

Concentrations of Carboxyhemoglobin in the Blood of Smokers and Non-Smokers

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Introduction

The main sources of carbon monoxide (CO) with which man comes into contact are emissions from cars and tobacco smoke.

A feature of CO is that it has an affinity for hemoglobin which is many times higher than that of oxygen. For example, in arterial blood a CO partial pressure of $\frac{1}{500}$ of a given O₂ partial pressure is sufficient to bind to 5% of the hemoglobin.

However, due to the high dependency of the CO binding curve on the O₂ partial pressure, the CO partial pressure for mixed venous blood is only $\frac{1}{700}$ of a given O₂ partial pressure (Zander, unpublished data). The result is that CO inhaled into the alveolar space is almost completely taken up by the blood with the formation of carboxyhemoglobin (COHb).

Once in the blood, carbon monoxide has two effects: It reduces the concentration of hemoglobin available for O₂ transport, and thus the arterial O₂ concentration, and it causes a leftward shift in the O₂ binding curve of the remaining (free) hemoglobin, thus reducing the O₂ partial pressure in the capillaries.

Therefore, it seemed advisable to re-examine the COHb concentrations in the blood of smokers and non-smokers. The CO concentrations in inhaled cigarette smoke are still similar to those of car emissions without the use of a catalyst, namely 1.5–4.5 vol.% (for comparison, the maximum allowed concentration in the work place (MAC) is 0.003 vol.%).

In addition, it appeared necessary to examine more closely the results reported by Hlastala et al. [2] by measuring the influence of CO on the shape of the O₂ binding curve using improved methods, under physiological conditions, in the range of 0–20% COHb, and especially in the middle, relevant range of the O₂ binding curve.

Methods

COHb concentrations, expressed as a percentage of total hemoglobin, were determined in the blood of cigarette smokers and non-smokers using a CO-oxymeter (Ciba Corning 2500). In all cases venous blood was examined. The blood samples from the non-smokers were taken in the morning.

The first blood sample was taken from the smokers in the evening between 10 and 12 p.m., after which smoking was discontinued. The second sample was taken the next day around 9 a.m., and the third one around 2 p.m. The so-called half-life was calculated from the COHb concentrations measured after smoking was discontinued. This half-life of cCOHb (%) indicates the time it takes after discontinuation of smoking for the COHb concentration to fall to half that of the starting value. During the whole day before the first blood sample was taken the smokers kept a record of how many cigarettes they smoked and the average number of inhalations per cigarette smoked.

In all cases the COHb concentrations were calculated as the mean value from 5 single measurements.

For the determination of the O₂ binding curve, heparinized venous blood from 12 subjects was equilibrated in a tonometer at 37°C (Instrumentation Laboratory 237) with gas mixtures of defined composition (precision gas mixer, Corning 192), whereby pO₂ values were set at 15, 25, 35, 55 and 90 mmHg (pCO₂ always 40 mmHg).

In all cases the pH value was checked and, if necessary, was adjusted to 7.40 ± 0.02 by addition of NaHCO₃.

In order to shorten the equilibration time, the blood samples were adjusted beforehand to COHb values of 5, 10, 15, 20 and 25% by mixing them with 100% COHb blood.

The measurement of sO₂ (%) as well as control of cCOHb (%) was performed using the CO-oxymeter (see above).

The O₂ binding curves for linear COHb values were obtained by graphic interpolation.

Results

The COHb concentration for non-smokers was found to be $1.2 \pm 0.2\%$ ($n = 11$). For the smokers, who had smoked 30.6 ± 16.6 cigarettes with 10.5 ± 2.7 inhalations per cigarette on the day the first blood sample was taken, a mean COHb concentration of $10.4 \pm 3.1\%$ ($n = 14$) was measured in the evening. The results are summarized in figure 1.

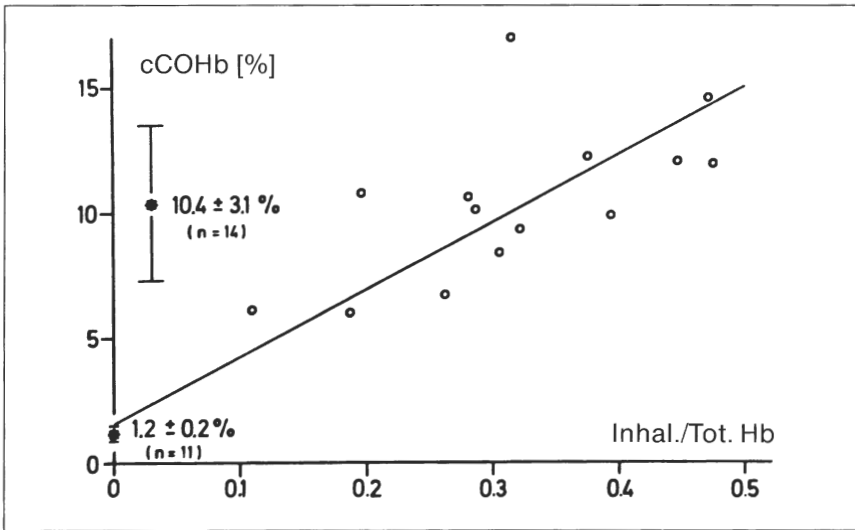


Fig. 1. Evening COHb concentrations in the blood of 14 smokers who had smoked an average of 30.6 ± 16.6 cigarettes on the same day, depicted as a function of the sum of inhalations per total hemoglobin of the subjects (blood volume \times Hb concentration). In comparison to 11 non-smokers with a mean COHb concentration of $1.2 \pm 0.2\%$, the smokers show a mean COHb concentration of $10.4 \pm 3.1\%$.

The wide range of individual COHb values is due to varying smoking habits. Apart from the number of cigarettes smoked, the number of inhalations per cigarette, the type of cigarette smoked (length, paper, filter) as well as the individual smoking habits (manner of inhalation) all play an important role. Since CO is almost exclusively present bound to hemoglobin, the COHb concentration has to be determined in relation to the amount of the total Hb of the subjects, which can be obtained from the product of the blood volume (ca. 8.5% of body weight) and blood Hb concentration. If the evening COHb concentrations are plotted against the sum of the number of inhalations per day (proportional to CO uptake) per total Hb in g (proportional to the CO distribution), a linear relationship with a positive gradient ($n = 25$, $r = 0.927$) is observed despite the wide range of measured values. The calculated half-life of COHb for all smokers is 8.3 ± 1.4 h; in other words, after discontinuing smoking it takes

on average 8.3 h for half of the CO to be eliminated from the body and for the COHb concentration in the blood to be halved.

The O₂ binding curves calculated for 1, 10 and 20% COHb are shown in figure 2.

It is obvious that the COHb concentrations found in smokers have a clear influence on the position and form of the O₂ binding curve; this is especially true for the middle and lower regions of the O₂ binding curve.

Discussion

Given a measured CO concentration in inhaled cigarette smoke of between 2 and 5 vol.% (Zander, unpublished data), a smoker takes up between 1.4 and 3.5 ml CO into the alveolar space with every inhalation (approximately 70 ml of smoke). This CO is almost completely taken up by the blood. Smoking a cigarette thus increases the COHb concentration in the blood by ca. 1–2%, depending on the distribution volume, i.e. the total hemoglobin of the subject. Because of the measured half-life of 8.3 h, CO, once taken up, is eliminated only very slowly. The COHb concentrations of smokers given here are in good agreement with the results of Anderhub et al. [1], i.e. 3–12% COHb, and are clearly below the maximum values for smokers as compiled from the literature by Pankow [3], namely 12–22% COHb.

Due to the wide variations in smoking habits and the distribution throughout the body described above, it is not possible to give even a rough prediction of the COHb concentration in the blood of smokers.

The influence of CO on the position and form of the O₂ binding curve in the range relevant for smokers of between 10 and 20% COHb is worth noting. It is exactly in the middle and lower range of the O₂ binding curve that is known to be relevant for the transfer of O₂ from blood to the tissue that there is a rapid leftward shift and loss of the S-form. The consequences are clear:

COHb causes a marked deterioration in the transfer of O₂ from blood to the tissue, especially in the organs which have a large arteriovenous O₂ difference (myocardium, cerebral cortex). The consequences at a Hb concentration of 15 g/dl for the myocardium with an avDO₂ of 12 ml/dl (difference in saturation 57.5%) are shown in figure 2. Thus, the coronary venous pO₂ would fall from 22 mmHg (at a physiological cCOHb of 1%) to only 12 mmHg (at 20% COHb).

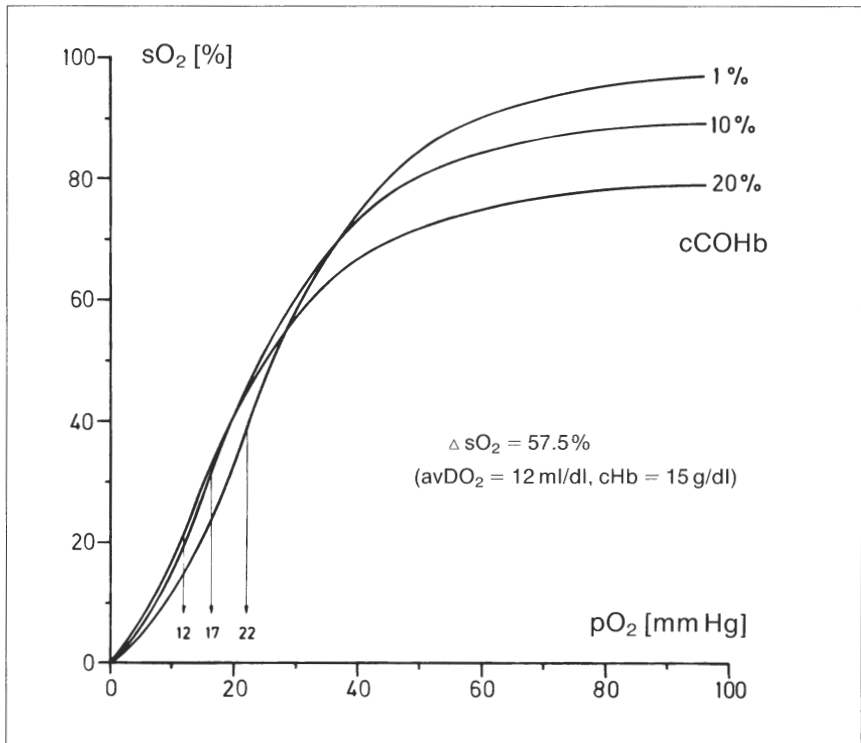


Fig. 2. O₂ binding curves, sO₂ (%) as a function of pO₂ (mmHg), under physiological conditions (37°C, pH = 7.40 ± 0.02, pCO₂ = 40 mmHg) in the presence of 1, 10 and 20% COHb. The decrease in the arterial O₂ concentration in connection with an increasingly leftward shift of the O₂ binding curve due to CO intoxication puts a strain on organs with a high avDO₂ (myocardium) with increasingly smaller venous (coronary venous) O₂ partial pressure values (22, 17, 12 mmHg).

If such a leftward shift of the O₂ binding curve caused by CO were to be combined with an additional leftward shift due to another cause, clinical consequences could be expected. For example, when stored blood also shows a leftward shift of the O₂ binding curve within a few days, the combination of these two effects can be significant (see [4]). Therefore, a routine determination of COHb is indispensable in an analysis of the arterial O₂ status.

Summary

Compared to non-smokers with approximately 1% COHb, cigarette smokers showed evening COHb concentrations of between 5 and 15%. The half-life of COHb determined in 14 smokers was approximately 8 h; in other words, after discontinuation of smoking it took about 8 h for the COHb concentration in the blood to fall by half.

Even in the range of 10–20%, COHb had a clear influence on the position and form of the O₂ binding curve, which can lead to a deterioration in the O₂ supply to the myocardium.

Since it is hardly possible at present to make an accurate prediction of the COHb concentration based on smoking habits, COHb should be determined with each routine analysis of the O₂ status.

References

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- 2 Hlastala, M. P.; McKenna, H. P.; Franada, R. L.; Dettler, J. C.: Influence of carbon monoxide on hemoglobin-oxygen binding. *J. appl. Physiol.* 41: 893–899 (1976).
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- 4 Zander, R.: Klinik der O₂-Bindungskurve am Beispiel von Rauchern und gelagertem Blut. *Anaesthesist* 37 (Suppl.): 95 (1988).