

ECG Body Surface Mapping changes in type 1 Diabetic Patients with and without autonomic neuropathy

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Short title: ECG body surface mapping in diabetic patients

Summary: ECG body surface mapping (BSM) parameters in the 1st type diabetic patients (DM1) are significantly different comparing to healthy nondiabetic subjects. Hypothesis that these changes are more pronounced in DM1 patients with autonomic neuropathy (AN) was tested. The parameters of BSM were registered by diagnostic system Cardiac 112.2 in 54 DM1 patients including 25 with AN and 30 control subjects. AN was diagnosed according to Ewing criteria when two or more Ewing tests were abnormal. In classic 12-lead ECG the heart rate was increased, QRS and QT shortened ($p < 0.01$) and QT_c prolonged in DM 1 patients. The VCG measurement of QRS-STT angles and spatial QRS-STT angle showed nonsignificant differences. The absolute values of maximum and minimum in depolarization and repolarization isopotential, isointegral, isoarea maps were significantly different in DM 1 patients in comparison with controls ($p < 0.01$). The changes were more pronounced in DM 1 patients with AN than in DM patients without AN ($p < 0.05$). The QT duration measured in 82 leads of thorax was significantly shortened in 68 leads of both DM 1 groups of patients ($p < 0.01$) when compared with controls. In 34 of them this shortening was more pronounced in DM 1 AN patients than in DM 1 patients without AN ($p < 0.05$). The results showed that the method of ECG BSM is capable to confirm the presence of autonomic neuropathy in diabetic patients.

Key words: Diabetes mellitus type 1, Autonomic neuropathy, ECG Body Surface Mapping

Introduction

Diabetes mellitus can be complicated by autonomic neuropathy even in the young DM1 patients (Alberti *et al.* 1978, Alberti and Zimmet 1989, Beckman *et al.* 2002). The relatively increased sympathetic control in patients with autonomic neuropathy is associated with the higher risk of sudden cardiac death (Alberti *et al.* 1978, Alberti and Zimmet 1989). In these patients the heart rate is accelerated and cardiac response to the different stimuli is pathological as far as autonomic neuropathy produces some abnormalities in heart electrical field (Javorka *et al.* 2005, Ruttkay-Nedecky 2001). In our previous work we found the differences in some parameters of heart electrical field in DM1 patients in comparison with controls (Žďárská *et al.* 2007). In the present study we tested hypothesis that autonomic neuropathy is associated with more pronounced changes on body surface mapping in the DM1 patients. The preliminary results have been published in abstract form (Palová *et al.* 2007).

Methods

The 1st type diabetic patients (DM1) were recruited in diabetic outpatient department of Medical department of Faculty hospital Prague Motol. 54 diabetic patients (31 men, 23 women) and 30 controls (11 men, 19 women) participated in the study (Tab 1). In DM 1 20 men and in DM 1 with AN 11 men were present. The parameters of heart electric field were evaluated together for men and women.

Inclusion criteria: 1st type diabetes mellitus

Age: between 18–50 years

Twelve leads ECG: within normal limits

Exclusion criteria: Another drug treatment than insulin

Signs of retinopathy

Presence of microalbuminuria

Serum creatinin or urea elevation

Any 12 lead ECG abnormalities

Another disease than 1st type diabetes mellitus

In diabetic patients the duration of DM1 was recorded. DM1 was diagnosed by the increased fasting blood sugar, low C-peptide, and positive findings of glutamic acid decarboxylase 65 (anti-GAD65) or/and to autoantibodies to islet cells (anti-ICA). The long-term metabolic compensation of diabetic patients was evaluated by HbA1c level according to IFCC (Pickup and Williams 2003). The average total daily insulin dosage was also recorded.

Autonomic diabetic neuropathy (AN) was diagnosed according to Ewing tests examined and evaluated according to previously published protocols (Alberti *et al.* 1978, Alberti and Zimmet 1989). Cardiovascular tests have been developed which indicate the presence or absence of autonomic neuropathy.

The following 5 autonomic function tests have been performed. The patients with at least two abnormal tests were labelled to suffer from AN (Alberti *et al.* 1978, Alberti and Zimmet 1989).

Ewing autonomic function tests:

1. Heart rate variation of deep breathing (beats/min) – abnormal < 10
2. Heart rate increase on standing at 15 seconds (beats/min) – abnormal < 12
3. Heart rate increase on standing, 30:15 ratio – abnormal < 1.00
4. Valsalva ratio – abnormal < 1.2
5. Postural systolic pressure fall at 2 min (mm Hg) – abnormal > 30

The electrocardiogram (ECG), vectorcardiogram (VCG) and body surface isopotential, isointegral and isoarea maps (BSM) were registered altogether using the diagnostic system CARDIAG 112.2 (Slaviček *et al.* 2001, Kittnar and Stovicek 1993) one hour after a light breakfast and an insulin application in diabetic patients in the morning hours. Healthy control subjects were also examined one hour after breakfast. 201 parameters of heart electrical field were registered in controls, DM1 patients without and with AN.

Heart rate, duration of PQ, QRS, QT and QTc intervals were recorded and evaluated by 12 leads ECG.

In the VCG evaluation, the Frank orthogonal lead system was used (Frank 1956). The depolarization, repolarization isopotential maps (DIPM, RIMP), isointegral depolarization and repolarization maps (DIIM, RIIM) and their maximum and minimum were recorded and evaluated by ECG body surface potential maps (BSM). Depolarization isoarea maps, their maximum and minimum in μVs (isointegral maps from the beginning of QRS until 40 msec – DIAM max 40, DIAM min 40) and repolarization areas and their maximum and minimum in μVs (isointegral maps from the point J to 40 msec – RIAM max 40 and RIAM min 40) were examined as well as isointegral minimum (Q-IIM), amplitude (IPMAM-Q) of Q wave as and QT duration in the chest electrodes. Activation time (ICHVAT in ms) was measured between the beginning of depolarization in an orthogonal lead and the R wave in the individual chest leads. The QT duration was measured in 85 thoracic leads.

All diabetic patients and control subjects signed informed consent forms prior to their inclusion in the study. The local ethical committee accepted the study protocol with respect to the 1964 Declaration of Helsinki.

Statistical evaluation: The mean value, standard deviation (SD) and standard error of mean (S.E.M.) of the measured parameters were calculated. T-test and chi square test were used for evaluation of basal characteristics. Kruskal-Wallis test was used for comparison of 12 leads ECG, vectorcardiogram and BSM parameters of all three studied groups (diabetic patients with or without AN and healthy control subjects). The non-parametric Mann-Whitney test was used for separate comparison of 12 leads ECG, vectorcardiogram and BSM parameters of 2 diabetic groups. The p-value below 0.05 was considered to be significant.

Results

The number of controls and diabetic patients with or without AN as well as the parameters of the compensation and treatment of DM1 are summarized in Tab. 1. In these characteristics no significant changes were present among the groups. Out of twenty-five patients with diagnosis of AN, twenty-one presented a score of 2 and the remaining four patients a score of 3 in pathological Ewing tests.

The results of 12 leads ECG are shown in Tab. 2. Comparing only two groups of patients with DM1, the significant difference was detected for heart rate which was faster in patients with AN ($p < 0.05$) and R-R shortened ($p < 0.05$). The duration of QRS was significantly shortened in DM1 patients with AN patients than in controls ($p < 0.01$). The shortening of QRS was more pronounced in DM1 patients with AN ($p < 0.01$) than in DM1 patients without AN ($p < 0.05$) when compared with controls. The duration of QT was shortened in both groups of diabetic patients due to the increase of heart rate. The QT_C was prolonged in both groups of diabetic patients, but nonsignificantly probably due to the wide range of variation in DM 1 patients.

The planar QRS-STT angles in VCG changed by different way in both DM groups of patients (Tab. 3). The QRS axis in frontal plane was unchanged in comparison with controls, similarly as the QRS-STT angle in frontal plane.

The QRS-STT angle in transversal (horizontal) plane was more opened only in DM 1 patients without AN, while in left sagittal plane the angle was opened in both groups. The spatial angle was opened by more pronounced way in DM 1 without AN than DM 1 with AN. The differences were not statistically significant (Mann-Whitney), the result of Kruskal-Wallis test was significant for the value of left sagittal plane angle and spatial angle (Tab. 3).

The parameters of heart electric field in the diabetic patients (both without or with AN) and control subjects during BSM measurement are summarized in Tab. 4.

The maximum in depolarization of isopotential maps (DIPMmax) was more positive until the 30th ms and more negative from the 50th ms. The minimum (DIPMmin) was more negative until the 40th ms of the QRS. The repolarization isopotential map maximum

(RIPMmax) was less positive in the 10th ms and minimum (RIPMmin) less negative from 50th to 80th ms from the beginning of the point J (terminal part of the QRS). The changes in DIPM maps were confirmed by measurement of depolarization isointegral and isoarea maps maximum and minimum (DIIM, DIAM). The amplitude of Q and S wave were more negative (Q-IPMAMmin, QS-IPMAMmin) similarly as the minimum in the isointegral map of the Q wave (Q-IIMmin) in DM patients than in controls. The activation time (ICHVAT) was faster in DM1 patients due to the increased heart rate (Tab. 4).

In 12 parameters from 19 (DIPMmax50, DIPMmin20, DIAMmax40, RIPMmax10, RIPMmin 50, 60, 70, 80, Q-IPMAMmin, Q-IIMmin, QS-IPMAMmin, ICHVAT) the DM1 patients with AN showed the more pronounced changes ($p < 0.01$), than DM1 patients without AN ($p < 0.05$) in comparison with controls. On the other side changes of 4 parameters (DIPMmax10, 30, DIPMmin10, DIIMmin) were more pronounced in DM1 without AN than in DM1 patients with AN ($p < 0.05$, Tab.4). The values of 3 parameters (DIPMmin30,40, DIPMmin40) were nonsignificantly different in both groups of DM patients (Tab. 4.), but significantly different in comparison with controls ($p < 0.01$).

In 68 thoracic surface leads of out 82 measured the QT duration was significantly shortened in the both groups of DM1 patients when compared with controls. In 34 leads the QT shortening was more pronounced in DM1 patients with AN ($p < 0.01$) than in DM1 patients without AN ($p < 0.05$). In 34 other leads the shortening of QT duration was not different in the DM1 patients with and without AN ($p < 0.01$) in comparison with controls. In the resting 14 leads the significant shortening of the QT duration ($p < 0.01$) was observed only in DM1 patients with AN and not in DM 1 patients without AN (not shown in Tab.).

Discussion

The present work showed the differences in parameters of heart electric field in DM 1 patients in comparison with controls: acceleration of heart rate, shortening of RR, QRS and QT intervals. Depolarization and repolarization isopotential, isointegral, isoarea maps (the absolute values of maximum and minimum) were significantly changed. Fourteen parameters were different in comparison of controls and DM 1 patients without AN, and twenty parameters were significantly different in comparison of controls and DM 1 AN patients. The duration of QT measured in 80 surface thoracic places was shortened in 68 of them in DM 1 patients without AN, but in all 82 places in DM 1 patients with AN. Comparison of two DM 1 patient groups showed the more pronounced changes of some heart electric field parameters in DM 1 AN patients than in patients without AN (Tab. 4). The present work confirmed our previous results in DM 1 without AN (Žďárská *et al.* 2007).

The statistically significant changes in localization of depolarization isopotential map minimum in 40th and 50th msec (DIPMmin40,50) were found. The values of minimum were placed 2 intercostal spaces lower in DM 1 AN patients than in controls ($p < 0.05$).

Diabetes mellitus can be considered as a vascular disease because it causes both microvascular and macrovascular complications (Alberti *et al.* 1978, Alberti and Zimmet 1989, Beckman *et al.* 2002, Pickup and Williams 2003, Kannel *et al.* 1986). Cardiovascular disease accounts for at least 66% of deaths in diabetic patients. Due to the sensitive neuropathy, the coronary artery disease in diabetic patients is frequently asymptomatic (Alberti and Zimmet 1989, Langer *et al.* 1991, Paillole *et al.* 1995, Wackers *et al.* 2004). This fact underlines the importance of the sophisticated laboratory testing in diabetic patients, mainly stress myocardial SPECT and stress echocardiography, for diagnosis evaluation (Langer *et al.* 1991, Paillole *et al.* 1995, Wackers *et al.* 2004). Twelve leads resting ECG can be within normal limits even in an advanced stage of coronary artery disease (Alberti and Zimmet 1989, Pickup and Williams 2003). In this respect, BSM might be useful tool in coronary ischemia detection (Kittnar *et al.* 1993, Green *et al.* 1985, Valouch *et al.* 2004) in asymptomatic diabetic patients. However, the changes due to diabetes itself must be defined first before the specific abnormalities for silent coronary ischemia are looked for on BSM in diabetic patients.

In our present study therefore the young 1st type diabetic patients with normal resting 12 leads ECG and without any clinically evident macrovascular complications have been evaluated. These patients represent group of subjects with very low probability of asymptomatic coronary artery disease (Alberti *et al.* 1978, Alberti and Zimmet 1989, Pickup and Williams 2003).

The parameters of VCG, QRS-STT angles, the spatial angle and QRS axis in frontal plane were nonsignificantly different in comparison with controls probably due to small number of patients (Tab. 3). However Kruskal-Wallis test showed the value near to the 5% signification level in spatial QRS-STT angle and in QRS-STT angle of frontal and left sagittal plane opening angle. Similarly the heigh level of SD in Tab. 3 is due to the small number of patients. On the other hand, the similar results were obtained in frontal plane, ie no change in QRS axis deviation and small change in QRS-STT angle in both groups of DM 1 patients in comparison with controls (Tab. 3).

We have shown the significant changes in heart depolarization and repolarization as well as in activation time and amplitude of Q wave in the young 1st type diabetic patients comparing to healthy nondiabetic controls. As far as no diabetic patient was known to suffer from coronary artery disease and only some of them fulfilled the criteria for autonomic neuropathy the body surface mapping changes could be attributed to the specific influence of

diabetes. Diabetic patients have fasting as well as postprandial hyperglycemia (Alberti *et al.* 1978, Alberti and Zimmet 1989, Pickup and Williams 2003). They are using more fatty acids and less glucose for cardiac muscle energy metabolism comparing to nondiabetic subjects (Pickup and Williams 2003). This could lead to some changes in cardiomyocytes membranes.

The comparison of DM1 patients with and without AN showed some different results in heart electric field parameters. The heart rate was significantly faster in DM1 patients with AN than in DM1 patients without AN, the R-R, QRS and QT intervals shortened more in DM1 with AN than in DM1 without AN.

DM1 patients with and without AN presented the similar changes of some parameters of heart electric field on BSM. However, several parameters of heart electric field on BSM were more significantly changed in DM1 patients with AN patients than without AN in comparison with controls.

The significant changes of depolarization and repolarization in body surface mapping seen in our young DM1 patients could be explained by the activation of adrenergic system. The changes of these parameters were more pronounced in DM1 patients with AN. The observation of no statistically significant differences of BSM when only two diabetic groups have been considered could be attributed to limited number of diabetic patients included into study. As far as autonomic neuropathy is associated with significantly increased sympathetic activity (Javorka *et al.* 2005, Ruttkay-Nedecky 2001), the results of our study are in favour of idea that body surface changes in 1st type diabetic patients are related to power of adrenergic activation.

The similar results of heart electric field parameters on body surface mapping were observed in depressive patients treated with the tricyclic anti-depressants (Paclt *et al.* 2003, Slavicek *et al.* 1998). This observation could be important as far as it is known that among diabetic patients there is high incidence of depressive subjects (Pickup and Williams 2003).

In summary, our results confirmed the significance of body surface mapping changes in 1st type young diabetic patients. The presence of more pronounced changes in the heart electric field parameters in DM1 patients with AN than without AN when compared with controls, supports hypothesis that BSM changes in DM1 patients are associated with the augmentation of the adrenergic activation.

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Tab. 1. The characteristics of controls and diabetic patients type 1 (DM 1) with or without autonomic neuropathy (mean±SD).

	Controls (n=30)	DM1 without AN (n=29)	DM1 with AN (n=25)	p
Age (years)	29.60±3.22	32.59±5.55	32.24±5.77	NS
DM1 duration (years)	–	13.23±7.82	13.82±6.64	NS
HbA1c (%)	–	6.41±1.28	6.96±1.11	NS
Insulin dosage (units/day)	–	42.44±10.71	46.18±11.47	NS
BMI (kg/m ²)	24.30±2.98	23.44±2.80	24.40±2.32	NS

AN – autonomic neuropathy, DM1 – type 1 diabetes mellitus, AN – autonomic neuropathy, HbA1c – glycolated hemoglobin, BMI – body mass index (kg/m²)

Tab. 2. Heart rate, PQ, QRS, QT, QTc and R/R duration (means±S.E.M.) in controls and DM1 patients with or without autonomic neuropathy measured by classic 12-leads ECG.

Parameter	Controls (n=30)	DM1 without AN (n=29)	DM1 with AN (n=25)	p M-W	p K-W
Heart rate (min ⁻¹)	73.97 ±1.79	82.24±2.54*	92.24±3.00**	<0.01	0.000
PQ (ms)	153.93±5.81	142.82±3.59	154.08±10.08	NS	0.743
QRS (ms)	87.60±2.28	78.62±1.51*	75.52±1.63**	<0.01	0.001
QT (ms)	376.53±4.05	352.24±5.90**	350.72±10.08**	<0.01	0.000
QTc (ms)	404.53±12.08	424.03±16.31	431.40±12.66	NS	0.241
RR (ms)	753.29±22.28	751.48±23.05	668.09±25.89*	<0.05	0.014

DM1 – type 1 diabetes mellitus, AN – autonomic neuropathy

* p<0.05; ** p<0.01 in comparison of controls and diabetic patients; comparison of heart rate and QRS duration in DM 1 patients with or without AN (second and third column) – p<0.05; p M-W – Mann-Whitney test; p K-W – test Kruskal-Wallis for all three groups

Tab. 3. VCG QRS-STT angles (expressed in degrees) in frontal, transversal, left sagittal planes and the spatial QRS-STT angle (Mean±S.E.M.).

Parameter	Controls (n=30)	DM1 without AN (n=29)	DM1 with AN (n=25)	p K-W
QRS-STT angle (frontal plane)	-1.03±8.25	-3.00±6.11	-0.52±4.33	0.923
QRS-STT angle (transversal plane)	-48.77±7.93	-68.38±7.95	-48.00±7.75	0.128
QRS-STT angle (left sagittal plane)	-43.77±4.13	-62.34±15.26	-64.60±9.27	0.092
QRS-STT spatial angle	44.10±6.33	59.24±6.17	49.12±5.02	0.094
QRS axis in frontal plane	38.30±6.84	37.66±5.89	38.12±5.41	0.833

p K-W – test Kruskal-Wallis; for more detailed description see Tab. 2

Tab. 4. The selected parameters of BSM (means±S.E.M.), the significant differences between DM1 patients with or without AN, and control subjects.

Parameter	Controls (n=30)	DM1 without AN (n=29)	DM1 with AN (n=25)	p K-W
DIPMmax10 (μV)	100.67±10.92	147.21±10.95**	145.24±21.73	0.001
DIPMmax30 (μV)	716.60±55.41	1016.20±68.17**	993.60±45.80	0.005
DIPMmax50 (μV)	1051.17±98.93	747.52±69.36	679.64±83.16*	0.015
DIPMmin10 (μV)	-48.50±4.06	-66.24±7.04*	-65.40±5.64	0.020
DIPMmin20 (μV)	-150.80±21.27	-239.03±41.11	-238.10±38.31*	0.017
DIPMmin30 (μV)	-370.13±61.07	-791.86±123.92**	-801.04±113.40**	0.000
DIPMmin40 (μV)	-792.43±102.80	-1502.14±158.60**	-1382.67±155.70**	0.000
DIIMmin (μVs)	-31.48±2.71	-46.21±4.28*	-38.17±3.61	0.026
DIAMmax40 (μVs)	14.11±1.05	19.84±1.31*	19.14±1.94**	0.004
DIAMmin40 (μVs)	-6.96±0.98	-14.15±2.13**	-12.75±2.16**	0.000
RIPMmax10 (μV)	144.67±21.09	114.45±16.46	84.6±18.65*	0.004
RIPMmin50 (μV)	-278.10±131.14	-127.26±11.64	-100.38±9.48*	0.026
RIPMmin60 (μV)	-316.67±120.94	-156.12±13.31	-130.00±11.56**	0.004
RIPMmin70 (μV)	-354.80±110.53	-188.52±16.19*	-157.62±12.63**	0.001
RIPMmin80 (μV)	-326.13±103.28	-178.50±16.02	-157.80±11.76**	0.005
Q-IPMAM min (μV)	-625.50±12.80	-850.34±17.85	-866.83±19.29*	0.023
ICHVAT (ms)	68.90±1.69	62.72±1.25*	59.70±1.35**	0.001
Q-IIM min (μVs)	-14.86±1.04	-21.96±2.63	-24.02±2.58*	0.016
QS-IPMAM min (μV)	-557.12±50.60	-772.17±98.92	-843.08±82.30*	0.036

DIPMmax, DIPMmin – maximum, minimum in depolarization isopotential map (μV)

DIIMmin – minimum in depolarization isointegral map (μVs)

DIAMmax 40, DIAMmin 40 – maximum, minimum in depolarization isoarea map from the beginning of QRS to 40th ms of depolarization (μVs)

RIPMmax, RIPMmin – maximum, minimum in repolarization isopotential map (μV)

Q-IPMAM – amplitude of Q wave (μV)

ICHVAT – activation time between the onset of electrical activation registered by orthogonal leads x, y, z and the thoracic surface ECG (ms)

Q-IIM – minimum in isointegral map of Q-wave (μVs)

QS-IPMAM – amplitude of Q and S wave (μV).

* p<0.05, ** p<0.01 in comparison with controls; p K-W – Kruskal-Wallis test for all three group