Level 10

Acute coronary syndromes—mastering the ST segment

In this chapter you'll learn about the acute coronary syndromes and how they affect the ST segment.

The acute coronary syndromes

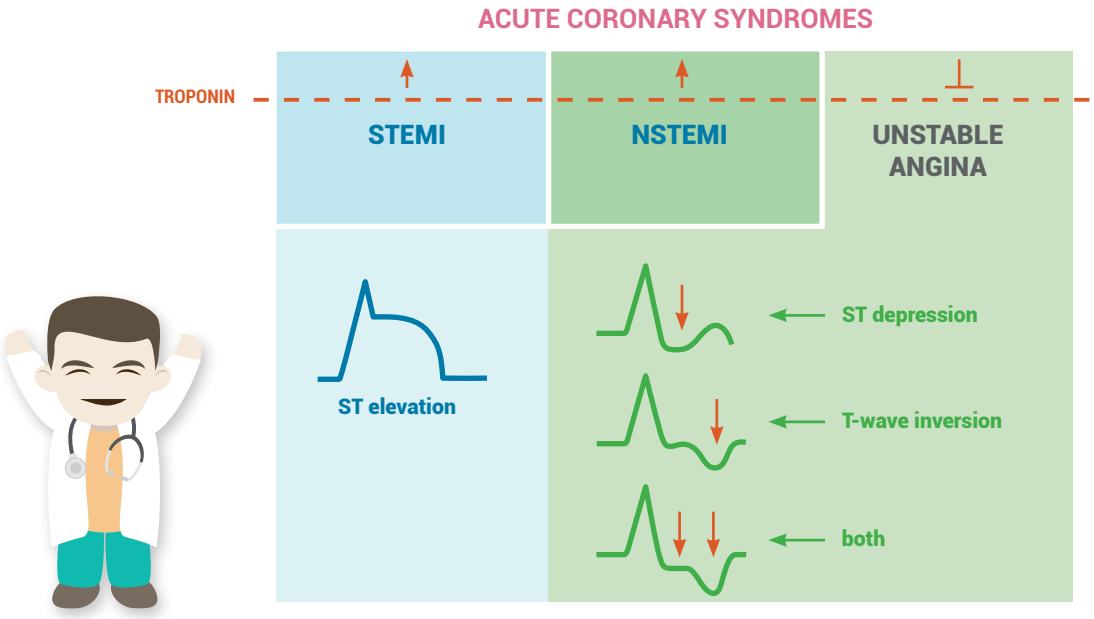
In the previous chapters we discussed what happens to the QRS complex in the setting of myocardial infarction. You learned that the QRS complex "drowns in negativity" when myocardial infarction occurs, which means that R-wave amplitudes decrease and Q waves emerge.

These QRS changes are signs of myocardial necrosis and/or scarring. Scars are usually irreversible, so these changes to the QRS complex are also **irreversible**.

However, myocardial infarction not only affects the QRS complex but also the ST segment, and these changes are usually **transient**.

Acute myocardial infarction is part of the so-called **acute coronary syndromes** (ACS). Acute coronary syndromes result from coronary arteries that are (partly) occluded either by a thrombus or ruptured plaque.

If you want to become a true master of the ST segment, you'll need a thorough understanding of the different acute coronary syndromes. So here they are.



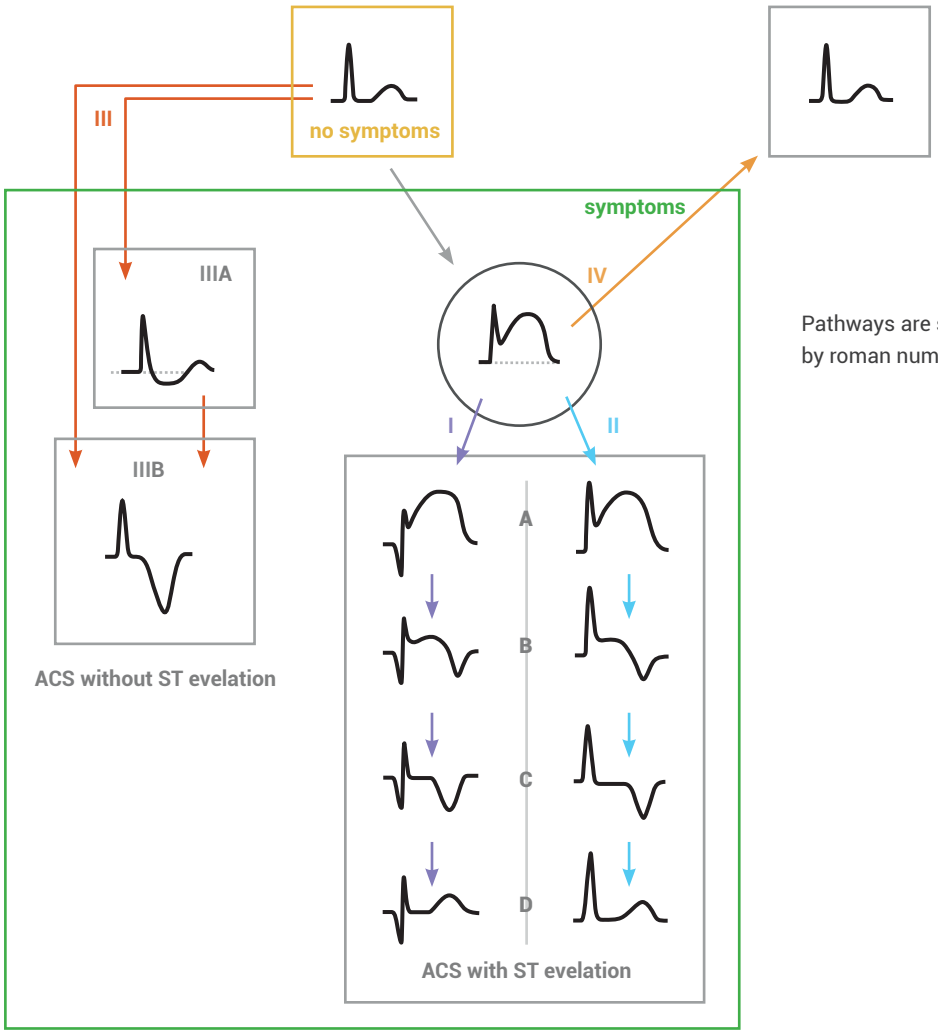
STEMI = ST elevation myocardial infarction
NSTEMI = non-STEMI

A few things to remember:

1. Both **STEMIs** and **NSTEMIs** are characterized by an **elevation of troponin** in the blood. Troponin is elevated because myocardial cells are dying off.
2. As the name implies, **STEMI** comes with an **elevation of the ST segment** (duh!), which discriminates it from NSTEMI and unstable angina.
3. In **NSTEMI and unstable angina**, changes to the ST segment can be subtle; there can be **ST depression, T-wave inversion**, or both.
4. ST changes are very similar in unstable angina and NSTEMI. However, in **unstable angina, troponin** (and other cardiac enzymes) are **NOT elevated**.

The terms "STEMI," "acute myocardial infarction," and "ACS with ST elevation" are sometimes used interchangeably. However, ACS doesn't necessarily lead to myocardial infarction (i.e., necrosis). Therefore, you should think of ST elevation as a sign of acute ischemia rather than infarction, although in general it is its first step.

The figure below shows the different pathways and different stages of acute coronary syndromes.



Pathways I and II—ACS with ST elevation

We start off with the normal heart, shown in yellow.

As symptoms develop, **ST-segment elevation appears** (ischemia). Now three pathways are possible (I, II, and IV). Let's first take a look at ST elevations with Q waves (pathway I in the previous illustration).

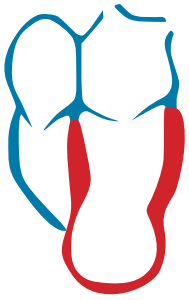
A few hours after the beginning of myocardial ischemia, pathologic **Q waves appear** as a sign of necrosis (IA in the illustration).

As mentioned above, ST elevation is a transient phenomenon. The process from ST elevation to its resolution is called **ST-segment resolution**. It starts with the ST segment going down and the T wave becoming negative (IB).

In the subacute phase of myocardial infarction (IC), the **ST segment has returned to the isoelectric line**, and **the T wave has become negative**. In some patients, this pattern persists forever.

In the chronic phase of myocardial infarction (ID), the T wave becomes positive again. There is no residual sign of infarction in the ST segment or T wave. The myocardial scar is only visible as a Q wave or QS complex.

Time until complete ST-segment resolution is variable. It strongly depends on time to revascularization. Usually, the ST segment starts to go down immediately after complete revascularization. In other cases ST elevation disappears only after several days. Persistence of ST-segment elevations for weeks after myocardial infarction is alarming as it is often caused by a left ventricular aneurysm.

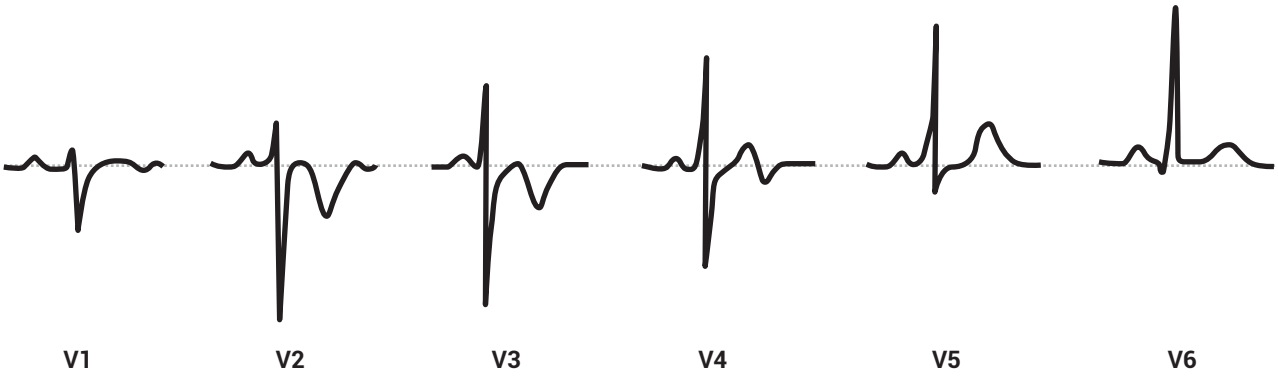


myocardial aneurysm

The time-dependent pattern of changes seen in the ST segment and T wave can also be observed in non–Q-wave infarction (and in patients with peri-myocarditis)—this is pathway II in the illustration.

Pathway III—ACS without ST elevation

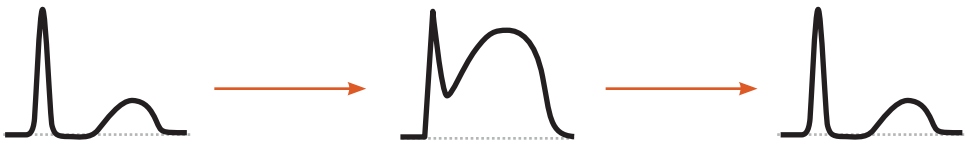
In NSTEMI and unstable angina, symptoms are associated with ST depression (IIIA in the illustration) or T-wave inversion (IIIB). To differentiate between NSTEMI and unstable angina, you'll have to look at whether cardiac enzymes are elevated.



NSTEMI in the territory of the left anterior descending artery (LAD). Leads V2, V3, and V4 are affected. Could also be diagnosed as unstable angina if troponin stays within normal limits.

Pathway IV—Prinzmetal angina: a special case

There is a form of myocardial ischemia that's commonly associated with ST elevation. This disease is called **variant angina** or **Prinzmetal angina**. Chest pain is typically of short duration (15 to 20 minutes) and appears at rest or even during sleep. Unlike other forms of angina, ST elevation returns to baseline immediately after symptoms disappear. Coronary occlusion is thought to be caused by coronary spasm in these cases.



Return to baseline after symptom resolution

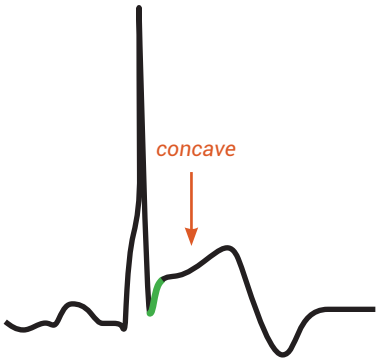
Perimyocarditis

In perimyocarditis, the ST segment is usually also elevated and shows the stages we have seen in IIA through IID. Perimyocarditis is a diffuse disease, and unlike infarction, it's not limited to the perfusion territory of one coronary artery. So it can be seen in most limb leads and many of the precordial leads.

Whenever you see ST elevations in areas that are not supplied by one single artery, you should think of perimyocarditis.

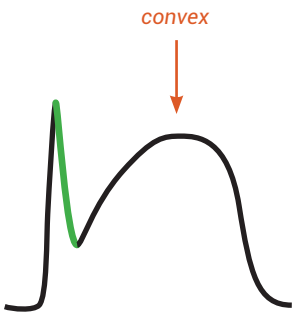
Typically, the ST elevation is not convex, as in myocardial infarction, but rather concave (as seen in the following figure). Furthermore, the ST segment usually originates from the ascending part of the QRS complex in perimyocarditis, whereas in STEMI it usually originates from the descending part of the QRS.

Perimyocarditis



ascending part of
the QRS complex

STEMI

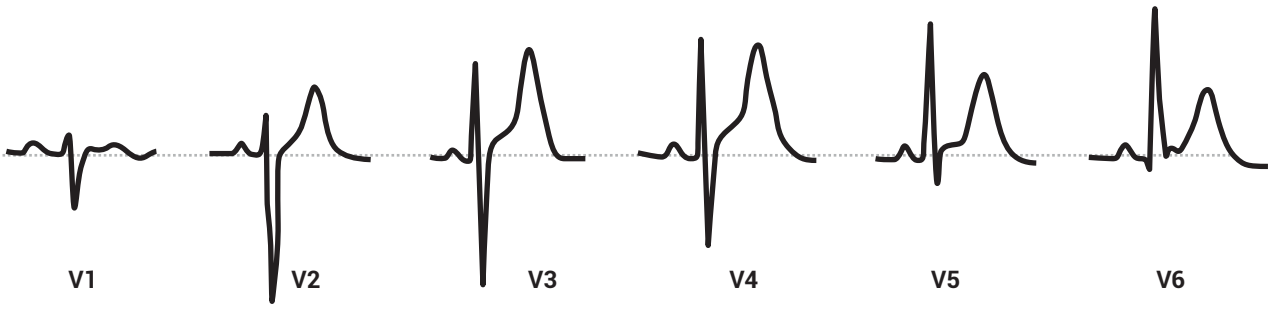


descending part of the QRS complex

In perimyocarditis you can also see the time-dependent changes in ACS with ST elevation, ST resolution, T-wave inversion, etc.

Vagotonia

And finally, there's one more form of ST-segment elevation that's rather innocent compared with the previous ones. This type of ST elevation can be seen in the setting of vagotonia (i.e., an increase in vagal tone). The elevation is up to 0.2 mV in amplitude, and it's usually accompanied by a tall and peaked T wave, as well as a low heart rate of <60 beats per minute.

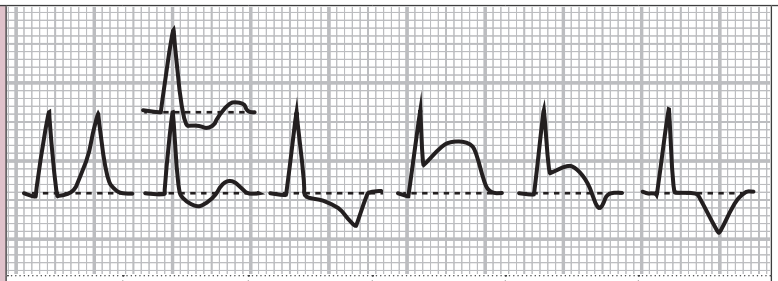


Case of vagotonia with ST elevation and a tall, peaked T wave.

With this knowledge in mind, we can now add the evaluation of the ST segment to the steps of our cookbook. Note that the ST segment should always be evaluated in combination with the QRS complex.

Question	Answer	Diagnosis
1. Rhythm	[coming later]	[coming later]
2. Heart rate	[coming later]	[coming later]
3. P waves	[coming later]	[coming later]
4. PR interval	a) >0.2 s (if PR interval constant for all beats and each P wave is followed by a QRS complex)	I° AV block
	b) <0.12 s and QRS complex normal	LGL syndrome
	c) <0.12 s and visible delta wave	WPW syndrome
5. QRS axis	Determine the axis according to leads I, II, and aVF	normal axis left axis deviation right axis deviation north-west axis
6. QRS duration	a) ≥0.12 s (always think of WPW syndrome as a differential)	complete bundle branch block
	b) >0.1 s and <0.12 s with typical bundle branch block appearance (notching)	incomplete bundle branch block
7. Rotation	Rotation is defined according to the heart's transition zone. Normally the transition zone is located at V4, which means that right ventricular myocardium is located at V1–V3 and left ventricular myocardium is at V5–V6.	transition zone at V5–V6: clockwise rotation transition zone at V1–V3: counterclockwise rotation NOTE: don't evaluate rotation in the setting of myocardial infarction, WPW syndrome, or bundle branch block
8. QRS amplitude	a) QRS amplitude <0.5 mV in all standard leads	low voltage
	b) Positive criteria for left ventricular hypertrophy	left ventricular hypertrophy
	c) Positive criteria for right ventricular hypertrophy	right ventricular hypertrophy
9. QRS infarction signs	abnormal Q waves, QS waves, missing R-wave progression	myocardial infarction; localization according to affected leads

10. ST–T segment



	tall T wave	ST depression	ST depression	ST elevation		negative T	
QRS normal	→						hyperkalemia, vagotonia
QRS normal	→	→					probably ischemia (DD: digitalis)
QRS normal	→	→	→				nonspecific repolarization abnormality
QRS normal	→	→	→	→			acute ischemia, perimyocarditis, variant angina
QRS normal	→	→	→	→	→		STEMI/ perimyocarditis in resolution
QRS normal	→	→	→	→	→	→	STEMI subacute, NSTEMI, perimyocarditis
QRS with Q wave	→	→	→	→	→	→	STEMI acute, STEMI in reso-lution, STEMI subacute
QRS: left ventricular hypertrophy	→	→	→				left ventricular hypertrophy with abnormal repolarization
QRS: right ventricular hypertrophy, bundle branch block, or WPW syndrome	→	→	→				In these situations an ST-segment deviation is almost always present and can-not be interpreted in and of itself. It has to be left out in the ECG report

11. QT duration, T-U waves

[coming later] — [coming later]