Bradycardia-dependent bundle branch block is a relatively infrequent form of rate-dependent bundle branch block. When it occurs in the setting of Mobitz I second degree heart block, it must be differentiated from ventricular escape and from Mobitz II block, both of which are much more common causes of wide QRS complexes. The differential diagnosis is important since Mobitz II block has a poorer prognosis and a greater need for pacemaker therapy. We present a case of second degree atrioventricular (AV) block initially felt to be Mobitz II block because of a left bundle branch block morphology. On closer scrutiny, the tracings were more consistent with a Mobitz I mechanism and an associated bradycardia-dependent bundle branch block. Subsequent electrophysiologic study revealed no evidence of infranodal conduction abnormalities.

**CASE REPORT**

A 45-year-old man with a history of hypertension was admitted to a community hospital with severe chest pain and 3 mm ST elevation in ECG leads 2,3 and avf. The CPK enzyme elevation and Q waves in leads 3 and avf ultimately confirmed the diagnosis of acute inferior myocardial infarction. On the fourth hospital day, he developed PR prolongation to .24 second. Shortly thereafter, he was noted to have occasional nonconducted P waves followed by aberrantly conducted beats, as seen in Figure 1. On the following day, he developed sustained 2:1 AV block with left bundle branch block morphology of the conducted beats (Fig 2). Systolic blood pressure was 75, and atropine did not reduce the degree of block. A temporary pacemaker was placed.

As late as the 11th hospital day, he still had occasional nonconducted P waves. Some were preceded by definite progressive PR prolongation typical of Mobitz I block. Others, however, were preceded by little if any PR prolongation and were therefore less clearly of a Mobitz I or II mechanism (Fig 3). The patient was referred to our institution for further evaluation.

Echocardiography confirmed an akinetic inferior wall consistent with infarction. Treadmill testing and rest-stress radionuclide ventriculography revealed no evidence of persistent ischemia. Twenty-four-hour Holter monitoring and three days of telemetry revealed no further instances of AV block. Electrophysiologic study revealed a normal HV interval of 45 ms, Mobitz I AV block occurring proximal to the His bundle at a paced cycle length of 350 ms, and no evidence of split His potentials or blocked conduction distal to the His deflection. The patient was discharged without a pacemaker and has had no cardiovascular symptom in the 12 months since discharge.

**DISCUSSION**

We propose that the aberrant beat in Figure 1 is an example of bradycardia-dependent bundle branch block occurring on the first beat of a Wenckebach cycle (ie, after the nonconducted beat). The narrow complex beats meet Wenckebach criteria with progressive PR prolongation preceding the nonconducted beat. If one considers the aberrant beat as conducted, there is PR prolongation from .21 second to .28 second prior to the nonconducted beat. While the Wenckebach phenomenon virtually excludes a Mobitz II mechanism as

**FIGURE 1.** Monitor lead tracing on the fourth hospital day revealing 5:4 Wenckebach cycle with bradycardia-dependent aberrant conduction of the QRS complexes following the blocked beats.
an alternate explanation for the aberrant beat, one cannot exclude on the basis of this tracing alone the possibility that the wide complex beats represent a ventricular escape mechanism. The fact that the PR interval preceding the two aberrant beats is identical while the RR interval varies slightly makes this possibility less likely.

In Figure 2, the uniform PR interval over a sustained period excludes the possibility of a ventricular escape mechanism. Mobitz I and II mechanisms, however, cannot be differentiated on the basis of this tracing alone. While the left bundle branch block might be considered evidence of more distal Mobitz II block, the analysis above of Figure 1 and the setting of an inferior infarction in fact make Mobitz I block with an associated bradycardia-dependent bundle branch block the more likely diagnosis.

The combined analyses of Figures 1 and 2, along with the data obtained at electrophysiologic study, support the diagnosis of bradycardia-dependent bundle branch block secondary to Mobitz I atrioventricular block. Bradycardia-dependent (or deceleration-dependent) bundle branch block remains a relatively uncommon form of rate-dependent bundle branch block. In a review of the literature in 1968, Massumi evaluated the previously reported cases and found that only four stood up to close scrutiny. Many cases could be explained equally well by other mechanisms such as ventricular escape or “supernormal” conduction of early, narrow complex beats. Since then, other reports have appeared including those of Gay and Brown and Lie et al which describe this phenomenon in the setting of acute inferior myocardial infarction. The appropriate diagnosis of bradycardia-dependent bundle branch block in this setting may prevent unnecessarily aggressive pacemaker therapy.

The proposed mechanism for bradycardia-dependent bundle branch block is the phenomenon of phase 4 aberrancy. With more prolonged diastolic intervals, there is more time during which phase 4 depolarization can reduce the resting transmembrane potential. A decreased resting potential may decrease the slope of phase 0 of the action potential thereby creating the electrophysiologic substrate for aberrancy or block.
While other mechanisms such as direct vagal influence have been proposed, phase 4 aberrancy seems the most plausible explanation for this unusual electrocardiographic finding.

REFERENCES