Published in final edited form as:

Curr Treat Options Cardiovasc Med. 2012 October ; 14(5): 529–535. doi:10.1007/s11936-012-0193-6.

Recommendations Regarding Dietary Intake and Caffeine and Alcohol Consumption in Patients With Cardiac Arrhythmias: What Do You Tell Your Patients To Do or Not To Do?

Kathryn A. Glatter, MD^{1,*}, Richard Myers, BS², and Nipavan Chiamvimonvat, MD^{2,3}

¹ Woodland Clinic Medical Group, 632 West Gibson Road, Woodland, CA 95695, USA

²Division of Cardiovascular Medicine, University of California, Davis, One Shields Avenue, GBSF 6315, Davis, CA 95616, USA

³Department of Veteran's Affairs, Northern California Health Care System, Mather, CA, USA

Abstract

Opinion statement—The etiology of arrhythmias including atrial fibrillation is multifactorial. Most arrhythmias are associated with comorbid illnesses like hypertension, diabetes, thyroid disease, or advanced age. Although it is tempting to blame a stimulant like caffeine as a trigger for arrhythmias, the literature does not support this idea. There is no real benefit to having patients with arrhythmias limit their caffeine intake. Caffeine is a vasoactive substance that also may promote the release of norepinephrine and epinephrine. However, acute ingestion of caffeine (as coffee or tea) does not cause atrial fibrillation. Even patients suffering a myocardial infarction do not have an increased incidence of ventricular or other arrhythmias after ingesting several cups of coffee. Large epidemiologic studies have also failed to find a connection between the amount of coffee/caffeine used and the development of arrhythmias. As such, it does not make sense to suggest that patients with palpitations, paroxysmal atrial fibrillation, or supraventricular tachycardia, abstain from caffeine use. Energy drinks are a new phenomenon on the beverage market, with 30-50 % of young adults and teens using them regularly. Energy drinks are loaded with caffeine, sugar, and other chemicals that can stimulate the cardiac system. There is an increasing body of mainly anecdotal case reports describing arrhythmias or even sudden death triggered by exercise plus using energy drinks. Clearly, there must be more study in this area, but it is wise to either limit or avoid their use in patients with arrhythmias. Moderate to heavy alcohol use seems to be associated with the development of atrial fibrillation. The term “holiday heart” was coined back in 1978, to describe patients who had atrial fibrillation following binge alcohol use. Thus, it is reasonable to recommend to patients with arrhythmias that they limit their alcohol use, although unfortunately this treatment will likely not completely resolve their arrhythmia.

Keywords

Arrhythmias; Energy drink; Atrial fibrillation; Alcohol; Caffeine; Sudden death

Disclosures

No potential conflicts of interest relevant to this article were reported.

Introduction

Treatment recommendations regarding caffeine intake in patients with cardiac arrhythmias

“Every time I drink an espresso, my heart just races and races!” lamented our forty-year old, otherwise healthy female patient. Her Holter monitor revealed paroxysmal atrial fibrillation. Ironically, she was in sinus rhythm on the monitor while drinking coffee, although she noted “heart palpitations” on the record at that time.

Should we restrict our patient’s caffeine intake? Would cutting out coffee help eliminate any arrhythmias?

Background on caffeine

Americans (and much of the world) have a love affair with coffee, drinking an average of three cups of coffee per person daily [1•, 2]. Coffee contains many different chemical substances. However, its main cardiovascular effects derive from caffeine. The caffeine content ranges from 40 to 180 mg per 150 ml of coffee, or 90 mg for a cup of espresso coffee [3]. Caffeine is the most widely consumed vasoactive substance in the world.

Chemical effects of caffeine

Caffeine is a methylxanthine compound related to theophylline, which has a number of interesting actions on the heart [4, 5]. Caffeine’s primary action is to stimulate the sympathetic nervous system through different mechanisms. It antagonizes adenosine receptors, which participate in cardiovascular regulation [4, 6, 7]. It increases cytosolic calcium concentrations by blocking the reuptake of calcium into the sarcoplasmic reticulum. Additionally, it inhibits phosphodiesterase, which gives caffeine its sympathomimetic effects.

However, as will be seen in the following literature review, except in rare cases, caffeine does not seem to promote arrhythmias in most patients. Serious cardiovascular effects seem to only occur in vivo at very high doses, much higher than what is normally ingested by humans. Finally, its various vasoactive actions may be counterbalanced and negated by its other effects in the body.

Clinical studies of caffeine and arrhythmias

Much of our information whether caffeine can provoke arrhythmias comes from the older clinical research, performing research studies that would probably not be permitted in today’s rigorous scientific arena.

In an elegant study, Dobmeyer et al. gave either an oral coffee or intravenous caffeine load to 19 patients who underwent an electrophysiology (EP) study before and after the caffeine dose [7]. The dose approximated two cups of coffee. The study participants were regular consumers of caffeine who normally drank an average of three to five cups of coffee per day. Caffeine shortened the refractory period of the right atrium, AV node, and the right ventricle, and prolonged the refractory period of the left atrium. The study did show that caffeine increased the serum concentrations of both epinephrine and norepinephrine, which could theoretically promote arrhythmogenesis [8, 9]. However, on balance, the caffeine load did not seem to induce arrhythmias in most patients, despite affecting the refractory periods in the heart.

Researchers in Oregon gave 22 habitual coffee drinkers with malignant ventricular arrhythmias an oral 275 mg caffeine load, similar to that found in two to three cups of coffee [10]. The authors found no change in the ventricular refractory period during EP study after

ingestion of the caffeine. There was no increase in the inducibility or even severity of ventricular arrhythmias seen, which was surprising since one might infer that coffee would provoke premature ventricular complexes (PVCs) in this relatively high-risk patient population.

In another fascinating study, Graboys et al. took 50 patients who had been hospitalized with ventricular arrhythmias [11]. They underwent a stress test and a Holter monitor before and after a 200 mg (two-cup of coffee) caffeine load. There was no increase in the PVC load after the caffeine ingestion, although again serum epinephrine and norepinephrine levels increased.

Other researchers have confirmed caffeine's lack of apparent effect upon measurable electrophysiologic parameters. Newcombe et al. took 34 healthy adults and performed a 12-lead EKG and 24-hour Holter monitor before and after acute ingestion of an average of seven cups of coffee [12]. They found no increase in arrhythmias, heart rate, or even occasional skipped heartbeats after such a huge caffeine load. Sutherland et al. gave an acute oral caffeine load of 1 mg/kg of body weight and performed 12-lead EKG and Holter-monitoring [13]. There was again no difference in heart rate and QT intervals post-caffeine. However, in 14 of 18 patients who had frequent PVCs at baseline, caffeine increased the amount of PVCs slightly, although it varied greatly among the individuals.

In a similar vein, Ammar et al. gave 400 mg of caffeine (equivalent to five or more cups of coffee) to ten healthy volunteers and found no acute changes in the PR or QT intervals on 12-lead EKG [14]. The heart rate also did not change following coffee ingestion. Interestingly, the systolic blood pressure did increase an average of 10 points (a significant amount) for several hours, although this is admittedly a huge caffeine bolus that most patients would not normally imbibe. As with so many of these studies in the literature, it is difficult to translate their findings to something clinically relevant for our patients.

Finally, one might speculate that caffeine could promote arrhythmias during a myocardial infarction (MI), since coffee promotes a surge in epinephrine and norepinephrine levels [15•]. One provocative study evaluated this question. The authors randomized 103 patients with an acute, ST-elevation MI to receive caffeinated versus decaffeinated coffee and observed its effect on heart rate variability (HRV) 5 days post-MI. Incredibly, the caffeine improved the autonomic nervous system function and increased the parasympathetic activity as measured by HRV. Normally, the finding of depressed HRV post-MI is associated with an increased long-term mortality. Coffee ingestion did not seem to promote arrhythmias post-MI, a finding replicated in other clinical trials.

Epidemiological studies of caffeine and arrhythmias

Given that caffeine stimulates the sympathetic nervous system, one might speculate that it could trigger arrhythmias or even sudden death in an electrically vulnerable period, such as immediately post-myocardial infarction. Although studies in individuals have failed to demonstrate these findings, perhaps larger-scale epidemiological trials might yield the data.

Investigators looked at the 11,323 patients post-MI in the GISSI-prevention trial, an Italian study originally designed to test the efficacy of omega-3 fatty acids and vitamin E on mortality after a heart attack [16]. As with so many of these studies, the authors relied on a simple dietary questionnaire where patients reported on their retrospective coffee use. An inverse relationship was found between coffee consumption and the risk of coronary heart disease events, stroke, and sudden death. This risk was highest for those patients drinking fewer than two cups per day and lowest for those drinking over four cups per day. However,

these findings did not reach statistical significance, which is important as this was a large study.

In the Women's Health Initiative observational study, the authors looked at the risk of sudden death and caffeine intake for 33,638 postmenopausal women [17•]. The data were again obtained by completing a baseline questionnaire on their coffee ingestion. As with the GISSI trial, the authors found that regular coffee and total caffeine use was not associated with an increased risk of sudden cardiac death. Regular tea intake and drinking decaffeinated coffee were also not associated with this risk.

Other epidemiological studies have provided conflicting data regarding if caffeine raises the mortality rate after a myocardial infarction. In the Stockholm Heart Epidemiology Program (SHEEP), 1,369 patients after an MI between 1993 and 1994 in Sweden were identified in this population-based cohort study [18•]. Subjects self-reported on their coffee intake in the year immediately prior to the MI. The authors found an inverse association with coffee intake and subsequent post-MI mortality. Those subjects who drank over seven cups of coffee per day had the lowest death rate, and those who drank one to three cups per day had the highest death rate.

The Onset Study yielded yet more conflicting data. This trial was conducted in 45 US community hospitals between 1989 and 1994 with 3,882 subjects who had suffered an acute MI [19]. Participants were again asked about their frequency of consumption of caffeinated tea and coffee during the year prior to the MI. The authors found that those with a higher coffee intake (over 14 cups per week) had a higher prevalence of ventricular arrhythmias, and those with a moderate tea intake (fewer than 14 cups per week) had a lower prevalence of malignant arrhythmias.

The Danish Diet, Cancer, and Health Study with 47,949 participants contradicted the previous studies [5]. They followed 555 subjects who developed atrial fibrillation (or atrial flutter) for an average of 5.7 years. They found no association between the consumption of caffeine and the risk for an atrial arrhythmia. In contrast, a Kaiser study found an inverse relationship between caffeine intake with hospitalization for arrhythmias in 3,137 persons hospitalized for arrhythmias [20•]. The authors concluded that total moderate caffeine intake will not increase the arrhythmia risk.

There are several reasons why such studies may yield differing results. One major point is that all such trials rely on participants to retrospectively report on their caffeine use in the previous year. Such memories may be flawed or unconsciously biased towards producing a desired study result. Patients may not accurately remember how many cups of coffee versus tea versus espresso they drank, or how much caffeine the cups contained. Finally, subjects can develop tachyphylaxis to caffeine when ingested in increasing doses over time.

Energy drinks and arrhythmias

Energy drinks are a relatively new source of caffeine and sugar that have been recently marketed. By self-report, 30-50 % of teens and young adults drink energy drinks regularly [21•, 22•]. Energy drinks are beverages that contain caffeine, taurine, sugar, vitamins and herbs. They are the fastest growing segment of the US beverage market, accounting for roughly \$9 billion in 2011 in the United States alone [21•, 22•].

The major ingredient of these energy drinks is caffeine. Many drinks contain 70-80 mg of caffeine per 8 ounce serving, which is often three times that in a regular cola [21•, 22•]. However, there may be even more caffeine hidden in other additives like a plant called guarana, which contains both caffeine and theophylline. What is concerning about these

energy drinks is that they are unregulated by the FDA, which limits the amount of caffeine in soft drinks but not in these energy drink beverages.

There are several case reports in the literature of arrhythmias following acute ingestion of an energy drink [21•, 22•]. One report notes a 14-year-old boy who developed acute atrial fibrillation (which is normally very rare in teens or children) after downing an energy drink and running a race [22•]. In another case, a 16-year-old boy also had atrial fibrillation after downing a vodka, Adderall, and a Red Bull [22•]. Finally, a 28-year-old man experienced a cardiac arrest after drinking seven to eight cans of an energy drink and competing in a motocross race [23•]. Such case reports may grow in the medical literature as more young people ingest energy drinks.

Treatment options for arrhythmias and caffeine

To summarize, caffeine does not appear to promote arrhythmias, at least for most patients who drink a moderate amount of caffeinated beverages daily. Although it is tempting to blame the double espresso for our patient's lone atrial fibrillation, it probably plays little role in its occurrence. We encouraged her to continue her espresso habit with moderation.

Treatment of arrhythmias and alcohol ingestion

Unlike caffeine, alcohol use may play some role in the initiation and maintenance of atrial fibrillation. Abstaining from at least heavy alcohol is a reasonable recommendation to a patient with atrial fibrillation, especially if the patient feels that alcohol may have triggered the arrhythmia initially.

Ettinger et al. coined the original term “holiday heart” in 1978 to describe episodes of atrial fibrillation in patients imbibing alcohol on the weekend or over the holidays [24]. The authors described 24 patients who were heavy users of alcohol and presented with rhythm abnormalities. Most subjects presented with atrial flutter, although a few had isolated premature ventricular complexes, atrial premature complexes, or junctional tachycardias. Thus, this classic paper did not refer only to alcohol causing atrial fibrillation, although that is how most physicians perceive which arrhythmias alcohol commonly causes.

Greenspon et al. undertook an elegant study that probably could not be done in today's more stringent research world [25]. They performed electrophysiology studies on 14 patients who chronically abused alcohol. The subjects were then given orally (or via IV) 80 proof whisky shots, and the EP study was then repeated. The HV interval prolonged, the heart rate increased, and the corrected sinus node recovery time all shortened with the acute ingestion of heavy alcohol. It was unclear if these findings actually provoked the atrial arrhythmias that were seen, or had significant clinical relevance.

However, such electrophysiologic findings have lent credibility to the concept that alcohol use can promote atrial fibrillation. Researchers at UCSF looked retrospectively at 381 patients who had atrial fibrillation, atrial flutter, or served as controls and evaluated their alcohol use by self-report [26]. All subjects underwent invasive electrophysiology study. Interestingly, heavier alcohol intake was associated with the development of atrial flutter in younger patients, although not with atrial fibrillation. There was also a trend towards shorter atrial refractory periods (which could promote atrial fibrillation) in those patients who drank alcohol as compared with those who did not drink. Such findings may support a mechanistic link between atrial fibrillation and ingestion of alcohol.

Epidemiological data for alcohol-inducing atrial fibrillation

There are ample data from observational studies demonstrating a relationship between increasing alcohol levels and the development of atrial fibrillation.

In the large Copenhagen City Heart Study, a population-based cohort study in Denmark, the association between self-reported alcohol use and atrial fibrillation was studied in 16,415 participants [27]. The authors found that male patients who had a high weekly alcohol intake (over 35 drinks per week) had the highest risk of atrial fibrillation. Heavy alcohol used was associated with atrial fibrillation in 5 % of the patients.

In a similar fashion, the large, population-based cohort study of the Framingham Study was begun in 1948 [28]. The authors identified 1,055 cases of atrial fibrillation and followed them for over 50 years. A weak dose-response association was found between the amount of alcohol consumed and the risk of developing atrial fibrillation. Again, those subjects who drank over three drinks per day (heavy alcohol use) had the highest risk of developing atrial fibrillation. There was little relationship between moderate alcohol ingestion and developing atrial fibrillation.

Conen et al. looked at data generated from the Women's Health Study, a randomized controlled trial of 34,715 women [29]. Again, participants answered questionnaires about their alcohol consumption, which was correlated with the risk of developing atrial fibrillation. The authors found that among these healthy middle-aged women, consuming up to two alcoholic beverages daily was not associated with an increased chance for atrial fibrillation. However, those subjects who did drink over two drinks per day faced a small but elevated risk for atrial fibrillation.

Most longitudinal studies have shown that mild to moderate alcohol use does not seem to increase the risk for developing atrial fibrillation. The Cardiovascular Health Study was a longitudinal study of nearly 6,000 patients aged 65 years or older who were followed long-term [30]. Patients self-reported on their alcohol consumption, and the diagnosis of atrial fibrillation was determined by EKG or discharge diagnoses. The authors found that current alcohol intake (high or low) was not associated with a higher risk for developing atrial fibrillation. As with all of these studies that rely on questionnaires to quantitate alcohol use, there were likely flaws based upon the accuracy of self-reporting alcohol use. Also, most studies were not able to differentiate between acute versus chronic alcohol use, and these may affect the development of atrial fibrillation [31••].

Effects of alcohol on ion channels

Although alcohol seems to have pro-arrhythmic properties, the molecular biology of how this occurs has not been well studied. The available studies present some conflicting data.

Laszlo et al. infused white rabbits with intravenous alcohol for 120 hours and then examined ion current flux of the different cardiac cells using the patch clamp technique [32•]. Alcohol infusions reduced the sodium current (I_{Na}) and the calcium current (I_{Ca-L}) but had no effect on the outward potassium current, I_{K0} . These findings could potentially shorten the atrial refractory period and help promote atrial fibrillation, which mirrors earlier researchers' findings [33]. However, the authors cautioned against extending these results to the clinical realm since their study only looked at the effects of acute alcohol infusion, which differs greatly from chronic ingestion.

Klein et al. also found a similar effect of alcohol on the cardiac sodium channel [34]. They took HEK-293 cells (human embryonic kidney 293 cells) that were transfected with sodium channels and infused them with ethanol. They similarly found that alcohol reduced I_{Na} .

Reduction of the cardiac sodium current may lead to a decrease in the action potential duration. With chronic alcohol use and downregulation of sodium channels, this can also help promote episodes of atrial fibrillation.

Several studies have contradicted these results and have suggested ways in which alcohol may actually have anti-arrhythmic effects [35, 36]. Kostis et al. found that infusion of alcohol in dogs led to an increase in the atrial fibrillation threshold [36]. Such findings would imply that alcohol use could decrease the risk of atrial fibrillation. These contradictory findings may explain in part why low to moderate alcohol use does not seem to increase the incidence of atrial fibrillation in those patients.

Treatment options for arrhythmias and alcohol

Heavy alcohol use may be associated with an increased risk of the development of atrial fibrillation, although the clinical and molecular literature provide conflicting data on this point. As with caffeine use, it is probably wise to advocate at most moderate alcohol ingestion in patients with arrhythmias.

Acknowledgments

Dr. Nipavan Chiamvimonvat has received a grant from the National Institutes of Health and the Veteran's Administration. Richard Myers has received an HHMI Training Grant Fellowship.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

• Of importance

•• Of major importance

- 1•• Di Castelnuovo A, di Giuseppe R, Iacoviello L, et al. Consumption of cocoa, tea, and coffee and risk of cardiovascular disease. *Eur J Intern Med.* 2012; 23:15–25. This is a well-written review article summarizing the cardiac effects including arrhythmias for different caffeinated products like tea and coffee. [PubMed: 22153525]
2. Rashid A, Hines M, Scherlag BJ, et al. The effects of caffeine on the inducibility of atrial fibrillation. *J Electrocardiol.* 2006; 39:421–5. [PubMed: 16919674]
3. Mattioli AV, Bonatti S, Zennaro M, et al. Effect of coffee consumption, lifestyle, and acute life stress in the development of acute lone atrial fibrillation. *J Cardiovasc Med.* 2008; 9:794–8.
4. Myers MG. Caffeine and cardiac arrhythmias. 1991; 114:147–50.
5. Frost L, Vestergaard P. Caffeine and risk of atrial fibrillation or flutter: the Danish Diet, Cancer, and Health Study. *Am J Clin Nutr.* 2005; 81:578–82. [PubMed: 15755825]
6. Chang CY, Yeh TC, Chiu HC, et al. Electromechanical effects of caffeine in failing human ventricular myocardium. *Int J Cardiol.* 1995; 50:43–50. [PubMed: 7558463]
7. Dobmeyer DJ, Stine RA, Leier CV, et al. The arrhythmogenic effects of caffeine in human beings. *N Engl J Med.* 1983; 308:814–6. [PubMed: 6835272]
8. Robertson D, Frohlich JC, Carr RK, et al. Effects of caffeine on plasma renin activity, catecholamines, and blood pressure. *N Engl J Med.* 1978; 298:181–6. [PubMed: 339084]
9. Levi L. The effect of coffee on the function of the sympathoadrenomedullary system in man. *Acta Med Scand.* 1967; 181:431–8. [PubMed: 6023454]
10. Chelsky LB, Cutler JE, Griffith K, et al. Caffeine and ventricular arrhythmias: an electrophysiological approach. *JAMA.* 1990; 264:2236–40. [PubMed: 2214101]
11. Graboyes TB, Blatt CM, Lown B. The effect of caffeine on ventricular ectopic activity in patients with malignant ventricular arrhythmia. *Arch Intern Med.* 1989; 149:637–9. [PubMed: 2465748]

12. Newcombe PF, Renton KW, Rautaharju PM, et al. High-dose caffeine and cardiac rate and rhythm in normal subjects. *Chest*. 1988; 94:90–4. [PubMed: 2454782]
13. Sutherland DJ, McPherson DD, Renton KW, et al. The effect of caffeine on cardiac rate, rhythm, and ventricular repolarization. *Chest*. 1985; 87:319–24. [PubMed: 3971755]
14. Ammar R, Song JC, Kluger J, et al. Evaluation of electrocardiographic and hemodynamic effects of caffeine with acute dosing in healthy volunteers. *Pharmacotherapy*. 2001; 21:437–42. [PubMed: 11310517]
15. Richardson T, Baker J, Thomas PW, et al. Randomized control trial investigating the influence of coffee on heart rate variability in patients with ST-segment elevation myocardial infarction. *Q J Med*. 2009; 102:555–61. This is an unusual study that randomized patients who were having an acute myocardial infarction to acute ingestion of coffee. Coffee actually improved the heart rate variability in these critically ill patients, which is a measure of the cardiac autonomic function.
16. Silletta MG, Marfisi RM, Levantesi G, et al. Coffee consumption and risk of cardiovascular events after acute myocardial infarction: results from the GISSIPrevenzione Trial. *Circulation*. 2007; 116:2944–51. [PubMed: 18056527]
17. Conen D, Chiuve SE, Everett BM, et al. Caffeine consumption and incident atrial fibrillation in women. *Am J Clin Nutr*. 2010; 92:509–14. This is a large cohort of over 33,000 women followed in the Women's Health Study. The authors found no connection between the use of caffeine and the risk of atrial fibrillation. [PubMed: 20573799]
18. Mukamal KJ, Hallqvist J, Hammar N, et al. Coffee consumption and mortality after acute myocardial infarction: the Stockholm Heart Epidemiology Program. *Am Heart J*. 2009; 157:495–501. This is a population-based study in Sweden looking at patients hospitalized with an acute myocardial infarction. They found that coffee consumption was inversely associated with postinfarction mortality. [PubMed: 19249420]
19. Mukamal KJ, Alert M, Maclure M, et al. Tea consumption and infarct-related ventricular arrhythmias: the determinants of Myocardial Infarction Onset Study. *J Am Coll Nutr*. 2006; 25:472–9. [PubMed: 17229893]
20. Klatsky AL, Hasan AS, Armstrong MA, et al. Coffee, caffeine, and risk of hospitalization for arrhythmias. *Permanente J*. 2011; 15:19–25. The authors report on over 3,000 patients hospitalized for cardiac arrhythmias. They found that moderate caffeine in-take does not seem to increase the arrhythmia risk.
21. Seifert SM, Schaechter JL, Hershorin ER, et al. Health effects of energy drinks on children, adolescents, and young adults. *Pediatrics*. 2011; 127:511–28. The authors search available databases for any information about adverse health effects with energy drinks. This is an excellent, comprehensive review about this timely topic. [PubMed: 21321035]
22. Di Rocco JR, During A, Morelli PJ, et al. Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports. *J Med Case Rep*. 2011; 5:18. Two healthy teens experience atrial fibrillation after ingesting energy drinks. Atrial fibrillation is normally quite rare in this patient population. [PubMed: 21247417]
23. Berger AJ, Alford K. Cardiac arrest in a young man following excess consumption of caffeinated “energy drinks”. *Med J Aust*. 2009; 190:41–3. The authors describe a healthy young man who has a cardiac arrest after drinking excessive amounts of energy drinks and competing in a motocross race. This report highlights the potential hidden dangers of these energy drinks. [PubMed: 19120009]
24. Ettinger PO, Wu CF, De La Cruz C, et al. Arrhythmias and the “holiday heart”: alcohol-associated cardiac rhythm disorders. *Am Heart J*. 1978; 95:555–62. [PubMed: 636996]
25. Greenspon AJ, Schaal SF. The “holiday heart”: electrophysiologic studies of alcohol effects in alcoholics. *Ann Intern Med*. 1983; 98:135–9. [PubMed: 6824246]
26. Marcus GM, Smith LM, Whiteman D, et al. Alcohol intake is significantly associated with atrial flutter in patients under 60 years of age and a shorter right atrial effective refractory period. *PACE*. 2008; 31:266–72. [PubMed: 18307620]
27. Mukamal KJ, Tolstrup JS, Friberg JS, et al. Alcohol consumption and risk of atrial fibrillation in men and women. The Copenhagen City Heart Study. *Circulation*. 2005; 112:1736–42.

28. Djousse L, Levy D, Benjamin EJ, et al. Long-term alcohol consumption and the risk of atrial fibrillation in the Framingham Study. *Am J Cardiol.* 2004; 93:710–3. [PubMed: 15019874]
29. Conen D, Tedrow UB, Cook NR, et al. Alcohol consumption and risk of incident atrial fibrillation in women. *JAMA.* 2008; 300:2489–96. [PubMed: 19050192]
30. Mukamal KJ, Psaty BM, Rautaharju PM, et al. Alcohol consumption and risk and prognosis of atrial fibrillation among older adults: the Cardiovascular Health Study. *Am Heart J.* 2007; 153:260–6. [PubMed: 17239687]
- 31••. Kodama S, Saito K, Tanaka S, et al. Alcohol consumption and risk of atrial fibrillation. *J Am Coll Cardiol.* 2011; 57:427–36. This is a meta-analysis looking at 14 studies which related alcohol and atrial fibrillation. The authors found that not drinking alcohol probably provides the most reduction for the risk of atrial fibrillation in terms of alcohol use. [PubMed: 21251583]
- 32•. Laszlo R, Eick C, Schwiebert M, et al. Alcohol-induced electrical remodeling: effects of sustained short-term ethanol infusion on ion currents in rabbit atrium. *Alcohol Clin Exp Res.* 2009; 33:1697–703. This is an interesting basic science paper in which the authors found that short-term ethanol infusion in rabbits alters atrial current densities as measured by patch clamp techniques. [PubMed: 19572985]
33. Fisher VJ, Kavalier F. The action of ethanol upon the action potential and contraction of ventricular muscle. *Recent Adv Stud Cardiac Struct Metab.* 1975; 5:415–22. [PubMed: 1081255]
34. Klein G, Gardiwal A, Schaefer A, et al. Effect of ethanol on cardiac single sodium channel gating. *Forensic Sci Int.* 2007; 171:131–5. [PubMed: 17129694]
35. Nguyen TN, Friedman HS, Mokraoui AM. Effects of alcohol upon experimental atrial fibrillation. *Alcohol Clin Exp Res.* 1987; 11:474–6. [PubMed: 3314563]
36. Kostis JB, Goodkind MJ, Skvaza H, et al. Effect of alcohol on the atrial fibrillation threshold in dogs. *Angiology.* 1977; 28:583–7. [PubMed: 900571]