A Review on Arrhythmias in ECG: Detection Using Spectral Methods

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ABSTRACT
Arrhythmia investigation of Electrocardiogram (ECG) signal plays a major role in diagnosing most of the heart diseases. This review paperdescribes about basic components of ECG, classification of arrhythmias according to their source of origin, different type of cardiac arrhythmias, spectra of heart rate variability and discusses a review of existing main techniques namely power spectral and wavelet transform used for the analysis of arrhythmias and severaltechniques used in detection and classification of arrhythmias.

Keywords: classification of arrhythmias, spectral methods

1. INTRODUCTION

Electrocardiogram (ECG) has normally used to gather a lot of estimations that contain specific evidence in the cardiac signals. The ordinary diagnostic technique is off-line investigation from the recorded data, and utilizing a cardiogram to recognize arrhythmic categories of the subjects.The ECG is a diagnosis device that revealed the electrical signal activity of heart repolarization and depolarization recorded by skin electrode(Silverman and Willis Hurst, 1992). The electrical signal activity is due to depolarization and repolarization of Na+ and K+ ions in the blood (Stanfield, 2013). It is a noninvasive system that implies the electrical signal activity is measured on the surface of human body, which is used in detection of the cardiac diseases(Stanfield, 2013) .The ECG complex morphology and heart rate variability reflects the cardiac health of human heart (Rajendra Acharya *et al.*, 2007). Any disorder of heart rate or rhythm, or change in the morphological pattern, is an indication of cardiac arrhythmia, which could be detected by analysis of the recorded ECG signals(Beheshti, Karthikeyan Umapathy and Sridhar Krishnan, 2016). The duration and strength of the P-QRS-T wave of ECG signals contains useful information about the idea of diseaserelated to the heart. The ECG signal provides the following information of a human heart(Moss, 1996): (i)heart location and its relative chamber size (ii) origin of impulse signals and its propagation (iii) morphological pattern, heart rhythm and conduction disturbances (iv)Level and position of myocardial ischemia (v) changes in electrolyte concentrations (vi) Drug-effects on the cardiovascular system.

The basic components of the ECG signal are illustrated in Figure 1. The first impulse, named as P wave, relates to the depolarization of the left and right atrium: the electrical signal spreads from the Sino- atrial (SA) node trench the atrium(Sörnmo and Laguna, 2005)(Dilaveris *et al.*, 2003). The PR (or PQ) interim is the time period between the onsets of ventricular and atriumdepolarization. A short duration isoelectric line is extant within the PR interim. This is alluded to as PR segment and reaches out from the finish of the P wave until the start of the QRS complex. Ventricular depolarization converts into the QRS complex(Durrer, 1968). It ought to be noticed that atrial repolarization happens amid ventricular depolarization and is covered up in the QRS complex.

The ventricular depolarization an isoelectric line is perceptible in the ECG record. This is called ST segment and stretches out from the finish of the QRS complex to the start of the T wave. It relates to an electrically neutral time for the heart, between ventricular depolarization and repolarization. Depression or elevation of the ST segment might be indicative of myocardial damage(Hurst, 1997). The T wave always follows the QRS complex because it represents ventricular repolarization. The ordinary T wave ought to be a similar way of the QRS complex and is marginally lopsided. The time between the end of ventricular repolarization and onset of ventricular depolarization is named as QT interval(Merritt and Tan, 2012). It is along these lines estimated from the earliest starting point of the Q wave to the finish of the T wave. The length of this interval changes as per age, sex, and for the most part heart rate variability.

Table 1. ECG complex features and their amplitude and duration of normal adult subject.

ECG Features	**Strength (mV)**	**Duration (S)**
P –Wave	0.25-0.30	0.08-0.10 S
PR Interval	-	0.12-0.21 (frequency dependent)
PR Segments	-	0.04-0.10 (frequency dependent)
QRS Complex	0.5-1.6 in limb leads up to 2 in chest leads	0.06-0.12 S
ST interval	-	No relevance
ST segments	-	80 to 120ms
QT interval	-	frequency dependent
RR interval	-	0.12-0.22 s
T wave	0.1to 0.3	120 to 160 mS

The U wave, with small amplitude that follows the T wave, signifies late ventricular repolarization. This wave is not always appearing in the ECG recorded data and its nonappearance is not a sign of cardiac disease. U waves are more easily noticeable with slower heart rates (Moss, 1993)(Sinha and Shahnaz, 2012). The value of basic components of ECG complex of normal adult subject is reported in Table 1.

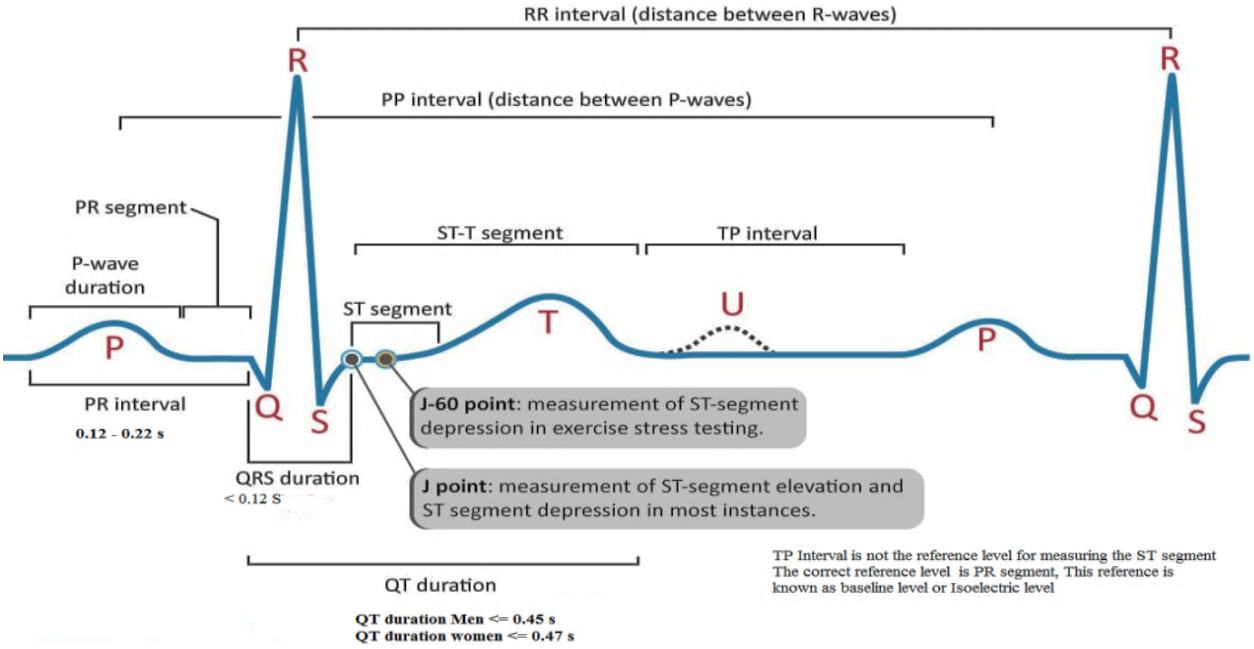

Figure 1: Basic components of the ECG complex of healthy human heart, ecgwaves.com/ecg-normal-P-wave-QRS-complex-st-segment-t-wave-j-point.

2. ARRHYTHMIAS

The term arrhythmia is disordered in the timing of heart beat or P-QRS-T pattern of the ECG complex.Arrhythmias are distinctin any cardiac rhythm except the normal sinus rhythm (NSR).The normal rhythm of the heart where there is no disease or disorder in the P-QRS-T pattern of ECG signal is called NSR. The heart rate of NSR is normally characterized by 60 to 100 bpm (beats per minute).But, particularly in kids and under normal conditions, it mightoccur slight to moderate changes relianton the times of breathing, the heart rate increasing with inspiration and vice versa(Issa and John Miller, 2008) (Mariano Llamedo Soria, 2012).Arrhythmia may be due to abnormalities in impulse formation or disorder in heart's electrical system, or both, but it is not always a disorder in heart rhythm(Tsipouras and Fotiadis, 2004)(Erik and Sigurd, 1991).Impulse formation may be ectopic or sinus, the rhythm regular or irregular and the heart rate faster (> 100bpm i.e. sinus tachycardia or tachycardia), or slow (< 60 bpm i.e. sinus bradycardia)(Goldberger, 2006)(Rakel, 2013). Despite that, it ought to be considered that sinus rhythm differs all through a 24-h period and sinus tachycardia and sinus bradycardia usually are a physiologic response to certain sympathetic (stress, exercise) or vagal (sleep, rest) stimuli (Luz E.J. S. *et al.*, 2016).Under such disorders, the occurrence of these heart rates ought to be considered normal(Mariano Llamedo Soria, 2012).The arrhythmiasaredepending on primary disease conditions related to heart and cruelty, some arrhythmias such as atrial fibrillation, heart block, ventricular atrial fibrillation and supraventricular tachycardia (SVT). As a result of arrhythmia, the heart may beat ineffectually, and the body may receive an insufficient blood supply.This may cause life-threatening symptoms(Khadra, Al-Fahoum and Binajjaj, 2005)(Jacobs and Oxer, 1990). The classification of cardiac arrhythmias depends onsource of the originating rhythm, which has been further explained in section 3.

3. CLASSIFICATION OF ARRHYTHMIAS

The four arrhythmogenic zones are shown in Figure 2, these zones used to classify arrhythmias according to their source of origin(TOMAS B. GARCIA and Geoffrey T. Miller, 2004)(Jane Huff, 2011)(Burghardt, 2005). The P-QRS-T pattern initiating in the atrium or atrio-ventricular (AV) junction, sino-atrial (SA)node, can be more generally known as supraventricular rhythms. The main types of arrhythmic events arebrief below using this classification and their characteristics are reviewed in the sections 4.1 to 4.6.

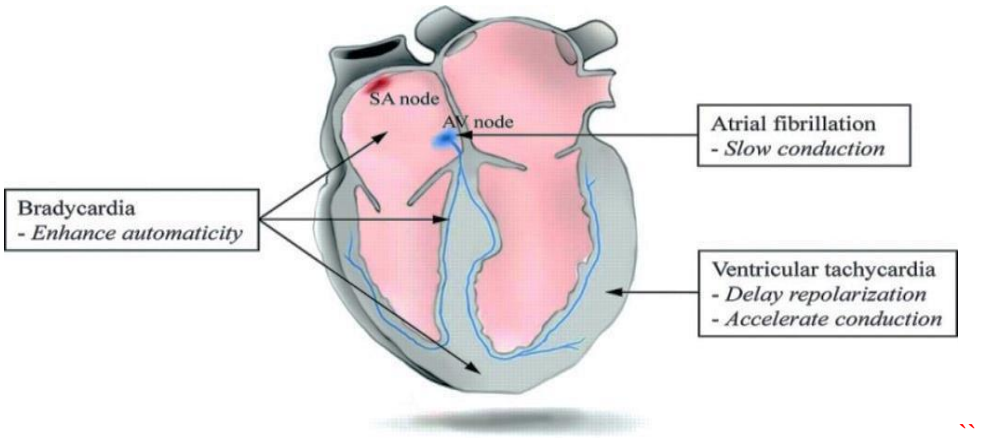

Figure 2: Four arrhythmogenic zones (Garcia and Miller, 2004).

3.1 *SINUS NODE ARRHYTHMIAS*

This type of arrhythmia originated from the SA node of the heart. As the electrical impulse is generated from the normal pacemaker, the characteristic of these arrhythmias is that P-wave of ECG morphology is normal. These arrhythmias are of the following types.

3.1.1 *SINUS ARRHYTHMIA*

This arrhythmia is not disordering the heart beat, but a normal, heart beat variation in the sinus rate with the phases of breath is shown in Figure 3. It is characterized by HR of 60-100 bpm, atrial and ventricular rhythm, P-wave and upright before QRS complex, length of RR interval lies between 0.12-0.22 Sec. QRS complex length varies between 0.06 -0.12 Sec and look alike.

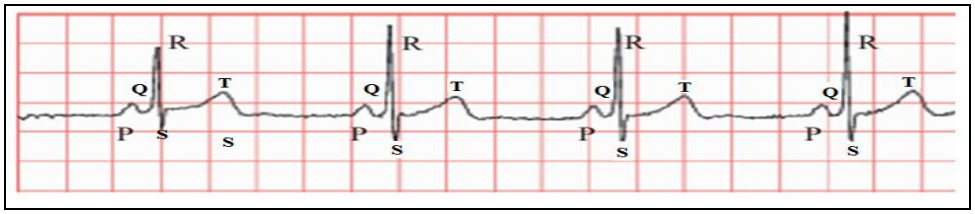

Figure 3: The normal sinus rhythm has regular ECG complex

3.1.2 *SINUS BRADYCARDIA*

In sinus bradycardia, the rhythm initiates from the SA node.It is characterized by heart rate of less than 60 bpm, atrial and ventricular rhythm, P-wave and upright before QRS complex, length of RR interval lies between 0.12-0.22 Sec.

QRS complex length varies between 0.06 -0.12 Sec and like look alike. Example is depicted in Figure 4. The ECG appears normal even slow heart rate. Mild sinus bradycardia (48-59 bpm) is generally asymptomatic, while marked sinus bradycardia (30-43bpm) may lead to hypotension and result in deficient perfusion of the brain and other vital organs.

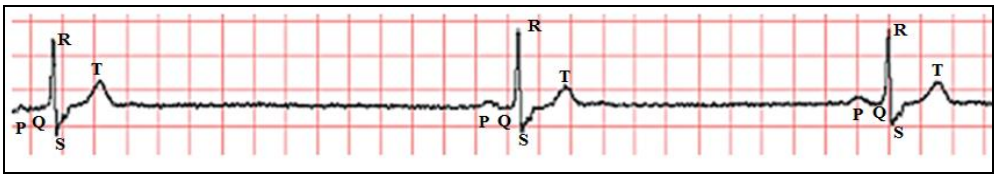

Figure 4: Sinusbradycardia, illustrated heart beats at a slow rate

3.1.3 SINUS TACHYCARDIA

Sinus Tachycardia is created due to a fast firing of the SA nodeand is shown in Figure 5. It is characterized by HR of 101 to 160 bpm, atrial and ventricular rhythm, P-wave and upright before QRS complex, length of RR interval lies between 0.12-0.22 Sec. QRS complex length varies between 0.06 to 0.12 Sec and like look alike.

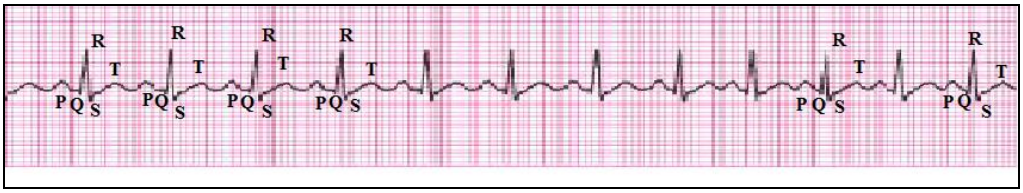

Figure 5: Sinus Tachycardia, the heart beatsis fast compared to NSR.

3.1.4 SINUS ARREST

In this arrest, the SA node spasmodically fails to fire. There is no P-wave and therefore no accompanying QRS-complex and no T-wave, is shown in Figure 6. This results in a pause in the electrical activity realized on theECG complex. Subsequently the automaticity of the SA node is irregular; the longest P-P interval (i.e. the pause or arrest" on the ECG) will not be several of the tiniest P-P intervals.

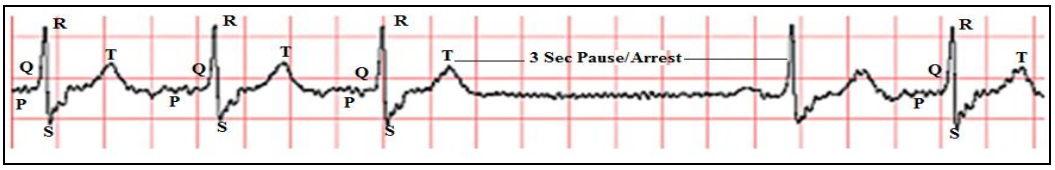

Figure 6: Sinus arrest is identified by a missing beat (P-QRS-T).

3.2 ATRIAL ARRHYTHMIAS

Atrial arrhythmias create outside the SA node but in the interiorof atria in the form of electrical pulses. There are various types of atrial arrhythmias summaries below.

3.2.3 Premature Atrial Contractions (PAC)

This arrhythmia comes about an unusual P-wave morphology took after by a normal QRS complex and a T-wave. This happens on account of an ectopic pacemaker terminating before the SA node. PACs may occur as a couplet

where two PACs are created continuously and is appeared in Figure 7. When three or more consecutive PACs occur, the rhythm is considered to be atrial tachycardia.

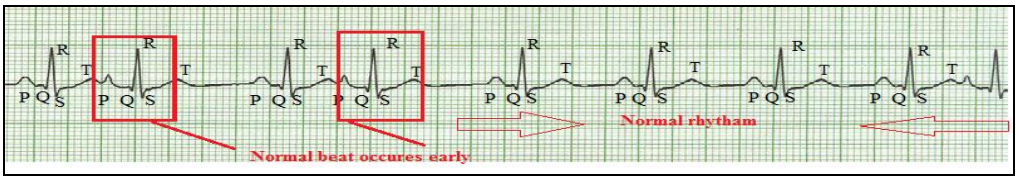

Figure 7: Premature Atrial Contractions

3.2.4 Atrial Tachycardia (Supraventricular Tachycardia)

The heart rate atrial tachycardia is fast and ranges from 150 to 240 bpm in atrial tachycardia. During an episode of supraventricular tachycardia (SVT), the heart's electrical system doesn't work right, causing the heart to beat very fast.

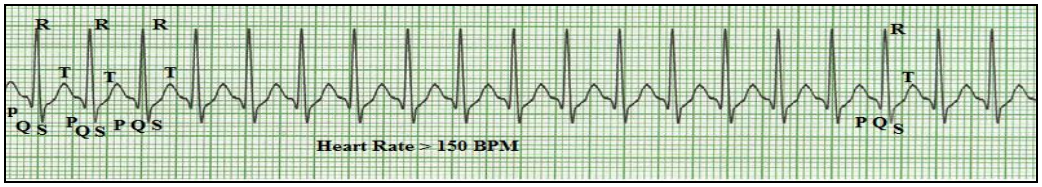

Figure 8: Supraventricular Tachycardia, heart rate ranges between 150 to 240 bpm.

3.2.5 ATRIAL FLUTTER

In atrial flutter (AFL), the atrial rate is very fast (from 240 to 360bpm). The abnormal P-waves occur regularly and so quickly that they take morphology of saw-tooth waveform which is called Flutter (F) waves, and is shown in Figure 9.In AFL, atria of the heart beat too fast, this result in atrial muscle contractions that are faster and out of synchronize with the ventricles.

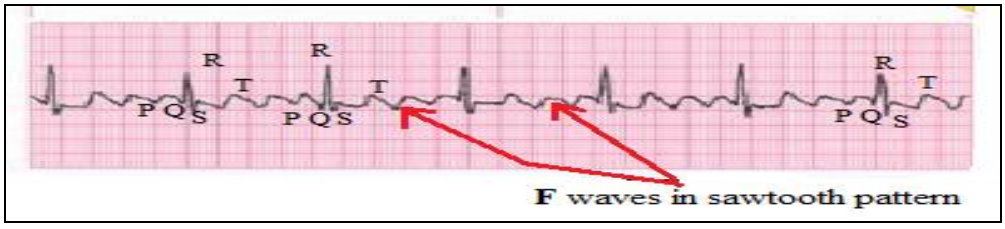

Figure 9: Atrial Flutter is characterized by sawtooth pattern

3.2.4 *ATRIAL FIBRILLATION*(AF)

In this arrhythmia, the atrial rate surpasses 350 bpm. This arrhythmia happens due to clumsy activation and retrenchment of various parts of the atria. . The higher atria rate and clumsy compression prompts incapable pumping of blood into the ventricles. Atrial fibrillation might be discontinuous, happening in paroxysms (short bursts) or incessant, is shown in Figure 10.

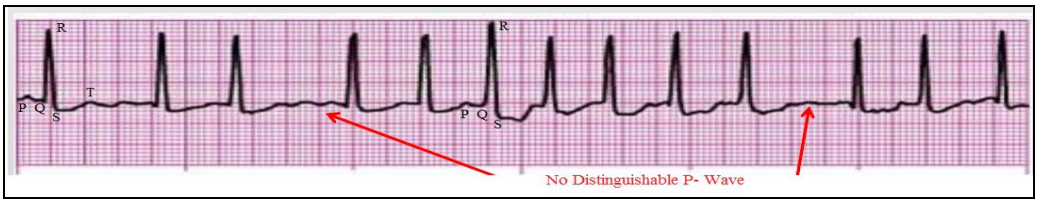

Figure 10: Atrial Fibrillation characterization atrial rate> 350bpm

3.3 JUNCTIONAL ARRHYTHMIAS

Junctional arrhythmias are instigated inside the AV junction as the impulse comprising the AV node and it's Bundle. In these arrhythmias, abnormal in P wave morphology occurs. The direction of the abnormal P-wave would be reverse to that of the normal direction P-wave since depolarization is transmitted in the reverse direction from the AV node to the atria.

3.3.1 PREMATURE JUNCTIONAL CONTRACTIONS (PJC)

It is a ventricular contraction originated by an ectopic pacemaker in the AV node. In premature junctional escape constriction, a typical looking QRS complex prematurely shows up, yet without a previous P-wave, however the morphology of T-wave is normal, and is shown in Figure 11.

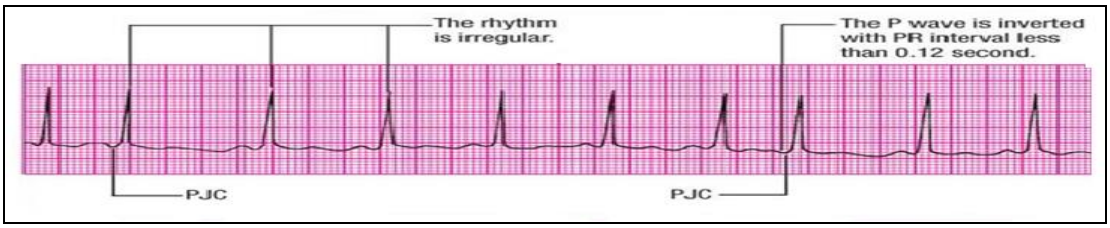

Figure 11: Premature Junctional Contractions in which P-wave is inverted

3.3.2 PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

The PSVT may take place because of a reentry circuit in the AV junction. It is also known AV nodal reentry tachycardia (AVNRT). It might likewise take place because of a reentry circuit including a frill pathway between the atria and a ventricle i.e. is also known as AV reentry tachycardia (AVRT). The beginning and end of PSVT is sudden, and may happen in rehashed episodes (paroxysms) that keep going for quite a long time, hours or days. In AVNRT, P-waves are generally hidden in the QRS complex and henceforth not obvious; while for AVRT the P waves might be perceptible. The heart rate ranges from 160 to 240 bpm. The ECG complex of PSVT is shown in Figure 12.

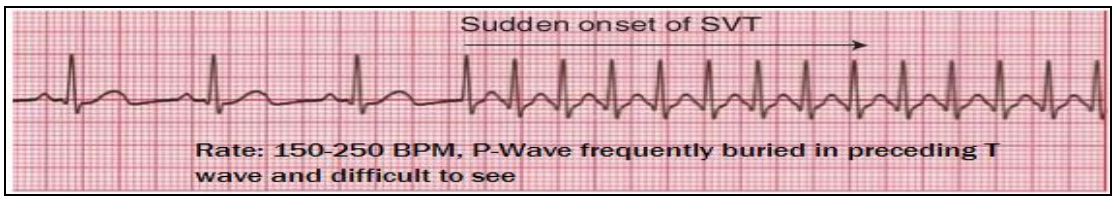

Figure 12: Premature Junctional Contractions, P-wave is difficult to see.

3.4 VENTRICULAR ARRHYTHMIAS

In this kind of arrhythmia, the impulses begin from the ventricles and move outwards to whatever remains of the heart. The QRS shape of this type of arrhythmias has wide and bizarre.

3.4.3 Premature Ventricular Contractions (PVC)

In PVC the heart rate abnormality is initiated from ventricles. The PVCs regularly do not show depolarization of atria or the SA node and thereforethe P-waves morphology keeps up their basic beat and happens at the normal time, is shown in Figure 13.The PVCs may occur any instant duration of heart beat cycle. PVCs are depicted as isolated if they occur independently, and as couplets if two continuous PVCs take place.

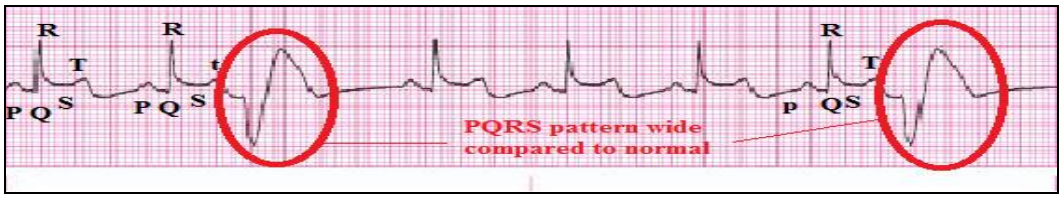

Figure 13: Premature Ventricular Contractions

3.4.4 VENTRICULAR TACHYCARDIA (VT)

In VT the QRS complex of ECG is bizarrely wide, out of the normal in shape, and of an unlike direction from the ordinary QRS complex, is displayed in Figure 14. The VT is considered perilous as the fast heart rate may avoid compelling ventricular filling and result in a drop in cardiovascular yield. The heart rate of ventricular tachycardia is 101 to 250 bpm.

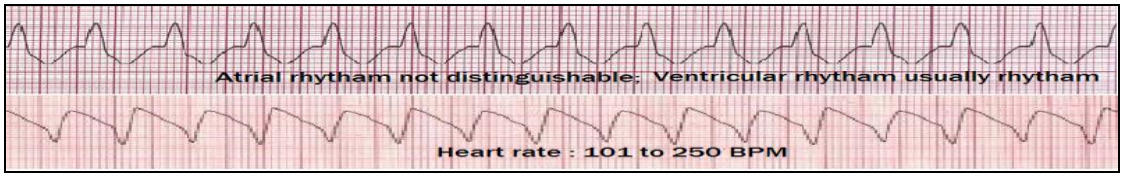

Figure 14: Ventricular Tachycardia

3.4.5 VENTRICULAR FIBRILLATION

The Ventricular fibrillation (VF) occurs when various ectopic pacemakers in the ventricles make different parts of the myocardium contract at not the same time in a non-synchronized fashion. Ventricular express displays an extremely quick ventricular rate with a saw-tooth waveform, is appeared in Figure 15.

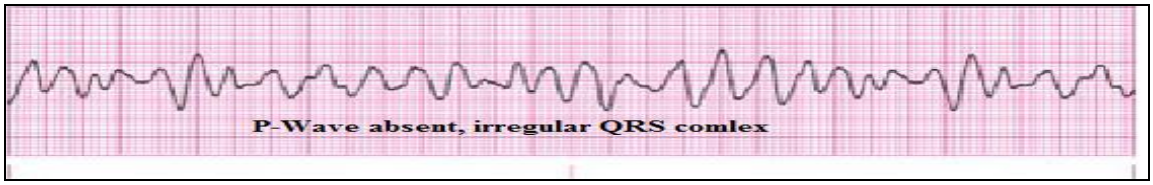

Figure 15: Ventricular Tachycardia

3.5 ATRIOVENTRICULAR (AV) BLOCKS

The AV blocksare characterized by the normal transmission of the electrical signal impulse along the conduction pathways to the ventricles, while the block may delay or totally counteract proliferation of the impulse to whatever is left of the conduction system.

3.5.3 First-Degree Atrioventricular Block

This AV block is befallen when entirely the P-waves are conducted to the ventricles and PR-interval is long, is presented in Figure 16.

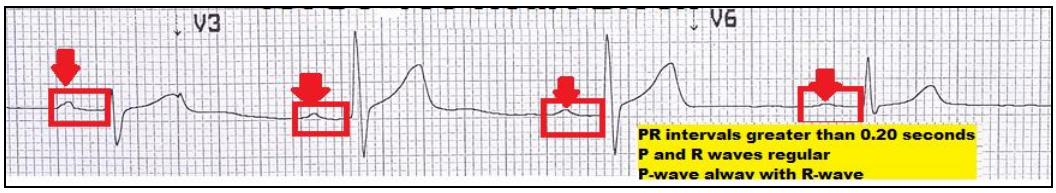

Figure 16: Ventricular Tachycardia

3.5.4 SECOND-DEGREE AV BLOCK

Second-degree AV blocks are occurred when some of the P- waves fail to conduct to the ventricles. The ECG complex of second-degree AV Block is shown in Figure 17. Here, conduction intermittently fails to go down the bundle of His and bundle branches. There is usually a pre-existing complete block in one bundle branch, such that when an intermittent block occurs in the remaining bundle branch, conduction down to the ventricles fails. As a result of the pre-existing bundle branch block, the QRS-complex is commonly wide. Commonly the conduction ratio is 4:3. or 3:2 P-waves to QRS-complexes. This type of AV block is dangerous as complete heart block can occur unpredictably, and is an indication for implantation of an artificial pacemaker.

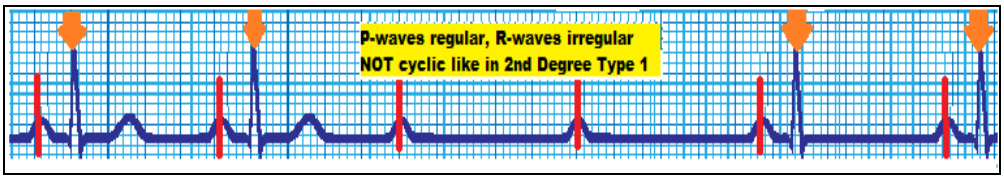

Figure 17: Second-Degree AV Block

3.5.5 THIRD-DEGREE AV BLOCK

In third-degree AV block, the rhythm of the P-waves is totally alienated from the beat of the QRS on ECG complex, and is portrayed in Figure 18. The ventricles and the atria beat at their own rate (Rajendra Acharya *et al.*, 2007).

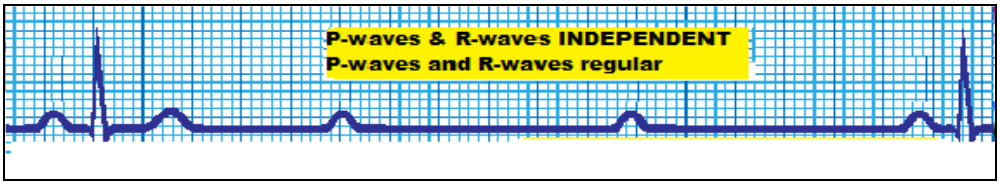

Figure 18: Third-Degree AV Block

3.5.6 PACED BEAT (IMPLANT)

Hearts implanted with an artificial pacemaker will generally beat around 60 to 70bpm depending on the setting of the artificial pacemaker. The pacingelectrode is commonly attached to the apex of the right ventricular cavity (ventricularpacemaker) or the right atrium (atrial pacemaker), or both (dual chamber pacemaker). The artificial pacemaker produces a narrow, often biphasic spike. Apacemaker lead positioned in an atrium produces a pacemaker spike followed by aP -wave. A pacemaker lead positioned in a ventricle produces a pacemaker spikefollowed by a wide, bizarre QRS-complex (Figure 19). Sometimes it is also called pacemaker rhythm.

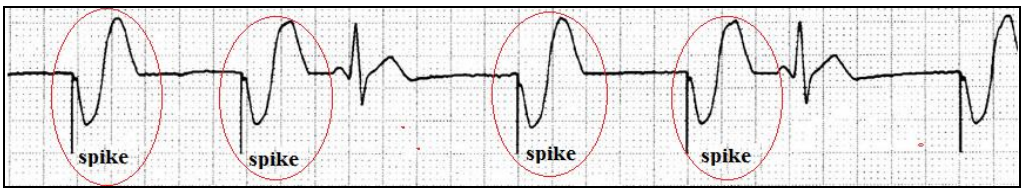

Figure 19: Paced Beat

4. FREQUENCY BAND OF HEART RATE VARIABILITY

The variation of consecutive R-R interval on the QRS templates of ECG records is known as heart rate variability (HRV). The HRV is influenced by multiple neural and hormonal inputs that generate specific observable rhythms in the series(Magagnin *et al.*, 2010).The HRV assist to measure the balance between sympathetic mediators of heart rate that is the effect of epinephrine and norepinephrine hormone released from sympathetic nerve fibers acting on the SA and AV nodes.It increases the rate of cardiac contraction and facilitates conduction at the AV node. The parasympathetic mediators of heart rate that is the influence of acetylcholine released by the parasympathetic nerve fibers acting on the SA and AV nodes leading to decrease in the heart rate and a slowing of conduction at the AV node. Sympathetic mediators appear to exert their influence over longer time periods and are reflected in the low frequency power (PLF) of the HRV spectrum (between 0.04Hz and 0.15 Hz).Vagal mediators exert their influence more quickly on the heart and principally affect the high frequency power (PHF) of the HRV spectrum (between 0.15Hz and 0.4 Hz). Thus at any point in time the PLF: PHFP ratio is a representation for the sympatho-vagal balance. Thus HRV is a noninvasive valuable tool to investigate the sympathetic and parasympathetic function of the autonomic nervous system (ANS)(Eckberg, 1983).

5. METHODS FOR ANALYSIS OF ECG SIGNALS

There are two basic approaches to quantifying the fundamental components of ECG signals: (i) use of global descriptive statistics to portray the distribution of heart periods (e.g. variance, range and standard deviation) and (ii) modeling of periodic shapes to extricate particular frequency components of difference that identify with useful techniques or physiological constituents,instance RSA(BERNTSON *et al.*, 1997). The firstapproach statisticsmostlycontemplate the total population of ECG component as isolated or independent data samples, whereas second approach focuses on the serial linkages among the data components. These two methodologies are not united with the refinement between time-domain and frequency-domain analyses. Time-domain techniques can be used to express periodic processes, and any frequency-domain technique can be transformed into the time-domain technique if the signals are stationary(David R. Brillinger, 2001). But,

the ECG signals are non-stationary time-varying signal from the statistical perspective. Outliers, noise and artifacts affect the sensitivity of the time domain techniques(Acharya *et al.*, 2006). The time domain analysis failed to detect or differentiate the ECG signals, when different rhythmic signals with same means and standard deviations. Hence, time domain analysis is not very useful to analyze the nonlinear and non-stationary signals. Numerous methods have been employed or proposed over the past several decades for analysis of ECG basic components and HRV. However, as the ECG signals have a place with the group of multicomponent non-stationary signals(Wood and Barry, 1996). It is challenge to obtain precise assessments of time-varying spectral signals. This problem can tackle by an appropriate time–frequency distribution method and resolute the multicomponent behavior of ECG signals.Some of the more commonly employed methods in frequency domain and time-frequency domain are summarized in the section 6.1,6.2 and 6.3 for analysis of arrhythmias.

5.1 *SPECTRAL METHODS*

Power spectral investigation of ECG components shows specific frequency variation in the cardiac record which reflects central neural control (CNC) of sinus node activity (SNA)(Fallen *et al.*, 1988)(Akselrod *et al.*, 1981)(Pagani *et al.*, 1986). Spectral approaches produce a disintegration of total disparity of an ECG data time series into its spectral components (Bravi, A. *et al.*, 2011). The ECG signals can be articulated in the form of a power spectral density function which illustrates spectral power as a variation of frequency(MV Kamath, 1993). Power spectral for a given spectrum band can then be measured by deriving the region under the spectral density variation contained by the specified frequency band(Kamath and Fallen, 1993). Kay and Marple(Kay and Marple, 1981) gave a broad outline of a considerable lot of the strategies accessiblefor spectral analysis. The two most common techniques like fast Fourier transform (FFT) technique(Akselrod *et al.*, 1985) and autoregressive (AR) modeling (Pagani *et al.*, 1986)are using for spectralanalysis of ECGcomponents. The fundamental distinction between the FFT and AR approaches is the way by which the data are viewed. In FFT, (i) The analysis expect that the time arrangement contains just deterministic parts. (ii) The spectrum computed is derived from all the data regardless of how well they fit a model based on peaks in the spectral distribution. (iii) The spectrum concentrates on the more significant peaks including with noise. In AR, (i) The analysis expect that the time arrangement as a composite of stochastic and deterministic segments. (ii) The time-domain data are used to identify a best-fit model from which a number of peaks and the final spectrum are derived. (iii) The technique concentrates on the more significant peaks, attempting to exclude noise. Thus, in its most exceedingly terrible fundamental application the FFT approach could be viewed as an expressive strategy and the AR approach would be more predictable with a statistical or stochastic approach for ECG components analysis. In Practical application, this refinement is obscured by the basic use of smoothing calculations or windowing to stabilizevariance estimates from FFT analyses. In spite of the fact that there are a number of disadvantages and advantages to each of these strategies, there are also numerous similarities that in practice usually lead to basically equivalent results(Parati *et al.*, 1995).

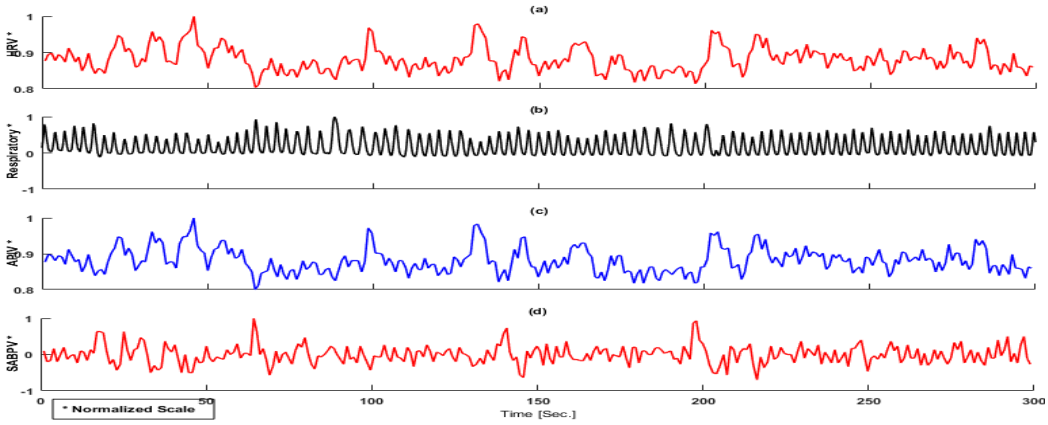

Figure 20: Raw data of (a) HRV (b) Respiratory (c) APIV (d) SABPV in time domain.

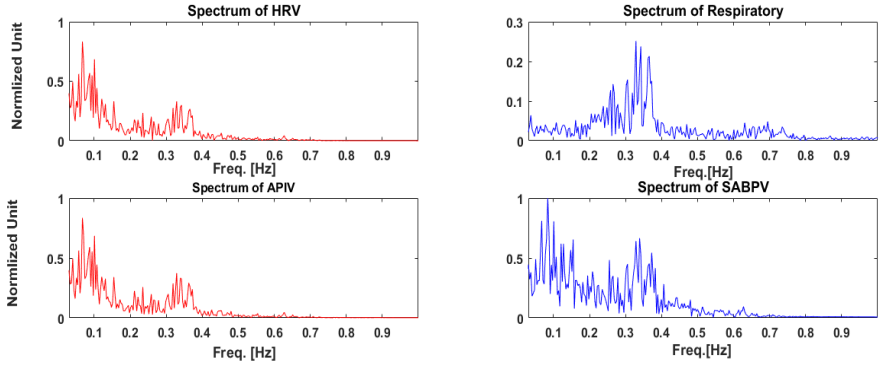

Figure 21: Power spectral of (a) HRV (b) Respiratory (c) APIV (d) SABPV using FFT.

We have studied the power spectral density (PSD) of the HRV (R-R interval components of ECG complex), respiratory, atrial pulse interval variability (APIV) and systolic atrial blood pressure variability (SABPV) of a healthy young subject in time domain (Figure 20) are shown in Figure 21. Which show that, the PSD of HRV consistently display three different power spectral peaks at around 0.03, 0.1, and 0.25-0.38 Hz respectively. The peak power at 0.1 Hz does not always occur exactly at this frequency but varies by up to 10% [63]. In this review paper, however, we will refer to Mayer wave oscillation as the 0.1 Hz peak. The MF around 0.1 Hz and HF around 0.25-0.38 Hz peaks are of particular interest to us. The 0.1-Hz peak reflects the oscillatory rhythm of the SABPV control system, containing the baroreceptor or blood pressure sensor part, afferent neural information from arterial baroreceptors to the cardio regulatory center, and the vagal and sympathetic efferent nerve traffic modulating the HR, myocardial contractility, and systemic vascular resistance (Nandagopal D. N., Fallen and Ghista, 1985)(Luczak and Laurig, 1973). The PSD peak at 0.25-0.3 Hz is due to respiratory sinus arrhythmia and reflects vagal activity. For synthetic data analysis of HRV and respiratory signal the equation (1), (2) and (3) can be employed.

$$X(t) = Re\left\{A_{iX,LF}(t)exp^{-i\,\theta_{iX,LF}(t)} + A_{iX,HF}(t)exp^{-i\,\theta_{iY,HF}(t)}\right\} + AWGN \qquad (1)$$

$$Y(t) = Re\left\{A_{iY,LF}(t)\,exp^{-i\theta_{iY,LF}(t)} + A_{iY,HF}(t)exp^{-i\,\theta_{iY,HF}(t)}\right\} + Al \quad (2)$$

The components of instantaneous amplitude as $A_{iX,LF}$, $A_{iX,HF}$ *and* $A_{iY,LF}$, , and instantaneous phase as $\theta_{iX,LF}$, $\theta_{iY,HF}$ *and* $\theta_{iY,LF}$, $\theta_{iY,HF}$ $in\,rad$ of signals of X and Y in LF and HF band derived as

$$A_{iX,LF}(t) = 4 + 3\sin(2\pi t) \;\; and \;\; A_{iX,HF}(t) = 4 + 1\cos(\quad (3)$$

5.2 *DETECTION OF ARRHYTHMIA USING SPECTRAL METHODS*

Automatic ECG components investigation is basic for determination and treatment of critically cardiac patients. Modeling and simulation of ECG under different conditions are vital in understanding the functioning of the cardiovascular system and also in the diagnosis of cardiac diseases. Arrhythmia signifies a serious threat to the patient recuperating from acute myocardial infarction, particularly ventricular arrhythmias like ventricular tachycardia (VT) and ventricular fibrillation (VF). Specifically, VT and VF are perilous conditions and create significant hemodynamic disintegration [66]. There is a requirement for quick distinguish recognizable proof of these conditions. Different arrhythmias like atrial premature contraction (APC), premature ventricular contraction (PVC) and supraventricular tachycardia (SVT) are not as fatal as VF, but rather are critical in diagnosing the disorders of the heart. The fast and reliable identification of these arrhythmias constitutes a challenge for a cardiovascular diagnostic system. Subsequently, significant measure of research has concentrated on the advancement of algorithm for exact diagnosis of cardiac arrhythmias.

Recently, several of techniques like Fourier transform, AR modeling, fuzzy adaptive resonance theory mapping as well as correlation waveform analysis (CWA)(Caswell *et al.*, 2002), time-frequency analysis like wavelet transform , stockwell transform and short time Fourier transform and Smoothed Pseudo-Wigner-Ville distribution(Afonoso and Tompkins, 1995) have been used for detection of arrhythmias. For detection of cardiac ventricular arrhythmias, different features as QRS and ST segment based values; HRV, spectral features (HF, LF, and VLF power) and AR modelingcoefficients are extracted from the ECG complex. Cardiac arrhythmias can be detected using the CWA including two intracardiac channels(Caswell *et al.*, 2002). The CWA was used to identify ECGcomplex morphologic variations in the intra cardiac electrogram channels, after compared to electrograms during normal sinus rhythm. In which, each electrogram had its particular formatand the ECG templates were acquired by signal averaging the P-QRS-T ECG waveform from a passage of normal sinus rhythm. Trigger software was used to line up the template with the P-QRS-T cycle being verified. Methods such as advanced cluster analysis methods with Fourier transform has been used for direct ECG feature detection and classification of arrhythmia(Zhou, Rautaharju and Calhoun, 1993)(Minami, Nakajima and Toyoshima, 1999). The fundamental target of the direct ECG feature identification was to investigate what number of various ventricular conduction defects classes can be framed by advanced cluster analysis methods, which minimize the number of classification parameters into a rationally small set for an expressive classification. The second target was investigated that what range to select a set of repolarization parameters. It would behelped in identification of differentvarious ventricular conductiondefects. In this study,

various featuresas QRS duration, T amplitude, T axis angle, R-R interval (HRV), spatial angle and QRS axis angle are extracted for detection of ventricular conduction defects(Barro *et al.*, 1989).

The VT and VF arrhythmia were detected by amethod, which is based on averaged threshold crossing intervals(Thakor, Zhu and Pan, 1990)(Zhang *et al.*, 1999). In this analysis, HRV (R-R interval) features were used for averaged threshold crossing intervals. Further, a modified sequential (MS) detection method was proposed to improve the sensitivity, accuracy and specificity of detecting VT and VF arrhythmias(Chen, Clarkson and Fan, 1996). A FFT based algorithm has been suggested for the detection of supraventricular tachycardia (SVT)rhythms from ventricular rhythms(Minami, Nakajima and Toyoshima, 1999).In which, power spectra computed from the QRS complexes extracted from ECG signals was classified using a neural network. High accuracy, specificity and sensitivity values greater than 98% have been reported for separating supraventricular rhythms from ventricular rhythms. A new algorithm based on complexity measuresusing extended Lyapunov exponents was proposed for the detection of NSR, VT and VF (Zhang *et al.*, 1999). The algorithm was tested for varying lengths of data and very high accuracy values100% for a test set of 204 body surface records (85 monomorphic VT,34 NSRand 85 VF) were achieved for data lengths of 7 sec for classifying NSR, VT and VF. This algorithm was also suggested for real-time practice in automatic detection of external defibrillators.

The AR method has been used broadly to investigateHRV and for power spectrum estimation of ECGcomplex ofarrhythmiassubjects(Mukhopadhyay and Sircar, 1996)(Pinna, Maestri and Di Cesare, 1996)(Bennett *et al.*, 2002). Features extracted from AR coefficient conjunction with other features have been used for arrhythmia classification(Arnold *et al.*, 1998)(Mainardi *et al.*, 1995)(Ham and Han, 1996). In this procedure, two AR coefficients, conjunction with the mean-square value of the QRS of ECG complex segments and HRV were utilized as features for classification of normal and abnormal PVC, where the prediction order was used only two. A fuzzy adaptive resonance theory mapping (ARTMAP) was used for classification and detection. The best result of PVC accuracy and sensitivity weremore than 92% under the ratios of the training data size and validation data size was 2 to 4(Ham and Han, 1996). It has been suggested that the increasing model order would not minimize the prediction error. This suggests that a linear predictor order of two is adequate for quickcardiac arrhythmia detection (S. Lawrence Marple, 1987).

The AR models are popular due to the linear form of the system of simultaneous equations involving the unknown AR model parameters and the availability of efficient algorithm for computing the solution(Anderson, Stolz and Shamsunder, 1998). Various arrhythmias including Atrial Premature Contraction (APC), Premature Ventricular Contraction (PVC), Supraventricular Tachycardia (SVT), Ventricular Tachycardia (VT) and Ventricular Fibrillation (VF) were classified using AR coefficients computed from the ECG signals and classifieras a generalized linear model (GLM). The accuracy of detecting NSR, APC, PVC, SVT, VT and VF were 93.2% to 100% using the GLM based classification algorithm(P and JA, 1989)(Ge, Srinivasan and Krishnan, 2002).The Phillip et al. (Staniczenko, Lee and Jones, 2009)have proposed a new technique based on measurement of spectral entropy to discriminate

between NSR of the heart and two types of cardiac arrhythmia like atrial fibrillation and atrial flutter. This spectral entropy measure is driven by typical differences in the power spectra of beat timings amid for three rhythms. In this process, the calculation is set to detect abnormal rhythms within 6 s, it concurs with 85.7% of the annotations of professional rhythm assessors; for a reaction time of 30 s, this increased to 89.5%, and with 60 s,it is 90.3%. Thus, this method is quick to distinguish atrial fibrillation, showing usable reaction times as low as 6 s.

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The rate of the fibrillation signals has been calculated by average refractory period of the atria(Capucci *et al.*, 1995). The foremost fibrillation frequency can be acquired using the maximum peak of thepower spectrum of the atrial signal(Holm *et al.*, 1998). The atrial average refractory period or the fibrillationfrequency is known to be pretentious by autonomic modulation. AF frequency has been used to monitorcardiac variations along with effects of sympathetic and parasympathetic activity. Studiesdepict that the fibrillation frequency increases in the morningand decreases during night(Bollmann *et al.*, 2000)(CJ *et al.*, 2001) andthat both carotid sinus massage (parasympathetic activity)(Bollmann *et al.*, 2001)and head-up tilt (sympatheticactivity)(Ingemansson, Holm and Olsson, 1998)changed the fibrillation frequency.The fibrillation frequency can also be used to detect impulsive AF activities and therapeuticeffects. Several studies have demonstrated the significant correlation between AF frequency and theprobability of spontaneous or drug-induced AF termination. A low fibrillation frequency has been provedto be a good predictor of spontaneous AF termination(Nilsson *et al.*, 2006). When the fibrillation recurrence is underneath 6 Hz the probability effective pharmaceutical cardio version is greater(Bollmann *et al.*, 1998)(Bollmann *et al.*, 2002). The risk ofearly AF recurrence is also higher for patients with higher AF frequency(Langberg, Burnette and McTeague, 1998). These are essential contemplations when choosing candidates for cardio AF arrhythmias(Sandberg, 2007).

6. CONCLUSION

In literature review, it has been concluded that the spectral methods in frequency domain and wavelet transform methods in time-frequency domain are a flexible decomposition tool for multicomponent signal which can form the foundation of useful ECG signal analysis. Along with these methods, number of classifiers is used for classification of cardiac arrhythmias. It is anticipated that the future will see advance utilization of the spectral and wavelet transform to the ECG as the developing technologies in view of them are groomed for practical purpose.

7. CONFLICT OF INTEREST: The authors declare that they have no conflict of interest.

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