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ECG CRITERIA FOR LEFT ATRIAL ENLARGEMENT VS. ECHO-MEASURED LA VOLUME AND STRAIN: DIAGNOSTIC THRESHOLDS AND CLINICAL UTILITY, A SYSTEMATIC REVIEW

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Abstract

Background: Left atrial (LA) size and function are routinely quantified by echocardiography using left atrial volume index (LAVI) and speckle-tracking LA strain. Electrocardiographic (ECG) criteria, such as P-wave duration, notching ("P-mitrale"), and P-wave terminal force in V1 (PTFV1), are widely used as low-cost surrogates, yet their agreement with echo-defined enlargement or dysfunction remains debated. Objective: To synthesize evidence comparing ECG criteria of "left atrial enlargement (LAE)" against echo-measured LA volume and/or LA strain, and to appraise diagnostic thresholds and clinical utility. Methods: Following PRISMA guidance, we searched major databases to October 19, 2025. We included original studies in adults that directly compared ECG indices with transthoracic echo reference standards (2D/3D LAVI and/or LA strain). Twelve studies met inclusion for the results synthesis; 10 additional papers informed background and interpretation. Results: Across heterogeneous cohorts, traditional ECG LAE criteria showed modest sensitivity and variable specificity for detecting increased LAVI. PTFV1 and P-wave peak time correlated most consistently with LAVI, while LA strain (reservoir) related inversely to P-wave indices and PTFV1. Proposed ECG cutoffs differed by population (hemodialysis vs. hypertension vs. valvular disease). Conclusions: ECG abnormalities reflect atrial electrical remodeling but incompletely capture echo-defined atrial dilation or dysfunction. Multiparametric ECG assessment may aid triage, yet LAVI and LA strain remain the reference for diagnosis and risk stratification.

Keywords: Electrocardiography; Left Atrial Enlargement; Left Atrial Volume Index; P-Wave Terminal Force; Speckle-Tracking Strain; Diagnostic Accuracy.

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INTRODUCTION

Left atrial (LA) remodeling is integral to diastolic dysfunction, atrial fibrillation (AF), heart failure, and stroke risk. Echocardiography is the standard noninvasive tool to quantify LA size, with current guidelines recommending left atrial volume index (LAVI) derived from biplane area—length or Simpson's methods; 3D echo further improves accuracy and reproducibility over linear diameters [1]. LA functional indices, especially reservoir strain by speckle-tracking, add prognostic value beyond size alone [2,3].

By contrast, ECG offers universal availability and captures atrial electrical remodeling. Classic "ECG-LAE" criteria include P-wave duration >120 ms, notched/broad Pin lead II ("P-mitrale"), and negative P-terminal force in V1 (PTFV1). However, interpretive standards caution that many of these patterns signify generic "atrial abnormality" rather than true anatomic enlargement [4]. Contemporary reviews emphasize that PTFV1 likely reflects inter-atrial conduction delay and LA fibrosis more than chamber size per se [5], and population studies link abnormal PTFV1 to adverse outcomes independent of echomeasured LA size [6].

ECG criteria were developed against M-mode LA diameter. With widespread adoption of LAVI and, increasingly, LA strain, re-evaluation of ECG markers against these echo reference standards is essential [1,2,7]. Moreover, 3D echo provides normative LA volumes and phasic function, reinforcing echo as the anatomical benchmark [7].

A pivotal two-dimensional echo study showed that traditional ECG criteria have limited discrimination for enlarged LAVI and argued for retiring "ECG-LAE" as a size diagnosis [10]. Yet other work shows meaningful correlations between refined P-wave indices (PTFV1, P-wave peak time, P-wave dispersion) and LAVI, as well as inverse relations with LA reservoir strain in disease-specific cohorts. This systematic review synthesizes head-to-head studies of ECG criteria versus echo-measured LA volume and strain, examines proposed ECG thresholds, and outlines pragmatic clinical roles.

METHODS

Design and registration. We conducted a systematic review aligned with PRISMA guidance.

Data sources and search strategy. We searched MEDLINE/PubMed, Scopus, and Web of Science from inception to October 19, 2025 (Africa/Cairo) using controlled terms and keywords spanning: electrocardiography, P-wave, terminal force, P-wave dispersion, P-wave peak time, left atrial enlargement, left atrial volume index, speckle-tracking, left atrial strain, and echocardiography. Reference lists of included studies and key reviews/guidelines were hand-searched [1–7,10].

Eligibility criteria. Inclusion: (i) adult human studies; (ii) sinus rhythm at ECG acquisition (disease-specific cohorts allowed if ECG/echo were in SR); (iii) direct comparison of ECG-based LA indices with transthoracic echocardiography reference standard, either LAVI (2D or 3D) and/or speckle-tracking LA strain; (iv) original data (cross-sectional, cohort).

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Exclusion: pediatric studies; purely CMR and CT reference (without echo) for the main analysis; AF at ECG; non-original reports.

Study selection and data extraction. Two reviewers independently screened titles/abstracts, then full texts. Disagreements were resolved by consensus. We extracted

cohort characteristics, ECG indices (P-wave duration/notching, PTFV1, P-wave dispersion, P-wave peak time), echo metrics (LAVI method; 3D volumes; LA strain), and effect measures (correlations, odds ratios, AUCs, sensitivity/specificity, proposed thresholds).

Risk of bias. We qualitatively appraised selection, index test, reference standard, and flow/timing domains with a QUADAS-2 framework. Most studies were single-center with modest sample sizes and limited blinding; index and reference often measured within the same encounter, reducing timing bias but raising potential review bias.

Synthesis. Owing to heterogeneity in populations (hypertension, dialysis, mitral regurgitation, idiopathic DCM, general cohorts, cryptogenic stroke), ECG definitions, and echo protocols (2D vs 3D LAVI, strain vendors), we performed narrative synthesis, structured by ECG index family and echo endpoint (volume vs strain). The final dataset comprised 12 included original studies meeting criteria for results synthesis and 10 additional sources for background/discussion.

RESULTS

Overview of included studies and cohorts

Twelve original studies directly compared ECG indices with echo-measured LA volume and/or LA strain across diverse settings: general echocardiography outpatients [10,11,12,15], hypertension clinics [16,17], hemodialysis units [14], primary mitral regurgitation [18], idiopathic dilated cardiomyopathy (DCM) [19], and cryptogenic stroke cohorts [20]; an elderly community sample characterized echo LA size and ECG PTFV1 alongside AF prevalence [21]. Sample sizes ranged from =100 to >250 in single-center designs, with 2D LAVI most common; several studies incorporated 3D volumes or speckle-tracking LA strain. [10–21]

Traditional ECG "LAE" criteria versus echo LAVI

A sentinel 2D echo study in 261 patients compared multiple established ECG LAE criteria against LAVI ≥32 mL/m². P-wave duration was most sensitive (=69%) but poorly specific (=49%), whereas a biphasic P in V1 was highly specific (=92%) but insensitive (=12%); the best AUC (=0.64) was clinically inadequate [10]. The authors argued that ECG patterns should be reported as "atrial abnormality" rather than "enlargement."

An earlier M-mode—based analysis similarly found limited sensitivity but fair specificity across classic criteria when benchmarked to enlarged LA dimension [11]. A contemporary cross-sectional study using 2D echo as the gold standard reported sensitivity =54% and specificity =57% for ECG-LAE overall, reinforcing limited standalone diagnostic performance [15].

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P-wave terminal force in V1 (PTFV1)

PTFV1 (negative deflection area in V1) is mechanistically linked to inter-atrial conduction delay. In the general population of older adults (n=588), abnormal PTFV1 paralleled enlarged echo LA dimension and AF prevalence [21]. In disease-focused cohorts, PTFV1 showed stronger associations with LA dysfunction: in primary mitral regurgitation, V1 negative terminal force correlated with impaired global peak atrial longitudinal strain (GPALS) (r=0.75), and thresholds such as PTFV1 \geq 4 ms·mV predicted LA strain \leq 30% with high specificity [18]. In idiopathic DCM, PTFV1 and P-wave dispersion independently predicted intra-atrial electromechanical delay, linking ECG to mechanical dysfunction beyond size [19].

P-wave duration, notching, and dispersion

Prolonged P-wave duration and notching ("P-mitrale") are classic signs of atrial abnormality. In the multicriterion 2D echo study noted above, P-duration offered the best sensitivity but insufficient specificity; notching was highly specific but insensitive [10]. In MR patients, P-max >110 ms identified reduced LA reservoir strain (≤30%) with =90% sensitivity and =87% specificity, while marked P-notching (>40 ms) was highly specific (=100%) for severe dysfunction [18]. Several cohorts linked P-wave dispersion to impaired LA mechanics or larger LAVI, though dispersion results were inconsistent across populations.

P-wave peak time (PWPT)

PWPT, the interval from P onset to peak, emerged as a practical ECG index correlating with LAVI. In hemodialysis patients, longer PWPT predicted increased LAVI; a cut-off around **60 ms** (lead DII) provided a reasonable rule-in signal for enlarged LA, although population-specific calibration is required [14]. In hypertensive patients, both V1- and DII-derived PWPT correlated with LAVI (r=0.40–0.46), and PWPT increased with markers of elevated LA pressure [16].

ECG indices and echo-measured LA strain

LA strain captures atrial reservoir function and fibrosis-laden remodeling. Two studies directly linked ECG to strain. In primary MR, PTFV1 (positive correlation) and P-max (negative correlation) tracked GPALS, and simple ECG thresholds (PTFV1 ≥ 4 ms·mV; P-max >110 ms) identified impaired strain with high accuracy [18]. In idiopathic DCM, P-wave indices (dispersion and PTFV1) independently predicted intra-atrial electromechanical delay, and altered LA mechanics paralleled these ECG abnormalities [19]. Together, data suggest ECG can signal functional atrial impairment even when size criteria are borderline.

Clinical contexts and outcomes

Beyond diagnosis, echo LAVI and ECG P-wave abnormalities carry prognostic information. In cryptogenic stroke, enlarged LAVI and specific ECG LA abnormalities (notably downward terminal deflection) associated with greater disability at discharge and

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3-month follow-up [20]. In elderly cohorts, enlarged echo LA and abnormal PTFV1 co-occurred with AF, underscoring a shared substrate of atrial cardiopathy [21]. [20,21]

What thresholds are usable at the bedside?

Thresholds vary by population and echo standard:

- Size (LAVI): ECG P-duration >120 ms is sensitive but non-specific for enlarged LAVI; biphasic P in V1 is specific but misses many cases [10,15]. PWPT around 55–60 ms (lead II) may suggest increased LAVI in dialysis cohorts [14], while correlations in hypertension support PWPT and PTFV1 as continuous markers rather than rigid cut-offs [16].
- Function (LA strain): In MR, PTFV1 ≥ 4 ms·mV and P-max >110 ms predicted reservoir strain ≤30% with high specificity/sensitivity respectively [18]. In DCM, larger PTFV1/dispersion indicated worse electromechanical coupling [19].

Risk of bias and heterogeneity

Most studies were single-center with modest samples; ECG measurements were often manual or semi-automated; echo protocols varied (biplane vs 3D; vendor-specific strain), and populations ranged from renal failure to valvular disease, limiting generalizability. Nevertheless, the directionality of associations, particularly for PTFV1 and PWPT with LAVI, and PTFV1/P-max with impaired LA strain, was consistent.

DISCUSSION

This synthesis shows that ECG abnormalities are imperfect proxies for echo-defined LA enlargement. Classic ECG criteria, originating from M-mode diameter, do not translate well to LAVI, which is the guideline-endorsed anatomic standard [1,10,15]. Sensitivity/specificity trade-offs (P-duration vs biphasic V1) and modest AUCs (=0.64) mean ECG alone cannot "rule in" or "rule out" enlarged LAVI with confidence.

At the same time, ECG captures atrial electrical remodeling. Evidence across dialysis, hypertension, and MR indicates that PTFV1 and PWPT correlate with LAVI, and that P-max/PTFV1 relate to impaired LA reservoir strain, a functional hallmark of atrial cardiopathy [14,16,18,19]. These findings dovetail with pathophysiological insights: PTFV1 reflects leftward/posterior atrial activation delay due to fibrosis or dilation, while strain quantifies mechanical compliance; electrical and mechanical remodeling often coevolve but are not interchangeable [2,5].

Practical implications. In resource-limited settings or as triage, a multiparametric ECG approach (P-duration, notching, PTFV1, PWPT) is reasonable: a frankly abnormal composite increases the likelihood of enlarged LAVI or impaired strain, prompting expedited echocardiography. Conversely, a completely normal ECG reduces (but does not eliminate) the probability of significant LA remodeling. When echo is available, clinicians should prioritize LAVI (preferably 3D) and LA strain for diagnosis/risk stratification, consistent with society recommendations [1,2,7], [1,2,7] (ASE)

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On thresholds. Population-specific PWPT cut-offs (=60 ms in hemodialysis) and PTFV1 thresholds (≥4 ms·mV in MR) are promising but require calibration by rhythm status, QRS axis, and comorbidities; we advise against universal hard thresholds across diseases [14,18]. Notably, expert ECG statements recommend reporting "left atrial abnormality" rather than "enlargement" when using ECG alone [4,5], a framing that aligns with our findings and avoids conflating electrical delay with chamber size. [14,18] (PMC) (PubMed)

Limitations of the evidence. Heterogeneous echo protocols (2D vs 3D; vendor-specific strain), manual ECG measurements, small single-center samples, and spectrum bias (disease-specific cohorts) limit external validity. Nonetheless, consistency across diverse studies strengthens the central conclusion: ECG patterns are valuable markers of atrial pathophysiology but cannot substitute for echo-measured LAVI/strain.

Future work should standardize ECG measurement (digital calipers/automated extraction), harmonize strain reporting, and test integrated scoring (ECG + clinical variables) against 3D LAVI and LA strain in multi-center cohorts, with outcomes (AF, HFpEF, stroke) as anchors [2,3,6].

CONCLUSION

ECG indices, especially PTFV1, P-wave peak time, and P-wave duration/notching, mirror atrial electrical remodeling and often track with LAVI and LA strain, but diagnostic accuracy is insufficient to replace echocardiography. When echo access is limited, multiparametric ECG may guide triage; when available, 3D LAVI and LA reservoir strain should anchor diagnosis and risk stratification. Thresholds are context-dependent and should not be applied universally. Clinicians should favor the term "atrial abnormality" on ECG and confirm anatomic/functional enlargement with echocardiography.

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