Early Repolarization Revisited

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For more than 60 years, physicians have been fascinated by a peculiar electrocardiographic pattern called “early repolarization.” When Google is queried, more than 1 million hits turn up on this subject. Although Boineau recently described the electrocardiographic features of early repolarization as being quite diverse, such features have one factor in common: slurring or notching that produces a positive hump, called a J wave, is found at the junction at the end of the QRS complex and the beginning of the ST segment. However, the location of the electrocardiographic leads showing the J wave may vary among patients, and dynamic changes may occur in the width and height of the wave. The J wave may show circadian changes, may not always be present, and usually disappears during exercise. The lead with the J wave commonly shows ST-segment elevation. The T wave can be negative, and the QRS width and QT time may be shorter than normal.

From an epidemiologic perspective, electrocardiographic features of early repolarization are commonly present in 2 to 5% of the population and are found mostly in men, young adults, athletes, and dark-skinned persons. Usually such changes, which can be easily differentiated from those caused by cardiac ischemia, have been considered to be benign, although isolated case reports, mostly from Asia, have mentioned such changes in the QRS–ST junction in men with idiopathic ventricular fibrillation.3–8

In this issue of the Journal, two groups of investigators focus attention on junctional changes in a large group of patients with otherwise normal hearts who were resuscitated after life-threatening ventricular arrhythmia. In a study involving 22 tertiary care arrhythmia centers, Haïssaguerre et al.9 found junctional changes on electrocardiography in 64 of 206 patients (31%) who were resuscitated after idiopathic ventricular fibrillation. These junctional changes consisted of slurring or notching at the end of the QRS complex and were confined to inferolateral leads. In a letter to the editor, Nam et al.10 describe similar junctional changes in 9 of 15 Korean patients (60%) with idiopathic ventricular fibrillation, but the authors do not state in which leads the changes were seen. Although Nam et al. do not provide information regarding the age and sex of their patients, in the study by Haïssaguerre et al., the mean (±SD) age of patients with idiopathic ventricular fibrillation was 35±13 years, and 46 of the 64 patients with this condition (72%) were male. All the patients who were described in the cited case reports3–8 were male, and 12-lead electrocardiography, when reported, showed the junctional changes in the inferolateral leads.

One question that arises is whether the described abnormality at the end of the QRS complex is indeed early repolarization or, rather, delayed activation of the inferolateral area. A late potential coincident with the terminal QRS abnormality was found on signal averaging in the patient described by Garg et al.3 However, in the study by Haïssaguerre et al. late potentials were found in only 5 of 44 patients (11%) in whom...
signal-averaged electrocardiography was performed. Disappearance of the junctional changes during exercise and infusion of isoproterenol also favors the diagnosis of early repolarization. In both studies reported in this issue and in the case report by Shinohara et al., a marked accentuation of the J wave was observed, a change that was followed by ventricular ectopic activity immediately preceding the ventricular tachyarrhythmia. Of interest is the observation that ectopic ventricular activity seemed to originate in the same area as the J wave.9

The mechanisms of the junctional changes and of the initiation and perpetuation of the ventricular tachyarrhythmia seen in these patients are not clear. Gussak and Antzelevitch11 investigated the cellular and ionic changes occurring in early repolarization and discussed the presence of possibly arrhythmogenic mechanisms. Boineau12 pointed to the possibility of deep invagination of Purkinje fibers to a subepicardial level, resulting in increased transmural activation followed by earlier repolarization. This shortening in the time of transmural activation is favored by increased trabeculation of the ventricular wall.12 Local disparity in the duration of the refractory period could favor reentrant arrhythmias. The various possible mechanisms have to be evaluated with the use of molecular techniques, morphologic imaging of the area showing the described junctional changes, and activation and voltage mapping on the epicardial surface of the suspicious area. Genetic profiling should be performed in familial cases. Careful autopsy studies of the ventricular area corresponding to the electrocardiographic changes might also be helpful.

How should the information presented by Haïssaguerre and Nam and their colleagues be applied? It is obvious that the junctional changes that are described as early repolarization are common and that the unfortunate reported events are quite rare. When confronted with a patient with ventricular tachyarrhythmias or syncope, the practitioner must rule out all ischemic and nonischemic causes, including the long-QT syndrome, the short-QT syndrome, the Brugada syndrome, and arrhythmogenic right ventricular dysplasia. Once these conditions have been excluded, it is clear that slurring or notching at the junction between the QRS and the ST segment in inferolateral leads (especially in men) can be an important diagnostic sign to detect high-risk persons with a history of unexplained syncope or a familial incidence of sudden death at a young age.

To help resolve this speculation, long-term electrocardiographic recordings might be used to document ventricular ectopic activity related to transient accentuation of J waves. The significance of these junctional changes could also be probed through epidemiologic studies, such as ones directed at electrocardiograms of athletes. In many countries, 12-lead electrocardiography has been routinely performed for many years. These tracings could be evaluated to determine the natural history of persons with inferolateral junctional changes.

With respect to treatment, placement of implantable cardiac defibrillators is the standard of care for patients who have been resuscitated after life-threatening ventricular arrhythmia. Pharmacologically, only quinidine has been found to prevent arrhythmic recurrences, but few patients have undergone evaluation. In the study by Haïssaguerre et al., a few patients who underwent catheter ablation at the area of ventricular ectopic activity had short-term elimination of arrhythmias. Long-term follow-up of such an intervention is needed, especially in relation to the size of the area showing changes in repolarization, along with analysis of the electrocardiographic characteristics of the ectopic beat.

What one would like to have is a test in asymptomatic persons with junctional changes in the inferolateral leads to identify those in whom manipulation (augmentation) of the J-wave height results in ventricular ectopic activity. Since J-wave amplitude is usually highest at slow heart rates, studies of the effects of carotid sinus massage and the administration of adenosine could be of interest. Until we have better data, we are left with the observation that in some persons with electrocardiographic changes suggesting early repolarization in the inferolateral area, life-threatening ventricular tachyarrhythmias may occur. Since there are many persons who fit such a picture but do not appear to have excess risk, we need further data to unravel how to identify patients who are at high risk for a catastrophic arrhythmia.

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