

The Effect of Controlled Drinking in Alcoholic Cardiomyopathy

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Background: Cardiomyopathy is a potentially fatal complication of alcohol abuse. In alcoholic persons who develop cardiac dysfunction, abstinence is thought to be essential to halt further deterioration of cardiac contractility. Some evidence indicates that reducing alcohol intake may also be beneficial.

Objective: To evaluate the effect of moderate “controlled” drinking on cardiac function in patients with alcoholic cardiomyopathy.

Design: 4-year prospective cohort study.

Setting: A university hospital in Barcelona, Spain.

Patients: 55 alcoholic men with cardiomyopathy who had been drinking a minimum of 100 g of ethanol per day for at least 10 years.

Measurements: Evaluation of ethanol intake and nutrition, clinical assessment of cardiac status, and sequential echocardiography and radionuclide cardiac angiography.

Results: After the first year of evaluation, all patients with car-

diomyopathy who abstained from alcoholic beverages demonstrated significant improvement in left ventricular function (average increase in left ventricular ejection fraction, 0.131 [95% CI, 0.069 to 0.193]). Patients who drank 20 to 60 g of ethanol per day showed a comparable mean improvement of 0.125 (CI, 0.082 to 0.168). In contrast, left ventricular ejection fraction deteriorated further in most patients who continued to abuse alcohol (>80 g/d). After 4 years, left ventricular ejection fraction had continued to improve in both abstinent patients and those who controlled their drinking. Ten patients who had continued to consume more than 80 g of ethanol per day died during the study.

Conclusion: In patients with alcoholic cardiomyopathy, both abstinence and controlled drinking of up to 60 g of ethanol per day (four standard drinks) were comparably effective in promoting improvement in cardiac function.

Ann Intern Med. 2002;136:192-200.

www.annals.org

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Alcoholic cardiomyopathy is a complication of long-standing alcohol abuse and is related to a person's total lifetime dose of ethanol (1, 2). The malady is not uncommon; in developed countries, it accounts for 3% to 40% of all cases of dilated cardiomyopathy (3–6). In our previous studies, one third of asymptomatic alcoholic persons showed some evidence of cardiac dysfunction (1, 2, 7). Although the natural course of chronic alcoholic cardiomyopathy is not precisely defined, abstinence from alcohol has been reported to improve cardiac function, or at least halt its deterioration (8–10).

The treatment of alcoholic cardiomyopathy is generally assumed to require abstinence (11), and any further alcohol intake is thought to be deleterious. Yet abstinence is not always possible, and a sizable proportion of persons treated for alcoholism report that they are able to drink moderately (12). We therefore designed a 4-year prospective cohort study of patients with alcoholic cardiomyopathy. Although total abstinence was urged for all patients at the initial interview and on subsequent occasions, fewer than one third stopped drinking completely. A similar number maintained a

controlled drinking pattern, and the remainder continued to abuse alcohol. We evaluated the effect of “controlled” drinking, defined as a daily intake of 20 to 60 g of alcohol per day (one to four standard drinks), in patients with alcoholic cardiomyopathy.

METHODS

Patient Selection

All patients were younger than 60 years of age and reported consuming more than 100 g of ethanol per day for at least 10 years. Over a 6-year period, 51 alcoholic patients were admitted to the cardiology unit or the emergency department of Hospital Clínic, Barcelona, Spain, with signs or symptoms of heart failure (New York Heart Association [NYHA] functional class II to IV) (13), left ventricular ejection fraction of 0.5 or less, and no evidence of any heart disease other than dilated cardiomyopathy. Alcoholic cardiomyopathy was diagnosed in all of these patients, and they were consecutively recruited for the study. Fifteen patients who presented to the outpatient alcoholism unit during the same period for assistance in terminating their depen-

dence on alcohol, displayed cardiac enlargement on radiography, and were found to have alcoholic cardiomyopathy (left ventricular ejection fraction < 0.5) were also included in the study. The cardiothoracic index was abnormally large in all patients (≥ 0.5). In addition, all but 4 of the patients had an end-diastolic diameter greater than 2 standard deviations above that of a group of healthy volunteers.

Of the 66 alcoholic persons initially evaluated, 11 were excluded. Five had severe withdrawal symptoms, 1 reported cocaine use, and 1 was infected with HIV. Two patients with evidence of coronary artery disease were not considered good candidates for the study. Two patients had such severe alcoholic cardiomyopathy (ejection fraction < 0.1) that they died shortly after admission. Thus, a total of 55 patients with alcoholic cardiomyopathy remained in the study. No patients objected to inclusion, and all gave informed consent for the various procedures. The Institutional Review Board of Hospital Clínic approved the study protocol.

All patients were white men of Spanish descent who lived with their families in or around Barcelona and had histories of stable employment. Most were skilled laborers or office workers, and none were indigent. As part of our standardized program designed to terminate dependence on alcohol, all patients were repeatedly urged to become abstinent. The proportion of patients who stopped drinking was similar to that reported by others (12, 14).

To assess the normal variability of cardiac function over time, we also evaluated 10 healthy men (median age, 45 years [range, 35 to 45 years]) who reported drinking less than 20 g of ethanol per day. It is important to note that alcoholic beverages are part of Spanish culture and that total abstention from alcohol is uncommon. The controls were studied at the beginning and end of the study in the same manner as the men with alcoholism.

Clinical Studies

One of two physicians obtained a detailed history of ethanol intake for each patient using repeated interviews and a validated structured questionnaire (15, 16). In our experience, intra- and interobserver reliability of this type of assessment are greater than 90%. Data were confirmed by consultation with family members. Minor discrepancies in alcohol intake were found in five cases

(9%), which were resolved by further consultation with the patients and their families. Frequency and amount of daily ethanol intake since patients began drinking habitually were recorded. Life events, such as marriage, military service, and work posts, were used as “anchor points” to help recollection (time-line follow-back method [15]). The total lifetime dose of ethanol was estimated by multiplying the amount of ethanol consumed per day by the number of years of each alcohol intake period multiplied by 365 and adding the total amounts ingested during these different periods (1, 7). Withdrawal symptoms were evaluated according to the Clinical Institute for Withdrawal Assessment Scale (17).

Laboratory and Nutritional Studies

Blood samples for laboratory tests were obtained the day after admission. Overall nutrition was assessed in terms of the proportion of actual weight to ideal weight. The lean body mass and muscular area of the arm were calculated from the circumference of the upper non-dominant arm and the thickness of the tricipital skin fold, respectively. The fatty area of the arm, calculated from the thickness of the tricipital fold, was considered indicative of total body fat (1, 18). Body mass index was determined as the body weight relative to the square of the body height (kg/m^2). Patients were considered to have caloric malnutrition if their body weight was less than 80% of their ideal weight or if the calculated lean body mass was more than 10% below the control value. Protein malnutrition was diagnosed when patients had abnormally low values for three of the following: hemoglobin, lymphocyte count, total protein level, albumin level, prealbumin level, retinol-binding protein level, or transferrin level (18, 19). Hepatic ultrasonography was performed in all patients. Percutaneous needle biopsies of the liver were performed when serum aminotransferase levels remained elevated to more than twice the normal values for more than 2 months or when ultrasonography showed liver abnormalities.

Cardiac Studies

Past and present signs and symptoms of heart failure were evaluated, and NYHA functional class was determined according to the Goldman activity scale (13). Three days after admission, when results of tests for blood alcohol were uniformly negative, chest radiogra-

Table 1. Baseline Epidemiologic, Nutritional, and Clinical Characteristics of 55 Alcoholic Men with Cardiomyopathy*

Characteristic	Value
Age, y	48.1 ± 7.3
Daily ethanol intake, g	208 ± 56
Duration of alcoholism, y	26.6 ± 7.3
Lifetime dose of ethanol, kg/kg body of weight	26.1 ± 8.1
Proportion of ideal body weight, %	101.7 ± 17.6
Lean body mass, kg	51.3 ± 4.5
Body mass index, kg/m ²	24.2 ± 4.6
Tricipital skin fold, mm	10.8 ± 5.9
Fat area of the arm, cm ²	11.0 ± 5.1
Hemoglobin level, g/L	141 ± 15
Lymphocyte count, ×10 ⁶ cells/L	1881 ± 661
Total protein level, g/L	70.8 ± 7.3
Albumin level, g/L	40.8 ± 6.6
Prealbumin level, mg/dL	26.4 ± 11.0
Retinol-binding protein level, mg/dL	5.2 ± 2.2
Transferrin level, mg/dL	250 ± 51
NYHA functional class, n (%)	
Class I	11 (20)
Class II	13 (24)
Class III	18 (32)
Class IV	13 (24)
Cardiothoracic index	0.53 ± 0.02

* Data expressed with a plus/minus sign are the mean ± SD. NYHA = New York Heart Association.

phy, conventional electrocardiography, bidimensional echocardiography (Toshiba SS-10 instrument, Otawara, Japan), and technetium-99 radionuclide angiocardiology (Picker-Dyna 4-15 gamma-camera, Haifer, Israel) were performed. End-diastolic and end-systolic diameters, the shortening fraction, and the mass of the left ventricle were measured according to the standards of the American Society of Echocardiography (20). Values for left ventricular ejection fraction were obtained by using isotopic angiography, which is reported to be a reliable method (1, 2, 21). The personnel who performed and evaluated these tests had no knowledge of the alcoholic history of the patients. To rule out ischemic heart disease, patients underwent a treadmill electrocardiographic test.

At the end of the first year and annually thereafter for up to 4 years, interviews, laboratory tests, and clinical examinations were repeated for all patients. Ethanol consumption, pharmacologic treatment, and incidental disease were recorded at each interview. At the end of the first year of follow-up, patients were classified into one of four groups according to level of alcohol consumption: 1) complete abstinence; 2) controlled drinking of 20 to 60 g of ethanol per day, a range commonly

accepted as moderate (22); 3) an indeterminate range of 60 to 80 g of ethanol per day; and 4) alcohol abuse, defined as more than 80 g of ethanol per day. For patients who died during follow-up, death was classified as cardiologic or noncardiologic.

Statistical Analysis

We used standard statistical methods from the SPSS Statistical Analysis System, version 9.0 (SPSS, Inc., Chicago, Illinois) (23). In each group of patients, we used the paired *t*-test when comparing variables from different time periods.

Role of the Funding Sources

The funding sources had no role in the design, analysis, or interpretation of the study or in the decision to submit the results for publication.

RESULTS

Clinical Characteristics

Patients with alcoholic cardiomyopathy ranged in age from 31 to 59 years. Alcoholic beverages were generally consumed continuously as part of everyday life, and no binge drinking was reported. Patients most often drank wine, beer, or brandy and less frequently drank anisette, whiskey, or gin. The mean daily ethanol intake was 208 g (range, 100 to 310 g) over 26.6 years (Table 1). The mean total lifetime dose of ethanol (26.1 kg/kg of body weight [range, 12 to 42 kg/kg]) was similar to that observed in previous studies (1, 2). Most of the patients (75%) had smoked one to two packs of cigarettes per day since the second decade of their lives. None of the patients used illicit drugs.

Nutritional Status and Laboratory Data

Nutritional and laboratory variables are shown in Table 1. Six patients exhibited mild caloric malnutrition, and 4 had slight protein malnutrition. Of the 28 patients who had liver biopsy, 3 had normal livers, 17 had cirrhosis, 6 had fatty liver, and 2 had mild alcoholic hepatitis. Among the 17 patients with cirrhosis, 6 had hepatitis C virus antibodies and 1 had antibodies against hepatitis B virus but was negative for surface antigen. None of the patients had signs or symptoms of liver failure; all were categorized as class A by Child-Pugh classification (24).

Initial Cardiac Status

At study entry, 24 patients were classified as NYHA functional class I to II and 31 were classified as class III to IV (Table 1). Electrocardiographic studies showed atrial fibrillation in 14 patients, conduction defects in 12, and premature ventricular contractions in 17. All patients had abnormally elevated end-diastolic and end-systolic diameters and left ventricular mass. They also had abnormally low left ventricular shortening fractions and ejection fractions (Table 2). All patients were normotensive (mean blood pressure [\pm SD], $127 \pm 14/77 \pm 14$ mm Hg). During the study, patients were treated according to NYHA functional class. In addition to ethanol abstinence, we recommended a dietary sodium intake of less than 100 mmol/d for all patients. Patients who had NYHA class II to IV disease ($n = 44$) were treated with an angiotensin-converting enzyme inhibitor (captopril, 25 to 50 mg three times per day) according to individual tolerance. In addition, 18 patients who had NYHA class III to IV disease and presented with congestive heart failure received furosemide (20 to 40 mg twice per day) and spironolactone (25 to 50 mg/d). Eleven patients with atrial fibrillation also

Table 2. Left Ventricular Function at Study Entry in Patients with Alcoholic Cardiomyopathy and in Healthy Volunteers*

Variable	Patients with Alcoholic Cardiomyopathy ($n = 55$)	Healthy Volunteers ($n = 10$)
End-diastolic diameter, mm	62.2 ± 8.9	48.0 ± 1.9
End-diastolic diameter index, mm/m ²	33.9 ± 5.2	27.7 ± 1.2
End-systolic diameter, mm	45.8 ± 11.3	30.3 ± 4.2
End-systolic diameter index, mm/m ²	24.9 ± 5.9	17.4 ± 2.4
Shortening fraction, %	26.5 ± 8.2	38.2 ± 7.2
Left ventricular mass, g	280 ± 99	183 ± 36
Left ventricular mass index, g/m ²	153 ± 51	106 ± 22
Ejection fraction	0.393 ± 0.113	0.667 ± 0.049

* Data are expressed as the mean \pm SD. End-diastolic and end-systolic diameters, shortening fraction, and left ventricular mass were measured according to the standards of the American Society of Echocardiography (20). Left ventricular ejection fraction was obtained by isotopic angiography.

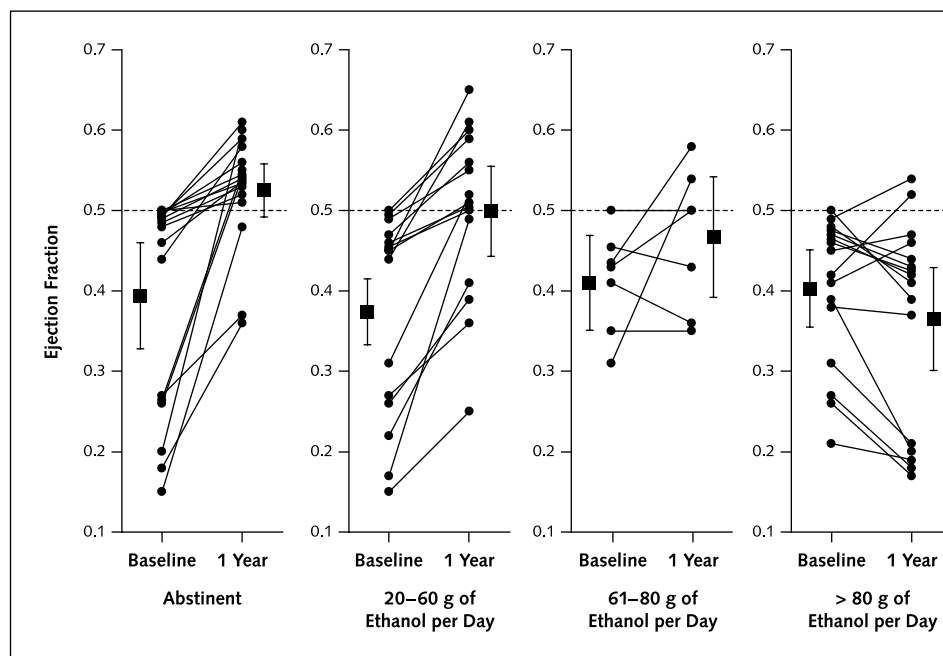
received digoxin (0.25 mg/d). No patient was treated with β -blockers or hydralazine during the study.

First-Year Follow-up Studies

Ethanol Consumption

During the first year of the study, 17 patients stopped drinking entirely, 15 reduced their ethanol in-

Figure. Changes in left ventricular ejection fraction in patients with alcoholic cardiomyopathy, according to daily ethanol intake during the first year of the study.



Group values (*squares*) are expressed as means; error bars represent 95% CIs.

Table 3. Changes in Cardiac Function at 1-Year Follow-up, according to Alcohol Consumption*

Variable	Abstinent (n = 17)			20 to 60 g of Ethanol per Day (n = 15)		
	Baseline	1 Year	Mean Difference (95% CI)	Baseline	1 Year	Mean Difference (95% CI)
End-diastolic diameter, mm	60.8 ± 11.1	56.7 ± 6.8†	-4.1 (-7.6 to -0.6)	63.0 ± 8.5	59.4 ± 7.8‡	-3.5 (-5.6 to -1.4)
End-diastolic diameter index, mm/m ²	32.9 ± 6.1	30.7 ± 3.4†	-2.2 (-4.1 to -0.3)	33.1 ± 4.9	31.2 ± 4.5‡	-1.8 (-2.9 to -0.7)
End-systolic diameter, mm	44.2 ± 11.8	38.9 ± 4.6†	-5.3 (-10.1 to -0.5)	48.2 ± 11.7	42.8 ± 9.6‡	-5.3 (-8.5 to -2.8)
End-systolic diameter index, mm/m ²	23.9 ± 6.0	21.0 ± 2.5†	-2.9 (-5.4 to -0.3)	25.2 ± 5.8	22.4 ± 4.9‡	-2.7 (-4.4 to -1.0)
Shortening fraction, %	27.3 ± 7.1	30.7 ± 5.0†	3.3 (0.2 to 6.5)	24.2 ± 10.8	29.9 ± 6.7†	5.2 (1.4 to 8.9)
Left ventricular mass, g	280 ± 108	248 ± 60	-32 (-68.5 to 4.5)	293 ± 80	261 ± 59	-32 (-66.3 to 2.3)
Left ventricular mass index, g/m ²	151 ± 56	131 ± 28†	-20 (-39.5 to -0.4)	153 ± 39	136 ± 30†	-17 (-34.2 to 0.1)
Ejection fraction	0.394 ± 0.135	0.525 ± 0.068§	0.131 (0.069 to 0.193)	0.374 ± 0.127	0.499 ± 0.107§	0.125 (0.082 to 0.168)

* Data expressed with a plus/minus sign are the mean ± SD.

† $P < 0.05$ when compared with baseline for each group (paired t -test).

‡ $P < 0.01$ when compared with baseline for each group (paired t -test).

§ $P < 0.001$ when compared with baseline for each group (paired t -test).

take to 20 to 60 g/d, 7 consumed 61 to 80 g/d, and 16 continued to drink more than 80 g/d. The level of ethanol consumption remained stable during the first year. Since alcoholic persons do not tend to exaggerate and often underreport the amount they continue to drink, we consider these reports to be minimal estimates. This modification of ethanol intake was similar to that observed in other studies (12, 14) and correlated with changes in γ -glutamyl transpeptidase levels. Even patients who continued to drink heavily reduced their alcohol consumption substantially. Among those who persisted in consuming more than 80 g of ethanol per day, the mean daily intake (\pm SD) decreased from 221 ± 51 g to 150 ± 38 g in the first year of follow-up. Nutritional, caloric, and protein variables did not differ at baseline among the four groups.

Cardiac Studies

Baseline cardiac status and therapy during the first year of follow-up were similar among the four groups of patients. During the first year of follow-up, 7 of the 17 patients who abstained from alcohol received only captopril and 6 were treated with captopril plus diuretics. Of the 15 patients who controlled their ethanol intake, 6 were treated with captopril and 6 were given captopril plus diuretics. In the group of patients who consumed 61 to 80 g of ethanol per day, 6 received treatment with captopril, 1 of them in addition to diuretics. Eight patients who continued drinking more than 80 g/d were treated with captopril, and 5 received captopril plus diuretics. Two patients who drank 60 to 80 g of ethanol per day and 3 patients in each of the other groups were

treated with digoxin. All patients adhered to recommended drug regimens during the first year of follow-up, and no patients died during this period.

By the end of first year of follow-up, left ventricular function improved in all abstinent patients (mean increase in left ventricular ejection fraction, 0.131 [CI, 0.069 to 0.193]) (Table 3, Figure). The most important and unexpected finding was related to moderate or "controlled" alcohol consumption, defined as 20 to 60 g/d (22). Cardiac contractility improved in all 15 patients with alcoholic cardiomyopathy who controlled their alcohol consumption. The mean increase in left ventricular ejection fraction was 0.125 (CI, 0.082 to 0.168), which is close to the increase seen in patients who abstained from alcohol (Table 3). Patients who drank at an intermediate level, defined as 61 to 80 g/d, had mixed results. As expected, left ventricular ejection fraction deteriorated further in most patients who continued to abuse alcohol (>80 g/d) (mean decrease, -0.038 [CI, -0.001 to -0.075]). Of interest, in this group, 4 patients who continued to drink heavily but had reduced their alcohol intake by approximately 50% actually demonstrated a functional improvement. End-diastolic and end-systolic diameters, left ventricular mass, and the shortening fraction also improved in patients who abstained from alcohol and those who drank moderately (Table 3). Indices of left ventricular function remained unchanged in healthy volunteers during the follow-up period.

As reported elsewhere (1, 25), age, nutrition, and the presence of cirrhosis did not influence the changes in functional measures. No significant changes in blood

Table 3—Continued

61 to 80 g of Ethanol per Day (n = 7)			>80 g of Ethanol per Day (n = 16)		
Baseline	1 Year	Mean Difference (95% CI)	Baseline	1 Year	Mean Difference (95% CI)
63.1 ± 4.5	61.5 ± 3.3	-1.6 (-4.7 to 1.4)	62.3 ± 8.9	61.0 ± 11.9	-1.3 (-3.7 to 1.1)
33.4 ± 3.8	32.5 ± 2.3	-0.9 (-2.5 to 0.6)	37.2 ± 6.0	36.4 ± 7.5	-0.8 (-2.3 to 0.7)
46.0 ± 7.1	44.3 ± 6.5	-1.6 (-5.3 to 2.0)	45.5 ± 13.3	46.6 ± 14.3	1.0 (-1.1 to 3.1)
24.3 ± 3.8	23.3 ± 2.6	-0.9 (-2.9 to 0.9)	27.2 ± 8.3	27.9 ± 8.9	0.7 (-0.6 to 2.0)
27.1 ± 6.9	28.3 ± 7.2	1.1 (-3.3 to 5.6)	24.3 ± 8.2	25.0 ± 9.2	0.6 (-1.6 to 2.8)
285 ± 63	285 ± 60	0 (-34.2 to 34.2)	268 ± 84	260 ± 79	-8 (-46.9 to 30.9)
152 ± 42	151 ± 34	-1.4 (-19.4 to 16.6)	159 ± 50	155 ± 47	-4 (-25.3 to 17.3)
0.41 ± 0.07	0.467 ± 0.089	0.057 (-0.037 to 0.151)	0.403 ± 0.094	0.365 ± 0.128†	-0.038 (-0.075 to -0.001)

pressure were detected during the study. At the end of the first year of follow-up, NYHA functional class had improved in 9 patients who abstained from alcohol, 8 who reduced their ethanol intake to 20 to 60 g/d, 2 who consumed 61 to 80 g/d, and 2 who continued to drink heavily. Conversely, NYHA functional class worsened in 1 patient who drank 70 g/d and 4 patients who continued to drink heavily. We adjusted therapy according to NYHA functional class and the presence of congestive heart failure, as described earlier. Digoxin was withdrawn in 3 patients whose atrial fibrillation was reversed.

Long-Term Follow-up Studies

Patients were studied for 3 years after the initial 1-year follow-up. Left ventricular ejection fraction worsened in 6 alcoholic patients who were abstinent during the first year of the study but then relapsed and con-

sumed more than 80 g of ethanol per day (mean decrease, 18.4% [CI, 9.9% to 26.9%]; $P = 0.007$). In contrast, left ventricular ejection fraction improved in 5 patients who drank more than 80 g/d for the first year and then became abstinent (mean increase, 13.2% [CI, 3.2% to 23.1%]; $P = 0.006$). Patients who drank 60 to 80 g of ethanol per day during follow-up tended to show a deterioration in left ventricular ejection fraction and a progressive increase in end-diastolic and end-systolic diameters. As expected, cardiac function decreased substantially in most patients who continued to drink more than 80 g of ethanol per day.

Because controlled drinking proved to be an effective way to improve left ventricular function during the first year, we examined whether such improvement was maintained during long-term follow-up (Table 4). Twelve alcoholic patients remained in the controlled

Table 4. Cardiac Function in Healthy Volunteers and in Alcoholic Patients with Cardiomyopathy Who Drank Moderately*

Variable	Healthy Volunteers (n = 10)			Patients Consuming 20 to 60 g of Ethanol per Day (n = 12)		
	1 Year	4 Years	Mean Difference (95% CI)	1 Year	4 Years	Mean Difference (95% CI)
End-diastolic diameter, mm	57.6 ± 5.8	55.0 ± 4.2†	-2.6 (-4.2 to -0.9)	60.3 ± 9.3	60.6 ± 11.0	0.3 (-3.5 to 4.1)
End-diastolic diameter index, mm/m ²	31.3 ± 3.4	29.9 ± 2.8†	-1.4 (-2.3 to -0.4)	31.7 ± 5.5	31.8 ± 5.8	0.1 (-1.8 to 2.0)
End-systolic diameter, mm	38.1 ± 3.9	36.8 ± 3.9	-1.2 (-3.2 to 0.7)	44.2 ± 10.5	47.2 ± 12.4	3 (-0.8 to 6.8)
End-systolic diameter index, mm/m ²	20.7 ± 2.2	20.0 ± 1.9	-0.6 (-1.6 to 0.4)	22.8 ± 6.1	24.3 ± 6.4	1.4 (-0.5 to 3.4)
Shortening fraction, %	33.0 ± 5.0	34.6 ± 2.6	1.6 (-1.7 to 5.0)	29.0 ± 6.7	25.4 ± 6.8	-3.6 (-7.0 to -0.1)
Left ventricular mass, g	241 ± 32	248 ± 40	7 (-8.5 to 22.6)	276 ± 47	287 ± 77	11 (-28.3 to 51.7)
Left ventricular mass index, g/m ²	130 ± 19	133 ± 22	3 (-8.4 to 14.4)	143 ± 27	147 ± 34	4 (-15.8 to 24.8)
Ejection fraction	0.52 ± 0.063	0.619 ± 0.065†	0.096 (0.044 to 0.149)	0.474 ± 0.105	0.515 ± 0.103†	0.041 (0.012 to 0.068)

* Data expressed with a plus/minus sign are the mean ± SD.

† $P < 0.01$ when compared with 1-year values for each group (paired t -test).

drinking group (20 to 60 g/d) for 3 additional years. Of these, left ventricular ejection fraction remained stable or improved further in 11 (mean increase, 0.041 [CI, 0.012 to 0.068]) and decreased by 0.02 in 1. This additional improvement in left ventricular ejection fraction was somewhat less than that observed in the long-term abstainers (mean increase, 0.096 [CI, 0.044 to 0.149]). At the end of the study, 2 patients who drank moderately but none of the patients who abstained continued to receive furosemide and captopril. After the 4-year study was completed, patients were followed for additional periods of 2 to 8 years. During this time, no patients had myocardial infarction or developed angina.

Mortality

No patients died in the first year of the study. Ten patients, all of whom continued to consume more than 80 g of ethanol per day after the first year of follow-up, died during the study. Seven patients died of end-stage heart disease, and 3 patients died of noncardiac diseases (hepatic failure, lung cancer, and pneumonia). Those who died of cardiac causes tended to show greater end-diastolic and end-systolic baseline indices and exhibited a progressive decrease in left ventricular ejection fraction and an increase in end-diastolic diameter.

DISCUSSION

It is generally agreed that the functional and morphologic characteristics of alcoholic cardiomyopathy do not differ from those of idiopathic dilated cardiomyopathy (26, 27). The diagnosis relies on the absence of known causes of dilated cardiomyopathy, the identification of excessive ethanol consumption, and an improvement in cardiac function after abstinence from alcoholic beverages. As we (1–3) and others (28–32) have previously observed, long-term alcohol abuse leads to impairment of left ventricular function. In evaluations of alcohol-induced tissue damage, studies of the heart offer an advantage over those of other organs since the assessment of contractile function is a quantitative measure.

In our current study, cardiac function improved in all abstinent patients and deteriorated in a large proportion of those who did not change their drinking patterns. Contributory factors other than ethanol, such as differences in drug treatment during the study, were excluded. Compensated liver disease itself does not ad-

versely influence cardiac function (25). No patients developed hypertension, and no evidence of ischemic heart disease was seen. We have followed more than 300 alcoholic patients for up to 10 years and have observed only 1 myocardial infarction and 1 case of angina. This situation probably reflects the protective effect of alcohol consumption on coronary artery disease (33).

Demakis and colleagues (10) showed that abstinence from alcohol is associated with a more favorable clinical course in patients with alcoholic cardiomyopathy. In a more recent study, Guillo and associates (34) concluded that left ventricular ejection fraction improves in alcoholic persons with NYHA class IV heart failure if complete abstinence is accomplished. Perhaps because most of our patients consumed alcoholic beverages during the entire day and with meals rather than in patterns of binge drinking, more than a quarter chose to limit ethanol intake to 20 to 60 g/d rather than abstain completely. Of interest, after 1 year in the study, patients who limited their ethanol intake showed improvements in left ventricular function similar to those in patients who abstained. The left ventricular mass index at baseline was significantly higher in alcoholic patients than in the healthy volunteers, a finding similar to that in previous studies (1, 3, 28). Patients who abstained from alcohol and those who controlled their drinking both experienced a regression of left ventricular hypertrophy, a situation reminiscent of that observed after the control of hypertension (35) or the replacement of damaged heart valves (36). By the end of the study, left ventricular ejection fraction had remained stable or improved in almost all patients who controlled their drinking. No cardiac deaths were recorded among alcoholic patients who reduced their alcohol intake to a moderate level, whereas the total cardiac mortality rate was 41% after 5 years in those who continued to abuse alcohol (>80 g of ethanol per day). Survival analysis comparing the different groups was limited by the number of cases, individual drinking patterns, and duration of follow-up. Although continued ethanol abuse usually had a deleterious effect on cardiac function, we noted that cardiac function actually improved in several alcoholic patients who continued to consume more than 80 g of ethanol per day. Each of these patients had reduced his previous alcohol intake by more than 50%.

Our data provide preliminary evidence of a threshold for alcohol consumption, probably about 60 g/d

(about four standard drinks), below which no damage is inflicted on the myocardium. A similar threshold has been suggested for alcoholic myopathy (37, 38) and alcoholic liver disease (39). Thus, in clinical practice, when cardiac function deteriorates in a presumably abstinent or near-abstinent alcoholic patient, the patient probably has another medical condition or is not being truthful about his or her alcohol consumption. It is noteworthy that not all patients with chronic alcoholism, even those who drink particularly large amounts of ethanol, develop cardiac dysfunction. A genetic component has been documented for idiopathic dilated cardiomyopathy (26), and we recently obtained preliminary evidence of a similar familial tendency in alcoholic cardiomyopathy (Unpublished data). Our current results, which show left ventricular improvement in alcoholic patients with dilated cardiomyopathy who converted to moderate drinking, should not be extrapolated to patients with other types of left ventricular dysfunction regardless of whether they have heart failure.

In summary, left ventricular function improved considerably in patients with alcoholic cardiomyopathy who achieved abstinence and to a similar extent in those who controlled their alcohol consumption. A larger study is needed to demonstrate key differences between groups (for example, whether patients who drink moderately survive longer than those who drink heavily). A management strategy recommending continued use of alcohol at a reduced level (as opposed to total abstinence) was not evaluated and cannot yet be advocated. Abstinence remains the cornerstone of any alcohol treatment program and continues to be recommended to all alcoholic patients with dilated cardiomyopathy.

From Institut d'Investigacions Biomèdiques August Pi i Sunyer, University of Barcelona, Barcelona, Spain; and Jefferson Medical College, Philadelphia, Pennsylvania.

Grant Support: By grants from Fondo de Investigaciones Sanitarias (FIS 98/0330 and 99/0115) and by Generalitat de Catalunya (CUIR 1999/SGR-279).

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