HEART DEPOLARIZATION VECTOR LOCUS CARDIOGRAM AND ITS CLINICAL DIAGNOSTIC APPLICATIONS

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This study demonstrates the development of the heart depolarization vector locus cardiogram (HPVLC, from limb leads and a modified Einthoven’s triangle) as a diagnostic measure of the left ventricular depolarization strength. Our work involves the reconstruction of the “equivalent heart electrical-activity vector (HAV)” for the QRS complex from limb leads voltages of a sample ECG recording, and plotting the progression of the cardiac vector during the QRS complex.

A realistic visualization of the progression of the equivalent-dipole HAV during the QRS complex is possible by staging the HPVLC of the QRS complex from the onset of the QRS till the end of the depolarization stage. This can enable the characterization of the HPVLC by means of an analytical function. By studying the HPVLC for various electro-cardiological disorders, it is possible to determine the ranges of the analytical function’s parameters for normal and disordered electro-cardiological states, for diagnostic purpose.

We have seen that the monitored ECG is theoretically derived from HAV components on the sides of the Einthoven triangle. Nevertheless, in cardiac practice, the monitored ECG is employed in diagnosis of heart diseases. From the ECG, we can obtain the heart rate, and therefrom the heart rate variability, which too has diagnostic applications. Many nonlinear methods have been proposed to analyze ECG and HRV for detection of cardiac abnormalities, using linear and nonlinear methods. Herein, we have shown how HRV signal can be analyzed in terms of four recurrence quantification analysis (RQA) features, which are then combined into an Integrated Index to enable better separation of normal and diabetic subjects.

**Keywords:** Heart vector; depolarization; QRS complex.
1. Introduction

According to the centric dipole assumption, the electrical activity of the heart (as sensed on the torso) can be represented by a single lumped dipole moment located at the center of the torso.\(^1\) For the depolarization (activation) of cardiac tissue, a double layer appears at the cardiac depolarization wave front, with the dipole orientation in the direction of propagation. The dipole consists of two monopoles of opposite sign but equal strength \(I_0\) (often termed source and sink) separated by a very small distance, \(b\). The quantity \(p\) is the dipole moment or dipole magnitude. The dipole is a vector whose direction is defined from the negative point source to the positive. A dipole of \(x\)-axis orientation is illustrated in Fig. 1, where the coordinate system origin is placed at the negative pole. The equation of extra-cellular current is given in Ref. 2 as:

\[
dI_o = \pi a^2 \sigma_i \frac{\partial^2 V_i}{\partial x^2} dx, \tag{1}
\]

where \(\sigma_i\) = conductivity of intra-cellular medium. The potential at point \(O\), due to the current source \(dI_o\), is \(dV_o(r_o)\) and is given by:

\[
dV_o(r) = \frac{dI_o}{4\pi \sigma_{co} r_x}, \tag{2}
\]

where \(\sigma_{co}\) = myocardial conductivity = 2 sm\(^{-1}\).

![Fig. 1. Dipole consisting of a sink \(-I_0\) at origin and a source \(I_0\) at radius vector \(x\), shown with respect to action potential. Also illustrated is a potential point at radius vector \(r\) and polar angle \(\alpha\).](image-url)
We use the definition of the electric field measured at point O, as given in Ref. 1, to define the total potential at point O due to a continuous leakage of current along the length of the dipole from the negative point source to the positive,\(^3\) as:

\[
V_o(r) = \frac{P \cos \alpha}{4\pi \sigma \omega r^2} = \frac{p \cdot r}{4\pi \sigma \omega r^3},
\]

wherein we define the magnitude \((P)\) of the electrical activity vector \((p)\) as \(P = \pi a^2 \sigma_{el} \Delta V\).

### 2. Modified Einthoven Triangle

We will now demonstrate how the 3-lead ECG voltages are derived from the projection of the equivalent-dipole heart electrical-activity vector (HAV) on the sides of the Einthoven triangle.\(^3\)

In the Einthoven electrocardiographic model, the cardiac source is a two-dimensional dipole in a fixed location on the frontal plane. The modified Einthoven Triangle is not an equilateral triangle as defined in the standard Einthoven triangle, but defined by taking the actual physical dimensional angles between the bipolar leads. It is assumed that the cardiac sources are represented by the equivalent-dipole HAV, located at the circumcenter O, as shown in Fig. 2.

![Modified Einthoven’s triangle](image)
The equation of potential difference between two points on the torso, in terms of the equivalent-dipole HAV, \( \text{OP} \), is as follows:

\[
V(r_2) - V(r_1) = \frac{\text{OP} \cdot R}{4\pi \sigma_\text{co} r^3},
\]

where \( R = r_2 - r_1 \).

The potential differences \( \text{A-B}, \text{A-C}, \text{and B-C} \) are considered to be the components of the HAV (along \( \text{AB}, \text{AC}, \text{and BC} \)), as shown in Fig. 2. The three bipolar lead voltages are expressed as the projections of the heart vector (\( \text{OP} \)) onto each side of the Einthoven triangle.\(^1\) Using (3), we can put down:

\[
\text{lead}\, I = V_I = V_B - V_A = \frac{P_{rAB} \cos \theta}{D} = \frac{P_x r_{AB}}{D},
\]

where (i) \( P \) is the magnitude of the cardiac vector \( \text{OP} \), (ii) \( P_x \) is the magnitude of the component of \( \text{OP} \) vector along the \( x \) axis (or along \( \text{AB} \)), (iii) \( r_{AB} \) is the length of side \( \text{AB} \) of the modified Einthoven triangle, (iv) \( D \) equals to \( 4\pi \sigma_\text{co} r^3 \), with the myocardial conductivity (\( \sigma_\text{co} \)) assumed to be 0.2 \( \text{Sm}^{-1} \), and (v) \( P(= \pi a^2 \sigma_\text{ci} \Delta V_i) \), with the intra-cellular medium conductivity \( \sigma_\text{ci} = 2 \text{Sm}^{-1} \).

\[
\text{lead}\, II = V_{II} = V_C - V_A = \frac{P_{rAC} \cos(\theta - 60^\circ)}{D} = \frac{(P_x \cos 60^\circ + P_z \sin 60^\circ) r_{AC}}{D},
\]

where (i) \( P_x \) and \( P_z \) are the magnitudes of the \( x \)-axis and \( z \)-axis components of the \( \text{OP} \) vector, (ii) \( r_{AC} \) is the length of the side \( \text{AC} \) of the modified Einthoven triangle, and (iii) \( \theta \), measured clockwise by convention from the horizontal axis, defines the \textit{instantaneous electrical axis} of the heart.

\[
\text{lead}\, III = V_{III} = V_C - V_B = \frac{P_{rBC} \cos(100^\circ - \theta)}{D} = \frac{(P_x \cos 100^\circ + P_y \sin 100^\circ) r_{BC}}{D},
\]

where (i) \( P \) is the magnitude of the cardiac vector \( \text{OP} \), (ii) \( P_x \) and \( P_y \) are the \( x \)-axis and \( y \)-axis components of the \( \text{OP} \) vector, (iii) \( \theta \), measured clockwise by convention from the horizontal axis, is defined as the \textit{instantaneous electrical axis} of the heart, and (iv) \( D \) equals to \( 4\pi \sigma_\text{co} r^3 \).

So, the three bipolar lead ECG voltages (as depicted in Fig. 3 theoretically represent the three projections of the heart vector onto each side of the Einthoven triangle. Conversely, since the three lead voltages can be monitored, they can be employed to reconstruct the HAV, from which we can then generate the vector cardiogram, as shown in the next section.
3. Reconstruction of HAV (From Limb Leads) and Heart Depolarization Vector Cardiogram

A simplified approach is used for the inverse reconstruction of the HAV OP from any two of the bipolar leads. Using lead I and lead II and Eqs. (5)–(7), we can determine the magnitude and direction of the HAV OP, in terms of its x and y components $P_x$ and $P_y$ given by:

$$
P_x = \frac{V_I D}{r_{AB}}
$$
$$
P_y = \left(\frac{V_{II} D}{r_{AC}} - P_x \cos 60^\circ\right) / \sin 60^\circ
$$
$$
\theta = \tan^{-1}\left(\frac{P_y}{P_x}\right)
$$

Now, by using values of lead I and II voltages, obtained from an ECG sample for Lead I, II, and III provided in Fig. 3 for the QRS complex (and tabulated in Table 1), we can determine $P_x$ and $P_y$, and hence the magnitude and direction of the HAV OP.

The HAV OP is now plotted on a plane representing the frontal plane of the torso, as depicted in Fig. 4. The line drawn by the tip of the HAV OP (derived from Leads I and II), as it traces the path of the depolarization electrical vector, during the progression of QRS complex, is the front-plane heart depolarization vector locus.

Fig. 3. Sample ECG recording used for sampling of lead I, II, and III voltages during QRS complex.
cardiogram (HPVLC), as it traces the path of the depolarization electrical vector during the progression of QRS complex. Thus, the HPVLC is actually a loop, with initial and terminal points at the origin (equivalent to the iso-electric baseline).

4. Results: Progression of the Vector — Locus Cardiogram During a QRS Complex

A realistic progression of the equivalent-dipole HAV during the QRS complex can be visualized by staging the HPVLC of the QRS complex from the onset of the QRS until the end of the depolarization stage.

Figure 5(a) is depicting the HPVLC during ventricular activation, when electrical impulses are first conducted down the left and right bundle branches on either side the septum. This causes the septum to depolarize from left-to-right as depicted by

<table>
<thead>
<tr>
<th>Point</th>
<th>$V_I$ (mV)</th>
<th>$V_{II}$ (mV)</th>
<th>$V_{III}$ (mV)</th>
<th>$P_x$</th>
<th>$P_y$</th>
<th>$P$ (mV)</th>
<th>$\theta$ (deg)</th>
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<tr>
<td>1</td>
<td>0</td>
<td>0.01</td>
<td>0.01</td>
<td>0.00E+00</td>
<td>-4.83E-05</td>
<td>4.83E-05</td>
<td>-89.99</td>
</tr>
<tr>
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<td>-0.2</td>
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<td>-0.25</td>
<td>-4.69E-05</td>
<td>-6.57E-05</td>
<td>8.07E-05</td>
<td>-125.48</td>
</tr>
<tr>
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<td>3.0</td>
<td>2.20</td>
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<td>1.18E-04</td>
<td>2.22E-04</td>
<td>32.16</td>
</tr>
<tr>
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<td>-0.1</td>
<td>-0.25</td>
<td>-0.15</td>
<td>-2.34E-05</td>
<td>-7.73E-06</td>
<td>2.47E-05</td>
<td>-161.72</td>
</tr>
<tr>
<td>5</td>
<td>-0.1</td>
<td>-0.3</td>
<td>-0.2</td>
<td>-2.34E-05</td>
<td>-1.55E-05</td>
<td>2.81E-05</td>
<td>-146.56</td>
</tr>
</tbody>
</table>

Fig. 4. HPVLC for a QRS complex. The plot was done by using five separate readings at interval of 6 ms (in Table 1) during the progression of the QRS complex.
the heart electrical activity vector (HAV). This vector is heading away from the positive electrode and therefore will record a small negative deflection (Q wave of the QRS complex).

As electrical wave propagation proceeds along the Purkinje fibers to the inner walls of the ventricles, and ventricular depolarization starts first from the left side of the inter-ventricular septum. The resultant dipole from this septal activation points to the right, as can be seen from the left orientation of the path of the depolarization vector, in Fig. 5(b).

Depolarization then propagates through the walls of the ventricles. Because the left ventricle wall is thicker, activation of the left ventricular free wall continues even after depolarization of a large part of the right ventricle has depolarized. As seen in

Fig. 5. Illustration of the progression of the Vector — Locus Cardiogram during a QRS Complex.
Fig. 5. (Continued)
Fig. 5(c), the resultant depolarization vector reaches its maximum, and points towards the left in this phase, due to non compensating right depolarization.

Figure 5(d) shows that the depolarization front continues propagation along the left ventricular wall toward the back. Finally, the ventricular wall surface area decreases continuously and (as shown in Fig. 5(e)) the magnitude of the resultant vector decreases until the entire ventricular muscle is depolarized.

By characterizing this HAV tip-locus (HAV-TL) curve by an analytical function of a known curve (such as a limaçon), we can determine the parameters (e.g., $p_1$ and $p_2$ for a limaçon) of the curve. By studying the HAV-TL for various electro-cardiological disorders, we can determine the ranges of the HAV-TL parameters for normal and disordered electro-cardiological states. This can facilitate the assessment of intra-myocardial depolarization activity, in terms of the values of the two parameters, plotted as points on the $(p_1, p_2)$ plane. On this plane, we can designate the zones of normal and diseased states for diagnostic usage.

5. Electrocardiography Employment in Cardiac Diagnosis

5.1. Using ECG signal parameters for diagnosis

One approach is to designate the parameters of the P-QRS-T complex and associate the various cardiac disorders with abnormalities of the P-QRS-T complex. This approach is depicted in our other paper in this issue on ECG Signal Generation and Heart Rate Variability (HRV) Signal Extraction: Signal Processing, Features Detection, and their Correlation with Cardiac Diseases.

For this purpose, the noise in the original ECG signals can be eliminated by passing through a low-pass filter of 15 Hz cut-off frequency. Baseline wander present in the ECG signal can be removed by passing the signal through a high-pass filter of cut-off frequency of 0.3 Hz. Then the 50 Hz band-reject filter may be used to remove the power-line interference noise. Median filter can be used to eliminate the baseline wander of the processed ECG signal. Finally, the $R$ peaks of ECG signal can be detected. RR interval is defined as the interval between two successive QRS complexes. Heart rate (HR) is the time series of unevenly spaced RR intervals; HR is $60/(R-R \text{ interval})$.

5.2. HRV signal analysis and diagnostic methods

Yet another approach for directly employing the ECG signal is to extract the HRV signal from the ECG signal, and employ it as the base signal to observe the functioning of the heart. The HRV signal is non-linear and non-stationary. Hence, in our other paper in this issue on HRV Signal Features and Automated Detection for Cardiac Disease Diagnosis using Recurrence Quantification Analysis, Recurrence Quantification Analysis (RQA) has been used to extract the important features from the HR signals. These features are fed to the Fuzzy, Gaussian Mixture Model (GMM) and Probabilistic Neural Network (PNN) classifiers for automated classification of...
cardiac bio-electrical contractile disorders. This approach is also developed in Sec. 4, on HRV Analysis in Cardiac Diagnosis.

### 5.3. Heart-depolarization vector cardiograms for distinguishing cardiac disorders

A third approach for electrocardiography based cardiac diagnosis is to (i) reconstruct the HAV at each instant from the ECG recordings (as shown in Fig. 4), (ii) plot the HDVC, and (iii) associate its shape with cardiac disorders, as depicted below.\(^3\)

So for this purpose, ECG recordings from the PTB\(^a\) Arrhythmia database have been used for the reconstruction of the HAV and the plotting of the HDVC. The sampling frequency is 1000 Hz with resolution 0.5 \(\mu\)V/bit, and 10s of each ECG recording was downloaded. The lead I and II voltages were normalized to the isoelectric baseline (0 V).

In Fig. 6, plots of the Heart-Depolarization Vector Cardiogram (HDVC) on the frontal plane are provided for a healthy control subject (Fig. 6(a)) and one for each abnormal electro-cardiological state in Figs. 6(b)–6(d).

The start of the Vector Cardiogram is assumed to be the first departure of either Lead I or II voltages from the isoelectric baseline. The end of Vector Cardiogram is assumed to be the return of Lead I or II voltages to the isoelectric baseline after \(S\) wave, whichever is later. Since the \(T\) wave can be negative for disordered electro-cardiological states and the \(ST\) segment can be depressed in ischemia, the return to isoelectric baseline for Vector Cardiogram of diseased states could be after \(ST\) segment. This is especially so for hypertrophy.

![Normalized Lead I and II ECGs and their corresponding HDVC for healthy controls and abnormal electro-cardiological states.](image)

(a)

**Fig. 6.** Normalized Lead I and II ECGs and their corresponding HDVC for healthy controls and abnormal electro-cardiological states. (a) Healthy electro-cardiological state (patient 105), (b) Ventricular Hypertrophy (patient 159), (b) Bundle branch block (patient 202), (c) Inferior Myocardial Infarction (patient 11).
Fig. 6. (Continued)
For diagnostic purpose, it is required to (i) determine the shape functions of these HDVC plots and their parameters, and then characterize the cardiac disorders in terms of these parameters.

6. HRV Analysis in Cardiac Diagnosis

HRV is a reliable and powerful tool for the assessment of sympathetic and parasympathetic functions of the autonomic system. Hence, HRV is widely used as a tool to monitor post myocardial-infarction patients and also diabetes subjects, because (as a chronic side effect) diabetes affects the peripheral and autonomous nervous system.

Since HRV is an important clinical sign of many cardiac diseases (including diabetes), it is important to understand the intrinsic mechanisms which are involved in its generation and perturbation. We have developed a biophysical model of HRV, based on neuroanatomical data about electrophysico-chemical mechanisms of sinoatrial node’s bioelectrical activity, involved in regulating heart-rate activity in healthy and diabetic subjects. We have numerically simulated the heart-rate variability phenomenon in normal and diabetic states (based on the known physiological and biophysical concepts about cardiac activity). Our model combines the analysis of the regulatory elements of the nervous system and of the pacemaker electrical activity of the sinoatrial node of the heart as illustrated below in Fig. 7.

We will now present a summary of how HRV can be analyzed diagnostically.

Figure 8 presents a system for the automated diagnosis of cardiac state using HRV signals. Firstly, the HR is evaluated by employing ECG signals using the Pan-Tompkins method. Thereafter, various linear and non-linear features are to be extracted from these signals. Then, clinically significant features are selected, by

![Fig. 7. Schematic representation of an idealized adrenergic neuron, synapse, and SA node cell with membrane currents involved.](image-url)
using ANOVA (Analysis of Variance) test. These selected features are fed to the classifiers for automated classification.

HRV is used as a base signal in this analysis. For signal analysis and feature extraction, continuous wavelet transform and fractal dimension are used to differentiate normal state from sick sinus syndrome, complete heart block, preventricular contraction, atrial fibrillation, and ischemic/dilated cardiomyopathy cardiac classes. Also, scalogram plots have been made for these cardiac classes, for differentiating cardiac diseases by (i) using detrended fluctuation analysis and correlation dimension and (ii) designating specific ranges for these cardiac classes. Then some unique ranges of values have been proposed for all the linear and non-linear features for these classes, along with unique phase space plots. In this way, nine cardiac abnormalities have been categorized by using various modeling techniques. The results show that the Auto Regressive Moving Average (ARMA) is superior compared to auto regressive (AR) and moving average (MA) methods. ARMA was able to classify the classes with an average accuracy of 83.83%, sensitivity of 81.72%, and specificity of 100% correctly.

An adaptive neuro-fuzzy network has also been used to classify heart abnormalities in 10 different cardiac states in Ref. 11. They have shown that this classifier was able to correctly classify to the tune of about 85%–100% accuracy. Recently, the spatial filling index and time-frequency analysis of HRV signal has been proposed for disease identification in Ref. 12. For detecting cardiac dysfunction, phase space and the time-frequency analysis of these cardiac signals, using spatial filling index and Renyi’s entropy, has been employed. Higher order spectra methods have also been used for analysis of HR signals for the detection of epilepsy, sleep stages, and cardiac disease identification.

6.1. Developing an integrated index for diabetes detection:

Data acquisition

ECG signals of 15 diabetic and 15 non-diabetic volunteers were obtained by using BIOPACTM equipment at Kasturba Medical Hospital, Manipal, India. The diabetic volunteers consisted of 8 males and 7 females while the non-diabetic volunteers consisted of 10 males and 5 females. From these ECG signals, 82 non-diabetic and 80 diabetic datasets were obtained at 15 min intervals, with the volunteers relaxed in supine position.

There from, 1,000 samples were recorded for each dataset with a sampling rate of 500 Hz. The volunteers under study were between 50 and 70 years of age (Mean ± standard deviation = 58.5 ± 6.42 years) and had diabetes between 5 to 15 years. The non-diabetic volunteers under study were between 40 to 60 years of age.
(Mean ± standard deviation = 50 ± 8.8 years). The Kasturba Medical Hospital ethics committee, consisting of senior doctors, approved for this data to be used in this research.

6.2. Data Processing

The raw data was pre-processed as follows:

(i) Low pass filtering of cut-off frequency 35 Hz, to remove unwanted high frequencies.
(ii) High pass filtering of cut-off frequency 0.3 Hz, to suppress baseline wander.
(iii) Notch filtering of cut-off frequency 50 Hz, to suppress power line interference noise.
(iv) Median filtering, to eliminate baseline wander.

To detect the R peaks of the signal, Tompkins’s algorithm was used. [Tompkin’s et al. 1985] The output was the primary data used in this research. Figures 9 and 10 show the processed HR signals of a non-diabetic subject and a diabetic patient, respectively.

Fig. 9. HRV signal of a non-diabetic subject.

Fig. 10. HRV signal of a diabetic patient.
6.3. Methodology

RQA quantifies the number and duration of recurrences of a dynamical system presented by its phase space trajectory. The RQA was developed to quantify differently appearing recurrence plots (RP)\textsuperscript{17} based on small-scale structures. The RPs are tools which visualize the recurrence behavior of the phase space trajectory of dynamical systems. They mostly contain single dots and lines, which are parallel to and perpendicular to the line of identity (LOI).

Lines parallel to the LOI are referred to as diagonal lines and the vertical structures are referred to as vertical lines. Because RP is usually symmetrical, horizontal and vertical lines correspond to each other, and hence only vertical lines are considered. The lines correspond to a typical behavior of the phase space trajectory, whereas, the diagonal lines represent segments which remain in the same phase space region for sometimes. Figures\textsuperscript{11} and \textsuperscript{12} show the RPs of a non-diabetic subject and a diabetic patient, respectively.

For normal subjects, the plot has a diagonal line and fewer squares, indicating more variation in the HR. In the case of abnormalities, a higher number of squares appears on the plot, indicating the inherent periodicity and lower HRV. Therefore, the RP of a diabetic subject has a more uniform pattern than the normal RP has.\textsuperscript{18,19}

Fig. 11. RP of a non-diabetic.
Four RQA features were evaluated and used in the formulation of the Integrated Index. They are as follows:

(i) Determinism (DET) is the percentage of recurrence points, which, diagonal lines in the recurrence plot are of minimal length. DET is given by:

$$\text{DET} = \frac{\sum_{l=l_{\text{min}}}^{N} \ell P(\ell)}{\sum_{i,j=1}^{N} R(i,j)},$$

where, $P(l)$ is the frequency distribution of the lengths ($l$) of the diagonal lines. DET is related with the predictability of a dynamical system.

(ii) Laminarity (LAM) is the amount of recurrence points which form the vertical lines. LAM is given by:

$$\text{LAM} = \frac{\sum_{v=v_{\text{min}}}^{N} v P(v)}{\sum_{v=-1}^{N} P(v)},$$

where, $P(v)$ is the frequency distribution of the lengths ($v$) vertical lines which have at least a length of $v_{\text{min}}$. LAM is related with the amount of laminar phases (intermittency) in a system.
(iii) $T_1$ is the recurrence time of the first type.
(iv) $T_2$ is the recurrence time of the second type.

6.4. **Integrated index**

The four features mentioned above are used to formulate the Integrated Index as follows:

$$\text{Integrated Index} = 40 \times (\text{LAM} - \text{DET}) + (T_2 - T_1)$$

The mean and standard deviation values for this Integrated Index is shown in Table 2.

The Integrated Index diagrammatic scale is shown in Fig. 13. We can see therein a distinct separation of non-diabetic and diabetic classes of subjects.

<table>
<thead>
<tr>
<th>DII</th>
<th>Mean</th>
<th>SD(+-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Diabetic</td>
<td>4.14</td>
<td>0.7</td>
</tr>
<tr>
<td>Diabetic</td>
<td>7.3</td>
<td>0.97</td>
</tr>
</tbody>
</table>

Table 2. Mean and standard deviation of non-diabetics and diabetics for the integrated index.

Fig. 13. Diagrammatic representation of index scale.
The scale shows no data overlap boundary of value 1.5 between the two classes. This is significant and important as the accuracy of the Integrated Index was proved.

These HR signals and patient information can be interleaved within the images with the different error correcting codes in a noisy environment without affecting the hidden information.\textsuperscript{20,21}

6.5. Conclusion

The aim of the Integrated index is to create an accurate diagnosis for diabetes, which we have achieved in doing, as seen in Fig. 13. If the value of an Index falls in a boundary where the scales of two classes overlap, then diagnosis of the patient cannot be made. The no data overlap boundary between the two classes means that this diagnosis issue can be successfully achieved.

However, improper feature extraction, instrumentation or human error can result in an Index value to not lie within the boundary limits of either of the two classes. Prompt rectification of respective errors should be performed to eliminate such issues.

As the Integrated Index is a non-invasive and fast technique, it can free clinicians for other tasks and can be a time-efficient and popular diagnostic tool for clinicians.

7. Discussion: ECG Based HAV Cardiogram and Its use in Cardiac Diagnosis and HRV Analysis Based Integrated Index for Diabetes Detection

In this paper, we have provided the theoretical basis of the modified Einthoven Triangle and shown the progression of the cardiac electrical activity vector developed from thereon. Herein, we have also discussed the diagnostic value of the heart depolarization vector locus cardiogram. Further, we are in the process of employing mathematical methods to fit the vector cardiograms with suitable parametric equations, so that we can then use these parameters to designate the zones of normal and diseased states for diagnostic usage.

We have seen that the monitored ECG is theoretically derived from HAV components on the sides of the Einthoven triangle. Nevertheless, in cardiac practice, the monitored ECG is employed in diagnosis of heart diseases. From the ECG, we can obtain the heart rate, and therefrom the heart rate variability, which too has diagnostic applications.

Many nonlinear methods have been proposed to analyze ECG and HRV for detection of cardiac abnormalities, using linear and nonlinear methods in Refs. 7–10. Herein in Sec. 4, we have shown how HRV signal can be analyzed in terms of four RQA features, which are then combined into an Integrated Index to enable better separation of normal and diabetic subjects.
References