CHAPTER I

INTRODUCTION

1.1. INTRODUCTION

Studying the ECG signal provides an insight to understand life-threatening cardiac conditions [21]. This typically is centered on the study of arrhythmias, which can be any disturbance in the rate, regularity and site of origin or conduction of the cardiac electric impulse. Not all arrhythmias are abnormal or dangerous but some do require immediate therapy to prevent further problems.

A person’s ECG information can be recorded using a portable Holter monitor which is worn by the person. A Holter monitor typically employs a few electrodes and stores a recording of the person’s heart rhythm as they go about their daily activities over a 24 to 48 hour period. The Holter monitor is then returned to a cardiologist who examines the recordings and determines a diagnosis. Examining these recordings is a time-consuming and hence any automated processing of the ECG that assists the cardiologist in determining a diagnosis would be of assistance.

Beat classification is an important step in arrhythmia analysis as many arrhythmias simply consist of a single aberrant beat as opposed to a sustained rhythm disturbance [23]. A beat classifier attempts to classify a heartbeat into a normal beat or into a class representing one of many different arrhythmias. The rhythm of some ECG signals can be determined by knowing the beat classification of a number of consecutive beats in the signal.

Beat classification is a candidate task for automatic pattern recognition as it involves the labeling of beats on the basis of ECG waveform shape and temporal position relative to surrounding beats. Other authors have described previous work on this problem Senhadji et al. [1] explored the use of the discrete wavelet transform to discriminate between three beat types. Using the Daubechies’
orthogonal wavelets, Spline wavelets and Morlet type wavelets, they employed a beat classifier modelled on linear discriminants processing input features derived from distributions of energy and local extrema in the details corresponding to different levels of decomposition. Their study was conducted on a database set of 53 beats consisting of 20 normal, 13 premature ventricular contractions and 20 beats with an S-T segment deviation [26]. The data was divided into training (25 beats) and testing (28 beats) data sets. The classifier achieved a good accuracy in classifying the beats and was found to outperform a classifier processing features derived from the maximum magnitude of the P, QRS and T waves, the P-R and ST intervals and power spectral density measurements. Until the results are validated on a significantly larger database it is difficult to draw any real conclusions from this work. The beat classifier designed by Yeap et al. [2] was modeled using a feed forward neural network. The classifier’s performance was tested on the American Heart Association database; beats were classified into normal or premature ventricular contractions beat types. Four of the 80 available ECG records were used to train the classifier; the remaining records (excluding the ventricular tachycardia records) were used to test the classifier. The neural network consisted of two hidden layers each with 20 processing units. The input feature vector consisted of five features: the QRS width, the R wave’s amplitude, a measure of the QRS offset, the T wave slope and a measure of the R-R interval with respect to its mean value [25].

The classification rates of automatic beat classifiers presented in the literature to date have not been high enough for the classifiers to gain wide spread clinical acceptance. Hu et al. [3] notes that certain beat types are sufficiently rare that to date not enough ECG data has been collected to obtain a representative sample of the these populations and hence classifier training procedures are unable to properly model these classes. In order to boost the classification performance, they suggested customizing a beat classifier to a specific patient (known as a local
classifier) and then combining it with a global classifier designed from a large database of ECG signals. They modeled a global classifier on a feed-forward neural network with one hidden layer of seven processing units [27]. They used self-organizing maps and learning vector quantization to design the local classifier. The two classifiers were then combined using a Mixture Of Experts (MOE) approach. The MIT-BIH Arrhythmia database was used to examine the MOE classifier. Thirteen recordings were used to train the global classifier and 20 recordings were used to simulate the records of 20 patients. The feature vector consists of the QRS width, the instantaneous RR interval, the average R-R interval and 9 elements representing the QRS template and the classifier considered normal, premature ventricular contraction and fusion beats only. The local classifier significantly enhanced the performance of the global classifier with the MOE classifier achieving better accuracy on the same data set. In practice this method requires a cardiologist to annotate a segment of a patient-specific ECG in order to implement the MOE approach. The main drawback of this approach lies in the expert input required to customize this approach to each patient.

1.2. ELECTROCARDIOGRAM

The ECG is a recording of body surface potentials generated by the electrical activity of the heart. The recording and interpretation of the ECG has a very long history and is an important aspect of the clinical evaluation of an individual’s cardiac status and overall health.

1.2.1. ECG Beat Morphology

The normal heart beat begins as an electrical impulse generated in the sinoatrial node of the right atrium. From there, the electrical activity spreads as a wave over the atria and arrives at the atrioventricular node about 200 ms later. The atrioventricular node is the only electrical connection between the atria and ventricles. In approximately 100 ms, the wave front emerges on the other side and
rapidly spreads to all parts of the inner ventricular surface via the His-Purkinje system. The activation of the entire ventricular myocardium takes place in 80 ms.

The ECG waveform corresponding to a single heart beat consists of three temporally distinct wave shapes: the P wave, the QRS complex and the T wave. The P wave corresponds to electrical excitation of the two atria and is roughly 0.2 mV in amplitude. The QRS complex corresponds to electrical excitation of the two ventricles and has a peaked shape approximately 1 mV in amplitude. The T wave corresponds to the repolarization of the ventricles. It varies greatly from person to person, but is usually 0.1–0.3 mV in amplitude and ends 300–400 ms after the beginning of the QRS complex. The region between the QRS complex and T wave, called the ST segment, is the quiescent period between ventricular depolarization and repolarization (Figure 1.1). Algorithms for detecting ECG beats invariably focus on the QRS complex because its short duration and high amplitude makes it the most prominent feature [25].

![Figure 1.1: A Sample ECG Signal showing P-QRS-T Wave](image)

The bandwidth (difference between the highest-frequency signal component and the lowest-frequency signal component) of the ECG is not rigidly defined, although most of the clinically relevant energy falls between 0.05 and 50
Hz. However, important morphological features of the ECG may contain very little of the waveform’s energy. Therefore, reproduction resulting in unchanged clinical interpretation may not necessarily be related to traditional engineering metrics such as preservation of the waveform energy.

1.2.2. Noise

It should be no surprise that artifacts can be a problem in ECG analysis. Fortunately, the Signal-to-Noise Ratio (SNR) is usually quite good in a person at rest (SNR compares the level of a desired signal to the level of background noise). In an active person, however, there can be substantial low frequency (< 15 Hz) noise due to electrode motion and high frequency (> 15 Hz) noise due to skeletal muscle activity. In addition, there is the possibility of noise at 60 Hz and its harmonics due to power-line noise [30].

1.2.3. Arrhythmias

All normal heartbeats begin as an electrical impulse in the sinoatrial node and a sequence of normal heartbeats is referred to as a normal sinus rhythm. The term arrhythmia refers to an irregularity in the rhythm. Most arrhythmias are associated with electrical instability and, consequently, abnormal mechanical activity of the heart. Arrhythmias are typically categorized by the site of origin of the abnormal electrical activity. Although all normal heartbeats originate in the sinoatrial node, abnormal beats can originate in the atria, the ventricles, or the atrioventricular node (Figure 1.2) [119].

Arrhythmias can consist of isolated abnormal beats, sequences of abnormal beats interspersed with normal beats, or exclusively abnormal beats. From a clinical perspective, the severity of the arrhythmia depends on the degree to which it interferes with the heart’s ability to circulate oxygenated blood to itself and to the rest of the body. Isolated abnormal beats typically do not interfere with cardiac
function, although they do indicate an underlying pathology in the cardiac tissue. Rhythms dominated by abnormal beats are often more problematic.

![Heart Diagram]

<table>
<thead>
<tr>
<th>Pacemaker</th>
<th>Intrinsic Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinoatrial (SA) node</td>
<td>60-80/min</td>
</tr>
<tr>
<td>Atrioventricular (AV) node</td>
<td>40-50/min</td>
</tr>
<tr>
<td>Bundle of His</td>
<td>30-40/min</td>
</tr>
<tr>
<td>Purkinje fibers</td>
<td>15-30/min</td>
</tr>
</tbody>
</table>

**Figure 1.2: Specialized Neural-Like Conductive Tissues and their Approximate Intrinsic Rates**

Many of them can be treated with medication, while the most severe arrhythmias are fatal if not treated immediately. Two especially dangerous arrhythmias are ventricular flutter and ventricular fibrillation. Ventricular flutter is produced by a focus of ventricular cells firing at a rate of 200–300/minute. In ventricular flutter the ventricles contract at such a high rate that there is not enough time for them to fill with blood, so there is effectively no cardiac output. Untreated ventricular flutter almost always leads to ventricular fibrillation, because the lack of blood supply causes many other foci in the ventricles to fire independently [28, 29].

In ventricular fibrillation, many foci of ventricular cells fire, each at its own rate. Because there are so many foci firing at once, each one causes only a small area of ventricle to depolarize, resulting in a chaotic twitching (Muscle contraction and relaxation) of the cardiac muscle and no cardiac output. Ventricular fibrillation is an emergency situation which requires treatment by electrical shock to restore the heart to a normal sinus rhythm.
1.2.4. Automated Arrhythmia Detection

Not surprisingly, much effort has been devoted to the development of automated arrhythmia detection systems for monitoring hospital patients. In such systems, the ECG signal is picked up by surface electrodes on the patient, amplified, lowpass filtered and digitized before processing. Many signal processing techniques have been studied for reducing noise and identifying relevant features of digital ECG signals. The output of the system is a diagnosis of the rhythm which is typically recorded and/or displayed on the monitor. In addition, the detection of rhythms requiring immediate attention generally triggers an alarm. Although detection of VF or VFIB should obviously generate an alarm, it is important to minimize the false alarms (number of normal traces that were classified as arrhythmic) produced by such systems. Experience has shown that automated arrhythmia detectors with high rates of false alarms are typically disabled or ignored by hospital staff [24].

1.3. GENERAL PRINCIPLES OF CARDIAC FUNCTION

The output of the heart per minute (cardiac output) is the paramount cardiovascular event required to sustain blood flow throughout the body. In addition to blood volume and contractile strength, the heart must sustain a regular cycle of relaxation and contraction if it is to fulfill its objective. This regularity is predicated on a series of complex electrophysiological events within the cardiac tissues that can be monitored using a device called the electrocardiogram. This device is variably referred to as an ECG or as an EKG, the latter based on the Greek term “kardia” for heart. Many prefer EKG to ECG because it is less likely to be confused verbally with EEG, the abbreviation for electroencephalogram.

The standard events required for a normal cardiac cycle are the rhythmic contraction and relaxation of the atria and ventricles. The heart is composed of two principal cell types: working cells and specialized neural like conductive cells. The working cells are the muscle or myocardium of the atria and ventricles.
Specialized cells include the Sinoatrial (SA) node, the Atrioventricular (AV) node, the bundle of His and the Purkinje fibers (Figure 1.2) [119].

These cells initiate and conduct electrical impulses throughout the myocardium and this regulates the rhythm of a cardiac cycle. In order to initiate impulses, specialized cells have a property called automaticity, which reflects an ability to initiate electrical impulses spontaneously. This is independent of any nerves or hormones, but their actual rate of firing can be influenced by autonomic nerves, with sympathetic increasing and parasympathetics decreasing their rate. Each cardiac cycle commences with an impulse, spontaneously generated by the SA node that subsequently spreads throughout the remainder of the neural-like conductive tissues and onto the muscle (myocardial) cells. Abnormalities within this conduction system will compromise cardiac output and are called arrhythmias or dysrhythmias synonymously.

1.4. ELECTROPHYSIOLOGICAL CONSIDERATIONS

To fully appreciate electrical impulses and the information provided by an ECG, we must first review fundamental concepts regarding electrical membrane potentials. All cardiac cell membranes are positively charged on their outer surfaces because of the relative distribution of cations (an ion or group of ions having a positive charge). This resting membrane potential is maintained by an active transport mechanism called the sodium-potassium pump. When the cell is stimulated, ion channels open, allowing a sudden influx of sodium and/or calcium ions and thereby reversing the resting potential. This period of depolarization is very brief because sodium channels close abruptly, denying further influx of sodium. Simultaneously, potassium channels open and allow intracellular potassium to diffuse outward while sodium ions are actively pumped out. This reestablishes a positive charge to the outside of the membrane, a process called repolarization that returns the membrane to its resting membrane potential. The processes of depolarization and repolarization are referred to collectively as an
action potential. This event self-propagates as an impulse along the entire surface of a cell and from one cell to another, provided that their membranes are connected (Figure 1.3) [119].

All human cells exhibit this phenomenon and its purpose varies according to the cell’s function. The purpose of action potentials in neurons is to initiate release of neurotransmitters that either excite or stabilize cell membranes of the tissue innervated (supply of nervous energy). In skeletal and cardiac muscle cells, action potentials release stored calcium ions that initiate the actual contractile process.

![Diagram of depolarization and repolarization of cell membranes](image)

(a) Polarized Resting Cell-Stage 1  (b) Depolarizing Cell-Stage 2
(c) Depolarized Cell-Stage 3  (d) Repolarizing Cell-Stage 4

**Figure 1.3: Depolarization and Repolarization of Cell Membranes.** (a) The Resting Cell Membrane is Charged Positively on the Outside and Negatively on the Inside. (b) Following a Stimulus (S), Positive Ions Enter the Cell Reversing this Polarity. (c) This Process Continues until the Entire Cell is Depolarized. (d) Ions are returned to their Normal Location and the Cell Repolarizes to its Normal Resting Potential

Cells comprising the heart’s conduction system are unique in two aspects. First of all, they possess automaticity. The physiological explanation for this property resides in the resting membrane’s partial permeability to calcium and/or
sodium ions. The gradual inward ‘‘leak’’ of cations decreases the voltage of the resting potential until a threshold is reached. At this point, all channels open and rapid cation influx depolarizes the membrane.

The second unique characteristic of this specialized tissue is the fact that, unlike classic neural tissue, these cells do not release neurotransmitters. Instead, they are in direct contact with cardiac muscle and their action potential initiates depolarization of the cardiac muscle cells directly.

Cardiac muscle cells are fused to one another by special attachments called intercalated discs. This allows them to function as a continuous sheet of cells called a syncytium. The atrial syncytium is separated from that of the ventricles by a layer of connective tissue that acts as an insulator. The SA node initiates depolarization of the atrial muscle, but the insulation precludes propagation into the ventricles except at one place, the AV node.

The AV node delays and finally relays the impulse along the common bundle of His, which penetrates the connective tissue to enter the ventricles. The impulse continues along the common bundle of His and its branches until it finally reaches the Purkinje fibers, which ignite the ventricular muscle syncytium. The action potential of an individual cell can be measured using microprobes inserted through its cell membrane. It is far too small an electrical event to be measured by surface electrodes. However, action potentials that spread throughout the muscle syncytia of the heart are great enough for surface electrodes to record and produce a tracing known as an ECG. It is important to appreciate that the ECG cannot record electrical events generated by the specialized cells of the conduction system; their voltages are far too small. However, other events can be deduced from the tracing.
1.5. THE ECG TRACING

The electrical sequence of a cardiac cycle is initiated by the sinoatrial node, the so-called pacemaker of the heart. This is because the SA node has a faster rate of spontaneous firing than the remaining specialized tissues as shown in figure 1.4. However, if this rate decreases, other portions of this specialized system can gain control, a phenomenon termed escape [119].

The baseline of an ECG tracing is called the isoelectric line and denotes resting membrane potentials. Deflections from this point are lettered in alphabetical order and following each, the tracing normally returns to the isoelectric point. The first deflection is the P wave and represents depolarization of atrial muscle cells. It does not represent contraction of this muscle, nor does it represent firing of the SA node. These events are deduced based on the shape and consistency of the P waves. One assumes that the SA node fires at the start of the P wave and one assumes that atrial contraction begins at the peak of the P wave. Although atrial repolarization follows depolarization, the ECG provides no evidence of this event. A popular misconception is that evidence of repolarization is obscured by the subsequent QRS complex. However, repolarization would be observed in cases where the QRS complex is delayed or absent, eg, AV blocks. The correct explanation is that atrial repolarization is too minor in amplitude to be recorded by surface electrodes.

The QRS complex represents depolarization of ventricular muscle cells. The Q portion is the initial downward deflection, the R portion is the initial upward deflection and the S portion is the return to the baseline, or the so-called isoelectric point. Often, the Q portion is not evident and the depolarization presents as only an “RS” complex. In any case, the complex does not represent ventricular contraction. One assumes that contraction will commence at the peak of the R portion of the complex. Unlike contraction of the atria, ventricular
contraction can be confirmed clinically by palpating a pulse or by monitoring a pulse oximeter wave form.

<table>
<thead>
<tr>
<th>Physiologic Event</th>
<th>ECG Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. SA node initiates impulse</td>
<td>Not visible</td>
</tr>
<tr>
<td>2. Depolarization of atrial muscle</td>
<td>P wave</td>
</tr>
<tr>
<td>3. Atrial contraction</td>
<td>Not visible</td>
</tr>
<tr>
<td>4. Depolarization of AV node &amp; Common Bundle</td>
<td>Not visible</td>
</tr>
<tr>
<td>5. Repolarization of atrial muscle</td>
<td>Not visible</td>
</tr>
<tr>
<td>6. Depolarization of ventricular muscle</td>
<td>QRS complex</td>
</tr>
<tr>
<td>7. Contraction of ventricular muscle</td>
<td>Not visible</td>
</tr>
<tr>
<td>8. Repolarization of ventricular muscle</td>
<td>T wave</td>
</tr>
</tbody>
</table>

**Figure 1.4: Summary of Events of a Cardiac Cycle**

A patient in cardiac arrest may have normal QRS complexes on his or her ECG; ventricular muscle cells are depolarizing, but there is no contraction. This phenomenon is called pulseless electrical activity. Following depolarization, ventricular muscle repolarizes and this event is great enough in amplitude to generate the T wave on the ECG tracing. The PR interval is measured from the beginning of the P wave to the beginning of the R portion of the QRS complex. (This is conventional because the Q portion of the complex is so frequently indiscernible.) Because the PR interval commences with atrial muscle depolarization and ends with the start of ventricular depolarization, one can assume that the electrical impulse passes through the AV node into the ventricle during this interval. If the PR interval is prolonged, one may presume that AV block is present (Figure 1.4) [119].

1.5.1. **Technical Considerations**

In 1901 a Dutch physiologist, Willem Einthoven, developed a galvanometer that could record the electrical activity of the heart. He found that a tracing can be produced as action potentials spread between negatively and positively charged electrodes. (A third electrode serves to ground the current.) He
found that tracings varied according to the location of the positive and negative electrodes and subsequently described 3 angles or leads in the form of a triangle with the heart in the middle. This is known today as Einthoven’s triangle and the 3 electrode arrangements are known as the primary limb leads I, II and III. As research continued throughout the 20th century, additional arrangements were discovered that enable physicians to analyze electrical events as they spread in many directions through the heart, much like an apple slicer sections an apple into various parts. Today, the cardiologist analyzes a 12-lead ECG to aid in diagnosing infarctions, hypertrophy and complex arrhythmias. However, other purpose is to identify only the basic arrhythmias that justify dynamic ECG monitoring during sedation and general anesthesia. For this purpose, a single-lead ECG is all that is required. Most often, lead II is selected because it generally records the largest waves [119].

1.6. ECG PAPER

An ECG monitor displays a tracing that lacks any grid as background. However, most of these monitors are equipped with optional printers that can generate a gridded printout if desired. As the stylus of the recording device is deflected by electrical currents, the recording paper is moving at a speed of 25 mm/s. This creates an ECG tracing whose components can be measured.

The vertical axis of an ECG denotes voltage and the direction of waveforms from the baseline. These considerations are generally irrelevant during routine monitoring, but have significance for diagnosing ischemia and infarction. The horizontal axis denotes time and sequence of events, both of which are essential for arrhythmia recognition. Standard ECG recording paper is divided into small and large squares. The former represents 0.04-second intervals. Five small squares constitute a large square, which represents 0.20 seconds. Notice, in Figure 1.5, that the lines between every 5 boxes are heavier, so that each 5-mm unit horizontally
corresponds to 0.2 seconds (5\times 0.04=0.2). The ECG can therefore be regarded as a moving graph with 0.04- and 0.2-second divisions [119].

![Figure 1.5: Standard ECG Paper](image)

1.7. ECG ANALYSIS

Dynamic ECG monitors display heart rate, but it can also be ascertained from a printed tracing using either of two methods:

1. When the heart rate is regular, count the number of large (0.2-second) boxes between 2 successive QRS complexes and divide 300 by this number [22]. The number of large time boxes is divided into 300 because 300\times 0.20 = 60 and heart rate is calculated in beats per minute or 60 seconds. For example, if there are 3 large boxes between QRS complexes, the heart rate is 100 beats/min, because 300/3=100. Similarly, if 4 large time boxes are counted between QRS complexes, the heart rate is 75 beats/ min (Figure 1.6).

![Figure 1.6: Normal ECG Tracing](image)
2. If the heart rate is irregular, the first method will not be accurate because the intervals between QRS complexes vary from beat to beat. In most cases, ECG graph paper is scored with marks at 3-second intervals. In such cases simply count the number of QRS complexes every 3 or 6 seconds and multiply this number by 20 or 10 respectively.

How one chooses to analyze an ECG rhythm strip is arbitrary. Each clinician must adopt a sequence of analysis that accommodates personal methods of reasoning. Always keep in mind that events during the PR interval pertain to supraventricular activity. When abnormalities are detected, try to establish the event as ventricular or supraventricular in origin. The following sequence represents one suggestion for analysis of an ECG tracing. It is described as 5-step analysis.

**Step 1: Is the Rhythm Regular or Irregular?**

If the intervals between QRS complexes (R-R intervals) are consistent, ventricular rhythm is regular. If intervals between P waves (P-P intervals) are consistent, the atrial rhythm is regular. In figure 1.6 the rhythm is regular.

**Step 2: Are all QRS Complexes Similar and Are they Narrow?**

The duration of the QRS complex should not exceed 0.10 seconds (2½ small squares). A widened complex indicates ventricular enlargement (hypertrophy) or that ventricular depolarization is being initiated by pacemaker issue below the AV node, eg, ventricular-paced rhythm. In this case, one ventricle depolarizes first and the current must spread into the second ventricle. This takes more time than when the current spreads down the bundle into both ventricles simultaneously. If QRS complexes are narrow, the rhythm is being initiated by a pacemaker at the AV node or higher and is described as a supraventricular rhythm. If the complexes are wide, the pacemaker is in the ventricles and is described as a ventricular rhythm. Should complexes vary in appearance, more than one
pacemaker is generating impulses. This phenomenon is referred to as ectopic pacemakers and the rhythm described as ectopy.

**Step 3: Are all P Waves Similar and Are PR Intervals Normal?**

If P waves are all similar and normal in shape, one can assume that the SA node is the primary pacemaker. In this case the rhythm is sinus in character. If P waves vary in shape or absent, other tissue(s) will function as pacers.

The PR interval is normally 0.12–0.20 seconds (3–5 small squares). Longer intervals indicate that the impulse is being delayed from entering the ventricles and the condition is designated AV block.

**Step 4: Is the Rate Normal?**

If the rhythm is regular, count the number of large squares between QRS complexes and divide 300 by this number. However, if the rhythm is irregular, count the number of QRS complexes in a 6-second segment and multiply by 10. Rates below 60 indicate bradycardia; those above 100 indicate tachycardia. In figure 1.6, there are approximately 4 large boxes between QRS complexes, so the rate is approximately 75.

**Step 5: Do Waves and Complexes Proceed in Normal Sequence?**

Each P wave should be followed by a QRS complex, which is followed by a T wave. This assures a normal sequence for each cardiac cycle.

1.8. **ARRHYTHMIA IDENTIFICATION**

Most basic courses in ECG interpretation emphasize the precise recognition of at least 15–20 arrhythmias. The primary objectives are rote memorization of a name for each rhythm and its deviant characteristics.

However, this approach nurtures an inability to assess the clinical significance of a particular arrhythmia. ECG analysis must be correlated with the patient’s appearance and vital signs. Collectively, these will establish the clinical
significance of the electrical disturbance and determine any indication for intervention.

1.8.1. Basic Arrhythmia Groups

By performing the first two steps described above, it is possible to organize all basic arrhythmias into 4 groups.

(i) **Rhythms in Group A**

During the first 2 steps of 5-step analysis, can find that the R-R intervals are regular and all QRS complexes are narrow. It denotes that the heart is being paced from tissue above the ventricle. For each, apply steps 3–5 of 5-step analysis.

(ii) **Rhythms in Group B**

During the first 2 steps of 5-step analysis, can find that the R-R intervals are irregular but all QRS complexes are narrow. It denotes that the heart is being paced from tissue above the ventricles. For each, apply steps 3–5 of 5-step analysis.

(iii) **Rhythms in Group C**

During the first 2 steps of 5-step analysis, can find that the R-R intervals are regular but all QRS complexes are wide. It denotes that the heart is being paced from tissue below the AV node, within the ventricles. For each, apply steps 3–5 of 5-step analysis.

(iv) **Rhythms in Group D**

During the first 2 steps of 5-step analysis, can find that the R-R intervals are irregular and that the QRS complexes vary in shape. For each, apply steps 3–5 of your 5-step analysis.
1.8.2. Extreme Low- and High-Frequency ECG

Although the accepted range of the diagnostic ECG is often quoted to be from 0.05 Hz (for ST analysis) to 40 or 100 Hz, information does exist beyond these limits. Ventricular Late Potentials (VLPs) are microvolt fluctuations that manifest in the terminal portion of the QRS complex and can persist into the ST-T segment. They represent areas of delayed ventricular activation which are manifestations of slowed conduction velocity (resulting from ischemia or deposition of collagens after an acute myocardial infarction). VLPs, therefore, are interesting for heart disease diagnosis [4–6]. The upper frequency limit of VLPs can be as high as 500 Hz [7].

On the low frequency end of the spectrum, Jarvis and Mitra [8] have demonstrated that sleep apnea may be diagnosed by observing power changes in the ECG at 0.02 Hz.

1.8.3. Spectral Nature of Arrhythmias

Arrhythmias, which manifest due to abnormalities in the conduction pathways of the heart, can generally be grouped into either atrial or ventricular arrhythmias. Ventricular arrhythmias manifest as gross distortions of the beat morphology since the depolarization begins in the ventricles rather than the atria. The QRS complex becomes broader due to the depolarization occurring along an abnormal conduction path and therefore progressing more slowly, masking the latent P wave from delayed atrial depolarization. Figure 1.7(c) illustrates a 5-second segment of Ventricular Tachycardia (VT) with a high heart rate of around 180 bpm (beats per minute) or 3 Hz and the accompanying power spectral density [Figure 1.7(d)]. Although the broadening of the QRS complexes during VT causes a shift in the QRS spectral peak to slightly lower frequencies, the overall peaks are similar to the spectrum of a sinus rhythm3 (Figure 1.8) and therefore, spectral separation between sinus and VT rhythms is difficult. Figure 1.7(a) shows a 5-
second segment of sinus rhythm ECG for the same patient before the episode of VT, with a relatively high heart rate (108 bpm).

![Graphs of Sinus, VT, VFL, and VFB](image)

**Figure 1.7: Spectral Nature of Arrhythmias**

Figure 1.7 (a) Sinus rhythm and (b) corresponding Power Spectral Density (PSD). (c) Ventricular Tachycardia (VT) and (d) corresponding PSD. (e) Ventricular Flutter (VFL) and (f) corresponding PSD. (g) Ventricular Fibrillation (V-Fib) and (h) corresponding PSD. Note that ventricular beats exhibit broader QRS complexes and therefore a shift in QRS energy to lower frequencies. Note also that higher frequencies (than normal) also manifest. VFL destroys many of the subtle ECG features and manifests as a sinusoidal like oscillation around the frequency of the (rapid) heart rate. VFIB manifests as a less organized and more rapid oscillation and therefore the spectrum is broader with more energy at higher
frequencies. (All PSDs were calculated on 5-second segments with the same parameters as in figure 1.8, but linear scales are used for clarity.)

Figure 1.8: Ten seconds of 125-Hz Typical ECG in Sinus Rhythm recorded with a Lead II Placement (Upper Plot) and Associated Linear and Log-Linear Periodograms (Middle and Lower Plots, respectively). A 256-point Welch Periodogram was used with a Hamming Window and a 64-point Overlap for the PSD Calculation

Note that although the P waves, QRS complexes and T waves are discernible above the noise, the main spectral component is the 1- to 2-Hz baseline noise.

When the ventricular activation time slows sufficiently, QRS complexes become severely broadened and Ventricular Flutter (VFL) is possible. This arrhythmia manifests as sinusoidal-like disturbances in the ECG and is therefore
relatively easy to detect through spectral methods. Figure 1.7(e) illustrates a 4-second segment of transient VFL and the corresponding power spectrum [Figure 1.7(f)]. If the ventricular arrhythmia is more erratic and manifests with a higher frequency of oscillation, then it is known as the extreme condition Ventricular Fibrillation (VFIB).

![Graph](image)

**Figure 1.9:** (a) Atrial Fibrillation (AF) and (b) corresponding PSD. Note the Similarity to Sinus Rhythm in figure 1.7 (a, b). (All PSDs were calculated with the Same Parameters as in figure 1.7)

Colloquially, the heart is said to be squirming “like a bag of worms,” with little or no coherent activity. At this point, the heart is virtually useless as a pump and immediate physical or electrical intervention is required to encourage the cardiac cells to depolarize/repolarize in a coherent manner. Atrial arrhythmias, in contrast to ventricular arrhythmias, manifest as small disturbances in the timing
and relative position of the (relatively low amplitude) P wave and are therefore difficult to detect through spectral methods. Figure 1.9 illustrates the ECG and its corresponding power spectrum for an atrial arrhythmia. Atrial arrhythmias do, however, manifest significantly different changes in the beat-to-beat timing and can therefore be detected by collecting and analyzing statistics on such intervals [9].

1.8.4. Standard Clinical ECG Features

Clinical assessment of the ECG mostly relies on relatively simple measurements of the intrabeat timings and amplitudes. Averaging over several beats is common to either reduce noise or average out short-term beat-to-beat interval-related changes. The complex heart rate-related changes in the ECG morphology (such as QT hysteresis) can themselves be indicative of problems. However, a clinician can extract enough diagnostic information to make a useful assessment of cardiac abnormality from just a few simple measurements.

Table 1.1:

Typical Lead II ECG Features and their Normal Values in Sinus Rhythm at a heart Rate of 60 bpm for a Healthy Male Adult (see text and figure 1.10 for definitions of intervals)

<table>
<thead>
<tr>
<th>Feature</th>
<th>Normal Value</th>
<th>Normal Limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>P width</td>
<td>110 ms</td>
<td>±20 ms</td>
</tr>
<tr>
<td>PQ/PR interval</td>
<td>160 ms</td>
<td>±40 ms</td>
</tr>
<tr>
<td>QRS width</td>
<td>100 ms</td>
<td>±20 ms</td>
</tr>
<tr>
<td>QT interval</td>
<td>400 ms</td>
<td>±40 ms</td>
</tr>
<tr>
<td>P amplitude</td>
<td>0.15 mV</td>
<td>±0.05 mV</td>
</tr>
<tr>
<td>QRS height</td>
<td>1.5 mV</td>
<td>±0.5 mV</td>
</tr>
<tr>
<td>ST level</td>
<td>0 mV</td>
<td>±0.1 mV</td>
</tr>
<tr>
<td>T amplitude</td>
<td>0.3 mV</td>
<td>±0.2 mV</td>
</tr>
</tbody>
</table>

ms-milliseconds, mV-milliVolt
Figure 1.10 illustrates the most common clinical features and table 1.1 illustrates typical normal values for these standard clinical ECG features in healthy adult males in sinus rhythm, with their upper and lower limits of normality.

Note that these figures are given for a particular heart rate. It should also be noted that the heart rate is calculated as the number of P-QRS-T complexes per minute, but is often calculated over shorter segments of 15 and sometimes 30 seconds. In terms of modeling consider the heart rate as the operating point around which the local interbeat interval rises and falls. Subsequently, it is simple to calculate a heart rate over any scale, up to a single beat. In the latter case, the heart rate is termed the instantaneous (or beat-to-beat) heart rate, \( HR_i = 60/RR_n \), of the \( n \)th beat. Each consecutive beat-to-beat, or RR, interval will be of a different length (unless the patient is paced) and a correlated change in ECG morphology is seen on a beat-to-beat basis.

Often, the RR interval will oscillate periodically, shortening with inspiration (and lengthening with expiration). This phenomenon, known as Respiratory Sinus Arrhythmia (RSA) is partly due to the Bainbridge reflex, the expansion and contraction of the lungs and the cardiac filling volume caused by variations of intrathoracic pressure [10]. During inspiration, the pressure within the thorax decreases and venous return increases, which stretches the right atrium resulting in a reflex that increases the local heart rate (i.e., shortens the RR intervals). During expiration, the reverse of this process results in a slowing of the local heart rate. In general, the normal beat-to-beat changes in morphology are ignored, except for derivations of respiration, although the phase between the respiratory RR interval oscillations and respiratory-related changes in ECG morphology is not static. The reason for this is that the mechanisms which alter amplitude and timing on the ECG are not exactly the same (although they are coupled either mechanically or neurally with a phase delay which may change from beat to beat). Changes in the features in table 1.1 and figure 1.10, therefore,
occur on a beat-to-beat basis as well as because of shifts in the operating point (average heart rate), although this is a second order effect.

![Diagram of ECG waves](image.png)

**Figure 1.10: Standard Fiducial Points in the ECG (P, Q, R, S, T and U) together with Clinical Features (Listed in Table 1.1)**

The PR interval extends from the start of the P wave to the end of the PQ junction at the very start of the QRS complex (that is, to the start of the R or Q wave). Therefore, this interval is sometimes known as the PQ interval. This interval represents the time required for the electrical impulse to travel from the SA node to the ventricle and normal values range between 120 and 200 ms. The PR interval has been shown to lengthen and shorten with respiration in a similar manner to the RR interval, but is less pronounced and is not fully correlated with the RR interval oscillations [11].

The global point of reference for the ECG’s amplitude is the isoelectric level, measured over the short period on the ECG between the atrial depolarization (P wave) and the ventricular depolarization (QRS complex). In general, this point
is thought to be the most stable marker of 0V for the surface ECG since there is a short pause before the current is conducted between the atria and the ventricles.

Interbeat segments are not usually used as a reference point because activity before the P wave can often be dominated by preceding T-wave activity. The QRS width is representative of the time for ventricles to depolarize, typically lasting 80 to 120 ms. The lower the heart rate, the wider the QRS complex, due to decreases in conduction speed through the ventricle. The QRS width also changes from beat-to-beat based upon the QRS axis, which is correlated with the phase of respiration and with changes in RR interval and therefore the local heart rate. The RS segment of the QRS complex is known as the Ventricular Activation Time (VAT) and is usually shorter (lasting around 40 ms) than the QR segment. This asymmetry in the QRS complex is not a constant and varies based upon changes in the Autonomic Nervous System (ANS) axis, lead position, respiration and heart rate.

The QRS complex usually rises (for positive leads) or falls to about 1 to 2 mV from the isoelectric line for normal beats. Artifacts (such as electrode movements) and abnormal beats (such as ventricular ectopic beats) can be several times larger in amplitude. In particular, baseline wander can often be the largest amplitude signal on the ECG, with the QRS complexes appearing as almost indistinguishable periodic anomalies. For this reason, it is important to allow sufficient dynamic range in the amplification (or digital storage) of ECG data. The point of inflection after the S wave is known as the j-point and is often used to define the beginning of the ST segment. In normals, it is expected to be isoelectric since it is the pause between ventricular depolarization and repolarization. The ST level is generally measured around 60 to 80 ms after the j-point, with adjustments for local heart rates. Abnormal changes in the ECG, defined by the Sheffield criteria [12], are ST level shifts $\geq 0.1$ mV (or about 5% to 10% of the QRS amplitude for a sinus beat on a V5 lead). Since only small deviations form the isoelectric level are significant markers of cardiac abnormality (such as ischemia),
the correct measurement of the isoelectric line is crucial. The interbeat segments between the end of the P wave and start of the Q wave are so short (less than 10 samples at 125 Hz), that the isoelectric baseline measurement is prone to noise. Multiple-beat averaging is therefore often employed. ST segment and j-point elevation, common in athletes, has been reported to normalize with exercise [13] and therefore j-point elevations may be difficult to distinguish from other changes seen in ECG.

The QT interval is measured between the onset of the QRS complex and the end of the T wave. It is considered to represent the time between the start of ventricular depolarization and the end of ventricular repolarization and is therefore useful as a measure of the duration of repolarization. The QT interval varies depending on heart rate, age and gender. As with some other parameters in the ECG, it is possible to approximate the (average) heart rate dependency of the QT interval by multiplying it by a factor $\alpha = (R’R) - 12$ where $R’R$ is the local average RR interval.

The resultant QT interval is called the corrected QT interval, QTc [14]. However, this factor works over a limited range and is subject dependent to some degree, over and above the usual confounding variables of age, gender and drug regime. Furthermore, ANS activity shifts can also change $\alpha$. In general, the last RR interval duration affects the action potential and hence the QT interval. It is also known that the QT-RR dependence is both a function of the average heart rate and the instantaneous interval, RRI [15]. Note that there is some variation in these parameters between lead configurations. Although interlead differences are sometimes used as cardiovascular markers themselves (such as in QT dispersion [16]), it is unclear whether there is a specific physiological origin to such differences or whether such metrics are just measuring an artifact which correlates with a clinical marker [17, 18].
One of the problems in measuring the QT interval correctly (apart from the
noise in the ECG and the resultant onset and offset ambiguities) is due to the
changes in the j-point and T wave morphology with heart rate. It has been
observed that as the heart rate increases, the T wave increases in height and
becomes more symmetrical [19]. Furthermore, in some subject groups (such as
athletes), the T wave is often observed to be inverted [13].

To summarize, the following changes are typically observed with
increasing heart rate [13, 19, 20]:

- The average RR interval decreases.
- The PR segment shortens and slopes downward (in the inferior leads).
- The P wave height increases.
- The Q wave becomes slightly more negative (at very high heart rates).
- The QRS width decreases.
- The R wave amplitude decreases in the lateral leads (e.g., V5) at and just
  after high heart rates.
- The S wave becomes more negative in the lateral and vertical leads (e.g.,
  V5 and aVF). As the R wave decreases in amplitude, the S wave increases
  in depth.
- The j-point often becomes depressed in lateral leads. However, subjects
  with a normal or resting j-point elevation may develop an isoelectric j-point
  with higher heart rates.
- The ST level changes (depressed in inferior leads).
- The T wave amplitude increases and becomes more symmetrical (although
  it can initially drop at the onset of a heart rate increase).
- The QT interval shortens (depending on the autonomic tone).
- The U wave does appear to change significantly. However, U waves may
  be difficult to identify due to the short interval between the T and following
  beat’s P waves at high heart rates.
It should be noted however, that this simple description is insufficient to describe the complex changes that take place in the ECG as the heart rate increases and decreases.

1.9. SUMMARY

This chapter gives the overview of the ECG and its analysis. The chapter gives the importance of the electrocardiogram in the bio medical field. It briefly presents the details about general principles of ECG and arrhythmia identification.