This also suggests that peripheral components such as muscle function had a share in the improvement seen in the intervention group (5). After all, it is not yet clear whether exercise training can induce an improvement in ventilatory efficiency in HFpEF. A larger, prospective, randomized controlled trial that is now underway (6) will contribute to clarification of this issue.

Frank Edelmann, MD
Götz Gelbrich, PhD
Rolf Wachter, MD
Martin Halle, MD
***Burkert Pieske, MD**

*Department of Cardiology
University of Graz
Auenbrugger Platz 15
A-8036 Graz
Austria
E-mail: burkert.pieske@meduni-graz.at

doi:10.1016/j.jacc.2011.11.062

REFERENCES

1. Edelmann F, Gelbrich G, Düngen HD, et al. Exercise training improves exercise capacity and diastolic function in patients with heart failure with preserved ejection fraction: results of the Ex-DHF (Exercise Training in Diastolic Heart Failure) pilot study. J Am Coll Cardiol 2011;58:1780–91.
2. Guazzi M, Myers J, Arena R. Cardiopulmonary exercise testing in the clinical and prognostic assessment of diastolic heart failure. J Am Coll Cardiol 2005;46:1883–90.
3. Smart N, Haluska B, Jeffriess L, Marwick TH. Exercise training in systolic and diastolic dysfunction: effects on cardiac function, functional capacity, and quality of life. Am Heart J 2007;153:530–6.
4. Kitzman DW, Brubaker PH, Morgan TM, Stewart KP, Little WC. Exercise training in older patients with heart failure and preserved ejection fraction: a randomized, controlled, single-blind trial. Circ Heart Fail 2010;3:659–67.
5. Haykowsky MJ, Brubaker PH, John JM, Stewart KP, Morgan TM, Kitzman DW. Determinants of exercise intolerance in elderly heart failure patients with preserved ejection fraction. J Am Coll Cardiol 2011;58:265–74.
6. Exercise Training in Diastolic Heart Failure. Current Controlled Trials. June 2011. Available at: http://www.controlled-trials.com/ISRCTN86879094/. Accessed March 2, 2012.

Prevalence of J-Point Elevation in Families With Sudden Arrhythmic Death Syndrome

We congratulate Nunn et al. (1) for their interesting report published recently in the *Journal*. They reported that J-point elevation in the inferolateral leads was more prevalent in the first-degree relatives of patients with sudden arrhythmic death syndrome (SADS) than in controls. They suggested that early repolarization was a potentially heritable proarrhythmic marker, risk modifier for lethal arrhythmia, or marker of proarrhythmia in SADS (1). Because J-point elevation is highly prevalent in the healthy population, the report by Nunn et al. (1) encourages the development of better clinical algorithms. Because this study was confined to the relatives of patients with SADS, the significance of J-point elevation found in clinically healthy individuals (electrocardiogram obtained for pre-operative clearance, sports suitability, or job-related check) in the absence of a family history of SADS would probably be minimal. Should it be important to obtain family history in individuals showing J-point elevation? The researchers highlighted that a gene association study or linkage analysis to identify genetic candidates is a logical next step. Although waiting for the availability of more extensive knowledge on the genetic basis of J-point elevation, the risk of missing a potential warning marker would continue to loom in clinical practice.

The group of J-wave syndromes is a spectrum of disorders that involve accentuation of the epicardial action potential notch in different regions of the heart that may predispose patients to develop phase 2 reentry and ventricular tachyarrhythmias. J-point elevation has been divided into 3 subtypes (2,3). An early repolarization pattern in the lateral precordial leads is rarely seen in survivors of ventricular fibrillation (VF) (type 1). On the other hand, J-point changes in inferior or inferolateral leads are usually associated with many cases of idiopathic VF (type 2), and global early repolarization patterns are associated with the highest risk for development of malignant arrhythmias, including VF storms (type 3).

We have observed J-point elevations in young carriers of mutations of various genes (including lamin A/C and plakophilin 2), as well as in healthy relatives of patients with mutation (Fig. 1). After the publication of Nunn et al. (1), this finding cannot be ignored, and it will be important to develop a consensus for the approach to healthy individuals with J-point elevation with and without a family history of SADS.

Alessandra Serio, MD
Nupoor Narula, BS
Antonio Frontera, MD
Fabiana Isabella Gambarin, MD
***Eloisa Arbustini, MD**

*Centre for Heritable Cardiovascular Diseases
IRCCS Foundation Policlinico San Matteo
Piazzale Golgi 19
27100 Pavia
Italy
E-mail: e.arbustini@smatteo.pv.it

doi:10.1016/j.jacc.2011.11.060

REFERENCES

1. Nunn LM, Bhar-Amato J, Lowe MD, et al. Prevalence of J-point elevation in sudden arrhythmic death syndrome families. J Am Coll Cardiol 2011;58:286–90.
2. Yan GX, Antzelevich C. Cellular basis for the electrocardiographic J wave. Circulation 1996;93:372–9.
3. Antzelevich C, Yan GX. J wave syndromes. Heart Rhythm 2010;7:549–58.

JACC Vol. 59, No. 18, 2012
May 1, 2012:1658–61

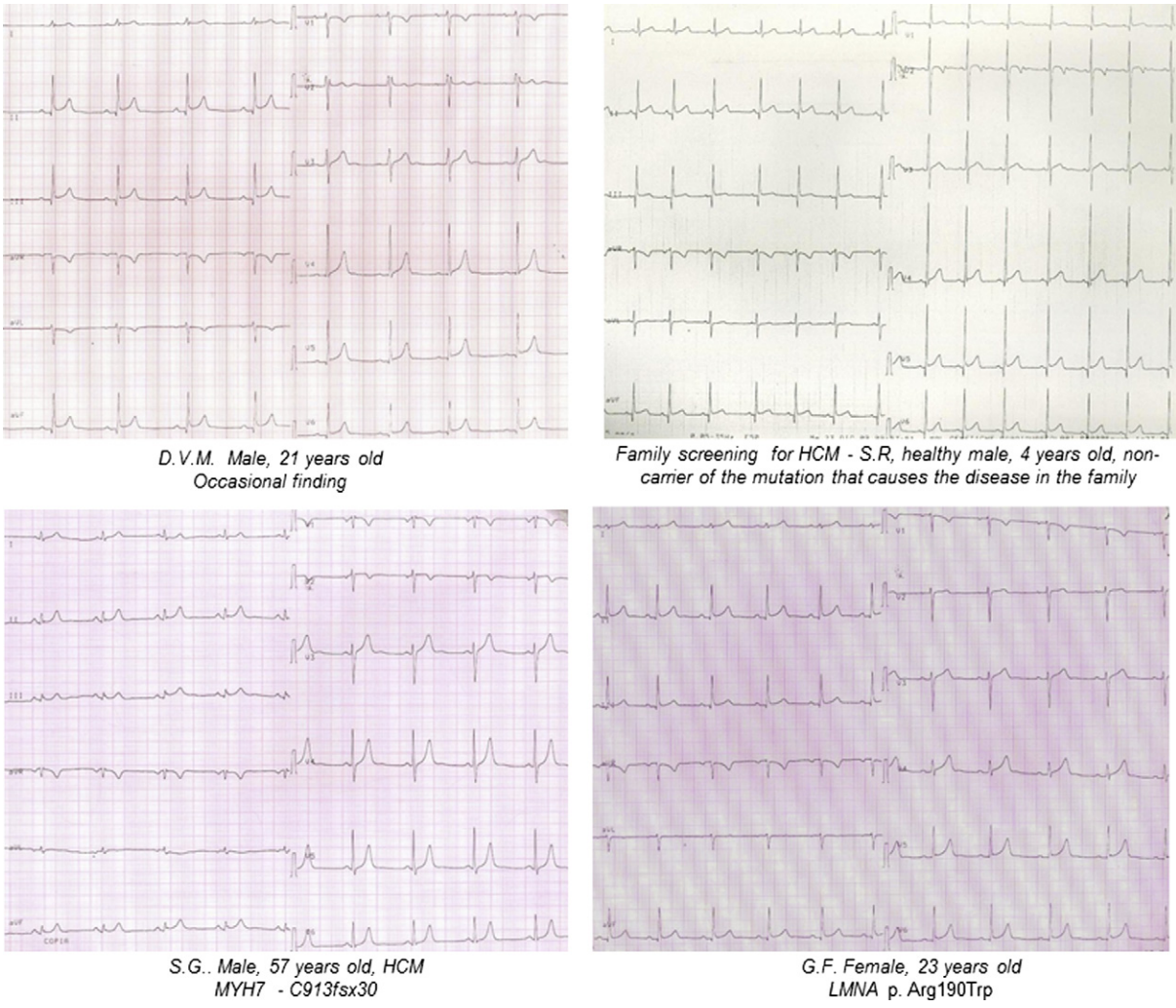

Figure 1 **Different Patterns of J-Point Elevation in Normal individuals and in Patients With HCM and DCM**

DCM = dilated cardiomyopathy; HCM = hypertrophic cardiomyopathy.

Reply

We thank Dr. Serio and colleagues for their interest in our paper (1) and for their letter highlighting the clinical challenge of asymptomatic early repolarization (ER). We noted the examples of J-point elevation in a healthy individual, 2 patients with genetic forms of cardiomyopathy, and a mutation-negative relative. These cases are entirely consistent with our hypothesis that ER may be an independent clinical trait that increases arrhythmic risk in genetically predisposed individuals, but they also illustrate the importance of context when deciding on the clinical significance of ER.

The fundamental clinical distinction is that between physiological J-point elevation, which conveys little or no arrhythmic risk to an individual, and a pathological ER phenomenon that predisposes patients to ventricular fibrillation (VF). In population studies, the presence of a J wave increases the risk of idiopathic VF from 3.4 per 100,000 to 11 per 100,000, making the odds that an individual has a fatal disease 1:10,000, which is too small to influence management (2).

A variety of associated electrocardiogram (ECG) parameters such as the location and amplitude of J-point elevation and a shorter QT interval have been suggested as indicators of an increased risk of VF. However, data from large population cohorts have indicated that the risk is greater in older patients with left ventricular hypertrophy and ischemic heart disease (3,4). These findings need confirmation in larger independent cohorts, and they indicate that other ECG anomalies may be important in determining the sudden death risk of the individual. In addition, further refinement and elucidation of the pattern of ER is still merited.

Based upon our observations and current understanding of the role of ER in VF mechanisms, we suggest that a family history of sudden arrhythmic death syndrome (SADS), epilepsy, or unexplained syncope are new considerations in the interpretation of ER on an ECG because these may indicate either pathological ER or ER in the context of a separate proarrhythmic condition.

In conclusion, at present, ER in the absence of symptoms, structural heart disease, or family history of SADS should not be seen as a major independent risk factor for sudden death. In patients