

Wavelet Power Spectral Analysis of abnormal ECGs

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Abstract

Many mathematical analysis of ECG provide somewhat meaningful interpretation, and there by proper diagnosis. One of the possibilities is analysis of time frequency spectra of the signals. Time frequency analysis reveals changes in signals with the optimum time and frequency resolution. Thus, it can help to detect low level and short time changes at the same time. Over the last a decade and half, the Wavelet transform (WT) has proven to be a valuable tool in many applications of non-stationary signals such as the biomedical signals in general and ECG in particular. The WT provides a time frequency representation of the signal, and thus suitable for the inspection of characteristic waves of the ECG signal at different scales with different resolutions. The application of Wavelet Power Spectrum (WPS) to ECG signals for extracting useful information is attempted here. The WPS is the distribution of energy of signal in the time scale plane expressed in power per frequency unit, like the spectrogram. ECGs of Normal, Hypertensive, Diabetic and Cardiac patients are included in this study.

Keywords: Wavelet Power Spectrum, ECG, discrete Wavelet transform, aVL lead

I. INTRODUCTION

The incidence of heart disease has doubled in the past 30 years and it is highly likely that cardiovascular disease (CVD) will soon become the number one killer. People in the high risk category are those who have a history of heart disease, stroke or diabetes, and/or those who have hypertension, high cholesterol levels in addition to those who are obese and smoke. The cumulative effects of aging on the cardiovascular system and the progressive nature of risk factors substantially increase the risk of heart disease.

Diabetes must be considered as a major risk factor causing CVD. CVD accounts for at least 66% of deaths in diabetic patients [1]. Diabetes mellitus can be considered as a vascular disease because it causes both microvascular and macrovascular complications [2]. In addition, studies suggest that diabetic patients are more prone to 'silent' or asymptomatic disease [3],[4],[5] which could be seen in Electrocardiogram (ECG) however with no symptoms and possibly poorer prognosis.

Hypertension (HT) is a major risk factor for coronary heart disease (CHD). CHD is the first cause of morbidity and mortality in hypertensive patients [6]. Early detection of changes in cardiac performance, before irreversible damage to the heart, can contribute substantially to a further decline in hypertension related death.

The following characteristics in ECG help to identify various problems associated with heart although to discriminate a patient whether he/she is Normal, Diabetic, Hypertensive or Cardiac is a difficult procedure even for physicians/specialists based on only ECG analysis [7].

1.1 Characteristics of Diabetic ECG

There are two types of diabetes mellitus Type 1 (young diabetics) or Insulin dependent Diabetes Mellitus (IDDM) which is treated with injection of insulin only. Type 2, or Non Insulin Dependent Diabetes Mellitus (NIDDM), is treated with tablets alone (Metformin, Glibenglamide, etc) or tablets with injection of insulin (Human insulin is preferable). Both Type 1 and Type 2 diabetes are independent risk factors for CVD [8][9].

The amplitudes/heights of R and T waves are less and that of P wave is higher in the ECG of patients suffering from cardiac autonomic neuropathy (CAN) [10] which is either induced by Type 1 or/and Type 2 diabetics. The lower amplitude of the T wave is in accordance with earlier findings [11], as well as its further drop in the course of advanced stage of diabetes [12]. QT interval (the distance between the beginning of Q wave and the end point of T wave) prolongation is a constant feature of CAN [13]. The prolongation of the QT interval, accompanied by the enhancement of vulnerable phase of the cardiac cycle [14] may be associated with poor prognosis as well as with the increased risk of sudden death in diabetic patients with CAN [15],[16]. ECG of a Type 2 diabetic with unstable angina exhibits ST (the distance between the end point of S wave (QRS complex) and the beginning of T wave) depression which is more than 0.05 mV or T wave inversions of more than 0.2 mV in at least 2 adjacent leads. Painless ST segment depression is common in diabetic patients. Their ECG with ST segment depression by 1 mm or more in any lead indicate the onset of Myocardial Ischemia [17]. Approximately half of all the ischemic ECG abnormalities are silent [18]. Myocardial Infarction is generally diagnosed by a typical chest discomfort with an appearance of ST elevation or new Q wave in ECG.

The heart rate of a diabetic patient is higher, the duration of the QRS complex is longer and the PQ interval is shorter than in control subjects. Diabetes mellitus leads to collagen accumulation in the interstitium of the cardiac muscle, resulting in diffuse damage of the myocardium [19]. Such a latent derangement of left ventricular function, as a feature of cardiomyopathy, can be manifested by decreased voltage of the R wave and prolongation of the QRS complex on ECG at rest. A low R wave was also reported by other authors [20],[21] and in the course of time it was found to diminish further [22]. A twelve lead resting diabetic ECG can be within normal limits in contrast with treadmill/exercise ECG even in an advanced stage of coronary artery disease [18]. Therefore stress test plays an important role in the detection of significant coronary stenosis (narrowing of cardiac valves) [23],[24].

1.2 Characteristics of Hypertensive ECG

Repolarization anomalies are frequently found in ECG of hypertensive patients, in particular negative T waves in the lateral leads (LI, aVL, V5 and V6) indicating systolic overload of the left ventricle, frequently associated with Left Ventricular Hypertrophy (LVH).

Both ventricular and atrial forms of arrhythmia are common in patients with HT. Atrial Fibrillation (AF) is the most common form of atrial arrhythmia associated with HT [25]. Patients with an electrocardiographic diagnosis of LVH have a 3.0 to 3.8-fold increased risk of developing AF [26]. Ventricular arrhythmia is usually triggered by simple or complex ventricular extra systole whereas the mechanism whereby tachycardia is perpetuated more usually involves a reentry circuit. Ventricular premature complex is more common in hypertensive subjects when there is concomitant LVH [27][28]. HT is associated with an increased risk of sudden death, essentially due to ventricular arrhythmia [29]. There is a multiplicity of mechanisms related to HT that lead to the development of Myocardial Ischemia [30]. This leads to an inequality between the transport and consumption of oxygen by the myocardium.

1.3 Characteristics of Cardiac ECG

Cardiac diseases either induced by long time effect of Diabetes and/or Hypertension or independently affect the heart adversely resulting in premature death. During acute cardiac complications, proper interpretation of ECG helps in saving the life. Characteristics of ECG under severe cardiac conditions are briefly discussed herein.

Many categories of patients are at risk of arrhythmia and/or sudden death. ECG findings associated with sudden death include; resting tachycardia [31], QT prolongation [32], frequent premature ventricular contractions [33], non-sustained ventricular tachycardia [34][35]etc. Prolonged QRS duration is also associated with sudden death [36]. Myocardial Infarction patients with documented VT (abnormal rapid heart rhythm that originates from ventricles) have significantly increased high frequency components corresponding to prolonged QRS durations and ventricular late potentials (VLPs). QT dispersion (QTd) is the difference between the maximal and minimal QT interval on the standard 12 lead ECG. [37] have shown that a QTd > 80 ms diagnosed 2–3 months after Myocardial Infarction identifies patients at increased risk of arrhythmic cardiac death in the next 2–3 years

ST segment depression on the other hand is associated with ischemia and may be a precursor to death or acute Myocardial Infarction [38]. Heart Rate Variability (HRV) abnormalities are

strongly associated with mortality [39],[40],[41]. HRV may also be useful for identifying high risk patients with an increased risk of fatal cardiac events [42].

1.4 Wavelet Power Spectrum

Mathematically, the WPS can be expressed as [43]:

$$WPS_f^\psi(j,k) = |DCWT_f^\psi(j,k)|^2 \quad \dots 1$$

where the discretized version of continuous Wavelet transform (DCWT) using the discrete wavelet $\psi_{j,k}(t)$ is expressed as:

$$DCWT_f^\psi(j,k) = \int f(t) \psi_{j,k}^*(t) dt \quad \dots 2$$

where $\psi_{j,k}(t)$ is the translated and scaled mother wavelet $\psi(t)$. By varying the wavelet scale and translating along the localized time index, the coefficients of DCWT are obtained which in turn result the WPS. In the WPS, variation of amplitude versus both time as well as scale is realized [44]. A non-orthogonal analysis such as the DCWT is highly redundant at large scales, where the wavelet spectrum at adjacent times is highly correlated. The non-orthogonal transform is useful for time series analysis, where smooth and continuous variations in wavelet amplitude are expected [45].

Based on a previous extended survey on wavelets suitable to ECG analysis [46], the wavelets namely, Morlet wavelet and Mexican hat wavelet are found to be useful in such a study.

1.4 Morlet Wavelet

The Morlet wavelet was formulated by J. Morlet in the field of seismic data analysis [47]. The Morlet wavelet is complex, symmetric with no corresponding scaling function which can only be used for CWT analysis. Although the Morlet wavelet has infinite support, its effective support is only in the range [-4 4]. It is one of the first wavelets to be used in practical time frequency analysis. A Morlet wavelet is formed by a plain wave modulated by a Gaussian function and it is given by [47]:

$$\psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} e^{i\omega_0 t} e^{-t^2/2} \quad \omega_0 \geq 5 \quad \dots 3$$

Morlet wavelet ensures better frequency localization however lacks time localization; this is expected by the Heisenberg's Uncertainty Principle [48]. Morlet wavelet provides the best

compromise between time and frequency resolution. When the analysis is focused on amplitude and phase changes, a complex wavelet such as the Morlet wavelet can be the most appropriate. This helps to retrieve the oscillatory behaviour of the data. Figure 1 shows the real and imaginary parts of the Morlet wavelet.

1.5 Mexican Hat Wavelet

Mexican hat or Marr wavelet is symmetric and is the second derivative of the Gaussian probability density function and is expressed as [49]:

$$\psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} (1-t^2) e^{-t^2/2} \quad \dots 4$$

The shape of this wavelet is similar to that of Mexican hat (Figure 2) This wavelet has two vanishing moments and evidently the wavelet and all its derivatives have Gaussian decay. This function has an infinite support; nevertheless its effective support is in the interval [-5, 5]. The Marr wavelet in its generalization to N dimensions is isotropic and therefore cannot discriminate different directions of the signal [50].

1.6 Mathematical Aspects of Mother Wavelets

The complete Morlet wavelet in contrast with Morlet wavelet given in equation (3) is defined as [51],[52]

$$\psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} \left(e^{i\omega_0 t} - e^{-\frac{\omega_0^2}{2}} \right) e^{-t^2/2} \quad \dots 5$$

where ω_0 is the central frequency of the mother wavelet. The second term in the brackets is known as the correction term, as it corrects for non zero mean of the complex sinusoid of the first term. In practice it becomes negligible when $\omega_0 \geq 5$.

In this case, the Morlet wavelet becomes

$$\psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} e^{i\omega_0 t} e^{-t^2/2}, \quad \omega_0 \geq 5 \quad \dots 6$$

This truncated Morlet wavelet is invariably found in the literature and often referred to as simply the Morlet wavelet. The Morlet wavelet is a complex sinusoid within the Gaussian envelope, where the central frequency, ω_0 in effect determines the number of significant oscillations of the complex sinusoid in the Gaussian window. The complex sinusoidal

waveform is contained in the term $e^{i\omega_0 t}$. The Gaussian envelope $e^{-\frac{t^2}{2}}$ has unit standard deviation and confines both the real and imaginary parts of the complex sinusoidal waveform. The imaginary part is phase shifted from the real part by a quarter period. The term $\frac{2}{\sqrt{3}}\pi^{-1/4}$ is a normalization factor which ensures that the wavelet has unit energy.

The admissibility constant, c_ψ of the wavelet is given as:

$$c_\psi = \int_0^\infty \frac{2\sqrt{2}}{3} \sqrt{\pi} \frac{e^{-(\omega-\omega_0)^2}}{\omega} d\omega \quad \dots 7$$

Note that various other normalizations are common in literature and each of which requires a different c_ψ to be used to ensure that the original signal and its transform contain the same energy.

The Fourier transform of the standard Morlet wavelet is given as:

$$\hat{\psi}(\omega) = \frac{2\sqrt{2}}{\sqrt{3}} \pi^{1/4} e^{-\frac{(\omega-\omega_0)^2}{2}} \quad \dots 8$$

and its energy spectrum is expressed as:

$$|\hat{\psi}(\omega)|^2 = \frac{8}{3} \sqrt{\pi} e^{-(\omega-\omega_0)^2} \quad \dots 9$$

which implicitly plays a significant role in the corresponding Wavelet transform applications.

Further, the complete Morlet given by equation (5) can be rewritten as:

$$\psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} \left(\cos \omega_0 t + i \sin \omega_0 t - e^{-\frac{\omega_0^2}{2}} \right) e^{-t^2/2} \quad \dots 10$$

The real part of equation (10) is given as:

$$\text{Re} \{ \psi(t) \} = \frac{2}{\sqrt{3}} \pi^{-1/4} \left(\cos \omega_0 t - e^{-\frac{\omega_0^2}{2}} \right) e^{-t^2/2} \quad \dots 11$$

On simplification by neglecting the higher order terms, the real part reduces to,

$$\text{Real}\{\psi(t)\} = \frac{2}{\sqrt{3}} \pi^{-1/4} \frac{\omega_0^2}{2} (1-t^2) e^{-t^2/2} \quad \dots 12$$

which is similar to the Mexican hat wavelet given by equation (4).

On the other hand, the complex Mexican hat wavelet which is also known as complex Gaussian wavelet (No.2) (Figure 3) can be formulated by setting the Fourier spectrum to zero for negative frequencies prior to inverse Fourier transform.

II. METHODOLOGY

The second cycles in aVL lead of ECGs pertaining to Normal, Diabetic, Hypertensive and Cardiac patients were denoised using the biorthogonal wavelet 'bior1.1' (Figure 4) before subjecting them for further analysis. After many experiments, biorthogonal wavelets were found to yield reliable results in signal reconstruction from approximation coefficients of the discrete Wavelet transform (DWT).

The WPS of known categories of denoised ECG (Normal, Diabetic, Hypertensive and Cardiac) were computed using Mexican hat (second derivative of Gaussian function) and Morlet wavelet. Figures 5a, 6a, 7a and 8a represent the second cycle of aVL lead and the corresponding WPS of the aforesaid four categories of ECG using Mexican hat wavelet is given in Figures 5b, 6b, 7b and 8b using Morlet wavelet is Figures 5c, 6c, 7c and 8c. To make the WPS analysis clearer, the Fourier period is used instead of wavelet scale as the dimension of the vertical axis.

III. RESULTS AND DISCUSSION

The investigated results of second cycle of aVL lead in all four types of (Normal, Diabetic, Hypertensive and Cardiac) ECGs have shown a fairly good response and are specified hereunder.

The Wavelet Power Spectrum (WPS) with both Mexican hat and Morlet wavelets result in symmetric energy distribution in Normal case with high energy (red colour) of 40 milli seconds duration approximately.

Prolonged high energy distribution (red colour) of approximate 80 milli seconds duration is observed in WPS of both Mexican hat and Morlet wavelet of Diabetic which may be representing QT interval prolongation as an indication of patient suffering from CAN [53].

Negative T wave in the aVL lead of Hypertensive ECG signifies repolarization anomaly and the corresponding WPS of Mexican hat wavelet (Figure 6 (b)) has prolonged high energy distribution between 220 to 280 milli seconds in the T wave region signifies Left Ventricular Hypertrophy (LVH).

A VLP detection method based on the analysis of the behavior of the wavelet energy density surface in a selected time frequency region occurring beyond the end of the QRS complex was proposed by [54]. The high energy distribution in the WPS of Mexican hat wavelet (Figure 7(b)) indicates presence of Myocardial Infarction (death of cardiac cells due to lack of oxygen) in the cardiac ECG (Figure 7(a))

$$\text{Morlet wavelet: } \psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} e^{i\omega_0 t} e^{-t^2/2}, \quad \omega_0 \geq 5$$

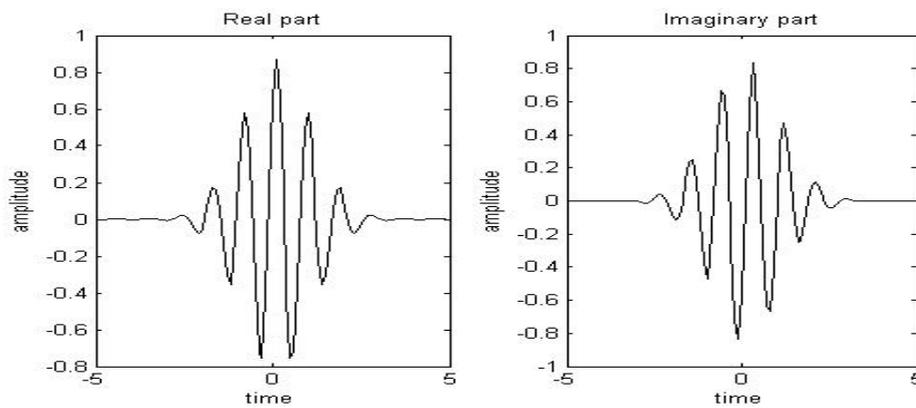


Figure1: Real and imaginary parts of Morlet wavelet

$$\text{Mexican hat wavelet: } \psi(t) = \frac{2}{\sqrt{3}} \pi^{-1/4} (1-t^2) e^{-t^2/2}$$

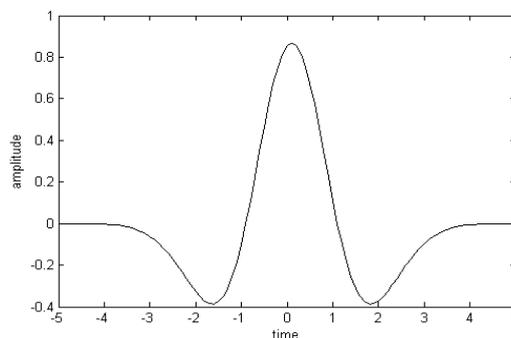


Figure 2: Mexican hat wavelet

complex Gaussian wavelet: $\frac{d^2}{dt^2} \left(e^{-i\omega_0 t} e^{-t^2} \right), \omega_0 \geq 5$

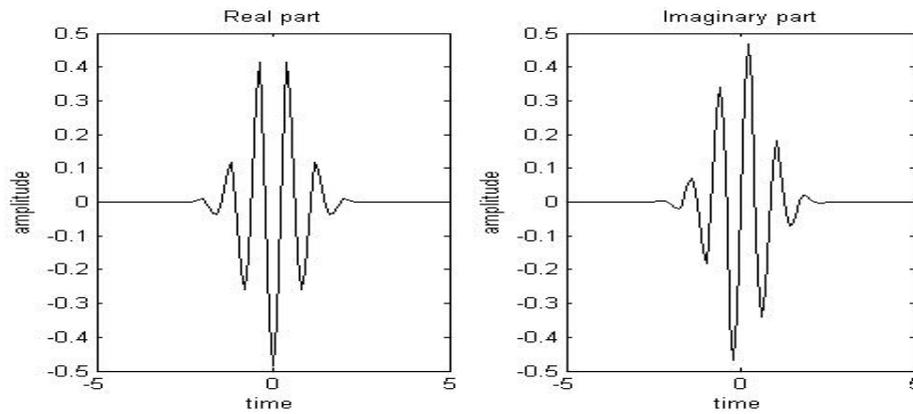


Figure 3: Real and imaginary parts of Gaussian wavelet

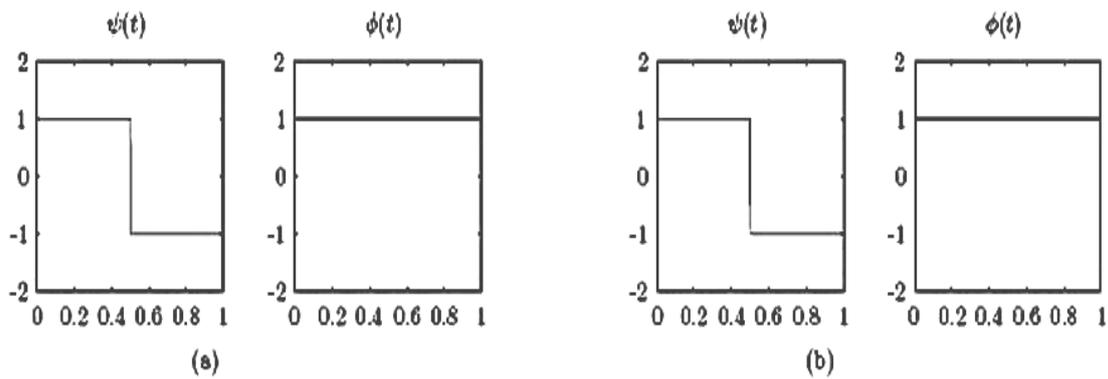


Figure 4: (a) Decomposition and (b) Reconstruction filters of 'bior1.1'

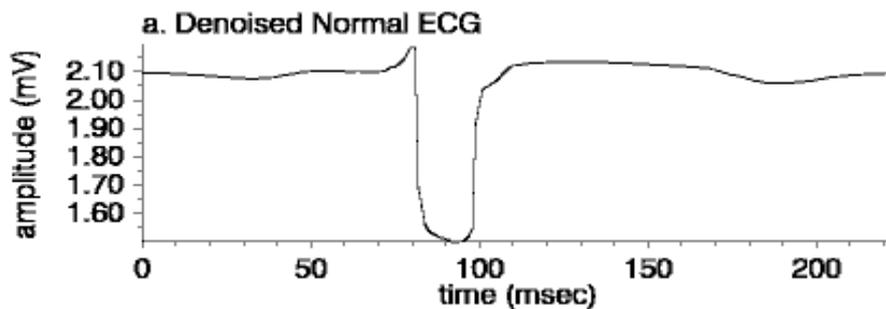


Figure 5 (a): The denoised Normal ECG of the second cycle of aVL lead

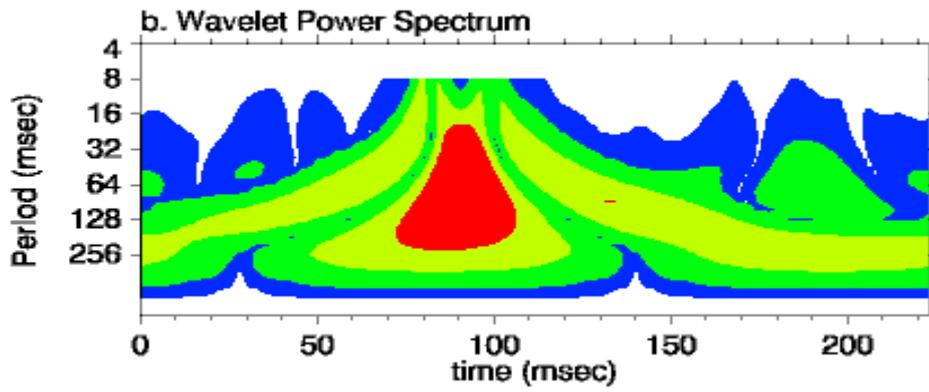


Figure 5 (b): The WPS using Mexican hat wavelet

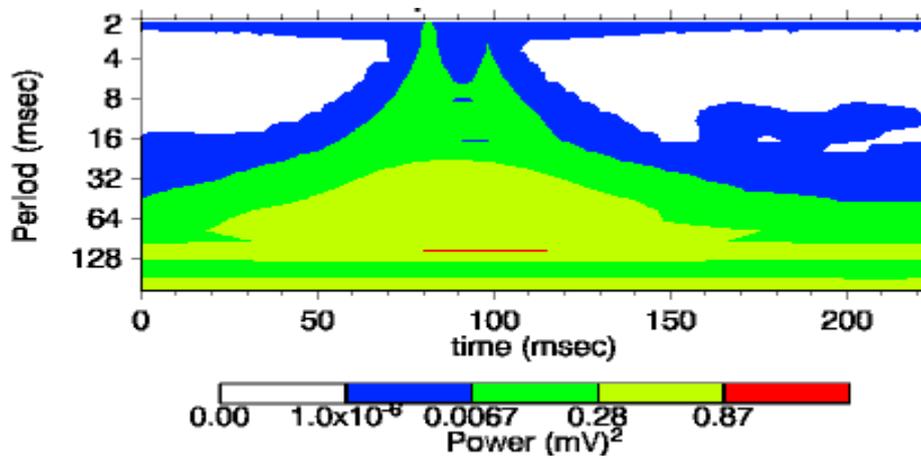


Figure 5 (c): The WPS using Morlet wavelet

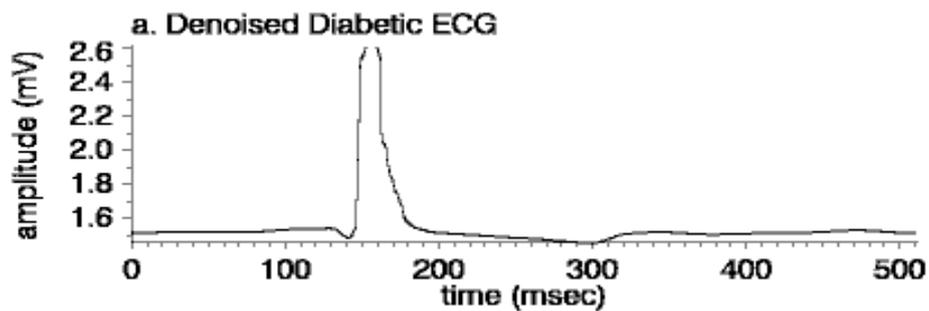


Figure 6 (a): The denoised Diabetic ECG of the second cycle of aVL lead

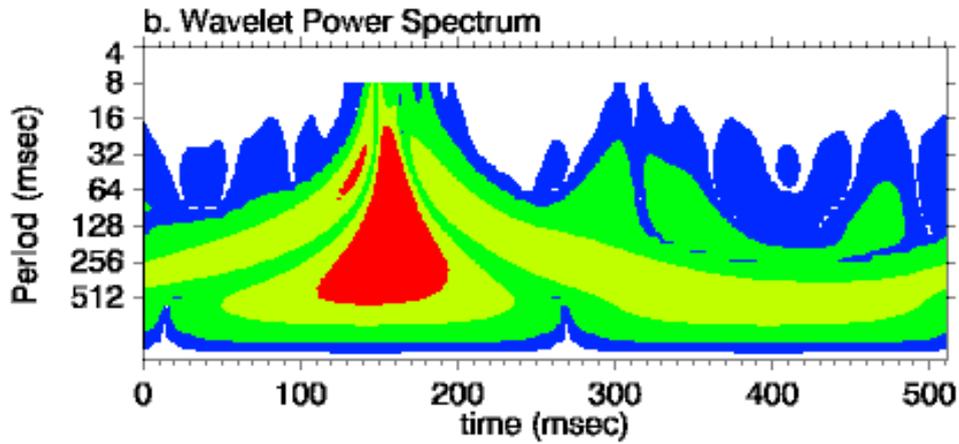


Figure 6 (b): The WPS using Mexican hat wavelet

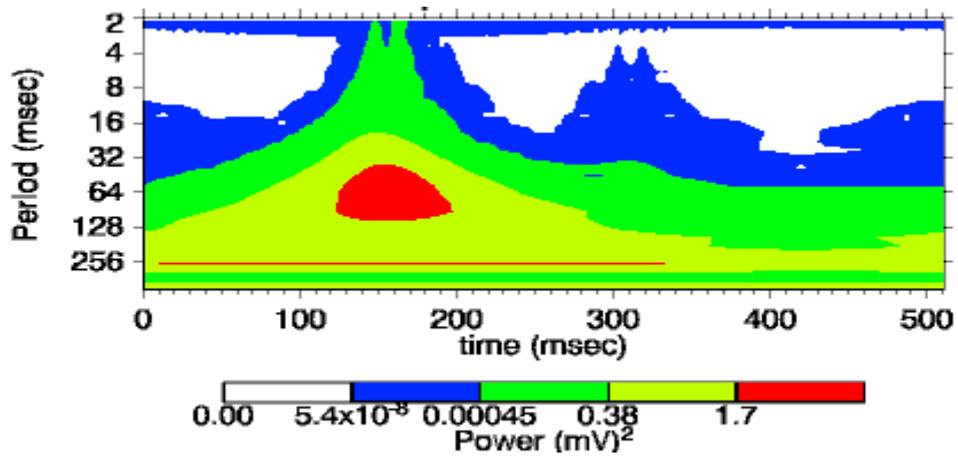


Figure 6 (c): The WPS using Morlet wavelet

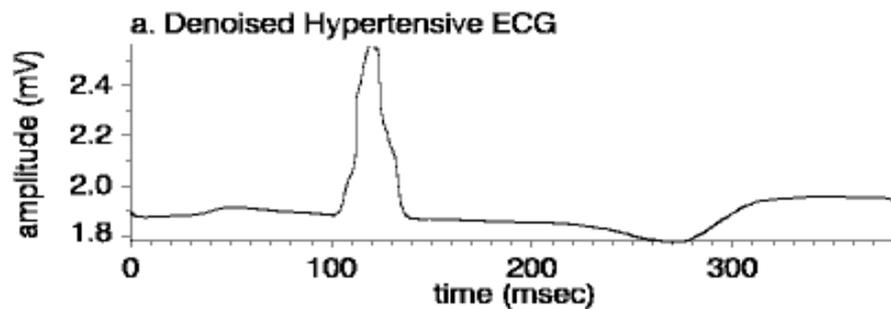


Figure 7 (a): The denoised Hypertensive ECG of the second cycle of aVL lead

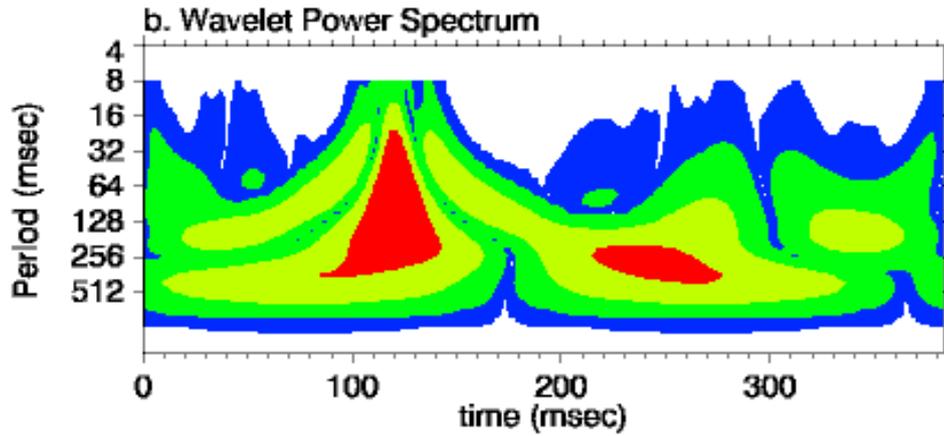


Figure 7 (b): The WPS using Mexican hat wavelet

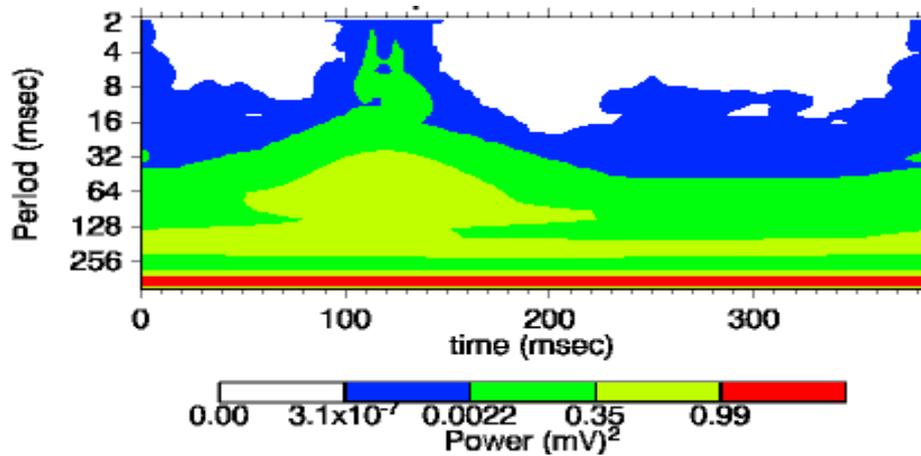


Figure 7 (c): The WPS using Morlet wavelet

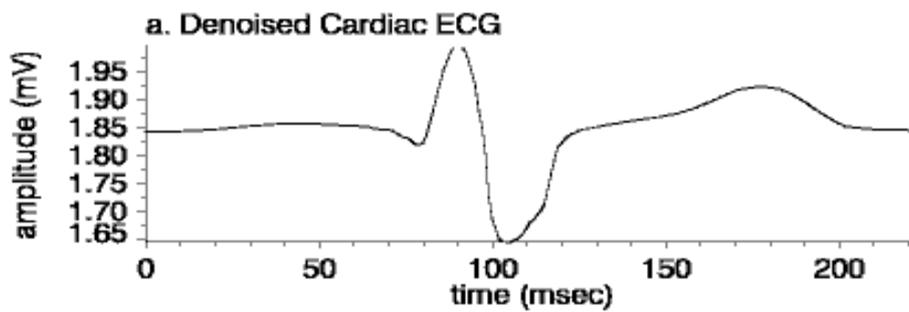


Figure 8 (a): The denoised Cardiac ECG of the second cycle of aVL lead

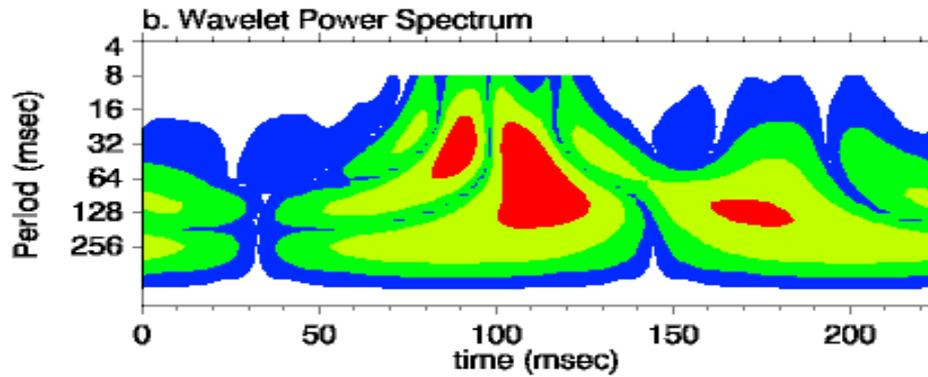


Figure 8 (b): The WPS using Mexican hat wavelet

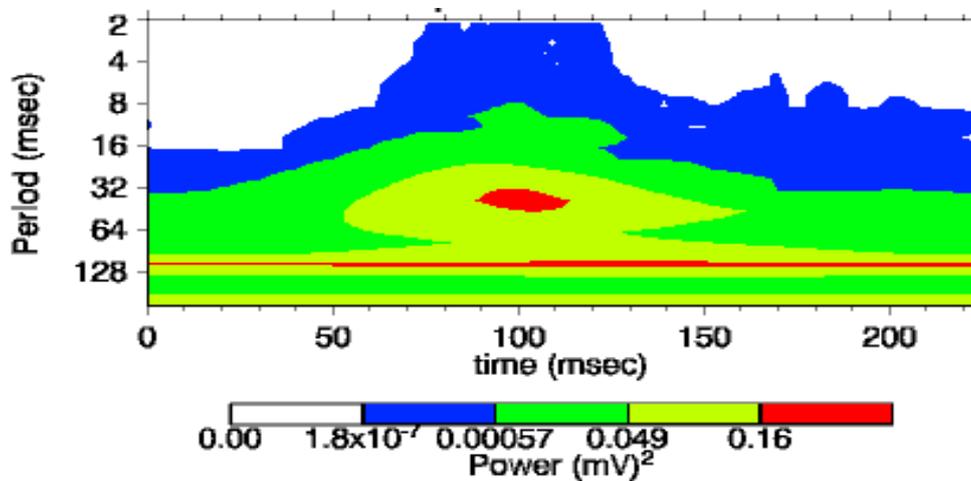


Figure 8 (c): The WPS using Morlet wavelet

References

- [1] Zimmet P Z and Alberti K G, 1997. The changing face of macrovascular disease in noninsulin-dependent diabetes mellitus: an epidemic in progress. *Lancet* 350 (suppl I):1-4.
- [2] Nathan D M, Meigs J and Singer D E, 1997. The epidemiology of cardiovascular disease in type 2 diabetes mellitus: how sweet it is... or is it? *Lancet* 350(suppl I): 4-9.
- [3] Kannel W B and McGee D L, 1979. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham study. *Diabetes Care*; 2:120-6.

- [4] Pyorala K, Laakso M and Uusitupa M, 1987. Diabetes and atherosclerosis: an epidemiologic view. *Diabetes Metab Rev*; 3 (2): 463–524.
- [5] Wingard D L, Barrett Connor E L, Scheidt Nave C and McPhillips J B, 1993. Prevalence of cardiovascular and renal complications in older adults with normal or impaired glucose tolerance or NIDDM. A population-based study, *Diabetes Care*; 16 (7): 1022–1025.
- [6] Collins R and McMahon S, 1994. Blood pressure, antihypertensive drug treatment and the risks of stroke and of coronary heart disease. *Br Med Bull* ; 50: 272-98.
- [7] Dr. Misra K P, 12th March, 2006. ECG: Myths and Facts, The Hindu, Hyderabad edition.
- [8] McGill H C Jr and McMahan C A, 1998. Determinants of atherosclerosis in the young. Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group. *Am J Cardiol*; 82 (10B): 30T–36T.
- [9] Wilson P W, D’Agostino R B, Levy D, Belanger A M, Silbershatz H and Kannel W B, 1998. Prediction of coronary heart disease using risk factor categories. *Circulation*; 97 (18): 1837–1847.
- [10] Page M and Watkins P J, 1977. The heart in diabetes: Autonomic neuropathy and cardiomyopathy. *Clin Endocrin Metab* ; 6: 377. 388.
- [11] Karlefors T, 1996. Exercise tests in male diabetics-electrocardiographic study. *Acta Med Scand*; 180: 3.18.
- [12] Persson G, 1977. Exercise tests in male diabetics. *Acta Med Scand*; S605: 7.23.
- [13] Ziegler D, Laude D and Akila F, 2001. Time and frequency-domain estimation of early diabetic cardiovascular autonomic neuropathy. *Clin Auto Res*;11:369-76.
- [14] Schwartz P and Malliani A, 1975. Electrical alternation of the T wave: clinical and experimental evidence of its relationship with the sympathetic nervous system and with the long QT syndrome. *Amer Heart J* 1975; 89: 45.50.
- [15] Kahn J K, Sisson J C and Vinik A I, 1987. QT interval prolongation and sudden cardiac death in diabetic autonomic neuropathy. *J Clin Endocrinol Metab*; 64: 751.754.
- [16] Bellavere F, Ferri M, Quarini L, Bax G, Piccoli A, Cardone C and Fedele D, 1988. Prolonged QT period in diabetic autonomic neuropathy: a possible role in sudden cardiac death? *Brit Heart J*; 59: 379.383.

- [17] Taddei A, Constantino G, Silipo R and Marchesi C, 1995. A system for the detection of ischemic episodes in ambulatory ECG, *Comput. Cardiol.*, pp. 705–708.
- [18] Wackers F J, Young L H, Inzucchi S E, Chyun D A, Davey J A, Barret E J, Taillefer R, Wittlin S D, Heller G V, Filipchuk N, Engel S, Ratner R E and Iskandrian A E, 2004. Detection of silent myocardial ischemia in asymptomatic diabetic subjects, the DIAD study. *Diabetes Care* 27(8):1954-61.
- [19] Regan T J and Weisse A B, 1992. Diabetic cardiomyopathy. *J Amer Coll Cardiol*; 19: 1165.1166.
- [20] Airaksinen K E J, 1985. Electrocardiogram of diabetic subjects. *Ann Clin Res*; 17: 135.138.
- [21] Karlefors T, 1996. Exercise tests in male diabetics-electrocardiographic study. *Acta Med Scand*; 180: 3.18.
- [22] Persson G, 1977. Exercise tests in male diabetics. *Acta Med Scand*; S605: 7.23.
- [23] Bigi R, Desideri A, Cortigiani L, Baw J J, Celegon L and Fiorentini C, 2001. Stress echocardiography for risk stratification of diabetic patients with known or suspected coronary artery disease. *Diabetes Care* 24: 1602-7.
- [24] Delorenzo A, Lima R S, SiqueiraFilho A G and Pantoja M R, 2002. Prevalence and prognostic value of perfusion defects detected by stress technetium-99 sestamibi myocardial perfusion single-photon emission computed tomography in asymptomatic patients with diabetes mellitus and no known coronary artery disease. *Am J Cardiol* 90(8): 827-32.
- [25] Madu E C, Baugh D S, Gbadebo T D, Dhala A and Cardoso S, 2001. Effect of ethnicity and hypertension on atrial conduction: evaluation with high-resolution P-wave signal averaging. *Clin Cardiol*; 24: 597-602.
- [26] Kannel W B, Wolf P A, Benjamin E J and Levy D, 1998. Prevalence, incidence, prognosis, and predisposing conditions for atrial fibrillation: population-based estimates. *Am J Cardiol*; 82(suppl): 2N-9N.

- [27] Messerli F H, Ventura H O, Elizardi D J, Dunn F G and Frohlich E D, 1984. Hypertension and sudden death. Increased ventricular ectopic activity in left ventricular hypertrophy. *Am J Med*; 77: 18-22.
- [28] Levy D, Anderson K M, Savage D D, Balkus S A, Kannel W B and Castelli W P, 1987. Risk of ventricular arrhythmias in left ventricular hypertrophy: the Framingham heart study. *Am J Cardiol*; 60: 560-565
- [29] Bayès de Luna A, Coumel P H and Leclercq J E, 1989. Ambulatory sudden death: mechanisms of production of fatal arrhythmias on the basis of data from 157 cases. *Am Heart J*; 117: 154-159.
- [30] Schwartz P, Larovere M T and Vanoli E, 1992. Autonomic nervous system and sudden death. Experimental basis and clinical observations for post-myocardial risk stratification. *Circulation*; 85(suppl I): 177-191.
- [31] Pedretti R F, Catalano O, Ballardini L, De Bono D P, Radice E and Tramarin R, 1999. Prognosis in myocardial infarction survivors with left ventricular dysfunction is predicted by electrocardiographic RR interval but not QT dispersion. *Int J Cardiol*; 68:83–93.
- [32] Zipes D P and Wellens H J J, 1998. Sudden cardiac death. *Circulation*;98: 2334–2351.
- [33] Naccarella F, Lepera G and Rolli A, 2000. Arrhythmic risk stratification of post-myocardial infarction patients. *Curr Opin Cardiol*; 15:1–6.
- [34] Marinchak R A, Rials S J, Filart R A and Kowey P R, 1997. The top ten fallacies of nonsustained ventricular tachycardia. *PACE*; 20: 2825–2847.
- [35] Cheema A N, Sheu K, Parker M, Kadish A H and Goldberger J J, 1998. Nonsustained ventricular tachycardia in the setting of acute myocardial infarction: Tachycardia characteristics and their prognostic implications. *Circulation*; 98: 2030–2036.
- [36] Jordaens L and Tavernier R, 2001. Miracle Investigators. Determinants of sudden death after discharge from hospital for myocardial infarction in the thrombolytic era. *Eur Heart J*; 22: 1153–1155.
- [37] Zareba W, Moss A J and Le Cessie S, 1994. Dispersion of ventricular repolarization and arrhythmic cardiac death in coronary artery disease. *Am J Cardiol*; 74: 550–553.
- [38] Patel D J, Knight C J, Holdright D R, Mulcahy D, Clarke D, Wright C, Purcell H and Fox K M, 1998. Long term prognosis in unstable angina: The importance of

- early risk stratification using continuous ST segment monitoring. *Eur Heart J*;19: 240–249.
- [39] Kautzner J, Stovicek P, Anger Z, Savlikova J and Malik M, 1998. Utility of short-term heart rate variability for prediction of sudden cardiac death after acute myocardial infarction. *Acta Univ Palacki Olomuc Fac Med*; 141: 69–73.
- [40] Lanza G A, Guido V, Galeazzi M M, Mustilli M, Natali R, Ierardi C, Milici C, Burzotta F, Pasceri V, Tomassini F, Lupi A and Maseri A, 1998. Prognostic role of heart rate variability in patients with a recent acute myocardial infarction. *Am J Cardiol*; 82: 1323–1328.
- [41] Lanza G A, Galeazzi M, Guido V, Lucente M, Bellocchi F, Zecchi P and Maseri A, 1999. Additional predictive value of heart rate variability in high-risk patients surviving an acute myocardial infarction. *Cardiologia*; 44: 249–253.
- [42] Dououlas A D, Flather M D, Pipilis A, Campbell S, Studart F, Rizos I K, Gialafos I H, Toutouzas P K and Sleight P, 2001. Evolutionary pattern and prognostic importance of heart rate variability during the early phase of acute myocardial infarction. *Int J Cardiol*; 77:169–179.
- [43] Qian, S and Chen D, 1996. *Joint Time-Frequency Analysis*, Prentice Hall.
- [44] Torrence C and Compo G, 1998. A practical guide to wavelet analysis: *Bull., Am. Meteor. Soc.*, 79, 61–78.
- [45] Lotric M B, Stefanovska A, Stajer D and Rovani V U, 2000. Spectral Components of Heart Rate Variability determined by Wavelet Analysis, *Physiology Measurements*, v. 21, p. 441-457.
- [46] Andreao R V, 2004. Segmentation de battements ECG par approche markovienne: application à la détection d'ischémies, INT-UFC, Evry, coopération CAPES-COFECUB.
- [47] Morlet J, Arens G, Fourgeau E and Giard D, 1982a. Wave propagation and sampling theory—part I: Complex signal and scattering in multilayered media: *Geophysics*, 47, 203–221. 1982b, Wave propagation and sampling theory—part II: Sampling theory and complex waves: *Geophysics*, 47, 222–236.
- [48] Daubechies I, 1992. *Ten lectures on wavelets*: Society for Industrial and Applied Mathematics.
- [49] Romain Murenzi, 1988. *Wavelets*. Springer, Berlin, Heidelberg, New York.
- [50] Farge M, 1992. Wavelet transforms and their applications to turbulence. *Annu. Rev. Fluid Mech.*, 24, 395–457.

- [51] Antoine J P, Murenzi R, Piette B and Duval Destin M, 1990. Image analysis with 2D continuous Wavelet transform: detection of position, orientation and visual contrast of simple objects.
- [52] Antoine J P, Carrette P, Murenzi R and Piette B, 1991. Image analysis with 2D continuous Wavelet transforms. In Wavelet Transforms and Multiresolution Signal Analysis, Willsky, IEEE Trans.
- [53] Lewandowski P, Meste O, Maniewski R, Mroczka T, Steinbach K and Rix H, 2000. Risk evaluation of ventricular tachycardia using wavelet transform irregularity of the high-resolution electrocardiogram Med. Biol. Eng. Comput. 38 666–73.