Alexandria Journal of Medicine (2013) **49**, 145–152

Alexandria University Faculty of Medicine

Alexandria Journal of Medicine

www.sciencedirect.com

ORIGINAL ARTICLE

Electrocardiogram as prognostic and diagnostic parameter in follow up of patients with heart failure

Mohamed Sadaka *, Alaa Aboelela, Sameh Arab, Mostafa Nawar

Faculty of Medicine, Cardiology, Cardiovascular Department, Egypt

Received 23 May 2012; accepted 10 September 2012
Available online 27 October 2012

KEYWORDS

Heart failure;
Electrocardiogram;
QRS voltage of chest leads;
QRS voltage of limb leads

Abstract *Introduction:* Most patients with HF due to systolic dysfunction have a significant abnormality on ECG. It is a widely available tool relatively inexpensive, simple to perform, and yields an instant result.

Aim: To determine whether heart failure (ECG) conveys prognostic and diagnostic information in patients with HF.

Methods: The study was carried out on 100 heart failure patients. All patients were subjected to history taking, clinical examination, standard 12 lead ECG (at admission, discharge, and 3 month follow up) for assessment of the following: (QRS voltage amplitude: in limb leads and in chest leads, total QRS voltage and transthoracic echocardiography were done for all patients (at discharge and at 3 month follow up (FU).

Results: There were 75 males and 25 females, aged between 21 and 86 years (mean = 59.14 ± 13.41 - years). Total QRS voltage, limb leads voltage and chest leads voltage; all significantly increased from admission values (98.54 ± 20.64, 37.87 ± 11.06, and 60.67 ± 15.26 mm) to (110.72 ± 21.28, 43.01 ± 11.92, and 67.71 ± 15.22) at discharge ($p < 0.001$ for all). Also there was a significant increase in voltage at FU (111.72 ± 21.28, 43.30 ± 11.96, and 68.61 ± 16.61 mm) ($p < 0.001$ for all). There was a negative correlation found between left ventricular end diastolic and systolic dimensions (LVEDD and LVESD) with total QRS voltage, chest leads voltage and limb leads voltage, both at discharge and at FU. The total QRS Voltage was significantly higher in patients having no lower limb (LL) edema than those with edema on admission, at discharge and on FU ($p < 0.001$).

Conclusion: Voltages of ECG are considered one of the most important parameters in diagnosis and FU of patients with heart failure. Voltage of chest leads well correlated with LVEDD and LVESD. Voltage of limb leads has important inverse correlation with LL edema.

* Corresponding author. Tel.: +20 01227498471.
E-mail address: mohsadaka2000@yahoo.com (M. Sadaka).

Peer review under responsibility of Alexandria University Faculty of Medicine.

ELSEVIER Production and hosting by Elsevier

http://dx.doi.org/10.1016/j.ajme.2012.09.002

1. Introduction

Heart failure (HF) is a common, costly, disabling, and potentially deadly condition. In developed countries, around 2% of adults suffer from HF, but in those over the age of 65, this increases to 6–10%.[1]

Patients with advanced HF represent almost 10% of the total HF population, have the highest short-term mortality, and consume the greatest percentage of resources. The direct costs are mainly attributed to hospitalizations (approximately 75%).[2,3]

The diagnosis of HF is dependent on a careful assessment of the symptoms and signs complemented by diagnostic testing, for example, chest X-ray, Electrocardiogram (ECG), Echocardiography, coronary angiography, and laboratory tests.[4]

ECG is a widely available tool that has a prognostic value in HF. It is relatively inexpensive, simple to perform, and yields an instant result. The measurement is objective and does not require specialized training to interpret.[5,6]

Most patients with HF due to systolic dysfunction have a significant abnormality on ECG. A normal ECG makes systolic dysfunction unlikely and is rare in patients with suspected HF and has an important role in guiding therapy.[6]

Low ECG voltage is a marker of the severity of HF and is a risk factor for adverse outcomes in patients with systolic HF at 1 year.[7]

The mechanism of the attenuation associated with peripheral edema (PERED) appears to be extracardiac in nature, and due to a short-circuiting effect on the electrical voltage generated at the epicardial surface, exerted by the passive volume conductor containing the heart; the short-circuiting effect in turn is due to the fluid-based reduction of the electrical impedance of the volume conductor (low resistivity of the excess fluid).[8–11]

Another hypothesis propose that QRS Voltage attenuation in patients with decompensated HF refer to a combination of heart-based influences, and the impact of the PERED, with different proportions of these two influences in different patients depending on the extent of changes in the heart (pressure, volume, and ischemia changes) and the passive body volume conductor (PERED)[10] (see Fig. 1).

This attenuation correlates well with weight gain from fluid overload, and this association can be put to work in the diagnosis and follow-up of such patients. Perusal of serial ECGs of patients with CHF and PERED indicates that the amplitude of QRS complexes are at its lowest when the patients show the highest weight and the most conspicuous PERED on physical examination. In contrast, the amplitude of QRS complexes is at its highest when the patients' weight is at its lowest, and the PERED has responded favorably to diuresis. This return to normalcy or improvement is also reproducibly experienced after hemodialysis[12,13] and may be seen in patients with HF and PERED who have end-stage renal failure and undergo this procedure.[14]

The attenuation of ECG voltage/PERED concept can be utilized in clinical practice and research, and can be employed either via a quantitative treatment of the 12-lead ECG, or by "eye-balling" the amplitude of leads I and II, at the patient's bedside, in the clinic or the office, summing the estimates of amplitude from these 2 leads and comparing serially such values with gain or loss of patient's weight or clinical evidence of PERED.[14]

ECG is relatively inexpensive, simple to perform and yields an instant result. The measurement is objective and does not require specialized training to interpret. The aim of this study was to determine whether heart failure (ECG) conveys prognostic and diagnostic information in patients with HF.

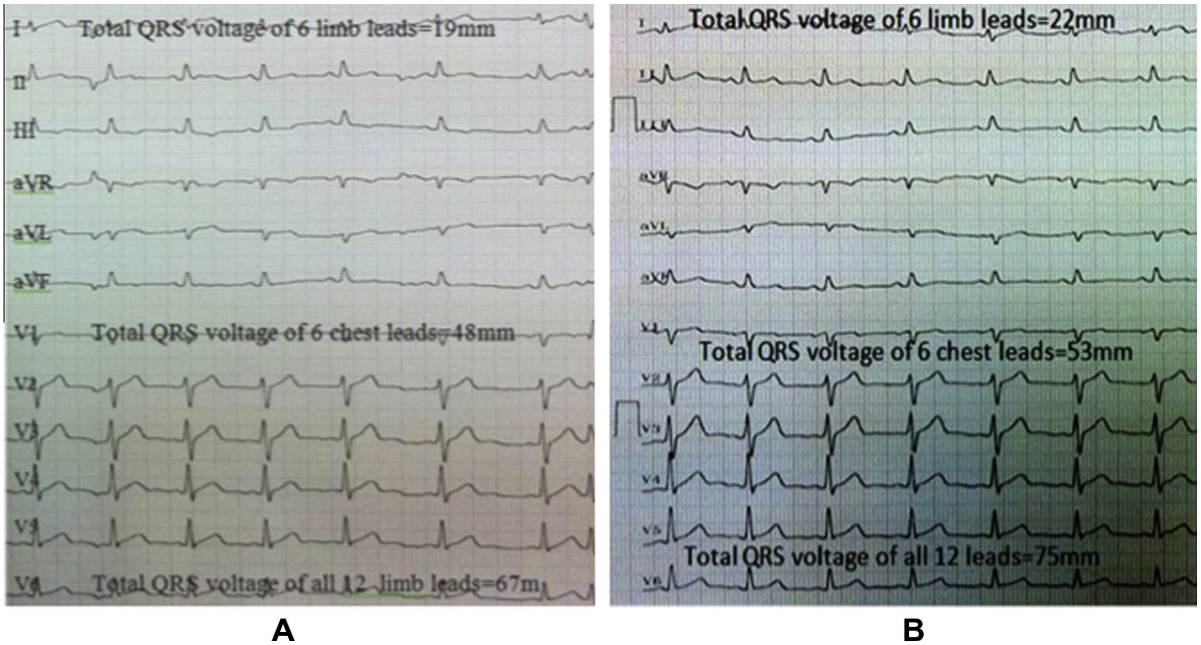

Figure 1 Case example: ECGs A (at admission) and B (at follow up). ECG was recorded at a speed of 25 mm/s. calibration of 10 mm = 1 mV. Measurements of peak-to-peak amplitudes of the QRS complexes were obtained. Sums of the 6 limb leads. Six precordial leads and all 12 ECG leads were manually calculated.

2. Methods

2.1. Study population

The study was carried out on 100 patients hospitalized in the department of cardiology, Alexandria Main University Hospital either with acute heart failure (AHF) or decompensated chronic heart failure (CHF), with the following Inclusion criteria: patient with AHF or decompensated CHF due to either ischemic heart disease or idiopathic dilated cardiomyopathy while the exclusion criteria were; patients with aortic stenosis and/or mitral stenosis, left ventricular outflow tract obstruction and atrial fibrillation (AF).

All patients were subjected to: thorough history taking, clinical examination and standard 12 lead ECG (at admission, discharge, and 3 month follow up) for assessment of the following:(QRS voltage amplitude: in limb leads and in chest leads, total QRS voltage was measured by summing the maximum deflection of the QRS complex above and below the base line.[15] Transthoracic Echocardiography was done for all patients (at discharge and at 3 months follow up) for evaluation of the following: wall motion abnormalities, systolic function: (fractional shortening and ejection fraction also, to measure ventricular dimensions).[16] All patients were followed up to 3 months as regards NYHA functional classification, rehospitalization, and death.

2.2. Statistical analysis

All clinical, ECG and echocardiographic data of all the patients were regularly recorded on standard forms and stored. Statistical analyses were performed with Microsoft Excel and SPSS software. Data were summarized using means and standard deviation (SD) for continuous variables, and frequency for categorical variables. Univariate analysis was performed either by student's *t*-test or Chi-square test, as appropriate. All *p*-values were significant if $\leqslant 0.05$

3. Results

The study included 100 patients, they were 75 males and 25 females, aged between 21 and 86 years (mean = 59.14 ± 13.41 - years). Nine patients (9%) had AHF, while (91%) had previously diagnosed CHF, with a mean duration of 3.48 ± 2.72 years (range = 3 months–11 years). The etiology of HF in all patients was either ischemic heart disease (IHD) in 50% of the cases, dilated cardiomyopathy (DCM) in 26%, hypertension (HTN) in 18%, or valvular heart disease (VHD) in 6%. Decompensation occurred due to acute coronary syndrome in 47%, chest infection in 14%, non-compliance to therapy in 11%, uncontrolled hypertension in 10%, arrhythmias in 9%, anemia in 5%, intake of negative inotropic drugs in 3%, or thyrotoxicosis in 1%. Seventy percent of the patients were hypertensive, 57% were diabetics, and 50% had dyslipidemia. Forty-four percent of the patients were smokers, 17% ex-smokers and 39% were non-smokers.

Patients were hospitalized and evaluated both clinically and by ECG. Treatment for each patient was individualized according to his condition. Before discharge and after a follow-up period of 3 months, all patients were subjected to ECG and echocardiogram. During the follow-up period, 34 patients were re-hospitalized, from which one case died at home.

On admission, 4 patients were in NYHA class II, 57 in class III, and 39 in class IV. Clinical improvement was encountered at discharge, where 32 patients were in NYHA class I, 56 in class II, and 12 in class III. At 3 months FU 37 patients were in NYHA class I, 44 in class II, 14 in class III, and 4 in class IV. According to Killip classification, 17 patients were in Killip class I, 70 in class II, 11 in class III and 2 in class IV.

3.1. Echocardiographic finding

The left ventricular end diastolic dimension (LVEDD), end systolic dimension (LVESD) and the ejection fraction (EF%) at discharge and on follow-up in all patients and the equivalent measurements for both groups {non rehospitalized group (NRG), rehospitalized group (RG)} are shown in Table 1.

There was a significant increase in LVEDD and LVESD with an insignificant increase in the EF when studying all patients. However in the NRG, there was no significant difference in the LVEDD and LVESD compared at discharge and at follow-up ($p = 0.06$, 0.051, respectively), but there is significant difference in EF compared at discharge and at follow-up ($p = 0.03$). As regards the RG, there was a significant increase in both LVEDD and LVESD and a significant decrease in EF% ($p < 0.001$ for all).

3.2. ECG findings (Table 2)

QRS voltage was measured by summing the maximum deflection of the QRS complex above and below the base line.[16] The mathematical summations of voltages in all limb leads and in all chest leads were calculated, as well as the summation of both values (Total QRS voltage). These parameters were calculated on admission, at discharge as well as at follow-up.

When studying all patients, the three parameters (Total QRS voltage, limb leads voltage and chest leads voltage) all significantly increased from admission values (98.54 ± 20.64, 37.87 ± 11.06, and 60.67 ± 15.26 mm) to (110.72 ± 21.28, 43.01 ± 11.92, and 67.71 ± 15.22) at discharge ($p < 0.001$ for all). Also there was a significant increase in voltage at follow-up (111.72 ± 21.28, 43.30 ± 11.96, and 68.61 ± 16.61 mm) ($p < 0.001$ for all).

Almost similar results were obtained when studying the 66 patients who required no re-hospitalization (NRG). The three parameters (Total QRS voltage, limb leads voltage and chest leads voltage) all significantly increased from admission values (105.58 ± 18.29, 39.29 ± 10.95 and 66.29 ± 13.16 mm) to (117.44 ± 19.66, 44.38 ± 11.89, and 73.06 ± 13.47 mm) at discharge ($p < 0.001$ for all) also there was a significant increase in voltage at follow-up (120.50 ± 19.99, 45.61 ± 11.70, and 75.15 ± 13.83 mm) ($p < 0.001$ for all).

Different results were obtained when studying the 34 patients who required further hospitalization (RG). The three parameters (Total QRS voltage, limb leads voltage and chest leads voltage) all significantly increased from admission values (84.88 ± 18.09, 35.12 ± 10.92 and 49.76 ± 13.10 mm) to (97.68 ± 18.20, 40.35 ± 11.70, and 57.32 ± 13.02 mm) at discharge ($p < 0.001$ for all), but there was a significant decrease

Table 1 Comparison between LVEDD, LVESD and EF at discharge and follow up.

All patients	LVEDD($n = 0$)		LVESD ($n = 100$)		EF ($n = 100$)	
	Discharge ($n = 100$)	3 months ($n = 99$)	Discharge ($n = 100$)	3 Months ($n = 99$)	Discharge ($n = 100$)	3 Months ($n = 99$)
Range	43.0–84.0	43.0–84.0	32.0–67.0	33.0–70.0	21.0–56.0	18.0–57.0
Mean ± SD	58.79 ± 8.10	60.21 ± 9.25	46.65 ± 8.88	49.76 ± 10.45	38.01 ± 7.32	37.63 ± 10.48
P	$<0.001^{*}$		$<0.001^{*}$		0.430	
NRG	LVEDD ($n = 66$)		LVESD ($n = 66$)		EF ($n = 66$)	
	Discharge	3 months	Discharge	3 months	Discharge	3 months
Range	43.0–84.0	43.0–84.0	32.0–67.0	33.0–70.0	21.0–56.0	20.0–57.0
Mean ± SD	56.24 ± 5.06	56.83 ± 8.46	44.55 ± 7.95	44.79 ± 8.83	39.98 ± 6.71	40.27 ± 7.73
P	0.060		0.051		$<0.03^{*}$	
RG	LVEDD ($n = 34$)		LVESD ($n = 34$)		EF ($n = 34$)	
	Discharge ($n = 34$)	3 months ($n = 33$)	Discharge ($n = 34$)	3 months ($n = 33$)	Discharge ($n = 34$)	3 months ($n = 33$)
Range	50.0–72.0	50.0–75.0	36.0–63.0	38.0–69.0	22.0–56.0	18.0–53.0
Mean ± SD	63.74 ± 5.56	66.97 ± 6.79	50.74 ± 9.27	57.70 ± 8.85	34.18 ± 7.0	28.33 ± 9.02
P	$<0.001^{*}$		$<0.001^{*}$		$<0.001^{*}$	

p: *p* Value for paired *t*-test.
* Statistically significant at $p \leqslant 0.05$.

in voltage at follow-up (94.15 ± 18.49, 38.70 ± 11.27, and 55.45 ± 13.74) ($p < 0.001$, 0.002, < 0.001), respectively.

3.3. Correlation between ECG voltage and Echo data

When studying all patients, a negative correlation was found between LVEDD and LVESD with the total QRS voltage, chest leads voltage and limb leads voltage, both at discharge and at follow-up. A similar finding (but positive correlation) was found with the EF% as shown in Table 3.

3.4. Lower limb edema (Table 4)

The Total QRS Voltage was significantly higher in patients having no lower limb (LL) edema than those with edema on admission, at discharge and on follow-up ($p < 0.001$). A similar significant finding existed when studying the Limb Leads Voltage ($p < 0.001$), but not with the Chest Leads Voltage when the difference was insignificant (p always > 0.4). These results were found in all patients and also in NRG. In RG, a significant correlation was only found with limb leads voltage.

4. Discussion

Electrocardiogram is a widely available tool that has a prognostic value in HF. It is relatively inexpensive, simple to perform, and yields an instant result. The measurement is objective and does not require specialized training to interpret.[4,5]

In our study, as regards the echocardiographic data, the left ventricular dimensions (both LVEDD and LVESD) were significantly higher among RG at discharge, with a significantly lower ejection fraction. At follow up, the latter showed a significant increase in LVEDD and LVESD with significant decline in ejection fraction ($p < 0.001$). In NRG, there were no significant changes in neither LVEDD nor LVESD between discharge and follow up, despite a significant increase in ejection fraction ($p < 0.03$).

In concordance with our study, Vasan et al.[17] revealed a low EF predict outcome in patients with CHF, they evaluated the echocardiograms of 73 patients with CHF (33 women, 40 men, and mean age 73 years) and 146 age- and gender-matched control subjects. Impaired LV systolic function was defined as an LV ejection fraction (LVEF) < 0.50. Thirty-seven CHF cases (51%) had a normal LVEF; 36 (49%) had a reduced LVEF. Women predominated in the former group (65%), whereas men constituted 75% of the latter group. During a median follow-up of 6.2 years, CHF cases with normal LVEF experienced an annual mortality of 8.7% versus 3.0% for matched control subjects. Congestive heart failure cases with reduced LVEF had an annual mortality of 18.9% versus 4.1% for matched control.

In our study, the ECG was studied, especially the QRS voltage. The QRS voltage was measured in all leads, and calculated the Chest Leads voltage (total voltage in the 6 chest leads), the Limb Leads voltage (total voltage in the 6 limb leads), and the Total voltage (mathematical summation of voltage in the 12 leads).

Table 2 Comparison between limb leads voltage, chest leads voltage, and total QRS voltage at admission, discharge, and follow up.

All patients (n100)	Limb leads voltage			Chest leads voltage			Total QRS voltage		
	Admission	Discharge	3 months	Admission	Discharge	3 months	Admission	Discharge	3 months
Range	14– 71	16– 84	16–81	26–94	35–102	34–109	45–156	52–169	54–172
Mean ± SD	37.87 ± 11.06	43.01 ± 11.92	43.30 ± 11.96	60.67 ± 15.26	67.71 ± 15.22	68.61 ± 16.61	98.54 ± 20.64	110.72 ± 21.28	111.72 ± 21.28
$p1$		$^{w}p < 0.001^{*}$	$^{w}p < 0.001^{*}$		$p < 0.001^{*}$	$p < 0.001^{*}$		$Wp < 0.001^{*}$	$^{w}p < 0.001^{*}$
$p2$			$^{w}p = 0.429$			$p = 0.022^{*}$			$^{w}p = 0.065$
NRG (n = 66)	Limb leads voltage			Chest leads voltage			Total QRS voltage		
	Admission	Discharge	3 months	Admission	Discharge	3 months	Admission	Discharge	3 months
Range	19–71	27–84	26–81	37–94	40–102	43–109	65–156	76–169	79–172
Mean ± SD	39.29 ± 10.95	44.38 ± 11.89	45.61 ± 11.70	66.29 ± 13.16	73.06 ± 13.47	75.15 ± 13.83	105.58 ± 18.29	117.44 ± 19.66	120.50 ± 19.99
$p1$		$^{w}p < 0.001^{*}$	$^{w}p < 0.001^{*}$		$p < 0.001^{*}$	$p < 0.001^{*}$		$< 0.001^{*}$	$< 0.001^{*}$
$p2$			$^{w}p < 0.001^{*}$			$p < 0.001^{*}$			$< 0.001^{*}$
RG (=34)	Limb leads voltage			Chest leads voltage			Total QRS voltage		
	Admission	Discharge	3 Months	Admission	Discharge	3 Months	Admission	Discharge	3 Months
Range	14–61	16–66	16–61	26–80	35–88	34–85	45–132	52–145	54–135
Mean ± SD	35.12 ± 10.92	40.35 ± 11.70	38.70 ± 11.27	49.76 ± 13.10	57.32 ± 13.02	55.45 ± 13.74	84.88 ± 18.09	97.68 ± 18.20	94.15 ± 18.4
$p1$		$^{w}p < 0.001^{*}$	$^{w}p < 0.001^{*}$		$p < 0.001^{*}$	$p < 0.001^{*}$		$< 0.001^{*}$	$< 0.001^{*}$
$p2$			$^{w}p < 0.001^{*}$			$p\ 0.002^{*}$			$< 0.001^{*}$

p: p value for Paired t-test.
^{w}p: p value for Wilcoxon signed ranks test.
$p1$: p value between admission and each other periods.
$p2$: p value between discharge and 3 months.
* Statistically significant at $p \leqslant 0.05$.

In the NRG, there was a significant increase in voltage from the admission level to that at discharge, and this remained to increase at follow-up. This applied to the total QRS voltage; limb leads voltage, and also chest leads voltage.

On the other hand, in RG, though there was a significant increase in voltage for the three voltage parameters from admission to discharge, there was a significant decline at follow-up. However, the voltage at follow-up was still significantly higher than the admission levels. This applied to all three parameters.

When studying all patients, a significant increase in voltage was noted between the admission and discharge ECG, which was maintained at follow-up. This applied to the three voltage parameters studied. However, when comparing the change in voltage from the value at discharge with its equivalent at follow-up, the only significant increase was that obtained from the summation of voltage in all chest leads, while the change in total voltage and limb leads voltages was insignificant. This could be explained by the opposed changes in NRG and RG.

Russell et al.[18] measured and summed the QRS voltage in all 12 leads of the ECG ($\sum$QRS) in two cohorts. The first included 415 patients with low left ventricular ejection fraction, who were followed-up in a HF clinic. The second cohort included 100 patients with advanced HF who had an ECG within 1 year preceding cardiac transplantation. They defined Low voltage as the lowest quartile of the clinic cohort ($\sum$QRS < 12 mV), and its prevalence was compared in the two cohorts. The associations of low voltage with 1-year outcomes were assessed in the clinic cohort.

In the clinic cohort, they found that the frequency of low voltage was higher in patients with NYHA class IV versus those with class I–III (34% versus 22% respectively, $p = 0.04$). The frequency of low voltage in the pre-transplant cohort (47%) was twice that of the clinic cohort (24%, $p < 0.001$). After 1 year of follow-up in the clinic cohort, low ECG voltage was associated with a higher rate of death (14% versus 5%, $p = 0.008$), and also with the composite end point of death or HF hospitalization (35% versus 20%, $p = 0.004$).

Russell et al.[18] concluded that the low ECG voltage is a marker of the severity of HF, and is a risk factor for adverse outcomes in patients with systolic HF at 1 year.

In our study, the total QRS voltage and the limb leads voltage were correlated and significantly higher in patients having no lower limb edema than those with edema on admission, at discharge and on follow-up. Such difference was insignificant when studying the chest leads voltage. These findings were found in all patients as well as the NRG. In RG, significant correlation was only found with limb leads voltage. A significant negative correlation existed between lower limb edema and voltage in limb leads, whereas it was insignificant as regards chest leads voltage.

In agreement with our results, Lumlertgul et al.[19] studied 20 patients with CHF by recording the ECG and weight on admission and at discharge. The amplitude of the QRS complexes in all ECG leads was measured and sums of leads I and II, the six chest leads, the six limb leads, and all 12 leads were calculated. There was a good correlation between the weight loss and the increase in the sums of the amplitudes of the QRS complexes from leads I and II, and the six limb leads, but a poor correlation with the V1–V6 leads and all 12 leads. They concluded that the sums of the amplitudes of the QRS complexes from leads I and II constitute a reliable, easily obtainable, ubiquitously available, bedside clinical index, which can be employed in the diagnosis, monitoring of management, and follow-up of patients with CHF.

Negative significant correlation was found between LVEDD and LVESD with both voltages in chest leads and total QRS voltage, while this correlation was not significant as regards voltage in limb leads, these results included the whole patients as well as both the groups.

Talbot et al.[20] studied 59 cases, 41 men and 18 women. Left ventricular volumes were measured by single plane ventriculography and coronary arteriography. The relation of the left ventricular end-diastolic volumes to the QRS voltage of the 12-lead electrocardiograms was examined.

Talbot et al.[20] found that QRS voltage was inversely correlated with volume for group 1 and for group 2; both correlations were the same ($r = 0$–68). There was no significant correlation for the group of patients with left ventricular hypertrophy. This could be ascribed to the opposite effect of hypertrophy on QRS voltage.

In our study the total QRS voltage was significantly lower in patients with lower limb edema than those without lower limb edema on admission, at discharge, and on follow-up ($p < 0.001$). A similar significant finding existed when studying the limb leads voltage ($p < 0.001$), but not with the chest leads voltage when the difference was insignificant (p always > 0.4). These results were found in all patients and also in NRG. In RG, significant correlation was only found with limb leads voltage.

Table 3 Correlation between voltage in limb leads, voltage in chest leads and total QRS voltage with LVEDD, LVESD and EF% in whole sample ($n = 100$).

		Voltage in Limb leads[a]		Voltage in Chest leads		Total QRS voltage	
		Discharge	3 months	Discharge	3 months	Discharge	3 months
LVEDD	*R*	−0.147	−0.202	−0.582*	−0.607*	−0.536*	−0.539*
	P	0.061	0.056	< 0.001	< 0.001	< 0.001	< 0.001
LVESD	*R*	−0.187	−0.178	−0.524*	−0.619*	−0.488*	−0.555*
	P	0.062	0.068	< 0.001	< 0.001	< 0.001	< 0.001
EF%	*R*	0.219*	0.256*	0.491*	0.619*	0.458*	0.563*
	P	0.028	0.010	< 0.001	< 0.001	< 0.001	< 0.001

r: Pearson coefficient.

* Statistically significant at $p \leqslant 0.05$.

[a] Logarithmic normalization of limb leads was used.

Table 4 Relation between LL edema with limb leads voltage, chest leads voltage, and total QRS voltage at each different period in the whole sample ($n = 100$).

	LL edema		Test of significance
	No	Yes	
Limb leads at admission			
Range	33.0–71.0	14.0–45.0	$^{MW}p < 0.001^{*}$
Mean ± SD	46.97 ± 9.78	32.05 ± 7.28	
Limb leads at discharge			
Range	38.0–84.0	16.0–54.0	$^{MW}p < 0.001^{*}$
Mean ± SD	52.08 ± 11.32	37.21 ± 8.12	
Limb leads at 3 months			
Range	39.0–81.0	16.0–54.0	$^{MW}p < 0.001^{*}$
Mean ± SD	52.33 ± 10.65	37.43 ± 8.69	
Chest leads at admission			
Range	30.0–90.0	26.0–94.0	$p = 0.567$
Mean ± SD	61.77 ± 15.99	59.97 ± 14.87	
Chest leads at discharge			
Range	37.0–102.0	35.0–101.0	$p = 0.599$
Mean ± SD	72.72 ± 16.06	67.07 ± 14.76	
Chest leads at 3 months			
Range	34.0–106.0	34.0–109.0	$p = 0.488$
Mean ± SD	70.05 ± 17.05	67.67 ± 16.39	
Total QRS voltage at admission			
Range	75.0–156.0	45.0–129.0	$p < 0.001^{*}$
Mean ± SD	108.74 ± 20.18	92.02 ± 18.27	
Total QRS voltage at discharge			
Range	87.0–169.0	52.0–140.0	$p < 0.001^{*}$
Mean ± SD	120.79 ± 21.16	104.28 ± 18.85	
Total QRS voltage at 3 months			
Range	86.0–172.0	54.0–149.0	$p < 0.001^{*}$
Mean ± SD	121.90 ± 22.71	105.10 ± 20.96	

p: p value for Mann Whitney test.
p: p value for Student's t-test.
* Statistically significant at $p \leqslant 0.05$.

Madias et al.[21] concluded that the resting electrocardiogram furnishes essential information for the diagnosis, management, and prognostic evaluation of patients with congestive heart failure (CHF). Almost any ECG diagnostic entity may turn out to be useful in the care of patients with CHF, revealing the nonspecificity of the ECG in CHF. Nevertheless, a number of CHF/ECG correlates have been proposed and found to be indispensable in clinical practice; they include, among others, the ECG diagnoses of myocardial ischemia and infarction, atrial fibrillation, left ventricular hypertrophy/dilatation, left bundle branch block and interventricular conduction delays, left atrial abnormality, and QT-interval prolongation. In addition to the above well-known applications of the ECG for patients with CHF, a recently described association of peripheral edema (PERED), sometimes even imperceptible by physical examination, with attenuated ECG potentials, could extend further the diagnostic range of the clinician. These ECG voltage attenuations are of extracardiac mechanism, and impact the amplitude of QRS complexes, P-waves, and T-waves, occasionally resulting also in shortening of the QRS complex and QT interval duration. PERED alleviation, in response to therapy of CHF, reverses all the above alterations. These fresh diagnostic insights have potential application in the follow-up of patients with CHF, and in their selection for implantation of cardioverter/defibrillator and/or cardiac resynchronization systems. If sought, PERED-induced ECG changes are abundantly present in the hospital and clinic environments; if their detection and monitoring are incorporated in the clinician's ''routine'', considerable improvements in the care of patients with CHF may be realized.

4.1. Conclusions

- Voltages of electrocardiogram are considered one of the most important parameters in diagnosis and follow up of patients with acute or decompensated heart failure.
- Voltage of chest leads correlates well with left ventricular end systolic and diastolic dimension.
- Voltage of limb leads has important inverse correlation with lower limb edema.
- Voltage of electrocardiogram improves with improvement of heart failure and is decreased in case of decompensation. Hence could be used in the follow up of these cases.
- ECG is a widely available tool that has a prognostic value in HF. It is an inexpensive tool, simple to perform, and yields an instant result. We recommend that ECG should be done not only during hospital course but also during follow up of patients with acute or decompensated HF.

- Considerable improvements in the care of patients with acute or decompensated heart failure may be realized if ECG monitoring and follow up are incorporated in clinician's routine work up.
- Further studies with longer follow up and larger sample of patients are needed to confirm our results.

References

1. Dickstein K, Cohen-Solal A, Filippatos G, McMurray JJ, Ponikowski P, Poole-Wilson PA, et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2008: the Task Force for the diagnosis and treatment of acute and chronic heart failure 2008 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association of the ESC (HFA) and endorsed by the European Society of Intensive Care Medicine (ESICM). *Eur Heart J* 2008;**29**(19):2388–442.
2. Hunt SA. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (Writing Committee to update the 2001 Guidelines for the Evaluation and Management of Heart Failure). *J Am Coll Cardiol* 2005;**46**(6):e1–e82.
3. O'Connell JB, Bristow MR. Economic impact of heart failure in the United States: time for a different approach. *J Heart Lung Transplant* 1994;**13**(4):S107–12.
4. Heart Failure Society of America. Evaluation and management of patients with acute decompensated heart failure. *J Card Fail* 2006;12(1):e86–103. Review. PubMed PMID: 16500576.
5. Senen K, Turhan H, Riza Erbay A, Basar N, Saatci Yasar A, Sahin O, et al. P-wave duration and P-wave dispersion in patients with dilated cardiomyopathy. *Eur J Heart Fail* 2004;**6**(5):567–9.
6. Khan NK, Goode KM, Cleland JG, Rigby AS, Freemantle N, Eastaugh J, et al. Prevalence of ECG abnormalities in an international survey of patients with suspected or confirmed heart failure at death or discharge. *Eur J Heart Fail* 2007;**9**(5):491–501.
7. Kamath SA, Meo Neto Jde P, 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.
8. Madias JE, Agarwal H, Win M, Medepalli L. Effect of weight loss in congestive heart failure from idiopathic dilated cardiomyopathy on electrocardiographic QRS voltage. *Am J Cardiol* 2002;**89**(1):86–8.
9. Madias JE, Song J, White CM, Kalus JS, Kluger J. Response of the ECG to short-term diuresis in patients with heart failure. *Ann Noninvasive Electrocardiol* 2005;**10**(3):288–96.
10. Madias JE. Standard electrocardiographic and signal-averaged electrocardiographic changes in congestive heart failure. *Congest Heart Fail* 2005;**11**(5):266–71.
11. Madias JE. On the mechanism of augmentation of electrocardiogram QRS complexes in patients with congestive heart failure responding to diuresis. *J Electrocardiol* 2005;**38**(1):54–7.
12. Madias JE. P waves in patients with changing edematous states: implications on interpreting repeat P wave measurements in patients developing anasarca or undergoing hemodialysis. *Pacing Clin Electrophysiol* 2004;**27**(6pt):156–749.
13. Madias JE, Narayan V. Augmentation of the amplitude of electrocardiographic QRS complexes immediately after hemodialysis: a study of 26 hemodialysis sessions of a single patient, aided by measurements of resistance, reactance, and impedance. *J Electrocardiol* 2003;**36**(3):263–71.
14. Madias JE. QTc interval in patients with changing edematous states: implications on interpreting repeat QTc interval measurements in patients with anasarca of varying etiology and those undergoing hemodialysis. *Pacing Clin Electrophysiol* 2005;**28**(1):54–61.
15. Madias JE. QRS Voltage Changes in Heart Failure: A 3-Compartment Mechanistic Model and its implications. *Indian Pacing Electrophysiol J* 2010;**10**(10):464–73.
16. Bounhoure JP, Massabuau P, Galinier M, Jordan C, Laurent JP, Marco J. Heart failure with preserved left ventricular function: clinical, echocardiographic, and clinical course features. Prognostic factors. *Bull Acad Natl Med* 2002;**186**(6):1003–13.
17. Vasan RS, Larson MG, Benjamin EJ, Evans JC, Reiss CK, Levy D. Congestive heart failure in subjects with normal versus reduced left ventricular ejection fraction: prevalence and mortality in a population-based cohort. *J Am Coll Cardiol* 1999;**33**(7):1948–55.
18. Russell M, Sandeep A, Januario de P, Uddin F, Kathleen H. 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.
19. Lumlertgul S, Chenthanakij B, Madias JE. ECG leads I and II to evaluate diuresis of patients with congestive heart failure admitted to the hospital via the emergency department. *Pacing Clin Electrophysiol* 2009;**32**(1):64–71.
20. Talbot S, Kilpatrick D, Jonathan A, Raphael MJ. QRS voltage of the electrocardiogram and Frank vectorcardiogram in relation to ventricular volume. *Br Heart J* 1977;**39**(10):1109–13.
21. Madias JE. The resting electrocardiogram in the management of patients with congestive heart failure: established applications and new insights. *Pacing Clin Electrophysiol* 2007;**30**(1):123–8.