effusions, anxiety, or pulmonary embolism. A patient with left ventricular systolic or diastolic failure may describe the acute onset of breathing difficulty when sleeping. This problem, called paroxysmal nocturnal dyspnea (PND), is caused by pulmonary edema that is redistributed in a prone position; it is usually secondary to left ventricular failure. These patients often notice the acute onset of dyspnea followed by coughing roughly 2 to 4 hours after going to sleep. This can be a very uncomfortable feeling, and it leads the patient to sit up immediately or get out of bed. Symptoms typically resolve over 15 to 30 minutes. Patients with left ventricular failure also often complain of orthopnea, which is dyspnea that occurs when one assumes a prone position. This is relieved by sleeping on multiple pillows or remaining seated to sleep.

Patients with sudden onset of dyspnea may be experiencing flash pulmonary edema, which is very rapid and acute accumulation of fluid in the lungs. This can be associated with severe CAD and may also be a cause of dyspnea in patients with coarctation of the aorta and renal artery stenosis. Sudden dyspnea is associated with pulmonary embolism, and this symptom is typically accompanied by pleuritic chest pain and possibly hemoptysis in such patients. Pneumothorax can cause dyspnea accompanied by acute chest pain. Dyspnea due to lung disease is present with exertion, although in severe cases it may be present at rest. This is often accompanied by hypoxia and is relieved by pulmonary bronchodilators or steroids or both. Dyspnea may also be an "angina equivalent." Not all patients with CAD develop typical anginal chest pain. Dyspnea that comes on with exertion or emotional stress, is relieved with rest, and is relatively brief in duration might be a manifestation of significant CAD. This type of dyspnea is also usually improved with the administration of nitroglycerine.

Palpitation

Palpitation is another symptom commonly seen in the cardiovascular patient. This is the subjective sensation of rapid or forceful beating of the heart. Patients often are able to describe in detail the sensation they feel, such as jumping, skipping, racing, fluttering, or an irregularity in the heartbeat. It is important to ask the patient about the onset of the palpitations because they may begin abruptly at rest, only with exertion, with emotional stress, or with ingestion of certain foods such as chocolate. One should also inquire about associated symptoms such as chest pain, dyspnea, dizziness, and syncope. It is important to note other medical issues, such as thyroid disease, and bleeding, which can lead to anemia, because these conditions may be associated with arrhythmias. A social history focusing on drug use and intake of alcohol is important because use of these substances can lead to certain rhythm disturbances. The family history is also important, because there are many inherited disorders (e.g., long-QT syndromes) that might lead to significant arrhythmias.

Potential etiologies of palpitation include premature atrial or ventricular beats, which are typically described as isolated skips and can be uncomfortable. Supraventricular tachycardias such as atrial flutter, AV nodal reentrant tachycardia, and paroxysmal atrial tachycardia often start and stop abruptly and can be rapid. Atrial fibrillation is usually rapid and very irregular. Ventricular arrhythmias are more often associated with severe dizziness or syncope. Gradual onset of tachycardia with a gradual decline in HR is more indicative of sinus tachycardia or anxiety.

Syncope

Syncope may be caused by a variety of cardiovascular diseases. It is the transient loss of consciousness due to inadequate cerebral blood flow. In the patient presenting with syncope as a primary complaint, one must try to differentiate true cardiac causes from neurologic issues such as seizure and metabolic causes such as hypoglycemia. Determination of the timing of the syncopal event and associated symptoms is very helpful in determining the etiology. True cardiac syncope is typically very sudden, with no prodromal symptoms. It is typically caused by an abrupt drop in cardiac output which may be due to tachyarrhythmias such as ventricular tachycardia or fibrillation, bradyarrhythmias such as complete heart block, severe valvular heart disease such as aortic or mitral stenosis, or obstruction of flow due to left ventricular outflow tract (LVOT) obstruction. True cardiac syncope often has no accompanying aura. In situations such as aortic stenosis or LVOT obstruction, syncope typically occurs with exertion. Patients usually regain consciousness rather quickly with true cardiac syncope.

Neurocardiogenic syncope involves an abnormal reflexive response to a change in position. When one rises from a prone or seated position to a standing position, the peripheral vasculature usually constricts and the HR increases to maintain cerebral perfusion. With neurocardiogenic syncope, the peripheral vasculature abnormally dilates or the HR slows or both. This leads to a reduction in cerebral perfusion and syncope. A similar mechanism is responsible for carotid sinus syncope and syncope associated with micturition and cough. The patient usually describes a gradual onset of symptoms such as flushing, dizziness, diaphoresis, and nausea before losing consciousness, which lasts seconds. When these patients wake, they are often pale and have a lower HR. In the patient with syncope due to seizures, a prodromal aura is typically present before loss of consciousness occurs. Patients regain consciousness much more slowly and at times are incontinent, complain of headache and fatigue, and have a postictal confusional state. Syncope due to stroke is rare, because there must be significant bilateral carotid disease or disease of the vertebrobasilar system causing brainstem ischemia. Neurologic deficits accompany the physical examination findings in these patients.

The history is very important in determining the cause of a syncopal episode. This was previously studied by Calkins and colleagues, who found that men older than 54 years of age who had no prodromal symptoms were more likely to have an arrhythmic cause of their episodes. However, those with prodromal symptoms such as nausea, diaphoresis, dizziness, and visual disturbances before passing out were more likely to have neurocardiogenic syncope. Many inherited disorders such as long-QT syndrome and other arrhythmias, hypertrophic cardiomyopathy with LVOT obstruction, and familial dilated cardiomyopathy lead to states conducive to syncope. For this reason, a very detailed family history is necessary.

Edema

Edema often accompanies cardiovascular disease but may be a manifestation of liver disease (cirrhosis), renal disease (nephrotic