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To assess effect of smoking on cerebral blood flow

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ABSTRACT

Background

Smoking is known to interfere with NO mediated endothelial function therefore impairing the cerebrovascular blood flow (CBF).Cigarette smokers also have raised fibrinogen levels and platelet count which makes the blood stickier. All these factors make smokers more vulnerable to cerebrovascular diseases.

Purpose of the Study

The purpose of the present study was to determine whether the level of cigarette use is a significant predictor CBF when age, gender, is controlled. The objective of the study was to assess and compare cerebral blood flow in smokers and non- smokers and to correlate the alterations in cerebral blood flow with the no. of cigarettes and duration of smoking.

Methods

The subjects consisted of 50 smokers and 50 non-smokers without any history of respiratory disease, cardiac disease or any other chronic disease. Smoking index was calculated and Mean cerebral blood flow (CBF) in Anterior (ACA), Middle (MCA) & Posterior (PCA) cerebral arteries were measured by the Trans-cranial Doppler method (TCD) method.

Results

Subjects had an average smoking index of (number of cigarette smoked per day) x year of smoking history) 450 +_ 164. CBF was measured using trans-cranial Doppler method. Simple linear regression analysis demonstrated a negative correlation of smoking index with CBF in all the major cerebral arteries (LMCA, r = -0.119), (RMCA, r = -0.34), (LACA, r = -0.36),(RACA, r = -0.34),(LPCA, r = -0.33) and (RPCA, r= .000).

Keywords: Smoking, Cerebral blood flow, Smoking index

INTRODUCTION

Endothelial dysfunction is well known to alter the blood flow and is a cause for much vascular pathology. It is influenced by many factors and smoking is one of them.

The vascular endothelial cells control the contractile state of blood vessels by release of

various substances (e.g. NO) [1,2,3]. Smoking is known to interfere with NO mediated endothelial function therefore impairing the cerebrovascular blood flow (CBF) [4,5].

Cigarette smokers also have raised fibrinogen levels and platelet count which makes the blood stickier [6].

All these factors make smokers more vulnerable to cerebrovascular diseases.

In the largest American prospective study age specific death rate for CVD (cerebrovascular disease) was 1.4 times higher in 55 to 64 year old male smokers than in non-smokers [7, 8].

Epidemiological Reports have also shown that cigarette smoking increases the risk of cerebrovascular disease (CVD).

The Framingham study and a large scale Japanese study also showed an excess risk of CVD in smokers [8, 9].

Wechsler concluded that cigarette smoking does not change CBF. Conflicting reports proposed that smoking as well as nicotine injections increase CBF [10].

In all of these earlier studies however, CBF was measured before and just after smoking and these results described only the acute effects of smoking.

Since these reports conflicted with the fact that cigarettes are a risk factor for CVD, [11, 12] the following study suspected that the acute pharmacological effects and chronic or long term effects of smoking on CBF were quite different. The present study, therefore, was done to study the chronic effects of smoking on CBF.

MATERIALS AND METHODOLOGY

The present cross sectional study was conducted In the Dept. of Physiology, BPS GMC for Women, Khanpur Kalan, in collaboration with Central Research Laboratory. Cerebral blood flow (CBF) in Anterior (ACA), Middle (MCA) & Posterior (PCA) cerebral arteries were measured by the Trans-cranial Doppler method (TCD) method. The study was carried out with subjects sitting on a chair with closed eyes in a quiet room.

Subjects

A total of hundred adult subjects above the age of 18 years, coming to hospital either for their own treatment or their relatives and the supporting staff were studied by measuring CBF. The subjects consisted of 50 smokers and 50 nonsmokers. Smoking index was calculated [Smoke Index = (Cigarettes smoked per day) X (year smoking history)] .In nonsmoking group consisting of 50 subjects, who had never smoked; therefore the mean smoking index was 00. In smokers the mean smoking index was 450 ± 164. The mean age of smokers & nonsmokers was identical. A detailed history of each subject was taken on a proforma.

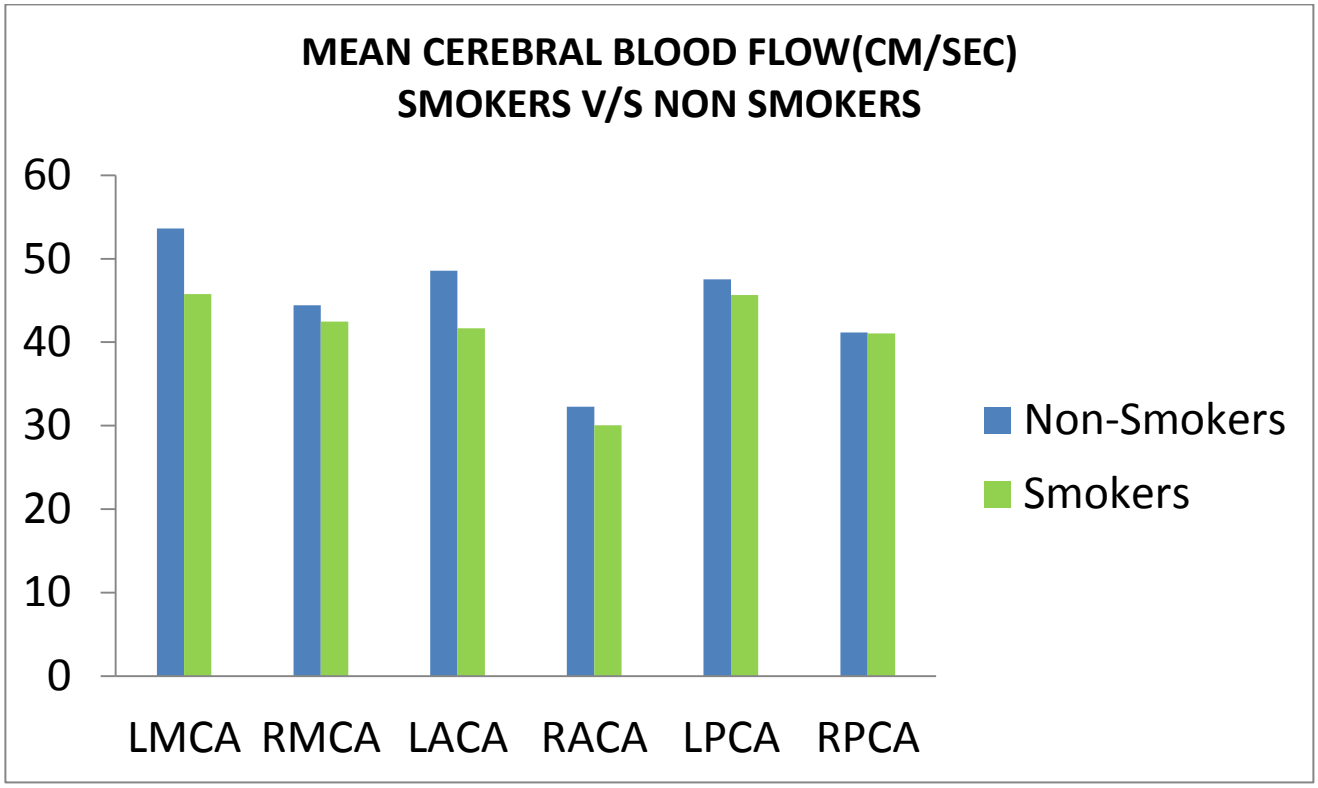
Normal healthy subjects without any history of cardiovascular, neurological, pulmonary dysfunction, acute and chronic illness were included in the study. Informed consent was taken from all cases and control subjects.

OBSERVATIONS AND RESULTS

Table-1: Mean Cerebral Blood Flow (Cm/Second) (Smokers V/S Non-Smokers)

S. No.	Cerebral Arteries	Non-smokers (n=50) (Mean ±S.D)	Smokers (n=50) (Mean ±S.D)	p-value
1.	LMCA	53.64 ± 11.467	45.75 ± 11.139	.001
2.	RMCA	44.44 ± 9.856	42.48 ± 10.723	.344
3.	LACA	48.56 ± 9.900	41.66 ± 10.143	.001
4.	RACA	32.26 ± 12.000	30.04 ± 14.165	.400
5.	LPCA	47.52 ± 13.637	45.64 ± 12.618	.476
6.	RPCA	41.18 ± 11.115	41.04 ± 11.308	.950

(LMCA-left middle cerebral artery; RMCA-right middle cerebral artery;
LACA-left anterior cerebral artery; RACA-right anterior cerebral artery;
LPCA-left posterior cerebral artery; RPCA-right posterior cerebral artery)



(LMCA-left middle cerebral artery; RMCA-right middle cerebral artery; LACA-left anterior cerebral artery; RACA-right anterior cerebral artery; LPCA-left posterior cerebral artery; RPCA-right posterior cerebral artery)

Table-2: correlation between smoking index and mean cerebral blood flow

Smoking Index	Pearson correlation (r)	LMCA	RMCA	LACA	RACA	LPCA	RPCA
		-.119	-.155	-.074	-.021	-.227	.000
	p-value	.411	.284	.609	.887	.113	.978
	N	50	50	50	50	50	50

TABLE-1 Shows distribution of mean cerebral blood flow (cm/sec.) in smokers & nonsmokers. CBF of each group was compared. CBF values of smokers were lower as compared to non-smokers.

There was significant difference in mean CBF of Left Middle and Anterior Cerebral artery (LMCA & LACA) of smokers as compared to nonsmokers.

TABLE-2 Present the correlation between mean CBF and Smoking index. The correlation was negative in all the vessels.

DISCUSSION

The cerebral mean artery blood flow measured in smokers was found to be significantly lower when compared to the control subjects.

It was also found that smoking index was negatively correlated to mean cerebral blood flow. It was found that people with higher smoking index show lower Cerebral Mean blood flow than those with lower smoking index.

CBF is mainly regulated by nitrergic (post-sympathetic, postganglionic) nerves and NO liberated from endothelial cells in response to sheer stress and stretch of vasculature. Cigarette smoking is well known to affect endothelial function and to degrade NO by producing oxygen radicals.

Nicotine acutely dilates cerebral vasculature in humans and experimental animals via release of NO from perivascular nitrergic nerves [13, 14].

Chronic exposure to nicotine elicits blunted NO induced vasodilatation in pial arteries and increased cortical blood flow. It also increases cerebral hypo-perfusion via degradation of NO by increasing oxidative stress [13, 14].

There is evidence that smoking impairs NO synthesis via endothelial nitric oxide synthase and neuronal endothelial oxide synthase (eNOS/nNOS) inhibition in cerebral vascular endothelial cells and nitrergic nerves leading to interference with cerebral blood flow [14,15,16,17,18].

Therefore smoking mediated chronic impairment in synthesis and action of NO may lead to alteration in CBF.

CONCLUSION

Cerebral blood flow is regulated by nitergic nerves and Nitric oxide released from the endothelial cells. Cigarette smoking impairs synthesis of NO by inhibiting endothelial nitric oxide synthase and neuronal nitric oxide synthase and increased production of oxygen radicles. Objective of this study was to compare cerebral blood flow in smokers and non-smokers and to see its correlation with smoking. It was found that CBF was lower in smokers than in Non-smokers. It also decreased proportionately with increased smoking index.

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