S
ince the introduction of the string galvanometer by Willem Einthoven more than a century ago, the ECG has become the most commonly performed cardiovascular test and an essential diagnostic tool in clinical cardiology.1–3 The transition from analogue to digital ECGs resulted in automated computer analysis of the ECG (ECG-C) assuming a larger role in the diagnostic interpretation.3 Yet, as the ECG-C has evolved into a necessary tool of modern medical practice, many physicians remain unaware of the hazards of relying on these preliminary diagnostic interpretations. The ECG-C clinical use should always be with the understanding that they are preliminary interpretations and require reading and confirmation by qualified electrocardiographer. With an increased reliance on these readings for point-of-care decision making, clinicians must remain mindful of many limitations of the ECG-C.2,3

The ECG-C was lower than that of cardiologists in interpreting ECGs in clinically validated cases of various cardiac disorders.5 The percentage of ECGs correctly classified by the ECG-C was lower than that for the cardiologists.4 The median sensitivity of the computer programs was also significantly lower than that of the cardiologists in diagnosing left ventricular hypertrophy, right ventricular hypertrophy, anterior myocardial infarction, and inferior myocardial infarction.4 The median total accuracy level (the percentage of correct classifications) was 6.6% lower for the computer programs (69.7%) than for the cardiologists (76.3%; P<0.001).4

More contemporary analysis of the accuracy of ECG-C similarly concluded that there are frequent errors in the interpretation of the cardiac rhythm. In evaluating the ECG-C interpretation of the cardiac rhythm, the ECG-C demonstrated an overall accuracy of 88.0%.5 Sinus rhythm was correctly interpreted in 95.0% of the ECGs with this rhythm. However, nonsinus rhythms were correctly interpreted with an accuracy of only 54%.5 The ECG-C interpreted sinus rhythm with a sensitivity of 95%, specificity of 66.3%, and positive predictive value of 93.2%.5 The ECG-C interpreted nonsinus rhythms with a sensitivity of 72%, a specificity of 93%, and a positive predictive value of 59.3%.5 In addition, incorrect rhythm interpretation by the ECG-C was frequently further compounded by additional major inaccuracies.5 Of incorrect rhythm interpretation, additional major errors were noted in 137 (54%) patients.5

The clinical impact of the ECG-C misinterpretation was evaluated by other investigators who demonstrated that 19% of ECG-C had the rhythm misinterpreted as atrial fibrillation.6 Failure of the physician ordering the ECG to correct the inaccurate interpretation resulted in change in management and initiation of inappropriate treatment, including antiarrhythmic medications and anticoagulation, in 10% of patients.6 Additional unnecessary diagnostic testing was performed based on the misinterpreted ECGs in 24% of patients.5

The ECG-C failed to identify many at-risk family members when used as a screening tool for long QT syndrome (LQTS).7 The ECG-C erroneously classified 6 of 23 family members known to have LQTS as normal.7 Half of the family members, proved to have the ion channel defect, received the diagnostic interpretation normal ECG.7 The investigators concluded that reliance on the ECG-C alone fails to identify many at-risk family members with LQTS.7 The investigators suggested that all first-degree relatives of an identified LQTS proband have a 12-lead ECG that is reviewed independently by a physician who is familiar with LQTS in an effort to improve screening for this potentially lethal syndrome.7

Accurate measurement of the QT interval and its adjustment for rate, sex, and QRS prolongation remains one of the major challenges in electrocardiography.3,8–13 The matter is of great importance to physicians, drug manufacturers, and regulatory agencies because of the relationship between prolongation of the QT interval and potentially lethal ventricular arrhythmias.3,8–13 In this respect, the original contribution by Lehman and his colleagues in this issue of Circulation, Arrhythmias and Electrophysiology has particular significance.14 In this investigation, all ECG-Cs analyzed by a single system (Marquette 12SL ECG Analysis Program, GE Healthcare, Milwaukee, WI) were evaluated. ECGs were included if sinus rhythm was present with a heart rate <100 beats per minute and QRS duration <120 ms, and a prolonged ECG-C QTc value was displayed in 16.7%.14

The ECG-C used an algorithm that defined the range of normal for the QT interval as <470 ms in women, and <460 ms in men.14 However, in only 47.5% of these ECGs with prolonged QTc did the automated interpretation include an accompanying
prolonged QT diagnostic statement. The authors note that such prolonged QT under-reporting was manifest across all patient environments, and reflected algorithmic suppression of the diagnosis. This was because of ECG waveform-based criteria in 52.5% of ECGs with prolonged QTc. Of the latter ECGs with prolonged QT diagnosis suppression, the computer declared 42.1% as normal, despite QTc prolongation. The authors concluded that in evaluating an adult patient, whose ECG-C lacks a prolonged QT diagnostic statement, physicians should examine the actual QTc value displayed on the report before concluding that this parameter is normal. They also conclude that assessment of the clinical impact of prolonged QT diagnosis suppression by ECG waveform-based criteria is warranted.

It is evident that automated evaluation of the QT interval is one of the particularly important functions of any ECG-C. Identification of a prolonged heart rate corrected QT interval (QTc) may identify an individual with an inherited LQTS at-risk for sudden cardiac death because of torsade de point. Specific technical and clinical recommendations for measurement of the QT and QTc intervals on the ECG have been made in a American Heart Association consensus document written to standardize the interpretation of ECGs. It is recommended that, in addition to rate, an adjustment for sex and age be incorporated into QTc measurement. As practical clinical limits for considering the QT interval as abnormal, this document recommends that the adjusted QT of 460 ms or longer in women and 450 ms or longer in men be considered a prolonged QT interval, and that QT of 390 ms and shorter be considered a short QT interval. It is recommended that linear regression functions rather than the Bazett formula be used for QT-rate correction, and that the method used for rate correction be identified in ECG analysis reports. It is further recommended that rate correction of the QT interval should not be attempted when RR interval variability is large, as often occurs with atrial fibrillation, or when identification of the end of the T-wave is unreliable. In view of the clinical importance of the QT-interval prolongation, it is essential to validate QT-interval prolongation reported by a computer algorithm visually. In addition to administration of QT-prolonging cardioactive drugs, a number of conditions can induce QT prolongation. It is often possible to identify a specific cause of QT prolongation, when appropriate clinical information is available. Electrolyte disturbances, including hypokalemia, hypomagnesemia, and hypocalcemia, can prolong phase 2 and phase 3 of the action potential and prolong the QT interval. A comprehensive and current list of all possible causes of QT prolongation is readily available. It is evident that presence in an ECG report of QTc prolongation should call for a careful clinical evaluation of possible causes.

The report by Lehman and colleagues placing emphasis on algorithmic suppression of abnormal QTc intervals serves to extend prior observations on the limitations of the ECG-C. As noted by the authors, the study was not designed to analyze physician responses or clinical outcomes related to ECG-C interpretations. Thus, the investigators were not able to assess either extent of physician under-recognition of prolonged QTc values or any possible adverse clinical impact resulting from algorithmic suppression of a prolonged QT diagnosis. Furthermore, the study was confined to versions of the ECG manufacturer’s software. A more recent software update, now in the implementation phase, continues to rely on the previously utilized ECG waveform-based criteria for prolonged QT diagnosis reporting and suppression. However, the newer version also offers an option of automated notification of user-defined critical QTc values.

Given the profound clinical implications of the ECG-C, all clinicians must be mindful of potential for erroneous interpretation resulting in unnecessary, potentially harmful medical treatment, and inappropriate use of medical resources. The publication by Lehman serves to remind clinicians that the appropriate use of the ECG-C is as a supplement, but not a substitute, for interpretation by an electrocardiographer. Although it has been known for 3 decades that measurements made by different automated ECG-C systems from standardized reference ECG databases can vary enough to alter ECG-C diagnostic interpretation, many clinicians remain unaware of the considerable limitations of the ECG-C. Although ECG-C diagnostic statements continue to improve, it remains evident that over-reading and confirmation is still required of every ECG-C, when used for screening or making any clinical decisions.

Disclosures
None.

References


KEY WORDS: Editorials ECG
Computerized Interpretation of ECGs: Supplement Not a Substitute
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Circ Arrhythm Electrophysiol. 2013;6:2-4
doi: 10.1161/CIRCEP.111.000097

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