

AMRL-TR-70-37

AD0717716

ADDITIONAL



EFFECTS OF SHORT-TERM LOW LEVEL CARBON MONOXIDE EXPOSURE ON HUMAN PERFORMANCE

ROBERT D. O'DONNELL, CAPTAIN, USAF

PETER MIKULKA, CAPTAIN, USAF

PATRICK HEINIG, LIEUTENANT, USAF

JAMES THEODORE, MAJOR, USAF, MC

JUNE 1970

20060707039

This document has been approved for public
release and sale; its distribution is unlimited.

STINFO COPY

AEROSPACE MEDICAL RESEARCH LABORATORY
AEROSPACE MEDICAL DIVISION
AIR FORCE SYSTEMS COMMAND
WRIGHT-PATTERSON AIR FORCE BASE, OHIO

NOTICES

When US Government drawings, specifications, or other data are used for any purpose other than a definitely related Government procurement operation, the Government thereby incurs no responsibility nor any obligation whatsoever, and the fact that the Government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data, is not to be regarded by implication or otherwise, as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use, or sell any patented invention that may in any way be related thereto.

Federal Government agencies and their contractors registered with Defense Documentation Center (DDC) should direct requests for copies of this report to:

DDC
Cameron Station
Alexandria, Virginia 22314

Non-DDC users may purchase copies of this report from:

Chief, Storage and Dissemination Section
Clearinghouse for Federal Scientific & Technical Information (CFSTI)
Sills Building
5285 Port Royal Road
Springfield, Virginia 22151

Organizations and individuals receiving announcements or reports via the Aerospace Medical Research Laboratory automatic mailing lists should submit the addressograph plate stamp on the report envelope or refer to the code number when corresponding about change of address or cancellation.

Do not return this copy. Retain or destroy.

The voluntary informed consent of the subjects used in this research was obtained as required by Air Force Regulation 169-8.

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY (Corporate author) Aerospace Medical Research Laboratory Aerospace Medical Div., Air Force Systems Command Wright-Patterson Air Force Base, Ohio 45433		2a. REPORT SECURITY CLASSIFICATION UNCLASSIFIED	
		2b. GROUP N/A	
3. REPORT TITLE EFFECTS OF SHORT-TERM LOW LEVEL CARBON MONOXIDE EXPOSURE ON HUMAN PERFORMANCE			
4. DESCRIPTIVE NOTES (Type of report and inclusive dates) Final Report, February 1969 - August 1969			
5. AUTHOR(S) (First name, middle initial, last name) Robert D. O'Donnell, Captain Patrick Heinig, Lieutenant Peter Mikulka, Captain James Theodore, Major			
6. REPORT DATE June 1970		7a. TOTAL NO. OF PAGES 51	7b. NO. OF REFS 25
8a. CONTRACT OR GRANT NO. b. PROJECT NO. 6302 c. Task No. 630202 d. Work Unit No. 630202013		9a. ORIGINATOR'S REPORT NUMBER(S) AMRL-TR-70-37 9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)	
10. DISTRIBUTION STATEMENT This document has been approved for public release and sale; its distribution is unlimited.			
11. SUPPLEMENTARY NOTES		12. SPONSORING MILITARY ACTIVITY Aerospace Medical Research Laboratory Aerospace Medical Div., Air Force Systems Command, Wright-Patterson AFB, OH 45433	
13. ABSTRACT The effects of 0, 50, 125, 200, and 250 ppm of carbon monoxide exposure on human time estimation, tracking, ataxia, galvanic skin response and heart rate were tested in 10 subjects. Mean carboxyhemoglobin levels from .96 to 12.37% were reached after 3 hours of exposure. No significant symptoms were reported by subjects, and no ability to detect the presence of CO was noted. No overall trend toward poorer estimates of a 10 second interval occurred as a function of CO uptake, and tracking performance did not become worse over the course of the exposure to CO. There were some indications that subjects under CO showed a different overall pattern of tracking over time than control subjects in that their performance curve was flatter. However, this observation must be considered tentative. No changes in absolute heart rate occurred during task performance in any group, but there was slightly less cardiac deceleration at the onset of a task in the CO groups during early exposure. No differences in galvanic skin response were found between any groups. It is concluded that the present data do not support the hypothesis that low level carbon monoxide exposure results in human performance decrements. Additional investigation is required to define the lower limit and extent of such exposure to resolve major conflicts in the literature. Key Words: Carbon Monoxide Time Estimation Galvanic Skin Response Human Performance Tracking Heart Rate Toxicology Ataxia			

SUMMARY

Problem

The present study was undertaken as part of the general program for the evaluation of atmospheric contaminants that may arise in confined spaces, which could have toxic effects on the human brain and nervous system. The purpose of this study was to evaluate the effects of short duration exposure to low concentrations of carbon monoxide (CO) on human performance. Since the brain is quite sensitive to CO and it is commonly present at low concentrations in a number of operational systems, its potential risk at sub lethal levels must be defined. This risk is especially important in the aerospace environment, since human performance is carried to its extreme limits in high performance aircraft and in space systems.

Approach

Performance was evaluated in ten human volunteers who performed various tasks during and following exposure to 0, 50, 125, 200, and 250 parts per million (ppm) CO for three hours. The performance tasks employed to evaluate central nervous system function included; the ability to estimate time intervals, a complex tracking task, a variety of tasks which measure balancing abilities, and the activity of the autonomic nervous system by assessing heart rate and sweating. Bloods were drawn from all subjects after the three-hour exposures to measure the amount of CO bound to hemoglobin (percent carboxyhemoglobin). The performance levels were compared with the amount of CO in the blood for all subjects.

Results

No significant symptoms were reported by any of the subjects, and they were unable to determine when CO was present. There were no significant changes in the performance tasks employed, which indicated that there was no decrement in human performance. However, a tentative observation noted was that the subjects during CO exposure showed some indications that the overall pattern of tracking performance was slightly different. In addition, there was a slight difference in heart rate deceleration at the onset of each performance task during CO exposure.

Conclusions

In this study, three-hour exposures to carbon monoxide at concentrations of 50 to 250 ppm caused no impairment of human performance. From the tasks employed and the abilities required to perform them, it was concluded that carbon monoxide exposure under the conditions described and with the attendant percentage of carboxyhemoglobin saturations would not impair certain critical aspects of a pilot's performance for instrument landings of an aircraft or space docking procedures, and that there would be probably no impairment of a driver's performance for driving an automobile at high speeds since it involves similar critical aspects of performance. Additional investigation is required to define the lower limits and the extent of such exposure, in order to resolve major conflicts in the literature.

FOREWORD

This research was performed in support of Project 6302, "Toxic Hazards of Propellants and Materials," Task 630202, "Pharmacological and Biochemical Mechanisms of Toxicity," Work Unit 630202013, "Toxic Effects on Nervous System and Performance". The work was accomplished between February 1969 and August 1969 in the Toxicology Branch, Toxic Hazards Division, of the Aerospace Medical Research Laboratory. This was a joint effort accomplished with the Flight Environment Branch of the Human Engineering Division.

The authors express their appreciation to Dr. Kenneth C. Back and Dr. Mel J. Warrick of the 6570th Aerospace Medical Research Laboratory. Through Dr. Back's stimulation and helpful criticism, this study became a reality and Dr. Warrick's review and thoughtful comments enhanced the preparation of the manuscript.

This technical report has been reviewed and is approved.

CLINTON L. HOLT, Colonel, USAF, MC
Commander
Aerospace Medical Research Laboratory

TABLE OF CONTENTS

<u>Section</u>	<u>Page</u>
I Introduction	1
II Procedures	3
III Results	14
IV Discussion	42
References	50

LIST OF FIGURES

<u>Figure</u>		
1	Schematic Representation of the Critical Instability Tracking Task Dynamics	7
2	Tracking Performance Under Three Levels of CO Over Time	17
3	Tracking Performance of CO Over Time at 200 and 250 ppm	23
4	Time Estimation Under Three Levels of CO Over Time	25
5	Exposures to 200 and 250 ppm CO	29
6	Heart Rate Pattern During Tracking Trials	31
7	Absolute Heart Rates for Pre- and Post-Start Intervals Over Three-Hour Exposure	32
8	"Cardiac Responsiveness" to the Start of Tracking Trials Over Three-Hour Exposure	34
9	GSR Scoring Points for Each Tracking Trial	37
10	GSR in Micromhos During Averaged Tracking Trial At the Start (15 Min.) and End (170 Min.) of Exposure	38

I

INTRODUCTION

Carbon monoxide (CO) has become an important byproduct of the advances in modern technology. The potential risk of this toxic substance applies not only to the general population, but also to military populations in many operational systems. This risk is especially important in the aerospace environment since human performance is carried to its extreme limits in high performance aircraft and in space systems. Even small effects on performance shown to result from CO exposure must be considered serious insofar as they affect the system operator. Mechanisms of CO action must be correlated with decremented performance in order to arrive at realistic generalizations from the laboratory situation to the operational environment.

The central nervous system is extremely sensitive to oxygen deprivation and is considered the prime locus of CO induced effects. The major mechanism of CO action is presumed to be mediated through tissue hypoxia (Dinman, 1968). Subjective symptoms rarely occur below carboxyhemoglobin (COHb) levels of 20 per cent, and acute signs of cardiovascular, respiratory, and central nervous system embarrassment occur at levels of COHb greater than 30 per cent (Haldane, 1895). However, a number of reports have indicated that the central nervous system may be impaired at levels of COHb as

low as 2 to 5 per cent. MacFarland, Roughton, Halperin & Niven (1944) demonstrated impairment of visual discrimination for brightness with COHb levels as low as 4 per cent. Lilienthal and Fugitt (1946) noted lowered flicker fusion at an altitude of 6000 feet with COHb levels between 5 and 10 percent. Decrement in limb coordination has been reported with the same COHb levels at normal atmospheric pressure (Trouton & Eysenck, 1961). Moreau (1964), Forbes, Dill, Desilva, and VanDeventer (1937), and Rockwell and Ray (1967) have all reported decrement of varying degrees in automobile driving ability as measured in simulators or in actual road driving, although these effects were seen only at relatively high COHb levels. Impairment in cognitive and psychomotor performance has been reported by Schulte (1963) at 5 per cent and less COHb concentration. Finally, decreased auditory discriminability was reported with COHb levels as low as 2 to 3 per cent (Beard & Wertheim, 1967).

Not all investigators have reported performance decrements at low CO levels. Clayton, Cook, & Frederick (1960) found no association between COHb levels and automobile accidents. Dorcus and Weigand (1929) did not find decrements in cognitive and psychomotor performance until the COHb level reached 25 to 30 per cent. Vollmer, King, Birren, and Fisher (1946) found no changes in flicker fusion, perimetry, or ataxia as a result of CO exposure, and no correlation between performance and the amount of COHb in the blood.

Cigarette smokers are estimated to have a COHb level between 3.8 and 6.8 per cent (Goldsmith & Landaw, 1968) and 12 to 14 per cent of employed persons have occupations in which there is a likelihood of exposure to high CO levels. If low CO levels produce significant performance decrements, then large segments of the population may be operating at depressed performance levels. It therefore becomes important to determine what effects are present, their mechanism of action, and the importance of these effects on performance of daily tasks.

The present study was undertaken to determine effects of CO on relatively simple applied performance tasks. Analyses were planned which would reveal if: (1) absolute performance levels changed as a result of CO exposure, and if (2) the CO exposure changed the pattern of performance over time.

II PROCEDURE

Subjects

All subjects used in the present study were male university students between 19 and 22 years of age. All had volunteered for the experiment and were paid for their participation. They were given a full explanation of procedures, including the fact that they were to be exposed to carbon monoxide (CO). However, although they were told the CO levels would be low, they were not informed what the precise

amounts would be. They were told that one exposure would be a control condition with no CO present. Neither they nor the experimenters knew when there was CO present. This double blind procedure was considered essential to preclude suggestion effects in the subjects, and to minimize any unintended communication of concern to the subjects by the experimenters.

Subjects were screened by a complete medical questionnaire. In a few cases where doubtful answers were given, a medical examination was performed. All experimental sessions were under medical supervision. In no case was it deemed necessary to eliminate a potential subject from the study for physical reasons. However, one subject, because of a recent knee injury, was not allowed to perform the post-exposure test of postural equilibrium described later. As one of the conditions for participating in the study, all subjects were to be non-smokers.

Environment

Carbon monoxide and control exposures were all carried out in the Thomas Domes, Wright-Patterson Air Force Base, Ohio. The physical properties and operating characteristics of these domes have been described elsewhere (Thomas, 1965). The dome is a completely closed environmental system into which a given contaminant can be introduced and maintained in precise amounts. Air flow is controlled by a series of blowers and vacuum pumps which produce a flow of 40 ft³/min,

yielding a complete atmospheric exchange every 20 minutes. The dome floor is roughly circular with a diameter of 12 feet. Subjects inside the dome can see into the surrounding room, but in the present experiment about eight feet of the dome windows were covered to eliminate distracting background movement from the subject's field of view while tracking. However, it was considered important to allow the subject to see "outside" during rest periods and to have space to move about in order to preclude possible sensory restriction effects which might mimic or confound CO effects. Temperature was controlled between 68 and 74 degrees F., and dome pressure was maintained at exactly 680 mm Hg at all times except during entry and exit. Entrance to the domes was accomplished through an airlock, allowing maintenance of a constant CO level at all times. At those times when subjects or experimenters entered or left the dome, it was necessary to establish ambient atmospheric pressure while the inner hatch was opened, and then to re-establish the standard pressure after it was closed. This caused a slight pressure imbalance in the subject's ear. None reported this pressure to be uncomfortable, and all indicated they had no trouble in "clearing" their ears. Illumination in the dome came from a single 100-watt light source located on the ceiling at a distance of 7 feet from the tracking target at an angle of 50°. The ambient sound level in the dome was high (85 db) but the subjects' earphones attenuated this about 15 db. This level was reported to be comfortable by all subjects. There were no beats or rhythmic alterations

of the sound which could have acted as a time cue to the subjects.

Experimental Measures

Time Estimation. In view of past results indicating that the "time sense" may be disrupted by carbon monoxide exposure, a time estimation task was included in the present experiment. Each subject was given the following instructions on the first time estimation trial of each day.

We are now ready to begin the time estimation task. You are to indicate the passage of each ten seconds by pressing the button on the arm of your chair. From the time I say start, estimate when ten seconds have elapsed and press the button. Release the button immediately, rest your arm, and estimate ten seconds from the moment you pressed the button. Do not look around while you are doing this. In fact, some people find it best to close their eyes during this task. Continue estimating these ten second periods until we tell you to stop. The task will last for several minutes.

After a brief rest period the subject was told to "start" and the task was continued until the first response beyond three minutes. No knowledge of results was given the subject.

Critical Instability Tracking Task

In order to determine if low level CO exposure affects performance which is readily quantified in terms of practical performance requirements of the human operator, the critical instability tracking task developed by Systems Technology, Inc., was included in the present study. This task requires an operator to stabilize a statically unstable controlled element by closing a compensatory control loop

around the system (Jex, 1967).

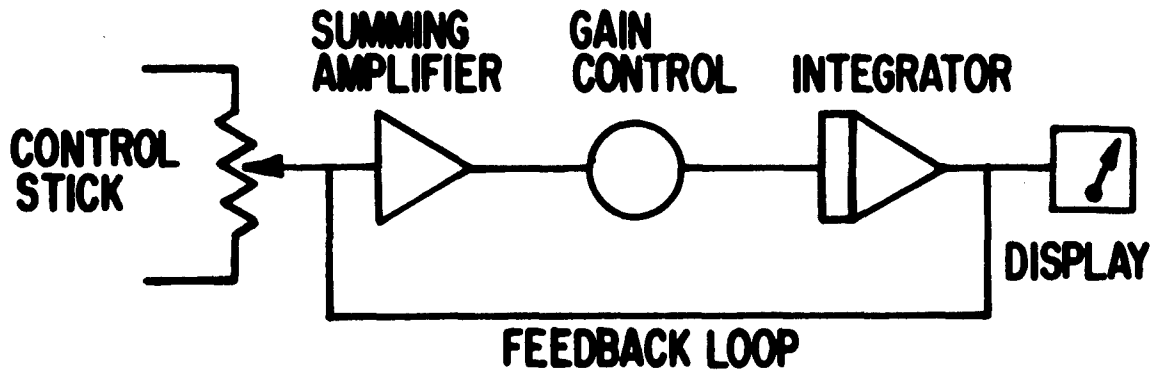


Figure 1. Schematic Representation of the Critical Instability Tracking Task Dynamics

Referring to Figure 1, it can be seen that any output from the integrator is fed back through the summing amplifier in such a way as to cause the output of the integrator to increase. If the operator, by moving his control stick, generates an input to the summing amplifier which exactly cancels the input fed back from the integrator, the output display will remain stationary. Any slight error in the timing or the amplitude of the subject's response will cause the output to change. Thus the operator's optimum strategy is to keep his control stick displacement exactly proportional to the display deflection.

In each experimental trial, the gain control was automatically

increased until the subject lost control of the system. This gain control changes the rate of divergence of the output for a given error, i.e., the divergence time-constant of the system. It has been shown (Jex, McDonnell & Phatak, 1966), that the value of the divergence time-constant at which the operator loses control of the system is inversely proportional to his "effective time delay" or effective reaction time in the tracking context.

Scoring on this task is based on this "effective time delay" (τ_e). The definition of τ_e is based on a partitioning of the various delays and lags involved in the eye/brain/limb/control stick system (Jex, McDonnell, & Phatak, 1966). Under certain conditions, the net phase lag can be closely approximated by an "effective" time delay which is the sum of the various delays, lags, and high frequency leads involved:

$$\tau_e = \bar{\tau}_d + \tau_r(t) + T_N - T_{Lhi}$$

where: $\bar{\tau}_d$ represents transport delays and central nervous system latencies (mean)

$\tau_r(t)$ represents the time varying component (average is zero)

T_N represents average neuromuscular lag

T_{Lhi} represents high frequency lead equalization beyond measurement bandwidth.

In essence, the subject is given a compensatory tracking task with a first-order divergent controlled element. The instability is increased until control is lost. This "critical instability" point, determined by the operator's effective delay time, has been shown to be an extremely stable measure (Jex, et al, 1966; Jex, 1967; McDonnell & Jex, 1967). The task itself, on theoretical grounds, has been analyzed to be sensitive to a number of stressors including hypoxia, drugs, g-level, low temperature and secondary workload (Rosenberg & Jex, 1966; Jex, 1967). Further, on the basis of the describing functions involved, it appears to be closely related to such activities as flying an instrument landing, controlling an automobile at high speed, manual aiming of such devices as cameras, and control of a spacecraft during docking and re-entry (Sadoff & Jex, 1968).

Pensacola Ataxia Test

One of the effects of relatively severe carbon monoxide exposure is a loss of balance and the appearance of other vestibular symptoms. To test for subtle changes in dynamic equilibrium, portions of the Pensacola Ataxia Test were used. This test battery consists of a number of balancing tasks which are performed either on narrow rails or on the floor (Graybiel and Fregly, 1965). For the present purposes, the following tasks were chosen:

1. The Sharpened Rhomberg (SR) Test, in which the subject

attempts to stand for 60 seconds in a heel-to-toe position with his arms folded and his eyes closed.

2. The WALK/EYES OPEN (W/EO) Test, in which the subject attempts to walk a rail $3/4$ inches wide and 8 feet long, with his arms folded and eyes open.

3. The STAND/EYES OPEN (S/EO) Test, in which the subject attempts to stand on the $3/4$ inch rail for 30 seconds with his arms folded and eyes open.

4. The STAND/EYES CLOSED (S/EC) Test, in which the subject attempts to stand for 60 seconds on a rail $2-1/4$ inches wide and 30 inches long.

5. The STAND/ONE LEG/EYES CLOSED (SOLEC) Test, in which the subject attempts to balance with one foot on the floor. This test is done for both the left and right feet.

6. The WALK A LINE/EYES CLOSED (WALEC) Test, in which the subject attempts to walk a 12-foot straight line with his eyes closed and his arms folded.

Design and Testing Procedures

In the major phase of this study, three levels of CO exposure were used: 125 parts per million (ppm), 50 ppm and a control condition, 0 ppm. The order in which subjects were exposed to these three levels was partially counterbalanced to preclude training or serial effects on any of the tasks. Originally, the

design called for nine subjects, with three receiving a 0-50-125 (a-b-c) order, three receiving the b-c-a order, and three receiving the c-a-b order. A tenth subject was to be used if one of the other subjects failed to complete the experiment. Since no subject failed to complete the experiment, four subjects received the a-b-c order. In addition, one subject was exposed out of his regular order due to a technician error. Because of the double blind procedure, this was not discovered until after the study was completed. Therefore, one subject received a b-a-c order, and only two subjects received the intended b-c-a order. Although this destroys the original counterbalancing, no essential harm was done since representative effects of ordering could still be examined.

After completing the major study, it was decided that increased CO levels should be given to a few subjects in order to explore additional hypotheses for future study. Five subjects were exposed to 200 ppm of CO. The design of this exploratory study was based on six experimental sessions, one being a "blank" or zero-control session. Thus, while not a balanced double blind experiment, the subjects and experimenters were never sure that CO was present. After these runs, three subjects were exposed to 250 ppm of CO in a single blind design. For both exposure levels, it was considered ethically necessary to inform the subjects that the CO, if present, would be at a higher level than before, although the precise amount

was again not specified.

Subjects in all experimental runs were scheduled at either 8 A.M. or 1 P.M. depending on their availability. Electrodes for taking GSR and EKG were attached, and approximately five minutes later the subject and experimenter entered the dome. Exposure time started at this moment. A respiration transducer was fixed around the subject's lower chest at the costal margin, headphones were put on, and after a final check of all recording and communications equipment, the experimenter left the dome through the airlock. The subject was then instructed to relax for a few minutes until he had been in the dome for exactly 15 minutes. The first series of measurements was then taken.

A performance session consisted of five trials on the tracking task, a brief rest, three minutes of time estimation, another brief rest, and five more tracking trials. After each session, the subject was told to relax for "about 15 minutes" until time for the next session. Thus, each subject performed for approximately 15 minutes out of each half hour.

Each dome exposure lasted three hours. Due to the regular occurrence of the testing sessions, subjects were usually aware of approximately how long they had been in the dome. In addition, after 90 minutes, the experimenter entered the dome, detached the electrode wires from their terminal boxes, and allowed the subject

to get up, stretch, walk around the dome, etc., in order to reduce the possibility of discomfort, fatigue, and boredom from sitting for three hours. After five minutes, the subject was again seated and re-connected to the system, which was again tested prior to the experimenter leaving the dome. As was noted earlier, entering and leaving the dome did not affect the level of CO, although it did require that dome pressure be brought down to ambient level and re-established after the experimenter had left.

Following the three hours exposure, the subject was immediately removed from the dome and a 10 cc blood sample was taken to determine hematocrit, hemoglobin, and carboxyhemoglobin levels. He was then escorted to a separate room (a distance of approximately 50 feet) and was given the Pensacola Ataxia battery. Following this, the subject breathed 100 percent oxygen for about ten minutes. This completed the experimental session, and an appointment was made for the next session not less than three days later.

III

RESULTS

Carbon Monoxide Exposure Levels

Carboxyhemoglobin (COHb) determinations on venous blood were made for each subject after each experimental session, using a modified gas chromatographic method of Dominguez, Christensen, Goldbaum, & Stembridge (1959). The results of these determinations are presented in Table I.

TABLE I

CARBOXYHEMOGLOBIN LEVELS FOR ALL SUBJECTS AT EACH EXPOSURE LEVEL

Subject	Order of Exposure	0 PPM (a)	50 PPM (b)	125 PPM (c)	200 PPM (d)	250 PPM (e)
1	abc	.7	2.8	6.5		
2	bca	1.2	2.4	6.6		
3	cabd	.8	2.7	6.5	9.8	
4	bcae	1.0	3.1	7.4		13.1
5	cab	1.0	3.0	6.8		
6	abcde	1.3	3.6	6.8	10.1	11.9
7	bacd	1.1	3.3	6.4	10.1	
8	abcd	.9	3.0	6.6	10.9	
9	cabde	.6	2.9	6.2	10.9	12.1
10	abc	3.5	4.5	7.4		
Mean*		0.96	2.98	6.64	10.35	12.37

* Does not include scores for subject No. 10.

It can be seen that the COHb levels reflect a direct relationship with

the level of ambient carbon monoxide. The data agree well with CO uptake curves based on time, exposure concentrations, and rates of ventilation constructed by Forbes, Sargent and Roughton (1945). One exception to this is noted in subject 10, who had a COHb level of 3.5 per cent in the 0 ppm condition. When this concentration was discovered, the subject was again questioned about his smoking, and admitted that on the day of the test, he had "one cigarette." His COHb level with no experimental CO exposure was higher than the mean level for those exposed to 50 ppm of CO. Since it must be assumed that this subject was a smoker, his data obviously could not be used in any group analyses.

Hematocrit levels for all subjects ranged from 42 to 52 and hemoglobin levels ranged from 12.8 to 17.6 gms per cent with no systematic variation due to CO exposure over all sessions.

Subjective Reports

All subjects were interviewed informally after each session to determine the incidence of subjectively perceived symptoms, changes in mood, or any other anecdotal observation of interest. Over the 39 experimental sessions, only one subject reported any symptoms -- subject 4 during exposure to 250 ppm. He reported a very slight headache which he said did not "interfere" with his performance. In addition, he reported that he felt better in general than during

his previous exposure, when he felt tired before and during the session. That previous session had been a 0 ppm control exposure. None of the other subjects reported headaches, dizziness, vegetative sensations, or any change in mood.

Subjects could not indicate, on questioning, whether CO had been present during a given experimental session. They did not perceive any change in their performance or in the effort expended to perform. Although several complained that their eyes became tired toward the end of the session, these complaints bore no relationship to the presence or absence of CO, and were probably related to the relatively low light level in the chamber. Most subjects used their free time during a session to read and study, and none reported difficulty in concentration or slowing of their thought processes. In leaving the dome, subjects were required to climb down a small ladder. No subject reported or displayed any unsteadiness or insecurity in negotiating this ladder.

Tracking Data

Scores from the critical instability tracking task were obtained in the form of the difficulty level reached by each subject on each trial before he 'lost control' of the task. The mean difficulty level was obtained for each series of ten tracking trials per 30 minute period. There were therefore six scores for

each subject during each dome exposure. All subjects had been trained to a plateau on the task prior to their first session.

Since the uptake of carbon monoxide within the body is cumulative during the entire three hours at these exposure levels, it is of interest to compare not only the differences between groups at the various time periods, but also the change over time within a given exposure level. Grouped data representing the mean scores for nine subjects at each time period are presented in figure 2. It can be seen, first of all, that no trend toward poorer performance over time appeared in any of the groups, even

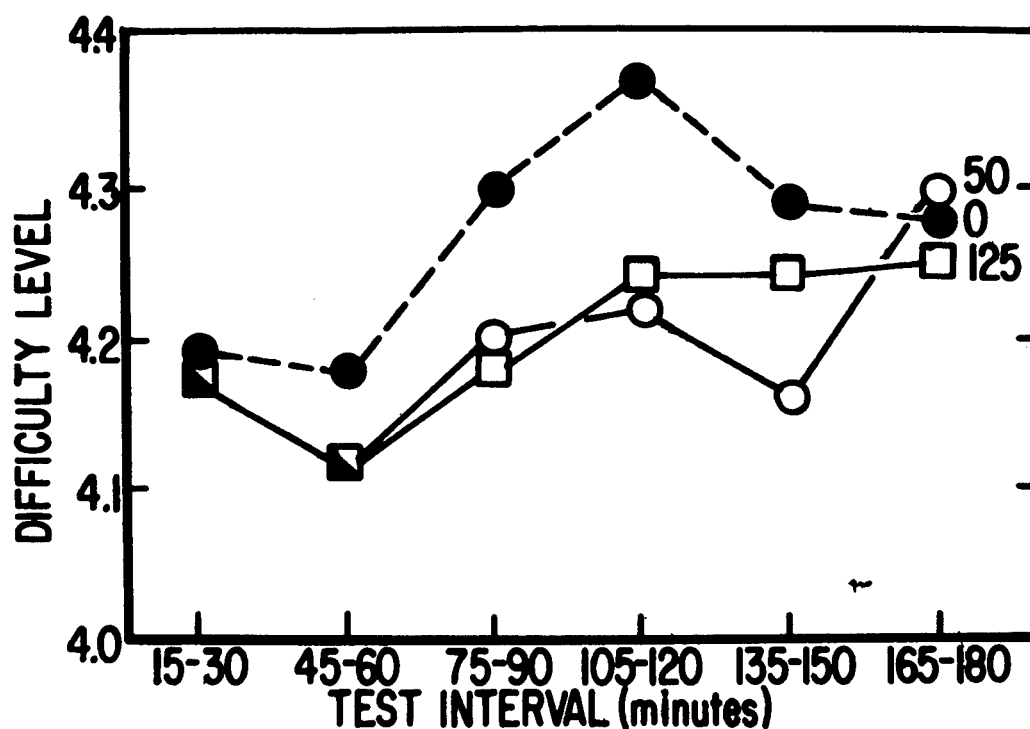


Figure 2. Tracking Performance Under Three Levels of CO Over Time.

under exposure to 125 ppm CO. There was an overall trend for all groups to improve their scores slightly over time in the dome. The data were therefore analyzed to determine the statistical significance of this effect. These analyses yielded F ratios of 1.84 for the 0 ppm condition, 1.81 for 50 ppm and 2.23 for 125 ppm. None of these is significant at the .05 level, although the value for 125 ppm is significant at .10. Thus, considered separately, no group showed a clearly reliable change in tracking performance over time, and the one group which showed the largest change indicated that tracking performance became somewhat better during exposure to 125 ppm CO. All groups appeared to show a slight but consistent improvement in performance during the second, and part of the third hour. In no case was performance worse in the second and third hours than it had been in the first hour. This finding answers one of the primary concerns of the present study. Tracking performance under low level CO exposure was not found to deteriorate with time over a 3 hour period.

To determine if there were relative differences between scores with and without CO present, the data was subjected to analyses of variance (repeated measures) at each time period. The results of these analyses are presented in table 2.

It can be seen that there were statistically significant differences in the performance level of the three groups at the

TABLE II

ANALYSES OF VARIANCE ON TRACKING SCORES FOR EACH TIME PERIOD

Time	Source	Sum of Squares	d.f.	Mean Square	F
20 Min	Between <u>Ss</u>	1.7645	8	.2206	.02
	Within <u>Ss</u>	.363	18	.0202	
	CO Effect	.001	2	.0005	
	Residual	.362	16	.0226	
50 Min	Between <u>Ss</u>	1.740	8	.2175	.41
	Within <u>Ss</u>	.453	18	.0252	
	CO Effect	.022	2	.0110	
	Residual	.431	16	.0269	
80 Min	Between <u>Ss</u>	3.112	8	.3890	2.35
	Within <u>Ss</u>	.308	18	.0171	
	CO Effect	.070	2	.0350	
	Residual	.238	16	.0149	
110 Min	Between <u>Ss</u>	2.860	8	.3575	4.53**
	Within <u>Ss</u>	.320	18	.0178	
	CO Effect	.116	2	.0580	
	Residual	.204	16	.0128	
140 Min	Between <u>Ss</u>	3.286	8	.4108	2.84*
	Within <u>Ss</u>	.297	18	.0165	
	CO Effect	.078	2	.0390	
	Residual	.219	16	.0137	
170 Min	Between <u>Ss</u>	3.297	8	.4121	.64
	Within <u>Ss</u>	.162	18	.0090	
	CO Effect	.012	2	.0060	
	Residual	.150	16	.0094	

* Sig at .10

** Sig at .05

110 and 140 minute periods. Newman-Keuls tests (Winer, 1962) were done on the 110 and 140 minute periods and it was verified that performance under the 0 ppm condition was better than either the 50 or 125 ppm conditions at 110 minutes ($P < .05$) and performance under the 0 ppm condition was better than under the 50 ppm condition at 140 minutes ($P < .10$). There was no significant difference in performance during the 50 ppm and 125 ppm conditions at either period. These results suggest that while there was no deterioration in performance over time under CO exposure, there may have been a CO-induced difference in the overall shape of the performance curves which manifested itself only in the middle of the three hour exposure.

In order to probe further into the nature of this possible effect, trend analyses were done on each condition over time (Winer, 1962). Table III summarizes the results of these analyses in terms of the percentage of the total mean square variance due to time which is accounted for by each polynomial component.

TABLE III
PERCENTAGE OF MS TIME ACCOUNTED FOR BY
EACH POLYNOMIAL COMPONENT

Component	0 PPM	50 PPM	125 PPM
Linear	37.9	45.0	62.4
Quadratic	31.3	1.0	0.9
Cubic	9.4	0.9	24.7
Quartic	5.8	41.6	11.6
Quintic	15.6	11.5	.4

It can be seen that there are distinct differences in the shapes of the curves and that these are most notably evident in a stronger tendency toward linear performance over time in the 125 ppm condition and in a much stronger quadratic or single-humped performance over time in the control condition than in any other. Average performance at 50 ppm shows a greater tendency to be erratic than in any other condition, as evidenced by the strong quartic component.

In summary, it is seen that if CO had any effect on the tracking scores seen in this study, it was not in the direction of causing a deterioration in absolute performance level. Rather, it was in disrupting the usual pattern of performance of the curves during the three hour period.

Overall inspection of the performance curves of individual subjects revealed no consistent effect of CO exposure. In four of the nine subjects, tracking performance under the 0 ppm condition was generally better than for the other conditions. In only one case was performance at 125 ppm consistently worse than in the other conditions, while in four cases performance under 50 ppm was worse than in the higher CO exposure. As CO uptake increased over the test periods, performance would be expected to show a decline over time if there was any simple relationship between the two. However, in no subject was an overall time-related decrement seen

in the 125 ppm condition, and two subjects showed a remarkably consistent improvement in performance over time in both the 50 and 125 ppm conditions.

It is of interest to examine the data from the one subject in the study who was assumed to be a smoker. In many respects, the curves for this subject are opposite to those found for the majority of subjects. Under the 0 ppm condition, this subject showed a fairly consistent decrement in performance as time in the dome increased. Conversely, under the 50 and 125 ppm exposures, this subject showed a slight, erratic improvement in performance. During the last 90 minutes of exposure, his tracking scores were slightly better than they were in the control condition. In addition, it might be noted that in absolute terms, this subject was the best tracker in the study, consistently performing at a higher level than the other subjects, no matter what the CO level.

Exposures to 200 and 250 ppm CO

Inspection of the data for the five subjects exposed to 200 ppm and the three subjects exposed to 250 ppm revealed no remarkable difference from the results already presented. As seen in Figure 3, tracking performance was generally better under 250 ppm than under any other condition for the same subjects. At the 110 minute point, the absolute tracking score fell slightly lower under the 200 and 250

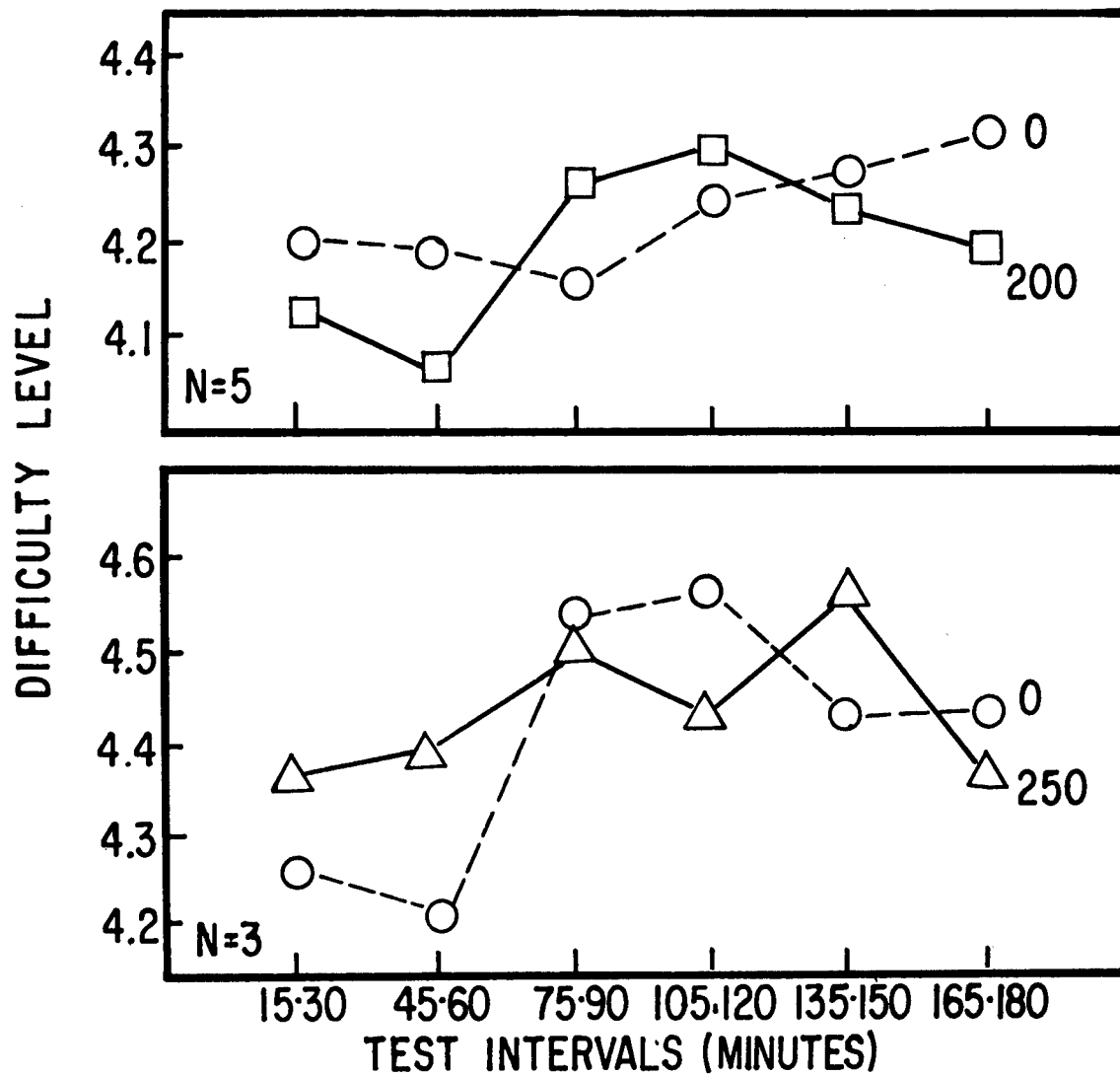


Figure 3. Tracking Performance of CO Over Time at 200 and 250 ppm

ppm conditions than it had been at zero. No overall performance decrement was seen over time, and in fact, overall performance improved slightly in both cases. There did appear to be a tendency for the performance curve under the 250 ppm condition to show a 'flatter' shape than it did in the control condition. This phenomenon has been noted in the data of the main experiment. Although these results are not directly relatable to the previous conditions, they tend to confirm the original findings that exposure to CO for three hours produces little absolute decrement in performance on the tracking task, but may disrupt the overall pattern of performance.

In view of these results, it must be concluded that under the conditions of the present study, no decrement in absolute tracking performance was found. There was an indication that carbon monoxide exposure resulted in a different pattern of performance than that seen in the control condition. This difference was most noticeable after the subjects had been exposed to CO for 110 minutes, and was manifested in a less improved tracking score than that seen in the control condition. However, after 170 minutes of exposure to CO, this difference had disappeared, and subjects were performing equally well with or without carbon monoxide.

Time Estimation Data

Under the procedure for time estimation used in the present

study, a series of approximately 18 estimates was obtained from each of six periods for each experimental session. The mean of these individual estimates was taken for each period and these means constituted the raw data for further analyses.

Grouped data representing these data points for nine subjects at each time period are presented in figure 4. It is evident that although there is some separation between the different conditions, no overall trend toward larger over-or-under estimation occurred

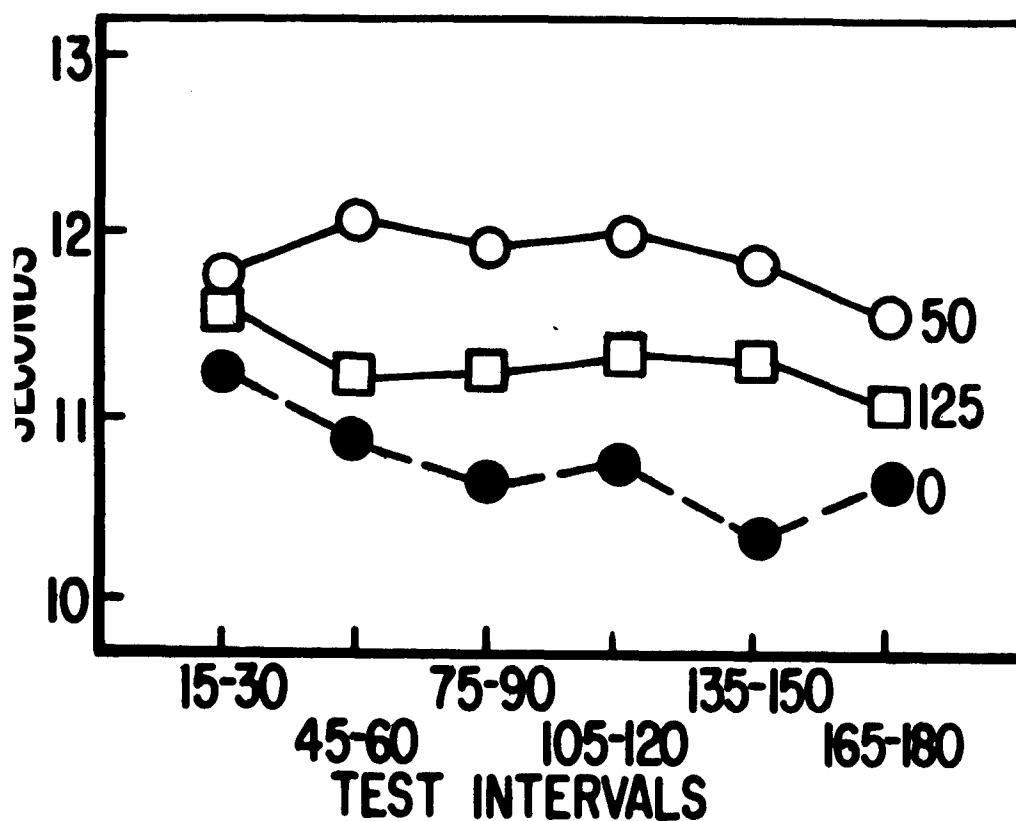


Figure 4. Time Estimation Under Three Levels of CO Over Time.

over time in any condition. The points are remarkably consistent under all conditions, with a very slight (non-significant) tendency for estimates to grow shorter for all three conditions during the second and third hours.

In order to test the relative differences between conditions at each time period, separate analyses of variance were performed on these data. The results of these analyses are presented in Table IV.

The only significant difference occurring in this table is at 140 minutes, where time estimates under 50 ppm were longer than under the control condition. Inspection of figure 4 reveals that this difference resulted from a decrease in the average estimates in the 0 ppm condition, and not from an increase in estimates attributable to CO. In no case were estimates under 125 ppm different from those under 0 or 50 ppm.

Inspection of the estimates of individual subjects similarly revealed no consistent effect attributable to CO exposure. In five subjects, time estimates were generally lower under the 0 ppm condition than under the other two. However, for the other four subjects, one or both CO conditions produced time estimates which were lower than those seen in the zero control. In only two cases did the 125 ppm condition produce the longest time estimates, while

TABLE IV
ANALYSES OF VARIANCE ON TIME ESTIMATION
AT EACH TEST INTERVAL

INTERVAL	SOURCE	d.f.	MEAN SQUARE	F RATIO
15-30	Between <u>Ss</u>	8	14.795	0.121
	Within <u>Ss</u>	18	3.718	
	CO	2	0.500	
	Residual	16	4.120	
45-60	Between <u>Ss</u>	8	13.109	0.824
	Within <u>Ss</u>	18	3.678	
	CO	2	3.090	
	Residual	16	3.751	
75-90	Between <u>Ss</u>	8	15.806	1.417
	Within <u>Ss</u>	18	2.678	
	CO	2	3.605	
	Residual	16	2.562	
105-120	Between <u>Ss</u>	8	18.201	1.663
	Within <u>Ss</u>	18	2.230	
	CO	2	3.455	
	Residual	16	2.078	
135-150	Between <u>Ss</u>	8	21.069	3.987*
	Within <u>Ss</u>	18	1.668	
	CO	2	4.980	
	Residual	16	1.249	
165-180	Between <u>Ss</u>	8	27.064	1.468
	Within <u>Ss</u>	18	1.233	
	CO	2	1.720	
	Residual	16	1.172	

* Significant at .05 level.

in five cases the 50 ppm condition produced the longest estimates.

It is interesting to note that for some subjects, an order effect appeared in the data. This is especially noteworthy since the task was unpracticed and no knowledge of results was given. In five cases, the first experimental session in the dome produced the longest estimates, regardless of the presence or absence of CO.

Time estimates made by the one subject who was assumed to be a smoker did not differ radically from the pattern already presented. From the first to the third hour under the 0 ppm condition, he showed an increase of about one second in his estimate. In the 50 ppm condition, he increased his estimate by one second in the second hour and then decreased it an equal amount in the third hour. Under the 125 ppm exposure, this subject showed a relatively consistent estimate between 11 and 11.5 seconds in the first two and one-half hours, and then a decrease to 10.5 seconds in the last half-hour.

Data for those subjects exposed to 200 and 250 ppm are presented in Figure 5. From this figure, it can be seen that in neither case was there a clearly defined effect of CO over time. For the exposures at 200 ppm, the curve is virtually identical to the control curve. At 250 ppm, the control curve produced consistent over-estimates of the 10 second interval, with the CO condition producing estimates which were under, but generally near, 10 seconds of real

time.

In summary, then, it may be concluded that under the conditions of the present experiment, no pattern of increase or decrease in the estimate of the empty time interval could be demonstrated as a result of CO exposure. In all conditions the averaged estimated interval for these subjects was longer than the actual 10 second clock time.

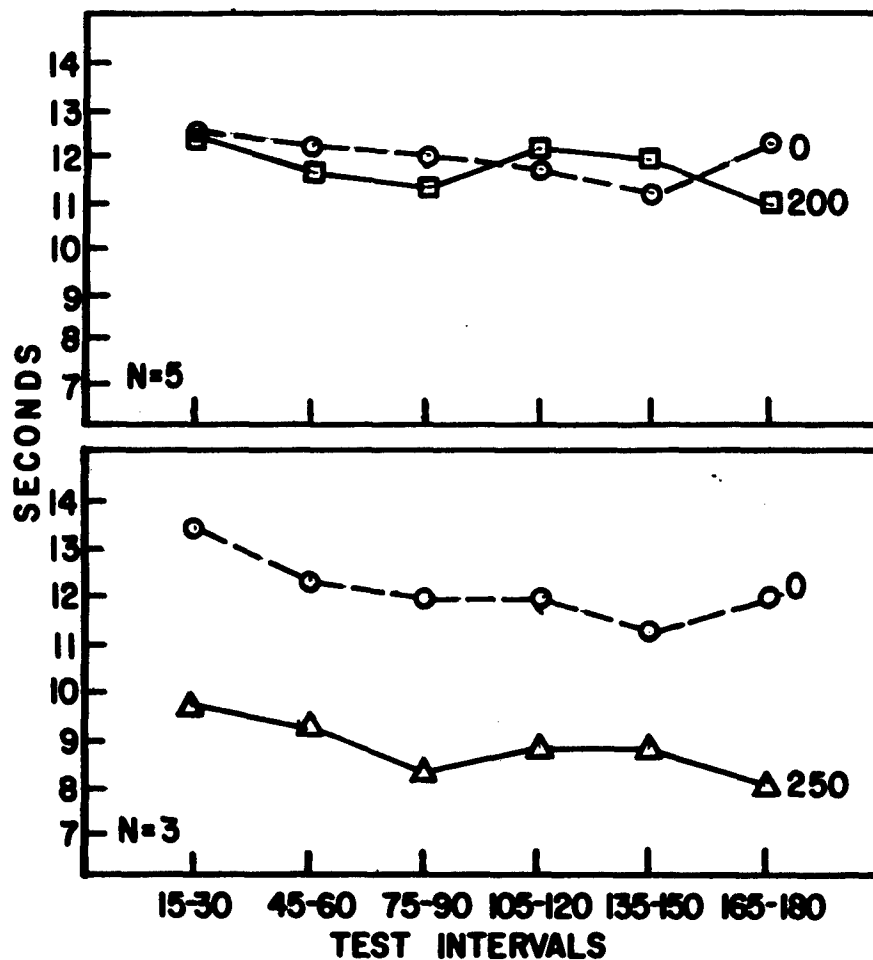


Figure 5. Exposures to 200 and 250 ppm CO

At 140 minutes, estimates under 50 ppm CO were significantly longer than under 0 ppm, due to a decrease in the estimates of subjects in the zero condition. This difference disappeared by 170 minutes.

Heart Rate Data

The subject's heart rate was monitored by two electrodes (Telec-trode 1801) placed approximately one inch below the pectoral muscles, and using the subject's left wrist as ground. Signals were amplified by a Sanborn Low Level Pre-Amplifier, model 350-1500, with a EEG/ECG module, model 350-3.

Heart rate was monitored continuously throughout each experimental session, except during the rest period 90 minutes after entry into the dome. Records were scored for the 10-second intervals bracketing the start and end of each tracking trial. Retrospectively, the subject's heart rate was scored for the 10 seconds prior to the start of a tracking trial (Prestart) and the 10 seconds immediately following start of the trial (Poststart). Likewise, pre-and post-End scores were obtained at the conclusion of a trial. Out of each block of five trials, only the data from the second and fourth trials were used for analysis.

Heart rate was examined in two ways to identify any possible effects of carbon monoxide. First, the absolute heart rate was examined for the pre- and post-intervals. Second, the amount of

change in heart rate from the pre to the post interval was analyzed in an attempt to assess any change in responsiveness to a specific stimulus.

Examination of Figure 6 shows the pattern of heart rate during the tracking interval. The pattern is uniform across CO levels and testing periods. Because of the symmetrical form of the cardiac

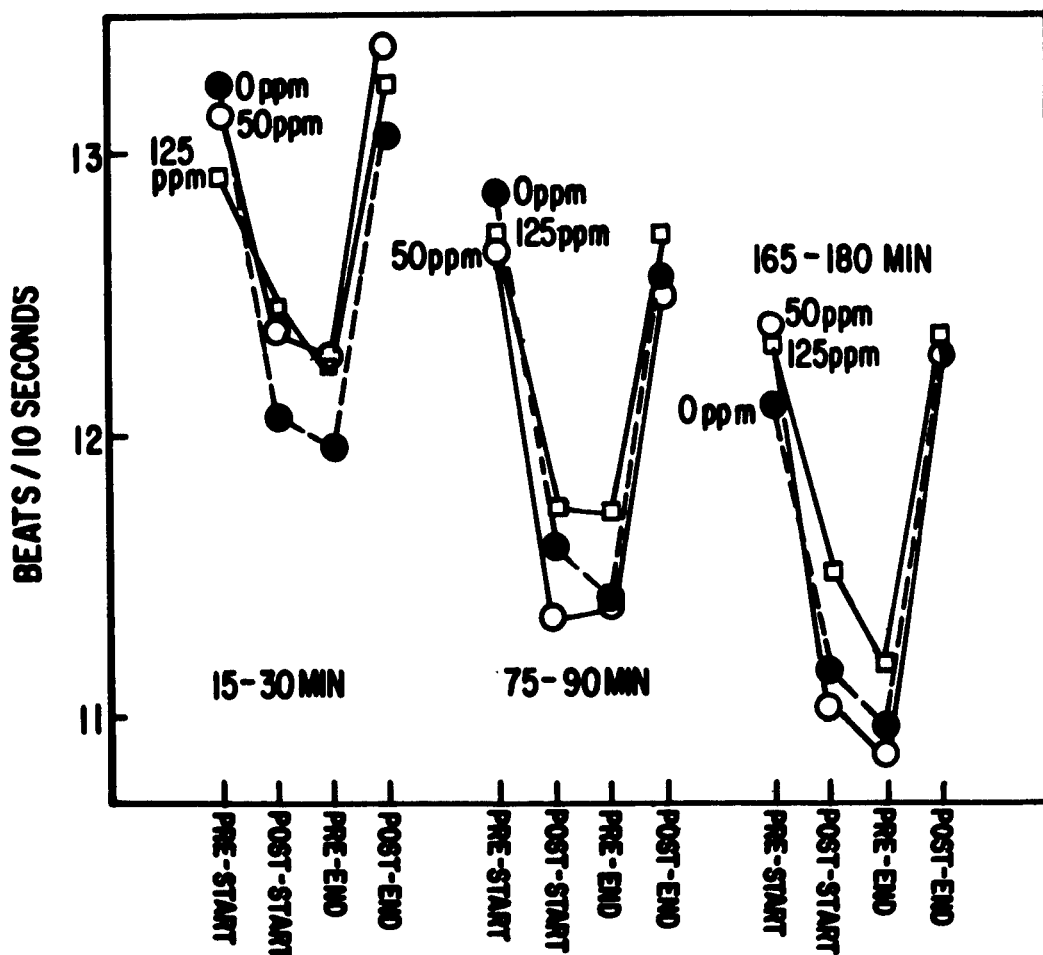


Figure 6. Heart Rate Pattern During Tracking Trials.

response only the pre-and post-start intervals were analyzed and these were assumed to reflect the pre-and post-end intervals.

Absolute Heart Rate

Figure 7 shows the absolute heart rates for the pre-and post-

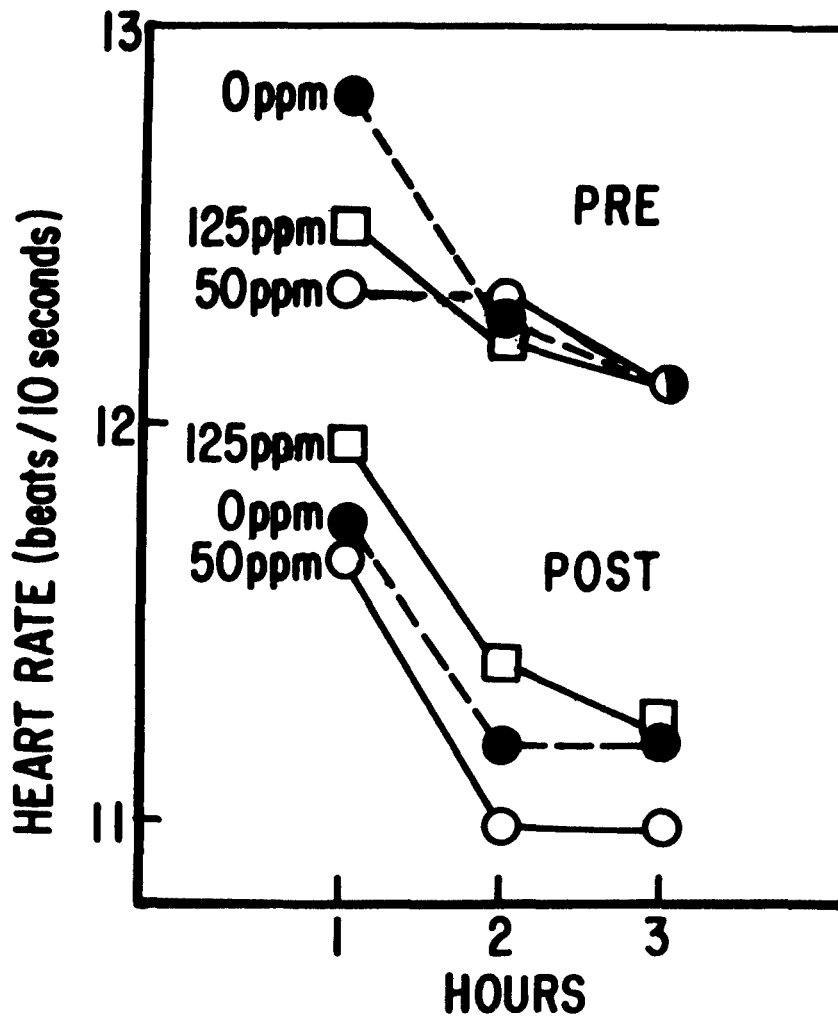


Figure 7. Absolute Heart Rates for Pre-and Post-Start Intervals Over Three-Hour Exposure.

start intervals. The only consistent pattern appears to be the slight drop in the absolute cardiac rate over the three hours of testing, the maximum decrease being on the order of 1 beat/10 seconds. Inspection of individual subjects' records revealed that this pattern is consistent over 8 out of the 9 cases. The remaining subject showed this pattern in some periods and not in others.

The absolute heart rate levels from two consecutive half-hour sessions were averaged and were analyzed by consecutive one-hour blocks. Separate analyses of variance were done comparing the three conditions at each hour. There were no significant effects of carbon monoxide in either the pre-or post conditions. Simply, the three levels of carbon monoxide did not yield any differences in the Pre-start rates, or in the Post-start rates. From these results it can be concluded that the carbon monoxide levels used in this study could not be shown to affect the absolute activity level of the heart.

Change in Heart Rate

The pattern of cardiac deceleration found during each tracking trial is consistent with previous findings on the physiological concomitant of the orienting response. Decelerative changes are commonly found in tasks where intellectual concentration is required. Therefore, it is important to consider whether the CO

exposures used here affected the magnitude of such responses. Accordingly, the heart rate for each "post-start" interval was subtracted from that during the "pre-start" interval for each scored trial, yielding a "cardiac responsiveness" score for the beginning and end of each trial. These were then averaged for each subject during a one-hour period. The grouped results of this analysis for the start periods are presented in Figure 8.

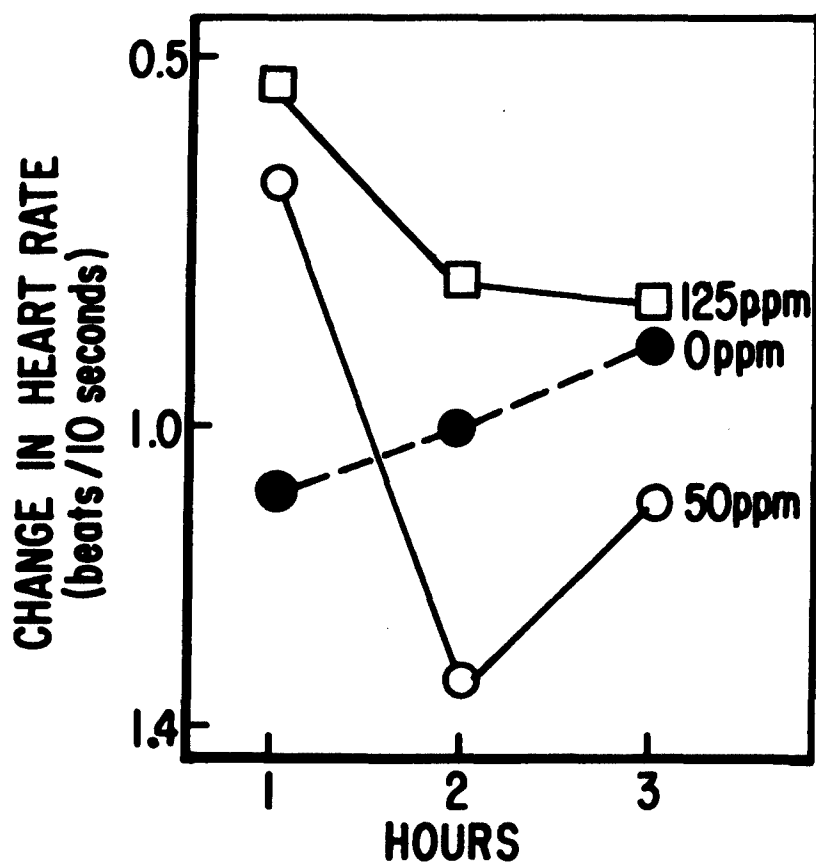


Figure 8. "Cardiac Responsiveness" To the Start of Tracking Trials Over Three-Hour Exposure.

Independent analyses of variance on these data (repeated measures) were done for each one-hour period, and these results are presented in Table V. It can be seen that significant differences in responsiveness to the start of a trial appeared in the first and second hours. Application of the Newman-Keuls test to these data (Winer, 1962) revealed that the 125 ppm condition produced significantly less deceleration than the 0 ppm condition in the first hour ($P < .05$) and significantly less deceleration than the 50 ppm condition

TABLE V
ANALYSIS OF VARIANCE TABLE ON CARDIAC RESPONSIVENESS
FOR THE PRE- POST- START INTERVAL

HOUR	SOURCE	SS	d.f.	MS	F
ONE	Between <u>Ss</u>	4.19	8	0.524	3.655**
	Within <u>Ss</u>	4.59	18	0.255	
	CO	1.44	2	0.720	
	Residual	3.15	16	0.197	
TWO	Between <u>Ss</u>	8.20	8	1.025	4.530**
	Within <u>Ss</u>	3.75	18	0.208	
	CO	1.35	2	0.675	
	Residual	2.39	16	0.149	
THREE	Between <u>Ss</u>	6.75	8	0.844	1.016
	Within <u>Ss</u>	3.47	18	0.193	
	CO	0.39	2	0.195	
	Residual	3.08	16	0.192	

** Significant at .05.

in the second hour ($P < .05$). By the third hour, no differences are found between conditions.

There is no obvious reason why cardiac responsiveness to the start of a task should be different in the first hour as a result of exposure to CO. Certainly, the cardiac differences which occurred in these subjects were not correlated with performance changes on the tracking or time estimation tasks. In view of the number of statistical tests performed and the lack of consistent patterns in these results, it is safest to conclude that any suggestion of altered cardiac responsiveness immediately upon being exposed to CO exposure should await further experimental verification.

In summary, no effect of low level CO exposure could be demonstrated on the absolute heart rate. There was some indication that the decelerative response to the onset of a task was different under CO levels, but this tentative finding cannot be fully explored within the present design.

Galvanic Skin Response (GSR) Data

Galvanic Skin response was obtained from the middle two fingers of the subject's left hand, using Telectrode 1801 disposable electrodes and Sanborn Redux paste. Skin resistance was monitored continuously. Only data from the second and fourth of each five tracking trials were used for scoring.

For each scored trial, six data points in absolute resistance were taken and subsequently converted to conductance (micromhos). Three identifiable stimulus points were chosen during each trial and the GSR response to each of these points was calculated. Each scored trial yielded six values, as illustrated in figure 9: one taken at the "ready" signal, one taken two to three seconds later, one at the start of the trial, the GSR to the start, one taken at the end of the trial, and one for the GSR to the end. These absolute conductance values were then averaged over each ten trial block, and represent the raw data used in further analyses. The grouped data for these values are presented in figure 10.

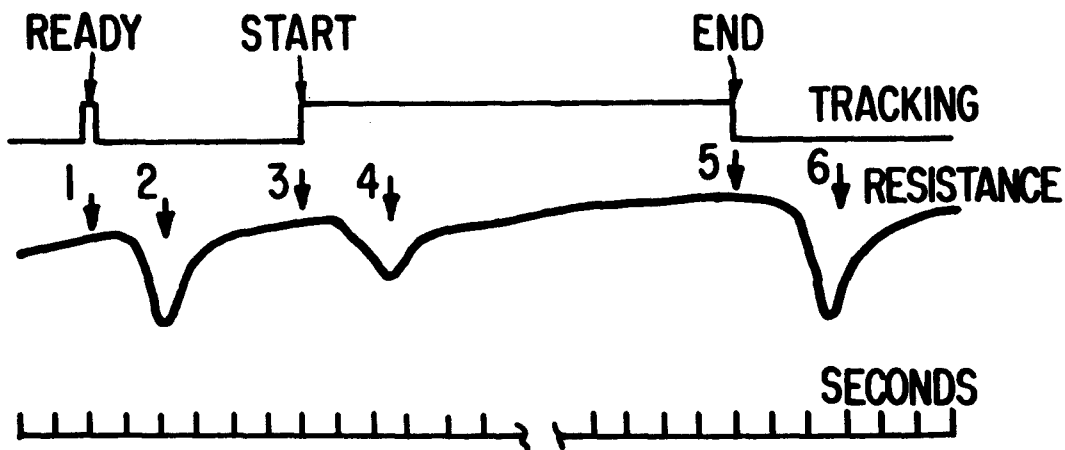


Figure 9. GSR Scoring Points for Each Tracking Trial.

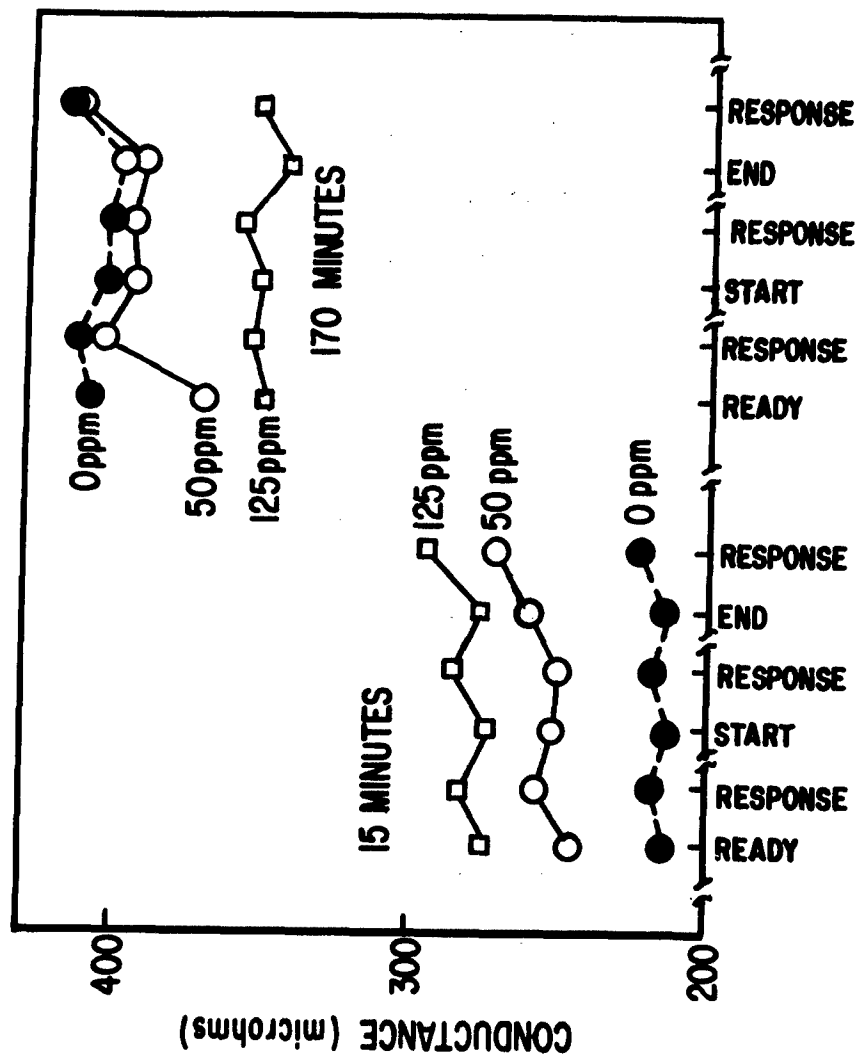


Figure 10. GSR in Micromhos During Averaged Tracking Trial At the Start (15 Min.) and End (170 Min.) of Exposure.

The general form of skin conductance changes during a tracking trial can be seen from this figure. "Conductance increases" (GSRs) occur to each stimulus event (ready, start, end). During the tracking trial itself there is a general, although not universal, tendency for the conductance level to decrease. It is also apparent that the general conductance level increased over time for all groups.

In order to determine if any GSR differences due to CO exposure could be demonstrated, separate analyses of variance were done at each time period and for each score within a given time period. None of these analyses yielded significant CO effects, and none of the F ratios approached significance. It must therefore be concluded that the CO levels used in the present study could not be shown to affect subjects' peripheral activation level as measured by the GSR.

The lack of GSR change found under the conditions of this study further confirm the previous indications that CO had little affect on the subjects. Certainly if there had been a maintenance of performance at the cost of greater or lesser activation in the subject, it should have demonstrated itself in changes in heart rate and/or GSR. No such indication was found in the present experiment.

Ataxia Test Data

As noted earlier, scores on the Ataxia test were obtained as

*

recommended by Graybiel and Fregly (1965). Subjects had been given four familiarization sessions on the test and had reached approximate plateau levels prior to being exposed in any experimental session. Details on tasks involved in each test of the battery have been presented in the procedure section and are spelled out in greater detail by the original authors.

Averaged results on all tests are shown in Table VI, along with the F ratios for the analysis of variance performed on each test. It can be seen that none of the differences between conditions are significant. In fact, in absolute terms, for four of the tests (SR,

TABLE VI
RESULTS AND ANALYSES OF VARIANCE ON ALL TESTS
OF THE PENSACOLA ATAXIA BATTERY

TEST	0 PPM	50 PPM	125 PPM	F RATIO
SR	204	221	217	0.7584
W/EO	14.00	14.13	13.75	0.1168
S/EO	32.25	29.75	30.88	0.0897
S/EC	81.13	80.75	81.13	0.0014
SOLEC-L	133.13	138.50	135.13	0.1032
SOLEC-R	140.50	150.00	141.50	1.6810
WALEC	17.13	13.38	10.50	0.9626

SOLEC-L, SOLEC-R and WALEC) the CO conditions yielded scores showing better dynamic equilibrium than the control condition. In only one case (S/EO) were scores under the CO conditions both worse than they were without CO.

In view of these results, it is clear that CO exposure had no effect on the kinds of abilities measured by these tests. It therefore seems safe to conclude that under the present conditions, no effect of CO exposure on these abilities could be demonstrated.

IV DISCUSSION

This study attempted to answer the question: does exposure to low-levels of carbon monoxide (CO) affect performance? To explore this question, a range of performance measures were taken: (1) time estimation of an "empty" interval, (2) psycho-motor tracking, (3) the Pensacola ataxia battery, and (4) heart rate and galvanic skin response (GSR). On an ordered scale, these measures run from the estimation of time, where the subject consciously supplies his own internal counting stimuli, possibly through a predominantly cortical mechanism, to the nonvoluntary, subcortical functions involved in heart rate and GSR. The results indicated that three hours of exposure to CO levels of 50 and 125 parts per million (ppm) produced no observable deterioration in functioning on the time estimation, tracking, or ataxia tasks, and no clearly reliable pattern of changes from the control (0 ppm) condition. In addition no changes indicating an overall increase or decrease in activation were observed in the heart rate or GSR measures.

The results of the present study conflict with several other studies which have found performance decrements under low levels of CO. Beard and Wertheim (1967) using a temporal discrimination task, found major disruptions under 50 ppm. Schulte (1963) using a battery of cognitive and perceptual tasks found performance disruptions with

reported carboxyhemoglobin (COHb) levels far below those obtained after three hours exposure to 125 ppm CO in the present study. Trouton and Eysenck (1961) reported impairment in control precision and multiple limb coordination in subjects when the concentration of COHb in their blood exceeded 5 per cent. However, Schulte (1963) found no change in muscular control as measured by reaction time, muscle persistence, and static steadiness up to 20 per cent COHb. In the present study, at 125 ppm CO (average COHb of 6.6 per cent) there was a lack of any evidence for a deterioration in performance on the Pensacola ataxia task, which would be sensitive to such motor impairment, or on the tracking task, which requires rapid and extremely accurate motor control.

Other studies have failed to find any decrement in performance until extremely high levels of COHb were reached. Dorcus and Weigand (1929) could only produce a decrement in psychomotor and cognitive functioning at COHb levels of 25 to 35 per cent. Vollmer, King, Birren and Fisher (1946) failed to find a deterioration in visual fields below 25 per cent COHb. Clayton, Cook, and Frederick (1960) found no relationship between COHb levels and auto accidents. Performance decrements in driving ability have been shown when COHb levels reached 20 to 25 per cent, but these could easily be overcome by increased attention (Forbes, Dill, Desilva, and Van Deventer, 1937; Rockwell & Ray, 1967; Dinman, 1968).

Ignoring obvious methodological differences and the diversity of performance measures between studies, it is difficult to account for the apparent contradictory results found in the literature, and for the differences between the present study and those indicating relatively dramatic effects at low CO levels. Working from the assumption that the primary mechanism for the effects of CO on the organism is through tissue hypoxia (Dinman, 1968) and since the central nervous system is extremely sensitive to oxygen deficiency, it could be predicted that the functions of the higher integrative cortical system would be the first to show decrement. From this viewpoint, the reported early decrements in cortically mediated tasks such as arithmetic calculations (Schulte, 1963) and temporal discrimination (Beard & Wertheim, 1967) are not surprising.

Close scrutiny of the above studies, however, raises serious questions concerning the validity of such a simple relationship. There is some reason to believe that Schulte underestimated the actual COHb levels present in his subjects, since he reports 0.00 per cent COHb in his control subjects, who were firemen from a large metropolitan city and were predominantly smokers. Recent data indicate that 1.2 per cent COHb is found in the average metropolitan non-smoker (Goldsmith & Landaw, 1968) which agrees well with the 0.96 per cent found for non-smokers under the control condition in the present study. In addition, the absolute number of errors in

arithmetic calculations reported by Shulte was higher under a reported COHb level of 1.0 per cent than under almost all of the other COHb levels up to 20 per cent. Similar questions could be raised with respect to other psychological tests reported in this study. In the study by Beard and Wertheim (1967) significant decrements in temporal discrimination were found during exposure to 50 ppm CO. It should be noted, however, that these authors confined their subjects in a sound-proof audiometer booth with a total volume of only 110 cubic feet and no outside visibility. In addition, none of the tasks involved much kinesthetic, proprioceptive, or visual input to the subjects. In view of the fact that even moderate degrees of sensory or motor restriction can cause significant perceptual and cognitive distortion (Schultz, 1965) it is possible that any CO effects reported could be accounted for, or at least are confounded, by sensory restriction effects.

The subjects in the present study were in a large dome with an approximate area of 600 cubic feet. They could see outside the dome, and were allowed to get up and walk around midway through the experiment. Additionally, the tasks involved a great deal of visual input and a significant amount of motor output. It was intended that these procedures would have the effect of minimizing the effects of sensory restriction and boredom and thus yield a less confounded estimate of CO effects. In view of this, it may not be surprising that the

present results do not show the same decrements seen in other studies. It is suggested therefore, that when the subjects' sensory and motor restriction is minimized, the effects reported in these other studies are not reproduced.

Given the results on the cortically dependent time estimation task, it is consistent to find no deterioration in performance on the tracking task used here, or on the ataxia rails. Although the tracking task involves relatively complex visual, neural, and neuromuscular circuits, it can be considered as a reflexive response to visual input, especially with the degree of overlearning given in the present case. Similarly, dynamic equilibrium appears to be primarily dependent on vestibular and kinesthetic reflexes. The present results indicate that neither of these performance measures showed a CO related decrement, which argues for a certain degree of functional integrity in these sub-cortical systems under the exposures and times used. Also, the available evidence from the heart rate and GSR indices of autonomic functioning indicated that the subject was not maintaining his pre-exposure level through an increased "activation" (i.e., he was not working harder to maintain performance under CO).

These findings have significant practical importance in many areas. They are pertinent to the situations in which humans are

exposed to CO when performing tasks of varying complexity and cortical involvement in a relatively enriched environment. Under these conditions, the human is able to perform as well after three hours of low level exposure as he did prior to, or just after, exposure. Therefore, at least certain critical aspects of the pilot's performance on landing, of the astronaut, or of the driver in traffic should not ordinarily deteriorate at these exposure levels. The results of Beard and Wertheim (1967) indicate that when similar tasks are used where conditions of CO exposure and sensory restriction are allowed to interact, the results might be quite different. It might be hypothesized that such a situation could arise where an automobile driver is on a long, monotonous road at night.

A further question can be raised concerning the performance of subjects under CO exposure; over a three-hour period, is the overall pattern of performance the same when subjects are exposed to CO as when they are not? On this question, the results of the present study are less clear. There was some indication that tracking performance was somewhat worse after 110 and 140 minutes of exposure to 50 and 125 ppm CO than after the same intervals with no exposure. However, these differences disappeared by 170 minutes. In absolute terms, these differences were small, amounting to only 6 to 10 milliseconds in the subjects' averaged reacting times. Overall,

the evidence indicates that there is no direct relationship between COHb in the blood and tracking performance. For the time estimation data, a significant difference indicating longer estimates for the group exposed to 50 ppm appeared at 140 minutes. This difference did not appear in comparisons involving the 125 ppm group and, as with the tracking data, by 170 minutes even this one difference had disappeared.

One possible explanation of these results postulates the existence of an adaptive mechanism which maintains performance at a fairly constant level even when COHb level is increasing in the blood. In this respect, the observation that the overall pattern of performance in the CO conditions was different from the control condition would be viewed as supporting the above hypothesis. In addition, the "cardiac responsiveness" changes could also indicate the existence of some adaptive mechanism acting rapidly after CO exposure to counteract its effects. Identification of such a system would be extremely difficult in view of the complexity of the physiological and performance systems involved. However, it would appear worthwhile to direct future investigation to identification of changes in patterns of performing rather than to search for absolute decrements in complex tasks.

It seems reasonable to conclude from the present study that CO, acting alone, does not necessarily produce decrements in absolute performance on several kinds of tasks. It remains for future research to determine whether the maintenance of high performance under low level CO involves a "cost" to the individual in terms of some adaptive mechanism, and, if so, whether this cost is prohibitive.

REFERENCES

- Beard, R.R., & Wertheim, G.A. Behavioral impairment associated with small doses of carbon monoxide. Amer. J. Pub. Health, 57:2012-2022, 1967.
- Clayton, G.D., Cook, W.A., & Frederick, W.G. A study of the relationship of street carbon monoxide concentrations to traffic accidents. Amer. Indust. Hyg. Assoc. J., 21:46-54, 1960.
- Dinman, B.D. Pathophysiologic determinants of community air quality standards for carbon monoxide. J. Occupat. Med., 10:446-456, 1968.
- Dominguez, A.M., Christensen, H.E., Goldaum, T.R., & Stenbridge, V.A. A sensitive procedure for determining carbon monoxide in blood and tissues utilizing gas-solid chromatography. Toxicol & Appl. Pharmacol., 1:135-143, 1959.
- Dorcus, R.M., & Weigand, G.E. The effect of exhaust gas on the performance of certain psychological tests. J. Gen. Psychol., 2:73-76, 1929.
- Forbes, W.H., Dill, D.B., DeSilva, H., & Van Deventer, F.M. The influence of moderate carbon monoxide poisoning on the ability to drive automobiles. J. Indust. Hyg. & Toxicol., 19:598-603, 1937.
- Forbes, W.H., Sargent, J., & Roughton, T.J.W. The rate of carbon monoxide by normal man. Amer. J. Physiol., 143:594-608, 1945.
- Goldsmith, J.R. & Landaw, S.A. Carbon monoxide and human health. Science, 162:1352-1359, 1968.
- Graybeil, A., & Fregly, A.R. A new quantitative ataxia test battery NSAM-919, NASA Order No. A-93, Naval School of Aviation Medicine, Pensacola, Fla., 1965.

Haldane, J.S. The action of carbonic acid on man. J. Gen. Physiol., 18:430-462, 1895.

Jex, J.R. Two application of a critical-instability task to secondary workload research. IEEE Transactions on Human Factors in Electronics, HFE 8:279-282, 1967.

Jex, J.R., McDonnell, J.D., & Phatak, A.V. A critical tracking task for manual control research. IEEE Transactions on Human Factors in Electronics, HFE 7:138-144, 1966.

Lilienthal, J.L., Jr., & Fugitt, C.H. The effect of low concentrations of carboxyhemoglobin on the "altitude tolerance of man". Amer. J. Physiol., 145:359-364, 1946.

MacFarland, R.A., Roughton, F.J.W., Halperin, M.H., & Niven, J.I. The effects of carbon monoxide and altitude on visual thresholds. J. Aviation Med., 15:381-394, 1944.

McDonnell, J.D., and Jex, H.R. A critical tracking task for man-machine research related to the operator's effective delay time: Part II. Experimental effects of systems input spectra, control stick stiffness, and controlled element order. NASA CR-674, 1967.

Moreau, H. Carbon monoxide as a test for air pollution in Paris due to motor vehicle traffic. Proc. Roy. Soc. Med., 57:1015-1020, 1964.

Rockwell, T.H., & Ray, A.M. An exploratory study of automobile driving performance under the influence of low levels of carboxyhemoglobin. Technical Report No. 1E-8, Systems Research Group, The Ohio State University, Columbus, Ohio.

Rosenberg, B.R., & Jex, J.R. A survey of stressors affecting the tracking performance and effective delay time of astronauts. Systems Technology, Inc., Working Paper No. 155-8, Hawthorne, Calif., 1966.

Sadoff, M., & Jex, J.R. Apollo applications human transfer function experiment, T007. Systems Technology, Inc., Working Paper No. 175-9, Hawthorne, Calif., 1968.

Schulte, J.H. Effects of mild carbon monoxide intoxication. Arch. Environ. Health, 7:524-530, 1963.

Schultz, D.P. Sensory Restriction: Effects on Behavior. Academic Press, New York, 1965.

Thomas, A.A. Low ambient pressure environments and toxicity. Arch. Environ. Health, 11:316-322, 1965.

Trouton, D., & Eysenck, H.J. The effects of drugs on behavior, Handbook of Abnormal Physiology, Eysenck, H.J., editor, pp 634, New York, Basic Books, Inc., 1961.

Vollmer, E.P., King, B.G., Birren, J.E., & Fisher, B.M. The effects of carbon monoxide on three types of performance at simulated altitudes of 10,000 and 15,000 feet. J. Exp. Psychol., 36:244-251, 1946.

Winer, B.J. Statistical Principles in Experimental Design, McGraw-Hill Book Co., Inc., New York, 1962.