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ORIGINAL ARTICLE

INFLUENCE OF HEART RATE ON EXERCISE INDUCED R WAVE AMPLITUDE CHANGES

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ABSTRACT: ECG is the most basic non-invasive, fairly informative and routine modality of investigation which gives clear and accurate information regarding cardiac function. An electrocardiogram associated with exercise is used as screening procedure for detecting latent coronary artery disease in asymptomatic patients. Present study is done to evaluate the R wave amplitude changes during exercise and to correlate heart rate changes that can influence ECG changes. It was seen during the study that R-Wave Amplitude increases after exercise upto heart rate of 120-130/min and decreases with further increase in the heart rate. This incidentally reflects end diastolic dimensions of left ventricle. **METHODS:** 64 normal non-athletic male subjects with a mean age of 19.06 years were chosen for the study. They were made to do exercise on Treadmill for 10mins, with a gradual increase in the speed every 2mins with constant inclination. ECG was recorded in standing position using a computer based serial communication electrocardiograph – CARDIOWIN – Genesis medical systems Pvt. Ltd. Heart Rate & R-Wave Amplitude were measured before exercise, immediately after exercise and 5mins after exercise. **RESULTS:** Heart rate changes, R Wave Amplitude expressed from lead II and average of chest leads from V1- V6 are taken as parameters. Paired t test was done. In majority of individuals RWA increased initially and later decreased. Corresponding values after 5mins of exercise showed that the decrease in Heart Rate is more pronounced with a reversal of R-Wave Amplitude. **CONCLUSION:** The study shows that with exercise the R wave amplitude increased in majority of individuals up to the heart rate of 120 – 130 and later the R wave amplitude decreased. It also shows that R wave amplitude changes are greatly influenced by changes in the heart rate which in turn are influenced by changes in systemic peripheral resistance. An increase in R wave Amplitude is seen in CAD due to coronary narrowing and ventricular dilatation leading to ventricular dysfunction. This change in R wave amplitude after exercise can give an approximate idea about the prognosis of the CAD patients.

KEYWORDS: Heart Rate; R Wave Amplitude (RWA); End diastolic Ventricular Volume, Coronary artery disease (CAD).

INTRODUCTION: Electrocardiography has become an integral part of clinical assessment of cardiovascular diseases. Assessment of cardiac function by ECG permits early detection of congenital defects and abnormalities associated with many cardiac diseases. During the past decades, there has been an exponential increase in the number of physiological tests designed to evaluate cardiovascular function. Till today, ECG is the most basic non-invasive, fairly informative and routine modality of investigation which gives clear and accurate information regarding cardiac function. ECG also provides valuable information in maintaining disease progression and response to treatment.

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Since Simonson made the first measurement of Electrocardio-graphic response to treadmill testing in 1953, the ECG in treadmill exercise has become a part of routine clinical examination.

Exercise induced changes in R wave Amplitude and Heart Rate in normal subjects were done earlier and it was shown that R wave Amplitude increased immediately after the starting of exercise but decreased gradually during exercise, increased again during the recovery phase.[1]
The influence of R wave amplitude on exercise induced ST depression was also studied and it was concluded that R wave amplitude greatly influences the magnitude of exercise induced ST segment depression and has to be taken into account in order to accurately interpret the exercise electrocardiogram.[2]

The influence of heart rate on exercise induced R wave amplitude changes in coronary patients and normal subjects was studied and it was seen that R wave amplitude changes are greatly influenced by heart rate.[3]

The present study was undertaken on normal and non-athletic subjects to emphasize the changes in R wave amplitude of ECG in relation to changes in heart rate after exercise on a Treadmill.

MATERIALS AND METHODS: This study was done among 64 young male adults in the Department of Physiology, Kurnool Medical College, Kurnool.

Inclusion Criteria: normal, non-athletic male subjects with a mean age of 19.06 years, SD $\pm$ 1.47, a mean body mass of 52.72 kgs, SD $\pm$ 8.76 and a mean height of 172.03 cms, SD $\pm$ 7.59 were choosen.

Exclusion Criteria: subjects with history of cardiovascular disease or any other disease which could be depicted to affect ECG, with chest cage abnormalities, with history of intake of vasodilator drugs or β- blockers and on drugs affecting ionic balance of body fluids were excluded.

The experimental details were explained to them prior to investigation and consent was obtained prior to participation. Ethical clearance was obtained from Institutional Ethical Committee.

MATERIALS AND INSTRUMENTS: ECG was recorded in standing position using a computer based serial communication electrocardiograph – CARDIOWIN (Genesis Medical Systems Pvt. Ltd, Hyd.). The same instrument was used throughout the study.

'Skintact' & 'Arbo' Adhesive disposable ECG electrodes were used to connect the chest leads. To keep them from further displacement during exercise, 'Gypsoplast' adhesive plaster was used. Clip electrodes were used to connect the limb leads.

A Treadmill manufactured by Messers Raghu industries – Chennai was used for exercise. Pulse oxymeter, Palkov was used to know the changes in Heart rate throughout the experiment.

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Test Methods: The height, weight & age of the subjects were recorded. The data was entered into the Cardiowin. The test was performed at a room temperature of 28^0 C. The adhesive chest leads were placed in position and clip electrodes were attached to the limbs using ECG gel. Normal ECG was recorded in standing position. Heart rate was noted directly from the Pulse oxymeter.

The leads were left in position. The treadmill speed was adjusted to 3.4 kmph, inclination 7% with subject on it and the subject was instructed to run on it for 2 minutes. The speed was increased for every 2 minutes to 5.1 kmph, 6.8kmph, 8.5 kmph and 10.2kmph with the same inclination for a total exercise period of 10 minutes and an ECG recording was made immediately. The subject was then made to relax on a flat bed for 5 minutes and another ECG was recorded at the end of recovery phase.

RESULTS:

Table 1: Relation between Heart rate and R – wave Amplitude

H. R. Group 100 – 110

Sl. No.	Subject	H.R	RWA LEAD H		RWA	Vi-V«
		IAE	I.A.E	B.E	I.A.E	B.E
2.	RK	108	0.909 ↓	1.020	0.706 ↓	1.114
10.	SR	104	0.456 ↓	0.565	0.318 ↓	0.572
15.	VK	100	0.642 ↑	0.465	0.442 ↓	0.512
22.	SD	100	0.707 ↓	1.188	0.672 ↓	1.014
24.	SY	100	0.772 ↓	1.182	0.624 ↓	1.146
36.	RG	102	0.999 ↑	0.988	0.872 ↓	0.958
39.	KH	108	0.421 ↓	0.529	0.172 ↓	0.441
41.	MH	100	0.565 ↓	0.581	0.488 ↓	0.565
44.	MV	100	0.037 ↓	0.439	0.216 ↑	0.197
59.	CK	106	0.998 ↑	0.819	0.533 ↑	0.519
62.	MR	102	1.406 ↑	1.218	0.732 ↓	1.012

Table 1: Relation between Heart rate and R – wave Amplitude

DOI: 10.18410/jebmh/2015/921

H. R. Group 111 -120

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E		B.E	RWA Vi-V« I.A.E		B.E
1.	GF	111	0.987	↓	1.249	0.812	↓	0.962
25.	HB	118	0.560	↓	0.834	0.476	↓	0.721
31.	KK	115	1.031	↑	0.971	0.924	↑	0.729
38.	RR	115	1.424	↑	1.249	1.276	↑	1.116
46.	GK	112	1.002	↑	0.786	0.878	↓	0.924
53.	VM	116	1.215	↑	1.015	1.272	↑	1.114
63.	MK	115	1.312	↑	0.890	1.124	↑	1.104

H. R Group 121 – 130

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E		B.E	RWA Vi - V_6 I.A.E		B.E
11.	AB	125	0.206	↓	0.501	0.512	↓	0.624
12.	RH	130	1.023	↓	1.372	0.099	↑	0.926
26.	CK	128	0.680	↑	0.649	0.541	↓	0.826
28.	MD	129	1.110	↑	1.101	0.856	↓	1.002
34.	EK	126	0.681	↓	0.880	0.653	↓	0.724
47.	YN	127	0.876	↑	0.636	0.412	↓	0.712
50.	RP	128	0.428	↓	0.668	0.712	↓	0.716
54.	AK	128	0.943	↑	0.819	0.928	↑	0.569
60.	NR	126	0.960	↓	0.987	0.633	↓	0.637

H. R. Group 131 – 140

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E		B.E	RWA Vi-V_6 I.A.E		B.E
6.	AM	131	1.048	↓	1.229	0.455	↓	0.965
7.	SW	137	0.489	↓	0.875	0.344	↓	0.658
17.	ML	131	0.477	↓	0.824	0.372	↓	0.918
19.	MH	140	0.479	↑	0.468	0.268	↓	0.512
21.	SV	134	1.028	↑	1.243	1.016	↑	0.962
30.	CS	139	0.961	↓	1.329	0.986	↑	0.906
37.	BP	138	0.816	↓	0.956	0.772	↑	0.649
43.	SS	134	0.840	↓	1.074	0.714	↓	1.248
48.	GA	131	0.916	↑	0.540	0.842	↑	0.441
49.	YA	136	0.642	↓	0.678	0.558	↓	0.723
55.	LL	140	0.852	↓	0.890	0.886	↑	0.516

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H. R. Group 141 – 150

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E	B.E	RWA Vi - V_6 I.A.E	B.E
3.	ES	146	1.061 ↓	1.256	0.401 ↓	1.216
5.	HS	148	0.319 ↓	0.491	0.382 ↓	0.542
8.	ZR	142	0.564 ↓	0.867	0.524 ↓	0.721
13.	ER	150	0.168 ↓	0.883	0.260 ↓	0.714
16.	SP	144	1.145 ↓	1.360	0.996 ↓	1.226
32.	GM	147	0.594 ↓	0.690	0.534 ↓	0.821
42.	VR	142	1.102 ↓	1.310	0.816 ↓	1.286
51.	VS	142	0.894 ↓	1.387	0.640 ↓	1.127
52.	GM	146	0.514 ↓	0.856	0.492 ↓	0.612
56.	PS	144	0.990 ↑	0.928	0.882 ↓	0.894
64.	RJ	145	1.270 ↓	1.337	0.905 ↓	1.096

H. R. Group 151 – 160

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E B.E		RWA Vi-V« I.A.E B.E	
4.	ND	159	1.419 ↑	1.316	0.956 ↓	1.249
18.	SS	158	0.128 ↓	0.340	0.116 ↓	0.472
20.	AJ	157	0.366 ↓	0.489	0.432 ↓	0.542
27.	SD	156	0.157 ↓	0.729	0.142 ↓	0.677
29.	KS	156	0.464 ↓	0.524	0.396 ↓	0.526
33.	NM	159	0.692 ↓	0.880	0.636 ↓	0.724
45.	AC	156	1.152 ↓	1.387	0.917 ↓	1.252
57.	SS	158	1.252 ↑	1.214	1.129 ↑	1.116
58.	SD	154	1.030 ↓	1.223	1.006 ↓	1.043

H. R. Group 161 – 170

Sl. No.	Subject	H.R IAE	RWA LEAD H I.A.E	B.E	RWA Vj-Vg I.A.E	B.E
9.	NR	163	0.678 ↓	0.856	0.899 ↓	0.932
14.	SR	162	0.971 ↓	1.223	0.646 ↓	1.043
23.	MJ	166	0.147 ↓	0.624	0.128 ↓	0.968
35.	RP	164	0.630 ↓	0.653	0.568 ↓	0.716
40.	AH	167	0.985 ↓	1.383	0.816 ↓	1.219
61.	CS	163	1.257 ↓	1.507	0.722 ↓	1.341

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H. R - Heart Rate / minute
RWA - Millivolts
BE - Before exercise
IAE - immediately after exercise

Table -2: Relation between Heart Rate and R – wave Amplitude

H. R. Group 70 – 80

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD II 5 min A.E	B.E	RWA Vi - V_6 5 min A.E	B.E
2.	RK	80	1.057 ↑	1.020	0.998 ↓	1.114
38.	RR	78	1.060 ↓	1.249	0.984 ↓	1.116
41.	MH	80	0.866 ↓	1.298	0.449 ↓	1.012
44.	MV	75	0.826 ↑	0.581	0.724 T	0.565

H. R. Group 81 – 90

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD II 5 min A.E	B.E	RWA V! - V_6 5 min A.E	B.E
4.	ND	81	1.457 ↑	1.316	1.256 ↑	1.249
22.	SD	85	1.006 ↓	1.188	1.416 ↑	1.014
24.	SY	87	0.802 ↓	1.182	0.768 ↓	1.146
30.	CS	85	1.386 ↑	1.329	1.214 ↑	0.906
36.	RG	86	1.026 ↑	0.988	1.233 ↑	0.958
37.	BP	86	1.256 ↑	0.956	1.072 ↑	0.649
46.	GK	86	1.248 ↑	0.786	0.856 ↓	0.954
48.	GA	88	1.034 ↑	0.540	1.102 ↑	0.441
50.	RP	89	1.011 ↑	0.668	0.953 ↑	0.716
51.	VS	84	1.216 ↓	1.387	1.019 ↓	1.127
56.	PS	87	0.898 ↓	0.928	0.653 ↓	0.894
60.	NR	85	0.925 ↓	0.987	0.602 ↓	0.637

H. R, Group 91 – 100

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD H 5 min A.E	B.E	RWA Vi - V_6 5 min A.E	B.E
1.	GF	94	1.277 ↑	1.249	1.264 ↑	0.962
6.	AM	97	1.247 ↑	1.229	0.955 ↓	0.965
10.	SR	93	0.422 ↓	0.565	0.428 ↓	0.572
11.	AB	93	0.206 ↓	0.501	0.316 ↓	0.624

DOI: 10.18410/jebmh/2015/921

15.	VK	100	0.427 ↓	0.465	0.412 ↓	0.512
16.	SP	97	1.531 ↑	1.360	1.232 ↑	1.226
26.	CK	92	0.691 ↑	0.649	0.735 ↓	0.826
42.	VR	98	1.458 ↑	1.310	1.256 ↓	1.286
47.	YN	94	0.839 ↑	0.636	0.726 ↑	0.712
52.	GM	96	1.406 ↑	0.856	1.213 ↑	0.612
59.	CK	91	0.488 ↑	0.439	0.216 ↑	0.197
62.	MR	93	0.783 ↓	0.819	0.623 ↑	0.519
63.	MK	97	0.965 ↑	0.890	0.576 ↓	1.104
49.	YA	100	0.628 ↓	0.678	0.548 ↓	0.723
21.	SV	100	1.347 ↑	1.243	1.219 ↑	0.962
39.	KH	95	0.561 ↑	0.529	0.513 ↑	0.441
25.	HB	95	0.821 ↓	0.834	0.766 ↑	0.721
52.	GM	96	1.046 ↑	1.024	1.224 ↑	0.569

H. R. Group 101 – IIP

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD II 5 min A.E	B.E	RWA 5 min AE	v,-v B.E
7.	SW	105	0.917 ↑	0.875	0.928 ↑	0.658
9.	NR	110	0.859 ↑	0.856	0.723 ↑	0.932
12.	RH	101	1.340 ↓	1.352	1.258 ↓	0.926
17.	ML	101	0.759 ↓	0.824	0.624 ↓	0.918
28.	MD	105	1.358 ↓	1.101	1.413 ↓	1.002
31.	KK	110	0.902 ↑	0.971	0.856 ↑	0.729
33.	NM	106	0.956 ↑	0.880	0.812 ↑	0.724
34.	EK	107	1.141 ↑	1.025	1.066 ↑	1.248
35.	RP	108	0.431 ↓	0.653	0.409 ↓	0.716
40.	AH	102	1.252 ↑	1.383	0.848 ↑	1.219
43.	SS	110	0.944 ↓	1.074	0.958 ↓	1.248
45.	AC	108	1.122 ↑	1.387	1.111 ↑	1.252
53.	VM	105	0.958 ↑	1.015	0.716 ↑	1.114
57.	SS	110	0.903 ↓	1.214	0.713 ↓	1.116

H. R. Group 111 – 120

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD H 5 min A.E	B.E	RWA Vi - V_6 5 min A.E	B.E
3.	ES	112	1.205 ↑	1.256	1.018 ↓	1.216
8.	ZR	113	0.674 ↑	0.837	0.566 ↑	0.721
19.	MH	116	0.443 ↓	0.468	0.482 ↓	0.512

DOI: 10.18410/jebmh/2015/921

27.	SB	119	0.791 ↓	0.729	0.514 ↑	0.677
32.	GM	116	0.520 ↓	0.690	0.423 ↑	0.821
55.	LL	112	0.856 ↑	0.890	0.754 ↓	0.576

61.	CS	113	1.625 ↑	1.507	1.138 ↑	1.341
23.	MJ	120	0.339 ↓	0.624	0.532 ↑	0.968

H. R. Group 121 - 130

Sl. No.	Subject	H.R 5 min A.E	RWA LEAD H 5 min A.E B.E		RWA Vi - V_6 5 min A.E B.E	
5.	HS	122	0.613 ↑	0.491	0.774 ↓	0.542
13.	ER	127	0.362 ↑	0.883	0.374 ↑	0.712
14.	SR	121	1.212 ↓	1.223	1.019 ↓	1.043
18.	SS	122	0.310 ↓	0.340	0.218 ↑	0.472
20.	AJ	128	0.352 ↓	0.489	0.473 ↑	0.542
58.	SD	124	0.963 ↑	1.223	0.908 ↓	1.043
64.	RJ	128	1.221 ↑	1.337	1.065 ↓	1.096

H. R. Group 131 – 140

Sl. No	Subject	H.R 5 min A.E	RWA LEAD II 5 min A.E B.E		RWA Vi - V_6 5 min A.E B.E	
29.	KS	135	0.735	0.524	0.738 t	0.526

H. R- Heart Rate / minute
RWA - Millivolts
BE - Before exercise
5Min A.E - Five minutes after exercise

Table 3: R – Wave Amplitude changes with progressively increasing Heart rate steps before exercise X immediately after exercise:

Heart rate steps (bpm)							
H.R	100-110	111-120	121-130	131-140	141-150	151-160	161-170
	↓↑	↓↑	↓↑	↓↑	↓↑	↓↑	↓↑
RWA Lead II	7/11 4/11	3/7 4/7	5/9 4/9	9/11 2/11	10/11 1/11	7/9 2/9	6/6 -
RWA V1-V6	9/11 2/11	3/7 4/7	7/9 2/9	7/11 4/11	11/11 0/11	8/9 1/9	6/6 -

DOI: 10.18410/jebmh/2015/921

Before exercise X 5 Minutes after exercise:

Heart rate steps (bpm)							
H.R	70-80	81-90	91-100	101-110	111-120	121-130	131-140
	↓↑	↓↑	↓↑	↓↑	↓↑	↓↑	↓↑
RWA Lead II	2/4 2/4	5/12 7/12	6/18 12/18	9/14 5/14	6/8 2/8	6/7 1/7	0/1 1/1
RWA V1-V6	3/4 ¼	5/12 7/12	8/18 10/18	9/14 5/14	7/8 1/8	5/7 2/7	0/1 1/1

Paired t test was done for changes in heart rate to changes in R wave amplitude. Changes in R wave amplitude are noticeable in lead II and also in chest leads. The difference between changes in lead II and $V_1 – V_6$ average are almost similar.

Table I shows relation between heart rate and R wave amplitude wherein we have taken 7 subgroups according to the heart rate changes. In the first group heart rate changes from 100 – 110, in the second group 111 – 120, 121 – 130 in the third, 131 – 140 in the fourth, 141 – 150 in the fifth, 151 – 160 in the sixth and 161 – 170 in the seventh group. The corresponding R wave amplitudes in L - II and average of R wave amplitudes from $V_1 – V_6$ are depicted in each tabulation. The increase in heart rate in each subject immediately after exercise corresponding to the change in the R wave amplitude after exercise was depicted and compared with the normal value i.e., before exercise. An increase is marked as ↑ and a decrease as ↓.

Table II shows relation between heart rate and R wave amplitude where we have taken 7 subgroups again according to the heart rare changes. In the first group heart rate changes from 70 – 80, in the second group 81 – 90, 91 – 100 in the third, 101 – 110 in the fourth, 111 – 120 in the fifth, 121 – 130 in the sixth & 131 – 140 in the seventh. The corresponding R wave amplitudes in L – II and average of R wave amplitudes from $V_1 – V_6$ are depicted in each tabulation. The change in heart rate in each subject 5 minutes after exercise corresponding to the change in the R wave amplitude 5 minutes after exercise was depicted and compared with the normal value i.e., before exercise. An increase is marked as ↑ and a decrease as ↓.

Table III shows a comparative statement of the results obtained in the present study showing the relationship between a proportionate change of heart rate resulting in the change of R wave amplitude. It gives the consolidation of results. The first part of the table shows R wave amplitude changes immediately after exercise and second part shows changes 5 minutes after exercise. The first part of the table gives us a clear picture that initial increase of heart rate up to 120 corresponds with a predominant increase of R wave amplitude and further increase of heart rate results in the decrease of R wave amplitude. Similar relationship 5 minutes after exercise was shown in the lower part of the table.

Increase in R wave amplitude in lead II occurred more up to 121 – 130 heart rate steps and then declined. In the heart rate stage of 161 – 170 all got decreased in R wave amplitude of both in lead II and average of V_1- V_6. The results show that R wave amplitude depends on heart rate which in turn effects the end diastolic volume and the dimensions of the ventricular changes. The more decrease of R wave amplitude in chest leads gives an approximation of axis deviation affecting the R wave amplitude since axis deviation is a common result of exercise.

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DISCUSSION: A lot of development occurred in the instrumentation and the recording of the ECG as well as its interpretation. In the diagnosis of coronary artery disease two important ECG changes are taken into consideration i.e., S – T segment displacement and R wave amplitude changes. Making the person to do exercise, to simulate transient coronary ischemia became a standard pattern of experimentation both in research and in patient care. It was studied that in a normal human at rest, the arterial saturation can be 95% and the venous 75% resulting in an AV-O_2 difference of 20%. This pattern is altered in cardiac patients with low outputs, so that the AV-O_2 difference may be as high as 40% due to a drop in venous oxygen to 55% or 60%. The coronary circulatory system does not have the capacity to adapt to this degree, because of its relatively high extraction rate of oxygen at normal work levels. Coronary sinus blood returning from the capillary bed of the myocardium is usually from 10% to 25% saturated, resulting in an AV-O_2 difference across the myocardium of 75% or more.[4]

Treadmill came into existence in this context, to impart graded exercise to the patient and to record the appropriate ECG changes both in apparently normal individuals and in CAD patients.

The initial stimulus to investigate the significance of R wave in exercise testing came from the work of Brody (1956) who suggested that exercise induced changes in left ventricular function and volume may be reflected in the electrocardiogram by alterations in QRS voltage.

Changes in the amplitude of QRS complexes can also occur due to slowed conduction through the ventricles which can lead to larger than normal amplitude of depolarization waves. Hypertrophy of left ventricle due to long standing high blood pressure, hypertrophy of right ventricle due to elevation of pulmonary arterial pressure can give rise to increased amplitude in the QRS complex.[5]

However in patients with coronary artery disease the development of myocardial ischemia is almost always associated with dilatation of the left ventricle. The intial finding of a decrease in the R wave in normal subjects and increase in R wave in patients with coronary artery disease appeared to fit well into Brody's hypothesis.

In normal heart it is likely that there is a progressive decrease in systolic and diastolic left ventricular volumes with exercise that is possibly sympathetically mediated. The role of intracardiac volume in controlling electrocardiographic R wave amplitude changes during acute myocardial ischemia was studied in open chest dogs.[6] It suggested that factors other than intracardiac volume determine R wave amplitude changes in the cause of myocardial ischemia.

The response of R wave amplitude to postural changes and to exercise emphasizes the fact.,[7] At rest in supine position the R wave amplitude was almost similar, but in sitting position at rest and at the begining of the exercise the R wave amplitudes were significantly higher in physically active healthy men than in sedentary healthy men or in infarction patients. Physical training during the first post infarction year induced only negligible changes in R wave amplitude response to postural changes and exercise.

An increase in R wave amplitude after exercise should be included among the variables with prognostic value for further coronary events.[8]

Effect of change in venous return on R wave amplitude was studied.[9] Various factors which affect venous return thus influencing the R wave amplitude were studied. A significant increase in R wave amplitude by 57.1% ($P<0.01$) was observed when subjects submerjed themselves in water in an upright position, which might be caused by an increase in venous

return due to the increased water pressure that acted on the lower part of the body. Exercise in supine position ex: swimming would be expected to be associated with a large stroke volume due to increased venous return to the heart as gravity is removed. At low levels of horizontal exercise, the heart rate is the main source of increase.(10)

In this study, the results obtained clearly demonstrate a specific correlation of the R wave amplitude to the heart rate. Subsequent increase in heart rate beyond 140 beats per minute definitely decreases the R wave amplitude. The gradual decrease in R Wave Amplitude during exercise may be related to gradual increase in heart rate and a gradual decrease in systemic peripheral resistance. and gradual increase in R Wave Amplitude after exercise may be related to gradual decrease in heart rate and a gradual increase in systemic peripheral resistance.

Since there is the possibility of exercise induced changes in the anatomic position of the heart and axis shifts in direction of the maximal QRS potentials towards chest wall, and to take up 16 primordial leads as an appreciation of the said results,(11) in the present study we have envisaged recording both R wave amplitudes in lead II for comparison with the average of V_1 – V_6. The results tallied in majority of the instances, but in R wave amplitude after exercise, the change in R wave amplitude in some instances varied with the average of V_1- V_6 compared to lead.[11] the results show that R wave amplitude depends on heart rate which in turn effects the end diastolic volume and the dimensions of the ventricular changes. The more decrease of R wave amplitude in chest leads gives an approximation of axis deviation affecting the R wave amplitude since axis deviation is a common result of exercise. Similar changes of R wave amplitude are obtained 5 minutes after exercise where in increase in amplitude occurred. The results indicate that heart rate is one of the most important factors affecting the R wave amplitude inspire of apparent decrease in venous return occurring 5 minutes after exercise, the lower heart rate having a higher diastolic filling capable of increasing R wave amplitude.

Administration of nitrates could reverse R wave amplitude changes during exercise. Nitrates are coronary vasodilators they augment coronary blood flow as well as oxygen supplement to the cardiac muscle, It has a definite role in reversing the effects of exercise and shortening the recovery period.(12)

Athens QRS score is a new parameter for analysis of stress ECG based on changes in Q, R & S wave amplitudes in resting and immediate post exercise ECG. Studies show that the amplitude of Q wave either increases or remains unchanged after exercise in normal individuals but fails to increase in the presence of CAD. R Wave amplitude increases with exercise in CAD patients S wave amplitude increases after exercise in normal individuals but a decrease in amplitude is seen after exercise in CAD patients.(13)

Since dilatation is the more common sequence of chronic CAD, which invariably is capable of causing increase in R wave amplitude, this change in R wave amplitude can give an approximate idea about the prognosis of the CAD patients. This increase has twice the probability of having an event compared with the remaining patients. Thus the increase in R wave amplitude after exercise can be postulated as a predictor of CAD and also is a very important factor in considering the CAD patient's prognosis.

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CONCLUSION: With exercise the R wave amplitude increased in majority of individual's upto the heart rate of 120 – 130 and later the R wave amplitude decreased. Changes in the R wave amplitude are noticeable in lead II and also in chest leads. Corresponding values for heart rates 5 minutes after exercise show that where the decrease in heart rate is more pronounced, reversal of R wave amplitude is more common. R wave amplitude increases with increase in the chamber dimensions either exercise induced or in dilatation occurring in chronic CAD patients. This change in R wave amplitude can give an approximate idea about the prognosis of the CAD patients.

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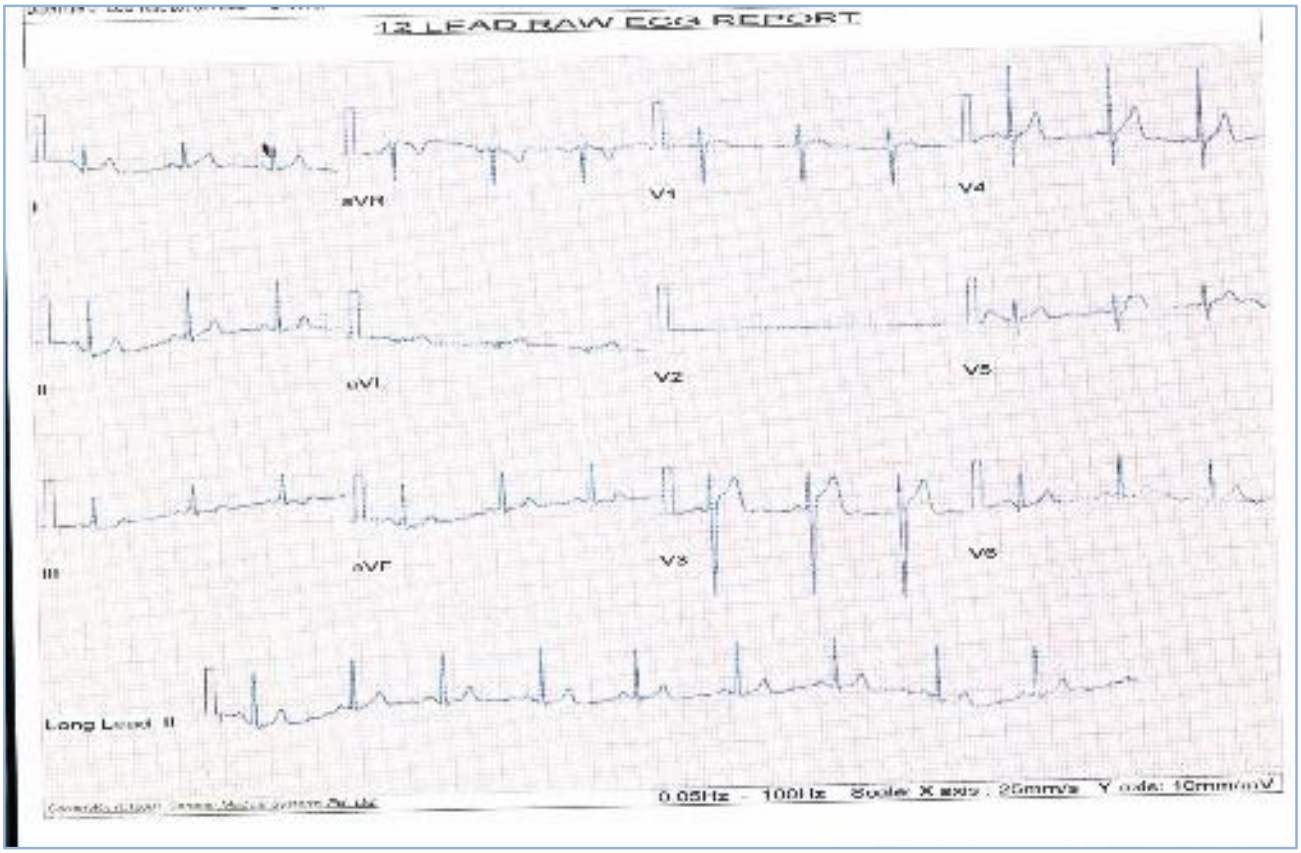

ECG

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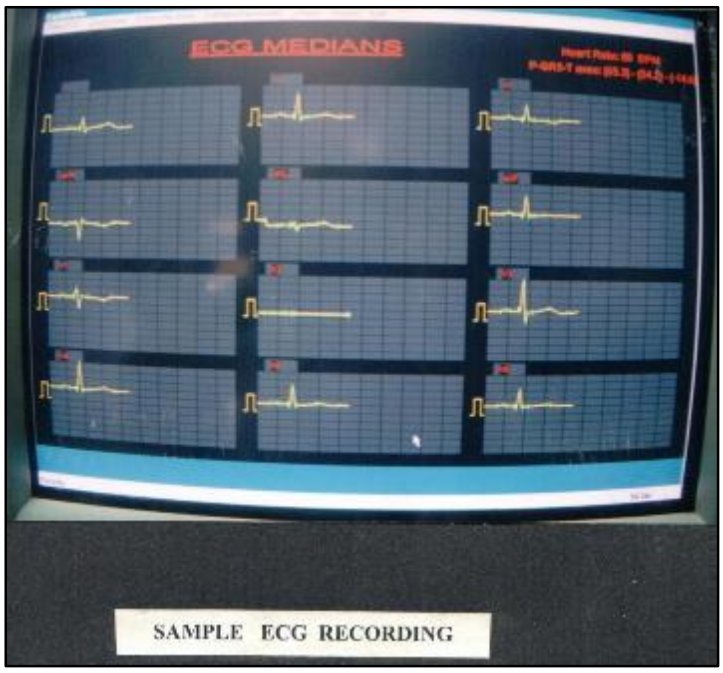

SAMPLE ECG RECORDING

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