Alcoholic Cardiomyopathy and Ventricular Arrhythmias

To the Editor:

We read with interest the article by Piano et al1 (May 2002) about alcoholic cardiomyopathy (ACM), with a complete clinical and physiopathologic overview on this leading cause of nonischemic heart failure. We agree with all the comments and conclusion of the author, and we thank Dr. Piano for the several citations of our work published in 2000.2 We would like to add a short comment on the problem of ACM and arrhythmias since this issue remains unclear. Alcohol intake is associated with biological changes, such as potassium depletion that may induce arrhythmogenesis. Our work was one of the very few that took into account some arrhythmia risk markers in the evaluation of ACM. Acute consumption of alcohol is a well-known etiology of paroxysmal atrial arrhythmias3,4 with the so-called “holiday heart syndrome,” but there is little information concerning the prevalence or incidence of ventricular arrhythmias in ACM. We found that the prevalence of atrial arrhythmias and of sustained or nonsustained ventricular tachycardia (VT) was not significantly different in either group of patients with idiopathic cardiomyopathy (IDCM) or ACM. A study has found that late ventricular potentials on signal-averaged ECG in long-term alcoholics without preexisting heart disease was associated with more severe steatosis, suggesting that this method could detect early alterations in the myocardium.5 However, we found no difference in the prevalence of late ventricular potentials on signal-averaged ECG in patients with ACM and IDCM.

Currently, our database includes 194 patients with nonischemic dilated cardiomyopathy: 119 patients with IDCM and 75 patients with ACM. Among the latter, we observed 47 patients with complete or almost complete alcohol abstinence and 28, respectively, without alcohol abstinence with a mean ± SD follow-up (FU) of 51 ± 42 months. We now have information on the occurrence of major arrhythmic events during FU (sudden death, sustained VT, ventricular fibrillation [VF]) that we did not published in our initial work of 2000. Figure 1 shows the actuarial curve of events (14 sudden deaths, 10 sustained VT/VF) for the three groups of patients. These data confirm that the prognosis appears similar concerning this aspect of the prognosis for patients with IDCM and ACM without abstinence, and appears much better for patients with ACM and abstinence (only 2% of events during FU).

Therefore, alcohol intake does not seem to worsen prognosis on ventricular arrhythmias by comparison to IDCM; however, alcohol abstinence in ACM is associated with a very good prognosis concerning the risk of sudden death, which may be related to the improvement of the left ventricular function. This is further argument to suggest that an aggressive approach to alcohol cessation is needed in these patients.

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Figure 1. Survival curves of sudden deaths and major arrhythmic events (sustained VT, VF) in patients with ACM and IDCM. The solid line indicates patients with ACM and alcohol abstinence, the small dashed line indicates IDCM, and the large dashed line indicates patients with ACM without abstinence. Compared to patients with ACM and alcohol abstinence, relative risk of events was 8.0 for patients with ACM without abstinence (log-rank test, p = 0.01) and 7.3 for patients with IDCM (log-rank test, p = 0.03).