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THE EFFECT ON THE CHIMPANZEE OF RAPID DECOMPRESSION TO A NEAR VACUUM

Edited by Alfred G. Koestler

Prepared under Contract No. T-27210G *by*
AIR FORCE SYSTEMS COMMAND
Holloman Air Force Base, New Mexico
for Manned Spacecraft Center

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ABSTRACT

The Effect on the Chimpanzee of Rapid Decompression to a Near Vacuum

6571st Aeromedical Research Laboratory
Holloman Air Force Base, New Mexico

Eight chimpanzees, used in nine separate tests, were decompressed from 179 mm Hg (100 per cent oxygen) to less than 2 mm Hg in 0.8 seconds and remained at this altitude from 5 to 150 seconds. After recompression to 179 mm Hg (again breathing 100 per cent oxygen), the subjects were kept at this altitude for 24 hours. Performance by all animals, on a complex operant schedule presented during and following rapid decompression, reached a baseline level of performance within a four hour post-decompression period. No central nervous system damage (as measured by behavior) could be detected.

Cortical EEG, ECG, and respiration were recorded before, during, and following decompression. Visual analysis of recorded physiological parameters was conducted and correlations with performance were attempted. EEG fast activity (10-12 cps) always preceded the end of the period of total behavioral impairment, while total behavioral recovery followed the return of normal EEG patterns.

Surgical procedures for implanting chronic cortical leads were developed. All subjects showed slight neutrophilia, increased transaminase, and facial edema which returned to normal within 72 hours after decompression. All subjects survived in good health and no lasting effects of rapid decompression to a near vacuum could be detected.

FOREWORD

This research program was accomplished between 1 July 1964 and 31 January 1965 by the 6571st Aeromedical Research Laboratory, Holloman Air Force Base, New Mexico, under contract (T-27210G) with the Environmental Physiology Branch, NASA Manned Spacecraft Center, Houston, Texas. Acknowledgment is made of contract monitorship by Drs. Wayland Hull and John Billingham of the Environmental Physiology Branch and of their continual interest in and enthusiasm for this research.

Special recognition is due the following technical and administrative support personnel of the 6571st Aeromedical Research Laboratory for their outstanding efforts throughout all phases of this program.

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A1C Jim Frasier - Drafting and Charts

Mrs. Sandra Gibson and Mrs. Evelyn Burley - Drafting of the report

Mrs. Bruna West - Typing of final copy

As a result of the findings in this research a replication of the tests involving 90, 120, and 150 second exposures will begin in April 1965. This will permit a more reliable statement of the limits of impairment and recovery which is needed for manned spaceflight programs at the earliest possible date.

This report has been reviewed and is approved for publication.

C. H. KRATOCHVIL
Major, USAF, MC
Commander
6571st Aeromedical Research Laboratory

CARE AND HANDLING OF THE SUBJECTS

The animals used in this study were handled in accordance with the "Principles of Laboratory Animal Care" established by the National Society for Medical Research.

ABBREVIATIONS AND SYMBOLS

AM	auditory monitoring
ARB	total avoidance return to baseline
CA	continuous avoidance
CARB	continuous avoidance return to baseline
DA	discrete avoidance
ECG	electrocardiogram
EEG	electroencephalogram
LP	lever presses
RD	rapid decompression
RT	reaction time
RTB	return to baseline
SRK	stimulus response key
TBR	total behavior recovery
TTI	time of total impairment
TUC	time of useful consciousness
VM	visual monitoring
$\overline{X}$	mean
N	number of cases
σ	standard deviation
t	Student's small sample test of significance of difference between means
r	Pearson's product moment correlation coefficient
r_{yx}	Pearson's product moment correlation coefficient (predicting y from x)

η	correlation coefficient for non-linear relations
R	Doolittle's multiple correlation coefficient
$F_{r/\eta}$	test of significance of difference between correlation coefficients
X	abscissa (X) axis in regression equation
Y'	ordinate (Y) axis in regression equation
σ_{yx}	standard error of the mean (predicting y from x)

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SECTION I

PERFORMANCE

Alfred G. Koestler
Herbert H. Reynolds, Major, USAF
Lewis M. Barker, A3C, USAF
Neil D. Catone, SSgt, USAF
Gordon L. Wilson

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

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ABSTRACT

Eight chimpanzees, used in nine separate tests, were decompressed from 179 mm Hg (breathing 100 per cent O_2) to less than 2 mm Hg in 0.8 seconds and remained at this altitude from 5 to 150 seconds. After recompression to 179 mm Hg (again breathing 100 per cent O_2), the subjects were kept at this altitude for 24 hours. Performance by all animals, on a complex operant schedule presented during and following rapid decompression, reached a baseline level of performance within a 4-hour post-decompression period. No central nervous system damage (as measured by performance) could be detected and all subjects survived in good health.

INTRODUCTION

Background

Prior to World War II, research on exposure to high altitude was primarily academic. With the advent of aircraft capable of flying above 12,000 meters the problems and the resulting research became more pragmatic. In 1940 investigations were undertaken concerning rapid decompression, as experienced during pressure loss in an aircraft. These investigations dealt exclusively with the physiological effects of decompression from sea level to altitudes between 13,700 and 22,000 meters. The altitude ceiling was dictated by the performance capabilities of military and civilian aircraft.

As early as 1670 Boyle (Ref. 2) exposed a viper to a high vacuum and observed a bubble within its eye, but the first systematic observations on the effects of low ambient pressure were made by Hoppe-Seyler in 1857 (Ref. 9), who found formation of gas bubbles in the blood. In 1909 Greenwood and Hill (Ref. 7), as well as others, ascribed these bubbles to nitrogen or air boiling out, while Armstrong (Ref. 1) credited them to water vapor. Polak and Adams (Ref. 12) concluded in 1932 that binding of the chest would prevent such aerebullosis, but Whitehorn, Lein, and Hitchcock (Ref. 18) showed that thoracic and abdominal bindings not only failed to protect the subject but also had detrimental effects during and after decompression.

When dogs, rabbits, and rats were subjected to explosive decompression from 2,400 to 13,700 meters in .019 seconds, Smith (Ref. 15) found that only 50 per cent of the rabbits survived, but no ill effects were experienced by dogs and rats. In 1944, Sweeney (Ref. 16) failed to detect any evidence of pathological changes in humans as a result of decompression from 2,500 to 10,700 meters in .09 seconds. Whitehorn, Lein, and Edelmann (Ref. 17) decompressed dogs from sea level to 18,300 meters and beyond but returned them almost immediately to sea level, all subjects surviving the experimental conditions.

In 1948, Hitchcock (Ref. 8) decompressed 150 human subjects at altitudes of from 3,050 to 10,700 meters and from 6,100 to 12,200 meters. The subjects were maintained at these altitudes for 60 and 90 minutes, respectively, after decompression. No significant changes were found in ECG recorded during the decompression test. Neither roentgenograms nor audiograms, taken before and after the experiment, revealed any noticeable pathology. However, Burch, Kemp, and Vail (Ref. 3) found serious damage to the myocardium of dogs when they were decompressed to an altitude of 21,950 meters in .02 seconds and exposed to this altitude for 60 seconds. Somewhat later, Henry (Ref. 6) and Luft (Ref. 11) indicated that automatic pressure garments would be essential for survival in cases of rapid decompression to altitudes beyond 15,250 meters. Luft demonstrated that rapid decompression of humans to altitudes of 10,700 meters and above was not accompanied by unconsciousness unless the exposure time exceeded 5-6 seconds at any altitude above 10,700 meters.

In 1955 Hall (Ref. 5) designed an experiment primarily to separate the effects of rapid decompression, hypoxia, and air embolism. A performance task was provided to evaluate disorientation in one phase of the experiment by training ten rats to discriminate between black and white escape alleys. Hearing loss was estimated by means of a conditioned escape response (crawling on a safety perch) to a 3,000 cycle tone of threshold intensity. When decompressed from zero to 18,300 meters and then immediately returned to sea level, the trained rats displayed no discernible disorientation, i.e., their behavior did not vary significantly from their pre-exposure conditioned behavior. Untrained rats were apparently afflicted by gross labyrinthine disorientation. Reaction times to the auditory stimuli were adversely affected during the auditory conditioning phase. The subjects seemingly heard the stimulus but had difficulty making the required escape response.

Since manned orbital flights have paved the way for long-term space exploration, the altitude ceiling has been raised to environmental pressures of 2 mm Hg and less. The occurrence of decompression accidents involving astronauts, such as cabin puncture by cosmic particles or suit rupture

during extra-vehicular exploration, has become a distinct possibility. The possibility of such occurrences has necessitated research on the effects of decompression to a vacuum on unprotected and performing organisms. A somewhat less exotic, yet equally impelling, reason has prompted studies of this kind. Missile components, entire spacecraft units, and various instruments which operate in pressureless environments must be tested in large decompression chambers. Personnel working in and around decompression chambers will be exposed to hazards the extent of which must be determined before mission rules and safety measures can be devised. Consequently, in 1963-1964 Dunn, Bancroft, and Haymaker (under NASA contract) decompressed 125 dogs to pressure levels of 2 mm Hg and less. Ninety-two dogs were autopsied within 30 minutes, 3 to 5 days, and 1 to 3 weeks post rapid decompression (RD). Their major findings are summarized in the following statement:

> "The most interesting finding was the absence of major pathological damage except in the lungs unless the exposure time exceeded 120 seconds. By varying the exposure time and the time of decompression (altitude to less than 2 mm Hg), it was possible to separate the pathological effect of anoxia versus time of decompression. In all dogs the severity of lung damage increased with the duration of the anoxic exposure. In groups with comparable exposure times, the dogs decompressed in 1 second exhibited pulmonary congestion, edema, and hemorrhage, while those decompressed in .2 seconds showed predominently more petechial hemorrhage and emphysematous changes. Denitrogenation appeared to reduce the incidents of severity of lung damage . . . Animals autopsied at later dates showed evidence of all lesions, especially in the lungs. For the exposures of more than 120 seconds, gross examinations of the brain and other organs showed increasing amounts of congestion and hemorrhage."

A further NASA effort was that of Rumbaugh (Ref. 14) who tested the learning ability of the squirrel monkey after exposure to a near vacuum. Thirty subjects were assigned to four groups on the basis of pre-RD performance, which consisted of 11 trials on a 100-object discrimination problem. Group 1 (N=10) constituted the control group. Group 2 (N=10) was decompressed from 180 mm Hg to less than 2 mm Hg in 1 second and then recompressed after 3 seconds to 760 mm Hg within 30 seconds. Group 3 (N=5) was decompressed to the same altitude in 1 second, held there for 60 seconds, and then recompressed to 760 mm Hg within 5 seconds. Group 4 N=5) differed from Group 3 only in the exposure time to altitude which was maintained for 90 seconds. Upon recovery the animals were retested. Two fatalities were encountered: one in Group 2 and one in Group 4. The survivors experienced no loss in learning set proficiency. Longer recovery times to normalcy were observed in animals which were held at 2 mm Hg for longer durations. Short-term interference with vision and hearing was noted.

The Russians have also evidenced interest in studies of exposure to decreased pressure environments. Ivanov (Ref. 10), using 20 young human adult males, found great individual differences in the appearance of subcutaneous emphysema and pain when his subjects' wrists were exposed to pressures of 41 and 8.5 mm Hg for periods of 17 minutes and longer.

Current Problem

It was the aim of this research program to determine the behavioral, physiological, and pathological sequelae of decompression of chimpanzees to a near vacuum with subsequent recompression to a habitable atmosphere in order that mission rules, safety procedures, and necessary engineering designs might be incorporated into manned spacecraft to assure maximum chances of mission success for astronauts. More specifically, this research attempted to answer the following questions:

1. Can an unprotected, highly developed organism such as the chimpanzee survive rapid decompression to a near vacuum?

2. If the chimpanzee can survive rapid decompression, what exposure times following such a decompression can it survive?

3. What is the effect of rapid decompression to a near vacuum on the performance of complex tasks by the chimpanzee?

4. What is the time of useful consciousness (TUC) following decompression to a near vacuum? TUC is defined as that time which elapsed between the onset of decompression and the last correct response to a meaningful stimulus before unconsciousness occurs.

5. What is the time of total impairment (TTI), that is, how much time elapses between loss of useful consciousness and the time when the chimpanzee is again able to respond to meaningful stimuli?

6. How does performance during and after decompression to a near vacuum compare with pre-exposure performance?

7. How much time elapses before the animal is again able to perform complex tasks at a level comparable to its pre-exposure efficiency (TBR)?

METHODOLOGY

Subjects

Eight chimpanzees (6 males and 2 females) from the chimpanzee colony at the 6571st Aeromedical Research Laboratory at Holloman Air Force Base, New Mexico, were assigned to this project. Their approximate ages (determined by dental eruption) and weights (established on the day of decompression) are summarized in Table I.

TABLE I

EXPERIMENTAL SUBJECT DATA

RD*	Name	Number	Sex	Weight (kg)	Age (Months)
1/05"	Jim	62	Male	21.09	81
2/30"	Duane	64	Male	22.23	80
3/60"	Jim	62	Male	21.77	81
4/90"	Rufe	114	Male	21.55	79
5/120"	Clayton	130	Male	18.14	67
6/150"	Shirley	116	Female	18.60	61
7/90"	Brownie	120	Male	22.68	75
8/120"	Lulu	121	Female	25.40	70
9/150"	Jake	109	Male	20.87	72

* Test Number and duration of exposure (in seconds) to less than 2 mm Hg.

Apparatus (See Appendix I-A)

Performance Schedule (See Appendix I-B)

Procedure

The decompression subject underwent intensive training on all tasks of the work schedule. This training, which involved operant conditioning techniques, took place in the Comparative Psychology Division. The preparation time varied from 6 to 12 weeks, depending on how tractable the subject was and how much previous training the animal had received on similar tasks. The subjects received their introduction to the program in a restraint chair. After the animal showed sufficient progress, training was continued in a training couch to acquaint the subject with the new body position, the restraint suit, and the new location of the work panel.

Two days prior to the scheduled decompression, the subject was transferred to the chamber area. The subject was kept in the chamber for approximately 7 to 8 hours per day. The door was sealed but the subject was kept at Holloman altitude with fresh air being constantly replenished and the temperature and humidity kept under controlled conditions. The animal, with all the physiological sensors in place, was restrained in the experimental couch for work on the experimental performance panel where experimental conditions were approximated as closely as possible. Performance programs were presented and responses recorded for baseline performance data during this time.

The preparation of the subject for experimentation began at 0730 on the day the animal was to be exposed to RD. Following a brief physical examination (a thorough examination was given a week earlier), all physiological sensors were attached. The subject, dressed in a loosely fitting net restraint suit, was then placed in the experimental couch and transferred to the chamber area. The shock plates were taped to the soles of the feet and the adhesive tape was cut to allow for foot expansion. In addition, the feet were loosely wrapped with elastic bandage to insure electrical conduction. Fidelity and compatibility checks of behavioral and physiological data recordings were accomplished after the couch was inserted into the chamber.

All chimpanzees were exposed individually to the following experimental conditions:

1. Within 15 minutes after sealing the chamber door, the subject was provided with a 100 percent O_2 environment for purposes of 3 hours of denitrogenation prior to going to altitude. Temperature and humidity were maintained at approximately 24^{o} C and 20 percent to 50 percent, respectively, throughout the entire period the animal occupied the chamber. One complete performance program was presented 30 minutes after the subject began pre-breathing 100 percent O_2 and a second presentation of the complete work program took place 2 hours after the subject entered the 100 percent O_2 environment. Performance on these two schedules had to be within the limits of the baseline performance for the test to continue.

2. The subject was then taken to a pressure altitude of 10, 700 meters (179 mm Hg) since this environment approximated arterial oxygen saturation of a resting chimpanzee breathing air at the altitude of Holloman Air Force Base, New Mexico (1, 250 meters). This change in altitude was accomplished in about 13 minutes. At this altitude the subject completed another performance schedule. If performance was not significantly different from baseline, the subject was then decompressed in 0. 8 seconds to a pressure of less than 2 mm Hg and kept there for periods of 5 seconds to 150 seconds, depending on the experimental design. Two minutes prior to decompression the preparatory avoidance schedule was introduced and the main program began simultaneously with decompression. Continuous motion picture photography and still pictures at intervals of 15 seconds were obtained for 30 minutes from this point onward. The shocking device (negative reinforcement) was turned off as soon as the subject was unable to respond, but the Avoidance Program stimuli were continued.

3. Recompression to 10, 700 meters (still 100 percent oxygen) was accomplished in 30 seconds following the specified time at less than 2 mm Hg. The total work schedule was presented in case the subject returned to consciousness and began to perform at baseline level during the first avoidance schedule (as in the case of the shorter duration exposures). If, however, as in the longer duration exposures, the animal failed to attend to the stimuli by the end of the avoidance portion of the

first program, the schedule was interrupted at this point. The avoidance tasks were re-introduced after 1 minute of rest until the animal began to respond at its baseline level.

4. Physiological recordings as well as TV monitors were constantly observed. If the animal showed signs of recovery on the basis of these observations, the shocking device was turned on and off for 1 minute alternately until the first response was recorded. Then continuous reinforcement was administered. After decompression-recompression, the work-rest cycle (30 minutes work - 15 minutes rest, or 22 minutes work - 13 minutes rest) continued for 6 hours followed by a 6 hour total rest period. The subject remained at an altitude of 10,700 meters on 100 percent O_2 for a period of 24 hours, continuing on a 6-hour work and 6-hour rest cycle, unless a decision was made to the contrary.

5. At the end of the 24 hour experimental period at 10,700 meters, the chamber atmosphere was lowered to Holloman altitude in approximately 18 minutes, and the subject removed from the chamber. The animal was then transferred to the Veterinary Sciences Division and removed from the couch. In the same area the subject was taken out of his restraint suit and the physiological sensors removed. The animal then underwent an immediate physical examination and was subsequently transferred to its regular living quarters.

The separate effects of rapid decompression, the subsequent effects of exposure to a pressureless environment, and the effects of anoxia were not delineated in the design of this research. However, the research simulated a space or space-related operations accident where these variables may occur concurrently.

Statistical Approach

1. Following each RD, tests of significance of difference (Student's t) were computed between the following means:

a. Baseline and experimental means derived from the response latencies of AM, VM, DA, and Oddity Discrimination.

b. Baseline and experimental means derived from the response efficiencies of AM, VM, DA, and Oddity Discrimination.

c. Baseline and experimental means derived from Oddity probability scores.

d. Baseline and experimental means derived from the response frequencies (LP/min) of CA.

2. Data defined as "baseline" consisted of performance recorded on the 2 days prior to rapid decompression (see page 7), plus the three performance sessions immediately preceding decompression.

3. Performance which began with the appearance of the first correct response to a meaningful stimulus following decompression-recompression and which ended with the animal returning to Holloman altitude, constituted the experimental data.

RESULTS

A total of nine decompression studies were accomplished. All subjects survived in excellent physical condition. They were all also able to perform within baseline limits before they were removed from the chamber. Follow-up studies, which are still in progress, have not detected long-lasting changes in performance. The results from these studies will be made available at a subsequent date.

The time of useful consciousness (TUC) and the time of total impairment (TTI), as defined on page 5, for each decompression test are summarized in Table II. The time of total behavioral recovery (TBR) is presented in the same table. Detailed information concerning recovery of individual performance tasks is furnished as Appendix I-C.

TABLE II

Summary of Rapid Decompression Effects

RD Experiment	Exposure Time to < 2 mm Hg (seconds)	TUC Time of Useful Consciousness (seconds)	TTI Time of Total Behavioral Impairment (minutes)	TBR Time of Total Behavioral Recovery (minutes)
1	5	11	.42	20.00
2	30	Not available	1.80	67.00
3	60	16.9	2.48	90.08
4	90	11.3	18.93	163.02
7	90	12.5	4.82	43.00
5	120	10.1	8.56	245.02
8	120	9.5	19.07	121.75
6	150	29.7	36.56	144.02
9	150	8.0	38.69	247.00

Summaries of performance during baseline and experimental sessions for each rapid decompression test are presented in Tables III through XI. The reaction times are given in tenths of seconds and fractions thereof.

Rapid Decompression Test No. 1 (Jim No. 62, 1-2 July 1964)

This subject was exposed to a near vacuum for 5 seconds and was incapacitated for a total of 25 seconds (TTI (36 seconds) - TUC (11 seconds)). Subject's performance was not significantly affected by the experimental condition except in two instances. Reaction time to the oddity problems was significantly lower (improved) and the average lever presses per minute on the continuous avoidance program were significantly reduced (a decrement). Discriminative efficiency on the oddity problems was not affected by the experimental conditions. See Table III.

Rapid Decompression Test No. 2 (Duane No. 64, 15-16 July 1964)

This subject experienced a 30 second exposure. Almost all reaction times were adversely affected with reaction time for Discrete Avoidance, Visual Monitoring, and Oddity Discrimination increasing significantly. Auditory Monitoring reaction time was faster, but not to a significant extent. In the avoidance portion of the schedule, the subject's efficiency was equal to that of pre-exposure performance, and the response rate on the continuous motor task was significantly increased. The discrimination task was performed in a superior fashion after exposure to altitude, with both discrimination efficiency and the probability of a correct choice improving significantly. See Table IV.

TABLE III

Rapid Decompression Test No. 1/05"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	6.65	278	1.76	6.69	199	1.87	-1.82
VM-RT	7.10	20	1.20	6.87	290	1.26	1.27
AM-RT	7.08	195	1.57	7.15	138	1.77	-.37
Odd-RT	10.47	686	3.59	9.27	472	3.51	5.71**
DA-Eff (%)	82.83	24	11.21	90.00	16	10.52	-2.00
VM-Eff	71.21	24	9.29	75.25	16	12.08	-1.37
AM-Eff	58.04	24	18.60	62.69	16	15.68	-.83
Odd-z	2.48	24	1.31	1.87	16	1.36	1.39
CA-LP/min	143.17	24	14.40	104.50	16	18.76	7.91**

** significant beyond .01 level

TABLE IV

Rapid Decompression Test No. 2/30"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	6.26	543	1.17	6.80	202	1.15	-5.74**
VM-RT	6.57	536	1.12	6.82	237	1.16	-2.22*
AM-RT	6.06	546	1.11	5.94	212	1.18	1.33
Odd-RT	12.27	1,127	3.94	13.45	412	7.10	-3.19**
DA-Eff(%)	97.03	40	4.40	90.31	16	16.62	1.54
VM-Eff	87.44	36	9.23	87.00	16	15.63	.10
AM-Eff	97.55	40	4.00	94.69	16	11.97	.91
Odd-z	2.85**	40	1.20	3.91**	16	.79	-3.79**
CA-LP/min	73.00	40	15.02	96.88	16	19.10	-4.34**

* significant beyond .05 level
** significant beyond .01 level

Rapid Decompression Test No. 3 (Jim No. 62, 29-30 July 1964)

Jim was the only animal which was decompressed on two occasions. His first exposure was of relatively short duration (5 seconds), and he showed insignificant changes during post-experimental performance; therefore, he was chosen as the subject for this experiment which involved a 60 second exposure. Experimental performance was highly variable; however, performance on the Discrete Avoidance task was not at all affected. Average response latency on the Visual Monitoring task was of significantly longer duration and experimental response efficiency dropped far below pre-exposure performance. Auditory responses were somewhat slower but their variance was not statistically different from pre-exposure responses. The efficiency, however, on this task suffered noticeably as did the response rate on the Continuous Avoidance task. Oddity Discrimination performance following decompression was inferior both in terms of response latency and efficiency of choice. The subject's average discrimination score was far above chance level before decompression, but following decompression discriminations were, on the average, at a chance level.

Follow-up data for this subject have yielded the following results. Reaction times to the DA task and the Oddity Discrimination task were still significantly slower 3 months after the second decompression than during baseline and experimental performance. VM reaction time, as well as the performance efficiency on DA, VM, and AM tasks, was improved as compared with baseline performance. Lever presses per minute on the continuous motor task did not change from experimental data to follow-up data, that is, this task performance was still below the baseline performance.

Discrimination efficiency on the Oddity task was still below baseline but had significantly improved over the experimental performance. This suggests that the decrement immediately following decompression was not stable and indicates a trend toward improvement. See Table V.

TABLE V

Rapid Decompression Test No. 3/ 60"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	6.47	265	1.74	6.40	206	2.51	.33
VM-RT	6.74	234	1.17	7.21	58	1.01	-3.13**
AM-RT	7.24	173	1.25	7.58	57	1.15	-1.89
Odd-RT	18.41	521	10.28	21.32	435	13.15	-6.47**
DA-Eff (%)	94.75	20	6.21	94.69	16	7.82	.02
VM-Eff	69.05	20	9.95	22.25	16	19.12	8.60**
AM-Eff	61.65	20	10.84	27.13	16	19.33	6.19**
Odd-z	3.03**	19	1.16	-.31	16	2.69	4.45**
CA-LP/ min	114.85	20	15.83	88.63	16	19.51	4.22**

** significant beyond .01 level

Rapid Decompression Test No. 4 (Rufe No. 114, 12-13 August 1964)

On this test the total exposure time was 90 seconds. The subject was quite consistent in its performance change following decompression. All reaction times were significantly longer than pre-exposure values and performance efficiency on all tasks suggested a decremental trend; however, no statistically significant pre- and post-exposure differences were detected. Performance on the Oddity Discrimination problems was well above chance during both pre- and post-experimental performance periods. See Table VI.

Rapid Decompression Test No. 5 (Clayton No. 130, 26-27 August 1964)

This subject was maintained at a near vacuum for 120 seconds. Comparison of pre-experimental and experimental reaction times on the DA task revealed that they came from the same population, while the mean experimental efficiency on this task dropped below baseline performance. Visual Monitoring was poorer both in reaction time and efficiency. The AM reaction time showed a tendency toward improvement; however, this improvement was not statistically significant. On the other hand, this subject committed more errors on this task than during pre-decompression. The subject took significantly less time after decompression to make choices on the Oddity Discrimination problems and his performance showed some, yet insignificant, improvement in efficiency. The subject's rate of response on the continuous motor task was markedly depressed. See Table VII.

TABLE VI

Rapid Decompression Test No. 4/90"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	5.67	211	2.15	7.00	125	1.31	-7.00**
VM-RT	6.15	216	1.14	6.49	137	1.07	-2.83**
AM-RT	6.10	216	.85	6.32	135	1.09	-2.00*
Odd-RT	13.11	330	5.20	16.21	228	5.76	-6.46**
DA-Eff (%)	94.31	16	6.17	77.42	12	26.31	2.09*
VM-Eff	79.19	16	8.07	66.92	12	31.97	1.25
AM-Eff	96.50	16	4.29	80.42	12	31.79	1.67
Odd-z	5.20**	16	2.68	5.19**	11	.41	.01
CA-LP/min	183.44	16	24.36	195.96	11	60.75	-.62

* significant beyond .05 level
**significant beyond .01 level

TABLE VII

Rapid Decompression Test No. 5/120'

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	6.94	249	1.27	7.15	188	1.11	1.86
VM-RT	5.03	302	.78	5.41	229	1.11	-4.22**
AM-RT	5.69	249	1.22	5.51	184	1.24	1.50
Odd-RT	8.34	413	3.35	7.69	372	3.64	2.50*
DA-Eff (%)	98.83	18	2.61	70.68	19	42.87	2.78**
VM-Eff	98.67	18	3.20	70.89	19	42.85	2.74**
AM-Eff	98.83	18	3.50	69.21	19	38.71	3.23**
Odd-z	2.33	18	1.29	2.76	15	.65	-1.23

* significant beyond .05 level
** significant beyond .01 level

Rapid Decompression Test No. 6 (Shirley No. 116, 9-10 September 1964)

This chimpanzee was kept at less than 2 mm Hg for 150 seconds. During training and baseline performance this subject did not achieve steady and reliable performance on the Oddity Discrimination problems. It was therefore decided to delete this portion of the performance task for RD Test No. 6 only. Response rate on CA, mean efficiency on Visual and Auditory monitoring showed no changes due to the experimental conditions. Reaction times to all discrete events (DA, VM, AM) were improved, with only DA reaction time showing no statistically significant improvement. The response efficiency on DA was below the baseline performance level. See Table VIII.

Rapid Decompression Test No. 7 (Brownie No. 120, 27-28 October 1964)

This subject was exposed to the near vacuum condition for 90 seconds. Due to mechanical failure, no Auditory Monitoring responses were recorded during the experimental period. All reaction times were of significantly longer duration during the experimental sessions. Response efficiency on all tasks, as well as response rate on CA, was not affected. See Table IX.

Rapid Decompression Test No. 8 (Lulu No. 121, 14-15 December 1964)

This test involved an exposure to the near vacuum condition for 120 seconds. All mean reaction times for the discrete events during the avoidance portion of the performance schedule, as well as the reaction times to the Oddity Discrimination problems, were significantly slower when compared to the pre-experimental data. Performance efficiency on all discrete events of the avoidance program suffered somewhat, but statistically these variations were due to chance and therefore not significant. Continuous Avoidance responding was significantly depressed by the decompression; however, this depression did not influence proficiency, that is, the subject did not receive any more shocks when compared to pre-exposure performance. On the Oddity problems the trend was toward improvement in discrimination, even though this improvement was not statistically significant. See Table X.

TABLE VIII

Rapid Decompression Test No. 6/150"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	7.19	196	1.09	7.09	249	1.10	.91
VM-RT	6.54	230	1.34	6.09	304	1.17	4.09**
AM-RT	6.43	181	1.34	5.91	213	1.28	4.00**
Odd-RT							
DA-Eff (%)	100	14	0	94.37	19	9.77	2.48*
VM-Eff	96.57	14	4.92	94.05	19	14.61	.68
AM-Eff	92.50	14	6.18	80.16	19	24.74	2.03
Odd-z							
CA-LP/min	123.79	14	20.28	122.37	19	21.82	.19

* significant beyond .05 level

** significant beyond .01 level

TABLE IX

Rapid Decompression Test No. 7/ 90"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	4.55	250	1.46	5.97	148	1.03	-10.92**
VM-RT	3.94	290	1.29	5.21	179	1.44	-9.07**
AM-RT	NOT AVAILABLE						
Odd-RT	15.99	435	5.11	18.90	301	6.79	-6.33**
DA-Eff (%)	95.28	18	8.61	91.42	12	11.36	.96
VM-Eff	94.72	18	10.07	90.42	12	11.84	1.00
AM-Eff	NOT AVAILABLE						
Odd-z	3.51	18	1.21	3.68	15	1.35	-.37
CA-LP/ min	161	18	25.24	172	12	13.89	-.93

** significant beyond .01 level

TABLE X

Rapid Decompression Test No. 8/ 120"

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	3.97	239	1.08	4.89	167	1.33	-7.67**
VM-RT	4.24	281	1.27	5.27	178	1.00	-9.36**
AM-RT	5.20	233	1.12	6.04	160	1.34	-6.46**
Odd-RT	8.28	433	4.76	11.93	334	5.25	-9.86**
DA-Eff	99.60	15	1.50	86.23	13	23.86	1.93
VM-Eff	98.67	15	2.21	88.69	13	20.44	1.68
AM-Eff	97.13	15	5.04	84.77	13	28.37	1.49
Odd-z	1.81	15	.74	2.37	13	1.27	-1.33
CA-LP/ min	122.47	15	16.77	91.62	13	14.21	5.07**

** significant beyond .01 level

Rapid Decompression Test No. 9 (Jake No. 109, 12-13 January 1965)

This subject was exposed for 150 secnnds. All reaction times to the discrete events were significantly slower, while performance efficiencies were not altered by the experimental conditions. Lever presses per minute on the Continuous Avoidance task were significantly depressed after exposure. Oddity Discrimination was poor in reaction time, efficiency, and in chance discrimination. See Table XI.

DISCUSSION

The post-exposure improvement shown in RD Test Number 2 (30 seconds) on the complex discrimination task is of considerable import, in that it suggests negligible trauma to the central nervous system -- that is, learning occurred immediately following decompression and this brought about a statistically significant improvement. A similar case was the complex discrimination exhibited during RD Test Number 8 (120 seconds). Although discrimination efficiency between baseline and experimental performance was not significantly different, it is noteworthy in one particular aspect. The animal did not discriminate significantly different from chance during baseline performance, but during the experimental period the subject improved its discrimination efficiency to such a level that on the average it discriminated significantly above chance. These two instances of improvement on a difficult task following rapid decompression strongly suggest that central nervous system functioning may not be seriously affected by exposure to a near vacuum for periods up to 150 seconds.

Statistical comparisons between baseline and experimental performance for several animals showed a number of decrements, primarily in reaction times to discrete stimuli. It should be noted, however, that these mean differences often lie in the hundredths-second range and would generally not be of operational importance.

TABLE XI

Rapid Decompression Test No. 9/150''

Performance	Baseline			Experimental			
	$\bar{X}$	N	σ	$\bar{X}$	N	σ	t
DA-RT	5.61	316	1.06	5.94	283	1.20	-3.67**
VM-RT	5.59	367	1.29	6.22	318	1.25	-6.30**
AM-RT	5.55	311	1.02	6.00	277	1.03	-5.63**
Odd-RT	17.39	514	6.73	11.30	511	4.86	16.46**
DA-Eff (%)	98.80	20	2.40	93.16	19	20.82	1.14
VM-Eff	96.60	20	4.57	89.05	19	18.46	1.69
AM-Eff	97.30	20	2.98	91.16	19	23.61	1.09
Odd-z	3.74	20	.80	2.08	18	1.11	5.19**
CA-LP/min	151.00	20	16.24	107.74	19	24.55	6.29**

** significant beyond .01 level

The fact that significant decrements in performance did occur during the experimental period, and the fact that all subjects did return to a baseline level of performance during the experimental period (See Table II) is only an apparent contradiction. The data for the experimental period included all responses following rapid decompression, and these data did reflect a distinct decrement in subject reaction time, efficiency, and rate of response, during the period immediately following exposure to a near vacuum. And, although each subject recovered while still in the chamber and returned to a baseline level of performance, the decrements in performance immediately following decompression were sufficient to lower the overall experimental means and thus bring about a statistical difference.

Product moment correlation coefficients were computed to demonstrate relationships among exposure time to the near vacuum, the time of total impairment (TTI), and the time of return to baseline performance on the total task (time of total behavioral recovery - TBR). These relationships are presented in Figures 1, 2, and 3. Since TTI and TBR appeared to be possible exponents of exposure time to a near vacuum, and TBR a possible exponent of TTI (Curvilinear relationship), rather than possessing a linear relationship, eta coefficients were also computed. F-ratios between the product moment correlation coefficients and the respective eta correlation coefficients proved to be insignificant (See Figures 1, 2, and 3).

Product moment correlation coefficients were also computed between exposure time, time to a near vacuum and the time of return to baseline of the individual performance tasks (See Appendix III). The correlation coefficients are presented in Table XII. From this group of ten performance variables, eight with the highest correlation with exposure time (Table XIII) were considered for computation of a multiple correction coefficient (Doolittle - R). However, inspection of the correlation matrix (Table XIV) indicated that variables 4, 5, 8, and 9 had the largest intercorrelation with the remaining variables and would thus add little or nothing to the multiple correlation. Therefore, variables 2, 3, 6, and 7 (See Table XIII) were selected to be incorporated into the multiple correlation with variable 1 (exposure time to near vacuum).

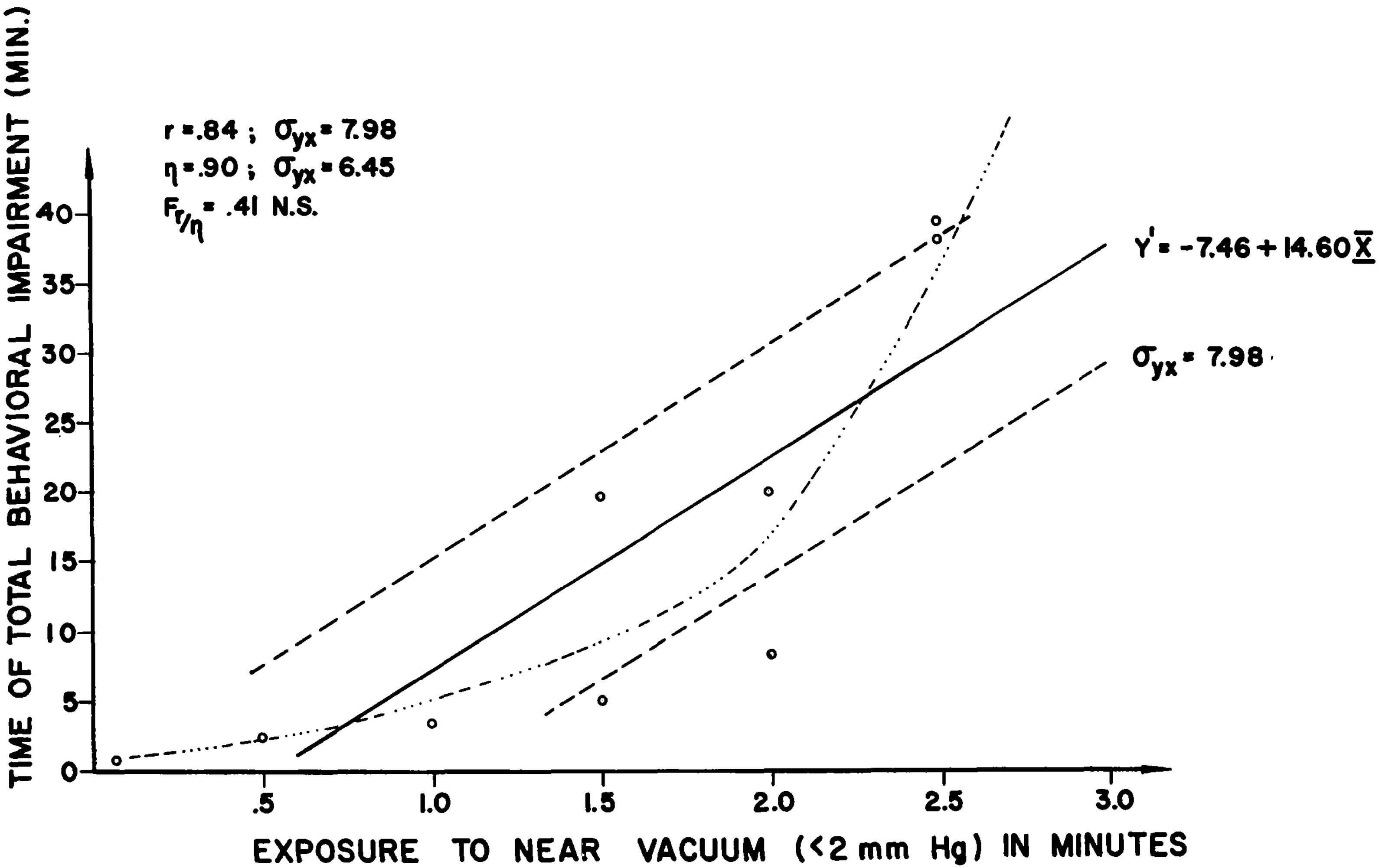

Figure 1. Relationship between exposure time to near vacuum and time of total behavioral impairment.

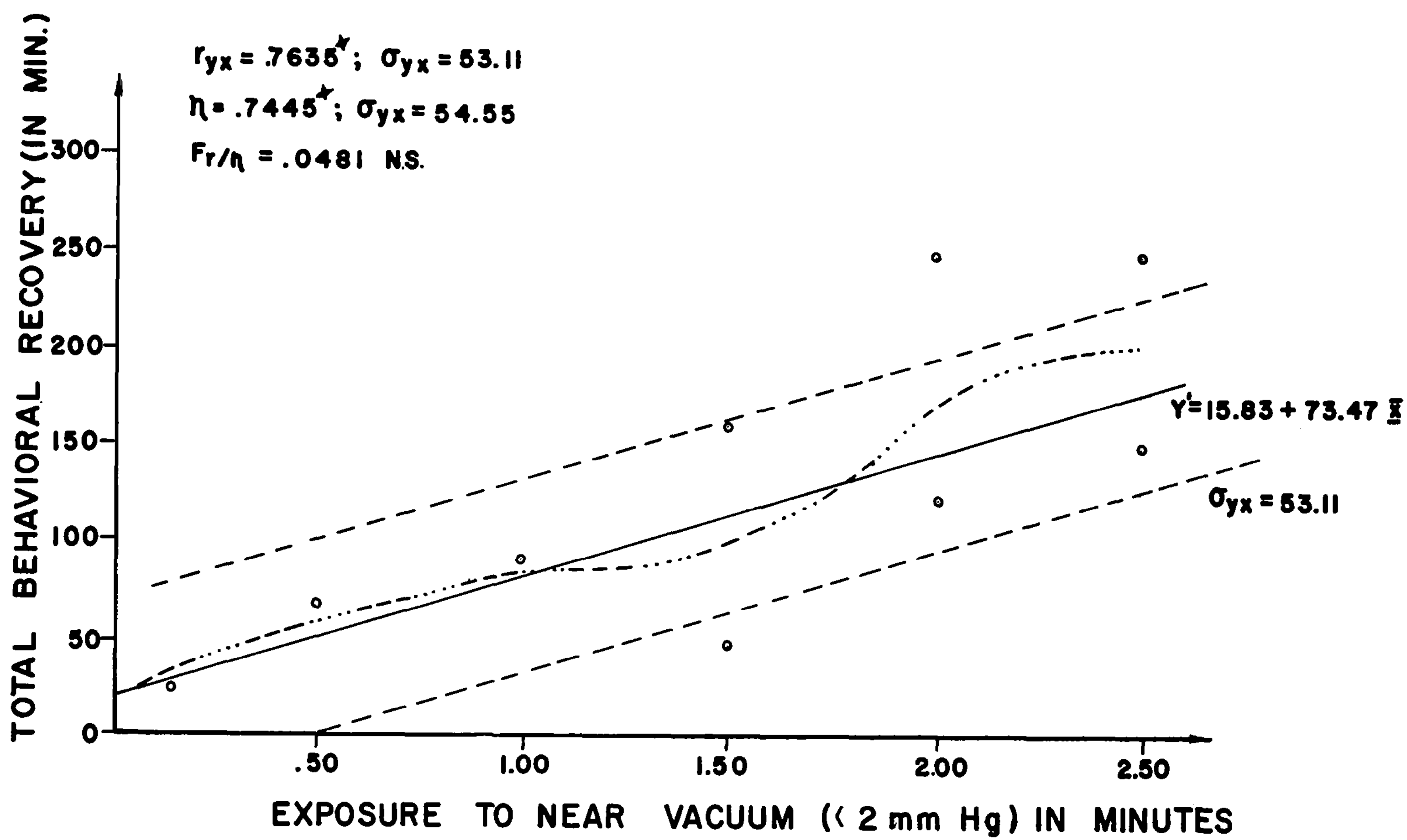

Figure 2. Relationship between exposure time to near vacuum and total behavioral recovery.

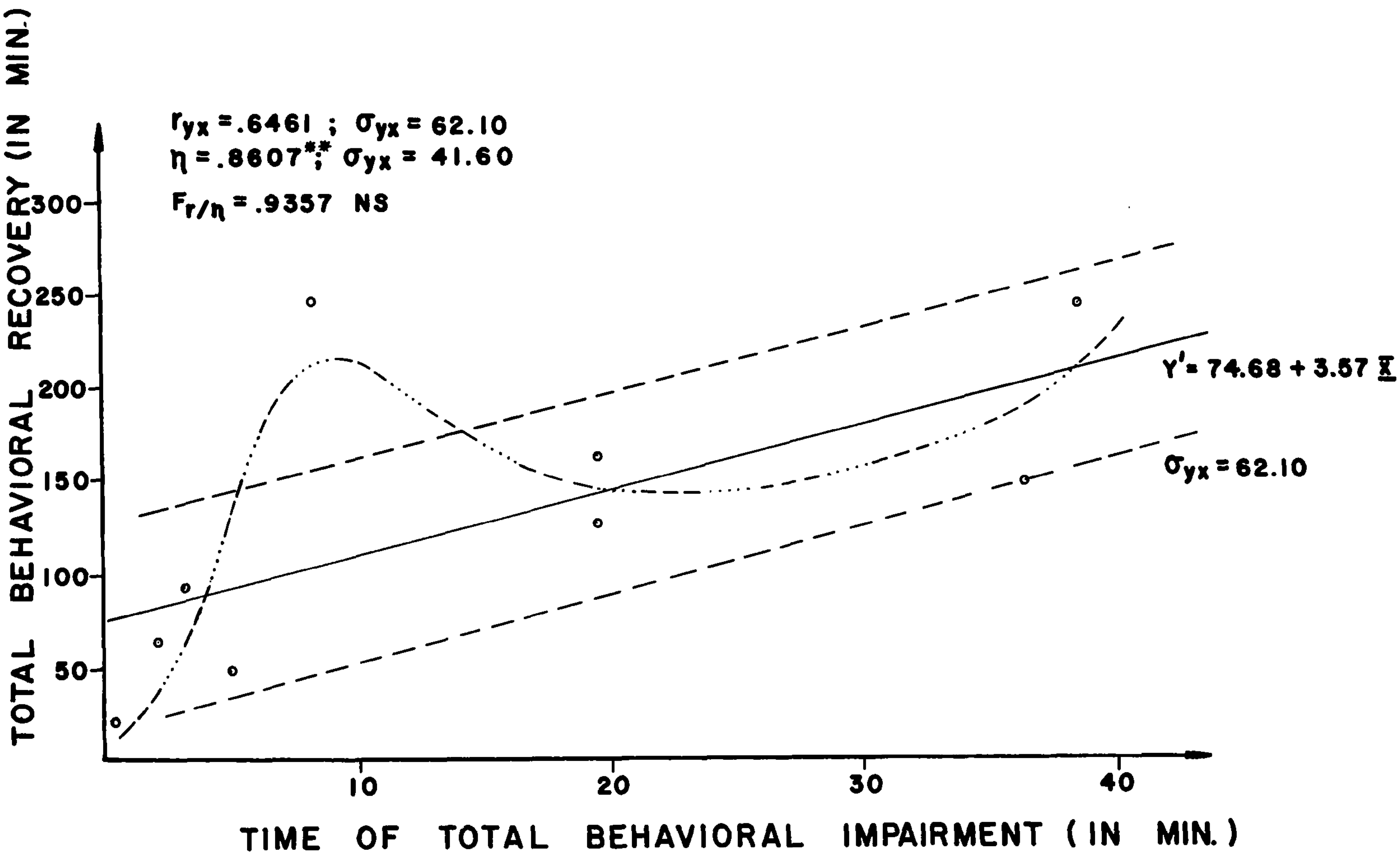

Figure 3. Relationship between time of total impairment and time of total behavioral recovery.

TABLE XII

Correlation Coefficients Between Exposure Time
to Near Vacuum and Time of Return to Baseline Performance

	DA-RT	Odd-Eff	AM-Eff	AM-RT	CA Dist.	CA Rate	DA-Eff	VM-Eff	VM-RT	Odd-RT
	r =	r =	r =	r =	r =	r =	r =	r =	r =	r =
Exposure time	.8370	.8247	.8214	.7930	.7405	.7366	.7063	.6883	.6348	.3254
	**	*	*	*	*	*	*	*	N. S.	N. S.

* significant beyond the .05 level

** significant beyond the .01 level

TABLE XIII

Variables for Multiple R

No. 1	Exposure time to a near vacuum in minutes	
No. 2	DA-RT	Return to baseline in minutes
No. 3	Odd-Eff	Return to baseline in minutes
No. 4	AM-Eff	Return to baseline in minutes
No. 5	AM-RT	Return to baseline in minutes
No. 6	CA Dist.	Return to baseline in minutes
No. 7	CA Rate	Return to baseline in minutes
No. 8	DA-Eff	Return to baseline in minutes
No. 9	VM-Eff	Return to baseline in minutes

TABLE XIV

Correlation Matrix of Variables for Multiple R

	X_2	X_3	X_4	X_5	X_6	X_7	X_8	X_9	X_1
X_2		.7978	.9061	.5969	.7638	.6651	.7598	.6191	.8370
X_3			.9310	.8366	.8459	.9136	.7060	.7403	.8247
X_4				.7018	.9022	.6507	.7817	.7828	.8214
X_5					.9094	.7683	.8549	.8896	.7930
X_6						.7185	.8873	.9523	.7405
X_7							.5696	.6836	.7366
X_8								.7628	.7063
X_9									.6883

Multiple $R_{1,2367}$ = .8773

$R^2_{1,2367}$ = .7697

Even though it is unnecessary to predict a known factor, i.e., exposure time to a near vacuum, the multiple correlation coefficient indicated that almost 77 percent of the variance in performance recovery was related to a given duration of exposure. This means that at least four of the performance variables were of considerable value in terms of their sensitivity to the experimental conditions, and the remaining six no doubt would account for a share of the remaining variance.

CONCLUSIONS

Perhaps the most important result of this series of tests is that all subjects survived the experimental conditions. The fact that the animals were capable of performing a complex task and achieving a level of performance equal to or superior to their pre-exposure performance provides clear-cut evidence of a functional capability not previously anticipated. The best and most cautious generalization that can be made at this time from the experimental findings is that the chimpanzee can survive, without apparent central nervous system damage (as measured by performance), the effects of decompression to a near vacuum up to 2.5 minutes and return within approximately 4 hours to baseline levels of functioning. Further research, in which the replication of longer duration exposures (90, 120, 150 seconds) is accomplished (perhaps with several different primate species) should determine the reliability of these findings and suggest the degree to which sub-human results may realistically be extrapolated to man.

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APPENDIX I-A

Apparatus

1. Closed Environmental System for Primates (comprized of four main mobile sub-assemblies)

Manufactured by:

General Electric Company
Missile and Space Vehicle Department
Aeromedical Engineering Operation
Philadelphia, Pennsylvania

The first sub-assembly was the chamber proper. Its internal dimensions were 76.2 cm in depth, 121.9 cm in height, and 121.9 cm in width, giving it a volume of 1.132 cubic meters. All wall surfaces were fully water-jacketed and connected in parallel by manifolds for maintaining wall temperature at any desired level or "heat pulsing" them from high to low extremes of temperature. A plexiglass window with a 21.6 cm by 24.1 cm area was provided in one wall over the pneumatic control panel for viewing the subject. The door of the chamber was modified to include a plexiglass panel 55.9 cm in width and 30.5 cm in height through which 16 mm moving pictures were taken throughout the period of decompression. Also provided were suitable electrical interfaces so that electrical signals could be transmitted from internal test instrumentation to outside monitors. Eight gas-sampling outlets were also provided. The pneumatic control console displayed an absolute pressure gauge, oxygen supply shut-off valve, "breathe-down" valves, bleed valves, a quick-vent valve, and a demand type precision oxygen regulator. A purge valve was also provided so that the desired atmosphere gas composition could be admitted and "air" expelled from the system.

The second sub-assembly was the life-support system. It was entirely enclosed with easily removable sheet metal covers, as was the entire system. This sub-system had the capability to maintain a liveable environment within the chamber proper. The atmosphere from the chamber was recirculated through the system at a rate of approximately .34 cubic meters per minute by means of an actual flow blower. After the "stale" air was removed from

the chamber, it passed through a CO_2 absorber. This device was capable of maintaining a CO_2 partial pressure below 2 mm Hg. The air then entered one of two cold traps which froze out most of the water vapor in the air stream. Two cold traps were necessary since they had to be alternately switched off the line and allowed to defrost as they became saturated. Frost heaters integrated with the refrigerant coils were utilized to perform this function. This freeze-out defrost cycle was programmed by an electro-mechanical timer which switched the flow path between cold traps by means of pneumatically actuated valves. The timer also terminated refrigerant flow path sequence and defrost heater operation. The cold air next entered the heater-humidifier where it was reheated and a controlled amount of moisture readmitted into the air stream before reentering the chamber. The life support sub-system also housed the samplers for the CO_2 and O_2 analyzers which were an integral part of the system. The CO_2 analyzer was of the infra-red type and monitored the CO_2 level at the inlet to the CO_2 absorber. The O_2 analyzer operated on the para-magnetic principle and continuously sampled from the system atmosphere at the cold-trap outlet. Hook-ups were provided to check the calibration of both these instruments without shutting down during the course of a test.

The electrical control console was the third sub-assembly of the system. This console displayed all the internal chamber environmental parameters and provided means for controlling them. Indicator lights were provided to indicate operations of various components within the system. The CO_2 analyzer display panel and the O_2 analyzer recorder-indicator were also housed in this assembly. An electrical interface was provided so that output from the environmental sensors could be fed to a continuous recorder if desired. An audible warning device indicated high or low extremes of either temperatures or pressure. The console was connected to the other parts of the system through a central junction box located on the life support sub-system.

The last sub-assembly to be described is the elevated wall temperature sub-system. This assembly provided the capability of heating the chamber walls to an elevated temperature and then cooling them back to ambient within a 10-minute period.

The system contained two insulated 151.4 liter tanks connected by three-way manual valves to an electrically-driven centrifugal pump. The first tank contained thermostatically-controlled immersion heater coils and the second was the cool tank which was cooled by refrigeration coils connected to a 1/3 Hp hermetic refrigeration unit. By manual regulation of the valves and pump, the desired temperature profile could be pulsed into the chamber walls. Additionally, this sub-system was used to maintain the chamber walls at the same temperature as the internal environment by means of a thermostatic thermistor temperature controller which cycled the water circulation pump to maintain a preset temperature. The decompression chamber was connected to a 84.9 cubic meter parasitic tank by a 45.7 cm diameter double-ported balanced decompression valve which was operated pneumatically.

2. Programming Equipment

Training program equipment consisted of commercially available standard relay equipment, while the experimental programming equipment was approximately 95 percent of the solid state variety. One complete equipment rack contained the necessary power supplies, task programming, and recording equipment. The rack contained a positive 28 volt unregulated power supply, a negative 12 volt regulated supply, and required a 110 VAC 60 cps power input to drive the supplies and various reinforcement equipment. Reinforcement for performance was both positive and negative. Positive reinforcement applied to both the Fixed Ratio and Oddity Discrimination programs and involved a choice (by the test subject) of either food or water. If the subject selected food, a one-gram flavored pellet (banana, maple, etc.), dropped through a chuting arrangement into a cup readily accessible to the subject. If water was selected, the subject could consume approximately 5 to 10 cc of liquid from a lip lever located just to the right of its head. Negative reinforcement was used in the Avoidance and Oddity Discrimination programs and consisted of a mild shock to the soles of the subject's feet. The duration and intensity was under the experimenter's control and was determined by individual subject differences in motivation. Counters and recording equipment were used to record all correct and incorrect responses, in some cases total number of lever responses (Continuous Avoidance

or Fixed Ratio responses) and totals of positive and negative reinforcements received. A digital printout counter recorded reaction time to within one-tenth of a second. A cumulative type recorder reflected the rate of response for the Continuous Avoidance and Fixed Ratio tasks. As a back-up, a Model 150, 8-channel sanborn oscillograph recorded reaction time, number of responses, and negative reinforcement. The Sanborn was correlated with IRIG timing to ascertain the exact time of occurrence of the various events.

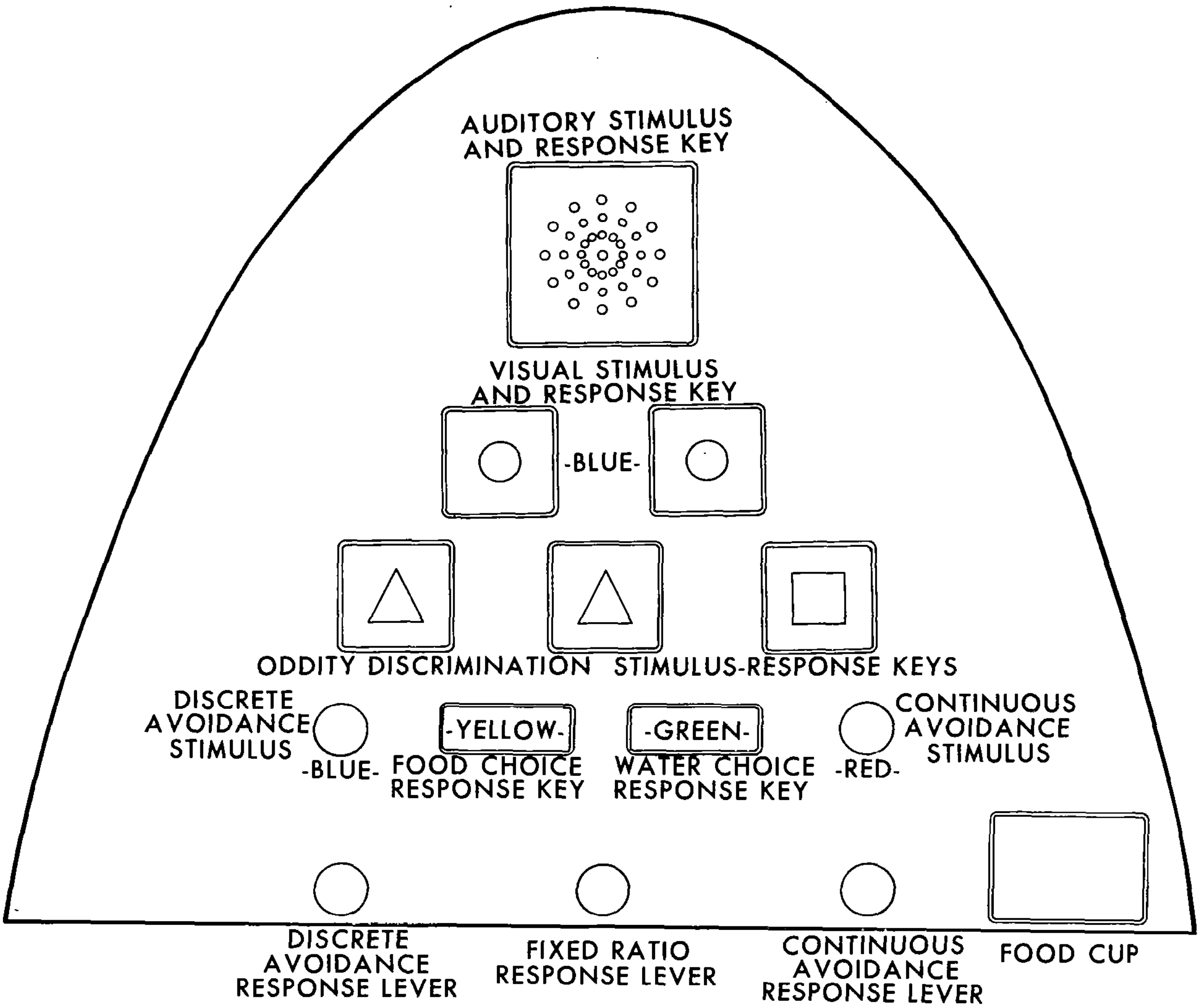
3. The Performance Panel

The performance panel consisted of a stimulus-response apparatus and was easily removable from four mounting points on the subject restraint couch. Immediately behind the performance panel were two feeders containing 1500 one-gram pellets each and a reservoir containing approximately 5,000 cc of water. Appropriate wiring terminated at electrical interfaces used in connecting the panel to the program rack. The performance panel is illustrated in Attachment 1 to this appendix. Basically, it contained eight stimulus-response keys (SRK's), two 2.54 cm diameter lights, and three levers. The SRK was a lucite pushbutton lever which was developed in this laboratory and is described in detail elsewhere (Ref. 13). The levers were 2.54 cm in diameter and protruded 5.72 cm beyond the face of the panel. A .91 kg force was required to activate each lever. Mounted behind the center SRK's, series 140 miniature rear projection one-plane readouts projected oddity symbols on the SRK's. The symbols were 1.6 cm in size. The one-plane readouts were manufactured by Industrial Electronic Engineers, Inc., 5528 Vineland Avenue, North Hollywood, California.

4. Experimental Restraint Couch

The restraint couch, made of fiberglass and mounted on an aluminum base, was of the form-fitting variety and kept the subject's thighs perpendicular to his back. The subject was tilted at approximately 15^{o} to the base when lying in the couch, and its hands were free to move about although baffling prevented it from reaching the area below its waist. A perforated plastic "bubble" covered the top part of the couch in order to keep the subject's hands away from the EEG probes in its head. Besides

numerous ventilation holes, the bubble had three 3.75 cm holes in front by the subject's mouth to enable it to feed itself following positive reinforcement. Excrement was collected in a closed container with a volume of approximately 4,900 cubic cm. It was located directly forward and below the subject and connected to the couch by a 10.2 cm diameter tube.



Attachment 1. Schematic Drawing of the Performance Panel

APPENDIX I-B

Performance Schedule

The primary objective of these experiments was the determination of the recovery of performance after rapid decompression to a near vacuum. A standard performance schedule for all subjects had been developed and is described below. The time sequence of events is provided in Attachment 1 to this Appendix.

1. Avoidance

a. The first ten minutes of the performance schedule constituted the Avoidance phase. It consisted of a basic program, Continuous Avoidance (CA), which covered the entire time period and required continuous motor behavior. The stimulus for CA was a red light in the lower right hand corner of the panel (see Attachment 1, Appendix I). The subject responded by depressing the lever below the light at least once every five seconds to avoid electric shock. Each lever press delayed the shock for five seconds. The dependent variables were lever presses per minute and total number of shocks. Concurrent with CA the subject had to respond to three additional avoidance programs: Auditory Monitoring (AM), Visual Monitoring (VM), and Discrete Avoidance (DA).

b. The stimulus for AM was a one-thousand-cycle tone of 60 db intensity which was presented for 1 second aperiodically. The stimulus source was a speaker mounted at the center top of the panel. The subject was required to terminate the tone by pushing the response plate covering the speaker. Failure to respond within 1 second of the onset of the auditory stimulus resulted in the delivery of an electric shock.

c. VM was basically identical to AM with the exception that the stimuli were blue lights behind the two SRK's (VM 1 and VM 2) located just below the AM speaker plate. Presentation of these stimuli was aperiodic, and the same response and reinforcement configuration as in AM were used.

d. The left lever and the blue light immediately above it were associated with the Discrete Avoidance (DA) task. The blue light was presented for 1 second aperiodically. A lever press had to be accomplished within 1 second of the onset of the stimulus to avoid electric shock.

Responses to 45 discrete events (14 DA, 17 VM, and 14 AM) concurrent with the CA program were necessary. It will be noted from the time sequence table (Attachment 1 to this Appendix) that 12 of the 45 discrete events occurred within the first 15 seconds of the work period. This design provided as many measures as possible during the critical time period immediately following decompression. The measured parameters of all discrete events consisted of response latency measured in tenths of seconds and of an efficiency percentage measure derived from the ratio of correct responses to total presentations.

2. Fixed Ratio

Since the animal had to spend considerable time confined in the decompression chamber, it had to be provided with an opportunity to feed itself. A stimulus light above the center lever was presented immediately following the avoidance schedule. This cued the subject to depress the lever on a fixed ratio 50:1 reinforcement schedule. When the subject had completed 50 responses, the SRK's above the cue light were illuminated. If food was desired, the subject pressed the left SRK (yellow) and was reinforced by a one-gram food pellet (Ciba) dropped into the feeder hopper by an automatic pellet dispenser. If the animal preferred water, it was conditioned to press the right SRK (green). This activated a green cue light at the water feeder for six seconds. During this time water was available to the subject. Biting the drinking tube opened a solenoid and the animal could drink for the allotted time. This program was in effect for 10 minutes and was followed by a 30-second rest break.

3. Oddity

The last 9.5 minutes of the performance schedule was a Negative Oddity program designed to evaluate discriminative efficiency and response latency. This program required the animal to select the odd symbol of three symbols presented. A total of 18 oddity problems was included in this program. Their sequence and the type of symbols used is shown in Attachment 2 to this Appendix. Three symbols were presented by three rear projection units through the SRK's located below the two VM buttons (Attachment 1, Appendix I-A). The subject had 10 seconds to select the odd symbol and respond by pressing the appropriate key. A correct discrimination within this time terminated the

presentation, and a 20-second waiting period was introduced after which the next problem was presented. Failure to make a response within the allotted time or an incorrect choice resulted in the delivery of an electric shock, termination of the stimuli, and the beginning of a 20-second waiting period, after which the same problem appeared again. If the subject failed to complete the 18 oddity problems within 9.5 minutes, the program was terminated. Performance on this task was measured in terms of response latency, discrimination accuracy (a ratio of correct responses to total presentations), and a "z score" indicating the subject's performance in relation to a chance discrimination.

The 30-minute performance schedules were separated by a 15-minute rest period.

4. Amendments to the performance schedule.

a. Avoidance Phase. The onset of the avoidance schedule coincided with the onset of decompression in the original program sequence. These events probably influenced simultaneously the initial responses of the experimental subject, one through a startle response, the other through the actual decompression effects. In order to eliminate these startle responses, 2 minutes of a "preparatory" avoidance schedule was added, preceding the main program. Starting with RD No. 3/60", the preparatory avoidance schedule consisted of continuous avoidance (CA). Thus the total work schedule was extended to 32 minutes and the rest period was shortened to 13 minutes. Six discrete events were superimposed on the 2 minutes of CA beginning with RD No. 7/90". The type of discrete events as well as the time sequence are also presented in Attachment 1 to this Appendix.

b. Fixed Ratio Phase. This phase of the program was deleted after the completion of RD Test No. 6/150".

c. Oddity Phase. The oddity program was modified concurrently with the change in the fixed ratio phase. The subject continued to perform on the 18 oddity discrimination problems and was allowed 10 seconds in which to make a discrimination. If the discrimination was correct, a shock to the soles of the feet was avoided, and a choice for food or water was presented for 5 seconds via the already described green

and yellow SRK's. The next problem was presented after a 20-second delay. If the discrimination was incorrect or if the subject failed to make a discrimination within 10 seconds, shock was delivered and the same problem was presented again after a 20-second delay. Starting with RD No. 8/120", each oddity problem was preceded by a 2-second warning light which directed the subject's attention to the upcoming problem and allowed the observation of EEG pattern changes. If all problems in the series were not completed at the end of the 9.75 minute work phase, the program was terminated.

These various performance program changes shortened the work schedule to 22 minutes: 12 minutes Avoidance, 15 seconds Rest, and 9.75 minutes of Oddity, followed by a 13-minute rest period.

Attachment 1

Avoidance Performance Schedule
for Rapid Decompression Studies

Time	Continuous Avoidance (5" R-S Interval)	Discrete Avoidance	Visual Monitoring (2 stimuli)	Auditory Monitoring
-2	Begin			
-1'45"				x
-1'15"		x		
- 45"			x_2	
- 30"		x		x
- 15"			x_1	
0	Decompression	x		x
+2"				x
+4"			x_2	x
+6"			x_1	
+8"		x		
10"		x	x_2	
12"		x	x_1	
14"				x
30"		x		
45"			x_1	x
60"				x
1'15"			x_2	
1'30"		x		x

Time	Continuous Avoidance (5" R-S Interval)	Discrete Avoidance	Visual Monitoring (2 stimuli)	Auditory Monitoring
2'0"			x_1	
2'15"				x
2'30"		x		
2'45"			x_2	
3'0"		x	x_1	
4'30"			x_2	
5'0"		x		
5'30"			x_1	
5'45"				x
6'0"		x		
7'0"			x_1	x
7'30"		x	x_1	
7'45"			x_2	x
8'15"			x_2	
8'30"		x		
8'45"			x_1	
9'0"				x
9'30"		x		x
9'45"		x		x
10'00"	end		x_1	

Attachment 2. Sequence of Oddity Problems

PROBLEM NO.	SYMBOL ON DISPLAY		
	1	2	3
1	○	△	○
2	△	△	○
3	○	○	□
4	△	○	○
5	△	○	△
6	□	□	△
7	○	□	□
8	△	□	□
9	△	△	□
10	□	○	□
11	□	△	△
12	○	□	○
13	○	○	△
14	○	△	△
15	△	□	△
16	□	○	○
17	□	△	□
18	□	□	○

APPENDIX I-C

Return to Baseline of Individual Performance Tasks

(in minutes)

RD Exp	DA-RT	DA-Eff	VM-RT	VM-Eff	AM-RT	AM-Eff	Odd-RT	Odd-Eff	CA Rate	CA Distr.
1	1.50	1.50	.75	.75	1.00	1.00	<u>20.00*</u>	<u>20.00*</u>	.60	.60
2	45.02	45.02	45.02	45.02	2.28	45.02	<u>67.00</u>	20.00*	1.80	43.00
3	5.00	5.00	7.50	<u>90.08</u>	45.08	5.75	20.00*	20.00*	43.00	43.00
4	36.50	<u>163.02</u>	73.08	118.08	118.02	73.02	93.00	48.00*	71.00	116.00
5	74.75	155.02	<u>245.02</u>	155.08	110.02	155.02	85.00*	85.00*	153.00	153.00
6	74.15	109.02	74.08	109.08	<u>74.08</u>	144.02	Not Available		72.00	107.00
7	33.02	33.02	33.08	33.08	Not Available		<u>43.00</u>	<u>43.00</u>	31.00	31.00
8	60.50	<u>121.75</u>	60.25	60.25	59.25	59.25	36.00*	36.00*	59.00	59.00
9	73.50	107.75	178.25	108.25	107.25	107.25	49.00*	84.00	<u>247.00</u>	107.00

Underlined times indicate total behavioral recovery on this RD experiment

* First performance opportunity on this task following RD

SECTION II

PHYSIOLOGY

Loyd M. Stephens, 1st Lt, USAF
John L. Hartman, Major, USAF, (MSC)
Oliver F. Lewis, MSgt, USAF
John M. Rhodes, Assoc. Prof., University of New Mexico

Physiology Branch
Bio-Effects Division
6571st Aeromedical Research Laboratory
Aerospace Medical Division
Air Force Systems Command
Holloman Air Force Base, New Mexico

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ABSTRACT

Cortical EEG, ECG, and respiration were recorded from immature chimpanzees before, during, and following rapid decompression to a near vacuum of less than 2 mm Hg. Subjects remained at this pressure altitude for up to two and one-half minutes. Visual analysis of the physiological parameters, as recorded on strip chart, was conducted and a correlation of these results to the animal's performance ability was attempted. EEG fast activity (10-12 cps) always preceded the end of total performance impairment. The continuous avoidance baseline level of response followed the return of EEG normalcy.

INTRODUCTION

In recent years there has been an accumulation of knowledge of brain function during anoxia and hypoxia (Refs. 1, 8, 10, 20), as reflected by the electroencephalogram. Little is known, however, about the electrical activity of the brain of higher primates following rapid decompression (RD) to pressures as low as 1-2 mm Hg.

The purpose of this portion of the report is to communicate preliminary results of electrophysiology and its relation to behavioral changes in eight chimpanzees following rapid decompression to a near vacuum pressure of less than 2 mm Hg. for periods of time varying from 5 to 150 seconds. Particular emphasis will be placed on cortical electroencephalographic activity and its correlation with performance changes during these exposures.

METHODS

Experimental Subjects

Eight chimpanzees with body weights ranging from 18.4 to 25.4 kg were decompressed to a pressure of less than 2 mm Hg. for periods of from 5 to 150 seconds. All subjects were decompressed only once except subject No. 62 who was decompressed twice (5 seconds and 60 seconds). No anesthetic or medication was used in any of the experiments.

Decompression Profile

The decompression profile and apparatus is discussed in the preceding section on Performance.

Electroencephalography

EEG activity was recorded by the following methods. Decompression Subjects 1 through 6 had six nylon and stainless steel electrodes (Fig. 1) mounted through the skull in three positions, bilaterally, in contact with the dura. All electrodes were parallel and 32.0 mm lateral to the midline. The anterior electrodes were 12.5 mm posterior to the supraorbital ridge; the posterior electrodes were 38.0 mm anterior to the inion; and the medial electrodes were centered between the anterior and posterior ones.

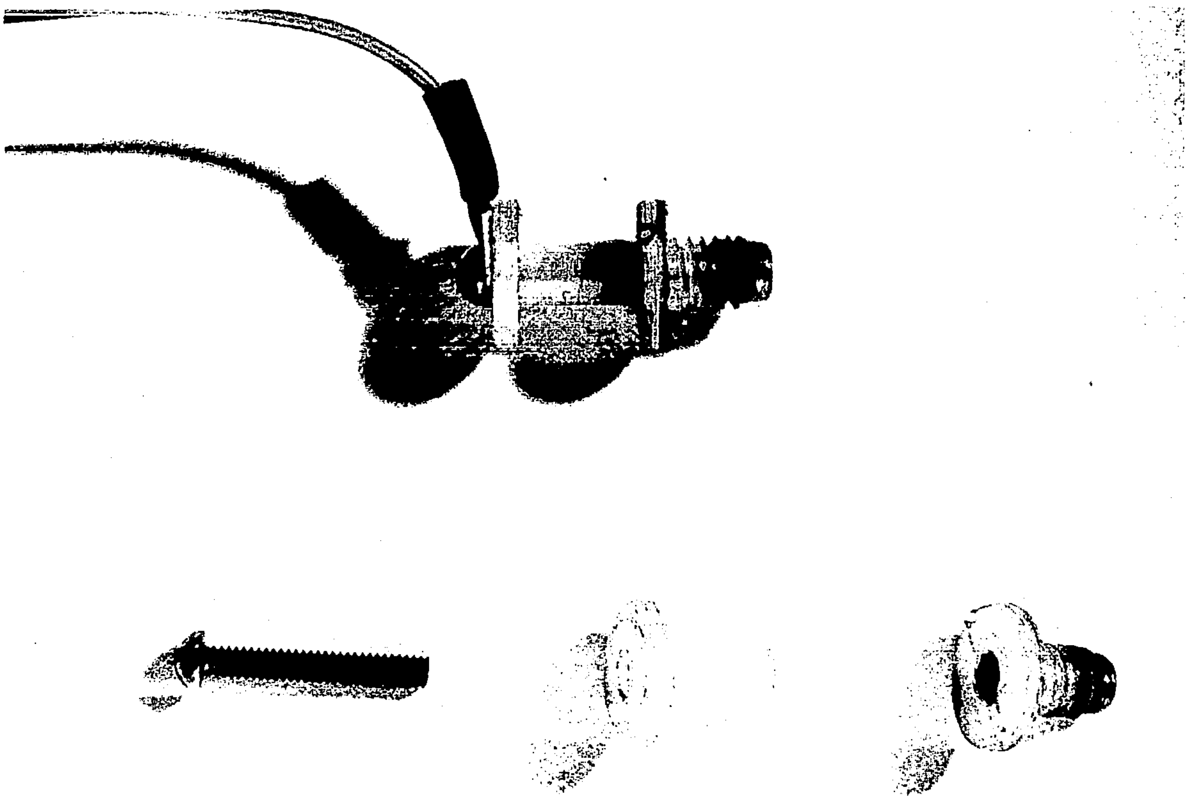


Fig. 1. Nylon and Stainless Steel RD Electrodes. The actual assembled length is 20 mm. The recording lead shown in the top view was attached during instrumentation on the day of the decompression.

Fig. 2. View of implanted receptacle following surgery on subject Jake (109) RD 9/150".

Decompression Subject 7 had stainless steel No. 4-40 screws placed in the skull without penetration to the dura in the same anatomical locations as described above and covered with dental acrilic cement. The lead wires were brought to an electrical connector on the vertex of the skull.

Decompression Subjects 8 and 9 had stainless steel No. 4-40 screws mounted in the same manner as described above in four locations bilaterally (frontal, central, parietal, and occipital), utilizing one ground electrode. The leads were brought to electrical receptacles (Fig. 2). Subject 9 damaged the implanted electrical receptacle to the extent that it was not usable. Therefore, suture leads of the type used for ECG were applied over the same areas as the implanted electrodes in order that an EEG signal could be detected. In all of the above methods the signals were recorded on an 8-channel Grass Model III A electroencephalograph.

ECG

The electrodes were suture type as described by Ward and Britz (Ref. 27). Lead A electrodes were located on mid-sternum and over the 5th lumbar vertebra with the ground lead located over the 8th thoracic vertebra. Lead B electrodes were located on the lower right axilla and on the left inner thigh. The ECG signals were recorded by the Grass electroencephalograph. Heart rate was obtained by feeding the lead A ECG signal into a Yellow Springs Instrument Co., Fels Cardiotachometer Model 21A and recording the output with a Sanborn 150 direct writing recorder.

Respiration

Respiration was recorded by the Grass Model III A from a copper sulfate ($CuSO_4$) respiration transducer located on the lower rib cage of the subject, which detected mechanical expansion and contraction of the thorax.

Skin Temperature

Skin temperature was recorded by the Sanborn 150 from a Yellow Springs Instrument Co., Type 409 Surface Thermistor located on the right inner thigh of the subject.

Magnetic Tape Recording

Rapid decompressions Number 8 and 9 had the physiological data recorded on magnetic tape using an Ampex FR 600 tape recorder. All data were recorded on individual tracks at 9.53 cm per second.

Time Code

IRIG 'C" time code was recorded during all decompressions on the Grass Model III A and the Sanborn 150 from an EECO Model 811 time code generator. IRIG 'B' was recorded on the Ampex FR 600 from the same source.

Sampling Procedure

Physiological recording began simultaneously with the closing of the decompression chamber door and continued until the completion of the first performance session. Recording was resumed one hour before decompression and was continued until the animal returned to behavioral baseline performance. Sample recordings consisting of ten minutes of each rest period and ten minutes of each work session were taken thereafter until the animal was removed from the chamber.

RESULTS

Electroencephalography

Each rapid decompression experiment has been analyzed separately with a description of the visible changes occurring in the EEG record. As the areas of recording remain the same throughout any single rapid decompression, only one record has channel descriptors.

Jim (62) RD 1/5"

No noticeable EEG changes occurred in this record other than artifacts due to the physical effects of decompression.

Fig. 3. EEG recording technique showing implanted chimpanzee (RD 9/150") lead wires and Grass Model III A Electroencephalograph.

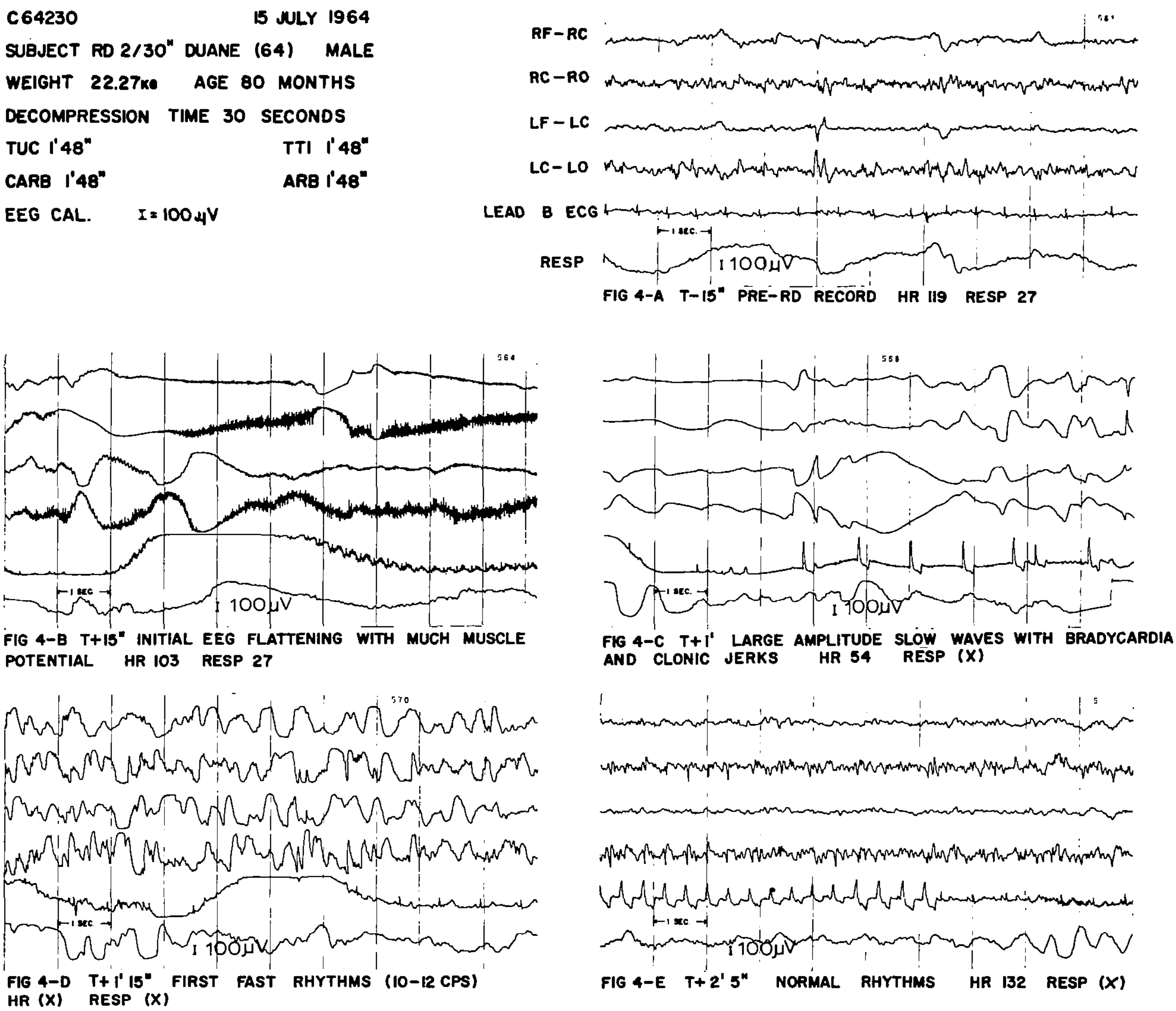


Fig. 4. Records of RD 2/30".

Duane (64) RD 2/30"

The results for this subject were shown in Figure 4. There is an initial flattening of the record with muscle action potential being the only visible activity (Fig. 4-B). Sixty seconds after RD there is a burst of high amplitude slow waves accompanied by clonus (Fig. 4C). Ten seconds later this activity, although still slow (2-4 cps), has bursts of 10-12 cps fast activity superimposed (Fig. 4-D). Within 80 seconds the fast activity has become dominant and the slow waves have been considerably reduced. Within 120 seconds the cortical rhythms have returned to essentially the same pattern as seen prior to the RD, although they are of higher amplitude (Fig. 4-E).

Jim (62) RD 3/60"

This record was unusable due to technical problems involved in recording the EEG. There is an indication, however, that this recovery pattern is similar to RD 5/120.

Rufe (114) RD 4/90"

This record shows the progression of recovery from a longer period (90 seconds) of decompression. Within 50 seconds following recompression, there is a burst of high amplitude slow waves with no visible fast activity (Fig. 5-B). This is followed by approximately two minutes of near electrical silence consisting of very low voltage slow waves of 1 or 2 cps. This period is followed by increasing slow waves (Fig. 5-C) with another 90 seconds passing before the first fast activity of 10-12 cps appears (Fig. 6-A). In three minutes the fast activity is beginning to dominate (Fig. 6-B) and the slow waves have virtually disappeared. It is not until eleven minutes later, however, that electrical shocks applied as reinforcement during the psychological program appear to activate the EEG record to the pre-RD level (Fig. 6-C).

Clayton (130) RD 5/120"

With a two minute period of decompression there is a decay of activity into electrical silence as expected. However, in this subject[1] the recovery period does not follow the usual pattern shown in

[1] Two decompression subjects'(RD 3/60" and RD 7/90") records were unusable due to technical problems, but they suggested a similar recovery pattern to RD 5/120".

this study. There is no early burst of high amplitude slow waves such as are found in experiments RD 2/30", RD 4/90", RD 6/150", RD 8/120" and RD 9/150". Rather, there are low amplitude slow waves appearing approximately 90 seconds after recompression, and they slowly increase (Fig. 7-B) for another minute before faster rhythms become noticeable (Fig. 7-C). The first rhythmic bursts of activity (10-12 cps) appear in another four minutes, although the record is still dominated by slow activity (Fig. 8-A). Within two minutes the fast activity (10-12 cps) has become dominant (Fig. 8-B). Approximately 20 minutes later the record has almost returned to the pre-RD pattern. Respiration is rhythmic as if the subject were drowsy (Fig. 8-C).

Shirley (116) RD 6/150"

This subject is one of two that were decompressed for two and one-half minutes. This record will therefore be described in more detail. Figure 9-B shows a burst of muscle activity and entrance into electrical silence at what behaviorally was considered the end of useful consciousness. There is bradycardia approximately one minute later during decompression (Fig. 9-C). Figure 9-D shows the moment of recompression with cortical electrical silence and an irregular but increased heart rate. A later period showing tachycardia is soon followed by the restoration of respiration (Fig. 9-E). The electrical silence of the cortex does not change for another 70 seconds at which time high amplitude slow waves occur (Fig. 10-A). The duration of this burst is approximately 25 seconds and is then followed by another 100 seconds of electrical silence. This last period of silence is followed by the appearance of low amplitude slow cortical activity (Fig. 10-B), which within 30 seconds has progressed to high amplitude slow waves (Fig. 10-C). This type of activity continues and increases slightly in frequency (Fig. 10-D). Twenty minutes following rapid decompression the first signs of a faster rhythm can be noted (Fig. 11-A). However, for the next ten minutes these rhythms do not consistently remain, but after a series of electrical shocks received during the behavioral program they become more noticeable. Six minutes later, although there is still a large amount of slow activity, the faster activity of 10-12 cps is clearly and continuously superimposed. This pattern, which continued with the rhythmic fast activity becoming more dominant, can be most clearly seen just preceding a performance period 72 minutes after RD when the subject was drowsy (Fig. 11-B). Five minutes

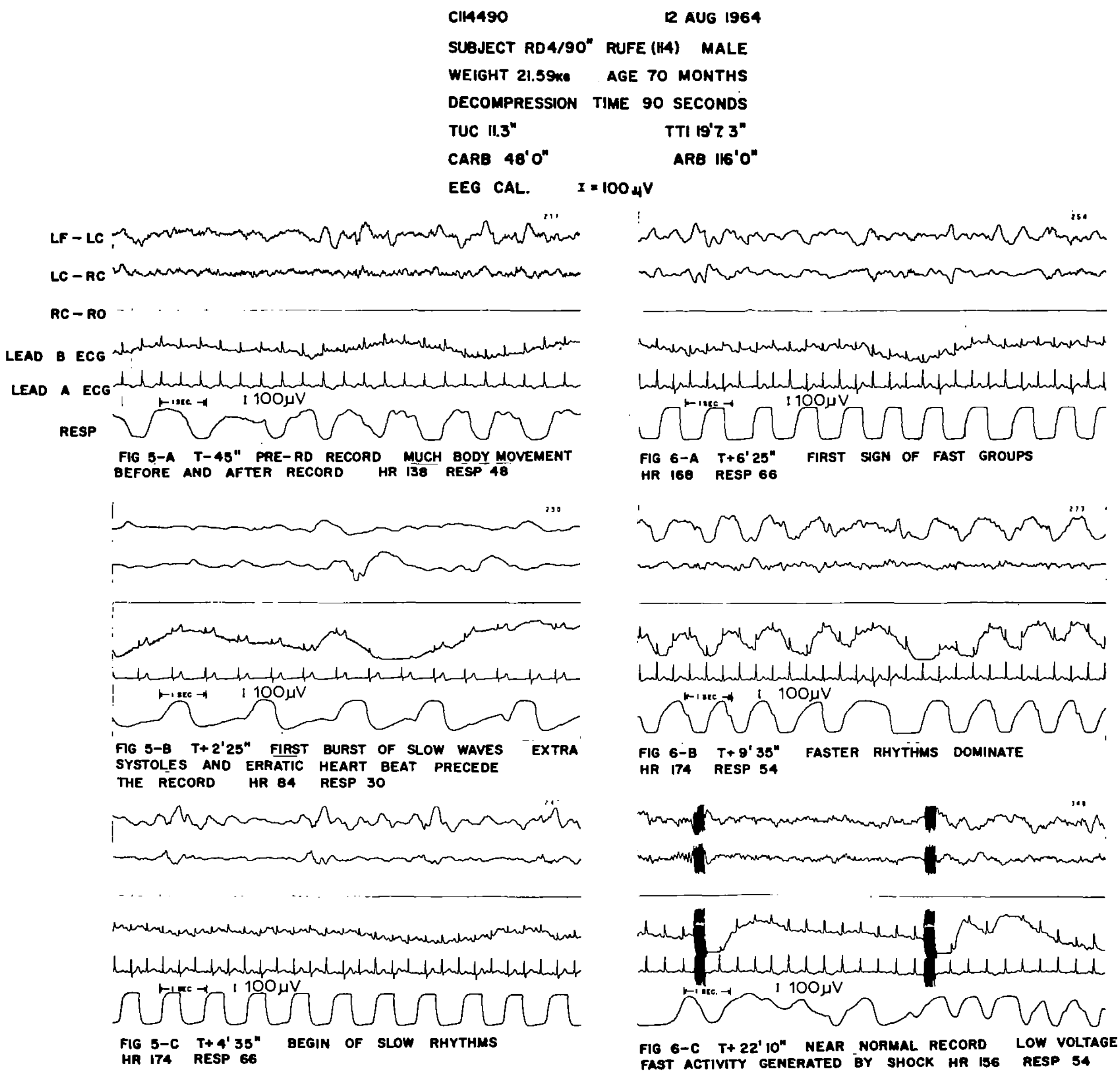


Fig. 5 & 6. Records of RD 4/90".

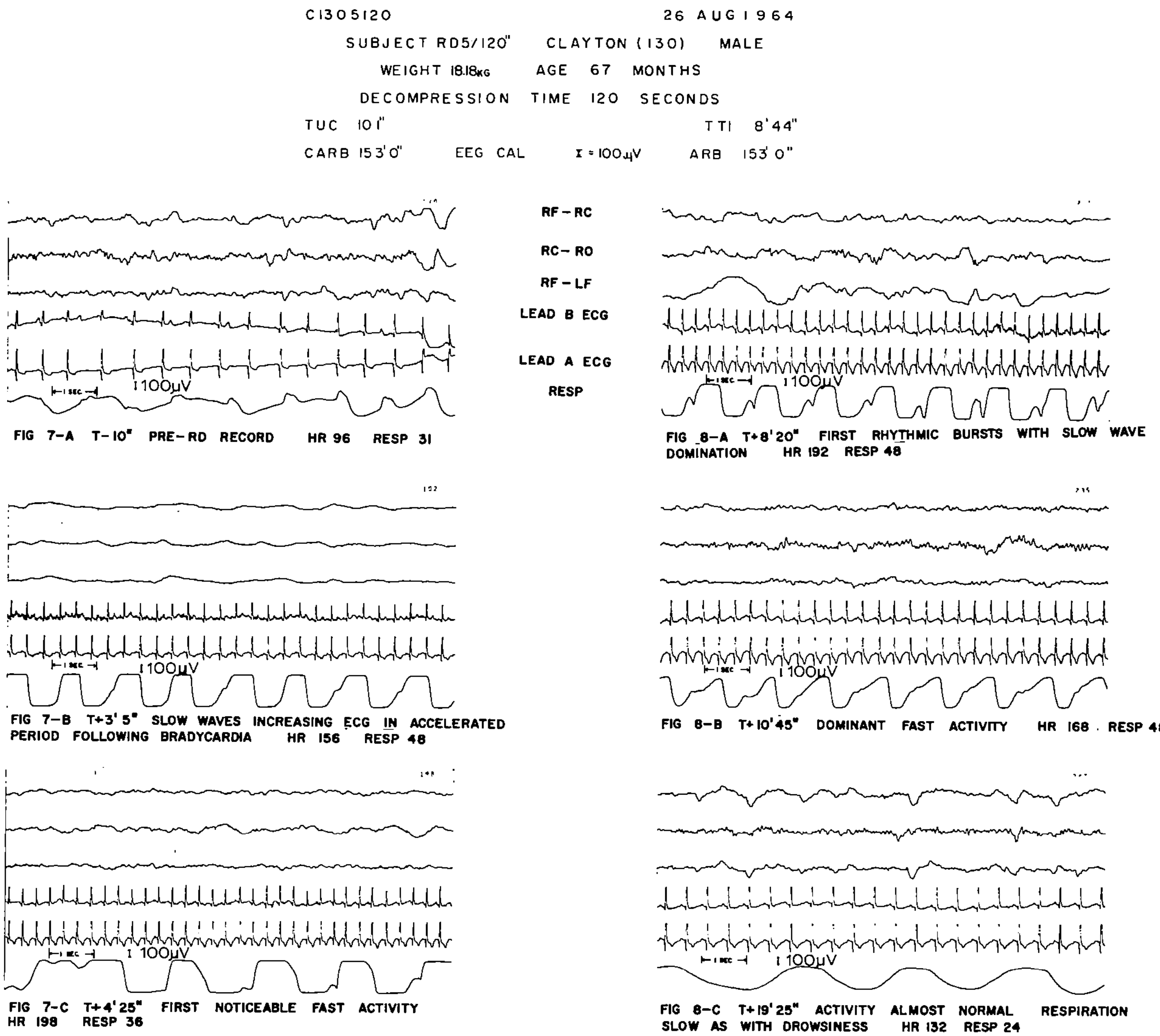


Fig. 7 & 8. Records of RD 5/120".

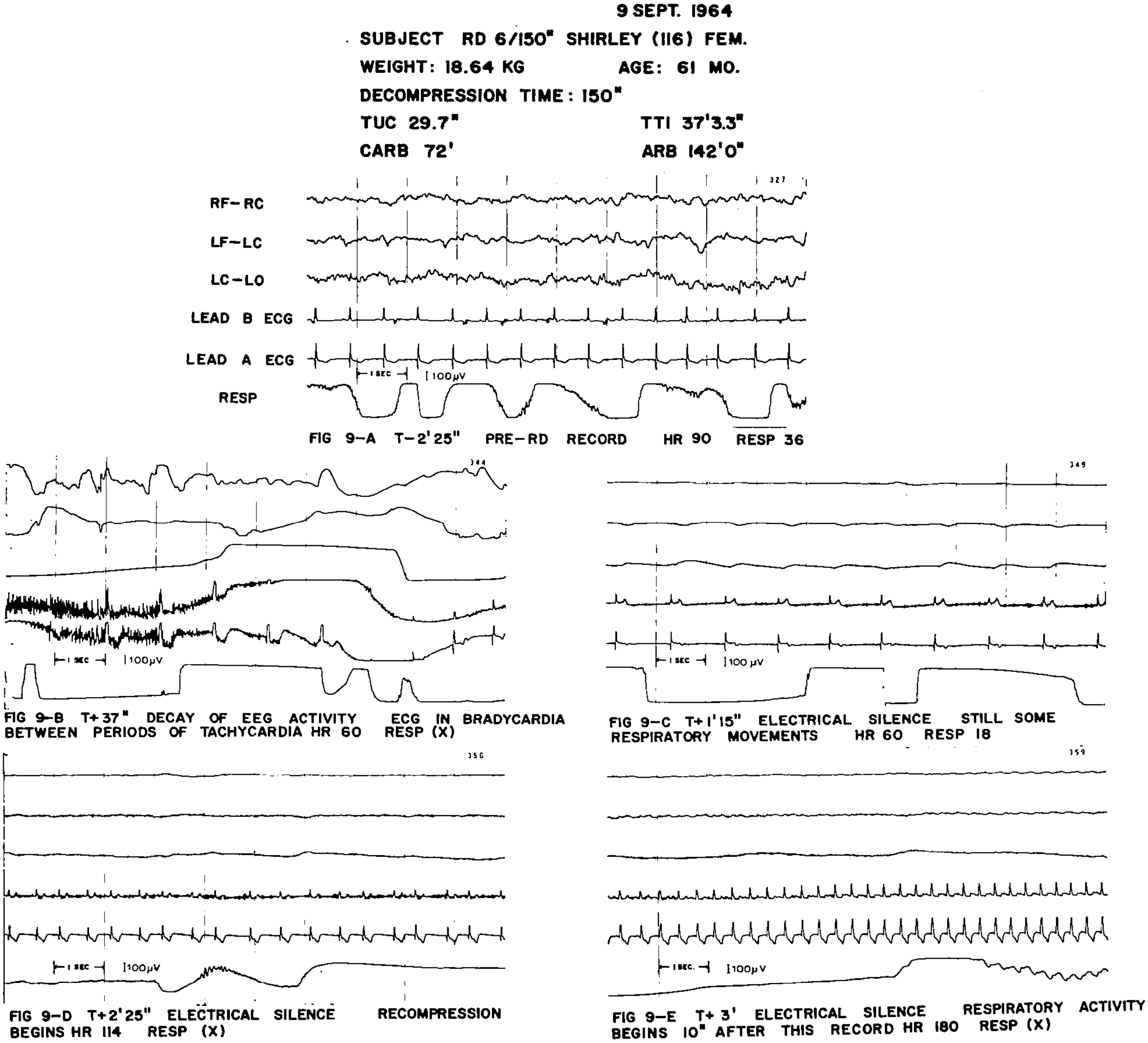


Fig. 9. Records of RD 6/150".

later, after a performance session (Fig. 11-C), the record, though marred by movement artifacts, is similar to the pre-RD pattern. However, even three hours after RD (Fig. 11-D), the pattern is still slower than that of a record taken several hours before RD (Fig. 11-E). The rhythmic respiration in Figure 11-D suggests a state of drowsiness which might explain, in part, the difference between the two records.

Brownie (120) RD 7/90"

This record was largely unusable due to electrical difficulties but resembles the recovery pattern of RD 5/120".

Lulu (121) RD 8/120"

Rapid decompression of this subject was followed by the usual pattern of cortical recovery. The first recovery signs from the cortex occur 60 seconds after recompression (Fig. 12-B) as high amplitude slow waves (1-2 cps) lasting about ten seconds. After two minutes of electrical silence, slow waves of moderate amplitude reappear but are soon followed by rhythmic fast activity of 10-12 cps (Fig. 12-C). Both types of activity continue during the next eight minutes with the slow waves becoming less dominant. Periods of electrical shock are followed by increased fast activity (Fig. 13-A). Within 24 minutes the record has a similar pattern to that of the pre-RD record, although it is of slightly lower amplitude (Fig. 13-B). An apparently normal record is seen after another five minutes (Fig. 13-C).

Heart Rate and Respiration

No attempt has been made to intensively analyze these parameters. The ECG electrodes were in a non-standard position, making interpretation other than heart rate difficult. In general, both measures confirmed what is already well known in the literature (Refs. 4, 16, 28). There was an initial tachycardia at the onset of decompression followed by respiratory arrest and a bradycardia. In the longer decompressions, there was a tachycardia following the bradycardia before recompression and continuing after recompression to rates as high as 198 beats per minute. (Fig. 14). There is no readily visible relationship of either heart rate or respiration to performance ability or cortical electrical activity, but they do

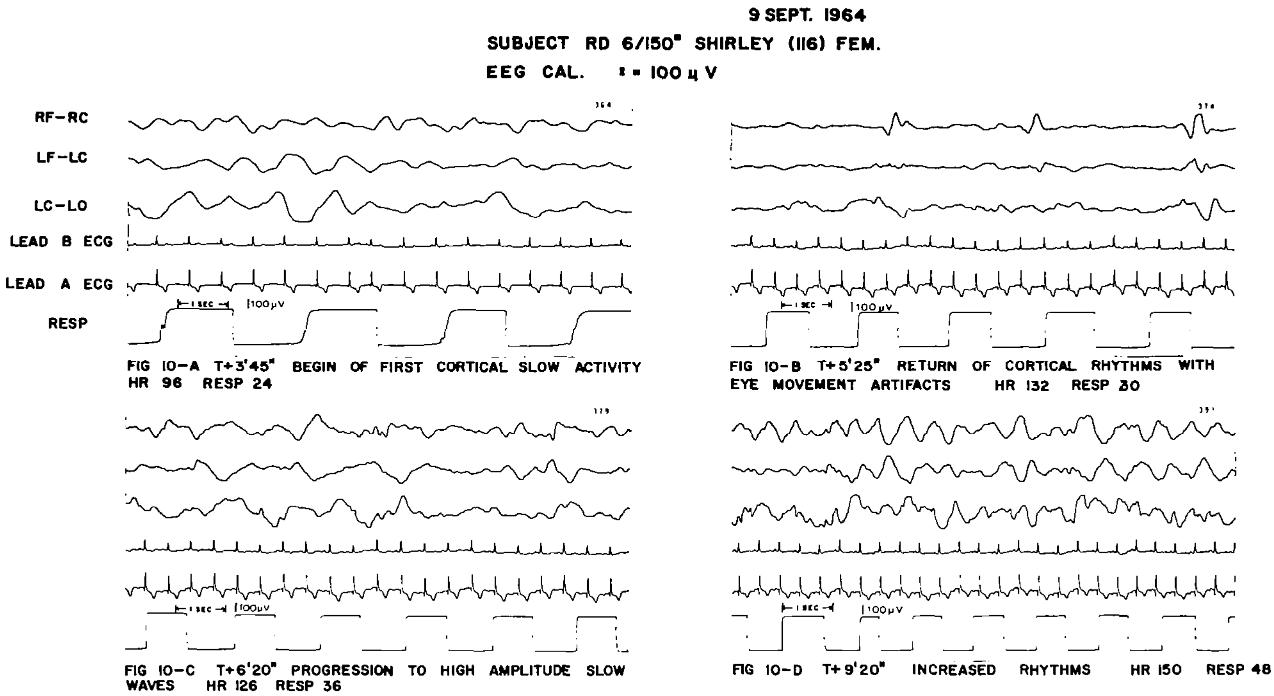


Fig. 10. Records of RD 6/150".

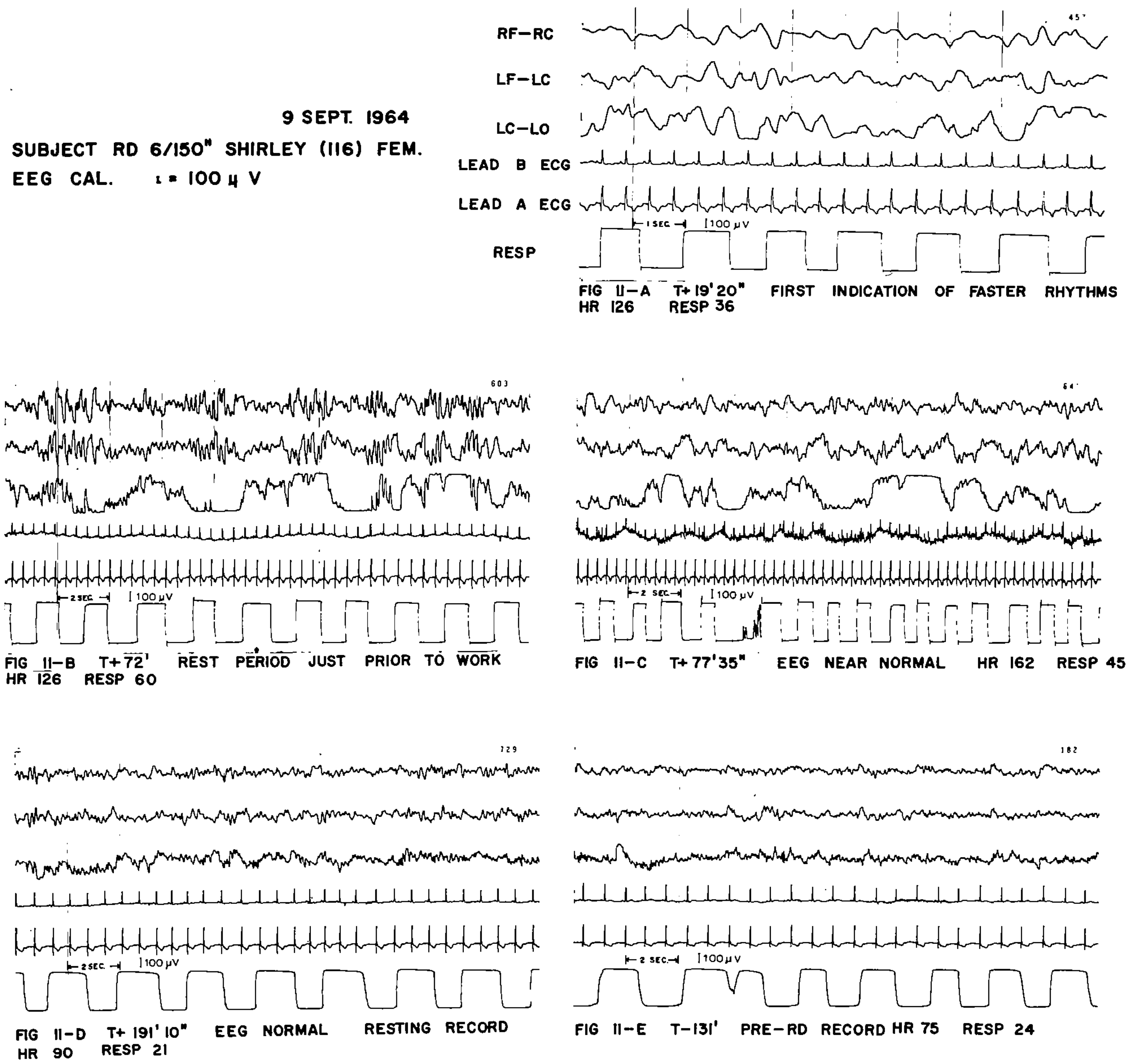


Fig. 11. Records of RD 6/150".

appear related to the stress phenomena of the decompression. The changes in both which do occur, however, suggest that other more sensitive measures such as blood flow and oxygen tension might reveal significant relationships particularly with the end of total impairment.

DISCUSSION

There are several areas of consideration pertinent to this evaluation of cortical electrical activity during rapid decompression.

Of most importance is the obvious feasibility of using the EEG as an indicator of performance impairment. On the basis of the preliminary results reported here, it would seem that performance will be lowered when the EEG activity is slower than normal. This is not to say that EEG normalcy dictates normal performance, but rather, without it fully effective performance does not apparently return. The other subtle factors which may be involved after EEG "normalcy" has returned cannot be evaluated from these records. More sophisticated techniques of analysis such as those possibly available through advanced computer technology will be necessary before one can arrive at a more complete interpretation of cortical electrical activity and its correlation to performance ability.

A further problem, in terms of correlating performance and EEG under these conditions, comes from other possible physiological side effects. Previous studies on hypoxia (Refs. 12, 14, 21), even without dramatic pressure changes, have indicated that visual fields, convergence, retinal sensitivity, and audition are affected. There is a latent period of visual and auditory impairment following anoxia. The tasks necessary to evaluate the performance of these subjects require both visual and auditory sensitivity. Also, the lights used to present the various tasks were of several colors and of different intensities making an interpretation of the performance level in relation to cortical electrical activity somewhat clouded by possible physiological impairment. This suggests that the use of deep implanted electrodes may show some differentiation in brain activity not readily visible in the cortical record. Such a procedure might well lead to the recognition of subtle patterns previously unnoticed in the cortical record which are related to visual and auditory defects.

C8121120 12 NOV 1964

SUBJECT RD8/120" LULU (121) FEMALE

WEIGHT 25.45KG AGE 70 MONTHS

DECOMPRESSION TIME 120 SECONDS

TUC 9.5" TTI 19'13.6"

CARB ARB 59'0"

EEG CAL. I = 100µV

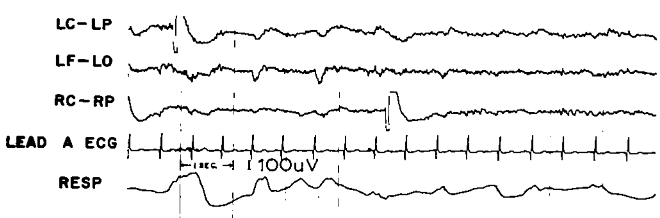


FIG 12-A T-30" PRE-RD RECORD HR 103 RESP (X)

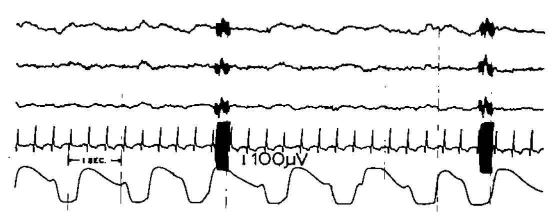


FIG 13-A T+10'50" INCREASED FAST ACTIVITY FOLLOWING SHOCKS HR 180 RESP 48

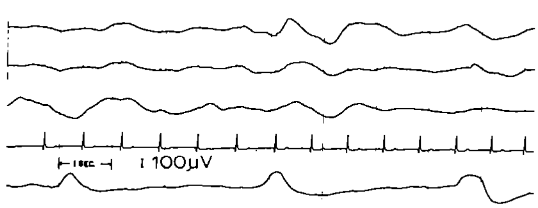


FIG 12-B T+2'55" FIRST BURST OF SLOW WAVES HR 84 RESP (X)

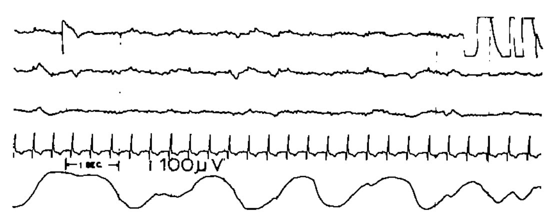


FIG 13-B T+25'50" NEAR NORMAL EEG HR 162 RESP 30

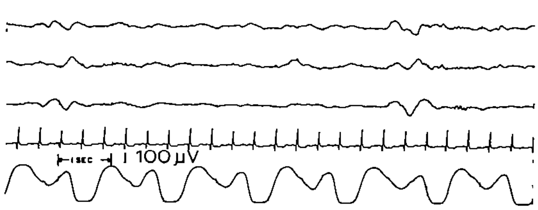


FIG 12-C T+5'30" FIRST FAST ACTIVITY HR 144 RESP 36

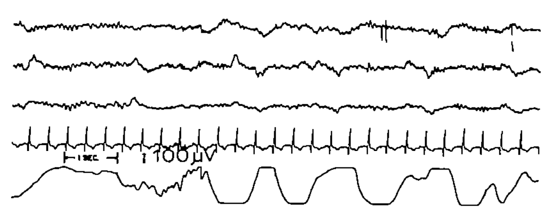
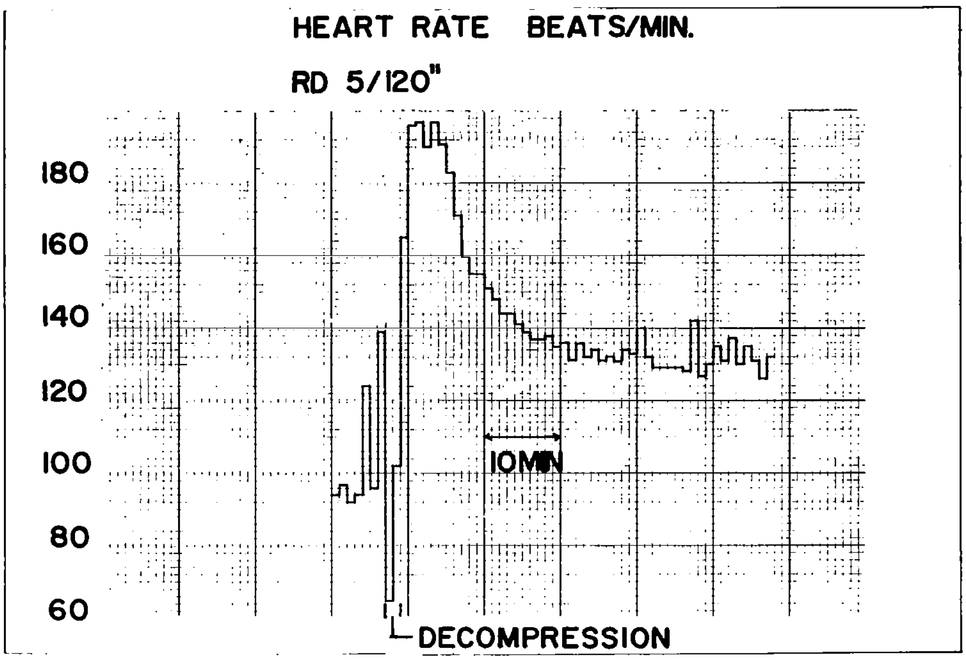


FIG 13-C T+28'35" NORMAL EEG HR 144 RESP 36

Fig. 12 & 13. Records of RD 8/120".



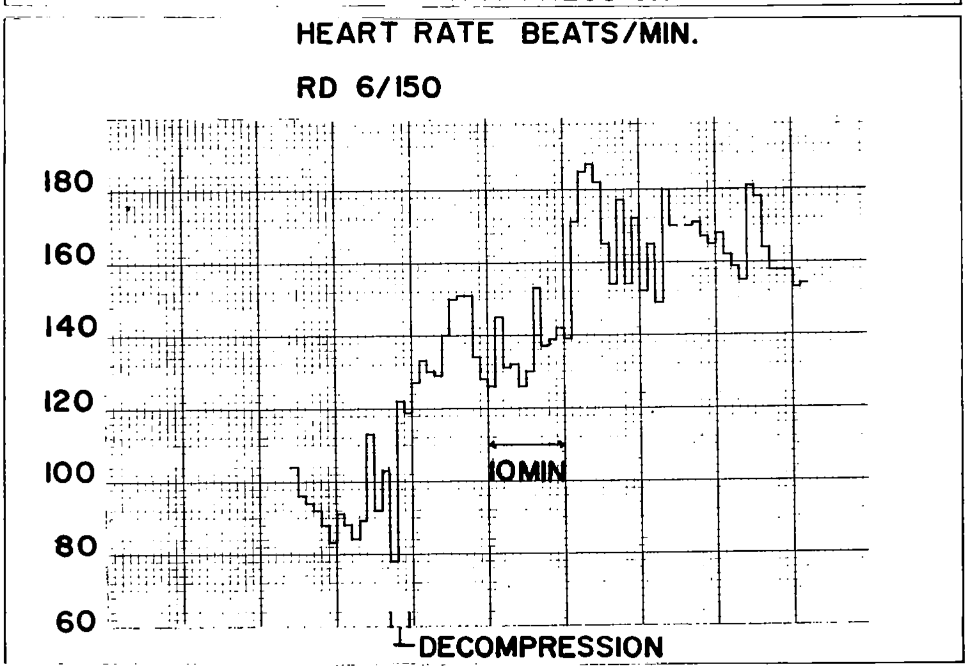


Fig. 14. Heart Rate records following Rapid Decompression which show two distinct patterns, immediate acceleration to a peak rate and slow continuous acceleration.

Finally, these EEG records show a recovery pattern after recompression that differs from anoxia and hypoxia experiments using lower mammals and to some extent from those experiments using humans. This suggests that many anoxia or hypoxia experiments may be misleading when applied to the extreme conditions of rapid decompression to less than 2 mm Hg. A possible cause for this deviation is that of methodology. Most hypoxia and anoxia studies involving the EEG have been conducted at much lower altitudes or at ground level with slow induction using gas mixtures having low oxygen content, decreasing the blood supply to the brain or other means. In light of these considerations it would seem wise to carry out more complete studies with the present methodology in order to be able to make valid comparisons with past work.

SUMMARY

Table I gives a summary of the EEG and behavioral data. It can be readily seen that the end of total impairment does not occur until the appearance of some fast activity. In fact, in the shorter decompression periods the EEG has essentially returned to normal. With the 30 second decompression, the end of total impairment, return to CA baseline, and a normal EEG occur at approximately the same time. With all other periods of low pressure, however, the return to CA baseline level of response did not return, until after normalcy of the EEG had been achieved.

While the present study has perhaps raised more questions than it has answered, it has demonstrated the feasibility of using the EEG as an indicator for evaluation of performance following rapid decompression to 1 to 2 mm Hg. Fast activity (10-12 cps) always preceded the end of total performance impairment. This relationship, in light of other neurophysiological studies, may well be a reflection of a particular system within the brain such as the ascending reticular activating system (ARAS) of Magoun. The work of Hugelin, et al, has strongly suggested that the lower brain stem is the most resistant to anoxia and presumably the first to recover. If these fast waves were produced by a particular system, separate from whatever system was producing slow waves, it would explain how the end of total impairment could come about even though slow waves were importantly present. This might further implicate a third system for normal performance to return which, in higher primates, might be rhinencephalic in origin.

TABLE I

Experiment	*First Cortical Activity		*Time to Consistent Activity	*Time to Fast Activity	**Time of Total Impairment	*Time to Normal EEG	**Time of Return to CA Baseline
	Latency to	Type of					
RD 2/30"	30"	High amplitude slow waves	30"	45"	1'48"	1'30"	1'48"
RD 4/90"	55"	High amplitude slow waves	3'5"	4'55"	19'0"	20'0"	48'0"
RD 5/120"	1'5"	Low amplitude slow waves	1'5"	2'25"	8'30"	28'30"	153'0"
RD 6/150"	1'15"	High amplitude slow waves	2'55"	16'50"	37'0"	75'5"	72'0"
RD 8/120"	55" =	High amplitude slow waves	32'0"	2'30"	19'13"	26'35"	59'0"

* Times from Recompression
** Times from Decompression

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SECTION III

EXPERIMENTAL SURGERY AND CLINICAL EVALUATION

Jerry Fineg, Major, USAF, VC
Donald C. Van Riper, Captain, USAF, VC
Phillip W. Day, Captain, USAF, VC
James R. Prine, Major, USAF, VC

Veterinary Division
657lst Aeromedical Research Laboratory
Aerospace Medical Division
Air Force Systems Command
Holloman Air Force Base, New Mexico

ACKNOWLEDGMENTS

The assistance of the following persons is gratefully acknowledged:

Loyd M. Stephens and James Bartlett	-	Development of the electrode plug
Robert C. Frantz and Bobby J. Teal	-	Surgical Assistants

In addition, appreciation is expressed to the clinical laboratory and veterinary technicians for their support.

ABSTRACT

Surgical procedures are outlined for implanting chronic cortical leads in the chimpanzee. Criteria for physical evaluation of the subjects are presented.

All subjects showed slight neutrophilia, increased transaminase levels and facial edema, which were transient in nature and returned to normal within 72 hours after decompression to less than 2 mm Hg. There seems to be no lasting effect of rapid decompression exposure to a neaı vacuum.

INTRODUCTION

In support of the experiment outlined in Sections I and II of this report, the veterinary responsibility was two-fold: (1) to establish an acceptable, chronic cortical lead implant procedure for electroencephalographic (EEG) measurement on the chimpanzee (Ref. Sect. II), and (2) to evaluate, pre- and post-rapid decompression (RD), the clinical status of the subjects based upon physical examination and laboratory analysis.

METHOD

Experimental Surgery

EEG activity was recorded on Subjects 1 through 6 utilizing the techniques outlined in Physiology, Sect. II of this paper. Measurements on Subjects 7-9 involved the same parameters, however, the implant procedures were modified in an effort to arrive at a more meaningful EEG tracing and a truly permanent 8 lead cortical implant. The procedure outlined in this paper is the one which has proven to be the most satisfactory and, in an effort to attain a standardized procedure to insure reproducibility of encephalographic tracings, is the planned approach for all future endeavors in this direction.

Chimpanzees selected for the RD study were given a complete physical examination prior to surgery (see Fig. 8). Subjects were prepared for the EEG implant procedures by administering Demerol* (11 mg/kg) and atropine or scopolomine (.2 to .4 mg total dose). The head was then clipped, treated with Surgex** and prepared for surgery. The chimpanzee was

* Meperdine hydrochloride, Winthrop Laboratories
** Depilatory, Crookes-Barnes

then placed in a prone position on the operating table and prepared for a general surgical procedure utilizing intubation and gas anesthetics (combinations of oxygen and Fluothane*) (Ref. 1). When the subject reached the surgical plane of anesthesia, the head was positioned in a stereotaxic apparatus (Fig. 1). Final preparation of the head was then made and the surgical area draped.

An incision was made 2.5 cm to the left of the midline extending 5 cm posteriorly from the anterior border of the supraorbital ridge. The skin and periosteum were reflected. A dental drill and small bur (No. 8 round) were used to form a hole in the skull at a point 2 cm posterior to the anterior angle of the initial incision. The hole was prepared by undermining the boney skull to form a concave cavity large enough to accommodate a size 4-40 screwhead**. Four holes were then drilled equi-distant (.5 cm) around the cavity to serve as anchor points for the Kadon*** base. The 9 lead single electrode plug, fitted with a single screw (head down), was then inserted into the center cavity and dental Kadon applied to form a base 2 cm in diameter (Fig. 2). Upon completion of this procedure, the incision was extended to form a ⊔ to accommodate eight holes, drilled approximately 3.2 cm lateral to the midline (4 holes per sagital plane). The anterior pair of holes was located 4 cm behind the supraorbital ridge; the posterior set was 2.5 cm anterior to inion. The 2 medial pairs of holes were drilled to divide the intervening space between the anterior and posterior positions into equal segments (Fig. 3). A 9th hole for the ground electrode was drilled 2.5 cm directly behind the main plug.

The hole positions described above represent a fixed area over the frontal, parietal and occipital lobes, respectively. At no time was the dura penetrated. The holes were connected with each other by a small groove, 1.5 mm in depth, made with a dental drill and burr (No. 557 fissure). EEG electrode screws (size 2-56)**** were then threaded into the holes. Small baked

---

* 2-bromo, 2-chloro, 1,1,10tri fluorethane, Ayerst

** 12.7 mm long flat head, stainless steel screw

*** Kadon Resin (R), L.D. Caulk Company, Milford, Delaware

**** 3.1 mm long, round head, stainless steel machine screw

enamel coated wires (.226 mm in diameter) originating at the rear of the main plug were soldered to the screw-heads, placed in the grooves, and cemented in place with Kadon (Fig. 4). The periosteum was closed with 2-0 plain gut, the skin was closed with interrupted 2-0 silk sutures (Figs. 5 and 6) and a pressure bandage was applied. Manipulation of the surgical area was prevented by utilizing a plexiglass dome over the subject's head for approximately 10 days. The dome is constructed to avoid interference with eating or drinking (Fig. 7).

Clinical Evaluation

All chimpanzees selected for the RD study were stabilized in the 6571st ARL laboratory for not less than one year. All had undergone some psychomotor conditioning (Ref. Sect. I, Psychology).

The pre-surgical evaluation of the subjects consisted of a complete physical examination as outlined in Figs. 8, 9, 10, 11, 12 and Ref. 2.

Upon completion of the surgery for EEG implantation, the chimpanzees were allowed to recover for a period of 3 weeks.

The 24 hour pre- and post-RD clinical evaluation of the subjects was limited to those measurements outlined in Figs. 9, 10, 11, 12 and 13.

CONCLUSIONS

All subjects, upon initial examination, following RD, showed slight elevations in neutrophiles and transaminase* levels in the blood which, in the authors' opinion are considered as normal reactions to stresses of this magnitude (Refs. 3, 4).

---

* Serum Glutamic Oxylacetic Transaminase (SGOT) and Serum Glutamic Pyrubic Transaminase (SGPT)

Three out of the 8 chimpanzees exposed to RD showed a slight increase in Serum Potassium and Sodium levels and elevated Hematocrits. It is suggested that these increases were probably due to transitory dehydration incident to the refusal of the subject to take advantage of all positive reenforcement (water and food) opportunities presented during the rapid decompression test period.

All subjects showed facial edema, injected sclera and occular discharge for 48-72 hours post-RD. No clinical signs of neurologic involvement were detected. All clinical laboratory values were within normal limits by 72 hours post-RD.

The latest EEG electrode implant procedures have proven quite satisfactory. An effort will be made to continue the improvement of procedures as more information is derived from testing and better monitoring equipment is developed.

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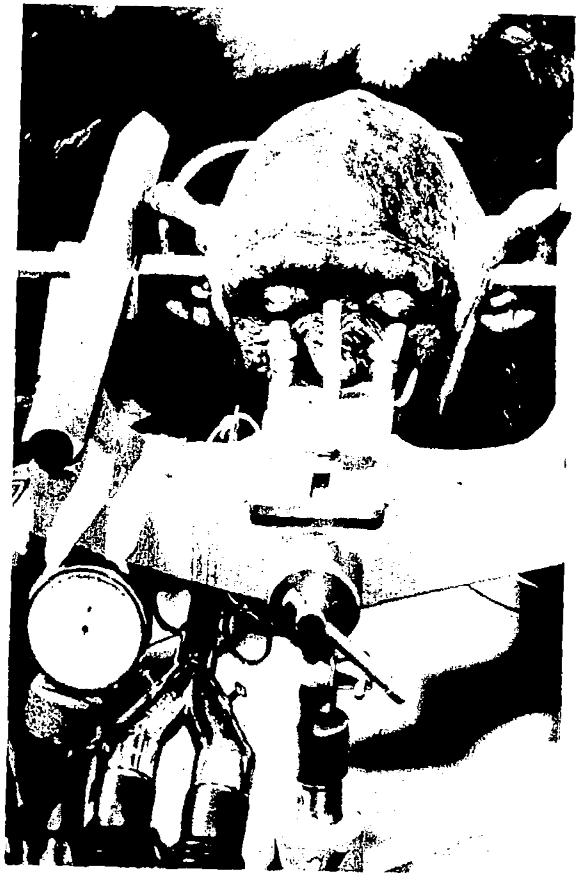


Fig. 1. Stereotaxic apparatus

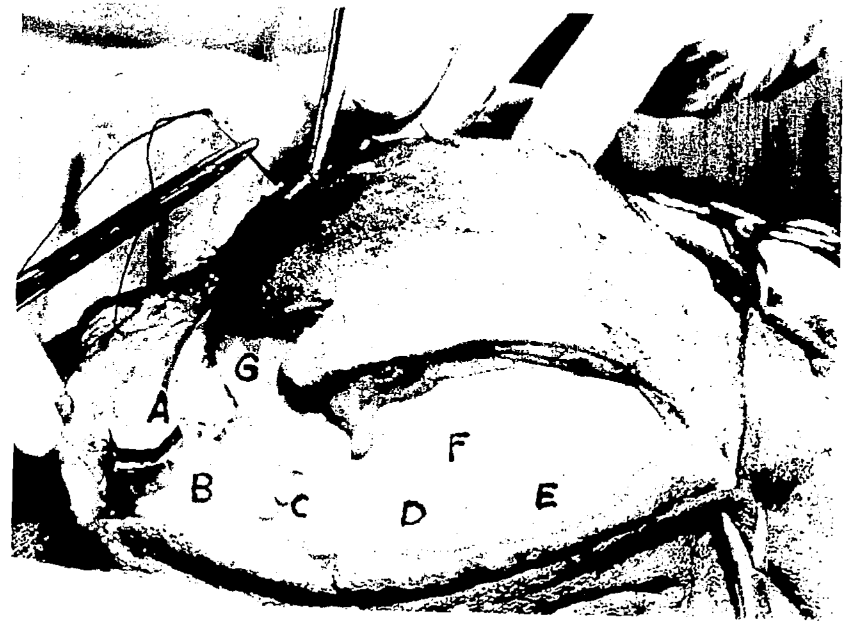


Fig. 2. Lateral view of head showing plug (A) with Kadon base (B), location of lateral screws (C), (D), (E) and groove connecting them (F), ground (G).

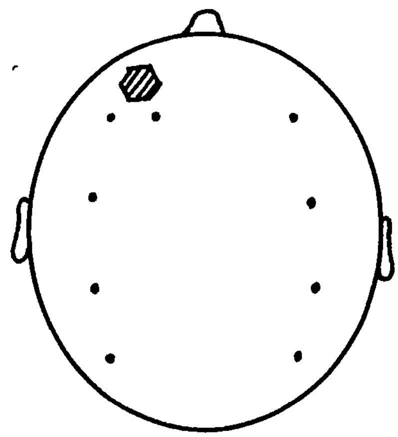


Fig. 3. Diagram showing location of plug and lateral screws.

Fig. 4. Filling the grooves with Kadon (R)

Fig. 5. Closure of skin with interrupted 2-0 silk sutures

Fig. 6. Closure of skin with interrupted 2-0 silk sutures

Fig. 7. Chimpanzee with post-operative protective helmet.

PHYSICAL EXAMINATION - PRIMATE

STATISTICS

IDENTIFICATION NR	NAME		SEX ♂ ♀	AGE *(Mos)*	WEIGHT ____KILO____LBS
	RESTRAINT	TEMP °F	PULSE/MIN	RESP/MIN	BLOOD PRES
BLOOD TYPE	NONE				
	STRAP				
[ ] ANNUAL [ ] SEMI ANNUAL [ ] QUARTERLY [ ] OTHER	ANESTHESIA				

If anesthesia is utilized, describe Type, Mode of Entry, Amount and Duration:

GENERAL APPEARANCE/CONDITION

	NORMAL	ABNORMAL *(Explain)*
SKIN		
EENT		
LUNGS *(Auscultation)*		
HEART *(Auscultation)*		
ABDOMEN *(Palpation)*		
EXTREMITIES *(Palpation)*		
REFLEXES		

CLINICAL TESTS

ECG
STANDARD ______
SPECIAL ______

EEG
STANDARD ______
SPECIAL ______

XRAY SETTINGS MA

CHEST ______ ABDOMEN ______ HEAD ______ EXTREMITIES ______ BITE WINGS ______

LABORATORY FORMS *(R-routine B-bacteriologic P-parasitic BC-biochemical X-special)*

[ ] **FECAL** [ ] **BLOOD** [ ] **URINALYSIS** [ ] **DENTAL** [ ] **OTHERS**

HISTORY

ORIGIN

PROGRAM UTILIZATION

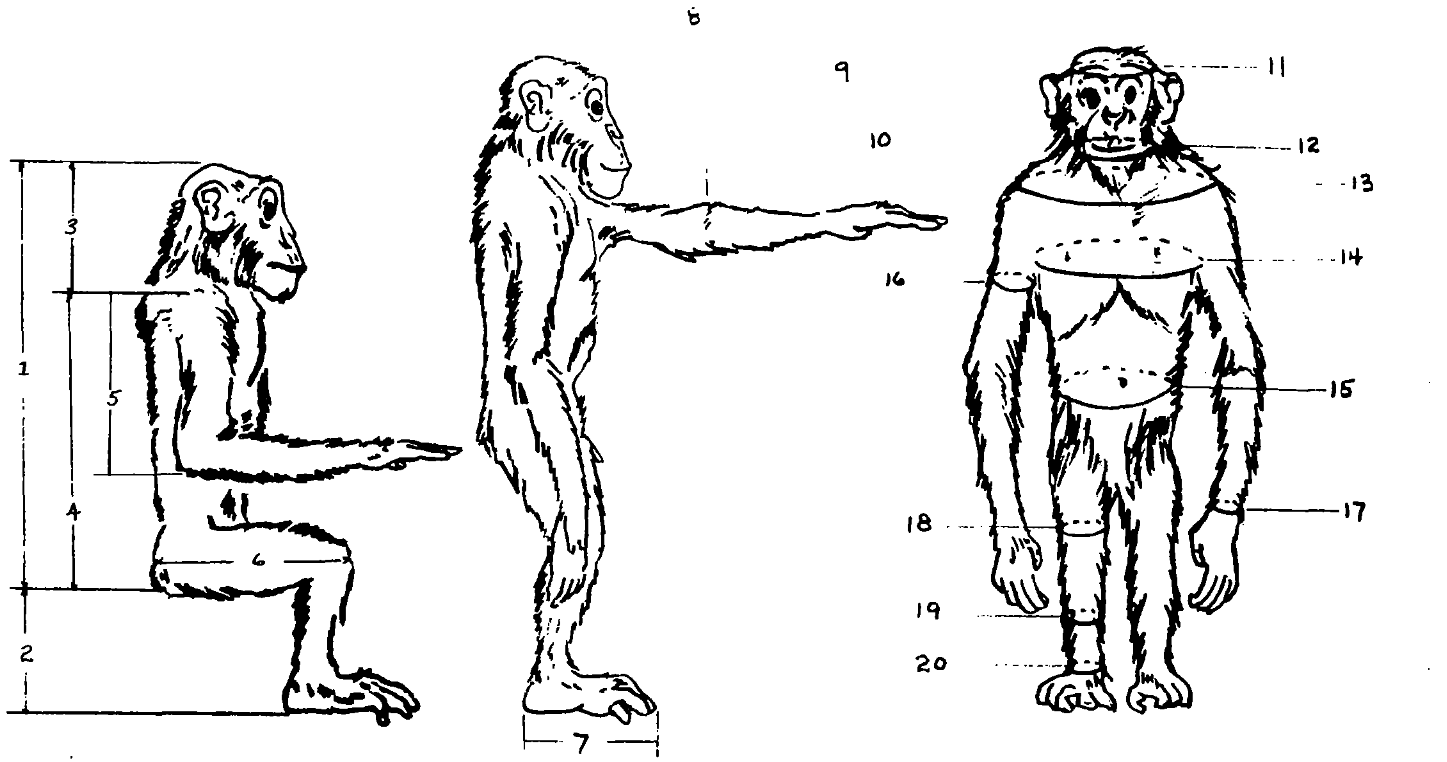
TUBERCULIN TEST [ ] POSITIVE [ ] NEGATIVE

DATE

REMARKS

ARL FORM DEC 64 **0-1** (ARSVV) PREVIOUS EDITIONS OF THIS FORM ARE OBSOLETE

Fig. 8. Physical Examination Form



LOCATION OF BODY MEASUREMENTS

LENGTH IN INCHES				CIRCUMFERENCE IN INCHES			
1. BUTTOCK - CROWN		6. BUTTOCK - KNEE		11. HEAD		16. BICEPS	
2. POPLITEAL HEIGHT		7. HEEL - TOE *(Foot)*		12. NECK		17. WRIST	
3. TOP HEAD - TOP SHOULDER		8. TOTAL ARM REACH		13. SHOULDER		18. THIGH *(Mid shaft)*	
4. ACROMIAL HEIGHT		9. FOREARM - HAND		14. CHEST		19. CALF	
5. SHOULDER - ELBOW		10. HAND		15. WAIST *(Hip)*		20. ANKLE	

REMARKS

VETERINARIAN / TECHNICIAN	DATE

Fig. 8a. Physical Examination Form

Rapid Decompression Study

Surgical
Pre
Post RD

REGISTER OR UNIT NO.	WARD NO.	☐ BED PATIENT ☐ AMBULATORY
REQUESTED BY		DATE OF REQUEST
DATE, TIME, AND METHOD OF COLLECTION		

PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

X	COLOR-APPEARANCE	
X	REACTION	
X	SPECIFIC GRAVITY	
X	ALBUMIN	
X	SUGAR	
	ACETONE	
	BILE	

X MICROSCOPIC: REMARKS

DATE OF REPORT	SIGNATURE *(Specify Lab. if not part of requesting facility)*
NAME OF MEDICAL FACILITY	

Standard Form 514—A—Rev. June 1959.
Bureau of the Budget Circular A-32 GPO c58—16—56268-6 URINALYSIS

Fig. 9. Tests run on urine

Rapid Decompression Study

Surgical
Pre
Post RD

REGISTER OR UNIT NO.	WARD NO.	☐ BED PATIENT ☐ AMBULATORY
REQUESTED BY AND DATE		DATE COLLECTED
CLINICAL DATA		

PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

X	W.B.C.		X	R.B.C.
X	DIFFERENTIAL COUNT		X	HEMATOCRIT
	NEUTROPHILES		X	HEMOGLOBIN
	BLASTS			BLEEDING TIME
	MYELOCYTES			COAGULATION TIME
	BANDS			BLOOD MORPHOLOGY; REMARKS
	LYMPHOCYTES			
	MONOCYTES			
	EOSINOPHILES			
	BASOPHILES			
	PLATELETS			
X	SEDIMENTATION RATE			
	C.S.R.			

DATE OF REPORT	SIGNATURE *(Specify Lab. if not part of requesting facility)*
NAME OF MEDICAL FACILITY	

Standard Form 514-B—Rev. June 1959.
Bureau of the Budget Circular A-32 GPO c58—16—56274-6 **HEMATOLOGY**

Fig. 10. Tests run on blood

Rapid Decompression Study

Surgical
Pre
Post RD

REGISTER OR UNIT NO.	WARD NO.	☐ BED PATIENT ☐ AMBULATORY
REQUESTED BY AND DATE		DATE AND HOUR COLLECTED
CLINICAL DATA		

PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

X	UREA N			TRANSAMINASE	
	GLUCOSE		X	Sodium	
	CHOLESTEROL		X	Potassium	
	ESTERS		X	Chloride	
	BILIRUBIN (TOTAL)		X	Calcium	
	BILIRUBIN (DIRECT)		X	Bicarb.	
	B.S.P.		REMARKS:		
	THYMOL TURBIDITY			pH SGOT	
	CEPHALIN FLOCCULATION	24 HR. 48 HR.		SGPT	
X	PROTEIN TOTAL		DATE OF REPORT	SIGNATURE *(Specify Lab. if not part of requesting facility)*	
	ALBUMIN				
	ALKALINE PHOSPHATASE		NAME OF MEDICAL FACILITY		
	ACID PHOSPHATASE				

Standard From 514-D—Rev. June 1959.
Bureau of the Budget Circular A-32

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BLOOD CHEMISTRY

Fig. 11. Chemical determinations run on blood

Rapid Decompression Study

Surgical
Pre
Post RD

REGISTER OR UNIT NO.	WARD NO.	☐ BED PATIENT ☐ AMBULATORY
REQUESTED BY AND DATE		DATE AND TIME COLLECTED
CLINICAL DATA		

PATIENT'S LAST NAME—FIRST NAME—MIDDLE NAME

X	APPEARANCE				
X	CONSISTENCY				
X	BLOOD—GROSS				
X	OCCULT				
X	PUS		REMARKS		
	MUCUS		Concentration method		
	BILE				
X	OVA AND PARASITES				
			DATE OF REPORT	SIGNATURE (*Specify Lab. if not part of requesting facility*)	
			NAME OF MEDICAL FACILITY		

Standard Form 514-G—Rev. June 1959.
Bureau of the Budget Circular A-32

U. S. GOVERNMENT PRINTING OFFICE: 1961-609973

FECES

Fig. 12. Tests run on feces

PHYSICAL EXAMINATION PRE AND POST TEST				
TEST	PROJECT/TASK NR	ANIMAL IDENTIFICATION	SEX	DATE
PAST CLINICAL HISTORY				

DATA	PRE TEST	POST TEST
WEIGHT/LBS RECTAL TEMP F^0 BLOOD PRESSURE PULSE/MIN RESPIRATION/MIN ANESTHESIA/RESTRAINT	IF USED AMOUNT, TYPE AND TIME	
E. C. G. X-RAY	OBSERVATIONS:	OBSERVATIONS:
MEASUREMENTS PERTINENT TO TEST		

REMARKS [General appearance and mental status, eye, ear, nose and throat, chest (lungs and heart sounds), abdomen (palpation), extremities]	
PRE	POST

ARL HQ FORM FEB 62 0-4 (ARSVV)

Fig. 13. Physical Examination Form

LABRATORY REQUIREMENTS
HEMATOLOGY *(Number of samples, times to be collected, type and amounts)*
URINALYSIS *(Number of samples, times to be collected, type and amounts)*
OTHERS
(Signature of Veterinarian/Assistant)

SECTION IV

PHYSICAL ENVIRONMENT

Thomas D. Magnuson, B.S.M.E.
Thomas L. Dobson, B.S.M.E.

Guidance and Control Directorate
Deputy for Guidance Test
Air Force Missile Development Center
Air Force Systems Command
Holloman Air Force Base, New Mexico

SUMMARY

A modified Closed Environmental System Chamber (See Section I, Appendix I-A for description) was connected by a pneumatic decompression valve to an 84.9 cubic meter tank and a 48 cubic meter "Stratosphere Chamber." With the decompression chamber at 179 mm Hg and both parasitic tanks at approximately 200 microns, the resultant initial pressure amounted to approximately 2 mm Hg after decompression.

Recompression to 179 mm Hg in 30 seconds was accomplished with bottled oxygen through a regulator adjusted to 1,552 mm Hg. Five-second emergency recompression was constantly available through an oxygen bottle (25,860 mm Hg pressure) connected directly to the system. The life support sub-system was separated from the decompression chamber 5 seconds prior to decompression, and flow was resumed immediately after the chamber was recompressed to 179 mm Hg.

The chamber instrumentation system was augmented for the special conditions of this experiment. In addition to the system oxygen analyzer-recorder, three Brown strip-chart recorders were installed. A dual-pen recorder charted chamber wall and air temperatures which were sensed by copper-constantan thermocouples. A single-pen unit recorded pressures from 1 to 660 mm Hg, sensed by a Statham ± 646.5 mm Hg strain-gauge pressure transducer through a bridge circuit. The third recorder was used for accurate pressure readings between 1 and 2 mm Hg which was sensed by a Pirani tube through a CEC Autovac gauge. After RD 1/05", a fourth unit was installed to record relative humidity as sensed by a Honeywell Q457A lithium chloride sensor.

Nine tests were completed between 1 July 1964 and 12 January 1965. The environmental conditions for these tests are recapitulated in Table I. Temperature, relative humidity, and percent oxygen values are averages of 15-minute readings.

The chamber, valve, and piping connected to the vacuum tank are shown in Figure 1. Figures 2 and 3 show the life support sub-system, gas supply and the behavioral and physiological data instrumentation. A subject restrained in a couch in the decompression chamber is shown in Figure 4. Figure 5 shows environmental instrumentation.

TABLE I. Summary of Environmental Conditions

	Rapid Decompression Test								
	1/05"	2/30"	3/60"	4/90"	5/120"	6/150"	7/90"	8/120"	9/150"
1. Time to 100% O_2 * (mins.)	41	15	23	20	18	30	13	26	22
2. Time at 100% O_2 prior to ascent (hrs.)	3.0	3.0	3.0	3.0	3.0	3.0	2.7	3.1	3.0
3. Time to ascend to 179 mm Hg (mins.)	14	12	13	13	13	12	13	13	12
4. Time at 179 mm Hg prior to RD (mins.)	45	** 28 & 28	54	98	49	55	86	42	41
5. Temperature prior to RD (mean $^\circ$ C)	26.04	24.92	25.20	25.59	24.47	25.26	23.02	24.47	23.74
6. Humidity prior to RD (mean % RH)	43.5	34.0	34.5	36.1	35	33	35.5	34	35
7. Oxygen prior to RD (% O_2)	96.4	94.3	98.4	97.5	98.4	98.1	98.2	98.2	98.3
8. Pressure at near vacuum (mm Hg)	1.3	2.2	1.7-2.1	1.9-2.5	1.7-2.5	1.5-2.6	2.3-3.0	2.1-2.4	2.25-2.8
9. Time at near vacuum (secs.)	5	30	60	90	120	150	90	120	150
10. Time to recompress to 179 mm Hg (secs.)	30	30	30	30	30	30	30	28	29
11. Time at 179 mm Hg post RD (hrs.)	24	24	24.75	24	24	24	24	19.50	24
12. Temperature post RD (mean $^\circ$ C)	25.20	23.80	24.92	24.86	24.14	24.25	23.91	23.97	24.25
13. Humidity post RD (mean % RH)	41.5	34.6	31	32.5	34.3	34	34	34.8	34.7
14. Oxygen post RD (% O_2)	93.6	96.1	95.1	95.5	96.3	95.1	97.5	97.8	99.2
15. Time to recompress to ambient (mins.)	8	6	7.5	7	7	6	7	7	6

* 95% is considered as 100% in this case.

** Air trapped in chamber caused O_2 % to drop during ascent. Pressure was increased to 226 mm Hg after 28 minutes at 179 mm Hg.

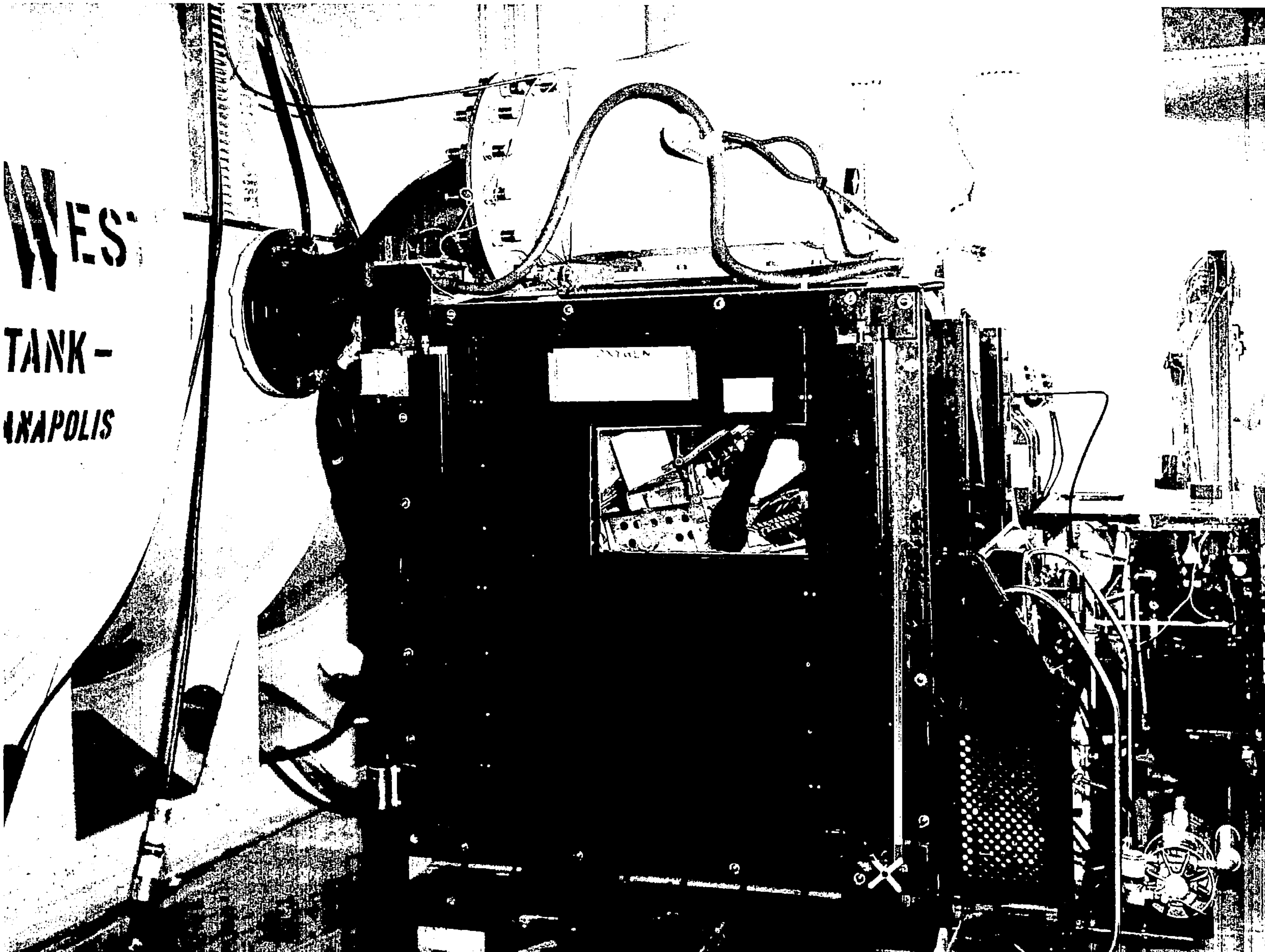

Figure 1. Decompression Chamber

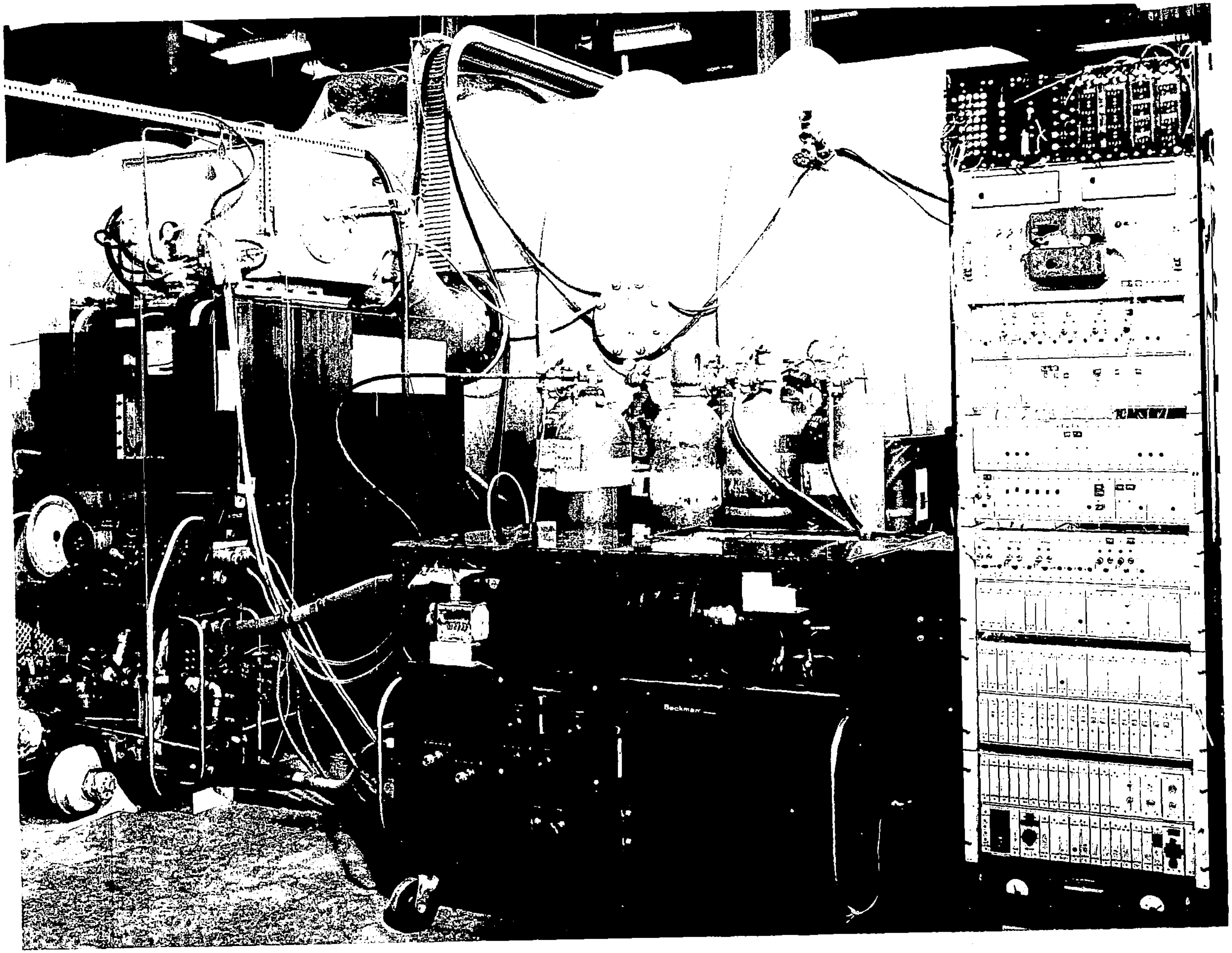

Figure 2. Life Support and Behavioral Equipment

Figure 3. Physiological Instrumentation

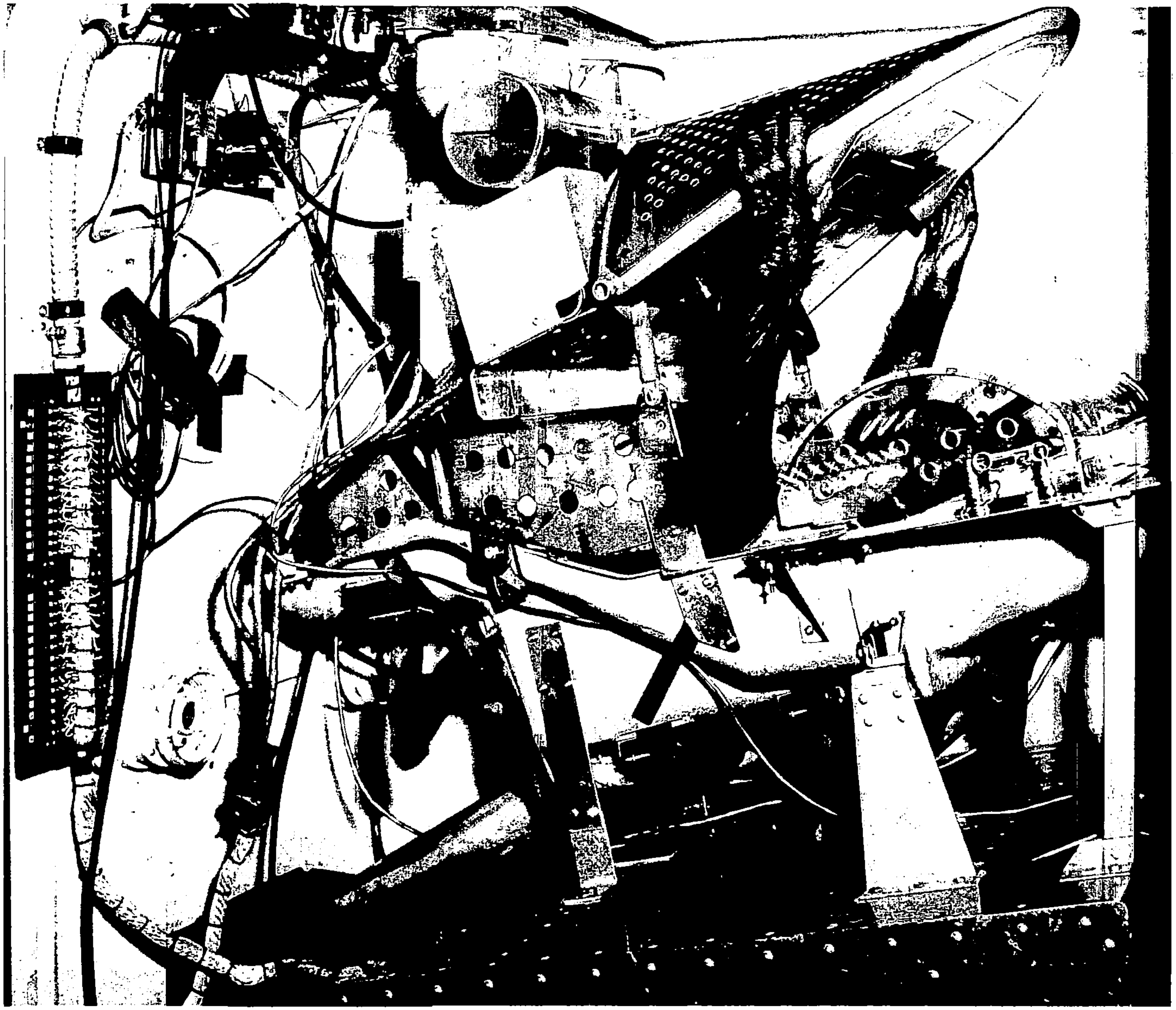

Figure 4. Subject Inserted in Chamber

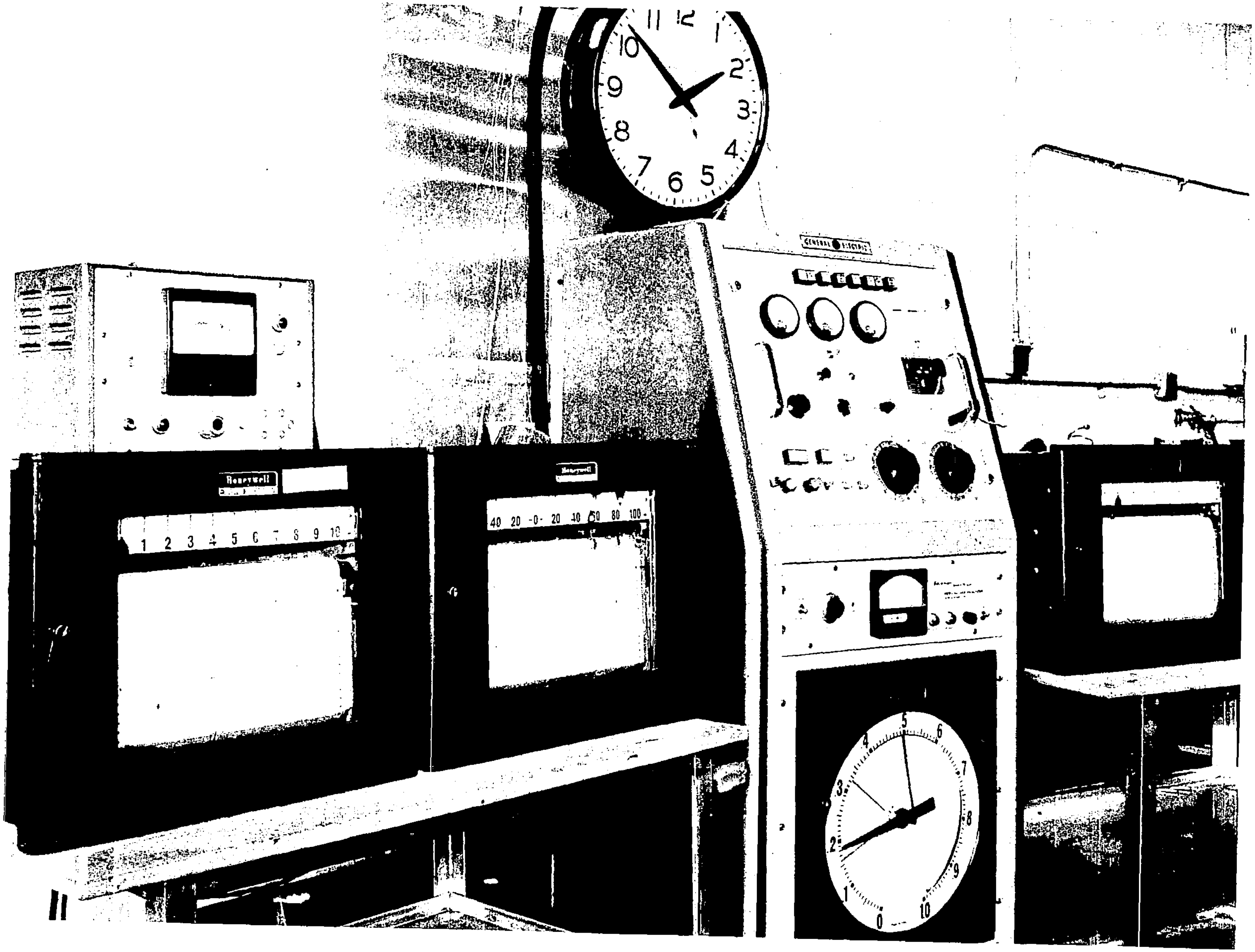

Figure 5. Environmental Instrumentation

NASA-Langley, 1965 CR-329