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

Running Title: Cappato et al.; QRS-ST changes in athletes with cardiac arrest.

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Abstract

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. Aim of our study was to investigate whether QRS-ST changes are markers of cardiac arrest (CA) of unexplained cause or sudden death (SD) in athletes.

Methods and Results. In 21 athletes (mean age 27 years, 5 females) with CA or SD, the ECG recorded prior to 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 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 case 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 (V4 to V6) were more frequently recorded in cases than in control athletes (28.6% vs 7.9%, P = 0.007). Among CA victims, 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 were found more frequently among athletes with CA/SD than in control athletes. Nevertheless, the presence of this ECG pattern seems not to confer a higher risk for recurrent malignant ventricular arrhythmias.

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, 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.

In athletes, changes of the QRS-ST segment may occur as a consequence of physiologic resetting of the balance between the sympathetic and parasympathetic tone ultimately regulating trans-membrane 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 experienced a resuscitated CA or SD due to 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 remaining 21 athletes (mean age 27±8, 14 to 43 years, 16 males) had no evidence of either structural heart disease, Brugada syndrome, pre-excitation, long or short QT on the basis of clinical, electrocardiographic and
echocardiographic data and represent our study population. Their demographic and clinical characteristics are presented in Table 1.

Of the 21 athletes, 19 had experienced CA and 2 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 electrocardiographic, 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 females) who underwent pre-participation screening and were considered healthy and eligible for competitive sport activity. Each of the controls 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, ECG analysed were those recorded prior 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 pre-event ECG analysis in 10 CA victims was not possible, because of unavailability of athletes’ files at referral center years after original recording. Of the control group, ECGs recorded at the time of pre-participation screening were analyzed.

The following QRS-ST changes were analyzed: “J point elevation” (J wave), “slurred QRS complex”, and “ST segment elevation”. Namely, “J waves” were defined as positive “hump-like” deflections immediately following 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 times 2 magnification. In order 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, aVF), lateral (I, aVL, and V4 to V6), or infero-lateral leads (II, III, aVF and V4 to V6). PR interval, QRS duration and QTc (QT interval corrected with Bazett’s formula and with Fridericia’s formula) were also measured in both groups. All ECGs were analyzed by two 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 pre-event ECG and in 115 control athletes in the control group, of which 56 presenting 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), signal averaging ECG (in 8) were performed in athletes with resuscitated CA. In 17 of 19 cases the electrophysiological study (EPS) was performed: a maximum of 2 or 3 extrastimuli were delivered from 2 ventricular sites. Patients with VF, polymorphic ventricular tachycardia (VT) lasting > 30 sec 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, 31-119), 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 were recorded relative to the level of training (or detraining), and use of antiarrhythmic drugs, or 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 for continuous variables and percentage values for non-continuous variables. Comparisons between groups were performed with Student’s t-test and Pearson’s chi square when appropriate. Results are displayed as the OR plus 95% CIs. All p values were considered significant at a 0.05 value. All these statistical calculations were done using a computerized statistical package (SPSS 13.0, Chicago, Il.). The propensity score analysis was performed using SAS software package (SAS Institute Inc., 1989. SAS/STAT user’s guide, version 8e. Cary, NC SAS Institute Inc.).

In order 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 is detected, with the following formula:

\[
P_{j} \text{(CA/SD)} = \frac{P \text{(CA/SD)} \times P_{ca/sd} \text{(J)}}{P \text{(CA/SD)} \times P_{ca/sd} \text{(J)} + P \text{(not CA/SD)} \times P_{not ca/sd} \text{(J)}}
\]

where \( P_{j} \text{(CA/SD)} \) 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 \( P_{ca/sd} \text{(J)} \) and \( P \text{ not ca/sd (J)} \),
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 CA would occur within the same time frame. Moreover, we hypothesized that about 5% of CA/SD events likely 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, one on stand-by during 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 stand-by 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, i.e., 16 of 21, were not preceded by symptoms. Of the remaining 5 athletes, 3 had suffered one or more syncope during physical activity a few months before CA occurred, which had remained unexplained and without documentation of arrhythmia. Two other subjects complained of 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 controls athletes (60±7 bpm vs 66±10, P = 0.02). PR interval (162±14 vs 159±23 msec) and QRS duration (88±8 vs 86±7 msec) were not statistically different. QTc corrected with Bazett’s formula was not statistically different in the cases and control group (411±21 vs 402±27
msec, \( P = 0.10 \)), whereas the QTc corrected with the Fridericia’s formula was longer in cases than in control group, with borderline statistical significance (412±18 vs 398±24, \( P=0.05 \)). However, all QTc values were within the normal variability range.

**J wave and QRS slurring**

Compared to controls, athletes with CA/SD showed more frequently a slurred QRS pattern in any leads (28.6% vs. 7.6%, \( P = 0.006 \)) and a J point elevation and/or slurred QRS in standard inferior plus pre-cordial V4 to V6 leads (28.6% vs. 7.9%, \( P = 0.007 \)) (Table 2). These differences were confirmed when the case subject population was compared with an age- and gender-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% vs. 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 (Fig. 1A) than in control athletes (Fig.1B) (38.1% vs. 15.6%, \( P = 0.04 \)), particularly in inferior-lateral leads (23.8% vs. 2.5%, \( P = 0.001 \)) (Table 3). These differences were confirmed when the case subject population was compared with an age- and gender-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 experiencing 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 presence of positive ventricular late potentials. On exercise testing, frequent ventricular ectopic beats or runs of non-sustained VT were induced in 14 (7 with QRS-ST changes) of 19 athletes. In no patients coronary or ischemic abnormalities were observed either during coronary angiography, cardiac magnetic resonance or exercise testing. The ECG Holter monitoring showed episodes of non-sustained VT in 6 of 9 cases with, and in 5 of 10 cases without QRS-ST changes. The EPS 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 31-119). All were strongly recommended to stop regular training and competition, but 3 of 19 were not compliant. All subjects were administered pharmacological treatment: beta-blockers (13), amiodarone (5), propafenone (2), or diltiazem (1). Of 19 athletes, 13 were treated with an ICD; 3 refused ICD implantation and another 3 were treated with anti-arrhythmic drugs. The distribution of non-compliant 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 wave 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 were 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 non-sustained VT. Of 10 athletes with J wave or slurred QRS, 3 experienced 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 experienced CA in 1983 during a soccer game, died suddenly ten years later at age 33 yrs at home. He had refused ICD implantation and was under propafenone at time of death.

**Discussion**

The pattern of QRS-ST elevation observed in the inferior and lateral ECG leads has been considered for long time to be an innocent finding in individuals without evidence of cardiac disease, in spite of several case-reports (14-17) and recent studies describing the association of this pattern with idiopathic VF in subjects with 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 presence of QRS slurring or J wave in the absence of ST elevation in the inferior-lateral ECG leads were associated with a marginally increased risk of experiencing 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 after-depolarizations and circum movement re-entry 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, most likely 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 manoeuvres. Nevertheless, this
interpretation appears unlikely, because all 5 athletes with CA and pre-event 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 time of 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, CA/SD victims 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 cathecolamine 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) may also have been a 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} likely 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 than in CA/SD athletes. These observations suggest that the various patterns of QRS-ST changes may reflect different electrophysiological mechanisms which have different and possibly opposite influences in heart vulnerability to life-threatening arrhythmias. In particular, ST segment elevation, mainly due to 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) suggest 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 (e.g.: presence in specific leads, time variability, response to provocative tests) are still unclear and should be further elucidated.

Limitations

The present population represents a selected group of CA victims and therefore cannot be extrapolated to the general population. In addition, it is unknown how many SD victims 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 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 could be observed in other geographical areas, such as with Brugada syndrome (29). A lower prevalence could be observed, if QRS slurring was supported by depolarization delay rather than repolarization defect. In our study this hypothesis appears unlikely, because of the similar distribution of late potentials...
in the two athlete populations with CA/SD, those with and those without J point elevation or
QRS slurring.

The ECGs were analyzed by two 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 pre-participation 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 electrocardiographic pattern of
QRS-ST changes in competitive athletes who experienced CA in 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.

Conflict of Interest Disclosures: None
References


Table 1. Demographics and clinical findings in the 21 athletes with CA/SD without heart disease.

<table>
<thead>
<tr>
<th></th>
<th>J w/QRS slurring (N = 10)</th>
<th>No J w/QRS slurring (N = 11)</th>
<th>P value</th>
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<tbody>
<tr>
<td>Male sex, no. (%)</td>
<td>8 (80%)</td>
<td>8 (72%)</td>
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<td>Age, yr (mean±standard deviation)</td>
<td>25.5±5</td>
<td>28.9±1</td>
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<td>Confirmation of idiopathic VF:</td>
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<td>- Coronary angiogram, no.</td>
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<td>6</td>
<td></td>
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<tr>
<td>- MRI, no.</td>
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<tr>
<td>Sport:</td>
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<tr>
<td>- Endurance-trained</td>
<td>10</td>
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<tr>
<td>- Weight lifter</td>
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<td></td>
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<tr>
<td>- Level: international/national</td>
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<td>4</td>
<td></td>
</tr>
<tr>
<td>- regional</td>
<td>5</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>- Duration (hours/week )</td>
<td>16.8±4</td>
<td>15.5±5</td>
<td>0.4</td>
</tr>
<tr>
<td>Resuscitated CA</td>
<td>10</td>
<td>9</td>
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<td>Sudden death</td>
<td>0</td>
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<tr>
<td>Activity at time of CA/SD:</td>
<td></td>
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<tr>
<td>- During effort</td>
<td>7</td>
<td>5</td>
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<tr>
<td>- On stand-by during game</td>
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<tr>
<td>- On stand-by after effort</td>
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<td>- Rest</td>
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<td>- Sleeping</td>
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<tr>
<td>ECG (post-CA) :</td>
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<tr>
<td>- PR msec</td>
<td>159±16</td>
<td>167±10</td>
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<tr>
<td>- HR bpm</td>
<td>57±5</td>
<td>61±7</td>
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<td>- QRS duration</td>
<td>89±6</td>
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<td>- QTc (Bazett’s formula)</td>
<td>405±19</td>
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<td>- QTc (Fridericia’s formula)</td>
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<td>415±17</td>
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<td>SAECG (8 cases)</td>
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<td>- Presence of late potentials</td>
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<td>3/4</td>
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<td>Holter Recording (19 cases)</td>
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<tr>
<td>- Non-sustained VT</td>
<td>6/9</td>
<td>5/10</td>
<td>0.43</td>
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<td>Exercise test (19 cases)</td>
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<td>- Complex VPB</td>
<td>5/10</td>
<td>3/9</td>
<td>0.46</td>
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<tr>
<td>- Non-sustained VT</td>
<td>2/10</td>
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<td>0.26</td>
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<td>EPS (17 cases)</td>
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<td>- Inducibility VT-VF</td>
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<td>6/9</td>
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<td>- LVWTh</td>
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<td>- LVSTh</td>
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</tbody>
</table>

CA = cardiac arrest, EPS = electrophysiological study, J w = J wave, LVWTh = left ventricular wall thickness, LVSTh = left ventricular septal thickness, MRI = magnetic resonance imaging, SAE CG = signal avering electrocardiogram, SD = sudden death, VF = ventricular fibrillation, VT = ventricular tachycardia.
Table 2. Distribution of QRS Slurring, J wave and ST segment elevation ≥ 0.05 mV in athletes with CA and control subjects.

<table>
<thead>
<tr>
<th></th>
<th>CA Subjects</th>
<th>Control Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N. 21</td>
<td>N. 365</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>%</td>
</tr>
<tr>
<td>J-Wave +/- Slurred QRS +/- ST elevation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any lead</td>
<td>10</td>
<td>47.6</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>2</td>
<td>9.5</td>
</tr>
<tr>
<td>Lateral lead</td>
<td>2</td>
<td>9.5</td>
</tr>
<tr>
<td>Inferior + V₄ to V₆</td>
<td>6</td>
<td>28.6</td>
</tr>
<tr>
<td>Slurred QRS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any lead</td>
<td>6</td>
<td>28.6</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>3</td>
<td>14.3</td>
</tr>
<tr>
<td>Lateral leads</td>
<td>1</td>
<td>4.7</td>
</tr>
<tr>
<td>Inferior + V₄ to V₆</td>
<td>2</td>
<td>9.5</td>
</tr>
<tr>
<td>J-Wave</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any lead</td>
<td>7</td>
<td>33</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>2</td>
<td>9.5</td>
</tr>
<tr>
<td>Lateral lead</td>
<td>4</td>
<td>19</td>
</tr>
<tr>
<td>Inferior + V₄ to V₆</td>
<td>1</td>
<td>4.7</td>
</tr>
</tbody>
</table>

CA = cardiac arrest , CI = confidence interval, OR = odds ratio; +/- means with or without, Pps = P value adjusted with propensity score analysis.
Table 3. Distribution of ST segment elevation >0.05 mV in athletes with CA and 365 control subjects.

<table>
<thead>
<tr>
<th></th>
<th>CA Subjects</th>
<th></th>
<th>Control Subjects</th>
<th></th>
<th>P Value</th>
<th>OR</th>
<th>CI 95%</th>
<th>Pps</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N. 21</td>
<td>N. 365</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST segment elevation &gt;0.05 mV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any lead</td>
<td>2</td>
<td>9.5</td>
<td>79</td>
<td>21.6</td>
<td>0.27</td>
<td>0.381</td>
<td>0.09-1.67</td>
<td>0.27</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>1</td>
<td>4.7</td>
<td>8</td>
<td>2.2</td>
<td>0.40</td>
<td>2.23</td>
<td>0.27-18.72</td>
<td>0.16</td>
</tr>
<tr>
<td>Lateral leads</td>
<td>1</td>
<td>4.7</td>
<td>44</td>
<td>12.1</td>
<td>0.49</td>
<td>0.365</td>
<td>0.05-2.79</td>
<td>0.39</td>
</tr>
<tr>
<td>Inferior + V₄ to V₆</td>
<td>0</td>
<td>0</td>
<td>27</td>
<td>7.4</td>
<td>0.38</td>
<td>--</td>
<td>--</td>
<td>0.22</td>
</tr>
<tr>
<td>J-Wave +/- Slurred QRS no ST elevation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any lead</td>
<td>8</td>
<td>38.1</td>
<td>58</td>
<td>15.8</td>
<td>0.04</td>
<td>2.86</td>
<td>1.1-7.43</td>
<td>0.02</td>
</tr>
<tr>
<td>Inferior leads</td>
<td>2</td>
<td>9.5</td>
<td>11</td>
<td>3</td>
<td>0.19</td>
<td>--</td>
<td>--</td>
<td>0.36</td>
</tr>
<tr>
<td>Lateral lead</td>
<td>1</td>
<td>4.7</td>
<td>38</td>
<td>10.4</td>
<td>0.48</td>
<td>--</td>
<td>--</td>
<td>0.49</td>
</tr>
<tr>
<td>Inferior + V₄ to V₆</td>
<td>5</td>
<td>23.8</td>
<td>9</td>
<td>2.4</td>
<td>0.001</td>
<td>10.99</td>
<td>3.25-37.15</td>
<td>0.009</td>
</tr>
</tbody>
</table>

CA = cardiac arrest, CI = confidence interval, OR = odds ratio; +/- means with or without, Pps = P value adjusted with propensity score analysis.
Figure legend:

Figure 1 - J wave, QRS slurring and ST segment elevation in athletes. A. Electrocardiogram of a 21-year old male, soccer player, who experienced cardiac arrest during practice. J waves >0.05 mV are present in leads II, III, aVF without ST segment elevation. In the lower panel tracings are magnified and the arrows indicate the J waves. B. Electrocardiogram of a 29-year old male cyclist in the control group showing a QRS slurring in leads II, III, aVF and a J wave in leads V₃ to V₆. ST segment elevation is evident in leads II, III, aVF and V₄ to V₆. In the lower panel tracings are magnified and the oblique arrows indicate QRS slurring in leads II, aVF and J wave in V₆; vertical arrows indicate ST segment elevation.
J Wave, QRS Slurring and ST Elevation in Athletes with Cardiac Arrest in the Absence of Heart Disease: Marker of Risk, or Innocent Bystander?

Riccardo Cappato, Francesco Furlanello, Valerio Giovinazzo, Tommaso Infusino, Pierpaolo Lupo, Mario Pittalis, Sara Foresti, Guido De Ambroggi, Hussam Ali, Elisabetta Bianco, Roberto Riccamboni, Gianfranco Butera, Cristian Ricci, Marco Ranucci, Antonio Pelliccia and Luigi De Ambroggi

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