# Study of Transthoracic Impedance Cardiogram for Assessment of Cardiac Hemodynamics in Atrial Fibrillation Patents

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***Abstract:*** *This study aims to test the usability of the transthoracic impedance cardiogram (ICG) for assessment of the quality of myocardial contractions in atrial fibrillation (AFIB) vs. sinus rhythm (SR), using signals recorded via defibrillation pads during external cardioversion (ECV). Data from 88 patients with persistent AFIB who received planned ECV are processed. AFIB is treated with cardioverter/defibrillator DG4000 (Schiller Médical, France) using a non-escalating protocol 200J/200J/200J. Successful ECV is defined as restoration of SR for >1min. The electrocardiogram (ECG), thoracic baseline impedance (Z) and dynamic impedance components dZ, dZ/dt captured via self-adhesive pads in antero-apical position are processed. Heartbeat contractions are evaluated by several measures extracted from the mean ICG patterns during systole: from dZ pattern – ICG (peak amplitude, range, area); from dZ/dt pattern – ICG velocity (peak, range, area) and left ventricular ejection time (LVET). The hemodynamical indices measured before and after ECV are: mean heart rate over 2 minutes (HR), standard deviation of HR (HRV), systolic (SysBP) and diastolic (DiaBP) blood pressure.*

*When the rhythm converts from AFIB to SR (74 patients), all measures on dZ, dZ/dt patterns significantly increase: dZ (64-102%), dZ/dt (31-67%), LVET (18%), $p < 0.05$. Significant decrease of HR (-36%), HRV (-53%), SysBP (-11%) and DiaBP (-19%) are also observed. Unsuccessful ECVs without conversion to SR (14 patients) are, however, associated with non-significant increase of dZ (10-21%), dZ/dt (0.3-29%), LVET (9%), $p > 0.05$ when comparing pre-shock AFIB vs. post-shock AFIB. No clear change in HR (-9%) and HRV (6%), and slight decrease of SysBP (-10%) and DiaBP (-8%) are observed.*

*The level of improvement of cardiac output quality in post-shock SR vs. pre-shock AFIB as estimated by ICG is related to a set of more than 60 clinical and hemodynamical parameters. Significant correlation coefficients are found to: Beta-Blocker (-0.25), Number of anti-arrhythmic drugs (-0.29), ΔST (0.37), pre-shock HR (0.43), ΔHR (-0.40), pre-shock HRV (0.30), ALT (0.46), ΔCK-MB (-0.32), ΔHR (-0.26), pre-shock DiaBP (0.24).*

***Keywords:*** *Impedance cardiography, ICG patterns, Hemodynamical status, Arrhythmia, Automated external defibrillators.*

## Introduction

The monitoring of cardiac hemodynamics could provide valuable additional information to the electrocardiogram (ECG) analysis for determination of the patient's clinical status. Impedance cardiography (ICG) is a technology that presents a cost-effective, noninvasive monitoring of hemodynamical parameters by applying a constant, low-amplitude, high-frequency current to the thorax and measuring the corresponding voltage to detect changes in thoracic electrical impedance. Recently, a number of reports on the clinical use of ICG are published. Authors have suggested that ICG measurements are useful for diagnosis of heart failure, monitoring of the patient clinical status, and assisting in medicine decisions [1]. The dynamic beat-to-beat changes in impedance are shown to be applicable for calculation of hemodynamical parameters, such as stroke volume and cardiac output [2, 3]. The common 4 electrode measurement of ICG is also shown to be a useful technique for monitoring the hemodynamic effect (stroke volume and cardiac output) of the electrical therapy in patients with atrial fibrillation or sinus bradicardia who restore sinus rhythm after cardioversion or stimulation [4]. The application of ICG acquired via defibrillation pads as an additional sensor for detection of pulseless electrical activity in defibrillators has been studied in [5].

The aim of this study is to test the usability of the transthoracic impedance cardiogram for providing information about the different quality of myocardial contraction in atrial fibrillation vs. sinus rhythm when using the signal recorded via the defibrillation pads during cardioversion.

## Materials

This study comprises clinical data from 88 patients admitted to receive elective external cardioversion (ECV) of persistent atrial fibrillation (AFIB) from May 2010 to March 2012 in the Intensive care unit, National Heart Hospital, Sofia. The ECV is following standard hospital procedures, all patients signing a written informed consent. The shocks are administered by a commercial cardioverter/defibrillator DG4000 (Schiller Médical, France) via standard self-adhesive defibrillation pads with active area ($2\times75cm^2$), placed in antero-apical position. AFIB is treated with a maximum of 3 biphasic shocks 200J/200J/200J, followed by a 360 J monophasic if required. Successful ECV is defined as a restoration of sinus rhythm (SR) for at least 1 minute, thus dividing the study population into successful group (*S*-group, 74 patients) and failure group (*F*-group, 14 patients). Clinical data related to patients' demographic information, diagnosis indicators, prehistory and supporting treatment are collected.

The ECV procedure is electronically recorded by a measurement device *Z*-Meter [6], storing signals captured via the defibrillation pads (sampling rate of 250 Hz, 12-bit resolution):

- ECG signal – acquired with a pass-range of (0.5-30) Hz;
- ICG signal – measured as the attenuation of a low-intensity, high-frequency current in the thoracic area, including the thoracic impedance baseline component ($Z$), the pulsatile component ($dZ$) in a pass-band of (1-45 Hz), and the first derivative ($dZ/dt$).

## Methods

The ECV interventions are retrospectively processed in Matlab 7.5 (MathWorks Inc.), extracting ECG and ICG strips in noise-free episodes (10 s to 2 min): *Episode 1* before ECV, *Episode 2* after the last ECV shock. Considering the *pre*- and *post-shock* patient rhythm, the extracted episodes are annotated as follows:

- *S*-group: 74 episodes with *pre-shock* AFIB + 74 episodes with *post-shock* SR;
- *F*-group: 14 episodes with *pre-shock* AFIB + 14 episodes with *post-shock* AFIB.

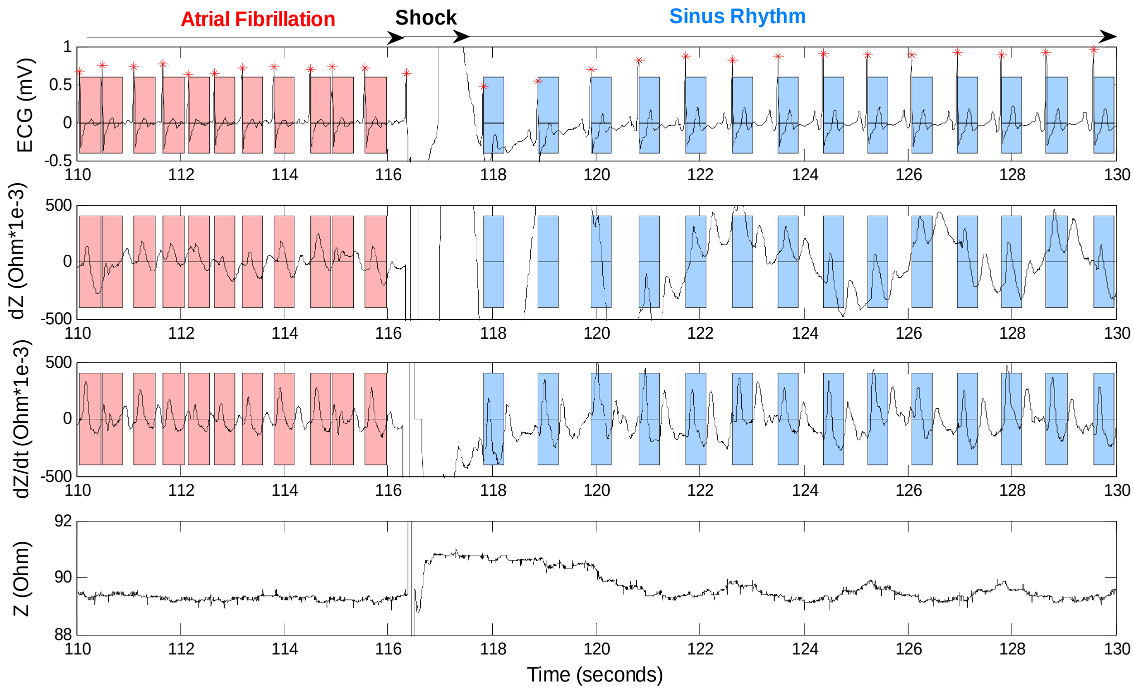



Fig. 1 Extract of 10-second segment, including an episode of *pre-shock* AFIB and the restored *post-shock* SR – ECG (1st trace), *dZ* (2nd trace), *dZ/dt* (3rd trace), baseline *Z* (4th trace). The R-peak reference points identified for all heartbeats are marked (‘*’). The defined ICG pattern windows during systole are depicted in red (AFIB) and blue (SR).

QRS detector is applied on the ECG channel to identify R-peaks as heartbeats’ reference points. ICG patterns (from *dZ* and *dZ/dt* signals) are then studied within a window of 500 ms after the reference points to analyse the impedance change, including the systole period. An example of recorded signals and identified patterns is shown in Fig. 1.

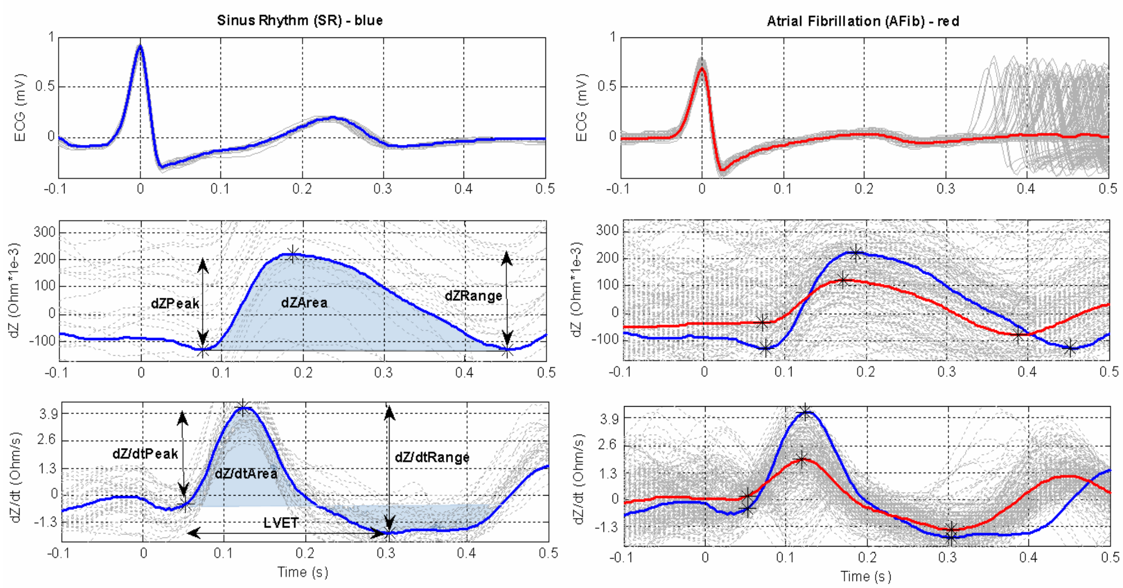
Signal averaging of all consecutive ICG patterns within one episode is applied to derive a mean ICG pattern with improved signal-to-noise ratio. By detection of extrema over the mean *dZ* and *dZ/dt* patterns, the following measures are defined to describe the ICG waveform during systole (see Fig. 2a):

- *dZPeak* – the ICG positive peak amplitude;
- *dZRange* – the ICG peak-to-peak range;
- *dZArea* – the ICG pattern area, accumulated during systole;
- *dZ/dtPeak* – the ICG peak velocity;
- *dZ/dtRange* – the ICG peak-to-peak velocity range;
- *dZ/dtArea* – the accumulated area under the ICG velocity curve during systole;
- *LVET* – left ventricular ejection time measured from *dZ/dt* pattern.

Two indexes are calculated to estimate the change of cardiac output quality in *post-shock* rhythms (SR for *S*-group, AFIB for *F*-group) compared to the *pre-shock* AFIB state, using the total impact of all *dZ*, *dZ/dt* measures:

$$Index(dZ) = \prod(dZPeak, dZRange, dZArea)\Big|_{Post-shock} \Big/ \prod(dZPeak, dZRange, dZArea)\Big|_{Pre-shock}$$

$$Index(dZ/dt) = \prod(dZ/dtPeak, dZ/dtRange, dZ/dtArea)\Big|_{Post-shock} \Big/ \prod(dZ/dtPeak, dZ/dtRange, dZ/dtArea)\Big|_{Pre-shock}$$




*a*) *b*)

Fig. 2 ECG, *dZ*, *dZ*/*dt* patterns for *post-shock* SR (*a*) compared to *pre-shock* AFIB (*b*) of the patient in Fig. 1. The mean patterns (bolded curves – blue (SR), red (AFIB)) are obtained after signal averaging of all superimposed patterns (gray dotted curves). The extrema used to identify *dZ*, *dZ*/*dt* measures are identified by '*'.

Cardiac hemodynamical parameters, measured before and after ECV, are defined as:

- *HR* as the mean heart rate over a 2 minutes period;
- *HRV* as the heart rate variability estimated by the standard deviation of the mean *HR*;
- *SysBP/DiaBP* as non-invasive measurements of systolic/diastolic blood pressure.

## Statistics

Statistical analysis is performed with the package Statistica 7.0 (StatSoft Inc.). Student's *t*-test is used to compare the means of all measures on *dZ* pattern, *dZ/dt* pattern, *Z*-baseline, *HR*, *HRV*, *SysBP* and *DiaBP* before and after ECV. The aim is to track significant *post-shock* changes in case of successful (*S*-group) and unsuccessful SR conversion (*F*-group), corresponding to improved and non-improved cardiac hemodynamics. The relative change of the *post-shock* means compared to the respective *pre-shock* means is also computed (denoted as Δ). A two-tailed *p*-value $< 0.05$ is considered statistically significant.

Linear regression models are used to study how patients' clinical data are influencing the level of improvement of cardiac output quality in *post-shock* SR vs. initial AFIB for the *S*-group, evaluated by *Index(dZ)* and *Index(dZ/dt)*.

## Results

Fig. 2b illustrates an example where the mean *dZ*, *dZ*/*dt* pattern waveforms in *pre-shock* AFIB are considerably suppressed in comparison to the *post-shock* patterns during SR.

In the *S*-group (74 patients) with successful SR restoration accompanied with improved *post-shock* cardiac hemodynamics, the comparison of *post-shock* SR vs. *pre-shock* AFIB (Table 1, 2$^{nd}$ column) reveals significant increase of all ICG measures (by 31 to 102%, $p < 0.05$), significant decrease of *HR*, *HRV*, *SysBP* and *DiaBP* (by 11 to 56%, $p < 0.05$), but with negligible drop of the baseline impedance *Z* (by 1%, $p > 0.05$).

Table 1. T-test for comparison of all measures on ICG patterns (*dZ*, *dZ*/*dt*), *Z*, and hemodynamical indices in case of: (1) *Pre-shock* AFIB vs. *post-shock* SR for the *S*-group; (2) *Pre-shock* AFIB vs. *post-shock* AFIB for the *F*-group. Delta of means (ΔSR/AFIB after successful ECV, ΔAFIB after non-successful ECV) is used to quantify the differences. **p* > 0.05: Non significant differences.

| | ***S*-group**, 74 patients | | | | ***F*-group**, 14 patients | | | |
|---|---|---|---|---|---|---|---|---|
| | ***Pre-shock*** **AFIB** mean±std | ***Post-shock*** **SR** mean±std | *p*-value | Δ SR/AFIB (%) | ***Pre-shock*** **AFIB** mean±std | ***Post-shock*** **AFIB** mean±std | *p*-value | Δ AFIB (%) |
| ***dZPeak***, [mΩ] | 136±92 | 245±108 | <0.001 | 80% | 98±40 | 108±41 | 0.94* | 10% |
| ***dZRange***, [mΩ] | 152±88 | 249±107 | <0.001 | 64% | 109±84 | 125±103 | 0.99* | 15% |
| ***dZArea***, [mΩ.s] | 3311±3211 | 6686±4704 | <0.001 | 102% | 1731±1027 | 2091±1398 | 0.44* | 21% |
| ***dZ/dtPeak***, [Ω/s] | 1.5±0.9 | 2.5±1.0 | <0.001 | 67% | 1.04±0.5 | 1.34±0.5 | 0.14* | 29% |
| ***dZ/dtRange*** [Ω/s] | 2.3±1.1 | 3.2±1.1 | <0.001 | 39% | 1.789±0.6 | 1.794±0.6 | 0.98* | 0.3% |
| ***dZ/dtArea***, [Ω] | 28.4±19.6 | 37.2±18.9 | 0.002 | 31% | 15.3±6.9 | 17.0±8.3 | 0.56* | 11% |
| ***LVET***, [ms] | 256±69 | 303±77 | <0.001 | 18% | 250±49 | 272±46 | 0.22* | 9% |
| ***Z***, [Ω/s] | 93±17 | 92±17 | 0.65* | -1% | 110±18 | 107±16 | 0.59* | -3% |
| ***HR***, [bpm] | 104±20 | 67±11 | <0.001 | -36% | 107±16 | 97±14 | 0.09* | -9% |
| ***HRV***, [bpm] | 19±5 | 9±7 | <0.001 | -53% | 18±5 | 19±6 | 0.42* | 6% |
| ***SysBP***, [mmHg] | 131±16 | 116±14 | <0.001 | -11% | 135±12 | 122±11 | 0.009 | -10% |
| ***DiaBP***, [mmHg] | 86±10 | 70±12 | <0.001 | -19% | 89±10 | 82±7 | 0.06* | -8% |

Table 2. T-test for comparison of the total ICG indexes for *S*-group vs. *F*-group

| | ***S*-group**, 74 patients mean±std | ***F*-group**, 14 patients mean±std | *p*-value |
|---|---|---|---|
| ***Index*(*dZ*)** | 9.5±22.1 | 0.66±0.13 | 0.14 |
| ***Index*(*dZ/dt*)** | 3.1±5.7 | 0.7±0.6 | 0.12 |

In the *F*-group (14 patients) with unsuccessful SR restoration associated with non-improved *post-shock* cardiac hemodynamics, the comparison of *post-shock* AFIB vs. *pre-shock* AFIB (Table 1, last column) reveals insignificant increase of all ICG measures (by 0.3 to 29%, $p > 0.05$), negligible decrease of the baseline *Z* (by 3%, $p < 0.05$), as well as the hemodynamical measures *HR*, *HRV*, *DiaBP* (by 6 to 9 %, $p > 0.05$), but *post-shock* reaction with significant decrease of *SysBP* (by 10%, $p < 0.05$).

The total ICG change (*post-shock* to *pre-shock*), estimated by *Index*(*dZ*/*dt*) and *Index*(*dZ*) is between 4 to 14-times larger in the *S*-group compared to the *F*-group (Table 2), although found to be non-significant, probably due to the small number of patients in the *F*-group and considerable deviations observed in the *S*-group.

The *S*-group is additionally studied in respect of the influence of patients' clinical data on the level of improvement of cardiac output quality in *post-shock* SR vs. *pre-shock* AFIB as estimated by ICG. Table 3 lists the correlation coefficients of *Index(dZ)* and *Index(dZ/dt)* in relation to a set of more than 60 clinical and hemodynamical parameters. The following significant dependencies (maximal correlations) are found:

- *Index*(*dZ*, *dZ*/*dt*) are correlated to: Beta-Blocker (-0.25), Number of anti-arrhythmic drugs (-0.29), Δ*ST* (0.37), *pre-shock HR* (0.43), Δ*HR* (-0.40), *pre-shock HRV* (0.30);
- Δ*LVET*: *ALT* (0.46), Δ*CK-MB* (-0.32), Δ*HR* (-0.26), *pre-shock DiaBP* (0.24).

Table 3. Correlation coefficients, presenting *Index(dZ), Index(dZ/dt)* in function of the patients' clinical data for 74 patients in the *S*-group. Significant correlations (*$p < 0.05$) indicate about *post-shock* vs. *pre-shock* change of ICG waveform related to the patient status.

| Clinical data | *Index dZ* | *Index dZ/dt* | Δ*LVET* SR/AFIB | Clinical data | *Index dZ* | *Index dZ/dt* | Δ*LVET* SR/AFIB |
|---|---|---|---|---|---|---|---|
| Age [yrs] | 0 | 0 | -0.05 | Previous ECV [y/n] | 0.21 | 0.19 | -0.03 |
| Gender | -0.01 | -0.07 | 0.06 | AFIB duration [days] | -0.17 | -0.11 | 0.02 |
| Weight [kg] | 0.17 | 0.17 | 0.17 | Arterial hypertension [y/n] | 0.16 | 0.21 | 0 |
| Height [cm] | 0.05 | 0.09 | 0.09 | Struct heart disease [y/n] | 0.12 | 0.16 | 0.09 |
| Body mass index BMI [kg/$m^2$] | 0.20 | 0.17 | 0.18 | Heart failure [y/n] | -0.07 | -0.12 | -0.17 |
| Body surface area BSA [$m^2$] | 0.12 | 0.13 | 0.06 | Diabetes mellitus [y/n] | -0.06 | -0.10 | 0.04 |
| Lean body weight LBW [kg] | 0.11 | 0.11 | 0.14 | Coronary artery disease [y/n] | -0.03 | -0.09 | -0.13 |
| Fat body weight FBW [kg] | 0.20 | 0.19 | 0.16 | Cardiomyopathy [y/n] | -0.09 | -0.11 | -0.08 |
| Chest circumference [cm] | 0.09 | 0.09 | 0.05 | Valvular heart disease [y/n] | -0.15 | -0.18 | -0.06 |
| Hemoglobin [g/l] | 0.04 | 0.09 | 0.16 | Chr. obstr. pulm. disease [y/n] | -0.08 | -0.7 | -0.09 |
| Hematocrit [g/l] | 0.03 | 0.07 | 0.16 | Pericarditis [y/n] | -0.05 | -0.03 | -0.11 |
| Glucose [mmol/l] | 0.01 | -0.01 | 0 | Thyroid dysfunction | 0.07 | 0.15 | 0.22 |
| Urea [mmol/l] | -0.10 | -0.10 | -0.04 | Beta-Blocker [y/n] | -0.25* | -0.23* | -0.19 |
| Creatinine [mmol/l] | 0.09 | -0.01 | 0.16 | Propafenone [y/n] | 0.01 | 0.02 | -0.05 |
| *WBC* [$10^9$/L] | -0.04 | -0.06 | 0.04 | Amiodarone [y/n] | -0.07 | -0.13 | -0.03 |
| *K* [mmol/l] | -0.07 | -0.08 | 0.02 | CCB [y/n] | -0.04 | -0.05 | -0.06 |
| *Na* [mmol/l] | 0.03 | 0.05 | 0.14 | Number Antiarrhytmic drugs | -0.29* | -0.29* | -0.21 |
| *AST* [U/L] | 0.06 | 0.06 | 0.02 | ACE inhibitor/ARB [y/n] | 0.06 | 0.06 | -0.07 |
| *ALT* [U/L] | -0.01 | 0 | 0.46* | Atropin before ECV [y/n] | -0.11 | -0.18 | -0.05 |
| Δ*CK* [umol/l] | -0.15 | -0.12 | -0.18 | Atropin after ECV [y/n] | -0.04 | -0.03 | -0.08 |
| Δ*CK-MB* [umol/l] | -0.06 | -0.18 | -0.32* | Dosis Propofol [mg/kg] | -0.01 | -0.14 | -0.22 |
| Δ*TnI* [umol/l] | -0.01 | -0.02 | -0.16 | Left atrial LA dimension [mm] | 0 | -0.07 | -0.13 |
| *ST0* [mm] | -0.07 | -0.04 | 0.02 | LV telesystolic dimension [mm] | 0.09 | 0.02 | 0.18 |
| Δ*ST* [mm] | 0.28* | 0.37* | 0 | LV telesdiastolic dimension [mm] | 0.14 | 0.11 | 0.19 |
| Number of shocks | -0.03 | -0.09 | -0.11 | LV telesystolic volume [ml] | 0.05 | -0.06 | -0.08 |
| Cumulative energy [J] | -0.03 | -0.09 | -0.10 | LV telediastolic volume [ml] | -0.03 | -0.13 | 0 |
| *Z*-baseline [Ω] | -0.03 | 0.01 | -0.04 | Ejection fraction EF [%] | -0.08 | -0.04 | 0.11 |
| **Hemodynamical parameters** | | | | | | | |
| *HR* (*pre-shock*) | 0.43* | 0.43* | 0.13 | *SysBP* (*pre-shock*) | 0.06 | 0.08 | 0.06 |
| *HR* (*post-shock*) | 0.10 | 0.06 | -0.09 | *SysBP* (*post-shock*) | 0.11 | 0.14 | 0.08 |
| Δ*HR* | -0.35* | -0.40* | -0.26* | Δ*SysBP* | -0.05 | -0.07 | -0.06 |
| *HRV* (*pre-shock*) | 0.29* | 0.30* | 0.17 | *DiaBP* (*pre-shock*) | 0.19 | 0.23 | 0.24* |
| *HRV* (*post-shock*) | -0.14 | -0.14 | -0.02 | *DiaBP* (*post-shock*) | 0.19 | 0.15 | 0.08 |
| Δ*HRV* | -0.19 | -0.21 | -0.12 | Δ*DiaBP* | 0.01 | -0.08 | -0.09 |

## Discussion and conclusion

The signal-averaging technique over ICG acquired via defibrillation pads is used for extraction of mean *dZ*, *dZ/dt* patterns with well-defined extrema during heart systole.

When the rhythm converts from initial AFIB to *post-shock* SR, all amplitude and time-related measures on *dZ* and *dZ/dt* patterns in Table 1 ($2^{nd}$ column) manifest significant increase: Δ*dZ* increases by 64-102%, Δ*dZ/dt* by 31-67%, Δ*LVET* by 18% ($p < 0.05$). On the contrary, for patients with initial AFIB vs. sustained *post-shock* AFIB (Table 1, last column), we observe a non-significant increase of Δ*dZ* by 10-21%, Δ*dZ/dt* by 0.3-29%, Δ*LVET* by 9% ($p > 0.05$).

These results could be paired to the observed rhythm and hemodynamics that is to say:

- When the rhythm converts from AFIB to SR, a significant decrease of *HR* and *HRV* (36% and 53% respectively) and a significant decrease of *SysBP* and *DiaBP*

(-11% and -19% respectively) is observed.

- When the rhythm does not convert from AFIB to SR, no clear change in *HR* and *HRV* (-9% and 6%) and a slight decrease of *SysBP* and *DiaBP* (resp. -10% and -8%) is observed.

Increase in *dZPeak*, *dZRange*, *dZArea* and *dZ/dtPeak*, *dZ/dtRange*, *dZ/dtArea*, *LVET* are related to an improvement of the patient's rhythm after delivery of an electric shock during ECV of AFIB. These parameters could be valuable descriptors of the myocardial contraction quality improvement in *post-shock* SR vs. initial AFIB, and could be used as markers of hemodynamics.

## References

1. Yancy C., W. T. Abraham (2003). Noninvasive Hemodynamic Monitoring in Heart Failure: Utilization of Impedance Cardiography, Congest Heart Fail, 9(5), 241-250.
2. Bayram M., C. Yancy (2009). Transthoracic Impedance Cardiography: A Noninvasive Method of Hemodynamic Assessment, Heart Failure Clinics, 5(2), 161-168.
3. Schultz M., R. Climie, S. Nikolic, K. Ahuja, J. Sharman (2012). Reproducibility of Cardiac Output Derived by Impedance Cardiography during Postural Changes and Exercise, Artery Research, 6, 78-84.
4. Palko T. (2004). Impedance-cardiography for the Assessment of Haemodynamic Effect of Electrocardiotheraphy, Polish J. Med. Phys. and Engineering, 10(4), 201-207.
5. Johnston P., Z. Imam, G. Dempsey, J. Anderson, A. Adgey (1998). The Transthoracic Impedance Cardiogram is a Potential Haemodynamic Sensor for an Automated External Defibrillator, European Heart J., 19, 1879-1888.
6. Mudrov Ts., V. Krasteva, I. Jekova, N. Mudrov, M. Matveev, T. Stoyanov (2010). Device for Data Collection during Cardioversion, Annual J. Electronics, 4(2), 142-145.

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