

Introduction:

The heart is an organ that serves as a pump to circulate the blood. The task of the heart is to pump enough blood to deliver a continuous supply of oxygen and other nutrients to the brain and the other vital organs. Many toxic agents such as cocaine can affect the heart performance. They can effect sympathetic tone, heart rate, blood pressure, myocardial contractility etc.

Methods:

The clinical trial which studied 24 healthy volunteer subjects (12 men and 12 women; 24 to 44 years of age) after written informed consents were obtained. All subjects were normotensive with no history of **cardiovascular** disease or substance abuse. No subject was taking any medications that had cardiovascular or autonomic effects. Volunteers received intranasal cocaine hydrochloride 2 mg/kg in a 10% solution.. Electrocardiogram, HR, and Mean Arterial Pressure recorded continuously using a multichannel digital data recorder.

Results:

T.1: Heart Rate and blood pressure Response to Cocaine

Intervention	Heart Rate (Beats/Min)	Mean Arterial Pressure (mm Hg)
Baseline	64	77
Cocaine	75	87

Discussion:

Most of the cardiac effects with cocaine can be traced by to two basic mechanisms: one is its ability to block sodium channels, leading to a local anesthetic, the second is its ability to block reuptake of catecholamines in the presynaptic neurons in the central and peripheral nervous system, resulting in increased sympathetic output and increased catecholamine.

Cocaine consumption produces quick and significant elevations in blood pressure through a variety of processes including potent sympathomimetic properties and arterial vasoconstriction, Our aortic stiffness parameters correlated with the duration and frequency of cocaine use in this clinical trial study. This suggests that cocaine's effect on arterial stiffness is due to a change in the proportion of elastic versus non-elastic content ratio, rather than vascular smooth muscle hypertrophy. Cocaine also induces high blood pressure by triggering the release of catecholamines such as norepinephrine and dopamine. Blood vessels become thinner as a

result of this constriction. Even when blood arteries are thinner than previously, they still need to carry the same amount of blood. Because the heart has to use more effort to push blood through narrower blood arteries. Cocaine suppress myocardial contractility, reduce coronary blood flow and causes electrical abnormalities in the heart. These effects will lead (fig: 1) to decrease myocardial oxygen supply and may increase demand. Thus, myocardial ischemia and/or infarction may occur. While cocaine also causes increase in heart rate by unbalancing the catecholamine level in the body. Cocaine stimulates the sympathetic nervous system by inhibiting catecholamine reuptake at sympathetic nerve terminals and stimulating

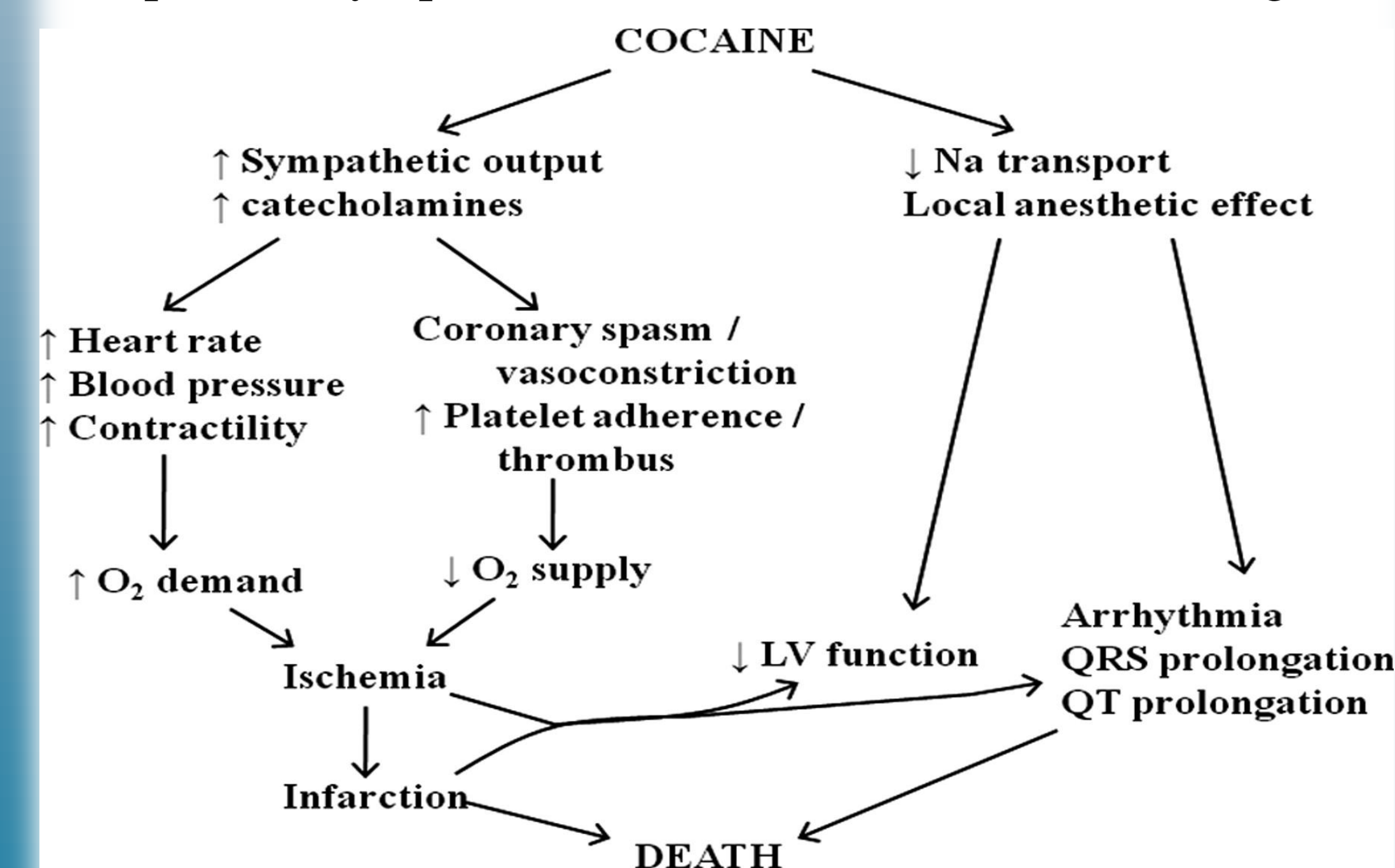


Fig: 1 complications of the cocaine.

central sympathetic outflow, and increasing heart rate. Norepinephrine are necessary for fight-or-flight response and causes increase in heart rate.

Conclusion:

Cocaine causes increase in the blood pressure and heart rate by potent sympathomimetic properties and arterial vasoconstriction due to catecholamine reuptake unbalancing in the body.



Reference:

1. Kozor R, Grieve SM, Buchholz S, et al. Regular cocaine use is associated with Increased systolic blood pressure, aortic stiffness and left ventricular mass in young otherwise healthy individuals. *PLoS One*. 2014;9(4):e89710. Published 2014 Apr 9. doi:10.1371/journal.pone.0089710
2. Cardiovascular Effects of Cocaine. *Circulation*. [https://www.ahajournals.org/doi/full/10.1161/circulationaha.110.940569#:~:text=Cocaine%20affects%20the%20cardiovascular%20system,which%20increase%20myocardial%20oxygen%20demand](https://www.ahajournals.org/doi/full/10.1161/circulationaha.110.940569#:~:text=Cocaine%20affects%20the%20cardiovascular%20system,which%20increase%20myocardial%20oxygen%20demand.). Published 2022.
3. Vongpatanasin W, Taylor J, Victor R. Effects of cocaine on heart rate variability in healthy subjects. *Am J Cardiol*. 2004;93(3):385-388. doi:10.1016/j.amjcard.2003.10.028