1.6: Twelve Leads and Special Rhythms

This module will examine a very limited number of 12 lead interpretation methods and things to watch for in practice. This is a limited look at 12 lead EKG interpretation and should not be used as a comprehensive learning module for 12 lead interpretation.

ST Elevation

ST elevation refers to the failure of the QRS complex to return to the baseline before the T wave commences. Elevation specifically refers to this slurring of the QRS-to-T wave that occurs above the isoelectric line. It looks like this:

![Image 1: ST Elevation](https://med.libretexts.org/Bookshelves/Nursing/An_EKG_Interpretation_Primer_(Christianson_et_al.)/01%3A_Chapters/1.06_12-Lead_interpretation_files/12_lead_interpretation_01.png)

Note that the S wave in the QRS complex does not return to the baseline before the T wave starts in V2. The S wave does return to the baseline in V6.

ST elevation of 1 mV (1 small box) or larger is considered to be clinically significant, and it must be replicable in more than one full cardiac cycle (ie: P to T wave).
The reciprocal change, ST depression, is the same finding but instead of failing to return to the baseline in favor of an elevated graph above the baseline, the S does not return to the baseline and remains below the baseline. ST depression looks like this:

![Image 2: ST depression](https://med.libretexts.org/Bookshelves/Nursing/An_EKG_Interpretation_Primer_(Christianson_et_al.)/01%3A_Chapters/1.06...)

**What Does ST Elevation Mean?**

It is not clearly known what causes ST elevation and depression - it is thought that it is an electrical defect caused by ventricular tissue that is ischemic and damaged or otherwise malfunctioning. One ST elevation and depression hypothesis is that it occurs because ischemic ventricular tissue stays fully polarized for longer than normal. That polarization (which is measured on the EKG as electrical activity) causes the EKG graph to measure as non-zero until after the T wave.

ST elevation in different leads can mean different things. A condition called pericarditis can cause mild diffuse ST elevation in most or all leads:

![Image 3: Pericarditis](https://med.libretexts.org/Bookshelves/Nursing/An_EKG_Interpretation_Primer_(Christianson_et_al.)/01%3A_Chapters/1.06...)

**Pericarditis** is an inflammation or infection of the sack around the heart (pericardium). It causes irritation to the overall heart tissue which presents as diffuse ST elevation.
ST elevation can also be an indicator of a specific type of myocardial infarction known as STEMI. This particular type of MI is seen as emergent because a true STEMI usually indicates a blockage of 90% or more of one of the coronary arteries. Patients with STEMI require immediate intervention; lack of intervention will lead to death or debilitating chronic heart conditions like congestive heart failure. To review, the coronary arteries that deliver blood to the heart itself are located as diagrammed below:

![Coronary arteries diagram](https://med.libretexts.org/Bookshelves/Nursing/An_EKG_Interpretation_Primer_(Christianson_et_al.)/01%3A_Chapters/1.06...

Figure 4: Image 4, Coronary arteries

Note that the posterior descending artery is located along the posterior wall of the heart. The left circumflex artery also wraps around to the posterior heart.

As we discussed above, ischemic heart tissue can produce ST elevation. In the case of STEMI, ischemia is caused by grossly inadequate or entirely absent blood flow due to an occlusion of one of the coronary arteries. The coronary artery affected can be approximately located using a 12 lead EKG to determine which areas are likely ischemic and therefore likely deprived of blood flow. While this method is not precise, it does give interventional cardiology a general idea of where they should start looking for coronary artery blockages.

In addition to ST elevation in specific leads, in a patient with STEMI you will often see ST depression in the reciprocal leads that are electrically opposite from the leads with elevation. This makes sense if you consider previous lessons on lead placement and what leads are designed to detect: electrical activity moving toward or away from them.
Notice that each color labeled area is supplied by a particular coronary artery.

The areas of the heart that are measured by particular leads are circled in different colors. Green represents the right ventricular region (right marginal artery) and is measured by V1. Purple represents the septal region; septal refers to the septum separating the two ventricles. The septal region is measured by V2 and V3. Light blue represents the anterior segment and is measured by V3 and V4. The red segment is the lateral and is measured by leads I, aVL, V5 and V6.

There is one remaining segment called the inferior segment that is not labeled with a color on the above diagram. The inferior segment is as the name suggests; the bottom portion of the heart that faces the diaphragm. It includes the inferior portion of the apex of the heart. This segment is measured using leads II, III, and aVF.

<table>
<thead>
<tr>
<th>Segment</th>
<th>Leads</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>V1</td>
</tr>
<tr>
<td>Septal</td>
<td>V2, V3</td>
</tr>
<tr>
<td>Anterior</td>
<td>V3, V4</td>
</tr>
<tr>
<td>Lateral</td>
<td>I, aVL, V5, V6</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, aVF</td>
</tr>
</tbody>
</table>

Of note: The right ventricular area is poorly visualized by a 12 lead EKG setup. If a right STEMI is suspected, additional leads are often attached. A posterior STEMI is also generally not visible on the 12 lead EKG, as it is measured by leads V7-9 which are not applied during a standard 12 lead EKG. Much like leads V1-6 wrap around the left side of the chest, leads V7-9 continue to wrap around the posterior section of the chest wall. We will not discuss leads or placement beyond the standard 12 lead in detail during this primer.

Additionally, many types of STEMI will show inverse changes in a reciprocal leads. Reciprocal leads refers to leads that show the opposite direction another lead. For example aVF and aVL are leads that go in almost perfectly opposite directions; if you have a patient with an inferior STEMI, in addition to ST elevation in aVF you will often also see ST depression in aVL. Reciprocal changes can include ST depression instead of elevation and inversions of particular
Prinzmetal’s Angina

Prinzmetal’s angina, or coronary artery vasospasm, is a specific type of angina that causes EKG changes. The condition itself causes spasm of the smooth muscle around the coronary arteries. Spasm of the muscles causes a temporary occlusion of the artery, resulting in ischemia that resolves when the spasm resolves. The spasms usually resolve spontaneously, but do sometimes require intervention.

Figure 7: Image 7, Coronary vasospasm

The EKG changes that occur with Prinzmetal’s angina depend upon the affected coronary artery, and usually present with changes that are otherwise consistent with STEMI. Prinzmetal’s angina should be treated as if it were a STEMI until proven otherwise; STEMI is lethal if not treated, Prinzmetal’s angina is not. It is never wrong to err on the side of caution when there is concern for a condition that can impact a patient’s life, limb, fertility, or vision.

Figure 8: Image 8, Inferior STEMI

The difference between a STEMI and a patient with Prinzmetal’s angina is the consistency of the EKG changes. A patient with a STEMI will not have spontaneous resolution of the EKG changes; a patient with Prinzmetal’s angina will sometimes spontaneously self-resolve, sometimes resolve entirely with medical intervention like nitroglycerin.

Figure 9: Image 9, Spontaneous Return to NSR

Prinzmetal’s angina typically presents with the same symptoms as cardiac ischemia or acute coronary syndrome: chest pain that may radiate into the left arm, jaw, neck, or left shoulder, difficulty breathing, diaphoresis, dizziness, feeling of impending doom. These symptoms spontaneously resolve when the coronary vasospasm resolves.

Common causes of Prinzmetal’s angina are exposure to cold, excessive stress, certain medications, and drug use (particularly cocaine). Cocaine-related Prinzmetal’s angina is sometimes colloquially referred to as “the cocaine chest.
pain” in emergency medicine as it is a fairly common and well known emergent problem in cocaine users.

Wellens Syndrome

Wellens Syndrome is an EKG change that has a strong correlation with impending anterior STEMI (approximately 75% of patients who present with Wellens Syndrome developed anterior STEMI within 1-2 weeks of noted EKG change in a study in the early 80s). It is identified as an inverted or biphasic T wave in leads V2 or V3 (sometimes both).

![Wellens Syndrome Image](image)

It is a helpful syndrome to identify because it can flag a patient who needs cardiac intervention before they have a life-threatening event like STEMI.

Bundle Branch Blocks

A bundle branch block (BBB) is a similar issue to the AV blocks discussed in an earlier segment with regards to physiology. AV blocks refer to dysfunction of the AV node. A bundle branch block refers to a malfunction of a branch of the bundle of His (the AV node is functioning normally).

![Bundle Branch Blocks Image](image)

Recall the anatomy of the bundle of His and the electrophysiology of the area. The bundle of His refers to the insulated bundle of cardiac tissue that allows electricity to travel toward the bottom of the ventricles before the ventricular depolarization begins, which causes the ventricles to pump from the bottom upward toward the semilunar valves. The bundle of His is made up of 2 branches (not mentioned previously): one for the right ventricle, one for the left. These are referred to as the “bundle branches.”

The bundle branches do not commonly malfunction at the same time, and a malfunction of the right bundle branch looks different from a malfunction of the left. It is also notable that, much like an AV block, a bundle branch block can be partial or complete. Bundle branch blocks can be acute or chronic, and it is important to compare them to a previous EKG if
A right bundle branch block (RBBB) occurs when the right branch of the bundle of His is not conducting correctly. This results in slowed conduction to the right ventricle, but normal conduction to the left ventricle, causing the left ventricle to pump normally but the right ventricle to pump slightly after the right ventricle with regards to time. This causes the QRS complex to look abnormally in most leads, and is most prominent in the leads that examine the right side of the heart.

The first 3 precordial leads (V1-3) are the most distinct, and you will notice a progression of decreasing severity of the QRS abnormality. V1 is typically the most abnormal looking, V2 will appear somewhat better, V3 will appear better yet, etc as the precordial lead view adjusts toward the normally functioning left view.

RSR' (pronounced R, S, R prime) is a phenomenon in which the R wave appears “interrupted” by the S wave. The R begins in the left ventricle, appears to stop abruptly during the normal S wave occurring in the left ventricle, meanwhile the R wave is occurring in the right ventricle after it occurs in the left because of the slowed conduction down the right bundle. So after the left ventricle’s S wave, the R appears on the EKG to restart (the second R is referred to as R’ or R prime). This is sometimes called a “bunny ears” appearance because of the two R wave peaks.

An RSR’ morphology in a QRS complex will almost always appear to have ST elevation, as it does in the above example, because the T wave in the left ventricle has usually started before or as the QRS complex in the right ventricle completes, so there is no visible return to baseline on the EKG graph. Again, the key to differentiating true ST elevation from a change caused by a problem like a bundle branch block is to examine the QRS morphology and the other leads. While a right STEMI can present with ST elevation visible exclusively in V1 (due to poor depiction of the right side on a standard 12 lead), a right STEMI should not also have an RSR’ pattern and may or may not have changes in any other adjacent leads.
Leads III, V6, and aVF are typically the most normal appearing in a right bundle branch block because their view of the heart is least impacted by the right bundle branch - lead 6 is furthest away from the right bundle branch and aVF is a view that looks from top to bottom at an angle toward the left ventricle.

Left Bundle Branch Block

A left bundle branch block (LBBB) appears somewhat more dramatically than a right bundle branch block does. The right ventricle is smaller than the left, as the right is only responsible for pumping to the pulmonary vasculature where the left is responsible for maintaining blood flow to the entire body; the differential in size (and thereby electrical output) causes somewhat more dramatic EKG changes compared to the right bundle branch block.

Additionally, while a right bundle branch block is best visualized in the right precordial leads V1-3 and sometimes not apparent at all in V6, a left bundle branch block is often apparent in all of the leads on a standard 12 lead and is most prominent in leads V5 and V6.

Figure \(\PageIndex{13}\): Image 13, Left Bundle Branch Blocks

Note that in the circled leads, the QRS complexes still look somewhat abnormal and mono-directional. In a left bundle branch block, the QRS complex in the right ventricle starts before it does in the left. This is best visible in the precordial V1-3 leads as a deep, dramatic S wave with minimal other visible waves in the QRS complex. Many of the leads appear to mostly go in one direction because the right side depolarizes first, then the left; as the electricity in the right side is flowing away from the unipolar electrodes during the S wave at the end of its QRS complex, the left side is beginning its depolarization as the right is ending, resulting in electricity flowing away from the right precordial electrodes in a much larger quantity than compared to normal depolarization (remember that the height of a wave correlates to the amount of electricity detected by the electrode). The R wave may or may not be visible, depending on the exact timing of the electrical flow; if it is, it is usually a very small upward notch at the beginning of the S wave.

The same RSR' morphology is sometimes present with a left bundle branch block, but is usually in the left-facing V5 lead. The above green circled complex is an RSR' morphology; sometimes there is a similar RS morphology (which is the same except the R' is absent).

You will also notice monophasic QRS complexes in many leads, prominently in lead I, aVL, and V6. The QRS complex may be monophasic, or consist of a singular R wave, or have a small notch near the top, as aVL does in this example.

R-on-T Phenomenon and Torsades de Pointes

R-on-T phenomenon is an anomaly in which a T wave (ventricular repolarization) is interrupted by an R wave (ventricular depolarization). This is most commonly caused by a PVC or an SVT. Prolonged QT intervals can also
predispose a patient to R-on-T phenomenon.

Occasionally this can cause irregularities in the depolarization-repolarization pattern in adjacent ventricles, leading to a unique type of ventricular tachycardia called torsades de pointes (“turning of the points”). The heart is most prone to this if it is already experiencing some degree of ischemia; this phenomenon is extremely rare absent any other cardiac pathology.

Torsades de pointes is a type of polymorphic ventricular tachycardia, or a ventricular tachycardia that changes in QRS morphology. Refer to our review of premature ventricular contractions: QRS morphology for ventricular rhythms is dependent on where the depolarization originates from. A polymorphic ventricular tachycardia originates from multiple different sites. In the case of torsades de pointes, it is a “rotating” of sites in a circular, gyrating pattern around the heart. This gyrating pattern of depolarization origination causes a pattern that seems to twist around the isoelectric line.

Torsades de pointes is a lethal arrhythmia and is treated with defibrillation. It can also be treated with IV magnesium, though the mechanism through which this treatment works is poorly understood. If it is not treated, it usually converts into ventricular fibrillation and ultimately asystole.

**Wolff-Parkinson-White Syndrome**

Wolff-Parkinson-White syndrome (WPW) is a common cause of sudden unexpected cardiac death in athletes and young adults. It is caused by an inherited abnormality in the electrophysiology pathways of the AV node. In a normal heart, the AV node is the only pathway between the atria and the ventricles and only allows electrical currents to flow in from atria to ventricles. WPW is caused by an extra pathway (called an accessory pathway) that also connects the atria to the ventricles. This means that the AV node is not a reliable gatekeeper between the atria and ventricles and electrical impulses can bypass the AV node, which causes the ventricles to begin the depolarization process in an abnormal pattern originating from the accessory pathway (referred to as pre-excitation). Additionally, the extra unregulated pathway means electricity from the ventricles can reach the atria.
The flow of electricity from the atria to the ventricles through the accessory pathway can be seen on an EKG as an early slurring from the baseline to the QRS complex, called a delta wave. The slurring can usually be seen in most or all leads, but will be more prominent in leads viewing the part of the heart the accessory pathway is located in (ie: if the accessory pathway is on the right as in the picture, the delta wave should be most prominent in V1-2 and aVR). A WPW patient may or may not always have visible EKG changes consistent with the condition as some accessory pathways are semi-selective in the same way the AV node does not let all impulses through every time.

The delta wave can be subtle and difficult to notice, and generally requires a moderate to large amount of EKG interpretation experience to detect.

WPW can cause sudden cardiac death in a similar manner to R on T complex. In short: The SA node starts an impulse, the impulse travels both normally to the AV node and abnormally through the accessory pathway. The accessory pathway excites and starts ventricular depolarization early. The AV node releases its impulse after the ventricles have already started depolarization, stimulating ventricular tissue that is not necessarily repolarized and ready for another depolarization. Similarly to R-on-T complex, this can cause spontaneous ventricular fibrillation or torsades de pointes.
WPW patients are also prone to a problem called reentrant tachycardia, which is a type of supraventricular tachycardia (SVT). This occurs when the electricity from the ventricular depolarization loops back up to the atria again using the accessory pathway, stimulating them to depolarize in fast succession (as in the drawing above). This results in an electrical loop that causes a rapid heart rate. It is difficult to correctly differentiate SVT causes based on EKG, but a reentrant SVT features P waves that may or may not be visible and may be after the QRS complex (similar to a junctional rhythm). The QRS morphology is usually normal; this specific type of reentrant SVT is referred to as orthodromic.

Occasionally the AV node also fails and allows retrograde electrical flow (ie: flow from the ventricles into the atria), which can cause a wide QRS complex and a circuit that loops in the opposite direction. This is referred to as an antidromic reentrant SVT. It is very difficult to differentiate both clinically and based on EKG from a ventricular tachycardia, as it is a wide complex tachycardia, and it is usually treated in the same way a ventricular tachycardia would be treated (synchronized cardioversion or defibrillation).

Wolff-Parkinson-White syndrome is, in most patients, asymptomatic and they are often unaware they have the condition. It is usually caught during an SVT event in which a patient presents for palpitations, dizziness, syncope or pre-syncope, chest pressure, or shortness of breath. It is also sometimes caught on routine EKG. If it is symptomatic or a patient is in an occupation that is considered to be high-risk, such as an athlete, the patient may undergo a cardiac ablation to remove the accessory pathway. Ablation is typically not recommended in otherwise asymptomatic patients with low occupational risk.
Brugada Syndrome

Brugada syndrome is an inherited dysfunction of the sodium channels. Recall the physiology of the heart: sodium cations (positive) are allowed to flood in via the voltage-gated fast Na+ channel, allowing the cardiac muscle cell to reach action potential and contract. Refer to the EKG Primer Part 0 segment on depolarization for more details about the fast Na+ channel’s function.

Dysfunction of the fast Na+ channel alters the way the heart depolarizes, which affects both the way the muscles contract and the electrophysiology of the heart. It can be difficult to diagnose or identify correctly on EKG, but it is most commonly identified as ST elevation in any of the leads V1-3 with an inverted or biphasic T wave in the same lead.

Figure \(\PageIndex{21}\): Image 21, Brugada syndrome

Brugada syndrome also requires clinical criteria for diagnosis, as the EKG changes alone are not specific enough to definitively diagnose. Other criteria include sudden cardiac death or known Brugada in a member of the immediate family at a young age (generally <40), known family history of similar EKG abnormalities, personal history of life-threatening arrhythmia like ventricular fibrillation, and syncope. Brugada diagnosis is an evolving area of cardiology.

Brugada patients have a high incidence of sudden cardiac death. Brugada syndrome is thought to be a cause of some cases of SIDS, as deaths from Brugada syndrome can occur at any point in the lifespan of an affected individual. It is thought to be a possible cause (or possibly the same syndrome, named differently) of other sudden cardiac death syndromes described in Southeast Asia and Africa such as SUNDS (sudden unexpected nocturnal death syndrome), Pokkuri syndrome, and bangangut.

Numerous genes that cause functional abnormalities in the fast Na+ channels which ultimately causes Brugada syndrome have been isolated, but many of the responsible genetic defects have not yet been identified. The description as a fast Na+ channel anomaly and name Brugada syndrome are fairly new in Western medicine (described for the first time in the West in 1992), though as discussed above other non-Western medical practices may have had other names and differing descriptions of this syndrome long predating Western recognition.