ECE 417 TERM PROJECT

Electrocardiogram (ECG) + Detection of Events

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May 2021

SUMMARY

In this article mentioned about: electrocardiogram, structure and function of heart, cardiac muscle and excitation process, signal of cardiovascular system, origin of ECG and type of waves, ECG electrode placement, modeling of ECG, heart rate, diseases, processing and feature extraction of ECG, derivative based method for QRS detection.

INTRODUCTION

Electrocardiogram (ECG) is the most commonly used biomedical signal in clinical diagnostics of the heart. The word “electrocardiogram” is a combination of three words: electro, pertaining to electric signal; cardio, which translates into heart; and gram, which stands for recording. The recording of the electric activity of the heart is called ECG.

An ECG is often used alongside other tests to help diagnose and monitor conditions affecting the heart. It can be used to investigate symptoms of a possible heart problem, such as chest pain, palpitations (suddenly noticeable heartbeats), dizziness and shortness of breath.[1]

An ECG can help detect:

Arrhythmias,

coronary heart disease,

heart attacks,

cardiomyopathy.[2]

In this report, first, the function of the heart as a pump will be discussed. Then, in order to fully understand the electric signals generated by the heart with respect to each contraction, some basic phenomena involved in the contraction process will be described. Finally, the formation, measurement, and processing of ECG and Detection of Events will be discussed.[3]

1) HEART

As a central part of the circulatory system, the heart is primarily responsible for pumping blood and distributing oxygen and nutrients throughout the body. Because of this task, the heart may be considered one of the most important organs of the body, such that even small dysfunctions or abnormalities may cause drastic changes or effects in the human organism. The heart is a muscle whose working mechanism is made possible by the many parts that operate together. The organ is divided into several chambers that take in and distribute oxygen-poor or oxygen-rich blood. These chambers are accompanied by veins and arteries that facilitate the same function. With all of its parts working together towards the same goal, the heart successfully pumps blood with ease. Normally, a good-functioning adult heart could go on three cardiac cycles or 72 beats per minute—this rate changes for children whose heart rates are normally and relatively faster. Heart is shown in **Figure 1.**

1.2) Structure of Heart

The heart can be found at the chest's center, underneath the sternum in a thoracic compartment. It comprises four chambers and several valves that regulate the normal flow of blood within the body.

Two chambers called atria are located in the upper portion of the heart with the left atrium receiving oxygen-rich blood and the right receiving oxygen-free blood. The valves that separate these chambers are called atrioventricular valves, composed of the tricuspid valve on the left and the mitral valve on the right.

On the other hand, ventricles are chambers found on the lower portion of the heart; they pump oxygen-enriched blood into the body's organs, reaching even the smallest cells. Similar to the atria, valves also separate the ventricular chambers. Collectively-termed as semilunar valves, these are comprised of the pulmonary and aortic valves. The heart also has a wall that is composed of three layers: the outer layer epicardium (thin layer), the middle layer myocardium (thick layer), and the innermost layer endocardium (thin layer). The myocardium is thick because it is made up of cardiac muscle fibers.

The heart structure is made more complex because of the mechanisms that allow blood to be distributed throughout the body and return to the heart. Facilitating this continuous process are two types of blood vessels: veins and arteries. The vessels that bring oxygen-free blood back into the heart are called veins; those that bring oxygen-rich blood away from the heart and to other body parts are called arteries.

Functioning in the left ventricle, the largest artery is called the aorta. The aorta is considered a main artery in the body. It further splits into two smaller arteries called common iliac arteries.

With regular functioning, the heart can continuously supply a sufficient amount of oxygen to all parts of the body. Structure of the heart is shown in **Figure 2 and 3.**

1.3) Function of Heart

The heart is the main organ in the circulatory system, the structure is primarily responsible for delivering blood circulation and transportation of nutrients in all parts of the body. This continuous task uplifts the heart's role as a vital organ whose normal operation is constantly required.

The heart’s blood-pumping cycle, called the cardiac cycle, ensures that blood is distributed throughout the body. The oxygen distribution process begins when oxygen-free blood enters into the heart through the right atrium, goes into the right ventricle, enters the lungs for oxygen refill and release of carbon dioxide, and transfers into the left chambers, ready for redistribution. About 5.6 liters of blood circulate the body, and three cardiac cycles are completed per minute.

The performance of the heart could now be easily monitored when any cardiovascular problem or disorder is suspected. For instance, a regularly abnormal heartbeat or beats per minute are characteristic of a heart-related illness. This is because a heartbeat is a manifestation of the heart's oxygen-reloading process that is made up of two phases.
The systole is a short period that occurs when the tricuspid and mitral valves close; the diastole is a relatively long period when the aortic and pulmonary valves close. The systole-

diastole relationship is the reference in measuring blood pressure. Other ways of physically determining the heart's regular functioning are examining the pulse rate (beats per minute). An adult's normal heart rate is at 72 beats per minute, while children normally yield higher heart rates. The main functionalities of the heart are provided by the structure and characteristics of the cardiac muscle to be described next.
Function of the heart is shown in **Figure 4. [4]**

1.3.1) Cardiac Muscle

The main function of the cardiac muscle can be summarized as follows. An action potential that causes the heart muscle cells to contract reduces the atrial and ventricular volume, respectively. This results in an increase in pressure, leading to an outflow through the valve when the pressure before the valve exceeds the pressure behind the valve. This process provides the pressure changes to open and close the valves and therefore perform the pumping role of the heart. The contraction part of the heart is called the systole. Each systolic period is followed by a rest period, which is called the diastole.
An important fact about the function of the cardiac muscle is that the depolarization period is a function of the frequency in which the cardiac muscle receives the initiation pulses. During this time and the subsequent repolarization period, theoretically, no new depolarization can be initiated. However, both the depolarization and the repolarization period are subject to shortening if the demand is there. The higher the repetition rate becomes, a shorter depolarization period is achieved. This is another fact differentiating the cardiac muscle from skeletal muscle. This characteristic of cardiac muscle allows the heart to adapt the state of physical exercise.
The remarkable difference with skeletal muscle in organizational structure is the presence of a wire mesh of excitable cells: nodal tissue, running through the cardiac muscle tissue. This network of excitable cells is in charge of control and adjustment of the heartbeat and is further described in the next section.

1.3.2) Cardiac Excitation Process

The nodal cells are located in the heart. They initiate, synchronize, and regulate the heart contractions by generating periodic action potentials.
The nodal cells in the heart do not possess a rest potential but have a constant ion leak. The persistent ion leak eventually causes these nodal cells to depolarize spontaneously at regular intervals when the cell membrane potential exceeds a certain threshold. The nodal cells will also produce a repolarization after each depolarization.
All the nodal cells are linked together electrically, such that any nodal cell can start the depolarization process. There is however an order of depolarization, i.e., some cells depolarize faster than others, giving the fastest cell always the upper hand. If, however, the faster cells are damaged, the slower cells will take the lead.

These nodal cells are organized in two general groups: the sinoatrial node (S-A node) and atrioventricular node (A-V node). These two nodes are called natural pacemakers. The S-A node, which is the main pacemaker of the heart, stimulates the atria and the A-V node in turn and, after a delay, stimulates the ventricles. The electric delay between the activation of the S-A and A-V nodes is created by the propagation of electric impulses via the bundle of tissues between the two nodes.

The S-A node is located in the wall of the hollow vein, on the upper side on the border with the right atrium. This is the primary nodal tissue, i.e., pacemaker, producing impulses in a regular fashion to excite the atrium first, before trickling down to the ventricle

through the A-V node. The S-A node is tunable by hormonal and parasympathetic nerve impulses.

The A-V node is positioned in the part of the septum close to the base of the heart, i.e., the border between the atria and the ventricles, just above a section called the annulus fibrosus. The depolarization rate of the A-V node is generally slower than that of the S-A node. Location of natural pacemakers and conduction system of the heart shown in **Figure 5.**

The cardiac muscle is different from most other cells; in the fact, that the cells themselves pass the depolarization signal on to only certain neighboring cells. All cardiac muscle cells in turn are connected to each other by a conducting cell wall section, the intercalated disk. The intercalated disk conduction is illustrated in **Figure 6.**
The intercalated disk transmits the depolarization wave to the adjacent cell, and this cell will respond as long as it is not in the repolarization phase. The fact that the cells themselves pass the depolarization signal on to only certain neighboring cell is of crucial importance for the understanding of how an ECG is formed at the electrodes.

If all cells are successfully excited at the apex of the heart, the migration of electric excitation wave front through the intercalated disks will provide a smooth depolarization. This depolarization front moves homogeneously upward toward the base of the heart, and as a result, the blood is effectively forced out off the ventricle with maximum efficiency. This description of the heart activity assumes a healthy normal working heart. Detectable deviations in the depolarization wave front will elicit any abnormalities in the ejection sequence and are therefore an integral part of diagnosing the proper functioning of the pump action of the heart.

2) ELECTROCARDIOGRAM: SIGNAL OF CARDIOVASCULAR SYSTEM

The depolarization and repolarization process during each heart cycle generates local electric potential differences, which can be measured on the skin using electronic recording equipment. This group of signals, called ECG, constitutes the most informative clinical signal commonly used in the diagnosis of the cardiovascular system.

2.1) Origin of ECG

The ECG represents the repetitive electric depolarization and repolarization pattern of the heart muscle. Typical healthy ECG signal is shown in **Figure 7.**
The ECG is characterized by five peaks, represented by the letters P, Q, R, S, T, and sometimes followed by a sixth peak, the U wave. The P wave is the result of the depolarization of the atrium, while the remaining waves are caused by the ventricle.
The electrocardiography made its introduction through the pioneering efforts of the Dutch scientist Willem Einthoven in 1903. He used a galvanometer to design a way to record the action potentials. He also introduced the markers P, Q, R, S, and T on the standard ECG. **[5]**

The P wave: Contraction of the atria is triggered by the SA-node impulse. The atria do not possess any specialized conduction system as the ventricles do; as such, contraction of the atrial muscles takes place in a slow squeezing manner, with the excitation stimulus being propagated by the muscle cells themselves. For this reason, the P wave is a slow waveform, with a duration of about 80 ms. The P wave amplitude is much smaller (about $0.1 - 0.2$ mV) than that of the QRS because the atria are smaller than the ventricles. The P wave is the epoch

related to the event of atrial contraction. (Atrial relaxation does not produce any distinct waveform in the ECG as it is overshadowed by the following QRS wave.)

The PQ segment: The AV node provides a delay to facilitate completion of atrial contraction and transfer of blood to the ventricles before ventricular contraction is initiated. The resulting PQ segment, of about 80 ms duration, is thus a “nonevent”; however, it is important in recognizing the baseline as the interval is usually isoelectric.

The QRS wave: The specialized systemof Purkinje fibers stimulate contraction of ventricular muscles in a rapid sequence from the apex upwards. The almostsimultaneous contraction of the entire ventricular musculature results in a sharp and tall QRS complex of about 1 mV amplitude and 80 − 100 ms duration. The event of ventricular contraction is represented by the QRS epoch.

The ST segment: The normally flat (isoelectric) ST segment is related to the plateau in the action potential of the left ventricularmuscle cells. The duration of the plateau in the action potential is about 200 ms; the ST segment duration is usually about 100 − 120 ms. As in the case of the PQ segment, the ST segment may also be called a nonevent. However, myocardial ischemia or infarction could change the action potentials of a portion of the left ventricular musculature and cause the ST segment to be depressedn or elevated. The PQ segment serves as a useful reference when the isoelectric nature of the ST segment needs to be verified.

The T wave: The T wave appears in a normal ECG signal as a discrete wave separated from the QRS by an isoelectric ST segment. However, it relates to the last phase of the action potential of ventricular muscle cells, when the potential returns from the plateau of the depolarized state to the resting potential through the process of repolarization. The T wave is commonly referred to as the wave corresponding to ventricular relaxation. While this is correct, it should be noted that relaxation through repolarization is simply the final phase of contraction: Contraction and relaxation are indicated by the upstroke and downstroke of the same action potential. For this reason, the T wave may be said to relate to a nonspecific event.
The T wave is elusive, being low in amplitude (0.1 − 0.3 mV) and being a slow wave extending over 120 − 160 ms. It is almost absent in many ECG recordings. Rather than attempt to detect the often obscure T wave, one may extract a segment of the ECG 80− 360 ms from the beginning of the QRS and use it to represent the ST segment and the T wave. **[6]**

Because of the electric nature of the heart muscle, the depolarization and the repolarization are separated by a relatively long time period. As a result, in the electric recording of the heart activity, the expected triphasic action potential phenomenon will never be seen.

2.2) ECG Electrode Placement

The ECG is not measured directly on the heart itself but on the exterior of the body. Various internationally accepted electrode placements are in existence, which are described next.
The oldest standard of ECG measurement is a three-point extremities recording on the arms and one leg, which is essentially the same as the original Einthoven electrode placement. The recording uses three bipolar measurements that are as follows: recording I is between left (L) and right (R) arm, i.e., VL − VR; recording II is between left leg (F, for foot) and right arm, i.e., VF − VR; and recording III is between left leg and left arm, i.e., VF − VL. This standard ECG configuration for the electrode placement is illustrated in **Figure 8.**
Another popular electrode placement arrangement is outlined in **Figure 9.**
A scrolling curve on the chest outlines the electrode placement starting from the right, with one electrode on either side of the sternum and three more electrodes trailing along the left side of the sternum at the fifth rib with the sixth electrode underneath the armpit on the sixth rib. This electrode placement using six electrodes is called V1–V6. These six electrodes are added to the ones at the three extremities. This ECG standard is called the Wilson placement. As can be seen, in this standard, the electrode placements follow the outline of the heart from top to bottom.
More accurate measurements can be made with the help of coronary electrode placement and ventricular electrode placement through insertion of a catheter equipped with electrodes. Most catheters have two or three electrodes on a single catheter, and several catheters can be inserted simultaneously.

2.3) Modeling and Representation of ECG

Einthoven introduced the concept that electric activity of the heart can be described by a single current dipole with a resulting three-dimensional (3-D) rotating field vector, called the heart vector. The concept of the heart vector is illustrated in **Figure 8**, where all the recordings at a 120° angle difference with each other are combined to give the geometrical projection in one resultant direction. The heart vector represents the magnitude and the direction of the dipole moment caused by the current dipole, which is the cumulative polarization and repolarization ion flow across the cardiac cells.
The main postulate in the forming and analysis of the heart vectors is that the electric activity of the heart at any time can be described by the heart vector. In order to provide a geometry that simplifies the calculations of the heart vector, the human body is considered as a sphere with the source of the heart vector located at the center. In this geometry, even though the two arms and the left leg all protrude from the sphere, they are assumed to be in one plane and at equal distances from the center. In forming the heart vectors, it is also assumed that the electric conduction is homogeneous and isotropic over the sphere.

2.4) Periodicity of ECG: Heart Rate

The leading ECG feature deciphering the hemodynamic phenomena is the frequency of relaxation and contraction of the heart muscle, i.e., the pulse or heart rate. A complete heart cycle is started by the atrial contraction, associated with the pulse represented by the P wave. After the atrial contraction, the ventricular contraction occurs, which is preceded by the QRS complex marking the systole. The cycle ends with rest in which both atria and ventricles are in relaxed state or diastole. This rest state then leads to atrial contraction and

repeat of the cycle. The repetition of this entire cycle makes ECG and all other heart signals periodic.
In an average person, the heart rate is approximately 75 beats per minute, which yields a period of 0.8 s on the ECG. The various stages of the contraction are spread out over this period in the following sequence. The atrial contraction in this case lasts approximately 0.1 s, followed by a ventricular contraction that lasts 0.3 s, with an end pause of 0.4 s. This means the atrial diastole lasts 0.7 s in this example, and the ventricular diastole lasts 0.5 s.

The heart rate is sensitive to internal and external stimuli that can increase or decrease the heart rate. Two different types of mechanisms that can affect the heart rate can be distinguished: intrinsic and extrinsic. The intrinsic mechanisms are due to the changes (e.g., stretching) in the S-A node, which directly alters the heart rate. Another intrinsic effect is temperature, which can affect the heart rate both in an upward and downward direction depending on raised or lowered temperature, respectively. The extrinsic regulatory mechanism includes both parasympathetic and sympathetic nervous systems. These autonomic nerve systems affect the release of acetylcholine or noradrenaline–adrenaline that changes the heart rate. The parasympathetic and orthosympathetic nervous system affects the heart rate through the nervi vagi. The final and most well-known mechanism in heart rate control is the hormone adrenaline released by the adrenal glands, which increases the heart rate for the fight-or-flight reaction.

Various deviations in the typical heart rhythm are often caused by either impulse generation malfunctioning or conduction distortion. The activation in the atria may be fully irregular and chaotic, producing irregular fluctuations in the baseline. As a consequence, the ventricular rate becomes rapid and irregular, even though the QRS contour may still look normal. This electric phenomenon is referred to as atrial fibrillation (AF). When the electric disturbance is confined to the ventricles, the resulting disease is referred to as ventricular arrhythmias.

3) CARDIOVASCULAR DISEASES AND ECG

These deviations from the normal functionality of the cardiovascular system are associated with certain pathological conditions, which can be either genetic or due to malfunctions such as infections, lack of oxygen, and obstruction of blood vessels that supply blood to the heart itself. In this section, some of the main cardiovascular diseases are briefly introduced and the changes in ECG and related diseases are discussed. This brief discussion of cardiovascular diseases will allow us to devise computational methods for the processing of ECG that can detect abnormalities in their early stages.

3.1) Atrial Fibrillation

AF is one of the most common arrhythmias that occur as a result of rheumatic disease, infections (such as pericarditis), atherosclerotic disease, or hyperthyroidism.
These conditions caused by AF are not as life threatening as some of the ventricular arrhythmias but provide an increased risk for stroke. Physical symptoms include palpitations, dizziness, and shortness of breath. Some people having AF never notice any sign of discomfort.
AF has a very rapid and chaotic ECG. AF results in rhythms of 150–220 beats per minute. The most prominent feature of the ECG of AF is an abnormal RR interval, while the ventricular rates are generally faster than of a healthy heart.

AF is also characterized by the lack of P wave in the ECG, or the P wave is very small and does not precede the relatively regular-looking QRS complex. **Figure 10** shows an example of a typical AF ECG that was captured using the Wilson placement combined with augmented Einthoven electrode recording, giving a total of nine recordings. One of the standard chart recordings are given by the augments Einthoven recordings aVR, aVL, and aVF, combined with the Wilson recording V1 through V6.

3.2) Ventricular Arrhythmias

In ventricular arrhythmias, ventricular activation does not originate in the A-V node. Moreover, the arrhythmia does not proceed in the ventricles in a normal way. In a normal heart in which the activation proceeds to the ventricles along the conduction system, the inner walls of the ventricles are activated almost simultaneously, and the activation front proceeds mainly radially toward the outer walls. As a result, the QRS complex of a healthy heart is of relatively short duration. In ventricular arrhythmias, however, since either the ventricular conduction system is broken or the ventricular activation starts far from the A-V node, it takes a longer time for the activation front to proceed throughout the ventricular muscle. This results to longer QRS complex. The criterion for normal ventricular activation requires the QRS interval to be shorter than 0.1 s. A QRS interval lasting longer than 0.1 s indicates abnormal ventricular activation. One example of ventricular arrhythmia is illustrated in **Figure 11.**
Another characteristic of ventricular disturbance is the premature ventricular contraction. A premature ventricular contraction is one that occurs abnormally early. If the origin of the disturbance is in the ventricular muscle, the QRS complex has a very abnormal form and lasts longer than 0.1 s. Usually the P wave is not associated with it. The arrhythmogenic complex produced by this supraventricular arrhythmia lasts less than 0.1 s.

3.3) Ventricular Tachycardia

A rhythm of ventricular origin may be a consequence of a slower conduction in ischemic ventricular muscle that leads to circular activation (reentry). This results in the activation of ventricular muscles at a high rate (over 120 beats per minute), causing rapid and wide QRS complexes. Such an arrhythmia is called ventricular tachycardia (VT). VT is often a consequence of ischemia and myocardial infarction. The main change in ECG that indicates the occurrence of VT is the very fast heart rate that can be easily detected in the Fourier domain using discrete Fourier transform, or DFT.

3.4) Ventricular Fibrillation

When ventricular depolarization occurs chaotically, the situation is called ventricular fibrillation. This is reflected in the ECG, which demonstrates coarse irregular undulations without QRS complex. The cause of fibrillation is the establishment of multiple reentry loops usually involving diseased heart muscle. In this type of arrhythmia, the contraction of the ventricular muscle is also irregular, and, therefore, the timing is ineffective at pumping blood. The lack of blood circulation leads to almost immediate loss of consciousness and even death within minutes. The ventricular fibrillation may be stopped with an external defibrillator pulse and appropriate medication.

3.5) Myocardial Infarction

If a coronary artery is occluded, the transport of oxygen to the cardiac muscle is decreased, causing an oxygen debt in the muscle, which is called ischemia. Ischemia causes changes in the resting potential and in the repolarization of the muscle cells. This abnormality is observed in ECG as changes in the shape of the T wave. If the oxygen transport is terminated in a certain area, the heart muscle dies in that region. This is called a myocardial infarction or heart attack. After a blockage in the blood vessels supplying the heart muscle with oxygen and nutrients, the muscle cells in the region are severely compromised. Some cells may die while others will suffer severe damage, all resulting in a decreased ability to conduct impulses by generating its own depolarization. The dead cells will eventually be replaced by collagen since the heart does not have the ability to regenerate.
An infarct area is electrically silent since it has lost its excitability. The loss of this outward dipole is equivalent to an electric force pointing inward. With this principle, it is possible to locate the infarction. The compromised cells will generate an action potential in a much slower fashion, causing a localized delay in the depolarization wave front. If this delay is enough to emerge at the time that healthy cells have already been repolarized, a subsequent delayed depolarization wave front may pass through the region of the heart that had just contracted. This generates a chaotic electric pattern and a disorganized contraction agreement.
Figure 12 shows nine sections of a recording in combined Einthoven and Wilson electrode placement of an inferior myocardial infarction. A heart attack can result in various deviating ECG patterns. In many heart attack cases, due to the existence of the dying cells in the heart muscle, there will be no significant dip between the QRS complex and the T wave. The period between the S part and the T wave will also seem continuous. This is referred to as ST elevation**. Figure 13** illustrates the effect of dying cells on the ST potential. The ST elevation is one of the most recognizable indicators of myocardial infarction.

3.6) Atrial Flutter

When the heart rate is sufficiently elevated so that the isoelectric interval between the end of T and beginning of P disappears, the arrhythmia is called atrial flutter.
The origin is also believed to involve a reentrant atrial pathway. The frequency of these fluctuations is between 220 and 300 beats per minute. The A-V node and thereafter the ventricles are generally activated every second or every third atrial impulse (2:1 or 3:1 heart block).

3.7) Cardiac Reentry

Under certain conditions, the electric depolarization can conduct back into the atrium from where it immediately conducts over the His bundle back into the ventricles. Another frequent form of reentry is a short circuit in the Purkinje fibers at the end of His bundle. The signal traveling through the His bundle does not conduct through one of the branches in one direction but will allow conduction in the opposite direction, providing a secondary activation of the ventricles through the healthy His branch. Cardiac reentry is one of the main causes of ventricular arrhythmias.

3.8) Atrioventricular Block

As mentioned earlier, the A-V node is slower than the S-A node, and, as a result of various illnesses, the conduction from S-A node to A-V node can be interrupted. This is called AV block. Under these circumstances, the atria will contract faster than the ventricles and the pump function will be severely compromised. As discussed earlier, if the P wave precedes the QRS complex with a PR interval of 0.12 s, the AV conduction is normal. If the PR interval is fixed but shorter than normal, either the origin of the impulse is closer to the ventricles or the AV conduction is utilizing an (abnormal) bypass tract leading to preexcitation of the ventricles. The latter is called the Wolff–Parkinson–White (WPW) syndrome and is discussed later. The PR interval may also be variable, such as in a wandering atrial pacemaker and multifocal atrial tachycardia. An example of the ECG recorded during a third-degree AV block is shown in **Figure 14.**
Based on the specific condition of the block, different types of AV blocks are defined. The conduction system defects producing a third-degree AV block may arise at different locations.

3.9) Wolf–Parkinson–White Syndrome

Technically, the ventricles and atria are supposed to be electrically isolated from each other. However, sometimes there may be a small amount of conduction passing directly from the atrium to the ventricle. In this situation, the A-V node is effectively circumvented, and the delay introduced by the A-V node and His bundle combination is no longer able to hold the ventricular contraction off until the ventricle is filled by the atria. The QRS will follow directly on the down slope of the P wave. Meanwhile, the A-V-node stimulus will still proceed and depolarize in sequence with the “shortened” atrium–ventricle passage. This will lengthen the QRS complex, making it terminate at the originally expected time period as during a normal ECG recording.
One cause for a broad QRS complex that exceeds the 0.12 s duration may be the WPW syndrome. The cause of the WPW syndrome is the passage of activation from the atrium directly to the ventricular muscle via an abnormal route. This bypass is called the bundle of Kent, which bypasses the AV junctions. This results in an activation of the ventricular muscle before normal activation reaches the ventricular muscle via the conduction system. This process is called preexcitation, and the specific ECG depends on the respective location of the accessory pathway. In WPW syndrome, the time interval from the P wave to the R wave is normal. The QRS complex in this case has an earlierthan-normal sharp onset called the delta wave. The premature ventricular excitation manifesting the delta wave results in a shortening of the PQ time. An illustration of the impact on the conduction pattern during WPW syndrome given in **Figure 15.**

3.10) Extrasystole

Both chemical and mechanical stimuli can trigger spontaneous depolarization of a single cardiac cell, which translates to the conduction of activation to the neighboring cells. The only difference between this type of disease and normal heart is the resulting excitation that can be caused by any cell located at any location in the heart muscle. This random pulse generation is called an ectopic stimulus. In this situation, the P wave is often missing in its entirety for the QRS complex.

4) PROCESSING AND FEATURE EXTRACTION OF ECG

The interpretation of the recorded ECG is often based on a physician's experience. The physician's decision is typically based on the previously mentioned "time-based criteria" described for the ECG of the diseased cases. Such criteria are often rooted in the physiology of the cardiovascular system and are very well understood by the physicians.
While the computer-aided diagnostic systems commonly used in many clinical electrocardiography clinics utilize all the time-based criteria mentioned earlier, they also use the typical signal processing and pattern recognition measures and techniques for diagnostics.
It has been reported that the computer-aided interpretation of characteristic patterns found in ECG provides better than 90% accuracy of recognizing the underlying electric and hemodynamic causes and factors for appropriate treatment. For majority of the diagnostic interpretations, it is either the measurement of time intervals (i.e., time-based criteria) that provides an insight in the underlying causes, or the altered shapes of the actual waveform that reveal the underlying causes of the hemodynamic complications. The purely signal processing measures and methods capture small changes in ECG that may go unnoticed by the human eyes. It is also important to note that even for measuring time-based criteria, the computer-aided systems can provide a more reliable and accurate measurement and description of the time-based measures such as the duration of the heart cycle. More specifically, the signal processing techniques can more accurately calculate the time intervals and analyze the resulting numbers.

4.1) Time-Domain Analysis

The most significant time-domain feature in the ECG is the duration of the heart cycle. The heart cycle duration is frequently derived by measuring the time span from one R wave to the next. Other features are essentially the duration of each wave (i.e., the duration of QRS complex) and the time separation among the waves (e.g., the time interval between T and P waves, i.e., TP interval).
Two specific time features are further described in the following. An important time domain feature is the duration of QRS complex as described earlier. Generally, the QRS complex is identified by the characteristic shape and relative stable time constant in the pattern. Another feature of interest is the time interval between the T wave and the subsequent P wave. The importance of this measure shows the separation between two important events, i.e., the pulse rate of the sinus node, which is expressed in the P wave, and the repolarization phenomenon that is the origin of the T wave.

4.2) Frequency-Domain Analysis

In any ECG waveform, the QRS complex is well localized as the high-frequency region. The P and T waves are mainly the low-frequency components. The ST segment in the ECG is time restricted with mostly low-frequency content.
The normal ECG and the deviating ECG often have significantly different frequency contents. Since the normal heart rate is in the range of 60–100 beats per minute, the fibrillation can exceed 200 beats per minute. In addition to the frequency differences, the depolarization and repolarization ramps also change under diseased conditions, requiring a much wider frequency bandwidth to describe each different phenomenon.

The standard ECG can be described by the first eight harmonics of the heart rate in the Fourier domain. This provides a basic representation as shown in **Figure 16**. **Figure 16a** shows the nine-electrode recording for a normal ECG as shown in **Figure 7** illustrates the superposition of the first eight harmonics reconstructing the ECG from **Figure 3**. Any minor high-frequency deviation from the normal ECG often creates variations in the ECG that requires a much larger number of harmonics to describe the frequency-domain features of the ECG. As a rule of thumb, a frequency analysis should span no less than the frequency range of 0–100 Hz for an apparently normal ECG. Arrhythmias may require a frequency analysis up to 200 Hz. However, entering even higher-frequency spectra, the spectrum will be dominated by noise and will not contribute additional information.

4.3) Wavelet-Domain Analysis

Since action potentials are mainly stochastic in nature, wavelet analysis of a single action potential may not provide the reliable data required for accurate diagnosis. However, when observing a repetitive signal (such as ECG) as a resultant of the summation of many action potentials, the wavelet-domain features can identify the relative contributions of the higher frequencies (lower scales).
The wavelet features used in the analysis of ECG often detect the existence of a scaled or shifted version of a typical pattern or wave. Wavelet decomposition using mother wavelet resembles the general shape of the QRS complex, which reveals the location, the amplitude, and the scaling of the QRS pattern quantitatively. Wavelet analysis is also performed using Daubeches and Coiflet wavelets.
A typical application of the wavelet analysis is the separation of the mother's and the baby's ECG. As mentioned earlier, the waveform of the fetal ECG is similar to that of the adult ECG in the wavelet transform (WT) domain, except for the scale of the signal. The wavelet decomposition of the observed signal can effectively separate the mother's ECG from the baby's, simply because the two ECG signals reside on different scales. **[7]**

5) DETECTION OF EVENT

5.1) Derivative based methods for QRS detection

Problem: Develop signal processing techniques to facilitate detection of the QRS complex, given that it is the sharpest wave in an ECG cycle.

Solution 1: The QRS complex is the largest slope (rate of change of voltage) in a cardiac cycle by virtue of the rapid conduction and depolarization characteristics of the ventricles. As the rate of change is given by the derivative operator, the d/dt operation would be the most logical starting point in an attempt to develop an algorithm to detect the QRS complex.

The derivative operator enhances the QRS, although the resulting wave does not bear any resemblance to a typical QRS complex.

The smoothed three-point first derivative y0(n) of the given signal
x(n) is approximated as
$y0(n) = |x(n) - x(n - 2)|$.

The second derivative is approximated as
$y1(n) = |x(n) - 2x(n - 2) + x(n - 4)|$.

The two results are weighted and combined to obtain
$y2(n) = 1.3y0(n) + 1.1y1(n)$.

The result y2(n) is scanned with a threshold of 1.0. Whenever the threshold is crossed, the subsequent eight samples are also tested against the same threshold. If at least six of the eight points pass the threshold test, the segment of eight samples is taken to be a part of a QRS complex. The procedure results in a pulse with its width proportional to that of the QRS complex; however, the method is sensitive to noise.
Illustration of application: **Figure 17** illustrates, in the topmost trace, two cycles of a filtered version of the ECG signal. The signal was filtered with an eighth-order Butterworth lowpass filter with fc = 90 Hz, down-sampled by a factor of five, and filtered with a notch filter with fo = 60 Hz. The effective sampling rate is 200 Hz. The signal was normalized by dividing by its maximum value.

The second and third plots in Figure 17, show the derivatives y0(n) and y1(n), respectively; the fourth plot illustrates the combined result y2(n). Observe the relatively high values in the derivative-based results at the QRS locations; the outputs are low or negligible at the P and T wave locations, in spite of the fact that the original signal possesses an unusually sharp and tall T wave. It is also seen that the results have multiple peaks over the duration of the QRS wave, due to the fact that the QRS complex includes three major swings: Q–R, R–S, and S–ST baseline in the present example (an additional PQ baseline–Q swing may also be present in other ECG signals).

The last plot in Figure 17, shows the smoothed result y3(n) obtained by passing y2(n) through the 8-point MA filter. We now have a single pulse with amplitude greater than 1.0 over the duration of the corresponding QRS complex.

A simple peak-searching algorithm may be used to detect each ECG beat. The net delay introduced by the filters should be subtracted from the detected peak location in order to obtain the corresponding QRS location.

Note that peak searching cannot be performed directly on an ECG signal: The QRS might not always be the highest wave in a cardiac cycle, and artifacts may easily upset the search procedure. Observe also that the ECG signal in the present illustration was filtered to a restricted bandwidth of 90 Hz before the derivatives were computed and that it is free of baseline drift**. [8]**

CONCLUSION

In this article, the function and structure of the heart as well as the origin of the ECG signal are briefly described. Also, introduces a number of cardiovascular diseases that can be diagnosed using ECG. The processing methods commonly used for processing of ECG and detection of event are also covered in this article.

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