Nicotine is a major tobacco smoke constituent that directly and/or indirectly facilitates the initiation of atrial and ventricular tachycardia and fibrillation (AT/AF and VT/VF respectively) in hearts with preexisting or evolving myocardial infarction. Acute nicotine promotes reentrant VF only in ventricles with electrical and structural remodeling. Similar concentrations of nicotine, which are also seen in the plasma of regular smokers, fail to promote conduction block and reentrant VF in normal canine hearts. Nicotine’s pro-VF mechanism may be related to its ability to steepen the slope of ventricular action potential duration restitution curve. Similarly, acute cigarette smoke inhalation is shown to cause greater reduction in VF threshold in canine hearts with acute ventricular ischemia than in normal hearts with no myocardial infarction. Chronic nicotine administration in dogs with an evolving isolated left ventricular myocardial infarction amplifies the induction of atrial fibrosis compared to atria of dogs with normal hearts leading to isthmus-based atrial flutter. Smoking cessation causes greater reduction in arrhythmic death in patients with advanced ischemic heart disease compared to patients without these advanced complications. It is concluded that both acute and chronic nicotine adversely affect the evolution of an existing cardiac disease by promoting potentially lethal cardiac arrhythmias.

Keywords: nicotine, smoking, cardiac arrhythmias