SPECIAL REPORT

The Year of 2007 in Electrocardiology

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Upon receiving the most prestigious invitation from the editor of this journal to review last year's most outstanding publications in electrocardiology, I was not at all sure that my search would produce 50 innovative articles as it did when critically reviewing the years 2005 and 2006.^{1,2} I was wrong. Most notable advances were again made in the realm of electrocardiology during this past year and it was even more difficult to limit the number of meritable publications to 50. Omissions were inevitable and I wish to apologize in advance to the authors who don't find their articles quoted. Together, let's look forward to similarly original and novel publications in 2008 as well.

ACUTE CORONARY SYNDROMES (ACS)

The consideration of both ST-segment elevation and depression in the standard 12-lead ECG recording significantly increased the sensitivity for the detection of acute myocardial infarction (MI) with only a slight decrease in the specificity.³ ACS patients with ST-segment shifts during the first 24 hours, documented by continuous monitoring, had a higher risk of death or MI; this prognostic information was incremental beyond the validated GRACE risk score and identified high-risk patients who benefit from early revascularization.⁴ ST-segment analysis by fully automated wireless ECG transmission network decreased door-to-intervention time in the STAT-MI investigation.⁵ A

more detailed ECG analysis, involving also Q- and T wave morphology, was found to be useful for rapid identification of high-risk patients in whom every effort should be made to transfer for primary intervention, or vice versa, for identifying low-risk patients in whom fibrinolytic therapy might be an alternative option.⁶ In non-ST-elevation ACS patients and an elevated troponin T the $\sum$ ST-segment deviation (depression) of ≥ 1 mm were an increased risk of death or MI.⁷ The importance of terminal QRS distortion, defined earlier by Birnbaum et al.* as grade 3 ischemia, was shown to be by the author's group to predict failure of ST-segment resolution after primary percutaneous coronary intervention for acute myocardial infarction.⁸ Green and coworkers⁹ found that in emergency situations leads III, aVL, and V₂ are sufficient for computerized prediction of ACS. On the other hand, Perron and associates¹⁰ advocated the addition of 7 leads, V₁, -V₂, -V₃, -aVL, -I, aVR, and III.

ST elevation on postdischarge ambulatory Holter monitoring in ST elevation MI patients was an independent predictor of mortality and nonfatal major cardiovascular events treated with fibrinolysis.¹¹ The importance of strict adherence to ECG entry criteria (degree of ST elevation, presence of ST depression, number of leads, new LBBB) in patients investigated for ST-elevation MI was stressed by Tjandrawidjaja and coworkers.¹²

Isolated right ventricular MI developed in a patient during percutaneous coronary intervention and the ECG showed ST elevation in inferior leads II, III, and aVF.¹³

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*Birnbaum Y, Mahaffey KW, Criger DA, et al. Grade III ischemia on presentation with acute myocardial infarction predicts rapid progression of necrosis and less myocardial salvage with thrombolysis. *Cardiology* 2002;97:166–174.

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THE QT INTERVAL

The morphology of the QT interval, that is, $T_{\text{peak}} - T_{\text{end}}$ interval, together with the QTc correlated with the risk of torsade de pointes.¹⁴ In 100% of patients with transmural ischemia QTc interval prolongation was the earliest ECG abnormality observed, suggesting that prolongation of repolarization might figure prominently in the ischemic cascade.¹⁵ In a prospective study, QT variability strongly predicted sudden cardiac death in asymptomatic subjects with mild or moderate left ventricular systolic dysfunction.¹⁶

Arnestad and coworkers¹⁷ demonstrated that ECG screening between the third and fourth weeks of life can probably identify most of the infants at risk for sudden death caused by LQTS, either in infancy or later on in life, which would thus allow the institution of preventive measures. The cost-effectiveness of neonatal ECG screening was questioned by another well-respected group of investigators.¹⁸ Gardiner and coworkers¹⁹ used a new technique of real-time fetal ECG measurement of fetal heart rate, electric PR interval, and analysis of various other waveforms and time intervals. Both electric PR interval recordings and Doppler mechanical PR intervals were good predictors of atrioventricular block in anti-Ro positive pregnancies. Etheridge et al.²⁰ studied the diagnosis and treatment of children with long QT syndrome, with a mean QTc of 487 ± 39 ms. During a mean follow-up of 3.5 years patients treated with a device, mainly dual-chamber devices and about one-third of them with ICDs, had a longer QT interval and were more symptomatic than the medically treated children.

Active and passive mental stress was shown to induce QT-interval prolongation and T-wave notching, reflecting sympathoadrenergic effects on myocardial repolarization.²¹ An interesting observation, based on a retrospective analysis of 24,370 ECGs showed that in males, but not in females, ≥ 18 years of age with a normal baseline ECG, the QTc interval was longest in October.²²

An important step toward a mutation-specific risk stratification was taken by Crotti and coworkers²³ who identified a common LQT1-causing mutation with high clinical severity, independently of the ethnic origin of the families. The identification of this mutation and possibly others in the future may improve risk stratification and management of this syndrome.

With all the excellent information becoming available about the correct ways to measure and to identify abnormal QT intervals, Taggart and coworkers²⁴ found diagnostic discordance to be present in about two-thirds of patients who sought a second opinion at their group for a suspected LQTS. Dr. Vetter* in her editorial comment stressed the importance to make the correct diagnosis and advocated for probable LQTS patients to start a beta-blocker, obtain an automated external defibrillator for the home and school, and continue an ongoing evaluation, including additional clinical and genetic testing.

CONGENITAL HEART DISEASE

The "wealth of information" the ECG provides to the clinical assessment of adults with congenital heart disease was reemphasized by Khairy and Marelli,²⁵ describing the pathogenomonic findings in the more frequently encountered congenital defects in adults and summarizing variations in the location of the sinus node, AV node, and His-Purkinje system relevant to ECG findings.

BAROREFLEX SENSITIVITY (BRS)

Ten to 14 weeks following a myocardial infarction, risk-assessment by 24-hour high-resolution Holter monitoring showed in patients with mildly reduced LV function that impaired BRS, heart rate turbulence, and abnormal TWA each predicted a significantly higher independent risk of the primary and secondary outcomes.²⁶ A depressed BRS 4–6 weeks in low-risk post-MI patients was found to identify, independently of age, a subgroup at long-term high risk for subsequent cardiovascular mortality.²⁷ Dr. Sleight** in his editorial commenting on these two papers stressed that BRS measurements, whether using phenylephrine-induced or spontaneously occurring blood pressure and RR-interval

*Vetter VL. Clues or miscues? How to make the right interpretation and correctly diagnose long-QT syndrome. *Circulation* 2007;115:2595–2598.

**Sleight P. New methods for risk stratification in patients after myocardial infarction. Autonomic control and substrate sensitivity. *J Am Coll Cardiol* 2007;50:2291–2293.

changes or indexes of HRV, the results are applicable in today's clinical context.

T-WAVE ALTERNANS (TWA)

In heart failure patients Cox and coworkers used spectral and modified-moving average analyses of TWA and found it to be a predictor of subsequent cardiac arrest on long-term prospective follow-up.²⁸ Microvolt TWA status was an effective tool in identifying patients, followed up for 27 ± 12 months, who most and least likely benefit from ICD therapy, with potential policy implications for ICD coverage.²⁹ While the high negative predictive value of this test was stressed in an editorial comment accompanying this study by Drs. Russo and Marchlinski,* it has also called this test only "promising" in predicting patients who might be most likely to benefit from ICD therapy. In heart failure patients with nonischemic cardiomyopathy an abnormal TWA test was associated with a fourfold higher risk of cardiac death and life-threatening arrhythmias over a 19-month follow-up.³⁰ By reclassifying an unsustained MTWA as a negative and the presence of ectopy and inability to reach adequate heart rate as a positive test, Chan and coworkers improved the predictive power of this test.³¹ Time domain TWA record during an exercise test powerfully predicted mortality in a general population.³²

In patients with ischemic and nonischemic cardiomyopathy, Selvaraj and coworkers³³ compared surface ECG TWA with intracardiac repolarization alternans; the spatial and temporal concordances found in this study seem to be a first step to link TWA to human ventricular arrhythmias.

THE PROBLEM OF ATHLETES

The search, together with the controversy, continues for test/s for the identification of athletes prior to their participation in competitive sport activities who are at high risk with potentially lethal arrhythmias and at danger of sudden cardiac deaths. An important investigation was published in 2007 concerning the implementation of 12-lead ECG for screening of young athletes:

working upon a program established by law and implemented in Italy since 1982, Pelliccia and coworkers³⁴ published that in a large and unselected population of young individuals undergoing cardiovascular screening, the prevalence of markedly abnormal ECG patterns suggestive for structural cardiac disease is low (about 5%), and should not represent obstacle for implementation of 12-lead ECG in the preparticipation screening program. The authors suggested that athletes with abnormal ECGs merit continued clinical surveillance. Prompted by this investigation, Drs. Corrado and McKenna** in their editorial comment, praised the Italian experiment and emphasized that it provides adequate sensitivity and specificity for detection of athletes affected by potentially dangerous cardiomyopathy or arrhythmia at risk of athletic field death and its implementation led to a reduction of mortality of young competitive athletes by about 90%, mostly by preventing sudden death from cardiomyopathy. These and other relevant investigations prompted discussions in the American literature whether ECGs should or should not be included in routine preparticipation screening of young athletes.

A decrease in QRS amplitude was observed following 21 months of intensive training in juvenile female athletes³⁵; this finding is in contrast with the classical hypothesis on the ECG diagnosis of left ventricular hypertrophy (LVH) and is in agreement with an alternative hypothesis on the relative voltage during the early stage of LVH development.

The prevalence of prolonged QTc in elite athletes was found to be 0.4% by Basavarajaiah and coworkers.³⁶ According to these authors, in this population a QTc of >500 ms is highly suggestive of LQTS, while a QTc of <500 ms in the absence of symptoms or familial disease is unlikely to represent LQTS.

ECG FOR PREDICTION

Five quantitative ECG measures: higher ventricular rate, longer PR interval, longer QRS duration, lower or higher Sokolow-Lyon voltage, and more negative ST-segment slope, were found by Lauer and coworkers³⁷ to be predictive of long-term outcome in patients, 74% males, who underwent CABG for severe coronary artery

*Russo AM, Marchlinski FE. Should microvolt T-wave alternans be utilized routinely in selecting patients for prophylactic implantable cardioverter-defibrillator insertion in the setting of ischemic heart disease. *J Am Coll Cardiol* 2007;49:59–61.

**Corrado D, McKenna WJ. Appropriate interpretation of the athlete's ECG saves lives as well as money. *Eur Heart J* 2007;28:1920–1922.

disease. Based on data on about 15,000 patients from the Women's Health Initiative (WHI), in asymptomatic postmenopausal women, clinically relevant baseline and incident ECG abnormalities were found to be independently associated with increased risk of cardiovascular events and mortality; the information was incremental to the established methods of risk stratification.³⁸ Interestingly, there were no significant interactions between hormone treatment and ECG abnormalities for risk prediction of cardiovascular end points.

In a predominantly male VA population, among 45,000 examinees, the 3.8% who had premature ventricular contractions (PVCs) on the routine resting ECG had an adverse prognosis of all-cause and cardiovascular mortality even if they had an otherwise normal baseline tracing. The combination of PVCs with an increased heart rate dramatically increased mortality.³⁹

Circadian variability analyses of Holter-derived RR and QT intervals were independent predictors of cardiac death in patients with stable heart failure, prognostic informations beyond those provided by the 24-hour averages of these parameters.⁴⁰ Fragmented QRS was an independent predictor of cardiac events in patients with coronary artery disease and was associated with significantly lower event-free survival for a cardiac event on long-term follow-up.⁴¹ A prominent R wave in lead aVR (aVR sign) was a risk factor for life-threatening arrhythmic events in patients with Brugada syndrome.⁴² Syncope, cardiac arrest, and sudden death were predicted by a longer QTc interval, together with female gender, LQT2 and cardiac events during childhood and adolescence.⁴³ In nonischemic cardiomyopathy patients, Iacoviello and coworkers found that nonsustained ventricular tachycardia and steeper QT/RR slope identified patients at particularly high risk for arrhythmic events over a mean follow-up of 39 months.⁴⁴

VENTRICULAR ASSYNCHRONY

The linkage between QRS duration and mechanical dyssynchrony was reevaluated by Haghighi and coworkers⁴⁵ who described that although both interventricular and intraventricular dyssynchrony increased with the increasing QRS duration, this correlation was weak. The incidence of dyssynchrony with a narrow QRS complex was found to be around 30%.⁴⁶ Diastolic and/or systolic asyn-

chrony was common in 61% of diastolic heart failure patients despite a narrow QRS complex.⁴⁷

MISCELLANEOUS

Regression of ECG parameters of left ventricular hypertrophy during antihypertensive therapy was associated with reduction in sudden cardiac deaths.⁴⁸ J wave ("the Osborne wave") was seen in a case where it was induced probably by acute ischemia.⁴⁹ Changes in P wave morphology and the frontal plane atrial vector were noted to be caused by atrial septal aneurysm in an asymptomatic woman.⁵⁰ A terminal deflection within or at the end of QRS complex, an epsilon wave, in V₁₋₃ with T wave inversion in these leads, was observed in a patient with arrhythmogenic right ventricular dysplasia; electrophysiological study showed that post-systolic or late fractionated electrogram temporarily correlated with the epsilon wave on the surface lead V₁.⁵¹

Finlay and coworkers⁵² demonstrated that the use of a lead selection algorithm is enhancing the 12-lead ECG and indicated also that repositioning just two precordial leads can provide the same level of information capture as that observed when all precordial leads are optimally placed.

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