benefits were patients with heart disease who had not returned to their jobs. Furthermore, only 10% had attempted either to be retrained or to seek a less strenuous position within their company.

In 1940, the New York State Employment Service sought assistance from the New York Heart Association in evaluating return-to-work status for workers with cardiac disease. The purpose of this evaluation was to determine a level of activity that would be safe and would allow recovered individuals to return to work and once again become productive members of society. This request eventually led to the establishment of the Work Classification Units or Work Evaluation Units.[5] These Work Evaluation Units were located in teaching hospitals, rehabilitation centers, and community hospitals all across the state.

The purpose of these units was threefold: (1) to provide a clinical service by using a team evaluation approach of the client's work capacity regardless of the type or severity of cardiac dysfunction and by offering an opportunity for appropriate job placement, (2) to serve as an educational instrument for training physicians and for informing the general public, and (3) to serve as a research opportunity for studying the effects of coronary artery disease on return to work. The cardiac Work Evaluation Units of the 1940s became the earliest approach to formalized cardiac rehabilitation programs.

As a result of the implementation of the Work Evaluation Units, many individuals were able to return to the labor force and once again become productive members of society. This reduced both the number of men receiving disability funds and the financial burden to the state. However, in spite of these positive results, by the 1950s, dissatisfaction over declining referrals to the units and the methods used to classify coronary artery disease disability caused many units to close. Additionally, the lack of any formal exercise intervention or follow-up evaluation led to client disinterest. Gradually, the effectiveness of the units dwindled and the programs closed.

DELETERIOUS EFFECTS OF PHYSICAL INACTIVITY

In 1952, Levine and Lown[6] openly questioned the need for enforced bed rest and prolonged inactivity after an uncomplicated MI. On the basis of earlier research,[6] they prescribed early sitting up at bedside and armchair exercises for patients recovering from MI. Their work concluded that long, continued bed rest ". . . decreases functional capacity, saps morale, and provokes complications."[6] Their highly acclaimed published report caught the attention of the medical community and elevated the level of investigation about the management of cardiovascular disease. Today, this article is recognized as a landmark article, demonstrating that early mobilization of patients with acute heart disease significantly reduces complications and mortality.[6]

At the 13th scientific session of the American Heart Association in 1953, the noted physician Louis Katz told the medical community, "Physicians must be ready to discard old dogma when they are proven false and accept new knowledge."[7] He recommended that new research findings on physical activity should be incorporated into the management of patients with cardiac disease. In 1958, two cardiologists, Turell and Hellerstein, urged physicians to provide a more positive and comprehensive approach for the treatment of coronary artery disease.[8] They recommended a graded step program (a prototype to contemporary cardiac rehabilitation) based on established energy requirements of physical activity and patient exercise tolerance while monitoring cardiovascular function, both founded on principles of work physiology. This set the stage for renewed interest concerning the effect of physical activity on patients with coronary artery disease.

This new approach, which incorporated exercise into the medical management of patients with coronary artery disease, was provided high visibility when President Dwight Eisenhower suffered a heart attack in the late 1950s, while in office. His physician Paul Dudley White, a man strongly committed to the positive effects of exercise, prescribed for his eminent patient a program of graded levels of activities, including swimming, walking, and golf. The results were so positive for the President that he created the President's Youth Fitness Council. In the 1960s, President John F. Kennedy renamed the council as the President's Fitness Council in order to encourage physical activity in individuals of all ages and foster an appreciation of its positive effects throughout the life span.

ADVANCEMENTS IN ACUTE CARDIAC CARE

The 1960s was a period of rapid advancement in the care of patients with coronary artery disease. The general public became better educated on the early warning signs of an impending heart attack. It was becoming clear that survival from MI was dependent on rapid transport to a hospital and immediate intervention to reduce the risk of sudden death and/or minimize the damage caused by the infarction. In 1966, Congress passed the Highway Safety Act. This landmark piece of legislation directed states to develop emergency medical service systems, whose mission was to provide emergency treatment in the field and rapid transportation to the hospital. The 1960s and 1970s saw improvement in prehospital emergency care, with emergency medical technician–paramedic personnel providing treatment in the field and in ambulances that were evolving into sophisticated mobile emergency units. The public was receiving instruction and certification in basic life support (BLS), whereas physicians, nurses, and allied health personnel were being trained in advanced cardiac life support (ACLS). Cardiac intensive care units (CICUs) were multiplying and flourishing: These units specialized in the acute care of patients in the early stage of evolving MI. In addition, the experience in the CICUs made the diagnosis of sudden death, which was most likely to occur at the inception

of a myocardial ischemic episode, perhaps reversible and/or preventable. The use of sophisticated diagnostic and monitoring equipment, like radionuclide imaging, Holter monitoring, and invasive hemodynamic pressure monitoring, was becoming the new standard of care in the management of patients in the acute phase of MI. Most recently, current outcomes research has confirmed that the likelihood of survival from MI increases when the earlier emergency treatment is instituted. "Every minute counts" and "time is muscle" are today's battle cries in the fight against heart disease.[9]

CARDIAC REHABILITATION

By the mid-1960s, numerous research studies had demonstrated the adverse effects of physical inactivity after an uncomplicated MI.[10-15] Saltin et al. reported that the functional capacity of normal subjects confined to bed for 3 weeks decreased approximately 33%. Equally important was the finding that, with physical training, subjects were able to achieve their pre–bed rest aerobic condition. After 3 months of twice-daily rigorous exercise programs, Saltin found that all subjects exceeded their control state.[13]

Cardiac Rehabilitation Programs as Formalized Interventions

As a result of the work of Wenger, Zohman, Hellerstein, and others, the concept of progressive supervised exercise for medically stable patients soon expanded to include more complicated patients with MI as well as patients following coronary artery bypass graft (CABG).[16-25] By the end of the 1970s, cardiac rehabilitation programs were stratified into four phases: phase 1—the hospital inpatient period; phase 2—the convalescent stage following hospital discharge; phase 3—the extended, supervised endurance training program; and phase 4—the ongoing maintenance period. Each phase had its own objectives for patient care and progression.[16-25]

Phases of Cardiac Rehabilitation From the Late 1960s to 1990s

Phase 1 Cardiac Rehabilitation

Many inpatient early mobilization hospital programs were originally 14 steps in length, which started in the CICUs and continued through the step-down phase (approximately 24 days). Activities appropriate to phase 1 were generally low-level, rhythmic, isotonic exercises that were calisthenic in nature. Early mobilization programs were designed for uncomplicated patients with acute MI in order to progressively increase activity levels in three areas—active exercises, activities of daily living (ADL), and educational activities (Fig. 1-1).[17,18] A patient was eligible for phase 1 cardiac rehabilitation when his or her clinical condition stabilized. This structured plan greatly assisted the patient toward discharge and an early return to everyday activities. The favorable outcome of these formalized programs led to the development of similar programs across the country. Soon many hospitals were observing the positive economic implications of early mobilization. These included a hastened recovery time, which decreased hospital stay and improved functional status at discharge; a decrease in depression; and an early return to work.[22,23] As CABG surgery became a routine intervention, many of these surgical patients were also included in the phase 1 programs. Eventually, the strong positive effects of these programs seemed appropriate for more complicated patients with coronary artery disease. See Box 1-1 for an example of an early mobilization phase 1 protocol dating from the late 1960s.

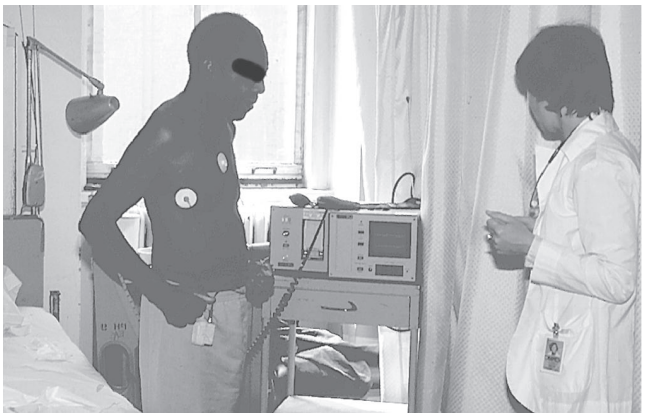

FIGURE 1-1 Physical therapist in a large metropolitan hospital helping a post–myocardial infarction patient to perform low-level exercises as part of a phase 1 cardiac rehabilitation program. Note the use of the portable bedside telemeter. Photograph taken in the late 1970s.

Phase 2 Cardiac Rehabilitation

Phase 2, the convalescent phase, followed hospital discharge and was originally referred to as the "home phase."[24] These early programs lasted 6 to 8 weeks, depending on the patient status. Physicians were acting on the notion that myocardial scar formation takes between 6 and 8 weeks. Thus, phase 2 allowed the heart muscle the time to heal. Patients were not allowed to return to work. They were discharged from the hospital and instructed to continue the exercises performed in the hospital and commence a walking or biking program. This transitional phase was often difficult for patients and families because they were each independently adjusting to the new diagnosis and were often uncomfortable with the implementation of progressive activities.[24]

In the early 1980s, many phase 2 programs were extended for up to 12 weeks. Family and physician consultation was done on a regular basis. Additionally, risk-factor modification and psychological and vocational outcomes were established.[25-28] In the early 1990s, phase 2 programs actually decreased in length as a result of reimbursement, severity of disease, and patient need.[26]

BOX 1-1

Summary of the 14-Step Wenger Program

Step 1
Exercise: Passive range of motion exercises to the upper and lower extremities.
Activities of daily living: Begin feeding self with trunk and arm support using pillows.
Activities: Initial interview—explain program.

Step 2
Exercise: Same as step 1.
Activities of daily living: Patient may wash hands and face and brush teeth in bed.
Activities: Finger and wrist craft activity in bed (eg, lacing a coin purse).

Step 3
Exercise: Passive and active range of motion exercises in bed.
Activities of daily living: Same as step 2.
Activities: Complete craft activity.

Step 4
Exercise: Active range of motion exercises progressing to minimal resistive exercises in bed.
Activities of daily living: Same as step 2. Add dressing self. Use bedside commode.
Activities: Begin copper tooling project.

Step 5
Exercise: Minimal to moderate resistive exercises.
Activities of daily living: Chair sitting with self-feeding 3× day.
Activities: Continue copper tooling project. Use higher-energy–level tools.

Step 6
Exercise: Begin use of 1- to 5-lb weights for upper and lower extremity exercises.
Activities of daily living: Self-care activities. Walk to bathroom. Bathe self in tub.
Activities: Refinish a precut wood project.

Step 7
Exercise: Add walking 50 ft down the hall.
Activities of daily living: Same as step 6.
Activities: Continue wood project.

Step 8
Exercise: Add trunk exercise. Add walking down one flight of stairs.
Activities of daily living: Self-care activities plus walking down stairs.
Activities: Continue wood project.

Step 9
Exercise: Add walking 50 ft × 2.
Activities of daily living: Self-care activities plus walking down stairs.
Activities: Begin metal hammering project.

Step 10
Exercise: Add trunk exercises using 1-lb weight.
Activities of daily living: Self-care activities plus walking down stairs.
Activities: Continue metal hammering project.

Step 11
Exercise: Same as step 10.
Activities of daily living: Self-care activities plus walking down stairs.
Activities: Continue metal hammering project.

Step 12
Exercise: Same as step 10. Add walking down two flights of stairs.
Activities of daily living: Begin homemaking activities. Introduce conservation of energy techniques.
Activities: Cut out wood project using a table saw.

Step 13
Exercise: Same as step 10. Add trunk exercises using a 2-lb weight.
Activities of daily living: Walk down stairs.
Activities: Continue wood project.

Step 14
Exercise: Add seated toe touches and walking up one flight of stairs.
Activities of daily living: Same as step 12.
Activities: Complete all projects.

Modified from Zohman LR, Tobis JS. *Cardiac Rehabilitation*. Orlando, FL: Grune & Stratton; 1970, with permission from Elsevier.

Phase 3 Cardiac Rehabilitation

Phase 3 followed approximately 6 to 12 weeks of convalescence at home.[24] Patients were medically supervised and frequently located in hospital-based outpatient departments or private cardiac rehabilitation facilities. Entrance into phase 3 began with the performance of a maximum, symptom-limited exercise test. The results of the test were used to write an exercise prescription, which was characterized by elevating the patient's heart rate to a relatively high level and maintaining it in a "training zone" for a prescribed period of time. The goal of such programs was the induction of an aerobic endurance training effect, which would allow the patient to participate in higher levels of activities before the onset of symptoms.[24-32] Patients were closely monitored during training sessions. After induction of this training effect, patients became candidates for phase 4 cardiac rehabilitation.

Phase 4 Cardiac Rehabilitation

Phase 4 programs were frequently located in YMCAs. Jewish community centers, university settings, or physical therapy private practices where patients could exercise and have their vital signs monitored.[24] Patients in phase 4 were considered medically stable and only occasionally monitored during moderate levels of exercise, which often included recreational activities like noncompetitive basketball, kickball, and volleyball. An electrocardiographic (ECG) monitor and crash cart were brought into the gym or other exercise area. Patients were instructed to monitor their own pulse and occasionally

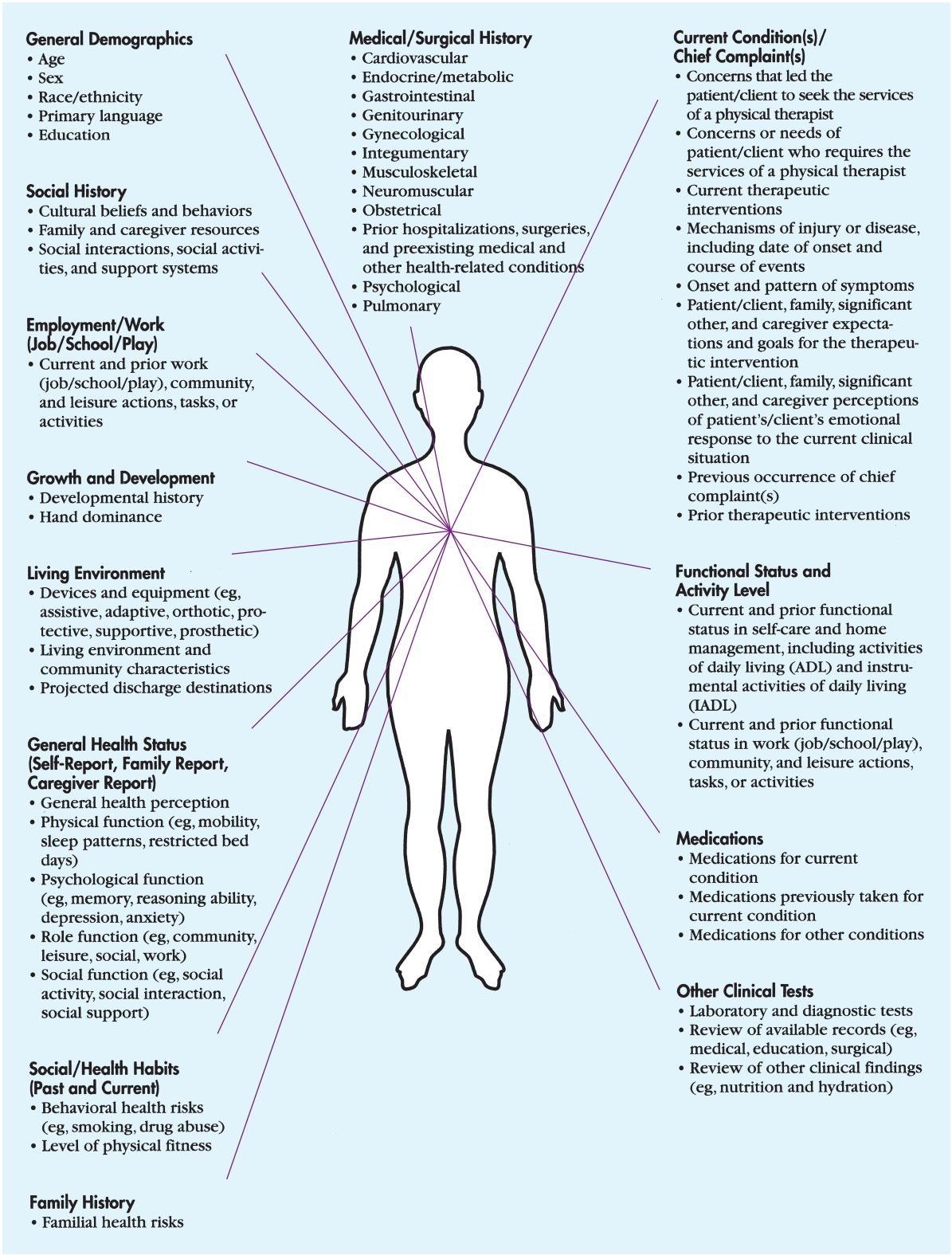

FIGURE 2-4 The five elements of patient/client management. (Reprinted from American Physical Therapy Association Guide to Physical Therapist Practice, 2nd ed. *Phys Ther*. 2001 Jan;81(1):9-746, with permission of the American Physical Therapy Association. This material is copyrighted, and any further reproduction or distribution is prohibited. All rights reserved.)

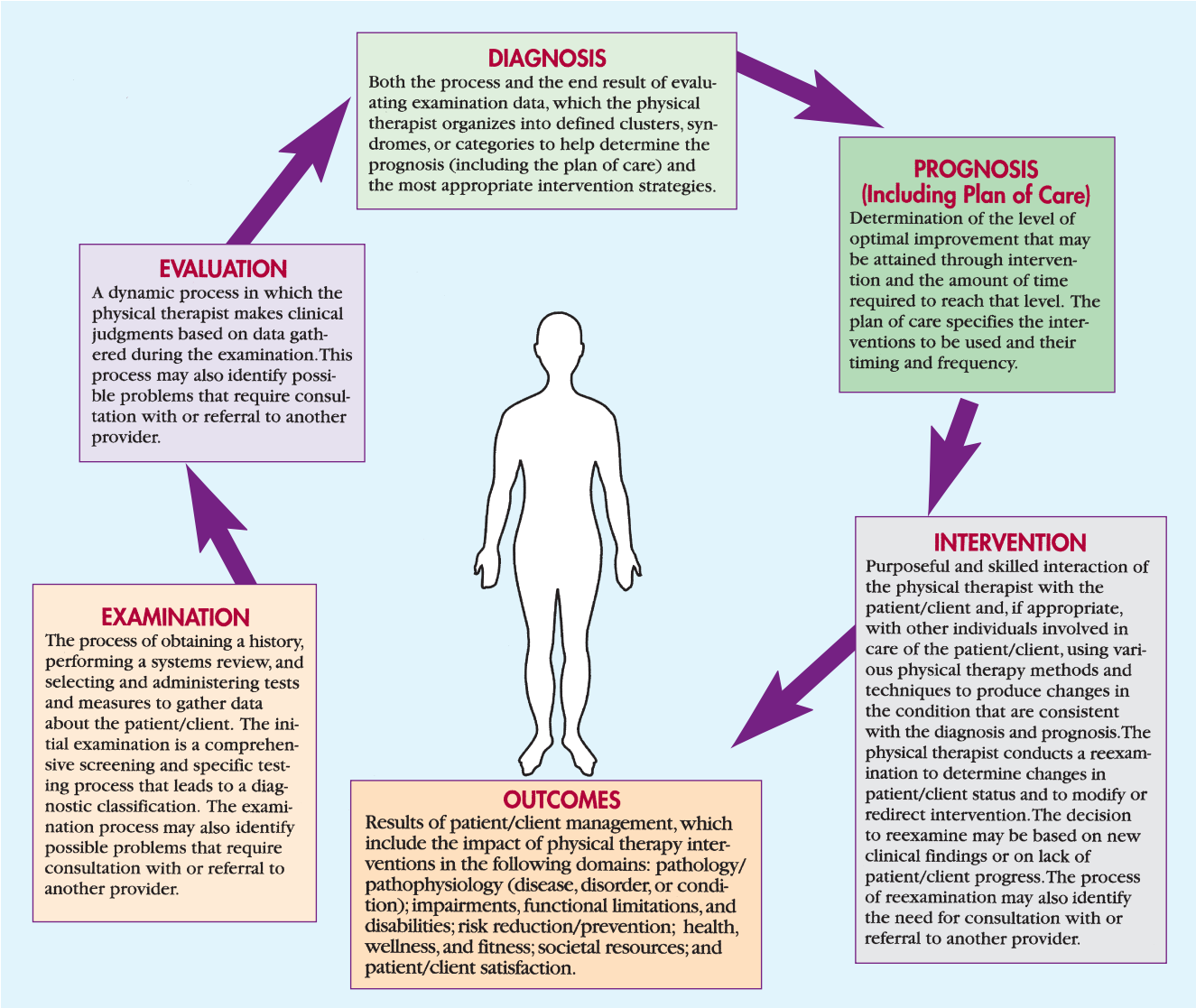

FIGURE 2-5 The types of data that may be generated from a patient/client history. (Reprinted from American Physical Therapy Association. Guide to Physical Therapist Practice, 2nd ed. *Phys Ther*. 2001 Jan;81(1):9-746, with permission of the American Physical Therapy Association. This material is copyrighted, and any further reproduction or distribution is prohibited. All rights reserved.)

PT to participate and direct PT intervention and assume a variety of roles. The types of data that may be generated from a patient/client history are shown in Fig. 2-5.[8]

Chapter 2 describes the tests and measurements that a PT may use in the management of patients or clients. Twenty-four categories of tests and measures have been presented as in the 1st edition,[2] but there is now an attempt to include clinical indications for the tests and measures within particular domains of disablement.[8] An example of the integration of the tests and measures within the Disablement Model is shown in Table 2-2.

Chapter 3 lists different interventions that may be provided by the PT. Figure 2-6 identifies the three components of physical therapy intervention (Coordination, Communication, and Documentation; Patient-/Client-Related Instruction; and Procedural Interventions) along with a listing of the nine specific procedural interventions.[8] All of the sections within Chapter 3 have been expanded including the clinical considerations, interventions, and anticipated goals and expected outcomes.

As in Chapter 2, an attempt to include clinical considerations for procedural interventions within particular domains of disablement has been made.[8] These changes are shown in Fig. 2-5. The breadth of PT is apparent in the variety of tests and measures as well as in the direct treatments that may be provided by a PT, which are listed in Chapters 2 and 3, respectively. These tests and measures as well as interventions are listed in the most appropriate domain of disablement. Examples of this are shown in Table 2-3. This is an important addition to the 2nd edition of the *Guide to Physical Therapist Practice*.[8]

skeletal muscle is matched by an adequate supply, exercise can be continued for a protracted period of time. **Activation of the long-term, or aerobic endurance, energy system tends to cause a "volume" workload on the heart, which places less stress on the heart than the higher "pressure" workload that is associated with the high-intensity, short-term energy system.**

CLINICAL CORRELATE

The heart rate response to activation of this system is fairly linear and proportionate to the workload. The rate at which systemic oxygen is consumed is predictable and proportionate to the workload as well. This fact allows the clinician to quantify the amount of exercise that a patient receives.

In the way that these three energy states have been presented, it may appear that the immediate, short-term, and long-term energy systems are three discrete entities that "turn on and off" without overlap. This is not the case. Movement across exercise states and utilization of appropriate fuel substrates occur as a smooth integration of systems. All three metabolic pathways are in continuous operation; the intensity and duration of exercise determine which system predominates[67] (Fig. 3-8).

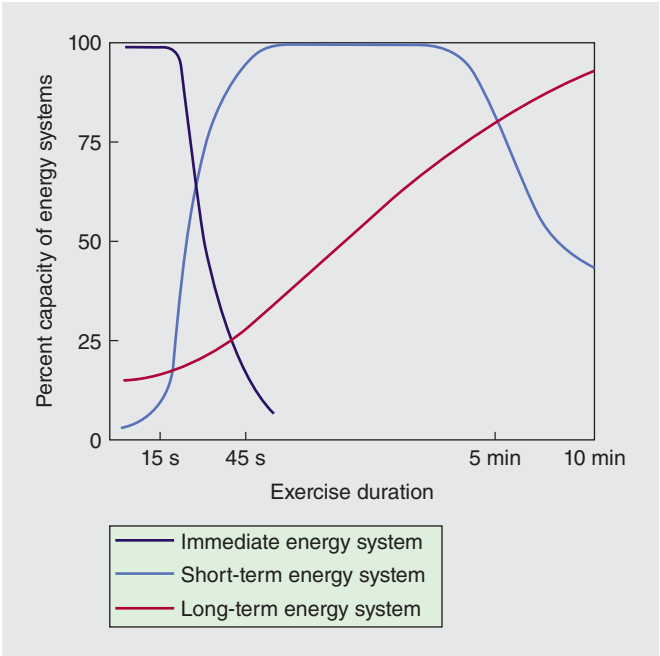

FIGURE 3-8 Utilization of three metabolic energy pathways in moving from the resting state to a moderate, prolonged level of exercise. (Modified with permission from McArdle WD, Katch FI, Katch VL. *Exercise Physiology: Energy, Nutrition, and Human Performance.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001.)

CARDIOVASCULAR AND PULMONARY RESPONSE TO EXERCISE

Immediate Response to Exercise: Cardiac

Exercise presents an ultimate challenge to the cardiovascular and pulmonary systems. The metabolic need for more oxygen and nutrients in working skeletal muscle during exercise initiates a long and complex series of feedforward and feedback mechanisms that occur through a dynamic interplay among somatosensory, musculoskeletal, cardiovascular and pulmonary systems. It is the amalgamation of these processes that ultimately propels the human body from point A to point B.

If metabolic need initiates the process, then cardiac output drives the process. Arterial blood carries oxygen and nutrients into metabolically active tissue, and venous blood removes metabolites and oxygen-reduced red blood cells from the area. This section will demonstrate by example what happens to these and other variables when a subject moves from rest to exercise.

Heart Rate: Revision of the Age-Related Maximum Heart Rate

During graded exercise, HR rises linearly with increasing workload. The increase in HR occurs as a result of a withdrawal in parasympathetic tone and by augmentation of sympathetic neural input to the sinoatrial node. HR linearity will continue until a maximum HR (HR_{max}) is achieved, at which point exercise must stop, as cardiac output is no longer able to match metabolic need. HR_{max} decreases with age and traditionally has been calculated as 220 – age. The notion of an age-related maximum HR (ARMHR) has been universally accepted and widely used for many years. It has served as an endpoint for maximal exercise testing and has also been used as a basis for prescribing intensity of effort in rehabilitation programs. The ARMHR is constant across both gender and state of training. However, the validity of the ARMHR equation has never been established, particularly for older adults.

A recent study has sought to validate the ARMHR among healthy men and women ranging widely in age.[68] Tanaka et al. performed a meta-analysis on a total of 351 peer-reviewed research papers that met the following criteria: (1) subjects were both men and women and analyzed separately; (2) subjects were nonmedicated nonsmokers; (3) subjects were adults; and (4) maximum exercise was determined by using objective criteria. Tanaka et al. went on to perform their own research study by exposing 514 healthy men and women to maximum exercise testing. Forward stepwise multiple regression analyses demonstrated that age alone accounted for 80% of the individual variance in HR_{max}. The regression formula obtained from the research experiment was virtually identical to that of the meta-analysis. There was no significant difference in the regression equation between men and women or between sedentary and endurance-trained individuals. It was concluded that the traditional 220 – age formula overestimates true HR_{max} in young adults and underestimates true

HR_{max} in persons older than 40 years. **The following formula more accurately identifies true HR_{max} among healthy adults across the life span:**

$$HR_{max} = 208 - 0.7 \times age.$$

Utilization of the new, revised formula has clinical implications. The revised formula allows older individuals to exercise to a higher HR before termination, resulting in better diagnostic validity as well as a higher level of training exercise intensity. However, it should be stressed that this formula, like 220 – age, provides only an estimate of HR_{max}. Significant variance exists at any given age. Indeed, 1 SD HR_{max} is 10 to 12 beats per minute (bpm). Finally, these results are only applicable to healthy normal adults. Individuals with overt cardiovascular disease may have a very different regression formula.

CLINICAL CORRELATE

It is thus inappropriate to apply the revised regression formula for HR_{max} to patients with cardiovascular disease for the purpose of writing an exercise prescription. These patients require a maximum symptom-limited exercise test in order to identify their specific HR_{max} and individuate their exercise prescription.

The Rate-Pressure Product

The rate–pressure product (RPP) is found by multiplying the HR and the SBP. It is usually expressed by a power of 3. Thus, for example,

$$\text{Heart rate} = 105 \text{ bpm},$$
$$\text{Systolic blood pressure} = 150 \text{ mmHg},$$
$$\text{RPP} = 15.7 \times 10^3.$$

There is a strong linear correlation between the RPP and myocardial oxygen consumption ($M\dot{V}O_2$) during progressive, aerobic lower-extremity exercise.[69,70] The RPP has particular utility for physical therapists who treat patients with heart disease in that both the HR and SBP response to exercise are often abnormal in these patients. HR and contractility, both major determinants of $M\dot{V}O_2$, may be compromised by way of ischemia or necrosis; alterations in afterload as a function of left ventricular mechanical dysfunction can also affect $M\dot{V}O_2$. Both of these findings are captured in the measurement of the RPP. The benefit of its use in monitoring tolerance to exercise and individuating an aerobic exercise prescription is that the dynamic interplay between both HR and BP is reflected in the equation.

The cardiac response to exercise is highly individual; that is, the HR and SBP responses to exercise vary widely across individuals, especially in persons with heart disease. However, recent work by Hui and colleagues presents normative values for resting and exercise RPP among healthy normal subjects.[71] Data obtained from 1,623 subjects were used to develop a multiple regression model that recognized several factors, including age, gender, and BMI, which contribute to calculation of RPP.

The reader will recall that quantifying the amount of exercise that the physical therapist prescribes to a patient through the use of METs provides a basis for comparisons between subjects. This is not the case with the RPP, which is highly variable across patients with heart disease.

CLINICAL CORRELATE

Use of the RPP to monitor exercise benefits to the patient with heart disease because it reflects cardiac function. While comparison across patients is not possible, it can be used to monitor individual patient progress. Successful acquisition of an aerobic endurance training effect is demonstrated by a reduction of the RPP at any given submaximal workload.

Cardiac Function Curve

A *cardiac function curve* is a graphical depiction of the heart's ability to receive blood from the venous system and to pump blood out through the arterial system. Examine the cardiac function curve of a healthy normal 24-year-old individual about to begin ambulation on a treadmill[72] (Fig. 3-9). This figure isolates left heart function (curved, moving from left to right) from right heart function (more linear, moving from right to left). Cardiac output represents a balance between blood coming into the heart from the periphery and blood leaving the heart from the left ventricle. The balance occurs where the two solid lines cross (point A), which indicates a cardiac output of 5.0 L/min at rest. Notice that right atrial pressure (Pra) is zero at point A: This healthy heart is pumping out the same volume that is coming in.

One of the first things that happen during the initiation of exercise is *activation of the skeletal muscle pump.* This causes an increase in venous return, a transient small increase in right atrial pressure, and thus a new right heart function curve (red line). Left heart function remains unchanged. There is new equilibration at point B, with a new cardiac output of 8.0 L/min. Within the next 15 to 20 seconds, the neurological system becomes activated. *The sympathetic nervous system turns on,* producing an increased force of contraction of both the left and the right heart and a new set of function curves that equilibrate at point C (12.5 L/min). A final development in this model is the onset of a reduction in resistance to blood flow, which occurs at the local skeletal muscle level, as a result of metabolites that cause *local vasodilatation*. Final cardiac output is at 21.0 L/min, a peak exercise level that is typical for a young, healthy normal adult.

oxygen requirements may be provoked by a number of factors including exercise, mental stress, or even spontaneous fluctuations in heart rate and blood pressure. Decreased oxygen supply may result from a reduction in coronary blood flow. (The reader may recall the already high extraction of oxygen from blood flowing through myocardial tissue, with the resultant dependence on coronary blood flow to meet myocardial demand. See Chapters 3 and 5.) Decreased blood flow may be due to decreased aortic driving pressure or increased coronary vascular resistance, which may be due to coronary vasospasm, platelet aggregation, or partial thrombosis.

It is a commonly held belief that coronary artery occlusion greater than 70% produces myocardial ischemia, which in turn provokes the symptoms that bring the patient to the doctor's office. The patient at this stage of atherosclerotic progression is comfortable at rest but will complain of chest pressure during mild-to-moderate exercise, which is relieved by rest. The diagnosis of ischemic heart disease is usually made on the basis of a formal exercise stress test.

Coronary atherosclerosis and coronary arterial spasm both reduce coronary blood flow and thus reduce myocardial oxygen supply. When this happens, myocardial ischemia and irritability occur, which may produce arrhythmias, impaired myocardial contractility (systolic dysfunction), and impaired myocardial relaxation (diastolic dysfunction). This diastolic dysfunction prolongs systole and reduces ventricular filling time. Ventricular compliance decreases and the ventricular end-diastolic pressure rises, causing aortic driving pressure to be further reduced. Myocardial ischemia often manifests itself on an electrocardiogram (ECG) as ST-segment displacement (see Chapter 11).

The threshold for myocardial ischemia can be either predictable or unpredictable. Abnormal endothelial function appears to play a role in the unpredictable, fluctuating threshold for ischemia. The majority of studies suggest that endothelium-dependent vasodilator mechanisms predominate in nondiseased epicardial coronary arteries. During interventions that normally induce increases in myocardial oxygen consumption and blood flow (eg, exercise, stress, induced tachycardia), epicardial vascular dilation occurs. This dilation is at least partially endothelial dependent. However, the presence of even nonocclusive, early atherosclerosis appears to *impair* the release of endothelium-relived relaxing factor (nitrous oxide), attenuating this vasodilator mechanism, which results in prevailing, unopposed vasoconstriction. Moderate vasoconstriction in an area of minimal occlusion may be of little hemodynamic consequence; however, the same degree of vasoconstriction in an area of greater occlusion may markedly decrease blood flow and induce ischemia.[73-75]

Stable angina—The classical symptom of myocardial ischemia is *angina pectoris*. This discomfort is described as pressure, heaviness, or tightness that may be located in the middle of the chest (substernal); over the heart (precordial); or in the shoulder, arm, throat, or jaw. Angina may be precipitated by exertion, stress, emotions, and heavy meals. Stable angina usually lasts for several minutes and is usually relieved by rest and/or nitroglycerin. The patient is pain free at rest.

Anginal pain arises within the myocardium and is thought to stimulate free nerve endings in or near small coronary vessels. Impulses travel in afferent unmyelinated or small myelinated cardiac sympathetic nerves through the upper thoracic ganglia to dorsal horn cells and through the spinothalamic tract of the thalamus to the cortex.[5,76] The cerebral cortex integrates and modifies these impulses. This modulation may contribute to the variability in the perception of angina across patients. Psychosocial and cultural factors may also influence the perception of pain at the cortical level.

Unstable angina—The term *unstable angina* is usually used to denote either a change in the anginal pattern or angina at rest. Unstable angina may occur with less exertion than previously described, may last longer, or become less responsive to medication. Angiography has shown that a high proportion of patients with unstable angina have complex coronary stenoses characterized by plaque rupture, ulceration, or hemorrhage with subsequent thrombus formation. This inherently unstable situation may progress to complete occlusion and infarction, or may heal, with reendothelialization and return to a stable though possibly more severe pattern of ischemia. New-onset angina is sometimes considered unstable, but if it presents in response to exertion and responds to rest and medication, it does not carry the same poor prognosis.

Prinzmetal (variant) angina—*Prinzmetal angina*, also called atypical or variant angina, is an unusual type of cardiac pain due to myocardial ischemia that occurs almost exclusively at rest. Prinzmetal and colleagues[77] hypothesized that variant angina was the result of transient increases in vasomotor tone or *vasospasm*. Vasospasm causes a transient, abrupt, marked decrease in the diameter of the coronary artery that results in myocardial ischemia. In such cases, no preceding increases in myocardial oxygen demand occur. Vasospasm can occur in both normal and diseased coronary arteries. Often the decrease in the diameter can be reversed by nitroglycerin.[10] Variant angina is usually not associated with physical exertion or emotional stress and is associated with ST-segment elevation, rather than with depression on ECG.[78] This form of angina is often severe and characteristically occurs in the early morning, awakening patients from sleep. It tends to involve the right coronary artery and is likely to be associated with arrhythmias or conduction defects.[5] Prinzmetal angina may be associated with acute MIs and severe cardiac arrhythmias, including ventricular tachycardia and fibrillation (Fig. 6-4).[79]

Asymptomatic (silent) myocardial ischemia—Many individuals have some episodes of "silent" ischemia (ischemia without symptoms); some patients have only silent ischemia. Asymptomatic ischemic episodes may be present in patients with any of the aforementioned ischemic coronary syndromes or after an MI. Some patients never complain of chest

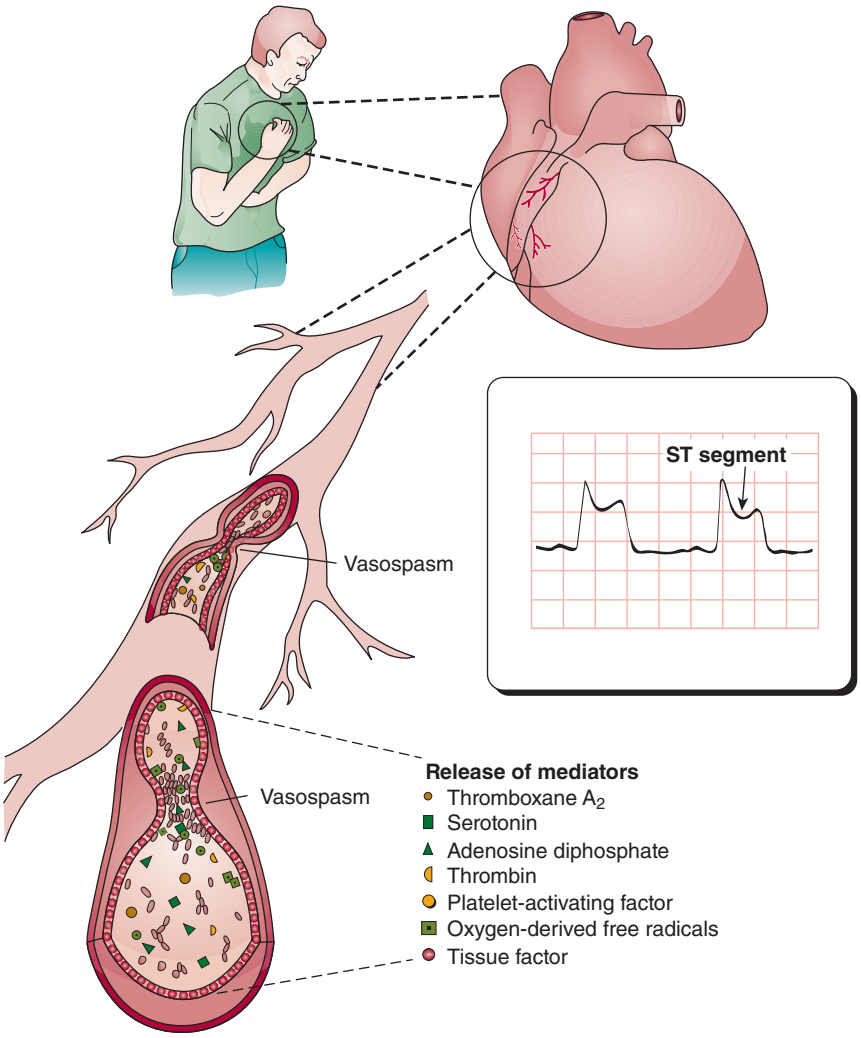

FIGURE 6-4 Clinical presentation, electrocardiographic, chemical, and arterial changes associated with coronary artery spasm. Note the ST-segment elevation above baseline.

pain with episodes of ischemia; others *inconsistently* report chest pain with episodes of ischemia. The true prevalence of silent ischemia is undetermined, but it is believed to be high. Important factors include age, the presence and extent of CAD, and other disease processes that include peripheral neuropathy as a component (eg, diabetes mellitus, alcoholic neuropathy).

Some clinicians have attempted to explain silent ischemia as angina that is less noxious than reported angina. The correlation between ECG evidence of ischemia and the report of anginal pain in patients with chronic stable angina is only fair.[80,81] Therefore, the most likely explanation is neurologic. Neuropathy with defective sensory efferent nerves occurs commonly in persons with diabetes. The variable expression of ischemic pain may be explained by modification of pain stimuli in the central nervous system. Patients with diabetes have a relatively high incidence of painless MIs and definite silent ischemic episodes as documented by ambulatory ECG recordings and exercise testing.[82-85]

***Anginal equivalents*—**These include dyspnea, fatigue, lightheadedness, and belching brought on by exercise or stress and relieved by rest or nitroglycerin. We have said that some patients with diabetes may not complain of chest discomfort due to impaired peripheral sensation (eg, silent ischemia). Alterations in neural processing can, by extension, also give rise to anginal equivalents. Ischemic episodes in this group can present as fullness in the throat and the jaw, a desire to cough, or dyspnea. Elderly patients and patients with peripheral neuropathies may also present with anginal equivalents.

The rich variety of radiation patterns associated with angina pectoris is determined by the levels of the spinal cord, which share sensory inputs with somatic structures (eg, gut) and the heart. The precise mechanisms causing angina and anginal equivalents are yet to be defined.

Myocardial Infarction

***Pathogenesis*—**MI results from prolonged myocardial ischemia and is precipitated in most cases by an occlusive

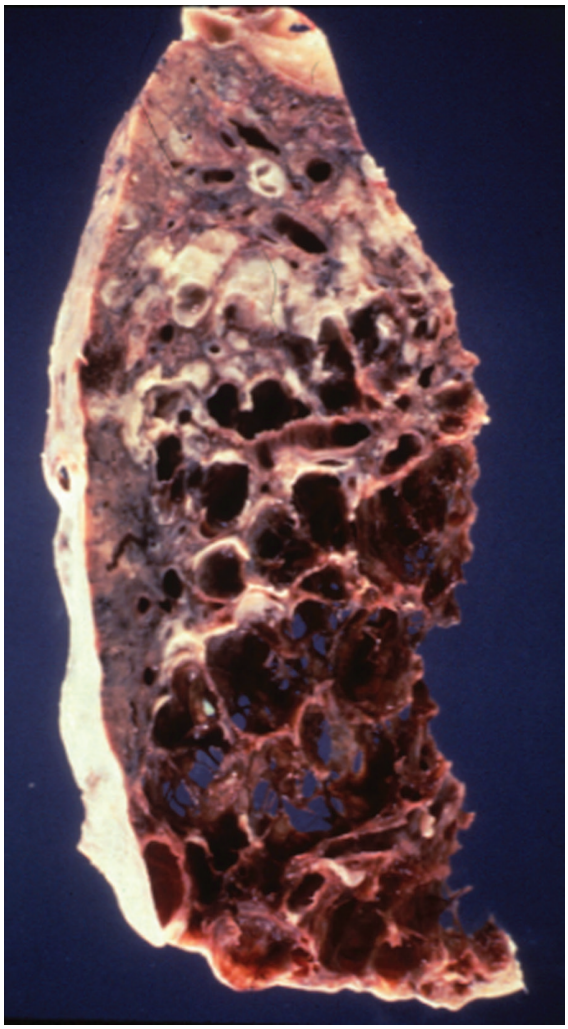

FIGURE 7-5 Emphysema. This disease results in the destruction of bronchioles and parenchymal tissue, which leads to the loss of elastic recoil properties of the lung. This results in the dilatation of airways, which leads to air trapping and hyperinflation. (Used with permission from Dana Gryzbicki, MD, University of Pittsburgh, PA.)

of the particles are removed through a well-developed lymphatic system of the lower lobes; however, the particles that are also deposited in the upper lobes are revoked at a slower removal rate due to the smaller size of its lymphatic system.[40] The loss of FEV_1 function occurs more than twice as fast as the aging process and declines by 25% for each pack year of smoking.[40,42,43] It is associated with a very insidious onset, which occurs over 30 to 40 years.

Cigarette smoking is associated with an increase in elevated inflammation and cellular apoptosis, early and excessive cell death. There is an increased accumulation of apoptotic cells and slow cell removal with macrophage dysfunction. There is also an increase in TNF-α and a decrease in surfactant protein. These changes lead to alteration of alveolar and small airway function, inflammatory and proteolytic activity, and changes in the endothelium and epithelium cells. The consequences of destruction of the alveolar wall, decrease in surface area, loss of functioning pulmonary capillary bed, and loss of the parenchyma lead to air trapping and ventilation–perfusion ($\dot{V}/\dot{Q}$) mismatch.[38,42,44,45]

The etiology of emphysema is based on the protease-antiprotease hypothesis in which there is an imbalance between protease, which causes tissue breakdown, and antiprotease enzymes. This imbalance leads to the loss of lung parenchyma and elastic recoil, which the small airways depend on return to their resting states during exhalation. The elastic property of the parenchyma also provides a normal level of airway resistance during inspiration. This loss of parenchyma tissue results in the loss of *radial traction* on the airways. The end result is dilation of airways, premature airway closure and air trapping, and an increase in RV.[40,46,47]

The nicotine in cigarette smoke attracts neutrophils, activates alveolar macrophages, and inactivates the protective nature of antiprotease.[48] The alveolar macrophages and neutrophils contain protease enzymes, which are capable of destroying the elastic property of the lung tissue, thus producing emphysema.[40] The cigarette smoke causes chronic inflammation that leads to proliferation of endothelial and smooth muscle cells, platelet aggregation, thrombus formation. The end results are the destruction of pulmonary capillaries.[41] The impairment to the small blood vessels within the pulmonary system may lead to the decrease in DLCO and the development of secondary pulmonary hypertension (PH).[38,43-45]

As a consequence of intrinsic pulmonary damage, hyperinflation of the lungs occurs, which eventually leads to the compensatory changes of the chest wall. This disruption of normal chest wall mechanics leads to dysfunction of the inspiratory muscles, particularly of the diaphragm. The dysfunction of the diaphragm is an important cause of respiratory failure in patients with emphysema. Hyperinflation causes shortening of inspiratory muscles and flattening of the diaphragm with the loss of sarcomeres. The result is a loss of diaphragmatic excursion and subsequent decline in the mechanical effectiveness of the diaphragm, and other respiratory muscles needed to support the increased demand of ventilation.[49]

The most common complaint of patients with emphysema is *dyspnea on exertion* (DOE). The common results of a physical examination reveal the following findings: diminished breath sounds and high-pitched wheezing, which are typically associated with exertion, and a prolonged expiratory phase.[38,42,50] The patient will present with an enlarged anterior-posterior dimension of the chest wall, called a *barrel chest,* with an increase in rib angle. The accessory muscles are commonly hypertrophied from overuse. There is hyperresonance sound upon mediate percussion, which is consistent with the hyperinflation of the lungs. The presence of a chronic cough and sputum production will vary and depend on the infectious history of the patient. As the disease advances, many patients become *cachectic,* or emaciated, and begin to show signs of right-sided heart failure due to secondary PH. The classic signs and symptoms of right-sided heart failure include peripheral pitted edema, weight gain, jugular vein distension, diminished appetite, right upper quadrant discomfort, and ventricular

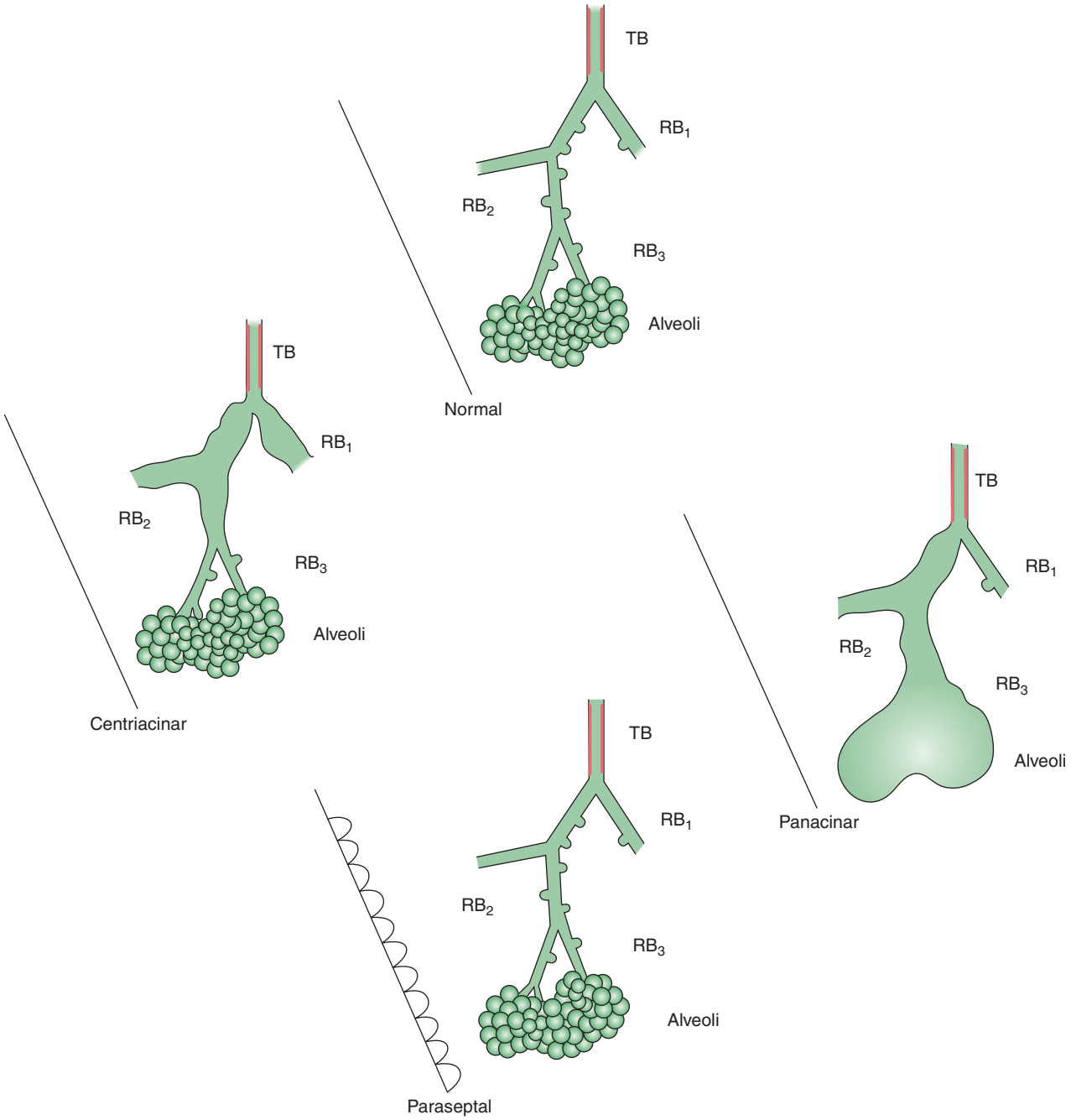

FIGURE 7-6 Types of emphysema. (Modified with permission from Gurney JW. Pathophysiology of obstructive airways disease. *Radiol Clin North Am*. 1998;36(1):15-27.)

gallop, S_3 heart sound (Fig. 7-7). Emphysema is considered as a systemic disease with the increase of the inflammatory process. Patients with emphysema commonly suffer from osteoporosis, skeletal muscle disease, depression, and an increase in incidence of cardiovascular disease.[42,46,51,52] Beyond the physical examination, PFT results are consistent with other obstructive airway diseases, which include a decline in FVC, FEV_1, and FEV_1/FVC ratio that indicates small airway disease. There is an increase in TLC and RV.[53] The chest X-ray reveals hyperinflation with a flattened diaphragm, decreased vascular markings, and possible enlargement of the right side of the heart. Because of the destruction of the gas-exchange areas of the lungs, there is also a mismatch between ventilation and perfusion $\dot{V}/\dot{Q}$ that is demonstrated on a $\dot{V}/\dot{Q}$ scan.[50]

Patients who present with the classic presentation of emphysema will have arterial blood gas analysis that typically reveals hypoxia and normal-to-slight hypocapnia. These patients present with tachypnea, labored breathing, and a normal-to-low body mass index (BMI). Some patients with emphysema will present with signs more associated with chronic bronchitis including hypoxemia, hypercapnia, signs of right-sided heart failure, copious secretions, and an