# 27

# Acute Medical Conditions: Cardiopulmonary Disease, Medical Frailty, and Renal Failure

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Medical rehabilitation medicine needs to encompass the needs of patients with cardiopulmonary disorders as well as patients with debility and renal dysfunction. There are ever greater numbers of patients who are now presenting with these issues, especially as the population ages. Cardiac disease is still the number one cause of mortality and disability in the United States, with chronic obstructive pulmonary disease (COPD) currently being the third leading cause of mortality.[105] Even if it is not primary rehabilitation for patients with these diseases, many patients with other disabilities will have cardiac, pulmonary, and renal disabilities, and a common theme among all of these conditions is a degree of frailty.

This issue of frailty underlies a great number of the common features of disability in these underlying conditions and will be discussed in the section on frailty, later. Key areas that will be addressed include sarcopenia, acute and chronic deconditioning, and the role of exercise as medicine to improve patients with all of these conditions.

Important contributors to the prevalence of all of these conditions are the aging of the population and the effects of combinations of these conditions on the ability to rehabilitate patients. The overall approaches to exercise treatment in these patients will be discussed as well as how to approach these conditions in the acute, rehabilitation, and outpatient settings.

## Cardiopulmonary Rehabilitation

It must be remembered that there are two types of cardiopulmonary patients, those with primary cardiac and pulmonary disease who need cardiac/pulmonary rehabilitation and those patients with other disabilities who have a cardiac or pulmonary secondary disability. Patients with respiratory failure and patients who have need for ventilatory support are also in this group but are beyond the scope of this chapter. Dual disability patients are more prevalent than ever in rehabilitation because more patients are currently older and have multiple comorbidities. Many patients with stroke, vascular disease, or other conditions can be included in active cardiac and pulmonary rehabilitation programs or benefit from the application of cardiopulmonary rehabilitation principles to their rehabilitation. Remember also that cardiopulmonary rehabilitation is one of the most underused yet most effective treatments for patients with cardiopulmonary disease. Because we work with frail older adults and other compromised populations, it is important for rehabilitation specialists to know how to provide cardiopulmonary rehabilitation in patients with either primary or secondary cardiopulmonary disability.

To use cardiac and pulmonary rehabilitation principles for patients with cardiopulmonary disease, whether it is a primary or secondary disability, it is necessary to review some of the basic principles of cardiac and pulmonary physiology and learn how to apply these principles to improve the exercise capacity of these patients. It is also essential to have an understanding of normal exercise physiology to appreciate the issues of patients with abnormal cardiopulmonary physiology.

## Assessment of Cardiopulmonary Function

### *History and Physical Examination*

A complete cardiopulmonary history and physical examination are essential parts of the evaluation of patients with cardiopulmonary disease who participate in rehabilitation. Key parts of the history include both verbal and nonverbal cues and will allow the establishment of goals and improve patient compliance with the treatment program.

#### *History*

The history should include emotional state, concurrent illnesses, other disabilities, functional history, occupational history, social history, personal habits, family dynamics, and the effect of disability and cardiopulmonary illness on the patient in the community. Both rest and activity symptoms are reviewed with particular emphasis on the following.[14]

**Dyspnea.** Shortness of breath is usually the prime complaint for patients with cardiopulmonary disease. The history of dyspnea helps to differentiate the role of cardiac or pulmonary disease in the patient's symptoms. Cardiac dyspnea can be from ischemic heart disease, congestive heart failure, valvular heart disease, and arrhythmias. Pulmonary dyspnea can come from pulmonary vascular disease, restrictive lung disease, and obstructive lung disease. In some patients both cardiac and pulmonary issues may be present, and in all cases physical conditioning needs to be assessed. Because psychological factors are also important, patients also should be screened for anxiety and depression. Finally, an assessment for hypoxemia should be done. See Table 27.1 for a list of common causes of dyspnea.

**Chest Pain.** Chest pain and tightness is not only a mark of coronary insufficiency but can also be seen with valvular heart disease or arrhythmia. Assessing the duration, quality, provocation, location of the pain, and any ameliorating factors can help to assess functional limitations and help to design the appropriate therapy program. In addition, lung conditions can cause chest pressure in both obstructive and restrictive lung disease and is very common in pulmonary vascular disease.

**Palpitations.** Symptoms of palpitations can be indicative of serious arrhythmias.

**Syncope.** Cardiac syncope is usually abrupt with no warning or only a brief warning (with the patient feeling as if he or she were about to pass out) and can be caused by aortic stenosis, idiopathic hypertrophic subaortic stenosis, primary pulmonary hypertension, hypercarbia, hypoxemia, ventricular arrhythmias, reentrant arrhythmias, high-degree atrioventricular block, or sick sinus syndrome. Pulmonary syncope is often gradual in onset and can be caused by hypercarbia, hypoxemia, or pulmonary vascular disease. Orthostatic syncope can be caused by autonomic dysfunction, neurologic disease, vagal stimuli, or psychological stimuli.

**Edema.** Peripheral edema may be an indication of heart failure and may indicate the onset of right ventricular failure in pulmonary vascular disease.

**Fatigue.** Fatigue is likely to be the most common complaint in cardiopulmonary disease and may be worsened by the presence of depression, physical exhaustion, medication side effects, and deconditioning.

**Cough.** Cough can be from both cardiac and pulmonary diseases. "Cardiac" cough is often nocturnal and postural, with little to no sputum production and relieved by assuming an upright position. Cough is common in both restrictive and interstitial lung disease, with or without sputum production.

### *Physical Examination*

A description of the complete and detailed physical examination of the patient with cardiopulmonary disease is beyond the scope of this chapter (see Chapter 1). Still, some important elements of the examination include a general survey of the patient for exophthalmos (possible thyrotoxicosis), xanthelasma (hypercholesterolemia), acrocyanosis (chronic hypoxemia), clubbing (chronic hypoxemia), ankylosis (aortic valve disease and conduction defects), Down syndrome (cardiac abnormalities), myasthenia gravis, or neuromuscular disease (cardiomyopathy, conduction disease, and ventilatory failure). A good cardiopulmonary examination and history can help to prevent complications in a cardiopulmonary rehabilitation

**TABLE 27.1 Causes of Dyspnea**

| | Site of Pathology | Pathophysiology |
|---|---|---|
| **Pulmonary Causes** | | |
| Airflow limitation | Airways | Limitation to ventilation through flow-through airways |
| Restriction (intrinsic) | Lung parenchyma | Poor lung compliance |
| Restriction (extrinsic) | Chest wall | Poor chest wall compliance with or without poor chest wall strength |
| Acute pulmonary disease | Lungs | Increased ventilation/perfusion (V/Q) mismatch |
| **Cardiac Causes** | | |
| Valvular disease | Heart valve stenosis or incompetence | Limited cardiac output |
| Coronary disease | Heart muscle ischemia | Coronary insufficiency leads to myocardial ischemia |
| Heart failure | Ventricular failure | Limited cardiac output from decreased stroke volume |
| **Circulatory** | | |
| Anemia | Low hemoglobin can be from blood loss or from hemoglobinopathies | Limited oxygen-carrying capacity |
| Peripheral circulation | Peripheral arterial disease | Inadequate oxygen supply to metabolically active tissues, leading to early anaerobic threshold |
| **Whole Body** | | |
| Obesity | Excess adipose tissue with associated physiologic changes | Increased work of movement, decreased efficiency<br>May have respiratory restriction if severe—both extrinsic chest wall restriction and upper airway obstruction |
| Psychogenic | Emotional | Hyperventilation, anxiety |
| Deconditioning | Multiple organ systems, muscle weakness, cardiac deconditioning | Loss of ability to effectively distribute systemic blood flow, inefficient aerobic metabolism |
| Malingering | Emotional | Inconsistent results |

program and should be done as a part of the physiatrist's initial history and physical examination.

*A few key cardiopulmonary examination findings are highlighted here.*

Cardiac auscultation can indicate an atrial septal defect, a midsystolic click may indicate mitral valve prolapse, and a murmur may indicate valvular heart disease. Pulmonary hypertension typically produces a heightened second heart sound, a noncompliant ventricle can be detected via an atrial gallop at the cardiac apex, and a left ventricular gallop may reveal heart failure. The pulse contour, split heart sounds, and the quality of the murmur can help to differentiate aortic sclerosis from aortic stenosis. In younger patients, pulmonary stenosis or valvular heart disease needs to be differentiated from idiopathic hypertrophic subaortic stenosis. Diastolic murmurs may be mitral stenosis or pulmonary hypertension with pulmonary valve regurgitation, and continuous murmurs may be from ventricular septal or atrial septal defects. New findings or changes in findings are important as they may indicate the need for further evaluation or alterations in the program of cardiopulmonary rehabilitation.

Lung physical examination may have decreased breath sounds and/or barrel chest in obstructive disease, whereas interstitial lung disease may have diffuse or basilar crackles. Inspiratory stridor may indicate upper airway obstruction, whereas expiratory wheezing and rhonchi can be seen with obstruction or secretions. It is also important to assess symmetry of breathing, accessory muscle use, and possible compromise to diaphragmatic function.

## Cardiac Anatomy and Physiology

### Cardiac Anatomy

To supervise cardiac rehabilitation, it is essential to be familiar with the normal distribution of the major arteries of the heart, the anatomy of the heart valves, and the distribution of ischemia or infarction from the coronary arteries.

The heart has paired atria and ventricles, with deoxygenated venous blood entering the right atrium, traversing the right ventricle through the tricuspid valve, and entering the pulmonary artery through the pulmonic valve. Oxygenated blood enters the left atrium, goes to the left ventricle through the mitral valve, and is ejected into the aorta through the aortic valve. The cardiac valves ensure unidirectional unobstructed flow of blood, with atrial contraction adding up to 15% to 20% to the total cardiac output (CO). Atrial contribution to blood flow is greater with increased heart rate and in conditions with decreased ventricular compliance.[131] Atrial fibrillation can cause a loss of this atrial "kick" and may contribute to cardiac dysfunction.

The cardiac conduction system allows coordinated contraction of the atria and ventricles at a controlled rate. The normal heartbeat is initiated at the *sinoatrial* node and then travels through three atrial internodal pathways to the atrioventricular node, where conduction is delayed to cause sequential atrial and ventricular contraction. Below the atrioventricular node, the signal passes into the bundle of His and divides into left and right bundles. All cardiac fibers then end in terminal branches, which excite the myocytes and cause contraction. The conduction system can be injured by myocardial infarction (MI), aging, and other conditions and can cause heart block or sick sinus syndrome. Accessory pathways that bypass the atrioventricular node can be seen in Wolff-Parkinson-White syndrome.

#### *Variation of Arteries*

There are three main distributions of coronary circulation. Normally the left main coronary artery divides into the left anterior descending and the circumflex arteries, whereas the right coronary artery continues on as a single vessel. Right dominant circulation is seen in 60% of individuals, whereas left dominant circulation with the posterior descending artery arising from the left circumflex is seen in 10% to 15% of individuals. The remaining 30% of individuals have balanced circulation with the posterior descending artery coming from the left circumflex and right coronary arteries.[4]

### Cardiac Physiology

Cardiac myocytes extract nearly 65% of oxygen from the blood at all levels of activity (compared with 36% for brain and 26% for the rest of the body). Cardiac myocytes prefer carbohydrates as an energy source (40%), with fatty acids making up most of the remaining 60%. With high oxygen extraction and coronary blood flow only during diastole, the heart is at high risk of ischemic injury, especially in the endocardium. Coronary vasodilation with exercise is normally done via nitric oxide–mediated pathways and increases blood flow with exertion. The goal of most medical and surgical therapies for ischemia is to restore or preserve myocardial perfusion through vasodilation, bypass, or endovascular procedures. Exercise can increase cardiac collateral circulation and improve arteriolar vasodilation and has long been known to be a primary therapy for cardiac ischemia.[86,112,114,148]

Another important issue to manage with patients with cardiac disease is fluid volume. Appropriate venous return can maintain appropriate cardiac "preload," whereas fluid overload can lead to too much venous return and exacerbate heart failure. In cases of mechanical cardiac constriction, surgery can restore CO, and in dilated heart failure, medical treatment aims to decrease the size of the ventricles to increase CO. In refractory or end-stage disease, left ventricular assist devices (LVADs) and cardiac transplantation are options.

## Pulmonary Anatomy and Physiology

### Pulmonary Anatomy

Important pulmonary anatomy includes the upper and lower airways (the oropharynx, larynx, trachea, main stem bronchi, and smaller bronchi), the lung parenchyma, the chest wall, and musculature (diaphragm, accessory muscles of breathing, rib cage, and pleura). Pulmonary limitations can come from abnormalities in any of these structures. The lungs also have a dual circulation with pulmonary arteries and veins, which deliver deoxygenated blood to the lungs and deliver oxygenated blood to the left atrium and intrinsic pulmonary artery circulation delivering oxygenated blood to the respiratory tree.

Stridor can result from upper airway obstruction from vocal cord paralysis or tumor, whereas asthma, bronchitis, or reactive airway disease may cause dyspnea from lower airway obstruction. Emphysema is a result of parenchymal lung disease with a loss of alveoli leading to decreased intrinsic recoil of the lung and subsequent hyperinflation and dyspnea. In interstitial lung disease and pulmonary fibrosis, there is interstitial scarring with increased recoil and decreased ability to diffuse oxygen through the lung tissues. In some patients, both restrictive and obstructive diseases can be present with one predominant over another (cystic fibrosis and sarcoidosis). In these cases, it is important to evaluate the lung parenchyma with imaging or physiologic testing (pulmonary function tests) to assess which condition may predominate.[13]

## Pulmonary Physiology

Normal breathing is regulated in the medulla oblongata by the respiratory center. Respiratory signals are carried by the phrenic and other somatic nerves to the diaphragm and secondary inspiratory muscles (intercostals, sternocleidomastoids, and pectorals) and cause rhythmic breathing by generating negative pressure in the chest wall. Normal exhalation is passive, resulting from the elastic recoil of the chest wall and the lung parenchyma. COPD and emphysema can create the need for active exhalation, markedly increasing the work of breathing. Interstitial lung disease with scarring decreases compliance of lung tissue so severely that lung volumes decrease and hypoventilation can result. Any disease affecting the brain, spine, phrenic nerves, respiratory muscles, or changing the mechanical properties of the chest wall or diaphragm can affect normal respiration.[99,100]

Pulmonary vascular disease can result in either primary or secondary pulmonary hypertension. Primary pulmonary hypertension can be idiopathic or can result from vasculitis, thromboembolic disease, or intrinsic parenchymal disease. Secondary pulmonary hypertension is from vascular congestion, often a result of left heart failure. Secondary pulmonary hypertension can lead to intrinsic vascular compromise if the condition is chronic. Chronic hypoxemia may also create pulmonary hypertension in individuals with obesity, obstructive sleep apnea, or high-altitude exposure through a mechanism of pulmonary vascular constriction. Chronic hypoxemia can lead to vascular intimal hypertrophy with resultant fixed pulmonary vascular resistance and pulmonary hypertension.

## Basic Terminology for Exercise

### *Aerobic Capacity*

Aerobic capacity ($Vo_{2max}$) is the measure of the work capacity of an individual and is expressed as the oxygen consumed by the individual (liters of oxygen per minute or milliliters of oxygen per kilogram per minute). Oxygen consumption ($Vo_2$) increases linearly with workload, up to the $Vo_{2max}$, where it reaches a plateau. Maximal exercise capacity assessment can assist in rating disability and planning exercise and recovery programs.[13]

### *Heart Rate*

Heart rate is a useful guide for exercise as a result of having a linear relationship to $Vo_2$. Maximum heart rate is best determined by testing and decreases with age. It can be estimated either by the Karvonen equation or by the equation heart rate = 220 − age.[70] Physical conditioning can alter the slope of the relationship of heart rate and $Vo_2$ with improved conditioning lowering the slope (less heart rate increase for a given $Vo_2$). A limitation to using heart rate can be the alteration of heart rate response in the setting of medications that alter vagal and sympathetic tone.[13]

### *Stroke Volume*

Stroke volume (SV) is the volume of blood ejected with a left ventricular contraction. Maximal SV can be increased with exercise and is sensitive to postural changes (least increases in supine), with the greatest increase during early exercise. Normally SV increases in a curvilinear manner, achieving maximum at approximately 40% of $Vo_{2max}$. SV declines with advancing age, with decreased cardiac compliance, after MI, and in heart failure.[40]

### *Cardiac Output*

CO is the product of the heart rate and SV. It has a linear relationship with work and is the primary determinant of $Vo_{2max}$. At maximum exercise, left ventricular ejection fraction (LVEF), and thus CO, is greater in the upright position compared with the supine position.[13,121]

### *Myocardial Oxygen Consumption*

Myocardial oxygen consumption ($Mvo_2$) is the $Vo_2$ of the heart muscle increasing in proportion to workload. When the $Mvo_2$ exceeds the maximum coronary artery oxygen delivery, an individual will have myocardial ischemia and angina. The rate pressure product (RPP) = [heart rate × systolic blood pressure (SBP)]/100 and has a direct relationship to the $Mvo_2$. Another consideration is that arm exercise, isometric exercise, and exertion in the cold, extreme heat, after eating, and after smoking all have a higher $Mvo_2$ for a given $Mvo_2$. Supine exercises have a higher $Mvo_2$ at low intensity and a lower $Mvo_2$ at high intensity compared with erect exercises.[13]

Basic static lung volumes and dynamic responses to exercise are helpful in the assessment of exercise capacity in individuals with lung disease. Although complete pulmonary function evaluation is beyond the scope of this chapter, some important values include:

Total lung capacity (TLC): Volume of air in the lungs at full inspiration
Vital capacity (VC): Volume of air between full inspiration and full expiration
Forced expired vital capacity (FVC): Maximum volume expired after a maximal forced expiration
Forced expiratory volume in 1 second ($FEV_1$): Maximum volume exhaled in 1 second
Maximal voluntary ventilation (MVV): Measurement of the maximal ventilation over 15 seconds
Residual volume (RV): Volume of the chest wall after a full expiration
Tidal volume (TV): Volume of regular resting breath
Diffusion of the lung for carbon monoxide ($D_{LCO}$): Diffusion of carbon monoxide (oxygen analog) across the alveolar membrane

The best evaluation of the capacity to exercise in cardiac and pulmonary conditions is with a cardiopulmonary exercise test (CPET). The CPET yields diagnostic, prognostic, and exercise prescription guidance in patients with cardiopulmonary disease. The interpretation of pulmonary exercise testing in a number of conditions is shown in Table 27.2.

## Interventions for Cardiopulmonary Disease

### *Aerobic Training*

Physical exercise that increases the cardiopulmonary capacity ($Vo_{2max}$) allows for aerobic training. All aerobic training prescriptions must include four components: intensity, duration, frequency, and specificity.

**Intensity.** How hard an exercise is. It can be prescribed by a target heart rate, metabolic (MET) level, or intensity (wattage). Usual intensity target for cardiac primary prevention is a heart rate of 80% to 85% of the predicted maximum heart rate/peak heart rate from the exercise tolerance test (ETT). For secondary prevention in patients with known cardiopulmonary disease, exercise should be at a safe level at 60% or more of the maximum heart rate to achieve a training effect.

**Duration.** How long a given bout of exercise is. Usual cardiopulmonary conditioning requires 20- to 30-minute sessions and

**TABLE 27.2 Effects of Physiologic Conditions on Cardiopulmonary Exercise Capacity**

| Abnormality | Physiologic Abnormality | Gas Exchange |
|---|---|---|
| Obesity | • Increased work with activity<br>• Inefficient exercise | • Rapid alveolar-arterial $p(A\text{-}a)O_2$ fall with exercise<br>• Low aerobic capacity ($VO_{2max}$)<br>• Rapid fatigue |
| Peripheral vascular disease | • Claudication can limit exercise | • Low $VO_{2max}$<br>• Increased lactic acidosis<br>• Associated deconditioning often present<br>• Low anaerobic threshold |
| Pulmonary vascular disease | • Impaired pulmonary blood flow<br>• Right ventricular failure or overload<br>• Possible right to left shunt | • Low $VO_{2max}$<br>• Low anaerobic threshold<br>• Rapid pulse at low exercise<br>• Hypoxemia<br>• Excessive dyspnea |
| Anemia | • Low oxygen-carrying capacity | • Low $VO_{2max}$<br>• Early anaerobic threshold<br>• Rapid pulse at low exercise<br>• Fatigue and dyspnea |
| Chronic obstructive pulmonary disease | • Impairment to expiratory phase of breathing<br>• Decreased alveolar ventilation | • Low $VO_{2max}$<br>• Low anaerobic threshold<br>• Rapid pulse at low exercise<br>• Submaximum heart rate achieved<br>• Retention of $CO_2$ that increases with exercise |
| Restrictive lung disease (intrinsic) | • Poor diffusion capacity<br>• Poor pulmonary compliance<br>• Pulmonary hypertension in later disease | • Low $VO_{2max}$<br>• Early anaerobic threshold<br>• Tachypnea at all levels of exertion<br>• Low pulmonary reserve<br>• High alveolar-arterial $p(A\text{-}a)O_2$ difference yielding marked hypoxemia<br>• Marked dyspnea<br>• Presence of pulmonary hypertension can cause severe hypoxemia and loss of hemodynamic response to exercise<br>• Can trigger cough with exercise |
| Restrictive lung disease (extrinsic) | • Poor chest wall compliance<br>• Chest wall muscle weakness<br>• Loss of neural control of breathing musculature | • Low $VO_{2max}$<br>• Early anaerobic threshold<br>• Tachypnea with low tidal volumes<br>• Low pulmonary reserve<br>• Submaximum heart rate achieved<br>• Oxygenation and $CO_2$ usually preserved until severe end-stage disease |
| Asthma | • Restricted expiratory phase of breathing from airway obstruction<br>• Decreased alveolar ventilation<br>• In exercise-induced asthma, peak flows drop 5–10 minutes into exercise | • Most findings are normal when not symptomatic and resemble obstructive disease with acute attack |
| Ventricular failure | • Compromised pulmonary blood flow<br>• In left ventricular failure, can have pulmonary vascular congestion | • Low $VO_{2max}$<br>• Early anaerobic threshold<br>• Tachypnea, dyspnea<br>• Exaggerated heart rate response to exercise<br>• May have hypoxemia with pulmonary congestion and loss of normal hemodynamic response to exercise |
| Ischemic heart disease | • Chest pain/cardiac ischemia<br>• Can precipitate ventricular failure | • Often normal at rest or until ischemia<br>• With onset of ischemia and ventricular stiffening/systolic dysfunction, can appear like mild ventricular failure<br>• Can have loss of normal hemodynamic response to exercise with onset of ischemia |
| Metabolic acidosis | • Metabolic acidosis, low $HCO_3^-$ | • Normal diffusion<br>• Exaggerated response of ventilation to exercise<br>• Low $VO_{2max}$ |

should have a 5- to 10-minute warm-up and cool-down period. The lower the intensity of an exercise, the longer the duration will need to be to achieve a similar training effect.

**Frequency.** How often exercise is performed over a fixed time period (usually a week). Moderate-intensity training programs should be done at least three times per week, and low-intensity programs should be done five times per week.

**Specificity.** The activity to be done in exercise. Training benefits are specifically related to the activities performed. Thus elliptical exercise is not as beneficial for walking as treadmill training. Specificity in prescription should be altered to adapt to the needs of each patient. For a patient with spinal cord injury, upper arm ergometry would be more functional, and for a patient with severe leg arthritis, cycle ergometry would be better than a treadmill. The law of specificity of conditioning should be remembered when designing a cardiopulmonary conditioning program.[2]

The benefits of aerobic training include the following:

Aerobic capacity: Maximum capacity increases with training. Resting $Vo_2$ is stable as is the $Vo_2$ at a given workload. The changes are specific to the trained muscles.

Cardiac output: Maximum CO increases, whereas resting CO is stable. Resting SV increases with a corresponding decrease in resting heart rate.

Heart rate: Heart rate is lower at rest and at any given workload, whereas maximum heart rate is unchanged. The lower heart rate at rest and submaximal exercise causes a lower $Mvo_2$ with normal activity.

Stroke volume: SV increases at rest and at all levels of exercise. CO is thus maintained at a lower heart rate and causes a lower RPP for a given level of exertion.

Myocardial oxygen capacity: After training, maximum $Mvo_2$ does not usually change but is less at a given workload. This reduces episodes of angina and increases safety for moderate activity. $Mvo_2$ can also increase after pharmacologic treatments or revascularization procedures.

Peripheral resistance: Exercise training decreases peripheral vascular resistance (PVR) by reducing "afterload" through lowering arterial and arteriolar tone. The reduction in PVR results in a lower RPP and a lower $Mvo_2$ at a given workload and at rest.

Minute ventilation: With improved conditioning, individuals will require a lower $Vo_2$ and thus a lower minute ventilation for a given activity. For patients with pulmonary and cardiac disease, this can lead to a large reduction in dyspnea.

Tidal volume: Exercise can lead to a higher TV on exertion, with a subsequent decrease in respiratory rate and decreased dyspnea.

Respiratory rate: As TV is improved, respiratory rate will be lower for a given minute ventilation, decreasing dyspnea.

The application of basic physiologic principles to the design of cardiopulmonary rehabilitation programs can improve function, decrease symptoms, and improve outcomes for patients with cardiopulmonary disease. The prime effect of cardiac conditioning is in reduction of cardiac risk and improved cardiac conditioning. Reduction of cardiac risk has been well established since 1989, when pooled data from 22 randomized studies of exercise in 4554 patients following acute MI demonstrated a 20% to 25% reduction in all-cause mortality, fatal MI, and cardiac mortality in a 3-year follow-up study.[7] These benefits of cardiac rehabilitation apply across populations, including older adults, women, and patients after bypass.[7] Similar benefits have also been shown for pulmonary rehabilitation in COPD with decreased hospitalizations, improved function, and improved quality of life,[75,109,110,123] and new studies are showing that interstitial disease and pulmonary vascular disease can also benefit from exercise.[33,43,59,63,132]

# Pulmonary Rehabilitation

## Abnormal Physiology: Lung

Patients with pulmonary disease demonstrate three main impairments: (1) obstructive lung disease, (2) restrictive lung disease, and (3) pulmonary vascular disease. Often more than one type of limitation may be present in a given patient and will increase the complexity of their condition. Understanding the underlying physiology can assist in the design of a specific exercise program for an individual patient.

For primary pulmonary disease, it is essential to know if the patient has primarily an obstructive or restrictive condition. Obstructive lung disease is marked by an inability to exhale resulting from either upper airway or large airway disease (sleep apnea, tracheomalacia, vocal cord disease, asthma, and bronchitis) or as a result of lower airway disease from either secretions or lung parenchymal disease (emphysema and bronchiectasis). Obstruction can also be exacerbated by a component of acute obstruction (asthma) combined with a chronic condition (COPD). The hallmark of severe COPD is carbon dioxide retention and active exhalation. Medical treatments are limited for COPD, with steroids and bronchodilators offering incomplete relief. Lung reduction surgery is appropriate only in selected individuals, and transplant is only for end-stage disease. For all levels of obstructive disease, pulmonary rehabilitation is appropriate, and in the "GOLD" recommendations for treatment of COPD, pulmonary rehabilitation is recommended for all patients with moderate to severe disease.[47,48]

In restrictive lung disease, the primary limitations are low TVs from an inability to expand the chest wall (extrinsic restriction) or from very noncompliant lung tissue (intrinsic restriction). In extrinsic restrictive disease (neuromuscular disease, paralysis, and kyphoscoliosis), the parenchyma of the lung is normal and gas exchange is preserved, meaning that treatment is usually respiratory muscle training and mechanical ventilatory support as needed. With intrinsic restrictive lung diseases (pulmonary fibrosis, sarcoidosis, etc.), there may be a profound associated hypoxemia from severely decreased diffusion capacity of scarred lung tissue. Patients with parenchymal restrictive disease classically have severe hypoxemia and may need high-flow supplemental oxygen. Patients with end-stage intrinsic restrictive disease can have ventilatory failure with hypercarbia and hypoxemia and may be refractory to ventilatory support, and lung transplantation is then often the only remaining treatment option. Table 27.3 shows some of the lung pathologies and effects on inspiratory reserve and RV (obstructive diseases) and the effects of various conditions on lung compliance (restrictive diseases).[15]

Finally, patients with pulmonary vascular disease have a similar presentation in many ways to patients with heart failure, and in the end stages of the disease, right ventricular heart failure is a major part of the condition and leads to excess mortality and morbidity.[82] Rehabilitation is focused on a program that resembles exercise for patients with heart failure, with the addition of close monitoring of oxygen saturation and the use of appropriate levels of supplemental oxygen to prevent hypoxemia.

**TABLE 27.3 Causes of Altered Lung Physiology**

| Restrictive Diseases | Obstructive Diseases |
| --- | --- |
| Loss of inspiratory reserve | Increase in residual volume |
| Intrinsic loss of inspiratory reserve | Intrinsic increase in residual volume |
| • Lung fibrosis<br>• Pulmonary hypertension<br>• Pulmonary edema | • Bronchial obstruction (acute asthma)<br>• Airways collapse (chronic obstructive lung disease/ emphysema)<br>• Bronchial obstruction (bronchiectasis, cystic fibrosis) |
| Extrinsic loss of inspiratory reserve | Extrinsic increase in the residual volume |
| • Chest wall rigidity<br>• Neurologic (central) weakness<br>• Neurologic (peripheral) weakness<br>• Chest wall restriction from bracing | • Neck obesity<br>• Tracheomalacia |

For patients with either intrinsic restrictive or obstructive disease, pulmonary rehabilitation is an important treatment to consider and should be offered for patients whether or not they have their pulmonary condition as a primary or a secondary disability. A brief overview of pulmonary rehabilitation programs for primary pulmonary disease is shown in Table 27.4.

## Cardiac Rehabilitation

### Abnormal Physiology: Heart

An understanding of abnormal cardiac physiology in disease is necessary for appropriate cardiac rehabilitation. In general, cardiac limitation is caused by either decreased CO, or ischemic disease, or a combination of these. Ischemia causes the myocardium to have lower contractility and lower compliance reducing SV. Valvular heart disease lowers maximum CO through stenotic valves (e.g., aortic or mitral stenosis) or valvular regurgitation (e.g., aortic or mitral insufficiency). Finally, heart failure is a state of low CO, often as a result of low SV, and is associated with a reduction of $Vo_{2max}$, increased resting heart rate, and often a greater $Mvo_2$ for a given $Vo_2$.

Arrhythmias decrease CO by lowering SV and increasing heart rates. For atrial arrhythmias, the mechanism can be by a loss of atrial ventricular filling (atrial "kick") during atrial fibrillation or supraventricular tachycardias, or from high heart rates without atrial coordination as in ventricular tachycardias and ventricular bigeminy.

Surgical treatments for heart disease either restore coronary circulation (e.g., bypass and intravascular procedures) or restore normal anatomy (e.g., valve replacement). Surgical treatment for heart failure can include LVADs or transplantation.[130,151] Medical treatment for heart disease either aims to improve coronary circulation for ischemia or works to improve blood flow and restore CO for heart failure by lowering afterload, reducing fluid overload, and increasing inotropy. Although medical treatment of ventricular arrhythmias has been limited, implantable defibrillators and pacemakers have been very successful. Severe end-stage heart disease of all types may require cardiac transplantation or an LVAD. In all of these conditions and treatments, cardiac rehabilitation has an important role to play. Some basic concepts to remember include that patients before transplantation are similar to patients with heart failure, whereas patients after transplantation have several physiologic changes that are unique, including high resting heart rate, limited increase in SV, and peak heart rate with exercise. The basic principles of cardiac rehabilitation are discussed as follows.[13]

Cardiac rehabilitation is either primary prevention, which includes risk factor modification and education before a cardiac event, or secondary prevention, which is cardiac rehabilitation after the onset of cardiac disease including both exercise and risk factor modification.

Primary prevention is usually performed in primary care settings rather than a rehabilitation setting. The focus is on the reduction of cardiac risk factors with a combination of education and exercise for patients in the community. Primary prevention can have a profound effect on the rate of cardiac disease with a decrease in obesity, blood pressure, and lipid profiles.[53,54,147] Ideally, behavior modification should begin in childhood with the establishment of healthy behavior and then maintained throughout life. Because populations who are disabled are generally sedentary and may have other risk factors, primary prevention should be an important component of the care of the disabled and should include management of hypertension and lipids along with encouraging exercise and consideration of antiplatelet agents. These are all cost-effective approaches and can decrease mortality and morbidity on a population-based scale, in addition to the individual benefits.[53,54,147]

After an episode of cardiac disease, it is essential to have secondary risk factor modification, which includes all of the features of primary prevention programs discussed earlier. In addition, disease-specific education and formal exercise is a part of the secondary prevention program. In both cardiac and pulmonary disease, smoking cessation is crucial as part of both primary and secondary prevention programs.[6,87,158]

### Pulmonary Rehabilitation Programs

Rehabilitation programs for patients with pulmonary disease are similar to cardiac rehabilitation programs. After severe acute exacerbations, some patients can benefit from a short acute inpatient rehabilitation, but the majority of pulmonary rehabilitation is done in an outpatient setting. For patients who are in an intensive care setting, early mobilization programs are now being used to limit debility in these vulnerable patients.[5,18,107] Outpatient pulmonary rehabilitation programs also have primary prevention for pulmonary disease with smoking prevention and cessation, occupational safety, and prevention of exposure to environmental and infectious agents. Secondary pulmonary prevention involves medication adherence and education, smoking cessation, supplemental oxygen use and education, and environmental modification for known environmental triggers.[75,110]

For patients with ventilatory failure that cannot be supported with noninvasive ventilation, lung transplant may become necessary. Rehabilitation before transplantation is focused on both the underlying condition and transplant-specific education, whereas rehabilitation after transplantation includes education and restoration of muscle strength, which is impaired from the medical regimen for patients after transplantation.[17]

### Cardiac Rehabilitation of the Patient After Myocardial Infarction

The standard model for cardiac rehabilitation after MI was first described by Wenger et al.[155] in 1971. Because revascularization is currently common and infarcts are smaller than in the past, there

**TABLE 27.4 Summary of Goals and Methods of Pulmonary Rehabilitation**

| Goals | Methods |
|---|---|
| **Primary and Secondary Prevention** | |
| Smoking cessation | Smoking cessation programs, emotional support, monitor and encourage abstinence |
| Immunization | Assure proper immunizations (flu and pneumonia), communicate with primary physician |
| Prevent exacerbations | Disease education<br>Self-assessment skills taught<br>Self-intervention taught<br>Instruct on accessing private physician |
| Appropriate medication use | Review medication<br>Focus on inhaler technique<br>Review dosing schedules<br>Review interactions and side effects<br>Focus on appropriate use of inhalers and nebulizers |
| Pulmonary toilet | Review bronchial hygiene<br>Teach cough techniques/huffing<br>Teach appropriate use of chest physiotherapy techniques to the patient and family |
| Appropriate use of oxygen therapy | Encourage acceptance of the need for $O_2$<br>Appropriate use of oxygen at rest and with exertion<br>Review self-monitoring with pulse oximetry<br>Review oxygen equipment and appropriate systems for a given patient<br>Emphasize the importance of supplemental oxygen use and the consequences of failure to use oxygen |
| Nutritional counseling | Aim to achieve ideal body weight<br>For $CO_2$-retaining individuals, avoid high-carbohydrate diet<br>Maintenance of low-sodium diets<br>Encourage balanced nutrition, avoidance of fad diets |
| Family training | Disease-specific training<br>Pulmonary toilet and chest physiotherapy<br>Medication and oxygen use<br>Family support group<br>Counseling as needed |
| **Dyspnea Relief: Exercise Training** | |
| Exercise | Multifaceted program individualized to each patient's needs |
| • Strengthening | Emphasis on gradual increase in strength with a focus on proximal muscle groups<br>Avoid injury to weakened musculotendinous structures that may have been weakened by disuse and medications<br>Focus on high-repetition, low-intensity training |
| • Conditioning | Aim to increase exercise tolerance with aerobic exercises<br>Cross-training program to avoid injury<br>Create an independent training program<br>Increase ambulation endurance with gait training<br>Appropriate oxygen titration during exercise |
| • Respiratory muscle training for selected conditions | Isocapnic hyperpnea<br>Inspiratory resistance training<br>Inspiratory threshold training |
| • Upper extremity training | Increase strength, focus on proximal muscles<br>Increase endurance for sustained activity, aim to decrease fatigue with ADL |
| • ADL training | Energy conservation and adaptive techniques<br>Teach anxiety and stress relief<br>Teach pacing in activities |
| **Dyspnea Relief: Lifestyle Modifications** | |
| Breathing retraining | Technique of pursed lip breathing, especially in obstructive conditions<br>Diaphragmatic breathing |
| Anxiety reduction | Stress relaxation techniques<br>Paced breathing<br>Autohypnosis<br>Visualization<br>Use of anxiolytics as needed<br>Evaluate and treat any underlying depression |

**TABLE 27.4 Summary of Goals and Methods of Pulmonary Rehabilitation—cont'd**

| Goals | Methods |
|---|---|
| Improve confidence | Build compensatory techniques<br>Build confidence in ability to exercise<br>Provide ability to self-assess and learn disease management techniques |
| **Disease Management** | |
| Disease acceptance | Family and patient education regarding disease process |
| Coping skills | Patient and family support group<br>Psychology and social work intervention as needed<br>Treatment of depression as needed |
| Quality of life improvement | Simplify ADL management, improved coping skills<br>Improve disease management strategies |
| Advance directives review | Counseling regarding end-of-life planning<br>Establishment of healthcare proxy<br>Clarification of intention for resuscitation<br>Assistance in preparing paperwork |
| Encouragement | Patient support group<br>Use of social work and psychological support |
| Continuing exercise and disease management compliance | Multidisciplinary team encouragement<br>Physician (specialty and primary care) consensus<br>Family education and involvement |

*ADL*, Activity of daily living.

have been modifications to the classical program with a reduction to three phases, eliminating the classical stage 2 recovery phase. A modern acute phase mobilization program is illustrated in Table 27.5.

The exception to bypassing the recovery phase for cardiac rehabilitation comes for surgical patients with sternotomy who may require recovery from their surgery before starting the training phase of rehabilitation. In summary, phase 1 rehabilitation is the acute phase in hospital immediately following a cardiac event and ends at discharge. Phase 2 is an outpatient training phase, with secondary prevention, intense education, and aerobic conditioning. Phase 3 is the most difficult, the maintenance phase in which patients seek to achieve continued aerobic exercise and maintenance of lifestyle modifications. Risk factor modification is performed at all phases. This model is similar for patients with pulmonary disease. For patients with cardiopulmonary disease who are not hospitalized, the goal is essentially phase 2 and phase 3 for all patients at the time of diagnosis. A more detailed description of each of the phases follows.

### Acute Phase (Phase 1)

The basics of the phase 1 program are illustrated in Table 27.5. Education about cardiopulmonary risk factor modification is introduced at the time of acute hospitalization. For patients with cardiac disease, all acute mobilizations should be done with cardiac monitoring with appropriate supervision by trained therapists or nurses. A post-MI heart rate increase with activity should be kept to within 20 beats/min of baseline and SBP kept within 20 mm Hg of baseline. A decrease of 10 mm Hg or more is indicative of further medical issues, and exercise should be halted. The target intensity at the end of the phase I program exercise is to a level of four METs, covering most of the daily activities patients may perform at home after discharge.

For patients with pulmonary disease, similar phase 1 goals exist and there is new emphasis on early mobilization in the intensive care unit (ICU) to prevent frailty and deconditioning. Patients are aggressively mobilized, some while still on the ventilator. Innovations, including extracorporeal membrane oxygenation, are also now allowing more aggressive mobilization of patients because sedation is less, and patients may maintain better nutritional status. These patients with pulmonary disease should be enrolled in outpatient pulmonary programs to maintain their early gains and complete a full program of education and exercise.

### Inpatient Rehabilitation Phase (Phase 1B)

To distinguish between patients who have a rapid recovery after their cardiopulmonary event (pure phase 1) and those patients who require either acute or subacute rehabilitation treatment before discharge home, the designation of phase 1B rehabilitation has been established. With advanced age or substantial comorbidities or other disabilities that make mobilization more difficult, many rehabilitation specialists will care for these patients in phase 1B. The guidelines for exercise are the same as they are for patients in phase 1 but with a longer recovery period extending their hospitalized care to an acute or subacute rehabilitation setting before discharge.

### Training Phase (Phase 2)

Classically, phase 2 cardiopulmonary rehabilitation starts after a symptom limited full-level ETT for patients with cardiac disease or a CPET for patients with complex pulmonary disease. This allows for setting target heart rates and target exercise intensity from the exercise. A target heart rate of 85% of the maximum heart rate on an ETT or a CPET is generally regarded as safe for patients at low risk. Exercise intensity targets are lower for patients at higher risk or those with underlying conditions. In patients with life-threatening arrhythmias or chest pain, target heart rates are chosen that are below notable end points. Because hypoxemia can add risk and limits participation with exercise, it is important to provide supplemental oxygen as needed (up to a rate of 15 L/

**TABLE 27.5 Acute Phase I In-Hospital Cardiac Mobilization Program**

| Day | Activity |
|---|---|
| Day 1 | Passive ROM, ankle pumps, introduction to the program, self-feeding |
| | Progress to dangle at side of bed, initiate patient education |
| | Progress to active assisted ROM, sitting upright in a chair, light recreation and use of bedside commode |
| | Increased sitting time by the end of the day, light activities with minimum resistance, continue patient education |
| | Progress to light activities with moderate resistance, unlimited sitting, seated ADLs by end of first day |
| Day 2 | Increased resistance, walking to bathroom, standing ADLs, up to 1-hour-long group meetings |
| | Progress walking up to 100 feet, standing warm-up exercises |
| | Begin walking down stairs (not up), continued education |
| | Progress exercise program with a review of energy conservation and pacing techniques |
| Day 3 | Advance exercise to include light weights and progressive ambulation |
| | Increase the duration of activities |
| | Progress stair activity to climbing two flights of stairs, continue to increase resistance in exercises |
| | Consolidate home exercise program teaching |
| | Aim to safely walk up and down two flights of stairs (ensures safety for normal activities), complete instruction in home exercise program and in energy conservation and pacing techniques |
| | Discharge planning and education |

*ADL,* Activity of daily living; *ROM,* range of motion.

min as needed) to maintain saturation greater than 90% for safe exercise. A target heart rate of 65% to 75% of maximum is safe and effective in a regular exercise program for patients at higher risk[54] and, with target rates as low as 60%, still provides a training benefit. Monitoring also needs to be customized to accommodate the underlying risk profile.

Classically, a cardiopulmonary training program is three sessions per week for 8 to 12 weeks. Cardiac rehabilitation is covered by most insurance plans, but the major limitation is a lack of referral and/or a lack of facilities in many areas. Creative and innovative care delivery programs have been developed to increase access and include home programs (patients at low risk), telemedicine programs, and community-based programs in nonmedical facilities. Because training continues after the 8- to 12-week period, it is important for patient self-efficacy that they learn to perform self-monitoring following the guidelines presented in standard references.[55,83] Patients need to learn to begin exercise with a stretching session, then a warm-up session, a period of training exercise at designated intensity, followed by a cool-down period. The principles of specificity of training need to be remembered because training benefits generally are seen in the specific muscles exercised.

### *Maintenance Phase (Phase 3)*

Although the maintenance phase of cardiopulmonary rehabilitation is the most important part of the program, it often receives the least attention. The benefits of a phase 2 program can be lost in as little time as a few weeks if a patient ceases to exercise. Because of this, patient education of the importance of making exercise a part of their new health habits has to be emphasized and the patient needs to integrate exercise as a part of a healthy lifestyle. To maintain capacity, patients should perform moderate exercise at the target intensity learned in their rehabilitation program for at least 30 minutes three times a week. With low-level exercise, the frequency has to be increased to five times a week for maintenance of gains. Although telemetry monitoring is usually not used with patients with cardiac disease, patients with pulmonary disease can benefit from the use of home pulse oximetry and should be taught to adjust their supplemental oxygen as needed with exercise to maintain adequate oxygenation.[16]

## Cardiac Rehabilitation Programs in Specific Conditions

### *Angina Pectoris*

Cardiac rehabilitation for angina aims to lower heart rate at rest and with given levels of activity to decrease angina by improving fitness. Exercise benefits for patients with angina include improved peripheral efficiency and improved coronary artery collateralization.

## Cardiac Rehabilitation After Revascularization Procedures

### *Postcoronary Artery Bypass Grafting*

Cardiac rehabilitation after coronary artery bypass grafting (CABG) emphasizes secondary prevention aims to improve conditioning and fitness. For patients with low ejection fractions and heart failure, closer telemetry monitoring should be done. If a patient had a sternotomy, arm exercises will have to be limited until sternal healing occurs, usually at approximately 6 weeks after surgery. Patients who have had percutaneous interventions usually pursue the program immediately and it is similar to the program after CABG.

## Cardiac Rehabilitation for Patients After Cardiac Transplantation

Because most patients after cardiac transplantation have severe heart failure and debility before transplantation, involvement in a heart failure pretransplant program can help to limit deconditioning and help to treat depression and anxiety. Heart transplantation usually improves cardiac function; therefore a posttransplant program can focus on conditioning, education, and secondary prevention. An added feature is that many patients after transplantation may have vascular and neurologic complications, which may mean a phase 1B program is needed before starting the phase 2 outpatient program. This is often done in either acute or subacute rehabilitation settings.

Remembering the alterations of cardiac physiology in the patients after transplantation is important. Transplanted hearts are denervated and have no direct sympathetic or vagal central regulation. In many patients, the loss of vagal inhibition creates a resting tachycardia of 100 to 110 beats/min. By contrast, because there is a loss sympathetic innervation, the chronotropic response to exercise is in response to circulating catecholamines, leading to a delayed and blunted heart rate response to exercise. Posttransplant, peak heart rates are usually 20% to 25% lower than in matched controls. Other cardiovascular effects that are seen include resting hypertension from the renal effects of calcineurin inhibitors (e.g., cyclosporine and tacrolimus) and prednisone, along with diastolic dysfunction in some patients.[18] Combined, these effects usually reduce maximum work output and maximum oxygen by approximately one-third compared with age-matched individuals. Of interest, despite no denervation of the heart, similar decreases in exercise capacity are also seen in patients after lung transplantation.[17] With exercise, patients after transplantation have a lower work capacity, reduced CO, lower peak heart rate, and lower oxygen uptake and a higher resting heart rate and SBP than normal individuals. In addition, resting and exertional diastolic blood pressures are usually higher for patients after heart transplantation.[25,142] The net effect of these alterations in exercise response is higher than normal perceived exertion, minute ventilation, and ventilatory equivalent for oxygen at submaximal exercise levels.

The focus of a cardiopulmonary rehabilitation program after transplantation is on conditioning and education. Target intensity for aerobic exercise is usually approximately 60% to 70% of peak effort for 30 to 60 minutes three to five times weekly. Intensity can be regulated with rating of perceived exertion target at 13 to 14 on the Borg Scale, approximately 5 to 6 on the modified Borg Scale, with the goal being to consistently increase the level of activity. Education focuses on learning the complicated medical regimen and vocational and psychological needs. For patients after cardiac transplantation, a program of rehabilitation can help to assist them to improve work output and exercise tolerance, with some patients able to participate in competitive athletics.[71,73,74]

### Cardiomyopathy

Fortunately, cardiac rehabilitation for heart failure is currently covered by insurance plans, since Medicare regulations started to cover rehabilitation for heart failure in March of 2014 (42 C.F.R. § 410.49(b)(1)(vii)). An important consideration for heart failure rehabilitation is the increased risk of complications such as sudden death, depression, and chronic cardiac disability. Closer monitoring of telemetry and vital signs is also needed because some patients with heart failure have inconsistent responses to exercise with increased fatigue, possible exertional hypotension, and syncope. Most patients also exhibit low endurance and chronic fatigue as a result of their low-exercise capacity. However, a positive effect can be realized in their fatigue and function with even a small improvement in $Vo_2$. These changes in capacity can lead to a marked improvement in quality of life and may help patients with heart failure to continue to live independently.[7]

Because of the increased risk for complications in patients with heart failure, a graded ETT is helpful before starting a cardiac rehabilitation program. Long warm-up and cool-down periods with gentle exercise at a limited workload helps to compensate for an impaired ability to generate a dynamic exercise response, and dynamic exercise is preferred to isometric exercise because isometric exercise can lead to an increase in diastolic pressure and cardiac afterload.[34] Heart rate targets are usually set 10 beats/min less than any notable end point found with cardiopulmonary exercise testing. Cardiac rehabilitation begins with cardiac monitoring especially when severe left ventricular dysfunction is present. Once the patient has demonstrated stability with an exercise program and has learned how to self-monitor, the patient should be taught a self-monitored program. Education of patients with heart failure also includes doing a daily body weight (to observe for fluid accumulation) and monitoring their blood pressure and heart rate responses to exercise.[7]

Patients who are on pharmacologic inotropic support or left ventricular mechanical support for end-stage heart failure can also exercise in a cardiac rehabilitation program with similar precautions to other patients with congestive heart failure.[7] Rehabilitation after an LVAD usually follows a classical postsurgical course and may include phase 1 and phase 1B rehabilitation followed by phase 2 and phase 3 programs. Patients with an LVAD seen in acute and subacute units require staff training, close cooperation with the LVAD team, and familiarity with the devices that are used locally. Because an LVAD often restores a reasonable CO, exercise tolerance is often only limited by the peak flow of the device. In addition to normal secondary prevention education, LVAD-specific family and patient education are also essential parts of post-LVAD rehabilitation.[12]

### Valvular Heart Disease

Cardiac rehabilitation for valvular heart disease resembles the program for cardiac heart failure. Postsurgical considerations are the same as for CABG, with the added consideration of anticoagulation for patients with mechanical valves. Because anticoagulation increases the risk of hemarthrosis and bruising, patients need to avoid impact exercises and need education regarding injury avoidance.[7] The overall training program is similar to that discussed for the patient post-CABG.[7]

### Cardiac Arrhythmias

An essential consideration for patients with cardiac arrhythmias is the need for telemetry monitoring with increases in intensity of exercise and new exercises. Patients at high risk can benefit from an automatic implantable cardiac defibrillator (AICD), which may offer protection form ventricular arrhythmias. Cardiac rehabilitation for patients with AICD needs to be done at intensities that avoid the heart rates at which the device is set to respond to ventricular tachyarrhythmias. An exercise stress test can help to set appropriate target heart rates for an exercise program. In addition to secondary prevention and education, AICD-specific education and emotional support are important to include in the rehabilitation program.[12]

### Cardiac Rehabilitation for Stroke

An emerging area of scientific exploration is using the cardiac rehabilitation model for stroke recovery and prevention.[93,94] This is a logical extension of the effectiveness of the cardiac rehabilitation model in reducing known coronary artery disease risk factors in regular participants. Because the risk factors for coronary artery disease are the same as the risk factors for stroke, it is logical to apply the same group exercise-based behavior modification model to a similar at-risk population. Participants must be screened carefully for balance impairments and muscle strength asymmetry that could increase the risk of falling in a group exercise setting. Asymmetric muscle fatigue must be scrupulously monitored and can contribute to degradation of gait or exercise form as exercise progresses. Subtle cognitive deficits may require close observation until it is determined that the stroke patient care set machine intensity levels accurately and independently. Meticulous blood pressure control must be maintained and may require more frequent measurements than regularly collected in cardiac patients. Any questions regarding safe blood pressure range should be directed to the referring neurologist. This is analogous to target heart-rate range directed by referring cardiologist when indicated.

### High-Intensity Interval Training

A new and exciting area of emerging research is the conditioning of cardiac patients with high-intensity interval training (HIIT).[124,156]

Following appropriate screening and evaluation, patients are enrolled in a traditional cardiac rehabilitation program. Once they are familiar with the execution of exercise intervals, an accurate setup of exercise machines trials of HIIT can be introduced. Patients are instructed to exercise at high intensity (>85% of $Vo_{2max}$) for short or ultra-short intervals followed by low-intensity or rest intervals.[126] It is recommended to start HIIT training on seated machines to reduce likelihood of falls on standing machines due to loss of balance, distraction during interval initiation or termination, and dizziness from blood pressure fluctuations. Meticulous blood pressure and telemetry monitoring should take place until the practitioner is confident they have established an individualized baseline response for each patient. Additional considerations are the potential for exercise-induced hypoglycemia—an expected and physiologically beneficial outcome. Symptomatic hypoglycemia can be quickly treated with any source of oral glucose. It is important to reassure cardiac patients that hypoglycemic symptoms are not the onset of cardiac ischemia to reduce negative associations with exercise. HIIT has been shown to provide significant conditioning benefits in the cardiac rehabilitation population with medically supervised selection of candidates.[88]

### *Home-Based Rehabilitation*

Demand for cardiac rehabilitation services is unable to keep up with the current and projected future supply throughout the world leading to innovative approaches to delivery included home-based and "hybrid" programs.[89,125,134] Numerous innovative models are emerging in both cardiac and pulmonary rehabilitation.[28] All models are initiated with an in-person evaluation and followed by various technology driven follow-up contacts. The telerehabilitation approach allows one-on-one live supervised sessions or weekly coaching via video link. A less technology-driven approach includes weekly phone calls and reviews of self-kept activity logs that may also include additional communication via text message.[84] Hybrid programs begin as a traditional facility-based, telemetry-monitored exercise program followed by rapid transition to home-based continuation.[44] Most hybrid programs do not extend telemetry monitoring into the home setting, although with technologic advances this has been demonstrated in at least two foreign studies.[22,80] In addition, current technology allows physiologic data to be collected and relayed to the provider via wearable technology and app- or web-based interfaces.[85,96,141] Regular follow-up appointments, whether in person or via video link or phone call, are recommended to ensure continuity of care. It has been demonstrated that home-based programs are not inferior to facility-based programs, but the medical literature is still developing regarding conclusions of superiority.[64] Home-based cardiac rehabilitation is an emerging and exciting trend. There is a wide variety of solutions being explored throughout the international cardiac rehabilitation community. Exercise has been repeatedly demonstrated to be safe for patients in the cardiac rehabilitation population—extrapolation from this well-accepted practice is leading to new delivery methods to an ever-expanding population.[72,146,152,153]

## Pulmonary Rehabilitation Programs in Specific Conditions

### *Emphysema*

Rehabilitation for patients with COPD is the standard for pulmonary rehabilitation. Goals of a pulmonary rehabilitation program include improving disease management and exercise capacity. Because pulmonary rehabilitation does not improve lung function, the goal of the rehabilitation program is to improve peripheral efficiency and decrease dyspnea. Energy conservation education (how to do a given activity at a lower level of exertion), anxiety reduction, and improved endurance all contribute to improved function and decreased dyspnea. Longer-duration exercise of moderate intensity is often used rather than high-intensity exercise. Investigations have started to evaluate a possible role for HIIT for patients with COPD, but this has not yet been proven to be more effective than the standard training program. Because isometric exercises increase intrathoracic pressures, they should be avoided in patients with COPD.[16] Appropriate supplemental oxygen should be given to maintain saturation greater than 90%, with education to lower supplemental oxygen after exercise back to baseline levels to prevent resting hypercarbia. Patients with COPD generally have relatively modest oxygen needs and can often maintain their oxygen saturation levels with 1 to 6 L of oxygen via a nasal cannula. Bilevel ventilation may have a role for patients with sleep apnea or ventilatory failure, and education for these patients should include the proper use of this modality. For patients being considered for lung volume reduction surgery, pulmonary rehabilitation is considered essential both to qualify for the surgery and after surgery to ensure adequate outcomes.[16]

Airway clearance and chest physical therapy has a role in the pulmonary rehabilitation of patients with substantial secretions. A combination of external percussion devices, vibration devices, and inhaled saline in combination with cough training and huffing may help to mobilize secretions. It is also important to include family training and education about inhaled medications, supplemental oxygen use, and management of equipment.[117]

### *Interstitial Lung Disease*

The basics of a program of pulmonary rehabilitation for interstitial lung disease are the same as for COPD. An essential issue for patients with interstitial lung disease is often profound hypoxemia that requires high-flow oxygen with exercise to maintain adequate saturation for activity. It is essential in this group of patients to avoid chronic hypoxemia to prevent secondary pulmonary hypertension because the coexistence of interstitial lung disease and pulmonary hypertension can lead to a markedly decreased life expectancy. Exercise intensity is often limited in patients with interstitial lung disease by oxygenation rather than dyspnea, and airway secretions are usually not an important issue. For some individuals with severe end-stage disease, there may be ventilatory failure with hypercarbia, but in those patients, rehabilitation may no longer be possible.[144]

Because interstitial lung disease is often progressive, transplant evaluation and education or end-of-life planning may be needed to permit as many patient goals as possible to be achieved.

### *Pulmonary Hypertension*

Patients with pulmonary hypertension have similar limitations as patients with heart failure and share many similar precautions. Effective medical treatment for pulmonary hypertension has made a once-fatal condition into a chronic disease for many patients. Patients with pulmonary hypertension currently have a much longer life expectancy, and improved functional status is essential for maintaining an active life. Major concerns for pulmonary rehabilitation are preventing debility and improving dyspnea. Because hypoxemia can worsen pulmonary hypertension, it is important to maintain oxygen saturation with exercise, and cardiac monitoring may be needed for patients with a history of arrhythmias and right ventricular failure. Education for this group of patients should include a review of their vasodilating medications and supplemental oxygen use. Intravenous and continuous subcutaneous vasodilator infusion is appropriate for a pulmonary rehabilitation program, but, similar to patients with heart failure, there may need to be long warm-up and cool-down periods. For patients with severe pulmonary vascular disease, the program should start with moderate- to low-level exercise. Definitive research of the efficacy and safety of pulmonary rehabilitation for patients with pulmonary hypertension is still ongoing.

### *Ventilatory Failure*

For alert patients on either invasive or noninvasive ventilation for ventilatory failure, a program of pulmonary rehabilitation can help to increase mobility and prevent complications. Exercise programs for patients on nocturnal or intermittent ventilatory support aim to improve efficiency and decrease fatigue while off the ventilator. The details of ventilatory support for patients requiring noninvasive ventilation is beyond the scope of this chapter. Table 27.6 provides an overview of the types of patients who may present with ventilatory failure. A summary of the indications for ventilatory support is listed in Table 27.7.[29]

## Cardiopulmonary Rehabilitation in the Physically Disabled

As the population ages and more patients survive a disabling condition, there is an increase in patients with both physical disability and cardiopulmonary disease. An issue for cardiac rehabilitation for patients with dual disability is the impaired mobility that can impair both evaluation and participation in a rehabilitation program. Individuals who are disabled tend to have lower activity levels, which puts them at increased risk of cardiac and pulmonary disease and may present obstacles for a standard rehabilitation program for a person who is newly disabled and who has preexisting cardiopulmonary limitations. For new-onset cardiac or pulmonary disease, cardiopulmonary rehabilitation is just as important and needs to be considered. Cardiopulmonary primary and secondary prevention also overlaps with the education needed for stroke and peripheral vascular disease and is especially important for patients with physical disabilities because they are often more sedentary with a higher prevalence of obesity and deconditioning. Finally, because mobility in individuals who are disabled requires greater energy expenditure, compromised work capacity from cardiopulmonary disease may impose an even greater degree of disability on an individual who is disabled than an individual who is able bodied.

Cardiopulmonary exercise prescription for individuals who are disabled has to be adapted for the individual disabilities that the patient has. Individuals who have a lower extremity impairment resulting from neurologic or orthopedic conditions can perform upper extremity ergometry or use modified lower extremity exercise equipment, whereas an adapted bicycle ergometer or Airdyne may be helpful for a patient with hemiplegia. The higher $MVO_2$ requirements for upper extremity exercise compared with lower limb exercise should be considered to adapt the cardiac rehabilitation program for patients who are disabled. Patients who are disabled also need to focus on task-specific activities while increasing their aerobic conditioning and endurance, with a goal of lowering the $MVO_2$ for any given task. Because of the expertise in dealing with physical disabilities and understanding the mechanics of motion, physiatrists are particularly well positioned to lead cardiopulmonary rehabilitation programs for the disabled. It is especially important when many traditional cardiac rehabilitation program teams are hesitant to work with patients who are physically disabled because of their lack of experience with physical disability.[120,147]

## Conclusion

Cardiopulmonary rehabilitation is an area where physiatry is uniquely positioned to help manage the patient who is multidisabled, and the multidisciplinary approach is well suited to address the education and team management needed for successful cardiopulmonary rehabilitation. A goal for cardiopulmonary rehabilitation is to increase the access to cardiopulmonary rehabilitation to a greater number of patients, including populations who are underserved in rural and urban areas, women and minority groups, and patients with dual disabilities as they become a larger proportion of the patients seen with cardiopulmonary disease.

## Frailty

### Movement and Function

Movement is an essential part of human life and is important for the preservation of function throughout the entire life cycle. Since the beginning of human history, a high degree of physical activity has been required to maintain a livable environment and to secure adequate nutrition to ensure survival. It is only following industrialization, a relatively recent event from an evolutionary point of view, that the diseases and conditions associated with inactivity and immobility began to manifest themselves throughout human societies. Obesity and the resultant conditions of diabetes, hyperlipidemia, and decreased cardiopulmonary reserve have increased steadily throughout the past century. Much attention is focused

**TABLE 27.6 Causes of Ventilatory Failure**

| Central Hypoventilation | Respiratory Muscle Failure | Chronic Respiratory Disorders | Other |
|---|---|---|---|
| Intracranial hemorrhage | Amyotrophic lateral sclerosis | Chronic obstructive lung disease | Congestive heart failure |
| Arnold-Chiari malformation | Congenital myopathies | Bronchopulmonary dysplasia | Congenital heart disease |
| Central nervous system trauma | Botulism | Cystic fibrosis | Tracheomalacia |
| Congenital and central failure of control of breathing | Muscular dystrophies | Interstitial lung disease | Vocal cord paralysis |
| Myelomeningocele | Myasthenia gravis | Kyphoscoliosis | Pierre Robin syndrome |
| High spinal cord injury | Phrenic nerve paralysis | Thoracic wall deformities | |
| Stroke | Polio/postpolio | Thoracoplasty | |
| Central alveolar hypoventilation | Spinal muscular atrophy | | |
| | Myotonic dystrophy | | |

**TABLE 27.7 Indications for Ventilatory Support**

| NIV Decision Tree | Mild | Moderate | Severe |
|---|---|---|---|
| Clinical syndrome | Substantial daytime $CO_2$ retention (>50 mm Hg with normalized pH) | Mild daytime or nocturnal $CO_2$ retention (45–50 mm Hg) with symptoms of hypoventilation | Substantial nocturnal hypoventilation or hypoxemia |
| Clinical diagnoses | COPD<br>Neuromuscular disease | Moderate neuromuscular disease<br>Paralysis<br>Chest wall deformity | Central hypoventilation<br>Marked obesity<br>Severe neuromuscular disease<br>End-stage lung disease |
| Maximum medical management options | Optimal medication<br>Pulmonary toilet<br>Airway management<br>Avoidance of pulmonary exacerbations | Add management of secretions<br>Airway protection<br>Consider nocturnal NIV rest with daytime off NIV | Treat reversible pulmonary conditions<br>Maximize posture and other supports<br>Considerations for augmentative communication and ensuring adequate nutrition |
| Indications for tracheostomy over NIV | Uncontrollable secretions | Uncontrollable secretions<br>Chronic aspiration and chronic pneumonias<br>Obstructive sleep apnea with failure to improve with continuous positive airway pressure<br>COPD with severe hypoventilation | Uncontrollable secretions<br>Chronic aspiration and chronic pneumonias<br>Failure of NIV<br>Inability to manage NIV<br>Patient/caregiver preference |

*COPD*, Chronic obstructive pulmonary disease; *NIV*, noninvasive ventilation.

on the "westernization" of diet, although less attention is focused on the "westernization of physical activity levels."

As physicians concerned with function, physiatrists intuitively understand the dangers of activity reduction in all settings from all causes, both medical and environmental. In fact, often physiatrists are the only physicians who have familiarity with the maintenance of function via physical activity using therapists, nurses, and family members. The knowledge of how to modify physical and social environments to maximize functional movement and overall function for their patients allows physiatrists to improve and maintain function in their patients. The physiatric focus on activities of daily living (ADLs) is an effort to return functional movements to an individual who is disabled, allowing them to maintain their baseline degree of physical activity required for autonomy and independent movement.

## Physiology and Consequences of Inactivity

The link between physical activity and cardiovascular disease has been well described since the 1950s,[102,103] when the relationship between workplace activity levels were directly related to higher rates of cardiovascular events. It is no surprise that less physically active daytime behavior (e.g., mail sorters vs. mail deliverers) affected the development of cardiovascular disease. A more interesting finding is the strength of this association; primarily seated workers developed almost twice the rate of cardiovascular disease. More research investigating and elucidating the cellular biology of inactivity needs to be done until this area of physiology is as well understood as exercise physiology.

Currently, the global workforce is becoming more sedentary in numerous sectors as desk-based work responsibilities dominate the work day and after-hours couch-based recreational activities, including home theaters, media centers, and ubiquitous social media, create prolonged voluntary immobilization after work. In the United States the amount of daytime sedentary hours is high, as revealed by data from the *National Health and Nutrition Examination Survey* (NHANES) database, which showed that 54.9% of waking hours in the population studied was spent during sedentary activities,[95] with late adolescents and older individuals being the most sedentary.

Rising "sedentarism" is not only an American phenomenon. In Australia a large population-based study found that sedentary behavior (television viewing time) was positively associated with abnormal glucose metabolism and metabolic syndrome.[35,36] More concerning is that these associations were preserved even when controlling for what would be considered active individuals who participated in sustained and moderate-intensity recreational activities. Prolonged physical inactivity appears to be a unique risk factor for maladaptive energy metabolism. In the future it is possible that number of hours spent sitting will be recognized as a risk factor for the development of cardiovascular disease. In the context of this discussion, these observational data support the hypothesis that the physiology of inactivity is a risk factor for poor health outcomes in numerous settings. This supports the idea that there should be a paradigm shift in how all physicians view physical inactivity in their patients, especially in the hospital setting where the physiatrist is in a key position to increase patient mobility.

In addition to increased cardiovascular risk, there are numerous associations between inactivity and poor health outcomes, including the metabolic syndrome, deep vein thrombosis, obesity, and serum insulin levels.

### *Metabolic Syndrome*

Although there have not been studies to elucidate the molecular biology linking prolonged sitting to metabolic syndrome, epidemiologic data are compelling. The metabolic syndrome is the presence of three out of five of the following findings: central obesity, elevated blood pressure, low serum high-density lipoprotein cholesterol, high triglycerides, and elevated fasting glucose. Patients with this constellation of findings are at increased risk for the development of cardiovascular disease and diabetes.[1] In recent years, the recognition of the metabolic syndrome and its prevention has produced a rich literature specific to this syndrome.

Prolonged inactivity (sitting) more than doubles the risk for development of metabolic syndrome.[41] Development of the metabolic syndrome has been shown to increase with each additional hour of sedentary television viewing, as opposed to having television in the background during other household activities.[35]

### *Deep Vein Thrombosis*

The correlation with prolonged sitting and development of deep vein thrombosis has been well described.[61] This can occur even in active individuals who are immobile for prolonged periods of time. Case reports from varied settings have reported seated individuals who developed deep vein thrombosis since the 1950s. These have included observations from air raid shelters, sitting in theater, sitting on extended airplane flights, and even prolonged sitting during video game playing.[104,108] Presumed causes include rheologic changes and hemoconcentration.[58,66]

### *Obesity*

The relationship between physical activity and body mass index is supported by the medical and epidemiologic literature. In varied populations, pediatric, adult, or older adult individuals who have a lower level of baseline physical activity generally have a higher rate of obesity. Sedentary behavior is a reversible cause of obesity in all populations,[9,38,135] and improvement of physical activity levels should begin with school-aged children.[50,154] This is especially true because there is evidence that an obese child has a significantly greater likelihood of continuing life as an obese adult.[57,157] Even high levels of physical activity in athletic adults may not be able to compensate for the deleterious effects of sedentary behavior.[68]

### *Insulin*

Insulin resistance is a component of the metabolic syndrome, and thus it is reasonable to assume that an association would exist between amount of insulin present and sedentary behavior. Reduced leisure time physical activity levels are associated with higher levels of insulin at baseline.[42]

Numerous studies have demonstrated the relationship between prolonged sitting, obesity, and the development of type 2 diabetes, an insulin resistance state. More interestingly, however, is the suggestion that insulin levels are elevated in individuals who have prolonged sitting, even in the presence of regular exercise.[56]

## Frailty Syndrome

The frailty syndrome is a recognized syndrome of decreased ability to adapt to stressors accompanied by reduced physiologic reserves and reduced energy metabolism. The frailty syndrome has been reported to vary widely depending on the population studied, ranging from 4% to 60%.[45] The frailty syndrome is clearly associated with advanced age and becomes more prevalent in older groups studied; however, it is considered to be a separate syndrome and not a variant of normal aging. If it were considered the end result of normal aging, it would be reasonable to expect all individuals to acquire the frailty syndrome if they lived long enough, which they do not. The potential for confusion exists when the descriptor "frail" is confused with the frailty syndrome. A universally accepted definition or set of variables to diagnose the frailty syndrome has not yet been agreed upon; however, the conversation in the medical literature is ongoing and evolving. Substantial opportunities exist for physiatric contribution to this discussion because the frailty syndrome is defined in most tools as having a strong functional component.

A true consensus on the definition of the frailty syndrome has yet to be agreed upon internationally; however, the following findings are discussed in the medical literature as possible variables in evaluating individuals thought to be at risk for the frailty syndrome:

- Increased inflammatory response
- Decreased cardiopulmonary and renal reserve
- Sarcopenia
- Weight loss
- Exhaustion
- Weak grip strength
- Slow walking speed
- Low level of physical activity

There are numerous frailty screening tools being used in a variety of settings. Two of the most commonly cited tools in the medical literature include the Fried Frailty Phenotype and the "Rockwood Indices" from the Canadian Study of Health. These measurement tools can be of use to physiatrists depending on the practice setting in which they are used.

## Fried Frailty Phenotype

Fried and colleagues[45] at Johns Hopkins University developed a screening tool that identifies frailty based on a positive score in three out of five possible domains: weight loss, exhaustion, low physical activity, slow walking speed, and reduced grip strength. A positive response was assigned a score of 1 or 0 for each category.

### *Weight Loss*

A positive value of 1 was assigned when participants responded "yes" to the following question, "In the last year, have you lost more than 10 pounds unintentionally?" Because there is considerable room for self-reported bias, follow-up measurements to confirm weight loss are recommended to confirm the initial findings.

### *Exhaustion*

Two questions extracted from the Center for Epidemiologic Studies Short Depression Scale (CES-D 10) were used as indicators of exhaustion: "I felt that everything I did was an effort" and "I could not get going." Scoring was based on the strength of participants' agreement with these statements. A positive score was assigned a value of 1.

### *Low Physical Activity*

Kilocalories per week expended were calculated with a self-reported description of voluntary activities adapted from the Minnesota Leisure Time Activity Questionnaire. This included questions about walking, chores, outdoor gardening, and numerous types of exercise. Interpretation of the answers should be clarified to ensure that negative answers are not based on different settings (urban vs. suburban) or culture. A positive value of 1 was assigned for participants with the lowest 20% of activity.

### *Slow Walking Speed*

Measurement was the time in seconds required to walk 15 feet. Stratification by gender and height took place. The slowest 20% of the population studied was defined as receiving a positive score and a value of 1.

### *Reduced Grip Strength*

Results were stratified based on gender and body mass index with values recorded in kilograms with a standard dynamometer. Participants who scored in the lowest 20% after adjustment for gender and body mass index were assigned a positive value of 1.

**TABLE 27.8 Example Variables from the Canadian Study of Health and Aging Frailty Index**

| Medical | Functional | Psychological | Neurologic | Musculoskeletal |
|---|---|---|---|---|
| Lung problems | Problems cooking | Depression | Tremor at rest | Impaired mobility |
| Congestive heart failure | Urinary incontinence | Delirium | History of Parkinson disease | Poor muscle tone in limbs |
| History of stroke | Toileting problems | Restlessness | Seizures | Poor standing posture |
| Skin problems | Falls | Mood problems | Cognitive symptoms | Poor coordination of trunk |

Modified from Jones D, Song X, Mitnitski A, Rockwood K: Evaluation of a frailty index based on a comprehensive geriatric assessment in a population based study of elderly Canadians, *Aging Clin Exp Res* 17:465–471, 2005.

After scores are totaled, individuals who score 0 are considered "robust." The presence of one to two of the criteria listed earlier are considered to be "prefrail," and three or more positive criteria are considered "frail." This phenotypic description has been correlated with notable clinical outcomes that are recognized as important in the geriatric population including falls, hospitalizations, and mortality. This clinical applicability and relevance to important measurable outcomes has made the Fried Frailty measure a popular tool in clinical research. There are no laboratory tests or psychosocial components taken into account when determining a score with this scale. Using these criteria, Fried identified individuals who met the criteria for the frailty syndrome in 7% of the 4317 participants included in the Cardiovascular Health Study, 30% of the participants older than 80 years of age, and 28% of the Women's Health and Aging Studies.[45]

## Canadian Study of Health and Aging Measurement Tools

The "Rockwood Index," also known as the Canadian Study of Health and Aging (CSHA) Frailty Index, and The Clinical Frailty Scale were both derived from the CSHA dataset, a prospective cohort of more than 10,000 participants. Both of these scales have been widely used in medical studies. The 70-item CSHA Frailty Index is driven by clinical judgment. This is a detailed tool in which clinical deficits are scored based on a 70-item index. The items include self-reported functional activities, mood, and motor symptoms, as well as signs and symptoms derived from medical history and physical examination. Each deficit is assigned a value between 0 and 1.0 to give a total score, which is then divided by 70. Some examples of variables collected are listed in Table 27.8, with division into categories provided as a conceptual framework.

This index determines clinical deficits and allows scoring based on evaluating participants for accumulation of impairments. The Clinical Frailty Index was developed as an attempt to integrate clinician judgment into a formal and universally applicable model to evaluate frailty. The authors recognized that simply evaluating frailty based on a limited number of phenotypic variables may correlate with mortality but does not give considerably more information across all populations. Although it is of benefit to identify participants at risk for increased overall mortality, it does not guide the physician to develop interventions that can address specific and more importantly correctable impairments. The Frailty Index gives structure to an intensive review of deficit accumulation that can serve as a starting point for interventions.

These same authors recognized the utility for a shorter more clinically oriented scale that could be used by clinicians across numerous specialties. In 2005, they developed and validated the CSHA Clinical Frailty Scale.[67] This is a descriptive scale with seven categories from "very fit" to "severely frail." It has been demonstrated to be an effective measure of frailty and offers predictive information about probability of survival and likelihood of institutionalization. This is a judgment-based scale that would be more applicable in the physiatric setting, whereas the more time-intensive Frailty Index would give more specific information to a primary care provider or geriatrician. Both tools are validated instruments of benefit in the research setting (Table 27.9).

## Frailty: A Complex Syndrome

In addition to the functional, psychological, and musculoskeletal changes, there are also altered organ system and homeostatic responses in the frailty syndrome. These include a reduced capacity to maintain homeostasis and an increased vulnerability to stressors caused by lower energy metabolism, sarcopenia, altered hormonal activity, and decreased immune function.

Changes in the endocrine axis, specifically the growth hormone/insulin-like growth factor 1 (GH/IGF-1) axis, affect numerous metabolic systems. Hepatic IGF-1 production is controlled by pituitary growth hormone secretion and is essential for normal metabolic processes in adults. Because growth hormone secretion declines with normal aging, IGF-1 levels also decline; this is often referred to as the "somatopause,"[69] which accounts for the normal age-related decline in endocrine function. Decreasing circulating levels of endocrine hormones contribute in part to osteoporosis, alteration in muscle/fat ratio, and cognitive decline in addition to sarcopenia. There is an association between the frailty syndrome and abnormally low IGF-1 levels.[91] Routine testing of IGF-1 levels is not recommended because it is neither diagnostic nor cost effective.[76] However, appreciating the metabolic setting in which the frailty syndrome is more likely to occur provides a framework for understanding this complex syndrome. In the Women's Health and Aging Study, participants who had disabilities in mobility and disabilities in ADLs were more likely to have an increase in the proinflammatory cytokine interleukin-6 (IL-6).[26] The combination of decreased IGF-1 and increased IL-6 could contribute to a theoretical shift from the normal slowly decreasing anabolic state of aging to a rapidly increasing catabolic state seen in the frailty syndrome. As the population ages, the incidence of the frailty syndrome will also increase. This complex syndrome will continue to be elucidated as advances in the molecular biology of normal aging progress. For the physiatrist, early recognition of the frailty syndrome may allow multidisciplinary intervention with the intended goal of preserving function as long as possible for these patients.

**TABLE 27.9 Comparison of Tools Used to Measure Frailty**

| Measurement Tool | Variables | Scoring | Utility |
|---|---|---|---|
| Fried Frailty Phenotype[45] | Domains of physical function | Each of five domains is scored between 0 and 1. Higher scores represent greater disability.<br>0: "Robust"<br>1–2: "Prefrail"<br>3+: "Frail" | Correlated with falls, hospitalizations, and mortality<br>Popular tool in clinical research |
| CSHA rules-based definition of frailty[129] | Rules-based ranking from fitness to frailty | 0: Independent and continent<br>1: Bladder incontinent only<br>2: Needs assistance with one or more ADLs, bowel or bladder incontinence, cognitive impairment with dementia<br>3: Totally dependent for transfers or at least one ADL, completely incontinent, dementia | Predictive of death or admission to an institution |
| CSHA Frailty Index[67] | Deficits-based ranking | 70 deficits scored in varying increments from 0–1.0. Total divided by 70 to give an index score | Predictive of frailty and death |
| CSHA Clinical Frailty Scale[67] | Disabilities, comorbidities, and cognitive deficit–based scoring | 1: Very fit<br>2: Well<br>3: Well, with treated comorbid disease<br>4: Apparently vulnerable<br>5: Mildly frail<br>6: Moderately frail<br>7: Severely frail | Predictive of death or admission to an institution |
| CSHA Function Scale[24] | ADL-based scoring | 12 ADLs scored as follows:<br>0: Independent<br>1: Needs assistance<br>2: Incapable | |

*ADL,* Activity of daily living; *CSHA,* Canadian Study of Health and Aging.

## *Zero Physical Activity: Hospital Immobility*

**Traditional Hospital Practices (Bed Rest, Sedation, and Immobilization).** There has been a long-standing culture of bed rest in hospital culture. The concept of "convalescence" is that an ill person's strength returns gradually and is enhanced through greater than normal rest. This word is believed to have entered regular use in the late 15th century[31] at a time when patients who were ill had few medical options other than rest. The deeply held belief that hospitals are places to "rest" influences sedation practices and encourages the overuse of bed rest despite there being a clear understanding of the dangers of immobility.

This is especially true in the traditional critical care setting.[118] Long-standing provider beliefs include the idea that undersedation during mechanical ventilation is painful, traumatic, and panic-inducing despite ample data to the contrary.[81,128] In a recently published study that explored nurse sedation practices in the ICU, 80% of nurses surveyed believed that sedation is necessary for patient comfort and 87% of the nurses surveyed would want sedation if they were ventilated themselves.[52] Although some degree of sedation is usually required, 56% of the nurses surveyed believed that patients who are "spontaneously moving hands and feet" are undersedated. Although overuse of sedation is associated with an increase in posttraumatic stress disorder (PTSD)[128] and prolonged mechanical ventilation[81] with the accompanying complications of total immobility, it is still a widely held belief in many institutions that "rest is best." These practices lead to accelerated and iatrogenic deconditioning resulting from immobilization.

**Iatrogenic Immobilization and Deconditioning.** The dangers of immobilization have been understood for a long time (Table 27.10). The often-cited 4% to 5% loss of muscle strength for each week of bed rest was derived from studies that involved young healthy test individuals without underlying disease or musculoskeletal conditions.[19] It is likely that the rate of deconditioning is even faster in older adult patients with multiple comorbidities, because ambulatory function and ability to perform basic ADLs have been shown to decline in one-third of hospitalized patients older than the age of 70 years.[133] Some of the complications of immobility include orthostatic intolerance, skeletal muscle changes, joint contractures, pulmonary atelectasis, urinary stasis, glucose intolerance, and pressure ulcers.[78] Traditionally, physiatrists have served as advocates for increased patient activity in the hospital setting because they evaluate and identify patients who can benefit from physical and occupational therapy.

## *Early Mobilization*

**Rationale.** The importance of early mobilization is well accepted as a "best clinical practice" in every hospital setting, not just the ICU. As the complications of immobilization became more widely understood, the importance of early mobilization throughout hospital organizations becomes a logical institutional goal, ideally approached through the quality improvement methodology.[18] Less universally agreed is how to design and implement a multidisciplinary early mobilization program and how to effect the accompanying culture change that is required for success. The physiatrist, working closely with colleagues in nursing, critical

**TABLE 27.10 Effects of Immobility**

| System | Impact of Immobility | Potential Functional Impact |
|---|---|---|
| Musculoskeletal | Atrophy of skeletal muscles<br>Decreased core strength<br>Joint contractures | Reduced muscle power<br>Reduced standing balance<br>Shift in center of gravity |
| Cardiovascular | Deep vein thrombosis<br>Reduced peripheral vascular resistance<br>Orthostatic hypotension<br>Reduced venous return | Potential lower extremity edema<br>Pooling of blood in lower extremities<br>Syncope/presyncope<br>Venous stasis |
| Pulmonary | Atelectasis<br>Pneumonia | Decreased vital capacity<br>Decreased endurance |
| Psychological | Delirium<br>Depression<br>Posttraumatic stress disorder | Extended length of stay<br>Decreased activities of daily living<br>Chronic depression/ anxiety |
| Dermatologic | Pressure ulcers | Prolonged hospital admission |

Modified from Kortebein P: Rehabilitation for hospital-associated deconditioning, *Am J Phys Med Rehabil* 88:66–77, 2009.

care, physical therapy, and occupational therapy is ideally suited to take a major role in the effort to bring mobilization to all patients who are hospitalized. The ICU is an ideal setting in which to initiate such a program because of the increased staffing to patient ratios, extended length of stay, and awareness within the critical care community as to the important role of physical medicine and rehabilitation in these programs.

**Culture of Immobility.** To successfully mobilize patients who are hospitalized, one must understand the many reasons that patients are ordered to bed rest and immobilization. Only then can the root causes of reduced patient activity be addressed. The concept of therapeutic bed rest can be a difficult idea to challenge. Therapeutic bed rest has been recommended for almost every medical problem at some point in medical history, including a variety of cardiac and pulmonary conditions in both the pediatric and adult populations.[21,46,60] In the intensive care setting, long-held beliefs that the experience of mechanical ventilation was traumatic and frightening for patients gave rise to a culture of complete sedation and resultant immobility. The idea that patients who were unconscious would recover faster, "fight the vent" less, and be spared psychological suffering was essentially unchallenged. Similar to the experience of patients with cardiac disease before unit-based cardiac rehabilitation efforts began in the 1970s, survivors of critical illness were profoundly weak with substantial disabilities resulting from their prolonged immobilization. This was often interpreted as proof of how tenuous their conditions were at the time of presentation, rather than the side effects of immobilization and oversedation.

**Culture of Mobility.** The first step to increase patient activity is gaining the trust and "buy-in" of colleagues in medicine and nursing. The importance of having evidence-based discussions with colleagues cannot be overstated. The common ground of all healthcare providers is commitment to patient care and improved functional outcomes. There is currently a developing medical literature on the benefits of early mobilization.[97,107,116] Journal clubs, consultative rounds, and a strong inpatient presence contribute to an understanding of the physiatric approach and will provide an appropriate venue for discussions about early mobilization.

**Physiatric Involvement.** As a physician who understands the important role of physical, occupational, and speech therapy, the physiatrist is ideally suited to emphasize the vital role that these services play in the hospital setting. Because of hospital-bundled payments, physical therapy expenditures have traditionally been seen as cost centers and not profit centers by hospital administrators. To address this perception, the physiatrist should be well aware of the importance and robust discussion taking place in the medical literature about the cost-effectiveness as well as clinical use of early mobilization programs in a variety of medical settings.[8,62,90,145] Familiarity with the current literature demonstrating the multiple savings to an institution is key in acquiring the resources needed to implement an early mobilization program. Having a physiatrist as part of the implementation team is optimal to represent the interests and contributions of the entire spectrum of physical medicine and rehabilitation providers.

**Ambulatory Devices.** Hospitalized patients who use assistive devices normally are generally unsafe to ambulate without their usual devices while in the hospital. Because of staffing ratios and constant surveillance, ambulation in the ICU would not take place unassisted; however, in a noncritical unit a mobilization program would need accommodation for patients who can ambulate independently as well as with assistance. A recently developed Fall Prevention Tool Kit includes bedside signs identifying risks and required assistive devices if they are determined to be necessary for safe ambulation.[37] In one study, 45% of falls were related to attempts by patients to reach the bathroom.[150] It is logical that removing assistive devices from ambulatory patients entering the hospital will result in increased falls when patients attempt to ambulate independently.

**Training.** Coordinated interdisciplinary training before implementation is essential for any mobilization program. Although early mobilization is often focused on physiatric oversight and physical therapy services, without appropriate coordination by all disciplines involved, successful early mobilization cannot take place. Multidisciplinary simulation training with case-based scenarios should take place in the unit where mobilization is planned. All members of the healthcare team should have clearly defined roles before simulation training to experience the difference between standard multidisciplinary care and interdisciplinary coordination of care, which is at the heart of early mobilization. Unit-based simulation training, ideally with an actor as the patient, is optimal when possible. Cases provided should address medical emergencies and the challenges of physical coordination of care. Allocating time following simulation training for team members to discuss their experiences further facilitates the team-building experience.

## Treatment Considerations in the Critical Care Setting

### *Hemodynamic Instability/Orthostatic Hypotension*

Patients hospitalized in critical care units almost universally have hemodynamic instability. The systemic inflammatory response syndrome causes peripheral vasodilation, cardiac dysfunction, capillary leak, and circulatory shunting leading to hypovolemia. A constantly changing cardiovascular environment makes daily evaluation essential. As a result of vasodilation, patients who are critically ill may be unable to tolerate bed elevation, let alone seated positioning. Observing hemodynamic response to simple turning is a bedside test of hemodynamic tone that is easily carried out by a single provider. Patients who cannot tolerate trunk elevation can be treated in a supine position with range of motion and progressive resistance. It is important for treating therapists to continually observe blood pressure response to intervention. It is strongly recommended that treating therapists consult with nursing providers to discuss any changes since last treatment resulting from rapid changes in physiologic state. With constant surveillance and gradual progression, mobilization of the patient who is critically ill is unlikely to produce any unexpected events.[18]

### *Ventilatory Dependence*

Hypoxemic failure causes numerous pathophysiologic effects culminating in requirement for ventilator support. These include hypoxemic failure caused by ventilation/perfusion (V/Q) mismatch, shunting, and altered oxygen exchange properties. Hypercapnic failure causes decreased minute ventilation relative to physiologic demand, especially in the setting of critical illness complicated by increased dead space ventilation. Coordination of therapy in conjunction with ventilator management should take place between physical medicine and rehabilitation and respiratory therapy. In some cases, therapists may be given parameters by the primary team as to titration of oxygen during treatment sessions. Oxygen titration should always take place in coordination with respiratory therapy or nursing. Similar to hemodynamic monitoring, ventilator status and oxygen saturation can vary quickly in response to activity and should be monitored at all times. During active ambulation, a respiratory therapist is essential to monitor oxygenation and the position of an endotracheal tube.

### *Postparalytic/Intensive Care Unit–Acquired Weakness*

ICU-acquired weakness is common following hospitalization in the intensive care setting. Approximately 50% of patients with prolonged mechanical ventilation, sepsis, or organ failure have some degree of neuromuscular dysfunction.[143] The presence of ICU-acquired weakness can cause abnormalities at any point in the gait cycle. Endurance is reduced in all patients following prolonged bed rest with or without paralytics. Ambulation trials should begin with standing at bedside and progress only out of the patient room once the entire team is assembled. This will ensure patient safety and continuous monitoring in the face of global weakness. Early and aggressive physical therapy intervention has been shown to improve recovery of muscle strength in patients in the ICU.[18,140]

## Psychology of the Patient in the Intensive Care Unit

### *Posttraumatic Stress Disorder*

Survivors of critical illness often have symptoms of PTSD. In a recently published study, 35% of ICU survivors following admission for acute lung injury reported PTSD symptoms during the 2-year period following critical care admission.[20] Symptoms of PTSD can be persistent, given that 62% of patients who reported symptoms confirmed their persistence at 24 months. Of these, 50% had taken psychiatric medications and 40% had seen a psychiatrist since hospital discharge.[20] Clearly, the complications following an episode of critical illness are multifactorial. Although it is too early to definitively state that early mobilization programs reduce symptoms of PTSD, it is likely that the benefits of such a program include reduced psychiatric complications.

### *Delirium*

Delirium in the intensive care setting frequently complicates patients' hospital courses with resulting negative health outcomes. It is important that all providers in the intensive care setting recognize delirium and understand how it can impact on early mobilization programs. By definition, delirium is a change in cognitive function hallmarked by a fluctuating course over a short period of time (hours to days).[3] In the critical care setting, delirium is not just a descriptive term; it is a measurable medical syndrome associated with poor outcomes. The Confusion Assessment Method (CAM) and CAM-S (short form) are measurement tools that have been validated for use in this setting.[65]

It is important for therapists to understand that the interventions the provider has been shown reduce delirium and improve functional outcomes.[138] Recent critical care practice guidelines state, "We recommend performing early mobilization of adult ICU patients whenever feasible to reduce the incidence and duration of delirium."[11] For the rehabilitation provider, even though the fluctuating nature of delirium has the potential to interfere with physical and occupational therapy, it is essential that therapy intervention be provided whenever possible. This often necessitates frequent reevaluations throughout the day until an appropriate window of intervention can be found.

### *Transition of Care*

The importance of care transitions is receiving greater attention in both the medical literature and the administrative realm of hospital management. An appreciation for the impact of poorly structured systems to transition care from one medical setting to another has become an area of intense focus across the healthcare spectrum. Transitional care programs have been studied using different combinations of licensed providers including social workers, pharmacists, nurses, and physicians with most of these programs showing some degree of benefit.[27,77,111] The use and feasibility of transitional care programs has been studied in varied environments where the transition begins including ICU, hospital, nursing home, emergency department, and rural care centers.[10,23,27,49,111] The effect of early mobilization programs on care transition has not been studied, but the current interest in ensuring continuity of communication emphasizes the importance in developing and studying systems to ensure transition of physical and occupational therapy. Without attention to appropriate transition of care of rehabilitation services, the benefits gained during early mobilization are rapidly lost following transfer. This is an area of emerging interest that justifies research efforts on the part of all disciplines involved in early mobilization programs.

## Application to Patients Not in the Intensive Care Unit

Although the importance of early mobilization in the critical care setting has received substantial attention, the principles of early mobilization are applicable throughout the healthcare continuum. Evaluating patients at risk for immobility or reduced mobility followed by appropriate referral to trained providers can increase patient activity levels throughout a medical system. The type of provider selected is determined by the degree of assistance required for a patient to safely increase their activity level. In the hospital setting, "bed rest" orders should be replaced by systemized evaluations regarding safe patient activity levels. Allowing patients who are hospitalized to use bedside commodes and assistive devices where appropriate is an economical way to increase patient activity without skilled intervention. Providing assistive devices to appropriate and carefully selected patients who are hospitalized can increase mobility and reduce incidence of falls in the context of a structured fall-prevention program.[37]

### *Community Mobilization*

**Healthy People 2020.** The US Department of Health and Human Services has initiated five "Healthy People" initiatives since 1979. These are comprehensive public health programs designed to provide structure and guidance to achieve more than 1200 objectives to improve the health of all Americans. Each health objective has a reliable data source, baseline measurement, and target for specific improvements to be achieved by the year 2020 (Table 27.11).[113]

The Healthy People 2020 website is a rich resource that includes leading health indicators, tips and tools for implementation of programs, and a consortium of organizations and agencies committed to achieving the Healthy People 2020 goals.[113] Community mobilization programs can play a role in achieving targets for some of the most important leading health indicators listed as follows.

**Wellness Centers.** Chronic diseases and their cost are responsible for approximately 75% of US healthcare costs.[122] The Prevention and Public Health Fund was established by the Patient Protection and Affordable Care Act of 2010 to administer the Community Transformation Grant program. More than 60 grantees have been funded to support Americans in adopting healthier lifestyles including healthy eating, active living, and tobacco-free living. Funding opportunities exist within this new structure to explore evidence-based community wellness initiatives.

Although the causes of preventable chronic diseases in the United States are clear (cigarette use, lack of physical activity, calorie-dense/nutrient-poor dietary patterns), the solutions are as varied as the communities throughout the country. There are many unique and innovative programs that intervene at different stages of the disease process. Preventative programs are some of the most appealing as long as the cost-benefit ratio strongly supports ongoing investment. Some programs may involve disease-specific secondary prevention, such as a traditional cardiac rehabilitation program. Others may be more broadly based and focus on increasing fitness in the context of a community.[51] Although wellness programs with general physical activity goals may be common in senior centers, some of the most innovative approaches to the overarching problem of obesity in the United States are taking place in community centers with a focus on modifying heath behavior in the context of community-based family education and counseling. The Growing Right Onto Wellness (GROW) program in Nashville, Tennessee, represents an innovative program with a population of 600 parent-child pairs who received educational interventions from 2013 to 2016 with a goal of preventing childhood obesity from developing in children.

Wellness centers can be freestanding but with community resources (community centers, houses of worship, schools, and employers) because locations for population-specific wellness programs are an efficient way to improve health outcomes.

Regardless of the population being targeted, all wellness programs can be developed based on a standard approach, listed as follows:

**Step 1**: Organize Advocates and Advisors

Wellness programs are multidisciplinary in nature. Identifying a group of committed and interested leaders and creating a working group is a logical first step. Having the input and involvement of advocate community members at an early stage can improve awareness and uptake in later stages.

**Step 2**: Determine the Target Population

The most common wellness programs are either community or workplace based in scope. A work-based program will by definition be multidisciplinary. A physiatrist would be ideally suited to spearhead this type of intervention within a medical center based on their understanding of function, occupational health concerns, and expertise in multidisciplinary work.

**Step 3**: Needs Assessment via Health Risk Appraisal

Surveying the target population by questionnaire will yield the most accurate and direct information regarding what types of conditions a population is at risk for. This in turn will guide the development of appropriate interventions. In the community setting, an open forum or informal sampling of health concerns and interests can serve the same purpose. A needs assessment survey is also an opportunity to determine the preferences and interests of the target community. For example, there are numerous ways to increase physical activity levels. Early identification of prevailing interests will guide the working group to direct its efforts where they will yield the greatest participation.

**Step 4**: Identify Health Goals for Intervention

Information from Health Risk Appraisals will direct the priorities for each organization or community. Some examples include: Exercise/Physical Fitness, Tobacco Reduction, Stress Management, Back Care, Nutrition, Weight Control, and Mental Health. There is no way to predict what topics will be of importance to a population without sampling opinion. In a 1994 study that queried medical students regarding priorities for a health promotion program, 49% were interested in financial planning initiatives and only 12% were interested in alcohol and drug abuse programming.[139] Targeted polling will inform where resources should be directed for maximum impact.

**Step 5**: Community Outreach and Resource Identification

All communities have resources: human, physical, and financial. Identifying available space, facilities, and activity leaders will clarify what types of programs are possible to develop. Publicizing the need for a service, provider, or programing space may identify participants who are willing to create programs of interest at little to no cost. Community leaders including those of faith-based organizations, politi-

**TABLE 27.11 Healthy People 2020 Leading Health Indicator Target Goals**

| Physical Activity | Adult Obesity | Obesity in Children/Adolescents |
|---|---|---|
| 20% of adults 18+ meet the current federal physical activity guidelines | Reduce incidence of obese adults by 14% from 36% to 30% by 2020 | Reduce incidence of obese children between ages 2 and 19 years from 17% to 15% |
| Goal met | Goal not met | Goal not met |

cal organizations, unions, and neighborhood organizations are often well aware of what resources are available for use. If community leaders have been involved from inception, they are more likely to be a committed stakeholder.

**Step 6**: Program Development

After analyzing community data, preferences, and resources, the project leadership should be able to draft an initial plan with staged interventions and a time line for deployment. Having a written document of program components, expected outcomes, and desired metrics before beginning will not ensure success, but it will create a structure where leadership can learn from experience what is working and what needs to be adjusted. There are ample resources available at the Wellness Council of America website: www.Welcoa.org.

**Step 7**: Evaluation Plan

Every intervention must have an evaluation plan before deployment. Specific data sampling methods should be agreed upon before implementation as should meetings to review and interpret collected data. There should be object measures collected on a regular basis that are reviewed by leadership to determine if goals are being met and, if not, what action is necessary.

**Step 8**: Incentive Plan

Human behavior is driven by reinforcement. Creating sustainable incentives (e.g., t-shirts vs. weekend getaways) will contribute to success over time. Newsletters, parties, recognition meals, snacks, and gift cards are all popular workplace incentives that have the added value of increasing visibility of a new program for nonparticipants. Workplace competition can be a healthy way to promote a program, especially if a substantial incentive is being offered as reward to the winning team. Geographic competitions, departmental teams, and intradisciplinary teams can all contribute to employee "buy-in" and team building.

**Step 9**: Implementation

Before implementation, additional community members, division heads, or other leaders should be included as part of the implementation team. It is just as challenging to develop programs with too many members as it is to implement a program launch with too few members on the launch team. Leaving some choices to be decided by the entire implementation team will improve participation and sense of involvement for all team members, leading to better enthusiasm and community participation following deployment.

Spearheading or participating in implementation of an employee wellness program can be a gratifying and worthwhile way to improve function on a population level rather than at the individual level. Highlighting the importance of the unique physiatric approach in the healthcare setting, as well as educating communities as to the important role physiatrists can play in keeping their community members healthy and active, are additional benefits to involvement in these types of programs.

## Renal Failure

### Physiology of Renal Failure

Scientific advances have expanded our understanding of what constitutes renal failure. Once thought to be solely attributable to volume overload, urea buildup, and hypocalcemia, it is currently known that uremia is a complex syndrome caused by the accumulation of organic waste products, some of which have yet to be identified. Hemodialysis cannot fully duplicate the complex actions of the nephron, which filters blood, reabsorbs water and solutes, secretes toxic substances, and excretes essential hormones.

Chronic kidney damage leading to kidney failure has numerous causes; however, the most common causes are uncontrolled hypertension, poorly controlled diabetes, and glomerulonephritis.[137] Uncontrolled hypertension leads to nephrosclerosis, or localized damage to the glomeruli. There are two proposed mechanisms of hypertensive nephrosclerosis, the first glomerular ischemia and the second hypertension-induced damage and resultant hyperfiltration. Diabetic glomerulopathy has excessive extracellular matrix as the most important pathologic feature. Glomerulonephritis can be primary or secondary, each of which has many causes, a full discussion of which is beyond the scope of this chapter. Infections, immune diseases, and vasculitides can all cause primary glomerulonephritis with resulting scarring of the nephrons. Secondary causes include diabetes, hypertension, and lupus, among others.

Alternatively, chronic kidney disease can be classified based on the anatomic part of the nephron that is affected. Glomerular, interstitial, tubulointerstitial, vascular, and obstructive are all anatomic classification examples that are listed in Table 27.12. Fluid overload with hypertension can occur when the glomerular filtration rate falls below 60 mL/min.

#### *Hyperkalemia*

The ability to maintain normal potassium levels is preserved until the glomerular filtration rate reaches approximately 10% of normal. Although patients with chronic kidney failure can secrete potassium until an advanced stage of renal failure, the rate of excretion is reduced, causing prolonged elevations in potassium following ingestion. Caution must be used when a patient is noted to be receiving spironolactone. Spironolactone is an aldosterone agonist that can result in dangerous hyperkalemia if not monitored closely.

#### *Hyponatremia*

Sodium metabolism is maintained until renal failure becomes advanced. As renal failure progresses, the ability to conserve sodium is compromised, resulting in hyponatremia.

#### *Uremia*

Uremia literally means "urine in the blood." It is a general term that began to be used before the understanding that end-stage kidney disease was more complex than just the inability to filter urea from the blood. As nephrons die, the remaining units increase their capacity in a process known as compensatory hyperfiltration.[136] Increased glomerular permeability is another adaptation to the reduced number of functioning nephrons.

#### *Metabolic Issues*

Metabolic acidosis is caused by the reduced ability of the failing kidney to excrete acid.[119] This is exacerbated by decreased ability to resorb bicarbonate. Bicarbonate serves as the main pH buffer in the body and is derived from bone stores.

#### *Bone Issues*

**Hypocalcemia.** Hypocalcemia is loss of calcitriol and leads to decreased calcium absorption and stimulation of parathyroid hormone release.

**Hyperphosphatemia.** Hyperphosphatemia is caused by impaired excretion. In the face of abnormal bone metabolism, soft tissues became the phosphate reservoir, causing increased vascular

**TABLE 27.12 Anatomic Classification of Renal Disease**

| Type | Example |
|---|---|
| Glomerular | Postinfectious<br>Viral<br>Rapidly progressive<br>Immune mediated<br>Membranous |
| Interstitial | Autoimmune (systemic lupus erythematosus, Sjögren syndrome, Wegener granulomatosis, Kawasaki syndrome)<br>Medication induced |
| Tubulointerstitial | Numerous conditions, including but not exclusively:<br>Autoimmune<br>Infection<br>Drugs<br>Vascular<br>Obstruction<br>Heavy metals<br>Granulomatous diseases |
| Vascular | Renal artery thrombosis<br>Thromboembolism<br>Aneurysm |
| Obstructive | Neurogenic bladder<br>Blood clot<br>Tumor |

calcification. This leads to increasing pulse pressure in advanced disease.

### *Weakness*

Anemia of chronic disease resulting from decreased erythropoietin and iron deficiency contributes to fatigue and weakness. Supplementation with erythropoietin is recommended and overseen by the primary nephrologist.

### *Debility*

Studies have shown that a glomerular filtration rate of less than 60 mL/min is associated with reduced well-being and overall function.[106,127] Patients who are dependent on dialysis have multiple possible sources of debility depending on the type of dialysis they receive. Peritoneal dialysis using the peritoneum as a membranous filter typically takes place in the patient's home, causing less functional impact than hemodialysis, a facility-based intervention. Both methods of dialysis have potential for infection resulting from indwelling catheters, but peritoneal dialysis is more likely to be complicated by subacute bacterial peritonitis as opposed to bacteremia in hemodialysis. SBP is more likely to result in hospitalization, whereas hemodynamically stable bacteremia will be treated on an outpatient basis with antibiotics and with less likelihood of iatrogenic complications during hospitalization. Patients who are hemodialysis dependent often report substantial fatigue following dialysis sessions; this impacts on their ability to participate not only with ADLs but also with rehabilitation efforts.

### *Issues of Dialysis: "Residual Syndrome"*

The term "residual syndrome" has been applied to the syndrome of partially treated uremia, because dialysis cannot fully replace all renal functions. Electrolyte imbalances and the resultant acid-base abnormalities are responsible in part for the uremic symptoms that are seen in patients who are dialysis dependent.

### *Sarcopenia/Uremia*

Uremic sarcopenia has been described in chronic kidney disease.[79,92] The presence of uremia causes changes in skeletal muscle fibers including mitochondrial depletion and atrophy of both slow- and fast-twitch muscle fibers.[39,101,149] This in turn contributes to the overall sense of fatigue and weakness reported by these patients. The role of exercise in maintaining health-related quality of life and exercise capacity has been described, and patients should be enrolled in an exercise program whenever possible to preserve skeletal muscle function as soon as possible following the diagnosis of renal insufficiency.[30,159]

## Rehabilitation for Patients Before Transplantation

Patients with chronic renal insufficiency are often candidates for rehabilitation in both the hospital as well as outpatient settings. Chronic deconditioning resulting from uremic sarcopenia, a predisposition to chronic pain and gait abnormalities, represents areas for substantial rehabilitative intervention.[32,98,115] Patients on dialysis should be instructed in a regular stretching program because of the potential for hip and knee contractures from prolonged sitting. Patients on hemodialysis are at risk for a substantial degree of sedentary behavior as a result of extended immobility during hemodialysis sessions and postdialysis fatigue following dialysis sessions. Three hours of dialysis followed by 2 hours of resting while watching television constitutes up to 15 hours of additional sedentary time a week. Early referral to a rehabilitation professional for institution of a home exercise program before dialysis is warranted for all patients with uremia. It has been shown to be beneficial for patients on hemodialysis to exercise during dialysis sessions, although this is rarely standard practice.

## Early Mobilization After Renal Transplantation

Similar to cardiac transplantation, patients following renal transplantation are substantially disabled as a result of a long-standing chronic medical condition: uremia instead of congestive heart failure. Although a patient on hemodialysis is typically more functional than a patient with end-stage heart failure, the principles of preprocedure rehabilitation or "prehab," posttransplant evaluation, and coordination of rehabilitative care are the same.

Following transplantation, reduced mobility, prescribed corticosteroids, and a sedentary lifestyle during recovery can greatly accelerate debility even in the face of a normally functioning kidney. Unlike cardiac transplantation, there is no expected increase in CO to boost energy metabolism. It is essential that all patients be evaluated following renal transplantation for potential rehabilitation intervention whether it be for aggressive mobilization and extended monitoring or for discharge to acute or subacute facilities.

Patients following renal transplantation have complex rehabilitative needs similar to patients who are critically ill in the medical intensive care setting. Both populations have multisystem organ damage, distributive shock, hemodynamic instability, and severe deconditioning and are at risk for functional decline. Unlike the medical ICU where a comprehensive early mobilization program may be implemented previously, the patient following renal transplantation may be more reliant on physiatric consultation and coordination of care to receive required services. Applying the early mobilization model to this population would necessitate evaluation for participation with physical medicine and rehabilitation on postoperative day 1. Patients requiring ventilator support can receive physical and occupational therapy if sedation is interrupted long enough to participate with the treating therapist. Physical therapy is often automatically ordered for all appropriate patients postoperatively and coordination with the primary team should take place in the morning with an agreed upon time for treatment should an interruption in sedation be needed. Daily physiatric consultation and review of treatment session notes can guide expectations for the following treatment session. It is crucial to ensure uninterrupted therapy at least three times a week because posttransplant physical reserve is reduced and potential for accelerated deconditioning in the presence of an immunosuppressive regimen that includes corticosteroids is substantial.

Outpatient rehabilitation programs following renal transplantation should focus on controlled conditioning and education regarding the importance of exercise in maintaining exercise capacity and lifelong function. The same target heart ranges can be used for patients following both cardiac and renal transplantation: 60% to 70% of peak effort for 30 to 60 minutes three to five times weekly. Rate of perceived exertion as described in the Borg Scale is a validated method to regulate intensity. It is important to provide consistent encouragement because patients following transplantation can be extremely debilitated and depressed. Advising transplant survivors that functional improvement may not be seen for 2 weeks or more following initiation of conditioning will help to maintain participation. A comprehensive rehabilitative program will offer numerous benefits following renal transplant. Physical therapy can be focused on achieving a baseline improvement to facilitate participation in activities that the patient following transplantation may have forgone in the face of declining function. Perhaps more than any other solid organ transplant, successful candidates for renal transplantation can hope to achieve maximum function with optimal rehabilitation.

## Pulmonary Rehabilitation in Coronavirus Disease 2019

The coronavirus disease 2019 (COVID-19) pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been the most significant public health crisis in 100 years, and it has affected all aspects of healthcare, including every area of rehabilitation medicine. At the time of publication, COVID-19 is still increasing worldwide, and with the high number of survivors who have suffered a severe acute respiratory distress syndrome (ARDS) with prolonged intubations or severe pneumonia, the need for rehabilitation interventions has never been higher.[26a] Even though recent evidence has suggested that most patients hospitalized with COVID pneumonia can be successfully treated without mechanical ventilation, there are severe pulmonary implications for many patients. All hospitalized patients should be discharged with clear recommended guidelines for safe and progressive conditioning at home as standard of care is now to provide medical oversight and guidance for patients at home whenever possible.[1a,126a] This has led to a rapid expansion of telehealth services across the medical continuum that includes all disciplines within physical medicine and rehabilitation even as the medical complexity of patients admitted to acute rehabilitation has increased.[46a]

The common admitting diagnoses to acute rehabilitation units during the pandemic has been deconditioning following acute hypoxic respiratory failure secondary to COVID pneumonia. A key principle guiding care of COVID-19 patients is the maintenance of adequate oxygenation at all times. For hospitalized patients the principles of early mobilization should be utilized whenever possible to prevent rapid deconditioning and further need for already limited rehabilitation services.[122a] Oxygen saturation should be maintained above 90% with liberal use of supplemental oxygen. Continuous oximetry monitoring may be indicated for patients whose oxygenation level declines with trial mobilization on room air. COVID-19 patients have also been found to be at higher risk for venous thromboembolic events (VTEs), and baseline laboratory studies for all patients admitted to a rehabilitation unit should include a panel screening for prothrombotic states that may indicate need for prophylaxis. Patients are at elevated risk for VTEs for an extended period following intensive care hospitalization for COVID-19 pneumonia, so a high index of suspicion should be maintained throughout the course of rehabilitation.[77a] As the incidence of COVID-19 decreases and acute rehabilitation units reopen, there will be ongoing opportunities to generate more objective data and high-quality literature on characteristics, complications, and outcomes of the COVID-19 population in the rehabilitation setting.

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