Real-world performance, long-term efficacy, and absence of bias in the artificial intelligence enhanced electrocardiogram to detect left ventricular systolic dysfunction

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Aims

Some artificial intelligence models applied in medical practice require ongoing retraining, introduce unintended racial bias, or have variable performance among different subgroups of patients. We assessed the real-world performance of the artificial intelligence-enhanced electrocardiogram to detect left ventricular systolic dysfunction with respect to multiple patient and electrocardiogram variables to determine the algorithm’s long-term efficacy and potential bias in the absence of retraining.

Methods and results

Electrocardiograms acquired in 2019 at Mayo Clinic in Minnesota, Arizona, and Florida with an echocardiogram performed within 14 days were analyzed (n = 44,986 unique patients). The area under the curve (AUC) was calculated to evaluate performance of the algorithm among age groups, racial and ethnic groups, patient encounter location, electrocardiogram features, and over time. The artificial intelligence-enhanced electrocardiogram to detect left ventricular systolic dysfunction had an AUC of 0.903 for the total cohort. Time series analysis of the model validated its temporal stability. Areas under the curve were similar for all racial and ethnic groups (0.90–0.92) with minimal performance difference between sexes. Patients with a ‘normal sinus rhythm’ electrocardiogram (n = 37,047) exhibited an AUC of 0.91. All other electrocardiogram features had areas under the curve between 0.79 and 0.91, with the lowest performance occurring in the left bundle branch block group (0.79).

Conclusion

The artificial intelligence-enhanced electrocardiogram to detect left ventricular systolic dysfunction is stable over time in the absence of retraining and robust with respect to multiple variables including time, patient race, and electrocardiogram features.
Introduction

Left ventricular systolic dysfunction (LVSD) is a common pathology associated with significant morbidity and mortality.\(^1\)–\(^3\) Although no routine screening strategies are currently recommended, robust efforts have been made to identify patients with LVSD.\(^4\)–\(^6\) Early identification may permit treatment with evidence-based therapies to mitigate morbidity and mortality.\(^1\)

The electrocardiogram (ECG) is a rapid, cost-effective, point-of-care test that is available in most clinical settings, both inpatient and outpatient. We have previously reported the development and clinical application of a deep learning network to detect LVSD using an artificial intelligence-enhanced ECG (AI ECG) in patients in primary care clinics and the emergency department.\(^7\)–\(^10\) The AI ECG remains promising as a non-invasive screening tool for patients with previously undiagnosed LVSD, including those with suspected myocardial dysfunction secondary to SARS-CoV-2 infection,\(^11\) for which it has received US Food and Drug Administration Emergency Use Authorization.

It is well known that a trained network is susceptible to data set shift errors when employed in a setting that differs from its original training environment. In some cases, AI models have to re-learn or recalibrate to better fit an environment or setting.\(^12\) Although our group has performed both internal and external validation of the AI ECG algorithm to detect LVSD with promising results,\(^13,14\) its stability with respect to potentially impactful clinical factors (i.e. patient age, ECG recording location, underlying arrhythmia, time since network training) remains undetermined. To test the hypothesis that the AI ECG network for LVSD is stable over time and across populations due to its broad training set, we assessed its accuracy with respect to time since development, patient age, patient race and ethnicity, ECG rhythm and morphology, and ECG recording location.

Methods

Study population

All ECGs acquired between 1 January and 31 December 2019 (the year following algorithm development) performed at Mayo Clinic in Rochester, Minnesota, Scottsdale, Arizona, Jacksonville, Florida, and Mayo Clinic Health System locations were evaluated (\(n = 406,916\) from 230,661 patients). Eligible ECGs were digital, standard 10 s 12-lead ECGs acquired in the supine position during the study timeframe. ECGs from patients who underwent transthoracic echocardiography within 2 weeks of an eligible ECG were included in this analysis. In the case of multiple echocardiograms for a single patient, the earliest echocardiogram was selected as the index echocardiogram. In the case of multiple ECGs in the 14-day window from echo, the ECG temporally closest to the first echo was selected for analysis (\(n = 44,986\) patients with valid ECG-echo pairs). None of the patients in this analysis participated in the derivation of algorithm, and all ECGs in the analysis were acquired after the model was fully developed.

Patient data

The institutional review boards of the Mayo Clinic Foundation approved of this study and protocol. Following institutional review board approval, patient age and self-reported race and ethnicity were collected from the Mayo Clinic unified data platform. The original protocol was modified to include patients <18 years of age, and the institutional review board approved this change. The ECG date, acquisition location, and rhythm were extracted from the MUSE system (GE Healthcare, Marquette, WI). For all ECGs, the final rhythm and other ECG findings were adjudicated by a technologist under cardiology supervision.

All echocardiograms had one or more ejection fraction (EF) measurement performed by a cardiologist. For studies with more than one left ventricular ejection fraction measurement, we used a heuristic technique to select the most accurate measurement.\(^7\) The preferred measurement
used for analysis was (from most accurate to least accurate): 3D echocardiography, biplane imaging using the Simpson method, 2D methods, M-mode measurements, and, in the absence of any of the preceding, the reported visually estimated EF.

Artificial intelligence model

The artificial intelligence model used in this study has been previously described. Briefly, the model uses a convolutional neural network that analyzes a matrix with 10 s, 12-lead ECG data resampled to 500 Hz (5000 × 12 values per ECG). Each matrix row contains the raw amplitude for each of the 12 leads for that timestamp. The model uses seven convolutional blocks, each with a convolutional layer, batch normalization, a ‘Relu’ activation function and a max-pooling layer, followed by two fully connected blocks. The model was developed using 44,959 unique patients and was tested on 52,870 patients not used to develop the model. In the original testing cohort, the model was able to detect an EF ≤ 35% with an area under the curve (AUC) of 0.93 and an EF ≤ 40% with an AUC of 0.91.

Although the model was developed and tested prior to the start date of the present study, to avoid any data leakage, patients used to develop the model were excluded from the current analysis, as we assume that the model might have an unfair advantage when testing the same patients again due to the biometric information in the ECG.

Main outcome

While the original publication focused on the detection of EF ≤ 35%, in this work we adjusted the threshold for the definition of low EF to ≤ 40%, in accordance with the Universal Definition and Classification of Heart Failure, European Society of Cardiology, and American College of Cardiology professional society guidelines. The AUC for the detection of EF ≤ 40%, was evaluated with respect to multiple factors: time (in 1-month blocks, for each month of 2019), patient age (in 10-year intervals), race and ethnicity, ECG recording location and situation (inpatient, outpatient or emergency department), echocardiogram measurement type used to assess EF, and ECG features at the time of screening.

The focus of the statistical analysis was providing descriptive data on the performance of the algorithm over the range of patient profiles identified above. No formal hypothesis testing was configured to test for differences from the original area under the receiver operating characteristic curve. Instead, differences in the point estimates of AUC between the original sample and the new validation cases greater than 3 percentage points were considered because the original study yielded differences in AUC among the groups exist. In this validation cohort, 43 more comorbid than patients with an EF >40%. Details of age, race, and comorbidities by EF value are described in Table 1. Expanded race and ethnicity characteristics are described in Supplementary material online, Table S1.

Temporal evaluation

Evaluation of the AUC by month is shown in Figure 1. The per-month AUC remains greater than 0.89 throughout the year of 2019 without significant variation. There is moderate variability of the confidence interval range on a month-to-month basis; however this does not significantly impact overall accuracy of the algorithm over time. This visual finding was supported with the time series model. In particular, the estimated slope of the performance over the 12-month period was 0.0002 consistent with temporal stability of the model.

Age group and sex evaluation

Evaluation of AUC with respect to age revealed robust test performance (AUC >0.87) for the age groups 0–10 years old and 20–80 years old (Figure 2A). The AUC was lower for patients with 10–20 years of age and greater than 80 years (AUC 0.831–0.843); however, the sample size of the number of patients aged under 20 years of age was small (n = 1183). The AUC was minimally different between male and female patients (Figure 2B).

Electrocardiogram accuracy with respect to race and ethnicity

To validate that the model is not impacted by patient race, we used self-reported race and ethnicity to determine whether significant differences in AUC among the groups exist. In this validation cohort, 43

| Table 1 Baseline cohort characteristics with and without left ventricular systolic dysfunction |
|-----------------------------------------------|-------------------|-------------------|
| Age (95% CI)                                 | Female (%)        | Race (%)          |
| 63.6 (63.4, 63.7)                            | 18270 (44.5)      | Non-Hispanic White| 36601 (89.1) |
| 68.5 (68.0, 69.0)                            | 1082 (27.5)       | Black             | 1810 (4.4)  |
|                                               |                   | Other             | 2647 (6.4)   |
|                                               |                   | Hispanic (%)      | 1776 (4.3)  |
|                                               |                   | Congestive heart failure (%) | 14530 (35.4) |
|                                               |                   | Myocardial infarction (%) | 6498 (15.8)  |
|                                               |                   | Hypertension (%)  | 27090 (66.0) |
|                                               |                   | Diabetes mellitus (%) | 10283 (25.0) |
|                                               |                   | Renal disease (%) | 11997 (29.2) |
|                                               |                   | Cerebrovascular disease (%) | 8645 (21.1)  |
|                                               |                   | Peripheral vascular disease (%) | 16947 (41.3) |
|                                               |                   | COPD (%)          | 11613 (28.3) |
|                                               |                   | Connective tissue/ rheumatologic disease (%) | 2963 (7.2) |
|                                               |                   | EF below 30% by TTE (%) | 0 (0.0)  |
|                                               |                   | EF below 50% by TTE (%) | 4262 (10.4) |

*CI, confidence interval; EF, ejection fraction; TTE, transthoracic echocardiogram.
302 patients self-reported race and ethnicity. The model had similar AUC among all races and ethnicities tested (AUC: 0.90–0.93) with slightly higher AUC for black and Hispanic patients, as noted in Figures 2C, 2D, and Supplementary material online, Figure S1. Patients with unknown racial demographics had slightly lower AUC (0.899; see Supplementary material online, Figure S1).

**Area under the curve by electrocardiogram features**

AUC characteristics stratified by ECG features are depicted in Figure 3. Electrocardiogram features/findings were evaluated on an individual basis as some ECG features are mutually exclusive (NSR and atrial flutter for example). The overall evaluation for all analyzed ECGs had an AUC of 0.903 (2948 of 44919 with LVSD), similar to the AUC for detection of low EF in our original work (AUC = 0.91 for EF ≤ 40%). Patients with an ECG rhythm of ‘normal sinus rhythm’ exhibited an AUC of 0.91 for detecting EF ≤ 40%. All other ECG rhythms or ECG diagnoses had AUCs between 0.791 (for left bundle branch block) and 0.91, as shown in Figure 3.

**Discussion**

We found that the AI ECG model for LVSD was robust without re-training even for the less stringent definition of LVSD of ≤ 40% EF. Specifically, it was stable over time, and functionally invariant with regards to patient sex and race. There was some minor variability in performance based on age, ECG recording location, and ECG features that may warrant further consideration.

Neural networks are susceptible to data shift errors when networks are applied to populations that differ from those initially used for training. Although the LVSD AI ECG neural network was trained on a large and broad population, and previous retrospective analysis had shown robustness with regards to patient age and ethnicity, this is the first real-world prospective use study to show temporal stability, and robustness over a wide range of geographical usage (with inherently different populations), clinical environments (outpatient, inpatient, emergency department), and ECG features. Through each of these evaluations we identified only minor variations in algorithm accuracy across each clinically important variable. This non-invasive point-of-care screening tool exhibits significant dataset stability on multiple fronts.

The minimal variation of AUC over time in 2019 suggests that the algorithm does not require recalibration on a monthly or annual basis. These data suggest that the AI ECG for LVSD is minimally susceptible to temporal dataset shifts. However, an underlying assumption is the use of a standard 12-lead ECG acquired using clinically accepted lead positions with the patient supine. The use of ECG signals from mobile or other form factors requires additional validation, as variations in cardiac position, adrenergic tone, resting and active heart rates, and ECG morphology could impact algorithm function. Although this evaluation indicates no anticipated performance degradation over time, re-evaluation in future years may be also merited in the event of shifting disease trends, population change, or alterations in the ECG system.

The AI ECG is considered a black box, since the specific ECG features used to make EF determination is unknown. Thus, there is a concern for the introduction of implicit bias, especially as it relates to underrepresented groups. Prior to using the model in practice, we demonstrated that in our testing cohort, the LVSD model worked well in all races and ethnicities in the testing set. In this prospective validation, we found that despite the known ECG features differences across races and ethnicities, the LVSD algorithm works equally well in those that

![Figure 1](image1.png)

**Figure 1** Artificial intelligence-enhanced electrocardiogram for left ventricular systolic dysfunction area under the curve by month in the year of 2019.
were tested. Similarly, we observed robust performance of the algorithm for both male and female patients.

There was a modest drop in test performance with advanced age, with an AUC of 0.87 for patients 80–90, and 0.84 for age above 90 (Figure 2A). Noteworthy is that the most clinically relevant age group for LVSD (20–80 years of age) has the greatest test performance (AUC 0.89). Irrespective of the performance degradation with advanced age, the test showed robust performance characteristics with an AUC >0.8 for all age groups with values remaining in line with other medical tests (typically AUC 0.75). Although we observed an acceptable AUC in patients 0–20 years, the number of patients with disease (n = 13) was quite small in these groups, the sensitivity for patients aged 20, 20 years was low (37.5–60.0%), and this experimental data represented a first-time application of the AI ECG for LVSD algorithm, in training or testing, to a paediatric patient population. As a result of these limitations, no conclusions can be drawn regarding algorithm use in these low prevalence, paediatric populations. Further investigation in a larger paediatric population must be completed to clarify appropriate application of this algorithm.

When validating LVSD accuracy by echocardiography, the AUC significantly decreased when the EF was measured by visual estimation (AUC <0.8). All other modes of measuring EF by echocardiography exhibited AUC greater than 0.9 (except 2D; AUC = 0.889), suggesting that the accuracy EF by visual estimation may be limited. This is consistent with previous reports describing the variability of EF measurement via visual estimation. User variability and clinical context (i.e. severely ill in emergency department with point-of-care echocardiogram) likely contribute to the variation in test performance. This raises the possibility that the AI ECG may, at times, be a more powerful predictor of LVSD than echocardiography when ventricular function is visually estimated, particularly in acute environments and clinical situations in which echocardiographic images may be challenging. However, given the limited number of patients in this subgroup (n = 133), it is difficult to draw definite conclusions without further investigation.

Location analysis reveals that the AUC was higher in the outpatient setting than in the emergency department or inpatient setting. This heightened test performance may reflect fewer confounding factors present in the clinic setting. A number of hypotheses may explain this phenomenon: (i) ambulatory patients may be less likely to have multiple active medical issues or an acute exacerbation of another disease, either of which might impact the ECG; (ii) ambulatory patients typically do not have external factors impacting clinical haemodynamics (i.e. intubation, positive pressure ventilation, use of intravenous vasoactive medications); (iii) there is likely less variation between ECG machines used in the clinic (i.e. limited number of machines at an outpatient location rather than next available ECG tech within the hospital/unit).

Finally, we hypothesized that variations in ECG rhythm and morphological features likely used by the model may impact its performance. In this analysis we sought to determine whether the presence of a paced rhythm or an intrinsic left bundle branch block (LBBB) are significantly used by the model to identify LVSD.

**Figure 2** Forest plot for artificial intelligence electrocardiogram subgroup performance. Location abbreviations in 2G are as follows: MN-Minnesota, AZ-Arizona, FL-Florida. Odds ratios (ORs) are ‘diagnostic odds ratio’ defined as the ratio between the odds of test positivity in a patient with disease and the odds of test positivity in a patient without disease.
(in which case, the AUC would approximate 0.5 in the context of LBBB, as not all individuals with LBBB actually have LVSD). However, irrespective of the absence or presence of specific ECG features, the model continued to perform with an acceptable AUC >0.8. This indicates that the model continues to effectively identify left ventricular dysfunction despite the presence of bundle branch blockade, hypertrophy, or other features listed in Figure 3. Similarly, model performance persisted in the presence of arrhythmias (Figure 3). While patients with ‘completely normal ECGs’ (i.e. absence of any arrhythmia, conduction delay, or other electrophysiologic abnormality) exhibited an AUC of 0.803, the sensitivity was quite low (19.4%) with very high specificity (99.1%) indicating the algorithm may have significant limitation as a screening tool in this subset of patients without adjusting the model threshold to have a higher sensitivity and lower specificity for patients with completely normal ECG.

### Conclusions

The AI ECG is robust screening tool for ventricular dysfunction, with strong performance over time, geography, clinical location, patient age, and ECG features. We similarly observed strong AI ECG performance with respect to variable race/ethnicity while acknowledging the limited diversity of this study population. The change in model performance with method of EF estimation emphasizes the need for objective, reproducible EF calculation methods.

### Lead author biography

David M. Harmon is an academic internist at the Mayo Clinic in Rochester, MN, USA. He is a member of the Alpha Omega Alpha Medical Honor Society and is directing research efforts at the Mayo Clinic through a National Institute of Health Stimulating Access to Research in Residency grant. His research is focused on the clinical development and application of artificial intelligence to the electrocardiogram. He aims to broaden the use of artificial intelligence technology, validating its application in...
wearable and handheld devices which hold implications for both at-home medical care and acute, emergent settings.

Supplementary material

Supplementary material is available at European Heart Journal – Digital Health.

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Data availability

Our raw data from this study is not available, however the analysis of our results are sharable.

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