SPECIAL REPORT

The Year of 2011 in Electrocardiology

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EARLY REPOLARIZATION (ER) PATTERN IN THE ECG

Undoubtedly, last year the subject of ER raised the most vivid discussion among electrocardiographers and electrophysiologists. Based on a follow-up of about 10 years of over 15,000 middle-aged subjects, Olson et al.¹ demonstrated that the characteristic ST-elevation at the J-point was associated with an increased sudden cardiac death (SCD) in whites and in females, but not in blacks or males.

Japanese investigators² reviewed the ECG records of nearly 6000 atomic bomb survivors who were examined at least once during biannual health examinations in Nagasaki between 1958 and 2004; ER pattern had an elevated risk of unexpected death and a decreased risk of cardiac and all-cause death, although the hazard ratio for unexpected death in ER pattern was lower than that in patients with Brugada (Br)-type ECG.

The characteristics of the ECG manifestations of ER in a general population was scrutinized by Tikkanen et al.;³ subjects with ER ≥ 0.1 mV and horizontal/descending ST-variant had an increased hazard ratio of arrhythmic death; an even higher hazard ratio of arrhythmic death was observed with higher amplitude ER in inferior leads and horizontal/descending ST-segment variant. In subjects with ascending ST, the relative risk for arrhythmic deaths was not increased.

The heritability basis of ER pattern in the general population was stressed by Noseworthy et al.,⁴ al-

though not ruling out that the increased incidence of the ER pattern among siblings of ER-positive subjects could arise in part from confounders, such as age, sex, or exercise. The same group of investigators⁵ demonstrated ER pattern in the inferior and lateral territory in about 25% of competitive athletes. As its prevalence increased significantly after intense physical training, an association between exercise and ER pattern can be assumed, but without suggesting that exercise training increases the risk of life-threatening arrhythmias. The ER pattern in the study was associated with black race, increased QRS voltage, and slower heart rate.

By scrutinizing nearly 30,000 ECG records with a median follow-up of over 70 years, Uberoi et al.⁶ demonstrated a decreased cardiovascular mortality (although not significant after adjustment for age) of individuals with ER, irrespective of the different patterns and components of this pattern. The same group of these investigators⁷ described a loss of the ER pattern over 10 years in more than half of their young clinical cohort.

In 100 patients with apparently unexplained cardiac arrest and preserved ejection fraction, Derval et al.⁸ found significant ER in 19 patients; J-point elevation in idiopathic ventricular fibrillation (VF) patients had higher amplitude and wider distribution than those with an established cause of cardiac arrest. Nunn et al.⁹ studied families with arrhythmic SCDs for the presence of J-point elevation in the inferior and lateral leads and found it to be more prevalent in them than in controls; according to the authors

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this indicated that ER is an important potentially inheritable proarrhythmic trait or marker of proarrhythmia in SCD. ER pattern was found more frequently by Watanabe et al.¹⁰ in patients with a short QT interval with sudden death and/or arrhythmias than in those without. The same group of investigators¹¹ studied 50 patients with ER and VF and found that with these patients heart rate was slower and the PR interval and QRS duration were longer in patients with idiopathic VF and loss-of-function mutations in *SCNSA*, supporting the hypothesis that decreased sodium current enhances VF susceptibility.

The ER syndrome and the Br syndrome, although differing with respect to the magnitude and lead location of the abnormal J-wave manifestation, represent a continuous spectrum of phenotypic expression, according to Antzelevitch et al.¹² and justifies thus the use of the terms *J-wave syndromes* and *ER*. The same group of investigators¹³ expressed their view that ER pattern predominantly in the inferior or inferolateral leads is associated with a certain level of risk, although ER pattern globally in the inferior, lateral, and right precordial leads, is associated with the highest level of risk for development of malignant arrhythmias and is often associated with VF storms. In contrast to this, Surawicz and Macfarlane¹⁴ expressed their view, that the use of both terms, the *J-wave syndromes* and the *ER*, are "inappropriate and confusing" and according to them, "J-point elevation" expresses sufficiently this condition and terminal QRS abnormalities can be adequately described as fragmentation, slurring, or notching.

BR SYNDROME

As known, although the ECG hallmark of the coved type ST-elevation with right bundle branch block (RBBB) pattern (type 1) seems to be quite clearly recognized as Br syndrome, the types 2 and 3 patterns require antiarrhythmic drug challenge to be unmasked and converted to type 1. For discrimination between the types 2 and 3 patterns and incomplete RBBB, Chevallier et al.¹⁵ proposed a careful examination of the angle between a vertical line and the downslope of the R wave (α) and the angle between the upslope of the S wave and the downslope of the R-wave (β). All their patients underwent ajmaline infusion: a positive result of the test (conversion to type 1) was associated with a mean β angle significantly larger as compared to the patients with negative tests; the optimal cutoff

value of 58° yielded a positive predictive value for the appearance of type 1 of 73% and a negative predictive value of 87%.

Brugada¹⁶ assumed that a $\geq 58^\circ$ of the β angle combined depolarization and repolarization abnormalities, probably related to a mutation of the cardiac sodium channel gene or to somatic mutations of the neural crest cells. Still, antemortem ECGs in cases of sudden death due to Br syndrome did not demonstrate a type 1 ECG pattern, suggesting that current risk factors in Br syndrome are insufficient to foretell SCD events in those affected.¹⁷

Take et al.¹⁸ found that in patients with Br syndrome, changes in ST level ≥ 0.2 mV were more frequent in patients with VF than without. Spontaneous ECG alterations and appearances of types 1 and 0 ECGs were more frequent in the VF+ group than in the VF- group ($P < 0.01$). These ECG alterations were associated with significant action potential heterogeneity that can initiate VF in these patients.

ARRHYTHMIAS

Differentiating between left ventricular (LV) from right ventricular (RV) outflow tract (OT), tachycardia is a challenge and Betensky et al. who developed a surface ECG measurement, the "V₂ transition ratio," for distinguishing LVOT from RVOT origin in patients with left bundle branch pattern idiopathic VT/premature ventricular contractions (PVCs) with lead V₃ precordial transition.¹⁹ A V₂ transition ratio of ≥ 0.6 predicted an LVOT origin with 95% sensitivity and 100% specificity; according to the authors, this simple ECG measurement will certainly be helpful when planning an ablation strategy. Studying idiopathic RVOT or PVCs originating from the RVOT, Yamashina et al.²⁰ demonstrated J waves to be present more commonly in these patients than in matched control subjects; however, the significance of the J waves was limited as far as the prognosis and VF were concerned.

Yoshida et al.²¹ studied the characterization of postinfarction ventricular tachycardias (VTs) which involved in 45% of the patients the interventricular septum; VTs involving the Purkinje fiber system had the narrowest QRS complexes and displayed a RBBB morphology. Intramural VTs had a left BBB (LBBB) with a QS complex in V₁ and a broad R wave in lead V₂. Endocardial VTs from the interventricular septum had the broadest QRS width.

Single channel ECGs recorded from 1968 through 1971 for the Framingham Heart Study of patients ≥ 60 years old showed over a median follow-up of 15.8 years no significant associations between P-wave dispersion with incidence of atrial fibrillation (AF) or mortality; however, maximum P-wave duration at the upper fifth percentile was associated with long-term AF risk in this community-based cohort.²²

A novel "cardiac rotation-corrected" transitional zone index was described by Yoshida et al.²³ who found it a useful marker for differentiating ventricular arrhythmias (VAs) with RVOT origin than with aortic sinus cusp origin.

In a canine model, Nishida et al.²⁴ demonstrated that chronic occlusion of coronary arteries supplying atrial tissues inducing ischemia/infarction creates substrates for both spontaneous ectopy and sustained reentry, promoting thus both AF triggers and the substrate for AF maintenance. Idiopathic VAs could be differentiated from scar-related LBBB VAs, identifying the latter if one of the following three criteria is met: a precordial transition beyond V_4 , or notching of the S-wave downstroke in lead V_1 or V_2 , or a duration from the onset of QRS to the S-nadir in $V_1 > 90$ ms.²⁵ In the acute stage of myocardial infarction 10-minute-long ECG recordings were extracted, one right before VT onset and another at least 1 hour before it. It was demonstrated that increased premature activations and unstable QT intervals precede the VT.²⁶

MYOCARDIOPATHIES

ECG criteria helped distinguish VA originating from arrhythmogenic RV dysplasia/cardiomyopathy (ARVD/C) from those with RVOT tachycardia (RVOT-VT).²⁷ The authors found that precordial transition at lead V_6 was exclusively seen in ARVD/C patients. QRS duration of ≥ 120 ms in lead I was sensitive for the diagnosis of ARVD/C whereas the presence of notching in the QRS and precordial transition at lead V_6 are specific for the diagnosis of ARVD/C. In idiopathic dilated cardiomyopathy patients with LV dysfunction, a fragmented QRS had a high predictive value for the combined end point of all-cause mortality and ventricular tachyarrhythmias.²⁸

Takotsubo cardiomyopathy could be differentiated from anterior ST-elevation myocardial infarction in its acute stage according to the magnitude of the ST-segment elevation measured at the J-point,

as in the former significantly lower prevalence of ST-elevation ≥ 1 mm in lead I was observed as compared to the patients with infarction. ST-elevation ≥ 1 mm in leads V_{3-5} without ST-elevation in V_1 identified this type of cardiomyopathy.²⁹

In patients with nonischemic dilated cardiomyopathy, Ikeda et al.³⁰ demonstrated that heart rate turbulence on 24-hour ECGs was a powerful index for cardiac mortality and sustained VTs during a follow-up of over 1 year.

ISCHEMIA AND INFARCT DETECTION AND PROGNOSTIC SIGNIFICANCE

Based on inverse calculation of heart-surface potentials from the body-surface ECGs, Horacek et al.³¹ proved that 12-lead ECG might provide useful estimates of the regions of myocardial ischemia that agree with those provided by scintigraphic techniques. Patients with an increased Selvester QRS score for scar size, based on 12-lead ECG, had during a follow-up for a median of 45.5 months significantly more VT/VF, than those with no scar.³²

New criteria for the diagnosis of multiple myocardial infarctions were derived from studies by Boineau³³, using ECGs and body surface potential distribution maps. As according to classical Q- and R-wave criteria, myocardial infarctions located in opposing walls are difficult to identify, the author replaced the opposing pathologic Q waves by multiple QRS deflections or baseline crossings, which are equally weighted in his scoring system, and used also to predict myocardial infarctions in LBBB, intraventricular conduction delay, and ventricular pacing.

In Japanese individuals,³⁴ 24-year follow-up demonstrated that ST-segment depression and/or T-wave abnormalities on the resting ECG, with or without left high R waves, are independent predictors of cardiovascular disease.

THE P WAVE

Rowlands et al.³⁵ found significant correlations between wavelet-based features extracted from the beat-averaged P wave and the left atrial diameter, area and volume index measures obtained from echocardiogram and cardiac magnetic resonance, demonstrating thus that the wavelet technique from the P wave may provide a good estimate of the size of the left atrium. In the National

Health and Nutrition Examination Survey, Magnani et al.³⁶ scrutinized several P-wave indices and demonstrated that P-wave duration is significantly associated with increased cardiovascular and all-cause mortality.

Militaru, Donoiu, and Ionescu³⁷ demonstrated that in normal population the filtered P wave duration (PWD) was higher in men and in patients with recurrent persistent AF. The root mean square (RMS) in the last 40 ms (RMS₄₀) and last 30 ms (RMS₂₀) were significantly correlated with age and P-wave duration. Patients with recurrent paroxysmal AF had higher PWD and lower RMS₄₀, RMS₃₀, and RMS₂₀.

A new filtering technique of the T wave described by Goldwasser et al.³⁸ helping to detect P waves hidden in the previous T wave and helped to distinguish between atrioventricular (AV) nodal reentry tachycardia and AV reentry tachycardia.

THE USE OF HOLTER METHOD

Abisse et al.³⁹ using three standard bipolar Holter leads, studied patients with subsequent life-threatening ventricular tachyarrhythmias during 3–4 years of follow-up and patients without. During mental stress and poststress recovery, Δ T-wave amplitude was almost 2-fold greater in the former than in the latter group, which may signal proarrhythmic repolarization response.

Holter monitoring-detected microvolt T-wave alternans in hypertrophic cardiomyopathy patients with NYHA functional class I or II was a useful predictor of nonsustained VT (nVT), but it did not improve the arrhythmia prediction beyond that obtained by other indices; postexercise nVT episodes had better sensitivity, specificity, and predictive values than spontaneous nVT episodes.⁴⁰

On 7-day continuous ECG monitoring in over 6000 patients with acute coronary syndromes without ST-elevation, 5.3% had both ST-depression of ≥ 1 mm lasting ≥ 1 minutes and VT. During a nearly 1 year follow-up, patients with either VT or ischemia were at increased risk of cardiovascular outcomes and a combination of the two identified a particularly high-risk population.⁴¹

In the GISSI-HF study,⁴² 24-hour Holter recordings were analyzed using a template-matching, semiautomatic algorithm to measure QT and heart rate time series in sequential 5-minute epochs over 24 hours; after a median follow-up of nearly 4 years a significant association was found between

total and cardiovascular mortality in patients with chronic heart failure.

By analyzing Holter recordings, Razak et al.⁴³ although using a relatively low sampling frequency (125 or 128 Hz), demonstrated that QT interval duration is strongly affected by noninstantaneous heart rate, dating back more than 60 seconds; this “memory affect” decays exponentially with time constant of 1 minute providing an accurate description of QT adaptation.

MISCELLANEOUS

According to Zareba et al.,⁴⁴ the benefit of cardiac resynchronization therapy in class I or II heart failure patients and EF $\leq 30\%$ is limited to those who exhibited an LBBB QRS pattern; no clinical benefit was observed in those with an RBBB or intraventricular conduction disturbances.

Gollob, Redpath, and Roberts⁴⁵ proposed new scale for the diagnosis of short QT syndrome (SQTS). On their scale, QTc ≤ 330 ms carries three points, QTc ≤ 350 ms, history of SCD, documented polymorphic VT/VF, first-/second-degree relative with high-probability SQTS, and a positive genotype (if the patient has ≤ 370 ms of QTc), each one carries two points, QTc ≤ 370 ms, J-point-T-peak interval < 120 ms, and some other clinical, family, and genetic information each one carries 1 point; patients with ≥ 4 points have high-, patients with 3 points have intermediate-, and patients with ≤ 2 points have low-probability SQTS.

In patients with Chagas' disease, Strauss et al.⁴⁶ described good correlation between 12-lead ECG scar quantification and magnetic resonance scar size; this makes ECG an attractive screening tool which may enhance clinical risk stratification of Chagas patients. In patients with cardiac sarcoidosis, signal-averaged electrocardiography disclosed a significant association of low amplitude signal duration > 42 with this diagnosis.⁴⁷

Ye et al.⁴⁸ studied a general population of over 1000 patients with sudden death, some of them resuscitated. A majority of patients for whom initial ECG rhythm was available had VT/VF. A prolonged T-peak to T-end interval in lead V₅ of the 12-lead ECG was found to be a significant predictor of SCD.⁴⁹ Independent component (IC) analysis obtained from digital ECG data from 10 channels using 20 surface electrodes in patients with genetically confirmed LQTS type 1 and normal subjects was done by Horigome et al.⁵⁰ who demonstrated

extra ICs during repolarization in all patients with LQT1, but none in normal subjects.

Premature supraventricular and ventricular contractions were more frequent, heart rate variability was decreased, and heart rate turbulence onset was abnormal in workers occupationally exposed to lead.⁵¹

In patients with atriofascicular or decremental atrioventricular pathways with absent delta waves, a high prevalence of terminal QRS slurring or notching was demonstrated by Liao et al.⁵² the authors stressed that these can be the sole manifestation of such accessory pathways during sinus rhythm.

Over 10,000 middle-aged apparently healthy subjects were followed by Finnish investigators for 30 ± 11 years. Prolonged QRS duration ≥ 110ms, without the criteria of complete or incomplete BBB and LV hypertrophy, in a standard 12-lead ECG was associated with increased all-cause and cardiac mortality and arrhythmic SCD; intraventricular conduction delay was most strongly associated with an increased risk of arrhythmic death.⁵³

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