ORİJİNAL ARAŞTIRMA ORIGINAL RESEARCH

DOI: 10.5336/cardiosci.2020-74783

Electrocardiographic Changes in Intensive Care Unit Patients During Their Last Hours of Life: Can We Use These Parameters as Prognostic Indicators?

Yaşamın Son Saatlerinde Yoğun Bakım Hastalarında Görülen Elektrokardiyografik Değişiklikler: Bu Parametreleri Prognostik Göstergeler Olarak Kullanabilir miyiz?

Ashok PAUDEL[a], Orkhan MAMMADOV[b], Erkan EKİCİBAŞI[a], Nihan YAMAN MAMMADOV[c], Ararso Kedir JIMA[b]

[a]Acıbadem Altunizade Hospital, Acıbadem Healthcare Group, Clinic of Cardiology, İstanbul, TURKEY
[b]Acıbadem Altunizade Hospital, Acıbadem Healthcare Group, Clinic of Intensive Care, İstanbul, TURKEY
[c]Van Training and Research Hospital, Clinic of Anesthesiology, Van, TURKEY

ABSTRACT Objective: QRS amplitude attenuation and prolonged QRS duration has been associated with increased mortality in various clinical conditions including critical care patients and general population. Relative bradycardia has been found to be associated with lower mortality in patients with septic shock, but there are no studies in literature evaluating the electrocardiographic (ECG) changes and changes in heart rate (HR) just before death. Our aim of this study is to calculate the gradual changes in these parameters in the last hours of life from II derivation telemetry records. **Material and Methods:** We included 30 patients who died in intensive care unit irrespective of their diagnosis during admission and follow up. HR, QRS amplitude and QRS duration were analysed from the telemetry recordings obtained from the last 10 hours of their life. **Results:** QRS duration prolongs and heart rate decreases during the last 10 hours of life and the changes in these parameters were more prominent in the last hours. QRS duration increased at rate of 5.43 ms per hour ($p<0.001$) and heart rate decreased at rate of 2.68/min each hour ($p<0.001$). QRS amplitude attenuation were more subtle (decreased by 0.23 mV per hour, $p=0.02$) compared to QRS duration and heart rate. **Conclusion:** During last 10 hours of life, there was widening of QRS complex, attenuation of QRS voltage and decrease in heart rate. Automated softwares could present these findings in graphics and can be used as a prognostic indicators to recognize a dying patient. This information could be used in certain acute reversible critical conditions such as fulminant myocarditis, anaphylactic shock, trauma patients as a sign of poor prognosis or on decision making regarding end-of-life in irreversible illness such as terminal cancer patients.

Keywords: Electrocardiography; heart rate; critical illness; terminal care

ÖZET Amaç: QRS amplitüdünde azalma ve uzun QRS süresi, yoğun bakım hastaları ve genel popülasyon dahil olmak üzere çeşitli klinik koşullarda artan mortalite ile ilişkilendirilmiştir. Rölatif bradikardi septik şokta olan hastalarda düşük mortalite ile ilgili saptanmıştır fakat ölüm öncesi kalp hızı değişkenliği ile ilgili yapılmış çalışma bulunmamaktadır. Bu çalışmadaki amacımız yaşamın son saatlerinde bu parametrelerdeki kademeli değişiklikleri II derivasyon telemetri kayıtlarından hesaplamaktır. **Gereç ve Yöntemler:** Yatış ve gözlem sırasındaki tanılarına bakılmaksızın yoğun bakım ünitesinde ölen 30 hasta çalışmaya alındı. Kalp hızı, QRS amplitüdü ve QRS süresi, yaşamlarının son 10 saatinden elde edilen telemetri kayıtlarından analiz edildi. **Bulgular:** Yaşamın son 10 saatinde QRS süresi artıyor ve kalp hızı azalıyor; bu değişiklikler son saatlerde daha belirgin hale geliyor. QRS süresi saatte 5,43 ms artıyor ($p<0.001$) ve kalp hızı saatte 2,68/dk kadar azalıyor ($p<0.001$). QRS amplitüdünde azalma QRS süresi ve kalp hızı değişikliklerine göre daha az belirgindi (saatte 0,23 mV azalma, $p=0.02$). **Sonuç:** Yaşamın son 10 saatinde hemodinamik bozulmanın kardiyak elektrik sistemi üzerindeki etkisi, QRS kompleksinde genişleme, QRS voltajında zayıflama ve kalp hızında azalma olarak kendini gösterir. Bu bulgular bilgisayar yazılımları yardımıyla grafik olarak gösterebilir ve ölen hastayı tanımak için bir belirteç olarak kullanılabilir. Bu bilgiler, fulminan miyokardit, anafilaktik şok, travma hastaları gibi bazı akut geri dönüşümlü kritik durumlarda hayat kurtarıcı bir alarm olarak veya terminal kanser hastaları gibi geri dönüşümsüz hastalıklarda yaşam sonu ile ilgili karar vermede kullanılabilir.

Anahtar Kelimeler: Elektrokardiyografi; kalp hızı; kritik hastalık; terminal dönem bakımı

Correspondence: Orkhan MAMMADOV
Acıbadem Altunizade Hospital, Acıbadem Healthcare Group, Clinic of Intensive Care, İstanbul, TURKEY/TÜRKİYE
E-mail: orkhan.orkhan@yandex.com

Peer review under responsibility of Turkiye Klinikleri Cardiovascular Sciences.

Received: 08 Mar 2020 ***Received in revised form:*** 13 May 2020 ***Accepted:*** 15 May 2020 ***Available online:*** 17 May 2020

2146-9032 / Copyright © 2020 by Türkiye Klinikleri. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Although electrocardiogram (ECG) changes are of great diagnostic value and have been used to predict the outcome in various cardiac conditions and different non-cardiac diseases with cardiac involvement, there have been only few reports evaluating the prognostic value of these parameters in critically ill patients.[1-3] In a cohort of nearly 10,000 critically ill patients in Irish population, an abnormal ECG was a powerful predictor of in-hospital mortality. Only 4 of 4,177 acutely ill patients with a normal ECG died within 24 hours. The other 55 patients who died within 24 hours had ECG abnormalities during admission.[4] In a study of centenarian population Romhilt-Estes left ventricular hypertrophy (LVH) criteria scores ≥5 points, low ECG QRS voltages (Sokolow-Lyon voltage <1.45 mV), and wide QRS complexes of ≥90 ms were predictive of 360 day mortality.[5] Similarly in a recent study performed in large population of 1486 critically ill intensive care unit patients, low QRS voltage (a combined lead I+II) was a major predictor of in-hospital mortality.[6] Besides critically ill patients, low QRS voltage was also correlated with increased mortality during one year follow-up in patients with systolic heart failure and patients waiting for cardiac transplantation.[7] Decrease in QRS amplitude is related with decrease in viable myocardium and decrease in cardiac function.[6] Disease affecting conduction system such as bundle branch block, manifest as increase in QRS duration.[8] A study performed in septic shock patients found that there was a mean decrease in QRS amplitude and prolongation in QRS duration compared to the controls. Also, these changes were reversible and returned to normal in patients who recovered from septic shock.[9] Sepsis is very often associated with multiorgan dysfunction, cardiomyopathy being one of them and 10% of all fatalities has been attributed to intractable heart failure in a review.[10] There is not any study showing the change in heart rate (HR) just before death. Our aim is to see the pattern how QRS amplitude, QRS duration and HR changes in intensive care unit patients during the last 10 hours of life.

MATERIAL AND METHODS

The study was performed by retrospective analysis of data stored in the telemetry recording system connected to patients during their stay in intensive care unit. Thirty patients, irrespective of their diagnosis during admission and follow up, who died in intensive care unit during January 1/2018-August 30/2018 were included in the study. Just after exitus, patients' telemetry recordings were analyzed, ECG prints were obtained on hourly basis starting from exitus time to 10 hours retrospectively (Figure 1, 2). QRS voltage was measured in lead II from the lowest negative deflection (i.e. the Q wave) to the highest positive deflection (i.e.the R wave) regardless of the ECG baseline and noted in millivolt (mV) units. QRS duration was measured from lead II starting from the first deflection of QRS at isoelectric line to the last deflection at isoelectric line and calculated in millisecond (ms) units. Duration between consecutive R wave (RR duration) was also calculated in lead II in ms units and HR was calculated by formula 60000/RR duration in ms. Arterial blood pressure was also measured simultaneously.

All data were entered in IBM SPSS Statistics 23, analyzed using Fitting Marginal Model and shown in graphs as Spaghetti Plots and Box Plots through time points.

Ethical approval of the study was obtained from Mehmet Ali Aydinlar Acibadem University Ethics Committee (Reference no: ATADEK 2018-17/16) which conformed to the principles outlined in the declaration of Helsinki.[11]

RESULTS

Baseline characteristics of the patients are shown in Table 1. The patients included in the study had hypertension in the ratio of 63.3%, diabetes mellitus in the ratio of 16.7%, coronary artery disease in the ratio of 33.3%, chronic obstructive pulmonary disease (COPD) in the ratio of 16.7% and malignancy in the ratio of 6% (Table 1).

There was consistent increase in QRS duration and decrease in HR during the last 10 hours of life and the most prominent increase were seen in the last hours. QRS duration increased at rate of 5.43 ms per hour ($p<0.001$) and HR decreased at rate of 2.68/min each hour ($p<0.001$). QRS amplitude attenuated in almost all patients although the changes were more subtle (decreased by 0.23 mV per hour, $p=0.02$)

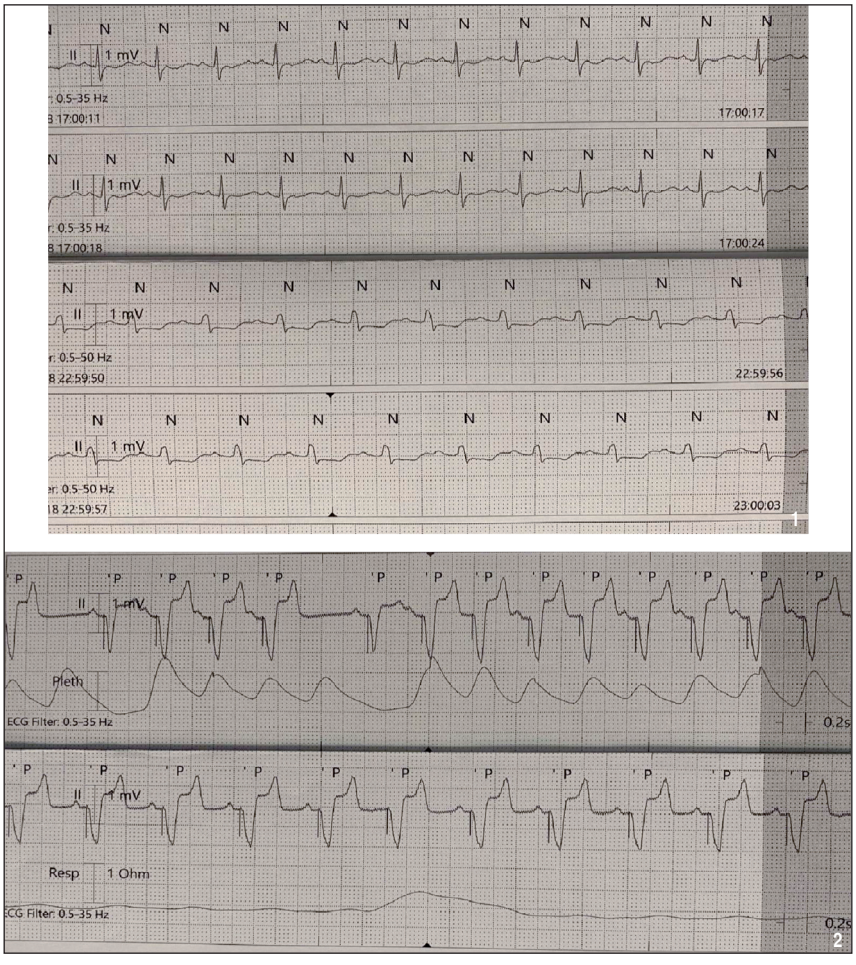


FIGURE 1, 2: ECG samples of two different patients during their last hours of life. In Figure 2 pacemaker ECG was presented to demonstrate changes in QRS voltage and QRS duration despite the same pacing site and same pacing voltage.

compared to QRS duration and HR. The findings of the study are shown by using spaghetti plots in Figure 3 and box plots through time points in Figure 4. The results are interpreted in Figure 5, Figure 6 and Figure 7.

DISCUSSION

In our study there was consistent decrease in QRS voltage, heart rate and increase in QRS duration during the last 10 hours of life. Although this study was conducted in small population of patients, the finding was consistent in all patients without exception.

The major cause of death in our patient population can be listed as follows: Pneumonia, malignancies, heart failure and cardiogenic shock, septic shock, cirrhosis, arrhythmia and intracranial bleeding.

TABLE 1: Baseline characteristics of the patients.

Age	73.8±13.2
Gender (women/men)	17/13
Hypertension	19 (63.3%)
Diabetes mellitus	5 (16.7%)
Coronary heart disease	10 (33.3%)
COPD	5 (16.7%)
Malignancy	1 (%6.0)

COPD: Chronic obstructive pulmonary disease.

Patients in septic shock had both decrease in QRS amplitude and increase in QRS duration. This effect was thought to be due to decrease in sodium current and loss of excitability of cardiac tissue secondary to sepsis, as has been shown in animal models. These ECG changes were reversible in patients who recovered.[9]

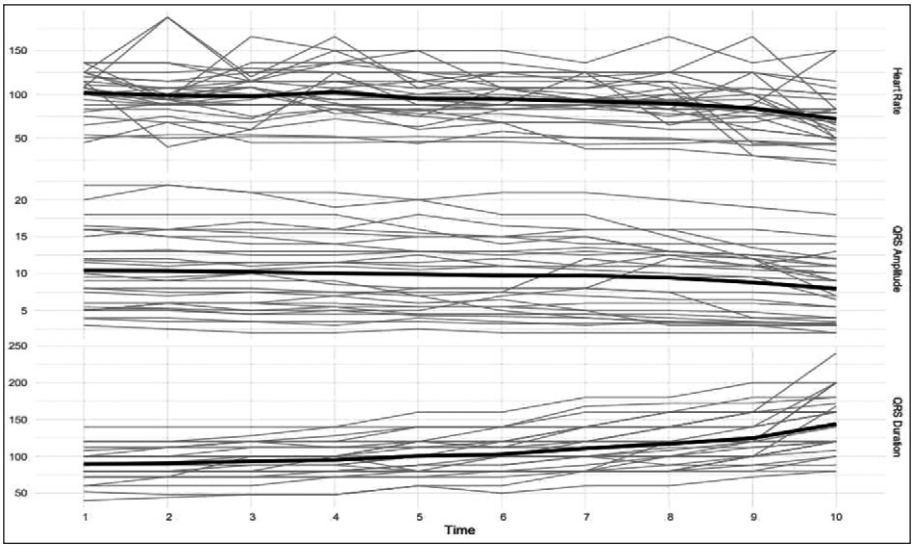


FIGURE 3: Gradual changes in QRS amplitude, QRS duration and heart rate during last 10 hours of their life shown by Spaghetti Plots.

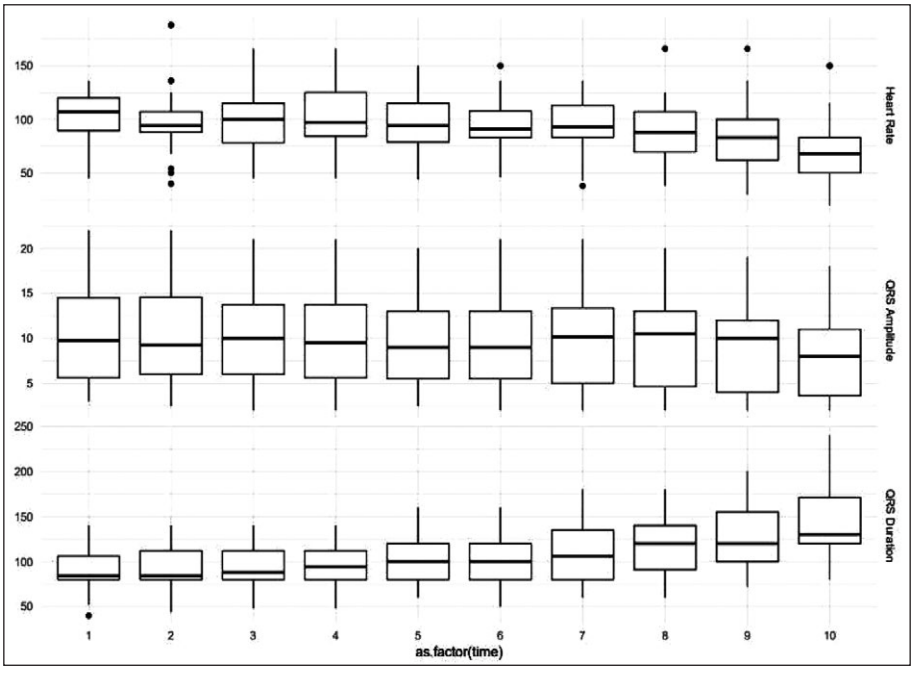


FIGURE 4: Gradual changes in QRS amplitude, QRS duration and heart rate during last 10 hours of their life shown Box Plots through time points.

In a large scale study of 12409 acute coronary syndrome patients, low QRS amplitude on admission ECG (<0.5 mV in all limb leads and <1.0 mV in all precordial leads) was an independent predictor of higher in-hospital mortality.[12] Similarly, an association between low QRS voltage and all cause mortality was found in a population of 6440 participants without any cardiovascular disease and mean age 60.[13]

A transient QRS attenuation has also been seen in takotsubo cardiomyopathy patients who returned to normal values once the cardiac function returned to normal and an increase in QRS amplitude specifically in aVR lead has been shown to be a good predictor of clinical improvement in decompensated heart failure.[14,15] In a study conducted in heart failure patients, a decrease in QRS amplitude was associated with adverse outcome and was a marker of disease severity.[6]

Fitting Marginal Models I - Heart Rate

$$HR_{ij} = \beta_0 + \beta_1 time_{ij} + Z_{ij}$$

Table 1: Coefficients Table for Heart Rate

	Value	Std.Error	t-value	p-value
(Intercept)	107.72	3.6	29.95	7.12e-92
time	-2.68	0.58	-4.63	5.45e-06

One unit increase in time is associated with a 2.68 decrease in the heart rate ($p < 0.001$).

FIGURE 5: Decrease in heart rate per hour.

Fitting Marginal Models II - QRS Amplitude

$$QRS_Amp_{ij} = \beta_0 + \beta_1 time_{ij} + Z_{ij}$$

Table 2: Coefficients Table for QRS Amplitude

	Value	Std.Error	t-value	p-value
(Intercept)	10.91	0.61	17.75	1.30e-48
time	-0.23	0.1	-2.33	2.06e-02

One unit increase in time is associated with a 0.23 decrease in the QRS Amplitude ($p = 0.02$).

FIGURE 6: Decrease in QRS amplitude per hour.

Fitting Marginal Models II - QRS Duration

$$QRS_Dur_{ij} = \beta_0 + \beta_1 time_{ij} + Z_{ij}$$

Table 3: Coefficients Table for QRS Duration

	Value	Std.Error	t-value	p-value
(Intercept)	76.92	3.58	21.48	1.79e-62
time	5.43	0.58	9.41	1.36e-18

One unit increase in time is associated with a 5.43 increase in QRS duration ($p < 0.001$).

FIGURE 7: Increase in QRS duration per hour.

In a prospective study of critically ill patients, low QRS voltage was also found in patients with anasarca type edema. A decrease in pacemaker spike has been noted during anasarca postulating that decrease in QRS sometimes might not be solely due to myocardial origin. It might be due to hypervolemic state and edema in the extremities where electrodes are placed.[16,17] Patients who died of cardiogenic shock and sepsis have increase in extracellular volume and decrease in myocardial function. Patients with liver failure, severe pulmonary infections and malignancies have low albumin levels and interstitial and extracellular edema. Both of these mechanisms may have contributed to the decrease in QRS voltage.

Prolongation of QRS duration has been associated with increased mortality in various studies. QRS duration, T wave inversion and QRS/T angle have been shown to be associated with sudden cardiac death and death from all causes in general population.[18] Delayed intrinsicoid deflection (DID) which results in prolongation of QRS duration has been associated with increased sudden cardiac arrest (SCA) independent of echocardiographic LVH, electrocardiographic LVH and reduced left ventricular ejection fraction (LVEF) in an ongoing prospective, population-based Oregon Sudden Unexpected Death Study (Oregon SUDS).[19] Increased QRS duration has been associated with poor in-hospital outcome and severe complications such as circulatory failure, ventricular tachycardia or fibrillation, ventilation requirement in Takotsubo cardiomyopathy and higher hospital mortality risk in fulminant myocarditis patients. Wide QRS implies mild or early stage progressive myocardial damage in myocarditis and takotsubo.[20,21] PR interval elongation and widened QRS complex has been shown to be independent risk factors for developing fulminant myocarditis in an adult patient with viral myocarditis.[22] In a patient population with corrected Tetralogy of Fallot (TOF), a longer QRS duration was associated with increased risk of developing malignant ventricular arrhythmia.[23] Intensive care patients in their last hours usually have haemodynamic collapse which results in myocardial depression, myocarditis, myocardial damage and impairment in normal conducting system of heart. All of these factors may have contributed to the widening of QRS as mentioned earlier.

Relative bradycardia was associated with lower 21 day mortality in critically ill patients with septic shock compared to never bradycardia (21% vs 34%) in a study.[24] But till date, there is not any study to reveal the correlation between bradycardia and higher

mortality in critically ill patients. Also, no study has been performed to show the changes in HR during the last hours of life. Our study shows that during last hours, along with the decrease in QRS amplitude and an increase in QRS duration, the HR decreases gradually before it comes to an ultimate stop.

The least prominent change was found in QRS amplitude. This could be explained by the change in position of leads during 10 hours. Usually the motorization leads were placed in the same anatomical landmarks. The change in lead position could change the amplitude of QRS but the duration and rate are not subject to change due to change in lead position.

Many measures of mortality have been proposed and are being used to predict the mortality of critically ill patients. QRS amplitude, QRS duration and HR can be easily calculated by automated machines and could be illustrated in graphics. We are collecting data from larger number of patients. If these findings will be consistent, then programs could be integrated in modern monitors to show the changes in QRS amplitude, QRS duration and HR in graphics. This could alert the physician that the mortality is high.

LIMITATIONS

This study was performed in a small number of patients and should be confirmed by large scale studies. Also, the changes in these parameters earlier than 10 hours before death has not been studied and whether it is consistent with our findings is not known. When the patients are in the last hours, they usually enter to an irreversible state and it is difficult to avoid mortality. The ECG changes might be very subtle and not apparent in the time period when there is still chance to reverse the clinical condition and prevent mortality. The changes earlier than last 10 hours should also be studied for this answer. Although we use standard position for the placement of telemetry leads, there might be subtle change in position during 10 hours. Although this subtle change in position has no effect in heart rate and QRS duration, amplitude might differ.

CONCLUSION

Calculation of QRS amplitude, QRS duration and HR from telemetry recordings is very practical. We found that during the last 10 hours of life the overall impact of the hemodynamic deterioration on cardiac electrical system is manifested as widening of QRS complex, attenuation of QRS voltage and decrease in HR. Automated softwares could be developed to present these findings in graphics and can be used as a prognosticator to recognize dying patient. This could have a profound influence on patients' and families' decision regarding end-of-life in irreversible illness such as terminal cancer patients. On the other hand, these finding could be a life saving alarm in certain acute reversible critical conditions such as fulminant myocarditis, anaphylactic shock, travma patients. Whether these findings will be different in those who respond to cardiopulmonary resuscitation and is a subject for further investigation.

Source of Finance

There has been no financial support for this work that could have influenced its outcome. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest

No conflicts of interest between the authors and/or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, shareholding and similar situations in any firm.

Authorship Contributions

Idea/Concept: Ashok Paudel; Design: Orkhan Mammadov; Control/Supervision: Ararso Kedir Jima, Nihan Yaman Mammadov; Data Collection and/or Processing: Orkhan Mammadov, Ararso Kedir Jima; Analysis and/or Interpretation: Ashok Paudel, Orkhan Mammadov; Literature Review: Nihan Yaman Mammadov, Erkan Ekicibasi; Writing the Article: Ashok Paudel, Orkhan Mammadov; Critical Review: Nihan Yaman Mammadov, Erkan Ekicibasi; References and Fundings: Ashok Paudel, Orkhan Mammadov; Materials: Ashok Paudel, Orkhan Mammadov, Ararso Kedir Jima.

REFERENCES

1. De Bacquer D, De Backer G, Kornitzer M, Blackburn H. Prognostic value of ECG findings for total, cardiovascular disease, and coronary heart disease death in men and women. Heart. 1998;80(6):570-7. [Crossref] [PubMed] [PMC]
2. Groot A, Bots ML, Rutten FH, den Ruijter HM, Numans ME, Vaartjes I. Measurement of ECG abnormalities and cardiovascular risk classification: a cohort study of primary care patients in the Netherlands. Br J Gen Pract. 2015;65(630):e1-8. [Crossref] [PubMed] [PMC]
3. Tan SY, Sungar GW, Myers J, Sandri M, Froelicher V. A simplified clinical electrocardiogram score for the prediction of cardiovascular mortality. Clin Cardiol. 2009;32(2):82-6. [Crossref] [PubMed] [PMC]
4. Kellett J, Deane B. The simple clinical score predicts mortality for 30 days after admission to an acute medical unit. QJM. 2006;99(11): 771-81. [Crossref] [PubMed]
5. Szewieczek J, Gąsior Z, Duława J, Francuz T, Legierska K, Batko-Szwaczka A, et al. ECG low QRS voltage and wide QRS complex predictive of centenarian 360-day mortality. Age (Dordr). 2016;38(2):44. [Crossref] [PubMed] [PMC]
6. Kellett J, Opio MO; Kitovu Hospital Study Group. QRS voltage is a predictor of in-hospital mortality of acutely ill medical patients. Clin Cardiol. 2018;41(8):1069-74. [Crossref] [PubMed] [PMC]
7. Kamath SA, de P Meo Neto J, Canham RM, Uddin F, Toto KH, Nelson LL, et al. Low voltage on the electrocardiogram is a marker of disease severity and a risk factor for adverse outcomes in patients with heart failure due to systolic dysfunction. Am Heart J. 2006;152(2): 355-61. [Crossref] [PubMed]
8. Demidova MM, Martín-Yebra A, Koul S, Engblom H, Martínez JP, Erlinge D, et al. QRS broadening due to terminal distortion is associated with the size of myocardial injury in experimental myocardial infarction. J Electrocardiol. 2016;49(3):300-6. [Crossref] [PubMed]
9. Rich MM, McGarvey ML, Teener JW, Frame LH. ECG changes during septic shock. Cardiology. 2002;97(4):187-96. [Crossref] [PubMed]
10. Müller-Werdan U, Prondzinsky R, Witthaut R, Stache N, Heinroth K, Kuhn C, et al. [The heart in infection and MODS (multiple organ dysfunction syndrome)]. Wien Klin Wochenschr. 1997;109 Suppl 1:3-24.
11. World Medical Association. World Medical Association Declaration of Helsinki: ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191-4. [Crossref] [PubMed]
12. Tan NS, Goodman SG, Yan RT, Tan MK, Fox KAA, Gore JM, et al; GRACE ECG substudy and Canadian ACS I Registry investigators. Prognostic significance of low QRS voltage on the admission electrocardiogram in acute coronary syndromes. Int J Cardiol. 2015;190:34-9. [Crossref] [PubMed]
13. Usoro AO, Bradford N, Shah AJ, Soliman EZ. Risk of mortality in individuals with low QRS voltage and free of cardiovascular disease. Am J Cardiol. 2014;113(9):1514-7. [Crossref] [PubMed]
14. Guerra F, Giannini I, Pongetti G, Fabbrizioli A, Rrapaj E, Aschieri D, et al. Transient QRS amplitude attenuation is associated with clinical recovery in patients with takotsubo cardiomyopathy. Int J Cardiol. 2015;187:198-205. [Crossref] [PubMed]
15. Durmus E, Hunuk B, Erdogan O. Increase in QRS amplitudes is better than N-terminal pro-B-type natriuretic peptide to predict clinical improvement in decompensated heart failure. J Electrocardiol. 2014;47(3):300-5. [Crossref] [PubMed]
16. Madias JE, Bazaz R, Agarwal H, Win M, Medepalli L. Anasarca-mediated attenuation of the amplitude of electrocardiogram complexes: a description of a heretofore unrecognized phenomenon. J Am Coll Cardiol. 2001;38(3):756-64. [Crossref]
17. Madias JE. Decrease/disappearance of pacemaker stimulus "spikes" due to anasarca: further proof that the mechanism of attenuation of ECG voltage with anasarca is extracardiac in origin. Ann Noninvasive Electrocardiol. 2004;9(3):243-51. [Crossref] [PubMed] [PMC]
18. Laukkanen JA, Di Angelantonio E, Khan H, Kurl S, Ronkainen K, Rautaharju P. T-wave inversion, QRS duration, and QRS/T angle as electrocardiographic predictors of the risk for sudden cardiac death. Am J Cardiol. 2014;113(7):1178-83. [Crossref] [PubMed]
19. Darouian N, Narayanan K, Aro AL, Reinier K, Uy-Evanado A, Teodorescu C, et al. Delayed intrinsicoid deflection of the QRS complex is associated with sudden cardiac arrest. Heart Rhythm. 2016;13(4):927-32. [Crossref] [PubMed] [PMC]
20. Sawamura A, Okumura T, Ito M, Ozaki Y, Ohte N, Amano T, et al; CHANGE PUMB Investigators. Prognostic value of electrocardiography in patients with fulminant myocarditis supported by percutaneous venoarterial extracorporeal membrane oxygenation-analysis from the CHANGE PUMP study. Circ J. 2018;82(8):2089-95. [Crossref] [PubMed]
21. Yamaguchi T, Yoshikawa T, Isogai T, Miyamoto T, Maekawa Y, Ueda T, et al. Predictive value of QRS duration at admission for in-hospital clinical outcome of Takotsubo cardiomyopathy. Circ J. 2016;81(1):62-8. [Crossref] [PubMed]
22. Sun LJ, Guo LJ, Cui M, Li Y, Zhou BD, Han JL, et al. [Related factors for the development of fulminant myocarditis in adults]. Zhonghua Xin Xue Guan Bing Za Zhi. 2017;45(12): 1039-43.
23. Russo G, Folino AF, Mazzotti E, Rebellato L, Daliento L. Comparison between QRS duration at standard ECG and signal-averaging ECG for arrhythmic risk stratification after surgical repair of tetralogy of fallot. J Cardiovasc Electrophysiol. 2005;16(3):288-92. [Crossref] [PubMed]
24. Beesley SJ, Wilson EL, Lanspa MJ, Grissom CK, Shahul S, Talmor D, et al. Relative bradycardia in patients with septic shock requiring vasopressor therapy. Crit Care Med. 2017;45(2):225-33. [Crossref] [PubMed] [PMC]