The end of a year is a time to reevaluate, resolve, and begin anew. In this Heartbeat, I want to share some news I’m not happy about, which has led to my New Year’s resolution for 2017.

Investigators are calling into question the long-held notion that “moderate” alcohol consumption offers health benefits, especially cardiovascular disease (CVD) benefits. They’re saying there may be some positive effects, but they exist at lower, not daily, “doses” of ethanol, and may even be nonexistent.

This is “hard to swallow” news, considering all the positive press in recent years given to the beneficial effects of moderate alcohol consumption. I’m a firm believer in two drinks/day as per our August 2016 Heartbeat on diet (maybe a touch over).

J Curve

There is a relationship between mortality from CVD and the average amount of alcohol consumed per day that is consistent with a J-shaped curve. A similar relationship has been reported for mortality from ischemic heart disease (IHD) in one meta-analysis. Compared to long-term abstinence, daily consumption of small or moderate amounts of alcohol was associated with reduced mortality from IHD, with the nadir of the relative risk curve at about 31 g of ethanol per day for men and a substantially lower amount for women (about 11 g/day for IHD mortality—less than one drink/day—and 14 g/day for IHD morbidity).1

(See graphic below) At higher daily intakes of alcohol, the risk reduction was gradually replaced by increased risk.

Lower recommendations for women is not misogynist, but based on the fact that alcohol metabolism in the liver is slower for women.

Sweet Spot

The sweet spot (lowest point of the J), where we always want to go (most benefit with the lowest risk) is moving left and seems smaller before the risk goes up.

The positive press in recent years given to the beneficial effects of moderate alcohol consumption is based on many observational studies without statistical support. The problem is the fact that bad news on the topic just doesn’t seem as likely to hit the headlines. There is
certainly a large audience for “good news” about alcohol consumption. Any possibility of benefit is welcome news to those who enjoy its consumption (me) and to those who profit from its sale.

Data from new methodologies, such as Mendelian randomization and data from large international meta-analysis studies, have led us to rethink this J-shaped relationship in that the dose-response has shifted to the left. In one Mendelian analysis of 261,991 individuals of European descent, reduction of alcohol consumption, even for light-to-moderate drinkers (one drink/day for women and two drinks/day for men), was beneficial for cardiovascular health.2

There are a large number of individual variables that can influence response to alcohol, including genetics, sex, race/ethnicity and socioeconomic status.3 In recent years, there has been growing concern regarding whether purported beneficial effects of moderate alcohol use are real or are attributable to confounding factors not adjusted for in the studies performed.4 People who drink one to two drinks per day usually have better health habits.

New information, in an analysis of data from 52 countries—the INTERHEART study—have “moved the J curve” left. In men and women, alcohol consumption exceeding one to two drinks/day is associated with an increase in the relative risk of hypertension. Binge drinking on a regular basis is likely to “acutely” increase blood pressure and the risk of stroke. Binge drinking, defined as six or more drinks in a single episode of drinking, significantly increases the risk of myocardial infarction (MI) over the next 24 hours (odds ratio: 1.4; p < 0.01) compared to less consumption of alcohol.5 This risk is even worse for people greater than 65 years old.

The protective association between alcohol use and MI was no longer significant when any alcohol was consumed more than four times a week—so much for the protective effects of a drink a day. “Alcohol use with a frequency of one to four times a week (again, not a day— a week) was associated with a reduced risk of MI; anything more than that had a deleterious effect on risk of MI.

Importantly, the INTERHEART investigators noted that the protective effect was not uniform among all regions or populations. Where there seemed to be a protective effect in Europe, North America and Australia/New Zealand, there was none at all seen for people in Southeast Asia. Also, the protective association of alcohol against MI was greater in women and in people ≥ 45 years of age.

Stroke is also an important consideration. Evidence suggests that the level of alcohol consumption reported to be protective for the heart is lost for the brain in terms of incident stroke and stroke mortality. In other words, levels of consumption, which seem protective for the heart, actually increase risk of stroke.

No Sweet Spot

The risk of atrial fibrillation begins at the lowest doses of alcohol consumption and increases in a dose-response fashion.6,7 An editorial comment in this month’s JACC concludes, “Alcohol is a potentially addictive and dangerous drug, both for the cardiovascular system and multiple other organ systems. The recent infatuation with the potential benefits of light-to-moderate drinking for CVD protection appears to be based on observational and subtly confounded data, rather than on randomized clinical trial evidence, and perhaps on more than a little wishful thinking.”8

Where are We Now?

• A low-to-moderate level of alcohol consumption (one to two drinks/day, but not every day) is probably not harmful to overall cardiovascular health—one for the ladies and two for the gentlemen—skipping at least three/day/week. This will be the hard part, which I feel is the “new safer moderate sweet spot.”

• In the absence of randomized controlled clinical trials, healthcare professionals should not recommend alcohol consumption as a primary or secondary lifestyle intervention to decrease risk, but rather should continue to recommend established strategies, such as physical activity and a healthy diet as per our July and August 2016 Heartbeats (on our website—www.SJHG.org).

• Lower levels may be appropriate for specific groups—such as the elderly or particular racial groups (e.g., in South Asia and the Middle East, and African-Americans).
• Those who currently abstain from alcohol should not begin drinking to reduce their risk of health problems.

**Diastolic Blood Pressure: What’s Optimal?**

Since we now all can relate to the J curve, I thought it would be good to apply it to another topic. For years, there has been a debate about not treating blood pressure (BP) too vigorously for fear of decreasing diastolic pressure too much.

Randomized data regarding the optimal BP target conflict, with the most recent data from SPRINT (Systolic Blood Pressure Intervention Trial) suggesting that lower is indeed better.\(^9\) Intuitively, though, it would seem that there should be a lower limit below which BP reduction would be harmful—the so-called J-curve.\(^10\) Coronary blood flow occurs predominantly during diastole; as such, a reduction in diastolic BP (DBP) would be expected to decrease coronary blood flow, especially if obstructive coronary artery disease (CAD) is present. Hypertension that results in left ventricular hypertrophy may further increase myocardial demand, but overly aggressive hypertension therapy may decrease coronary perfusion pressure, creating a supply/demand mismatch.\(^11\) Several observational analyses support the existence of a J-curve.\(^12\)

An important study by McEvoy et al. presents an insightful analysis of 11,565 subjects from the ARIC (Atherosclerosis Risk in Communities) cohort.\(^13\) Investigating the association between high-sensitivity cardiac troponin-T (hs-cTnT) levels and DBP, they found that compared with people with DBP of 80 to 89 mm Hg, those with a DBP of 60 to 69 mm Hg had higher prevalence of baseline hs-cTnT ≥14 ng/l. Those with DBP < 60 mm Hg had an even higher prevalence. The hypothesis suggested by these associations is that low DBP causes ongoing subclinical myocardial damage that leads to chronic troponin elevation, explaining an association between low DBP and adverse cardiac outcomes.

Compared with a DBP of 80 to 89 mm Hg, a DBP < 60 mm Hg was significantly associated with incident CAD and mortality. Importantly, there was not an association with stroke. The association between DBP and incident CAD was strongest when the baseline level of hs-cTnT was ≥14 ng/l (interaction \(p < 0.001\)), suggesting that troponin elevation may not only have been a marker of myocardial damage, but also on the causal pathway for coronary events. These associations were most pronounced among patients with baseline systolic blood pressure (SBP) ≥ 120 mm Hg, which meant that those with an elevated pulse pressure (PP = SBP - DBP) > 60 mm Hg were at particular risk.

**Conclusion**

Particularly among adults with a SBP ≥ 120 mm Hg, and thus elevated PP, low DBP was associated with subclinical myocardial damage and CHD events. **When titrating treatment to SBP < 140 mm Hg, try to ensure that DBP levels do not fall below 70 mm Hg, and particularly not below 60 mm Hg.**
References


