ventricular volume overload or left atrial volume overload, and to the morphologic characteristics of the mitral valve (i.e., simple mitral valve prolapse vs. flail leaflet or the presence of severe myxomatous infiltration), cannot yet be analyzed. Recently, it has been suggested that mitral valve prolapse is uniformly a good prognosis (3). It is unclear how these various components of the clinical presentation of patients with mitral valve diseases contribute to the outcome, in particular to the risk of sudden death. We agree that further studies are needed on the outcome of various types of mitral valve disease. Such studies will require analysis of large population-based groups of patients with mitral valve disease. At this point, our study allows us to define the notable risk of sudden death incurred under conservative management by patients with flail leaflets, who represent a large group of candidates for surgical correction of mitral regurgitation.

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REFERENCES

U-Wave Alterations: Singular Noninvasive Electrocardiographic Diagnostic Markers

The recent absorbing report by Miwa et al. (1) is, secondarily, a reaffirmation of the considerable utility of the 12-lead electrocardiogram (ECG) and why it continues to be the most frequently used cardiovascular laboratory procedure. It was refreshing to learn that exercise-induced prominent U waves implicated significant left circumflex or right coronary artery disease and that patients with good collateral vessels could be identified by the finding of exercise-induced U-wave alterations. Furthermore, these alterations predicted the development of acute myocardial infarction or hemodynamic instability for low-risk patients upon abrupt closure of a stenotic coronary artery during coronary angioplasty.

There are three practical tenets regarding the at-rest negative U wave on the ECG. There are diverse cardiovascular etiologies, and only with knowledge of the history and physical findings will the full cognitive base residing in the experienced electrocardiographer’s repertoire be mobilized. It is an extremely important wave, as it may be the earliest and only marker of an evolving myocardial infarction (2), and it is an important clue in identifying the congenital long QT syndromes, such as the Jervell and Lange-Nielsen and the Romano-Ward syndromes, which harbor a malignant arrhythmogenic potential (3). Negative U waves may also be recorded in the presence of left ventricular enlargement; left anterior descending coronary artery disease (4); valvular heart disease such as aortic stenosis, mitral regurgitation and aortic insufficiency; and hypertension and variant angina (5). Second, transient U-wave inversion may be seen with both hypertension and variant angina; it can be differentiated on the ECG by initial or terminal negative deflections within the TP segment—the latter as related to myocardial ischemia (5). Finally, Miwa et al. (1) found a lower ejection fraction in patients with severe angina, exercise-induced U-wave alterations and good collateral vessels. Interestingly, in this vein, some 20 years ago, Gerson and McHenry (4) reported that at-rest U-wave inversion was an indicator of stenosis of the left anterior descending coronary artery, and they also found that U-wave negativity was a significant predictor of left ventricular dysfunction, usually segmental anteroapical akinesia or dyskinesia. Miwa et al. (1) and Gerson and McHenry (4) are applauded.

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REFERENCES