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THE EFFECTS OF SMOKING, ALCOHOL AND DRUGS ON CARDIOVASCULAR SYSTEM- A SYSTEMATIC REVIEW ON CARDIOLOGY DISEASES

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ABSTRACT:

Tobacco use is a leading cause of morbidity and mortality from cardiovascular disease (CVD). There is little evidence linking smoking to various subtypes of CVD, both fatal and non-fatal outcomes. Current smoking increases the risk of nearly all CVD subtypes, at least doubling the risk of many, including AMI, cerebrovascular disease, and heart failure. Paroxysmal tachycardia is a newly discovered smoking-related risk. The effects of alcohol on cardiovascular (CV) health are complex. The links between alcohol consumption and CV diseases such as hypertension, coronary heart disease, stroke, peripheral arterial disease, and cardiomyopathy have been extensively researched and are summarised in this review. Although many behavioural, genetic, and biologic variants influence the link between alcohol use and CV disease, the dose and pattern of alcohol consumption appear to be the most influential. Several drug classes may cause heart failure in patients who do not have concurrent cardiovascular disease or may hasten the onset of heart failure in patients who already have left ventricular impairment. Using the MEDLINE database and lateral references, we conducted a review of the literature on drug-induced heart failure.

Key words:

Alcohol consumption, Drug induced diseases, Tobacco consumption, Cardiovascular problems

Introduction

Cardiovascular disease

Cardiovascular disease is the dysfunction or damage to the tissues of the heart or blood vessels (e.g. cardiac muscle, arterial walls) that results in reductions of their inherent capacity to carry out their normal functions. These functions include the regular and consistent 'pumping' of blood throughout the body, which is done to supply all parts of the body (including the brain) with nutrients and oxygen.[1]

1.2 CONDITIONS AND COMPLICATIONS RELATED TO CARDIOVASCULAR DISEASE

1.3 HYPERTENSION

Hypertension, or high blood pressure, is a condition in which the heart must work harder than usual to keep blood circulation at acceptable levels. Many cases of hypertension are linked to age-related increases in blood pressure[2]. Estimates suggest that approximately 33% of all adults over 20 years of age are affected by hypertension[3]. Hypertension may be a risk factor in the development of atherosclerosis, and thus coronary artery disease[2].

1.4 DRUG INDUCED HYPERTENSION:

A myriad variety of therapeutic agents or chemical substances can induce either a transient or persistent increase in blood pressure, or interfere with the blood pressure-lowering effects of antihypertensive drugs. Some agents cause either sodium retention or extracellular volume expansion, or activate directly or indirectly the sympathetic nervous system[4].

An accurate and detailed medical history should, therefore, include specific inquiries concerning foods, poisons, and medications. This is particularly important with regard to such things as over-the-counter drugs, nutritional supplements, diets, and health foods, which are often not considered to be drugs and, therefore, are frequently omitted from the history.[5]

1.5 CONGESTIVE HEART FAILURE

Congestive heart failure is a syndrome that can be caused by a variety of abnormalities, including pressure and volume overload, loss of muscle, primary muscle disease or excessive peripheral demands such as high output failure. In the usual form of heart failure, the heart muscle has reduced contractility.[6]

1. DRUG INDUCED HEART FAILURE

Congestive heart failure is predominantly caused by cardiovascular diseases, such as coronary artery disease, hypertension and valvular heart disease [7,8]. However as several categories of drugs may exert unfavorable

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hemodynamic effects, these drugs may act as a precipitating factor for a relapse in patients with previously compensated CHF. According to MEDLINE Data base potential role in the occurrence of heart failure of cytostatics, immunomodulating drugs, antidepressants, calcium channel blocking agents, nonsteroidal anti-inflammatory drugs, antiarrhythmics, beta-adrenoceptor blocking agents, anesthetics and some miscellaneous agents.[9]

2. ARRHYTHMIAS

Cardiac arrhythmias are irregularities in heartbeat, or the rate at which blood is pumped around the body by the heart. A common form of these is atrial fibrillation (also known as A-fib), related to disorders of an atrium[10]

3.1 DRUG INDUCED ARRHYTHMIAS

Many widely used medications may cause or exacerbate a variety of arrhythmias Perhaps less familiar to clinicians is the fact that drugs can also trigger other arrhythmias, including bradyarrhythmias, atrial fibrillation/atrial flutter, atrial tachycardia, atrioventricular nodal reentrant tachycardia, monomorphic ventricular tachycardia, and Brugada syndrome. Some drug-induced arrhythmias (bradyarrhythmias, atrial tachycardia, atrioventricular node reentrant tachycardia) are significant predominantly because of their symptoms; others (monomorphic ventricular tachycardia, Brugada syndrome, torsades de pointes) may result in serious consequences, including sudden cardiac death. [11]

3. MYOCARDIAL INFARCTION

Myocardial infarction (MI), more commonly known as heart attack, is the impairment of blood flow to some part of the heart, which may result in extensive damage to cardiac muscle and other tissues due to oxygen starvation.[12] Eventually, this results in enough cumulative cardiac damage—and symptoms noticeable enough—to become apparent as a heart attack.[1]

4.1 DRUG INDUCED MYOCARDIAL INFARCTION

Some drugs and chemical substances can cause MI, and their mechanisms can be diametrically different: from a direct toxic effect on cardiomyocytes to probably the most common mechanism, which is vasospasm. Also documented is the risk of MI after ingestion of such substances as ecstasy, methamphetamine, LSD, cocaine, heroin[17] and volatile substances (propane-butane)[19].

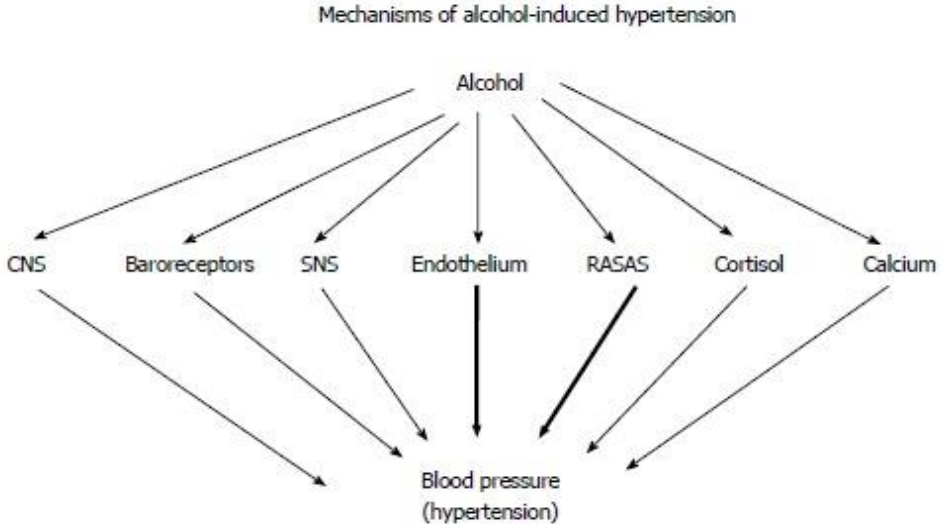
4. Alcohol induced cardio vascular diseases –

Alcohol (ethanol, EtOH) has pleiotropic effects and causes a variety of acute and long-term effects as a result of direct interactions with alcohol targets, as well as the effects of alcohol metabolites and metabolism.

Numerous studies have shown that moderate to low alcohol consumption provides significant health benefits, particularly for the cardiovascular system [20].

In addition, long-term alcohol misuse causes liver damage and cardiac disease[21,22,23]. (for example, alcoholic cardiomyopathy),

Alcoholic cardiomyopathy is linked to heavy alcohol consumption (regardless of beverage type) [24].



According to the World Hypertension League, the substantially higher effect of alcohol on systolic blood pressure compared to diastolic blood pressure could reflect an imbalance between central nervous system components influencing cardiac output and the peripheral vascular effects of alcohol[26,27].

There has been a considerable rise in plasma renin activity in individuals who consume heavy alcohol compared to those who consume mild or moderate alcohol[32,34,35].

5.1 Myocyte loss

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Necrosis or apoptosis (programmed cell death) can cause myocyte loss or cell death[40].
Capasso et al.[41] discovered that rats fed ethanol in their drinking water for 8 months lost a large number of myocytes in the LV. Chen et al.[42] investigated the effects of acute alcohol (500 and 1000 mg/dL) consumption (24 h) on the apoptotic process utilising primary newborn myocyte cell cultures.

5.2 Calcium homeostasis

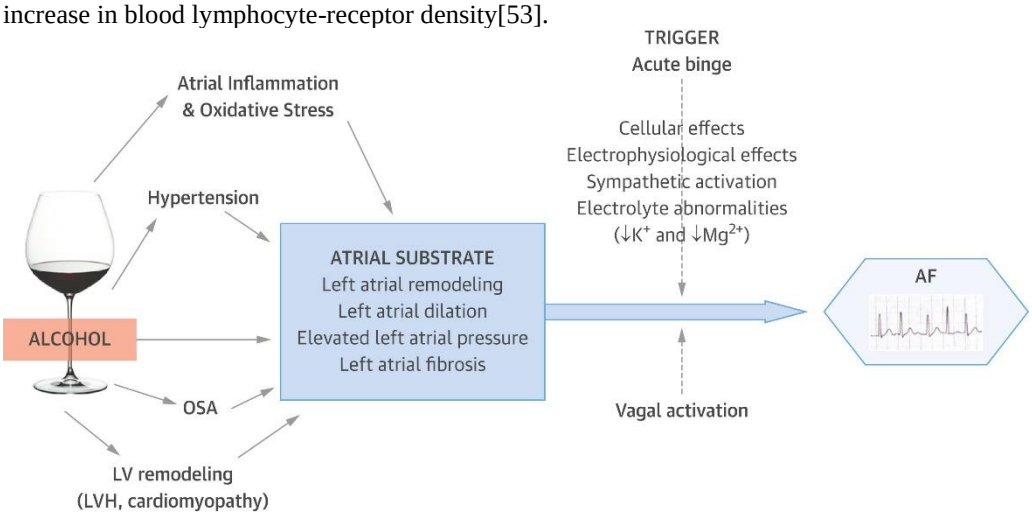
Normal cellular function requires calcium homeostasis. Alcohol consumption for a short length of time has been linked to impaired cytosolic Ca2+ transients[43] and decreased myofilament Ca2+ sensitivity, according to reports.[44]

5.3 Oxidative stress

Both acute[47] and chronic[48]ethanol treatment has been shown to enhance myocardial lipid peroxidation and protein oxidation while decreasing mitochondrial GSH content,[49] implying that reactive oxygen species (ROS) play a role in the beginning of cardiac damage.

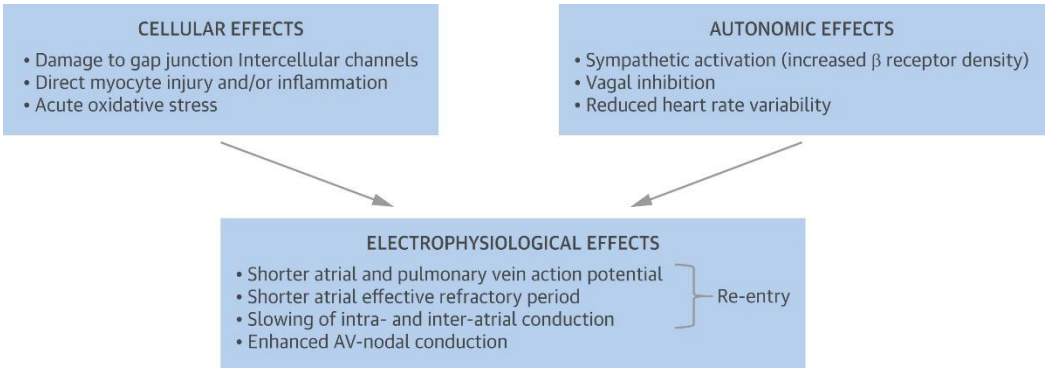
5.5 Autonomic effects of alcohol

Alcohol has an influence on autonomic regulation, which may have a role in the development of AF. In patients with past alcohol-induced AF, Mäki et al. [52] found a sympathetic response to 1.25 g/kg alcohol, as well as a 29 percent increase in blood lymphocyte-receptor density[53].



5.6 Atrial structural effects of alcohol

Alcohol causes tissue fibrosis and has direct effects on atrial excitation-contraction coupling. Myofilament calcium sensitivity was reduced in rats who consumed alcohol for two months, as was their reaction to inotropes [54].



5. Vascular actions

Alcohol's effect on the vasculature varies depending on its concentration and the type of blood vessel[55,56].

6. Cardiac actions

Alcohol has been found to directly reduce heart muscle contractility in a dose-dependent way [58,56] This negative inotropic effect can be seen in the isolated heart or when the autonomic nervous system has been blocked[58]

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The sympathetic nervous system's activity appears to hide alcohol's direct inhibitory effect on the heart. Indeed, we've found that after using a beta blocker, the alcohol-induced increases in heart rate and cardiac output are reduced[59]

7.1 Oxidative status

Several in vitro investigations on polyphenols from wine, beer, and vegetables have revealed that these chemicals have antioxidant properties, despite the fact that alcohol is known to cause oxidative stress.

7.2 Inflammation

The link between alcohol intake and blood concentrations of C-reactive protein (CRP), interleukin 6 (IL-6) and tumour necrosis factor (TNF-) was not significant in a recent meta-analysis[61].

8.What You Need To Know About Smoking And Cardiovascular Disease[64]

According to the 2014 Surgeon General's Report on Smoking and Health, smoking is a major cause of cardiovascular disease (CVD) and accounts for one out of every four CVD deaths. CVD is the leading cause of death in the United States, claiming the lives of over 800,000 individuals each year. Heart disease affects more than 16 million Americans.

8.1 How Smoking Harms The Cardiovascular System

Cigarette smoke contains chemicals that cause the cells that line blood vessels to swell and become inflamed. This can cause blood vessels to narrow, which can lead to a variety of cardiovascular problems.

Atherosclerosis occurs when fat, cholesterol, and other compounds in the blood produce plaque that builds up in the walls of arteries, narrowing them and making them less flexible. As plaque builds up inside the arteries, the opening narrows, and blood can no longer circulate freely to various regions of the body. The production of plaque in blood arteries is accelerated by smoking.

Coronary Heart Disease develops when plaque narrows or clots block the arteries that provide blood to the heart muscle. Cigarette smoke contains chemicals that cause blood to thicken and clot inside veins and arteries. A clot can cause a heart attack, which can result in death.

A stroke is a loss of brain function caused by a disruption in blood flow within the brain. Strokes can result in long-term brain damage as well as death. Smoking raises the risk of having a stroke. Smokers are more likely to die from strokes than former smokers or persons who have never smoked.

Peripheral Arterial Disease (PAD) When blood vessels narrow and the flow of blood to the arms, legs, hands, and feet is diminished, this is known as peripheral vascular disease (PAD). When blood flow is limited, cells and tissue are deprived of oxygen. An infected limb may need to be amputated in extreme circumstances. The most common preventable cause of PAD is smoking.

Abdominal Aortic Aneurysm An abdominal aortic aneurysm is a bulge or weakened area in the aorta that runs through the abdomen. The primary artery that delivers oxygen-rich blood throughout the body is the aorta. Smoking has been linked to early damage to the abdominal aorta, which can result in an aneurysm.

7. Smoking induced HTN[65]

Cigarette smoking has been linked to coronary artery vasoconstriction. Cigarette smoking may increase coronary blood flow in people who do not have coronary heart disease, but it may reduce coronary blood flow in the presence of coronary disease.

Tobacco Use and Cardiovascular Disease

Cigarette smoking is a foremost cause of CVD, and one study found that cigarette smoking increases the risk of coronary heart disease (CHD) even in people with low cholesterol levels in their blood.[66]

The development of atherosclerotic alterations with narrowing of the arterial lumen and induction of a hypercoagulable state, both of which increase the risk of acute thrombosis,[67] are the general processes by which smoking causes cardiovascular events.

Furthermore, there is strong evidence that smoking leads to the development of atherosclerotic plaque[68-71].

Summary CHD, stroke, aortic aneurysm, and PAD are all caused by cigarette smoking and involuntary exposure to cigarette smoke. The risk is seen both as an increased risk of acute thrombosis of narrowed vessels and as an increased degree of atherosclerosis in the blood vessels involved.The quantity of cigarettes smoked and the length of time spent smoking enhance the cardiovascular risks associated with cigarette smoking. However, even moderate amounts of cigarette smoke, such as secondhand smoke, significantly increase the risk of CVD. Cigar users, on the other hand, appear to have lower CVD risks than cigarette smokers.

Pathophysiology

Mechanisms

Cigarette smoking causes acute myocardial ischemia by disrupting the equilibrium between the demand for oxygen and nutrients in the heart and the supply of oxygen and nutrients to the heart (Figure 1). Nicotine activation of the

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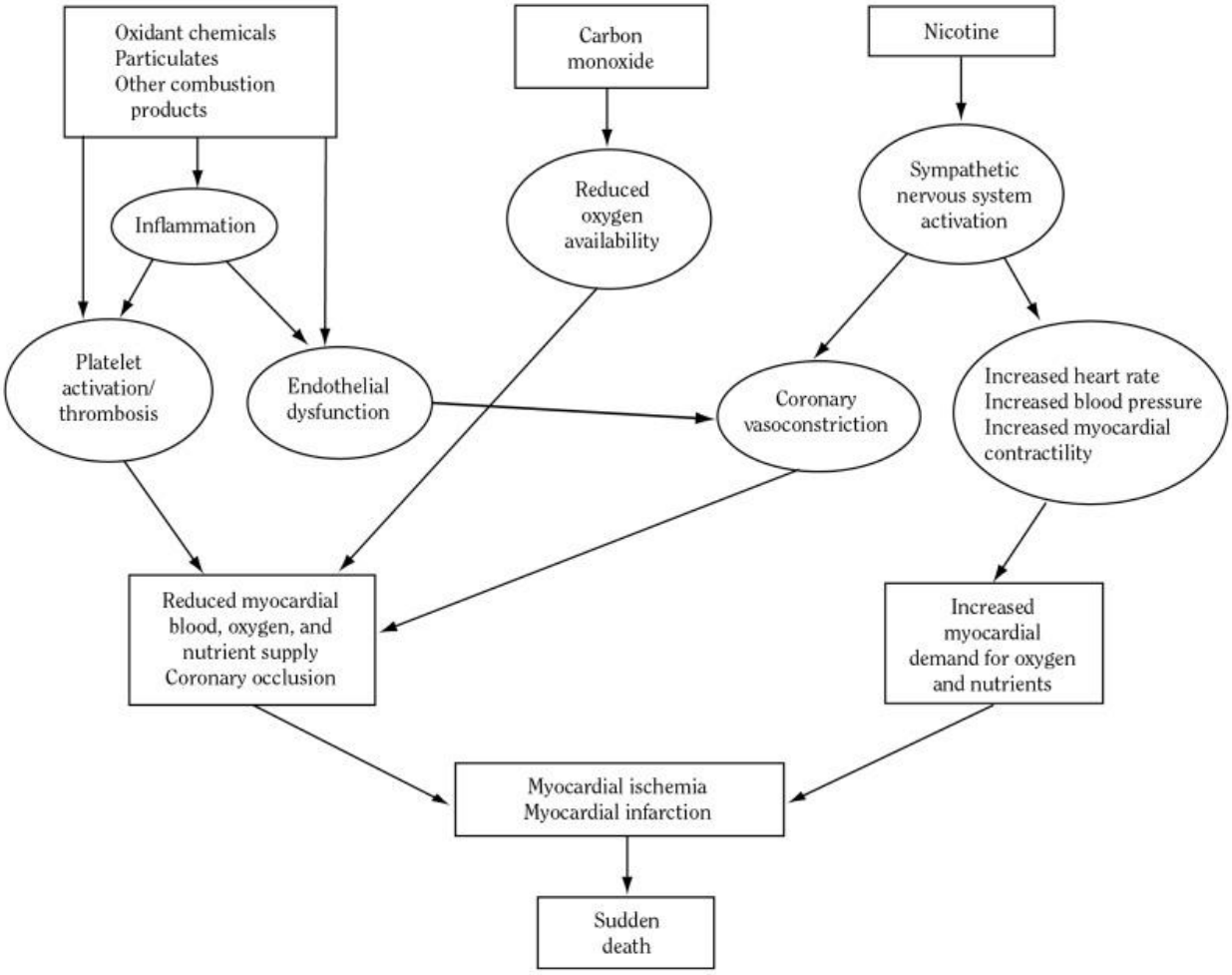
sympathetic nervous system and the heart results in an increase in oxygen demand in the myocardium. Cigarette smoking raises plasma norepinephrine and epinephrine levels and increases norepinephrine and epinephrine urine excretion for 24 hours.[72]



Overview of mechanisms by which cigarette smoking causes an acute cardiovascular event. *Source:* Benowitz 2003. Reprinted with permission from Elsevier, © 2003.

Conclusion:

Cigarette smoking raises the risk of AMI through a variety of factors. Smoking boosts blood LDL-cholesterol and triglyceride levels while lowering serum HDL-cholesterol levels, which contributes to atherogenesis. Furthermore, cigarette smoke causes LDL to be damaged by free radicals, resulting in a buildup of oxidised LDL-cholesterol within the artery wall. Smoking tends to contribute to the vascular inflammation associated with atherosclerosis, as seen by smokers having greater blood C-reactive protein levels than nonsmokers[95]. It's unclear if these atherogenic processes are more prevalent in the right or left coronary artery circulation. Because atherosclerosis progression tends to occur at similar rates in present and past smokers[98] some of the negative effects of smoking on atherogenesis may not be quickly reversible after smoking cessation.

Smoking affects the sympathetic nervous system (SNS) via increasing heart rate and systolic blood pressure, primarily due to nicotine content. Increased myocardial oxygen needs occur from an increase in the rate-pressure product. Increased SNS activity caused by nicotine also causes coronary artery vasoconstriction[97], reducing myocardial blood flow at a time when oxygen demand is rising. In addition to raising myocardial oxygen demand and lowering coronary blood flow.

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Cigarette smoking raises blood carboxyhemoglobin levels, thus reducing oxyhemoglobin-mediated myocardial oxygen supply. It's unclear whether cigarette smoking causes greater right-sided coronary artery vasoconstriction than left-sided coronary artery vasoconstriction. Nasal cocaine causes equal vasoconstriction in the left and right coronary arteries[99]. Unfortunately, there hasn't been a similar comparison done before and after cigarette smoking.

Conflict of interests: No conflicts of interest

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