EFFECTS OF POSITIVE ACCELERATION ON THE ELECTROCARDIOGRAM

By

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Introduction

Accelerative forces resulting in inertial vectors acting from head to foot are termed positive G or +G. The cardiovascular system is more profoundly affected by +G than any other system of the body.

Although the cardiovasular reactions to +G have been studied by various workers, the electrocardiographic changes resulting from +G accelerations continue to be the subject of much discussion. The relative importance of the various factors producing such changes in the ECG are as yet not completely explained.

This paper presents the results of an analysis of E.C.G. records obtained during the Centrifuge runs carried out at the Institute of Aviation Medicine, IAF.

Material and Methods

The centrifuge runs were carried out on 86 subjects who were exposed to varying levels of +G between 2 to 4.5 G. The subjects comprised of normals (14), head injury cases (62) and cases with other disabilities (10). The results of these runs form the material of this review. The rate of build up of +G varied between 0.2 G/second and 0.5 G/second. The peak G ranged between +2G and +4.5G with the duration of peak kept below 15 seconds.

All subjects were fully instrumented for recording the standard (biopolar) leads I, II and III. Base line records at rest were obtained prior to the run. Recordings were continued throughout the run and for 5 minutes following the run. All the monitoring leads coming from the test person were connected to a junction box in the gondola and the signals were conveyed *via* the slip ring assemby mounted on the centrifuge arm to the rack incorporating the monitoring instruments. The monitoring equipment consisted of six channel oscilloscope type PSI, triple ECG amplifier model E 33 and a 6 channel jet recorder.

All ECG tracings were taken at 25 mm/second speed and records so obtained were critically analysed groupwise with particular reference to the following: Rhythm, Heart rate, P wave changes, P-R interval, QRS complex, T wave, and ST segment.

Results

The mean and ranges of age, height, weight and blood pressure of the subjects in the 3 groups are given in Table I.

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TABLE I

Mean and range of age, height, weight and blood pressure in the normal, head injury and miscellaneous groups of subjects
(Figures in brackets represent range)

Group	No.	Age (Yrs.)	Height (inches)	Weight (lbs.)	Basal Systolic BP (mm. Hg.)	Basal Diastolic BP (mm. Hg.)
Normal ..	14	32 (21-43)	66.5 (62-72)	143 (120-191)	127 (118-140)	84 (76-98)
Head Injury ..	62	26.5 (20-46)	67.6 (64-72)	138 (104-171)	129 (108-155)	85 (60-100)
Miscellaneous ..	10	25 (20-35)	66.3 (64-69.5)	137 (127-160)	124 (122-130)	85 (80-92)

The three groups are seen to be more or less similar with reference to all the variables. The mean age of the normals, however, is about 5-7 years higher than that of the other two groups. The 'normals' are members of the Institute staff who are of a higher age group while those who come for medical evaluation generally are aircrew and are of a younger age group.

Statistical analysis of the ECG data at rest and at various peak levels of +G has been carried out for the 14 normal subjects, 62 head injury cases and 10 miscellaneous cases suffering from various disabilities.

For the evaluation of ECG data 3 levels of peak G have been chosen. These 3 levels are +2G (includes 2.5G), +3G (includes 3.5G) and +4G (includes 4.5G).

Tables II, III and IV reveal significant trends in the distribution of some of the ECG components, with increasing acceleration, as against their corresponding values at rest. This is seen in all the three groups of subjects, though with varying magnitude. The changes that have occurred with increasing +G are:

(*a*) linear increase in the average heart rate;
(*b*) increase in average P wave amplitude;
(*c*) decrease in average P-R interval; and
(*d*) decrease in the median value of T wave amplitude in all cases; at higher levels of +G, T becomes flat, in a large number of cases and frankly inverted T are seen in some cases only.

It is also noticed that two cases showed ventricular extra systoles. Definite ST segment changes have been seen in five cases.

As agaist the above changes, no marked deviations in the other ECG components, *viz.*, electrical axis, QRS amplitudes and QRS interval have been observed.

Discussion

In evaluating the ECG changes during positive acceleration, it is necessary to proceed with the greatest caution. This is so because there is hardly any process which so profoundly disturbs the normal circulatory mechanism and produces in a few seconds grossly different working conditions for the

TABLE II

Average (median) and range of the various components of E.C.G. at rest and at different levels of +G in normal subjects
(Figures within brackets represent range)

Experimental Level (+G)	Number of subjects	Heart Rate (per mt.)	Elect. Axis (degrees)	P Amp. (mm.)	P-R Interval (Secs.)	QRS Amp. (mm.)	QRS Interval (Secs.)	T Amp. (mm.)	ST Segment*
Rest	14	81	50	1.25	.14	6	.06	2	13 Isoelectric (93%)
		(71-93)	(—20-70)	(Just positive to 1.75).	(.12-0.18)	(3-20)	(.06-.08)	(.25-3)	1 J depression (7%)
+2G	4	103	Not readable	1.5	.14	8	.08	Not readable	4 Isoelectric (100%)
		(100-103)		(1-1.5)	(.12-.16)	(6-8)	(.06-.08)		
+3G	14	111	60	1.75	.14	7	.06	1	11 Isoelectric (79%)
		(100-140)	(—30—75)	(1-2)	(.12-.16)	(3-21)	(.06-.12)	(just positive to 3)	1 Elevated (7%) 1 Depressed (7%) 1 J depression (7%)
+4G	9	125	50	1.75	.12	7	.06	1.5	6 Isoelectric (67%)
		(107-142)	(—10—80)	(1-2.5)	(.10-.16)	(1.5-12)	(.06-.08)	(Flat to 4)	1 Equivocal (11%) 2 J depression (22%)

* For ST segment, the figures represent the breakdown in terms of numbers and percentage of its several features.

TABLE III

Average (median) and range of the various parameters of E.C.G. at rest and at different levels of +G in head injury cases.
(Figures with brackets represent range)

Experimental Level (+G)	Number of subjects	Heart Rate (per m.)	Elect. Axis (degrees)	P Amp. (mm.)	P-R interval (Secs.)	QRS Amp. (mm.)	QRS Interval (Secs.)	T Amp. (mm.)	ST Segment*
Rest	61	75	50	1.5	.14	7 †	.06	2	59 Isoelectric (98%)
		(65-100)	(—30-80)	(Just positive to 2.25)	(.10-.18)	(0.5-19)	(.06-.12)	(Just positive to 4.5)	1 Elevated (1%) 1 Depressed (1%)
+2G	62	107	50	2	.14	7.5	.06	1	60 Isoelectric (97%)
		(79-136)	(—30—90)	(1-3.5)	(.08-.18)	(2-13)	(.06-.12)	(Flat to 4)	2 Depressed (3%)
+3G	61	120 (81-142)	55 (—30—80)	2 (1-4)	.12 (.08-.16)	8 † (2.5-20)	.06 (.06-.12)	(Flat Inverted to 3.5.)	59 Isoelectric (97%) 2 Equivocal (3%)
+4G	24	125 (96-136)	55 (—30—70)	2 (1-3.25)	.12 (.08-.06)	7.5 † (1-21)	.06 (.06-.12)	(Flat Inverted to 4)	21 Isoelectric (88%) 2 Equivocal (8%) 1 Depressed (4%)

* For ST segment, the figures represent the breakdown in terms of numbers of and percentage of its several features.

† All these extreme values pertain to the same case.

TABLE IV

Average (median) and range of the various parameters of E.C.G. at rest and at different levels of +G in miscellaneous groups of cases
(Figures within brackets represent range)

Experimental Level (+G)	Number of subjects	Heart Rate (per mt.)	Elect. Axis (degrees)	P Amp. (mm.)	P-R interval (Secs.)	QRS Amp. (mm.)	QRS Interval (Secs.)	T Amp. (mm.)	ST Segment*
Rest	10	83	55	1.25	.16	8	.06	2	10 Isoelectric (100%)
		(68-98)	(30 to 80)	(0.75-2)	(.14-.18)	(5.5-14)	(.06-.10)	(1.25-3.25)	
+2G	8	120	70	2.5	.14	9	.06	1.25	7 Isoelectric (88%)
		(83-136)	(30 to 70)	(1.5-2.5)	(.12-.16)	(3.5-14)	(.06-.08)	(Flat to 1.5)	1 Equivocal (12%)
+3G	7	136	45	1.75	.12	6	.06	1	5 Isoelectric (71%)
		(107-150)	(30 to 90)	(Just positive to 3)	(.12-.14)	(5-9)	(.06-.08)	(Just positive to 2.5)	2J Depression (29%)
+4G	6	136	60	2.5	.12	7	.06	Flat	6 Isoelectric (100%)
		(111-158)	(45 to 70)	(1.75-3)	(.10-.14)	(5-14)	(.06-.08)	(Flat to just positive)	

* For ST segment, the figures represent the breakdown in terms of numbers and percentage of its several features.

heart as do accelerative forces acting along the longitudinal axis of the large blood vessels.

Three main factors that modify the action of the heart and consequently the ECG are:

(1) Variation in the tone of extra cardiac nerves;
(2) variation in the filling of the individual chambers of the heart; and
(3) change of position/configuration of the heart during acceleration stress.

It is postulated that there is increased cardiovascular sympathetic tone during exposure to positive G.[8] In this phase, vagal tone is completely eliminated, but as soon as the centrifugal force is diminished, the vagal tone is greately increased.

During exposure to +G, the blood flow to both the atria is diminished and consequently the minute output is decreased. On the other hand, during deceleration, when recovery occurs, the atria and ventricles are suddenly distended with an excessive amount of blood.

When exposed to +G, the heart is elongated and is relatively displaced downwards.

Rhythm

An exaggeration of the normal sinus arrhythmia is very commonly seen. This may merely reflect the extra respiratory effort needed to raise the diaphragm.[8]

In the literature in this field, abnormality of rhythm has been reported only by Zuidema *et al.*[13] They exposed 5 subjects to varying levels of +G. Four subjects showed increasing myocardial irritability; three had premature ventricular contractions, and one had premature contractions of nodal or atrial origin. These authors have postulated that relative myocardial ischaemia is responsible for the increased cardiac irritability observed in their cases.

Bondurant[1] repeated the experiments with the same subjects under the same conditions a year later and was unable to produce any of the changes noted earlier. These changes are not what usually occur, but can be seen sometimes. Extra systoles, usually of ventricular origin, are seen occasionally under accelerative stress, particularly during deceleration after exposure to +G. A review of over two thousand runs on the RAF centrifuge has also shown that disorder of rhythm in the form of occasional extra-systoles do appear.[8]

Ventricular extra systoles of this nature are probably due to varying tonus of the extra cardiac nerves during and following acceleration stress.[10] In our own series of cases under review, we have seen two cases of ventricular extra systoles and one case of sinus arrest. These abnormalities appeared during the phase of deceleration. It is interesting to note that there was no occurrence of any arrhythmia in the group of 14 normal cases. Sinus arrest was seen in a subject who had suffered a head injury earlier and was asymptomatic when subjected to +G. Extra systoles were seen in a case of postural hypotension and a recovered case of encephalitis. At present, it is difficult to comment on the statistical as well as physiological significance of these findings on account of the small number of cases.

Heart Rate

The mean percentage rise in heart rate with increase in +G has been calculated and is given in Table V. It has been found that mean percentage rise is a better

index than the absolute increase of heart rate.

TABLE V

Mean % rise in Heart Rate with +G in Normal Head Injury and Miscellaneous groups of subjects

Group	Rest to 2 & 2.5G	Rest to 3 & 3.5G	Rest to 4 & 4.5G
Normal ..	22	32	63
Head Injury ..	35	51	62
Miscellaneous ..	47	73	76

It is seen that the heart rate increases significantly in all three groups, particularly with acceleration above +4G. While the increase for normals at 2G and 3G is gradual, it is rapid for head injury and miscellaneous groups of subjects. It is also seen that in the miscellaneous group, the percentage increse is larger than the other two groups.

Increase in heart rate per G has also been calculated. In normals, this increase per G is 10 per minute and in the head injury cases it is 11 per minute. The rate could not be calculated for the miscellaneous group due to inconsistency of heart rate changes.

Increase in heart rate in proportion to +G stress has been observed as early as 1937 by Buhrlen.[6] In general, the rate increases directly in relation to positive acceleration and finally stabilizes at a higher level. The increased pulse rate is due to reflex stimulation through the baroceptors, mainly in the carotid sinus.[7] During deceleration, a variable degree of bradycardia is always noted.

P-R Interval

The mean percentage fall in P-R interval has been calculated for the three groups of subjects. The P-R interval shows a tendency to shortening with increasing +G loads, in all the three groups as seen in Table VI.

TABLE VI

The mean % fall in P-R interval with +G in Normal, Head Injury and Miscellaneous groups of subjects

Group	Rest to 2 & 2.5G	Rest to 3 & 3.5G	Rest to 4 & 4.5G
Normal ..	*Nil*	6	12
Head Injury ..	7	12	14
Miscellaneous ..	9	17	21

As can be seen from the above table, the P-R interval decreases progressively with increasing acceleration, but never falls below the normal lower limit (0.1 second). This has been observed by Browne and Fitzsimons.[3] It is well known that with a rise in heart rate from any cause, the P-R interval is diminished. The reduction of P-R interval with increasing +G is purely due to increase in heart rate and there is no evidence to indicate any abnormality in conduction from SA node to AV node.

P Wave

The average (median) values of the P wave amplitude has been found to increase with +G. Table VII below shows the increases for the normals and head injury cases. The miscellaneous group has not been included for lack of consistency.

TABLE VII

Median rise of P amplitude (mm.) with +G in the Normal and Head Injury groups of subjects

Group	Rest to 2 & 2.5G	Rest to 3 & 3.5G	Rest to 4 & 4.5G
Normal ..	*	0.25	0.5
Head Injury ..	0.5	1.0	1.0

* Insufficient number of readings.

P wave is found peaked in leads II and III under G loads at all levels. Increase in amplitude of P wave is found statistically significant. It is more marked in head injury cases as compared to the normals.

Cohen and Brown [4] have also observed peaking of P waves in leads II, III and aVF. It has been postulated by these authors that P wave changes are qualitatively similar to those as occur during orthostasis or during stimulation with epinephrine and other sympathomimetic drugs. They were able to revert the ECG changes under positive G to the normal pattern by the administration of a beta adrenergic blocking agent like propranalol (Inderal).

QRS Complex

In our study the QRS complex shows very little change either with respect to amplitude or duration (interval). This is probably because we have used the bipolar leads only. Pryer *et al.* [10] have observed that the QRS complexes remain within normal limits.

When unipolar chest leads and limb leads are used, Bondurant [1] has found an increase in amplitude in the right sided chest leads and sometimes in aVL and aVF, although the size of the complex is reduced in leads from the left side of the chest. It has been suggested that the changes in R and S waves denote right ventricular preponderance.

Pryor *et al.* [10] have carried out spatial vector analysis and have found that the QRS and T vectors shift in opposite directions, resulting in an increase in the QRS-T angle. They also comment that the normal range of variation is very great.

Electrical Axis

Our study confined only to standard 3 leads (frontal plane triaxial system of Bayley), did not reveal any significant changes in the electrical axis of the heart. Browne and Fitzsimons [3] found that the changes in the manifest electrical axis with +G are small compared to changes due to respiration and cannot be used as indications of mean anatomical axis. An attempt to relate the change in the vector during respiration with changes in the anatomical axis measured on X-ray photographs produced extremely uncertain results. In this study, which included vector cardiography, they found little value in determining the change in electrical axis. The only conclusion which could be drawn was that the electrical axis during positive G moved in the same general direction as it did with inspiration. Bondurant [1] also failed to note any significant deviation in the electrical axis of the heart.

T Wave and ST Segment

The amplitude of the T wave shows conspicuous changes with acceleration in all the three groups. The changes are more significant in the head injury and miscellaneous groups. With +G many of the T waves which are upright became flat or inverted. The proportion of such cases increases as higher +G loads are imposed. In those cases where the T wave remains positive, a decrease in the mean value of amplitude has been observed (*See* Table VIII).

TABLE VIII

Mean decrease in T Amp. (mm.) for those remaining positive

Group	Rest to 2 & 2.5G	Rest to 3 & 3.5G	Rest to 4 & 4.5G
Normal ..	0.3	0.8	0.5
Head Injury ..	0.8	1.0	0.9
Miscellaneous ..	0.4	0.5	1.0

An overall estimate of the T wave changes can be obtained from Table IX which gives a break down of the total number of cases

TABLE IX

*** Number and percentage of the total number of cases that show changes in T wave**

Atributes	NORMAL †			HEAD INJURY				MISCELLANEOUS			
	Rest (n=13)	3 & 3.5G (n=14)	4 & 4.5G (n=9)	Rest (n=61)	2 & 2.5G (n=62)	3 & 3.5G (n=61)	4 & 4.5G (n=22)	Rest (n=9)	2 & 2.5G (n=8)	3 & 3.5G (n=7)	4 & 4.5G (n=6)
Positive ..	13 (100%)	9 (65%)	6 (67%)	57 (93%)	43 (69%)	28 (45%)	10 (45%)	9 (100%)	4 (50%)	3 (43%)	*Nil*
Just Positive ..	—	5 (35%)	1 (11%)	3 (5%)	—	1 (2%)	1 (5%)	—	—	1 (14%)	2 (33%)
" J " shaped ..	—	—	—	—	—	1 (2%)	—	—	—	—	—
Equivocal ..	—	—	1 (11%)	—	1 (2%)	1 (2%)	—	—	2 (25%)	1 (14%)	1 (17%)
Flat ..	—	—	1 (11%)	—	15 (24%)	26 (42%)	9 (41%)	—	2 (25)%	2 (29%)	3 (50%)
Inversion ..	—	—	—	1 (2%)	3 (5%)	4 (7%)	2 (9%)	—	—	—	—

* Distribution (Number and Percentage) of T wave configuration at rest and at various levels of peak ' G '.

† T wave changes at 2 & 2.5G are excluded for lack of sufficient number of readings.

in terms of the types of changes taking place in the T waves with positive accelerations as compared to these types at rest.

It is seen in the head injury group, where the large number of cases permits a probable conclusion to be drawn, the T wave is found flat in 24%, 42% and 41% at 2 (2.5) G, 3 (3.5) G and 4 (4.5) G respectively as against Nil at rest. The percentage of inversion also increases with higher +G in the same group. It is also noted that the T wave in four of the six cases in the miscellaneous group is not positive at 4 (4.5) G.

T wave and ST segment changes are usually considered of particular importance in the diagnosis of myocardial ischaemia and coronary insufficiency. ST segment changes in our study have been equivocal. The tachycardia usually seen during peak G makes accurate assessment of changes difficult. In our series, only 5 cases have shown ST segment changes out of a total of 89 cases. Experience on the RAF Centrifuge has been similar to ours. Flatenning or inversion of T wave without obvious depression or elevation of the ST segment is not usually of great diagnostic significance. Most authors at the present time are not prepared to accept that the flatenning and inversion of T wave which are almost universally noted during +G as indicative of acute myocardial ischaemia.

Zuidema *et al.*[13] noted that four out of their five subjects showed marked ECG changes namely cardiac arrhythmia. The fifth subject complained of substernal pain. In only one of the subjects who showed cardiac arrhythmia was marked ST segment changes noted. They attribute these changes to myocardial ischaemia. Pryor *et al.*[10] in their study did not find any significant ST segment shift to suggest coronary insufficiency in any subject. Brown and Fitzsimons[3] considered that the descent of the diaphragm during positive G could account for the comparatively small T wave variations in their subjects and that there was no evidence of myocardial ischaemia. Cohen and Brown[5] have discussed the possible mechanism responsible for the T wave and ST segment changes. They feel that these are reflex mediated and are an exaggeration of the changes seen in orthostatis and during stimulation with epinephrine and sympathomimetic drugs. In their study administration of a beta adrenergic blocking agent like propranolol completely normalised the ECG and the changes noted during positive G which they feel are a reflection of increased sympathetic tone and that there is no evidence to support the presence of myocardial ischaemia during positive G. McKee *et al.*[9] have also shown how administration of propranolol can reverse T wave and ST segment changes in cases of stress induced ECG changes.

More work is necessary before a final answer can be obtained as to the exact significance of the T wave and ST segment changes. Coronary perfusion studies under positive G conditions when undertaken may provide the answer.

Conclusion

As a result of the study, the following results have been noted:—

1. There is an increase in the heart rate with increase in +G, with bradycardia during deceleration.
2. The P-R interval is correspondingly reduced.
3. A consistent (peaking of P wave occurs with +G).
4. The QRS complex does not show any significant change.

5. The amplitude of T is universally diminished: the T wave becomes flat in a majority of cases or inverted in some cases.

6. There have been equivocal changes in the configuration of ST segment in a few cases.

7. The cardiac rhythm is not markedly changed during + G except that we found an exaggeration of the sinus arrhythmia in a few cases. Similarly, in two cases, extra systoles were found.

Acknowledgement

The authors wish to acknowledge the encouragement of Group Captain J. H. F. Manekshaw, Officer Commanding, Insitute of Aviation Medicine, during the preparation of this paper. The help of Mr. P. L. N. Rao, Statistician, is gratefully acknowledged.

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