dysfunction is best defined as significant bradycardia associated with symptoms plausibly attributable to bradycardia.

Modest persistent bradycardia is often asymptomatic. When symptoms occur, they are commonly nonspecific, such as fatigue, listlessness, or dyspnea, making the attribution of symptoms to resting bradycardia difficult. Sinus bradycardia may also exacerbate congestive heart failure and limit effective use of β-blocker therapy, a cornerstone of therapy for heart failure, coronary disease, and tachyarrhythmias. When inappropriate sinus bradycardia is persistent, especially when severe, plausible symptoms are present, and alternate causes of symptoms have been excluded, pacemaker implantation is reasonable. Asymptomatic sinus bradycardia should rarely be treated with pacing unless a need for medical therapy is expected to further exacerbate bradycardia.

Chronotropic Incompetence

Cardiac output during exercise is increased by augmentation in stroke volume and an increase in heart rate. If heart rate rise with exercise is inadequate, exertional symptoms such as fatigue or dyspnea may ensue. As in the case of resting sinus bradycardia, unless severe, attribution of symptoms to chronotropic incompetence is difficult. Various criteria for this condition have been proposed that rely on the inability to achieve a set fraction of age-predicted heart rate or heart rate reserve. As for resting sinus bradycardia, the decision to implant a pacemaker for chronotropic incompetence is a matter of judgment more than criteria.

Sinus Pauses or Arrest

An abrupt failure of sinus node automaticity or failure of propagation from the sinus node to the atrium can result in a pause in atrial activity. P waves are absent, and if of adequate duration and not accompanied by a competent subsidiary escape mechanism, it can result in abrupt symptoms of lightheadedness, presyncope or true syncope. Sinus pauses of less than 3 seconds are commonly seen for normal subjects, who are rarely symptomatic. Sinus pauses exceeding 3 seconds and not occurring during sleep are often pathologic and may result in symptoms. Sinus pauses associated with simultaneous symptoms and documentation of pauses lasting 3 seconds or longer in patients with a history of symptoms plausibly related to bradycardia are indications for pacemaker therapy.

Sinoatrial Exit Block

Sinus node dysfunction is often accompanied by significant atrial fibrosis, which may lead to a block in the tissues surrounding the sinus node complex and impede propagation to the atrial tissue. Bradycardia due to sinus node dysfunction may result, not from a failure of automaticity, but from failure of propagation from the sinus node complex to the atrium. Because sinus node activity is not directly apparent from the surface ECG, the diagnosis is made indirectly by the observation of abrupt halving in the sinus P-wave rate, followed by an abrupt return to the baseline sinus rate (Fig. 9-3C and D). Although other patterns may be observed, 2 : 1 exit block is the most common. Therapy for sinoatrial exit block is identical to that for intermittent sinus bradycardia (discussed earlier).

Bradycardia-Tachycardia Syndrome as a Consequence of Sinus Node Dysfunction

Bradycardia-tachycardia ("brady-tachy") syndrome refers to a clinically significant tachyarrhythmia sometimes accompanied by clinically significant bradycardia. The term may be confusing because the mechanism of tachycardia is often unrelated to the mechanism of bradycardia.

This syndrome most commonly manifests as intermittent pathologic atrial arrhythmias, often intermittent AF with concomitant sinus node dysfunction resulting in long pauses or symptomatic sinus bradycardia when the patient is in sinus rhythm. A typical manifestation of this syndrome is a prolonged period of asystole after termination of AF (see Fig. 9-3E) due to slow recovery of sinus node automaticity with resultant presyncope or syncope.

The combination of two seemingly independent processes is in part a consequence of the high prevalence of AF and sinus node dysfunction in the elderly and the need to use potent drugs to decrease ventricular response during AF with resultant unintended secondary sinus node dysfunction between periods of atrial arrhythmias. This type of bradycardia-tachycardia syndrome represents an important form of clinical sinus node dysfunction and is a common indication for pacemaker implantation.

Sinus node dysfunction causing bradycardia-tachycardia should be distinguished from a common, unrelated form of bradycardia-tachycardia syndrome, which is characterized by chronic rather than intermittent AF with periods of rapid and slow ventricular responses. This condition is often incorrectly referred to as *sick sinus syndrome*. However, in this syndrome, the atrium is chronically fibrillating, and the sinus node therefore has no influence on heart rate. Bradycardia or protracted pauses in the setting of chronic AF is a consequence of impaired AV conduction and is unrelated to sinus node dysfunction.

Atrioventricular Conduction Disturbances

AV conduction disturbances include disorders in which the normal physiologic AV relationship is not maintained due to pathologic delay in AV conduction or to intermittent or complete loss of AV conduction. The PR interval includes three distinct phases of AV conduction. Although the individual components of AV conduction can be readily recorded by a His bundle catheter in an electrophysiology laboratory, the salient features of AV conduction disturbances can usually be elucidated by careful interpretation of the surface ECG without resorting to invasive recording techniques.

The right atrial conduction time from the area of the sinus node where the P wave begins to the region of the AV node occupies a short first portion of the PR interval and usually lasts no more than 30 milliseconds. Because the atrial conduction time is short and does not change much over time in a given patient, it can conveniently be ignored when assessing AV conduction. The second portion of the PR interval is the propagation time through the AV node, which is normally 50 to 120 milliseconds. The last component of the PR interval is the time for propagation through the His bundle and bundle branches, which is typically 30 to 55 milliseconds. Although this last portion,