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# Acute alcohol consumption is associated with increased interatrial electromechanical delay in healthy men

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## Abstract

**Background:** *Acute alcohol consumption can cause atrial fibrillation in patients with, and without, heart disease. Increased atrial electromechanical delay (EMD) has been associated with atrial fibrillation. We evaluated the atrial conduction properties by tissue Doppler imaging (TDI) echocardiography in healthy men following acute alcohol intake.*

**Methods:** *Thirty healthy male volunteers were included in this study. Baseline ECG, heart rate, blood pressure, and TDI echocardiographic findings were compared to readings taken one hour after drinking six 12-oz cans of beer (76.8 g of ethanol).*

**Results:** *Although the blood pressure and heart rate remained similar before and one hour after alcohol intake, Pmax and Pd values were significantly prolonged (114.2 ± 10.4* vs *100.8 ± ± 10.6, $p = 0.002$; 50.6 ± 9.6* vs *34.5 ± 8.8, $p < 0.0001$). Interatrial EMD was significantly increased after drinking alcohol compared to the baseline (19.8 ± 9.2* vs *14.0 ± 5.5 ms, $p < 0.0002$).*

**Conclusions:** *Acute moderate alcohol intake was associated with an increased interatrial EMD obtained by TDI echocardiography. This finding may help explain how these patients express increased susceptibility to atrial fibrillation.* (Cardiol J 2011; 18, 6: 682–686)

**Key words: alcohol, atrial fibrillation, echocardiography**

## Introduction

Moderate alcohol consumption is associated with reduced risk of certain cardiovascular events [1]. On the other hand, ingesting excessive amounts of alcohol within a short period of time ('binge drinking') has been related to an increased risk of myocardial infarction, stroke, and atrial fibrillation (AF) [2–6]. Alcohol causes AF in patients with and without structural heart disease by affecting atrial refractoriness and conduction [4, 7–9]. The increased secretion of cathecholamines, elevated level of plasma free fatty acids, and an indirect effect through the primary metabolites of alcohol are the alternative mechanisms of alcohol-induced arrhythmogenesis [10, 11]. Electromechanical delay (EMD) has been defined as the temporal delay between the detected onset of electrical activity and the realization of force in the myocardium. On the other hand, atrial EMD measured by tissue Doppler imaging (TDI) echocardiography is also associated with AF [12, 13]. We hypothesized that atrial EMD could be affected following acute alcohol consumption in subjects without structural heart disease, and this ef-

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fect may be associated with the development of an AF episode. Therefore, we examined atrial EMD in healthy subjects with acute alcohol intake.

## Methods

### Study population

Thirty healthy male volunteers with no medical problems consented to participate in this observational study. All subjects had normal findings on physical examination and chest radiography. Thyroid function tests and complete metabolic panel were within normal limits. Body mass index (BMI), age, smoking, and alcohol consumption pattern were recorded. All the subjects were very light drinkers (0.1–4.8 g daily). The local ethical committee approved this study.

### Study design

After three days of alcohol abstinence, on the study day the subjects consumed the same standard 1,600 kcal meal with low fat content at 6pm. Two hours after the meal, baseline ECG, echocardiography, heart rate and blood pressure (BP) measurements of the subjects were recorded. All study participants consumed six 12-oz cans of beer (76.8 g of ethanol). The average level of consumed ethanol was 0.95 ± 0.12 g/kg body weight (range 0.80––1.20 g/kg body weight). One hour after drinking, ECG, echocardiography, heart rate, and BP measurements of all subjects were measured again.

### Echocardiography

All examinations (Vivid 3 Pro, GE, Horten, Norway, 2–4 MHz phased array transducer) were performed by a single cardiologist blind to the patient characteristics and clinical data. During echocardiography, one lead ECG was recorded continuously. Measurement of the left ventricle (LV) and left atrium (LA) diameters was performed on M-mode traces recorded from the parasternal long axis view according to established standards [14]. TDI echocardiography was performed with a transducer frequency of 3.5 to 4.0 MHz, adjusting the spectral pulsed Doppler signal filters to obtain the Nyquist limit of 15 to 20 cm/s, and using the minimal optimal gain setting. The monitor sweep speed was set at 50 to 100 mm/s to optimize the spectral display of myocardial velocities. In apical four-chamber view, the pulsed Doppler sample volume was placed at the level of the LV lateral mitral annulus, and subsequently at the septal mitral annulus and right ventricular (RV) tricuspid annulus. The sampling window was positioned as parallel as possible to the myocardial segment of interest to obtain the optimal angle of imaging. EMD was defined as the time interval from the onset of P wave on the surface ECG to the beginning of the late diastolic wave (Am wave). It was measured from the lateral mitral annulus (mitral lateral EMD), septal mitral annulus (septum EMD), and RV tricuspid annulus (tricuspid EMD). All EMD intervals were averaged over three consecutive beats. The difference between the lateral and tricuspid EMD intervals was defined as interatrial EMD, and the difference between the septum and tricuspid AEC intervals was defined as intraatrial EMD [15].

### P-wave measurements on 12-lead ECGs

All standard 12-lead ECGs were obtained simultaneously using a recorder (Hewlett Packard, Pagewriter 300 pi) set at a 50 mm/s paper speed and 2 mV/cm standardization. All recordings were performed in the same quiet room during spontaneous breathing, following 10 minutes of adjustment in the supine position. The ECGs were numbered and presented to the analyzing investigators without name or date information. All measurements of P-wave duration were made blindly by two medically qualified observers. P-wave durations was measured manually in all simultaneously recorded 12 leads of the surface ECG. The mean P-wave duration for at least three complexes were calculated in each lead. The onset of the P wave was defined as the point of first visible upward slope from baseline for positive waveforms, and as the point of first downward slope from baseline for negative waveforms. The return to the baseline was considered the end of the P-wave. The Pmax measured in any of the 12 leads of the surface ECG was used as the longest atrial conduction time. The difference between the Pmax and the Pmin was calculated and defined as Pd [16].

### Statistical analysis

Statistical analysis was carried out with a commercially available statistical package (SPSS for Windows version 10.0; SPSS Inc, Chicago, IL, USA). Data was presented as mean ± standard deviation. Mann-Whitney U test was used for continuous variables and the $\chi^2$ test was used for categorical changes. Relationships between variables were examined with Pearson correlation coefficients. A p-value $< 0.05$ was considered to indicate statistical significance. Reproducibility of EMD obtained by TDI was assessed by coefficient of variation (CV) between measurements. CV is calculated as the standard deviation of the differences between the

**Table 1.** Baseline demographic, laboratory, and echocardiographic parameters.

| | |
|---|---|
| Age (years) | 45 ± 10 |
| Body mass index [kg/m²] | 28.7 ± 2.8 |
| Smoking | 50% |
| Glucose [mg/dL] | 85 ± 8 |
| Total cholesterol [mg/dL] | 180 ± 34 |
| LDL cholesterol [mg/dL] | 100 ± 25 |
| Left atrium diameter [cm] | 3.5 ± 0.3 |
| LV systolic diameter [cm] | 3.4 ± 0.4 |
| LV diastolic diameter [cm] | 5.2 ± 0.5 |
| IVS thickness [cm] | 1.02 ± 0.11 |
| Posterior wall thickness [cm] | 0.90 ± 0.08 |
| Left ventricular ejection fraction (%) | 65.2 ± 5.2 |

LDL — low-density lipoprotein; LV — left ventricle; IVS — interventricular septum

repeated measurements divided by the averages of the repeated measurements and is expressed as a percentage.

## Results

Average age and BMI of the subjects was 45 ± 10 years, and 28.7 ± 2.8% of the study population were current smokers. Two-dimensional echocardiographic measurements were within the normal limits (Table 1).

BP, heart rate, P wave measurements, and atrial EMD parameters at baseline and after the drinking period are set out in Table 2. Although the systolic and diastolic BPs decreased and the heart rates increased after drinking, these differences were not statistically significant. In comparison with the baseline, Pmax and Pd values were significantly prolonged after the drinking period (114.2 ± 10.4 *vs* 100.8 ± 10.6, p = 0.002; 50.6 ± 9.6 *vs* 34.5 ± ± 8.8, p < 0.0001). Although Pmin values decreased after the drinking period, the difference was not statistically significant. Mitral lateral EMD, septum EMD, and tricuspid EMD were significantly increased after the drinking period compared to the baseline (68.2 ± 8.2 *vs* 52.4 ± 7.1 ms, p < 0.0001; 48.4 ± 10.1 *vs* 38.4 ± 5.2 ms, p < < 0.0001; 46.5 ± 8.2 *vs* 37.5 ± 4.5 ms, p < 0.0001). Interatrial EMD was significantly higher after the drinking period compared to the baseline (19.8 ± ± 9.2 *vs* 14.0 ± 5.5 ms, p < 0.0002). Intraatrial EMD increased after the drinking period but the difference was not statistically significant (1.9 ± ± 7.8 *vs* 0.9 ± 3.8, p = NS).

There were no complications or arrhythmia in subjects during the study period. Intraobserver and interobserver reproducibility of the ECG measurements were evaluated. CV averaged 8.1% and 10.4% for Pmax and 7.0% and 9.5% for Pd. The values of CV for intraobserver variability were 5.8% for mitral lateral EMD, 6.8% for septum EMD, and 5.8% for tricuspid EMD.

## Discussion

We found that atrial EMD parameters and interatrial EMD measured by TDI echocardiography were significantly increased in otherwise healthy men after acute moderate alcohol consumption. Secondly, the ECG-derived Pmax and Pd values were also increased after the drinking period com-

**Table 2.** Blood pressure, heart rate, P wave measurements, and atrial conduction parameters at baseline and after drinking period.

| | Baseline | After drinking period | P |
|---|---|---|---|
| Systolic blood pressure [mm Hg] | 123.4 ± 11.5 | 121.6 ± 8.8 | NS |
| Diastolic blood pressure [mm Hg] | 82.2 ± 6.4 | 75.5 ± 7.7 | NS |
| Heart rate [bpm] | 67 ± 6 | 70 ± 5 | NS |
| Pmax [ms] | 100.8 ± 10.6 | 114.2 ± 10.4 | 0.002 |
| Pd [ms] | 34.5 ± 8.8 | 50.6 ± 9.6 | 0.0001 |
| Pmin [ms] | 66.3 ± 5.4 | 63.6 ± 7.4 | NS |
| Mitral lateral EMD [ms] | 52.4 ± 7.1 | 68.2 ± 8.2 | 0.0001 |
| Septum EMD [ms] | 38.4 ± 5.2 | 48.4 ± 10.1 | 0.0001 |
| Tricuspid EMD [ms] | 37.5 ± 4.5 | 46.5 ± 8.2 | 0.0001 |
| Interatrial EMD [ms] | 14 ± 5.5 | 19.8 ± 9.2 | 0.0002 |
| Intraatrial EMD [ms] | 0.9 ± 3.8 | 1.9 ± 7.8 | NS |

Pmax — maximum P wave duration; Pd — P wave dispersion; Pmin — minimum P wave duration; EMD — electromechanical delay

pared to the baseline. These results could help explain the impact of alcohol on the development of AF.

Alcohol reduces myocardial contractility and causes atrial and ventricular arrhythmias. After consuming large quantities of alcohol over several years, alcoholic cardiomyopathy may develop, which has the characteristic of dilation and impaired contractility of the left or both ventricles.

The connection between alcohol intake and AF in healthy individuals has been demonstrated in clinical studies [4]. Even modest alcohol intake can trigger paroxysmal AF in some patients [17–19]. Increased sympathetic system activity, altered conduction and refractory times, vagal reflexes, and myocardial damage have been suggested as potential mechanisms of AF in these individuals [10, 11]. In this study, we found that acute alcohol intake might cause interatrial EMD, which in turn may cause AF.

Koskinen et al. [20] reported that acute intake of alcohol could decrease heart rate variability due to diminished vagal modulation in healthy men. In another study [21], acute alcohol consumption caused a decrease in vagal modulation with a later shift to sympathetic predominance in patients with coronary artery disease. Our results agreed with these studies and suggested that the acute alcohol effect on the atrial impulse conduction might be a mechanism for AF.

In general, after the consumption of one standard drink, the amount of alcohol in the blood (BAC, blood alcohol concentration) peaks within 30–45 min. A standard drink is defined as 12 fluid ounces of beer, 5 fluid ounces of wine, or 1.5 fluid ounces of 80-proof distilled spirit, all of which contain the same amount of alcohol [22]. We recorded the ECG, echocardiographic images, heart rate, and BP measurements of all subjects one hour after the drinking period. A number of factors can influence the absorption process, including the presence and type of food in the gastrointestinal tract when alcohol is consumed [23–25]. Furthermore, women absorb and metabolize alcohol differently than men. They have higher BACs after consuming the same amount of alcohol. Alcohol consumption affects the metabolism of a wide variety of medications, increasing the activity of some, and diminishing the activity, and thereby the effectiveness, of others. Therefore, in our study, all the subjects were men, and they ate the same standard 1,600 kcal meal with low fat content at the same time. None of the study participants was on any type of medication.

## Limitations of the study

The major limitation of our study was the lack of repeat testing one day after the elimination of alcohol from the system. We did not check the BAC in the study participants. All measurements were obtained using blinded manual conventional methods. The manual measurement of P-wave duration in standard 12-lead ECGs is more feasible and reliable when performed on the high-resolution screen of a digital ECG system than with more conventional methods involving paper-printed ECGs [26]. Therefore, manual measurement of P-wave duration performed on standard paper-printed ECGs has limited accuracy.

In this study, we evaluated the acute effects of alcohol in healthy subjects and did not include patients with AF. Since we did not perform continuous Holter recordings, we could not be sure about clinically silent paroxysmal AF episodes. However, we did not observe AF while we were performing the clinical tests, and none of the study participants reported any symptom suggestive of arrhythmia. Therefore, we do not know whether increased interatrial conduction time can predict AF.

For this purpose, a large-scale prospective study is needed to determine whether increased EMD parameters could predict the development of AF after acute alcohol intake. Finally, we evaluated the short-term effects of alcohol ingestion on echocardiographic changes, and we did not follow up these changes.

## Conclusions

Acute moderate alcohol consumption is associated with an increased interatrial EMD measured by TDI echocardiography. These findings may help explain how these patients express increased susceptibility to AF.

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The authors do not report any conflict of interest regarding this work.

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