J Wave, QRS Slurring, and ST Elevation in Athletes With Cardiac Arrest in the Absence of Heart Disease

Marker of Risk or Innocent Bystander?

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Background—QRS-ST changes in the inferior and lateral ECG leads are frequently observed in athletes. Recent studies have suggested a potential arrhythmogenic significance of these findings in the general population. The aim of our study was to investigate whether QRS-ST changes are markers of cardiac arrest (CA) of unexplained cause or sudden death in athletes.

Methods and Results—In 21 athletes (mean age, 27 years; 5 women) with cardiac arrest or sudden death, the ECG recorded before or immediately after the clinical event was compared with the ECG of 365 healthy athletes eligible for competitive sport activity. We measured the height of the J wave and ST elevation and searched for the presence of QRS slurring in the terminal portion of QRS. QRS slurring in any lead was present in 28.6% of cases and in 7.6% of control athletes ($P=0.006$). A J wave and/or QRS slurring without ST elevation in the inferior (II, III, and aVF) and lateral leads (V_4 to V_6) were more frequently recorded in cases than in control athletes (28.6% versus 7.9%, $P=0.007$). Among those with cardiac arrest, arrhythmia recurrences did not differ between the subgroups with and without J wave or QRS slurring during a median 36-month follow-up of sport discontinuation.

Conclusions—J wave and/or QRS slurring was found more frequently among athletes with cardiac arrest/sudden death than in control athletes. Nevertheless, the presence of this ECG pattern appears not to confer a higher risk for recurrent malignant ventricular arrhythmias. **(*Circ Arrhythm Electrophysiol*. 2010;3:305-311.)**

Key Words: cardiac arrest ■ sudden death ■ early repolarization ■ athletes

The presence of QRS-ST changes in inferior and lateral ECG leads is a common finding in the general population (2% to 10%)[1–5] and is even more frequent in trained athletes, particularly those engaged in endurance disciplines (such as cycling, rowing, or marathon running).[6–10] In young and healthy individuals, this condition has commonly been considered to represent an innocent finding. However, experimental studies,[2,11–13] case reports,[14–17] and recent studies on healthy subjects surviving a cardiac arrest[18] or with primary ventricular fibrillation (VF)[9,19,20] have suggested an association between J-point elevation and/or QRS slurring in the inferior and lateral ECG leads and the risk of VF.

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In athletes, changes of the QRS-ST segment may occur as a consequence of physiological resetting of the balance between the sympathetic and parasympathetic tone, ultimately regulating transmembrane ionic currents.[2,21] Whether such changes may be arrhythmogenic and ultimately lead to VF has not been systematically investigated. The aim of the present study was to assess whether QRS-ST–segment changes observed at baseline ECG may represent a marker of risk for cardiac arrest (CA) or sudden death (SD) in competitive athletes in the absence of heart disease.

Methods

Study Population

We reviewed the database of 86 athletes who had a resuscitated CA or SD caused by cardiovascular causes between 1982 and 2007 and who were referred to our Sport Center Unit in San Donato, Milan, or to Villa Bianca, Trento, for diagnostic and therapeutic purposes or for legal advise. Of the 86 athletes, 65 showed some evidence of underlying cardiac disease potentially responsible for CA. The

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Table 1. Demographics and Clinical Findings in the 21 Athletes With CA/SD Without Heart Disease

	J Wave/QRS Slurring (n=10)	No J Wave/QRS Slurring (n=11)	*P* Value
Male sex, no. (%)	8 (80)	8 (72)	0.7
Age, y, mean±SD	25.5±5	28.9±1	0.34
Confirmation of idiopathic VF			
Coronary angiogram, No.	5	6	
MRI, No.	6	7	
Sport			
Endurance-trained	10	10	
Weight lifter	0	1	
Level: International/national	5	4	
Level: Regional	5	7	
Duration, h/wk, mean±SD	16.8±4	15.5±5	0.4
Resuscitated CA	10	9	
SD	0	2	
Activity at time of CA/SD			
During effort	7	5	
On standby during game	1	1	
On standby after effort	2	3	
Rest	0	1	
Sleeping	0	1	
ECG after CA, mean±SD			
PR, ms	159±16	167±10	0.18
Heart rate, bpm	57±5	61±7	0.11
QRS duration	89±6	86±10	0.32
QTc, Bazett formula	405±19	415±22	0.28
QTc, Fridericia formula	409±19	415±17	0.45
SAECG (8 cases)			
Presence of late potentials	2/4	¾	0.46
Holter recording (19 cases)			
Nonsustained VT	6/9	5/10	0.43
Exercise test (19 cases)			
Complex VPB	5/10	3/9	0.46
Nonsustained VT	2/10	4/9	0.26
EPS (17 cases)			
Inducibility of VT-VF	4/8	6/9	0.49
Echo wall thickness, mm, mean±SD			
LVWTh	9.6±1.2	10.2±1.4	0.55
LVSTh	10±1.7	10.1±1.4	0.89

SAECG indicates signal-averaging ECG; VPB, ventricular premature beats; EPS, electrophysiological study; LVWTh, left ventricular wall thickness; and LVSTh, left ventricular septal thickness.

remaining 21 athletes (mean age, 27±8 years; range, 14 to 43 years; 16 men) had no evidence of either structural heart disease, Brugada syndrome, preexcitation, or long or short QT on the basis of clinical, ECG, and echocardiographic data and represent our study population. Their demographic and clinical characteristics are presented in Table 1.

Of the 21 athletes, 19 had CA and 2 had SD. All the 19 subjects with CA were resuscitated by means of on-site or early defibrillation. In each of the 19 athletes, the circumstances of CA/SD, family history of unexplained SD, symptoms preceding the event (declared by victims or collected by witnesses), sporting discipline, and level of athletic achievement were recorded. In each, diagnostic testing performed before the event, including ECG, echocardiographic, exercise testing, 24-hour Holter monitoring, and electrophysiological data, were collected when available.

Control Group

The control group consisted of 365 athletes (mean age, 28±8 years; 36 women) who underwent preparticipation screening and were considered healthy and eligible for competitive sport activity. Each of the control subjects had no history of syncope or heart disease, showed normal baseline ECG, and had no evidence of heart disease on clinical and echocardiographic examination.

ECG Analysis

Of the study group, ECGs analyzed were those recorded before the event (available in the 2 subjects with SD and in 9 subjects with resuscitated CA) and ECGs recorded after the event (available in all 19 with CA). Access to preevent ECG analysis in 10 subjects with CA was not possible because of unavailability of athletes' files at referral center years after the original recording. Of the control group, ECGs recorded at the time of preparticipation screening were analyzed.

The following QRS-ST changes were analyzed: J-point elevation (J wave), slurred QRS complex, and ST-segment elevation. J waves were defined as positive "humplike" deflections immediately after a positive QRS complex at the onset of the ST segment; slurred QRS complex was defined as a gradual transition from the QRS complex to the ST segment; ST-segment elevation was measured at its most horizontal section. The height of the J point and/or ST-segment elevation from the baseline was measured with a caliper under a magnifying glass providing ×2 magnification. To consider athletes as carriers of QRS-ST changes, we adopted the following criteria: elevation of the QRS-ST junction >0.05 mV above the baseline level, either as J wave or QRS slurring, with or without ST-segment elevation >0.05 mV above the baseline level in the inferior (II, III, and aVF), lateral (I, aVL, and V_4 to V_6), or inferolateral leads (II, III, aVF, and V_4 to V_6). PR interval, QRS duration, and QTc (QT interval corrected with the Bazett formula and with the Fridericia formula) were also measured in both groups. All ECGs were analyzed by 2 independent cardiologists, and controversial interpretations were rediscussed with a third observer. Grading was by consensus. Variability of QRS-ST changes was investigated by means of serial ECG recording in the 9 case patients with availability of preevent ECGs and in 115 control athletes in the control group, of which 56 presented with and 59 without QRS-ST changes at baseline ECG. Changes below the 0.05-mV threshold value of the QRS-ST segment observed in subsequent ECGs from the same subject were judged as small variations.

Other Testing

In addition, ECG exercise testing (in 19), 24-hour Holter monitoring (in 19), coronary angiography (in 11), cardiac magnetic resonance (in 9), and signal-averaging ECG (in 8) were performed in athletes with resuscitated CA. In 17 of 19 cases, the electrophysiological study was performed: A maximum of 2 or 3 extrastimuli were delivered from 2 ventricular sites. Patients with VF, polymorphic ventricular tachycardia (VT) lasting >30 seconds, or monomorphic fast VT (>240 bpm) requiring intervention for termination were classified as inducible.

Follow-Up

Survivors of CA were followed up for a median of 36 months (interquartile range, 31 to 119), during which all ECGs available were collected and analyzed, and a new one was recorded in each subject at the end of follow-up. In addition, information was recorded relative to the level of training (or detraining) and use of antiarrhythmic drugs or implantable cardioverter-defibrillator (ICD).

The recurrence of ventricular arrhythmias in the study group during follow-up was recorded by means of careful subject history, Holter recording, ICD telemetry, and clinical files, as applicable.

Statistical Analysis

When designing the study, it was estimated that a sample size of 350 control subjects would provide adequate statistical power to detect predictors of CA or SD in the case subject group. Descriptive analysis was performed using mean±SD values for continuous variables and percentage values for noncontinuous variables. Comparisons between groups were performed with the Student *t* test and Pearson χ^2 when appropriate. Results are displayed as the odds ratio plus 95% confidence intervals. All probability values were considered significant at a value of 0.05. All these statistical calculations were done using a computerized statistical package (SPSS 13.0, Chicago, Ill). The propensity score analysis was performed using SAS software package (SAS/STAT User's Guide, Version 8e. 1989, SAS Institute Inc, Cary, NC).

To put the present results into clinical perspective, we used, as proposed by Rosso et al,[9] the Bayes theorem[22] to determine the conditional probability of having CA or SD, when J-point elevation or slurring was detected, with the following formula: Pj (CA/SD)=P (CA/SD)×Pca/sd (J)/P (CA/SD)×Pca/sd (J)+P (not CA/SD)×P not ca/sd (J), where Pj (CA/SD) is the probability of having idiopathic CA or SD on the basis of a documented or presumed VF episode, when J-point elevation (J wave and/or QRS slurring) is present. The parameters entered into the formula Pca/sd (J) and P not ca/sd (J), expressing the probability of having a J-point elevation for an athlete with CA or SD and for an athlete without CA or SD, respectively, were derived from the results of the present study; P (CA/SD), the incidence of CA or SD in the athlete population, was entered as 0.2/100 000. This figure was based on epidemiological data showing that the risk of SD for the athlete population is 2.1 of 100 000[23] and assuming that a similar number of resuscitated CAs would occur within the same time frame. Moreover, we hypothesized that about 5% of CA/SD events probably occur in absence of structural heart disease. Finally, P (not CA/SD), representing the incidence of athletes without CA/SD, was approximated to 1.

Results

Of the 10 athletes with CA and early repolarization on 12-lead ECG, 7 collapsed during effort, 1 on standby during a soccer game and 2 about 30 minutes after termination of effort. In the case group (9 subjects) without early repolarization on 12-lead ECG, 5 athletes collapsed during effort, 3 on standby during (1 patient) or after effort (2 patients), and 1 at rest (Table 1). All patients were resuscitated without long-term neurological impairment. Two other subjects had SD, 1 about 15 minutes after a training session and 1 during sleep. The majority of the events, that is, 16 of 21, were not preceded by symptoms. Of the remaining 5 athletes, 3 had 1 or more syncope events during physical activity a few months before CA occurred, which had remained unexplained and without documentation of arrhythmia. Two other subjects reported short episodes of palpitations after exercise, associated with premature ventricular beats.

ECG Analysis

Heart rate was lower in the group of competitive athletes with CA/SD than in control athletes (60±7 versus 66±10 bpm, $P=0.02$). PR interval (162±14 versus 159±23 ms) and QRS duration (88±8 versus 86±7 ms) were not statistically different. QTc corrected with the Bazett formula was not statistically different in cases and control subjects (411±21 versus 402±27 ms, $P=0.10$), whereas the QTc corrected with the Fridericia formula was longer in cases than in control subjects, with borderline statistical significance (412±18 versus 398±24, $P=0.05$). However, all QTc values were within the normal variability range.

J Wave and QRS Slurring

Compared with control subjects, athletes with CA/SD more frequently showed a slurred QRS pattern in any leads (28.6% versus 7.6%, $P=0.006$) and a J-point elevation and/or slurred QRS in standard inferior plus precordial V_4 to V_6 leads (28.6% versus 7.9%, $P=0.007$) (Table 2). These differences were confirmed when the case subject population was compared with an age- and sex-matched control population of 63 healthy athletes.

ST-Segment Elevation

The presence of ST-segment elevation was not statistically different in athletes with CA/SD and in control athletes, even though this pattern was much less commonly observed in the group with CA/SD (9.5% versus 21.6%) (Table 3). On the other hand, the presence of J wave and/or QRS slurring without ST-segment elevation was more frequently observed in athletes with CA/SD (Figure, A) than in control athletes (Figure, B) (38.1% versus 15.6%, $P=0.04$), particularly in inferior-lateral leads (23.8% versus 2.5%, $P=0.001$) (Table 3). These differences were confirmed when the case subject population was compared with an age- and sex-matched control population of 63 healthy athletes.

Diagnostic Power of J-Point Elevation

In this study, the probability of having J-point elevation (J wave and/or QRS slurring) was 0.48 in CA/SD athletes and 0.29 for control athletes. According to the Bayes formula of conditional probabilities,[22] finding a J wave and/or QRS slurring would increase the probability of having CA/SD from approximately 2 per million to 3.5 per million athletes.

Other Diagnostic Testing

Within the study group of 21 athletes with CA/SD, only 5 subjects (2 with and 3 without QRS-ST changes) showed the presence of positive ventricular late potentials. On exercise testing, frequent ventricular ectopic beats or runs of nonsustained VT were induced in 14 (7 with QRS-ST changes) of 19 athletes. In no patients were coronary or ischemic abnormalities observed during coronary angiography, cardiac magnetic resonance, or exercise testing. The ECG Holter monitoring showed episodes of nonsustained VT in 6 of 9 cases with and in 5 of 10 cases without QRS-ST changes. The electrophysiological study was performed in 8 of 10 subjects with and 9 of 11 without QRS-ST changes: Sustained polymorphic VT was induced in 4 cases with and in 6 without QRS-ST changes.

Follow-Up

Survivors of CA had serial evaluation over a median of 36 months (interquartile range, 31 to 119). All were strongly recommended to stop regular training and competition, but 3 of 19 were not compliant. All subjects were administered pharmacological treatment: β-blockers,[13] amiodarone,[5]

Table 2. Distribution of QRS Slurring, J wave, and ST-Segment Elevation ≥0.05 mV in Athletes With CA and Control Subjects

	CA Subjects (n=21)		Control Subjects (n=365)					
	N	%	N	%	*P* Value	OR	95% CI	Pps
J wave with/without slurred QRS with/without ST elevation								
Any lead	10	47.6	108	29.5	0.09	2.16	0.89–5.24	0.1
Inferior leads	2	9.5	16	4.4	0.25	2.3	0.49–10.72	0.33
Lateral lead	2	9.5	63	17.2	0.55	0.50	0.11–2.22	0.54
Inferior+V_4 to V_6	6	28.6	29	7.9	0.007	4.63	1.67–12.85	0.007
Slurred QRS								
Any lead	6	28.6	28	7.6	0.006	4.81	1.73–13.38	0.007
Inferior leads	3	14.3	8	2.1	0.017	7.44	1.82–30.43	0.01
Lateral leads	1	4.7	6	1.6	0.32	2.99	0.34–26.05	0.14
Inferior+V_4 to V_6	2	9.5	14	3.8	0.21	2.64	0.56–12.46	0.19
J wave								
Any lead	7	33	101	27.7	0.58	1.3	0.5–3.3	0.56
Inferior leads	2	9.5	12	3.2	0.13	3.4	0.7–16.4	0.11
Lateral lead	4	19	60	16.4	0.65	1.3	0.4–4	0.64
Inferior+V_4 to V_6	1	4.7	29	7.9	0.31	0.49	0.06–3.7	0.21

CI indicates confidence interval; OR, odds ratio; and Pps, *P* value adjusted with propensity score analysis.

propafenone,[2] or diltiazem.[1] Of 19 athletes, 13 were treated with an ICD; 3 refused ICD implantation, and another 3 were treated with antiarrhythmic drugs. The distribution of noncompliant athletes to detraining, type of antiarrhythmic agents, and ICDs did not differ in the 10 athletes with versus the 9 without J wave or slurred QRS. The pattern of J waves or slurred QRS did not disappear during the follow-up in 49 serial ECGs recorded from 8 of 10 athletes with CA and showed small variations in the remaining 2. In the 9 athletes without J wave or slurred QRS on the ECG at time of CA, J wave or slurred QRS was never observed in 34 serial ECGs recorded during the follow-up. Similarly, in the 5 patients showing early repolarization before CA, no changes were observed after CA. In the control group, small variations of QRS-ST were found during follow-up in 14% of athletes with and in 10% without early repolarization findings at baseline ECG.

Recurrence of ventricular arrhythmias was recorded in 14 of 19 case subjects. The incidence of recurrent arrhythmias did not differ significantly in subjects with and without J wave or slurred QRS. Specifically, among 9 athletes without J wave or slurred QRS, 6 presented few episodes of sustained VT (3 during effort) reverted by the ICD intervention (ATP), 2 had episodes of VF interrupted by ICD shock, and 1 had episodes of nonsustained VT. Of 10 athletes with J wave or

Table 3. Distribution of ST-Segment Elevation >0.05 mV in Athletes With CA and 365 Control Subjects

	CA Subjects (n=21)		Control Subjects (n=365)					
	N	%	N	%	*P* Value	OR	95% CI	Pps
ST-segment elevation >0.05 mV								
Any lead	2	9.5	79	21.6	0.27	0.381	0.09–1.67	0.27
Inferior leads	1	4.7	8	2.2	0.40	2.23	0.27–18.72	0.16
Lateral leads	1	4.7	44	12.1	0.49	0.365	0.05–2.79	0.39
Inferior+V_4 to V_6	0	0	27	7.4	0.38	–	–	0.22
J wave with/without slurred QRS no ST elevation								
Any lead	8	38.1	58	15.8	0.04	2.86	1.1–7.43	0.02
Inferior leads	2	9.5	11	3	0.19	–	–	0.36
Lateral lead	1	4.7	38	10.4	0.48	–	–	0.49
Inferior+V_4 to V_6	5	23.8	9	2.4	0.001	10.99	3.25–37.15	0.009

CI indicates confidence interval; OR, odds ratio; and Pps, *P* value adjusted with propensity score analysis.

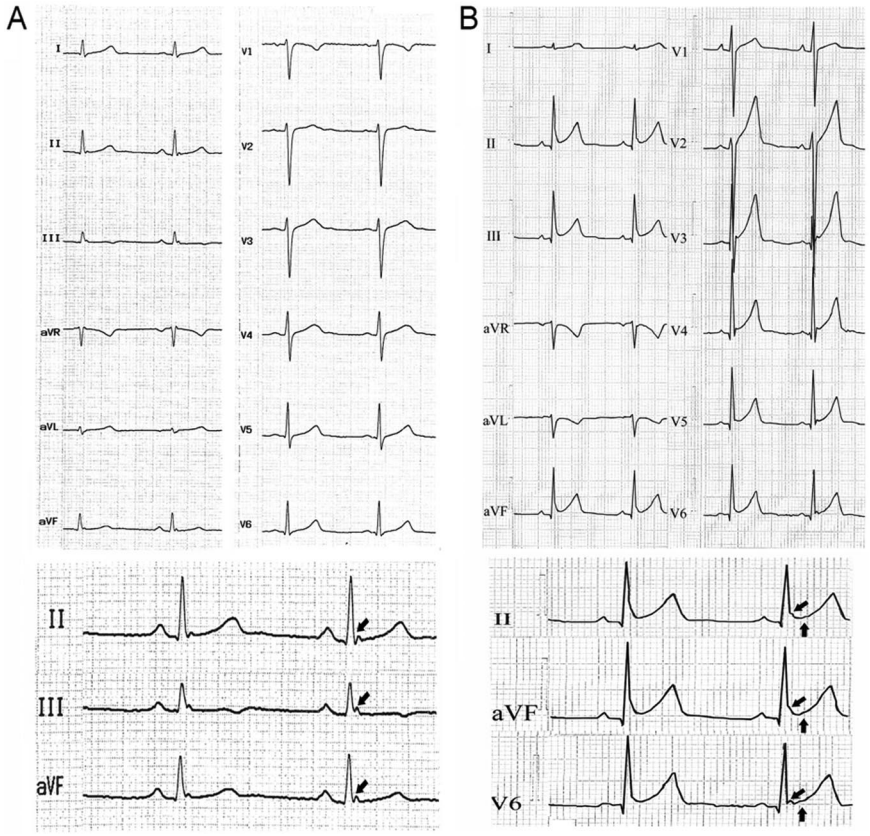

Figure. J wave, QRS slurring, and ST-segment elevation in athletes. A, ECG of a 21-year-old man, a soccer player, who had CA during practice. J waves >0.05 mV are present in leads II, III, and aVF without ST-segment elevation. In the lower panel, tracings are magnified and arrows indicate the J waves. B, ECG of a 29-year-old male cyclist in the control group showing a QRS slurring in leads II, III, and aVF and a J wave in leads V_3 to V_6. ST-segment elevation is evident in leads II, III, aVF, and V_4 to V_6. In the lower panel, tracings are magnified and oblique arrows indicate QRS slurring in leads II and aVF and J wave in V_6; vertical arrows indicate ST-segment elevation.

slurred QRS, 3 had episodes of polymorphic VT and VF, interrupted by the ICD, 2 had sustained VT, and 3 presented polymorphic premature ventricular beats. One athlete with J wave, slurred QRS, and ST-segment elevation who had CA in 1983 during a soccer game died suddenly 10 years later at age 33 years at home. He had refused ICD implantation and was receiving propafenone at the time of death.

Discussion

The pattern of QRS-ST elevation observed in the inferior and lateral ECG leads has been considered for a long time to be an innocent finding in individuals without evidence of CD, despite several case reports[14–17] and recent studies describing the association of this pattern with idiopathic VF in subjects with a structurally normal heart.[9,18–20] In the present study, we sought to determine whether the presence of specific changes of the QRS-ST segment in competitive athletes without structural heart disease may represent a marker of increased arrhythmic risk.

Overall, our study suggests that the presence of QRS slurring or J wave in the absence of ST elevation in the inferior-lateral ECG leads was associated with a marginally increased risk of CA or SD, whereas the presence of ST-segment elevation did not appear to increase such risk. This is in agreement with the hypothesis that J wave and QRS slurring are the body surface expression of enhanced transmural repolarization heterogeneity favoring phase-2 early afterdepolarizations and circum movement reentry tachycardia, as reproduced in arterially perfused ventricular wedge preparation.[2] Our findings are consistent with those reported by Haissaguerre et al[18] in a sedentary population, although the prevalence of QRS-ST changes in our study is higher in both groups of subjects with and without CA, probably because our study population comprised younger competitive athletes. Of note, the prevalence of J wave with or without QRS slurring in our control is similar to that recently reported in a similar population by Rosso et al[9] (22%).

The QRS-ST changes observed in athletes with CA could be the results of myocardial ischemia associated with VF and of resuscitation maneuvers. Nevertheless, this interpretation appears to be unlikely because all 5 athletes with CA and preevent early repolarization did not change their ECG pattern during follow-up. Lack of significant QRS-ST changes during serial ECG recording both in case and control groups suggests that these findings are quite stable.

Despite the larger proportion of J wave and/or QRS slurring in case subjects with cardiac events, our data suggest that these patterns did not confer an additional risk for recurrent ventricular arrhythmias during an intermediate follow-up, as suggested by the similar incidence of recurrent events in CA survivors with and without these repolarization abnormalities. These data differ from those reported by Haissaguerre et al,[18] who described a significant increase in the risk for recurrent ventricular arrhythmias after CA in patients with J wave and/or QRS slurring. A possible explanation for this discrepancy may be related to the precipitating role of strenuous exercise in CA athletes at the time of the index event; elimination of exercise in these patients may have played a protective role against arrhythmia recurrences during follow-up.[24] Another explanation may be related to an age-related increased risk of recurrent arrhythmias; in fact,

those with CA/SD were younger in our study than in the study by Haissaguerre et al.[18]

QRS slurring could be interpreted as intraventricular conduction delay. However, both in our case subjects (Table 1) as well as in those reported by Haissaguerre[18] no significant association was found between presence of late ventricular potentials and QRS slurring. Consistent with findings from experimental models, QRS slurring or prominent J wave may be secondary to an increase of I_{to} currents.[2,11,25] The higher prevalence of CA during or immediately after effort in our patients is not consistent with a previous report showing reduction or disappearance of early repolarization abnormalities in response to catecholamine infusion.[18] In that report, subjects were not exposed to the mechanisms associated with competitive sport activity possibly precipitating CA in patients at risk. In addition, sport-induced rate-dependent repolarization inhomogeneities,[21] unknown mutations of ion channels regulating cardiac repolarization, and alterations of repolarization secondary to possible intake of illicit drugs[26] also may have been precipitating factors.

The most frequent expression among all QRS-ST changes in control athletes was the presence of J wave in lateral leads alone (63%), whereas in CA athletes QRS slurring or J wave appeared much more frequently (80%) in the inferior leads, alone or associated with leads V_4 to V_6 (Table 2). These observations are consistent with those reported by Rosso[9] and suggest that in athletes, J-point elevation in leads V_4 to V_6 probably represents a "benign" finding.

Two striking findings in our study are that (1) the pattern of J wave or QRS slurring without ST-segment elevation was more frequently observed in CA/SD than in control athletes (Table 3); and (2) ST-segment elevation was more common in healthy control subjects than in CA/SD athletes. These observations suggest that the various patterns of QRS-ST changes may reflect different electrophysiological mechanisms that have different and possibly opposite influences in heart vulnerability to life-threatening arrhythmias. In particular, ST-segment elevation, mainly caused by an increased parasympathetic tone, may reflect a diffuse depression of action potential dome acting in turn as a stabilizing factor on the electrophysiological substrate.[2,11,12]

Clinical Implications

QRS-ST changes are a common finding in athletes, and major arrhythmic events leading to CA and SD are rare.[23,27] The present study, in agreement with Rosso et al[9] suggests that the incidental finding of a J wave or QRS slurring in a healthy population, including athletes, should be taken as a marker of minimally increased arrhythmic risk. By applying the Bayes formula of conditional probabilities,[22] presence of J wave or QRS slurring pattern increases the probability of CA or SD from about 2 per million to 3.5 per million in our population of competitive athletes. On the contrary, the presence of ST-segment elevation does not increase the probability of CA or SD in this population; rather, it may limit the increased risk associated with J wave or QRS slurring when present in combination. However, the true clinical significance of the various ECG patterns warrants prospective, long-term epidemiological studies. In particular, the significance of various QRS-ST changes (eg, presence in specific leads, time variability, and response to provocative tests) are still unclear and should be further elucidated.

Limitations

The present population represents a selected group of subjects with CA and therefore cannot be extrapolated to the general population. In addition, it is unknown how many subjects with SD were not referred for assessment within the same time frame and geographical area. Also, it is possible that the prevalence of QRS-ST changes observed in the present study does not reflect the true prevalence in the investigated populations. Although no major changes of the QRS-ST segment were observed during serial ECG recording in case and control groups, we cannot exclude that a higher incidence of changes may have occurred if ECGs were recorded more frequently, particularly in temporal proximity to VF.[9,15,17–20,28] Similarly, different prevalences than in our study, such as with Brugada syndrome, could be observed in other geographical areas.[29] A lower prevalence could be observed if QRS slurring was supported by depolarization delay rather than repolarization defect. In the present study, this hypothesis appears to be unlikely because of the similar distribution of late potentials in the 2 athlete populations with CA/SD, those with and those without J-point elevation or QRS slurring.

The ECGs were analyzed by 2 independent cardiologists, but the reading was not performed blindly; thus a possibility for bias could be present.

Given the inability to accurately retrieve information about use of illicit drugs administered to increase cardiovascular performance, it is not possible to exclude that some of these drugs may have contributed to precipitate CA at least in some of our patients. Finally, the low number of case subjects in our study reflects the very low prevalence of CA/SD in athletes with no heart disease and preparticipation screening selection. The observed data should therefore be taken with caution. However, the highly significant differences in the investigated parameters between case and control subjects outline the potential role of these parameters in predicting vulnerability to spontaneous ventricular arrhythmias.

Conclusion

The present study showed a higher prevalence of the ECG pattern of QRS-ST changes in competitive athletes who had CA in the absence of heart disease than in healthy athletes. Because QRS slurring and J-point elevation could reflect an underlying abnormality of repolarization, which makes the myocardium more sensitive to various and still not well-defined arrhythmogenic triggers, the finding should always be taken into consideration, particularly in subjects with other risk factors for arrhythmias.

Disclosures

None.

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CLINICAL PERSPECTIVE

The ECG pattern of early repolarization (ie, J wave, QRS slurring, and/or ST-segment elevation) in the inferior and lateral ECG leads is a common finding in the general population and is even more frequently observed in athletes. Recent studies have suggested a potential proarrhythmic significance of these findings in the general population, but data are lacking in athletes. We investigated whether QRS-ST changes are markers of risk for cardiac arrest or sudden death in athletes without underlying heart disease. In a selected group of 21 young competitive athletes who had cardiac arrest in the absence of heart disease, the prevalence of J wave and/or QRS slurring in the inferior (II, III, and aVF) and lateral leads (V_4 to V_6) was significantly higher in cases than in control athletes. After sport discontinuation during 36-month follow-up, arrhythmia recurrences did not differ between subgroups with and without J wave or QRS slurring. Because of discrepancy between the frequency of early repolarization pattern on 12-lead ECG and the rarity of cardiac arrest/sudden death, the incidental finding of a J wave/QRS slurring in a healthy athlete should be considered as a marker that minimally increases the arrhythmic risk. The present findings provide novel insights on clinical profiles of athletes at possible risk of cardiac arrest.

J Wave, QRS Slurring, and ST Elevation in Athletes With Cardiac Arrest in the Absence of Heart Disease: Marker of Risk or Innocent Bystander?

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