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**Pulmonology**

# Measurement of Exhaled CO level in Active, Passive & Non-Smokers

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| Abstract | Original Research Article |
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Cigarette smoking is the single most preventable cause of morbidity and mortality, causing five million deaths worldwide each year. Exhaled carbon monoxide (CO) is a biological indicator to assess smoking status. Turn the CO Check ON by pressing by ON/OFF Power key. The subject should be encouraged to hold the breath for 15 seconds (CO Check pro). Press the select function key and the display will show the blow icon. Alternately, set the hold breath timer to desired number of seconds. The unit will count down the period during which the subject should hold the breath. When the period expires, the unit will display the blow icon. Then the carbon check pro is given exhaled CO levels and then calculate CO-HB percentage. CO PRO Check levels were assessed in a total of 205 subjects; 73 of them were active smokers 63 of them was passive smokers (62men; 1 woman) and 69 of them was non- smokers (28 men; 41 women). Active smokers were 35.6%, Passive Were 30.7% and non-smokers were 33.65%. The active smokers, the mean daily consumption of cigarettes was 5.458cigarettes/day. The mean exhaled CO level was 15.47ppm for healthy smokers and 1.65ppm for non-smokers and 4.5ppm for passive smokers. The mean exhaled CO level was significantly higher in healthy smokers than passive smokers and non-smokers. Our results shown that the optimal cut-off was 4.2ppm, giving 94% sensitivity and 85% specificity. There is a direct relationship between the smoking status of a given individual and the concentration of carboxyhaemoglobin (CO-Hb) in their blood. The estimation of (CO-Hb) is used to estimate the patient's health status. The CO levels to ensure the patients' health condition to wards CO poisoning risks, decreased morbidity and mortality due to smoking and CO poisoning.
**Keywords:** Carboxyhaemoglobin, Exhaled carbon monoxide, Non-smokers, Passive smokers, Smoking status.

## INTERDUCTION

Carbon monoxide is a colourless, practically odourless and tasteless gas that is poorly soluble in water, but it is soluble in alcohol and benzene. It is a product of incomplete combustion of carbon-containing fuels. Carbon monoxide burns with a violet flame and it is classified as an inorganic compound. It has a slightly lower density than air [1]. Cigarette smoking is the single most preventable cause of morbidity and mortality, causing five million deaths worldwide each year [2]. A systematic study of biological, behavioural, and environmental factors is necessary to identify specific patterns of increased disease risks among various subgroups of smokers [3]. Exhaled carbon monoxide (CO) is a biological indicator to assess smoking status [4, 5]. Exhaled CO is also considered as a biomarker of some pulmonary diseases like asthma, chronic obstructive pulmonary disease, primary ciliary dyskinesia, cystic fibrosis and bronchiectasis [6, 7]. It is

suggested that exposure to CO can induce myocardial ischemia in subjects with coronary artery disease [7]. Exposure to CO leads to various health effects through affecting cardiovascular system, lungs, and blood and central nervous systems depending on health and physiological status of exposed person, pollutant concentration, and exposure time. One of the important outcomes of exposure to CO is reaction with blood haemoglobin molecules to make carboxyhaemoglobin (CO-Hb); reducing oxygen supply to brain and other body organs. CO-Hb concentration in blood has been utilized as an indicator for health consequences of exposure to CO and various symptoms have been linked to different concentrations of CO-Hb in blood (CO-Hb%). In general, signs and symptoms of acute CO poisoning appears at CO-Hb concentrations ranging from 3 to 24%. It is stated that the symptoms of exposure to CO appears in CO-Hb% more than 3 percent in blood [8]. The main mechanism by which CO causes heart disease is production of hypoxia. The

effects of CO are more profound in the myocardium than in peripheral tissues because of very high oxygen extraction by the myocardium at rest [9, 10]. Carbon monoxide may also have direct myocardial effects. In isolated rat hearts, CO caused a greater decrease in heart rate and pulse pressure compared to the same degree of anoxia produced by the inhalation of nitrogen [11]. Central nervous system (CNS) effects in individuals suffering acute CO poisoning cover a wide range, depending on severity of exposure: headache, dizziness, weakness, nausea, vomiting, disorientation, confusion, collapse, and coma [1]. In practice, measuring the concentration of CO in the exhaled air, is noninvasive, cheap, quick, portable, and does not require special technical background. The aim of this study was to compare the breath CO levels in established smokers, Passive smokers and non-smokers and to investigate factors that may affect breath CO levels by measuring exhaled CO levels. Finally, all subjects were asked to provide one breath into CO PRO CHEK meter because of a previous study has been reported the first reading to be significantly higher than the second.

## METIRIALS & METHOD:

It is An Institutional based Prospective observational study. Conducted at Govt.CD & TB hospital, Bheemaram, Hanamkonda. Two hundred- and five-members subjects were included the study. The subjects were informed of the purpose of the CO PRO CHEK meter and were reassured that the results were confidential in order to encourage accurate reporting of smoking habits. Background information about their health, age, gender, smoking habits, occupational state and passive smoke was obtained. An active smoker was defined as a person who currently smoked at least one cigarette a day. Passive smoking was defined as they had never smoked cigarettes or Exposure to environmental tobacco smoke (ETS) was ascertained using data derived from the same questions asked to the subjects. A person was deemed to have been exposed to ETS if a household member had regularly smoked cigarettes in their presence or if a co-worker smoked in the same indoor room in their presence for more than one year during the past 10 years. Finally, all subjects were asked to provide one breath into CO PRO CHEK meter.

## PATIENT'S SELECTION
### INCLUSION CRITERIA
* 18 years above
* smokers every day
* smoked above 1 year

### EXCLUSION CRITERIA
* <18 years
* quit smoking
* Pregnancy women

**CO PRO CHEK METER**

Insert the 9V PP3 battery (#3 supplied) by removing the battery cover and clipping the battery in place, and then replace the battery cover. Insert the safe Breath filtered cardboard mouthpiece (#4).

Turn the CO Check ON by pressing by ON/OFF Power key. The device will display the version number before starting the countdown timer. The unit will countdown for 10 seconds to ensure that the sensor is stabilised. When the countdown timer reaches zero it will display the current environment reading if enabled.

If the environmental monitor is disabled, it will zero to ambient air automatically. The subject should be encouraged to hold the breath for 15 seconds (CO Check pro) or 10 secs (CO Check Baby). Press the select function key and the display will show the blow icon.

Alternately, set the hold breath timer to desired number of seconds. The unit will count down the period during which the subject should hold the breath. When the period expires, the unit will display the blow icon.

If the environment reading is disabled, the original countdown will be based on hold breath setting, provided it is more than 10 seconds.

The subject should place their lips around the carboard mouth piece and blow gently and continue blowing until their lungs are completely empty. CO is collected in the last portion of the breath (alveolar breath).

**DATA ANALYSIS**

The collected data was analyzed primarily by using MS-EXCEL 2010.

## STATISTICAL ANALYSIS

All statistical analyses were done using Graph Pad Prism (version-7). Results were expressed as mean+SD. ANOVA was used to compare all exhaled CO levels between groups. A value of P less than 0.05 was considered significant. Spearman correlation analyses were used to evaluate the relationship between the exhaled CO levels-daily cigarette consumption, and CO levels-Exhaled CO-Hb% in Active smokers.

## RESULTS

CO PRO Check levels were assessed in a total of 205 subjects; 73 of them were active smokers 63 of them was passive smokers (62men; 1 woman) and 69 of them was non- smokers (28 men; 41 women)

**Table-1: Age wise distribution of subjects in the study**

| AGE | SUBJECTS | PERCENTAGE |
|-----|----------|------------|
| 15-20 | 15 | 7.3% |
| 21-25 | 80 | 39% |
| 26-30 | 61 | 29.7% |
| 31-35 | 18 | 8.7% |
| 36-40 | 11 | 5.3% |
| 41-45 | 4 | 1.9% |
| 46-50 | 5 | 2.4% |
| 51-55 | 6 | 2.9% |
| 56-60 | 1 | 0.48% |
| 61-65 | 2 | 0.97% |
| 66-70 | 2 | 0.97% |

Active smokers were 35.6%, Passive Were30.7% and non-smokers were 33.65%. Active, passive and non-smokers distribution

**Table-2**

| Smoking | No. of subjects | Percent |
|---------|-----------------|---------|
| Active | 73 | 35.6% |
| Passive | 63 | 30.7% |
| Non-Smoker | 69 | 33.65% |

The mean age of active smokers 27.27, SD is 10.18 and SE of mean is 1.997. The Mean age of passive smokers 30.84, SD is 9.9 and SE of mean is 1.25. The mean Age of non-smokers 27.75 and SD is 6.363 and SE of mean is 0.766. The active smokers, the mean daily consumption of cigarettes were 5.458cigarettes/day. SD 3.12 Standard error of mean 0.36 and lower limit of mean is4.725, upper limit of mean 6.192. All of them reported that they had smoked on the day of testing. The mean exhaled CO level was 15.47ppm for healthy smokers and 1.65ppm for non-smokers and 4.5ppm for passive smokers. The mean exhaled CO level was significantly higher in healthy smokers than passive smokers and non-smokers. The mean exhaled CO level was higher in passive smokers than non-smokers. There was a significant positive correlation between CO levels and daily cigarette consumption, and CO levels and Exhaled CO-Hb% in active smokers (r =0.0665, p&lt; 0.0001).

## DISCUSSION

Nicotine, cotinine, or thiocyanate levels in the plasma or urine may be used to indicate smoking status [12]. However, the blood tests are invasive and neither the blood nor the urine tests provide an immediate assessment. The measurement of breath CO level may provide an immediate, non-invasive method of assessing smoking status [13]. This study supports that measuring breath CO levels provides an immediate, non-invasive, simple, and effective way of confirming a patients' smoking status.

Jarvis *et al*. shown that the second breath CO level was significantly higher than the first. In contrast Middleton and Moricereported that the first breath CO level was significantly higher than the second and they recommended that a single Smokerlyser assessment should usually be sufficient, provided that there is adequate technique. For this reason, in present study exhaled CO levels were assessment with the first measurement [14]. In a previous study it was stated that exhaled CO may be affected by ambient CO and that this influence may be reduced by subtracting ambient CO from exhaled CO [15]. In contrast, Zetterquist *et al*.'s [5] study shown that ambient air did not affect the exhaled CO levels when subjects held their breath for 10s. Since there seems to be no contribution of CO from the conducting airways it must have its origin from the alveoli. The increase in CO concentrations after breath-hold also supports this view. The inhaled CO concentration may affect the concentration gradient of CO over the alveolar membranes (and possibly in the airways), and should not be compensated for by direct subtraction. A standardised time of breath-hold of 15s was used in all the experiments which should have been sufficient for equilibrium to take place [5]. Since we also ask to the subjects to hold their breath for 15s, we did not consider the impact of ambient air.

In our study CO PRO Check levels were assessed in a total of 205 subject; 73 of them was active smokers 63 of them was passive smokers (62men; 1 women) and 69 of them was non- smokers (28 men; 41 women). And Active smokers was 35.6%, Passive was 30.7% and non-smokers was 33.65%.

Our results shown that there was a significantly positive correlation between daily consumption of cigarettes, co levels and CO-Hb% in active smokers. Likewise, Gonzalez *et al*. reported that CO in expired air correlated significantly with the number of smoked cigarettes. Smokers who develop chronic obstructive pulmonary disease, besides consuming a greater number of cigarettes, smoke them with a particular inhalation pattern (they inhale a greater volume of smoke and more deeply), thus permitting a higher quantity of oxidant substances [15]. Cunnington *et al*. [16]. demonstrated that the mean breath CO concentrations increased in direct proportion to the number of cigarettes smoked.

Exhaled CO levels were higher in active smokers than in non-smokers [13, 17]. Also, in our study, the exhaled CO level with the CO Pro Check was significantly higher in active smokers than passive and non-smokers and the values of exhaled CO in smoking and non-smoking subjects were similar to those of previous study [13, 17]. There is a direct relationship between the smoking status of a given individual and the concentration of carboxyhaemoglobin (COHb) in their blood [18, 19]. Exhaled CO reflects COHb accurately only if the lung acts as an effective tonometer, and exhaled CO is in dynamic equilibrium with COHb [18]. The measurement of exhaled CO is widely used to estimate COHb and, as such, to monitor the smoking habits of patients. Considering that a COHb concentration >2% is generally used in the clinical arena to separate smokers from non-smokers [20]. Our results shown that exhaled CO levels may be used to distinguish smokers from non-smokers as same as the others.

Our CO Pro Check shows 1-3 non-smokers, 4-6 passive smokers, 7-10 light smokers, 11-15 smokers and above 16 were heavy smokers. Any exposure to CO may occur in normal day-today life, due to environmental pollution, passive smoking, and occupational exposure, the most likely cause of high levels of exposure is smoking.

CO in expired air has been reported to be an indirect measurement for the quantity of passive smoking [21] and exhaled CO can be used as an indicator of indoor smoking. In our study, the exhaled CO levels were 3.50 ppm in passive smokers. Laranjeira *et al.* reported that exposure to environmental tobacco smoke is the most likely cause for the increase in CO levels among non-smoking waiters. In this study it is reported that pre-exposure exhaled CO level was 1.0ppm and post-exposure exhaled CO level was 3.5 ppm. In our study, 6 of passive smokers (n = 63) were business people, 33 was passive smokers were students and they explained that they usually spend their free time at this university canteen, 16 subjects were farmers who exposed to environmental CO, and 8 peoples were job in automobile exhaust system.

As expected, passive smokers had higher CO concentration than healthy non-smokers, in previous studies; the mean exhaled CO concentrations were usually similar in healthy non-smokers. For example, exhaled CO levels were determined 1.5+0.1ppm in Zayasu *et al*. study, 1.2+0.9ppm in Yamaya *et al*. study and 1.2+0.3ppm in Yamaya *et al*. study. In our study, the exhaled CO levels were 3.61ppm in healthy non-smokers. These results were high compared with other studies results. This may be due to excessive environmental pollution, faulty automobile exhaust system, and home heating system in our city. Our results shown that the optimal cut-off was 4.2ppm, giving 94% sensitivity and 85% specificity. Similarly, Middleton and Morice1 reported that the optimal cut-off was 6ppm (selectivity 96%, sensitivity 94%). Jarvis *et al*. reported that the optimal cut off was 8ppm (sensitivity 90%, selectivity 89%), and Crowley *et al*. also reported that a breath CO level 48ppm was strongly associated with a self-report of current smoking. When exhaled CO at 7ppm or over differentiated ''smokers'' from ''non-smokers'', sensitivity was 93% and specificity was 95% for detecting smokers. Likewise Hewat *et al*. shown that exhaled CO levels were all below 7ppm, within the normal range for non-smokers. Many studies using breath CO monitors have tended use 10ppm as the cut-off [22, 23]. In our study, we found the cut-off was 10.5ppm, giving 75% sensitivity and 98% specificity. This result suggests that 10.5ppm is too high to be a cut off for a screening test, as it will reduce its sensitivity.

# COUNCLUSION

The measurement of Mean exhaled CO levels in active smokers was 15.47ppm
The measurement of Mean exhaled CO levels in passive smokers was 4.5ppm
The measurement of Mean exhaled CO levels in non-smokers was 1.65ppm
Determining of exhaled CO level 4.5ppm strongly suggests that subject is a smoker. So, Measure the CO levels to ensure the patients' health condition to wards CO poisoning risks, decreased morbidity and mortality due to smoking causes respiratory, cardiac, and neuronal and other problems and CO poisoning.

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