Report No. 6
RF Project 2045

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THE OHIO STATE UNIVERSITY

RESEARCH FOUNDATION

1314 KINNEAR ROAD COLUMBUS, OHIO 43212

FINAL REPORT - PART II

CARDIOVASCULAR EFFECTS OF VIBRATION

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NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
Washington, D. C. 20546

Grant No. NGR 36-008-041

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FINAL - PART II

REPORT

By

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On CARDIOVASCULAR EFFECTS OF VIBRATION

For the period 31 July 1968 - 31 July 1969

Submitted by Lester B. Roberts

Department of Preventive Medicine

Date July 1969

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I. HUMAN VIBRATION AND THE ECG

A. INTRODUCTION

Part I of the Final Report dealt with human response to Gz (vertical) vibrations. This report deals with Gx (front-back) and Gy (transverse) vibrations.

B. APPARATUS AND TESTS

Each of two volunteer subjects was sinusoidally vibrated; unrestrained, Gx and Gy, while seated, at various intensities as shown in Table I.

Frank lead orthogonal ECG's, obtained using special equipment and techniques described previously,[3] were recorded on magnetic tape during the complete test and control periods. Selected portions were reproduced as x, y, and z scalar ECG's. Beat-by-beat pulse rate curves and certain vectocardiographs were also obtained from the stored recorded data. These curves for various vibration intensities are shown in Figs. 1-71. Also shown on the pulse rate curves are blood pressure readings (cuff) obtained approximately 30 seconds after termination of each vibration.

C. RESULTS AND DISCUSSION

1. X, Y, Z, Scalar ECGs

Inspection of the orthogonal scalar electrocardiograms considered together shows them to be generally acceptable for clinical evaluation. Under certain conditions individual leads show excessive noise. As previously noted[9] this substantiates our recommendation to record multiple leads in this kind of study.

It was not considered necessary to apply signal averaging techniques to these data.[3]

No evidence of extrasystoles was detected in the recordings of either subject during this experiment. In a previous experiment[9] Subject K's record shows an atrial or nodal extrasystole occurring during standing Gz vibration.

Changes in the subjects' ECGs which occurred during vibration did not persist after vibration nor did the changes suggest undue stress or any other than normal physiological variations.

2. Pulse Rate Curves

In contrast to our findings for Gz[9] vibration there seems to be no consistent pattern of pulse rate change for either subject when vibrated either Gx or Gy.

3. VCGs

There are interesting pattern changes in the VCGs. These may reflect electrical or physical heart center movement, but presently are considered unexplained. The magnitude of VCG change during vibration can be seen to be considerably less than the variations seen during deep breathing.

Table I. Vibration intensities used in experiment, expressed as frequency and peak amplitude and approximately as peak G

Amplitude (inches)	Frequency (Hz)	Acceleration (peak G)	Duration (sec)
1/8	2	.05	60
	3	.11	60
	5	.31	60
	7	.61	60
	9	1.01	60
	12	1.80	60
	0-12-0	to 1.80	60
	0-12-0	to 1.80	30
1/4	2	.10	60
	3	.22	
	5	.62	
	7	1.22	
	8	1.60	
	11	3.00	
	0-11-0	to 3.00	60
	0-11-0	to 3.00	30
1/2	2	.20	60
	3	.45	
	5	1.24	
	7	2.45	
	0-8-0	to 3.20	60
	0-8-0	to 3.20	30

D. CONCLUSIONS

(1) Clinically useful ECGs can be obtained from human subjects while they are being vibrated, even at relatively severe vibration intensities, if our recommendations to accomplish this are followed. The ability to obtain such ECGs makes the ECG available as a useful tool in cardiovascular vibration research.

(2) Subject to confirmation by evaluation of a larger number of experimental subjects our research shows: (a) That short-term vibrations in the practical range used were physically only moderately stressing to healthy subjects. (b) That vibration, particularly Gz vibration <u>changing</u> in frequency, can elicit premature extrasystoles in man. (c) That changes in configuration of ECGs observed during vibration were transient, terminating with termination of vibration, and of a magnitude comparable to normal changes of deep breathing. While the changes are not considered clinically significant, nevertheless they are of physiological interest. (d) That patterns of pulse rate changes during vibration are variable. During Gz vibration consistent patterns exist for individuals, but not between individuals. The magnitude of change is related to vibration intensity and pulse rate before vibration. This variability in pulse rate response to vibration may be important. Wide variability in subjective responses to vibration has been observed by us and others (10). It has been rationalized on the basis that physiological and psychological responses are mediated through initial physical responses which, being functions of mass, elasticity and dampening, vary widely between subjects because of variation in man's physical make-up. The interrelationship between the responses is complicated and made more variable by feed back of physiological and psychological response back to physical. On this basis, it is perhaps not surprising that pulse rate responses should be variable. It indicates, however, that man's pulse rate response to vibration is largely subjective; at least more subjective than we had previously considered it. This may have important implications for longer term vibration exposures; it implies "individual susceptability" to vibration which suggests a choice in assigning personnel to perform critical tasks in vibration environments. If confirmed it makes vibration tolerance specifications and even empirical vibration tolerance assessment less precise than we had anticipated earlier.

(3) A more comprehensive evaluation of the cardiovascular effects of vibration on man will require additional research which should include blood pressure and cardiac output measurements. Noninvasive methods to measure these parameters during vibration should be developed.

E. RECOMMENDED EQUIPMENT AND TECHNIQUES FOR RECORDING ECGs OF HUMAN SUBJECTS WHILE THEY ARE BEING VIBRATED

(1) Only high-quality ECG electronic equipment shall be used. Precautions will be taken to minimize electric interference pickup.

(2) For research, Frank or McFee orthogonal lead ECGs shall be recorded. For monitoring only, electrodes shall be placed on fleshy parts of the body.

(3) NASA type electrodes and electrode paste (described by Day and Lippitt[7]) shall be used. Electrodes will be checked for noise as described.[7]

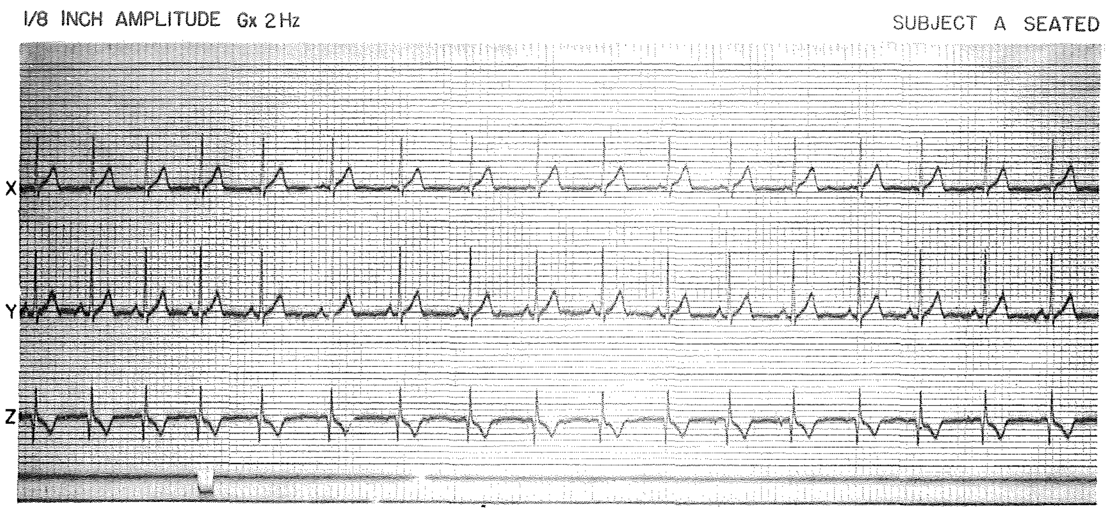
(4) The skin at electrode placement sites shall be prepared by 10-15 strippings (alternate application and removal) of Scotch* transparent tape. Electrical resistance of pairs of applied electrodes can be tested.[2]

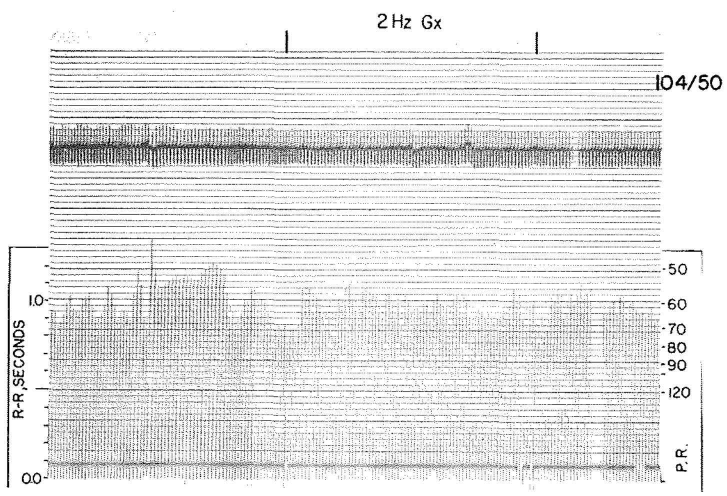
(5) Leads to electrodes will be flexible, light weight, and shielded, and shall have low microphonic noise characteristics. The leads shall be taped to the subject's body with slack to prevent undue tension or pulling during vibration.

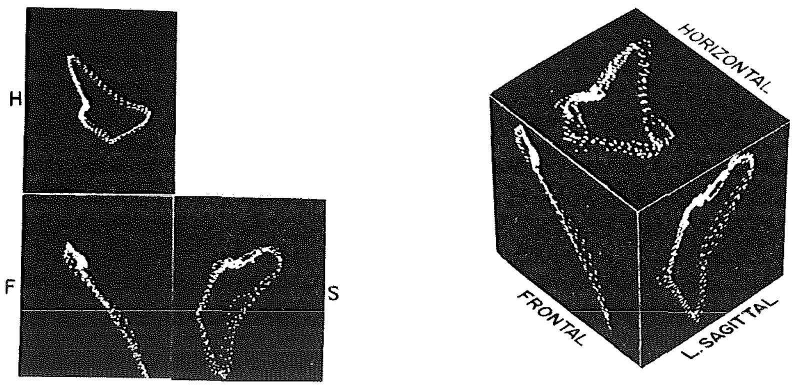
(6) If necessary, noise reduction of ECG signals can be accomplished using signal-averaging techniques described in Ref. 1.

(7) If Frank orthogonal leads are used, equipment described in Report No. 3 (page 3) can be used to determine the line of the center of the heart.

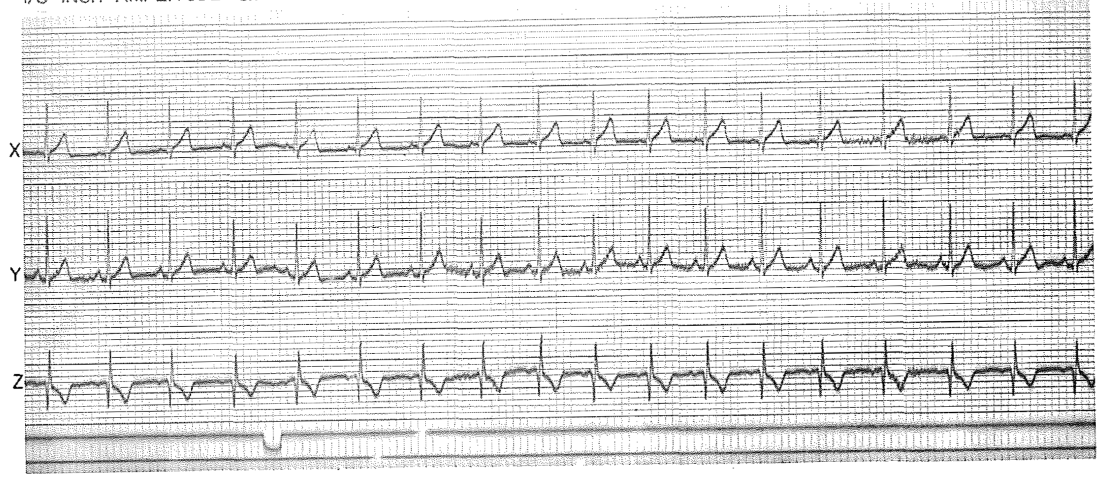
*Registered trademark

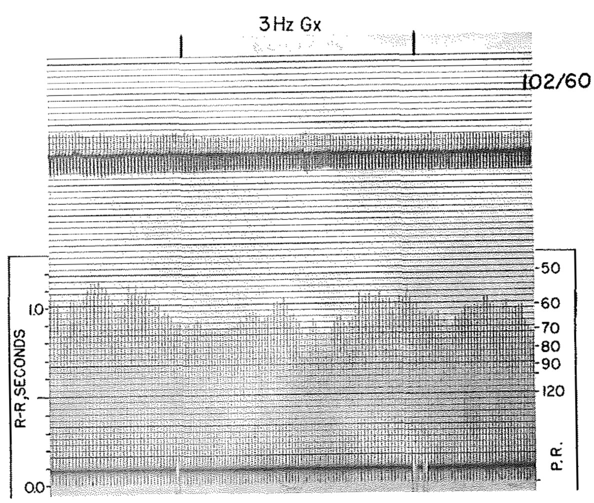

1/8 INCH AMPLITUDE Gx 2 Hz
SUBJECT A SEATED
X
Y
Z

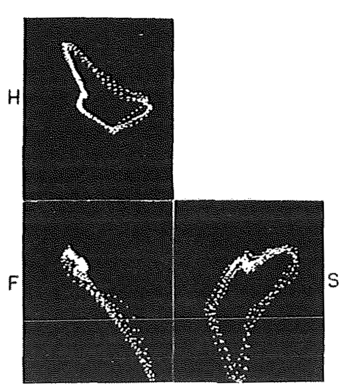

2 Hz Gx
104/50
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

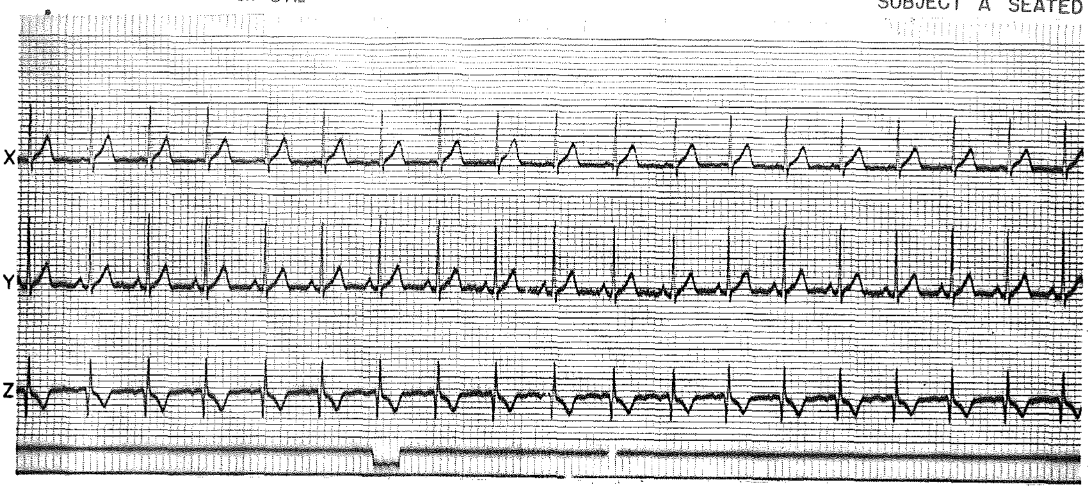

H
F
S
HORIZONTAL
FRONTAL
L. SAGITTAL

1/8 INCH AMPLITUDE Gx 3Hz SUBJECT A SEATED








1/8 INCH AMPLITUDE Gx 5 Hz
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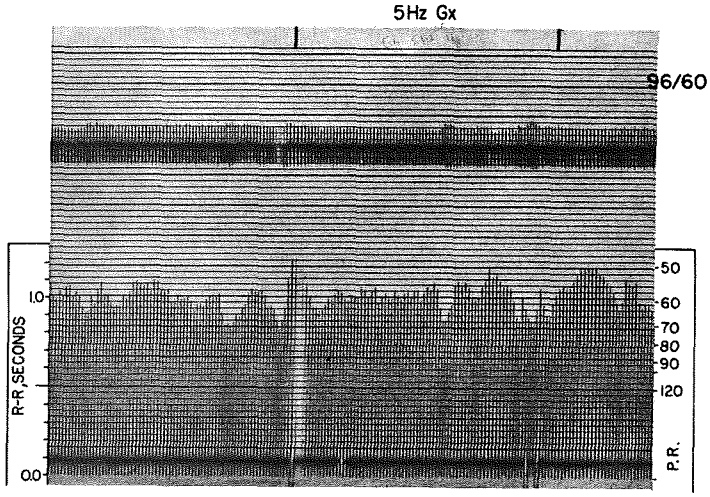
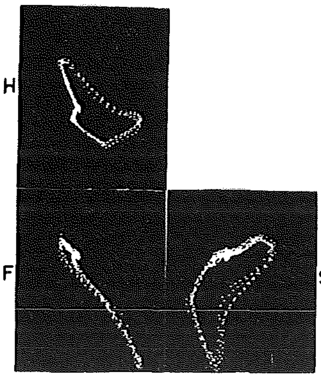
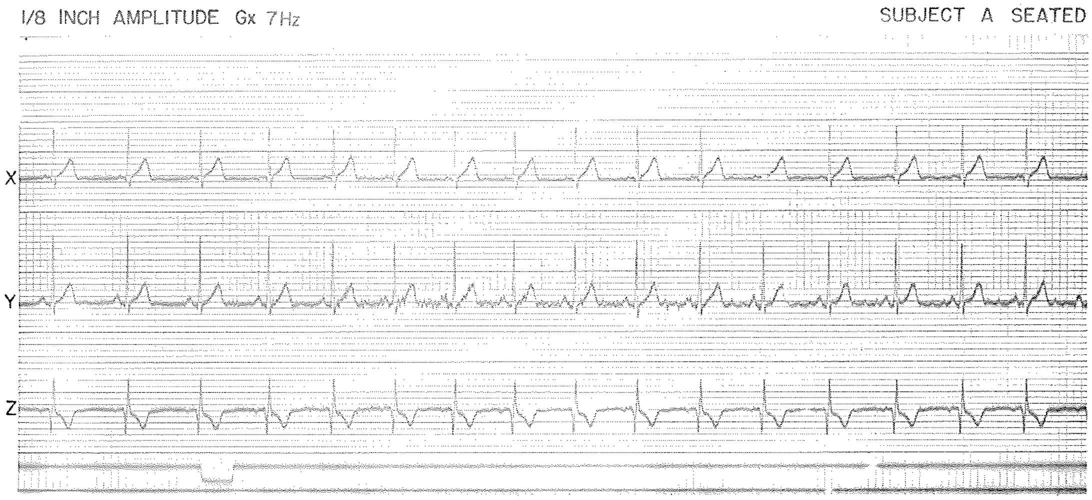
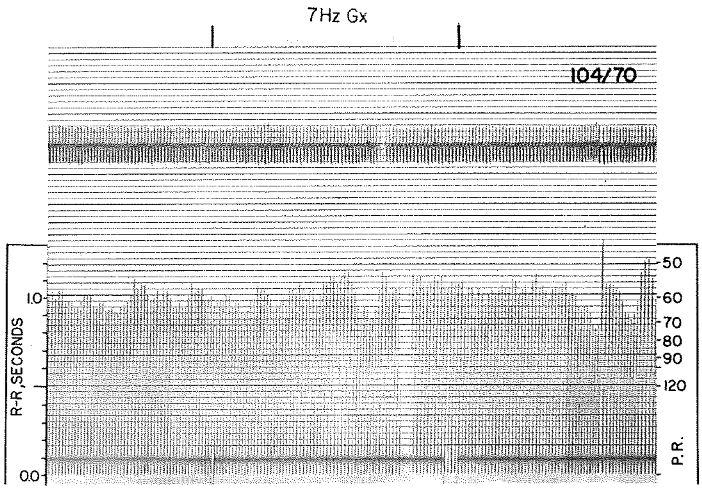
Y
Z


5Hz Gx
96/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

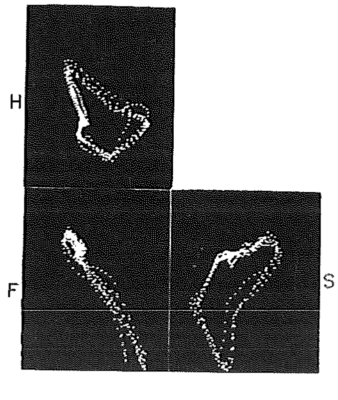

H
F
S



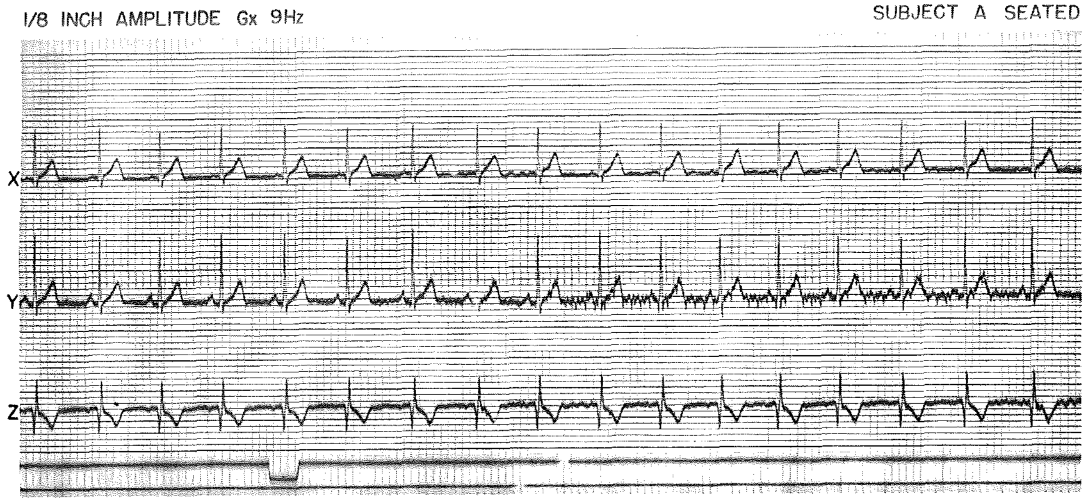
1/8 INCH AMPLITUDE Gx 7Hz
SUBJECT A SEATED
X
Y
Z



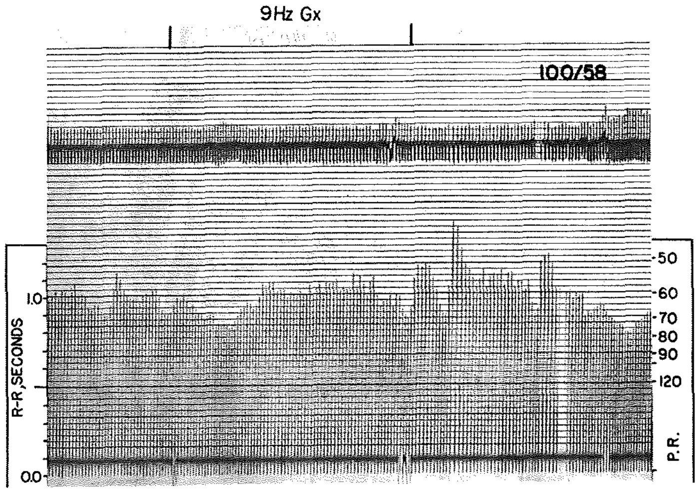
7Hz Gx
104/70
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.



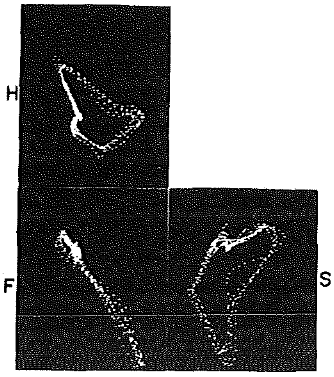
H
F
S



1/8 INCH AMPLITUDE Gx 9Hz
SUBJECT A SEATED
X
Y
Z

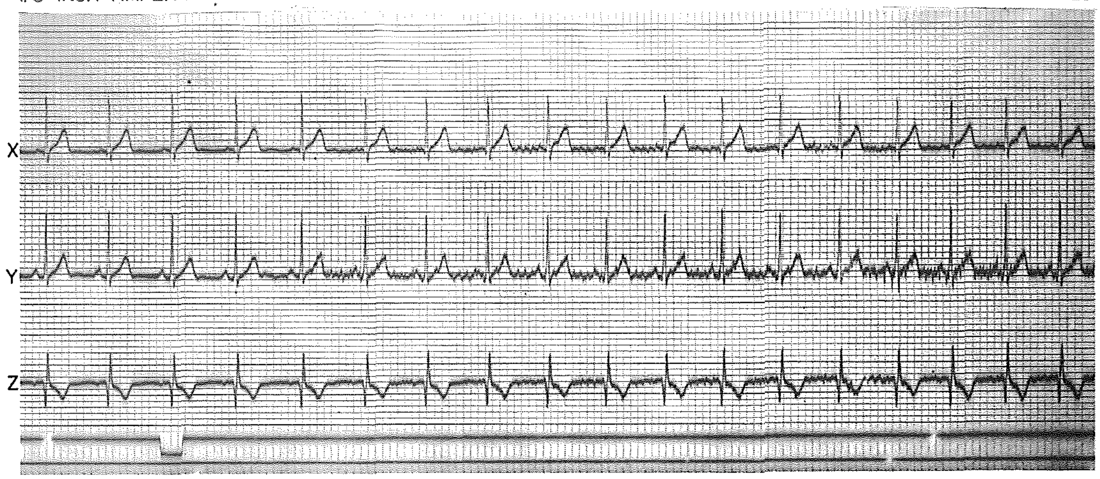


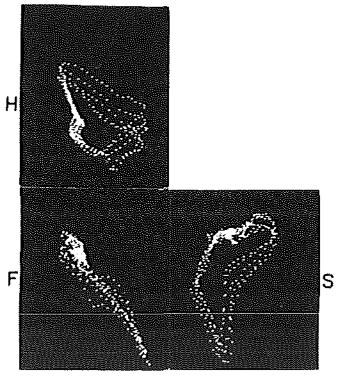
9Hz Gx
100/58
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

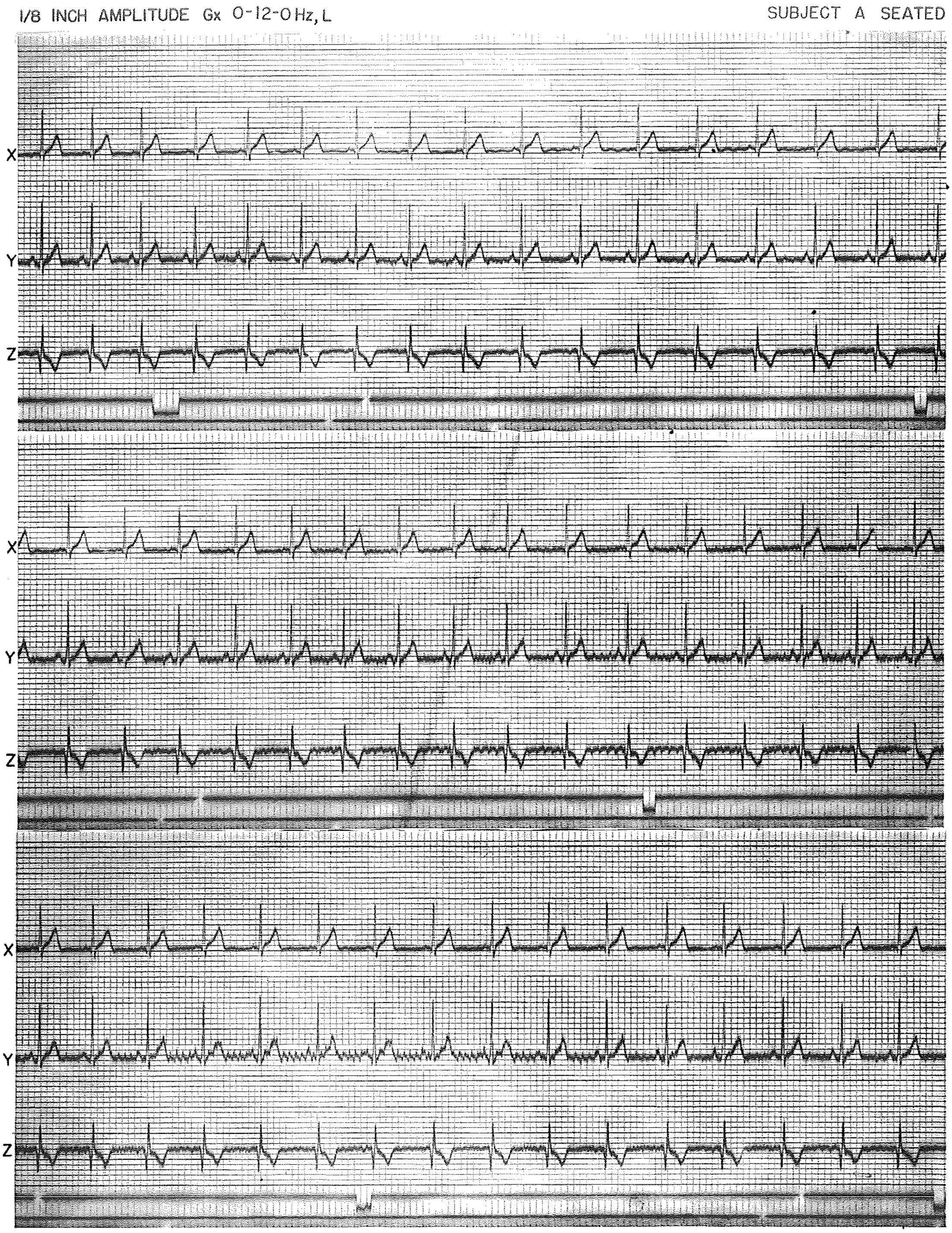


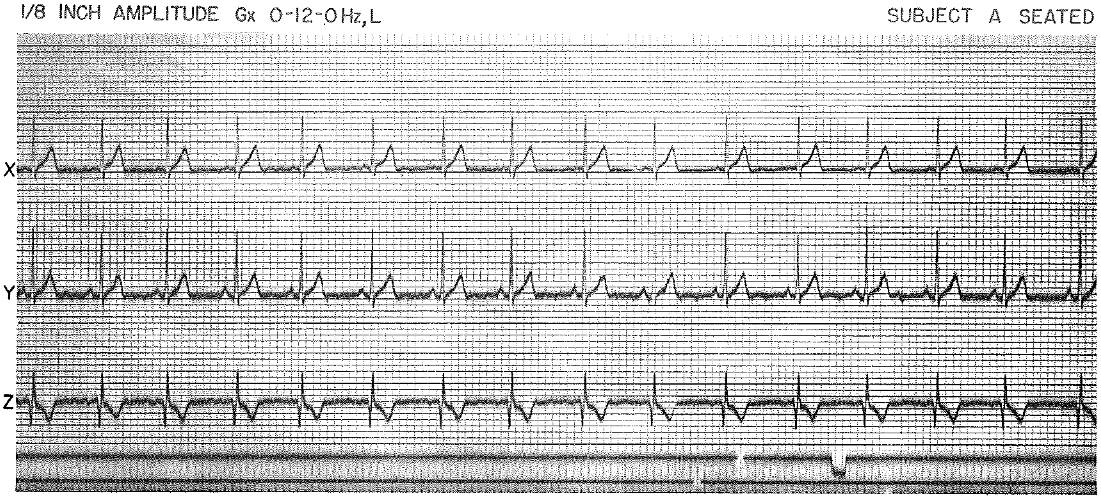
H
F
S

1/8 INCH AMPLITUDE Gx 12 Hz SUBJECT A SEATED

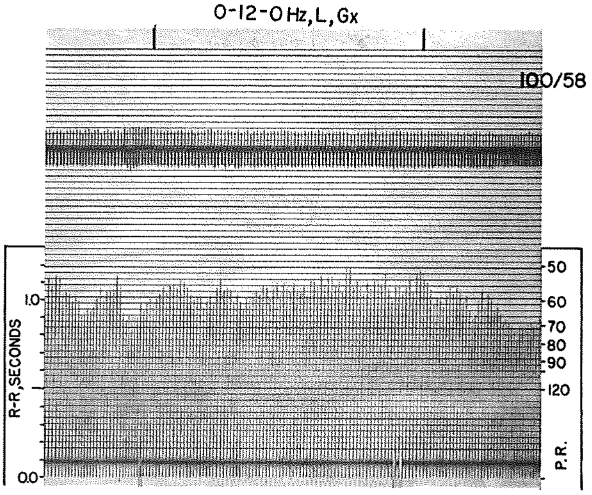





1/8 INCH AMPLITUDE Gx 0-12-0 Hz, L
SUBJECT A SEATED
X
Y
Z
X
Y
Z
X
Y
Z



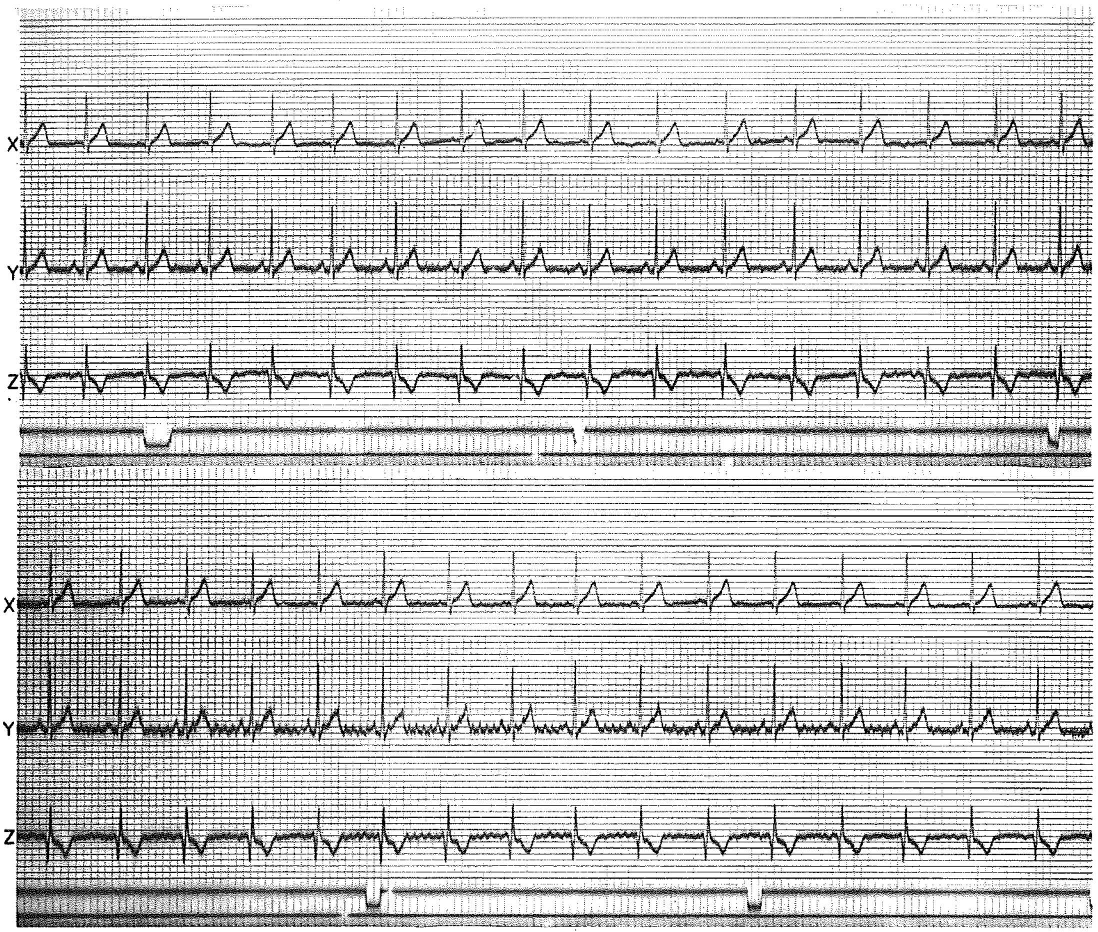
1/8 INCH AMPLITUDE Gx 0-12-0 Hz, L
SUBJECT A SEATED
X
Y
Z

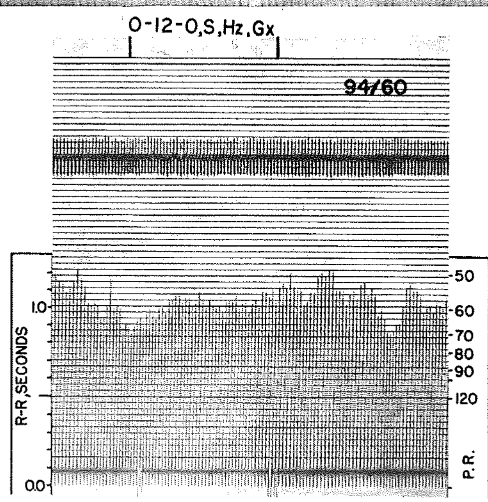


0-12-0 Hz, L, Gx
100/58
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

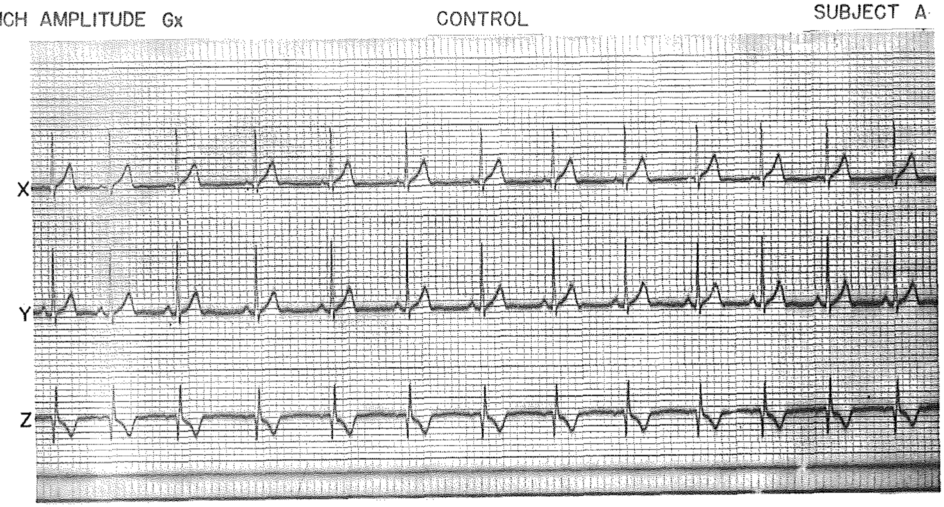
1/8 INCH AMPLITUDE Gx 0-12-0,S

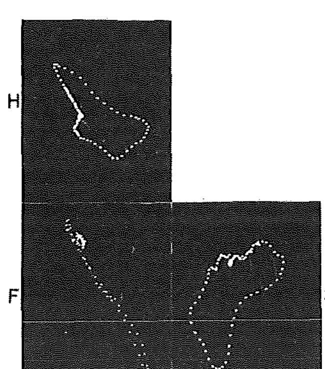
SUBJECT A SEATED





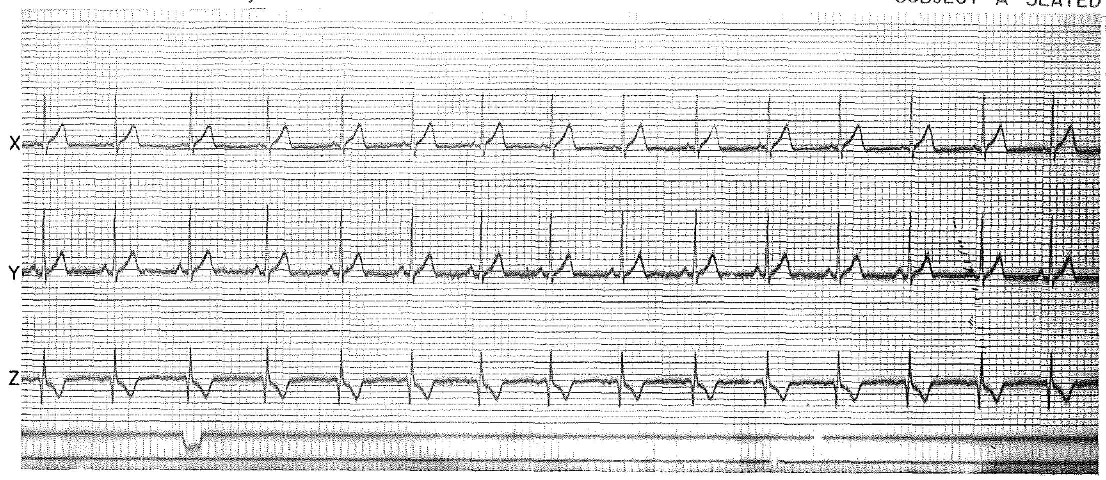
1/8 INCH AMPLITUDE Gx CONTROL SUBJECT A SEATED



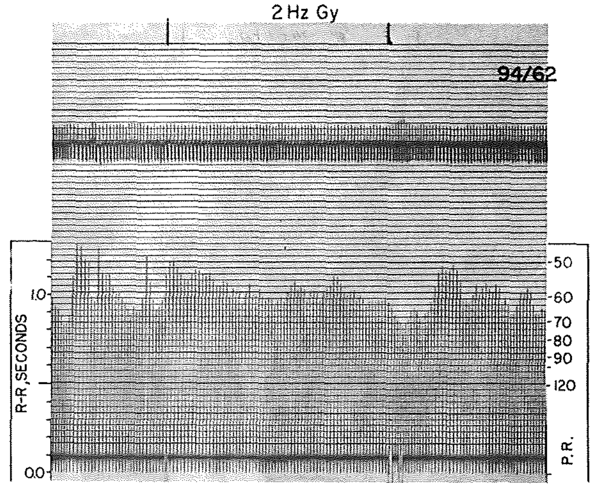


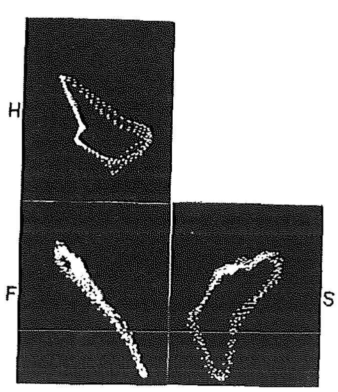
1/8 INCH AMPLITUDE Gy 2 Hz

SUBJECT A SEATED

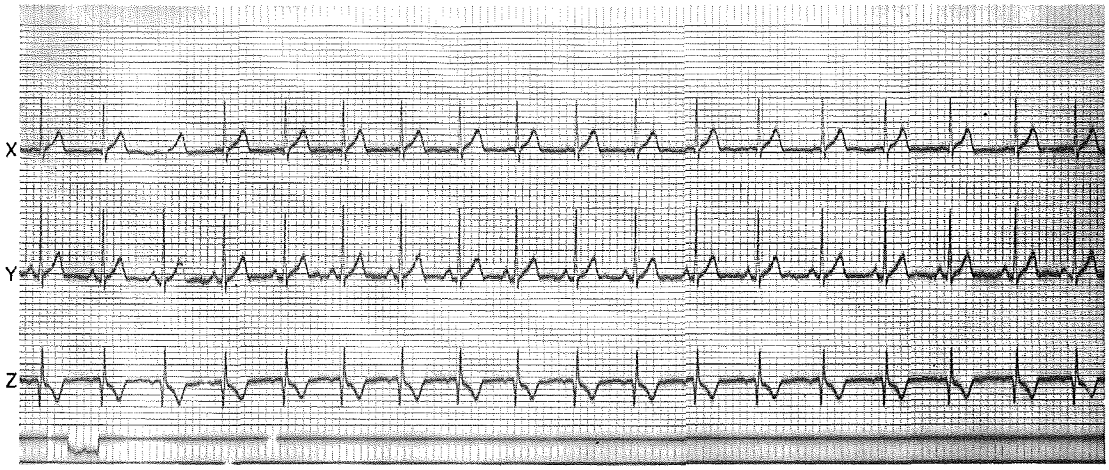


2 Hz Gy

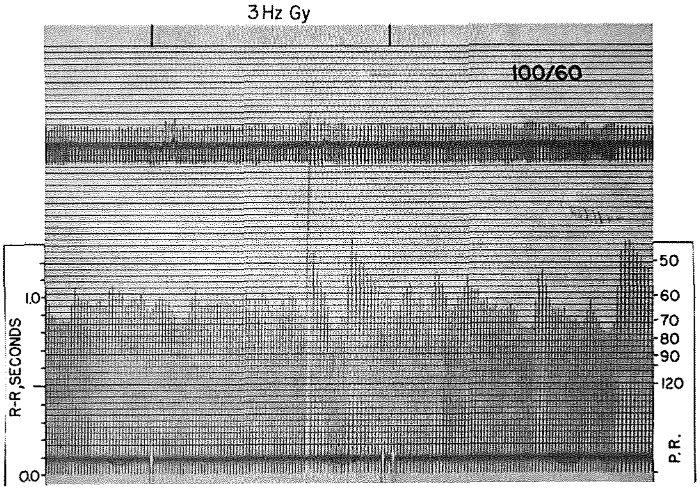


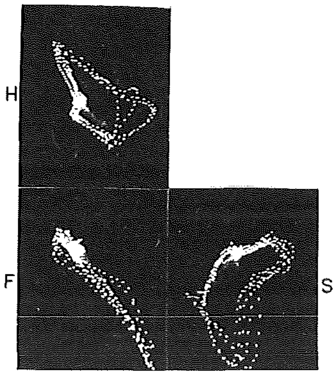


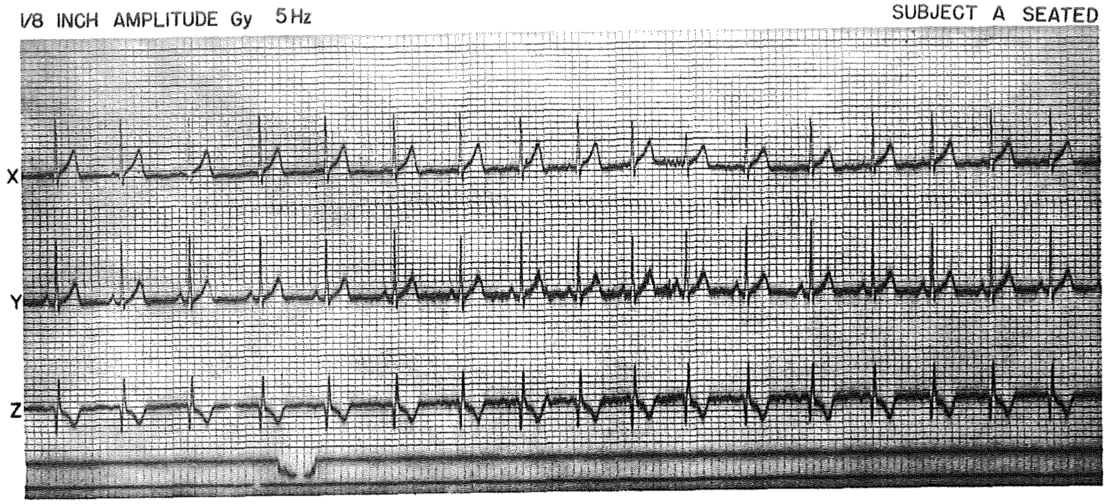
1/8 INCH AMPLITUDE Gy 3 Hz SUBJECT A SEATED

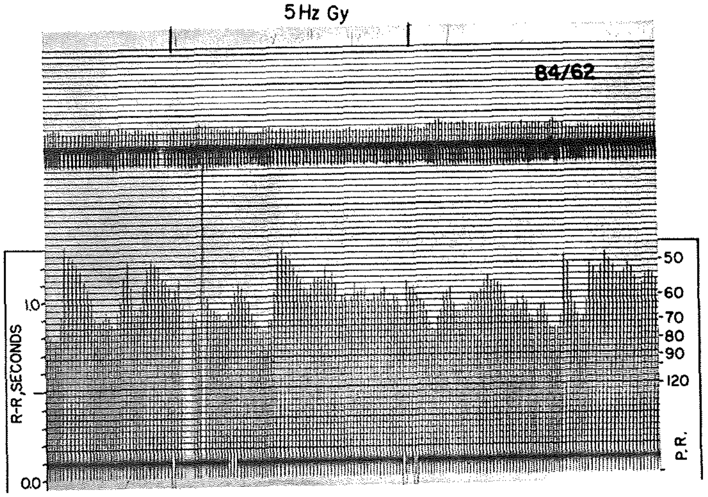


3 Hz Gy






1/8 INCH AMPLITUDE Gy 5 Hz
SUBJECT A SEATED
X
Y
Z


5 Hz Gy
84/62
R-R, SECONDS
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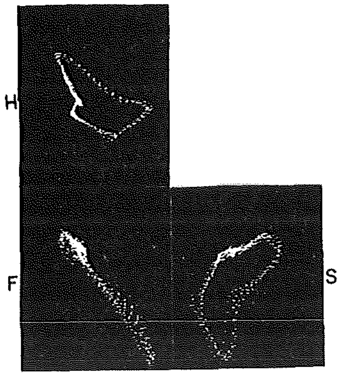
50
60
70
80
90
120
P.R.


H
F
S

1/8 INCH AMPLITUDE Gy 7Hz

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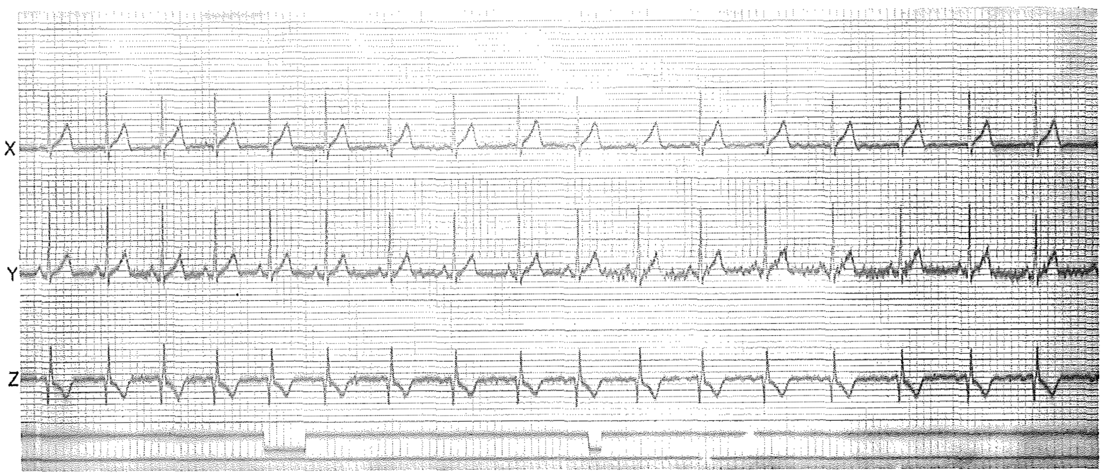
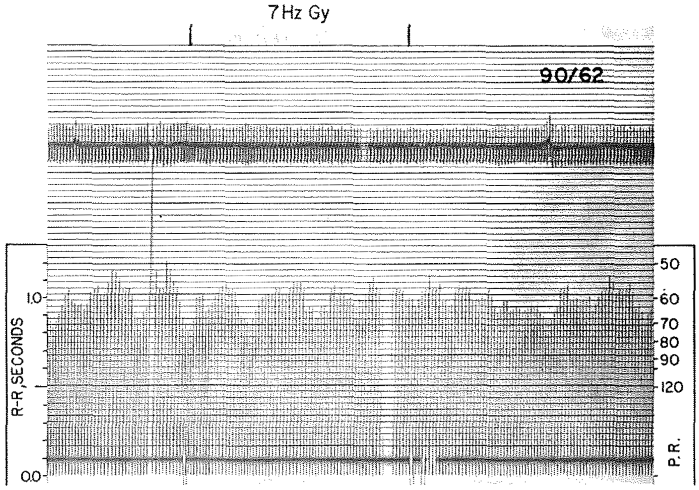
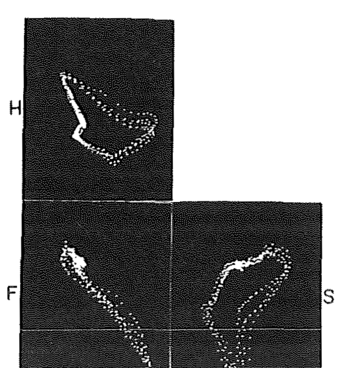
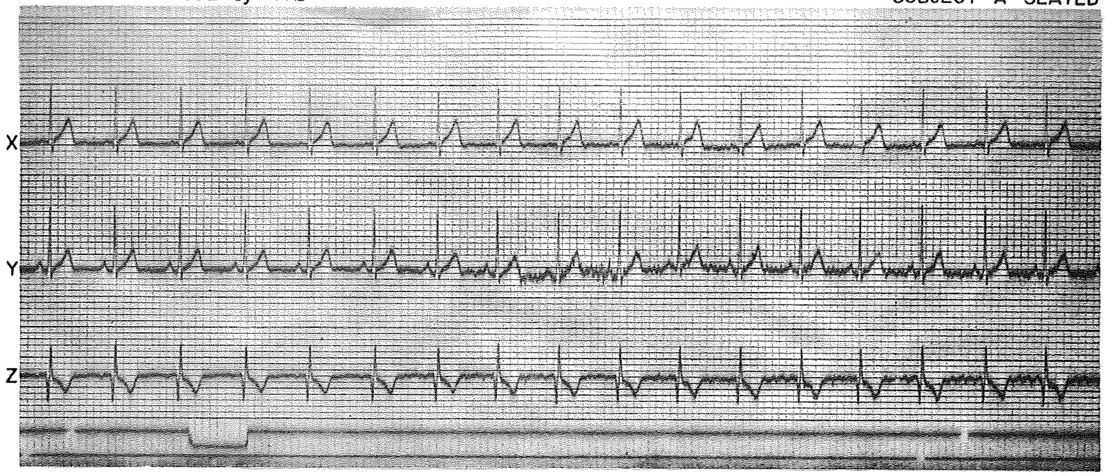


7 Hz Gy

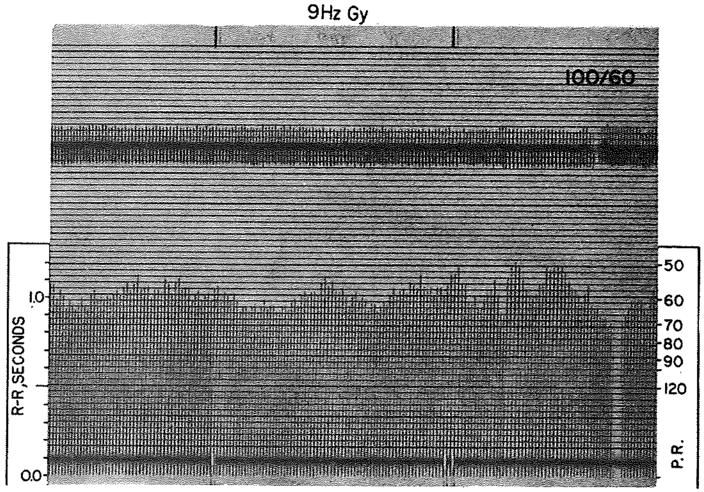


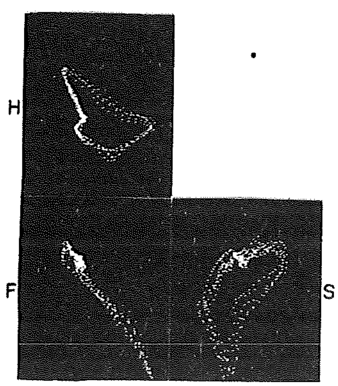


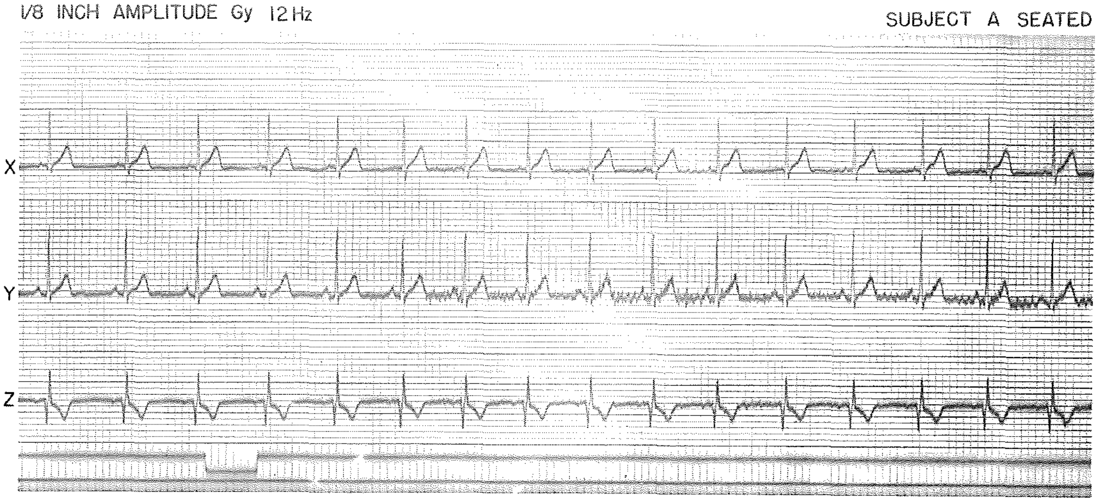
1/8 INCH AMPLITUDE Gy 9Hz SUBJECT A SEATED

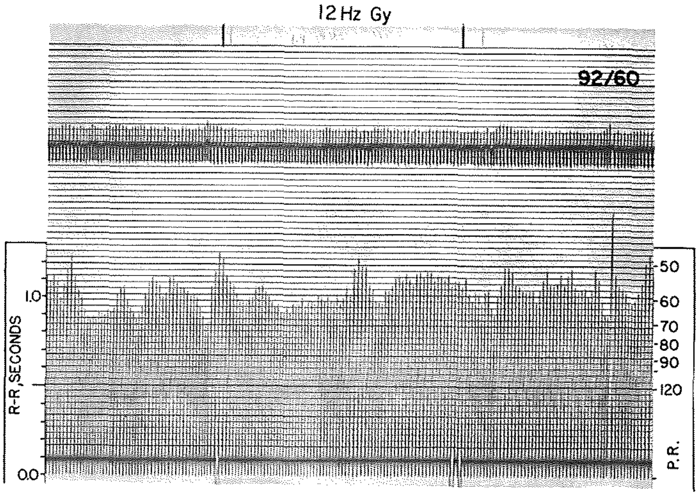


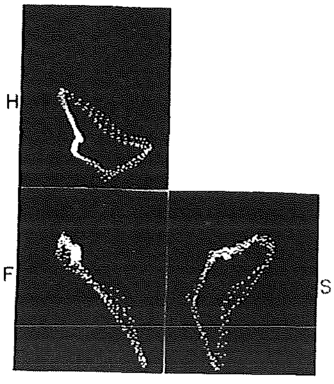
9Hz Gy





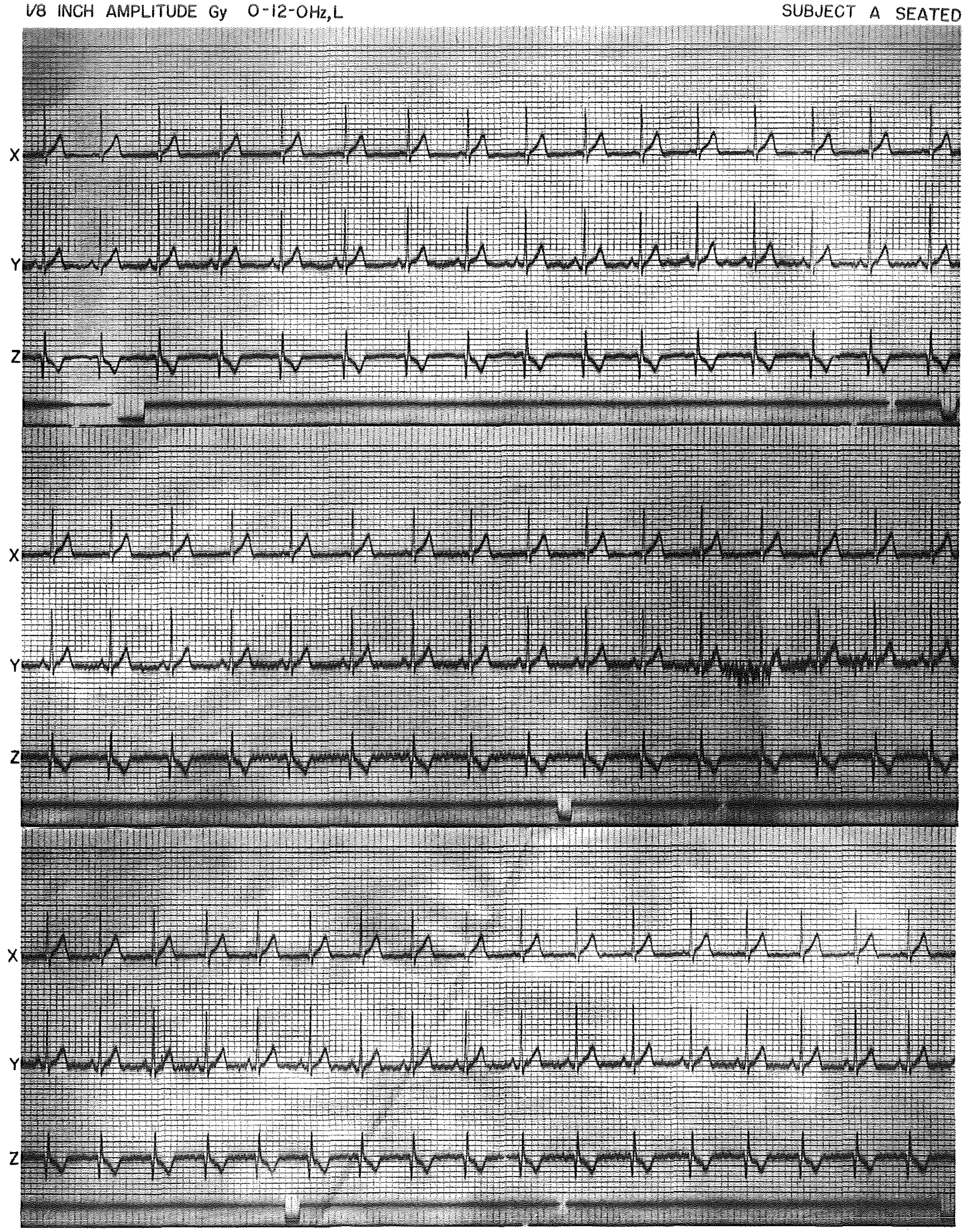

1/8 INCH AMPLITUDE Gy 12 Hz
SUBJECT A SEATED
X
Y
Z


12 Hz Gy
92/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

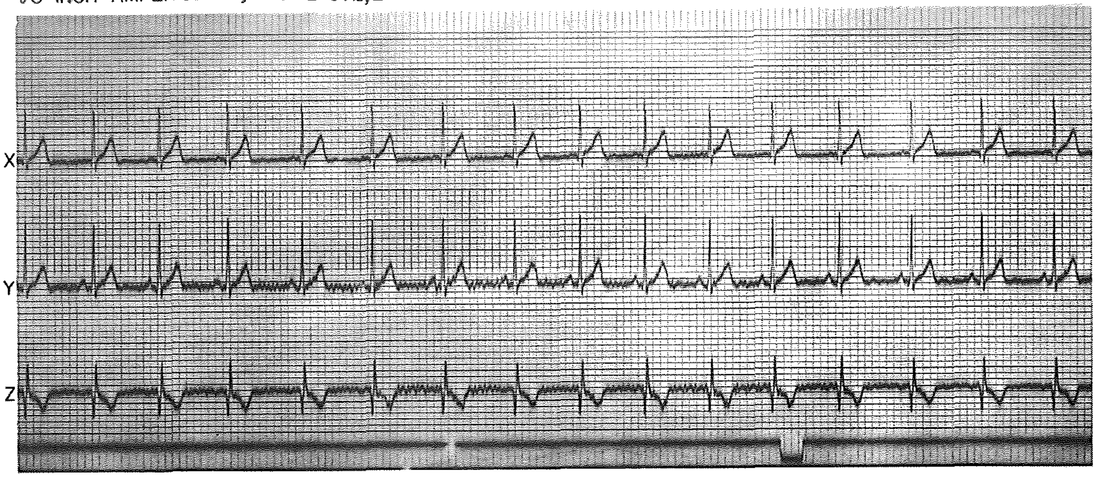

H
F
S

1/8 INCH AMPLITUDE Gy 0-12-0Hz,L

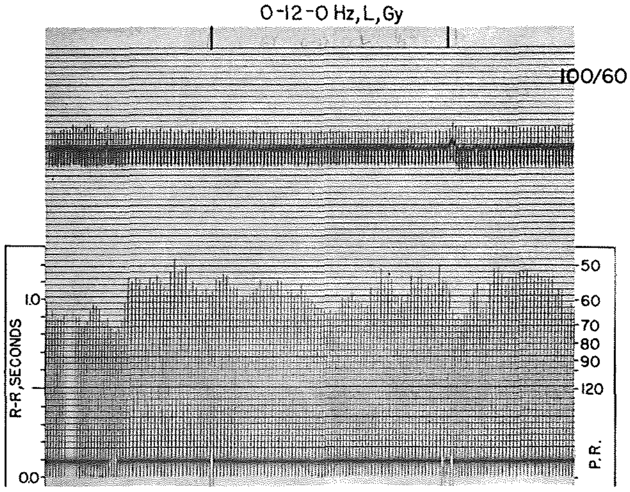
SUBJECT A SEATED



1/8 INCH AMPLITUDE Gy 0-12-0 Hz, L SUBJECT A SEATED



0-12-0 Hz, L, Gy



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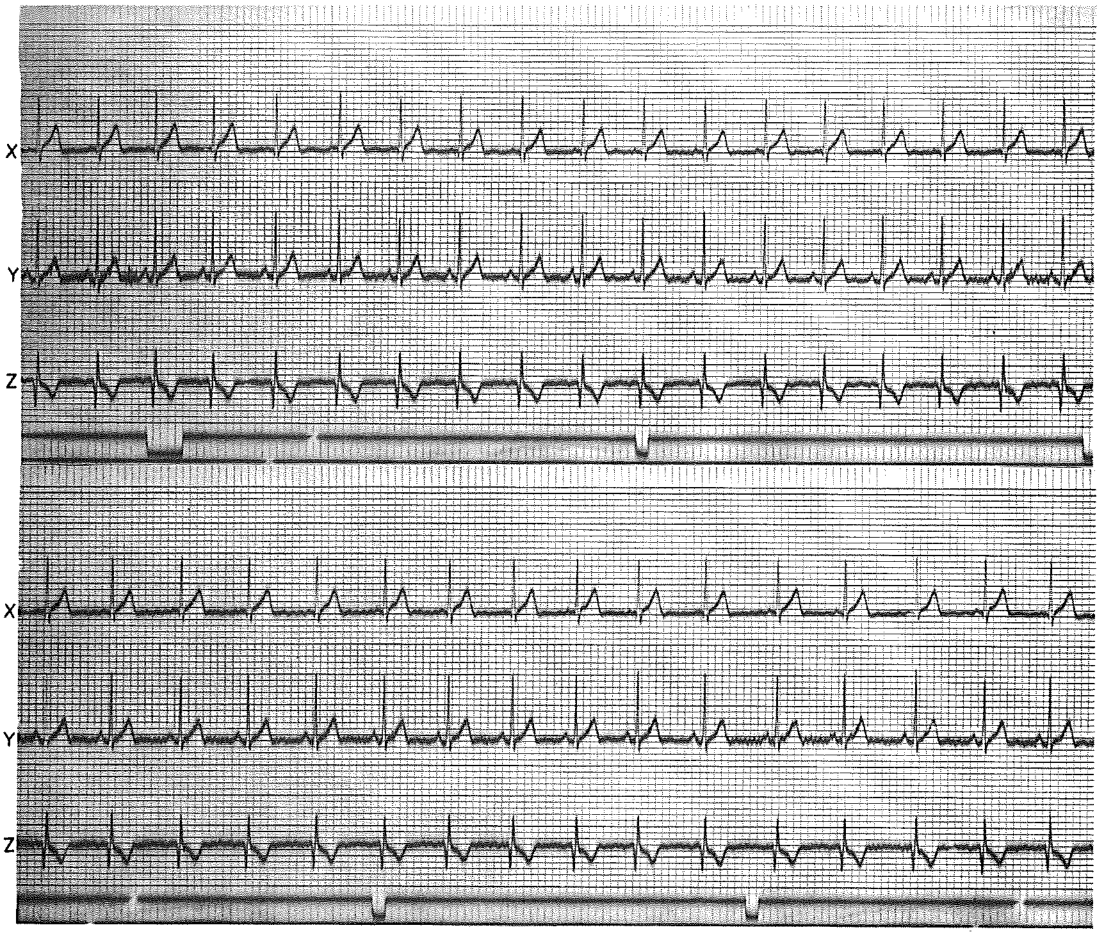
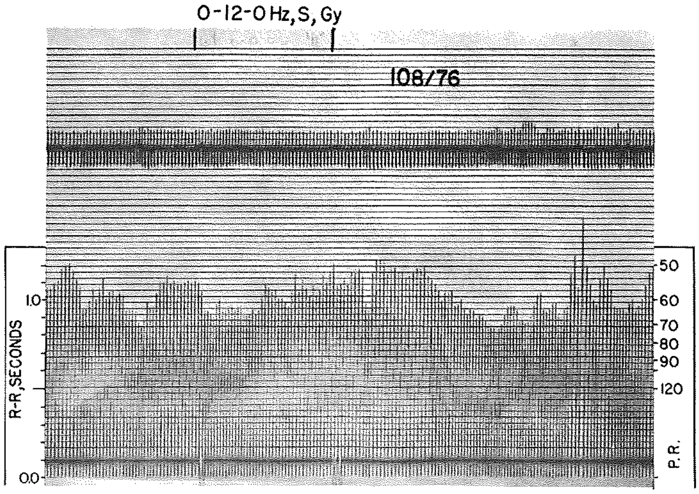
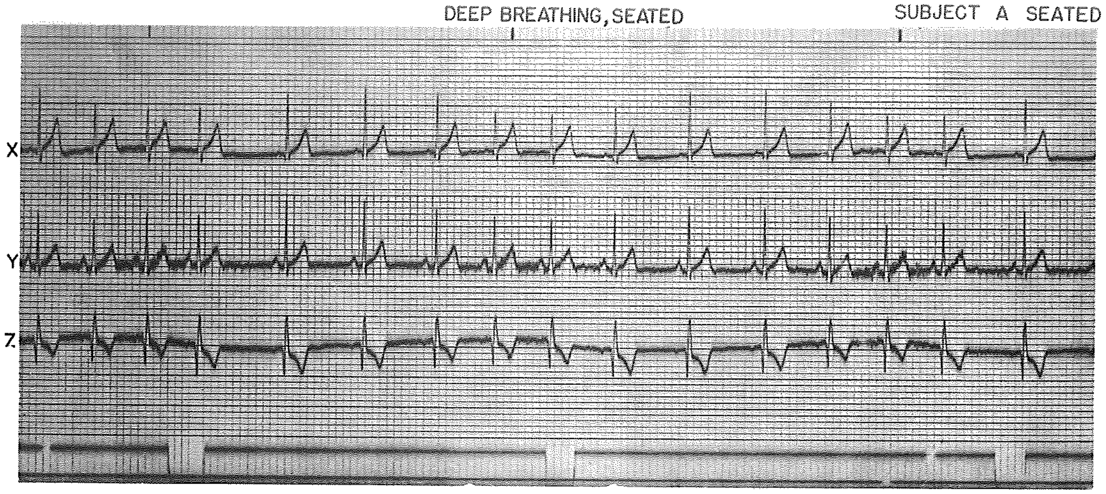
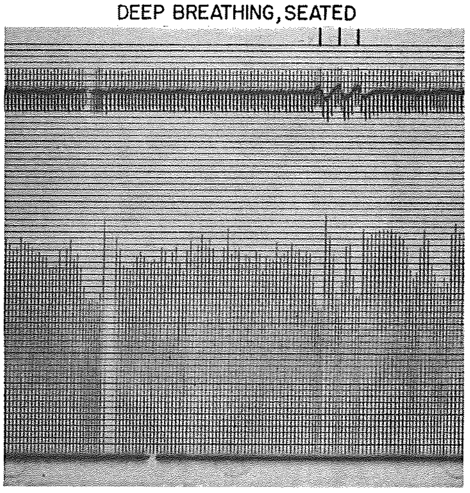


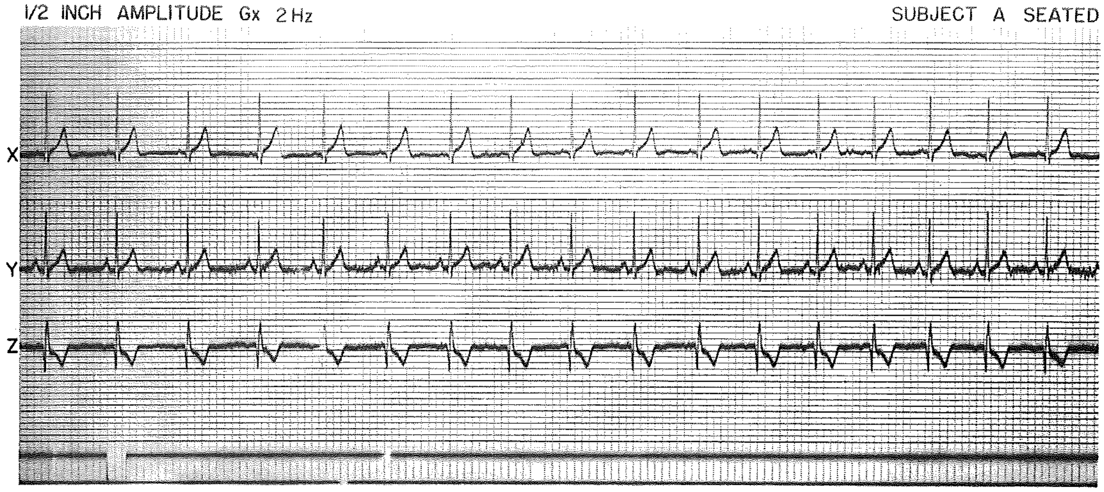
0-12-0 Hz, S, Gy



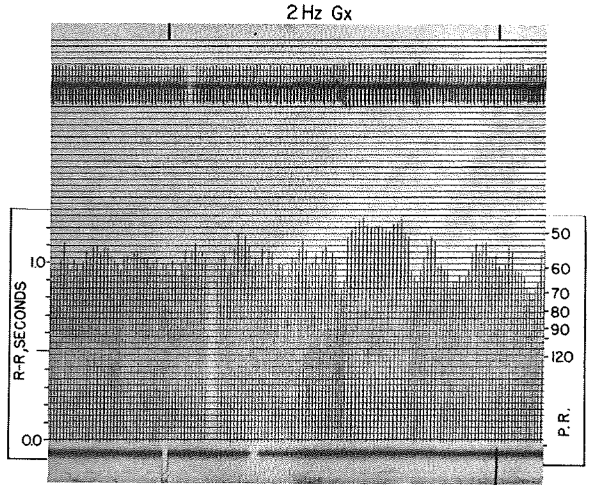

DEEP BREATHING, SEATED
SUBJECT A SEATED
X
Y
Z


DEEP BREATHING, SEATED

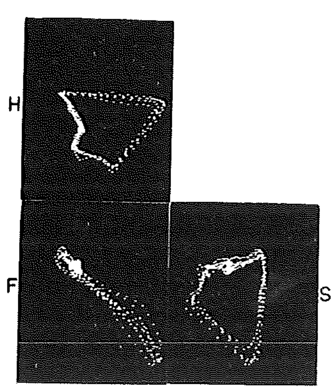
1/2 INCH AMPLITUDE Gx 2 Hz SUBJECT A SEATED



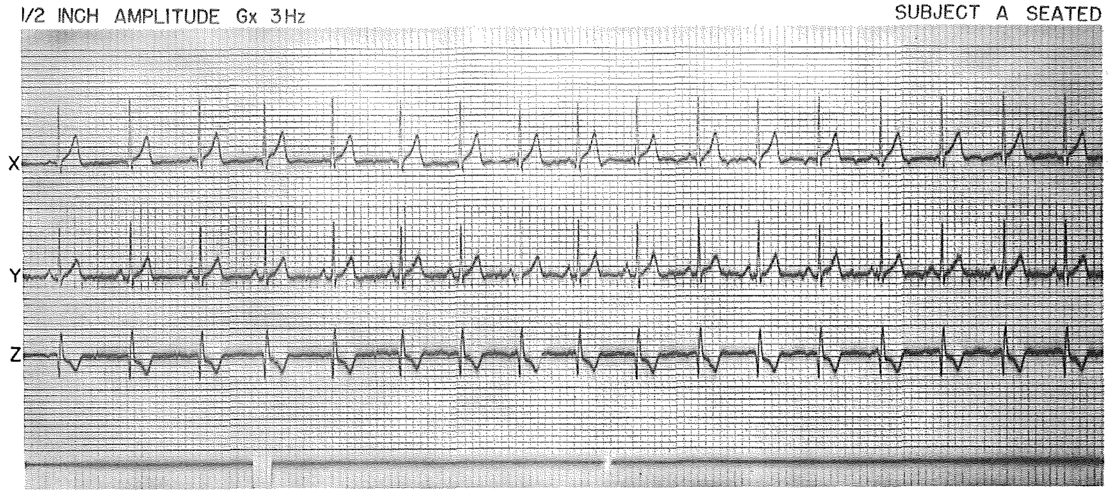
2 Hz Gx



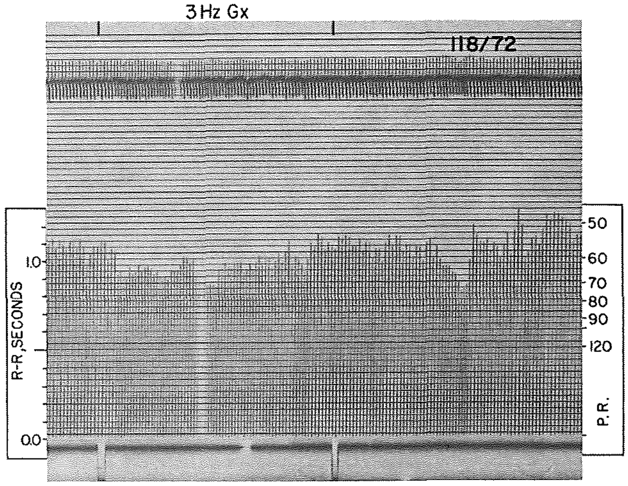
118/70

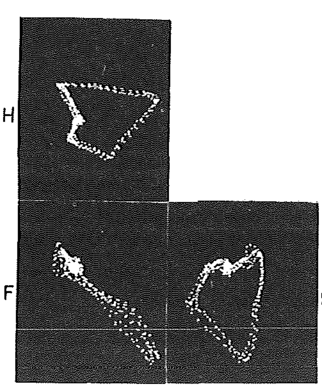


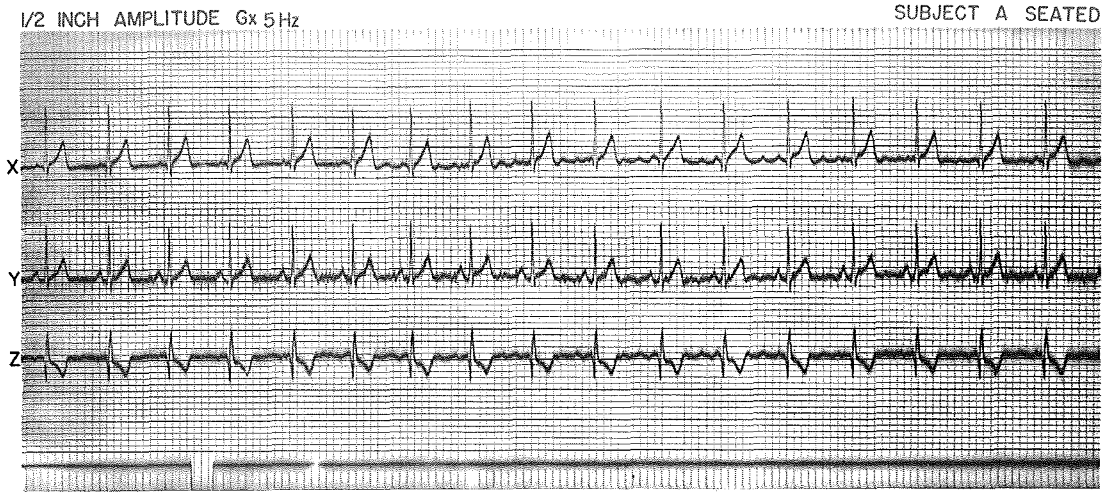
1/2 INCH AMPLITUDE Gx 3Hz SUBJECT A SEATED



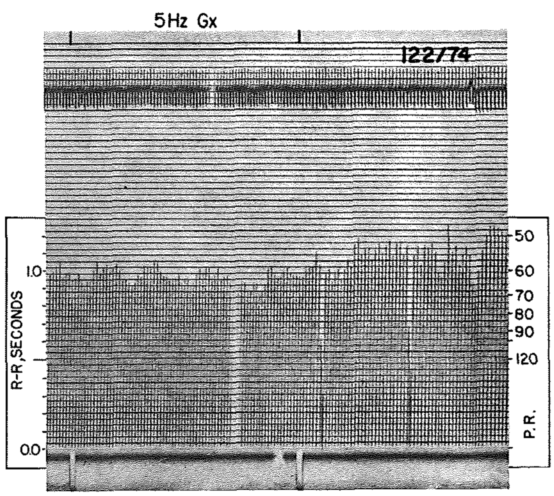
3 Hz Gx



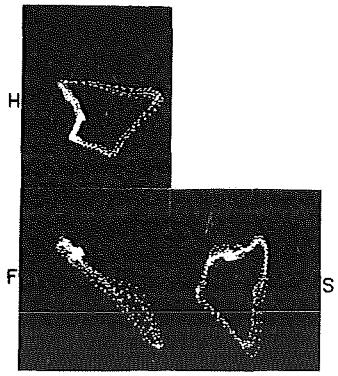




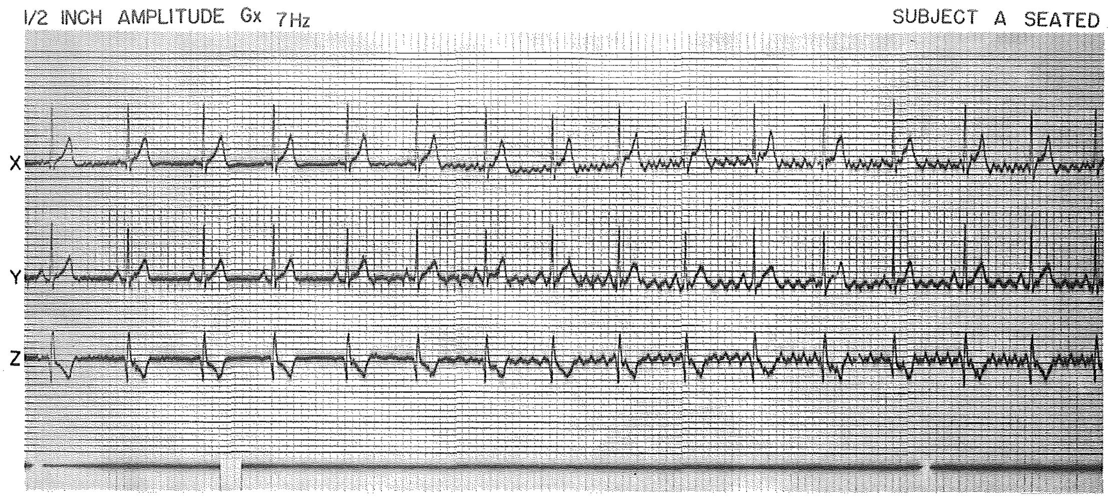
1/2 INCH AMPLITUDE Gx 5 Hz
SUBJECT A SEATED
X
Y
Z



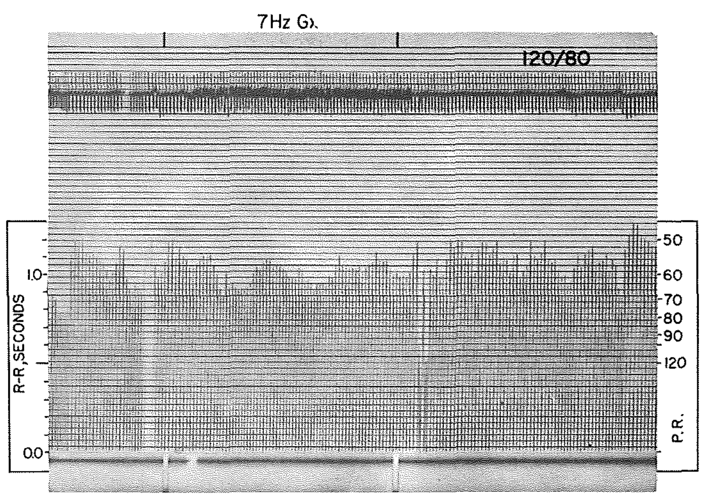
5Hz Gx
122/74
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.



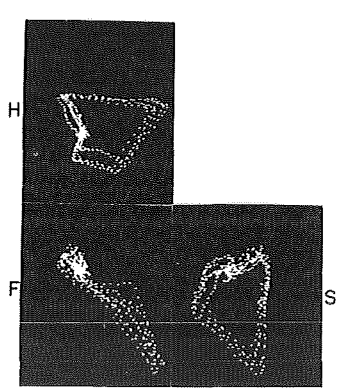
H
F
S



1/2 INCH AMPLITUDE Gx 7Hz
SUBJECT A SEATED
X
Y
Z



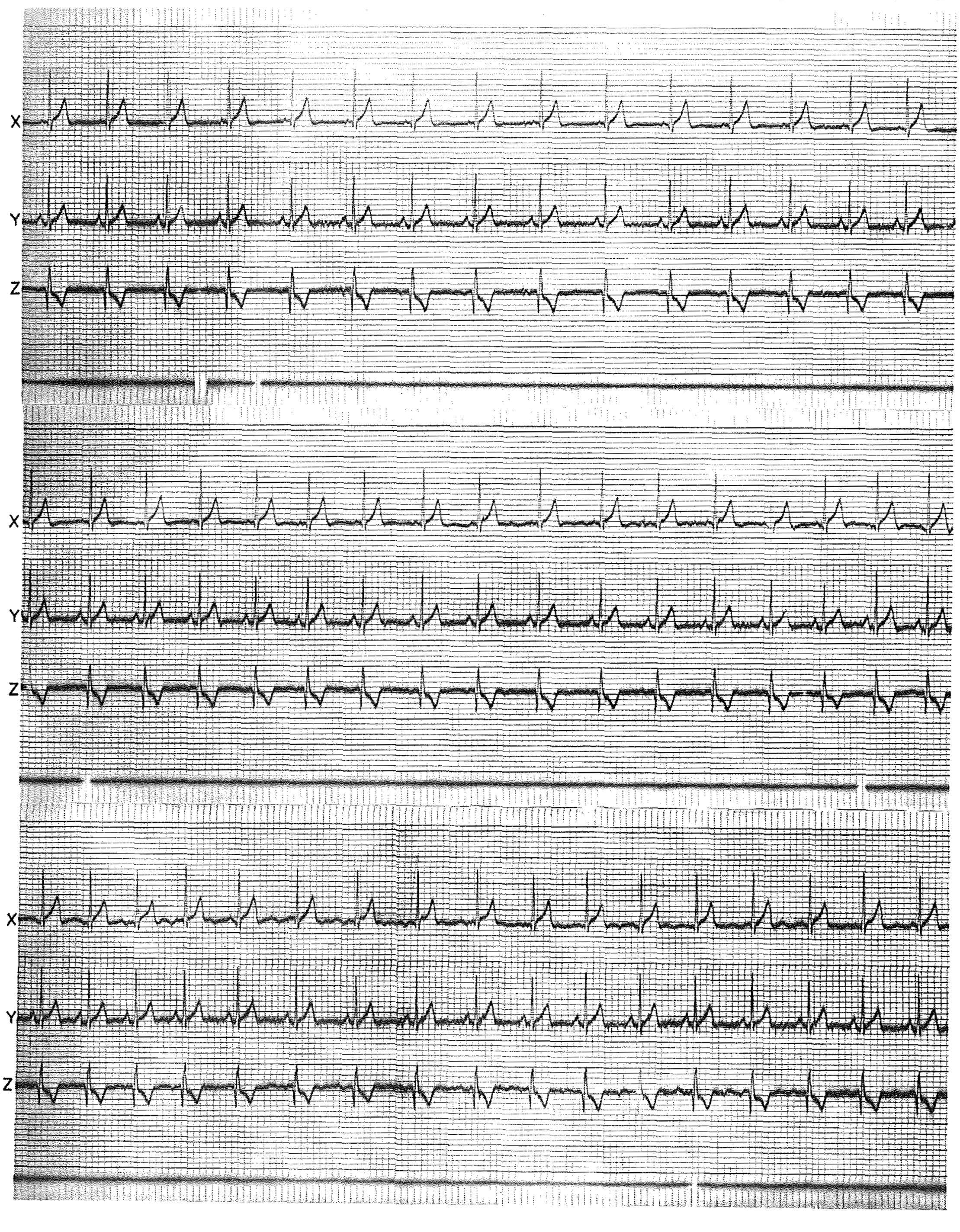
7Hz Gx
120/80
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

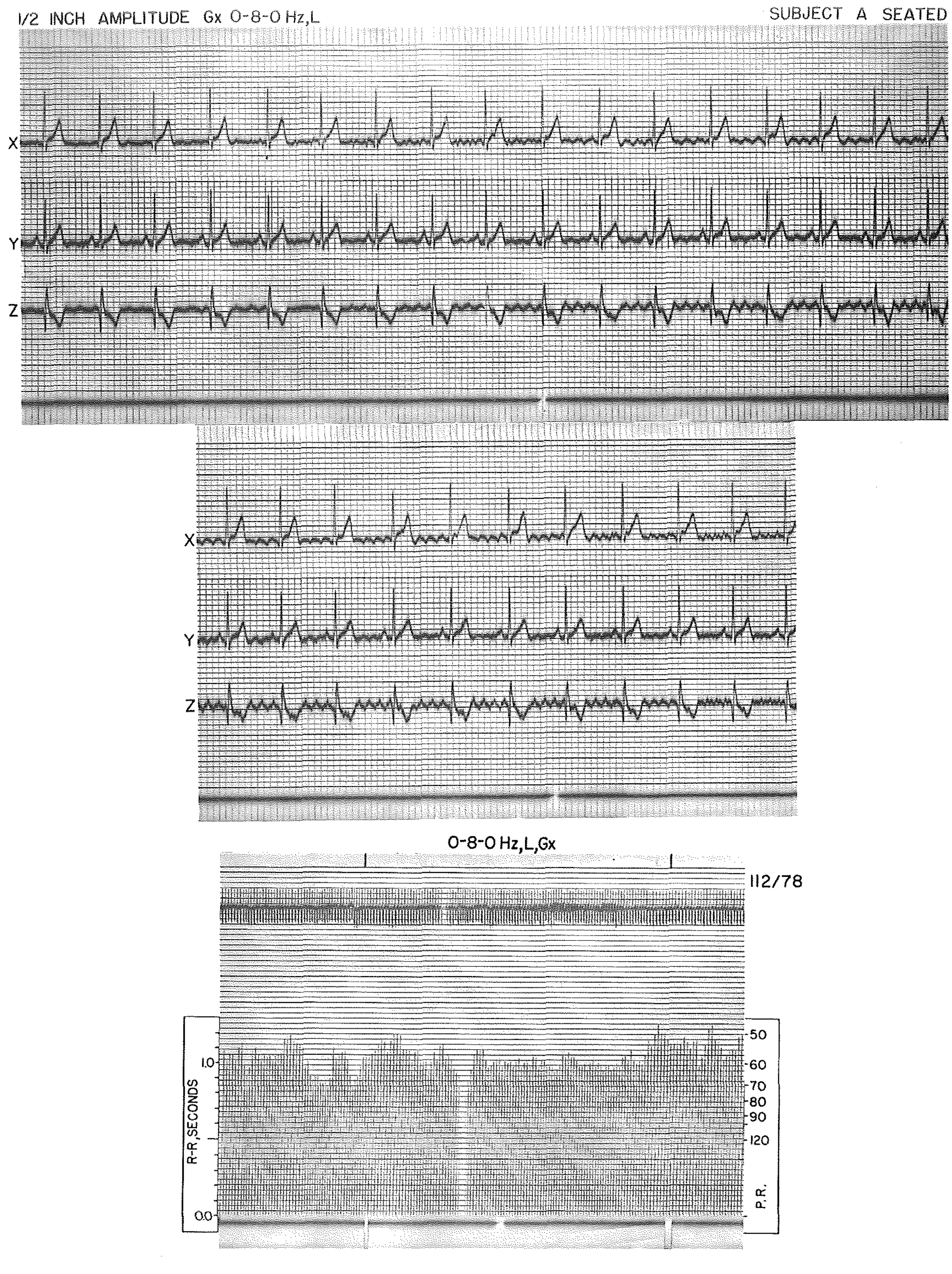


H
F
S

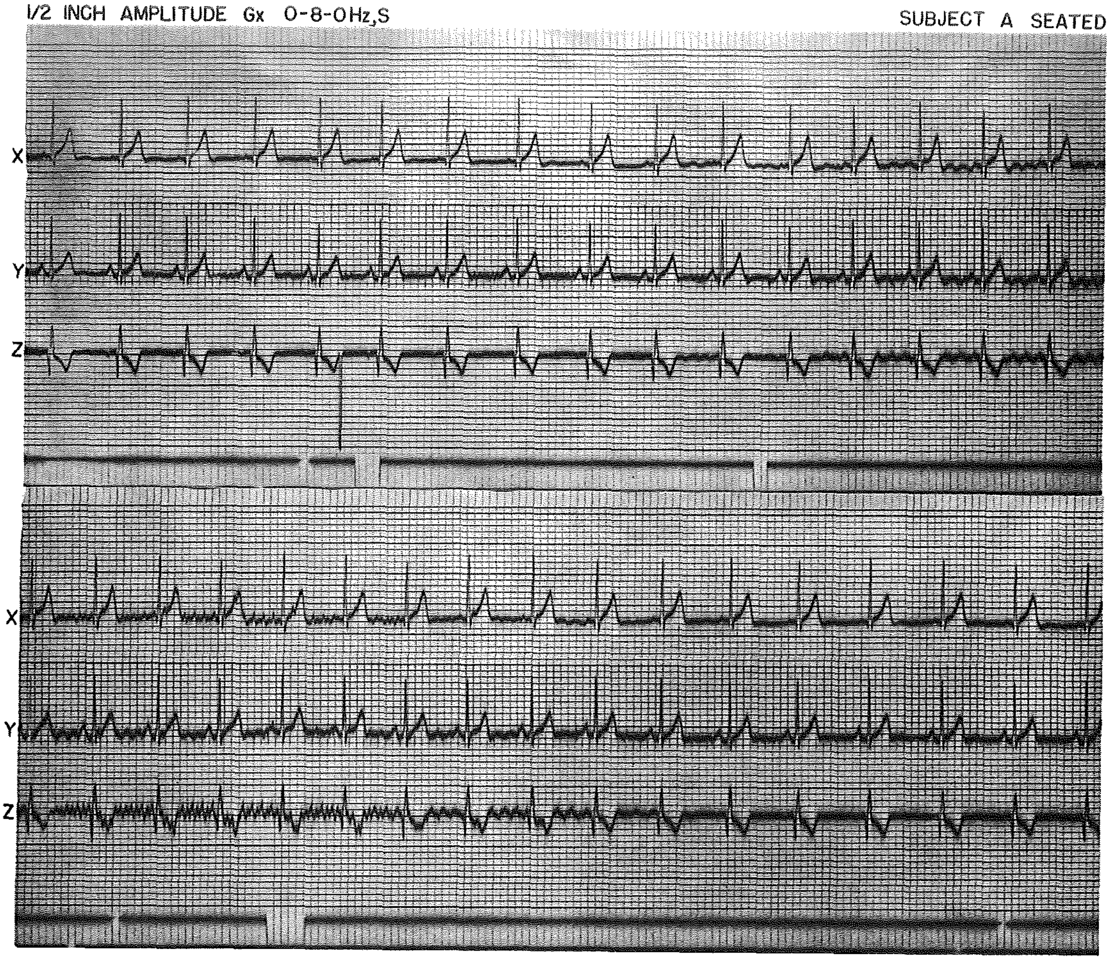
1/2 INCH AMPLITUDE Gx 0-8-0 Hz,L

SUBJECT A SEATED

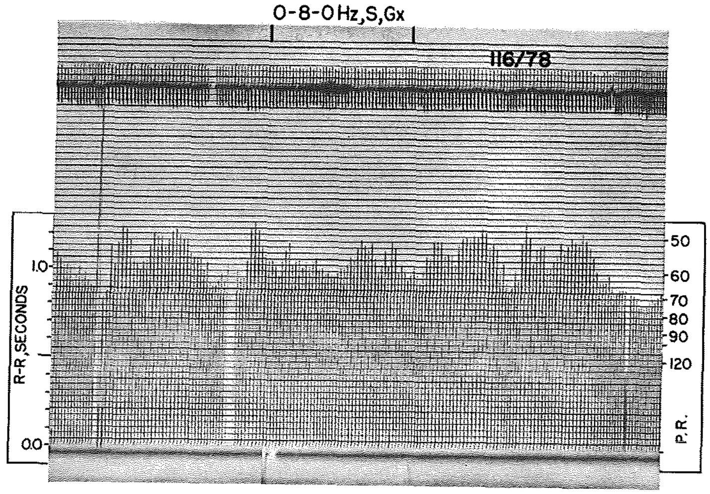


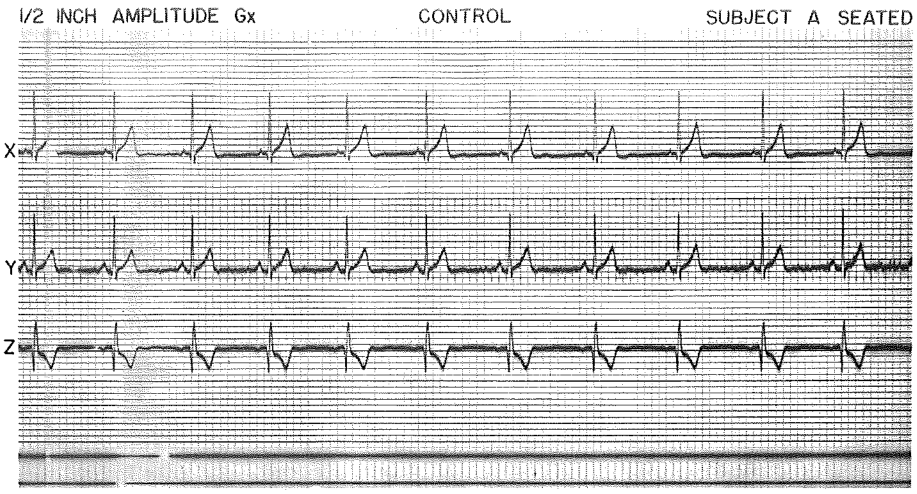

1/2 INCH AMPLITUDE Gx 0-8-0 Hz,L
SUBJECT A SEATED
X
Y
Z
X
Y
Z
0-8-0 Hz,L,Gx
112/78
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

1/2 INCH AMPLITUDE Gx 0-8-0 Hz,S SUBJECT A SEATED

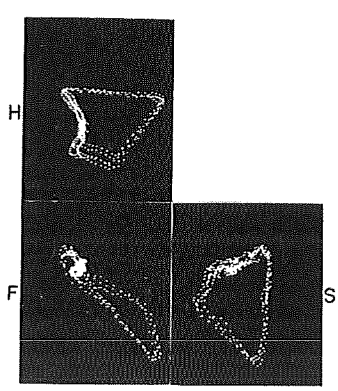


0-8-0 Hz,S,Gx

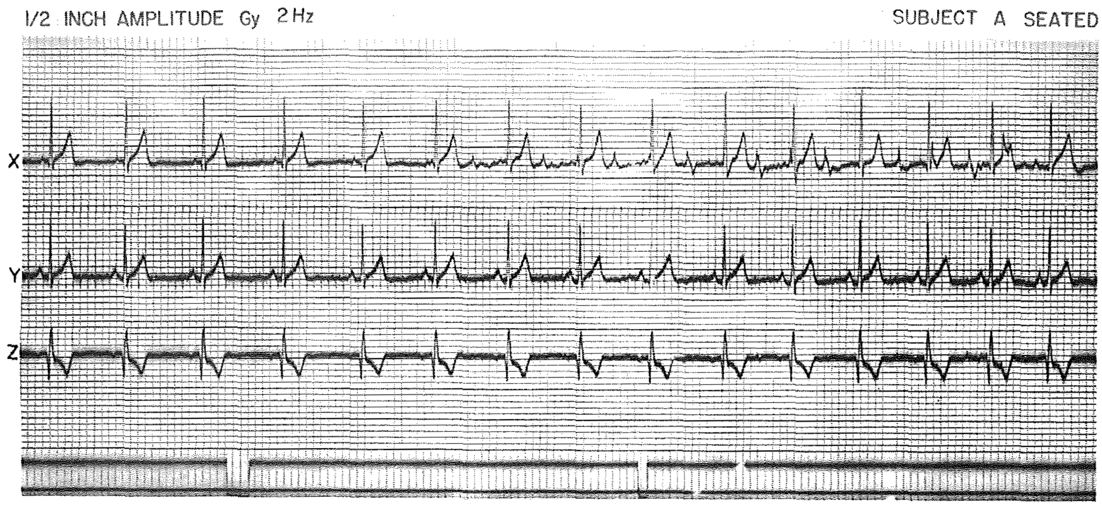


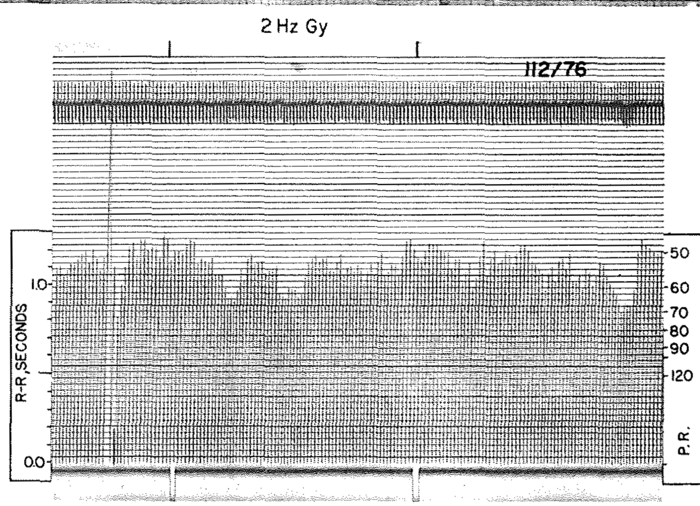


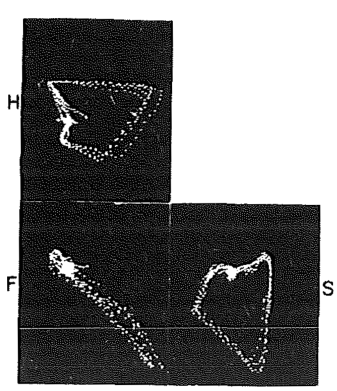
1/2 INCH AMPLITUDE Gx
CONTROL
SUBJECT A SEATED
X
Y
Z

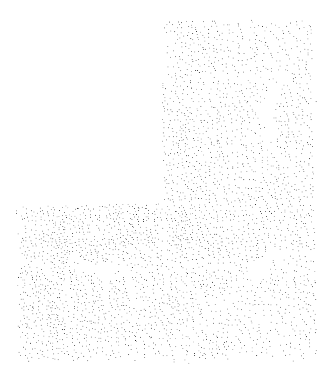


H
F
S

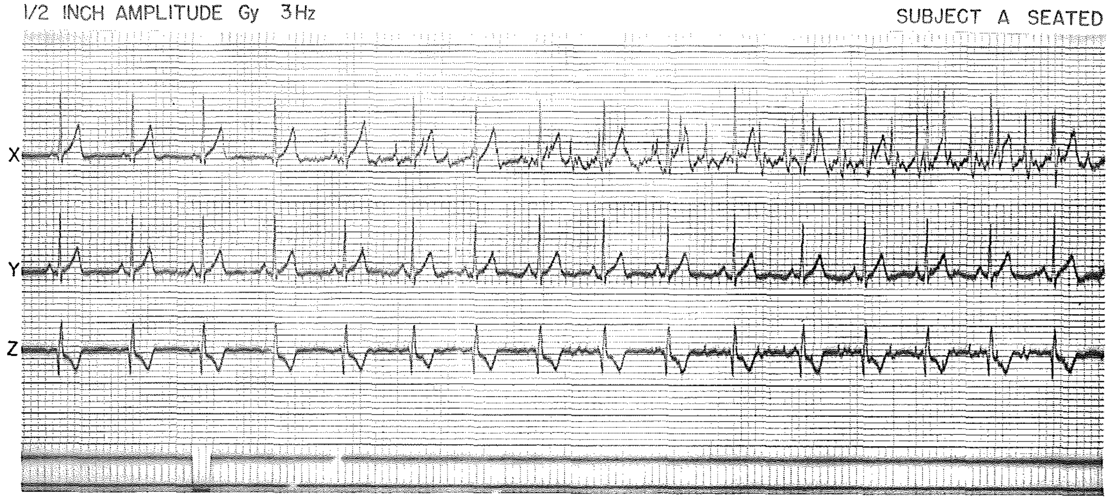

1/2 INCH AMPLITUDE Gy 2 Hz
SUBJECT A SEATED
X
Y
Z


2 Hz Gy
112/76
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

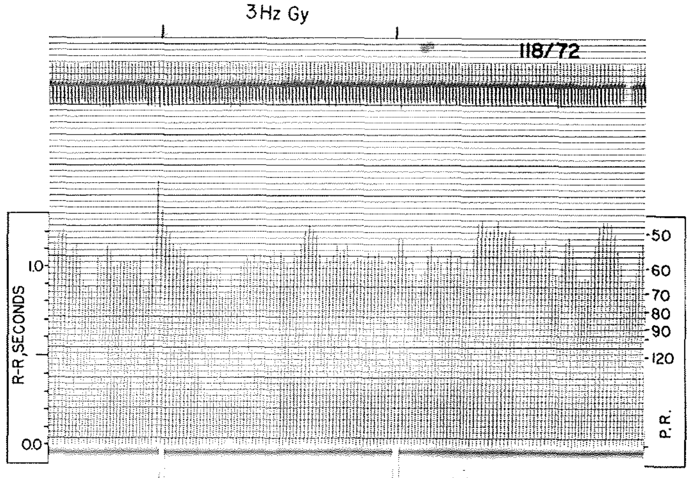

H
F
S

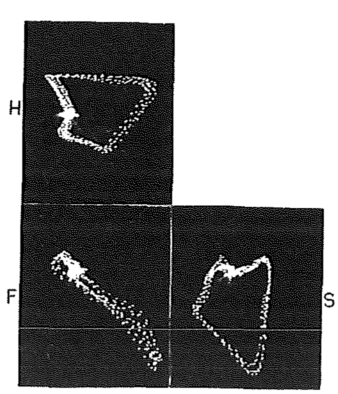


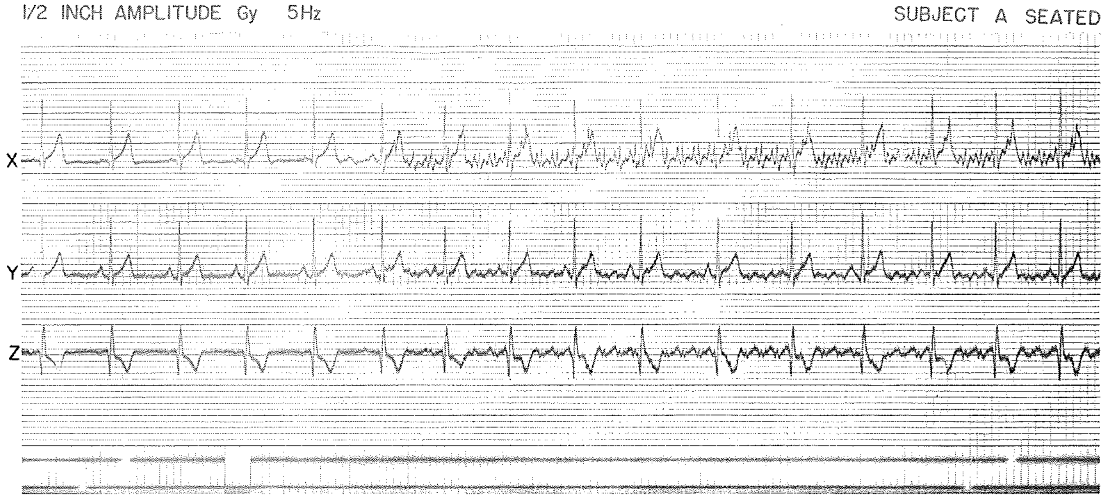
1/2 INCH AMPLITUDE Gy 3Hz SUBJECT A SEATED

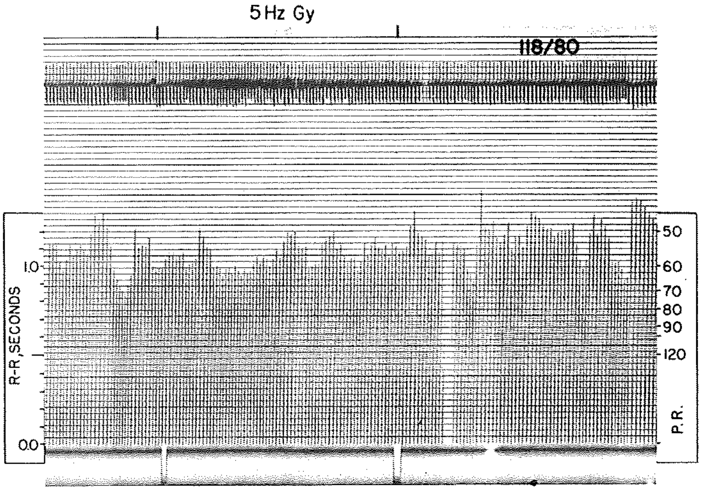


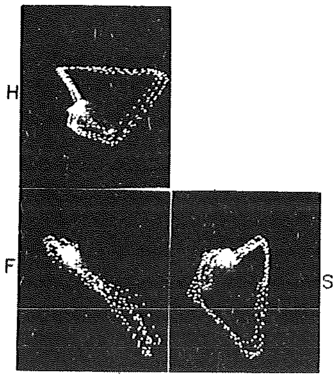
3Hz Gy



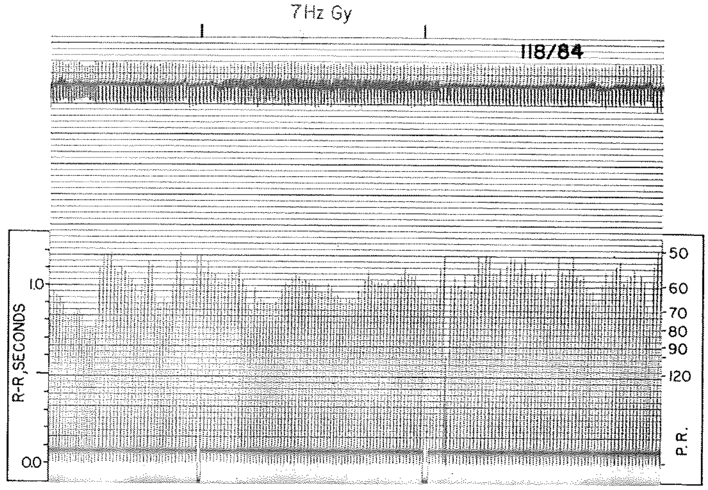


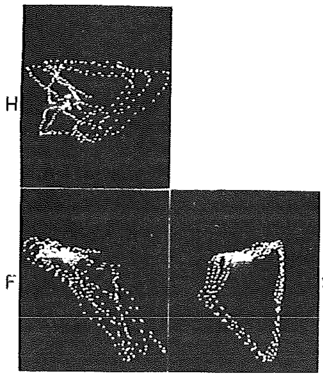

1/2 INCH AMPLITUDE Gy 5Hz
SUBJECT A SEATED
X
Y
Z


5 Hz Gy
118/80
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

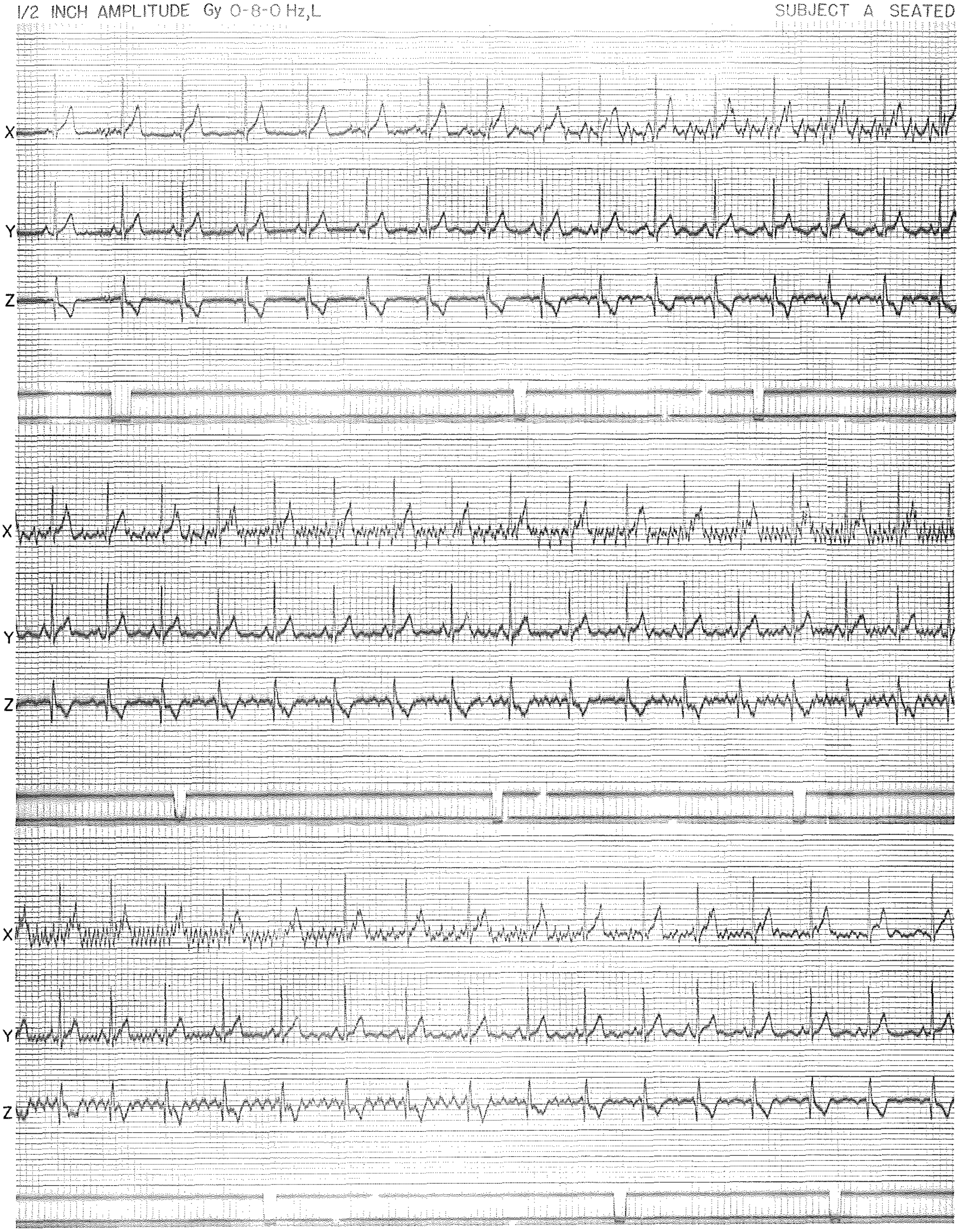

H
F
S

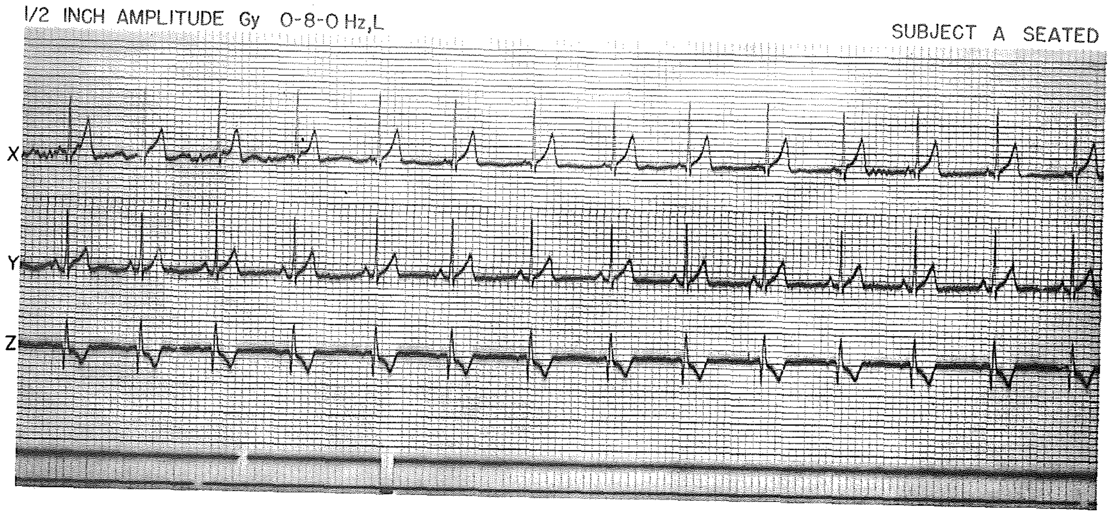

1/2 INCH AMPLITUDE Gy 7Hz
SUBJECT A SEATED
X
Y
Z

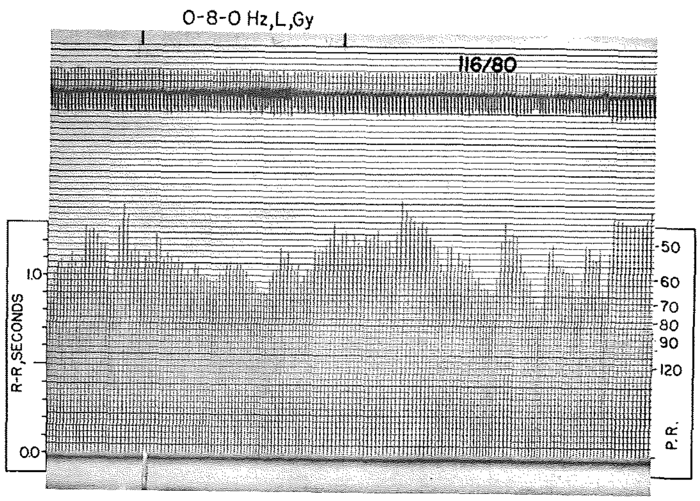

7Hz Gy
118/84
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

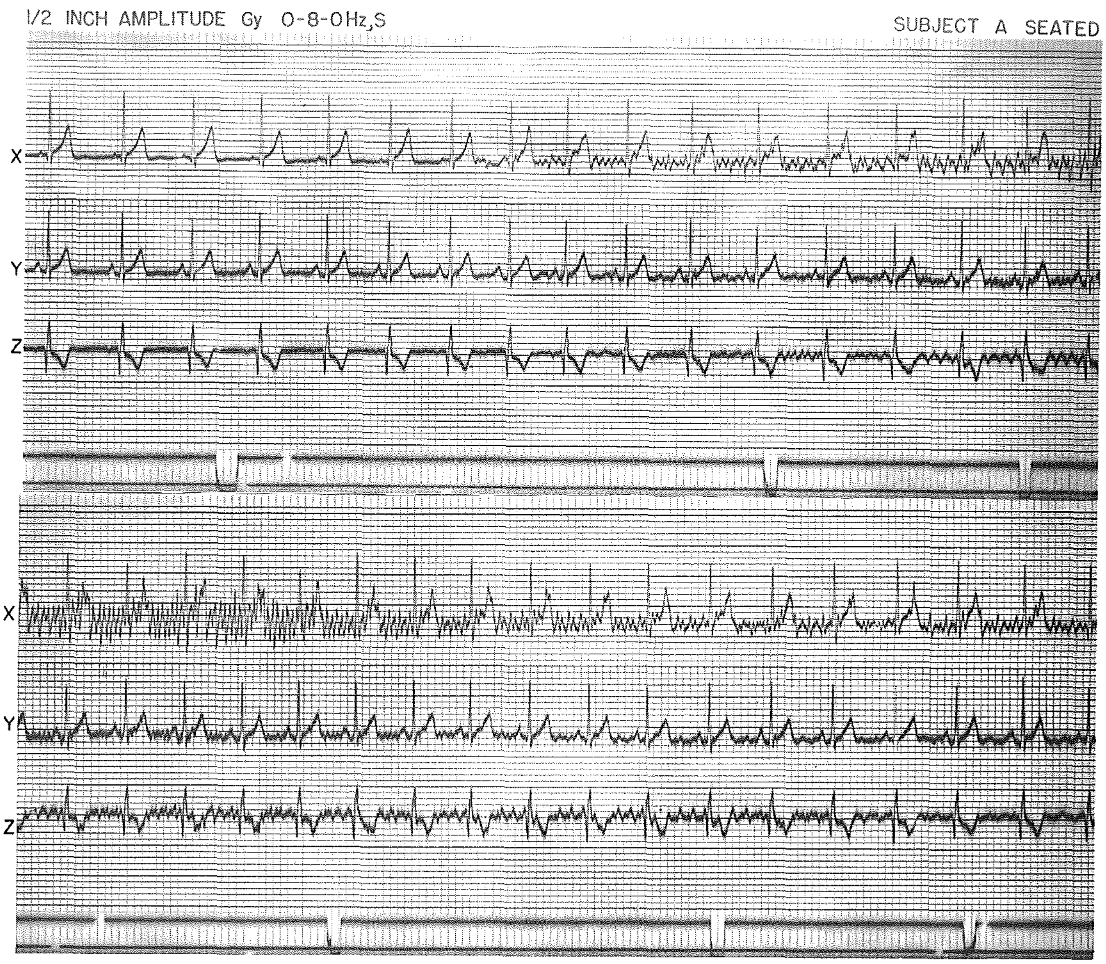

H
F
S

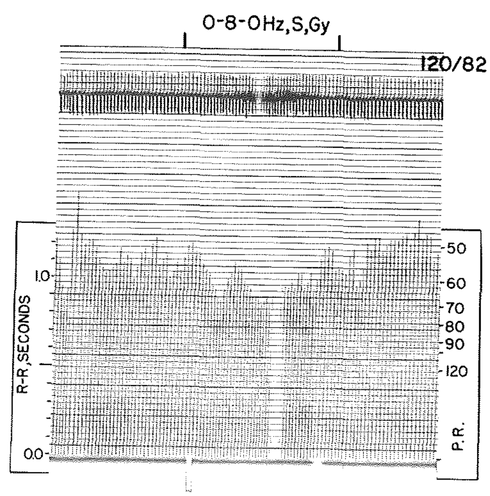
I/2 INCH AMPLITUDE Gy O-8-O Hz,L SUBJECT A SEATED




1/2 INCH AMPLITUDE Gy 0-8-0 Hz,L
SUBJECT A SEATED
X
Y
Z

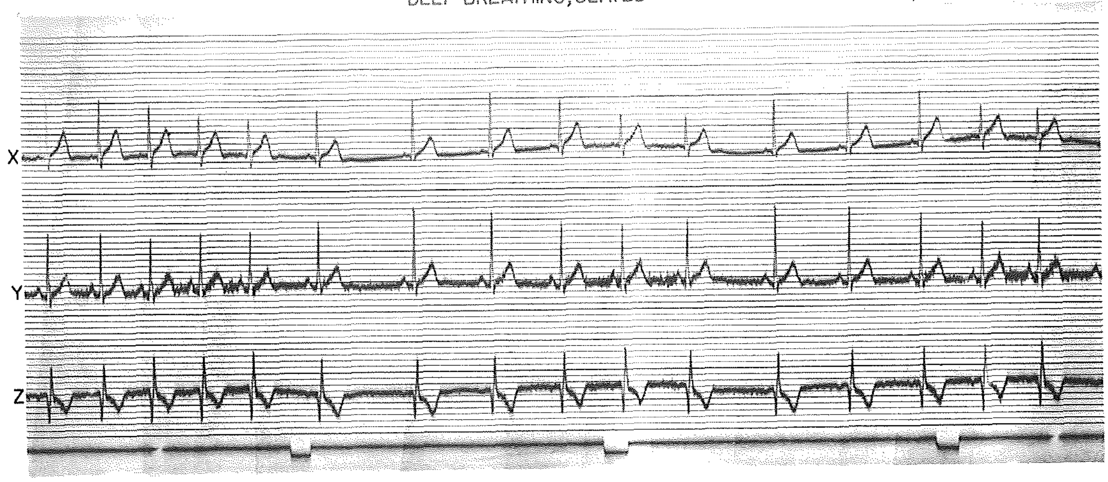

0-8-0 Hz,L,Gy
116/80
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

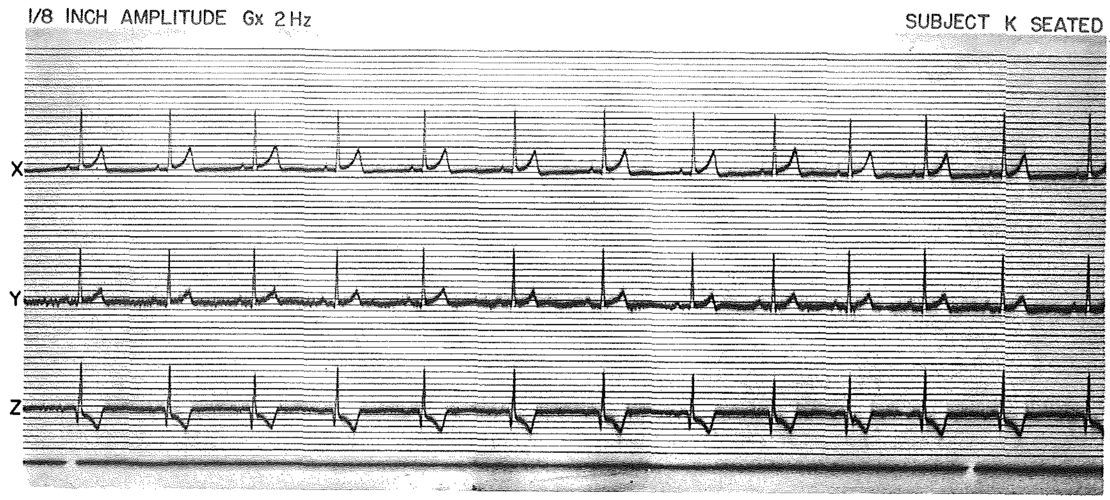

1/2 INCH AMPLITUDE Gy 0-8-0Hz,S
SUBJECT A SEATED
X
Y
Z
X
Y
Z

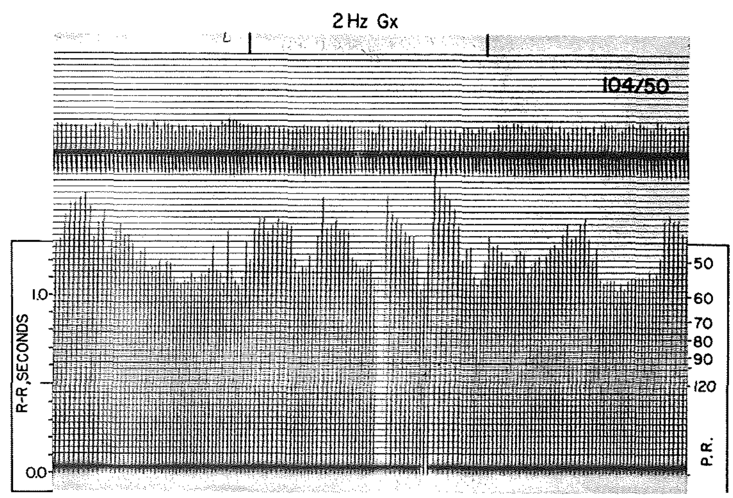

0-8-0Hz,S,Gy
120/82
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

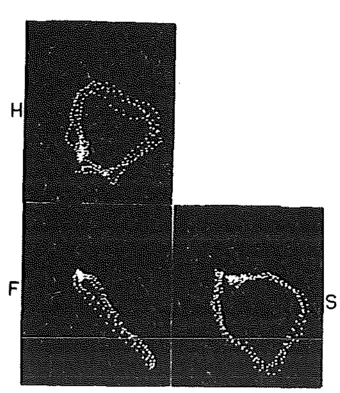
DEEP BREATHING, SEATED

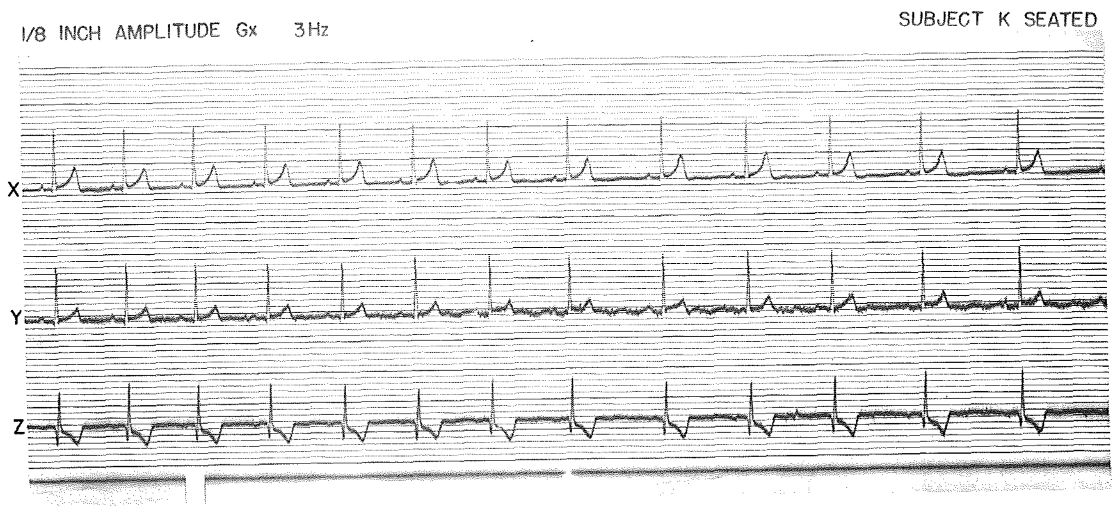
SUBJECT A SEATED

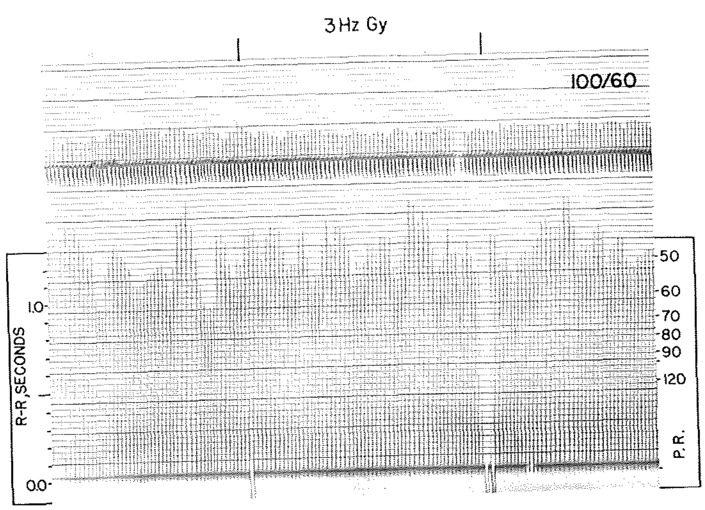


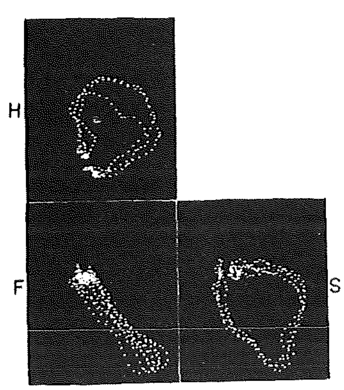

1/8 INCH AMPLITUDE Gx 2 Hz
SUBJECT K SEATED
X
Y
Z

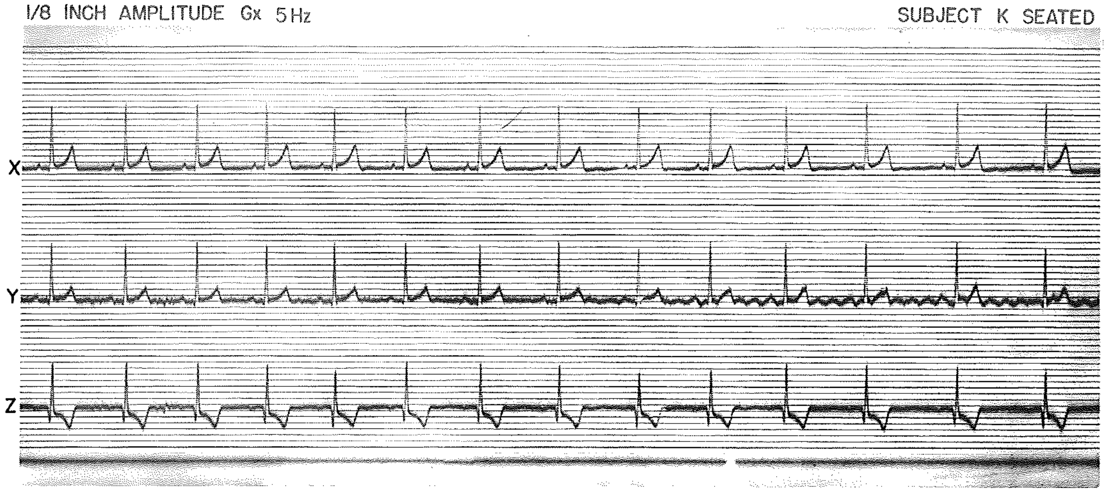

2 Hz Gx
104/50
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

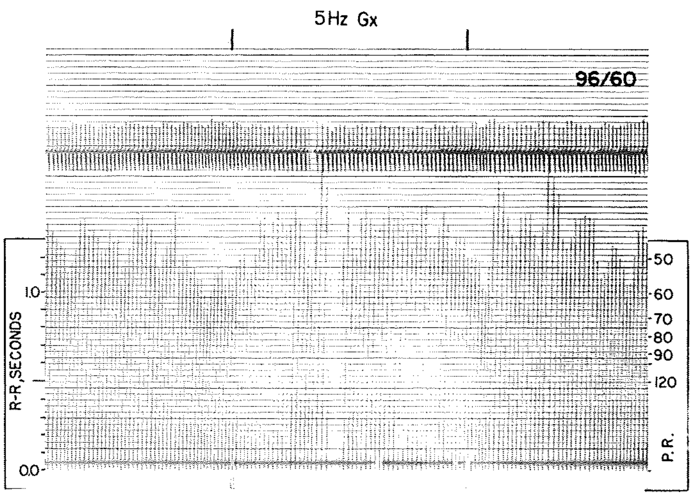

H
F
S

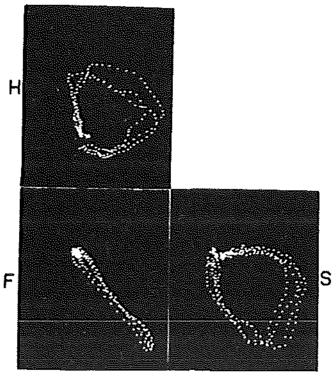

1/8 INCH AMPLITUDE Gx 3 Hz
SUBJECT K SEATED
X
Y
Z

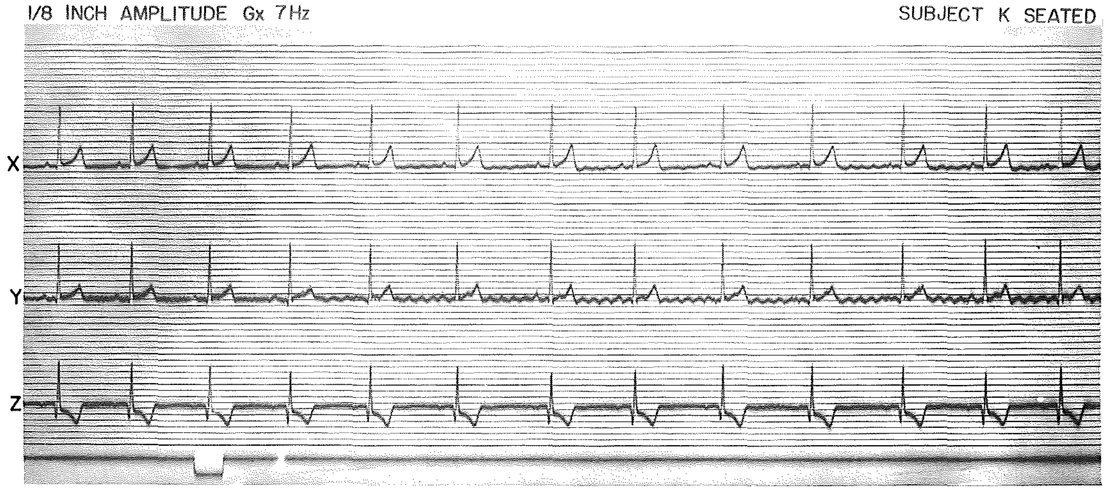

3 Hz Gy
100/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

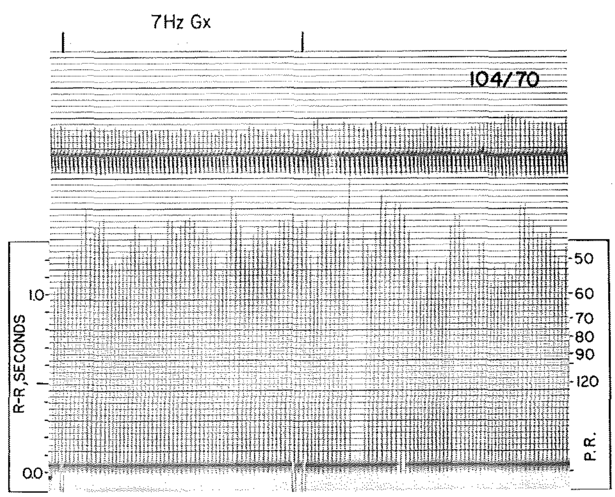

H
F
S

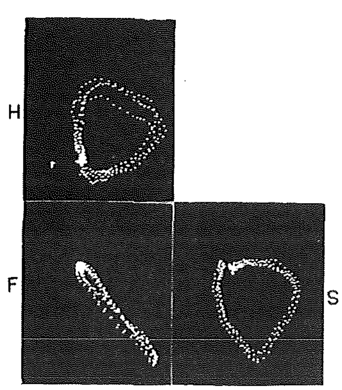

1/8 INCH AMPLITUDE Gx 5 Hz
SUBJECT K SEATED
X
Y
Z


5 Hz Gx
96/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.


H
F
S

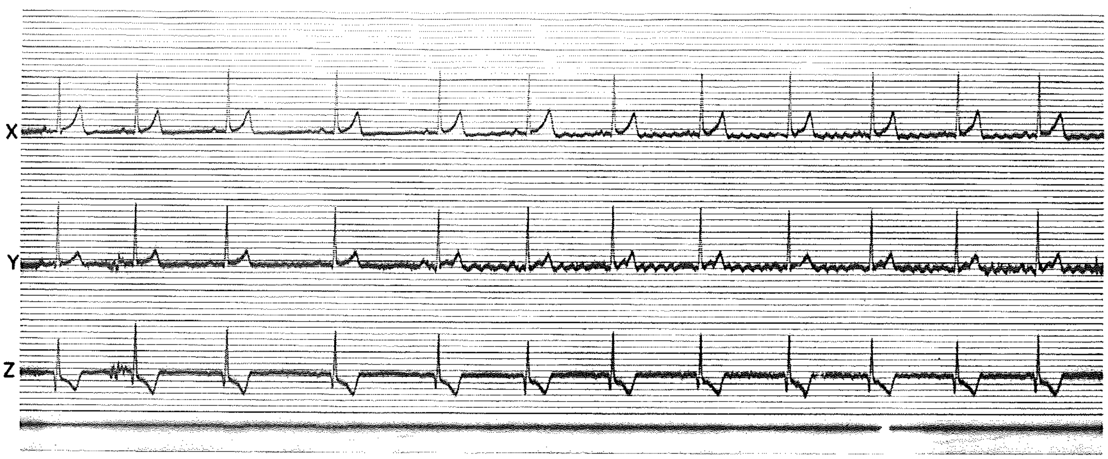

1/8 INCH AMPLITUDE Gx 7Hz
SUBJECT K SEATED
X
Y
Z

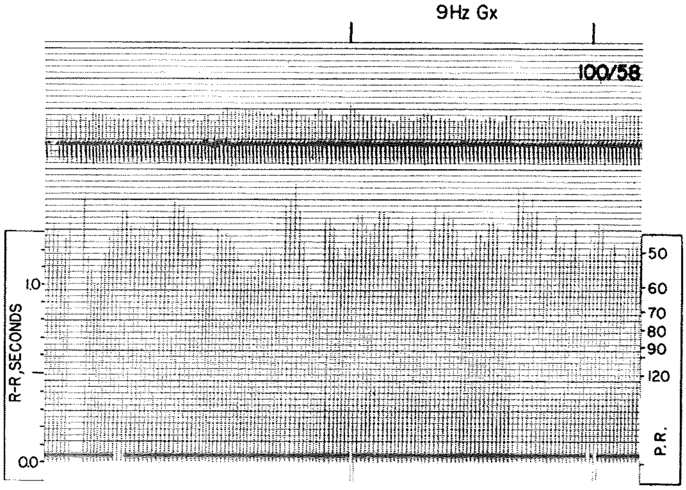

7Hz Gx
104/70
1.0
R-R, SECONDS
0.0
50
60
70
80
90
120
P.R.

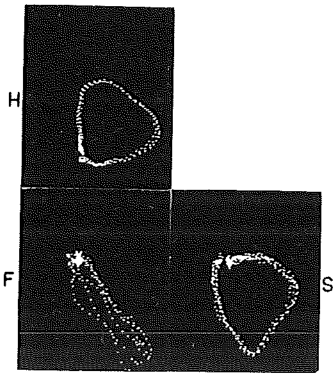

H
F
S

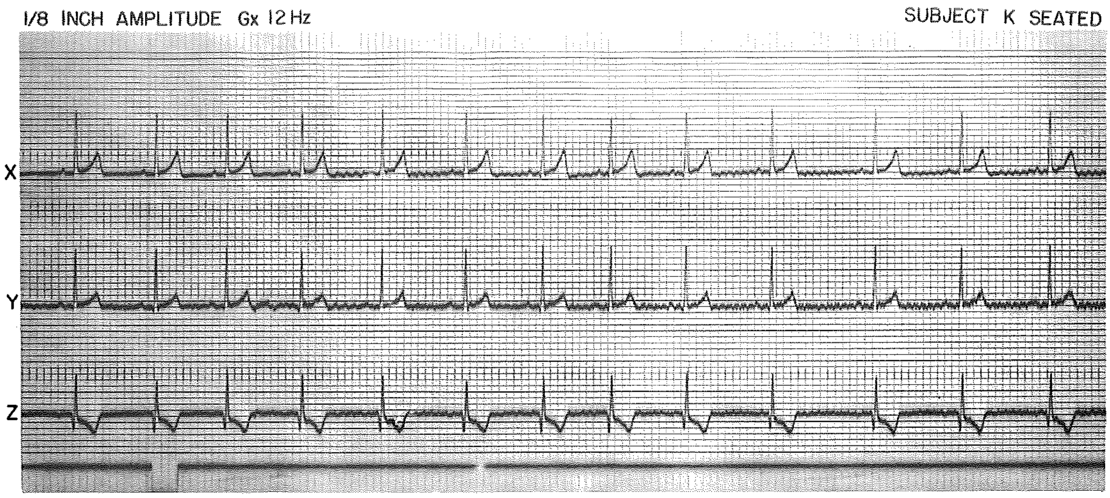
1/8 INCH AMPLITUDE Gx 9Hz

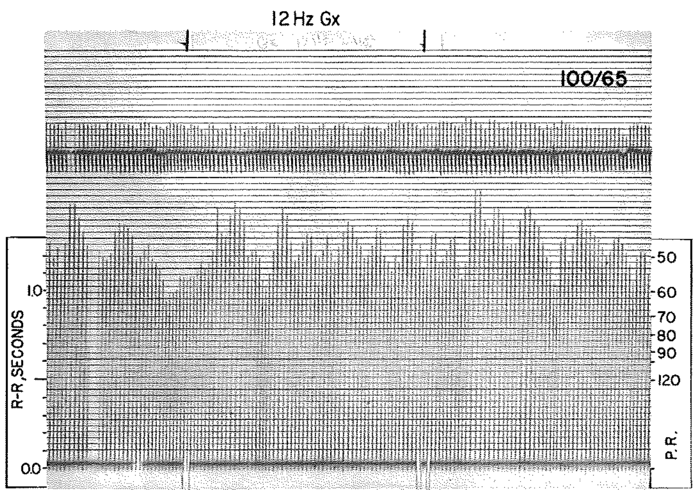
SUBJECT K SEATED

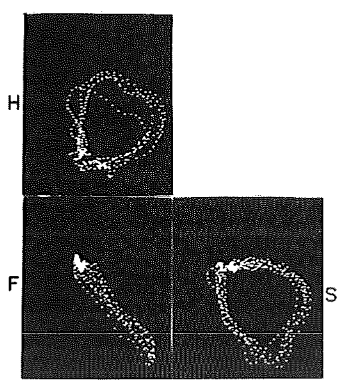




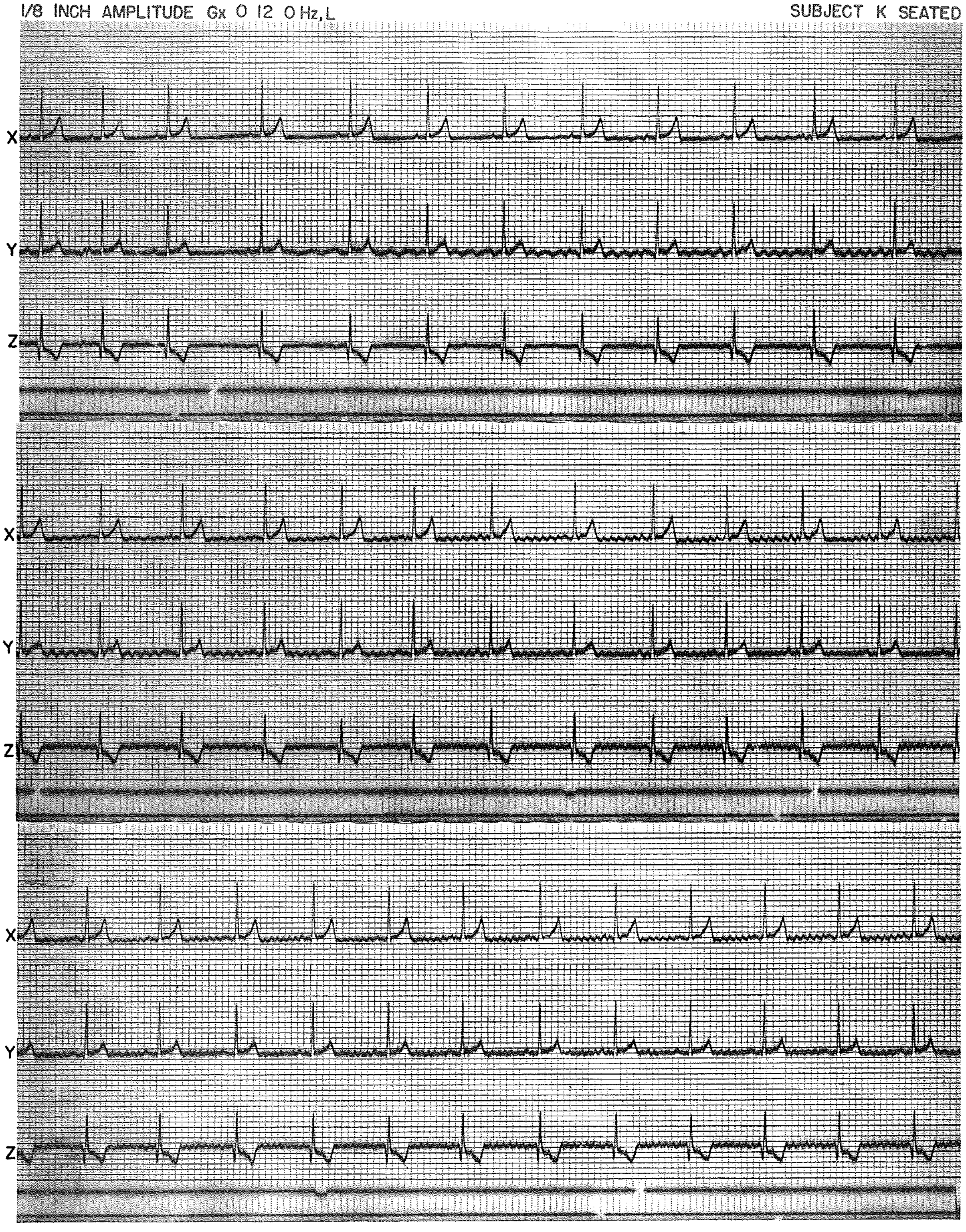



1/8 INCH AMPLITUDE Gx 12 Hz
SUBJECT K SEATED
X
Y
Z


12 Hz Gx
100/65
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

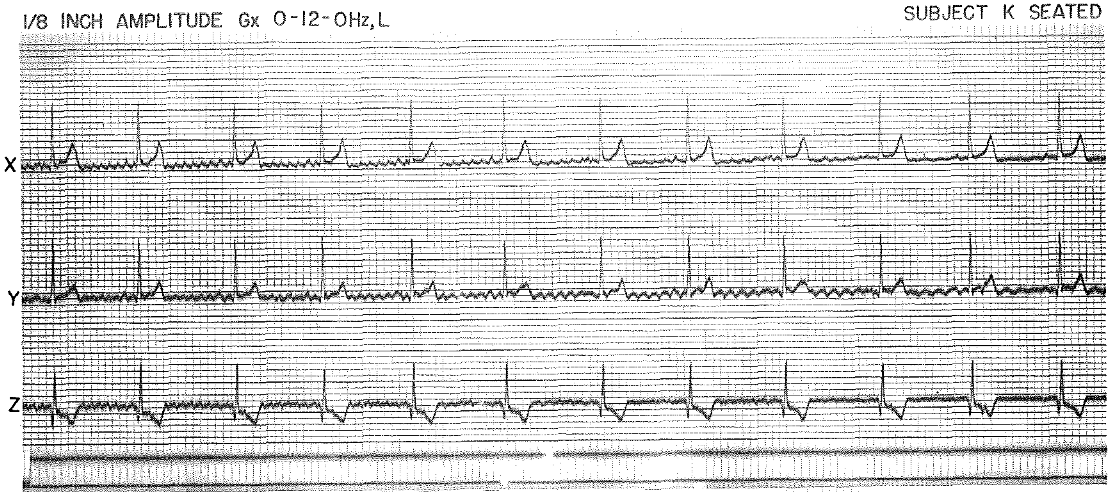

H
F
S

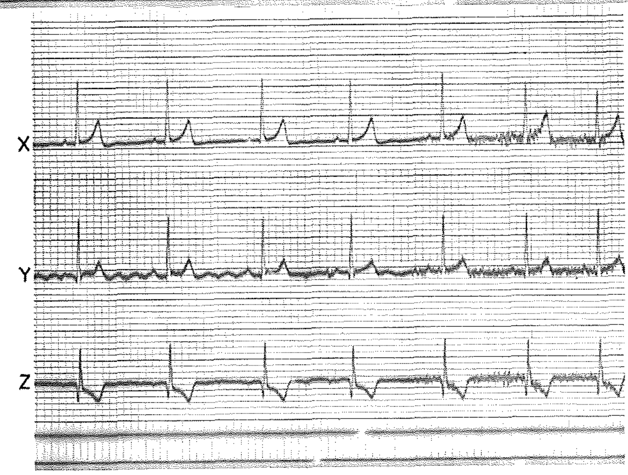
1/8 INCH AMPLITUDE Gx 0 12 0 Hz, L SUBJECT K SEATED



1/8 INCH AMPLITUDE Gx 0-12-0Hz, L

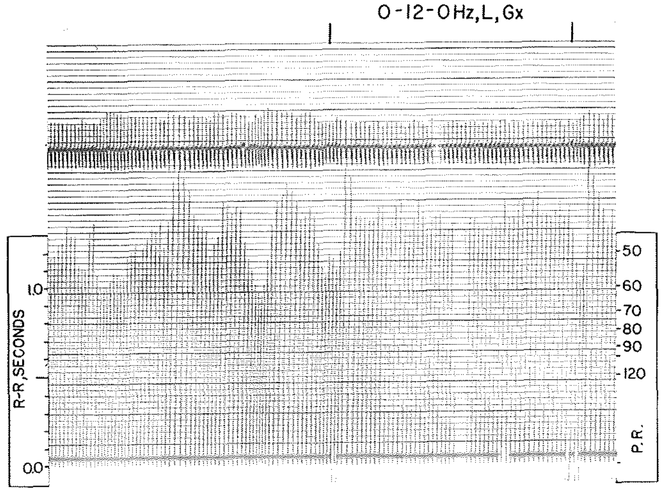
SUBJECT K SEATED

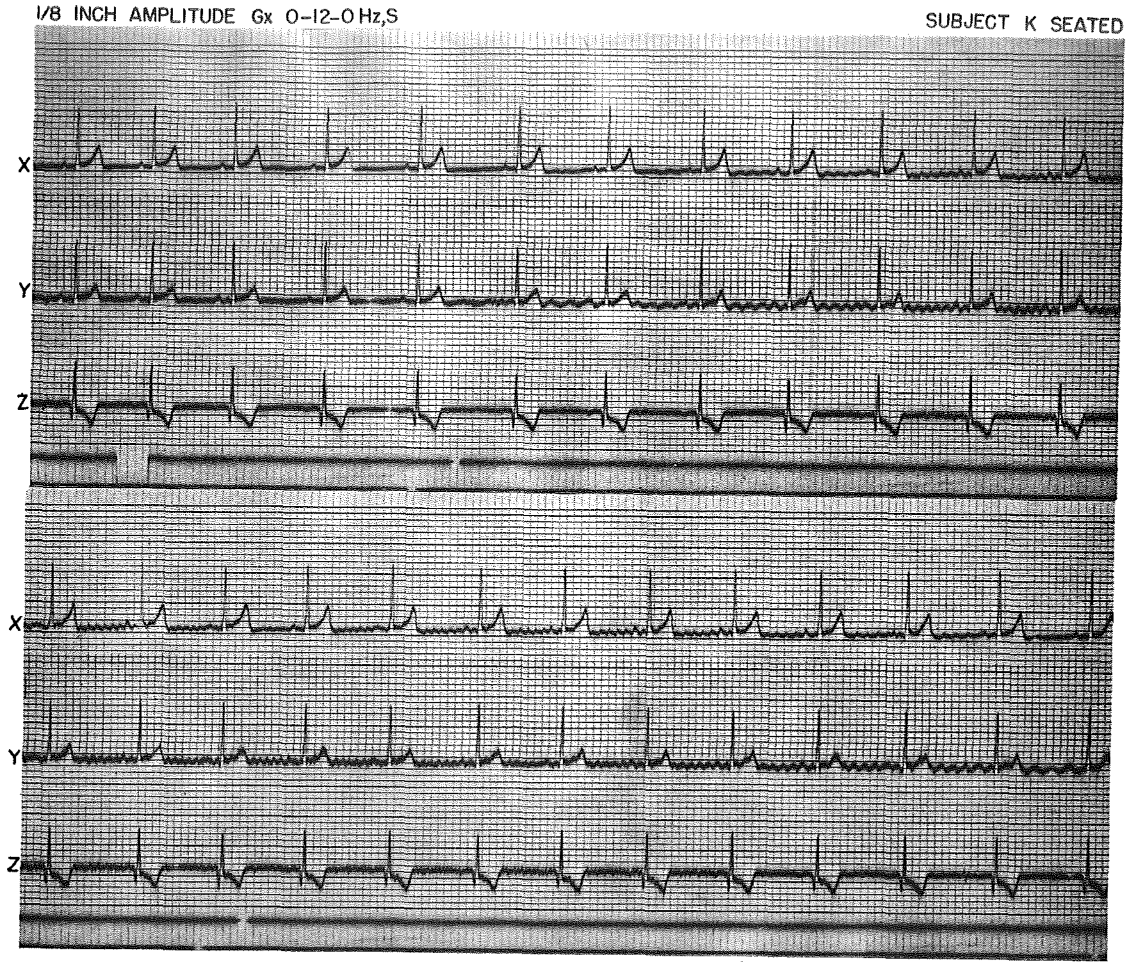


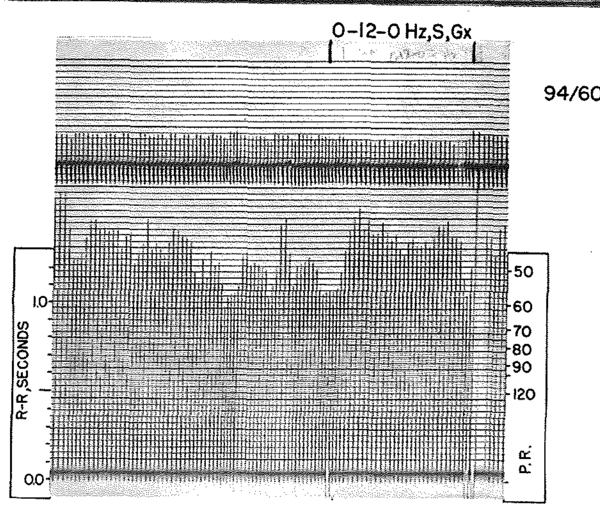


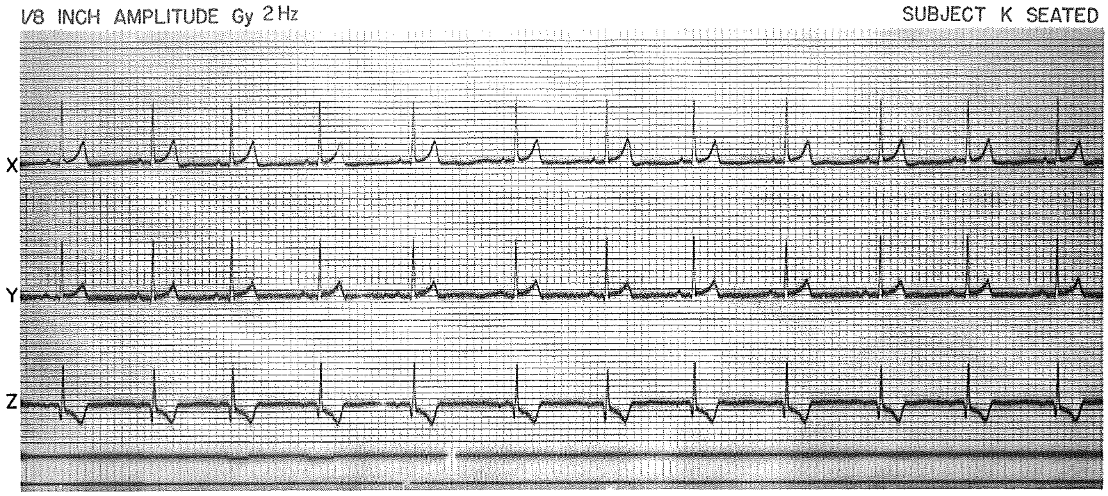
0-12-0 Hz, L, Gx

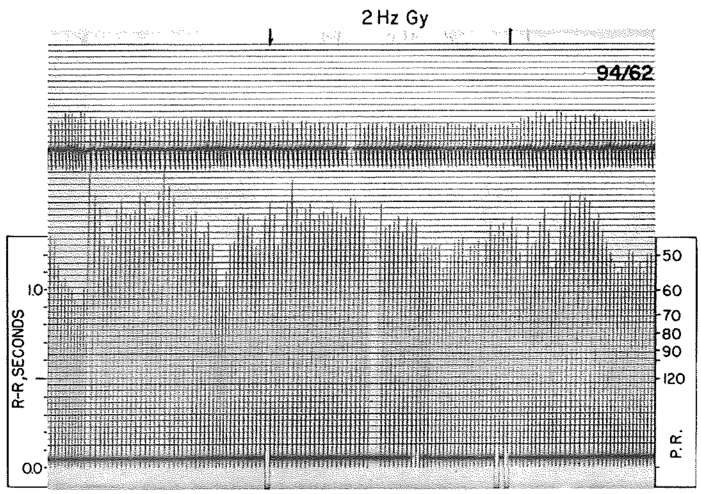
100/58

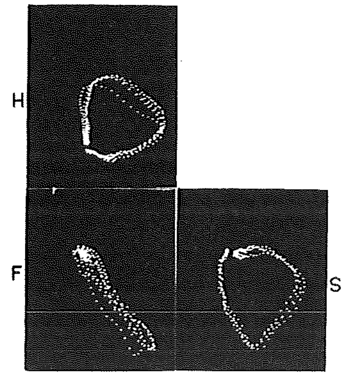


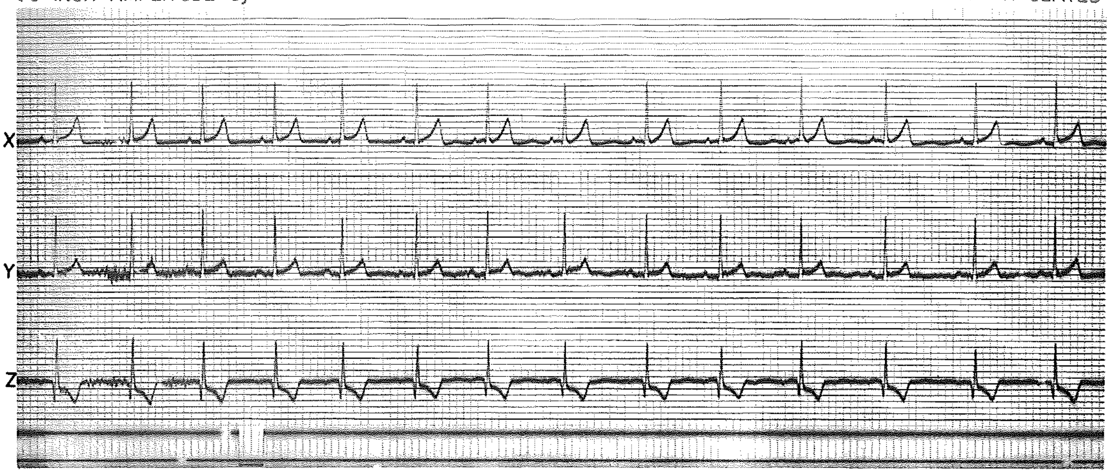

1/8 INCH AMPLITUDE Gx 0-12-0 Hz,S
SUBJECT K SEATED
X
Y
Z
X
Y
Z


0-12-0 Hz,S,Gx
94/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

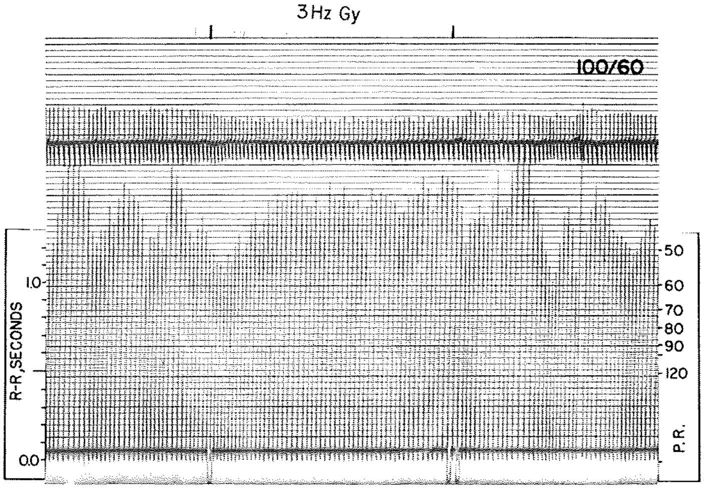

1/8 INCH AMPLITUDE Gy 2 Hz
SUBJECT K SEATED
X
Y
Z


2 Hz Gy
94/62
R-R, SECONDS
1.0
0.0
P.R.
50
60
70
80
90
120

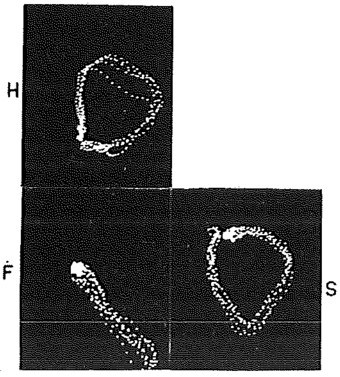

H
F
S



1/8 INCH AMPLITUDE Gy 3Hz
SUBJECT K SEATED
X
Y
Z



3Hz Gy
100/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

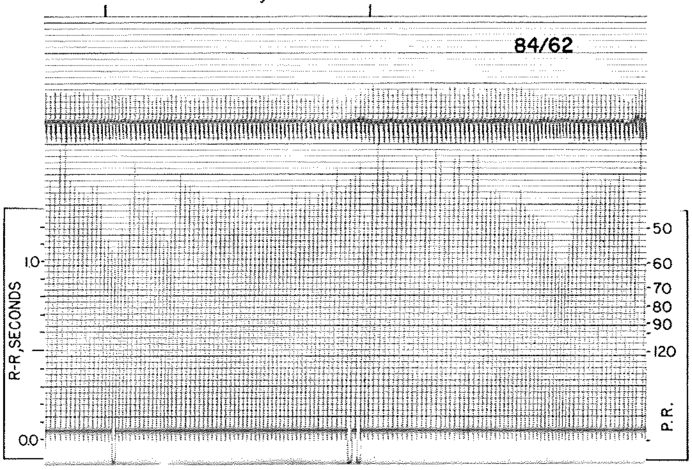


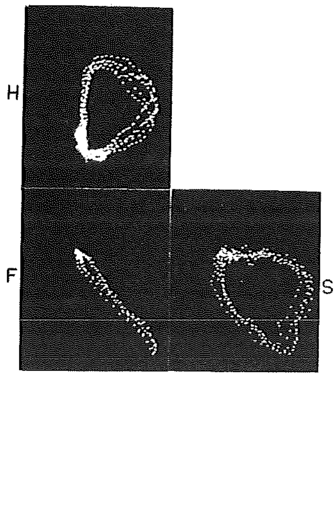
H
F
S

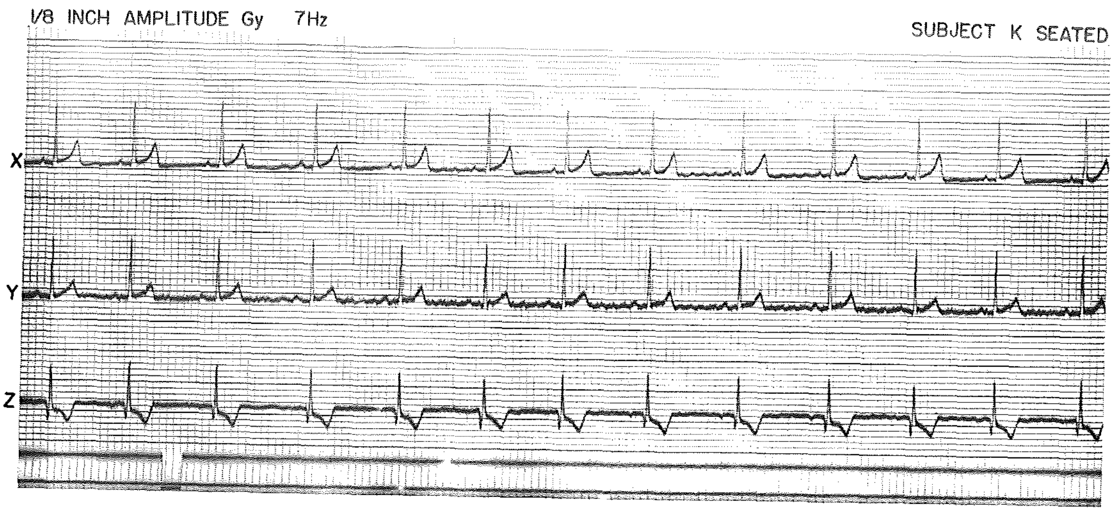
1/8 INCH AMPLITUDE Gy 5Hz

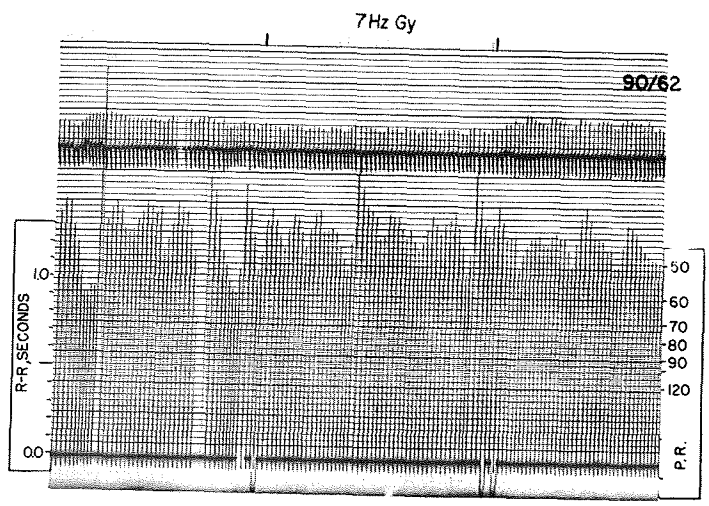
SUBJECT K SEATED

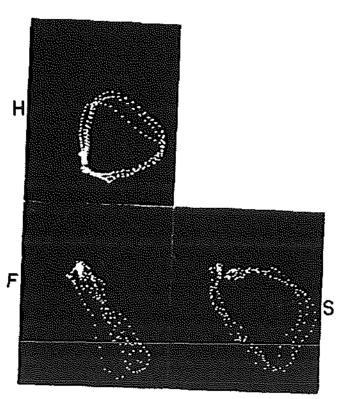
5 Hz Gy

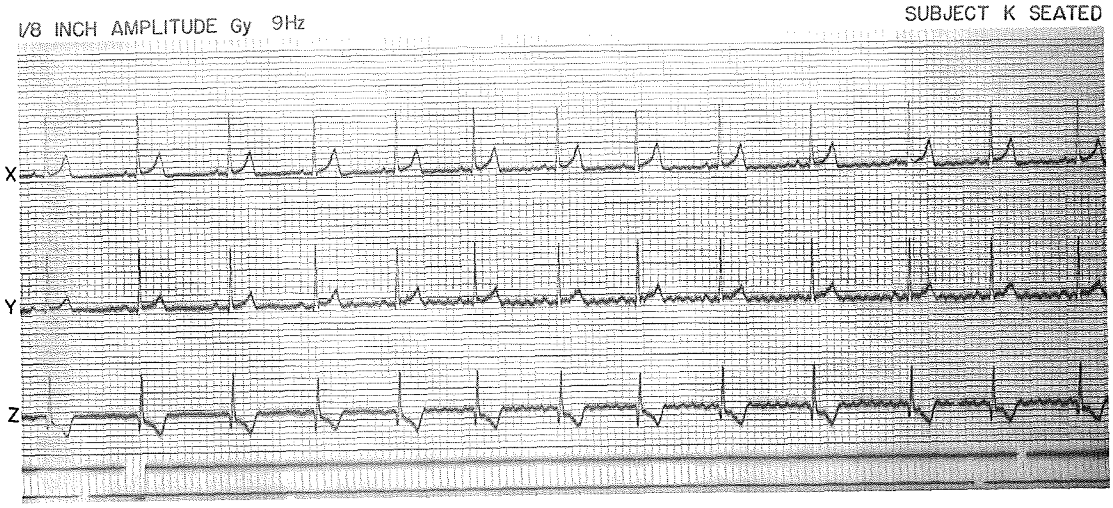


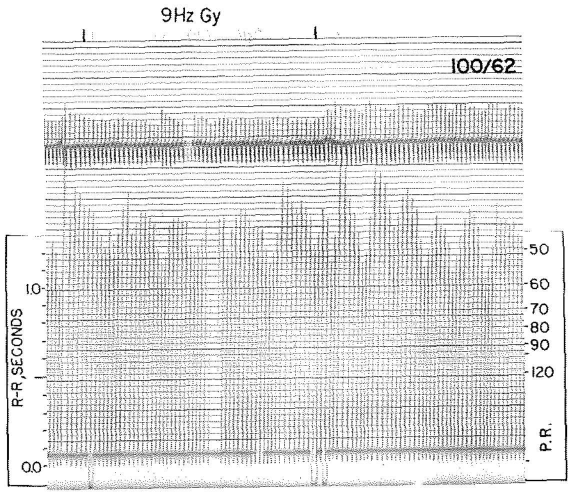


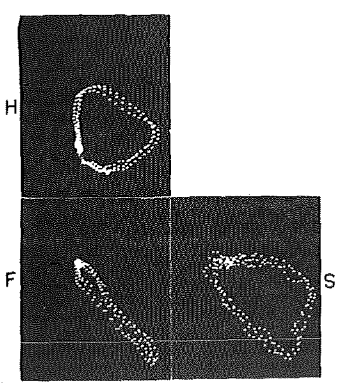

1/8 INCH AMPLITUDE Gy 7Hz
SUBJECT K SEATED
X
Y
Z

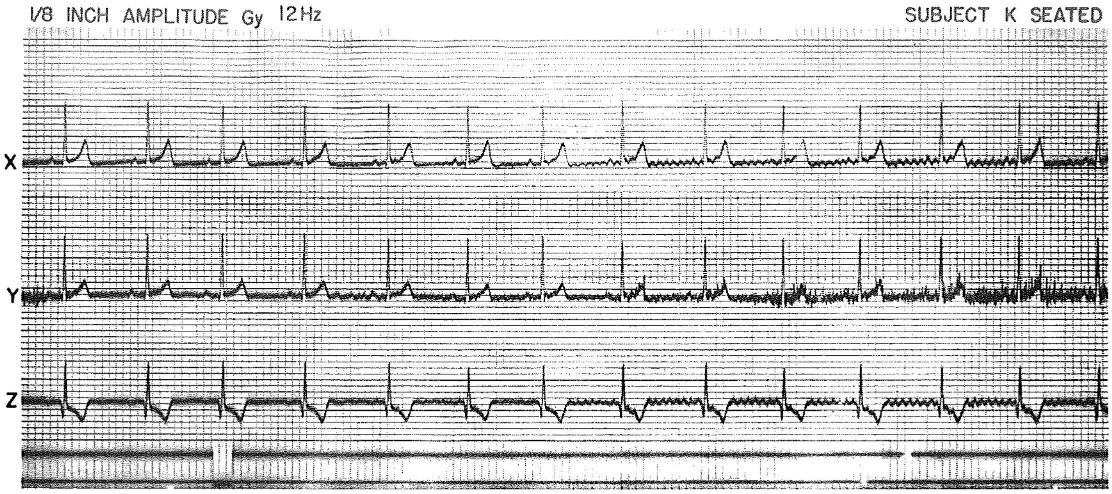

7Hz Gy
90/62
1.0
R-R, SECONDS
0.0
50
60
70
80
90
120
P.R.

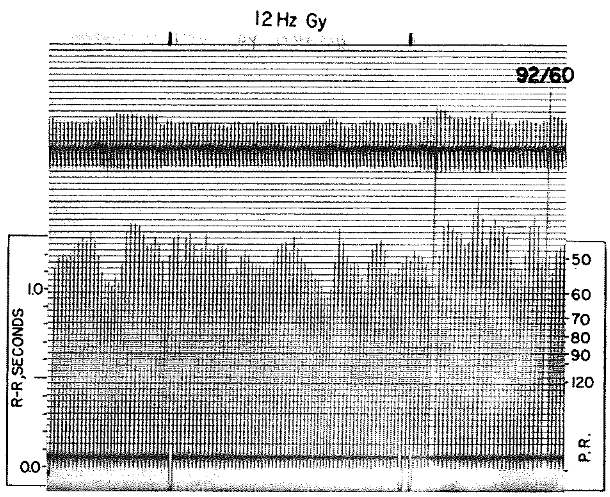

H
F
S

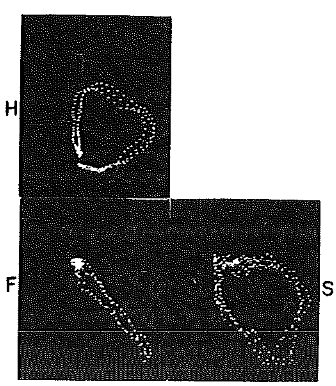

1/8 INCH AMPLITUDE Gy 9Hz
SUBJECT K SEATED
X
Y
Z


9Hz Gy
100/62
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.


H
F
S

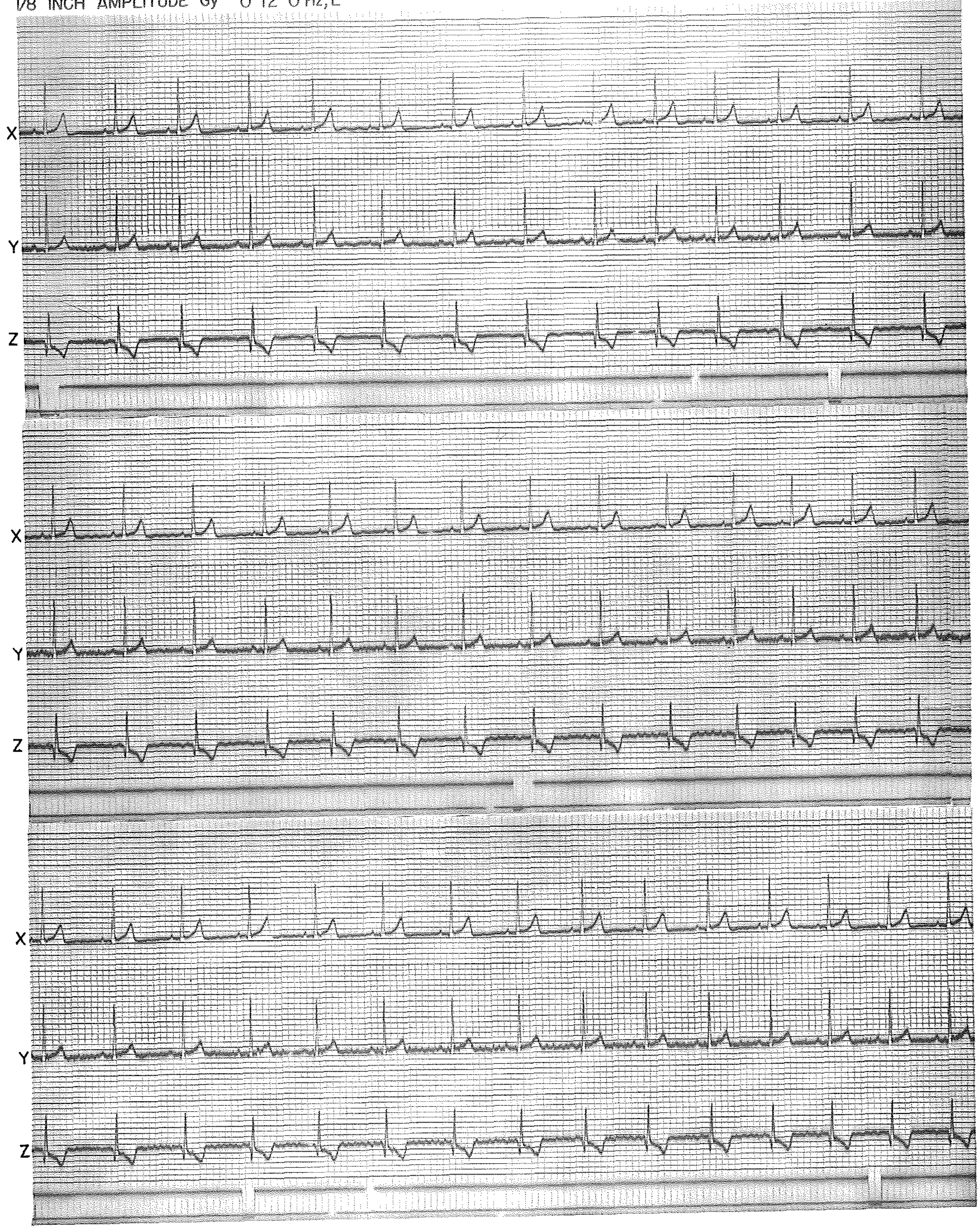

1/8 INCH AMPLITUDE Gy 12 Hz
SUBJECT K SEATED
X
Y
Z


12 Hz Gy
92/60
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.


H
F
S

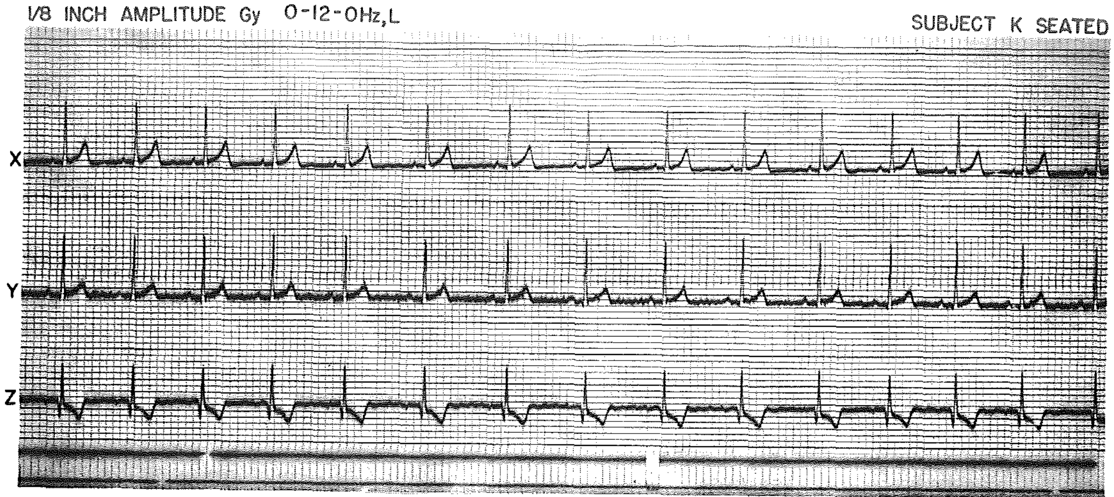
1/8 INCH AMPLITUDE Gy O 12 O Hz, L

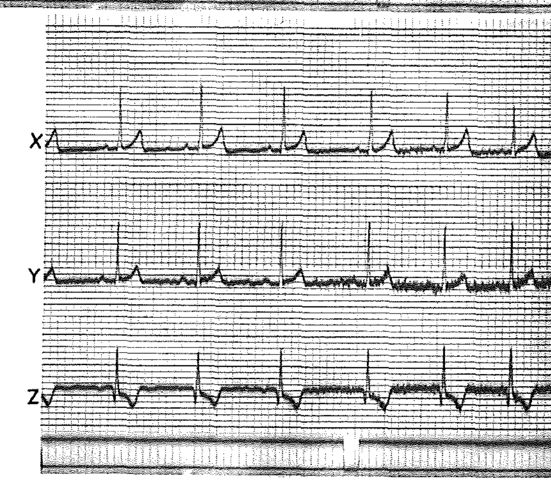
SUBJECT K SEATED



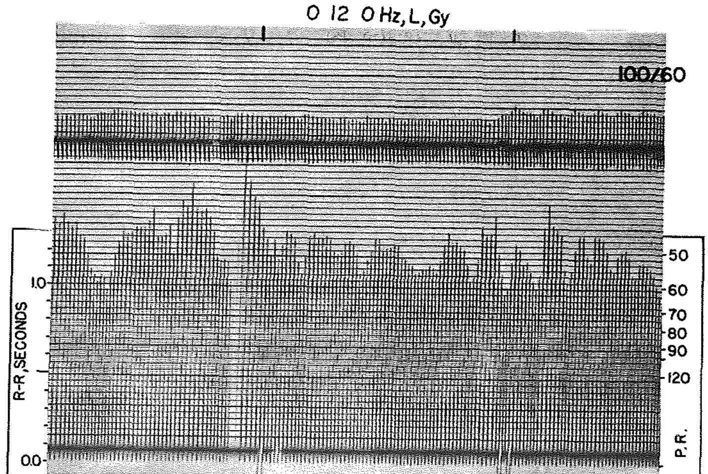
1/8 INCH AMPLITUDE Gy 0-12-0Hz,L

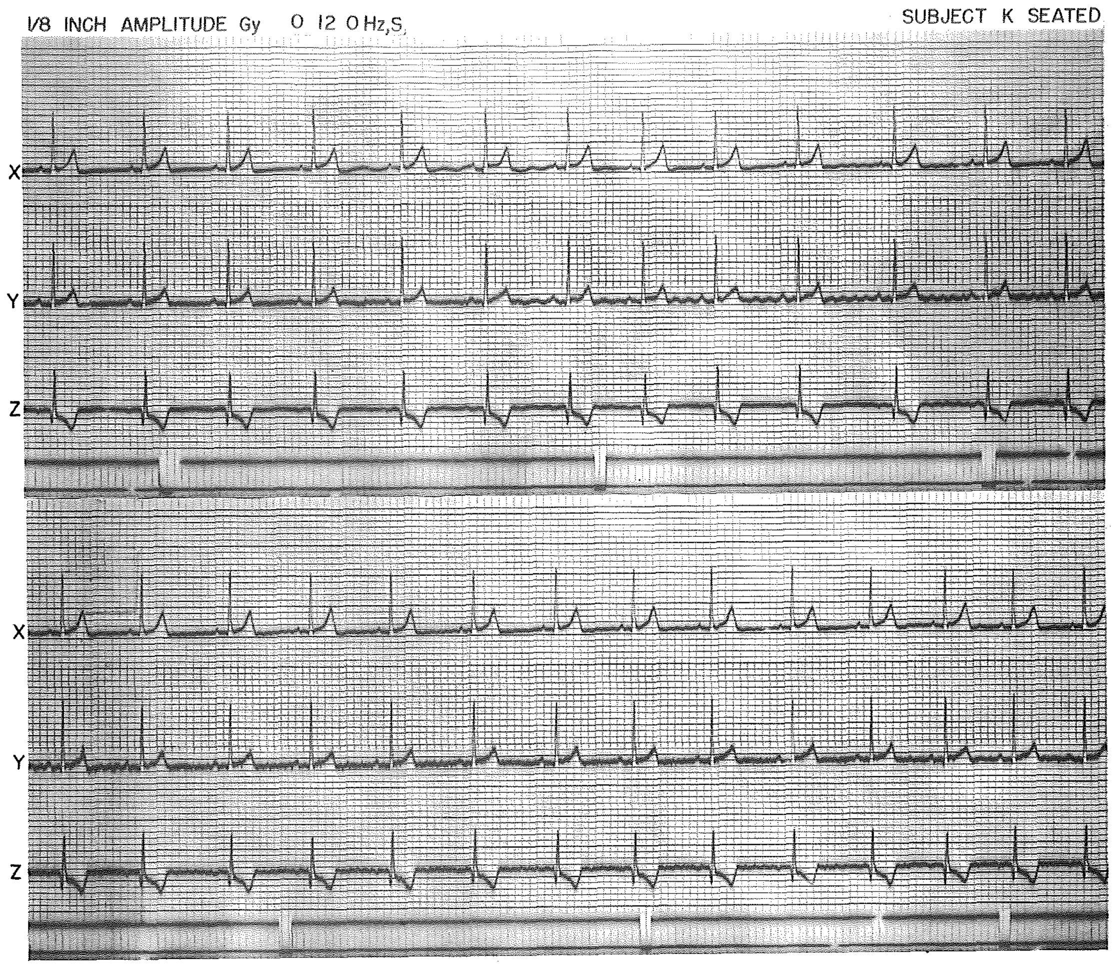
SUBJECT K SEATED

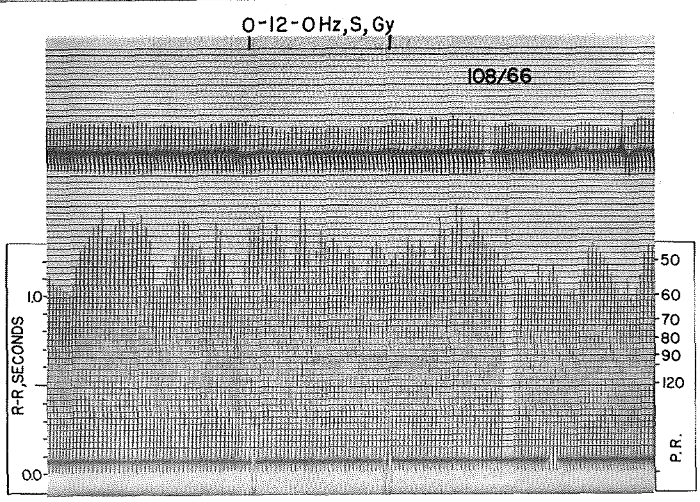


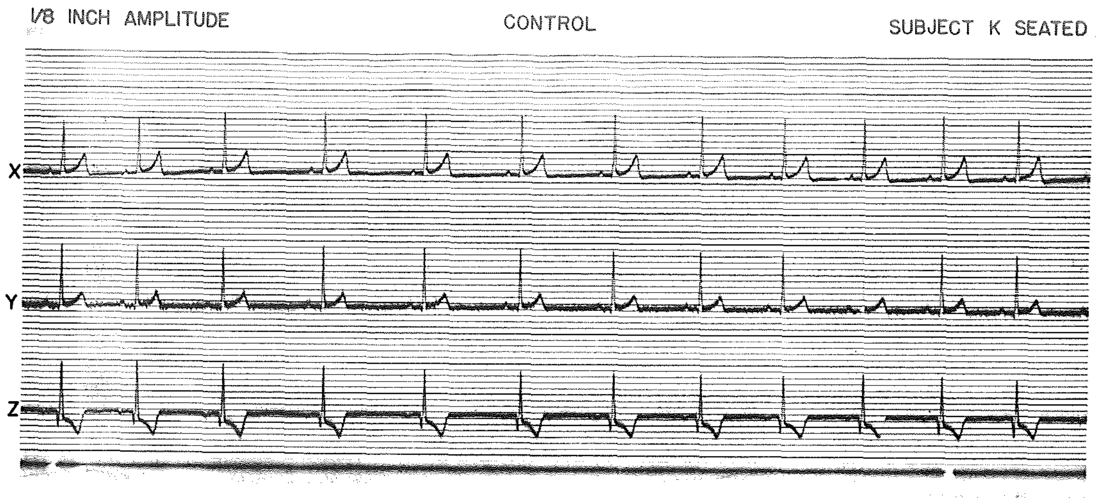


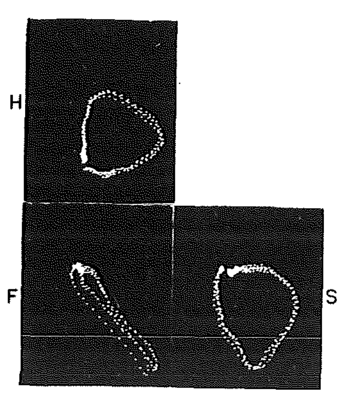
0 12 0 Hz, L, Gy

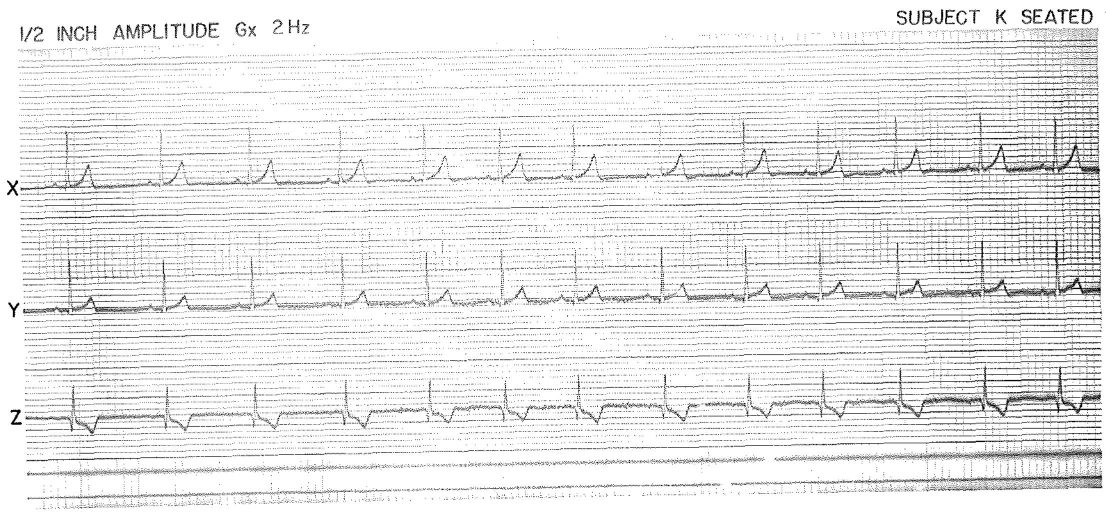


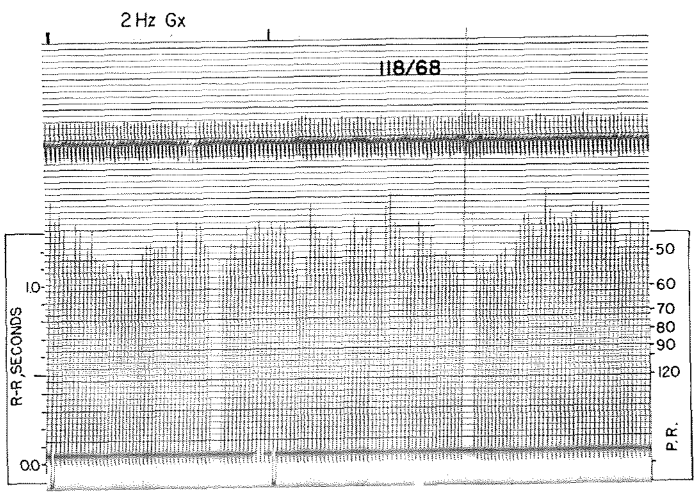

1/8 INCH AMPLITUDE Gy 0 12 0 Hz,S,
SUBJECT K SEATED
X
Y
Z
X
Y
Z

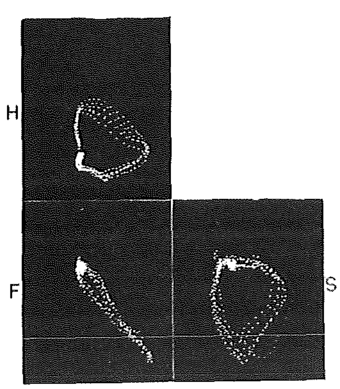

0-12-0 Hz,S, Gy
108/66
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

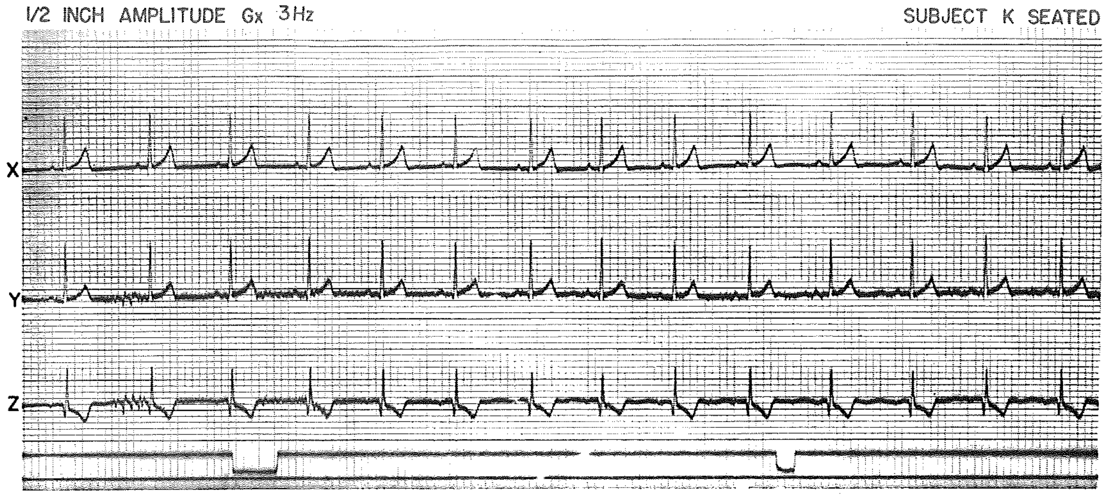

1/8 INCH AMPLITUDE
CONTROL
SUBJECT K SEATED
X
Y
Z

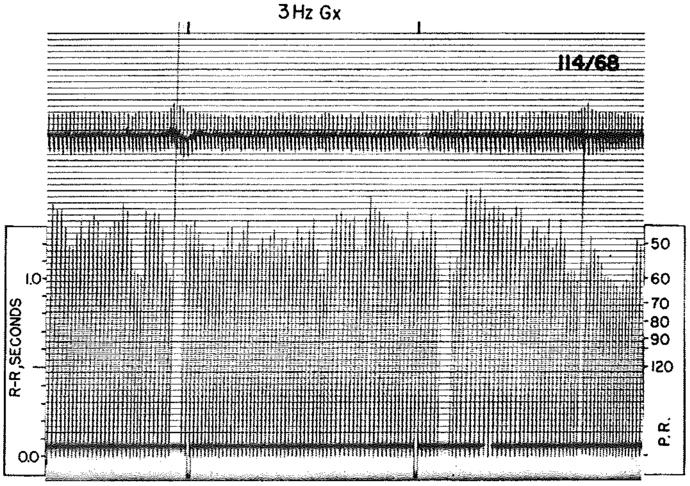

H
F
S

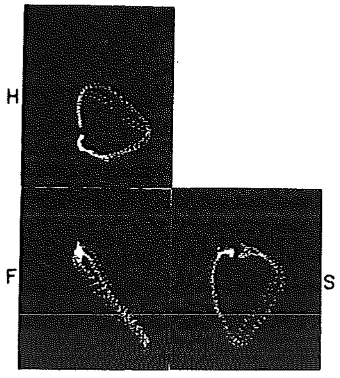

1/2 INCH AMPLITUDE Gx 2 Hz
SUBJECT K SEATED
X
Y
Z

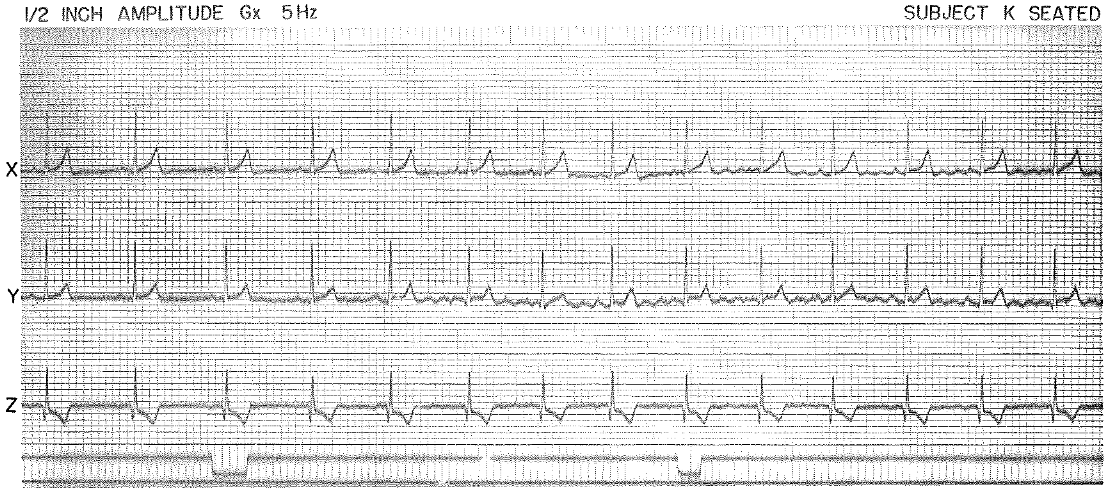

2 Hz Gx
118/68
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

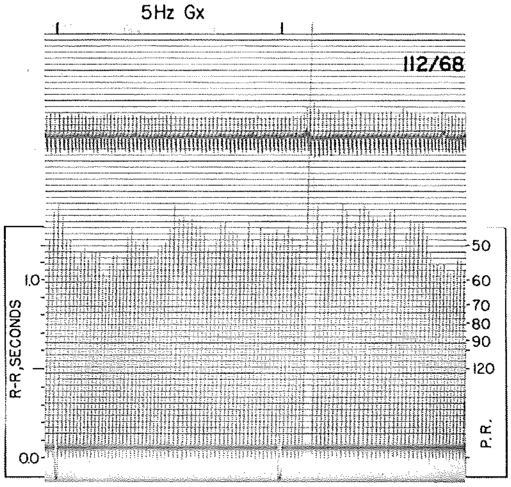

H
F
S

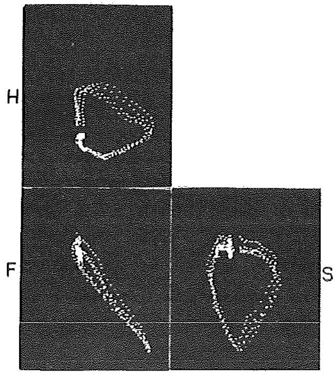

1/2 INCH AMPLITUDE Gx 3Hz
SUBJECT K SEATED
X
Y
Z

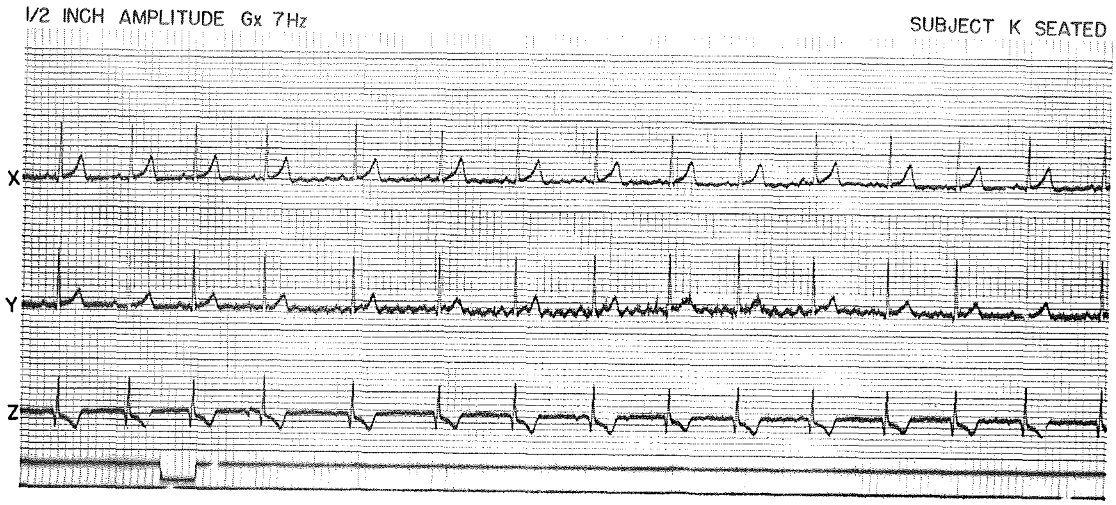

3 Hz Gx
114/68
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

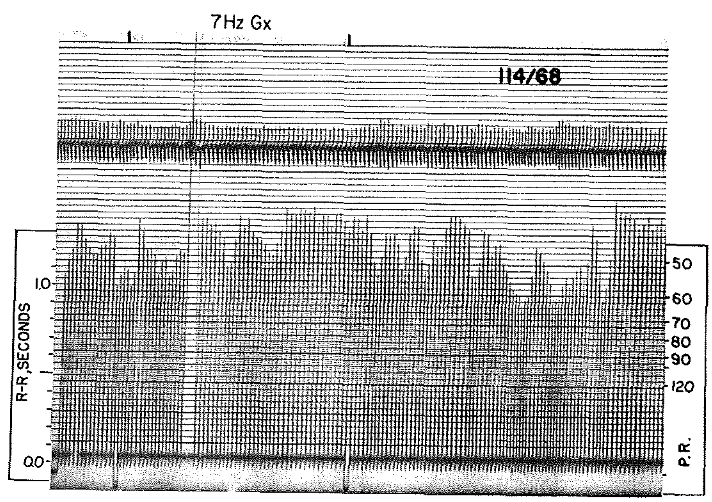

H
F
S

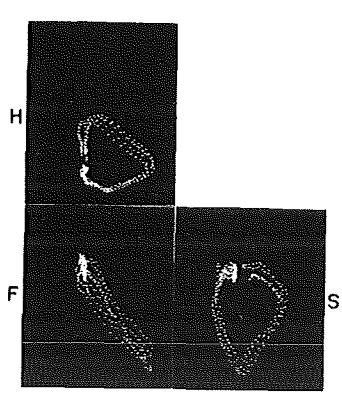

1/2 INCH AMPLITUDE Gx 5 Hz
SUBJECT K SEATED
X
Y
Z


5Hz Gx
112/68
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

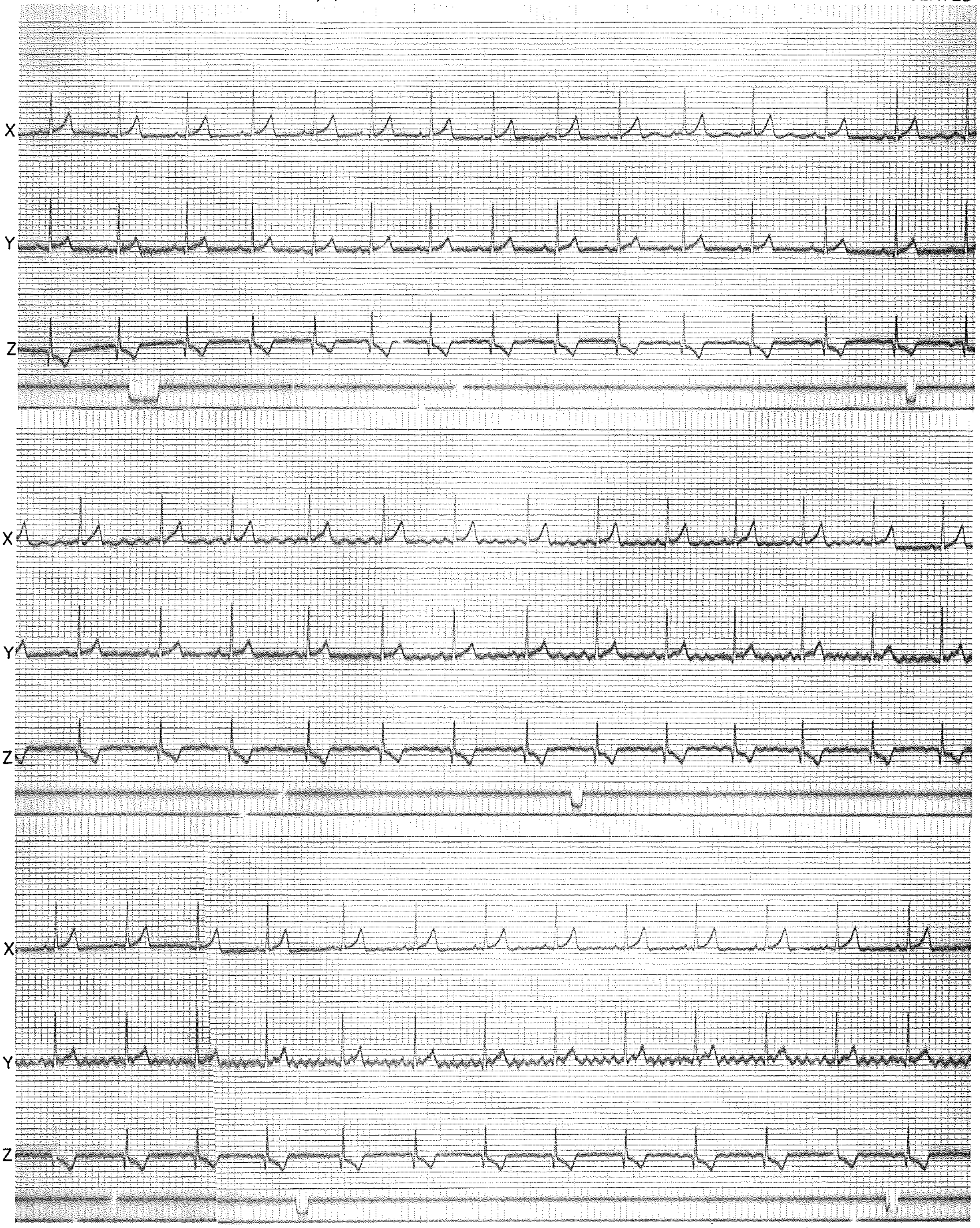

H
F
S

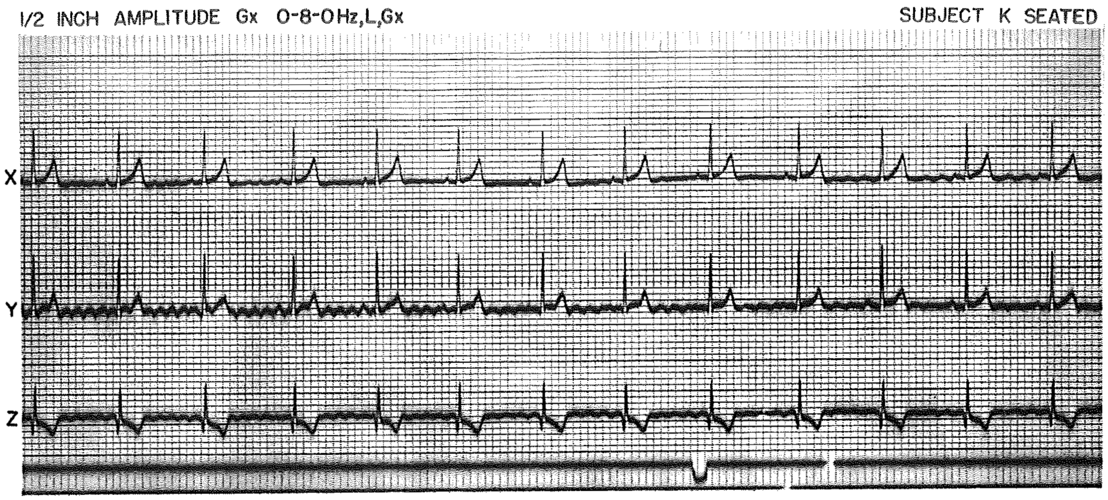

1/2 INCH AMPLITUDE Gx 7Hz
SUBJECT K SEATED
X
Y
Z

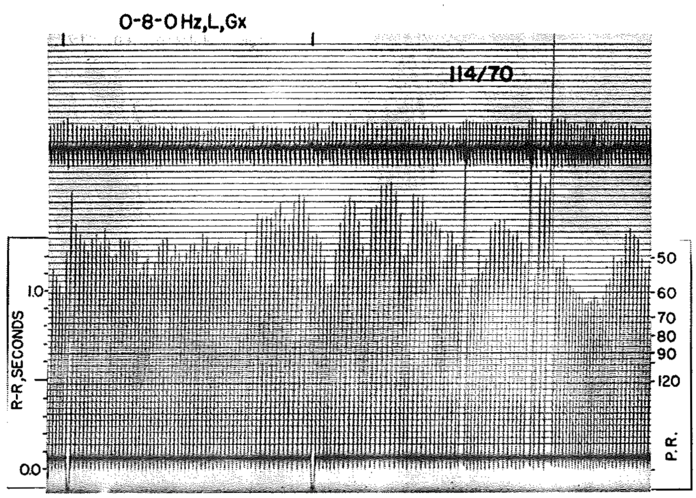

7Hz Gx
114/68
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

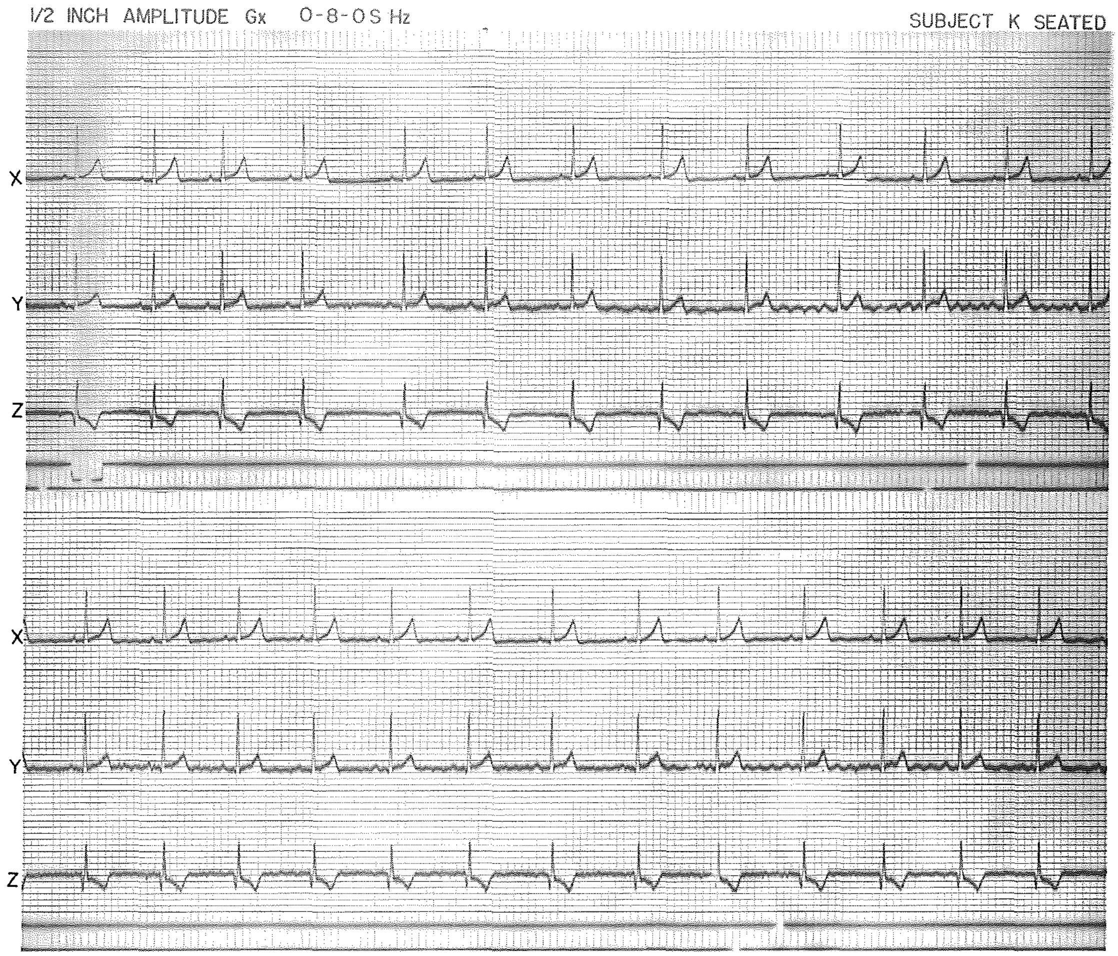

H
F
S

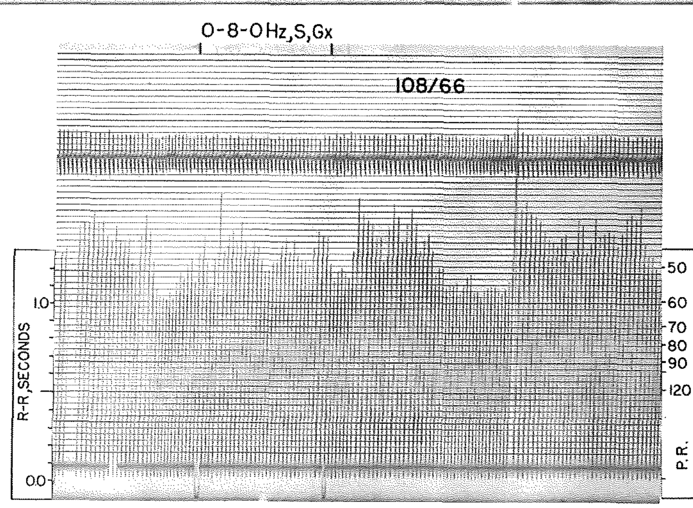
1/2 INCH AMPLITUDE Gx 0-8-0Hz,L,Gx SUBJECT K SEATED

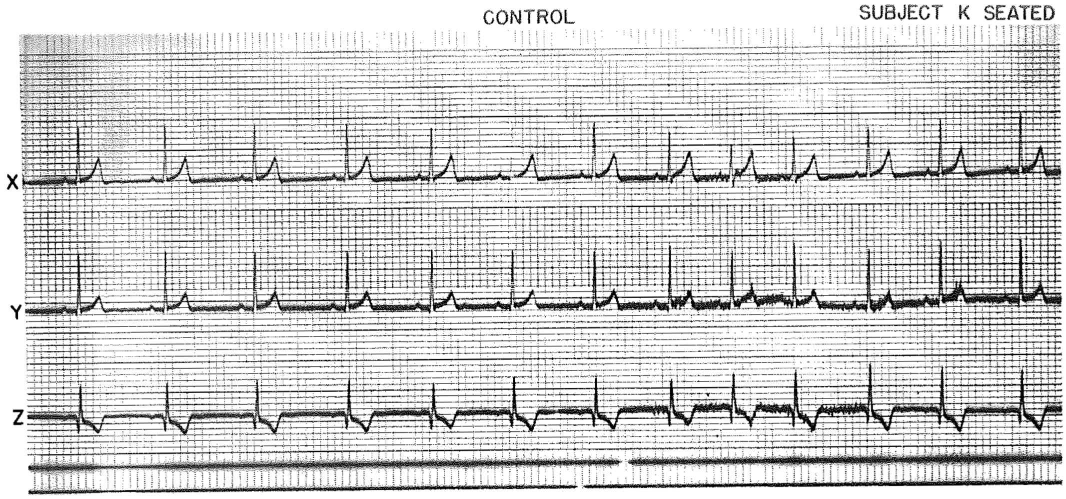


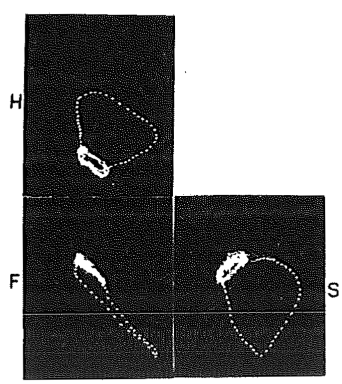

1/2 INCH AMPLITUDE Gx 0-8-0 Hz,L,Gx
SUBJECT K SEATED
X
Y
Z

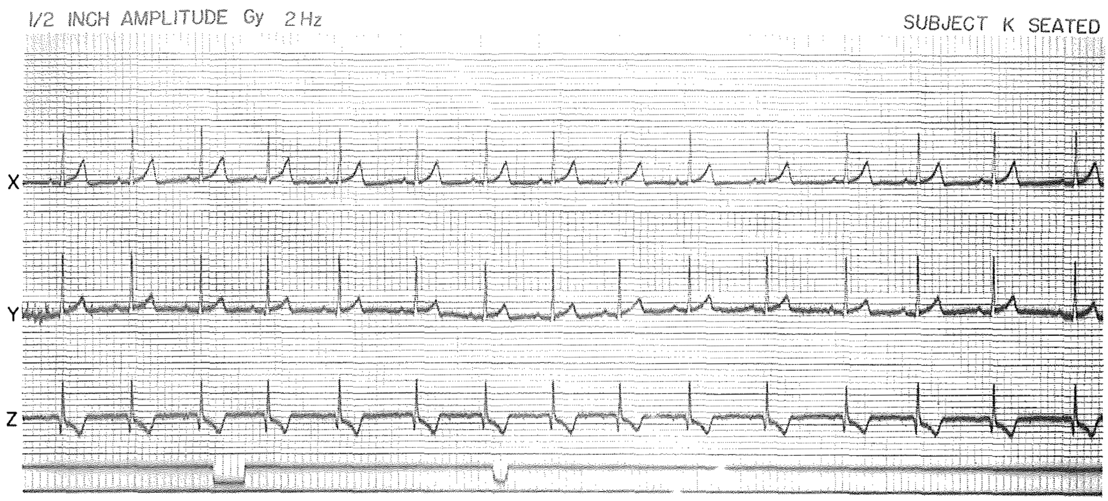

0-8-0 Hz,L,Gx
114/70
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

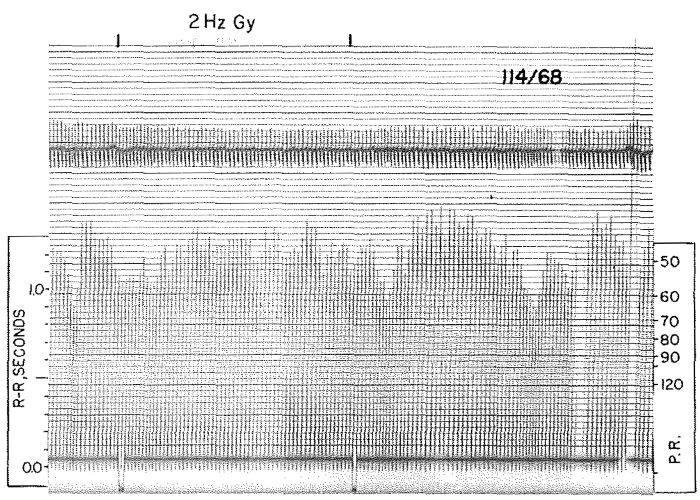

1/2 INCH AMPLITUDE Gx 0-8-0S Hz
SUBJECT K SEATED
X
Y
Z
X
Y
Z

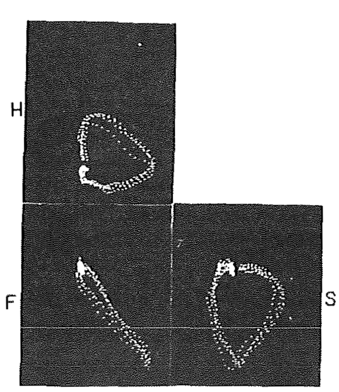

0-8-0Hz,S,Gx
108/66
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.


CONTROL
SUBJECT K SEATED
X
Y
Z

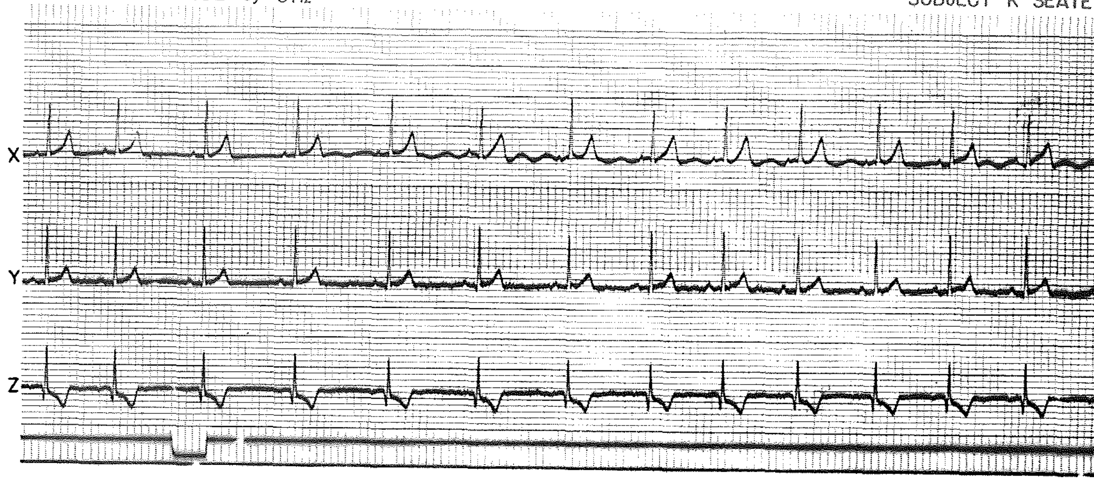

H
F
S


1/2 INCH AMPLITUDE Gy 2 Hz
SUBJECT K SEATED
X
Y
Z

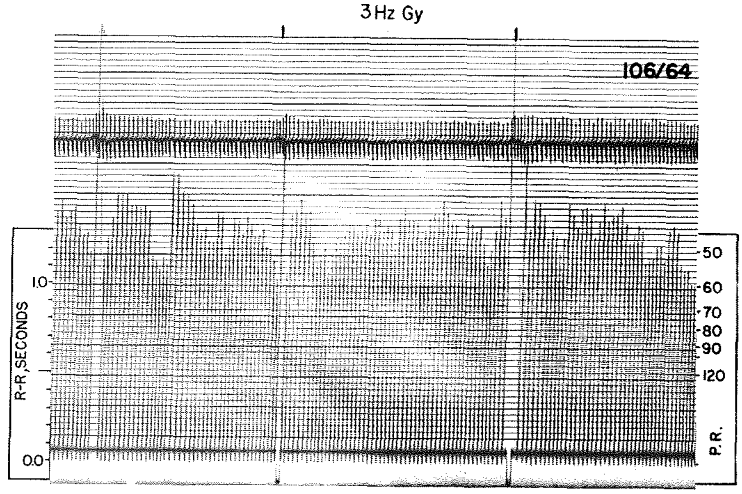

2 Hz Gy
114/68
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

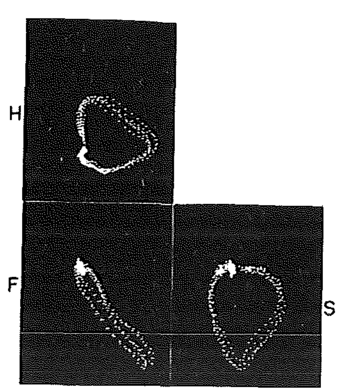

H
F
S

1/2 INCH AMPLITUDE Gy 3 Hz SUBJECT K SEATED

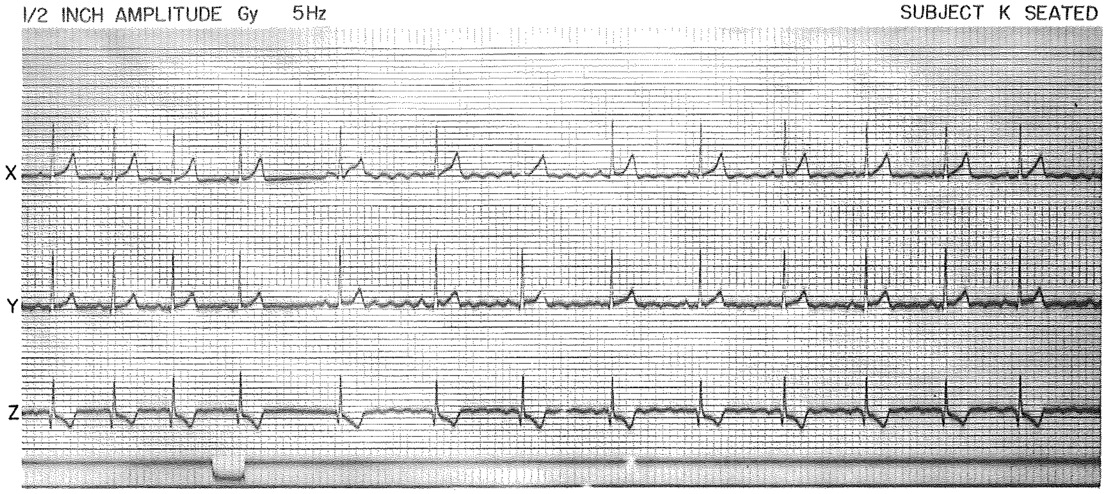


3 Hz Gy

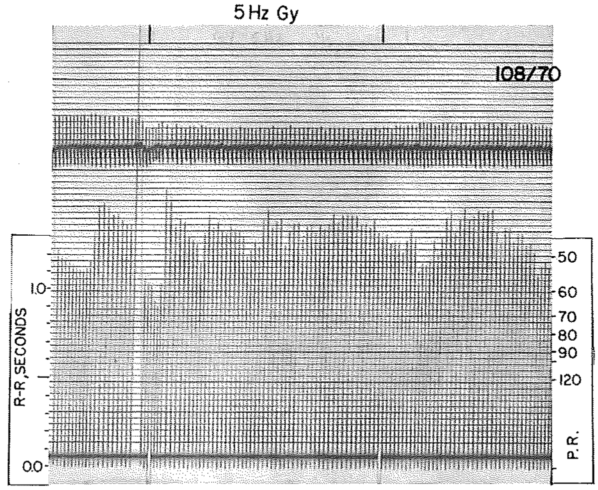


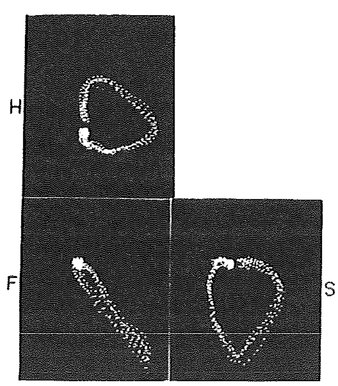


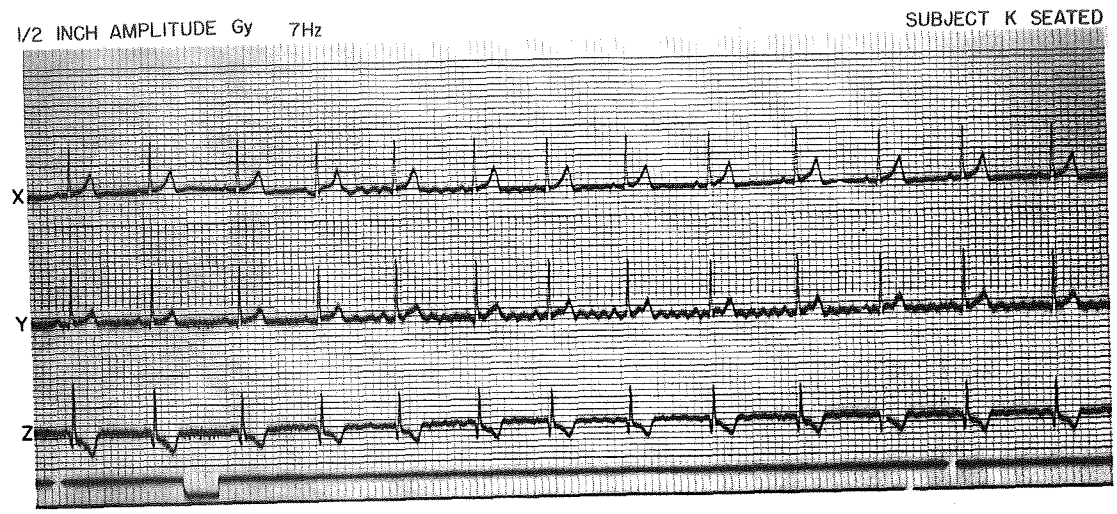
I/2 INCH AMPLITUDE Gy 5Hz SUBJECT K SEATED

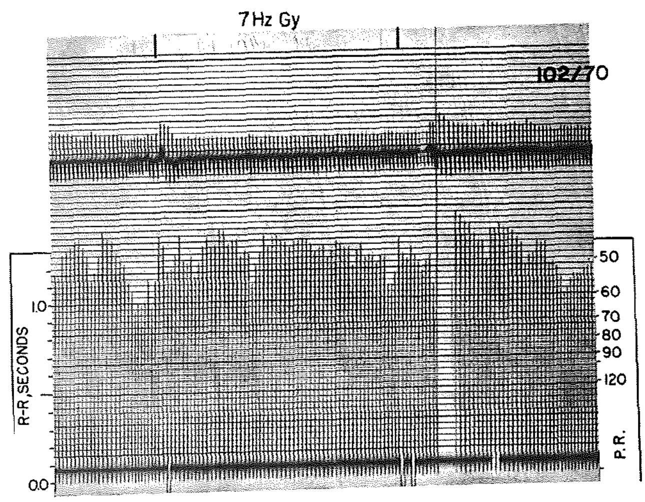


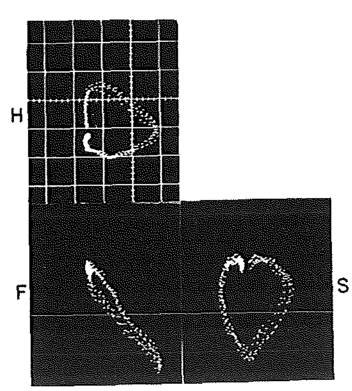
5 Hz Gy





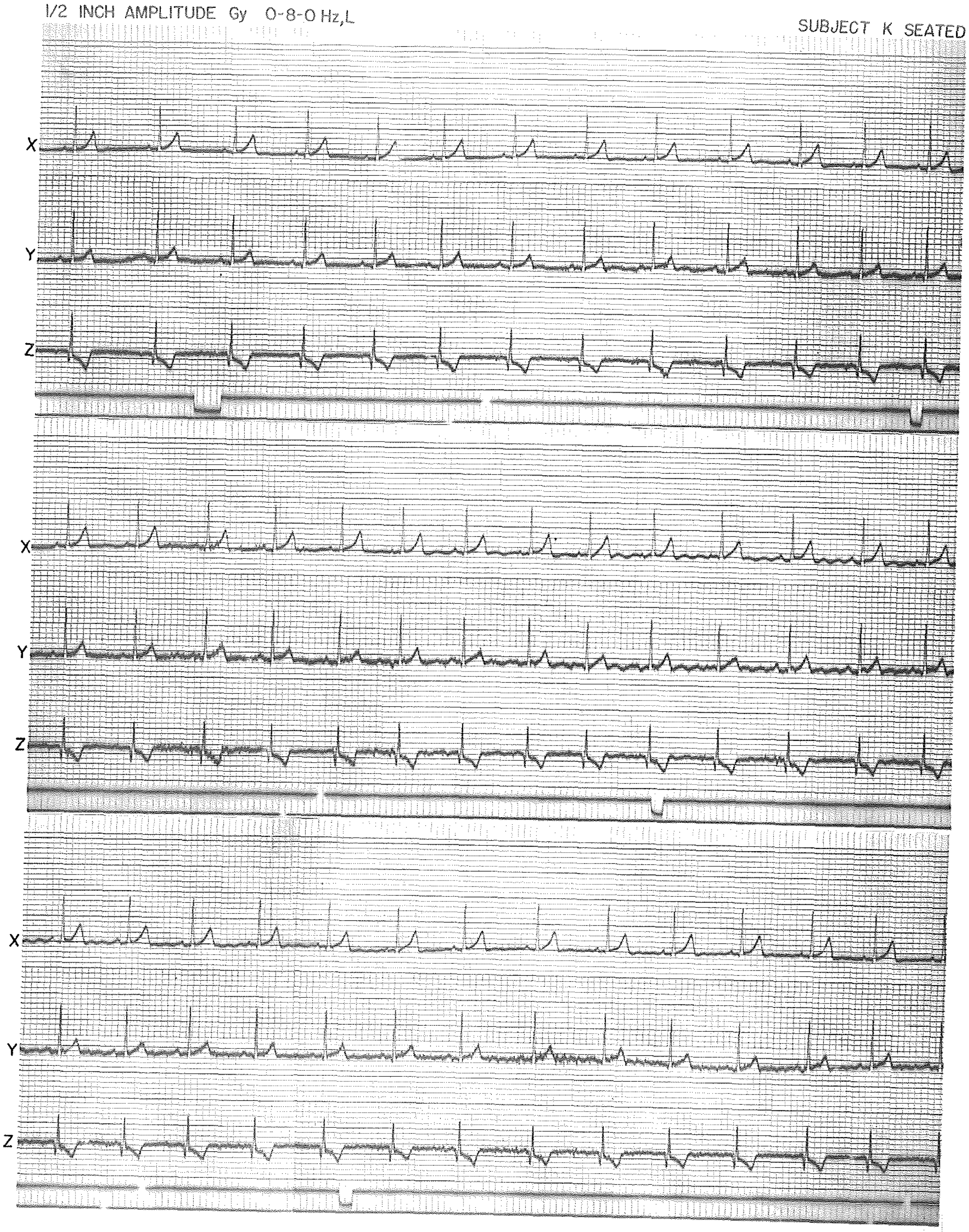

1/2 INCH AMPLITUDE Gy 7Hz
SUBJECT K SEATED
X
Y
Z

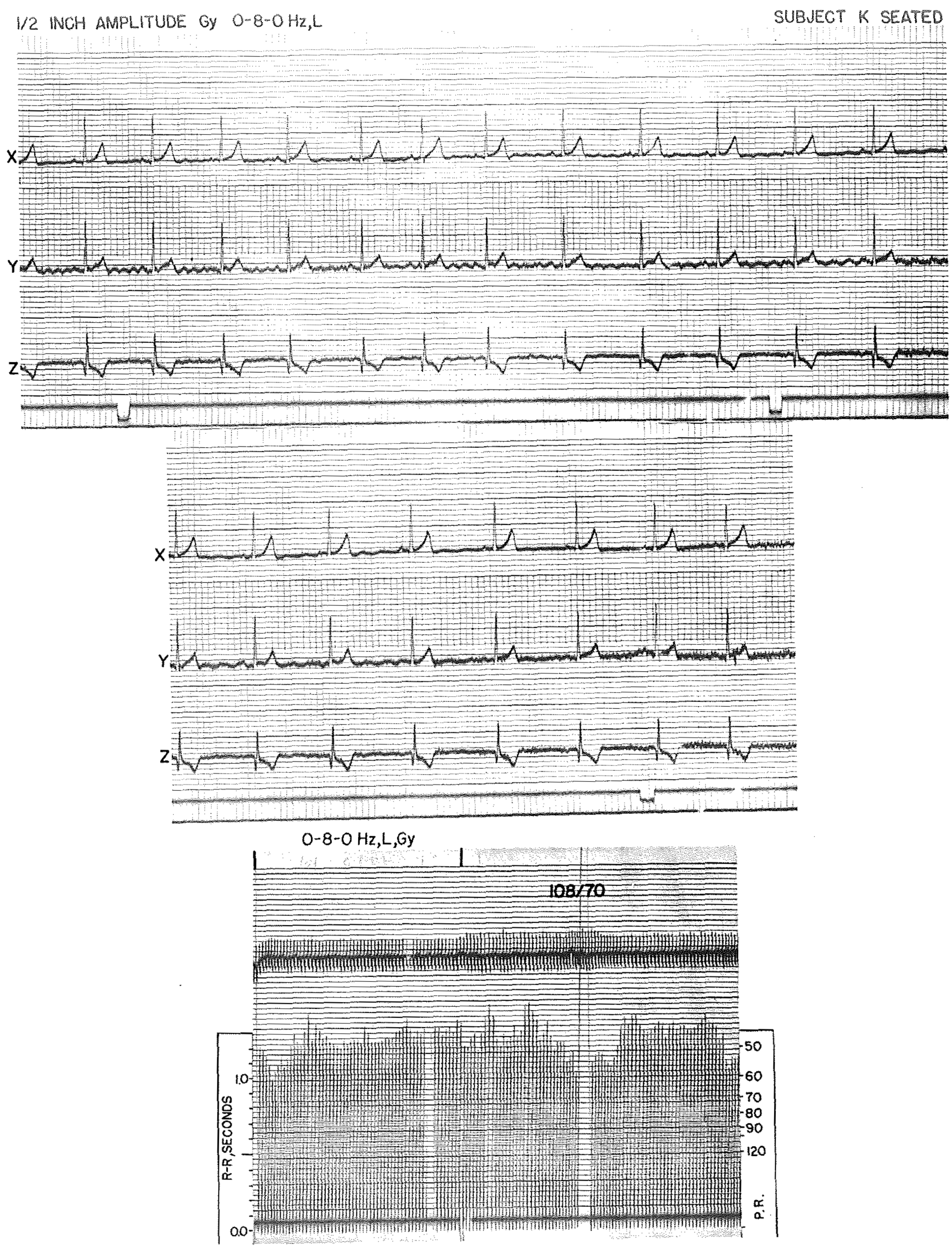

7 Hz Gy
102/70
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.


H
F
S

1/2 INCH AMPLITUDE Gy 0-8-0 Hz,L

SUBJECT K SEATED

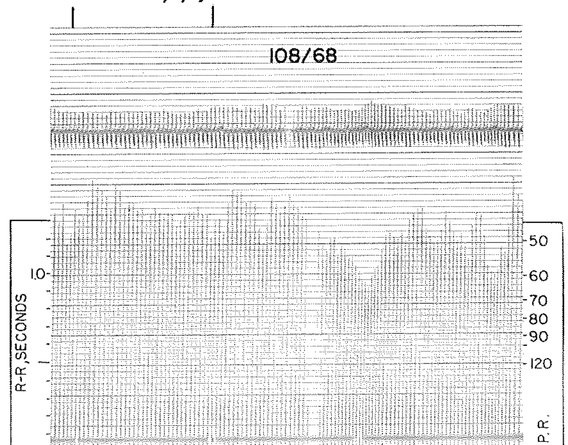


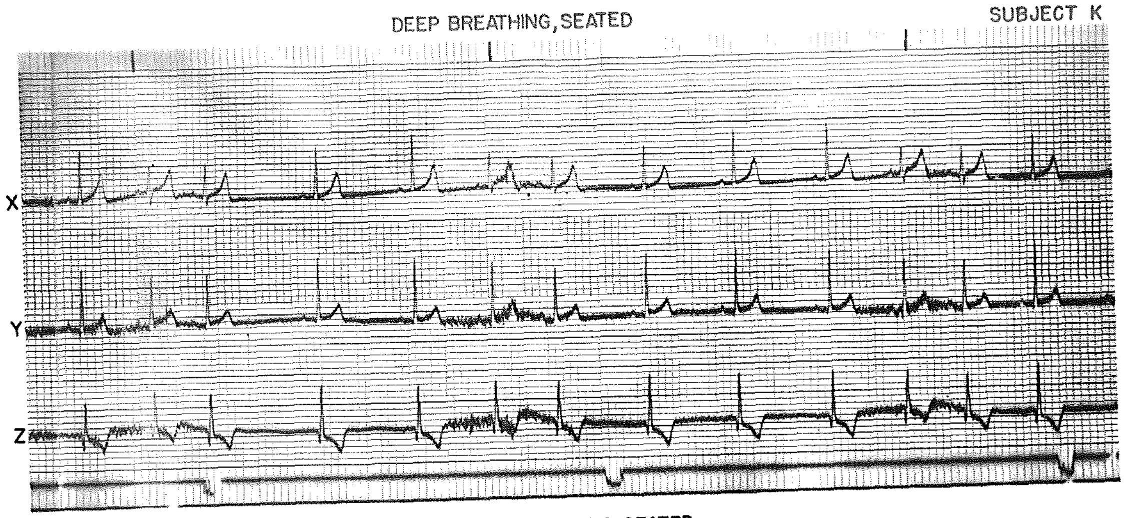


1/2 INCH AMPLITUDE Gy 0-8-0 Hz,L
SUBJECT K SEATED
X
Y
Z
X
Y
Z
0-8-0 Hz,L,Gy
108/70
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.

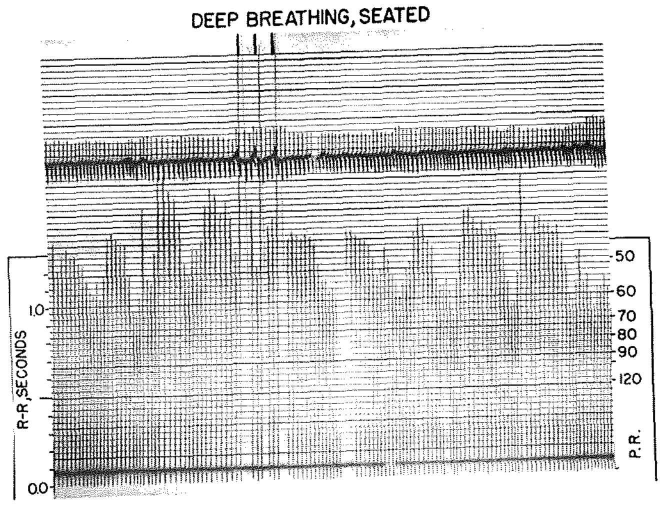
1/2 INCH AMPLITUDE Gy 0-8-0 Hz SUBJECT K SEATED

0-8-0 Hz,S,Gy

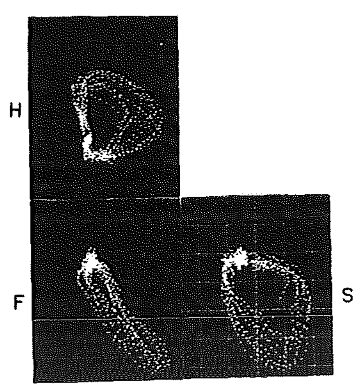




DEEP BREATHING, SEATED
SUBJECT K
X
Y
Z



DEEP BREATHING, SEATED
R-R, SECONDS
1.0
0.0
50
60
70
80
90
120
P.R.



H
F
S

II. ACUTE, TRANSIENT CARDIOVASCULAR EFFECTS OF VIBRATION IN DOGS*

Robert L. Hamlin, D.V.M., Ph.D.**+; John H. Dines, M.D.***; Lester B. Roberts, B.S.***; and Edward J. Whitehead***

A. INTRODUCTION

Transient cardiovascular changes observed by us in healthy man and in anesthetized but otherwise unmedicated dogs when either is subjected to moderately severe vibration do not immediately suggest the vibration response mechanisms involved. Since vibration can conceivably elicit responses from a variety of centers and since there may be intermediate physical response mechanisms involved, a better understanding is needed if we hope to be able to evaluate vibration on any basis other than empirical. This study of cardiovascular responses of the anesthetized dog to vibration before and after receiving alpha and beta adrenergic and vagal blockade was designed to help indicate the cardiovascular response systems involved.

B. MATERIALS AND METHODS

Thirty-two healthy dogs of equal sex distribution and weighing between 10 and 16 kg (mean weight 14.5 kg) were selected for this study. All were anesthetized intravenously, to plane iii of Stage 3, with either dolophine-chloralose-urethane or fentanyl-droperidol-pentobarbital. Dogs anesthetized with both regimens responded similarly to vibration. Since the latter is administered more easily and permits dogs to maintain their normal respiratory sinus arrhythmia with average ventricular rate below 80 beats per minute, fentanyl-droperidol-pentobarbital was used for the last 11 experiments. Dogs which, after being anesthetized, did not maintain this arrhythmia with average rate below 80 were dropped from the study.

Dogs were placed in dorsal-supine recumbency in form-fitting plaster casts which coupled them firmly but did not rigidly restrain them to a vibration table.

A special catheter-tip pressure transducer,[2] compensated to cancel

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out the effect of vibration, was placed in the left ventricle through a carotid artery.

A second catheter was placed in the descending thoracic aorta via the femoral artery.

These catheters furnished electrical signals for the simultaneous recording of left ventricular pressure (LVP), its derivative (dp/dt), high gain (LVP) for end-diastolic pressure measurement (EDP) and mean arterial pressure (AP).

Cardiac output (CO) estimations were obtained by the indicator-dilution method in which indocyanine green dye was injected into the right atrium and arterial blood was sampled continuously via another femoral artery. Stroke volume (SV) was calculated by dividing (CO) by heart rate (HR) obtained from lead II electrocardiogram. Systemic vascular resistance (SVR) was calculated by dividing AP by CO.

Recordings were made before and after 15 seconds of vibration from dogs: (1) unmedicated (except for anesthesia), (2) after beta adrenergic block with propranolol (0.5 mg/kg), (3) after vagal block either by atropine (0.1 mg/kg) or bilateral cervical vagotomy, and (4) after alpha adrenergic block with phenoxybenzamine (15 mg/kg).

The above sequence produced sequential blockade in the order given from none to total denervation.

The dogs were vibrated at 1/2-inch total displacement and 7 Hz in the cranio-caudal axis.

C. RESULTS

Alterations in parameters measured reached their peak within 10 to 20 seconds after onset of vibration and returned to or near control values after 45 seconds of vibration. They remained at control values for the 15 minutes between periods of vibration. Consistent responses in all untreated dogs were (1) increases in HR, CO, dp/dt_{max}; (2) decreases in AP, svr; and (3) no change in EDP, SV.

Variations in heart rate (sinus arrhythmia) were reduced or abolished.

Propranolol caused slight or no reduction in HR, CO, dp/dt_{max}, AP and did not alter the pattern of response to vibration of the untreated dogs.

Following atropine or bilateral cervical vagotomy, characteristic increases in HR, CO, and dp/dt_{max} occurred. Vibration in dogs treated with propranolol and atropine or bilateral cervical vagotomy, or after the above and alpha adrenergic block with phenoxybenzamine produced

(1) decrease in AP, svr; (2) increase in CO, SV; and (3) no change in HR, dp/dt_{max}, EDP.

Figures 72 and 73 show typical pattern of blood pressure and pulse rate change during vibration. Values shown below the curves are cardiac outputs before and during vibration.

D. DISCUSSION

Responses to vibration in the untreated dogs were consistent with a fall in svr, countered by tachycardia, increased CO and dp/dt_{max}, and constant LVEDP, all mediated over decreasing parasympathetic efferent activity and/or increasing sympathetic efferent activity. Since, following beta adrenergic block with propranolol, the responses of all dogs were similar to these of the untreated group, we conclude that the response may result from either decreased parasympathetic efferent activity or increased sympathetic efferent activity, but that a decrease in parasympathetic efferent activity alone could produce all the effects.

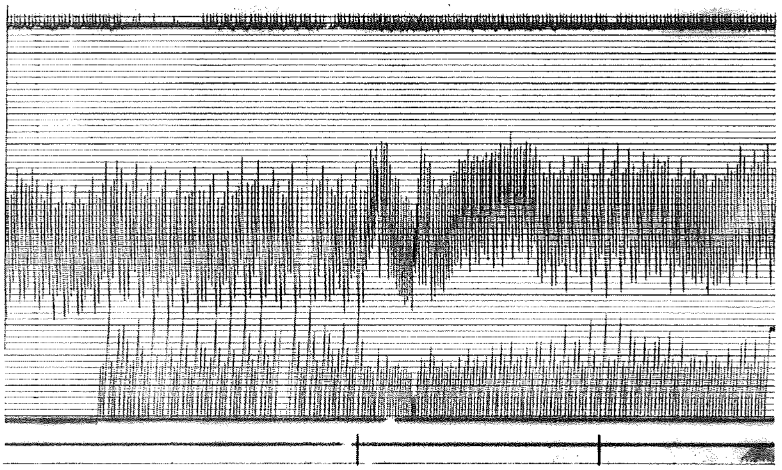
Following propranolol and atropine or bilateral cervical vagotomy, the heart could be considered denervated. That is, when either vagus was stimulated electrically at 15 V at 60 Hz, no bradycardia was produced; and when 0.5 μg isopropyl arterenol/kg body weight was given, no increase in either heart rate or dp/dt_{max} was produced. Vibration of the denervated dogs produced a fall in AP, a fall in svr, and an increase in CO. From this, we may conclude that vibration possibly caused direct stimulation to the heart which increased CO by increasing stroke volume, or that it caused a fall in systemic vascular resistance by direct vasodilatation or by interruption with alpha adrenergic efferent activity. The decrease in svr and fall in AP would then permit a greater SV at a constant SW.

Since the same responses were produced following alpha adrenergic block, we conclude that vibration of the normal dog causes direct vasodilatation; and that all other responses are mediated either through waning vagal efferent activity and waxing sympathetic efferent activity or the preceding coupled with increase in CO produced by increase in SV in presence of the fall in AP.

Too few experiments were performed and most changes were too small to justify statistical treatment of data. Direction and magnitude of alterations, however, were consistent. Homeostatic mechanisms (e.g., tachycardia), we believe, countered the fall in svr and minimized the reduction in AP produced by vibration. That these mechanisms were invoked no doubt impinged upon the ability of the system to respond to further stimuli. In any case, magnitudes of change could not be considered "clinically significant"; that is, a fall in AP of 15 mm Hg would hardly be indicated as a cause for alarm in healthy subjects. This could, however, be serious in subjects with preexisting disease (e.g., coronary artery disease, hyperthyrodism) or in unanesthetized human

POST PROPRANOLOL

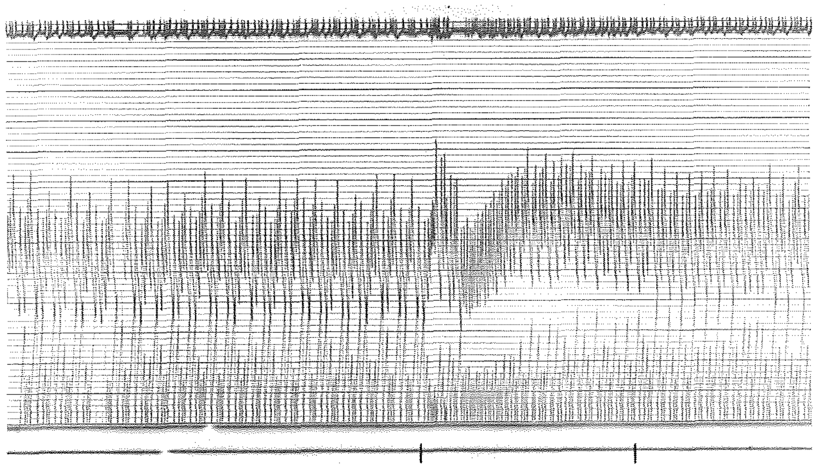
D-6



CONTROL
1.028

11 Hz
1.356
(+.328)

NO DRUGS D-6



CONTROL 0.956 C.O. 11 Hz 1.055 (+ 0.099)

beings subjected to moderation or intensification of these responses by psychic influences.

When man is vibrated pulse rate changes also occur. The pattern of change from the onset of vibration is consistent for an individual subject, but not consistent between individuals. The general patterns could result from temporary changes in systemic vascular resistance and the compensating sequences above postulated for dogs.

In some instances mean arterial pressure rose, while in others, mean arterial pressure fell immediately after onse of vibration. In all instances systemic vascular resistance decreased and heart rate increased. Variation in mean arterial pressure may be explained as follows.

Heart rate is fixed by vagal efferent activity which reflects degree of stimulation to baro-receptors in the systemic arterial tree. Stimulation to these receptors is produced more effectively by a pulsating pressure which obtains a certain mean than by a constant (or nonpulsatile) mean pressure. Thus, when heart rate accelerates during vibration, arterial pulsations decrease as pulse pressure falls, and a higher mean arterial pressure is required to produce cardiodeceleration equivalent to that produced by a lower mean arterial pressure but with a pulse pressure of greater magnitude.

For instances in which mean pressure increased during vibration, we propose that the cardioacceleration decreased the "effective" mean arterial pressure responsible for vagal efferent activity.

During this study an interesting but unexplained effect was observed. Three dogs with spontaneous unifocal ventricular extrasystoles, which occurred at the end of long asystole during expiration, reverted at the onset of vibration to normal sinus arrhythmias but without ventricular premature beats. Duration of diastole during expiration was as long during vibration as before; therefore, ventricular ectopic beats could have occurred via the "vagal escape" mechanism. That they did not occur during vibration could be explained by one of the following reasons:

1. Vibration abolished or overrode the "vagal escape possibly by its parasympatholytic effect;

2. "Vagal escape" was not the mechanism even before vibration.

Future studies on larger numbers and on unanesthetized subjects should be performed. Also the precise time-course of alterations in parameters measured here should be followed. Most importantly, genesis of the vibration-induced fall in systemic vascular resistance must be elucidated. Here, too, we might postulate (1) direct effect or (2) alteration in metabolism--possibly O_2 debt or CO_2 build-up in the vibrating dog.

III. TAPE RECORDER MODIFICATION FOR STUDY OF REPETITIVE NONPERIODIC SIGNALS*

Lester B. Roberts**

Transducer signals from cardiovascular systems such as ECG, arterial pressure, etc., are examples of more or less repetitive signals which are nonperiodic. They are more or less repetitive since while their general configuration remains constant, certain details may vary in time and from beat to beat. They are nonperiodic since their occurrence changes with change of heart rate. Signals of this type can often best be studied by displaying them on a persistent screen or storage oscilloscope or x-y recorder triggered so that succeeding traces are superimposed on preceding traces. How easily this can be accomplished depends on the temporal relationship between the portion of the signal of interest and a suitable signal trigger point. When the portion of the signal of interest occurs after a suitable triggerable signal point, a satisfactory display for study can be obtained simply by conventional triggering on this signal point. However, if, as is generally the case, the portion of the signal of interest precedes a suitable trigger point, it is necessary to employ special signal and trigger delay techniques to obtain the desired oscilloscope display.

An instrument tape recorder equipped with separate record and reproduce heads can be used to delay the signal (see Fig. 74). The signal is recorded at record head A and played back at record head B after a fixed delay equal to the time necessary for a point on the tape to travel from head A to head B. The signal is also fed to TD where it triggers an adjustable pulse delay circuit. The delayed pulse initiates the oscilloscope sweep. Adjustment of the trigger, the trigger delay, and the oscilloscope sweep time thus control the "superimposed" display of selected portions of the signal occurring anywhere, within limits, ahead or after the trigger point.

The above applies to on-line signal study and has been used by Hon[8] to store fetal ECG signals in register into a computer for signal-averaging noise reduction. Various schemes have been devised to permit similar studies of off-line pre-recorded data.[1,8] We have suggested [5] a method which requires modifications of the tape recorder by providing one or more reproduce heads ahead of the normal reproduce heads (see Fig. 75). The signal from the first head is the trigger signal. The signal from the second head is fed to the vertical amplifier of the oscilloscope. The operation is the same as described above.

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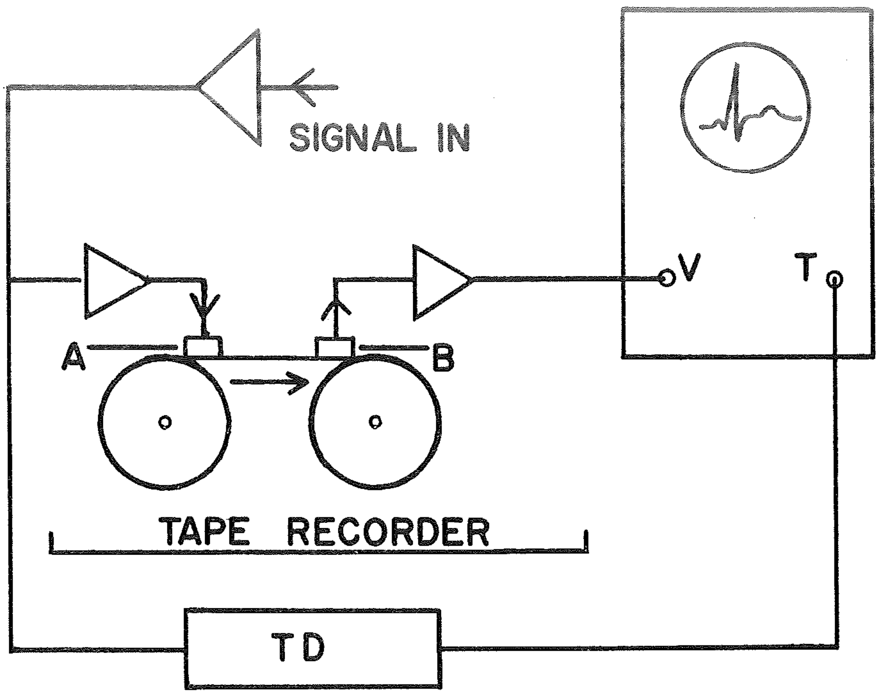
SIGNAL IN
V
T
A
B
TAPE RECORDER
TD

Fig. 74

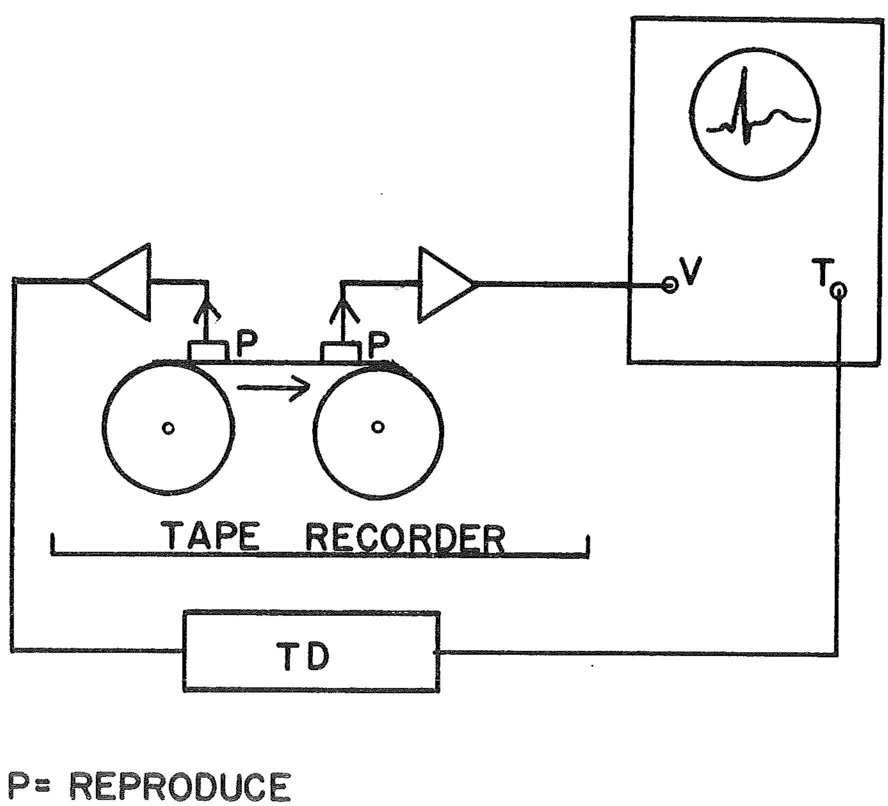
V
T
P
P
TAPE RECORDER
TD
P= REPRODUCE

Fig. 75

An Ampex SP-300 four-channel tape recorder has been modified as we have suggested. Since the record heads of this recorder are combination record-reproduce and since they are located ahead of the normal reproduce heads, it was possible to make the modification by simply wiring the existing record heads for reproduction. The installation is represented schematically in Fig. 76. In the actual modification, switching circuitry is provided between the reproduce amplifier and all of the forward heads. This provides a choice of trigger channels, which is an important feature in many studies.

The method has broad application and can be used for multi-channel signal comparisons. Used with an analog-to-digital interface it can provide a digital computer with oscilloscope type "delayed sweep" flexibility.

Following are some examples of application of the method to cardiovascular research.

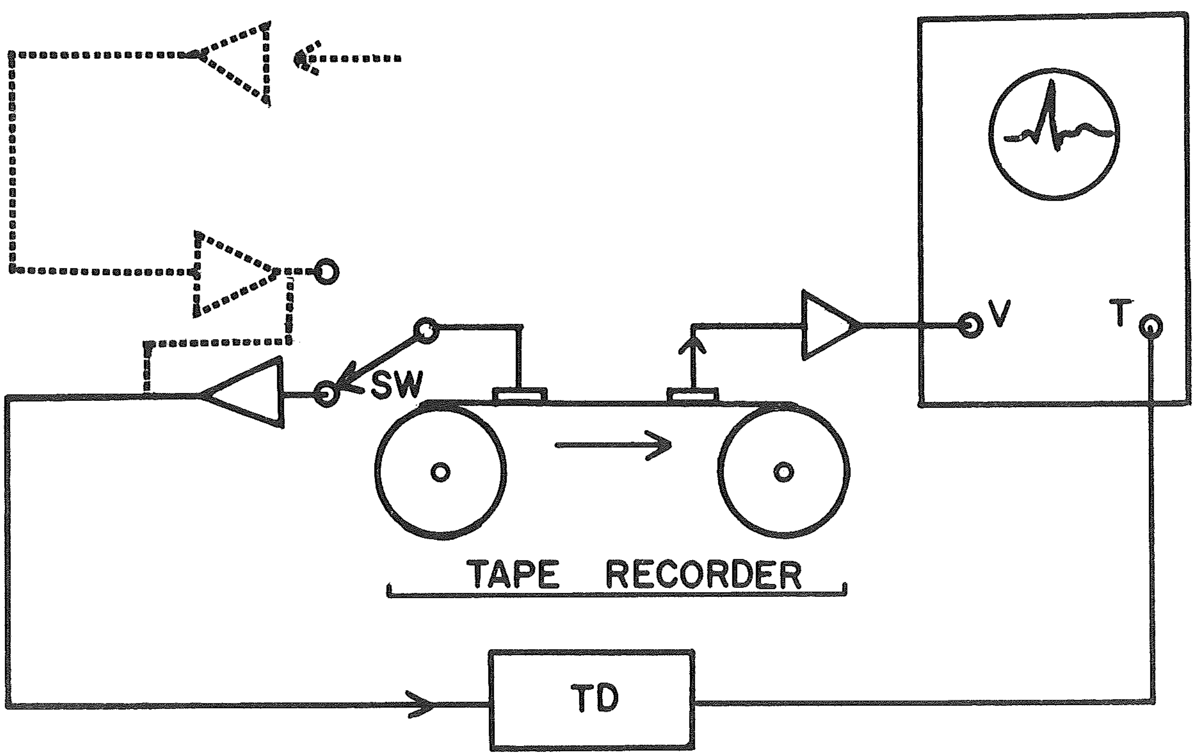

Fig. 76

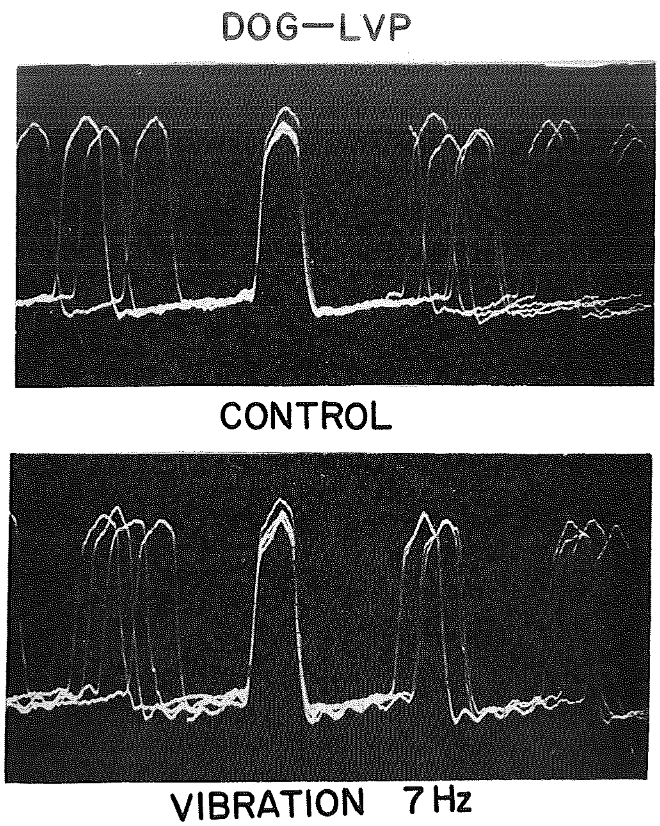

Fig. 77 - Blood Pressure in Left Ventricle of Dogs Heart During Control Period and Period Dog is Being Vibrated

Center complex shows four pressure curves superimposed. Complexes to left and right of center shows pressure curves preceding and following superimposed pressure curves.

Sweep time approximately three seconds.

Trigger approximately midway on ascending limb of LVP curve.

The small variability of the LVP curves both during control and during vibration can be seen. The average curves can be compared. Pulse intervals and interval variability can be compared; the curves demonstrate a more uniform pulse during vibration.

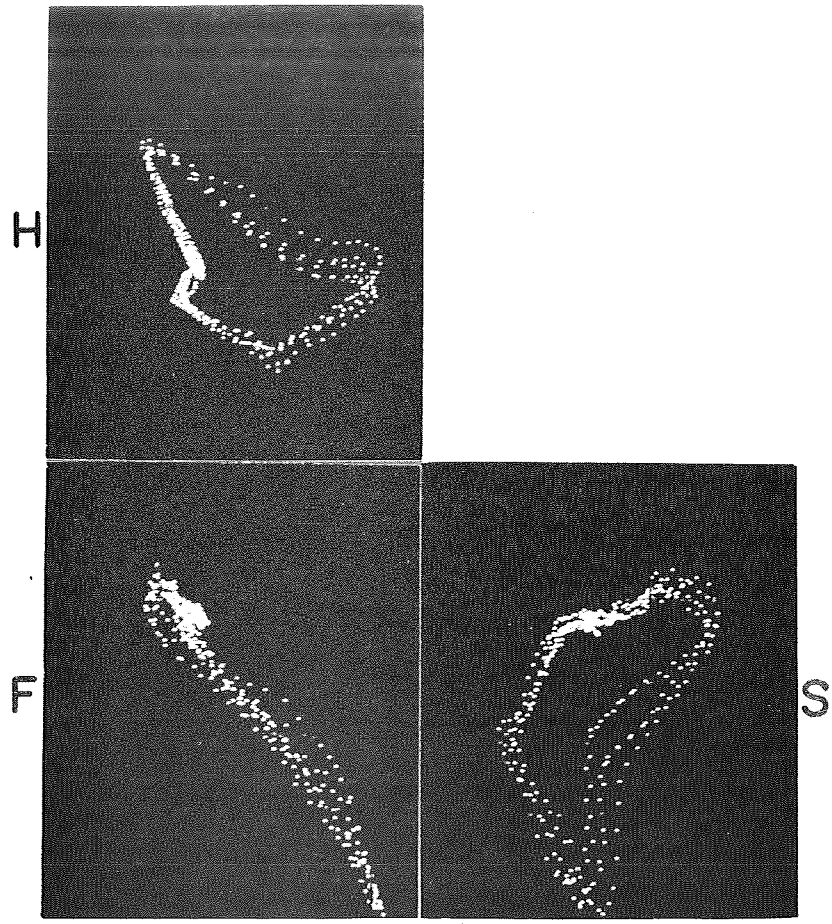

Fig. 78 - Vectocardiographs Taken From Human Experimental Subject While Being Vibrated

The vectocardiograms are photographs of oscilloscope plots (x-y, x-z, y-z) of the recorded three-lead orthogonal x, y, z, scalar electrocardiograms. Only the QRS portions of the vectocardiograms are shown. The trigger for all vectocardiograms was obtained from channel y. At the end of each trigger delay period the gate is opened which feeds a 1000 Hz modulating signal to the oscilloscope beam circuitry producing time marking during QRS period.

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