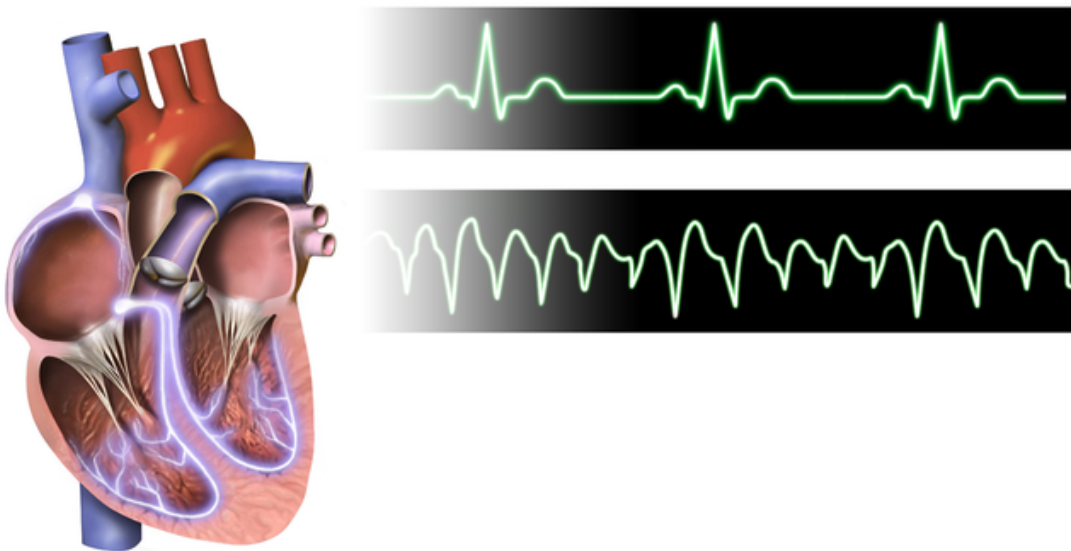


Ventricular Tachycardia (VT) — Types and ECG

[See online here](#)

Ventricular tachyarrhythmias are a group of arrhythmias that result in a heartbeat greater than 100 beats per minute and originate anywhere in the ventricle. There are three main types of ventricular tachyarrhythmias: ventricular fibrillation, monomorphic ventricular tachycardia, and polymorphic ventricular tachycardia. Each one is associated with a high mortality rate. Symptoms include chest pain, palpitations, hemodynamic collapse, and end-organ damage. If the ventricular tachyarrhythmia is suspected, start advanced cardiac life support measures immediately.



Ventricular Tachycardia

Definition of Ventricular Tachycardia

Ventricular tachycardia (VT) refers to any heart rate **greater than 100 beats per minute** that begins in the ventricles. Its **different classification systems** are based on duration, morphology, and hemodynamic effect. For example, a non-sustained VT is short, lasting less than 30 seconds, while a sustained VT is longer than 30 seconds.

Additional classifications are based on the origin of the ectopic beat. If it repeatedly originates in the same place, it is classified as a monomorphic ventricular tachycardia. If it originates in different places around the ventricle, it is called a polymorphic ventricular tachycardia. Torsade de Pointes is an example of a life-threatening, polymorphic ventricular tachycardia.

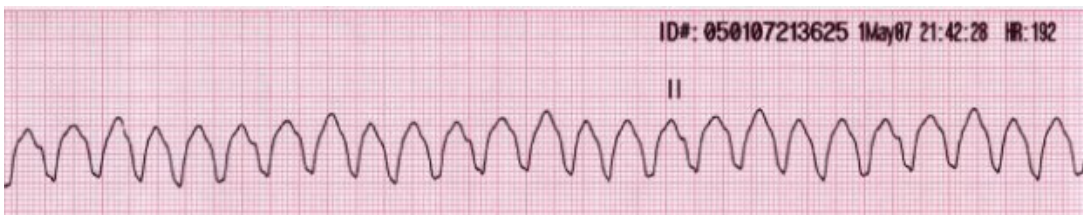
VTs can be caused by a variety of conditions, including:

- [Ischemic heart disease](#)
- Electrolyte abnormalities
- Idiopathic (RVOT)
- Structural heart disease
- Medication side effects

There are three important types of ventricular tachyarrhythmia:

- **Monomorphic ventricular tachycardia**
- **Ventricular fibrillation (VF)**
- **Polymorphic ventricular tachycardia (Torsade de Pointes)**

Monomorphic ventricular tachycardia

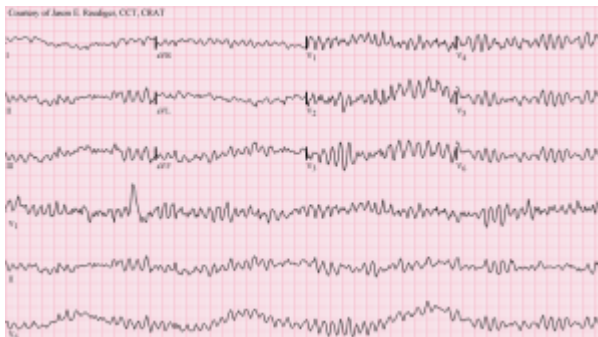


[Image](#): "Monomorphic ventricular tachycardia" by Glenlarsen. License: Public Domain

Monomorphic ventricular tachycardia is a **simple, rapid heart rate with an ectopic beat** originating in the ventricle. There is a risk for **hemodynamic collapse** and **sudden cardiac death** in all patients with ventricular tachycardia.

Ventricular tachycardia is further divided into pulse and pulseless types. In **pulseless tachycardia**, the heart rate is too rapid, and the rhythm is too uncoordinated to pump [blood](#). This condition should be treated like ventricular fibrillation. Advanced cardiac life support measures should be started immediately, including cardiac monitoring, oxygen, medication, and cardioversion, if necessary.

Ventricular fibrillation (VF)



[Image](#): "Ventricular fibrillation" by Jer5150. License: [CC BY-SA 3.0](#)

Ventricular fibrillation (VF) is a more severe ventricular tachyarrhythmia. In this arrhythmia, there is a **rapid and sporadic beat**, resulting in immediate hemodynamic collapse. Death is likely within minutes unless advanced cardiac life support measures are started immediately.

Polymorphic ventricular tachycardia

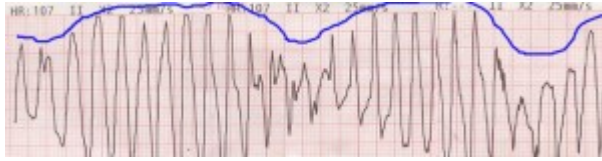


Image: "Torsade de Pointes. The blue line shows the characteristic "twist" around the isoelectric baseline" by Panthro.
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Polymorphic ventricular tachycardia (**Torsade de Pointes**) is a ventricular tachyarrhythmia with a **rapidly changing rate and rhythm**. The rate can change between 150–250 beats per minute, and the amplitude also changes when observed by ECG. This arrhythmia may spontaneously revert to normal or progress into ventricular fibrillation.

Epidemiology of Ventricular Tachycardia

Spread of ventricular tachyarrhythmias

VT makes up **about half the cardiac mortality rate** in the United States. The **incidence increases with age**, as the incidence of coronary artery disease increases. Half of all deaths due to coronary artery disease are caused by VF. Most VF cases occur **more frequently in men**; however, Torsade de Pointes is more frequently found in women.

Etiology of Ventricular Tachycardia

Coronary artery disease & MI

The most common cause of VT is an **ischemic disease**, often after an MI. The risk of VT is also increased by scar formation via surgery or infiltrative cardiomyopathies due to systemic inflammatory diseases, such as [sarcoidosis](#), amyloidosis, and hemochromatosis.

Coronary artery disease is a major risk factor for VF, and extensive coronary artery occlusion is the most common pathological finding. Restoring coronary artery flow reduces the risk of VF.

Non-ischemic cardiomyopathy

Dilated cardiomyopathy increases the risk of VF. It is the most common type of non-ischemic cardiomyopathy and is associated with ventricular remodeling and an increased ventricular volume. **Hypertrophic cardiomyopathy** is frequently a genetic disorder ([autosomal dominant](#)) and also increases the risk of VF.

Hypertrophic cardiomyopathy is the most common cause of ventricular fibrillation in patients under 30 years old. **Takotsubos** cardiomyopathy increases the risk of developing Torsade de Pointes. Takotsubos (AKA broken heart syndrome) is associated with acute heart wall weakness and is often associated with severe stress.

Long QT

Some patients have a naturally long QT interval. The QT interval is measured on an ECG strip from the start of the Q wave to the end of the T wave and represents the **depolarization and repolarization of the ventricles of the heart**.

Certain medications can also prolong the QT interval (antiarrhythmics, antibiotics such as azithromycin, and psychiatric medications such as haloperidol, methadone, and ziprasidone). A prolonged QT interval **can result in polymorphic ventricular tachycardia and Torsade de Pointes**. Other conditions can prolong the QT interval, such as [hypothyroidism](#). It is also common in type 1 [diabetes](#).

Electrolyte imbalance

Electrolyte imbalance can lead to heart arrhythmias. The heart is most sensitive to low levels of **potassium** (hypokalemia), **calcium** (hypocalcemia), and **magnesium** (hypomagnesemia).

Digitalis toxicity

Digitalis toxicity may result from the excessive use of or an abnormal reaction to the drug and increases the risk of developing monomorphic ventricular tachycardia.

Pathology and Pathophysiology of Ventricular Tachycardia

Ventricular tachyarrhythmias are **abnormal, potentially deadly** arrhythmias caused by an abnormal ectopic contraction in the ventricle. The condition may be benign if the ectopic signal is regular and stationary, and the cardiac output is maintained. However, if the signal moves around the ventricle or the rate changes rapidly, then arrhythmia could rapidly result in cardiovascular collapse and death. The most common cause of VT is ischemic change after coronary artery disease or a myocardial ischemic event due to scar formation.

The normal electrical signal slows as it passes through the scar tissue, which allows the surrounding ventricle to repolarize. The slowed signal depolarizes the repolarized ventricular tissue before the supraventricular signal can pass through the ventricular conduction fibers.

The ectopic beat causes the ventricle to beat very fast (greater than 100 beats per minute) and **out of sequence with the atria**. The result is a **drop in cardiac output** and, potentially, sudden death. If the rapid ventricular contraction rate is tolerated, then arrhythmia may cause cardiomyopathy over time.

Torsade de Pointes is closely associated with a prolonged QT interval due to abnormal ion regulation in the heart's [cardiac muscles](#). Any medications and genetic disorders that result in a prolonged or delayed repolarization may cause early afterdepolarization and Torsade de Pointes.

Symptoms of Ventricular Tachycardia

Signs of ventricular tachyarrhythmias

Symptoms include:

- Palpitations
- Lightheadedness
- Syncope
- Sudden cardiac death, especially during ventricular fibrillation

These symptoms often cause anxiety and agitation in the patient. Hypoperfusion to the brain may result in lethargy or coma. Additionally, the patient may describe a sensation of neck fullness and difficulty breathing as the venous blood pools and venous pressure increases.

On physical exam, patients with VT will show **tachycardia**. Due to the low cardiac output, they may also show **hypotension and tachypnea**. VT is also associated with cannon A waves. In this condition, the atria and ventricle contract at the same time, producing a strong pressure wave against the mitral and tricuspid valves of the heart. The wave may be visible, passing up the jugular vein.

Other symptoms include jaw pain and cough. Other conditions produce a cannon A wave, including pulmonary hypertension and complete heart block (third-degree AV block).

The patient may experience some short-term or permanent physical changes after VT has been converted to sinus rhythm. Displacement of the point of maximal impulse is possible. Any structural heart changes that result in valve disease may produce a murmur. An S3 gallop may also develop.

Diagnosis of Ventricular Tachycardia

Ventricular tachyarrhythmias are **potentially life-threatening**. If VT is a concern, then advanced cardiac life support measures should be activated while making the diagnosis.

ECG

An [electrocardiogram](#) and physical findings are sufficient to diagnose VT. **Tachycardia** (greater than 100 beats per minute) **with wide QRS complexes** (> 120 ms) is sufficient for diagnosis.

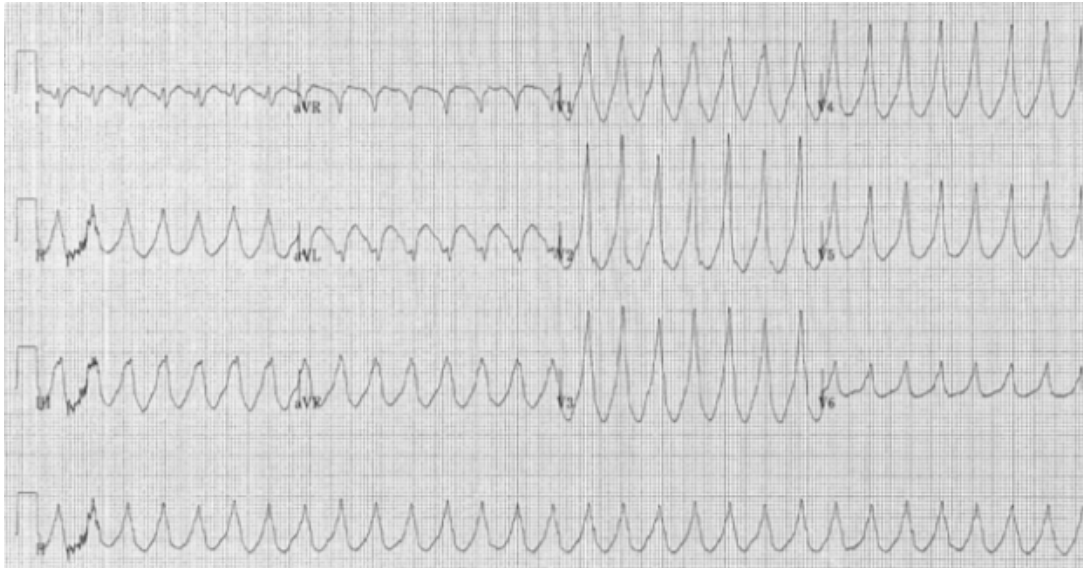


Image: "12 lead electrocardiogram of ventricular tachycardia" by Karthik Sheka, M.D. License: Public Domain [CC BY-SA 2.5](#)

Monomorphic ventricular tachycardia

The ectopic beat is constant, and the QRS wave on ECG will be abnormal but consistent.

Polymorphic ventricular tachycardia

The ectopic beat has many foci in the ventricles and produces a waveform of QRS complexes that appear to twist around an axis. The most common polymorphic ventricular tachycardia condition is Torsade de Pointes or "twisting of the points."

Laboratory studies

Measure electrolytes for abnormally high or low levels, especially **potassium, calcium, and magnesium**. Measure the blood for **medications** that may prolong QT or induce VT, such as digoxin or tricyclic antidepressants. Measure for recreational **drugs** such as cocaine and methamphetamines. Evaluate the heart for damage by measuring **cardiac enzymes**, including troponin I and troponin T.

Differential Diagnoses of Ventricular Tachycardia

- [Atrial flutter](#)
- [Atrial fibrillation](#)
- Premature ventricular contractions
- [Myocardial infarction](#)

Therapy of Ventricular Tachycardia

Treatment of ventricular tachyarrhythmias

Ventricular tachycardia treatment can be broken up into short term and long term treatment.

Short term

Ventricular tachycardia may result in hemodynamic collapse and require **lifesaving measures** to restore normal sinus rhythm. Signs and symptoms of hemodynamic collapse (if these are present, administer lifesaving measures):

- **Dyspnea**
- Chest pain
- Hypotension
- Altered mental status

The first-line treatment for hemodynamic instability secondary to ventricular tachycardia is direct current cardioversion.

A stable patient with prolonged monomorphic ventricular tachycardia but no signs of end-organ damage should be treated with medication-induced cardioversion with **procainamide, sotalol, or lidocaine**. Patients with left ventricular dysfunction should undergo cardioversion with amiodarone or lidocaine. These antiarrhythmics are preferable because they will not exacerbate left heart failure where procainamide may.

A stable patient with polymorphic ventricular tachycardia should be treated with **IV magnesium sulfate, cardioversion**, and an antiarrhythmic that will not prolong the QT interval (procainamide and amiodarone are contraindicated).

If ventricular tachycardia results from digitalis toxicity, treat with anti-digitalis antibody and cardiovert to normal sinus rhythm. If electrolyte imbalance is suspected, then carefully and quickly restore electrolyte concentrations to normal and cardiovert.

Long term

The goal of long-term treatment is to prevent ventricular tachycardia with the most effective and conservative treatment available. Treatment options include medication (**antiarrhythmics**), **ablation**, and **implantable cardioverter-defibrillator implantation**. Additionally, evaluate the QT interval and remove and change medications that may unnecessarily prolong the QT interval.



Cardiovert if unstable!



Antiarrhythmics:

- Amiodarone
- Procainamide
- Sotalol



If regular and monomorphic, may consider adenosine instead

"Treating VT" Image by Lecturio

Progression and Prognosis of Ventricular Tachycardia

Prognosis depends on several characteristics but mainly left heart function. Patients with ventricular tachycardia and left heart dysfunction are much more likely to suffer from hemodynamic collapse and sudden cardiac death.

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