DOKUZ EYLÜL UNIVERSITY
GRADUATE SCHOOL OF NATURAL AND APPLIED SCIENCES

ARRHYTHMIA CLASSIFICATION WITH SOM

by
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ARRHYTHMIA CLASSIFICATION WITH SOM

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ARRHYTHMIA CLASSIFICATION WITH SOM

ABSTRACT

The electrocardiogram carries a lot of clinical information for a cardiologist, especially the width or duration of the waves in the ECG are widely used to define conduction in the heart and to stratify patients at risk of cardiac arrhythmia. The manual annotation to the waves is a strenuous task; as a result several automated methods have been developed to relieve the cardiologist.

This study presents an Artificial Neural Network using Self-Organizing Map architecture, the evaluation of its performance in the classification of QRS waves of the electrocardiogram (ECG) from patients with cardiac arrhythmias and the classification of data from MIT/BIH Arrhythmia Database.

Keywords: Arrhythmia, Self Organizing Map (SOM), Artificial Neural Network, Electrocardiogram (ECG)

SOM KULLANILARAK ARİTMİ SINIFLANDIRILMASI

ÖZ

Elektrokardiyogram bir kardiyolog için birçok klinik bilgi içerir. Özellikle ECG' deki dalgaların genlikleri ve oluşma süreleri, kalpteki iletimi tanımlamak ve aritmi riski olan hastaları saptamak için sıkça kullanılır. Bu yüzden kardiyologlara yardımcı olabilmek için birçok metot geliştirilmiştir.

Bu çalışmada Self-Organising Map yöntemi kullanılmıştır. Aritmisi olan hastalardan alınan ECG kayıtlarındaki R dalgalarının ve MIT/BIH Aritmi Veritabanı'ndan alınan ECG kayıtlarının sınıflandırılması yapılmış ve Self-Organizing Map yönteminin bu sınıflandırmadaki performansı ölçülmüştür.

Anahtar Sözcükler : Aritmi, Self Organising Map (SOM), Yapay Sinir Ağları, Elektrokardiogram (ECG)

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CHAPTER ONE
INTRODUCTION

1. Introduction

The heart is a pump that functions by pushing the blood through its four chambers. The blood is "pushed" through in a controlled sequence of muscular contractions. The sequence is controlled by bundles of cells which control the electrical activity of the heart. When the sequence is disturbed, heart arrhythmias occur.

Arrhythmias are abnormal rhythms of the heart. They cause the heart to pump blood less effectively. Most cardiac arrhythmias are temporary and benign. Most temporary and benign arrhythmias are those where your heart skips a beat or has an extra beat. The occasional skip or extra beat is often caused by these occasional changes can be brought on by strong emotions or exercises. Nonetheless, some arrhythmias may be life-threatening and require treatment. Arrhythmias are diagnosed by electrocardiogram (ECG) as they have differences in morphology. The electrocardiogram carries a lot of clinical information for a cardiologist, especially the width or duration of the waves in the ECG are widely used to define conduction in the heart and to stratify patients at risk of cardiac arrhythmia Mitchell et al., (1992). ECG classification has always been a very difficult task in the realization of computer aided ECG diagnosis. Computer-based methods for analysis and interpretation of electrocardiograms (ECG's) have been subject to intense research for nearly four decades. Artificial neural networks (ANN's) are one of the most recent techniques in this field and primarily have been considered for classification of ECG's into different diagnostic groups.

Baig et al., (2001) investigated a set of efficient methods to extract important features from the ECG data applicable in the localization of cardiac arrhythmia. They used SOM, LVQ and Beat Detection methods. The ECG data is digitizing at a frequency of 16 KHz to detect the pacemaker pulses digitally. The signal is then

downsampled to 250 Hz. Data is allowed to pass through a bandpass filters at 0.16 Hz-300 Hz. One filter is used for QRS & ST detection and one filter is used for QRS & ST classification. They have developed a novel method for arrhythmia localization from potential mapping data. The QRS integral maps that display only spatial information are based on the whole QRS complex, the SOM & LVQ approach uses both spatial and temporal QRS information.

Simelius et al., (1997) developed a novel method for ventricular tachycardia (VT) source localization using body surface potential mapping (BSPM). Kohonen self-organizing maps (SOM) are utilized for the classification of different types of VTs. The method is a two-step process. First, a large number of potential patterns from Vt, QRS complexes are presented to the SOM. Second, QRS time traces on this SOM are created to find the representative patterns for different types of V. The localization can be performed using these time traces as a reference. The presented method utilizes both spatial and temporal aspects of the recorded VT to create a representative pattern of tachycardia. The data was obtained from 27 patients with ventricular tachycardia; 21 were used for teaching set and 6 for the test set.

Braccini et al., (1997) presented a method for classifying ECG complexes from the MIT-BIH Arrhythmia Database based on Self-Organising maps (SOM). QRS complexes were extracted and RR intervals were calculated. The information contained in each beat was represented by the coefficients of the decomposition with the first three Hermite basis functions. These features in combination with measurements of RR intervals were clustered with a SOM. The resulting classifications performed by a more conventional method based on cross-correlation. The percentage of misclassifications with the SOM method was 2.4 % which was substantially lower than the 4.4 % of the correlation based method.

Prasad G. K., & Sahambi J. S., (2003) proposed a method to accurately classify ECG arrhythmias through a combination of wavelets & artificial neural networks (ANN). A set of discrete wavelet transform (DWT) coefficients, which contain the maximum information about the arrhythmia, is selected from the wavelet

decomposition. These coefficients in addition the information about RR interval (the difference between the present and previous QRS peaks) are fed to the back-propagation neural network which classifies the arrhythmias. The ECG data is taken from standard MIT-BIH Arrhythmia database. The proposed method is capable of distinguishing the normal sinus rhythm and 12 different arrhythmias. The overall accuracy of classification of the proposed approach is 96.77%. The result of the analysis was found to be more accurate than those of the existing methods. To check the robustness of the algorithm, three types of noise, i.e., muscle noise, power-line interference and base-line wander, were added with SNR values ranging from 0 dB to 10 dB to the signal and the accuracy was found to be well within the clinical limits. It was observed that the effect of base-line wander on the accuracy of detection was less than the other disturbances.

Heidari et al., (1998) investigated the analysis of the sustained ventricular arrhythmias from the signal-averaged electrocardiogram (SAECG) using artificial neural network and fuzzy clustering algorithm. Patients with ventricular tachycardia and ventricular fibrillation have a potential for sudden death. After myocardial infraction the chance to get sustained ventricular tachycardia or ventricular fibrillation increases, thus reduction in number of sudden death requires advanced predictive procedures. In their study, frequency domain feature extraction, clustering and classification models are combined for providing an integrated system for the sustained ventricular arrhythmias. The radial basis function network and the fuzzy c-means algorithm for training clustering and classification were investigated. These techniques do not have limitation of the previous classical procedures. The value of sensitivity and specificity, on the data was used here, for the RBFN were found to be 92.3% and 71.4%, respectively, also the value of sensitivity and specificity for the FCM were found to be 84.6% and 71.4, respectively.

Szilagyi, L., (1998) studied on the application of the Kalman Filter in cardiac arrhythmia detection. Their main objective was to investigate whether the study of time varying spectra of the R-R interval time series can give any information about the identification of short segments of arrhythymia and the detection of the onset and

termination of such arrhythmia. The variability of the R-R intervals in the ECG signal contains valuable information about the various types of arrhythymia that may be present. It has been recently suggested, that the identification of cardiac arrhythymia might be possible by applying spectral analysis method. This study intends to investigate the efficiency of a spectral analysis method, namely the application of the Kalman filter identifier in the calculation of time varying spectra of the R-R interval time series. The efficiency of the method is tested using the MIT-BIH database and; in particular cases of bigeminy, trigeminy, second degree block and ventricular flutter have been tested. Tests have revealed that this technique in most cases can detect the onset of arrhythymia and can also identify the arrhythymia that is present.

Owis et al., (2002) presented a study of the nonlinear dynamics of electrocardiogram signals for arrhythymia characterization. The correlation dimension and largest Lyapunov exponent are used to model the chaotic nature of five different classes of ECG signals. The model parameters are evaluated for a large number of real ECG signals within each class and the results are reported. The presented algorithms allow automatic calculation of the features. The statistical analysis of the calculated features indicates that they differ significantly between normal heart rhythm and the different arrhythymia types and, hence, can be rather useful in ECG arrhythymia detection. On the other hand, the results indicate that the discrimination between different arrhythymia types is difficult using such features. The results of this work are supported by statistical analysis that provides a clear outline for the potential uses and limitations of these features.

Osowski, S., & Linh, T. H., (2001) studied on the application of the fuzzy neural network for electrocardiographic (ECG) beat recognition and classification. The new classification algorithm of the ECG beats, applying the fuzzy hybrid neural network and the features drawn from the higher order statistic has been proposed in this study. The cumulants of the second, third and fourth orders have been used for the feature selection. The hybrid fuzzy neural network applied in the solution consists the fuzzy self-organizing subnetwork connected in cascade with the multilayer perceptron

working as the final classifier. The c-means and Gustafson-Kessel algorithms for the self-organization of the neural network have been applied. The results of experiments of recognition of different types of beats on the basis of the ECG waveforms have a confirmed good efficiency of the proposed solution. The investigations show that the method may find practical application in the recognition and classification of different type heart beats.

Hosseini et al., (2001) studied on a multi-stage network including two multilayer perceptron (MLP) and one self-organizing map (SOM) networks is presented. The input of the network is a combination of independent features and the compressed electrocardiogram (ECG) data. The proposed network as a form of data fusion performs better than using the raw data or individual features. They classified six common ECG waveforms using ten ECG records of the MIT/BIH arrhythmia database. An average recognition rate of 0.883 was achieved within a short training and testing time.

The objective of the present study is to classify arrhythmias by using Self-Organizing Map. It is carried out in two ways; arrhythmia classification and beat detection.

In Chapter 2, The Heart from an Electrical Perspective, ECG, in Chapter 3, Arrhythmias, in Chapter 4 Self-Organizing Map, in Chapter 5, Application and Results, in Chapter 6, Conclusion and Discussion will be mentioned.

CHAPTER TWO
THE ELECTRICAL ACTIVITY OF THE HEART

2. The Heart from an Electrical Perspective

The intrinsic heart rate (HR) generated by sinoatrial node in the absence of any neural or hormonal influence is about 100 to 120 bpm. In a healthy individual, the HR estimated at any given time represents the net effect of the parasympathetic (vagus) nerves, which slow HR, and the sympathetic nerves, which accelerate it. At rest, both sympathetic and parasympathetic nerves are tonically active with the vagal effects dominant.

The most obvious effect of vagal stimulation is to slow or even to stop the heart. The response time of the sinus node is very short and the effect of a single vagal impulse depends on the phase of the cardiac cycle at which it is applied. Thus, vagal stimulation results in an immediate response within one or two heart beats after its onset. After cessation of vagal stimulation, HR rapidly returns to its previous level. An increase in HR can also be achieved by reduced vagal activity or vagal block. Thus, any sudden changes in HR are parasympathetically mediated.

2.1. The Electroconduction System of the Heart

The heart possesses the property of autonomic and rhythmic conduction. It has the inherent ability to initiate and conduct impulses which stimulate muscular contraction. This ability is located in the specialized neuromuscular tissue known as the conduction system which is shown in Figure 2.1 consists of:

- The sinoatrial node (SA) node
- The internodal atrial pathways
- The atrioventricular (AV) node
- The bundle of His
- The right and left bundle branches, an

- The Purkinje system

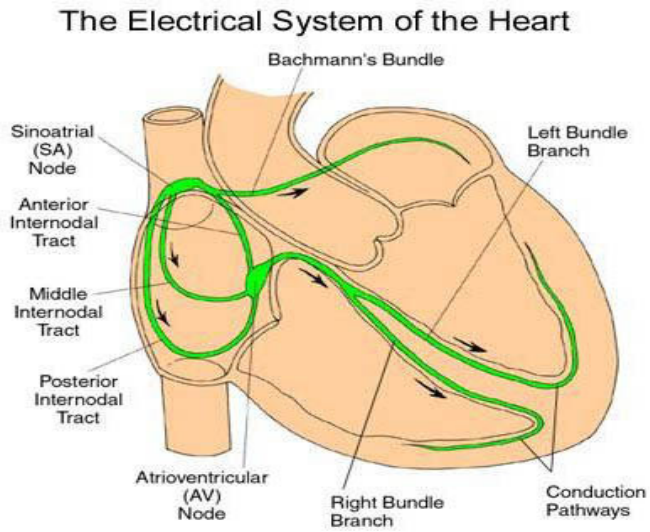

Figure 2. 1 The Electrical System of the Heart

SA Node

The heartbeat is normally controlled by rhythmic impulses which arise in the SA node, and the latter is therefore the cardiac pacemaker. It consists of a bundle of specialized neuromuscular tissue measuring approximately 5 x 200 mm which lies on the endocardial surface of the right atrium at the junction of the superior vena cava and right atrial appendage. The impulse then spreads through both atria, producing the P wave.

Internodal Atrial Pathways

Conduction through the atria occurs through 3 bundles of myocardium which contain Purkinje type fibers: (1) the anterior tract (Bachman) leaves the SA node in a forward direction and curves about the superior vena cava and the anterior wall of the right atrium. There it divides into 2 bundles of fibers, one entering the left atrium and the other coursing over the anterior portion of the interatrial septum descending obliquely behind the root of the aorta to enter the anterior-superior margin of the AV node. (2) The middle internodal tract (Wenckebach) leaves the posterior margin of

the SA node, curves behind the superior vena cava, and courses along the posterior portion of the interatrial septum to enter the superior margin of the AV node. (3) The posterior internodal tract (Thorel) leaves the posterior margin of the SA node and follows the course of the crista terminalis and eustachian ridge to enter the posterior margin of the AV node. Lateral extensions from this tract arborize over the dorsum of the right atrium. Between all 3 tracts there are interconnecting fibers that merge just above the AV node. Some of these fibers do not enter the AV node but bypass it; they can reenter the conducting system at a place distal to AV node.

AV Node

This bundle of specialized neuromuscular tissue measures 2 x 5 mm. It is located on endocardial surface on the right side of the interatrial septum just inferior to the opening of coronary sinus. The impulse which has spread through both atria enters the AV node and is normally delayed for approximately 0.07 s.

Bundle of His

The bundle of His is in direct continuity with the lower portion of the AV node. It is approximately 20 mm long and is located on the endocardial surface of the right side of the interatrial septum, immediately superior to the interventricular septum. The bundle of His is not a homogeneous mass of conducting tissue but consists of multiple individual longitudinal tracts. It is likely that specific tracts continue specified bundle branches.

Bundle Branches

The right bundle branch arises from the bundle of His and traverses the endocardial surface of the right side interventricular septum. Distally, the right bundle divides into 3 divisions (anterior, lateral, posterior). Purkinje fibers arborize from these and spread over the endocardial surface of the right ventricle and the distal portion of the interventricular septum.

On the left side of the septum, 3 fascicular radiations arise from the bundle of His. The more proximal is the left posterior fascicle, which spreads as a broad band of fibers over posterior and inferior endocardial surfaces of the left ventricle. Immediately distal to the origin of the posterior fascicle is the left anterior fascicle, which spreads as a narrower band of fibers over anterior and superior endocardial surfaces of the left ventricle. Separate fibers arise from the proximal portions of the left anterior and posterior fascicles and cover the endocardial surface of the left side of the interventricular septum. This is the septal fascicle. Those from the anterior fascicle enter the anterior and superior surfaces of the septum, and those from the posterior fascicle enter the posterior and inferior portions of the septum. Thus the bundle branch system consists of 4 fascicles:

- A right bundle branch
- A left posterior fascicle
- A left anterior fascicle and
- A left fascicle

The midportion of the interventricular septum is normally activated from left to right. Purkinje fibers arise more proximally from the divisions of the left bundle than from the right bundle branch and enter the left side of the septum, which they activate initially. This wave of excitation from left to right produces the initial negativity of the left ventricular cavity and the initial positivity of the right ventricular cavity. It results in a force oriented to the right and anteriorly.

Purkinje System

After traversing the right bundle branch and the left fascicular bundle branches, the impulse passes into the multiple ramifications of the Purkinje system which cover the subendocardial surfaces of both ventricles. The impulse then travels perpendicularly from the endocardial to the epicardial surface of the myocardium. It is the propagation of the impulses through the Purkinje system into the ventricular myocardium which produces the remainder of the QRS complex (after activation of

the interventricular septum from left to right). The anteroseptal region of the right ventricle is the earliest site of free wall ventricular activation. The posterobasal region of the left ventricle, the pulmonary conus, and the uppermost portion of the interventricular septum are activated last Gülsün, E., (2002).

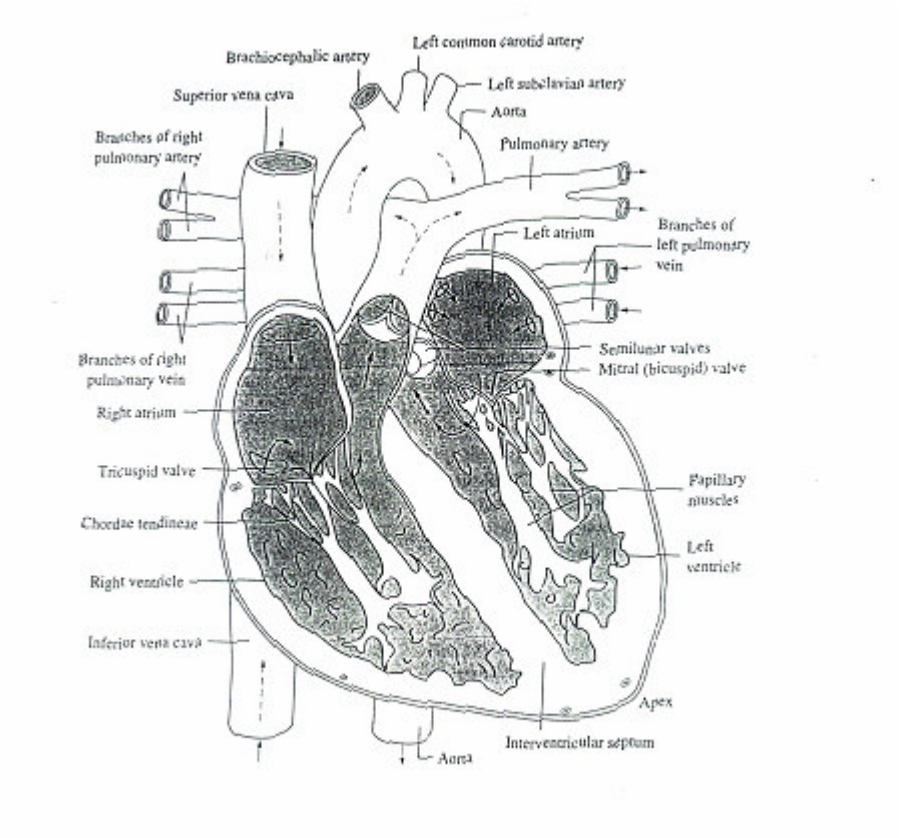

Figure 2. 2 Cross-sectional View of the Human Heart

2.1.1 Electrophysiology of the Heart

The following factors are involved in the genesis of the ECG:

- Initiation impulse information in the primary pacemaker (sinus node)
- Transmission of the impulse through the specialized conduction system of the heart
- Activation (depolarization) of the atrial and ventrical myocardium, and
- Recovery (repolarization) of all the above areas

In order for one to have an understanding of the ECG, it is necessary to have a basic knowledge of intracellular and surface potentials.

Intracellular Potentials

If one electrode is placed on the surface of a resting muscle cell and a second indifferent electrode is placed in a remote location, no electrical potential (i.e., zero potential) will be recorded because of the high impedance of the cell membrane. However, if the cell membrane is penetrated by a capillary electrode, a negative potential of about 90 millivolts (mV) will be recorded. This is known as the membrane resting potential (MRP). The major factor that determines the MRP is the gradient of the potassium ions (K^+) across the cell membrane. The intracellular concentration of K^+ is approximately 150 mEq/L, and the extracellular concentration is approximately 5 mEq/L. This K^+ gradient of 30:1 is sufficient to explain the recorded MRP (-90 mV). On the other hand, an opposite gradient exists for the sodium ions (Na^+). There is a relatively high extracellular Na^+ concentration in relation to intracellular Na^+ concentration. This Na^+ gradient, opposite in polarity to that of the K^+ gradient, does not appreciably after the MRP because the cell membrane in the resting state is 30 times more permeable to K^+ than Na^+.

At the onset of the depolarization of a cardiac muscle cell (eg, a ventricular muscle cell), there is an abrupt change in permeability of the cell membrane to sodium. Sodium ions (and calcium ions to a less degree) enter the cell and result in a sharp rise of intracellular potential to positivity (approximately 20 mV). This designated as phase 0 and represents the fast inward current typical of normal myocardial cells and Purkinje fibers. Pacemaker cells of the SA node and cells in the proximal region of the AV node are depolarized by a slow via sodium channels is inhibited can be depolarized by the slow inward current via calcium channels.

Following depolarization, there is a relatively slow and gradual return intracellular potential to MRP (phase 4). This is repolarization and is divided into 3 phases:

Phase 1: An initial rapid return of intracellular potential to 0 mV. This is largely the result of abrupt closing of the sodium channels. It has been suggested that chloride ions entering the cell may contribute to phase 1.

Phase 2: A plateau phase of repolarization owing to the slow entrance of calcium ions into the cell. These are the same channels that can result in the slow inward type of depolarization.

Phase 3: This represents the slow, gradual return of the intracellular potential to MRP. It results from extrusion of potassium ions out of the cell, which reestablishes the normal negative resting potential. However, the cell is left with an excess of sodium ions and a deficit of potassium ions. To restore the original ion concentration, a cell membrane sodium-potassium pump mechanism becomes effective. The energy required for this pump is derived from conversion of ATP to ADP. This pump removes sodium from the cell and permits potassium influx.

The summation of all phase 0 potentials of ventricular muscle cells produces the QRS complex. Phase 2 correlates with the ST segment and phase 3 with the T wave of the ECG.

The entire curve of intracellular potential is the monophasic action potential. The duration of this curve from the onset of depolarization to the termination of repolarization the duration of action potential.

The monophasic action potential curve of an atrial muscle cell is different from that of a ventricular muscle cell. Phase 4 (MRP) and phase 0 (depolarization) are similar, but the duration of repolarization, and hence the duration of action potential, is shorter in an atrial muscle cell. This is largely due to a shortening and steepening of the slope in phase 2.

The monophasic action potential curve of a cell in the sinoatrial (SA) node is markedly different from the above:

- There is a lower MRP (-60 to –70 mV) at the onset of diastole.
- A prepotential is present in diastole (phase 4). Instead of the MRP remaining at a constant level during diastole, as is typical of ventricular and atrial muscle cells, there is a gradual rise of the MRP during diastole. It is this prepotential that explains the automatic function of the sinus pacemaker.
- Depolarization is slower and does not reach sufficient positive potential to be recorded on a surface electrogram.
- The peak of the action potential is rounded, and repolarization is a single slow curve in which phases 1, 2 and 3 cannot be defined.

The major portion of the AV node does not have the property of prepotential. The configuration of the action potential of cells in the bundle of His and Purkinje fibers is similar to that of a ventricular muscle cell except that some degree of prepotential is present which allows these centers to assume pacemaker activity under appropriate conditions. The duration of action potential is longer in a Purkinje fiber than in any other site. This is due to prolongation of phases 2 and 3 and results in the U wave of the ECG.

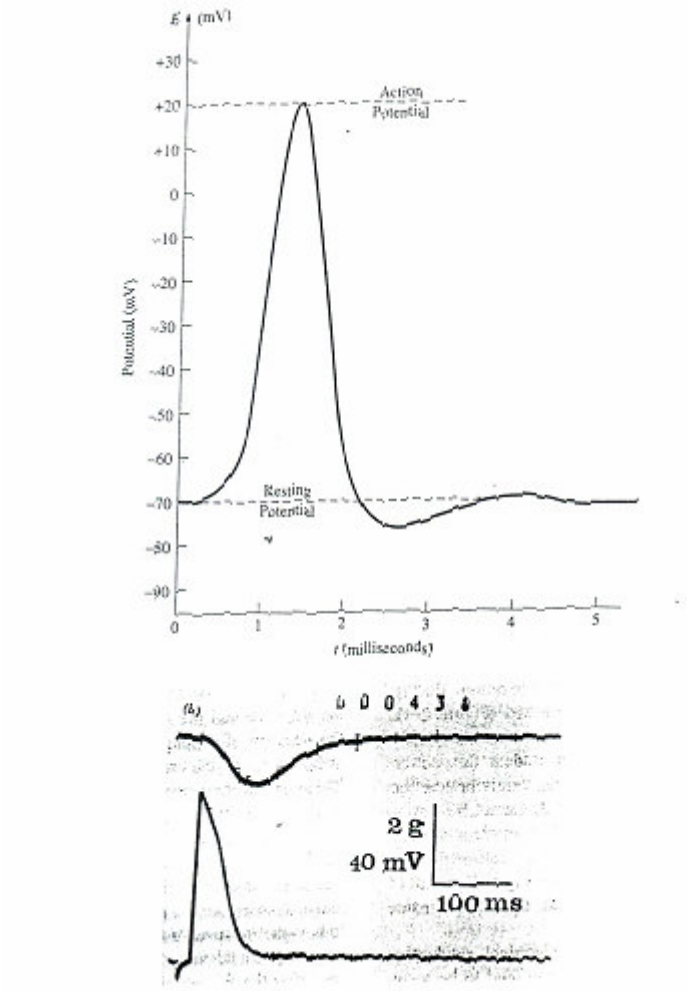

Figure 2. 3 Action potential duration with time: (a) typical cell action potential; (b) contraction (upper trace) and action potential (lower trace) from a Guinea Pig Myocardium

Conduction Velocity

The speed at which the electrical potential spreads through the heart varies considerably depending upon the inherent properties of different portions of the specialized conduction system and the myocardium. Velocity is most rapid in Purkinje fibers and slowest in the midportion of the AV node. The following figures are averages of many experiments done on various animal species: SA node, 0.05 m/s; atrial muscle, 0.8-1 m/s; AV node, 0.05 m/s; bundle of His, 0.8-1 m/s; Purkinje fiber, 4 m/s; and ventricular muscle, 0.9-1 m/s.

Excitation & Threshold Potential

Excitation of cardiac muscle occurs when the stimulus reduces the transmembrane potential to a certain critical level: the threshold potential. This is approximately –60 mV in atrial and ventricular muscle cells. Thus, excitation will result from a relatively weak stimulus if the MRP is lowered and therefore closer to the level of threshold potential providing other factors such as membrane resistance are constant. Conversely, excitation will require a stronger stimulus if the MRP is increased and therefore farther removed from the level of threshold potential.

Refractoriness of Heart Muscle

That period of time in the action potential curve during which no stimulus will propagate another action potential is known as the absolute refractory period. This period includes phases 0, 1, 2 and part of phase 3. Following this, there is a period during which a strong stimulus can evoke a response. This is relative (or effective) refractory period. It begins when the transmembrane potential in phase 3 reaches the threshold potential level (about –60 mV) and ends just before the termination of phase 3. This is followed by a period of supernormal excitability (terminal parts of phase 3 and beginning of phase 4), during which time a relatively weak stimulus can evoke a response.

Cell-to-Cell Conduction

It had been assumed that cell-to-cell conduction and impulse transmission occurred through ‘intracellular bridges’ between muscle cells. However, with electron microscopy it was shown that cells are bounded on all sides by membranes, and no ‘bridges’ are present. The membrane of the cell has a high resistance that should make a electrical conduction impossible; for this reason, chemical transmission was postulated. However, it is now known that, although the cells have a true membrane along their longitudinal axes, the intercalated disks that cross the short axes of the cells are low resistance membranes. These disks have a resistance of

1/1000 of cell membrane and thereby readily permit electrical transmission from cell to cell.

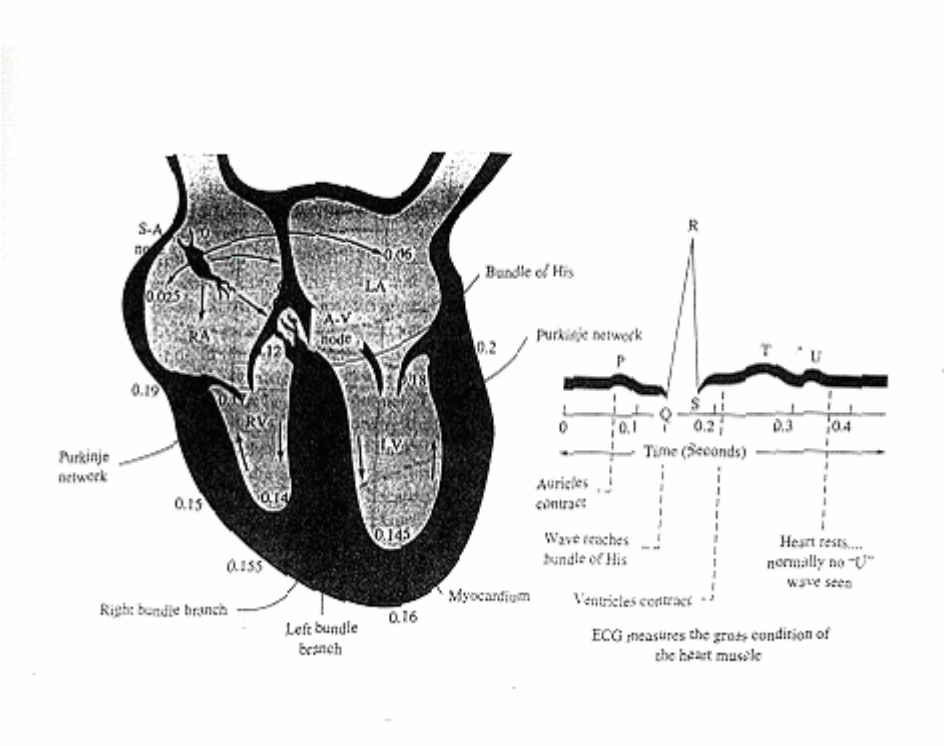

Figure 2.4 Electroconduction system of the heart and resulting ECG waveform

2.2 Introduction to Electrocardiography

The electrocardiogram (ECG) is a graphic recording of the electric potentials produced in an association with the heartbeat. The heart is unique among the muscles of the body in that it possesses the property of autonomic rhythmic contraction. The impulses that precede contraction arise in the conduction system of the heart. The impulses result in excitation of the muscle fibers throughout the myocardium. Impulse formation and conduction produce weak electrical currents that spread through the entire body. By applying electrodes to various positions on the body and connecting these electrodes to an electrocardiographic apparatus, the ECG is recorded. The connections of the apparatus are such that an upright deflection indicates positive potential and a downward deflection indicates negative potential.

2.2.1 The Usefulness of the Electrocardiogram

With advances in electrocardiography, the accuracy of electrocardiographic diagnosis has been greatly increased. The ECG is of particular value in the following clinical conditions:

- Atrial and ventricular hypertrophy
- Myocardial ischemia and infarction: Multiple leads, vectorcardiograms, and modern exercise testing have increased the accuracy of diagnosis and of estimates of extent of disease.
- Arrhythmias: Not only can more exact diagnosis be made, but unipolar and intracardiac electrocardiography have also contributed substantially to our basic understanding of the origin and conduction of abnormal rhythms.
- Pericarditis
- Systematic diseases that affect the heart
- Effect of cardiac (non cardiac) drugs, especially digitalis and quidine
- Disturbances in electrolyte metabolism, especially potassium abnormalities
- Evaluation of electronic pacemaker function

2.2.2 Electrocardiographic Apparatus

In modern electrocardiography, 2 types of apparatus are used: the string galvanometer and the radio amplifier. The former records its pattern on photographic paper which must then be developed. It requires more experience to operate, and caution must be taken to prevent damage to valuable string. The radio amplifier has been combined with a direct writer; it is a compact, light, and mobile unit which is very simple to operate, and there is much less chance of damaging the machine by technical errors of operation. It has the additional advantage of producing an instantaneous recording, thus making the record immediately available for interpretation. Many modern machines record multiple (3 or 6) leads simultaneously. Oscilloscopic viewing of the ECG is commonly used in clinical medicine. This produces a constant electrocardiographic pattern on a fluorescent screen, and

permanent records can be obtained by connecting the machine to a direct-writing apparatus. Such pieces of equipment are now routine in coronary and intensive care units and in surgery.

Small electrocardiographic tape recorders can be attached to a patient and continuous recordings obtained while patient is ambulatory (or at rest) for 24-hour periods. The tape is then reviewed by the physician. This is of special value in the study of patients with arrhythmias and myocardial ischemia.

ECGs can be transmitted via telemetry or telephone lines, thus permitting constant or temporary monitoring and interpretation by a physician many files from the patient. Memory loops are available to record events prior to the onset of an arrhythmia. Computer facilities are available not only for electrocardiographic interpretation but for the recognition and quantization of arrhythmias.

2.2.3 ECG Tracings

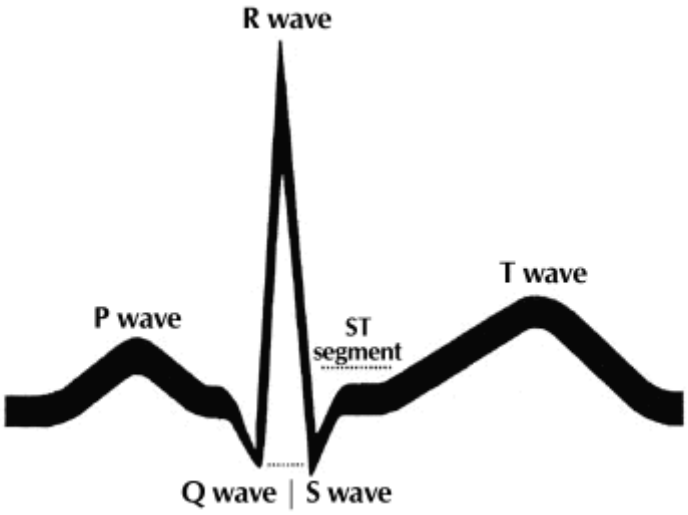

Figure 2.5 ECG Tracings

The first little upward notch of the ECG tracing is called the "P wave." The P wave indicates that the atria (the two upper chambers of the heart) are contracting to pump out blood.

The next part of the tracing is a short downward section connected to a tall upward section. This next part is called the "QRS complex." This part indicates that the ventricles (the two lower chambers of the heart) are contracting to pump out blood to the body.

The next short upward segment is called the "ST segment." The ST segment indicates the amount of time from the end of the contraction of the ventricles to the beginning of the rest period before the ventricles begin to contract for the next beat. The next upward curve is called the "T wave." The T wave indicates the resting period of the ventricles Başçı, E., (2002).

CHAPTER THREE
ARRHYTHMIAS

3. Arrhythmias

An arrhythmia (also called dysrhythmia) is an abnormal rhythm of the heart, which can cause the heart to pump less effectively. Specialized electrical system cells form a small fraction of cardiac mass. At the junction of the superior vena cava and the high right atrium, a cluster of cells, the sinoatrial or sinus node, forms the primary electrical generator (pacemaker) of the normal heart. These cells produce a rhythmic discharge modulated by autonomic innervations and by circulating catecholamine. Sinoatrial node activity is not seen on the surface ECG but occurs 80 to 120 msec before the onset of the P wave, which represents depolarization of atrial myocardial cells. Impulse transmission from the sinoatrial node through the atrium to the atrioventricular node appears to be through normal unspecialized myocardial cells. However, a preferential route of conduction is dictated by the muscle bundles that form the atrium.

The atria are electrically insulated from the ventricles except by the atrioventricular node, whose tortuous conduction pathway delays impulse transmission. The atrioventricular nodal refractory period usually is longer than that of other heart tissue, is heart rate dependent, and is modulated by autonomic tone and by catecholamine, adjusting activation of the ventricles relative to the atria to maximize cardiac output for any given heart rate.

The atrioventricular node is on the atrial side of the annulus fibrosus. Specialized conduction tissue, the His bundle, runs along the tricuspid valve ring to the valve trigone, penetrating the annulus fibrosus and continuing through the membranous interventricular septum. Where the membranous septum becomes muscular septum, the His bundle divides. The right bundle branch continues down the right ventricular endocardial surface to reach the anterior and apical muscle of the right ventricle. Impulses are contained within the branch until its final ramifications. The main left

bundle branch crosses the summit of the muscular interventricular septum to emerge on the left side of the heart just below the noncoronary cusp of the aortic valve. The left bundle divides in a variable manner but functionally gives rise to a left posterior fascicle (which innervates the septum) and a left anterior fascicle. Disease affecting these fascicles may produce characteristic ECG changes.

Arrhythmias can cause problems with contractions of the heart chambers by:

- Not allowing the chambers to fill with an adequate amount of blood, because an electrical signal is causing the heart to pump too fast.
- Not allowing a sufficient amount of blood to be pumped out to the body, because an electrical signal is causing the heart to pump too slowly or too irregularly.

In any of these situations, the heart may not be able to pump an adequate amount of blood to the body with each beat due to the arrhythmia's effects on the heart rate. The effects on the body are often the same, whether the heartbeat is too fast, too slow, or too irregular.

3.1 Types of Arrhythmias

3.1.1 Bradycardia

The adult heart (at rest) beats at about 60 to 80 beats per minute. Fifty-five to 60 beats per minute would be considered bradycardia for an adult. Infants, however, have a much higher at rest heart rate (110 to l30 beats per minute), thus; bradycardia in infants would be a rate below 100 beats per minute.

Slower than average heart rates are normal in people who are physically fit and people who are sleeping. Many athletes who train regularly have resting heart rates of 40 to 60 beats per minute.

Bradycardia can also occur secondary to certain illnesses (such as decreased thyroid function, certain gastrointestinal disorders, and jaundice), or the abuse of certain drugs. People with known heart disease (including hypertension) who are being treated with medications that slow the heart (such as beta-blockers and certain calcium channel blockers) can experience bradycardia. It may be a temporary consequence of certain types of heart attack. Bradycardia is common in elderly people (whether or not they suffer from arteriosclerosis) and infants with certain types of congenital heart disease.

Symptoms of Bradycardia

When symptoms occur, they are usually fatigue, shortness of breath, light-headedness or fainting. Athletes and those with "trained" hearts generally have no symptoms.

Diagnosis of Bradycardia

Determination of bradycardia can be made by the nurse or physician in the office. Further testing to determine the cause may involve blood tests, an EKG or a heart monitor.

Treatment of Bradycardia

If the bradycardia does not cause symptoms, no treatment is necessary. If there are symptoms, medications can be given to increase the rate of the heartbeat. If fainting or serious symptoms persist despite medication, a permanent pacemaker may need to be implanted. In specific instances, certain medications may have to be withdrawn because of their slowing effect.

Severe bradycardia (fewer than 30 beats per minute) can be an emergency situation, leading to brain oxygen deprivation and convulsions. Death may result

unless immediate medical measures are taken to increase the heart rate, from http://www.stronghealth.com/services/cardiology/conditions/bradycardia

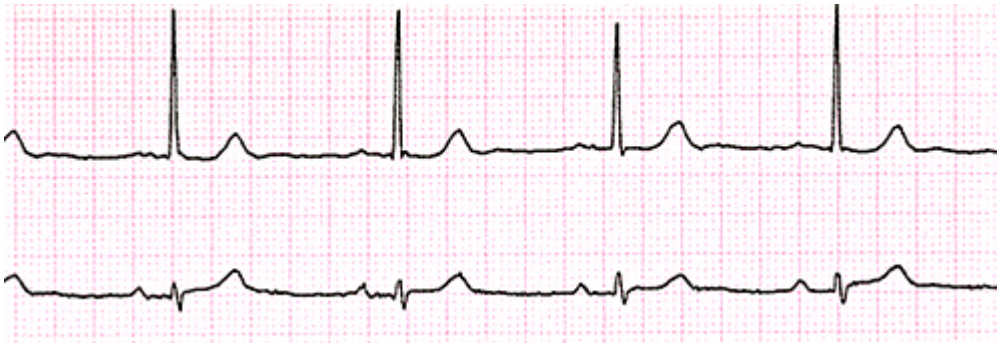

Figure 3.1 The electrocardiographic image of bradycardia

3.1.2 Tachycardia

The heart normally beats at a rate of about 60 to 100 beats per minute at rest. A rate faster than 100 beats a minute in an adult is called tachycardia. Most people experience transient rapid heartbeats, called sinus tachycardia, as a normal response to excitement, anxiety, stress, or exercise. If tachycardia occurs at rest or without a logical cause, however, it is considered abnormal.

Description of Tachycardia

The two main types of tachycardia are abnormal supraventricular tachycardias (which originate in the upper chambers of the heart, the atria) and ventricular tachycardias (which originate in the lower chambers of the heart, the ventricles).

The most common forms of tachycardias are:

1. Paroxysmal supraventricular tachycardia, which generally has a rate of 140 to 200 beats per minute, develops spontaneously, and stops and starts suddenly, but may recur

2. Atrial flutter, in which the atria beat at 240 to 300 beats per minute, although the actual pulse rate is much slower, because not all of these impulses are translated into contractions of the ventricles

3. Ventricular tachycardia, a very serious arrhythmia initiated in the ventricles, in which the heart rate is usually between 150 and 250

4. Atrial fibrillation

Causes and Risk Factors of Tachycardia

Sinus tachycardias are most likely to occur in those who are easily excitable, suffer anxiety, or drink a lot of caffeine-containing beverages. They may also been seen in people with thyroid disease, with fevers, or with certain drugs (especially asthma and allergy medications).

The occurrence of tachycardias under any of these circumstances does not necessarily imply underlying heart disease.

More severe types of tachycardia tend to occur in those who have underlying heart disease. They may be caused by an electrical disturbance within the heart without an anatomic deformity, or by congenital defects, coronary artery disease, chronic disease of the heart valves, or chronic lung disease.

Tachycardias may also occur in the course of a heart attack (or myocardial infarction).

Symptoms of Tachycardia

The main symptom is awareness of a rapid heartbeat, commonly called "palpitations." Depending on the cause and extent of the tachycardia, other symptoms may include shortness of breath, dizziness, actual syncope (fainting), chest pain, and severe anxiety.

Diagnosis of Tachycardia

Your physician will take a complete medical history and perform a physical examination. Blood tests may be done. He or she may perform an electrocardiogram (EKG) or use a heart monitor to assess your heart's electrical activity.

Treatment of Tachycardia

Medical treatment depends on the cause and type of the tachycardia. Sinus tachycardias usually do not require treatment other than therapy for the underlying cause, if any. A supraventricular paroxysmal tachycardia may respond to certain simple maneuvers that your physician will explain to you. This may involve holding one's breath for a minute, bathing the face in cold water, or massaging the carotid artery in the neck. In other cases, medication may be prescribed to slow the heartbeat on a continual basis.

If tachycardia is severe, or arises from the ventricle, immediate injectable medication or electric shock (electroconversion) may be required to stimulate the heart to return to a normal rate. In rare, severe and resistant cases of ventricular tachycardias, a defibrillation device (AICD, similar to a pacemaker) may be implanted surgically to help maintain a normal heart rhythm.

In elderly people or those with underlying heart disease, it is important to treat tachycardias within a few hours, if at all possible, because a prolonged rapid rate may result in decreased heart function and complications, from http://www.americanhealth.org/presenter.html

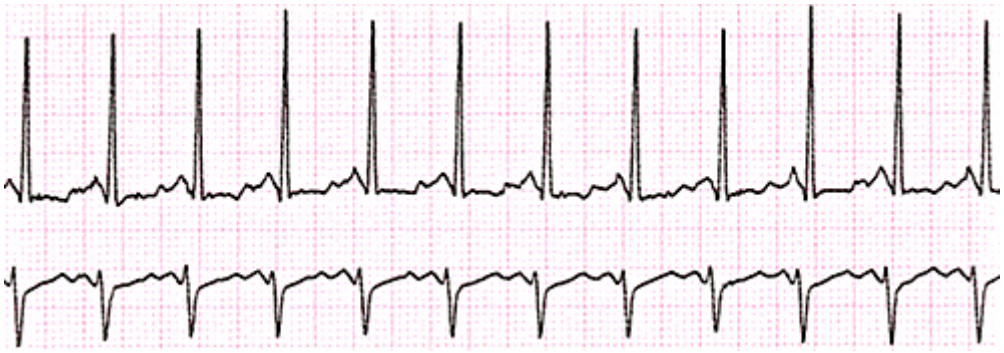

Figure 3.2 The electrocardiographic image of tachycardia

3.1.3 Premature Ventricular Contractions

Heart rate is variable. P wave is usually obscured by the QRS, PST or T wave of the PVC. The wideness of the QRS complex is more than 0.12 seconds and its morphology is bizarre with the ST segment and the T wave opposite in polarity. QRS complex may be multifocal and exhibit different morphologies.

The impulse originates below the portion of the Bundle of His; full compensatory pause is characteristic. Its rhythm is irregular. PVC's may occur in singles, couplets or triplets; or in bigeminy, trigeminy or quadrigeminy.

PVC's can occur in healthy hearts. For example, an increase in circulating catecholamines can pause PVC's. They also occur in diseased hearts and from drug (such as digitalis) toxicities.

Treatment is required if they are;

- associated with an accute MI,
- occur as couplets, bigeminy or trigeminy,
- are mutifocal, or
- are frequent (>6/min)

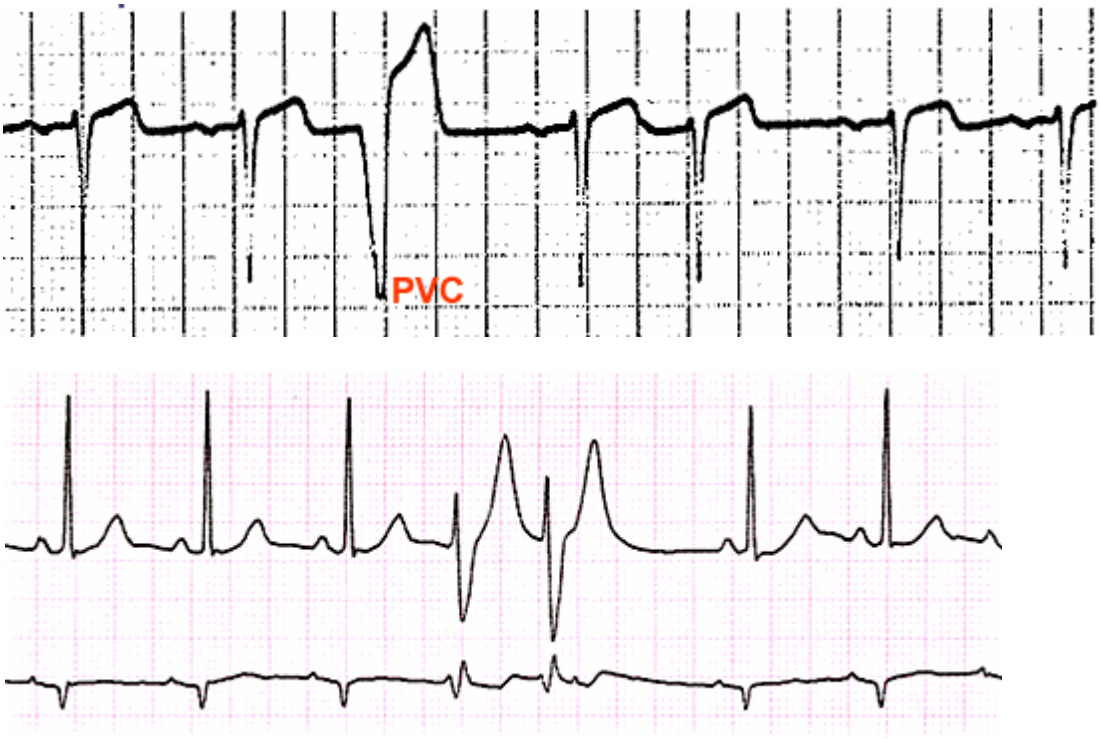

Figure 3.3-a) The electrocardiographic image of PVC
b) The electrocardiographic image of PVC couplet

3.1.4 Ventricular Fibrillation

Ventricular fibrillation occurs when parts of the ventricles depolarize repeatedly in an erratic, uncoordinated manner. The EKG in ventricular fibrillation shows random, apparently unrelated waves. Usually, there is no recognizable QRS complex.

Ventricular fibrillation is almost invariably fatal because the uncoordinated contractions of ventricular myocardium result in ineffective pumping and little or no blood flow to the body. There is lack of a pulse and pulse pressure and the patients lose unconsciousness rapidly. When the patient has no pulse and respiration the patient is said to be in cardiac arrest. A person in cardiac arrest must receive CPR immediately.

Electrical defibrillation, by passage of current at high voltage, may be successful in restoration of a normal regular rhythm. The electrical current stimulates each

myocardial cell to depolarize simultaneously. Following synchronous repolarization of all ventricular cells, the SA node assumes the role of pacemaker and the ventricular myocardial cells can resume the essentially simultaneous depolarization of normal sinus rhythm.

Ventricular fibrillation is associated with drug toxicity, electrocution, drowning and myocardial infarction.

Diagnosis and Treatment

Ventricular fibrillation is suspected when a person suddenly collapses and does not have a pulse or blood pressure. The diagnosis is confirmed by electrocardiography (ECG).

Ventricular fibrillation must be treated as an extreme emergency. Cardiopulmonary resuscitation (CPR) must be started within a few minutes. It must be followed by defibrillation (an electrical shock delivered to the chest) as soon as the equipment is available. Antiarrhythmic drugs may then be given to help maintain the normal heart rhythm.

When ventricular fibrillation is treated within a few minutes (before lack of oxygen damages the brain or other vital organs), some people recover completely. However, people who are successfully resuscitated from ventricular fibrillation due to coronary artery disease are at high risk of another episode. They should usually be evaluated with cardiac catheterization (coronary angiography) or electrophysiologic testing.

If possible, the disorder causing ventricular fibrillation is treated. Otherwise, a defibrillator is surgically implanted. Alternatively, antiarrhythmic drugs are given to prevent recurrences. People who have severe coronary artery disease and poor heart pumping function are less likely to survive, even when defibrillation is promptly done, from http://www.emedicine.com/EMERG/topic633.htm

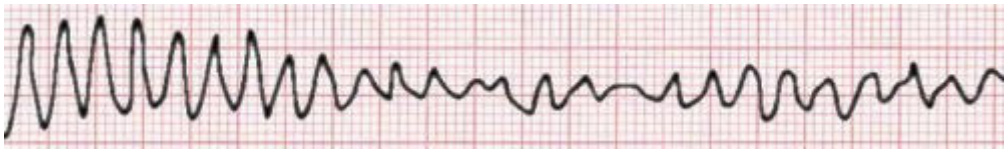

Figure 3.4 The electrocardiographic image of ventricular fibrillation

3.1.5 Bigeminy

Bigeminy is a heart arrhythmia in which heart beats occur in pairs with a pause between each pair. A typical example is ventricular bigeminy which is a normal beat followed by a premature ventricular beat, also known as a premature ventricular contraction (PVC). Following the PVC there is a pause and then the normal beat returns - only to be followed by another PVC. The continuation of this pairing of beats is an example of bigeminy.

Bigeminy can be associated with the following conditions:

- hypoxia
- ischemia
- acute myocardial infarction
- medication overdose

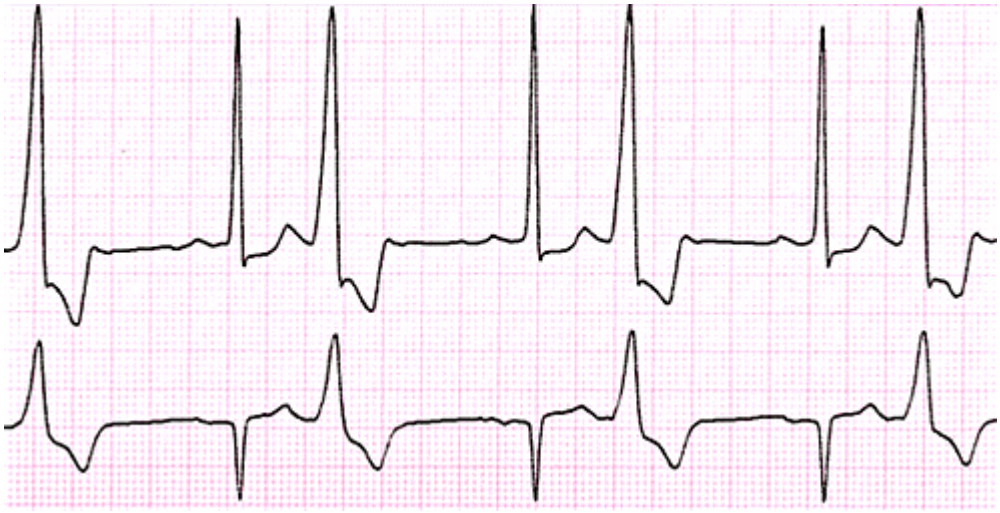

Figure 3.5 The electrocardiographic image of bigeminy

3.1.6 Trigeminy

Trigeminy is a type of PVC pattern. The PVC follows very two normal QRS complexes. Possible causes are electrolyte imbalances, hypoxia, ischemia, acute myocardial infarction, and medication toxicity.

In ECG heart rate is usually normal and 60-100 beats per minute. Rhythm is irregular. A normal QRS complex is followed by a wide QRS complex.

3.1.7 R on T

It is a very dangerous arrhythmia which appears when ventricle suddenly contracts while it is repolarising. This event shows itself as a QRS complex on the T wave. If RR interval is divided into three parts, T wave appears in the first part of this interval. If RR interval appears in a shorter time than the one over three of previous average RR interval time and it is followed by an RR interval which compensates it, R on T arrhythmia is diagnosed.

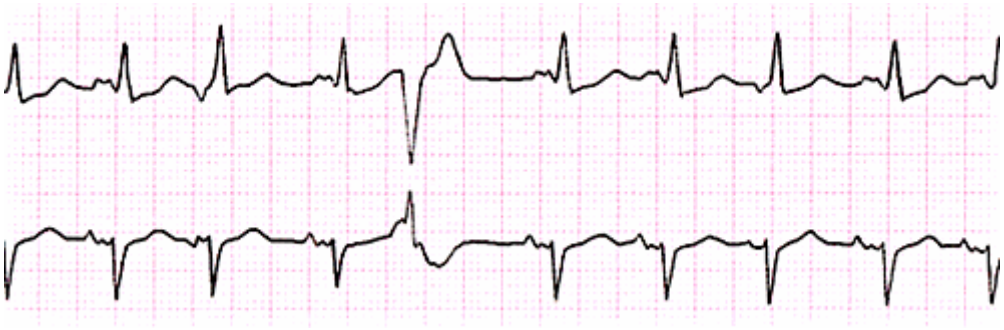

Figure 3.6 The electrocardiographic image of R on T

3.1.8 Skipped Beat

If RR interval is two times bigger than the previous RR interval, it means that one of the R wave is lost and it's named as skipped beat. However, there should not be extra beat that remove the difference, from http://www.londoncardiac.ca/pages/palp.html

3.1.9 Sinus Arrhythmia

This is characterized by variations in heart rate from beat to beat that are greater than would be expected from normal respiratory variation. It is irregular and caused by fluctuations of autonomic tone that result in phasic changes of the discharge rate. During inspiration the parasympathetic tone falls and the heart rate quickens, on expiration the heart rate falls. Such conditions may be detected as intermittent pauses or complexes of premature beats and are normal in the young and in those who are athletic.

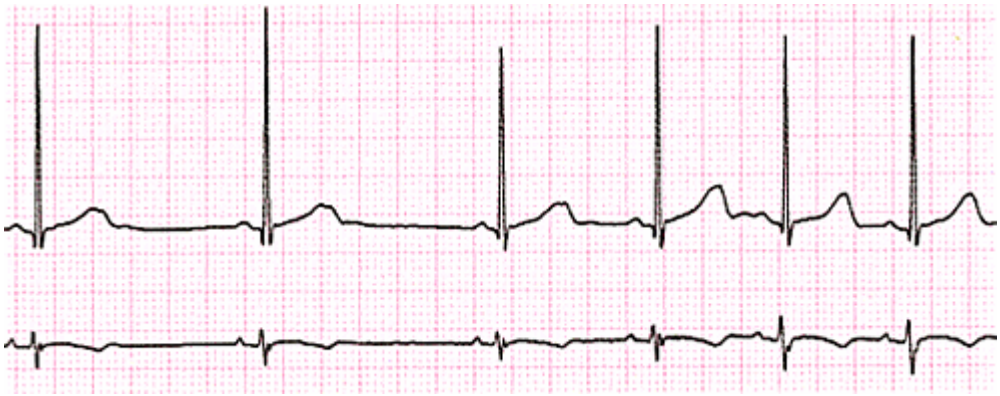

Figure 3.7 The electrocardiographic image of sinus arrhythmia

3.1.10 Atrial Fibrillation

Atrial fibrillation (AF) is an electrical rhythm disturbance of the heart affecting the atria. Abnormal electrical impulses in the atria cause the muscle to contract erratically and pump blood inefficiently. The atrial chambers are thus not able to completely empty blood into the ventricles.

Pooling of blood in the atria can cause red blood cells to stick together and form a clot. Most clots within the heart form in a portion of the left atrium known as the left atrial appendage. The most worrisome complication of atrial fibrillation is dislodgement of a clot and embolism of the clot material to one of the major organs of the body (e.g., the brain).

A clot that embolizes to the brain can interrupt blood flow to a portion of the brain, resulting in a cerebrovascular accident, more commonly known as a stroke. Most individuals with atrial fibrillation are advised by their doctors to take one of a number of medications to prevent clot formation within the heart (and therefore reduce the risk of stroke secondary to clot embolism from the heart). The most commonly used medications are warfarin and aspirin. Atrial fibrillation is the most common form of irregular heartbeat (arrhythmia). Normally, your heart's electrical system controls the rhythm at which your heart beats. See illustrations of the heart and its electrical system.

During a normal heartbeat, the electrical impulses that cause the atria to contract come from the sinus node, a small area of the right atrium. During atrial fibrillation, however, these impulses come from all over the atria, triggering 300 to 500 contractions per minute within the heart's upper chambers. Under normal circumstances, the atrioventricular node would receive these impulses and conduct them to the ventricles (the lower two chambers of the heart that do the pumping). During atrial fibrillation, however, the atrioventricular node becomes overwhelmed by all of the impulses it receives from the atria, and the result is an irregular and rapid heartbeat — 80 to 160 beats per minute versus a normal range of 60 to 100 beats per minute.

The rapid and irregular heartbeat produced by atrial fibrillation cannot pump blood out of the heart efficiently. As a result, blood tends to pool in the heart chambers, increasing the risk of a blood clot forming inside the heart. Blood clots can travel from the heart into the bloodstream and circulate through the body. Ultimately, they may become lodged in an artery, causing pulmonary embolism, stroke and other disorders. In atrial fibrillation, abnormal electrical impulses cause the upper chambers of the heart (atria) to fibrillate, or quiver, resulting in irregular and rapid beating of the ventricles, the heart's main pump. As a result, the heart pumps less efficiently, reducing blood flow to the body and to the heart muscle itself. For most people, this aspect of atrial fibrillation in itself is usually not life-threatening.

However, people with atrial fibrillation are at increased risk for life-threatening strokes, especially if they are not taking anticoagulant medications. Inefficient pumping of the atria allows blood to pool and clot there. If these clots are pumped out of the heart and into the bloodstream, they can lodge in the brain's blood vessels, resulting in stroke. In addition, if the heart rate is fast and uncontrolled over a long period, atrial fibrillation can damage the heart and lead to heart attack and heart failure, from http://.heart-disease.health-cares.net/cardiac-arrhythymia.php

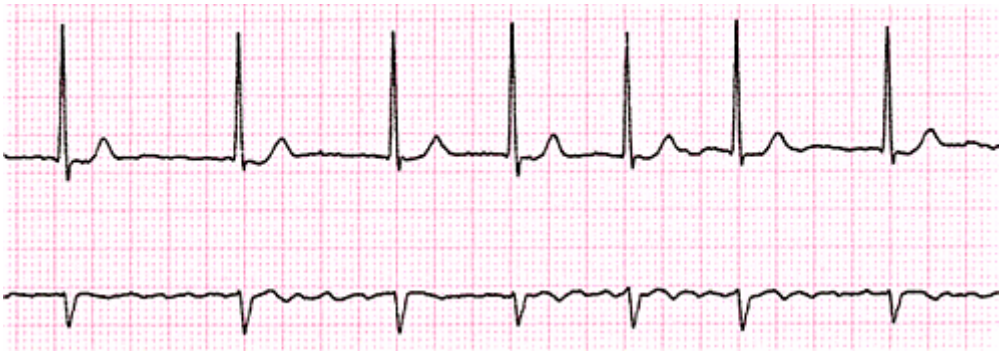

Figure 3.8 The electrocardiographic image of atrial fibrillation

3.1.11 Atrial Flutter

Atrial flutter is a rhythmic, fast rhythm that occurs in the atria of the heart. This rhythm occurs most often in individuals with organic heart disease (i.e.: pericarditis, coronary artery disease, and cardiomyopathy). Atrial flutter is a regular, rhythmic tachycardia originating in the atria. The rate in the atria is over 220 beats/minute, and typically about 300 beats/minute. The morphology on the surface EKG is typically a sawtooth pattern.

The ventricles do not beat as fast as the atria in atrial flutter. The AV node acts as a safety valve in the event of any fast rhythm of the heart, including atrial fibrillation and atrial flutter. The AV node slows down conduction of the electrical activity, and if it receives the next action potential before it is ready, the impulse will be blocked at the AV node level, and never reach the ventricles. In the case of atrial flutter, there is a very particular block pattern at the AV node level. In atrial flutter, the AV node typically will block every other electrical impulse, or three out of four impulses. If

every other impulse is blocked, known as 2:1 block, while the atrial rate is 300 beats/minute, the ventricular rate will be 150 beats/minute. If three out of four beats are blocked, known as 4:1 block, while the atrial rate is 300 beats/minute, the ventricular rate will be 75 beats/minute.

In many individuals, the degree of block is variable - sometimes every other beat is transmitted, sometimes two beats are dropped before the third is transmitted, etc. This is known as varying block. For reasons that are not well understood, a stable 3:1 block is not commonly seen in individuals with atrial flutter. A single individual can have varying degrees of block at different times. The varying degree of block is due to a multitude of factors, including catecholamine release and the use of any drugs that inhibit conduction through the AV node, such as beta blockers, digitalis, and calcium channel blockers.

Atrial flutter can sometimes degenerate to atrial fibrillation and sometimes atrial fibrillation will organize back into atrial flutter and at times even one atrium may be in an organized rhythm and the other atrium may be in a disorganized rhythm. Atrial flutter may arise in otherwise healthy people without any evidence of heart disease, or may be secondary to scarring or stretching diseases of the atrium, just like AF. Atrial flutter shares some features with AF in that it causes symptoms similar to those of AF, it can also increase the risk of stroke by blood clot formation in the heart which breaks off and migrates to the brain. An important difference with atrial fibrillation, though, is that many patients with atrial flutter can have their arrhythmia treated by catheter ablation with a very good probability of success. At times, when antiarrhythmic drugs are used for patients with atrial fibrillation, atrial fibrillation may be organized by the antiarrhythmic drug into atrial flutter and catheter ablation can then be successfully performed, a treatment sometimes referred to as hybrid therapy. In this circumstance, the antiarrhythmic drug is continued after the ablation has been performed to prevent recurrences of AF, from http://www.emedicinehealth.com/articles/10851.asp

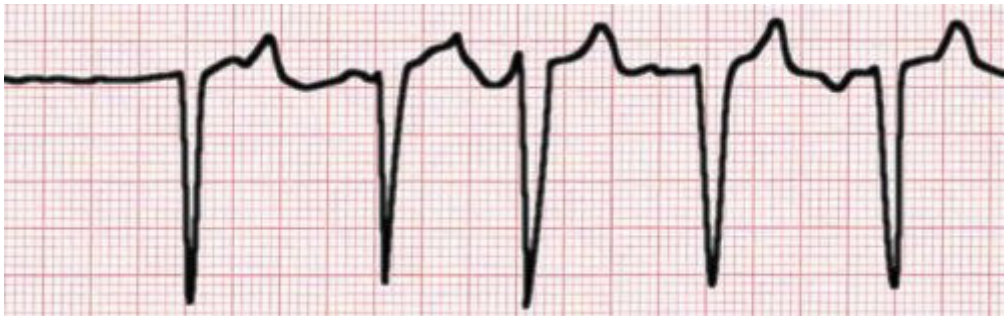

Figure 3.9 The electrocardiographic image of atrial flutter

3.1.12 Bundle Branch Block

Bundle branch block (BBB) is a disruption in the normal flow of electrical pulses that drive the heart beat. Bundle branch block belongs to a group of heart problems called intraventricular conduction defects (IVCD). There are two bundle branches, right and left. The right bundle carries nerve impulses that cause contraction of the right ventricle (the lower chamber of the heart) and the left bundle carries nerve impulses that cause contraction of the left ventricle. The two bundles initially are together at a junction called the bundle of His. Nerve impulses come through the sinus node of the heart to the bundle of His and then move into the right and left bundle branches. Bundle branch block is a slowing or interruption of nerve impulses. A problem may exist in any of the three bundles.

Patients with BBB are generally without symptoms unless the disease is severe enough to cause a complete infranodal A-V block and very slow heart rate. In patients with right bundle branch block (RBBB), the nerve impulse is conducted slowly or not at all. The right ventricle finally receives the impulse through muscle-to-muscle spread, outside the regular nerve pathway. This mechanism of impulse transmission is slow and results in a delayed contraction of the right ventricle. There are several types of left bundle branch block (LBBB), each producing its own characteristic mechanism of failure. In each case, the nerve impulse is blocked or delayed. Patients with LBBB may have left ventricular disease or cardiomyopathy.

Bundle branch block is usually found incidentally on an electrocardiogram (ECG) done for some other reason. It often causes no symptoms. Bundle branch block results from a slowing or complete blockage of one pathway (branch) taken by the electrical impulses that cause your heart to beat. Normally, these electrical impulses originate in the upper-right (atrial) chamber of your heart. They move through the middle of your heart to the lower chambers through right and left electrical conducting branches. These electrical impulses activate the lower chambers, allowing your heart to pump blood to your body and lungs.

If a blockage occurs in one of the branches, the electrical impulse takes a detour to its destination. When this happens, it doesn't affect the rate and rhythm of your heartbeat. But the alternative pathway that the electrical impulse takes shows up on an ECG. Bundle branch block almost never needs treatment. But more severe blockage of the electrical pathways, such as complete heart block, may lead to a very slow heart rate (bradycardia). In this case, a doctor may recommend a pacemaker. It's important to understand that blockage of the electrical system isn't the same as narrowing or blockage of the coronary arteries, which provide blood to the heart.

Usually a person with bundle branch block shows no symptoms, but this block shows up on the EKG as an abnormality. If you have bundle branch block, it may have only been noticed when you had an EKG. You may feel fine, although some people may either faint (syncope) or feel as if they're going to faint (presyncope).

If both bundles are diseased, heart block may result, producing syncope or presyncope. When this happens, the heartbeat may be so slow that an artificial pacemaker is implanted.

Bundle heart block may be caused by damage to the heart muscle resulting from a heart attack. In most cases, bundle branch block does not need treatment. But patients who have bundle branch block along with another heart condition may need treatment. For example, if bundle branch block develops during a heart attack, you may need a pacemaker. After a heart attack, your heart is fragile, and bundle branch

block may cause a very slow heart rhythm (bradycardia). A pacemaker will help regulate the heart's rhythm after a heart attack.

For patients with both bundle branch block and dilated cardiomyopathy, a new type of pacing called cardiac resynchronization treatment (CRT) may be used. Normally, pacemakers pace only one of the lower heart chambers (the ventricles) at a time. But CRT re-coordinates the beating of the two ventricles by pacing them at the same time. Recent studies have shown that CRT works for certain patients with both bundle branch block and dilated cardiomyopathy. Even if you do not have other conditions, you should still see your doctor regularly so that he or she can be sure there are no other changes in your heart, from http://.heart-disease.health-cares.net/cardiac-arrhythymia.php

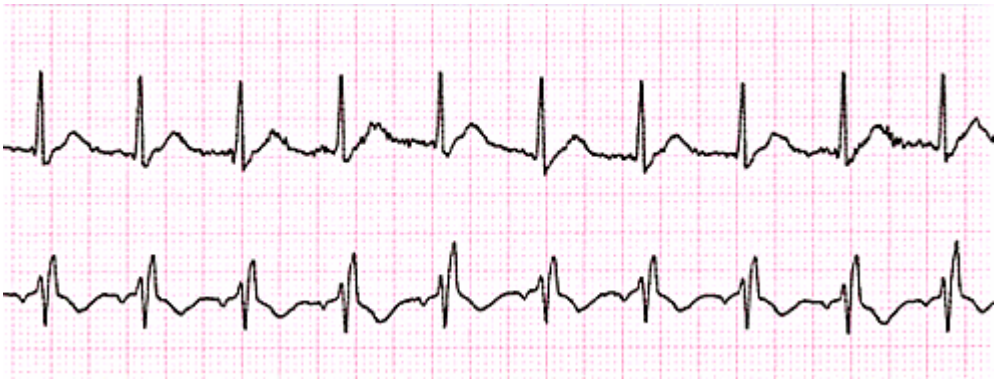

Figure 3.10 The electrocardiographic image of RBBB

3.1.13 Sick Sinus Syndrome

Sick sinus syndrome, also called Bradycardia-tachycardia syndrome is a group of abnormal heartbeats (arrhythmias) presumably caused by a malfunction of the sinus node, the heart's "natural" pacemaker. Sick sinus syndrome is a disorder of the sinus node of the heart, which regulates heartbeat. With sick sinus syndrome, the sinus node fails to signal properly, resulting in changes in the heart rate.

The sinus node in the heart functions as the heart's pacemaker, or beat regulator. In sick sinus syndrome, patients normally will experience bradycardia, or slowed heart rate. Also, it is not uncommon to see fluctuations between slow and rapid heart

rate (tachycardia). This makes the diagnosis and treatment of sick sinus syndrome more complicated than most other cardiac arrhythmias (irregular heart beats). A sick sinus node may be responsible for starting beats too slowly, pausing too long between initiations of heartbeats, or not producing heartbeats at all.

Sick sinus syndrome is a type of bradycardia in which the sinoatrial node (the heart's natural pacemaker) is not functioning as it should. This means that the electrical signal that starts a heartbeat either moves too slowly through the SA node (sinoatrial block) or that there are pauses in delivery of the electrical signal (sinus arrest). SSS can also cause tachycardia (heart rates that are too fast) or bradycardia-tachycardia syndrome (heart rates that fluctuate between being too slow and too fast), from http://.heart-disease.health-cares.net/cardiac-arrhythymia.php

3.1.14 Short QT Syndrome

Short QT syndrome is a genetic disease of the electrical system of the heart. It is made up of a constellation of signs and symptoms, made up of a short QT interval on EKG (≤ 300 ms) that doesn't significantly change with heart rate, tall and peaked T waves, and a structurally normal heart. Short QT syndrome appears to be inherited in an autosomal dominant pattern, and a few affected families have been identified.

Individuals with short QT syndrome frequently complain of palpitations and may have syncope (loss of consciousness) that is unexplained. Due to the autosomal dominant inheritance pattern, most individuals will have family members with a history of unexplained or sudden death at a young age (even in infancy), palpitations, or atrial fibrillation. Short QT syndrome is associated with an increased risk of sudden cardiac death, most likely due to ventricular fibrillation. The diagnosis of short QT syndrome is made up of characteristic history and findings on EKG and electrophysiologic testing. There are currently no set guidelines for the diagnosis of short QT syndrome. The characteristic findings of short QT syndrome on EKG are a short QT interval, typically ≤ 300 ms, that doesn't significantly change with the heart

rate. Tall, peaked T waves may also be noted. Individuals may also have an underlying atrial rhythm of atrial fibrillation.

In the electrophysiology lab, individuals with short QT syndrome are noted to have short refractory periods, both in the atria as well as in the ventricles. Also, ventricular fibrillation is frequently induced on programmed stimulation. The etiology of short QT syndrome is unclear at this time. A current hypothesis is that short QT syndrome is due to increased activity of outward potassium currents in phase 2 and 3 of the cardiac action potential. This would cause a shortening of the plateau phase of the action potential (phase 2), causing a shortening of the overall action potential, leading to an overall shortening of refractory periods and the QT interval. In the families afflicted by short QT syndrome, two different missense mutations have been described in the human ether-a-go-go gene (HERG). These mutations result in expression of the same amino acid change in the cardiac IKr ion channel. This mutated IKr has increased activity compared to the normal ion channel, and would theoretically explain the above hypothesis.

Currently, the only effective treatment option for individuals with short QT syndrome is implantation of an implantable cardioverter-defibrillator (ICD). A recent study has suggested that the use of certain antiarrhythmic agents, particularly quinidine, may be of benefit in individuals with short QT syndrome due to their effects on prolonging the action potential and by their action on the IK channels1. While the use of these agents alone is not indicated at present, there may be benefit of adding these agents to individuals who have already had ICD implantation to reduce the number of arrhythmic events, from http://.heart-disease.health-cares.net/cardiac-arrhythymia.php

3.1.15 Wollf-Parkinson-White Syndrome

Wolff-Parkinson-White syndrome (WPW) is a syndrome of pre-excitation of the ventricles due to an accessory pathway known as the bundle of Kent. This accessory pathway is an abnormal electrical communication from the atria to the ventricles.

Unlike the AV node, which is the normal electrical pathway from the atria to the ventricles, the accessory pathway in WPW does not have slow conductive properties. This means that electrical activity that originates from the SA node with conduct to the ventricles faster via the accessory pathway than via the AV node. This causes the characteristic EKG pattern consisting of a delta wave at the beginning of the QRS complex. The delta wave is due to pre-excitation of the ventricle due to anterograde conduction via the accessory pathway. The EKG will exhibit a short PR interval and a widened QRS interval.

Patients with WPW often exhibit more than one accessory pathway, and in some patients as many as eight additional abnormal pathways can be found. Though it can be treated with medication, in the long term the treatment of choice is destruction of the abnormal electrical pathway by radiofrequency catheter ablation. Wolff-Parkinson-White syndrome is sometimes caused by Leber hereditary optic neuropathy (LHON), a form of mitochondrial disease.

Blood is circulated through the heart and body by a muscular pump and valve system involving the atria and ventricles. The right atrium receives oxygen-lacking blood returning to the heart from the body. The blood is passed from the right atrium into the right ventricle, which contracts and sends blood out to the pulmonary artery. The pulmonary artery sends the blood into the lungs, where carbon dioxide is removed, and fresh oxygen is added. The left atrium receives blood with oxygen from the lungs and passes this arterial blood to the left ventricle, where it is emptied into the aorta, the main artery of the heart.

These functions are directed by electrical signals within the heart. In patients afflicted with Wolff-Parkinson-White syndrome, an abnormal pathway exists that causes additional electrical signals to pass between the atria and ventricles, possibly causing rapid heart rate.

Wolff-Parkinson-White syndrome is characterized by attacks of rapid heart rate (tachycardia). The heartbeat is regulated by electrical impulses that travel through the atria (upper chambers of the heart) to a knot of tissue known as the atrioventricular

node, and then to the ventricles (lower chambers of the heart). Usually, electrical impulses pause at the atrioventricular node before prompting the ventricles to contract. In Wolff-Parkinson-White syndrome, an extra pathway conducts the electrical impulses to the ventricles, without the normal delay, or bounces the electrical impulses back to the atria. The heart rate can reach over 200 beats per minute, when the normal resting heart rate is around 70 to 80 or so. Four out of every 100,000 people are thought to have Wolff-Parkinson-White syndrome. The condition can be managed with medications and surgery, from http://.heart-disease.health-cares.net/cardiac-arrhythymia.php

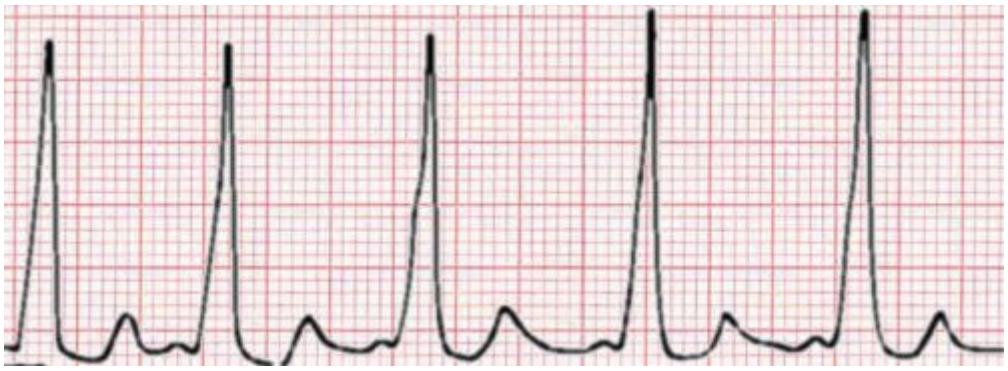

Figure 3.11 The electrocardiographic image of WPW

3.1.16 Ventricular Flutter

This is especially dangerous when the heart rate exceeds 250 beats per minute. This is called ventricular flutter. The chambers of the heart contract so quickly that there is hardly any time for the blood to flow into and fill the chambers. In this situation, the heart transports only a little blood into the circulation. The person who is experiencing this is close to unconsciousness.

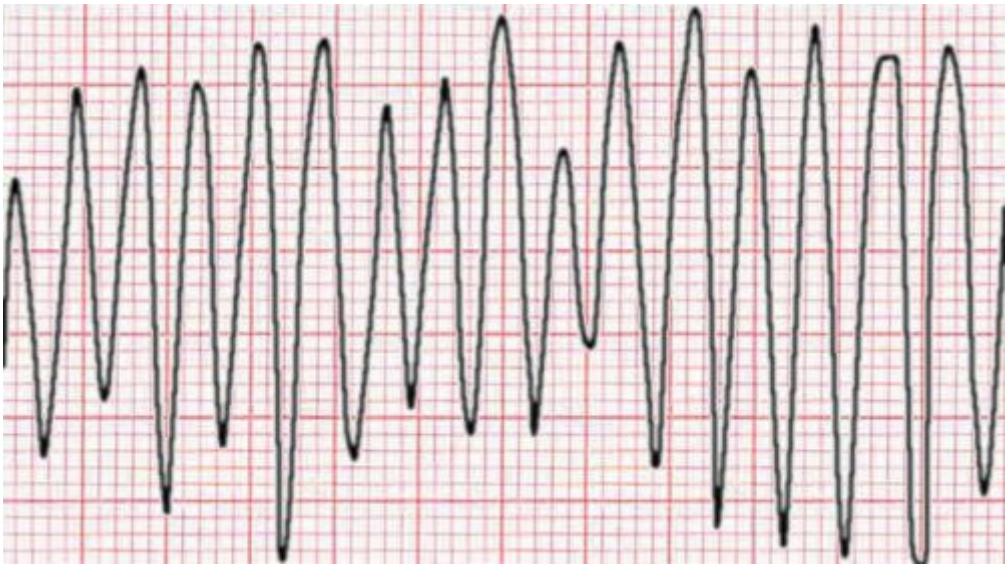

Figure 3.11 The electrocardiographic image of ventricular flutter

CHAPTER FOUR
SELF-ORGANIZING FEATURE MAPS

4. Self- Organizing Maps

Maps of sensory surfaces are discovered in many parts of the brain. These maps seem to indicate that some aspects of information processing for a sensory modelity are related to the physical localizations of the cells on a surface. This is referred to as a topographic representation.

The common occurrence of the topographic mapping in so many different sensory systems is a strong indication that it serves important information-processing function. It is not certain how the biological mapping relations are developed in the nervous system. Current belief is that synaptic connections are from probably not completely determined in a biological neural system by genes to achieve the various mappings. Perhaps multiple mechanisms are involved during development. Learning or “conditioning” may be one of the mechanisms for creating such mappings. Once a map is formed, some systems can show considerable plasticity in the maps in response to various environmental stimuli.

Two types of topographic maps are considered. A topographic map is a topology-preserving (in other words, neighborhood relation-preserving) map from the input space to the output space. The output space is usually a set of units, arranged either in a one-dimensional line or a two-dimensional plane. The basic idea is that inputs that are closed together in the input space according to a metric should be mapped to output units that are close together.

In the first type of topographic map, there are only a small number of continuous input variables, and the metric is defined in the Cartesian coordinate space. The inputs are to be mapped to an output array of units. For example, if the real-valued input variables are (x_1, x_2), the output may be an array of units in a two-dimensional plane. Another example is the case of three real-valued input variables (x_1, x_2, x_3) that

are mapped to two-dimensional array of units, with the three input variables constrained to assume values on a sphere.

In the second type of topographic map the input variables (x_{ij}) are given on an array, which is normally two-dimensional. The map is to transform the activity of the input array to an activity in an output array, which is also normally two-dimensional. For example, the input array may have units turned on in a small neighborhood. This activity is to be transferred to a small neighborhood of units in the output array.

There is also another type of map, the feature map, in which similar features, such as orientation and pattern, are mapped to nearby units. The difference is that the output map is organized according to the similarity of features in the input patterns, not their location in the input space. This normally cannot be achieved by one layer of units.

The common characteristics of the self-organizing network is to asses the input patterns, organize itself to learn, on its own, similarities among the collective set of inputs, and categorize (or cluster) them into groups of similar patterns. Therefore these networks learn without a 'teacher', and the learning is called unsupervised learning.

Unsupervised learning, from large amounts of (redundant) input data, the network learns the underlying latent characteristics of the input patterns, and the synaptic weights of the network, contains this information.

Kohonen Self-Organizing Map (SOM)

The self organizing map developed by Kohonen is an unsupervised, competitive learning, clustering network. In this network only one neuron is on at a time. The SOM is an artificial system that emulates certain mappings that occur in the brain (for example; topographic mappings in the visual system).

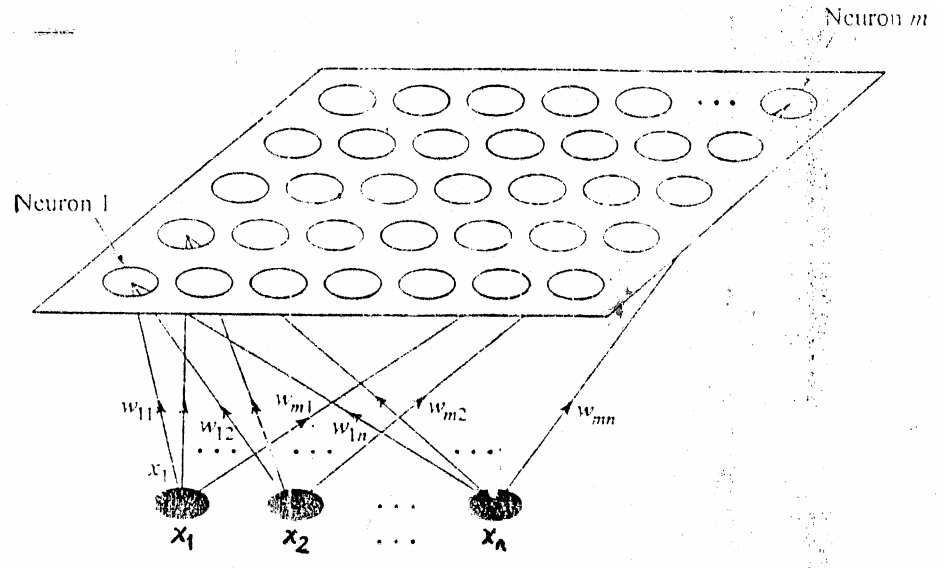

Figure 4.2 Self-Organising map, conventional feature mapping architecture (the inputs are fully connected to each output neuron; however only a few connections are shown)

The SOM transforms input patterns to a one-dimensional or two dimensional map of features in a topological ordered fashion.

Even there are no lateral connections between output neurons, neurons in the neighborhood of the neuron with the best match to the input (i.e., the winning neuron) are modified so that they respond more like the winning unit than they did previously. Lateral connections provide correlated learning by spatially neighboring neurons.

The inputs to the network

$$X = [x_1, x_2, ..., x_n]^T \qquad (4\text{-}1)$$

The synaptic weight vector of neuron *i* in the two dimensional array is given by

$$W_i = [w_{i1}, w_{i2}, ..., w_{in}]^T \qquad i = 1,2,....,m \qquad (4\text{-}2)$$

m: the total number of output neurons in the 2-D array

The best match of the input vector x with the synaptic weight vector w_i is determined from

$$q(x) = \min \left\| X - w_i \right\|_2 \qquad i = 1,2,....,m \tag{4-3}$$

q(x) is the index that specifically identifies the winning neuron with equation (4-3) a continuous input space is mapped onto a discrete array of neurons.

The learning rule for updating the synaptic weight vector associated with the winning neuron and the neurons within a defined neighborhood of the winning neuron is given by

$$W_i(k+1) = W_i(k) + \eta_{qi}(k)[X(k) - W_i(k)] \tag{4-4}$$

Where

$$\eta_{qi}(k) = \begin{cases} \mu(k) & \text{within } Nq \text{ (neighborhood set for winning neuron } q) \text{ where } 0<\mu(k)<1 \\ 0 & \text{outside } Nq \end{cases} \tag{4-5}$$

From (4-4) and (4-5) we can write the learning rule as;

$$W_i(k+1) = \begin{cases} Wi(k) + \mu(k)[X(k) - Wi(k)] & \text{if } i \in Nq(k) \\ Wi(k) & \text{if } i \notin Nq(k) \end{cases} \tag{4-6}$$

Where $0 < \mu(k) < 1 \qquad (\text{the learning rate parameter})$

It is advantageous to let the neighborhood set $N_q(k)$ be relatively wide in the beginning of training and then shrink monotonically with time (this is illustrated in Figure 4.2)

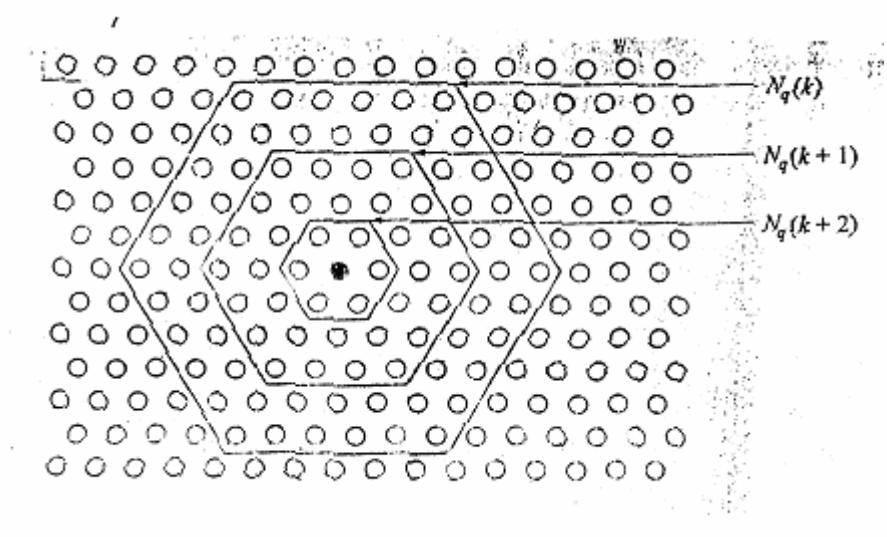

Figure 4. 3 Topological neighborhood examples, showing a monotonic decrease in the neighborhood

A biological lateral interaction can have a bell-shaped curve. To incorporate this into the kernel function, we denote the coordinates of the neurons q and i by the vectors r_q and r_i respectively. The typical choice for η_{qi} in equation (4-4) is

$$\eta_{qi} = \eta_0 \exp\left(-\left\|r_i - r_q\right\|_2^2 / \sigma^2\right) \qquad (4\text{-}7)$$

With $\eta_0 = \eta_0(k)$ and $\sigma = \sigma(k)$ chosen as suitable decreasing functions of time.

The learning rate equation (4-4) drags the weight vector w_q associated with the winning unit towards X, and it also drags w_i's of the closest units along with it.

During the learning process there are two related phases, the ordering phase and the convergence phase.

Ordering Phase

During this phase topological ordering of the weight vectors is carried out. The training process attempts to cluster the nodes on the topological map to reflect to

range of class types found in the training data. This is a coarse mapping, where the network is discovering how many classes the map must eventually identify, and where should lie in relation to each other on the map. During this phase, the learning parameter should be set close to unity and then gradually decreased (but not allowed to go below 0.1).

Convergence Phase

Once a stable coarse representation is found, the nodes within the localized regions of the map are fine-tuned to the input training vectors. To achieve this fine-tuning much smaller changes must be made to the weight vectors at each node. For this the learning rate parameter should attain relatively small values for a long time. The learning rate parameter should be on the order of (or less than) 0.01.

The training algorithm will produce clusters for all the class types found in the training data. Once the network has self-organized the internal representation, the clusters on the feature map can be labeled to indicate their class so that the network can be used to classify unknown inputs. The network forms the internal features without supervision, but the classification labeling must be done by hand, once the network has been fully trained, Bose, N.K., & Liang, P., (1996).

CHAPTER FIVE
APPLICATION AND RESULTS

5. Application and Results

In this thesis, the arrhythmia detection is carried out in two different ways. According to the used data the first method can be named as pattern classification while the second one can be named as beat classification.

First Way

Ideally, the EKG line must be smooth but because of noise the shape is changed. So, a microcomputer can't detect P and T waves which have small amplitudes. However the detection of R wave, which has relatively bigger amplitude, is easier. In this algorithm, the location of R wave in time domain is used in spite of the amplitude.

Here, eleven kinds of arrhythymia is used for detection algorithm. They are normal sinus rhythm, bradycardia, tachycardia, ventricular fibrillation, skipped beat, premature ventricular contractions, R on T, bigeminy, trigeminy, extrasystol, and, atrial premature beat. The procedure is described below.

1. The vectors are constituted from the graph shown below.

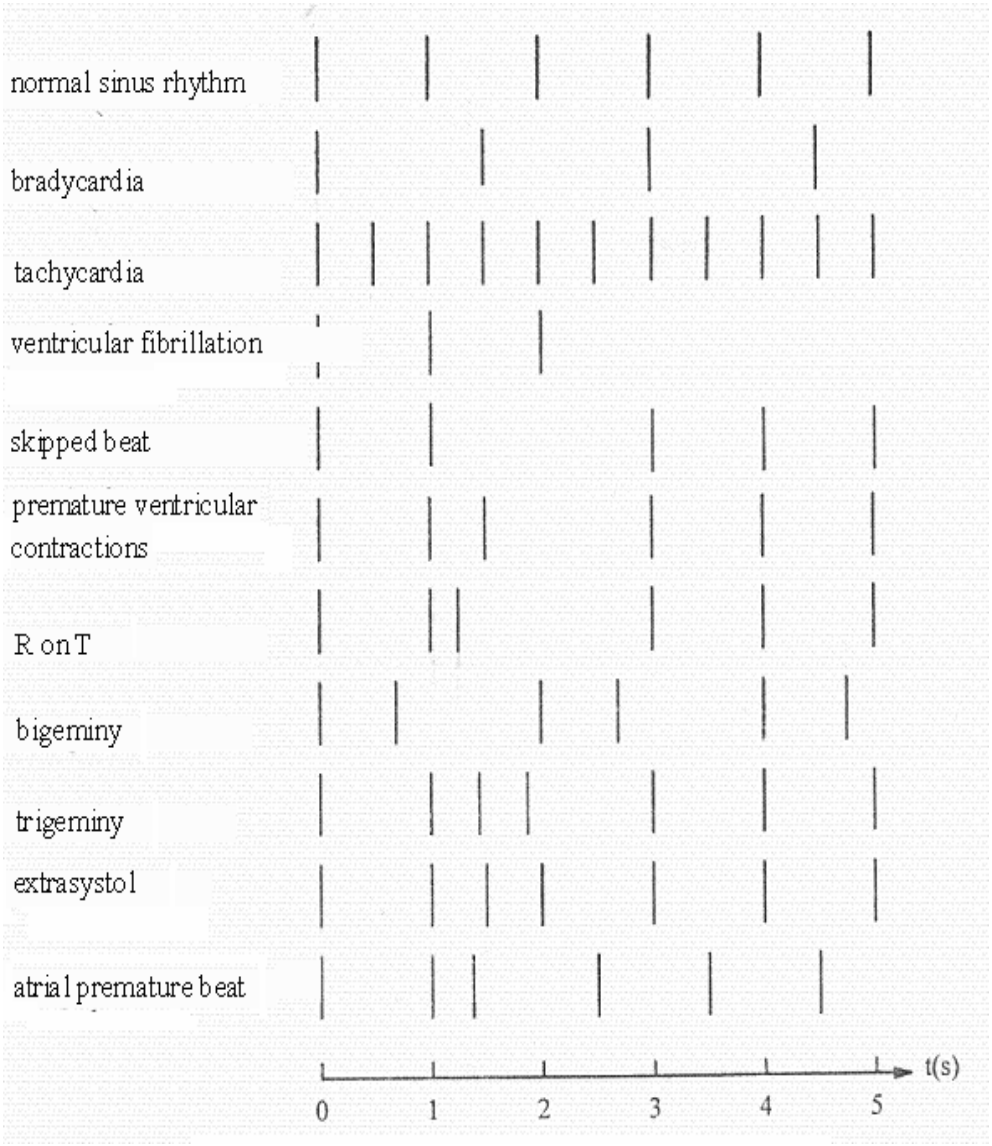

Figure 5.1 R waves in some kinds of arrhythmias

2. 5 sec. long ECG recordings are divided into equal segments to form equal sized vectors.
3. The location where R waves take place are marked as 1 and the others are marked as zero. So, eleven vectors of arrhythmias are constituted. The size of each vector is 51x1 and they consist of ones and zeros.
4. As an algorithm, the demosm2 program from Matlab toolbox is used. A 6 by 6 SOM is used to classify the vectors constructed. It is expected that each

neuron would respond to a different region of the map, and neighboring neurons would respond to similar vectors.

5. Self-organizing map is created by the command ‘newsom’. For topology function, distance function, ordering phase learning rate, ordering phase steps, tuning phase learning rate and tuning phase neighborhood distance, default values are used.

Table 5.1 Default values

	Name	Default Values
Di	Size of ith layer dimension	[5 8]
TFCN	Topology function	Hextop
DFCN	Distance function	Linkdist
OLR	Ordering phase learning rate	0.9
OSTEPS	Ordering phase steps	1000
TLR	Tuning phase learning rate	0.02
TND	Tuning phase neighborhood distance	1

6. After training, each vector is tested to show which neuron is activated.
7. The results show that each arrhythmia vector respond to a different region of the map. The graph of the result of the first part is shown below.

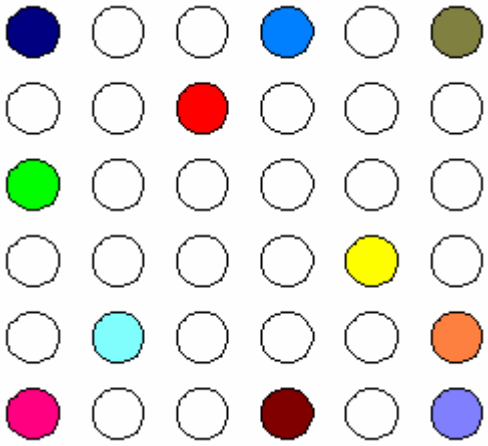

Figure 5.2 Neurons activated by the arrhythmias

: Normal sinus rhythm

: Bradycardia

: Tachycardia

: Ventricular fibrillation

: Skipped beat

: Premature ventricular contraction

: R on T

: Bigeminy

: Trigeminy

: Extrasystol

: Premature atrial contraction

8. To see, if the noise added vectors activate the different regions of the map; 110 noisy arrhythmia vectors are constituted, by adding ones randomly to each vector for ten times. Three or four zeros are changed with ones to add noise to the vectors.

9. Each groups of noisy vectors activate the different region of the map. The graph of the activated neurons of the noise added vectors are shown below.

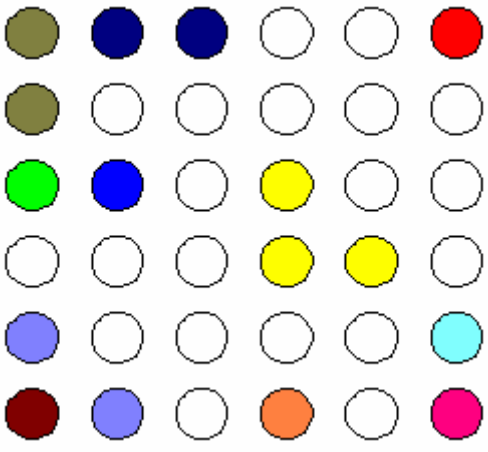

Figure 5.3 Neurons activated by the noise added arrhythmias

: Noise added normal sinus rhythm.

: Noise added bradycardia

: Noise added tachycardia

: Noise added ventricular fibrillation

: Noise added skipped beat

: Noise added premature ventricle contraction

: Noise added R on T

: Noise added bigeminy

: Noise added trigeminy

: Noise added extrasystol

: Noise added premature atrial contraction

Second Way

In the second part, the ECG data which is taken from MIT/BIH Arrhythmia Database, is used in the algorithm. Procedure of the second way is described below.

1. The database is downloaded from the Physionet web page. PhysioNet offers free access via the web to large collections of recorded physiologic signals and related open-source software.
2. The data is taken from MIT/BIH Arrhythymia Database in PhysioBank Archives. The MIT-BIH Arrhythmia Database contains 48 half-hour excerpts of two-channel ambulatory ECG recordings, obtained from 47 subjects studied by the BIH Arrhythmia Laboratory between 1975 and 1979. Twenty-three recordings were chosen at random from a set of 4000 24-hour ambulatory ECG recordings collected from a mixed population of inpatients (about 60%) and outpatients (about 40%) at Boston's Beth Israel Hospital; the remaining 25 recordings were selected from the same set to include less common but clinically significant arrhythmias that would not be well-represented in a small random sample. The recordings were digitized at 360 samples per second per channel with 11-bit resolution over a 10 mV range. Two or more cardiologists independently annotated each record; disagreements were resolved to obtain the computer-readable reference annotations for each beat (approximately 110,000 annotations in all) included with the database.
3. In this algorithm the arrhythmia database belonging to the patients numbered 100, 105, 106, 109, 111, 114, 116, 119, 124, 200, 207, 209, 212 and 214, have been considered.
4. They are preprocessed before using in the algorithm. The central QRS wave is obtained and 30 QRS wave in the left, 30 in the right are taken to constitute new data. Each arrhythmia vector consists of 61 points. The data numbered thirty one is the central and there is 30 data in the left and 30 data in the right. The 3:63 value of each arrhythymia database row is a vector.

5. The size of constituted arrhythymia database matrix is 17211x63. Each row of the matrix is formed as; [‘numberofpatient’ ‘typeofarrhythymia’ ‘data’].
6. There are seven types of arrhythmias as shown below.

Table 5.2 Number of arrhythmias used in the database

MIT/BIH	Arrdb	Name	Number
N	O	Normal Beat	2237
L	1	Left Bundle Branch Block Beat	8070
R	2	Right Bundle Branch Block Beat	3440
A	3	Atrial Premature Beat	564
V	4	Premature Ventricular Contraction	2323
E	5	Ventricular Escape Beat	105
!	6	Ventricular Flatter Wave	472
TOTAL			17211

7. Number of each vector is different, but same number of vectors are used in the algorithm. Randomly 65 vectors are chosen for each kind of arrhythymia database.
8. First train data for each vector and self-organizing map is constituted. Train data is formed by skipping one element in train data vector to reduce the size of the vector. The size of each train data is 31x1.
9. After training the same vectors are used for testing.
10. In the end of testing, some kinds of arrhythmias respond the same region of neurons. To come over this problem number of training epochs and neurons are increased. Algorithm is repeated again but the same problem occurred.
11. In the second step the number of arrhythmias reduced to six and the same procedure is repeated, again some of the arrhythmias respond the same region of neurons.
12. The number of arrhythmias reduced to five, four and three one by one. The algorithm couldn’t succeed to separate the arrhythmias until three kinds of data remain. Especially right bundle branch block beat and atrial premature

beat, ventricular escape beat and ventricular flatter wave, premature ventricular contraction and ventricular flatter wave datas are mixed with each other.

13. The percentage of misclassifications with six kinds of arrhythmias is 56.36%, with five kinds of arrhythmias is 40.4%, with six kinds of arrhythmias is 28%,
14. When only three kinds of arrhythmias are used in the algorithm, arrhythmias respond different region of neurons. Left bundle branch block beat and atrial premature beat, atrial premature beat and ventricular escape beat, left bundle branch block beat and ventricular flatter wave can be separated from each other properly. The graphs of results are shown below.

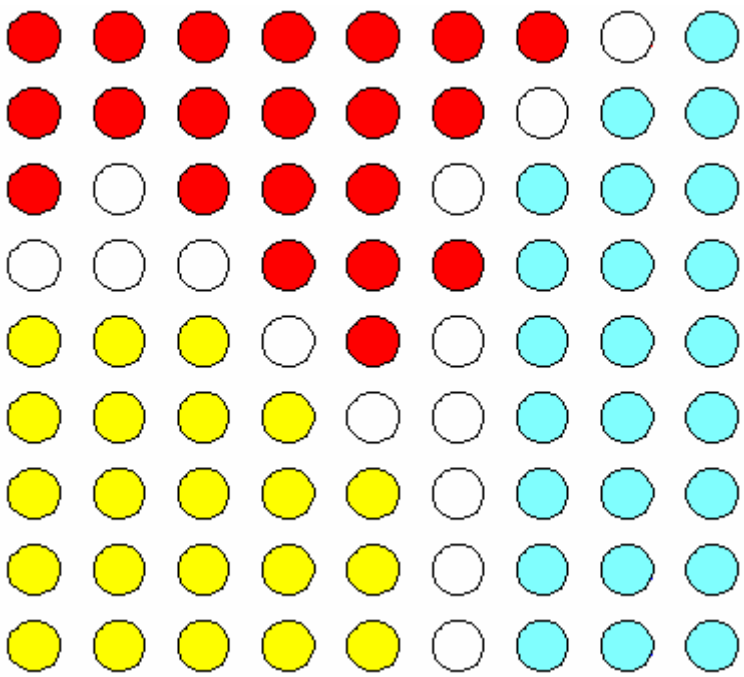

Figure 5.4 Neurons activated by arrhythmias

: Normal beat

: Ventricular escape beat

: Atrial premature beat

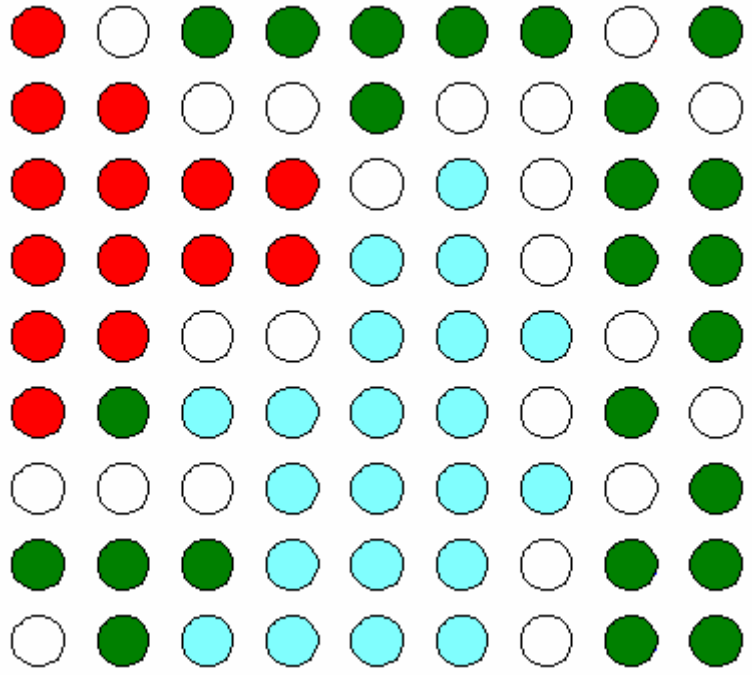

Figure 5.5 Neurons activated by the arrhythmias

: Normal beat

: Left bundle branch block

: Atrial premature beat

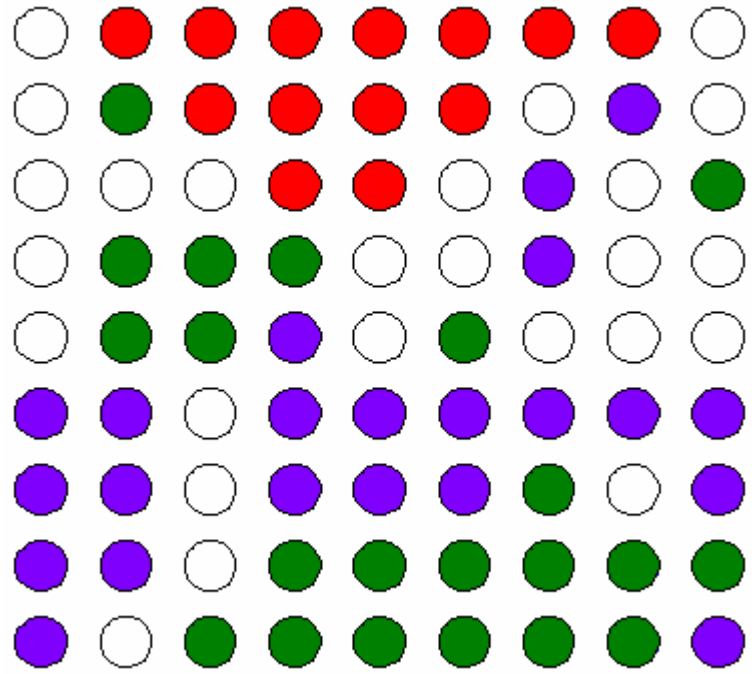

Figure 5.6 Neurons activated by the arrhythmias

: Normal beat

: Left bundle branch block

: Ventricular flatter wave

CHAPTER SIX
DISCUSSION AND CONCLUSION

In this study, the classification of different types of arrhythmias by using Self-Organizing Map is performed. The method is studied in two different ways. According to the data used the first one can be named as pattern classification and the second one can be named as beat classification.

In the beat classification, the algorithm succeeded to classify only three kinds of arrhythmias. If more than three kinds of arrhythmias are used, some of them respond to the same region of cells. This is due to fact that some of the arrhythmias may have the same morphologic characteristics such as the amplitude of the QRS complex, the period of the RR interval and the location of the R wave.

There are similar studies in the literature but they didn't use the same properties used in the presented algorithm. Braccini G., et al., (2001) calculated the RR intervals of the ECG, Prasad G. K., et al., (2003) calculated the DWT coefficients. Unlike them, the recordings of ECG are used without feature extraction in this study. Instead of the whole recording, a definite part of the recording is used in the algorithm.

The recordings in the study are obtained from MIT/BIH Arrhythmia Database. Osowski S., Linh T. H., (2001) used raw data, too. They studied on the application of the fuzzy neural network for electrocardiographic beat recognition and classification. In their study the recognition of the normal and different types of beats representing the arrhythmias have been done with a good accuracy. However in this thesis, SOM algorithm can classify only three types of arrhythmias, it fails when constituted more than three types.

In the continuation of this study, the data can be used by taking their Wavelet or Fourier Transforms in the algorithm. Feature extraction of ECG recording can give better results in the classification of a large number of arrhythmias. Also the outputs

of SOM can be used as the inputs of Learning Vector Quantization (LVQ) method

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APPENDIX

```
%THE PROGRAM WHICH CLASSIFIES THE ARRHTHMIAS ACCORDING TO
THE R WAVES

clear all

in1=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 0 0 0 1]';

in2=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
0 0 1 0 0 0 0 0]';

in3=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0
0 0 1 0 0 0 0 1]';

in4=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0]';

in5=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 0 0 0 1]';

in6=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 0 0 0 1]';

in7=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
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in8=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 1 0 0 0]';

in9=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 0 0 0 1]';

in10=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 1]';

in11=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0 0
0 0 0 1 0 0 0 0]';

in=[in1 in2 in3 in4 in5 in6 in7 in8 in9 in10 in11];

% We will use a 6 by 6 layer of neurons to classify the vectors above. We would
% like each neuron to respond to a different region of the rectangle, and
% neighboring neurons to respond to adjacent regions.  We create a layer of
% 36
% neurons spread out in a 6 by 6 grid:
```

```
net =newsom([0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0
1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0
1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1],[6 6]);

net.trainParam.epochs =1000;
net =train(net,in);

p1=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a1 =sim(net,p1);

p2=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;1;0;0;0;0;0];
a2 =sim(net,p2);

p3=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;
0;0;0;0;1;0;0;0;0;1];
a3 =sim(net,p3);

p4=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;
0;0;0;0;0;0;0;0;0;0];
a4 =sim(net,p4);

p5=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a5 =sim(net,p5);

p6=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a6 =sim(net,p6);

p7=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a7 =sim(net,p7);

p8=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;1;0;0;0];
a8 =sim(net,p8);

p9=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a9 =sim(net,p9);

p10=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1];
a10 =sim(net,p10);
```

```
p11=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0
;0;0;0;0;1;0;0;0;0;0];
a11 =sim(net,p11);
```

```
%THE PROGRAM WHICH CLASSIFIES THE NOISE ADDED ARRHTHMIAS
ACCORDING TO THE R WAVES

clear all

in11=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in12=[1 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in13=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in14=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in15=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in16=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in17=[1 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in18=[1 0 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in19=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in110=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 1]';

in21=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in22=[1 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in23=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in24=[1 0 0 0 0 0 0 0 0 0 1 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in25=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in26=[1 0 0 0 0 0 0 0 1 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in27=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in28=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 0]';
in29=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 1 0 0]';
in210=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 1
0 0 0 0 1 0 0 0 0 0]';
```

```
in31=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in32=[1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in33=[1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in34=[1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in35=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in36=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in37=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in38=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in39=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0
0 0 0 1 0 0 0 0 1]';
in310=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1
0 1 0 0 1 0 0 0 0 1]';

in41=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in42=[1 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in43=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in44=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in45=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in46=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0
0 0 0 0 0 0 0 0 0]';
in47=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0 0]';
in48=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 0]';
in49=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 1 0 0 0 0 0 0]';
in410=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 1 0 0]';

in51=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in52=[1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in53=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
```

```
in54=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in55=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in56=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in57=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 1 0
0 0 0 0 0 0 0 0 1];
in58=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 1 0 0 0 0 0 1];
in59=[1 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in510=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 1 0 0 1];

in61=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in62=[1 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in63=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in64=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in65=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 1 0 0 0 0 1];
in66=[1 0 0 0 0 0 0 0 1 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in67=[1 0 0 0 0 0 0 0 0 0 1 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in68=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in69=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
1 0 0 0 0 0 0 0 1];
in610=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 1 0 1];

in71=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in72=[1 0 0 0 1 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in73=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in74=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in75=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1];
in76=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0
0 1 0 0 0 0 0 0 1];
```

```
in77=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in78=[1 0 0 0 0 0 0 1 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in79=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in710=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 1
0 0 0 0 0 0 0 0 1]';

in81=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in82=[1 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in83=[1 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in84=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in85=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in86=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in87=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in88=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 1 0 0 0 1 0 0 0]';
in89=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 1 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 1 0 0 0]';
in810=[1 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 1 0 0 0 0 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 1 0 0 0]';

in91=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in92=[1 0 0 1 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in93=[1 0 0 0 0 0 0 1 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in94=[1 0 0 0 0 0 0 0 0 0 1 0 1 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in95=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 1 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in96=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in97=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 1 0
0 0 0 0 0 0 0 0 1]';
in98=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0
0 0 1 0 0 0 0 1]';
in99=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 1 0 0 1 0
0 0 0 0 0 0 0 0 1]';
```

```
in910=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 1 0 0 1]';

in101=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in102=[1 0 0 1 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in103=[1 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in104=[1 0 0 0 0 0 0 0 0 0 1 0 0 1 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in105=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 1 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in106=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 1 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in107=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 1
0 0 0 0 0 0 0 0 0 1]';
in108=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 1
0 0 0 0 0 0 0 0 0 1]';
in109=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 1 0 0 0 0 0 1]';
in1010=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1
0 0 0 0 0 0 1 0 0 1]';

in111=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in112=[1 0 1 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in113=[1 0 0 0 0 0 0 1 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in114=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in115=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in116=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 1 0 0 0 0 0 1 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in117=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 1 0
0 0 0 0 1 0 0 0 0 0]';
in118=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 1 0 0]';
in119=[1 0 0 1 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 0 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';
in1110=[1 0 0 0 0 0 0 0 0 0 1 0 0 0 1 0 0 0 0 0 0 0 0 0 0 0 1 0 0 0 0 0 1 0 0 1 0 0 0 0 0
0 0 0 0 1 0 0 0 0 0]';

in1=[in11 in12 in13 in14 in15 in16 in17 in18 in19 in110];
in2=[in21 in22 in23 in24 in25 in26 in27 in28 in29 in210];
in3=[in31 in32 in33 in34 in35 in36 in37 in38 in39 in310];
```

```
in4=[in41 in42 in43 in44 in45 in46 in47 in48 in49 in410];
in5=[in51 in52 in53 in54 in55 in56 in57 in58 in59 in510];
in6=[in61 in62 in63 in64 in65 in66 in67 in68 in69 in610];
in7=[in71 in72 in73 in74 in75 in76 in77 in78 in79 in710];
in8=[in81 in82 in83 in84 in85 in86 in87 in88 in89 in810];
in9=[in91 in92 in93 in94 in95 in96 in97 in98 in99 in910];
in10=[in101 in102 in103 in104 in105 in106 in107 in108 in109 in1010];
in11=[in111 in112 in113 in114 in115 in116 in117 in118 in119 in1110];

in=[in1 in2 in3 in4 in5 in6 in7 in8 in9 in10 in11];

% We will use a 6 by 6 layer of neurons to classify the vectors above. We would
% like each neuron to respond to a different region of the rectangle, and
% neighboring neurons to respond to adjacent regions.  We create a layer of 36
% neurons spread out in a 6 by 6 grid:

net =newsom([0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0
1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0
1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1;0 1],[6 6]);

net.trainParam.epochs =10;
net =train(net,in);

p1=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a1 =sim(net,p1);
p11=[1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1];
a11 =sim(net,p11);
p12=[1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1];
a12 =sim(net,p12);
p13=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1];
a13 =sim(net,p13);
p14=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a14 =sim(net,p14);
p15=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a15 =sim(net,p15);
p16=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a16 =sim(net,p16);
p17=[1;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a17 =sim(net,p17);
```

```
p18=[1;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a18 =sim(net,p18);
p19=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a19 =sim(net,p19);
p110=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a110 =sim(net,p110);

p2=[1;;0;0;0;0;0;0;0;0;0;1;;0;0;0;0;1;;0;0;0;0;0;0;0;0;0;0;0;0;0;1;;0;0;0;0;0;0;0;0;0;
1;;0;0;0;0;1;;0;0;0;0;0];
a2 =sim(net,p2);
p21=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a21 =sim(net,p21);
p22=[1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a22 =sim(net,p22);
p23=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a23 =sim(net,p23);
p24=[1;0;0;0;0;0;0;0;0;0;1;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a24 =sim(net,p24);
p25=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a25 =sim(net,p25);
p26=[1;0;0;0;0;0;0;0;1;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a26 =sim(net,p26);
p27=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a27 =sim(net,p27);
p28=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;0;];
a28 =sim(net,p28);
p29=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;1;0;0;];
a29 =sim(net,p29);
p210=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;
1;0;0;0;0;1;0;0;0;0;0;];
a210 =sim(net,p210);

p3=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;
0;0;0;0;1;0;0;0;0;1];
a3 =sim(net,p3);
```

```
p31=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a31 =sim(net,p31);
p32=[1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a32 =sim(net,p32);
p33=[1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a33 =sim(net,p33);
p34=[1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a34 =sim(net,p34);
p35=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a35 =sim(net,p35);
p36=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a36 =sim(net,p36);
p37=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a37 =sim(net,p37);
p38=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a38 =sim(net,p38);
p39=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;0;0;0;0;1;];
a39 =sim(net,p39);
p310=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1;0;1;0;0;1;0;0;0;0;1;];
a310 =sim(net,p310);


p4=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;];
a4 =sim(net,p4);
p41=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;];
a41 =sim(net,p41);
p42=[1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;];
a42 =sim(net,p42);
p43=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;];
a43 =sim(net,p43);
p44=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;];
a44 =sim(net,p44);
```

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p45=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0
;0;0;0;0;0;0;0;0;0;0;];
a45 =sim(net,p45);
p46=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0
;0;0;0;0;0;0;0;0;0;0;];
a46 =sim(net,p46);
p47=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0
;0;0;0;0;0;0;0;0;0;0;];
a47 =sim(net,p47);
p48=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0
;0;0;0;0;0;0;0;0;0;0;];
a48 =sim(net,p48);
p49=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0
;0;0;0;1;0;0;0;0;0;];
a49 =sim(net,p49);
p410=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;
0;0;0;0;0;0;0;0;1;0;0;];
a410 =sim(net,p410);


p5=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a5 =sim(net,p5);
p51=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a51 =sim(net,p51);
p52=[1;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a52 =sim(net,p52);
p53=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a53 =sim(net,p53);
p54=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a54 =sim(net,p54);
p55=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a55 =sim(net,p55);
p56=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a56 =sim(net,p56);
p57=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;1
;0;0;0;0;0;0;0;0;0;1;];
a57 =sim(net,p57);
p58=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;1;0;0;0;0;0;1;];
a58 =sim(net,p58);
```

```
p59=[1;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a59 =sim(net,p59);
p510=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;1;0;0;1;];
a510 =sim(net,p510);

p6=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a6 =sim(net,p6);
p61=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a61 =sim(net,p61);
p62=[1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a62 =sim(net,p62);
p63=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a63 =sim(net,p63);
p64=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a64 =sim(net,p64);
p65=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;1;0;0;0;0;1;];
a65 =sim(net,p65);
p66=[1;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a66 =sim(net,p66);
p67=[1;0;0;0;0;0;0;0;0;0;1;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a67 =sim(net,p67);
p68=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;1;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a68 =sim(net,p68);
p69=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;1;0;0;0;0;0;0;0;1;];
a69 =sim(net,p69);
p610=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;1;0;1;];
a610 =sim(net,p610);

p7=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;0;0;0;1];
a7 =sim(net,p7);
p71=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1;];
a71 =sim(net,p71);
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p72=[1;0;0;0;1;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a72 =sim(net,p72);
p73=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a73 =sim(net,p73);
p74=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a74 =sim(net,p74);
p75=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a75 =sim(net,p75);
p76=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;1;0;0;0;0;0;1;];
a76 =sim(net,p76);
p77=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a77 =sim(net,p77);
p78=[1;0;0;0;0;0;0;1;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a78 =sim(net,p78);
p79=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;1;];
a79 =sim(net,p79);
p710=[1;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;1;0;0;0;
1;0;0;0;0;0;0;0;0;1;];
a710 =sim(net,p710);

p8=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;
0;0;0;0;0;0;1;0;0;0;];
a8 =sim(net,p8);
p81=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
a81 =sim(net,p81);
p82=[1;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
a82 =sim(net,p82);
p83=[1;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
a83 =sim(net,p83);
p84=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
a84 =sim(net,p84);
p85=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
a85 =sim(net,p85);
p86=[1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;1;0;0;0;];
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a86 =sim(net,p86);
p87=[1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;];
a87 =sim(net,p87);
p88=[1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;1;0;0;0;];
a88 =sim(net,p88);
p89=[1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;];
a89 =sim(net,p89);
p810=[1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;1;0;0;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;];
a810 =sim(net,p810);

p9=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1];
a9 =sim(net,p9);
p91=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a91 =sim(net,p91);
p92=[1;0;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a92 =sim(net,p92);
p93=[1;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a93 =sim(net,p93);
p94=[1;0;0;0;0;0;0;0;0;0;0;1;0;1;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a94 =sim(net,p94);
p95=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;1;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a95 =sim(net,p95);
p96=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a96 =sim(net,p96);
p97=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;];
a97 =sim(net,p97);
p98=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;1;];
a98 =sim(net,p98);
p99=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;0;1;];
a99 =sim(net,p99);
p910=[1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;0;1;];
a910 =sim(net,p910);
```

```
p10=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1
;0;0;0;0;0;0;0;0;0;1];
a10 =sim(net,p10);
p101=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a101 =sim(net,p101);
p102=[1;0;0;1;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a102 =sim(net,p102);
p103=[1;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a103 =sim(net,p103);
p104=[1;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a104 =sim(net,p104);
p105=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;1;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a105 =sim(net,p105);
p106=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;1;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a106 =sim(net,p106);
p107=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;
1;0;0;0;0;0;0;0;0;0;1;];
a107 =sim(net,p107);
p108=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;1;0;
1;0;0;0;0;0;0;0;0;0;1;];
a108 =sim(net,p108);
p109=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;
1;0;0;1;0;0;0;0;0;0;1;];
a109 =sim(net,p109);
p1010=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0
;1;0;0;0;0;0;0;1;0;0;1;];
a1010 =sim(net,p1010);

p11=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0
;0;0;0;0;1;0;0;0;0;0;];
a11 =sim(net,p11);
p111=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a111 =sim(net,p111);
p112=[1;0;1;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a112 =sim(net,p112);
p113=[1;0;0;0;0;0;0;1;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a113 =sim(net,p113);
p114=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
```

```
a114 =sim(net,p114);
p115=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a115 =sim(net,p115);
p116=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;1;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a116 =sim(net,p116);
p117=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;1;
0;0;0;0;0;1;0;0;0;0;0;];
a117 =sim(net,p117);
p118=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;1;0;0;];
a118 =sim(net,p118);
p119=[1;0;0;1;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;0;0;0;1;0;0;0;0;
0;0;0;0;0;1;0;0;0;0;0;];
a119 =sim(net,p119);
p1110=[1;0;0;0;0;0;0;0;0;0;1;0;0;0;1;0;0;0;0;0;0;0;0;0;0;0;1;0;0;0;0;0;1;0;0;1;0;0;0;0
;0;0;0;0;0;1;0;0;0;0;0;];
a1110 =sim(net,p1110);
```

```
%THE PROGRAM WHICH CLASSIFIES ARRHTHMIAS

clear all

TraningSize = 65 ;

% Normal beat 2237
NormalBeat_LowerBound =  0 ;
NormalBeat_UpperBound =  2237 ;

vector0=CreateRandomVector(NormalBeat_LowerBound,NormalBeat_UpperBound
,TraningSize);
train0 = vector0(1:50);
test0 = vector0(51:65);

% Left bundle branch block beat 8070
LeftBundleBranchBlockBeat_LowerBound = NormalBeat_UpperBound + 1 ;
LeftBundleBranchBlockBeat_UpperBound = NormalBeat_UpperBound + 8070 ;

vector1=CreateRandomVector(LeftBundleBranchBlockBeat_LowerBound,LeftBund
leBranchBlockBeat_UpperBound,TraningSize);
train1 = vector1(1:50);
test1 = vector1(51:65);

% Right bundle branch block beat 3440
RightBundleBranchBlockBeat_LowerBound =
LeftBundleBranchBlockBeat_UpperBound + 1 ;
RightBundleBranchBlockBeat_UpperBound =
LeftBundleBranchBlockBeat_UpperBound + 3440 ;

vector2=CreateRandomVector(RightBundleBranchBlockBeat_LowerBound,RightB
undleBranchBlockBeat_UpperBound,TraningSize);
train2 = vector2(1:50);
test2 = vector2(51:65);

% Atrial premature beat 564
AtrialPrematureBeat_LowerBound = RightBundleBranchBlockBeat_UpperBound +
1 ;
AtrialPrematureBeat_UpperBound = RightBundleBranchBlockBeat_UpperBound +
564 ;

vector3=CreateRandomVector(AtrialPrematureBeat_LowerBound,AtrialPremature
eat_UpperBound,TraningSize);
train3 = vector3(1:50);
test3 = vector3(51:65);

% Premature ventricular contraction 2323
```

```
PrematureVentricularContraction_LowerBound= AtrialPrematureBeat_UpperBound
+ 1 ;
PrematureVentricularContraction_UpperBound = AtrialPrematureBeat_UpperBound
+ 2323 ;

vector4=CreateRandomVector(PrematureVentricularContraction_LowerBound,Prem
atureVentricularContraction_UpperBound,TraningSize);
train4 = vector4(1:50);
test4 = vector4(51:65);

% Ventricular escape beat 105
VentricularEscapeBeat_LowerBound =
PrematureVentricularContraction_UpperBound + 1 ;
VentricularEscapeBeat_UpperBound =
PrematureVentricularContraction_UpperBound+ 105;

vector5=CreateRandomVector(VentricularEscapeBeat_LowerBound,VentricularEsc
apeBeat_UpperBound,TraningSize);
train5 = vector5(1:50);
test5 = vector5(51:65);

% Ventircular flatter wave 472
VentircularFlatterWave_LowerBound = VentricularEscapeBeat_UpperBound + 1 ;
VentircularFlatterWave_UpperBound = VentricularEscapeBeat_UpperBound + 472 ;

vector6=CreateRandomVector(VentircularFlatterWave_LowerBound,VentircularFlat
terWave_UpperBound,TraningSize);
train6 = vector6(1:50);
test6 = vector6(51:65);

Result = '' ;

%clear all

load arrdb;

%indis=find(arrdb(:,2)==0);
data0=arrdb(train0,3:2:63);
train_data0=data0';

%indis=find(arrdb(:,2)==1);
data1=arrdb(train1,3:2:63);
train_data1=data1';

%indis=find(arrdb(:,2)==2);
data2=arrdb(train2,3:2:63);
train_data2=data2';
```

```
%indis=find(arrdb(:,2)==3);
data3=arrdb(train3,3:2:63);
train_data3=data3';

%indis=find(arrdb(:,2)==4);
data4=arrdb(train4,3:2:63);
train_data4=data4';

%indis=find(arrdb(:,2)==5);
data5=arrdb(train5,3:2:63);
train_data5=data5';

%indis=find(arrdb(:,2)==6);
data6=arrdb(train6,3:2:63);
train_data6=data6';
%size(train_data6)

train_data=[train_data0 train_data1 train_data2 train_data3 train_data4 train_data5
train_data6];


% We will use a 9 by 9 layer of neurons to classify the vectors above. We would
% like each neuron to respond to a different region of the rectangle, and
% neighboring neurons to respond to adjacent regions.  We create a layer of 81
% neurons spread out in a 9 by 9 grid:

net = newsom([0 1; 0 1;0 1; 0 1;0 1; 0 1;0 1; 0 1;0 1; 0 1;0 1; 0 1;0 1; 0 1;0
1; 0 1;0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1; 0 1],[9 9]);

net.trainParam.epochs = 1000;
net = train(net,train_data);

testdata0=arrdb(test0,3:2:63);
test_data0=testdata0';
a0=sim(net,test_data0)

testdata1=arrdb(test1,3:2:63);
test_data1=testdata1';
a1=sim(net,test_data1)

testdata2=arrdb(test2,3:2:63);
test_data2=testdata2';
a2=sim(net,test_data2)

testdata3=arrdb(test3,3:2:63);
test_data3=testdata3';
a3=sim(net,test_data3)
```

```
testdata4=arrdb(test4,3:2:63);
test_data4=testdata4';
a4=sim(net,test_data4)

testdata5=arrdb(test5,3:2:63);
test_data5=testdata5';
a5=sim(net,test_data5)

testdata6=arrdb(test6,3:2:63);
test_data6=testdata6';
a6=sim(net,test_data6)
```