AD-777 604

PHYSIOLOGIC RESPONSES TO HIGH SUSTAINED + GZ ACCELERATION

Sidney D. Leverett, Jr., et al

School of Aerospace Medicine
Brooks Air Force Base, Texas

December 1973

DISTRIBUTED BY:

NTIS

National Technical Information Service
U. S. DEPARTMENT OF COMMERCE
5285 Port Royal Road, Springfield Va. 22151

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

AD-777604

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: SAM-TR-73-21

2. GOVT ACCESSION NO.

3. RECIPIENT'S CATALOG NUMBER

4. TITLE (and Subtitle): PHYSIOLOGIC RESPONSES TO HIGH SUSTAINED $+G_z$ ACCELERATION

5. TYPE OF REPORT & PERIOD COVERED: Final, 6 June 1972 - 6 July 1972

6. PERFORMING ORG. REPORT NUMBER

7. AUTHOR(s): S. D. Leverett, Jr., Ph.D.; R. R. Burton, D.V.M., Ph.D.; R. J. Crossley, Flt Lt, RAF; E. D. Michaelson, Lt Col, USAF, MC; S. J. Shubrooks, Jr., Maj, USAF, MC

8. CONTRACT OR GRANT NUMBER(s)

9. PERFORMING ORGANIZATION NAME AND ADDRESS: USAF School of Aerospace Medicine (VNB), Aerospace Medical Division (AFSC), Brooks Air Force Base, Texas 78235

10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: 7930-03-25

11. CONTROLLING OFFICE NAME AND ADDRESS: USAF School of Aerospace Medicine (VNB), Aerospace Medical Division (AFSC), Brooks Air Force Base, Texas 78235

12. REPORT DATE: December 1973

13. NUMBER OF PAGES: 73

14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office)

15. SECURITY CLASS. (of this report): Unclassified

15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

Reproduced by
NATIONAL TECHNICAL INFORMATION SERVICE
U S Department of Commerce
Springfield VA 22151

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

G-suits studied and evaluated; USAF CSU-12/P G-suit evaluation; RAF mini-G-suit evaluation; RAFIAM/USAF G-tolerance study; USAF/RAFIAM G-tolerance study; G-forces, high sustained; acceleration increased tolerance by G-suits; G-tolerance via G-suits; G-tolerance via M-1/PPB methods; M-1 respiratory straining maneuver,

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

This high sustained $+G_z$ acceleration study consisted of two parts: (part 1) to compare the respective G tolerances afforded subjects by the USAF CSU-12/P G-suit and the RAF prototype mini-G-suit; and (part 2) to compare the utility and effectiveness of the M-1 respiratory straining maneuver and positive pressure breathing (PPB) methods, respectively, to increase G tolerance for subjects wearing the USAF CSU-12/P G-suit. A total of 12 men (6 in each of the two parts of the study) participated in these experiments. Each subject was exposed to two

DD FORM 1 JAN 73 1473 EDITION OF 1 NOV 65 IS OBSOLETE

Unclassified

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

19. Key Words (continued)
G-stressed; PPB method of increasing G-tolerance; G-tolerance increased by PPB; medical effects of high G-forces; high-sustained-G medical effects.

20. Abstract (continued)
series of similar 60-sec exposures of 3, 6, and 8 G, followed by a 60-sec period at 1 G during which the subject performed a maximum M-1 or PPB straining maneuver. Observations were made concerning: electrocardiograms; direct systemic eye-level arterial pressures; direct esophageal and gastric pressures; direct central venous pressures; heart mass position movement during G; lower body superficial venous pressure (only in part 1 of the study); and arterial blood gases and pH (only in part 2). Except for two subjects, all tolerated 3 and 6 G for 60 sec. Only 10 of 24 exposures of 60-sec duration at 8 G were completed, and tolerance to 8 G was not improved by either the mini-suit or PPB. In the mini-G-suit—at 6 G, severe pain in the calf region of the leg occurred in 5 of 6 subjects; but at 3 or 8 G, this pain was usually absent. No correlation was found between pressure in an ankle vein and leg pain; but because of this severe pain, serious doubts arose regarding the acceptability of the mini-G-suit as an anti-G operational garment. In the case of PPB subjects, less fatigue was experienced and Pa_{O_2} was statistically increased, as compared with the M-1 subjects at the end of the 3 and 6 G exposures. The Pa_{C_2} and calculated arterial saturations were similar for both the M-1 and the PPB at 8 G. The conclusion was that the PPB method increased man's tolerance to high sustained G forces.

NOTICES

This joint study was initiated between the Royal Air Force Institute of Aviation Medicine (RAFIAM), Farnborough, England, and the USAF School of Aerospace Medicine (USAFSAM). The arrangements for, and details of, this research project extended from August 1971 to January 1973; the actual study was accomplished by the Biodynamics Branch, Environmental Sciences Division, USAFSAM, between 6 June and 6 July 1972, under task No. 79300325.

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

The voluntary informed consent of the subjects used in this research was obtained in accordance with AFR 80-33.

This report has been reviewed and cleared for open publication and/or public release by the appropriate Office of Information (OI) in accordance with AFR 190-17 and DODD 5230.9. There is no objection to unlimited distribution of this report to the public at large, or by DDC to the National Technical Information Service (NTIS).

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

SIDNEY D. LEVERETT, JR., Ph.D.
Project Scientist

EVAN R. GOLDMAN, Colonel, USAF, MC
Commander

ACCESSION for
NTIS White Section ☑
DDC Buff Section ☐
UNANNOUNCED ☐
JUSTIFICATION
BY
DISTRIBUTION/AVAILABILITY CODES
Dist. AVAIL. and/or SPECIAL
A

PREFACE

This joint study was approved by the U. S. Department of the Air Force and the Ministry of Defense of the United Kingdom of Great Britain. Dr. Sidney D. Leverett, Jr., of the USAF School of Aerospace Medicine (USAFSAM), was overall project director; Major Samuel J. Shubrooks, Jr., was USAFSAM project engineer; Flight Lieutenant Roger J. Crossley, of the RAF Institute of Aviation Medicine (RAFIAM), was project engineer; and Dr. Russell R. Burton was USAFSAM data analyst and principal author of the final report.

For their respective efforts on behalf of this research project, especial appreciation is expressed to Dr. Billy E. Welch (USAFSAM) and to Group Captain Peter Howard (RAFIAM).

The following individuals deserve special acknowledgment for their enthusiasm in serving as volunteer subjects during the intensive and potentially hazardous series of centrifuge runs:

CMSgt Harry F. Lund

TSgt William Matthews	Sgt Robert Sardá
Sgt James R. Burrell	Sgt Ronnie Zegers
Sgt Robert Davies	AlC Gene Davidson
Sgt Robert P. Monahan	AlC Robert Laymon
Sgt Larry Patterson	AlC Stephen Swann
Sgt Thomas Pfeiffer	AlC Robert Willette

Also, the authors express their appreciation to the following individuals for the high degree of technical expertise in resolving the many mechanical or electronic problems that arose during the course of such an extensive centrifuge study:

Mr. Hubert Tomerlin, Chief Centrifuge Operator	MSgt Douglas Krueger
Mr. Rufus V. Fairchild	MSgt Albert J. Marchetti
Mr. Bernard McMullin	MSgt Joe C. Nichols
	SSgt Earl V. Fraley
	SSgt Donald R. Power
	Sgt James M. Hanners

Finally, we would like to acknowledge the outstanding assistance of Dr. Phelps P. Crump, Mr. Darwell E. Stowe, Mr. Edward J. Engelken, Mr. Robert J. Fuchs, Mr. Paul A. Lozano, and Mr. Kennith W. Stevens of the Biometrics Division.

CONTENTS

Page

INTRODUCTION 7
MATERIALS AND METHODS 8
Description of Subjects 8
Training of Subjects 8
Order, Duration, and Levels of Acceleration 10
Dress for Subjects 10
Measurement of Acceleration 10
Characteristics of Anti-G Valve 10
Methods of Obtaining Electrocardiograms 12
Measurement of Arterial Pressure 12
Measurement of Esophageal and Gastric Pressures 12
Measurement of Central Venous Pressure 13
Estimations of Heart Mass Movement 14
Measurement of Lower Body Superficial Venous Pressure . . . 15
Measurement of Anti-G-suit Pressure 16
Measurement of Mask Pressure 16
Calibration of Transducers 16
Sampling of Arterial Blood 16
Regulator for PPB 16
List of Recorded Information 17
Questions on Subjective Impressions 17
RESULTS 17
Subjects' Time at Peak G 17
Effects on Heart Rate 19
Alterations in Heart Rhythm 21
Responses of Arterial Pressure 23
Preacceleration Resting Values 30
Preacceleration Peak 30
Deceleration Peak 31
Postacceleration Minimum 31
Postacceleration Peak 31
Time of Recovery 31
Final Resting Value 31
Determinations of Central Venous Pressure 31
Superficial Venous Pressures at Ankle Level 36
Changes in Arterial Blood Gases and pH 40
Change in Heart Size 49

Preceding page blank

(CONTENTS, Cont'd)

Page

Measurements of Esophageal and Gastric Pressures 52
Comparison of Data from Parts 1 and 2 of Study 52
DISCUSSION 52
Factors in Assessing G-tolerance 53
Responses of Heart Rate and Rhythm, and Arterial Pressure . 54
Preacceleration Response 54
Acceleration Response 54
Postacceleration Response 56
Responses of Superficial (Ankle) Venous Pressure 57
Description of Heart Volume Calculations 58
Causes of Arterial Oxygenation Responses at 8 G 61
Relation of Subject Fatigue to the M-1 and PPB. 62
REFERENCES 66
APPENDIX A 69
ABBREVIATIONS 71

ILLUSTRATIONS

Figure No.

1. The RAF prototype "mini-suit"--compared with a standard USAF multibladder G-suit, CSU-12/P. 9
2. A subject instrumented for measurement of esophageal and gastric pressures. 13
3. The tubes-balloons arrangement for measuring esophageal and gastric pressures. 14
4. Heart-rate responses to 60 sec of $+G_z$ with the subjects performing the M-1 at 1, 6, and 8 G. 20
5. Comparison of heart rates, at 1 G, for the same subjects while performing the M-1 and PPB, respectively. . 21
6. Recordings of the five types of cardiac arrhythmias observed during and after exposures to respective accelerations. 22
7. R-R intervals before and after a 6 G exposure, showing the presence of marked sinus arrhythmia during the recovery period. 23
8. A comparison of systemic arterial and esophageal pressure tracings obtained from three subjects who were exposed to +8 G_z while performing either M-1 or PPB activities. 26
9. Systolic and diastolic arterial pressures at eye-level are compared for the various G-levels before and after acceleration exposure. 30

(CONTENTS, Cont'd)

Page

10. The effects of time, at various levels of G, on ankle venous pressure are compared with calculated hydrostatic pressure. 37
11. Effect of exposure time at peak 8 G on Pa_{O_2} for both M-1 and PPB subjects. 44
12. Pa_{O_2} as a function of G, comparing the M-1 with PPB. 45
13. Arterial oxygen saturation as a function of exposure time to +8 G_z for both M-1 and PPB subjects. 47
14. Percent arterial oxygen saturation as a function of G for both M-1 and PPB groups. 48
15. The Pa_{O_2} data of Michaelson are compared with the M-1 data from Figure 12. 65
A-1. The arterial blood sampling device. 70

Table No.

1. The order of G intensity of 6 different subjects in parts 1 and 2, respectively. 11
2. Time spent at each level of acceleration by each subject in parts 1 and 2 of study. 18
3. Reasons for and incidence of stopping fourteen 8 G runs at less than 60 sec, and the mean time at peak G for the subjects affected. 19
4. Respective experimental conditions and incidence of PVCs and PACs in the 11 subjects exhibiting these abnormalities. 24
5. Systolic arterial blood pressure, pulse pressure, and esophageal pressure are compared during inspiration and expiration at 1 G, 6 G, 8 G in subjects performing the M-1 or PPB. 25
6. Mean values of eye-level arterial pressures and the time at which each occurred. 29
7. Central venous pressures, after correction for transducer/subject diaphragm distance, for subjects in CSU-12/P G-suits performing the M-1 at 1, 6, and 8 G. 33
8. Central venous pressures, after correction for transducer/diaphragm distance, for subjects in mini-suits performing the M-1 at 1, 6, and 8 G. 34
9. Central venous pressures, after correction for transducer/diaphragm distance, for subjects in CSU-12/P G-suits performing PPB at 1, 6, and 8 G. 35

(CONTENTS, Cont'd)

Page

10. The measured cumulated mean of ankle vencus pressure, for five subjects wearing the RAF mini-suit, grouped according to G-level exposure and M-1 maneuver. . . . 38
11. The occurrence of various amounts of leg pain in relation to G-levels and ankle venous pressure. 40
12. Arterial P_{O_2} levels of each subject at the end of a specific level of acceleration while performing the M-1 maneuver and PPB, respectively. 41
13. Arterial P_{CO_2} levels of each subject at the end of a specific level of acceleration while performing the M-1 maneuver and PPB, respectively. 42
14. Arterial pH levels of each subject at the end of a specific level of acceleration while performing the M-1 maneuver and PPB, respectively. 43
15. Percent arterial oxygen saturations, determined from data in Tables 12 and 13, are compared for the M-1 and PPB subjects at 3, 6, and 8 G. 46
16. Heart height as related to specific acceleration levels and conditions for each subject. 50
17. Statistical comparison of heart heights for control and specific acceleration groups. 51
18. Heart width as related to specific acceleration levels and conditions for each subject. 51
19. Heart shadow areas have been calculated, compared for each subject, and grouped according to G-level. 59
20. Statistical comparison of heart area between control and specific acceleration groups. 60
21. Percentage reduction in cardiac output, as determined from data of Lindberg et al. (20), compared with percentage reductions in heart volume as a function of G. 61

PHYSIOLOGIC RESPONSES TO HIGH SUSTAINED $+G_z$ ACCELERATION

INTRODUCTION

Future military aircraft will have performance characteristics which could expose the aircrew to high sustained $+G_z$--accelerations above 6 G--for periods of at least 2 minutes. Until recently, the studies of human tolerance to $+G_z$ acceleration were generally conducted on "relaxed" subjects for short durations (15 sec) only. As a result, little was learned about the effects of high sustained accelerations. Although the relaxed subject approach has provided useful evidence on the relative value of anti-G systems and physiologic responses to $+G_z$ acceleration, the experimental situation has little resembled inflight conditions. Under actual conditions, for example, a pilot is alert, tenses his muscles, and continually shifts his body in the cockpit in order to see his target.

Accordingly, interest has been focused on the responses to accelerations, up to a maximum of 9 G for 45 sec (23) of anti-G-suited subjects trained to perform the M-1 maneuver. This maneuver consists of forced expiration through a partially closed glottis, with tensing of the arm, leg, and abdominal musculature. The presumed action of the M-1 is to increase arterial pressure by increasing intrathoracic pressure, while the muscular tensing maintains a positive pressure gradient between the abdominal cavity and thorax sufficient to maintain venous return, thus assuring an adequate cardiac output. During the early studies of high sustained $+G_z$ acceleration (8 G/45 sec), arterial pressure measurements had confirmed that maintenance of vision was accompanied by arterial pressures which would ensure adequate cerebral perfusion (25).

Arterial pressure can also be increased by positive pressure breathing (PPB) and muscular tensing through a mechanism similar to the M-1 maneuver (7). The fall in venous return associated with continuous pressure breathing can be reduced or abolished when the subject wears an inflated anti-G suit and tenses his skeletal musculature. Since, under PPB conditions, the increase in intrathoracic pressure is produced by a machine and not by the subject's musculature, the same anti-G benefits could probably be obtained from PPB as from the M-1, and with less effort by the subject (as recently considered by Shubrooks, ref. 26).

Another interesting area in acceleration protection has been a continuous search for comfortable anti-G suits. Both the RAF and the USAF

have studied prototype G-suits without lower leg bladders (mini-suits) which generally were more comfortable and would also be cooler in the tropics. On relaxed subjects, the mini-suit was reported to give between 0.1 and 0.4 G less protection than a full-length suit (5, 18). However, a later study suggested no G-protective difference existed between the suits (6): Some relaxed centrifuge subjects reported severe lower leg pain above 4 G--a response not confirmed by pilots who wore this suit in flight (16, 24). The pain was thought to be evoked by high pressures in the leg veins (5).

Therefore, our joint study (17) not only investigated several physiologic effects of accelerations, up to 8 G for 1 min, but also compared the RAF mini-suit with a standard full-length suit--as well as PPB and M-1 as methods of increasing tolerance to high sustained $+G_z$ accelerations.

MATERIALS AND METHODS

Because of the large number of $+G_z$ exposures required and the limited number of vascular catheterization procedures which could safely be carried out on a subject, the study was divided into two parts. Different subjects were used in each part. In part 1, in which the RAF mini-anti-G suit was compared with the USAF CSU-12/P anti-G suit, all of the subjects performed the M-1 maneuver. The only difference between the two suits was that the RAF mini-suit had no lower leg (calf) bladders, whereas the CSU-12/P employed the usual number (5) of interconnecting bladders (Fig. 1). In part 2, in which the M-1 maneuver was compared with PPB, all of the subjects wore the USAF CSU-12/P anti-G suit.

Description of Subjects

Twelve informed volunteer military men were used (6 in each part of the study). They had recently passed a USAF Flying Physical Examination (Class II) and were experienced in riding the human centrifuge. The ages of the 6 subjects ranged: in part 1, from 18 to 32 years; and, in part 2, from 20 to 37 years.

Training of Subjects

To ensure the adequate performance of all subjects at high accelerations, each was instructed in the proper performance of the M-1 maneuver. Each subject then practiced this maneuver on the human centrifuge for 8 sessions, during which both the level and the duration of acceleration were progressively increased up to 8 G for a minimum of 30 sec. Moreover,

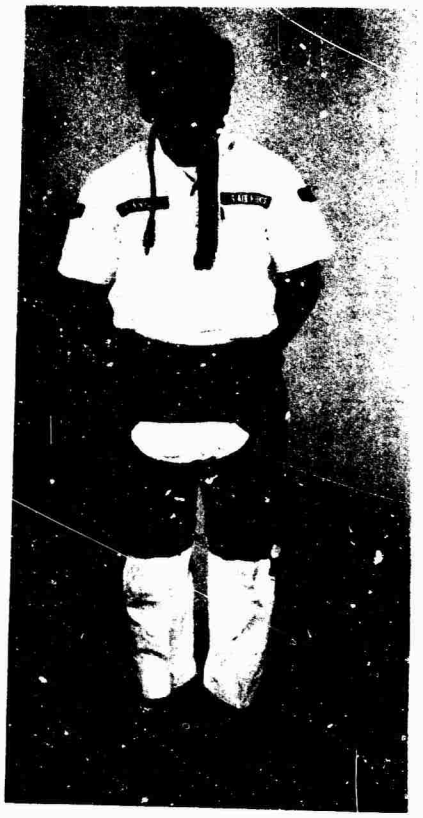


Photo A: RAF Mini-G-suit

Photo B: USAF CSU-12/P suit

Figure 1. The RAF prototype "mini-suit" (photo A)--compared with a standard USAF multibladder G-suit, CSU-12/P (photo B). (The major difference is the absence of calf bladders in the mini-G-suit.) The subject also was fitted with the RAF soft helmet and P/Q oxygen mask.

the subjects in part 2 were trained to pressure breathe (30 mm Hg) at 1 G, but did not have the opportunity to pressure breathe during acceleration before the actual experiment. In the experiment, they were instructed not only to use PPB but also to exert as much muscular effort as necessary during acceleration to maintain clear vision.

Order, Duration, and Levels of Acceleration

In parts 1 and 2 of this study, each subject had 2 experimental sessions on the centrifuge. Each session consisted of several rapid onset (1 G/sec) acceleration runs at various levels, as outlined in Table 1. Although the intent was for each run to have a 60-sec duration at each G-level, the following four criteria, (a) to (d), were established for stopping the run at an earlier time: (a) subject's choice--viz, at his own discretion and for any reason, the subject could stop the run by releasing the "dead man" brake switch; (b) vision--viz, subject's central vision was reduced to 50% dimming, as he judged by the central red light; (c) medical--viz, if the medical officer monitoring the run observed gross abnormalities in the ECG, or if the subject exhibited prolonged low eye-level arterial pressures (< 20 mm Hg); and (d) technical--viz, if a failure occurred in the recording system, so that the medical monitor was unable to guarantee the subject's safety.

Dress for Subjects

In addition to the appropriate anti-G suit, the subjects wore green surgical shirts and trousers, flying socks and boots, RAF soft helmet, and integrated harness to the ESCAPAC (McDonnell Douglas Co.) seat which was installed in the centrifuge gondola. Subjects were also fitted with the RAF P/Q oxygen mask (as required for PPB, in part 2).

Measurement of Acceleration

Acceleration was measured by a Pace Accelerometer (Model CA19R+20 G, mounted at the subject's heart level) which was calibrated by rotating the centrifuge at a rate calculated to produce 1, 2, 5, and 10 G.

Characteristics of Anti-G Valve

The anti-G suits were inflated with the RAF anti-G valve (VAG109-006). This valve had two pressure settings (4), the higher of which was always used in this study. The pressure supplied to the suit was 1.5 (n-2), in which n is the level of acceleration; viz, the valve began to supply pressure at 2.0 G. The anti-G valve was mounted so that the operating weight

TABLE 1. THE ORDER OF G INTENSITY (AND DURATION EXPOSURES) OF 6 DIFFERENT SUBJECTS IN PARTS 1 AND 2, RESPECTIVELY

PART 1						PART 2					
Mini-suit			CSU 12/P suit			PPB			M-1		
(Subjects)	(G)	(Sec)	(Subjects)	(G)	(Sec)	(Subjects)	(G)	(Sec)	(Subjects)	(G)	(Sec)
RD*, GD, JB*	3	15	RD, GD*, JB	3	15	RS, HL*, JW*	3	15	RS*, HL, JW	3	15
	3			6			6			3	
	6	60		3	60		3	60		6	60
	8			8			8			8	
	1			1			1			1	
WM, SS, LP*	3	15	WM*, SS*, LP	3	15	RL, TP*, RZ	3	15	RL*, TP, RZ*	3	15
	6			3			3			6	
	3	60		6	60		6	60		3	60
	8			8			8			8	
	1			1			1			1	

* Indicates first G exposures.

was at the same level as the accelerometer. Inlet-filtered air to the anti-G valve was supplied from a main compressor at 125 psi.

Methods of Obtaining Electrocardiograms

The ECG was simultaneously recorded and visually observed for changes in rate and rhythm from two sets of electrodes--sternal and bi-axillary. Standard electrodes (of the disposable adhesive type) were used throughout the study. Each subject's skin was abraded at the electrode sites in order to reduce skin-electrode resistance and thus minimize baseline shift and noise.

Measurement of Arterial Pressure

A Longdwel Teflon (18-gage) cannula was inserted into the radial artery (after the subject received local anesthesia). The cannula was connected, via a 3-way stopcock and sterile saline-filled polyethylene tubing, to a miniature Statham transducer (Model P-37). This transducer was sewn to the subject's soft helmet at the level of his eyes (Fig. 2). The other limb of the 3-way stopcock was used for flushing the catheters with heparinized saline.

Measurement of Esophageal and Gastric Pressures

The subject was required to swallow 2 empty latex balloons--the gastric balloon was 3 cm in diameter, whereas the esophageal balloon was 1 x 10 cm. So that both balloons could be swallowed at one time, they were first fitted to two 18-gage polyethylene tubes (Fig. 3). The subject's nose was sprayed with a mixture of 3.75% hexylcaine HCl and 0.25% phenylephrine HCl, and his throat with 5.0% hexylcaine HCl. The balloons were lubricated with water-soluble jelly and inserted into one nostril. By sipping water at the same time that they swallowed each balloon, the subjects experienced only limited difficulty. The balloons were finally positioned so that the lower point of the esophageal balloon was 45 cm from the external nares, at which cardiogenic artifacts are minimal (22). The distance between the lower point of the esophageal balloon and the upper point of the gastric balloon was 20 cm. The amount of air then inserted into the esophageal balloon was 3 ml; and into the gastric balloon, 10 ml. Previous tests had shown that when these amounts of air were used, the pressure in each balloon was zero relative to atmosphere. Gastric pressure was measured by a Statham P23DE pressure transducer (Fig. 2, point J). Esophageal pressure was measured by using a Sanborn 267BC pressure transducer (Fig. 2, point C).

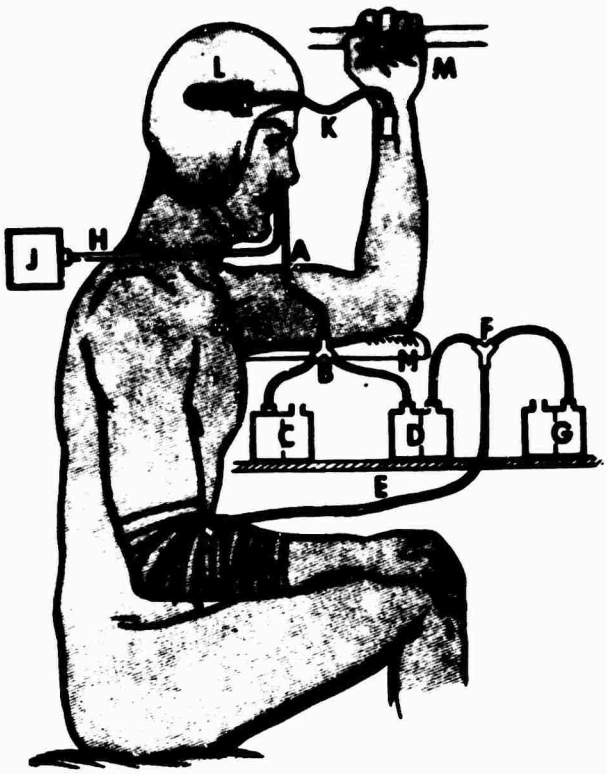


Figure 2. A subject instrumented for measurement of esophageal and gastric pressures. (A = tube from the esophageal balloon; B and F = 3-way connectors; C, D, G, and J = differential pressure transducers; E = central venous catheter; H = tube from gastric balloon; K = arterial catheter; L = miniature Statham pressure transducer; M = rigid bar; and N = arm rest.)

Measurement of Central Venous Pressure

Central venous pressure was measured by means of a 24-in. polyethylene intracatheter inserted via the median cubital vein. The position of the catheter tip was located by fluoroscopy at a point 1 to 2 in. above the right atrium in the superior vena cava. The catheter was connected via a 3-way stopcock and a sterile polyethylene cannula to a 3-way connector (Fig. 2, point F). The other limb of the stopcock was used to flush the catheter with heparinized saline. One limb of the 3-way connector, F, was connected to a Statham P23DE pressure transducer (Fig. 2, point G). The other limb was connected to the positive side of the diaphragm of a

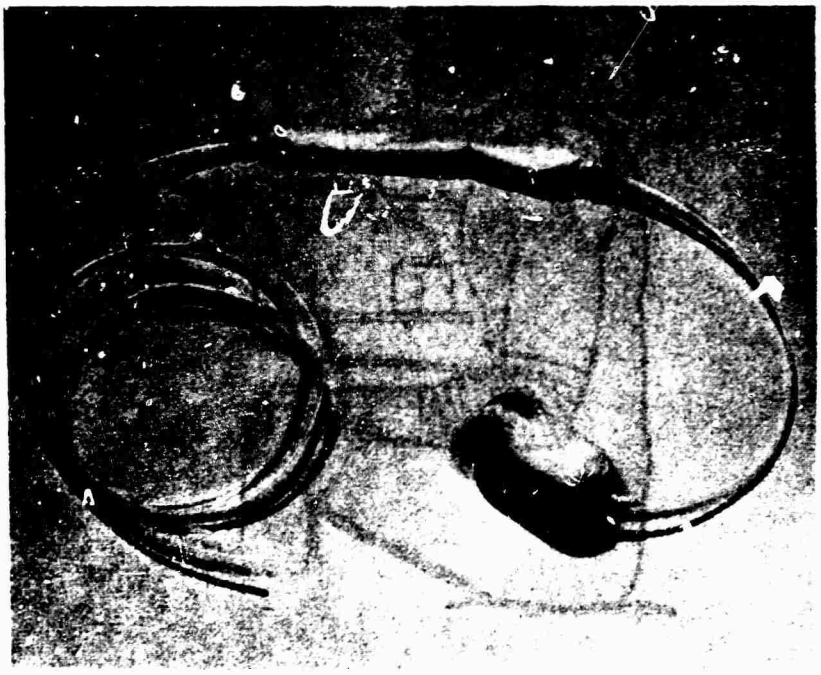


Figure 3. The tubes-balloons arrangement for measuring esophageal and gastric pressures. (Two tubes enter the elongated esophageal balloon; but only one of the tubes passes through this balloon and leads into the gastric balloon, which is approximately 3 cm in diameter.)

Sanborn 267BC differential pressure transducer (Fig. 2, point D). A similar 3-way connector also was used with the polyethylene catheter connected to the esophageal balloon (Fig. 2, point B) so that the third limb of this stopcock was connected to the negative side of the diaphragm of transducer D. In this way, transducer G measured central venous pressure which is a function of intrathoracic pressure and blood pressure. Consequently, in an attempt to eliminate the intrathoracic pressure effect and obtain the net blood pressure, transducer D was used to measure the pressure differential between transducers C (intrathoracic pressure) and G (central venous pressure).

Estimations of Heart Mass Movement

Because heart position apparently is altered during $+G_z$ acceleration (28), central venous pressure measurements can be affected during

acceleration due to the change in the height of the column of fluid between the heart and the transducer. To correct for these possible pressure artifacts, an anterioposterior (AP) chest radiograph was taken during each run with transducer D positioned to serve as a point of reference on the x-ray film. The radiographic film plate was inserted into the back of the seat. A Picker medical-type x-ray unit (Model KM2000R) was installed in the centrifuge and focused so that a readable (AP) picture of the complete chest was obtainable. A control exposure (1 G, resting) was taken with the resting subject strapped into the seat. Pictures were taken after 20 sec at peak G during each 60-sec run, including one picture at 1 G, with the subject performing the M-1 or PPB. Radiographic intensities standard for chest exposures were used with appropriate modifications at high G, where the lower lung region increased in density. The focal point of the tube was set between 41 in. and 43 in.

Lead markers were placed on each subject's back, and the distances between them were measured. These markers, together with the size of the ECG electrodes and the buckles of the harness which was strapped firmly to the subject, made it possible to estimate the magnification of the x-ray. Hence the absolute distance from the transducer to a specific point on the heart could be calculated. Since taking more than 9 x-rays on each subject was considered unsafe, radiographic exposures were not made during the second session of the 3-G 60-sec run. Also, 2 subjects in each part of the study were not x-rayed at all; for they had participated recently in another study using radiography, and exposing them to additional radiation was considered unsafe.

Measurement of Lower Body Superficial Venous Pressure

In part 1 of the study, lower body venous pressure was measured at the ankle, using a superficial vein of subjects who were wearing the mini-suit. A polyethylene catheter was inserted into the most accessible superficial vein of the left ankle. This catheter was connected, by means of a 3-way stopcock and sterile polyethylene tubing, to a Statham P-37 miniature pressure transducer. The third limb of the stopcock was used for flushing the catheter with heparinized saline. The transducer was mounted on a fixed bar at the level of the subject's ankle. Mild external counterpressure was supplied by an elastic bandage, applied at the site of catheter insertion into the vein to avoid extravascular hemorrhage. The catheter tip was always above the counterpressure site.

Measurement of Anti-G-Suit Pressure

Anti-G-suit pressure was measured using a Giannini pressure transducer (type 451212-42) connected, by means of a T-tube, to the hose joining the anti-G suit to the anti-G valve.

Measurement of Mask Pressure

Mask pressure was measured in part 2 of the study while the subjects were using PPB--via polyethylene tubing connecting the mask cavity to a Statham P-23DE pressure transducer.

Calibration of Transducers

All of the transducers were calibrated, before and after each session, by means of a mercury manometer. Each transducer was mounted so that its diaphragm was parallel to the G_z vector. In addition, the centrifuge was run on separate days at 8 G, after the transducers were calibrated, to ensure that no shift occurred in the recorded zero line due to acceleration.

Sampling of Arterial Blood

In part 2 of the study, arterial blood samples were obtained by using an automatic blood-sampling device (described in appendix A). This device was connected to the third limb of the 3-way stopcock connected to the cannula in the radial artery, and was operated remotely with a switch located in the control room. Samples were taken after exposure, at the beginning of deceleration. The P_{O_2}, P_{CO_2}, and pH of the arterial blood were determined immediately by using a pH-blood-gas analyzer (Instrumentation Laboratories, IL Model 113-S-2).

Regulator for PPB

In part 2 of the study, when the subjects were required to use PPB, the mask was connected to an RAF MR 11 oxygen regulator. This regulator supplied a safety pressure of 3 - 5 mm Hg and a positive pressure of 28 - 30 mm Hg. The regulator was modified to supply pressure when exposed to more than 2 G. Filtered inlet air to the oxygen regulator was supplied from a compressor at 65 psi pressure. The regulator was mounted at the level of the accelerometer and anti-G valve.

List of Recorded Information

The following information was recorded on a Brush MK-200 8-channel recorder: acceleration; one ECG; arterial pressure; both absolute and differential central venous pressures; esophageal pressure; gastric pressure; and either mask pressure (part 2) or superficial ankle venous pressure (part 1), together with a time mark and an analog time code. The other ECG, suit pressure, and acceleration were recorded on a Brush 440 4-channel recorder.

All data were simultaneously recorded on magnetic tape using a Sangamo 4700 14-channel tape recorder.

Questions on Subjective Impressions

In part 1 of the study, each subject was questioned about the occurrence of lower leg pain after each exposure to acceleration. In part 2 of the study, each subject was asked to compare PPB with the M-1 maneuver, particularly with respect to the degree of fatigue experienced.

RESULTS

Subjects' Time at Peak G

In Table 2 is shown the time spent at each level of acceleration by the respective subjects (identified by initials) in both parts of the study. All subjects completed 60 sec at 3 G and 6 G, except for two subjects in part 2. Of these exceptions ("RS" and "JW"), subject RS experienced technical difficulties at 6 G, because the inlet hose to his mask became occluded; and subject JW completed only 39 sec at 6 G, apparently because he had problems coordinating his muscular straining with PPB.

At 8 G, however, several subjects failed to complete 60 sec at peak acceleration. The longer periods at peak G spent by subjects who wore the mini-suit as compared with the full suit (part 2)--and those who used PPB as compared with the M-1--are not statistically significant according to Student's paired t-test. Also, no significant differences (analysis of variance testing) existed between the G tolerances obtained during part 1 and those during part 2. Of 24 sessions at 8 G, 13 subjects (approximately 50%) tolerated 50 sec or more exposure time. The reasons for stopping the 8 G runs are given in Table 3. It is of interest that subject JW, who had trouble with PPB at 6 G, also had the shortest stay at 8 G (viz, 8 sec, in contrast to 32 sec at 8 G--the maximum achieved by the "second lowest" ranking subjects).

TABLE 2. TIME SPENT AT EACH LEVEL OF ACCELERATION BY EACH SUBJECT (IN RESPECTIVE TEST G-SUITS AND MANEUVERS) IN PARTS 1 AND 2 OF STUDY

Subjects	Time (in sec) per acceleration level per subject, in test conditions					
	3 G		6 G		8 G*	
(Part 1)	Mini	CSU-12/P	Mini	CSU-12/P	Mini	CSU-12/P
RD	60	60	60	60	60	60
GD	60	60	60	60	48	60
JB	60	60	60	60	60	60
WM	60	60	60	60	39	18
SS	60	60	60	60	60	8
LP	60	60	60	60	60	46
Mean:	60	60	60	60	54.5	42.0
SD:	--	--	--	--	8.98	23.3
(Part 2)†	M-1	PPB	M-1	PPB	M-1	PPB
RS	60	60	33‡	60	50	43
HL	60	60	60	60	38	56
JW	60	60	60	39	20	8
RL	60	60	60	60	14	32
TP	60	60	60	60	53	60
RZ	60	60	60	60	60	60
Mean:	60	60	55.5	56.5	39.2	43.2
SD:	--	--	11.0	8.57	18.7	20.5

* Statistical analyses (by analysis of variance or paired t-tests, as appropriate) showed no significant difference in tolerances at 8 G for the groups tested.

† All subjects wore CSU-12/P G-suits.

‡ Inlet hose (to mask) occluded and prevented breathing.

SD = standard deviation.

TABLE 3. REASONS FOR AND INCIDENCE OF STOPPING FOURTEEN 8 G RUNS AT LESS THAN 60 SEC, AND THE MEAN TIME AT PEAK G FOR THE SUBJECTS AFFECTED

Reasons for stopping runs	No. of subjects affected	Mean time at 8 G (sec)
Subject choice (fatigue)	2	44
Visual loss	8	28
Medical (low arterial pressure)	0	--
Medical (ECG abnormalities)	0	--
Medical (head drooping toward chest)	3	41
Technical	1	39

Note: A subject's head drooping toward his chest usually is an indication of his tiring; and, if this factor (noted in 3 subjects) is added to fatigue (the reason for which 2 subjects voluntarily stopped the run), then the mean G duration tolerance for this group of exhausted subjects is 42 sec. Visual loss, however, usually occurred earlier in the run--at a mean time of 28 sec (as shown above).

Effects on Heart Rate

No significant differences for heart-rate values at the same G-level were found for M-1 subjects wearing either the mini-suit or CSU-12/P. Consequently, heart-rate data were combined for the 3 groups where the M-1 was performed, and were compared for the G-levels before, during, and after acceleration exposure (Fig. 4).

Approximately 20 sec before acceleration exposure, heart rate increased rapidly, especially for the 6 G and 8 G runs. A similar response was noted by Parkhurst et al. (23) and attributed to excitement in anticipation of the immediate G exposure.

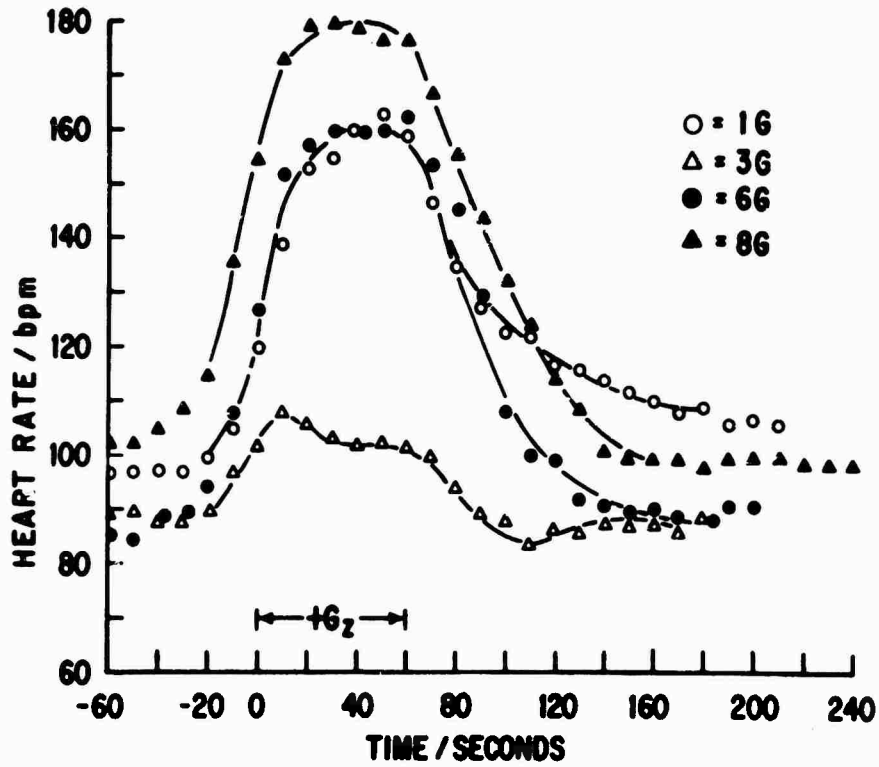


Figure 4. Heart-rate responses to 60 sec of $+G_z$ with the subjects performing the M-1 at 1, 6, and 8 G. (The M-1 was not performed during the 3 G exposure.)

Performing the M-1 at 1 G caused a statistically significant increase in heart rate above resting levels. This rise was similar to that at 6 G, where the subjects also were required to perform an M-1 maneuver. However, the maximum M-1 effort which the subjects were asked to perform at 1 G usually exceeded the effort necessary to maintain vision at 6 G. Although an increase in heart rate was also found in subjects at 3 G who were not performing the M-1, this rise was less statistically significant than that noted in subjects at 1 G who were performing the M-1. Heart rates at 6 and 8 G therefore appear to have two bases: M-1 effort; and acceleration per se.

The time for heart rate to recover appears to depend on both the total exposure to G during the entire day's session and the total effort in performing the M-1. Recovery periods for 3 G and 6 G are the same, although the 3 G recovery included a period of bradycardia which was not apparent following 6 G. The 8 G run, which caused the most severe

physiologic stress, had a recovery duration longer than that for the lower G runs; however, after the M-1 maneuver at 1 G (which was always the last procedure in a day's session), heart-rate recovery was considerably prolonged. The recovery after PPB at 1 G was significantly more rapid than when the same subjects performed the M-1 (Fig. 5). Subjectively, everyone felt more fatigued at the end of the M-1 session than after PPB, thus suggesting that the duration of heart-rate recovery might be an indicator of fatigue.

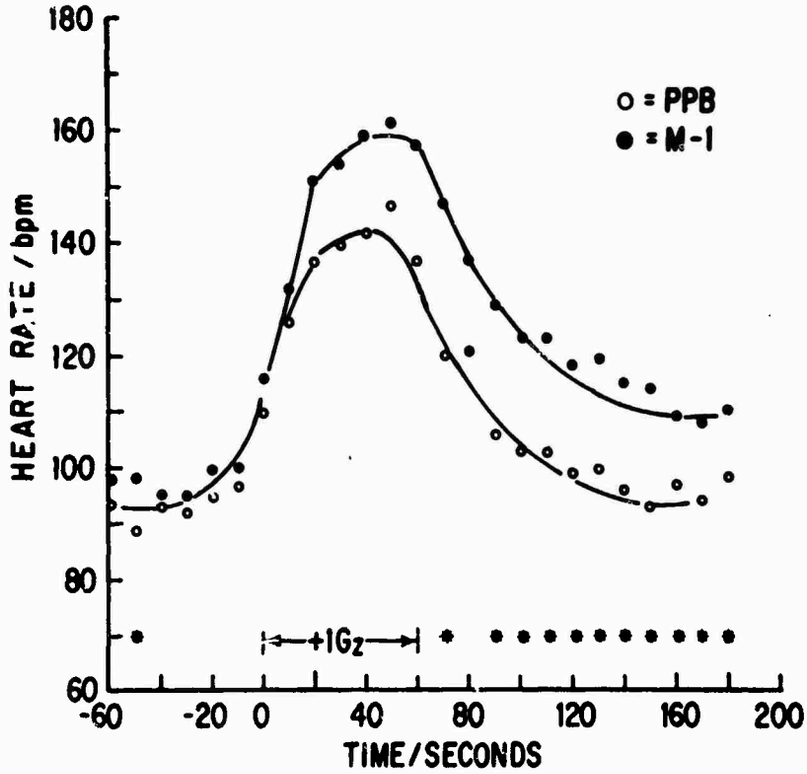


Figure 5. Comparison of heart rates, at 1 G, for the same subjects (part 2) while performing the M-1 and PPB, respectively. (Asterisks indicate significant differences, $P < .05$, between groups.)

Alterations in Heart Rhythm

Five types of disturbance of normal cardiac rhythm were observed (examples in Fig. 6); and, of these, the sino-atrial block and the junctional

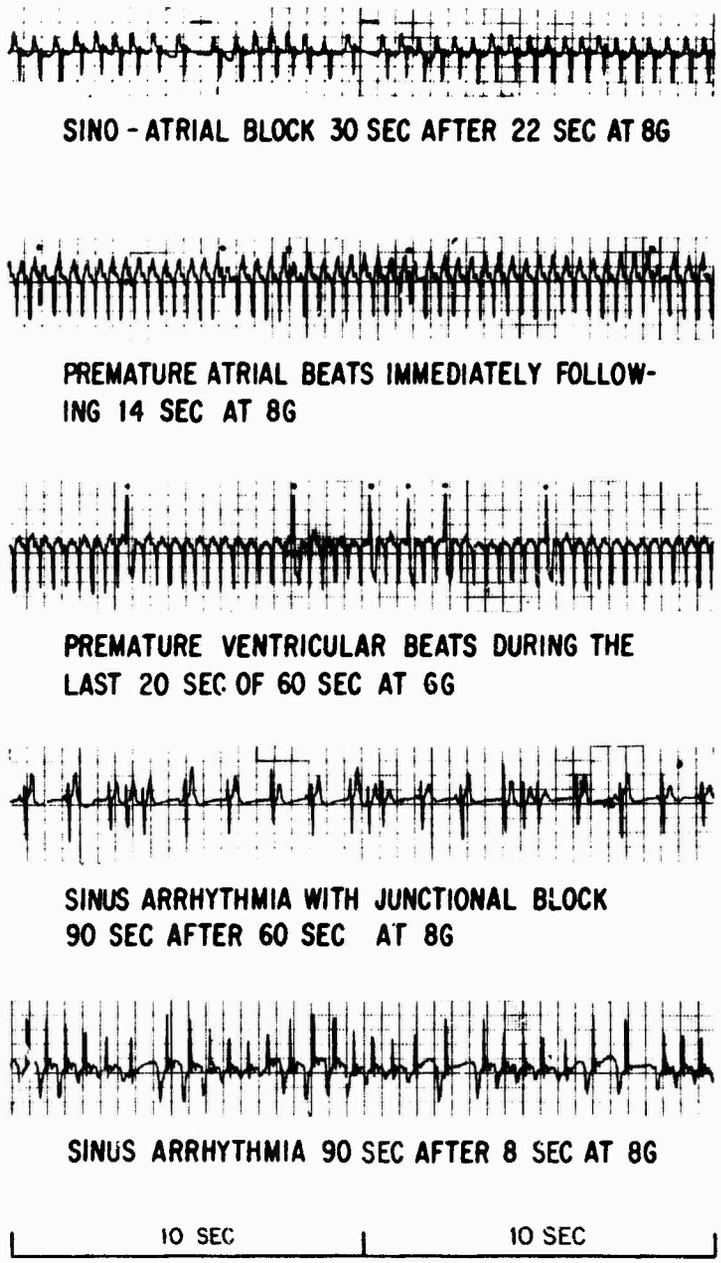


Figure 6. Recordings (sternal electrodes) of the five types of cardiac arrhythmias observed during and after exposures to respective acceleratic s. (Scale is shown beneath figure.)

block occurred only once. Most subjects exhibited a normal respiratory sinus arrhythmia during the resting periods. During the recovery period, a characteristic R-R interval pattern emerged (Fig. 7).

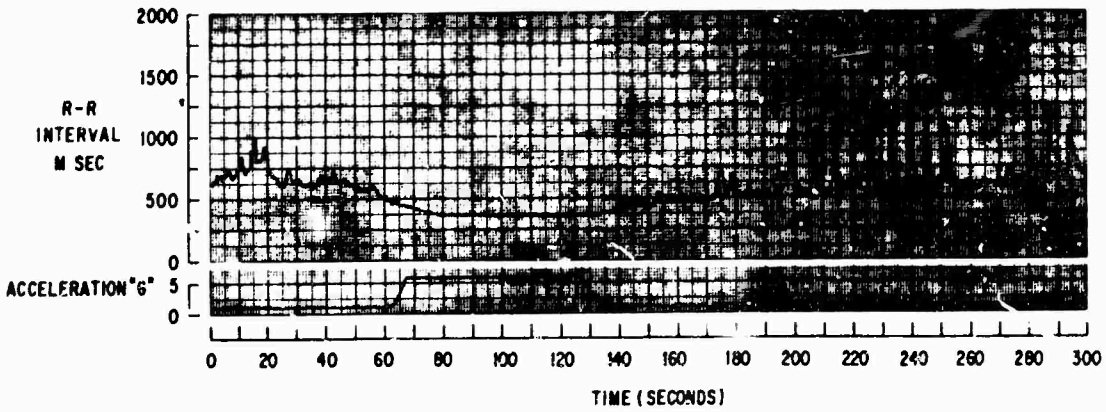


Figure 7. R-R intervals before and after a 6 G exposure, showing the presence of marked sinus arrhythmia during the recovery period.

Shown in Table 4 is the incidence of premature ventricular contractions (PVCs) and of premature atrial contractions (PACs). Apart from subjects "HL" (both sessions) and "SS" (one session), the remaining 10 subjects did not exhibit many unusual or abnormal ECG rhythms. Only one subject, "GD," exhibited any abnormal beats at 3 G, whereas 3 persons (including GD) had these beats at 1 G while doing the M-1 or PPB. Of the 12 subjects, however, 11 exhibited abnormal beats on at least one occasion; but these beats did not occur at any particular time in the run and (except in subject HL) were not paired. The unpredictability of these ECG abnormalities makes them difficult to correlate with physiologic events.

Responses of Arterial Pressure

During the M-1 maneuver or PPB, fluctuations in esophageal and arterial pressures were observed and are shown in Figure 8 for 3 subjects during 8 G exposure. Changes in arterial pressure are directly correlated with changes in esophageal pressure (viz, both in frequency and amplitude). Esophageal and arterial pressures obtained during 1, 6, and 8 G are compared for the M-1 and PPB in Table 5. Fluctuations in pressures corresponding with inhalation and exhalation resulted in a cyclic phenomenon

TABLE 4. RESPECTIVE EXPERIMENTAL CONDITIONS AND INCIDENCE OF PVCs AND PACs IN THE 11 SUBJECTS EXHIBITING THESE ABNORMALITIES

Subjects	No. of G's	Condition (activity & G-suit)*	Premature contractions (No.)	(Type)
RD	6	B	1	PVC
GD	1	B	1	PAC
	3	B	1	PAC
	6	B	1	PAC
	6	A	1	PAC
JB	8	B	2	PAC
	8	A	3	PVC
			2	PAC
WM	6	B	2	PAC
	8	B	2	PAC
SS	6	B	22	PVC
LP	6	B	1	PVC
RS	6	B	3	PVC
	6	C	1	PVC
	8	C	7	PVC
HL	8	E	29	PVC†
	6	C	30	PAC†
RL	[illegible]	B	1	PAC
	6	B	9	PAC
	8	B	6	PAC
	8	C	2	PVC
TP	6	B	4	PVC
RZ	1	C	3	PVC
	8	C	2	PVC

* A = M-1/Mini; B = M-1/CSU-12/P; C = PPB/CSU-12/P.
† Some paired.

TABLE 5. SYSTOLIC ARTERIAL BLOOD PRESSURE, PULSE PRESSURE, AND ESOPHAGEAL PRESSURE ARE COMPARED DURING INSPIRATION (MINIMUM) AND EXPIRATION (MAXIMUM) AT 1 G, 6 G, 8 G IN SUBJECTS PERFORMING THE M-1 OR PPB

	M-1			PPB		
	1 G	6 G	8 G	1 G	6 G	8 G
	$\bar{X}\pm SE$ (n)	$\bar{X}\pm SE$ (n)	$\bar{X}\pm SE$ (n)	$\bar{X}\pm SE$ (n)	$\bar{X}\pm SE$ (n)	$\bar{X}\pm SE$ (n)
SAP						
Insp. (min.)	101±10.1 (6)	58.3±13.0(6)	18.4±12.3 (6)	106±7.57 (5)	52.7±14.3(6)	21.9±19.8 (6)
Exp. (max.)	166±8.21 (6)	135±13.9 (6)	94.8±17.9 (6)	160±11.5 (5)	111±9.01 (6)	90.4±22.1 (6)
PP						
Insp. (min.)	35.8±5.43(6)	37.8±7.07(6)	30.6±2.06 (6)	33.9±5.70(5)	31.2±9.42(6)	24.9±10.3 (6)
Exp. (max.)	59.0±9.15(6)	83.4±7.57(6)	72.5±10.4 (6)	68.2±3.51(5)	76.3±3.75(6)	72.9±13.9 (6)
EP						
Insp. (min.)	-8.98±5.40(6)	*-21.3±1.90(6)	†-25.6±3.82 (6)	6.17±2.21(6)	*7.03±1.78(6)	†4.54±1.86 (6)
Exp. (max.)	‡61.6±3.45(6)	48.5±8.49(6)	70.9±5.10 (6)	‡33.2±4.30(6)	37.8±5.18(6)	58.4±10.1 (6)
Differential**	†70.0±8.19(6)	†68.6±10.2(6)	†96.4±6.04 (6)	†27.1±5.88(6)	†30.8±6.96(6)	†59.2±11.3 (6)

* $P < .001$
† $P < .05$
‡ $P < .01$
** Differential EP = Max. EP - Min. EP.
EP = esophageal pressure.

(n) = number of subjects per group.
PP = pulse pressure.
SAP = systolic arterial blood pressure.
SE = standard error.
$\bar{X}$ = mean.

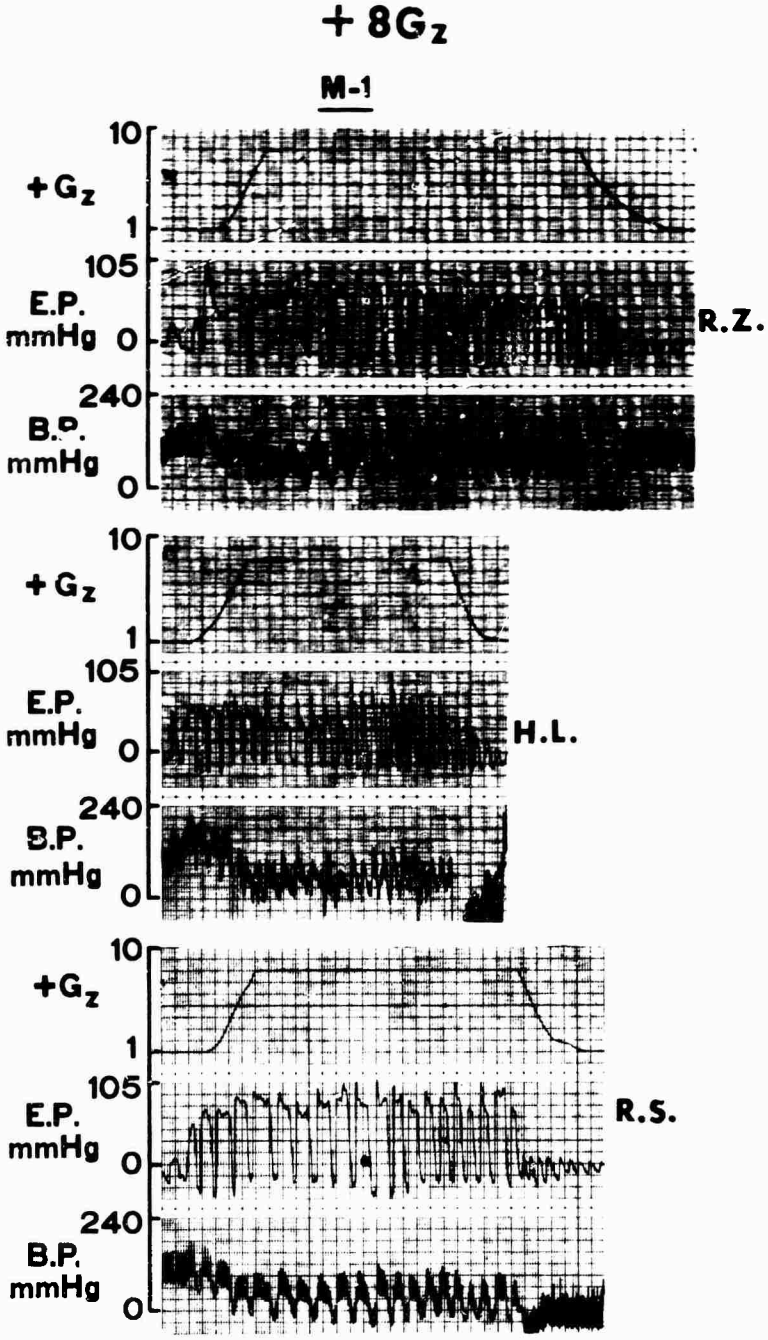
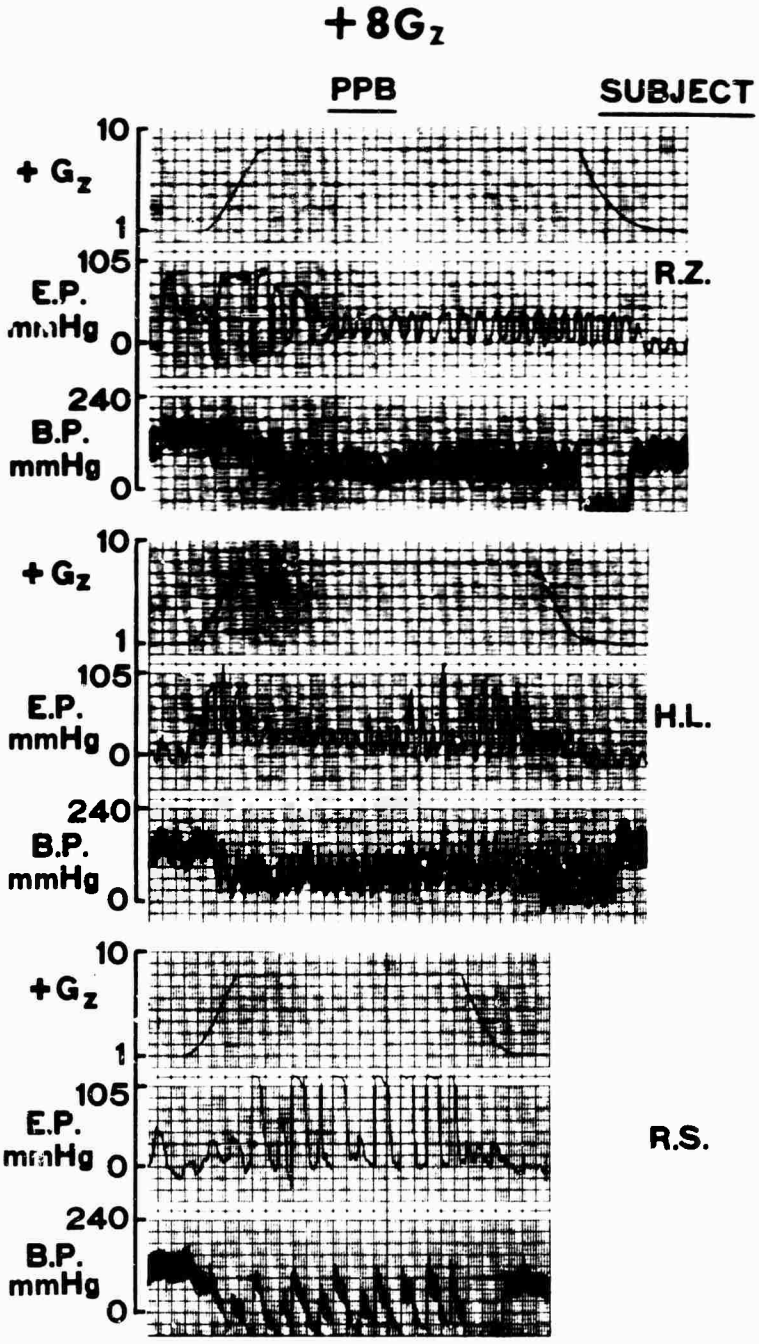


Figure 8. A comparison of systemic arterial and esophageal pressure tracings obtained from three subjects who were exposed to +8 G_z while performing either the M-1 or PPB activities.

(Cont'd on facing page--)



(Fig. 8--Cont'd from preceding page)

R.Z., H.L., and R.S. = three subjects for the M-1 and PPB tests;
E.P. = esophageal pressure; and
B.P. = blood pressure.

which exhibited minimum and maximum values for systolic arterial pressures, pulse pressures, and esophageal pressures--the minimum value occurring during the rapid inspiratory phase of respiration, and the maximum value corresponding with the prolonged period of forced expiration.

Systolic arterial blood pressure and pulse pressure were not affected differently by the M-1 or PPB. Systolic pressures were lower during inspiration than expiration, and each was reduced as the G-level increased. Mean systolic pressures at 8 G during inspiration were approximately 20 mm Hg, the arterial level at which central light loss (CLL) occurs (15).

Regarding individuals, it was not uncommon during 8 G runs for the arterial pressure to be zero during the inspiratory phase of the M-1, yet the subjects reported no loss of vision. However, if the subject's rate of inspiration was sufficiently slowed, and the lower pressures were maintained for longer periods--then subjects reported visual loss. Of course, peripheral light loss (PLL) occurred when eye-level pressure at all phases of the respiratory cycle dropped to near 30 mm Hg. The mean systolic blood pressure during expiration at 8 G was approximately 90 mm Hg, which is more than sufficient to maintain adequate cerebral and retinal blood circulation.

Pulse pressures were lower during inspiration than expiration yet appeared to be unaffected by the G-level, suggesting that adequate cardiac output is maintained during the entire 8 G exposure duration.

Esophageal pressures were affected by PPB quite differently than by the M-1. Generally speaking, during inspiration, esophageal pressures were lower in those persons doing the M-1 than in those doing the PPB. Conversely, during expiration, esophageal pressures were higher in subjects doing the M-1 than in those doing the PPB--resulting in differential esophageal pressures approximately twice as large during M-1 as during PPB.

Comparison of individual pressure recordings revealed characteristic patterns which were specific for a given G-level, but independent of subjects or conditions (i.e., M-1 or PPB). These patterns are illustrated in Figure 9 and comprise: a rise in pressure preceding the onset of acceleration; a transient peak during deceleration (only observed in part 1, since pressure recording stopped during collection of blood in part 2); a second, more prolonged peak after the centrifuge had come to rest; and eventual recovery to control levels. Systolic and diastolic levels were identified by computer, and mean values for relevant phases of the runs are given in Table 6 (data from M-1 and PPB runs being pooled, since no statistically significant difference was demonstrated).

TABLE 6. MEAN VALUES OF EYE-LEVEL ARTERIAL PRESSURES AND THE TIME AT WHICH EACH OCCURRED (FOR ALL SUBJECTS, IN PARTS 1 AND 2 OF STUDY)

G-levels	Preacceleration resting level (Pressure)		Preacceleration peak (Pressure)		Deceleration peak			Postacceleration minimum			Postacceleration peak			Final resting level		
					(Time)	(Pressure)		(Time)	(Pressure)		(Time)	(Pressure)		(Time)	(Pressure)	
	S	D	S	D	(sec)	S	D	(sec)	S	D	(sec)	S	D	(sec)	S	D
								(PART 1: Mini-suit and CSU-12/P)								
1	110	55	125	66	-	-	-	10.1	92	43	46.6	135	64	83.0	109	52
3	128	56	140	63	-	-	-	-	-	-	25.7	145	62	82.9	121	56
6	130	55	149	70	8.2	124	60	16.1	105	43	56.0	162	67	131.4	127	56
8	132	58	155	89	7.2	156	80	20.7	113	51	66.9	174	74	166.0	129	57
	1<3,6,8		1<3,6,8	8>1,3,6							3<1,6,8	1,3<6,8	8>6>3,1	8,6>3,1		
P<	.001	NS	.01	.001							.01	.01	.01	.001	NA	NA
								(PART 2: PPB and M-1)								
1	105	51	117	57	*			*			58.0	133	60	90.0	109	51
3	124	53	133	60	*			*			36.7	139	62	73.4	117	52
6	130	55	144	77	*			*			66.3	176	74	153.5	135	54
8	127	57	155	92	*			*			68.6	182	81	184.5	132	57
	1<3,6,8		1<3,6,8	8>6,3,1							3<1,6,8	1,3<6,8	8>6>3,1	8>6>3<1		
P<	.001	NS	.01	.01							.01	.01	.01	.05	NA	NA

* Time of blood withdrawal.
S = systolic; D = diastolic; sec = second(s); NS = not statistically significant (P > .05); NA = not appropriate.

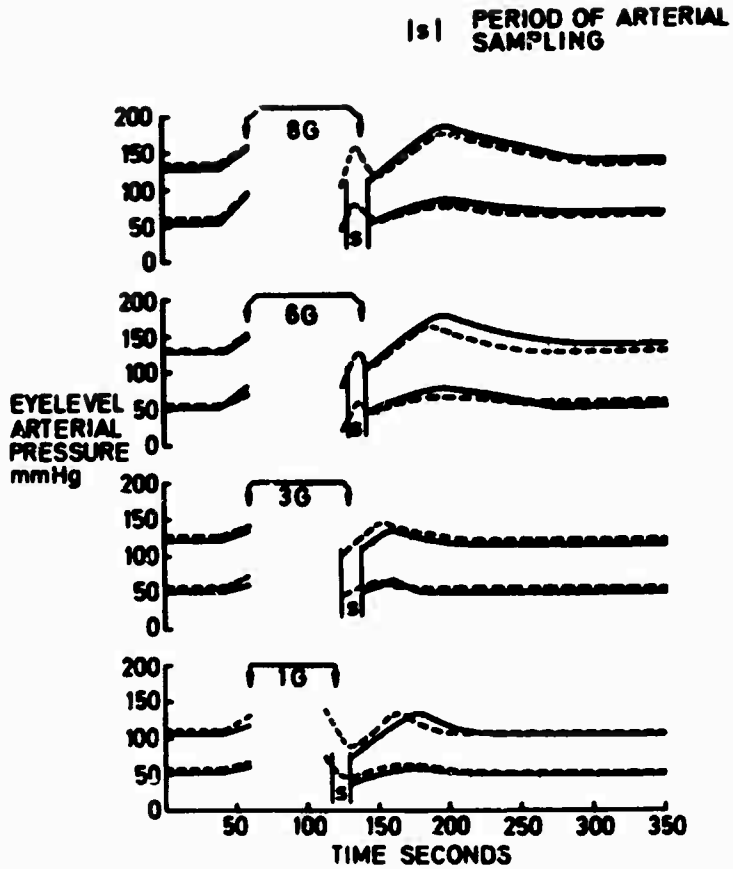


Figure 9. Systolic and diastolic arterial pressures at eye-level are compared for the various G-levels before and after acceleration exposure. (The values for both groups, in part 1, are shown by dashed lines--in part 2, by solid lines.)

Preacceleration resting values. No significant difference existed between the resting diastolic pressures for any of the preacceleration groups. Systolic pressures, however, for the pre-1 G were significantly ($P < .001$) lower than at 3, 6, and 8 G for both studies.

Preacceleration peak. An arterial pressure rise was observed in the 20 sec before acceleration, with the peak value occurring just

before the start of the run. This rise probably was an anticipatory response to the impending G exposure and was closely correlated to a pre-G heart-rate increase (Fig. 4).

Deceleration peak. This pressure overshoot, as identified only in part 1 (arterial samples were taken during this time period in part 2 of the study), is not significantly different from resting blood-pressure levels and did not occur at 3 G. Presumably, therefore, this deceleration peak results from the M-1, because the subjects at 6 and 8 G continued to perform this maneuver during deceleration.

Postacceleration minimum. Postacceleration pressure minimum also occurred during blood sampling in part 2, and consequently was recorded in part 1 only. This response did not occur at 3 G, and was significantly ($P < .001$) lower than that at the resting level. The pressure minimum occurred later after high-G runs than after 1 G runs. The data suggest, therefore, that this response is a function of both the M-1 and the intensity of G.

Postacceleration peak. This pressure phenomenon likewise appears, to some extent, to be a function of G and muscular straining; for its time of occurrence is greater under M-1 or PPB conditions, after exposure at 1, 6, and 8 G than at 3 G (no straining). Since this pressure response is greater for the 6 and 8 G groups, however, its magnitude appears to be due primarily to the level of prior G exposure.

Time of recovery. The required time for these arterial pressures to return to original resting values varies directly with the higher G levels. Apparently, therefore, arterial pressure recovery is primarily a function of prior acceleration intensity--the efforts required for PPB or the M-1 are not effective here.

Final resting value. The final resting values were not significantly different from the preacceleration resting levels.

Determinations of Central Venous Pressure

Some of the subjects had to be excluded from the analysis of this measurement for the following reasons:

a. The x-rays were not sufficiently clear to allow for calculation of the correction factor.

b. Occasionally, the transducer's response was damped by small air bubbles which could not be removed.

c. Two subjects were not permitted to undergo x-ray exposure.

During resting periods, the fluctuations of venous pressure throughout the cardiac or respiratory cycles were small; consequently, only a mean value was considered for each 10-sec period. During acceleration, however, large fluctuations of pressure were observed during both the cardiac and respiratory cycles. Therefore, four pressure points on the recordings were identified: (a) inspiration systolic, (b) inspiration diastolic, (c) expiration systolic, and (d) expiration diastolic. During each 10-sec period, a mean value for each of these points was determined.

In order to correct these measurements for the position of the transducer, two approaches were considered: First, the hydrostatic indifference point (HIP) was estimated from the x-ray shadow, and was arbitrarily determined to lie one-third of the distance below the upper shadow of the heart (10). Corrections for the distance from HIP and the transducer were attempted. Second, the venous hydrostatic column length was calculated, using the diaphragm as a reference point. In a manner similar to that in the first approach, the measurements were corrected for the distance between the transducer and the subject's diaphragm.

The figures obtained from corrections based on the HIP showed a wide range of values between subjects; viz, for a particular condition and G-level, the range extended from -20 to +44 mm Hg. On the other hand, when the transducer/subject diaphragm method was used, less variation between subjects was observed. Consequently, these corrected values were assumed to represent a more accurate determination of central venous pressure.

In order to compare G-levels, the central venous pressures were combined from all subjects (in both parts of the study) wearing CSU-12/P suits and performing the M-1 maneuver (Table 7).

No change in pressure with time occurred throughout the resting periods or periods of steady acceleration. Mean resting values were the means of the pressures measured in the six 10-sec control periods preceding acceleration. Mean values during acceleration were obtained from those periods during steady peak G, and mean recovery values from the twelve 10-sec periods immediately after deceleration. Some of the diastolic values obtained from subject "TP" were quite different from

TABLE 7. CENTRAL VENOUS PRESSURES (mm Hg), AFTER CORRECTION FOR TRANSDUCER/SUBJECT DIAPHRAGM DISTANCE, FOR SUBJECTS IN CSU-12/P G-SUITS PERFORMING THE M-1 AT 1, 6, AND 8 G

Subjects (CSU-12/P)	Central venous pressures (mm Hg), during M-1, at: 1 G				6 G				8 G			
	MR	I	E	M.Rec	MR	I	E	M.Rec	MR	I	E	M.Rec
JB	4	11/3*	18/9	5	5	39/28	45/34	4	4	32/17	42/27	6
HL	6	12/3	20/8	6	8	26/16	37/25	14	8	34/13	48/30	12
RL	3	11/1	31/14	6	4	36/21	44/31	5	5	38/28	50/37	6
TP	1	14/-2	21/3	2	-1	23/-1	36/9	1	0	31/12	44/2	7
LP	7	9/3	33/23	8	8	26/8	40/16	9	6	40/25	59/24	7
Mean:	4	11/2	25/11	5	5	30/14	40/23	7	5	35/19	48/24	8

MR = mean resting; I = inspiration; E = expiration; and M.Rec = mean recovery.
* Systolic and diastolic pressures (expressed conventionally for arterial pressure).

those of the other subjects at the same G-levels, but a review of his data prevented its exclusion on technicalities. The values obtained at 1 G were significantly ($P < .01$) less than those at 6 and 8 G.

These pressures, however, were calculated for the level of the diaphragm. The resting values might be assumed to represent the effect of the hydrostatic column between the diaphragm and the HIP. The use of a hydrostatic column-length correction factor to calculate absolute pressures at this point during G resulted in wide variation of values between subjects, similar to the data obtained using the HIP method.

Corresponding results, using transducer/subject diaphragm methods, for the mini-suit and PPB runs are given in Tables 8 and 9. Most of the data obtained with the mini-suit are similar to those with the CSU-12/P, while the values obtained during PPB are variable. The data are, however, insufficient to permit any conclusion as to the effect of either condition on central venous pressure.

Superficial Venous Pressures at Ankle Level

Superficial venous pressures at the ankle were successfully measured in five subjects who wore the RAF mini-suit at 1, 3, 6, and 8 G. Continuous pressure recordings obtained before, during, and after acceleration exposures showed the existence of a dynamic pressure rise during increased G and at 1 G while the subjects performed the M-1. Sampling these data at 10-sec intervals permitted satisfactory reconstruction of the pressure curves (Fig. 10). Consequently, statistical data analyses for G and M-1 effects were performed using these (10-sec) values. Shown in Table 10 are statistical comparisons of the G and/or M-1 effect on ankle venous pressure. Statistically significant differences are found between comparisons for all groups, except between 6 and 8 G. Although the mean pressure value was highest for 8 G, the individual variation was too great to be of statistical significance when compared with 6 G pressures.

Of particular interest is the significant increase in ankle pressure during the performance of the M-1 at 1 G--the increase suggesting some disturbance in blood flow and/or vascular resistance. Similar pressure responses due to the M-1 while at G would be anticipated if the M-1 maneuver was performed during the acceleration exposure. However, although the M-1 effort at 1 G was maximal (as indicated earlier in this report), it is difficult to know the ankle-pressure effect of the M-1 during increased G; for, during acceleration exposure, the physiologic characteristics of the venous system in this region may be altered (viz, all factors that may affect or alter venous wall compliance and resistance).

TABLE 8. CENTRAL VENOUS PRESSURES (mm Hg), AFTER CORRECTION FOR TRANSDUCER/DIAPHRAGM DISTANCE, FOR SUBJECTS IN MINI-SUITS PERFORMING THE M-1 AT 1, 6, AND 8 G

Subjects (Mini-suit)	Central venous pressures (mm Hg), during M-1, at: 1 G				6 G				8 G			
	MR	I	E	M.Rec	MR	I	E	M.Rec	MR	I	E	M.Rec
GD					4	9/-6	13/-3	2	1	12/-1	33/15	2
JB	8	19/4*	37/19	9								
TP	7	13/5	22/12	8	6	24/12	32/13	6	10	30/20	43/22	6
Mean:	8	16/5	30/16	9	5	17/3	23/5	4	6	21/10	38/19	4

MR = mean resting; I = inspiration; E = expiration; and M.Rec = mean recovery.
* Systolic and diastolic pressures (expressed conventionally for arterial pressure).

TABLE 9. CENTRAL VENOUS PRESSURES (mm Hg), AFTER CORRECTION FOR TRANSDUCER/DIAPHRAGM DISTANCE, FOR SUBJECTS IN CSU-12/P G-SUITS PERFORMING PPB AT 1, 6, AND 8 G

Subjects (CSU-12/P)	Central venous pressures (mm Hg), during PPB, at: 1 G				6 G				8 G			
	MR	I	E	M.Rec	MR	I	E	M.Rec	MR	I	E	M.Rec
HL	8	7/5*	14/12	9					9	16/4	27/15	14
JW	7	8/3	12/7	7								
RL					7	33/17	45/29	7	10	66/59	70/54	8
TP	1	2/-3	6/-3	1	0	5/-9	12/-6	-1	0	17/-8	29/2	0
Mean:	5	6/2	11/5	6	4	19/4	29/12	3	6	33/18	42/24	7

MR = mean resting; I = inspiration; E = expiration; and M.Rec = mean recovery.

* Systolic and diastolic pressures (expressed conventionally for arterial pressure).

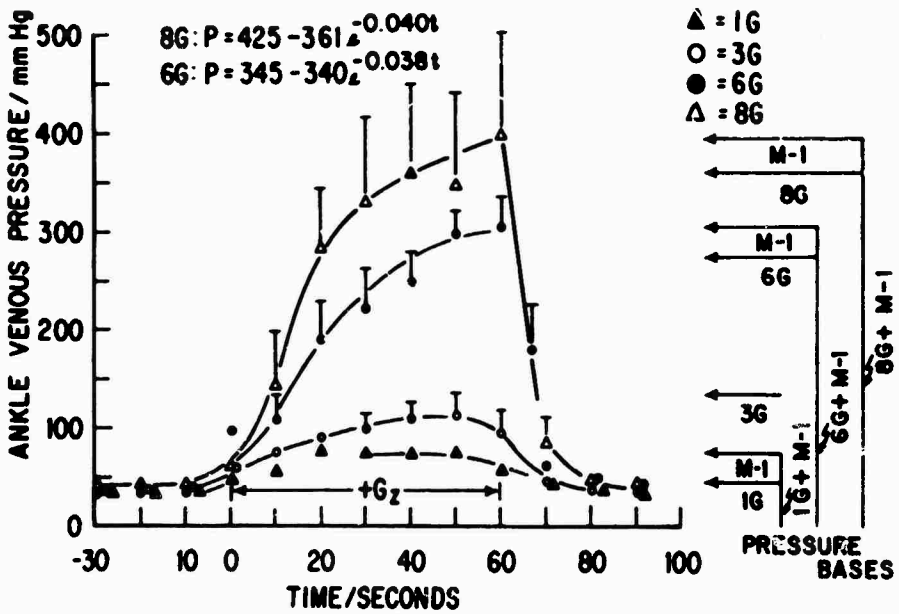


Figure 10. The effects of time, at various levels of G, on ankle venous pressure (mm Hg) are compared with calculated hydrostatic pressure.

The rate of ankle-pressure change was qualitatively similar for 1 and 3 G with M-1, yet distinctively different from pressure changes recorded at 6 and 8 G. The 1 and 3 G pressures increased slowly during the run, reaching a peak at 40 - 50 sec of exposure.

The rates of change of ankle venous pressure, however, during 6 and 8 G, were qualitatively different from those during 1 and 3 G. Before 6 and 8 G acceleration exposure, some increase in venous pressure was found; and the increase continued at a rapid exponential rate for the first 10 sec of G.

This rate of pressure increase for the remaining 50 sec acceleration, however, changes and becomes a negative exponential function with a time-limit asymptote--

6 G:

$$P = 345 - 340\, e^{-0.038t} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (1)$$

and 8 G:

$$P = 425 - 361\, e^{-0.040t} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (2)$$

In both equations

P = pressure (in mm Hg), and

t = time at G (in sec).

TABLE 10. THE MEASURED CUMULATED MEAN OF ANKLE VENOUS PRESSURE (MM HG), FOR FIVE SUBJECTS WEARING THE RAF MINI-SUIT, GROUPED ACCORDING TO G-LEVEL EXPOSURE AND M-1 MANEUVER. (THE VALUES SHOWN--MEAN ± STANDARD ERROR--FOR A SPECIFIC G WERE CALCULATED USING ALL SUBJECTS SAMPLED AT INTERVALS OF 10 SEC THROUGHOUT THE DURATION OF G.)

$+G_z$ (activity)	Ankle pressure (mm Hg)	Statistical significance* of differences between G groups: 1 G (M-1)	3 G†	6 G (M-1)	8 G (M-1)
1 G†	45.0 ± 2.14‡	0.001	0.001	0.001	0.001
1 G (M-1)	69.7 ± 4.29	-	0.05	0.001	0.001
3 G†	91.9 ± 6.13	-	-	0.001	0.001
6 G (M-1)	224 ± 17.1	-	-	-	NS
8 G (M-1)	266 ± 31.9	-	-	-	-

* Student's t-probability (P <).
† No M-1 maneuver.
‡ Mean ± standard error.
NS = (P > .05).

Functions of this type allow a mathematical prediction of a limit that the pressure will approach but not exceed. Mathematics of this type suggest that some regulatory mechanism (feedback) is functioning and, in this instance, controlling the pressure response. The predicted pressure limits of 345 mm Hg at 6 G (Eq. 1) and of 425 mm Hg at 8 G (Eq. 2) were 89% completed after 60 sec at 6 G, and 93% after 60 sec at 8 G. After high sustained G, the ankle venous pressure rapidly returned to pre-G levels (Fig. 10).

Calf muscle pain frequently occurred in subjects who wore the mini-suit and were exposed to 6 G. This pain usually occurred abruptly 5 to 10 sec into the run, and continued unabated until deceleration started. Occasionally, however, the pain continued for several minutes after high-G exposure; and some swelling and tenderness in the calf region was apparent. This leg discomfort became so painful to the subjects that on several occasions they considered halting the runs. It is of interest that, quite unexpectedly, the leg pain in the same subject at 8 G was considerably reduced and often absent.

Data on superficial ankle venous pressure are compared at the various G-levels with the occurrence of leg pain (Table 11). The mean ± standard error of the ankle vein pressure (mm Hg), for subjects grouped according to pain responses only at 6 and 8 G, are: no pain, 191 ± 23.2; moderate pain, 384 ± 46.3; and severe pain, 214 ± 19.8. Persons exhibiting moderate pain had, statistically, significantly higher pressure than those with severe pain. The respective ankle venous pressure of persons exhibiting severe pain was not significantly different from that of persons without leg pain at high G.

Regardless of the interrelationships between superficial venous pressure, acceleration level and duration, and the occurrence of pain, the fact remains that the majority of subjects experienced severe pain almost immediately at 6 G. Since 6 G is a common level for combat aircraft, serious doubts should be entertained about the acceptability of this G-suit.

TABLE 11. THE OCCURRENCE OF VARIOUS AMOUNTS OF LEG PAIN IN RELATION TO G-LEVELS AND ANKLE VENOUS PRESSURE

Subjects	Occurrence of varying amounts of:					
	Pain	Pressure*	Pain	Pressure*	Pain	Pressure*
	(3 G)		(6 G)		(8 G)	
SS	None	-	Severe	151	None	114
LP	None	114	None	234	Moderate	407
JB	None	55	Severe	244	Moderate	360
GD	None	114	Severe	204	None	135
WM	None	97	Severe	257	None	298

*Mean ankle pressure (in mm Hg) determined from data in Table 10.

Changes in Arterial Blood Gases and pH

Arterial O_2 and CO_2 tensions and pH values obtained at the end of each G run are shown for each subject in Tables 12, 13, and 14, respectively. Analysis of variance indicated that significant differences existed between groups with respect to Pa_{O_2} and G-level (Table 12). Not all of the subjects in the 8 G group, however, tolerated this level of acceleration for the entire 60 sec (Table 2)--the tolerance range being 8 sec to 1 min. Blood was withdrawn during the deceleration period at the termination of the 8 G exposure period, regardless of the duration of tolerance. Since a wide range of exposure times existed, it was possible to estimate the change in arterial oxygen tension as a function of exposure time to 8 G (Fig. 11). When the mean arterial Pa_{O_2} of the 1 G group was considered as approximating 0 time at 8 G, a rapid reduction in oxygen tension occurred during the first 20-sec exposure to 8 G. However, continued acceleration exposure for an additional 40 sec caused little additional reduction in Pa_{O_2}. This relationship between Pa_{O_2} and time (in seconds) at 8 G is most accurately (highest correlation coefficient) expressed as Pa_{O_2} being proportional to a negative power of time (in seconds) at 8 G:

$$Pa_{O_2} = 104\ T - 0.196 \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (3)$$

in which

Pa_{O_2} = arterial oxygen tension (mm Hg)

T = time at 8 G (in sec)

r = 0.90 ($P < .01$).

Exposure time during sustained high G is obviously an important consideration in arterial oxygen tensions. Consequently, a mean value of Pa_{O_2}, representative of 8 G in a sustained acceleration study (if compared with other sustained G-levels), should include only those values obtained after the subject had been at peak G for some period of time. A minimum of 50-sec exposure to 8 G was considered, therefore, as representative of a sustained high-G exposure. This time period was chosen not only because, in this study, it included an even number of subjects (3 each) performing the M-1 or PPB, but also because 50 sec had been chosen by Glaister (ref. 11: Fig. 10-8) as representative of blood gas changes for a specific sustained G-level.

TABLE 12. ARTERIAL P_{O_2} LEVELS OF EACH SUBJECT AT THE END OF A SPECIFIC LEVEL OF ACCELERATION WHILE PERFORMING THE M-1 MANEUVER AND PPB, RESPECTIVELY

Subjects	Arterial P_{O_2} levels at the end of:							
	1 G		3 G		6 G		8 G	
	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)
RS	92.0	102.0	91.0	95.0	-*	59.0	49.0	55.0
HL	115.0	111.0	51.0	78.0	39.0	43.0	37.0	37.0
JW	92.0	91.0	69.0	77.0	74.5	80.0	57.5	73.0
RL	107.0	95.0	81.0	87.0	62.5	76.0	65.0	63.0
TP	104.0	105.0	76.0	96.0	56.5	62.0	48.0	48.0
RZ	98.0	111.0	70.5	95.0	49.0	64.0	48.0	52.5
Mean:	101.3	102.5	73.1†	88.0†	56.3†	64.0†	50.8	54.8
SE:	±3.7	±3.4	±5.5	±3.6	±6.0	±5.4	±3.9	±5.1

* Insufficient blood taken for analysis.

† Statistically significantly different at the same G-level using paired t-test ($P < .05$).

SE = standard error.

TABLE 13. ARTERIAL P_{CO_2} LEVELS OF EACH SUBJECT AT THE END OF A SPECIFIC LEVEL OF ACCELERATION WHILE PERFORMING THE M-1 MANEUVER AND PPB, RESPECTIVELY

Subjects	Arterial P_{CO_2} levels at end of:							
	1 G		3 G		6 G		8 G	
	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)
RS	34.00	25.50	-*	28.50	-*	28.50	26.00	29.00
HL	21.25	-*	27.50	23.75	28.50	26.50	28.75	-*
JW	22.00	31.00	33.25	33.00	34.75	33.50	30.50	29.50
RL	26.50	30.00	29.50	32.50	30.50	31.50	23.75	28.50
TP	29.50	28.00	33.50	28.50	33.50	29.00	30.00	27.00
RZ	31.00	23.75	31.50	29.25	32.50	32.50	33.25	30.50
Mean:	27.38†	27.65	31.05†	29.25	31.95†	29.92	28.71	28.90
SE:	±2.10	±1.35	±1.14	±1.37	±1.11	±0.93	±1.38	±0.58

* Insufficient blood taken for analysis.

† Significantly different 1 vs. 3 G and 1 vs. 6 G using paired t-tests ($P < .05$).

SE = standard error.

The effect of sustained exposure to various G-levels upon Pa_{O_2} is shown in Figure 12. The Pa_{O_2} relationship to G was mathematically determined using individual subject data for those performing the M-1 (n = 21) and the PPB (n = 20)--

PPB:

$$Pa_{O_2} = 111 - 8.0\ G \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (4)$$

in which

Pa_{O_2} = arterial oxygen tension (mm Hg) and,

G = acceleration exposure for a minimum of 50 sec,

$r = 0.91$ ($P < .01$);

M-1:

$$Pa_{O_2} = 102\ G^{-0.348} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (5)$$

in which

symbols = same as in Eq. 4,

$r = 0.87$ ($P < .01$).

TABLE 14. ARTERIAL pH LEVELS OF EACH SUBJECT AT THE END OF A SPECIFIC LEVEL OF ACCELERATION WHILE PERFORMING THE M-1 MANEUVER AND PPB, RESPECTIVELY

Subjects	Arterial pH levels at end of:							
	1 G		3 G		6 G		8 G	
	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)
RS	7.45	7.50	7.45	7.47	-*	7.48	-*	7.48
HL	7.51	7.51	7.46	7.50	7.49	7.51	7.50	7.48
JW	7.47	7.42	7.43	7.39	7.41	7.43	7.47	7.44
RL	7.35	7.37	7.42	7.37	7.43	7.45	7.45	7.46
TP	7.40	7.41	7.43	7.47	7.45	7.47	7.46	7.46
RZ	7.41	7.48	7.45	7.47	7.43	7.44	7.41	7.43
Mean:	7.43	7.45	7.44	7.45	7.44	7.46	7.46	7.46
SE:	±0.02	±0.02	±0.01	±0.02	±0.01	±0.01	±0.01	±0.01

* Insufficient blood taken for analysis.
SE = standard error.

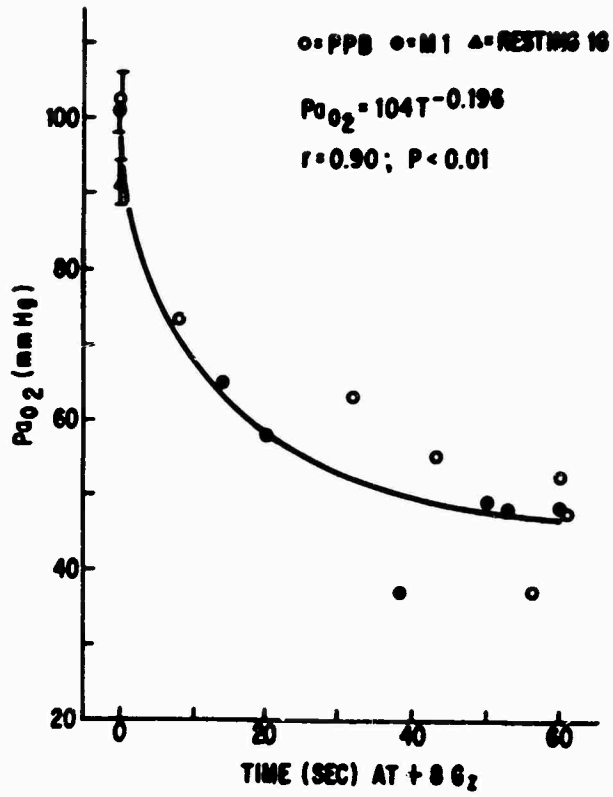


Figure 11. Effect of exposure time at peak 8 G on Pa_{O_2} for both M-1 and PPB subjects. (The resting value is from Michaelson (21).)

Quantitatively different relationships between Pa_{O_2} and G exist for the M-1 and PPB for the G-levels in this study. At 8 G, however, the equations converge--indicating that the protective benefits of PPB occur before 8 G (viz, at the 3 and 6 G-levels).

Arterial oxygen saturations were determined mathematically from the Pa_{O_2}, pH, and Pa_{CO_2} data (Table 15). Arterial desaturation which occurred at 8 G was a function of acceleration exposure time (Fig. 13). However, unlike the sharply inflected log:log relationship found for Pa_{O_2} (Fig. 11), a simple rectilinear arterial saturation:time function appeared to offer the best fit:

$$Sa_{O_2} = 96.4 - 0.197\,T \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (6)$$

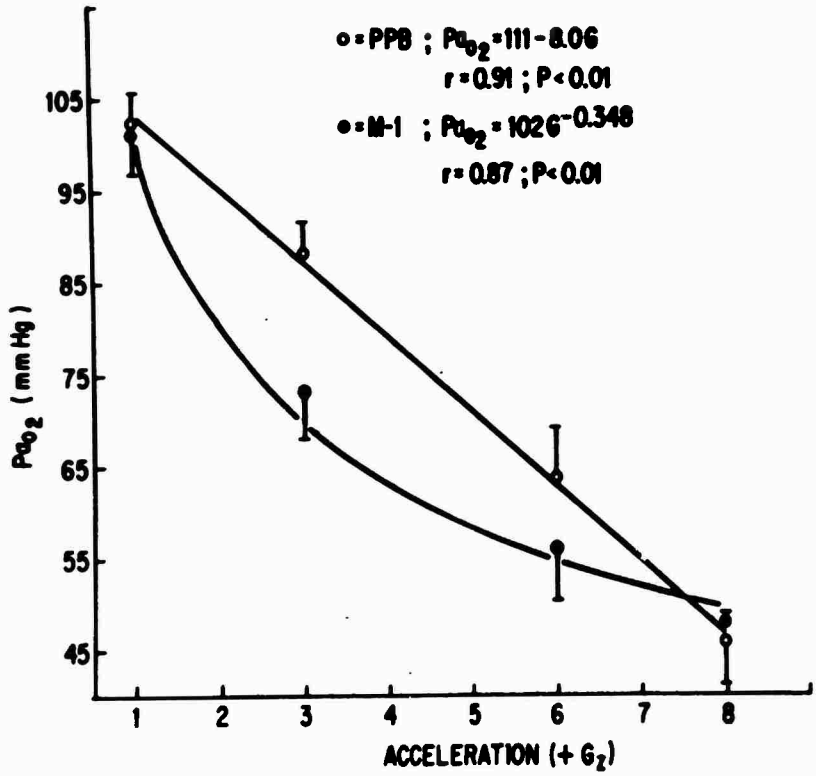


Figure 12. Pa_{O_2} as a function of G, comparing the M-1 with PPB. (Mean values at 3 and 6 G are significantly ($P < .05$) different for M-1 and PPB.)

in which

Sa_{O_2} = arterial saturation (%),

T = same as in Eq. 3,

$r = 0.73$ ($P < .01$).

The different relationship between time at 8 G and Pa_{O_2} (Eq. 3) and percent arterial oxygen saturation (Eq. 6) is a function of the sigmoidal oxygen saturation curve. It is important, therefore, to note here that although the rate of Pa_{O_2} reduction is much less after 20 sec at 8 G (Fig. 11), arterial desaturation appears to continue at the same rate (Fig. 13). One subject ("HL") had lower Pa_{O_2} and arterial oxygen

TABLE 15. PERCENT ARTERIAL OXYGEN SATURATIONS, DETERMINED FROM DATA IN TABLES 12 AND 13, ARE COMPARED FOR THE M-1 AND PPB SUBJECTS AT 3, 6, AND 8 G

Subjects	Comparison of arterial oxygen saturations (%) at:					
	3 G		6 G		8 G	
	(M-1)	(PPB)	(M-1)	(PPB)	(M-1)	(PPB)
RS	97.0*†	97.0	-	92.2	88.3	90.6
HL	89.0	96.1	80.0	87.8	77.8	77.8†
JW	94.8	96.0	94.6	96.3	91.3	95.8
RL	96.5	97.0	93.1	96.0	95.0	91.9
TP	96.0	97.0	89.8	93.4	87.8	87.1
RZ	95.2	97.0	86.0	93.5	85.4	89.0
Mean:	94.8	96.7	88.7‡	93.2‡	87.6	88.7
SE:	1.20	0.20	2.63	1.26	2.38	2.49

* 97.0 = Maximum saturation possible when breathing air at 1 atmosphere pressure.

† Pa_{CO_2} not available; saturation estimated using mean Pa_{CO_2} value for appropriate group (Table 13).

‡ Significantly different at 6 G, using Student's paired t-test ($P < .05$).

SE = standard error.

saturation than the other subjects. During sessions (M-1 or PPB) he halted the 8 G run because of fatigue. It is of interest that he had a prolonged history as a moderate-to-heavy cigarette smoker and was the oldest subject (38 years of age).

In Figure 14, the effects of sustained G (minimum of 50 sec) upon arterial saturation are compared, using individual subject data for the M-1 (n = 15) and PPB (n = 16). Arterial saturation is reduced by exposure to increasing levels of sustained G; the relationship for the M-1 is rectilinear and qualitatively similar to that found for the 8 G exposure

with time (Eq. 6)--as expressed in Eq. 7. For this M-1 relationship:

$$Sa_{O_2} = 99 - 1.59\ G \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (7)$$

in which

Sa_{O_2} = same as in Eq. 6,

G = same as in Eq. 4,

$r = 0.70\ (P < .01)$.

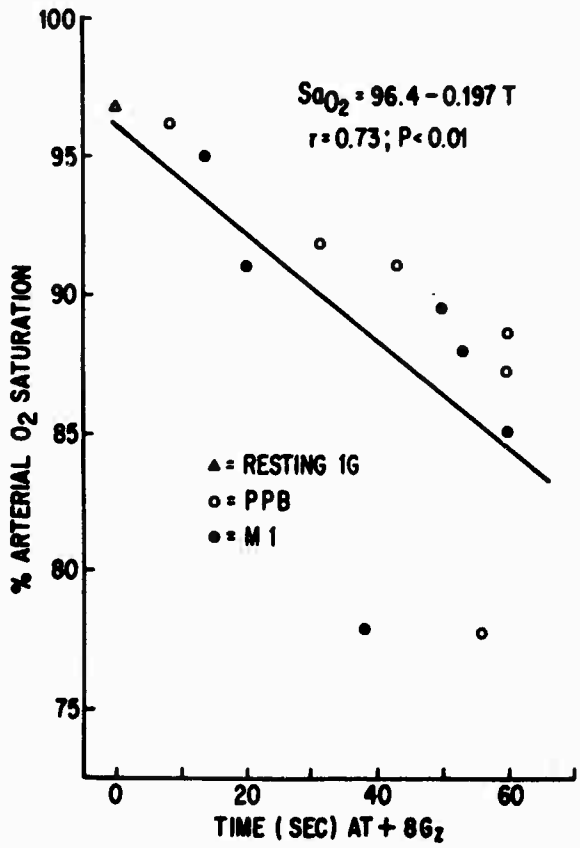


Figure 13. Arterial oxygen saturation (%) as a function of exposure time to +8 G_z for both M-1 and PPB subjects (97% is assumed to be maximum saturation).

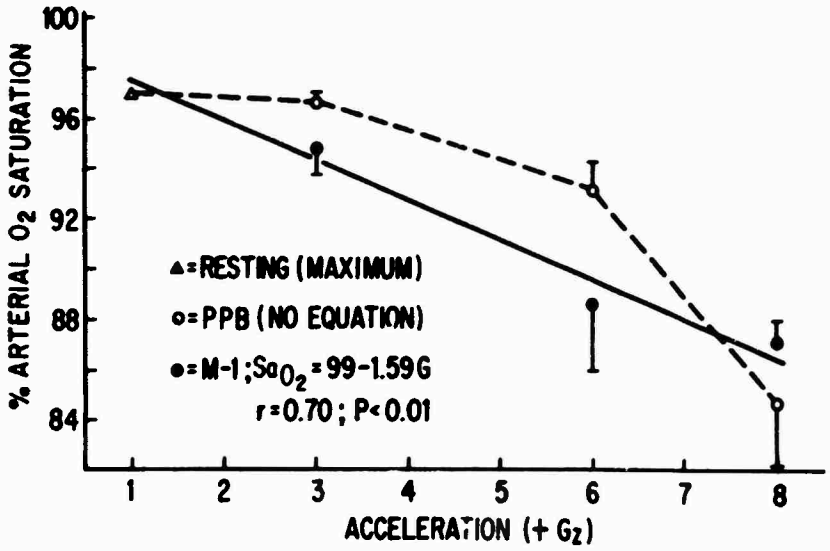


Figure 14. Percent arterial oxygen saturation as a function of G for both M-1 and PPB groups. (Mean values for 6 G differ significantly ($P < .05$) between M-1 and PPB; 97% is assumed to be maximum saturation.)

However, no simple mathematical equation could be fitted to the PPB data. As determined for the Pa_{O_2}, the benefit of PPB is apparently in the lower G (less than 8 G) range; arterial saturations were significantly different (according to paired t-tests) only at the 6 G level (Table 15). Since the benefit of PPB is absent at 8 G, arterial saturations are probably quite similar for both M-1 and PPB above 8 G. The same reasoning might apply also for Pa_{O_2} above 8 G (Fig. 12).

Arterial carbon dioxide tensions (Table 13) were not statistically altered by G specifically, although an analysis of variance showed that differences existed between G-levels for the M-1--those at 1 G having statistically lower (according to Student's paired t-test) Pa_{CO_2} levels than those at 3 and 6 G. Although Pa_{CO_2} values at 1 and 8 G were similar, the differences--comparing 8 G with 3 and 6 G--were not statistically significant. Similarly, at 1 and 8 G the subjects performed a maximum M-1 effort. It would appear therefore that a lower Pa_{CO_2} has some relationship to the M-1 maneuver (i.e., that some fall in CO_2 accompanies a maximum M-1 effort). On the other hand, Pa_{CO_2} levels

in PPB subjects were more consistent and not statistically different between acceleration levels. At the same G-level, however, Student's paired t-tests showed that no significant differences existed between Pa_{CO_2} levels for the M-1 and PPB groups. An absolute evaluation of the Pa_{CO_2} data is not possible because "resting" 1 G data were not obtained in this study.

Arterial pH levels (Table 14) were not significantly different (analysis of variance) between acceleration groups for either the M-1 or PPB.

Analysis of variance determination for arterial P_{CO_2} and pH at all G-levels showed significantly ($P < .001$) less variation within the respective subject than between subjects, indicating that these values were characteristic of the individual.

Change in Heart Size

AP radiographs of the thorax of subjects were obtained during the experiment, and were analyzed for possible changes in heart-shadow size relative to G-level. Heart height and width could be measured directly from the radiographs of 8 subjects. Heart height was taken as the vertical distance (in centimeters) between the diaphragm and the level at which heart shadow merged with the mediastinal shadow. Heart width was considered as the horizontal distance (in centimeters) between the extreme right and left points of the heart shadow. These data for heart height, after correction for magnification--as discussed under "Materials and Methods" (in the subsection: "Estimation of Heart Mass Movement")--are shown for each subject at specific G-levels (Table 16). Analysis of variance determinations showed significant differences ($P < .001$) between acceleration groups, and these differences between specific G groups were statistically identified using Student's t-test (Table 17).

The relationship between G-level and heart height is inverse; and, by using each subject's cardiac measurement ($n = 31$ pairs), a regression analysis shows the following correlation with G:

$$H = 9.68\, e^{-0.063\, G} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (8)$$

in which

H = heart height (cm),

G = acceleration level, and

$r = 0.75$ ($P < .01$).

On the other hand, heart width had no statistically significant relationship with G, and on all x-rays appeared to be quite consistent (Table 18).

TABLE 16. HEART HEIGHT AS RELATED TO SPECIFIC ACCELERATION LEVELS AND CONDITIONS FOR EACH SUBJECT

Subjects	Respective heart heights for:				
	Controls*	1 G†	3 G	6 G	8 G
			(cm)		
JB	8.1	7.5	6.9	5.8	5.8
LP	12.0	10.1	10.4	8.0	7.1
GD	10.0	8.3	7.6	5.5	6.4
SS	11.8	9.4	8.4	7.4	6.6
HL	10.7	10.5	8.1	6.5	5.6
JW	11.0	9.8	9.4	8.8	-
TP	11.2	10.7	6.3	7.2	5.1
RL	10.6	8.8	6.5	5.6	5.0
Mean:	10.68	9.39	7.95	6.85	5.94
SE:	±0.43	±0.39	±0.51	±0.43	±0.30

* Control data were obtained from subjects resting at 1 G.
† Data from subjects performing the M-1 at 1 G.
SE = standard error.

TABLE 17. STATISTICAL COMPARISON OF HEART HEIGHTS (USING STUDENT'S T-TEST) FOR CONTROL AND SPECIFIC ACCELERATION GROUPS

G-levels	Heart heights for acceleration groups: Control*	1*	3	6
1*	.05†	-	-	-
3	.01	.05	-	-
6	.001	.001	NS	-
8	.001	.001	.01	NS

* Control data differ from 1 G as defined in Table 16.
† P < (NS) = (P > .05).

TABLE 18. HEART WIDTH AS RELATED TO SPECIFIC ACCELERATION LEVELS AND CONDITIONS FOR EACH SUBJECT

Subjects	Respective heart widths for: Control*	1 G*	3 G	6 G	8 G
			(cm)		
JB	17.4	15.5	18.8	18.8	15.7
LP	16.5	14.3	16.2	16.9	16.2
GD	10.9	8.9	13.6	14.9	12.2
SS	14.5	14.6	16.6	11.9	15.9
HL	14.8	14.5	11.0	13.1	12.1
JW	12.9	12.3	13.0	14.3	-
TP	14.6	13.3	13.1	15.2	11.1
RL	14.5	14.6	14.6	11.3	12.5
Mean:	14.5	13.5	14.6	14.6	13.7
SE:	0.71	0.74	0.88	0.89	0.82

* Control differs from 1 G as defined in Table 16.
SE = standard error.

Measurements of Esophageal and Gastric Pressures

The esophageal pressure, as an indirect measurement of intrathoracic pressure, depended on the effort expended by the subjects. This effort varied not only with each subject's physical ability but also with his natural G tolerance, for he was instructed not to exert himself beyond the point at which he maintained clear vision. When subjects performed the M-1 maneuver, the pressures during peak effort varied from 60 to 110 mm Hg maximum and from -15 to -40 mm Hg minimum. During PPB, the maximum pressures were 30 mm Hg (the output pressure of the Mr 11 regulator) and the minima ranged from 0 to 20 mm Hg.

Much difficulty was encountered in obtaining satisfactory gastric pressures, since the gastric balloon became empty on many occasions. Endeavors to find the cause of this difficulty proved fruitless; however, by means of filling the balloon immediately before a run, the emptying--as indicated by a fall in recorded pressure--was observed to be slow. Maximum pressures at the beginning of the run were in the same range as the esophageal (60 to 110 mm Hg); and, although there was a gastric pressure fluctuation with respiration, this fluctuation was smaller (minimum pressures 30 to 60 mm Hg) even where the anti-G suit was not inflated. Because of the difficulties encountered in obtaining these data, little significance can be attached to them.

Comparison of Data from Parts 1 and 2 of Study

For each parameter that was common to both part 1 and part 2 of this study (viz, when the subjects wore the CSU-12/P anti-G suit and performed the M-1 maneuver), there was never any significant difference between the data.

DISCUSSION

The two-fold intent of this study (as already explained in the "Introduction") has been to compare: (a) physiologically, the RAF prototype mini-suit and the standard USAF CSU-12/P G-suit, at high sustained G-levels; and (b) the PPB technique and the M-1 maneuver as methods of increasing man's tolerance to acceleration. As frequently happens in research, however, the number of questions raised by the experiments rivaled the number answered! In this attempt to investigate numerous variables affecting human tolerance to high sustained $+G_z$ acceleration, quick decisions were at times necessary--such as having to sacrifice one measurement in order to obtain others. (For

this reason, little information was gleaned about gastric pressure, and some of the information on central venous pressure was lost.) The overall information obtained, however, adds significantly to the hitherto limited knowledge of this subject.

Factors in Assessing G-tolerance

A man's tolerance to $+G_z$ acceleration can be assessed in two ways. The first method (used since the effects of G were first studied, before and during World War II) measured the G-level at which a relaxed subject first lost peripheral vision--the greyout threshold. This method, if one assumes that subjects truly can be relaxed, measures basically the effects on and responses of the cardiovascular system to anti-G systems or other factors. As such, this approach is useful and has contributed much to the knowledge of G tolerances (viz, the basis on which the anti-G systems of both the RAF and USAF have been developed). On the other hand, this approach has two basic deficiencies: (a) The levels of acceleration which produce blackout in relaxed subjects are well below those expected from modern aircraft. (b) No pilot or navigator is relaxed when accelerated in flight--a factor which alters G tolerances and the bases for acceleration protection.

The second method (alternative to G-tolerance measurements in a relaxed state) is to investigate, using various physiologic and mechanical protective techniques and devices, the maximum G which a man can tolerate before losing his vision. Studies using this condition are less easy to evaluate than "relaxed measurements"; for the condition depends not only on levels of G, but also on time at G, and on the physical as well as the emotional stamina of the respective subject. Moreover, at some subsequent G-level (or time), an aircrew member may exceed the mechanical stress limits of critical organs (such as heart, lungs, or major vascular beds) without the preceding blackout--but with catastrophic results. Therefore, until more is known about high sustained G, some compromise between maximum and relaxed tolerances must be accepted.

In this study, maximum G tolerances were not attained with several men (Table 2) who successfully completed 60 sec at 8 G--the arbitrary limit set for this experiment. On the other occasions (apart from a single technical failure), however, maximum G tolerances were attained, with the tolerance criteria being fatigue or visual loss (Table 3). The question which arises, in the five instances of fatigue, is whether this condition would have occurred in flight. The motivation and morale of these subjects were high. Considerable local publicity surrounded the project

and, in addition, the subjects maintained an unofficial competition as to who was the most capable of tolerating the acceleration. Whenever a subject had failed to complete 60 sec at 8 G during his first session, he announced most strongly his intent to "make it this time" on his second exposure. This additional motivation apparently was not effective, because G tolerances (seconds at 8 G) for the second exposure (46.6 sec) did not differ statistically from those of the first (42.8 sec). Of course, a fighter pilot undergoing imminent physical threat, or rapidly approaching a favorable gun-bearing position, is most likely to exert any physical effort required to maintain vision and consciousness. USAFSAM centrifuge data--based on the experiences of more than 75 Tactical Air Command (TAC) pilots (of F-4E aircraft) who were exposed to >7.5 G/30 sec for the first time--indicate that pilots can be rapidly trained to withstand these forces through proper indoctrination, motivation, and competition

Responses of Heart Rate and Rhythm, and Arterial Pressure

Three cardiovascular parameters (heart rate and rhythm, and arterial pressure) are considered together, because their responses to high sustained G suggest that they are functionally related. These parameters are presented here chronologically and in relation to G exposures.

Preacceleration response. The preacceleration rise, in both heart rate and arterial pressure, apparently is due to anticipation of the approaching stress (23). Arterial pressure before a 1 G run was significantly lower than before an acceleration run.

Acceleration response. In observed cardiovascular parameters, the differences between the effects of PPB and the M-1 maneuvers during acceleration were not great, but included: (a) lower heart rate at 1 G during PPB; and (b) more rapid heart-rate recovery after several G exposures in those men using PPB.

These results offer physiologic evidence in favor of the use of PPB as a method of protecting a subject against high sustained $+G_z$ acceleration. This study found no physiologic reasons against PPB and in favor of the M-1; but some technical objections to PPB may arise, since it would demand a more complex aircrew support system. As evidenced by the high heart rates and fatigue which the subjects experienced, 8 G apparently exerts extreme physical and emotional stress on the subject. In addition, the stress of 8 G for 60 sec exceeds the acceleration tolerance of the subjects--thus indicating that improved

G protective devices and techniques are necessary if aircrews are to operate effectively in a high sustained G environment.

The occurrence of premature beats may be a cause for concern. Although no subjects in this study produced long runs of PVCs, such runs did occur in earlier studies, as reported by Parkhurst et al. (23) and Shubrooks (25). Possible causes of these abnormal beats, as discussed in detail by Shubrooks (25), are: high heart rate (possibly due to an excessive sympathetic activity); changes in heart position; and some degree of cardiac ischemia and/or possibly changes in cardiac filling and blood volumes. Owing to the possibility of myocardial ischemia or severely decreased heart volumes, it should be emphasized here that--although these cardiac rhythm changes are not "serious"--additional stresses could precipitate "a sustained serious rhythm disturbance" (25).

Shubrooks (26) reported less respiratory-induced variations in systemic arterial blood pressure with PPB than with the M-1 at high sustained G. In our study, we were unable to demonstrate consistently a similar finding (Table 5). In contrast, similar minimum and maximum systolic arterial pressures and pulse pressures were obtained from 3 subjects (RJ, RS, and HL) at 1, 6, and 8 G whether using PPB or M-1. Three types of arterial pressure responses appear to be associated with PPB or M-1, as shown in Figure 8: (a) for subject RZ, less pressure fluctuation with PPB (similar to that reported by Shubrooks, ref. 26); (b) for RS, less pressure variation with M-1; and, (c) for HL, similar pressure responses with M-1 or PPB.

Several differences exist, however, between our study and Shubrooks' (26), which may explain these apparent discrepancies. Shubrooks (26) had fewer yet probably better trained subjects who used 40 mm Hg PPB, whereas our men used 30 mm Hg PPB. Considering both studies, however, we may conclude that: (a) PPB can reduce, significantly, the marked variations in arterial pressures at eye-level in specific individuals (viz, RZ in our study, and subject "P" in Shubrooks' study); and (b) although this marked reduction in arterial pressure fluctuation is not apparent in other subjects while pressure breathing, PPB is as effective as the M-1 in maintaining eye-level arterial pressure. PPB significantly reduced marked variations in esophageal pressures associated with respiration while performing the M-1 (Table 5). (This esophageal pressure response is discussed in some detail subsequently in the subsection on "Subject fatigue associated with the M-1 and PPB.")

Postacceleration response. Two particularly interesting facets of this period are: (a) the marked sinus arrhythmia, and (b) the slow rise of the arterial pressure to a peak level. The postacceleration eye-level arterial pressure "overshoot," observed in these experiments, was primarily a function of pulse pressure (in part 1, for example, the 3-G postacceleration peak: 145 systolic - 62 diastolic = 83 mm Hg pulse pressure; and the post 8-G peak: 174 systolic - 74 diastolic = 100 mm Hg pulse pressure). The occurrence of this arterial overshoot was considerably delayed beyond the post-G-deceleration reflex-type pressure "rebound" overshoot frequently reported following short-duration acceleration exposures--clearly shown for man after a 5-G run (19: Fig. 17).

Arterial pressure overshoots after high-acceleration exposures have been studied in some detail in animals (2, 12, 27). These pressure overshoots were reported to occur 0.5- to 3-min post-G exposure, the degree and duration of which appeared to be directly dependent upon: (a) extent and duration of cardiovascular accommodation during G; and (b) inactivity of the carotid sinus (27), or activity of the carotid sinus (12). Burton (2) reported that the magnitudes of arterial and pulse pressure overshoots which occurred in miniature swine, after 15-sec exposure to +2 to +8 G_z, were independent of G-suit inflation or prior Gs; but the occurrence of such overshoots was significantly delayed after the higher G levels, viz, occurring at 28.6 ± 5.4 sec (mean ± standard error) at +7 G_z.

Although these arterial pressure overshoots apparently may be exaggerated by tampering with cardiovascular pressure control mechanisms, it is still evident that--even in the control (no surgery) dogs--after 1 min of +9 G_x exposure (27), an arterial pulse pressure increase of 7% to 9% occurs within 1 min post-G. Conceivably, therefore, these slowly occurring post-G pressure overshoots may have a noncardiovascular (possibly metabolic) basis and may be quite independent of the altered cardiovascular system during G exposure. However, if the physiology of the mechanism of cardiovascular homeostasis is altered significantly (e.g., denervation of the carotid sinus) and an increase in pulse pressure occurs (regardless of origin), the return to preforced levels will be quite delayed.

Combined information--on the work performed by the subjects at G, and a reflex peripheral vasoconstriction which would have been present at G--suggests that in many vascular beds a buildup in metabolites may have occurred; viz, the result of a high oxygen debt (lactic acid formation) and slow blood flow. The pattern of the post-G arterial

pressure peak was as might be expected, considering a metabolically induced response. Also, the time required to reach a peak was directly correlated to G-level, the longest time being in those situations where the largest buildup of metabolites would occur.

There are several possible explanations for the observed sinus arrhythmia: (a) respiratory activity, or (b) variations in the volume of blood entering the heart. In these experiments, both respiratory drive and cardiac output were taxed. Consequently, in the wake of repaying oxygen debt and clearing anaerobic metabolites, the marked sinus arrhythmia was a likely cardiac response. Also, the duration of this sinus arrhythmia pattern paralleled the post-G arterial pressure peak phenomenon (Figs. 4 and 9). Since a fast heart rate usually precludes a sinus arrhythmia, the impetus for the sinus arrhythmia may have been present but imperceptible until the heart rates became low enough for irregularity to begin.

Responses of Superficial (Ankle) Venous Pressure

The change in ankle pressure is directly correlated with G-level, as might be expected in considering the physical effect of acceleration on hydrostatic pressures:

$$P = h\,d\,g \qquad (9)$$

in which

P = hydrostatic pressure,

h = column height,

d = specific density of blood, and

g = acceleration force (G).

Expected (theoretical) hydrostatic pressures were calculated for each G-level by using equation 9. The column height was considered as the distance between the heart base and ankle (pressure transducer location), as determined for a 5-ft 10-in. man (i.e., 28-in. water pressure which, at 1 G, equals 52.7 mm Hg). These calculated ankle venous pressures are compared with measured values in Figure 10. The measured pressures at 6 and 8 G are slowly approaching what appears to be an asymptotic limit, and this limit corresponds to calculated hydrostatic pressures at the 6 and 8 G levels.

For blood to flow from the leg during $+G_z$, the intravenous pressures would have to be at least above the physical restraint of these calculated hydrostatic pressures. Since these values are below, yet approaching these pressures, it would appear that blood is not flowing from the lower leg region. However, because deep venous pressures of the lower leg were not measured and, because the majority of blood flow from the leg occurs through deep leg veins, sufficiently high venous pressures for blood flow could be occurring undetected by us.

With these limited data, it is difficult even to speculate as to the physiologic meaning of these pressure:time relationships. Here attention should be called to the complex interrelationships which exist between blood flow, volume, and pressures on apparent venous distensibility of man, as recently suggested by the studies of Hollenberg and Boreus (13).

Unfortunately, this aspect of acceleration physiology (blood flow from the legs during high G) is little understood. The findings of this investigation clearly show the need for further studies and also for experiments in three specific areas: (a) superficial venous pressure responses at 1 G to various degrees of upper leg external pressures (tourniquet); (b) deep venous pressure responses in the lower leg during various sustained G-levels, and their relationships to superficial ankle pressures; and (c) the relationship of these pressures and pressure gradients to blood flow. The third experiment area, (c), could probably be most successfully conducted on an experimental animal such as a miniature swine (3).

Description of Heart Volume Calculations

Heart height, as detected on x-ray, was significantly reduced during G; but no significant change was found in heart width, thus suggesting that the total heart shadow area is less at G. Franks et al. (9) also reported that (in man) during increased G, AP x-ray films showed a "marked reduction" in cardiac shadow size as compared with 1 G films. The extent of this heart-size reduction, however, was not reported. A qualitatively similar response in the heart shadow size of dogs, during acceleration exposure, also has been reported by Agadyhanyan and Mansurov (1). They found a reduction in total heart area (in square centimeters) during acceleration exposure, progressing from control values of 32 - 35 cm^2 to 22 - 24 cm^2 at +8 G_z. This reduction in heart area affected primarily the "transverse section"; the "longitudinal section" remained essentially unchanged.

Heart shadow area was calculated for the human from the data of Tables 16 and 18 by arbitrarily using the area equation of an ellipse. These calculated values for the different G exposures are shown in Table 19.

TABLE 19. HEART SHADOW AREAS (cm^2) HAVE BEEN CALCULATED,* COMPARED FOR EACH SUBJECT, AND GROUPED ACCORDING TO G-LEVEL (WITH HEART VOLUME† CALCULATIONS BEING IN PARENTHESES)

Subject	Heart shadow areas at acceleration levels				
	Control‡	1 G‡(†)	3 G(†)	6 G(†)	8 G(†)
JB	109(1138)	85(893)	100(1000)	86(797)	71(578)
LP	156(1948)	111(1169)	132(1517)	105(1076)	89(840)
GD	86(797)	56(419)	81(729)	63(500)	61(476)
SS	135(1568)	107(1106)	109(1138)	69(573)	82(743)
HL	123(1364)	117(1265)	69(573)	65(524)	53(386)
JW	112(1185)	94(911)	96(941)	98(970)	--
TP	128(1448)	110(1154)	63(500)	86(798)	43(282)
RL	119(1298)	101(1015)	73(624)	49(343)	48(333)
Mean:	121(1343)	98.1(992)	90.4(878)	77.6(698)	63.9(523)
SE:	7.23(119)	6.84(94)	8.24(121)	6.78(89)	6.59(80)

* Heart shadow area (cm^2) for each subject was calculated from heart height and width data (Tables 16 and 18), using the area equation of an ellipse: πab, in which a and b are the radii of the ellipse.

† Heart volume estimations are calculated as heart shadow area raised to the 3/2 power.

‡ Control values differ from 1 G, as defined in Table 16.

SE = standard error.

The heart shadow area is smaller at the higher G levels than at 1 G (M-1) or at 1 G at rest (control). Analysis of variance was significant ($P < .01$), indicating that one or more of the accelerations were statistically different. Table 20 shows that subjects at the higher G levels (6 and 8 G) had statistically smaller hearts than when resting or performing the M-1. An especially interesting fact is that the M-1 activity significantly reduced heart size compared with resting at 1 G (control). Consequently, it appears that the size of the heart shadow (like the heart rate), at 6 and 8 G, is a function of both acceleration per se and the M-1.

TABLE 20. STATISTICAL COMPARISON OF HEART AREA (USING STUDENT'S PAIRED T-TEST) BETWEEN CONTROL AND SPECIFIC ACCELERATION GROUPS

G-levels	Heart area for acceleration groups: Control*	1*	3	6
1*	.001*†	--	--	--
3	.01	NS	--	--
6	.01	.05	NS	--
8	.001	.05	.001	NS

* Control data differ from 1 G as defined in Table 16.
† $P <$ (NS) = $P > .05$.

Heart shadow area should have a relationship to the 3/2 exponent with heart volume ($a \alpha v^{2/3}$); and, since heart rate differs only slightly at 6 and 8 G, heart volume estimations may function as a crude index of cardiac output. Heart volume, as estimated from the heart shadow area, increased to the 3/2 exponent, and is shown (in parentheses) for each subject in Table 19.

Lindberg et al. (20), using dye-dilution techniques, determined cardiac output in men wearing inflated G-suits, exposed to +2, 3, and 4 G_z. They reported a significant reduction in cardiac output at increased G. From their data, it was possible to calculate the relationship of $+G_z$ to percentage reduction in cardiac output:

$$CO\ (\%) = 5.9\ G - 5.5 \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (10)$$

in which

CO (%) = percent reduction in cardiac output compared with 1 G relaxed controls; and

G = level of acceleration exposure.

By using this equation, percentage reduction in cardiac output from 1 G controls was estimated at 3, 6, and 8 G and compared with percentage reduction in heart volume as a function of 1 G performing the M-1 (Table 21). Similar reductions are found between cardiac output and heart volume. Consequently, it appears that heart shadow area is correlated to cardiac output at high sustained $+G_z$ acceleration.

TABLE 21. PERCENTAGE REDUCTION IN CARDIAC OUTPUT, AS DETERMINED FROM DATA OF LINDBERG ET AL. (20), COMPARED WITH PERCENTAGE REDUCTIONS IN HEART VOLUME AS A FUNCTION OF G

G-levels	Reductions in: Heart vol. (This study)	Cardiac output (Lindberg et al. (20))
	%	
1*	--	--
3	12	12
6	30	30
8	47	42

* 1 G performing the M-1.

Causes of Arterial Oxygenation Responses at 8 G

After decreases in pulmonary ventilation, some finite period of time is required for reductions in Pa_{O_2} and saturation to occur; e.g., after breath holding, approximately 20 - 30 sec elapse before a reduction in arterial saturation and Pa_{O_2} is noted (8). Similarly, encroachment on pulmonary function occurs at high sustained G; and here, also, some finite period of elapsed time precedes reductions in Pa_{O_2} or arterial

saturation. Figure 11 suggests that the reduction in Pa_{O_2} at 8 G begins immediately; viz, at T = 10 (Eq. 3), Pa_{O_2} approximates 70 mm Hg--a reduction of 20 mm Hg during the first 10 sec at peak 8 G. Of course, each point in Figure 11 represents only 1 subject; and, since individual variation occurs, the shape (slope) of this Pa_{O_2}:time at 8 G relationship during the first 20 sec is still somewhat obscure. However, this apparent instantaneous lowering of Pa_{O_2} at 8 G (Fig. 11) does not account for the time at G prior to attaining the 8 G level; viz, at least 7 sec elapse (at the standard rapid onset rate of 1 G/sec) before reaching 8 G. Quite obviously, physiologic responses to acceleration are occurring, but are usually undetected (because of experimental techniques) during this G onset duration. In the past, minimal consideration has been given to the effect of this G onset rate upon peak G data, because at lower G levels the onset duration was short. As the peak G increases, however, the duration of G onset increases. The probable result is an exponential increase in the onset of the G effect, raising it to a point at which this effect becomes significant and accountable--especially if a time dependency at G is to be determined.

Relation of Subject Fatigue to the M-1 and PPB

All of the men were fatigued after each day's acceleration exposures. Each subject reported that PPB was less fatiguing than the M-1, although at 8 G no significant difference in acceleration tolerance was found. The two subjects who had less tolerance with PPB than with the M-1 (especially JW) had difficulty in positive pressure breathing at G without also performing an "M-1 like" maneuver. They appeared to distrust PPB as an aid to G tolerance because PPB required such little effort on their part.

Fatigue appeared to be directly related to the energy expended by a subject, inasmuch as maximum work effort produced exhaustion in a relatively short period of time. No attempts were made to measure energy expenditure during G; but the following parameters show differences between PPB and the M-1 which suggest how PPB is less fatiguing:

The smaller amplitude of the esophageal (intrathoracic) pressure cyclic fluctuations found with PPB (Fig. 8; and Table 5) suggests that less work (energy) was required to maintain vision during acceleration (intrathoracic pressure differential being, to a large extent, a function of chest wall muscular activity).

Heart rate during G conditions is a function of the M-1 or PPB as well as acceleration; however, Figure 5 indicates that less tachycardia occurs during PPB than during the M-1 at 1 G, and that recovery after several G runs is more rapid with PPB.

Blood gas differences also may suggest some basis for this greater fatigue with the M-1 than PPB, because lower oxygen tensions were observed with the M-1 at the end of 3 and 6 G. More important, a significant difference in lowering of oxygen saturation between the PPB and M-1 groups was found at 6 G, suggesting less oxygen delivery to the tissues in those persons doing the M-1. The probable result is an increase in anerobic metabolism (lactic acid formation) which could contribute to the development of fatigue.

The basis for the higher Pa_{O_2} during PPB at 3 and 6 $+G_z$, as compared to the M-1 maneuver, could be due at least in part to increased oxygen utilization during the latter. However, it can also be explained by the effect of PPB G-induced changes in the lung. The fact that the airways in the dependent portions of the lungs have a tendency to collapse during $+G_z$ (14) exaggerates the gross ventilation-perfusion inequality ($\dot{V}_A/\dot{Q}$) caused by redistributed blood and results in venous admixture. If functional residual capacity (FRC) is increased by PPB, airway closure will be minimized. This mechanism could also explain the similarity of the values of Pa_{O_2} for the M-1 maneuver and PPB at $+8\ G_z$ since, at this level, anti-G-suit inflation limits lung expansion (21). Also, a further increase in $\dot{V}_A/\dot{Q}$ inequality at $+8\ G_z$ may assume increasing importance, compared to the effects of PPB, in terms of impaired gas exchange. The beneficial effects of a given level of PPB may be limited by reduction in cardiac output, overexpansion of areas of normal lung, and increase in the vertical nonuniformity of blood flow in the lung. Nevertheless, the differences we observed would appear to give a physiologic advantage to a pilot in a G-on-G maneuver during which transient levels of 7 - 10 $+G_z$ may be reached from a 3 - 6 $+G_z$ plateau.

The effect of sustained acceleration on Pa_{O_2} in subjects performing the M-1 (refer to Eq. 5) was determined in this study by using data obtained at 3 levels of increased acceleration; and the slope (-0.348) suggests a lessening of G influence upon lowering Pa_{O_2} especially in the higher G range. Using equation 5, an extrapolation to 10 G predicts a Pa_{O_2} of 46 having 83% saturation--a reduction of only 1 - 2 mm Hg below the 8 G level (a comforting thought!). A similar prediction regarding arterial saturation may be made by extrapolating to 10 G (using Eq. 7).

A previous study by Michaelson (21), in which the subjects also breathed air and performed the M-1, suggested that greater reductions in Pa_{O_2} would occur in the higher (8 - 10 G) $+G_z$ range. He measured Pa_{O_2} from blood extracted during the last 20 sec of 45-sec G-exposure periods (at 3, 5, and 7 G). Considering the rapid reduction in Pa_{O_2}, with time, which occurs during high sustained G (Fig. 11; Eq. 3), Michaelson's data (21) at 5 G and 7 G would probably match values obtained in this study by end-acceleration sampling. Therefore, the Pa_{O_2} data from both studies were compared in Figure 15 and their relationships to G were mathematically determined:

$$Pa_{O_2} = 97.5\, e^{-0.093G} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad (11)$$

in which

symbols are the same as in Eq. 4, and

$r = 0.996$ ($P < .01$).

This exponential equation differs qualitatively from the logarithmic equation (Eq. 5), determined as best fit for the data presented herein. Both regression lines are compared also in Figure 15; and, although qualitatively dissimilar, the curves approximate each other over the G-range studied. If, however, we extrapolate to 10 G (using Eq. 11), the Pa_{O_2} is lowered to 38 mm Hg with an approximate 76% oxygen saturation. This situation is more serious than that predicted using the parabolic equation (5). Additional investigations at high sustained G are necessary to identify the exact descriptive mathematical function.

Although PPB is no more effective regarding oxygen saturation at 8 G than the M-1, PPB <u>is</u> of significance because of our renewed interest in high-acceleration physiology stemming from the new generation of aircraft. Present-day fighter aircraft, although occasionally reaching 8 G, usually perform in the lower 2 to 3 G range for the entire engagement period of 1 to 2 min. The newer aircraft with higher G limits might also be generally used at less than their maximum G capability, although their routine G-level in aerial engagements may well increase from 3 to 6 G. Sustained 6 G for 1 to 2 min is decidedly fatiguing, and here is where PPB is superior to M-1 breathing.

If, during aerial maeuvers, a pilot using PPB chooses to move from 6 G's to 8-10 G's, he might have a greater oxygen tension in arterial blood upon entering the higher G-level. In our study, although

arterial oxygen tension and saturation were similar for both PPB and M-1 at 8 G (Figs. 12 and 14), the PPB might have been superior if the subjects had been at 6 G for several seconds before continuing to a final 8 G exposure. It appears, therefore, that PPB, because it is most effective in the 6 G range, would have its greatest operational value for pilots of planes with G capabilities far exceeding the levels at which PPB functions effectively as an anti-G device.

Our opinion, resulting from this study, is that--at levels of 6 G and above--a pilot using PPB will maintain a distinct advantage over an adversary using M-1 because fatigue is lessened and arterial oxygen tension is higher with PPB.

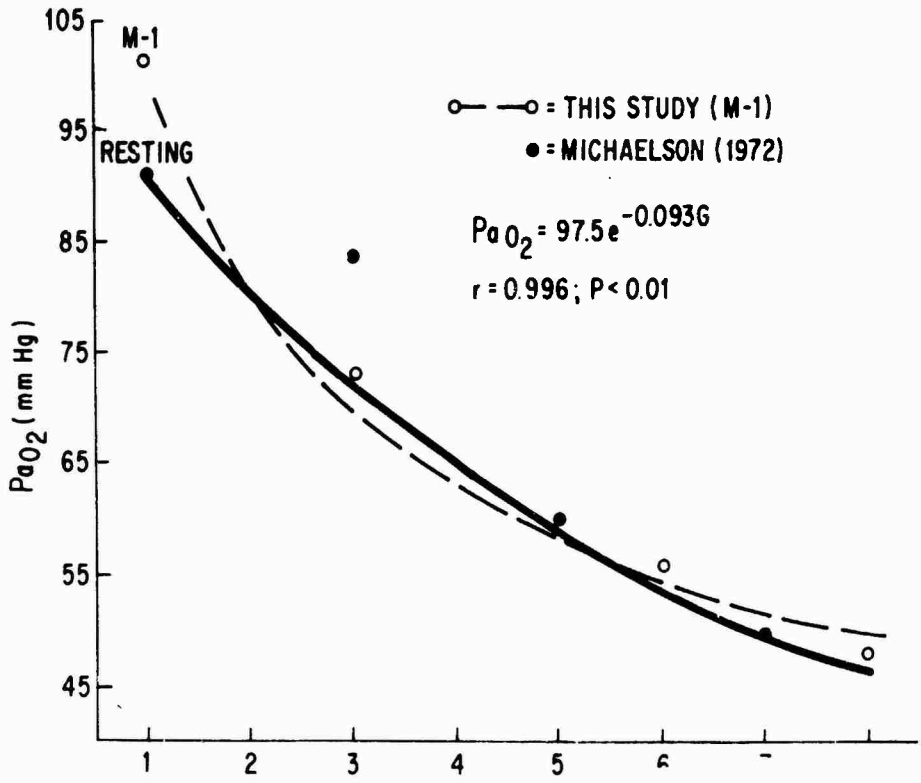


Figure 15. The Pa_{O_2} data of Michaelson (21) are compared with the M-1 data from Figure 12. (The exponential equation, represented by the continuous line, is developed using values from both studies.)

REFERENCES

1. Agadyhanyan, N. A., and A. R. Mansurov. The effect of oxygen deficiency and prolonged radial acceleration on an animal organism. Bull. Exp. Biol. Med. 53(4):42-46 (1962). (Biol. Eksp. Biol. Med. (Moskva, USSR))

2. Burton, R. R. Cardiovascular responses of miniature swine to $+G_z$ acceleration. Physiologist 15:97 (1972).

3. Burton, R. R. The development of the miniature swine as a human analog for positive acceleration research. Aerosp. Med. 44:294-298 (1973).

4. Crossley, R. J. A survey of pilots' use of two gradient anti-G valves. RAF Institute of Aviation Medicine, RAFIAM Report No. 95, Farnborough, Hants, England, 1970.

5. Crossley, R. J., and D. H. Glaister. Evaluation of a "mini" anti-G suit. RAF Institute of Aviation Medicine, RAFIAM Report No. 484, Farnborough, Hants, England, 1970.

6. Crossley, R. J., J. H. Lemon, and G. M. Turner. An evaluation of the proposed mini anti-G suit. RAF Institute of Aviation Medicine, RAFIAM Report No. 200, Farnborough, Hants, England, 1972.

7. Ernsting, J. Some effects of raised intrapulmonary pressure in man. AGARDograph 106, pp. 229-312. London: MacKay, 1966.

8. Ferris, E. B., et al. Voluntary breathholding. III. The relation of the maximum time of breathholding to the oxygen and carbon dioxide tensions of arterial blood, with a note on its clinical and physiological significance. J. Clin. Invest. 25:734-743 (1946).

9. Franks, W. R., W. K. Kerr, and B. Rose. Some effects of centrifugal force on the cardio-vascular system in man. J. Physiol. 104:9P-10P (1945).

10. Glaister, D. H. The effect of acceleration on the cardiovascular system. In Glaister, D. H. (ed.). The effects of gravity and acceleration on the lung. AGARDograph 133, ch. 5, p. 69. Technivision Services, Slough, England, 1970.

11. Glaister, D. H. The effect of acceleration on gas exchange, arterial oxygen saturation, and alveolar shunting. In Glaister, D. H. (ed.). The effects of gravity and acceleration on the lung. AGARDograph 133, ch. 10, p. 166. Technivision Services, Slough, England, 1970.

12. Greenfield, A. D. M. Effect of acceleration on cats, with and without water immersion. J. Physiol. 104:5P-6P (1945).

13. Hollenberg, N. K., and L. O. Boreus. The influence of rate of filling on apparent venous distensibility in man. Can. J. Physiol Pharmacol. 50:310-316 (1972).

14. Jones, J. G., S. W. Clarke, and D. H. Glaister. Effect of acceleration on regional lung emptying. J. Appl. Physiol. 26:827-832 (1969).

15. Lambert, E. H., and E. H. Wood. Direct determination of man's blood pressure on the human centrifuge during positive acceleration. Fed. Proc. 5:59 (1946).

16. Lemon, J. H. An evaluation of an anti-G suit without calf bladder. RAF Institute of Aviation Medicine, RAFIAM Report No. 96, Farnborough, Hants, England, 1970.

17. Leverett, S. D., Jr., et al. Panel: Physiological response to high $+G_z$ acceleration. Aerosp. Med. 44:351 (1973).

18. Leverett, S. D., Jr., R. U. Whitney, and G. D. Zuidema. Protection devices against acceleration. In Gravitational stress in aerospace medicine, p. 215, ch. 21. Boston, Mass.: Little, Brown, and Co., 1961.

19. Lindberg, E. F., and E. H. Wood. Acceleration. In Brown, J. H. U. (ed.). Physiology of man in space, p. 97. New York: Academic Press, 1963.

20. Lindberg, E. F., et al. Measurement of cardiac output during headward acceleration using the dye-dilution technique. Aerosp. Med. 31:817-834 (1960).

21. Michaelson, E. D. Blood oxygenation in man during high, sustained $+G_z$. Preprints of the Annual Scientific Meeting, pp. 89-90. Aerospace Medical Assoc., National Airport, Washington, D.C., 1972.

22. Milic-Emili, J., J. Mead, and J. M. Turner. Topography of esophageal pressure as a function of posture in man. J. Appl. Physiol. 19:212-216 (1964).

23. Parkhurst, M. J., S. D. Leverett, Jr., and S. J. Shubrooks, Jr. Human tolerance to high, sustained $+G_z$ acceleration. Aerosp. Med. 43:708-712 (1972).

24. Reader, D. C. An in-flight assessment of an anti-G suit without calf bladders. RAF Institute of Aviation Medicine, RAFIAM Report No. 242M, Farnborough. Hants, England, 1972.

25. Shubrooks, S. J., Jr. Changes in cardiac rhythm during sustained high levels of positive ($+G_z$) acceleration. Aerosp. Med. 43:1200-1206 (1972).

26. Shubrooks, S. J., Jr. Positive pressure breathing as a protective technique during $+G_z$ acceleration. J. Appl. Physiol. 35:294-298 (1973).

27. Shul'zhenko, Ye. B. Neuroflex mechanisms for regulation of hemodynamic changes during rapidly and slowly increasing acceleration. Kosm. Biol. Med. 1(1):45-49 (1967). (Kosmicheskaya Biologiya i Meditsina (Moskva, USSR))

28. Sieker, H. O. Effect of acceleration on the heart, ch. 9, pp. 55-56. In Gauer, O. H., and G. D. Zuidema (eds.). Gravitational stress in aerospace medicine. Boston, Mass: Little, Brown, and Co., 1961.

APPENDIX A

MODE OF OPERATION OF THE ARTERIAL BLOOD SAMPLING DEVICE.

Details of the USAFSAM arterial blood sampling device are shown in Figure A-1. With the device, blood is withdrawn from the subject's radial artery by means of a roller pump. This pump can be operated locally through an on/off control, or remotely from the centrifuge control room. The blood is pumped into a manifold to which two 10-ml syringes are connected. The dead space ("d.s.") syringe is free to move until the plunger comes into contact with an adjustable d.s. stop, while movement of the sample syringe plunger is restricted by the overriding spring. When the d.s. syringe reaches the stop, the sample syringe fills. The d.s. stop can be adjusted so that the d.s. syringe will fill with all the d.s. fluid. The volume of blood collected by the sample syringe is controlled by the length of time the control button is operated. In these experiments, the d.s. has been computed as 4.0 ml; the d.s. stop was adjusted so that 4.5 ml fluid were collected in the d.s. syringe; and, by operation of the control button for 8 sec, 6 ml blood were collected in the sample syringe.

Both syringes can be isolated from the manifold by stopcocks to ensure that air does not enter the system. The whole system can be flushed with heparinized saline which can then be emptied into the waste bottle. This flushing process is facilitated by unlatching and swinging the d.s. stop out of the way of the syringe plunger.

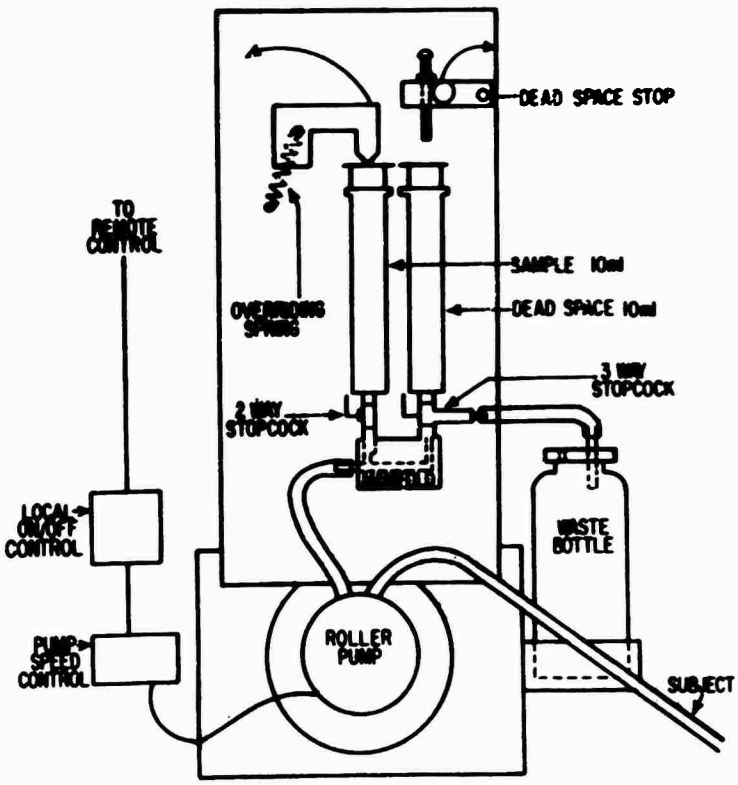


Figure A-1. The arterial blood sampling device (schema) developed at the USAF School of Aerospace Medicine by the Biodynamics Branch, Environmental Sciences Division, under the advice (oral communication) of D. H. Glaister, RAFIAM.

ABBREVIATIONS

AP	anterioposterior
bpm	beats per minute
cm	centimeter(s)
ECG	electrocardiogram
FRC	functional residual capacity
G (or) $+G_z$	positive acceleration
Hg	mercury
HIP	hydrostatic indifference point
in.	inch(es)
M-1	forced exhalation against a partially closed glottis, repeated by the subject during G
min	minute(s)
mm	millimeter(s)
msec	millisecond(s)
NA	not appropriate
NS	not significant ($P > .05$)
P	statistical probability of chance occurrence
PAC	premature atrial contraction
PLL	peripheral light loss (grey-out)
PPB	positive pressure breathing
PVC	premature ventricular contraction (beats)
R-R	interval between R waves (in ECG)
SE	standard error
sec	second(s)