The effect of an acute increase in heart rate on left ventricular function has been well documented in patients with paroxysmal arrhythmias. There is general agreement that cardiac output remains relatively constant despite a decrease in stroke volume and end-diastolic volume. These changes may be accompanied by minor wall motion abnormalities. Nonetheless, in the absence of other heart disease, initiation of supraventricular tachycardia has not resulted acutely in any overall decrease in ejection fraction compared with baseline function in sinus rhythm (1). Similarly, no appreciable change in ejection fraction has been observed in normal subjects despite atrial pacing to rates of 120 to 160 beats/min (2-4).

The effect of chronic tachycardia on otherwise normal myocardium is less certain; it remains unclear in reported cases whether left ventricular dysfunction results from chronic tachycardia or merely represents aggravation of a coexisting cardiomyopathy. In the past, treatment was confined to pharmacologic therapy, often with agents possessing negative properties. The development of catheter and intraoperative techniques to localize and ablate sources of arrhythmia has allowed this old dilemma of “which came first” to be reexamined, since these interventions effect a return to sinus rhythm without modifying the myocardial substrate.

In a previous issue of the Journal, Gillette et al. (5) reported the largest series to date of ectopic atrial tachycardia treated by direct ablative techniques. Termination of tachycardia was achieved by catheter ablation in 2 of 4 patients and by direct cryosurgery in 13 of 14 patients. Ventricular function was believed to have clinically improved in 9 of 10 patients with abnormal left ventricular function, and was confirmed by echocardiographic variables in 7 of the 9. As the authors acknowledge, the use of echocardiography to identify “improvement” is somewhat limited, since the initial measure of left ventricular function may have been artificially low because of a rapid heart rate. Nonetheless, the continued late improvement noted supports the concept that chronic tachycardia may have resulted in a reversible form of cardiomyopathy.

**Chronic tachycardia and left ventricular dysfunction.** Chronic supraventricular tachycardia was once regarded as a benign process without adverse myocardial sequelae (6,7). Subsequently, however, a relation between incessant tachycardia and cardiomyopathy has been suggested by a number of investigators (8-14). Data from my laboratory support this concept (15). Nine patients (aged 5 to 57 years) were studied with clinically significant left ventricular dysfunction presumed to be related to uncontrolled tachycardia of 2.5 to 41 years’ (mean 15.1) duration. Eight of the nine had incessant tachycardia of 0.5 to 6 years’ (mean 2.6) duration. Six of the nine had myocardial biopsy with no specific pathologic diagnosis. The rate of tachycardia ranged from 120 to 139 beats/min in two, 140 to 159 beats/min in three and was greater than 160 beats/min in three. Radionuclide angiography was performed in all patients immediately before and 11 to 51 months (mean 24) after surgery. Left ventricular function improved markedly in seven of nine patients at rest and in an additional patient during exercise. The mean ejection fraction was 19 ± 9% (range 10 to 35) in the baseline state. Early follow-up study (mean 8.4 days) showed an increase in rest ejection fraction to 33 ± 7% (p = 0.01), and late follow-up study (mean 17.4 months) showed the rest ejection fraction to be 45 ± 15% (p = 0.002). Eight of the nine patients were noted to be asymptomatic 11 to 51 months (mean 24) after the corrective procedure and had resumed normal activities.

**Pathophysiology.** The underlying pathophysiologic link between chronic incessant tachycardia and ventricular dysfunction is unknown, but might be explained by a reversible depletion of high energy substrates. Coleman et al. (16) studied six dogs that developed congestive heart failure after 13 to 29 days of pacemaker-induced tachycardia. Twenty-four hours after cessation of tachycardia, persistent heart failure was manifested by elevated left ventricular end-di-
astolic pressure and decreased ejection fraction. The maximal isovolumic end-diastolic tension developed for a given end-diastolic volume and the maximal shortening velocity (Vmax) were significantly lower than in control dogs without congestive failure. The left ventricular stores of creatine, creatine phosphate and adenosine triphosphate (ATP) were also significantly depleted. The investigators concluded that left ventricular dysfunction might be related to a depletion of left ventricular energy stores.

The rapid repletion of such substrates may explain the reversal seen in the patients reported on by Gillette et al. as well as by others (12,14,15). Improved function has been observed as early as 7 days in canine studies in which high energy phosphate levels were depleted by transient ischemia. Kløner et al. (17) found that canine ATP and creatine phosphate stores (depleted after 15 minutes of coronary artery occlusion) and accompanying left ventricular dysfunction returned to normal within 7 days after treatment, with a further improvement of ejection fraction noted in long-term follow-up study.

Therapeutic implications. A detailed commentary on the various therapeutic modalities available is beyond the scope of this editorial. Nevertheless, some issues raised by the report of Gillette et al. (5) should be critically examined. Although the arrhythmias treated were believed to be due to abnormal automaticity, diphenylhydantoin (a drug useful in treating such arrhythmias) was noticeably absent in the drug trials described. Similarly, one might speculate about the potential utility of amiodarone in these cases. Although amiodarone has caused a number of toxic side effects in adults, it is generally well tolerated in children and might have been considered for short-term management of the arrhythmias described. Indeed, suppression of arrhythmia was achieved in the single instance in which amiodarone was utilized.

Role of catheter ablation techniques. Much remains to be learned of the long-term effects of catheter ablation. Although this new technique appears extremely promising, it remains to be demonstrated that the "small visible scar" will remain invisible clinically in the years after the procedure. A small growing heart, for example, may be more prone to develop arrhythmogenic sequelae from such a scar, a situation increasingly evident in the long-term follow-up of surgically corrected cases of congenital heart disease. No short-term complications were encountered in the series of Gillette et al. (5), although it would appear prudent to advise against early discharge of these patients because of possible late tamponade.

The introduction of ablative techniques for the treatment of tachycardia, especially those employing catheter delivery systems, has clearly broken new ground; the diagnostic study now carries the potential for a therapeutic result. In view of the investigative nature of these techniques, more long-term follow-up study of data acquired in carefully controlled series will be necessary to assess the benefit-risk ratio of this promising approach to treatment.

Until the "chicken-egg" dilemma is resolved, the hypothesis that significant left ventricular dysfunction may result from chronic uncontrolled tachycardia in the absence of other cardiac disease should be carefully considered in any patient presenting with these findings, since the dysfunction appears to be reversible with definitive control of the tachycardia.

References