SCIENTIFIC LETTER

Relations between alcohol consumption, heart rate, and heart rate variability in men

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We have previously reported a strong positive association between average day time and nighttime heart rate measured during 24 hour ambulatory blood pressure monitoring and usual alcohol intake.[1] Acute alcohol consumption has been previously described to reduce ECG indices of vagal activity, and men who are severely dependent on alcohol have been reported to have significantly lower indices of cardiac vagal nerve activity than normal volunteers.[2–5] We performed 24 hour ambulatory ECG monitoring and power spectral analysis of heart rate variability in apparently healthy middle aged men to examine the association between alcohol consumption, heart rate, and indices of heart rate variability.

METHODS

Twenty eight men aged 50 (9) years (range 33–68 years) who were free from clinically apparent alcohol related or cardiovascular diseases and receiving no medication were selected from volunteers responding to an advertisement in a local newspaper. The subjects' blood pressures (128 (12)/75 (9) mm Hg), heart rate (72 (9) beats/min), body weight, and height were measured and their demographic details recorded. A detailed alcohol consumption history was taken by an experienced alcohol and other drugs nurse counsellor (JR). The information obtained included the duration that they had been consuming their current alcohol intake, the days of the week that they usually drank, and the amount of alcohol consumed on each day, their usual weekly alcohol intake (in grams), and their alcohol intake over the preceding week. All had normal left ventricular function and left ventricular mass measured by cine magnetic resonance imaging (MRI). None of the subjects undertook regular exercise.

The subjects were fitted with a Rozinn Holter 3.3 ambulatory ECG recorder (Cardiac Agency, Belmont, NSW, Australia) and a 24 hour ECG recording was performed while they assumed their normal daily activities. At the end of the 24 hour monitoring period the subjects were asked whether they consumed alcohol while the monitor was worn, the time of their last drink, and the amount of alcohol consumed during their last drinking session. They were also asked to estimate the time that they fell asleep, the time that they woke, and whether they had a normal nights' sleep. The ECG data were downloaded and analysed using Rozinn Holter 3.3 software. Indices of heart rate variability were calculated following fast Fourier transformation of the R–R interval data. Ectopic cardiac beats were edited from the data before analysis. Indices of heart rate variability calculated were the standard deviation of successive R–R intervals (SDNN), the standard deviation of the root mean square successive R–R intervals (rMSSD) pNN50, low frequency power, high frequency power, and total power. These indices were calculated for the full 24 hour period, during sleep and during waking hours, and were included in uni- and multivariate regression analyses using alcohol intake as the independent variable.

Total power was measured up to 0.500 Hz and the division between high and low frequency power was 0.150 Hz. Estimates of heart rate variability during sleep were calculated from the data recorded from 10 pm to 6 am, while awake estimates were calculated from the remainder of the day. Statistical analysis was performed using the Statistica 5.5 software package. The study protocol was approved by the South-Eastern Sydney Health Service ethics committee and all subjects provided written informed consent.

RESULTS

The men consumed, on average, 361 (245) g of alcohol per week (median 378 g, range 0–840 g). Most of the subjects drank daily (median seven days per week). The mean period of time from their last drink to when the ambulatory ECG monitor was removed was 18 (10) hours (range 4–42 hours). The amount of alcohol that the subjects consumed during their last drinking session before removal of the ECG monitor was 52 (32) g (range 0–140 g). Six of the subjects were current smokers.

On univariate analysis, alcohol consumption was a significant predictor of 24 hour heart rate ($\beta = 0.510$, $p = 0.006$), SDNN, awake SDNN, sleep SDNN, rMSSD, awake rMSSD, sleep rMSSD, pNN50, sleep pNN50, low power, awake low power, sleep low power, high power, sleep high power, total power, awake total power, and sleep total power. The association between alcohol consumption and SDNN during sleep is presented in fig 1. As body mass index was also found to be positively associated with heart rate and alcohol intake and inversely associated with several measures of heart rate variability, multivariate analysis was performed using alcohol intake and body mass index as independent variables.

On multivariate analysis, alcohol consumption remained an independent predictor of heart rate ($\beta = 0.390$, $p = 0.008$) and of all of the above parameters except for awake rMSSD. In contrast, body mass index no longer remained a significant predictor of any of the ECG parameters. When low and high power

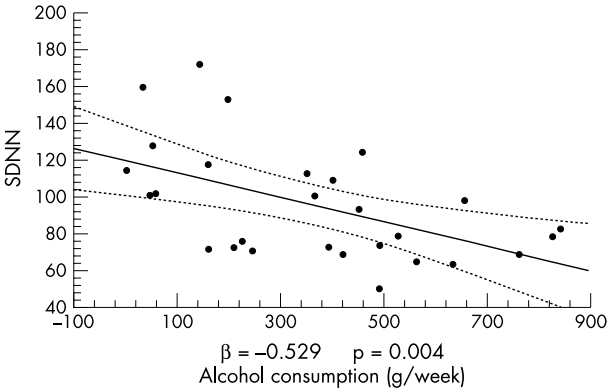

Figure 1 Relation between usual alcohol consumption (g/week) and standard deviation of successive R–R intervals (SDNN) measured following power spectral analysis of 24 hour ambulatory ECG monitoring results. One unit of alcohol (standard drinks) = 10 g of alcohol.

were corrected for total power (low/total power and high/total power) associations with alcohol consumption were no longer apparent except for high/total power during sleep which was strongly and positively associated with alcohol intake.

Measures of heart rate variability were not related to time since the last drink but were significantly related to the amount of alcohol consumed during the last drinking session. However, the amount of alcohol consumed during the last drinking session was highly correlated with the subjects' usual weekly alcohol intake.

DISCUSSION

This study principally differs from previous reports of associations between alcohol intake and heart rate variability[2–7] in that the relations between alcohol intake and measures of heart rate variability following correction for changes in total power have also been assessed.

The results suggest that the previously reported universe associations between alcohol intake and heart rate variability, which have suggested that alcohol consumption is associated with reduced vagal activity, may be mainly due to a positive association between alcohol intake and heart rate.[8] The reason for a positive association between alcohol intake and heart rate is unclear but possibilities include an increase in sympathetic activity secondary to vasodilation or increased calcium entering into cardiac myocytes.[9]

In conclusion, the results of the present study suggest that the association between alcohol intake and decreased heart rate variability may be mainly secondary to an increase in heart rate rather than a central or peripheral effect of alcohol on cardiac vagal nerve activity.

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