The Physiological Mechanism for Alcohol to Raise Blood Pressure

1. What is the physiological mechanism for alcohol to raise blood pressure (and how much alcohol!)?

Question submitted by: Graham E. White, Parksville, British Columbia

When we think about how long alcohol has been consumed, and how much is consumed around the world, it is quite disappointing to admit that the answer to your question is still unclear. It is very clear that alcohol consumption is associated with an increased incidence of hypertension and stroke.

In my opinion, the most likely explanation lies in activation of the sympathetic nervous system. It has been shown that alcohol consumption triggers widespread sympathetic discharge and acutely raises blood pressure. It is suspected that chronic alcohol consumption leads to chronic stimulation and persistent hypertension. In addition, sympathetic stimulation increases the response to angiotensin II in the secretion of aldosterone, which directly contributes to hypertension.

Alcohol consumption is also related to increased insulin secretion, which may contribute to hypertension by directly influencing sodium excretion, so that more sodium is re-absorbed by the kidneys back into the body. Short-term sympathetic stimulation may also increase platelet stickiness, contributing to the increased acute events that occur with excess alcohol.

The second part of your question, how much alcohol will trigger hypertension, is also unclear. Research studies suggest that there is a linear relationship between the amount of alcohol consumed and blood pressure elevation. On the other hand, we also know that moderate amounts of alcohol (1 to 2 oz daily) are associated with a decreased risk of myocardial infarction and lower mortality. This implies that the contribution of such moderate amounts of alcohol to hypertension is minimal. As in much of life, moderation is the key.

Answered by:
Dr. Wayne Warnica

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ASA Use for Primary Prevention of CVD

2. Is there a benefit to prescribing ASA for a hypertensive patient with no other CV risk factors and a Framingham risk score of less than 10%?

Question submitted by: T. Petraglia, Québec

Using ASA for the primary prevention of CVD events increases the risk for major bleeding events in men and women, including hemorrhagic stroke and serious gastrointestinal bleeding requiring transfusion.[1] Gastrointestinal bleeding risk increases with age and the concomitant use of NSAID therapy more than triples this risk.[2] Nonetheless, in the absence of gastrointestinal ulcer, ASA use for the primary prevention of CVD provides more benefit than harm.

Meta-analyses of randomized control trials have attempted to quantify these benefits and risks.[3] For a hypothetical group of 1,000 men younger than 60-years-of-age with a 6% 10-year baseline risk for myocardial infarction, ASA use can be expected to prevent approximately 19 myocardial infarctions and cause approximately one hemorrhagic stroke and eight major bleeding events. Likewise, for a similar low-risk hypothetical group of 1,000 women, aspirin use will prevent approximately 10 strokes and cause approximately four major bleeding events.[3]

In short, ASA use in primary prevention saves lives, but it needs to be individualized.

Reference:
1. Ridker PM, Cook NR, Lee IM, et al: A Randomized Trial of Low-dose Aspirin in the Primary Prevention of Cardiovascular Disease in Women. N Engl J Med 2005;352:1293-304.
2. Hernández-Díaz S, García Rodríguez LA: Cardioprotective Aspirin Users and Their Excess Risk of Upper Gastrointestinal Complications. BMC Med 2006;4:22
3. Berger JS, Roncaglioni MC, Avanzini F, et al. Aspirin for the Primary Prevention of Cardiovascular Events in Women and Men: a Sex-specific Meta-analysis of Randomized Controlled Trials. JAMA 2006;295:306-13.

Answered by:
Dr. Theodore K. Fenske

Prescribing Amiodarone for Treatment of Tachyarrhythmias

3. Is there any reason cardiologists prescribe Amiodarone for tachyarrhythmias? Are there no other choices? It is my understanding that it remains in the body for five to six weeks. What happens if you develop toxicity or a significant drug-drug interaction?

Question submitted by: Dr. Richard Wianecki, Ajax, Ontario

Amiodarone is a "broad spectrum" antiarrhythmic that is useful in both supraventricular and ventricular arrhythmias. It has little negative inotropic effect; so, it can be used when treating heart failure. It also has the lowest "proarrhythmic" action of all antiarrhythmic drugs. On the other hand, it is pharmaceutically unwieldy, with a very long half-life of 60 days or more. Finally, as has been implied, it does have significant adverse effects, with liver, lung, thyroid, skin and eye problems being most prominent. All these are dose related and may regress over time when the drug is stopped. Important drug interactions to note include increased warfarin and digoxin effects. A newer drug, dronedarone, seems to have fewer adverse effects, but it is less efficacious in preventing recurrent atrial fibrillation.

Answered by:
Dr. Thomas W. Wilson