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QUANTITATIVE EFFECTS OF ABRUPT DECELERATION
ON
PULMONARY DIFFUSION IN MAN

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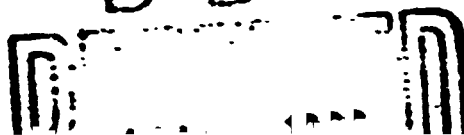
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6571st Aeromedical Research Laboratory
Aerospace Medical Division
Air Force Systems Command
Holloman Air Force Base, New Mexico

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FOREWORD

This study was made by the Bio Effects Division of the 6571st Aeromedical Research Laboratory under NASA Contract No. T-13335G. The research was conducted from 1 September 1965 to 1 February 1966.

This technical report has been reviewed and is approved for publication.



C.H. KRATOCHVIL, Lt Colonel, USAF, MC
Commander

ABSTRACT

Pulmonary diffusion capacity was measured in nine subjects using the steady state method to determine if this physiological measurement was altered by impact. Each subject rode the Daisy Decelerator twice backward (+G_x) at 25 G, twice laterally (-G_y) at 15 G, and experienced one sham ride. Carbon monoxide diffusion capacities were measured immediately before and after each ride, and 3 and 24 hours afterwards. No significant change in pulmonary diffusion capacity was associated with impact. There was no correlation between observed DL_{CO} and predicted DL_{CO} based on oxygen consumption, however, observed DL_{CO} and oxygen consumption were highly correlated. The validity of the prediction formula as described by Donevan et al., (Ref. 4), is questioned, but this may be related to the increased altitude (1400 ft) at which the studies were done.

I

INTRODUCTION

Little is known of the physiological effects of abrupt deceleration (impact) on man. Present knowledge of the effects of impact at levels not producing obvious injury is primarily subjective. Human subjects sustaining high G rides on the Daisy Decelerator of the 6571st Aeromedical Research Laboratory frequently complain of dyspnea for up to one minute following impact. High speed photography reveals marked chest movement during (+G_x) impact with compression of the chest to approximately one-half the initial transverse diameter.

Bears which have sustained high G impact rides show severe intrathoracic trauma (Ref. 1). Pulmonary trauma consists mainly of intra-alveolar hemorrhage with disruption of alveolar membranes. Similar studies performed on chimpanzees reveal pulmonary hemorrhage and formation of petechiae (Ref. 2). It appears as if the intrathoracic and intracranial contents are most susceptible to trauma from severe impact (Ref. 1). No apparent injuries have occurred to either of these areas in studies on humans undergoing impact testing at what is considered to be subjectively tolerable levels of impact.

Pulmonary diffusion capacity (D_{LCO}) appears to be reduced under continual G forces as produced on human centrifuges but is most probably related to ventilation-perfusion inequalities (Ref. 3). The authors, therefore, postulated that if abnormal physiological function was produced by impact at levels which produce no subjective or objective trauma, that it would most likely occur to the intrathoracic or intracranial contents. Carbon monoxide diffusion capacity (D_{LCO}) may be considered a sensitive measure of pulmonary function, thus it was hypothesized that inapparent trauma to the lungs, (i.e., petechiae or disruption of alveolar membranes) would alter pulmonary diffusion capacity. The current research program to measure steady state carbon monoxide diffusion capacities was undertaken using human subjects, to evaluate the effects of impact rides which were subjectively tolerable without objective changes in chest X-rays or physical examinations.

II

METHODS AND MATERIALS

Ten volunteer subjects were chosen from the pool of sled subjects maintained by the 6571st Aeromedical Research Laboratory for the purpose of impact testing. All had normal physical examinations, electrocardiograms, and chest X-rays and were in excellent physical condition. One subject failed to complete the series due to a tympanic membrane perforation on a lateral ride, probably due to pressure effect of the ear impacting the head rest. The perforation subsequently healed, but the data were eliminated because of failure to complete the testing sequence. Impact testing was performed on the Daisy Decelerator at velocities of 30 feet/second and onset of 1200 to 1400 G/second. Tests were conducted in the backward orientation (+G_x) at 25 G and in the right lateral orientation (-G_y) at 15 G. In the lateral position, head and torso enclosures were used. The restraint system consisted of lap and V-belt and shoulder harness. G forces recorded on accelerometers on the subjects ranged from 30 to 60 G in the direction of the applied force. Each subject sustained two rides at 25 G in the backward orientation and two rides at 15 G in the lateral orientation. Each subject experienced one sham ride in either of the two positions in which the entire operational procedure was followed but the firing pressure was reduced and the sled did not reach the brake. The subject was not aware that the sham ride would occur.

Steady state pulmonary diffusion capacities were measured using the method of Donevan, et al., (Ref. 4) modified as described below. One tenth percent carbon monoxide in air was used as the test gas. The subject walked at 2.5 miles per hour on a 4 degree (7 percent) grade breathing room air for 4 minutes and then switched to the carbon monoxide mixture for 5 minutes to insure that stabilization of expired carbon monoxide had occurred. Following this, expired air was collected for 1 minute in a 120 liter Tissot Spirometer.¹ Gas temperature, volume, exact time of collection, and respiratory rate were recorded and the expired

¹ Warren E. Collins, Inc., 220 Wood Road, Braintree, Massachusetts

air was analysed for oxygen and carbon dioxide using a Beckman F-3 oxygen analyser.² The actual concentration of oxygen was read on a John Fluke voltmeter.³ Carbon monoxide was continually monitored on a Beckman IR 215 Infrared Spectrophotometer.⁴

Background carbon monoxide measurements were performed by breathing 100 percent oxygen for 3 minutes followed by rebreathing for 3 minutes into a 13-liter anesthesia bag through carbon dioxide and water absorbants. This measurement performed before and after each diffusion study was used to calculate a correction factor for carboxyhemoglobin back pressure. The results indicated that correction for carbon monoxide uptake as described by Linderholm (Ref. 5) yields approximately the same results as measuring a total background at the end of each study. Physiological dead space was assumed on the basis of tidal volume as described by Asmussen and Nielsen (Ref. 6). Using an average atmospheric pressure of 660 mm. hg. at Holloman AFB, alveolar P_{O_2} was calculated to be approximately 510 mm. hg. after rebreathing² O_2 . This value was verified by measuring the alveolar P_{O_2} after rebreathing O_2 in four subjects and averaged 507 mm. hg. Predicted D_{LCO} was based on oxygen consumption and age. The observed D_{LCO} varied from 110 to 140 percent of the predicted values, and D_{LCO} could be reproduced to within 5 percent in each subject over any 24-hour period. The increase in observed D_{LCO} over the predicted values were assumed to be due to increased carbon monoxide uptake at an altitude of 4,400 feet where the average hemoglobin content of the subjects is expected to be 10 to 20 perc at higher than at sea level.

Baseline pulmonary diffusion studies were made from 3 to 5 times on consecutive days in each subject to both train the subject in the use of respiratory equipment and to determine a valid baseline measurement.

Impact rides were conducted at intervals of not less than 10 days and diffusion capacity was measured immediately before and after each ride and at 3 and 24 hours following the ride. Figure 1 is a schematic diagram of the pulmonary diffusion apparatus and the rebreathing apparatus. All data were transferred to punch cards for computation and statistical analysis.

²Beckman Instruments, Corp., Fullerton, California

³John Fluke Manufacturing Co., Seattle, Washington

⁴Beckman Instruments, Corp., Fullerton, California

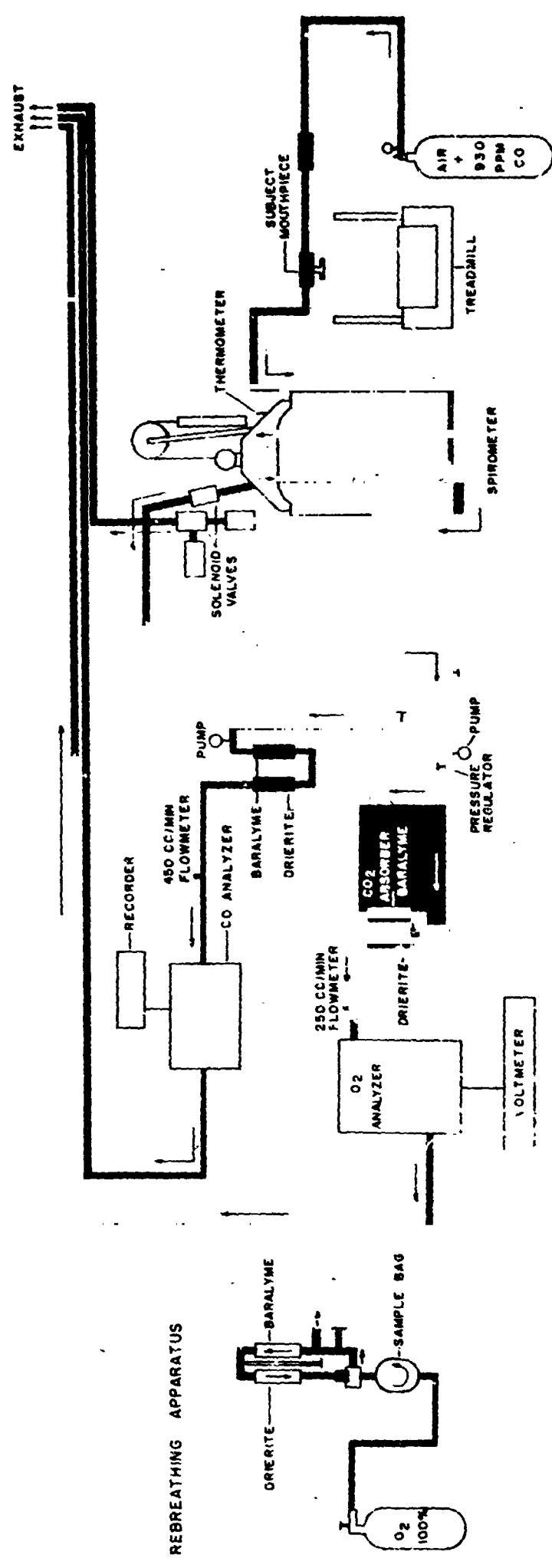


Figure 1. Schematic Diagram of Steady State Pulmonary Diffusion Apparatus

III

RESULTS

The individual values for all nine subjects on whom complete data were obtained is summarized in Table I. For each subject, there are results for two separate runs under the $+G_x$ and $-G_y$ columns and for the single sham run. Table II presents the mean values for all subjects for each time and position. The significance of the results was tested using a three-way analysis of variance comparing subjects, positions and time. The main effects due to subject and position and the subject-position interaction effect are all highly significant ($p = < .0005$). The variations between subjects was recognized prior to the test, and was not a significant consideration. The high significance associated with the subject-position interaction merely reflects the variation in performance of several of the subjects over the 3-month period in which the study was conducted. Further analysis of the position effect shows that the difference between the sham run and the lateral facing runs is within the 99 percent confidence limits but the difference between the backward facing and the sham runs is insignificant. In addition to being an indication of the effect of direction of impact, the position effect also is a function of a possible long-term time effect, as the backward-facing runs were all conducted during the first half of the study, and the sham runs were randomly conducted during the first two-thirds of the study. To get a better understanding of the significance of the position effect, the data were normalized and each pre-run test was considered as a control for a particular run. Analysis of the normalized data shows that there is no significance to the position effect. This would indicate that the observed change was not a function of position but a shift in the subject's baseline over the 3-month period of the study, and is related to adaptation to the equipment and loss of anxiety concerning breathing carbon monoxide.

Each computed diffusion capacity (D_{LCO}) was compared with a predicted value based on the formula:

$$D_{LCO} \text{ (predicted)} = 37 - (.53 \times age) + (8.75 \times O_2 \text{ consumption})$$

TABLE I. COMPOSITE DATA OF ALL PULMONARY DIFFUSION CAPACITIES

SUBJECTS	SHAMS				+G _x (BACKWARD)				-G _y (LATERAL)			
	PRE	POST	3 HR.	24 HR.	PRE	POST	3 HR.	24 HR.	PRE	POST	3H	24HR.
(C.A.)	46.7	45.9	41.8	48.9	49.5	46.6	46.6	46.6	45.3	42.1	42.6	44.9
(R.B.)	39.3	39.5	35.9	39.7	45.8	43.5	45.3	40.3	42.7	39.6	35.4	33.1
(L.B.)	56.4	47.3	51.1	59.2	50.9	49.5	54.7	54.5	51.0	53.2	51.4	54.2
(P.C.)	57.9	55.8	49.9	55.3	52.2	55.9	51.4	53.9	43.8	42.9	40.8	44.7
(P.F.)	44.9	46.8	37.9	45.9	40.7	42.2	38.6	38.2	57.7	46.5	47.7	43.2
(B.L.)	34.9	35.9	36.6	32.8	40.4	42.4	41.2	37.7	37.6	39.3	37.8	38.9
(T.M.)	40.3	43.3	35.3	41.5	39.2	38.2	39.0	38.7	42.2	43.2	37.7	35.4
(J.S.)	51.5	51.5	52.6	56.7	53.4	46.5	49.4	45.8	49.3	45.6	44.2	45.1
(R.T.)	46.7	43.2	43.1	42.6	48.3	47.2	46.2	43.6	41.5	39.0	43.9	42.1
					43.4	43.3	43.1	44.2	44.6	42.1	40.4	47.6

TABLE II. AVERAGE VALUES FOR ALL SUBJECTS FOR TIME AND POSITION

	PRE	POST	3 HR.	24 HR.
SHAM	46.3	44.9	42.2	46.0
BACKWARD (+G _x)	45.6	44.4	45.6	44.9
LATERAL (-G _y)	45.7	42.9	42.5	43.1

TABLE II. AVERAGE VALUES FOR ALL SUBJECTS FOR TIME AND POSITION

	PRE	POST	3 HR.	24 HR.
SHAM	46.3	44.9	42.2	46.0
BACKWARD (+ G _x)	45.6	44.4	45.6	44.9
LATERAL (- G _y)	45.7	42.9	42.5	43.1

This formula was derived by Donevan, et al. (Ref. 4) in BTPS terms and converted to STPD as described by Richert (Ref. 7). It was expected that graphic portrayal of D_{LCO} would yield two lines which were parallel. Figure 2 presents graphically values for two of the nine subjects which are representative of the entire group. These lines are markedly divergent as well as showing the observed D_{LCO} to average 110 to 150 percent of the predicted value. This latter observation is in part attributable to the altitude of 4400 feet and barometric pressure of 660 mm. hg. under which the studies were done, assuming that carbon monoxide uptake is related to hemoglobin concentration, which is one to two grams higher on the average at this altitude; however, the divergency of the lines is not explained entirely by increased hemoglobin concentration.

The observed D_{LCO} 's for the entire group versus the corresponding oxygen consumptions were compared graphically. Figure 3 provides a regression line based upon a highly significant correlation coefficient (r) of 0.42 ($p = < .001$) with a standard error of estimate (σ_{xy}) of ± 5.9 . The relationship of oxygen consumption to D_{LCO} is obviously very high in this group in which age is a fairly restricted variable (24 to 30 years).

Two possible theories are advanced to explain these observations. First, that the prediction formula for D_{LCO} as presented by Donevan (Ref. 4) is incorrect, either due to its inapplicability of prediction in a group of subjects with a narrow age range, or second, where the altitude has not been taken into consideration. These data do not answer these questions nor the question of whether or not D_{LCO} is truly a measure of diffusion, but rather may only be a measure of the capacity of the hemoglobin to capture carbon monoxide and bind it in the same way oxygen is bound.

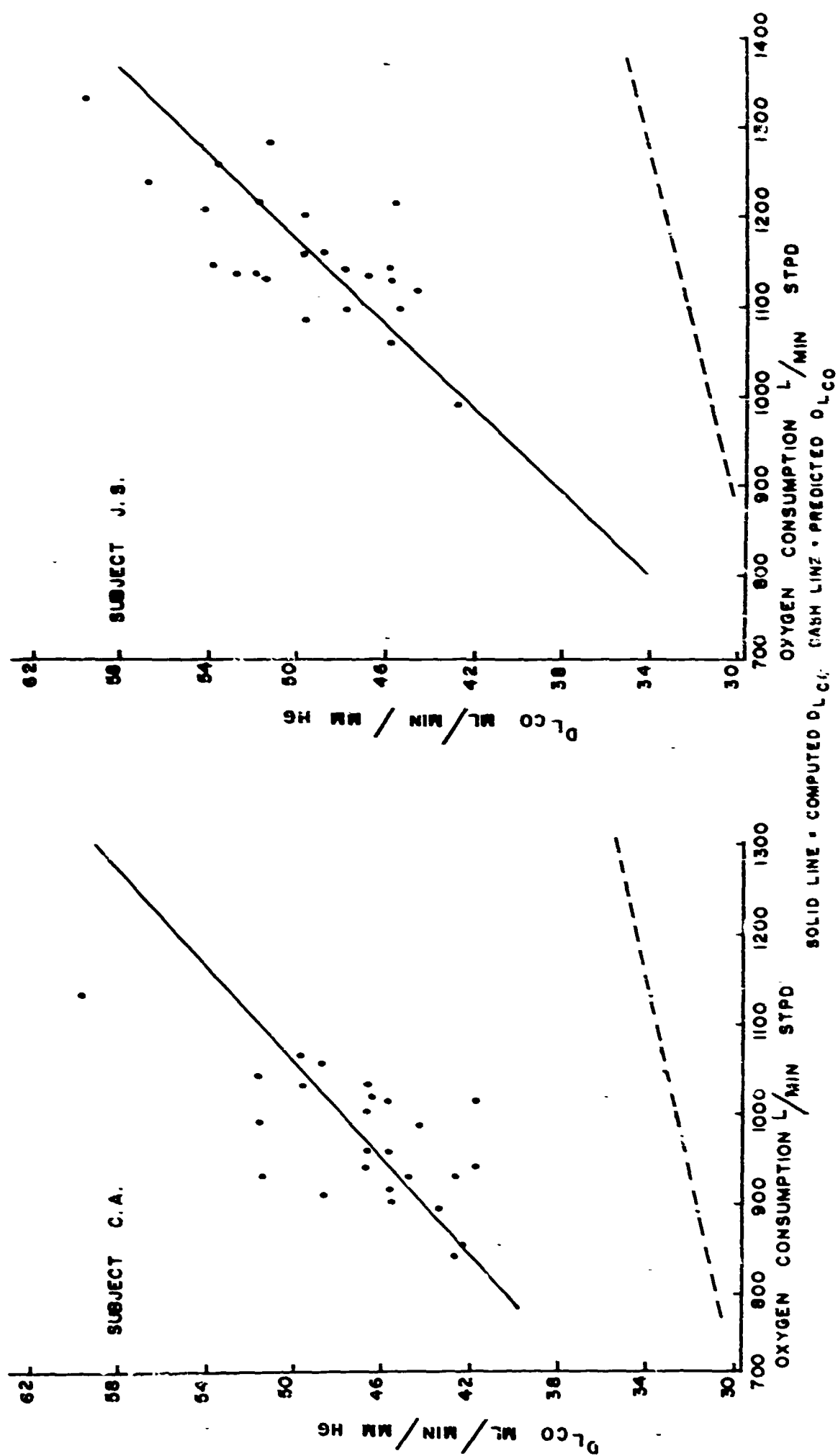


Figure 2. Representative Graphs of DL_{CO} vs. Oxygen Consumption for Observed and Predicted Values of Two Subjects

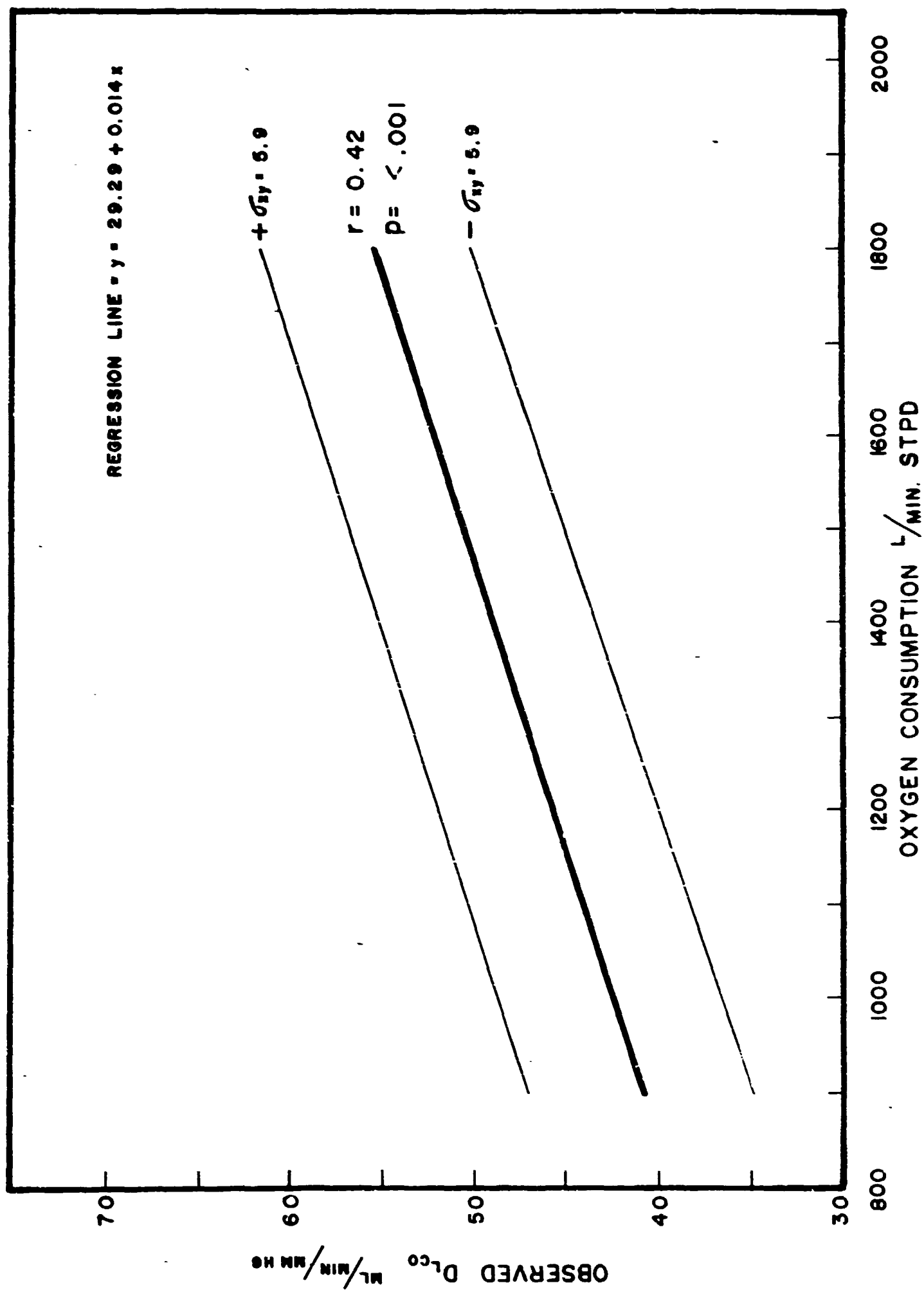


Figure 3. Observed D_{LCO} vs. Oxygen Consumption for all Nine Subjects

IV

CONCLUSION

If measurement of carbon monoxide diffusion capacity is a true measure of membrane diffusion, the data derived from this study suggest that impact at this level produced no changes in this physiological function. The levels of impact used were less than those producing pulmonary hemorrhage and petechial in animals (approximately 40 G and above), but man generally seems to be more susceptible to the effects of impact than are animals. Whether pulmonary changes occur at levels of 15 - 25 G or not is problematical--they are not demonstrable by X-ray or obvious symptomatology. Lack of abnormality of pulmonary diffusion capacity after impact lends credence to the lack of clinical evidence of pulmonary damage but should not be construed as being conclusive.

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APPENDIX

CARBON MONOXIDE DIFFUSION CAPACITY WORK SHEET FOR CALCULATIONS

Name _____ P_B _____ Temp _____
 Age _____ Wt. _____ lbs. Time of collection _____ min.
 Exercise: mph _____ Grade _____ Time total _____ min. Time on CO _____ min.
 Tissot readings: Initial _____ Final _____ Diff _____ x _____ = _____ L.
 $F_{I_{O_2}}$ _____ $F_{I_{CO_2}}$ _____ $F_{I_{CO}}$ _____ $F_{I_{N_2}}$ _____ $F_{E_{O_2}}$ _____
 $F_{E_{CO_2}}$ _____ $F_{E_{N_2}}$ _____ $F_{E_{N_2}} = \frac{F_{E_{N_2}}}{F_{I_{N_2}}} =$ _____ $F_{E_{CO(uncorr)}}$ _____ x
 $(1 - F_{E_{CO_2}}) = P_{E_{CO}} =$ _____ (corr) Resp. rate _____

$$V_E \text{ (L, uncorr)} \times \text{Corr Factor} = \frac{V_E}{V_E} \text{ L(STPD)}$$

$$\times \text{Corr Factor} = \frac{V_E}{V_E} \text{ L(BTPS)}$$

$$V_{E(STPD)} \div \text{Time} \text{ min.} = \dot{V}_E \text{ L/min. (STPD)}$$

$$V_{E(BTPS)} \div \text{Time} \text{ min.} = \dot{V}_E \text{ L/min. (BTPS)}$$

$$\dot{V}_E \div \text{Resp. rate} \text{ /min} = V_T \text{ L(BTPS)}$$

$$V_{D(\text{Subject})} \text{ cc } V_{D(\text{Valve})} \text{ cc } V_{D(\text{Total})} \text{ cc}$$

$$\dot{V}_{O_2} = \dot{V}_E (STPD) \left[\frac{F_{E_{N_2}}}{F_{I_{N_2}}} (F_{I_{O_2}}) - F_{E_{O_2}} \right] = \text{cc (STPD)}$$

$$\dot{V}_{CO_2} = \dot{V}_E (STPD) \left[F_{E_{CO_2}} - \frac{F_{E_{N_2}}}{F_{I_{N_2}}} (F_{I_{CO_2}}) \right] = \text{cc (STPD)}$$

$$R = \frac{V_{CO_2}}{V_{O_2}} =$$

$$\dot{V}_{CO} = \dot{V}_E (STPD) \left[\frac{F_{E_{N_2}}}{F_{I_{N_2}}} (F_{I_{CO}}) - F_{E_{CO_2}} (\text{corr}) \right] = \text{cc (STPD)}$$

Background CO:

$$F_{SCO} \text{ (background)} = \underline{\hspace{2cm}}$$

$$P_{ACO} \text{ (background)} = F_{ACO} \times (P_B - 47) = \underline{\hspace{2cm}}$$

$$S_{CO} \text{ (background)} = \frac{210 \times 100 \times P_{ACO}}{P_{O_2} + (P_{ACO} \times 210)} = \underline{\hspace{2cm}} \quad (1)$$

Mean Capillary CO:

$$S_{CO} = \frac{\dot{V}_{CO} \times T(\text{min}) \times 15.625}{\text{Wt. (lbs)}} = \underline{\hspace{2cm}} \quad (2) \quad \text{Total } S_{CO} = (1) + (2)$$

$$P_{CCO} = \frac{S_{CO}}{210} = \underline{\hspace{2cm}}$$

Alveolar CO:

$$P_{ACO} = \frac{B-47}{V_{T(BPTS)} - V_{D(Total)}} \times \left[(F_{ECO} \times V_T) - (F_{ICO} \times V_D) \right] = \underline{\hspace{2cm}}$$
$$P_{ACO} - P_{CCO} = \underline{\hspace{2cm}}$$

Diffusion:

$$D_{LCO} \text{ (Observed)} = \frac{\dot{V}_{CO}}{P_{ACO} - P_{SCO}} = \underline{\hspace{2cm}}$$

$$D_{LCO} \text{ (Predicted)} = 37.7 - (0.53 \times \text{age}) + (8.75 \times \dot{V}_{O_2}) = \underline{\hspace{2cm}}$$

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KEY WORDS

Man
Abrupt deceleration
Pulmonary diffusion capacity
Steady state method
Oxygen consumption
Physiological effects

LINK A

LINK B

LINK C

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