A Stability Analysis in PTa Interval Dynamics Using ARX Model

Project Report

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TRIVANDRUM

CERTIFICATE

This is to certify that this project report entitled **"A Stability Analysis in PTa Interval Dynamics Using ARX Model"** is a bonafide record of the work done by **Niyas P**, under our guidance towards partial fulfilment of the requirements for the award of the Degree of **Master of Technology in Electronics and Communication with specialization in Signal Processing**, of the A P J Abdul Kalam Technological University during the year 2017-2019.

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My sincere thanks is extended to all the teachers of the department of ECE and to all my friends for their help and support.

Above all, I thank God for the immense grace and blessings at all stages of the project.

Niyas P

Abstract

Mathematical modeling of PTa and PP interval dynamics of ECG is of interest in finding the relationship between the heart rate and repolarization variability. The goal of this project was to assess the bounded input bounded output (BIBO) stability in PTa interval (PTaI) dynamics using autoregressive exogenous (ARX) model and to investigate the reason for causing instability in the atrial repolarization process.

The ECG data collected from normal synus rhythm (NSR) subjects and atrial tachycardia (AT) subjects were analysed. The ARX model showed better prediction of PTa interval dynamics in both groups. A more complex model is needed to better identify and characterize healthy heart dynamics.

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Chapter 1

Introduction

Alternans of action potential duration (APD) create a repolarization dispersion which causes atrial fibrillation directly. Initiation of cardiac arrhythmias is generally due to the unstable dynamics of APD at the cellular level which is responsible for the alternans in the repolarization phase. In atrial ECG event, the Ta wave is represented as the repolarization phase and the PTa interval is the measure of total atrial ECG component which is the counterpart of QT interval of the ventricles.

Abnormal atrial repolarization in ECG may be the key marker for different types of atrial arrhythmias. The atrial repolarization may be modified in its spatial orientation or in its duration by factors related to ischemic or necrotic phenomena and conditions that act upon the atria due to tachycardia, exercise and hyperthyroidism. These factors primarily affect repolarization, and the changes they produce are considered as primary alterations of atrial repolarization.

APD restitution refers to the cardiac action potential duration and its conduction velocity both depend on the previous diastolic interval (DI). The slope of APD restitution curve has been an indicator for the instability in the APD dynamics, and the cause of instability in the APD is mainly due to the occurrence of small perturbation in DI which results in a large (>1) APD restitution slope. However, earlier studies have reported that the prediction of arrhythmia occurrence is not only due to APD restitution slope but also been attributed to the presence of short-term memory.

The presence of PA in the ECG which is directly related with the unstable action potential dynamics could alter the normal PTa variability.These unstable segments of the PTa dynamics could initiate arrhythmias like sustained atrial tachycardia in acute myocardial

infarction (AMI) patients. The instability in the PTa dynamics could be a prognostic marker of the arrhythmia susceptibility for diseased human heart having prolonged PTa interval. There introduced a short term linear ARX model for the prediction of the unstable segments in the ventricular repolarization characteristics and demonstrated the effect of the PA in QT interval dynamics stability by calculating the frequency of PA in ECG by Imam et al [1].

In this project, the main aim is to assess the BIBO stability in PTaI dynamics in normal sinus rhythm (NSR) and atrial tachycardia (AT) subjects from the ECGs. A linear ARX model is used in this study to predict the PTaI dynamics. The functions are then transfer from time domain to their respective z-domain to predict the unsta ble segments. In addition, the ARX model complexity change is examined for the identification of the PTaI dynamics for NSR subjects in comparison to the AT patients.

1.1 Literature Survey

The instability in QT interval was studied by Xiaozhong Chen and Natalia A [2] by using ARX model in both AT and NSR subjects. This methodology can be applied in the clinic to monitor the development of QTI instability and thedevelopment of arrhythmia risk.

The stability analysis in PTa interval was studied by J. Sivaraman [3] by usingARX model in both AT and NSR subjects. In the study, it is found that the presence of unstable segments in the AT patients was generally due to premature activation. But the results of this study showed that similar number of unstablesegments was seen for both the AT and NSR subjects due to the HRV.

Restitution properties and occurrence of ventricular arrhythmia in LQT2 type of long QT syndrome was studied by S. Yamauchi et al. The aim of the study was to clarify the ventricular tachy-arrhythmia mechanism induced by the IKr-blocking agent E4031, simulating the LQT2 form. Electrophysiological properties were examined in 13 canines before and after administration of E4031. Thirty-six needle electrodes were inserted into the anterior left ventricular wall. From each needle, local unipolar electrograms were obtained from four intramural sites. Activation time (AT) and activation-recovery interval (ARI) were measured. To evaluate the susceptibility to ventricular arrhythmia, intramural ARI

dispersions and the restitution relationship between ARI and diastolic interval were calculated. After E4031 administration, ARI prolonged uniformly in each myocardial layer. However, ARI dispersion was not augmented compared with control. The slope of the ARI restitution curve after E4031 was significantly steeper than control. A steep slope may result from augmented ARI alternans. In 11 of the 13 canines, ventricular tachyarrhythmia was induced by programmed stimulation after E4031, whereas no arrhythmia was induced by the same protocol in control. Steepness of electrical restitution may play a major role in arrhythmogenicity in LQT2 hearts.

Relation between ventricular repolarisation duration and cardiac cycle length during 24-hour Holter recordings was studied by M. Merri et al. The study was based on the findings in normal patients and patients with long QT syndrome. A computer algorithm was developed to quantify the RTm and preceding RR intervals for each of more than 50,000 beats on 24-hour ambulatory electrocardiographic (Holter) recordings to evaluate the dynamic relation between repolarization duration and cycle length. The relation of RTm to the preceding RR interval (RTm/RR slope) was determined by the best-fit linear regression equation between these two parameters. Eleven normal subjects and 16 patients with long QT syndrome (LQTS) were investigated. Six of the normal subjects had Holter recordings obtained before and after beta-blocker therapy. beta- Blockers were associated with a significant (p = 0.005) reduction in the RTm/RR slope from 0.13 +/- 0.02 to 0.10 +/- 0.02. The mean value of the RTm/RR slope was significantly (p = 0.003) larger in the LQTS patients (0.21 +/- 0.08) than in normal subjects (0.14 +/- 0.03).These findings indicate that 1) quantification of the dynamic relation between ventricular repolariza tion and RR cycle length can be obtained on a large number of Holter-recorded heart beats; 2) beta-blockers reduce the RTm/RR slope in normal patients; and 3) LQTS patients have an exaggerated delay in repolarization at long RR cycle lengths.

Chapter 2

System Development

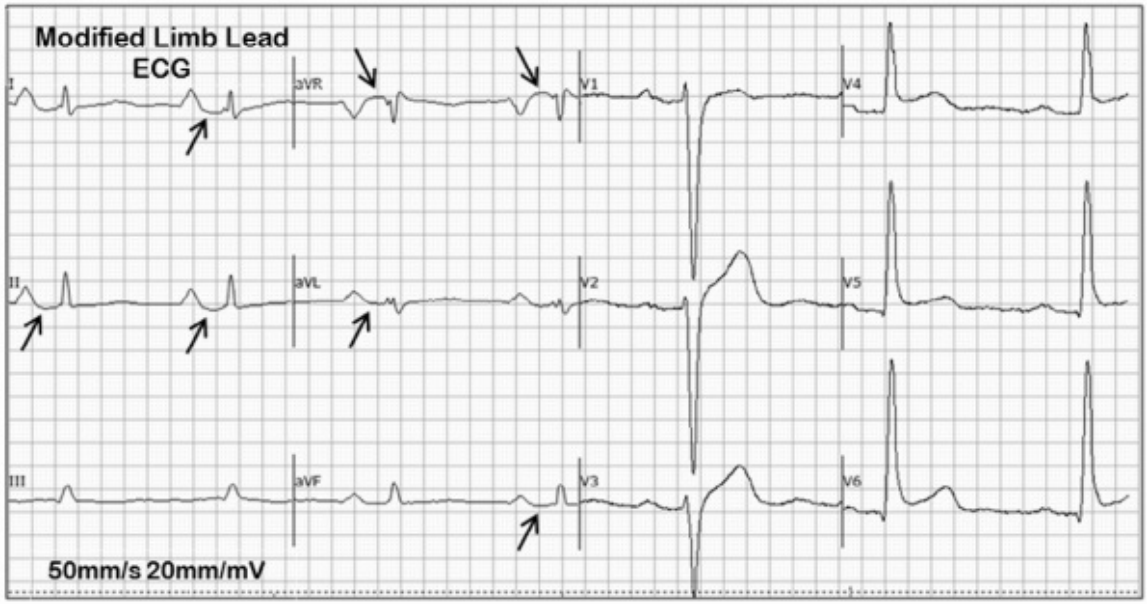
The methodology for identifying the instability in QTI dynamics of both groups is carried out in two parts. First, a mathematical model is represented as dependence of each PTaI on several prior PTaIs and PPIs. Second, the instability in PTaI dynamics of the the model is determined in the z-domain.

2.1 ECG data acquisition and analysis

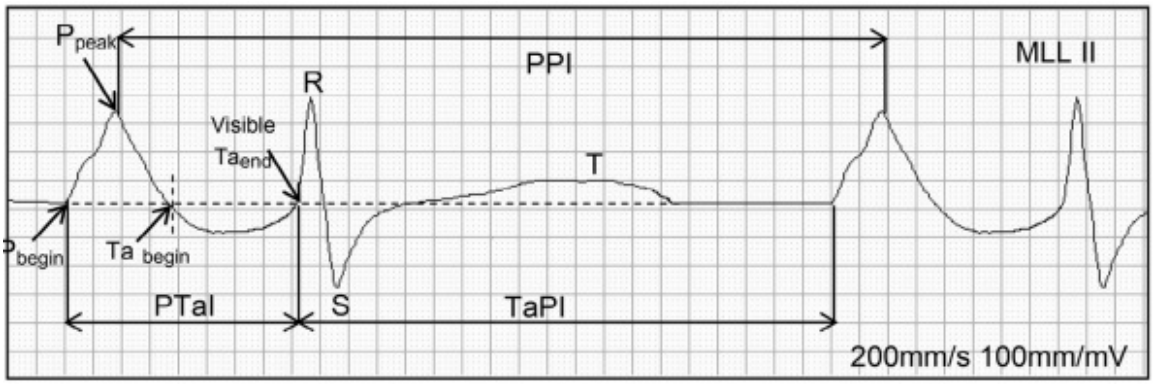
The MLL (Modified Limb Lead) system was used to record ECGs in the present study[1]. Five minute long, MLL ECGs (in lead II configuration Fig. 2.1) were recorded at a standard ECG paper speed of 25 mm/s and 10 mm/mV in supine position in NSR and AT subjects using a digital electrocardiograph (EDAN SE-1010 PC ECG system, EDAN Instruments, Inc.,) operating at 1000 samples per second, with a frequency response of 0.05 Hz to 150 Hz. ECGs could be printed at variable gain from 2.5 mm/mV to 100 mm/mV and variable paper speed of 5 mm/s to 200 mm/s for the better delineation of ECGs. All the ECGs were recorded and transferred to a computer and stored for subsequent off-line processing. The digital data analysis of ECGs was performed using MATLAB (R 2017a) for Windows. Each 5 min ECG segments were then divided into 1 min long segments having 5 segments for each subject like the method presented by Chen et al. [2] and Imam et al. [1]. The analysis of HRV between the two groups was measured and analyzed separately for comparative purpose. The PTa interval, TaP interval (TaPI) and the total PP interval (PPI) were measured using the smart ECG measurement and interpretation programs of EDAN ECG

[1]Data collected by Sivaraman et al. is used in the project

machine in the present study, where PPI = PTaI + TaPI.



(a)



(b)

Figure 2.1: (a) Modified limb lead ECG of a sinus rhythm subject clearly shows the presence of atrial Ta wave as a depression in the PR segment of leads I, II and aVL with the corresponding reciprocal elevation in the lead aVR. (b) Modified limb lead ECG of a subject in sinus rhythm and the annotations of P_{begin}, Ta_{begin},P_{peak}, visible Ta_{end}, TaPI and PPI. The ECG is replayed in 200 mm/s and 100 mm/mV for better delineation. One box has a width of 25 ms and a height of 0.05 mV.

2.2 Definitions

In the MLL system ECG trace, the beginning of the P wave is denoted as P_{begin} and the peak of the P wave is denoted as P_{peak}. The end of the P wave was defined as the beginning of the Ta wave (Ta_{begin}) at which the ECG trace crossed the isoelectric line [4]. The beginning of the Ta wave is denoted as Ta_{begin} and the visible end of Ta wave is denoted as visible Ta_{end}. The interval from the P_{begin} to the visible Ta_{end} was defined as the P-Ta interval. The Ta_{begin} to the visible Ta_{end} was defined as the visible Ta duration. All durations were annotated using the smart ECG measurement and interpretation programs of EDAN ECG machine to obtain PTaI,TaPI, and PPI as illustrated in Fig. 2.1.

2.3 PTa-PP ARX Model Formation

The ARX model for the PTaI dynamics is established using the system identification techniques.The dependence of the PTaI on the previous PTaIs and PPIs, an ARX model is developed for each 1 min ECG segment. The model equation is given by:

$$PTaI_n = \sum_{i=1}^{M} a_i' \times PTaI_{n-i} + \sum_{i=1}^{N} b_i' \times PPI_{n-i} \tag{2.1}$$

Where n is the beat number in 1 min ECG segment; PTaI and PPI are two discrete-time signals of the same length. $PTaI_n$, $PTaI_{ni}$, and PPI_{ni} are the values of the signal for beat n and n i respectively. The weight constants a_i and b_i for each preceding PTaI and PPI, respectively, contribute to $PTaI_n$. M and N represent the model order or the model parameters i.e., number of poles and zeros. The autoregressive term is $\sum_{i=1}^{M} a_i' \times PTaI_{n-i}$, whereas the term $\sum_{i=1}^{N} b_i' \times PPI_{n-i}$ represents the exogenous input. In Eq. 2.1, we used PPI instead of TaPI, because TaPI is affected by the preceding PTaI, and thus it is not an independent exogenous input. In this study, we have used M = N as described by Imam et al. which indicates that the memory effect of heart rate and repolarization were considered in the model for prediction of PTaI dynamics. The parameter of each ARX model is evaluated using the System Identification Toolbox functions in MATLAB 9.2 (R2017a).

2.4 Detection of stability in PTaI dynamics

To detect and to determine the stability of the ARX model, the a_i and b_i coefficients are redefined and Eq. 2.1 can be rearranged as:

$$a_0{}'PTaI_n - \sum_{i=1}^{M} a_i{}'a_0{}' \times PTaI_{n-i} = \sum_{i=1}^{N} b_i{}'a_0{}' \times PPI_{n-i} \tag{2.2}$$

$$a_0{}'PTaI_n + \sum_{i=1}^{M} (-a_i{}'a_0{}') \times PTaI_{n-i} = \sum_{i=1}^{N} b_i{}'a_0{}' \times PPI_{n-i} \tag{2.3}$$

Assuming, $a_0{}' = a_0$; $-a_i{}'a_0{}' = a_i$; $b_i{}'a_0{}' = b_i$

$$a_0 \times PTaI_n + \sum_{i=1}^{M} a_i \times PTaI_{n-i} = \sum_{i=1}^{N} b_i \times PPI_{n-i} \tag{2.4}$$

$$\sum_{i=0}^{M} a_i \times PTaI_{n-i} = \sum_{i=1}^{N} b_i \times PPI_{n-i} \tag{2.5}$$

Eq. 2.5 can be expanded as:

$$\begin{aligned} &a_0 PTaI_n + a_1 PTaI_{n-1} + ... + a_M PTaI_{n-M} \\ &= b_1 PPI_{n-1} + b_2 PPI_{n-2} + ... + b_N PPI_{n-N} \end{aligned} \tag{2.6}$$

Eq. 2.6 is the discrete-time expanded form of Eq. 2.5. To study about stability of the PTa-PP ARX model, Eq. 2.6 is transformed into z-domain and represented in Eq. 2.7. For each iteration of the model, the value of M was determined by increasing it from 1 for each step. The value of M was examined for each step whether the PTaI dynamics was accurately predicted in the minECG. The minimum number of poles required to detect the unstable segments in the PTaI is defined as M_{min} (low-order model), and the number of poles needed to achieve the predefined prediction capability is defined as M_{max} (high-order model). The value of M is increased from 1 sequentially up to the value when the model became unstable for the first time is calculated as M_{min}. M_{max} is the first value of M where the model achieved a predefined prediction value of the PTaI. In this study, the predefined accuracy of the mean square error between the predicted value and the measured PTaI value is smaller than 6 ms^2.

2.5 BIBO stability analysis in PTaI dynamics

The BIBO stability criterion was carried out for the ARX model of each ECG segments of the NSR subjects and AT patients. The ARX model was transformed from the time domain into the z-domain, where z is a complex number. The z transform is then applied to Eq. 2.6, resulting in:

$$\begin{aligned} &a_0 PTaI(z) + a_1 z^{-1} PTaI(z) + ... + a_M z^{-M} PTaI(z) \\ &= b_1 z^{-1} PPI(z) + b_2 z^{-2} PPI(z) + ... + b_N z^{-N} PPI(z) \end{aligned} \tag{2.7}$$

The transfer function representation of Eq. 2.7 is given in Eq. 2.8.

The equation 2.1 can be transformed to z domain as:

$$H(z) = \frac{PTaI(z)}{PPI(z)} = \frac{b_1 z^{-1} + b_2 z^{-2} + ... + b_N z^{-N}}{a_0 + a_1 z^{-1} + ... + a_M z^{-M}} \tag{2.8}$$

The factorized form of Eq. 2.8 to represent the poles (α_M) and zeros (β_N) of the model is given in Eq. 2.9 using which the polezero plot is illustrated. The above equation can be represented in the factorized form as:

$$H(z) = \frac{PTaI}{PPI} = g\frac{(z-\beta_1),(z-\beta_2),...,(z-\beta_M)}{(z-\alpha_1),(z-\alpha_2),...,(z-\alpha_M)} \tag{2.9}$$

Eq. 2.9 is the transfer function H(z) of the ARX model in the z-domain. Where β_1,.....,β_M are the zeros and α_1,.....,α_N are the poles and g is the constant. Pole-zero cancellation occurs when they are equal. In this study, if the difference between a pole and a zero is smaller than 0.05, then a pole is practically canceled by a zero. If any pole magnitude is greater than 1 (i.e. $\mid pole \mid > 1$) and when at least one pole was found to be outside the unit circle (i.e. $\mid z \mid = 1$) in the pole zero map, the model was considered unstable.

Chapter 3

Results and Discussion

3.1 Results

Using the MLL ECG recordings from both groups, an ARX model was constructed for each minECG. The accuracy of the ARX model was studied by predicting the each value of PTaI in minECG. The atrial rate, duration of PPI, PTaI and TaPI of both the groups are shown in Table 3.1. Using the PPI of the minECG as input, the output of the model was computed for each subjects in this study. The ARX model output and the prediction error for an individual AT patient are shown in Fig. 3.1.

Table 3.1: Statistical summary of PPI, PTaI and TaPI of healthy subjects and AT patients.

Measurement		Sinus rhythm subjects	Atrial tachycardia patients
Heart rate	bpm	73 $\pm$ 2.41	110$\pm$ 7.61
PPI	ms	827$\pm$ 76.15	548 $\pm$ 107.09
PTaI	ms	209$\pm$ 12.23	163 $\pm$ 14.38
TaPI	ms	618$\pm$ 70.63	385$\pm$ 95.18

The ARX model was able to predict the measured PTaI dynamics accurately in one of the AT patients for $M_{max} = 14$ as shown in Fig. 3.1b.While for $M_{min} = 6$, the model was not able to predict with the predefined mean square error of $5ms^2$ as shown in Fig. 3.1a. The dependence of the prediction error on M for the same minECG is shown in Fig. 3.1c. The same ARX model was also used to predict the measured PTaI dynamics in an individual NSR subject accurately for $M_{max} = 15$ and did not predict accurately for $M_{min} = 6$ as shown in Fig. 3.2b and 3.2a respectively. The dependence of the prediction

error on M is shown in Fig. 3.2c. The values of Mmin and Mmax for both groups are shown in Table 3.2

Table 3.2: Model order values of AT and NSR groups.

Feature	Atrial tachycardia (AT) subjects	Normal sinus rhythm (NSR) subjects
M_{min}	11 $\pm$ 9.69	12 $\pm$ 12.4
M_{max}	11$\pm$ 11.84	14.2$\pm$ 1.36
Values are presented as mean $\pm$ S.D		

The pole zero plots for the same minECG was obtained in the z domain.The stability analysis of the PTaI in the z domain for minECG of the same AT patient and NSR subject is shown in Fig. 3.3. The PTaI dynamics of this minECG for both groups are assessed as unstable, because the poles are outside the unit circle as seen in Fig. (3.3a, 3.3b) for M = 6 and M = 14 for AT patient and M = 6 and M = 15 for NSR subject in Fig. (3.3c, 3.3d).

3.2 Discussions

3.2.1 BIBO stability analysis in PTa-PP model

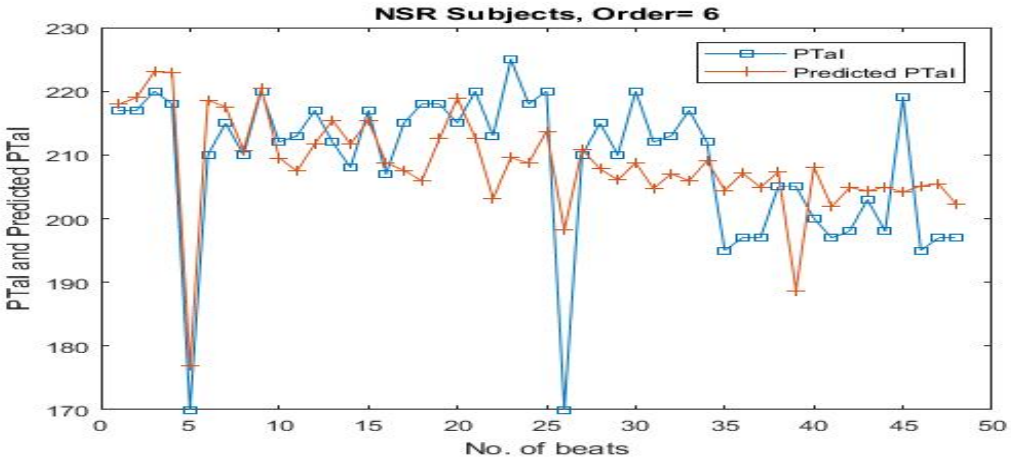
The autoregressive model used by Chen et al. [2] was aimed for the stability analysis of ventricular repolarization process with RR interval used as the exogenous input in the model. In the study done by Chen et al., the authors did not consider any additional noise term in their model that may induce instability in the QTI dynamics. In the project, the same methodology was used to predict the BIBO stability in the atrial repolarization process with PP interval as the exogenous input in the PTa-PP model.The derived ARX model was able to predict the PTaI dynamics in NSR and AT subjects, and the model was validated with the measured PTaI and PPI, and the mean square error was found to be within the tolerance ($6ms^2$).

3.2.2 Effect of heart rate variability on the PTaI dynamics

In the project, the ARX model was used to study the effect of HRV on PP interval and PTa interval in NSR subjects and AT patients. Since the PTa interval affects the subsequent TaP interval, we studied the HRV based on analyzing the PP interval which includes both the TaP and PTa interval within it. Several previous studies have established that QTI is not only affected by heart rate variability, and other factors like respiration, temperature, gender, age, genetic profile and autonomic nervous system have an effect on the QTI [5, 6]. Similar to the QTI variability, Debbas et al. [4] studied the effects of the sinus rate, pacing and drugs on the Ta wave in heart block patients and noted the variations in the PTaI dynamics.

Recently, Sivaraman et al. [7] studied the P and Ta wave morphology in healthy subjects and noted that the increase in age prolonged the P and Ta wave duration and increase in the heart rate shortened the observable P-Ta interval in the healthy subjects. From the results of this study, it is evident that the PAs are not the only reason in causing the unstable segments in NSR subjects likewise in the AT patients. Seen in the light of these findings, it is obvious that the cause of unstable segments of PTaI in NSR subjects is mainly due to the HRV and also due to the other intrinsic factors as described in Refs. [5, 6]. Acharya et al. documented that healthy heart showed high HRV and more asymmetry compared with the diseased heart. Perhaps this could be the reason why the ARX model in this study actually needed high-order model (Mmax) to predict the PTaI dynamics accurately in NSR subject than the AT patients. Since the healthy heart involves intricate dynamics, more complex model is required to understand such effect on PTaI dynamics.

From this preliminary investigation, it is found that the presence of PA might not be the only factor for causing the instability in PTaI in NSR subjects. The instability in NSR subjects is mainly due to high HRV within the subject which required high-order model to detect the instability. Further analysis to find the prediction capability of the ARX model was achieved by increasing the model order in predicting the PTaI dynamics in both groups.



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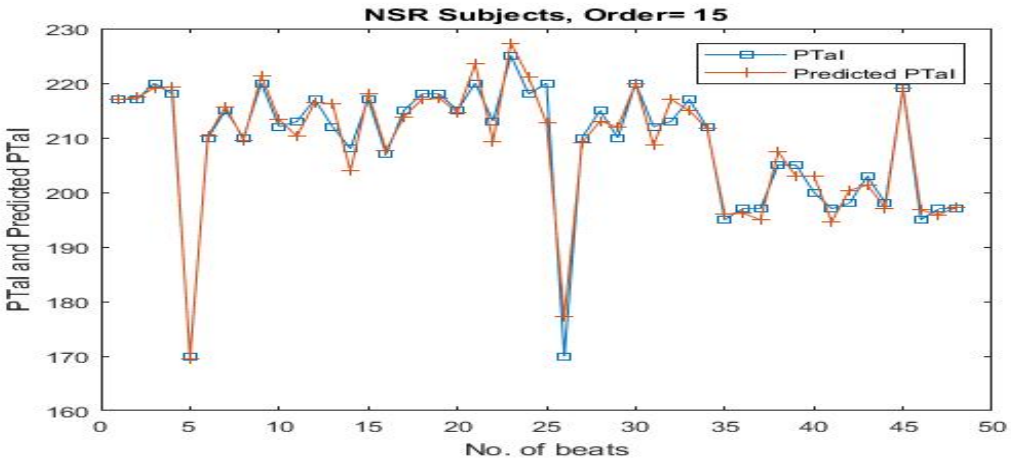

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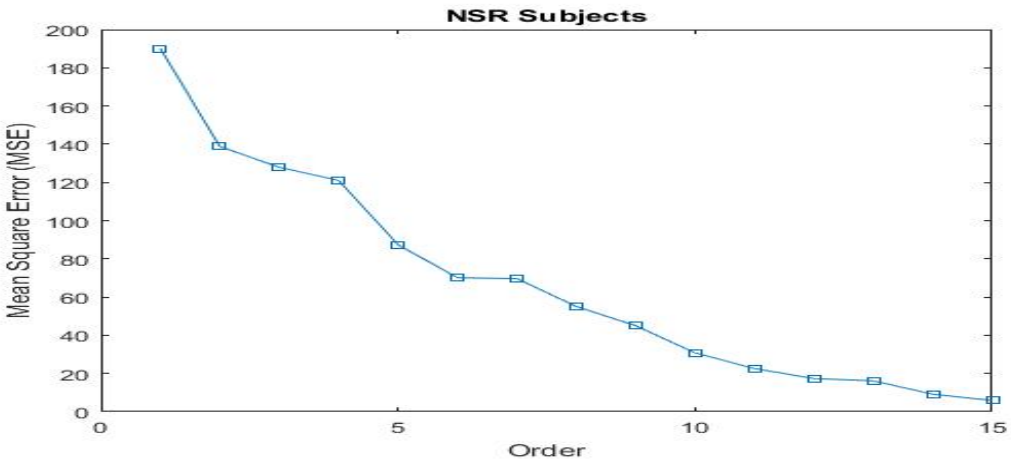
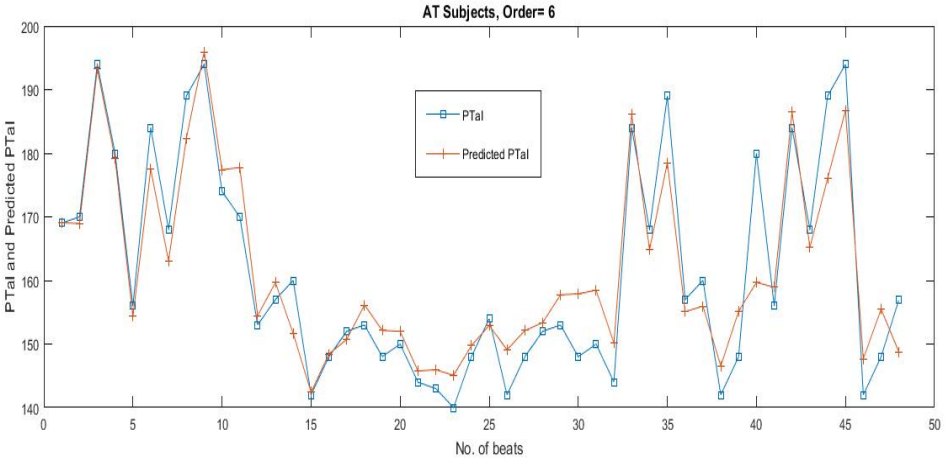


(c)

Figure 3.1: Predicted PTaI dynamics of a minECG for an individual NSR subject by ARX model. (a) PTaI dynamics extracted from the minECG for $M_{min} = 6$. (b) PTaI dynamics extracted from the minECG for $M_{max} = 15$. (c) The dependence of the prediction error on M for the same minECG.



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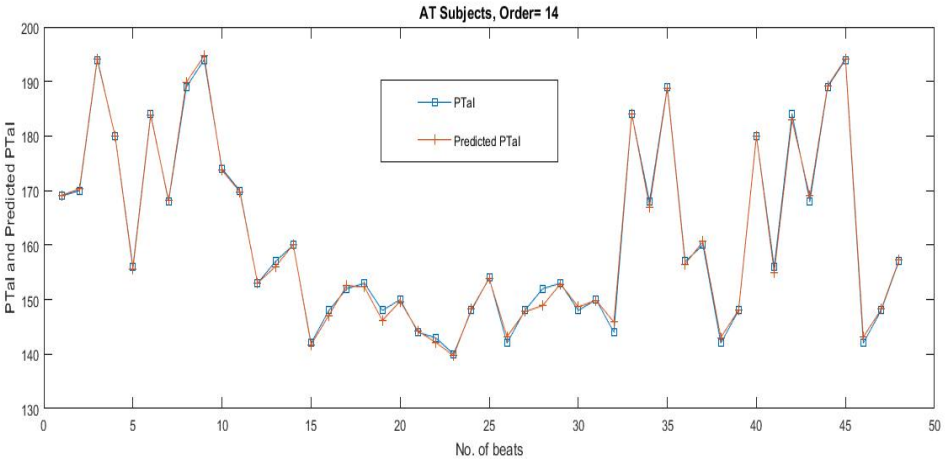

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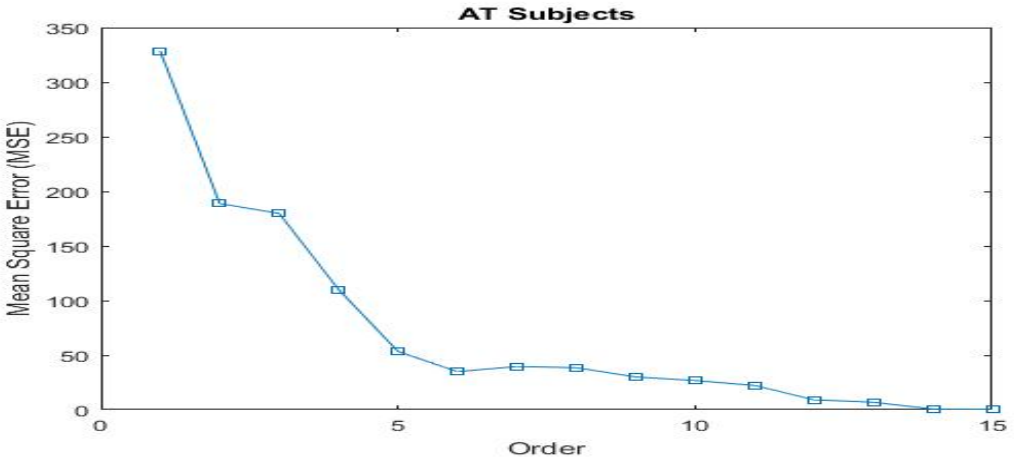

(c)

Figure 3.2: Predicted PTaI dynamics of a minECG for an individual AT patient by ARX model. (a) PTaI dynamics extracted from the minECG for $M_{min} = 6$. (b) PTaI dynamics extracted from the minECG for $M_{max} = 14$. (c) The dependence of the prediction error on M for the same minECG.

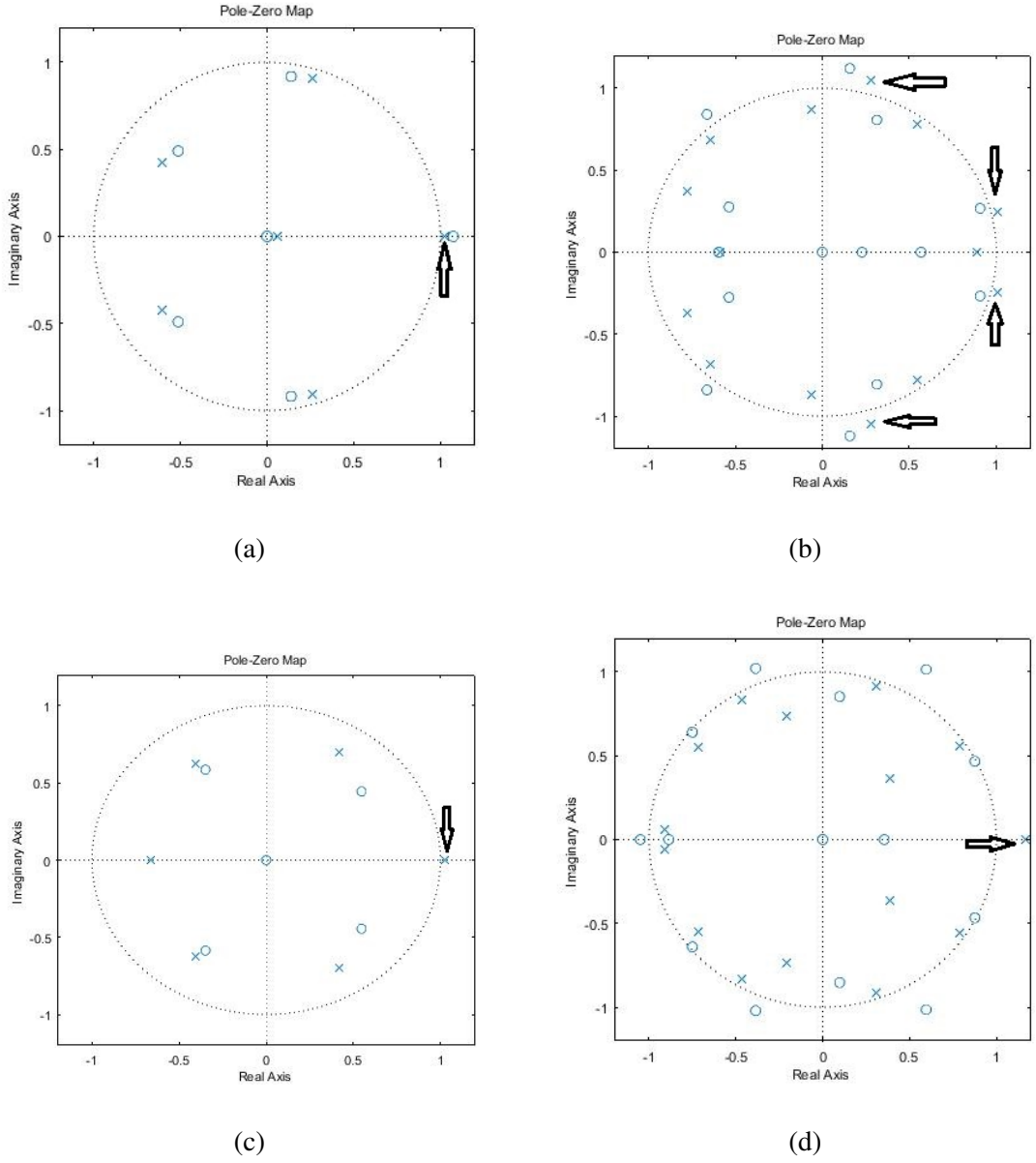

Figure 3.3: Pole-zero plots of minECG for an individual AT and NSR subject using ARX model. (a, b) Pole-zero plot in AT patient for $M_{min} = 6$ and $M_{max} = 14$. (c, d) Pole-zero plot in NSR subject for $M_{min} = 6$ and $M_{max} = 15$.

Chapter 4

Conclusion

The derived ARX model predicted the PTaI dynamics in NSR and AT subjects, and the model was validated with the measured PTaI and PPI. It is found that the presence of unstable segments in the AT patients was generally due to premature activation. Similar number of unstable segments was seen for both the AT and NSR subjects. The unstable segments in NSR subjects are mainly due to the HRV within the subjects. Obviously, future larger studies using more complex models such as ARMAX are needed to shed light on the prediction of the PTaI dynamics and the presence of PAs in healthy heart dynamics.

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Appendix A

Matlab Code

```
clear all;
close all;
clc;

%%NSR Subjects
%minECG
ny1=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'B2:B49');
nu1=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'C2:C49');
ndata1=iddata(ny1,nu1,1/1000);

ny2=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'F2:F49');
nu2=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'G2:G49');
ndata2=iddata(ny2,nu2,1/1000);

ny3=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'J2:J49');
nu3=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'K2:K49');
```

```
ndata3=iddata(ny3,nu3,1/1000);

ny4=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'R2:R49');
nu4=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'S2:S49');
ndata4=iddata(ny4,nu4,1/1000);

ny5=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'V2:V49');
nu5=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',2,'W2:W49');
ndata5=iddata(ny5,nu5,1/1000);

%5minute ECG
nyy=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',3,'A2:A241');
nuu=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',3,'B2:B241');
ndataa=iddata(nyy,nuu,1/1000);
%%Tachycardia Subjects
%minECG
tu1=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'B2:B49');
ty1=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'C2:C49');
tdata1=iddata(ty1,tu1,1/1000);

tu2=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'F2:F49');
ty2=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'G2:G49');
```

```
tdata2=iddata(ty2,tu2,1/1000);

tu3=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'J2:J49');
ty3=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'K2:K49');
tdata3=iddata(ty3,tu3,1/1000);

tu4=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'N2:N49');
ty4=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'O2:O49');
tdata4=iddata(ty4,tu4,1/1000);

tu5=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'R2:R49');
ty5=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',4,'S2:S49');
tdata5=iddata(ty5,tu5,1/1000);

%5 minute ECG
tuu=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',5,'A2:A241');
tyy=xlsread('C:\Users\lashe\Desktop\Niyas\M2SP\Mini_Project\
   ECG_Data\PTa_interval_Original.xls',5,'B2:B241');
tdataa=iddata(tyy,tuu,1/1000);


%ARX Models
for i=1:15
    tmodel1(1,1,i)=arx(tdata1,[i i 0]);
    [y,fit,x0]=compare(tdata1,tmodel1(1,1,i));
```

```
ty1p(:,:,i)=y.y;
tfit1(i)=fit;
tmse1(i)=immse(ty1p(:,:,i),ty1);

tmodel2(1,1,i)=arx(tdata2,[i i 0]);
[y,fit,x0]=compare(tdata2,tmodel2(1,1,i));
ty2p(:,:,i)=y.y;
tfit2(i)=fit;
tmse2(i)=immse(ty2p(:,:,i),ty2);

tmodel3(1,1,i)=arx(tdata3,[i i 0]);
[y,fit,x0]=compare(tdata3,tmodel3(1,1,i));
ty3p(:,:,i)=y.y;
tfit3(i)=fit;
tmse3(i)=immse(ty3p(:,:,i),ty3);

tmodel4(1,1,i)=arx(tdata4,[i i 0]);
[y,fit,x0]=compare(tdata4,tmodel4(1,1,i));
ty4p(:,:,i)=y.y;
tfit4(i)=fit;
tmse4(i)=immse(ty4p(:,:,i),ty4);

tmodel5(1,1,i)=arx(tdata5,[i i 0]);
[y,fit,x0]=compare(tdata5,tmodel5(1,1,i));
ty5p(:,:,i)=y.y;
tfit5(i)=fit;
tmse5(i)=immse(ty5p(:,:,i),ty5);



nmodel1(1,1,i)=arx(ndata1,[i i 0]);
[y,fit,x0]=compare(ndata1,nmodel1(1,1,i));
```

```
    ny1p(:,:,i)=y.y;
    nfit1(i)=fit;
    nmse1(i)=immse(ny1p(:,:,i),ny1);

    nmodel2(1,1,i)=arx(ndata2,[i i 0]);
    [y,fit,x0]=compare(ndata2,nmodel2(1,1,i));
    ny2p(:,:,i)=y.y;
    nfit2(i)=fit;
    nmse2(i)=immse(ny2p(:,:,i),ny2);

    nmodel3(1,1,i)=arx(ndata3,[i i 0]);
    [y,fit,x0]=compare(ndata3,nmodel3(1,1,i));
    ny3p(:,:,i)=y.y;
    nfit3(i)=fit;
    nmse3(i)=immse(ny3p(:,:,i),ny3);

    nmodel4(1,1,i)=arx(ndata4,[i i 0]);
    [y,fit,x0]=compare(ndata4,nmodel4(1,1,i));
    ny4p(:,:,i)=y.y;
    nfit4(i)=fit;
    nmse4(i)=immse(ny4p(:,:,i),ny4);

    nmodel5(1,1,i)=arx(ndata5,[i i 0]);
    [y,fit,x0]=compare(ndata5,nmodel5(1,1,i));
    ny5p(:,:,i)=y.y;
    nfit5(i)=fit;
    nmse5(i)=immse(ny5p(:,:,i),ny5);
end

%Plots

figure(1)
```

```
x=linspace(1,15,15);
plot(x,nmse4,'-s');
xlabel('Order');
ylabel('Mean Square Error (MSE)');
title('NSR Subjects');

figure(2)
x=linspace(1,15,15);
plot(x,tmse1,'-s');
xlabel('Order');
ylabel('Mean Square Error (MSE)');
title('AT Subjects');

figure(3)
x=linspace(1,48,48);
plot(x,ny4,'-s',x,ny4p(:,:,6),'-+');
legend('PTaI','Predicted PTaI');
xlabel('No. of beats');
ylabel('PTaI and Predicted PTaI');
title('NSR Subjects, Order= 6');

figure(4)
x=linspace(1,48,48);
plot(x,ny4,'-s',x,ny4p(:,:,15),'-+');
legend('PTaI','Predicted PTaI');
xlabel('No. of beats');
ylabel('PTaI and Predicted PTaI');
title('NSR Subjects, Order= 15');

figure(5)
x=linspace(1,48,48);
plot(x,ty1,'-s',x,ty1p(:,:,6),'-+');
```

```
legend('PTaI','Predicted PTaI');
xlabel('No. of beats');
ylabel('PTaI and Predicted PTaI');
title('AT Subjects, Order= 6');

figure(6)
x=linspace(1,48,48);
plot(x,ty1,'-s',x,ty1p(:,:,14),'-+');
legend('PTaI','Predicted PTaI');
xlabel('No. of beats');
ylabel('PTaI and Predicted PTaI');
title('AT Subjects, Order= 14');
ident;
```