HORT COMMUNICATION

EFFECT OF SMOKING ON ELECTROCARDIOGRAM AND BLOOD PRESSURE

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Summary: Smoking was found to casuse a significant increase in heart rate and blood pressure (both systolic and diastolic) but not appreciable changes in E.C.G. wave forms.

Key words: smoking E.C.G. blood pressure

INTRODUCTION

Smoking of tobacco has a positive effect on heart rate and blood pressure and causes cardiographic alterations. Roth *et al.* (7) in their study on 66 subjects found that after smoking two standard cigarettes there was an increase in heart rate and blood pressure and decrease in amplitude of QRS complex. Cryer *et al.* (2) also found an increase in heart rate and blood pressure on cigarette smoking. There appears to be a correlation between smoking and atherosclerosis (1). However, the extent of damage to the cardiosvascular system remains controversial Lefkowitz (6) has recommended the use of more eloborate electrocardiographic monitoring in such investigations. The present study was undertaken in the light of these recommendations.

MATERIALS AND METHODS

Following a 30 minute rest, the blood pressure and E.C.G. (lead II) was measured in 20 normal healthy habitual smokers (males only) of 20-45 years of age. Each subject was directed to smoke and inhale two standard sized cigarettes and recording of blood pressure and E.C.G. was repeated immediately after the last puff.

RESULTS AND DISCUSSION

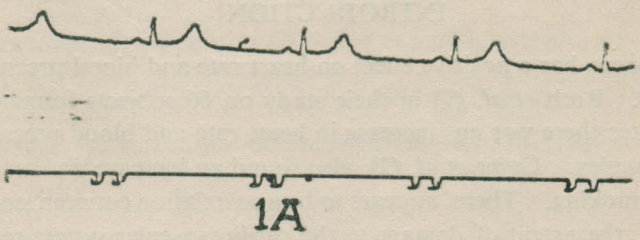
After smoking there was a significant increase in heart rate and both systolic and diastolic pressure ($P > 0.05$). There was an apparent decrease in duration of PR interval, T wave and amplitude of QRS but slight increase in duration of QRS complex and amplitude of T wave ($P > 0.05$) (Table I and Fig 1 A and Fig. 1 AB). QRS complex in few exceptions exhibited an increase in amplitude though mean value showed a decrease.

These findings are generally consistent with the findings of other workers who have also recorded a similar increase in blood pressure, heart rate and change in E.C.G. Cryer *et al.* (2) have shown that smoking increased the pulse rate from 72 ± 3/min to 86 ± 3/min and systolic and

TABLE I: Mean heart rate. B. P. and E.C.G. changes before and after smoking.

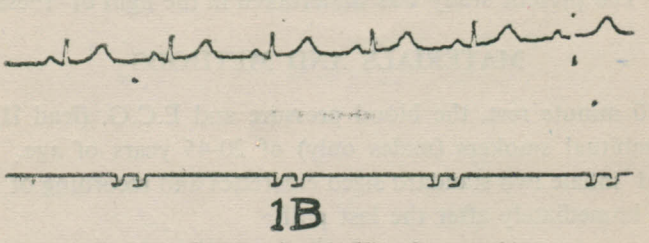
	Heart rate per minute	*Blood pressure*		*Duration in seconds*			*Amplitude (mm)*	
		Systolic (mmHg)	*Diastolic (mmHg)*	*PR*	*QRS*	T	*QRS*	T
Before smoking	66±8	121±10	77±5	0.162± 0.028	0.063± 0.016	0.17± 0.025	11.5± 5.8	4.0± 1.77
After smoking	73±97	128±13	82±5	0.155± 0.082	0.064± .07	0.169± 0.019	11.05± 4.52	4.065± 1.79
P	* <0.05	* <0.05	* <0.05	>0.05	>0.05	>0.05	>0.05	>0.05

*Statistically significant
± S.D.



1A

Fig. 1A: E.C G. (Lead II) before smoking.



1B

Fig. 1B: E.C G. (Lead II) after smoking.

diastolic blood pressure from 108±4 mmHg to 114±4 mmHg and from 67±4 mmHg to 75±4 mmHg respectively. Grabiel *et al.* (4) found an increase in heart rate and blood pressure in 80% and a dimunition of QRS, T and PR interval in 50% of 45 subjects studied. Several constituents of cigarette smoking have been implicated as having a causal effect on pathogenesis of various diseases. The two major ones are carbon monoxide and nicotine. Carbon monoxide promotes development of atherosclerosis like lesion in vessels (6) and nicotine causes stimulation of sympathetic nervous system and chemoreceptors, leading to vasoconstriction, tacy-

cardia and elevation of blood pressure (3). Cryer *et al.* (2) have attributed their findings to the release of sympathetic neurotransmitters (Norepinephrine) as well as adrenomedulary hormone (Epinephrine). The increase of these neurohumoral transmitters have actually been found by these workers.

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