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Acute Effects of Cigarette Smoking and Inhalation of Carbon Monoxide During Maximal Exercise*

K. Klausen, C. Andersen, and S. Nandrup

Laboratory for the Theory of Gymnastics, August Krogh Institute, University of Copenhagen, Universitetsparken 13, DK-2100 Copenhagen, Denmark

Summary. The acute effect of inhaling the smoke of three cigarettes was compared to the effect of inhalation of an amount of carbon monoxide (CO), giving the same CO-saturation of the arterial blood as smoking during rest and during maximal exercise on a Krogh cycle ergometer. Sixteen male subjects were tested in the morning (1) after about 8 h without smoking (control), (2) after inhalation of the smoke of three cigarettes (smoke), and (3) after CO-inhalation (CO). It was found that the average maximal rate of O_2-uptake ($\dot{V}_{O_2}$ max) decreased during both smoke and CO by about 7%. Endurance time at $\dot{V}_{O_2}$ max decreased 20% during smoke but only 10% during CO. A significant decrease in maximal heart rate (HR), and an increase in HR at rest, was demonstrated only during smoke. The peak lactate concentration (HLa) following maximal exercise was significantly decreased after smoke. The results suggest that the decrease in $\dot{V}_{O_2}$ max during smoke is due to the CO-saturation of the blood, and hence to a decrease in the oxygen capacity of the blood, while the decrease in endurance time during smoke is a combined effect of the CO-saturation and an increased cost of breathing caused by the smoke particles. It is further suggested that nicotine, or possibly some other components of the smoke, have an enhancing effect on the heart at rest, while an inhibition is seen during maximal exercise. Finally it was found that the subjects who had a $\dot{V}_{O_2}$ max above the average for all subjects investigated were less susceptible to the effects of smoking than subjects with a $\dot{V}_{O_2}$ max below the average.

Key words: Smoking – Carbon monoxide – Maximal oxygen uptake – Peak lactate – Physical fitness

Offprint requests to: Klaus Klausen (address see above)
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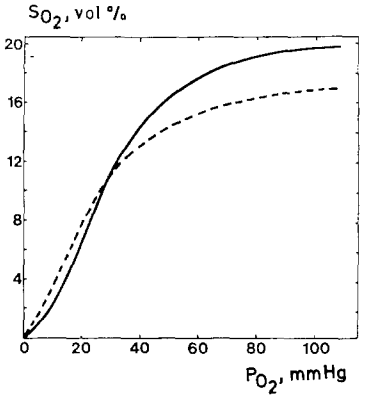

Fig. 1. Oxygen dissociation curve of a non-smoker (continuous curve) and of a heavy smoker (dashed curve). Abscissa = partial pressure of oxygen in the blood, ordinate = oxygen content of the blood. Note that at a P_{O_2} of 100 mm Hg the heavy smoker has an oxygen capacity which is 15% lower than that of the non-smoker

Introduction

Tobacco contains about 200 different components and the tobacco smoke from a cigarette contains more than 260 components (Kensler 1960). Some of these are highly dangerous in larger doses, but most of them are present in the smoke in concentrations so small that they have no measurable effect on the human organism.

The effect of smoking during exercise is most often related to carbon monoxide (CO), nicotine, and smoke particles ("tar"). The effects of these and maybe other components vary somewhat from one subject to another, partly because of a different sensitivity to tobacco smoke (Karpovich and Hale 1951), and maybe especially because the absorbtion of the different components into the body depends on the subject's smoking habit. Thus, depending on the intensity of smoke inhalation, the amount of nicotine taken up in the body by smoking one cigarette may vary from a few to almost 100% of the total nicotine content of the cigarette, and CO may occupy from 3–4% to more than 15% of the oxygen capacity of the blood.

Most interest has been focused on the effect of CO on working capacity. Figure 1 illustrates the effect of CO on the oxygen dissociation curve. As can be seen, the oxygen capacity of the blood is decreased corresponding to the amount of CO bound to the hemoglobin, and further the release of oxygen, e.g., to the exercising muscles, is impeded due to the shift to the left of the oxygen dissociation curve. Both effects will tend to decrease a subject's aerobic working capacity. This has been confirmed by several investigations. Thus, Nielsen (1971) in three subjects with an average CO-saturation of the blood (S_{CO}) of 28% found an average decrease in the maximal rate of oxygen consumption ($\dot{V}_{O_2}$ max) of about 24%, and Mollerup (1974) found in two subjects that smoking two cigarettes which gave an S_{CO} of 4% caused a decrease in $\dot{V}_{O_2}$ max of 2%. However, the nicotine, which among other things influences the autonomic nervous system, and the smoke particles, which influence the airways and the lungs, may also affect maximal working capacity. The purpose of the present experiments, therefore, is to investigate if there is any difference in the decrease of $\dot{V}_{O_2}$ max and endurance due to smoking or due to an S_{CO} of the same magnitude caused by inspiring CO-containing air.

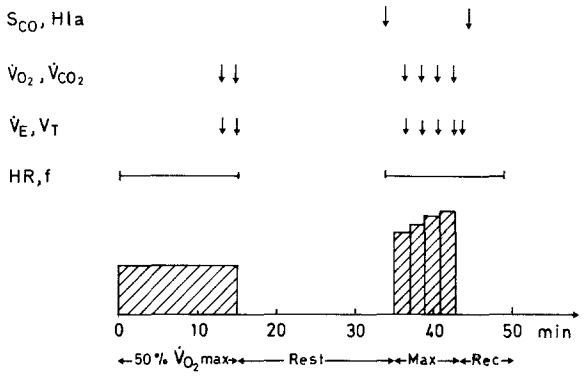

Fig. 2. Sequence of pertinent events before, during and after a maximal exercise test

Material and Methods

The subjects were 16 healthy, young males. Their average age, height and weight were 24.5 years, 180.6 cm and 75.0 kg, and their $\dot{V}_{O_2}$ max was 51 ml × kg^{-1} O_2 × min^{-1}. Most were moderate smokers (5–15 cigarettes per day), but one subject smoked 25 cigarettes per day, and two subjects were pipe smokers (about 5 fills per day).

All subjects performed a maximal test in the following three situations on 3 different days: (1) control experiment without smoking or CO-inhalation at least 8 h prior to the experiment (control), (2) after smoking three unfiltered, regular-size cigarettes immediately before the test (smoke), and (3) after inhalation of an amount of CO which gave the subject the same degree of CO-poisoning as smoking the three cigarettes (CO). The sequence of the three test situations was varied from one subject to the other, except that the smoke experiments always were performed before the CO-experiments.

Before the actual experiments, on a separate day, a work load corresponding to the subject's $\dot{V}_{O_2}$ max ($\dot{W}$ max) was extrapolated from the steady state HR obtained at three submaximal work loads and a measured maximal HR (HR max). The sequence of events during the three test situations are given in Figure 2. The subject started to exercise on the Krogh cycle ergometer for 15 min at a work load corresponding to about 50% of his $\dot{V}_{O_2}$ max. He then rested on a couch for 20 min. During control, the subject did nothing during the 20-min rest period, while during smoke he inhaled three cigarettes, and during CO he inhaled an amount of CO which gave him approximately the same CO-saturation in the blood (S_{CO}) as he had after smoking three cigarettes. At the end of the 20-min rest blood samples were obtained from a preheated fingertip for determination of S_{CO} and lactate concentration (HLa). Then the maximal exercise test was performed on the Krogh cycle ergometer as follows: The subject began to ride the ergometer at a pedalling frequency of 70 rpm and a work load corresponding to about 90% of his $\dot{W}$ max. The work intensity was increased by 5% every second minute until the subject was no longer able to keep the pedalling frequency of 70 rpm. After the end of exercise the subject rested sitting on the ergometer for 6 min with the legs in an elevated position.

During the maximal test, expired air was collected in Douglas bags during the last 45 s of each 2-min period. Respiratory frequency (f) and HR were registered continuously during the whole test. $\dot{V}_{O_2}$, pulmonary ventilation ($\dot{V}_E$) and tidal volume (V_T) were calculated for each 2-min period. During the 6-min recovery period HR and f were registered each minute, and, within the first 2 min of recovery, samples of arterialized capillary blood were obtained from a fingertip for determination of (HLa) and S_{CO}.

Mean values and standard errors of all variables from the 16 subjects were calculated. Differences between the three test situations were analyzed by the Student *t*-test for paired observations. Significance was set at the 0.05 level of confidence.

Table 1. Maximal values of different variables measured during a maximal exercise test in the three different test situations

	Maximal test			
	n	Control	Smoke	CO
Work time (min)	16	8.77 ± 0.34	7.01 ± 0.46[a]	7.84 ± 0.33[a, b]
$\dot{V}_{O_2}$ max ($l \cdot min^{-1}$)	11	3.83 ± 0.17	3.54 ± 0.21[a]	3.59 ± 0.18[a]
$\dot{V}_{O_2}$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)	11	51.2 ± 2.0	47.4 ± 2.7[a]	48.0 ± 2.3[a]
V_E max ($l \cdot min^{-1}$)	11	150.3 ± 6.1	143.6 ± 8.3	146.7 ± 7.4
f max ($resp \cdot min^{-1}$)	15	53.7 ± 3.4	53.3 ± 3.0	49.5 ± 3.2[b]
V_T max (l)	10	2.95 ± 0.18	2.75 ± 0.17[a]	2.94 ± 0.19[b]
HR max ($beats \cdot min^{-1}$)	16	187.2 ± 2.0	181.1 ± 2.4[a]	184.9 ± 1.7

[a] Significantly different from control
[b] Significantly different from smoke

Results

The subjects average S_{CO} after smoking was 4.51%, and 5.26% after CO-inhalation. From Table 3 it can be seen that part of the CO was eliminated during the maximal exercise test. Thus the average S_{CO} in the three test situations was: Control: 1.19%, smoke: 3.55% and CO: 4.33%. Part of the explanation for the higher S_{CO} in the CO-experiments is a faulty dosage of CO in two subjects. If these two subjects are omitted the average S_{CO} in the CO-experiments is 3.94%.

Average values (± 1 SE) from the maximal exercise tests in the three different test situations are presented in Table 1: There is a 7% decrease of $\dot{V}_{O_2}$ max from control to smoke and CO. On the other hand, worktime has decreased 20% during smoke but only 10% during CO. The slight reductions in $\dot{V}_E$ max during smoke and CO are not significant. Of further note is the 7% reduction of V_T max and the 3% reduction of HR max during smoke.

From Table 2 it can be seen that the HR at rest and 6 min after the end of the maximal test are significantly increased during smoke as compared to both control and CO. Further that f at rest and 6 min after the maximal test is lower during CO compared to both control and smoke.

Besides the S_{CO} values mentioned above, the (HLa) before and after the maximal exercise tests are presented in Table 3. As can be seen the (HLa) after exercise during smoke is significantly lower than in control.

Discussion

The present experiments have demonstrated that the reduction of $\dot{V}_{O_2}$ max is the same in Smoke as in CO. Thus, one reason for the decrease of $\dot{V}_{O_2}$ max after smoking is the increase in S_{CO} in the blood. On the other hand, the marked decrease in endurance time after smoking reveals that components in the smoke other than CO-influence work capacity. Here it is worth noting that several

Table 2. Heart rate and respiratory frequency at rest before the maximal exercise test and after 6 min recovery in the three different test situations

	Rest before max test			After 6 min recovery		
	Control	Smoke	CO	Control	Smoke	CO
HR (beats · min^{-1})	81.0 ± 2.9	95.0 ± 2.6[a]	82.7 ± 2.9[b]	100.5 ± 1.3	107.0 ± 2.3[a]	101.2 ± 1.2[b]
f (resp · min^{-1})	18.0 ± 1.2	20.8 ± 1.1	15.8 ± 1.1[a]	23.3 ± 1.2	24.7 ± 1.2	22.8 ± 0.9

[a] Significantly different from control
[b] Significantly different from smoke

Table 3. Carbon monoxide saturation of the blood and blood lactate concentration at rest before the maximal exercise and 2 min after the test in the three different test situations

	Rest before max test			2 min after max test		
	Control	Smoke	CO	Control	Smoke	CO
S_{CO} (%)	1.51 ± 0.22	4.51 ± 0.27	5.26 ± 0.45	0.86 ± 0.19	2.59 ± 0.23	3.39 ± 0.32
[HLa] (mmol · l^{-1})	1.3 ± 0.07	1.3 ± 0.09	1.3 ± 0.10	10.4 ± 0.40	9.3 ± 0.52[a]	9.8 ± 0.41

[a] Significantly different from control

investigations seem to indicate that particles in the smoke cause acute bronchoconstriction (Clarke et al. 1970; Nadel and Comroe 1961). This would increase the airway resistance and hence increase the energy cost of maintaining a given pulmonary ventilation. Thus one possible explanation for the marked decrease in work time during smoke could be that an increase in the cost of breathing would demand a greater fraction of the total pulmonary O_2-uptake, and hence cause a reduction in the oxygen supply available for the exercising leg muscles. An increase in airway resistance in the present experiments is to some extent indicated by the significant decrease in V_T max found during smoke (Table 1). The increase of HR at rest after smoking found in the present experiments confirm previous investigations (Behr et al. 1981; Burn 1960; Coffman and Javett 1963; Goldbarg et al. 1971; Irving and Yamamoto 1963; Kerrigan et al. 1968; Moses et al. 1964; Rottenstein et al. 1960). This effect of smoking on HR at rest is apparently in contrast to the decrease in HR max found during smoke. The present experiments do not allow any conclusive explanation of this discrepancy. Most investigations on the effect of smoking at rest assume that the increase in HR is due to the effect of nicotine (Coffman and Javett 1963; Goldbarg et al. 1971; Irving and Yamamoto 1963; Moses et al. 1964; Rottenstein et al. 1960). The same explanation is used for the finding that HR during submaximal exercise is elevated after smoking (Goldbarg et al. 1971; Krone et al. 1972). In some of these experiments it has further been shown that the increase in HR is associated with a trend towards an increased stroke volume (SV) and an increased mean arterial blood pressure (MABP). These changes seem to show that nicotine has a stimulating effect on the sympathetic nervous system. However, in some experiments (Coffman and Javett 1963; Rottenstein et al. 1960) it has been found that nicotine causes an increase of muscle blood flow and a decrease of skin blood flow, and further that smoking reduces the venous return to the heart both at rest and during exercise (Krone et al. 1972), in some cases leading to a reduction of SV (Goldbarg et al. 1971; Krone et al. 1972). Behr et al. (1981) on the other hand found that neither HR, MABP, or cardiac output were influenced by smoking during severe, submaximal exercise. These different findings indicate that the effect of nicotine on the circulatory system at rest and during submaximal exercise at the moment is not fully elucidated. When it comes to the decreased HR max after smoking seen in the present experiments, a stimulating effect of nicotine cannot be the explanation. On the contrary, a slight inhibitory effect on the heart is indicated.

HR max is related to work time, as can be seen from Fig. 4: The longer the maximal exercise is continued, the higher the HR max. Therefore, the lower HR max during smoke must obviously have been influenced by the shortened work time. However, the average HR during the CO and the control experiments at the time of exhaustion during smoke (7 min in Fig. 4) was still higher than the actual average HR max in the smoke experiments (c.f. dashed line on Fig. 4). Thus the above suggestion of an inhibitory effect of nicotine (or rather of smoking) on the heart during maximal exercise seems justified. This possibility is further supported by the fact that the average maximal "oxygen-pulse" [i.e., ($\dot{V}_{O_2}$ max $\times$ 1,000): HR max] is decreased during smoke as compared to control and to some extent also as compared to CO (Fig. 3 left). If

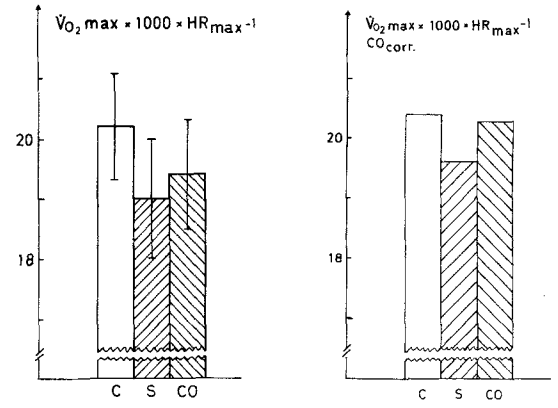

Fig. 3. Left half shows the average maximal "oxygen pulse" [ml O_2 per heart beat (± 1 SE)] in the three experimental situations (C = control, S = smoke). Right half shows the average oxygen pulses corrected for the amount of blood saturated with carbon monoxide ["(oxygen + CO) pulse"]

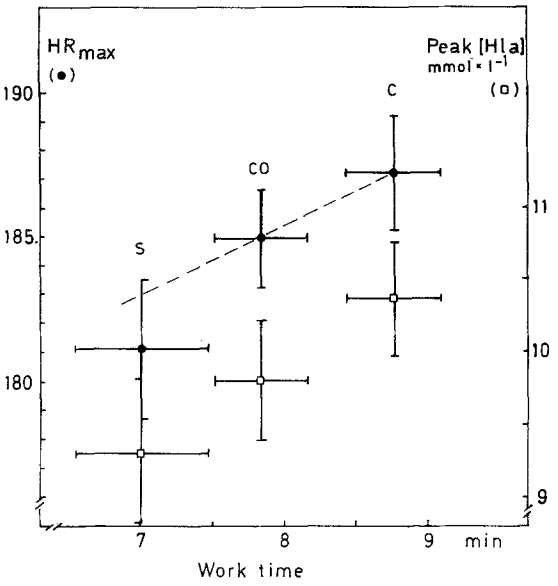

Fig. 4. ● = maximal HR in relation to work time in the three experimental situations (C = control, S = smoke). Dashed line is explained in the text. □ = maximal blood lactate concentration [peak (HLa)] in relation to work time in the three experimental situations

correction is made for the average CO-content of the blood in the three experimental situations the "(oxygen + CO) pulse" may express the stroke volume of the heart, and as can be seen from the right half of Fig. 3, this variable is decreased during smoke, while the value during CO is virtually unchanged as compared to control.

Thus the present experiments seem to show that nicotine, or possibly some other components of the smoke, have an enhancing effect on the heart at rest, while an inhibition is seen during maximal exercise.

It may seem surprising that the maximal (HLa) after the end of exercise is lowest during smoke, since the oxygen supply to the muscles in this situation is probably reduced more than during CO or the control experiments. However, as can be seen from Fig. 4, there is a linear relation between work time and (HLa)

in the three test situations. Thus the blood (HLa) mainly reflects the amount of HLa which can be produced by the muscles in the time available during the maximal exercise before exhaustion.

Finally it should be mentioned that $\dot{V}_{O_2}$ max determinations were completed in all three test situations on 11 subjects (Table 1). Based on the $\dot{V}_{O_2}$ max determinations in the control experiments, these 11 subjects were divided into two groups: group I with an average $\dot{V}_{O_2}$ max of 57.3 ml $\times$ kg^{-1} $\times$ min^{-1}, and group II with an average of 46.5 ml $\times$ kg^{-1} $\times$ min^{-1}. The reaction of these two groups to smoke was rather different: The reduction in HR max and $\dot{V}_{O_2}$ max was on average 1.0 and 4.4% respectively in group I, while in group II it was 4.3 and 12.5%. Also the reduction in work time was different for the two groups (14.3% in group I and 18.0% in group II). Thus, it seems that subjects with a high state of physical fitness, i.e., high $\dot{V}_{O_2}$ max, are less sensitive to the effects of smoking on physical performance.

In conclusion, the present experiments have clearly shown that smoking reduces maximal performance of 7–9 min duration. It has been demonstrated that the CO-content of the blood is in the main responsible for the reduction in aerobic power. Nicotine, smoke particles and other components of the tobacco smoke may further contribute to the decrease in maximal performance. It should be noted that the effect of CO is not persistent, since ample amounts of the blood born CO are washed out during exercise (Table 3), but the effect of CO may still be measurable even after ½ h of heavy exercise. Finally, it seems as if well trained subjects are less susceptible to the destructive effects of smoking on physical performance.

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