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**Keywords**

Electrocardiogram (ECG) - Dipole - Vectorcardiogram - QRS complex - Heart electrical activity vector - VCG loops - Einthoven triangle - Visualization

**Foot note**

information
Frontal Plane Vectorcardiograms: Theory and Graphics
Visualization of Cardiac Health Status

Dhanjoo N. Ghista · Rajendra U. Acharya · Nagenthiran Nagenthiran

Abstract The electrocardiogram (ECG) is a representative signal containing information about the condition of the heart. The shape and size of the P-QRS-T wave, the time intervals between its various peaks, may contain useful information about the nature of disease afflicting the heart. However, these subtle details cannot be directly monitored by the human observer. Besides, these signals are highly subjective, and the symptoms may appear at random in the time scale. It is very taxing and time-consuming to decipher cardiac abnormalities based on these ECG signals. The Vectorcardiogram (VCG) is the vector loop in the 2-D frontal plane, indicating the magnitude and direction of the instantaneous heart electrical activity vector (HAV), which represents the sum of the dipole vectors located along the instantaneous depolarization wavefront. The HAV is constructed from the monitored 3-lead ECG signals, placed at the three vertices of the modified Einthoven triangle formed by the 3-lead system in the frontal plane of the torso. The VCG examines the electrical activities within the heart, using the ECG signals along the three sides of the modified Einthoven triangle, and displays electrical events in the 2-dimensional frontal plane. This study demonstrates the development of the heart-depolarisation vector-locus cardiogram (using modified Einthoven's triangle), as a diagnostic measure of the left ventricular depolarisation strength. Our work involves the reconstruction of the “equivalent heart vector” for the QRS complex from limb lead voltages of a sample ECG, and plotting the progression of the cardiac vector during the QRS complex. We have demonstrated the construction of the frontal plane heart-depolarization vector cardiogram (HDVC), as the path of the locus of the tip of the heart electrical activity vector, with initial and terminal points at the origin. In this work, we have shown characteristic patterns of HDVC for cardiac states namely, normal, bundle branch block, ventricular hypertrophy and myocardial infarction. We have demonstrated how HDVC can be diagnostically employed to characterize cardiac disorders, such as ventricular hypertrophy bundle branch block and inferior myocardial infarction.

Keywords Electrocardiogram (ECG) · Dipole · Vectorcardiogram · QRS complex · Heart electrical activity vector · VCG loops · Einthoven triangle · Visualization

Introduction and scope

ECG, HAV & VCG

The Electrocardiogram is the electrical activity of the heart recorded from the body surface to evaluate rapidly the functioning of the heart. Pathological alterations observable by ECG are cardiac rhythm disturbances (or arrhythmia), dysfunction of myocardial blood perfusion (or cardiac ischemia) and chronic alteration of the biomechanical structure of the heart. Cardiac rhythm disturbances can lead to life threatening conditions. WHO in 2002 reported that, in the United States, diseases of the heart are the...
leading cause of death, causing a higher mortality than
cancer (malignant neoplasms) [20]. Coronary heart disease
is responsible for 1 in 5 deaths in the U.S. Thus the
detection of abnormalities in intensive care units is very
essential. Recently extensive research is being done to
detect the cardiac abnormalities automatically by applying
various bioengineering techniques. The continuous moni-
toring and automatic analysis of ECG in intensive care
units, will aid the clinical staff in the absence of the doctor.
The electrocardiogram (ECG) is a graphical recording of
the electrical signals generated by the heart as it contracts
and relaxes. Ions are exchanged in the process, resulting in
the progression of the depolarization wavefront within the
heart. Along a depolarization wavefront are located several
dipoles. Each dipole has a dipole vector associated with it,
representing the strength of the depolarization activity. The
resultant of these dipole vectors along a depolarization
wavefront constitutes the instantaneous dipole vector or the
heart electrical-activity vector (HAV). This HAV varies in
magnitude and direction during a cardiac cycle. The HAV
generates potential differences between points within the
heart as well between points in the torso. This information
can be captured by means of electrodes attached to the skin
at specific locations on the body, to detect this signal from 3
to 12 leads.

The gold standard for recording the electrical signals
from the heart (electrocardiogram or ECG) is the 12-lead
ECG. However, even though both the 3-lead and 12-lead
ECG contain a lot of diagnostic information derived from
evidence-based medicine, these ECG signals essentially
constitute 1-D time-varying potential signals, derived from
the 2-D heart electrical activity vector (HAV) located in the
frontal plane of the torso. However, the valuable informa-
tion of the strength and orientation of the intrinsic HAV is
lost in the process. This is the reason behind the generation
of HAV from the multi-lead ECG, and its employment in
the form of VCG, to yield more detailed and precise
information of heart diseases, as they influence the
generation and propagation of depolarization wavefront.

The bioelectrical state of cardiac health is generally
reflected in the shape of ECG signal waveforms. It may
contain important pointers to the nature of diseases
afflicting the heart. However, bio-signals being non-
stationary signals, this reflection may occur at random in
the time scale (that is, the disease symptoms may not show
up all the time, but many manifest at certain time periods
during the day). Therefore, for effective diagnostics, the
study of ECG pattern may have to be carried out over
several hours. Thus, the volume of the data being
enormous, the study is tedious and time consuming. This
is also why the VCG is more useful, because it provides a
visual representation of HAV variation during a cardiac
cycle.

VCG & construction

The HAV can be reconstructed from its component 1-D
signals (monitored by electrodes attached to the skin) by
means of the modified Einthoven triangle formed by the 3-
lead system in the frontal plane of the torso. This HAV
is assumed to be located at the centroid of the modified
Einthoven triangle (MET), and the monitored ECG poten-
tials at the 3 leads are taken to be located at the 3 vertices
of MET. These monitored potential differences between the 3
vertices are expressed in terms of the magnitudes of the HAV
components along the sides of the MET. The HAV is then
reconstructed from its components vectors along the sides of
MET. The reconstructed HAV (simulating the actual HAV) is
seen to vary in magnitude and direction during a cardiac
cycle. The tip of the HAV traces loops of varying shapes and
patterns, specific to heart normality and disease states.

The VCG is obtained by mapping the reconstructed
instantaneous HAV vectors in the 2-D frontal plane. The
tips of these vectors form a loop, which is designated as the
VCG loop. The geometries of these VCG loops, in the 2-D
frontal coordinate plane, are specific to cardiac diseases.
Hence by means of measures to depict and quantify these
VCG loops, we can detect heart diseases. The contours of the
HAV tip loops are important aspects of VCG diagnostic
criteria, that have no direct counterpart in the 3-lead ECG or
even in the 12-lead (ECG). Therefore, attempts have been
made to synthesize VCGs from 12-lead ECGs for diagnostic
purposes [14]. Current uses of vectorcardiograms have been
mainly in the diagnostic classification of bundle branch
blocks and myocardial infarction. VCG has also been
recently used in the assessment of left ventricular size [39].

Nowadays, the vectorcardiogram (VCG) is being increas-
ingly used in cardiology, as it provides additional informa-
tion over the conventional ECG in the form of spatial
information of HAV. In this paper, we will develop the theory
and generation of VCG closed-contours in the frontal plane,
derived from the QRS complex of ECG. The VCG provides
two-dimensional contours of the two-dimensional HAV
(heart electrical activity vector) in the frontal plane. Hence
these VCG contours (of the loop generated by the tip of
HAV) depict the origin and pathways of propagation of the
electrical impulses in the heart. These loops have a unique
shape for each type of cardiac disease [34]. VCGs have also
been used as classifiers to detect the cardiac failure or
severe pulmonary disease, depending on the QRS config-
uration in the transverse plane [16].

Diagnostics based on VCG

Presently, vectorcardiograms are used in the identification
of myocardial infarction. They have also been recently used
in the evaluation of myocardial infarct size [41] and in the
assessment of right ventricular overload in patients with chronic pulmonary disease [25]. The VCG showed more sensitivity than the ECG for the diagnosis of multiple infarctions, associated with left anterior fascicular block (LAFB) [5]. The vectorcardiogram was more useful in the diagnosis of Brugada syndrome [3]. The VCG showed higher diagnostic sensitivity as compared to the ECG in acute myocardial infarction (AMI), with left anterior fascicular block (LAFB) [23].

In many cardiac patients, ECG quality and/or scan efficiency was reduced due to imprecise R-wave ability to trigger the scan due to noise on the electrocardiogram (ECG) caused by the magnetic resonance (MR) environment [8]. Triggering system using spatial information of the vectorcardiogram (VCG) was used to minimize the effects of MR-related noise on triggering. The subtle morphologic variability in the electrocardiogram (ECG)/vectorcardiogram (VCG) is also complicated by the presence of respiration and muscular activity noises. A method was recently presented to reduce the influence of such noise by performing spatial and temporal alignment of VCG loops [2]. The alignment was performed in terms of scaling, rotation and time synchronization of the loops.

A Vectorcardiographic lead system was designed for the analysis of atrial fibrillation (AF) [38]. They have shown that the Frank lead system was suboptimal for estimating the equivalent dipole components (VCG) during AF, and the alternate electrode configuration should include at least one electrode on the back. Characteristic vectorcardiographic patterns in patients with bundle-branch block and acute myocardial infarction were produced by [15]. Their pattern was able to diagnose the acute myocardial infarction in the presence of left bundle-branch block with an accuracy of 71%.

Diagnostic methods have also been created by using vectorcardiogram orientations, such as the spatial direction of the vector of the QRS loop having the greatest magnitude, and the spatial direction of the vector of the T loop having the greatest magnitude [13]. Other VCG variables used for diagnostic purposes are (i) the QRS vector difference, which reflects changes in the shape of the QRS complex, and (ii) the ST vector magnitude, representing the deflection of the ST segment from the isoelectric level [27].

Myocardial infarctions have been analyzed, using the following parameters: (i) the location of the initial 20 msec QRS vector, (ii) the amplitude and duration of the initial rightward, anterior and superior QRS vectors, (iii) the magnitude of the maximal leftward deviation of the initial superior QRS vectors, and (iv) the maximal anterior QRS accession time [31]. In another study involving old inferior myocardial infarctions [42], the leftward and inferior frontal plane QRS vectors were chosen for analysis, as these vectors showed the least variation in normal subjects and were suitable for patients with inferior infarction.

Commonly used methods for the derivation of the VCG are the Dower inverse transform [12] and singular value decomposition (SVD) [32]. The Dower inverse transform computes Frank leads for the 12 lead ECG, and SVD derives the VCG to represent the ECG on its three axes. The usefulness of conventional vectorcardiography (VCG) and computerized analysis of spatial VCG changes for diagnosis of perioperative myocardial infarction were studied by [11], and implemented for the interpretation of VCG after the cardiac surgery.

An algorithm to visualize the cardiac diseases based on the VCG was proposed by Chee et al. (7). In this work, the features like PR interval, QRS width and ST interval are extracted from the magnitude and phase plot of different lead combinations. These features are displayed on a Cartesian quadrant as different curves, with a menu driven display strategy to visualize the ECG for a chosen interval.

Cardiac conditions studied in this work

In this work, we have characterized VCG patterns for four types of cardiac signals, which are briefly explained below.

Types of cardiac conditions

i) Normal Sinus Rhythm (NSR) is characterized by a heart rate of 60 to 100 beats per minute and varies with the breathing. The range of PR interval, QRS width and P wave amplitude are (0.12–0.21) second, (0.06–0.08) second and (2.0–2.5) millimeter respectively.

ii) Bundle Branch Block (BBB) is partial or complete blockage to the conduction of the electrical impulses through the left or right bundle branches. The duration of the QRS complex on the ECG usually exceeds 120 ms [24, 36].

iii) Left Ventricular Hypertrophy (LVH) occurs due to the heart working harder than the normal to pump the blood throughout the body. A tall R wave (greater than 25 mm in V5 or V6) and a deep S wave in V1 or V2 can be seen [24, 36], in the case of LVH.

iv) Myocardial infarction (MI) is a state of interruption of the blood supply to a part of the heart due to rupture of a vulnerable plaque. In inferior myocardial infarction, the cells die in the inferior wall of the thick muscle in the left ventricle. The common ECG features of MI are ST segment depression or T wave inversion [24, 36].

Feature of paper: VCG loops generated for detecting cardiac disorders

The action potential generates the electrical potential field, which can be measured on the surface of the body using scalar.
ECG. This ECG (scalar) represents the potential differences on the body surface versus the time. The conventional ECG has three main waves (P, QRS and T) [17]. They are as follows:

- **P wave**—indicates the spread of the stimulus through the atrium (atrial depolarization).
- **QRS complex**—represents the depolarization of the ventricular myocardium.
- **T wave**—corresponds to the repolarization of the ventricles.

The instantaneous cardiac dipole vector represents the cardiac electric generator. In VCG, the magnitude and direction of this HAV dipole vector (due to the heart’s electrical activity) is represented in the form of loops. We have reconstructed the HAV (or VCG vector) from the 3-lead ECG data, using the modified Einthoven triangle, MET. We have then plotted the instantaneous HAV in the 2-D coordinate frame of the frontal plane. The path traversed by this HAV represents the VCG, and indicates the details of the bioelectrical phenomena (such as depolarization and repolarization of the heart). Hence, the different cardiac abnormalities will have different shapes and different orientations in the 2-D frontal coordinate plane. The loop traced by the tip of this HAV in the 2-D frontal coordinate plane constitutes the VCG loop.

In our work, we have used only the QRS complex to plot the VCG. As the heart beats, the loop corresponding to the QRS complex is plotted. The shape, size and orientation of the loops, and their locations in the display quadrants of the loop are all useful indicators that can be used to determine any cardiac disorder.

### Methods

In this section, the data and methods used for the analysis are discussed in detail.

### Data used

- **ECG data for the analysis** was obtained from Physikalisch-Technische Bundesanstalt (PTB) arrhythmia database [22].
- **Prior to analysis,** the ECG signals were processed to remove noise due to power line interference, respiration, muscle tremors, spikes etc [1, 28, 37, 40]. The age and number of subjects chosen for each of the four classes of cardiac conditions (mentioned in "Diagnoses based on VCG") are given in Table 1. Each dataset consists of around 10,000 samples and the sampling frequency of the data is 1 kHz.

### Method used for generating the equivalent heart vector

In this section, the reconstruction of the “equivalent heart vector” from limb lead voltages for the ECG recording is explained and derived. The concepts of cardiac dipole, modified Einthoven’s triangle, and the reconstruction of the HAV from limb leads are explained below:

#### Cardiac dipole

In the cardiac depolarization (or activation) process, the cardiac depolarization wave front propagates across the conducting cardiac tissue medium. This depolarization wavefront can be deemed to consist of a double layer of a series of adjacent dipoles, oriented in the direction of the wave propagation.

In a conducting cardiac tissue unit or fiber (within this cardiac tissue medium), this depolarization wavefront can be represented by a collection of these adjacent dipoles (of all the cardiac tissues units or fibers along the wavefront), which in turn can be represented, (at any given time instant) as a single equivalent dipole (located across the wavefront) whose magnitude and direction varies throughout the heart cycle.

Thus according to the centric dipole concept, the electrical activity of the heart (as sensed by the three ECG leads on the torso, (forming the Einthoven triangle), can be represented (at any time instant) by a single lumped dipole moment located on the instantaneous depolarization wavefront in the heart [29]. The electrical field of the heart can then be studied as a field of this single dipole, termed here as the equivalent-dipole heart electrical-activity (HAV) vector. The curve traced by the tip of this vector during a cardiac cycle is the vector cardiogram (VCG).

In order to derive an expression for the generation of this VCG, let us consider a conducting cylindrical cardiac tissue fiber oriented along the x axis (as shown in Fig. 1). When the in vivo cardiac tissue is depolarized by the action potential \( V_0(x) \), the depolarization wavefront (caused by the action potential) can be represented by an equivalent dipole consisting of two monopoles of opposite but equal strength \( I_0 \) (often termed as source and sink) separated by the very small length (b) which is depolarized by the action potential.

Figure 1 illustrates the action potential \( V_0(x) \) and the associated dipole, whose negative pole is located at the origin and the positive pole is located along the x axis at a distance b from it. The dipole moment is the vector whose direction is defined from the negative point source to the positive, along the x-axis.

#### Table 1 Age and the number of subjects used in each class

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<th>Number of subjects</th>
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<td>Normal Sinus Rhythm</td>
<td>41.7±14.1</td>
<td>3F,8M</td>
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<td>Bundle Branch Block</td>
<td>53.25±16.4</td>
<td>2F,3M</td>
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<tr>
<td>Ventricular Hypertrophy</td>
<td>66.25±9.43</td>
<td>1F, 3M</td>
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<tr>
<td>Myocardial Infarction</td>
<td>58.6±11.4</td>
<td>5F, 10M</td>
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### Diagnostic class

- **Normal Sinus Rhythm**
- **Bundle Branch Block**
- **Ventricular Hypertrophy**
- **Myocardial Infarction**
Potential at a point exterior to the depolarized cardiac tissue

The current $I(x,t)$ flowing within this tissue cylinder is given (by Ohm’s law), as

$$I(x,t) = \frac{\Delta V_i}{R} = \frac{\partial V_i}{\partial x} \cdot \frac{R}{\sigma_{ci}}$$

Putting $R = \sigma_{ci}/\pi a^2$ (based on Ohm’s law), we get

$$I(x,t) = -\sigma_{ci} \frac{\partial V_i}{\partial x}$$

where $\sigma_{ci}$ = conductivity of the intra-cellular medium = 2 Sm$^{-1}$.

Then, the expression for the extra-cellular current or the transmembrane current $dI_o$ (leaking out through the membrane into the external medium) is given (as depicted in Fig. 2) by:

$$dI_o = I(x,t) - I(x+dx,t)$$

$$= \frac{\partial I}{\partial x} \cdot dx$$

$$= \pi a^2 \sigma_{ci} \frac{\partial V_i}{\partial x} \cdot dx$$

Using the definition of electrical potential at a point O (due to a small current source $dI_o$), the potential at point O ($r$) due to this small leakage current $dI_o$ is given by

$$dV_o(r) = \frac{dI_o}{4\pi \sigma_{co} r^2}$$

where $\sigma_{co}$ is the myocardial conductivity = 0.2 Sm$^{-1}$.

Combining Eqs. (3) and (4), we get

$$dV_o(r) = \frac{\pi a^2 \sigma_{ci}}{4\pi \sigma_{co} r^2} \frac{\partial^2 V_i}{\partial x^2} \cdot dx$$

$$= \frac{\pi a^2 \sigma_{ci}}{4\pi \sigma_{co} r^2} \frac{\partial^2 V_i}{\partial x^2} \cdot dx$$

The total potential at point O due to a continuous leakage of current along the length $b$ of the dipole (or the extent of the action potential) within the cylindrical cardiac tissue, from the negative point source to the positive) is obtained by integrating the contribution of all the elements of the current within the dipole:

$$V_o(x) = \frac{\pi a^2 \sigma_{ci}}{4\pi \sigma_{co}} \int_0^b \left( \frac{\partial^2 V_i}{\partial x^2} \right) \left( \frac{1}{r} \right) \cdot dx$$

With respect to Fig. 1, the distance of point O from the dipole segment $dx$ in terms of $x$ is given by

$$r_x = r - x \cos \alpha$$

Substituting Eq. (7) into (6), Eq. (6) becomes

$$V_o(x) = \frac{\pi a^2 \sigma_{ci}}{4\pi \sigma_{co}} \int_0^b \left( \frac{\partial^2 V_i}{\partial x^2} \right) \left( \frac{1}{r} \right) \cdot dx$$

Using integration by parts, Eq. (8) becomes

$$V_o(x) = \frac{\pi a^2 \sigma_{ci}}{4\pi \sigma_{co} r^2} \int_0^b \frac{\partial V_i}{\partial x} \cdot dx + \frac{\pi a^2 \sigma_{co} \cos \alpha}{4\pi \sigma_{co} r^2} \int_0^b \frac{\partial^2 V_i}{\partial x^2} \cdot dx$$

Since $\frac{\partial V_i}{\partial x} |_{x=0} = 0$ and $\frac{\partial V_i}{\partial x} |_{x=b} = 0$ (see Fig. 1), Eq. (9) is simplified to

$$V_o(x) = \frac{\pi a^2 \sigma_{ci} \cos \alpha}{4\pi \sigma_{co} r^2} \int_0^b V_i(0) - V_i(b)$$

Defining the magnitude ($P$) of the electrical activity vector ($p$) as $P = \pi a^2 \sigma_{ci} \Delta V$, we can rewrite Eq. (10) as

$$V_o(x) = \frac{P \cos \alpha}{4\pi \sigma_{co}} \int_0^b \frac{dx}{dx} - \frac{V_i(b)}{\pi a^2 \sigma_{co} r^2}$$

where $\sigma_{ci}$ = conductivity of the intra-cellular medium = 2 Sm$^{-1}$.

$$\sigma_{ci}$$

$$\sigma_{co}$$

$$V_o(x) = \frac{P \cos \alpha}{4\pi \sigma_{co}} \int_0^b \frac{dx}{dx} - \frac{V_i(b)}{\pi a^2 \sigma_{co} r^2}$$

$$I(x,t)$$

$$I(x+dx,t)$$

$$dI_o$$

$$dx$$

$$A = \pi a^2$$

$$I_i$$

$$I_o$$

$$P$$

$$\Delta V$$

$$\sigma_{ci}$$

$$\sigma_{co}$$

$$V_i(0) - V_i(b)$$

$$V_o(x) = \frac{\pi a^2 \sigma_{ci} \cos \alpha}{4\pi \sigma_{co} r^2} \int_0^b \frac{dx}{dx} - \frac{V_i(b)}{\pi a^2 \sigma_{co} r^2}$$

Fig. 1 Illustration of the cardiac tissue cylinder (of radius $a$) along the $x$ axis. In its depolarization wavefront segment (of length $b$, from the sink $-L_0$ to the source $+L_0$), due to the action potential (or transition from depolarized to resting state), there will be variation of potential within (i) the cylinder’s internal medium, as well as (ii) across the cylindrical membrane between the internal and external medium.

Fig. 2 Segment of the Cardiac tissue cylinder into which the depolarization wave front has advanced during depolarization, showing the leakage current given by Eq. (3).
Now let us consider two points $A(r_1)$ and $B(r_2)$ in the external myocardial medium far from the conducting tissue fiber, such that $r_1 = r_2 = r$. Then

$$V_o(A) = V_o(r_1) = \frac{d}{4\pi\sigma_{cor} r^3}$$

$$V_o(B) = V_o(r_2) = \frac{d}{4\pi\sigma_{cor} r^3}$$

Therefore, the potential difference between points $A$ and $B$ is given by:

$$V_o(r_1) - V_o(r_2) = \frac{d}{4\pi\sigma_{cor} r^3} (r_2 - r_1)$$

This is the expression for the potential difference between two external points $A$ and $B$ due to the electrical activity vector $d$ of magnitude $P = \pi a^2 \sigma d \Delta V_i$.

**Modified Einthoven’s triangle**

Let us now go back to the equivalent-dipole heart electrical-activity (HAV) vector $d$, and place it at point $O$ (the centroid of the modified Einthoven triangle, MET), as $\overrightarrow{OP}$, shown in Fig. 3a. In the Einthoven electrocardiographic model, the cardiac bioelectrical source is the two-dimensional dipole HAV, located at the centroid (O) of MET in the frontal plane. The modified Einthoven’s triangle is not an equilateral triangle as defined in the standard Einthoven triangle, but is defined by taking the actual physical dimensions of the three bipolar leads.

According to the centric dipole assumption, the cardiac electrical activity at any time instant can be represented by the equivalent-dipole heart electrical-activity vector (HAV) $\overrightarrow{OP}$ located at the centroid $O$ of MET [29], as shown in Fig. 3. As mentioned earlier, the magnitude and direction of this HAV vector ($\overrightarrow{OP}$) will vary from instant to instant during a cardiac cycle.

We now employ Eq. (14), to develop the expressions for the potential differences between the vertices (A & B, B & C, C & A) of the Einthoven triangle. The equation of potential difference between two points on the torso (i.e., between $A$ & $B$, $B$ & $C$, $C$ & $A$), in terms of the equivalent-dipole heart electrical-activity vector (HAV) $\overrightarrow{OP}$, is then obtained as:

$$V(r_2) - V(r_1) = \frac{\overrightarrow{OP} \cdot \overrightarrow{R}}{4\pi\sigma_{cor} r^3}$$

where $R = r_2 - r_1$.

The potential differences between $A$–$B$, $A$–$C$ and $B$–$C$ are thus considered to be due to the scalar components of the HAV (along AB, AC and BC), as shown in Fig. 3b. These three bipolar lead voltages are expressed in terms of the projections of the heart vector ($\overrightarrow{OP}$) onto each side of the Einthoven triangle [30]. Using Eq. (15), we can then put down:

$$\text{lead } I = V_1 = V_B - V_A = \frac{P r_{AB} \cos \theta}{D} = \frac{P_x r_{AB}}{D}. \quad (16)$$

where (i) $P$ is the magnitude of the cardiac vector $\overrightarrow{OP}$, (ii) $P_x$ is the magnitude of the component of $\overrightarrow{OP}$ vector along the x axis (or along AB), (iii) $r_{AB}$ is the length of side AB of the Einthoven triangle.

**Fig. 3** Modified Einthoven triangle. a Modified Einthoven’s triangle, in which the vertices $A$, $B$, $C$ are the three bipolar leads and the HAV $\overrightarrow{OP}$ is located at its centroid. A typical sample set of dimensions of the Einthoven triangle is provided in the figure, $r_a = r_b = r_c = r = 36 \text{ cm}$, $\sigma_{ci} = 2 \text{Sm}^{-1}$; $\sigma_{co} = 0.2 \text{Sm}^{-1}$. b Projections of the heart vector onto the leads, with corresponding limb lead ECG voltages.
the modified Einthoven triangle, (iv) $D$ equals to $4\pi \sigma_{e} r^2$, and (v) $P = \pi a^2 \sigma_{e} \Delta V$, $\sigma_{e} = 2 \text{Sm}^{-1}$ where in the myocardial conductivity ($\sigma_{e}$) is assumed to be 0.2 Sm$^{-1}$.

\[ \text{lead II} = V_{\text{II}} = V_{C} - V_{A} = \frac{P_{AC} \cos (60^\circ - \theta)}{D} = \left( P_{x} \cos 60^\circ + P_{y} \sin 60^\circ \right) r_{AC}, \quad (17) \]

where (i) $P_{x}$ is the magnitude of the $x$-axis component of $\overrightarrow{OP}$ vector, (ii) $r_{AC}$ is the length of the side AC of the modified Einthoven triangle, and (iii) $\theta$, measured clockwise by convention from the horizontal axis, is defined as the instantaneous electrical axis of the heart.

\[ \text{lead III} = V_{\text{III}} = V_{C} - V_{B} = \frac{P_{AC} \cos (180^\circ - \theta)}{D} = \left( P_{x} \cos 180^\circ + P_{y} \sin 180^\circ \right) r_{AC}. \quad (18) \]

**Reconstruction of HAV from limb leads and VCG generation**

This simplified approach is used for the inverse reconstruction of the HAV $\overrightarrow{OP}$ from any two of the bipolar leads. Using lead I and lead II and Eqs. (16–18), we can determine the magnitude and direction of the HAV $\overrightarrow{OP}$, in terms of its $x$ and $y$ components $P_{x}$ and $P_{y}$ given by

\[ P_{x} = V_{I} \frac{D}{r_{AB}}, \quad P_{y} = \left( V_{II} D - P_{AC} \cos 60^\circ \right) / \sin 60^\circ, \quad \theta = \tan^{-1} \left( \frac{P_{y}}{P_{x}} \right), \quad (19) \]

Using values of lead I and II voltages obtained from an ECG sample for the QRS complex, we can determine $P_{x}$ and $P_{y}$, and hence the magnitude and direction of the HAV $\overrightarrow{OP}$.

$\overrightarrow{OP}$ is the HAV $\overrightarrow{OP}$ is now plotted on a plane representing the frontal plane of the torso. The line drawn by the tip of the HAV $\overrightarrow{OP}$ (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 Cardiogram (HPVLC). Thus, the HPVLC is actually a loop, with initial and terminal points at the origin (equivalent to the iso-electric baseline).

**Results of VCG for cardiac disease states**

VCG plots for disease states

Plots of the heart-depolarization vector cardiogram (HDVC) on the frontal plane for the normal subject and abnormal subjects are shown in Fig. 4. The left hand side of the figures of Fig. 4a, b, c and d shows the ECG voltages (normalised with respect to the bioelectric baseline) at leads I and II. In this work, we have used the Cartesian coordinate system to explain the display system. Figure 4a shows the HDVC for the normal subject. In this plot, the loop is more pointing more downwards, and is prominent in the first quadrant with a small portion in fourth quadrant.

In the case of ventricular hypertrophy, the loop of the vector cardiogram is pointing upwards and is distributed in the first and second quadrant (Fig. 4b). Figure 4c shows the VCG for the bundle branch block disease. The loop of the VCG is spread in the first and second quadrants, and is pointing downwards. There is a small closed loop present in the third quadrant. The VCG graph of the inferior myocardial infarction is shown in Fig. 4d. The loop of the plot is almost equally distributed in the first and second quadrants, with a small area in the fourth quadrant.

Table 2 summarizes the loop area distribution in the four quadrants, for different disease states. With more data and VCG loop features, coupled with detailed classifier norms, one can classify the disease states more accurately.

**Combined vectorcardiograms plots**

Figure 5a shows the graphs of two vectorcardiograms for six cardiac diagnostic classes (normal, ventricular hypertrophy, blocked branch bundle, inferior, antero-septal and anterior myocardial infarction). The combined plot of these 12 VCGs gives an idea of the extent of variation within each diagnostic class and between diagnostic classes. It also provides us with a visual image of the path of the equivalent-dipole heart electrical-activity vector (HAV) in the frontal plane.

It can be seen from the plots, that, the loop area is greater for normal and BBB subjects. This loop area is less for the other abnormalities. Figure 5b and c shows the VCG graphs without myocardial infarction and with myocardial infarction, respectively. It can be seen from the graph that the loop area decreases for all the type of myocardial infarction due to the loss of the R wave amplitude (inferior MI and anterior MI).

**Progression of the heart depolarization locus during QRS complex**

A realistic progression of the equivalent-dipole heart electrical-activity vector during the QRS complex can be visualized by illustrating the heart-depolarization vectorcardiogram (HDVC) of the QRS complex in different stages, from the onset of the Q wave till the end of the depolarization stage (S wave). The progression of the HDVC for a normal subject is shown in Fig. 6.

Frontal plane vectorcardiographic parameters

In addition to the VCG plots, two additional diagnostic parameters namely, VCG loop area and loop sling length...
have been introduced in this work. These two parameters are explained below:

**Frontal vectorcardiographic QRS loop area**

The area of the vectorcardiographic loop is of great clinical significance, and is more convenient to employ for cardiac disease diagnosis compared to 1-D ECG signals [30]. Let us assume two vectors \( \vec{A} \) and \( \vec{B} \). The result of their cross-product \( \vec{A} \times \vec{B} \) is a vector perpendicular to both \( \vec{A} \) and \( \vec{B} \) is \( \vec{C} \). The absolute value of \( \vec{C} \) is two times the area spanned by the vectors \( \vec{A} \) and \( \vec{B} \). Calculating the area for the whole loop means calculating each separate area between vectors pointing from point 1 (at the origin) to point \((i+1)\) and \((i+2)\), where \(i=1 \ldots N-2\) for a loop containing \(N\) points [29].

Thus, the QRS loop area is given by

$$\sum_{i=2}^{N-2} \frac{1}{2} |\vec{a}_i - \vec{a}_1 \times \vec{a}_{i+1} - \vec{a}_1|$$

where \(\vec{a}_i\) is the position vector of point \(i\).

**Fig. 4** Typical lead I and II ECG signals and resulting VCG plots for (a) normal case, (b) ventricular hypertrophy case, (c) bundle branch block case, (d) inferior myocardial infarction case. a Leads I and II ECG signals and resulting VCG for normal subject (patient 105). b Leads I and II ECG signals and resulting VCG, for ventricular hypertrophy subject (patient 159). c Leads I and II ECG signals and resulting VCG, for bundle branch block subject (patient 202). d Leads I and II ECG signals and resulting VCG for inferior myocardial infarction subject (patient 11)
The length of the loop, which we call the sling length, is calculated by adding the distances between consecutive points together. As mentioned earlier, the loop sling length could provide correlated information about the length of the QRS complex. The distance between 2 points is given as

\[ D = \sqrt{(X_i - X_{i+1})^2 + (Y_i - Y_{i+1})^2} \]  

**Table 2** Variation of VCG loops in different quadrants for normal and ventricular hypertrophy

<table>
<thead>
<tr>
<th>Disease</th>
<th>Quadrant I</th>
<th>Quadrant II</th>
<th>Quadrant III</th>
<th>Quadrant IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Sinus Rhythm</td>
<td>+++</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Ventricular Hypertrophy</td>
<td>++</td>
<td>+++</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>

Notations: + small loop area; ++ big loop area; +++ significant loop area; – no loop area
During ventricular activation, impulses are first conducted down the left to the right bundle branches on either side of the septum. This causes the septum to depolarize from left-to-right, as depicted by the vector in Fig. 6a. This vector is heading away from the positive electrode and therefore will record a small negative deflection (Q wave departs from iso-electric baseline).

Propagation proceeds along the Purkinje fibers to the inner walls of the ventricles. Ventricular depolarization starts first from the left side of the inter-ventricular septum...
Fig. 6 Illustration of the progression of the heart depolarisation vector during a QRS complex
and therefore the resultant dipole from this septal activation points to the right (this could be seen from the left orientation of the path of the depolarization vector in Fig. 6b). Depolarization 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. The resultant depolarization vector reaches its maximum and points towards the right in this phase (shown in Fig. 6d), due to no compensating right depolarization. Figure 6d shows that the depolarization front continues propagating along the left ventricular wall towards the back.

Finally, the ventricular wall surface area decreases continuously and the magnitude of the resultant vector decreases until the whole ventricular muscle is depolarized (S wave reaches isoelectric baseline).

**HDVC parametric space**

Figure 7 provides the HDVC parametric space covered by the loop area and loop sling length parameters, for a total of 35 subjects representing healthy control and abnormal electro-cardiological states. It can be seen that each diagnostic class has its own zone in the parametric space. To verify the results obtained in Fig. 7, we need to study the correlation between the ECG features for abnormal states and the parametric space ranges occupied by data from each diagnostic class.

One can see from Fig. 7 that the HDVCs from the diagnostic class of “Bundle Branch Block” have long loop length but relatively small loop area. This is in agreement with the expected long QRS duration (>120 ms) but small R wave for this abnormal class.

HDVCs from subjects with “Ventricular Hypertrophy” occupy the parametric space with long loop length and large loop area. This is in correlation to the large R wave (>15 mm) expected of such diagnostic class. Similarly, HDVC loop area and loop length are small for the “myocardial infarction” cases, due to the loss of R waves.

**Discussion**

A novel visualization technique is proposed to provide a visual graphics display of various cardiac disorders. The orientation and the magnitude of the loop indicate the type of disease. The subtle changes in the ECG are reflected more distinctly in the VCG loop and in the VCG parametric space. Hence, it helps to identify the disease at an early stage.

VCG has been found to be more reliable than the electrocardiogram, for the diagnosis of atrial enlargement and right ventricular hypertrophy [10]. It is more sensitive than the electrocardiogram in the detection of myocardial infarction, especially if the infarction is inferior or if it occurs in the presence of left bundle branch block or left anterior hemiblock. It is also helpful in the diagnosis of ventricular pre-excitation and in the localization of the bypass tract. Some repolarization abnormalities are more clearly demonstrated by the vector display.

Increased creatine kinase concentrations after elective percutaneous transluminal coronary angioplasty (PTCA) has been shown to be associated with increased late cardiac mortality [26]. Continuous VCG monitoring during elective PTCA was shown to be a promising method for immediate detection of patients, at increased risk of procedure-related myocardial infarction. Patients with implanted stent showed significantly higher VCG values (0.05<P<0.001) than the group without a stent. There was a relationship of (P<0.06) between increased creatine kinase concentration and stent implantation.

Vectorcardiographic criteria for the diagnosis of acute right ventricular infarction was proposed by [6], based on serial vectorcardiographic and cardiac scintigraphic studies. The criteria were: (i) the direction of the maximal spatial ST vector points either to the right-anterior-inferior or to the right-posterior–inferior octant, and (ii) the magnitude of the projection of the maximal spatial ST vector is 0.15 mV in the horizontal plane. These criteria correlated with scintigraphic results to the tune of 92% sensitivity and specificity of 98%.

Recently, Chee et al., have proposed a technique to visualize cardiac abnormalities using vectorcardiograms [7]. They have used the magnitude and phase information of different lead combinations plotted in polar format, to yield a graphical cardiac vector plot over a short period of time.
It has been shown, that the vectorcardiogram is more effective compared to the ECG in many particular situations, such as (i) in the evaluation of electrically inactive areas, (ii) in intraventricular conduction disorders combined and/or in association to inactive areas, (iii) in the identification and location of ventricular pre-excitation, (iv) in the differential diagnosis of patterns varying from normal electrical axis deviation, and in the estimation of the severity of some enlargements [35].

The VCG provides two dimensional information of the electric activity of the atria and the ventricles, showing better spatial orientation and the magnitude of the vectors at every moment than shown by the ECG signals [9, 35]. The computerized VCG generates the unique graphs of the loops for each heart disease, and the length and area of the loop corresponding to the QRS complexes can be evaluated easily. Using this VCG method, the cardiac state can be analyzed immediately with greater accuracy compared to the conventional method [19, 20, 35].

In spite of the studies showing VCG to have a greater diagnostic capacity than ECG, the VCG is still evolving. Also, it will always be didactic useful to teach electrocardiology, besides it representing a low-cost method, with great diagnostic value in different situations [4, 18, 33].

In our work, we have developed the heart-depolarisation vector-locus cardiogram from limb leads and a modified Einthoven's triangle to identify the different cardiac classes. Similar concept can be applied to other cardiac diseases like cardiomyopathy, which is difficult to diagnose using the ECG. We have demonstrates different loops for different diseases at different quadrants. However, with more data and good features coupled with classifier, one can classify the diseases accurately.

Conclusion

A study of the usefulness of vectorcardiography in the diagnosis of congenital heart disease is made in this work. A reconstruction of the equivalent heart vector for the QRS complex from limb lead voltages of a sample ECG is carried out. A plot of the progression of the cardiac vector during the QRS complex is made in the frontal plane heart depolarization vector cardiogram (HDVC).

Thus, a visual display platform of VCG, for the study of subtle changes in the QRS complex using bipolar lead voltages of ECG, is developed. Hence, the VCG parametric space derived from the frontal plane VCG is able to define classification zones for different ECG abnormalities. This display technique is useful for identification of abnormalities easily. The orientation and shape of these loops are unique for each disease. This technique is simple to operate and does not involve extensive computations. This method can be used to test the efficacy of the drugs, treatment and therapy. Different patterns of HDVC for cardiac states such as normal, bundle branch block, ventricular hypertrophy and myocardial infarction are considered in this paper. Here, especially the QRS loop vector is studied for both normal and abnormal cases using the modified Einthoven triangle. Significant changes in morphology, contour, and spatial orientation of QRS vectors can be seen in the VCG loops. The VCG is a useful adjunct to electrocardiography in the diagnosis of congenital heart disease. For diagnosis of many cardiac bioelectrical abnormalities and disease conditions, it even proves to be a superior technique, since it provides instantaneous recording of HAV and VCG loops vectors in 2-D, that cannot be done with the use of electrocardiogram. The more illustrative display of the electrical events in the VCG makes this technique very valuable in the diagnosis of various forms of congenital and acquired heart diseases.

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