Limitations in the Clinical Assessment of Obesity: Comments on the American Heart Association’s 2006 Statement

To the Editor:

The American Heart Association (AHA) update on obesity has recently been published.1 The purpose of this letter is to comment briefly on the limitations of the clinical assessment of obesity.

The link between obesity and cardiovascular risk (CVR) has traditionally been controversial. Although the Framingham study already observed in 1967 that higher body weight raised an individual’s probability of cardiovascular disease (CVD),2 it was only in 1998 that the AHA (American Heart Association class) first recognized obesity as a major independent cardiovascular risk factor.3 The explanation to this problem lies precisely in the limitations of obesity assessment, which should be expressed as a percentage of body fat,4,5 but is difficult to quantify clinically.6 Numerous anthropometric variables have been used for this purpose, but body mass index (BMI) is the most highly extended to assess body weight and, along with abdominal circumference, is the method recommended by the AHA.1

The key limitations in the clinical assessment of obesity include the following:

1. Available indexes do not identify the percentage of body fat (ie, they do not discriminate between muscle, fat, and bone).
2. The fat-to-muscle ratio varies with age, gender, ethnic group, and race.5,6,7
3. The BMI varies with body proportions and may tend to underestimate the prevalence of obesity in taller subjects and to overestimate it in shorter subjects, although this should be confirmed in larger populations.8 Moreover, there are multiple confounding factors that can mask the actual relationship between obesity and CVR.
5. Comorbidity of the obese patient.1
6. Underweight subjects (BMI <18.5) have an elevated prevalence of smoking, chronic diseases, and risk of death from cancer.9
7. CVR varies with height10 and is lower in tall subjects.11
8. Other: physical exercise,12 diet,13 etc.

In summary, the main clinical indexes for defining obesity (BMI, abdominal circumference, and even waist/hip circumference14) have limitations, although there are practical alternatives such as a combined assessment of weight1 and degree of physical activity.13,15 This would make it possible to identify sedentary obese individuals, a subgroup with greater CVR12 and, theoretically, a higher percentage of body fat.

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REFERENCES
To the Editor:

Brugada syndrome, an entity described in 1992, is characterized by episodes of syncope or unexpected sudden death in patients with a structurally normal heart, and a characteristic electrocardiogram consisting of a right bundle-branch block (RBBB) pattern and ST segment elevation in the unipolar precordial V1 through V3 leads. The electrocardiographic pattern may be present, intermittent, or occult (only demonstrable with a test done with flecainide, procainamide, or ajmaline). The arrhythmic event can occur at rest, triggered by stress, or with no apparent relationship, with variations in the autonomic nervous system. There are 3 types: type 1, which presents coved ST-segment elevation ≥2 mm, followed by negative T-wave; type 2, with saddleback ST segment elevation and J point ≥2 mm, followed by positive T-wave; and type 3, with ST segment elevation and J point <1 mm and variable morphology (coved or saddleback).

We describe a patient with the characteristic ECG features of Brugada syndrome who was found to have sustained monomorphic ventricular tachycardia (SMVT) during exercise testing, an observation for which we found no references in the scientific literature.

Exercise testing is a procedure used for the diagnostic and prognostic assessment of patients with ischemic heart disease that is also used in other subjects, both healthy and ill, with nonischemic heart disease.

A 38-year-old man with no personal or family history of interest was referred for exercise testing due to episodes of chest pain. He was not receiving any therapy.

The baseline electrocardiogram (ECG) was performed without medication and showed the characteristic image of type 1 Brugada syndrome, with RBBB and ST segment elevation in V1, V2, and V3 (Figure 1).

The exercise test was done using the Bruce protocol, with 10:08 minutes of exercise. After 1 minute of recovery, the patient presented SMVT with RBBB morphology at a rate of 180 bpm that lasted 40 s but showed no hemodynamic repercussions (Figure 2). He did not present chest pain at any time during the test, which was clinically and electrically negative for ischemia.

The patient was admitted to our hospital, where transthoracic echocardiography showed normal systolic function, with no regional contractility abnormalities, or other pathological findings of interest. Left catheterization for coronary angiography and ventriculography yielded normal results.

Electrophysiological study was later performed, in which 3 extrastimuli were applied to the apex of the right ventricle, but only nonsustained ventricular tachycardia was achieved. An implantable cardioverter defibrillator was indicated, based on the possibility of malignant arrhythmia, which had been documented with the stress test.

Figure 1. Baseline electrocardiogram shows the classic pattern of Brugada syndrome.