ECG CHANGES IN YOUNG COMPETITIVE INDIAN ATHLETES

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HOW TO CITE THIS ARTICLE:

M. Padma Geethanjali, P. S. N. Raju, V. Seetharama Raju. "ECG Changes in Young Competitive Indian Athletes". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 04 January 12; Page: 682-701, DOI: 10.14260/jemds/2015/99

ABSTRACT: BACKGROUND: The Purpose of this study was to compare the changes in ECG's of young competitive Indian athletes with that of normal young non athletic persons. **METHODS:** The study included 100 subjects out of which 50 were competitive athletes and 50 were non-athletes age ranging between 16-30 yrs. Following parameters were accessed: Resting Heart Rate. **Rhythm.** **Intervals:** PR, QRS, QT, QTc. Axis. These parameters were obtained using ECG machine 1101 with 12 lead European mode, the data was statistically analysed using graphpad software, the data was compared using student t test. **RESULTS:** Heart Rate There was significant Bradycardia in 26% of Athletes and none with Tachycardia. The Heart Rate was in normal range in 98% of Non-Athletic Group and 2% showed Tachycardia. On Comparison by paired t test, Athletic Group had Significant Bradycardia ($P = 0.0001$). **RHYTHM:** Sinus Rhythm was assessed in all ECG's of either of the groups. **PR interval:** PR interval was found to be normal (0.12-.20 s) in 96% of Athletes and 4% showed prolonged PR interval. PR interval was normal in all non-athletes. There was no statistical significance ($P=0.074$) in PR intervals of both groups. **QRS duration:** QRS duration of 86% athletes showed normal duration (0.08 – 0.12s) and 14% showed prolonged QRS duration. QRS duration of 96% non-athletic group showed normal duration, 2% showed prolonged and 2% showed decreased duration. There was significant difference in QRS duration ($P=0.003$) between the two groups. **QT interval:** QT interval is normal (0.36-0.44s) in 50% of athletic group and 50% showed prolonged duration. QT interval was normal in 100% Non-athletic group. There was significant increase in QT interval ($P=0.0001$) between the two groups. **QT c interval:** QTc interval was normal (0.43-0.45s) in 96% of athletes, and 4% showed prolonged QTc interval. QTc was normal in 100% non-athletic group. There was significant difference ($P=0.0001$) in QTc interval between both the groups. **Axis Deviation:** Among athletes 16% showed Left Axis Deviation and 4% showed Right Axis Deviation. Among Non-athletes 4% showed LAD, 2% RAD. **CONCLUSION:** There was significant Bradycardia, Prolonged QRS duration, Prolonged QTc intervals in athletic group when compared to non-athletic group. There was 16% left axis deviation, 4% RT axis deviation in athletic group. There was 4% LAD and 2% RAD in Non athletic group.

KEYWORDS: Athletes, ECG, QRS, QT, QTc Intervals, Competitive, Indian athletes.

INTRODUCTION: Regular sports participation is encouraged by the medical community as it improves fitness and reduces cardiovascular morbidity and mortality. A large proportion of the young population participates in competitive or recreational sports activity. Cardiovascular exercise involves the use of large muscles in a repetitive fashion, activating muscle fibers programmed for endurance and utilizing a heart rate range anywhere from 40 to 85 percent of our maximum heart rate. When performing cardiovascular exercise, blood flow is directed toward working muscles and away from areas that aren't doing much (such as your arms during running, or the digestive tract). There is increased blood flow, and blood volume returning to the heart. As the heart registers a larger

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blood volume, over time the left ventricle adapts and enlarges. This larger cavity can hold more blood, and ejects more blood per beat, even at rest. Over time, with chronic cardio training, our resting heart rate drops because each beat delivers a bigger burst of blood, and fewer beats are needed. This takes work off your heart and is why cardio exercise is recommended for healthy heart. However, cardiovascular exercise can also produce stress.

If we get into over-training, we may hit a point where we are drowning in cortisol. This eventually leads to immune-suppression and fat gain around the abdomen and face. The heart adapts to this by increasing the thickness of the left ventricle wall. This thickness derived from chronic weight training is healthy, whereas the thickness from chronic high blood pressure is not. What's the difference? The healthy heart only has to work under pressure for two to three hours of strength training per week, whereas the heart with high blood pressure has to work 24 hours a day, seven days a week. The second heart may exhaust, whereas the healthy heart becomes stronger with a lower resting heart rate. Exercise also stimulates the production of new blood vessels. As we make more blood vessels, there are more places for blood to flow, which results in more efficient circulation. Cardiovascular exercise increases the number of new blood vessels while resistance training increases the size of those blood vessels. There will be a profound improvement in cardiovascular function. The basal heart rate decreases due to increased vagal tone. Stroke volume increases due to increased myocardial muscle mass. A trained subject achieves the required cardiac output during exercise mainly by increasing the stroke volume rather than by heart rate, where as an untrained individual achieves the same cardiac output mainly by increasing the heart rate.

MATERIALS AND METHODS: The Study included 50 young competitive male athletes of age group ranging from 16-30 years; All the Subjects are of Indian origin hailing from Hyderabad and Visakhapatnam undergoing training at Lal Bahadur Sastry Stadium at Hyderabad, Railway stadium at Secunderabad, Port Stadium at Visakhapatnam comparing with 50 normal healthy subjects with irregular sports activity.

Sedentary non-athlete individuals of same age groups were selected.

Criteria for primary selection: Daily physical exercise category of 3-4 hrs/day since the last 5 years and specially trained by coach or a trainer.

My study is done as per the European Society of Cardiology including the recommendations alongside history and physical examination the inclusion of a resting 12-lead electrocardiogram (ECG).

The Data was statistically analyzed using Graphpadsoftware, the data was compared using unpaired t test.

RESULTS:

Heart Rate: Normal 60-100 beats/min,Bradycardia< 60, Tachycardia>100

	Normal	Bradycardia	Tachycardia
Athletes -50	37	13	-
Non-Athletes -50	49	-	1

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26% of Athletes had Sinus bradycardia and none had tachycardia, Average Heart rate in this group was 66 beats/min, Tabular form above showed difference in men and women.

Among the Non-Athlete group 98%(49/50 subjects) showed normal heart rate and just 2% (1 subject) showed tachycardia changes.

PR Interval: (Normal PR Interval 120-200 msec): In athletes, 96%(48/50 subjects) showed PR intervals ranging from 120-200msec and 4%(2/50) showed increase in PR intervals time.

Among on athletes 100% subjects showed normal range of PR interval.

	Normal(120-200 msec)	↑> 200 msec	↓< 120 msec
Athletes -50	48	2	-
Non-Athletes -50	50	0	0

QRS Duration: Normally- The QRS wave's duration is 0.08-12 sec (80-120msec).

Among athletes 14% (7/50) subjects showed increased QRS interval period, none had any momentum of decreased QRS intervals.

Among Non-athletes 2% (1/50) showed increased QRS interval, 2% (1/50) showed decreased QRS interval duration.

QRS Duration	Normal(80-120 msec)	↑> 120 msec	↓< 80 msec
Athletes -50	43	7	0
Non-Athletes -50	48	1	1

QT INTERVAL: Normal QT is (0.36 – 0.44 s) equal to or below 0.40s (400msec).^{4,5,7,8,10} Among athletes 50% (25/50) had prolonged QT interval. Average QT interval of this group is 401 msec, which exceeded the expected range of 400 msec.

Non-of the Non-Athletic group had any prolonged or decreased QT interval, the average QT interval is being 351msec.

QT Interval	Normal(360-120 msec)	↑> 120 msec	↓< 80 msec
Athletes -50	43	7	0
Non-Athletes -50	48	1	1

Corrected QT interval (QTc):

- Borderline "QTc" in males is 431-450 ms, and in females 451-470 msec.
- An "abnormal" QTc in males is a QTc above 450 ms, and in females, above 470 ms.
- Athletes showed any average QTc intervals of 420 msec which is within normal range, but 4% (2/50) male athlete subjects showed increased level of QTc interval.

All Non-athlete subjects were well in normal range in this duration.

QTc Interval	Normal	↑ Increased QTc
Athletes -50	48	2
Non-Athletes -50	50	0

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AXIS: A normal heart axis is between -30 and +90 degrees.

- A left heart axis is present when the QRS in lead I is positive and negative in II and AVF. (between -30 and -90 degrees)
- A right heart axis is present when lead I is negative and AVF positive. (Between +90 and +180).

Among Athletes 16% (8/50) subjects showed Left Axis deviation (LAD), 4% (2/50) showed Right Axis Deviation (RAD).

Among Non-Athletes 4% (2/50) subjects showed Left axis deviation (LAD), 2% (1/50) showed Mild Right deviation of axis diagnosed to have RBBB (RAD).

SL. No.	Name	Age	Sex	HR	P Durat	Intervals			T Durat	QTc	Axis	Remarks
						PR	QRS	QT				
1	RK	26	M	62	108	122	82	390	206	382	47°	
2	SS	26	M	50	101	132	81	↑470	194	429	28°	Sinus Bradycardia
3	RA	26	M	54	↑123	192	96	↑453	202	430	62°	Sinus Bradycardia
4	PN	26	M	70	110	145	82	407	235	440	-44° - Lt	
5	RK	25	M	55	102	120	101	425	212	410	64°	Sinus Bradycardia
6	SS	23	M	68	112	155	87	410	215	439	36° - Lt	
7	SD	24	M	74	112	130	97	370	195	411	38°	
8	BS	28	M	76	108	121	90	420	212	473	-60°	

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9	V	27	M	54	96	156	↑128	446	201	422	-48° - Lt	Sinus Bradyc, RBBB, LAD
10	VR	21	M	73	↑121	153	88	378	211	417	40°	
11	PA	24	M	70	117	170	97	397	197	429	72°	
12	JR	27	M	67	↑130	↑204	92	420	265	443	60°	1° AV Block
13	SA	25	M	61	120	152	95	417	237	422	55°	
14	PI	24	M	66	↑141	188	106	424	221	445	-75° - Lt	
15	SFR	23	M	60	106	124	87	425	206	425	-10°	
16	PRR	25	M	69	100	148	83	403	199	433	-22° - Lt	
17	SW	23	M	73	↑141	181	↑136	391	203	432	110° Rt	Incomplete RBBB
18	MH	17	M	67	↑130	177	100	397	265	420	-36°	
19	AY	20	M	54	96	156	↑128	446	204	422	-45° - Lt	Sinus Bradycardia
20	GV	21	M	63	104	132	80	401	196	411	0°	
21	PR	16	M	78	108	132	76	348	208	397	-10°	
22	MS	26	M	63	↑121	174	103	390	222	400	25°	
23	Ps	27	M	45	111	168	109	418	219	362	12°	Sinus Bradycardia
24	PSa	23	M	55	↑121	144	112	411	215	394	28°	Sinus Bradycardia
25	PM	21	M	69	↑124	181	103	391	240	419	65°	
26	BP	24	M	59	108	132	68	408	214	405	10°	Sinus Bradycardia
27	TV	26	M	49	↑129	152	98	407	221	388	26°	Sinus Bradycardia
28	UA	23	M	65	↑140	172	↑136	414	231	459	-45° - Lt	Lt Ventricular Hypertro
29	MF	28	M	81	109	132	94	378	219	439	10°	
30	KV	20	M	59	↑161	↑201	↑125	448	226	443	120°	Sinus Bradycardia
31	SS	26	M	62	↑126	141	104	400	229	404	18°	
32	PS	25	M	48	113	168	↑131	469	209	374	30°	Sinus Bradycardia
33	JK	24	M	82	102	126	94	388	216	454	-50° - Lt	
34	Sm	24	M	63	↑152	187	97	412	225	423	-20°	
35	KV	25	M	62	98	121	83	391	201	398	0°	
36	R	21	M	52	93	109	↑121	378	206	405	18°	Sinus Bradycardia

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37	RS	19	M	68	112	131	91	371	209	395	22°	
38	Pk	21	M	72	106	128	88	378	216	415	10°	
39	TR	22	M	76	111	136	92	385	221	434	-12°	
40	BS	23	M	68	103	132	86	401	208	427	0°	
41	BP	23	F	70	111	154	87	378	211	408	10°	
42	TB	24	F	73	106	121	96	404	221	446	-36° - Lt	
43	SY	22	F	74	112	128	89	367	203	408	28°	
44	PP	23	F	80	98	123	83	363	208	419	18°	
45	SJ	19	F	81	101	121	89	374	224	435	-20°	
46	JS	23	F	68	107	121	98	391	219	416	12°	
47	CJ	21	F	76	112	136	101	386	231	434	0°	
48	M	20	F	80	111	128	93	344	219	397	20°	
49	SL	19	F	81	104	126	89	364	216	423	-15°	
50	KP	24	F	59	101	116	91	408	221	405	28°	Sinus Bradycardia

Table 1: Master sheet Athletes ECG Values

SL. No.	Name	Age	Sex	HR	P Durat	Intervals			T Durat	QTC	QRS Axis	Remarks
						PR	QRS	QT				
1	P	21	M	82	111	139	89	331	203	387	36°	
2	S	25	M	101	108	138	90	327	162	424	30°	Sinus Tachycar
3	A	25	M	83	116	141	90	339	208	399	-45° - Lt	
4	S	23	M	73	90	131	77	362	201	399	40°	
5	R	28	M	93	115	134	95	338	215	421	-6° - Lt	
6	G	26	M	85	122	157	85	345	204	410	25°	
7	P	25	M	81	108	135	96	349	125	405	20°	
8	K	21	M	67	141	162	1129	391	217	413	110° - Rt	RBBB
9	S	25	M	87	120	159	88	340	201	388	35°	
10	P	20	M	86	118	153	91	377	221	421	25°	
11	J	17	M	72	98	128	81	361	203	396	45°	
12	S	16	M	74	115	140	96	375	226	417	48°	
13	P	21	M	86	113	142	98	334	216	400	45°	
14	P	24	M	80	107	136	96	354	208	409	30°	
15	C	23	M	68	111	139	86	331	202	352	60°	
16	R	20	M	78	119	146	92	341	210	389	30°	
17	S	21	M	72	108	144	91	389	232	426	22°	
18	M	21	M	75	117	140	100	371	201	415	55°	

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19	K	23	M	82	106	129	80	351	206	410	45°	
20	V	28	M	73	101	132	69	387	192	405	60°	
21	K	25	M	90	110	141	91	329	222	403	15°	
22	C	16	M	89	108	139	91	331	216	403	45°	
23	G	19	M	80	109	121	84	361	206	417	30°	
24	C	19	M	73	121	154	96	352	221	388	26°	
25	G	23	M	81	110	131	94	361	221	420	42°	
26	K	26	M	80	113	148	96	348	221	402	48°	
27	E	20	M	71	118	142	91	334	208	363	35°	
28	V	22	M	78	116	140	88	346	217	395	45°	
29	K	21	M	76	108	131	90	368	212	414	20°	
30	D	25	M	68	112	139	96	363	208	386	40°	
31	R	20	M	80	106	121	81	341	210	394	15°	
32	S	23	M	78	116	136	90	339	212	387	-10°	
33	A	23	M	84	101	128	90	336	211	398	28°	
34	N	17	M	78	102	124	88	363	206	414	75°	
35	P	19	M	72	98	141	81	385	212	422	60°	
36	S	26	M	62	112	138	84	390	208	395	30°	
37	S	26	M	78	110	149	98	378	221	410	48°	
38	J	26	M	73	112	144	78	361	216	398	64°	
39	P	25	M	72	116	152	92	345	208	378	46°	
40	S	26	M	88	112	142	90	324	202	393	52°	
41	K	24	F	89	108	150	84	321	208	391	50°	
42	G	18	F	84	106	136	80	329	205	389	10°	
43	T	20	F	91	110	153	80	328	201	404	-22°	
44	S	25	F	82	114	150	91	342	208	400	50°	
45	K	22	F	81	118	148	89	349	216	406	45°	
46	V	19	F	70	107	131	86	352	215	380	56°	
47	M	18	F	76	108	128	91	346	220	389	18°	
48	K	21	F	84	111	132	93	332	218	393	12°	
49	A	24	F	80	106	124	88	361	212	417	72°	
50	S	21	F	86	102	120	91	330	203	395	35°	

Table 2: Master sheet NON Athletes ECG Values

Figure 1: Athletes **Figure 2: Non-Athletes**

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Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Figure 8

Figure 9

Figure 10

Figure 11

DISCUSSION: The interpretation of the athlete's ECG is often left to personal experience and usually made according to traditional ECG criteria used in the general (non-athletic) population.

Electrocardiogram changes in athletes are common and usually reflect structural and electrical remodeling of the heart as an adaptation to regular physical training (athlete's heart).

IN THIS STUDY: Among the Athletes, 26% had Sinus bradycardia, none with tachycardia, Average Heart rate in this group was 66 beats/min.

Among the Non-Athletic group 98%(49/50 subjects) showed normal Heart Rate and just 2% (1 subject) showed tachycardia changes.

Bradycardia is the result of physiological adaptive changes of the autonomic nervous system and reflects the level of athletic conditioning. Only profound sinus bradycardia and/or marked sinus arrhythmia (heart rate < 30 beats/min and or pauses 3sec during wake hours) need to be distinguished from sinus node disease. Sino atrial node dysfunction can be reasonably excluded by demonstrating that: (i) Symptoms such as dizziness or syncope are absent;(ii)Heart rate normalizes during exercise, sympathetic maneuvers or drugs, with preservation of maximal heart rate, and (iii)

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Bradycardia reverses with training reduction or discontinuation. But few old athletes tend to complain of decreased heart rate might continue all their life even after they discontinue active sport and continue to work up at the least exercise levels to maintain standard of life. Few trainers now who were long standing athletes have complains of some discomfort if exercise levels are lowered with age building up with them. One of this being Sports Medicine Specialist and a boxer himself and a participant in national boxing championship 30 years back has an everlasting complaint of dizziness/syncopal attacks when resting or out of sports activity for more than 2 or 3 days, he retains his normal healthy status after he begins to exercise getting back to his practice.

D'Souza A¹ et al supported in their study that training-induced bradycardia is not a consequence of changes in the activity of the autonomic nervous system but is caused by intrinsic electrophysiological changes in the sinus node so concluded Exercise training reduces resting heart rate via down regulation of the funny channel HCN4.

Farahani B⁴ et al in their study, Prevalence of different electrocardiographic patterns in Iranian athletes, explored the abnormalities in Iranian athletes electrocardiogram to find any relation with body fat. 239 international athletes were involved in this cross sectional study. Body-fat percentage and resting 12-lead ECGs were recorded from all participants. Of 239 participant athletes, 212 were male and 27 female. 60% of participants had sinus bradycardia.

ATRIO-VENTRICULAR BLOCK: First-degree AV block and Mobitz Type-I (Wenkebach) second degree AV block are common in trained athletes, and 2% (1/50) of athlete's ECGs respectively. As with sinus bradycardia, AV conduction slowing and block are mediated by increased parasympathetic tone or by decreased sympathetic tone. A very rare finding of Second-degree Mobitz Type-II and Type-III degree heart block in athletes to confirm that's it is due to cardiac adaptation activity.

Doutreleau S² et al in their study of Exercise-induced second-degree atrioventricular block in endurance athletes.

Training induces volume- and time-dependent morphological and functional changes in the heart. Heart rhythm disorders, such as atrial arrhythmia (including atrial fibrillation and atrial flutter), are a well-established consequence of such long-term endurance practice. Although resting bradycardia and first-degree atrioventricular block persist in veteran athletes, higher conduction system impairment has never been reported neither at rest nor during exercise.

ISOLATED INCREASE OF QRS VOLTAGES: Intensive athletic activity causing morphological cardiac changes, including increased cavity dimensions, wall thickness and ventricular mass which is reflected in the 12-lead ECG. Physiological LV hypertrophy in trained athletes usually manifests as an isolated increase of QRS amplitude, with normal QRS axis normal atrial and ventricular activation patterns. Criteria for LV hypertrophy has been reported in trained athletes as its noticed here in one of the subject, which has increased QRS voltage and diagnosed to have LVH.

These subjects have been correlated ECG changes with cardiac morphology assessed by echocardiography. The ECG was labeled as 'abnormal' in (40%) athletes; however an isolated increase of QRS voltage accounted for 14% (7/50) of such changes. No athletes with isolated increase of QRS voltages had evidence of structural heart disease such as HOCM (Hypertrophic Obstructive Cardio Myopathy) in this workup but rather many athletes had been discovered to have diagnosed with HOCM.

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Athletes who show pure QRS voltage criteria for LV hypertrophy on 12-lead ECG do not require systematic echocardiographic evaluation, unless they have relevant symptoms, a family history of cardiovascular diseases and or sudden cardiac death (SCD) or non-voltage ECG criteria suggesting any pathological LV hypertrophy.

Pelliccia A³ et al in their study showed that highly trained athletes show morphologic cardiac changes (i.e, athlete's heart) that are the consequence of several determinants, including type of sport, gender, and, possibly, inherited genetic factors. The extent of physiologic cardiac remodeling may occasionally be substantial in highly trained athletes and may raise a differential diagnosis with structural cardiac disease, such as cardiomyopathies. In addition, athletes demonstrate a spectrum of alterations in the 12-lead electrocardiogram (ECG) pattern, including marked increase in precordial R-wave or S-wave voltages, ST segment or T-wave changes, and deep Q waves suggestive of left ventricular hypertrophy, that may raise the possibility of pathologic heart condition, but have also been viewed as a consequence of the cardiac morphologic remodeling induced by athletic conditioning. To evaluate the clinical significance of these abnormal ECGs, the authors compared ECG patterns to cardiac morphology and function (assessed by two-dimensional echocardiography in individual athlete) in a large population of 1005 elite athletes engaged in a variety of sporting disciplines. Forty percent of the athletes had abnormal ECGs, and a subgroup of about 15% showed distinctly abnormal and often bizarre patterns highly suggestive of cardiomyopathies, such as hypertrophic cardiomyopathy, in the absence of pathologic cardiac changes. Such alterations are likely the consequence of athletic conditioning itself and represent another potential component of athlete's heart syndrome.

INCOMPLETE RIGHT BUNDLE BRANCH BLOCK: The Prevalence of incomplete right bundle block (RBBB) (QRS duration 120 ms) has been estimated to range from 4% in athletes compared with less than 2% in young sedentary controls. This ECG pattern is more often noted in athletes engaged in endurance sports. It has been suggested that the right ventricular (RV) conduction delay is not within the specialized conduction system, but is caused by the enlarged RV cavity size /Increased cardiac muscle mass and the resultant increased conduction time. The RBBB morphology has been shown to be reversible with deconditioning.

EARLY REPOLARIZATION: Early repolarization has traditionally been regarded as an idiopathic can be benign ECG phenomenon, with an estimated prevalence in healthy young individuals of 1–2%, and a clear male preponderance. The early repolarization ECG pattern is the rule rather than the exception among highly trained athletes, in whom it is observed in 50–80% of resting ECGs. The early repolarization ECG shows elevation of the QRS–ST junction (J-point) of at least 0.1mV from the baseline, associated with notching or slurring of the terminal QRS complex which may vary in location, morphology and degree.

Slowing of heart rate exaggerates ST-segment elevation, whereas sinus tachycardia occurring during exercise. Early repolarization in athletes reflects the development of a training related hypervagotonia, and ECG abnormalities are reversible phenomenon which reduces or disappears with deconditioning.

Early repolarization is a physiological and benign ECG pattern in the general population of younger people and athletes and does not require further evaluation.

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But none of the ECG's in athletes showed any early repolarization changes.

ECG abnormalities with the cardiovascular diseases include repolarization abnormalities such as inverted T-waves and ST-segment depression, pathological Q wave conduction disease including left-axis deviation, ventricular pre-excitation, long-and short QT interval and Brugada-like repolarization changes.

Unlike the ECG changes characteristic of athlete's heart, such ECG abnormalities are relatively uncommon (5%) and training-unrelated. Further diagnostic work-up is mandatory for those athletes who exhibit such ECG changes in order to confirm (or exclude) an underlying cardiovascular disease.

De Asmundis C⁵ et al in their study of Prevalence and electrocardiographic characteristics of early repolarization pattern (ERP) in young teen athletes, Of the 122 included subjects, The overall prevalence of ERP in their study population was 36%, Interestingly, incomplete right bundle-branch block (IRBBB) was significantly lower among all subjects with ERP (N = 15; 34.1%), compared to those without ERP (N = 49; 63%) (P < 0.002). And henceforth concluded that ERP is a common finding in young teen athletes.

LEFT VENTRICULAR HYPERTROPHY: Electrocardiogram offers the potential to distinguish between pathological and physiological hypertrophy, given that ECG abnormalities of structural heart diseases manifesting with LV hypertrophy, such as cardiomyopathies, valve diseases, or hypertensive heart disease overlap only marginally with training-related ECG changes.

One of the Athletes namely subject number 28 has shown changes of LVH in ECG as:

- Markedly increased LV voltages: Huge precordial R and S waves that overlap with the adjacent leads (SV2 + RV6 >> 35 mm).
- R-wave peak time > 50 ms in V5-6 with associated QRS broadening.
- LV strain pattern with ST depression and T-wave inversions in I, aVL and V5-6.
- ST elevation in V1-3.
- Prominent U waves in V1-3.
- Left axis deviation.

Diagnosed as Sinus Rhythm with LVH as this subject was send for further investigations ECHO test, a procedure of choice for LVH, showed changes as concentric Left Ventricular Hypertrophy, Ejection Fraction is 70%, and no systolic dysfunction seen. So likewise an athletes heart is a growing extra thick as if it is under constant stress and need extra thickened muscle to provide the strength to pump out blood sufficiently to the peripheral parts, so this is usually a life style effect in athletes and most of the times not life threatening.

Arbab-Zadeh A⁶ et al.Cardiac Remodeling in Response to 1 year of Intensive Endurance training One year of prolonged and intensive endurance training leads to cardiac morphologic adaptations in previously sedentary young subjects similar to those observed in elite endurance athletes; however it is not sufficient to achieve similar levels of cardiac compliance and performance. Contrary to conventional thinking, the left ventricle responds to exercise with initial concentric not eccentric remodeling during the first 6-9 months after commencement of endurance training depending on the duration and intensity of exercise. Thereafter, the left ventricle dilates and restores the baseline mass to volume ratio. In contrast, the right ventricle responds to endurance training with eccentric remodeling at all levels of training.

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Caselli S⁷et al Differentiating left ventricular hypertrophy in athletes from that in patients with hypertrophic cardiomyopathy.

Aimed in the study was to revise the ability of simple echocardiographic and clinical variables for the differential diagnosis of HC versus athlete's heart. Twenty-eight athletes free of cardiovascular disease were compared with 25 untrained patients with HC, matched for LV wall thickness (13 to 15 mm), age, and gender. Clinical, electrocardiographic, and echocardiographic variables were compared. Athletes had larger LV cavities (60 ± 3 vs 45 ± 5 mm, $p < 0.001$), aortic roots (34 ± 3 vs 30 ± 3 mm, $p < 0.001$), and left atria (42 ± 4 vs 33 ± 5 mm, $p < 0.001$) than patients with HC. LV cavity < 54 mm distinguished HC from athlete's heart with the highest sensitivity and specificity (both 100%, $p < 0.001$). Left atrium > 40 mm excluded HC with sensitivity of 92% and specificity of 71% ($p < 0.001$).

ST SEGMENT DEPRESSION: Although ST segment elevation due to early repolarization is a common finding in the basal ECG of trained athletes, resting ST segment depression is rarely observed, none of the subjects showed any changes in ST segment.

RIGHT ATRIAL ENLARGEMENT AND RIGHT VENTRICULAR HYPERTROPHY: ECG evidence of right atrial enlargement and/ or RV hypertrophy is uncommon finding in athletes. The presence of either congenital or acquired heart diseases associated with an increased right atrial size and/or pathological RV dilatation/ hypertrophy should be excluded.

T-WAVE INVERSION: Recent studies on large athletic populations disproved the traditional idea that T-wave inversions are common and training-related ECG changes in the athlete.

T-wave inversion in young and apparently healthy athletes may represent the initial phenotypic expression of an underlying cardiomyopathy prior to the development of morphological changes detectable on cardiac imaging.

T-wave inversions 2 mm in two or more adjacent leads is rarely observed on the ECG of healthy athletes, where as it is a common finding in patients with cardiomyopathy and other cardiac disease.

VENTRICULAR PRE-EXCITATION (WOLFF- PARKINSON-WHITE): None of the subjects were found to have any WPW abnormalities.

They prevalence of ventricular pre-excitation in the general population varies from 0.1% to 0.3% and is similar in athletes. Most individuals with WPW pre-excitations syndrome remain asymptomatic throughout their lives. When symptoms do occur, they are usually secondary to reciprocating supraventricular tachycardia which causes disabling symptoms.

LONG-QT INTERVAL: The duration of the QT interval(i.e.the interval between the beginning of the QRS complex and the end of the T-wave) changes with heart rate and it is usually corrected (QTc) by using,

<p>Bazett's formula $QT_B = \frac{QT}{\sqrt{RR}}$</p>

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QT INTERVAL: Normal QT is (0.36 – 0.44 s) equal to or below 0.40s. (400msec).

Among athletes 50% (25/50) had prolonged QT interval. Average QT interval in this group being 401 msec, which has over leaped the expected range of 400 msec.

Among the Non-Athletes nobody had any prolonged or decreased QT interval, in this group average QT intervals being 351msec.

The QT interval should be measured in L2,V3, or V5;the longest value (return to baseline)should be considered.

The calculation of the QT interval in athletes has inherent limitations due to sinus arrhythmia, slightly widened QRS complexes and T-U complexes. Corrections for heart rate may be inaccurate at heart rates below 40and above 120 bpm.

QT interval is modulated by gender and therefore different cut off values are used after puberty. A QTc interval is traditionally considered long in men when it is more than 440 ms and in women when it exceeds 460 ms, but some suggested values upto and above 470 ms in males and 480 ms in female.

In general QT interval is longer in athletes than in non-athletic control because of the lower resting heart rate associated with athletic training, and this workup has demonstrated the average QT interval in Indian athletes is 401ms, whereas average QT interval in Non-athletes is 351ms, while the QTc of the athletic group is within normal limits, though toward the upper limit.

Recording of QTc intervals beyond the normal cut-off values should raise the suspicion of either acquired or congenital long QT syndrome (LQTS).

Omiya K⁸ et al Influence of gender and types of sports training on QT variables in young elite athletes. Influence of gender and sports training on QT variables such as QT interval and dispersion (QT dispersion: QTD) in young elite athletes were evaluated.

Concluded Maximum and minimum QTc were significantly longer in female athletes than in male athletes (max: 414.2 vs. 404.5 ms, min: 375.1 vs. 359.2 ms, $p < 0.0001$ respectively), whereas QTc dispersion (QTcD) was shorter in female athletes than in male athletes (39.2 vs. 45.3 ms, $p < 0.0001$). QTcD was significantly shorter in female athletes than in the female control group (39.2 vs. 45.2 ms, $p < 0.05$). However, no statistically significant difference was observed between male athletes and the male control group. Male gymnasts exhibited significantly longer QTcD than the control group ($p < 0.01$), but female gymnasts had significantly shorter QTcD than the control group ($p < 0.05$). Maximum QTc intervals were prolonged in the male static training group compared with non-athletes, and QTcDs in the static training group were prolonged compared with the endurance training group. However, no significant difference was observed in the female group. In conclusion, both gender and different characteristics of sports training may affect QT variables even in young elite athletes. Vigorous static exercise training may independently prolong QT variables.

Griffet V⁹ et al in their workup of Athlete's heart in the young, while recording the ECG and echocardiographic patterns in 107 French athletes and hence concluded Adolescent athlete's heart is normal. If QTc interval is not normal, be afraid of a QT long syndrome. Furthermore, when interventricular septal thickness is > 11 mm or left ventricular end diastolic diameter > 55 mm, myocardiopathy will have to be ruled out.

CONCLUSION: Now after determining ECG changes which are physiological (common and training-related ECG abnormalities) and that which are pathological (uncommon and training-unrelated ECG

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abnormalities) is expected to reduce the traditional high number of false positives, so wise reducing the investigations.¹¹

In this study, the ECGs were classified into three subgroups, as distinctly abnormal, mildly abnormal, and normal (or with minor alterations).

Abnormal ECG was identified in (6%) athletes of which distinctly abnormal in 2% (1/50), and mildly abnormal in 4% (2/50).

In the remaining 94% athletes (47/50), ECGs were normal in or exhibited only minor alterations, which were deemed typical of the athletes heart, of the 3 athletes with ECG abnormalities previously classified as abnormal and suggestive of cardiac disease, 1 case exhibited an isolated increase of QRS voltage.

But this workup will be helpful in making certain rules in our country to bring up the compulsory pre-participation screening program for upcoming athletes in schools (while on the go certain sports schools have already taken up this screening program voluntarily) and to prevent and early diagnosis of cardiac diseases.

Analyzed ECG changes	Number of patients, n (%)
Sinus bradycardia < 60/min	13 (26%)
Severe sinus bradycardia ≤ 40/min	0(0%)
Prolonged QT interval	2(4%)
Prolonged QTc interval as per Bazett's	0(0%)
Ventricular Arrhythmias	0(0%)
Shortened PR interval < 120 ms	0(0%)
Prolonged PR Interval > 200 ms	2(4%)
Left bundle branch block	0(0%)
Right bundle branch block	2(4%)
Heart Axis Deviation QRS > -30°	39(78%)
Right Axis Deviation > +90°	2(4%)
Left Axis Deviation < -30°	9(18%)
Incomplete RBBB	1(2%)
Early Repolarization	0(0%)
Isolated Voltage for Left Ventricular Hypertrophy	1(2%)
Features of left atrial hypertrophy	0(0%)
T Wave inversions(negative T wave)	0(0%)
R wave in V5 or V6 > 3.0 mV	1(2%)
S wave in V1 or V2 > 3.0 mV	1(2%)
Table 3: Changes in the ECG of 50 Athletes	

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Date of Submission: 19/12/2014.
Date of Peer Review: 20/12/2014.
Date of Acceptance: 02/01/2015.
Date of Publishing: 10/01/2015.