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ECG changes following cardioversion and defibrillation

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Summary

Principles: changes of the QRS amplitude following defibrillation or cardioversion have never been reported in humans.

Methods: prospective analysis of patients externally cardioverted or defibrillated for ventricular and supraventricular tachyarrhythmias. Patients with coronary artery disease (CAD) and acute coronary syndrome (ACS) formed group A and patients without CAD but with external cardioversion/defibrillation formed group B. Patients in the control group (group C) experienced a shock by an Internal Cardioverter Defibrillator (ICD). All patients underwent the same study protocol: serial ECG's were recorded and sums (∑) of the QRS amplitude created separately for the precordial and peripheral leads. ∑ were then compared with baseline values and changes indicated as percentage (%).

Results: We included a total of 45 patients in our study: 21 patients (47%) in group A, 11 patients (24%) in group B and 13 patients (29%) in group C. Median age was 66 years in group A, 55 in group B and 52 in group C. In group A mean change of the R amplitude was –35% in precordial and –16% in the peripheral leads. In group B mean change of the R amplitude was –16% in the precordial and –2% in the peripheral leads. The QRS amplitude changed –23% in the precordial leads in group A and –14% in group B. 13 patients with external defibrillation or cardioversion of group A + B and all patients of the control group (n = 13) showed no voltage changes. The most pronounced R and QRS attenuation was seen in patients with acute coronary syndrome, CAD and those in whom manual chest compressions had been necessary. Changes appeared after a mean period of 23 hours and returned to normal after a mean of 62 hours.

Conclusions: we report the phenomenon of reversible voltage loss after external defibrillation or cardioversion. A possible explanation for this phenomenon might be tissue oedema in the chest area after electrical and traumatic injury. An alternative reason might be myocardial stunning. The exact pathophysiological mechanism leading to reversible voltage attenuation remains unclear and needs further exploration in studies with a larger sample of patients.

Key words: defibrillation; cardioversion; voltage loss; reversible; edema; stunning

Introduction

The differential diagnosis of low QRS voltage on the surface electrocardiogram (ECG) is broad and a variety of diseases (from adrenal insufficiency to pericardial effusion) can cause it. Transient attenuation of QRS voltage is a phenomenon, which has been rarely reported, with peripheral oedema as the proposed cause [1, 2].

Electrocardiographic changes, such as ST segment elevation following delivery of electrical energy for defibrillation or cardioversion are well studied [3–10]. To our knowledge, changes of the QRS amplitude following cardioversion or defibrillation have never been reported in humans. Prompted by the observation of an unexplained reversible voltage loss in a patient (fig. 1), we prospectively evaluated changes of the QRS amplitude following cardioversion and defibrillation.

There are no conflicts of interest to disclose.

Patients and methods

Patients

Serial, standard 12-lead ECG were recorded in 32 patients who underwent external cardioversion or defibrillation for conversion of ventricular or supraventricular tachyarrhythmias. Thirteen patients who underwent Internal Cardioverter Defibrillator (ICD) testing after implantation served as controls were submitted to the same study protocol.

Three groups were created for analysis. Group A consists of patients with known coronary artery disease (CAD) including those with acute coronary syndrome (ACS). Group B consists of patients without CAD excluded by coronary angiography. Group C consists of patients who experienced a shock by an ICD.

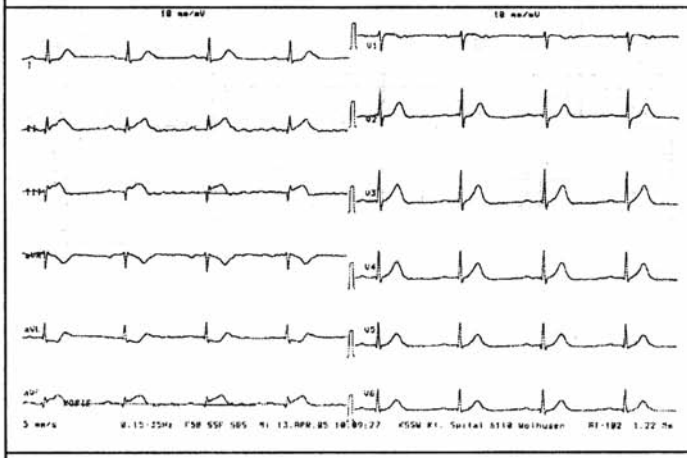
ECG recordings and interpretation

ECG's were recorded at baseline after cardioversion, then 6-hourly during the first 24 hours and thereafter every 24 hours as needed. The peripheral leads were placed on the extremities. Throughout the study the

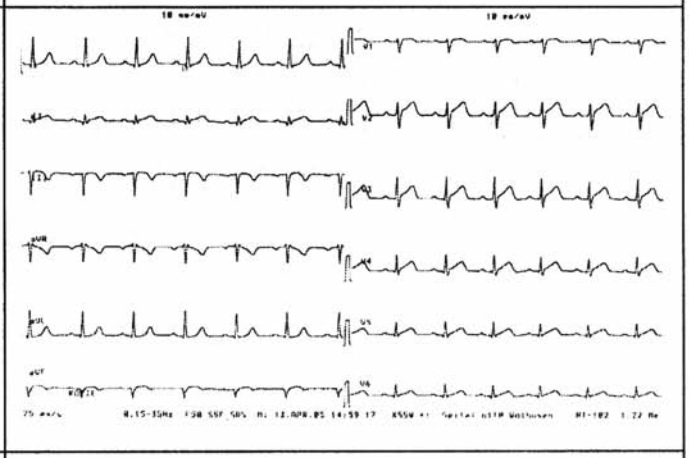
Figure 1

(a–e) ECG changes in a patient after defibrillation.

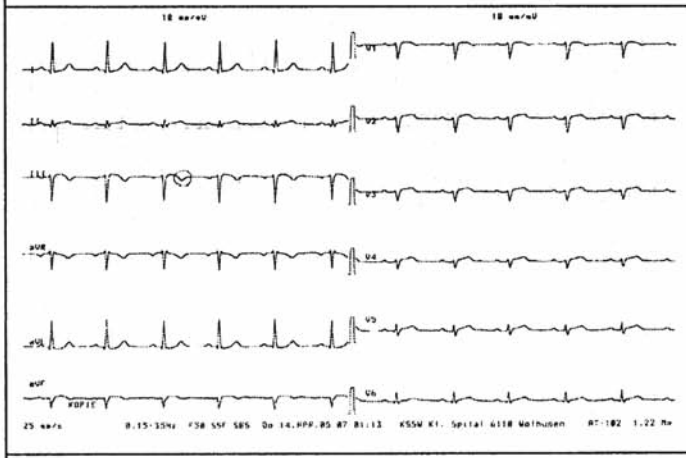
1a – Initial presentation – Acute inferior infarction – CPR with defibrillation



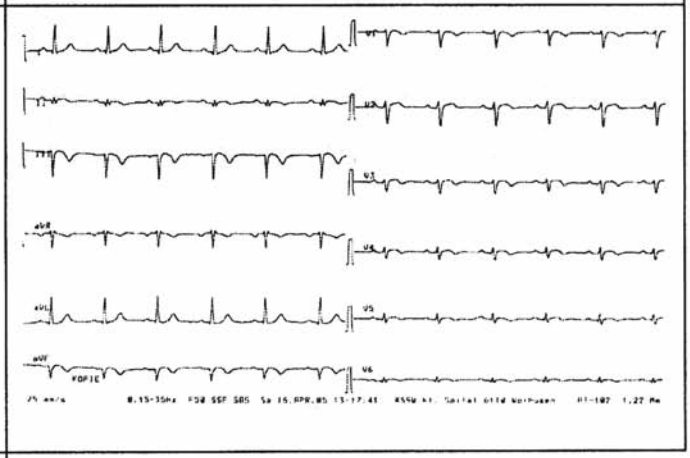
1b – After percutaneous coronary revascularisation Normalisation of the inferior ST-elevation



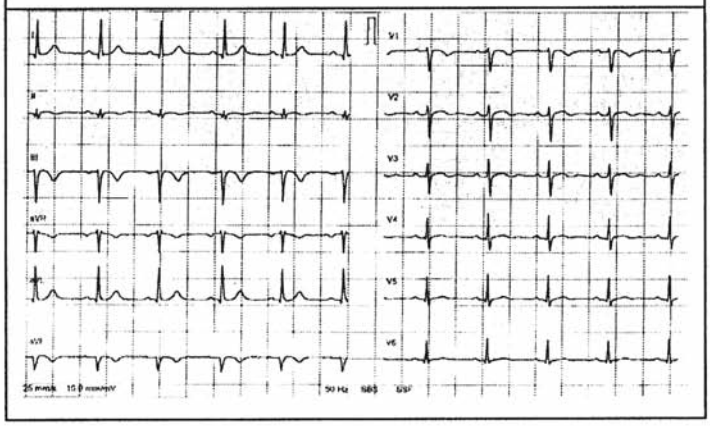
1c – Next day – low voltage in the anterior leads



1d – Persistence of the voltage loss 3 days after defibrillation



1e – Normalisation of the R waves 5 days after defibrillation



same ECG recorder (Schiller CS-100) was used. If no changes occurred within 72 hours of defibrillation/cardioversion, no further ECGs were recorded. If voltage loss occurred, then further ECG's were recorded every 24 hours until normalisation. The time points of maximal voltage loss and of voltage recovery were registered. Emphasis was laid on an unaltered placement of the electrodes during the serial ECG recordings. To this end the ECG electrodes were not removed after the first ECG and the same electrodes were used for serial recordings. The sum (Σ) of the R wave and QRS complex amplitudes were created separately for the precordial (V1–V6) and peripheral leads (I, II, III, aVF, aVL, aVR). The sums at baseline were then compared with the minimal sums reached in subsequent ECGs and loss of voltage was calculated as percentage (%).

The following information was obtained in all patients: Age, sex, body mass index, presence of an acute coronary syndrome (ACS), type of arrhythmia, amount of energy (joules) used for the conversion of the arrhythmia, and the need for chest compressions. In patients with ACS we also obtained the maximum value of Troponin T measured during hospitalisation.

Echocardiography to assess ejection fraction (EF), regional wall motion abnormalities and exclusion of pericardial effusion was performed in all patients.

Statistical analysis

Baseline characteristics of the patients are reported as median and interquartile range (IQR; bracketed values in table 1). Experienced voltage losses in % and time to appearance and normalisation are reported as mean values and standard deviation (SD). STATVIEW software from the SAS Institute (www.statview.com) was used for statistical analysis.

Table 1

Baseline characteristics of patients with external (Group A and B) and internal (Group C) defibrillation/cardioversion. Median values with IQR (in brackets).

	Group A (n = 21)	Group B (n = 11)	Group C (n = 13)
Age	66 (20)	55 (29)	52 (14)
Male	86%	73%	77%
BMI (kg/m^2)	25 (7)	26 (6)	25 (8)
Energy used (joules)	400 (200)	200 (175)	20 (0.5)
Chest compressions (%)	33	55	0
Ejection fraction (%)	40 (26)	55 (27)	33 (28)

Group A: Patients with Coronary Artery Disease (incl. ACS); Group B: Patients without CAD; Group C: Patients with an internal defibrillation by an ICD

Table 2

Mean voltage loss after defibrillation affecting the QRS and R amplitude in patients with CAD and ACS (group A) and patients without CAD (group B).

	QRS amplitude voltage loss (%)		R amplitude voltage loss (%)	
	Peripheral leads	Precordial leads	Peripheral leads	Precordial leads
Group A (n = 21)	–14	–23	–16	–35
Group B (n = 11)	–1	–14	–2	–16

Results

Baseline characteristics and results are presented in table 1. We evaluated a total of 32 patients with external cardioversion or defibrillation and 13 patients with shock applied by an ICD.

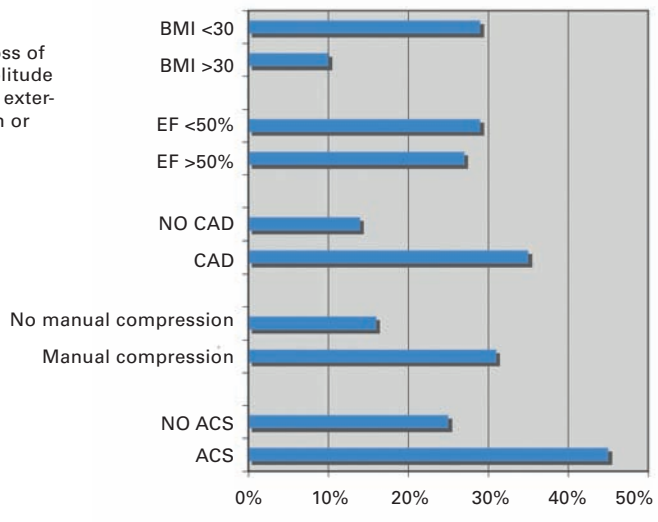


Figure 2

Mean voltage loss of the R wave amplitude in patients after external defibrillation or cardioversion.

External cardioversion and defibrillation (group A and B)

There were 21 patients in group A (with CAD and including 6 patients with ACS) and 11 patients in group B (patients without CAD but with external defibrillation/cardioversion). The median age of was 66 years in group A and 55 in group B.

6 out of 21 patients in Group A had ACS. In 13 patients (7 in group A and 6 in group B) manual chest compressions were applied for resuscitation. In group A 10 patients (48%) had ventricular fibrillation, 10 patients (48%) ventricular tachycardia and 1 patient (4%) torsade de pointes. In group B, 4 patients (37%) had ventricular fibrillation, 2 patients (18%) ventricular tachycardia, 3 patients (27%) atrial fibrillation and 2 patients (18%) torsade de pointes. The median amount of energy used for the conversion of the arrhythmias was higher in group A compared to group B (400 *vs* 200 joules). The interquartile range was 200 joules for group A and 175 joules for group B.

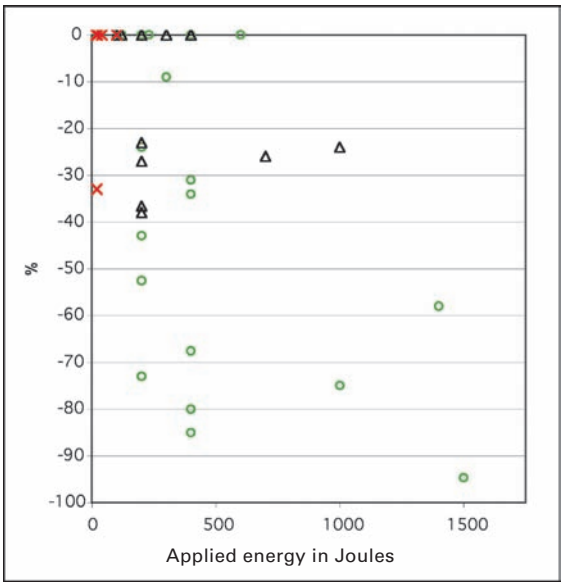


Figure 3

Voltage loss affecting the R wave amplitude in the precordial leads in relation to energy levels used for defibrillation or cardioversion (n = 45). Group A = green circles (n = 21); Group B = black triangles (n = 11); Group C = red crosses (n = 13).

The median ejection fraction was 40% (IQR 26) in group A and 55% (IQR 27) in group B. None of the patients had pericardial effusion.

The voltage loss affecting the R (ΣR) and QRS (ΣQRS) amplitudes is summarised in table 2. Group A reached higher voltage losses than group B (–35 *vs* –16% for the R amplitude in the precordial leads and –23 *vs* –14% for the QRS amplitude in the precordial leads). The maximum R voltage loss reached –95%. 13 patients showed no voltage changes. Attenuations of the R wave amplitudes are shown in comparison between different subgroups in figure 2. The attenuation of the R amplitude was higher in patients who experienced manual compressions for resuscitation (–31 *vs* –16%), those who had an ACS (45 *vs* 25%) and those with CAD (35 *vs* 14%).

Figure 3 shows the attenuation of the R wave in relation to the energy used to convert arrhythmias. There was a weak linear correlation (R^2 = 0.24) between energy levels used and attenuation of the R wave.

Minimum voltage values were reached after a mean of 23 hours for the QRS and the R wave (SD 8.3 *vs* 8.9 hours). The mean time to normalisation was 64 hours for the R wave and 62 hours for the QRS amplitude (SD 22 hours for both).

ST segment elevation was seen in all patients with ACS but not in the patients without ACS during follow-up ECG's. The mean Troponin T value in patients with ACS was 0.8 ug/l (SD 2.6).

ICD testing (group C)

The group with internal defibrillation involved 13 patients with a mean age of 54 years. 10 patients (77%) were males. Other characteristics of this group are shown in table 1. ICD testing did not lead to any amplitude changes in the analysed patients following ICD shock with a median of 20 joules.

Discussion

We report a phenomenon of voltage loss after external defibrillation or cardioversion, which appears after about 23 hours and is most prominent in the R waves of the precordial leads. The voltage loss was most conspicuous in patients who had required manual compressions for CPR and in patients with known CAD or ACS (table 2 and fig. 2). Additionally, higher defibrillator energy levels appeared to cause more prominent changes (fig. 3). In the cases evaluated in this study the voltage returned to previous levels after a mean time of 62 hours.

To our knowledge, the phenomenon of voltage loss after external defibrillation or cardioversion has never been reported. There are several possible explanations for this phenomenon. According to Ohm's law, voltage is inversely proportional to resistance, as is the case in pericardial effusion. Echocardiography excluded pericardial effusion in all of our patients. Chronic infarction is associated with low voltage in the corresponding leads, but in our cases low voltage occurred acutely and disappeared within days. Because we included patients with (n = 6) and without (n = 26) acute coronary syndromes, it is unlikely that acute ischaemia resulting from the shock played a major role. However, voltage losses were more striking in patients with ACS, a factor that may accentuate the phenomenon of voltage loss after external defibrillation or cardioversion.

Application of high electrical energy and additional physical trauma during CPR might cause chest wall oedema and thereby increase the resistance. This could explain both why precordial leads were predominantly affected and why pronounced R wave amplitude losses occurred in patients subjected to very high energy levels (fig. 3).

A further explanation for our observation of voltage loss after external defibrillation or cardioversion relates to defibrillation-associated post-resuscitation stunning which is common after prolonged cardiac arrest [11] . However, and despite the fact that we did not address the issue of stunning in our analysis, the phenomenon of voltage loss was also observed in patients without regional wall motion abnormalities, indicating a minimal or no role of stunning. The fact that patients with internal cardioversion by an ICD shock did not show any voltage change would indicate that the changes occurring are related to total electrical energy traversing the tissue to the myocardium.

It is also possible that a combination of different mechanisms (thoracic oedema plus stunning)

contribute to this phenomenon. The pathophysiological mechanisms underlying the phenomenon of voltage loss after external defibrillation/cardioversion need further exploration in studies with a larger sample of patients. The observation does indicate that an early assessment by echocardiography is helpful in exclusion of pericardial effusion and ongoing ischaemia. If stunning can be identified as a cause of the phenomenon then an appropriate drug therapy might be indicated to restore normal cardiac function more rapidly. If the cause cannot be identified then serial ECGs should be performed to document the reversibility of this probably benign phenomenon related to defibrillation or cardioversion.

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