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Detection of left ventricular hypertrophy through ECG: diagnostic performance and comparison of ten ECG scores in different hypertrophic conditions

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ABSTRACT

Background: Different electrocardiographic (ECG) criteria were proposed to improve ECG-based detection of left ventricular hypertrophy (LVH), but external validation data are limited and their diagnostic performances in different conditions leading to LVH are not extensively assessed.
Aim: to assess the robustness and limitations of ECG scores across distinct LVH phenotypes.
Methods: The LVH-MORE registry included 408 patients across four cohorts: HTN ($n = 103$), HCM ($n = 104$), sAS ($n = 101$), and controls ($n = 100$). LVH was defined by echocardiographic left ventricular mass index (LVMi) >115 g/m2 in male subjects and >95 g/m2 in female subjects. Ten ECG criteria were calculated, including Peguero–Lo Presti, Sokolow–Lyon, Cornell, Perugia, Romhilt–Estes, and others. Diagnostic performance was assessed using ROC curves (AUC), sensitivity (Sn), specificity (Sp).
Results: In the HTN cohort, Peguero–Lo Presti achieved the highest AUC for LVH detection (0.752 [95% CI: 0.616–0.888]), with Sn = 31% and Sp = 94%. Other scores showed similar or lower AUCs, with Perugia score showing higher sensitivity (54%). The highest specificity were reached by Sokolow-Lyon and Gubner-Ungerleider (100%).
In the HCM cohort, all ECG scores showed modest diagnostic performance (AUC 0.529–0.628). The Perugia and Romhilt–Estes scores achieved the highest sensitivities (94% and 71%, respectively), whereas Sokolow–Lyon showed the highest specificity (90%).
In the sAS cohort, AUCs ranged from 0.519 to 0.671. Sensitivity was higher for Perugia (66%) and Romhilt–Estes ≥4 (57%). Highest specificity was shown by Gubner-Ungerleider (95%).
Conclusions: No single ECG criterion provided consistent diagnostic accuracy across different causes of LVH. Voltage-only ECG scores performed better in HTN patients while multi-parametric scores showed relatively better performance in HCM and sAS.

Introduction

Several electrocardiographic (ECG) scores have been proposed and introduced in clinical practice in the past years for the diagnosis of left ventricular hypertrophy (LVH) [1–8].

The latest one, known as “Peguero-Lo Presti”, was validated in 2017 in patients with arterial hypertension and showed a statistically significant superior predictive performance as compared with other clinically validated ECG score [9].

However, there is a lack of extensive comparison between the scores validated so far with previous studies focusing on a few of them at once and in specific populations [10–15].

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Furthermore, these scores were originally validated in hypertensive patients while LVH may be present in different types of heart disease; in fact, it can occur both in primary conditions (such as hypertrophic cardiomyopathy [HCM]) and as a response to other forms of increased afterload, for example, in the presence of severe aortic stenosis (sAS) [16].

In sAS, LVH represents a maladaptive remodeling independently associated with systolic dysfunction and greater risk of heart failure [17]. Furthermore, LVH in HCM has important clinical and prognostic implications, being associated with adverse outcomes [18].

HCM and sAS are often characterized by ST-T abnormalities such as negative T waves and ST depression, rarely by increased voltages alone; [19–27] to date, there is a lack of specifically validated LVH ECG scores in such diseases and a comprehensive external validation of existing scores in HCM and sAS cohorts is missing.

In the present study, we aim to provide a direct comparison of ten different ECG scores for the diagnosis of LVH and to test their diagnostic performance, robustness and limitations in different hypertrophic conditions such as HCM and sAS.

Methods

Study design

The "Diagnosis of Left Ventricular Hypertrophy in Patients with Different Heart Conditions Using Electrocardiographic Criteria" (LVH-MORE) registry was a retrospective, single-center observational study conducted at our tertiary cardiology center.

The registry was designed to collect electrocardiographic and echocardiographic data from patients with various cardiac conditions associated with LVH, as well as from a control population without structural heart disease.

The study was approved by the local ethics committee (protocol ID: 0022270/25) and conducted in accordance with the principles of the Declaration of Helsinki.

Study population

The LVH-MORE registry was designed to include four cohorts of patients, each representing a different clinical condition associated with LVH, to test the study hypotheses. The cohort consisted in:

1. HTN cohort: we retrospectively included the first 120 consecutive outpatients evaluated at our outpatient clinic between June 2025 and August 2025, who had both a standard 12-lead ECG and a transthoracic echocardiogram performed within a 3-month interval and with a diagnosis of HTN. HTN was defined as an average systolic or diastolic home blood pressure (BP), respectively $\geq$135 or $\geq$85 mmHg, or average systolic or diastolic office BP respectively $\geq$140 or $\geq$90 mmHg as per latest ESC guidelines[28]. Patients with concomitant hypertrophic cardiomyopathy or severe aortic stenosis were excluded.
2. HCM cohort: this cohort included 120 consecutive patients followed at our dedicated cardiomyopathy outpatient clinic between January 2015 and August 2025. HCM was defined as a left ventricular wall thickness $\geq$15 mm in any myocardial segment or an apical-to-basal wall thickness ratio $\geq$1.5 in case of apical variant in the absence of other causes of LVH. Patients with previous surgical myectomy, alcohol septal ablation or under active treatment with myosin inhibitor mavacamten at the time of ECG/echo acquisition were excluded.
3. sAS cohort: this cohort included 120 consecutive inpatients referred to our cardiology department for elective transcatheter aortic valve implantation/replacement (TAVI/TAVR) between January 2017 and August 2025. All patients had sAS according to current ESC guidelines[29]. Patients with acute heart failure, or whose ECG/echocardiogram were performed after TAVI/TAVR, were excluded.
4. Control cohort: A control group of 120 consecutive outpatients was selected from our echocardiography laboratory among individuals referred for cardiovascular screening (e.g., pre-participation or preventive evaluation). Subjects with HTN, HCM, severe aortic stenosis, or any acute or structural heart disease were excluded to create a "normal" non-LVH reference group.

All patients with complete left or right bundle branch block or ventricular paced rhythm were excluded from the study. We also excluded all confirmed cases of cardiac amyloidosis given its association with low voltages and discrepancy between LVMi by echocardiogram and electrocardiographic features [30.31].

Echocardiographic analysis

Transthoracic echocardiography was the method of choice to estimate left ventricular mass. All echocardiograms were acquired using a Philips Epiq 7 and a GE Vivid E9. Left ventricular mass (LVM) was estimated using the Devereux formula with an optimal left parasternal long axis view in a left lateral decubitus position (Fig. 1). LVH was defined as a left ventricular mass indexed (LVMi) to body surface area (BSA) >115 g/m2 in male subjects and >95 g/m2 in female subjects. All cases were reviewed by an operator (F.P.) blinded to the results of ECG analysis.

Electrocardiographic analysis

A standard 12-lead electrocardiogram (ECG) was obtained for each patient at a paper speed of 25 mm/s and 1 mV/cm calibration. All the ECG were acquired using a Cardioline 100+ (CE; Cardioline S.P.A.). Whenever possible, the ECG recorded on the same day as the echocardiogram was selected; if unavailable, the closest available tracing within the same hospitalization or within 3 months was used. All ECGs were independently reviewed by an experienced cardiologist (K.S.).

Individual leads were analyzed by measuring Q, R and S waves in all precordial and limb leads, using the PR segment as the baseline; in the presence of multiple complexes within the same lead, the highest voltage was selected for analysis (Fig. 1). Left ventricular strain was defined as ST-segment depression and asymmetric T wave inversion in lateral leads.

Ten different ECG criteria LVH were systematically calculated for each patient: Lewis voltage, Gubner–Ungerleider, Sokolow–Lyon, R aVL, Romhilt–Estes, Cornell voltage, Perugia score, Framingham criterion, Cornell product, and Peguero–Lo Presti.

Detailed definitions and threshold values for LVH according to each criterion are provided in the Supplementary Materials.

Statistical analysis

Continuous variables were presented as mean value ± standard deviation and compared using the Student's *t*-test or Mann-Whitney U test, as appropriate. Categorical variables were presented as numbers and percentages and compared using the Chi-square test. The diagnostic performance of each ECG score for LVH was assessed using receiving operator curves (ROC) analysis, and the area under the curve (AUC) was calculated with the corresponding 95 % confidence interval (CI). Comparisons between the two AUCs were estimated using the method proposed by DeLong and DeLong[32].

We also computed sensibility (Sn) and specificity (Sp) for the prespecified cut-offs of the various ECG scores (as reported in Supplementary material) with their respective 95 % CI. McNemar test was used to determine a possible lack of agreement comparing the electrocardiographic criteria against the LVMi calculated by echocardiographic analysis: a p value <0.05 was considered to define lack of agreement.

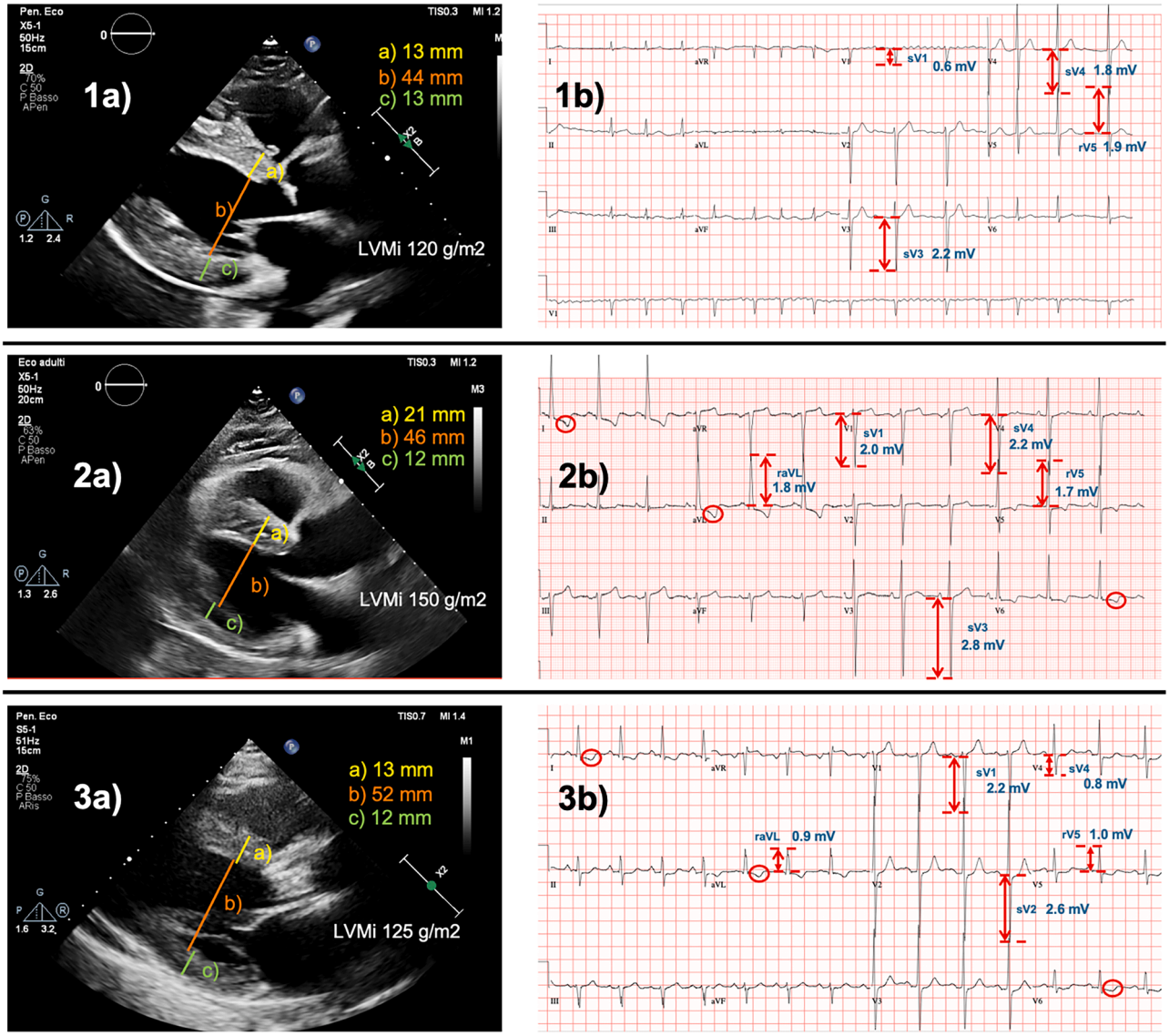

Fig. 1. Echocardiographic and ECG analysis to assess LVH in the three cohorts examined. 1a and 1b) hypertensive patient. 2a and 2b) hypertrophic cardiomyopathy patient. 3a and 3b) severe aortic stenosis patient.
LVMi: left ventricular mass index; mm: millimeters; mV: millivolt.

We performed sensitivity analyses to evaluate the robustness of the diagnostic performance of the ECG scores. First, we calculated AUC in subgroups that might influence ECG diagnostic power: (i) for overweight and obese individuals, we performed the analysis for patients with a BMI $\geq$ 25 kg/m2. Considering the potential underestimation of LVMi by BSA indexation in this specific subgroup, LVM was also normalized to height to an allometric power of 2.7 as suggested by previous studies [33,34]). Accounting for ECG voltages attenuation in overweight/obese patients, a last sensitivity analysis was conducted using a BMI-corrected Perugia score as proposed by Angeli et al. [34] maintaining LVM/height$^{2.7}$ as the reference standard; (ii) patients aged $\geq$65 years.

Second, within the HCM cohort, we performed separate analyses for AUC, Sn and Sp in the subgroup without apical HCM.

All analyses were performed using R software version 4.2.2 (R Foundation for Statistical Computing, Vienna, Austria) on macOS.

Results

Baseline characteristics

We included in the LVH-MORE registry 408 patients. Considering the 4 cohorts of HTN, HCM, sAS and control patients, respectively 17, 16, 19 and 20 patients for each group were excluded from the final analysis due to poor quality echocardiography. The baseline characteristics, different echocardiographic and ECG parameters are reported respectively in Table 1, Table S1 and S2.

HTN cohort

HTN patients showed higher BMI, higher prevalence of diabetes mellitus (DM) and coronary artery disease (Table 1); furthermore, higher antihypertensive therapy rates than other cohorts were observed (Table 1). Mean LVMi by echocardiogram was 79 $\pm$ 21 g/m2. 13 patients were found to have LVH assessed by echocardiographic parameters (Table 1). Fig. 2 reports the results of AUC curves for the ECG analysis results in the HTN cohort, while the results of the DeLong test

Table 1
Demographic characteristics of the population stratified by different LVH subgroups.

	Control (n = 100)	HTN (n = 103)	HCM (n = 104)	sAS (n = 101)	pvalue
Age	58 ± 15	69 ± 10	62 ± 13	79 ± 8	<0.001
Female	55 (55)	39 (38)	40 (39)	51 (51)	0.027
Body Mass Index	24 ± 4	28 ± 5	27 ± 5	26 ± 5	<0.001
Body Surface Area	1.79 ± 0.22	1.92 ± 0.25	1.88 ± 0.22	1.80 ± 0.20	<0.001
Left ventricular mass index, g/m2	64 ± 15	79 ± 21	123 ± 37	112 ± 35	<0.001
Left ventricular hypertrophy	0	13 (13)	63 (61)	61 (60)	<0.001
Diabetes Mellitus	6 (6)	31 (30)	15 (15)	26 (26)	<0.001
Hypertension	0	103 (100)	58 (56)	84 (86)	<0.001
HR, beats/min	72 ± 14	73 ± 16	70 ± 14	72 ± 13	0.585
AF	10 (10)	20 (19)	24 (23)	38 (39)	<0.001
CAD	1 (1)	18 (18)	4 (4)	41 (42)	<0.001
MI	1 (1)	11 (11)	5 (5)	17 (18)	<0.001
PCI	1 (1)	16 (16)	6 (6)	31 (32)	<0.001
CABG	0	4 (4)	0	8 (8)	<0.001
HF					<0.001
HFrEF	1 (1)	3 (3)	5 (5)	16 (16)	
HFmEF	0	3 (3)	3 (3)	16 (16)	
HFpEF	0	1 (1)	3 (3)	3 (3)	
COPD	2 (2)	6 (6)	5 (5)	10 (10)	0.088
PAD	0	2 (2)	2 (2)	9 (9)	<0.001
Stroke					0.507
Ischaemic	2 (2)	6 (6)	5 (5)	7 (7)	
Haemorrhagic	0	0	1 (1)	1 (1)	
ACE-I	2 (2)	31 (31)	17 (16)	18 (19)	<0.001
ARB	0	32 (32)	21 (20)	17 (18)	<0.001
Betablockers					<0.001
Bisoprolol	7 (7)	23 (23)	36 (35)	28 (30)	
Metoprolol	3 (3)	8 (8)	28 (27)	16 (17)	
Other	6 (6)	18 (18)	11 (11)	19 (20)	
MRA	8 (8)	13 (13)	13 (13)	32 (34)	<0.001
Digoxin	1 (1)	1 (1)	3 3)	7 (8)	0.028
DHP CCB	0	37 (37)	24 (23)	25 (27)	<0.001
Non-DHP CCB(%)	0	2 (2)	5 (5)	3 (3)	0.438

Values are mean standard deviation or n (%). ACE-I, angiotensin-converting enzyme inhibitors; AF, Atrial fibrillation; ARB, angiotensin receptor blockers; CABG, Coronary Artery Bypass Graft; CAD, Coronary artery disease; COPD, Chronic obstructive pulmonary disease; DHP CCB, dihydropyridine calcium channel blockers; HF, Heart Failure; HR, Heart rate; MI, Myocardial Infarction; MRA, mineralocorticoid receptor antagonist; Non-DHP CCB, non-dihydropyridine calcium channel blockers PAD, peripheral arteria disease; PCI, Percutaneous Coronary Intervention.

are reported in Table S3. When analyzed as a continuous variable, the Peguero-Lo Presti had the nominally higher predictive performance for LVH (AUC: 0.752; 95 % CI 0.616–0.888), followed by Perugia Score (AUC: 0.736; 95 % CI 0.593–0.879, P for difference = 0.818) and Romhilt-Estes (AUC: 0.659; 95 % CI 0.475–0.842, P for difference = 0.358) (Fig. 2).

In the HTN cohort, the Peguero-Lo Presti showed a Sn of 31 % (95 % CI 9–61) and a Sp of 94 % (95 % CI 88–98 %). The nominally higher Sn was reached by the Perugia Score (54 %; 95 % CI: 25–81 %) followed by Romhilt-Estes (38 %; 95 % CI 14–68 % if score $\geq$ 4 was considered and 31 %; 95 % CI 9–61 % if score $\geq$ 5 was considered). The overall best specificity was reached by Sokolow-Lyon (100 %; 95 % CI 96–100 %) and Gubner-Ungerleider (100 %; 95 % CI 96–100 %). The results of Sn and Sp for the other scores are reported in Table 2.

The Peguero Lo-Presti score showed agreement with the echocardiographic gold standard considered in the present analysis ($P = 0.423$). Similar results were found for the Romhilt-Estes, Perugia, R-aVL, Cornell and Lewis score (Table 2).

Sensitivity analyses in overweight/obese and older patients were broadly consistent with the primary findings (Fig. 2, Table S5, Figure S1). In participants aged >65 years, the Sokolow–Lyon score showed improved diagnostic performance, with the AUC increasing from 0.656 (95 % CI 0.460–0.852) to 0.763 (95 % CI 0.576–0.949) (Fig. 2). When LVM was indexed to height$^{2.7}$, Perugia score showed a lowered diagnostic performance, with AUC decreasing from 0.736 (95 % CI 0.539–0.879) to 0.660 (95 % CI 0.478–0.842) (Figure S1); conversely, diagnostic performance for both Peguero-Lo Presti and Cornell Product increased (Figure S1).

HCM, sAS cohorts and control cohort

Patients with HCM were younger compared with those in the HTN and sAS cohorts. Conversely, patients with sAS exhibited a higher prevalence of common cardiovascular comorbidities, including coronary artery disease, heart failure, and atrial fibrillation (Table 1).

Both the HCM and sAS groups showed significantly higher LVMi values compared with HTN patients (123 ± 37 g/m^2 and 112 ± 35 g/m^2, respectively; $P < 0.001$, Table 1), with HCM patients presenting the greatest mean interventricular septal thickness (16.2 ± 4.5 mm; $P < 0.001$, Table S1). LVH was present in 63 (61 %) of HCM patients and 61 (60 %) of sAS patients, both significantly higher compared with the HTN cohort ($P < 0.001$, Table 1).

The control cohort showed a younger age and fewer cardiovascular comorbidities compared to HTN (Table 1), with the lowest LVMi and interventricular septal thickness among all cohorts (respectively 64 ± 15 g/m^2 and 7.9 ± 1.2 mm; $P < 0.001$, Table 1 and S1).

Since no patients in the control cohort was diagnosed with LVH at echocardiographic assessment we did not perform the analysis of diagnostic performance of the scores.

Diagnostic performance of the scores in the HCM cohort

ECG analysis showed that the HCM cohort reached the highest values for all the scores examined (Table S2). Fig. 2 shows the results of the ROC analysis for the predictive performance of the scores used as continuous variables. The nominally higher predictive performance for the diagnosis of LVH in the HCM cohort was achieved by Framingham Criterion (AUC: 0.628; 95 % CI 0.487–0.714) followed by Cornell, Cornell Product (AUC: 0.610; 95 % CI 0.535–0.720 and AUC: 0.610; 95 % CI 0.496–0.724 respectively) and Peguero-Lo Presti (AUC: 0.600; 95 % CI 0.487–0.714) (Fig. 2). No significant difference was found at the DeLong test (Table S3). Framingham Criterion showed a 52 % (95 % CI 39–65 %) Sn and 73 % (95 % CI 57–86 %) Sp. The nominally highest Sn was reached by Perugia Score (94 %; 95 % CI 85–98 %), albeit with low Sp (12 %; 95 % CI 4–26 %). The highest Sp overall was achieved by Sokolow-Lyon (90 %; 95 % CI 77–97 %) with a 27 % (95 % CI 17–40 %) Sn. According to the McNemar test, the only score to show agreement with the gold standard reference was the Romhilt Estes both for values $\geq$ 4 and $\geq$ 5. ($P = 0.568$ and 0.136, respectively, Table 3). The results of the diagnostic parameters for the other scores are reported in Table 3. Sensitivity analyses in overweight/obese and older patients were broadly consistent with the primary findings (Fig. 2, Table S5, Figure S1).

Among the HCM cohort, 75 patients (72.1 %) had a non-apical HCM phenotype (sigmoidal, reverse curve, or neutral). In this subgroup, the best diagnostic performance was observed for the Romhilt–Estes score (AUC 0.833; 95 % CI 0.789–0.877), followed by the Perugia score (AUC 0.797; 95 % CI 0.754–0.840) (Fig. 3). The Perugia score confirmed the highest sensitivity (76 %; 95 % CI 68–83 %) and was the only score showing agreement with the echocardiographic reference according to the McNemar test ($P = 0.242$). The results of other scores are presented in Fig. 3 and Table S4.

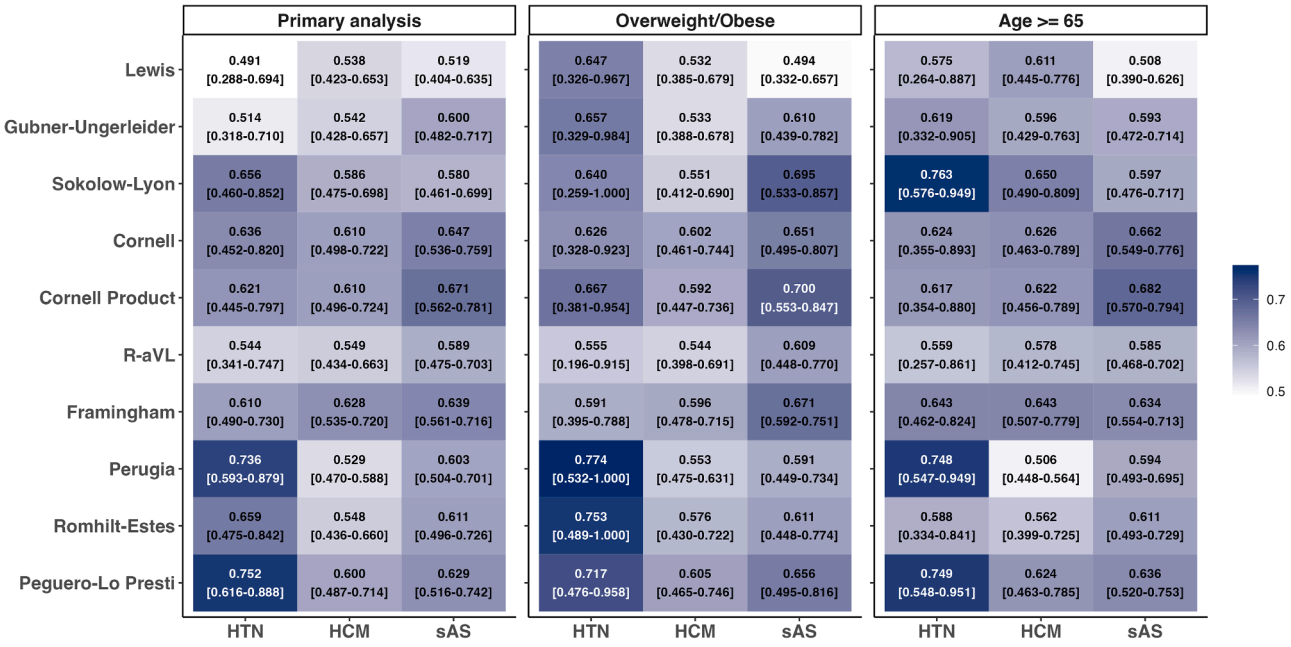

Fig. 2. Results of the ROC curve analysis for the different hypertrophic cohorts in the primary and sensitivity analyses vs echo.
AUC, Area under the curve; CI, confidence interval.

Table 2
McNemar Test Among the Electrocardiographic Criteria Against the Left Ventricular Mass Index in the hypertensive cohort.

	Sensitivity (95 % CI)	Specificity (95 % CI)	McNemar Test*
Lewis	0.23 [0.05–0.54]	0.89 [0.81–0.95]	1.000
Gubner-Ungerleider	0.15 [0.02–0.45]	1.00 [0.96–1.00]	0.003
Sokolow-Lyon	0.15 [0.02–0.45]	1.00 [0.96–1.00]	0.003
Cornell	0.31 [0.09–0.61]	0.98 [0.92–1.00]	0.070
Cornell Product	0.08 [0.00–0.36]	0.99 [0.94–1.00]	0.006
R-aVL	0.15 [0.02–0.45]	0.92 [0.85–0.97]	0.480
Framingham	0.23 [0.05–0.54]	0.99 [0.94–1.00]	0.016
Perugia score	0.54 [0.25–0.81]	0.93 [0.86–0.98]	1.000
Romhilt-Estes(≥ 5)	0.31 [0.09–0.61]	0.98 [0.92–1.00]	0.070
Romhilt-Estes (≥ 4)	0.38 [0.14–0.68]	0.98 [0.92–1.00]	0.114
Peguero-Lo Presti	0.31 [0.09–0.61]	0.94 [0.88–0.98]	0.423

* A p value <0.05 indicates lack of agreement. CI = confidence interval.

Table 3
McNemar Test Among the Electrocardiographic Criteria Against the Left Ventricular Mass Index in the HCM cohort.

	Sensitivity (95 % CI)	Specificity (95 % CI)	McNemar Test*
Lewis	0.25 [0.15–0.38]	0.80 [0.65–0.91]	<0.001
Gubner-Ungerleider	0.17 [0.09–0.29]	0.85 [0.71–0.94]	<0.001
Sokolow-Lyon	0.27 [0.17–0.40]	0.90 [0.77–0.97]	<0.001
Cornell	0.37 [0.25–0.50]	0.85 [0.71–0.94]	<0.001
Cornell Product	0.24 [0.14–0.36]	0.83 [0.68–0.93]	<0.001
R-aVL	0.27 [0.17–0.40]	0.83 [0.68–0.93]	<0.001
Framingham	0.52 [0.39–0.65]	0.73 [0.57–0.86]	0.005
Perugia score	0.94 [0.85–0.98]	0.12 [0.04–0.26]	<0.001
Romhilt-Estes (≥ 5)	0.57 [0.44–0.70]	0.46 [0.31–0.63]	0.568
Romhilt-Estes (≥ 4)	0.73 [0.60–0.83]	0.32 [0.18–0.48]	0.136
Peguero-Lo Presti	0.48 [0.35–0.61]	0.71 [0.54–0.84]	0.003

* A p value <0.05 indicates lack of agreement. CI = confidence interval.

Diagnostic performance of the scores in the sAS cohort

In ECG analysis, the nominally higher predictive performance for LVH was exhibited by Cornell Product (AUC: 0.671; 95 % CI 0.562–0.781), followed by Cornell (AUC: 0.647; 95 % CI 0.536–0.759) and Peguero-Lo Presti (AUC: 0.629; 95 % CI 0.516–0.742). The results of the ROC analysis for the sAS are reported in Fig. 2. Cornell Product showed a 31 % (95 % CI 20–44 %) Sn and 85 % (95 % CI 70–94 %) Sp.

The highest sensitivity was reached by Perugia score (66 %; 95 % CI 52–77 %) followed by Romhilt-Estes especially for values ≥ 4 (57 %; 95 % CI 44–70 %). The highest overall specificity was achieved by Gubner-Ungerleider (95 %; 95 % CI 83 %−99 %). At McNemar test, the only two scores that showed agreement against LVH were Romhilt-Estes (≥ 4) and Perugia score (respectively $P = 0.082$ and $P = 0.749$). All the results of the diagnostic performance are reported in the Table 4.

Sensitivity analyses in overweight/obese and older patients were broadly consistent with the primary findings (Fig. 2, Table S5, Figure S1).

Discussion

In this study, we sought to provide a direct comparison of ten different ECG scores for the diagnosis of LVH and to test their diagnostic performance in HTN, HCM and sAS.

The main findings of the present study can be summarized as follows: i) in the HTN cohort, only 13 % of patients had LVH. In this cohort, the Peguero–Lo Presti score showed the highest nominal predictive performance for LVH, with the highest sensitivity (54 %) reached by Perugia Score and highest specificity (100 %) by Sokolow-Lyon and Gubner-Ungerleider; ii) within the HCM cohort, the Perugia score achieved the highest sensitivity (94 %), making it the most effective score for ruling out LVH, whereas the Sokolow–Lyon criterion showed the highest specificity (90 %); iii) the diagnostic performance of the ECG scores in the HCM cohort varied according to the HCM phenotype. In particular, performance differed when patients with apical HCM were excluded, highlighting that ECG sensitivity and specificity are influenced by the type of hypertrophic involvement; iv) in the sAS cohort, highest sensitivity (66 %) and highest specificity (95 %) were showed by Perugia and Gubner-Ungerleider respectively with suboptimal predictive performances across the board; and iv) Perugia and Romhilt-Estes were the only two scores to consistenly show agreement against LVMi.

As reported in the first validation study of the Peguero–Lo Presti score[9], this criterion showed the highest predictive performance for

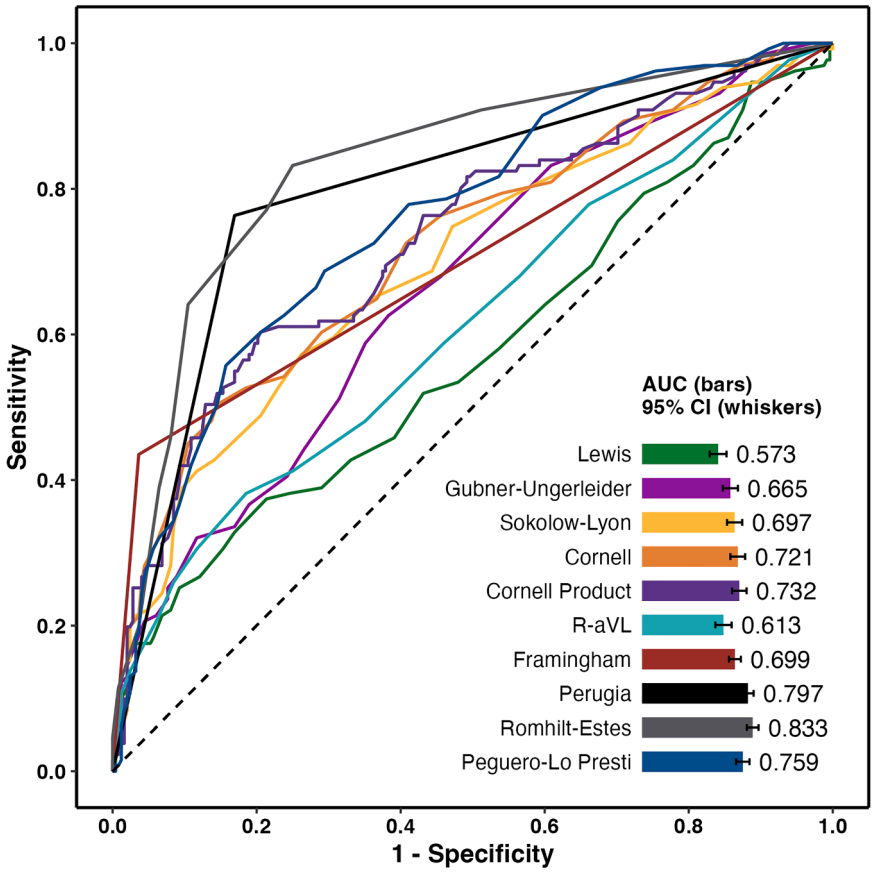

Fig. 3. AUCs of the HCM cohort of the different ECG scores to predict left ventricular hypertrophy excluding apical form.
AUC, Area under the curve; CI, confidence interval.

Table 4
McNemar Test Among the Electrocardiographic Criteria Against the Left Ventricular Mass Index in the sAS cohort.

	Sensitivity (95 % CI)	Specificity (95 % CI)	McNemar Test*
Lewis	0.18 [0.09–0.30]	0.88 [0.73–0.96]	<0.001
Gubner-Ungerleider	0.07 [0.02–0.16]	0.95 [0.83–0.99]	<0.001
Sokolow-Lyon	0.13 [0.06–0.24]	0.88 [0.73–0.96]	<0.001
Cornell	0.31 [0.20–0.44]	0.88 [0.73–0.96]	<0.001
Cornell Product	0.31 [0.20–0.44]	0.85 [0.70–0.94]	<0.001
R-aVL	0.18 [0.09–0.30]	0.90 [0.76–0.97]	<0.001
Framingham	0.38 [0.26–0.51]	0.90 [0.76–0.97]	<0.001
Perugia score	0.66 [0.52–0.77]	0.55 [0.38–0.71]	0.749
Romhilt-Estes (≥ 5)	0.34 [0.23–0.48]	0.78 [0.62–0.89]	<0.001
Romhilt-Estes (≥ 4)	0.57 [0.44–0.70]	0.65 [0.48–0.79]	0.082
Peguero-Lo Presti	0.43 [0.30–0.56]	0.75 [0.59–0.87]	<0.001

* A p value <0.05 indicates lack of agreement. CI = confidence interval.

ECG-based detection of LVH in HTN patients when compared with traditional voltage scores. In our cohort, the Peguero–Lo Presti score similarly achieved the highest AUC value among all the tested scores (AUC = 0.75), although slightly lower than that reported in the original validation cohort (AUC = 0.80).

These findings confirm the good diagnostic accuracy of the Peguero–Lo Presti score for identifying LVH through electrocardiographic criteria, while minor differences in performance may be explained by diversities in the study populations.

In the original article[9], the Peguero–Lo Presti was tested in two different cohorts: a test cohort comprising of 47 patients presenting with hypertensive crisis and 47 normotensive subjects (4 of whom had a previous history of arterial hypertension) with LVMi respectively of 126 ± 42 g/m^2 and 78 ± 18 g/m^2, 60 % of the total group had LVH by echocardiogram; a validation cohort of 122 consecutive patients referred for an echocardiogram that had a concomitant electrocardiogram for review regardless of the initial admitting diagnosis, 69 % had history of arterial hypertension, 42 % presented with LVH by echocardiogram with a mean left ventricular mass index of 107 ± 37 g/m^2.

In our hypertensive cohort, the mean LVMi was lower (79 ± 21 g/m^2), and the definition of hypertension differed slightly from that used in the original study. These differences likely account for the lower prevalence of LVH in our population (≈10 %), which in turn influences the observed diagnostic parameters. Specifically, we found a sensitivity of 31 % and a specificity of 94 %, compared with 57 % and 90 %, respectively, in the original validation cohort. This discrepancy is not unexpected, since in a population with a lower prevalence of LVH, a higher specificity and lower sensitivity are anticipated, reflecting the intrinsic trade-off of diagnostic performance metrics in low-event-rate settings[35].

When considering all scores (other than voltage-only scores), Perugia score showed a similar predictive performance (AUC 0.736) with higher sensitivity (54 %) and similar specificity (93 %). Similar results were found in the original Perugia validation cohort, even if a lower sensitivity was exhibited[6]. Compared to Peguero-Lo Presti, the highest sensitivity can be explained by the multi-parametric nature of the score encompassing a voltage criterion, an ECG strain criterion and a Romhilt-Estes ≥ 5 which is a multi-parametric score by itself.

Analogously, similar results were reached in the HCM cohort where the Peguero-Lo Presti showed only a modest ability to detect LVH, similar to other voltage-only ECG scores criteria tested; multi-parametric scores, particularly Perugia, overall yielded a higher diagnostic performance in this cohort. These results were maintained in the

non-apical HCM subgroup (Table S4); furthermore, sensitivity, specificity and AUCs greatly increased across all multi-parametric scores (except for Perugia sn which decreased to 76 %), while this effect was not seen in voltage-only criteria (Table S4, Fig. 3).

Several factors may account for these findings. First, up to 20 % of patients with HCM may present with normal left ventricular mass by echocardiography[18]. Moreover, the heterogeneous distribution of hypertrophy in HCM limits the accuracy of the Devereux formula, potentially leading to discrepancies between a negative echocardiographic criterion and a positive ECG finding[36]. Indeed, our sensitivity analysis conducted only in patients without apical HCM showed an improvement in the diagnostic performance of various scores, with the Perugia score being the one with the best sensitivity both in the main and in the sensitivity analysis. This result could be explained by the ECG modifications both seen in overt apical HCM and mild apical HCM ("relative" apical HCM), leading to a discrepancy between LVH measured by echocardiogram and LVH detected by ECG scores [37].

Second, distinct aetiologies of LVH (including HCM), often produce multiple electrical abnormalities such as increased voltages in specific leads, ST–T segment alterations, pathologic Q waves and QRS fragmentation rather than a single, uniform ECG modification[25]. Third, cardiac magnetic resonance (CMR) is the gold standard to quantify LVMi especially in HCM patients where yields higher diagnostic accuracy compared to echocardiography, which produces non-neglectable discrepancies in evaluating LVH and left ventricular wall thickness (LVWT) [38–41].

Previous studies have consistently shown that voltage-based criteria alone provide variable and generally modest diagnostic accuracy for HCM. Rowin et al. analyzed ECGs from 114 patients with HCM and found that 62 (54 %) had a Romhilt–Estes score $\geq$4, 64 (56 %) had negative T waves, 28 (25 %) had ST-segment deviations, and 49 (43 %) showed pathologic Q waves, while only 2 % fulfilled isolated QRS voltage criteria for LVH [42]. These results were coherent with other studies in similar cohorts [23.24].

In light of these findings, it is not surprising that multi-parametric scores incorporating repolarization abnormalities, such as Romhilt–Estes and Perugia scores, showed moderate to high sensitivity (71 % and 94 %, respectively) with Romhilt-Estes being the only score to show agreement against LVMi. However, data on the use of multi-parametric scores in HCM patients are scarce. Dohy et al. investigated 146 HCM patients showing a higher diagnostic performance for LVH of Romhilt-Estes compared to traditional voltage-only criteria[19] but to date no studies have investigated the potential role of Perugia score in HCM patients.

Our results suggest that Perugia score, specifically, can be used consistently to rule out LVH in HCM patients when negative [43] and a further confirmation in a larger HCM population is advisable. In this cohort, Sokolow-Lyon was confirmed as an optimal rule-in score for LVH.

In the sAS cohort Perugia Score showed again the highest sensitivity (66 %) even though several other ECG scores showed a slightly but not significantly higher AUC. Romhilt–Estes ($\geq$4) and Perugia were the only ECG scores showing agreement against LVMi; both scores incorporate not only QRS voltage criteria but also repolarization abnormalities and left ventricular strain patterns, which are frequently observed in patients with severe aortic stenosis. Thus, their diagnostic performance in this specific cohort is explained by their multi-parametric nature whereas traditional voltage-only ECG criteria showed a modest only diagnostic performance. These findings may be further explained considering that 17 patients presented a low-flow low-gradient sAS and 5 a paradoxical low-flow low-gradient [44]; furthermore, 17 sAS patients presented eccentric hypertrophy.

Lastly, Gubner-Ungerleider confirmed its potential role as a rule-in score for LVH in sAS patients.

When we conducted specific sensitivity analyses in older and overweight/obese patients for each subgroup, particularly indexing LVM to height^{2,7} and applying a BMI-corrected Perugia score, no substantial differences were observed compared with the main analysis, except for small deviations for Sokolow-Lyon, Perugia and Cornell Product in the HTN subgroup. Considering these findings, the incremental value of systematically performing such alternative indexation strategies and score adjustments in HCM and sAS cohorts remains uncertain.

The application of artificial intelligence (AI) in complex diseases such as sAS and HCM could help in the diagnosis of LVH in these settings, but appropriate studies must be conducted [45–48].

Study limitations

Some limitations of our study should be acknowledged. First, this was a single-centre, retrospective study, and potential confounders related to the regional prevalence of the investigated diseases cannot be excluded. Second, as our institution is a tertiary referral centre in the Emilia-Romagna region (Italy), the enrolled patient cohorts may not be fully representative or generalisable to other populations. Third, the diagnosis of LVH was based exclusively on echocardiographic criteria. In particular, in patients with HCM, cardiac magnetic resonance (CMR) is considered the gold standard for assessing LVMi, and its absence may have led to some degree of misclassification[36,38–40]. Fourth, inter- and intra-observer variability was not formally assessed; however, the ECG and echocardiographic analyses were performed by independent, blinded operators, which likely mitigates this source of bias.

Finally, future prospective multicentre registries are warranted to confirm the diagnostic performance of the criteria analysed in specific clinical settings such as HCM and severe aortic stenosis.

Conclusion

The prevalence and pattern of echocardiographic LVH differed markedly across the clinical conditions examined. Accordingly, the diagnostic performance of ECG scores varied substantially between cohorts. Voltage-only ECG scores performed better in HTN patients while scores incorporating non-voltage and morphological features showed relatively better performance in conditions characterized by more complex hypertrophic patterns, such as HCM and sAS.

Declaration of competing interest

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ejim.2026.106725.

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