Deceleration Capacity Alterations before Non-Sustained Ventricular Tachycardia Episodes in Post Myocardial Infarction Patients

Petros Arsenos[1], George Manis[2], Stavros Nikolopoulos[3], Konstantinos Gatzoulis[1], Polychronis Dilaveris[1], Christodoulos Stefanadis[1]

[1] First Dept. of Cardiology, National Kapodistrian University of Athens, Greece
[2] Dept. of Computer Science and Engineering, University of Ioannina, Greece
[3] School of Electrical and Computer Eng., National Technical University of Athens, Greece

Abstract

While Non-Sustained Ventricular Tachycardia (NSVT) can be characterized as innocent in healthy persons, such arrhythmias in post-infarction patients can be associated with an increased risk for Arrhythmic Sudden Cardiac Death (SCD). The Autonomic Nervous System (ANS) may influence the electrical status before fatal arrhythmias initiations. In this work we study the differences between the behavior of Deceleration Capacity (DC) of heart rate before the onset of NSVT and in the rest of the signal. Twenty (20) patients having presented NSVT episodes are examined. Nine (9) of them have been classified as high risk for SCD after 16 months of follow up, while the rest (11) have been considered as low risk. For each NSVT episode the 30 min period before the episode and the 150 min period exactly before this period were compared. Windowed analysis was performed. Mean values showed that DC is reduced before NSVT episodes in both high and low risk patients. High risk patients presented lower mean values for DC compared to the low risk.

1. Introduction

Its approximately thirty years now since it was proposed that the dynamics of cardiac cycles may reveal hidden instabilities preceding the onset of arrhythmias episodes[1]. Since cardiac cycles are influenced from ANS activity, methods for the quantification of this activity were investigated during the past two decades [2]. Advances in heart rate analysis made possible the study of Heart Rate Variability (HRV) before ventricular arrhythmia episodes [3] something that can give us an impression of the status of ANS [4]. Furthermore, Implantable Cardioverter Defibrillators with adequate ECG storage function offered the opportunity of evaluating the heart rate dynamics exactly before the detected arrhythmic events [5].

Deceleration Capacity of heart rate [6] quantifies the inter-beat deceleration duration reflecting (at least partly) the Parasympathetic Nervous System (PNS) activity on sinus node. Experimental and clinical data indicate that PNS has a protective role for the heart against malignant arrhythmias [7]. Impaired PNS activity reflected through decreased DC values may be of prognostic value.

We study the differences between the behavior of Deceleration Capacity of heart rate before Non-Sustained Ventricular Tachycardia episodes and the rest of the signal in patients suffered myocardial infarction. We study two groups of subjects that presented such episodes, those (9 patients) characterized as high risk for Sudden Cardiac Death after 16 months of follow up and those (11 patients) considered as low risk. For each Non Sustained Ventricular Tachycardia episode, we compare the 30 min period before the episode and the 150 min period before the first period. We perform window analysis to study Deceleration Capacity for these periods. Mean values show that Deceleration Capacity is reduced before Non Sustained Ventricular Tachycardia episodes in both high and low risk patients.

The rest of the paper is structured as follows. Section 2 outlines the method for the computation of Deceleration Capacity and methodology proposed in this paper for studing the decrease of Deceleration Capacity before NSVT episodes. Section 3 describes the dataset we used for our experiments the results of which are presented in section 4. The last section summarizes the most important points of this work.

2. Methods

2.1. Deceleration capacity

A method for the computation of Deceleration Capacity has been proposed in [6]. *Anchor* points are defined as RR intervals which are longer than their preceding intervals. Intervals which differ more than 5% from their pre-

ceding interval are excluded from computation in order to avoid possible artifacts. The line segments around anchors points are used for the computation of Deceleration Capacity. We align those segments according to the anchors points (*phase rectification* [8][9]). Aligned segments are then averaged.

Deceleration Capacity is given by the following formula:

$$DC = \frac{X(0) + X(1) - X(-1) - X(-2)}{4} \quad (1)$$

where $X(O)$ is the average for all anchor points, $X(1)$ is the average of all points following an anchor point, $X(-1)$ the average of all points before an anchor point and $X(-2)$ the corresponding average values of the points being two points before an anchor point.

2.2. Proposed methodology

We used windowed analysis in order to study the differences of Deceleration Capacity 30 minutes before the episode and 150 minutes before this 30 minutes period. Thus, we broke 3 hours of the signal just before the episode into 36 overlapping windows. The size of each window was 20 minutes and the overlap between two successive windows 5 minutes.

Next, we computed Deceleration Capacity for each one of these windows. We computed the average values of Deceleration Capacity for both the above described periods. We compared the mean values and computed the p-values for the two groups of subjects.

3. Data

We study 20 patients with ischemic heart failure. All recordings were acquired in the First Department of Cardiology, Medical School, National and Kapodistrian University of Athens, supervised by clinical experts. Patients underwent physical examination, chest X Ray, blood and biochemical tests, 12 lead-ECG, ECHO, Signal Averaged ECG (SAECG) and Holter Monitoring (HM), while personal, family history and medications were registered. All patients provided informed consent and the study was approved by our institution's Ethics Committee.

From those patients, 9 were characterized as *high risk* for SCD after 16 months of follow up (ICD's appropriate activation: 5 patients, Clinical VT/VF: 1 patient and confirmed SCD: 3 patients) and the rest 11 arrhythmia free patients considered as *low risk*. Further details on the data can be found on table 1.

Totally there were examined 24 NSVT episodes, 12 from the low risk group and 12 from the high risk group. In a period 3 hours before each one of these 24 episodes, no other NSVT episode was observed.

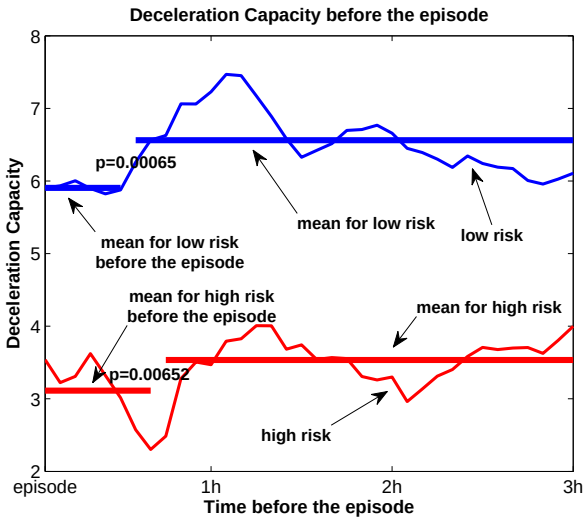

Figure 1. DC is reduced before an NSVT episode.

Table 1. Description of the dataset

	Low Risk	High Risk	p-value
Recordings	11	9	
Episodes	12	12	
Age (years)	62	72	0.035
Male Gender	7	10	0.440
LVEF(%)	40.9	40.5	0.894
Heart rate (bpm)	70	73	0.444

Table 2. Mean Deceleration Capacity for the two groups

	30 min before episode	30-180 min before episode	p-value
High Risk	3.11 ± 0.46	3.53 ± 0.33	0.00652
Low Risk	5.91 ± 0.06	6.56 ± 0.42	0.00065

4. Results

In table 2 we can see the mean values for both group of subjects for the two examined periods before the episode. The same information is graphically available in figure 3. From both the table and the figure we can easily draw some conclusions.

The high risk patients present much lower values of Deceleration Capacity in all examined periods than the low risk patients. The mean value of Deceleration Capacity for

Table 3. Reduction of Deceleration Capacity for the two groups before the NSVT episode

	absolute	percentage
High Risk	0.42	11.90%
Low Risk	0.65	9.91%

the high risk patients just before the episode is 3.11 much lower than the corresponding value of the low risk group (5.91). In accordance, in the second period (30-180 min before the episode) the mean values were 3.53 and 6.56 respectively.

Both groups of subjects present lower Deceleration Capacity in the period just before the episode. We can see a clear reduction of the capability of the heart to decelerate its rhythm before NSVT episodes. This value is reduced from 3.53 to 3.11 for high risk patient and from 6.56 to 5.91 for low risk patients.

The reduction of Deceleration Capacity before the episode is more significant in low risk patients. The p-value which characterizes the discrimination between the two periods is approximately 10 times larger in high risk patients and falls from 0.00652 to 0.00065.

The absolute and percentage reduction of Deceleration Capacity before the episode is shown in table 3. The absolute reduction of Deceleration Capacity for the low risk patients is 0.65, while the absolute reduction for the high risk group is (0.42). By calculating the percentage average reduction for each group we can see that the percentage average reduction for the low risk patients is 9.91%, lower than that of the high risk patients (11.90%).

The p-values for the discrimination of mean values of Deceleration Capacity in both examined periods is almost equal to zero.

5. Conclusions

We analyzed the periods before NSVT episodes for low and high risk patients for Arrhythmic Sudden Cardiac Death. We concluded that both groups of subjects presented a reduction of Deceleration Capacity exactly before those episodes. Furthermore, the high risk patients presented a larger percentage reduction than the low risk patients. In all our experiments the results appeared statistically significant with very low p-values.

References

[1] Goldberger AL, Findley LJ, Blackburn MR, Mandell AJ. Nonlinear dynamics in heart failure: implications of long-wavelength cardiopulmonary oscillations. American Heart Journal 1984;107:612–615.

[2] Dilaveris PE, Gialafos JE. Dynamics of heart rate before arrhythmias. In Malik M, Camm AJ (eds.), Dynamic Electrocardiography. NY: Blackwell Futura, 2004; 571.

[3] Valkama JO, Huikuri HV, Koistinen MJ, Yli-Mayry S, Airaksinen KEJ, Myerburg RJ. Relation between heart rate variability and spontaneous and induced ventricular arrhythmias in patients with coronary artery disease. J Am Coll Cardiol 1995;25(2):437–443.

[4] Arsenos P, Gatzoulis K, Dilaveris1 P, Manis G, Tsiachris D, Archontakis S, Vouliotis AH, Sideris S, Stefanadis C. Arrhythmic sudden cardiac death: Substrate, mechanisms and current risk stratification strategies for the post-myocardial infarction patient. Hellenic J Cardiol 2013;54:301–315.

[5] Lombardi F, Porta A, Marzegalli M, Favale S, Santini M, Vincenti A, De Rosa A. Heart rate variability patterns before ventricular tachycardia onsert in patients with an implantable cardioverter defibrillator. Am J Cardiol 2000;86:959–963.

[6] Bauer A, Kantelhardt JW, Barthel P, Schneider R, Makikallio T, Ulm K, Hnatkova K, Schomig A, Huikuri H, Bunde A, Malik M, Schmidt G. Deceleration capacity of heart rate as a predictor of mortality after myocardial infarction: cohort study. Lancet 2006;367(9523):1674–81.

[7] Vanoli E, De Ferrari GM, Stramba-Badiale M, Jr Hull SS, Foreman RD, Schwartz PJ. Vagal stimulation and prevention of sudden death in conscious dogs with a healed myocardial infarction. Circ Res 1991;68:1471–1481.

[8] Bauer A, Kantelhardt JW, Bunde A, Barthel P, Schneider R, Malik M, Schmidt G. Phase-rectified signal averaging detects quasi-periodicities in non-stationary data. Physica A Statistical Mechanics and its Applications 2006;364(0):423 – 434.

[9] Campana LM, Owens RL, Clifford GD, Pittman SD, Malhotra A. Phase-rectified signal averaging as a sensitive index of autonomic changes with aging. J Appl Physiol March 2010; 108(6):1668–1673.

Address for correspondence:

George Manis
Dept. of Computer Science and Engineering
University of Ioannina
P.O. Box 1186, Ioannina, 45110, Greece
manis@cs.uoi.gr