CHAPTER

10

Prevalence and Significance of Early Repolarization (a.k.a. Haïssaguerre or J-Wave Pattern/Syndrome)

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INTRODUCTION

This contribution should begin with a clarification of the point of view that will be taken. The previous 2 chapters have been written by 2 of the most accomplished electrophysiologists of our time. In their clinical practice and research, they have seen a number of patients with rare condition of idiopathic ventricular fibrillation (VF). They were 2 of the first investigators of a new channelopathy, where the arrhythmic events in patients with idiopathic ventricular tachycardia/VF (IVT/VF) were often preceded by the dynamic appearance of large J waves[1] similar to the Osborne waves of hypothermia (Figure 10.1). Though their writings are hopeful in tone, neither has presented convincing data nor enthusiastic support that any ECG finding on the routine 10-second ECG taken at a stable period in the course of these patients' disease will identify them as having the channelopathy.

In contrast, in spite of extensive clinical contact with cardiology patients and screening of athletes for the risk of sudden cardiac death (SCD), this author has never diagnosed an individual with this new channelopathy. Furthermore, in spite of many years of interpreting ECGs from patients and athletes as well as reading Holter and event recorders, he has never observed dynamic J waves. This is not to say they do not exist, but to stress the point that they are rare, and most of us will only see the exciting examples of them in the literature. Hopefully, now that awareness is widespread, more cases will be recognized and patients helped.

My main interest in this new syndrome has been in analyzing the screening, at-rest 10-second ECG to demonstrate components of the downslope of the R wave or S-wave upslope that could identify individuals at a risk of sudden death. This unprecedented interest in phenomena occurring on the downslope of the R wave or S-wave upslope (i.e., the general area where ventricular depolarization and repolarization overlap) has highlighted other complicating issues. These include how should QRS end be measured when they are present (are current normal values for QRS duration correct?), what is the J point, where is the ST level set, and do the current automated ECG programs make these measurements correctly when slurring or J waves are present?

Interest peaked in this new channelopathy with the seminal paper in the *New England Journal of*

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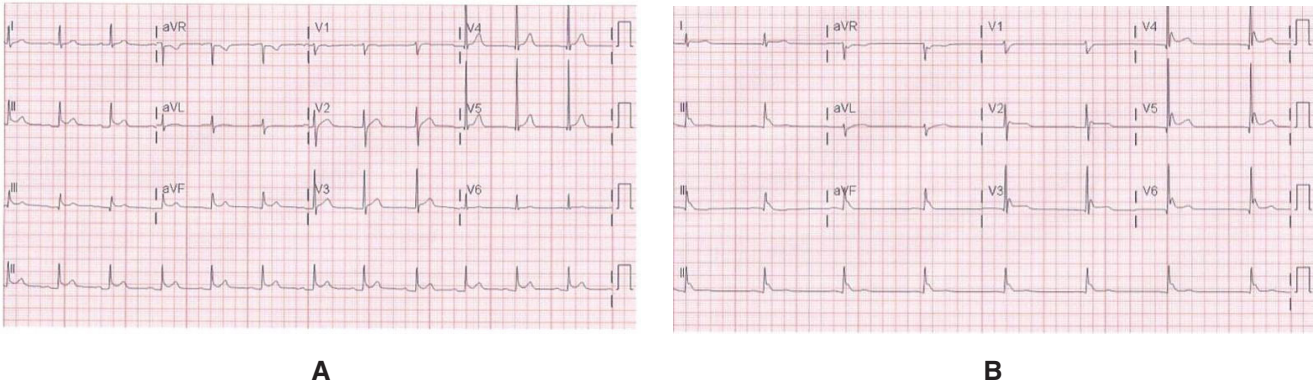

Figure 10.1. Example of Osborne waves. The 2 ECGs were obtained from a 56-year-old male with cerebral palsy admitted to a local hospital in pneumosepsis from a nearby long-term care facility. Cardiology called because of unusual ECG and the next day the patient's core temperature dropped to 33°C. The patient was treated with antibiotics and then the ECG returned to baseline. (Courtesy Dr. Bing Liem). **A.** Admission ECG. **B.** ECG from the next day (unfortunately serial ECGs are not available but Osborne waves disappeared while discharging).

Medicine (NEJM) from the French Multicenter study of patients with IVT/VF and interest sparked by the title inclusion of the term "Early Repolarization (ER)."[2] To most of the American physicians, that was a term associated with a benign ECG pattern of ST elevation (sometimes accompanied by J waves and slurring), often noted in healthy young athletes of African descent.[3] The only clinical concern with it was the similarity between it and the ST-elevation patterns of pericarditis, STEMI, variant angina, and acute transmural ischemia.[4] The implication that this ECG pattern could be predictive of arrhythmic death seemed unlikely, but any hope of confirming a new marker for sudden death in young athletes certainly was worth exploring. The possibility that the rest ECG could be used in population studies to predict arrhythmic death was supported by the unique Finnish study with a 30-year follow up published shortly after, also in the NEJM.[5] Subsequently, we hypothesized that this study and others supporting the routine rest ECG as a predictor of CV death were due to the association of J waves and slurs with Q waves, ST depression, and T-wave abnormalities, but that has yet to be resolved.[6]

Semantics were coming to the forefront of deciphering these new findings. ER had a definition long used by clinical electrocardiographers and clinicians (ST elevation on an otherwise normal ECG); to electrophysiologists and cellular physiologists, it was the first part of repolarization on the myocytes, action potential, and it was now being attached to J wave and slurs at the end of the QRS complex.[7] Figure 10.2 contrasts "classic" ER to the "new" ER on the surface ECG. While Viskin and Kukla first suggested the new channelopathy be called the Haïssaguerre syndrome,[8,9] Wagner suggested J-wave syndrome to be another option, but most electrophysiologists still (as the Editors of this book) use the confusing "ER."

Our research laboratory has a large, digital ECG database of veterans and athletes, and so when a group of young researchers coalesced in the summer of 2010, we began a series of investigations. The first step was to perform a careful literature review and determine the methodology of the ECG measurements in the new studies. The semantic confusion and lack of details became very evident. Terms such as "J-point," "QRS end," and "ST level," which had been

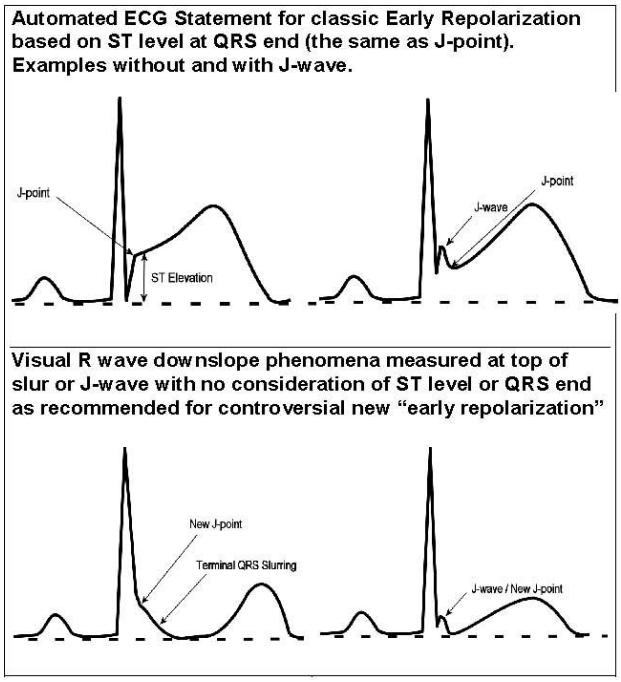

Figure 10.2. Contrasting surface ECG representations for "ER." Classic ER also required the ECG to not exhibit diagnostic Q waves, ST depression, or T-wave inversion, but not so for the "new" ER. Classic ECG interpretation considered the J point to be the end of the QRS complex, where the ST level for diagnosis of ischemia was set, while the "new" ER considered it to be the top of the J wave or slur.

clearly defined in guidelines and position papers were not being used consistently. Often the methods used could only be deciphered from the ECG examples provided. It appeared that the dynamic ECG waveforms of this new syndrome were causing confusion in the interpretation of the standard 10-second ECG. We initiated an email group of those working in this field to exchange ideas and found that experts no longer were agreeing on how to determine QRS duration or ST level. Furthermore, cellular physiologists were not in agreement regarding whether the J waves and slurs were late depolarization or ER phenomena. There was a concern that action potentials from isolated sections of dog hearts had less importance in the etiology of this channelopathy than studies of the electrical activity of the entire heart. This led to the collaboration with the Editor of the *Journal of Electrocardiography* and a special issue of the subject including many of the world experts.[10]

Most of the automated ECG programs used in U.S., including the GE MUSE 12-lead program used by all Veterans Affairs (VA) medical centers, make an interpretative statement of "ER" but this is based on ST elevation and normality of the Q and T waves (and certainly no ST depression). None of the commercially available programs code or measure terminal QRS J waves or slurs and in general they include them as a part of the QRS complex. Therefore, our ECGs had to be re-read and coded for J waves and slurs and QRS end confirmed. While facilitated using computerization for recall and display, this was still time-consuming. Having personally re-read and coded 4000 ECGs from veterans with a 10-year follow up for CV death, it was very disappointed to find no association. We are now in the process of validating our measurement techniques and expanding our coding to 20,000 ECGs with a 15-year follow up for CV death.

The databases we have, though, did have reliable measurements of ST level, and so we initiated a series of studies relating to ST elevation in athletes[11] and the Universal Definition of Myocardial Infarction (UDMI) for diagnosing ischemia.[12] Listed below are the causes of ST elevation (Table 10.1) and next the UDMI criteria (Table 10.2).

The UDMI criteria were empirical, too complex, and did not consider ethnicity, so we considered how many stable clinic patients fulfilled them and were false positives. These parallel efforts included studies of athletes and serial ECG studies in patients to demonstrate the natural history of ER. We demonstrated that the original ER based on ST elevation was related to age, gender, ethnicity, and heart rate as shown in Table 10.3. Figure 10.3 is an example of ST elevation due to pericarditis that can only be distinguished from classic ER by these features. We also noted that J waves and slurs were commonly associated with ST elevation in healthy and young individuals with normal ECGs, and were more common in elderly patients with Q waves or T-wave inversion with or without ST elevation than those with normal ECGs.

Table 10.1. Causes of ST elevation on resting ECG.

Acute (Dynamic)
1. Ischemia (localizes, arrhythmogenic).
2. Variant angina.
3. ST-elevation MI.
4. Before event in some patients with idiopathic VT/VF.
Chronic (Stable)
1. ER—changes with heart rate.
2. Pericarditis.
3. Over Q waves associated with LV aneurysm/wall motion abnormalities (WMA).
4. Spinal cord injury and mental patients—vagal tone?
5. Brugada patterns (V_{123})—but syndrome dynamic.
6. In 31% of idiopathic VT/VF (lateral, inferior).

NATURAL HISTORY OF THE J WAVE, SLURS, AND ST-ELEVATION PATTERNS

While ST elevation, J waves, and slurs are more common in the young, suggesting that it recedes with age, there have been limited studies of its prognosis and its natural history is uncertain. Demonstrating that these end-QRS phenomena can disappear naturally rather than its decreasing prevalence being due to death would supplement appropriate studies using survival analysis. Therefore, we published the natural history of lateral and inferior lead occurrences utilizing serial ECGs in an ambulatory clinical population.

Of the 250 patients selected with the greatest amplitude of ST elevation, J waves, or slurs in the lateral leads, after 6 were excluded for ECG abnormalities, 122 had another ECG at least 5 months later.[13] Their average age was 42 ± 10 years and average time between the first and second ECG was 10 years. Of the 122 patients, 47 (38%) retained the amplitude criteria while the majority (62%) no longer fulfilled

Table 10.2. Criteria for ST elevation ischemia according to the third UDMI. New ST elevation at the J point in 2 contiguous leads (with V_1, III, and aVR excluded) using the lead gender and age cut-points.

1. All leads, ages, and gender other than leads V_2–V_3 = ≥0.1 mV.
2. For leads V_2 to V_3:
a. ≥0.2 mV in men ≥40 years
b. ≥0.25 mV in men <40 years
c. ≥0.15 mV in women

Table 10.3. Features in otherwise-normal ECGs differentiating ST elevation that is physiological from pathological conditions.

1. Heart rate (low, not high).
2. Age (young, not old).
3. Gender (male, not female).
4. ST level (<0.2 mV, not more).
5. Ethnicity (Afro-American, not other).
6. Athletic status (yes, not sedentary)

the amplitude criteria. This was not due to a higher heart rate, a longer time between the ECGs, death, acute disease, or alterations in ECG diagnostic characteristics. Figures 10.4 and 10.5 are the examples of ECGs from the serial lateral lead study.

We next studied the patients selected with the greatest amplitude of end QTS phenomena in the inferior leads, which became more important once some of the prognostic studies isolated mortality to them. Starting from the highest amplitude, we carefully reviewed the ECGs and medical records from the first 85%.[14] From this convenience sample, 36 were excluded for abnormal patterns (myocardial ischemia or infarction and pericarditis). The remaining 257 patients were searched for another ECG at least 5 months later, of whom 136 satisfied this criteria. All these ECGs were paired for comparison, printed, and coded by 4 interpreters. Their average age was 47 ± 13 years and average time between the first and second ECGs was 10 years. Of the 136 subjects, 64 (47%) retained the patterns while 72 (53%) no longer fulfilled the amplitude criteria. While no significant differences were found in initial heart rate or time interval between ECGs, those who lost the pattern had a greater difference in heart rate between the ECGs (ΔHR of 0.0 vs. 10 bpm) and higher percentage of cardiovascular events over the interval. In conclusion, the ECG pattern of ER was lost over 10 years in over half of this young clinical cohort. The change in heart rate and higher incidence of cardiovascular events partially explained the loss but it was not due to the time interval between the ECGs, association with lateral lead J waves/slurs, or alterations in other ECG diagnostic characteristics. The following are the examples of ECGs from the serial inferior lead study (Figures 10.6–10.8).

PREVALENCE OF END-QRS ECG PHENOMENA

To study the prevalence of all of the end-QRS components of interest (ST elevation, J waves, and slurs), we retrospectively studied 5085 ECGs obtained in a multiethnic clinical population from 1997 to 1999 at the Veterans Affairs Palo Alto Healthcare System, analyzing 4041 after excluding those with confounding ECG abnormalities. We also examined ECGs obtained from the preparticipation exams of 1114 Stanford University varsity athletes in 2007 and 2008.[15] Criteria for components of ER were as follows: ST elevation ≥1 mm from the end of the QRS complex; J wave as an upward deflection on the QRS downslope peaking ≥1 mm above the isoelectric line; and slur of the R-wave downslope as a decrease in the slope beginning at similar amplitude.

Components of ER were most prevalent in males, African Americans, and particularly in athletes, with the greatest variations demonstrated in the lateral

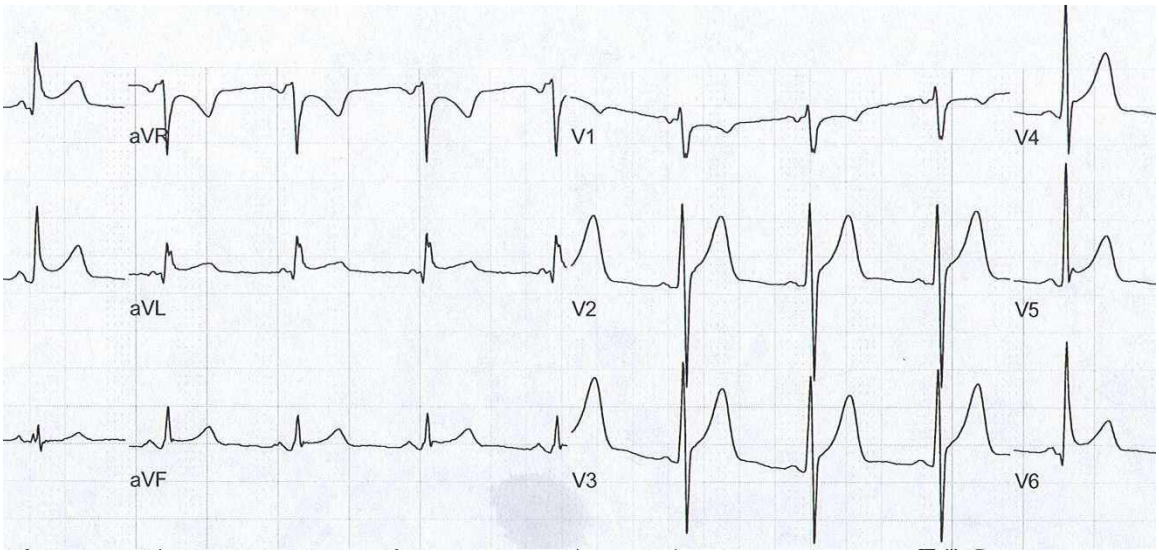

Figure 10.3. ECG in a 60-year-old white male with pericarditis (HR of 85 bpm due to β-blockers).

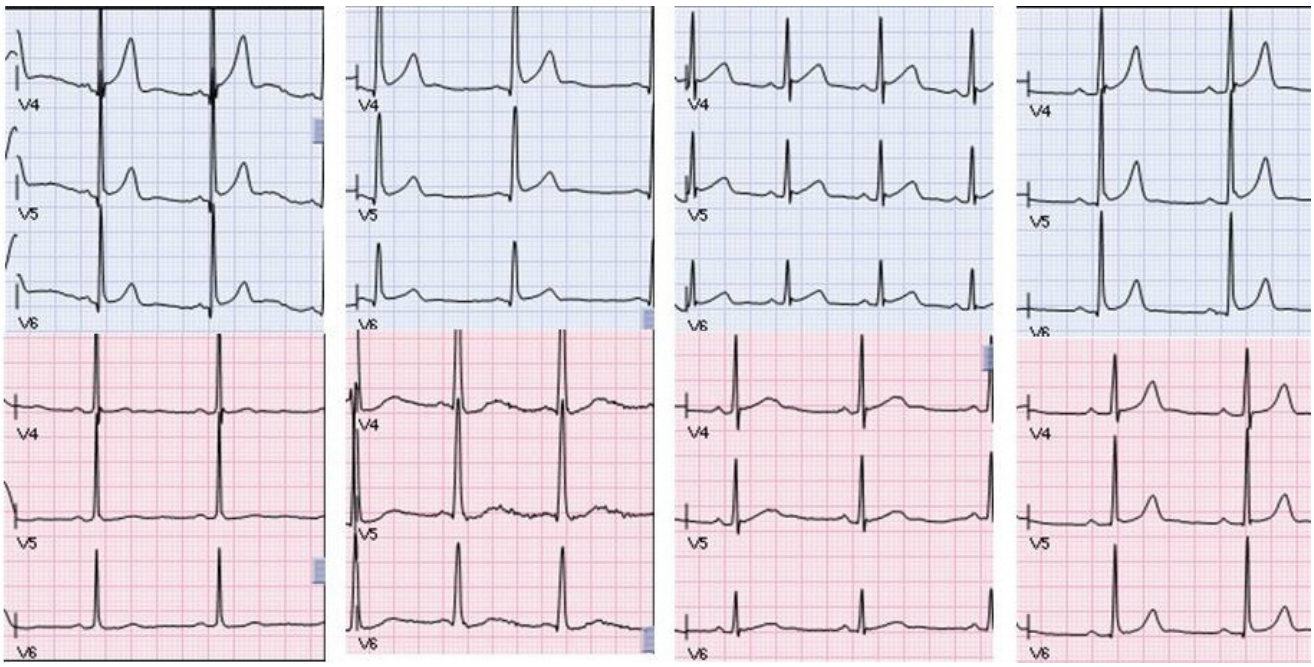

Figure 10.4. Four examples of paired ECGs with the greatest difference in amplitudes between ECGs consistent with loss of slurs and J waves in lateral leads (which occurred in 62%).

leads. ST elevation was most common, occurring in nearly one-third of male, African-American athletes in the lateral leads. Inferior J waves and slurs, previously linked to cardiovascular risk, were observed in 9.6% of clinical subjects and 12.3% of athletes. Figure 10.9 illustrates these findings.

PROGNOSTIC VALUE OF END-QRS ECG PHENOMENA

In those who manifest the Haïssaguerre pattern (R-wave downslope notching and slurring) on the stable, surface ECG, it will be necessary to identify

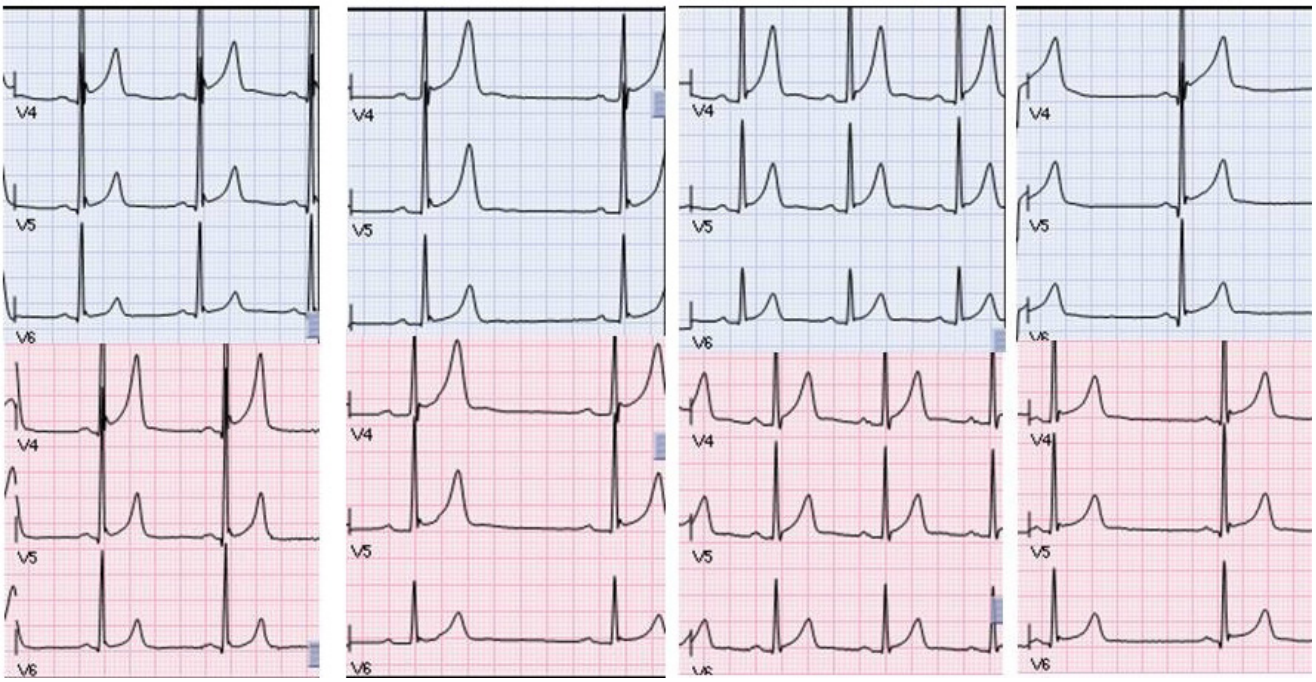

Figure 10.5. Four examples of paired ECGs with the least difference in amplitudes between ECGs consistent with retention of lateral slurs and J waves (which occurred in 38%).

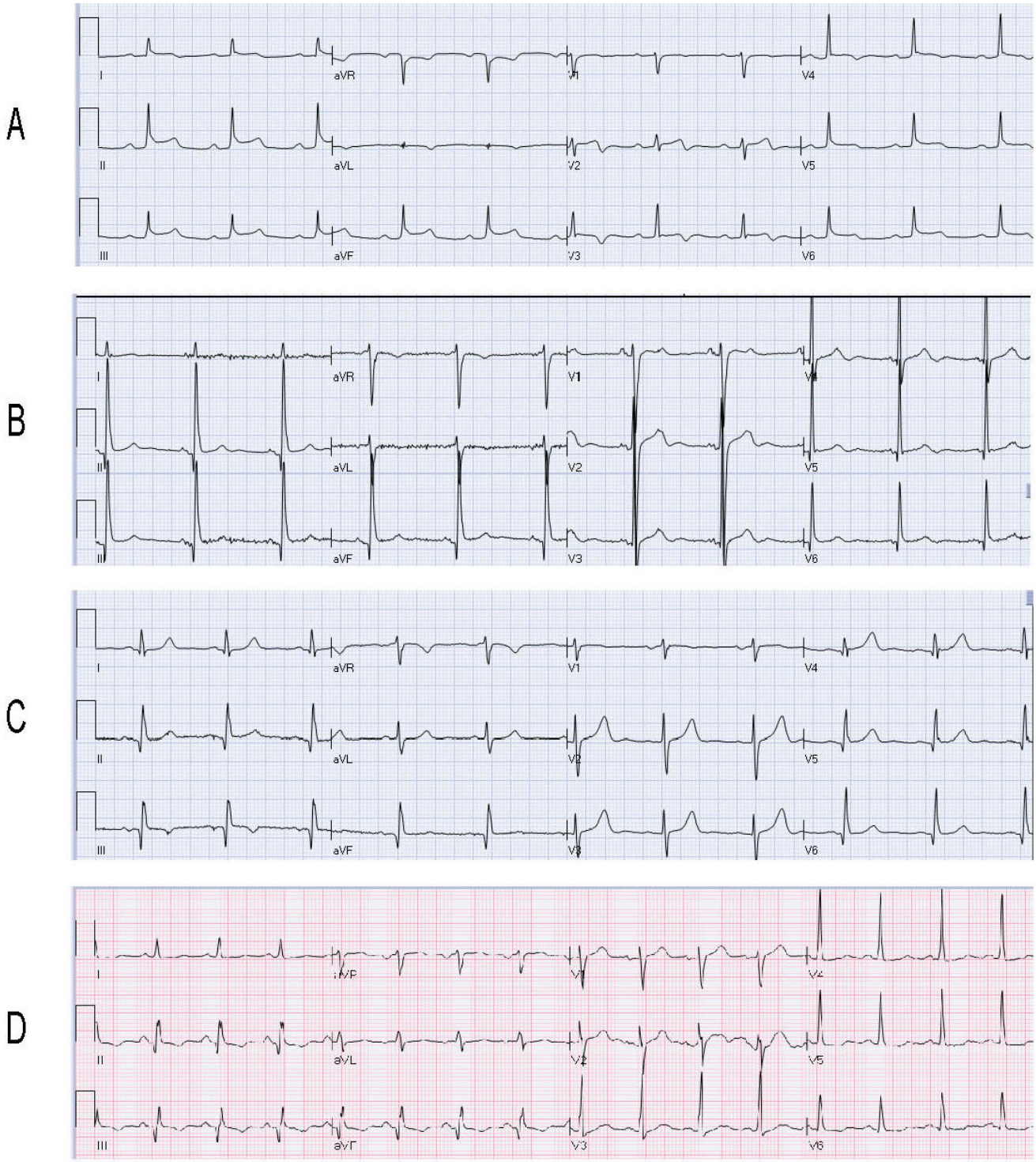

Figure 10.6. Examples of ECGs excluded from the inferior lead serial study. These were excluded because of (**A**) pericarditis, (**B**) HCM Q waves, (**C** and **D**) old inferior MIs. Such abnormalities were frequently associated with inferior J waves and slurs.

additional ECG markers that can predict risk of cardiovascular death and/or syncope, and thus develop the Haïssaguerre syndrome. This is of major importance, since we know that 50% of sudden deaths in young people occur with morphologically normal hearts.[16] Prognostic studies of this and similar ECG patterns have had differing results, but some suggest that end-QRS notching and slurring, particularly when occurring in the inferior leads and accompanied by downward-sloping ST segments, have associated risk of sudden or cardiovascular death. The differences in the studies appear to be due to terminology and

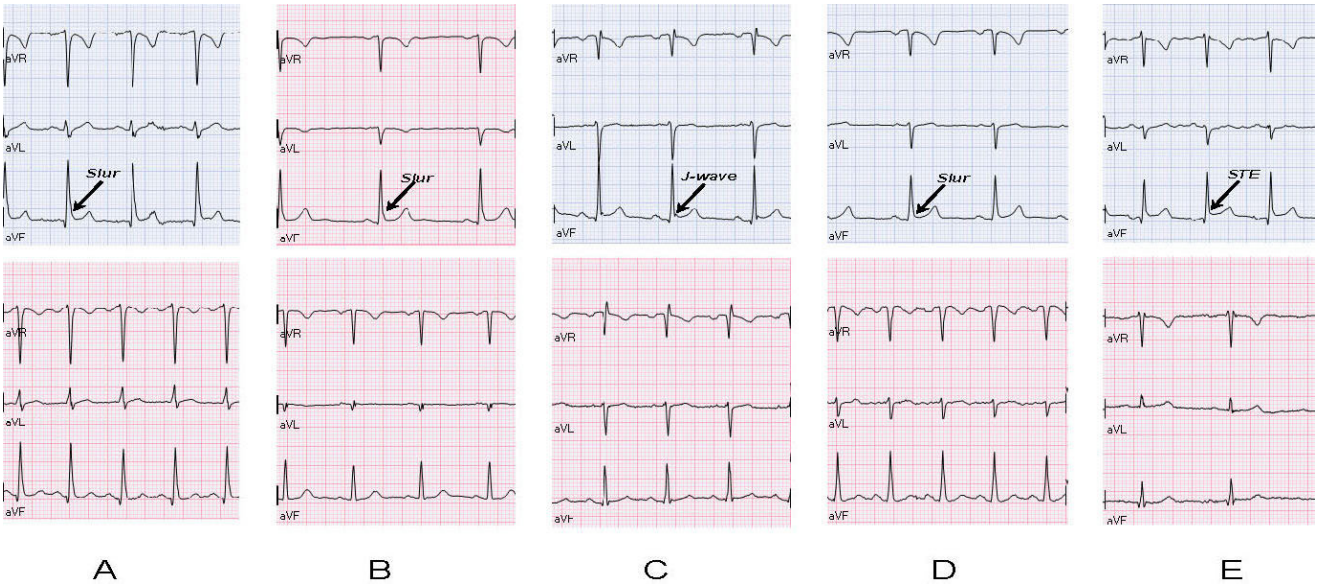

Figure 10.7. Five examples of paired ECGs with lost inferior J wave/slurs (which occurred in 53%).

methodology issues as well as design shortcomings.[17,18] A total of 8 prognostic studies were available as of February 2013 (Table 10.4).

Clearly, the best follow-up study and unlikely to be repeated is that by Tikkannen et al.[19] This classic study was only "limited" by noncomputerized ECG acquisition; the paper ECG recordings were gathered over 30 years ago (requiring the use of adjacent lead criteria for accuracy, unlike modern ECG analyses that rely on waveforms averaged over 10 seconds). Only inferior lead J waves or slurring were associated with CV or arrhythmic death risk. No risk was found for lateral lead slurring or J waves, and the anterior leads were not studied. The follow-up period of 30 years is important since the Kaplan–Meier survival curves only began to separate after 10 to 15 years. Furthermore, this study benefits from a national policy of standardized autopsies and investigations to determine the cause of death including arrhythmic deaths. Sinner et al[20] documented an increased hazard ratio for CV mortality associated with the Haïssaguerre pattern, especially in the inferior leads. However, they used a case-cohort design that only considered a younger subset of their community-based population. When

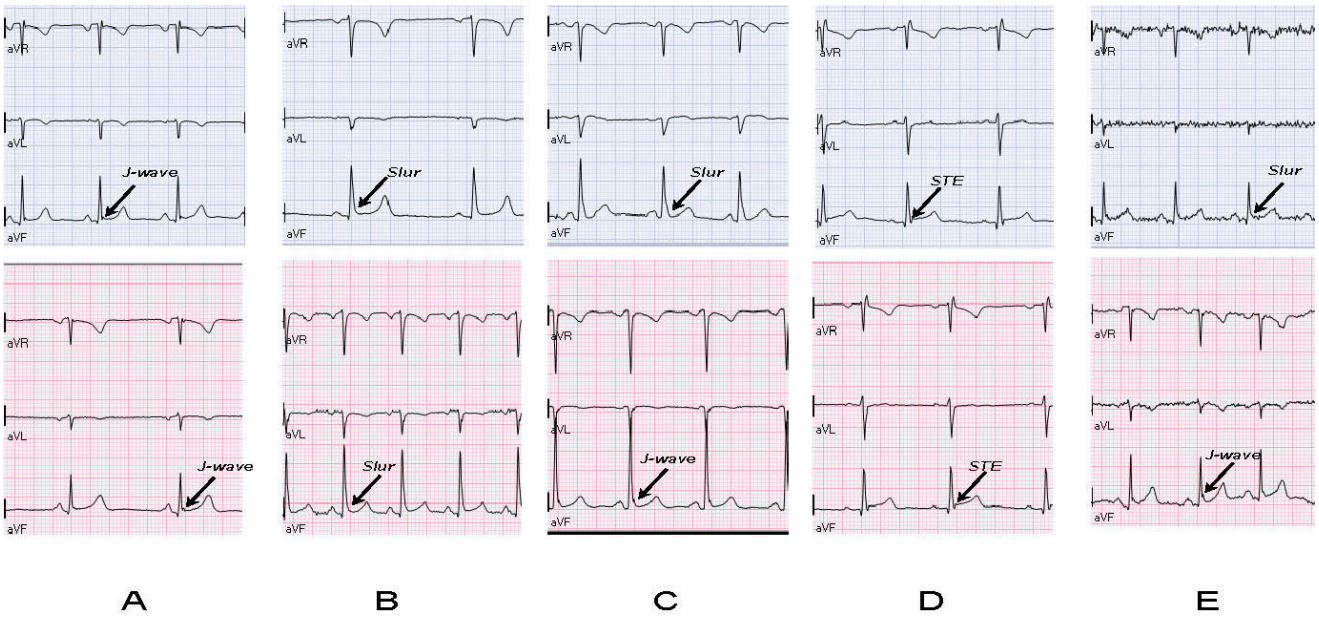

Figure 10.8. Five examples of paired ECGs with retained inferior J wave/slurs (which occurred in 47%).

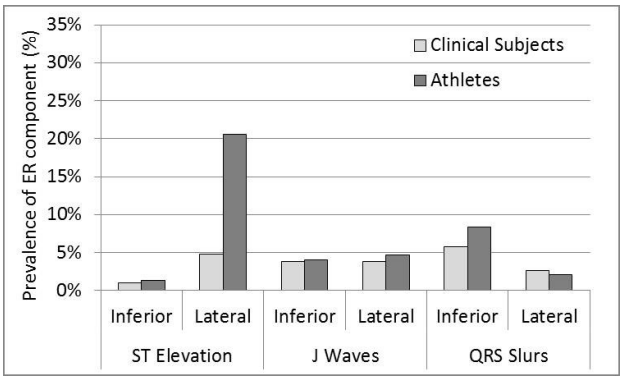

Figure 10.9. Prevalence of components of ER in clinical subjects and athletes.

analysis failed to demonstrate a risk in the younger subset, they were "enriched" by all those who died with J waves and slurs in the older segment of their population. This violated the Cox's model assumptions and results in age, not the Haïssaguerre pattern, being associated with death. The third outcome study, by Haruta et al[21] concluded that ER was only predictive of unexplained death. Although *unexplained death* was intended to be a surrogate for SCD, the main category included in this coding was unexplained accidental death.

The fourth study by Stavrakis et al[22] considered 852 consecutive patients with ST elevation ≥0.1 mV in the inferior or lateral leads from the VA ECG system and randomly selected 257 age-matched patients with normal ECGs as controls. They considered J waves or slurs and the ST elevation to be associated with a modest increased all-cause mortality compared to controls (hazard ratio of 1.5). Comparison to controls rather than the total population from the sampling period violates the assumptions of the Cox model. The fifth study was that of Rollins et al[23] with the French participants in the MONICA study. It was a retrospective study of 1161 southwestern French subjects aged 35 to 64 years. This relatively small study did not isolate the risk to the inferior leads and is therefore hard to reconcile with the larger Finnish study.

The sixth and seventh studies by Olson et al and Hisamatsu et al[24] were excellent population studies but did not consider R-wave downslope phenomena (Haïssaguerre/J-wave pattern) but only ST elevation and included the anterior leads. Uberoi et al[25] from the Veterans Affairs was one of the largest multiethnic population studies and considered R-wave downslope phenomena (Haïssaguerre/J-wave pattern) as well as ST elevation.

Two of the studies did not consider the Haïssaguerre/J-wave pattern but only reported ST elevation and should not be included. Six studies remain, but one had all-cause mortality as an end point, was really a case-control rather than a prospective study, and only reported a weak hazard ratio (1.5), leaving 5 studies for tabulation. Four of these 5 considered the Haïssaguerre pattern and found a hazard for CV and/or arrhythmic death. In tabulating the results of these studies, the impact of positive publication bias must be considered: authors and editors favor positive, exciting results over negativity. While

Table 10.4. Descriptors of the prognostic studies of "ER."

Lead Author	Year, Journal	Population Size	Female (%)	African Descent (%)	Mean Age (SD)	FU (yrs)	Nationality	Design
Tikkannen	2009, NEJM	10,864	48	0	44 ± 8	30	Finnish	community-based, prospective
Sinner	2010, PLOS	1945	51	0	35 - 45	18.9	German	MONICA, case-control, enhanced with deaths in older subjects*
Uberoi	2011, Circulation	29,281	13	13	55 ± 14	7.6	USA	clinic-based, prospective
Haruta	2011, Circulation	5976	56	0	45	24	Japan	atomic bomb survivors
Olson	2011, EHJ	15,141	56	27	54 ± 6	17	USA	ARIC population-based, prospective
Stavrakis	2012, ANEC	825 ER 255 controls	1	40	49 ± 12	6.4	USA	clinic-based, case-control
Rollin	2012, AJC	1161	48	0	50 ± 9	14.2	French	MONICA, prospective
Hisamatsu	2013, Circ Japan	7630	59	0	52 ± 4	15	Japan	National Circulatory Survey

shortcomings of these studies have been pointed out above, the majority favor a weak association of CV risk with the Haïssaguerre pattern. The average hazard ratio of approximately 3 is meaningful for a risk factor that can be modified (i.e., HBP, cigarette smoking), but does not provide adequate power for clinical decision-making, particularly when the risk may require 10 to 15 years before it manifests.

The strongest data supporting the prognostic risk of R-wave downslope phenomena (Haïssaguerre/J-wave pattern) is from the Finnish study. It demonstrates a risk only in the inferior leads and that there is no risk for the same ECG pattern in the lateral leads, where it is more common. The study suggests that the risk only is apparent after 10 to 15 years of follow-up and is an indicator of arrhythmic risk in older men with CAD. The hazard of up to 4 times has meaning for risk factors that can be modified but little value for a condition without a safe therapy.

When the Haïssaguerre study of idiopathic VT/VF is more closely examined, it becomes apparent that the Haïssaguerre pattern is only 33% sensitive for a condition with a high risk of sudden or cardiovascular death, and not very specific. The dynamic patterns with giant J waves are rare and may have different clinical implications. Provocative tools such as the Valsalva maneuver, drug challenges, further ambulatory monitoring, and exercise testing may help with further risk stratification. There may also be a better ECG marker of arrhythmic risk in phase 1 of repolarization. Certainly, the dream of identifying the cause of SCD in young individuals with normal hearts lies in this phase, but it appears that we are far from identifying it. The controversy regarding whether the Haïssaguerre/J-wave pattern is due to ER or late depolarization makes this ever more apparent.[26] Much research and careful epidemiological studies with modern ECG recording technology are very much needed.

ECG MEASUREMENT ISSUES

Before the prognostic significance of the Haïssaguerre pattern can be demonstrated, there must be agreement on precisely what measurements should be made. It appears that for stable ECG patterns with a QRS duration (including an end-QRS J wave/slur) less than 120 ms, we should follow the Computer Society of Electrocardiography (CSE) measurement statement (1985)[27] and consider the J point (also known as QRS end, J-junction, ST0 [0 ms], or ST beginning) to occur after the R-wave downslope notch/slur/or J wave as determined across all 12 leads. The measurement baseline should be set in an interval immediately preceding QRS onset as per the CSE measurement statement. Some of the bizarre and dynamic ECGs may require other rules for measurements but for now the CSE statement should be followed.

The major methodological issue in the studies is that ER and the J point is not consistently coded. Some defined ER as the presence of ST elevation (classic ER), while others defined it as the presence

Table 10.5. End points, results, and measurements used in the prognostic studies of "ER."

Lead Author	End Points	"ERP"	CVD Hazard	Measurement (1 mm)	Leads	J Waves/ Slurs	ST Elevation
Tikkannen	CV mortality, arrhythmic deaths	5.8%	2-3× inferior only	visual, 2 contig	Inf, Lat	yes	no
Sinner	CV mortality	13%	2-4×	visual, 2 contig	Inf, Lat	yes	no
Uberoi	CV mortality	14%	none	GE12SL, ST0/vis	Inf, Lat	yes	no
Haruta	CV mortality, unexpected and accidental deaths	24%	none, unexplained deaths only	visual, 2 contig	Inf, Lat	yes	no
Olson	Sudden Cardiac Death	STE 12.3%	1.2× (white females 2×)	GE 12SL ST0	Ant, Inf, Lat	no	yes
Stavrakis	all-cause mortality	NA	NA, 1.5× all cause	visual, 2 contig	Inf, Lat	yes	no
Rollin	CV mortality	13%	3 to 8× inf and lat	visual, 2 contig	Inf, Lat	yes	no*
Hisamatsu	CV mortality	STE 3.5%	2.5× anterior leads (>2 mm)	visual, any lead	Ant, Inf, Lat	no	yes

Vis, visual; contig, contiguous; GE12 L, General Electric MUSE program; ST0, ST at 0 ms; beginning of ST segment [J-point], Inf, inferior III, aVF, II; lat, lateral (V456,I,aVL); ant, anterior (V_{123}).

of R-wave downslope phenomena (J waves or slurs), which is most consistent with the definition used by Haïssaguerre (new ER). Is the resting ECG otherwise normal, as is the case for classic ER? Many subtle but important questions regarding appropriate use of the terminology remain: Is the J point the beginning of the ST segment (the classic J point) or is it the top of the J wave or slur (the new J point)? What is the QRS duration? Does it include J waves/slurs when they are present? Table 10.5 lists the definitions and measurements made by 8 studies. Even close reading of these studies does not always provide an answer to these questions.

CONCLUSIONS

The new channelopathy, preferably called the Haïssaguerre or J-wave syndrome, is a rare, new condition characterized by death during sleep and most notably the dynamic appearance of large J waves with or without ST elevation prior to idiopathic VT/VF. Unfortunately, it has been labeled "ER" by researchers, who have caused much confusion among clinicians, who have been taught that ER is physiological ST elevation occurring in an otherwise normal ECG. It is unlikely to cause SCD in athletes during exercise. Furthermore, it is unlikely that phenomena on the routine 10-second ECG will predict who will manifest idiopathic VT/VF. Cross-sectional studies suggesting a higher prevalence of J waves in patient groups is likely explained by their association with Q waves and other ECG abnormalities. All of the studies of ER, particularly the follow-up studies, are difficult to decipher because of confused semantics.[28] It is sad that the lack of consideration of established definitions used by all current automated ECG programs will probably cause more harm than good due to the "J wave- implantable cardioverter-defibrillator reflex."[29]

REFERENCES

1. Derval N, Lim H, Haïssaguerre M. Dynamic electrocardiographic recordings in patients with idiopathic ventricular fibrillation. *J Electrocardiol.* 2013;46(5):452–455.
2. Haïssaguerre M, Derval N, Sacher F, et al. Sudden cardiac arrest associated with early repolarization. *N Engl J Med.* 2008;358:2016–2023.
3. Kambara H, Phillips J. Long-term evaluation of early repolarization syndrome (normal variant RS-T segment elevation). *Am J Cardiol.* 1976;38:157–161.
4. Jayroe JB, Spodick DH, Nikus K, et al. Differentiating ST elevation myocardial infarction and nonischemic causes of ST elevation by analyzing the presenting electrocardiogram. *Am J Cardiol.* 2009;103(3):301–306.
5. Tikkanen JT, Anttonen O, Junttila MJ, et al. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med.* 2009;361:2529–2537.
6. Uberoi A, Sallam K, Perez M, et al. Prognostic implications of Q waves and T-wave inversion associated with early repolarization. *Mayo Clin Proc.* 2012;87(7):614–619.
7. Perez MV, Friday K, Froelicher V. Semantic confusion: The case of early repolarization and the J point. *Am J Med.* 2012;125(9):843–844.
8. Viskin S. Idiopathic ventricular fibrillation "Le Syndrome d'Haïssaguerre" and the fear of J waves. *J Am Coll Cardiol.* 2009;53(7):620–622.
9. Kukla P, Jastrzebski M. Haïssaguerre syndrome – a new clinical entity in the spectrum of primary electrical diseases? *Kardiol Pol.* 2009;67(2):178–184.
10. Froelicher V, Wagner G. Co-editors of symposium issue on J wave patterns and a syndrome. *J Electrocardiol.* 2013;46:381–382.
11. Leo T, Uberoi A, Jain NA, et al. The impact of ST elevation on athletic screening. *Clin J Sport Med.* 2011;21(5):433–440.
12. Thygesen K, Alpert JS, Jaffe AS, et al. Joint ESC/ACCF/AHA/WHF task force for the universal definition of myocardial infarction. *Eur Heart J.* 2012;50(184):2173–2195.
13. Adhikarla C, Boga M, Wood AD, Froelicher VF. Natural history of the electrocardiographic pattern of early repolarization in ambulatory patients. *Am J Cardiol.* 2011;108(12):1831–1835.
14. Stein R, Sallam K, Adhikarla C, et al. Natural history of early repolarization in the inferior leads. *Ann Noninvasive Electrocardiol.* 2012;17(4):331–339.
15. Muramoto D, Singh N, Aggarwal S, et al. Spectrum of ST amplitude: athletes and an ambulatory clinical population. *J Electrocardiol.* 2013;46(5):427–433.
16. Perez M, Fonda H, Le VV, et al. Adding an electrocardiogram to the pre-participation examination in competitive athletes: A systematic review. *Curr Probl Cardiol.* 2009;34(12):586–662.
17. Perez MV, Friday K, Froelicher V. Semantic confusion: The case of early repolarization and the J point. *Am J Med.* 2012;125(9): 843–844.
18. Surawicz B, Macfarlane PW. Inappropriate and confusing electrocardiographic terms: J-wave syndromes and early repolarization. *J Am Coll Cardiol.* 2011;57(15):1584–1586.
19. Tikkanen JT, Anttonen O, Junttila MJ, et al. Long-term outcome associated with early repolarization on electrocardiography. *N Engl J Med.* 2009;361(26):2529–2537.
20. Sinner MF, Reinhard W, Müller M. Association of early repolarization pattern on ECG with risk of cardiac and all-cause mortality: a population-based prospective cohort study (MONICA/KORA). *PLoS Med.* 2010;7(7):e1000314.
21. Haruta D, Matsuo K, Tsuneto A. Incidence and prognostic value of early repolarization pattern in the 12-lead electrocardiogram. *Circulation.* 2011;123(25):2931–2937.

22. Stavrakis S, Patel N, Te C, et al. Development and validation of a prognostic index for risk stratification of patients with early repolarization. *Ann Noninvasive Electrocardiol.* 2012;17(4):361–371.
23. Rollin A, Maury P, Bongard V, et al. Prevalence, prognosis, and identification of the malignant form of early repolarization pattern in a population-based study. *Am J Cardiol.* 2012;110(9):1302–1308.
24. Hisamatsu T, Ohkubo T, Miura K, et al. Association between J-point elevation and death from coronary heart disease. *Circ J.* 2013;77(5):1260–1266.
25. Uberoi A, Jain NA, Perez M, et al. Early repolarization in an ambulatory clinical population. *Circulation.* 2011;124(20):2208–2214.
26. Borggrefe M, Schimpf R. J-wave syndromes caused by repolarization or depolarization mechanisms: A debated issue among experimental and clinical electrophysiologists. *J Am Coll Cardiol.* 2010;55(8):798–800.
27. The CSE Working Party. Recommendations for measurement standards in quantitative Electrocardiography. *Eur Heart J.* 1985;6:815–825.
28. Froelicher V. Early repolarization redux: the devil is in the methods. *Ann Noninvasive Electrocardiol.* 2012;17(1):63–64.
29. Martini B, Wu J, Nava A. A rare lethal syndrome in search of its identity: sudden death, right bundle branch block and ST segment elevation. In: Wu Y, Wu J, eds. *Sudden Death: Causes, Risk Factors and Prevention.* New York, NY: Nova Science Publishers Inc.; 2013:2–12.