

ECG Signal analysis used as confirmative tool in quick diagnosis of Myocardial Infarction and ECG hysteresis index used as confirmative tool in cardiac health assessment.

Manjusha Joshi*, Dr.K.D.Desai**

*(Electronics Department, Assistant Professor and Research Scholar M.P.S.T.M.E,
Bhhakti vedant marg, J.V.P.D., Vile Parle (w),Mumbai-53.
Email: Manjusha.joshi@nmims.edu.)

**Dean(academics) /Director K.C. college of engineering Thane(E), Mumbai.
Email: kddesai@hotmail.com.

ABSTRACT: Apart from accidents, myocardial infarction is the most common reason of sudden deaths. As the time from the onset of myocardial infarction to the start of treatment is very crucial, quick diagnosis is essential. Paper proposes an easily deployable, cheap and non-invasive technique that can be used as confirmative diagnostic tool that diagnoses myocardial infarction and also the indices that can be used for prevention from the same. The changes in ambulatory service in case of patient gap complaining of acute chest pain. This can help of the normal patient. The degree of variation can be used as a diagnostic tool. With the advent of computer and signal processing the crucial time gap between the onset of symptoms and treatment will be reduced. The tool also helps in assessing the health of the heart as prevention from myocardial ischemia and infarction. ECG signal of the person suffering from myocardial infarction or reduced heart rate that paramedical staff to prepare for the treatment before the physician is available and crucial time variability are different to that compared with technology, the tool can be deployed as an

Keywords: Myocardial Infarction, heart rate variability, autocorrelation, crosscorrelation, ECG hysteresis

I. Introduction: Cardiovascular disease is the world's leading killer, accounting for 16.7 million or 29.2 per cent of total global deaths in 2003. The World Health Organization (WHO) estimates that 60 per cent of the world's cardiac patients will be Indian by 2010. Also it has been found

out that India has more number of cardiovascular deaths claiming younger population than in America [1]. The major reasons contributing to sudden deaths are due to myocardial infarction (commonly known as heart attack). Apart from deaths due to accidents myocardial infarction is the most commonly found episode in sudden casualty. Myocardial infarction is caused due to death of heart wall muscle due to reduced blood supply to the heart. This is a slow process and does not show any symptoms till the permanent damage has occurred. Therefore it is very essential that early diagnosis of the symptoms leading to onset on of myocardial infarction should be as quick as possible. The beginning of myocardial infarction results in to the changes in ECG of the patient. These changes are not visible morphologically in ECG but reduce the variation in the heart rate as the demanded by different activities of body. The normal person's ECG that shows more changes in the heart rate during different activities is compared to that of the patient. The method of computing heart rate variability proposed in the above paper takes into consideration the non linearity of ECG signal and hence is more accurate then the prevailing methods used in time and frequency domain techniques.[3] The proposed paper discusses the

confirmatory changes in ECG in case of different types of myocardial infarction and compares the patient's ECG with normal ECG. The ECG images available are converted in to signal so that the signal processing tools can be applied to it. The signal processing tool of cross correlation and auto correlation can confirmatively assess the degree of similarity/dissimilarity between the two signals.[2] Further there are certain dynamic ratio indices which depict the health of the heart. Computation of these parameters from the normal and patient's ECG. If patient is suffering from angina, there will not be significant changes in ECG and that possibility can be ruled out. Also the possibility of ectopic beats which can be falsely considered as a case of arrhythmia can be ruled out by the proposed test. The proposed paper uses the ECG of the patient and the normal ECG of the patient if available before onset of the disease.[4] If the normal ECG of the same patient is not available, then any other normal ECG can be used for tool for comparison.

II. Overview of Myocardial Infarction:

Myocardial Infarction refers to damage or death (infarction) of heart muscle (myocardium). The damage results from the interruption of blood supply to part of the heart causing heart cells to die. This is most commonly due to occlusion (blockage) of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (cholesterol and fatty acids) and white blood cells in the wall of an artery. If left untreated for a long period, this leads to permanent death of heart wall muscle. Since heart wall expansion and contraction is essential for proper functioning of heart, the damage of the wall affects the functioning of the heart resulting into heart failure. The type of infarction depends upon which part of the heart wall is damaged. The heart wall is mainly divided in three zones as

shown in figure-1.

The prominent types of infarction are –

1. Anterior infarction
2. Posterior infarction
3. Inferior infarction

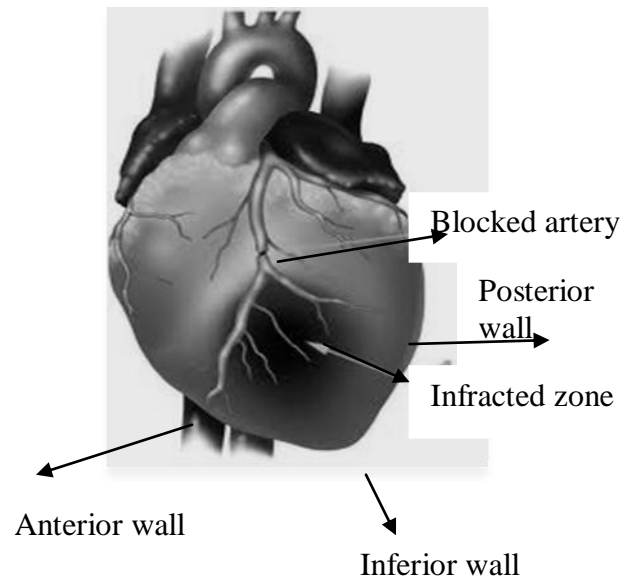


Figure-1

Acquiring ECG is non invasive, safe tool that gives instantaneous results. The ECG of an infarcted heart is different than that of a normal heart. The change in ECG is the dependent upon the location of infarct.[5]

The anterior wall infarction can be properly represented by AVL and V5-V6 leads. (Refer figure 2a)

The inferior myocardial infarction will be properly represented by AVF and V2-V3 leads. (Refer figure 2b)

The posterior infarction is not clearly visible in the ECG. Over all changes in ST segment can be the confirmative indication. These changes in the ECG can be compared to that with the normal ECG. (Refer figure 2c).

The ECG is observed to be changing depending upon the duration of the infarct.

By and large the confirmatory change in ECG is the ST segment depression and tall T wave with symmetrical taper.

The corresponding ECGs are as shown in the figure.2 [5]

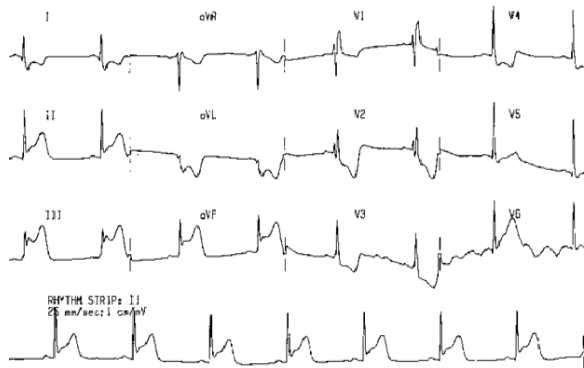


Figure 2a ECG of Posterior Infarction.

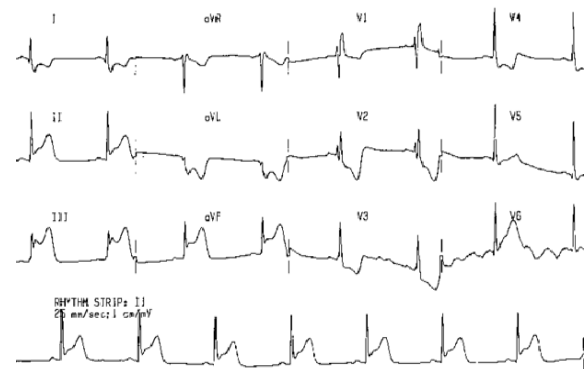


Figure 2b Required ECG leads representing Inferior Infarction

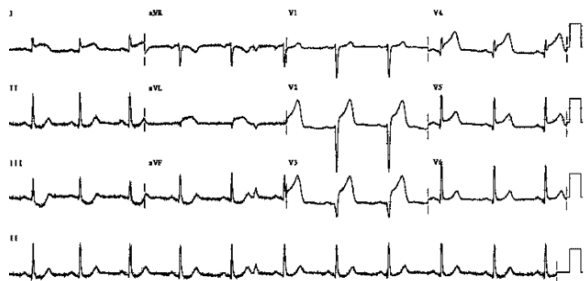


Figure 2c Required ECG leads representing Anterior Infarction

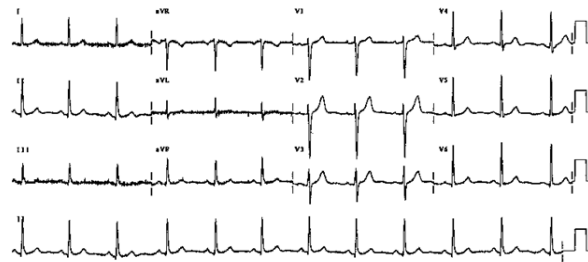


Figure 2c Normal ECG

III. Need to use cross correlation

Bio signals are highly subjective. Though there are some standard parameters of the ECG signals, the parameters tend to vary from person to person time to time. So it is not possible to build confirmative heuristic diagnostic algorithm to detect the abnormality. The suitable tool for the confirmative diagnosis is by using the cross correlation of the normal and abnormal signal. The accuracy of the diagnosis can be achieved by cross correlating the two signals over sufficiently long duration.

IV. Method used to compute HRV indices

All of the traditional HRV indices employ techniques that assume stationarity in the data. If part of the data in the window of analysis exhibits significant changes in the mean or variance over the length of the window, the HRV estimation technique can no longer be trusted. A cursory analysis of any real RR tachogram reveals that shifts in the mean or variance are a frequent occurrence. For this reason it is common practice to detrend the signal by removing the linear or parabolic baseline trend from the window prior to calculating a metric. Also the method of detrending should not remove the variations in the spectral or amplitude.

Nonstationarities in the ECG manifest both in an interbeat basis (as RR interval timing changes) and on an intrabeat basis (as morphological

changes). Although the former changes are often thought of as rhythm disturbances and the latter as beat abnormalities, the etiology of the changes are often intricately connected. Non stationarities in the ECG timing and ECG morphology can actually be well represented by nonlinear models. The transition between rhythms is a non stationary process. Heart rate hysteresis is a nonlinear technique of heart health assessment.[3]

V. Heart Rate Hysteresis

Changes in heart rate due to control of autonomic nervous system (ANS) and the morphological changes are asymmetric. As a result, changes in the QT and RR interval do not remain the same. Hence an ellipsoid like trajectory is observed in QT-RR plane. Figure 3.1 illustrates this point, with each of the central contours indicating a response of either tachycardia (TC) and bradycardia (BC) or normal resting. From the top right of each contour, moving counterclockwise (or anticlockwise); as the heart rate increases (the RR interval drops) the QT interval remains constant for a few beats, and then begins to shorten, approximately in an inverse square manner. When the heart rate drops (RR interval lengthens) a similar time delay is observed before the QT interval begins to lengthen and the subject returns to approximately the original point in the QT-RR phase plane. The difference between the two trajectories (caused by RR acceleration and deceleration) is the QT hysteresis, and depends not only on the individual's physiological condition, but also on the specific activity in the ANS. Although the central contour defines the limits of normality for a resting subject, active subjects exhibit an extended QT-RR contour. The 95% limits of normal activity are defined by the large, asymmetric dotted contour, and activity outside of this region can be considered abnormal.[3]

The standard QT-RR relationship for low heart rates (defined by the Fridericia correction factor $QTc = QT/RR^{1/3}$) is shown by the line cutting the phase plane from lower left to upper right. It can be seen that this factor, when applied to the

resting QT-RR interval relationship, overcorrects the dynamic responses in the normal range (illustrated by the striped area above the correction line and below the normal dynamic range) or underestimates QT prolongation at low heart rates. Many QT correction factors have been considered that improve upon Bazett's formula ($QTc = QT/\sqrt{RR}$), including linear regression fitting ($QTc = QT + 0.154(1 - RR)$), which works well at high heart rates, and the Fridericia correction ($QTc = QT/RR^{1/3}$), which works well at low heart rates.

Y axis variation-300-550/X axis variation-400-2000

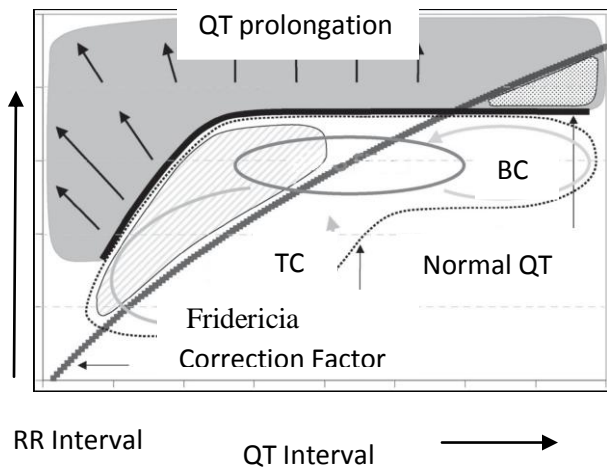


Figure-3.1 Normal dynamic QT-RR relationship

VI. Material and methods used:

ECG Images of normal and different types of infraction are used from standard MIT-BIH database. The duration of signals used for computing is for 120 sec. which is sufficiently large to ensure accuracy of results.

It is essential to convert the images to signals to ascertain the accuracy of the result. [6] This can be done by the implementing gray level slicing and then boundary extraction algorithm. The extracted boundary can be used as signal fit to use for applying further cross correlation and auto use for applying further cross correlation and auto correlation techniques. [7]

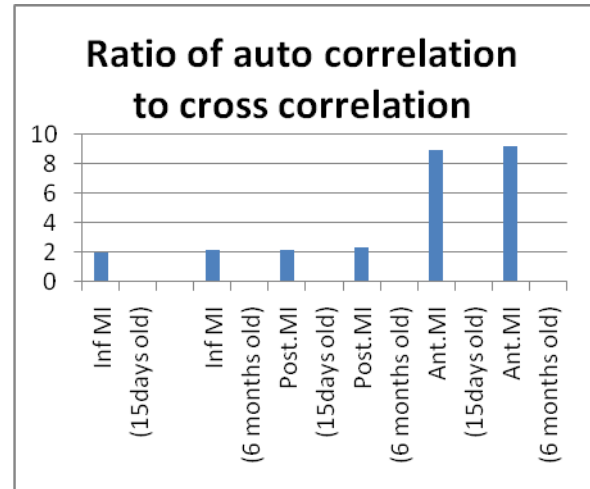
Type of specimen	Auto correlation	Cross correlation	Ratio of auto correlation to Cross correlation
Normal	1.4702e+10	-	-
Inferior Myocardial infarction (2 week old)	1.4310e+10	0.73774e+10	1.94
Inferior Myocardial infarction (12weekold)	1.3831e+10	0.66670e+10	2.07
Posterior Myocardial infarction (2week old)	1.3821e+10	0.6658e+10	2.08
Posterior Myocardial infarction (12weekold)	1.0188e+10	0.45242e+10	2.25
Anterior Myocardial infarction (2week old)	2.6013e+10	0.29013e+10	8.97
Anterior Myocardial infarction (12weekold)	2.2013e+10	0.24013e+10	9.17

The resulting summation values for autocorrelation and cross correlation can be used as confirmative parameters to diagnose the disease.

For calculation of HRV indices, ECG records are taken from MIT-BIH database and the average QT-RR interval is computed from the normal and patient suffering from infarction, ischemia at different wall locations are compared with that of the reference values given in figure 3.1. The ratio

of average QT- RR variation is computed to be 0.2999.

Table 1



VII. Results and Discussion

It is evident from table-1 and also graph-1 that the cross correlation values are at least half that of the autocorrelation. It is a confirmative positive diagnostic indication that proves the ECG period changes are prevalent over a sufficiently long period of time. Also the autocorrelation values are found to be higher for the disease that is 15 days old compared to that if the disease is six months old. This proves the fact that longer the duration of myocardial infarction higher are the ratio indices.

It has also been evident from the table that values of anterior infarction are much higher than the other diseases. This is purely incidental as we have taken the specimen of one patient.

The reduced HRV indicative indices are computed from the RR-QT ratios as shown in table2. The computed values of HRV indices from the theoretical are consistent with the values computed from MIT-BIH database. The

index for normal condition shows 0.3 and that for disease condition shows around 4.0. The computed ratios are observed to be in the same range.

Patient Details	disease	QT in msec (Avg)	RR in msec (Avg)	QT-RR Ratio (Avg)
Age-36 Male	Normal	32	96	0.333
Age 55 Male Acute chest Pain	Acute Inferior Myocardial Infarction	56	13.6	0.411
Age-63 Female 10 hour chest Pain, Sweat	Acute Anterior Myocardial Infarction	40	96	0.416
Age-60 Female 3 hour Chest Pain	Acute Posterior Myocardial Infarction	40	64	0.625
Age-53 Male	Ischemic heart disease	44	100	0.44

Table 2

VIII. Further scope

More work can be done by taking more samples and results and discussion can be further more confirmative. Also it is possible to explore more by taking different time interval windows of myocardial infarction. More analysis is also possible if one takes ECGs specific to one disease and actual patient data.

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