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Link between coffee and atrial fibrillation debunked?

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1. Introduction

The relationship between atrial fibrillation (AF) and coffee is controversial, although it carries important clinical implications due to the fact that caffeine is one of the most widely consumed active substances in the world and that AF is the most common arrhythmia in adults. The major intake of caffeine in adults comes from coffee, whereas young people consume caffeine in beverages like cola soda and energy drinks (EDs) [1,2].

The estimated caffeine from coffee consumption in the Mediterranean area is about 198 mg/day per person, similar to the 210 mg/day reported in the USA [2].

It is important to distinguish between coffee and caffeine effects. Caffeine exerts a stimulating effect on the heart, increasing cardiac contractility and stroke volume. Coronary vessels dilate while brain vessels constrict and blood flow to the brain decreases [1].

Coffee, additionally to caffeine, contains many different substances, including high levels of phenolic antioxidants consisting principally of chlorogenic acids (CGAs). The total content of CGA in a ‘regular’ cup of coffee (200 mL) varies between 70 and 350 mg [2]. Therefore, if coffee is consumed throughout the day, it may provide up to two thirds of total daily dietary antioxidants [3].

The most recent approach to cardiovascular disease prevention underlines the critical role of lifestyle, nutrition, and physical activity. Core components of a healthy diet include limiting intakes of saturated and trans fats, carbohydrate, alcohol, and sodium and high intakes of fruit, vegetables, (rich in antioxidants) and dietary fibers. The Mediterranean Diet meets these healthy diet requirements. The very recent Mediterranean Diet pyramid included at the basis the physical activity as well as moderate consumption of both coffee and wine [4–6].

2. How can it be that coffee and caffeine have been related to arrhythmias?

Caffeine-rich beverages have been traditionally considered the main driving environmental factor implicated in a variety of ordinary but unspecific symptoms (like palpitations and heartbeat irregularities) in clinical practice. Physicians are particularly worried that these symptoms might be mediated by potentially dangerous arrhythmic events.

Hansson et al. found that the 25% of patients regard coffee as a triggering-factor for arrhythmia [7].

However studies evaluating caffeine effects on supraventricular and ventricular ectopic beats are not linking caffeine dosage with development of arrhythmias.

In a recent meta-analysis, evaluating caffeine effects on ventricular arrhythmias, the overall relative risk for occurrence of ventricular premature beats in 24 h attributed to caffeine exposure was 1.00 (95% CI 0.94–1.06; P n.s.). Sensitivity analysis for caffeine dose, different designs, and subject profile was performed and no major differences were observed [8].

The authors concluded that there is no scientific evidence that supports a clinical recommendation to decrease or avoid caffeine consumption in patients at risk or with suspicious symptoms of arrhythmias. Similarly, prospective clinical trials did not demonstrate an increase in clinically significant ventricular or supraventricular arrhythmias, even after exposure to high doses of caffeine [9].

The Danish Diet, Cancer and Healthy suggested that caffeine consumption was not associated with hospitalizations for AF in a population with high caffeine intake [10]. Moreover, a recent analysis of the Danish Cohort after a median follow-up of 13.5 years found that compared with no intake, coffee consumption was inversely associated with AF incidence, with multivariable-adjusted hazard ratios of 0.93 for more than none to <1 cup/day, 0.88 for 1 cup/day, 0.86 for 2–3 cups/day, 0.84 for 4–5 cups/day, 0.79 for 6–7 cups/day, and 0.79 for >7 cups/day (p-linear trend = 0.02). Results suggested that higher levels of coffee consumption were associated with a lower rate of incident AF [11].

Similarly, in the Women’s Health Study, caffeine consumption was not associated with an increased risk of incident AF in middle-aged women. Women in the highest quintile of caffeine consumption had a similar risk of developing AF compared with women in the lowest quintile [12].

Larsson et al. summarized the current evidence on the relationship between regular coffee and new onset AF and found no significant relationship between coffee intake and incident AF [13]. Of the six studies, one of them showed...
a statistically significant reduced risk of AF when comparing the highest with the lowest category of coffee consumption, but there was no dose–response relationship [13].

When results from all studies were combined, the RR of AF comparing the highest with the lowest category of coffee consumption was 0.96 (95% CI 0.84–1.08).

In a sensitive analysis, which included six studies of coffee consumption and two studies of caffeine intake in relation to AF risk, the overall RR for the highest versus lowest category of coffee or caffeine intake was 0.95 (95% CI, 0.86–1.06; I² = 46.3 %). There was no evidence of a nonlinear relationship between coffee consumption and AF risk. The RR per 2-cups/d increment of coffee consumption was 0.99 [13].

Cheng and coworkers showed that the risk of developing AF decreases by 6% for each increase of habitual intake of 300 mg of coffee, leading to consider that usual intake of caffeine may even be protective against arrhythmic outcomes. They suggested that habitual caffeine consumption might reduce AF risk by its anti-fibrosis effect [14].

At last, the protective effect of caffeine might be related to co-occurring phytochemicals because caffeine is normally consumed in the form of plant-derived products and extracts that invariably contain other potentially bioactive phytochemicals [15].

In conclusion, caffeine exposure based solely on coffee consumption seems to not influence the risk of AF.

3. The new frontier: caffeine from EDs

Evidence from clinical studies suggests that caffeine might not be arrhythmogenic in most contexts except in special circumstances and at very high doses, and very high doses are included in EDs [16,17]. Several case reports suggest a trigger effect of these beverages on arrhythmias [17]. Caffeine toxicity produces supraventricular tachycardia, AF, and ventricular fibrillation [1,2].

EDs were originally conceived for athletes, to combat fatigue, but nowadays, young adults and teenagers represent their new main market segment.

Caffeine is a widely used energetic substance and many papers suggested great effects during endurance activities.

A systematic review was carried out on randomized placebo-controlled studies investigating the effects of caffeine on endurance performance and a meta-analysis was conducted to determine the ergogenic effect of caffeine on endurance time-trial performance. Caffeine showed a small but evident effect on endurance performance when taken in moderate doses (3–6 mg/kg). Similarly an overall improvement following caffeine compared to placebo in mean power output (3.03 ± 3.07%; effect size = 0.23 ± 0.15) and time-trial completion time (2.22 ± 2.59%; effect size = 0.41 ± 0.2) was reported. However, differences in responses to caffeine ingestion have been shown, with two studies reporting slower time-trial performance, while five studies reported lower mean power output during the time-trial [18].

Consumption of EDs by young people has increased dramatically in recent years in order to improve their physical and psychological performance [18–20]. The literature shows contradictions about the capacity of EDs to enhance psychophysical results. These drinks are very diffuse within young people because they are sold in cans, are unrestricted and marketing campaign identify it as safe products able to improve the physical and mental performance [19].

Currently, there is not a basic formula or ingredient included in commercial EDs. However, there are a number of substances, i.e. methylxanthines, often present in high quantities that give the beverage psychostimulants characteristics [17]. The EDs contain caffeine, taurine, creatine, carnitine, B vitamins, maltodextrin, inositol, and glucuronolactone; there are also herbs such as guarana (containing caffeine), ginseng, and ginkgo biloba [19].

The guarana plant and berry has one of the naturally highest levels of caffeine and there are also traces of theophylline and theobromine.

Being a working student was significantly positively associated both to EDs (OR 1.5) and ginseng use (OR 1.4). The most frequently reported academic and other reasons for ED use were: ‘to study longer’ (47.5%), and ‘to socialize’ (29.1%). The most often used combinations were ED containing alcohol (65.6%) and ginseng-coffee beverages (71.8%) [20].

A paper described the cases of two boys (14 and 16 years old) without previous cardiac event that developed AF after consuming EDs [20]. The first boy developed palpitations and chest pain after taking an ED containing high dose of caffeine. The same symptoms had been warned five days before having consumed another popular ED. The second boy experienced vomiting, attention problems, hyperactivity, asthma, and chest pain after taking a mix of EDs, spirits, and vodka, together with amphetamines. Mattioli et al. also reported cases of AF developed after ingestion of EDs [17,19].

With the increasing popularity of EDs in the young population, physicians should be aware of the arrhythmogenic potential associated with highly caffeinated beverage consumption. The FDA is considering new regulation of these beverages, but product specificity is needed to evaluate safety.

In conclusion, there is no relationship between coffee consumption and arrhythmias, however concern exists about the EDs could act as a trigger for arrhythmias. More studies are needed to explore this specific effect of EDs.
References

Papers of special note have been highlighted as either of interest (-) or of considerable interest (---) to readers.

--- Opinion paper from the EFSA.


