Electrocardiographic Signs of Left Ventricular Hypertrophy in Obese Patients: What Criteria Should be Used?

Giuseppe Germano

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Abstract Overweight and obesity are estimated at high prevalence and progression in adults; they are major contributors to chronic diseases and a major public health challenge. An obese habitus changes body-surface electrocardiograms (ECGs). Obesity is responsible for geometric changes to the heart and torso, as well as for deleterious electrophysiological changes of the heart. Common ECG changes, reduced voltages in the precordial leads, and axis deviation have made the search for left ventricular hypertrophy (LVH) even more problematic. Identification of LVH by ECG is difficult and time consuming but ECG is fundamental to reveal abnormalities of clinical relevance associated with obesity. The QTc dispersion assessment and the comparison with magnetic resonance imaging are the frontiers to clarify the connection between ECG LVH signs and overweight and obesity.

1 Introduction

Hypertension guidelines [1, 2] recommend, as the first step of risk stratification, the 12-lead electrocardiographic identification of left ventricular hypertrophy (LVH), which represents a marker of cardiac organ damage and has been related to increased risk of cardiovascular morbidity and mortality at the population level as well as in different clinical conditions. Mechanical and hormonal stimuli may be involved in the activation of myocyte growth, increasing the risk of arrhythmias, impaired coronary flow reserve and

decreased response of coronary flow to vasodilator management. The consequences of electrocardiographic electrogenesis can be summarized in: (1) a rotation of the heart around its three axes, anterior–posterior, longitudinal and transverse; (2) an increase in the voltage of the QRS prevalent in the leads that capture the potential of the enlarged ventricle; (3) increased QRS duration; its width increases from 0.08'' to 0.10''; (4) a delay in the onset of the intrinsicoid deflection in the precordial leads facing the enlarged ventricle: delay exceeding 0.03'' in the right precordial leads and 0.05'' in the left precordial leads [3].

Many features (up to 30) of LVH have been proposed (Table 1) [4], but the most widely used are based on a composition of voltage and non-voltage criteria including left ventricular strain, left atrial enlargement and the duration of intrinsicoid deflection (Table 2) [5].

The sensitivity of ECG criteria, when compared with the left ventricular mass by echocardiography is limited. David Pewsner's review of 21 studies in different healthcare settings found that sensitivity ranged from 10.5 % for the Grubner index to 21 % for the Sokolow–Lyon index and that specificity ranged from 89 % for the Sokolow–Lyon index to 99 % for the Romhilt–Estes score [13]. When the left ventricular mass indexed to height was used in middle-aged participants, compared with the older ones, the Cornell voltage had a higher sensitivity [14]. The Ikaria population study showed that when LVH was indexed for body surface area or for height, Framingham criteria had the highest, albeit not significant, sensitivity among the elderly, although the Cornell criteria had the highest sensitivity for middle-aged participants [15]. In any case, regression of ECG signs of LVH occurred during long-term antihypertensive regimen improving cardiovascular prognosis [16]. This difficulty in establishing a common criterion led to a prospective study that found a simple measurement of the

G. Germano (✉)
Dipartimento di Scienze Cardiovascolari, Respiratorie,
Nefrologiche, Anestesiologiche e Geriatriche, Sapienza
University of Rome, Rome, Italy
e-mail: ncgerman@tin.it

Table 1 Electrocardiographic features and thresholds of LVH

	Amplitude
<i>Limb lead voltage</i>	
(RI – SI) + (SIII – RIII)	>16 mm
RI + SIII	>25 mm
RI	>15 mm
RaVL	>11 mm
RaVF	>20 mm
Q or SaVR	>19 mm
R + S in any limb lead	>19 mm
<i>Precordial lead voltage</i>	
SV ₁	>23 mm
SV ₂	>25 mm
SV ₁ + RV ₅	>35 mm
SV ₂ + RV _{5,6}	>45 mm
SV _{1,2} + RV _{5,6}	>35 mm
SV _{1,2} + RV ₆	>40 mm
R + S any precordial lead	>35 mm
RV ₅ :RV ₆	>1.0 mm
R ₁ any precordial lead	>26 mm
SV ₂ + RV _{4,5}	>45 mm
RV ₅	>33 mm
RV ₆	>25 mm
<i>Combination of limb and precordial voltage</i>	
RSaVF + V ₂ + V ₆ (>30 years)	>59 mm
RSaVF + V ₂ + V ₆ (>30 years)	>93 mm
SV ₃ + RaVL (men)	>28 mm
SV ₃ + RaVL (women)	>20 mm
Total 12-lead voltage	>175 mm
<i>Combination of voltage and nonvoltage</i>	
Voltage-STT-LAA-axis-QRS duration	Point score
(RaVL + SV ₃) × QRS duration	>2,436 mm/s
Total 12-lead voltage × QRS duration	>1,742 mm/s
<i>Criteria for use with left anterior fascicular block</i>	
SV ₁ + RV ₅ + SV ₅	>25
SV _{1,2} + RV ₆ + SV ₆	>25
SIII + max R/S any lead (men)	>30
SIII + max R/S any lead (women)	>28
<i>Criteria for use with right bundle-branch block</i>	
Max R/S precordial lead (with LAD)	>29 mm
SV ₁	>2 mm
RV _{5,6}	>15 mm
S III + max R/S precordial (with LAD)	>40 mm
RI	

According to ‘AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram’. Amplitudes are given in millimeters, where 1 mm = 0.1 mm = 0.1 mV. Adapted from Hancock et al. [4]

LAD left axis deviation

R wave in aVL in the diagnosis of LVH and the prediction of cardiovascular risk [17].

Obesity, as defined by science advisory and expert committee recommendations (Table 3) [18, 19], has an important role in many of the hemodynamic and non-hemodynamic mechanisms involved in the hypertrophic adaptation of the heart and circulation, such as systolic and diastolic dysfunction, abnormal left ventricular geometry, high blood pressure, autonomic nervous system imbalance, endothelial injury, insulin resistance and increased systemic inflammation. Overweight and obesity are currently viewed as conditions characterized by growing prevalence and progression in adults and are major contributors to chronic diseases that represent a serious health challenge. An obese habitus has an impact on body-surface electrocardiograms compared with normal values.

2 Left Ventricular Hypertrophy in Obese Patients

The body-surface electrocardiogram changes include leftward shift of the P wave, QRS and T wave axes, enhancement of the P-terminal force in lead V₁ and prolongation of P wave duration at the interpeak notch in lead II, possible prolongation of the PR interval, possible prolongation of the QRS duration, low QRS voltage, which is of great importance here, and left ventricular hypertrophy. In addition, it is common to find T wave flattening in the inferior and lateral leads, prolongation of the corrected QT interval, increase in QT interval dispersion, an abnormal signal-averaged ECG and abnormal heart rate variability [20]. Obesity is also responsible for the geometric changes of the heart and torso. Obesity-related geometric alterations explain first of all left ventricular hypertrophy, but abdominal enlargement and heart displacement must be taken into account [21] even more than abundant breast tissue, especially in women who have practically negligible effect [22]. Therefore, low voltage on electrocardiogram is an usual condition in obesity when no wave positive or negative in a peripheral bipolar lead exceeds 5 mm or the sum of all positive and negative deflections in the three standard limb leads does not exceed 15 mm and in the precordial leads the maximum height of the QRS (R + S) reaches 9 mm in V₂ and 7 mm in V₆, but it is not a significant feature [23] and too small to be detected in clinical vectocardiography [24]. In addition, there is a relatively high frequency of left axis deviation (horizontalization) associated with the clockwise rotation around the longitudinal axis, found when the diaphragm is raised in the obese, as in brachytype with hypotonic abdominal walls. Typical of

Table 2 The most widely used electrocardiographic criteria of LVH

1. Cornell voltage criteria: $RaVL + SV_3 \geq 20$ mm for women and ≥ 28 mm for men [6]
2. Cornell duration criteria: $(RaVL + SV_3) \times \text{QRS duration}$; for women, add 8 mm, $\geq 2,440$ mm milliseconds [7]
3. Sokolow–Lyon voltage criteria: $SV_1 + RV_5$ or $V_6 \geq 35$ mm [8]
4. Romhilt–Estes score: higher R or S amplitude ≥ 30 mm in the horizontal plane or ≥ 20 mm in the frontal plane or strain pattern in lead V_5 or V_6 (if using digitalis, this means only one point) or left atrium increase according to Morris index (3 points); electric axis of AQRS (spatial axis of QRS on the frontal plane) greater than -30° (2 points); QRS duration ≥ 90 ms in V_5 or V_6 , or ventricular activation time ≥ 50 ms in V_5 or V_6 (one point). According to this score, LVH is diagnosed when the sum is ≥ 5 points [9]
5. Perugia score: LVH is diagnosed by the presence of one or more of the following findings: Cornell criteria, considering the limit ≥ 20 mm for women and ≥ 24 mm for men, Romhilt–Estes score, and strain pattern [10]
6. R wave of a VL ≥ 11 mm [11]
7. Presence of strain pattern: defined as a convex ST-segment depression with asymmetric T-wave inversion opposed to QRS complex in leads V_5 or V_6 [12]

Table 3 Classification of body weight according to BMI in adults and children*Adults*

Underweight BMI <18.5 ; normal BMI 18.5–24.9; overweight 25–29.9

Obese BMI ≥ 30 ; class 1 BMI, 30–34.9; class 2 BMI, 35.5–39.9; class 3 BMI ≥ 40 ; class 4 BMI ≥ 50 ; class 5 BMI ≥ 60

Children (2–18 years)

Overweight BMI at 85 to 94th percentile

Obese BMI at 95th percentile or BMI ≥ 30 , whichever is lower

Severe obesity BMI at 99th percentile

BMI $\cong 30$ –32 for youths 10–12 years of age; BMI 34 for youths 14–16 years of age

horizontal hearts with clockwise rotation is the appearance of Q waves of various depths in D_3 associated generally with S in D_1 . In this association, horizontalization and clockwise rotation, the left ventricle is placed further back and down and the septum becomes more horizontal [3]. One study has demonstrated that Cornell voltage criterion increases with body weight [25]; another study finds however that although $RaVL$ may be positively associated with BMI, SV_3 is not associated, rendering the sum of both unrelated to BMI [26]. Studies have also demonstrated a reduced sensitivity of Sokolow–Lyon criteria in detecting LVH and a slight relation was noted between Sokolow–Lyon criterion and LVMI in the Framingham study with a higher prevalence of being overweight or obesity [12]. The same conclusions are reported by studies that analyzed the influence of voltages of limb lead recordings [12, 27]. Conversely, Cornell voltage, voltage-duration criteria and the multifactorial Perugia criterion did not show differences in diagnostic performance between obese and nonobese individuals in a Brazilian population, while sensitivity statistically significantly decreased in obese patients with regard to Sokolow–Lyon voltage, Romhilt–Estes criterion and strain pattern [5].

3 Conclusions

The identification of LVH by electrocardiogram in obese patients is difficult and time consuming, but is fundamental in revealing abnormalities of clinical relevance associated with obesity such as alterations of P wave, QRS and T wave, prolongation of QRS and QT duration, and dispersion and arrhythmias. Only in hypertensive Chinese patients a significant correlation of heart mass with the QTc interval was found [28]. The increase in QT dispersion (a measure of inhomogeneous repolarization and an indicator of arrhythmogenicity, calculated as the difference between maximum and minimum QT interval), is a sign of pathological LVH not present however when we compare with the same myocardial mass in athletes [29]. LVH is a key determinant of QTc dispersion in normotensive obese patients. Regression of LVH associated with weight loss after bariatric surgery decreases QTc and QTc dispersion [30]. A recent study, using magnetic resonance imaging, showed no relation between voltage and anatomic criteria, supporting the idea that the electrical changes in a hypertrophied heart are to be considered differently from anatomic changes [31] and new studies shall clarify this relationship.

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