HEART RATE VARIABILITY IN VENTRIVULAR TACHYCARDIA

Η ΜΕΤΑΠΤΥΧΙΑΚΗ ΕΡΓΑΣΙΑ ΕΞΕΙΔΙΚΕΥΣΗΣ

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Από την

Σπαή Μαρία

ως μέρος των Υποχρεώσεων

για τη λήψη

του

ΜΕΤΑΠΤΥΧΙΑΚΟΥ ΔΙΠΛΩΜΑΤΟΣ ΣΤΗΝ ΠΛΗΡΟΦΟΡΙΚΗ ΜΕ ΕΞΕΙΔΙΚΕΥΣΗ ΣΤΙΣ ΤΕΧΝΟΛΟΓΙΕΣ-ΕΦΑΡΜΟΦΕΣ

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To my Parents, Family and Friends who always stand by my side...

CREDITS

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ABSTRACT

Maria Spai, MSc Department of Computer Science and Engineering, University of Ioannina, Greece. September, 2015 Heart Rate Variability in Ventricular Tachycardia Thesis Supervisor: George Manis, Assistant Professor

Ventricular Tachycardia, is a type of tachycardia that arises from improper electrical activity of the heart presented as a rapid heart rhythm that starts in the bottom chambers of the heart, called ventricles. The ventricles are the main pumping chambers of the heart, so this is a potentially life threatening arrhythmia because it may lead to ventricular fibrillation, asystole, and sudden death.

In this thesis we study heart signals, derived from patients with ventricular tachycardia and we apply various heart rate variability methods. Our main focus was to investigate how heart rhythm adapts before an incident. But we also compared some categories of groups, based on age, weight or the kind of ventricular tachycardia in order to see variations between each case.

Time domain, time- frequency domain and other methods were implemented in order to investigate the variability of the signals. Finally, we inserted some results derived of the methods computed, as input to a support vector machine in order to predict if those are from the time space prior to the episode or from the time space some minutes before.

The results indicate changes in heart rate, prior to an episode of ventricular tachycardia, with some metrics performing better than others.

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ΠΕΡΙΛΗΨΗ ΣΤΑ ΕΛΛΗΝΙΚΑ

Μαρία Σπαή, MSc Τμήμα Μηχανικών Η/Υ και Πληροφορικής, Πανεπιστήμιο Ιωαννίνων Σεπτέμβριος 2015 Μεταβλητότητα καρδιακού σε κοιλιακή ταχυκαρδία. Επιβλέπων: Γεώργιος Μανής, Επίκουρος Καθηγητής.

Κοιλιακή Ταχυκαρδία είναι ένας τύπος ταχυκαρδίας που προκύπτει από ακατάλληλη ηλεκτρική δραστηριότητα της καρδιάς και παρουσιάζεται ως ένας ταχύς καρδιακός ρυθμός, που ξεκινά στις κάτω κοιλότητες της καρδιάς, που ονομάζονται κοιλίες. Οι κοιλίες είναι οι κύριοι θάλαμοι άντλησης της καρδιάς, έτσι αυτή είναι μία δυνητικά απειλητική για τη ζωή αρρυθμία, επειδή μπορεί να οδηγήσει σε κοιλιακή μαρμαρυγή, ασύστολή, και αιφνίδιο θάνατο.

Σε αυτή την εργασία μελετάμε σήματα της καρδιάς που προέρχονται από ασθενείς με κοιλιακή ταχυκαρδία και εφαρμόζουμε διάφορες μεθόδους μεταβλητότητας του καρδιακού ρυθμού. Κύριος στόχος μας, ήταν να διερευνήσουμε πώς προσαρμόζεται ο ρυθμός της καρδίας πριν από ένα τέτοιο περιστατικό. Αλλά επίσης συγκρίναμε, την συμπεριφορά υποκατηγοριών του συνόλου των σημάτων, με βάση την ηλικία, το βάρος ή το είδος της κοιλιακής ταχυκαρδίας.

Μέθοδοι από το πεδίο του χρόνου, το πεδίο χρόνου- συχνότητας και άλλες εφαρμόστηκαν με σκοπό να διερευνηθεί η μεταβλητότητα των σημάτων. Τέλος κάποια από τα αποτελέσματα που προέκυψαν, δόθηκαν ως είσοδος σε μία μηχανή διανυσματικής υποστήριξης, με σκοπό να δούμε πόσο καλά διαχωρίζονται η πιο μακρινή από την πιο κοντινή στο περιστατικό περιοχές.

Τα αποτελέσματα δείχνουν μεταβολές στον ρυθμό της καρδιάς, πριν από ένα επεισόδιο κοιλιακής ταχυκαρδίας, με κάποιες μετρικές να αποδίδουν καλύτερα από άλλες.

1.1 Goals

1.2 Contribution

1.3 Thesis Structure

1.1. Goals

Ventricular Tachycardia is a type of a rapid heartbeat that comes from improper electrical activity of the heart, which starts in the bottom chambers of the heart, called ventricles. A healthy adult heart normally beats 60 to 100 times a minute when a person is at rest. When ventricular tachycardia occurs the heart rate in the upper or lower chambers of the heart, or both, is increased.

In this thesis, our goal is to study hearts behavior before those episodes. More specifically, we study heart rate variability before the episodes using time, and time–frequency domain methods in order to determine how heart rhythms change before ventricular tachycardia.

We split the signals in time intervals, overlapping and non-overlapping, and we compute various methods in those intervals. We focused our interest in the difference between the values derived between the firsts and lasts intervals, meaning a time space apart from the episode and a time space immediately before the episode, so we can conclude about signals variability.

1.2. Contribution

In our research, we studied the heart rhythm prior to Ventricular Tachycardia in order to see if the variability of each signal is significant in a way to predict the episode.

It's the first time that the variability is studied in order to compute changes in time intervals prior to the episode. Also the data used where from the only database that is freely available and weren't many. For those data we implemented various metrics and most of them gave very good results for the separation of the time intervals of our interest.

We also compared results involving different patient groups, distinguishing them based on age, weight or the type of ventricular tachycardia they were suffering from, in order to study in which cases heart behave differently before an upcoming episode.

Finally we show, by inserting some results as input in a support vector machine, that the separation of the two areas, the most distant from the nearest to the incident, is possible, although we did not get great results.

1.3. Thesis Structure

This thesis consists of 7 Chapters. In the current chapter, we did a small introduction about the goals, the contribution and the previous works on this field.

In Chapter 2, we describe Tachycardia, the illness study, its types, how can be diagnosed and how can be treated.

Next, we introduce what heart rate variability is and we continue by describing Timedomain, time-frequency domain and other methods that we used in this thesis for our computations. In chapters 5 and 6 we show the results obtained from our experiments and in the last chapter we conclude our work and suggest future extensions of this work.

CHAPTER 2. TACHYCARDIA

- 2.1 Tachycardia2.2 Types of Tachycardia2.3 Diagnosis
- 2.4 Treatment

2.1. Tachycardia

The heart is one of the most important organs of the human body. It is responsible for pumping the blood to every cell in our body in order to make it function.

Heart is made up of four chambers, two upper chambers (atria) and two lower chambers (ventricles). The left side of the heart, atria and ventricle, is providing oxygenated blood from the lungs to the rest of the body. The right side is sending the deoxygenated blood back to the lungs to reoxygenate again. The two sides are separated from a wall of tissue, called septum, in order to prevent bloods passing from one side to the other.

The rhythm of the heart is normally controlled by a natural pacemaker, the sinus node, located in the right atrium. The sinus node produces electrical impulses that normally start each heartbeat. From the sinus node, electrical impulses travel across atria, causing the atria muscles to contract and pump the blood into the ventricles. Then the electrical impulses arrive at a cluster of cells, called the atrioventricular node, AV node, which slows down the electrical signal before sending it to the ventricles.



Figure 2.1 "Diagram of the human heart. Licensed under CC BY-SA 3.0

Hearts rhythm is controlled by electrical signals sent across heart tissues. Tachycardia occurs when an abnormality in the heart produces rapid electrical signal and as a result heart may not effectively pump blood to the rest of the body, depriving the organs and tissues of oxygen. Common symptoms are dizziness, shortness of breath, lightheadedness, rapid pulse rate, chest pain, fainting and some of the reasons that can cause the disruption of the normal electrical impulses that control the rate of heart pumping action are damage to the heart tissues due to some heart disease, anemia, exercise, sudden stress, high blood pressure, smoking, fever, drinking too much alcohol or many caffeinated beverages, medication side effects or abuse of recreational drugs, overactive thyroid. However, in some cases, the exact cause of tachycardia cannot be determined.

2.2. Types of Tachycardia

Tachycardia occurs from different malfunctions in hearts electrical circuitry. So different types of it occurs. Common types of tachycardia include the following:

- Atrial fibrillation is a rapid heart rate caused by chaotic electrical impulses in the atria. These signals result in rapid, uncoordinated, weak contraction of the atria. Atrial fibrillation may be temporary, but some episodes won't end unless treated.
- Atrial flutter occurs when the heart's atria beat very fast but in a regular rate and is caused by irregular circuitry within the atria. The fast rate results in weak contraction of the atria. The rapid signals entering the AV node cause a rapid and sometimes ventricular rate.
- Supraventricular tachycardia which originates somewhere above the ventricles, is caused by abnormal circuitry in the heart, usually present at birth, that creates a loop of overlapping signals. An abnormality in the AV node may split an electrical signal into two, sending one signal to the ventricles and the other to the atria. Another abnormality is the presence of an extra electrical pathway from atria to the ventricles that bypasses the AV node. This may result in a signal going down one pathway and up the other.
- Ventricular fibrillation which occurs when rapid, chaotic electrical impulses cause the ventricles to quiver ineffectively instead of pumping necessary blood to the body. This serious problem is fatal if the heart isn't restored to a normal rhythm within minutes. Most people who experience ventricular fibrillation have an underlying heart disease or have experienced serious trauma, such as being struck by lightning.
- Ventricular tachycardia which is a rapid heart rate that originates with abnormal electrical signals in the ventricles. The rapid heart rate doesn't

allow the ventricles to fill and contract efficiently to pump enough blood to the body. Ventricular tachycardia is often a life-threating medical emergency.

In this thesis we focus on ventricular tachycardia. We collected signals, both from online databases and other, concerning spontaneous ventricular tachycardia and no sustained ventricular tachycardia, and we studied their variability.

2.3. Diagnosis

The diagnosis of ventricular tachycardia is made based on the rhythm seen on either an Electrocardiogram (ECG), which is the most common way, or a telemetry rhythm strip. An ECG shows the electrical activity of the heart. Electrodes are placed on the chest, legs and arms and detect the electrical changes on the skin that arise from the heart muscle depolarizing during each heartbeat.

It may be very difficult to differentiate between ventricular tachycardia and a widecomplex supraventricular tachycardia in some cases. In particular, supraventricular tachycardia with aberrant conduction from pre-existing bundle branch block are commonly misdiagnosed as ventricular tachycardia.

Various diagnostic criteria have been developed to determine whether a wide complex tachycardia is ventricular tachycardia or a more benign rhythm. In addition to these diagnostic criteria, if the individual has a past history of a myocardial infarction, congestive heart failure, or recent angina, the wide complex tachycardia is much more likely to be ventricular tachycardia.

The proper diagnosis is important, as the misdiagnosis of supraventricular tachycardia when ventricular tachycardia is present is associated with worse prognosis. This is particularly true if calcium channel blockers, such as verapamil, are used to attempt to terminate a presumed supraventricular tachycardia. Therefore, it is wisest to assume that all wide complex tachycardia is Ventricular Tachycardia until proven otherwise.

2.4. Treatment

Therapy may be directed either at terminating an episode of the arrhythmia or at suppressing a future episode from occurring. The treatment for stable Ventricular Tachycardia is tailored to the specific patient, with regard to how well the individual tolerates episodes of ventricular tachycardia, how frequently episodes occur, their comorbidities, and their wishes. Patients suffering from pulseless Ventricular Tachycardia or unstable Ventricular Tachycardia are hemodynamically compromised and require immediate cardioversion.

- Cardioversion: If a person still has a pulse, it is usually possible to terminate the episode. This should be synchronized to the heartbeat if the waveform is monomorphic if possible, in order to avoid degeneration of the rhythm to ventricular fibrillation. An initial energy of 100J is recommended. If the waveform is polymorphic, then higher energies and an unsynchronized shock should be provided (also known as defibrillation). As this is uncomfortable, shocks should ideally only be delivered only to someone who is unconscious or sedated.
- Defibrillation: A person with pulseless Ventricular Tachycardia is treated the same as Ventricular Fibrillation with high-energy (360J with a monophasic defibrillator, or 200J with a biphasic defibrillator) unsynchronized cardioversion .They will be unconscious. The shock may be delivered to the outside of the chest using the two pads of an external defibrillator, or internally to the heart by an Implantable Cardioverter-Defibrillator (ICD) if one has previously been inserted. An ICD may also be set to attempt to overdrive pace the ventricle. Pacing the ventricle at a rate faster than the underlying tachycardia can sometimes be effective in terminating the rhythm. If this fails after a short trial, the ICD will usually stop pacing, charge up and deliver a defibrillation grade shock.

- Medication: For those who are stable with a monomorphic waveform the medications may be used. As hypo magnesia is a common cause of Ventricular Tachycardia, magnesium sulphate can be given for torsade's or if hypomagnesemia is found/suspected. Long-term anti-arrhythmic therapy may be indicated to prevent recurrence of Ventricular Tachycardia.
- Surgery: An implantable ICD is more effective than drug therapy for prevention of sudden cardiac death due to Ventricular Tachycardia and Ventricular Fibrillation, but may be constrained by cost issues, as well as patient co-morbidities and patient preference. Catheter ablation is a possible treatment for those with recurrent Ventricular Tachycardia. Remote Magnetic Navigation is one effective method to do the procedure. There was consensus among the task force members that catheter ablation for Ventricular Tachycardia should be considered early in the treatment of patients with recurrent Ventricular Tachycardia. In the past, ablation was often not considered until pharmacological options had been exhausted, often after the patient had suffered substantial morbidity from recurrent episodes of Ventricular Tachycardia and ICD shocks. Antiarrhythmic medications can reduce the frequency of ICD therapies, but have disappointing efficacy and side effects. Advances in technology and understanding of Ventricular Tachycardia substrates now allow ablation of multiple and unstable Ventricular Tachycardia's with acceptable safety and efficacy, even in patients with advanced heart disease.

CHAPTER 3. HEART RATE VARIABILITY

- 3.1 Introduction
- 3.2 Time Domain Methods
- 3.3 Time Frequency Domain Methods
- 3.4 Other Methods

3.1. Introduction

Heart rate variability (HRV) is the physiological phenomenon of variation in the time interval between heartbeats. It is measured by the variation in the beat-to-beat interval. Methods used to detect beats include: ECG, blood pressure, ballistocardiograms and the pulse wave signal derived from a photoplethysmograph (PPG). ECG is considered superior because it provides a clear waveform, which makes it easier to exclude heartbeats not originating in the sinoatrial node. In the following picture we can see an electrocardiogram (ECG) recording of a heart that illustrates beat-to-beat variability in RR interval (top) and hear rate (bottom), where R is a point corresponding to the peak of the QRS complex of the ECG wave, and RR is the interval between successive Rs.



Figure 3.1 ECG example showing RR intervals.

The QRS complex is a wave resulting from the depolarization of the left and right ventricles. The Q wave is the first negative deflection, followed by the R wave which is the first positive deflection. S is the last negative deflection. The RR interval measures the length between two successive R waves. A typical QRS can be seen in the figure above.



Figure 3.2 The QRS Complex.

Reduced HRV has been shown to be a predictor of mortality after myocardial infraction [10], although others have shown that the information in HRV relevant to acute myocardial infraction survival is fully contained in the mean heart rate.

To perform HRV analysis the most widely used methods can be grouped under time domain, frequency domain and time- frequency domain methods. Other methods have been proposed such as non- linear methods. Multiple papers with regard to the study of cardiac signals, have used HRV analysis to draw conclusions in each topic [5, 6, 7, 9, 10].

3.2. Time-domain Methods

Time- domain methods are based on the beat-to-beat or NN intervals, where "NN" is used in place of RR to emphasize the fact that the processed beats are "normal" beats. Time domain methods can be separated into simple time domain variables, statistical methods and geometrical methods and can either be measured over the whole signal, or over smaller periods. From time domain methods we can extract information about the kind of variability that exists in a signal and those methods are less affected by the presence of artifacts in the signal.

Simple time domain variables include the mean NN interval and the rate NN. Those methods are easy to obtain while they hold important information for various heart conditions.

Statistical methods include the standard deviation of the NN intervals (SDNN) which can be computed over the whole signal but it can give more information when computed over shorter periods. Another important method is the root mean square of successive differences between adjacent NNs (RMSSD). This method is associated with short term, rapid changes in the heart rate.

$$RMSSD = \sqrt{\frac{1}{N-1} \left(\sum_{i=1}^{N-1} ((R-R)_{i+1} - (R-R)_i)^2 \right)}$$

where N = number of RR interval terms

There is also the proportion of the number of pairs of successive NNs that differ by more than 50ms, NN50, divided by total number of NNs (PNN50). This method also measure short-term variation in the NN interval because is entirely based on comparisons between successive beats.

$$pNN50 = NN50count / n$$

Where

$$NN50count = \sum_{i=1}^{n} x_{i+1} - x_i \ \alpha \nu \ x_{i+1} - x_i > 50 \text{ms}$$

And n is the number of heart beats.

3.3. Time- Frequency domain Methods

As we mentioned previously time-frequency domain methods combine characteristics from both time and frequency spaces. Frequency domain methods concern the spectral analysis component and the spectral density of the signal. These analyses are divided into two categories depending the length of the signal studied, in small duration measurements (2-5 minutes) and long duration measurements (24 hours).

One of the most popular methods of time-frequency domain are wavelets. Wavelets are mathematical functions that cut up data into different frequency components, and then study each component with a resolution matched to its scale. They have advantages especially in situations where the signal contains discontinuities and sharp spikes.

The basic idea of wavelet transforms is that the transformation should allow only changes in time extension, but no in shape. This is effected by choosing suitable basis functions that allow this. Changes in time extension are expected to conform to the corresponding analysis frequency of the basis function. Based on the uncertain principle of signal processing,

$$\Delta t \Delta \omega \geqq \frac{1}{2}$$

Where t represents time and ω angular frequency ($\omega = 2\pi f$, where f is temporal frequency).

The higher the required resolution in time, the lower the resolution in frequency has to be. The larger the extension of the analysis windows is chosen, the larger is the value of Δt .

When Δt is large results:

- Bad time resolution
- Good frequency resolution
- Low frequency, large scaling factor

When Δt is small results:

- Good time resolution
- Bad frequency resolution
- High frequency, small scaling factor



Figure 3.3 Examples with different frequency.

The first step while working with wavelets is to choose a family of wavelets to work with. Depending with the problem we can choose from:

• Haar, which is discontinuous and resembles a step function



Figure 3.4 The Haar wavelet.

- Daubechies, which are orthogonal wavelets defining a discrete wavelet transform and characterized by a maximal number of vanishing moments for some given support.
- Biorthogonal, which is a wavelet where the associated wavelet transform is invertible but not necessarily orthogonal.
- Gaussian etc.

After choosing a wavelet family, next step is analyzing the signal in various scales. By doing that we process data at different scales. By looking at a signal with a wide "window", we would notice mixed features. Similarly, if we look at a signal with a small "window", we would notice small features. With wavelet analysis we can get both results.

For each scale we compute the approximation and detail coefficients.

approximation:
$$\frac{x(i) + x(i+1)}{2}$$
detail:
$$\frac{x(i) - x(i+1)}{2}$$

The variability in each scale of resolution is given from the standard deviation of the detail coefficients as follows

$$s_N = \sqrt{\frac{1}{N} \sum_{i=1}^{N} (x_i - \overline{x})^2}$$


Figure 3.5 A 3 scale wavelet.

So from time domain methods we can learn how variable a signal is, from frequency domain methods which rhythms are hiding in the signal and which normal procedure they reveal and from time-frequency domain methods we can combine information both from time and frequency domains.

3.4. Other Methods

Another method used to analyze heart rates variability is Deceleration Capacity [3] which computes the capability of the heart to decelerate its rhythm. Beat to Beat Deceleration Capacity stands for the deceleration capacity of the heart rate from successive differences only. Beat to beat deceleration capacity is computed as follows and examines what happens per beat basis

and since x(0) is always larger than x(-1), beat to beat deceleration capacity is always positive.

Another form of deceleration capacity is SignDC, which is a different approach of deceleration capacity and is computed as follows



Figure 3.6 The formula for DC.

SignDC investigates deceleration in a longer term basis, and given that a series of segments is deceleration, how well the heart decelerates this period.

Both SignDC and BBDC are variations of the original method DC, Deceleration Capacity. In this method we define anchor points as the RR intervals which are bigger from their previous. The intervals which differ more than 5% from their previous are excluded and then the intervals around the anchor are aligned and the DC value is computed:

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

Where X(0) is the mean value derived from all anchor points, X(1) is the mean value derived from all the points following the anchor point and X(-1), X(-2) the mean value derived from the previous points respectively.

When computing SignDC the way filters are computing is different and also the way intervals are characterized as deceleration or acceleration. More specifically, assuming we have the intervals

$$RRi = RR1, RR2, \dots, RRN$$

we define the vectors

$$xi = (RRi, RRi+1, RRi+2, RRi+3)$$

Where

$$1 \le i \le N - 3$$

Then the vector v_i is defined as follows:

$$v_{1} \cdot \left(\begin{array}{c} 1, & \left| \frac{x(4) - x(3)}{x(3)} \right| \le 0.05, \left| \frac{x(3) - x(2)}{x(2)} \right| \le 0.05, \left| \frac{x(2) - x(1)}{x(3)} \right| \le 0.05, \\ 0, & \text{otherwise} \end{array} \right)$$

Where $x_i(1), x_i(2), x_i(3), x_i(4)$ are computed respectively to the original method. This computation results that if one of the inequalities is not satisfied for some points, then this points are excluded.

$$acdc_{i} = \frac{x_{i}(4) + x_{i}(3) - x_{i}(2) - x_{i}(1)}{4}$$

The value

is also defined and if this value is bigger than 0 a vector is characterized as deceleration

$$dc_{i} \cdot \begin{pmatrix} 1, & \text{if acdc} > 0 \\ 0, & \text{otherwise} \end{pmatrix}$$

and as acceleration if is smaller than 0

$$ac_{1} \cdot \begin{pmatrix} 1, & \text{if acdc} < 0 \\ 0, & \text{otherwise} \end{pmatrix}$$

The vectors

$$\overline{dc} = (\overline{dc_i^*v_i^*x_i(1)}, \overline{dc_i^*v_i^*x_i(2)}, \overline{dc_i^*v_i^*x_i(3)}, \overline{dc_i^*v_i^*x_i(4)})$$
$$\overline{ac} = (\overline{ac_i^*v_i^*x_i(1)}, \overline{ac_i^*v_i^*x_i(2)}, \overline{ac_i^*v_i^*x_i(3)}, \overline{ac_i^*v_i^*x_i(4)})$$

are also computed and from them is computed the final value of SignDC.

CHAPTER 4. DATA AND IMPLEMENTATION

4.1 Data

4.2 Implementation

4.1. Data

For the purposes of this thesis, we first searched an online database bank for ECGs derived from patients suffering from ventricular tachycardia, Physionet Data Bank [19]. We both searched for whole databases concerning this illness and for signals included in other databases but containing ventricular tachycardia episodes. Although ventricular tachycardia isn't a rare malady, we couldn't find an oversupply of signals. We found and used for our experiments the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc¹. We also used data from 20 patients with ischemic heart failure. Those signals were first used in a previous research [2], and were acquired in the First Department of Cardiology, Medical School, National and Kapodistrian University of Athens, supervised by clinical experts. At this point, we would like to thank Dr. Petros Arsenos, from the First Department of Cardiology, Medical School, National and Kapodistrian University of Athens and this clinic for the courtesy of those data.

¹ http://physionet.org/physiobank/database/mvtdb/

The online database (VT) contains 135 pairs of RR interval time series, recorded by implanted cardioverter defibrillators in 78 subjects. Episodes were retrieved from implanted devices during the patient's scheduled follow-ups and during subsequent unscheduled visits. Those signals concern both ventricular tachycardia and ventricular fibrillation Incidents. For the purposes of this thesis we only used the signals related to ventricular tachycardia. After excluding some signals, we arrived to a database with 86 signals with the 1024 most recently measured RR intervals just prior a ventricular tachycardia event is detected. Given the information the database provided we could also split the signals based on the age of the patients and on their weight.

The second database (NSVT) consists of 20 patients, with some of them suffering from multiple episodes of non-sustained ventricular tachycardia. Totally, we examined 35 NSVT episodes, also stored in the a equivalent way with those from the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc., meaning that the last beat of the signal is the just prior to each episode.

4.2. Implementation

Matlab code was produced in order to process all the data and implement our experiments. The online data didn't need some preprocessing but the other did. So we needed to parse every patients signals into sub signals in order to have only files ending with the beat just prior to the episode.

Next we implemented our metrics, described in Chapter 3. For each signal we compute each metric in time window intervals. After experimenting with the windows number we arrived at using 16 time windows for each signal and we computed each metric for both non-overlapping and overlapping windows. Then we parted those windows into three areas, the first area was consisted from the first 5 time windows and the third from the last 5 time windows. The second included all the in between

intervals. We focused our interest in the first and third area in order to see if hearts variability changes drastically right before an episode.

So we computed each metric and based on those results we computed the p-values for each metric for the three areas of the signal. P-value is a function of the observed function results that is used for testing a statistical hypothesis. In this case, the hypothesis is that the two groups comparing each time, are coming from distributions with equal mean value. If the p-value is bigger than 0.05, the hypothesis is accepted, otherwise is rejected. We assume that our samples are well separated when their p-value is smallest than 0.05.

Finally, the results of some of the metrics were given as input to a support vector machine in order to be found if the 2 areas could be correctly categorized.

CHAPTER 5. EXPERIMENTAL RESULTS

5.1 Results

5.1.1 All Signals
5.1.2 Age of the patient
5.1.3 Weight of the patient
5.1.4 NSVT Signals
5.2 VT and NSVT Comparison

5.1. Results

The goal of this thesis was to examine the variability of the heart before a ventricular tachycardia episode, in order to conclude whether hearts behavior changes before those episodes. In this chapter, we will introduce the results of our experimental work. We will initially present results, concerning the total of the signals used from the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc. Subsequently, we will show some results for some subsets of the initial database. Finally, we will show the outcomes for the NSVT database.

5.1.1. All Signals

As described at Chapter 4, we used signals derived from the online database Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc. We used 86 signals from a total of 102, which concerned ventricular tachycardia. Those where from males patients, aged from 20 to 78 years old and weighed from 61 to 113 kg.

Follow the plots for each metric computed, for overlapping and non-overlapping time windows and a table showing the p-value computed over each two areas. We also present the box plot for each metric, for the first and last area of the signal.

Firstly we computed the RateNN for non-overlapping and overlapping windows. As we can conclude from all the following plots, both in overlapping and nonoverlapping windows computations heart rate increases right before the episode. That means that heart accelerates its rhythm preceding to ventricular tachycardia.



Figure 5.1 The RateNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean RateNN.



Figure 5.2 The mean RateNN from all the samples over 16 non-overlapping windows



Figure 5.3 The RateNN for all the samples, over 16 overlapping windows, the blue line is representing the mean RateNN.



Figure 5.4 The mean RateNN from all the samples over 16 non-overlapping windows

In the following pictures we show the box plots for rate concerning area1 and area3 of the signals. As we can see from both pictures, the mean value of rate is completely different in the two areas, although the values that rate takes aren't completely different, something that was pretty obvious from the previous plots. That difference is shown at the p-value of those area too, as we will see later.



Figure 5.5 Box plot for RateNN, area1 and area2, with non-overlapping windows



Figure 5.6 Box plot for RateNN, area1 and area2, with overlapping windows

Next we computed MeanNN respectively. To a full much with rate, MeanNN is decreasing right before the episode as expected.



Figure 5.7 The MeanNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean MeanNN.



Figure 5.8 The mean MeanNN from all the samples over 16 non-overlapping windows



Figure 5.9 The MeanNN for all the samples, over 16 overlapping windows, the blue line is representing the mean MeanNN.



Figure 5.10 The mean RateNN from all the samples over 16 non-overlapping windows

As we can see from the box plots following, the mean value of MeanNN is completely different in the two areas, although the values that MeanNN takes aren't completely different in the two areas of our concern.



Figure 5.11 Box plot for MeanNN, area1 and area2, with non-overlapping windows



Figure 5.12 Box plot for MeanNN, area1 and area2, with overlapping windows

Then we computed the RMSSD. As we can see from the following plots, the way our signals behave result a not so smooth plot for RMSSD. We can see a sharp edge at the beginning of the second area followed from a not smooth also increment at the third area too.



Figure 5.13 The RMSSD for all the samples, over 16 non-overlapping windows, the blue line is representing the mean RMSSD.



Figure 5.14 The mean RMSSD from all the samples over 16 non-overlapping windows



Figure 5.15 The RMSSD for all the samples, over 16 overlapping windows, the blue line is representing the mean RMSSD.



Figure 5.16 The mean RMSSD from all the samples over 16 overlapping windows.

From the box plots following we can see as expected, the values of RMSSD in area1 and area2 are overlapping.



Figure 5.17 Box plot for RMSSD, area1 and area2, with non-overlapping windows



Figure 5.18 Box plot for RMSSD, area1 and area2, with overlapping windows

We next computed PNN50. We also can observe an abrupt change on this metric although the mean value is also finally increasing when we have non-overlapping windows. In case of overlapping this final Increment is follows from as small decrement.



Figure 5.19 The PNN50 for all the samples, over 16 non- overlapping windows, the blue line is representing the mean PNN50.



Figure 5.20 The mean PNN50 from all the samples over 16 non-overlapping windows



Figure 5.21 The PNN50 for all the samples, over 16 overlapping windows, the blue line is representing the mean PNN50.



Figure 5.22 The mean PNN50 from all the samples over 16 non-overlapping windows.

As we can see from the following box plots, despite the irregular form of the plots, area1 and area3 mean values using PNN50 metric, are different, but with a big number of outliers.



Figure 5.23 Box plot for PNN50, area1 and area2, with non-overlapping windows



Figure 5.24 Box plot for PNN50, area1 and area2, with overlapping windows

We next compute the SDNN. Although in both case SDNN shows an increasing behavior, when computed with overlapping windows the graph show a sharp form.



Figure 5.25 The SDNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean SDNN.



Figure 5.26 The mean SDNN from all the samples over 16 non-overlapping windows



Figure 5.27 The SDNN for all the samples, over 16 overlapping windows, the blue line is representing the mean SDNN.



Figure 5.28 The mean SDNN from all the samples over 16 non-overlapping windows.

As we can see from the following box plots in both cases, area1 and area3 mean values using SDNN metric, are different, but with a big number of outliers. Box plot for SDNN with non overlapping windows



Figure 5.29 Box plot for SDNN, area1 and area2, with non-overlapping windows



Figure 5.30 Box plot for SDNN, area1 and area2, with overlapping windows

Continuing, we computed the two deceleration capacity methods described in Chapter 3 section 4. First, we present the results for Beat to Beat Deceleration Capacity. In both scenarios, we can see an abnormal behavior. We have multiple edges and a quite different behavior in non-overlapping and overlapping windows. In the first case, despite all the sharp variations, BBDC is obviously decreasing in the third area, while in the second case, it concludes to a value close to where it started.



Figure 5.31 The BBDC for all the samples, over 16 non-overlapping windows, the blue line is representing the mean BBDC.



Figure 5.32 The mean BBDC from all the samples over 16 non-overlapping windows



Figure 5.33 The BBDC for all the samples, over 16 overlapping windows, the blue line is representing the mean BBDC.



Figure 5.34 The mean BBDC from all the samples over 16 overlapping windows.

Although the changes described previously, box plots in this case doesn't give a good separation, which come accordingly to those areas p-values that we will present to you.



Figure 5.35 Box plot for BBDC, area1 and area2, with non-overlapping windows



Figure 5.36 Box plot for BBDC, area1 and area2, with overlapping windows

This measurement, SignDC, also results really sharp lines. There is also a slight difference in the two cases. In the second case, at the third area the plots shows a clear Increment, fact which is not the case in the first.



Figure 5.37 The SignDC for all the samples, over 16 non-overlapping windows, the blue line is representing the mean SignDC.



Figure 5.38 The mean SignDC from all the samples over 16 non-overlapping windows.



Figure 5.39 The SignDC for all the samples, over 16 overlapping windows, the blue line is representing the mean SignDC.



Figure 5.40 The mean SignDC from all the samples over 16 overlapping windows.



Although the number of outliers is big in both cases, we can see from the box plots, that in the case of overlapping windows, SignDC separates well the two areas.

Figure 5.41 Box plot for SignDC, area1 and area2, with non-overlapping windows



Figure 5.42 Box plot for SignDC, area1 and area2, with overlapping windows

The following table shows the results concerning the p-values for all the previous methods.

	p-value Area1/Area3	p-value Area1/Area2	p-value Area2/Area3
RateNN Non-overlapping	0.019096	7.8E-5	0.078285
RateNN Overlapping	2.2E-5	3.19E-4	0.032876
MeanNN Non-overlapping	0.014897	8.5E-5	0.057253
MeanNN Overlapping	3.5E-5	3.22E-4	0.013079
RMSSD Non-overlapping	0.884934	0.024604	0.05728
RMSSD Overlapping	0.285321	0.120064	0.491467
PNN50 Non-overlapping	0.010573	0.968353	0.008685
PNN50 Overlapping	0.030309	0.903082	0.034366
SDNN Non-overlapping	0.037206	0.011862	0.029971
SDNN Overlapping	0.013056	0.024575	0.021974
SignDC Non-overlapping	0.078924	0.064140	0.624847
SignDC Overlapping	0.001662	0.215122	0.045420
BBDC Non-overlapping	0.173067	0.440522	0.304844
BBDC Overlapping	0.093777	0.499556	0.449341

Table 5-1 p-values for each method, between all the areas.

As we can observe from the above results, MeanNN and RateNN give the best p-values for area1 and area3, given that we assume that every value smaller than 0.05 is better for separating our data. We can also see that PNN50 and SDNN perform in a satisfying way, as well as SignDC computed over overlapping windows.

Finally, we used wavelets, with three different families of them, Haar, Daubechies and Biorthogonal and we computed the p-value for area1 and area3, for scales 1 to 8. For each scale we computed the standard deviation in each time window interval.

For Haar, we get our best result at scale 8, with p-value 0.0049873 between area1 and area3.



Figure 5.43 Standard deviation of Haar wavelet in scale 8.

For Daubechies we get our best result at scale 2, with a p-value 0.034 for the two areas.



Figure 5.44 Standard deviation of Daubechies wavelet in scale 2.



For biorthogonal we also get the best p-value at scale 2 and is equal to 0.0023

Figure 5.45 Standard Deviation of Biorthogonal wavelet, in scale 2

Below is shown the table containing all the p-values for all the scales tested, for the three wavelets families.

	Scale1	Scale2	Scale3	Scale4	Scale5	Scale6	Scale7	Scale8
Haar	0.049	0.011	0.582	0.043	0.266	0.243	0.064	0.004
Daubechies	0.072	0.003	0.393	0.261	0.199	0.339	0.044	0.080
Biorthogonal	0.048	0.002	0.559	0.047	0.246	0.245	0.093	0.678

Table 5-2 p-values for all the wavelets standard deviation, for all the scales, between area1 and area3.

5.1.2. Age of the patient

The Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc., provided with information about patient's age, weight, height etc. So, since it is known that age and weight are factors that affect heart conditions [8], we decided to split the dataset based on those two operators and do some more experiments. We isolated each group of signals and computed all the metrics that we computed for the whole database, but we also compared the behavior of those metrics on young to elderly and normal weight to overweight patients. In this section, we will present you the results based on the age of the patient.

The database is not equally split by the age of the patients, since we have 65 signals derived from patients older than 63 years old and 21 signals from patients younger than 63 years old.

Since, this is a subclass of the initial database, we will only show the plots comparing the results between young and old patients. For fullness, we will cite the tables containing, all the p-values for each group of signals.

	p-value	p-value	p-value
	Area1/Area3	Area1/Area2	Area2/Area3
RateNN Non-overlapping	0.017021	5,50E-10	0.102475
RateNN Overlapping	7,77E-09	2,62E-11	0.077476
MeanNN Non-overlapping	0.025439	4,82E-10	0.098419
MeanNN Overlapping	3,11E-10	2,33E-11	0.053737
RMSSD Non-overlapping	0.944832	0.019128	0.024796
RMSSD Overlapping	0.488118	0.017400	0.050359
PNN50 Non-overlapping	0.016175	0.953651	0.003658
PNN50 Overlapping	0.132349	0.983754	0.117329
SDNN Non-overlapping	0.033717	0.013049	0.026556
SDNN Overlapping	0.020773	0.004619	0.034088
SignDC Non-overlapping	0.141775	0.227303	0.415861
SignDC Overlapping	0.008571	0.371669	0.024733
BBDC Non-overlapping	0.369812	0.258937	0.593788
BBDC Overlapping	0.092819	0.265730	0.231173

Table 5-3 p-values for each method, between all the areas, for the oldest patients.

	p-value Area1/Area3	p-value Area1/Area2	p-value Area2/Area3
RateNN Non-overlapping	0.143260	0.114298	0.149699
RateNN Overlapping	0.242031	0.017643	0.747767
MeanNN Non-overlapping	0.071911	0.036891	0.088998
MeanNN Overlapping	0.056926	0.040360	0.199596
RMSSD Non-overlapping	0.484356	0.924551	0.319100
RMSSD Overlapping	0.605948	0.817417	0.508314
PNN50 Non-overlapping	0.024432	0.518892	0.033727
PNN50 Overlapping	0.002759	0.738720	0.004571
SDNN Non-overlapping	0.038954	0.063859	0.011197
SDNN Overlapping	0.049508	0.087176	0.070311
SignDC Non-overlapping	0.246524	0.195188	0.789693
SignDC Overlapping	0.011281	0.630899	0.098357
BBDC Non-overlapping	0.987427	0.362112	0.405209
BBDC Overlapping	0.196859	0.216131	0.720670

Table 5-4 p-values for each method, between all the areas, for the youngest patients.

Starting with RateNN we can see, that in both cases, in area1, rate differs a lot. That can easily be understood since we are having people from different age groups. The interesting outcome from this measurement, is that despite the age of the patient heart increases its rhythm just prior to an episode and actually following the same pattern, despite the fact that the values of rate in the group of oldest people are lower.



Figure 5.46 Comparing plot for RateNN, between old and young patients with nonoverlapping windows.



Figure 5.47 Comparing plot for RateNN, between old and young patients with overlapping windows.
Continuing with the MeanNN, as expected we can make the same observations as with RateNN.



Figure 5.48 Comparing plot for MeanNN, between old and young patients with nonoverlapping windows.



Figure 5.49 Comparing plot for MeanNN, between old and young patients with overlapping windows.

Then we compared the RMSSD metric. In this case we can observe, that when we compute RMSSD over overlapping time windows, the plots differs in area 3. For young patients RMSSD decreases while for older patients increases. This doesn't happened for non-overlapping windows.



Figure 5.50 Comparing plot for RMSSD, between old and young patients with nonoverlapping windows.



Figure 5.51 Comparing plot for RMSSD, between old and young patients with overlapping windows.

Follow, the plots for PNN50. As with all the previous measurements, for both groups, both plots follow the same pattern. When computed with overlapping windows, the values of PNN50 for older people are clearly higher, so comes and the separation between the two groups, but the behavior of the plots is quite similar.



Figure 5.52 Comparing plot for PNN50, between old and young patients with nonoverlapping windows.



Figure 5.53 Comparing plot for PNN50, between old and young patients with overlapping windows.

We next compared the SDNN for both groups. As it is obvious from the plot following both resulted almost the same with a small exception at the end of the plot, in the case of overlapping windows. At that point the standard deviation for older people Increases while for young decreases.



Figure 5.54 Comparing plot for SDNN, between old and young patients with nonoverlapping windows.



Figure 5.55 Comparing plot for SDNN, between old and young patients with overlapping windows.

Then we compared both BBDC and SignDC. For BBDC we can see that in area1 beat to beat deceleration capacity differs between young and older people. Young patients score lower values of deceleration capacity but just prior to the Incident, they almost have the same mean value as can be seen from the first plot.



Figure 5.56 Comparing plot for BBDC, between old and young patients with nonoverlapping windows.



Figure 5.57 Comparing plot for BBDC, between old and young patients with nonoverlapping windows.



Concerning SignDC we can also see a similar outcome as with BBDC.

Figure 5.58 Comparing plot for SignDC, between old and young patients with nonoverlapping windows.



Figure 5.59 Comparing plot for SignDC, between old and young patients with overlapping windows.

Summarizing, we can conclude that both in young and in older patients, just prior to the incident, the heart tends to adapt in the same way. Although some metrics showed a clear difference in area1 between young and older patients, all of them showed a similar trend for area3.

Finally, we computed the p-value of the change between the two plots for area1 and area 2. The results can be seen in the following table.

	p between the change in area1 and
	change in area 3
RateNN Non-overlapping	0.073126003185100
RateNN Overlapping	3,96E+11
MeanNN Non-overlapping	0.032279989720613
MeanNN Overlapping	0.005604145248088
RMSSD Non-overlapping	0.627374357547858
RMSSD Overlapping	0.524820113700616
PNN50 Non-overlapping	0.483772088100754
PNN50 Overlapping	0.430484240504543
SDNN Non-overlapping	0.867978480485922
SDNN Overlapping	0.107431673703546
SignDC Non-overlapping	0.771917254174947
SignDC Overlapping	0.440277419561200
BBDC Non-overlapping	0.393210707068313
BBDC overlapping	0.524820113700616

Table 5-5 P-values of the change between area 1 and area 3.

5.1.3. Weight of the patient

In the same approach with the previous section, we will present results categorizing the data based on patient's weight.

This partitioning consist of one group of 36 overweight patients, weighted over 90 kgs, and 50 normal weight patients. Same as before, the results from all the experiments will be shown at the appendix and in this section we will limit ourselves in to showing the results comparing the two groups.

Since, this is a subclass of the initial database, as previously, we will only show the plots comparing the results between young and old patients. For fullness, we will cite the tables containing, all the p-values for each group of signals.

	p-value Area1/Area3	p-value Area1/Area2	p-value Area2/Area3
RateNN Non-overlapping	0.0138683	5,38E-11	0.0581633
RateNN Overlapping	4,14E-10	0.0023621	0.0344021
MeanNN Non-overlapping	0.0083249	5,96E-11	0.0382104
MeanNN Overlapping	4,71E-10	0.0026693	0.0170296
RMSSD Non-overlapping	0.3400980	0.4583237	0.0949467
RMSSD Overlapping	0.5882471	0.4476670	0.0957566
PNN50 Non-overlapping	0.0059191	0.4951441	0.0163065
PNN50 Overlapping	0.2001720	0.9553997	0.3030982
SDNN Non-overlapping	0.0326903	0.0875797	0.0316093
SDNN Overlapping	0.0844086	0.0184606	0.0078823
SignDC Non-overlapping	0.1537665	0.1582048	0.7687733
SignDC Overlapping	0.0300598	0.5392965	0.1596989
BBDC Non-overlapping	0.8231831	0.7415869	0.9715906
BBDC Overlapping	0.125698	0.205845	0.6570614

Table 5-6 p-values for each method, between all the areas, for the normal weight patients.

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	p-value Area1/Area3	p-value Area1/Area2	p-value Area2/Area3
RateNN Non-overlapping	0.031482	1,49E-11	0.125240
RateNN Overlapping	2,99E-10	4,43E-10	0.047551
MeanNN Non-overlapping	0.035051	5,90E-11	0.108940
MeanNN Overlapping	6,96E-10	1,66E-11	0.021167
RMSSD Non-overlapping	0.181021	0.009214	0.307719
RMSSD Overlapping	0.019989	0.029174	0.666196
PNN50 Non-overlapping	0.689204	0.271627	0.044620
PNN50 Overlapping	0.135420	0.175974	0.650613
SDNN Non-overlapping	0.044990	0.001018	0.028122
SDNN Overlapping	0.003305	7,13E-11	0.097009
SignDC Non-overlapping	0.123665	0.127079	0.512617
SignDC Overlapping	0.009720	0.096396	0.034298
BBDC Non-overlapping	0.063087	0.320469	0.066906
BBDC Overlapping	0.082281	0.256731	0.272896

Table 5-7 p-values for each method, between all the areas, for overweight patients.

Starting with the RateNN, we can see a similar behavior as before. Although in both cases the rate is following the same pattern, we can see that in the first area normal weighted people have higher values than overweighed. In the third area rate behaves in a similar way for both groups.



Figure 5.60 Comparing plot for RateNN, between normal and overweight patients with non-overlapping windows.



Figure 5.61 Comparing plot for RateNN, between normal and overweight patients with overlapping windows.

Continuing with MeanNN, we can see the same effect as expected, with the only difference that in the case of overlapping windows, despite that the plot follows the same pattern overweight peoples MeanNN gets higher values.



Figure 5.62 Comparing plot for MeanNN, between normal and overweight patients with non-overlapping windows.



Figure 5.63 Comparing plot for MeanNN, between normal and overweight patients with overlapping windows.

Then we compared RMSSD. In this case, both plots evolve with same way but each one has very different values. We can see in both pictures following, that the RMSSD for overweight people is significantly higher compared with the RMSSD for normal weight people.



Figure 5.64 Comparing plot for RMSSD, between normal and overweight patients with non-overlapping windows.



Figure 5.65 Comparing plot for RMSDD, between normal and overweight patients with overlapping windows.

Next we can see the results for PNN50. In this case also we have some distinctions. Values for overweigh patients are also higher during the whole signal, but we can also see a small difference in the way the plot evolve, although both are Increasing.



Figure 5.66 Comparing plot for PNN50, between normal and overweight patients with non-overlapping windows.



Figure 5.67 Comparing plot for PNN50, between normal and overweight patients with non-overlapping windows.

Below we can see the comparison for the SDNN. When comes to non-overlapping windows we can observe that SDNN follows the exact same pattern for both groups, although the values for the first are higher for overweight patients. On the other hand, in the case of non-overlapping windows, the pattern is also the same but the values for SDNN, are higher for overweigh people during the whole signal.



Figure 5.68 Comparing plot for SDNN, between normal and overweight patients with non-overlapping windows.



Figure 5.69 Comparing plot for SDNN, between normal and overweight patients with overlapping windows.

Subsequently we present the results for BBDC and SignDC.

As expected, values at the first area differ for BBDC but both groups BBDC follows the same pattern and results to almost the same value.



Figure 5.70 Comparing plot for BBDC, between normal and overweight patients with non-overlapping windows.



Figure 5.71 Comparing plot for BBDC, between normal and overweight patients with non-overlapping windows.



Exactly the same we can observe, when it comes to SignDC.

Figure 5.72 Comparing plot for SignDC, between normal and overweight patients with non-overlapping windows.



Figure 5.73 Comparing plot for SignDC, between normal and overweight patients with overlapping windows.

In full correspondence with the previous section, we can conclude that although the values that almost all the metrics get for overweight people are higher than those for normal weight the pattern they follow is always the same. That means that the way the heart adjust prior to an episode is the same for all the groups we studied. The arithmetical is capable, as we can see for the p-value between groups, to separate sometimes the patients according to the group they belong, but mostly in the area 1, which is the area farthest from the ventricular tachycardia episode.

Finally, we computed the p-value of the change between the two plots for area1 and area 2. The results can be seen in the following table.

	p between the change in area1 and change in
	area 3
RateNN Non-overlapping	0.007965143549565
RateNN Overlapping	0.128303307568418
MeanNN Non-overlapping	0.007184277396305
MeanNN Overlapping	0.025681020755104
RMSSD Non-overlapping	0.196578262918137
RMSSD Overlapping	0.087103558982400
PNN50 Non-overlapping	0.045282613038112
PNN50 Overlapping	0.030709110788530
SDNN Non-overlapping	0.097566244870082
SDNN Overlapping	0.107534647039655
SignDC Non-overlapping	0.781814043516266
SignDC Overlapping	0.143260403456648
BBDC Non-overlapping	0.196578262918137
BBDC overlapping	0.087103558982400

Table 5-8 P-value results for the change between area1 and area3.

5.1.4. NSVT Signals

In this section we will present the results for all the above methods for the non-sustain ventricular tachycardia database. As mentioned previously in Chapter 4 section 1, this database consists of 35 NSVT episodes. Those signals are processed and studied with the same way as the previous signals. Those signals contained more information, since they were longer in duration, but they were stored and processed in a corresponding manner. The last beat of every signal was the beat just prior to each NSVT episode and thereafter they were stored and used for our experiments. We will now cite the results for every method computed, correspondingly to those mentioned in 5.1.1 for the signals of the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc.

Starting with RateNN, we can see some difference relative to the previous results. In this case rate has a descending trend in the first two areas, while is increasing only in the third area.



Figure 5.74 The RateNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean RateNN.



Figure 5.75 The mean RateNN from all the samples over 16 non-overlapping windows.



Figure 5.76 The RateNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean RateNN.



Figure 5.77 The mean RateNN from all the samples over 16 non-overlapping windows

As we can see from the box plot following RateNN can separate quite well the first and the third area in both cases, as despite the overlapping values the mean values of each area differ.



Figure 5.78 Box plot for RateNN, area1 and area2, with non-overlapping windows.



Figure 5.79 Box plot for RateNN, area1 and area2, with overlapping windows

Continuing with MeanNN, we can see corresponding various, since this time MeanNN starts with Increment and at the third area decreases, while with the first dataset was a continuous decrement.



Figure 5.80 The MeanNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean MeanNN.



Figure 5.81 The mean MeanNN from all the samples over 16 non-overlapping windows



Figure 5.82 The MeanNN for all the samples, over 16 overlapping windows, the blue line is representing the mean MeanNN.



Figure 5.83 The mean RateNN from all the samples over 16 overlapping windows.

As we can see from the following figures, box plots for MeanNN are quite similar with those for RateNN, as expected.



Figure 5.84 Box plot for MeanNN, area1 and area2, with non-overlapping windows



Figure 5.85 Box plot for MeanNN, area1 and area2, with overlapping windows

Then we computed RMSSD, as before isn't a smooth line. In both this and the previous database, is modifying abruptly and finally Increases in the third area. Although in this case, when computed with overlapping windows, has a decrement in towards the end of the signal.



Figure 5.86 The RMSSD for all the samples, over 16 non-overlapping windows, the blue line is representing the mean RMSSD.



Figure 5.87 The mean RMSSD from all the samples over 16 non-overlapping windows.



Figure 5.88 The RMSSD for all the samples, over 16 overlapping windows, the blue line is representing the mean RMSSD.



Figure 5.89 The mean RMSSD from all the samples over 16 overlapping windows.

As expected, the box plots cannot give a good and clear result of separation using the RMSSD measurement.



Figure 5.90 Box plot for RMSSD, area1 and area2, with non-overlapping windows.



Figure 5.91 Box plot for RMSSD, area1 and area2, with overlapping windows.

Continuing, we present the results for the PNN50 method. This method result similar plots for those data, as for those of the online database. There also exist sharp variations, but an Increasing trend exists.



Figure 5.92 The PNN50 for all the samples, over 16 non-overlapping windows, the blue line is representing the mean PNN50.



Figure 5.93 The mean PNN50 from all the samples over 16 non-overlapping windows.



Figure 5.94 The PNN50 for all the samples, over 16 overlapping windows, the blue line is representing the mean PNN50.



Figure 5.95 The mean PNN50 from all the samples over 16 overlapping windows

As expected, due to the sharp variations, box plots could not result a good separation of the two areas.



Figure 5.96 Box plot for PNN50, area1 and area2, with non-overlapping windows



Figure 5.97 Box plot for PNN50, area1 and area2, with overlapping windows

Following, we can see the results for SDNN. In addition to the previous results, for this data SDNN results also a sharp line with large changes.



Figure 5.98 The SDNN for all the samples, over 16 non-overlapping windows, the blue line is representing the mean SDNN.



Figure 5.99 The mean SDNN from all the samples over 16 non-overlapping windows



Figure 5.100 The SDNN for all the samples, over 16 overlapping windows, the blue line is representing the mean SDNN.



Figure 5.101 The mean SDNN from all the samples over 16 non-overlapping windows.

As expected, box plots cannot give a good a good result of separation.



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Figure 5.102 Box plot for SDNN, area1 and area2, with non-overlapping windows.

Figure 5.103 Box plot for SDNN, area1 and area2, with overlapping windows.

Next, follow the results for BBDC and SignDC. Let's first see how BBDC performs for this data. As before, we result a line with sharp edges. The main difference is, that with the previous data BBDC ended to a value smaller just prior to the Incident, while now ends with a higher value.



Figure 5.104 The BBDC for all the samples, over 16 non-overlapping windows, the blue line is representing the mean BBDC.



Figure 5.105 The mean BBDC from all the samples over 16 non-overlapping windows



Figure 5.106 The BBDC for all the samples, over 16 overlapping windows, the blue line is representing the mean BBDC.



Figure 5.107 The mean BBDC from all the samples over 16 non-overlapping windows

Although, mean values differ for each area we still did not got a great result of separation neither in this case.



Figure 5.108 Box plot for BBDC, area1 and area2, with non-overlapping windows



Figure 5.109 Box plot for BBDC, area1 and area2, with overlapping windows
Same as before, SignDC results strong diagrams, very close to those we got as results for the first database.



Figure 5.110 The SignDC for all the samples, over 16 non-overlapping windows, the blue line is representing the mean SignDC.



Figure 5.111 The mean SignDC from all the samples over 16 non-overlapping windows



Figure 5.112 The SignDC for all the samples, over 16 overlapping windows, the blue line is representing the mean SignDC.



Figure 5.113 The mean SignDC from all the samples over 16 overlapping windows.

Considering the large changes on the plots, box plots for SignDC, have multiple outliers and did not show that SignDC can separate area1 and area3.



Figure 5.114 Box plot for SignDC, area1 and area2, with non-overlapping windows.



Figure 5.115 Box plot for SignDC, area1 and area2, with overlapping windows

Now we will cite a summary table, containing all the p-values, for each metric, between the three areas.

	p-value Area1/Area3	p-value Area1/Area2	p-value Area2/Area3
RateNN Non-overlapping	6,29E-09	0.004388647766871	0.003979165005753
RateNN Overlapping	2,16E-11	7,90E-11	0.006163878410999
MeanNN Non-overlapping	7,61E-09	0.002444545313195	0.003883097751930
MeanNN Overlapping	1,03E-11	7,12E-11	0.006922809853529
RMSSD Non-overlapping	0.295294382913156	0.024851295997571	0.307308061823022
RMSSD Overlapping	0.048707725649728	0.063212585005851	0.654934903049683
PNN50 Non-overlapping	0.004197863794658	0.115931791233024	0.211559369264154
PNN50 Overlapping	0.011382590887603	0.029486596987287	0.333182731277476
SDNN Non-overlapping	0.002151920346042	0.051207825586515	0.002511260947578
SDNN Overlapping	0.018875243185822	0.045449056956394	0.008128885016169
SignDC Non-overlapping	0.054439653757489	0,035177613	0.122132656271298
SignDC Overlapping	0.061080017529610	0.061137166937230	0.304437920875539
BBDC Non-overlapping	0.080667001977926	0.135328241472596	0.242967281695785
BBDC Overlapping	0.048049121009530	0.170680672181119	0.421194629428976

Table 5-9 p-values for each method, between all the areas.

We also used wavelets, with three different families of them, Haar, Daubechies and Biorthogonal and we computed the p-value for area1 and area3, for scales 1 to 8. For each scale, we computed the standard deviation in each time window interval.

In this case Haar separates best the two areas, in the first scale with a p-value 0.009.

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Figure 5.116 Standard deviation for Haar wavelet, in scale 1.

Daubechies also performs best in the first scale with a p-value of 0.002 also between the two areas.



Figure 5.117Standard deviation for Daubechies wavelet, in scale 1.

Biorthogonal, also performs best at the first scale with a p-value of 0.008



Figure 5.118 Standard deviation for Biorthogonal wavelet, in scale 1.

Follows a table containing the p-values for all the scales, between area1 and area3. Summarizing we can see that, Daubechies performs better both in scale 1 and scale 2.

	Scale1	Scale2	Scale3	Scale4	Scale5	Scale6	Scale7	Scale8
Haar	0.009	0.389	0.125	0.349	0.842	0.938	0.557	0.011
Daubechiess	0.002	0.012	0.287	0.213	0.763	0.826	0.232	0.387
Biorthogonal	0.008	0.229	0.076	0.582	0.669	0.882	0.151	0.481

Table 5-10 p-values for all the wavelets standard deviation, for all the scales, between area1 and area3.

5.2. VT and NSVT Comparison

Next, and since we already had all the needed information, we compared the HRV methods results, for the data of the first database, VT signals, and the data from the NSVT database, NSVT signals.

We will next cite plots for each metric, containing the result for both datasets, and displaying the p-value between area1 for each and area3. Meaning we computed the p-value between VT's and NSVT's area1 and respectively for area3.

First we compared the RateNN. As expected from the previous results, each data behave differently and we can see that p-value is very small for both areas. We can see that Rate in VT patients Increases continuously before the episode, while for the NSVT has a slight Increment just prior to it.



Figure 5.119 Comparing plot for RateNN, between VT and NSVT samples with nonoverlapping windows.



Figure 5.120 Comparing plot for RateNN, between VT and NSVT samples with overlapping windows.

Accordingly are the results for MeanNN. In this case, we can also see that the two conditions score really small p-values in both areas.



Figure 5.121 Comparing plot for MeanNN, between VT and NSVT samples with nonoverlapping windows.



Figure 5.122 Comparing plot for MeanNN, between VT and NSVT samples with overlapping windows.

RMSSD also show a good difference between the two databases. Although both plots eventually Increase their value prior to the episode, we can observe that VT signals have values almost equal to half of the values of NSVT and that results the really small p-values we get for both areas.



Figure 5.123 Comparing plot for RMSSD, between VT and NSVT samples with nonoverlapping windows.



Figure 5.124 Comparing plot for RMSSD, between VT and NSVT samples with overlapping windows.



Exactly the same effect we can observe with the PNN50 measurement.

Figure 5.125 Comparing plot for PNN50, between VT and NSVT samples with nonoverlapping windows.



Figure 5.126 Comparing plot for PNN50, between VT and NSVT samples with overlapping windows.

As we can see with SDNN, the differentiation between the datasets continues with this measurement too. Although the plots are completely different, the p-value in the third area is quite big due to the values of results.



Figure 5.127 Comparing plot for SDNN, between VT and NSVT samples with nonoverlapping windows.



Figure 5.128 Comparing plot for SDNN, between VT and NSVT samples with overlapping windows.

BBDC results as all the above methods. As we can see SignDC is the only exception from all this measurements. Plots in this case follow quite similar patterns and also take values within the same range. That's the reason why with this measurement we cannot get small p-values in any area.



Figure 5.129 Comparing plot for BBDC, between VT and NSVT samples with nonoverlapping windows.



Figure 5.130 Comparing plot for BBDC, between VT and NSVT samples with overlapping windows.



Figure 5.131 Comparing plot for SignDC, between VT and NSVT samples with nonoverlapping windows.



Figure 5.132 Comparing plot for SignDC, between VT and NSVT samples with overlapping windows.

Finally, we computed the p-value of the change between the two plots for area1 and area 2. The results can be seen in the table 5-9.

	p-value of the change area1-area2
RateNN Non-overlapping	0.009237814890361
RateNN Overlapping	1,57E-05
MeanNN Non-overlapping	0.003049808563208
MeanNN Overlapping	6,23E-06
RMSSD Non-overlapping	0.109132848099495
RMSSD Overlapping	0.033000298815369
PNN50 Non-overlapping	0.251969137031038
PNN50 Overlapping	0.131535273651693
SDNN Non-overlapping	0.042475623228109
SDNN Overlapping	0.001197092663634
SignDC Non-overlapping	0.090444918324349
SignDC Overlapping	0.097080783354982
BBDC Non-overlapping	0.007576864421602
BBDC overlapping	4,41E-05

Table 5-11 p-value of the change between the plots of VT and NSVT for area1 and area3.

After seeing all the above comparisons we can conclude, that except the case of SignDC, VT and NSVT could be well separable in both areas.

CHAPTER 6. SUPPORT VECTOR MACHINE

6.1 Support Vector Machines

6.2 Support Vector Machine Performance

6.1. Support Vector Machines

Support Vector Machines are a set of learning methods used for classification problems. Is one of the most popular methods of classification, becoming the best choice for problems such as text categorization, hand writing recognition and biomedical data [16, 17, 18]. The method can be used for two-class problems, but also for multiclass problems.

A characteristic feature of SVMs, is that their ability in learning is independent of the dimensions of the space of characteristics. SVMs measure the complexity of cases, based on the distance that can separate the data, and not on the number of characteristics. This means that we can generalize even with the presence of too many features, if our data can be separated by a wide margin using functions from the assumptions space.

While training, the SVM is trying to find a hyper level, in order to separate the training data. Actually an SVM is trying to find the best hyper level, called maximum margin hyperplane, with the capacity to separate the groups of examples with the

greatest possible distance. This means that the nearest distance between a point on a hyperplane and a separated point in another hyperplane is maximized. In fact, the examples should me correctly classified and the hyperplane should be maximized, so the SVM has to solve a constraint optimization problem. Because of the fact, that in most problems data are not linear separable, SVMs use a different approach while training and they try to find a soft margin hyperplane, meaning the level that separates the data with the smallest error.

Moreover, they can develop effective functions, for each specific problem, in order to achieve even better results. So, when linear classifier does not perform well and calculations in bigger dimensions are needed, then support vectors machines based on the kernel are used. One of the most used functions is RBF kernel (Radial Basis Function) and this function is used in this thesis.

More specifically, if the training samples are linearly separable, there exists a linear function [4] on the formula of

$$f(\mathbf{x}) = \mathbf{w}^T \mathbf{x} + b$$

Such that $y_i f(x_i) \ge 0$, or $f(x_i) \ge 0$ for $y_i = +1$ and $f(x_i) < 0$ for $y_i = -1$.

The SVM wants to find the hyperplane that maximizes the separating margins between the two classes, which can be found by minimizing the cost function

$$J(\mathbf{w}) = \frac{1}{2}\mathbf{w}^T\mathbf{w} = \frac{1}{2}\|\mathbf{w}\|^2$$

Subject to the separability constraints

$$y_i(\mathbf{w}^T\mathbf{x}_i+b) \ge 1, \quad i=1,\ldots,l.$$

When the training data is not completely separable by a hyperplane, a set of slack variables $\xi_i \ge 0$, i=1, ..., l, is introduced, that represents the amount by which the linearity constraint is violated. In that case, the cost function is modified to take into account those violations. So, the function to be minimized becomes

$$J(\mathbf{w},\xi) = \frac{1}{2} \|\mathbf{w}\|^2 + C \sum_{i=1}^{l} \xi_i$$

In this case, C gives the significance of the constraints violations with respect to the distance between the points and the hyperplane and ξ is a vector containing the slack

variables. This problem can be solved by using Lagrange multipliers and the calculation of w becomes

$$\mathbf{w} = \sum_{i=1}^{l} \alpha_i y_i \mathbf{x}_i$$

where $\alpha_i \geq 0$, i=1,...,l the Lagrange multipliers associated with the previous constraints and know the problem is expressed as

$$\max_{\alpha_i} \left\{ \sum_{i=1}^l \alpha_i - \frac{1}{2} \sum_{i=1}^l \sum_{j=1}^l \alpha_i (y_i y_j \mathbf{x}_i \mathbf{x}_j) \alpha_j \right\}$$

With the constraints

$$\alpha_i \ge 0, \quad \sum_{i=1}^l \alpha_i y_i = 0$$

Therefore, the classification function is

$$f(\mathbf{x}) = \operatorname{sign}\left(\sum_{i=1}^{l} (\alpha_i y_i \mathbf{x} \mathbf{x}_i + b)\right).$$

As said before in this section, for the many datasets a hyperplane will not result a good classifier and a decision boundary with more complex geometry is needed. One way to achieve this is to map the attribute vector in a new space of higher dimensionality and look for a hyperplane in that new space, leading to kernel-based SVMs.

Let $\Phi(\cdot)$ be a nonlinear operator mapping the input vector x to a higher dimensional space. The optimization problem for the points $\Phi(x)$ becomes

min
$$J(\mathbf{w},\xi) = \frac{1}{2} \|\mathbf{w}\|^2 + C \sum_{i=1}^{l} \xi_i$$

With the constraints

$$y_i(\mathbf{w}^T \Phi(\mathbf{x}_i) + b) \ge 1 - \xi_i, \quad \xi_i \ge 0, \quad i = 1, \dots, l.$$

The only formula in which the mapping appears is in terms of

$$K(x_i, x_j,) = \Phi^T(x_i)\Phi(x_j)$$

and finally the problem to be solved is

$$\max_{\alpha_i} \left\{ \sum_{i=1}^l \alpha_i - \frac{1}{2} \sum_{i=1}^l \sum_{j=1}^l \alpha_i (y_i y_j K(\mathbf{x}_i, \mathbf{x}_j)) \alpha_j \right\}$$

subject to the constraints

$$\alpha_i \ge 0, \quad \sum_{i=1}^l \alpha_i y_i = 0$$

and so the classifier becomes

$$f(\mathbf{x}) = \operatorname{sign}\left(\sum_{i=1}^{l} (\alpha_i y_i K(\mathbf{x}, \mathbf{x}_i) + b)\right)$$

We used Radial Basis Function Kernel (RBF) with type

$$K(x, x') = \exp(\gamma ||x - x'||^2) \,\,\dot{\eta} \,\, K(x, x') = \exp\left(-\frac{||x - x'||^2}{2\sigma^2}\right)$$

Where γ is parameter that can be seen as the inverse of the radius of influence of the training set.

6.2. Support Vector Machine Performance

Therefore, support vector machines take as input some data coming from different groups and try to find the best hyper level, in order to separate them. Then those data are able to be correctly classified.

In our case, we build two groups of data, one with samples from area1 and one with samples from area2. We applied exhaustive search evaluation of the features RateNN, MeanNN, RMSSD, PNN50, SDNN, BBDC and SignDC. After testing the result of the classification using from 4 to 7 features, we ended using the results from RateNN,

MeanNN, PNN50, SDNN and SignDC. The classifier took as input all the above data, and with 10-fold cross validation, stored each time a train and a test set. SVM gets

trained with the train set and then predicts the class of the test set.

We tried both linear and RBF function for our experiments and repeat our experiment for 250 times, for each function. When using the RBF kernel function we had to set the cost and sigma (σ) parameters. As we mentioned before the cost parameter (C) trades off misclassification of training examples against simplicity of the decision surface. A low C makes the decision surface smooth, while a high C aims at classifying all training examples correctly by give the model freedom to select more samples as support vectors. The sigma parameter defines how far the influence of a single training example reaches, with low values meaning 'far' and high values meaning 'close'. We tried different values for these parameters and we ended using C= 0.9 and sigma= 0.2.

We counted the performance of the SVM using accuracy, sensitivity and specificity. Sensitivity measures the proportion of positives that are correctly identifies as such and specificity measures the proportion of negatives that are correctly identified as that. In our case we define as positive the sample from area3 and respectively as negative the data from area1. So accuracy is the proportion of total true results among the number of all the cases examined, true and false.

The Linear function the mean value of success was 71.78% with standard deviation of 0.075. For the RBF function the mean value of success was 73.5% with standard deviation 0.071. Follows a table with the results of our SVMs performance.

SVM Classifier	Accuracy	Sensitivity	Specificity
Linear	71,80%	73,60%	71,10%
RBF	73,50%	75,20%	68,90%

Table 6-1 Classification Results for the SVM

Finally, we normalized the values of the features inserted as input to the SVM in order to see if we could get a more accurate result. As we can see from the following table for the kernel classifier we get a significant improvement of about 3.74% in accuracy, 5.2% in sensitivity and 3.8% in specificity. The improvement on the result from the linear SVM is small as we can observe in the Table 6-2.

SVM Classifier	Accuracy	Sensitivity	Specificity
Linear	73.80%	74.12%	70.63%
RBF	77.24%	80.40%	72.70%

Table 6-2 Classification Results for the SVM, after value normalization.

CHAPTER 7. CONCLUSION

7.1 Conclusion

7.2 Future Work

7.1. Conclusion

The purpose of this work was to investigate how the rhythm of the heart, from patients suffering from ventricular tachycardia, change before an episode. We used heart rate variability methods, which are widely used in the study of heart signals, in order to study the signals in a different way, examining their change prior to a cardiac episode.

We implemented and computed HRV methods, from time and time-frequency domain and we also computed the deceleration capacity of the heart. All the above methods, were computed over time window intervals, we used non-overlapping and overlapping windows, and we focused our interest in the five first and five last windows. As area1 is the farthest from the episode and area3 is the nearest. We used signals from an online database, the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc., and from a NSVT database which was given to us.

First, we focused our interest in investigating the how each signal behave before a ventricular tachycardia episode. We can now conclude that some methods, as RateNN, MeanNN,SDNN and SignDC perform well in distinguishing area1 from area3. The p-values between those areas for the above metrics are smaller than 0.05 in

addition to those for RMSSD and BBDC. The same methods performed well for the NSVT signals as well.

Then, we compared the results based on age and weight for the samples of the Spontaneous Ventricular Tachyarrhythmia Database Version 1.0 from Medtronic, Inc. database. When comparing the patients by diving them to younger and older, we observed that while at the beginning of the signal their values might differ, when approaching the episode their values were really close. Also in this case the shape of the plots were almost the same. So we can conclude, that despite the age of the patient, prior to a ventricular tachycardia Incident heart behaves the same.

Splitting the patients using their weight we observed a similar behavior as before, but with the exception that in the cases of RMSSD and PNN50, both the values and the curve of the plots were different between the two groups of patients. Both methods focus on short term changes of the signal, since there are both based in differences and comparisons between adjacent beats.

Continuing we compared the VT and NSVT signals. In this case the results were different. With all the methods except SignDC, both the values and the curves of the plots were different. That fact led the p-values between area1 of the VT and area1 of the NSVT to be really small, as well as for area3 respectively.

Finally the results of the methods perform better for the VT signals, were given as input to a support vector machine. The performance of the machine was 73.5%.

7.2. Future Work

This work constitutes an early study of exploring the variability of heart prior to a ventricular tachycardia episode. Significant expansion of the thesis for the future, is to increase the number of data, in order to be able to generalize the results. Moreover, the study of longer duration data will be useful. Also other methods both from time

domain but also from frequency domain could apply on this data. More experimenting with the number and size of windows and also with the areas in focus could be also done. Another important factor is the contribution of specialized doctors, in order to explain those results and guide the researchers of this field.

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