drainage position may induce a productive cough.

- Place the patient in the proper, or modified, position, with assistance from other staff if necessary. Watch for any signs of intolerance.
- Maintain position for 5 to 20 minutes, depending on the quantity and tenacity of secretions and patient tolerance.
- Have the patient cough or perform suctioning before changing positions.
- If several positions are being used, it is best to limit total treatment time to 30 to 40 minutes because of the stress placed on the patient. Always treat the most critical areas first.
- Encourage the patient to cough periodically after treatment, as some secretions may take 30 to 60 minutes to clear.
- Patients should never be left in the head-down position unsupervised unless they are alert and independent in functional mobility, so that they can reposition themselves or call for assistance if needed.
- Better times is early morning and before sleep at night, and during the day as needed.

- Manual techniques, such as percussion and vibration, are usually applied during bronchial drainage to facilitate the mobilization of secretions.

Manual techniques used to assist postural drainage therapy:

In addition to the use of body positioning, deep breathing and effective cough to facilitate clearance of secretions from the airways, a variety of manual techniques are used in conjunction with postural drainage to maximize the effectiveness of the mucociliary transport system. They include:

1. Percussion

- This technique is used to further mobilize secretions by mechanically dislodging viscous or adherent mucus from the lungs.
- Percussion is performed with cupped hands over the lung segment being drained. The therapist's cupped hands alternately strike the patient's chest wall in a rhythmic fashion. The therapist should try to keep shoulders, elbows, and wrists loose and mobile during the maneuver. Mechanical percussion is an alternative to manual percussion techniques.

- Percussion is continued for several minutes or until the patient needs to alter position to cough.
- This procedure should not be painful or uncomfortable. To prevent irritation to sensitive skin, have the patient wear a lightweight gown or shirt. Avoid percussion over breast tissue in women and over bony prominences.
- Relative contraindications prior to implementing, percussion in a postural drainage program, the therapist must compare the potential benefits with the possible risks to the patient. In most instances, avoid the use of percussion.
    - Over fractures, spinal fusion, or osteoporosis.
    - Over tumor area.
    - If a patient has a pulmonary embolus.

- If a patient has a condition in which hemorrhage could easily occur, such as in the presence of a low platelet count, or if a patient is receiving anticoagulation therapy.
- If a patient has unstable angina.
- If a patient has chest wall pain, for example, after thoracic surgery.

2. Vibration

- The technique is used in conjunction with percussion in postural drainage. It is applied only during expiration as the patient is deep breathing to move the secretions to the larger airways.
- Vibration is applied by placing both hands directly on the skin and over the chest wall (or one hand on top of the other) and gently compressing and rapidly vibrating the chest wall as the patient breathes out .
- Pressure is applied in the same direction as that in which the chest is moving.
- The vibrating action is achieved by the therapist's isometrically contracting (tensing) the muscles of the upper extremities from shoulders to hands.

3. Shaking

- Shaking is a more vigorous form of vibration applied during exhalation using an intermittent bouncing maneuver coupled with wide movements of the therapist's hands.
- The therapist's thumb are locked together and the open hands are placed directly on the patient's skin and fingers are wrapped around the chest wall. The therapist simultaneously compresses and shakes the chest wall.

Pulmonary rehabilitation

Introduction

Pulmonary rehabilitation should be integral to the management of people with chronic respiratory disability, not an optional extra. The need is greater now that patients are being discharged from hospital 'quicker and sicker'.

It is rewarding because it can improve independence for people with inactivity and helplessness.

Rehabilitation does not reverse lung damage but it modifies the disability that derives from it and normally shows greater benefit than medication. Participants report a sense of well-being due to gaining control over symptoms, especially the fear of breathlessness.

Definition:

Pulmonary rehabilitation is an individually tailored, multidisciplinary program of care for patients with chronic respiratory impairment that is designed to optimize physical and social performance and autonomy and aims to control and alleviate symptoms, reduce disability, improve physical function and health-related quality of life, and possibly reduce overall health care services utilization.

The components of a pulmonary rehabilitation program:

- Patient assessment
- Patient and family education and training regarding the disease process, exacerbating factors and management strategies (smoking cessation, proper nutrition for weight loss or gain, stress management, environmental modifications, and flu and pneumonia immunizations), symptom management, and treatment interventions
- Optimal disease management (medications, bronchial hygiene treatments, and supplemental oxygen)
- Individually prescribed, medically supervised aerobic and resistance exercise training, flexibility exercises

- Nutrition counseling
- Psychosocial support and counseling
- Promotion of long-term adherence

Benefits of pulmonary rehabilitation:

- ↓ Breathlessness by 65%.
- ↑ Exercise capacity and quality of life, even for severely impaired patients.
- ↓ Health care costs, with reduced hospitalization.
- ↑Ability to wean from mechanical ventilation.
- Improvement of quality of life.

The team:

Potential team members should be involved:

- Physiotherapist
- Occupational therapist
- Respiratory nurse
- Physician
- Dietician
- Clinical psychologist or social worker
- One of whom is the co-ordinator.

Participants:

A programme includes about eight to 10 participants, with stratification for mild, moderate and severe disease.

Suggested selection criteria are the following:

- Breathlessness limiting activity.

- Motivation towards self-help and lifestyle change.
- Stable condition.
- Optimal medical management.
- Adequate ability to hear or communicate.
- Ability to attend the full programme.

Inclusion criteria:

COPD, asthma, cystic fibrosis, interstitial or other restrictive disease, neuromuscular disorders, following surgery or long-term hospitalization. People with restrictive lung disease show benefit in the early stages, although the following precautions are advisable:

- For interstitial disease, monitoring for hypoxaemia during exercise
- For neuromuscular disease, preventing overuse of compensating muscles.

The set up:

The options are:

- An outpatient programme.
- An inpatient programme in a dedicated rehabilitation ward.
- A discharge programme after exacerbation, either in a pre-discharge ward or at home.
- A home-based programme, useful for severely disabled people or as a cost-effective alternative to hospitalization for mild exacerbations.
- A community-based programme in a day centre, physiotherapy practice or other facility that has single-story access and a more upbeat atmosphere than hospital.

Resources:

The following are needed for a programme based in a hospital or day centre:

- Large, warm room with easily opened windows, cheerful atmosphere, wall space and

- non-slip floor, free from dust-collecting furniture, and with acoustics that can cope with choruses of coughing.
- Comfortable upright chairs.
- Treadmill, exercise bike, trampoline, quoits, weights, stretchy bands, springs and other gym equipment.
- Steps.
- Rolla tors and high walking frame.
- Full length mirror.
- Fan.
- Demonstration inhalers.
- Oxygen.
- Oximeter.
- Nebulizer system and drugs.
- Sputum pots and tissues.
- Audiovisual teaching aids.
- Handouts, exercise booklets, diaries, writing materials.
- Name labels to encourage group interaction.
- Refreshments.
- Crash trolley and team members trained iii life support.
- Individual transport arrangements such as taxis or cars to avoid the stress and delays of public or ambulance transport.

Structure and timing:

The session time: around an hour.

Frequency of sessions: twice weekly for 6-12 weeks, during daylight hours and avoid early mornings or rush hour travel.

The program of the session: education on a specified topic, a break for socializing, an exercise session and relaxation.

The supervision of physiotherapy session: is a one-to-one basis or with half the group at a time,

The session is formed of:

Assessment, identification of participant needs, goal setting, breathing re-education and a suggested home programme.

Assessment

Assessment should take account of:

- Respiratory impairment: ↓ lung function, e.g. Fev_1
- Respiratory disability: the effect of this impairment, e.g. Anxiety or ↓ exercise capacity
- Respiratory handicap: social and other disadvantages.

There is much variation between degree of impairment and an individual's disability or handicap. Rehabilitation is aimed at symptoms rather than the disease process.

Contraindications:

Acute disease, symptomatic angina, recent embolism or myocardial infarct, second or third-degree heart block, deep vein thrombosis and resting systolic BP above 240 mmHg or diastolic above 120 mmHg.

Relative contraindications:

Disabling stroke or arthritis, haemoptysis (depending on the cause), metastatic cancer, unstable asthma, resting heart rate (HR) below 100 and resting systolic pressure above 180 mmHg or diastolic above 95 mmHg.

Contact the physician if $PaCO_2$ is above 8 kPa (60mmHg).

People with insulin-dependent diabetes benefit from exercise training, which can also improve glucose tolerance.

Steroid induced osteoporosis is not a contraindication and indeed is an indication for sensible weight bearing exercise.

People with heart failure usually benefit from exercise training: those with mild disease may

take longer to recover from activity and those with more advanced disease require a low-intensity programme.

People with intermittent claudication can improve their walking distance.

The following drug history is relevant:

•Drugs such as beta-blockers render the BP and pulse unreliable for monitoring purposes.

•If prescribed and indicated, bronchodilators and anti-angina drugs should be taken before exercise

•Steroids should be at the lowest effective dose to minimize muscle weakness.

Respiratory function tests:

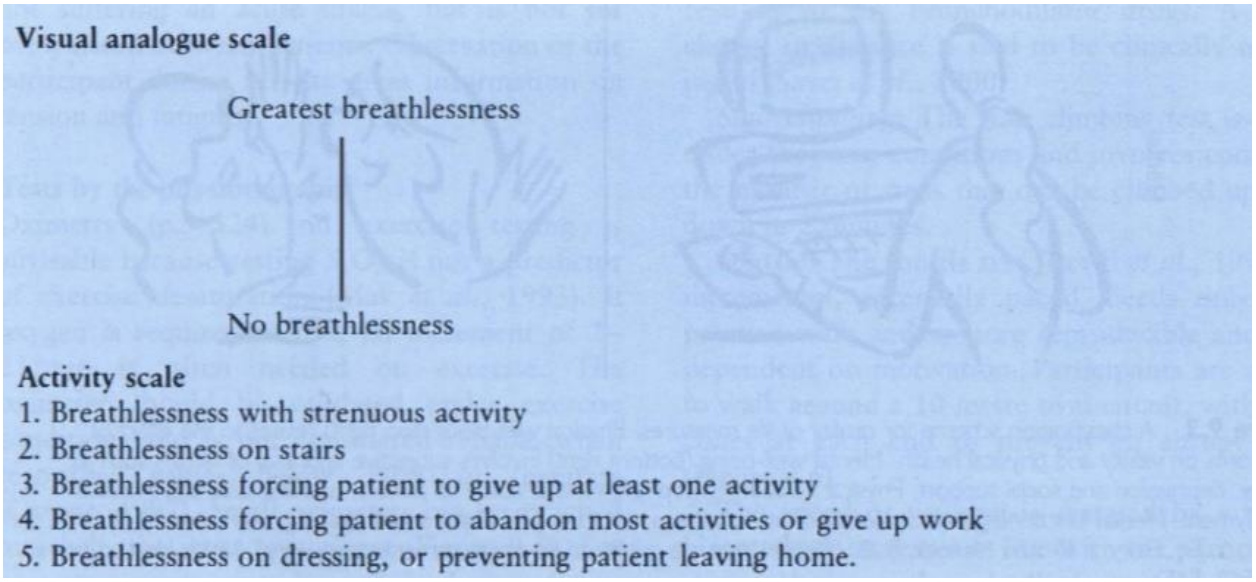
RFTs are generally unhelpful for outcome measurements. FEV_1 does not relate to disability, but it helps distinguish COPD from asthma, and may assist stratification, e.g.:

- FEV 1 > 60% predicted indicates mild COPD: aerobic programme suitable.
- FEV 1 40-60% predicted indicates moderate COPD: peripheral muscle strengthening and use of multigym suitable.
- FEV 1 < 40% predicted indicates severe COPD: isolated muscle group work and range of movement suitable.

Breathlessness and quality of life:

It is helpful to ask participants how breathlessness affects their lives and why they think they are breathless. Visual analogue scales help patients to quantify the symptom. It is easier for participants when they are vertical rather than horizontal. Breathlessness must be explained to participants so that they distinguish it from sensations such as fatigue or chest tightness.

Visual analogue scale



Activity scale

1. Breathlessness with strenuous activity
2. Breathlessness on stairs
3. Breathlessness forcing patient to give up at least one activity
4. Breathlessness forcing patient to abandon most activities or give up work
5. Breathlessness on dressing, or preventing patient leaving home.

Health-related quality of life (QoL) scales characterize wellbeing and include the effect of deconditioning caused by a lifestyle restricted to minimize breathlessness. Also known as health status or functional status scales, QoL scales can be generic or disease-specific.

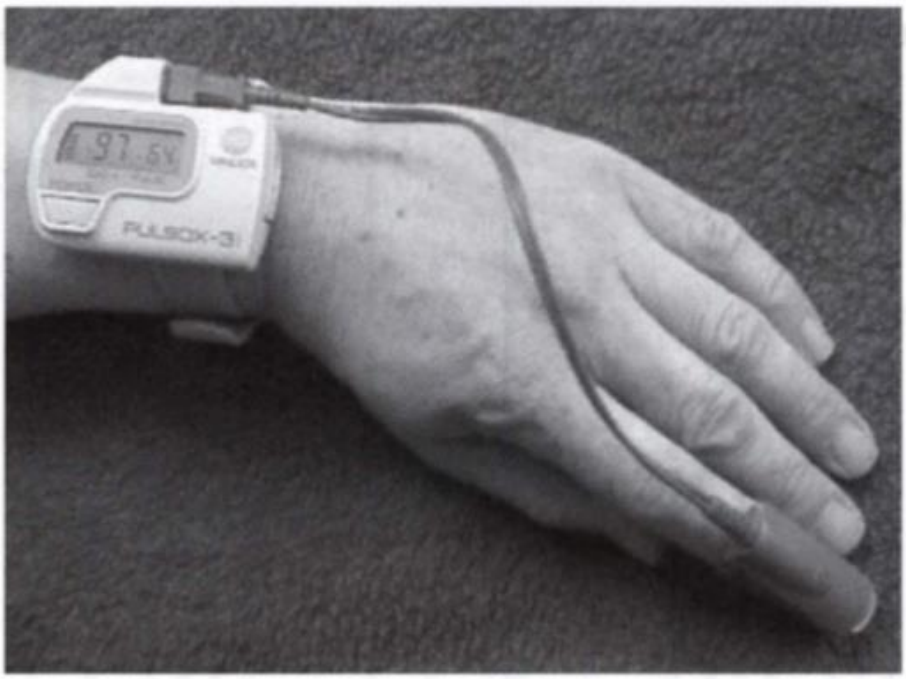
Exercise testing:

Exercise testing can be measured objectively by walking or stair-climbing. This gives an accurate indication of progress so long as the patient is not suffering an acute illness, but is not for comparison between patients. Observation of the participant during activity gives information on tension and fatigue.

Tests by the physiotherapist:

Oximetry on exercise testing is advisable because resting SaO_2 is not a predictor of exercise

desaturation. If oxygen is required at rest, an increment of 1- 2 L/min is often needed on exercise. The oximeter should be validated under exercise conditions and is not considered reliable when recording values below 90% on exercise. Small oximeters can be attached to a belt, the wrist or a finger. Ear oximeters may not be valid during heavy



Oxygen prescription:

Is based on the flow required to maintain SaO_2 at over 91 % during the last 30 seconds of a 5-minute corridor walk. Transient desaturation is acceptable. For severely impaired patients, a brief test such as sit-to-stand or stand-up-and-go can be used. For patients who are able, the following are available.

Six-minute distance:

For endurance testing, participants are asked to walk for 6 minutes as fast as reasonably possible along a measured flat corridor, following standardized instructions. Stopping to rest is allowed but included within the 6 minutes. Participants should feel at the end that they have performed to their maximum capacity. The physiotherapist can inform the participant when each minute is completed but should not walk alongside because this might influence his/her speed. The data to record are the 6-minute distance, symptoms, HR and SaO_2. Modifications are required if there are orthopaedic or neurological problems. Three or four practice walks are needed, with 20 minute rests in between. Repeat tests should be performed at the same time in relation to any bronchodilator drugs. A 15% change in distance is said to be clinically meaningful.

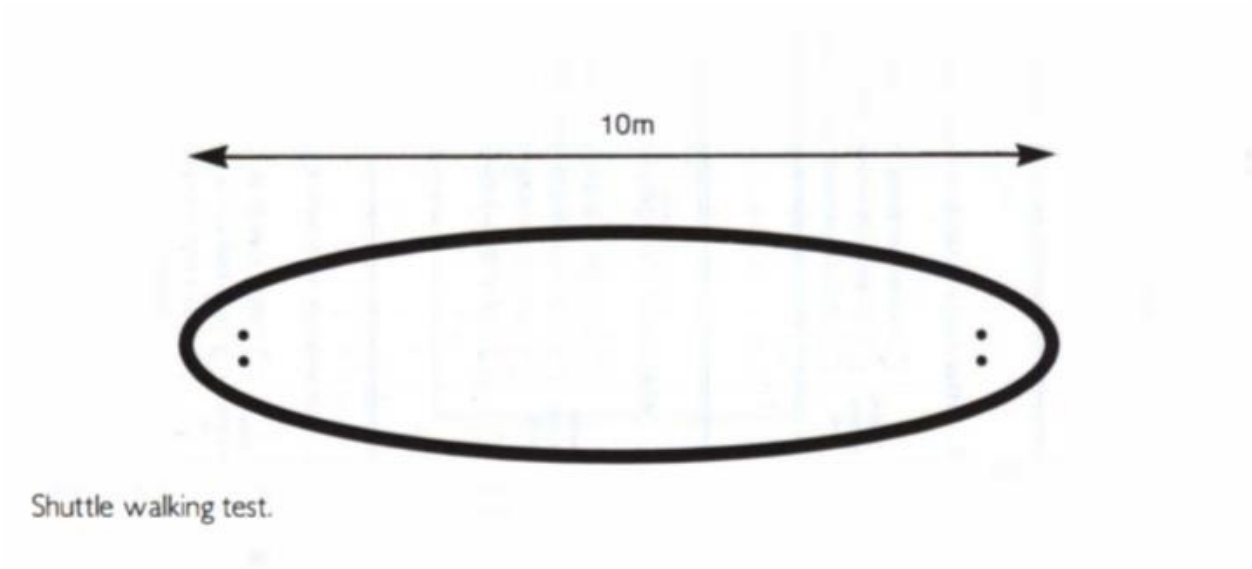
Stair climbing:

The stair climbing test is done under the same conditions and involves counting the number of steps that can be climbed up and down in 2 minutes.

Shuttle:

The shuttle test is incremental, externally paced, needs only one practice walk and is more reproducible and less dependent on motivation. Participants are asked to walk around a 10-metre oval circuit, with two cones at each end to prevent an abrupt turn. The speed of walking is dictated by a taped bleep which increases in line with the participant raising their speed gradually from 1 to 5 miles per hour. The tape (Appendix C) gives standardized instructions,

and no verbal encouragement is given. The physiotherapist walks alongside for the first minute to discourage the participant from exceeding the initial speed. Thereafter, if the cone is reached early the participant waits for the beep before continuing. The end point is when symptoms prevent the participant completing a circuit in the time allowed. Ideally the maximum should be reached within 10-15 minutes. Half-an-hour's rest is needed after the practice test. The shuttle is closely related to maximum oxygen consumption (VO_2 max). Some participants have trouble coordinating the cones with the beep.



Shuttle walking test.

Tests in the laboratory:

Exercise testing based on treadmill-walking or cycle ergometry is unfamiliar to participants, unreliable in relation to everyday activity and less related to exercise capacity than breathlessness. However, measurable workloads can be imposed in the laboratory while monitoring minute ventilation, CO_2 output, HR, BP, SaO_2, blood gases and oxygen consumption (VO_2). This helps to highlight the interaction between vanous systems involved in oxygen delivery to the tissues.

An ECG stress test detects myocardial ischaemia by identifying ST segment changes.

Exercise testing can help determine the cause of exercise limitation. Respiratory disease is likely if breathlessness is the limiting factor. If a person reaches the anaerobic threshold early,

i.e. at less than 40% predicted VO_2max, or if maximum predicted HR is reached early, limitation is probably due to cardiovascular disease.

Education:

Is the most cost-effective aspect of rehabilitation. It increases participants' confidence and reduces uncertainty and fear, whereas ignorance can breed a catastrophizing of life events. Most participants want to know as much as possible about their condition but are often reluctant to ask questions in a clinical setting. A rehabilitation programme provides time and a non-threatening atmosphere for discussion. Age does not itself hinder intellectual ability, but elderly participants may need time for processing information, and hypoxaemia may impair memory.

Retention of information is optimal if:

- the room is free of distractions
- the teaching plan is set out clearly
- the most important points are made first
- teaching sessions are brief
- language is simple and jargon-free
- advice is specific rather than general
- information is reinforced regularly throughout the programme
- booklets and handouts are included.
- participants are reminded to check body tension, take medication and practise their breathing: suggestions include memory aids such as stickers on kettles, reminders on toothbrushes, use of dead time such as queues or TV advertisements.

The respiratory nurse teaches the understanding and practical management of oxygen and medication.

The dietician identifies individual nutrition problems, suggests six-meal-a-day menus, advises on healthy eating and explains which foods are mucus-forming, gas-forming, constipating or hard to digest.

The physician answers medical questions and discusses advance directives in relation to life-prolonging treatment.

Participants are reminded that rehabilitation is not a course of treatment to make them better but more of a life plan.

Educational topics include:

- How we breathe, the relation between symptoms and pathology, the nature of breathlessness.
- Medical tests, procedures, interpretation of results.
- Oxygen therapy: effects, side effects, equipment, consequences of non-adherence Drug therapy: effects, side effects, inhalers and nebulizers, consequences of non-adherence, records and charts to aid memory.
- Fluids and nutrition.
- Smoking cessation.
- Relation between symptoms and interventions such as relaxation, breathing re-education, chest clearance, exercise training and energy conservation.
- Self-assessment, symptom management, recognition and management of exacerbations, recognition of the need for medical attention, e.g. change in symptoms or new symptoms.
- Prevention of infection, e.g. avoidance of people with respiratory infections, influenza vaccination.
- Management of the environment, e.g. indoor exercise if outside air is polluted, covering nose and mouth when exercising in cold weather, bowls of water by radiators, prevention of dust.

- Community resources, benefits and entitlements (with corresponding advice to welfare agencies on the needs of the 'invisible' respiratory patient).
- Vocational guidance to improve self-esteem and social participation while avoiding jobs or hobbies with respiratory irritants or excess energy expenditure.
- Tips such as a walking stick to advise motorists of a slow walking pace, advice to carers on simple massage techniques.
- Advice for participants before visiting the doctor, e.g. write down questions to ask in advance, clarify points that are not understood.
- Management of panic attacks.
- Home equipment.
- Travel tips, including use of nebulizers and oxygen abroad and while travelling.

Motivation:

Motivation is the best predictor of the success of rehabilitation. Over 70% of patients with COPD do not adhere to treatment, which may be because of inadequate information or depression.

Motivation is essential if participants are to practise at home. A twice-a-week programme of structured exercise is not enough by itself to improve exercise tolerance. Participants are unlikely to ignore their own beliefs and goals in order to follow a prescriptive approach, and education is not achieved by simply feeding information into an empty vessel and pressing the right buttons. Motivation is enhanced by participants taking responsibility for their own management.

Factors that increase motivation are:

- clear advance information in large print
- realistic expectations
- active participation, e.g. self-monitoring, invitations to question, comment, design programmes, contribute ideas
- verbal commitment from participants

- praise, warmth, humour, honesty and responsiveness from the rehabilitation team
- family involvement
- focus on health rather than disease
- short simple regimes
- understanding the rationale of each component
- early success, reinforced by progress charts
- access to notes
- continuity of personnel
- certificate of completion.

Factors that decrease motivation are:

- fatigue
- fear of failure
- anxiety or depression
- advice that is inconvenient or difficult to follow
- embarrassment
- boredom, e.g. repetitive exercise, 12-minute walking test, waiting for transport
- coercion
- lack of recognition of the individual as a whole.

Smoking cessation:

Is the first priority in the management of people with COPD. Some 70% of smokers want to give up but their endeavours are hampered by the tenaciously addictive properties of nicotine, as well as less specific obstacles such as comradeship amongst smokers, stress or boredom. Multiple reinforcements are more successful than a single intervention, and every strategy for encouragement should be employed because failure drains the will. A quit date should be decided, preferably when something unusual is happening and with a reward for success at the end. Goals can be set, low priority cigarettes stopped, a diary initiated, habits linked with

smoking can be changed, e.g. orange juice instead of coffee and avoidance of passing the cigarette shop.

Alternative strategies can be devised for difficult situations or in case of relapse. Although total cessation is best, reduced levels of smoking can be an alternative so long as compensatory deep inhalation is avoided.

Plenty of water is advised, and distraction from cravings might include:

- Sucking mints, chewing gum or dried fruit, eating an apple
- Brushing teeth, taking a shower
- Phoning a friend or helpline.

A pre-planned phone call to the participant is helpful a few days after the quit date, when motivation, determination and support from others might be waning.

Advice about the effects of tobacco on the smoker's family, suggestions on alternative uses of the £1000 per year spent on the other good habits, and the fact that, while smoking appears to help clear the chest, it only does so by irritating the airways and creating extra secretions.

Participants need to understand the physical and psychological difficulties of withdrawal but also the pleasures of sweeter-smelling breath and clothes, improved appetite and bank balance, reduced cough and even some recovery of lung function.

Temporary irritability can strain relationships but should be understood as the body recovering rather than a reason to return to smoking.

Nicotine replacement can double success rates. Up to two patches can be used over 24 hours.

A variety of drugs are available to assist withdrawal.

Changing to 'light' cigarettes is normally unhelpful because of altered smoking patterns.

Herbal cigarettes contain no nicotine but produce tar and carbon monoxide.

Other tips include posters, acupuncture, hypnotherapy, biofeedback by carbon monoxide monitoring, group counselling and role play, e.g. asking friends not to smoke around them. Participants themselves provide ideas for each other and can set up a 'buddy system' by swapping phone numbers. Positive support helps counteract patients' previous experience of being treated as if they should be punished for smoking (maybe some of this punitive energy could be directed at the tobacco companies!).

Reduction in breathlessness

Many techniques variety of resting positions, relaxation, breathing control, mobilization of the thorax.

Thoracic mobility:

Thoracic mobility may be impaired by chronic muscle tension, shortening of anterior chest and shoulder muscles, abnormal mechanics of breathing and a forward head posture. This can add a restrictive element to an obstructive condition and cause pain, sometimes misdiagnosed as pleurisy.

Beneficial effects of thoracic mobility:

- In forward-lean-sitting: Maitland mobilizations to vertebral and scapular joints
- Sitting astride a chair to fix the pelvis: passive thoracic rotation
- Half-lying with a roll under the thorax: thoracic extension assisted by passive arm elevation
- For some participants who have developed a stiff hyperinflated chest: manual compression on exhalation in a bucket-handle direction
- Stretches to muscles around the shoulder.

Flexibility exercise to mobilize the chest:

1. Head movements

- While breathing in, look up to ceiling. While breathing out, slowly bring chin down to

chest.

- Keeping shoulders still, move your head sideways to bring your ear towards your shoulder. Repeat the other side.
- Turn your head to look over your shoulder. Repeat the other side. Maintain steady breathing throughout.

2. Shoulder girdle circling: Circle shoulders slowly forward, upward, backward, downward. Relax. Repeat m opposite direction .
3. Chest stretch: Hands behind back, breathe in, push chest forward and shoulders back. Breathe out and relax.
4. Back stretch: Lock hands, stretch hands forward at shoulder level, feel stretch between shoulder blades, relax.
5. Arm circling: Hold arm sideways at shoulder height, circle arm in progressively increasing circles for count of 4, then decrease for count of 4. Repeat with other arm. Maintain relaxed breathing throughout.
6. Trunk rotation: With arms folded across chest, keeping pelvis still and knees forward, breathe in. While breathing out, rotate trunk to look over shoulder. Repeat other side. Maintain steady breathing.
7. Trunk rotation: As above, with hands behind head.
8. Trunk extension: With hands behind head, lean over back of (low-backed) chair while breathing in. Return while breathing out.
9. Trunk side-flexion: With hands across chest or behind head, and keeping buttocks flat on the chair, bend from side to side.
10. Trunk flexion: Breathe in gently. While breathing out, bend forward towards toes. Sit up slowly while breathing in.
11. Pelvic circling: Standing with hands on hips: rotate pelvis in slow circle.
12. Calf stretch: Standing with one foot in front of the other: lean forward and bend front leg, keeping back heel down. Repeat with other leg.
13. Pectoral stretch: Standing holding the inside of a door frame: while breathing in, step

through frame with one foot and feel stretch across front of chest. While breathing out, step back. Repeat with other foot.

14. Arms up: Standing with elbows straight: while breathing in, lift rolled-up newspaper above head in one hand, pass to other hand. While breathing out, bring arms down.

Exercise training:

Pulmonary rehabilitation aims at endurance, flexibility and some strength, which emphasize factors such as nutrition and physical fitness, rather than maximal capacity, which depends on ventilation, gas exchange and oxygen delivery to the respiratory muscles.

The programme must be individually planned, acceptable to the participant, accessible, safe, show tangible benefits and be designed so that it can be maintained unsupervised at home. Longterm commitment is needed because de-training occurs faster than training.

The benefits of exercise:

- Respiratory patients who train have shown improved exercise tolerance, cardiovascular fitness and raised anaerobic threshold. Maximal oxygen consumption (VO_2max) can be increased in people with less severe disease, and improved muscle strength can be comparable to that in healthy young people. Prior deconditioning means that modest exercise is likely to induce a physiological training effect, even in elderly people.
- Breathlessness is relieved by improved exercise tolerance, activity-related sensory input and reduced gas trapping because of freer airflow, represented by a minor 7% improvement in FEV_1. The distress component of breathlessness decreases more than the intensity component, showing how desensitization and reduced anxiety have a direct effect on the perception of breathlessness.
- A sense of well-being and confidence, along with reduced anxiety and depression, is consistently reported and is greater than objective change.
- Exercise reduces smoking, BP and risk of chest infection.
- It promotes relaxation and sleep, stabilizes blood sugar and reduces gut problems.

Mechanism of training:

The severity of disease dictates how training improves exercise tolerance.

People with moderate COPD (FEV_1 > 1.2L) can reach their anaerobic threshold and develop lactic acidosis, which occurs at a higher percentage of VO_2 max (e.g. 80-90% versus 60-70% in those with normal lungs).

In severe COPD (FEV_1 < 1.2 L), exercise is commonly limited by ventilatory function and gas exchange abnormalities, and improved exercise tolerance is thought to be due to greater mechanical skill, which reduces the oxygen cost of exercise, a more efficient ventilatory pattern, and desensitization to breathlessness. Major improvements in endurance with unchanged cardiorespiratory fitness, and suggested a further mechanism of improved neuromuscular coupling. People with COPD generate lactic acidosis at low exercise levels. Hypercapnic patients may find it particularly difficult to work above the anaerobic threshold because CO_2 is generated by lactic acid buffering, and fatigue may be caused by the excess ventilation required to compensate for metabolic acidosis.

Technique:

Even when a conventional training response is not anticipated, the three principles of training are followed:

- Overload, i.e. Intensity must be greater than the muscle's normal load
- Reversibility, i.e. Cessation of training loses the benefit gained
- Specificity, i.e. Only the specific activities practiced will show improvement.

Endurance training, comprising low-resistance, high-repetition exercise, is more suitable for respiratory patients than strength training. Endurance training forestalls the onset of inefficient anaerobic metabolism and enhances the use of oxygen.

Exercise prescription:

Four components make up the exercise prescription: mode, intensity, duration and frequency.

The mode of exercise relates to the participants' lifestyles. Many choose walking, stair climbing or occupation-based exercise. Some prefer the stationary bike or treadmill because they feel in control, can use oxygen easily and have support for their shoulder girdle. About 85% of body weight is supported by a bike, and large muscle groups can be exercised with less strain than walking. Treadmill-walking enables participants to learn the feel of different speeds, so that they can structure their home programme. Other participants enjoy simple activities that can be continued at home, such as chair exercises. Low intensity individual limb exercises are well tolerated and translate into improved whole body exercise capacity. Upper limb exercise needs to be included, and hobbies such as bowls are both enjoyable and useful for chest mobility.

Circuit training can involve six to ten exercise stations, choosing from examples in Box below. Exercises are best alternated between upper/ lower limb exercises, and easy/difficult exercises. Gentle progressive arm exercises reduce the breathlessness associated with upper limb activities, with a carry-over effect on the respiratory muscles that can be equivalent to inspiratory muscle training. Unsupported arm exercise should be included unless this causes abdominal paradox. Loss of shoulder girdle support forces the intercostal and accessory muscles to stabilize the arms and torso, which shifts the breathing load to the diaphragm, creating a challenge for people with COPD.

Each exercise is continued for 1 minute. Repetitions are recorded by participants on individual clipboards. Participants rest between each exercise at 'breathing control stations' until breathlessness returns to baseline, usually in about 1 minute. Instructions are best pinned to the wall.

1. **Knee tensing.** Long-sitting on plinth, bed or sofa with knees on a pillow or coffee jar: tighten thigh muscles, hold for count of 4, relax. Repeat with other leg. Maintain steady breathing throughout.
2. **Biceps curl.** Sitting with elbows on table: lift weight or bag of sugar, lower slowly. Repeat with other arm. Maintain steady breathing throughout.
3. **Heel–toe.** In sitting, raise alternate heels and toes, in time with breathing.
4. **Ball throwing and catching**
5. **Quadriceps exercises.** In sitting, straighten one knee, hold for count of 4, lower leg slowly. Repeat with other leg.
6. **Towel wringing.** In sitting, wring towel tightly, hold for count of 4. Slowly untwist towel. Keep breathing steadily throughout.
7. **Lift ups.** In sitting, inhale gently. While breathing out, push down with both hands and lift pelvis off seat. While breathing in, let yourself down slowly.
8. **Step ups.** Breathe out, step up with one foot. Breathe in, bring up other foot. Step down with one foot, then the other.
9. **Walking sideways.**
10. **Wall press-ups.** Stand with feet a comfortable distance from the wall, put hands on wall, bend at elbow (keeping heels on floor), push arms straight again.
11. **Abdominal contractions.** In sitting, pull in abdominal muscles, relax and breathe.
12. **Static bike, hula-hoops, trampet.**
13. **Bounce ball off wall.**
14. **Calf exercise.** Holding back of chair, go up on toes, return heels to floor.
15. **High knee marching.** Holding back of chair with one hand, march on the spot, lifting knees high.
16. **Arm raise.** Sitting or standing, raise arm, with or without weight, above head. Lower slowly. Repeat with other arm.
17. **Bend down, stretch up.** In time with breathing.
18. **Sit to stand.** Using dining room chair, sit–stand–sit. Repeat, holding a ball.

The intensity of exercise: Some methods are described below.

1. The predicted maximum HR can either be estimated as (220 – age) or measured during an incremental stress test. Exercise is traditionally maintained at 70% of maximum HR, but respiratory patients can achieve a training effect at 30-40% of

maximum. HR is linearly related to VO_2max, so the pulse can be taken at, say, 50% of V02max and used as a target.

2. Multiples of V02max are expressed as METs. These complicated methods of monitoring exercise intensity are widely described but may not be suitable because:

- Many respiratory patients are too breathless to reach true maximal HR or V02max
- HR is affected by cardiorespiratory drugs such as beta-blockers, digoxin and salbutamol
- Even people with normal lungs show a wide variation in HR and V02 max.

3. Breathlessness scales can be kept on clipboards for participants to assess their perception of breathlessness (Borg shortness of breath scale). Exercise can be increased gradually while maintaining breathlessness at a constant tolerable level. Breathlessness scales are reproducible, correlate with physiological measures of exercise intensity and even in people with normal lungs have been shown to result in greater improvement in endurance than when using HR. It is thought safer for the participant to choose the level of breathlessness rather than the physiotherapist.

a) Borg shortness of breath scale, which assigns numerical values to degrees of breathlessness. The level chosen by the participant is maintained while the amount of exercise gradually increases.

0	–	Nothing at all
0.5	–	Very very slight
1	–	Very slight
2	–	Slight
3	–	Moderate
4	–	Somewhat severe
5	–	Severe
6		
7	–	Very severe
8		
9	–	Very very severe
10	–	Maximal

b) Breathlessness rating scale. The participant chooses to work at rating 2,3 or 4.

Rating 1	Comfortable breathing throughout
Rating 2	*During*: deeper breathing *After*: recovery 2–5 min *Day after*: comfortable
Rating 3	*During*: harder breathing *After*: recovery 4–7 min *Day after*: comfortable
Rating 4	*During*: breathless but not speechless *After*: recovery 5–10 min *Day after*: not tired
Rating 5	*During*: breathless and speechless *After*: recovery > 10 min *Day after*: tired

Participants achieve a moderate training response if they are able simply to exercise enough to achieve an increase in breathlessness at a constant tolerable level while avoiding distress or desaturation. This allows for the variation in intensity that suits different individuals. Some exercise gently while others can exercise safely at over 80% of their maximum.

The balance of duration and frequency depends on individual preference because the result is similar if total work is the same. Low intensity, unstructured programmes appear to be as beneficial as high-intensity, highly structured programmes.

The duration: Supervised training sessions usually last for 30-60 minutes but, for home practice, respiratory patients find it more acceptable to exercise for one or more short sessions a day with brief warm up and cool down periods.

Severely breathless people may prefer interval training, which alternates 15- to 30-second episodes of exercise with rest.

People who tend to rush at their exercise in an attempt to get it over quickly may find that counting breaths with their steps helps them to pace themselves in the early stages, e.g.: in/one, out/one, or in/one, out/one/two.

For others this disturbs their rhythm and distracts them from focusing on awareness of their breathing and level of effort. Stair-climbing may be more efficient if performed by inhaling as one leg is raised, exhaling as the body is raised and interspersing every few steps with a rest.

Cool down

Participants are asked to slow down their activity for a few minutes, e.g. by slow walking, to prevent sudden pooling of blood in the lower extremities.

Progression

Participants progress by increasing duration or frequency, usually in weekly increments. Intensity usually stays the same but some participants are happy to increase this. Improvement usually continues for 4-6 months and, when a plateau is reached, moderate exercise should be maintained at a minimum 15 minutes a day. An indoor programme is substituted when there is air pollution, wind or rain. Urban patients are advised to choose the least polluted times and places for training. A fixed routine at a regular time of day helps adherence to the programme. Progression includes adaptation to uneven terrain and any anticipated problems identified by

the patient. Figures 9.15 and 9.16 are suggested documentation to record results and monitor progress.

Once a week, participants should put themselves back on the same programme as on the final day of their training. If this is difficult, they have lost fitness and will need to increase their maintenance exercise. If training is interrupted by illness or holiday, the programme is restarted at a lower level. The difficult task of maintaining fitness needs follow-up encouragement from the rehabilitation team.

Inspiratory muscle training

The concept that strengthening the respiratory muscles would allow a patient to sustain a higher level of ventilation during exercise is an attractive one if this increased exercise tolerance, but does it work?

Question 1

Respiratory disease can make inspiratory muscles either weaker or stronger than normal. How?

Strong muscles develop by working against the resistance of obstructed airways or stiff lungs.

Weak muscles are due to:

- Poor nutation, in which case dietary management is indicated, which can improve inspiratory muscle strength by 40%.
- Inadequate oxygen delivery to the muscles because of heart failure or blood gas abnormalities, which may respond to fluid, drug and oxygen therapy
- Steroid-induced weakness, which should respond to drug review
- Mechanical disadvantage, which might respond to breathing re-education or exercise training.

Therefore, for strong inspiratory muscles, it is presumed inappropriate to add a further load. For weak muscles, the cause should be addressed.

Question 2

Training can make the diaphragm either more or less susceptible to fatigue. How?

A diaphragm that becomes more susceptible to fatigue after training is thought to have reached maximum adaptability and can improve performance no further. It is already chronically fatigued and is more likely to benefit from nutrition than training. Claim that inspiratory muscle training (IMT) can override the protective mechanism of fatigue and lead to exhaustion and desaturation. Fatigue responds to rest. A diaphragm that becomes less susceptible to fatigue after training is in a fit state to adapt to the training stimulus. This is when IMT may be appropriate, especially as people with COPD show parallel decrements in strength of limb and respiratory muscles, each contributing independently to reduced exercise capacity.

Effects of inspiratory muscle training

Increased strength: ability to generate greater force

Increased endurance: ability to generate the same work for a longer time.

When used appropriately, IMT can improve inspiratory muscle strength and endurance but there is limited evidence that this benefits the patient.

Results have been mixed but, in rested and nourished patients, the following outcomes have been reported:

• ↓ breathlessness and ↑ exercise tolerance.

• ↑ nocturnal saturation.

• ↑ motivation to exercise.

One study found there was no effect unless nutrition was providing 1.5 times the basal metabolic needs. Some patients have shown reduced breathlessness by training the expiratory muscles. Expiratory muscle training may benefit people with multiple sclerosis whose poor

cough pressures are caused by weak expiratory muscles, partly due to deconditioning because of inactivity.

Indications and contraindications of inspiratory muscle training

How can we select those patients with weak muscles for whom training might provide protection against chronic fatigue, and avoid overburdening those with fatigued muscles? The clinical symptoms of fatigue and weakness are similar but the two states are distinguishable (p. 7-8).

Fatigued muscles are unsuited to training, and excessive exercise may split fibres, create 'overuse atrophy' and cause muscle damage. Patients with weak muscles may benefit from training, regardless of how breathless they are, and are most likely to respond if:

- They are fearful of activity, because IMT can be used to desensitize them to breathlessness prior to venturing into exercise training
- They are unable to do exercise training for other reasons, in which case IMT can be a substitute
- They find breathing re-education difficult, in which case using the device might familiarize them to an altered breathing pattern, before progressing to self-regulation of breathing
- They enjoy it!

Most studies have investigated people with COPD, but benefits have been reported for people with CF, asthma, restrictive disease and those awaiting heart transplantation. Others include patients with steroid-induced respiratory muscle weakness and those in respiratory failure who fail to wean from mechanical ventilation because of respiratory muscle atrophy.

For people with neurological disease, damaged nerves must be respected and weak muscles not overworked. However, exercise induced injury has not been reported, possibly because patients would not tolerate fatiguing loads. Disorders that leave the intact muscles unaffected, such as quadriplegia, have shown improvement with both inspiratory and expiratory muscle

training. Progressive disorders such as muscular dystrophy have shown some benefit before the stage of advanced disease when CO_2 is retained.

Some benefit has been shown for those without disease, e.g. the elderly and sportsmen who want to maintain their fitness when injury prevents training.

Technique of inspiratory muscle training

Devices are cheap and simple. The principles of training are followed:

- Alternate exercise with rest
- Avoid distressing levels of fatigue
- Progress by time and/or resistance.

For strength training, the target is generally 80 % of MIP, and for endurance training it is 60%, but benefits have been found at 30% of maximum.

If the aim is desensitization to breathlessness, resistance should be at a level that leaves the patient more breathless than normal but not speechless or distressed. More simply, a resistance can be set that the patient can tolerate for 10 minutes. Patients should be relaxed but inhale with sufficient force to overcome the resistance. They should work at different ranges to prevent muscle fatigue, while avoiding excess hyperinflation. If oxygen is needed, nasal cannulae can be used.

If progressing by time, this increases from about 5 minutes twice a day to about 15 minutes three times a day. If the patient prefers, the timing remains stable, e.g. five 2-minute periods three times a day, with resistance increased fortnightly for the first 6 weeks and then monthly.

When patients have mastered the art, training can be combined with watching TV or reading. Adherence is reasonable when IMT fits into the patient's schedule and the resistance is not uncomfortably high. Training diaries and further details of technique are provided by manufacturers (Appendix C).

A flow-dependent device (Figure a) sets resistance by the size of various inspiratory orifices, but this load can be lessened by the patient taking slow breaths to reduce turbulence. These devices are less likely to produce a training effect and are best used for desensitization to breathlessness. PEP masks can be used as flow-dependent inspiratory muscle trainers by attaching the resistance to the inspiratory port.

A pressure-threshold device (Figure b) incorporates a spring-loaded one-way valve, which opens to permit airflow only when a preset inspiratory pressure has been reached. The load is independent of airflow and can be set at a percentage of MIP. This obliges the patient to generate a set inspiratory force with every breath and is able to create a training effect.

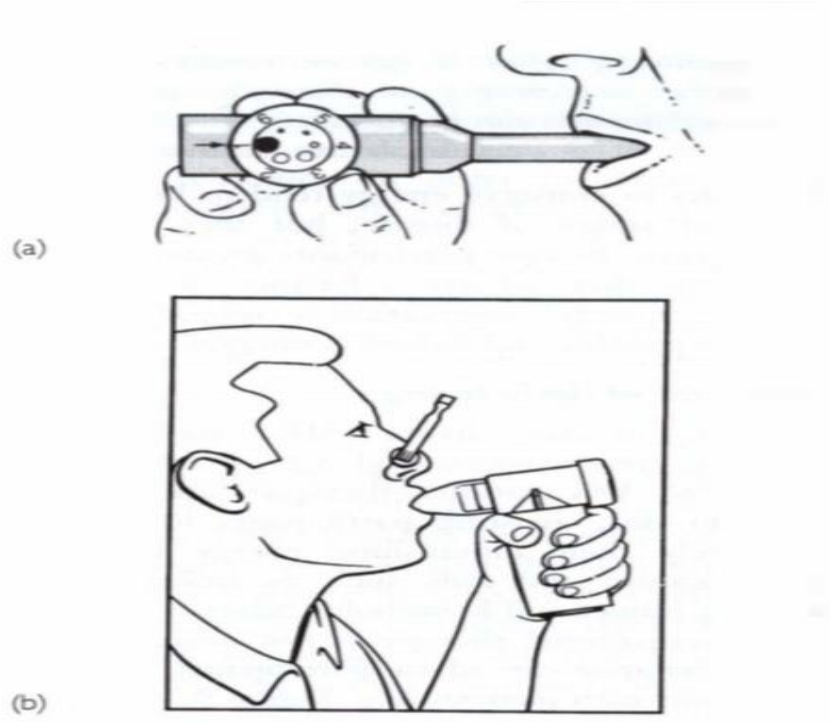


Figure 9.17 (a) Flow-dependent inspiratory muscle trainer. (b) Pressure-threshold inspiratory muscle trainer.

Incentive spirometry has been shown to provide sufficient resistance to create a training response in some elderly people.

Energy conservation

Strategies to conserve energy tend to be used in the later stages of disease, but they are best taught early to give participants greater control over how they achieve a balance of rest and exercise. Energy conservation is compatible with exercise training and indeed is integral to it.

Activities of daily living

Activities of daily living (ADL) training can improve breathlessness and SaO_2. Occupational therapists are valuable allies in this, assisting participants to allocate selectively their

diminishing energy by work simplification and aids such as trolleys, high walking frames and household gadgets. If occupational therapy is not available, the physiotherapist can advise participants, or make a handout with pictures, based on the following:

- Prioritize activities, eliminate non-essentials.
- Plan in advance, allow time, alternate hard and easy chores, spread energy-demanding tasks over the week, pace activities and work in stages.
- Organize chores by location to avoid multiple trips.
- Co-ordinate breathing, e.g. inhale with pulling and exhale with pushing, bending or the strenuous part of an activity ('blow as you go').
- Move smoothly, avoid extraneous movements, use a rolla tor rather than a Zimmer frame, which destabilizes the shoulder girdle and requires twice the oxygen consumption.
- Lean on shopping trolleys.
- Organize work space to reduce clutter and minimize reaching and bending.
- Ensure that work surfaces are the correct height.
- Keep heavy items on top of the work surface.
- Rest elbows on worktop for arm activities.
- Develop economic lifting methods using leg power rather than back and shoulders.
- Slide pots and pans along the worktop rather than lifting them.
- Prepare large one-dish meals such as casseroles, serve in baking dish, freeze leftovers.
- Soak washing up.
- Use a stool for kitchen work and ironing, as this can save 24% of energy.
- Sit to dress, put on two items at once e.g. underwear with trousers or skirt.
- Reduce bending by crossing one leg over the other to put on socks, trousers and shoes.
- Avoid aerosols or strongly scented perfumes.
- For bed-making, have a raised bed on casters away from walls, unfold sheets on the bed, make only one trip round the bed.

- Use non-iron clothes, electric toothbrush, long-handled sponge, soap-on-a-rope, towelling bathrobes, slip-on shoes or Velcro closures.
- Plan ahead for socializing because the energy expenditure can equal that of walking.

Stress reduction

People with chronic lung disease suffer muscle tension from breathlessness, stress and the body positions needed to ease their breathing. A rhythmically active muscle such as the diaphragm is in particular need of relaxation in order to return to its resting position after contraction, especially when it is being overused to maintain hyperinflation. Some have become accustomed to muscle tension and forget how it feels to be relaxed. Relaxation helps breathing and breathing helps relaxation. It should be taught early and reinforced throughout.

Relaxation

Relaxation can be taught by several methods, or self-taught from books, tapes or classes. Daily practice is needed until the sensation is appreciated and the skill mastered, whereupon a degree of relaxation is integrated into everyday life by identifying stressful situations and practicing in different positions. Relaxed walking can be consciously maintained. Spot checks during the day can identify body tension. Relaxation can be achieved in other ways. Participants often have their own ideas, e.g. sewing, jigsaws or, for insomniacs, watching a lighted aquarium at night. Activities such as circle dancing or Tai Chi provide rhythmic exercise with a meditative effect, which emphasizes trunk rotation, and improve balance, posture, immune function and conditioning.

Complementary therapies

Complementary therapies may help ease breathlessness and stress, depending on the practitioner. It is useful to have some knowledge of complementary therapies and local resources for participants who request this information. Yoga incorporates breathing

techniques, meditation and postures that consume minimal energy and induce physiological effects characteristic of deep relaxation.

Mechanical rest

For chronically fatigued patients, non-invasive ventilation at home may be part of rehabilitation.

Follow-up, home management and self help

Follow-up plans should be set at the start of the programme. It takes a minimum of 6 weeks, and often longer, for participants to see an improvement. If expectations are not met, they may lose heart. Some supervised training is best continued for a period after the initial programme, to prevent detraining and demotivation. Thereafter, follow-up in patients' homes or by telephone, newsletter or further training sessions may be needed, 3- and then 6-monthly.

The hospital may be able to provide a regular venue, or a leisure centre may be appropriate. Transport to follow-up meetings and social get-togethers may be available through voluntary organizations.

Home visits are especially useful for people who are elderly, anxious, forgetful, using new equipment or at the end-stage of disease. The home environment is where people feel most in control and are most responsive to advice. Patients are now cared for at home when they have relatively acute disease or when using intravenous therapy, tracheostomies or home ventilators.

Home visits provide the opportunity to check for adequate heating, and health or safety hazards. They are also supportive for the family. Spouses may be stressed, neglect their own health, feel guilty or be fearful of sleeping lest their partner die in the night. Children may lack attention and be caught up in conflicting emotions. Between visits, patients and families need a contact telephone number.

Respiratory patients can withdraw into social isolation because of the nature of their symptoms, and the mutual support that develops between participants during the rehabilitation programme

may become one of its most enduring assets. This support can be built into self-help groups using the Internet, coordinated programs, or peer outreach programmes in which patients are visited by volunteers with lung disease who have been selected for their ability to cope with their disabilities. Social outings, monthly lunches and annual celebrations may develop, which are particularly supportive for people who do not like to be seen in public with their oxygen.

Outcomes

Evaluation of rehabilitation can be by the following:

- Number of participants completing the programme
- Scales on breathlessness and QoL
- Diary review
- Hobbies, job (if wanted)
- GP visits, admissions to hospital, time in hospital
- Anxiety and depression scores
- Smoking
- Medication e.g. ↓ $beta_2$ agonists
- Independence in ADL.
- Video evidence of improved flexibility, posture and gait
- Weight ↓ or ↑ as appropriate
- Specifically in relation to exercise training: ↑ walking distance, ↑ shuttle test, ↑$V0_2$ max, ↓ exercise hr, ↓ blood lactate levels
- Improved well-being of carers.

Student activities

Course title: P.T for Cardiothoracic Diseases and its Surgeries. level : Third

Academic year: 2024/2025 - First semester

Student name: …….. ID #:……Lab section #:…….

1. **Attendance**

Lectures														
Practical sections														

2. **Quizzes :**

Quiz 1	**Quiz 2**	**Quiz 3**	**Quiz 4**	**Quiz 5**	**Quiz 6**	**Quiz 7**	**Quiz 8**	**Quiz 9**	**Quiz 10**
Student average mark									

2. Practical assignment:

……..

3. Teamwork research assignment

…….

Student total mark	

Lab section demonstrator **lecturer of the course**

…… **…….**