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Acute Consumption of Alcohol and Discrete Atrial Fibrillation Events

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Background: Patients' self-reports suggest that acute alcohol consumption may trigger a discrete atrial fibrillation (AF) event.

Objective: To objectively ascertain whether alcohol consumption heightens risk for an AF episode.

Design: A prospective, case-crossover analysis.

Setting: Ambulatory persons in their natural environments.

Participants: Consenting patients with paroxysmal AF.

Measurements: Participants were fitted with a continuous electrocardiogram (ECG) monitor and an ankle-worn transdermal ethanol sensor for 4 weeks. Real-time documentation of each alcoholic drink consumed was self-recorded using a button on the ECG recording device. Fingerstick blood tests for phosphatidylethanol (PEth) were used to corroborate ascertainments of drinking events.

Results: Of 100 participants (mean age, 64 years [SD, 15]; 79% male; 85% White), 56 had at least 1 episode of AF. Results of PEth testing correlated with the number of real-time recorded drinks and with events detected by the transdermal alcohol sensor. An AF episode was associated with 2-fold higher odds of 1 alcoholic drink (odds ratio [OR], 2.02 [95% CI, 1.38 to 3.17]) and greater than 3-fold higher odds of at least 2 drinks (OR, 3.58 [CI, 1.63 to 7.89]) in the preceding 4 hours. Episodes of AF were also associated with higher odds of peak blood alcohol concentration (OR, 1.38 [CI, 1.04 to 1.83] per 0.1% increase in blood alcohol concentration) and the total area under the curve of alcohol exposure (OR, 1.14 [CI, 1.06 to 1.22] per 4.7% increase in alcohol exposure) inferred from the transdermal ethanol sensor in the preceding 12 hours.

Limitation: Confounding by other time-varying exposures that may accompany alcohol consumption cannot be excluded, and the findings from the current study of patients with AF consuming alcohol may not apply to the general population.

Conclusion: Individual AF episodes were associated with higher odds of recent alcohol consumption, providing objective evidence that a modifiable behavior may influence the probability that a discrete AF event will occur.

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Atrial fibrillation (AF) is the most common arrhythmia encountered in clinical practice and is expected to affect more than 12 million persons in the United States by 2050 (1). Although research has focused on risk factors for the disease and therapies to treat it, patients with AF themselves are especially concerned with the acute triggers responsible for a discrete AF episode (2). Large epidemiologic studies have demonstrated that greater amounts of long-term alcohol consumption predict incident AF (3), and a recent randomized trial showed that alcohol abstinence over several months can reduce AF burden (4). We and others have shown that long-term alcohol consumption is associated with adverse atrial remodeling, potentially establishing an atrial substrate that is rendered more prone to fibrillate (5, 6). However, patients report alcohol as an acute trigger (2, 7), and the relationship between alcohol and AF initially arose from observations that discrete AF episodes seemed to be associated with recent heavy alcohol consumption (2, 7). However, because alcohol is so commonly consumed (8), objective determination of whether a given drinking event meaningfully enhances the risk for a particular AF event is difficult. In addition, if such a temporal relationship were present, the timing of risk after alcohol is ingested and the amounts of alcohol that may be relevant remain unknown.

We therefore sought to test the hypothesis that acute consumption of alcohol (drinking within a few hours before the episode) is independently associated with increased risk for a discrete AF episode.

Methods

To do a case-crossover analysis, we enrolled 100 consecutive patients aged at least 21 years with documented paroxysmal AF who consumed on average at least 1 standard alcoholic drink per month. Patients were recruited from outpatient clinics specializing in general cardiology or cardiac electrophysiology at the University of California, San Francisco. Medical records were initially screened to identify persons with documented AF, and only those without prior documentation of substance use disorder (alcohol or other) or clear alcohol abstinence were approached. We excluded those with a

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known history of alcohol or substance use disorder and those who exhibited evidence of severe alcohol use disorder by scoring greater than 19 on the Alcohol Use Disorders Identification Test consumption questions (9). In addition, we excluded patients undergoing planned changes in management of their AF, such as changes in antiarrhythmic drugs or catheter ablation, during the study period (a stable dose of an antiarrhythmic drug was allowed given documented AF while receiving that drug). We also excluded those who had a latex allergy, had a previous allergic or hypersensitivity reaction to electrocardiogram (ECG) recording adhesives, or were unwilling to wear an alcohol sensor.

Medical history, medications, and habits were determined using medical record reviews and patient interviews. The first 27 participants were fitted with a LifeWatch ACT (ambulatory cardiac telemetry) monitor (LifeWatch), with a goal wear time of 4 weeks, and the remainder received 2 successive Zio patches (iRhythm) worn for 2 weeks each. These devices continuously record single-lead ECGs and use algorithms routinely used in clinical care to detect episodes of AF with high accuracy (10). The time and duration of every AF episode longer than 30 seconds were determined. Recurrent AF episodes were considered separate regardless of the duration of non-AF rhythms between them. The ECG monitor wear time constituted each participant's individual study time of interest.

Participants were instructed to press a patient activator button on the ECG monitor only when and every time they had a standard alcoholic drink, which they were told would equal a standard glass of wine (the amount expected to receive when poured a glass of wine at a restaurant), a 12-ounce bottle or can of beer, or a shot of spirits; pressing the button then time-stamped every standard drink consumed. We refer to these as real-time, self-reported drinking events. Participants were also fitted with a transdermal alcohol sensor placed around the ankle for passive alcohol monitoring: the SCRAM (Secure Continuous Remote Alcohol Monitor) (SCRAM Systems). The SCRAM reliably detects alcohol consumption above a level of 2 to 3 drinks per occasion (11) but can also detect lower amounts of alcohol (12). Although patients could not swim or bathe with the SCRAM device, they were allowed to shower in concordance with device specifications. A "UCSF Cardiology" sticker was prominently displayed on the SCRAM ankle device to mitigate perceived stigma (13) of such a device by some participants.

Participants returned for in-person visits 2 and 4 weeks into the study to ensure adherence to devices and place new devices (such as the second Zio patch) if needed. Staff actively inquired about the previous 2 weeks to provide participants the opportunity to communicate any real-time, self-reported drinking events (button presses on the ECG monitor) that may have occurred during activities other than consuming a standard drink of alcohol (none were ultimately reported). During those visits, a fingerstick blood spot (United States Drug Testing Laboratories) was collected to test for phosphatidylethanol (PEth), an abnormal phospholipid formed in the blood only in the presence of alcohol use. It is detectable for 2 to 4 weeks after repeated, high-risk consumption of alcohol (defined as >60 g/day for men and >40 g/day for women) and has a half-life of 4 to 10 days (14, 15). Although test sensitivity increases with increased alcohol consumption, PEth is detectable at lower levels of alcohol use, including after a single drink (16). The most common homolog, 16:0/18:1, was measured. Exceeding the limit of detection (≥8 ng/mL) was considered a positive result (14).

The study was approved by the University of California Institutional Review Board, and all participants provided informed, written, and witnessed consent.

Statistical Analysis

Variables were summarized using means and SDs, medians and interquartile ranges (IQRs), or percentages, and they were compared using *t* tests, Wilcoxon rank-sum tests, and χ^2 or Fisher exact tests, as appropriate. The associations between detectable PEth in each 2-week period and the number of alcoholic drinks consumed over this period, as ascertained using real-time recording, were assessed using a logistic model including a fixed effect for 2-week period and using robust SEs to account for within-individual clustering. In a case-crossover analysis (17–19) restricted to participants with at least 1 AF episode, the association between having an AF episode and the number of recent alcoholic drinks ascertained by either real-time recording or the transdermal sensor was assessed using conditional logistic models. After dividing each day into twenty-four 1-hour periods, we included the first with AF on any calendar day as a case period. After excluding all subsequent periods on a calendar day with AF (including any with AF) and the 12 preceding periods, potentially including some on the previous calendar day, we then used all remaining 1-hour periods as controls. A sensitivity analysis that included the 12 preceding 1-hour periods as controls was also done. Only the first AF episode in a given calendar day was examined as a case. Lookback for ascertaining drinks ended at the time of the AF episode in case periods and at a randomly selected minute within the hour in control periods; lookback durations of up to 12 hours were examined. All case and control periods for each participant were pooled to form a single matched set. To control confounding by timing, day of week and time of day (grouped in 4-hour blocks) were included as fixed effects in the model. Spearman correlation coefficients were calculated to corroborate different methods of alcohol consumption ascertainment. We used the Transdermal Alcohol Sensor Data Macro (20) to process the alcohol sensor data to estimate areas under the curve of the alcohol levels for each drinking occasion. Because of the relative infrequency of transdermal alcohol ascertainment and the expected delay between alcohol consumption and detection in perspiration by the sensor, the lookback period for transdermal sensor events was set to 12 hours. To assess for heterogeneity in the relationship between real-time, self-reported drinking events and AF, we calculated the intraclass correlation coefficient in a random-effects logistic model.

Two-tailed *P* values were considered statistically significant. Analyses were done using Stata, version 16 (StataCorp). The statistical code is available at https://github.com/evittinghoff/Marcus_alcohol_monitors for interested readers.

Role of the Funding Source

The National Institute on Alcohol Abuse and Alcoholism provided funding for this study. The funding source had no role in the study design; the collection, analysis, or interpretation of the data; or the decision to approve publication of the finished manuscript.

RESULTS

Participants wore the ECG monitor for a median of 27 days (IQR, 15 to 28 days), and 90% wore it for more than 21 days (**Supplement Table 1**, available at Annals.org). **Table 1** shows the baseline characteristics of participants who did and did not have an AF episode during the study. During the monitoring period, real-time recordings of alcohol consumption showed a median of 19 drinks (IQR, 10 to 38 drinks) on a median 12 different days (IQR, 7 to 21 days). Participants drank a median of 1 drink (IQR, 1 to 2 drinks) per day throughout the study period. Atrial fibrillation occurred on a median of 5 different days (IQR, 2.5 to 12.5 days). **Supplement Tables 2** to **10** (available at Annals.org) show the distribution of AF episodes; real-time, self-reported drinking events; and SCRAM-detected events by time of day versus day of week, as well as by time of day alone and day of week alone.

Of 133 PEth tests, 95 (71%) had positive results; the median measurement was 37 ng/mL (IQR, 19 to 81 ng/mL; total range, 8 to 312 ng/mL). A positive PEth result correlated with both the number of real-time recorded drinking events and the area under the curve of the transdermal sensor–detected events (**Supplement Tables 11** and **12**, available at Annals.org). **Figure 1** shows box plots of relationships between PEth and both real-time, self-reported drinking events and sensor-detected episodes. Adjusting for study week and taking repeated measurements within individuals into account, we found that every additional drinking event based on participant activation was associated with 23% greater odds of a positive PEth result (odds ratio [OR], 1.23 [95% CI, 1.09 to 1.39]; $P < 0.001$) and that each additional unit of the area under the curve of alcohol consumption detected by the transdermal sensor was associated with 6% greater odds of a positive PEth result (OR, 1.06 [CI, 1.00 to 1.11]; $P = 0.032$). The Spearman correlation between real-time recordings of alcohol consumption and daily areas under the curve for SCRAM-detected events was 0.52 ($P < 0.001$). The distribution of real-time, self-reported drinking events is shown in **Supplement Table 13** (available at Annals.org).

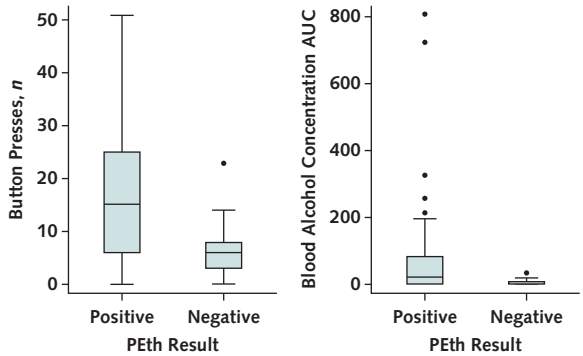
An AF episode was associated with 2-fold greater odds of 1 alcoholic drink and more than 3-fold greater odds of 2 or more drinks within the preceding 4 hours (**Table 2**; **Supplement Figure**, available at Annals.org). These relationships remained consistent when we examined the preceding

Table 1. Participant Characteristics Among Those With and Without at Least 1 AF Episode During the Monitoring Period

Characteristic	No AF Episode (*n* = 44)	AF Episode (*n* = 56)
Mean age (SD), *y*	64.8 (11.6)	63.8 (16.9)
Male sex, *n (%)*	32 (72.7)	46 (82.1)
Race/ethnicity, *n (%)*		
White	37 (84.1)	48 (85.7)
Asian	4 (9.1)	5 (8.9)
Black	2 (4.5)	1 (1.8)
Latinx	0 (0)	1 (1.8)
Other	1 (2.3)	2 (3.6)
Hypertension, *n (%)*	25 (56.8)	23 (41.1)
Diabetes, *n (%)*	5 (11.4)	6 (10.7)
Coronary artery disease, *n (%)*	10 (22.7)	6 (10.7)
Congestive heart failure, *n (%)*	3 (6.8)	3 (5.4)
Smoking, *n (%)*		
Never	20 (45.5)	30 (53.6)
Former	23 (52.3)	24 (42.9)
Current	1 (2.3)	1 (1.8)
Antiarrhythmic medications, *n (%)*		
Amiodarone	1 (2.3)	0 (0.0)
Dronedarone	1 (2.3)	0 (0.0)
Propafenone	2 (4.5)	5 (8.9)
Flecainide	12 (27.3)	5 (8.9)
Sotalol	0 (0.0)	1 (1.8)
Dofetilide	4 (9.1)	8 (14.3)
Median alcoholic drinks (IQR), *n**	18 (7.0–33.0)	20 (11.0–41.0)
Median time ECG monitor worn (IQR), *d*	28.5 (15.0–30.0)	29.0 (23.0–30.0)
Median AF episodes (IQR), *n*	0.0 (0.0–0.0)	15.0 (5.0–64.0)
Any real-time, self-reported drinking event, *n (%)*	43 (97.7)	55 (98.2)
Any transdermal alcohol event, *n (%)*	21 (47.7)	38 (67.9)
Positive PEth result, *n (%)*	19 (67.9)	31 (68.9)

AF = atrial fibrillation; ECG = electrocardiogram; IQR = interquartile range; PEth = phosphatidylethanol.
* Determined by real-time recording (button presses on the continuous ECG monitor).

Figure 1. PEth results compared with measures of alcohol consumption during the preceding 2 weeks.



Box-and-whisker plots illustrating the 25th percentile (bottom of the boxes), 75th percentile (top of the boxes), and median (line in the middle of the boxes); the error bars, or "whiskers," represent the greatest value not extending beyond 1.5 times the interquartile range, and the circles represent outlier values. The left panel illustrates relationships with button presses (real-time, self-reported drinking events), and the right panel illustrates relationships with the median AUC of transdermal alcohol sensor results. AUC = area under the curve; PEth = phosphatidylethanol.

6 and 8 hours (Table 2). In the sensitivity analyses that included the twelve 1-hour periods before AF episodes as controls, the estimates were somewhat attenuated but remained of similar general magnitude and statistically significant (Supplement Table 14, available at Annals.org). Examining discrete 2-hour intervals before an AF event produced the highest odds of alcohol consumption 3 to 4 hours prior, with a gradual reduction in the magnitude of the point estimates thereafter and no statistically significant association 11 to 12 hours prior (Figure 2).

Episodes of AF were also associated with increased blood alcohol concentration measured using the transdermal alcohol sensor during the previous 12 hours: The odds of AF were 38% greater per 0.1% increase in peak blood alcohol concentration in the past 12 hours (OR, 1.38 [CI, 1.04 to 1.83]; $P = 0.024$). Episodes of AF were also associated with the total area under the curve of alcohol exposure in the past 12 hours (OR, 1.14 [CI, 1.06 to 1.22] per 4.7% increase in total alcohol exposure; $P < 0.001$).

The intraclass correlation coefficient between real-time, self-reported drinking events and discrete AF episodes was 0.44, suggesting moderate heterogeneity in that relationship among all participants who had any AF. No apparent threshold effects existed between the amount of alcohol consumed and risk for a discrete AF event (Supplement Table 15, available at Annals.org).

DISCUSSION

We showed that alcohol consumption substantially increased the chance of a discrete AF episode within a few hours. The relationship seemed to be fairly linear—the more alcohol consumed, the higher the risk for an acute AF event—without clear evidence of a threshold effect. These observations mirror what has been reported by patients with AF for decades, but this is the first objective, measurable evidence of these phenomena. Indeed, to our knowledge, this is the first objective evidence that a modifiable exposure may influence the chance that an AF episode will occur in the near term.

Previous studies have demonstrated a relationship between long-term alcohol consumption and the development of an AF diagnosis. Although some prominent investigations failed to identify statistically significant relationships (21, 22), the literature as a whole has favored a positive association (3). A limitation to most of this work has been the reliance on self-report to describe general patterns of alcohol consumption; however, we used alcohol access laws as an instrumental variable (23) and health care provider coding of alcohol abuse (24) to confirm higher risks for an AF diagnosis with greater alcohol exposure. Given evidence from observational data that alcohol abstinence was associated with lower AF risk (25), a recent prospective randomized trial showed that instructions to abstain from alcohol could reduce the burden of AF (4).

The mechanism by which alcohol ingestion might lead to AF remains largely unknown. Extrapolating from the observations that long-term alcohol consumption can lead to a ventricular cardiomyopathy in some patients and that the atria are more prone to fibrosis than the ventricles (26), invasive cardiac electrophysiology studies have demonstrated that persons who consume more alcohol exhibit lower atrial voltages (a marker of more fibrosis) (6, 27). Similarly, using serial echocardiograms in the Framingham Heart Study, we showed that the long-term relationship between alcohol consumption and AF was at least in part mediated by a larger left atrium (5). A limitation of these studies evaluating long-term, alcohol-related effects is that AF itself is known to lead to adverse atrial remodeling (26), and short-term effects of alcohol on occult AF could not be excluded.

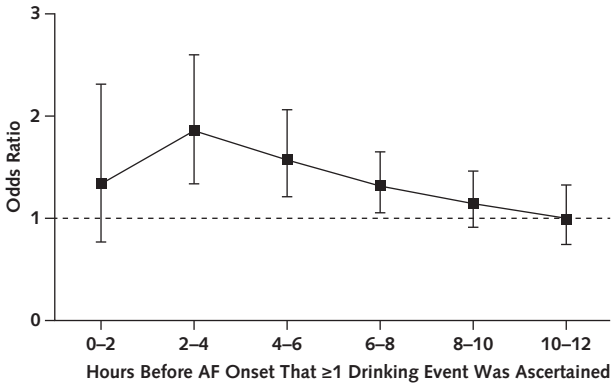
Despite their focus on long-term alcohol consumption and initial development of AF, the early studies that indicated an alcohol–AF relationship described patients

Table 2. Odds of a Real-Time, Self-reported Drinking Event Before an AF Event

Real-Time, Self-Reported Drinking Events	Odds Ratio (95% CI)			
	4 h Before an AF Episode	6 h Before an AF Episode	8 h Before an AF Episode	12 h Before an AF Episode
Any	2.26 (1.50–3.40)	2.51 (1.84–3.42)	2.34 (1.78–3.06)	1.81 (1.73–2.29)
1	2.02 (1.38–3.17)	2.34 (1.66–3.31)	2.05 (1.50–2.78)	1.73 (1.31–2.29)
≥2	3.58 (1.63–7.89)	3.05 (1.83–5.10)	3.13 (2.11–4.66)	1.98 (1.40–2.81)

AF = atrial fibrillation.

Figure 2. Odds of any real-time, self-reported drinking event restricted to 2-hour increments before an AF episode.



Error bars denote 95% CIs. AF = atrial fibrillation.

presenting with acute AF shortly after imbibing large amounts of alcohol (28, 29). Such observations were, by definition, case series of anecdotes with no readily available comparators. Given the ubiquity of alcohol consumption (8) combined with the most common arrhythmia (AF), such observations are prone to an availability heuristic (30), where recent exposures might naturally (and yet potentially erroneously) be inferred to be causal, leading to an illusory correlation (31). We previously attempted to combat this by comparing alcohol as a trigger in AF versus other supraventricular tachycardias (7) and by showing that alcohol was the most commonly described trigger of AF among a list of many others (2), but such investigations remained reliant on patient self-report.

In the current study, patient activation by pressing a button on the ECG monitoring device to indicate drinking events could not be prone to recall bias because it occurred in real time, without knowledge of the future AF event. Indeed, real-time assessments of alcohol consumption have been shown to be more reliable than conventional approaches relying on reports at a baseline or follow-up study visit (32). We acknowledge that some participants may have forgotten to press the button or may have minimized the number of button presses because of embarrassment or shame. Recognizing that this approach might not be reliably accurate, we corroborated the number of such patient activation events via comparison with PEth, an established marker of alcohol consumption in the past 21 days (33), and showed a correlation with SCRAM events. Separately and independently of any action on the part of the participant, alcohol drinking events identified by the transdermal sensor also presaged discrete AF events.

Atrial fibrillation is associated with substantial health care use and reductions in quality of life (34, 35). These data suggest that reducing or avoiding alcohol consumption may help mitigate these adverse consequences. These findings also may seem counter to a prevailing perception that alcohol is "cardioprotective" (36). However, the protective effects of moderate alcohol consumption are predominately in the context of coronary artery disease and myocardial infarction (37), even within the same cohorts where, over the same period of time and using the same methods, alcohol has been shown to heighten risk for AF (23). The current study expands on the established literature pointing to alcohol as a risk factor for the initial diagnosis of AF and shows that acute ingestion of alcohol seems to substantially enhance the risk that a particular episode of AF will soon occur. More optimistically, these data suggest that curbing alcohol consumption may have immediately beneficial effects in the prevention of AF.

We could not identify a clear and consistent relationship between the timing of alcohol consumption and AF episodes, although most alcohol–AF associated pairs occurred within a few hours of each other. This may reflect variability in alcohol metabolism. Of note, the effect did not generally seem to be immediate, favoring a consequence of alcohol or its metabolites rather than, for example, some instantaneous phenomenon related to esophageal exposure. The variable timing may also suggest that alcohol primarily heightens the *probability* that an AF event will occur; some other as yet unidentified factor, such as the timing of a premature atrial beat or a particular change in autonomic tone, may finally initiate AF within a vulnerable window of time. These findings fit with a recent, double-blind, randomized trial of intravenous ethanol versus masked placebo in patients having catheter ablation procedures for AF (38): Ethanol was associated with a significant reduction in atrial effective refractory periods in the pulmonary veins, a phenomenon expected to render the atria more prone to fibrillate, providing mechanistic evidence to support the findings of the current study. In addition, the immediate assessment during that randomized trial found no differences in AF episodes, consistent with the current observation that AF events occur several hours after alcohol exposure. We were similarly not able to identify a clear threshold of alcohol concentration that mattered, but rather the relationships seemed to be fairly linear. Contrary to a common belief that heavy alcohol consumption is required to influence AF, it seems that even 1 alcoholic drink may be enough to increase the risk for a discrete AF event. Of note, most participants did not show particularly excessive alcohol use according to PEth levels previously associated with adverse events (39, 40), suggesting that the potency of an alcohol–AF relationship may be even more evident among heavier drinkers.

Both unique strengths and limitations of the case-crossover design should be acknowledged. Individual participants contributed both case and control data, minimizing confounding related to interindividual differences. Such a study examining real-time effects is vulnerable to confounding related to exposures that are reliably contemporaneous with alcohol consumption, such as those that might occur during socializing occasions. Because no other behavior has been objectively shown to enhance the risk for a discrete AF event, such possible confounders are based on speculation. Smoking can frequently accompany alcohol consumption, and although smoking has not been shown to increase short-term risk for an AF event, those who smoke have a heightened risk for the disease in general (41). However, only 1 participant with AF reported smoking. Despite a common belief that caffeine may increase risk for arrhythmias, recent evidence suggests that long-term consumption of

caffeine is associated with *lower* risk for AF (42), and it seems unlikely that simultaneous consumption of caffeine and alcohol would be common. Sleep disruption, even independent of obstructive sleep apnea, has recently emerged as a risk factor for AF (43), and patients report lack of sleep as a trigger for discrete episodes (2). Because alcohol may contribute to insomnia (44), poor sleep might serve as a mediator in the observed relationship, but in that case it would be part of a mechanistic explanation along the causal pathway rather than a true confounder.

Other more general limitations should be recognized. The study was limited to persons with paroxysmal AF. Although this enabled a feasible assessment to document a proof of concept, the risk for a discrete AF event among the general population consuming similar amounts of alcohol cannot be directly extrapolated from these data. Risk for an acute AF event in the setting of a drinking episode likely has multifactorial determinants, including the amount of alcohol and the individual's underlying propensity to AF. Indeed, the intraclass correlation coefficient suggests heterogeneity in the AF response to alcohol. Similarly, alcohol use is clearly not necessary for AF to occur, and the current study did not elucidate other factors that influence the probability that a particular AF paroxysm will happen. Of note, some patients may be especially prone to alcohol-induced AF and may have ceased consuming alcohol, rendering them ineligible for the current study–therefore, at least for some patients, our observed effects may have underestimated the strength of the association. Finally, because exact measurements of alcohol consumed were not done, the numbers of drinks recorded as real-time, self-reported drinking events should be interpreted as rough estimates rather than well-validated, specific quantities. Indeed, in the sensitivity analyses that included twelve 1-hour periods before AF episodes as controls, the effect sizes were somewhat attenuated. These results ultimately suggest the proof of concept that acute alcohol consumption is associated with near-term AF events, but these data should not be used to extrapolate precise amounts of alcohol related to that observation.

In conclusion, alcohol consumption, documented objectively and in real time, substantially increased the risk for an AF episode within a few hours. These data show that the occurrence of AF may be neither random nor completely unpredictable, but rather that identifiable, common, and even imminently modifiable exposures are associated with discrete AF events.

From University of California, San Francisco, San Francisco, California (G.M.M., E.V., S.J., V.Y., G.N., E.P.G., J.D.M., R.J.L., B.K.L., Z.H.T., V.V., J.E.O., M.M.S., H.H., R.G., S.F., E.L., C.F., K.O., R.F., J.A.H.); and Lewis Katz School of Medicine at Temple University, Philadelphia, Pennsylvania (I.R.W.).

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Reproducible Research Statement: *Study protocol:* Available from Dr. Marcus (e-mail, greg.marcus@ucsf.edu). *Statistical code:* Available at https://github.com/evittinghoff/Marcus_alcohol_monitors. *Data set:* Not available.

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