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The 38th International Congress on Electrocardiology

Kingston, Ontario, Canada, June 8–11, 2011

With more than 120 participants from all around the world, the 4-day journey was incredibly productive, not only because of its amazing scientific level but also for the opportunity to socialize with old and new friends. New research collaborations were planned during the meeting and the International Society of Electrocardiology has confirmed its intention to explore new frontiers and to expand to all regions of the globe.

There were over 50 abstracts accepted for presentation and they are presented in this issue of "Cardiology Journal". We would like to recognize abstracts and their authors who received Young Investigator Awards:

1st PRIZE: Walther Schulze

Critical times based activation time imaging

Walther H.W. Schulze[1], Martin W. Krueger[1], Kawal Rhode[2], Reza Razavi[2], Olaf Doessel[1]

[1]*Institute of Biomedical Engineering, Karlsruhe Institute of Technology, Germany*
[2]*Division of Imaging Sciences, King's College London, United Kingdom*

2nd PRIZE: Rodrigo Miranda

The right ventricular (RV) septum presents the optimum site for maximal electrical separation (MES) in biventricular pacing

Rodrigo Miranda, Kevin Michael, Hoshiar Abdollah, Adrian Baranchuk, Christopher Simpson, Damian Redfearn

Queen's University, Kingston, Canada

3rd PRIZE: Helen Pang

Reverse atrial electrical remodeling induced by CPAP in patients with severe obstructive sleep apnea

Helen Pang, Damian Redfearn, Christopher Simpson, Kevin Michael, Effie Pereira, Peter Munt, Michael Fitzpatrick, Adrian Baranchuk

Kingston General Hospital, Kingston, Ontario, Canada

We are glad that this congress provided a forum for the presentation of early work by many young investigators interested in the field of electrocardiology.

Adrian Baranchuk, MD, FACC
Chair, 38th International Congress on Electrocardiology

The microstructure of heart rate asymmetry during sleep in relation to the severity of obstructive sleep apnea syndrome

Kokab Awan, Adrian Baranchuk, Przemyslaw Guzik, Tomasz Krauze, Jaroslaw Piskorski, Carlos A. Morillo, Damian P. Redfearn, Christopher S. Simpson, Michael Fitzpatrick

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Background: Heart rate decelerations and accelerations have unequal input to heart rate variability (HRV) and patterns created by consecutive cardiac cycles — this phenomenon is known as heart rate asymmetry (HRA). The analysis of monotonic runs of heart rate decelerations and accelerations provides a detailed insight into the HRA microstructure and thus of HRV. The aim of the study was to evaluate the relation between the severity of obstructive sleep apnea (OSA) and the HRA microstructure during sleep.
Materials and methods: Seventy eight patients with suspected OSA underwent overnight polysomnography. The 300-minute ECGs from the polysomnography were selected and analyzed. The HRA microstructure was quantified by measuring (1) the contribution of monotonic runs of decelerations or accelerations of different lengths to the number of all sinus beats, and (2) the length of the longest deceleration and acceleration runs.
Results: There were 19 patients with no/mild OSA (AHI 5.1 ± ± 2.5/h), 18 with moderate OSA (AHI 21.8 ± 4.0/h) and 41 with severe OSA (AHI 42.8 ± 17.4/h). Patients with severe OSA had significantly reduced deceleration and acceleration runs of length 1 compared to the moderate OSA group, and compared to patients with no/mild OSA they had an increased number of longer runs (from 5 to 10 for accelerations and from 5 to 8 for decelerations; $p < 0.05$ for all comparisons). The longest acceleration runs were significantly longer in severe OSA group ($p < 0.05$) than in subjects with no/mild OSA.
Conclusions: HRA microstructure is related with OSA severity. An increased number of longer deceleration and acceleration runs is more common in severe OSA patients.

Parkinson disease mimicking ventricular arrhythmia

Thiago Midlej Brito, Fernando Figuinha, Gabriel Carmo, Fausto Santos, Nelson Samesima, Mucio Oliveira Jr., Carlos Alberto Pastore

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Male, 80 years old, hypertensive, dyslipidemic, myocardial infarction (stent in LAD) and Parkinson disease, daily use of ASA, propanol, enalapril, simvastatin and levodopa. Admitted to the ER with atypical precordial pain of moderate intensity, duration < 20 min, not irradiating, with neither improvement nor worsening. On clinical examination he was normal, HR 60 bpm, BP 120 × 70 mm Hg. ECG showed tachycardia (HR 250 bpm), with wide QRS complexes. Due to the intense muscular tremor ECG had to be repeated after immobilization of all limbs, revealing sinus rhythm (HR 55 bpm), 1st degree atrioventricular block, infarction in (inactive) anteroseptal and possibly inferior areas, compatible with the myocardial scintigraphy that showed discrete left ventricular dysfunction (LVEF 50%), as well as inferior and septal akinesia. Taking into consideration the patient's coronary heart failure and his ECG, it made us think of tachycardia of ventricular origin (monomorphic VT or ventricular flutter) as a differential diagnosis. However, the initial physical examination did not agree with the above mentioned possibilities, since the heart rate then (250 bpm) is usually linked to hemodynamic instability. Tremor at rest in Parkinson disease is present in up to 100% of patients, showing 4- to 5-Hz frequency, which is compatible with the initial ECG, which featured 5 QRS complexes every second. Therefore, we conclude that the rhythmic muscular tremor in Parkinson disease can mimic a tachycardia of ventricular origin.

Negative sequence is a new law in physics and medicine

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Background: Abnormal inscription directions (AID) of the P-loop, which are linked with the partial negative sequence or complete negative sequence in physics, have been documented. Can they be found in T-loop of patients with acute myocardial infarction (AMI)? Could it be possible that the heart behaves like a biological generator in physics?
Materials and methods: One hundred and seventy eight consecutive patients with suspected acute coronary heart disease within 24 h of their admission into the Coronary Care Unit enrolled in this serial emergency Frank vectorcardiographic study.
Results: Total of 473 serial emergency vectorcardiographic tracings were recorded by one physician from 148 consecutive patients with AMI. Their age was 61 ± 9.8 years, and 129 were male, 19 female. A total 137 (93%) out of 148 patients had AID of the T-loop. Twenty-three (16%) of the 148 patients developed ventricular fibrillation or tachycardia or both. Twenty (87%) among these 23 had AID of the T-loop.
Conclusions: The heart is a biological generator, which has also inherited the same problems as generators. The types of figure-of-eight or clockwise rotation of the T-loop during repolarization phase of the heart are linked with the partial or complete negative sequence in physics. From findings in diseased hearts (in biology) and well-established malfunctioning generators (in mathematical physics), the negative sequence is a new law in physics and medicine.

Negative sequence voltages in acute subarachnoid hemorrhage: A preliminary report

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Background: Is there any evidence of negative sequence voltages (NSV) in patients with acute SAH?
Materials and methods: A total of 26 patients with suspected acute SAH received emergency vectorcardiographic examinations before and after their emergency brain surgery.
Results: Eleven patients (6 female, 5 male, age 55 ± 19.5 years) had their vectorcardiograms (VCG) prior to their emergency brain surgery for a confirmed acute SAH. In the atria, VCG revealed three patients (27%) with NSV in the P-loop (2 with figure-of-eight ['8'] in the Right Sagittal [RS] plane, 1 with clock-

wise rotation in the Frontal [F] plane). In the ventricles, VCG showed six patients (54%) with NSV in the T-loop (1 with '8' in the F plane; 5 with '8' in the Horizontal [H] plane: 1 with additional '8' in both F and RS planes, the other one with additional anti-clockwise in the RS plane). Due to electrical interference, only 6 patients completed second VCG in the surgical ICU. In the atria, 1 had '8' of the P-loop in the RS plane turning into normal. One had clockwise rotation in the F plane turning into '8' in both F and RS planes. Two normal changed into one with '8' in both the F & RS planes, the other one in RS plane only. In the ventricles, 3 normal changed into worse condition: 1 with '8' in the RS plane, 1 with anti-clockwise in the RS plane, 1 with '8' in the F plane and clockwise in the H plane. One had '8' in the three planes turning into anti-clockwise in the RS plane, but normal rotation in the F plane. One with '8' in the H plane returned to normal, but the F plane changed from normal into '8'. One with only '8' in the H plane downgraded into '8' in both H and RS planes after surgery.

Conclusions: In patients with acute SAH, one-third of them have NSV in the atria. Half of these patients have NSV in the ventricles. Emergency brain surgery can alter NSV in the heart, but surgery itself can also contribute to iatrogenic NSV in the heart.

Progression of QTc in normal newborns from birth to 30 days

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The purpose of this study is to measure and analyze the progression of the QT interval in normal newborns from birth to 30 days. There were 25 at 0–24 hours, 112 at 1–3 days, 22 at 4–7 days, and 71 at 8–30 days, totalling 230 babies. Measurements were of heart rate, QT duration (QT) QTc (Bazett) and QTc (Fridericia), the QT was highest at 1–3 days but because of the highest heart rate at 0–24 hours, the QTc (Bazett) was lower at 0–24 hours than at 1–3 days despite lower QT. Although heart rate increased to newborn levels at 8–30 days, QTc increased once again indicating an additional separate factor, the QT duration (not corrected) progressively decreased from 1–3 days to 8–30 days, as did the QTc by Fridericia's method. In summary, much but not all of the changes in Bazett's QTc from 0–30 days are related to changes in heart rate, but actual measures are better reflected by Fridericia's method. The high QTc (Bazett) often seen at 1–3 days may in fact be because of the low heart rate.

Lossless redundancy of the 12-lead ECG

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Background: Identification and removal of redundant leads is a necessary step in reducing the amount of 12-lead ECG data in emerging electronic data platforms. By expressing the ECG as a system of 12 linear lead equations of the 9 electrode terms {*RA, LA, LL, C1, C2, …, C6*}, the system is found to be rank-8, and hence theoretically reducible to 8 equations of 8 terms without any loss in electrode data.

Materials and methods: For lead equation reduction, it is observed that the 6 frontal lead equations is rank-2, hence any 4 of the frontal leads may be expressed in terms of the remaining 2 frontal leads, leaving 8 non-redundant ECG equations at any given time. The 4 frontal leads are first expressed in terms of the 3 frontal electrodes {*RA, LA, LL*}, which are then expressed in terms of 2 remaining frontal leads using the Moore-Penrose pseudoinverse. For electrode term reduction, it is observed that the sum of all coefficients is zero for each of the 12 ECG equations. Hence any one of the 9 electrode terms may be eliminated by subtracting each of the 9 terms by that term.

Results: Given 6-choose-2 permutations of equation reduction and 9-choose-1 permutations of term reduction, the 135 permutations to relate leads and electrodes as 8 equations of 8 terms are individually computed and listed. In order to minimize propagation of small digitization errors in the electrode data to the lead data, each 8 × 8 system of equations is evaluated for backward stability by its condition number. Of the 135 permutations, three best and equally well-conditioned permutations are found in the expression of (1) {*I, II, V1, V2, …, V6*} in terms of {*LA–RA, LL–RA, C1–RA, C2–RA, …, C6–RA*}, (2) {*I, III, V1, V2, …, V6*} in terms of {*RA–LA, LL–LA, C1–LA, C2–LA, …, C6–LA*}, and (3) {*II, III, V1, V2, …, V6*} in terms of {*RA–LL, LA–LL, C1–LL, C2–LL, …, C6–LL*}.

It is further discovered that the permutation (3) produces a linear combination that is itself a fundamental involution, such that the lead-to-electrode function is *identical* to the electrode-to-lead function.

Conclusions: Reduction of the 12-lead ECG equations may be useful in optimizing hardware and software design of the ECG. And identification of best-conditioned and involutive electrode-lead relations may provide more sampling flexibility in future mobile ECG devices.

Study of spectral analysis of the resting ECG for ischemia classification

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Background: Historically, spectral analysis of the ECG has been applied more toward rhythm analysis than to quantification of shape. This blinded pilot study applies spectral quantification of the shape of the JT segment in the resting ECG to detection of cardiac ischemia. ECG findings are assessed alongside concurrent clinical diagnosis of exercise-induced ischemia by myocardial perfusion imaging (MPI).

Materials and methods: For each of the 502 consenting subjects in this study, conventional 10-second ECG signals were digitally acquired. The recurring shape of the ECG segment from the J-point to the peak of the T loop (JT segment) was isolated by windowing, resampling, and averaging in the time domain. Harmonics were subsequently generated from the isolated JT segments by discrete cosine transform. An indicator termed "QED" was derived from a ratio of the frequency-domain harmonics and computed for each of the 12 leads. At the single visit in this study, standard evaluative examinations of stress ECG and myocardial perfusion imaging were conducted on each subject after acquisition of the resting ECG. Characterization of ischemia as positive (MPI-1) mild, moderate, and severe or negative (MPI-0) for each vascular territory was performed.

Results: One way analysis of variance (ANOVA) was employed to distinguish various normal and ischemia groupings, with statistical significance defined as $p < 0.05$. In analysis of the MPI-0 *vs*. MPI-1 groups, where the ischemic group for each vascular territory numbered between 27 and 32 cases among the 495 analyzed, the QED showed significance in leads I, aVR, V1, V4, V5,

and V6 for the RCA territory, in leads I, III, aVL, V5, and V6 for the LAD territory, and in leads I, II, aVR, aVL, V1, V4, V5, and V6 for the LCx territory. When the combined MPI-0 and MPI-1 mild groups were compared with the combined MPI-1 moderate and severe groups, where the ischemic group for each vascular territory numbered between 14 and 16 cases among the 495 analyzed, the QED showed significance in leads V4, V5, and V6 for the RCA territory, in leads I, III, aVL, V1, and V6 for the LAD territory, and in leads I, V1, V4, V5, and V6 for the LCx territory.
Conclusions: QED analysis of signals obtained from resting 12--lead ECG may be useful as a screening method for predicting exercise-induced cardiac ischemia. Furthermore, "forgotten leads" aVR and III are shown to be informative for ischemia detection in the frequency domain, further demonstrating time-frequency complementarity in ECG analysis.

Maximum Ito expression in subepicardial cardiomyocytes determines the extent of body surface early repolarization potentials. A simulation study

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Background: The association of the presence of J waves in infero-lateral and right precordial ECG leads with occurrence of malignant arrhythmias has been reported. A better understanding of the early repolarization potentials (ERP) genesis could improve their usefulness as prognostic markers. The aim of this study was to verify whether a maximum Ito conductance (gto) gradient in the left ventricle can explain, by itself, the body surface distribution of ERP.
Materials and methods: We used: an anatomical model for the ventricles and a human thorax with 370 electrodes, the Luo-Rudy II (2000) dynamical model for the action potentials and data reported in the literature for electrophysiological parameters in the ventricular strata. We ran a series of simulations in which the only variable parameter was a gto in the range of 70–375 ms/ /mV in the subepicardium. Two thresholds (0.05 and 0.1 mV) were chosen for the amplitude of body surface ERP at 10 msec after the ventricular activation end.
Results: The distribution of potentials on the simulated thorax was visually comparable with that present in normal body surface maps of healthy individuals. Leads with ERPs above threshold occupied a continuous region that covered a small anterolateral area with lower gto values, extended to the inferior leads and to the right with higher values. Both the amplitude and the number of leads with a potential above threshold increased linearly with the maximum Ito conductance, in the range of 0 to 24% of the simulated thorax surface for a 0.1 mV threshold.
Conclusions: The ERP amplitude variability can be explained solely by a plausible interindividual variability of the gto transmural gradient that is homogenous along the subepicardial myocardium. This does not rule out a possible influence of heterogeneity of other electrophysiologic parameters in the final expression of ERP on the body surface.

Multifactorial QT interval prolongation: The risk of polypharmacy

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Background: Acquired long QT (LQT) interval is often thought to be a consequence of drug therapy and electrolyte disturbances. However, multiple clinical risk factors have also been implicated in the potentiation of this arrhythmia. We report a series of cases of multifactorial acquired LQT interval that highlight the potential effects of polypharmacy on QT interval and that demonstrate the role of multiple clinical risk factors that may allow for the manifestation of torsades de pointes (TdP).
Materials and methods: Case series of 10 patients presenting to 4 tertiary care hospitals with LQT and at least 2 risk factors for developing LQT. Clinical characteristics, type of medications, electrolyte disturbances and course in hospital were analyzed.
Results: Mean age was 53.6 ± 16.8 years with 7 females. Five had hypertension. One patient demonstrated complete AV block. Average QTc interval at presentation was 632.3 ± 96.5 ms. Seven patients developed TdP. In 3 cases, LQT was not initially detected and amiodarone was administered, followed by development of TdP. Patients were taking an average of 2.3 ± 1.1 QT-prolonging medications, including an antidepressant in 7 cases and a diuretic in 7 cases. All patients had an electrolyte abnormality; 6 patients presented with severe hypokalemia (< 3.0 mmol/L). Average serum potassium and magnesium were 3.29 ± 1.34 mmol/L and 0.76 ± 0.10 mmol/L, respectively. There were no deaths. According to the Naranjo Adverse Drug Reaction Probability Scale, the medications that most probably affected QT interval were: amiodarone, venlafaxine, quetiapine, citalopram, escitalopram and domperidone.
Conclusions: This case series highlights the risks of polypharmacy in the development of LQT and TdP. It illustrates the importance of recognizing impending interactions between medications and clinical risk factors and demonstrates the significance of early detection of LQT in patients with multiple risk factors in ensuring appropriate treatment.

Bradycardia as a cause of angina: The new 'Bradyangina syndrome'

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Background: Angina is defined as a sensation of pressure or retrosternal pain with a rather specific pattern of irradiation, with physical or emotional stress, or even at rest, and secondary to a decrease in myocardial oxygen supply. Bradycardia is defined as a persistent or transitory decrease in heart rate below 60 beats per minute, due to primary, secondary, and reversible or irreversible causes. There is an association between heart rate and

cardiac output. Could it be possible that a reduction in the heart rate results in a reduction in cardiac output to a point that it is responsible for myocardial ischemia, and as such, angina?

Materials and methods: This is a retrospective–prospective study of patients with angina of unknown origin and sinus bradycardia diagnosis who presented to our cardiology service between August 1st 2007 and August 31st 2009. Diagnostic approach included coronarography, or non invasive measures such as stress echocardiography or myocardial perfusion test with radiotracers. Patients were treated with pacemaker implantation, discontinuation of medications with negative chronotropic effect and/or follow up in those with transitory or non pharmacologic causes of bradycardia. After performing the interventions, all patients were followed up and evaluated for the presence of angina or bradycardia, and persistence of symptoms.

Results: A total of 60 patients were evaluated; 70% were men. Mean age was 52.5 ± 16.3 (SD) years. Pacemaker implantation was performed in 78.3% of patients. Medication associated with bradycardia was discontinued in 11.7% of patients. During follow up, 100% of patients reported not having new episodes of angina after the intervention was performed.

Conclusions: With coronary disease discarded, the study of rhythm anomalies as a potential etiology of the symptomatology should be performed. The association between bradycardia and chest angina could be newly defined as "Bradyangina syndrome".

Left atrial size is associated with ventricular arrhythmias in the 24-hours Holter in hypertensives without left ventricular hypertrophy

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Background: To examine whether left atrial (LA) size is associated with ventricular arrhythmias in patients with hypertension without left ventricular hypertrophy.

Materials and methods: This study included 144 senior adult and elderly (median age 65.17 ± 7.49 years, range 55–89) hypertensive subjects (12.5 per cent were males). The inclusion criteria were: aged ≥ 55 years, left ventricular ejection fraction > 55%, and did not present previous cardiovascular events by questioning and physical examination; we excluded those subjects with atrial fibrillation and/or severe regurgitation valvular (mitral or aortic). In all patients, anthropometric data and blood pressure were measured; and an echocardiogram and a 24-h Holter study were performed. Planimetered LA area was measured from the apical four-chamber-view. The patients with left ventricular hypertrophy were excluded (left ventricular mass index ≥ 136 g/m^2 in male and ≥ 110 g/m^2 in female). An analysis of Spearman correlation was used to determine the association between LA area and presence of ventricular arrhythmias. A logistic regression model was used to evaluate the effect of gender, age, systolic blood pressure, indexed LV mass, and LA area on presence of ventricular arrhythmias. P < 0.05 was considered statistically significant.

Results: Men presented ventricular arrhythmias more than women (80.0 *vs*. 42.5%, respectively, p = 0.016). The overall mean of LA area was 13.75 ± 3.15 cm^2; in males it was 14.90 ± ± 3.23 cm^2 and females 13.46 ± 3.08 cm^2 (p = 0.049). LA area was significantly correlated with presence of ventricular arrhythmias (r = 0.285; p = 0.012). Only in women, the logistic regression analysis showed that LA area (OR = 1.254, 95% CI = 1.025–1.534, p = 0.028) predicted independently the presence of ventricular arrhythmias.

Conclusions: LA area appears to predict ventricular arrhythmias in elderly subjects with hypertension without left ventricular hypertrophy. The increase of LA area might constitute physiological and anatomical early alterations preceding detectable changes in ventricular geometry in hypertensive heart disease.

A low E/A ratio and a protracted isovolumetric relaxation time is a new marker for severe ventricular arrhythmias in patients over 55

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Background: Diastolic dysfunction measured using the transmitral E/A index correlates with the occurrence of ventricular tachycardia, which might be an indicator of the risk of sudden death. The aim of this study was to determine the best predictor of severe ventricular arrhythmias (SVA) in patients aged over 55.

Materials and methods: 145 persons with an ejection fraction > 55%, no coronary heart disease and atrial fibrillation underwent echocardiography with the emphasis on diastolic function and 24-hour Holter monitoring to classify ventricular arrhythmias according to the Lown criteria. SVA was defined as Lown ≥ III.

Results: 21.23% of the patients presented with SVA. These patients, compared to those with no SVA, presented with higher systolic pressure, left atrial size and indexed left ventricle mass, a greater prevalence of E/A < 1 and a significant lengthening of isovolumetric relaxation time (IVRT). E/A < 1 relaxation was associated with a 2.5 times higher risk of severe arrhythmia (OR = = 2.545; p = 0.034; IC 95% 1.052–6.160). An IVRT > 100 ms in itself gave a 3.5 times higher risk of SVA (OR = 3.516; p = 0.003; IC 95% 1.514–8.165). Patients with an E/A ratio < 1 and IVRT > 100 ms have more SVA than those with normal patterns (OR = 4.572; p = 0.005; IC 95% 1.51–18.83), a risk which is maintained even when adjusted for age, LV hypertrophy and systolic artery pressure (RPC = 3.61; IC 95% 1.39–9.36). In multivariable analysis, only E/A < 1 + IVRT > 100 was related to SVA (p < 0.008). Even in patients with normal geometry, an IVRT > 100 ms was associated with a 3.65 times higher risk of SVA.

Conclusions: A low E/A ratio and a protracted IVRT > 100 ms is a new, independent predictor of SVA in patients over 55 years of age, even in individuals with normal ventricular geometry.

The role of interacting proteins in human Ether-a-go-go-related gene (hERG) channel membrane stability

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The human *Ether-a-go-go* related gene (hERG) encodes a cardiac potassium channel, I_{Kr}, which is critical for maintaining heart rhythm. Decreases in hERG current delay cardiac repolarization, and therefore the returning of cardiac myocytes to their resting membrane potential. This leads to long QT syndrome (LQTS), a cardiac electrical disorder with high risk of sudden cardiac death. Our lab recently found that hypokalemia destabilizes hERG from the cell membrane and causes LQTS in experimental rab-

bits. The present study aims to characterize the roles of hERG-interacting proteins, KCNQ1, KCNE1 and KCNE2, in endocytosis of hERG channels induced by hypokalemia. After transfection of these hERG-interacting proteins into a hERG stable cell line, expression levels and function of hERG were analyzed using whole-cell voltage clamp, immunocytochemistry and Western blot analysis. Our data show that the effect of KCNE1 and KCNE2 was minimal, as no difference in hERG expression and function was observed between KCNE1/KCNE2-transfected hERG stable cells and control cells. Interestingly, KCNQ1, the pore-forming subunit of I_{Ks}, had a profound effect in stabilizing the hERG protein in the membrane, allowing hERG to pass current even after a 6-hour hypokalemic challenge, whereas there was a complete lack of current in cells solely expressing hERG under the same conditions. Overall, the susceptibility of the mature hERG channel to hypokalemia-induced internalization and degradation is reduced when hERG-expressing cells were coexpressed with KvLQT1. These results reveal a stabilizing role of KCNQ1 in hERG function, and extend our understanding of cardiac electrophysiology and LQTS.

Race- and gender-specific left ventricular hypertrophy thresholds for automated ECG analysis

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Background: We sought to refine race- and gender-specific classification criteria for left ventricular hypertrophy (LVH) by Cornell Voltage (CV) and Sokolow-Lyon Voltage (SL) to enhance their clinical utility.
Materials and methods: We used original CV and SL LVH classification thresholds and thresholds established previously for 95% specificity in the Cardiovascular Health Study (CHS) population of older adults with echocardiographic (Echo) LVH as an independent standard. Subsequently we evaluated LVH prevalence by the original and modified criteria in the National Health and Examination Survey (NHANES 3) population. Excluded were ECGs with QRS duration > 120 ms and major non-LVH related ECG abnormalities by the Minnesota Code. The rationale for threshold modification was the known low specificity of SL criteria in blacks and for the prevalence comparison that with equal specificity, the prevalence in NHANES 3 by CV and SL criteria should be reasonably equal if their sensitivity is equal. In the community-based US population of CHS Echo-LVH prevalence was 15% (within 3%) in all subgroups by race and gender.
Results: Threshold modifications needed for CV were small but substantial for SL. LVH prevalence estimated by CV criteria was 10 to 13% although lower (6%) in white males, and 2% or lower in all subgroups by SL criteria reflecting their known poor sensitivity. The reason for the apparent poor performance of ECG-LVH, SL criteria in particular is largely due to differing impact on LVH prevalence by ECG amplitude reduction in overweight, old myocardial infarction etc. Known differing influence of overweight on ECG-LVH criteria in various subgroups by gender and race needs to be considered to further improve LVH criteria.
Conclusions: Cornell voltage performance appears reasonable but clinical utility of SL criteria for LVH remains questionable.

Three dimensional phase space ECG and complex sub-harmonic frequencies predict ventricular arrhythmia in ICD recipients

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Background: Risk stratification for sudden cardiac death (SCD) remains problematic. We hypothesized that a contemporary algorithm capable of detecting aperiodic complex sub-harmonic frequencies (CSF) may detect differences in the ECG spectra of patients (pts) at risk for SCD.
Material and methods: The cohort consisted of 75 pts in whom an ICD was implanted. The mean time for ICD implantation was 5.02 ± 1.67 years. All pts were not pacemaker dependent. Group A consisted of 37 pts who experienced appropriate device therapy (DT). There were 38 Group B pts who appropriately did not undergo DT. The groups were comparable with regard to age, EF and NYHA class. Three-dimensional (3D) orthogonal lead, 1 kHz data was recorded during native rhythm and examined using a 3D Fast orthogonal search (FOS) technique. The presence of CSF structures was detected in Groups A and B using FOS to extract 3D elements which is the potential ventricular arrhythmia (VA) substrate. These subspace elements were quantified in magnitude using a phase space clustering algorithm. Correlation between presence of CSF and DT was evaluated.
Results: In Group A, CSF and its associated patterns were observed in 31 of 37 pts receiving DT. In Group B, CSF were observed in 3 of 38 pts. Using a non-linear phase space clustering algorithm, Group A and B pts could be predicted by CSF 83% and 92% of the time respectively ($p < 0.001$) with a sensitivity of 91% (95% CI 75–98%) and specificity of 85% (95% CI 70–94%).
Conclusions: This novel 3D analysis found an increase in CSF from ICD pts who had received appropriate DT compared to those pts without VA. This analysis appears to differentiate pts with VA substrate, and may serve as a better risk stratification agent than EF alone.

Atrial-His and His-Ventricle intervals short-term variability is asymmetric

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Background: Heart rate decelerations have a larger contribution than accelerations to short-term heart rate variability at supine rest. We aimed at the evaluation of asymmetric properties of the short-term variability of the Atrial-His (AH) and His-Ventricle (HV) intervals.
Materials and methods: Intracardiac tracings were recorded in 10 consecutive patients (18–66 years old; 8 female) during a routine electrophysiological study. The AH and HV intervals were recorded from the His bundle electrograms. The collected time series (5 separate 1-minute tracings for each patient) of consecutive AH and HV intervals were analyzed with the Poincare plot. The part of short-term variance related to the prolongations of the AH or HV intervals was measured by SD1p_AH

or SD1p_HV, respectively, and related to the AH or VH shortenings by SD1s_AH and SD1s_HV, respectively. The Wilcoxon test for paired data compared the descriptors of the AH or HV intervals asymmetry. The contribution of the parts of variance related to the prolongations of the AH (C1p_AH) or VH (C1p_HV) were normalized to the respective short-term variances. The binomial test checked whether the proportion of recordings with C1p_AH > 50% and C1p_HV > 50% is significantly different from 0.5. The results are shown as median and interquartile range (IQR).

Results: The AH interval was 79.2 ms (IQR: 76.1–85.5 ms) and HV interval was 42.5 ms (IQR: 48.4–50.4 ms). The C1p_AH was 51.5% (IQR: 48.6–53.45%) and C1p_HV was 52.0% (IQR: 49.6–55.3%). The SD1p_AH (1.8 [IQR: 1.5–2.3] ms) was significantly larger than SD1s_AH (1.7 [IQR: 1.4–2.2] ms; $p = 0.0142$), and SD1p_HV (1.5 [IQR: 1.4–1.7] ms) was larger than SD1s_HV (1.4 [IQR: 1.3–1.6] ms; $p = 0.0006$). The proportion of C1p_AH > 50% was 0.68 ($p = 0.0153$) and for C1p_HV > 50% it was 0.74 ($p = 0.0009$).

Conclusions: The AH and HV intervals prolongations contribute significantly more to their short-term variability. This study shows that short-term variability of the AH and HV intervals is asymmetric.

Increased rate of fast-changing microstructure of heart rate asymmetry predicts mortality in patients undergoing a clinically indicated exercise test

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Background: Unequal contribution of heart rate decelerations and accelerations to heart rate variability is called heart rate asymmetry (HRA). The number of deceleration runs, which describe HRA microstructure, is reduced in high-risk post infarction patients. We aimed at the analysis of the predictive value for total mortality of 2 indices of fast-changing HRA microstructure related to decelerations (MSD1) and accelerations (MSA1) in patients undergoing a clinically indicated exercise test.

Materials and methods: Pre-exercise ECG recordings of at least 1 minute duration were taken from prospectively collected 944 consecutive patients (mean age 58.0 ± 12.7 years; 326 women) from the Finnish Cardiovascular Study. For MSD1 and MSA1 calculation, RR intervals of sinus origin were used. The predictive value of MSD1 and MSA1 was analyzed with Receiver Operating Characteristics for Area Under Curve (AUC) value and Kaplan-Meier survival curves for hazard ratio.

Results: During mean follow-up of 56.9 ± 11.0 months there were 87 deaths for any reason. The AUC for total mortality for MSD1 was 0.73 (95% confidence interval [CI] 0.70–0.76; $p < 0.0001$) and for MSA1 was 0.72 (95% CI 0.69–0.75; $p < 0.0001$). The defined cut-off points were > 17.69% for MSD1 and > 16.85% for MSA1, respectively. The hazard ratio for MSD1 was 5.74 (95% CI 3.63–9.07; $p < 0.0001$) — and for MSA1 4.48 (95% CI 2.88–6.98; $p < 0.0001$).

Conclusions: In a general population undergoing a clinically indicated exercise test, patients at high risk of death have increased values of MSD1 and MSA1, which show that the rate of fast-changing microstructure is abnormally increased both for heart rate decelerations and accelerations. This study shows that heart rate decelerations and accelerations analysis in pre-exercise ECG of at least 1 minute duration carries important prognostic information. The clinical value of the analysis of heart rate asymmetry microstructure needs further exploration.

Hemodynamics and their variability in post-infarction *vs.* heart failure patients with implanted defibrillating device: Preliminary results of the Poznan-Team study

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Background: We compared selected hemodynamic parameters and the total variability of their beat-to-beat values between post myocardial infarction (PMI) and heart failure (HF) patients who underwent elective implantation of defibrillating device in the past.

Materials and methods: The prospective Poznan-Team project aims at predicting adverse clinical outcomes in patients with implanted defibrillating devices. The first 92 patients (mean age 64.0 ± 9.8 years; 76 male) underwent continuous 10-minute, noninvasive hemodynamic measurement by cardiac impedance. The mean values of hemodynamic parameters and the standard deviations of beat-to-beat hemodynamic data were used for the evaluation of hemodynamic variability.

Results: There were 56 PMI and 36 HF patients. There were no statistically significant differences in resting systolic and diastolic blood pressure or in cardiac index (CI) 2.7 ± 0.6 *vs.* 2.6 ± ± 0.5 L/min/m^2) or pre-ejection period (PEP) (111.6 ± 28.8 *vs.* 118.3 ± 27.4 ms) between PMI and HF patients. However, PMI compared to HF patients had significantly higher stroke index (SI) (41.9 ± 8.6 *vs.* 37.3 ± 9.7 mL/m^2; $p = 0.026$), longer left ventricular ejection time (LVET) (318.5 ± 44.4 *vs.* 292.2 ± 44.1 ms; $p = 0.005$), slower heart rate (64.7 ± 10.1 *vs.* 69.9 ± 9.1 beats/ /minute; $p = 0.007$) and reduced systolic times ratio (STR) (0.36 ± ± 0.11 *vs.* 0.42 ± 0.14; $p = 0.023$). In PMI patients the variability of SI was significantly higher (4.9 ± 1.4 *vs.* 4.4 ± 1.4 mL/m^2; $p = 0.049$) whereas the variability of PEP (16.7 ± 17.6 *vs.* 19.6 ± ± 10.0 ms; $p = 0.011$) and STR (0.07 ± 0.6 *vs.* 0.9 ± 0.5; $p = 0.013$) was significantly lower.

Conclusions: Both absolute values of hemodynamic parameters and their variability are different between PMI and HF patients with implanted defibrillating devices. Patients with PMI seem to have better preserved myocardial contraction and slower heart rate, with better variability of stroke index and more stable PEP and STR on a beat-to-beat basis.

Cardiac index on admission predicts mortality in patients with acute coronary syndrome with ST-segment elevation

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Background: This study aimed at the evaluation of the prognostic value of cardiac index (CI) measured noninvasively before emergency percutaneous coronary intervention (PCI) by cardiac impedance in patients with acute coronary syndrome with ST-segment elevation (STEMI).

Materials and methods: Noninvasive hemodynamic monitoring by cardiac impedance was started in 154 consecutive STEMI patients (mean age 59.5 ± 11.0 years; 50 female) instantly after admission. Receiver Operating Characteristics analysis with Area Under Curve (AUC) were used to define the optimal cut-off point for CI measured in 10-minute recordings collected before PCI. Univariate and multivariate logistic regression adjusted to age and gender was used to calculate the odds ratio (OR) with 95% confidence interval.

Results: Ten (6.7%) patients died of any cause during the maximal follow-up of 46 months. The mean value of CI on admission was 3.2 ± 0.7 L/m^2 in the whole group. At the admission, patients who died during the follow up compared with survivors were older (67.2 ± 9.7 *vs*. 58.8 ± 10.9 years; p = 0.02 Mann-Whitney test), had reduced systolic (102.4 ± 31.9 *vs*. 129.2 ± 26.6 mm Hg; p = 0.012) and diastolic blood pressure (62.3 ± 14.4 *vs*. 81.6 ± 16.0 mm Hg; p = 0.001), and CI (2.4 ± 0.3 *vs*. 3.5 ± 0.6 L/m^2; p = 0.016). The AUC for the association of CI with total mortality was 0.702 (p = 0.043) and the optimal cut-off point for high-risk group was set at CI < 2.79 L/m^2. The unadjusted odds ratio for this cut-off of CI was 6.1 (95% confidence interval 1.4–24.6; p = 0.012), and the adjusted CI was 4.8 (95% confidence interval 1.09–21.5; p = 0.039).

Conclusions: Upon hospital admission, cardiac index is reduced in STEMI patients with increased risk of mortality. This risk is particularly increased in patients with CI < 2.79 L/m^2, and the predictive value of reduced CI seems to be independent of age and gender. Further prospective studies are necessary to explore the clinical value of this observation.

A novel metric for quantifying percentage fractionation in AF electrograms and comparison with expert opinion

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Background: Clinicians Takahashi et al. have shown that catheter ablation in sites with a high percentage of continuous electrical activity is associated with a positive clinical outcome. We have developed an algorithm to compute this and we compare our percentage fractionation (PF) algorithm with that of current software, with the opinion of expert electrophysiologists as the gold standard.

Materials and methods: The opinion of 12 experienced electrophysiologists was gathered on 80 4-second signals showing left atrial electrograms recorded from 18 patients prior to catheter ablation using a visual analog scale from 0–100. PF was calculated as a percentage from 0 to 100. As well, results from a contemporary algorithm that assesses the mean cycle length of signals (CFE mean [St. Jude Medical]) were retrieved. PF was compared to median annotation with Pearson correlation, and CFE mean with Spearman correlation (ranked in descending order). The experts agreed to ablate at a cutoff rating of 70, and a ROC curve was generated for PF versus the decision to ablate.

Results: Spearman correlation between CFE mean and the gold standard was 0.27 (p = 0.016, 95% CI 0.045–0.49). Pearson correlation for PF was 0.78 (p < 0.01, and 95% CI 0.68–0.86). ROC curve sensitivity and specificity were 0.7727 and 0.8103 at the optimal cutoff point of 58.45 PF with AUC 0.89 CI (0.80–0.99). PF displayed on color-coded geometries differed from CFE mean maps qualitatively and quantitatively.

Conclusions: The agreement between the PF algorithm and the gold standard shows that PF can be used to guide operators to ablation sites. This, together with the clinical result of Takahashi et al., indicates that PF is a more accurate and precise metric for atrial activity.

Effectiveness of sodium channel blockers for preventing ventricular tachycardia in patients with LQT2

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Background: Experimental studies have demonstrated efficacy of sodium channel blockade to prevent torsades de pointes and abbreviate the QT interval in patients with LQT2. Clinical implementation of this approach is presented.

Materials and methods: Four children from four unrelated LQT2 families were studied and followed up for more than 4 years. The diagnosis was confirmed by course of disease and family history, QT prolongation and T-wave morphology on surface ECG, QTc dynamics on stress-test, specific features Body Surface Potential Mapping, and genetic analysis (in three cases).

Results: The three probands, 11 year-old, 13 year-old and 17 year-old, were diagnosed as a LQTS patient at age 5 to 7. Syncope, QT prolongation on ECG (QTc = 490 ms), abnormal T-wave morphology, bradycardia and rare single and couple polymorphic ventricular premature beats (VPB) without beta-blockers (BB) were detected. An extensive negative zone during ventricular repolarization on the body surface potential mapping suggested the LQT2. The patients were genotyped as LQT2 (KCNH2-IVS3+1G/C splicing mutation; KCNH2-L1045F and KCNH2-G604S). BB therapy (atenolol) did not fully prevent syncope and VPB, despite a full dose and a good compliance. Combined BB and sodium channel blocker (IC) therapy led to shortening of the QT interval, elimination of VPB and normalization of T-wave morphology. No syncope were observed during 27 to 72 months follow-up. Another 12 year-old boy clinically characterized as LQT2 was successfully treated with sodium channel blockers IC.

Shortening of the QT interval and no syncope were registered during 20 months.
Conclusions: These cases suggest that sodium channel blockade may be a valuable therapy in LQT2 patients in association with BB. Further studies are needed to better define the role of sodium channel blockers in LQT2.

The diagnostic value of stress-test in children with LQT1 and LQT2

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Background: Experimental and clinical data demonstrates the specific response of QT interval duration on adrenergic stimulation in patients with the most prevalent long-QT syndrome (LQTS) variants (LQT1 and LQT2). We aimed to evaluate the predictive value of specific features of ventricular repolarization in children with LQT1 and LQT2 by analyzing QTc dynamics on exercise stress test (EST).
Materials and methods: 24 children aged 6 to 17 (13 ± 3 years) from unrelated families with LQTS were enrolled in the study. The diagnosis was confirmed by QT prolongation, family history and course of the disease. Control group consisted of 15 healthy children aged from 8 to 16 (12 ± 2 years). Treadmill EST was performed using the Bruce walking treadmill protocol to examine QTc before (QTc1) and during EST on maximum heart rate (QTc2).
Results: All pts were divided according to the dynamics of QTc on maxHR (max HR = 132 ± 17 bpm). In 14 pts QTc2 was longer than QTc1 with a difference of +5 ms or more. Comparison with the genetic data showed that 93% of them (13 pts) had LQT1. In 9 pts QTc2 was shorter than QTc1 (≤ –5 ms); 8 of them (89%) had LQT2. In 1 child (LQT1) no difference was found. Baseline QT duration in pts with LQT1 was significantly higher than that in LQT2 pts (409 ± 34 and 457 ± 32). There was no difference in QTc1 between LQT1 and LQT2 pts (462 ± 41 and 492 ± 34). QTc2 in LQT1 pts was significantly longer than those in LQT2 pts (485 ± 29 ms and 453 ± 39). Children from the control group were characterized by normal value of QTc1 (428 ± 11) and significant shortening of QTc on EST (QTc2 = 411 ± 7).
Conclusions: EST revealed distinct response in QTc dynamics between LQT1 and LQT2 children and could be useful for pregenotype diagnosis of these variants. The sensitivity of stress test was 0.93; the specificity was 0.89, and the positive predictive value was 0.90.

Influence of sleep deprivation on cardiovascular parameters: a study with non-invasive methods for autonomic regulation assessment

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Background: Sleep deprivation has a significant influence on cardiovascular parameters through the autonomic regulation. Side effects of sleep deprivation on autonomic regulation and cardiovascular functions may have important clinical implications. It is well known that sleep deprivation increases sympathetic nervous system activity. The increased activity is considered to be a pathophysiological factor of cardiovascular and autonomic disturbances. The aim of the study was to test a hypothesis that the effects of sleep deprivation on cardiovascular functions can be tested using non-invasive electrophysiological methods.
Materials and methods: The study was performed on 19 healthy men (none on medication), any factors that might have influenced measured parameters were strictly controlled during whole experiment; (mean ± SD) age (30.5 ± 5.4 years); height (1.8 ± 0.0 m); weight (83.2 ± 11.1 kg); BMI (24.5 ± 2.4 kg/m^2); BSA (2.0 ± 0.1 m^2); basal SBP (116.8 ± 6.4 mm Hg); basal DBP (73.1 ± 5.7 mm Hg). Cardiovascular parameters and baroreceptors sensitivity were measured with non-invasive techniques implemented in Task Force Monitor system. Measurements were obtained twice, once after a normal sleep, and again after 32hours of sleep deprivation.
Results: In comparison with normal sleep, sleep deprivation resulted in an increase in: heart rate — HR (normal sleep *vs*. sleep deprivation = 53.4 ± 8.1 *vs*. 58.1 ± 7.9 1/min, p = 0.05); systolic blood pressure — SBP (normal sleep *vs*. sleep deprivation = = 116.8 ± 6.4 *vs*. 121.7 ± 6.5 mm Hg, p = 0.05); cardiac output — CO (normal sleep *vs*. sleep deprivation = 6.3 ± 1.2 *vs*. 6.9 ± 1.1 l/min, p < 0.05) and a decrease in baroreceptors reactivity — Slope (normal sleep *vs*. sleep deprivation = 32.6 ± 12.3 *vs*. 25.0 ± ± 12.5 ms/mm Hg, p < 0.05). Diastolic blood pressure, stroke volume and total peripheral resistance were not significantly changed by sleep deprivation. Sleep deprivation causes an increase in heart rate and systolic blood pressure and a decrease in baroreceptors reactivity and no changes in stroke volume. Thus, sleep deprivation results in an increase of sympathetic nervous activity which has an influence on cardiovascular functions in healthy subjects.
Conclusions: We conclude that applied non-invasive methods are a useful tool to investigate a hemodynamic state in different physiological conditions.

Individually adjusted standard torso model for solving the inverse problem of electrocardiology

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Background: For precise inverse problem solution in electrocardiology, an accurate realistic torso model is desirable. The possibility of using an individually adjusted parametrized standard torso model was examined in a simulation study.
Materials and methods: 12 small subendocardial and subepicardial ischemic lesions were modeled in left ventricular myocardium close to main coronary arteries and surface ECG potentials were simulated in 3 inhomogeneous torso models with electrode placement obtained from patient MRI. From simulated body surface potentials, the positions of lesions were then sought using an inverse solution to a single dipole. In the inverse computations, 4 types of torso models were tested for each of the 3 cases: (a) original torso with electrodes from MRI, (b) Dalhousie standard torso model with regularly placed electrodes, (c) standard torso model with dimensions adjusted for each case and regularly placed electrodes, (d) adjusted standard torso model with electrodes shifted as close as possible to real electrode pos-

itons. Distance between the center of the modeled lesion and inversely calculated dipole position was evaluated as the lesion localization error (LE).
Results: In all 3 cases, best results in inverse localization of the lesions were obtained with the original torso models from MRI (mean LE 0.52 ± 0.06 cm) and the second best results were achieved for adjusted torso with properly shifted electrodes (mean LE 0.74 ± 0.24 cm). Results on other positions depended on the case; the worst result in one case was achieved with torso model (b) (LE 2.88 cm) and in two cases with torso model (c) (LEs 1.31 cm and 1.80 cm).
Conclusions: Using a properly adjusted standard torso model instead of realistic chest geometry can give acceptable inverse solution if dimensions of the standard torso are properly adjusted in accordance with patient's chest dimensions and actual elelectrode positions are considered.

"Net QRS area" of lead aVR: An index of all six limb leads with potential utility for the follow-up of patients with heart failure

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Background: Modern ECG machines measure only leads I and II and calculate on line the other 4 limb leads. Lead aVR could be used as an index of all 6 limb leads, employing it in serial ECGs for the monitoring of patients with edematous states, including heart failure (HF), considering the equation aVR = – (I + II)/2, as reported recently based on the zenith to nadir amplitude(s) [Amp(s)] (PACE 2009; 32: 1567–1576)].
Materials and methods: Using a consecutive series of 1,784 ECGs, the Amp, "net QRS area", "absolute QRS area", and root mean square (RMS) of lead aVR were correlated with the corresponding parameters of the sums of all 6 limb leads (Σ6limbleads). Two analyses of the "net QRS areas" were carried out, one with Σ6limbleads considering the algebraic sign (Σ6limbleads "net QRS area"-C), and one ignoring it (Σ6limbleads "net QRS area"-I).
Results:

Variables		r
aVR "net QRS area"	*vs*. Σ6limbleads "net QRS area"-I	0.61
aVR Amp	*vs*. Σ6limbleads Amp	0.62
aVR RMS	*vs*. Σ6limbleads-RMS	0.67
aVR "absolute QRS area"	*vs*. Σ6limbleads "absolute QRS area"	0.70
aVR "net QRS area"	*vs*. Σ6limbleads "net QRS area"-C	0.78

P < 0.0001, for all above correlations.

Conclusions: The aVR "net QRS area" and Σ6limbleads "net QRS area"-C showed the best correlation, and since it is provided in many contemporary ECG management systems, it may be used as an index of Σ6limbleads "net QRS area"-C, for monitoring of patients with HF.

Fragmented ECG in Chagas' cardiomyopathy (FECHA Study)

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Background: Implantable cardioverter defibrillators (ICD) are proven to be an effective therapy to prevent sudden death in patients with chronic Chagasic cardiomyopathy (CChC). Identification of predictors of appropriate therapies delivered by ICDs remains a challenge. The aim of this study is to determine whether fragmentation on surface ECG helps in identifying patients with CChC and ICDs who are at higher risk of receiving appropriate ICD therapies.
Materials and methods: Retrospective study involving 14 centers in Latin America. All patients with CChC and ICDs were analyzed. Pacing-dependent patients were excluded. Clinical demographics, surface ECG and ICD therapies were collected. Bivariate and multivariate analyses were performed.
Results: A total of 98 patients from 14 Latin American centers were analyzed. Four cases were excluded due to pacing dependency. Males accounted for 63.8% of patients, mean age was 55.4 ± ± 10.4 years (26–75), mean LVEF was 39.6 ± 11.8%. Secondary prevention was the indication for implanting in 71.3% of cases. Fragmented surface ECG was found in 56 patients (59.6%). The location of fragmentation was inferior in 57.1%, lateral in 35.7% and anterior in 44.6%. Rsr pattern was the most prevalent (57.1%). Predictors of appropriate therapy in the multivariate model included: increased age (p = 0.01), secondary prevention indication (p = 0.01) and ventricular pacing > 50% of the time (p = 0.004). Male gender showed a positive trend (p = 0.07). The presence of surface ECG fragmentation did not identify patients at higher risk of receiving appropriate therapies delivered by the ICD (p = 0.87), regardless of QRS interval duration.
Conclusions: Fragmented surface ECG is highly prevalent among patients with CChC. It is a poor predictor of appropriate therapies delivered by ICDs in this population.

Repetitive monomorphic ventricular tachycardia triggered by fever in a patient with Brugada syndrome

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Background: Repetitive monomorphic ventricular tachycardia (MVT) occurring in the Brugada syndrome (BrS) is a potentially lethal event. Repetitive MVT triggered by fever has only been described in a few cases.
Results: A 39 year-old male with BrS (syncope, type 1 ECG pattern, negative genetics), was implanted with an ICD two years

ago. He was admitted with lightheadedness, fever and irritative lower urinary tract symptoms. Temperature: 39.2°C; HR: 110 bpm, irregular; BP 90/60 mm Hg. Laboratory investigations were unremarkable. In hospital, he experienced an episode of presyncope and the ECG showed a type 1 Brugada pattern and MVT with left bundle branch block and extreme left axis. He was treated with ice water packs, antipyretics and isoproterenol. ICD therapies were turned off. The patient experienced a total of 27 episodes of non-sustained MVT that abated when his body temperature normalized.

Conclusions: While an array of electrophysiological disturbances have been described in the setting of BrS, episodes of MVT triggered by fever are rare. Temperature-dependent dysfunction of sodium channels has been previously demonstrated, both with SCN5A mutations and other familial diseases including epilepsy and erythromelalgia. Where the SCN5A defect has been specifically related to late potentials and other phenomena in the RV outflow tract, this channel has been found throughout the myocardium. With a wide degree of variation in the location and sensitivity of sodium channels, there exists a possibility for a wide spectrum of electrophysiological dysfunction in BrS, of which fever-triggered MVT is a rare manifestation.

Early repolarization syndrome, Brugada syndrome or both?

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Background: Early repolarization syndrome (ERS) and Brugada syndrome (BrS) are two syndromes of abnormal ventricular repolarization. While BrS is well known for predisposing to life-threatening ventricular arrhythmias, it has only recently come to light that ERS, which was previously believed to be benign, may also have arrhythmogenic potential. These two syndromes share many common characteristics in terms of response to heart rate, pharmacologic agents and neuromodulation. Here, we present a patient with manifestations of both syndromes, raising the possibility that these two syndromes may have more in common than we had initially thought.

Results: A 20 year-old male athlete presented for assessment, complaining of two pre-syncopal episodes and one syncopal episode. His past medical history was unremarkable. His family history was positive for the unexplained sudden death of a paternal uncle at age 35. He denied consumption of any drugs. The physical examination was unremarkable. An echocardiogram was normal. Genetic testing for BrS was negative. His initial ECG showed sinus bradycardia. There was a J-wave with concave-up ST elevation in leads V4–6 and the inferior leads. These features are considered to be consistent with ERS. A second ECG, recorded 72 h later, showed sinus bradycardia with a type 1 Brugada ECG pattern in leads V1–3. Accentuation of the J-wave can be seen in the precordial and limb leads.

Conclusions: The appearances of both ERS and BrS in the same patient lends further strength to the notion that ERS may not be as benign as previously believed. The diagnostic value of the J-wave as a marker of arrhythmogenicity and the possibility of overlap between these syndromes is yet to be determined.

Maximal electrical separation (MES)-guided placement of right ventricular (RV) lead improves responders in cardiac resynchronization defibrillator therapy (CRT-D)

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Background: CRT-D is widely used for treatment of heart failure. Little is known about optimal placement of the RV lead. Recent data suggests electrical separation during LV pacing varies considerably within the RV. We hypothesized placement of the RV lead guided by MES would improve response to CRT compared with standard apical placement.

Materials and methods: Patients eligible for CRT-D were enrolled. LV lead placement was performed at the CS branch providing optimal parameters. The RVOT, septum and apex were mapped during LV pacing and MES recorded. At this point patients were randomized to receive either apical placement or at the site mapping MES. LV ejection fraction (EF), 6-minute walk distance (6MWD) and NYHA functional class (FC) were recorded at baseline and 3 months by blinded observers. Response (R) was defined as at least one of: an increase by > 1 FC; a 5% absolute increase in EF, or a 50 m increase in 6MWD. Primary endpoint was improvement in EF at 3 months.

Results: Of 52 patients recruited, follow-up is available in 43 (22 MES-guided and 21 apical). There was no significant difference in groups at baseline in terms of age, gender, NYHA FC, % ischemic etiology, QRS duration, baseline EF, 6MWD. Final RV lead position was septal in 20/22 and apical in 2/22 MES-guided patients. No RV lead was repositioned as a result of suboptimal defibrillation testing results. Main results are shown in the table. No dislodgement or adverse events were reported.

Conclusions: Significant improvements in EF and NYHA FC were observed in MES-guided patients compared to those undergoing standard apical positioning. Septal positioning of the RV ICD lead in CRT-D cases should be studied in larger trials.

	EF (delta)	Echo R	6MWD R	NYHA R	MES
MES {22}	7.5 ± 1.1	17	16	19	165.5 ± 22
Apical {21}	3.0 ± 1.8	8	11	10	152.1 ± 27
P	0.01	0.01	0.17	0.01	0.01

The right ventricular (RV) septum presents the optimum site for maximal electrical separation (MES) in biventricular pacing

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Background: Resynchronization therapy (CRT) benefits selected heart failure (HF) patients but the optimal placement of the RV lead has not been assessed. Greater physical separation between left (LV) and RV leads has been associated with better clinical outcomes. The relationship between electrical separation (ES) measured at implant and physical separation on CXR remains unexplored; moreover the site of MES in the RV is unknown.

Materials and methods: Prospective study of 38 CRT patients. The LV lead was placed in a postero-lateral branch of the coronary sinus. ES was recorded at three sites within the RV during LV pacing at 600 ms CL. The median ES was recorded with a roving deflectable catheter at the RV apex, mid septum and outflow tract (RVOT). Subsequent lateral CXR was used to calculate inter-lead distance and correlate with the ES recorded on final RV lead position.
Results: Mean age 67 ± 7 y/o, 28 male (73.7%). Twenty one had ischemic etiology (55.3%). Mean LVEF 25 ± 7%, six-minute walk test 384 ± 85 m, QRS duration pre and post 165 ± 26 ms and 138.5 ± ± 15.6 respectively ($p < 0.001$). Implant was successful in 37 (97%) patients, no immediate complications. MES was greater in the RV septum (174 ± 32 ms) compared with RVOT (155 ± 27 ms) and apex (148 ± 19 ms), $p = 0.01$. MES was most commonly found at the mid-septum and only rarely at the apex (3, 7.8%). Significant correlation between lateral inter-lead distance on CXR and final separation was observed ($r = 0.45$, $p = 0.006$).
Conclusions: MES was observed most commonly at the RV septum and rarely at the RV apex. Final ES measured at implant demonstrates correlation with physical distance on CXR. Septal placement may present the optimal placement of RV leads in CRT.

The simple heart examination and exercise test by means of a pocket-size trans-telephonic electrocardiograph for members of a sports club

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Background: The utility of pocket size trans-telephonic electrocardiograph (TTECG) for protecting cardiac events in a sports club was examined.
Materials and methods: In 6,703 applicants in the sports club (Musashi group in Japan), the ECG with the RA-V5 bipolar lead by TTECG (CG2100: wire-less non-loop memory type) was recorded as the screening test. Moreover, the V5R-V5 bipolar lead TTECG (CG6106: Loop-memory with wire type) was recorded for 124 applicants who were exercising by the ergometer in the sports club.
Results: Abnormal ECGs (Grade: G1–G5) were found in 353/6,703 (5.3%) with TTECG (CG2100). As a grade higher than G3, LVH with ST depression (5), atrial flutter (2), PSVT (1), SSS (1), OMI (1), WPW (1), LBBB (1), abnormal ST-T (22) and atrial fibrillation (10) in 33 cases (50 abnormal findings) were seen, including the dangerous example of 2 suggested by HCM and 1 of ST depression with frequent VPCs. On the other hand, TTECG (CG6106) of 124 people was able to be recorded easily as the exercise tolerance test by the ergometer. In 124 cases, 2 had significant increase of ST depression with no symptoms. Though VPC increased in 5 cases and disappeared in 4 cases, there was no relationship between the movement of VPCs and the ST-T changes.
Conclusions: It seems that TTECG is a simple method of screening and exercise test of the heart and might be useful in the prevention of heart events in a sports club.

Atrial fibrillation prevalence and treatment with oral anticoagulation in patients with permanent pacemakers

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Background: Atrial fibrillation (AF) is the most important risk factor for stroke and thromboembolic events (TE). The aim of this study is to determine the prevalence of AF among patients with permanent pacemakers (PPM), the percentage of anticoagulated patients and the prevalence on TE in this population. Secondary outcome was to determine the "level of knowledge" about indications of anticoagulation for AF patients.
Materials and methods: Descriptive and retrospective study on consecutive patients referred for PPM implantation. Cardiovascular risk factors, indications for pacing, prior history of AF, TE and anticoagulation indication were analyzed. In order to determine possible causes for not indicating anticoagulation, an electronic survey was sent to all doctors who usually refer patients for PPM implant and follow-up to our clinic.
Results: Among 934 patients, 26% (244) presented AF of which 34% were anticoagulated. 77.3% presented a CHADS2 score of ≥ 2 while only 2% had absolute contraindication for anticoagulation. The prevalence of TE was 9%. More than 60% of the doctors answered the survey. More than 40% acknowledged the CHADS2 score but only 33% were able to recognize all variables included in the score and 23% were able to determine when to indicate anticoagulation properly.
Conclusions: A low anticoagulation rate was detected among patients with AF and PPM with a high prevalence of TE and stroke. An extremely low adherence to international guidelines was detected among doctors who usually deal with this sort of patient.

Atrioventricular nodal reentrant tachycardia (AVNRT) radiofrequency (RF) ablation: Long--term outcome and results of a simplified technique

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Background: The ablation of the slow AVN pathway in AVNRT is successful as therapy, with very good outcomes. The aim of this study is to describe the long term outcome of RF in patients with AVNRT using a simplified technique and short applications of radiofrequency.
Materials and methods: Retrospective study involving patients with diagnosed SVT caused by AVNRT. Only two catheters were used for the EPS. AVN evaluation was carried out taking as references the HRA recordings and the beginning of the QRS complex in lead II (S2-QRS; S3-QRS), proving dual AVN physiology in case a jump > 50 ms of the S3-QRS were observed in respect of the previous one. Decremental atrial extrastimulation of 10 ms was used. The AVNRT mechanism was proved by presence of dual AVN physiology, atrial echoes, AVNRT induction with 1:1 AV relationship or VA interval of less than 60 ms. His recording was taken as reference of the Koch's triangle and RF lesions were applied between the medioseptal region and the CS os. RF settings: 50 W and 60°C. RF attempts of 10 s were used, considered effective when junctional rhythm were obtained. A total of 5 effective attempts was the goal. Successful procedure was considered the lack of AVNRT induction under basal and isoproterenol infusion conditions.
Results: A total of 344 patients were included, mean age: 49 ± ± 16 years (7–86); 74% women. Follow-up period: 21 ± 13 months (3–36); initial success rate of 100% with 1.45% of recurrent cases. No major complication was observed. Mean procedure time: 29.45 ± 9.6 min (18–70); mean fluoroscopy time: 10.87 ± 2.36 min (6–20); mean RF attempts: 7.79 ± 2.23 (5–14); mean RF effec-

tive attempts: 4.63 ± 0.62 (3–6); mean RF effective attempts time: 55 ± 8.03 s (40–90).
Conclusions: Slow pathway RF ablation using a simplified technique and short RF attempts, is safe, fast and effective, with excellent outcomes sustained over time.

Follow up of patients with implantable automatic cardioverter defibrillators without defibrillation threshold testing

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Background: Despite intraoperative defibrillation threshold testing (DFT) of implantable cardioverter defibrillators (ICD) being considered standard practice, ICD implants with no DFT have not been systematically evaluated yet. The aim of this study is to assess clinical outcomes of patients who underwent ICD implantation without performing (DFT) in a mid-term follow-up.
Materials and methods: Retrospective study involving patients who underwent ICD implantation without DFT. Sensing parameters, pacing threshold, and integrity of the system with sub-threshold pulses were tested at implant and at 7 and 30 days and then every 3 months. Clinical outcomes including appropriate and inappropriate therapies, lead dislodgement and ICD failure were evaluated during the follow-up.
Results: A total of 216 patients underwent ICD implantation without DFT. During a follow-up of 34 ± 22 months (3–86); 66 (30.55%) patients presented 199 episodes of ventricular arrhythmia. Almost 45% of the episodes were self limited and required no therapy. Antitachycardia pacing successfully terminated the arrhythmia in 52 episodes. A first shock of 20 Joules (5 episodes) and > 30 Joules (38 episodes) correctly treated ventricular arrhythmia. Inappropriate therapy was seen in 6% of the cases. Implant parameters: P wave: 3 ± 1 mV; R wave: 13 ± 4 mV; VD threshold: 0.7 ± 0.3 V; VD impedance: 748 ± 236 Ω; defibrillation integrity system: 48 ± 5 Ω. These values were stable during follow-up. Total mortality during follow-up was 6.8% and none of the deaths were sudden or related to device failure.
Conclusions: ICD implantation without DFT was safe and feasible. No additional complications compared to standard practice were seen. Randomized control trials are needed to confirm this observation.

Reverse atrial electrical remodeling induced by CPAP in patients with severe obstructive sleep apnea

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Background: Obstructive sleep apnea (OSA) has been associated with atrial enlargement in response to high arterial and pulmonary pressures and increased sympathetic tone. Continuous positive airway pressure (CPAP) is the gold standard treatment for OSA although its impact on atrial electrical remodeling has not been investigated. Signal average P-wave (SAPW) is a non-invasive quantitative method to determine P-wave duration, an accepted marker for atrial electrical remodeling. The aim of this study is to determine whether CPAP induces reverse atrial electrical remodeling in patients with severe OSA.
Materials and methods: Prospective study in consecutive patients attending the Sleep Clinic at Kingston General Hospital. All patients underwent full polysomnography. Severe OSA was defined as apnea-hypopnea index (AHI) ≥ 30 events/hour. SAPW was determined pre- and post-intervention with CPAP. Patients were treated with CPAP for a period of 4–6 weeks (titrated during the polysomnography study).
Results: A total of 16 patients were included in the analysis. Twelve were male, mean age 50.3 ± 11.9 years, mean BMI 34.4 ± ± 4.1 kg/m^2, 31.3% were hypertensive, 19% were smokers, and no patient presented with structural heart disease. Mean AHI was 40.8 ± 12.4 events/hour (range 30–60), minimum O_2 saturation 80.3 ± 7.1%. Mean SAPW duration pre-CPAP was 131.4 ± ± 8.6 ms and post-CPAP was 127.9 ± 8 ms ($p < 0.001$).
Conclusions: CPAP induced reverse atrial electrical remodeling in patients with severe OSA as represented by a significant reduction in P-wave duration.

Effect of exercise and emotional stress tests on ventricular arrhythmia

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Background: There is some information in literature about a unidirectional effect of exercise training testing (ETT) and an emotional Stroop Color and Word Test (SCWT) which induces myocardial ischemia in some subjects with coronary artery disease. The aim of our study was to examine the dynamics of ventricular arrhythmias (VA) with ETT and SCWT.
Materials and methods: 15 patients, mean age 41.7 ± 17.4 years, with a noncoronary VA of high grades: single ventricular ectopic complexes were 8,321.6 daily in all patients, predominantly daylong type of arrhythmia. Unstable ventricular tachycardia was in 5 patients (33.3%). A treadmill test was performed according to standard Bruce protocol to reach a submaximal heart rate (HR). SCWT was used to simulate neurophysiologic processes which occurred during emotional stress.
Results: During both tests, a response of sinus rhythm was observed: in ETT initially HR = 85.1 ± 6.2 bpm and HR max = = 162.3 ± 14.6 bpm, in SCWT initially HR = 68 ± 9.7 bpm and HR max = 84.9 ± 16.5 bpm ($p < 0.005$). Reaction of BP was normotensive: in ETT BP = 122.3/81.2 mm Hg, BP max = 157.7/ /88 mm Hg, in SCWT BP = 122.8/77 mm Hg and BP max = 131/ /78.6 mm Hg. Behavior VA during the tests: VA progressed in ETT and SCWT in 21.4% and 35.7%, resp. ($p > 0.005$), did not change in 7.1% and 50.0%, resp. ($p < 0.005$) and decreased or disappeared in 71.5% and 14.3%, resp. ($p < 0.005$). So, SCWT had no effect on VA in half of the group, and helped to identify it in one third of the cases, whereas ETT on the contrary caused VA decrease or disappearance (71.5%) more often. Such dependence is explained by significant differences in heart rate at the peak of tests, and different mechanisms of regulation during stress and ETT.
Conclusions: Both tests provoked arrhythmia in a small number of patients, however SCWT helped to identify VA in more cases than ETT.

Ventricular electrical activation after percutaneous mitral balloon valvoplasty: The significance of body surface potential mapping

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Background: Percutaneous mitral balloon valvoplasty (PMBV) is indicated for symptomatic patients with severe mitral stenosis (MS) and favorable valvar morphology. Body surface potential mapping (BSPM) was used to characterize noninvasively the pattern of ventricular electrical activation before and after PMBV.
Materials and methods: BSPM was performed in 31 patients with rheumatic MS before PMBV, and at 1-day, 1/3/6 months from the procedure. Data was compared to 20 healthy controls. Global and regional (RV, septum and LV) ventricular activation times (VATs), and also differences between regional RV-LV, Septum--RV and Septum-LV VATs were analyzed. Nonparametric T test, Kruskal-Wallis and Fisher statistics were used, with $p < 0.05$.
Results: MS patients were younger (39 ± 12 *vs*. 56 ± 15 years, $p < 0.0001$), and predominantly female (90% *vs*. 45%, $p = 0.0008$). PMBV was successful: mitral valve area 1.05 ± 0.22 *vs.* 1.76 ± ± 0.31 cm^2, $p < 0.0001$, pulmonary artery pressure 40.3 ± 10.7 *vs*. 32.7 ± 6.9 mm Hg, respectively after and before PMBV, $p = = 0.0058$. Analysis of ventricular electrical activation showed significantly greater global/regional VATs ($p < 0.0001$), and shorter regional VAT differences in MS patients before PMBV compared with controls, (RV-LV = $p < 0.0001$; Septum-RV = $p < 0.1073$; Septum-LV = $p < 0.0053$). However, even at 6 months from PMBV, no changes were found in ventricular electrical activation.
Conclusions: To the best of our knowledge, this was the first study analyzing ventricular activation after PMBV in patients with severe MS. However, although PMBV showed hemodynamic/ /clinical improvement, at 6 months patients did not show any cardiac electrical reverse remodeling.

Compensatory properties of heart rate asymmetry

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Background: Heart rate asymmetry (HRA) is a physiological phenomenon by which the contributions from decelerations to short-time heart rate variability (HRV) is greater than that of accelerations, and the contributions from accelerations to long--time HRV is greater than that of decelerations. This suggests the existence of some compensation between short- and long--term HRA. The aim of this study is to find the frequency of the compensation mechanism of HRA in healthy people at rest.
Materials and methods: The studied group consisted of short, 30 min resting ECG recordings from 420 healthy subjects (aged 20–40 years; 136 men) belonging to the control group of the Poznan-Team Study on predicting clinical events in patients with implanted cardiac defibrillating devices. For the quantification of short-term and long-term HRA, the contribution of heart rate decelerations to short-term HRV and long-term HRV were described by C1d and C2d, respectively. The short-term HRA was present if $C1d > 0.5$; long-term HRA was present if $C2d < 0.5$; whereas HRA compensation was defined when both conditions were satisfied i.e. $C1d < 0.5$ and $C2d > 0.5$. The same analyses were repeated for the same sets of RR intervals after shuffling their order. The binomial test was used to establish the existence of each type of HRA and HRA compensation.
Results: Short-term asymmetry was observed in 77.6% of subjects ($p < 0.0001$, binomial test), long-term asymmetry in 69.3% ($p < 0.0001$) and both types of HRA coexisted in 66.9% ($p < 0.0001$) of the whole group. In a group of individuals with at least one type of HRA i.e. $C1d > 0.5$ or $C2d < 0.5$, the compensation phenomenon was present in 83.6% ($p < 0.0001$).
Conclusions: The compensation phenomenon is present in short recordings of 30 min in approximately 67% of healthy people at rest. This phenomenon is related to the original structure of ECG and disappears after the order of RR intervals is completely random after shuffling.

Clinical utility of QT subintervals and QRS|T angle in acute coronary syndrome patients

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Background: Global repolarization time (RT) prolongation and wide mean QRS|T angle (Theta(QRS|T)) are considered as markers of abnormal repolarization with adverse prognostic implications. We evaluated repolarization in a reference group of 5,376 normal men and women and in 126 acute coronary syndrome (ACS) patients with, and 658 without, diagnostic ST elevation (STEMI and NSTEMI, respectively) using a recently developed repolarization model. RT estimates were derived for the epicardial and terminal RT (RTepi and RTterm), and for the initial and terminal QRS|T angles.
Materials and methods: The model uses as covariates rate-adjusted QT (QTa), QTpeak (QTpa) and (QTa–QTpa) intervals, and the obliques crossmural RT gradient (ΔRTxm) = Tp-Tx interval from Tp to the inflection point at the descending limb of the global T wave (Tx) and Theta(T,Tref), the deviation angle from the direction of repolarization in the normal group. RTepi = = QTpa if CosTheta(T|Tref) > 0, otherwise RTepi = QTpa + + ABS(CosTheta(T,Tref))*ΔRTxm.
Results: Theta(QRS|T) in the reference group was rate-invariant but 10% of the observed Theta(QRS|T) in STEMI and NSTEMI was due to heart rate (HR) with apparent sympathetic overdrive. QT subintervals and initial and terminal Theta(QRS|T) differed substantially in STEMI and NSTEMI groups from the reference group and there were large differences in initial and terminal Theta(QRS|T) in all subgroups. Gender differences and differences between STEMI and NSTEMI in QT subintervals and QRS|T angles were significant ($p < 0.001$ for all) in men and in women.
Conclusions: There are large differences in QT subintervals between the reference and MI groups not revealed by the global QT due to prolongation and shortening in opposite directions and drastic differences in Theta(QRS|T) between initial and terminal repolarization periods not evident from the mean Theta(QRS|T).

Cost benefit of a telecardiology service in the state of Minas Gerais: Minas Telecardio project

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Background: Telecardiology is a tool that can aid in cardiovascular care, especially in remote areas. Economic evaluations on this subject are scarce and with controversial results. The aim of the study was to evaluate the cost-benefit of implementing a telecardiology service in remote, small towns of the state of Minas Gerais, Brazil.

Materials and methods: The study utilized the database from the Minas Telecardio (MTC) Project, developed from June 2006 to November 2008, in 82 towns in the hinterland of the state. The towns were selected considering the population (up to 10,500 inhabitants) and the healthcare service coverage rate by public primary care program > 70%. Each municipality received a microcomputer with digital electrocardiograph, with the possibility of transmission of ECG tracings and the communication with duty of cardiology at the University hospital. The cost-benefit analysis was performed comparing the cost of performing an ECG in the project versus the cost of performing such examination by referral to another city. All values were adjusted for the date of June 15, 2008, considering a currency exchange rate of 1.63 Brazilian reais for each USA dollar.

Results: During the 30-month period of the study, a total of 62,865 electrocardiograms were carried out in 42,664 patients. The average cost of an ECG in the MTC project was R$ 17.74, broken down into R$ 4.96 referring to the cost of implementation and R$ 12.78 to maintenance. The simulation cost of the ECG by referral ranged from R$ 18.96 to R$ 33.48, with cost-benefit relation always favoring the project, independently of the mode of the distance calculation and considering both the point-of-view of the financier and of society. The sensitivity analysis with variations of calibration parameters confirmed these results.

Conclusions: The implementation of a telecardiology system in support of primary care in small Brazilian towns is feasible and economically beneficial, and may be transformed into a regular program of the public health system.

Exercise training slows down heart rate and improves deceleration and acceleration capacity in heart failure patients

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Background: The phase rectified signal averaging applied to RR intervals gives two variables, deceleration capacity (DC) and acceleration capacity (AC), both of which are predictors of total mortality in post-infarction patients. Although DC and AC are heart rate variability (HRV) measures and reflect autonomic modulation of heart rate, they have not been studied in patients with congestive heart failure (CHF). Cardiac rehabilitation in CHF patients improves autonomic control and reduces mortality. In this study we evaluated the effects of cardiac rehabilitation on mean RR interval, DC, AC and SDNN measuring total HRV in CHF patients.

Materials and methods: We enrolled twenty patients with CHF and left ventricular ejection fraction of no more than 40%, who were treated with optimal pharmacotherapy. Ten patients (training group) took part in a 24-week cardiac rehabilitation program with physical training planned 3 times a week. The other ten, age and gender matched, CHF patients (control group) were followed up for 24 weeks with no participation in cardiac rehabilitation. Mean RR interval, SDNN, DC and AC were calculated in resting 12-minute electrocardiogram recorded at the study entry and the end of follow-up.

Results: In the control group there were no significant changes in mean RR intervals (930.7 ± 133.9 *vs*. 862.8 ± 147.1 ms), SDNN (34.6 ± 18.8 *vs*. 26.6 ± 13.6 ms), DC (8.6 ± 4.1 *vs*. 6.2 ± 4.1 ms) and AC (–8.3 ± 3.5 *vs*. –5.9 ± 3.7 ms). In the training group there was a significant prolongation of mean resting RR interval (988.6 ± ± 148.2 *vs*. 1,088.0 ± 155.8; p = 0.0371), increase of DC (7.0 ± ± 3.3 *vs*. 10.4 ± 5.7 ms; p = 0.0488) and reduction in AC (–6.8 ± ± 3.4 *vs*. –9.7 ± 4.8 ms; p = 0.0273) but no change in SDNN (34.9 ± 18.4 *vs*. 38.7 ± 20.2 ms).

Conclusions: The applied 24-week cardiac rehabilitation program slowed down heart rate and improved DC and AC values with no influence on SDNN in CHF patients. Further prospective studies are needed to evaluate the clinical value of these findings.

Index of homogeneity of ventricular electrical activation: New concept on the ventricular depolarization phenomenon

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Background: The electrical phenomenon of ventricular contraction takes 40–90 ms in normal individuals, its onset at the RV, then the septum, ending by LV activation. Noninvasive characterization of global and regional (RV, septum and LV) electrical activation, using body surface potential mapping (BSPM), led to definition of an Index of Homogeneity (IH) of ventricular electrical activation, with the reasoning that the smaller the interval, the more homogeneous and uniform this activation will be.

Materials and methods: BSPM was performed in 20 normal individuals, 70 ± 14 years, 55% (11) male. Mean, minimum and maximal values of global and regional ventricular activation times (VAT) were automatically obtained, then IH was defined as the difference maximum-minimum VATs from the 87 BSPM leads (Global IH), and from leads related to RV, Septum and LV areas (Regional IHs). Since this index has not been reported yet, we studied healthy individuals to establish IH normal values (global and regional). Fisher, nonparametric T test, Kruskal-Wallis statistics were used, with p < 0.05.

Results: Ventricular electrical activation sequence in normal individuals was RV-Septum-LV as expected, with global VAT 45.4 ± ± 7.6 ms; and regional VATs respectively for RV: 35.2 ± 7.4 ms, Septum: 49.5 ± 8.5 ms, LV: 51.3 ± 9.0 ms. IH analysis of ventricular electrical activation resulted in Global IH: 72.6 ± 14.9 ms; and regional values: RV-IH 28.5 ± 19.7 ms, Septum-IH 25.4 ± 8.1 ms, and LV-IH 68.6 ± 15.5 ms. A regional LV-IH significantly greater than RV-IH (2.4×) and Septum-IH (2.7×), p < 0.0001 can be explained by the greater LV mass in relation to the other two areas.

Conclusions: The new Index of Homogeneity of ventricular electrical activation, in association with global and regional VAT values, as assessed by the BSPM, allowed a more detailed characterization of the ventricular electrical phenomenon in a normal population.

Critical times based activation time imaging

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Background: Methods for the non-invasive imaging of atrial activation times could provide valuable information on pathological excitation conduction patterns. In this study, the source representation functions used in the critical times method (Greensite et al. 1997) are expanded with a range adjustment to generate more accurate activation time maps from ECG measurements.
Materials and methods: Excitation conduction in the atria was simulated for various excitation origins. Body surface potential maps were obtained using a bidomain approach. The method of critical times can be used to quantitatively localize critical point locations and times, and to reconstruct surface activation in a qualitative manner (Greensite et al. 1995). To this end, all atrial surface nodes were treated as critical points and the corresponding critical times were reconstructed using the zero-crossing method, which is the subtraction of the two representation functions.
Results: For the heart surface nodes, it was observed that the minuend representation function in the zero-crossing term is often by magnitudes greater than the subtrahend. For the minuend to not dominate the subtrahend before the desired zero-crossing, which is supposed to occur at the time of depolarization, the minuend was therefore weighted with a sigmoid function and normalized to the range of the subtrahend.
Conclusions: Atrial activation times were reconstructed with both the new method and the method by Greensite. Two effects were observed. The overall reconstruction quality of the established method improves in the presence of 30 dB AWGN. This effect results from a gradual offset that is imposed on the reconstructed critical times under these circumstances (Huiskamp and Greensite 1997). Second, it could be shown that a significant reduction of reconstruction error can be achieved in the absence of noise with the sigmoid-weighted adaptation of the formula.With sigmoidal normalization, quality of reconstruction can be improved significantly if noise levels are below 30 dB.

QT/RR relationship in children in a population study "ECG screening of children and adolescents of the Russian Federation"

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Background: QT interval measurement has clinical importance for the electrocardiographic detection of phenomena associated with sudden cardiac death, syncope and severe arrhythmias. The study aims to analyze links of QT interval with RR interval, age, and sex in a large sample of healthy children.
Materials and methods: A representative sample of pediatric population was built according to the population volume and structure. The standard 12-lead ECG was recorded in 5,909 children aged 0–18 residing in the selected 14 regions of Russia. Subjects with previously known or screened underlying pathology were excluded from the study. 5,458 observations remained for further analyses. RR and QT intervals were measured in leads II and V5 in two from 5 to 10 consecutive cardio cycles. OLS regression was used to estimate relationship of the QT interval with RR interval, age, and sex. Bazett, Fridericia, Framingham and our own correction formulae were used for estimation of QT corrected by RR.
Results: A strong association between QT and RR that explains over 70% of QT variance out of 75.5% attributable to all explanatory variables was found. QT tends to increase with age. Only at ages 13+, QT is significantly associated with sex. In children, strong initial QT/RR correlation ($r = 0.84$) diminishes, but still remains significant after corrections with the Bazett ($r = –1.17$), Fridericia ($r = 0.39$), and Framingham formulae ($r = 0.44$). Two linear formulae significantly diminished QT/RR correlation in children. At ages 0 to 3 we apply [QTL1 = QT + 0.230 • (1–RR); $r = 0.0001$] and at age 4 to 17 years — [QTL2 = QT + 0.149 • (1––RR); $r = 0.0001$].
Conclusions: In normal pediatric population QT tends to increase with age, with RR being under control. QT of females exceeds that of males only beginning from age 13. Among all famous formulae for QT/RR correction, the Bazett's QTc showed the lowest correlation with RR. Linear formulae for improvement of the QT correction in children aged 0 to 3 and 4 to 17 are proposed.

Pediatric criteria for bradycardia and QTc limits established in a population study

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Background: This project "ECG screening of children and adolescents of the Russian Federation" was carried out in 2003–2008 in 14 randomly selected regions of the Russian Federation to identify the normal limits for ECG parameters in pediatric patients and to analyze their links with sex and physiological changes across age.
Materials and methods: A representative sample of pediatric population was built according to the population volume and age structure**.** The standard 12-lead ECG was recorded in 5,909 children aged 0–18 residing in the selected 14 regions. Prior to inclusion in the study, all subjects were screened for cardiovascular and/or any chronic abnormalities. Those subjects with previously known and/or screened underlying pathology were excluded from the study. Of the 5,909 accepted subjects, the data of a further 9% of subjects were excluded for the reasons of incomplete protocol (368), or newly identified underlying rhythm pathology (non-sinus rate or wandering pacemaker, WPW phenomenon, sinoatrial block, I–II degree AV-block, bundle branch block, supraventricular and ventricular extrasystole, atrial flutter, long QT syndrome [154]). The study results of 5,387 subjects were accepted (2,705 male and 2,682 female). Lower and upper limits of heart rate (HR), P wave duration, PQ and QRS intervals were defined as 2nd and 98th percentiles of their distribution. The equivalent limits of QT interval were defined as 5th and 95th percentiles of its distribution. Relationship between ECG characteristics and ECG parameters' dependency on age and sex were studied. A comparison of results with the most comprehensive studies of ECG variables in childhood was performed.
Results: The HR lower limits in bpm were identified as follows: 100 (age < 12 m), 80 (age 1–2), 70 (age 2–7), 60 (age 7–11), 55 (age 11–16), 50 (age 16+). The QTc interval in ms (Bazett's formula) ranged from 339–361 (5th percentile) to 449–465 (95th percentile). There was no significant effect of age and sex (age < < 13). There was a slight effect of sex at age above 13 years. The correlation of QT with HR was established at $r = –0.85$ ($p < < 0.002$) and the correlation of QTc with HR at $r = 0.13$ ($p < 0.05$).

Conclusions: The bradycardia limits appear to be lower than those found by Davignon A and Rijnbeek PR, especially at age ranges of 2m–9m, 1–3 and 10+ yrs. Presence of significant QTc--HR correlation questions the Bazett's formula as optimal method for QT over HR correction in children.

The influence of individual positioning of the heart on the inverse solution

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Background: The method for identification of small ischemic lesion by inverse solution to one dipole was used on well documented real data. The results obtained using standard torso model (STM) were compared to the results obtained for individually defined vertical position of the heart in the torso model (ITM).
Materials and methods: The inverse solution to one dipole was computed for the identification of ischemic lesion from the real data recorded in 117 leads during induced myocardial ischemia described in Horacek et al. J. Electrocardiol. Suppl. 2001/34. The inverse solution was computed from difference integral maps (IM) computed by subtraction of the IM measured during baseline activation from the IM at the ischemic state. The STM consisted of the Dalhousie torso model and the realistically shaped model of myocardial ventricles with cavities filled with blood. In ITM the vertical position of the heart septum was approximated for each observed subject individually as the position of an equivalent dipole inversely computed from the short interval after the Q wave onset. Both models were used in the inverse solution.
Results: From 14 cases with left anterior descending disease, we obtained the correct result on the anterior site of the heart model in STM only in 1 case, in ITM only in 2 cases, and in next 2 cases the result was located on the apex. Better results were obtained for the left circumflex disease. In STM or ITM, 10 or 12 results were correctly identified laterally on the left ventricle or in inferior septum, and 4 or 2 were identified on the anterior side. For STM 11 results for right coronary artery disease laid at the inferior side of septum and 3 were situated laterally, while for ITM all 14 results were correct.
Conclusions: Although the use of ITM increased the number of correctly located results in comparison with STM, it did not provide sufficiently reliable results of the inverse solution. Additional geometrical information is needed.

Cardiac impedance measurement predicts no reflow phenomenon of infarct-related coronary artery in patients with acute coronary syndrome with ST-segment elevation undergoing percutaneous coronary intervention

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Background: Patients with acute coronary syndrome with ST--segment elevation (STEMI) and no reflow phenomenon (TIMI flow grade < 3) are at risk of developing heart failure and increased mortality. We evaluated the predictive value of noninvasive hemodynamics measured before percutaneous coronary intervention (PCI) in STEMI patients for the no reflow phenomenon. Cardiac impedance, used for noninvasive measurement of hemodynamic variables in 158 consecutive patients with STEMI (age 35–81 years; 51 female), was started instantly after admission. The 10-minute means of stroke index (SI), heart rate (HR), left ventricular ejection time (LVET) and isovolumetric relaxation time (IVRT) measured just before PCI were used in statistical analysis.
Materials and methods: There were 22 patients with TIMI < 3. The mean value of SI was 40.7 ± 10.1 mL/m^2, HR 80.6 ± 16.2 bpm, LVET 289.0 ± 43.1 ms and IVRT 141.9 ± 55.6 ms. The Area Under Curve in the Receiver Operating Characteristics analysis was significantly different from 0.5 for SI (0.70; p = = 0.003), HR (0.71; p = 0.002), LVET (0.72; p = 0.001) and IVRT (0.69; p = 0.005). The optimal cut-offs for no reflow phenomenon were 37.7 mL/m^2 for SI, HR 83 bpm for HR, 270 ms for LVET and 110 ms for IVRT. In univariate logistic regression the odds ratios for the TIMI < 3 were as follows: 4.3 (95% confidence interval [CI] 1.6–11.3; p = 0.003) for reduced SI, 2.9 (95% CI 1.1–7.5; p = 0.035) for increased HR, 5.9 (95% CI 2.2–15.3; p < < 0.001) for shorter LVET and 4.8 (95% CI 1.9–12.4; p = 0.001) for shorter IVRT.
Conclusions: Patients with STEMI with reduced SI, LVET, IVRT and increased HR before PCI are at increased risk of no reflow phenomenon of infarct related artery after PCI. It seems that restoration of normal coronary blood flow during PCI is impaired in STEMI patients with impaired hemodynamic status at hospital admission. Cardiac impedance measurements appear to have a prognostic value for no reflow phenomenon of the infarct related artery in STEMI patients undergoing PCI.

Electromechanical coupling associated with long QT syndrome in children

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Background: The congenital long QT syndrome is a primary electrical disease characterized by QT interval prolongation. Genetic and electrocardiographic characteristics of LQTS are well described, but there is less evidence linking the syndrome with mechanical function and electromechanical interaction of the heart. The aim of the study is to evaluate the mechanical and electrical systolic function of the heart in young patients with LQTS.
Materials and methods: Echocardiography (ECHO, VIVID 7, GE) was performed in 52 LQTS patients (LQTS1 and LQTS2) aged from 4 to 19 years (12.3 ± 4.4 years) and 17 healthy controls matched by age (13.6 ± 3.4 years) and baseline heart rate. Electromechanical delay (Q-Ao, Q-Sm), electromechanical dissociation time (EMD) — interval from the aortic valve closure or the end of systolic wall motion to the end of QT interval and systolic time (Sm) were assessed by spectral (SpD) and tissue Doppler (TD) (in 12 segments of LV).
Results: In comparison with the control subjects, LQTS patients were characterized by the earlier start of mechanical systole (Q-Ao = 54.2 ± 12.5 *vs*. 67.3 ± 9.0 ms in control, p = 0.001; Q-S = = 58.1 ± 8.5 *vs*. 68.9 ± 11.1 ms in control, p = 0.0002), prolonged electromechanical dissociation time (EMD SpD = 98.4 ± 46.7 *vs*.

1.3 ± 8.4 ms in control, p = 0.0001; EMD TD = 94.6 ± 52.3 *vs*. 4.7 ± 14.4 ms in control, p = 0.0001). There were no significant differences in the duration of the systolic time between groups (Sm = 318.3 ± 22.5 *vs*. 307.9 ± 20.7 ms in control, p = 0.135).
Conclusions: In simultaneous analysis of ECHO and ECG parameters, LQTS patients exhibit pronounced features of electromechanical function i.e. accelerated coupling and electromechanical dissociation, that distinguish them from healthy children.

Improved electrocardiographic criteria for catheterization-proven myocardial infarction

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Background: The purpose of the study was to optimize the ability of the 12-lead electrocardiogram (ECG) to detect prior inferior myocardial infarction (IMI) and anterior myocardial infarction (AMI).
Materials and methods: We analyzed the digital ECG data from 1,138 patients (mean age = 53 years, 426 women) with suspected coronary artery disease who had undergone elective cardiac catheterization. Each ECG had been obtained 1 day before the catheterization. Evidence of the presence of IMI, AMI or neither was obtained by coronary angiography and left ventriculography. Using the digital ECG data, we analyzed a total of 135 combinations of Q, R and T durations (milliseconds) and amplitudes (microvolts) in Leads 2, 3 and aVF for possible IMI and 135 similar combinations in Leads V2, V3 and V4 for possible AMI. We used receiver-operating characteristic curves to determine the best ECG criteria for IMI and AMI and chi-square analysis to compare their performances in the same patients to each of 3 widely used commercial ECG diagnostic algorithms.
Results: There were 366 patients with prior IMI, 275 patients with prior AMI and 497 normals. For IMI, the best criterion was the algebraic sum of the Q and T amplitudes in Leads 3 and aVF. At 98% specificity, the respective sensitivities of the new criterion and the 3 commercial algorithms were 73%, 56%, 57% and 49%. For AMI, the best criterion was the algebraic sum of the Q, R and T amplitudes in Leads V2 and V4. At 98% specificity, the respective sensitivities of the new criterion and the 3 commercial algorithms were 74%, 61%, 50% and 19%. The superior performances of the new criteria for IMI and AMI were highly statistically significant.
Conclusions: Systematic statistical analysis of digital ECG data identifies diagnostic criteria for prior IMI and AMI whose performances are superior to those of 3 widely used commercial ECG diagnostic algorithms.

Hemodynamic and baroreflex responses to acute cold exposure (WBC) in healthy subjects

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Background: Whole-body cryotherapy (WBC) was introduced into clinical practice in the 1970s, essentially in Japan and Germany. It is primarily used to decrease inflammation and pain symptoms. In Poland, WBC is used widely in sports medicine and biological regeneration. WBC induces a rapid decrease of skin temperature and also a slight drop of body core temperature. Hemodynamic and baroreflex sensitivity changes are observed due to skin vasoconstriction. The aim of the present study was to determine the effect of acute cold exposure on the hemodynamic parameters and baroreflex reactivity.
Materials and methods: Twenty five healthy males (none on medication): (mean ± SD) age (31.5 ± 5.6 years); height (1.7 ± ± 0.05 m); weight (83.7 ± 10.1 kg); BMI (23.9 ± 2.8 kg/m^2); BSA (2.0 ± 0.1 m^2); basal SBP (121.2 ± 8.2 mm Hg); basal DBP (76.8 ± 6.5 mm Hg) were involved in the study. Each subject was exposed to 3 minutes WBC at –120°C and cardioimpedance techniques were used to analyze cardiovascular functions. All parameters were recorded with beat-to-beat method and baroreceptors reactivity was assessed with sequence method implemented in Task Force Monitor. Three examinations were performed: before/after/3 h after WBC.
Results: Mean values (mean ± SD) of hemodynamic parameters; before/after/3 h after WBC: RRI (R-R intervals) 985.0 ± ± 157.0/1,063.8 ± 172.5/953.1 ± 117.8 ms ($p < 0.05$); HR (heart rate) 62.8 ± 9.7/58.3 ± 10.1/64.7 ± 8.3 1/min ($p < 0.05$); SV (stroke volume) 100.2 ± 22.4/106.1 ± 21.7/101.0 ± 24.0 mL ($p < 0.05$); SI (stroke index) 49.3 ± 11.2/52.4 ± 11.6/49.94 ± ± 12.1 mL/m^2 ($p < 0.05$); CO (cardiac output) 6.2 ± 1.4/6.1 ± ± 1.3/6.4 ± 1.4 l/min ($p > 0.05$); CI (cardiac index) 3.0 ± 0.7/3.0 ± ± 0.6/3.1 ± 0.6 l/(min*m^2) ($p > 0.05$); TFC (thoracic fluid content) 34.4 ± 3.9/33.7 ± 4.0/34.7 ± 4.1 1/Ohm ($p < 0.05$); SBP (systolic blood pressure) 121.28 ± 8.2/122.3 ± 8.6/125.2 ± 9.2 mm Hg ($p < 0.05$); DBP (diastolic blood pressure) 76.8 ± 6.5/ /79.5 ± 8.3/78.2 ± 7.4 mm Hg ($p > 0.05$); TPR (total peripheral resistance) 1,210.8 ± 256.9/1,225.3 ± 253.5/1,199.3 ± 324.5 dyn*sec/cm^5 ($p > 0.05$); TAC (total arterial compliance) 2.2 ± ± 0.5/2.4 ± 0.5/2.1 ± 0.5 mL/mm Hg ($p < 0.05$); IC (index of contractility) 55.0 ± 16.11/57.9 ± 15.8/56.9 ± 17.9 1,000/s ($p >$ > 0.05); ACI (acceleration index) 78.0 ± 23.9/83.8 ± 26.9/80.6 ± ± 26.9 100/s^2 ($p > 0.05$); HI (Heather index) 0.3 ± 0.1/0.3 ± 0.1/ /0.3 ± 0.1 1/s^2 ($p > 0.05$); LVET (left ventricle ejection time) 311.0 ± 19.5/322.0 ± 20.19/305.6 ± 20.8 ms ($p < 0.05$); PEP (pre-ejection period) 114.8 ± 12.2/118.7 ± 10.2/111.8 ± 11.8 ms ($p < 0.05$); ER (ejection rate) 32.2 ± 3.6/31.0 ± 3.6/32.4 ± 2.8% ($p < 0.05$); Total Events Count 29.7 ± 15.8/19.0 ± 17.0/23.04 ± ± 16.3 n/1 ($p < 0.05$) Mean Slope 32.6 ± 20.4/44.6 ± 37.6/27.7 ± ± 17.0 ms/mm Hg ($p < 0.05$).
Conclusions: WBC causes significant changes in body hemodynamic state — increase of heart preload without changes of heart afterload and increase in baroreceptors sensitivity. No changes were observed in heart muscle contractility parameters. All changes were observed shortly after acute cold exposure; 3 hours after WBC, most parameters were back to their initial state.

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