in the patients we described, acute ischemia is a much less plausible explanation for distal AV block during exercise than is a fixed conduction system abnormality. This assessment is based not only on the absence of clinical evidence of ischemia during exercise, but also on the observation that block occurred at the onset of atrial pacing and during spontaneous increases in atrial rate unrelated to exercise—instances when the likelihood of ischemia was remote.

It is unlikely that ischemia was present but undetected because of low ventricular rates due to AV block in our patients. Patient 3 reached 85% of his predicted maximal heart rate during exercise, and Patient 2 underwent extended rapid ventricular pacing at a rate of 160 beats/min. In addition, low ventricular rates that limit myocardial oxygen consumption might be expected to prevent the occurrence of ischemia altogether, rather than simply to minimize the manifestations of ischemia on the electrocardiogram.

The study of Wayne et al. (Wayne's Reference 2) should not be used to suggest that our patients had undetected ischemia. Although coronary artery disease was documented in 12 of their 16 patients, all 12 had angina pectoris, and 6 even had a prior myocardial infarction. Certainly the prevalence of coronary disease in patients with exercise-induced conduction abnormalities who do not have angina pectoris would be expected to be much lower; in fact, the prevalence in the four such patients in the study of Wayne et al. was zero. In addition, Wayne et al. did not prove that ischemia was responsible for the exercise-induced conduction disturbance in their patients; the presence of coronary artery disease does not necessarily imply the presence of functional acute ischemia during exercise. Although exercise thallium scintigraphy was performed in a few of their patients (with positive results in only 50%), the best method to determine whether acute ischemia was present and directly responsible for the bundle branch block might have been to observe whether treatment of ischemia prevented bundle branch block from appearing during subsequent exercise.

Regarding autonomic influence on distal conduction, we agree that the His-Purkinje system may be sensitive to autonomic tone, and we emphasized only that its sensitivity is far less than that of the AV node. However, even if autonomic changes during exercise improve His-Purkinje conduction and increase the heart rate at which AV block appears, as perhaps occurred in the patient described by Chapman (Wayne's Reference 3), this would not account for the opposite finding in our Patient 3 that AV block appeared at a higher rate during atrial pacing than during exercise. Finally, by citing the occasional nonreproducibility of the heart rate at which AV block appears on serial exercise tests, we meant only to point out that if this rate may be different on serial exercise tests alone, a difference observed between an exercise test and an atrial pacing trial should not be expected.

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Idiopathic "Myocarditis"?

I am concerned about the epidemic of idiopathic "myocarditis" determined by endomyocardial biopsy as described by Zee-Cheng et al. (1). Their experience is contrary to our own. Figure 1 of their article, which is described as revealing "grade I lymphocytic myocarditis," was shown in a blinded fashion to five staff pathologists. All agreed that the photomicrograph showed nuclear enlargement and variation in size. They also agreed that there was no evidence of inflammation. Since most of the cases of myocarditis reported in this article were of the mild variety and since our pathologists did not agree with the reading of Figure 1, it may be that recent "epidemics" of myocarditis (1–3) are related more to histologic interpretation than to geographic differences in the incidence of this condition.

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Diastolic Atrial Compression in a Premature Infant

Kronzon et al. (1) demonstrated by two-dimensional echocardiography that diastolic compression of the right atrial free wall was a sensitive marker of cardiac tamponade in nine adults with pericardial effusion. This finding was recently observed in a preterm infant.

Case report. This 2,000 g premature infant girl (DOB 8/29/83) was evaluated for cardiomegaly by two-dimensional echocardiography and was found to have truncus arteriosus type 1, a large secundum atrial septal defect and a large pericardial effusion 36 hours after birth. Associated anomalies included imperforate anus, single umbilical artery and duodenal atresia. Physical examination showed all peripheral pulses to be present and bounding. The right arm blood pressure was 60/30 mm Hg. The heart rate was 140 beats/min. Cardiac examination revealed a normal first heart sound, a loud and single second heart sound and a grade 3/6 systolic ejection murmur along the left lower sternal border. The chest roentgenogram showed cardiomegaly with increased pulmonary vascularity. The electrocardiogram showed right atrial enlargement. Medical management of congestive heart failure included administration of digoxin and diuretic drugs and restriction of fluids.