Indian Journal of Basic and Applied Medical Research; March 2018, Vol.-7, Issue- 2, P. 641-646

Original research article

Evaluation of carbon monoxide in breath and carboxyhemoglobin percentage in young smokers

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Abstract:

INTRODUCTION: Cigarette smoking is the main cause of chronic bronchitis, emphysema and bronchial carcinoma. Carbon monoxide (CO) is produced by incomplete combustion of carbon containing material, notably tobacco, Biomass fuel and fossil fuel. CO present in cigarette smoke, when Inhaled binds to hemoglobin after displacing O_2 with high affinity to form carboxyhemoglobin, leading to serious compromise of O2 transport. COHb is a sensitive physiogial marker of atmospheric CO from both indoor and outdoor sources. CO concentration in breath is considered a reliable indicator of COHb percentage in the blood So breath analysis done COHb in the blood.

MATERIALS AND METHODS: 60 clinically healthy male individuals between the age group of 18-35 years and divided into control and study group. Control group includes 30 non smokers and study group includes 30 smokers of the same age distribution. Exclusion criteria includes Females, age less than 18 yrs more than 35 years, any respiratory & cardio vascular disorders, any ENT diseases

RESULTS: The mean values of the exhaled air CO between the non-smokers and the smokers were compared. Smokers(6.4 ± 2.78) have statistically significant increase in exhaled air CO than non smokers(1.26 ± 0.44) (p value approx 0.00).

CONCLUSION: This study has revealed a highly significant increase in COHb% in smokers than non smokers that cigarette smoking invariably increase the COHb% level even in smokers with less smoking index and thereby it impairs the O 2 delivery property of Hb, hence active smoking, passive smoking should be avoided at any cost.

INTRODUCTION

Cigarette smoking has deleterious effects on the respiratory system and this is clearly implicated in the aetiology of number of diseases of respiratory system which mainly include chronic bronchitis, emphysema and bronchial carcinoma (WHO, 1993). Overall life expectancy is also considered to be significantly decreased from 10 to 17.9 years (WHO, 2008). Carbon monoxide (CO) is produced by incomplete combustion of carbon containing material, notably tobacco, Biomass fuel and fossil fuel. Carbon monoxide exposure occurs mainly through cigarette smoke though small amount also occurs through vehicle emission (Deller et al, 1992). CO present in cigarette smoke, when Inhaled binds to hemoglobin after displacing O_2 with high affinity (200 times greater than for oxygen) to form carboxyhemoglobin (COHb), leading to serious compromise of O2 transport. The COHb complex is very stable additionally rendering a proportion of Hb unavailable for O_2 transport. It also causes O_2 dissociation curve to be shifted to the left, making even the functional Hb give up less O_2 to the tissues at any given pressure. Level of CO in the body is determined by ambient CO concentrations, alveolar ventilation, lung diffusibility, total hemoglobin

mass, and COHb level (IPCS). COHb is a sensitive physioogial marker of atmospheric CO from both indoor and outdoor sources. CO concentration in breath is considered a reliable indicator of COHb percentage in the blood (Jarvis MJ, 1908). So breath analysis being non invasive, easy procedure, is preferred for measurement of COHb in the blood (Verhoeff AP 1983).

(Maloney G)Expired CO has attracted some attention as a possible biomarker to encourage smoking cessation[Beard. E, et alMeredith SE etal] but requires specialized measuring equipment such as an electrochemical analyzer . Hence the present study was taken up in young smokers to estimate and compare the COHb % between smokers and non smokers by measuring exhaled air carbon monoxide in the sample group.

MATERIALS AND METHODS

This cross sectional study was conducted after obtaining approval from Institutional Ethical Committee (IEC) and a written informed consent from the individuals participating in the study. A scientifically adjusted sample size was taken from the general population with 60 clinically healthy male individuals between the age group of 18-35 years and divided into control and study group. Control group includes 30 non smokers and study group includes 30 smokers of the same age distribution. Exclusion criteria includes Females, age less than 18 yrs more than 35 years, any respiratory & cardio vascular disorders, any ENT diseases and neurological diseases,

Exhaled air CO	Non smoker	Smoker	P value
Mean	1.26	6.4	0.00*
SD	0.44	2.78	

Any metabolic and endocrinal diseases. COHb% was measured by the levels of CO in ppm in single exhaled breath sample using "CO Check + " CO breath analyzer.). The subject were instructed to blow as a single continuous, sustained, slow exhalation through the mouth piece of "CO check+ "machine until a beep sound was heard from the machine. Both exhaled air CO level and corresponding COHb% were displayed at the end of the maneuver and the values were noted. Breath CO monitors function based on the detection of carbon monoxide gas with an electrochemical gas sensor. Monitors incorporate an LCD display that provides a concentration level in a numeric format that correspond to various concentration ranges.

RESULTS AND ANALYSIS

The data obtained were statistically analyzed using SPSS 17.0 software and $p<0.05$ is considered significant. Data were expressed as mean ± standard deviation.

The mean values of the exhaled air CO between the non-smokers and the smokers were compared. Smokers(6.4 ± 2.78) have statistically significant increase in exhaled air CO than non smokers(1.26 ± 0.44) (p value approx 0.00).

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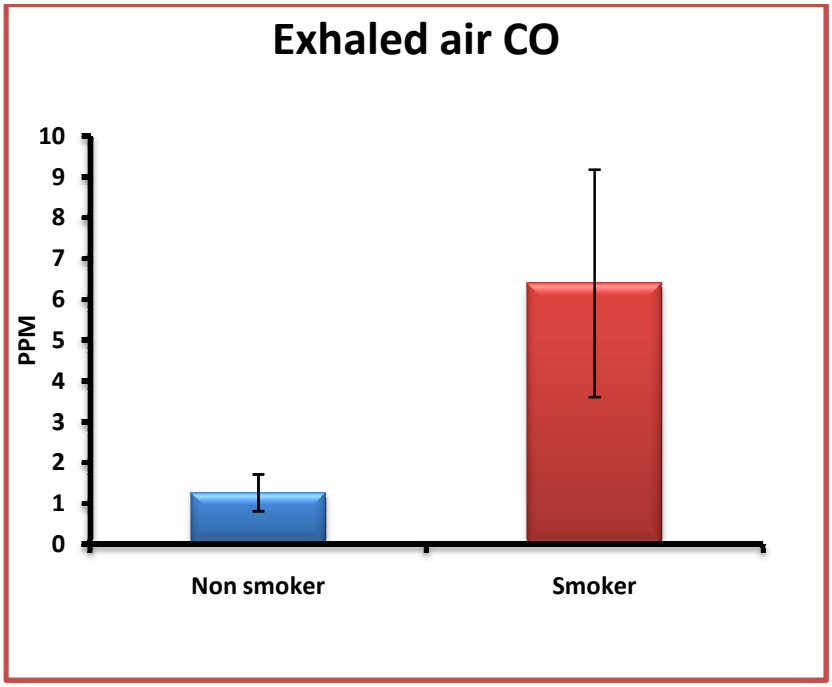
COMPARISON OF MEAN VALUES OF EXHALED AIR CO BETWEEN SMOKERS AND NON SMOKERS

*p value < 0.05 -Significant

Comparison of mean values of exhaled air CO between smokers and non smokers



COHb%

The mean values of the COHb % between the non-smokers and the smokers were compared. Smokers(1.03 ± 0.44) have statistically significant increase in COHb % than non smokers(0.21± 0.03) (p value approx 0.00)

COMPARISON OF MEAN VALUES OF COHb% BETWEEN SMOKERS AND NON SMOKERS.

*p value < 0.05 – Significant

GRAPH

COHb%	Non smoker	Smoker	P value
Mean	0.21	1.03	0.00*
SD	0.03	0.44	

Comparison of mean values of COHb % between smokers and Non smokers

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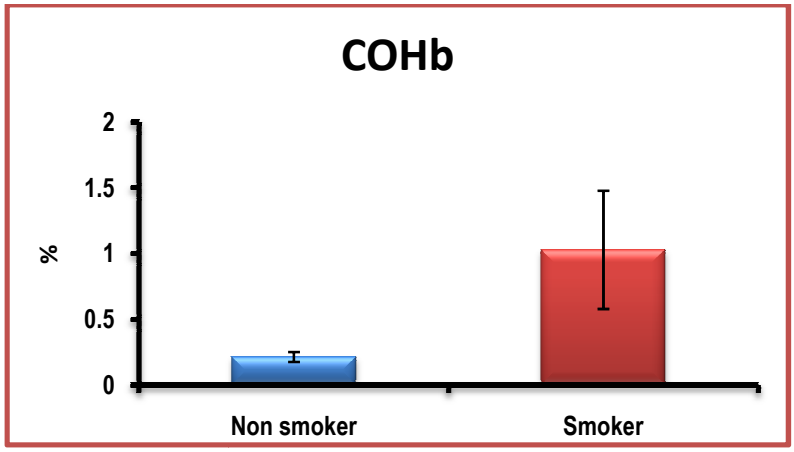

DISCUSSION

The mean value of COHb% in smokers was 1.03 ± 0.44 %. These values were significantly higher than the mean value obtained in non smokers. This is substantiated by Light, et al., 2007, who observed that the average value of COHb % of the 33 smokers was 5.04%, while the average value for the 27 non-smokers in a smoking environment was 2.49%. COHb levels ranged from 1-6 % in the non-smokers and 1-14% in the smoking group. Castleden, C. M. and Cole, P. V,1975 also studied the levels of COHb% in smokers and non smokers. They found that the COHb% levels were higher in smokers(mean 5-8 %) when compared to non-smokers (mean 1-3 %). Cohen SI,et al.,1971, Aronow WS et al.,1971, also showed that all types of cigarettes increased the COHb%. Tobacco smoke contains a mix of 7000 chemicals ; Hundreds are harmful , about 70 can cause cancer (13,12,11). Smoking increase the risk for serious health problems , diseases and death (13,12).

People who stop smoking greatly reduce their risk for diseases and early death. Although the health benefits are greater for people who stop at earlier ages, there are benefits at any age (13,12,14,15) Stopping smoking is associated with following health benefits (13,12, 14,15)

- Lowered risk for lung cancer and many other types of cancer
- Reduced risk of Atherosclerosis and its consequences such as Stroke, Myocardial infarction and Peripheral vascular disease
- Reduced Heart disease risk within 1- 2 years of quitting
- Reduced Respiratory symptoms , Such as Coughing, Wheezing and dyspnoea .Sometimes these symptoms may not disappear , but they do not continue to progress at the same rate among people who compared with those who continue to smoke.
- Reduced risk of developing some lung diseases, such as chronic obstructive lung disease (COPD)
- Reduced risk for Infertility in women of childbearing age. Women who stop smoking during pregnancy also reduce their risk of having a low birth weight baby.

This study has revealed a highly significant increase in COHb% in smokers though they were not symptomatic. It has been documented that cessation of smoking at any age can slow down the decline in lung functions and improves respiratory symptoms (mahajan et al.,1983, gupta, et al., 1975).

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The mean level of exhaled carbon monoxide in active smokers was 12.57 ppm with higher levels found in men. The highest mean level of eCO was found in participants with body weight between 60 kg and 80 kg (mean eCO = 13.39 ppm). The highest levels were observed in participants living in towns with 51,000-100,000 inhabitants. In passive smokers, the mean level of exhaled carbon monoxide was 3.55 +/- 1.26 ppm with higher levels found in men. 1. Concentration of carbon monxide in air and increase of blood carboxy-hemoglobin level(% COHb) were positively correlated with smoking amount. 2. Increase of blood carboxy-hemoglobin in passive smokers, in average, were about seventy six percent of that in smokers, as 2.2% vs. 2.9%. 3. Comparison with published data showed that Peterson's equation gave most similar result to this study in estimation of increase of blood carboxy-hemoglobin level. 4. During the exposure, flicker values fell steadily in both experimental groups and control groups as time passed. Flicker value were, however, elevated again in experimental groups after exposure, despite the fact that values still fell in control groups. 5.

After 30 minutes exposure to air with high levels of tobacco smoke (no other details) increased levels of carbon monoxide in the exhaled air and carboxyhaemoglobin in blood were found in non-smokers (California EPA 1997).

In non-smokers exposed to tobacco smoke in closed rooms, the carboxyhaemoglobin levels in blood reached 2 % to 3 % (Adlkofer 1992, California EPA 1997, Scherer et al. 1992). The endogenous carbon monoxide production (Coburn et al. 1966) results in carboxyhaemoglobin levels of 0.4 % to 0.8 % in healthy non-smokers, whereby the inter- individual differences can be enormous (Werner 1978) . -smokers (number not specified) were exposed for 8 hours to various concentrations of tobacco smoke; in the first experiment the carbon monoxide concentration was 10 ml/m 3and the particle concentration 1.0–1.5 mg/m 3, in the second the carbon monoxide concentration was 25 ml/m 3 and the particle concentration 3.0 to 4.0 mg/m 3 .

In the second experiment, smokers who smoked 20 cigarettes during the study were also included for comparison. The level of thioether excretion in the urine of passive smokers was about 44 % of that excreted by smokers. Determined from the thioether excretion in the urine, the non-smokers had “smoked” 9 cigarettes (Adlkofer 1992).

In another study in which 5 smokers each smoked 24 cigarettes during 8

CONCLUSION

This study has revealed a highly significant increase in COHb% in smokers than non smokers that cigarette smoking invariably increase the COHb% level even in smokers with less smoking index and thereby it impairs the O 2 delivery property of Hb.

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