women and there was a higher rate of cardiovascular risk factors, comorbidities, heart failure, permanent atrial fibrillation, and severe symptoms, and greater embolic and hemorrhagic risk. These patients also had worse left ventricular ejection fractions and renal function, as well as lower hemoglobin levels. Most of the drug classes were more frequently prescribed in patients with SHD, except for angiotensin receptor blockers (prescribed with a similar frequency) and antiarrhythmics and direct anticoagulants (prescribed less often). Overall, 27.23% of the patients had heart failure, with differential characteristics with respect to the sample, similar to patients with SHD, with a few exceptions (Table 2). Studies in Spain have reported a prevalence of coronary artery disease of between 10% and 20% in anticoagulated patients with NVAF, a similar prevalence to that reported in our study. The CALIFA registry is the only one of these studies to report frequencies of hypertensive heart failure (15.7%) and valve disease (4%) in Spain. These frequencies are similar to those reported in our registry (11.4% and 2%, respectively). It is possible that exclusion of patients with moderate or severe mitral regurgitation could partly explain this low frequency of heart disease. In the case of heart failure, previous studies have reported frequencies between 22% and 24%, which are similar to those observed in this series. A limitation of the present study is that several design features (anticoagulation in the 6 months prior to inclusion, exclusion of hospitalized patients, the willingness of the physicians involved in the registry, etc) could have resulted in a biased sample, and so extrapolation of our results to the overall population with NVAF should be made with caution. Furthermore, classification of heart disease was performed using medical records, which, although a true representation of everyday clinical practice, may have heterogeneous application of diagnostic criteria. Nevertheless, our results, obtained in a large Spanish sample of consecutive patients with NVAF, suggest that almost half have SHD and more than quarter have heart failure. These patients had a different clinical profile to the other patients with NVAF and they received direct anticoagulants less frequently.

Martín Ruiz Ortiz, Inmaculada Roldán, Vicente Bertomeu, Javier Muñiz, Francisco Marín, and Manuel Anguita on behalf of the investigators of the FANTASIA study

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studied. 35.79% were obese, and 79.26% were obese or overweight. Patients with a higher BMI had a higher prevalence of hypertension, diabetes mellitus, dyslipidemia, and obstructive sleep apnea. No significant differences were found between BMI groups regarding treatment with lipid-lowering therapy, beta-blockers, angiotensin-converting enzyme inhibitors, antiplatelets, aldosterone antagonists, amiodarone, and digoxin. During the 8.65 ± 0.34 years of follow-up, 104 deaths (16%) were registered. Specifically, 24 patients (17.80%) with a normal BMI, 46 (16.30%) overweight patients, and 34 (14.60%) obese patients died. No differences were observed between the 3 groups regarding the number of hospital admissions. The response to cardiac resynchronization therapy was also similar between groups. No differences were found in terms of appropriate shocks, inappropriate shocks, or electrical storms. Likewise, the Kaplan-Meier survival curves showed no differences in mortality for obese and overweight patients vs normal weight patients (Figure).

The parameters shown to be predictors of mortality included age, valve disease, heart rate > 70 bpm, anemia (hemoglobin < 13 mg/dL), dyslipidemia, female sex, atrial fibrillation, left ventricular dysfunction (left ventricular ejection fraction < 25%), and renal failure (creatinine > 1.3 mg/dL). No relationship was found between BMI and mortality. On multivariable analysis, there were no differences in mortality between the overweight and obese subgroups (overweight, hazard ratio [HR] = 0.94; 95% confidence interval [95%CI],
Catheter Ablation of Premature Ventricular Contractions From the Left Ventricular Summit

Ablación con catéter de extrasístoles ventriculares del summit ventricular izquierdo

To the Editor,

Approximately 12% of idiopathic left ventricular (LV) arrhythmias (VA) originate from the LV summit: a triangular region of the epicardial LV outflow tract with the apex at the bifurcation between the left anterior descending and left circumflex coronary arteries with its base formed by an arc connecting the first septal perforator branch of the left anterior descending coronary artery with the left circumflex coronary artery. It is transected laterally by the great cardiac vein (GCV) at its junction with the anterior interventricular vein (AIV) into an area accessible to ablation inferiorly and an inaccessible area superiorly. Electrocardiographically, right bundle branch block morphology with inferior rightward axis is typically observed. During recent years, ablation of LV summit VA has received increasing attention in the literature, given its significant frequency and the challenging technical aspects of catheter ablation.1–4

We report the case of a 59-year-old man with a history of ischemic heart disease and normal LV function who presented with palpitations and high density (30%) premature ventricular contractions (PVC) on 24-hour Holter recording. Beta-blockers and amiodarone were ineffective and he was scheduled for an

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Figure. Kaplan-Meyer survival curves according to body mass index.

0.57–1.54; P = .805; obesity, HR = 0.837; 95%CI, 0.49-1.42; P = .507). Similarly, there were no differences in the number of admissions for cardiovascular causes (obesity, HR = 0.986; 95%CI, 0.547-1.468; P = .663; overweight, HR = 0.981; 95%CI, 0.611-1.575; P = .936).

The conclusion drawn from this study, based on BMI analysis, is that obesity and overweight show no prognostic differences compared with normal weight for cardiovascular mortality, cardiovascular hospitalization, and appropriate and inappropriate therapies in this population of patients with HF and an ICD implant for primary prevention of SD.

However, the interpretation of these study results should take into account the limitations of the study. First, the conclusions are drawn from BMI analysis, which does not differentiate body fat from lean body mass. Second, we did not analyze distribution of body weight (peripheral vs abdominal) or other measurements of adiposity such as body fat percentage. In addition, no information was available on the proinflammatory and nutritional status of the study population. Furthermore, the available information on BMI was taken from the time of implantation only; therefore, possible changes in this parameter at follow-up were not considered. Lastly, the retrospective design of the study increased the risk of bias.