Arrhythmia Detection Using ECG Versus PPG

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Introduction

Heart arrhythmia (also known as arrhythmia, dysrhythmia or irregular heartbeat) is a group of conditions in which the heartbeat is irregular, too fast (tachycardias) or too slow (bradycardias). [1,2]

There are four main types of arrhythmia - extra beats, supraventricular tachycardias, ventricular tachycardia and bradycardia.

(1) Extra beats include premature atrial contractions, premature ventricular contractions and premature junctional contractions.

(2) Supraventricular tachycardias include atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia.

(3) Ventricular tachycardia is a type of ventricular arrhythmias, which includes ventricular fibrillation.

(4) Bradycardia is a condition typically defined wherein an individual has a resting heart rate of under 60 beats per minute in adults.

Sudden cardiac death is the cause of about half of deaths due to cardiovascular disease and about 15% of all deaths globally. [3,4] About 80% of sudden cardiac death is the result of ventricular arrhythmias. [3] Arrhythmias may occur at any age but are more common among older people.
1. Heart beat analysis using ECG and PPG

Arrhythmias can occur both in the atrial and ventricular regions of the heart. An electrocardiograph (ECG) is a graph of voltage versus time of the electrical activity of the heart derived using multiple electrodes placed on the skin. The device is called an electrocardiogram. By placing twelve leads in different position of the body a multi-directional snap-shot of the heart is produced. This can help clinically determine type and origin of the arrhythmia.

However, before we go into understanding the complex twelve lead ECG let us first understand the electrical signal corresponding to a two lead ECG placed across the chest. In the case the subject has a normal sinus rhythm (NSR) a single heart beat will be electrically represented as a unique waveform called the PQRST waveform. The P wave corresponds to the depolarization of the atria, the QRS complex corresponds to the depolarization of the ventricles and the T wave corresponds to the repolarization of the ventricles. The repolarization of atria is hidden in the RWS complex.

A single heart beat can also be optically observed using a photoplethysmogram (PPG) which is a measure of blood volume changes in the microvascular bed of tissue. The RR distance in the ECG corresponds to the inter-beat-interval (IBI) of the PPG. A PPG is clinically obtained using a finger-based pulse oximeter.

Heart rate variability can be computed using various techniques like (1) time domain methods, (2) frequency domain methods or (3) non-linear methods. Both heart rate and heart rate variability can be derived from the timeseries RR distances computed from an ECG or the timeseries IBI computed from a PPG.
2. The 12 Lead ECG

A 12 lead ECG has six leads called Einthoven or Goldberger leads (I, II, III, aVF, aVR and aVL) that gives us information about the heart’s vertical axis. Whereas the other six leads called Wilson leads (V1, V2, V3, V4, V5 and V6) gives us information about the heart’s horizontal axis. The figure 1 shows the heart’s vertical and horizontal axis as well as the physiological location of the 12 leads. The 12-lead ECG is the gold standard for ECG diagnosis and is used for both resting and stress ECGs.

![Diagram of ECG leads and heart](image)

So, in effect an ECG uses 4 limb electrodes, to compute data from 6 frontal leads that provide information about the heart’s vertical plane as shown in Fig. 3.
Fig 3 - The limb leads, and augmented limb leads in a 6 lead ECG (Wilson’s central terminal is used as the negative pole for the latter in this representation). [Courtesy: Wikipedia]

A normal ECG reading is shown in Figure 4. The clinical procedure to interpret the ECG reading is explained in Appendix 1. Here are some of the factors that are used to interpret the results.

1. Regular sinus rhythm
   (1) 60 - 100 beats per minute (specifically 82 bpm).
2. P wave:
   (1) Upright in leads I, aVF and V3 - V6
   (2) Normal duration of less than or equal to 0.11 seconds
   (3) Polarity is positive in leads I, II, aVF and V4 - V6; diphasic in leads V1 and V3; negative in aVR
   (4) Shape is generally smooth, not notched or peaked
3. PR interval:
   (1) Normally between 0.12 and 0.20 seconds.
4. QRS complex:
   (1) Duration less than or equal to 0.12 seconds, amplitude greater than 0.5 mV in at least one standard lead, and greater than 1.0 mV in at least one precordial lead. Upper limit of normal amplitude is 2.5 - 3.0 mV.
   (2) Small septal Q waves in I, aVL, V5 and V6 (duration less than or equal to 0.04 seconds; amplitude less than 1/3 of the amplitude of the R wave in the same lead).
   (3) Represented by a positive deflection with a large, upright R in leads I, II, V4 - V6 and a negative deflection with a large, deep S in aVR, V1 and V2
   (4) In general, proceeding from V1 to V6, the R waves get taller while the S waves get smaller. At V3 or V4, these waves are usually equal. This is called the transitional zone.
5. ST segment:
   (1) Isoelectric, slanting upwards to the T wave in the normal ECG
   (2) Can be slightly elevated (up to 2.0 mm in some precordial leads)
   (3) Never normally depressed greater than 0.5 mm in any lead
6. T wave
(1) T wave deflection should be in the same direction as the QRS complex in at least 5 of the 6 limb leads
(2) Normally rounded and asymmetrical, with a more gradual ascent than descent
(3) Should be upright in leads V2 - V6, inverted in aVR
(4) Amplitude of at least 0.2 mV in leads V3 and V4 and at least 0.1 mV in leads V5 and V6
(5) Isolated T wave inversion in an asymptomatic adult is generally a normal variant

7. QT interval
   (1) Durations normally less than or equal to 0.40 seconds or males and 0.44 seconds for females.

Figure 4 - Normal ECG
### 3. Arrhythmia detection with 12 Lead ECG

<table>
<thead>
<tr>
<th>Type of arrhythmia</th>
<th>Arrhythmia</th>
<th>How to detect [5]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Extra beats</td>
<td>Premature atrial contractions (PAC)</td>
<td>Is recognized by three distinct features in leads II and V1, (1) a premature and unusually shaped P wave (designated P') (2) a QRS complex resembling a normal sinus beat (3) a following cardiac cycle that is less than compensatory in duration. Irregular shape of the P' wave, irregular duration of the PP interval and extended duration of the P'R interval to greater than 0.12 seconds.</td>
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<tr>
<td>Wandering atrial pacemaker</td>
<td>Is recognized by flattened, niched or diaphasic P-waves in lead II.</td>
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<tr>
<td>Premature ventricular contractions</td>
<td>Is recognized as single or paired unifocal beats, with no preceding P wave, a wide QRS complex of increased amplitude characteristically lasting greater than 0.14 seconds, and a T wave demonstrating polarity opposite to that of the PVC in lead II.</td>
<td></td>
</tr>
<tr>
<td>Prolonged QT interval</td>
<td>Is recognized best in lead II.</td>
<td></td>
</tr>
<tr>
<td>Junctional Arrhythmia</td>
<td>Junctional rhythm</td>
<td>Is recognized by absence of P wave or presence of an inverted P wave in lead II.</td>
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<tr>
<td>Junctional tachycardia</td>
<td>Is recognized by (1) The p-wave may be inverted in leads II, III and aVF or may not be visible (2) Narrow QRS complexes.</td>
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<tr>
<td>Premature junctional contractions</td>
<td>Is recognized by normally shaped ventricular complex or QRS complex, not preceded by any atrial complex or P wave or preceded by an abnormal P wave with a shorter PR interval.</td>
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</tr>
<tr>
<td>Supraventricular tachycardias</td>
<td>Atrial fibrillation</td>
<td>Is recognized by missing P waves in leads I and III. Atrial fibrillation is common in patients with rheumatic heart disease, pulmonary emboli, cardiomyopathy, pericarditis, ischemic heart disease and thyrotoxicosis. It causes minimal hemodynamic compromise and often the patient presents complaining of palpitations as the only symptom.</td>
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<tr>
<td>Atrial flutter</td>
<td>Is recognized by &quot;sawtooth&quot; flutter waves (also called F</td>
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<tr>
<td>Arrhythmia Type</td>
<td>Description</td>
<td></td>
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<tr>
<td>---------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Atrial tachycardia</td>
<td>Is recognized by different morphology of P wave depending on exactly where it originates. This is referred to as an “ectopic atrial rhythm” or “ectopic P wave.”</td>
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<tr>
<td>AV reentry</td>
<td>Is recognized by narrow QRS complex with a duration less than 0.2 seconds and a conduction ratio of 1:1. P waves that are always present outside of the QRS complex, while their polarity depends on the atrial insertion of the accessory pathway.</td>
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</tr>
<tr>
<td>AV nodal reentry</td>
<td>Is recognized by narrow QRS complex with a duration less than 0.2 seconds and a conduction ratio of 1:1. P waves are buried within the QRS complex. Due to the fact that atrial activation originates from the inferior aspect of the right atrium, P wave polarity is negative in leads II, III and aVF.</td>
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<tr>
<td>Ventricular tachycardia</td>
<td>Monomorphic ventricular tachycardia</td>
<td></td>
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<tr>
<td>Accelerated idioventricular rhythm</td>
<td>Similar to those of Monomorphic ventricular tachycardia with wide QRS complexes (QRS&gt;0.12 seconds).</td>
<td></td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>Is recognized by unformed QRS complexes without any clear P waves in all leads.</td>
<td></td>
</tr>
<tr>
<td>Torsades de pointes</td>
<td>Is recognized by twisting of QRS complexes without any clear P waves in all leads.</td>
<td></td>
</tr>
<tr>
<td>Sinus bradycardia (primarily caused by heart block)</td>
<td>Is recognized by three distinct features, (3) P waves: Upright, consistent, and normal in morphology and duration. (4) PR interval: Between 0.12 and 0.20 seconds in duration. (5) QRS complex: Less than 0.12 seconds in width, and consistent in morphology.</td>
<td></td>
</tr>
</tbody>
</table>

Some other conditions such as myocardial infarction (MI) that a 12 Lead ECG can detect [5],

1. Acute anterolateral MI - is recognized by ST segment elevation in leads I, aVL and the precordial leads overlying the anterior and lateral surfaces of the heart (V3 - V6).

2. Acute inferior MI - is recognized by ST segment elevation and or Q and T wave inversion in leads II and III, and aVF.
(3) Acute posterior MI - is recognized by ST segment depression in leads V1 and V2.

(4) Acute right ventricular MI - is recognized by ST segment elevation by more than 1mm in lead aVR.

(5) Acute septal MI - is recognized by ST segment elevation and or Q and T wave inversion in leads V2 and V3.

(6) Complete heart block - is recognized by P waves that does not produce QRS complex in I, II, III and aVR, aVL and aVF.

(7) Digitalis effect – is recognized by ST shortening in V4, V5 and V6.

(8) Dual chamber pacemaker – is recognized by presence of two closely located PQRST wave in all lead.

(9) Hyperkalemia (Potassium overdose) – is recognized by the QRS complex diffusely broadened and continuous with the tall, tented T wave in all leads.

(10) Hypokalemia – is recognized by progressive ST depression, progressive flattening or inversion of the T waves, the development of U waves, increased amplitude and duration of the P waves and QRS complexes as well as a slight increase in the duration of the PR interval in all leads.

(11) Left atrial enlargement – is recognized by an increase in the terminal portion of the P wave seen in lead II. This deflection does not usually affect the amplitude of the P wave but may increase its duration to greater than 0.12 seconds.

(12) Left ventricular hypertrophy (LVH) – is recognized by increased R wave voltage and duration in leads over the right ventricle V5 and V6. LVH is clinically corelated with the risk of other cardiovascular diseases including myocardial infarction, congestive heart failure, stroke, arrhythmia and sudden death.

(13) Left bundle branch block – is recognized by wide QRS complex (greater than 0.12 seconds in duration) with an abnormal morphology in leads I, V1, V6.

(14) Pericarditis - is recognized by diffuse ST segment elevation in all leads except aVR and V1.

(15) Pulmonary embolism – is recognized by increase in the normal Q wave amplitude, minimal ST segment elevation, and often shallow T wave inversion only in lead III.

(16) Right atrial enlargement – is recognized by P wave shape is peaked (duration is unaffected) and its amplitude is increased to greater than 2.5 mm in leads II, III, aVF (also sometimes in V1).

(17) Right bundle branch block – is recognized by a wide QRS complex lasting greater than 0.12 seconds. Altered terminal QRS forces produce a terminal R wave in lead aVR and terminal S waves in leads I, aVL, V5 and V6. Triphasic complexes are identified as the late intrinsicoid “m-shaped” RSR’ complex in lead V1, and the early intrinsicoid qRS complex in lead V6.

(18) Single chamber pacemaker – is recognized by ”pacemaker spikes” identified by their abrupt vertical spike (arrows below), preceding the atrial or ventricular complex, depending on which chambers the pacemaker is responsible for.

(19) Wolff-Parkinson-White Syndrome – is recognized by delta waves. These waves resemble pathological Q waves and represent initial slurring of the QRS complex as a result of early ventricular depolarization through this accessory pathway in leads II, V1 and V6. As a result, the PR interval is shortened to less than 0.12
seconds and the QRS direction is altered is lead III, while its duration is extended to greater than 0.10 seconds. Secondary T wave anomalies resulting from abnormal ventricular repolarization are often demonstrated in leads II, III, V2, V3 and V4.

4. Arrhythmia detection with 5, 3 and 1 Lead ECG and PPG

A 6-Lead ECG uses 4 limb leads and 1 chest lead (corresponding to Einthoven or Goldberger Leads). This displays the bipolar leads I, II, and III and one of the V1-V6 depending on the position of the chest lead. It helps improve ST elevation readings but it’s still inferior to the 12-lead ECG.

A 3-Lead ECG uses 3 electrodes that are labeled white, black, and red (not universal). These 3 leads monitor rhythm monitoring but doesn’t reveal sufficient information on ST elevation activity. This displays the bipolar leads I, II and III.

A 1-lead ECG uses 2 electrodes. These are used typically for continuous or remote patient monitoring, where clinicians are interested in detecting early warning signs for cardiovascular diseases. With the advent of wearable medical devices such as iRhythm, AliveCor or Apple Watch these are bridging the gap between fitness wearables and remote patient monitoring. Although it was believed, that medical relevance of one lead ECGs is very limited recently Apple Watch and AliveCor has got FDA approval for detecting Atrial Fibrillation, Bradycardia and Tachycardia.

The table below shows different types of arrhythmias that can be distinguished by different types of ECG. As we decrease the number of leads, they detect a smaller range of arrhythmias as well as the ability to distinguish between different sub-types of arrhythmias. For example, a 1 Lead ECG can detect tachycardia but not Torsades de pointes. The table shows how a PPG based sensor can outperform a 1 Lead ECG. However, when a 1 Lead ECG and a PPG is combined in a single device, it is still qualitatively limited compared to a 5 lead ECG in their ability to detect the range and type of arrhythmias.

<table>
<thead>
<tr>
<th>Type of arrhythmia</th>
<th>Usage -&gt;</th>
<th>Clinical</th>
<th>Holter Monitor</th>
<th>Remote Patient Monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>12 Lead</td>
<td>5 Lead</td>
<td>3 Lead</td>
</tr>
<tr>
<td>Extra beats</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premature atrial contractions (PAC)</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wandering atrial pacemaker</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Premature ventricular contractions</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X [6]</td>
</tr>
<tr>
<td>Prolonged QT interval</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

*Usage*: Clinical, Holter Monitor, Remote Patient Monitoring
<table>
<thead>
<tr>
<th>Arrhythmia</th>
<th>Junctional rhythm</th>
<th>X</th>
<th>X</th>
<th>X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Junctional tachycardia</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Premature junctional contractions</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Supraventricular tachycardias</td>
<td>Atrial fibrillation</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td></td>
<td>Atrial flutter</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atrial tachycardia</td>
<td>X</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>AV reentry</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>AV nodal reentry</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Monomorphic ventricular tachycardia</td>
<td>X</td>
<td>X</td>
<td>X</td>
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<tr>
<td></td>
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<td>X</td>
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<td>Ventricular fibrillation</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td></td>
<td>Torsades de pointes</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Bradyarrhythmia</td>
<td>Sinus bradycardia (primarily caused by heart block)</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

X – Can detect

**References**

1. [https://www.nhlbi.nih.gov/health-topics/arrhythmia](https://www.nhlbi.nih.gov/health-topics/arrhythmia)
2. [www.nhlbi.nih.gov/health/health-topics/topics/arr/signs](http://www.nhlbi.nih.gov/health/health-topics/topics/arr/signs)
3. Global public health problem of sudden cardiac death

5. https://meds.queensu.ca/central/assets/modules/ECG/


Appendix A - How to read a multi-lead ECG [5]

When a physician sees an ECG, these are the following preliminary analysis they do,

(1) Determination of heart rate.

There are a number of strategies for determining the heart rate. A simple, quick technique is to first find a QRS complex that falls on a major vertical grid-line, and then count the number of large squares to the next QRS complex. Dividing this number into 300 gives you the heart rate. In the ECG below, there are 2 large squares between QRS complexes. 300/2 gives a heart rate of 150 beats per minute.

![Sample ECG](image)

Fig 2 – Sample ECG

(2) Determination of PR, QRS, and QT intervals.

The measurement of important electrocardiographic intervals usually includes the PR interval (<0.2 sec), the QRS interval (<0.12 sec) and the QT interval. At a standard paper speed of 25 mm/second, the width of each small square (1mm) represents 0.04 seconds. One large square (5mm) represents 0.2 seconds.

(3) Calculate the electrical axis.

Use the leads I and aVF to calculate an approximate axis. If Leads I and aVF equally positive, the axis will be midway between 0° and 90° (normal axis). If both Leads I and aVF are positive and Lead I is positive than aVF, then the axis will be oriented more toward 0°. If the Lead I is positive, and Lead aVF almost equiphase the the axis will be ~0°.

(4) Evaluate the cardiac rhythm.

Check if the rhythm is regular, the RR interval should be constant throughout the ECG. This can be checked by marking on a piece of paper the distance between two R waves and comparing this distance between pairs of QRS complexes on the ECG. Next, check to see if a P wave is present before each of the QRS complexes.
(5) Inspect P waves for atrial enlargement

The P waves in leads I, II, III and V1 should be inspected for evidence of right or left atrial enlargement. Usually, lead II will have the clearest P wave.

- P wave amplitude should not exceed 3 small squares (3 mm or 0.3mV). If it does, this represents right atrial enlargement.
- In lead V1, the terminal negative deflection of the P wave represents left atrial depolarization and should not exceed 1 mm (0.1mV). If it does, this is indicative of left atrial enlargement.

(6) Inspect QRS wave for ventricular hypertrophy

In the setting of Left Ventricular Hypertrophy (LVH), the left ventricle enlarges and so the leads oriented to the left ventricle (V5, V6, aVL) will "see" more electrical activity moving towards them. As well, the leads oriented away from the left ventricle (V1, V2) will "see" more activity moving away from them. In LVH therefore, leads V5, V6 and aVL will have tall R waves, while leads V1 and V2 will have deep S waves.

(7) Inspect QRS complexes for bundle branch block or fascicular block

The normal QRS interval is 0.12 seconds (3 mm or 3 small squares) on the ECG. To correctly determine the QRS interval, use the lead with the widest QRS complex. If the QRS complex is less than or equal to 0.12 seconds, then no further analysis is necessary. If it is greater than 0.12 seconds, then one determines the reason for the abnormally long QRS interval.

The type of bundle branch block can usually be determined from the examination of three key leads: I, V1 and V6.

(8) Assessment of Q waves

The Q waves should be assessed, and their significance determined, particularly in regard to the diagnosis of myocardial infarction. Small Q waves are commonly a normal finding in the inferior leads III and aVF, and in the anterolateral leads aVL, I, V5 and V6. Q waves of 0.04 seconds (1 mm) duration and greater than one third the R wave’s amplitude in the same lead may be pathological.
Fig 3 - The pathological Q waves seen in V1 - V6 indicate that this patient has had an anterior MI in the past. This patient also has evidence of an acute inferior MI as shown by the ST segment elevation in leads III and aVF.

(9) Access ST segments and T waves

Assess the ST segment for the presence of elevations or depressions, together with T wave abnormalities. ST elevation can indicate the presence of conditions such as acute myocardial injury, Prinzmetal's (variant) angina, pericarditis, ventricular aneurysm or myocardial ischemia.

Fig 4 - This ECG is from a patient with an acute inferior MI. Note the ST elevation in the inferior leads (II, III and aVF). The ECG also shows ST depression in leads V1, V2 and V3 - likely a result of reciprocal changes associated with the MI.

(10) Measure QT interval for specific diagnosis
The QT interval can be prolonged secondary to metabolic disorders and drug effects. It must be corrected for heart rate since it is rate dependent. The corrected QT interval is calculated using the following formula:

$$\text{QTI corrected} = \frac{\text{QTI observed}}{\sqrt{\text{RR interval}}}$$

The QTI corrected is often reported with computerized ECG interpretation.