NASA CONTRACTOR REPORT

NASA CR-236

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THE RESPONSE OF SQUIRREL MONKEYS TO HIGH ACCELERATIVE FORCES

by John N. Mebelas and Bruce W. Pine

Prepared under Contract No. NASw-851 *by*
SPACE/DEFENSE CORPORATION
Birmingham, Mich.
for

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION • WASHINGTON, D. C. • JUNE 1965

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FOREWORD

This final report is prepared in accordance with the requirements of Contract NASw-851, "Research on Simian and Human Response to High Accelerative Forces," between the National Aeronautics and Space Administration and Space/Defense Corporation. The Human Factors and Biotechnology Division, Office of Advanced Research and Technology, arranged for support and provided technical administration under the supervision of Mr. John Fuscoe (Code RBB). Performance was an eleven-month period starting December 12, 1963. The research reported herein is exploratory in nature and provides a survey, based on preliminary experimental evidence, of the capability of the selected subject simian -- the squirrel monkey -- to withstand high accelerative forces. Since the experimental exposures included various orientations, peak G's, and dwell times, the results of the survey are a substantial contribution to the understanding of the ability of a particular organism to survive in a dynamic environment. Due to the number of experimental cells employed it is not possible to assign statistical validity to the data. The general trends are unmistakable, however, and statistically valid results would be expected to be quite close to the preliminary results reported herein. On the basis, and in full appreciation of the complexities and lack of satisfactory scaling relationships, the possible significance of the information is considered in its application to man in similar high G environments.

This program was conducted under the general technical supervision of Bruce W. Pinc, and John N. Mehelas, M.D., was the project scientist. The report is co-authored by these investigators. A number of other individuals, however, contributed substantially to this effort. Acknowledgment is therefore made to the following: Michael Brian, of Space/Defense Corporation, inventor of the unique articulating centrifuge which permitted the program to be accomplished; Lenord Williams and Barbara B. Gordon, both of Space/Defense Corporation, whose assistance in the laboratory was essential for the actual experiments; Fred H. Gasow, D.V.M., who provided the veterinary support for the care of the animals; James R. Brayshaw, M.D., who participated in the evaluation and analysis of the electrocardiograms; Paul Wolf, M.D., Associate Professor of Pathology, Wayne State University, who is largely responsible for the enzyme study analysis, and his assistant Patricia Vazquez, who prepared the tissues for analysis; Robert Geil, D.V.M., International Research and Development Corporation, who assisted in the selection, preparation, and evaluation of the Hematoxylin and Eosin Process histopathology, and to Arlene M. Gresham, Drusilla Lundahl, Janet Dillon, and Cheryl Bond for manuscript typing. Finally, the authors especially acknowledge, with deep gratitude,

the editorial assistance of Alonzo D. Tuttle, Space/Defense Corporation, who organized, participated, and supervised all aspects of the preparation of this report with a dedication, determination and perseverance which converted a multitude of raw laboratory data into a meaningful and informative report. This document, at Space/Defense Corporation, is designated TR64-111.

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NOTATIONS

a	=	acceleration, ft/sec^2, (cm/sec^2)
A-P	=	anterior-posterior direction
AV	=	auriculoventricular
b	=	constant of proportionality
cc	=	cubic centimeter
cm	=	centimeter
C_{on}	=	onset rate (rate of change of accelerative force, ft/sec^3)
C_{off}	=	offset rate (rate of change of accelerative force, ft/sec^3)
CO_2	=	carbon dioxide
cliffhanger	=	marginal fatality of subject, expires within a few minutes post-run
D_r	=	total dwell duration of a specific run, seconds
D_t	=	time at G_o necessary to reach K_t, seconds
DPN	=	Diphosphopyridine Nucleotide - reagent
DPNH	=	Diphosphopyridine Nucleotide Hydrogen - reagent
ECG	=	electrocardiogram or electrocardiographic
f	=	function
F	=	force, pound, or gram
FM	=	frequency modulation
g	=	gravitational constant 32.2 ft/sec^2, (970 cm/sec^2)

G	=	$\frac{F}{W}$ or $\frac{a}{g}$ non-dimensional units
g, gm	=	grams
G_o	=	G stress at K_t
$+G_x$, $-G_x$	=	axes of G force, AGARD anatomical convention
$+G_y$, $-G_y$	=	axes of G force, AGARD anatomical convention
H & E	=	Hematoxylin and Eosin staining technique
J point	=	descriptive term for portion of ECG trace
K_{max}	=	maximum value for K
K_p	=	peak value for K for a specific run
K	=	total product of effects of all parameters
KC	=	kilocycles
kg	=	kilogram
K_t	=	empirical stress-weight factor at threshold,
	=	1.0×10^4 kg - G - sec.
LD	=	Lethal Dose (dosage)
m	=	number of experimental points considered
ml	=	milliliter
mm	=	millimeter
mv	=	millivolt
n	=	accelerative force units normalized to the acceleration of gravity (G units)
p	=	parameter
PIA	=	post introduction (initial) of anesthetic

P,Q,R,S,T,U	=	components of the ECG trace used to describe various cardiac activity
PMI	=	point of maximal impulse of the heart as felt through the chest wall
RPM (rpm)	=	revolutions per minute
SFAPS	=	Space Flight Acceleration Profile Simulator: articulated centrifuge model used in the experiment
t	=	time, seconds
t	=	subscript, threshold
VCO	=	variable cycle oscillator
V_2, V_3, V_4	=	standard pattern types of ECG traces, resulting from different locations of the heart electrode relative to the cardia
W	=	weight of the animal, kilograms
W_o	=	weight of the animal, at K_t
x	=	subscript, referring to x axis
y	=	subscript, referring to y axis
z	=	subscript, referring to z axis
Δ_{rt}	=	$D_r - D_t$, exposure above threshold stress level, seconds

THE RESPONSE OF SQUIRREL MONKEYS TO HIGH ACCELERATIVE FORCES

By John N. Mehelas, M.D. and
Bruce W. Pinc, M.S.
Space/Defense Corporation

SUMMARY

Two hundred twenty squirrel monkeys (Saimiri sciureus) were exposed to accelerative forces from 50 G to 430 G at increments of approximately 50 G. Dwell periods ranged from 2 seconds to 386 seconds applied in one of four axes ($+G_x$, $-G_x$, $+G_y$, $-G_y$) in an unique centrifuge. Data recorded included clinical observations, electrocardiograms (ECG), gross pathology and histochemical changes in studied tissues. From these data it was concluded that Saimiri sciureus is capable of sustaining and surviving the continuous application of $+430\ G_x$ for 115.6 seconds. The test animal also survived lower G loads for longer dwell times (i.e., $-50\ G_y$ for 386 seconds), and appears capable of surviving even higher G loads applied over shorter time bases. Differences in acceleration tolerance were observed in accordance with the direction of load application.

The pathologic response to stress appeared to differ between low (50-150 G), middle (200-250 G), and high (300-430 G) ranges. An initial statistical survey developed similar trends. Protection of the animal against certain aspects of stress was possible and extension of his tolerance to stress appeared feasible.

Cardiac events indicated by ECG changes included evidence of anatomical displacement under stress, with severe muscle noise which obscured the cardiac pattern. Clear patterns of ischemia, myocardial infarction and arrhythmias were seen, with tachycardia (to +20%), bradycardia (to -90%), and variations in amplitudes and durations of the trace elements. Other observed events included cardiac arrest (for 2-4 seconds), heart blocks, extrasystoles, flutter and other singular occurances. Dependent upon length of exposure beyond the initial ECG indication of injury, either terminal bradycardia or preliminary recovery occured. Special enzyme histochemical techniques supported the findings

from ECG and the routine Hematoxylin and Eosin (H & E) stained tissue microscopic pathological findings.

A safe anesthetic procedure was developed.

A mathematical hypothesis was developed which permitted apparent calibration of Saimiri to G stress at constant onset and offset rates. The hypothesis was tested sucessfully with numerous predictions regarding the lethality or non-lethality of Saimiri exposures. An opportunity also arose under another concurrent program sponsored by the Aerospace Medical Laboratories, U. S. Air Force, to apply the hypothesis to guinea pigs used as test subjects under contract 33(615)1893. The survival of six guinea pigs (exposed to peak G ranging from 100 G to 400 G) was successfully predicted. It is believed possible, with additional data, that extrapolation to man can be envisioned.

Finally, validation of the hypothesis may be the first step in the unification of impact data with that obtained from conventional centrifugation, eventually leading to a mathematical model fully applicable to man.

INTRODUCTION

The research into simian response to high accelerative stresses for various durations reported herein is a major event in a series of investigations in acceleration biophysics.

BACKGROUND

The staff scientists and engineers of Space/Defense Corporation have been pursuing investigations in basic and applied acceleration biophysics since mid-1961. Accident survival (1), the experiments of Beeding (2), Black-Schaffer (3), Kornhauser (4), Stapp (5), and other work (6) led us to speculate that men properly supported and protected by advanced techniques could survive much higher acceleration for longer time periods than had been previously possible. These investigators were initially stimulated by the realization that important practical applications might ensue if information (then lacking) were available on the ability of organisms to survive extremely rigorous acceleration-

time exposures. The nature of these potential applications will be discussed in greater detail elsewhere in this Introduction, but possibilities included are:

Use in manned and unmanned spacecraft systems;

Reducing personnel trauma and fatality in aircraft accidents;

Use in automotive crash injury research.

A review of the literature and the then-current state-of-the-art revealed that, of the many areas in acceleration biophysics that needed further study, the most unexplored area was that of unconventionally high (100-500 G), brief (2-10 seconds), accelerative loads. This dynamic environment had been ignored previously, but not because it was without intrinsic interest or devoid of practical potential. It had been ignored only because no device was available that could easily, accurately, inexpensively, and repetitively produce this particular dynamic environment. A review and analysis of the then-extant concepts rejected as inadequate the conventional approaches. These included sled tracks or drop towers which were limited to abrupt accelerations of less that 2 seconds duration, or conventional centrifuges which were limited to long-term accelerations of 20 seconds or more with inherent slow rates of onset and offset. Instead, a new device was designed and developed and was put into operation at the contractor's facility in Birmingham, Michigan.

A more detailed description of the machine is available in the Test Equipment section of this report, but briefly, the machine is a variable force-time profile accelerator with an articulating arm producing a 6-foot swept diameter. It is presently capable of exposing small animals to a maximum of 430 G in a minimum start to stop time of 20 seconds. Dwell at peak is unlimited.

This unique device, appropriately instrumented to record both physical and biological events during the experiment, was fully tested and then employed in a preliminary investigation of the physiologic responses of squirrel monkeys (Saimiri sciureus) to conventional and unconventional accelerative environments. The results of this study are reported in detail in the literature (7), but briefly nineteen animals were exposed to accelerative experiences on the articulating centrifuge. Thirteen tests above +300 G_x over approximately 20.0 second time bases were conducted,

of which eleven exceeded +400 G_x. One death and some injuries occurred and a characteristic general response was elicited, notable for certain neurologic and cardiovascular signs. Among the latter the most striking was apparent electrocardiac arrest with spontaneous re-initiation of rhythmic contraction.

The data obtained from these preliminary results revealed a much higher potential ability to accept accelerative stress than has been generally previously supposed, even after the most generous allowance was made for mass-to-surface ratios and scaling effect ratios when extrapolating from other simian data. The finding also tended to support our original hypothesis that man can accept and survive much higher accelerations for longer times than formerly thought possible. But before men were to be risked, prudence dictated the use of primates to gain data and develop equipment and techniques. We recognized that extrapolation of data from non-human primates to humans is unwise because of ignorance of accurate scaling relationships, however, primates were selected because of their previous use as test subjects in dynamic environment studies.

Thus, it was clear to us that this work should be pursued further and that the basic questions of simian physiological baseline response to both "conventional" and "unconventional" accelerative loads had to be thoroughly defined before more sophisticated investigations could be pursued with other animals.

Accordingly, a program of investigation was proposed to the National Aeronautics and Space Administration and on November 12, 1963, Contract NASw-851 was let to perform "Research on Human and Simian Response to High Accelerative Forces." The purpose of this report is to describe the work performed under that contract.

OPERATIONAL UTILIZATION

Assuming that over the next several years investigation validates the hypothesis that organisms can tolerate more accelerative stress than was previously thought allowable, of what practical utility is the knowledge? Perhaps it would be useful to briefly speculate upon some of the functional applications of greater acceleration tolerance.

Manned Space Systems

Escape from very large liquid fueled vehicles prior to and during launch presents difficulties, many of which are aggravated by present limitations on accelerative loads. Novel personnel ejection subsystems or powered flight of an escape module now become more attractive solutions if greater acceleration tolerance is possible. Escape from disabled orbiting space vehicles also becomes feasible using light-weight personal escape systems, if acceleration tolerance envelopes can be expanded. Of course, major reductions in manned vehicle weight can be achieved if steep re-entry angles are allowed (with concomitant increase in accelerative loads). The probable major reduction in terminal requirements for fuel, propulsion, guidance, control, and other subsystems become particularly attractive when the logistics of manned flights to Mars and Venus are examined.

Manned Military Space Systems

Personal escape modules, as noted above, now become feasible and their application in manned military systems is obvious. Here the demands of long duration operation, rapid response requirements and possible hostile action all emphasize the need for escape subsystems. In addition, certain re-entry configurations potentially attractive to the military are now more attractive if man's accelerative tolerance is appreciably improved. Also, solid-fueled boosters, with their characteristic acceleration profiles could now be more fully exploited for manned use if man's accelerative tolerance were increased. The ready-alert or rapid response potential for attack, identification, neutralization, or rescue missions is worth investigating.

Unmanned Space Systems

Biological probes carrying a wide variety of animals to investigate certain responses of organisms to the space environment are too infrequently flown. One reason is low re-entry accelerations are required so that the results of in-flight experiment will not be prejudiced by the supposedly degenerative effect of the decelerative stresses. This, in turn, penalizes the spacecraft designer who pays for the accelerative restrictions in complexity, weight, and eventually dollar costs. Better understanding of accelerative tolerances, their effects, etc., may

make simpler, cheaper bioprobes more readily available, resulting in additional valuable scientific data.

Crash Injury

Understanding of the mechanisms of improved acceleration tolerance cannot but help to improve our understanding of the traumatic events of crash injury and its prevention. The application in aircraft and automotive transport are too evident to need exposition. One aspect not usually considered is that of mass transportation, a subject becoming more and more prominent today. All the plans for mass transportation (whether it be by "private vehicles" using "public systems" or "public transportation" in the generally accepted sense) implicate high speed transportation. Doubtless, high reliability and safety will be built into such systems. But the inevitable system failures could be catastrophic to passengers unless protected by advanced protective systems that now can be contemplated if it is true that man's acceleration tolerance can be extended.

In summary, this early portion of our research investigations is intended to provide data leading to eventual resolution of two general questions:

Are the presently accepted accelerative tolerance values for man set too low?

Can the actual tolerance of man to accelerative stress be improved?

PURPOSE OF THE INVESTIGATION REPORTED

Before these questions can be definitively answered, evidently much work must be done. The short-term and long-term programs required are discussed at length in the experiment plan section. For the purposes of this introduction, however, it may be well to define the purpose of the investigation reported herein: to establish the ability of small primates to tolerate very high accelerative environments for various periods of dwell time, in various axes but with fixed rates of onset and offset.

SCOPE OF INVESTIGATION

The present investigation's scope was strictly limited to the area defined above, but clearly is related to a sequential series of investigations to establish cause and prevention of irreversible trauma and lead the way to solution of the practical operational problems outlined earlier.

RELATIONSHIP TO OTHER WORK

To our knowledge there is no similar work since the dynamic regime reproduceable on our accelerator is unique. The work reported herein assumes a dual significance because of this fact. It is unique in and of itself, but it also constitutes a "bridge" between two existing bodies of knowledge on conventional impact and long-term acceleration biophysics.

EXPERIMENT PLAN

The experiment plan used was designed with two principal objectives: first, by adhering to principles of orderly investigation, to provide that body of facts which would permit valid conclusions about the tolerance of small primates to "unconventionally" high accelerative forces; and, second, to form the base for a continuing investigation in acceleration biophysics which would allow the development of a biophysical acceleration technology around small primates, eventually extrapolatable to men in operational environments. Before describing the plan used in the work being reported, it is perhaps illuminating to briefly describe the entire primate investigation program as originally envisioned. This done, it is then possible to sketch the next (and long-term) programs which will eventually allow manned operational application.

PRIMATE PROGRAM

This primate program was envisioned as comprising three phases, the first of which is essentially complete and is the

subject of this report. The two other phases remain to be done.

Phase One - Response

This phase has as its objective determination of the ability of small primates to accept accelerative loads in a novel regime. The ability to survive, the frequency of death, the histologic and clinical events associated with survival or death are the principal entities to be measured. Phase one, the prosecution of which forms the body of this report is devoted to determining "when do they die?"

Phase Two - Cause

The objective of Phase Two is to attempt to determine the cause(s) of death in small primates exposed to the lethal regimes established in Phase One. The mode of failure of which organ system (or combination of systems) responsible for death must be determined, or "why do they die?"

Phase Three - Prevention

Once the cause of death has been established, it should be possible to devise protective measures to improve survivability. These will probably be mechanical aids, but certain physiologic, pharmacologic, bio-hydraulic or bio-pneumatic or other approach may have merit. Perhaps a combination of approaches may prove useful in supporting several of the organ systems involved. The goal is to "keep them from dying"--or at least raise the threshold again!

Additional Phases

Once lethality, cause of death, and prevention of death at a formerly lethal level have been thoroughly established, the base for additional investigation will have been laid. This should include use of larger primates in an attempt to develop accurate size-G scaling relationships, development of better protective devices, and finally exposure of humans on a man-rated centrifuge to conservatively extrapolated, high accelerative stress regimes. A series of careful experiments using men should clearly define

the acceleration envelope which men can tolerate without imposition of irreversible trauma. This known, a new set of design standards can be provided to space system designers permitting the exploration of whole new families of escape systems and operational vehicle configurations heretofore impossible.

SPECIFIC PROGRAM
PHASE ONE - RESPONSE

The experiment plan devised for the study reported herein can be found in detail in Space/Defense Corporation Report TM64-101, dated January 13, 1964, submitted to the National Aeronautics and Space Administration in compliance with contract requirements. Briefly, the goal of the investigation was to establish the ability of small primates to tolerate very high accelerative environments for various periods of dwell time, in various axes, but with fixed rates of onset and offset. Further, clinical observations, electrocardiographic (ECG) and gross and microscopic (histochemical) pathologic studies would be used on survivors and fatalities alike to record the events associated with survival and death. Two hundred twenty animals were to be used, divided into two groups: controls and experimental animals.

Control Series

Controls were of two types, procedural and anesthetic. The purpose was to establish what effect handling and anesthesia had on the subjects. One control group was handled, anesthetized, and prepared for experimentation, but not exposed to centrifugation; the second group, it was proposed, would be handled, anesthetized, couched, but permitted to recover from anesthesia, and then accelerated. This procedure was proposed based on results obtained from our prior research using intraperitoneal injection techniques.

Experimental Series

Two hundred fifteen animals were to be exposed to a variety of accelerative experiences. Each animal was to see but one experience. These were to be three controlled variables: peak G, dwell at peak G, and G orientation (or mode). Onset and offset

rates were to be maximum achievable operating as a conventional centrifuge, to eliminate the suspected contribution of "jolt" to accelerative trauma. Peak G's were to be 50, 100, 150, 200, 250, 300, 350, 400, and 430 G. Dwells at each peak were to be 0.2, 2.0, 20.0, 200.0 seconds, and one other time period chosen as a result of experimental findings. The G acceleration to be used included $+G_x$, $-G_x$, $+G_y$, and $-G_y$ (8). These AGARD Standard inertial resultants of body acceleration refer, respectively to inertial force acting on the body: from front to back; from back to front; from left to right; and from right to left -- front, back, right, and left are relative to the usual biologic frontal-saggital convention. The experimental matrix is shown as Table I.

Modifications to Plan

Minor modifications to the experimental plan were required as the program proceeded. Permission for modification was obtained from the Government in all cases. The modifications are described below.

Dwell at peak G. - Development of a harmonic vibration in the centrifuge when sustained runs above 400 G were made caused the amount of time dwell at 430 G to be curtailed in most cases. Three animals were run at 430 G, but it was unwise to make runs at the longer dwells at this maximum value.

Also, early in the program, it became apparent that it might be possible to predict survivors or fatalities by application of a mathematical relationship developed by one of the investigators. This necessitated flexibility in programming dwell at peak G as a function of the subject weight. The development of the concept, the derivation of the mathematics, and the final time-G exposures are indicated elsewhere in this report.

Matrix completion.-- Due to restriction of funds, but presented with the opportunity to explore this potentially valuable G-time-weight relationship, some of the animals in some of the matrix cells were transferred to other cells where it was deemed desirable to have larger populations. Accordingly, selected $+G_y$ and $-G_y$ samples at certain G values were not run, but never

TABLE I. PROPOSED TEST CELL ARRANGEMENT

Set No.	Peak G	G-Sec. at Dwell	Load Axis $+G_x$	Load Axis $-G_x$	Load Axis $+G_y$	Load Axis $-G_y$	Cell Totals	Set Totals
1	50	1×10^1	1	1	1	-	3	
		1×10^2	1	1	1	-	3	
		1×10^3	1	1	1	-	3	
		1×10^4	1	1	1	-	3	
		1×10^x	1	1	1	-	3	
			5	5	5	0		15
2	100	2×10^1	1	1	-	1	3	
		2×10^2	1	1	-	1	3	
		2×10^3	1	1	-	1	3	
		2×10^4	1	1	-	1	3	
		2×10^x	1	1	-	1	3	
			5	5	0	5		15
3	150	3×10^1	1	1	1	-	3	
		3×10^2	1	1	1	-	3	
		3×10^3	1	1	1	-	3	
		3×10^4	1	1	1	-	3	
		3×10^x	1	1	1	-	3	
			5	5	5	0		15
4	200	4×10^1	1	1	-	1	3	
		4×10^2	1	1	-	1	3	
		4×10^3	1	1	-	1	3	
		4×10^4	1	1	-	1	3	
		4×10^x	1	1	-	1	3	
			5	5	0	5		15
5	250	5×10^1	1	1	1	-	3	
		5×10^2	1	1	1	-	3	
		5×10^3	1	1	1	-	3	
		5×10^4	1	1	1	-	3	
		5×10^x	1	1	1	-	3	
			5	5	5	0		15
6	300	6×10^1	2	2	1	1	6	
		6×10^2	2	2	1	1	6	
		6×10^3	2	2	1	1	6	
		6×10^4	2	2	1	1	6	
		6×10^x	2	2	1	1	6	
			10	10	5	5		30
7	350	7×10^1	2	2	1	2	7	
		7×10^2	2	2	1	2	7	
		7×10^3	2	2	1	2	7	
		7×10^4	2	2	1	2	7	
		7×10^x	2	2	1	2	7	
			10	10	5	10		35
8	400	8×10^1	2	2	2	1	7	
		8×10^2	2	2	2	1	7	
		8×10^3	2	2	2	1	7	
		8×10^4	2	2	2	1	7	
		8×10^x	2	2	2	1	7	
			10	10	10	5		35
9	430	8.6×10^1	2	2	1	2	7	
		8.6×10^2	2	2	1	2	7	
		8.6×10^3	2	2	1	2	7	
		8.6×10^4	2	2	1	2	7	
		8.6×10^x	2	2	1	2	7	
			10	10	5	10		35
							Total Task	210

both: either + or - at any level (except 430) was run to provide exemplar data, once it had been shown that $+G_y$ and $-G_y$ results did not differ appreciably. This shifting in cell population is reflected elsewhere in this report.

Additional Investigations

In the course of the program, several monkeys died during the holding period prior to experimentation. These animals presented an opportunity to examine the organs after natural death; this was accomplished to provide a small sample of indicators for comparison to the results due to accelerative stress. These natural deaths were replaced later with acceptable specimens.

To provide data helpful in later work toward determining extrapolative and other scaling factors, the tail weight of 139 of the subjects was recorded. Additional data on morphology required to provide satisfactory restraint was obtained on eight of the subjects, early in the program. These data are summarized elsewhere in the report.

TEST PROCEDURES

Reproducible experimental results required an orderly conduct of daily procedures as well as a knowledge of and a familiarity with the peculiarities of the test animal, the equipment to which it would be exposed, and the animals' reaction to the test environment. Appendix B lists the chronologic events of our actual daily test run schedule. Examples of forms required and utilized in the detailed conduct of each experiment are also displayed in Appendix B.

The remaining aspects of the test procedures have been arranged into the following separate categories and are discussed in the following sections:

The Test Animal

The Test Equipment

Animal Handling Procedures

THE TEST ANIMAL

Saimiri sciureus, the squirrel monkey, is classed among the primates of the New World. This section describes the animal by briefly discussing these salient points:

Place among the primates

Distribution and taxonomy

Behavioral characteristics

Motile activities

Captivity

Pathology and husbandry

Physiological data

Place Among the Primates

Briefly, the definition, classification, and history of S. sciureus are reviewed below.

Definition of a primate. - The range of structure displayed by the primates is so great, their origin so ancient, and their subdivision so extensive that an inclusive definition of all of them can be provided only by the precise terms of Dr. St. G. Mivart:

> Unguiculate, claviculate placental mammals with orbits encircled by bone; three kinds of teeth, at least at one time of life; brain always with a posterior lobe and calcarine fissure; the innermost digit of at least one pair of extremities opposable; hallix with a flat nail or none; well developed caecum; penis pendulous; testis scrotal; always two pectoral mammae.

Primate classification. - There are about 750 known kinds of living primates of 244 species grouped into 80 genera which in turn, fall into the following 12 fairly well-defined major divisions. These divisions correspond roughly to families in the technical sense, but they are not all exactly of that status from the taxonomic point of view and at least one, that of the pithecoids is, according to current scientific acceptance, a composite assemblage. The living primates may be summarized (9) as follows:

1. Tupaioids, 2. Lorisoids, 3. Lemuroids, 4. Tarsioids, 5. Hapaloids, 6. Pithecoids, 7. Ceboids, 8. Cercopithecoids, 9. Cynopithecoids, 10. Coloboids, 11. Simoids, 12. Homonoids.

New World monkeys.- The Ceboids (10), which include the Squirrel Monkey, are the monkeys of the New World. The cytogenetics of this group have been studied by Bender and Mettler, 1958 and by Bender and Chew. However, the paucity of ecological and behavioral studies on this important group of primates, with the exception of the work of Carpenter and his studies on the howler monkeys of Panama, makes it difficult to comment on the adequacy of the distinction made in the past among the various species and genera. It is also very difficult to consider the history of any contemporary Ceboid groups, because the fossil record is so poor; indeed, it is almost non-existent. A rather large gap in our knowledge of the primates occurs in the material available for assessing the relationship of the Prosimii to the South American Ceboidea. The fact that the South American primates are considered to be of the monkey grade of organization, and are called monkeys, has stressed a probably spuriously close relationship between Old and New World forms.

Contemporary thought generally concedes that the Ceboids originated from a different group of Prosimii than that from which the Old World monkeys sprang. The similarities between the two groups are more apparent than real. It is, indeed, for this reason that the Ceboids are potentially such an important group of monkeys for students of primate evolution. The independent development, in a second primate stock, of structures and functional adaptations, though often exactly parallel functional adaptations of the Old World monkeys, is a datum about primate evolution that has not received proper consideration. It may also have bearing upon similar parallelisms in functional adaptation seen in Hominaedi, which may be of particular interest to the present investigators. The test animal in these series of experiments herein reported is a Ceboid and its peculiar name is Saimiri sciureus, or as it is commonly known, the Squirrel Monkey.

Distribution and Taxonomy (11)

The animal is distributed over an extensive region of the South American continent. Four, possibly five, distinct species of Saimiri may be recognized at the present time, some possessing minor color variations. It is possible that some of the species

may, with further knowledge, be reduced to the rank of subspecies. The various forms may be primarily divided into two groups depending upon whether the crown is or is not concolorous with the back. When the crown is black, the specimen may be from the group containing the Central American forms (oerstedi) or the southern race (boliviensis). Between these two extremes lies the wide territory occupied by the sciurea group characterized by the crown and back being approximately the same color.

The following key serves to distinguish the five potential species provisionally recognized.

Group I, sciurea group.- Head more or less concolorus with back; posterior border of palate simple, malar foramin normal in position, zygoma weak: head grayish (sciurea); head bluish-gray (madeirae); head golden yellow ticked with black (usta).

Group II, oerstedi group.- Head not concolorous with the back, but black or blackish; malar foramin near orbital margin of the maxilo-malar suture, zygoma short and broad: back reddish (oerstedi); back grizzled grey (much as in sciurea) (boliviensis).

Behavioral Characters

Most of the information available (9), (10), (11), relates to the common S. sciureus. Little attention is paid to the distinctions of species in the literature involving experimentation. The squirrel monkey is a highly excitable, noisy, and rather aggressive little monkey-like primate that lives in huge tribal units along river banks throughout an enormous area of North and South America. Squirrel monkeys are extremely active, forever on the move to the point of restlessness. They are highly and almost entirely arboreal; running, leaping, and climbing among the forest trees and creepers which line the banks of the waterways in the Guianas and Amazonia. They live in very large associations up to several hundreds, as many as 550 being seen together in Guiana. The troops are, moreover, very numerous, hardly ever less than two troops being visible at once. Squirrel monkeys are thus probably the commonest primates in South America, the ones most frequently seen both in the wild state and in captivity. In range they are confined to the immediate neighborhood of the riverbank and the edges of forest tracts in the savannah areas. They are not found outside the belt of forest beyond a few hundred yards from the riverbanks. Their ecological niche is thus a specialized

one -- festooned with creepers bearing flowers, fruits, and other edibles upon which they subsist. In eastern South America they reside in the Avicennia scrub. Within this tangle, definite roadways are produced through constant use by troops of monkeys leading within to the shady regions of the forest and upwards to the tops of the highest trees which rise between 80 feet and 100 feet on the fringes of the main forest. An occasional ascent in search of insects also occurs. Along the trackways the troop usually proceeds in single file following a leader. Single file is always used in leaping a chasm between neighboring masses of creepers.

Motile Activities

Progression is truly quadrupedal and, even when resting, support is normally by both pairs of limbs, the hands being relieved only during feeding or in handling foreign substances. Erect posture, however, is attainable, the body being balanced in these circumstances by the stiffened tail serving as a third limb of a tripod. The tail is an organ of considerable importance in the squirrel monkeys as may be inferred from its relatively great length and thickness. Though not in any way prehensile, it is exceedingly mobile and an organ of which the owner appears acutely conscious at all times. Besides its normal use as a balancing organ and as a temporary support in the resting animal, it is also used as a wrap or boa, being flexed between the legs and then spirally over the back or shoulders. The lips, though very mobile, are not characterized by much power of protrusion. The ears are large and in some species adorned by hair tips. They have highly distinctive vocabularies and make noises unlike those of any other animals. Fear and anger are expressed by a wide opening of the mouth accompanied by shrill, protracted vocal emission. In sleep squirrel monkeys huddle together and there is much squabbling for a central position in the party, presumably to escape the chill of tropical nights.

Diet

Though subsisting principally on a diet of fruit, nuts, buds, and similar vegetable fare, protein seems to be an essential element in the natural menu. Deprivation of protein in captivity will speedily result in ill health. Most commonly, insects including flies, butterflies, and mosquitos and also spiders sup-

ply the need in the wild state. Small birds, live or freshly killed, are also eaten. The animals need a great deal of water in captivity because dehydration is rapidly fatal.

The alimentary transit is rather rapid. Feces are solid, pultaceous, and formed. In color they vary according to the diet: bright yellow when mainly fruit eating; darker when plenty of insectile food is provided. Defecation takes place over the edge of their perch which thus frequently becomes foul.

Captivity

Squirrel monkeys are frequently tamed and kept as pets by the natives of South America and consequently they are among the commonest neotropical monkeys transported alive to menageries and pet shops, where their gentleness (when tamed), liveliness, large liquid eyes, cheerful voices, and other attributes make a ready appeal. In the London Zoo, Mitchell, 1911, gives the average length of life based on 44 individuals as nine months. The longest duration, nine years, was quoted by Flower in 1931. However, other researchers have shown the average life span to be two years. There is one record of a female being kept for 20 years. She had been treated as a pet although many observations of a nutritional study were made on her; she was maintained in health by vitamin D and ultraviolet light treatment. At necropsy she was thin and showed senile changes in the heart, blood vessels, and kidneys; granulose cell tumors were discovered in both ovaries as were multiple uterine fibromyomata.

The monkey has an intense interest in strange objects and has been noted to exercise this inquisitiveness by several researchers.

Emotions are expressed vocally and by facial movements. Eyes are particularly expressive. Exercise of the orbicularis oculi is noted when the animal is scrutinizing strange objects or when frightened. Anger is shown with open mouth and display of teeth, and high pitched vocalization. One researcher found an upset animal's eyes to be filled with tears and described the phenomenon as true weeping.

Squirrel monkeys, particularly the young, display an inordinate fondness for hairy objects. In the absence of members of their own kind, foreign objects will be adopted.

Pathology and Husbandry

Squirrel monkeys are very susceptible to cold, especially if kept singly, which precludes mutual warming and protection received from companions. They are consequently extremely liable to respiratory infections and this is particularly so in a dry atmosphere even if not unduly cold, the explanation being the desiccation of the respiratory mucosa. A humid atmosphere, even if not particularly hot, is, therefore, more favorable than a hot, dry one.

Although platyrrhine monkeys are less susceptible than Old World species to tuberculosis, the present genus lacks the immunity of the marmosets. When present, however, the disease is abdominal or generalized rather than primarily pulmonary. This suggests the bovine bacillus as responsible and therefore presumably the route of invasion is alimentary. Doubtless, young animals are reared by natives on infected cow's milk prior to export, explaining such occurrences rather than infection after their arrival in the dealer's hands.

Presumably, so-called "Monkey Virus B" or Simian Herpetic Encephalitis is pandemic in these animals; they are treated with the care accorded the carrier of a frequently fatal disease.

Pyroplasmosis, and various forms of helminthiasis are the commonest parasitic diseases. The former induces symptoms similar to human malaria and is amenable to anti-malarial drugs. Helminthic diseases include filiarisis, intestinal helminthiasis, as well as infestation with the lung form. They induce a severe anemia in captive animals especially when the diet is deficient. Intestinal worms include the nematode and the acanthocephalid. Vermifuges do not always improve the condition of the host which normally lives in harmony with the worms. Indeed it often seems to produce deleterious effects. Proper attention to dietetic requirements seems to be preferable to chemotherapy, especially giving the animals access to spiders or other invertebrate sources of protein or merely the occasional administration of a small live or freshly killed bird. Dietary deficiencies probably account for the development of sores in the tail, rump, and extremities so frequently met with in captive specimens. But infection from soiled perches may account for some of this.

TABLE II

SQUIRREL MONKEY PHYSIOLOGICAL DATA

	Sex	Body Weight in gms.	No. of Measure-ments	Body Temperature in °F		Respiratory Rate		Heart Rate	
				Mean	Range	Mean	Range	Mean	Range
1.	F	324	22	98.1	96.6-100.4	66	55-110	276	260-360
2.	M	375	19	100.1	99.0-103.4	78	64-112	292	250-330
3.	M	498	25	100.0	97.6-102.8	54	40- 92	242	180-300
4.	F	368	23	98.8	97.5-100.1	63	50- 80	230	180-300
5.	F	490	21	98.9	97.4-100.6	67	60- 80	290	270-320
6.	F	332	20	97.6	96.5- 99.2	72	60- 84	264	230-310
7.	F	339	17	102.8	101.1-104.8	106	76-160	242	220-300
8.	F	376	22	99.4	97.7-102.2	61	50- 88	243	200-290
9.	F	495	24	100.9	99.0-102.6	76	64- 84	245	220-280
10.	M	349	25	98.3	95.2-102.4	51	40- 60	185	140-260
				99.5	95 -105	68	40-160	250	140-360

AFTER BEISCHER

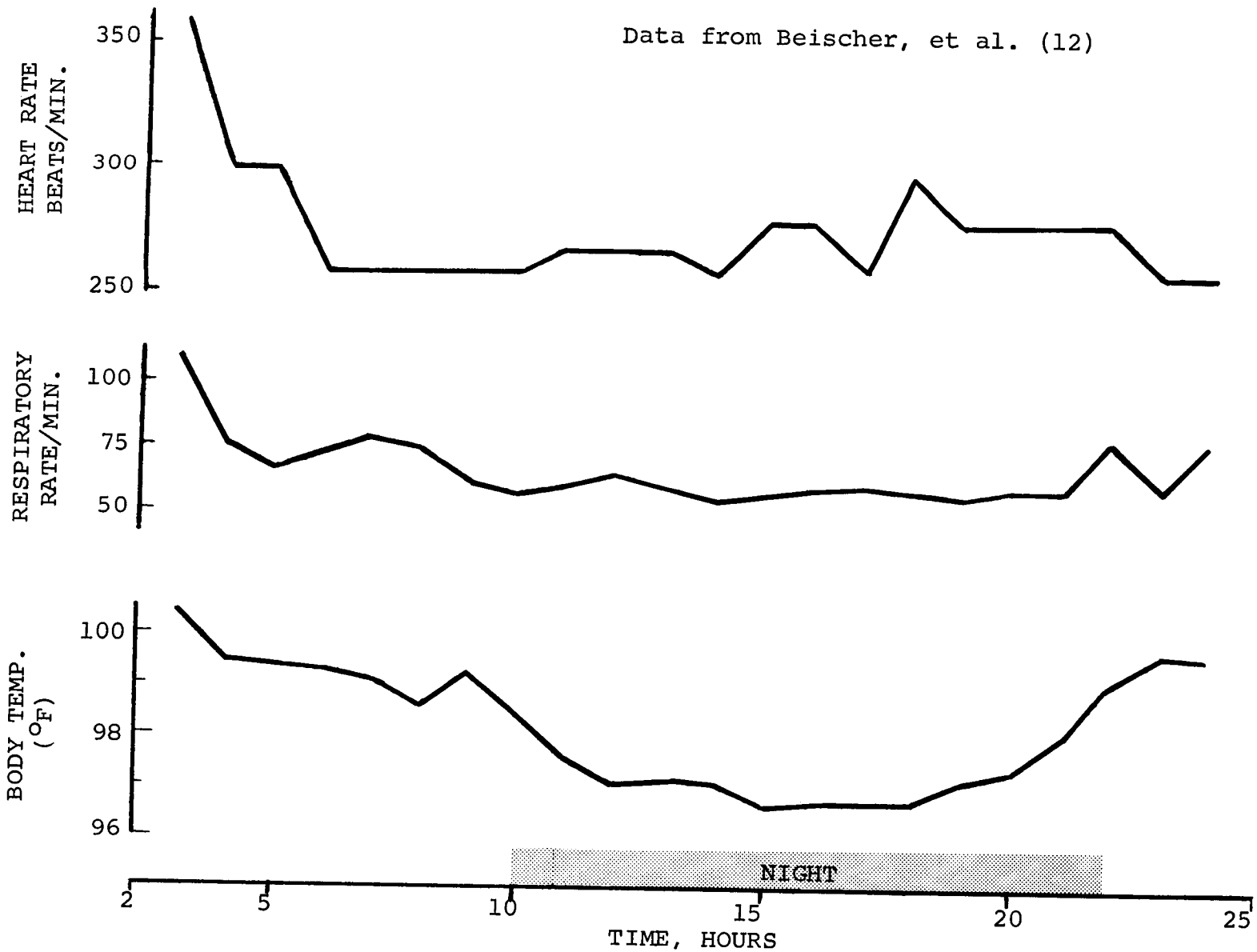

FIGURE 1. VARIATION OF PHYSIOLOGICAL MEASUREMENTS ON ONE MONKEY

Physiological Data

Detailed information on basic physiological parameters of S. sciureus is not readily available in the literature. Table II and Figure 1, drawn from Beischer's paper (12)on the animal, are among the best information on the subject, thus are included as supporting data for comparison to these experimental results.

TEST EQUIPMENT

The equipment used in the conduct of these experiments was designed, developed, and installed in our laboratories during previous privately sponsored research programs. Although much of the installation comprises combinations of commercial research equipment, critical items are unique and singular in construction and design. The test system, comprising an articulated centrifuge, instrumentation, data reduction, and control devices is described briefly below, including some of the salient information gained about performance of these devices during the experimentation.

Centrifuge

The test subject is couched and encapsulated in the payload capsule of the articulating centrifuge. The purpose of the machine is to permit investigation of the effects of variable inertia forces in conjunction with rapid and controllable onset rates, both being independently variable.

Comparison of devices.- The conventional centrifuge with the same power sources could achieve peak forces of the same magnitude, but would be severely limited in the choice of onset/offset rates. The impact testing machine, on the other hand, can achieve much greater onset rates but is severely limited in time of application. Additionally, the accuracy of the impact machine is not entirely or consistently predictable.

While the need for both the conventional and the impact type machines is obvious, there is a wide disparity between the data obtained. Because of this, an attempt has been made to design a machine that is flexible enough to fulfill both functions by spanning the entire range between the two types. In practice, the machine cannot compete with the impact type accelerator for very

abrupt accelerations; however, it exceeds the capabilities of the ordinary centrifuge by a considerable margin.

Description of prototype model.- A prototype model, employing the articulating concept, has been constructed and is called the "Space Flight Acceleration Profile Simulator" (SFAPS). The device is 6 feet in diameter, weighs about 2500 pounds and is powered by a 20 horsepower direct current motor. The whole structure is situated in a pit 10 feet square by 6 feet deep. The pit is partially enclosed at the top by steel plates leaving an octagonal opening about 5 feet across through which SFAPS can be observed.

SFAPS, shown in Figure 2, consists of two primary arms, two secondary arms, payload capsule, a balancing capsule (at opposite end of secondary arms), controls, instrumentation, and recording equipment. The primary arm rotates about the hub of the complete system (primary axis). The secondary arms are attached to the outer end of the primary arms via the secondary shaft. It is possible to rotate the secondary arms independently of the primary arms. Each arm has been balanced so that rotation of both, or either, will not affect the radius of gyration. The payload capsule is also free to rotate under certain conditions. Primary and secondary arm lengths are equal. The machine can be operated by one of two modes:

1. As a conventional centrifuge;
2. As an articulated centrifuge.

Conventional operational mode.- To operate as a conventional centrifuge, the two sets of arms are aligned and locked together with the payload capsule outermost (i.e., at periphery of rotation).

Articulating operating mode.- To operate as an articulating centrifuge, the two sets of arms are aligned and locked together with the payload capsule innermost (i.e., at the primary axis). Starting the motor causes the primary and secondary arms to commence rotation as a single integral unit. By applying a brake to the payload capsule, the system is constrained to rotate around the capsule; the capsule is held stationary by virtue of the brake torque. In this way it is possible to use a minimal power source since the time taken in increasing angular velocity of the system does not affect the stationary payload. When the desired rotation rate is attained, the payload capsule is spun-up in a few seconds to the same rate as the arms, but remains locked at the primary axis. The spin-up of the capsule is a two-phase operation: First,

LEGEND

1. Support Structure
2. Secondary Arms
3. Secondary Axis
4. Control Mechanisms
5. Primary Axis
6. Payload Capsule
7. Primary Arms
8. Drive Gear Box
9. 20 HP Electric DC Drive Motor
10. Mounting Base

Payload Capsule shown at Center of Rotation of Primary Arms.

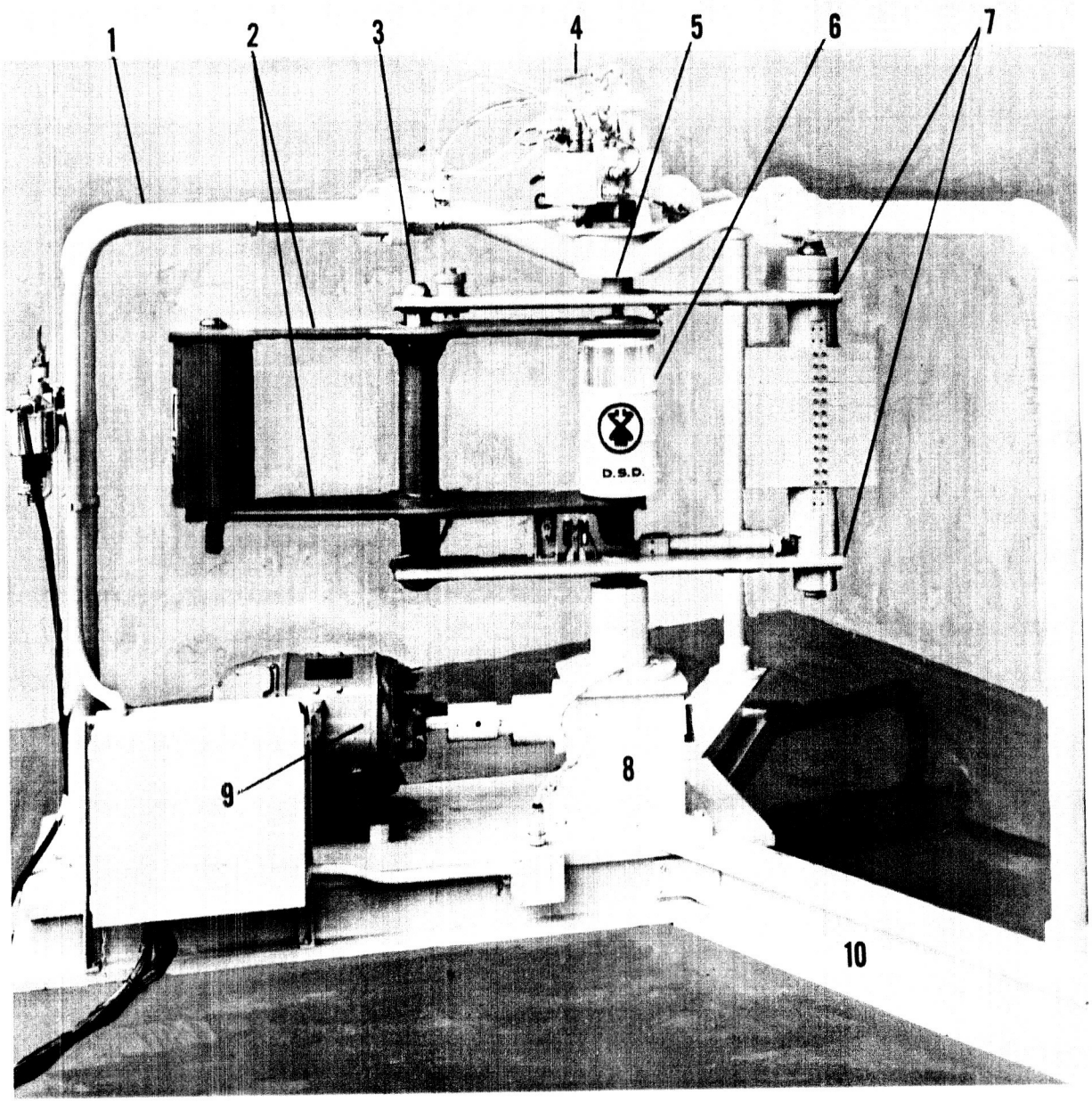

FIGURE 2. PHOTOGRAPH OF THE SFAPS

as indicated, the capsule must be spun-up to the same speed as the arms, or, stated in another manner, it must be brought to rest relative to the secondary arms. Second, in stopping the capsule relative to the secondary arm, it must be precisely oriented. This insures the payload will experience the inertia loads in the desired direction during the independent rotation of the secondary arm. Immediately following the speed synchronization and orientation of the payload capsule, the secondary arms are unlocked from the primary arms. The aerodynamic drag acting on the system will cause the secondary arm to rotate independently about the secondary axis. As the payload capsule traverses the space from the primary axis to the periphery of rotation, so the centrifugal force increases in direct proportion. The payload capsule is oriented by a 2:1 gearing system) so that it maintains a fixed attitude toward the primary axis. This insures that the inertia forces experienced do not vary in direction relative to the payload. A more complete description of SFAPS is given in Brian's paper (13).

The prototype model presently in operation does exhibit some faults which prevent its effective use as an articulating device. The faults are of a mechanical nature and can be corrected when the pending modifications are incorporated. In the meantime the machine is operating as a conventional centrifuge with the following onset (C_{on}) and offset (C_{off}) rate characteristics:

$$C_{on} = n^{.595}$$

$$C_{off} = n^{.65}$$

Where C_{on} and C_{off} are the mean rates and n is the peak G force.

In view of the high resilience exhibited by the test subjects, it was considered advantageous to operate as a conventional centrifuge to achieve the necessary extended time base.

Instrumentation

The instrumentation system must gather, store, and display data describing the physiological status of the test subjects during experimental periods. These data must be produced prior to, during, and after exposure to the accelerative forces produced by SFAPS, then related to the forces on the animal. The two parameters measured were electrocardiac activity and accelerative force.

Capsule equipment.- The payload capsule contains ten silver-coated slip-rings; seven are required for the two accelerometers. One accelerometer is mounted radially and the other tangentially to the line of accelerative force on the back of the animal couch. Experience has shown that the tangential accelerometer has little value and its use has been discontinued. The reason for omitting the second accelerometer relates to the characteristics inherent in the design. The cross axis sensitivity of the strain-gage type used is $\pm 1\%$, thus the tangential accelerometer is swamped by the radial force and registers an erroneous value. Other types of devices have no better cross-axis sensitivity.

The remaining rings are used to process the physiologic data, utilizing F.M. telemetry land link principles. The signal from the animal is picked up through three special electric conductive jelly-filled electrodes constructed to fit the test animal. Figure 3 shows the design of these devices schematically, developed during prior research specifically for this subject. The signal is amplified and multiplexed by the couch-mounted electronic components, then fed through a single slip-ring. The other two rings are used for electronic power supply and common. Although the electronic components have been exposed to many cycles of G stress, including 430 G for over three minutes per cycle, no difficulty has been experienced with the modules or components.

Initially, it was desired to record both an electrocardiogram and the heart sounds. However, the heart sounds were difficult to separate from other extraneous physiologic and mechanical noises, although a working system had been devised. Therefore, it was decided to abandon this particular parametric measurement in favor of a second ECG trace. Thus, two ECG's can be recorded.

F.M. telemetry link.- The multiplexed physiological signals and the acceleration information are fed through approximately 20 feet of coaxial cabling to the instrument and control console of the system. The accelerometer signal is amplified through a Honeywell two-channel carrier-amplifier system, containing its own calibration and regulated power supply for excitation. The carrier-amplifier, operating at a frequency of 5,000 cps, drives the recording galvanometer 8 inches peak to peak up to 1,000 cps, recording any transient phenomena that may occur in the SFAPS system. The ECG signals are demultiplexed by subcarrier discriminators, with an accuracy of $\pm 0.1\%$, one using a passband of 50 CPS, the other a passband of 110 CPS. Two ECG traces were

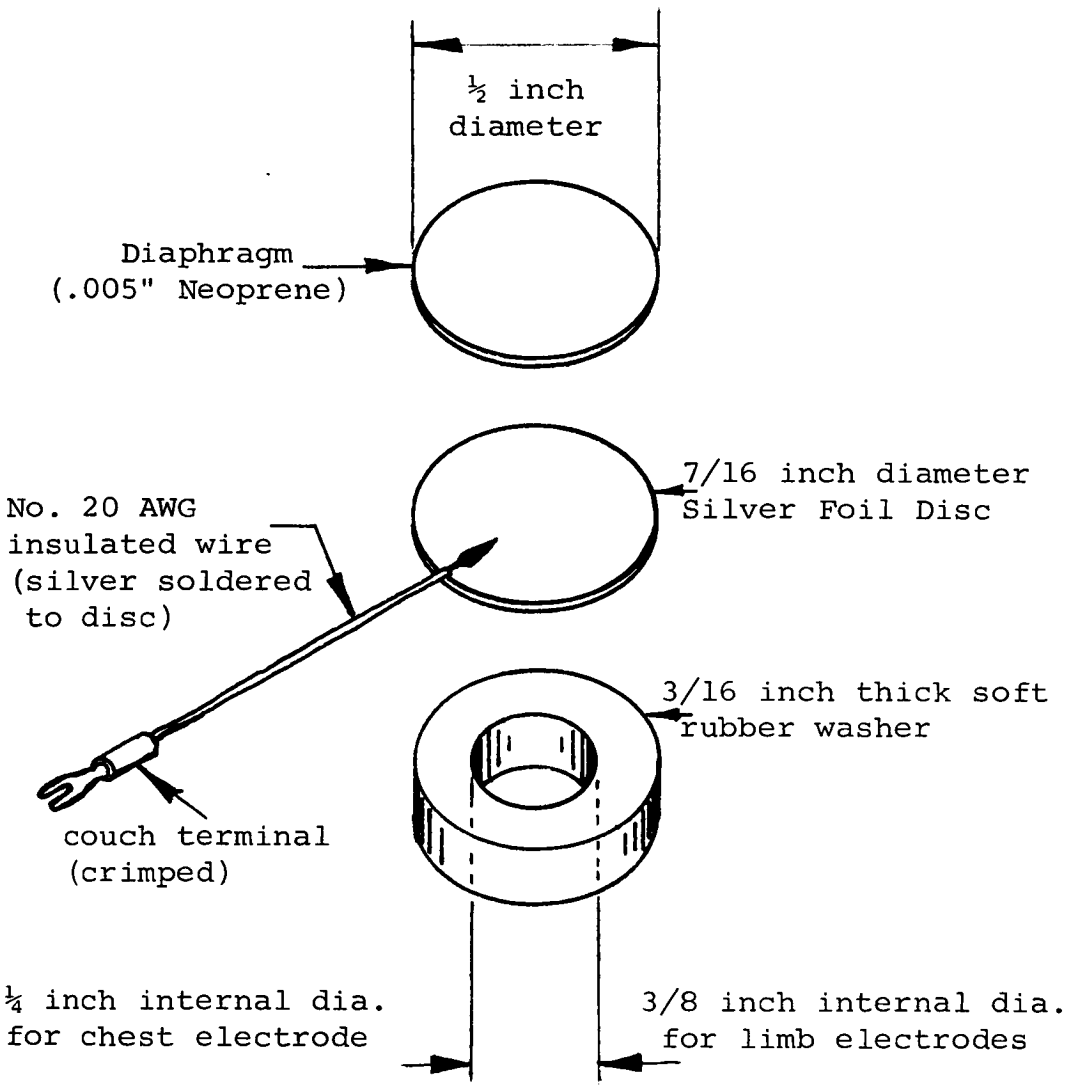

FIGURE 3. ECG ELECTRODE-EXPLODED VIEW

recorded, the first being a mirror image of the second except for the electronic filtering used. The primary image is reversed from the normal ECG pattern.

Data reduction system.- The data generated is demultiplexed and displayed as five channels of information on a multi-channel oscillogram recorder. Although not used for these experiments, it is possible to record all of the data, either real time or multiplexed for later read-back, on a multichannel magnetic tape recorder. The physiological data was monitored on an oscilloscope utilizing a dual-trace plug-in pre-amplifier. A schematic of the electronic data system is shown in Figure 4.

The oscillogram record shows the following traces:

1. Primary ECG recording, 50 cps passband; reverse image of normal ECG trace;

2. Transverse accelerometer recording (not used, straight line only);

3. Secondary ECG recording, mirror image of Trace 1, except filter 110 cps passband (normal ECG position);

4. Radial accelerometer recording, setting 50 G/cm;

5. Event marker, run, start, and stop.

In the $-G_x$ mode the reversal of the signal from the accelerometer necessitated relocating Trace No. 4 to a different galvanometer above Trace 1 on the paper, so the deflection of the trace would remain within the borders of the paper. The oscillogram record also includes fixed horizontal (length of paper) reference lines, 2 mm apart, with the centimeter (5th) lines accented and vertical (across the paper) time lines spaced one second apart. Although other timing intervals were available, this time interval was unchanged through the study. By reference to the horizontal grid, the excursion of a trace can be measured; by reference to the time lines, the temporal relationship of events can be measured. Changes in paper speed simply compress the trace, with the time lines remaining one second apart. However, the time lines have an accuracy within $\pm 2\%$ and, therefore, over a period of minutes some difference between real time and recorded time may be apparent. The event marker and test blip on the acceler-

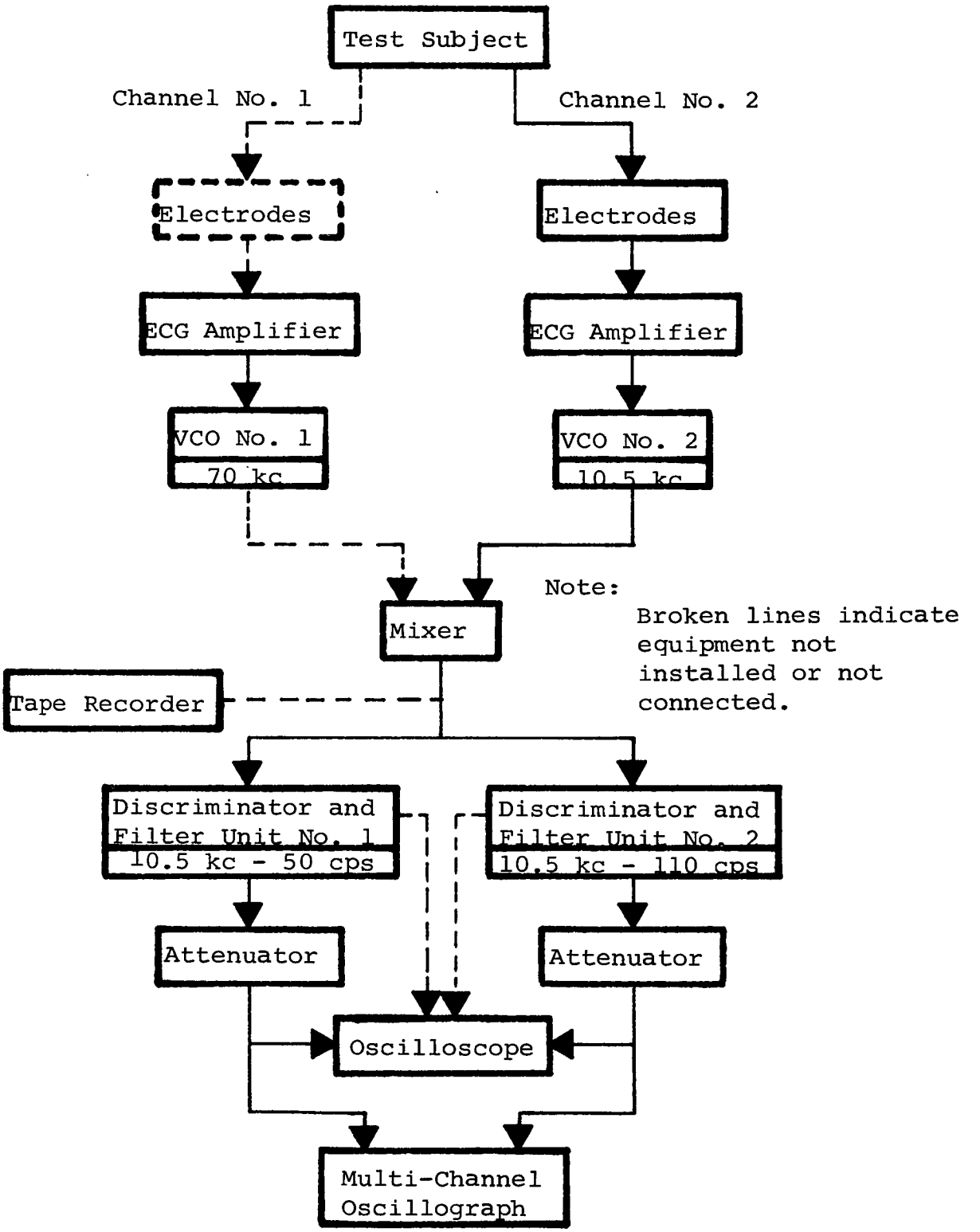

FIGURE 4. BLOCK SCHEMATIC - PHYSIOLOGIC DATA GATHERING AND RECORDING SYSTEM

ometer trace were used, in conjunction with a timer, to record events in real time as scheduled providing points at which time line errors can be determined. Selected ECG traces are shown in Appendix C.

The demultiplexed ECG signals were amplified through a galvanometer amplifier with adjustable gain so that adequate signals could be displayed. However, once set during baseline checks, the gain was not readjusted throughout the event. The usual setting was 10 millevolts per 3 centimeters (10mv/3cm) but when needed 1 mv/cm was used. On several occasions, other settings were used.

Peak G control.- When adjusting the centrifuge for the correct RPM to obtain the selected peak G, the initial setting of the speed control is made by reading a tachometer, the final adjustment by use of an electronic counter sensing the actual machine speed through a magnetic pick-up mounted in the gearing. It was found that the motor speed control could be marked by trial, so that the final adjustment to peak G would be less than one percent of the desired value. Although this is an open loop system, the "wow" and drift experienced have been negligible. The radial accelerometer trace is not used as a form of speed control or limiting value since the accuracy of reading the trace is much less than the digital read-out of the electronic counter.

It was found impossible to utilize the identical setting of the speed control from day to day to obtain repeatable rotational speeds. Each day, therefore, and again after a change in axis, the control setting was checked for each level to be run and recalibrated. Thus, assurance that the test subjects would be exposed at the desired value of accelerative stress was obtained.

Safety Precautions

Procedures developed in previous research were followed to guard against inadvertent mishaps. The inherent inertial forces involved in this type of experimentation had occasioned the installation of several interlocking safety controls.

The access to the centrifuge pit is equipped with switches to remove power from the control panel. It is thus impossible to start the machine until the area is cleared and control transferred. The last occupant of the pit was required to check the area for loose equipment and manually throw a switch before leav-

ing the area. With this switch closed the pit access door switch would allow operation from the panel.

Entrance to the experimental area was restricted without escort during the tests. A red caution light outside the entrance, controlled from the console, indicated tests in progress. A further precaution installed on the panel is a locked "stop" switch which requires a key to deactivate. In this manner operation of the machine is restricted to the persons to whom keys are issued.

ANIMAL HANDLING PROCEDURES

A regular routine was followed in the handling of animals throughout the course of this experiment. The animals were but recently captured and were not conditioned to experimental routines. After an initial short period of quarantine at the supplier, these animals were subjected to de-worming procedures. Six to nine animals would be shipped in a gang cage to our animal-holding facility managed, under contract, by a licensed veterinarian.

Holding Procedures

From the gang cage the animals were transfered to individual squeeze cages. Each cage had its own individual water and food supply containers attached. A regular feeding and watering schedule, as well as observation schedule, was maintained. Purina monkey crackers, bananas, and grapes were fed, with water ad libitum. The animals were ordinarily held at the veterinarian holding facility several days prior to their utilization in any given experiment.

Animal maintenance.- Usually the squirrel monkey survived this non-jungle environment. Since the animals were not acclimated to Michigan weather, and a major portion of this experiment took place in the winter months, several problems in animal maintenance became apparent. During one disastrous episode an entire shipment of squirrel monkeys contracted and expired from pneumonia because of a sharp overnight change in weather conditions from quite warm to sudden chill. Several other animals contracted pneumonitis at the veterinarian holding facility; they succumbed to the pneumonitis in rapid and dramatic fashion.

Some animals seemed prone to develop skin abscesses of the periorbital tissues. Such a development in these animals proceeded to death within a matter of a day or so.

Transfer Procedures

For such reasons, then, as the susceptibility of this animal to pneumonitis with the slightest changes in the weather, special precautions were taken in the handling of the animals involved. During the winter months a station wagon, preheated, was employed to transport the squirrel monkeys from the animal-holding facility across the street to the laboratory. Enough animals were brought over each day to complete that day's program of centrifugation and/or autopsy. The procedure was reversed for returning the animals after experimentation.

Health Survey

Each animal was subjected to an initial health survey prior to centrifugation where general appearance and conditions in the head, eyes, ears, nose, and throat, thorax, abdomen, limbs, and tail were noted. Also noted was activity in the cage such as normally active behavior, passive behavior, excessive huddling of the monkey (sick monkey position), or aggressiveness when provoked. The animal's food and water intake were noted as to whether or not they were grossly normal. Stools were noted as to coloration. Other abnormalities were also noted. The chart form used to record these observations is shown in Appendix B.

Preparation

With the operator wearing heavy animal-handling gloves, the squeeze panel would be slipped toward the front of the cage, forcing the animal out of the squeeze cage and into the specially constructed anesthesia box. The animal was then taken to the weighing station where the weight of the animal was recorded in grams.

Anesthesia and identification.- A measured amount of ether anesthetic, according to the predetermined dosage schedule, was then poured on a gauze pad and dropped into the anesthesia box containing the animal (Figure 5). After anesthetization, the

FIGURE 5. - ANESTHETIC ADMINISTRATION

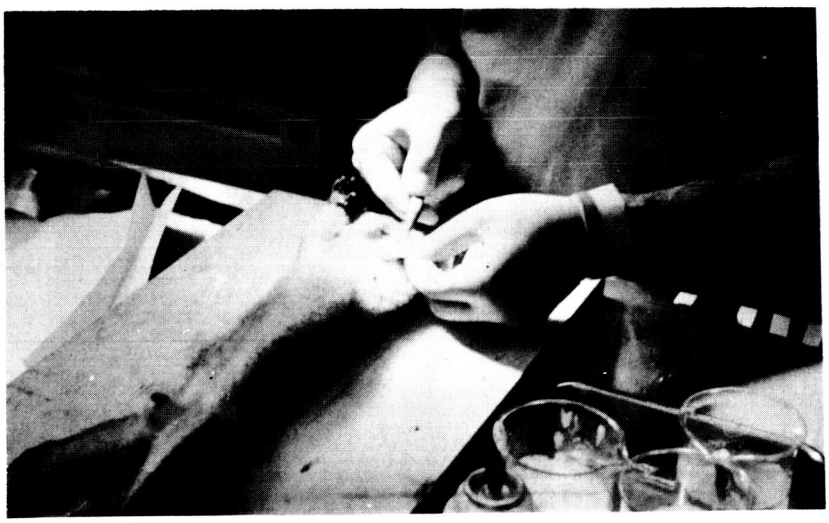

FIGURE 6. - EAR TAG INSTALLATION

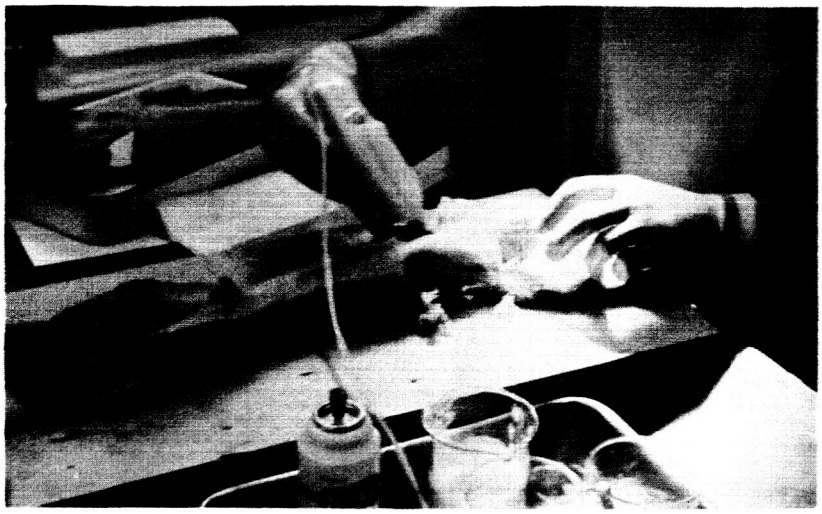

FIGURE 7. - SHAVING CHEST - BEAKER OVER MUZZLE

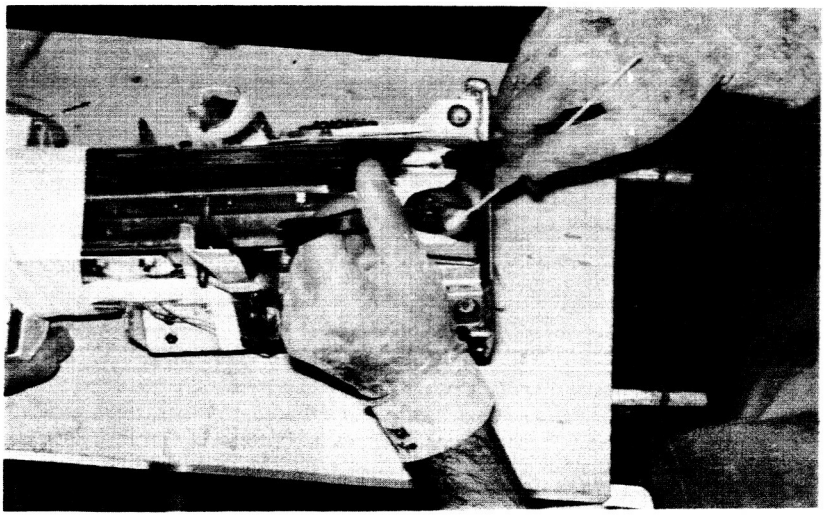

FIGURE 8. - CLEARING AIRWAY AFTER COUCHING

animal was removed from the box and transferred to the animal preparation board where the animal was placed in the supine position and its four extremities were tied to individual pegs. A numbered identification tag (Figure 6) was placed in the ear and recorded.

Electrode placement.- An electric shaver was used to remove the fur from the animal's chest, left lower leg, and right forearm. See Figure 7. These bare areas were then scrubbed with 20% soap solution and residual fuzz was removed with a safety razor. The newly shaved areas were then sponged, cleansed with 70% alcohol solution, and allowed to air dry. Small discs of adhesive tape, 2.5 to 3.0 cm in diameter, were then placed on the outside posterior of the lower left leg, inside the right forearm, and over the point of maximal impulse of the heart on the anterior chest wall. These areas were sprayed with a plastic surgical dressing (Vibesate). When the dressing had dried the discs were lifted off with forceps, exposing a small circular area of bare skin. Control of the animal's anesthesia state was maintained by inserting the original ether soaked gauze pad from the anesthesia box into a 150 ml beaker and allowing the subject to rebreathe his respiratory CO_2 and ether vapor.

Three electrodes, described in the Test Equipment Section, filled with electrical conducting paste were then placed over the prepared areas and were firmly taped in place to restrain the electrode in a given position against the forces of centrifugation. On the extremities a figure-8 configuration was used to hold the electrode tighter to the limb. A tape strip was applied around the entire thorax of the animal, such that the chest lead was held in place, but not so tight as to impede respiratory movements which appear to be largely abdominal in the animal.

Couching the Animal

After the electrodes were secured in place the animal would be transferred to the animal couch described in the Test Equipment Section. The animal's extremities were secured to the couch with tape so that animal body movement was minimized. The face plate of the animal couch was then closed down over the animal and secured in place with a transverse pin. Foam rubber was utilized to restrict the animal's head movements as required, as well as to make the face plate a snug fit. The animal's tail was then

taped down in such fashion that the movement of the animal with respect to the test couch was held to a minimum. At this time the airway of the animal was cleared with a swab to assure that the normal salivation response to the ether anesthetic would not introduce a respiratory artifact. Figure 8 shows the general appearance of the test at this point. The animal and couch were then transferred to the electronic test equipment where a baseline check of continuity of the electrocardiogram circuit could be ascertained.

Weighing and Balancing

A critical step in safety and satisfactory centrifugation of live specimens required the best possible balance and weight distribution. Off balance operation of the centrifuge increased the possibility of excessive vibrations and extraneous noise in the record as well as the introduction of unknown elements into the animal's response.

The couched animal was weighed on a calibrated spring balance. The difference between this measured weight and design weight of payload (2175 gm) was added in the form of variously sized washers to previously determined positions on the couch itself. The couch with the animal was balanced so that the center of mass was within limits. Molding clay was added as required to the couch to bring the center of gravity into the desired line and to readjust the total weight. When large animals were encountered, the specimen's weight restricted the amount of balancing weights which could be attached to the couch prior to the run. Therefore, only animals which could be balanced perfectly or within very narrow ranges were spun at the very high G loads. The few animals impossible to balance closely were reserved for lower G ranges.

In spite of these restrictions acceptable weight range spreads were obtained in all of the G modes and along the entire scale of the experimental G loads.

Installation on SFAPS

After balancing, the animal couch was then taken into the pit, the interconnecting wiring harness attached and the couch bolted to the capsule. The front plate of the capsule, equipped with a viewing window, was then installed. Ventilating air was

provided via holes drilled in the bottom and top of the capsule. The pit area was then isolated and ten minutes allowed, after starting, for warm-up of the motor generator. The warm-up period reduced perturbations in rpm to a minimum and accuracy of G loading improved to a point of acceptable consistency. Other equipment, such as the recorder, also used this warm-up to advantage.

A further advantage of this procedure was that essentially complete recovery from the anesthetic experience could be guaranteed since the animal remained anesthetized for a five-minute period after removal from the anesthesia box, whereas the total handling and weighing procedure lasted approximately one-half hour.

The animal's ECG trace was continuously monitored on the oscilloscope. At an appropriate time, a baseline electrocardiogram was taken.

The experiment parameters were then chosen (centrifuge rpm, G load, and run time) based on calculations made as described in the section on mathematics. The G mode (direction of application of force) had been previously determined. A mathematical prediction as to survival or fatality of the subject was made at this point, prior to the run, and recorded in the experiment log.

Experimental Procedures

After warm-up was completed, the area was cleared, the test recording equipment turned on, the centrifuge started, and the run completed. At the end of the run the animal would be observed in place on the centrifuge as to post-run level of consciousness and condition, nystagmus with respect to type and direction, head position, and any anomalous condition such as bleeding, destruction of tissue, viability of the animal, and individual post-run characteristics. These comments were entered on the centrifugation record, an example of which as shown in Appendix B.

At that time measures necessary to help the animal in its respiration was taken. These interventions were not elaborate, however, but consisted solely of attempting to maintain a free airway. Anomalous positions of the head incompatible with a clear airway were corrected, excessive oral-pharyngeal secretions were removed with a cotton-tipped swab and locked jaws pried

open; otherwise, the animal was left in its couch unmolested until it became definite whether it was a survivor or a run fatality. Figures 9 and 11 show two examples of conditions as seen by the observer.

After an appropriate interval the electrocardiographic record was halted and the couch removed from the machine. Couch and animal were then removed to the laboratory. Figure 10 shows an example. If the animal were a fatality, an immediate autopsy would be performed with appropriate tissue preparation techniques as described elsewhere. Figure 12 is an example of the procedure used. (If the animal survived, it was quickly removed from the couch, electrodes and tape taken off, and the animal returned to its cage.)

Post-Run Procedures

The caged animal was observed in a quiet area for the remainder of the working day. Appropriate comments as to animal condition, respirations, nystagmus, spatial orientation, activities in the cage, attempts to eat and drink, state and level of consciousness, and comments on the animal's prognosis were entered on the clinical post-run progress record whenever appropriate.

Upon completion of the day's activities, the animals would once again be placed in the heated station wagon and transferred to the animal-holding facility where ward routines for feeding, watering, and observation were reinstituted. The animals would remain in the veterinarian animal-holding facility until the designated time for autopsy. At that time the cycle of delivery to the laboratory site would begin once again with the autopsy animals and the new run animals arriving as previously described via the preheated station wagon.

Cautions Observed

In view of the heavy worm infestations seen in autopsy, and presumptive infection with herpetic encephalitis, precautions beyond normal sanitary rules were followed. All papers and material exposed to fecal matter were burned in the facility incinerator and the investigators wore surgical gloves and masks when working with or near the animals. The special safety precautions mentioned in the Test Equipment and Anesthesia sections were also followed.

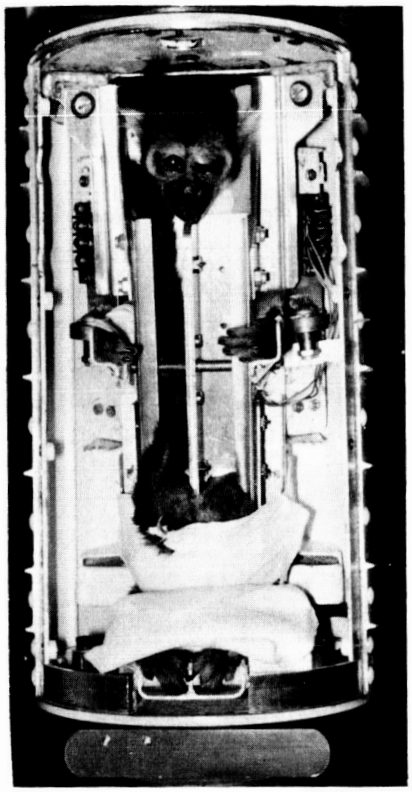

FIGURE 9. - VIEW AFTER $-G_x$ RUN
COUCH IN CAPSULE
ON CENTRIFUGE -
TEST ANIMAL AS
SEEN POST-RUN

Note: Hematoma, left eye, and head position -

Animal # 69.

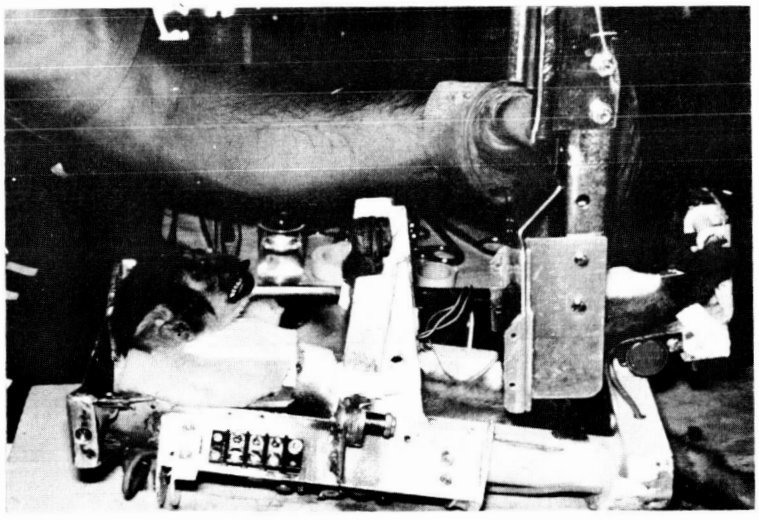

FIGURE 10. - COUCH FRONT PLATE LIFTED -
POST-RUN $-G_x$ MODE

Tongue protruding, head forward, animal strangled - Animal # 69.

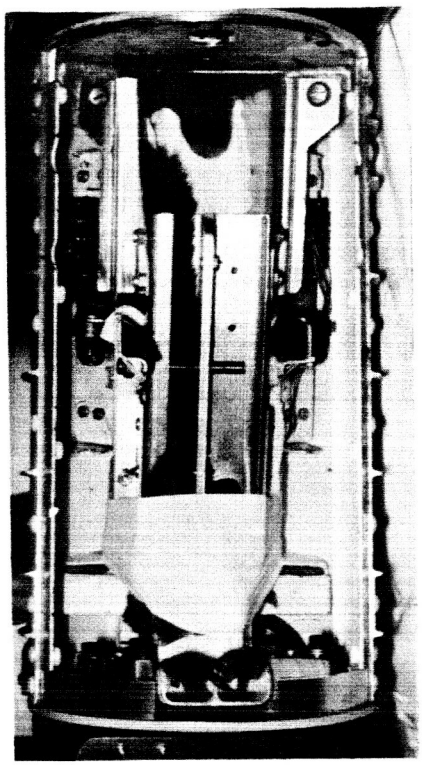

FIGURE 11. - POST-RUN $+G_X$ MODE ON CENTRIFUGE

Run survivor
Animal # 51.

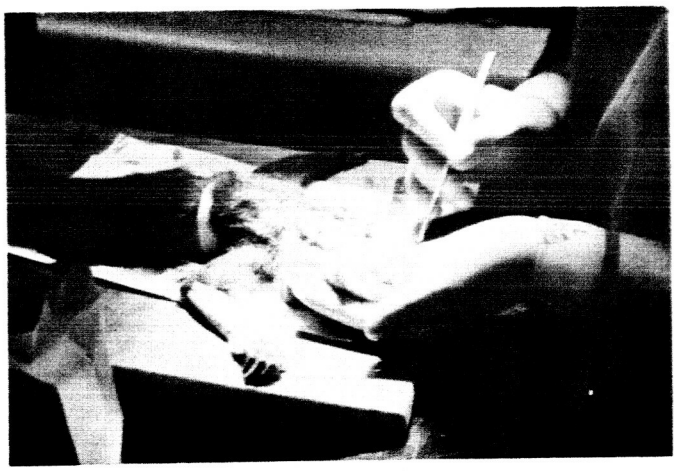

FIGURE 12. - AUTOPSY POST-RUN - BRAIN DISSECTED, GROSS PATHOLOGY UNDER EXAMINATION.

EXPERIMENTAL RESULTS

A large amount of experimental information was acquired during the investigation conducted. From a practical consideration it is not feasible to include all experimental data in this report; however, it is also unnecessary to do so. Appendix A lists the essential parameters of each experiment. A synthesis of the data and appropriate explanatory discussion are presented so that representative information is made available here for study. We have arranged these into the following separate categories:

1. Anesthesia
2. Clinical Observations
3. Electrocardiography
4. Gross Pathology
5. Histochemical Studies
6. Measurements Study
7. Mathematical Discussion

In the sections which follow each of the above categories is discussed in some depth.

ANESTHESIA

Development of a safe handling procedure for the squirrel monkey was essential for this investigation. Since the animals comprising our test colony were in the wild state (only days removed from the jungle with no period of conditioning or behavioral training) they could not be relied upon to cooperate in any phase of the experiment.

Preparation of the animal for electrode emplacement and for couching on SFAPS presented a difficult practical problem. It was necessary to develop a procedure for anesthesia which would not only permit safe animal handling, but which would insure viability of the test animal. Furthermore, another criterion was that the anesthetic should not influence the animal's subsequent response to the accelerative forces.

Test of Injectable Anesthetic Agent

A literature search revealed no information on anesthetization of the squirrel monkey; therefore, an entirely new and original procedure (we believe) was developed for this animal. Initially, pentobarbital was used. Animals No. 24 and No. 25 weighing 693 grams and 900 grams, respectively, were selected as initial anesthetic controls. A dose of 12.8 mg. per pound of animal weight was administered simultaneously to both animals; two minutes later it was noted that corneal reflexes disappeared briefly. The heavier animal initiated purposeless struggling; the lighter animal voided its bladder. Both animals lost consciousness within the following two-minute period.

Observations of subjects.- One hour post initial introduction of anesthetic (PIA) animal No. 25 was stirring slightly, but was still profoundly depressed; after one hour and ten minutes (PIA) animal No. 24 opened its eyes and raised its head briefly. Number 25 demonstrated a twitching of the extremities, stretching of the neck and tail. Both animals exhibited normal coloration of the palms, ears, and skin. After two hours and ten minutes (PIA) animal No. 24 was up and in the so-called "sick monkey" position -- that is, the crouch, head down between both legs, tail curved up over its head, and both hands grasping each other in front of the lower hindlegs. Animal No. 25 was still unconscious. After three hours and five minutes elapsed time (PIA) animal No. 25 was up and scratching, and animal No. 24 became active.

At three and a half hours (PIA) both animals were sitting in the sick monkey position, were rousable and phonating, but still seemed depressed with slow reflexes. At four hours (PIA) both animals were perching, but only with difficulty. At five hours (PIA) the animals were perching well but obviously were fatigued; heads were down and resting. It was noted that seven full hours (PIA) were required for complete recovery from anesthesia.

Since acceleration exposure durations, according to the initial test cell set up, were to range from 2 to 200 seconds, the response reported above was unsatisfactory. It seemed that this mode of anesthesia would completely mask the animal's response to the test situation, both during the run and immediately post-run. Consequently, pentobarbital was rejected as an anesthetic agent for this experimental program. The investigators rationalized

that it was necessary for the animal to be fully conscious throughout the entire test experience. It was believed that this was the only way in which the full response of the animal could be assessed without questioning the results. In view of the above position it was decided that a volatile anesthetic agent must be utilized. Its benefits would include fast depressive action and a relatively short period of recovery from anesthesia.

Diethyl Ether Tests - Part A
Utilizing Clinical Signs Criteria

Diethyl ether was the second anesthetic agent used experimentally. A small unmeasured amount of diethyl ether was poured on a gauze pad. The animal was transferred into a box of unknown volume along with the saturated ether pad. The animal was observed to be deeply anesthetized in three and a half minutes. The animal (No. 26) was promptly removed and placed in a squeeze cage for observation. The subject regained consciousness in 4 minutes, was perching in 7 minutes, and in 8 minutes was drinking water and eating monkey chow (biscuits).

Cone administration.- The same procedure was tried on another animal (No. 28). The subject was anesthetized in approximately 3 minutes and began to awaken in approximately 4 minutes after removal from the box. Additional ether was supplied by cone, drop by drop. The animal's respirations became slow and deep, even though the color was quite good. The heart rate slowed from 300 to 60 per minute, then respirations became greatly depressed and finally ceased. Despite aggressive resuscitative procedures the animal expired in respiratory and then cardiac arrest.

Burette and cone administration.- Animal No. 26 was given ether in the box in the same manner as before and removed the moment it became unconscious -- approximately 3 to 4 minutes later. The animal started to stir approximately 3 minutes after removal from the box, so it was returned to the box briefly, then taken out again. This time a burette with a drop-by-drop application of ether to an anesthesia cone was employed. With this method the animal was effectively maintained at the second to the third plane of anesthesia and preparations for the placement of electrodes and removal of the animal to the test couch proceeded without any major incident. The animal was then successfully run on the centrifuge.

Animal No. 23 was placed in the anesthesia box. It was decided to permit the animal to remain in the box one additional minute after the animal became unconscious in order to induce a deeper anesthetic level so that additional time could be obtained for handling the animal after removal from the box. However, the momentum of the animal's response to anesthesia was very great; the animal continued its downward trend into deeper and deeper planes, and finally succumbed a few minutes after removal from the box.

Diethyl Ether Tests - Part B
Utilizing Quantitative Data

Clearly, clinical observation of the animal and its response to the anesthetic agent was not a reliable method for anesthetization. A quantitized procedure for delivery of anesthetic would have to be developed, and a detailed observation of the animal's response made to assure meeting the previously established criteria. The volume and weight of the anesthesia box were determined to be 8 liters and 700 grams. By weighing the box containing an animal, then subtracting the box weight, an accurate weight of each subject was obtained. The volume of the box provided a means of determining the approximate concentration of gaseous anesthetic in the breathing space.

Response experiment.- The next anesthesia experiment included three animals: Animal A, weighing 713.2 grams; Animal B, weighing 635.0 grams; and Animal C, weighing 661.6 grams. Times given below are post initial introduction of anesthetic (PIA).

Animal A was treated as follows: one cc. of diethyl ether was taken up into a 10 cc. syringe and the total volume of the syringe injected (sprayed) into the 8 liter container at repeated intervals. After 7 cc. in 21 minutes, the animal was still conscious. Finally at 25 minutes (PIA) with no additional anesthetic the animal was observed to be anesthetized. It was removed from the box and placed in the squeeze cage where it had an almost immediate recovery (within one minute).

Animal B was treated as follows: 1 cc. of diethyl ether was placed on folded gauze pads which were then dropped into the anesthesia box. This process was repeated at 3 minute intervals. At the end of 6 minutes (total delivery of 2 cc.) no effect was noted. At 9 minutes (PIA) 2 cc. were added; the animal began to

show swaying motions, licking of the tongue, weaving motions of the head and body, gradually increasing in amplitude. At 10 minutes (PIA) the animal was grasping the walls of the container for stability. At 11 minutes the animal was retching; at 11½ minutes the animal exhibited loss of stability; at 12 minutes (PIA) the animal was anesthetized and returned to its cage. At 15 minutes (PIA) the animal was fully recovered.

Animal C was treated as follows: 3 cc. were initially placed on the gauze pad and introduced into the ether box with the animal. At one minute the animal was weaving. At two minutes it blinked. At three minutes it appeared unstabilized, was looking around, licking its tongue, and salivating slightly. At six minutes the animal appeared very agitated and tried to get out of the box. At eight minutes the animal appeared to be losing consciousness, and at nine minutes the animal was anesthetized. The animal was immediately transferred to the centrifuge couch; electrodes were emplaced. The test ECG obtained showed a heart rate of approximately 300 per minute. The animal was then allowed to recover consciousness and was replaced in the squeeze cage in the awakened state directly from the test couch. A safety screen around the test couch was employed for protection of the investigators.

A preliminary analysis of the test described indicated promise, but additional data were necessary before a working plot of dosage versus animal weight could be obtained. Therefore, the experiment for anesthesia was continued with animals Nos. 27, 29, and 30.

Confirmation experiment.- Animal No. 27 which weighed 642 grams was placed in the ether box along with a gauze pad holding 6 cc. of ether. In 20 seconds the animal appeared to be feeling the effects of the anesthetic; at 1 minute it was shaking its head. At 1 minute 25 seconds there was a profound salivary response. At 1 minute 30 seconds there was a blinking of the eyes; at 3 minutes the animal was still responsive to light. At 4 minutes 30 seconds the animal was noted to be weaving; at 5 minutes response to light was still obtainable. At 6 minutes the animal was shaking its head, weaving violently about the ether box, salivating profusely, struggling to leave the container, then was noted to be chirping very loudly. At 6 minutes 45 seconds the animal definitely was loosing coordination; at 7 minutes the animal was unconscious, breathing was rapid. At 7 minutes and 30 seconds breathing was slowing down; at 9 minutes the animal was removed from the box in the fully anesthetized state and transferred to the preparation table.

At 14 minutes (PIA), approximately 5 minutes after removal from the box, the animal began showing signs of recovery from the anesthetic. The anesthetic administration was continued using the same pad and enriching it with ether applied to the pad through a cone, drop by drop.

At 20 minutes an additional 1 cc. of ether was applied to the pad. At 22 minutes the animal was noted to be trembling; at 24 minutes, an additional 1 cc. was administered. The animal began retching. Its neck was extended manually to the side and straightened out. At 40 minutes the animal was fully instrumented with electrodes emplaced and ready for acceleration exposure run.

A 10-minute warm-up period was required for the equipment. Initial muscle noise and irregularity of electrocardiographic trace on the oscilloscope decreased during the period. Just before the run the animal quieted down and the ECG came through clearly for the baseline reading. The slightest amount of audible noise in the room disturbed the animal with introduction of a great deal of electrical "noise" through the electrocardiographic leads.

A total of one hour and twenty minutes preparation was required in order to run this animal for four and one half seconds on the centrifuge. Immediately post-run the animal was supine and vertical nystagmus of an intermittent variety was noted. The animal was replaced in the squeeze cage, still exhibiting disorientation. The disorientation was aggravated when the animal was forced to move; the animal was observed to be scratching itself and drinking vigorously. Whenever the animal was forced to move, he seemed to fall over his right shoulder and backwards.

Second confirmation.- Monkey No. 29 weighed 713 grams. Nine cc. of ether were put on the pad initially. At one minute and 30 seconds after the introduction of ether, violent shaking of the head was noted with an extreme excitation of the animal. At 3 minutes and 30 seconds the animal was struggling in the box. At 3 minutes and 45 seconds the animal was falling from side to side striking its head on the side of the box. At 4 minutes the animal began losing its balance and exhibited uncoordinated movements. At 4 minutes and 25 seconds a rapid respiratory rate was noted, and at 5 minutes the monkey was anesthetized. At 5 minutes and 30 seconds the animal was removed from the box and placed on the preparation board.

At 14 minutes and 10 seconds the monkey began stirring on the preparation board, and a few seconds later became "light". Control was maintained by the introduction of additional ether applied on the gauze pad inserted in an inverted beaker placed over the animal's head and the beaker further served as protection against biting. Twenty-six minutes were required to complete the preparation of the animal; it was awake at the end of that time. In 35 minutes the animal was in place on the centrifuge. Preparation time for this animal, as compared to No. 27, was 40 minutes less. This run was at 250 G for 20 seconds.

Postrun, the couched animal was noted to be awake before it was removed from the centrifuge. No nystagmus was observed; the animal was looking about, seemed to be well tempered, and responded amiably to stroking of the head. It chirped when its tail was accidently pinched during removal from the restraint couch. The animal was removed from the couch and transferred via the ether box to his cage. The animal began shaking his head laterally in a horizontal plane, and then was observed to be falling over his right shoulder and backward. The animal then perched but was shaking back and forth laterally in any position on the perch. One hour after the introduction of the anesthetic the animal was still completely disoriented. After one hour and 45 minutes the animal appeared quiet; there were slight lateral movements of the head; very slight horizontal nystagmus was barely noticeable. After three and a half hours the animal was offered monkey chow which he seized and ate rapidly.

Third trial.- Animal No. 30, weighing 734 grams, was given 12 cc. of anesthetic on the gauze pad initially. This was an aggressive, truculent animal which bared its fangs and screamed at the investigators. At 1 minute the animal was vocalizing and shaking its head; at 1 minute 30 seconds the animal was blinking. At 2 minutes it was salivating profusely. At 2 minutes 10 seconds it was bobbing and weaving; at 2 minutes 20 seconds it started losing its coordination. At 2 minutes 30 seconds it was bobbing and weaving; at 2 minutes 35 seconds total loss of coordination was noted. At 3 minutes the animal was unconscious, lying in the bottom of the box and breathing rapidly. At 4 minutes and 30 seconds the respirations began to slow and the animal was removed from the ether box for preparation. Three minutes after removal from the box (7 minutes 30 seconds PIA), the animal became light, anesthetically. Ten minutes after introduction of the anesthetic the animal was noted to be salivating.

At 25 minutes preparation was complete, with electrodes emplaced and animal couched. The slightest noise and disturbance in the room was immediately reflected in the ECG trace on the oscilloscope, but after such distractions ceased, the animal quickly quieted.

This animal was run at 250 G for 200 seconds; immediately after the run the animal was noted to be unconscious and unresponsive to stimuli. Four minutes post run, still encouched, the subject's pupils were not reacting to light. The animal was noted to be making periodic retching movements about 2 seconds apart. Peripheral color looked good. The respirations were stertorous and slower than normal. The animal was removed to the squeeze cage for observation. Fifty-two minutes after application of the anesthetic the animal began to show return to consciousness. One hour and six minutes after anesthesia, horizontal nystagmus was noted. The arboreal reflex was maintained, but if dislodged from his perch, the animal was noted to fall over his right shoulder and backwards. With every opportunity the animal grasped firmly to the side of the cage and tried not to be dislodged. When he was dislodged a short time later, he fell over his left shoulder.

At one hour fifteen minutes after the experimental procedure began, the animal was noted to have quick nystagmal components to the left, and it was beginning to fall alternately over the right shoulder and then over the left. The animal was thoroughly disoriented. After making a scrambling movement at the bottom of the cage, he would lie exhausted. The horizontal nystagmus continued. At 1 hour and 30 minutes after the experiment began, the animal was still falling both to the right and to the left, but was making much more purposeful movements. He did not exhibit any desire to drink.

Diethyl Ether Tests - Part C
Preliminary Working Curve

Data from Animals No. C, 27, 29, and 20 were reviewed and preliminary curves plotted to reflect animal weight, amount of ether administered, and time required for unconsciousness. After assessment it was decided that the duration parameter (time) should probably be held to a value of around five minutes. Therefore, an initial working curve was drawn showing the quantity of ether required to obtain anesthesia in five minutes, for squirrel

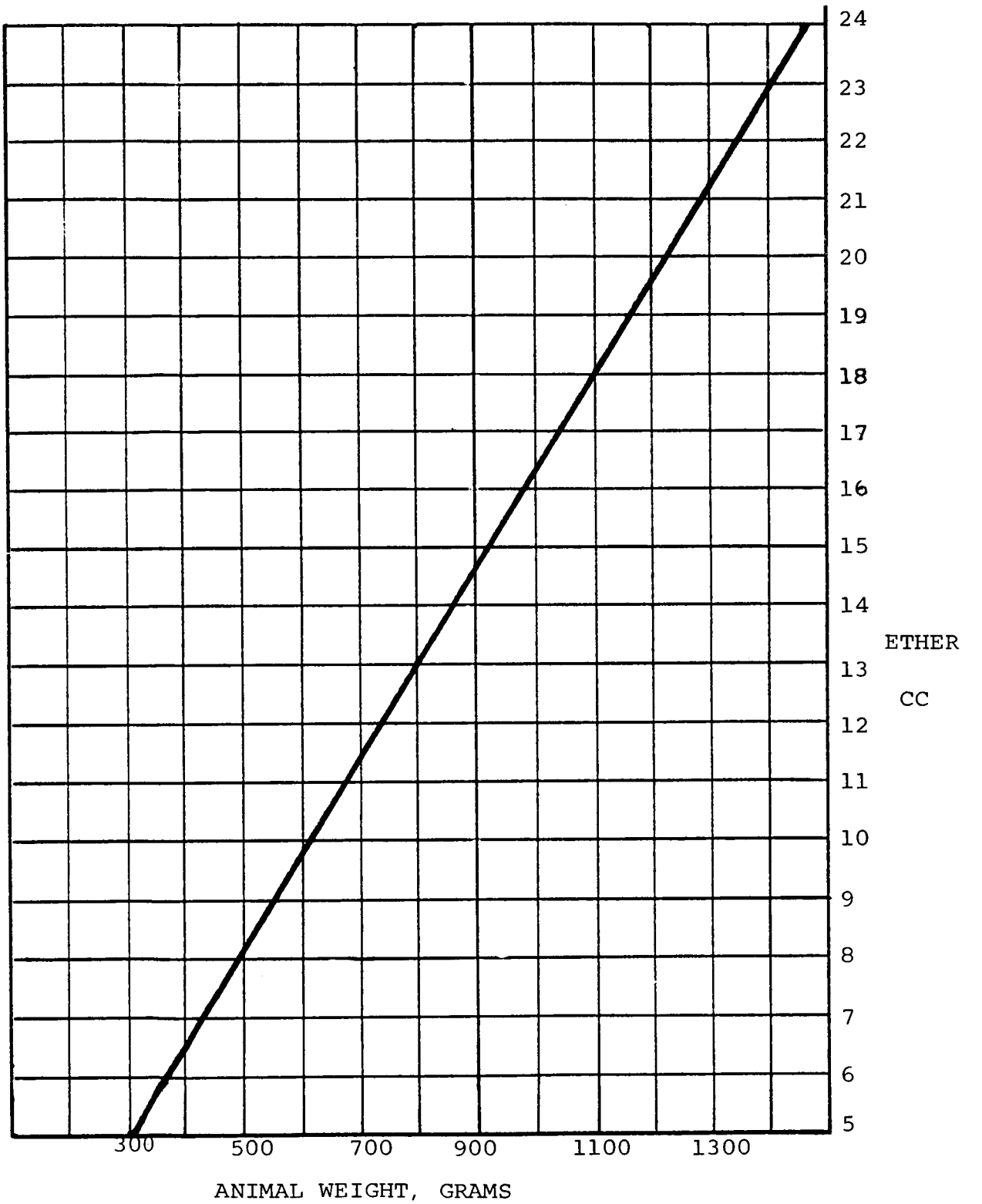

FIGURE 13. INITIAL ETHER DOSAGE SCHEDULE

monkeys of different weights. This curve is included here as Figure 13.

Augmentation and reconstruction of preliminary working curve. The above working curve was used for determining the precise amount of ether to be employed for the next sixteen experimental animals. It was utilized with success for fifteen animals without mishap. The sixteenth animal, No. 45, however, which weighed 353 grams, was given 6 cc. of anesthesic; anesthestized at 5 minutes and 30 seconds, and then was seen to go into anesthetic shock. Since this was a very young animal, and a very light animal, it was decided to pay special attention to these animals in the future. The axiom (in humans) that the very young and the very old do not respond well to anesthesia appeared to be a consideration in the treatment of these animals. The curve was reconstructed and modified appropriately in accordance with the additional data.

Diethyl Ether Tests - Part D
Application of Revised Dosage Schedule

An additional 15 anesthetizations were carried out successfully before another mishap occured, with Animal No. 61. This animal weighed 802.5 grams and, according to the graph, was given 13 cc. of anesthetic. It was anesthetized fully at 1.0 minutes; it was removed from the anesthesia box at 3 minutes completely inert. Apparently the animal had gone into deep anesthetic planes very quickly; it was noted to have excessive secretions from the mouth and, in spite of resuscitative efforts to clear its airway and to augment its respiration, the animal continued to go downhill and was declared an anesthetic death.

First amendment.- It was decided to alter the anesthetic dosage schedule by limiting the dosage of ether to 10 cc., regardless of the weight of the animal. With this limitation, 30 additional anexthetic procedures were carried out without mishap until animal No. 92 was encountered.

Second amendment.- Animal No. 92 weighed 464 grams and was given a dosage of 7½ cc. He was anesthetized in 5 minutes and removed from the box a minute and a half later. The animal continued on a downhill course and could not be recovered in spite of resuscitative efforts. The ether dosage schedule was revised once more with the stipulation that, wherever possible, if an

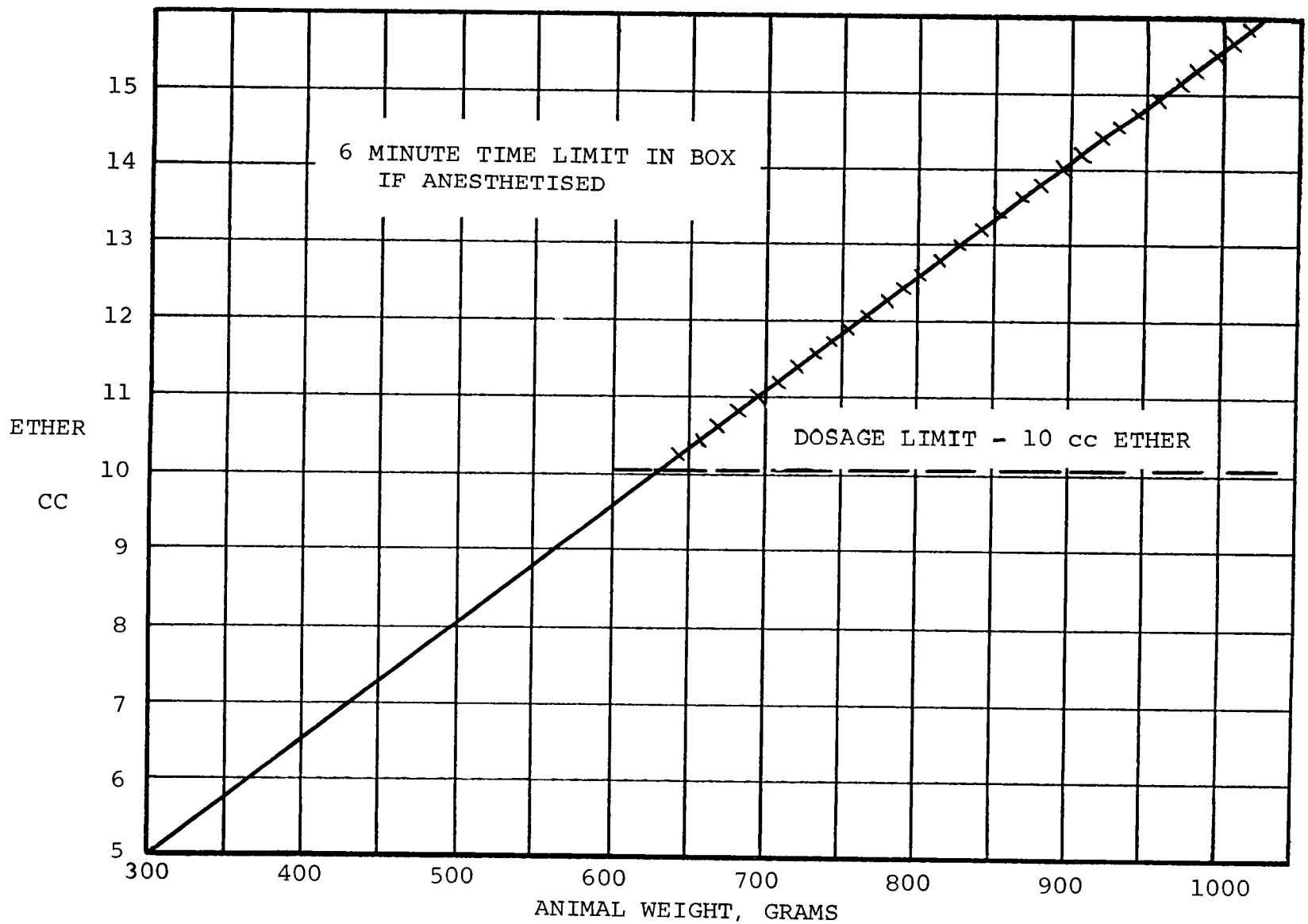

FIGURE 14 REVISED ETHER DOSAGE SCHEDULE

animal was anesthetized prior to six minutes, he would not be allowed to remain in the box for more than 6 minutes.

Third amendment.- No additional ether other than the original amount on the pad in the ether box was utilized. The animal was allowed to rebreath its own CO_2 and exhaled ether under the 150 ml head-control beaker if an additional minute or two was needed for electrode application.

Summary of limitations.- Consequently, the limitations of no more than 10 cc. of ether anesthetic and no more than 6 minutes of exposure time to dosage were employed as limiting factors. With these limitations the experiment proceeded over the next groups of animals to termination of the contract without any additional anesthetic mishap.

Diethyl Ether Tests - Part E
Application of Finalized Dosage Schedule

Consequently, the ether dosage schedule shown in Figure 14 with limitations just described was judged a safe anesthetic procedure for diethyl ether. It gave the experimenters approximately 5 minutes of working time to remove the animal from the ether box, to prepare it for the emplacement of electrodes, and to emplace it in the couch. This choice of dosage permitted the total preparation of the animal and its removal to the SFAPS machine within half an hour on the average. Generally, each animal had 25 minutes to recover from the ether dosage while still in the preparatory stage, and 10 additional minutes for the warm-up of the master generator of SFAPS.

These procedures made approximately 30 to 35 minutes of time available to the animal to recover from the initial dosage of ether anesthetic. As a consequence the animal was fully conscious, coordinated, and oriented at the beginning of each run. Therefore, an accurate assessment could be made of its response to the accelerative load both during the run and immediately post run, free of any alterations secondary to the anesthetic agent.

Extra Precautions

It is understood, of course, that diethyl ether is a highly volatile substance, subject to standard safety precautions in storage, handling, and use. Open flames, smoking, and spark-producing devices were excluded from the preparation area and the procedures must include purging the anesthesia box of residual vapors.

CLINICAL OBSERVATIONS

A series of clinical observations were made on each animal as it was processed through the program. The initial observations were conducted immediately after receipt at the animal holding facility. A health survey was instituted and findings recorded following several instances of severe bronchial inflammation in new arrivals at the colony.

Experimental Records

A chart was kept for each individual animal as the experiment progressed. Examples are shown in Appendix B. Clinical remarks were noted on the chart covering: pre-run activity; response to anesthesia; post-run consciousness; animal general condition post-run; apparent trauma or anomalous conditions; observable reaction to stimuli; head position, post-run; and development of nystagmus if appropriate, including direction and type. Additional recovery notations were made on survivors as appropriate, covering progress and observable changes in symptoms and reactions. Experimental data was also maintained on: animal weight, pre-run, post-run, and at autopsy; anesthetic dosage used; and basic run data.

Summary of Observations

In general, the animals were at a high level of alertness prior to initial handling and anesthesia. The reaction to squeeze cage operation and transfer to the preparation room elicited a wide range of response, from passive cooperation to aggressive challenge. The smaller (generally younger or male) were, with notable exceptions, the more passive. Female animals screamed loudly and often.

When ignored for a time, the animals quieted, but maintained an alert watchfulness of the investigators and other activity around them. Some correlation of rapidity of anesthetic effect to degree of activity just prior to insertion of the anesthetic-soaked pads was noted. The more excitable animals succumbed to the anesthetic earlier than their quieter fellows.

Pre-run observation.- After preparation and encouchment, movements of the animal were readily observable on the electrocardiographic traces. A valuable check on animal response to environmental stimuli other than accelerative stress was seen.

Observation of 2 and 20-second dwell cases.- Regardless of the peak G exposure, animals stressed for 2 or 20 seconds were conscious at first post-run observation in the capsule. Their respirations were noted as essentially unchanged from pre-run values. Auditory and visual stimuli routinely elicited normal response. Some of the animals chirped upon removal of the capsule cover plate. One animal in particular (No. 51) was remarkable in his vocalizing, after exposure to 400 G for 20 seconds. The general condition of animals held at peak G for 2 or 20 seconds appeared very little different from that at pre-run.

Generally, a few minutes after the run, but occasionally immediately after, a horizontal nystagmus would appear. On several occasions the nystagmus would be vertical. In either case, the condition was aggravated by attempts of the animal to move his head about in the couch, or to follow visual cues originated by the investigator. The head was routinely seen in the anterior-posterior direction at the initial look post-run. After removal from the couch and release into the cage, the animal would show disorientation, and a tendency to fall backwards over the right shoulder.

Anomalous conditions routinely seen included hematomas of one eye in the $+G_y$ and $-G_y$ modes and of both eyes in the $-G_x$ mode. In the $-G_x$ mode lacerations of the tongue over the incisor teeth, with blood spattered on the exterior capsule plate was seen. Occasionally, bleeding of the tail would be noted where the tail had contacted a sharp edge in the couch.

Observations of Longer Dwell and Higher G Cases

At the higher levels of stress (300 G and above) and also after long dwell periods at any stress level, the animals were routinely seen to be unconscious. The eyes were open, fixed in the anterior-posterior direction (relative to the skull), with dilated pupils and rarely with any discernable pupillary reflex.

No nystagmus was noted on opening the capsule. The animals remained couched on the centrifuge until clinical death occurred or sign of returning consciousness were noted. In many instances, nystagmus supervened at this point.

At levels approaching the point of lethality, immediately post-run respirations were noted as retching in type, very poor in force, with extreme intercostal space retractions, and at a rate of approximately one per second. If the animal survived the exposure, the respirations grew stronger and increased in rate within a few seconds post-run. Those animals which expired soon after the runs were seen to go into respiratory arrest and finally by ECG monitor into cardiac arrest. Those animals which expired prior to termination of the run were noted to be in respiratory arrest upon first observation post-run. ECG activity was profoundly bradycardiac in these latter animals.

Survivors

If the animal survived the experiment, changes in the brain observed later in the pathologic and histochemical studies apparently were not severe enough to alter the animal's basic senses of sight, hearing, taste, touch, and smell. The animals were seen to clinically respond to stimuli affecting these senses, sometimes almost immediately after exposure to high G loadings. As examples, offers of fruit were greeted with obvious pleasure. Monkey chow was held, palpated, and handled normally. The monkeys continued to participate in mischievous tail pulling amongst themselves. Cases of recognition of white laboratory coats as a threatening situation were seen in the survivors. In one case, recognition of the noise of the centrifuge as an extreme threat evoked radical escape reactions.

Certainly, as indicated by Animals No. 157 and No. 241, held for three months post run, and No. 100, 7½ months post run, the

animals were capable of normal activity within their cages and appeared to demonstrate behavioral patterns in the post-run period similar to pre-run patterns.

ELECTROCARDIOGRAPHIC FINDINGS

Probably the most spectacular findings of this investigation were the electrocardiographic (ECG) findings. It was the collective opinion of the investigators that a detailed study of these findings alone would constitute in itself another lengthy paper. As each animal's experience was recorded, the researchers could not help but be impressed with the occurrence of bizarre patterns and unique combinations of events, some of which were no less than fantastic. Evidence of recurring patterns was seen, although not in all cases identical. Unique events were noted on individual records. This chapter summarizes the findings of these examinations; further details of selected ECG's are discussed in Appendix C.

Experimental Animal Configuration

Each animal in this experiment was instrumented for an electrocardiogram. Three electrodes were utilized as follows: one to the right arm, the second to the left leg, and the third to the precordium. The electrodes were taped rather firmly to the extremities and to the chest in order to insure contact throughout the experiment.

The point at which the precordial lead was placed on the chest was obtained by manual palpation of the thoracic wall until the point of maximal impulse (PMI) was identified. The electrode was placed over the PMI. Routinely this proved to result in a wave pattern compatible with the V_3 patterns. It was noted that with shifts (1) of the cardia with respect to the electrode, (2) of the electrode with respect to the cardia, (3) or of both the heart and electrode with respect to each other, there would be reflected in the ECG a change in the QRS wave form toward a pattern similar to V_2 or to a pattern similar to V_4.

Naturally the direction of axis of spin will determine to a degree some of the alterations in wave form obtained. Some of the

changes for example would be as follows:

In the $+G_x$ mode the linear motion is forward. The heart is shifted nearer to the spinal column; i.e., a greater distance from the anterior thoracic electrode with a subsequent diminution of ECG voltage compared to baseline.

In the $-G_x$ mode the linear motion is backwards. The heart is shifted closer to the anterior thoracic wall; i.e., closer to the chest electrode. The right ventricle is pressed against the sternum with a probable flattening of the cardiac chambers.

In the $+G_y$ mode the linear motion is to the right. The heart is shifted to the left with respect to the chest electrode. This motion changes the configuration of the ECG from a V_3 to a V_2 pattern. The heart is stretched in the direction of the normal path followed by a beating heart in the thorax.

In the $-G_y$ mode the linear motion is to the left. The heart is shifted to the right with respect to the electrode. This motion changes the ECG pattern from a V_3 to a V_4 pattern. There are probably other rotational anatomic changes in all modes.

The Electrocardiogram Tracing

The electrocardiogram was continuous from before the start of the event to several minutes after the termination of the event. A dynamic record was thus obtained of sequential events of cardiac significance for each animal.

Electrocardiographic equipment considerations.- Adjustment of ECG equipment available were: (a) time lines; (b) paper speeds; (c) traces and filters; (d) sensitivity; (e) electrode configuration and size. These are discussed in detail in the section on Test Equipment in detail and are briefly described below.

Time lines.- The electrocardiographic records have vertical time lines. The time distance between two adjacent vertical lines is equal to one second, $\pm 2\%$, so no matter what the measured distance is between two adjacent vertical lines, the time elapsed is one second within the accuracy of measurement.

Paper speeds.- The only change made during the ECG recording was the speed of the paper. Three speeds were utilized for convenience. They were 100 mm per second, 50 mm per second, and 25 mm per second. These three speeds are readily identifiable on any one record by simple examination of the space between any two vertical lines.

Traces and filters.- Two ECG traces were used, from the single set of electrodes through different band pass filters, to help interpret the cardiac activity when partially masked by muscular noise or organ movements due to the dynamic stress. The regular trace filter value was 110 cycles per second and the mirror image trace filter was 50 cycles per second, both at 10.5 KC input.

Sensitivity.- The sensitivity was usually adjusted to 10 millivolts per three cm (10 mv/ 3 cm). When the recorded signal was inadequate at this level, a setting of 1 mv/cm was used. On several occasions a particular animal's response required a different sensitivity adjustment.

Electrode configuration and size.- The outside diameter of electrodes was ½ inch. The inside diameter for the arm and leg electrodes was 3/8 inch. The inside diameter of the precordial electrode was ¼ inch.

ECG Analysis Plan

As an initial approach to the analysis of the ECG, a protocol was set up, dividing the total ECG into twelve phases. These phases were approximate and arbitrary in nature and were designed solely to aid in the analysis identification and correlation of the series of ECG's covering different stress levels and varying periods of actual dwell time. Figure 15 shows the relationships of the phases described below.

Phase I, baseline, pre-run.- This short record was taken on each animal after it had been placed in the capsule and had sufficient time to quiet down from the handling and preparation, and had become used to the noise of the machine during warm-up.

Phase II, baseline, pre-event.- The ECG record was begun 3 or 4 seconds prior to energizing the centrifuge, after the machine warm-up period had been completed. This portion of the trace indicates, in comparison with Phase I, any variation of the subject's

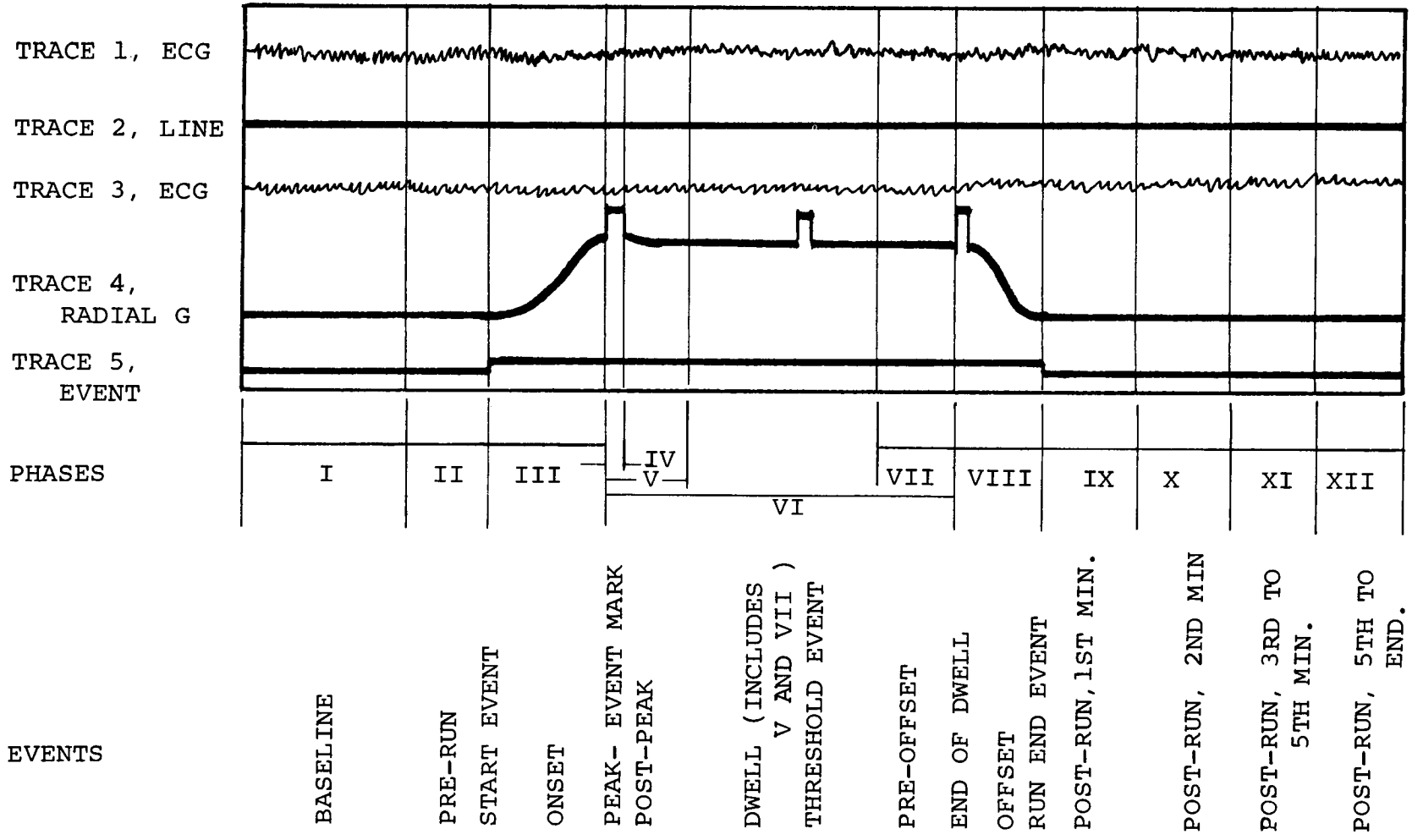

FIGURE 15. ELECTROCARDIOGRAM PHASES AND EVENTS FOR ANALYSIS

cardiac output due to the machine noise or other disturbing elements.

Phase III, onset.- At the instant that the centrifuge drive switch is closed an event mark is automatically placed on the ECG by deflection of Trace One. Phase 3 begins at this instant and continues until the preselected value of G is reached.

Phase IV, initial overshoot.- As the centrifuge approaches the G level selected, the inertia carries the arm to a speed slightly higher than desired. This is termed overshoot, and exists for one to two seconds before the machine stabilizes at the desired G level.

Phase V, initial dwell.- This phase was intended to cover the first few seconds after attainment of the target G and is a particular portion of the following phase, VI.

Phase VI, dwell.- This covers the entire dwell period from Phase IV to the beginning of the offset phase.

Phase VII, pre-offset.- This phase covers events occurring in the last few seconds of dwell, just prior to the beginning of offset, and is a portion of Phase VI.

Phase VIII, offset.- At the predetermined time at dwell, the centrifuge power was returned to neutral and the machine brought to a stop. When the arm ceased to rotate, the power was cut off, returning the event marker to zero position. This operation is termed offset.

Phase IX, post-run, first minute.- This phase extends for the first minute after the "end of event" marker.

Phase X, post-run, second minute.- This is self-explanatory.

Phase XI, post-run, third to fifth minutes.- This is self-explanatory.

Phase XII, post-run, final.- This phase covers any period after the end of the fifth minute post-run.

Record examples.- Two hundred ten electrocardiographic records were obtained during the experiment. A precursory examination of all 210 records resulted in a broad spectral view of the

overall results to be expected. A group of 25 records was selected as shown in Table No. III as a representative summary of all results obtained. Included are examples of tests in all G modes, and at all G levels. Fourteen records were chosen to show the marginal fatalities, which were called "cliffhangers". The remaining eleven show survival or death at the extremes tested.

Four records representative of most of the findings were chosen for display in this report. These are discussed in detail with illustrative figures in Appendix C. The records are from Animal No. 190 which was subjected to $-50\ G_x$ for 286.5 seconds and which was a fatality; No. 239, which was exposed to $+430\ G_x$ for 115.6 seconds and which was a clinical survival; and a most interesting combination of comparisons: Animal No. 241 - 242. This animal, as No. 241, was exposed to $+200\ G_x$ for 213 seconds. After three months, during which time it gained approximately 94 grams in weight, this animal was then relabeled Animal No. 242, spun at $+200\ G_x$ for 202.6 seconds and again was a clinical survival postrun. This animal (No. 241-242) was also the "Rerun Control" in the histochemical enzyme study. Enzymatic changes which support the changes in the electrocardiograms illustrated here and in Appendix C are identified in the chapter on histochemistry.

Method of examination.- Each ECG was examined for certain artifacts. Among these were possible representations of loose electrodes, 60 cycle noise, muscle tremor, or equipment noise. For purposes of standardization an initial approach to each electrocardiogram was constructed as follows. The amplitude of the baseline trace deflections were taken as 100%. For convenience, the amplitude of the trace deflections are quoted in units, representing 2 mm on the recording, or the space between the longitudinal marks. Measurable changes in amplitude were then compared to this baseline by percent. Each electrocardiogram was examined for rhythm as to whether it was regular or irregular and as to type such as tachycardia, block, extrasystole, flutter, or fibrillation. The heart rate was determined at various periods by measuring the distance between five or ten peaks, dividing by the distance between time lines, and adjusting the resulting beats per second into an equivalent rate per minute. Rates thus obtained are approximate and essentially instantaneous, and should not be directly compared with normal rates averaged over 15 or 30 seconds. Changes in these rates with respect to differing periods

TABLE III

ECG SELECTION FOR ANALYSIS

Typical Animals									
No.	Mode	nG	Time	Result	No.	Mode	nG	Time	Result
138	$+G_x$	50	257	Survivor	127	$+G_y$	400	171	Fatality
148	$+G_x$	50	341	Fatality	100	$-G_y$	400	200	Survivor
239	$+G_x$	430	115	Survivor	124	$+G_y$	400	200	Fatality
240	$+G_x$	430	124	Survivor	Re Run Animal				
229	$-G_x$	400	114.2	Survivor	241	$+G_x$	200	213	Survivor
230	$-G_x$	400	118.3	Fatality	242	$+G_x$	200	202	Clinical Survivor
126	$+G_y$	400	174	Survivor					

Post Run Fatalities - (Cliff Hangers)									
No.	Mode	nG	Time	Result	No.	Mode	nG	Time	Result
174	$+G_x$	100	223	Fatality CH	153	$+G_x$	400	200	Fatality CH
173	$+G_x$	250	180	Fatality CH	237	$+G_x$	430	120	Fatality CH
183	$+G_x$	250	195.6	Fatality CH	190	$-G_x$	50	286	Fatality CH
146	$+G_x$	250	200	Fatality CH	194	$-G_x$	100	163	Fatality CH
145	$+G_x$	250	211	Fatality CH	203	$-G_x$	250	132	Fatality CH
147	$+G_x$	300	200	Fatality CH	228	$-G_x$	400	200	Fatality CH
164	$+G_x$	350	173.8	Fatality CH	235	$-G_x$	400	115	Fatality CH

of the spin profile were noted, as were any changes in pattern. The PR interval was noted as either normal, long, or irregular. The QRS complex was examined as to height and width and as to whether it was normal, longer, or irregular. The QT interval was also examined. The P wave was examined for height, width, normal shape, whether it was absent, extended, or irregular. The T wave was examined for height, width, normality, irregularity, length, or inversion. The ST segment was examined as to whether it was normal, shorter, longer, raised, or depressed. Electrocardiographic evidence of the presence of conduction deformities was looked for. (Various unique events were noted in each record as they occurred.) Each record was examined for evidence of myocardial ischemia and injury.

Findings

Generally certain findings were present, but these changes were not necessarily specific to any individual record. The major reoccurring events are discussed below.

Onset period.- Following the initiation of onset, a tachycardia was usually noted, with the rate going up to 360 beats per minute from the baseline standard of 300. Heavy electrical noise obscured the trace details, differing strongly from the records obtained in previously privately supported research (7), where the animals had been anesthetized.

Dwell period.- At the beginning of dwell a period of cardiac arrest was often noted lasting from one to several seconds. At other times a bradycardia would be noted. During dwell at Target G a return of the heart rate to some intermediate bradycardic rate to some intermediate bradycardiac rate was noted after the initial bradycardic event, usually from 60-80 beats/minute higher. Further alterations of the heart rate were noted, rarely increasing even to the pre-run rate, but much more often stabilizing at a rate far below that of the baseline rate.

During the dwell period the amplitude of the QRS complex was sometimes greater, more often less, than baseline. The T wave became diphasic and would give the appearance on the monitor scope of "marching back" to the QRS complex, finally merging with the QRS complex, and becoming indistinguishable from it. If the experiment was terminated at the point where the T wave "met" the QRS complex the animal went on to become a clinical survival. If the T wave merged with the QRS complex and the

exposure to G stress continued for approximately one minute longer, there would almost always be a post-run bradycardia.

Post-run period.- If this post-run bradycardia were abolished within 30 seconds and cardiac activity returned to at least 180 beats per minute, clinical survival of the animal was guaranteed. If the heart rate did not increase within 30 seconds post-run, the animal was also clinically observed not to be exchanging air while still encouched on the apparatus. Heart rates of one beat per second (60 per minute) for periods up to and surpassing one minute with clinical survival of the animal, were occasionally observed. The general observation, however, was that if the heart rate returned within 30 seconds post-run to 120 beats/minute, the animal would be clinically noted to be breathing while still on the apparatus. Empirically then, the longer the period of stress after the T wave had merged with the QRS complex, the greater the period of bradycardia post-run and the less chance the animal had of becoming a clinical survival.

Certain of the electrocardiographic records showed a beat of one per second for a period of time post-run, extending as much as one-half hour. At autopsy this source of electrocardioactivity was found to be coming from a pulsating auricle with simultaneous ventricular standstill, so the source of this pulse then was from the SA node which initiated the auricular pulsations seen for periods extending more than half an hour past clinical death of the animal.

Muscle tremor noise.- Pronounced muscle tremor noise was noted on each ECG during the onset phases and well on into the dwell. However, it was also noted that when the empirical mathematical point of 1.0×10^4 Kg-G-sec (or threshold K_t) had been attained, the muscle tremor totally disappeared. From that point on, the electrocardiographic record was totally devoid of muscle tremor noise. This observation was taken to indicate that the animal was totally unconscious. Again, see reference (12).

Description of PQRST complexes.- For a description of the PQRST complexes, the following brief information is given:

Baseline:- For most of these records amplitude of the baseline R wave was consistent at approximately 12 units (24 mm) which was taken as a reference value for the rest of the electrocardiogram. As noted earlier, this sensitivity was set during

baseline checks. The baseline cardiac rate was approximately 300 per minute. The width of the QRS complex was 1.5 mm. There was a ½ space depression of the J point, the QT interval routinely could not be pinpointed accurately; however, there was a QU duration of 13 mm noted. The P wave routinely had an amplitude of 2 units and a duration of 2 mm.

Onset:- Generally during onset it was very difficult to distinguish electrocardiac activity from the gross amount of muscle tremor noise that was observed. However, where it was possible to do so with a high degree of certainty, the cardiac activity was noted to increase slightly from 300 to 360 per minute. Usually at the start of onset it was possible to see the start of an S wave which was not previously present. This would most likely suggest a rotation of the anatomical axis of the heart. In the $+G_x$ mode such a rotation would bring the nodal plane of the main QRS vector into line with the precordial lead and could partly account for the loss of the QRS complex during this phase of the noise. This point is speculative, however.

Dwell:- During the early parts of the dwell period a bradycardia of around 54 to 60 would manifest itself. Ventricular activity seemed to return first followed by atrial activity. The amplitude of the R wave of the complex routinely became reduced as compared to the baseline, on occasion reaching a value of 25% of baseline. The question is raised as to whether or not this would represent a loss of electrical function of the majority of the normal conducting passages in the ventricles. A gradual return of rate to approximately 120 would be noted and a slight increase in amplitude of the R wave to approximately 50% of prerun level would be noted. Occasionally one would see a pause followed by sinus beat, then a return to an idioventricular rhythm. The brief period noted at these points was almost Wenckebach in nature. A pattern characteristic of sinus arrest was often seen. Changing heart blocks were routinely noted; one would see a change in the amplitude from one R wave to the next which would be compatible with expiratory movements of the thorax. A QRS duration of almost 2 mm would often be noted which would be rather prolonged with respect to the baseline and therefore abnormal. This is comparable to a left tunnel branch block pattern or left ventricular conduction defect. The ST shift and the inverted T wave could then be considered changes secondary to an interventricular conduction defect.

Late dwell:- Later on in the dwell period an extremely bizarre QRS complex with marked widening of the QRS, ST, and T segments was seen with an approximate width of 8 times the baseline QRS duration. A slowing of rate would then routinely follow. If the duration of the run were of sufficient length, a fusion of these three components would become manifest on the oscilloscope.

Another series of events after slowing of the heart rate at dwell would be a widening of the PR interval followed by first degree block and then further widening of the QRS complex with definite episodes of ventricular tachycardia intervening. P waves occasionally were seen at intervals representing an episode of complete block, possibly a functional block due to the ventricular tachycardia and the fact that the P wave would find the AV node refractory The electrical activity during these phases was certainly agonal. There were episodes of ventricular tachycardia, ventricular fibrillation, isolated beats conducted from above through a prolonged PR interval. In agonal animals, QRS duration was quite slowed, the rate very low (as low as 40 per minute), then routinely a complete disassociation of atrial and ventricular foci would be noted.

Offset:- Often during the offset phase the pattern of complete heart block would persist with P waves bearing no relationship to the QRS, P wave activity being at a markedly slower rate at about 40 per minute, and with the ventricular activity being in the range of 30 per minute with bizarre ventricular complexes. A marked reduction in QRS amplitude was often seen during the offset period.

Post-run:- On the post-run period the most fantastic observations would occur. It seems as if the superventricular focus of conduction would take over, the rhythm becoming sinus bradycardia at some slower rate, with the PR interval as wide as it was ever measured, at about 3 mm, constituting a first-degree heart block. A diphasic T was observed and was interpreted as indicative of subendocardial ischemia.

In the animals destined to be survivors, the next fascinating thing that occurred would be the pick-up of the atrial rate with a development of 2 to 1 heartblock (second-degree heartblock). For example, the atrial rate would be 60 and the ventricular rate 30, or the atrial rate 120 and the ventricular rate 60. Then the heart would get through this 2 to 1 block and experience a period

of 1 to 1 conduction at either a rate of 60 per minute, or 120 per minute within the first 30-second period post-run. Very soon thereafter the rate would increase to 180. There was an almost immediate return of the QRS duration to baseline value. The PR interval was slightly prolonged, on the average being 8 mm compared with a baseline of 6 mm. The ST segment seemed to come back down toward baseline routinely with diminution in the amplitude of T. Rate then gradually increased becoming sinus rhythm at approximately 200 beats per minute; the T wave usually remained somewhat peaked and diphasic. If animals were to survive, the rate would then continue to reapproach the baseline rate of 300. In the case of lost animals, the rate of the heart would remain at 60 beats per minute or less in the post-offset phase. In the animals whose ventricles had surpassed tolerance an initial period of what seemed to be recovery was followed by an increasingly profound bradycardia with ever-decreasing rates from 180 to 120 to 60, then to 30, then to no beats at all.

Summary

In summary, during onset a slight tachycardia generally occurred. Immediately post-onset most animals either had a complete cessation of electroconductivity within the heart or had a change in the electrical axis such that the QRS voltage was not appreciated by the electrode. During dwell, there was a gradual development of electrical activity within the heart, but since there was no proof that there was actual blood flow through the heart, the electrical activity of the heart would have to be, at this time, considered that and that alone. (The ECG in itself cannot tell anything about blood flow.) During the dwell there routinely would be a gradual deterioration of electrical activity manifested by abnormal bouts of ventricular tachycardia and other abnormalities in rate and rhythm. Evidences of myocardial ischemia and injury were manifest during the dwell period. Post-run in the surviving animals it appeared that the ventricle would initially go through a recovery period manifested in the ECG by a gradual increase in rate. One could surmise at this point that blood flow to the myocardium was increased with improved oxygen flow to the heart. In non-surviving animals the gradual change in the ST and T waves with the development of the full-blown pattern of acute myocardial injury was noted. As these patterns became firmly established the rhythm returned to that of a marked sinus bradycardia, finally becoming a terminal bradycardia.

Questions are raised in mind as to the ability of the heart to resuscitate itself. It is well known, of course, that irreversible brain damage in man occurs between 3 and 5 minutes post-ischemia. Certainly the period of time for irreversible myocardial damage is probably somewhat longer than this. It may be that the bradycardia observed terminally here is simply a manifestation of myocardial injury developed during the course of the run to the point of irreversibility. In retrospect it would certainly be of interest to get comparison electrocardiograms in 5 days to a week following spins in animals which survived. Here one might see the development of T waves and other manifestations of myocardial infarction. An attempt at this sort of investigation was made with the inclusion in Appendix C of the initial ECG of Animal No. 241 and then of its subsequent ECG when it was rerun as Animal No. 242 three months after its initial experience of near tolerance G-stress. Figures 83 and 84, Appendix C, offer visual evidence of the differences in patterns between initial baseline and post-run 241 and re-run 242 baseline.

GROSS PATHOLOGY

All animals in this study were subjected to gross pathologic observation. Autopsies were performed immediately on animals which either did not survive the run or which expired a few minutes post-run. The remaining autopsies were distributed over a 24-hour period, a 48-hour period, a 72-hour period post-run, or beyond. The reason for the distribution of autopsies on an hour basis post-run was to enable the investigator to assess the animal's response to damage and the animal's ability to resolve damage at varying periods of time after exposure to the G load. The distribution of post-mortem examinations is shown in Table XIX, Appendix A, which indicates the distribution not only by G mode and G stress load, but also by the elapsed time post-run at which the post-mortem examination was held. An example of the Necropsy-Necrotomy Protocol is listed in Appendix B. All tissues listed in the protocol were routinely examined. Occasionally other tissues of interest were examined and data on these tissues were listed in the appropriate chart space labelled "other".

Findings in $+G_x$ Mode

A summary of each G stress level in the $+G_x$ mode now follows. A detailed listing of significant pathologic changes in this mode by animal number appears in Appendix D. Other modes utilized in this experiment are compared to the $+G_x$ mode, and their results are also tabulated in Appendix D.

Summary of $+50\ G_x$.- The lungs, liver, adrenal glands, and the brain appeared to show significant alterations in the $+50\ G_x$ series of experiments immediately after exposure. Congestion of the brain was well developed, especially by the 48th hour. Regardless of duration of stress, maximal changes in the response of the animal to the stress were noted in the lungs and in the adrenal glands at 24 hours. The generalized worm infestation evident in these animals apparently did not alter significantly the animals' response to stress. As stress duration increased the spleen showed significant changes as did the GI tract.

Summary of $+100\ G_x$.- At the $+100\ G_x$ level of stress, evidence of post-load stress on lung tissue is still present at 72 hours and inflammatory processes are still underway. In fact, at 72 hours the reparative process appears to be near its maximal rate. In the animals not surviving the run, or dying immediately post-run, there is evidence of overwhelming respiratory and circulatory disturbance. Early changes in the adrenal gland are routinely noted in all animals. Generally changes in the adrenal gland occur early in the post-run period. There certainly is a gradation of response with increasing exposure to the G load, with longer stress times resulting in ever-increasing damage to the tissues. The pronounced extent of the inflammatory response evidenced by the mildly exposed animals 72 hours post-run was surprising even though clinically the animals concerned had made a rapid recovery.

Summary of $+150\ G_x$.- In the $+150\ G_x$ series of experiments, some inconsistencies as to the animals' response to the stress appeared. No. 176 did not show any significant changes in the adrenal gland for the amount of stress to which it was exposed. But this animal was examined early in the post-run period which suggests that at least 72 hours are required for the loss of normal adrenal gland fat and color at this stress level. See the

results shown for Animal No. 142 in Appendix D. Generally, the responses to the stress were well developed by the 48th hour if the animal survived the run. The overwhelming congestion of the lungs, the liver, and of the heart and brain were of special significance in Animal No. 181 which did not survive the exposure. Other animals showed moderate changes in all tissues except the lungs which were heavily affected.

Summary of +200 G_x.- It seems as if the animal can tolerate +200 G_x up to 200 seconds and show only moderate response, not only post-run but also 24 to 48 hours after the run. However, beyond 200 seconds of dwell the response of the animal to stress appears to be overwhelming. Notable changes include the appearance of intraluminal GI fluid and early adrenal and splenic changes.

History of Animal No. 241-242.- In this group of +200 G_x subjects, animal No. 241 was particularly interesting. This animal appeared clinically intact post-run and was allowed to survive for three months; at the end of which time its electrocardiogram showed significant abnormality as opposed it its original prerun electrocardiogram. (This latter observation will be amplified in the ECG section and Appendix C.) After this three-month period, it was relabeled Animal No. 242 and re-exposed to stress as the "Rerun Control" for the enzymatic studies discussed in the histochemistry section. As Animal No. 241 it had been subjected to +200 G_x for 213 seconds. Three months later as Animal No. 242, it was resubjected to +200 G_x for 202 seconds. It had gained 94 grams between the two runs. The animal survived both runs and immediately post-run as No. 242, was sacrificed deliberately.

Detailed autopsy findings of No. 241-242:- This animal was noted to have posterior lobe hemorrhage in both lungs; the intestinal tract was slightly injected but otherwise not remarkable. The heart was heavily infiltrated with fat and was flabby. The pancreas was friable and injected. The liver showed hemorrhagic infarcts over its entire surface. The gall bladder was moderately tense. The spleen was moderately injected; the kidney was slightly injected and fatty. The adrenal glands were the normal chalky yellow color. (Apparently the adrenal gland had grossly recovered fully from the first run and was again of the normal coloration.) The brain was slightly injected throughout. The dura over the

petrous bone area also seemed injected and was therefore retained for possible future examination of the cochlea. From the gross pathology point of view this animal after its second exposure in three months to +200 G_x (approximately to the same amount of stress, see Appendix A) could have clinically survived after its second exposure had it not been sacrificed.

In summary, squirrel monkeys seem to be able to absorb loads of 200 +G_x for periods of time, up to 200 seconds dwell. Beyond that, with perhaps 10 seconds of additional exposure, an overwhelming and lethel response to the stress is produced. The inference here is that a very fine calibration of this animal occurs at +200 G_x for its range of weights.

Summary of +250 G_x.- The general impression gained is that the animal appears to be able to accept +250 G_x rather well up to a fine point around 180 seconds dwell, not demonstrating too much pathology at 24 hours, 48 hours, or 72 hours, post-run. Beyond this apparent point of calibration, the animal spills over into a zone of very pronounced response to the stress. Two hundred fifty G then compares well with the responses seen at 200 G.

There is some indication here that the animal is able to successfully manage loads of +200 G_x or +250 G_x of less than 180 seconds exposure, while at higher time relationships, the animal dies. This strongly suggests that the mechanism of damage may be different in the 50-150 G stress loads than in the 200-250 range.

Summary of +300 G_x.- The tremendous force applied to the animal is emphasized by the separation of the skull suture lines in animal No. 151 while the otherwise negligible responses of the many tissues (other than liver and lung) is also underscored. If the animal survives the initial stress experience, its tissues show maximal response by the 24th hour.

The mechanism of death in the +300 G_x animals appears to be slightly different than those seen between 50-150 G and 200-250 G. Plastic extrusion of animal tissues through or around rigid structures may be the primary mode of mechanical damage in this high stress range, rather than the congestion and anoxia usually seen between 50 and 150 G and the precipitous stress-

related failure past 180 seconds dwell at 200-250 G.

Summary of $+350\ G_x$.- Advance into the $+350\ G_x$ range imposes tremendous forces, separating the animal's skull at the suture lines and possibly extruding brain tissue through the various foramina, all augmenting the anoxic and congestive phenomena present. In addition to the massive damage to lung, liver, and brain, these animals also show intraluminal GI fluid and free blood. Spleen and adrenal changes appear at 24 hours.

Again it appears that the response of $+350\ G_x$ animals is not attributable to congestion and anoxia alone as in the lower G runs for more protracted periods of time. Rather it is suggested that death is due to other events supervening along with the congestion and anoxia. Most probably destructive plastic deformations take place.

Summary of $+400\ G_x$.- The lungs and liver routinely show damage secondary to congestion. Intraluminal GI fluid is still present at the 48th hour. All other body tissues (except the liver and lung) are not as severely affected as in the lower G levels. Here again, extravasation of blood through the skull suture lines and into the brain spaces seems to be implicated as a cause of death.

G Mode Relationships

Generally speaking the animals exposed to the $+G_y$ mode and $-G_y$ mode produce similar gross pathologic changes at approximately the same levels of G and time as do the $+G_x$ animals. The $-G_x$ animals, however, produce pathologic changes at lower levels of stress loading. Except for this consideration, the heart, intestinal tract, pancreas, spleen, kidney, and adrenals had essentially common responses in the $-G_x$ mode when compared to $+G_x$, $+G_y$, and $-G_y$ modes.

Temporal Response to Injury

The animals' response to injury seems to build around 24th to the 48th hour and certainly, by the 11th day post-run, the tissues have well returned to either normal morphology or a repaired morphology compatible with life.

Description of Tissue Damage

The general effect of G stress upon each organ is summarized below.

Brain. - Injection of the brain is constantly present, increasing in severity as a function of increase G-time loads. Deepest injection occurs in the direction opposite the line of linear motion. At very high stress loads cranial sutures are disrupted with free blood exuding through the suture gap. The dura mater was rarely lacerated, but free blood was seen in the subdural spaces.

Lung. - Certainly the damage to the lung continues to be a prominent feature throughout all G ranges. In the $+G_x$ mode, the lungs' posterior bases showed heavy injection. In the $-G_x$ mode the anterior surfaces were most heavily injected and in $-G_y$ (eyeballs right) the right surface of the right lung, lateral surface, and the medial surface of the left lung were grossly injected; whereas in the $+G_y$ (eyeballs left) the left lateral surface of the left lung was injected and the left medial aspect of the right lung was grossly injected. Occasionally in the G_y modes one lung would be found atelectatic and the other emphysematous. Post run, the lung exhibits varying stages of inflammation which most often resolve or organize the lung damage. Occasionally lung damage is overwhelming, leading to pneumonitis and death.

Spared tissues. - Usually the urinary bladder, the testes, the prostate, ovaries, and uterus were spared. However, in the higher stress ranges a spermatic cord hematoma would be seen and slight injection of the other G/U tissues.

Cochlea. - The dura over the petrous bone was occasionally injected and, depending on mode, seemed to correlate with post run clinical observations of vestibular dysfunction.

Liver.- The liver seems to uniformly respond to damaging stress in all G ranges, with injection, frank hemorrhage or mechanical failure evident depending on the G-time load produced.

Adrenal glands.- The adrenal glands are also very responsive, with major changes evident between 24-48 hours.

Kidney.- The kidney grossly shows minimal response. At high stresses the capsule was found to be ruptured and petechiae were noted on the cortical surfaces.

The pancreas.- The pancreas routinely showed little response in the $+G_x$, $+G_y$, and $-G_y$ modes. In the $-G_x$ mode, however, instances of moderate to heavy congestion of the pancreas were seen with severity increasing with G load. Several instances of frank pancreatitis were seen. Rarely gangrenous pancreatitis was found and in one instance a pseudo-cyst of the pancreas was seen.

Spleen.- The spleen was moderately labile in $+G_x$, $+G_y$, and $-G_y$ modes. In the $-G_x$ mode the spleen was subjected to severe punishment, becoming edematous and congested and occasionally necrotic. Splenic damage tended to persist for long durations post-run.

Heart.- Grossly very little is notable about the heart, except that in non-survivors the heart is found to be in systole and usually devoid of blood. Often the auricles would be seen to continue beating while the ventricles were at standstill.

The intestinal tract.- The intestinal tract shows graded response generally; there have been stress ulcers noted sometimes immediately post-run, but most often within the 24-hour period in the $-G_x$ animals, rather than in the $+G_x$ animals; with increasing stress, both a straw colored fluid as well as free blood was found. Intraluminally petechiae were routinely noted.

Gastric ulcers.- The distribution of gastric ulcers was an interesting sidelight. In $+G_x$ mode they occurred only at 350 G, at $-G_x$ they occurred from 200 to 300 G, and in $+G_y$ they ranged from 250 to 400 G, while in $-G_y$ they ranged from 100 to 350 G.

Eye.- The eye is uniformly spared in the $+G_x$ animal; however, in the $+G_y$ (eyeballs left), the left eye becomes grossly discolored and hemorrhagic, whereas in the $-G_y$ (eyeballs right), it is the right eyeball that becomes enlarged and discolored. It appears that the nasal bones of the facial bone complex provide support for the opposite eye. In the $-G_x$ mode both eyes extrude forward, become heavily discolored, and engorged. However, when the animal's head is elevated 90^o, the floor of the orbital cavity protects the eye and minimal congestion of the bulbar musculature occurs.

Tongue.- Also in the $-G_x$ animal, changes were noted in the tongue with the tongue becoming 4 to 5 times its original size, occluding in many instances the original oral cavity. This, in many instances, accounted for the animal's death, due to its inability to move such a massive tongue and to respire. This size increase occurred when the animal's head was held in the anterior-posterior (A-P) direction; however, when the chin was elevated 90^o and placed against a flat plate such that the tongue was pressed forward (in the $-G_x$ direction) against the floor of the mandible, the tongue did not change in size.

Mechanism of Damage

The clinical responses of the animal to the various G loadings is discussed in another chapter. The grading of the gross pathologic changes was a most difficult task in that not only were the animals exposed to different G loadings, different dwell times, and different modes, but they were also sacrificed at different periods, post-run. Consequently, a simple grading system could not be employed very readily in the assessment of gross pathologic damage. However, the coded chart of tissue damage included as Appendix D reflects damage levels at a glance.

Lower G levels.- In the lower G levels it appears that the method of damage is due to circulatory and respiratory causes. Pooling of blood in the venous circulation probably produces a stagnant hypoxia which can be readily reversed if the animal survives the run. To what extent adreno-cortico-hormonal insufficiency plays a role could not be determined within the scope of this experiment. The responses to 50-150 G and 200-250 G

seem subtly different, with less evidence in the 200-250 G range that hydraulic failure is the only or principal cause of death.

Higher G levels.- As the G loading increases into the 300 ranges and beyond, mechanical failures begin to appear. Separation of cranial suture lines was noted. Extrusion of central nervous system tissue through the foramina, or a compression of these elements upon themselves may play an important role in the death of the animal.

Combined mechanisms.- Certainly it can be surmised that these mechanisms of damage interdigitate to varying degrees as the scale of G stress and dwell at G increases. However, it has empirically been surmised that if the animal has been subjected to stresses near or at tolerance values, in whatever G form, in the lower G levels it will apparently succumb to overwhelming congestion and anoxia of tissues; whereas, as we proceed to higher levels of G loadings and dwell times, mechanical, plastic deformation seems to be an added factor.

Repair of damage.- If the animal survives the run, axiomatically it is capable of reversing the major portion of damage sustained secondary to stress.

HISTOCHEMICAL STUDIES

A total of 220 animals was studied histologically. The number of slides studied were in excess of 1500; all principal tissues were studied histochemically. The 32 figures shown in Appendix E were selected as representative of the tissue damage observed throughout the entire range of G loadings and exposure times.

Histologic and Clinical Relationships

Certain animals died during the run. Others expired within a few minutes after the run and were labeled "cliff-hangers." These animals were seen to be breathing at the rate of approximately one respiration per second just at the termination of the run. Soon, however, respirations were noted to cease and the animal rapidly deteriorated and finally died. Subsequent to the exposure, the time to death was usually 5 to 10 minutes, but an occasional specimen would survive for a half hour or longer. Still another group

of animals followed a steady downhill course, finally expiring several days after exposure to G stress.

Survivals.- With few exceptions if the animals were able to survive the run, they went on to long-term clinical survival. These survivals were divided into four broad groups according to the elapsed time post-run to sacrifice. Sacrifices were routinely conducted 24 hours, 48 hours, and 72 hours post-run. A small group of animals was allowed to survive for more than 72 hours, and two were allowed to survive 3 months before sacrifice. One animal was held 7½ months for long-term recovery studies. By this means both short-term and long-term histopathologic changes could be studied. Table No. X included in Appendix A shows the time of autopsy and thus the distribution of the histochemical investigation, from immediate death to periods well beyond the original stress. Since initial observations of animals exposed to low G loads for short periods of time indicated rapid clinical recovery, it was decided to concentrate the bulk of the histologic studies to tissues prepared at times between 24 and 72 hours post-run. This schedule was altered in certain instances to examine a particular tissue response.

Autopsy and specimen preparation.- Those animals that died on the run were autopsied within a few minutes after the run record was complete. The autopsy was quickly accomplished. At no time did more than a half hour elapse after a run fatality until all of the tissues were in formalin. Run survivors were sacrificed as scheduled by the administration of 1 cc of pentobarbital followed by exsanguination by the cutting of the aorta, vena cava, or abdominal aorta just as the animal entered the second plane of anesthesia. Immediately after exsanguination, the tissues were placed in 10% formalin solution in preparation for further tissue- and staining-processing. All of the tissues listed on the necropsy-necrotomy protocol form shown in Appendix B were studied.

Routine H & E stain.- The stain employed for the routine preparation of most of the tissues studied was the Hematoxylin and Eosin stain (H & E) usually employed on formalin-fixed tissues or on tissues fixed on non-acidified Zenker's solution. Hematoxylin stains the chromatin of the nucleus a deep blue to black and thus imparts an overall intense basophilic coloration. The cytoplasm is stained pink to red (acidophilic) by eosin and provides a sharp color contrast for the darkly stained nuclei.

Temporal relationship of altered tissue morphology to stress.- As the histologic study of the tissues progressed and microscopic changes were noted in the tissues, the question was raised as to the temporal relationship of tissue damage to the run stress. It was not clear if the changes noted on the routine H & E stain were indicative of artifacts in the staining techniques, of old damage from previous disease, or, in fact, of changes produced by the G stress. For this reason it was decided that the H & E procedure alone was not enough and would have to be supplemented by and correlated with specially designed enzyme studies.

Enzymatic Study

The enzymatic study was utilized to identify, in any one tissue area, the immediate changes which occurred as a result of the stress exposure. Such changes are indicated by clumping of the enzyme in the tissue. Well progressed damage or irreversible old damage is indicated by depletion of enzyme from its otherwise normal locale and concentration. An experiment utilizing both oxidative enzymes and a special H & E technique for frozen tissue was constructed to augment the routine H & E histologic study.

Control animals.- Three animals were used: a "No Run Control," (No. 243) which at no time had been exposed to G stress; a "Run Control Animal," (No. 157) which had survived in excess of 3 months after exposure to +300 G_x for 100 seconds; and a "Rerun Control" (No. 241) which had survived in excess of 3 months after exposure to +200 G_x for 213 seconds. The Rerun Control was retagged as animal No. 242 and re-exposed to +200 G_x for 202.2 seconds. It survived the second run.

Anesthetic check.- As a cross-check against the effects of anesthesia, the following stipulations were inserted into this special study:

1) Animal No. 243, the No-Run Control, was given 1 cc of pentobarbital intraperitoneally. Just as the animal entered the second plane of anesthesia, it was exsanguinated.

2) The Run Control animal, No. 157, was given ether anesthetic for handling purposes, then tied down to the animal board, allowed to clinically recover from the anesthetic, and was then exsanguinated.

3) Animal No. 241 (No. 242) labelled the Re-Run Control was noted to be alive, although unresponsive to stimuli. It was tied down to the animal board and immediately exsanguinated.

Tissue handling procedures - Frozen section enzyme.- As each tissue for the enzyme study was removed it was individually wrapped in a piece of polyethylene film, placed in a sterile jar, and immediately transferred to a freezer. Upon completion of the dissections the jars containing the tissue specimens were placed in an aluminum foil lined box and packed with dry ice. The tissues were then immediately transported to the processing laboratory where they were replaced in a freezer. The frozen tissues were then cut at -25° Centigrade in a Linderstrohm-Lang cryostat microtome and mounted ready to be immersed into the substrate.

Brief statement of principle and technique.- The slides with fixed tissue mounted were incubated at 25°C., 15 minutes for DPNH diaphorase, and 30 minutes for malic, lactic, and succinic dehydrogenase. In the presence of appropriate tissue enzymes and added co-enzymes the tetrazoleum is reduced to form a lightly colored insoluble formazan which precipitates at sites of enzyme in tissue. The formulation for each enzyme substrate solution is given in Appendix B. The step-by-step protocol for the enzyme study and for the special H & E frozen section technique that was employed are also included in Appendix B.

HISTOLOGIC CONSIDERATIONS

Tissue damage observed was in some cases reversible and in other cases severe enough to proceed to necrosis of either part or all of the tissue. Considerations which constitute the basic guidelines against which the histologic response of these animals was measured are discussed below.

Necrosis.- Necrosis signifies cell death due to disease or injury. Necrosis can be considered from the following aspects: one, causes of cell death; two, morphologic alterations within the cell that indicate death; three, specific morphologic types of necrosis; and, four, physiochemical changes occurring within such cells. Causes of necrosis are discussed in some detail below. Morphologic alterations are outlined as part of the explanation of photomicrographs in Appendix E, and the other aspects are alluded to briefly in this section.

Causes of necrosis.- The many agents that cause necrosis of cells may be classified in five groups: one, ischemia or anoxia; two, physical agents; three, chemical agents; four, biologic agents; and five, others. Certain aspects of each of these five agents can be considered as contributing to the end results observed in these experiments. Each of these agents will be considered briefly.

Ischemia:- Loss of blood supply to an area leads to death of cells through deprivation of their oxygen and nutrients unless immediate collateral circulation is brought in from adjacent areas to satisfy their metabolic needs. Cell death due to ischemia (ischemic necrosis) is also known as infarction and is manifested by a characteristic histologic appearance that is called coagulation necrosis.

Anoxia played a major role as a cause of tissue injury in these experiments. Alterations of the ECG compatible with myocardial ischemia and injury were observed. Histologically, focal necrosis secondary to ischemia was observed in the heart and in the kidney; ischemic changes were also seen in the brain. Specimens taken from many of the test animals showed both blood deprivations as well as congested (hyperemic) tissue. For example, in a typical non-surviving $-G_x$ animal, the frontal lobes of the brain were hyperemic while the occipital lobes were devoid of blood.

In either case anoxia supervened, secondary to either blood deprivation or to stagnation of blood flow (stasis).

Physical agents:- Trauma may act directly upon the cell. Physical trauma to a cell causes rupture of cell membranes, destruction of the integrity of the nucleus, and total disruption of the normal relationships of cell elements. Trauma may also produce its effect indirectly by damage to the blood vessels supplying a tissue. Contused, lacerated, and crushed tissues were routinely observed, both grossly and histologically. The liver was a prime example as was the lung. The lung was particularly liable to trauma with atelectasis and emphysema noted. Gross hemorrhaging into tissues was commonplace. Some instances of retinal detachment were seen. Physical separation of skull suture lines was a feature at high G loads, although not examined histologically. In some animals fractures of the long bones were seen and some of the abdominal viscera were torn from their

attachments, particularly the pancreas and spleen.

Chemical agents:- Necrosis can also be caused by endogenous chemicals. The tissues of the body, of course, have varying sensitivities to specific chemical substances. Some of these substances demonstrate local effects at their points of absorption or excretion; that is, where their concentration is highest. The kidney tubule or the gastric mucosa are examples of such areas. Endogenous products of metabolism may be toxic to tissues and may lead to death of cells by their accumulation in high concentration. This process has been called autointoxication. Autointoxication is also frequently spoken of in reference to the absorption of breakdown products of large areas of destruction of tissue.

The pancreas was occasionally seen to reflect changes compatible with acute pancreatitis; rarely, an acute peritonitis was also in evidence. The spillage of highly toxic pancreatic enzymes secondary to trauma is certainly implicated.

A pseudocyst of the pancreas was observed in animal No. 232 which was autopsied 25 days after exposure to -400 G_x. Localized bile induced peritonitis secondary to leakage of bile through a lacerated liver bile duct was seen, although rarely. Endogenous chemical deprivation is a probable feature of tissue injury. Alterations in lipid content of the adrenal glands point to hormonal insufficiency. Since bioassays of adrenocortical products are placed in Phase II of this experimental plan, the exact physiologic dysfunction of the adrenals based on histologic alterations observed is largely speculative at this time.

Stress:- Certain changes were noted which support other findings reported in the literature.

Adrenal gland necrosis with simultaneous findings of a gastric ulcer were noted in the animals shown in Table IV below.

Less obvious indications of adrenal gland necrosis were seen in other animals. See Appendix D.

TABLE IV

ANIMALS WITH ADRENAL GLAND NECROSIS AND GASTRIC ULCER

Animal Number	Stress nG	Dwell D_r	Autopsy at
#159	+350 G_x	171 sec.	24 hours
#201	-200 G_x	119 sec.	48 hours
#212	-300 G_x	120 sec.	24 hours
#210	-300 G_x	121 sec.	48 hours
#117	+250 G_y	203 sec.	24 hours
#134	+400 G_y	152 sec.	24 hours
#122	+400 G_y	139 sec.	24 hours
# 86	-100 G_y	200 sec.	24 hours

Biologic agents:- Worm infestations, viruses, bacteria, etc., release endotoxins or ectotoxins which are also lethal to cells. Among the test subjects of this experiment, such agents are common. Heavy worm infestation was seen, in spite of control measures. Subsequent to stress exposure, some animals developed a severe pneumonitis and respiratory insufficiency, resulting in death. Saprophytic bacteria, finding a fertile medium for growth in damaged lung tissue, may have been partly responsible for the death of these animals, secondary to pneumonitis.

Summary of Routine H & E Studies

Histochemical studies utilizing the routing H & E stain formed the bulk of the microscopic investigations. The results of these studies correlated very well with the gross pathologic observations. In addition, insight was gained into the degree and types of damage sustained at cellular level. A brief summary of microscopic findings by tissue type follows.

Lung.- Hyperemia, atelectasis, acute emphysema, extravasation of blood into alveolar spaces and into supporting structures, pneumonitis, filiriasis, and non-specific acute inflammatory changes.

Heart.- Hyperemia, focal necrosis, myocardial infarction, and

rare worm infestation.

Gastro-intestinal tract (GI).- Congestion, petechial hemorrhages, intraluminal fluid, and extravasation of blood into the lumen ascending in that order of severity. Post-run gastro-duodenal ulceration, with perforation and hemorrhage; gangrene; non-specific acute inflammatory changes; heavy tapeworm infestation of the large bowel and heavy hookworm infestation of intra-peritoneal spaces.

Pancreas.- Congestion, non-specific inflammatory changes, occasional laceration, and disruption of cellular continuity.

Kidney.- Congestion, focal necrosis, and rarely, extravasation of blood from glomerulus.

Adrenals.- Hyperemia, focal necrosis, and lipid depletion.

Central nervous system (CNS).- Congestion, extravasation of blood into subdural and subarachnoid spaces at very high G stress.

Eye.- Congestion, hemorrhage and edematous musculature and periorbital tissues, and occasional retinal detachment.

Tongue.- Laceration, heavy congestion in $-G_x$ animals, less hyperemia in $\pm G_y$ animals, and no changes in $+G_x$ mode.

Skin.- Laceration, hyperemia, non-specific acute inflammatory changes, and abscesses.

Bone.- Fracture of long bones, and separation of skull sutures with extravasation of blood.

Genito-Urinary (G/U).- Tissues of G/U tract were devoid of findings at low G levels. At high G's hyperemia, laceration of ligamentous structures were featured; a hematoma of the spermatic cord was often seen.

Cochlea.- Grossly, the cochlea showed few changes. Clinically the animals were coordinated and oriented by 24 hours. The cochlea was not extensively studied. Several samples were retained for later examination; preliminary results showed hyperemia and edematous changes.

Representative microphotographs of samples of some of the above tissues are in Appendix E.

Summary of Histochemical Studies on the Brain, Heart, Kidney, and Adrenal Glands

Histochemical studies using the special frozen section enzyme technique were conducted on the brain, heart, kidney, and adrenal glands from three animals, No. 157, No. 243, and No. 241-242. Four histochemical studies were performed: DPNH diaphorase, lactic dehydrogenase, malic dehydrogenase, and succinic dehydrogenase.

The best results seen on these tissues were, in descending order: first, DPN-DPNH diaphorase; second, malic dehydrogenase; third, succinic dehydrogenase; and, fourth, lactic dehydrogenase.

The "No-Run Control" studied showed normal enzymatic localization in the cytoplasm of cells in the brain, myocardium, renal tubules, and renal cortex.

There were no distinct abnormalities of the "Run Control" in comparison to the "No-Run Control."

The "Rerun Control" showed distinct abnormalities with an obvious clumping and depletion of enzyme in the cells of the cerebral cortex and medullary sites. There was clumping and minimal depletion of enzyme (especially DPN and malic and succinic dehydrogenases) in the cytoplasm of the myocardial cells. There was obvious depletion of enzyme in proximal and distal convoluted kidney tubules in the "Rerun Control" which corresponded to the findings of focal necrosis of the tubules in routine stains. There was minimal depletion of enzymes of the renal cortex in the "Rerun Control" in comparison to the "No-Run Control", but there is no evidence of clumping. Comparison of the Sudan black stains of the adrenal cortices in the "No-Run," the "Run," and the "Re-Run Control" shows obvious lipid depletion in the "Re-Run Control".

Preliminary results of histochemical and special staining of the vital organs of the simians demonstrate a loss of enzyme and clumping of enzyme in the "Re-Run Control" animal in the brain, heart, kidneys, and adrenal. The method of anesthesia and sacrifice did not appear to influence the histochemical results.

MEASUREMENTS

The morphologic characteristics of S. sciureus were investigated at the beginning of this study. A review of the literature showed extremely limited data on the subject, although the investigators are aware that this animal has been used in research for many years. Hill (11) was the best source uncovered, but detailed body information was not included. Some organ weights were found (12) but the overall lack of data was startling. Since both anthropometric and physiologic information was required to assure that the existing equipment was adequate for the study task and to develop data for future extrapolation scaling factors, two side experiments were conducted in the course of the basic task; basic body requirements and a tail weight study.

Basic body measurements.- Measurements of four female and four male specimens were taken from the initial colony of monkeys. The dimensions obtained were required for the design of the forward restraint plates of the couch. The animals were chosen to cover the maximum available spread of sizes at the time. The results are tabulated as Table V.

To supplement the dimensional data and provide an accurate overall configuration, Animal No. 26 was X-rayed showing lateral and anterior-posterior views, displayed as Figure 16. Further utilization of the X-ray was to obtain an approximation of total lung volume (for use in the ether anesthetic experiment) as well as the organ configuration and general location of the heart for purposes of electrode configuration, dimensions and optimum placement.

These data compare favorably with that displayed in Table VI.

Tail weight study.- Details of the tail weights obtained are included in Appendix A for each animal measured. A total of 134 animals was studied, covering a weight range of 285 to 837 grams, and including both male and female specimens. The average tail weight by percent of total weight was 5.60%.

Weight distribution.- Charts of the weight distribution in groups of 25 grams span of 87 male and 143 female animals are included as Figures 17 and 18.

TABLE V

MEASUREMENTS OF VARIOUS SQUIRREL MONKEYS

Measurement	ANIMAL NUMBER							
	34	35	36	43	44	46	50	51
Sex	F	M	M	F	F	M	F	M
Date Measured	19 Feb.	19 Feb.	19 Feb.	2 Mar.	2 Mar.	2 Mar.	8 Mar.	8 Mar.
Run Weight, gm	508	836.5	704	469.4	429.5	423.7	479.6	469.2
Sacrifice Weight, gm	-	-	-	428.2	394.6	387.4	496.8	415.5
Forehead-Back of Head	2-1/4	2	2	2	2	2	2	5.5cm
Back of Head-Muzzle	2-1/2	2-1/4	2-1/2	2-3/8	2-3/4	2-3/4	6.5cm	
Back-Chest	2-1/2	2-1/2	2-1/2	2	2-1/2	2-1/4	6.0cm	5.5cm
Back-Navel	1-1/2	2		1-1/2	1-1/2	1-3/4	3.5cm	4 cm
Ear-Ear	2	2-1/4	2-1/4	1-1/2	1-3/4	1-3/4	4.5cm	4 cm
Seat-Muzzle	8-3/4	10-1/4		9-1/2	9-1/8	8-3/8	9-1/2	
Seat-Top of Head		11-1/2		10-1/2	10-1/4	9-3/4	11	
Shoulder-Seat				8-1/2	8	8-3/4	8-1/2	
Thigh	1-1/4	1-5/8	1-1/4	-	-	-	-	
Chest Width-Shoulder to Shoulder	2-1/2	3-1/4	3	-	-	-	-	
Hip-Side to Side	2-1/2	2-3/4	2-1/2	-	-	-	-	
Belly-Side to Side	1-1/2	2	2	-	-	-	-	

Note: All dimensions in inches unless noted.

Animal No. 26
Wt. 734 grams

FIGURE 16. FULLSIZE X-RAY, P-A & LATERAL VIEWS

TABLE VI

MORPHOLOGICAL DATA ON THE SQUIRREL MONKEY
After Carmichael and MacLean (14)

Measurement	Number of Animals	Mean	Standard Deviation
Weight	76	717.0 g	± 170.40
Body length	49	26.0 cm	2.47
Tail length	50	36.0 cm	2.14
Total length	48	63.0 cm	3.57
Head length	45	5.4 cm	0.22
Head width	45	4.1 cm	0.36
Brain weight	21	26.0 g	1.72
Brain length	29	4.9 cm	0.21
Brain width	30	3.5 cm	0.71

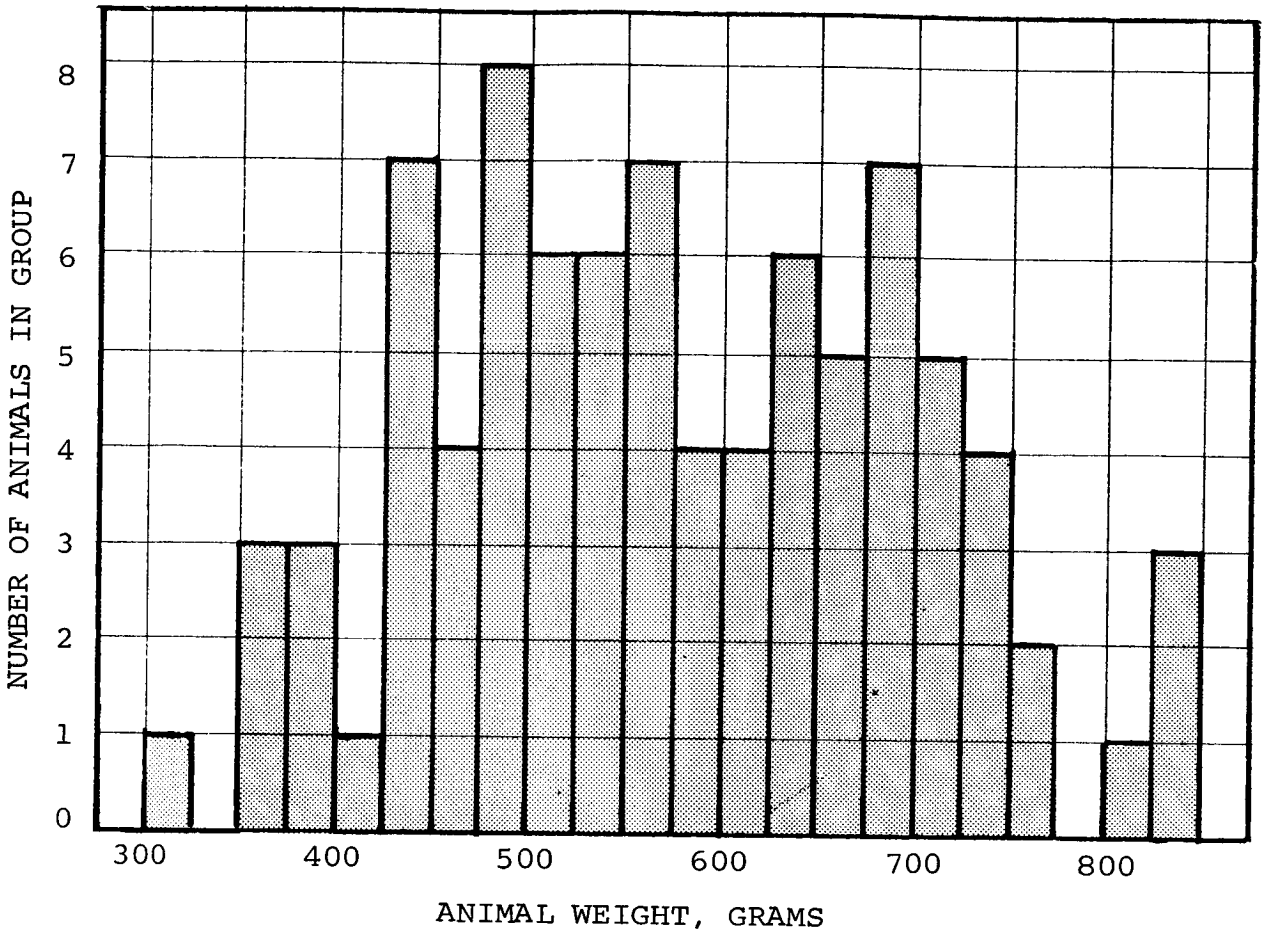

FIGURE 17. DISTRIBUTION OF 87 MALE ANIMALS IN GROUPS OF 25 GRAMS SPAN

Figure 17 shows a double peak, occuring at 475 grams and 675 grams for males. The minimum weight recorded for young (undeveloped) males was 313 grams, the minimum for mature males was 353 grams. There were three specimens weighing between 836 and 837 grams as maximum.

Figure 18 shows a typical Gaussian distribution for the female members of the experimental colony, peaking at 530 grams. The minimum weight recorded for a young female was 285 grams, for an adult female was 367 grams. The maximum was 723 grams.

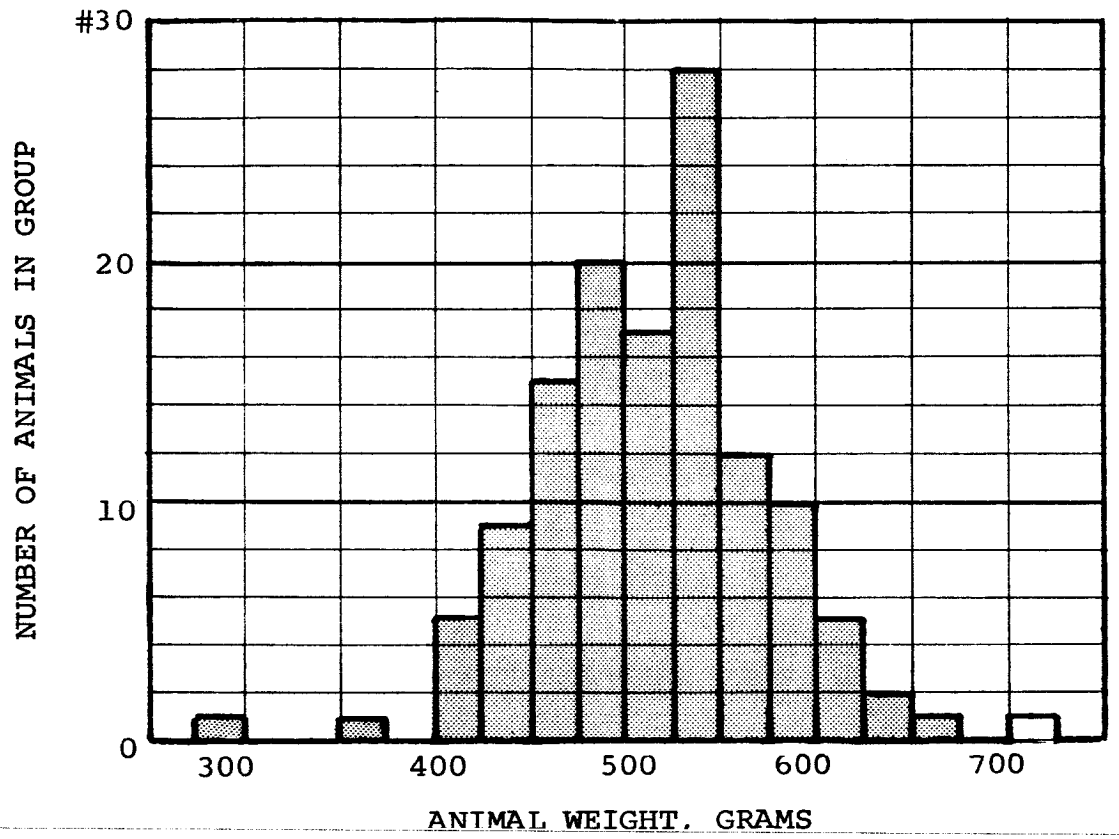

FIGURE 18. DISTRIBUTION OF 143 FEMALE ANIMALS IN GROUPS OF 25 GRAMS SPAN

The weight of a female squirrel monkey (Baker) was recorded over a 4 year period,(12), as shown in Figure 19. The plateaus before and after gestation have a remarkable coincidence with the maximum groups shown in Figure 18. Figure 19, although referring to a single animal, indicates that the 450-550 gram weight group may be safely described as mature females.

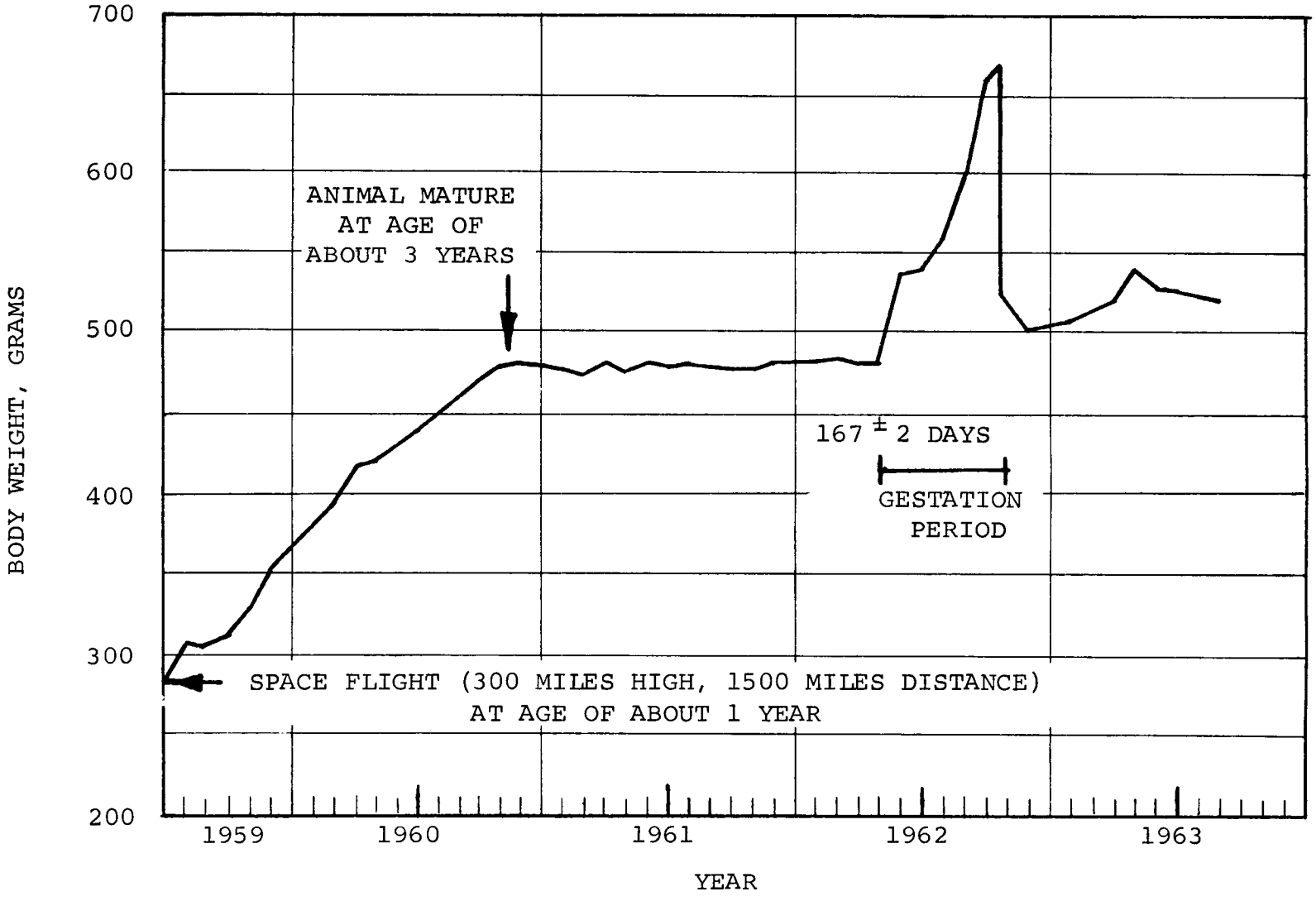

FIGURE 19. BODY WEIGHT OF ONE ANIMAL OVER 4 YEARS
(AFTER BEISCHER AND FURRY)

MATHEMATICS

In the past it was generally considered inconceivable that any mammal could survive G stress forces of the magnitude designed into this experiment. Therefore, the test cells for the experiment as described in an earlier chapter of this report were surveyed for an overview of the results to be expected. After the successful completion of the first few test cells it became surprisingly apparent that this particular animal could, as postulated, survive G stresses far in excess of those previously tested.

The results of the first few runs were compared and a simple survivability chart by G mode, as illustrated in Table VII, was constructed. This initial chart showed that in the $+G_x$ mode there seemed to be no apparent limit to the level of G stress that this animal could absorb for a two-second period. An analysis of the 20-second dwell line indicated that the animal could absorb unlimited stress within the constraints of this experiment at 20 seconds.

It was not until the 200-second dwell section of each test cell was approached that interesting results began to occur. It was noted that serial animals were able to survive 50 G's at 200 seconds, 150 G's at 200 seconds, 200 G's at 200 seconds, and 250 G's at 200 seconds, whereas at 300 G's, 200 seconds, the first loss was recorded and at 350 G's at 200 seconds the second loss was recorded.

However, and most interestingly, an exceptional loss of Animal No. 39 occurred at 100 G's, 200 seconds. This was a most important loss because (as indicated by Table VII) this fatality was bracketed by one survivor below 100 and by three survivors above the 100 G stress level at 200 seconds. This loss was unexplainable. A search was made for a possible explanation. The several parameters having to do with the animal, the test equipment, the mode, etc., were compared against each other for any possible correlative relationship that might be evolved. The various parameters are described below.

TABLE VII

INITIAL SURVIVABILITY LIST BY MODE

	G_x AXIS TIME, SECONDS			nG	G_y AXIS TIME, SECONDS			
	2	20	200		2	20	200	
	+	+	o	400	o	+	o	
	+	+	-	350	+	+	o	
	+	+	-	300	+	+	o	
	+	+	+	250	+	+	-	
$+G_x$	+	+	+	200	o	o	o	$+G_y$
	+	+	+	150	+	+	-	
	+	+	(-)	100	o	o	o	
	+	+	+	50	+	+	+	
	+	+	+	50	o	o	o	
	+	+	-	100	+	+	+	
	+	+	-	150	o	o	o	
	+	+	-	200	+	+	+	
$-G_x$	+	+	-	250	o	o	o	$-G_y$
	o	+	o	300	+	+	+	
	o	-	o	350	+	+	-	
	o	o	o	400	+	+	+	

+ = Survivor, - = Fatality, o = No Test

(-) Animal 39, Inconsistant Loss

Parametric Envelope

The parameters involved in this experiment were diverse and complex. Among them were:

1. External physical parameters which included humidity, temperature, baropressure, noise, light;

2. Machine parameters which included direction of force application, i.e., G mode, cross axis forces - transverse G or Coriolis effect, rate of onset of force, rate of offset of force, vibration;

3. Dynamic parameters which included level of force - nG, duration of exposure to stress - dwell, weight of animal;

4. Physiologic parameters which included age, health of animal, sex of animal, possible states of gestation of female, weight distribution, tail weight effect, randomicity of the colony, idiopathic capacity to absorb stress, individual variations within species;

5. Others such as conditional reflexes of animal to previous experience, fright, and still others not readily identifiable.

A rapid survey of these parameters indicated that they most probably could be expressed as series of percentage values which in some fashion would alter the overall response of each of the animals to the stress.

Integration of the Parameters

Each parameter has an effect on the response of the test specimen to G stress. The exact effect of each parameter was unknown. The letter K was arbitrarily chosen to represent the total product of the effects of all of these parameters. Therefore K could be expressed as follows:

$$K \propto p_1 \times p_2 \times p_3 \times p_4 \times \cdots p_m \qquad (1)$$

where K is proportional to the total product of the parameters and where arbitrarily:

p_1 = parameter 1 = w — weight, in kilograms

p_2 = parameter 2 = n — G units

p_3 = parameter 3 = D_r — dwell time, seconds

p_4 = parameter 4 = $(\pm G_x, \pm G_y)$ — mode or axis of stress

p_5 = m^{th} parameter = etc.

Elimination of Certain Parameters from Initial Proportion

It soon became apparent that several of these parameters were either constant or could be arbitrarily fixed and could therefore be ruled out of the initial proportion by setting them equal to one.

The physiologic factors were grouped together and held in abeyance. It is felt that although the sample was too small for an accurate determination of these factors, they do have a small but significant percentage value and should become determinable at a later time when more data is available.

The machine factors, such as mode, rate of G onset, and rate of G offset, etc., are fixed constants for each series of runs and, therefore, they could be considered as equal to unity. Their overall influence on the basic expression for K could be determined later when more data was available.

Physical and other factors were also set equal to unity for the first approximate determinations. They were recorded, however, for possible future considerations.

Therefore all parameters with the exception of the dynamic factors were eliminated from the proportion such that a first approximation of the absolute value of K could be obtained. The expression for K then reduces to:

$$K = b\ W(n)\ t \qquad (2)$$

Where b = a constant of proportionality, initially setting b equal to unity, Equation 2 becomes:

$$K = Wnt \qquad (3)$$

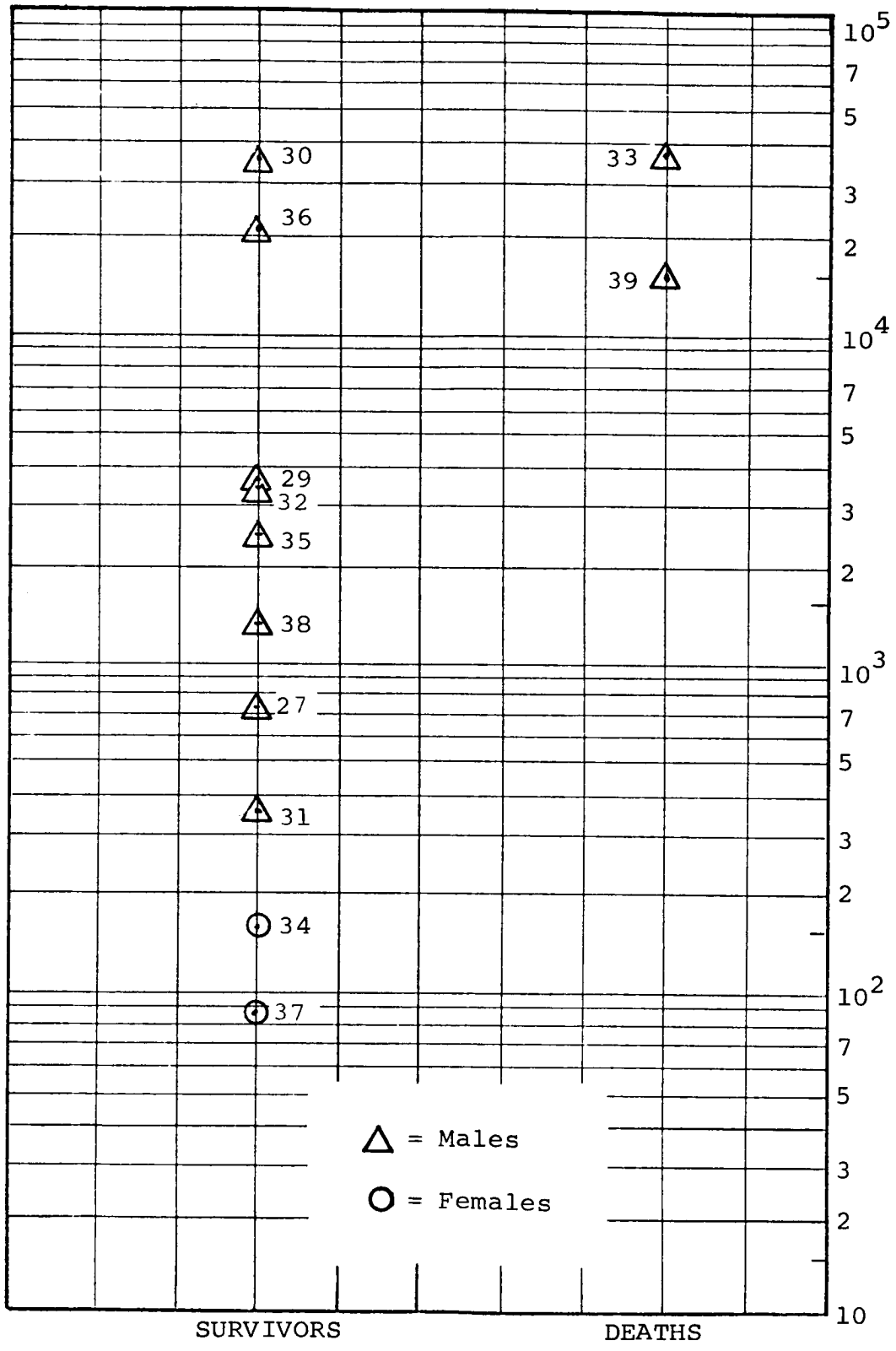

FIGURE 20 SURVIVABILITY - ANIMALS 27 THROUGH 39
$+G_x$ MODE VERSUS K

animals were exposed, and thus the animal fell into the assumed 50% lethality (LD_{50}) zone. It was hypothesized that this was a method to explain No. 39's death. Beginning with Animal No. 40, Equation 3 was applied to each animal prior to the experimental run, and the predicted outcome as well as the experimental outcome was recorded in the experiment log book. The results for Animals No. 40 through No. 51 tended to confirm the hypothesis set forth above.

Graphs by mode.- The $+G_x$ mode graph (Figure 21) was constructed, covering both the groups of No. 27 through No. 39 and No. 40 through No. 51. There were three losses and three survivals above the 10^4 line; all points below this line were survivors. The distribution of survivors and fatalities between K values of 1.5×10^4 and 4.0×10^4 was approximately equal with a maximum 3.5×10^4 survival recorded. No females were tested above $K = 10^4$ in this group.

As the $-G_x$ tests began, the first constraint for the rule of thumb, which assumed 100% survivability below $K = 10^4$ appeared to fail. As 17 animals were studied in this mode, the values were plotted (Figure 22) for $-G_x$ on the same basis as for $+G_x$. There were 12 survivors, all below 4.0×10^3, and 5 losses falling on or above this value. A sharp demarcation occurred between 3.5×10^3 and 4.0×10^3, separating survivals and fatalities. Although the series was small, the probability that the "threshold K" value for the $-G_x$ mode might be smaller than that for $+G_x$ became significant.

As 14 animals were studied in the $+G_y$ mode, a similiar graph (Figure 23) was constructed. There were two losses, both above the 10^4 line, and comparable to the $+G_x$ mode. A fairly large gap existed between survivals and fatalities, with all 12 survivors well below the 10^4 line. The decreased number of losses in $+G_y$ (as compared to $+G_x$) was not considered greatly significant, because of the smaller sample tested.

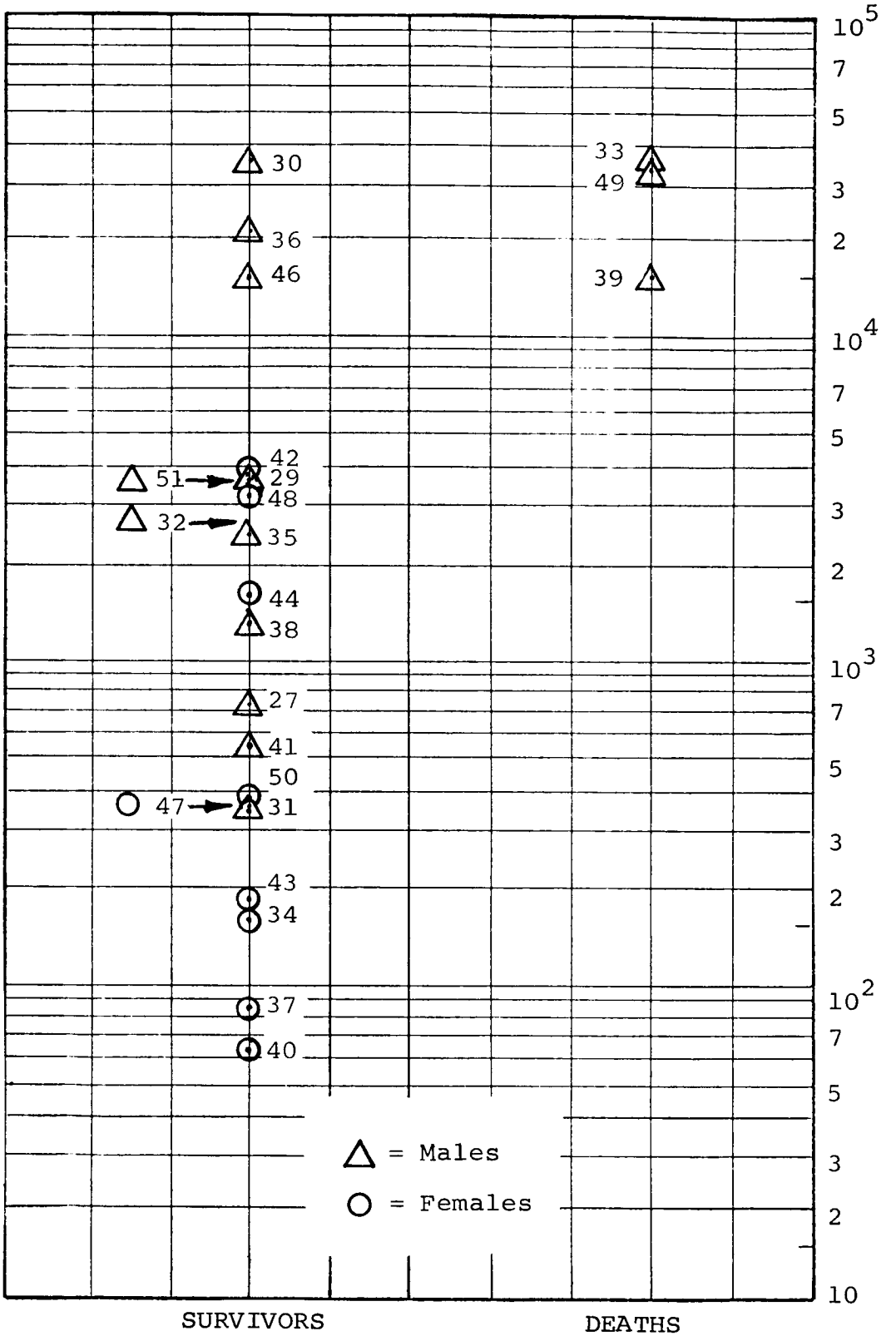

FIGURE 21 SURVIVABILITY - ANIMALS 27 THROUGH 51
$+G_x$ MODE VERSUS K

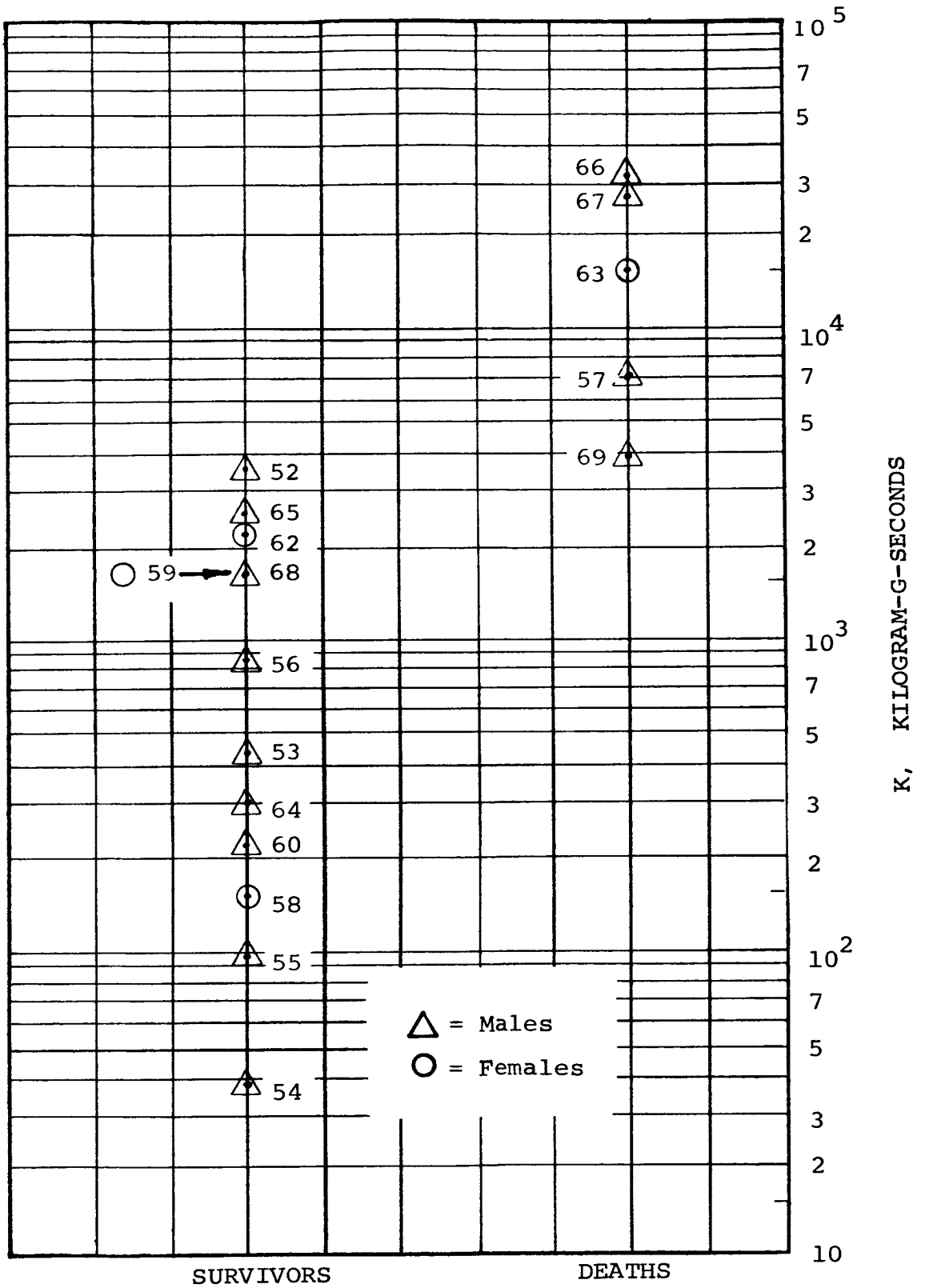

FIGURE 22 SURVIVABILITY - ANIMALS 52 THROUGH 69
$-G_x$ MODE VERSUS K

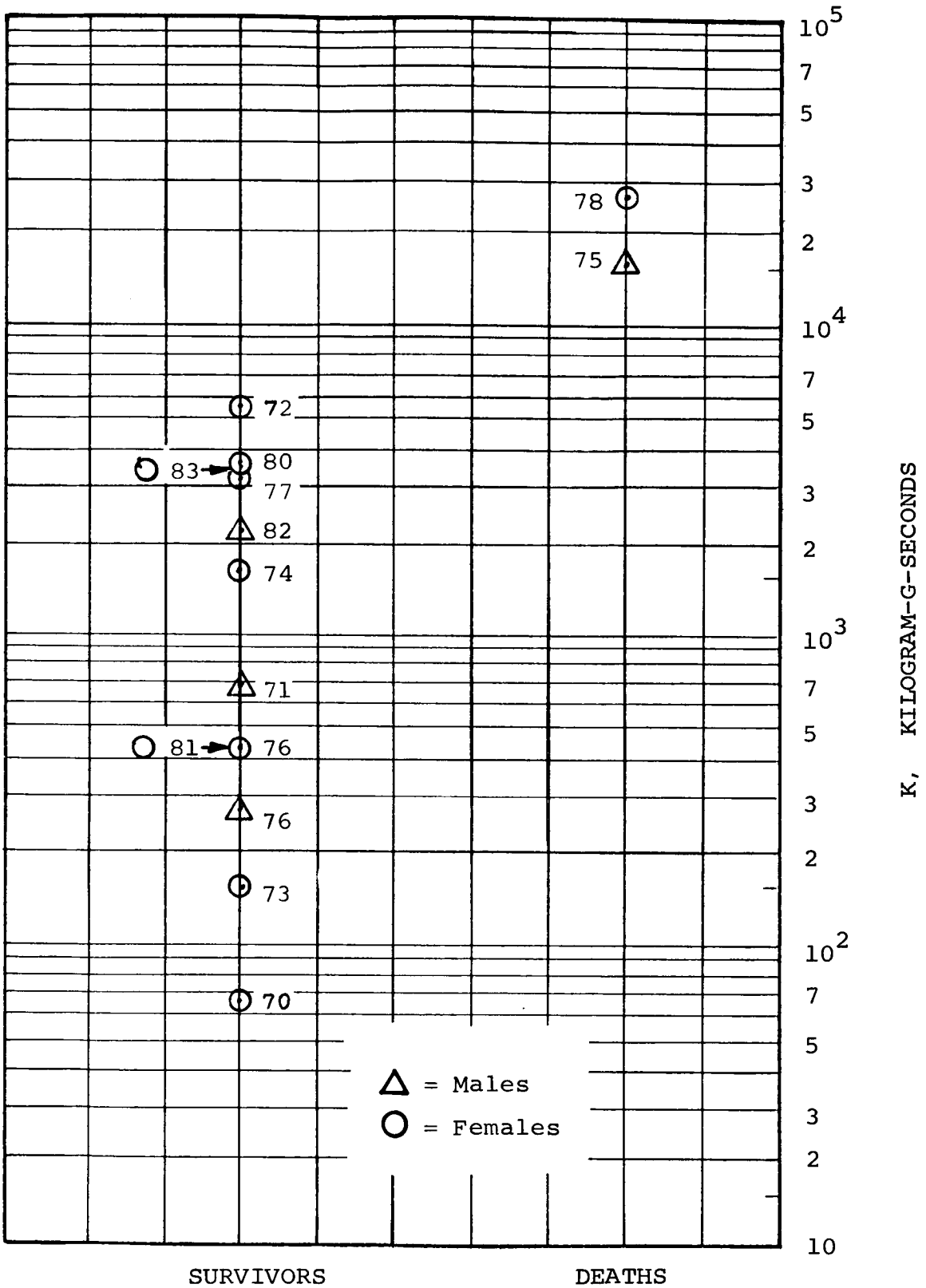

FIGURE 23 SURVIVABILITY - ANIMALS 70 THROUGH 83
$+G_y$ MODE VERSUS K

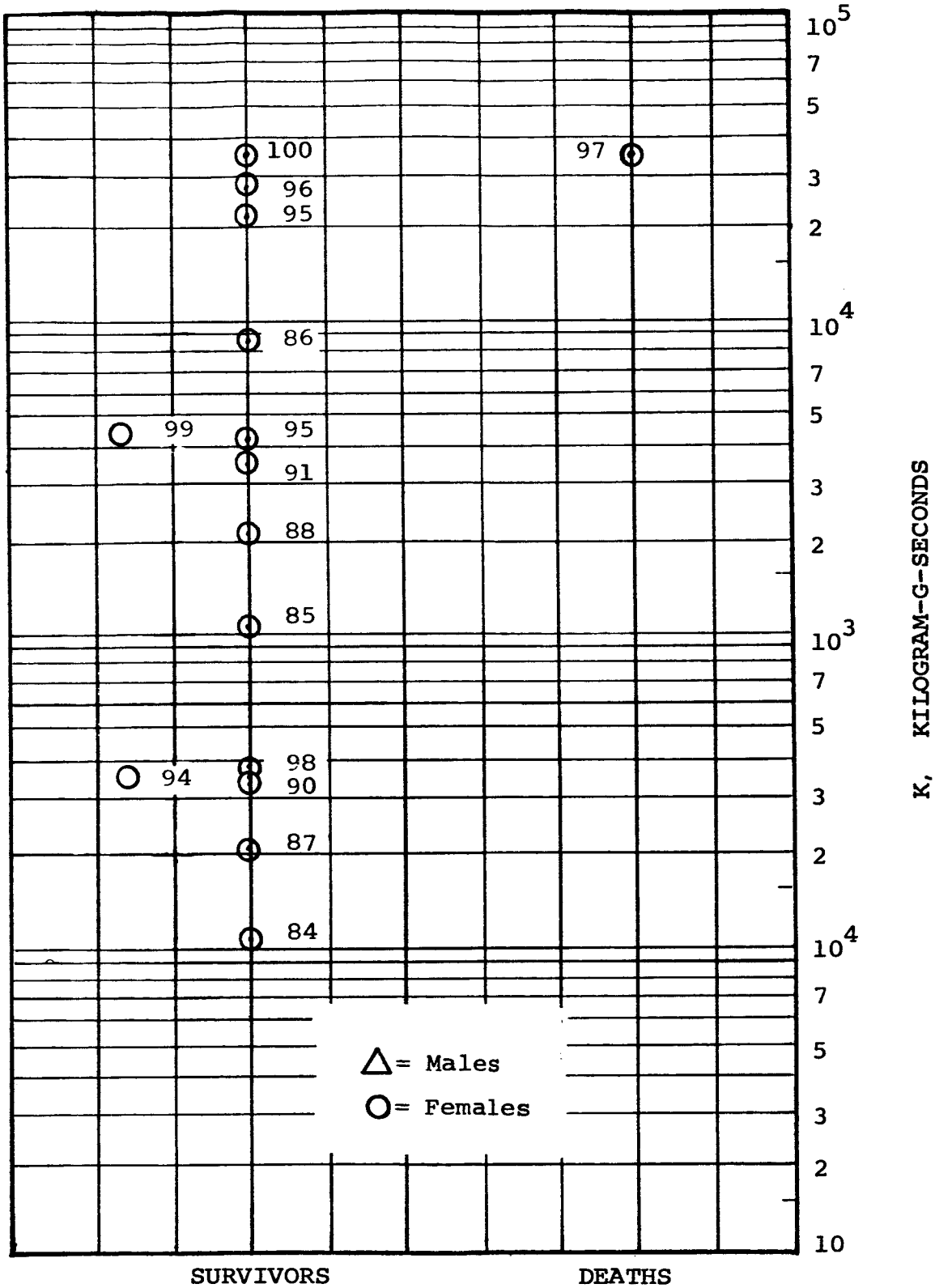

FIGURE 24 SURVIVABILITY - ANIMALS 84 THROUGH 100 $-G_y$ MODE VERSUS K

The $-G_y$ graph (Figure 24) was constructed as 16 animals were tested, recording 1 loss and 3 survivors above the $K = 10^4$ line. The remaining 12 survivors all plotted below this line. The values recorded for Animal No. 100 and No. 97 are noteworthy. Animal No. 97 did not survive at 3.64×10^4, yet No. 100 did survive at 3.65×10^4, one of the highest values recorded in the entire study. This interesting comparison is discussed further below. By chance, all the animals tested in this group and mode were female. Additional testing using male specimens would be necessary to determine if the indicated tolerance is germane to both sexes.

Since all of these survivability plots (Figures 21 through 24) use the same scales and notations, direct comparisons can be made between modes. The animal number is indicated by each point so that reference can be made to Appendix A for the basic data for each specimen if desired.

Threshold Concept and Event Marker

Because of the experimental observation that animals exposed to K values above 10^4 entered into a zone of increasing peril to life, the value $K = 10^4$ was termed the "Threshold K" or "K_t".

Each animal, of course, had a different weight and since K_t was set equal to 10^4, the selection of nG and the animal's weight determined the number of seconds necessary to achieve "threshold" or K_t. A special event marker was entered on each ECG to indicate the moment of dwell at which K_t was achieved. Any dwell time exceeding this value was considered as an additional exposure to stress in the zone of increasing lethal dosage ($K > 10^4$ kg - G - seconds).

This was needed as a laboratory working tool for the determination of the degree of stress to which each animal would be exposed. A mathematical basis was thus available to control and evaluate exposures above K_t.

The value $K_t = 10^4$ was selected empirically (see Figures 19, 21, 22) as threshold for all modes except $-G_x$. For $-G_x$ mode, laboratory data up to that time (see Figure 20) indicated that the K_t value for $-G_x$ mode should be chosen as equal to 4.0×10^3 rather than 1.0×10^4.

Subsequent experiments showed, however (as indicated by the results of Animal No. 202 which achieved a value of 1.67×10^4 and survived), that this was a low estimate probably based on the artifactual death of early $-G_x$ animals (by extrusion through the head restraint plate). Later in the experiment other animals achieved higher K values than 4.0×10^3 in the $-G_x$ mode and survived. The difference in the results was basically due to an improved method of constraint of the animal.

The type of constraint in the early aspects of the experiment of $-G_x$ mode held the animal's head in an anterior-posterior direction with the chin horizontal, parallel to the horizontal plane. This resulted in early strangulation of the animal. The couch configuration was changed to rotate the head upwards 90^o and furnish support for the chin. This change alone perhaps explains the higher values of K achieved by animal No. 202 and others in the $-G_x$ mode. Therefore, as indicated by Figures 164 to 172 (Appendix F), the maximum values for K for $-G_x$ ranged between 7.0×10^3 and 2.8×10^4, and the initial empirical value selected for K_t in the $-G_x$ mode was conservative.

Discussion of the Mathematical Curves

Similarities of two animals compared.- On April 28, 1964, it was noted that one of the $-G_y$ animals (No. 100) had a calculated value for K of 3.65×10^4. Another, Animal No. 97, was lost on the run with a calculated value for K of 3.64×10^4. Both were female. Both were $-G_y$ mode animals.

Similarly, Animal No. 97 entered the "threshold zone" as explained above at 55 seconds of elapsed dwell time with 145 seconds of exposure beyond "threshold", whereas Animal No. 100 entered the "threshold zone" at 54 seconds, 1 second earlier. No. 100 remained in the "zone of peril" for 146 seconds. These were similar exposure times.

Differences.- One difference in the two animals was the weight. No. 97 weighed 521 grams whereas No. 100 weighed 456 grams, a difference of 65 grams. The G load difference was 50 G's with No. 97 run at $-350\ G_y$ and No. 100 at $-400\ G_y$.

G load and animal weights were implicated parameters which could explain the difference. Animal No. 97 weighed more, but was stressed to a lower nG than No. 100, and conversely, No. 100 weighed less, but was stressed to a higher nG than No. 97. Analysis of results based on the non-integrated parameters would be difficult.

Math analysis.- The results experienced by the two animals could, however, be conveniently explained from the mathematical point of view in that experimentally there occurred an LD_{50} at a K value of 3.64×10^4.

Refinement of the simple "rule of thumb."- The gratifying results of the mathematical comparison of the above two animals stimulated the refinement of the "rule of thumb" as outlined below such that all subsequent experiments could be analyzed mathematically.

Threshold K.- Since the numerical value for K of 1.0×10^4 was designed at K_t the basic equation:

$$K = Wnt \tag{3}$$

was rewritten as:

$$K_t = W_o \times G_o \times D_t \tag{4}$$

where W_o is equal to the weight of the animal in kilograms, G_o is equal to the G stress and D_t is equal to the time in seconds at G_o necessary to reach K_t. The onset period was ignored in these assumptions.

Solving for D_t:

$$D_t = \frac{K_t}{W_o \times G_o} \tag{5}$$

Peak K.- The peak value of K, to which any animal would be exposed, was designated as K_p. Substituting symbols, Equation (3) becomes

$$K_p = W_o \times G_o \times D_r \tag{6}$$

where K_p equals the peak value of K for that run, W_o is equal to the weight of the animal in kilograms, G_o is equal to the G stress, and D_r is equal to the total dwell duration of the specific run, in seconds. Solving Equation 6 for D_r:

$$D_r = \frac{K_p}{W_o G_o} \tag{7}$$

Exposure time above threshold.- The difference between D_r and D_t defines the time, in seconds, of exposure to stress above K_t. Setting

$$\Delta_{rt} = D_r - D_t \tag{8}$$

and subtracting Equation 5 from Equation 7 and substituting

$$\Delta_{rt} = \frac{K_p}{W_o G_o} - \frac{K_t}{W_o G_o} \tag{9}$$

$$\Delta_{rt} = \frac{1}{W_o G_o} \left(K_p - K_t \right) \tag{10}$$

Working curves.- Now Δ_{rt} in seconds, G_o in stress, and weight in grams could be utilized as data to plot a series of graphs. For each value of G_o selected for the test cells a curve was plotted of Δ_{rt} versus weight. Tables of values of Δ_{rt} were constructed for a family of curves based on the initial test cell constraints of 2, 20, and 200 seconds. These graphs are included as Figures 148 to 163 in Appendix F.

Sample solution.- Each experiment from Animal No. 101 to the termination of the experiment was treated as follows: The animal was weighed and the G level was selected according to the test cell plan. Then a calculation for K_t would be made (data from Animal No. 241):

$$D_t = \frac{K_t}{W_o \, G_o} \tag{5}$$

$$D_t = \frac{1.0 \times 10^4}{.601 \times 200} = 83.0 \text{ seconds}$$

where

$K_t = 1.0 \times 10^4$ kg - G - Sec., $W_o = .601$ kg and

$G_o = 200$ G's

Empirical selection of Δ_{rt} value.- Following the calculation for D_t the experimenter went to Appendix F for No. 241 and the Δ_{rt} value for the animal was empirically selected based on previous run results in that area of weight.

Since extrapolation between previous run results indicated questionable survivability at $\Delta_{rt} = +130$ seconds for animals weighing from 550-650 grams, this value for Δ_{rt} was selected. From Equation 8 it was then possible to calculate the number of seconds for the duration of the new experimental run.

In this example:

$$D_r - D_t = \Delta_{rt} = +130$$

Solving for D_r, when $D_t = 83.0$ sec.

$$D_r - 83 = 130$$

$$D_r = 213 \text{ seconds}$$

Solution for K_p value:- Substituting in Equation 6, the values for W_o, G_o, and D_r given above and solving:

$$K_p = .601 \times 300 \times 213 = \underline{2.56 \times 10^4 \text{ kg - G - sec.}}$$

Data from Animals No. 27 through No. 100 were used to plot K_p versus weight for each G stress level. Data from $+G_x$, $+G_y$, and $-G_y$ were combined, while $-G_x$ data was used to prepare separate plots.

K_p curves.- Examples of these plots for 50 G and 400 G are included as Figures 25 and 26. Straight lines were drawn through the fatalities.

Figure 25:- The straight line connecting fatalities slopes upward from left to right. The inference is drawn that as the animal's weight increases so does its capability to absorb stress at 50 G's.

Figure 26:- This figure illustrates that the optimal weight for the animal to absorb stress at 400 G's appears to be between 500 and 550 grams. To the left of this region the capability to absorb stress falls off sharply, whereas to the right of the optimal weight range, the capability of the animal to absorb stress values falls less dramatically.

Flexibility of parameter value selections.- It can readily be seen that at one time it might be desirable to select a value of Δ_{rt} for a particular run and at another time it might be desirable to select a value of K_p for a particular run. Consequently

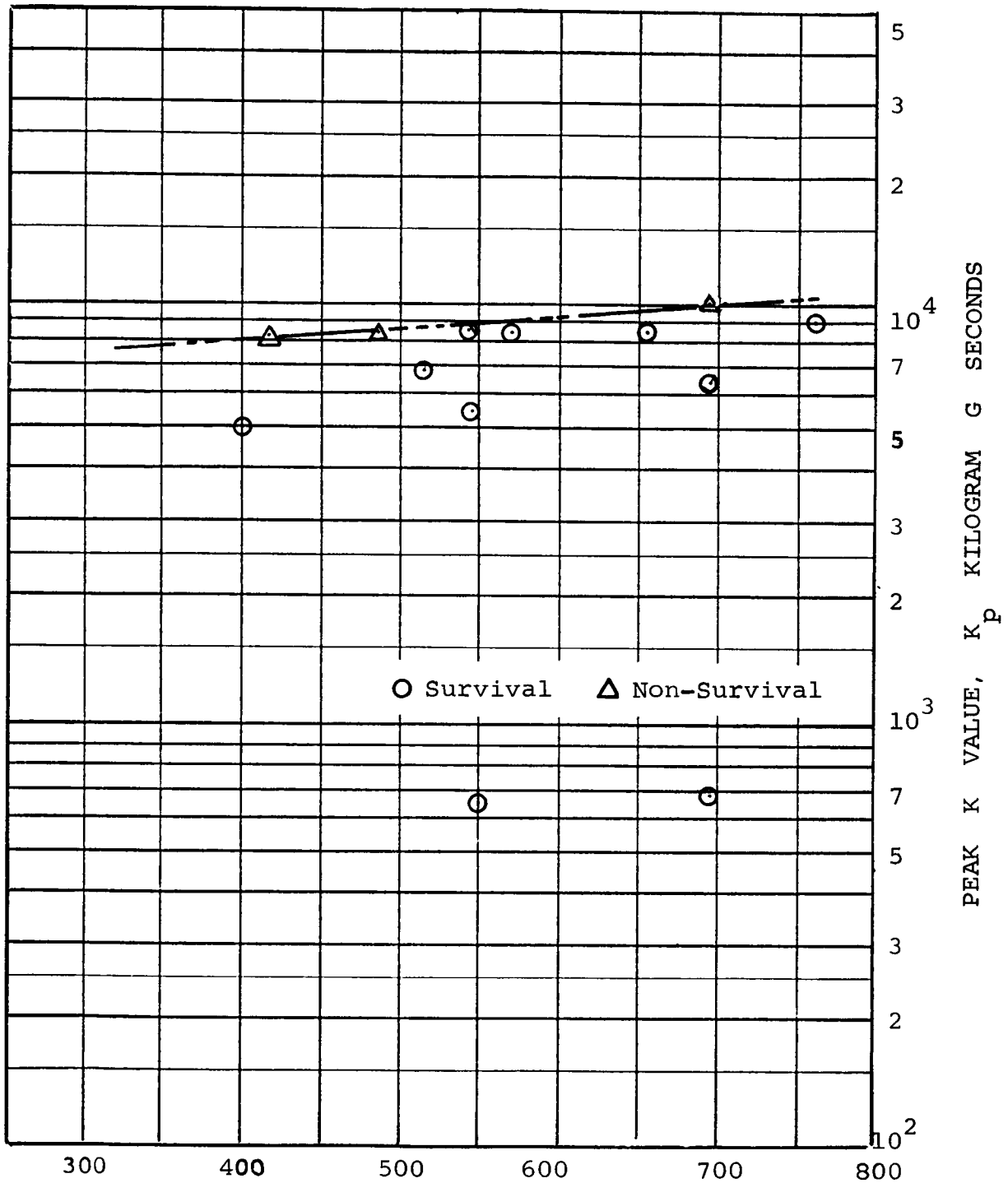

FIGURE 25. PEAK K VALUE VERSUS ANIMAL WEIGHT
50 G CASE

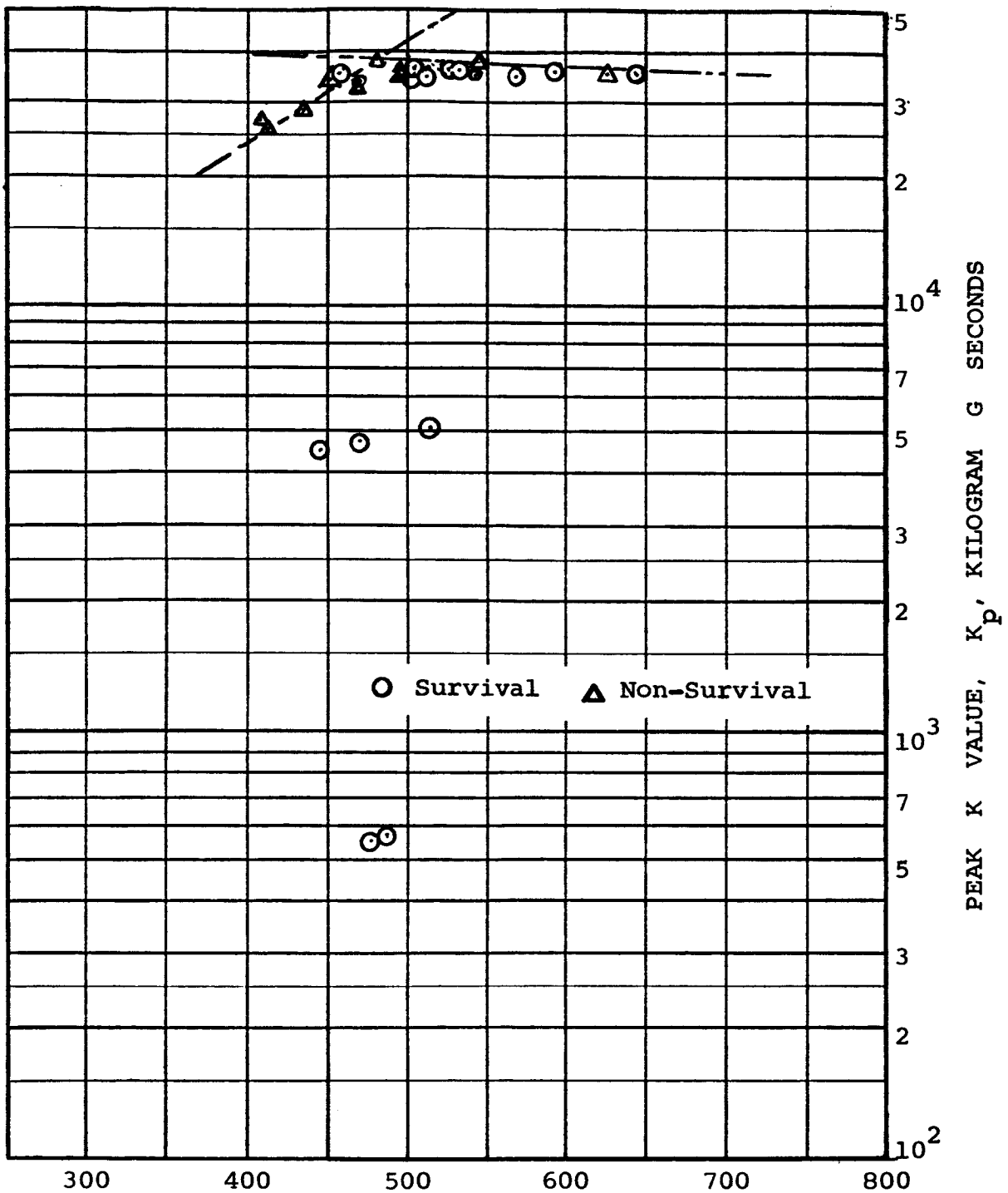

FIGURE 26. PEAK K VALUE VERSUS ANIMAL WEIGHT
400 G CASE

the two sets of curves, Δ_{rt} versus weight, and K_p versus weight, for each G stress level became laboratory working tools throughout the course of the remaining experiments. Δ_{rt} curves are shown in Figures 148 through 163 in Appendix F.

Direct solution for D_r.- With a selection of K_p rather than Δ_{rt}, D_r could be directly determined from Equation 6. Then from Equation 8 the value of Δ_{rt} could readily be calculated.

Results.- Graphic representations of experimental results are shown in Figures 164 through 172 in Appendix F, with K_p plotted against animal weight for each G mode and level.

Three Dimensional Plot

Study of the curves of Δ_{rt} versus weight and of K_p versus weight resulted in an attempt to express the relationship of K_p versus W_o versus G_o in a three dimensional plot. The relationships of the three parameters may be such that sets of points can be computerized to determine values of constants modifying W_o and G_o such that a surface curve of K versus W_o and G_o can be plotted.

K_p curve.- The inference drawn from the sets of K_p versus weight curves is that a three-dimensional model can be constructed with K_p rising vertically, W_o increasing to the right and G_o (as planes) increasing in the +Z direction. This model essentially creates a three-dimensional curved surface defining a relationship of K_p to weight and G_o.

The investigator has the freedom to manipulate the values of K_p, weight, and G_o such that a point lying at the surface described by the relationship of the three parameters can be obtained for any chosen weight or G_o. If (K_1, K_2, K_3, ...K_p) are assumed to represent increasing values of K, a series of surfaces can be described -- layered each above the other -- in such a fashion that the area between any two surfaces described by:

$\left(K_1, W_o, G_o\right)$ and $\left(K_2, W_o, G_o\right)$

can be regarded as a zone of equal lethal dosage or $(LD_{1,2})$.

Hopefully, the initial relationship of the surface described by K_1, W_o, and G_o is such that if an animal is exposed to a point beneath this surface, its survivability can be guaranteed as if a protective umbrella were spread over certain ranges of these three variables. As K increases, the space between two adjacent surfaces may represent advancing ranges of lethal dosage expressed as a percentage of Lethal Dosage until 100% fatalities are experienced. The three-dimensional model is illustrated below as Figure 27.

Maximum value of K.- Experimentally it was noted that no animal was able to survive if its value for K was above 3.65. Therefore maximum value for K (K_{max}) was empirically chosen as equal to 4.0×10^4. This observation altered the initial constraints on K. These constraints are expressed below.

$$LD_0 = K < 10^4 \quad (11)$$

$$LD_{50} = 10^4 < K < 4.0 \times 10^4 \quad (12)$$

$$LD_{100} = K > 4.0 \times 10^4 \quad (13)$$

Note: No attempt has been made to present a rigorous mathematical proof of the hypothesis outlined in this section. Rather, it is an attempt to quantify an empirical observation which was supported by over 200 experimental results. Laboratory use of this working tool has been gratifying, resulting in 14 specific cases where, within the biological variability, the actual outcome was predicted as marginal prior to the event.

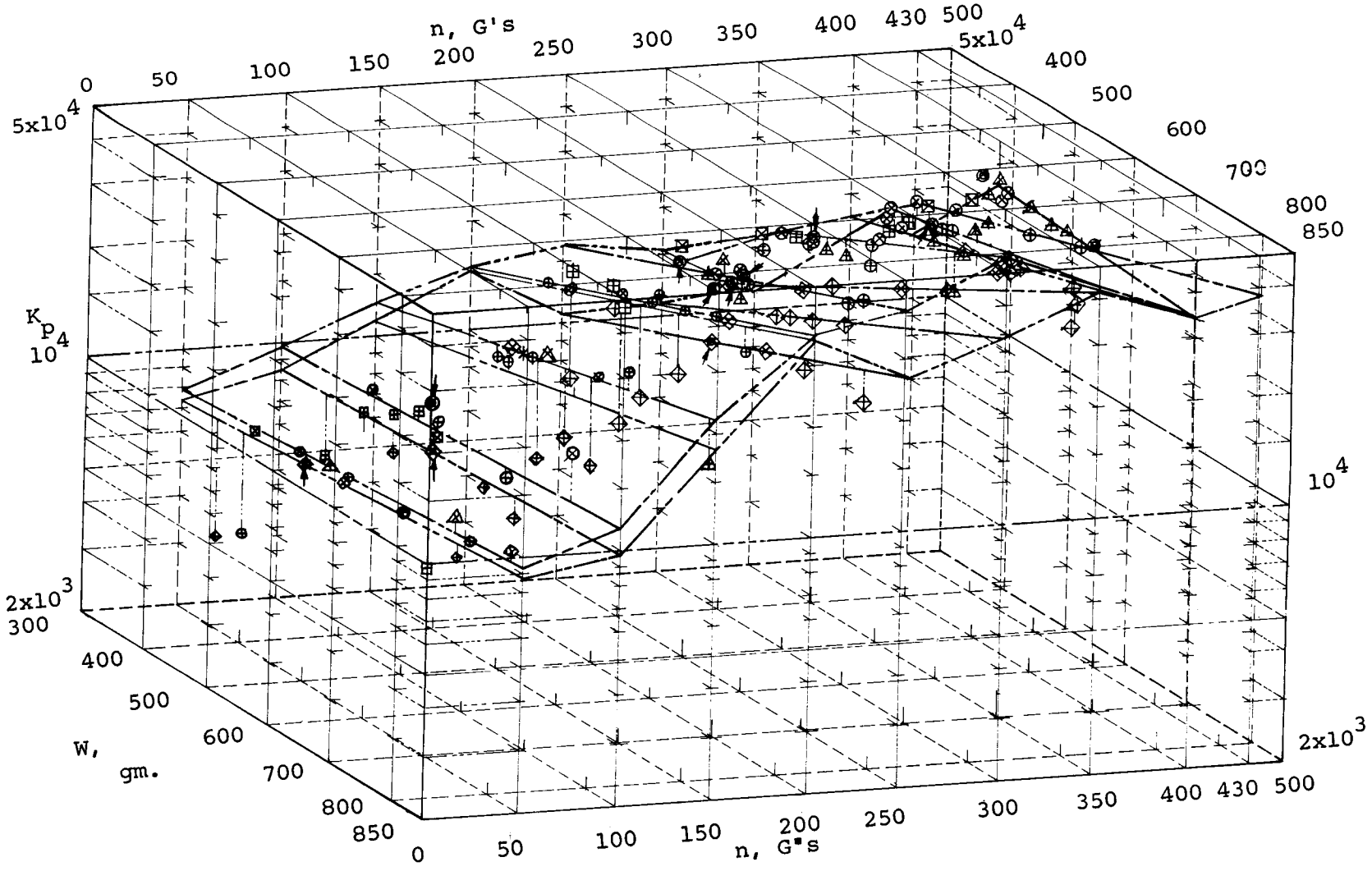

FIGURE NO. 27 THREE DIMENSIONAL MODEL, K_p vs G vs W

A Computerized Program for the Evaluation of the Surface Defined by K, Weight, and G

Computer programs are available which fit polynomials by a least squares fit to triplets of experimental points. The greater the number of sets of points of K, weight, and G that are obtainable, the more accurately will the surface be described. But of course the greater the number of sets of points, the greater will be the number of equations and unknowns that would have to be solved.

For a computerized calculation for the construction of an approximate curve to fit the experimental data the following considerations apply:

Definition: $K = WGT = f(W, G)$ (14)

where the letter G is substituted for n in Equation 3 for purposes of the derivation set forth below:

Attempting to approximate $f(W, G)$ by a polynomial in W and G of order N, thus:

$$f(W, G) = a_{oo} + a_{10}G + a_{11}W + a_{20}G^2 + a_{21}WG + a_{22}W^2$$

$$+ a_{30}G^3 + a_{31}WG^2 + a_{32}W^2G + a_{33}W^3 + \ldots a_{n0}G^n + a_{n1}WG^{n-1}$$

$$+ a_{n2}W^2G^{n-2} + \ldots \left[a_{ni}W^iG^{n-i} \right] + \ldots a_{nn}W^n + \ldots a_{NN}W^N \quad (15)$$

or $$f(W,G) = \sum_{n=o}^{N} \sum_{i=o}^{n} a_{ni}W^iG^{n-1} \quad (16)$$

Now given m experimental points, [that is, m sets of points (K, W, G)], the maximum number of determinable 2_{ni} would be m. Thus, the series must terminate at $n = N$ in such a manner that the total number of terms is less than or equal to n.

For each value of n there are $(n + 1)$ values of i, since:

$$o \leqq i \leqq n \quad \text{and} \quad i \quad \text{is an integer}$$

Thus:

$$\sum_{n=o}^{N} (N = 1) \leqq m \tag{17}$$

$$= \sum_{n-1}^{N+1} n = \frac{(N+1)\ (N+2)}{2} \tag{18}$$

Thus:

$$(N+1)\ (N+2) \leqq 2m \tag{19}$$

Thus examining:

$$(\alpha + 1)\quad (\alpha + 2) = 2m \tag{20}$$

where α is a number, not necessarily an integer, such that $\alpha \leqq N$

$$\alpha^2 + 3\alpha - (2m-2) = 0 \tag{21}$$

$$\alpha = -3 \frac{\pm \sqrt{9 + 4\ (2m-2)}}{2} \tag{22}$$

Ignoring the negative solution, since it is of no interest, and allowing N_{max} to be the largest integer less than or equal to α, then N_{max} is the largest integer satisfying condition 19. This is the largest N which could be used in Equation 16 to solve for all of the a_{ni}. If a lesser value for N were to be selected, it would produce a less accurate result, since only

$\frac{(N+1)\ (N+2)}{2}$ experimental points can be applied.

Given:

$m = 210$ triplets of points (number of animals in this study)

Then:

$\alpha = 17$ constants, thus $N_{max} = 17$, from Equations 19 and 20

If $N_{max} = 17$, then a set of 171 equations with 171 unknowns are involved. This would require the services of a computer. Alternatively, if only 80 points were to be considered (i.e., $m = 80$), then

$$\alpha = -3 \frac{\pm\sqrt{9 + 4\ (158)}}{2} = 11$$

Thus:

$$N_{max} = 11$$

and $$\frac{(11 + 1)\ (11 + 2)}{2} = 6 \times 13 = 78$$

Thus this involves a set of 78 equations with 78 unknowns. Similarly, an even rougher approximation of the final equation would result with the use of a lesser number of sets of points. The experimental sets of points obtained from 14 "cliff-hanger" animals are suggested as those that should be used in the initial computer solution for a first approximation of the surface described by the dynamic parameters. These triplets of points are included in Table VIII below; points 1 through 8 and point 14 are for the $+G_x$ surface while points 9 through 13 are for the $-G_x$ surface.

The approximate shape of the curve described by these points is shown by the three dimensional view of K versus G_o versus W_o (Figure 27).

TABLE VIII

TRIPLETS OF POINTS, CLIFFHANGER ANIMALS					
POINT	ANIMAL		STRESS		
	No.	W(gms)	nG	K_p ($\times 10^{-4}$)	G Mode
1	145	441.7	250	2.3	$+G_x$
2	146	545.5	250	2.7	$+G_x$
3	147	495.3	300	3.0	$+G_x$
4	153	455.0	400	3.7	$+G_x$
5	164	531.0	350	3.2	$+G_x$
6	173	500.0	250	2.3	$+G_x$
7	174	546.5	100	1.2	$+G_x$
8	183	529.5	250	2.6	$+G_x$
9	190	495.5	50	0.71	$-G_x$
10	194	548.6	100	0.9	$-G_x$
11	203	494.7	250	1.6	$-G_x$
12	228	505.2	400	2.4	$-G_x$
13	235	482.5	400	2.2	$-G_x$
14	237	533.4	430	2.5	$+G_x$

A brief examination of the experimental data resulted in the comparison shown in Figure 28. As the stress level at 95% survivors increases, the time decreases, but it is apparent that the slopes of the curves change radically at three points. These breaks in the curves roughly correspond to the changes seen in the gross pathology and histopathology reported earlier. It is thought likely that the time factor assumes increasing importance as the stress level increases, perhaps as some power function of time. If the maximum allowable G stress is expressed as:

$$n_{allowable} = c\, t^{x}$$

Then possibly the data might be fit so that

$$x = K_1 \quad \text{for Phase 1 failure}$$
$$= K_2 \quad \text{for Phase 2 failure}$$

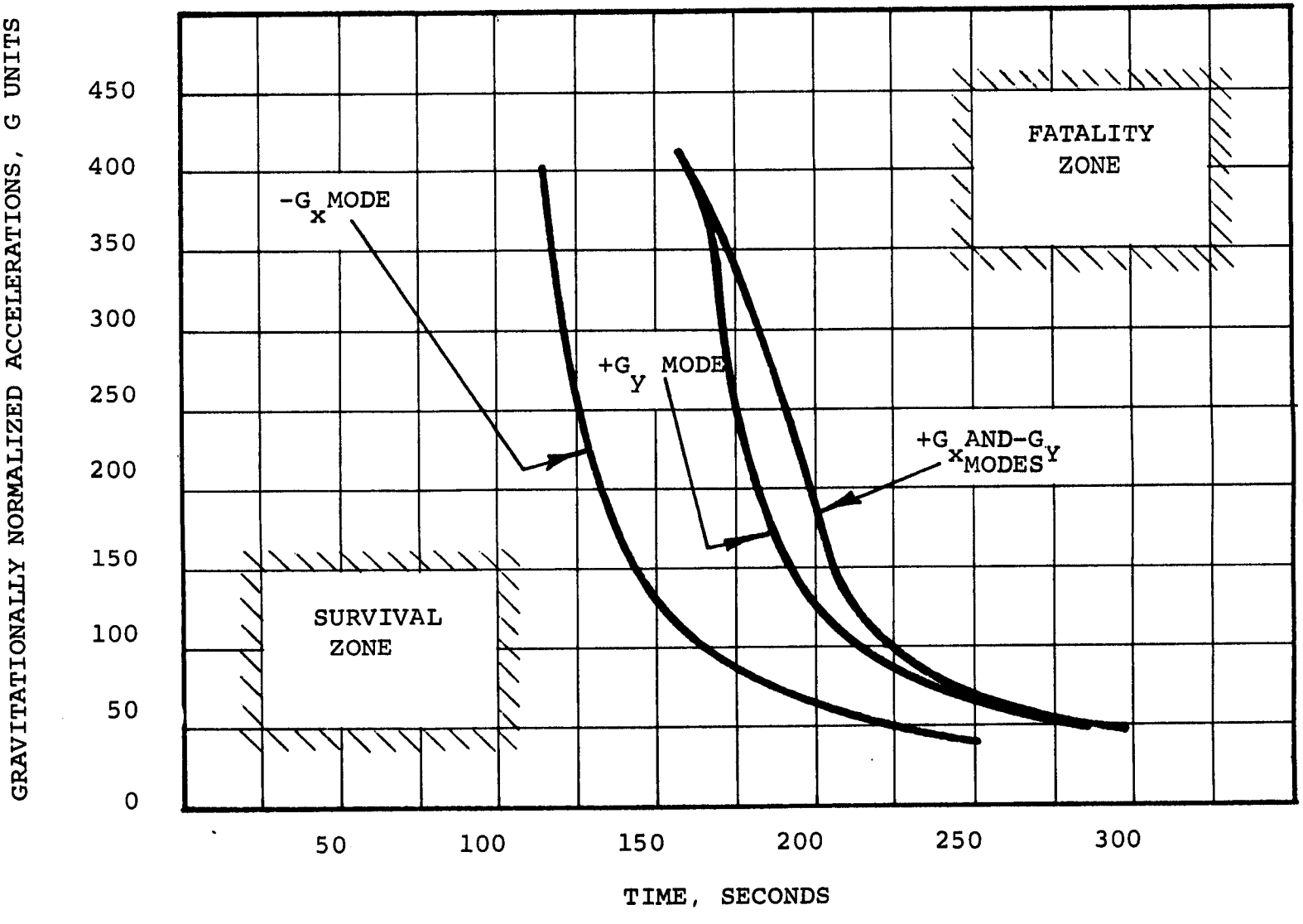

FIGURE 28 GROSS SURVIVABILITY STATISTICAL CURVES
(95 PERCENT SURVIVORS)

$= K_3$ for Phase 3 failure, etc.

Preliminary checks suggest that $K_3 \approx -4$, for the upper portion of the $+G_x$ curve. Further study of this effect of time is needed.

Non-rigorous Application of "K" Principle to Other Mammalia and to Man

It is well known that different species have differing propensities to absorb stress. However, it should be possible to expect that as each species differs slightly from another, so should their various natural responses to differing environmental situations. These differences can be expected to vary in a graded manner, such that they should lend themselves to a mathematical expression, if the parameters can be quantified.

Once this is achieved, a scaling factor should become evident that will permit the cross correlation of the response of one species to that of another. The immense importance of this concept has been well appreciated and forms the basis of research efforts aimed at the extrapolation of animal data to man.

Recently a parallel study of guinea pigs was conducted under an Air Force research program (Contract 33(615)1893) while the FEAT program was in progress, but not as a part of FEAT. We took the liberty of assuming that guinea pigs also had a "K" factor of their own, possibly much higher in value than that for a primate such as S. sciureus. A "non-rigorous" application of the value of K for primates to the guinea pig was utilized. This was done merely to try for a pre-run prediction of the survival of guinea pigs under G stress.

Consequently, as was done for the primate, the dynamic factors chosen by the Air Force Project manager were examined, assuming that $K=10^4$ for a first approximation fit. A pre-run prediction of survival was made for each animal. Six animals were tested, and they were exposed to G stress for approximately one second. The results are reflected in the following table. As can be seen from Table IX, the highest K_p achieved by any animal was 1.18×10^2, a value well below the 10^4 region.

TABLE IX

GUINEA PIG PREDICTION

RUN No.	G.P. No.	WT. Kg.	nG	TIME	K_p	P.O.	A.O.
1	7	.315	100	1 sec.	3.15×10^1	+	+
2	10	.315	100	1 sec.	3.15×10^1	+	+
3	8	.295	200	1 sec.	5.90×10^1	+	+
4	2	.265	200	1 sec.	5.30×10^1	+	+
5	9	.295	400	1 sec.	1.18×10^2	+	+
6	12	.295	400	1 sec.	1.18×10^2	+	+

% Correct = 100%

P.O. = Predicted Outcome
A.O. = Actual Outcome

Application to man.- A non-rigorous application of the principle to man was attempted. The recent experience of Stapp (5) to $-44\ G_x$ was selected as an example. Stapp's data, of course, is from the Holloman sled run and falls in the realm of impact stress. Consequently, the onset and offset rates experienced by this researcher differed greatly from those utilized in the experiments with Saimiri sciureus. For that reason the cross-correlation of these data at this time is tenuous. However, it can be surmised that the high rate of onset/offset of stress seen by Stapp most probably was more injurious than the onset/offset rates utilized in the FEAT experiments. In the future it may become possible to cross-correlate the effect of differing onset/offset rates.

Strictly for purposes of illustration, the assumption was made that the K factor of Saimiri sciureus might apply to other primates as well. If it is valid for man, Stapp's data can be reconstructed as follows:

Assuming:

1. Onset/offset rates are the same as those of FEAT;

2. Weight of subject, 70 kg;

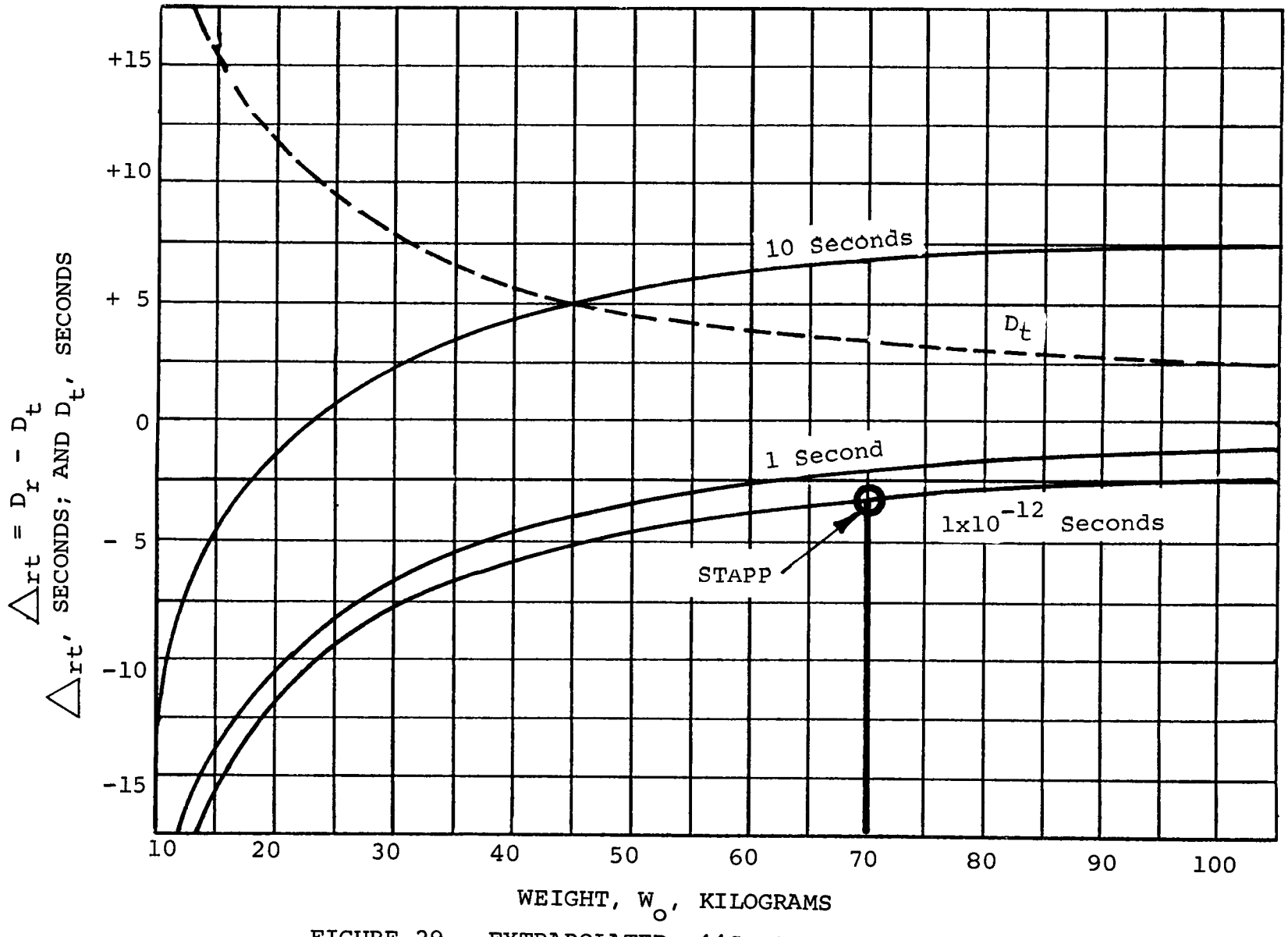

FIGURE 29. EXTRAPOLATED $44G_x$ CURVE FOR MAN

3. $nG = -44\ G_x$;

4. Time of application of load: less than one second.

Applying these data to Equation 3,

$$K_p = W_o\ G_o\ t \tag{3}$$

$$K_p = 70 \times 44 \times 1 = 3.1 \times 10^3 \text{ max.}$$

The graph drawn as Figure 29 would apply. Reference Δ_{rt} curves are drawn for time of dwell at nG for 10 seconds, one second, and a very small fraction of one second.

According to the hypothesis set forth above, this subject could have withstood at least an additional two seconds of stress application without sustaining irreparable systemic damage.

Resemblance of pathology; animal to man.- The resemblance of the damage sustained by the above subject on the sled run and the damage sustained by test subjects in the FEAT experiments are notably of the same type and approximate severity.

CONCLUSIONS

As a result of these experiments we arrived at a number of conclusions. Some of them were noted as a direct result of the experimental data. Still others were inferred by insight gained from a comparison of specific experimental results against previous knowledge in the field of accelerative stress.

The conclusions can be divided into several broad categories as follows:

1. The Test Animal
2. Pathologic Changes
3. Protection Against Damage
4. Supportive Care
5. Extension of Tolerance

6. Technical Procedures

7. Extrapolation of Data

   a. To Other Test Subjects

   b. To Other Fields of Study

In the sections that follow these conclusions will be examined briefly. Detailed discussion of each of the main points is contained within individual sections of the main body of this report.

THE TEST ANIMAL

Certain conclusions were reached which applied to the test subject itself. These were:

1. That the selected test animal, properly handled, is an acceptable test specimen in this type of an experiment. Handling procedures designed in support of the experimental tasks proved to be adequate and, in most cases, succeeded in delivering a viable healthy specimen to the test laboratory.

2. That the test animal, as hypothesized, can absorb high accelerative loads in the range of 50 G to 430 G. The amazing capacity of the squirrel monkey to absorb high levels of G force was repeatedly demonstrated under the constraints of constant onset/offset rates. It proved in these experiments that it could easily withstand most of tests designed into the experimental matrix and indicated that even higher levels of stress could probably be successfully absorbed.

3. That the test animal, as hypothesized, can accept these high accelerative loads of 50 to 430 G for extended periods of time -- as much as 200 seconds and more. In every mode but one, two-thirds of the dwell times designed into the experimental matrix were exceeded. It was not until the higher levels of G stress were examined that the animals experienced difficulty in attaining dwells of 200 seconds. They were in every case routinely capable of exceeding 100 seconds dwell. Inferred is that the animal can absorb even higher stresses than those seen in this experimental matrix over shorter time bases.

4. That a fine calibration of the test animal's response to high accelerative loads is possible, based on the mathematical hypothesis outlined in this report. Experimental data showed that the application of the mathematical hypothesis to new untried levels of G stress yielded a gratifying number of runs near the animal's tolerance level to accept such stress.

5. That there appears to be a subtle difference in the ability of the test subject to absorb stresses of this kind in the different axes (by standard AGARD terminology). In particular the test subject appears to be most susceptible to stresses applied in the $-G_x$ axis. In these particular series of experiments, the animal repeatedly succumbed to G stresses which were easily handled in the remaining three modes studied. Differences in these latter modes were more subtle.

PATHOLOGIC CHANGES

Certain conclusions were reached regarding the five categories of pathologic changes listed below:

1. The major types of response to damaging stress encountered may be placed into three ranges: Low G range, to 150 G. Congestion and pooling of blood with anoxemic changes secondary to lack of blood flow. 2 - Mid-G range, 200 - 250 G. Relative resistance to damage up to a maximal point near 200 seconds dwell. Beyond that dwell point there occurred overwhelming congestive and anoxic changes. 3 - High G range, 300 - 430 G. Traumatic lacerations of tissues, separation of bony sutures of the calvarium and fatal plastic deformation of vital structures such as the brain and brain stem. These ranges correspond to those shown in Figure 29.

2. The heart is apparently capable of surviving very high stresses of this type, but not without residual damage to the myocardium. The oxidative enzyme study demonstrated acute ischemic changes. These tended tc support the finding of myocardial necrosis by routine stain.

3. As far as the myocardium is concerned, exposure to this type of stress may be a "one shot" proposition. Otherwise, a second exposure may precipitate profound cardiovascular changes. The notable differences observed in the ECG traces of animal

No. 241-242 taken at comparable times of exposure to similar stress revealed the early development of arrhythmias and myocardial infarction during the animal's second run. If man were to be exposed to this type of stress, it may be that he will be able, in terms of "myocardial safety", to absorb such stress one time and one time only.

4. It may be possible electrocardiographically to predict the point at which a test subject should be exposed to no further stress. This electrocardiographic alteration is characterized by the accentuation of the T wave and its progression toward and its convergence with the ST - QRS complex and occurs after the advent of QRS configuration changes indicative of myocardial ischemia.

5. If the animal is stressed to near tolerance levels, but not beyond tolerance, then the major portion of the damages sustained are apparently reversible and compatible with clinically normal behavior post-run. Animals which survived the run behaved normally, as described in the Test Animal section. Gross and histopathologic findings tended to support this view.

PROTECTION AGAINST DAMAGE

Certain conclusions were reached with respect to measures which might protect the test subject. Among these were:

1. That on the basis of a few changes in the constraints of the animals during the experiment, it was possible to extend their tolerance to stress in the $-G_x$ mode by taking advantage of certain natural supportive structures for labile organs, such as the eyes and tongue.

2. That pharmacologic, mechanical, or other methods may prove useful in protecting the animal against damage to the vital organs or to vital centers. The provision of continuous flow oxygen under positive pressure during stress exposure may sustain the minimal air exchange necessary for survival and may help prevent plastic deformation of the thoracic cavity. These measures and others are worthy of experimental trial.

SUPPORTIVE CARE

Clinical observations supported the following conclusions:

1. That it appears to be possible to clinically support the test subject after he has sustained systemic damage secondary to the application of high accelerative loads. Such immediate post-run clinical support appears to be of the following types: support of the respiration of the animal, correction of acidosis, support of the dehydrative states which ensue when the animal has sustained tolerance levels of stress and is so incapacitated that it cannot take in an adequate amount of fluids to support its metabolic needs, treatment of myocardial infarction, steroid hormone support of the animal secondary to possible adrenocortical insufficiency. The dangers and complications of hormone therapy are, of course, to be borne in mind. Possible use of biologicals in the post-run period to reduce the advent of secondary saprophytic invaders of the respiratory system, and finally close attention to the temperature and humidity requirements of the test subject and other adequate measures necessary to support the animal.

2. That the determination of the precise response of the animal to G stress of this type by standard bioassay techniques is of fundamental importance to any attempt to extend the test subject's tolerance to stress.

EXTENSION OF TOLERANCE

Certain conclusions were drawn with respect to tolerance extension. Among these were:

1. That the test results achieved by the utilization of a conscious animal throughout the series of experiments has provided a firm base against which measurement is possible of the effectiveness of such devices as pharmacologic, mechanical and other agents in the attempt to extend knowledge of the test subjects' ability to sustain and survive even higher accelerative stresses than those employed in this experimental series.

2. That a greater number of animals from a known, controlled colony should be exposed to these high G environments such that

statistical validity of the animal's response to these stresses may be strengthened.

3. That a certain number of animals should be exposed in the $\pm G_z$ AGARD convention for the proper elucidation of animal response in this mode.

TECHNICAL PROCEDURES

The several technical procedures involved in this experiment produced a number of conclusions. Among these were:

1. That a safe anesthetic procedure for use in the preparation of this test subject for experimental purposes has been developed. Diethyl ether proved to be a safe, effective predictable anesthetic. The special enzyme study supported the impression that the anesthetic agent itself caused no residual animal tissue damage.

2. That special oxidative enzyme-staining techniques are necessary to positively identify anoxemic changes secondary to stress. Correct interpretation of the temporal response of tissues correlated to the stress event itself is of utmost importance in the assessment of damage sustained by the animal.

3. That a high speed magnetic tape readout would be a valuable adjunct in the analysis and interpretation of ECG results.

4. That more elaborate methods are necessary to identify such items as blood flow, air exchange, P wave identification (by esophageal electrode) and other physiologic parameters which would pinpoint the animal's dynamic physiologic response.

EXTRAPOLATION OF DATA

The experimental data was examined critically from the standpoint of extrapolation to man, for in the final analysis this is the goal toward which experiments such as this one are aimed. Among these conclusions were:

1. That if the hypothesis of the inter-reaction of the dynamic parameters as outlined herein is valid, then the experimenter has the freedom to manipulate the parameters in such a way that the level of stress and the time base over which the subject is exposed to this stress are such that the test subject remains within his tolerable limits to accept such stress.

2. That in the application of the K principle to a group of six guinea pigs exposed to varying ranges of G, the predicted experimental results by application of the formula were substantiated by the actual experimental results. It is concluded that the application of the K principle to other species of Mammalia as well as primates, is worthy of trial for the substantiation of the validity of the principle to the Mammalia as a group.

3. That it is possible to extend this hypothesis of increased capability of stress tolerance to higher forms of primates and eventually to man.

4. It is further concluded that should the mathematical hypothesis advanced in this paper be proved valid that the problems of extrapolation of experimental data from lower forms of primates to man may become less of a problem than heretofore realized.

5. That a "preliminary application" of the K principle to a human test subject, having sustained $-44\ G_x$ over a short time base on the order of less than one second indicates that were this test subject exposed to SFAPS onset/offset rates, it could have absorbed the continued application of this G load over a time base of at least two additional seconds. The validity of such a hypothesis, of course, is dependent on further examination of the response of individuals to stresses of the type sustained by the human test subject above, and to the standardization of experimental parameters from impact studies to conventional methods of centrifugation.

6. That three phases of failure are apparent, but that five phases are suspected. See Figure 29. For a given onset/offset rate it is suspected that the time factor assumes increasing importance as the G level increases.

Examining the test data of all of the runs the correlation between subject weight and time exposure at a specific G level is not sufficiently clear at present for the promulgation of a

rigorous mathematical law. However, extended research in the attempt to unify impact studies and conventional centrifugation with our regime of study may clarify the inter-reaction of these dynamic parameters, as well as others across the entire field of impulse studies and may eventually result in a mathematical model for man.

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APPENDIX A
BASIC SUMMARY TABLES

This Appendix contains the two basic summaries of the experimental results, Tables X and XI.

TABLE X, SUMMARY OF TEST RESULTS BY ANIMAL NUMBER

This table reflects the sequential accumulation of data, since animal numbers were assigned in daily sequence. Points in the course of the study when different tacks were taken in the approach to experimental problems are apparent. Reference to this table will agument the information contained in the body of the report, and presents a consise summation of the results for any individual animal. The entries and column heading of the table use the same notation as used throughout the report.

TABLE XI, SUMMARY OF TEST CELLS BY MODE AND G LEVEL

This table summarizes each test cell by the direction of force application and G Level. The information presented is a duplication of that given in Table X, but is arranged for entry by exposure levels rather than by individual tests. A comparison of the results obtained within each test cell is thus made easier. The dwell times are arranged in order of magnitude, within each cell.

This table is arranged to permit correlation with Table I, which shows the original test plan matrix and with Table XIX, Gross Pathology Results, in Appendix D. It can also be used readily in the interpretation of Figures contained in Appendix E. The entries are self-explanatory.

TABLE X. SUMMARY OF TEST RESULTS BY ANIMAL NUMBER

ANIMAL					STRESS							ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time hr.	No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time hr.
23	-	-	-	-	-	-	-	-	-	A	0	53	M	437	-	-	$-G_x$	50	20	458	4.4×10^2	+	48
24	M	692	-	-	-	-	-	-	-	A	7 days	54	M	380	-	-	$-G_x$	50	2	527	3.8×10^1	+	48
25	M	900	-	-	-	-	-	-	-	A	7 days	55	M	492	-	-	$-G_x$	100	2	208	9.8×10^1	+	48
26	M	704	-	-	$+G_x$	250	2	-	3.6×10^2	+	72	56	M	429	-	-	$-G_x$	100	20	233	8.6×10^2	+	48
27	M	642	-	-	$+G_x$	250	2	62.4	3.2×10^2	+	72	57	M	355	-	-	$-G_x$	100	200	282	7.0×10^3	-	Zero
28	-	600+	-	-	-	-	-	-	-	A	None	58	F	500	-	-	$-G_x$	150	2	133	1.5×10^2	+	24
29	M	713	-	-	$+G_x$	250	20	56.1	3.5×10^3	+	72	59	F	568.5	-	-	$-G_x$	150	20	117.5	1.7×10^3	+	24
30	M	734	-	-	$+G_x$	250	200	54.6	3.7×10^4	+	72	60	F	537	-	-	$-G_x$	200	2	93	2.1×10^2	+	24
31	M	608	-	-	$+G_x$	300	2	55	3.6×10^2	+	72	61	M	802	-	-	-	-	-	-	-	A	Zero
32	M	587	-	-	$+G_x$	300	20	56	3.5×10^3	+	72	62	F	538	-	-	$-G_x$	200	20	93	2.2×10^3	+	24
33	M	598	-	-	$+G_x$	300	200	55	3.7×10^4	-	Zero	63	F	518	-	-	$-G_x$	150	200	128.5	1.6×10^4	-	Zero
34	F	508	-	-	$+G_x$	150	2	131.5	1.5×10^2	+	48	64	M	600	-	-	$-G_x$	250	2	66.6	3.0×10^2	+	48
35	M	836	-	-	$+G_x$	150	20	79.6	2.5×10^3	+	48	65	M	507	-	-	$-G_x$	250	20	78.8	2.5×10^3	+	48
36	M	704	-	-	$+G_x$	150	200	94.8	2.1×10^4	+	48	66	M	638	-	-	$-G_x$	250	200	62.8	3.2×10^4	-	Zero
37	F	433	-	-	$+G_x$	100	2	235	8.7×10^1	+	72	67	M	711	-	-	$-G_x$	200	200	70.5	2.8×10^4	-	Zero
38	M	653	-	-	$+G_x$	100	20	153.5	1.3×10^3	+	72	68	M	566	-	-	$-G_x$	300	20	58	1.7×10^3	+	48
39	M	764	-	-	$+G_x$	100	200	130	1.5×10^4	-	Zero	69	M	568	-	-	$-G_x$	350	20	50.4	4.0×10^3	-	Zero
40	F	623	-	-	$+G_x$	50	2	315	6.2×10^1	+	48	70	F	656	-	-	$+G_y$	50	2	305	6.5×10^1	+	24
41	M	549	-	-	$+G_x$	50	20	350	5.5×10^2	+	48	71	M	698	-	-	$+G_y$	50	20	287	7.0×10^2	+	24
42	F	400	-	-	$+G_x$	50	200	500	4.0×10^3	+	48	72	F	549	-	-	$+G_y$	50	200	365	5.4×10^3	+	24
43	F	469	-	-	$+G_x$	200	2	109	1.9×10^2	+	48	73	F	546	-	-	$+G_y$	150	2	122	1.6×10^2	+	24
44	F	429	-	-	$+G_x$	200	20	117	1.7×10^3	+	48	74	F	560	-	-	$+G_y$	150	20	119	1.7×10^3	+	24
45	M	353	-	-	-	-	-	-	-	A	Zero	75	M	532	-	-	$+G_y$	150	200	125	1.6×10^4	-	Zero
46	M	423	-	-	$+G_x$	200	200	119	1.7×10^4	+	48	76	M	564	-	-	$+G_y$	250	2	72.4	2.8×10^2	+	48
47	F	517	-	-	$+G_x$	350	2	55	3.6×10^2	+	48	77	F	613	-	-	$+G_y$	250	20	65.4	3.1×10^3	+	48
48	F	482	-	-	$+G_x$	350	20	60.5	3.4×10^3	+	48	78	F	560	-	-	$+G_y$	250	200	71.4	2.8×10^4	-	Zero
49	M	504	-	-	$+G_x$	350	200	56.7	3.5×10^4	-	Zero	79	F	723	-	-	$+G_y$	300	2	46.2	4.3×10^2	+	48
50	F	479	-	-	$+G_x$	400	2	52.3	3.8×10^2	+	72	80	F	596	-	-	$+G_y$	300	20	55.8	3.5×10^3	+	48
51	M	469	-	-	$+G_x$	400	20	53.3	3.8×10^3	+	48	81	F	617	-	-	$+G_y$	350	2	46.1	3.4×10^2	+	48
52	M	353	-	-	$-G_x$	50	200	566	3.5×10^3	+	48	82	YM	313	-	-	$+G_y$	350	20	91.2	2.2×10^3	+	72

TABLE X (CONTINUED)
SUMMARY OF TEST RESULTS BY ANIMAL NUMBER

ANIMAL					STRESS							ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.	No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.
83	F	445	-	-	$+G_y$	400	20	56.2	3.6×10^3	+	72	113	M	502	29.5	5.88	$-G_y$	400	169.8	49.8	3.4×10^4	+	48
84	F	583	-	-	$-G_y$	100	2	171.5	1.2×10^2	+	24	114	F	497.2	27.8	5.59	$-G_y$	350	177.5	57.5	3.1×10^4	+	48
85	F	547	-	-	$-G_y$	100	20	183	1.1×10^3	+	24	115	F	519	30	5.78	$-G_y$	50	266	386	6.9×10^3	+	48
86	F	438	-	-	$-G_y$	100	200	227	8.8×10^3	+	24	116	M	692.9	34.7	5.01	$-G_y$	50	189	289	6.4×10^3	+	48
87	F	514	-	-	$-G_y$	200	2	97.3	2.1×10^2	+	24	117	F	486.6	24.2	4.97	$+G_y$	250	202.6	82.6	2.5×10^4	+	24
88	F	542.8	-	-	$-G_y$	200	20	92.5	2.2×10^3	+	24	118	M	570.5	40.6	7.11	$+G_y$	150	200	117.1	1.7×10^4	-	Zero
89	F	465	-	-	-	-	-	-	-	S	Zero	119	M	693	35.8	5.17	$+G_y$	50	289	289	1.0×10^4	-	Zero
90	F	567	-	-	$-G_y$	300	2	58.9	3.4×10^2	+	24	120	F	511	25.5	4.99	$+G_y$	300	185.2	65.2	2.9×10^4	+	24
91	F	579	-	-	$-G_y$	300	20	57.7	3.5×10^3	+	24	121	M	541	32.7	6.09	$+G_y$	50	320	370	8.6×10^3	+	48
92	F	464	-	-	-	-	-	-	-	A	Zero	122	M	680	38	5.86	$+G_y$	400	138.7	38.7	3.6×10^4	+	24
93	F	540	-	-	$-G_y$	200	200	92.8	2.2×10^4	+	24	123	F	598.9	34	5.68	$+G_y$	350	177.8	47.8	3.7×10^4	-	Zero
94	F	500	-	-	$-G_y$	350	2	57.1	3.5×10^2	+	24	124	F	477.5	26.2	5.49	$+G_y$	400	200	52.4	3.8×10^4	-	24
95	F	591	-	-	$-G_y$	350	20	48.4	4.2×10^3	+	24	125	F	466.3	26	5.58	$+G_y$	400	178.7	53.7	3.3×10^4	-	Zero
96	F	465	-	-	$-G_y$	300	200	71.9	2.8×10^4	+	24	126	M	506.5	30.9	6.10	$+G_y$	400	174.4	49.4	3.5×10^4	+	24
97	F	529	-	-	$-G_y$	350	200	54.2	3.7×10^4	-	Zero	127	F	542.0	23.9	4.41	$+G_y$	400	171.1	46.1	3.7×10^4	-	Zero
98	F	483	-	-	$-G_y$	400	2	51.8	3.9×10^2	+	24	128	YF	519.1	28.4	5.47	$+G_y$	400	164	48.2	3.4×10^4	+	72
99	F	516	-	-	$-G_y$	400	20	48.5	4.1×10^3	+	24	129	F	537.7	27.2	5.06	$+G_y$	350	163.2	53.2	3.1×10^4	+	72
100	F	456	-	-	$-G_y$	400	200	55.0	3.7×10^4	+	8 mo.	130	F	539.5	26.9	4.99	$+G_y$	250	174.2	74.2	2.4×10^4	+	72
101	F	525.5	28	5.33	$-G_y$	100	210	191	1.2×10^4	+	48	131	F	543.4	26.0	4.78	$+G_y$	300	184.5	61.6	3.0×10^4	+	24
102	F	462	-	-	$-G_y$	200	228	108	2.1×10^4	-	Zero	132	M	712.5	35.5	4.98	$+G_y$	300	140	46.8	3.0×10^4	+	24
103	YF	284.7	-	-	$-G_y$	300	200	116.5	1.7×10^4	-	Zero	133	F	570.3	28.0	4.91	$+G_y$	400	153.8	43.8	3.5×10^4	+	24
104	F	409	25.9	6.33	$-G_y$	300	201.5	81.5	2.5×10^4	-	Zero	134	F	597.5	32.2	5.39	$+G_y$	400	151.8	41.8	3.6×10^4	+	24
105	M	456.5	28.6	6.27	$-G_y$	350	162.7	62.7	2.6×10^4	+	96	135	M	837.0	36.9	4.41	$+G_y$	150	129.5	79.5	1.6×10^4	+	24
106	YF	435.5	26	5.97	$-G_y$	400	167.4	57.4	3.0×10^4	-	Zero	136	F	585.0	30.6	5.23	$+G_y$	350	148.9	48.9	3.0×10^4	+	24
107	M	554.5	34.4	6.32	$-G_y$	100	181	181	1.0×10^4	-	Zero	137	F	469.1	27.6	5.88	$+G_y$	400	178.4	53.4	3.4×10^4	+	24
108	M	528.6	33.3	6.30	$-G_y$	200	209.2	94.7	2.2×10^4	+	72	138	M	653.5	37.8	5.78	$+G_x$	50	257	307	8.5×10^3	+	24
109	M	686.0	40.4	5.89	$-G_y$	350	100	41.6	2.9×10^4	+	72	139	M	761.3	37.9	4.98	$+G_x$	50	238	263	9.1×10^3	+	24
110	M	678.7	35.9	5.16	$-G_y$	100	100	147	6.7×10^3	+	48	140	M	555.2	31.9	5.75	$+G_x$	100	200	180	1.1×10^4	+	24
111	F	486.2	26.7	5.49	$-G_y$	100	200	206	9.7×10^3	+	48	141	F	498.2	28.2	5.66	$+G_x$	150	183.5	133.5	1.4×10^4	+	48
112	F	420.7	24.2	5.76	$-G_y$	50	386	476	8.1×10^3	-	Zero	142	F	510.9	28.1	5.50	$+G_x$	150	180.5	130.5	1.4×10^4	+	48

TABLE X (CONTINUED)
SUMMARY OF TEST RESULTS BY ANIMAL NUMBRR

ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.
143	YM	486.2	30.2	6.21	$+G_x$	200	203	103	2.0×10^4	+	48
144	F	450.2	24.4	5.42	$+G_x$	100	242	222	1.1×10^4	-	Zero
145	F	441.7	28.8	6.34	$+G_x$	250	211	91	2.3×10^4	-	Zero
146	F	545.5	31.2	5.72	$+G_x$	250	200	73.5	2.7×10^4	-CH	Zero
147	YM	495.3	32.2	6.50	$+G_x$	300	200	67.5	3.0×10^4	-CH	Zero
148	F	488	31.2	6.39	$+G_x$	50	341	411	1.0×10^4	-	Zero
149	M	488	21.2	4.34	$+G_x$	300	188.4	68.4	2.8×10^4	+	24
150	F	581.7	31.1	5.35	$+G_x$	300	174.4	57.4	3.1×10^4	+	24
151	M	441.7	26.9	6.09	$+G_x$	300	205.5	75.5	2.7×10^4	-	Zero
152	YF	462.0	28.7	6.21	$+G_x$	200	208.5	108.5	1.9×10^4	-	Zero
153	F	455.0	24.1	5.29	$+G_x$	400	200	55	3.7×10^4	-CH	Zero
154	F	464.0	24.1	5.19	$+G_x$	350	191.7	61.7	3.1×10^4	-CH	Zero
155	F	536.0	30.2	5.63	$+G_x$	400	163.7	46.7	3.5×10^4	+	48
156	F	494.6	31.7	6.41	+G	300	189.7	67.7	2.8×10^4	-	Zero
157	YM	396.6	-	-	$+G_x$	300	100	84.0	1.2×10^4	+	101 days
158	F	411.3	26.2	6.37	$+G_x$	300	181.2	81.2	2.2×10^4	+	24
159	F	557.3	37.5	6.73	$+G_x$	350	171.4	51.4	3.4×10^4	+	24
160	F	460.0	25.8	5.61	$+G_x$	350	182.3	62.3	2.9×10^4	-	Zero
161	F	541.2	30.8	5.69	$+G_x$	350	172.8	52.8	3.3×10^4	-	Zero
162	F	486.7	29.1	5.98	$+G_x$	350	168.9	58.9	2.9×10^4	-	Zero
163	F	547.0	27.9	5.10	$+G_x$	350	172.3	52.3	3.3×10^4	+	48
164	F	531.0	27.9	5.25	$+G_x$	350	173.8	53.8	3.2×10^4	-CH	Zero
165	F	543.5	25.9	4.77	$+G_x$	400	161.2	46.2	3.5×10^4	+	48
166	YM	495.0	29.8	6.02	$+G_x$	400	170.5	50.5	3.4×10^4	-	Zero
167	F	417.0	24.2	5.80	$+G_x$	400	160	60	2.7×10^4	-	Zero
168	F	531.3	28.4	5.35	$+G_x$	400	165	47	3.5×10^4	+	48
169	F	494.2	30.2	6.11	$+G_x$	400	180	50.7	3.4×10^4	-	Zero
170	F	629.3	35.2	5.59	$+G_x$	400	140	39.1	3.5×10^4	-	Zero
171	F	547.1	25.3	4.62	$+G_x$	150	191.5	121.5	1.6×10^4	+	48
172	F	500	24.3	4.86	$+G_x$	250	200	80	2.5×10^4	-	Zero

ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.
173	M	500	29.1	5.82	$+G_x$	250	180	80	2.3×10^4	-CH	Zero
174	F	546.5	27.4	5.01	$+G_x$	100	223	183	1.2×10^4	-CH	Zero
175	F	593.3	29.2	4.92	$+G_x$	200	200	84.4	2.4×10^4	+	24
176	YF	536.0	27.7	5.17	$+G_x$	150	194.5	124.5	1.6×10^4	+	24
177	F	545	26.7	4.90	$+G_x$	250	183.4	73.4	2.5×10^4	+	24
178	F	535	30.4	5.68	$+G_x$	250	185	75	2.5×10^4	-	Zero
179	F	569.0	29.3	5.15	$+G_x$	50	300	352	8.5×10^3	+	24
180	M	663.8	31.2	4.70	$+G_x$	100	151	151	1.0×10^4	+	24
181	M	657.4	32.7	4.97	$+G_x$	150	191.5	101.5	1.9×10^4	-	Zero
182	M	738.6	35.1	4.75	$+G_x$	200	167.8	67.8	2.5×10^4	+	24
183	F	529.5	30.7	5.80	$+G_x$	250	195.6	75.6	2.6×10^4	-CH	Zero
184	F	516.8	28.1	5.44	$+G_x$	250	192.5	77.5	2.5×10^4	-	Zero
185	F	550.0	31.0	5.64	$+G_x$	250	192.9	72.9	2.7×10^4	+	24
186	M	643.2	40.1	6.23	$+G_x$	200	200	78	2.6×10^4	+	24
187	M	749.0	34.9	4.66	$-G_x$	50	206.5	106.5	7.8×10^3	+	24
188	M	836.2	44.7	5.35	$-G_x$	50	239	95.6	1.0×10^4	-	Zero
189	F	558.2	32.2	5.77	$-G_x$	50	287	144	8.0×10^3	-	Zero
190	F	495.5	22.3	4.50	$-G_x$	50	286.5	161.5	7.1×10^3	-CH	Zero
191	M	680	33.3	4.90	$-G_x$	100	118	59	8.0×10^3	+	24
192	F	630	31.6	5.01	$-G_x$	100	138.5	63.5	8.8×10^3	+	24
193	F	487.7	30.2	6.20	$-G_x$	100	157.2	82.2	9.7×10^3	+	24
194	F	548.6	35.9	6.54	$-G_x$	100	163.2	73.2	9.0×10^3	-CH	Zero
195	F	483.5	28.2	5.83	$-G_x$	150	110.4	55.2	8.0×10^3	-	Zero
196	F	559.4	30.0	5.36	$-G_x$	150	100	47.7	8.4×10^3	+	24
197	M	647.2	30.0	5.08	$-G_x$	150	103	41.3	1.0×10^4	+	24
198	F	602.7	33.0	5.48	$-G_x$	150	119.4	44.4	1.1×10^4	+	24
199	F	542.1	26.8	4.94	$-G_x$	200	92	36.9	1.0×10^4	+	96
200	F	575.1	31.2	5.43	$-G_x$	200	109.8	34.8	1.3×10^4	+	24
201	M	451	27.0	5.99	$-G_x$	200	119.4	44.4	1.3×10^4	+	48
202	M	631.5	34.8	5.51	$-G_x$	200	131.7	31.7	1.7×10^4	+	96

TABLE X (CONCLUDED)
SUMMARY OF TEST RESULTS BY ANIMAL NUMBER

ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.
203	M	494.7	32.2	6.51	$-G_x$	250	132.4	32.4	$1.6x10^4$	-CH	Zero
204	F	641.3	36.2	5.64	$-G_x$	250	120	25	$1.9x10^4$	+	24
205	M	583.1	35.6	6.11	$-G_x$	250	127.5	27.5	$1.9x10^4$	-	Zero
206	M	731.3	35.2	4.81	$-G_x$	250	104	21.9	$1.0x10^4$	+	24
207	F	504.1	29.5	5.85	$-G_x$	300	125	26.5	$1.9x10^4$	-	Zero
208	F	539.6	34.3	6.36	$-G_x$	300	117.4	24.7	$1.9x10^4$	+	24
209	F	489.5	25.3	5.17	$-G_x$	300	127.3	27.3	$1.9x10^4$	-	Zero
210	F	435.5	26.1	5.99	$-G_x$	300	120.6	30.6	$1.6x10^4$	+	48
211	F	367	22.4	6.10	$-G_x$	300	116.3	36.3	$1.3x10^4$	-	Zero
212	F	452.7	24.4	5.39	$-G_x$	300	119.5	29.5	$1.6x10^4$	+	24
213	M	498.0	31.2	6.25	$-G_x$	300	116.8	26.8	$1.7x10^4$	+	24
214	F	462.0	24.5	5.31	$-G_x$	300	128.9	28.9	$1.8x10^4$	-	Zero
215	F	440.5	26.1	5.93	$-G_x$	350	65	25.9	$1 x10^4$	+	96
216	YM	431.0	-	-	$-G_x$	350	112.5	26.5	$1.7x10^4$	+	-
217	F	486.0	26.0	5.35	$-G_x$	350	113.5	23.5	$1.9x10^4$	-	Zero
218	F	459.0	26.3	5.73	$-G_x$	350	112	24.9	$1.8x10^4$	-	Zero
219	M	463.2	24.5	5.29	$-G_x$	350	111	24.7	$1.8x10^4$	-	Zero
220	F	485.8	23.4	4.82	$-G_x$	350	108.5	23.5	$1.8x10^4$	+	120
221	M	506.3	25.8	5.10	$-G_x$	350	107.6	22.6	$1.9x10^4$	+	96
222	M	583.5	31.8	5.45	$-G_x$	350	94.6	19.6	$1.9x10^4$	+	120
223	YM	377.8	17.5	4.63	$-G_x$	350	115.3	30.3	$1.5x10^4$	+	22 days

ANIMAL					STRESS						
No	Sex	Wt gm	Tail Wt gm	Tail Wt %	Mode	nG	D_r	D_t	K_p	+/-	Autopsy Time Hr.
224	F	566.1	34.5	6.09	$-G_x$	350	105.2	20.2	$2.1x10^4$	+	22 days
225	F	495.0	29.0	5.86	$-G_x$	400	95.2	20.2	$1.9x10^4$	+	24
226	F	604.0	38.0	6.29	$-G_x$	400	83	16.6	$2.0x10^4$	+	24
227	F	508.0	29	5.71	$-G_x$	400	108	19.7	$2.2x10^4$	+	48
228	F	505.2	36.6	7.24	$-G_x$	400	120	19.8	$2.4x10^4$	-CH	Zero
229	F	521.0	30.4	5.84	$-G_x$	400	114.2	19.2	$2.4x10^4$	+	24
230	F	430.0	23.3	5.42	$-G_x$	400	118.3	23.3	$2.2x10^4$	-	Zero
231	M	648.8	38.4	5.92	$-G_x$	Not run				*	Zero
232	M	612.6	-	-	$-G_x$	400	98	16.3	$2.4x10^4$	+	25 days
233	F	498.5	25.4	5.1	$-G_x$	400	115.5	20.5	$2.3x10^4$	-	Zero
234	F	610.0	32.0	5.25	$-G_x$	400	106.4	16.4	$2.6x10^4$	+	17 days
235	YM	482.5	28.8	5.97	$-G_x$	400	115.7	20.7	$2.2x10^4$	-CH	24
236	M	561.0	-	-	$-G_x$	350	120.4	20.4	$2.4x10^4$	+	25 days
237	M	533.4	29.9	5.61	$+G_x$	430	120.0	43.6	$2.8x10^4$	-CH	Zero
238	M	544.8	29.3	5.38	$+G_x$	200	207	92	$2.3x10^4$	-	Zero
239	F	511.6	27.9	5.28	$+G_x$	430	115.6	45.6	$2.5x10^4$	+	11 days
240	YM	430.5	-	-	$+G_x$	430	124.1	54.1	$2.3x10^4$	+	25 days
241	M	601.5	See 242		$+G_x$	200	213	83	$2.6x10^4$	+	see 242
242	M	695	-	-	$+G_x$	200	202.5	72.5	$2.8x10^4$	+	Zero
243	?	?	-	-	No Run Control - Pento Only						Zero

LEGEND

+ = Survival

- = Fatality

-CH = "Cliff Hanger"

D_r = Seconds at dwell

D_t = Seconds to threshold

K_p = Kilogram - G - seconds

TABLE XI. SUMMARY OF TEST CELLS K_p BY MODE AND G LEVEL

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
+ 50 G_x						
2	1.0×10^2	.623	6.2×10^1	+	F	40
20	1.0×10^3	.549	5.5×10^2	+	M	41
200	1.0×10^4	.400	4.0×10^3	+	F	42
238	1.3×10^4	.761	9.1×10^3	+	M	139
300	1.3×10^4	.653	8.5×10^3	+	M	138
300	1.5×10^4	.569	8.5×10^3	+	F	179
341	1.7×10^4	.488	1.0×10^4	-	F	148
+ 100 G_x						
2	2.0×10^2	.433	8.7×10^1	+	F	37
20	2.0×10^3	.653	1.3×10^3	+	M	38
151	3.5×10^4	.664	1.0×10^4	+	M	180
200	2.0×10^4	.555	1.1×10^4	+	M	140
200	2.0×10^4	.764	1.5×10^4	-	M	39
223	2.2×10^4	.547	1.2×10^4	-	F	174
242	2.4×10^4	.450	1.1×10^4	-	F	144
+ 150 G_x						
2	3.0×10^2	.508	1.5×10^2	+	F	34
20	3.0×10^3	.836	2.5×10^3	+	M	35
180	2.7×10^4	.511	1.4×10^4	+	F	142
183	2.7×10^4	.498	1.4×10^4	+	F	141
192	2.9×10^4	.547	1.6×10^4	+	F	171
192	2.9×10^4	.657	1.9×10^4	-	M	181
195	2.9×10^4	.536	1.6×10^4	+	YF	176
200	3.0×10^4	.704	2.1×10^4	-	M	36
+ 200 G_x						
2	4.0×10^2	.469	1.9×10^2	+	F	43
20	4.0×10^3	.429	1.7×10^3	+	F	44
168	3.6×10^4	.739	2.5×10^4	+	M	182
200	4.0×10^4	.423	1.7×10^4	+	M	46
200	4.0×10^4	.593	2.4×10^4	+	F	175
200	4.0×10^4	.643	2.6×10^4	+	M	186
203	4.1×10^4	.462	2.0×10^4	+	YM	143
203	4.1×10^4	.695	2.8×10^4	+	M	242
207	4.1×10^4	.545	2.3×10^4	-	M	238
208	4.1×10^4	.462	1.9×10^4	-	YF	152
213	4.3×10^4	.602	2.6×10^4	+	M	241
+ 250 G_x						
2	5.0×10^2	.642	3.2×10^2	+	M	27
2	5.0×10^2	.734	3.6×10^2	+	M	26
20	5.0×10^3	.713	3.6×10^3	+	M	29
180	4.5×10^4	.500	2.3×10^4	-	M	173
184	4.6×10^4	.545	2.5×10^4	+	F	177
185	4.6×10^4	.535	2.5×10^4	-	F	178
193	4.8×10^4	.517	2.5×10^4	-	F	184

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
+ 250 G_x (Continued)						
193	4.8×10^4	.550	2.7×10^4	+	F	185
196	4.9×10^4	.530	2.6×10^4	-	F	183
200	5.0×10^4	.500	2.5×10^4	-	F	172
200	5.0×10^4	.546	2.7×10^4	-	F	146
200	5.0×10^4	.734	3.7×10^4	+	M	30
211	5.2×10^4	.442	2.3×10^4	-	F	145
+ 300 G_x						
2	6.0×10^2	.608	3.6×10^2	+	M	31
20	6.0×10^3	.587	3.5×10^3	+	M	32
100	3.0×10^4	.397	1.2×10^4	+	YM	157
131	3.9×10^4	.411	2.2×10^4	+	F	158
177	5.3×10^4	.582	3.1×10^4	+	M	150
189	5.7×10^4	.488	2.8×10^4	+	M	149
190	5.7×10^4	.495	2.8×10^4	-	F	156
200	6.0×10^4	.495	3.0×10^4	-	YM	147
200	6.0×10^4	.598	3.6×10^4	-	M	33
206	6.2×10^4	.442	2.7×10^4	-	M	151
+ 350 G_x						
2	7.0×10^2	.517	3.4×10^2	+	F	47
20	7.0×10^3	.482	3.5×10^3	+	F	48
169	5.9×10^4	.487	2.9×10^4	-	F	162
171	6.0×10^4	.567	3.4×10^4	+	F	159
172	6.0×10^4	.547	3.3×10^4	+	F	163
173	6.1×10^4	.541	3.3×10^4	-	F	161
174	6.1×10^4	.567	3.4×10^4	-	F	171
182	6.4×10^4	.460	2.9×10^4	-	F	160
192	6.6×10^4	.464	3.1×10^4	-	F	154
200	7.0×10^4	.504	3.5×10^4	-	M	49
+ 400 G_x						
2	8.0×10^2	.479	3.8×10^2	+	F	50
20	8.0×10^3	.469	3.8×10^3	+	M	51
140	5.6×10^4	.629	3.5×10^4	-	F	170
160	6.4×10^4	.417	2.7×10^4	-	F	167
161	6.4×10^4	.543	3.5×10^4	+	F	165
164	6.6×10^4	.536	3.5×10^4	+	F	155
165	6.6×10^4	.531	3.5×10^4	+	F	168
171	6.8×10^4	.495	3.4×10^4	-	YM	166
180	7.2×10^4	.494	3.4×10^4	-	F	169
200	8.0×10^4	.455	3.7×10^4	-	F	153
+ 430 G_x						
116	5.0×10^4	.512	2.5×10^4	+	F	239
120	5.2×10^4	.533	2.8×10^4	-	M	237
124	5.3×10^4	.431	2.3×10^4	-	YM	240

TABLE XI. (CONTINUED)
SUMMARY OF TEST CELLS K_p BY MODE AND G LEVEL

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
		- 50 G_x				
2	1.0×10^2	.380	3.8×10^1	+	M	54
20	1.0×10^3	.437	4.4×10^2	+	M	53
200	1.0×10^4	.353	3.5×10^3	+	M	52
206	1.0×10^4	.749	7.8×10^3	+	M	187
239	1.2×10^4	.836	1.0×10^4	-	M	188
286	1.4×10^4	.495	7.1×10^3	-	F	190
287	1.4×10^4	.558	8.0×10^3	-	F	189
		- 100 G_x				
2	2.0×10^2	.492	9.8×10^1	+	M	55
20	2.0×10^3	.429	8.6×10^2	+	M	56
118	1.2×10^4	.680	8.0×10^3	+	M	191
138	1.4×10^4	.630	8.8×10^3	+	F	192
157	1.6×10^4	.488	9.7×10^3	+	F	193
163	1.6×10^4	.549	9.0×10^3	-	F	194
200	2.0×10^4	.355	7.1×10^3	-	M	57
		- 150 G_x				
2	3.0×10^2	.500	1.5×10^2	+	F	58
20	3.0×10^3	.568	1.7×10^3	+	F	59
100	1.5×10^4	.560	8.4×10^3	+	F	196
103	1.5×10^4	.647	1.0×10^4	+	M	197
110	1.7×10^4	.484	8.0×10^3	-	F	195
119	1.8×10^4	.603	1.1×10^4	+	M	198
200	3.0×10^4	.518	1.6×10^4	-	F	63
		- 200 G_x				
2	4.0×10^2	.537	2.2×10^2	+	F	60
20	4.0×10^3	.538	2.2×10^2	+	F	62
92	1.8×10^4	.542	1.0×10^4	+	F	199
110	2.2×10^4	.575	1.3×10^4	+	F	200
119	2.4×10^4	.451	1.3×10^4	+	M	201
132	2.6×10^4	.632	1.7×10^4	+	M	202
200	4.0×10^4	.711	2.8×10^4	-	M	67
		- 250 G_x				
2	5.0×10^2	.600	3.0×10^2	+	M	64
20	5.0×10^3	.507	2.5×10^3	+	M	65
104	2.7×10^4	.731	1.9×10^4	+	M	206
120	5.0×10^4	.641	1.9×10^4	+	F	204

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
		- 250 G_x (Continued)				
128	3.2×10^4	.583	1.9×10^4	-	M	205
132	3.3×10^4	.495	1.6×10^4	-	M	203
200	5.0×10^4	.638	3.2×10^4	-	M	66
		-300 G_x				
20	6.0×10^3	.566	3.4×10^3	+	M	68
116	3.5×10^4	.367	1.3×10^4	-	F	211
117	3.5×10^4	.468	1.7×10^4	+	M	213
117	3.5×10^4	.540	1.9×10^4	+	F	208
120	3.6×10^4	.453	1.6×10^4	+	F	212
121	3.6×10^4	.436	1.6×10^4	+	F	210
125	3.8×10^4	.504	1.9×10^4	-	F	207
127	3.8×10^4	.490	1.9×10^4	-	F	204
129	3.9×10^4	.462	1.8×10^4	-	F	214
		- 350 G_x				
20	7.0×10^3	.568	4.0×10^3	-	M	69
65	2.3×10^4	.441	1.0×10^4	+	F	215
95	3.3×10^4	.584	1.9×10^4	+	M	222
105	3.7×10^4	.566	2.1×10^4	+	F	224
108	3.8×10^4	.506	1.9×10^4	+	M	221
109	3.8×10^4	.486	1.8×10^4	+	F	220
111	3.9×10^4	.463	1.8×10^4	-	M	219
112	3.9×10^4	.459	1.8×10^4	-	F	218
113	4.0×10^4	.431	1.7×10^4	+	YM	216
114	4.0×10^4	.486	1.9×10^4	-	F	217
115	4.0×10^4	.378	1.5×10^4	+	YM	223
		- 400 G_x				
83	3.3×10^4	.604	2.0×10^4	+	F	226
95	3.8×10^4	.495	1.9×10^4	+	F	225
98	3.9×10^4	.613	2.4×10^4	+	F	232
106	4.2×10^4	.610	2.6×10^4	+	M	234
108	4.3×10^4	.508	2.2×10^4	+	F	227
114	4.6×10^4	.521	2.4×10^4	+	F	229
116	4.6×10^4	.483	2.2×10^4	-	YM	235
116	4.6×10^4	.499	2.3×10^4	-	F	233
118	4.7×10^4	.430	2.2×10^4	-	F	230
120	4.8×10^4	.505	2.4×10^4	-	F	228

TABLE XI. (CONCLUDED)
SUMMARY OF TEST CELLS K_p BY MODE AND G LEVEL

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
		+ 50 G_y				
2	1.0×10^2	.656	6.6×10^1	+	F	70
20	1.0×10^3	.698	7.0×10^2	+	M	71
200	1.0×10^4	.549	5.5×10^3	+	F	72
289	1.5×10^4	.693	1.0×10^4	-	M	119
320	1.6×10^4	.541	8.6×10^3	+	M	121
		+ 150 G_y				
2	3.0×10^2	.546	1.5×10^1	+	F	73
20	3.0×10^3	.560	1.2×10^3	+	F	74
130	2.0×10^4	.837	1.6×10^4	+	M	135
200	3.0×10^4	.532	1.6×10^4	-	M	75
200	3.0×10^4	.571	1.7×10^4	-	M	118
		+ 250 G_y				
2	5.0×10^2	.564	2.8×10^2	+	M	76
20	5.0×10^3	.613	3.1×10^3	+	F	77
174	4.4×10^4	.540	2.8×10^4	+	F	130
200	5.0×10^4	.560	2.8×10^4	-	F	78
203	5.1×10^4	.487	2.4×10^4	+	F	117
		+ 300 G_y				
2	6.0×10^2	.723	4.3×10^2	+	F	79
20	6.0×10^3	.596	3.5×10^3	+	F	80
140	4.0×10^4	.713	3.0×10^4	+	M	132
185	5.6×10^4	.511	2.9×10^4	+	F	120
185	5.6×10^4	.543	3.0×10^4	+	F	131
		+ 350 G_y				
2	7.0×10^2	.617	4.3×10^2	+	F	81
20	7.0×10^3	.313	2.2×10^3	+	M	82
149	5.2×10^4	.585	3.0×10^4	+	F	136
163	5.7×10^4	.538	3.1×10^4	+	F	129
178	6.2×10^4	.599	3.7×10^4	-	F	123
		+ 400 G_y				
20	8.0×10^3	.445	3.6×10^3	+	F	83
139	5.6×10^4	.648	3.5×10^4	+	M	122
152	6.1×10^4	.598	3.6×10^4	+	F	134
154	6.2×10^4	.570	3.5×10^4	+	F	133
164	6.6×10^4	.519	3.4×10^4	+	YF	128
171	6.8×10^4	.542	3.7×10^4	-	F	127
174	7.0×10^4	.507	3.5×10^4	+	M	126
178	7.1×10^4	.469	3.4×10^4	+	F	137
179	7.2×10^4	.466	3.3×10^4	-	F	125
200	8.0×10^4	.478	3.8×10^4	-	F	124

STRESS		ANIMAL				
Time Sec.	G-Sec.	kg wt	K_p	+/-	Sex	No.
		- 50 G_y				
189	9.5×10^3	.693	6.4×10^3	+	F	116
266	1.3×10^4	.519	6.9×10^3	+	F	115
386	2.0×10^4	.420	8.1×10^3	+	F	112
		- 100 G_y				
2	2.0×10^2	.583	1.2×10^2	+	F	84
20	2.0×10^3	.547	1.1×10^3	+	M	85
100	1.0×10^4	.378	6.8×10^3	+	M	110
181	1.8×10^4	.555	1.0×10^4	-	M	107
200	2.0×10^4	.486	9.7×10^3	+	F	111
200	2.0×10^4	.438	8.8×10^3	+	F	86
210	2.1×10^4	.576	1.2×10^4	+	F	101
		- 200 G_y				
2	4.0×10^2	.514	2.1×10^2	+	F	87
20	4.0×10^3	.543	2.2×10^3	+	F	88
200	4.0×10^4	.540	2.2×10^4	+	F	93
210	4.2×10^4	.529	2.2×10^4	+	M	108
228	4.6×10^4	.462	2.1×10^4	-	F	102
		- 300 G_y				
2	6.0×10^2	.567	3.4×10^2	+	F	90
20	6.0×10^3	.579	3.5×10^3	+	F	91
200	6.0×10^4	.465	2.8×10^4	+	F	96
200	6.0×10^4	.285	1.7×10^4	-	F	103
202	6.1×10^4	.409	2.5×10^4	-	F	104
		- 350 G_y				
2	7.0×10^2	.500	3.5×10^2	+	F	94
20	7.0×10^3	.591	4.2×10^3	+	F	95
100	3.5×10^4	.686	2.4×10^4	+	M	109
163	5.7×10^4	.457	2.5×10^4	+	M	105
178	6.2×10^4	.497	3.1×10^4	+	F	114
200	7.0×10^4	.529	3.7×10^4	-	F	97
		- 400 G_y				
2	8.0×10^2	.483	3.9×10^2	+	F	98
20	8.0×10^3	.516	4.1×10^3	+	F	99
167	6.7×10^4	.436	3.0×10^4	-	YF	106
170	6.8×10^4	.502	3.4×10^4	+	M	113
100	8.0×10^4	.456	3.7×10^4	+	F	100

APPENDIX B
CHARTS AND PROTOCOLS

This Appendix contains the various charts and protocols used in this experiment. The formats demonstrated herein are arranged as follows:

LISTS OF LABORATORY MATERIALS

Table XII. - Run List
Table XIII. - Autopsy List

RECORD CHARTS

Figure 30. - Log Book Form
Figure 31. - Initial Health Survey, Centrifugation Record and Clinical Progress Record
Figure 32. - Necropsy-Necrotomy and Histopathology Protocol

PROCEDURAL PROTOCOLS

Table XIV. - Centrifugation Experiment Daily Time Schedule
Table XV. - Tissue Preservation Protocol
Table XVI. - Tissue Selection Criteria
Table XVII.- H & E Frozen Section Technique
Table XVIII.-Incubating Solution for Oxidative Enzymes

Usage.- With the exception of Table XVII and Table XVIII, the above materials, charts, and protocols were utilized on a daily basis. Table XVII and Table XVIII were utilized on only three animals No. 241-242, No. 157, and No. 243. The completed forms on each of the test animals are on file at the investigator's laboratory. Reference is made to these formats in the main body of the report. The entries are self-explanatory.

TABLE XII. LIST OF RUN LABORATORY MATERIALS AND EQUIPMENT

PREPARATION TABLE

Restraint board with four thongs
Instrument tray;
- Forceps, 1 large, 2 small
- Scissors, 3 pair
- Hemostats, 2 large, 1 small
- Screwdriver-- for couch
- Ether cone in 150 ml beaker
- Ear punch
- Ear tags
- Safety razor, fresh blade

Supply tray:
- Gauze sponges, 4 x 4's
- Beaker--soapy water
- Beaker--alcohol soaked sponges
- Aeroplast-(Plastic spray dressing)
- Q-Tips, 6" size
- Adhesive tape, 2 rolls
- Adhesive discs, 4 per animal
- Bandaids, 5 per animal

LABORATORY COUNTER

Centrifuge couch
Electrodes, 2 sets of 3 each
Electrode jelly
Small spatula to fill electrodes
Shaver, electric
Alcohol
Scales, triple beam and weights
Ether
Beaker, 30 ml., and watch-glass cover
Hypodermic syringe (to measure ether)
Triple gauze sponge, one per animal
Covered container for used sponges
Stethoscope
Stopwatch
Laboratory notebook
Pen
Appropriate animal folders
Surgeon's gloves
Surgical masks

ANIMAL TABLE

Papers to cover table
Animals in respective squeeze cages
(Brought in warmed station wagon from animal holding facility)
Animal handling gloves, 2 pairs
Anesthetic box (for animal)
Container to hold drinking tubes
Hook to remove perch from cages
Tags for cages

TABLE XIII LIST OF AUTOSPY LABORATORY MATERIALS AND EQUIPMENT

AUTOPSY TABLE

Restraint board with four thongs
Dissecting board
Dissecting pans, 2
Beakers, 600 ml., water filled, (2)
Specimen jars, 10% formaldehyde, 2/3 filled
Paper towels
Gauze sponges, 4 x 4's
Paper bags, large size for burnable wastes
small for extraneous tissues
Instrument tray:
Scalpel, #15 blade
Rongeur
Forceps, plain & mouse-toothed
Probe
Hemostats, straight--1 small, 2 large
toothed-- 1 small
Scissors, assorted sizes, 3 pairs
Dissecting pins

LABORATORY COUNTER

Bone saw
Scales, triple beam and weights
Pento (Sodium Pentobarbital)
Hypodermic syringe, 20 gauge needle
Surgeon's gloves
Necropsy-necrotomy protocol forms
Clip board and carbon paper
Pen
Equipment checklist, grease pencil
Scratch pad
Scrub suits
Surgical masks

ANIMAL TABLE

Papers to cover table
Animals in respective squeeze cages
(Brought in warmed station wagon
from animal holding facility)
Animal handling gloves, 2 pairs
Anesthetic box (for animal)
Container to hold drinking tubes
Hook to remove perch from cages

Run No. _____ Intention Summary _____ Page No. 94

Date: _____

Monkey No.: _____ Sex: _____ G_o _____

Personnel: _____ D_r _____

Weight Determination: D_t _____

$\frac{B + A}{- B}$ _____ K_t _____

A K_p _____

$W_o = A \times 10^{-3}$ _____ W_o _____

DB _____ WB _____ RH _____ Time: _____ Initial Anesthetic Delivered

Type: _____

Amount: _____

Calculations:

Predicted Outcome: _____

Actual Outcome: _____

Threshold Calculation: $D_t = \frac{K_t}{W_o \cdot G_o} = \_\_\_\_ =$

Duration of Run: $D_r = \frac{K_p}{W_o \cdot G_o} = \_\_\_\_ =$

$D_r - D_t =$

Time:

Clock	Exp.	Anesthetic Delivered _____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

WITNESSED _____

WITNESSED _____ SIGNATURE _____

FIGURE 30. LOG BOOK FORM

Monkey No. ______

INITIAL HEALTH SURVEY

Item	Checked	Comments
1. General Appearance		
2. HEENT		
3. Thorax		
4. Abdomen		
5. Limbs		
6. Tail		
7. Abn. Activity in Cage		
8. Normally Active		
9. Passive		
10. Huddles		
11. Aggressive when Provoked		
12. Food Intake		
13. Water Intake		
14. Stools Coloration		
15. Consistency		
16. Urine Coloration		
17. Other		

CENTRIFUGATION RECORD

1. Experimental Intention a/o Deviation	
2. Type of EKG Leads	
3. Pre-Run Consciousness	
4. Post-Run Consciousness	
5. Post-Run Animal Condition	
6. Nystagmus Type/Direction	
7. Head Position	
8. Anomalous Conditions	

Monkey No. ______

CLINICAL PROGRESS RECORD

Date	Time	Comments

FIGURE 31. INITIAL HEALTH SURVEY, CENTRIFUGATION RECORD AND CLINICAL PROGRESS RECORD

NECROPSY-NECROTOMY PROTOCOL

Project #500-NS-119 Code _____

Animal #_____ Species _____ Sex _____

Date Sacrificed _____ Method _____

Terminal Body Wt. _____

Prosector _____ Recorder _____

NECROPSY:

NECROTOMY: Tissue	Description	Sample Sent
Lung: Bronchi		
Heart & Gt. Vessels		
Esophagus		
Stomach		
Duodenum		
Small Intestine		
Large Intestine		
Pancreas		
Liver		
Gall Bladder		
Spleen		
Kidney		
Adrenals		
U. Bladder		
Ovaries/ Testes		
Uterus/ Prostate		
Brain		
Spinal cord		
Eye		
L/Node		
Other		

HISTOPATHOLOGY PROTOCOL

Project #500-NS-119 Code _____

Animal #_____ Species _____ Sex _____

Date Sacrificed _____ Method _____

Terminal Body Wt. _____

Prosector _____ Recorder _____

SUMMARY:

Tissue	Description	Sample Examined
Lung: Bronchi		
Heart & Gt. Vessels		
Esophagus		
Stomach		
Duodenum		
Small Intestine		
Large Intestine		
Pancreas		
Liver		
Gall Bladder		
Spleen		
Kidney		
Adrenals		
U. Bladder		
Ovaries/ Testes		
Uterus/ Prostate		
Brain		
Spinal Cord		
Eye		
L/Node		
Other		

FIGURE 32. NECROPSY-NECROTOMY AND HISTOPATHOLOGY PROTOCOL

TABLE XIV
CENTRIFUGATION EXPERIMENT TIME SCHEDULE

Time		Time	
0800-0900	Laboratory Preparation for Centrifugation a. Locate animal table and restraint board. b. Attach tie down thongs to restraint board. c. Prepare electrodes. d. Prepare masking discs. e. Position "Aeroplast" can. f. Cover bench with paper for protection. g. Prepare scrub suits, masks and gloves. h. Prepare anesthetic materials. i. Place sterile tray. j. Check gauze pad supply.	0900-0930	Animal Preparation a. Transfer to anesthesia box. b. Weigh. c. Anesthetize - see anesthetic schedule. d. Transfer to restraint board. e. Tag animal. f. Shave for electrodes. (R) arm (L) leg (L) chest. Mask with discs and spray with "Aeroplast" g. Secure electrodes. h. Place animal in couch and restrain. i. Test ECG output. j. Check animal for level of conciousness. k. Transfer to payload balance station; balance. l. Place couch on centrifuge. m. Secure pit area.
0830	Delivery of Animals		
0830	Electronics Equipment Warm up		
0900	Electronics Equipment Report AOK _____ No Go _____		

TABLE XIV (Cont'd.)
CENTRIFUGATION EXPERIMENT TIME SCHEDULE

Time		Time	
0930-0940	Master Generator Warm-up	1045-1100	Processing of ECG paper.
	Animal adaptation to centrifuge.		Prepare lab for animal #3.
0940	Base line ECG.	1100	Equipment status report.
	Verify run parameters.	1100	Preparation of animal #3. Repeat procedure of run #1.
0940	Test Run #1	1130-1140	Master Generator Warm-up
0945	Remove animal from couch and return to cage. Commence clinical observation.	1140	Base line ECG.
		1140	Test Run #3.
0945-1000	Processing of ECG paper. Prepare for animal #2.	1145	Remove animal from couch. and return to cage. Commence clinical observations.
1000	Run equipment status report.	1145-1200	Processing of ECG paper. Shutdown of equipment.
1000	Preparation of animal #2. Repeat procedure of run #1.	1200-1245	Lunch
1030-1040	Master Generator Warm-up	1245-1345	Laboratory Clean-up
1040	Base line ECG.		Gather instruments and sterilize.
1040	Test Run #2.		Scrub down laboratory.
1045	Remove animal from couch. and return to cage. Commence clinical observations.		

TABLE XV. TISSUE PRESERVATIVE PROTOCOL

Preparation of Preservative 1. 10% solution, 37% Formaldehyde 9 parts distilled H_2O to 1 part 37% Formaldehyde 2. Add 20 gm/ liter Sodium Acetate for buffer. 3. Use a minimum of 10 volumes of fixative, buffered, to 1 volume of tissue.
Preservation Technique 1. Fill bottles 2/3 full with solution. 2. Add tissue samples. 3. Fill to ¼ inch below mouth. 4. Insure all sample parts are covered by solution.

TABLE XVI. TISSUE SELECTION CRITERIA

1. Take 5-8 mm thick section of tissue with no unusual characteristics. 2. If any special feature is present on or in organ, take some normal tissue as well as the area of interest. Assure that the sample does not come apart. 3. When in doubt, send a sample.

TABLE XVII. HEMATOXYLIN AND EOSIN STAINING TECHNIQUE FOR FROZEN TISSUE SECTIONING

Step	Action	Material	Remarks
1	Cut section	Cryostat Microtome	6 Microns
2	Air dry		
3	Warm	37° C. Oven	1 Hour
4	Stain	Hemotoxylin	3 Minutes
5	Rinse	Tap water	
6	Decolorize	1% Acid Alcohol	
7	Rinse	Tap water	
8	Soak	Scotts Solution	1½ Minutes
9	Wash	Running tap water	10 Minutes
10	Dip	80% Alcohol	
11	Dip	95% Alcohol	
12	Stain	Eosin	1-2 Dips
13	Dip	95% Alcohol	
14	Dip	95% Alcohol	
15	Dip	100% Alcohol	
16	Dip	100% Alcohol Xylol	
17	Dip	Xylol	
18	Dip	Xylol	
19	Dip	Xylol	
20	Cover slip in Permount		

TABLE XVIII. INCUBATING SOLUTIONS FOR OXIDATIVE ENZYMES

DIAPHORASE:

1. 12 ml PO4 or Tris buffer (0.1M; pH 7.4)
2. 3 ml NBT (Nitro Blue Tetratsolium - 2 mg/ml of water)
3. 5 mg DPNH (Diphosphopyridine Nucleotide Hydrogen) Dissolve in buffer.
4. Incubate at 26 C for 15-30 minutes.
5. Washed in H20 - Rinsed 80% EtOH for 5 minutes - Washed in H20
6. Mounted in glycerol-gel

SUCCINIC DEHYDROGENASE:

1. 7 ml PO4 buffer (0.1M; pH 7.4)
2. 3 ml NBT (2 mg/ml H20)
3. 5 ml Succinate (Succinic Acid Disodium Salt - MW=270.16; 0.1M; 2.7 gm/100 ml)
4. Incubated at 26 C for 30-45 minutes.
5. Washed in H20 - Rinsed 80% EtOH for 5 minutes - Washed in H20
6. Mounted in glycerol-gel

NOTE - If sections were pretreated in organic solvents to remove lipids - add to the above incubating solution, 1 mg/ml (15 mg) of phenasine methosulfate.

LACTIC DEHYDROGENASE:

1. 7 ml Tris buffer (0.1M; pH 7.4)
2. 3 ml NBT (2 mg/ml H20)
3. 5 mg DPN (Diphosphopyridine Nucleotide) Dissolved in buffer.
4. 5 ml Lactic Acid (D or DL Lactic Acid Calcium Salt - MW=299.2; 0.08M; 17.95 mg/ml H20 - Dissolve by heating and neutralize to pH 7.2 with NaOH.)
5. Incubated at 26 C for 30 minutes.
6. Washed in H20 - Rinsed 80% EtOH - Washed H20
7. Mounted in glycerol-gel

MALIC DEHYDROGENASE:

1. 7 ml Tris buffer (0.1M; pH 7.4)
2. 3 ml NBT (2 mg/ml H20)
3. 5 mg DPN (Dissolved in buffer)
4. 5 ml Malic Acid (L-Malic Acid - MW=134.1; 0.07M; 9.39 mg/ml H20 - Dissolve by heating and neutralize with NaOH).
5. Incubated at 26 C for 40-60 minutes.
6. Washed in H20 - Rinsed in 80% EtOH for 5 minutes - Washed in H20
7. Mounted in glycerol-gel

NAPHTHYL ACETATE: (ESTERASE REACTION)

1. 20 mg Naphthyl Acetate in 1 ml of acetone
2. 20 mg Brentamine Fast Blue B in 15 ml of PO4 buffer (0.1M; pH 7.6)
3. Mix 2 solutions together, wait until mixture clears, filter and use. Incubate section 10 minutes. Wash in H20 and mount in glycerol-gel.

NOTE -

1. Store substrates for above reactions at -3° C (freeze), don't make more than 100 ml at one time as substrates deteriorate after about 6 months.
2. Keep buffers at 3-5 C.
3. Keep DPN and DPNH at 3-5 C in dark desiccated bottles.
4. To prepare stock solution of NBT, dissolve 400 mg of NBT in 208 ml H20 and store at 3-5 C in a dark bottle.
5. Substrates must be neutralized to pH 7.0 before using.

APPENDIX C
SELECTED SAMPLES OF ELECTROCARDIOGRAMS

This appendix is divided into four sections. Section One describes the recordings and the settings used for the traces. Section Two contains selected portions of the ECG from animal No. 190. Section Three contains records from animal No. 239, while Section Four compares the two records obtained three months apart on animal No. 241-242. The stress history on each animal is outlined at the beginning of each section.

SECTION ONE. DESCRIPTION OF RECORDING

The ECG examples shown are reproduced from the actual photogalvanometer record obtained during each run. The record has been photographically reduced to approximately 60% full size. The timing lines were set at one per second. Three paper speeds were utilized; these were 25, 50, and 100 mm/second. The paper speed can be determined by measuring the distance in millimeters between any two vertical time lines which are always one second apart, and correcting for the photo-reduction. Within the accuracy of the instrument, cumulative time errors were noted, and corrected at points determined by real time events, as explained in the Test Equipment section of this report.

The electrocardiogram traces, Trace No. 1 and Trace No. 3, were set during baseline checks for each animal so that a 10 millivolt output caused an excursion of three centimeters. These settings were not readjusted throughout the record. Trace No. 1 is the normal lead placement; Trace No. 3 is its mirror image. Filtration of the noise is the only other difference between these traces.

The accelerometer trace (Trace No. 4) was set at 50 G's per centimeter but is used only as a reference, rather than the prime speed control, because the actual peak G value was set by reference to an electronic counter as explained in the Test Equipment section. When the direction of load was negative ($-G_y$; $-G_x$), the trace was moved to the top of the chart paper so that the direction of galvanometer displacement would be toward the center of the record.

Trace No. 2 is a straight line trace of a stationary galvanometer used solely to check for paper warpage. The event marker

Trace No. 5 records the start and finish of the run. Trace No. 4 was also deflected and used as a marker to indicate beginning and ending of dwell, and at D_t.

Retouching of ECG was minimal and was utilized solely to accentuate faint lines for photographic purposes. The actual records are untouched and are available for study; as are the recordings for all the remaining animals.

SECTION TWO. ANIMAL NO. 190, SEX FEMALE

This animal was exposed to $-50\ G_x$ for a total dwell of 286.5 seconds. The animal's weight was 495.5 grams. Its calculated D_t was 161.5 seconds and D_r was 286.5 seconds. The K_p reached was 7.1×10^3. The animal was a cliff-hanger.

Figure 33. Animal No. 190 - Phase I, Baseline

The heart rate is 270 beats/min. Rhythym is sinus. QRS deflection measures 18 units (36 mm) and R wave measures 11 units (22 mm). Note the initial position of Trace No. 4 - this location is chosen due to deflection requirements for the $-G_x$ configuration.

Figure 34. Animal No. 190 - Phase II - Pre Run

Rate has dropped from 270 to 240 beats/min. Rhythym is sinus. Note the Trace No. 5 marker signifying the start of the run at the extreme right.

Figure 35. Animal No. 190 - Phase III - Onset, 0-2 Seconds

Note muscle tremor noise between one and two second timelines. Sinus arrythmia is demonstrated.

Figure 36. Animal No. 190 - Phase III - Onset, 2-4 Seconds

Sinus arrhythmia rate is 240. Note changes in voltage of QRS complex. Trace No. 4 is seen to be deflecting towards Trace No. 1. Note G levels attained at each time tic. Note difficulty in isolating P wave.

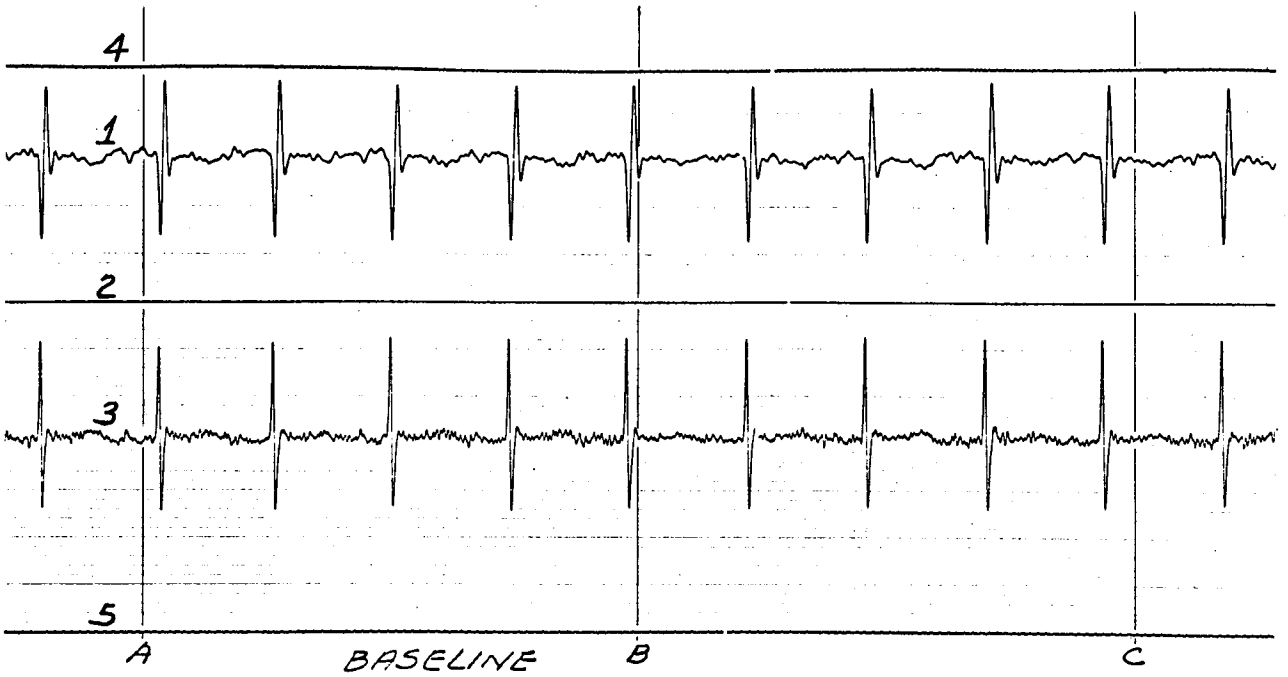


FIGURE 33. ANIMAL NO. 190. PHASE I, BASELINE

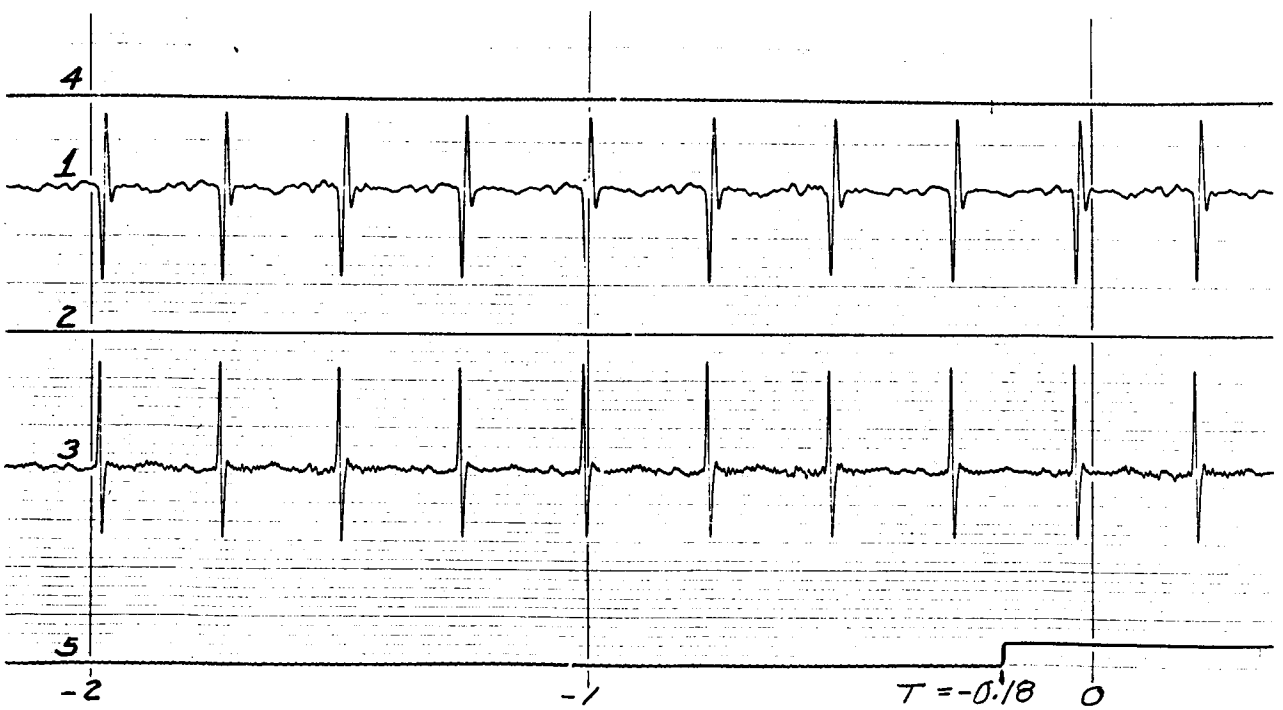


FIGURE 34. ANIMAL NO. 190. PHASE II, PRE-RUN

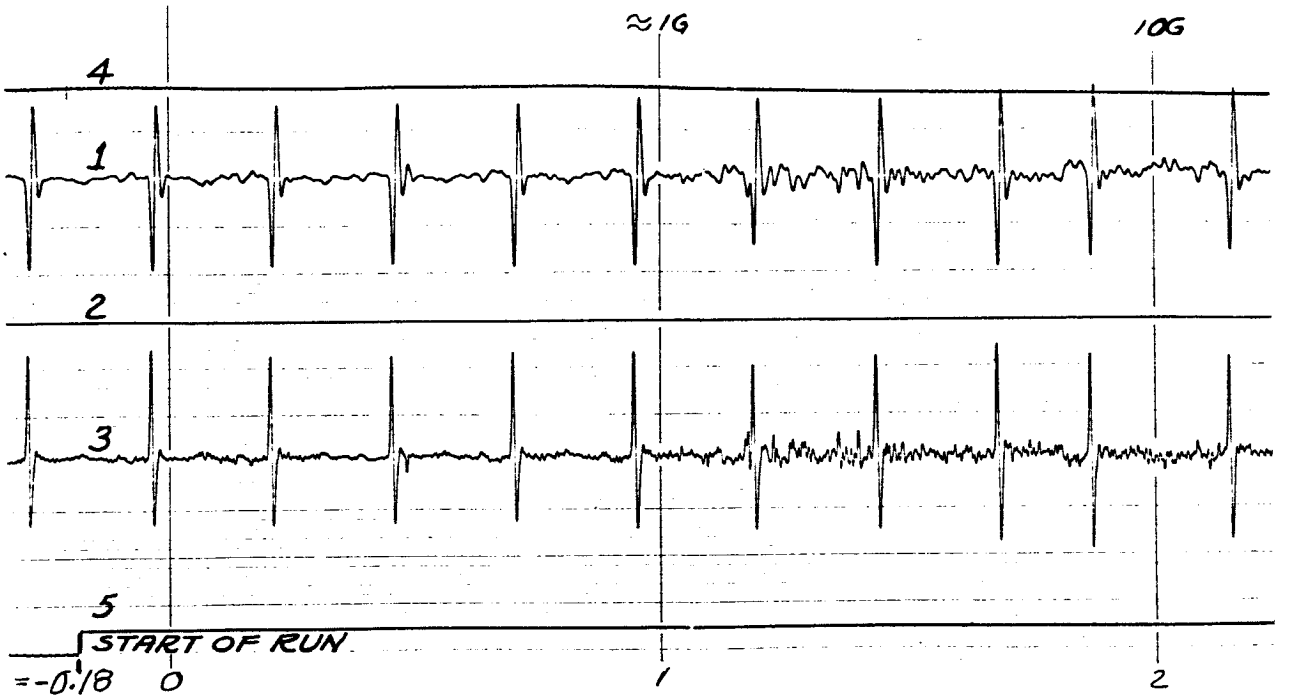


FIGURE 35. ANIMAL NO. 190. PHASE III - ONSET, 0-2 SECONDS

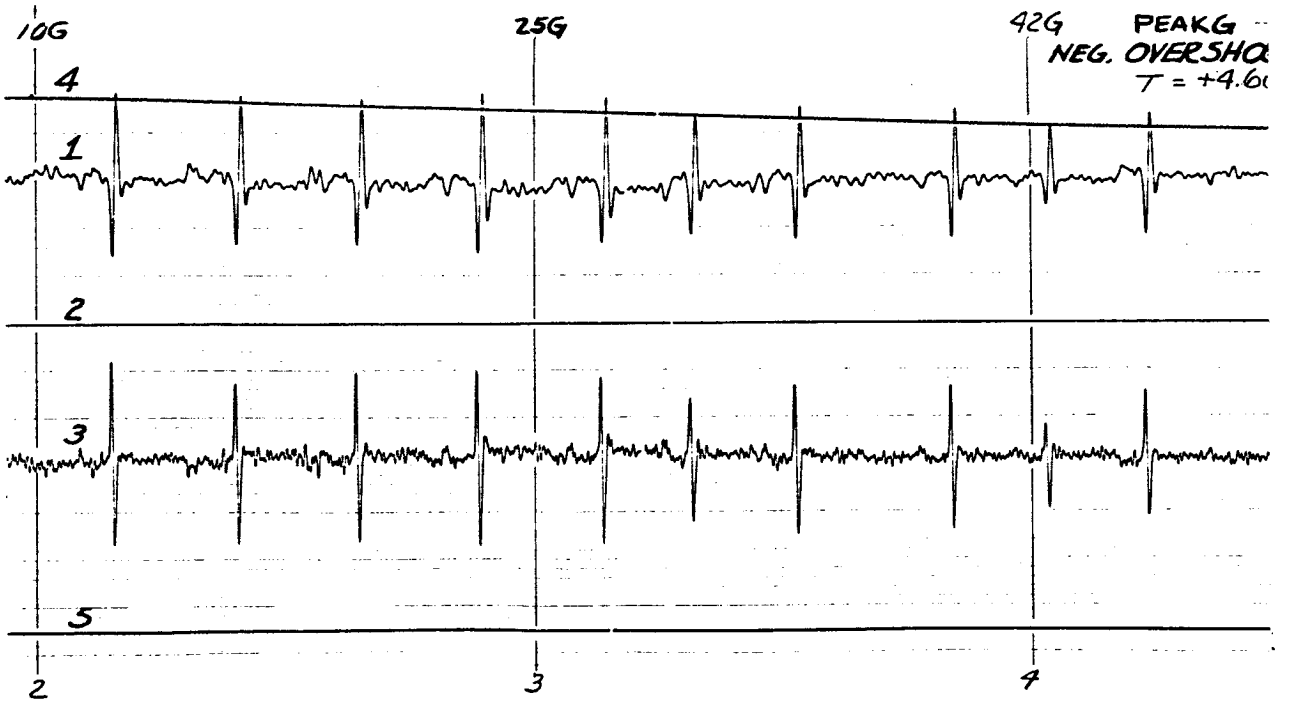


FIGURE 36. ANIMAL NO. 190. PHASE III - ONSET, 2-4 SECONDS

Figure 37. Animal No. 190 - Phase IV - Overshoot, 4-6 Seconds

Peak G is attained. Note wide swings of ECG trace. Note changes in voltage amplitude of QRS. Note ventricular extrasystole at elapsed time of 5 seconds at peak G. Trace No. 4 indicates that the machine has stabilized at 50 G at the 6 second time mark.

Figure 38. Animal No. 190 - Phase V - Initial Dwell, 5-7 Seconds

Note ventricular extrasystole. QRS displacement is at 25 units (50 mm) on the right.

Figure 39. Animal No. 190 - Phase VI - Dwell, 7-9 Seconds

Demonstrated here is the beginning of a 2.5 second period of electrocardiographic standstill. QRS displacement is 24 units (48 mm) in the complex just prior to this event.

Figure 40. Animal No. 190 - Phase VI - Dwell, 9-14 Seconds

Demonstrated here is the termination of the 2.5 second period of electrocardiographic standstill followed by a QRS complex typical of a premature ventricular contraction (P.V.C.) which is then followed by a second refractory period of 2.5 sec. Note change in paper speed from 100 through 50 to 25 mm/sec.

Figure 41. Animal No. 190 - Phase VI - Dwell, 11-19 Seconds

The refractory period to the left is followed by another P.V.C. which is then followed by a four second refractory period. Ventricular rate is extremely low.

Figure 42. Animal No. 190 - Phase VI - Dwell, 18-27 Seconds

Note the sharp shift in the QRS pattern. An abnormal Q wave is seen at the first arrow. There is also elevation of the ST segment with T wave inversion (second arrow), suggesting myocardial infarction and idioventricular pacemaker control. Rate is now 30/min.

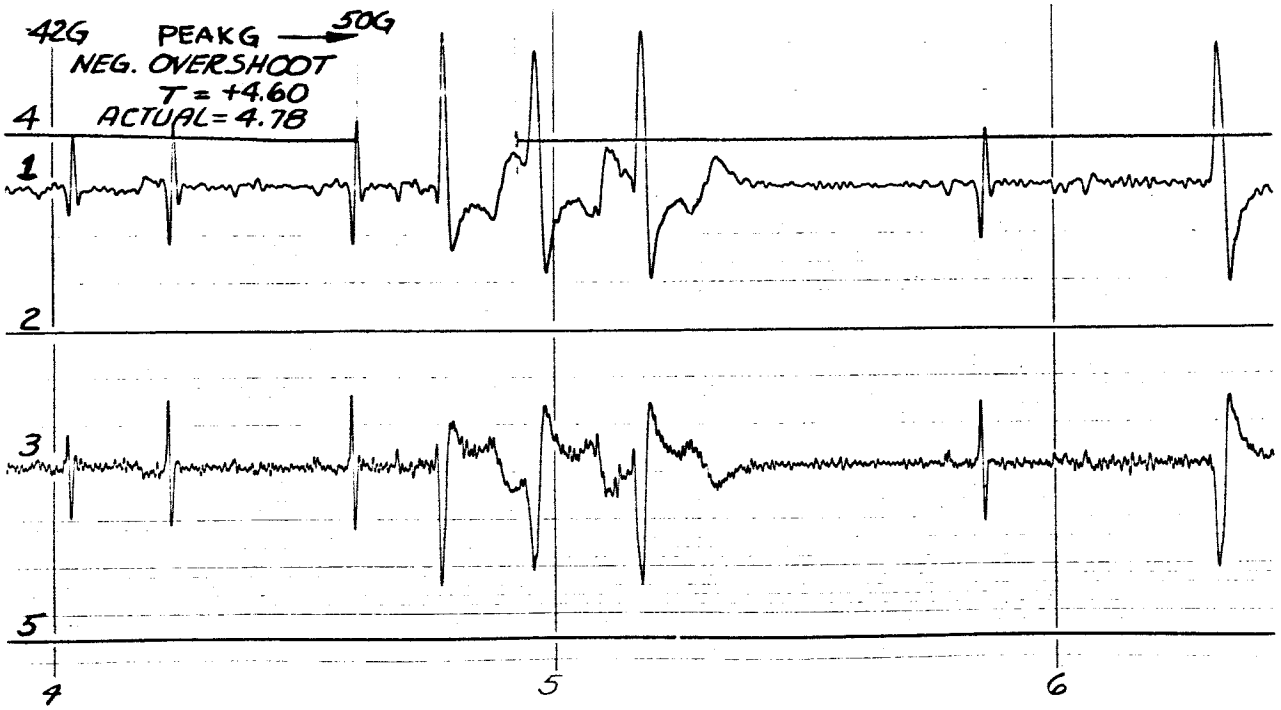


FIGURE 37. ANIMAL NO. 190. PHASE IV - OVERSHOOT, 4-6 SECONDS

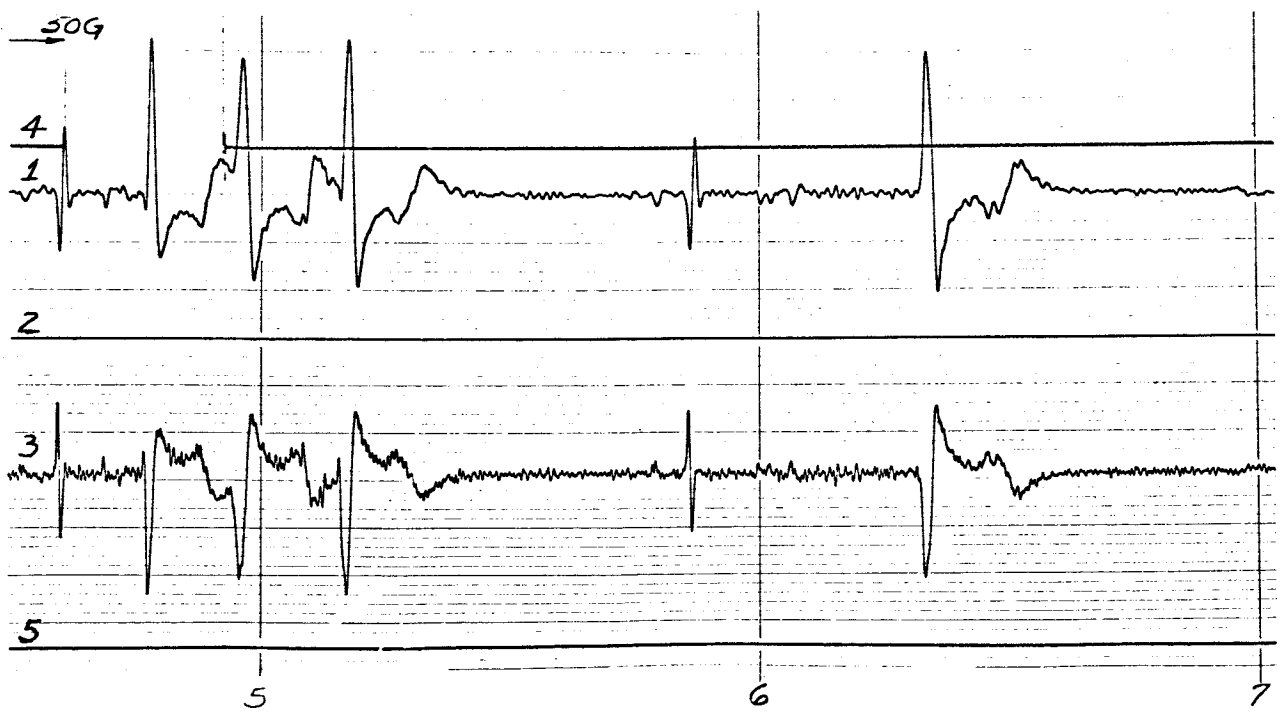


FIGURE 38. ANIMAL NO. 190. PHASE V - INITIAL DWELL, 5-7 SECONDS

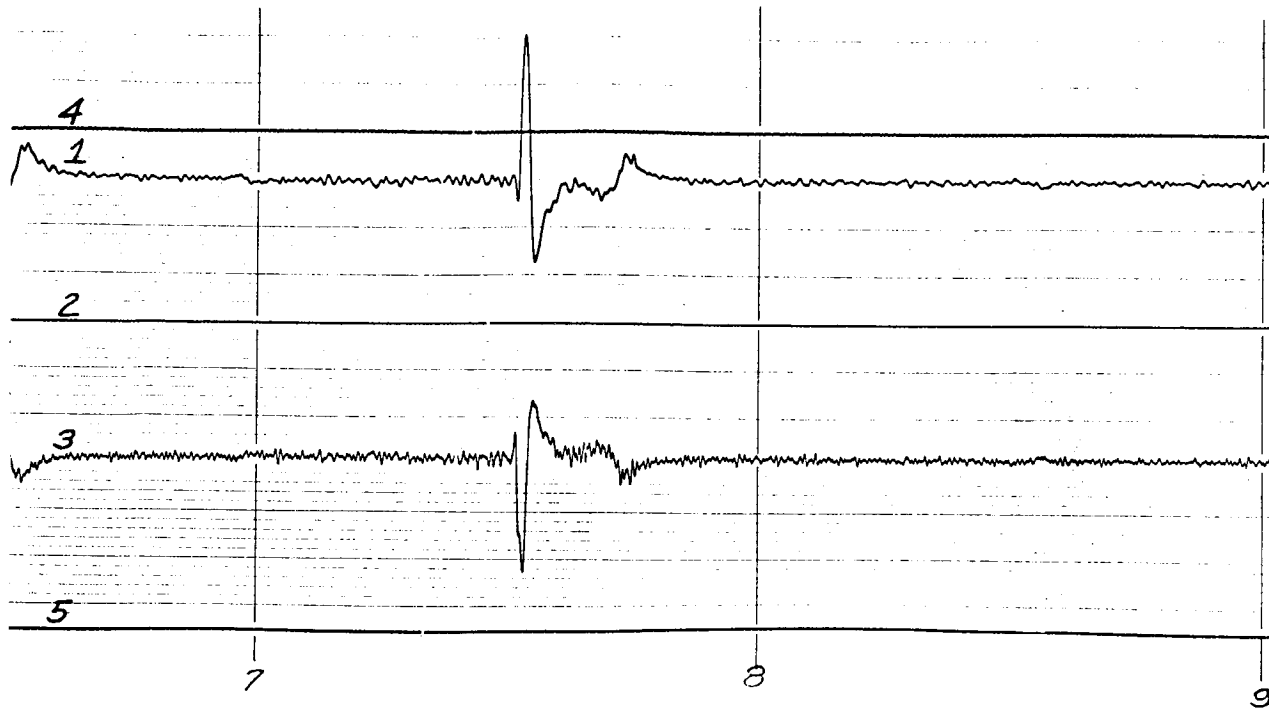


FIGURE 39. ANIMAL NO. 190. PHASE VI - DWELL, 7-9 SECONDS

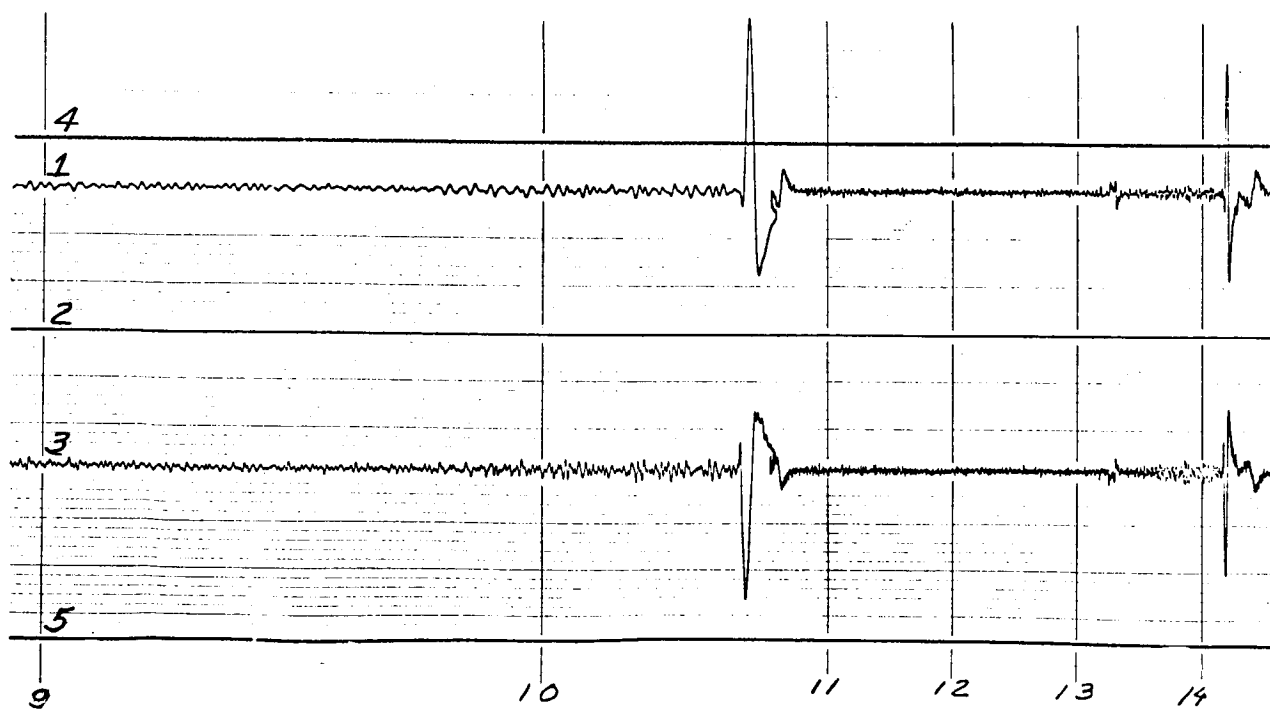


FIGURE 40. ANIMAL 190. PHASE VI - DWELL, 9-14 SECONDS

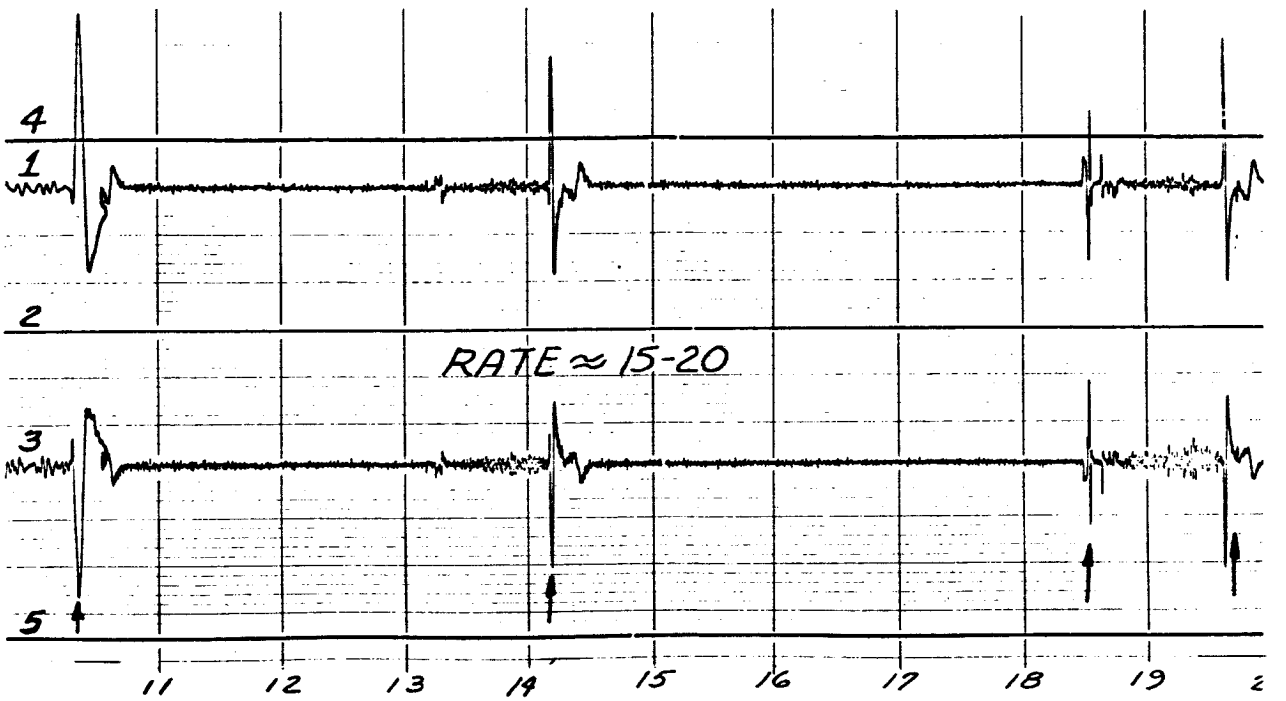


FIGURE 41. ANIMAL NO. 190. PHASE VI - DWELL, 11-19 SECONDS

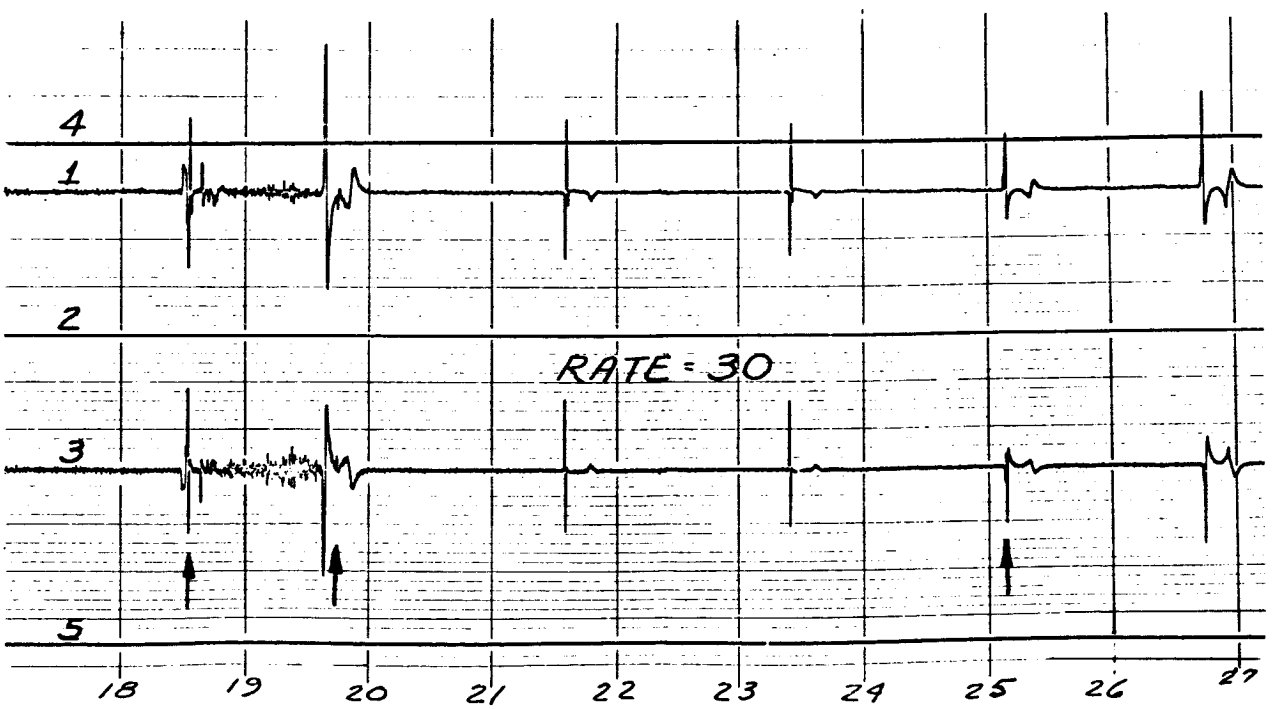


FIGURE 42. ANIMAL NO. 190. PHASE VI - DWELL, 18-27 SECONDS

Figure 43. Animal No. 190 - Phase VI - Dwell, 27-36 Seconds

Note abnormal Q wave, elevated ST segment and early T wave inversion suggesting myocardial infarction and idioventricular pacemaker. The heart rate increases to approximately 45/min. after tremor at 31 seconds.

Figure 44. Animal No. 190 - Phase VI - Dwell, 54-63 Seconds

Note the wandering of the sinus pacemaker. This is probably secondary to ischemia. On the right is demonstrated a deflection typical of a parasystole. (T=63) Rate now approximately 60/min.

Figure 45. Animal No. 190 - Phase VI - Dwell, 62-71 Seconds

The arrow on the left (at T=63) points to a complex typical of a parasystole. There are two pacemakers present, one atrial, the other idioventricular. Profound bradycardia is present. Approximate heart rate is 60/min. The pattern of ischemia injury is well developed. Note alternating QRS wave pattern, between 63-69 seconds.

Figure 46. Animal No. 190 - Phase VI - Dwell, 76-85 Seconds

Sinus pacemaker has regained control and rhythym is sinus. Heart rate has increased to 100/min. Note the slight rhythymic changes in QRS amplitude. Compared to baseline QRS, voltage is low. Note the diphasic T wave.

Figure 47. Animal No. 190 - Phase VI - Dwell, 110-120 Seconds

The arrows point to several premature ventricular contractions. The rate has increased to 150/min. Note slight changes in QRS voltage amplitude which is compatible with thoraric movement. T wave is diphasic. Also, note how T wave seems to be "marching back" to the QRS complex.

Figure 48. Animal No. 190 -
Phase VI - Dwell, K_t mark, 162-174 Seconds

Rhythym is sinus. QRS has low voltage, and T wave is diphasic. The marker for elapsed dwell time (D_t) to threshold is shown at T=166 or 161.5 seconds of dwell. A two second adjustment is made at 164-166 seconds to agree with actual elapsed time. Error is 1.7% within instrument error. Note lack of muscle noise. An extrasystole occurs at 169 seconds of run time. Heart rate is approximately 60/min.

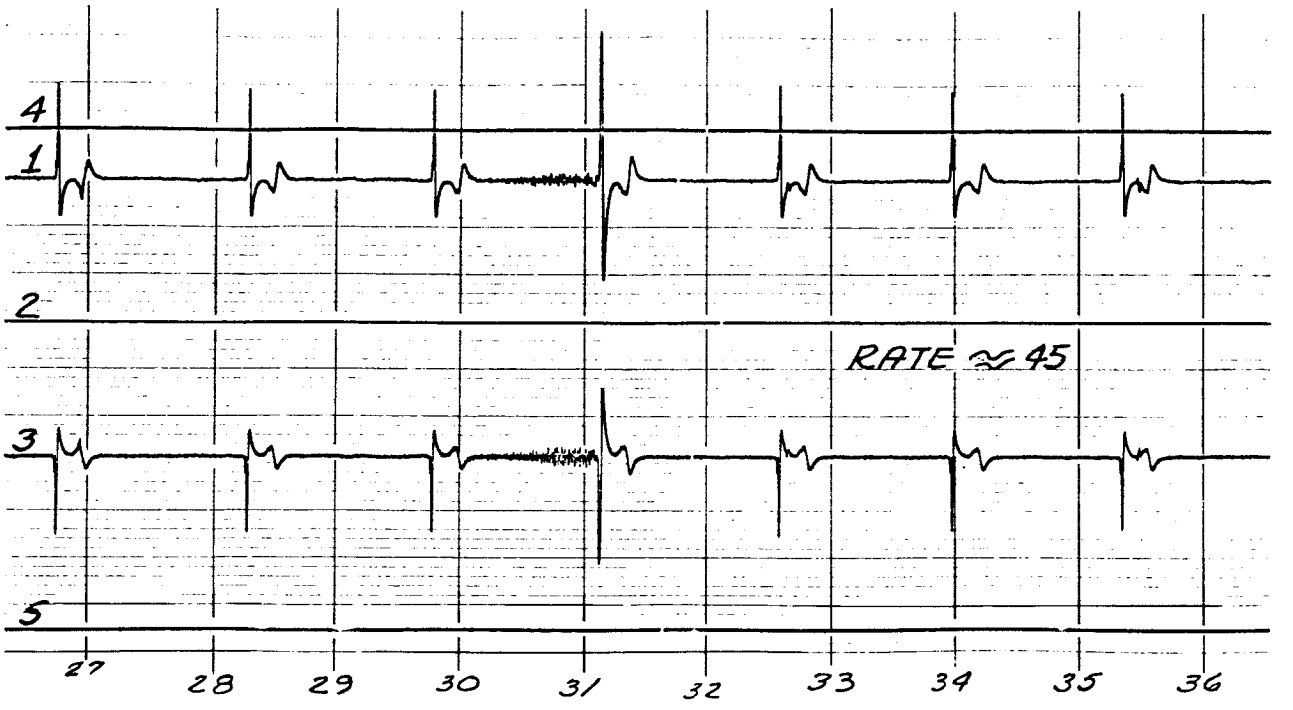


FIGURE 43. ANIMAL NO. 190. PHASE VI - DWELL, 27-36 SECONDS

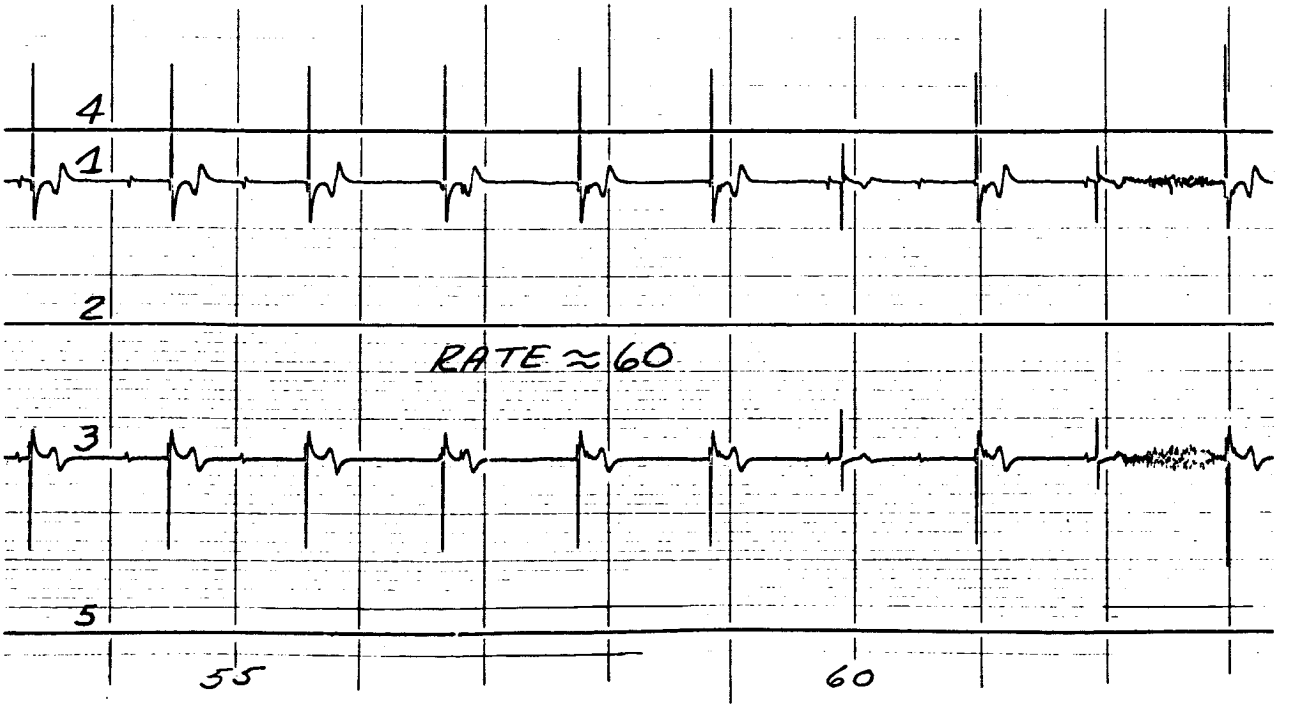


FIGURE 44. ANIMAL NO. 190. PHASE VI - DWELL, 54-63 SECONDS

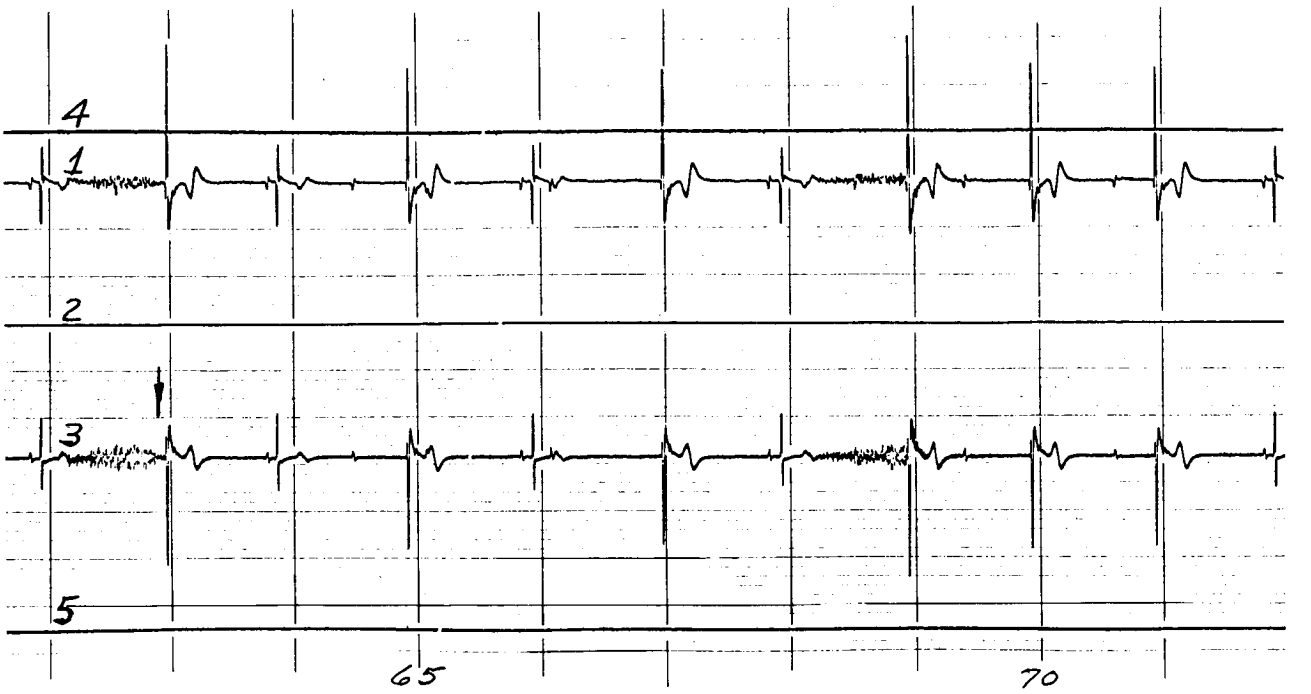


FIGURE 45. ANIMAL NO. 190. PHASE VI - DWELL, 62-71 SECONDS

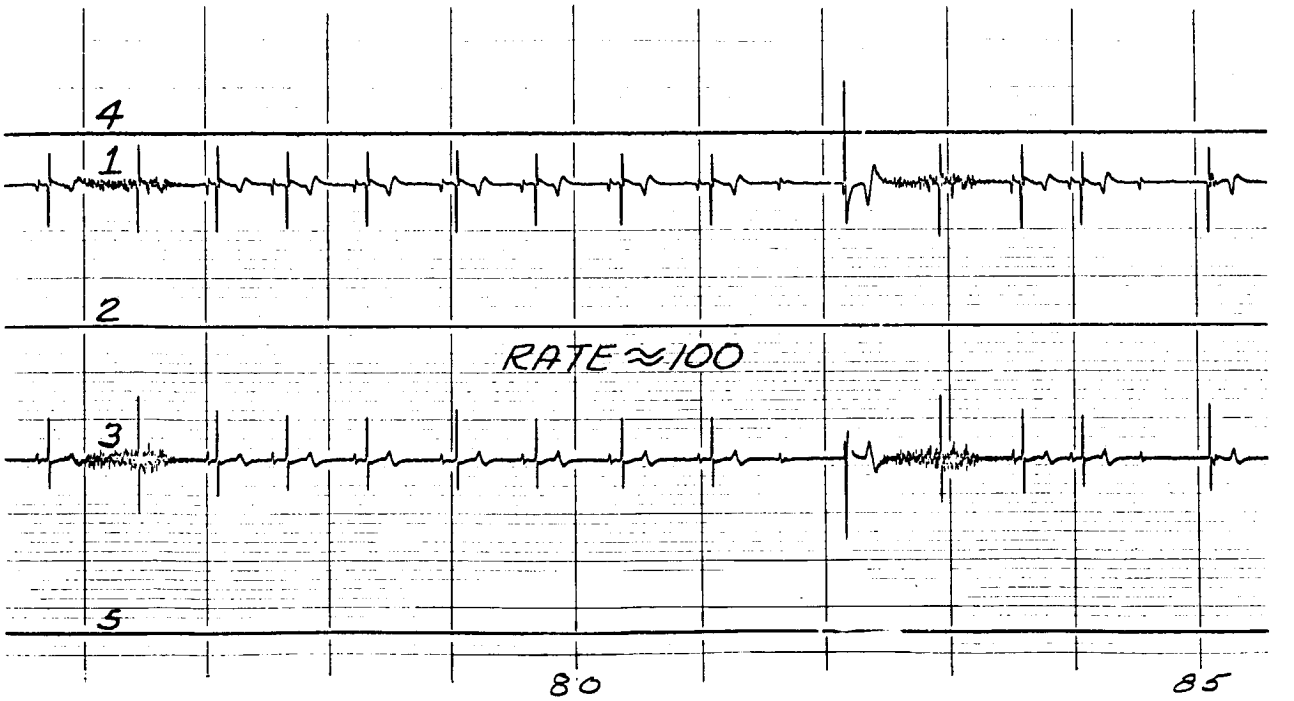


FIGURE 46. ANIMAL NO. 190. PHASE VI - DWELL, 76-85 SECONDS

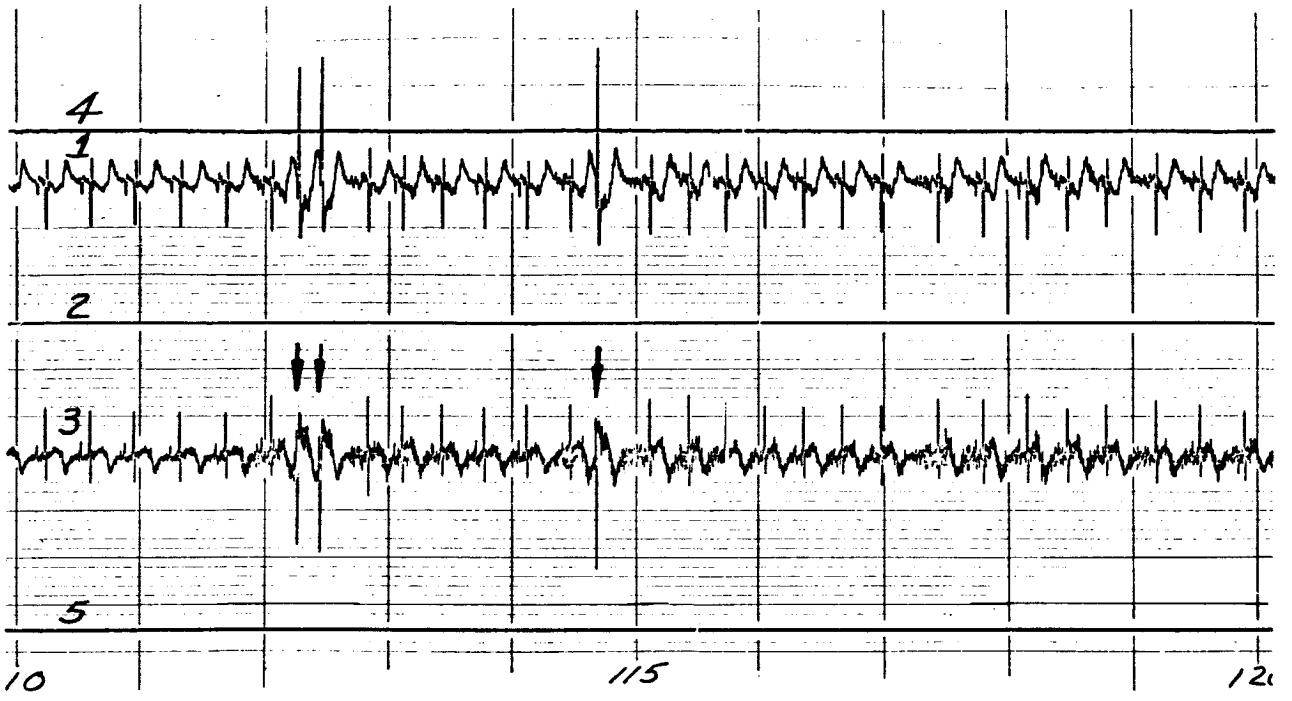


FIGURE 47. ANIMAL NO. 190. PHASE VI - DWELL, 110-120 SECONDS

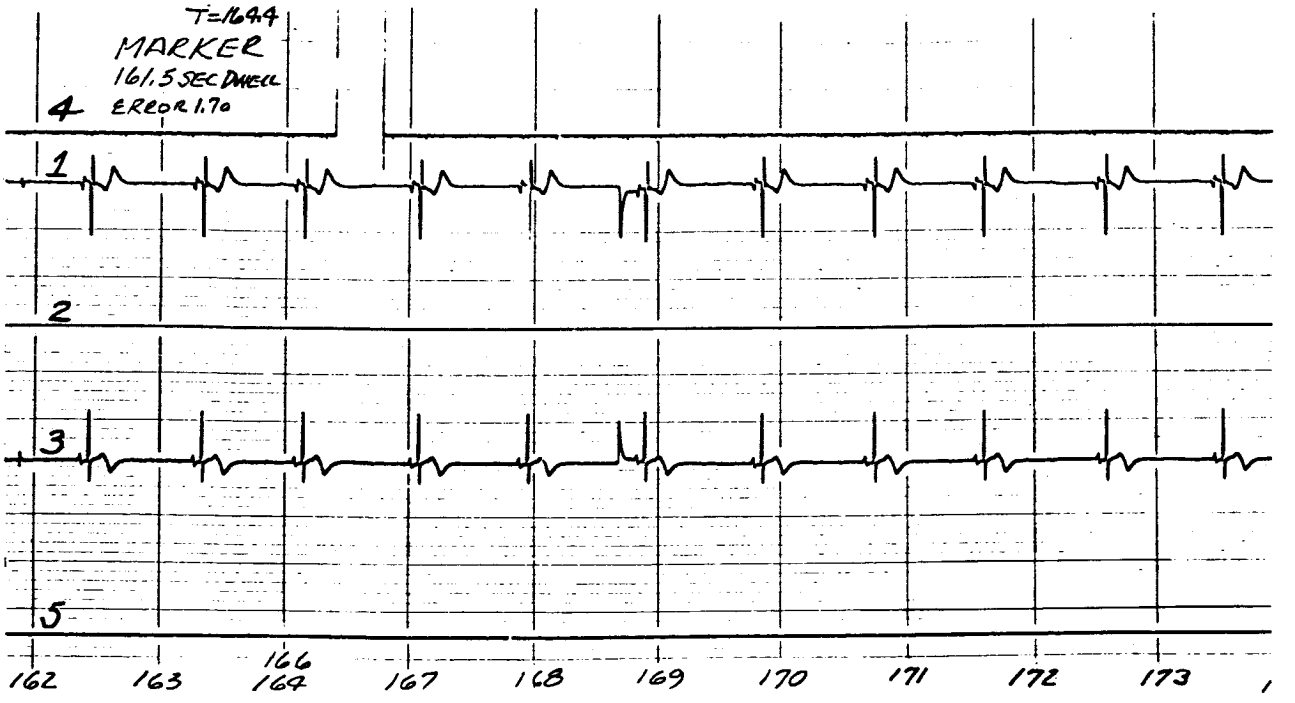


FIGURE 48. ANIMAL NO. 190.
PHASE VI - DWELL, K_t MARK, 162-174 SECONDS

Figure 49. Animal No. 190 - Phase VI - Dwell, 200-210 Seconds

Demonstrated here is a 2:1 A.V. block followed by a period of sinus arrest, which is then followed by additional 2:1 A.V. blocks. Heart rate remains at 60/min. QRS deflection is 7 units (14 mm). Also demonstrated is a P.V.C. on the right.

Figure 50. Animal No. 190 - Phase VI - Dwell, 226-235 Seconds

Demonstrated on the trace on the left is a temporary 3:1 A.V. block. Rate is 60/min. Also note how T wave appears to have "marched back" to the QRS complex.

Figure 51. Animal No. 190 - Phase VI - Dwell, 263-271 Seconds

Paper speed is 25 mm/sec. Note the profound bradycardia deepening from 60 to 30 min. A sinus arrhythmia is present at T=266-267.

Figure 52. Animal No. 190 - Phase VII - Pre-Offset, 282-292 Seconds

Heart rate has picked up to 60/min. again. Note marker on Trace No. 4 denoting the end of the dwell period, at T=291. QRS deflection is 7 units (14 mm). A 2:1 A.V. block is at T=283-284. There is a sinus arrhythmia present at T=290.

Figure 53. Animal No. 190 - Phase VIII-Offset, 289-298 Seconds

Note marker at T=291 denoting end of dwell. Also note deflection of Trace No. 4 towards baseline. Note the increased QRS deflection on the complex at the one G level at 296 seconds, followed by a 2:1 A.V. block.

Figure 54. Animal No. 190 - Phase IX -
Postrun 1st Minute, 297-206 Seconds

Intermittent 2:1 to 3:2 A.V. blocks are demonstrated here. The T wave is diphasic and has almost converged with the QRS complex. The ST segment is elevated.

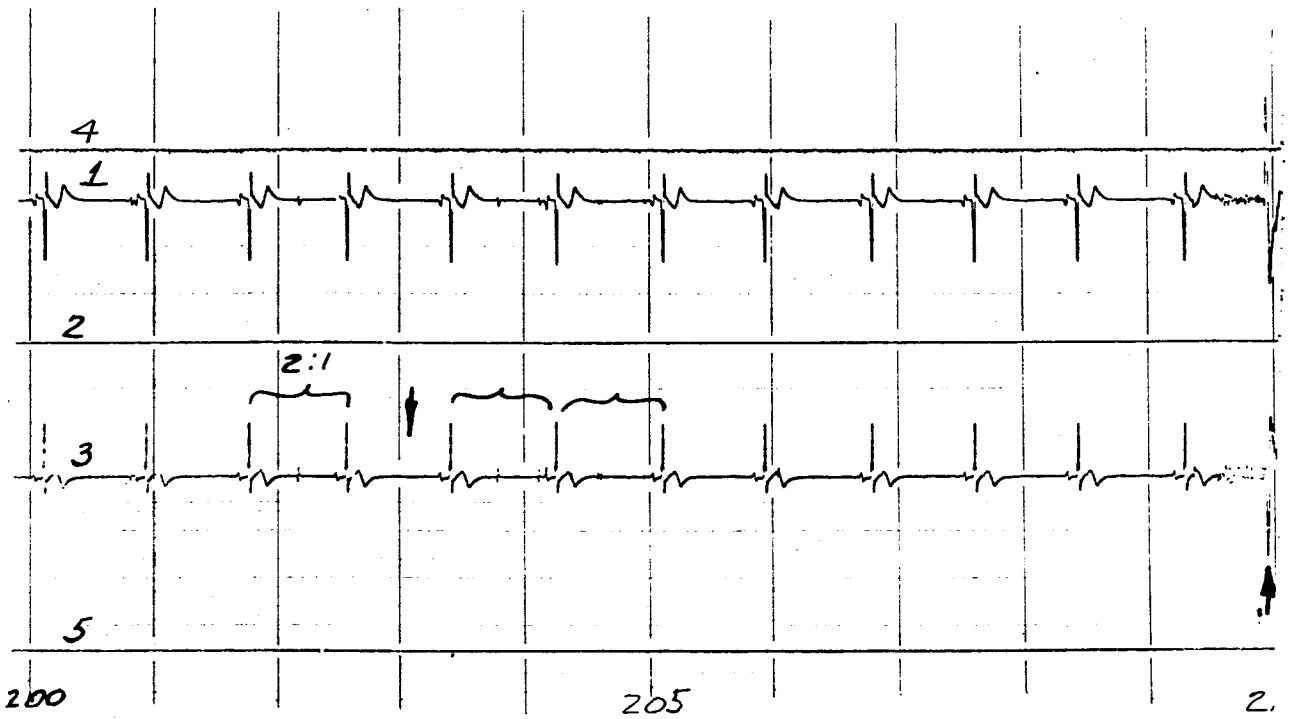


FIGURE 49. ANIMAL NO. 190. PHASE VI - DWELL, 200-210 SECONDS

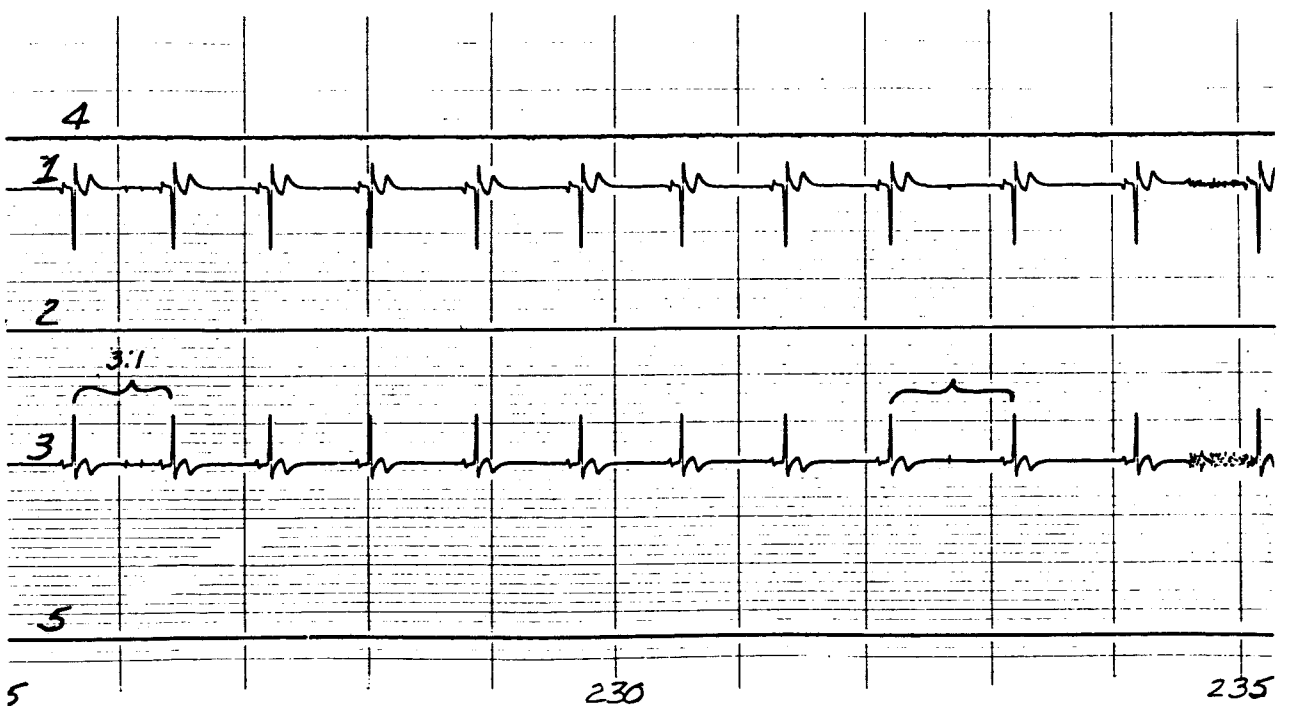


FIGURE 50. ANIMAL NO. 190. PHASE VI - DWELL, 226-235 SECONDS

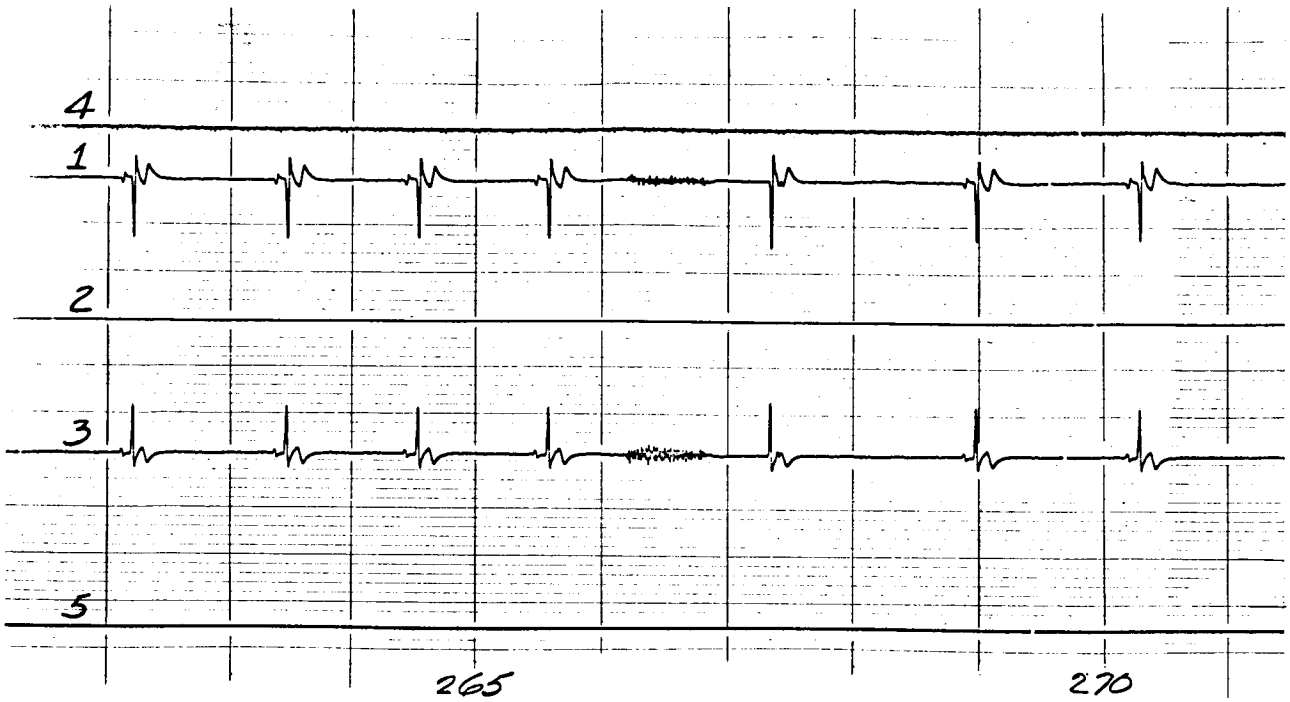


FIGURE 51. ANIMAL NO. 190. PHASE VI - DWELL, 263-271 SECONDS

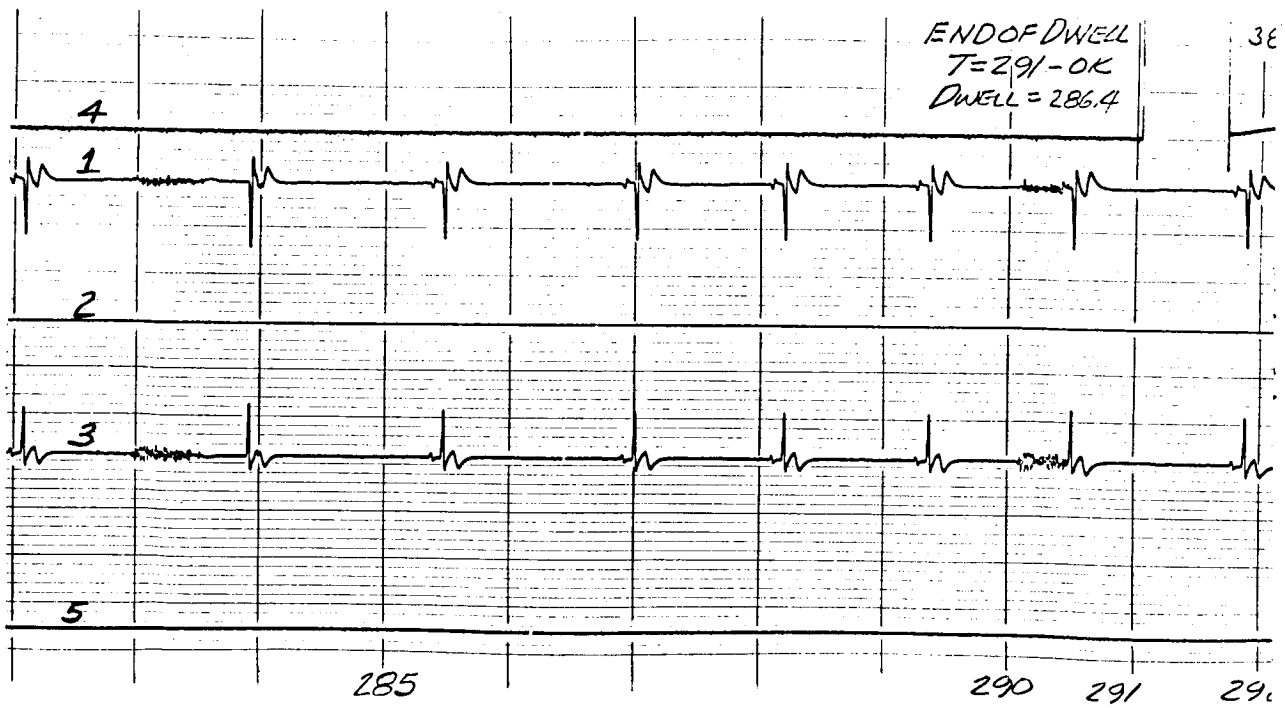


FIGURE 52. ANIMAL NO. 190.
PHASE VII - PRE-OFFSET, 282-292 SECONDS

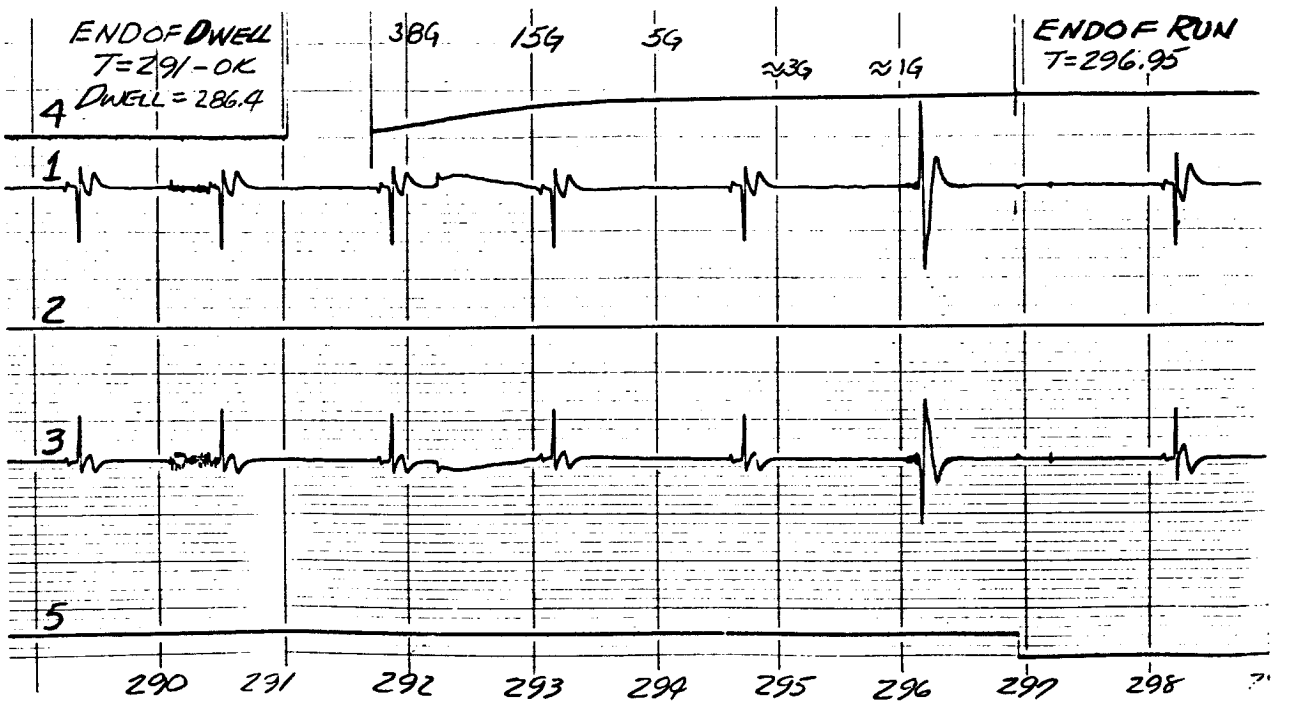


FIGURE 53. ANIMAL NO. 190. PHASE VIII - OFFSET, 289-298 SECONDS

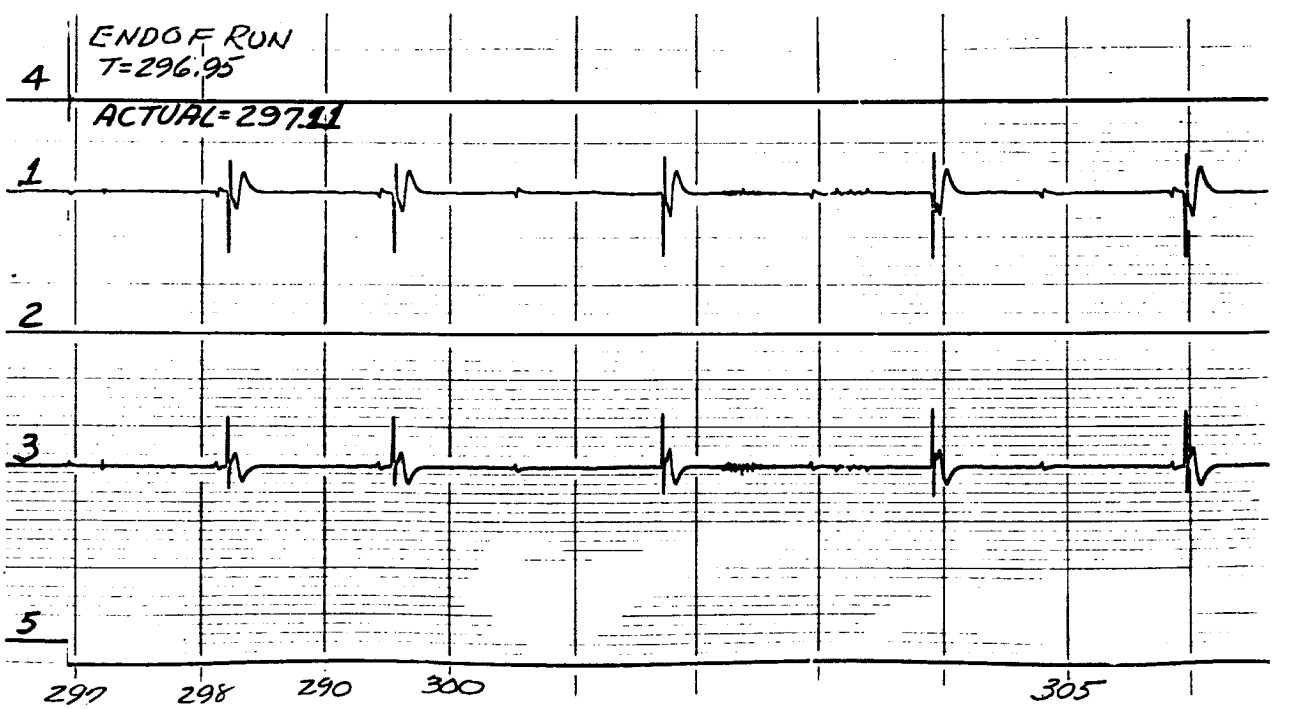


FIGURE 54. ANIMAL NO. 190.
PHASE IX - POSTRUN 1ST MINUTE, 297-306 SECONDS

Figure 55. Animal No. 190 -
Phase IX, Postrun 1st Minute, 315-324 Seconds

Demonstrated here are changing A.V. blocks, elevated ST segment, diphasic T wave, and on the right a P.V.C.

Figure 56. Animal No. 190 -
Phase X, Postrun 2nd Minute, 364-373 Seconds

Paper speed here is 25 mm/sec. This trace demonstrates complete A.V. dissociation. The QRS is from an idioventricular pacemaker, and shows increased amplitude of QRS deflection. Note complete merger of the T wave with the QRS complex. Note widened QRS duration.

Figure 57. Animal No. 190 -
Phase XI, Postrun 3rd to 5th Minute, 431-433 Seconds

Paper speed here is 100 mm/sec. Note the extreme width of the QRS complex as compared to baseline (Figure 33). Rate is 60/min. Also note complete merger of QRS and T components into one sweeping wave form.

Figure 58. Animal No. 190 -
Phase XIII, Postrun 5th Minute to End, 595-604 Seconds

Heart rate is 60/min. Complete A.V. dissociation with auricular rate of 120/min. and ventricular rate of 60/min. The QRS is from an idioventricular pacemaker. This animal was observed to be breathing immediately postrun at a respiratory rate of one/min. Respirations ceased and the animal finally died. It never showed ECG evidence of recovery.

SECTION THREE. ANIMAL NO. 239, SEX FEMALE

This animal was exposed to +430 G_x for a total dwell of 115.6 seconds. The animal's weight was 511.6 grams. Its calculated D_t was 45.6 seconds and D_r was 115.6 seconds. The K_p reached 2.4×10^4. The animal was a survivor.

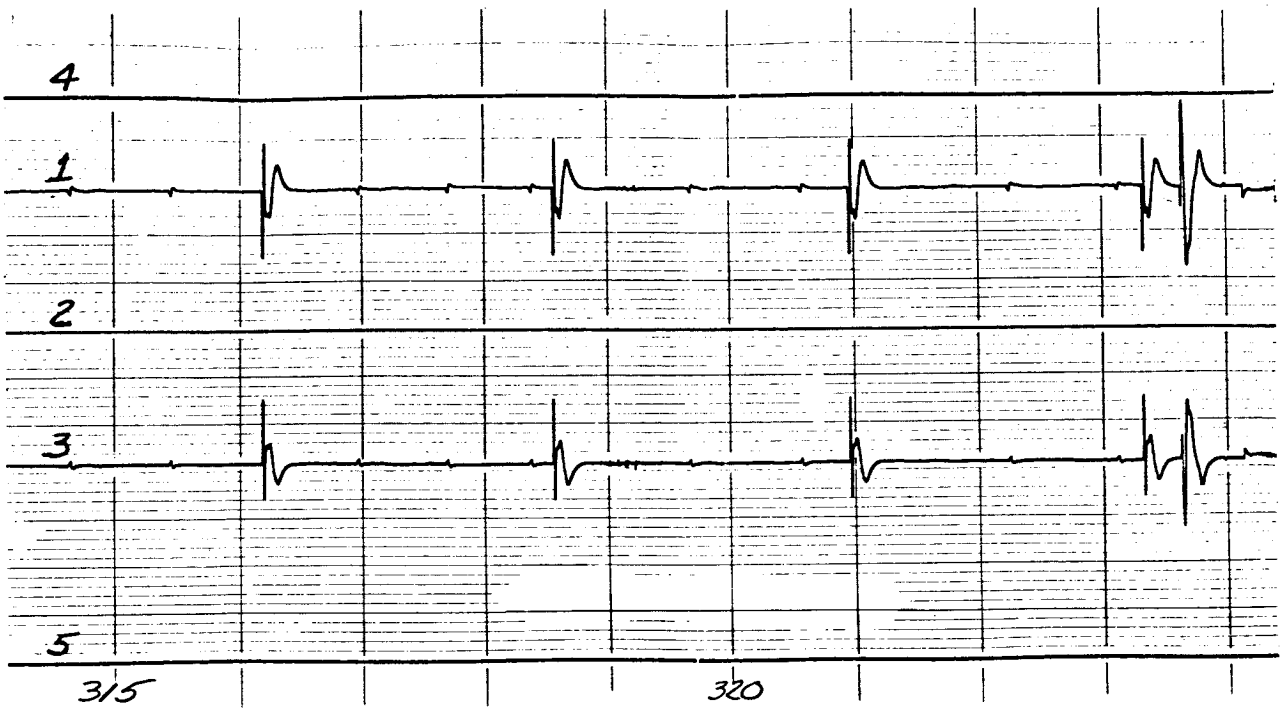


FIGURE 55. ANIMAL NO. 190.
PHASE IX - POSTRUN 1ST MINUTE, 315-324 SECONDS

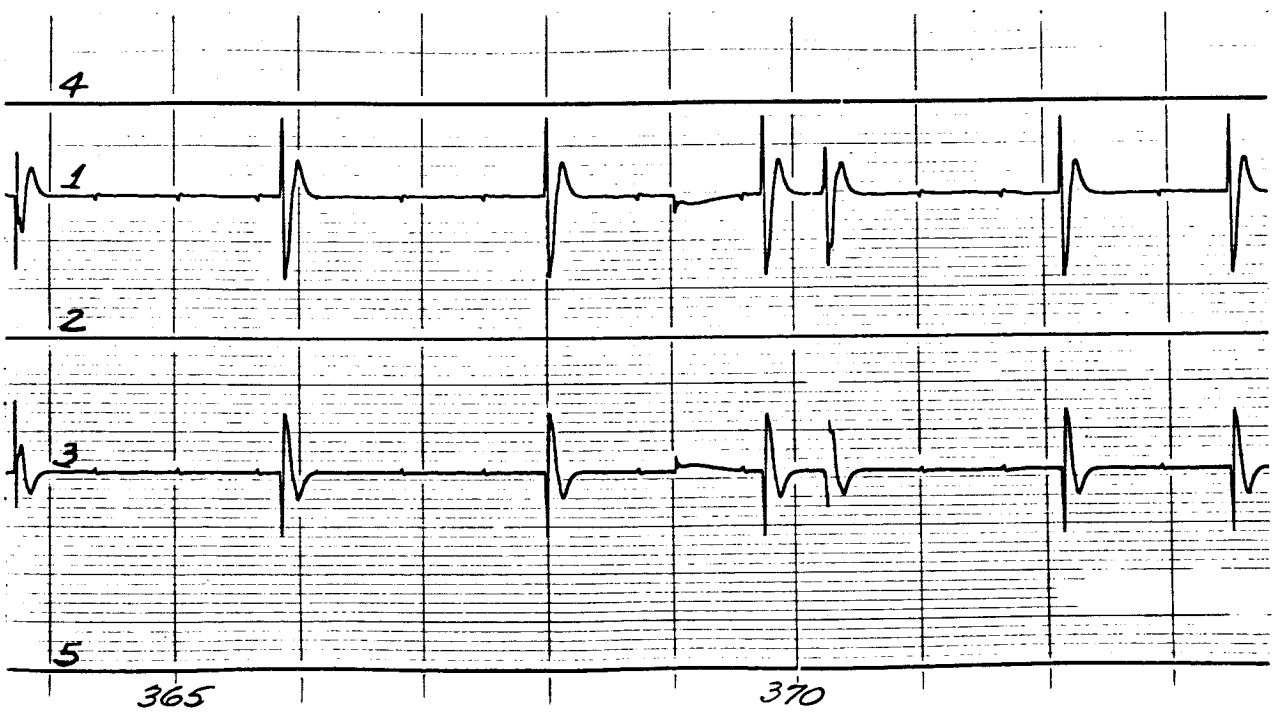


FIGURE 56. ANIMAL NO. 190.
PHASE X - POSTRUN 2ND MINUTE, 364-373 SECONDS

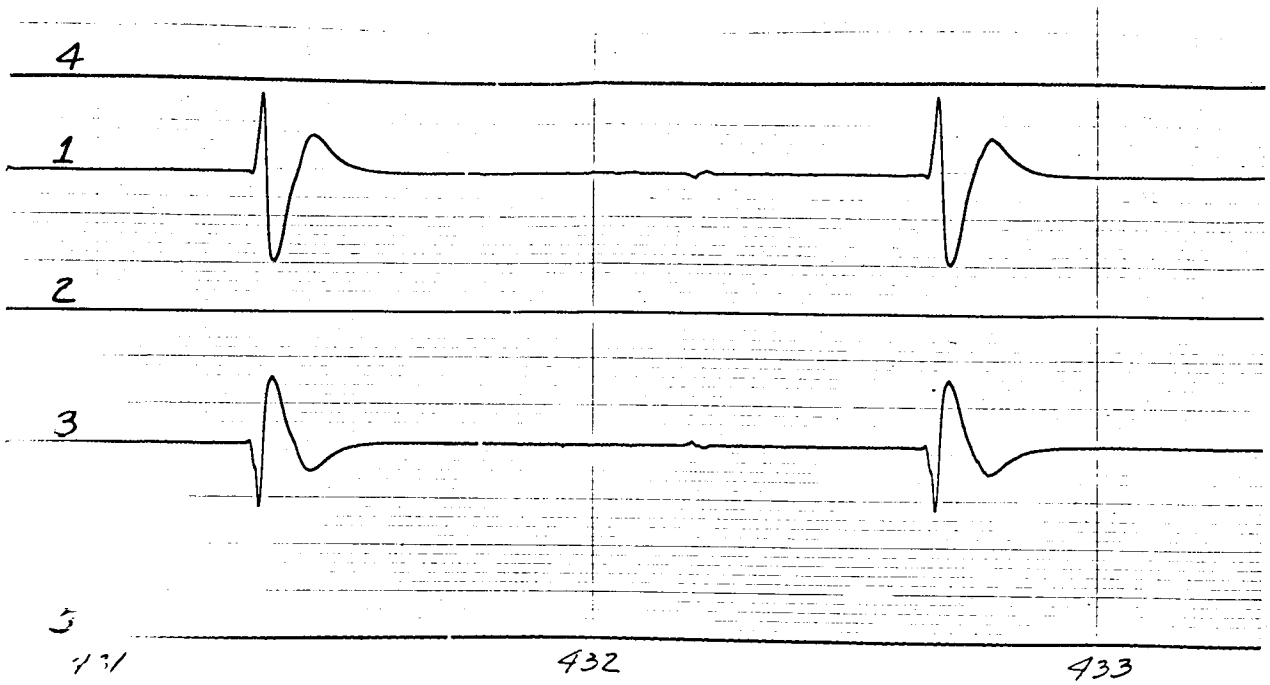


FIGURE 57. ANIMAL NO. 190.
PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 431-433 SECONDS

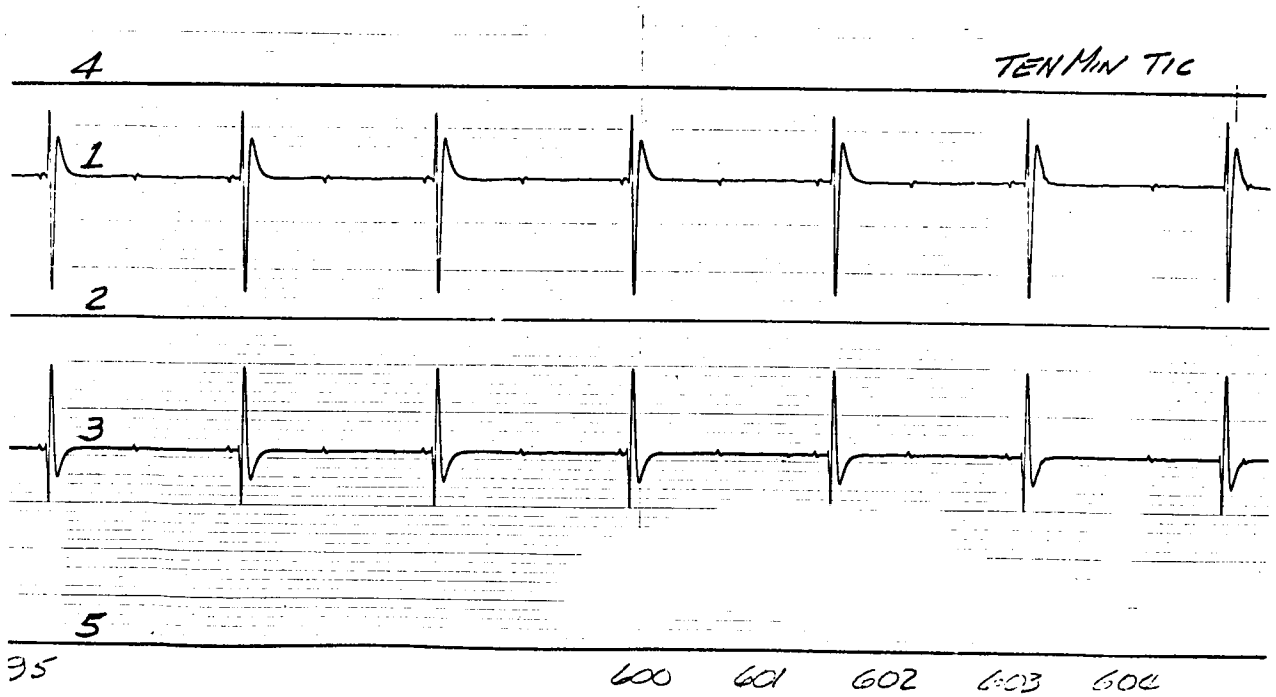


FIGURE 58. ANIMAL NO. 190.
PHASE XII - POSTRUN 5TH MINUTE TO END, 595-604 SECONDS

Figure 59. Animal No. 239 - Phase I, Baseline

This figure shows a QRS complex varying between 14 and 16 units, which is compatible with respiratory movements of the thorax. The respiratory rate here appears to be approximately 90/min. The QU duration is 13 mm. R wave is 10-12 units (20-24 mm) and the P wave is 2 units (4 mm) in amplitude and 2 mm in duration.

Figure 60. Animal No. 239 - Phase II, Pre-run Baseline

No change from the above figure. The clearing effect of the 50 cps. filter in Trace No. 1 is apparent.

Figure 61. Animal No. 239 - Phase III, Onset, 0-2 Seconds

Muscle noise appears on both traces almost instantly after start of run. The dimensions of the QRS vary widely, reducing to 10½ units (21 mm) at the extreme right. At 10 G (T=2 seconds) the shift in QRS pattern from V-3 in type to V-2 is abrupt and very noticable.

Figure 62. Animal No. 239 - Phase III, Onset, 2-4 Seconds

The left hand arrow indicates the point at which the wave form shifts. The right hand arrow indicates the beginning of a wide shift of the thorax. The 3-4 sec. segment shows what might be a slight tachycardia. See Trace No. 1. The P and T waves are difficult to identify.

Figure 63. Animal No. 239 - Phase III, Onset, 4-6 Seconds

The thoracic movement is in full evidence here. The tachycardia appears to be persisting at approximately 360 beats/min. Further details are obscured. Trace No. 4 is seen rising towards peak G. At the arrow (T=5 sec.) the QRS pattern seems to steady with a pattern approaching V-4. Note the 50% diminution in voltage, the QRS complex has a height of 6 units (12 mm). Approximate G levels are noted at each time line.

Figure 64. Animal No. 239 - Phase III, Onset, 6-8 Seconds

The heart rate is slightly more than 300 beats/min. between 6-8 sec. The QRS trace gradually increases to a 10 unit (20 mm) average. Trace No. 4 continues to rise.

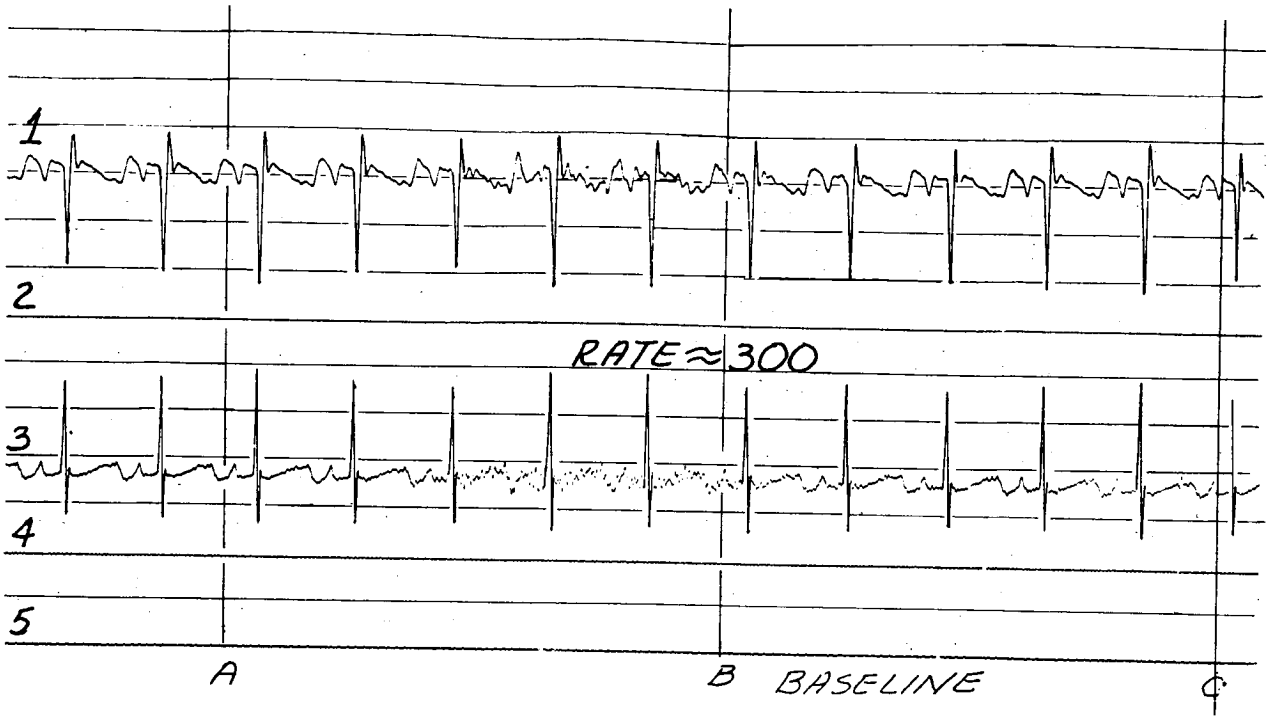


FIGURE 59. ANIMAL NO. 239. PHASE I - BASELINE

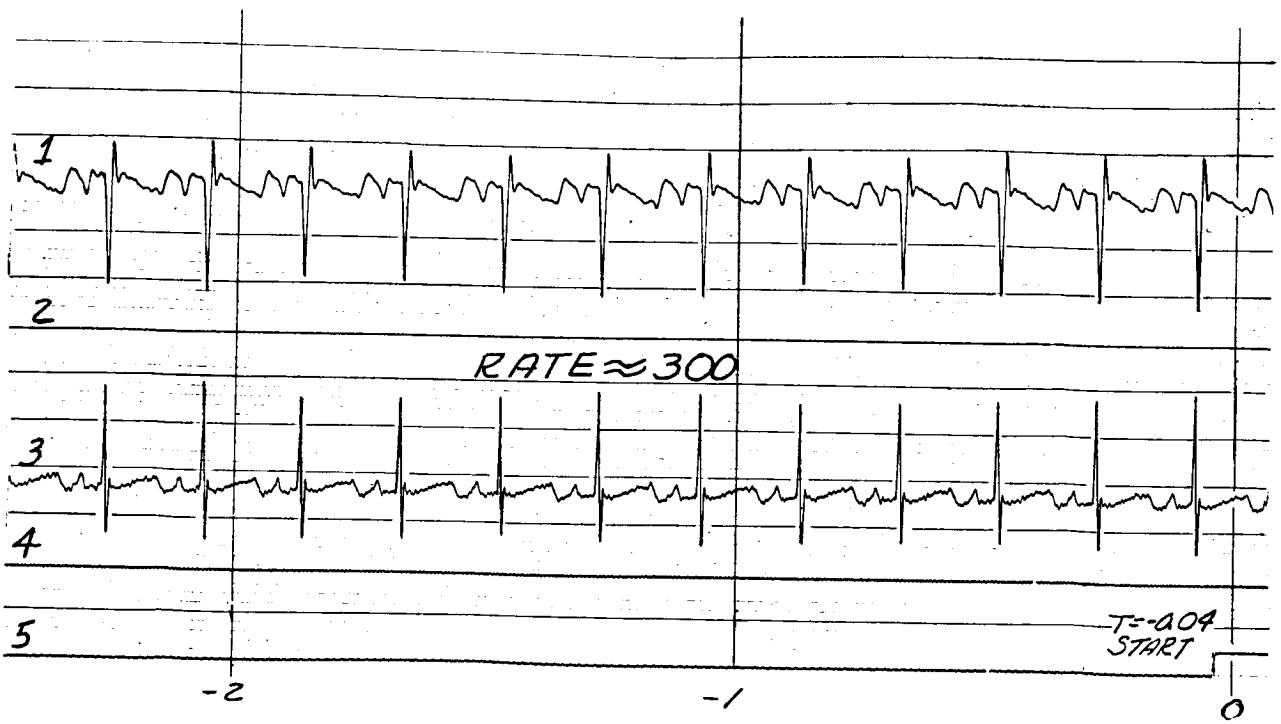


FIGURE 60. ANIMAL NO. 239. PHASE II - PRE-RUN BASELINE

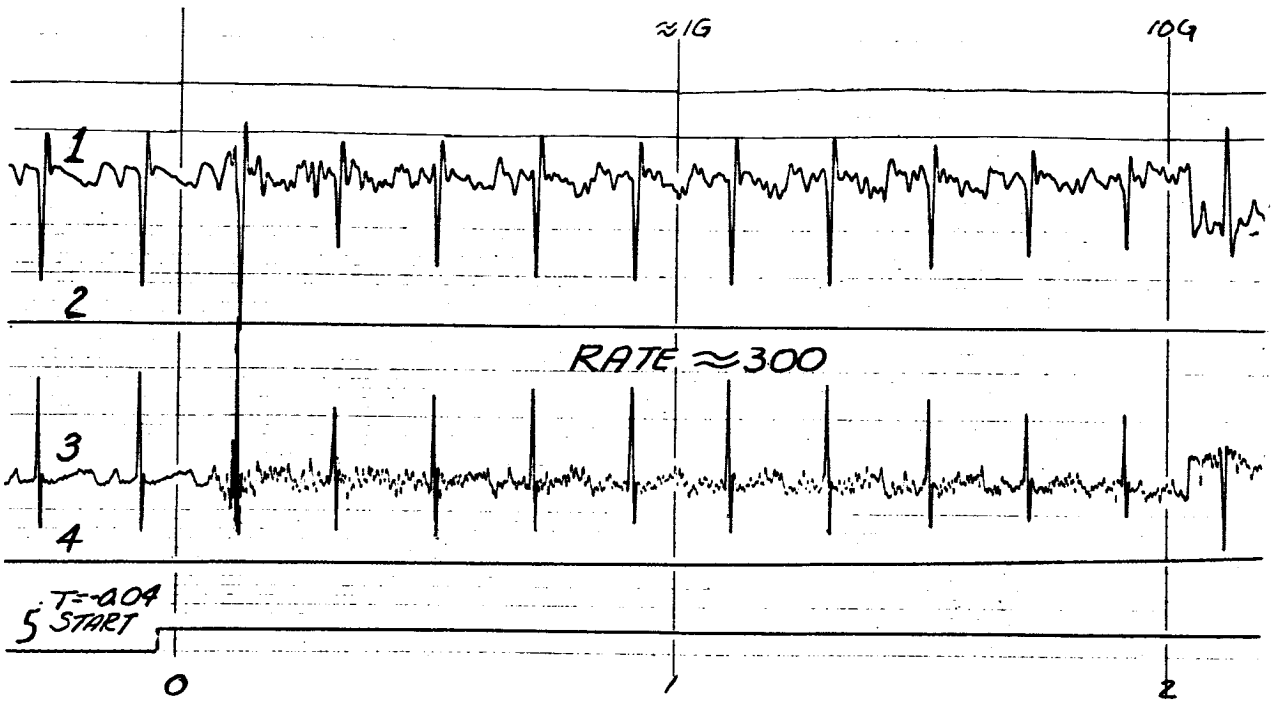


FIGURE 61. ANIMAL NO. 239. PHASE III - ONSET, 0-2 SECONDS

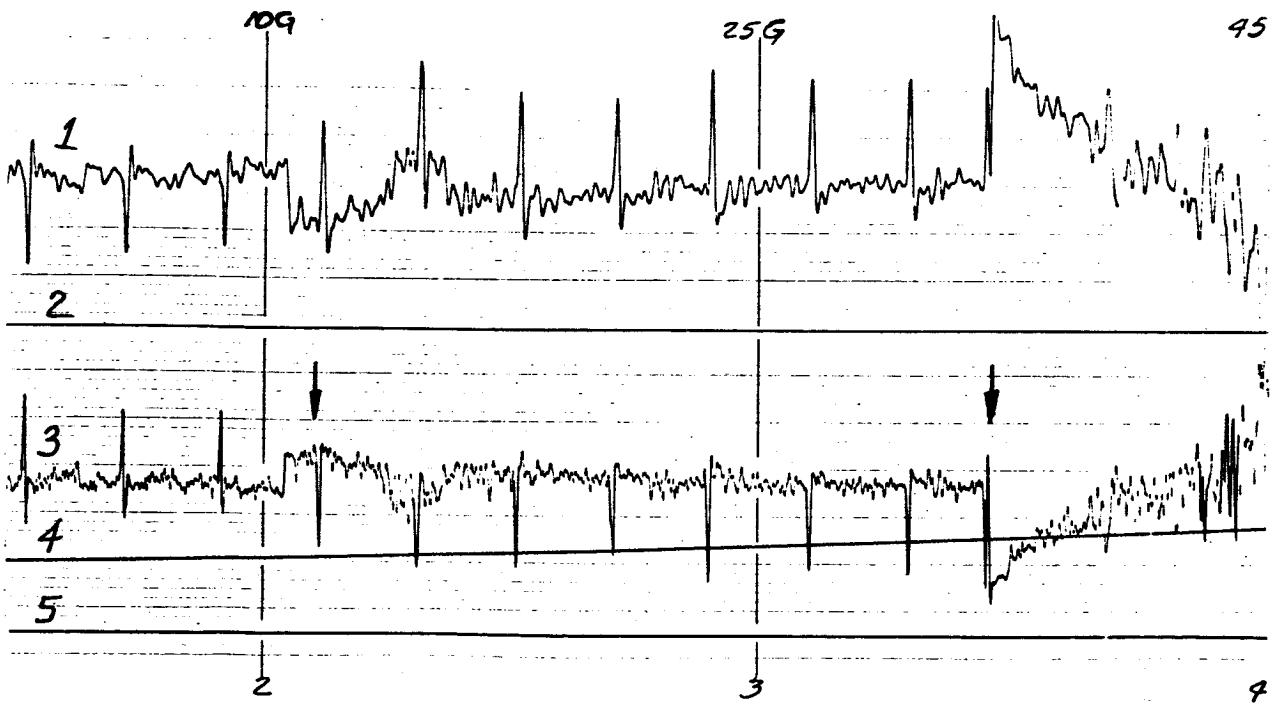


FIGURE 62. ANIMAL NO. 239. PHASE III - ONSET, 2-4 SECONDS

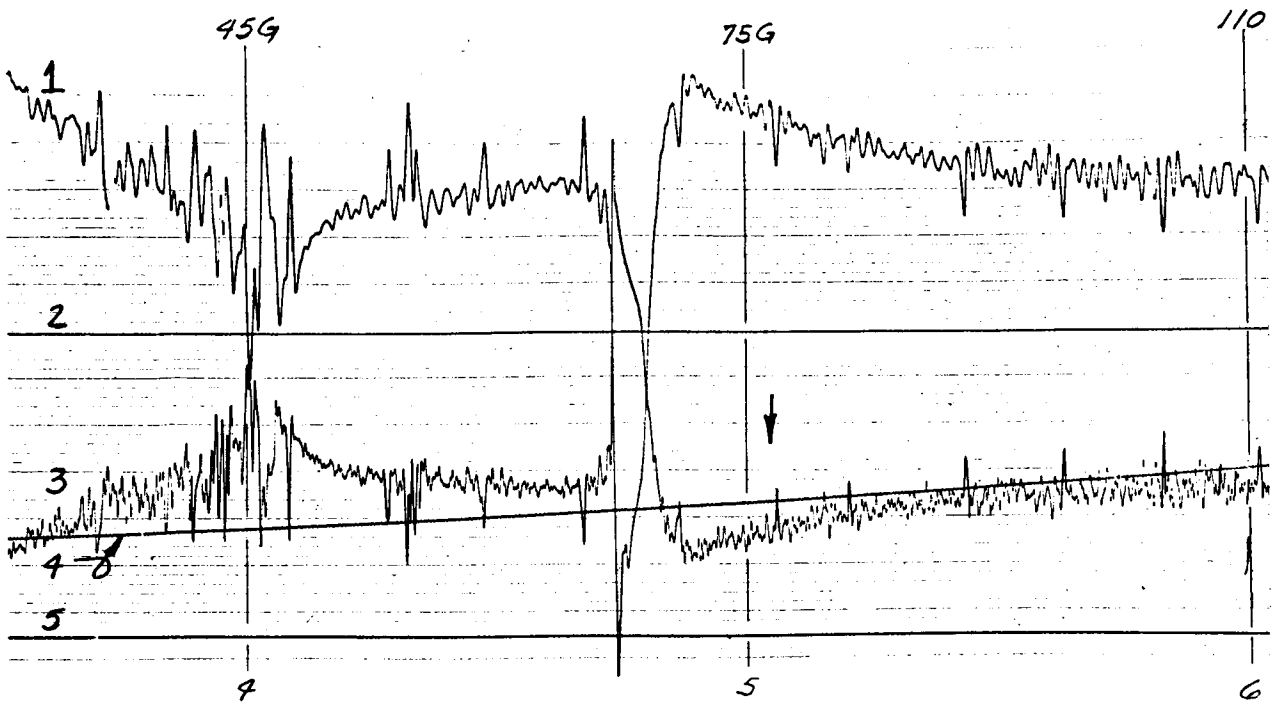


FIGURE 63. ANIMAL NO. 239. PHASE III - ONSET, 4-6 SECONDS

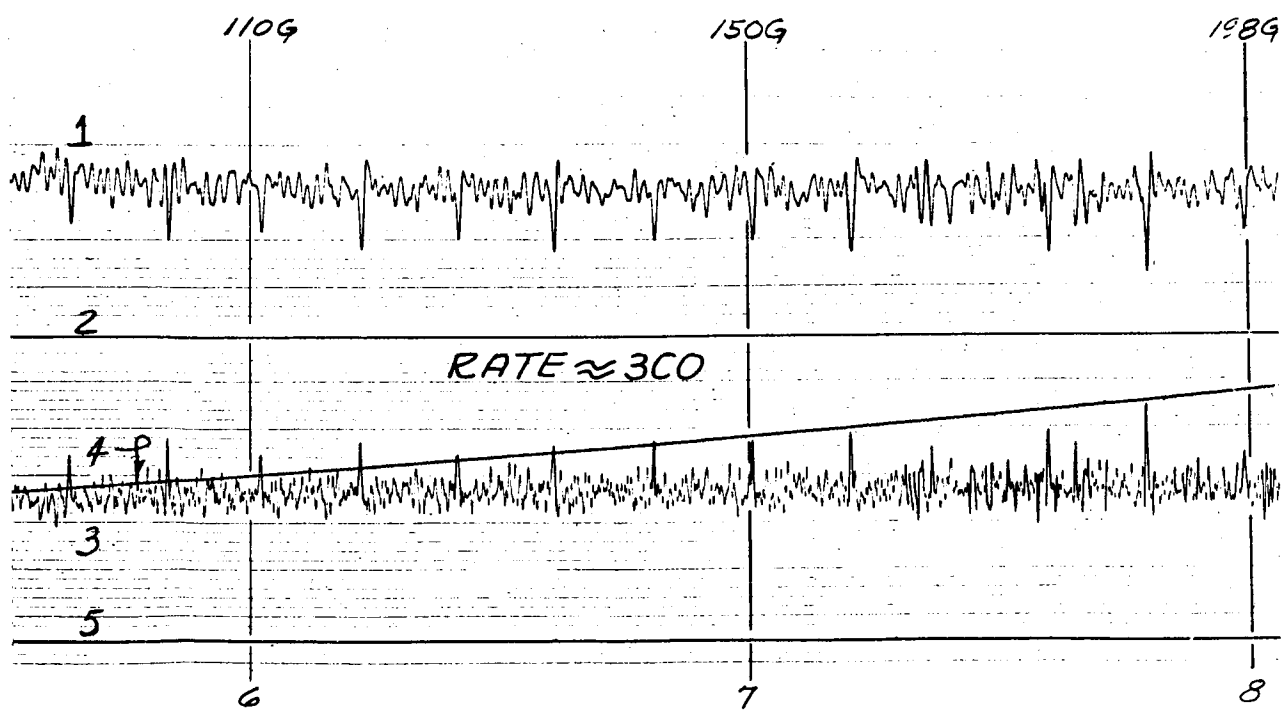


FIGURE 64. ANIMAL NO. 239. PHASE III - ONSET, 6-8 SECONDS

Figure 65. Animal No. 239 - Phase III, End of Onset, 11-13 Seconds

Note extreme muscle noise obscuring ECG pattern. Target G of 430 is attained at an elapsed time of 12.25 sec. Trace No. 4 indicates peak of overshoot at 460 G at 13 sec. elapsed time. There is a suggestion of tachycardia in Trace No. 1. A shifting QRS pattern is also suggested.

Figure 66. Animal No. 239 - Phase IV, Overshoot, 13-15 Seconds

Trace No. 4 achieves a peak of 460 G (approximately 107% of target) - tachycardia may be present. Overshoot lasts for approximately 3 seconds. Target G level re-attained at 15+ sec. elapsed time. Note alteration in height of complexes, between 14 and 15 seconds.

Figure 67. Animal No. 239 - Phase VI, Dwell - K_t Mark, 57-67 Seconds

The marker at the left of the trace indicates the dwell time to threshold (45.6 seconds), and it is achieved at the total elapsed time of 58.29 seconds. From the start of run, the gradual diminution of muscle noise once threshold is attained is noteworthy. Average QRS amplitude is 11 units. Heart rate has slowed from the baseline rate of 300. The average rate here is 150. The periodicity in the height of the QRS complex is compatible with respiratory movement. The paper speed has been reduced to 25 mm/sec.

Figure 68. Animal No. 239 - Phase VI, Dwell, 69-75 Seconds

The animal has quieted almost completely. Arrows point to sites of extrasystole. The QRS amplitude averages 7 units. A shift in wave forms at the extreme right, (T=75) is shown.

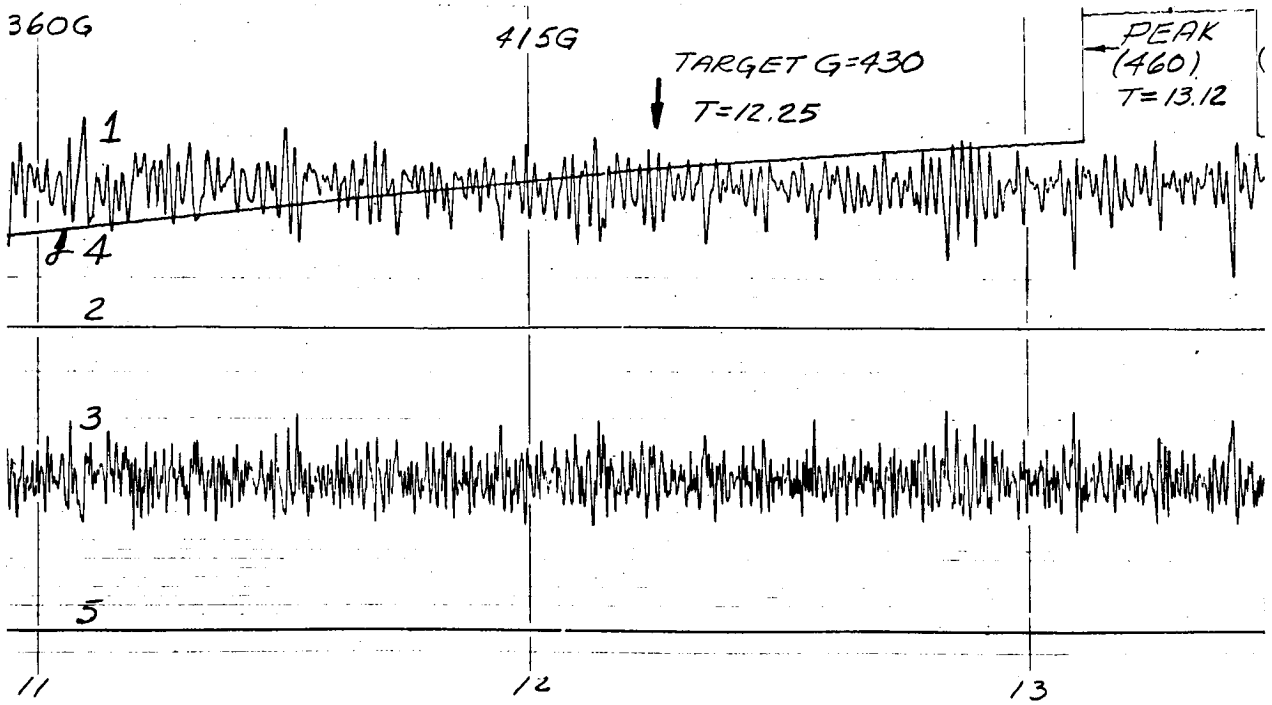


FIGURE 65. ANIMAL NO. 239.
PHASE III, END OF ONSET, 11-13 SECONDS

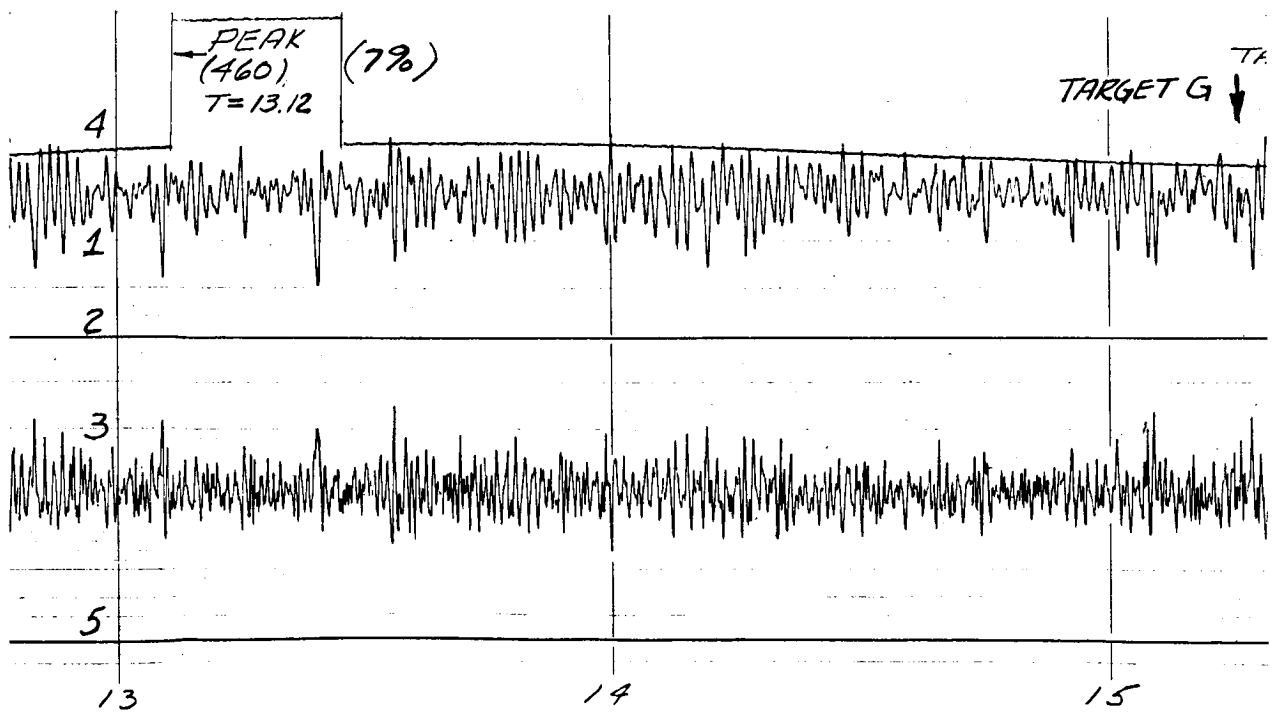


FIGURE 66. ANIMAL NO. 239. PHASE IV, OVERSHOOT, 13-15 SECONDS

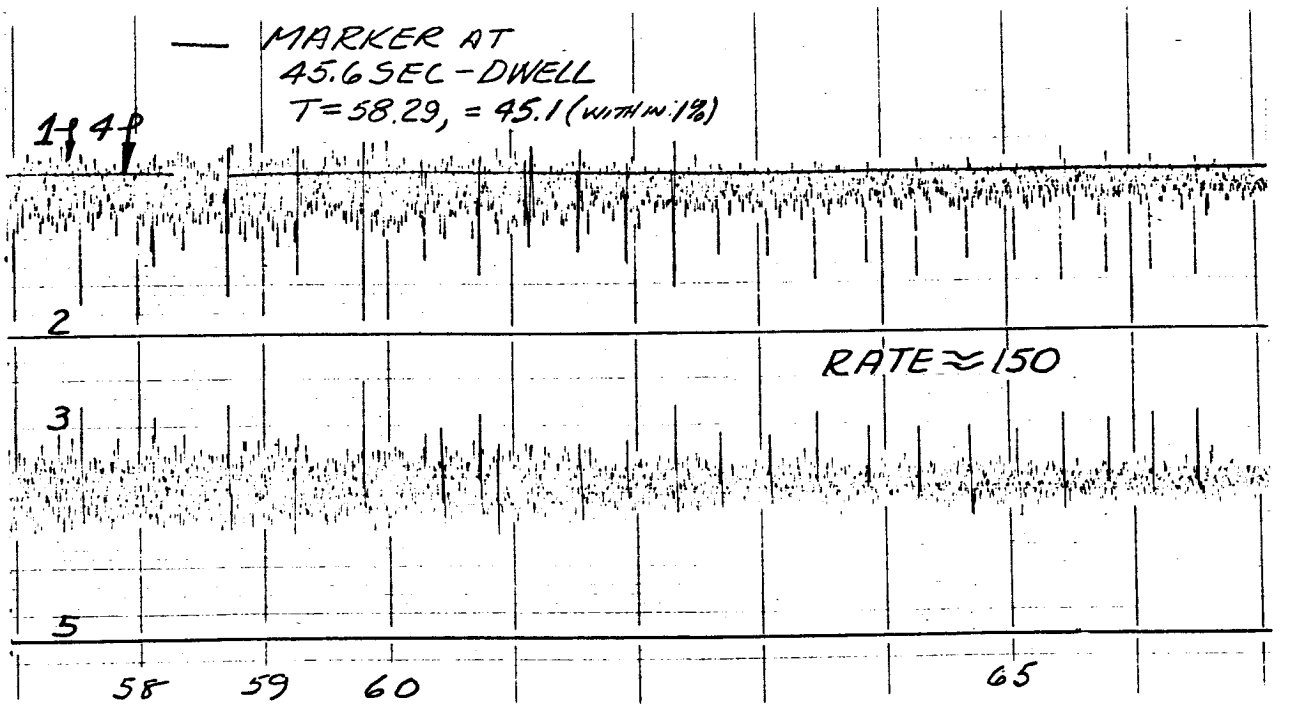


FIGURE 67. ANIMAL NO. 239.
PHASE VI - DWELL - K_t MARK, 57-67 SECONDS

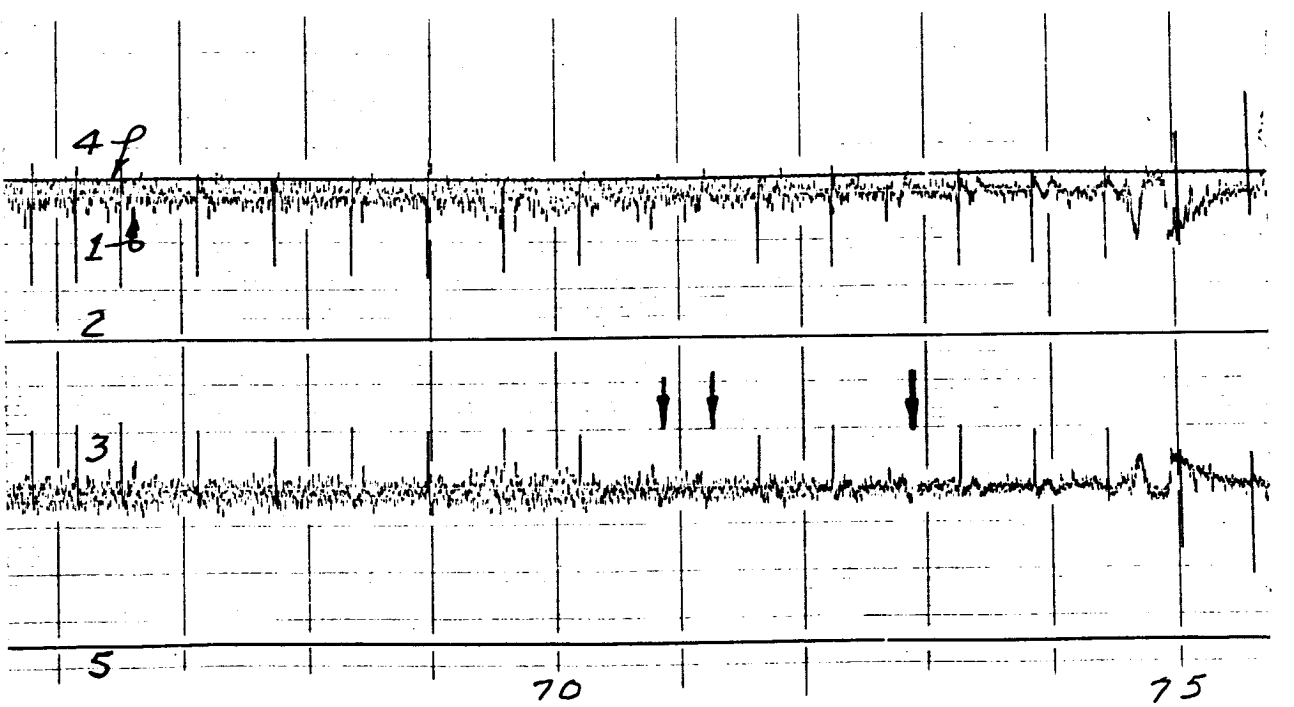


FIGURE 68. ANIMAL NO. 239. PHASE VI - DWELL, 69-75 SECONDS

Figure 69. Animal No. 239 - Phase VI, Dwell, 75-84 Seconds

The sharp shift in wave form is clearly demonstrated. The change in QRS configuration may be due to a change in position of the electrodes or a change in the anatomic position of the heart.

Figure 70. Animal No. 239 - Phase VI, Dwell, 105-114 Seconds

The periodic change in amplitude of the QRS voltage is very pronounced here and suggests thoracic movement. The electrical "noise" has again increased obscuring the fine detail of the complexes. This is assumed to be compatible with the suggested thoracic movement.

Figure 71. Animal No. 239 - Phase VII, Pre-Offset, 118-128 Seconds

Note the time marker at the extreme right indicating the end of dwell. This trace shows the few seconds of the pre-offset period. Note the alterations in amplitude of QRS pattern (6 units) at 122 sec. elapsed time. A time adjustment is made at the beginning of offset to correct for the slight error in timelines.

Figure 72. Animal No. 239 - Phase VIII, Offset, 128-138 Seconds

The wild swing of the ECG trace, suggests great thoracic movement. The approximate G level is noted at each time line. After 150 G level is reached ECG trace becomes very clear. A very sharp diminution of QRS voltage is recorded. The "end of run" marker at the extreme right indicates the centrifuge ceased rotation; and the power has been shut off.

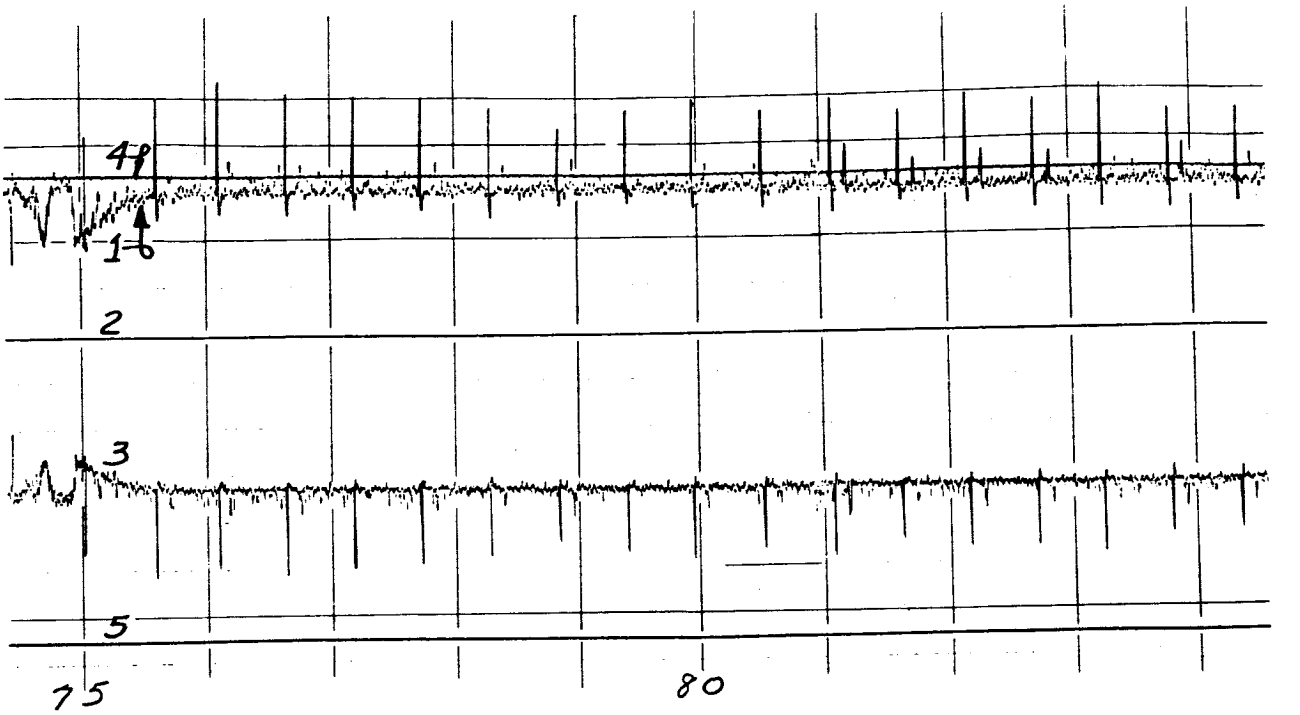


FIGURE 69. ANIMAL NO. 239. PHASE VI - DWELL, 75-84 SECONDS

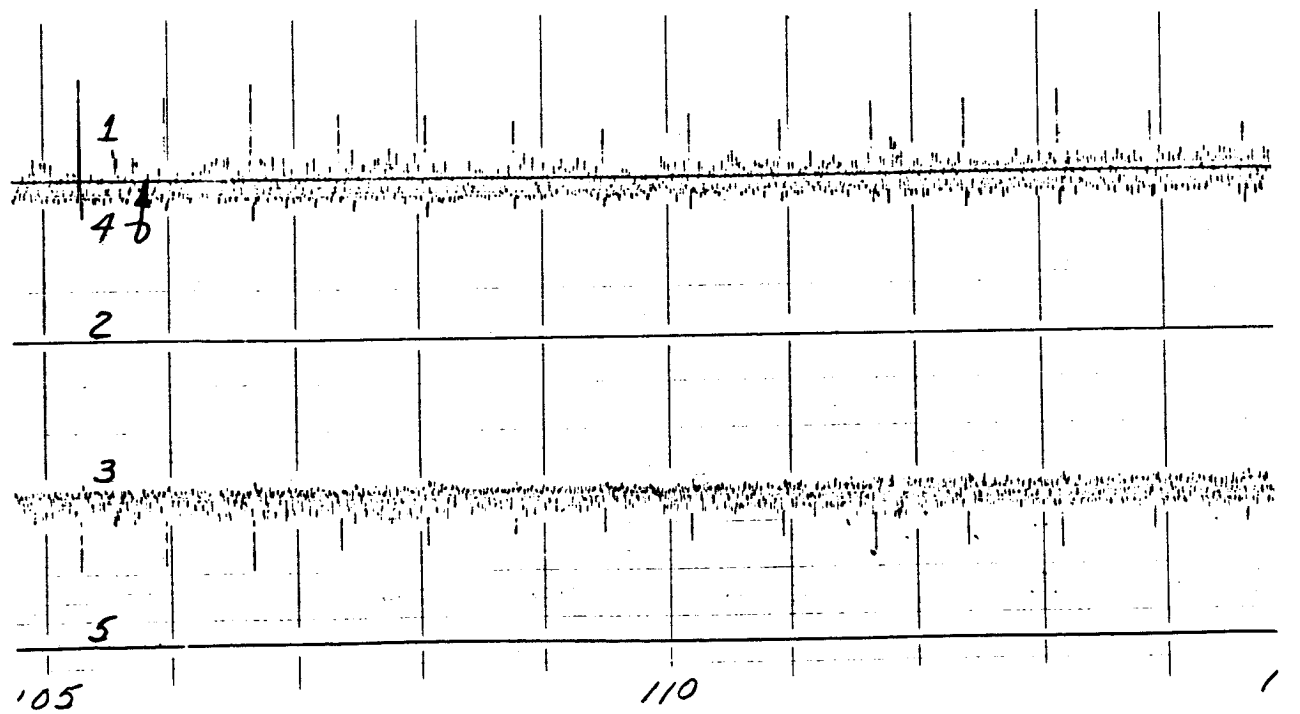


FIGURE 70. ANIMAL NO. 239. PHASE VI - DWELL, 105-114 SECONDS

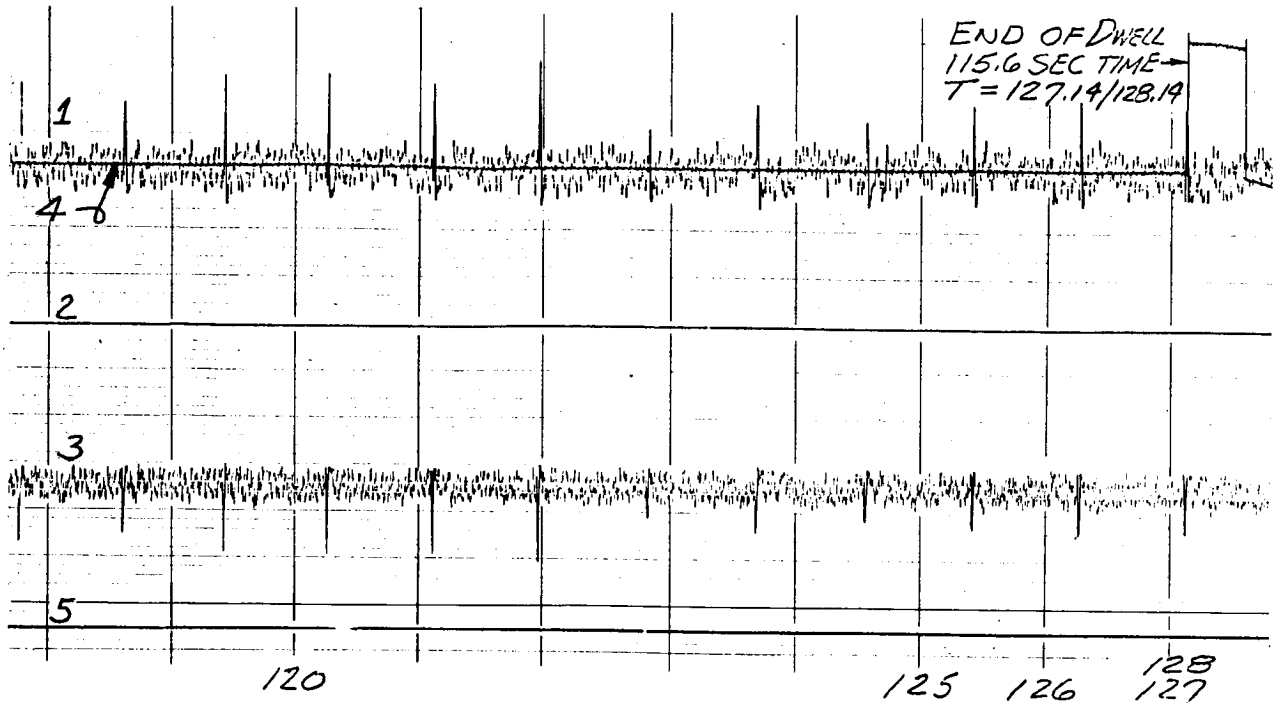


FIGURE 71. ANIMAL NO. 239. PHASE VII, PRE-OFFSET, 118-128 SECONDS

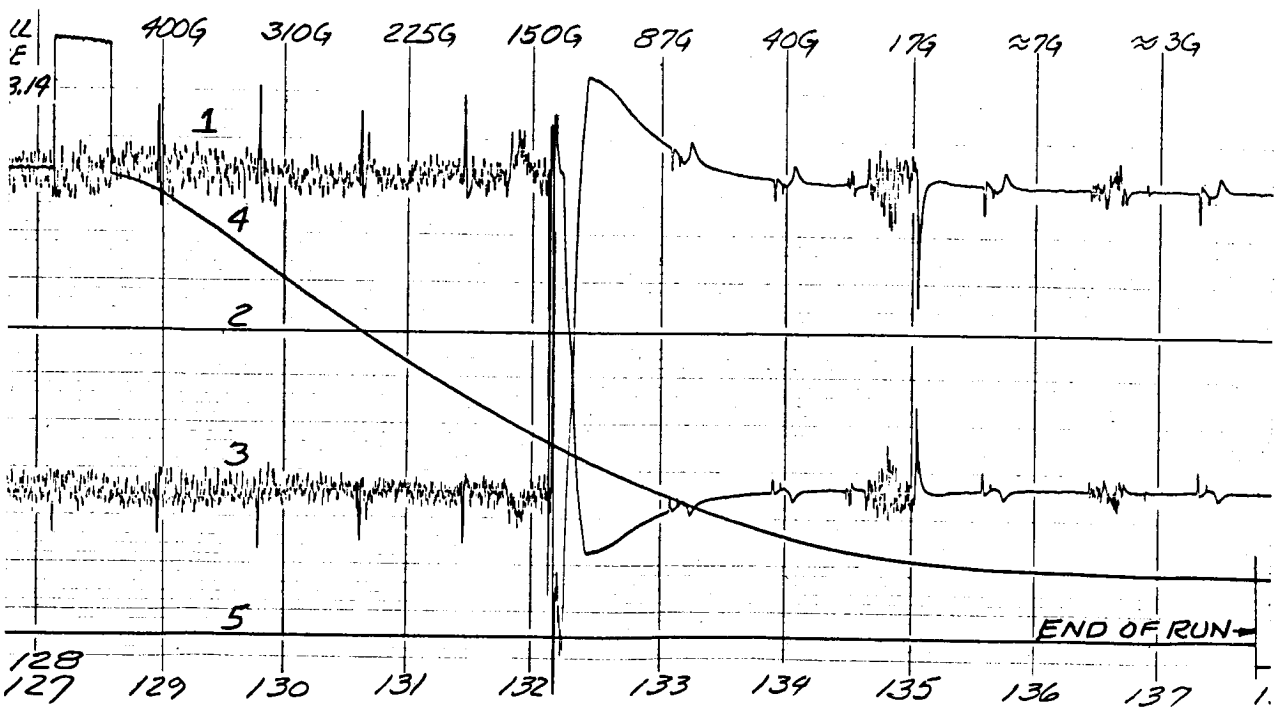


FIGURE 72. ANIMAL NO. 239. PHASE VIII, OFFSET, 128-138 SECONDS

Figure 73. Animal No. 239 -
Phase IX, Postrun 1st Minute, 138-147 Seconds

Compared to the baseline, the rhythm now is nodal with retrograde conduction to the auricular pacemaker. In addition, the coving of the ST segment and the inversion of the T wave, are evidence of ischemia and myocardial injury. The rate has been established at 60/min. QRS complex averages 3½ units (7 mm) in amplitude.

Figure 74. Animal No. 239 -
Phase IX, Postrun 1st Minute, 165-174 Seconds

The heart rate of 60 beats/min. is firmly established. Retrograde conduction to the auricular pacemaker continues. Comparing the QRS pattern shown to the baseline record, (figure 59) shows the amplitude changed from 16 units (32 mm) to 8 units (16 mm), a 50% reduction, while the duration remained constant. The paper speed here is 25 mm/sec. The animal appears to be unconscious.

Figure 75. Animal No. 239 -
Phase X, Postrun 2nd Minute, 194-204 Seconds

The QRS amplitude is 10½ units (21 mm). A temporary decrease in QRS voltage occurs at the extreme right of the trace at which point also the paper speed is changed from 25 mm/sec. to 100 mm/sec. At this point also a change in QRS configuration is seen. The rate is 60/min. diphasic with the T wave present.

Figure 76. Animal No. 239 -
Phase X, Postrun 2nd Minute, 206-208 Seconds

This figure shows the ECG pattern at 100 mm/sec. paper speed and reflects nodal rhythm with a shifting pacemaker.

Figure 77. Animal No. 239 - Phase X,
Postrun 2nd Minute, 208-210 Seconds

Paper speed 100 mm/sec. Heart rate 60/min. Rhythm is nodal with shifting of the nodal pacemaker indicated by the arrows.

Figure 78. Animal No. 239 -
Phase X, Postrun 2nd Minute, 215-224 Seconds

The increase of rate to approximately 90/min., is variable with the distances between complexes shifting. Note the well developed diphasic T wave. Arrows indicate sites of ventricular extrasystole. The paper speed is again 25 mm/second.

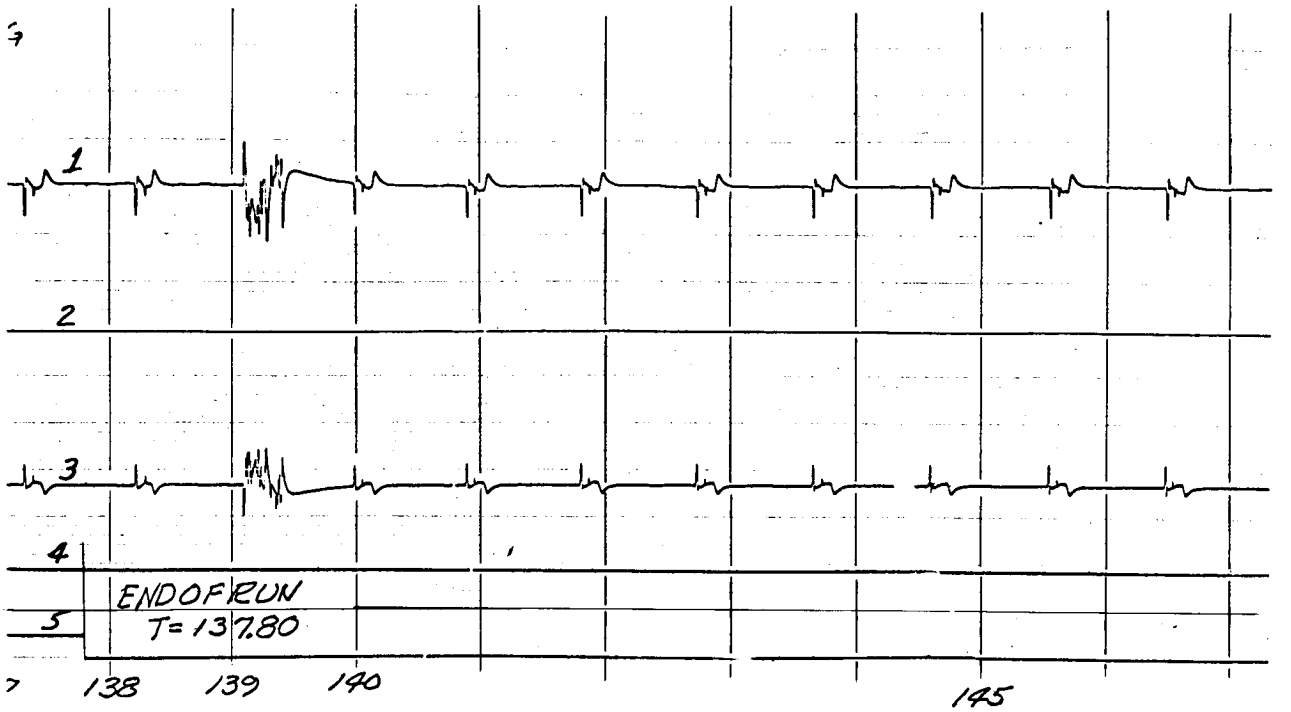


FIGURE 73. ANIMAL NO. 239.
PHASE IX, POSTRUN 1ST MINUTE, 138-147 SECONDS

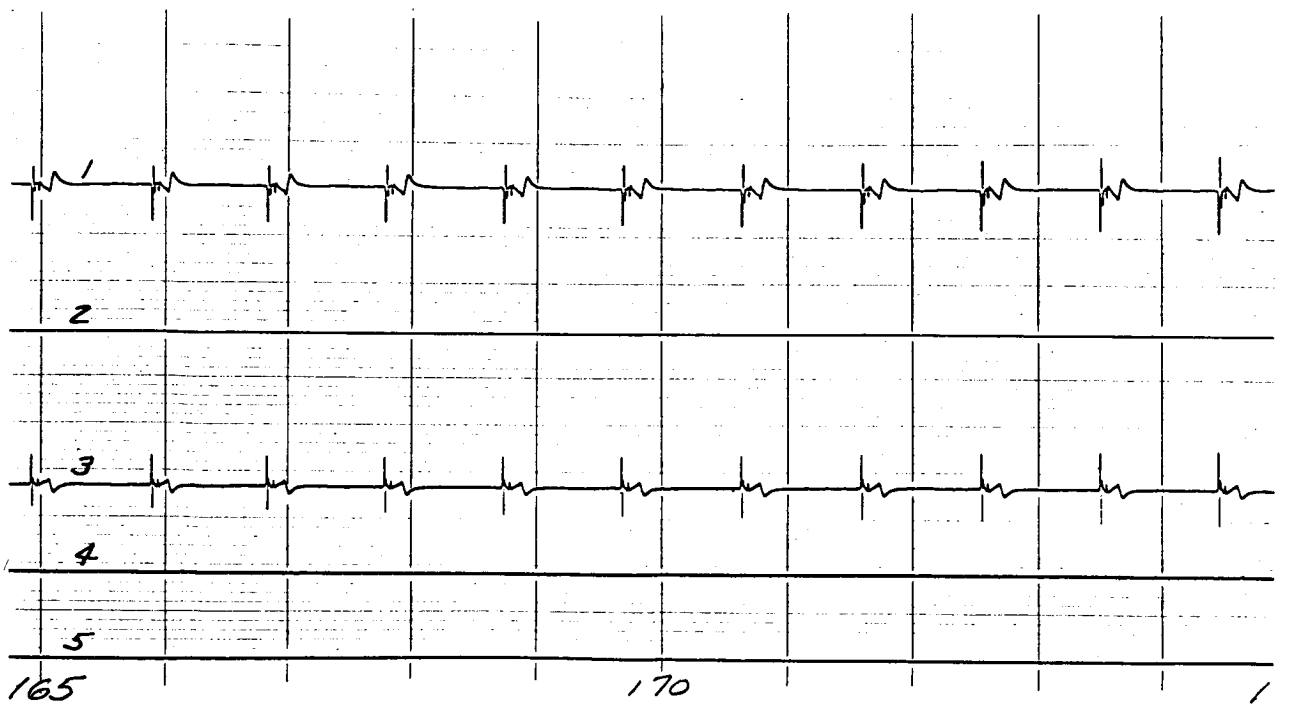


FIGURE 74. ANIMAL 239.
PHASE IX, POSTRUN 1ST MINUTE, 165-174 SECONDS

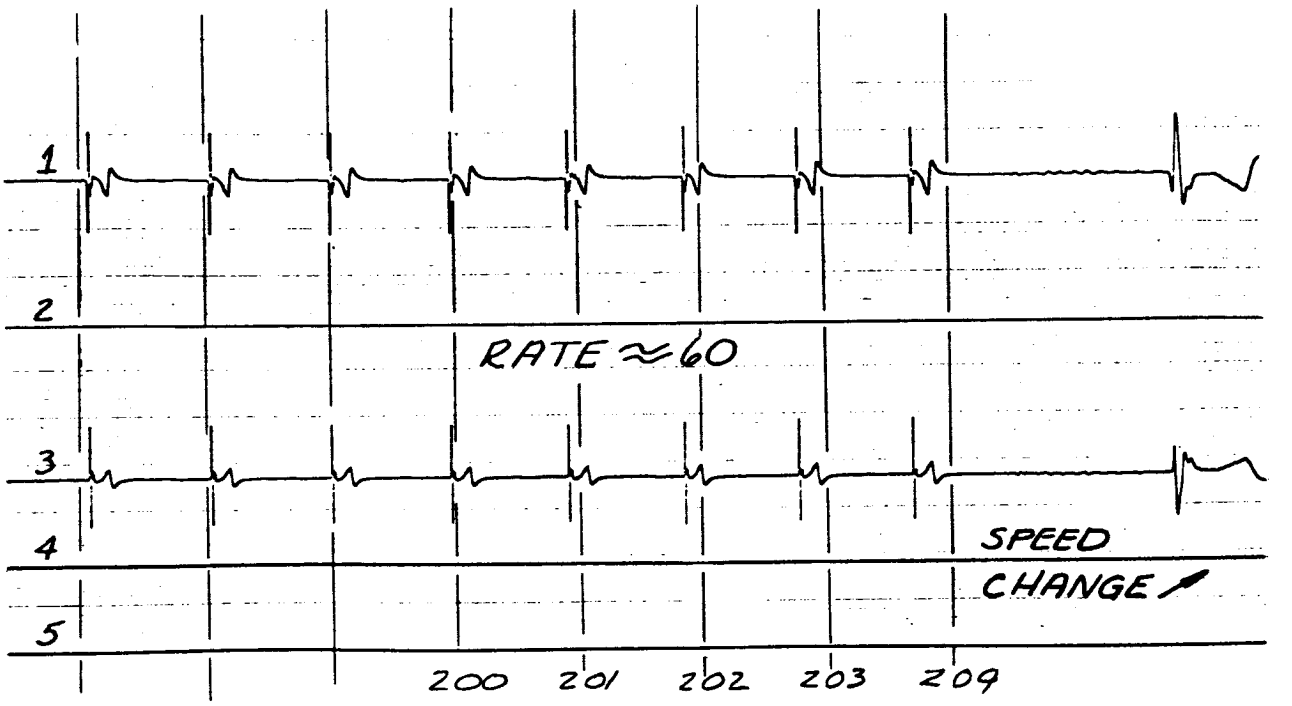


FIGURE 75. ANIMAL NO. 239.
PHASE X, POSTRUN 2ND MINUTE, 194-204 SECONDS

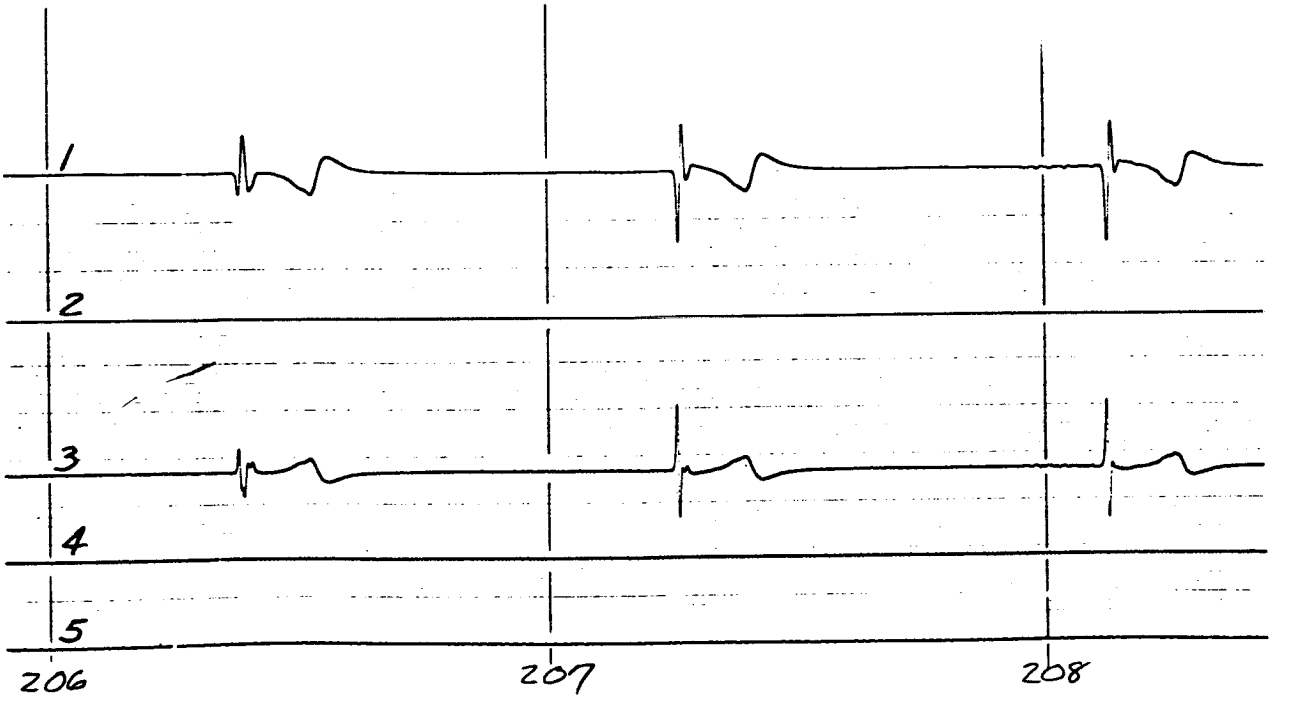


FIGURE 76. ANIMAL NO. 239.
PHASE X, POSTRUN 2ND MINUTE, 206-208 SECONDS

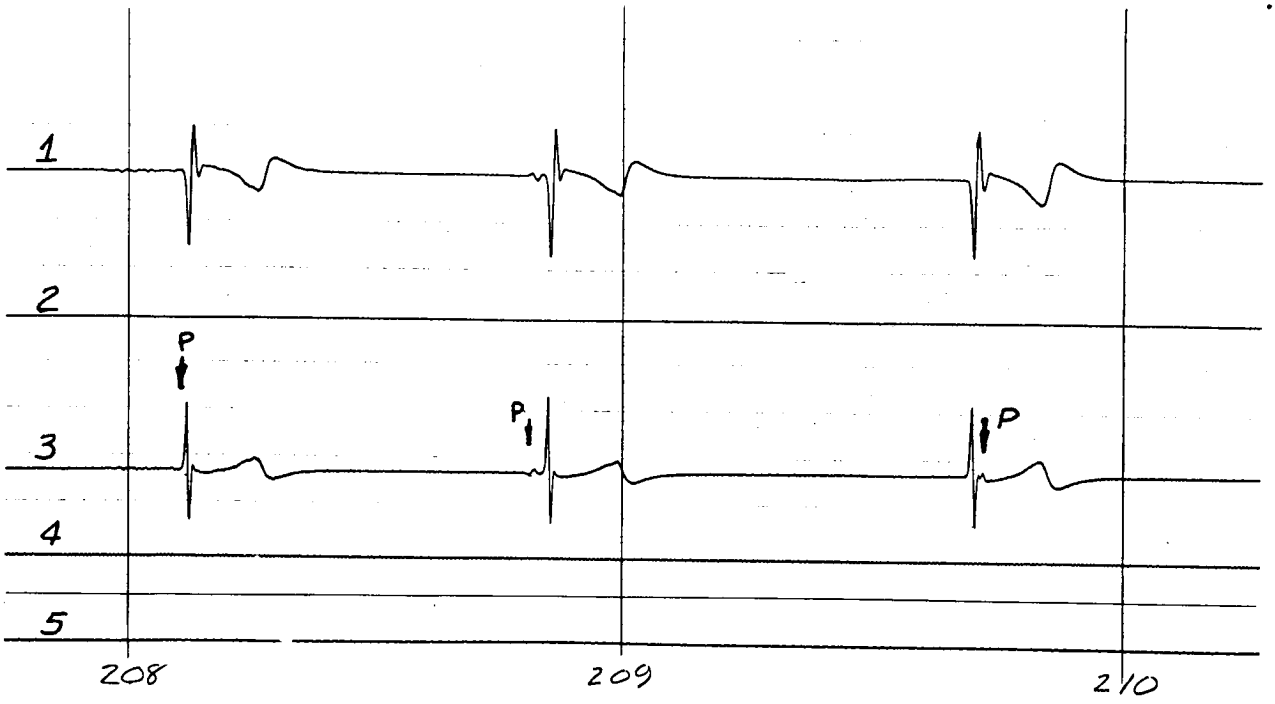


FIGURE 77. ANIMAL NO. 239. PHASE X, POSTRUN 2ND MINUTE, 208-210 SECONDS

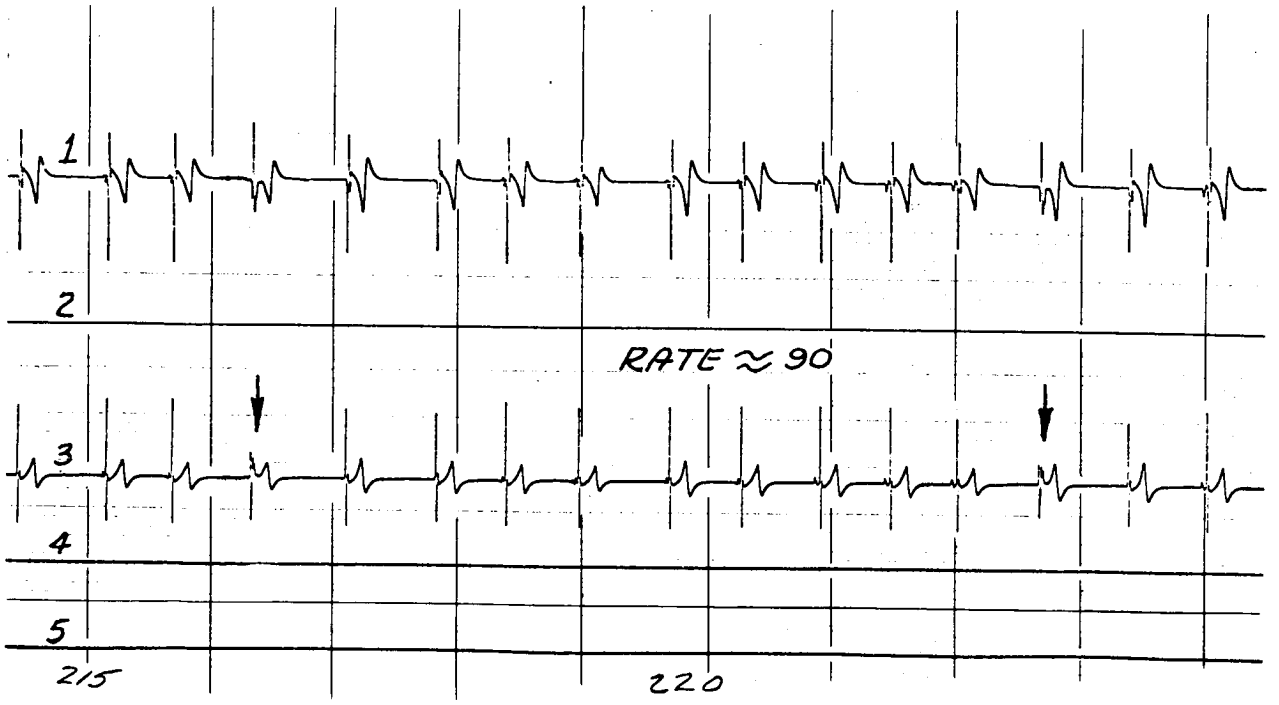


FIGURE 78. ANIMAL NO. 239. PHASE X, POSTRUN 2ND MINUTE, 215-224 SECONDS

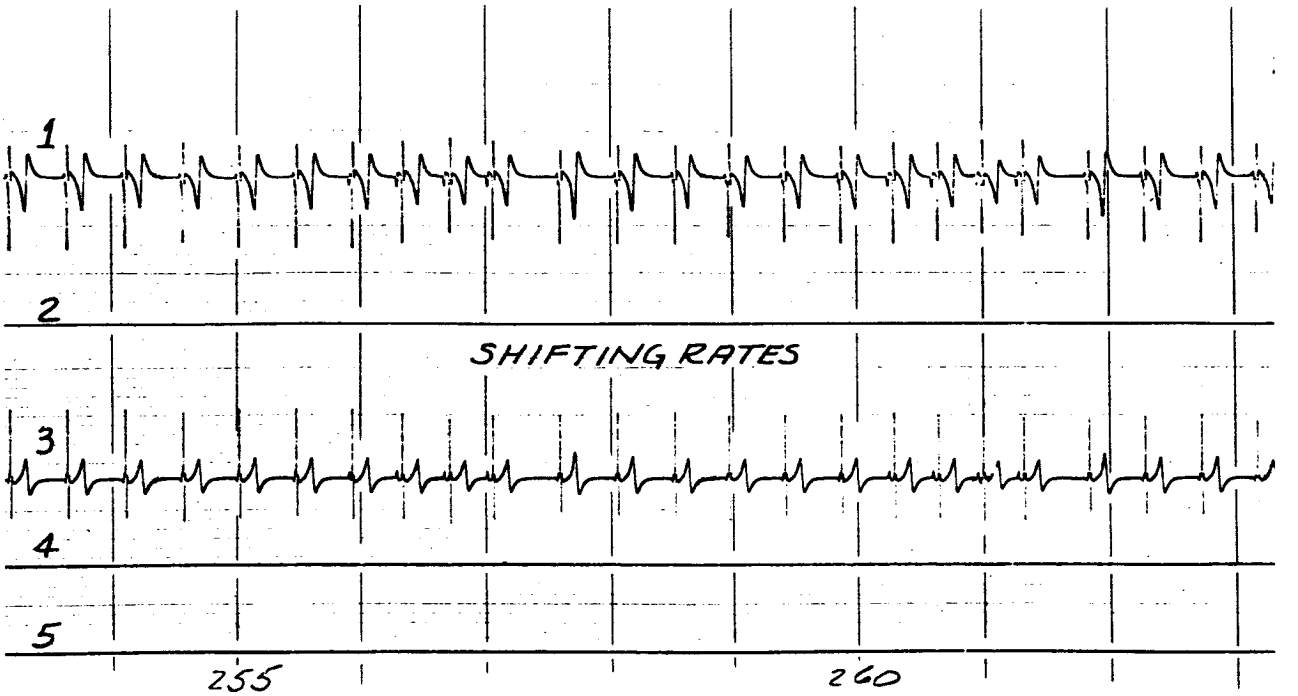


FIGURE 79. ANIMAL NO. 239.
PHASE XI, POSTRUN 3RD MINUTE, 257-263 SECONDS

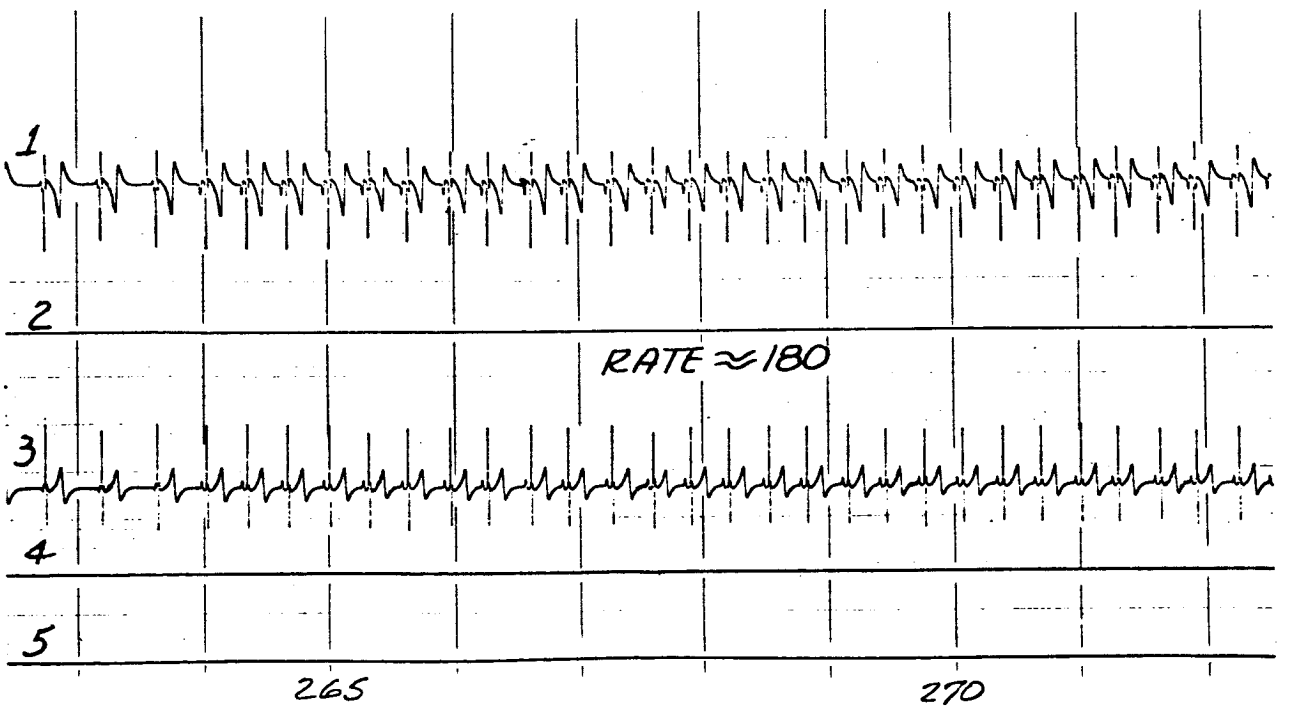


FIGURE 80. ANIMAL NO. 239.
PHASE XI, POSTRUN 3RD MINUTE, 263-272 SECONDS

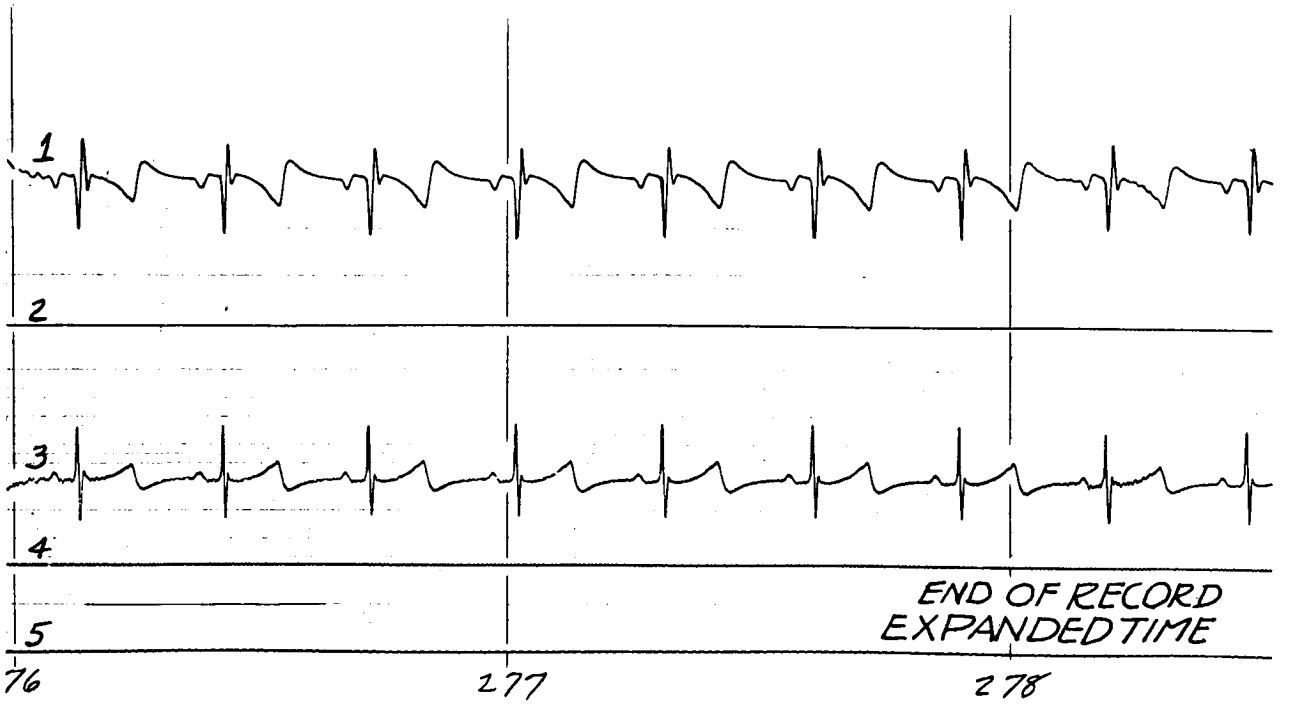


FIGURE 81. ANIMAL NO. 239.
PHASE XI, POSTRUN 3RD MINUTE, 276-278 SECONDS

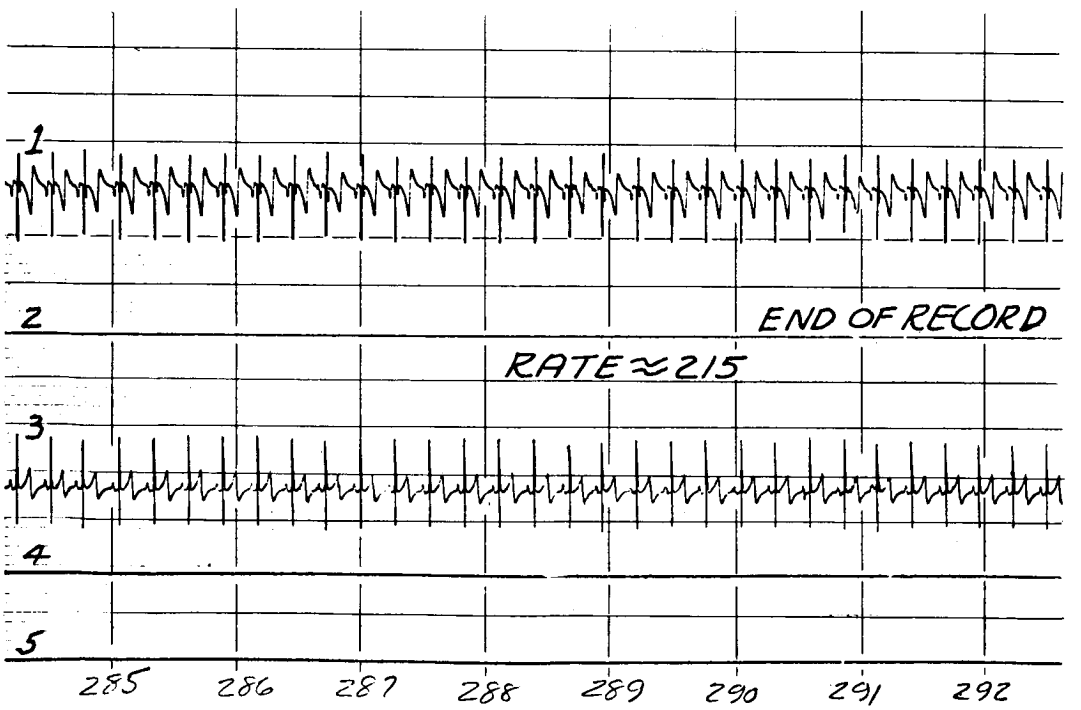


FIGURE 82. ANIMAL NO. 239. PHASE XI, POSTRUN 3RD MINUTE

Figure 79. Animal No. 239 -
Phase XI, Postrun 3rd Minute, 257-263 Seconds

Note the shifting rates of the heart as it goes from 120 to 180 then back to 120/min. Control of the heart has been regained by the SA node and the rhythm is sinus. The diphasic T wave persists.

Figure 80. Animal No. 239 -
Phase XI, Postrun 3rd Minute, 263-272 Seconds

Rate is well established at 180. The rhythm is sinus. Note the diphasic T wave. QRS duration is at pre-run value. QRS height is 10 units.

Figure 81. Animal No. 239 -
Phase XI, Postrun 3rd Minute, 276-278 Seconds

Paper speed 100 mm/sec. The T wave is observed to be less diphasic. Rate has increased to approximately 215/min. and is sinus.

Figure 82. Animal No. 239 -
Phase XI, Postrun, 3rd Minute

Rhythm is firmly established as sinus. The diphasic T wave persists, suggesting myocardial damage and ischemia. Paper speed is now 25 mm/sec. This is the end of the ECG record. The animal survived.

SECTION FOUR. ANIMAL NO. 241-242, SEX MALE

On June 30, 1964, this animal as No. 241 was exposed to +200 G_x for a total dwell of 213 seconds. Its weight was 601.5 grams. Calculated D_t was 83 seconds, D_r was 213 seconds. The K_p reached was 2.6×10^4. The animal survived. Three months later, September 11, 1964, as No. 242 this animal was exposed to +200 G_x for a total dwell of 202.5 seconds. Its new weight was 695 grams, a weight gain of 94 grams. Its calculated D_t was 72.5 seconds, D_r was 202.5 seconds. The K_p reached was 2.8×10^4. The animal was a clinical survivor. It was then exsanguinated and labeled the Rerun Control for the special enzyme study.

Figure 83 (A). Animal No. 241 - Phase I - Baseline

Paper speed is 100 mm/sec. Baseline is shown of animal No. 241 prior to its run on 30 June 1964. Compare to figure below and to figure 84A which follows.

Figure 83 (B). Animal No. 242 - Phase I - Baseline

Paper speed is 100 mm/sec. This is the pre-run baseline of animal No. 242 on 11 September 1964. Compare to figure above and to figure 84B which follows.

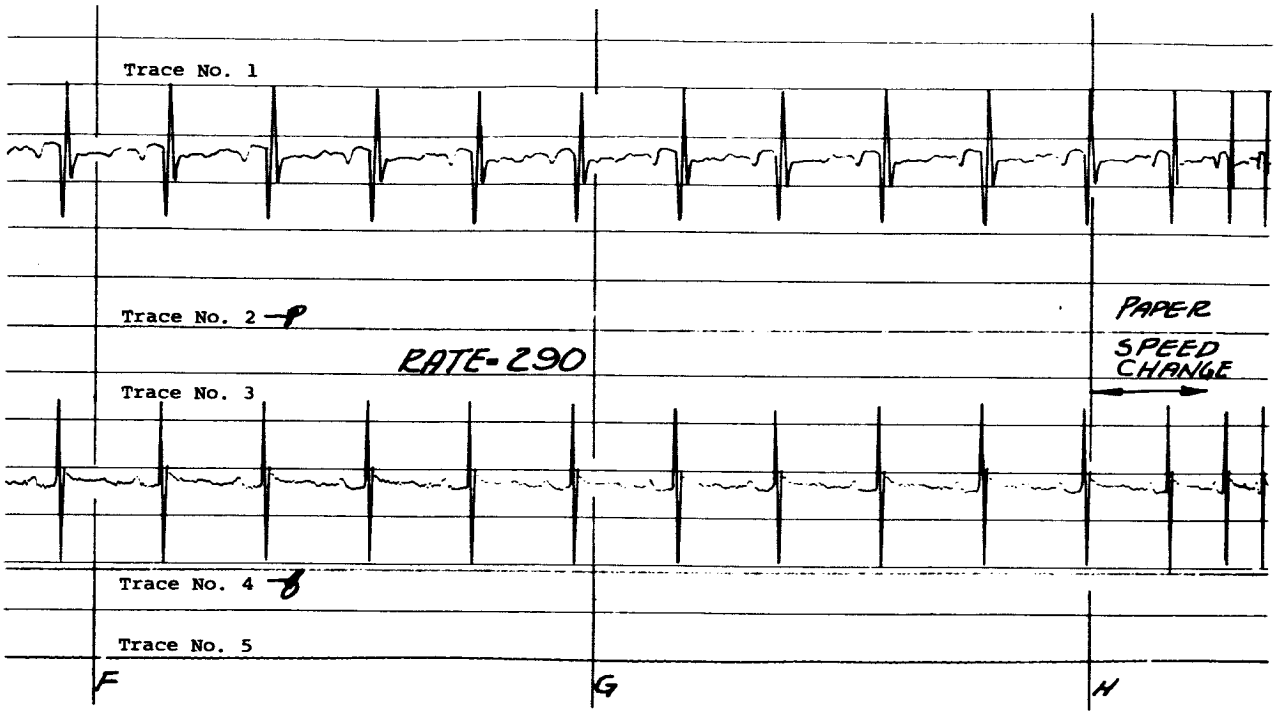


FIGURE 83 (A). ANIMAL NO. 241. PHASE I - BASELINE

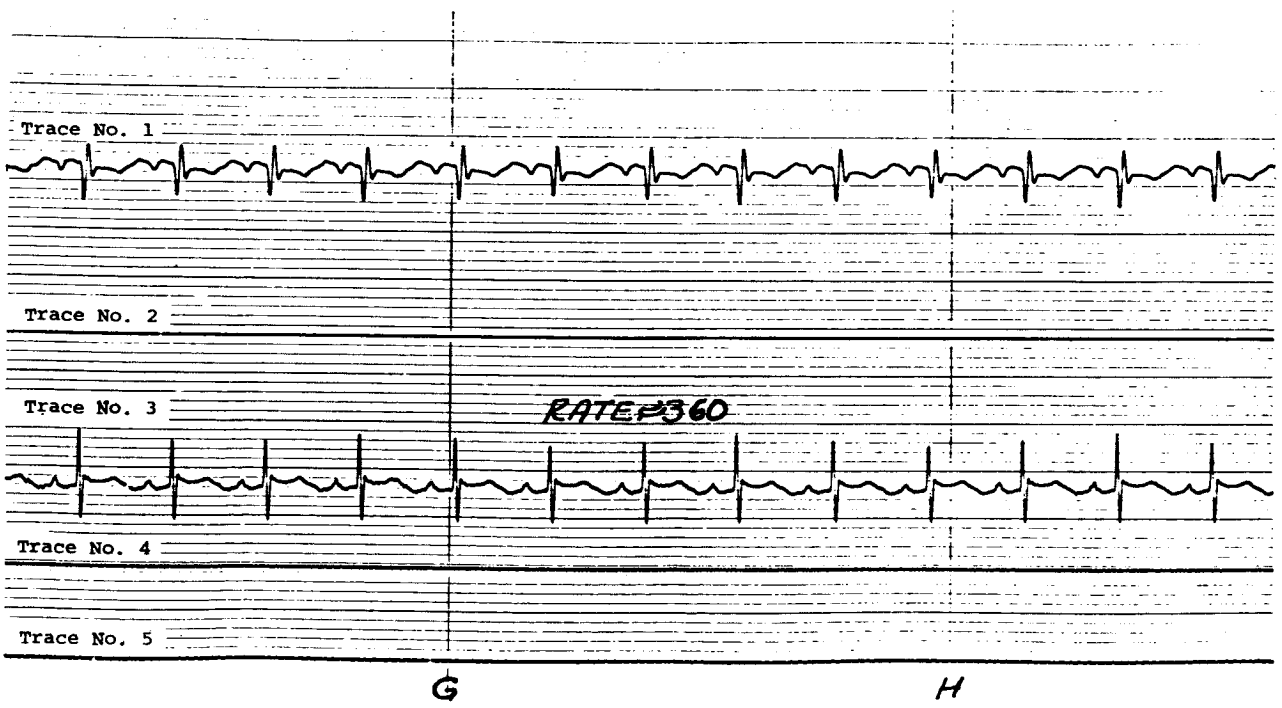


FIGURE 83 (B). ANIMAL NO. 242. PHASE I - BASELINE

Figure 84 (A). Animal No. 241 -
Phase XII - End of Run, 418-420 Seconds

This trace demonstrates the end of the post-run period for animal No. 241. Date of run was 30 June 1964. Paper speed is 270/min. Note the widened QRS diphasic T wave and coving of ST segment, all cudicative of myocardial ischemia and damage. QRS amplitude is 11 units (22 mm).

Figure 84 (B). Animal No. 242 -
Phase I - Baseline (Animal No. 241 after 3 Months)

This figure (identical to figure 83B) demonstrates the ECG of the same animal described above. Date of this trace was 11 September 1964. Animal is henceforth labeled No. 242. Baseline rate is 360/min. QRS amplitude is 9 units (18 mm).

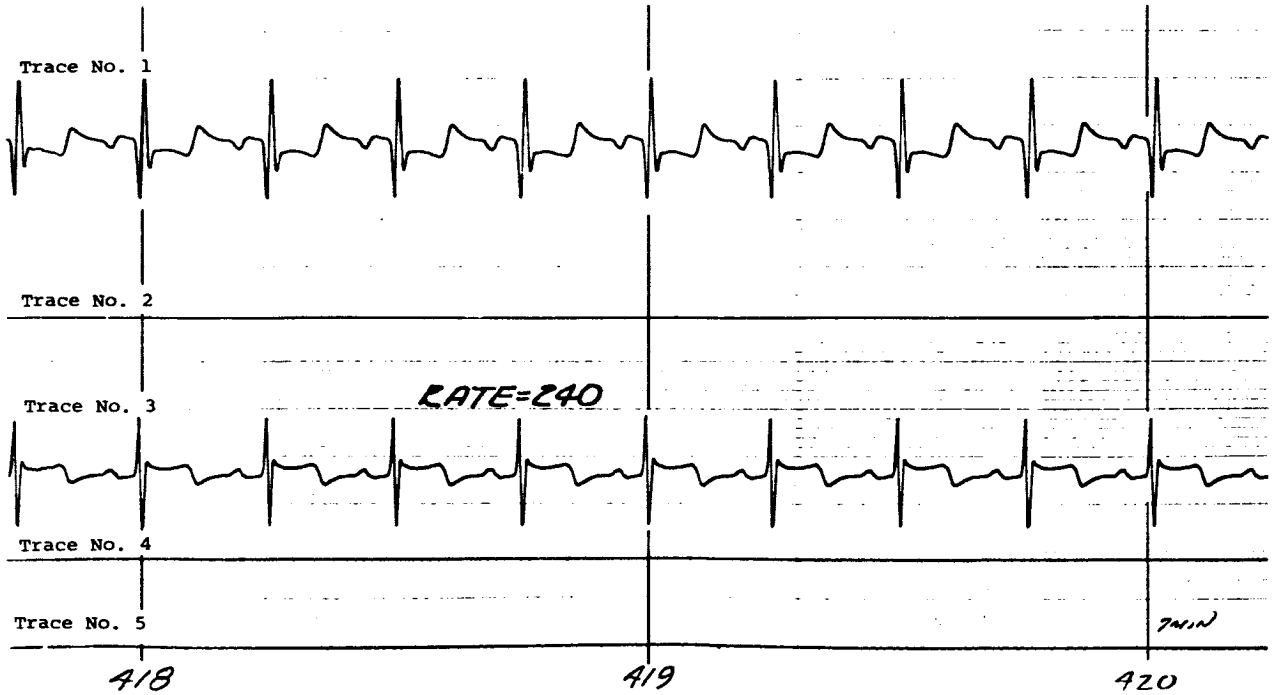


FIGURE 84 (A). ANIMAL NO. 241.
PHASE XII - END OF RUN, 418-420 SECONDS

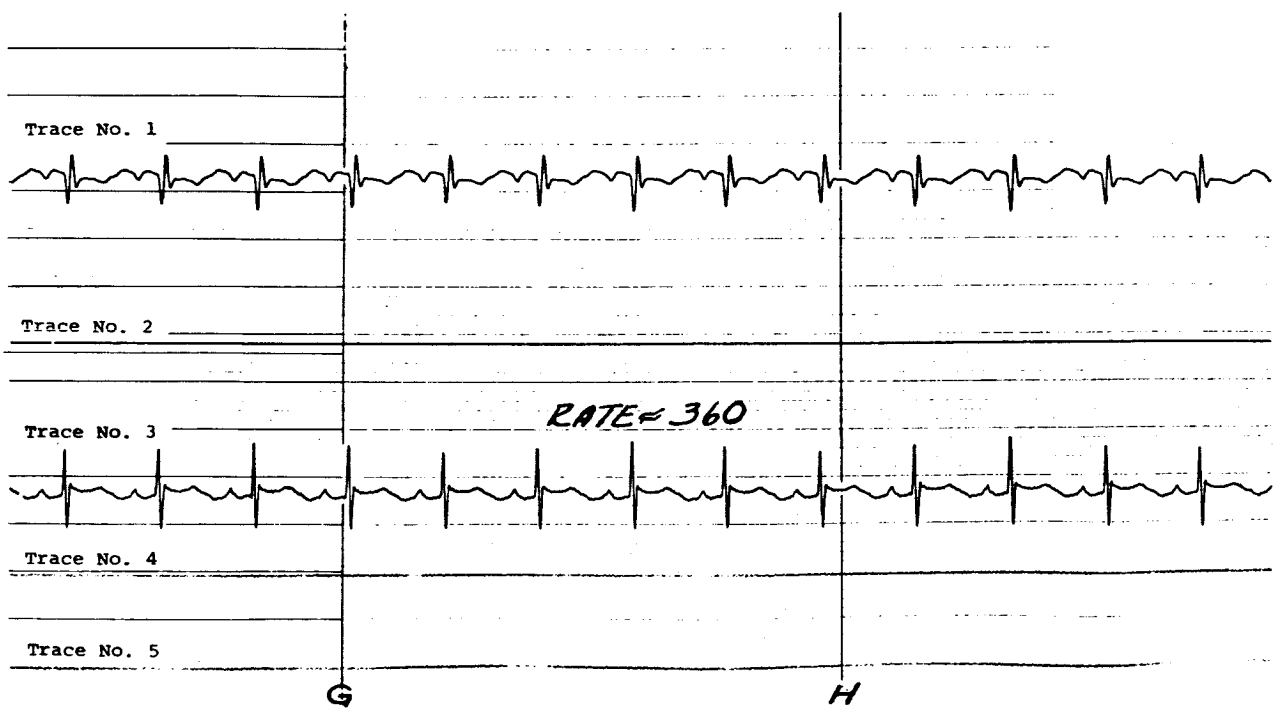


FIGURE 84 (B). ANIMAL NO. 242.
PHASE I - BASELINE (ANIMAL NO. 241 AFTER 3 MONTHS)

Figure 85 (A). Animal No. 241 - Phase III - Onset, 0-1 Seconds

Heart rate is 300/min. Note muscle tremor noise. QRS voltage is 16 units (32 mm). Note changes in peak to peak QRS voltage.

Figure 85 (B). Animal No. 242 - Phase III - Onset, 0-2 Seconds

Note tachycardia. Heart rate is 360/min. Note muscle tremor noise. QRS voltage is 8 units. QU interval is 12 mm. Note peak to peak change in QRS voltage.

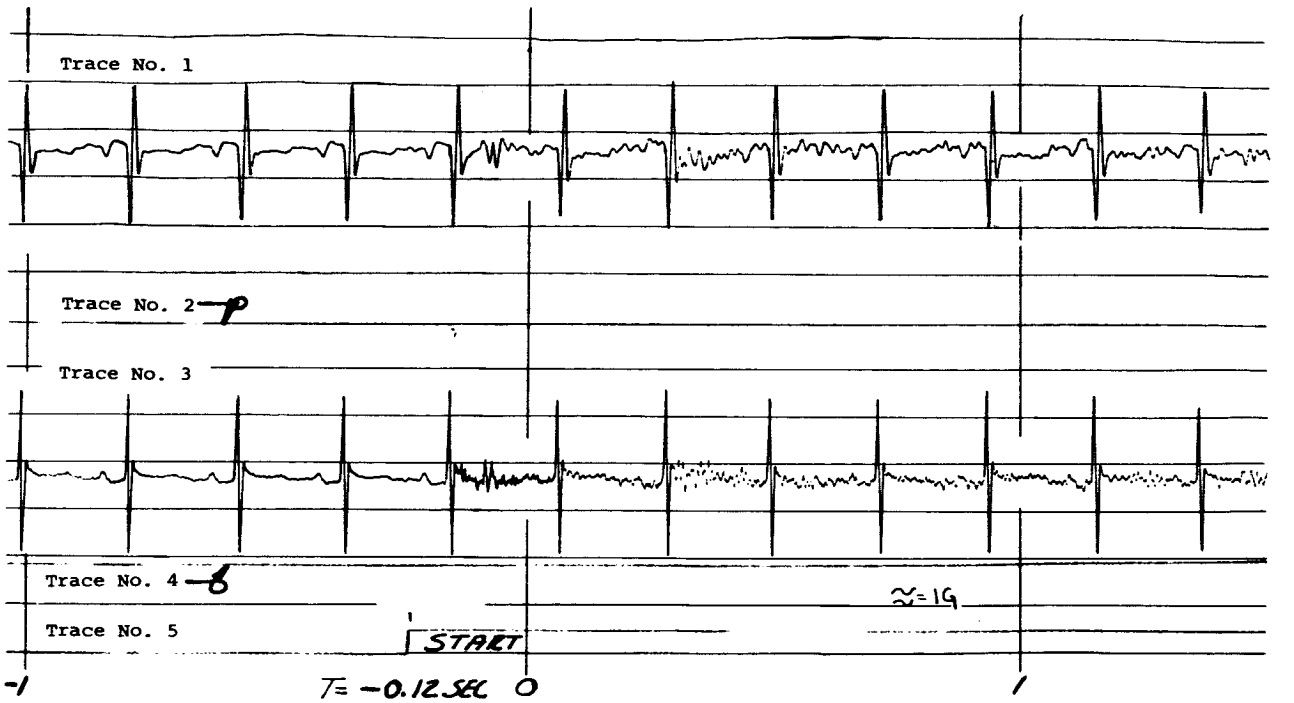


FIGURE 85 (A). ANIMAL NO. 241. PHASE III - ONSET, 0-1 SECONDS

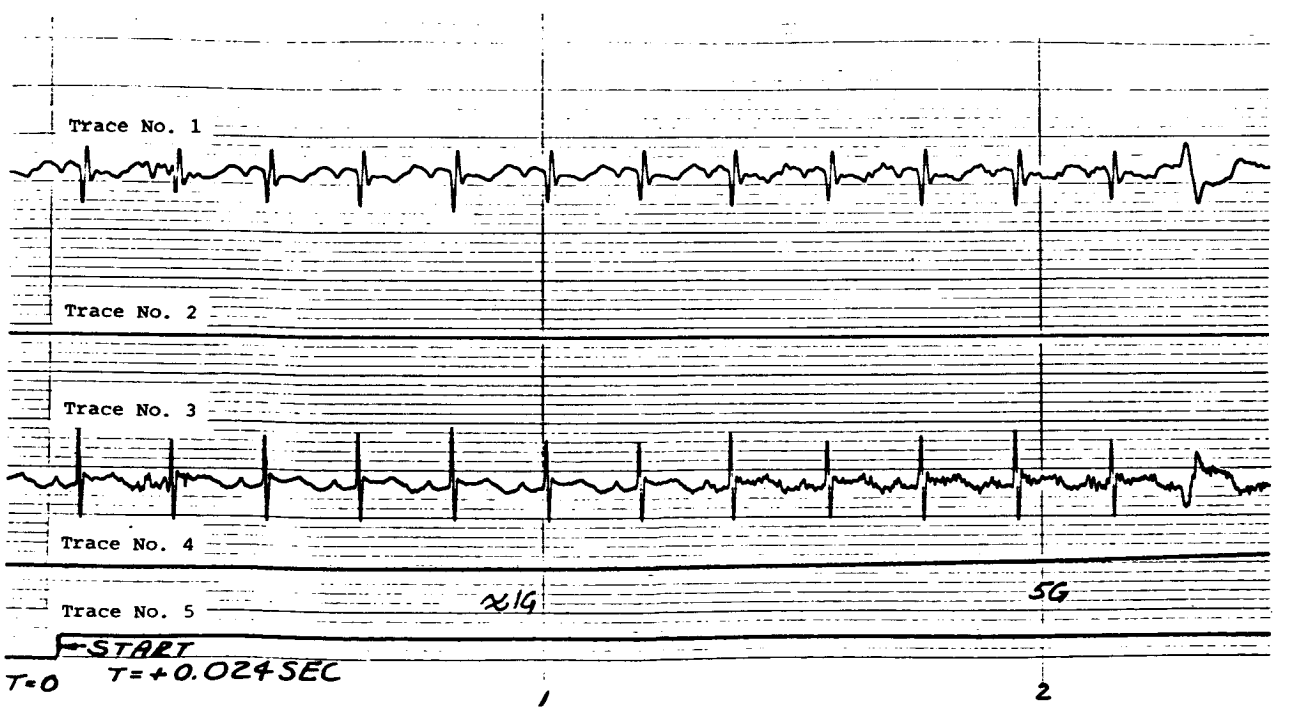


FIGURE 85 (B). ANIMAL NO. 242. PHASE III - ONSET, 0-2 SECONDS

Figure 86 (A). Animal No. 241 -
Phase III - Onset, 6-8 Seconds - Target G

Target G is reached by Trace No. 4 on the right at arrow. G levels are noted at timelines. Heart rate is 240/min. An area of questionable cardia arrest is seen at 8 seconds of elapsed run time. There is a definite diminution of voltage of QRS. Muscle tremor noise is in evidence.

Figure 86 (B). Animal No. 242 -
Phase III - Onset, 6-8 Seconds - Target G

Target G is reached by Trace No. 4 on the right at arrow. An area of questionable cardia arrest is seen at the right at 8 seconds of elapsed run time. There is a definite drop in the QRS voltage. Muscle tremor noise is evident.

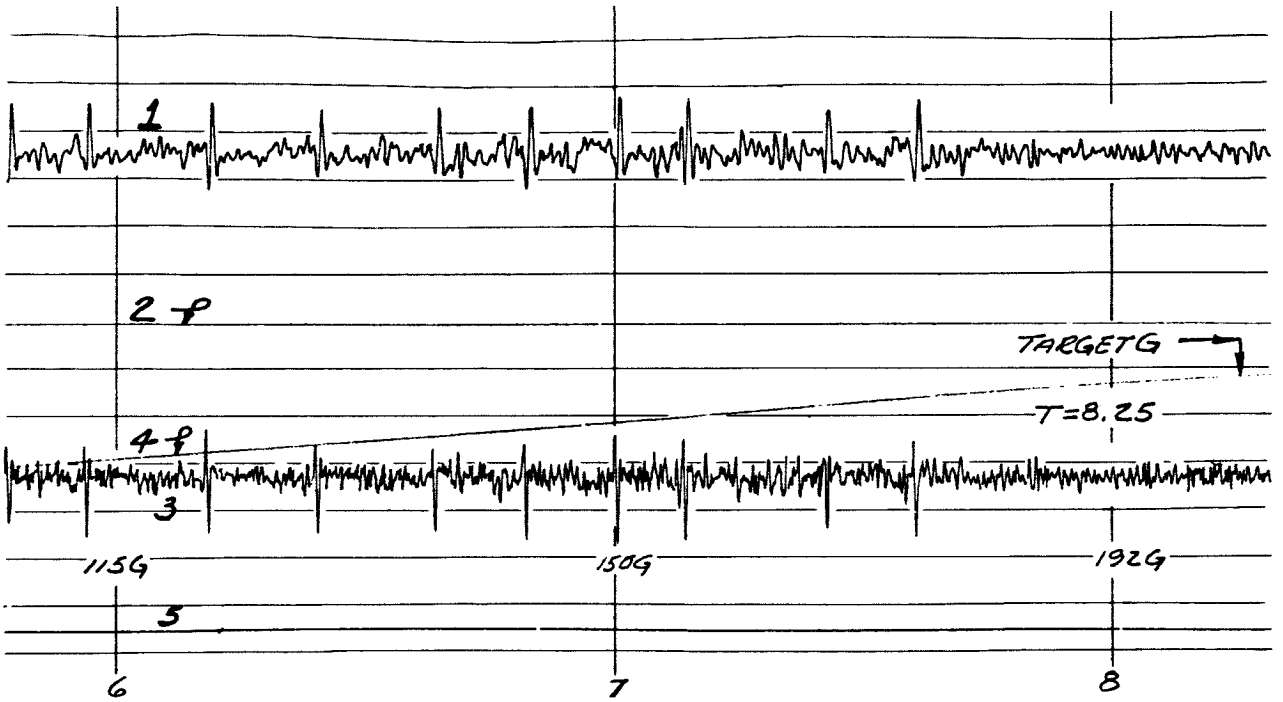


FIGURE 86 (A). ANIMAL NO. 241.
PHASE III - ONSET, 6-8 SECONDS - TARGET G

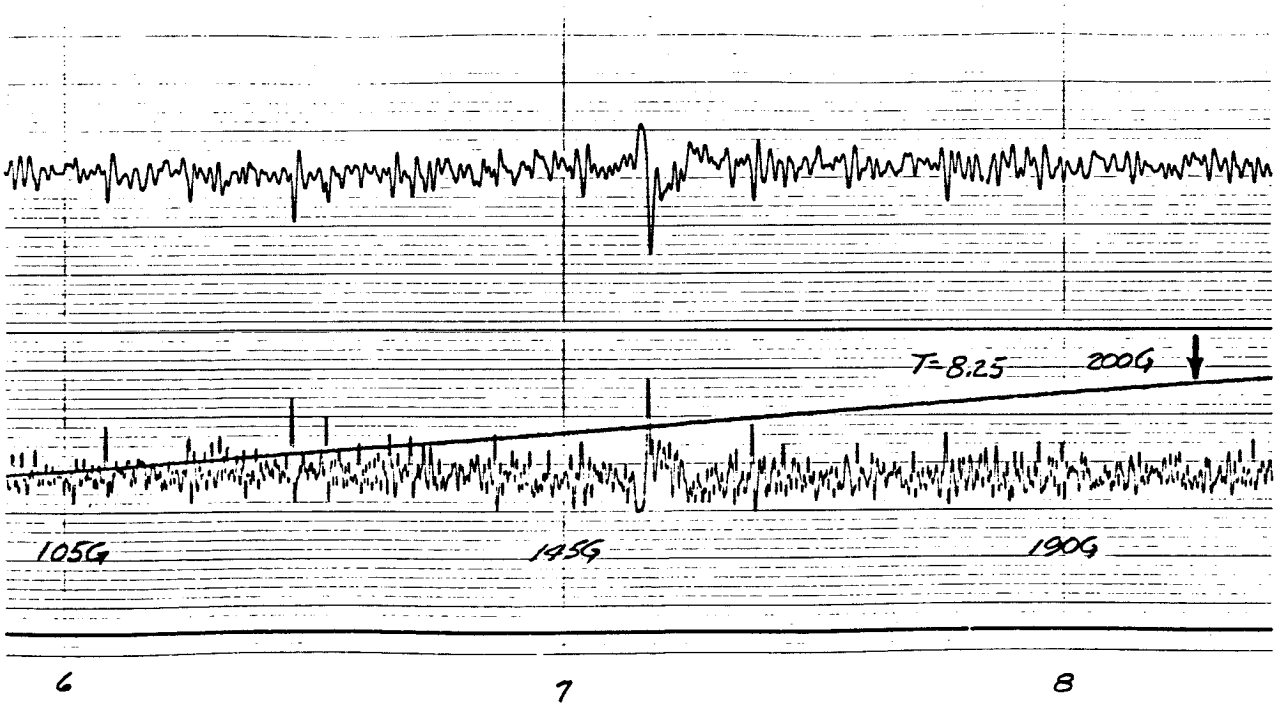


FIGURE 86 (B). ANIMAL NO. 242.
PHASE III - ONSET, 6-8 SECONDS - TARGET G

Figure 87 (A). Animal No. 241 - Phase IV - Overshoot, 9-10 Seconds

Note marker showing attainment of peak G and beginning of dwell. Muscle tremor noise is in evidence. An apparant period of cardiac arrest is seen between 8 and 9 seconds with a definite complex at 10 seconds. Arrow at extreme right indicates end of overshoot.

Figure 87 (B). Animal No. 242 - Phase IV - Overshoot, 9-10 Seconds

Note marker showing attainment of peak G and beginning of dwell. Muscle tremor noise is in evidence. Obscures QRS complexes, and does not permit any definite statement about trace. Target G is again attained at the arrow on the right.

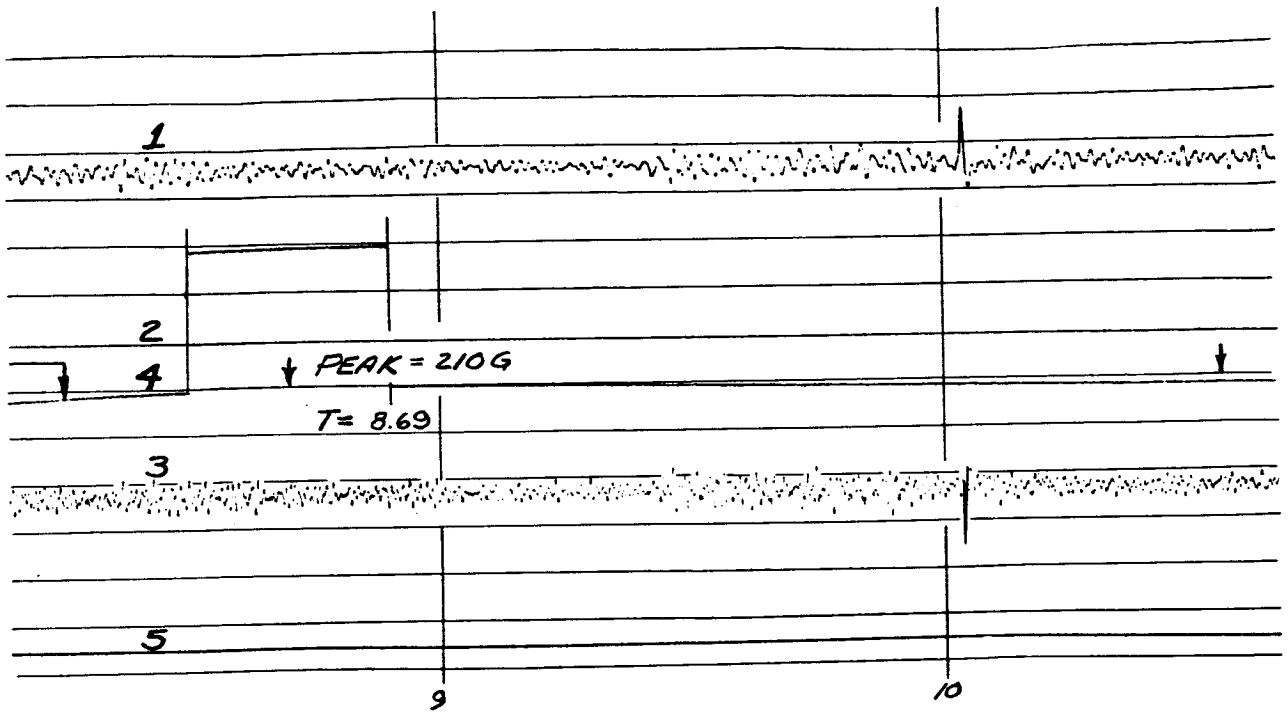


FIGURE 87 (A). ANIMAL NO. 241. PHASE IV - OVERSHOOT, 9-10 SECONDS

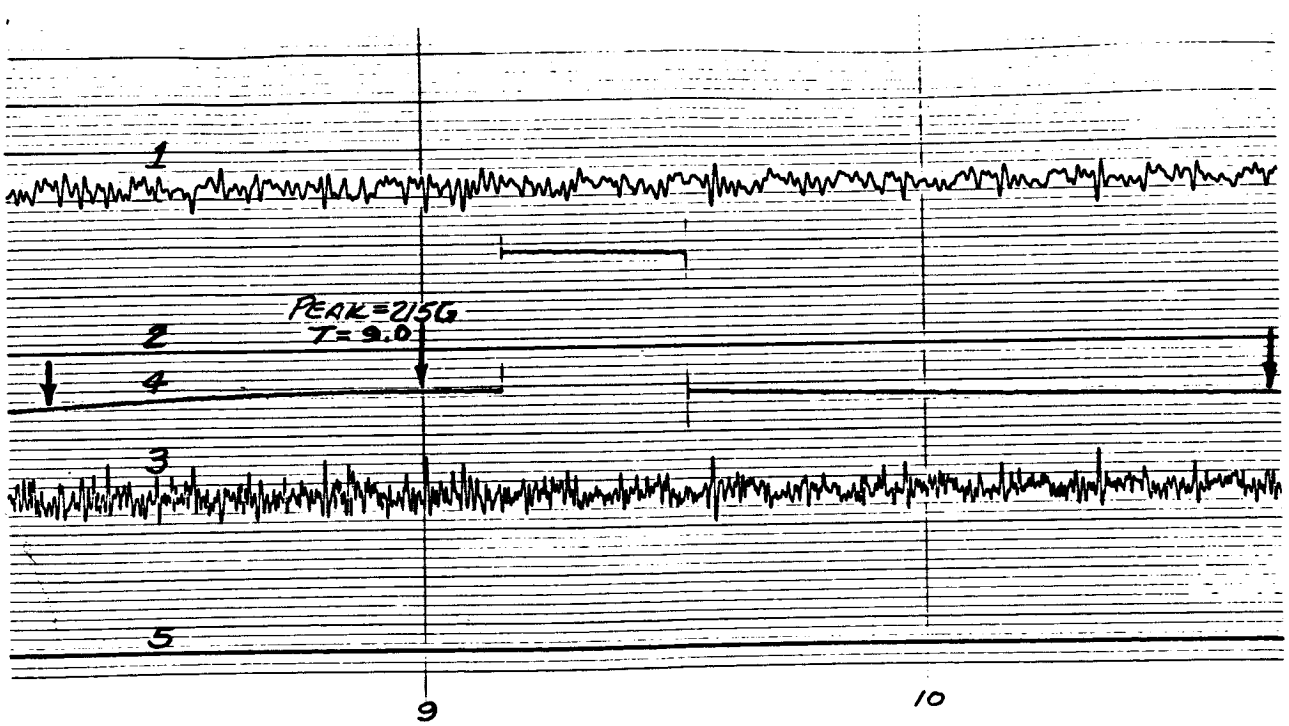


FIGURE 87 (B). ANIMAL NO. 242. PHASE IV - OVERSHOOT, 9-10 SECONDS

Figure 88 (A). Animal No. 241 -
Phase V - Initial Dwell, 11-13 Seconds

This trace demonstrates an area of decreased appreciation for QRS voltages. There may be a period of cardiac arrest. P waves cannot be distinguished from muscle tremor noise.

Figure 88 (B). Animal No. 242 -
Phase V - Initial Dwell, 11-13 Seconds

Decreased QRS voltages are in evidence. This area may represent a period of cardiac arrest. Muscle noise is present.

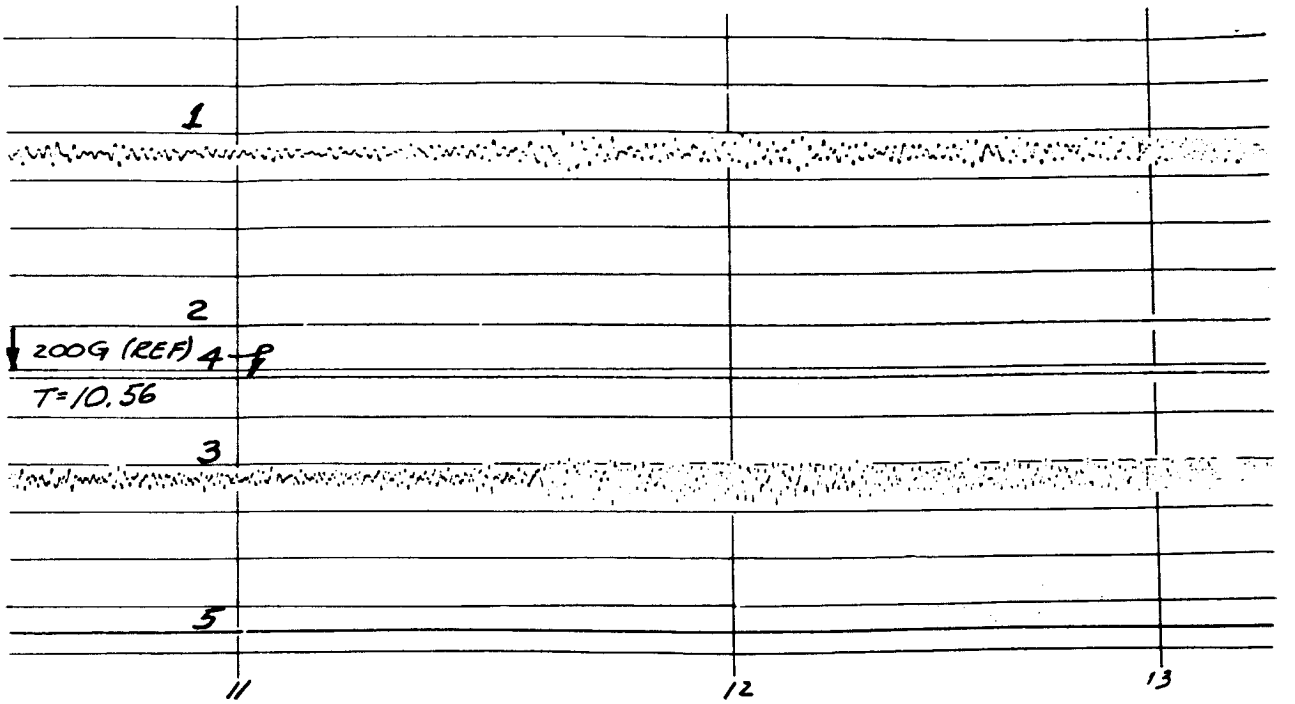


FIGURE 88 (A). ANIMAL NO. 241.
PHASE V - INITIAL DWELL, 11-13 SECONDS

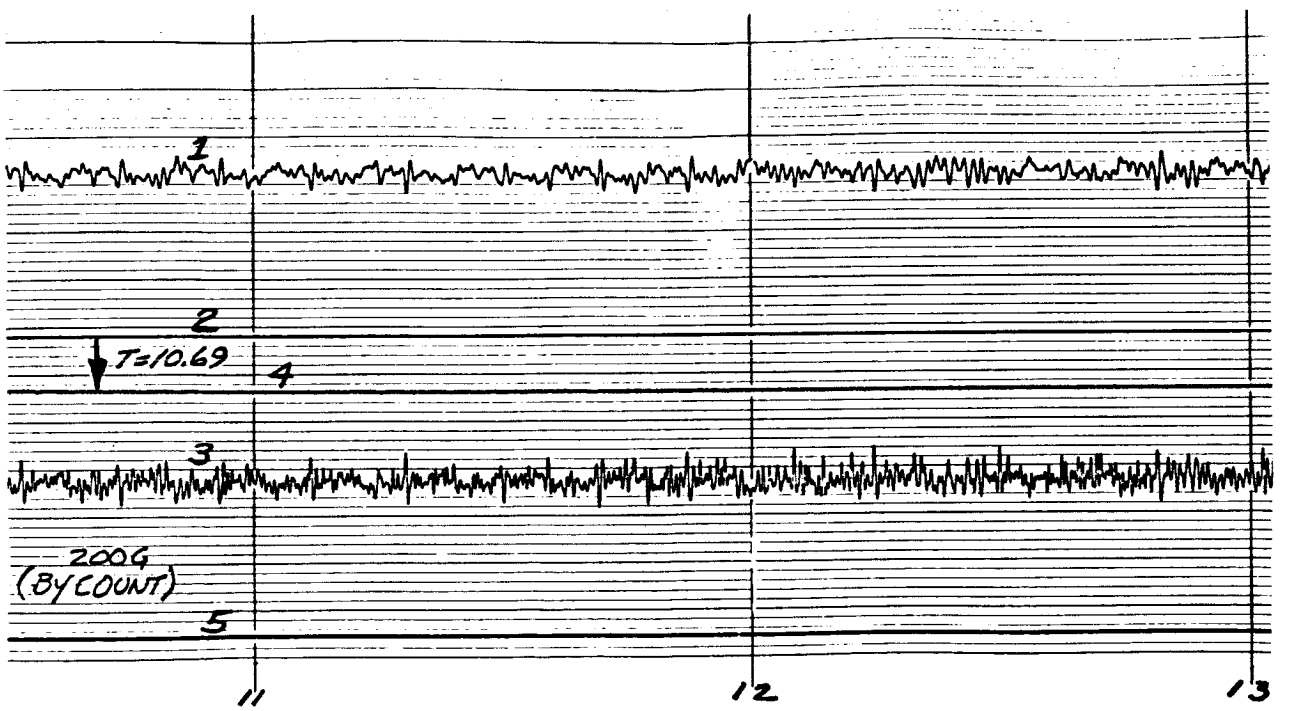


FIGURE 88 (B). ANIMAL NO. 242.
PHASE V - INITIAL DWELL, 11-13 SECONDS

Figure 89 (A). Animal No. 241 - Phase VI - Dwell, 13-22 Seconds

Areas of questionable electrocardiographic activity. Muscle tremor noise continues to obscure record. Probable QRS complexes after 16, 19 and 21 seconds of elapsed run time.

Figure 89 (B). Animal No. 242 - Phase VI - Dwell, 16-18 Seconds

Areas of questionable ECG activity. Muscle tremor noise continues to obscure record.

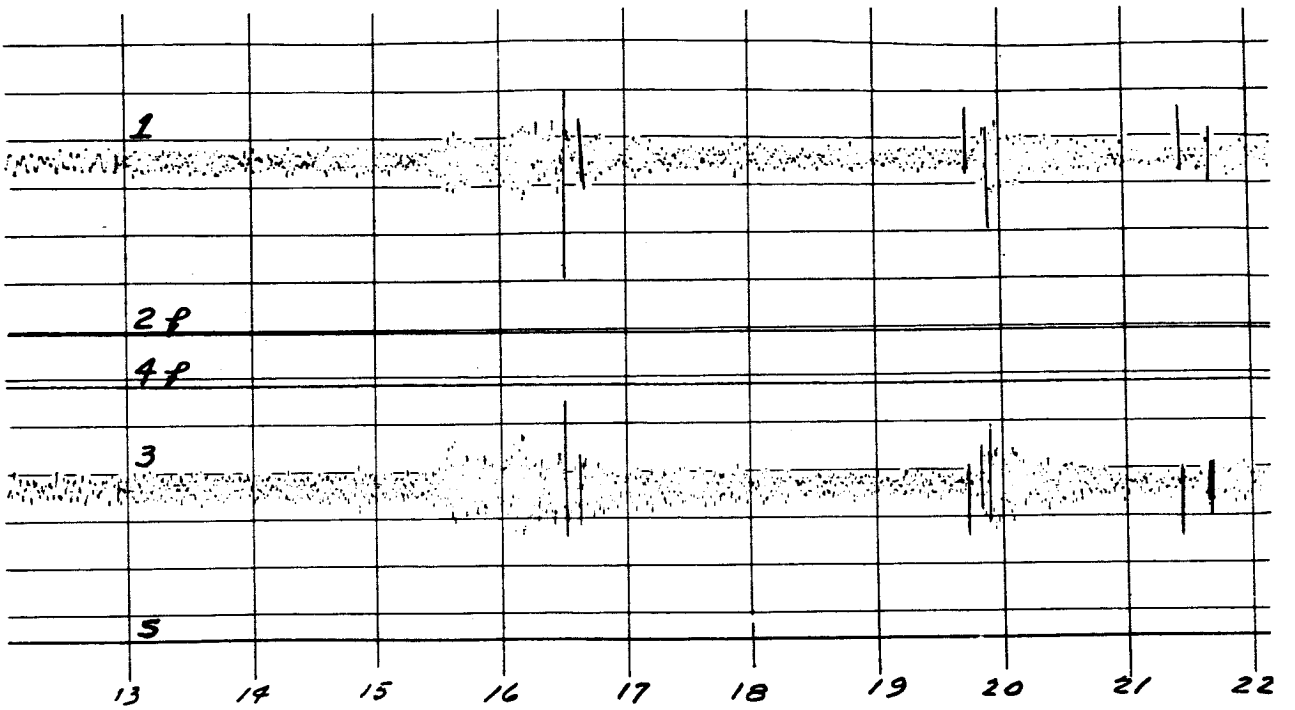


FIGURE 89 (A). ANIMAL NO. 241. PHASE VI - DWELL, 13-22 SECONDS

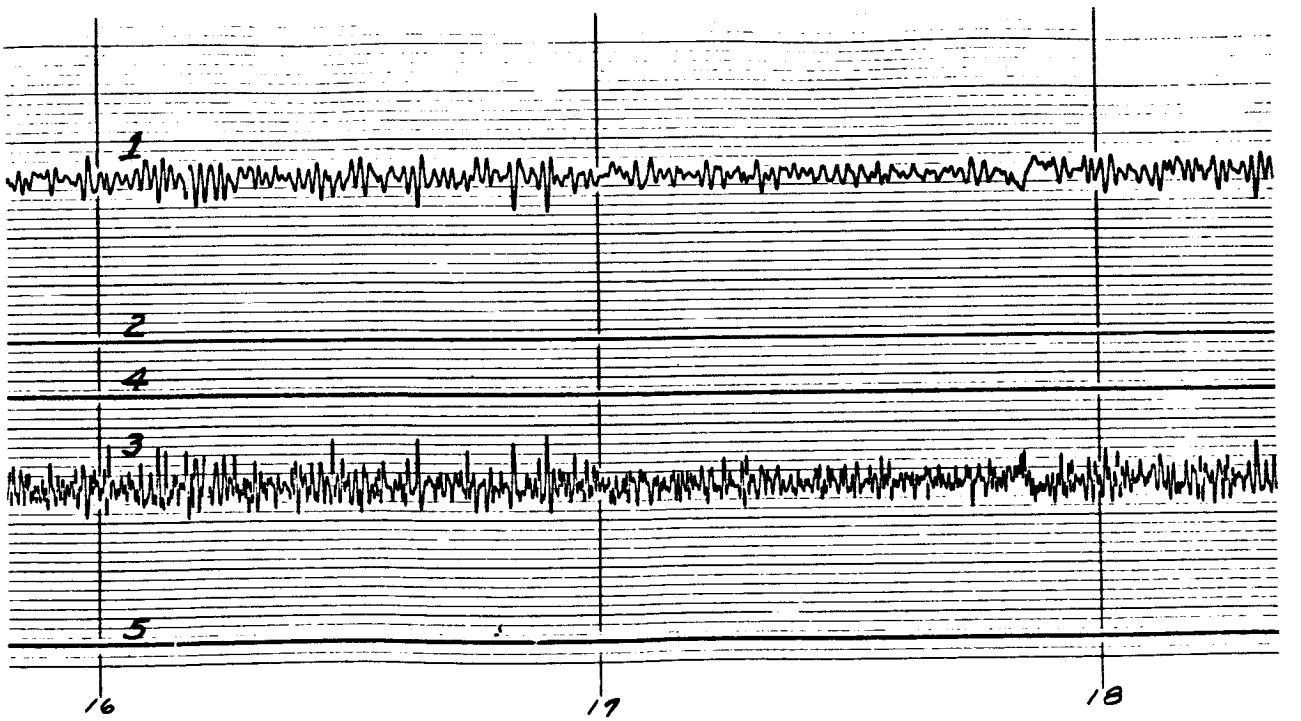


FIGURE 89 (B). ANIMAL NO. 242. PHASE VI - DWELL, 16-18 SECONDS

Figure 90 (A). Animal No. 241 - Phase VI - Dwell, 21-30 Seconds

There is a definite return of QRS activity. Heart rate is approximately 120/min. Peak to peak QRS amplitude and duration varies. There is a definite shift in QRS configuration. Paper speed is 25 mm/sec.

Figure 90 (B). Animal No. 242 - Phase VI - Dwell, 21-23 Seconds

Paper speed is 100 mm/sec. QRS complexes still largely obscured by noise. Heart rate appears to be 120/min. by comparison of complexes of Trace No. 1 and Trace No. 3. P wave is not discernable.

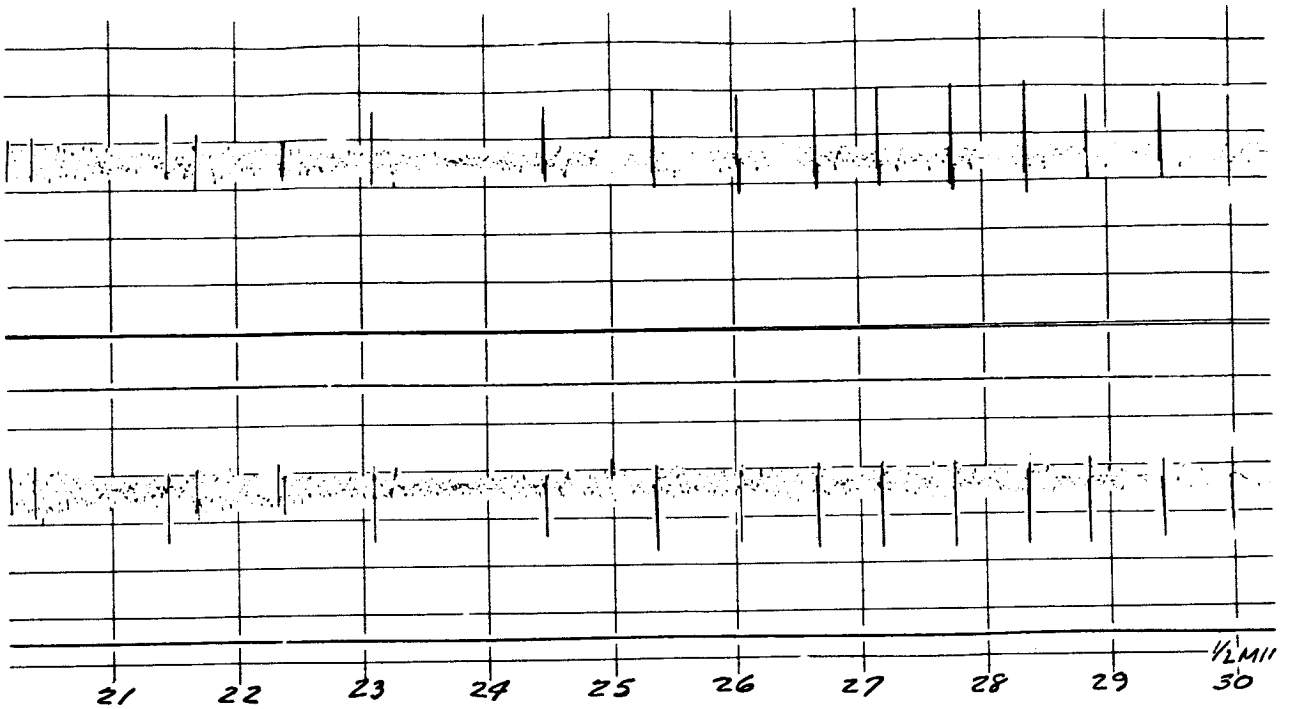


FIGURE 90 (A). ANIMAL NO. 241. PHASE VI - DWELL, 21-30 SECONDS

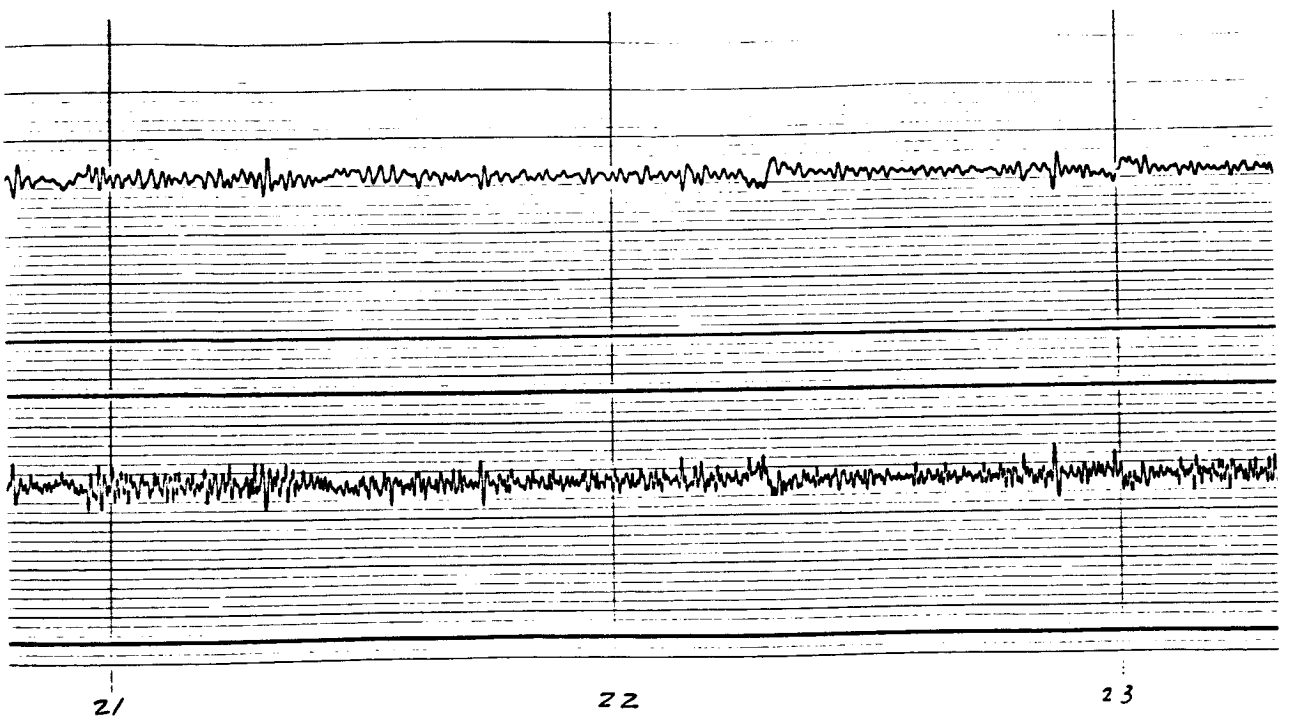


FIGURE 90 (B). ANIMAL NO. 242. PHASE VI - DWELL, 21-23 SECONDS

Figure 91. Animal No. 242 - Phase VI - Dwell, 26-30 Seconds

Sinus arrythmia is present. Heart rate is 120/min. There is a question here if the origin of the QRS is nodal. R waves are marked by arrows. The paper speed here is 50 mm/sec. and this figure is presented separately to complete the comparison with figure 90A preceeding.

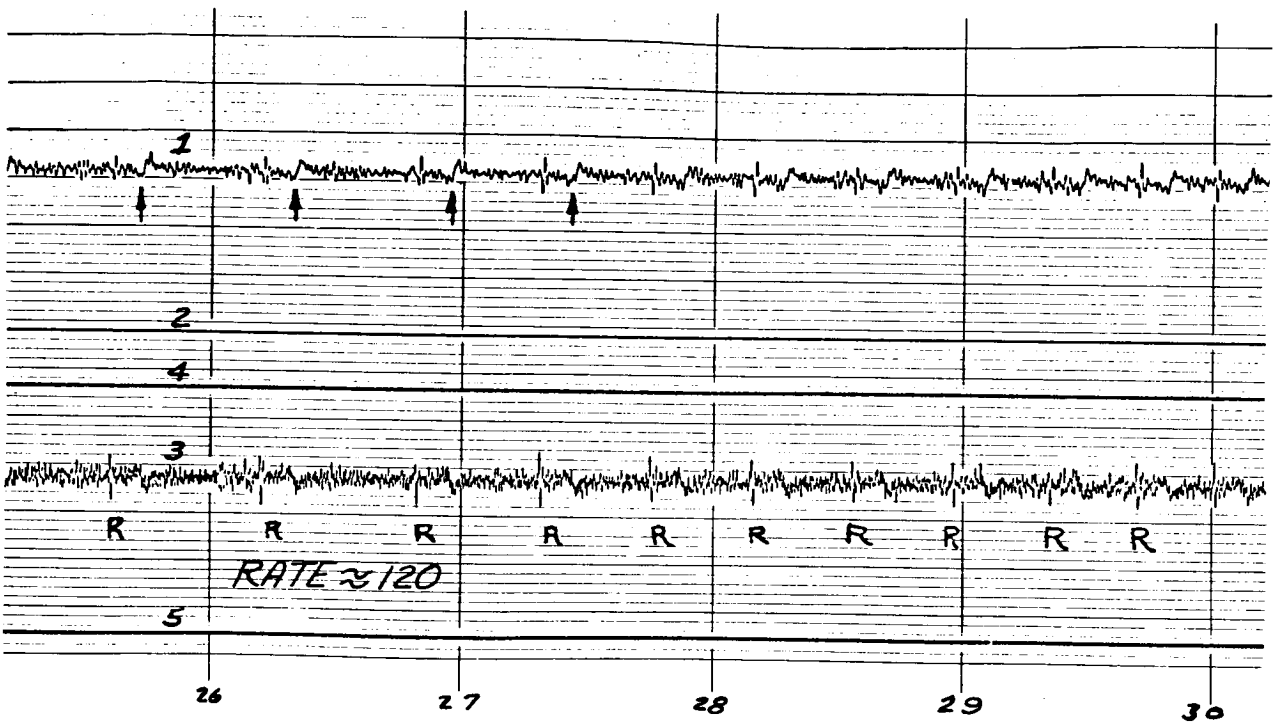


FIGURE 91. ANIMAL NO. 242. PHASE VI - DWELL, 26-30 SECONDS

Figure 92 (A). Animal No. 241 - Phase VI - Dwell, 30-38 Seconds

Heart rate fluctuating between 150 and 180/min. QRS excursion measures 9 units (18 mm).

Figure 92 (B). Animal No. 242 - Phase VI - 30-34 Seconds

Heart rate is 240/min. QRS voltage has diminished to 5½ units (11 mm).

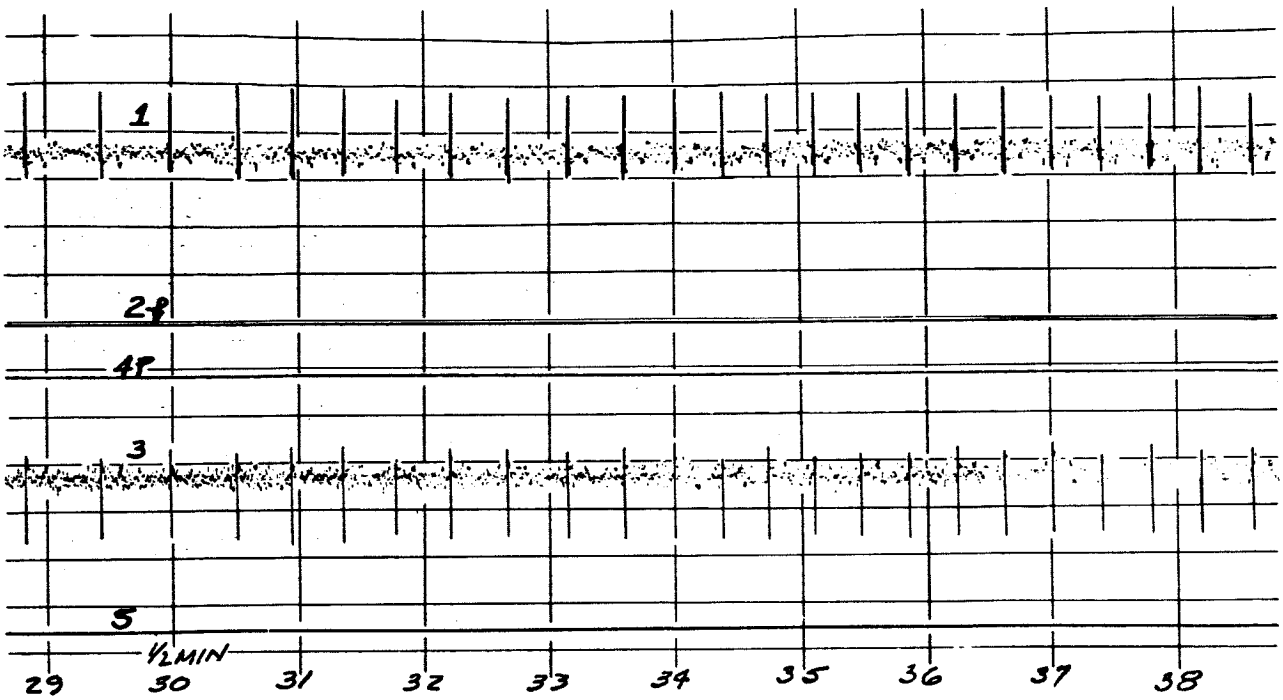


FIGURE 92 (A). ANIMAL NO. 241. PHASE VI - DWELL, 30-38 SECONDS

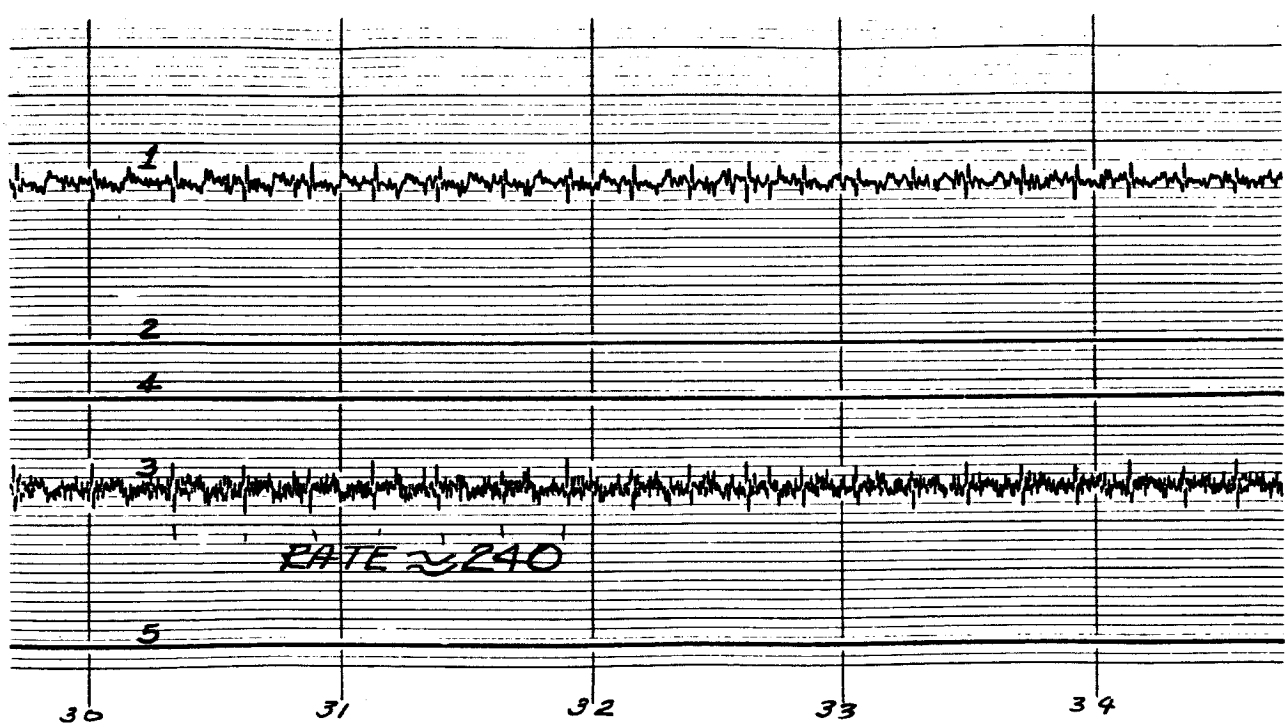


FIGURE 92 (B). ANIMAL NO. 242. PHASE VI - 30-34 SECONDS

Figure 93 (A). Animal No. 241 - Phase VI - Dwell, 42-51 Seconds

The QRS amplitude averages seven units (14 mm). The wave form is V_2 in type. Peak to peak QRS amplitude varies. Heart rate is 180/min.

Figure 93 (B). Animal No. 242 - Phase VI - Dwell, 44-48 Seconds

The QRS amplitude averages seven units (14 mm). The wave form is V_3 in type. Peak to peak QRS voltage varies. This is compatible with thoracic movement. Arrows point to extrasystole events. Heart rate appears to be 300 to 360/min.

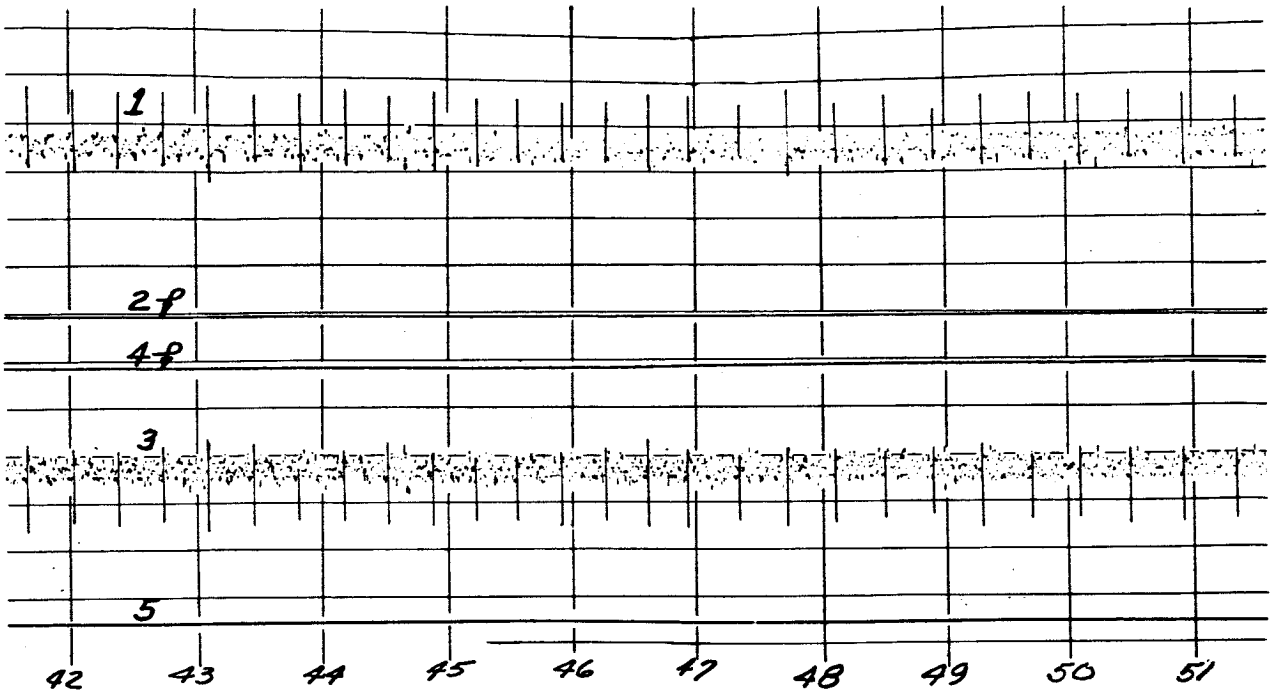


FIGURE 93 (A). ANIMAL NO. 241. PHASE VI - DWELL, 42-51 SECONDS

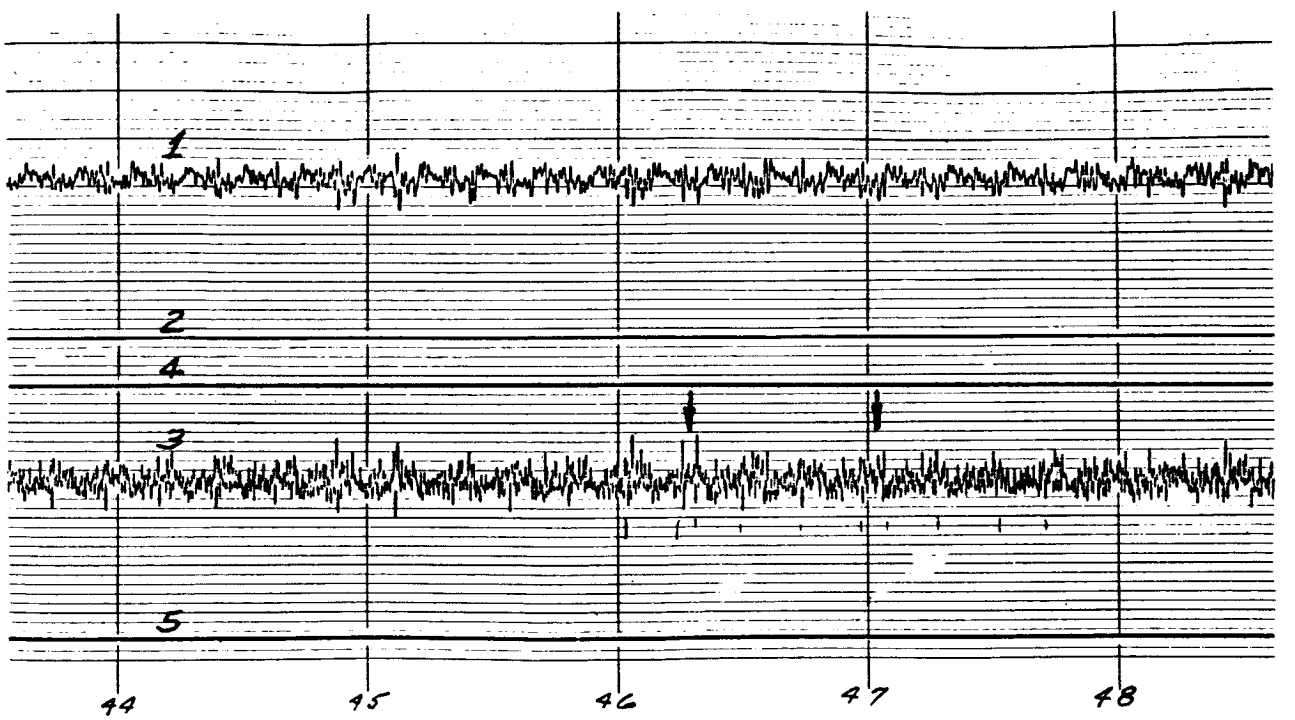


FIGURE 93 (B). ANIMAL NO. 242. PHASE VI - DWELL, 44-48 SECONDS

Figure 94 (A). Animal No. 241 - Phase VI - Dwell, 48-57 Seconds

Heart rate is 145/min. Muscle tremor noise is lessening, but P wave is still obscured.

Figure 94 (B). Animal No. 242 - Phase VI - Dwell, 56-60 Seconds

Heart rate is 100/min. Muscle tremor noise is lessening. Very difficult to identify the P wave.

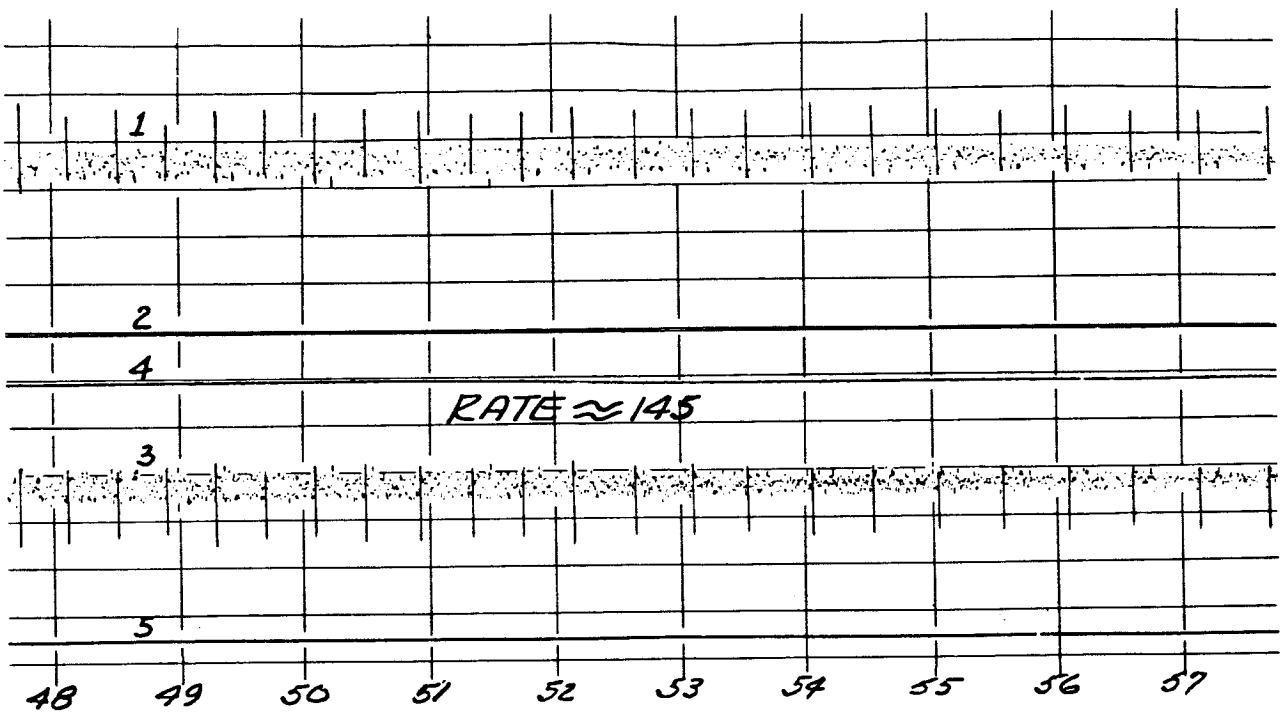


FIGURE 94 (A). ANIMAL NO. 241. PHASE VI - DWELL, 48-57 SECONDS

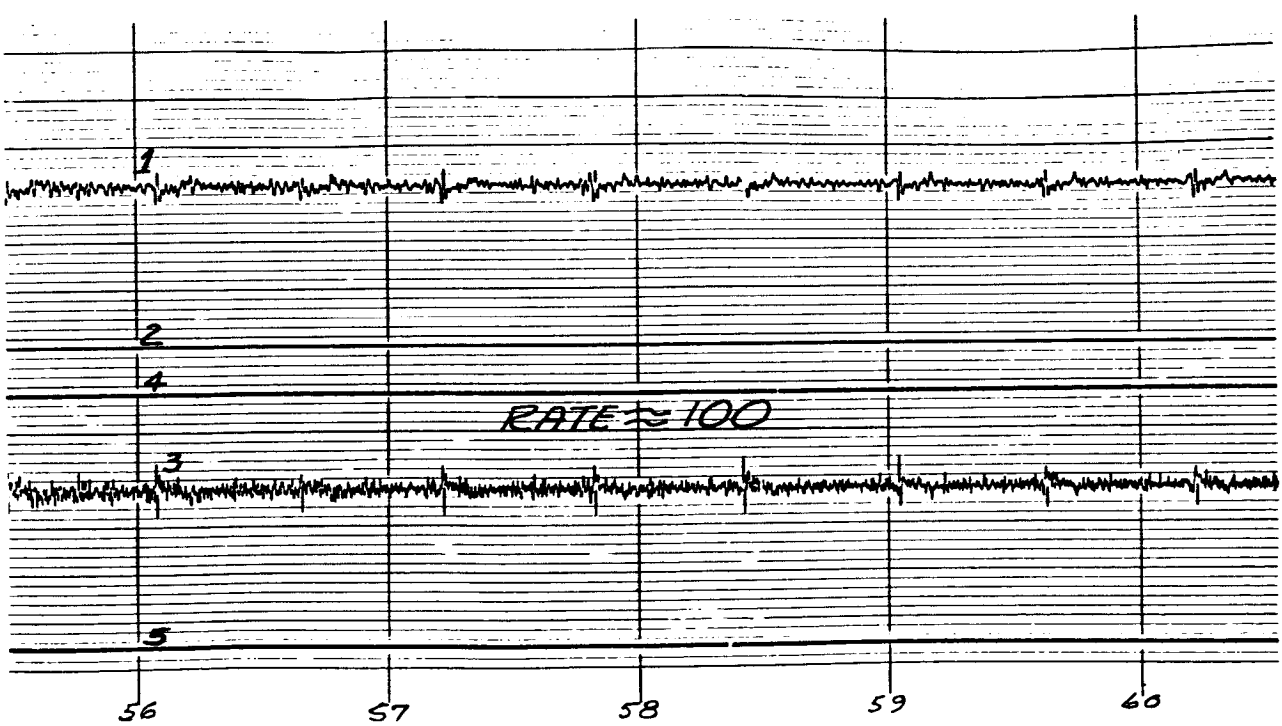


FIGURE 94 (B). ANIMAL NO. 242. PHASE VI - DWELL, 56-60 SECONDS

Figure 95 (A). Animal No. 241 - Phase VI - Dwell, 61-71 Seconds

Noise is definitely less. P wave is distinguishable. T wave is peaked. QRS amplitude measures 6½ units.

Figure 95 (B). Animal No. 242 - Phase VI - Dwell, 65-69 Seconds

Noise is definitely less. P wave is distinguishable and appears slightly diphasic. T wave appears inverted and slightly diphasic. QRS amplitude measures 5 units.

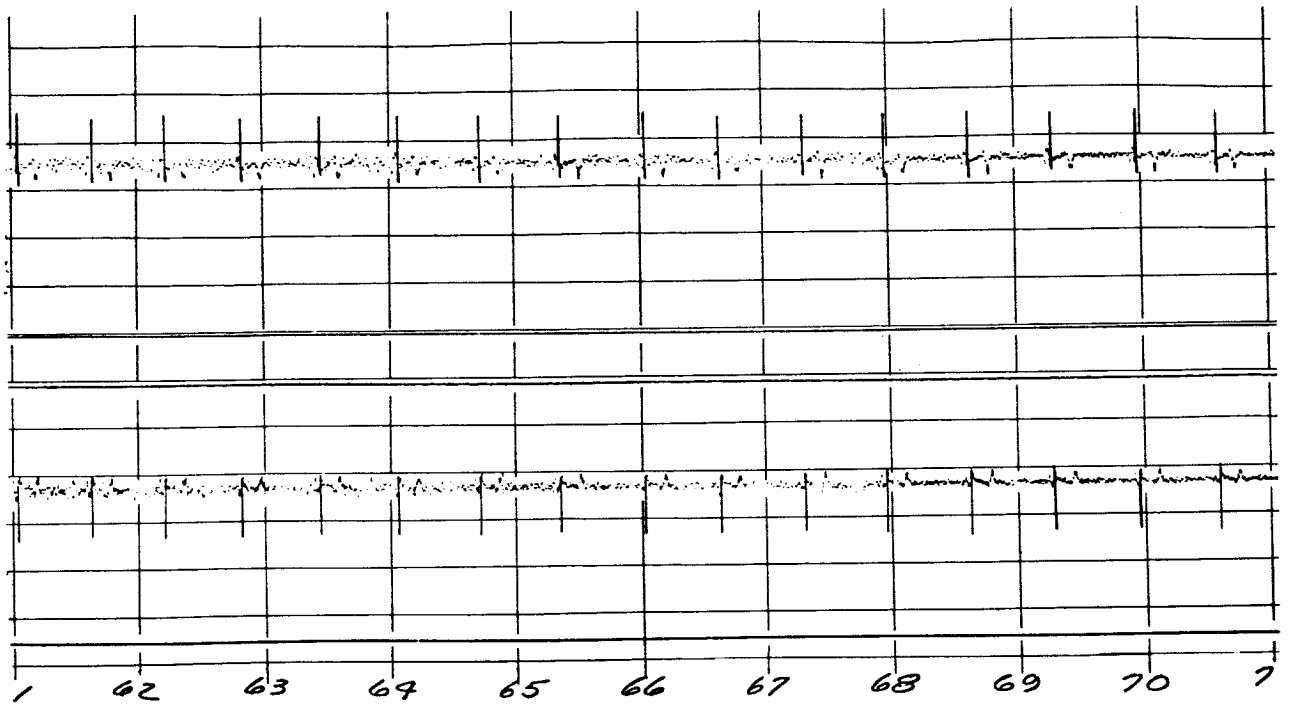


FIGURE 95 (A). ANIMAL NO. 241. PHASE VI - DWELL, 61-71 SECONDS

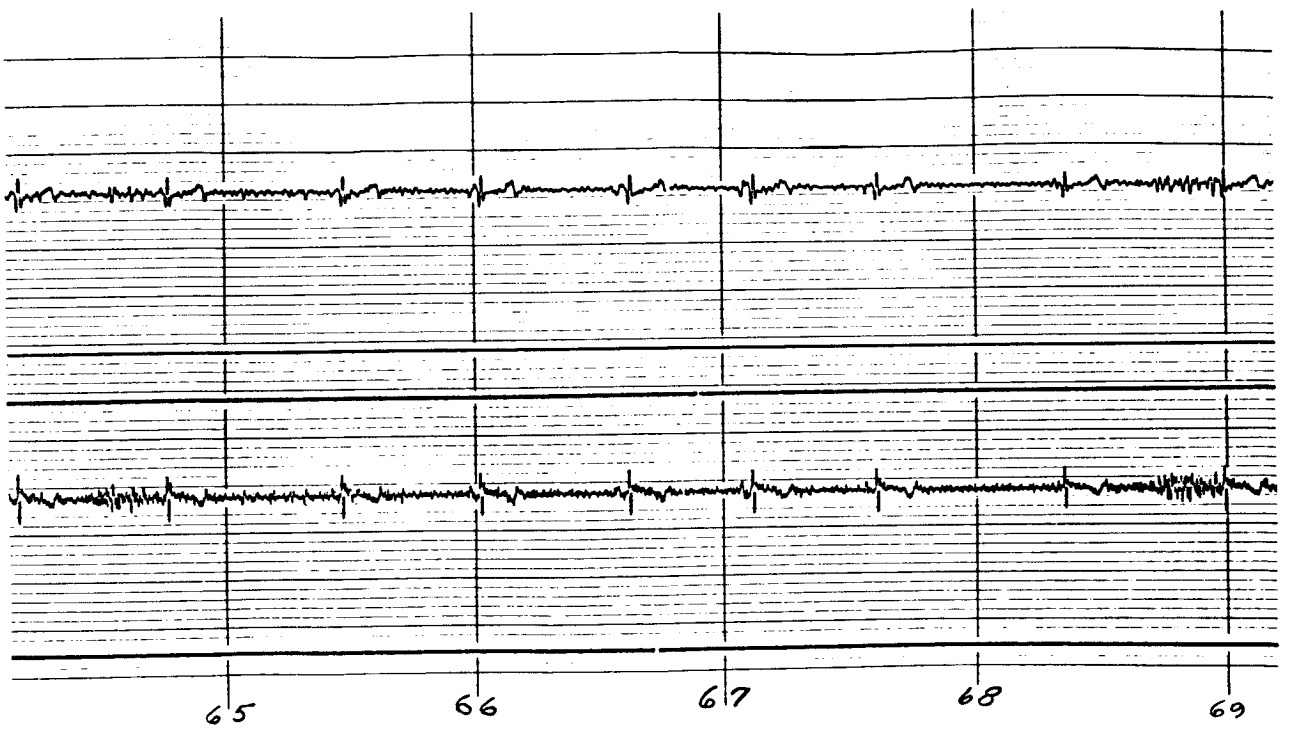


FIGURE 95 (B). ANIMAL NO. 242. PHASE VI - DWELL, 65-69 SECONDS

Figure 96 (A). Animal No. 241 - Phase VI - Dwell, 70-79 Seconds

The trace shows sinus rhythm and alternating QRS patterns with a peaked T wave. The heart rate is 120/min. Peak to peak QRS amplitude varies.

Figure 96 (B). Animal No. 242 - Phase VI - Dwell, 72-76 Seconds

The QRS complex holds rather steady at 5 units (10 mm). P waves are indicated on the trace. The left arrow points to an extrasystole, the remaining three arrows point to areas of sinus arrest. Rate is 90/min. A 2nd degree heart block is present. There is a suggestion of atrial tachycardia.

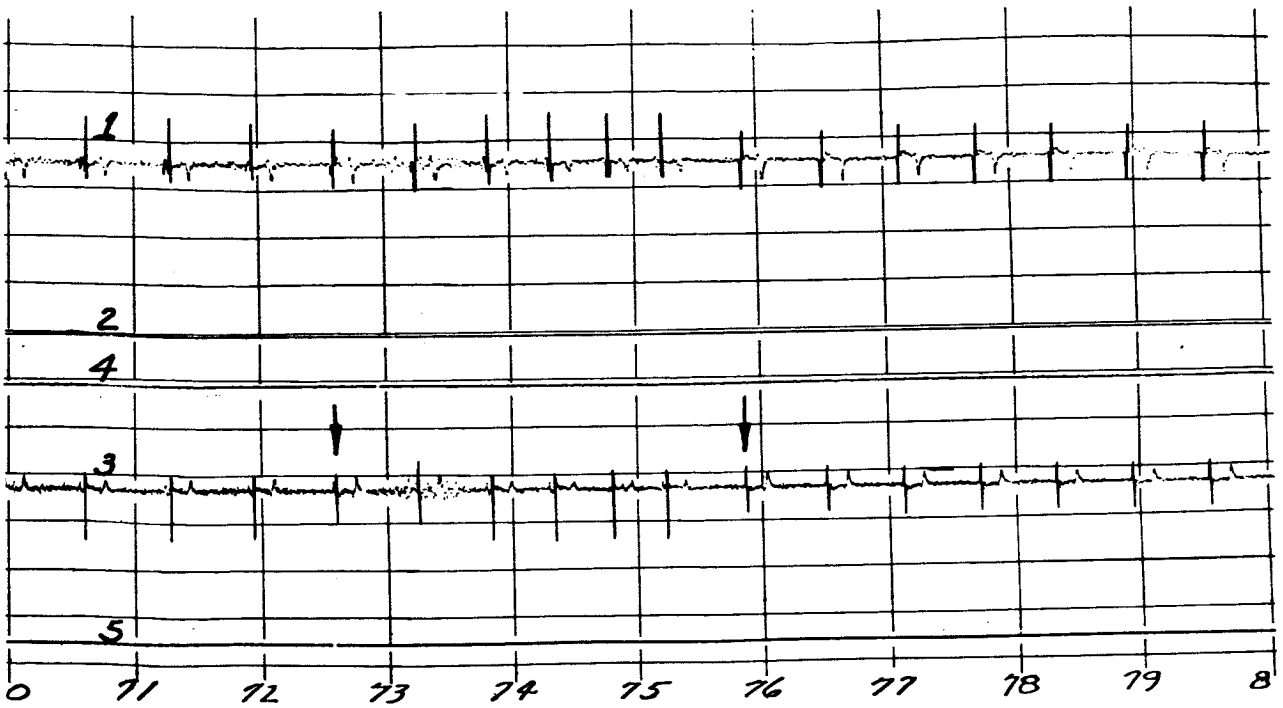


FIGURE 96 (A). ANIMAL NO. 241. PHASE VI - DWELL, 70-79 SECONDS

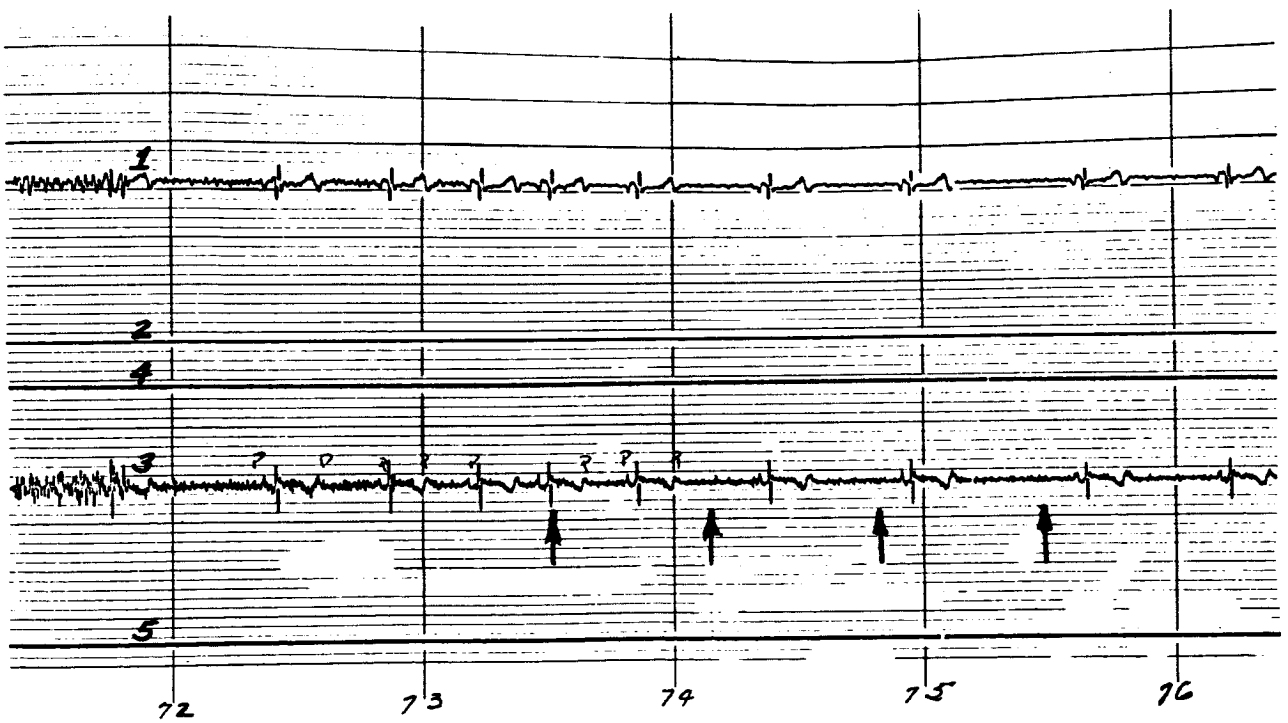


FIGURE 96 (B). ANIMAL NO. 242. PHASE VI - DWELL, 72-76 SECONDS

Figure 97 (A). Animal No. 241 -
Phase VI - Dwell, 84-93 Seconds - K_t Marker

Note marker at dwell time at which K_t is attained. The P wave is diphasic. QRS amplitude measures 9 units. The K_t for animal No. 241 was attained later in dwell because of its lesser weight. Note changing heart rates. Arrows point to changes in QRS voltage.

Figure 97 (B). Animal No. 242 -
Phase VI - Dwell, 76-80 Seconds - K_t Marker

Note marker on right showing attainment of K_t. QRS voltage is 5 units. There is an inverted diphasic T wave and a peaked P wave. Left hand arrows point to probable P wave. 2:1 block persists. Rate is 120/min.

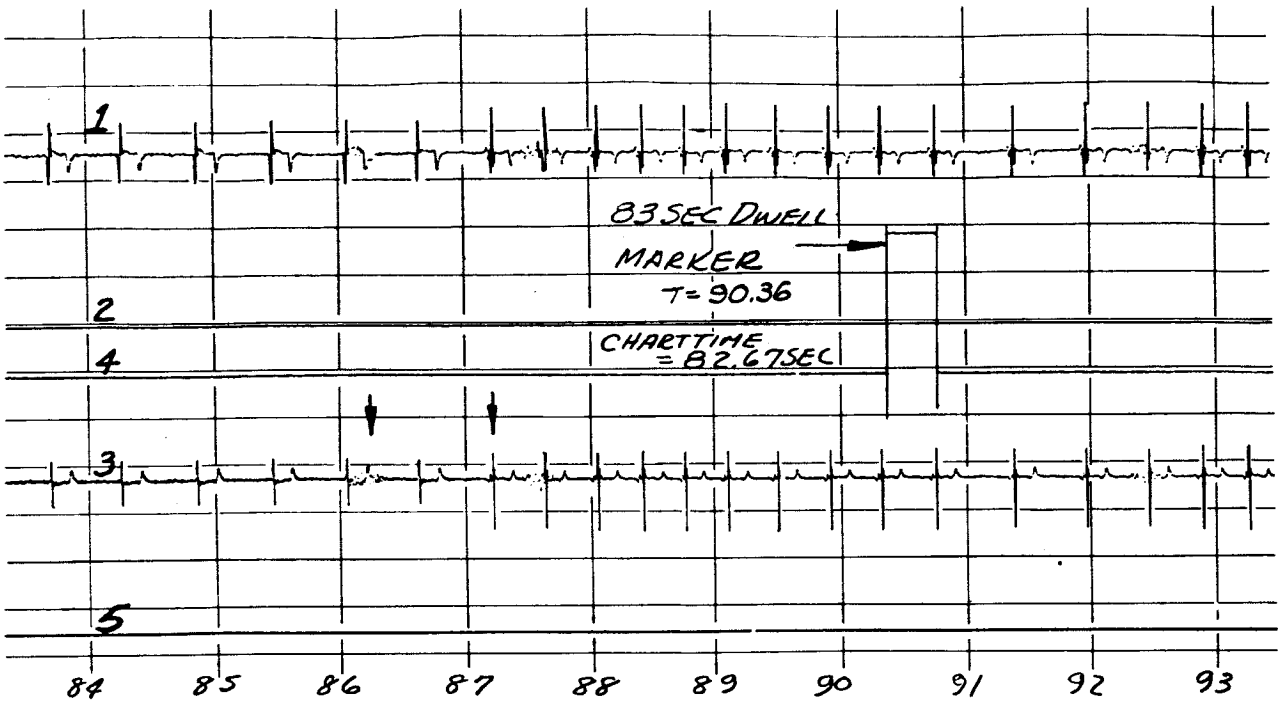


FIGURE 97 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 84-93 SECONDS - K_t MARKER

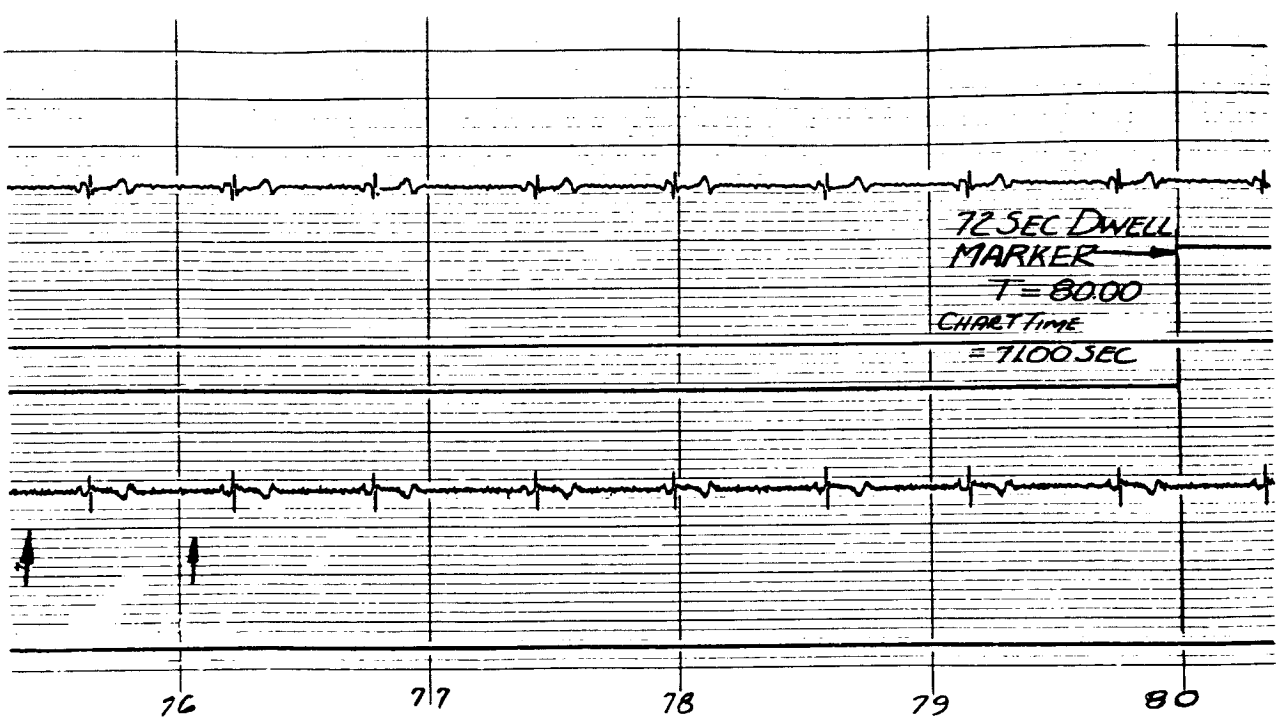


FIGURE 97 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 76-80 SECONDS - K_t MARKER

Figure 98 (A). Animal No. 241 - Phase VI - Dwell, 88-97 Seconds

K_t has been attained. Noise is virtually absent past K_t marker. QRS voltage is rather steady at 9 units. A small variance peak to peak is noticeable however.

Figure 98 (B). Animal No. 242 - Phase VI - Dwell, 80-84 Seconds

K_t has been attained. Again, noise is virtually absent. T wave is diphasic. The heart rate is 90/min. with a diphasic P wave.

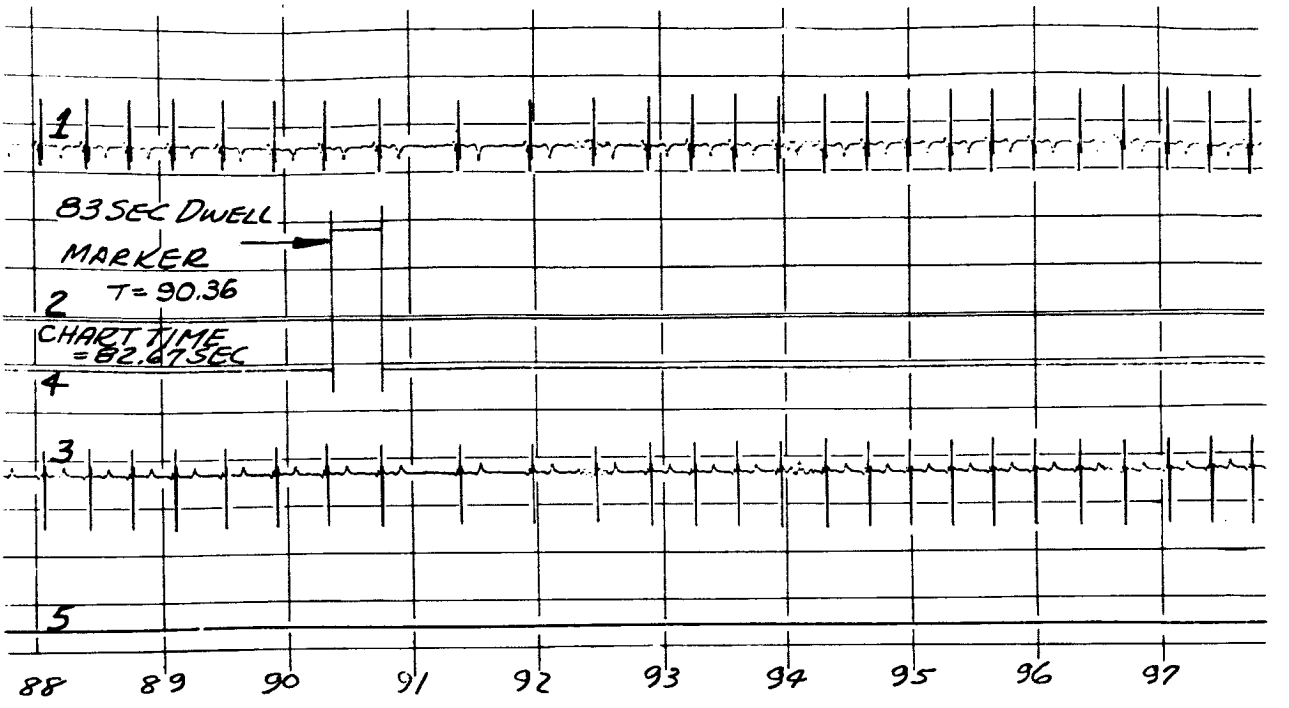


FIGURE 98 (A). ANIMAL NO. 241. PHASE VI - DWELL, 88-97 SECONDS

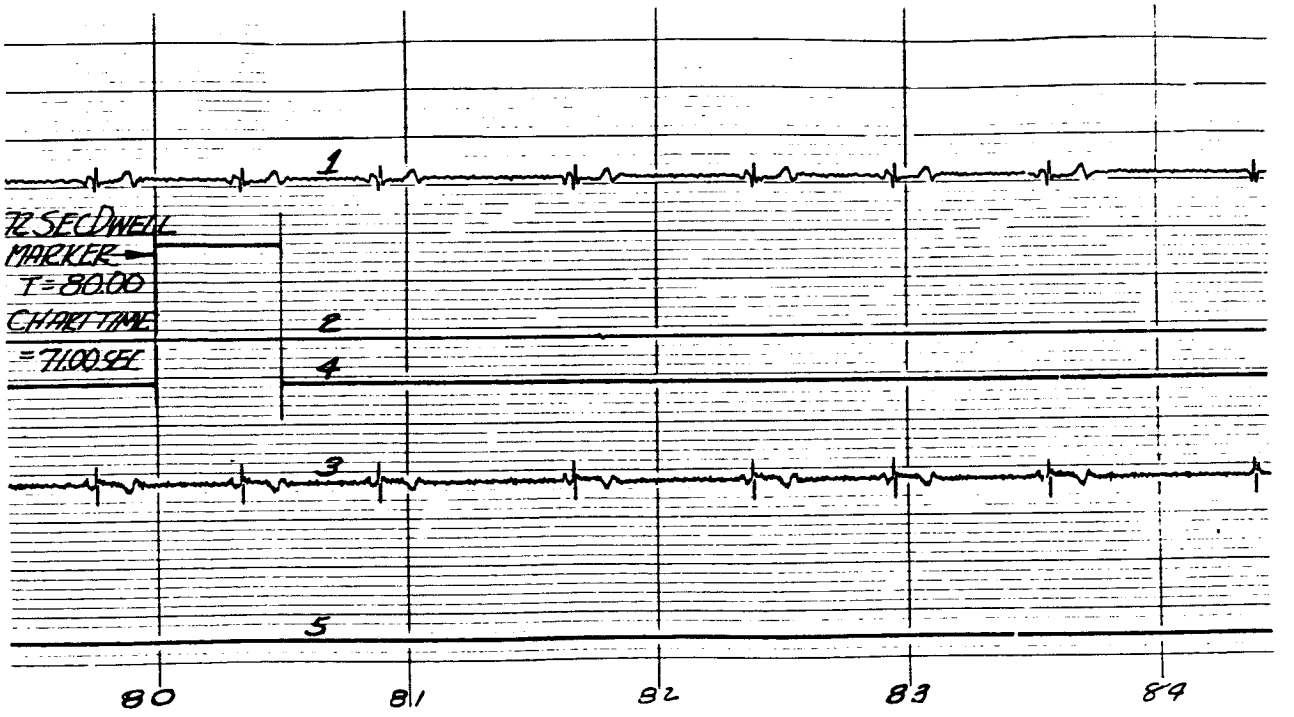


FIGURE 98 (B). ANIMAL NO. 242. PHASE VI - DWELL, 80-84 SECONDS

Figure 99 (A). Animal No. 241 - Phase VI - Dwell, 102-111 Seconds

Heart rate has come back strongly to 180/min. QRS amplitude is steady at 9 units. Note the diphasic T wave and definitely diphasic P wave. The difference between the strength of the QRS signal and that shown below is marked.

Figure 99 (B). Animal No. 242 - Phase VI - Dwell, 93-101 Seconds

A 2:1 A.V. block is demonstrated here. The heart rate varies from 90 to 120/min. QRS voltage has dropped to 4 units. Diphasic P wave and inverted T wave are in evidence. Slight elevation of ST segment is present.

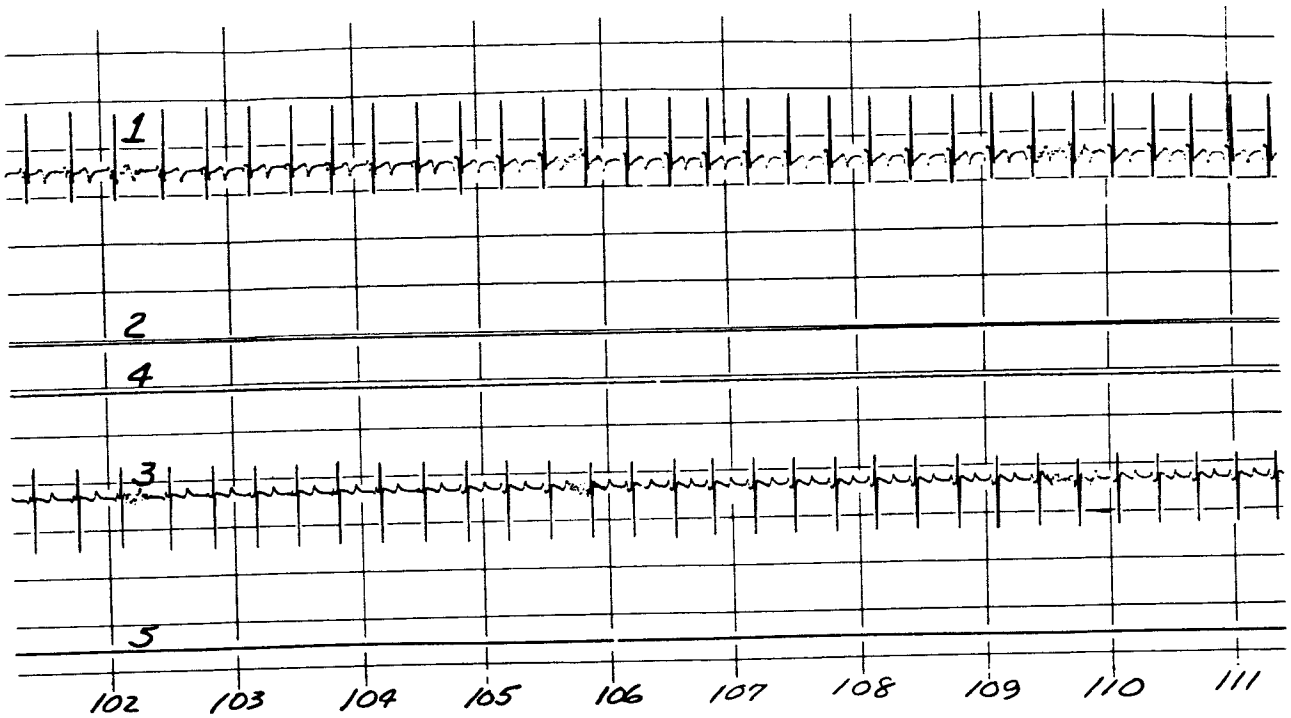


FIGURE 99 (A). ANIMAL NO. 241. PHASE VI - DWELL, 102-111 SECONDS

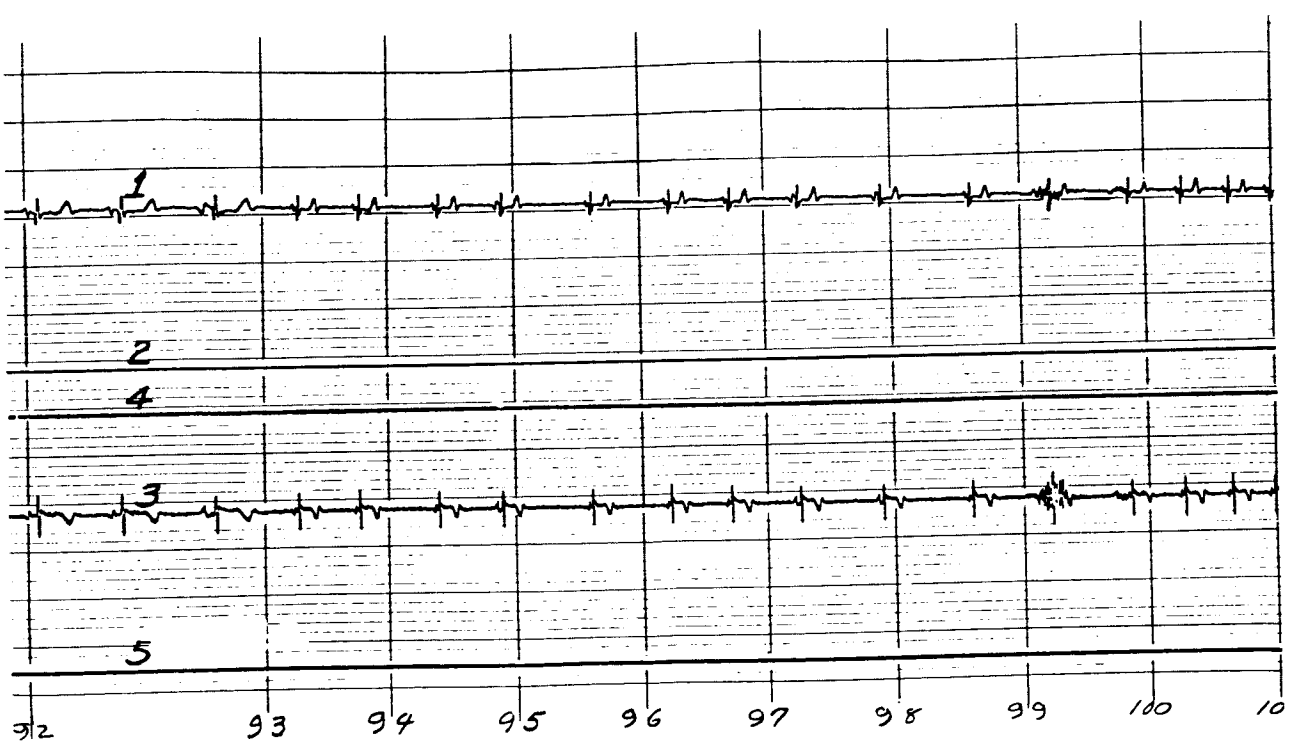


FIGURE 99 (B). ANIMAL NO. 242. PHASE VI - DWELL, 93-101 SECONDS

Figure 100 (A). Animal No. 241 -
Phase VI - Dwell, 106-115 Seconds

Heart rate is 180/min. Diphasic P and T waves are present. QRS is 7 units. There is same general exposure to stress as in the figure below.

Figure 100 (B). Animal No. 242 -
Phase VI - Dwell, 107-116 Seconds

In this trace ventricular extrasystole are indicated by the arrows. A 2:1 heart block is evident. Heart rate is 120/min. QRS measures 3 units. Developing pattern of myocardial ischemia and damage is present. Compare to figure above.

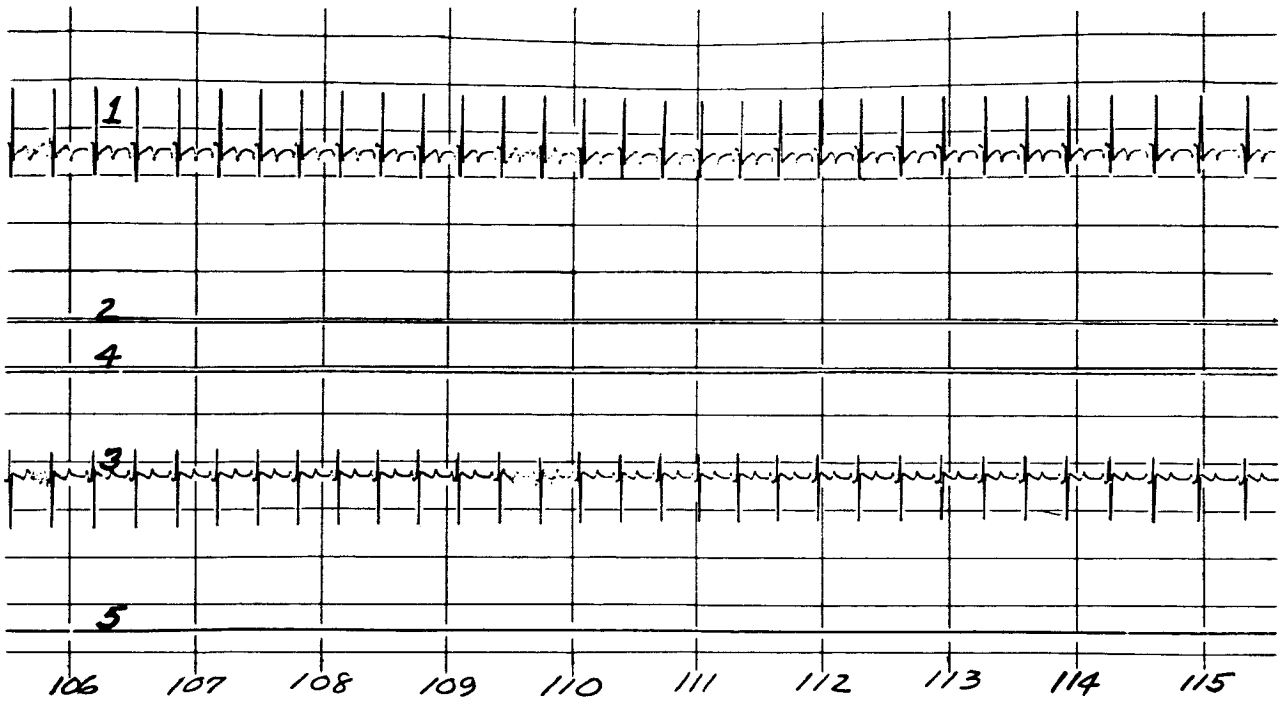


FIGURE 100 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 106-115 SECONDS

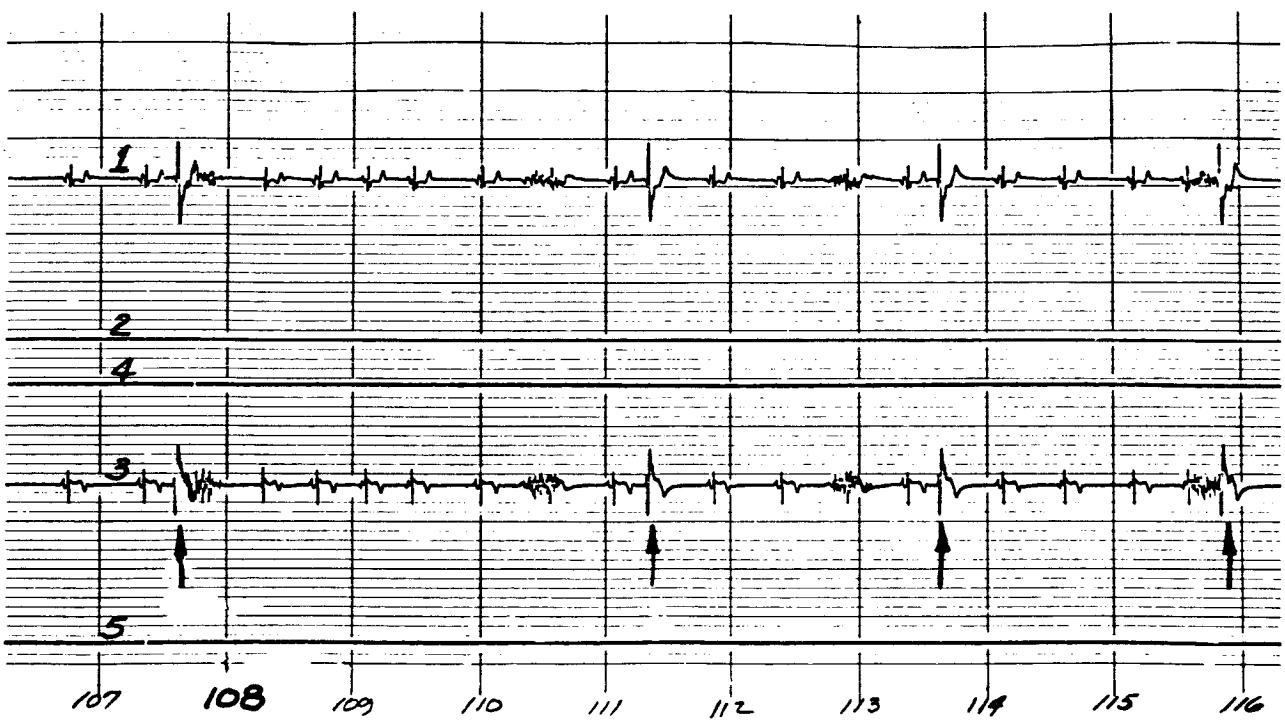


FIGURE 100 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 107-116 SECONDS

Figure 101 (A). Animal No. 241 -
Phase VI - Dwell, 112-121 Seconds

Heart rate changes abruptly to 120/min. at 119 seconds.

Figure 101 (B). Animal No. 242 -
Phase VI - Dwell, 111-120 Seconds

Pattern of myocardial ischemia and damage developing. Arrows point to P.V.C.

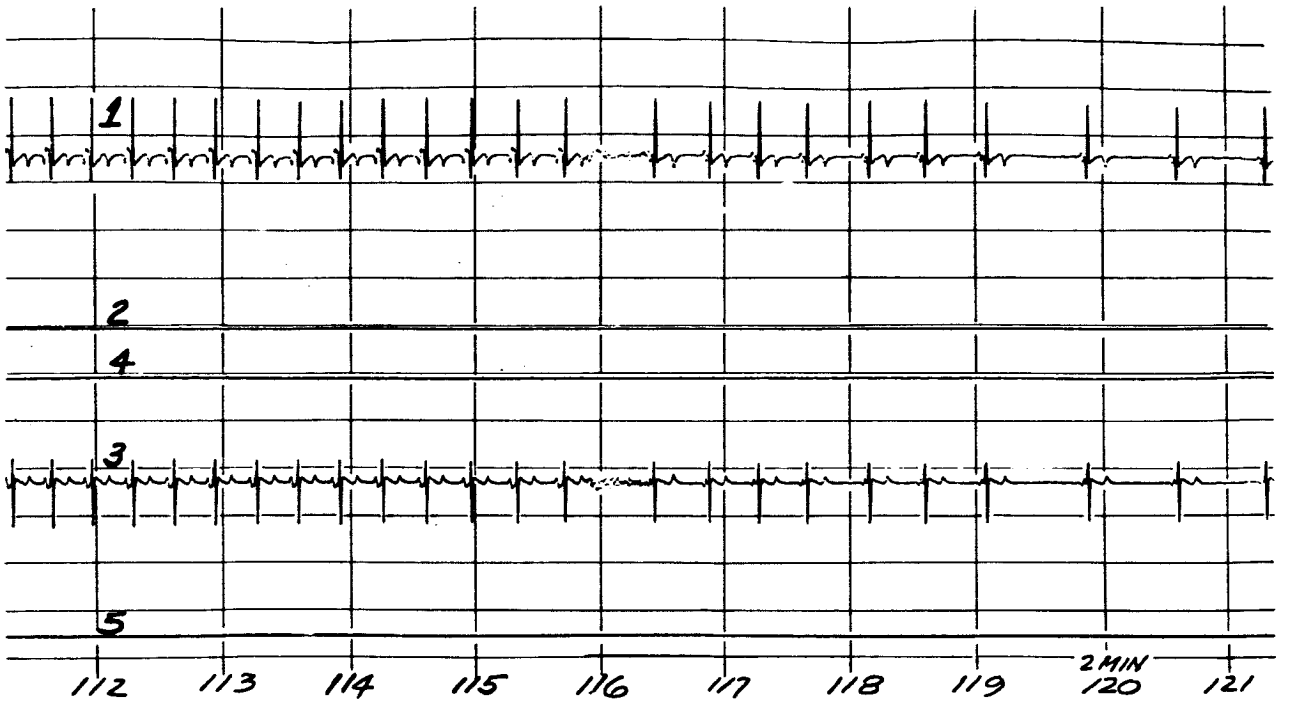


FIGURE 101 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 112-121 SECONDS

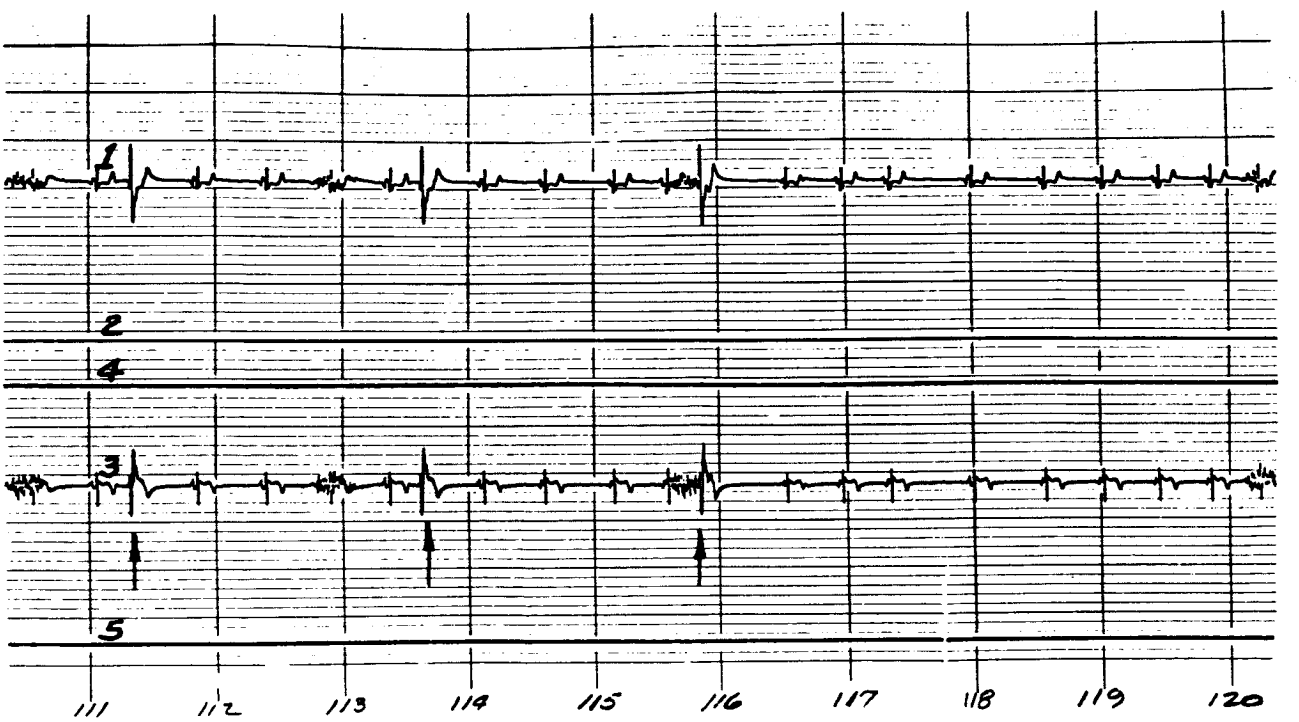


FIGURE 101 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 111-120 SECONDS

Figure 102 (A). Animal No. 241 -
Phase VI - Dwell, 118-127 Seconds

Heart rate is undergoing changes from 60 to 120/min. Average is 90/min. Coving of ST segment, diphasic P wave, and 2nd T wave are apparent.

Figure 102 (B). Animal No. 242 -
Phase VI - Dwell, 122-131 Seconds

A 2:1 heart block with periods of sinus arrest are seen. Two P.V.C. are indicated by the single arrow. Pattern of ischemia and damage is seen, as is a changing heart rate at the right.

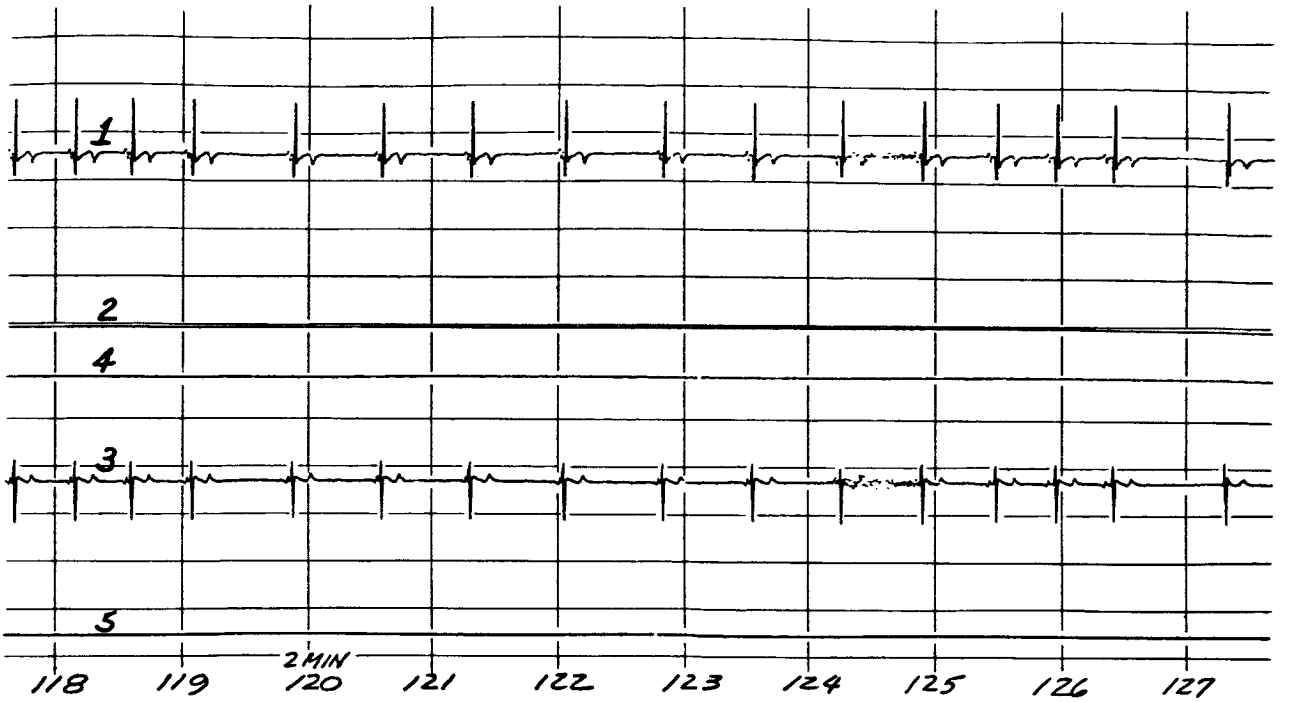


FIGURE 102 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 118-127 SECONDS

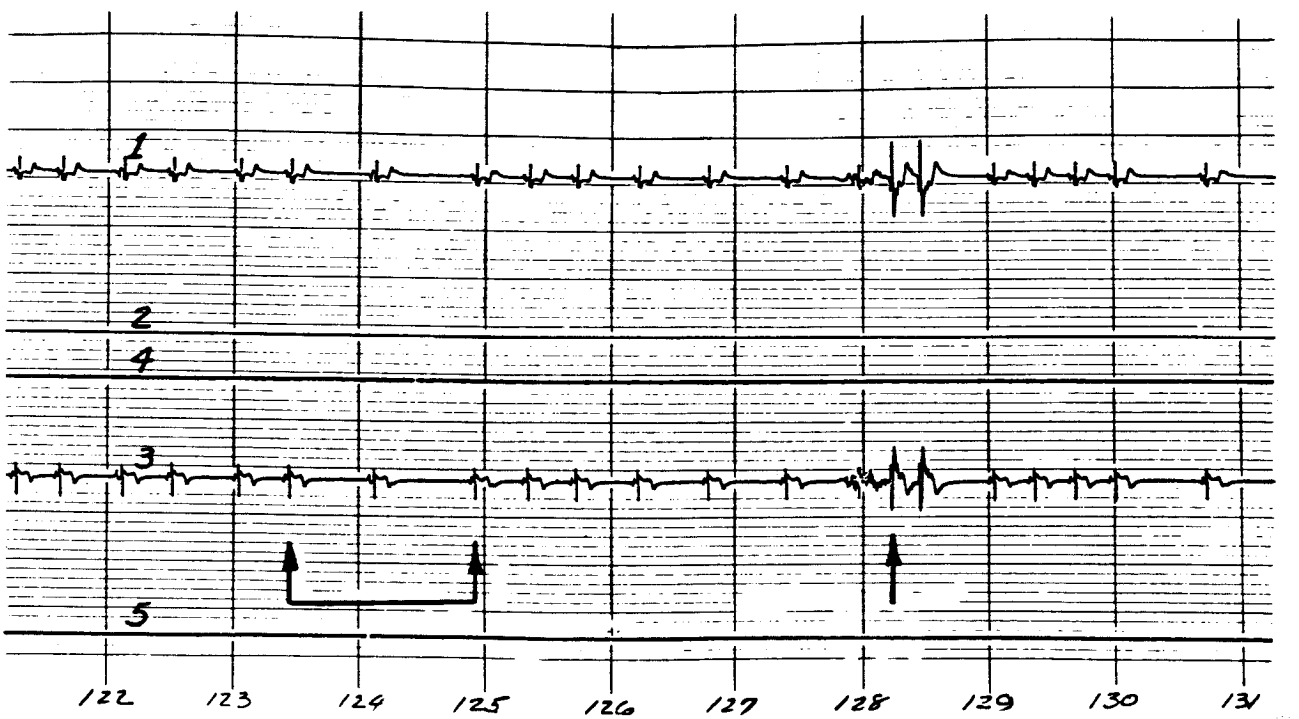


FIGURE 102 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 122-131 SECONDS

Figure 103 (A). Animal No. 241 -
Phase VI - Dwell, 152-161 Seconds

QRS voltage is slightly diminished. Heart rate is approximately 75/min.

Figure 103 (B). Animal No. 242 -
Phase VI - Dwell, 155-164 Seconds

A fully developed pattern of ischemia and damage is seen. Heart rate is increasing from 90 to 150/min.

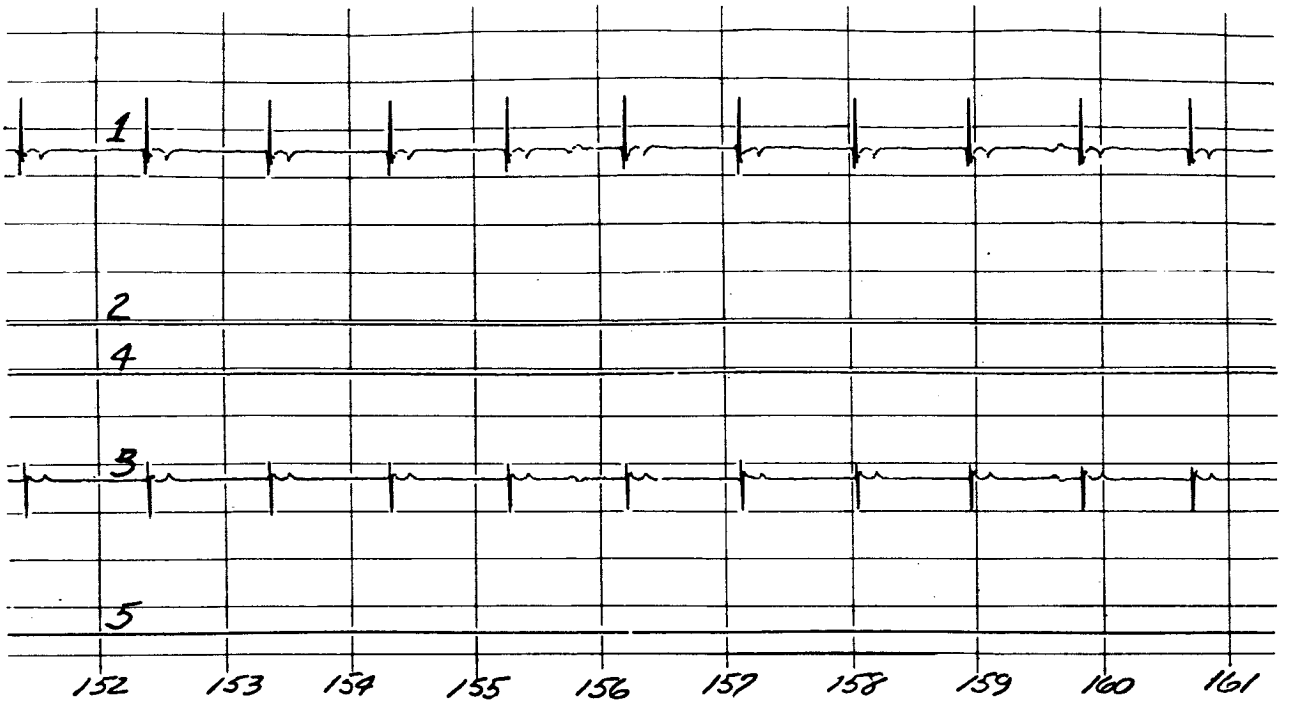


FIGURE 103 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 152-161 SECONDS

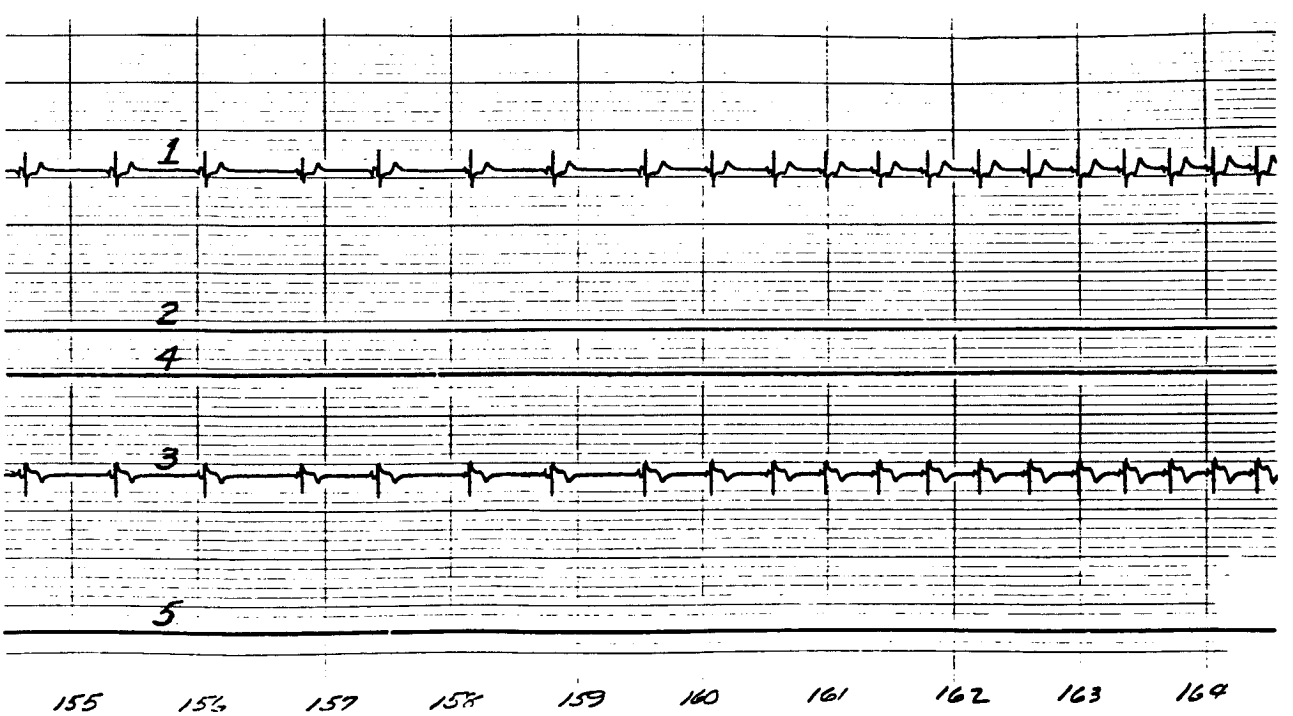


FIGURE 103 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 155-164 SECONDS

Figure 104 (A). Animal No. 241 -
Phase VI - Dwell, 164-173 Seconds

A 2:1 heart block is fully developed. Atrial rate is 120/min. and ventricular rate is 60/min.

Figure 104 (B). Animal No. 242 -
Phase VI - Dwell, 163-172 Seconds

Heart rate is steady at 180/min. Pattern of ischemia and injury is deepening. The small figure on the left is the classical pattern of ischemia (compare to Trace No. 143).

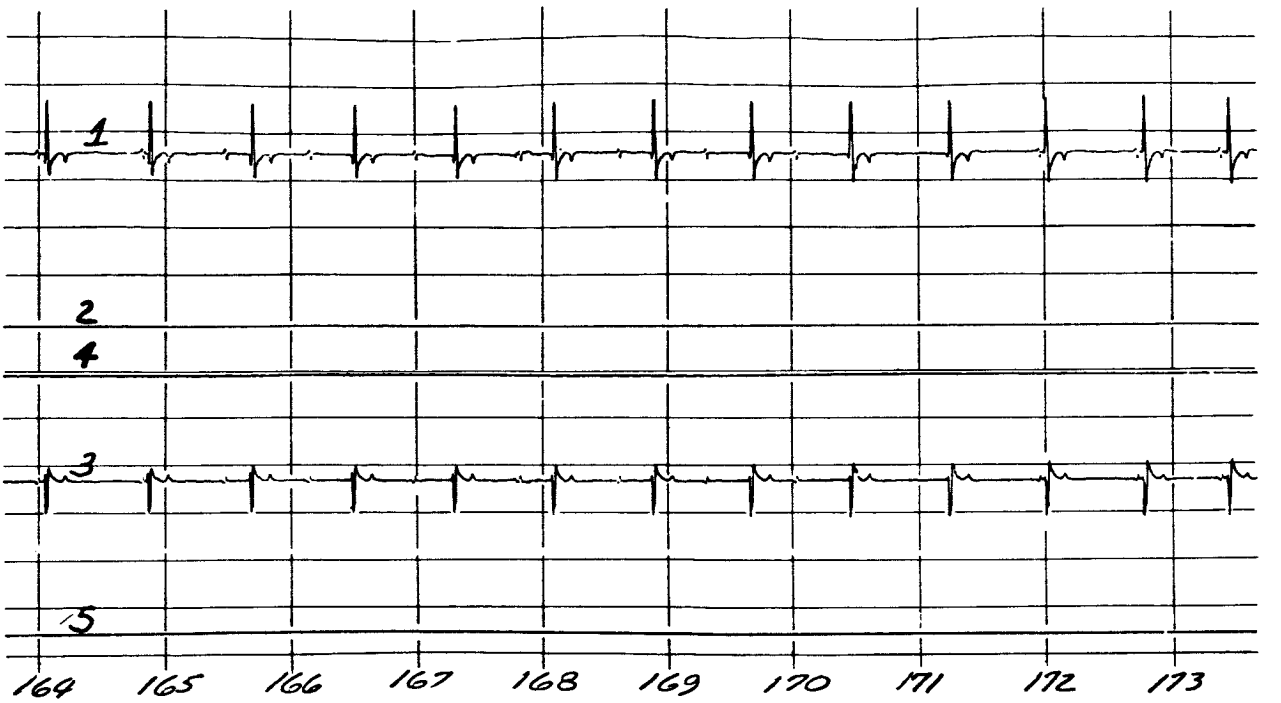


FIGURE 104 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 164-173 SECONDS

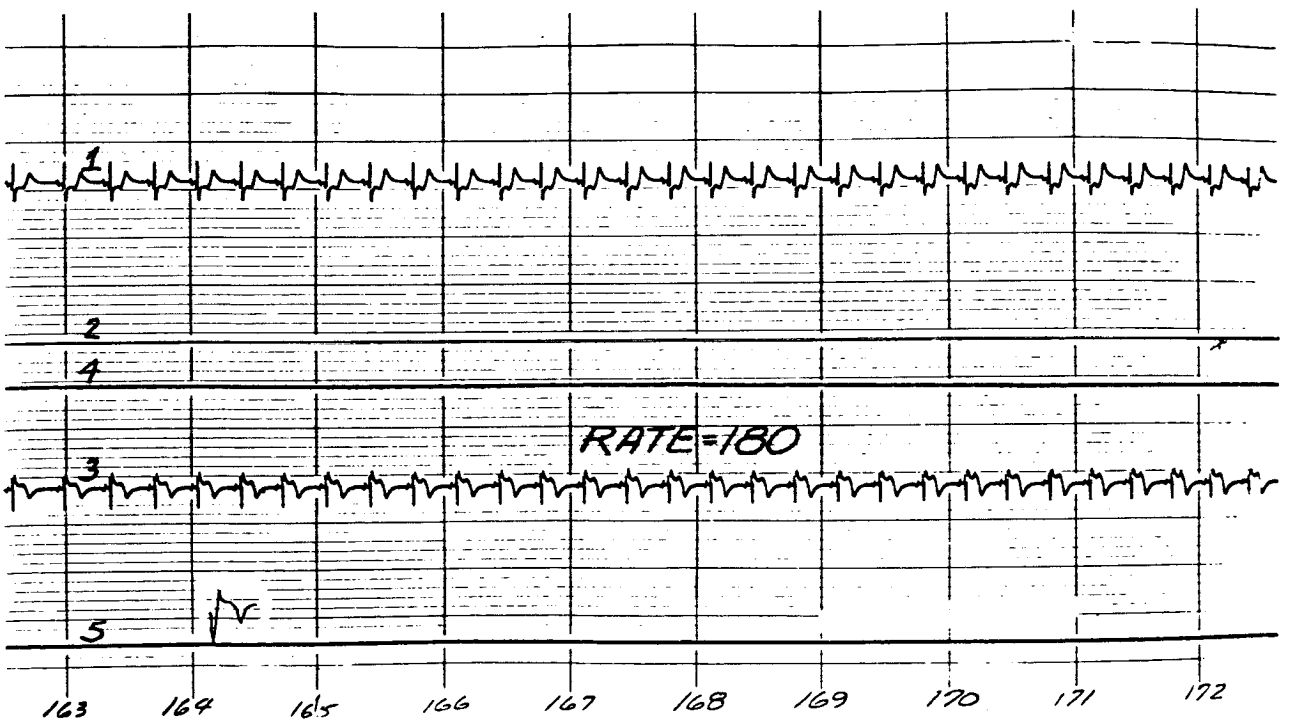


FIGURE 104 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 163-172 SECONDS

Figure 105 (A). Animal No. 241 -
Phase VI - Dwell, 179-188 Seconds

Heart rate is steady at 90/min. There is a definite elevation of ST segment and a widening of QRS duration. A merger of the QRS and T components seems to be taking place.

Figure 105 (B). Animal No. 242 -
Phase VI - Dwell, 187-196 Seconds

The amplitude of the QRS voltage is low with marked elevation of ST segment. In addition the T wave is inverted with periods of sinus arrest (at arrows). The heart rate is falling from 180 to 120/min.

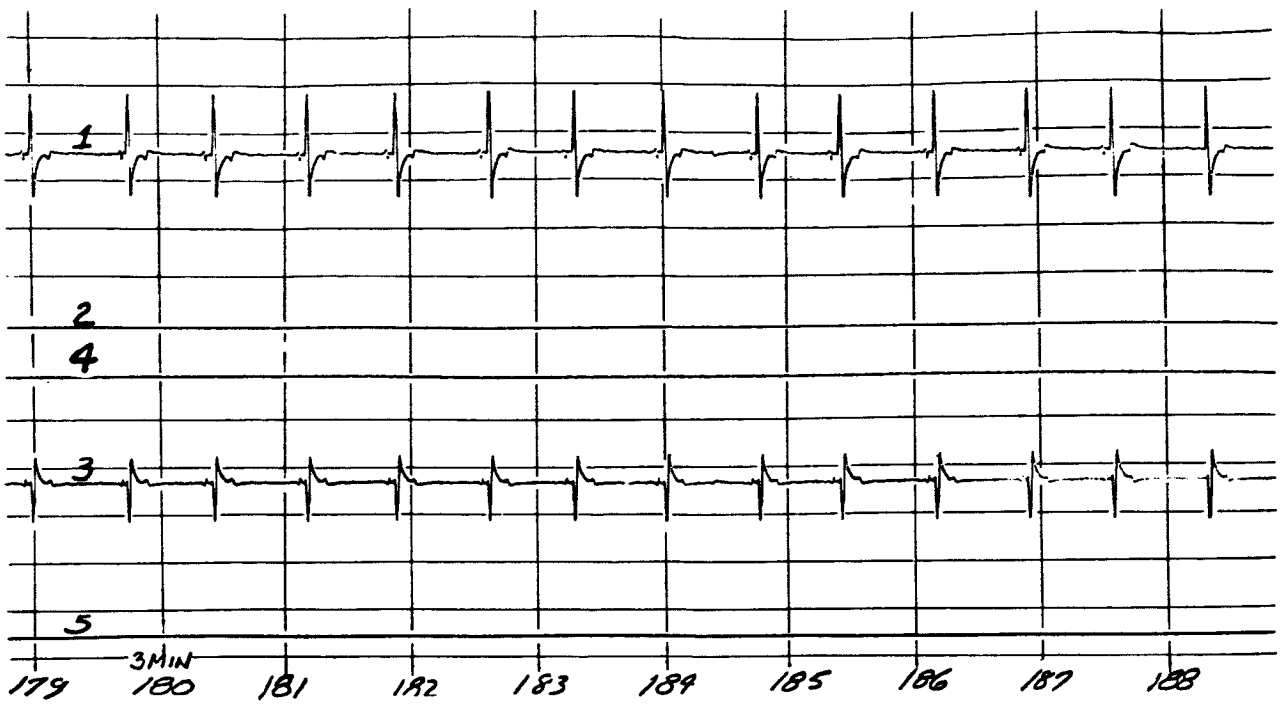


FIGURE 105 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 179-188 SECONDS

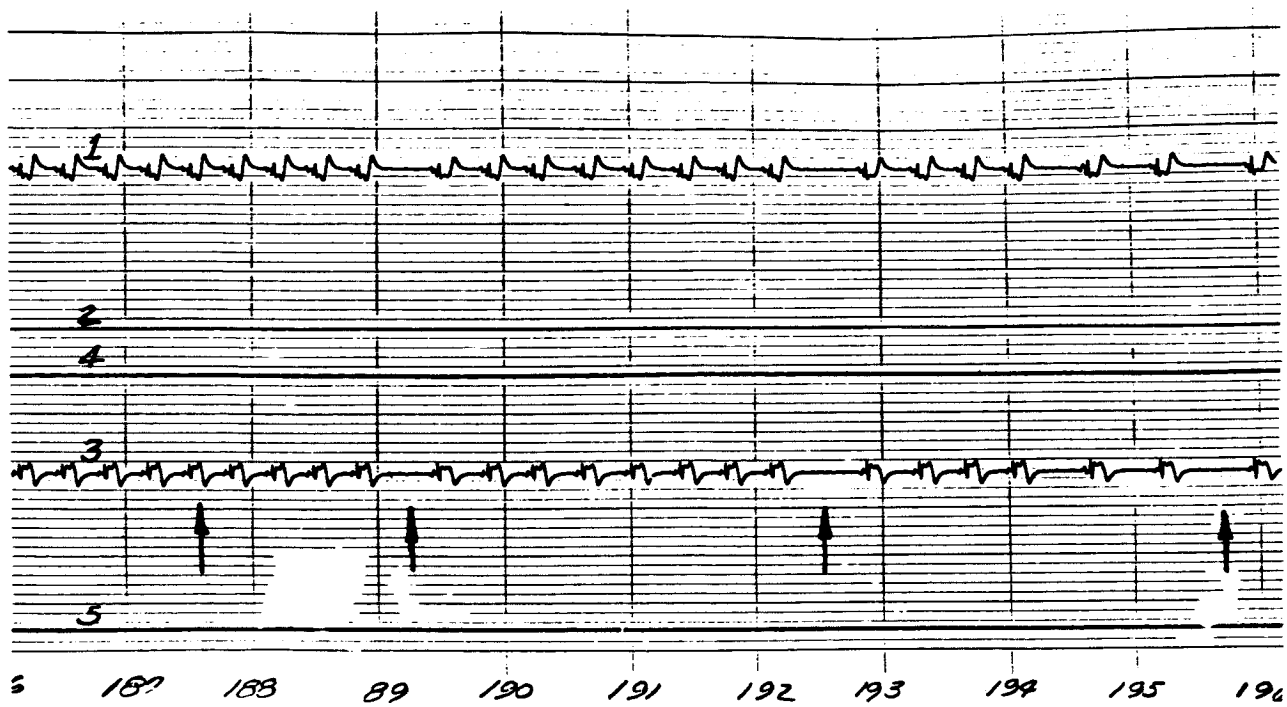


FIGURE 105 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 187-196 SECONDS

Figure 106 (A). Animal No. 241 -
Phase VI - Dwell, 190-199 Seconds

There is a developing pattern of ischemia and injury.

Figure 106 (B). Animal No. 242 -
Phase VI - Dwell, 191-200 Seconds

Sinus arrest is clearly demonstrated.

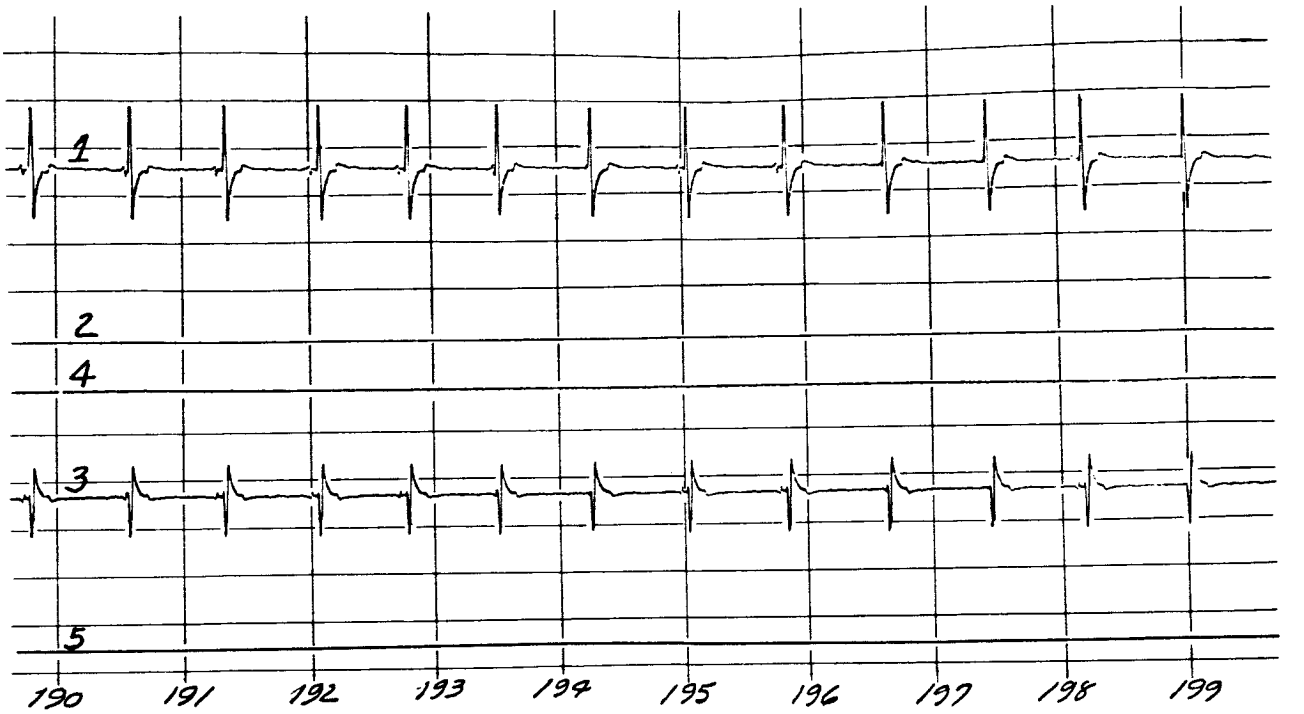


FIGURE 106 (A). ANIMAL NO. 241.
PHASE VI - DWELL, 190-199 SECONDS

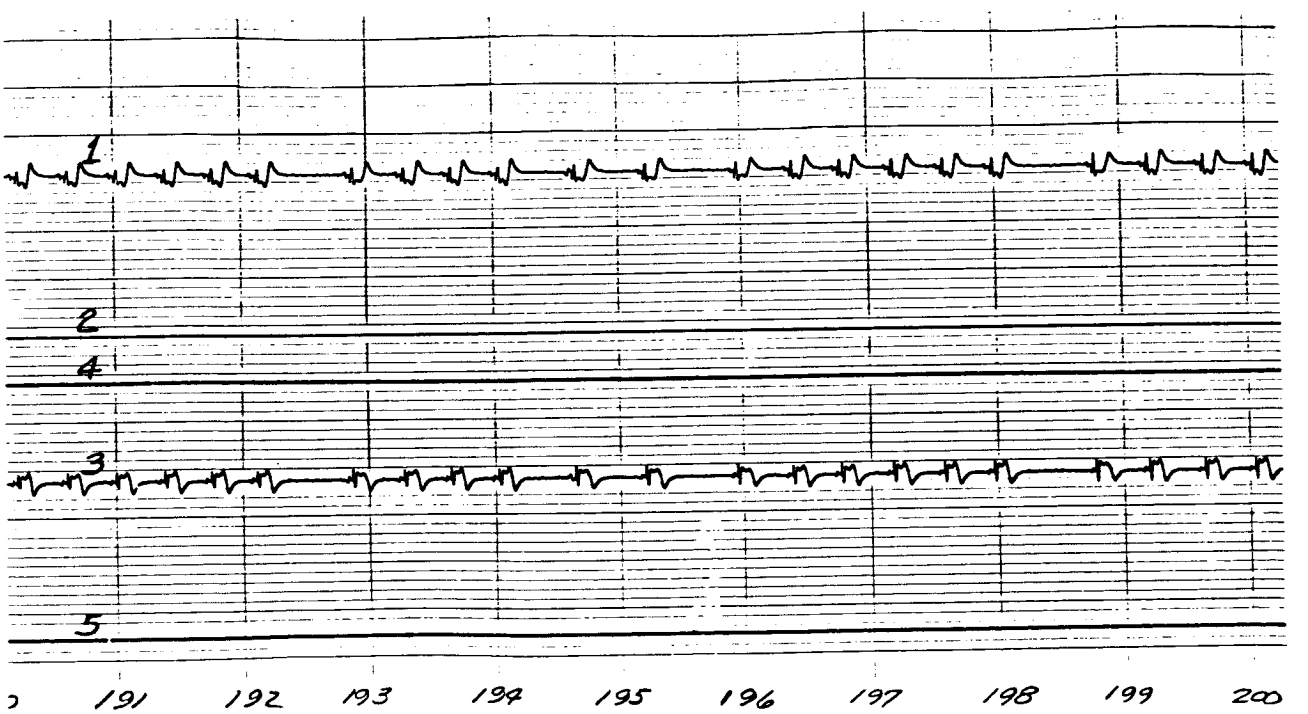


FIGURE 106 (B). ANIMAL NO. 242.
PHASE VI - DWELL, 191-200 SECONDS

Figure 107 (A). Animal No. 241 -
Phase VII - Pre-Offset, 212-221 Seconds

These are the last few seconds of dwell at peak G of 200. The end of dwell marker is on the right. The pattern of ischemia and injury is clearly demonstrated. Heart rate is 120/min.

Figure 107 (B). Animal No. 241 -
Phase VII - Pre-Offset, 200-211 Seconds

These are the last few seconds of dwell at peak G of 200. It is to be remembered that this is the second exposure the animal has had to 200 G. Its previous experience is recorded in figure A above. Note end of dwell marker. Heart rate is 120/min.

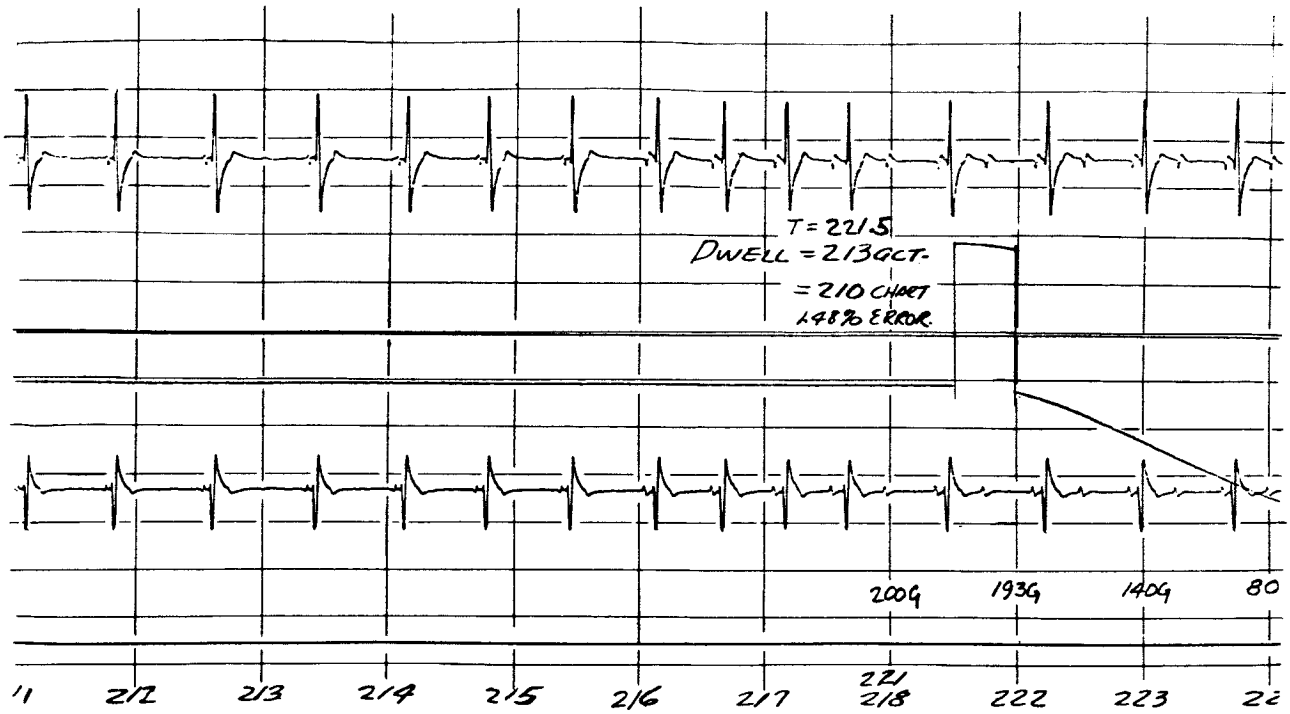


FIGURE 107 (A). ANIMAL NO. 241.
PHASE VII - PRE-OFFSET, 212-221 SECONDS

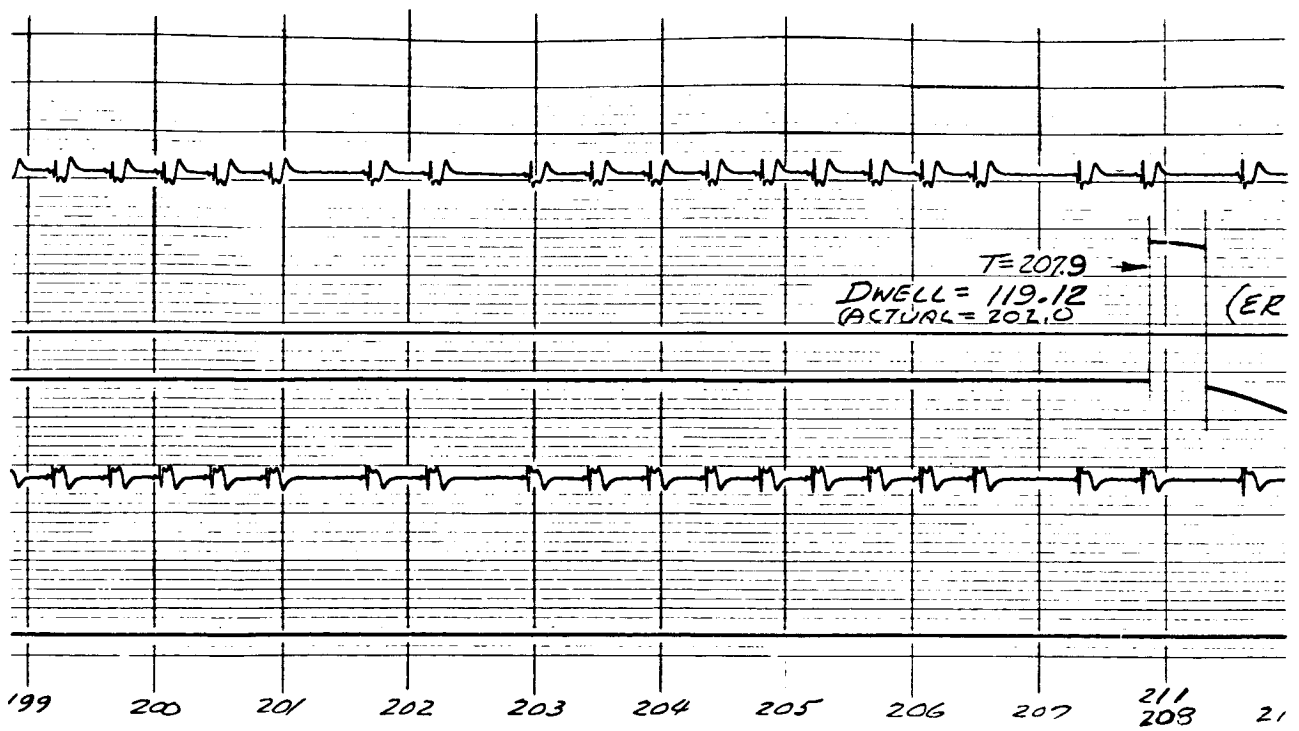


FIGURE 107 (B). ANIMAL NO. 242.
PHASE VII - PRE-OFFSET, 200-211 SECONDS

Figure 108 (A). Animal No. 241 -
Phase VIII - Offset, 221-230 Seconds

This is the offset period. The G levels are noted at each timeline. The heart rate is 90/min. A rapid drop in QRS amplitude is apparent with drop off of G stress. QRS voltage at right is 4 units. The end of run marker is shown at the right.

Figure 108 (B). Animal No. 242 -
Phase VIII - Offset, 211-219 Seconds

This is the offset period. Periods of sinus arrest persist. Changing heart rate is in evidence.

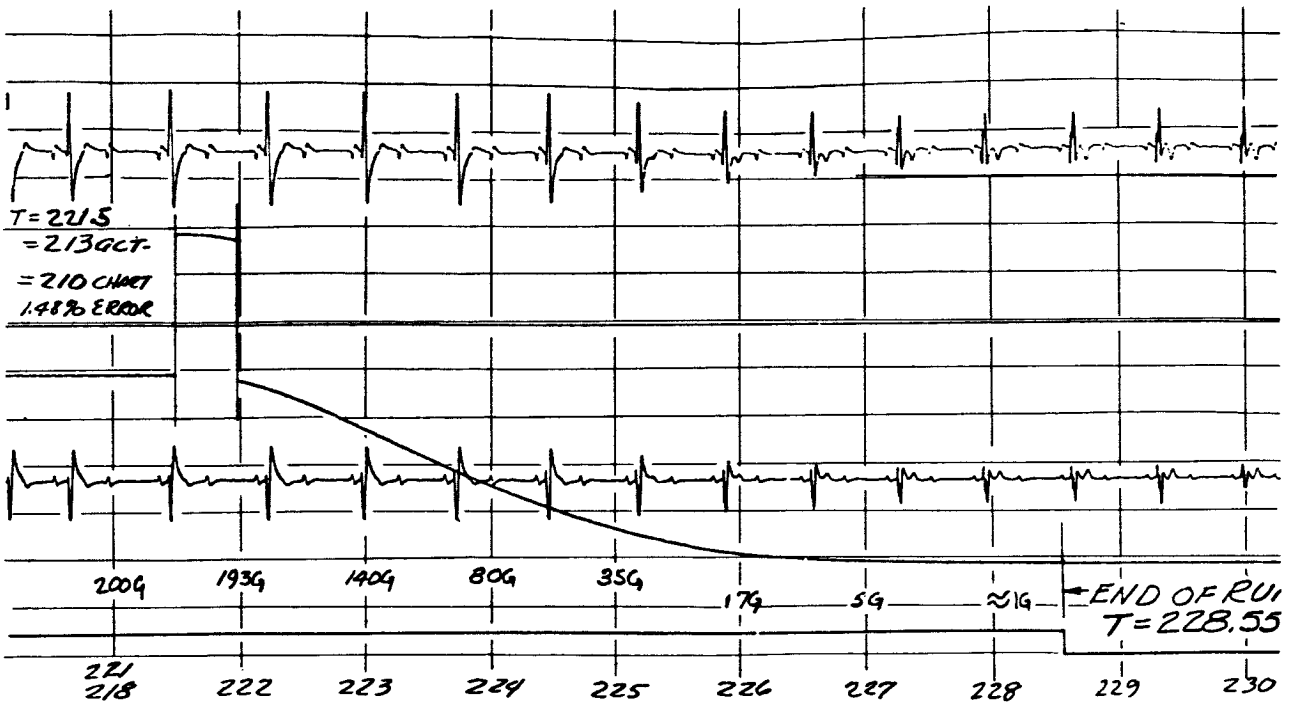


FIGURE 108 (A). ANIMAL NO. 241.
PHASE VIII - OFFSET, 221-230 SECONDS

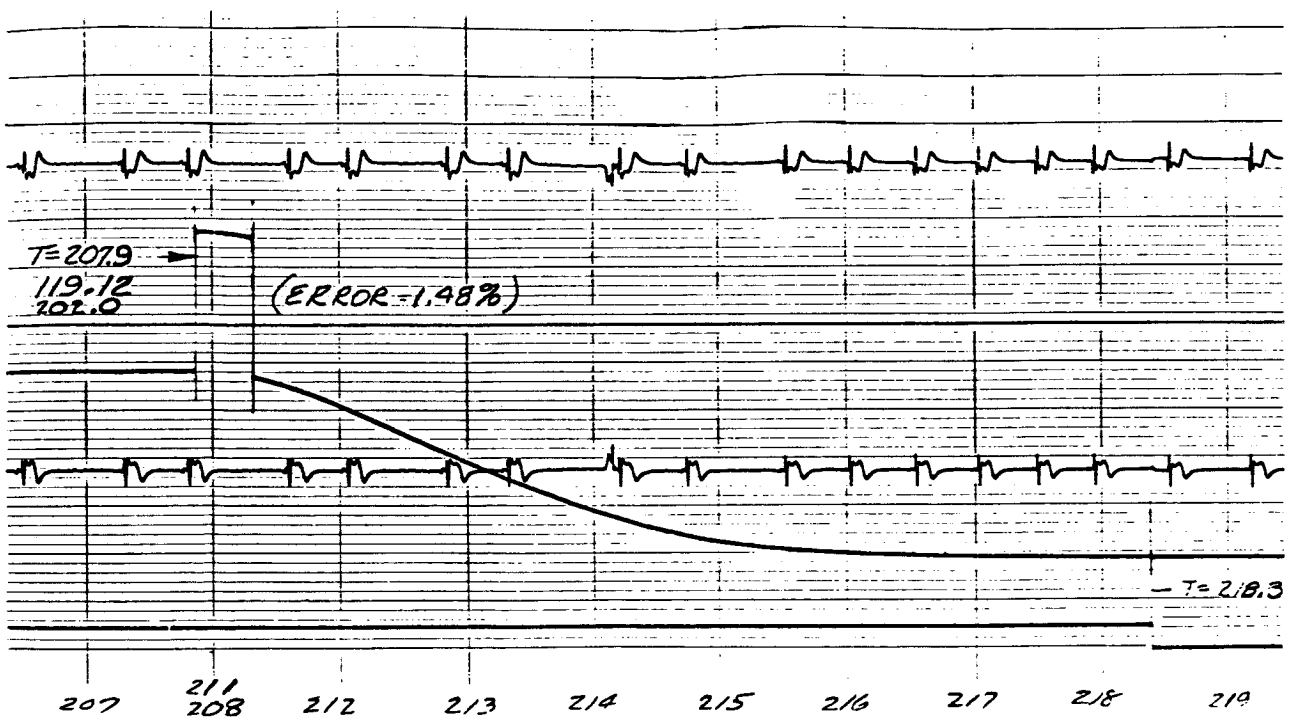


FIGURE 108 (B). ANIMAL NO. 242.
PHASE VIII - OFFSET, 211-219 SECONDS

Figure 109 (A). Animal No. 241 -
Phase IX - Postrun 1st Minute, 229-238 Seconds

The QRS amplitude gradually increases after the end of run. The rate also increases from 90 to 142/min. The ST segment is elevated. A 2:1 heart block is in evidence on the left and in the center of the trace. One to one conduction is shown on the right.

Figure 109 (B). Animal No. 242 -
Phase IX - Postrun 1st Minute, 219-228 Seconds

The heart rate averages 113/min. The P wave is wandering. ST segment is coming down to isoelectric line. T wave is diphasic; the pattern of injury persists.

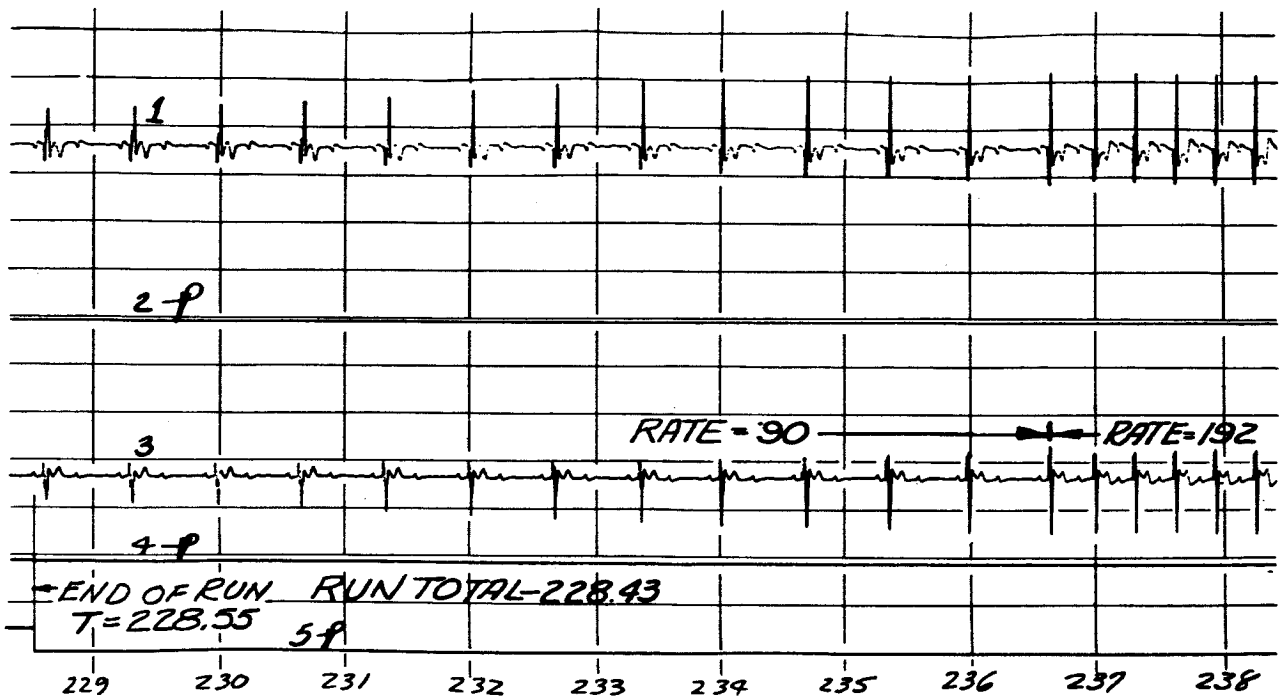


FIGURE 109 (A). ANIMAL NO. 241.
PHASE IX - POSTRUN 1ST MINUTE, 229-238 SECONDS

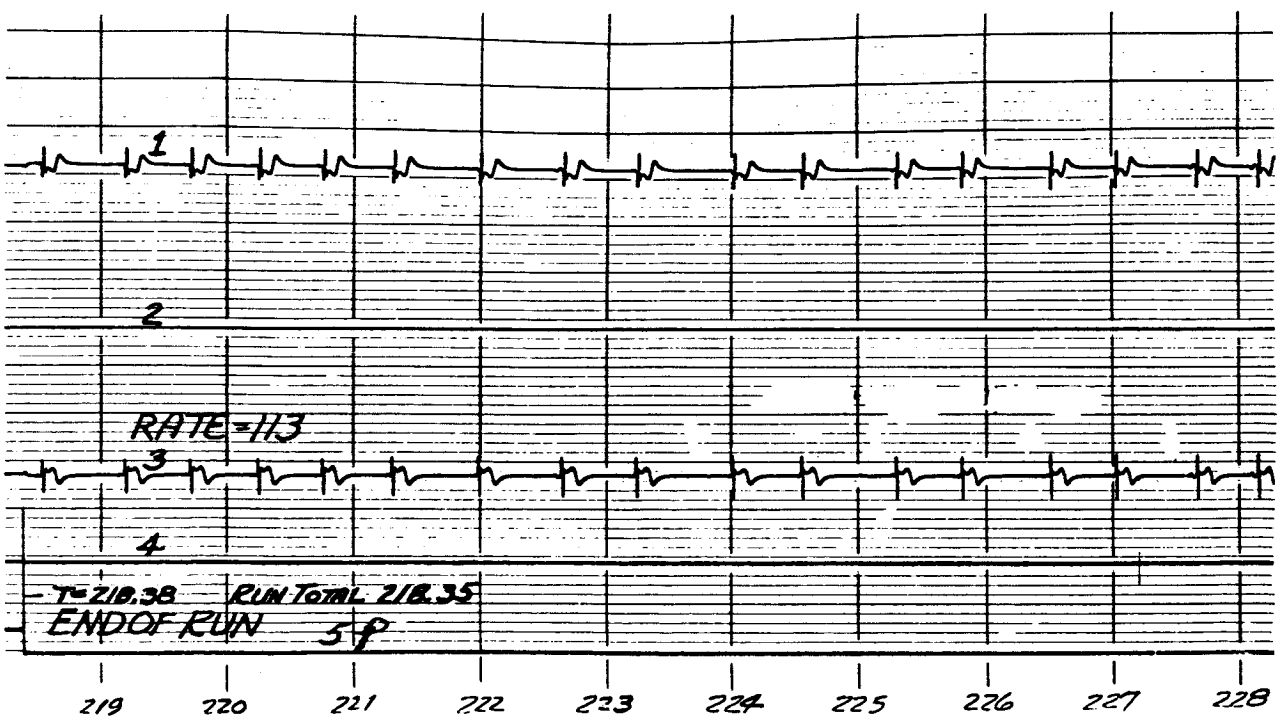


FIGURE 109 (B). ANIMAL NO. 242.
PHASE IX - POSTRUN 1ST MINUTE, 219-228 SECONDS

Figure 110 (A). Animal No. 241 -
Phase IX - Postrun 1st Minute, 234-243 Seconds

A 2:1 heart block is in evidence. The heart gets through this and demonstrates one to one conduction on the right. Rate change is from 90 to 186/min. QRS voltage increases from 9 to 12 units. A change in the configuration of the QRS takes place. Pattern of ischemia and damage persist. T wave is diphasic.

Figure 110 (B). Animal No. 242 -
Phase IX - Postrun 1st Minute, 229-237 Seconds

Heart rate increases from 138 to 178/min. QRS voltage is at 5 units. ST segment is approaching isoelectric line. T wave is diphasic.

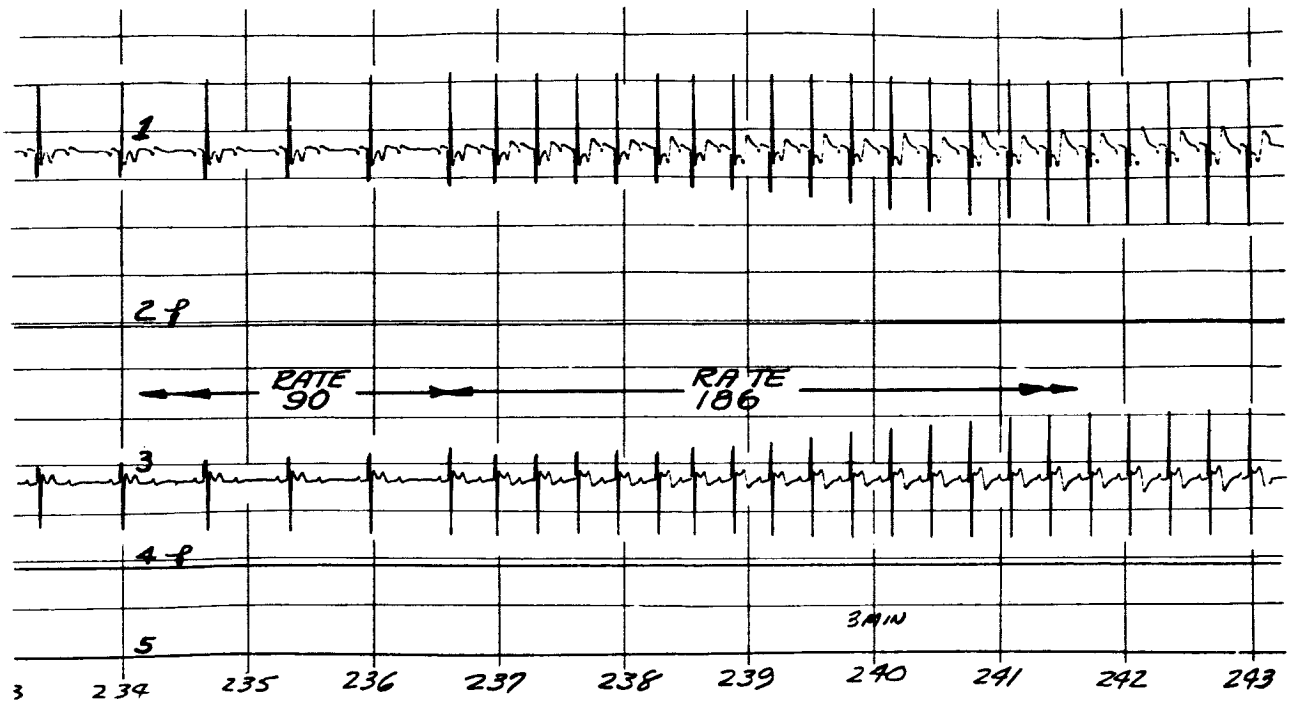


FIGURE 110 (A). ANIMAL NO. 241.
PHASE IX - POSTRUN 1ST MINUTE, 234-243 SECONDS

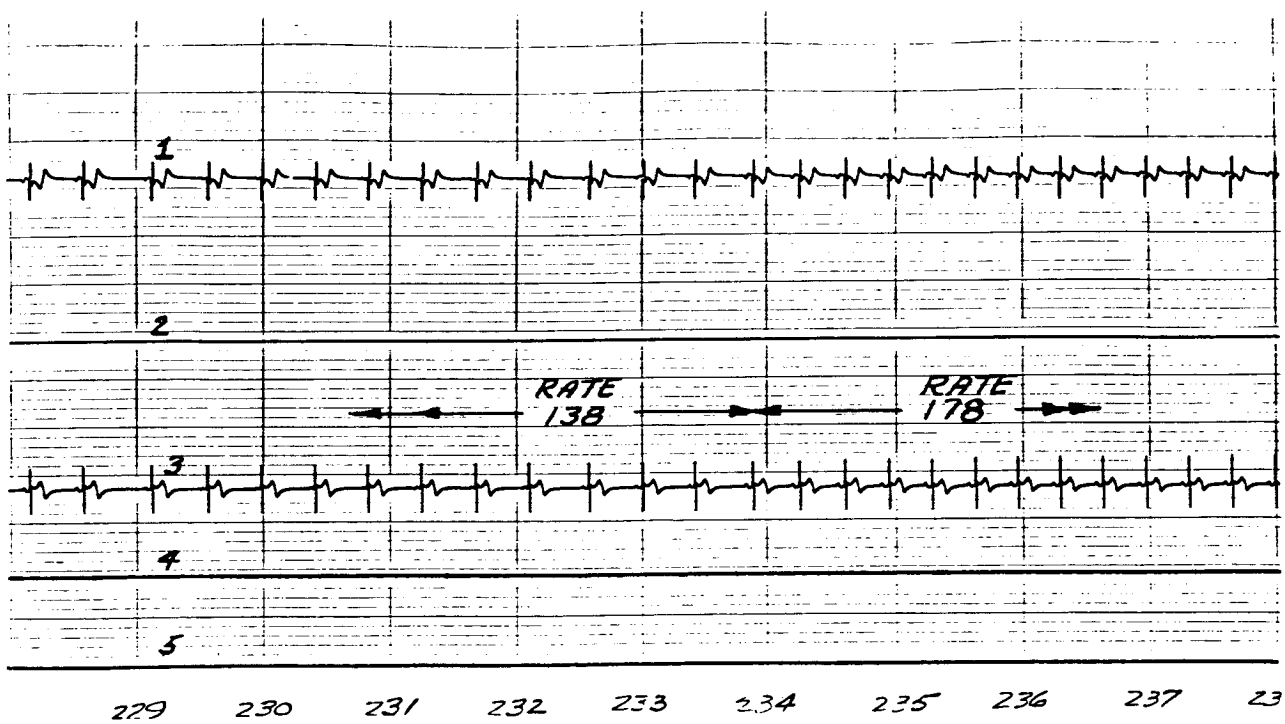


FIGURE 110 (B). ANIMAL NO. 242.
PHASE IX - POSTRUN 1ST MINUTE, 229-237 SECONDS

Figure 111 (A). Animal No. 241 -
Phase IX - Postrun 1st Minute, 246-247 Seconds

Paper speed is 100 mm/sec. Heart rate is 180/min. QRS is 3 mm in duration. T wave is inverted. ST segment is approaching isoelectric line.

Figure 111 (B). Animal No. 242 -
Phase IX - Postrun 1st Minute, 246-254 Seconds

Paper speed is 25 mm/sec. Heart rate is 180/min. Peak to peak QRS changes. This animal was visually observed to be breathing. QRS voltage is 6 units.

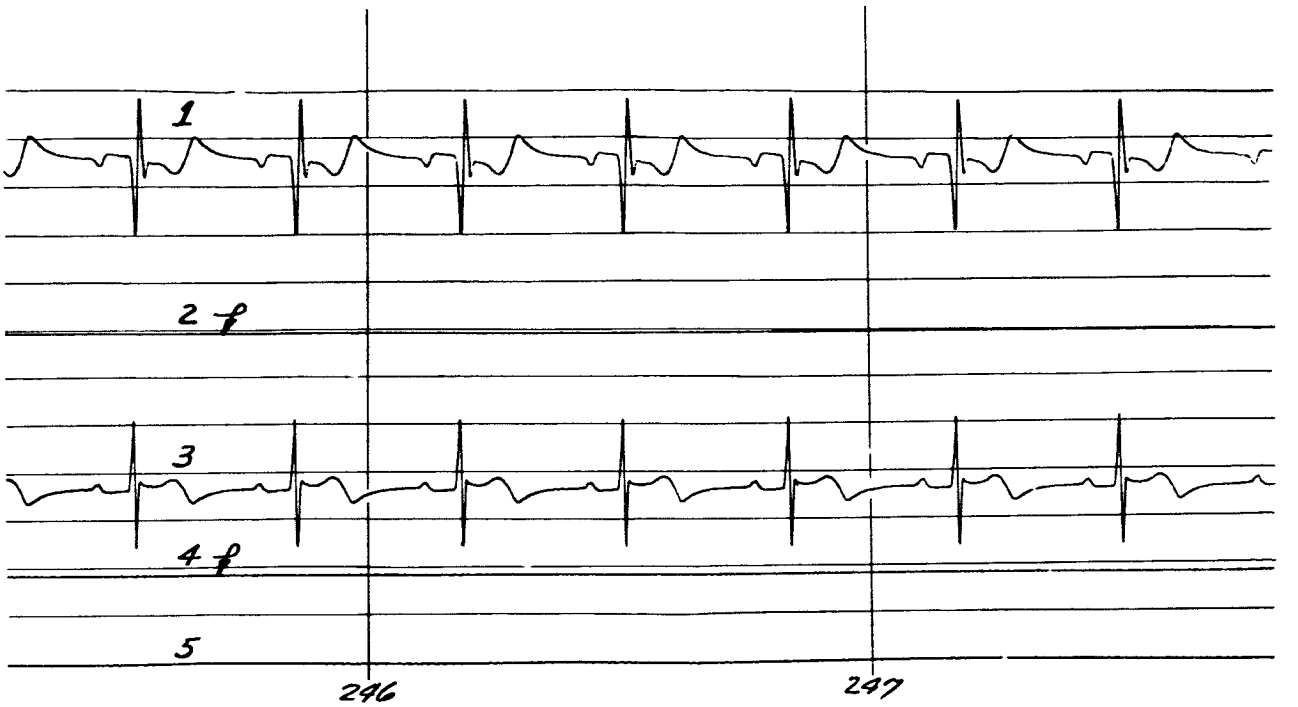


FIGURE 111 (A). ANIMAL NO. 241.
PHASE IX - POSTRUN 1ST MINUTE, 246-247 SECONDS

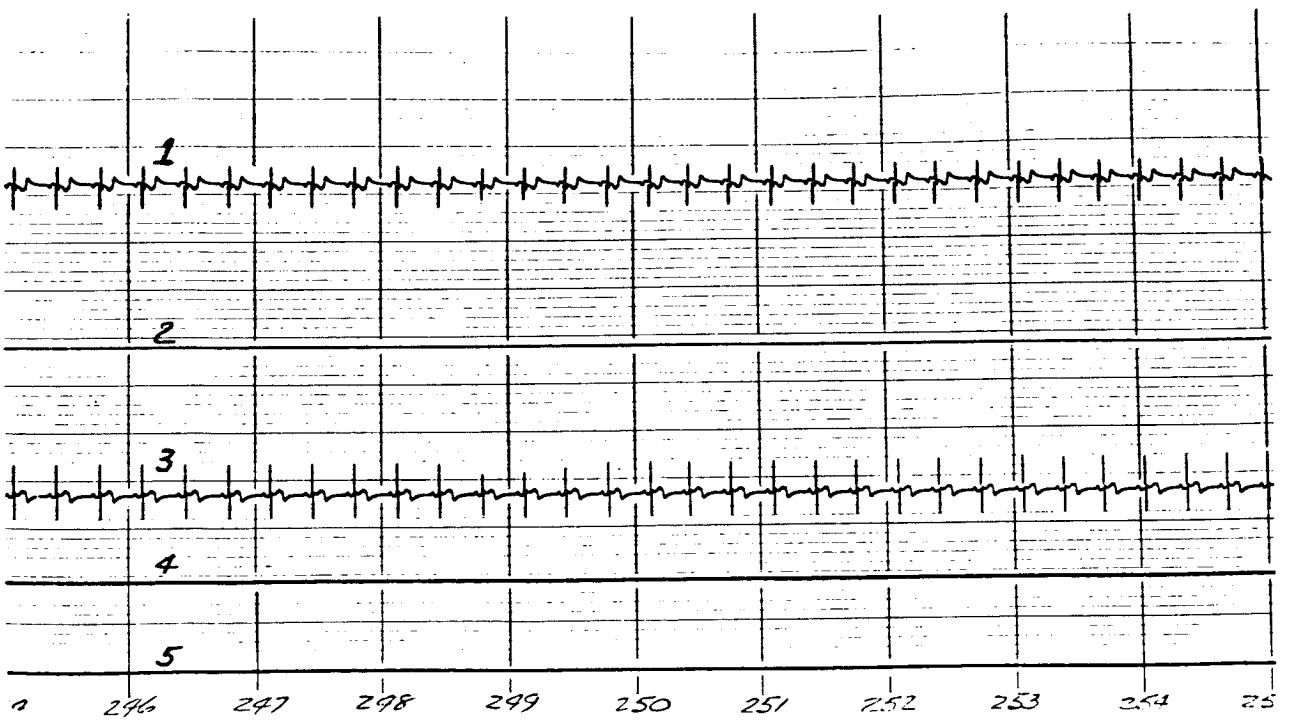


FIGURE 111 (B). ANIMAL NO. 242.
PHASE IX - POSTRUN 1ST MINUTE, 246-254 SECONDS

Figure 112 (A). Animal No. 241 -
Phase X - Postrun 2nd Minute, 280-289 Seconds

Heart rate is 180/min. The arrow indicates change in peak to peak QRS voltage. QRS voltage averages 12 units.

Figure 112 (B). Animal No. 242 -
Phase X - Postrun 2nd Minute, 275-284 Seconds

Heart rate increases from 180 to 240/min. ST segment is shifting down to baseline. A ventricular extrasystole is indicated by the right arrow. The left arrow shows a change in peak to peak QRS voltage.

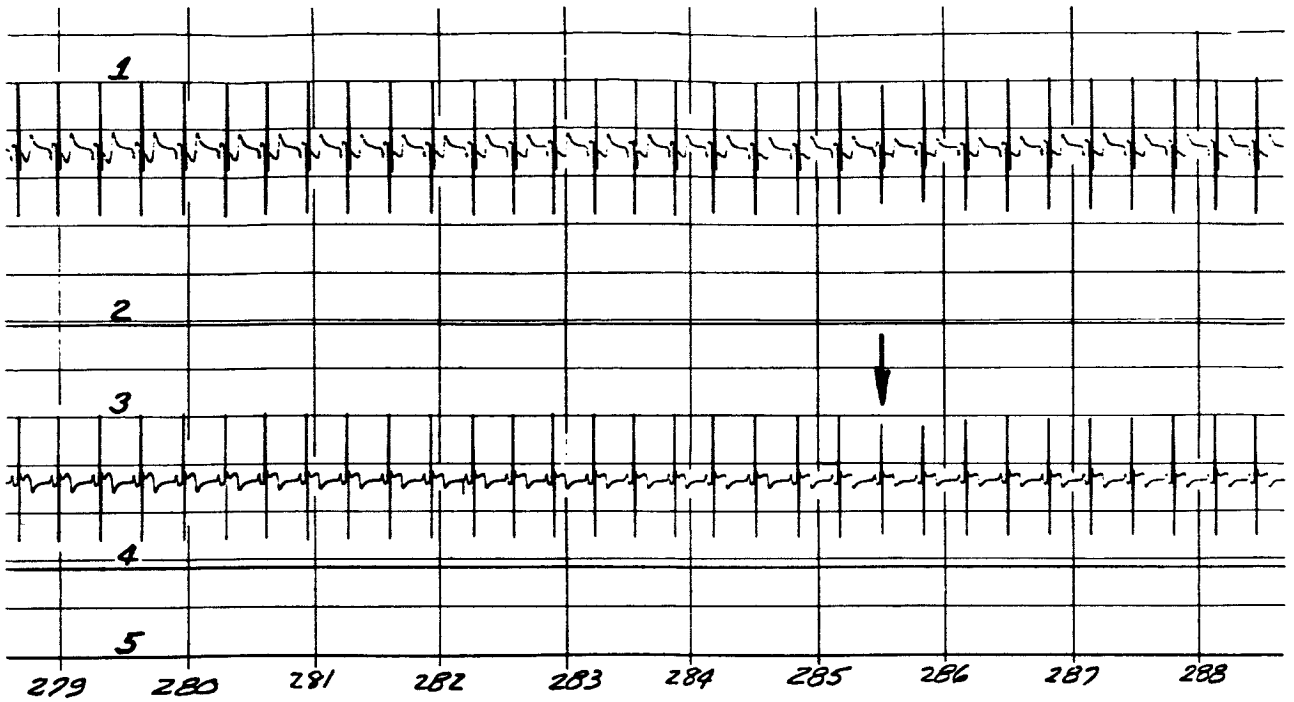


FIGURE 112 (A). ANIMAL NO. 241.
PHASE X - POSTRUN 2ND MINUTE, 280-289 SECONDS

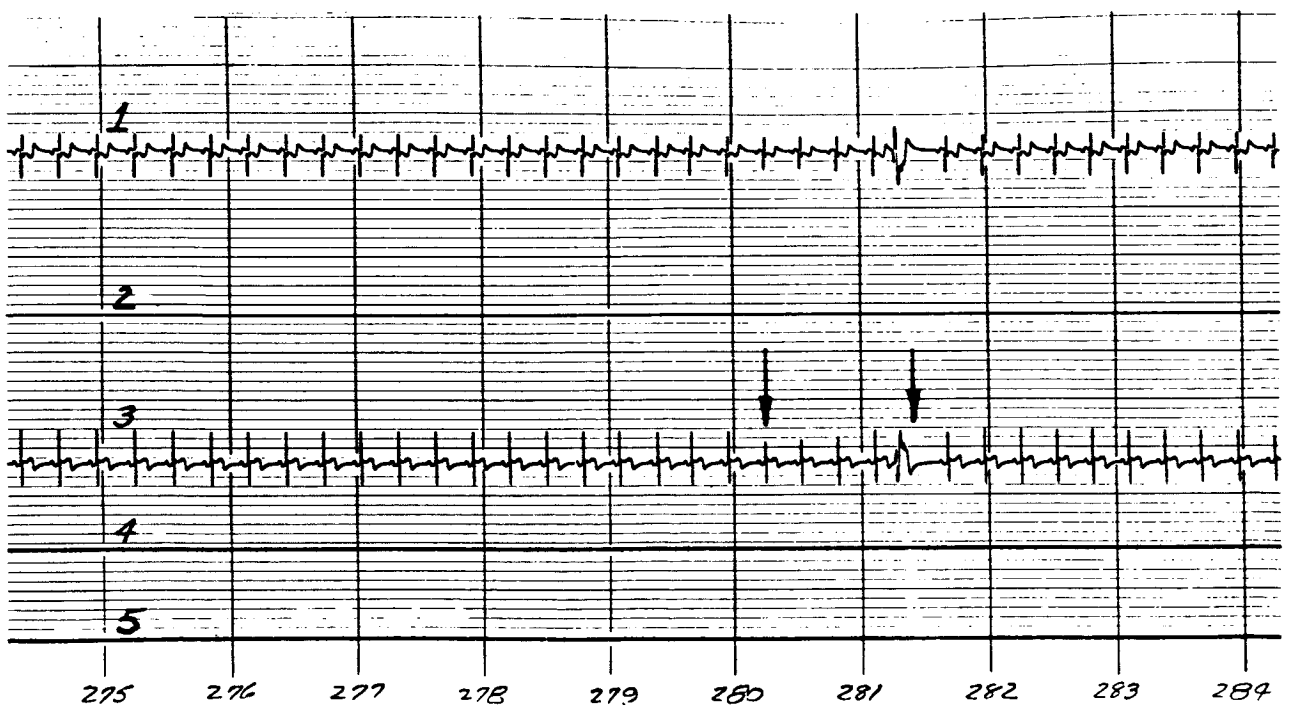


FIGURE 112 (B). ANIMAL NO. 242.
PHASE X - POSTRUN 2ND MINUTE, 275-284 SECONDS

Figure 113 (A). Animal No. 241 -
Phase XI - Postrun 3rd to 5th Minute, 345-354 Seconds

Heart rate is 240/min. Pattern of ischemia and injury persist. ST segment elevation is marked. T wave is diphasic.

Figure 113 (B). Animal No. 242 -
Phase XI - Postrun 3rd to 5th Minute, 342-344 Seconds

Paper speed is 100 mm/sec. A widening of the QRS duration as compared to baseline is nodal. Heart rate is 240/min. ST segment is virtually isoelectric.

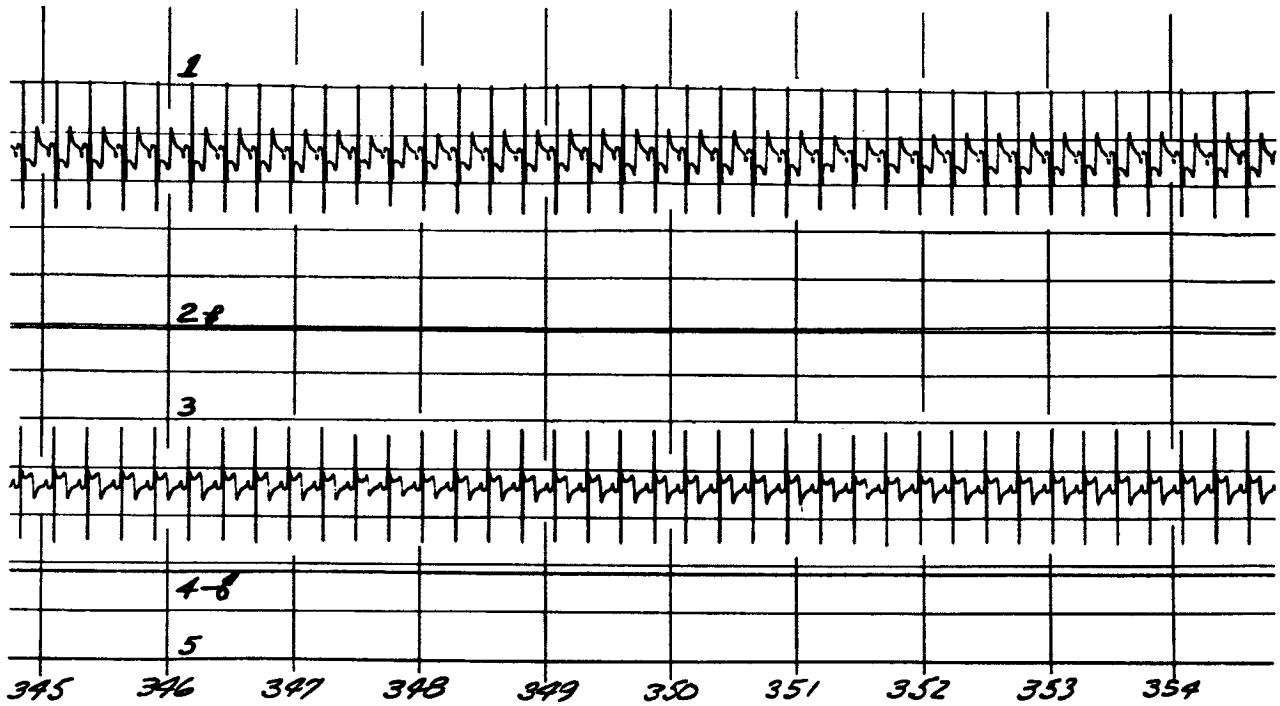


FIGURE 113 (A). ANIMAL NO. 241.
PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 345-354 SECONDS

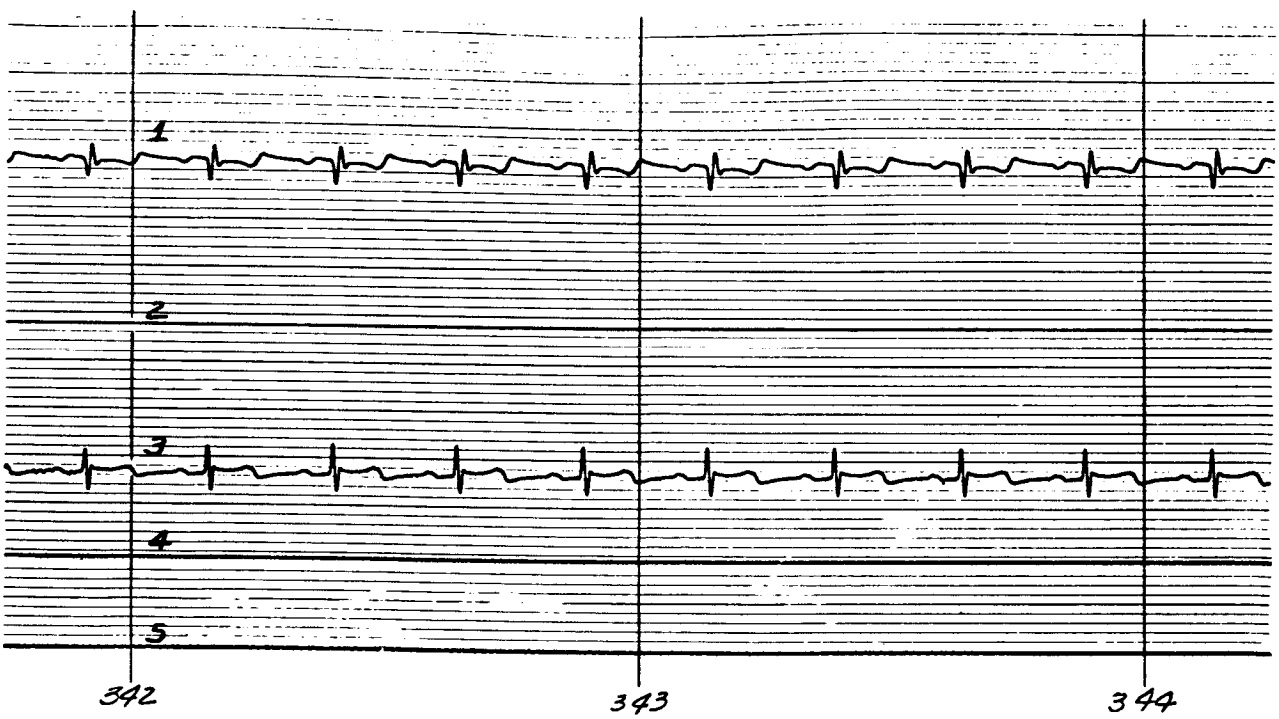


FIGURE 113 (B). ANIMAL NO. 242.
PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 342-344 SECONDS

Figure 114 (A). Animal No. 241 - Phase XI -
Postrun 3rd to 5th Minute, 418-420 Seconds - Expanded Time Base

Paper speed is 100 mm/sec. Compare this to pre-run baseline. QRS duration is 2.5 mm. T wave is inverted and ST segment is elevated. Heart rate is 240/min. QRS voltage is 11 units.

Figure 114 (B). Animal No. 242 - Phase XI -
Postrun 3rd to 5th Minute, 382-383 Seconds - Expanded Time Base

Heart rate is 240/min. Compare this to pre-run baseline (figure 83B). QRS duration is 2 mm and QRS voltage is 4½ to 5 units.

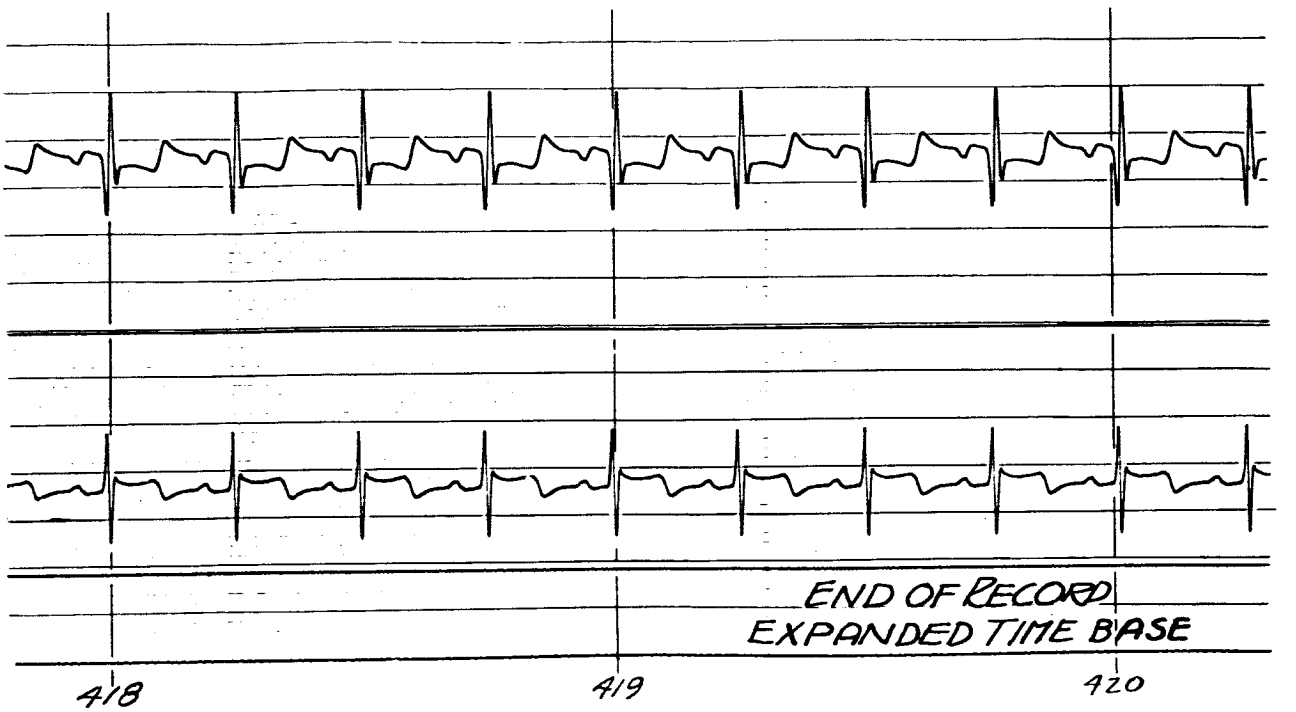


FIGURE 114 (A). ANIMAL NO. 241. PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 418-420 SECONDS - EXPANDED TIME BASE

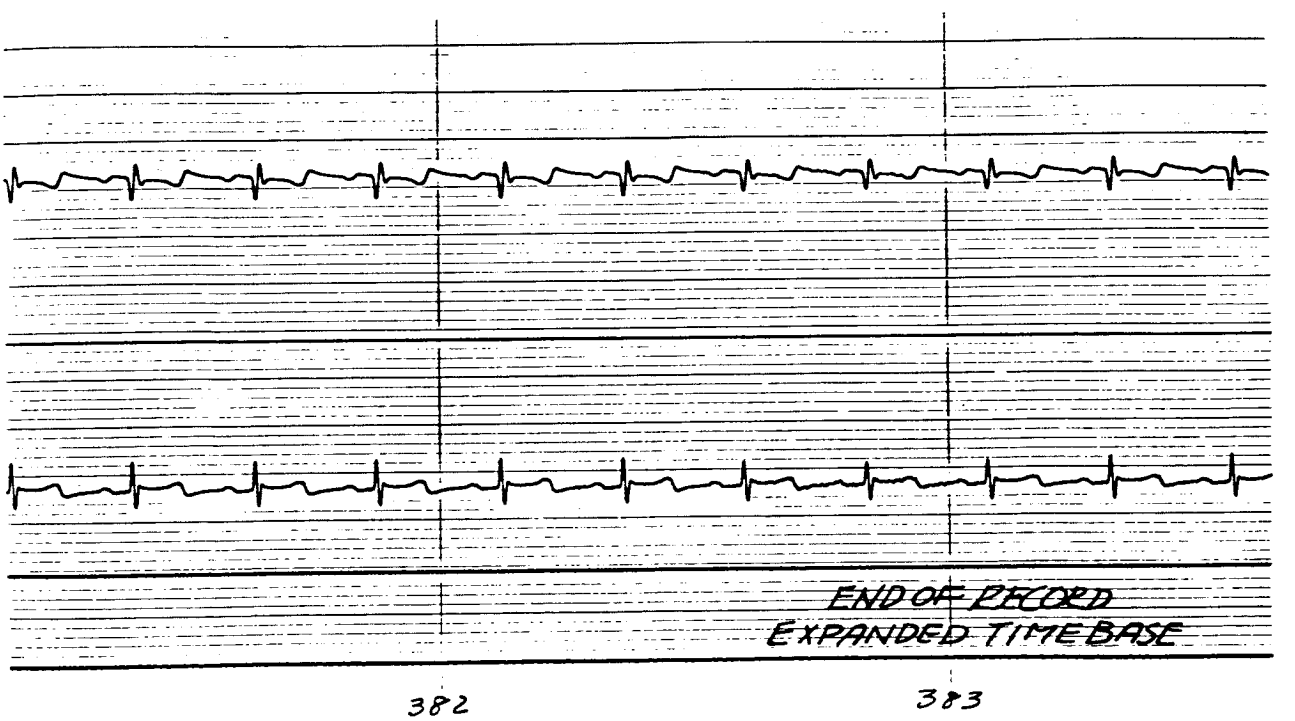


FIGURE 114 (B). ANIMAL NO. 242. PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 382-383 SECONDS - EXPANDED TIME BASE

Figure 115 (A). Animal No. 241 - Phase XI -
Postrun 3rd to 5th Minute, 405-413 Seconds - End of Record

Heart rate is 240/min. Pattern of ischemia and injury persist. This animal went on to clinical survival. Its postrun ECG after its 2nd exposure is shown below.

Figure 115 (B). Animal No. 242 - Phase XI -
Postrun 3rd to 5th Minute, 426-435 Seconds - End of Record

Heart rate is 270/min. Elevation of ST segment is still evident. T wave is diphasic. This animal survived the run. It was exsanguinated only after its clinical recovery was assured and as the "Rerun Control", it was used in the enzyme histochemistry study.

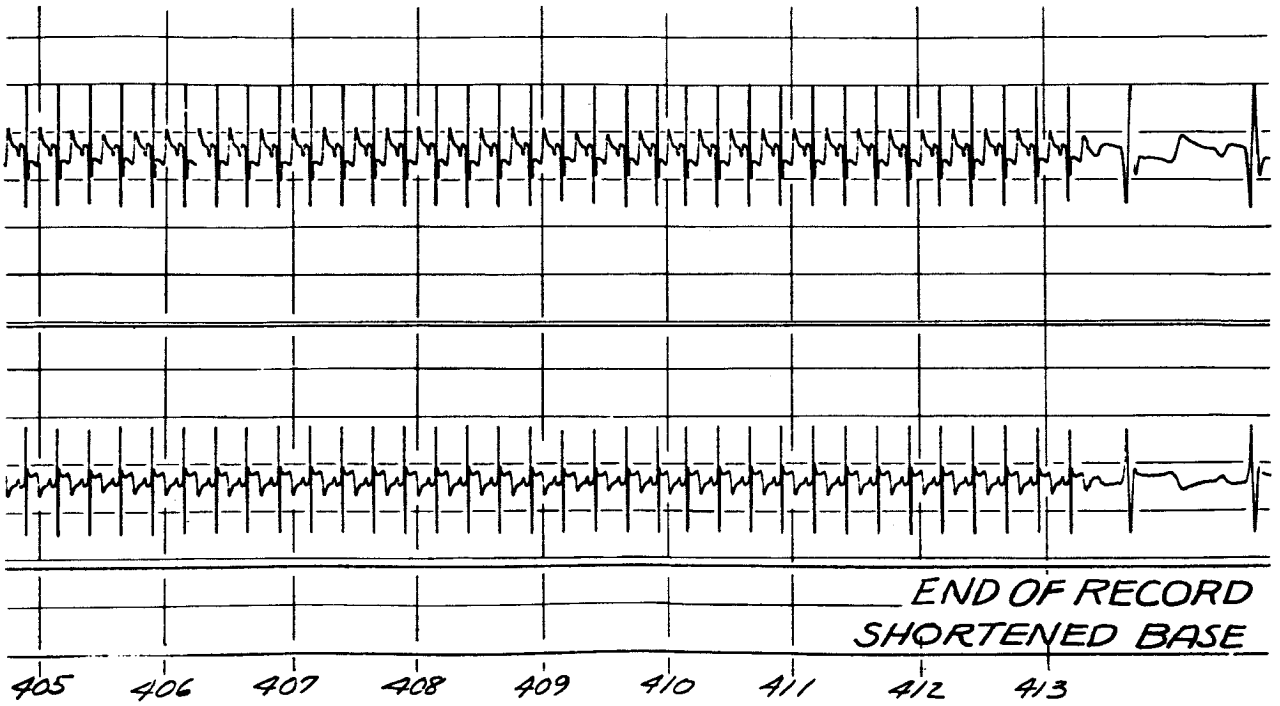


FIGURE 115 (A). ANIMAL NO. 241. PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 405-413 SECONDS - END OF RECORD

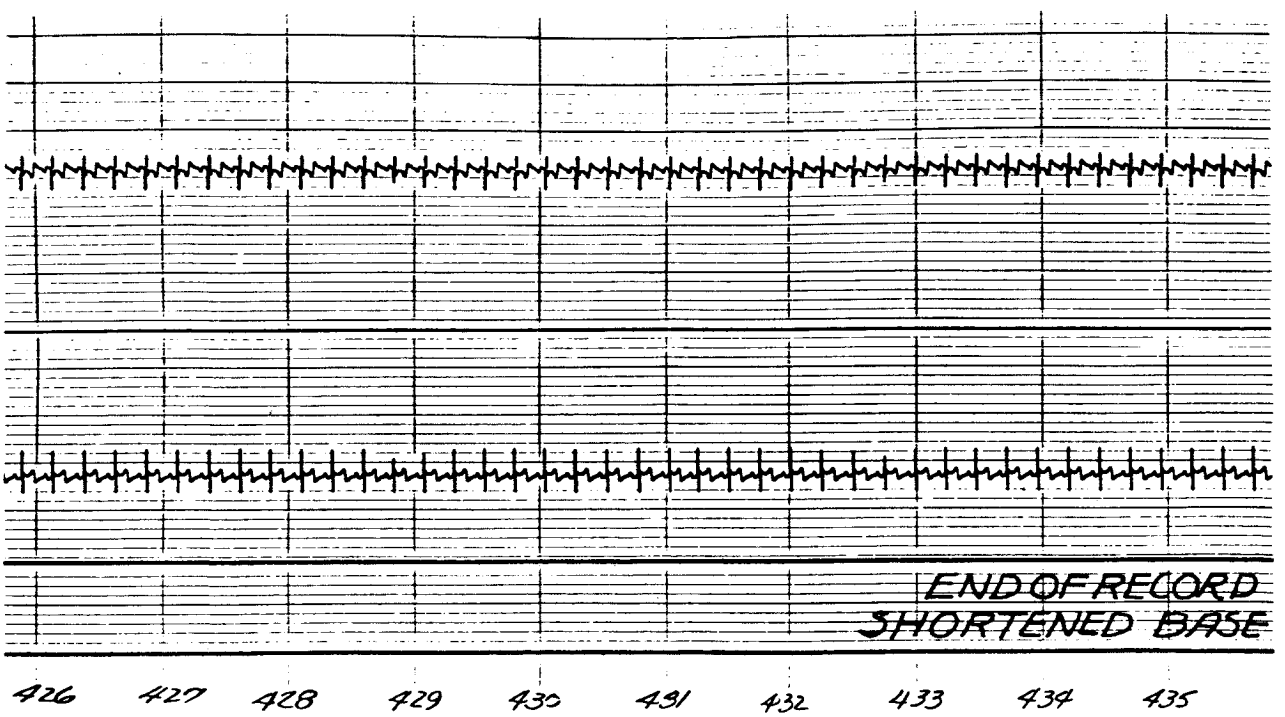


FIGURE 115 (B). ANIMAL NO. 242. PHASE XI - POSTRUN 3RD TO 5TH MINUTE, 426-435 SECONDS - END OF RECORD

APPENDIX D
GROSS PATHOLOGY

The tabulation of the results of the gross pathology study presented a difficult problem. The sheer number of detailed separate observations alone would take up an enormous amount of space. Secondly, there existed a varying profile of exposure to stress from animal to animal. Thirdly, the postrun time at which an observation of the reparative process was to be made varied also.

For these reasons the coded Table XIX Gross Pathologic Results included in this section was developed. There are 12 tissues listed and 220 animals studied. On the average, three separate observations are included per tissue. This table, therefore, presents approximately 8,000 bits of data.

This table by no means attempts to replace the detailed gross pathologic report available on each animal. But rather it attempts to summarize major findings in an easily read format. The tables are divided by tissue type proceeding in the horizontal direction. On the left hand edge of the table are a group of identifying notations. These are G mode, G stress, seconds of exposure to stress, arranged in descending order of magnitude, and hours of elapsed time after stress to autopsy, grouped by periods in ascending order.

<u>One word of caution</u> will prevent possible misinterpretation of tabulated results. Although animals in this table are listed according to decreasing dwell time in seconds in each G mode, their individual response to stress may not appear in a decreasing order of magnitude. The explanation for this is twofold. One explanation is that animals listed lower in each table may actually weigh more than animals above them. To confirm the correctness of the animal's response according to the mathematical hypothesis described in another section, check the animal's number in appendix A and observe its weight and K_p value. The other explanation is biologic variability.

An easily remembered code of letters and numbers is included and is self-explanatory. As an example on how to read the chart, perform the following exercise:

Go to +250 G_x

Find Animal No. 184

Read that this animal died on the run
(zero hours to autopsy)

Read that it received 193 seconds of exposure to
250 G

Under GI tract read:

HC = heavy congestion
4 = petechial hemorrhages
I-6= intraluminal fluid
3 = intraluminal free blood
W = worm infestation present

Under eye read:

N = normal

TABLE XIX GROSS PATHOLOGY SUMMARY CODE

A - Adhesions (intraperitoneal)
B - Bruise - Bruised
C - Congestion - (Hyperemia)
D - Darker hue of normal color
E - Pale
F - Fatty (or) Friable tissue
G - Gangrenous (Necrotic)
H - Heavy
I - Intraluminal
J - Laceration
K - Mottled mosaic pattern
    purple
    white
    grey
    red
L - Large
M - Moderate
N - Normal NSA
O - Occlusion by thrombus
P - Purple
Q - Cardiac arrest diastole
R - Bright red
S - Slight
T - Tense - distended
U - Ulcer - Ulceration
V - Cardiac arrest systole
W - Wormy
X - Tongue
Y - Yellow
Z - Tail

1 - Emphysematous
2 - Atelectatic
3 - Free Blood
4 - Petechial
5 - Edematous
6 - Fluid
7 - Empty
8 - Left
9 - Right
10 - Skull sutures discolored
11 - Skull sutures separated
12 - Abrasion
14 - Cochlea studied
15 - Animal died in cage - post run
16 - Neck muscles congested
17 - Healing excoriations
18 - Compressed tissue
19 - Foreign body, thorn
20 - Bilirubinate stones present
21 - Cystic
22 - Small piece of metal, kidney area
23 - Bloody feces
24 - Stye (L) ear
25 - Lacerated scalp
26 - Fractured
27 - Naso-oro pharymgeal
28 - Hematoma

TABLE XIX. GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan-creas	Liver	G/B	Spleen	Kid-ney	Adre-nals	CNS	Eye	Other
Zero	148	341	HRC	VN	C-W	N	HC-A	T	N	A	N	SC	N	
24	179	300	SC-K	N	SCW	N	SC	ST	LDP	F	C	SC	N	Z
	138	257	SK	N	HW SC	N	A HC	T20	LDP	A	D	SC	N	
	139	238	RCRG	N	MC 4W	N	MC	T	LDP	A	D-C	SC	N	
48	42	200	HC	VN	HC W	N	HCJ	N	N	N	N	C-19	N	
	41	20	HC	FN	HC	N	MC	N	N	N	N	HC	N	
	40	2	MC	L	MC	N	MC B	N	N	N	FN	MC	N	
						+ 50 G_x								
Zero	144	242	HC	D	A AW	N	HC	T	N	A	D	C-18	N	
	174	223	MRC	V	HC	N	HC	T	LDP	N	NC	HC	N	17
	39	200	HC	N	T	N	HC	N	N	C	DC	HC-5	N	16
24	140	200	MKRC	N	N W	N	B MC	N	N	N	N	SC	N	
	180	151	MKC SG	N	HC 4W	N	MC B	T	DP	DF	MC D	MC	NF	
48														
	38	20	MCK	N	T	N	MC	7	L	N	N	MC 5	N	
72	37	2	HC-F-1	N	T G	N	F SC	N	N	N	N	SC 5	N	
						+ 100 G_x								
Zero	181	192	HRCK	IO	SC W	DC	HC	T	LAC	LF	DC	HC	N	
24	176	195	HC	N	SC 4W	N	SC	T	N	N	N	MC	N	
48	36	200	HC	N	MC	N	MC	DT	DC	N	N	MC	N	
	171	192	HC	N	SC W	N	MC	T	D	A	N SC	SC	N	
	141	183	HRCK	QN	SC WA	N	MC	T	LDP	A	N	MC	N	
	35	20	MC	LN	SC	N	SC B	N	LD	C	N	SC	N	
	34	2	C	N	SC	N	SC	N	N	N	N SC	SC	N	
72	142	180	DHC	Y	H CGW	HC	HC B	HCT	G	A	DG	SC	N	15
						+ 150 G_x								
Zero	152	208	HC-G	D	SC	N	HC	T	N	N	HC Y	HC 5	N	
	238	207	HRC	V-7	MC4-I-3	N	HC	T	N	F-N	MC Y	HC	N	N
24	175	200	HC	N	SC WA	N	MC A	T	L A	C A	D	MC	N	
	186	200	HC G	N	Z-Y-6 WA	N	MC	T	A	F A	DC	SC	N	
	182	168	HC	N	I-Y6 F WA	MC	MC B	T	LDPA	FA-4	Y-C	SC	N-F	F
48	143	203	HRC K	N	SC	N	MC	T	LDP	N	Y-C	MC	N	
	46	200	MC K	N	SC	N	MC	N	N	N	N	SC	N	
	44	20	MC-5	N	SC	N	SC B	N	N	N	DYC	S-C	N	
	43	2	S-C	N	N	N	SC	N	D	N	N	S-C	N	
72														
3Mo.	241	213	-	-	-	-	-	-	-	-	-	-	-	-
Imm.	242	203	HC	F	S-C	F	MC	MT	MC	MC F	N	S-C	-	14
						+ 200 G_x								
Zero	145	211	HRC	V	SC-4 WA	N	HC-B	T	N	A	E	HC	N	
	146	200	HRC	V	MC-4 W	N	HC-B	T	DPC	E	EY	HC	N	
	172	200	HRC	LO	SC-4-I-6-W	EPC	HC	T	EPC	N	EC	SC-10-P	N	Z-12
	183	196	HRC	V-O	HC-4-I-6-W	N	HC	T	EPC	F	C-4	HC-3	N	
	184	193	HRC	V-O	HC-4-I-6-3-W	4	HC	T	N	N	D-C	MC	N	
	178	185	HRC-U	V-D	S-C-4	N	HC	T	N	N	MC	MC-10	N	
	173	180	HRC	V-D	M-C	EP	HC	T	LDP	N	MC	HPC	N	
24	185	193	HC W	N	EPC-I-6 WA	N	MC	T	LDPA	A	S-C	SC	N	
	177	184	HC	N	SC W	N	SC	T	LDP	N	D-SC	MC	N	
48														
					+ 250 G_x (Continued in next column)									

TABLE XIX. (CONTINUED) GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan-creas	Liver	G/B	Spleen	Kid-ney	Adre+nals	CNS	Eye	Other
72	30	200	E SD	N	N	N	N-4	N	N	N	E	E-C F5	N	
	29	20	E SD	S-C	SC	N	B	N	N	N	N	N	N	
	27	2	SC	E	N	N	N-4	N	N	N	N	N F-5	N	
	26	2	SC	N	SC	N	N	N	N	9-T	N	SC-5	N	
			+ 250 G_x (Concluded)											
Zero	151	206	HC	N	SC	N	MC-B	T	N	N	MC-Y	HC-11-3	N	
	147	200	HC	V	SC-3W	N	HC-B	T	21	N	SC Y	HC	N	
	33	200	HC	MC	N	N	HC	N	N	N	SC	S-C ?3	N	Z-12
	156	190	MC	VD	SC I-6-4	N	HC	T-D	D	D	D-SC	SC	N	
24	149	189	HC-G	N	SC-I6 WA	N	MC A	T	LDP	A	MC	SC	N	
	150	177	HC	F	SC-I6-WA	N	MC	T	DP	E	N	SC F	N	
	158	131	HRCK G	Q	SC-I6-4W	N	MC A	T	DP	D	D MC	SC	N	
48														
72	32	20	MC K	L	SC	N	SC	SD	D	N-22	N	SC	N	
	31	2	SC K	N	N	N	SC B	N	N	N	N	SC-5-F	N	
3Mo.	157	100	N	N	F N	N	SC	ST	N	F	F	N	N	14
			+ 300 G_x											
Zero	49	200	HRC	D O	SC-I-6 WT	EPC	HC-3	T	E D	E	DHC	HC	N	
	154	192	HRC	N	SCI-6-3-4WA	N	HC	T	N	A	DC	MC	N	Z 23
	160	182	HRC	D-O	SCI-6-4	N	HC	T	N	N	EC	HC-11-3	N	
	164	174	HRC	V-F	MC-U-4-3WA	N	HC-A-W	T	D-P-F	A F	EC	HC	F	Z 23
	161	173	HRC	D-O	SC-I3-4-T	N	HC	T	N	F	N	HC-11	N	
	162	169	HC	D	SC	N	HC	T	N	F	EC	HC	N	
24	159	171	HC-K	Q	MC-U-4	N	MC	T	DP	F	D MC	SC	N	
48	163	172	MC K	N	SC-I-6W	N	MC	T	DP	N	E MC		N	
	48	20	SC K	N	SC T	J MC	MC B	ET	N	N	E	5-E		
	47	2	SC K	N	SC	JFMC	MC B	N	N	N	N	SC 5		
72														
			+ 350 G_x											
Zero	153	200	HC-19	V	SC-U-W	E	HC-19	T	N	N	E Y	HC-3	N	19
	169	180	HRC	LDO	SC-4 W	N	HC	T	DP	D	EYC	HC-3	N	19
	166	171	HRC	V	SC-4	N	HC B	T	N	N	DMC	HC	N	24
	167	160	HRC	D	SC-4W	N	HC	T	N	DA	D	HC	N	Z-12
	170	140	HRC	N	SC-4	N	MC	T	N	F	Y-MC	HC-11-3	N	
24														
48	168	165	HRC	N	SC-I6 WA	N	MC-A	T	N	A	E-MC	SC	N	
	155	164	SC-K	F Q	SC-I6 WA	N	MC A	T	DP	A	D MC	SC	N	25
	165	161	MC K	Q	SC W	HC-G	MC	T	G	J	E MC	SC	N	
	51	29	MC F	N	?U ET	N	SC	N	N	E	N	SC	N	
72	50	2	SC	N	SC	F	SC	N	N	E	N	SC-55	N	
			+ 400 G_x											
Zero	237	120	HRC	V-D	HC-4	EPC	HC	T	N	J	MC	HC	N	
24														
48														
72														
11day	239	116	EC K	N	N-W	EPC	SB	T	N	N	N	N	N	
25day	240	124												
			+ 430 G_x											

TABLE XIX. (CONTINUED) GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan-creas	Liver	G/B	Spleen	Kid-ney	Adre-nals	CNS	Eye	Other
Zero	189	287	HRC K	V	E-PC-4W	N	HC	T	LDP	N	MC	HC	8-9-5-HC'	XL3-27-3
	190	286	HRC	V	EPC-I-6-WA	N	HC A	T	LDP	A	MC	MC	8-9-5-HC	XL3-27-3
	188	239	HRC	V	EPC-I-6-WA	MC	HC	T	EPC A	F	DMC	HC-3	8-9-5-HC	XL3-27-3
24	187	206	HRC	QF	T-SC-F-WA	S-CF	L SC	T	DPC	F	FMC	E-SC-10	8-9-5-MC	XMC
48	52	200	MC	N	N	N	SC	F	N	N	N	HC	9-5-MC	14
	53	20	MC K	N	N W	N	N	N	N	N	N	SC	N	
	54	2	MC K	N	N W	N	SC	N	LD	N	N	N	N	
72														
							- 50 G_x							
Zero	57	200	MC K	N	S-CI-6-3W	N	HC-J	T	N	E	N	HC	8-9-5-HC-3	XL3-27-3
	194	163	HRC-K	V-D	EPC-4 W	S-C	MC-B	T-SC	G	F	MC	MC	8-9-5-HC	XL3-27-3
24	193	157	EPC-K	Q	SC-W	MC	MC	T	DP-A	F	MC	SC	8-9-5-MC	XL-MC-5
	192	138	EPC-W	N	S-C-4-W	N	MC-B	T	EPC	L	MC	MC	8-9-5-MC	XL-MC-5
	191	118	EPC	N	S-C FW	SC-F	SC	T	N	9-28	S-C	S-C	8-9-MC	XMC-5
48	56	20	K	N	N	N	SC	N	LD	N	N	N	9-SC	
	55	2	DP K	N	N	N	SC	N	LD	N	N	N	8-9-SC	
72														
							- 100 G_x							
Zero	63	200	HRC-3	SC	E	J	4 J	N	E-L	E	E Y	HC	8-9-5HC	XL3-2F-3
	195	110	HRC	V-7	MPC-4	SC-F	SC	T	F	N	MC	HC	8-9-5-HC	XL3-2F-3
24	198	119	HRC	N	SC-I-6W	N	SC	T	LDP	N	MC	MC	8-9-5-SC	XL-5
	197	103	HRC-K	N	SC-W	N	SC	T	LDP	N	MC	MC	8-9-5-MC	X-D
	196	100	HRC-K	Q-D	EPC-4WA	N	HC-A	T	LDP A	A-F	DMC	MC	8-9-5-MC	X-D
	59	20	ESC	F	N-W	S-28	SC	T	L	N	N	SC	8-9-5-SC	14
	58	2	ESC	4	E	4	ESC	N	LK	N	EY	SC	8-5-SC	14
48														
72														
							- 150 G_x							
Zero	67	200	HRC	O	N	N	HC	T	L	N	N	HC	8-9-5-HC	27-3-14
24	200	200	MC	N	EPC-4-WA	N	MC-J-B-G	T	RPC A	A	MC	MC	8-9-5MC	X-D
	62	20	SC	N	N WA	N	E	N	N	E	EY	N	8-9-5-SC	14
	60	2	SC	N	N	4	SC-J-4	N	N	E	EY	N	8-5-S-C	14
48	201	119	HRC-K-G	L	SC-U-4-WA	HC-J	SC	T	DRP	N	D	SC-5	8-9-5-SC	10-3
72														
96	202	132	EPC-K-G	N	EPC-TWA	N	HC	T	LDP A	N	DMC	MC	8-9-5-SC	X-D
	199	92	21-K	N	SCW	EPC	SC	N	RP	N	N	SC	8-9-5-SC	
							- 200 G_x							
Zero	66	200	HRC	O	SC	N	HC	T	L	N	N	HC	8-9-5-HC	XL3-27-3
	203	132	HRC-K-G	V-O	HC-4-3-WA	N	MC	T	RPA	214A	MC	HC-3	8-9-5-HC	XL3-27-3
	205	128	HRC	V-7	HCI-6-3-4WA	N	HC	T	DPA	A	MC	HC	8-9-5-HC	XL3-27-3
24	204	120	MC-K	N	SC-4-WA	N	MC	T	LDPA	A	DMC	MC	8-9-5-MC	X-D
	206	104	HCDP	V	MC-4-3 WA	N	HC-A	T	LDP A	A	DMC	MC	8-9-5-MC	XLD
48	65	20	MCDP	NW	N	N	SC	N	N	N	N	SC	8-9-5-SC	XLD
	64	2	MC-K 9182	N	N	N	N	T	N	N	N	SC	8-9-SC	XLD
72														
							- 250 G_x							

TABLE XIX. (CONTINUED) GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan-creas	Liver	G/B	Spleen	Kid-ney	Adre-nals	CNS	Eye	Other
Zero	214	129	HC	VDO	HCI-34WA	N	HC	T	A	N	DMC	HC	8-9-5-HC	XL3-27-3
	209	127	HC	V-7	HC-D WA	MC	MC-A	T A	DRP	A MC	DMC	HC10-28	8-9-5-HC	XL3-27-3
	207	125	HRC	V-O	HC- WA	HC	HC-A	T	DRPA	A	DMC	HC10-28	8-9-5-HC	XL3-27-3
	211	116	HC	V	HC 4 WA	N	HC	T	N	E	MC	HC10-28	8-9-5-HC	XL3-27-3
24	212	120	MCK-G	N	HC-U-3 WA	N	SC	T	DRPA	A	DHC	MC	8-9-5-MC	
	208	117	HRC	V-7	SC-I-6 WA	N	HC-A	T	DRPA	A	DHC	MC	8-9-5-MC	
	213	117	MC-K-G	MC	SCI-6 WA	N	MC-B	T	DRPA	F	ESC	SC10-28	8-9-5-SC	
48	210	121	DRP	VDO	SCUTGW	MC	HC	T	DRP	N	DHC	HC	8-9-5-MC	15 XD
	68	20	MC K	N	N	SC	MC	T	DRP	N	DHC	HC10-28	8-9-5-SC	XL3-27-3
							- 300 G_x							
Zero	217	114	HRC	VO	SC4I-6 W	N	MC	T	N	N	EY	HC10-28	8-9-5-HC	
	218	112	HRC	VO	SC4I-6-W	N	HC	T	N	4	HC	HC10-28	8-9-5-HC	
	219	111	HRCK	VO	MC 4 W	N	HC	N	N	J	DHC	HC10-28	8-9-5-SC	XSC 27-3
	69	20	HC	VO	N	N	HC	T	E	N	N	HC10-28	8-9-5-MC	
24														
48														
72														
96	221	108	MC-K	N	HC-W	SC	HC-4	T	N	J	MC	MC	8-9-5 MC	
	215	65	HC	V-O	MC-G	HC	HCG	G	DRP	N	DMC	HC-3-G	8-9-5-SC	15
120	220	109	HC	V-O	HC-G W	SC	MC-A	T	RP	A-G	DRC	HC	8-9-5-HC	26
	222	95	HC	V-O	HC-GW	HC-G	HC-G	TG	DRP	G	DHC	HC-GF	8-9-5-HC	15
22day	223	115	MC-K	N	SC-4-W	MC	MC	D	D	N	MC	SC	N	
	224	105	K	N	MC-4	HC	HPC-4	DT	D	L	DPMC	E	N	
25day	236	113												
MR DOC	231	-	HC	VO	SCI-6	HC	HC	T	D	N	MC	HC	N	
							- 350 G_x							
Zero	228	120	HRC	VO	SCI-6 WA	SC	HC A	T	A	A	DMC	HC	N	
	230	118	HRC	VO	MC-4-TWA	HC	HC A	T	RPA	A	MC	HC	8-5-MC	X-J-3
	233	116	HRC	VO	M-C4I-6W	SC	HC	T	RP	N	MC	HC	8-5-MC	
	235	116	HRPC-G	V	MC WA	HC	HC	T	A	NSC	MC	HC10-28	8-9-5-MC	X-28
24	229	114	DRC	N	SC-4W	N	SC	T	DRP	J	DMC	MC	8-9-5MC	XLD
	225	95	HC	N	MC	N	MC	T	DRPA	21	DMC	SC	8-9-SC	XD
	226	83	MCK	N	SC WA	HC	SC	T	DRPC21	A	MC	SC	8-5-SC	XD
48	227	108	MRC	V-7	HC-TWA	HC	HC	TD	RPA	A	DMC	HC	9-5-SC	15 XD
17day	234	106	MC K	QO	SC4 WA	HC	HC	DT	DRPG	L	MC	SC-5-F	SC	
25day	232	98	EPC-K	N	N	21	N	F	N	N	EY	SC	N	
							- 400 G_x							

TABLE XIX. (CONTINUED) GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan creas	Liver	G/B	Spleen	Kid-ney	Adre-nals	CNS	Eye	Other
Zero	119	289	HRC	N	MC WA	N	MC A	T	A	A	D MC	SC	N	
24	72	200	MC	N	SC W	N	SC W	20	N	N	N	MC	N	14
	71	20	MDRPC	N	N	N	SC	N	D	N	N	SC	N	
	70	2	MDRPC	N	SC	N	SC-19	N	N	N	N	SC	N	
48	121	320	EPC	N	SCW	N	SC	T	RPC	N	DMC	MC	N	
							+ 50 G_y							
Zero	118	200	HDRPC-G	V	SC W	N	SC B	T	N	E	DMC	SC	N	
	75	200	HRC	N	SC	N	HC	N	E	N	8DMC	8 HC	8-5-MC	
24	135	130	MC	N	SC-FWA	F	E B	T	DRP	F	DMC	SC	8-5-SC	
	74	20	MDRPC	Q	MC-4N	J-SC	HC B	T	D	N	N	SC	8-5-SC	
	73	2	MDRPC	N	SC	J	SC	N	N	N	N	MC	8-5-SC	
							+ 150 G_y							
Zero	78	200	HDRPC	E-O	4-W	SC-J	MC	T	E	N	9-J	8-HC	8-5-HC	X8D5-14
24	117	203	HC B	N	U-SC-W	N	SC	T	G	A	DMC	SC	8-5-HC	
48	77	20	MC-K	Q	N	N	E-SC	N	D-L	N	N	SC	8-5-MC	
	76	2	MC-K	N	SC-W	N	E-SC	N	L	E	EY	SC	N	
72	130	174	MEPCK	N	SC WA	N	SC	T	LRP	A	DY	SC	8-5-MC	
							+ 250 G_y							
Zero	-	-	-	-	-	-	-	-	-	-	-	-	-	-
24	131	185	8 DRC	N	SC-4	N	SC-B	T	LRP	E	DRYC	SC	8-5-MC	
	120	185	MC-G	N	SC-W	N	SC	T	N	A	DRYC	SC	8-5-MC	
	132	140	MC-K	N	MCW	N	MC	T	D	D	DMC	SC	8-5-SC	
48	80	20	MC-K	N	U WA	N	SC-A	T	AD	A	EY	N	8-5-SC	
	79	2	MC-K	L	MC 4	N	MC	T	LDRP	N	N	SC	8-5-SC	14
							+ 300 G_y							
Zero	123	178	8-2HC 9-1	0	MC	N	MC	T	DJB	A	DMC	HC	8-5-HC	X8LD-27-3
24	136	149	HC	N	MC WA	N	SC B	T	N	A	DMC	MC	8-5-MC	
48	81	2	MCRPC	D	N	N	SC	T	DR	N	N	SC	8-5-SC	
72	129	163	9HC8-G	N	WA	N	SCA-W	T	N	AW	9 D	SC	8-5-MC	
	82	20	K	N	W	N	SC	T	E	E	N	SC	8-5-MC	
							+ 350 G_y							
Zero	124	200	HRC	V	N WA	N	SC	T	B	A	DMC	MC	8-5-MC	
	125	179	MRC	V-O	HC-U-4 WA	N	HC	T	A	A	DMC	HC	8-5-MC	XDC
	127	171	HRC	N	SC	N	HC-B	T	A	A	DMC	8HC-9E	8-5-HC	X-5DC
24	137	178	NRC	B	SC WA	N	SC-B	T	N	A	DMC	SC	8-5-MC	
	126	174	HRC	Q	MC W	HC	HC-J	T	N	EA	DMC	MC	8-5-HC	XD 28-14
	133	154	RPC	N	SC WA	N	SC-B	T	DRP W-21	A	DMC	E	8-5-MC	
	134	152	HRC	N	SC-U WA	N	SC B	T	N	A	DMC	SC	8-5-MC	
	122	139	MRC	N	SC-U-4WA	N	MC	T	LRP	E	DMC	SC-E	8-5-SC	
48														
72	128	164	EPC	N	?U-SC-W	N	SC-J	T	N	N	N	MC	8-5-SC	14
	83	20	MC-K	Q	SC-W	N	HC-WA	E-F	E	EA	N	SC-19	8-5-SC	
							+ 400 G_y							

TABLE XIX. (CONCLUDED) GROSS PATHOLOGY SUMMARY

#Hrs.	No.	D_r Sec.	TISSUE											
			Lung	Heart	GI	Pan-creas	Liver	G/B	Spleen	Kid-ney	Adre-nals	CNS	Eye	Other
Zero	112	386	HRC	J-V	HC-4 WA	4	HC-WA	T	DWA	A	DMC	HC	N	
24														
48	115	266	MC-K	N	HC-4	HC	HC-B	DT	N	N	DMC	SC	N	
	116	189	MC-K	N	SC-4	N	SC-B	T	N	E	EY	SC	N	
							- 50 G_y							
Zero	107	181	MC	V	HC 4 WA	N	SC	T	A	A	DRYC	HC	9-5-MC	27-3
24	101	210	MC	N	SC-W	N	SC-B	T	DRP	E	DYC	MC	9-5-SC	
	86	200	HRC	N	MCU-W	N	MC-J-B	N	A	DA	DMC	SC	9-5-SC	
	85	20	MC	F	N-W	N	MC-B	N	N	N-A	EY	N	9-5-SC	
	84	2	MC-K-W	N	SC-UFW	N	MC	N	LD	A	DYC	SC	N	
48	111	200	HRC	N	?USCWA	N	SC-WA	T	DRP	A	N	MC	N	
	110	100	HRPC	Q	SC WA	N	SC-W-A	TD	DRP	A	DYMC	SC	9-SC	
							- 100 G_y							
Zero	102	228	HRC	V	HC WA	SC	HC	T	N	E	DMC	9HC 8E	9-5-SC	
24	93	200	HC	Q	SC W	N	SC-B	N	N	E	N	MC	9-5-MC	
	88	20	D-2 K	Q	E-3WA	N	SC-WA	N	D A	D A	DYC	N	9-5-MC	
	87	2	SC-K	N	S-C 4 WA	N	A	N	DRP	A	N	N	9-5-SC	
48														
72	108	210	HC-K	Q	SC ? U	N	SC	T	D	A	N-J	HC	9-S5	
							- 200 G_y							
Zero	104	202	HRC	V	4-W	N	MC	T	N	N	N	9 HC-8E	8-9-MC	
	103	200	HRC	V	SC-W	N	SC	T	N	E	D	HC	9-S-5	
24	96	200	HRC	V	SC-4 WA	N	HC A	N	D-A	A	D	HC-W	9-5-MC	
	91	20	HRC	N	SC WA	N	A	N	N-A	A	D	E	9-5-HC	
	90	2	MC	Q	SC	N	SC B	N	D	D	D	MC	9-5-MC	
48														
							- 300 G_y							
Zero	97	200	HRC	V	SC-4-W	E	HC	T20	E	N	N	9 HC 8E	9-HC	Z-D 14
24	95	20	MCK	N	E-WA	E	E B	T	D	A	D	E	9-MC	
	94	2	SC K	Q	4-WA	N	SC	L	N	E	D	SC	9-MC	14
48	114	178	HRC	N	W	N	SC	T	D	N	N	E	9-MC	
72	109	100	MC-K	Q	U WA	N	H-4	L-T	DRP	DA	D	SC	9-5-SC	
96	105	163	EPC-K	N	SC	N	N	T	DP	N	N	N	9-5-SC	
							- 350 G_y							
Zero	106	167	9HC 8RC	VO	H4-WA	SC	SC-A	T	DA	A	DYMC	HC	9-5-SC	
24	99	20	HRC	Q	AW	N	SC-A	N	A	A	DYMC	E	9-5-MC	
	98	2	MRC	Q	SC-4	SC	SC	N	N	A	D	SC	9-5-SC	
48	113	170	MRC	N	WA	N	B	T	N	E	D	HC	9-5-SC	
+	100	200	[illegible]			Died pneumonitis Dec. 6, 1964								
							- 400 G_y							

APPENDIX E
HISTOLOGICAL STUDY DATA

MICROPHOTOGRAPHS

Microphotographs of selected slides from the histologic study are displayed on the following pages. These slides were chosen as examples of typical tissue damage at various levels of accelerative stress and dwell times at stress. The slides were chosen from animals Nos. 33, 39, 49, 57, 63, 66, 67, 69, 242, and 243.

Stress History

The stress history of these animals is summarized below:

Animal No. 39.- A run fatality, exposed to 100 $+G_x$ for 200 seconds. Figures 117 and 118.

Animal No. 33.- Run fatality, exposed to 300 $+G_x$ for 200 seconds. Figure 116.

Animal No. 49.- Run fatality, exposed to 400 $+G_x$ for 200 seconds. Figure 119.

Animal No. 57.- Run fatality, exposed to 100 $-G_x$ for 200 seconds. Figures 120, 121, 122, 123, 124.

Animal No. 63.- Run fatality, exposed to 150 $-G_x$ for 200 seconds. Figures 125, 126, 127.

Animal No. 67.- Run fatality, exposed to 200 $-G_x$ for 200 seconds. Figures 131, 132, 133.

Animal No. 66.- Run fatality, exposed to 250 $-G_x$ for 200 seconds. Figures 128, 129, 130.

Animal No. 69.- Run fatality, after initial ECG indications of impending recovery. Exposed to -350 $-G_x$ for 20 seconds. Figures 134, 135, 136, 137.

Animal No. 242-241.- Re-run control, enzyme study. As No. 241, survived exposure to 200 $-G_x$ for 213 seconds, and three months later, as No. 242 was re-exposed to 200 $-G_x$ for 202

seconds. Was classed as clinical survivor and then autopsied.

Animal No. 243.- No-run control, enzyme study. This normal animal was sacrificed without exposure to accelerative stress. The tissue taken are compared directly with the similar sections from animal No. 242. Figures 138 through 147.

MORPHOLOGIC CHANGES

Most of the Morphologic changes indicative of cell death are biochemically initiated and produced by enzymes of either intra- or extra-cellular origin following the death of the cell.

Temporal Considerations

Since enzymatic action occurs slowly, some time must elapse, depending on the enzyme involved, before modification of cell structure becomes apparent. Sudden death of cells and immediate fixation with standard techniques for microscopic study may not disclose any alteration in morphology. Thus, if the animal should die suddenly of an occlusion of the coronary vessel with myocardial infarction, at post-mortem examination no morphologic alteration can be seen in the heart. If the animal survived 24 hours or longer after the occlusion, sufficient enzymatic change should occur to permit identification of injured myocardium upon subsequent microscopic examination.

Therefore, morphologic modification represents a dynamic process occurring over a period of time, progressively becoming better defined and apparent and eventually resulting in total destruction and removal of the necrotic cells.

The method of spacing of autopsies employed in animals exposed to near tolerance levels permitted the full development of tissue response to stress damage. Furthermore, our specially designed oxidative enzymatic study was successful in picking up acute morphologic changes immediately post-run, as shown in Figures 138 through 147.

Nuclear Changes

In general, the histologic changes that indicate death of a cell are more prominent in the nucleus than in the cytoplasm. Conversely, degeneration and infiltration compatible with ultimate survival of the cell tend to affect chiefly the cytoplasm. Perhaps the first observable change in the nucleus of a dying cell is a loss of the normal distribution of the chromatin. Under examination, the nucleus appears shrunken and more deeply stained, assuming a homogenous dark discoloration and the nuclear membrane loses its smooth contour becoming somewhat irregular and wrinkled. This is called pyknosis. Later the nuclear membrane ruptures and the nucleus may undergo karyorrhexis which is a fragmentation of the nucleus and release of the nuclear contents into the cytoplasm. Complete dissolution of the nuclear membrane may not occur immediately after rupture. Then, following an initial period of increase of staining reaction, the nucleus may become progressively more faintly stained and eventually reach a point called karyolysis at which all basophilism is lost and the nucleus cannot be distinguished. See Figure No. 124. This figure illustrates hyperemia of the myocardium. Also illustrated is myocardial necrosis. In the upper right hand corner of the microphotograph, note the dark discoloration of one of the cells. This change is typical of pyknosis and heralds the death of the cell.

Cytoplasmic Changes

In the earliest changes seen, the cell may appear somewhat larger and the cytoplasm more granular than normal. This is sometimes referred to as a "cloudy swelling" of the cytoplasm. Over a span of hours to days the cytoplasm becomes more acidophilic, dense, and opaque, and loses its fine granularity. Granular coagulation and fragmentation then occur, concomitant with the appearance of fat droplets in the necrotic cytoplasm. By this time the nucleus has disappeared by either karyorrhexis or karyolysis. Eventually the quality of the cell membrane becomes lost as it fuses with the opaque cytoplasm. Shadow outlines of cells may persist for a time, but eventually the entire cell undergoes enzymatic dissolution and disappears. The entire cell becomes an amorphous, granular, opaque, anuclear mass. See Figure No. 124. This figure illustrates changes typical of an injured cell. In the cell described above for nuclear changes and in the cell in the lower left hand of the illustration, note the increased affinity of the injured cell for acidophilic eosin dye. Also note increased density and opaqueness of the cytoplasm.

Transitory Changes

If, of course, the damage to the cell is transitory in nature or of insufficient quantity to cause terminal cellular changes, the tissue cell will show a weak reconstitution. At a later time, it may be difficult to even find evidence that a cell was damaged slightly.

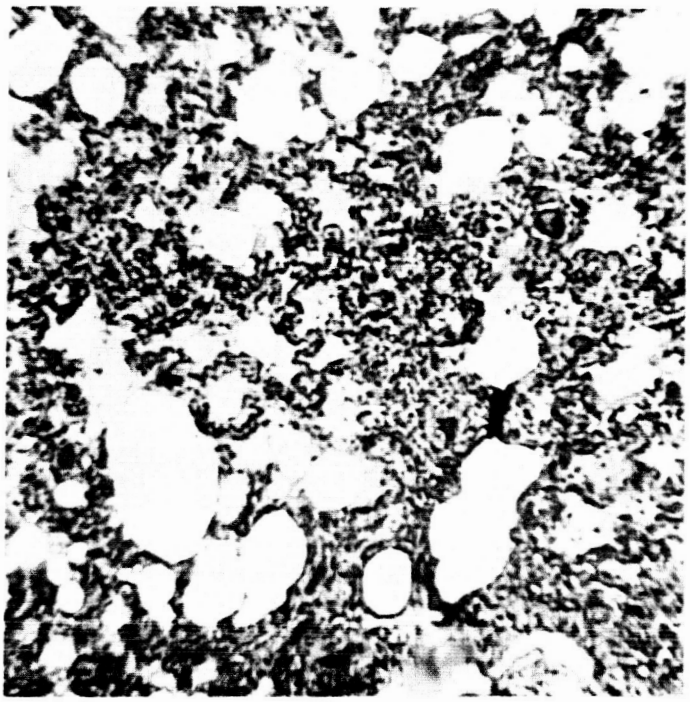


FIGURE 116. NO. 33. LUNG ATELECTASIS, CONGESTION (x90)

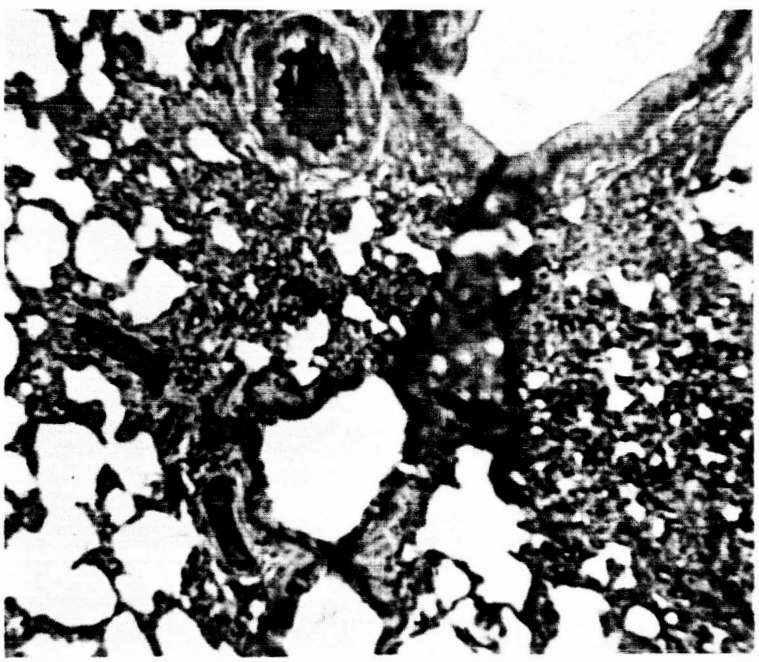


FIGURE 117. NO. 39. LUNG ATELECTASIS, AND CONGESTION (x135)

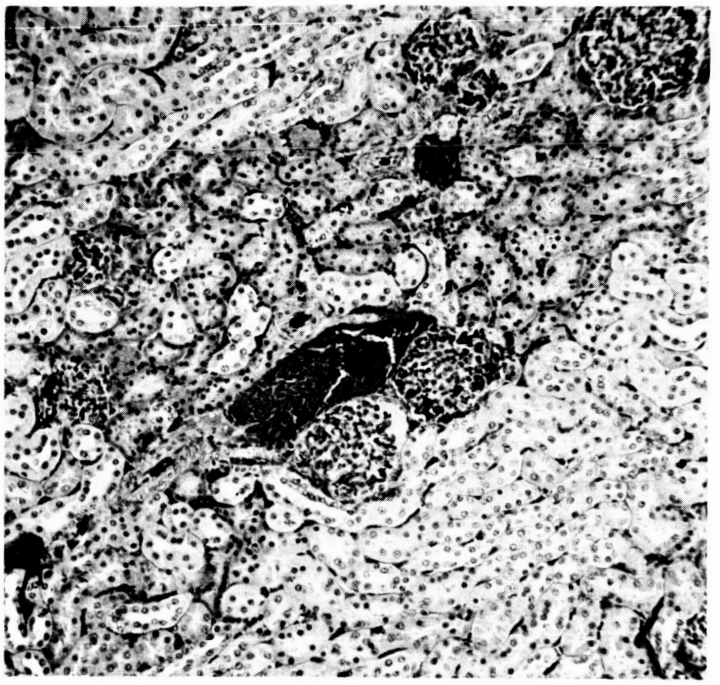


FIGURE 118. NO. 39. KIDNEY CONGESTION, FOCAL NECROSIS (x90)

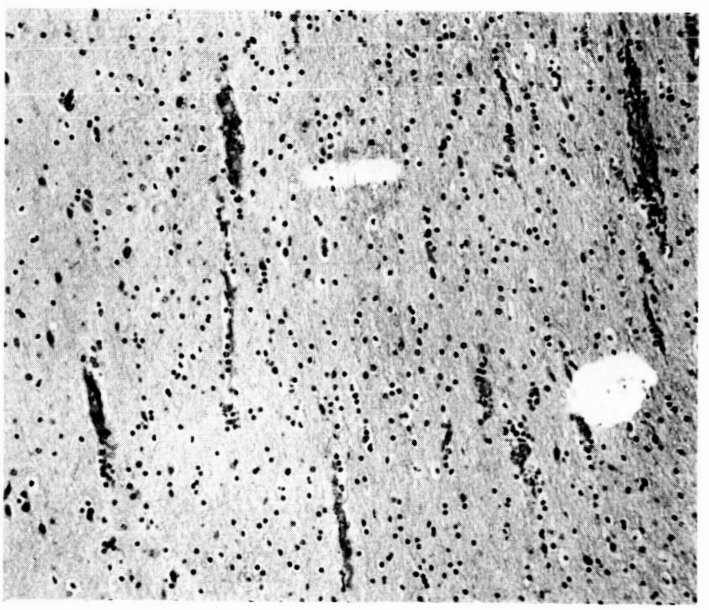


FIGURE 119. NO. 49. BRAIN CONGESTION (x90)

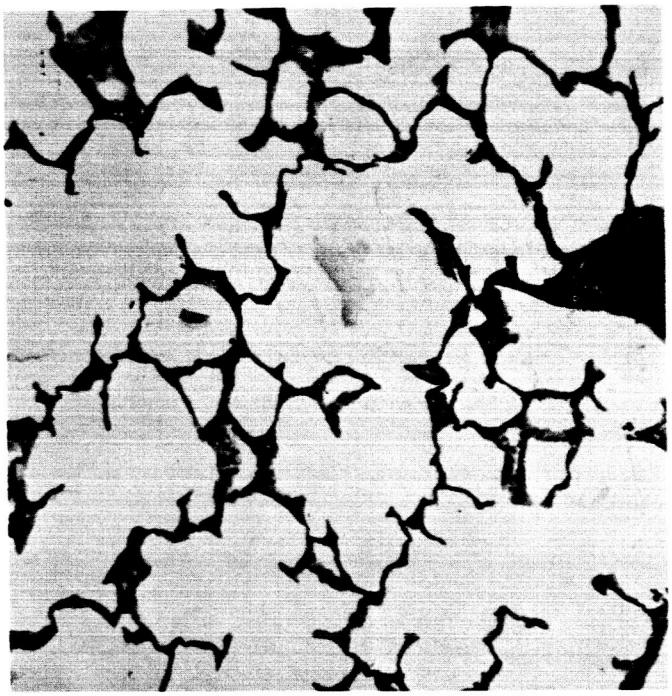


FIGURE 120. NO. 57. LUNG, ACUTE EMPHYSEMA (x90)

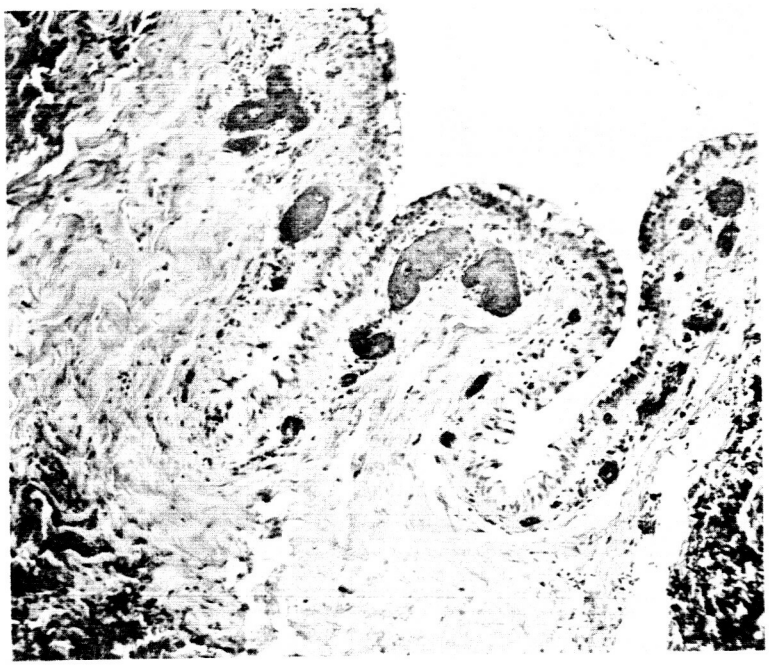


FIGURE 121. NO. 57. RESPIRATORY TRACT, HEMORRHAGE (x90)

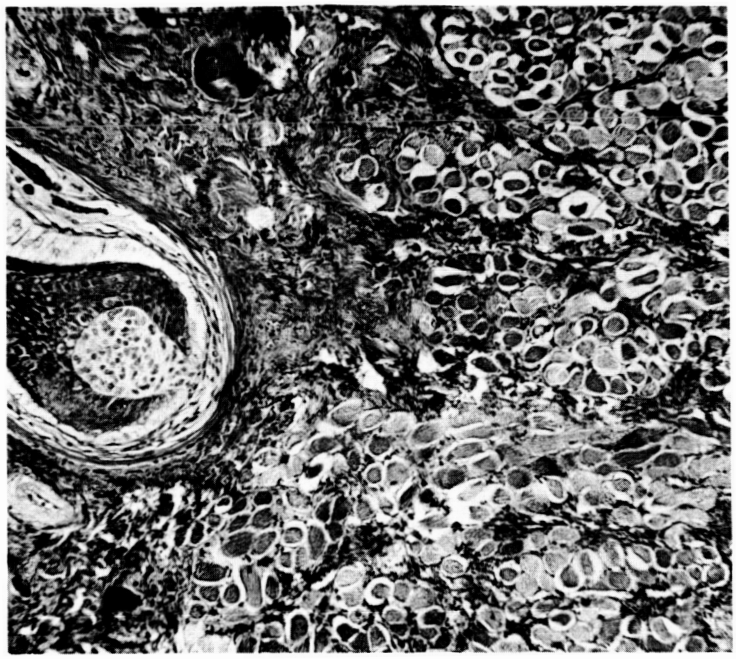


FIGURE 122. NO. 57. SKIN, HEMORRHAGE (x90)

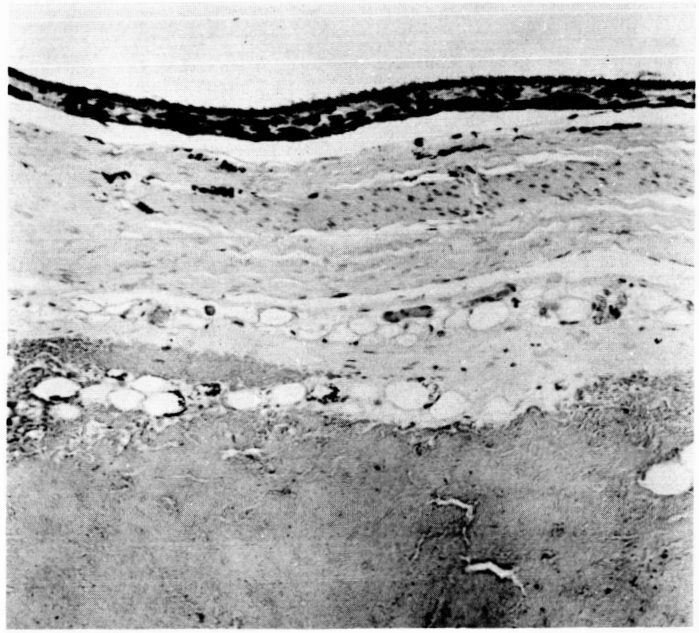


FIGURE 123. NO. 57. EYE, HEMORRHAGE (x90)

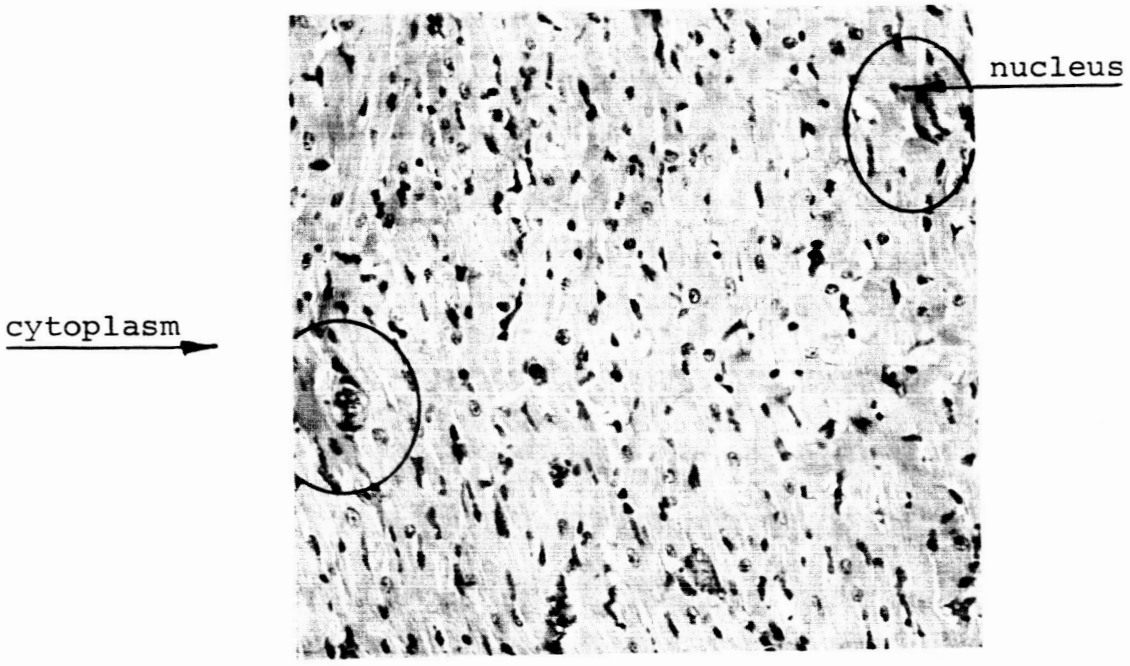


FIGURE 124. NO. 57. MYOCARDIAL NECROSIS AND HYPEREMIA (x90)

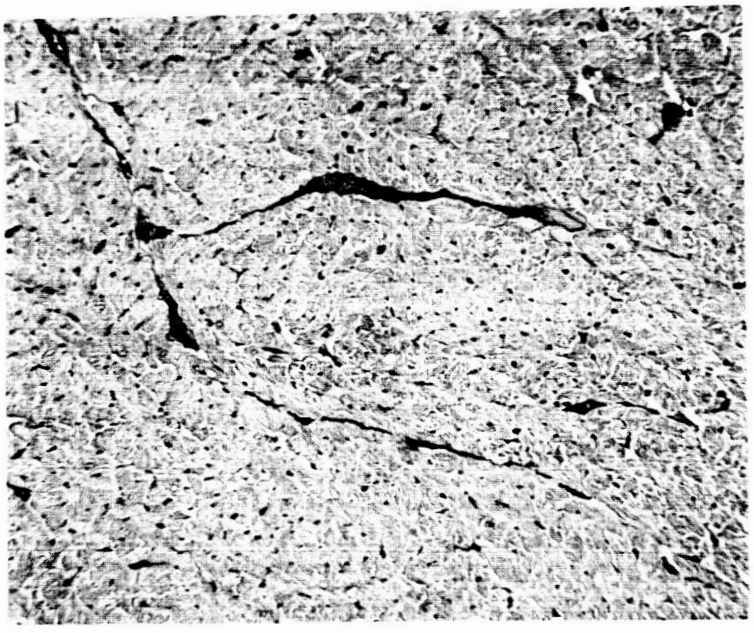


FIGURE 125. NO. 63. MYOCARDIAL NECROSIS AND CONGESTION (x90)

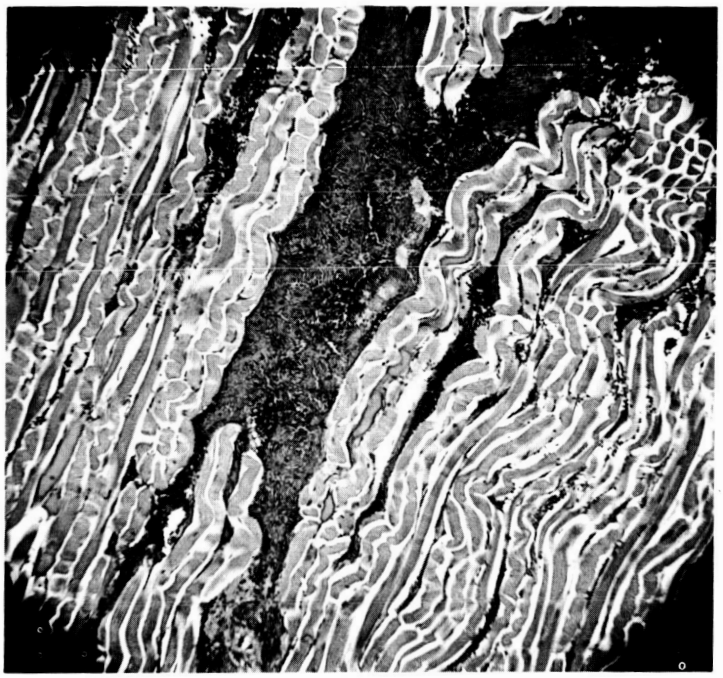


FIGURE 126. NO. 63. NECK MUSCLES, HEMORRHAGE (x90)

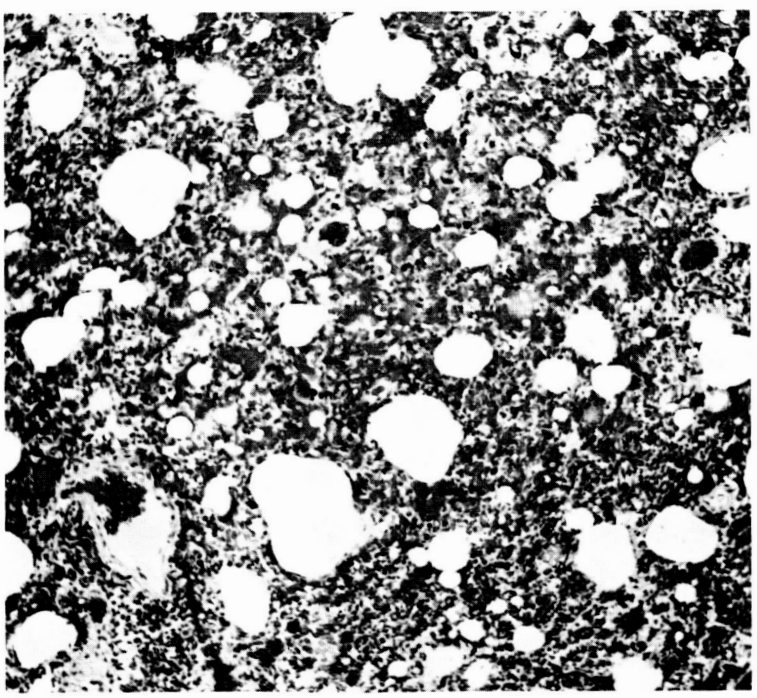


FIGURE 127. NO. 63. LUNG, HEMORRHAGE, AND ATELECTASIS (x90)

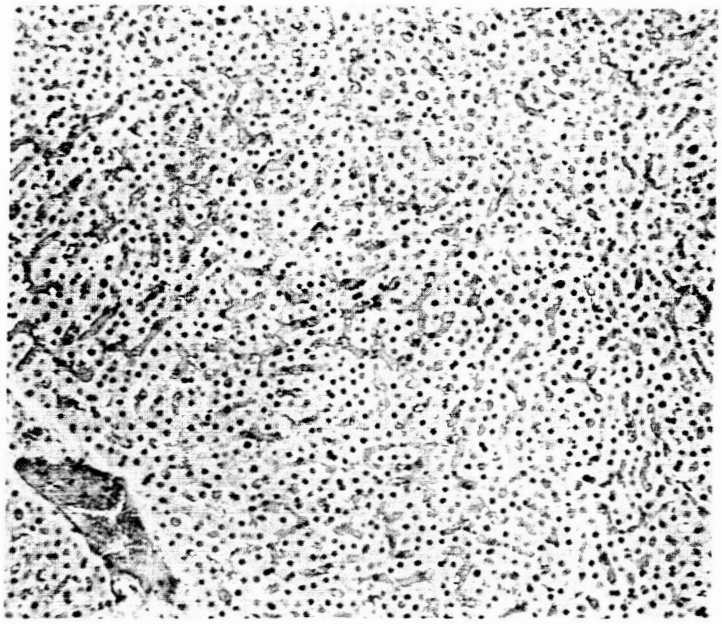


FIGURE 128. NO. 66. LIVER, CONGESTION (x90)

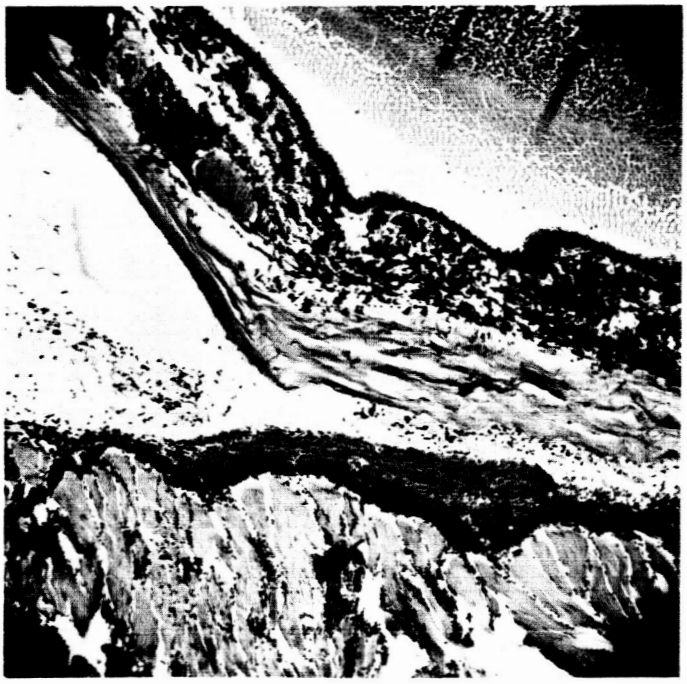


FIGURE 129. NO. 66.
HEMORRHAGE OF THE EYE MUSCULATURE AND RETNAL DETACHMENT (x90)

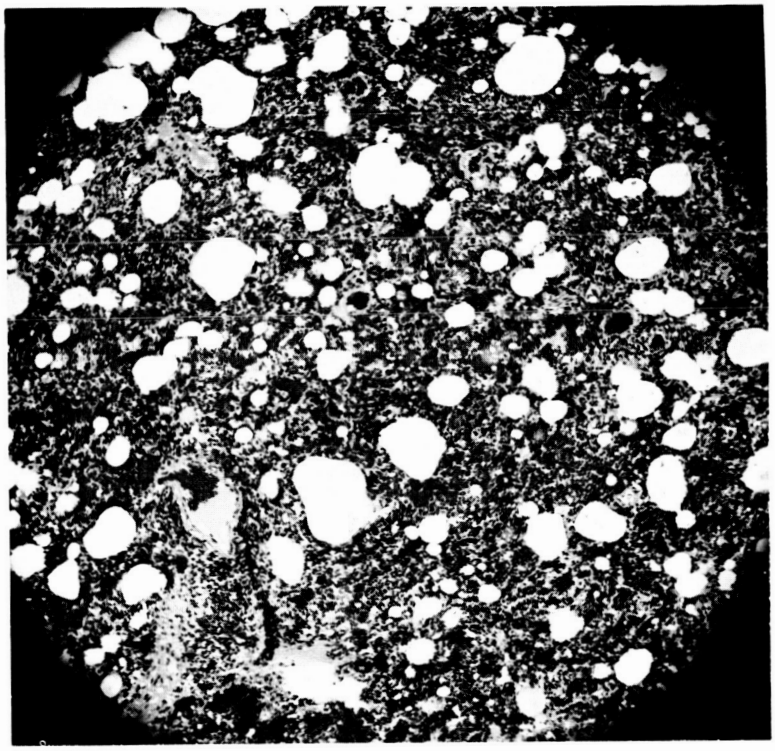


FIGURE 130. NO. 66. LUNG, HEMORRHAGE AND ATELECTASIS (x90)

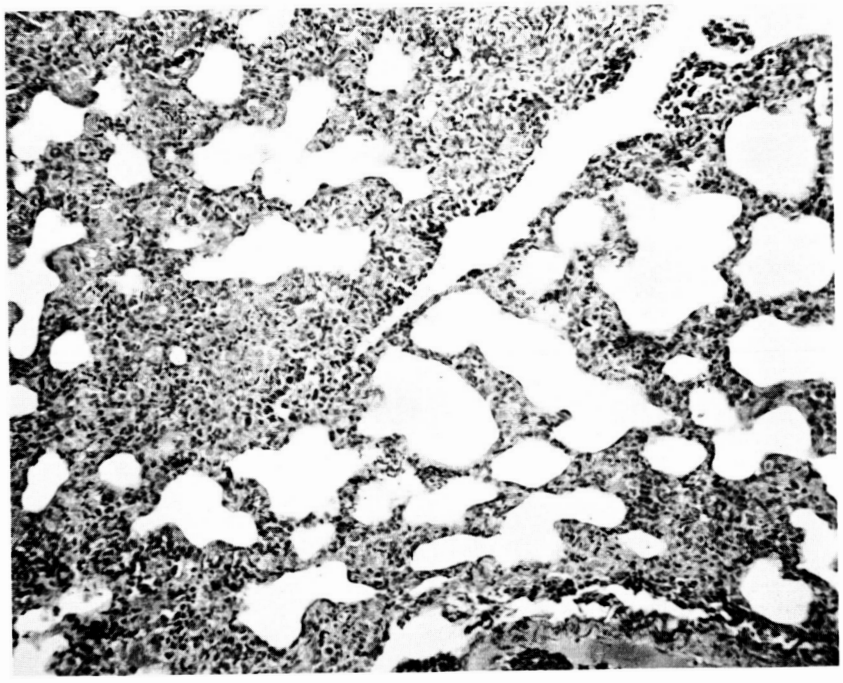


FIGURE 131. NO. 67. LUNG, HEMORRHAGE AND ATELECTASIS (x90)

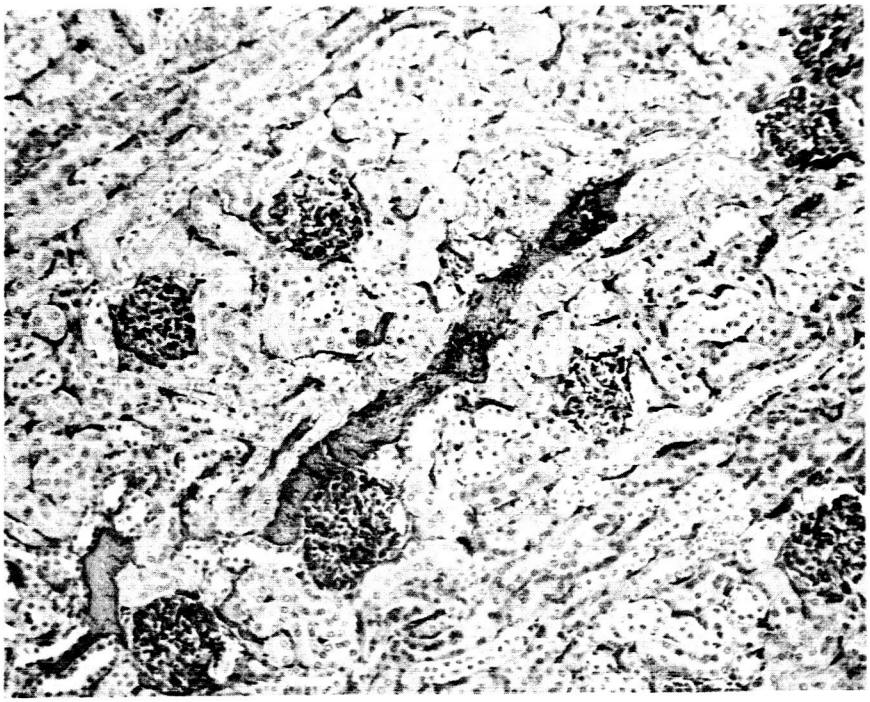


FIGURE 132. NO. 67. KIDNEY, CONGESTION, AND FOCAL NECROSIS (x90)

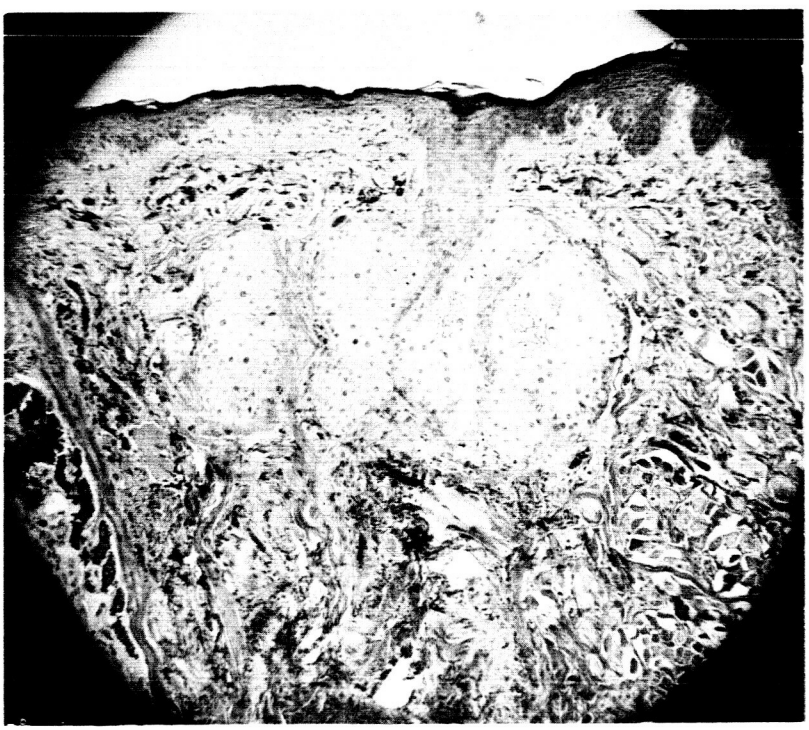


FIGURE 133. NO. 67. SHOWING SKIN HEMORRHAGE (x90)

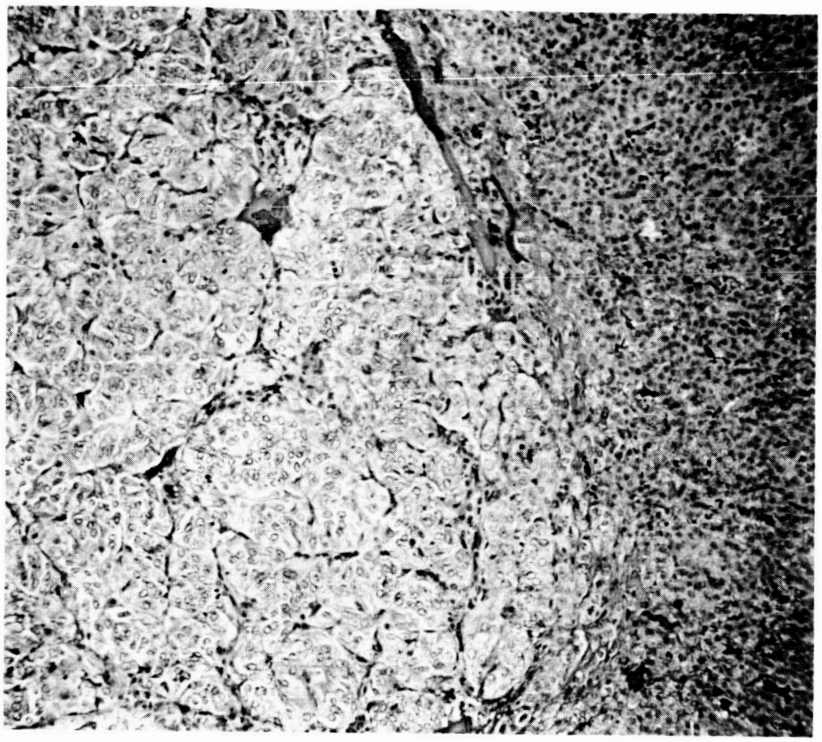


FIGURE 134. NO. 69. ADRENAL GLAND, CONGESTION (x90)

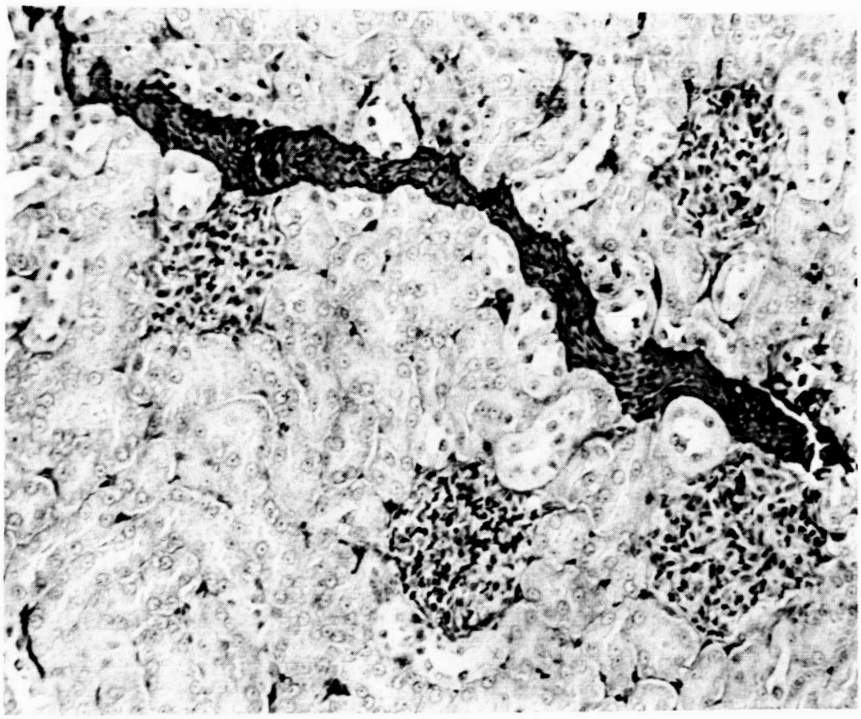


FIGURE 135. NO. 69. KIDNEY, CONGESTION AND FOCAL NECROSIS (x 135)

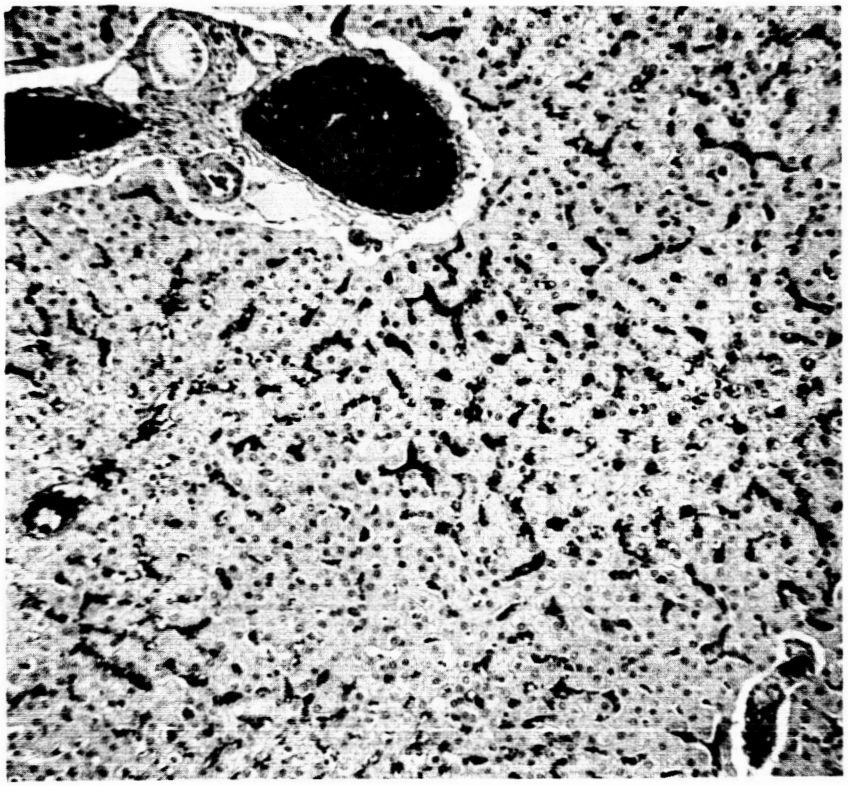


FIGURE 136. NO. 69. LIVER, CONGESTION (x90)

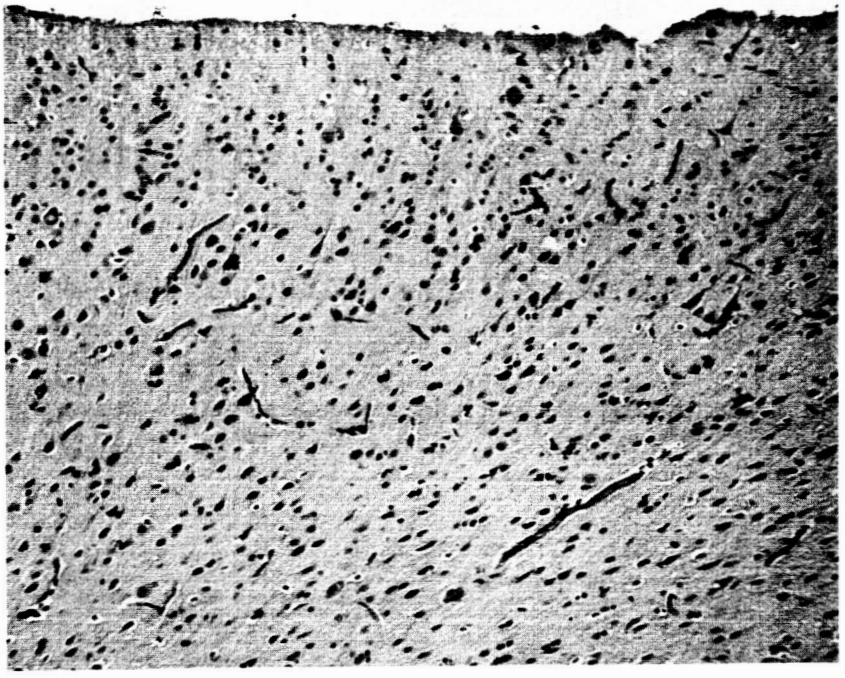


FIGURE 137. NO. 69. BRAIN, CONGESTION (x90)

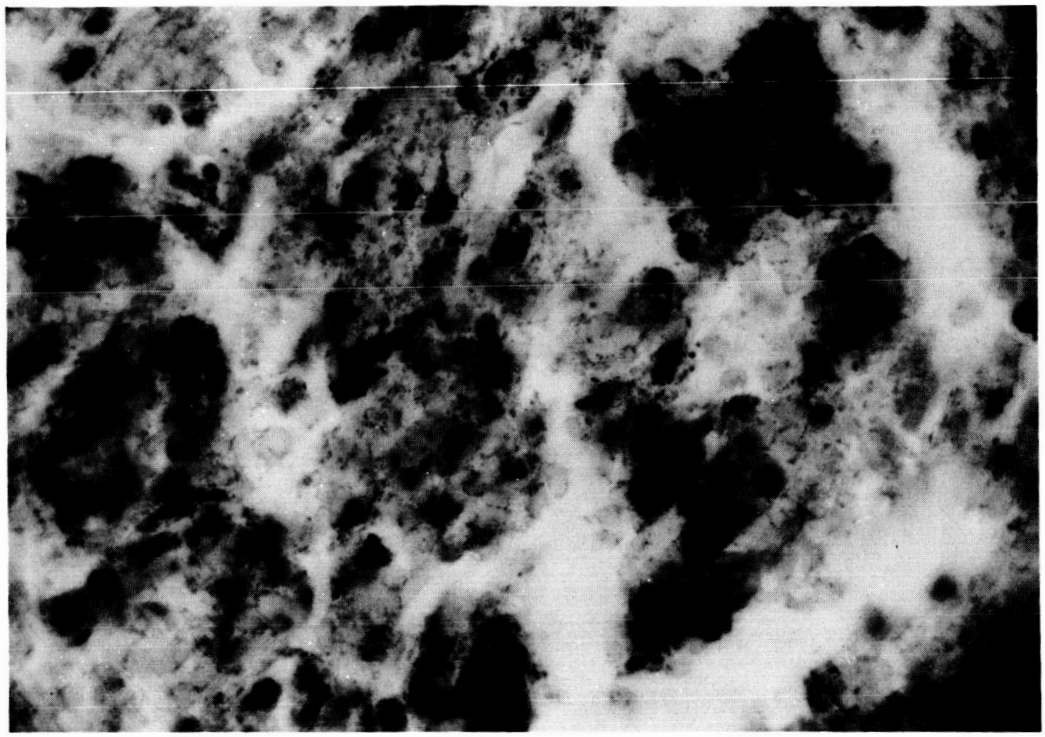


FIGURE 138. NO. 242. BRAIN, DPNH ENZYME LOSS, CLUMPING (x430)

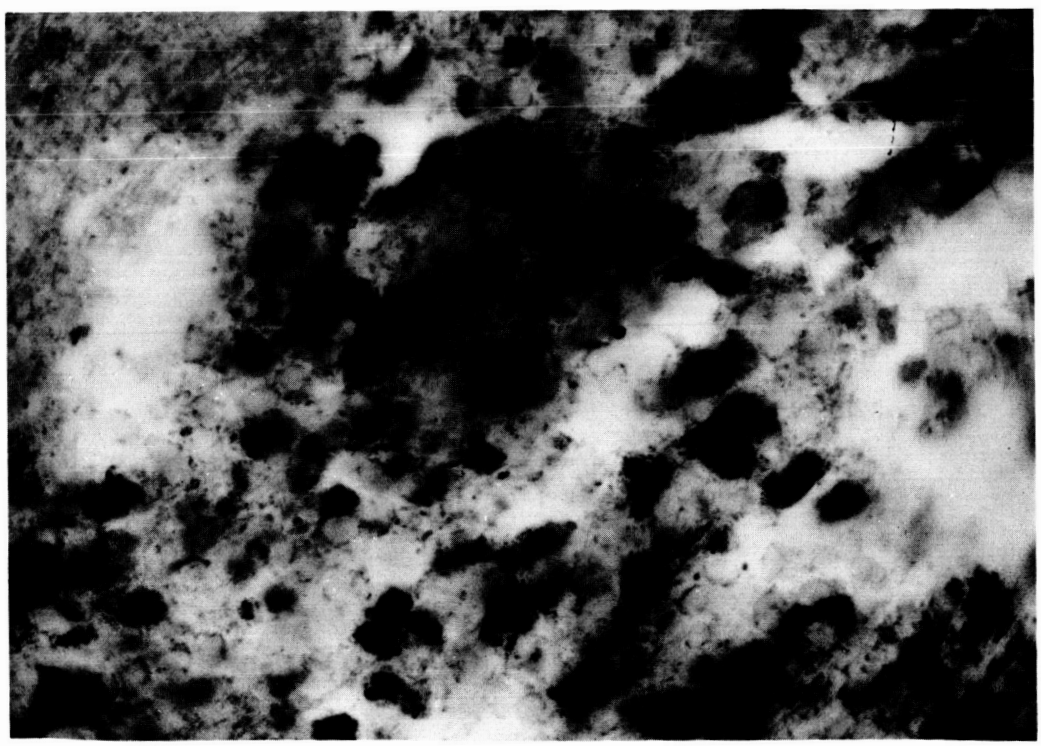


FIGURE 139. NO. 243. BRAIN, DPNH ENZYME, NORMAL (x430)

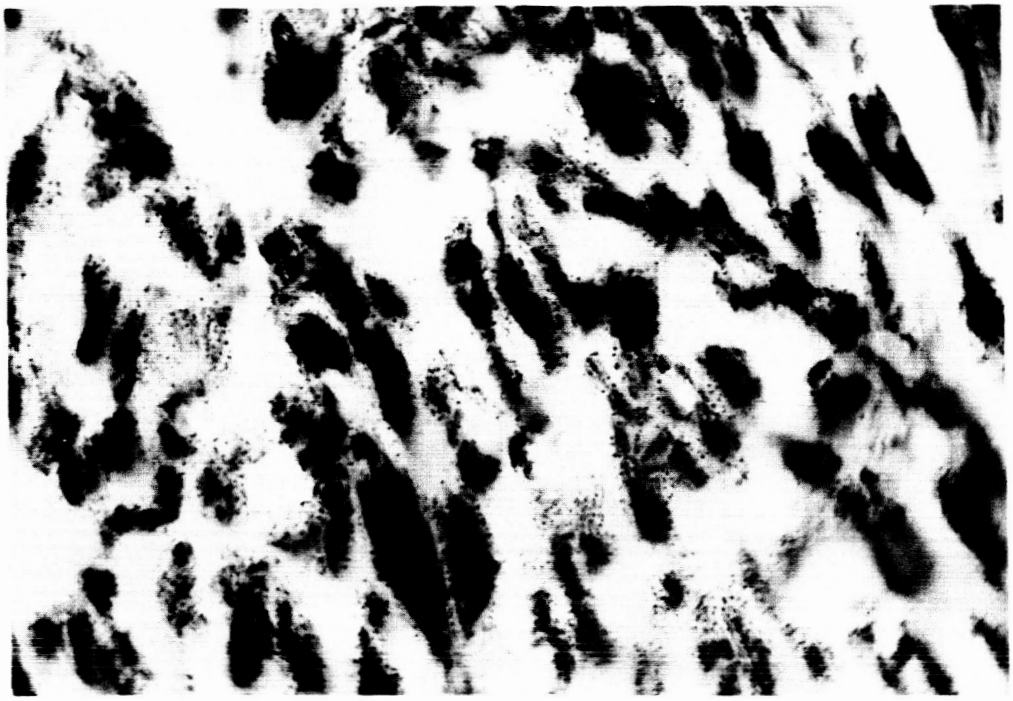
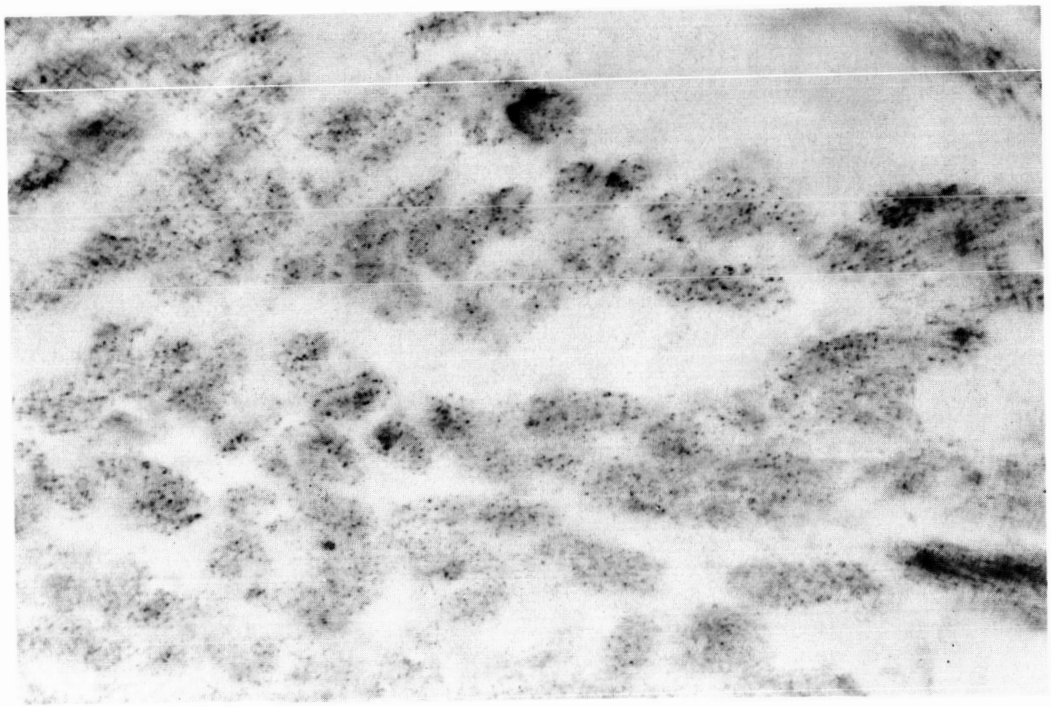


FIGURE 140. NO. 242. (x 430)
HEART, CLUMPING OF SUCCINIC DEHYDROGENASE ENZYME

FIGURE 141. NO. 243. (x 430)
HEART, NORMAL SUCCINIC DEHYDROGENASE ENZYME



(x430)

FIGURE 142. NO. 242. HEART, DECREASED MALIC ACID DEHYDROGENASE

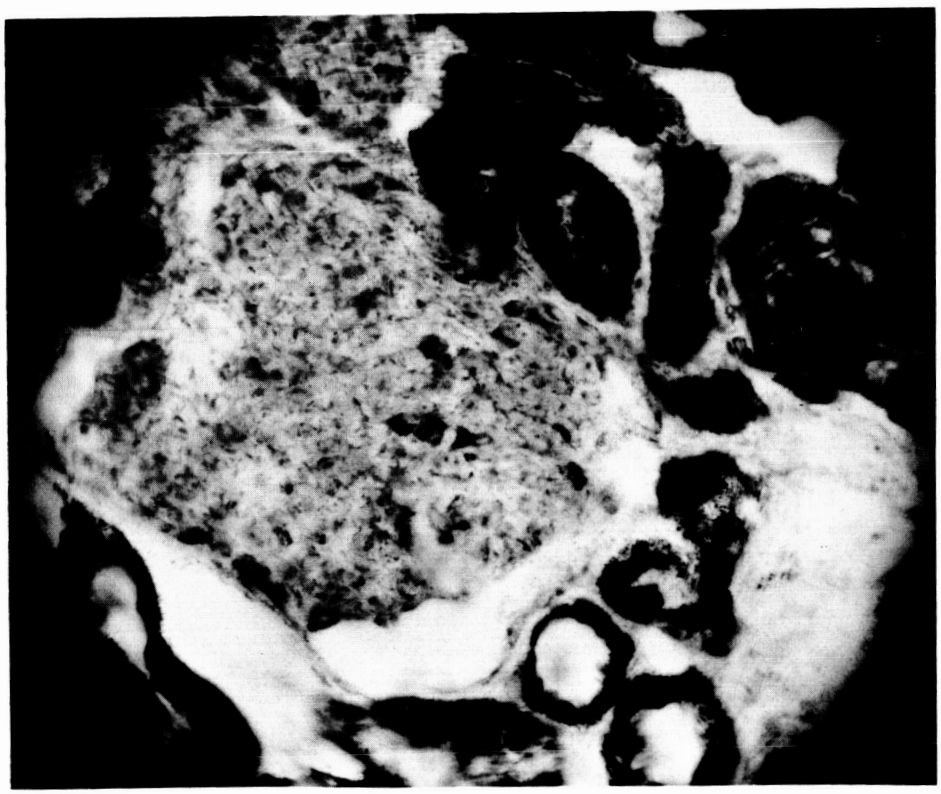


FIGURE 143. NO. 243. DPN, NORMAL KIDNEY (x 430)

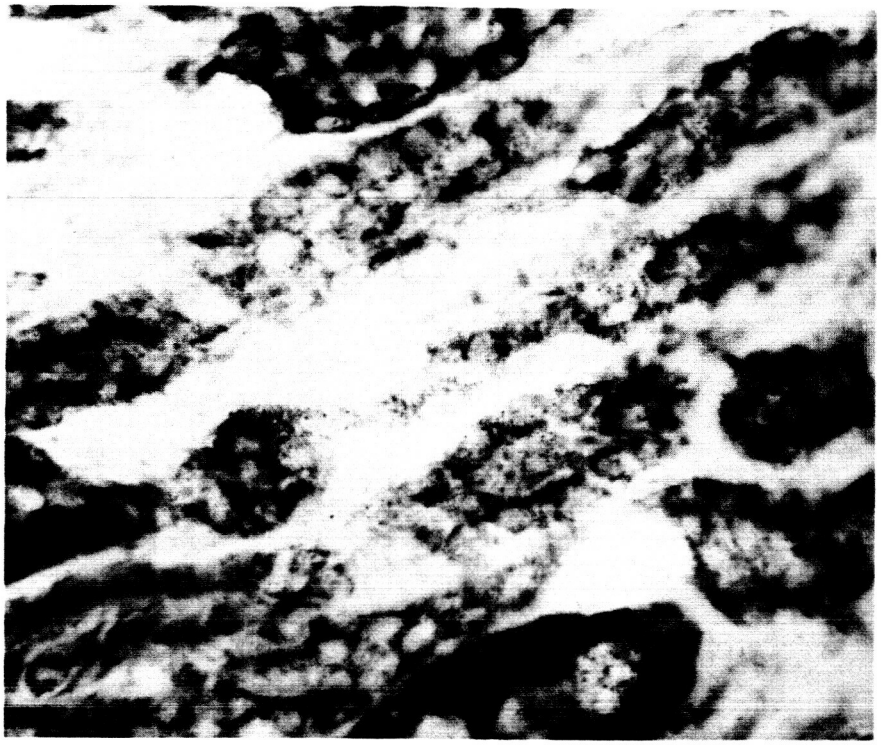


FIGURE 144. NO. 242. KIDNEY TUBULES, DPN LOSS (x235)

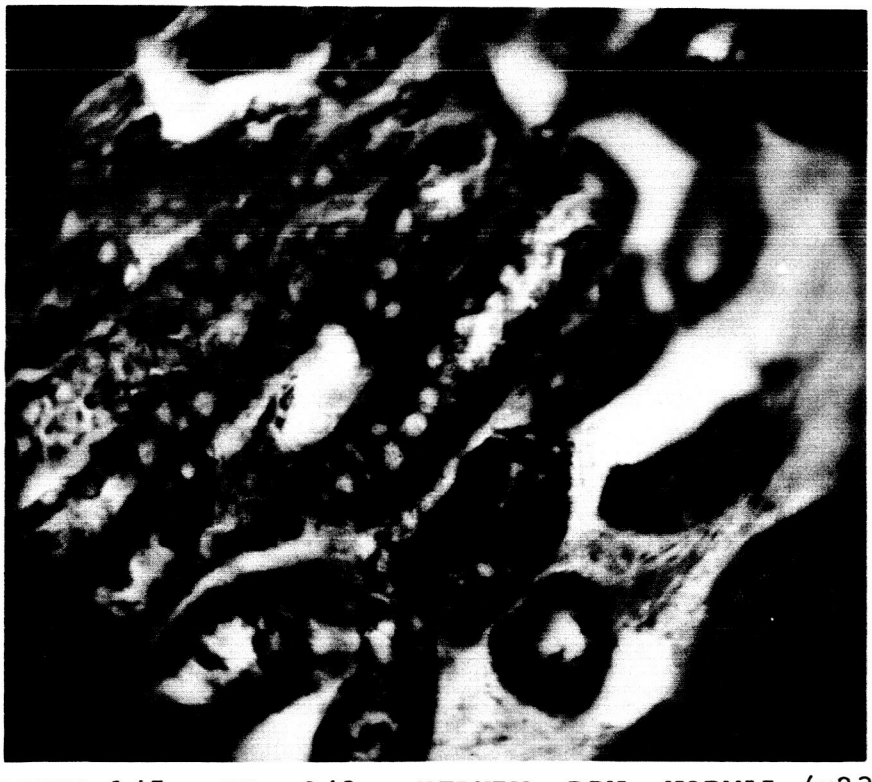


FIGURE 145. NO. 243. KIDNEY, DPN, NORMAL (x235)

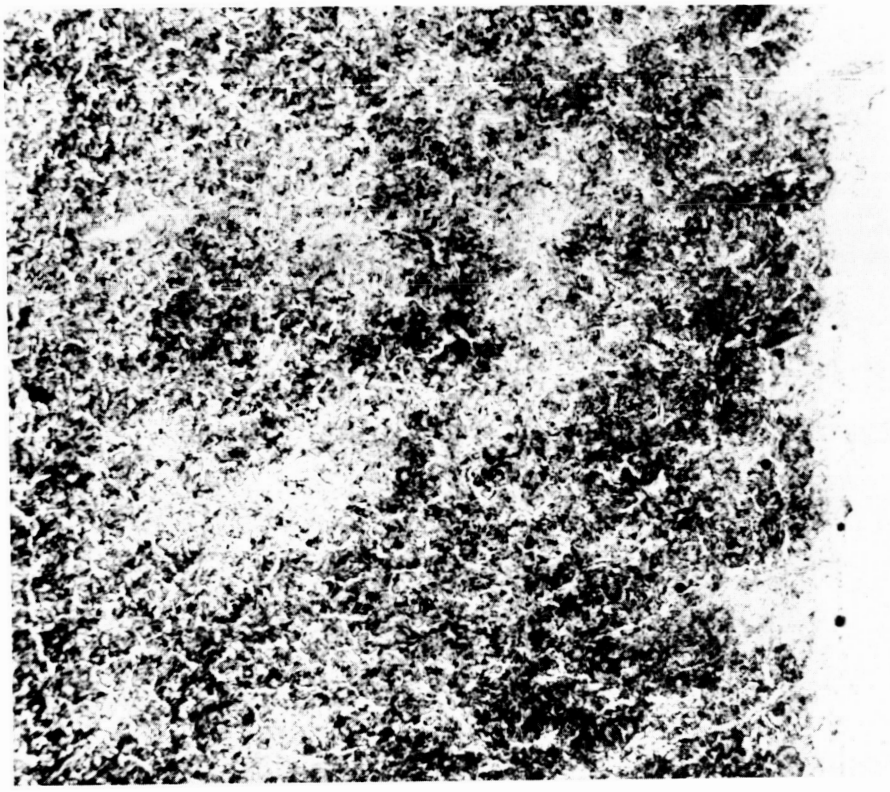


FIGURE 146. NO. 242. ADRENAL, SUDAN BLACK, LIPID LOSS (x135)

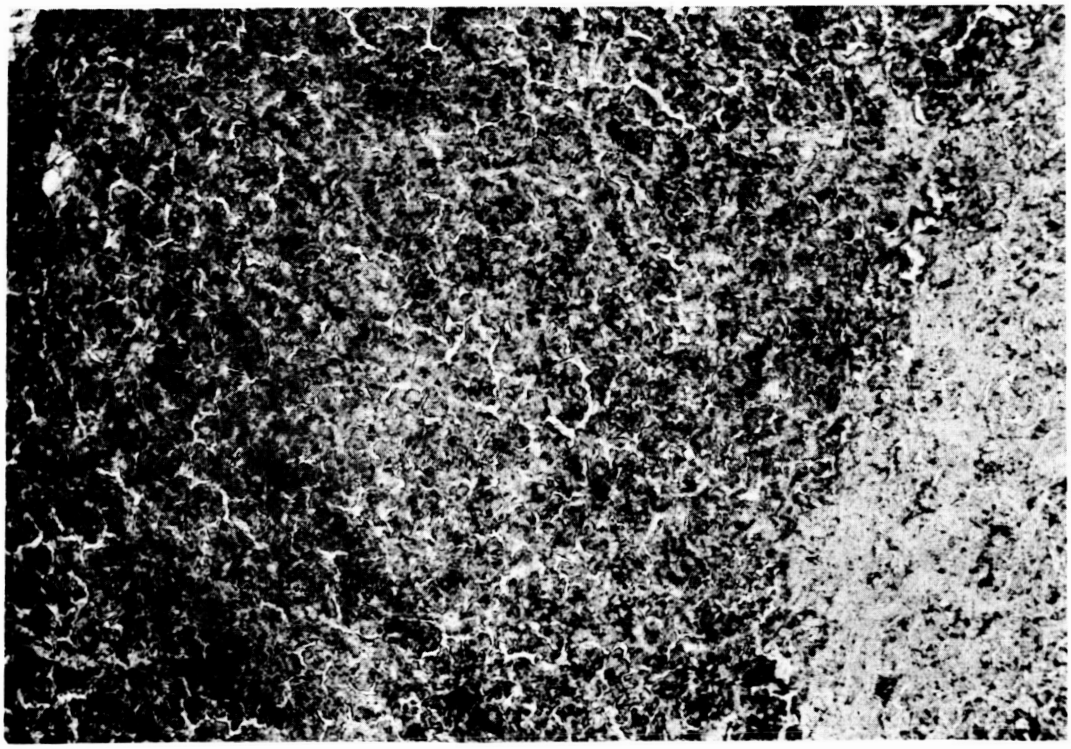


FIGURE 147. NO. 243. ADRENAL, SUDAN BLACK, NORMAL LIPID (x135)

APPENDIX F
MATHEMATICS

This appendix includes the laboratory working curves constructed during the course of the experiment as well as curves summarizing the experimental findings.

The laboratory working graphs, Figures 148 to 163, show Δ_{rt} plotted against weight. The vertical abscissa is divided in units of time (seconds) greater than (+) or lesser than (-), a value of $\Delta_{rt}=0$, (see text for explanation). The horizontal abscissa represents animal weight in grams. The D_t curve is included on each graph as is the D_r-D_t family of curves. These are provided for reference purposes.

A survivor is designated by a small circle, a fatality by a small pyramid, and a cliff hanger by a small pyramid with an arrow pointing to it.

Figures 164 to 172 are of the K_p versus weight type graphs for 50 to 400 G's. These graphs show the final distribution of experimental results in all modes. The legend identifies each mode and each result as to survivor or fatality.

Each of these graphs stressed the point that K values consistent with survival increased from left to right with increasing weight. Also stressed is that K values consistent with survival increased with G stress. Finally these graphs also show that a range of lethal dosage exists for each stress level. A brief discussion of the figures in this appendix follows.

Figure 148.- Figure 148 is a Δ_{rt} versus weight graph for the 50 G profile. This graph was used to apply to the $+G_x$, $+G_y$ and $-G_y$ modes. On it are drawn (D_r-D_t) curves for D_r of 2 seconds, 20 seconds, 200 seconds, 300 seconds, and 400 seconds. On this graph the zero line represents Δ_{rt} equal to zero, i.e., the difference between the run time and the threshold time is zero. The graph shows that for all weights of the test subject a Δ_{rt} of approximately -100 and below virtually assured animal survival.

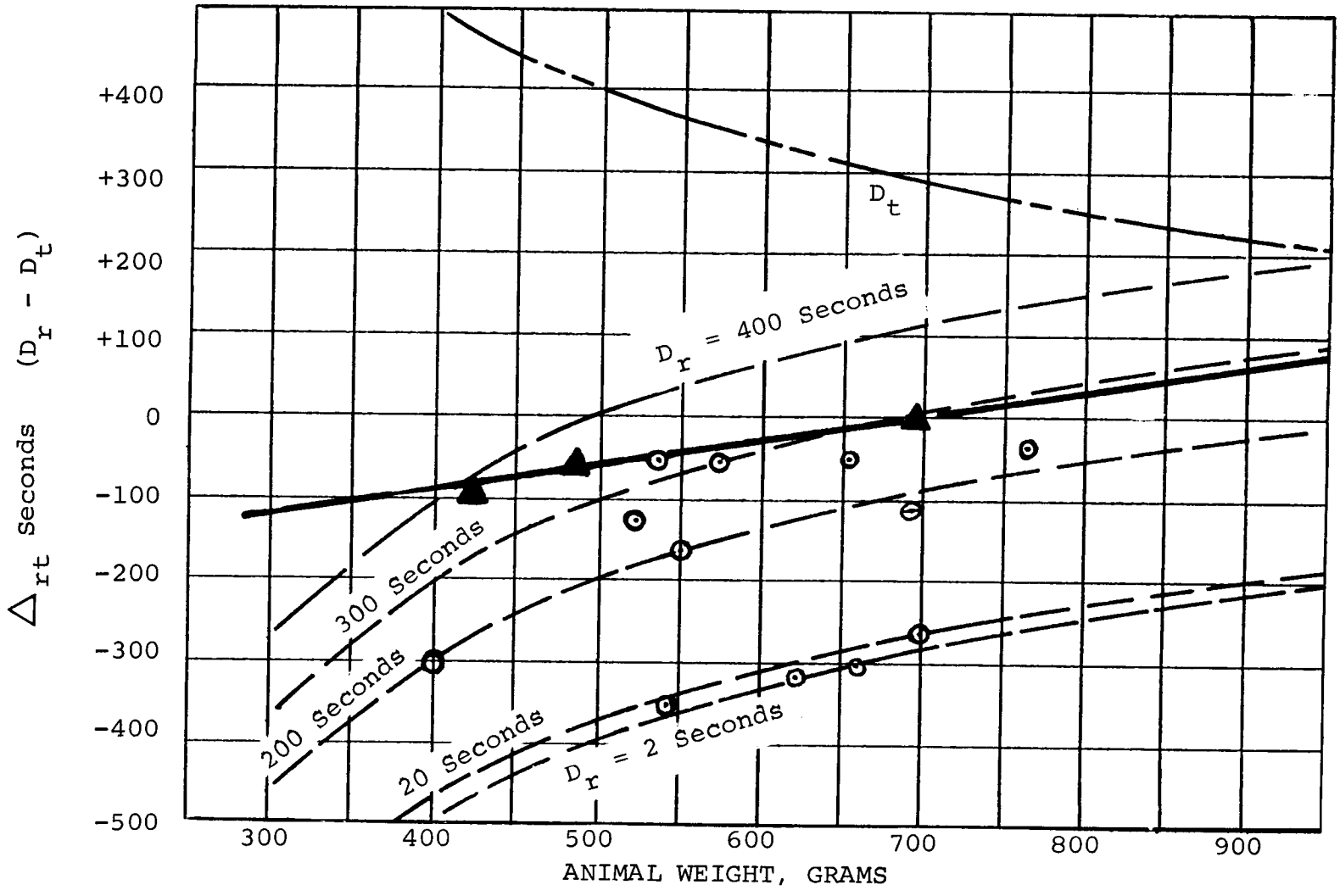


FIGURE 148 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
50 G CASE

The left hand edge of the graph shows losses occurring at -100 Δ_{rt} at approximately 440 grams, and -70 Δ_{rt} at approximately 480 grams. A line drawn between the various losses shows a progression of losses occurring with decreasing Δ_{rt}, negativity of (or a progression in the positive direction for Δ_{rt}) as weight increases to the right. The slope of this line is approximately 10^{o}. The inference drawn from this curve is that the initial assessment of K_t equal to 1.0×10^{4} is essentially correct.

Figure 149.- Figure 149 is a similar type of graph for 100 G's for the $+G_x$, $+G_y$, and $-G_y$ modes. Here we see a shift of the Δ_{rt} curve upwards and to the left for D_r of 2, 20, 200, and 300 seconds. An animal loss is recorded at approximately 550 grams and at Δ_{rt} equals zero (in other words at threshold). Three survivors occurred just above threshold values of K, but at a very minimal difference of Δ_{rt} (on the order of 20 seconds). A loss occurred at a Δ_{rt} of +65, at 735 grams. Again a line drawn between the losses from one edge of the spectrum to the other parallels the findings in the 50 G curve, namely a sloping line of approximately 10^{o} with a positive slope. Inference of this particular working graph is that again the selection of K_t equal to 1.0×10^{4} is essentially correct for 100 G's.

Figure 150.- Figure 150 is a similar Δ_{rt} versus weight graph for 150 G's; reference curves for D_r of 2, 20, 200, and 300 seconds are again drawn. In this case, as in the 50 and 100 G graphs, a D_t curve is also included. We see a lowering of the D_t curve toward the Δ_{rt} line equal to zero and a generalized flattening of the curve. This graph shows an interesting grouping of events occurring at the approximate optimum weight of the monkey, namely between 500 and 600 grams, at values of Δ_{rt} of 50 to 65 plus. There is a grouping of four survivors just below a grouping of four losses. A line drawn again from the left hand side of losses to the right hand side of the graph shows a slope of approximately 10^{o}. The inference of this graph is that for 150 G's K_t equal to 1.0×10^{4} is a little bit low; in

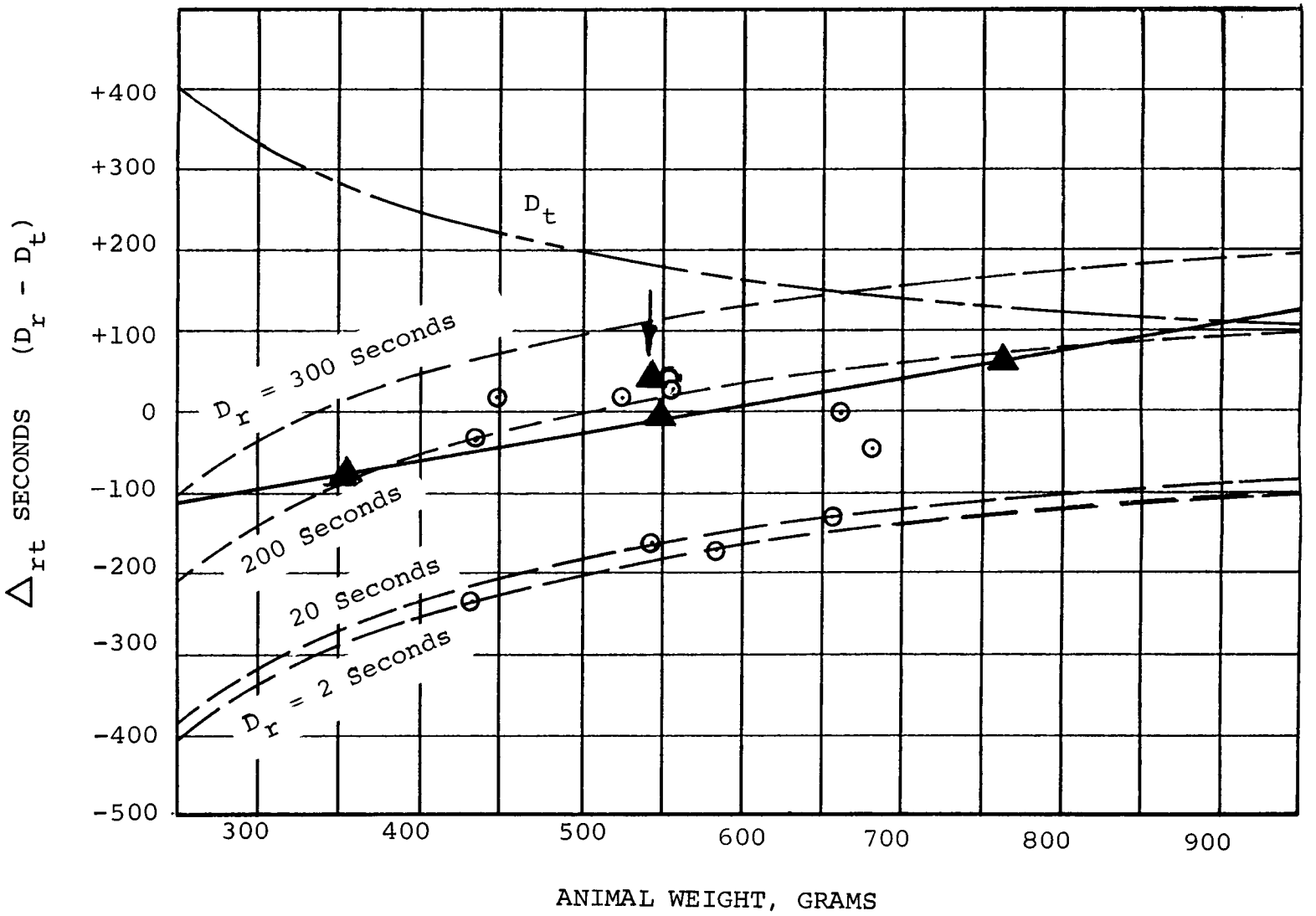


FIGURE 149 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
100 G CASE

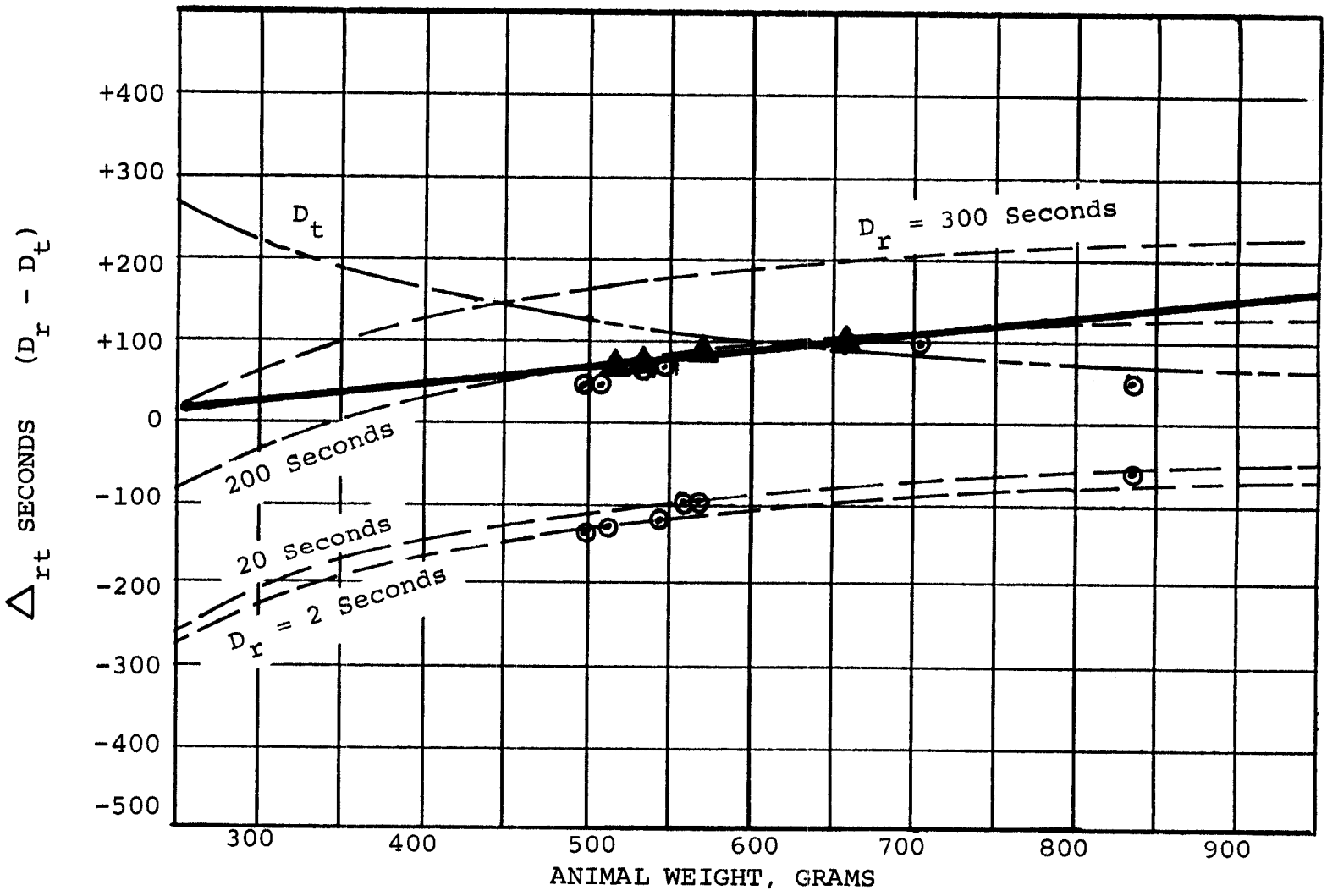


FIGURE 150 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
150 G CASE

other words, K threshold should have a higher value.

Figure 151.- Figure 151 is a D_r-D_t graph versus weight for 200 G's. Here reference curves for D_r of 2, 20, and 200 seconds are drawn. Animals whose Δ_{rt} values are in the minus Δ_{rt} regions are survivors. At values of Δ_{rt} of approximately +100 a fine distribution of survivors and losses occur within a few seconds of each other in the range of 450 grams to approximately 750 grams. There is a general flattening of the reference curves as well as of the D_t curve; and now the slope of the line connecting the losses at the left and right hand ends of the weight spectrum appear to flatten from 10^{o} to approximately 5^{o}. The interesting thing about this graph is that it supports the case for an increased value of K_t at the 200 G level. Furthermore, it appears that the particular animal under study can tolerate 200 G's better than it can any other G thus far studied in that in weight ranges from 450 to 700 grams only one loss occurred at a value of approximately +100 Δ_{rt}.

Figure 152.- Figure 152 is for the 250 G range of exposures. Here a greater flattening of the reference curves for D_r of 2, 20, and 200 seconds is noted. The D_t curve is also flatter. The shift noted in this graph is most interesting in that values of Δ_{rt} just above 100 seconds plus, a very striking grouping of events has occurred. This grouping occurs between 400 grams and 600 grams where the graph indicates 4 fatalities, 4 survivors, and 4 cliff-hangers. (By cliff-hangers is meant animals which survive the run are seen to be breathing immediately after the run, and which then proceed to die within the first half-hour after the run.) The D_t and reference curves approach each other such that the margin of safety may be interpreted as becoming less.

Figure 153.- Figure 153 represented the 300 G profile. Here there is a greater flattening of all curves, reference as well as D_t. A line drawn from the left to the right hand end of the spectrum connecting fatalities again achieves a value of approximately 10^{o} positive slope. All values of Δ_{rt} below zero are those of survivors. Interesting enough, it is seen that a value of Δ_{rt} equal to +100 seconds is a safe exposure from 400 to 700

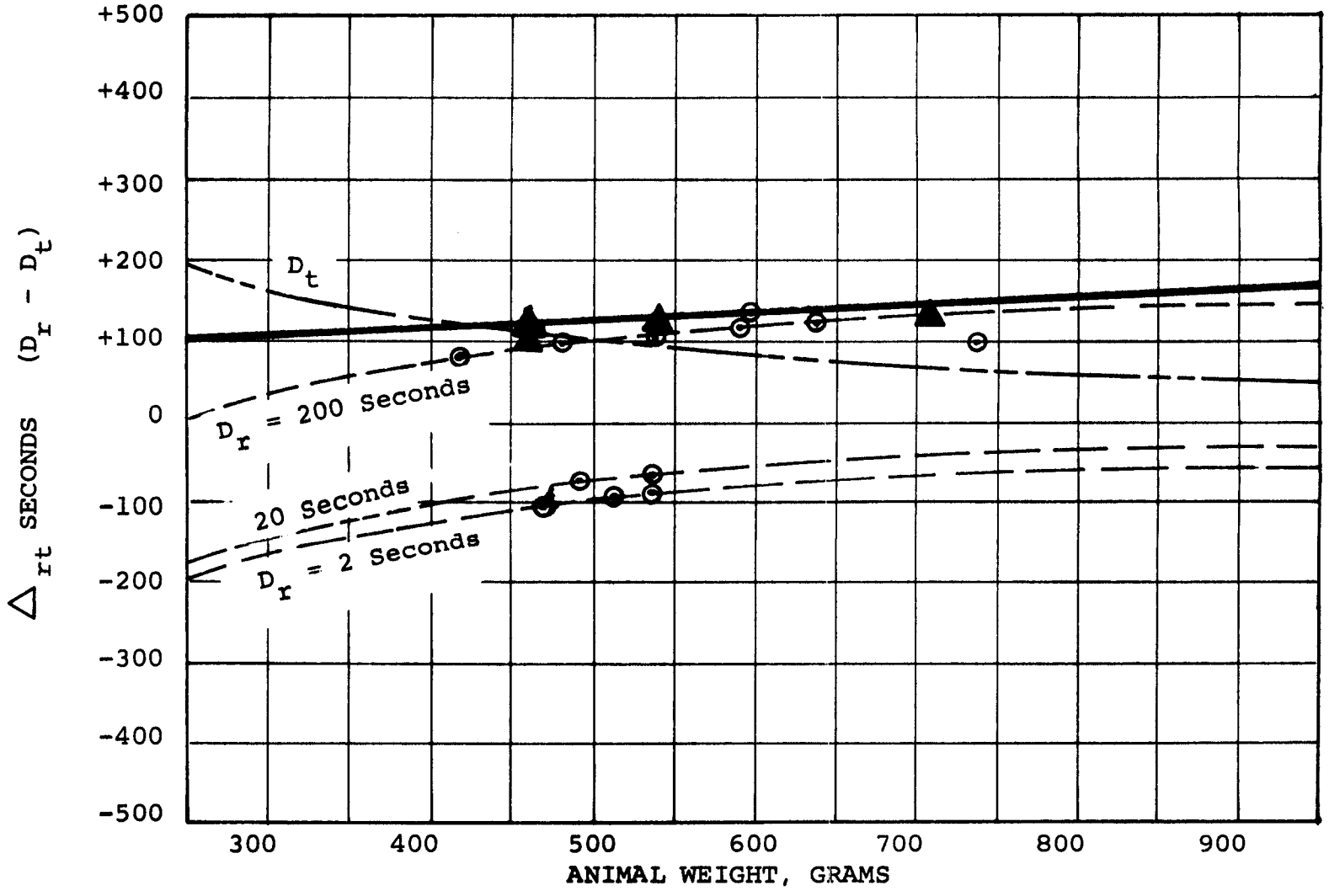


FIGURE 151 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
200 G CASE

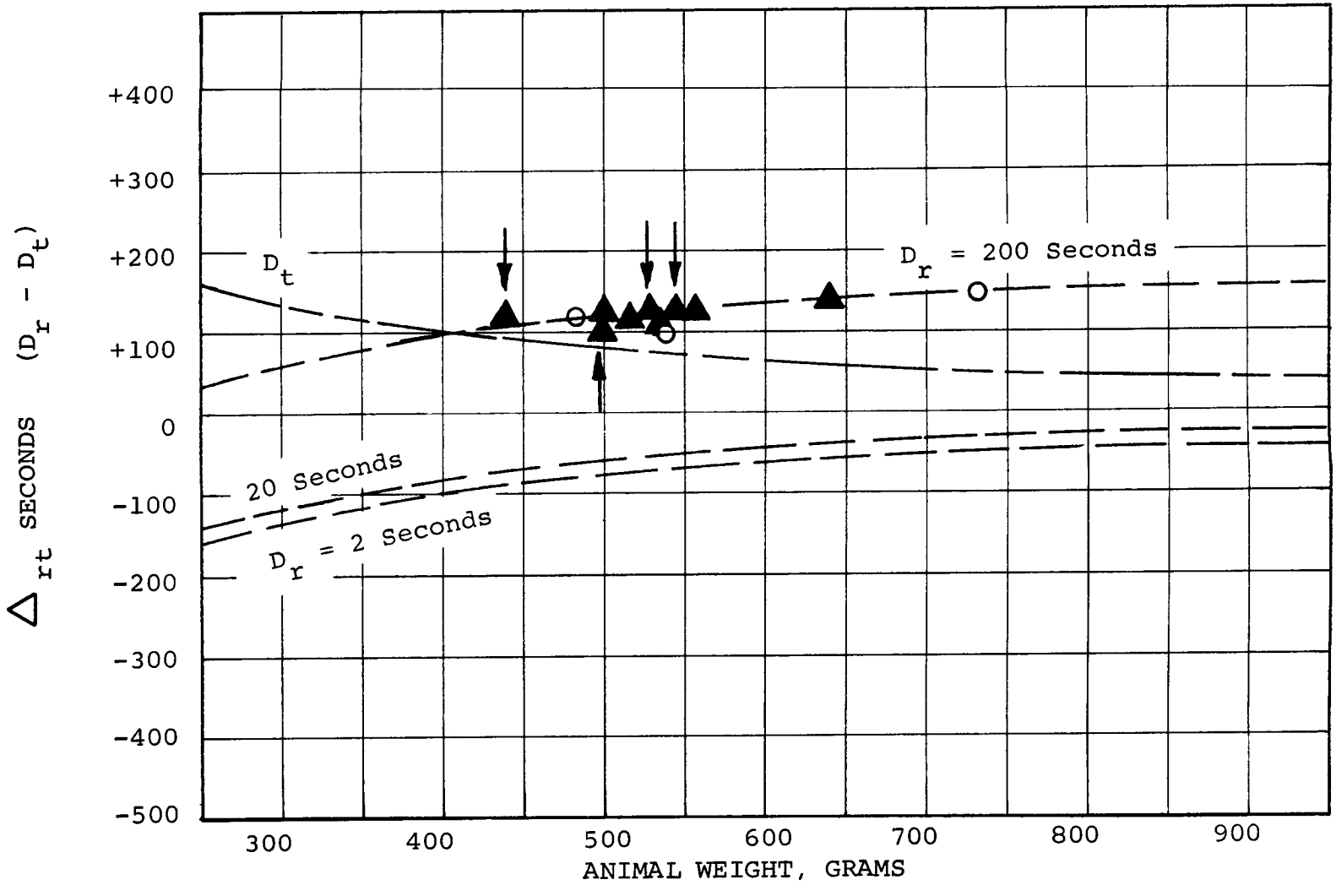


FIGURE 152 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
250 G CASE

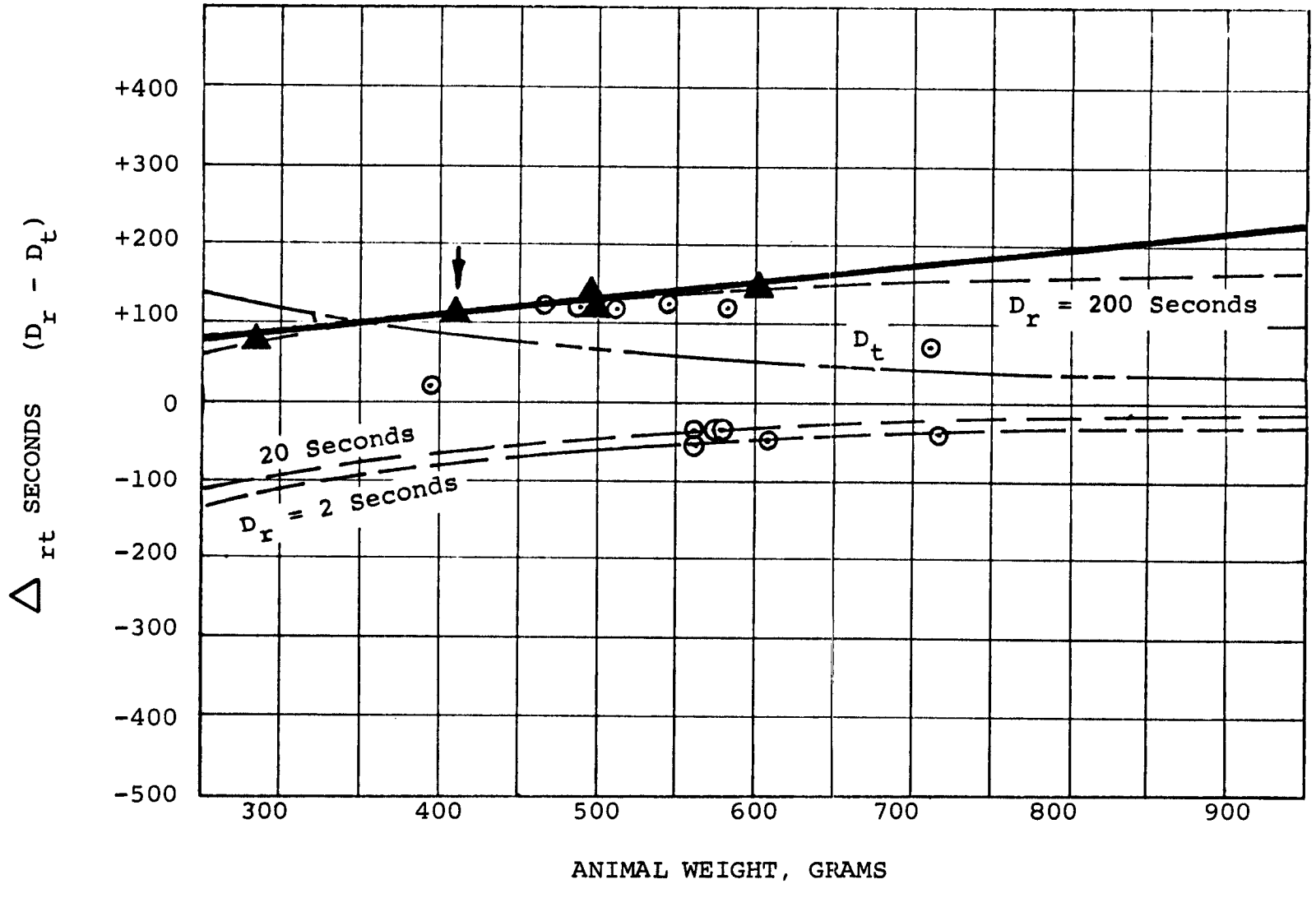


FIGURE 153 EXPOSURE ABOVE THRESHOLD VERSUS ANIMAL WEIGHT
300 G CASE

grams, whereas at the left hand edge of the curve it is a lethal value. Here an animal weighing approximately 280 grams was overcome at a Δ_{rt} of +75 seconds. Otherwise, a fine grouping of events occurs all along the plus 100 second Δ_{rt} line, where only one death occurs at the 400 gram level. One cliff-hanger is also at this level of weight, six survivors are above the 100 second line, and three deaths occur above the 100 second mark. The inference drawn from this graph is that a higher K_t can be selected for this 300 G profile level. Furthermore, there appears to be a limit to the K which the animal is capable of absorbing at the left hand edge of the weight spectrum. The slight negative slope of K at the right hand end of the weight spectrum is less dramatic, but noteworthy nevertheless.

Figure 154.- Figure 154 represents the 350 G profile. The curves for reference and D_t are flatter, the slope of the line connecting the left hand edge to the right hand edge of fatalities is flatter (5 degrees) and the sharper zone of fatalities to the left and to the right of the 500 gram mark at Δ_{rt} values of 100 seconds and above is noted. The inference here is that as progression to the left or to the right of the 500 gram mark takes place, not only do the Δ_{rt} values decrease, but so does the K_p. This may indicate that an optimum weight exists for the absorption of this type of stress.

Figure 155.- Figure 155 is for the 400 G profile and is a dramatic illustration of the preceding principle. At above +100 seconds Δ_{rt}, a very sharp drop to the left and to the right of the 500 gram mark is noted on the graph. The inference here is that apparently there is an optimum weight, an optimum G, an optimum time of G load application that applies to this particular animal.

Figure 156.- Figure 156 is a construction of the 50 $-G_x$ values of G loading with Δ_{rt} again on the left abscissa and weight along the horizontal abscissa of the graph. Initial selection, as previously stated, of K_t equal to 4.0×10^3 is indicated by the zero line. Most interestingly, it is noted that values above this K_t are achieved, both at the left and at the right hand ends of the weight spectrum. Three survivors exist below the zero line. The inference here is that perhaps instead

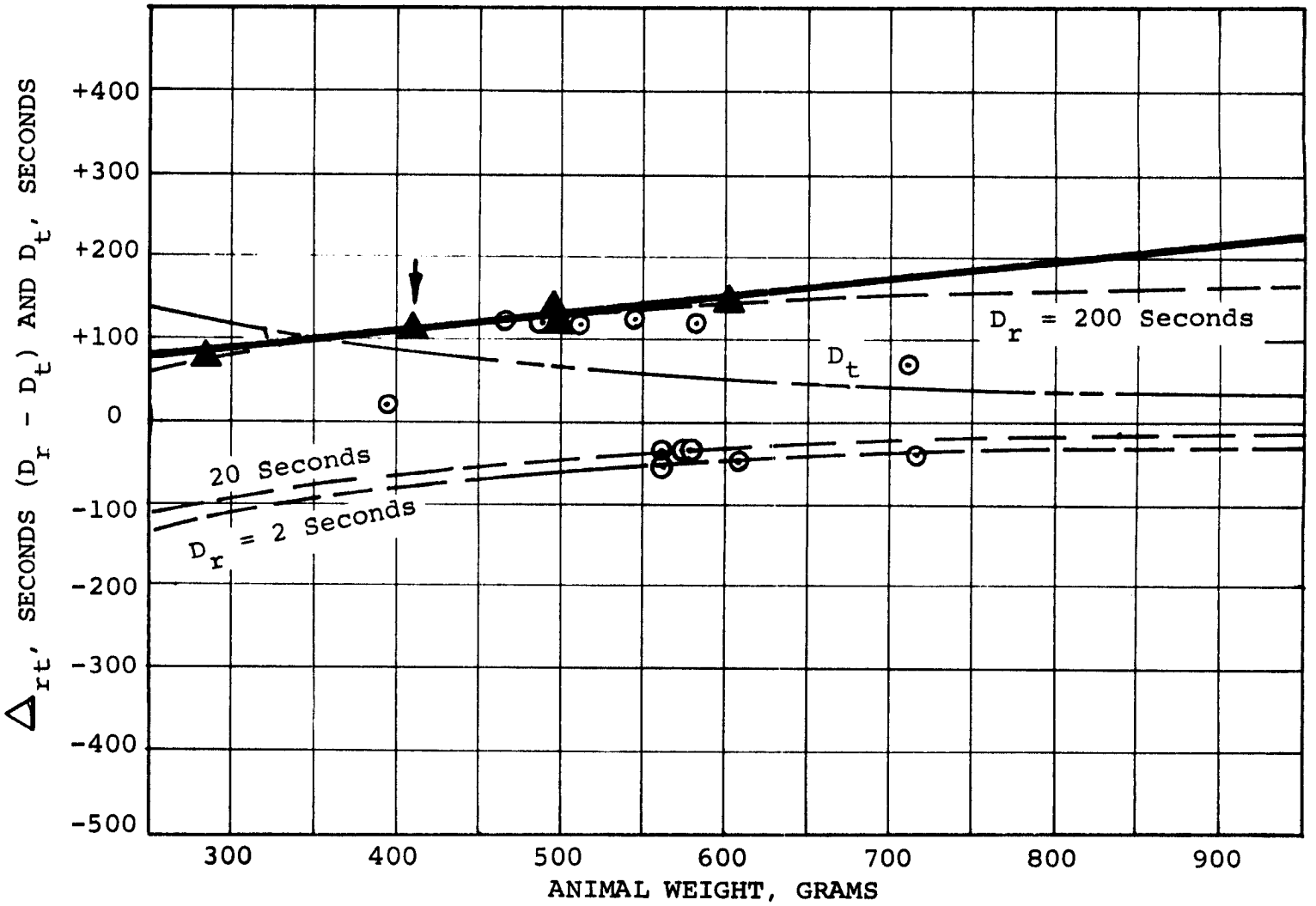


FIGURE 154 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
350 G CASE

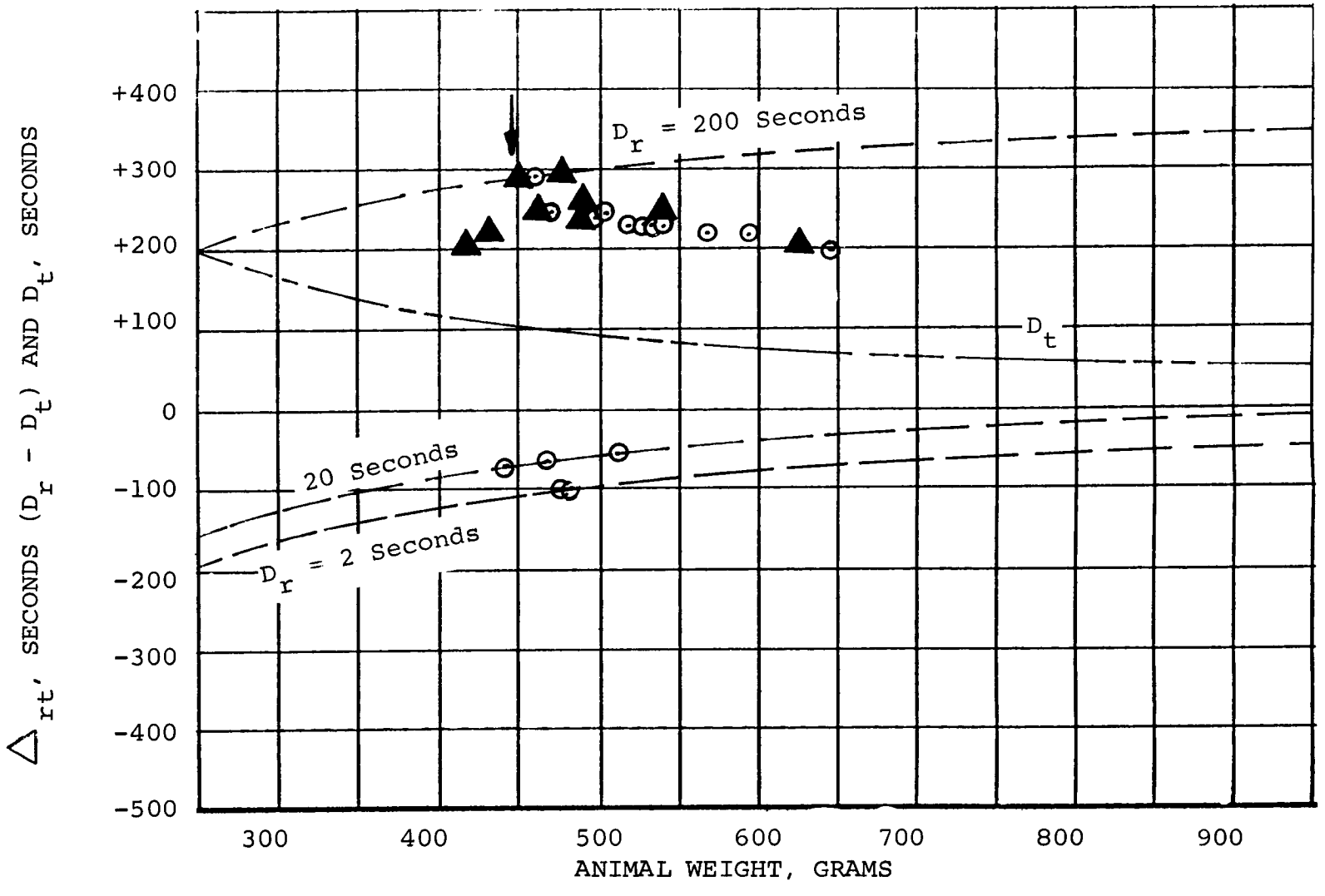


FIGURE 155 EXPOSURE ABOVE THRESHOLD VERSUS ANIMAL WEIGHT
400 G CASE

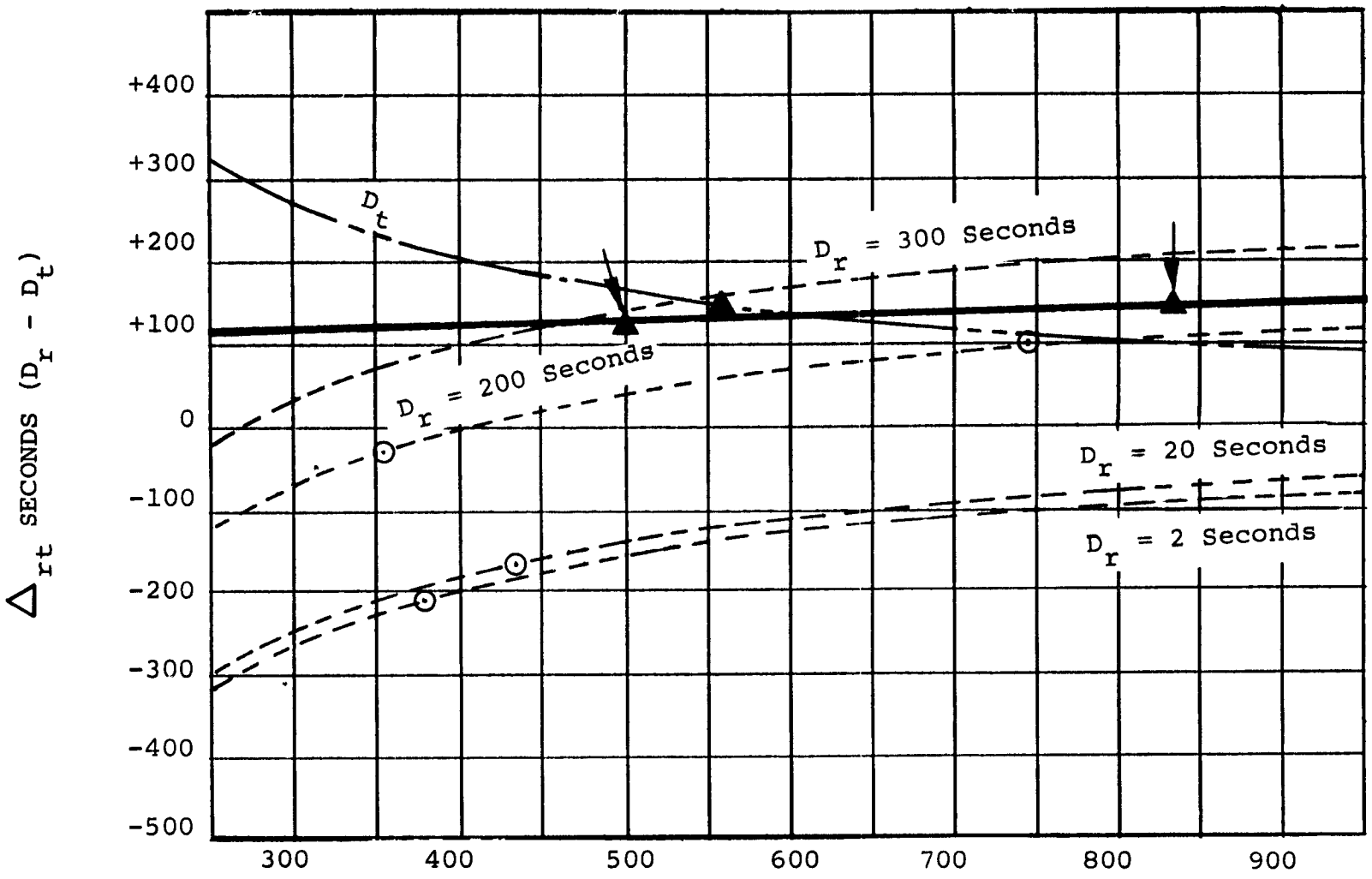


FIGURE 156 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-50 G_x CASE ONLY

of a K_t equal to 4.0×10^3, the range of values of K_t for $-G_x$ made at 50 G's, quite possibly ranges from 7.1×10^3 on the left to 1.0×10^4 on the right. Note the slope of the line connecting the fatalities which appears to be 5 to 10^o.

Figure 157.- Figure 157 is for 100 $-G_x$ and shows that the selection K_t of 4.0×10^3 is essentially conservative, because there are three survivors above the Δ_{rt} zero line, and one cliff-hanger at the optimum weight. Again, there is a slight positive slope of the line connecting the fatalities. The loss at the left-hand edge is at K value of 7.0×10^3.

Figure 158.- Figure 158 is a Δ_{rt} graph for 150 $-G_x$. Here, one loss is recorded at +55 seconds which is possibly artifactual. Since several survivors occur above this value of Δ_{rt}, this curve illustrates the capability of the animal to achieve values of K above 1.0×10^4.

Figure 159.- Figure 159 is a Δ_{rt} versus weight graph for 200 $-G_x$. This graph illustrates a gradual rise in the total Δ_{rt} value capable of being absorbed by this animal in the $-G_x$ mode. Animals with Δ_{rt} values in the minus zone are again survivors, but so are animals exposed to Δ_{rt} values of +100. The first fatality in this series of investigations did not occur until a K_p of 2.84×10^4 (Δ_{rt} + 175 sec.) was achieved. The inference of this curve is that, again, the value of K_t of 4.0×10^3 for the $-G_x$ mode was most conservative. Also illustrated is that the animal appears capable of absorbing 200 G's rather well.

Figure 160.- Figure 160 is a Δ_{rt} versus weight graph for -250 G_x. This graph provides us with a great deal of information in that a cliff-hanger and a death occur at +100 Δ_{rt}, whereas four survivors occur below this level and one death occurs above. The implication of this particular curve is that there is a lessening of the K value in the $-G_x$ mode up to and beyond 200

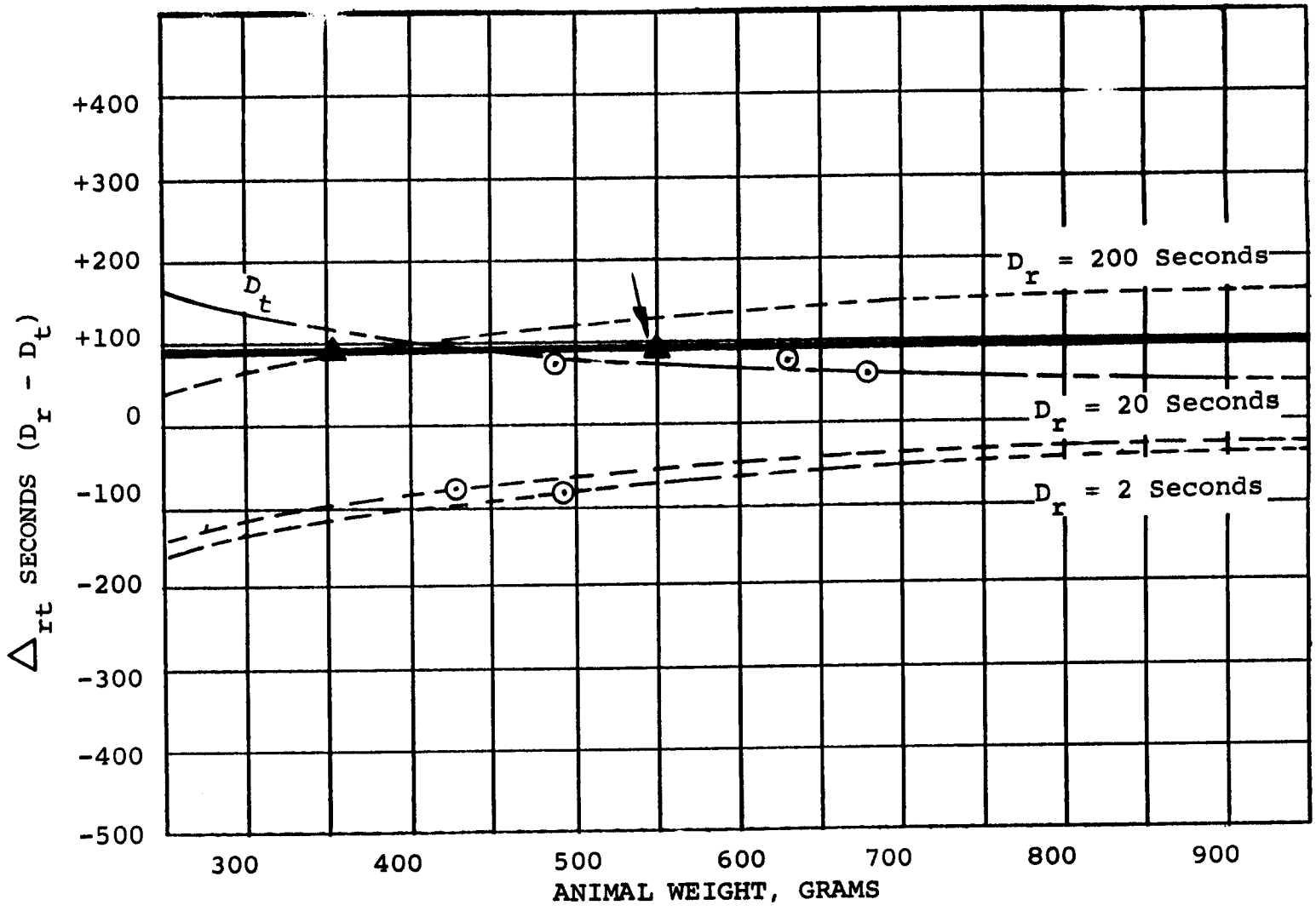


FIGURE 157 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-100 G_x CASE ONLY

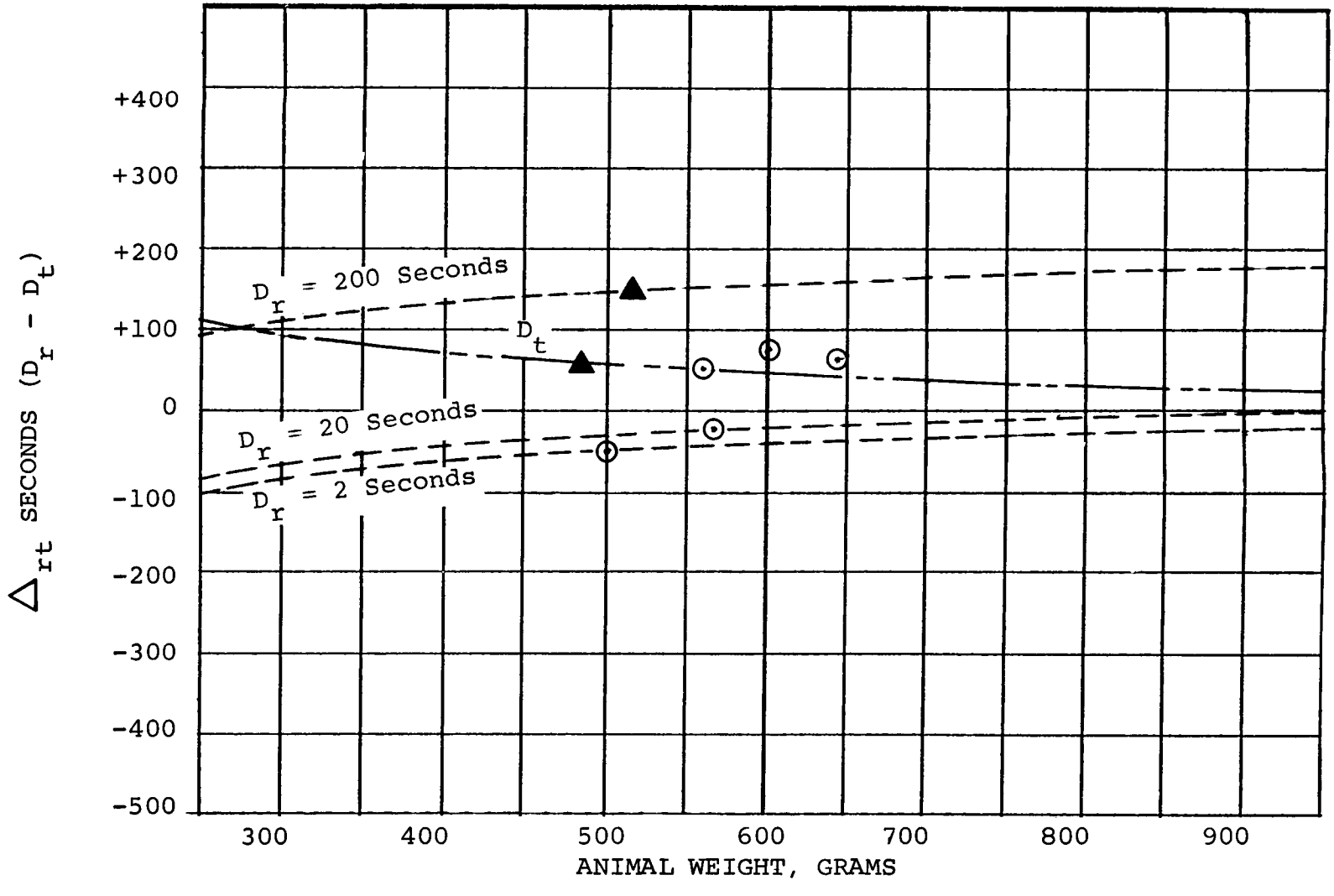


FIGURE 158 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-150 G_x CASE ONLY

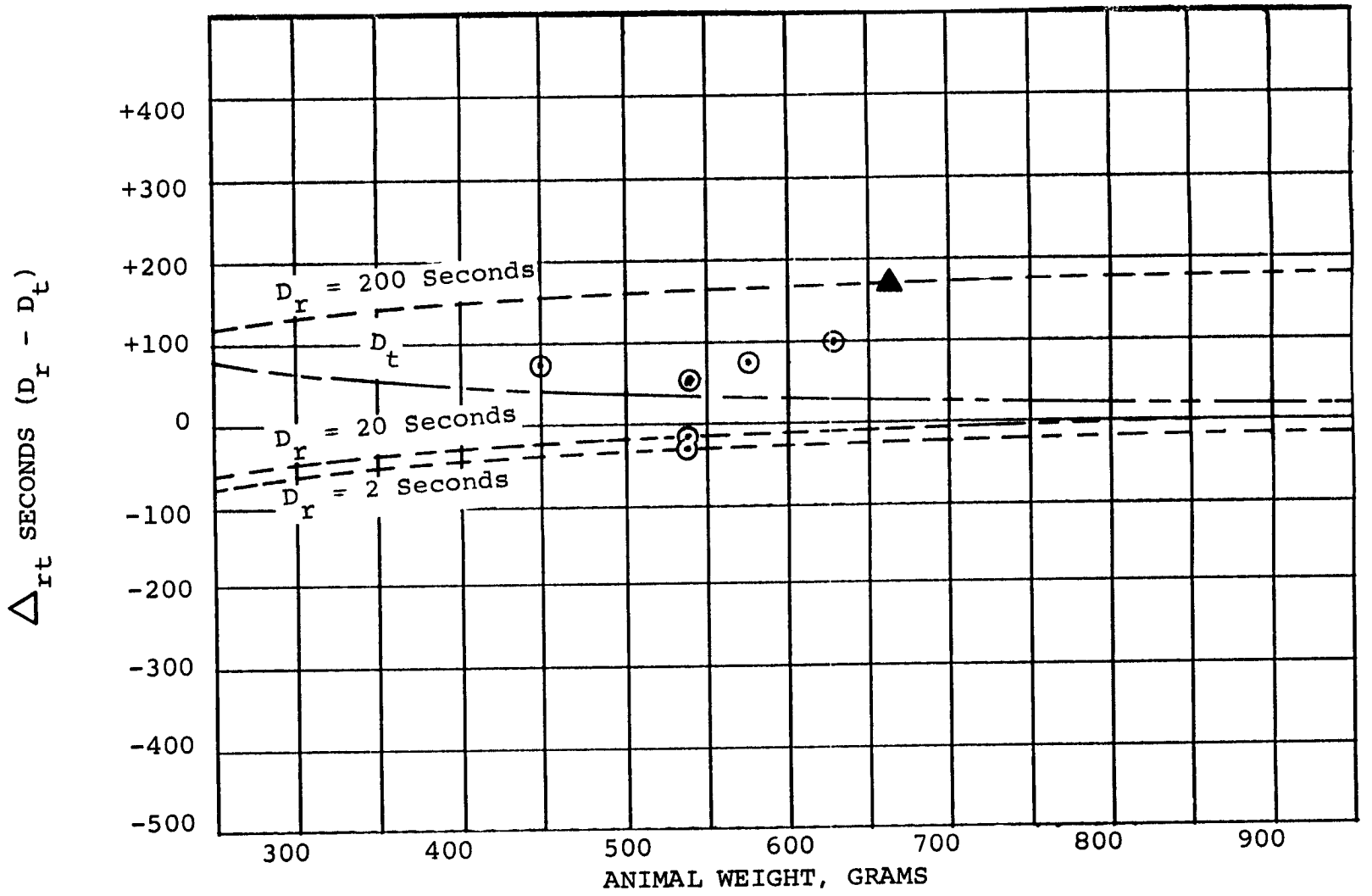


FIGURE 159 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-200 G_x CASE ONLY

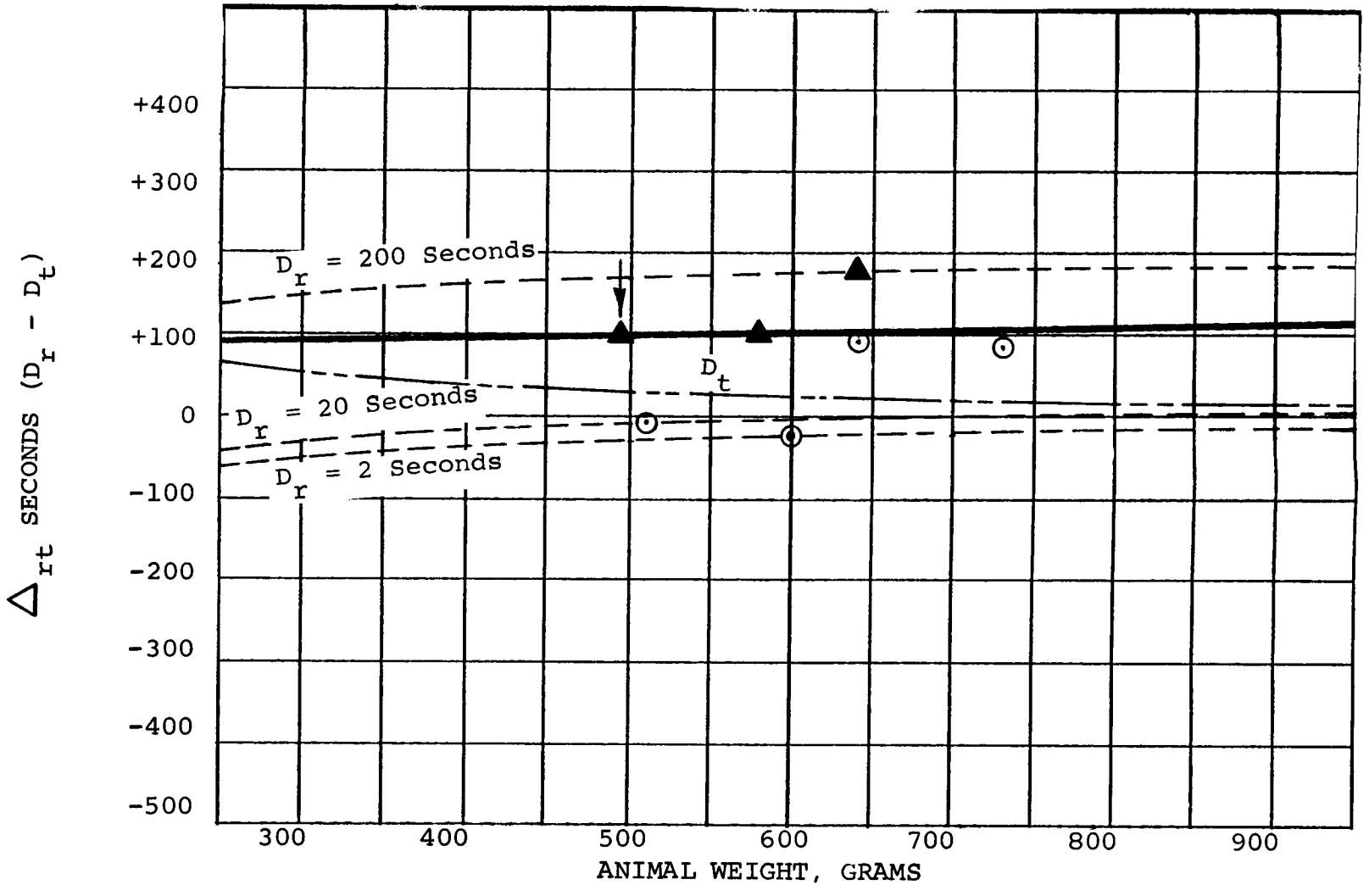


FIGURE 160 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-250 G_x CASE ONLY

G's. The maximum value for K_p achieved in this series was 1.6×10^4.

Figure 161.- Figure 161 is a Δ_{rt} versus weight graph for 300 $-G_x$. Nine animals were studied in this configuration. A great deal of difficulty was encountered with control of the animal in that the animals were able during the pre-spinup period to dislodge the faceplate retainer with subsequent extrusion of their neck and head through the faceplate. Consequently, the assessment of their individual capability to absorb G could not be made and their fatalities were listed as artifactural. However, four survivors occurred at values just below +100 Δ_{rt} on the plus side of the line and one fatality occurred at a K_p value of 1.87 (Δ_{rt} +100 seconds). The inference here is that the K_p values on previous graphs for this range of weight apparently also hold at 300 $-G_x$.

Figure 162.- Figure 162 is a Δ_{rt} graph for 350 $-G_x$. A generalized flattening of all curves is noted. A fine distribution of survivors below the value of Δ_{rt} equal to +80 seconds is noted with an interesting survival of an animal of mass 555 grams at a value of +100 Δ_{rt}. Again, this supports the hypothesis that an optimal weight for tolerance of G stress of the animal studied appears to be at the 500 to 550 grams level. The one loss which occurred (illustrated on this graph at a Δ_{rt} of zero, K_p of 3.9×10^3, weight of 555 grams) is considered artifactual. This is animal No. 69. It extruded its head and neck through the faceplate and died prematurely by strangulation. It is shown in the animal handling section and some slides of its tissues are in the histochemistry appendix.

Figure 163.- Figure 163 is a Δ_{rt} versus weight graph for 400 $-G_x$. Again, a very sharp division between survivors and fatalities occurs at the level of Δ_{rt} from +85 to +100 seconds. A few of the losses in this mid range are cliff-hangers. The slope of the line connecting the fatalities from left to right hand ends of the weight spectrum in this graph, as in previous graphs, also seems to illustrate a slope of approximately 10^o plus.

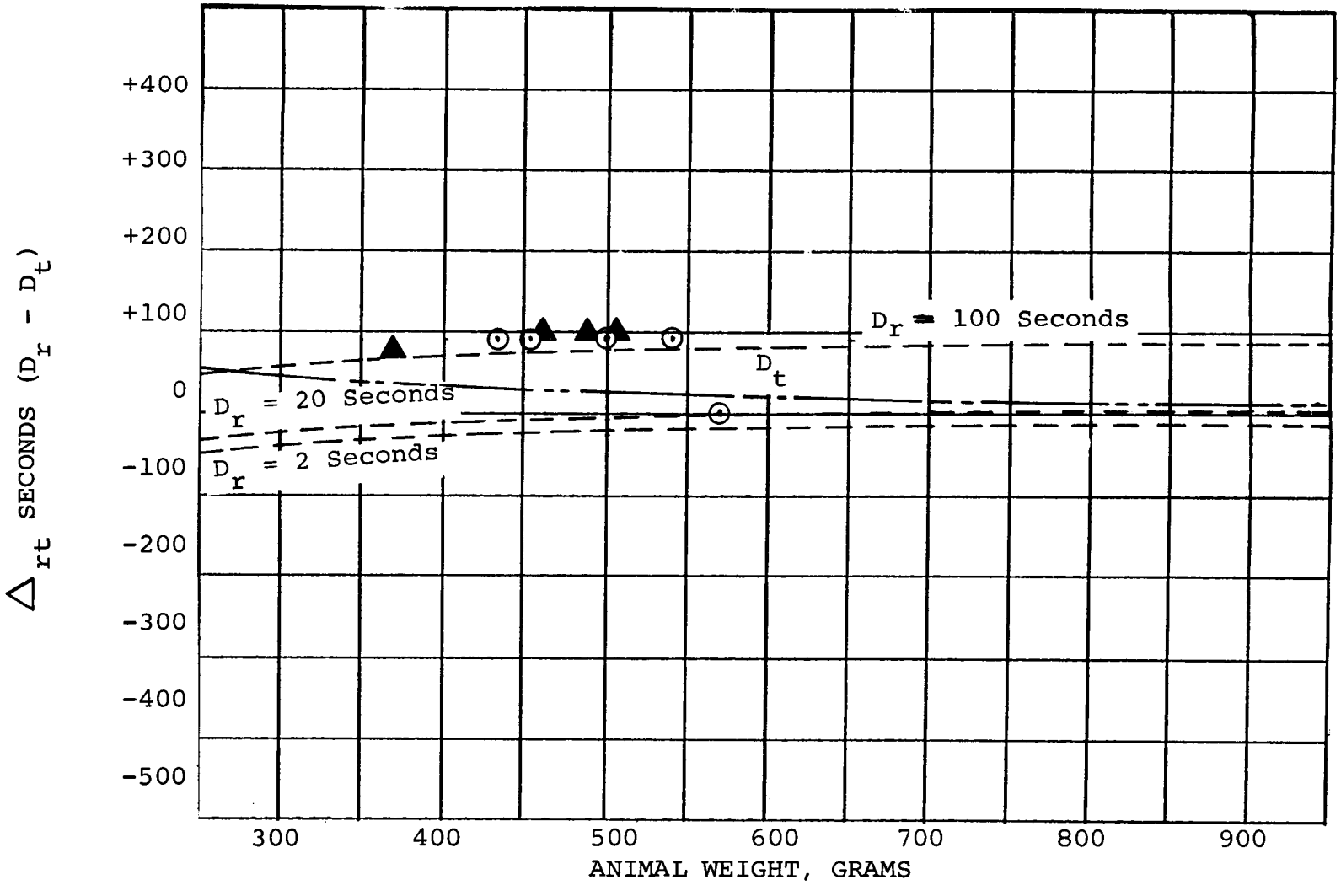


FIGURE 161 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-300 G_x CASE ONLY

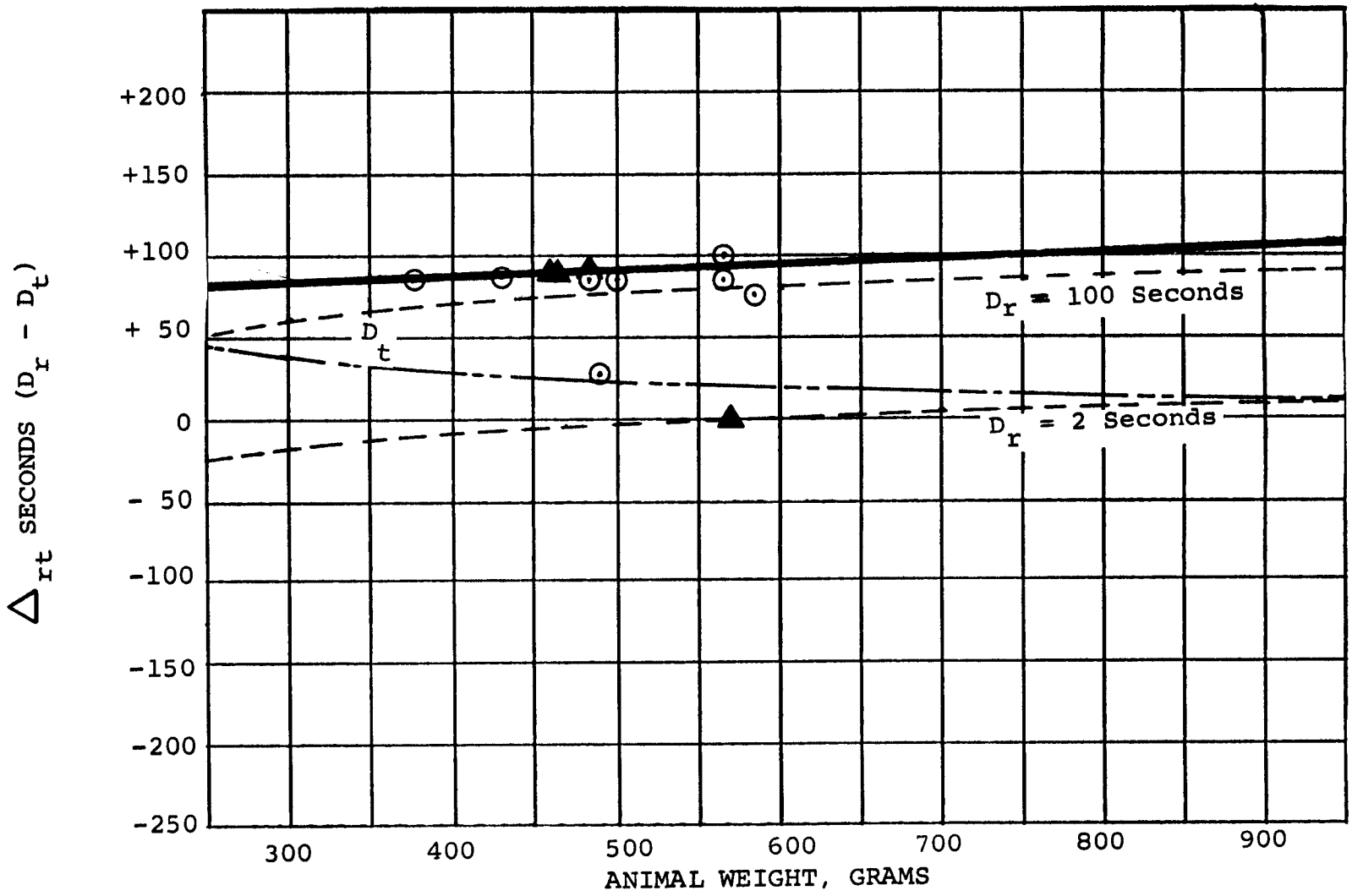


FIGURE 162 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-350 G_x CASE ONLY

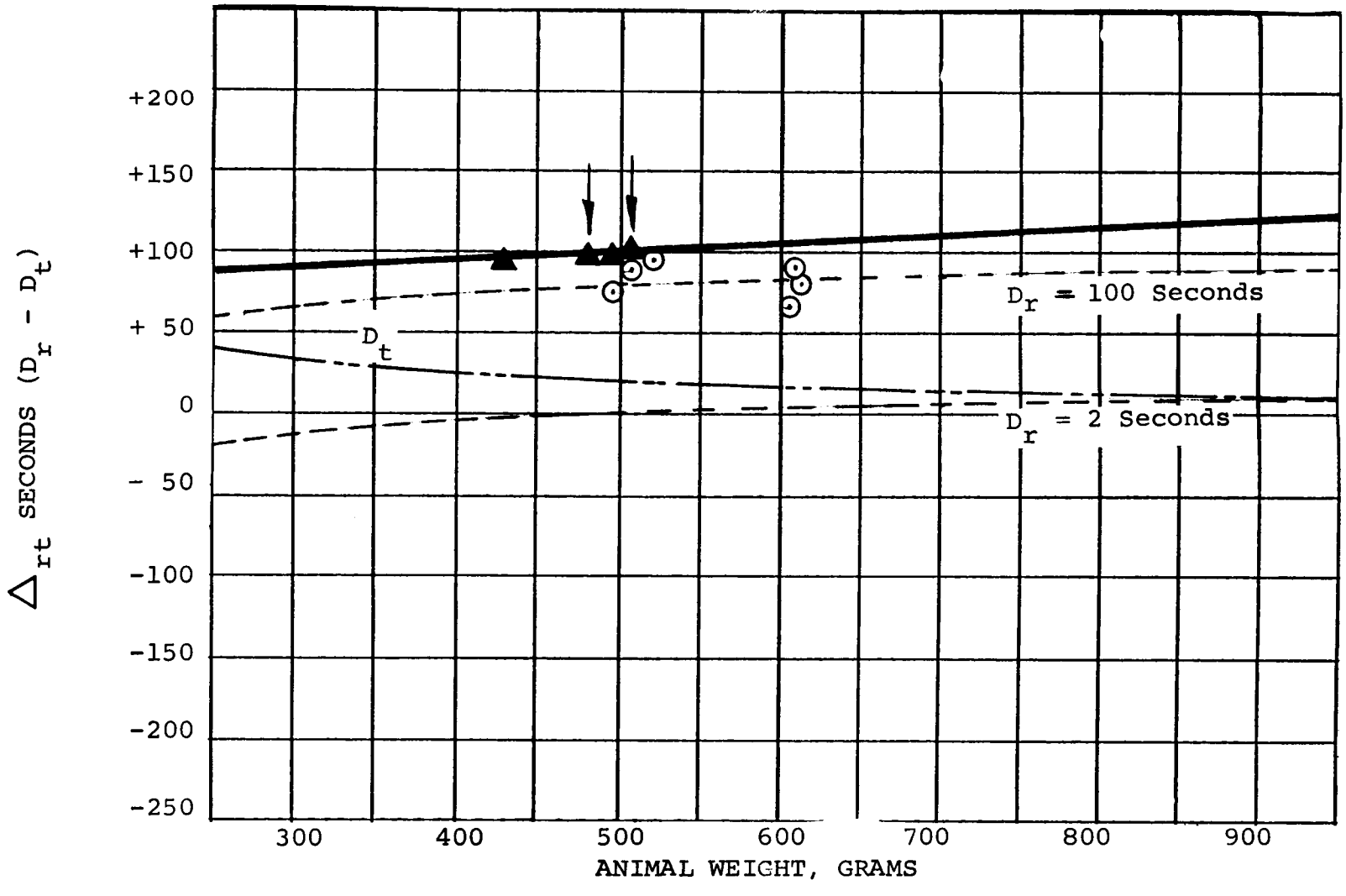


FIGURE 163 EXPOSURE ABOVE THRESHOLD VERSUS WEIGHT
-400 G_x CASE ONLY

Figures 164 through 172 show the K_p values plotted against weight, for each level of G stress tested. Symbols are used to indicate the direction of stress application and the experimental end results. Arrows point to the so-called "cliff-hanger" animals.

Lines were constructed on each graph by either using the lowest of the fatalities shown, or by approximating a L/D50 point. Where the results for the $-G_x$ axis seemed appreciably lower than the experience in other axes, an additional line is shown, parallel to the first, to illustrate the difference. Where an appreciable break in the line occurs, two intersecting straight lines were used.

The figures illustrate a reasonable degree of correlation to a "zone of fatality" which gradually rises with increasing weight and with increasing values of G until an upper limit of K_p is reached at an approximate value of 3.6. The results shown reflect the preponderance of animals between 450-550 grams weight.

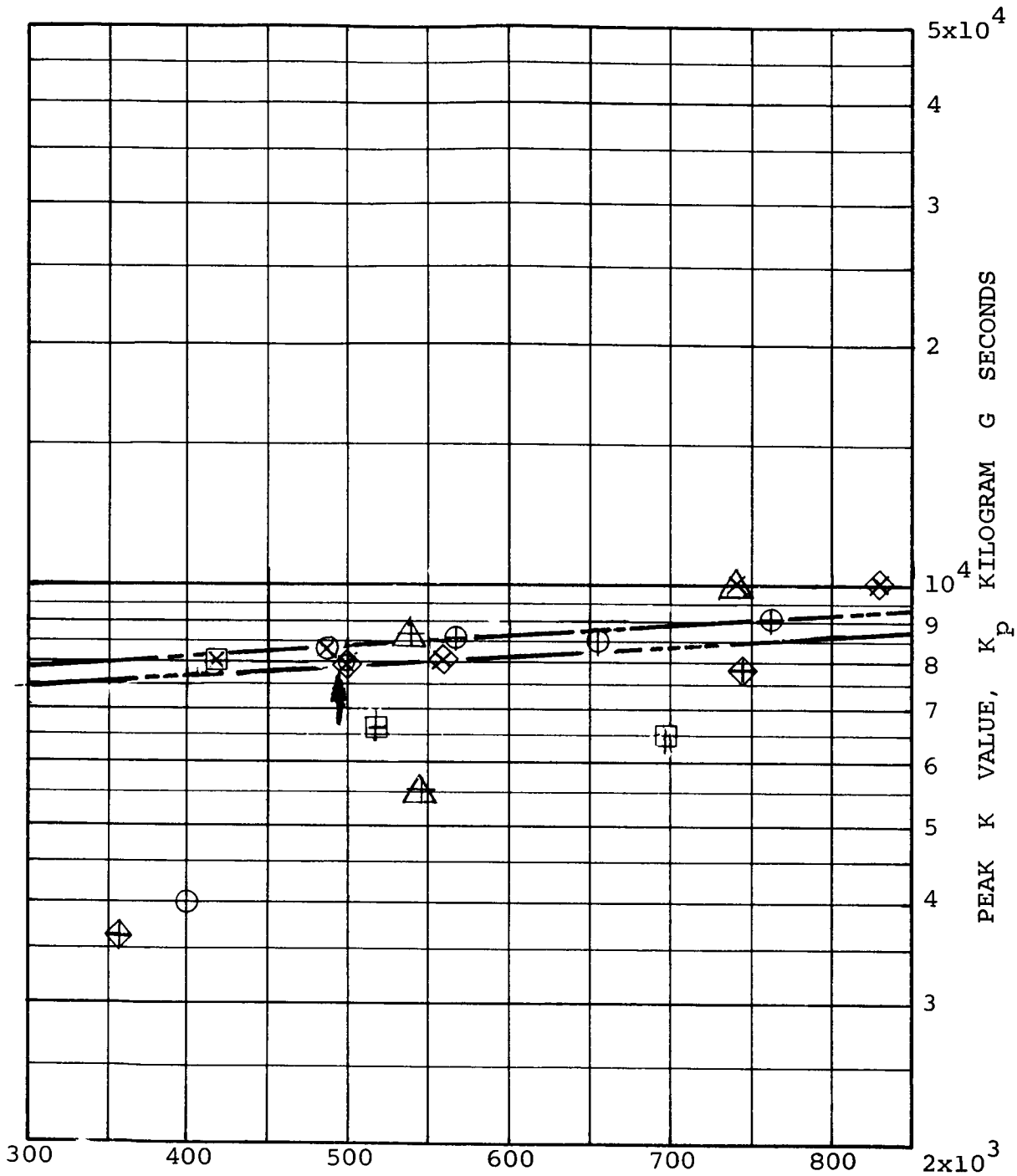


FIGURE 164. PEAK K VALUE VERSUS WEIGHT
50 G CASE

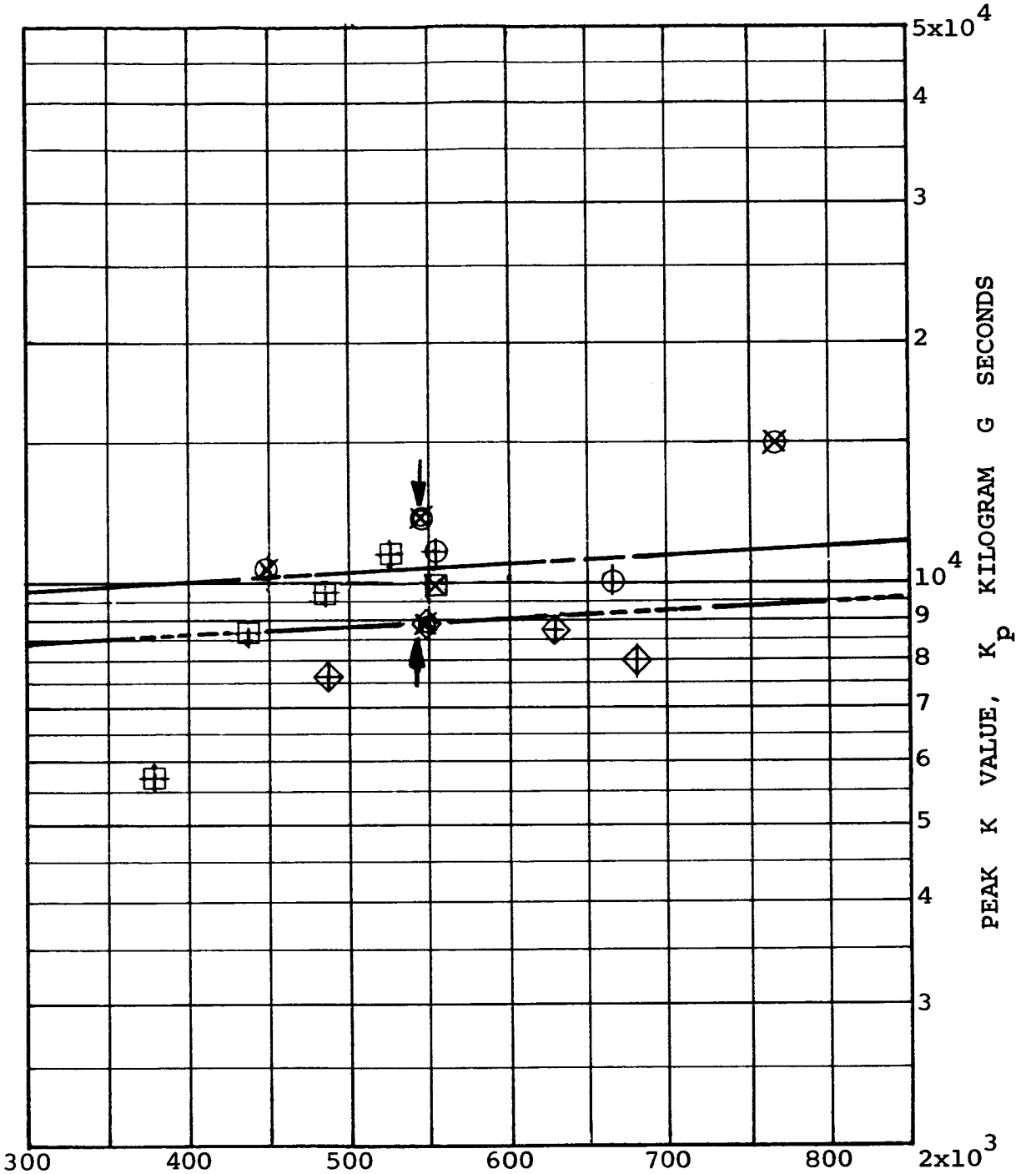


FIGURE 165. PEAK K VALUE VERSUS WEIGHT
100 G CASE

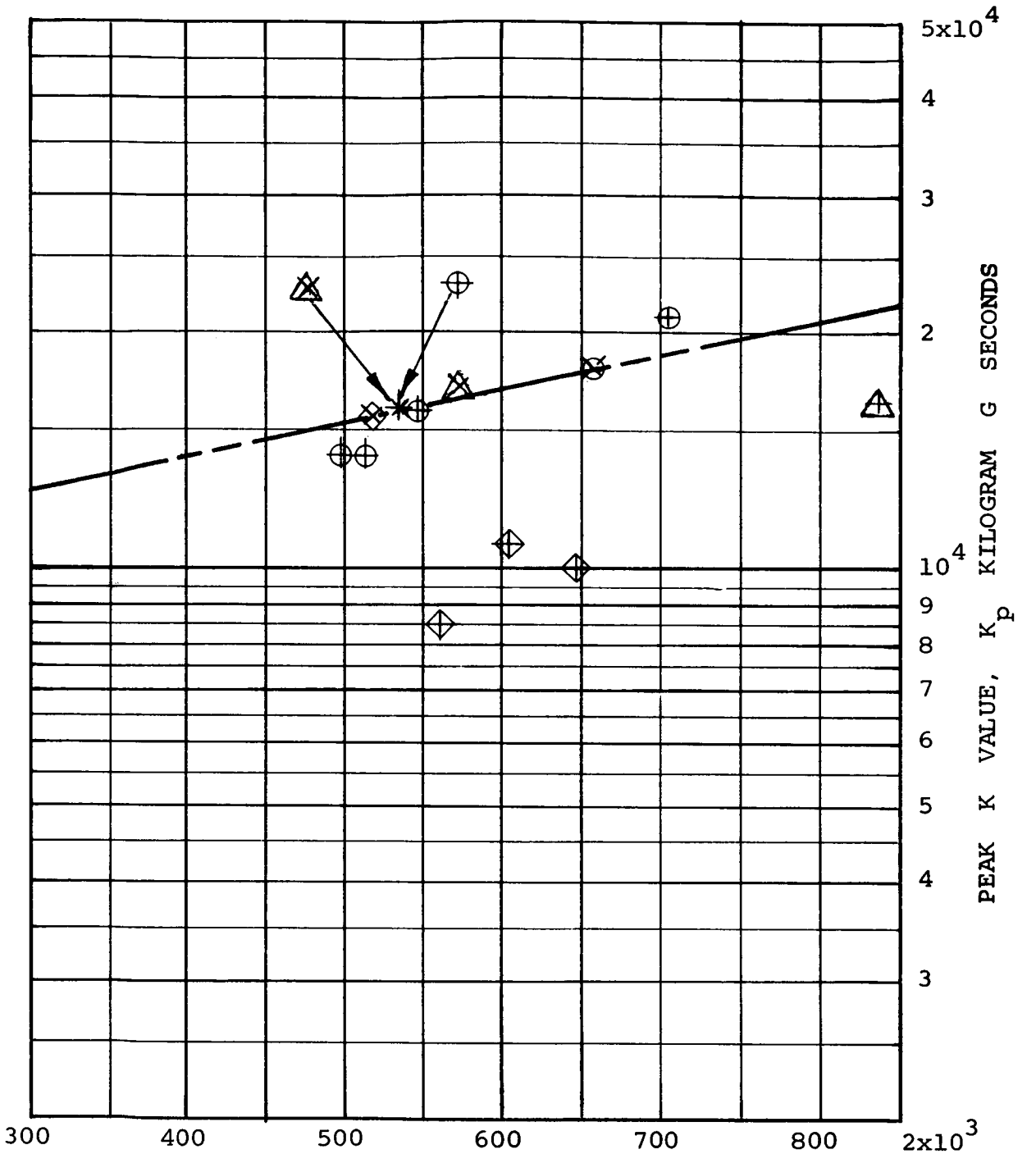


FIGURE 166. PEAK K VALUE VERSUS WEIGHT
150 G CASE

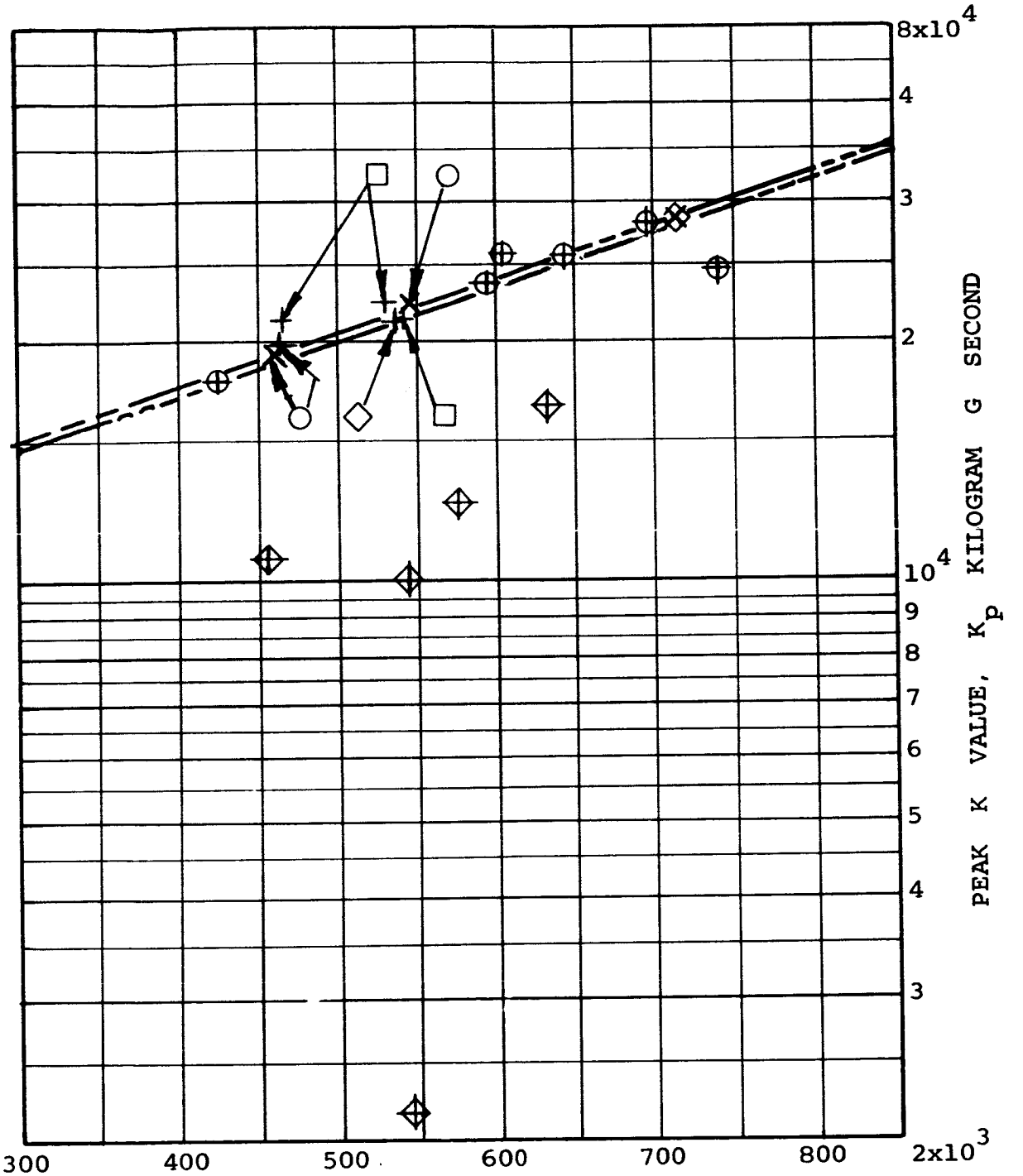
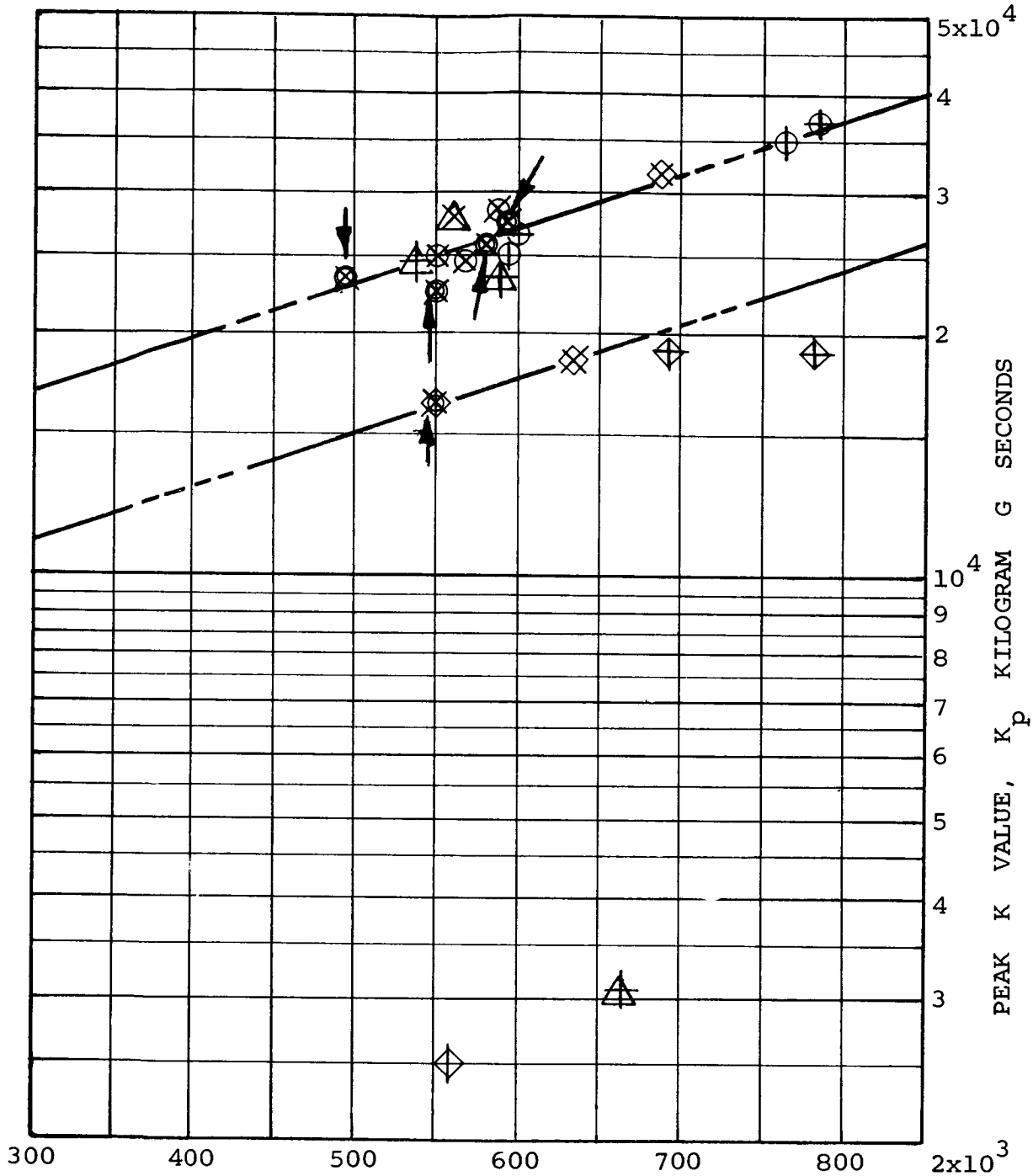


FIGURE 167. PEAK K VALUE VERSUS WEIGHT
200 G CASE



+ Survivor, x Fatality, ⊠ "cliff-hanger"

○= $+G_x$, ◇= $-G_x$, △= $+G_y$, □= $-G_y$

FIGURE 168. PEAK K VALUE VERSUS WEIGHT
250 G CASE

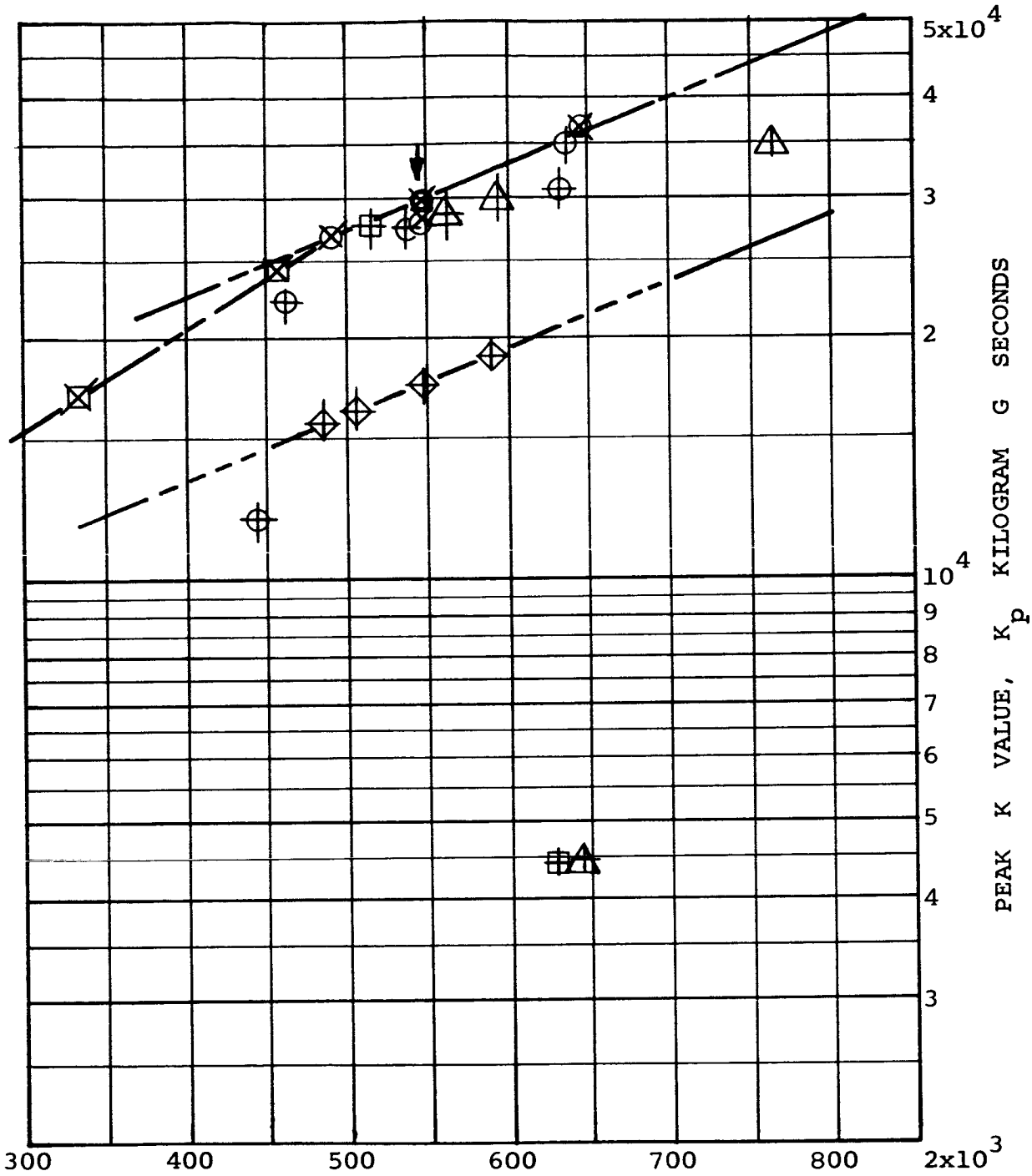
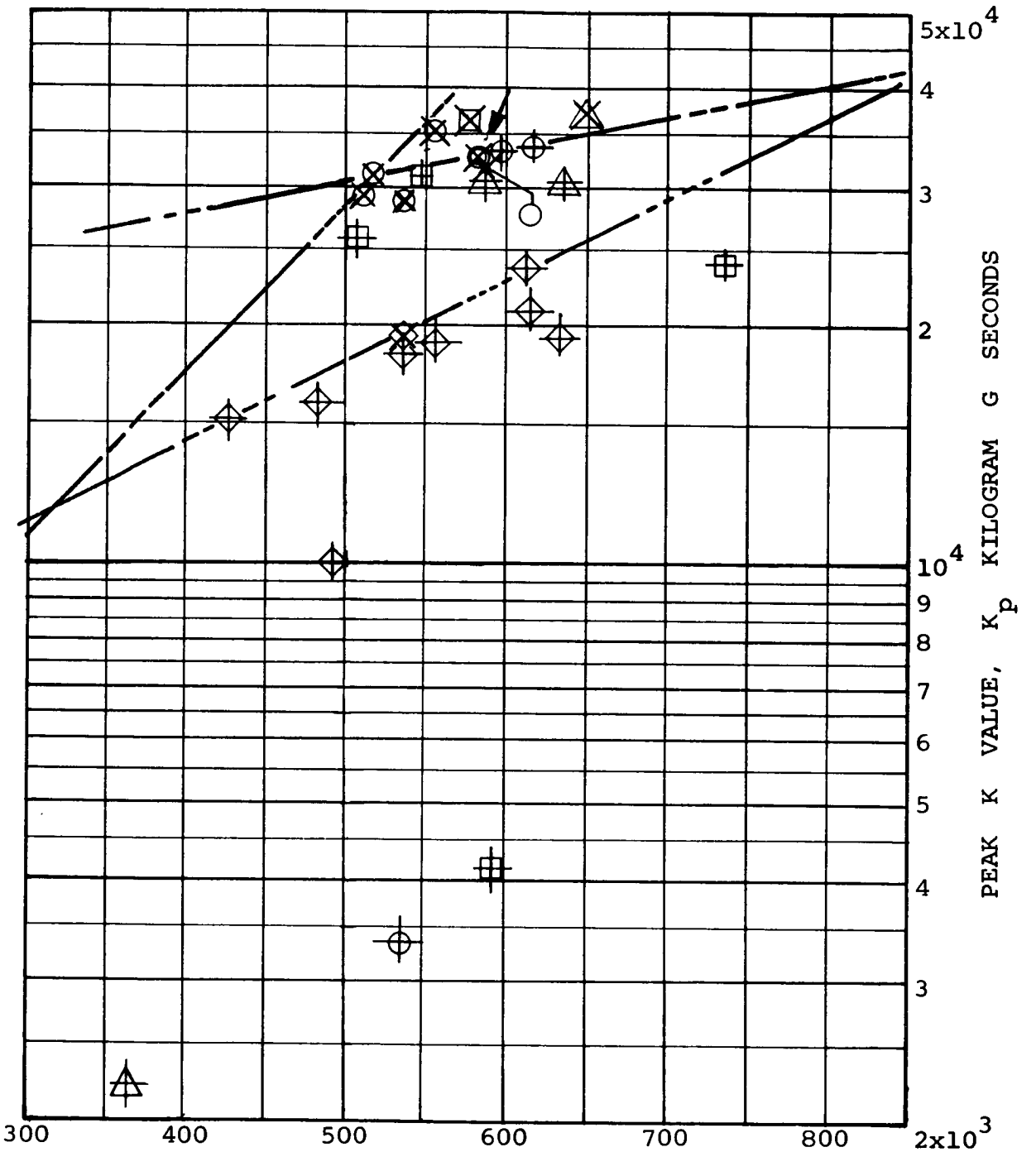


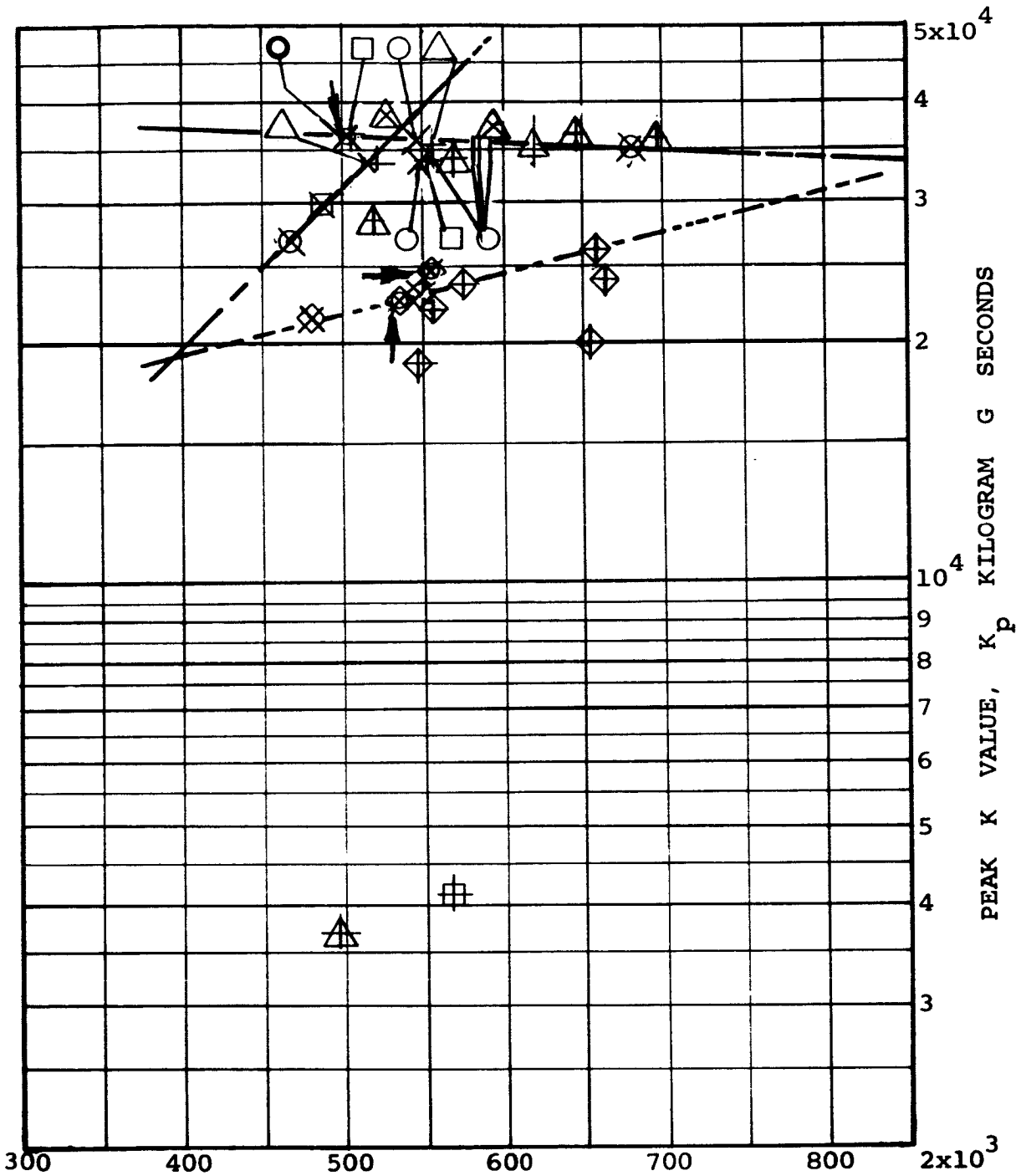
FIGURE 169. PEAK K VALUE VERSUS WEIGHT
300 G CASE



+ Survivor, x Fatality, ⊠ "cliff-hanger"

○ = $+G_x$, ◇ = $-G_x$, △ = $+G_y$, □ = $-G_y$

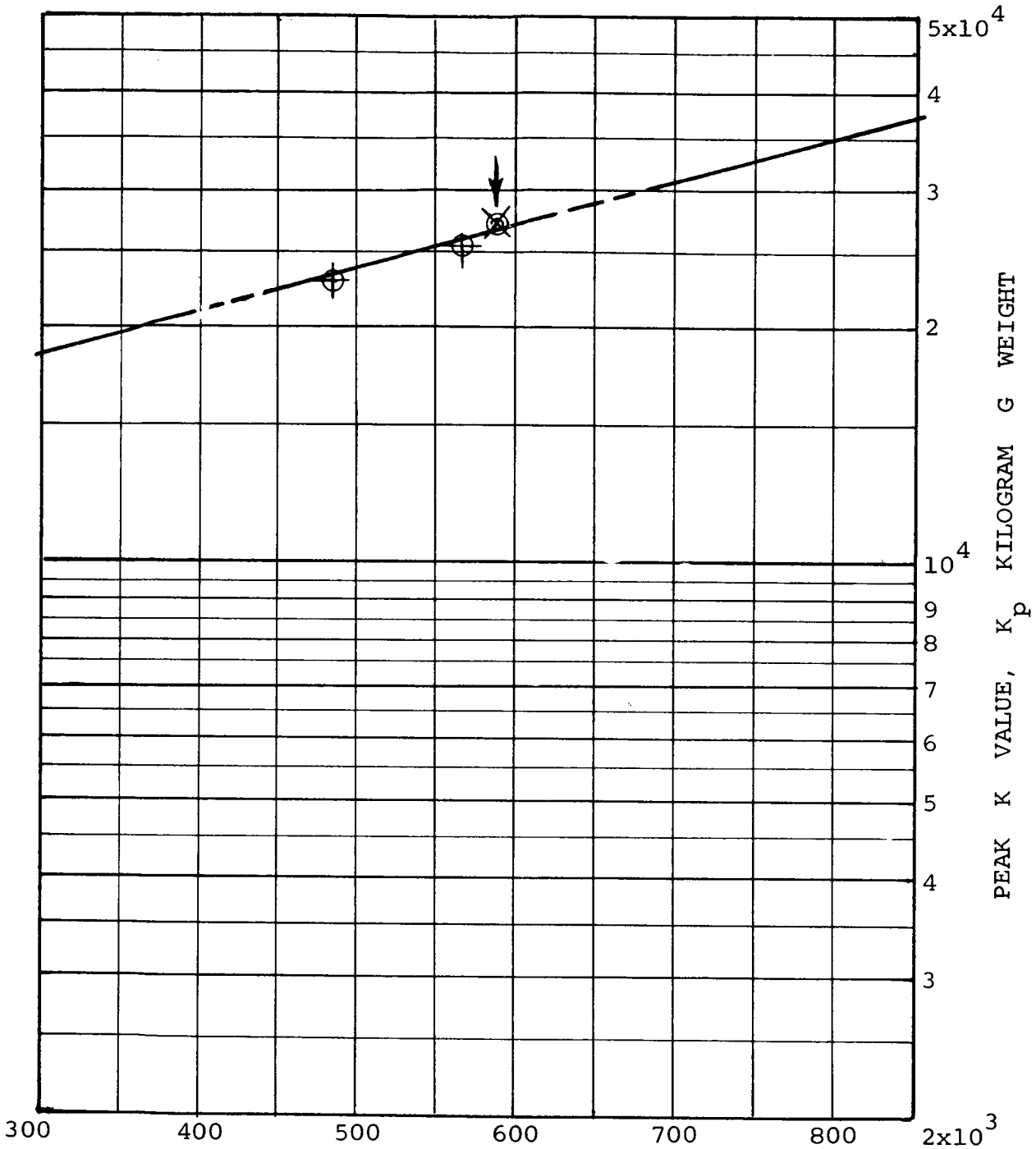
FIGURE 170. PEAK K VALUE VERSUS WEIGHT
350 G CASE



+ Survivor, x Fatality, ▣ "cliff-hanger"

○= $+G_x$, ◇= $-G_x$, △= $+G_y$, □= $-G_y$

FIGURE 171. PEAK K VALUE VERSUS WEIGHT
400 G CASE



+ Survivor, x Fatality, ⊠ "cliff-hanger"

◯ = $+G_x$ Mode

FIGURE 172. PEAK K VALUE VERSUS WEIGHT
430 G CASE