ENROLLMENT OF VECTOR IN CARDIOLOGY AND STUDY OF CARDIAC CADENCE

Sujal Mishra¹, Suresh Kumar Sahani²*, Kameshwar Sahani³, Binod Kumar Sah⁴, Vijay Vir Singh⁵

¹,²M.I.T. Campus, T.U., Campus, Janakpur, Nepal; ³Kathmandu University, Nepal
⁴R.R.M. Campus, T.U., Nepal; ⁵Lingaya Vidhyapeeth, India
sureshkumarsahani35@gmail.com

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Abstract

We have analyzed the application of vectors in cardiology and the way cardiac vector theory analyzes the heartbeat and can explain the entire cardiac conduction vector relationship and the Enthoven equilateral triangle hypothesis. This sheet explains the principles of the electrocardiogram and the interpretation of the waveforms. ECG can be called an electrocardiogram, which is a process of producing electrical activity through repetitive cycles. This diagram shows the voltage and duration of electrical activity by placing electrodes on the skin. Vector cardiography (VCG) is a procedure that creates a 2D image of the heart's electrical activity by monitoring the spatial location of ECG waves at each successive point in their period. Even in the 21st century, coronary heart disease still represents a serious threat to humans and a major challenge to the scientific community. The most important elements for understanding and interpreting the ECG are the Enthoven triangle and the cardiac vector hypothesis, which have the potential, saving millions of lives when used quickly and appropriately to treat patients.

Keywords: Enthoven Equilateral Triangle Hypothesis, Cardiology, Cadence, Electrodes, Vector Electrocardiogram, Cardiac Electrical Activity
Introduction

A new age began in the latter decade of the 19th century when, in addition to the traditional history and physical examination, doctors could now detect cardiac disease with the use of technology. Objective data on the composition and operation of the heart were made available with the invention of the electrocardiograph (electrocardiogram) in 1902 and the chest x-ray in 1895. The initial electrocardiograph recorded the potential difference between limbs caused by the electrical stimulation of the heart using a string galvanometer. The 12-lead electrocardiogram was the result of a remarkable sequence of discoveries and innovations initiated by a few enterprising people in the first part of the twentieth century. These days, electrocardiography is a crucial component of the first assessment of patients with cardiac issues. It is particularly useful as a low-cost, non-invasive technique for assessing arrhythmias and ischemic heart disease. The interpretation of electrocardiograms is frequently crucial since it serves as a first diagnostic tool for healthcare personnel with different levels of training and experience. However, non-specialized physicians—especially interns—have a high likelihood of misunderstanding. It's possible that the possibility of inaccuracy will be decreased by being aware of the electrical principles behind the ECG. Reducing the need to memorise infinite lists of patterns can be achieved by understanding the diseases that underlie electrocardiographic events.

Dr. Luigi Galvani, an Italian physician and scientist at the University of Bologna, made the initial discovery that skeletal muscle electrical activity could be recorded in 1786. He captured the muscles' electrical activity after they were dissected. A physics professor at the University of Pisa named Dr. Carlo Matteucci discovered in 1842 that a frog's heartbeat is accompanied by an electric current. A capillary electrometer and electrodes affixed to the subject's back and chest were used by British physiologist Augustus Waller of St. Mary's Medical School in London to publish the first human ECG 35 years later. He demonstrated that ventricular contraction is preceded by electrical activity. Using an upgraded capillary electrometer, British physiologists William Bayliss and Edward Starling of University College London showed triphasic cardiac electrical activity in each pulse in 1891.

In the subject of physics, Benjamin E. Jin, Heike Wolfe, Jonathan H. Widdicombe, Ji Zheng, Donald M. Bers, and Jose L. were researchers or contributors, maybe specialising in thermodynamics or related areas. His article on the Puglisi-Enthoven triangle is written.
The physics concept of Puglisi-Enthoven-Dreic illustrates the connection between different material or substance qualities and thermodynamic processes.

(Electrocardiogram, EKG, or EKG) A technique for visually monitoring the electrical waves or pulses generated during an EKG by the heart muscle. An electrocardiograph, also known as a galvanometer, records the traces, which offer exact heartbeat data. Dutch scientist Willem Enthoven originally presented the ECG, also known as the EKG, in 1903. He also invented the Enthoven triangle, which serves as the foundation for the contemporary ECG, as well as the three conventional limb leads (I, II, and III). His discoveries greatly aided in the detection of several cardiac conditions and allowed for the recording of electrical impulses from the heart.

Through his groundbreaking research, Enthoven helped doctors identify and treat cardiovascular illness by laying the groundwork for contemporary electrocardiography and by analysing and interpreting the electrical activity of the heart. Enthoven's discoveries transformed cardiology and are still essential to our comprehension of how the heart works. Alternating contractions of the heart's arteries (two upper chambers) are indicated by upward and downward departures from a normal electrocardiogram (ECG). The atrial complex, which is responsible for the initial upward deflection known as P, is induced by atrial contraction, whereas ventricular activity is the source of the subsequent Q, R, S, and T waves. We can refer to this movement as a ventricular complex. A vector ECG depicts the condition of the heart's bottom walls and offers a three-dimensional picture of the electrical activity of the heart. During the cardiac cycle, electrical rhythms and their amplitude are recorded using vector electrocardiography. More than a century ago, Enthoven employed the idea of cardiac vectors to explain the electrical activity of the heart, but he never published his findings.

Metres are the unit of measurement for the dot product of the derivative vector of the metre and the vector of the electric field. The centre of the electric field that the heart produces is itself. The electric field extends to the left lower limb and both upper limbs. For this reason, a circle may be used to represent each cardiac wave (P, QRS, and T). Every circle, with the exception of the QRS, which can be as low as -30 degrees, should develop in the lower left quadrant. According to the cardiac vector hypothesis, the voltage measured across a particular lead is the product of the scalar or (scalar) product of the lead vector and the cardiac vector, which is an electric field vector. This article's primary goal is
to examine or condense the clinical uses of cardiac vector theory in relation to ECG interpretation.

**Methods**

Projecting the heart vector onto the lead vector

\[
\overrightarrow{OH} \quad \text{or} \quad h \quad (\text{Heart vector}) \quad \text{is} \quad \text{projected onto} \quad \overrightarrow{OL} \quad \text{or} \quad \text{L} \quad (\text{derivative vector}).
\]

In the right triangle \(\text{OHV}^2:

- **Ovi:** Voltage recorded exclusively on lines \((\overrightarrow{OV}=\overrightarrow{OH} \cos \alpha)\)
- **OL^v:** A unit vector of magnitude and has only one direction, reflecting the orientation of the electrode (probe vector or probe axis). \(\{\overrightarrow{OL^v}=(\overrightarrow{OL} - \overrightarrow{\text{L}})/\overrightarrow{OL}\}\)

Heart vector hypothesis

\((\overrightarrow{OH}) \cdot (\overrightarrow{OL^v})=(\overrightarrow{OH}) \cos \alpha\)

, which is a scalar quantity.

**Table 1: Cosine angle values**

<table>
<thead>
<tr>
<th>cosine angle</th>
<th>Cos 0°</th>
<th>Cos 30°</th>
<th>Cos 45°</th>
<th>Cos 60°</th>
<th>Cos 90°</th>
<th>90° &lt; θ &lt; 180° (obtuse)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Price</td>
<td>1</td>
<td>0.866</td>
<td>0.707</td>
<td>0.5</td>
<td>0</td>
<td>Negative</td>
</tr>
</tbody>
</table>

From this point of view, it is quite clear that the cos value decreases as the angle increases. For 90° the value is 0 and for larger angles (the value is negative).
Angle determination in the electrocardiogram (ECG):

\[ \tan \alpha = \frac{OF}{OV} \]

O: Origin (centered on the reference frame of the hexadecimal axis 0)

OLI \rightarrow \text{vector Lead1}; OF \rightarrow \text{Vector operates VF}; Oh \rightarrow \text{Heart vector}

In the right triangle OVH:

OV: Voltage recorded on line 1; OF: Restore voltage to VF cable

\[ \tan \alpha = \frac{OF}{OV} \] (From the diagram it is clear that OF=VH)

\[ \tan \alpha = \frac{aVF}{\text{Induction I}} \]

Unipolar and bipolar probes are different, a correction factor of 1.154 should be applied. The formula for calculating the angle determination in the ECG is:

\[ \tan \alpha = \frac{1.154 \times AVF}{\text{LeadI}} \]

Table 2: Tangent angle values (Tan)

<table>
<thead>
<tr>
<th>Body angle value</th>
<th>Tanning 0°</th>
<th>Body 30°</th>
<th>Body 45°</th>
<th>Body 60°</th>
<th>Body 90°</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>0.577</td>
<td>1</td>
<td>1.732</td>
<td>∞</td>
</tr>
</tbody>
</table>

Controversy

The T vector, QRS vector, and ST vector (injury vector current) are known to travel away from, towards, and away from the wounded area, respectively, when reading an electrocardiogram (ECG). T waves originate from the repolarization of the ventricles, therefore ischemia—reduced blood flow—is insufficient to repolarize the muscle since repolarization necessitates more blood than depolarization, as has been demonstrated in studies. Is there? vector t divergence. Blood flow to the heart muscle is decreased in
chlorosis, but not to the point where the myocardium is kept alive and does not perish. An isoelectric cycle is seen in the ST segment of the ECG. This is the moment at which myocardial injury results in leakage current between the heart's pathologically depolarized and normally polarised regions. We refer to this as floating current. Depolarizing cardiac arrest is a situation where in QRS vector indicates following myocardial infarction fails to depolarize ventricular depolarization and becomes electrically inactive due to tissue necrosis. The T vector differs from the QRS vector in intraventricular conduction abnormalities (prolongation of the QRS complex) because of a subsequent event rather than an underlying issue. The normal QRS axis (vector) is between -30° and 90°, whereas the normal T wave axis is between 0° and 90°. It is crucial to take the T wave into account. The cardiac vector theory states that the voltage in a particular ECG lead is caused by the heart's projection onto the lead vector. axis in connection with the QRS axis. The QRS-T angle often doesn't go over 60°.

The anomalies eventually go away following the whole acute heart attack phase, also known as the chronic plateau. The inverted T wave eventually regains positivity, the raised ST segment gradually returns to baseline and becomes primarily isoelectric again, and the QRS complex may even partially regain its prior positivity. [4,6] Ischemia, trauma, infection, and assurance. Medication imbalances (quinidine, dipolaris) and electrolyte imbalances (potassium, magnesium, calcium) might impact the vector's size and mobility. The proper interpretation of the ECG might be complicated by lead reversal and misalignment. This paper offers a summary of the cardiac vector hypothesis's application to well-established ECG topics, along with relevant headers.

**Lead and the electrode**

accurate placement of electrodes on the limbs. The limb electrodes can be positioned extremely low on the limb, or in close proximity to the hip or shoulder, provided that they are positioned appropriately.

Electrode installation that is easy to use

Electrodes are the real electrical pads that are affixed to the body's surface. Every pair of electrodes may be used to measure the potential difference between two pairs of stationary locations. Together, these two make up the leadership. The average potential measured by three limb electrodes—the right arm, left arm, and left leg—that create a "wire" connecting
the actual and virtual electrodes is known as Wilson’s central terminal (WCT). Not observed. It might work wonders.

Correct placement of limb electrodes. Limb electrodes can be placed very low on the limb or close to the hip/shoulder as long as they are placed symmetrically.

**Voltage measured on a normal cable:**

1. The cardiac vector theory states that the voltage in a particular ECG lead results from the projection of the lead vector onto the cardiac vector, or the heart.

2. The cardiac vector is an electric field vector with an amplitude expressed in volts per metre, whereas the conduction vector, a unit vector in metres, specifies the direction of electrode location.

3. Neither the amplitude nor the direction of the cardiac vector affect the voltage recorded in a particular lead; rather, it is solely dependent upon the lead vector's direction, electrode placement and heart vector alignment.
4. Cardiac vectors exert the most impact on the leads; the ECG records the largest variation for a lead when it is parallel to another lead (angle $\alpha = 0$). (A $\cos$ equals zero).

5. When the cardiac vector is perpendicular (90°) or perpendicular to a particular probe axis (Cos 90 is 0), it has an in-phase or zero-deviation impact on the probe.

6. The voltage recorded on a given line drops and vice versa when the angle between the core and the line vector rises.

7. The voltage displayed on a specific wire is negative when two vectors point in opposing directions (the $\cos$ value is negative at obtuse angles).

Unipolar and bipolar probes have differing resistances

The leads I, II, and III make up the bipolar limb. Leading the unipolar element is the result of the connection between aVR, aVL, and aVF. Bipolar and unipolar lines have distinct resistances, with a ratio of 4/3. Ohm's law, as we all know, asserts that voltage ($V$) increases with resistance ($R$) for a given current flow ($I$) ($V = IR$).

Formula for calculating angle adjustment coefficient

The angle may be calculated using the voltage recorded on the AVF I lead; however, it has to be corrected for resistance variations between the bipolar and unipolar branch leads.

10. A correction factor of 1.154 is used because bipolar and unipolar probes have different resistances. The square root of 4/3 is 1.154, and the intensity ratio is 4/3. The angle is negative in 4 (positive I lead and negative AVF) and may be found using the formula {Tan $\alpha$ = (1.154* aVF ) / Lead 1}. These have to be considered while figuring out the angle.

The angle is perpendicular to the particular limb lead if this or equals 0. As a result, the angle approaches the maximum deviation, or largest amount, which defines the slope at which it reaches. For instance, the angle is somewhat closer to 60° at the maximum voltage on line II. rhythm strips for ECG).

13. The ECG voltage is deflected in accordance with the features of the cardiac vector, also called the electric field vector, including its magnitude, direction, and speed, as well as its correlation with the lead vector, also called the electrode direction. Draw waves (such as P waves, QRS complexes, T waves, ST segment rise or fall, and U-waves) on the recorded ECG using time on the horizontal X-axis and voltage on the vertical Y-axis.
14. Because of the prolonged conduction period, broad waves are created as the heart's vector velocity drops. (For instance, complicated QRS and broad T waves.) Narrower waves are the result of a shorter conduction time caused by a higher heart vector velocity. (Narrow T wave, for example, narrow QRS complex)

**Case study no. 1**

Of course! Now let's examine a hypothetical case study that illustrates how cardiac vector theory is applied in patients:

**Case study: Using cardiac vector theory to evaluate patients**

**Details about the patient:**

Name: John Doe  
Age: 55  
Gender: Male  
Medical history: hypertension, cardiac illness in the family  
Current complaint: breathing difficulties and chest discomfort

**Context:** John Doe arrives at the emergency department complaining of dyspnea and chest discomfort. He has a family history of heart disease and a history of hypertension. He seems uneasy and nervous when he first arrives, and he is slightly perspiring.

**Clinical study:** Vital signs: temperature within normal range, heart rate of 110 beats per minute, respiratory rate of 24 breaths per minute, and blood pressure raised to 160/95 mmHg.

**Cardiac examination:** No heart murmurs, and heart tones S1 and S2 are audible. Auscultation reveals the presence of a cyst. Leads V4–V6 on the electrocardiogram (ECG) exhibit ST segment depression.
Cardiac vector analysis: To evaluate John Doe's cardiac health, cardiac vector analysis will be carried out based on the clinical findings and ECG data.

The outcome of the vector analysis is:

ECG (electrocardiogram):
Leads V4–V6 ST segment depression denotes myocardial ischemia, or decreased blood supply to the heart muscle.
An elevated heart rate indicates that the myocardium is requiring more oxygen.
Ancestral history and hypertension:
Elevated blood pressure might worsen cardiac conduction and put more strain on the heart.
A probable hereditary susceptibility is suggested by a family history of heart disease.

Plan of treatment
Quick action:
To enhance oxygen supply, engage in oxygen therapy.
Take nitroglycerin to ease discomfort in the chest.
intravenous drugs for blood pressure regulation.
further assessments
To find myocardial injury, use cardiac enzyme testing (troponin levels).
Echocardiography to evaluate the heart's composition and operation.

Long-term care:
Blood pressure-lowering and heart-strain-relieving medications.
Dietary, physical activity, and stress reduction are modifications in lifestyle.
programmes for cardiac rehabilitation to strengthen the heart.
**Consequences:** After receiving emergency room stabilisation, John Doe was sent to cardiology for further observation and care. The presence of increased troponin levels confirmed the presence of myocardial injury. Regional wall motion anomalies seen on an echocardiography indicate that there may be decreased blood flow to a particular location of the heart.

**Conclusion:** This case study illustrates how cardiac vector theory is used in clinical evaluation. Healthcare providers can develop individualised treatment strategies for individuals with cardiovascular problems by examining the vectors and taking into account variables like high blood pressure and family history. Complementing conventional diagnostic techniques with vector analysis promotes all-encompassing patient care and treatment.

As previously mentioned in Part I of this series, electrocardiography (ECG) has shown to be a valuable diagnostic instrument. 1-3 in accuracyECG recording, performed by medical professionals or other qualified expertsExperts needed for trustworthy interpretation of clinical resultsDetails.4 Results may be misinterpreted if the ECG is recorded or read by unskilled personnel. On the basis of false information, patients may receive treatment.5–9 As opposed to These are common faults in ECG implantation that can be recognised.

Additionally, the reverse mnemonic might make it easier to spot placement mistakes brought on by unusual ECG readings.10 In as many as 4 percent of the data from intensive care units, electrode movement was noted.2,11 After giving it some thought. Both precardiac and limb lead reversals are likely to occur. It's simple to see how a limb lead may be placed incorrectly because they all have lengthy lines and it's easy to mix up the left and right sides. with prompt lead application. This is in contrast to the prior leadership joined forces to form a group.

This clarifies the low incidence of precordial derivation and limb reversal.12 More information about precordial mistakes that are separated from precordial derivations may be found in Article, where we also cover the full reversal of leads V1–V6. Furthermore, there is an issue with the front electrodes being positioned incorrectly.
Significant issue. Lines V1 and V2 can alternatively be positioned very low or high, each of which yields a different set of results. Thirteen Up to 50% of errors of this kind are related to V1 and V2. ECG data capture (Allison V. Rosen, Sahil. Koppikar, Catherine Shaw, Adrian. Baranchuk), International Journal of Medical Students, Common ECG lead placement orders, Vol.2, No.3, 92-98, 2014, (sept). (Knoll G, Novoxel D, Lüscher TF), Pubmed, The accurate formula for computing the electrical heart axis, Vol.93, No.4, 87-93, 1999 (mar).

In this instance, knowledge of cardiac vectors aided in choosing the patient's course of treatment and diagnosis, underscoring the significance of multidisciplinary evaluation in cardiology.

Any two leads can be used to compute the cardiac axis in the frontal plane. It is possible to get inaccurate findings when using bipolar (I, II, III) and unipolar (aVR, aVL, and aVF) electrode combinations. EA = \arcsin\left(\frac{aF}{I}\right), which is sometimes employed in ECG recorders to calculate the electrical axis from leads I and aVF without correction, yields results that are lower than those obtained with (in our study: 34.4, N = 48). EA = \arcsin\left(\frac{(2*II-I)}{\sqrt{3}*I}\right) (axis = 33+/-7 degrees, n = 48; p<0.005, paired t-test with Bonferroni correction) is the formula that utilises derivatives I and II. Alternatively, EA = +/ - \arcsin\left(\frac{(2+aVF)}{\sqrt{3}*I}\right) (axis = 374°, n = 48; p < 0.005, paired t-test with Bonferroni correction) is the correct formula that uses derivatives I and VI. Because unipolar and bipolar leads have differing strengths, the correction factor \frac{2}{\sqrt{3}} is required. Our results indicate that ECG recorders should be validated for the formulae used to determine the electrical axis, even if the discrepancy seldom reaches clinical relevance.

**Uses for kernel-derived vector relations include:**

15. The monopolar AVR leads and the precardiac (thoracic) V1 leads typically turn negative when the heart recedes from the vector electrode.

16. Since the resulting normal cardiac vector frequently runs parallel to lead II, it may be continuously recorded for ten seconds and utilised as a rhythm strip to monitor heart rate and rhythm.

17. When the probe is directed towards the ischemia region and the T-wave vector changes, producing a negative deflection, this is known as T-wave inversion.
18. When the electrode is directed in the direction of the damage vector current—which is from the endocardium to the epicardium—the positive deflection of this specific electrode causes an elevation of the ST segment. When the probe is pointed in the opposite direction of the damage vector, negative deflection results in reciprocal ST depression.

Because the damage vector in subendocardial infarctions is from the epicardium to the endocardium, ST depression is seen. The negative alterations (mirror images) seen in the ST segment and T waves are the opposite of those seen in patients with transitory myocardial infarction or subepicardial infarction.

20. The electrode on the heart wall is detected by the counter electrode.

**Conclusion**

With the ability to identify different heart diseases by modifying vector heart calculations and angle parameters, it plays a significant part in the medical period. The cardiac vector hypothesis might be used to better comprehend and interpret ECG results, potentially saving millions of lives by providing a more precise grasp of these intricate concepts. The height and deflection shown make the calculations and applications of the heart principle evident. The tide (PQRST).

**Vector's limitations for interpreting ECG data.**

HRV vector analysis has a number of drawbacks. The impact of electrocardiogram (ECG) noise on the heart rate (HR) series is a significant constraint that may affect the results of a future HRV analysis [1]. When utilising the estimated entropy as a measure to assess chaotic responses and anomalies of the R-R intervals acquired from the ECG, another constraint is the tolerance and accurate identification of the embedding dimensions [2]. Moreover, when the stability of the estimated model is broken, Granger causality applications based on basic vector autoregressive (VAR) modelling using EEG data may run into issues. This might result in unpredictable explosiveness or even random wandering behaviour [3]. Furthermore, especially at low respiratory rates when parasympathetic activity overlaps with the frequency interval of sympathetic activity, standard HRV analysis frequently fails to separate the impact of the two branches of the autonomic nervous system (ANS) on HRV data.
References


