**Cardiomyopathy**

**Cardiomyopathy is a general term for diseases of the heart muscle, where the walls of the heart chambers have become stretched, thickened or stiff. This affects the heart's ability to pump blood around the body.**

Most types of cardiomyopathy are inherited. It can also be caused by other conditions, or risk factors, but for some people the cause is unknown. Cardiomyopathy can affect people of all ages.

**Dilated cardiomyopathy**

In dilated cardiomyopathy the muscle walls of the heart become stretched and thin, so they cannot squeeze (contract) properly to pump blood around the body.

**How serious is it?**

If you have dilated cardiomyopathy, you're at greater risk of heart failure, where the heart fails to pump enough blood around the body at the right pressure.

Heart failure typically causes shortness of breath, extreme tiredness and ankle swelling. Learn more about the symptoms of heart failure.

There's also a risk of heart valve problems, an irregular heartbeat and blood clots. You'll need to have regular appointments with a GP so the condition can be monitored.

**Who's affected?**

Dilated cardiomyopathy can affect both children and adults.

The following can all play a role in the condition:

* inheriting a changed (mutated) gene that makes you more vulnerable to the condition
* an underlying medical condition
* uncontrolled high blood pressure
* an unhealthy lifestyle, such as a lack of vitamins and minerals in your diet, drinking too much alcohol and using recreational drugs
* a viral infection that causes inflammation of the heart muscle
* a heart valve problem
* a disease of the tissues or blood vessels – such as granulomatosis with polyangiitis (GPA), sarcoidosis, amyloidosis, lupus, polyarteritis nodosa, vasculitis or muscular dystrophy
* pregnancy – peripartum cardiomyopathy is rare and sometimes develops during pregnancy, or within 3 months of the baby's birth

But for many people, the cause is unknown.

**Hypertrophic cardiomyopathy**

In hypertrophic cardiomyopathy, the heart muscle cells enlarge and the walls of the heart chambers thicken.

The heart chambers are reduced in size so they cannot hold much blood, and the walls cannot relax properly and may stiffen. Also, the flow of blood through the heart may be obstructed.

**How serious is it?**

In most cases, hypertrophic cardiomyopathy will not have an impact on daily life. Some people do not have any symptoms and do not need treatment.

But that does not mean the condition cannot be serious. Hypertrophic cardiomyopathy is the most common cause of sudden unexpected death in childhood and in young athletes.

The main heart chambers can become stiff, leading to back pressure on the smaller collecting chambers. This can sometimes worsen the symptoms of heart failure and lead to abnormal heart rhythms (atrial fibrillation).

Blood flow from the heart may be reduced or restricted (called obstructive hypertrophic cardiomyopathy).

Also, the mitral heart valve can become leaky, causing blood to leak backwards. Find out more about mitral valve problems.

You'll also be at greater risk of developing a heart infection (endocarditis).These heart changes can cause dizziness, chest pain, shortness of breath and temporary loss of consciousness.

If you have severe hypertrophic cardiomyopathy, you'll need to see your doctor regularly so your condition can be monitored.

Your doctor will advise about the level and amount of exercise you can do and recommend lifestyle changes you can make.

**Who's affected?**

Hypertrophic cardiomyopathy is thought to affect 1 in 500 people in the UK. Most people inherit the disease from their parents.

**Arrhythmogenic cardiomyopathy (ACM)**

Arrhythmogenic cardiomyopathy (ACM) is an inherited condition that affects the left or right ventricles, or both. It's sometimes called arrhythmogenic right ventricular cardiomyopathy (ARCVM).

In arrhythmogenic cardiomyopathy (ACM), the proteins that usually hold the heart muscle cells together are abnormal. Muscle cells can die and the dead muscle tissue is replaced with fatty and fibrous scar tissue.

The walls of the main heart chambers become thin and stretched and cannot pump blood around the body properly.

People with ACM usually have heart rhythm problems. Reduced blood flow from the heart can also lead to . symptoms of heart failure

It can affect teenagers or young adults and has been the reason for some sudden unexplained deaths in young athletes.

There's increasing evidence that prolonged, strenuous exercise makes the symptoms of ACM worse. It's important that people with or at risk of ACM discuss this in detail with their heart specialist (cardiologist).

**Diagnosing cardiomyopathy**

Some cases of cardiomyopathy can be diagnosed after various heart scans and tests, such as:

* electrocardiogram (ECG)
* echocardiogram
* MRI scan
* heart rhythm monitor (24 or 48-hour ECG monitor)
* exercise tests
* a detailed family tree drawn by specialists may be required for the diagnosis of a cardiomyopathy

If you've been diagnosed with an inherited type of cardiomyopathy, you may be advised to have a genetic test to identify the faulty gene (mutation) that caused this.

Your relatives can then be tested for the same mutation and, if they have it, their condition can be monitored and managed early.

**Treating cardiomyopathy**

There's usually no cure for cardiomyopathy, but the treatments can be effective at controlling symptoms and preventing complications. Some types of cardiomyopathy have specific treatments and early diagnosis is very important.

Not everyone with cardiomyopathy will need treatment. Some people only have a mild form of the condition they can control after making a few lifestyle changes.

**Lifestyle changes**

Whether the cause of cardiomyopathy is genetic or not, it should generally help to:

* eat a  healthy diet and do gentle exercise
* quit smoking (if you smoke)
* lose weight (if you're overweight)
* avoid or reduce your intake of alcohol
* get plenty of  sleep (as well as diagnose and treat any underlying  sleep apnoea)
* manage stress
* make sure any underlying condition, such as diabetes, is well controlled

**Medicines**

Medicines may be needed to control blood pressure, correct an abnormal heart rhythm, remove excess fluid or prevent blood clots.

Find out more about:

* treatments for high blood pressure
* beta-blockers to treat an irregular heartbeat or heart failure
* anticoagulants such as  t warfarin o prevent blood clots
* medicines to treat heart failure

**Hospital procedures**

In some people with obstructive hypertrophic cardiomyopathy, the wall dividing the left and right side of the heart (septum) is thickened and bulges into the main heart chamber. They may need to have either:

* an injection of alcohol into their heart – this is to reduce part of the muscle in the septum
* a septal myectomy – heart surgery to remove part of the thickened septum (the mitral valve may be repaired at the same time, if necessary)

Those with heart rhythm problems may need to have arrhythmia ablation. This treatment carefully alters the diseased heart tissue that causes the heart rhythm problems.

Or they may have a device implanted, such as:

* a pacemaker to regulate the heart rate
* an implantable cardioverter defibrillator (ICD) to prevent a life-threatening abnormal heart rhythm

Find out more about having a pacemaker implanted.

Find out more about implantable cardioverter defibrillators from the British Heart Foundation.As a last resort, a heart transplant may be necessary.

**Takotsubo cardiomyopathy (Broken heart syndrome)**

Some people who experience significant emotional or physical stress, such as bereavement or major surgery, go on to experience a temporary heart problem.

The heart muscle becomes suddenly weakened or "stunned", causing the left ventricle (one of the heart's main chambers) to change shape. It may be caused by a surge of hormones, particularly adrenaline, during a period of stress.

The main symptoms are chest pain and breathlessness, similar to those of a heart attack. Always call 999 if you or someone else experiences these.

The condition – known medically as Takotsubo cardiomyopathy, or acute stress cardiomyopathy – is more common in women. It's temporary and reversible. It's unusual for it to happen again.

**Therapies**

Ways to treat cardiomyopathy or an irregular heartbeat without surgery include:

* **Septal ablation.** This shrinks a small part of the thickened heart muscle. It's a treatment option for hypertrophic cardiomyopathy. A doctor threads a thin tube called a catheter to the affected area. Then, alcohol flows through the tube into the artery that sends blood to that area. Septal ablation lets blood flow through the area.
* **Other types of ablation.** A doctor places one or more catheters into blood vessels to the heart. Sensors at the catheter tips use heat or cold energy to create tiny scars in the heart. The scars block irregular heart signals and restore the heartbeat.

**Surgery or other procedures**

Somes types of devices can be placed in the heart with surgery. They can help the heart work better and relieve symptoms. Some help prevent complications. Types of cardiac devices include:

* **Ventricular assist device (VAD).** A VAD helps pump blood from the lower chambers of the heart to the rest of the body. It's also called a mechanical circulatory support device. Most often, a VAD is considered after less invasive treatments don't help. It can be used as a long-term treatment or as a short-term treatment while waiting for a heart transplant.
* **Pacemaker.** A pacemaker is a small device that's placed in the chest to help control the heartbeat.
* **Cardiac resynchronization therapy (CRT) device.** This device can help the chambers of the heart squeeze in a way that's more organized and efficient. It's a treatment option for some people with dilated cardiomyopathy. It can help those with ongoing symptoms, along with signs of a condition called left bundle branch block. The condition causes a delay or blockage along the pathway that electrical signals travel to make the heart beat.
* **Implantable cardioverter-defibrillator (ICD).** This device may be recommended to prevent sudden cardiac arrest, which is a dangerous complication of cardiomyopathy. An ICD tracks heart rhythm and gives electric shocks when needed to control irregular heart rhythms. An ICDdoesn't treat cardiomyopathy. Rather, it watches for and controls irregular rhythms.

Types of surgery used to treat cardiomyopathy include:

* **Septal myectomy.** This is a type of open-heart surgery that can treat hypertrophic cardiomyopathy. A surgeon removes part of the thickened heart muscle wall, called a septum, that separates the two bottom heart chambers, called ventricles. Removing part of the heart muscle improves blood flow through the heart. It also improves a type of heart valve disease called mitral valve regurgitation.
* **Heart transplant.** This is surgery to replace a diseased heart with a donor's healthy heart. It can be a treatment option for end-stage heart failure, when medicines and other treatments no longer work.

SOURCE

NHS (2023) *Cardiomyopathy*. Available at: https://www.nhs.uk/conditions/cardiomyopathy/

**Broken Heart Syndrome**

Broken heart syndrome (takotsubo cardiomyopathy) is a sudden weakness in your heart muscle. This happens right after a physically or emotionally stressful event. The condition can last a few days or weeks. With medicine, most people recover completely.

Broken heart syndrome temporarily weakens your heart muscle.

**What is broken heart syndrome?**

Broken heart syndrome is a short-term condition where some of your heart muscle weakens rapidly. This typically happens after a sudden physical or emotional stressor. When part of your heart isn’t working well, the other parts may work harder.

Weak heart muscle can disrupt your heart’s supply of blood and its ability to pump. If your heart isn’t pumping well, that harms your whole body. Every cell in your body relies on the steady supply of oxygen that your blood carries.

There are many other names for, and types of, broken heart syndrome, including:

* Takotsubo cardiomyopathy.
* Apical ballooning cardiomyopathy (or transient apical ballooning syndrome).
* Stress cardiomyopathy (or stress-induced cardiomyopathy).
* Gebrochenes-Herz syndrome.

**Types of broken heart syndrome**

The four different types of broken heart syndrome are:

* **Apical**. This is the most common type, making up more than 80% of cases. It affects the lower half of your heart.
* **Mid-ventricular**. This type affects the middle section of your heart’s lower chambers(ventricles). The affected area looks like a belt or ring around your heart. The areas of your heart above and below the belt still function as they should.
* **Basal**. Similar to mid-ventricular, the affected area looks like a ring or belt but is higher up. The area below the belt is the only area that functions normally. This type is very rare and makes up about 2% of cases.
* **Focal**. This is the rarest type, making up about 1% of cases, and it involves a much smaller area than the other types. The affected area forms a bulge that sticks out noticeably from the rest of your heart. The opposite side of your heart curves inward toward the bulge.

**How common is this condition?**

Broken heart syndrome occurs in about 2% of people who visit a provider for a suspected heart attack. But researchers believe the true number of cases is higher because providers often don’t recognize the condition.

Takotsubo cardiomyopathy mostly affects women, who make up about 89% of reported cases. This is especially likely after menopause (mean age range of 58 to 77).

One possible explanation is that the hormone estrogen protects your heart against any harmful effects of hormones your body releases in response to stress. As the level of estrogen declines with age, women might be more susceptible to the effects of sudden stress.

**Symptoms and Causes**

**What are the symptoms?**

You may feel broken heart syndrome symptoms within minutes up to hours after the stressful event. The release of stress hormones temporarily stuns your heart muscle, producing symptoms similar to a typical heart attack.

Signs and symptoms of broken heart syndrome include:

* Sudden, severe chest pain (angina) — a main symptom.
* Shortness of breath — a main symptom.
* Weakening of the left ventricle of your heart — a main sign.
* Irregular heartbeats (arrhythmias).
* Low blood pressure (hypotension).
* Heart palpitations.
* Fainting (syncope).

**Broken heart syndrome vs. heart attack**

Because broken heart syndrome has symptoms like those of a heart attack, you may think you’re having one. Both conditions cause shortness of breath and chest pain. But with broken heart syndrome, you don’t have blocked coronary arteries and typically don’t have permanent heart damage. And you usually make a fast and full recovery.

**What causes broken heart syndrome?**

Researchers can’t pinpoint broken heart syndrome causes, but they believe a stressful event like a divorce, car accident or job loss can cause it. When you react to physical or emotional stress, your body releases stress hormones in your blood. Experts think that these hormones temporarily interfere with your heart’s function.

A small percentage of people with broken heart syndrome (takotsubo cardiomyopathy) can’t identify any stressors that may have triggered their episode.

There’s no evidence to suggest that a parent can pass broken heart syndrome down to their children.

**What kinds of emotional and physical stress can cause broken heart syndrome?**

Examples of sudden emotional stressors include:

* Grief from the death of a loved one or other large or meaningful loss (relationship, home, money or a beloved pet).
* Good news (surprise parties, winning the lottery).
* Bad news.
* Traumatic events like accidents or earthquakes.
* Intense fear (public speaking, armed robbery).
* Extreme anger.

Examples of sudden physical stressors include:

* Severe pain.
* An exhausting physical event.
* Health issues, including asthma attacks, difficulty breathing, seizure, stroke, high fever, low blood sugar (hypoglycemia), large blood loss or surgery.

**What are the risk factors for broken heart syndrome?**

You’re more likely to get broken heart syndrome if:

* You’re female.
* You’re older than 50 years of age.
* You’ve had a psychiatric disorder like anxiety or depression.
* You’ve had a neurologic disorder like seizures or a stroke.

**What are the complications?**

Broken heart syndrome complications are rare, but may include:

* Pulmonary edema.
* Rupture of the left ventricle of your heart.
* Blockage of the blood flow from your left ventricle.
* Heart failure.
* Blood clot in the wall of your left ventricle.
* Hypotension (low blood pressure).
* Abnormal heart rhythm (arrhythmia).
* Cardiogenic shock.
* Heart block.
* Death.

**Diagnosis and Tests**

**How is broken heart syndrome diagnosed?**

A healthcare provider will complete a physical exam and review your medical history. Then, they may order several tests, like:

* A blood test to check for a specific enzyme from damaged heart muscle cells.
* An EKG (electrocardiogram).
* Coronary angiography.
* Echocardiography.
* Chest X-ray.
* Heart MRI (magnetic resonance imaging).
* Ventriculogram (a provider injects dye into your heart’s left ventricle, then takes X-rays that show the size and pumping efficiency of this heart chamber).

Imaging can show damaged heart areas, but you need coronary angiography to help rule out a heart attack. Unlike a heart attack, broken heart syndrome doesn’t involve blocked arteries in your heart.

**Management and Treatment**

**What is the treatment for broken heart syndrome?**

Although there’s no cure for broken heart syndrome (takotsubo cardiomyopathy), most people make a full recovery after taking medicine.

Medications for broken heart syndrome treatment include:

1. Aspirin to improve circulation and prevent blood clots.
2. ACE (angiotensin-converting enzyme) inhibitors or ARBs (angiotensin receptor blockers)to lower blood pressure and fight inflammation.
3. Beta-blockers to slow your heart rate.
4. Diuretics to decrease fluid buildup.

If your heart needs help pumping, you may need an intra-aortic balloon pump or left ventricular assist device. This is rare.

**Complications/side effects of treatment**

In general, some of the possible side effects of takotsubo cardiomyopathy treatment include:

* Allergic or negative reactions to the medications or interactions between medications and other drugs you take.
* Bleeding, infections, blood clots, stroke or a heart attack from a left ventricular assist device or intra-aortic balloon pump.

**How soon after treatment will I feel better?**

Most people with broken heart syndrome start to feel better as they receive treatment. That can happen while you’re in the hospital or within hours or days of receiving treatment.

**Prevention**

**Can broken heart syndrome be prevented?**

There are no known ways to prevent broken heart syndrome. However, learning stress management and problem-solving techniques can help you limit physical and emotional stress.

Relaxation techniques can also be helpful. Some examples include:

* Practicing yoga, meditation, journaling or mindfulness.
* Taking a warm bath.
* Lighting scented candles.
* Taking long, deep breaths and slowly exhaling.

Depending on the source of your stress, you may be able to join a support group to talk about your stress and share coping skills. A professional counselor can help, too.

In addition, healthy habits can help you manage physical or emotional stress. These habits include:

* Eating nutritious foods like those in the Mediterranean diet.
* Getting regular exercise (at least five times a week for 30 minutes).
* Getting seven to nine hours of sleep  each night.
* Spending time with others.
* Keeping your medical appointments for checkups and screenings.
* Avoiding tobacco product use, recreational drug use and excessive alcohol use. (Your healthcare provider can guide you to resources to help you quit.)

**Outlook / Prognosis**

**What can I expect if I have broken heart syndrome?**

Broken heart syndrome (takotsubo cardiomyopathy) is a temporary condition for most people. You’ll likely recover without any long-term heart problems.

If an ongoing health problem — like stroke, asthma or seizures — triggered your broken heart syndrome event, check with your healthcare provider for help managing these health issues.

In some cases, your provider may want to do a follow-up echocardiogram about four to six weeks after your event. They’ll want to make sure you don’t have any heart health problems and the left ventricle of your heart is working normally again.

**How long this condition lasts**

People usually make a full recovery a few days to a few weeks after a stress-induced event. But many people have low energy levels for months after getting broken heart syndrome. This can lead to depression. If this happens to you, be sure to ask your provider for help.

**Outlook for broken heart syndrome**

People who get broken heart syndrome from a medical issue (like illness or surgery) tend to have a worse outcome than those who get the condition from an emotional event.

Although men are less likely to have broken heart syndrome, they’re more likely to have a worse prognosis. This is due to having critical illnesses.

Researchers have linked taking ACE inhibitors or ARBs with better survival.

It’s unlikely that you’ll die from broken heart syndrome. Estimates of death from it range from 0% to 8%. In most cases, broken heart syndrome is a temporary condition with a full recovery.

**Living With**

**How do I take care of myself?**

You may need to keep taking prescribed medicines for three to six months. If you have broken heart syndrome, the best thing to do to take care of yourself is to take your medication and see your provider as recommended. These are both important because of the long-term risks that come with takotsubo cardiomyopathy.

You can get broken heart syndrome again or have other health problems weeks or years after the first event. The condition happens again in 4% to 10% of people who have it.

**When should I see my healthcare provider?**

Contact your provider if you notice any new symptoms or changes in existing symptoms, especially if they affect your normal routine. Otherwise, your provider will schedule follow-up appointments as needed.

**When should I go to the ER?**

**Because broken heart syndrome shares symptoms with a heart attack, you should go to a hospital if you have any heart attack symptoms**. Those include:

* Chest pain (angina).
* Trouble breathing or shortness of breath.
* Unexpected fainting or passing out, or multiple instances where you become dizzy and nearly pass out.
* Heart palpitations.
* An unusually slow or fast heartbeat or one that skips or adds beats.

If you have any of the symptoms of broken heart syndrome (takotsubo cardiomyopathy), seek emergency care. Tests are the only way to know if you’re experiencing broken heart syndrome, a heart attack or another medical issue.

**What questions should I ask my doctor?**

Questions you may want to ask your provider include:

* How long do I need to take the medicines you prescribed for me?
* Do I need any follow-up testing?
* How often do I need follow-up appointments with you?

**Additional Common Questions**

**Does day-to-day stress from ordinary life cause broken heart syndrome?**

Most likely, no. Symptoms start after a sudden or extremely stressful event. If you have frequent chest pain or shortness of breath when facing day-to-day moderate stress, see your healthcare provider.

Ongoing symptoms are usually not a sign of broken heart syndrome. Your provider can help you figure out how to cope with stress, prescribe medication if anxiety is a problem, or order tests if they suspect an undiagnosed health problem.

**Is broken heart syndrome serious?**

Rarely, yes. People whose condition is severe or unstable will need close monitoring and more advanced types of care, like mechanical support devices.

Depending on how weak your heart muscle is, your healthcare provider may also recommend cardiac rehabilitation.

It’s common to hear people talk about a “broken heart” when they’re talking about their emotions. But broken heart syndrome (also known as takotsubo cardiomyopathy) is real and can happen after sudden emotional or physical stressors affect you.

The good news is that it’s a temporary condition that usually doesn’t cause any permanent heart damage. But because its symptoms are like those of a heart attack, never try to self-diagnose and convince yourself that you have broken heart syndrome. Always get checked at an emergency care center. Only tests can determine if your heart symptoms are a heart attack, broken heart syndrome or some other health issue.

SOURCE

Cleveland Clinic (2023) *Broken Heart Syndrome*. Available at: https://my.clevelandclinic.org/health/diseases/17857-broken-heart-syndrome

**HEART FAILURE**

What Is Heart Failure?

Heart failure, also known as congestive heart failure, is a condition that develops when your heart doesn’t pump enough blood for your body’s needs. This can happen if your heart can’t fill up with enough blood. It can also happen when your heart is too weak to pump properly. The term "heart failure" does not mean that your heart has stopped. However, heart failure is a serious condition that needs medical care.

More than 6 million adults in the United States have heart failure, according to the Centers for Disease Control and Prevention. Children can also have heart failure, but this health topic focuses on heart failure in adults.

Heart failure can develop suddenly (the acute kind) or over time as your heart gets weaker (the chronic kind). It can affect one or both sides of your heart. Left-sided and right-sided heart failure may have different causes. Most often, heart failure is caused by another medical condition that damages your heart. This includes coronary heart disease, heart inflammation, high blood pressure, cardiomyopathy, or an irregular heartbeat. Heart failure may not cause symptoms right away. But eventually, you may feel tired and short of breath and notice fluid buildup in your lower body, around your stomach, or your neck. Heart failure can damage your liver or kidneys. Other conditions it can lead to include pulmonary hypertension or other heart conditions, such as an irregular heartbeat, heart valve disease, and sudden cardiac arrest.Your doctor will  diagnose  heart failure based on your medical and family history, a physical exam, and results from imaging and blood tests.

Currently, heart failure is a serious condition that has no cure. However,  reatment such as healthy lifestyle changes, medicines, some devices, and procedures can help many people have a higher quality of life. Visit the  Living With section to learn more.

**Symptoms**

Share

Symptoms of heart failure depend on the type of heart failure you have and how serious it is. If you have mild heart failure, you may not notice any symptoms except during hard physical work. Symptoms can depend on whether you have left-sided or right-sided heart failure. However, you can have symptoms of both types. Symptoms usually get worse as your heart grows weaker.

Heart failure can lead to serious and life-threatening complications.

*The picture shows an image of the body and labels which parts are affected by the major symptoms of heart failure.*

One of the first symptoms you may notice is feeling short of breath after routine activities like climbing stairs. As your heart grows weaker, you may notice this while getting dressed or walking across the room. Some people have shortness of breath while lying flat.

Older adults who do not get much physical activity may not experience shortness of breath. However, they may feel tired and confused.

People who have **left-sided** heart failure may have the following symptoms.

* Trouble breathing
* Cough
* Fatigue (extreme tiredness even after rest)
* General weakness
* Bluish color of finger and lips
* Sleepiness and trouble concentrating
* Inability to sleep lying flat

People who have **right-sided**heart failure may also have the following symptoms:

* Nausea (feeling sick in the stomach) and loss of appetite
* Pain in your abdomen (area around your stomach)
* Swelling in your ankles, feet, legs, abdomen, and the veins in your neck
* Needing to pee often
* Weight gain

What problems can heart failure cause?

Heart failure can cause some serious problems.

* **Kidney or liver damage is caused by** reduced blood flow and fluid buildup in your organs.
* **Fluid may build up in or around your lungs.**
* **Malnutrition** from nausea and swelling in your abdomen (the area around your stomach) can make it uncomfortable for you to eat. Reduced blood flow to your stomach can make it harder to absorb nutrients from your food.
* **Other heart conditions** such as an irregular heartbeat, leaking heart valves, or sudden cardiac arrest can be caused by heart failure.
* **Pulmonary hypertension** may also be caused by this condition.

# Diagnosis

## How will I find out if I have heart failure?

Your doctor will diagnose heart failure based on your medical history, a physical exam, and test results. Bring a list of your symptoms to your appointment, including how often they happen and when they started. Also, bring a list of any prescription and over-the-counter medicines you take. Let your provider know if you have any risk factors for heart failure.

You may also be referred to a cardiologist for these tests and treatment. A cardiologist is a doctor who specializes in diagnosing and treating heart diseases.

## Diagnostic tests and procedures

### Blood tests

Your provider may order blood tests to check the levels of certain molecules, such as brain natriuretic peptide  (BNP). These levels rise during heart failure. Blood tests can also show how well your liver and your kidneys are working.

### Tests to measure your ejection fraction

Your provider may order an echocardiography (echo) or other imaging tests to measure your ejection fraction. Your ejection fraction is the percent of the blood in the lower left chamber of your heart (the left ventricle) that is pumped out of your heart with each heartbeat. Ejection fraction measures how well your heart pumps. This helps diagnose the type of heart failure you have and guides your treatment.

* **If 40% or less** of the blood in your left ventricle is pumped out in one beat, you have heart failure with reduced ejection fraction.
* **If 50% or more** of the blood in your left ventricle is pumped out in one beat, you have heart failure with preserved ejection fraction.
* If your ejection fraction is somewhere in between 41% to 49%, you may be diagnosed with heart failure with borderline ejection fraction.

### Other tests

* **Other imaging tests** show how well your heart is working, such as a cardiac CT scan, cardiac MRI, or nuclear heart scan. You may also need cardiac catheterization with coronary angiography to look inside the arteries in your heart and see if they are blocked.
* **Tests for your heart’s electrical activity** may also be necessary. This might include an electrocardiogram (EKG) or a Holter or event monitor that you wear for 24 to 48 hours or more while going about your normal activities.
* **A**  stress test measures how much exercise your body can handle and how well it works during physical activity. Some heart problems are easier to diagnose when your heart is working hard and beating fast.

Causes and Risk Factors

Long-term, or chronic, heart failure is often caused by other medical conditions that damage or overwork your heart. Sudden, or acute, heart failure can be caused by an injury or infection that damages your heart, a heart attack, or a blood clot in your lung.

To understand heart failure, it helps to know how the heart works. The right side of your heart gets oxygen-low blood from your body. It pumps the blood to your lungs to pick up oxygen. The left side of your heart pumps oxygen-rich blood to the rest of your body.

What causes left-sided heart failure?

Left-sided heart failure is more common than right-sided heart failure. There are two types of left-sided heart failure, each based on how well your heart pumps. This measurement is called the ejection fraction. The Diagnosis section has more information about ejection fraction.

* **In heart failure with reduced ejection fraction (HFrEF),** the left side of your heart is weak and can’t pump enough blood to the rest of your body. Chronic conditions that damage or weaken the heart muscles are the main cause of heart failure with reduced ejection fraction. For example, coronary heart disease or a heart attack can prevent your heart muscle from getting enough oxygen. Other causes of this type of heart failure include faulty heart valves, an irregular heartbeat, or heart diseases that you are born with or inherit .
* **In heart failure with preserved ejection fraction (HFpEF),** the left side of your heart is too stiff to fully relax between heartbeats. That means it can't fill up with enough blood to pump out to your body. High blood pressureand other conditions that make your heart work harder are the main causes of heart failure with preserved ejection fraction. Conditions that stiffen the chambers of the heart such as obesity and diabetes are also causes of this type of heart failure. Over time, your heart muscle thickens to adapt, which makes it stiffer.

The Diagnosis section includes more about heart failure with preserved or reduced ejection fraction and how doctors diagnose it.

What causes right-sided heart failure?

Over time left-sided heart failure can lead to right-sided heart failure.

In right-sided heart failure, your heart can't pump enough blood to your lungs to pick up oxygen. Left-sided heart failure is the main cause of right-sided heart failure. That’s because left-sided heart failure can cause blood to build up on the left side of your heart. The build-up of blood raises the pressure in the blood vessels that carry blood from your heart to your lungs. This is called pulmonary hypertension, and it can make the right side of your heart work harder.

Congenital heart defects or conditions that damage the right side of your heart such as abnormal heart valves can also lead to right-side heart failure. The same is true for conditions that damage the lungs, such as chronic obstructive pulmonary disease (COPD).

What raises my risk for heart failure?

Many things can raise your risk of heart failure. Some things you can control, such as your lifestyle habits, but many others are out of your control, including your age, race, or ethnicity. Your risk of heart failure goes up if you have more than one of the following.

* **Aging** can weaken and stiffen your heart**.** People 65 years or older have a higher risk of heart failure. Older adults are also more likely to have other health conditions that cause heart failure.
* **Family history of heart failure** makes your risk of heart failure higher. Genetics may also play a role. Certain changes, or mutations , to genes can make your heart tissue weaker or less flexible.
* **Unhealthy lifestyle habits,** such as an unhealthy diet, smoking, using cocaine or other illegal drugs, heavy alcohol use, and lack of physical activity, increase your risk of heart failure.
* **Heart or blood vessel conditions, serious lung disease, or infections such as HIV or SARS-CoV-2** raise your risk. This is also true for long-term health conditions such as obesity, high blood pressure, diabetes, sleep apnea, chronic kidney disease, anemia,  thyroid  disease, or iron overload. Cancer treatments such as radiation and chemotherapy can injure your heart and raise your risk as well. Atrial fibrillation, a common type of irregular heart rhythm, can also cause heart failure.
* **Black and African American** people are more likely to have heart failure than people of other races, often have more serious cases of heart failure and experience heart failure at a younger age.

Heart failure is common in both men and women, although men often develop heart failure at a younger age than women. Women more commonly have heart failure with preserved ejection fraction (HFpEF), which is when the heart does not fill with enough blood. Men are more likely to have heart failure with reduced ejection fraction (HFrEF), [DEF]. Women often have worse symptoms than men.

**Treatment**

Heart failure has no cure. But treatment can help you live a longer, more active life with fewer symptoms. Treatment depends on the type of heart failure you have and how serious it is.

Your healthcare providers may include a cardiologist (a doctor who specializes in treating heart conditions), nurses, your primary care provider, pharmacists, a dietitian, physical therapists and other members of a cardiac rehabilitation team, and social workers.

Healthy lifestyle changes

Your provider may recommend these heart-healthy lifestyle changes alone or as part of a cardiac rehabilitation plan:

* **Lower your sodium (salt) intake.** Salt may make fluid buildup worse. View our Tips To Reduce Salt and Sodium fact sheet.
* **Aim for a healthy weight** since extra weight can make your heart work harder.
* **Get regular physical activity.** Ask your healthcare provider about how active you should be, including during daily activities, work, leisure time, sex, and exercise. Your level of activity will depend on how serious your heart failure is. Sometimes, your provider might recommend outpatient cardiac rehabilitation services to improve your exercise level and reduce your risk factors.
* **Quit smoking.** Smoking and Your Heart has for more information. For free help quitting smoking, you may call the National Cancer Institute’s Smoking Quitline at 1-877-44U-QUIT (1-877-448-7848).
* **Avoid or limit alcohol.** Your provider may recommend that you limit or stop drinking alcohol. You can find resources and support at the National Institute on Alcohol Abuse and Alcoholism’s Alcohol Treatment Navigator.
* **Manage contributing risk factors.** Controlling some of the factors that may worsen heart failure like blood pressure, heart rhythm, and anemia will often improve heart health.
* **Manage stress.** Learning how to manage stress and cope with problems can improve your mental and physical health. Relaxation techniques, talking to a counselor, and finding a support group can all help lower stress and anxiety.
* **Get good-quality sleep.** Sleep disorders such as sleep apnea are common in people who have heart failure. Treating your sleep disorder helps improve your sleep and may help improve your heart failure symptoms.

Read about healthy lifestyle changes in Heart-Healthy Living.

Medicines

Left-sided heart failure

The following medicines are commonly used to treat heart failure with reduced ejection fraction.

* **Medicines that remove extra sodium and fluid from your body,** including diuretics and aldosterone antagonists (such as spironolactone) lower the amount of blood that the heart must pump. Very high doses of diuretics may cause low blood pressure, kidney disease, and worsening heart failure symptoms. Side effects of aldosterone antagonists can include kidney disease and high potassium levels.
* **Medicines to relax your blood vessels** makes it easier for your heart to pump blood. Examples include ACE inhibitors and angiotensin receptor blockers (ARBs). Possible side effects include cough, low blood pressure, and short-term reduced kidney function.
* **Medicines to slow your heart rate,** such as beta blockers and ivabradine make it easier for your heart to pump blood and can help prevent long-term heart failure from getting worse. Possible side effects include a slow or irregular heart rate, high blood pressure, and fuzzy vision or seeing bright halos.
* **Newer medicines,** including two new groups of medicines approved to lower blood sugar in patients with diabetes, called sodium-glucose cotransporter-2 (SGLT-2) inhibitors and glucagon-like peptide (GLP) agonists. They may also reduce heart failure hospitalizations. Their use in treating heart failure is currently being studied.
* **Digoxin** makes your heart beat stronger and pump more blood. This medicine is mostly used to treat serious heart failure when other medicines do not help improve your symptoms. Side effects may include digestive problems, confusion, and vision problems.

Currently, the main treatments for heart failure with preserved ejection fraction are diuretics. Your doctor also may prescribe blood pressure medicines to help relieve your symptoms.

Right-sided heart failure

If you have right-sided heart failure, your doctor may prescribe two types of medicines.

* **Medicines that remove extra sodium and fluid from your body,** including diuretics and aldosterone antagonists (such as spironolactone) lower the amount of blood that the heart must pump. Very high doses of diuretics may cause low blood pressure, kidney disease, and worsening heart failure symptoms. Side effects of aldosterone antagonists can include kidney disease and high potassium levels.
* **Medicines to relax your blood vessels** make it easier for your heart to pump blood. Examples include angiotensin converting enzyme inhibitors and angiotensin receptor blockers. Possible side effects include cough, low blood pressure, and short-term reduced kidney function.

Procedures and surgeries

If you have heart failure with reduced ejection fraction and it worsens, you may need one of the following medical devices:

* **A** **biventricular pacemaker,** also called cardiac resynchronization therapy, can help both sides of your heart contract at the same time to relieve your symptoms.
* **A mechanical heart pump,** such as a ventricular assist device or a total artificial heart may be used until you have surgery or as a long-term treatment.
* **An** **implantable cardioverter defibrillator** **(ICD)** checks your heart rate and uses electrical pulses to correct irregular heart rhythms that can cause sudden cardiac arrest.

You may also need heart surgery to repair a congenital heart defect or damage to your heart. If your heart failure is life-threatening and other treatments have not worked, you may need a heart transplant.

For people with heart failure and preserved ejection fraction, there are no currently approved devices or procedures to improve symptoms. Researchers are continuing to study possible treatments.

**Living With Heart Failure**

If you have heart failure, you will likely have to follow a treatment plan for the rest of your life. Even with treatment, heart failure often gets worse over time. However, you can take steps to have a higher quality of life.

How to manage heart failure at home

Following your treatment plan can help relieve symptoms and make daily activities easier. It also can lower the chance that you’ll have to go to the hospital.

* **Take your medicines as prescribed.** Tell your provider if you have side effects from any of your medicines. They might adjust the dose or change the type of medicine you take to reduce side effects.
* **Make** **heart-healthy lifestyle changes** recommended by your provider. Habits can be hard to change. Let your provider know if you’re having a hard time sticking with any of the changes. You may also be asked to limit the amount of salt and liquids that you drink to reduce fluid buildup.
* **Get medical care for other conditions** that can worsen heart failure. These include obesity, diabetes, high blood pressure, sleep apnea, and lung, kidney, or liver disease. Tell your provider and pharmacist about all the medicines you’re taking. Taking medicines together can raise the risk of side effects. Also, certain medicines can worsen your heart failure symptoms.

Know when to seek help

Watch for signs that heart failure is getting worse, such as new or worsening symptoms. Weight gain, ankle swelling, or increasing shortness of breath may mean that fluids are building up in your body. Ask your provider how often you should check your weight and when to report weight changes.

Your symptoms may suddenly get worse. Ask when to make an office visit or get emergency care. Keep the following handy:

* Phone numbers for your provider, the hospital, and someone who can take you for medical care
* Directions to the doctor's office and hospital
* A list of all the medicines you’re taking

Get support and know your options

Living with heart failure may cause fear, anxiety, depression, and stress. Talk to your healthcare provider or a professional counselor. They can help you find or learn ways to cope.

* **Get treatment for depression.** If you are depressed, your provider may recommend medicines or other treatments that can improve your quality of life.
* **Join a patient support group.** You can learn how other people who have similar symptoms have coped with them. Your provider may be able to help you find local support groups, or you can check with an area medical center.
* **Seek support from family and friends.** Letting your loved ones know how you feel and what they can do to help can help lower your stress and anxiety.

**Know your treatment options.** If your heart failure is very serious, palliative or hospice care can improve your quality of life and help make your daily life more comfortable. This type of care focuses on managing your symptoms, helping you avoid unnecessary tests or treatments, and providing support to your loved ones.

SOURCE

National Heart, Lung, and Blood Institute (2022) *Heart Failure - What Is Heart Failure?*. Available at: https://www.nhlbi.nih.gov/health/heart-failure

**Heart Failure (HF)**

**(Congestive Heart Failure)**

**Heart failure (HF) is a syndrome of ventricular dysfunction. Left ventricular (LV) failure causes shortness of breath and fatigue, and right ventricular (RV) failure causes peripheral and abdominal fluid accumulation; the ventricles can be involved together or separately. Diagnosis is initially clinical, supported by chest x-ray, echocardiography, and levels of plasma natriuretic peptides. Treatment includes patient education, diuretics, angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, beta-blockers, aldosterone antagonists, sodium-glucose cotransporter-2 inhibitors, neprilysin inhibitors, sinus node inhibitors, specialized implantable pacemakers/defibrillators and other devices, and correction of the cause(s) of the HF syndrome.**

* **Physiology**|
* **Pathophysiology**|
* **Etiology**|
* **Classification**|
* **Symptoms and Signs**|
* **Diagnosis**|
* **Prognosis**|
* **Treatment**|
* **Key Points**|
* **More Information**

**Topic Resources**

* + **3D Models (0)**
  + **Audios (0)**
  + **Calculators (0)**
  + **Images (7)**
  + **Tables (2)**
  + **Videos (1)**

**Heart failure affects about 6.5 million people in the US; > 960,000 new cases occur each year. About 26 million people are affected worldwide.**

**(See also Heart Failure in Children.)**

**Physiology of Heart Failure**

**Cardiac contractility (force and velocity of contraction), ventricular performance, and myocardial oxygen requirements are determined by**

* + **Preload**
  + **Afterload**
  + **Substrate availability (eg, oxygen, fatty acids, glucose)**
  + **Heart rate and rhythm**
  + **Amount of viable myocardium**

**Cardiac output (CO) is the product of stroke volume and heart rate; it is also affected by venous return, peripheral vascular tone, and neurohumoral factors.**

**Preload is the loading condition of the heart at the end of its relaxation and filling phase (diastole) just before contraction (systole). Preload represents the degree of end-diastolic fiber stretch and end-diastolic volume, which is influenced by ventricular diastolic pressure and the composition of the myocardial wall. Typically, left ventricular (LV) end-diastolic pressure, especially if higher than normal, is a reasonable measure of preload. LV dilation, hypertrophy, and changes in myocardial distensibility (compliance) modify preload.**

**Afterload is the force resisting myocardial fiber contraction at the start of systole. It is determined by LV chamber pressure, radius, and wall thickness at the time the aortic valve opens. Clinically, systemic systolic blood pressure at or shortly after the aortic valve opens correlates with peak systolic wall stress and approximates afterload.**

**The Frank-Starling principle describes the relationship between preload and cardiac performance. It states that, normally, systolic contractile performance (represented by stroke volume or CO) is proportional to preload within the normal physiologic range (see figure Frank-Starling principle). Contractility is difficult to measure clinically (because it requires cardiac catheterization with pressure-volume analysis) but is reasonably reflected by the ejection fraction (EF), which is the percentage of end-diastolic volume ejected with each contraction (stroke volume/end-diastolic volume). EF can generally be adequately assessed noninvasively with echocardiography, nuclear imaging, or MRI.**

**The force-frequency relationship refers to the phenomenon in which repetitive stimulation of a muscle within a certain frequency range results in increased force of contraction. Normal cardiac muscle at typical heart rates exhibits a positive force-frequency relationship, so a faster rate causes stronger contraction (and corresponding greater substrate requirements). During some types of heart failure, the force-frequency relationship may become negative, so that myocardial contractility decreases as heart rate increases above a certain rate.**

**Cardiac reserve is the ability of the heart to increase its performance above resting levels in response to emotional or physical stress; body oxygen consumption may increase from 250 to ≥ 1500 mL/minute during maximal exertion. Mechanisms include**

* + **Increasing heart rate**
  + **Increasing systolic and diastolic volumes**
  + **Increasing stroke volume**
  + **Increasing tissue extraction of oxygen (the difference between oxygen content in arterial blood and in mixed venous or pulmonary artery blood)**

**In well-trained young adults during maximal exercise, heart rate may increase from 55 to 70 beats/minute at rest to 180 beats/minute, and CO may increase from 6 to ≥ 25 L/minute. At rest, arterial blood contains about 18 mL oxygen/dL of blood, and mixed venous or pulmonary artery blood contains about 14 mL/dL. Oxygen extraction is thus about 4 mL/dL. When demand is increased, oxygen extraction may increase to 12 to 14 mL/dL. This mechanism also helps compensate for reduced tissue blood flow in heart failure.**

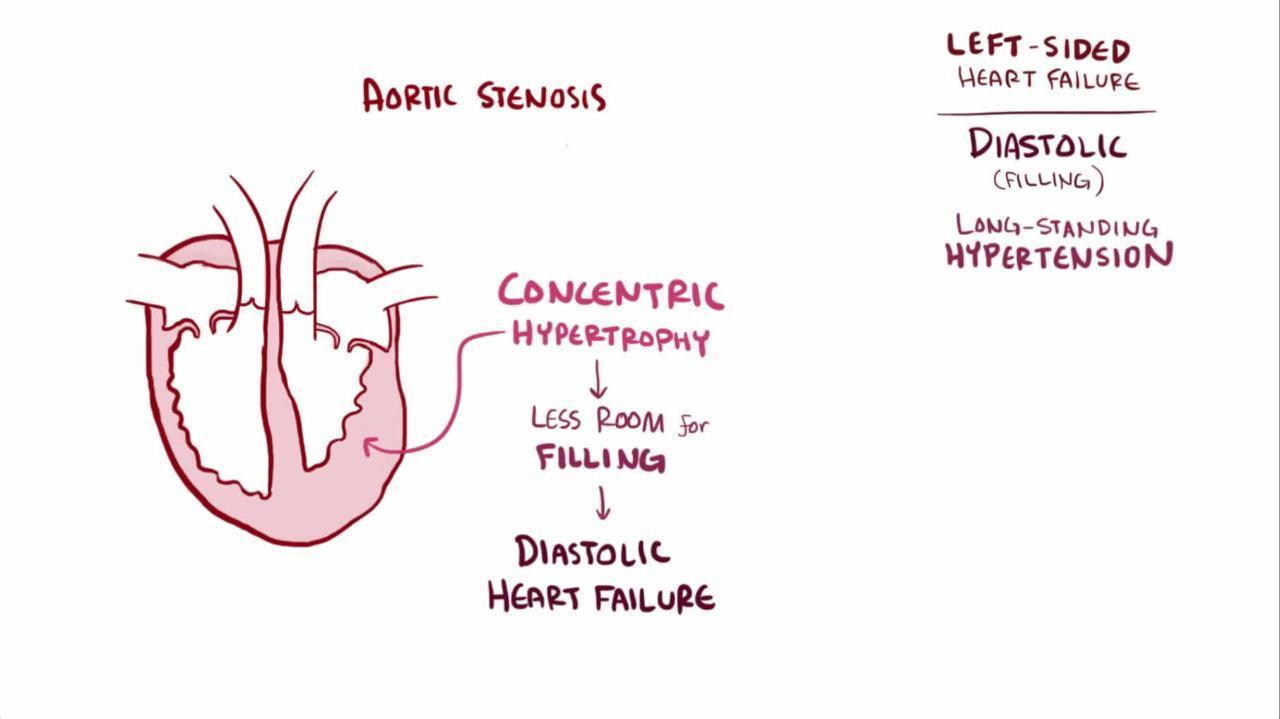
**Frank-Starling principle**

| Normally (top curve), as preload increases, cardiac performance also increases. However at a certain point, performance plateaus, then declines. In heart failure (HF) due to systolic dysfunction (bottom curve), the overall curve shifts downward, reflecting reduced cardiac performance at a given preload, and as preload increases, cardiac performance increases less. With treatment (middle curve), performance is improved, although not normalized.  Frank-Starling principle |
| --- |

**Pathophysiology of Heart Failure**

**In heart failure, the heart may not provide tissues with adequate blood for metabolic needs, and cardiac-related elevation of pulmonary or systemic venous pressures may result in organ congestion. This condition can result from abnormalities of systolic or diastolic function or, commonly, both. Although a primary abnormality can be a change in cardiomyocyte function, there are also changes in collagen turnover of the extracellular matrix. Cardiac structural defects (eg, congenital defects, valvular disorders), rhythm abnormalities (including persistently high heart rate), and high metabolic demands (eg, due to thyrotoxicosis) also can cause HF.**

**Overview of Heart Failure**

****

**VIDEO**

**Heart failure with reduced ejection fraction (HFrEF)**

**In HFrEF (also called systolic HF), global LV systolic dysfunction predominates. The LV contracts poorly and empties inadequately, leading to**

* + **Increased diastolic volume and pressure**
  + **Decreased ejection fraction (≤ 40%)**

**Many defects in energy utilization, energy supply, electrophysiologic functions, and contractile element interaction occur, with abnormalities in intracellular calcium modulation and cAMP production.**

**Predominant systolic dysfunction is common in heat failure due to myocardial infarction, myocarditis, and dilated cardiomyopathy. Systolic dysfunction may affect primarily the LV or the right ventricle (RV); LV failure often leads to RV failure.**

**Heart failure with preserved ejection fraction (HFpEF)**

**In HFpEF (also called diastolic heart failure), LV filling is impaired, resulting in**

* + **Increased LV end-diastolic pressure at rest or during exertion**
  + **Usually, normal LV end-diastolic volume**

**Global contractility and hence ejection fraction remain normal (≥ 50%).**

**However, in some patients, marked restriction to LV filling can cause inappropriately low LV end-diastolic volume and thus cause low CO (cardiac output) and systemic symptoms. Elevated left atrial pressures can cause pulmonary hypertension and pulmonary congestion.**

**Diastolic dysfunction usually results from impaired ventricular relaxation (an active process), increased ventricular stiffness, valvular disease, or constrictive pericarditis. Acute myocardial ischemia is also a cause of diastolic dysfunction. Resistance to filling increases with age, reflecting both cardiomyocyte dysfunction and cardiomyocyte loss, and increased interstitial collagen deposition; thus, diastolic dysfunction is particularly common among older adults. Diastolic dysfunction predominates in hypertrophic cardiomyopathy, other disorders with ventricular hypertrophy (eg, hypertension, significant aortic stenosis), and amyloid infiltration of the myocardium. LV filling and function may also be impaired if marked increases in RV pressure shift the interventricular septum to the left.**

**Diastolic dysfunction has increasingly been recognized as a cause of HF. Estimates vary, but about 50% of patients with heart failure have HFpEF; the prevalence increases with age and in patients with diabetes. It is now known that HFpEF is a complex, heterogenous, multiorgan, systemic syndrome, often with multiple concomitant pathophysiologies. Current data suggest that multiple comorbidities (eg, obesity, hypertension, diabetes, chronic kidney disease) lead to systemic inflammation, widespread endothelial dysfunction, cardiac microvascular dysfunction, and, ultimately, molecular changes in the heart that cause increased myocardial fibrosis and ventricular stiffening. Thus, although HFrEF is typically associated with primary myocardial injury, HFpEF may be associated with secondary myocardial injury due to abnormalities in the periphery.**

**Heart failure with mildly reduced ejection fraction (HFmrEF)**

**International societies have put forth the concept of HF with mildly reduced ejection fraction (HFmrEF), in which patients have an LV ejection fraction of 41 to 49%. It is unclear whether this group is a distinct population or consists of a mixture of patients with either HFpEF or HFrEF.**

**LV failure**

**In heart failure that involves left ventricular dysfunction, CO decreases and pulmonary venous pressure increases. When pulmonary capillary pressure exceeds the oncotic pressure of plasma proteins (about 24 mm Hg), fluid extravasates from the capillaries into the interstitial space and alveoli, reducing pulmonary compliance and increasing the work of breathing. Lymphatic drainage increases but cannot compensate for the increase in pulmonary fluid. Marked fluid accumulation in alveoli (pulmonary edema) significantly alters ventilation-perfusion (V/Q) relationships: Deoxygenated pulmonary arterial blood passes through poorly ventilated alveoli, decreasing systemic arterial oxygenation (PaO2) and causing dyspnea. However, dyspnea may occur before V/Q abnormalities, probably because of elevated pulmonary venous pressure and increased work of breathing; the precise mechanism is unclear.**

**In severe or chronic LV failure, pleural effusions characteristically develop, further aggravating dyspnea. Minute ventilation increases; thus, PaCO2 decreases and blood pH increases (respiratory alkalosis). Marked interstitial edema of the small airways may interfere with ventilation, elevating PaCO2—a sign of impending respiratory failure.**

**RV failure**

**In heart failure that involves right ventricular dysfunction, systemic venous pressure increases, causing fluid extravasation and consequent edema, primarily in dependent tissues (feet and ankles of ambulatory patients) and abdominal viscera. The liver is most severely affected, but the stomach and intestine also become congested; fluid accumulation in the peritoneal cavity (ascites) can occur. RV failure commonly causes moderate hepatic dysfunction, with usually modest increases in conjugated and unconjugated bilirubin, PT (prothrombin time), and hepatic enzymes (particularly alkaline phosphatase and gamma-glutamyl transpeptidase [GGT]). The impaired liver breaks down less aldosterone, further contributing to fluid accumulation. Chronic venous congestion in the viscera can cause anorexia, malabsorption of nutrients and drugs, protein-losing enteropathy (characterized by diarrhea and marked hypoalbuminemia), chronic gastrointestinal blood loss, and rarely ischemic bowel infarction.**

**Cardiac response**

**In HFrEF, left ventricular systolic function is grossly impaired; therefore, a higher preload is required to maintain CO. As a result, the ventricles are remodeled over time: During remodelling, the LV becomes less ovoid and more spherical, dilates, and hypertrophies; the RV dilates and may hypertrophy. Initially compensatory, remodelling ultimately is associated with adverse outcomes because the changes eventually increase diastolic stiffness and wall tension (ie, diastolic dysfunction develops), compromising cardiac performance, especially during physical stress. Increased wall stress raises oxygen demand and accelerates apoptosis (programmed cell death) of myocardial cells. Dilation of the ventricles can also cause mitral or tricuspid valve regurgitation (due to annular dilation) with further increases in end-diastolic volumes.**

**Hemodynamic responses**

**With reduced CO, oxygen delivery to the tissues is maintained by increasing oxygen extraction from the blood and sometimes shifting the oxyhemoglobin dissociation curve (see figure Oxyhemoglobin dissociation curve) to the right to favor oxygen release.**

**Reduced CO with lower systemic blood pressure activates arterial baroreflexes, increasing sympathetic tone and decreasing parasympathetic tone. As a result, heart rate and myocardial contractility increase, arterioles in selected vascular beds constrict, venoconstriction occurs, and sodium and water are retained. These changes compensate for reduced ventricular performance and help maintain hemodynamic homeostasis in the early stages of heart failure. However, these compensatory changes increase cardiac work, preload, and afterload; reduce coronary and renal perfusion; cause fluid accumulation resulting in congestion; increase potassium excretion; and may cause cardiomyocyte necrosis and arrhythmias.**

**Renal responses**

**As cardiac function deteriorates, renal blood flow decreases (due to low cardiac output). In addition, renal venous pressures increase, leading to renal venous congestion. These changes both result in a decrease in GFR (glomerular filtration rate), and blood flow within the kidneys is redistributed. The filtration fraction and filtered sodium decrease, but tubular resorption increases, leading to sodium and water retention. Blood flow is further redistributed away from the kidneys during exercise, but renal blood flow improves during rest.**

**Decreased perfusion of the kidneys (and possibly decreased arterial systolic stretch secondary to declining ventricular function) activates the renin-angiotensin-aldosterone system (RAAS), increasing sodium and water retention and renal and peripheral vascular tone. These effects are amplified by the intense sympathetic activation accompanying heart failure.**

**The renin-angiotensin-aldosterone-vasopressin (antidiuretic hormone [ADH]) system causes a cascade of potentially deleterious long-term effects. Angiotensin II worsens heart failure by causing vasoconstriction, including efferent renal vasoconstriction, and by increasing aldosterone production, which enhances sodium reabsorption in the distal nephron and also causes myocardial and vascular collagen deposition and fibrosis. Angiotensin II increases norepinephrine release, stimulates release of vasopressin, and triggers apoptosis. Angiotensin II may be involved in vascular and myocardial hypertrophy, thus contributing to the remodeling of the heart and peripheral vasculature, potentially worsening HF. Aldosterone can be synthesized in the heart and vasculature independently of angiotensin II (perhaps mediated by corticotropin, nitric oxide, free radicals, and other stimuli) and may have deleterious effects in these organs.**

**Heart failure that causes progressive renal dysfunction (including renal dysfunction caused by drugs used to treat HF) contributes to worsening HF and has been termed the cardiorenal syndrome.**

**Neurohumoral responses**

**In conditions of stress, neurohumoral responses help increase heart function and maintain blood pressure and organ perfusion, but chronic activation of these responses is detrimental to the normal balance between myocardial-stimulating and vasoconstricting hormones and between myocardial-relaxing and vasodilating hormones.**

**The heart contains many neurohumoral receptors (alpha-1, beta-1, beta-2, beta-3, angiotensin II type 1 [AT1] and type 2 [AT2], muscarinic, endothelin, serotonin, adenosine, cytokine, natriuretic peptides); the roles of all of these receptors are not yet fully defined. In patients with heart failure, beta-1 receptors (which constitute 70% of cardiac beta receptors) are downregulated, probably in response to intense sympathetic activation. The result of downregulation is impaired myocyte contractility and increased heart rate.**

**Plasma norepinephrine levels are increased, largely reflecting sympathetic nerve stimulation as plasma epinephrine levels are not increased. Detrimental effects include vasoconstriction with increased preload and afterload, direct myocardial damage including apoptosis, reduced renal blood flow, and activation of other neurohumoral systems, including the renin-angiotensin-aldosterone-vasopressin system.**

**Vasopressin is released in response to a fall in blood pressure via various neurohormonal stimuli. Increased vasopressindecreases renal excretion of free water, possibly contributing to hyponatremia in heart failure. Vasopressin levels in patients with HF and normal blood pressure vary.**

**Atrial natriuretic peptide is released in response to increased atrial volume and pressure; brain (B-type) natriuretic peptide (BNP) is released from the ventricle in response to ventricular stretching. These peptides enhance renal excretion of sodium, but in patients with HF, the effect is blunted by decreased renal perfusion pressure, receptor downregulation, and perhaps enhanced enzymatic degradation. In addition, elevated levels of natriuretic peptides exert a counter-regulatory effect on the renin-angiotensin-aldosterone system and catecholamine stimulation.**

**Because endothelial dysfunction occurs in HF, fewer endogenous vasodilators (eg, nitric oxide, prostaglandins) are produced, and more endogenous vasoconstrictors (eg, endothelin) are produced, thus increasing afterload.**

**The failing heart and other organs produce tumor necrosis factor (TNF) alpha. This cytokine increases catabolism and is possibly responsible for cardiac cachexia (loss of lean tissue ≥ 10%), which may accompany severely symptomatic HF, and for other detrimental changes. The failing heart also undergoes metabolic changes with increased free fatty acid utilization and decreased glucose utilization; these changes may become therapeutic targets.**

**Changes with aging**

**Age-related changes in the heart and cardiovascular system lower the threshold for expression of heart failure. Interstitial collagen within the myocardium increases, the myocardium stiffens, and myocardial relaxation is prolonged. These changes lead to a significant reduction in diastolic left ventricular function, even in healthy older people. Modest decline in systolic function also occurs with aging. An age-related decrease in myocardial and vascular responsiveness to beta-adrenergic stimulation further impairs the ability of the cardiovascular system to respond to increased work demands.**

**As a result of these changes, peak exercise capacity decreases significantly (about 8%/decade after age 30), and CO at peak exercise decreases more modestly. This decline can be slowed by regular physical exercise. Thus, older patients are more prone than are younger ones to develop HF symptoms in response to the stress of systemic disorders or relatively modest cardiovascular insults. Stressors include infections (particularly pneumonia), hyperthyroidism, anemia, hypertension, myocardial ischemia, hypoxia, hyperthermia, renal failure, perioperative IV fluid loads, nonadherence to drug regimens or to low-salt diets, and use of certain drugs (particularly NSAIDs [nonsteroidal anti-inflammatory drugs]).**

**Etiology of Heart Failure**

**Both cardiac and systemic factors can impair cardiac performance and cause or aggravate heart failure.**

**TABLE**

**Causes of Heart Failure**

| **Type** | **Examples** |
| --- | --- |
| Cardiac | |
| Myocardial damage | Cardiomyopathy  Myocardial infarction  Myocarditis  Some chemotherapy drugs |
| Valvular disorders | Aortic stenosis  Mitral regurgitation  Tricuspid regurgitation |
| Arrhythmias | Bradyarrhythmias  Tachyarrhythmias |
| Conduction defects | AV node block  Left bundle branch block |
| Reduced substrate availability (eg, of free fatty acids or glucose) | Ischemia |
| Infiltrative or matrix disorders | Amyloidosis  Chronic fibrosis (eg,  Hemochromatosis |
| Systemic | |
| Disorders that increase demand for CO | Anemia  Hyperthyroidism  Paget disease |
| Disorders that increase resistance to output (afterload) | Aortic stenosis  Hypertension |
| AV = atrioventricular; CO = cardiac output. | |

**Classification of Heart Failure**

**The most common classification of heart failure currently in use stratifies patients into**

* + **Heart failure with reduced ejection fraction ("systolic HF")**
  + **Heart failure with preserved ejection fraction ("diastolic HF")**

**Heart failure with reduced ejection fraction (HFrEF) is defined as heart failure with left ventricular ejection fraction (LVEF) ≤ 40%.**

**Heart failure with preserved ejection fraction (HFpEF) is defined as heart failure with LVEF ≥ 50%.**

**Patients with LVEF between 41% and 49% are in an intermediate zone, and have recently been categorized as HF with mildly reduced ejection fraction (HFmrEF—**[**1**](https://www.msdmanuals.com/professional/cardiovascular-disorders/heart-failure/heart-failure-hf#v31269678)**).**

**The traditional distinction between left and right ventricular failure is somewhat misleading because the heart is an integrated pump, and changes in one chamber ultimately affect the whole heart. However, these terms indicate the major site of pathology leading to heart failure and can be useful for initial evaluation and treatment. Other common descriptive terms for heart failure include acute or chronic; high output or low output; dilated or nondilated; and ischemic, hypertensive, or idiopathic dilated cardiomyopathy. Treatment differs based on whether the presentation is acute or chronic HF.**

**LV failure characteristically develops in ischemic heart disease, hypertension, mitral regurgitation, aortic regurgitation, aortic stenosis, most forms of cardiomyopathy, and congenital heart disorders (eg, ventricular septal defect, patent ductus arteriosuswith large shunts).**

**RV failure is most commonly caused by previous LV failure (which increases pulmonary venous pressure and leads to pulmonary hypertension, thus overloading the RV) or by a severe lung disorder (in which case it is called cor pulmonale). Other causes are multiple pulmonary emboli, RV infarction, pulmonary arterial hypertension, tricuspid regurgitation, tricuspid stenosis, mitral stenosis, pulmonary artery stenosis, pulmonic valve stenosis, pulmonary venous occlusive disease, arrhythmogenic RV cardiomyopathy, or congenital disorders such as Ebstein anomaly or Eisenmenger syndrome. Some conditions mimic RV failure, except cardiac function may be normal; they include volume overload and increased systemic venous pressure in polycythemiaor overtransfusion, acute kidney injury with retention of sodium and water, obstruction of either vena cava, and hypoproteinemia due to any cause resulting in low plasma oncotic pressure and peripheral edema.**

**Biventricular failure results from disorders that affect the whole myocardium (eg, viral myocarditis, amyloidosis, Chagas disease) or long-standing LV failure causing RV failure.**

**High-output HF results from a persistently high cardiac output, which may eventually result in an inability of a normal heart to maintain adequate output. Conditions that may increase CO (cardiac output) include severe anemia, end-stage liver disease, beriberi, thyrotoxicosis, advanced Paget disease, arteriovenous fistula, and persistent tachycardia.**

**Cardiomyopathy is a general term indicating disease of the myocardium. Most commonly, the term refers to a primary disorder of the ventricular myocardium that is not caused by congenital anatomic defects; valvular, systemic, or pulmonary vascular disorders; isolated pericardial, nodal, or conduction system disorders; or epicardial coronary artery disease (CAD). The term is sometimes used to reflect etiology (eg, ischemic vs hypertensive cardiomyopathy). Cardiomyopathy does not always lead to symptomatic HF. It is often idiopathic and is classified as dilated, congestive, hypertrophic, infiltrative-restrictive, or apical-ballooning cardiomyopathy (also known as takotsubo or stress cardiomyopathy).**

**Classification reference**

* 1. **1. Heidenreich PA, Bozkurt B, Aguilar D, et al: 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*145:e876–e894, 2022, doi: 10.1161/CIR.0000000000001062**

**Symptoms and Signs of Heart Failure**

**Manifestations of heart failure differ depending on the extent to which the LV and RV are initially affected. Clinical severity varies significantly and is usually classified according to the New York Heart Association (NYHA) system (see table NYHA Classification of Heart Failure); the examples of ordinary activity may be modified for older, debilitated patients. Because HF has such a broad range of severity, some experts suggest subdividing NYHA class III into IIIA or IIIB. Class IIIB is typically reserved for those patients who recently had a heart failure exacerbation. The American College of Cardiology/American Heart Association has advocated a staging system for HF (A, B, C, or D) to highlight the need for HF prevention.**

* + **A: High risk of HF but no structural or functional cardiac abnormalities or symptoms**
  + **B: Structural or functional cardiac abnormalities but no symptoms of HF**
  + **C: Structural heart disease with symptoms of HF**
  + **D: Refractory HF requiring advanced therapies (eg, mechanical circulatory support, cardiac transplantation) or palliative care**

**Severe LV failure may cause pulmonary edema or cardiogenic shock.**

**TABLE**

**New York Heart Association (NYHA) Classification of Heart Failure**

**History**

**In LV failure, the most common symptoms are dyspnea and fatigue due to increased pulmonary venous pressures, and low cardiac output (CO, at rest or inability to augment CO during exertion). Dyspnea usually occurs during exertion and is relieved by rest. As HF worsens, dyspnea can occur during rest and at night, sometimes causing nocturnal cough. Dyspnea occurring immediately or soon after lying flat and relieved promptly by sitting up (orthopnea) is common as heart failure advances. In paroxysmal nocturnal dyspnea (PND), dyspnea awakens patients several hours after they lie down and is relieved only after they sit up for 15 to 20 minutes. In severe HF, periodic cycling of breathing (Cheyne-Stokes respiration—from a brief period of apnea, patients breathe progressively faster and deeper, then slower and shallower until they become apneic and repeat the cycle)—can occur during the day or night; the sudden hyperpneic phase may awaken the patient from sleep. Cheyne-Stokes breathing differs from PND in that the hyperpneic phase is short, lasting only 10 to 15 seconds, but the cycle recurs regularly, lasting 30 seconds to 2 minutes. PND is associated with pulmonary congestion, and Cheyne-Stokes respiration with low CO. Sleep-related breathing disorders, such as sleep apnea, are common in HF and may aggravate HF. Severely reduced cerebral blood flow and hypoxemia can cause chronic irritability and impair mental performance.**

**In RV failure, the most common symptoms are ankle swelling and fatigue. Sometimes patients feel a sensation of fullness in the abdomen or neck. Hepatic congestion can cause right upper quadrant abdominal discomfort, and stomach and intestinal congestion can cause early satiety, anorexia, and abdominal bloating.**

**Less specific heart failure symptoms include cool peripheries, postural light-headedness, nocturia, and decreased daytime micturition. Skeletal muscle wasting can occur in severe biventricular failure and may reflect some disuse but also increased catabolism associated with increased cytokine production. Significant weight loss (cardiac cachexia) is an ominous sign associated with high mortality.**

**In older people, presenting complaints may be atypical, such as confusion, delirium, falls, sudden functional decline, nocturnal urinary incontinence, or sleep disturbance. Coexisting cognitive impairment and depression may also influence assessment and therapeutic interventions and may be worsened by the HF.**

**Examination**

**General examination may detect signs of systemic or cardiac disorders that cause or aggravate heart failure (eg, anemia, hyperthyroidism, alcohol use disorder, hemochromatosis, atrial fibrillation with rapid rate, mitral regurgitation).**

**In LV failure, tachycardia and tachypnea may occur. Patients with severe LV failure may appear visibly dyspneic or cyanotic, hypotensive, and confused or agitated because of hypoxia and poor cerebral perfusion. Some of these less specific symptoms (eg, confusion) are more common in older patients.**

**Central cyanosis (affecting all of the body, including warm areas such as the tongue and mucous membranes) reflects severe hypoxemia. Peripheral cyanosis of the lips, fingers, and toes reflects low blood flow with increased oxygen extraction. If vigorous massage improves nail bed color, cyanosis may be peripheral; increasing local blood flow does not improve color if cyanosis is central.**

**Cardiac findings in HFrEF include**

* + **Diffuse, sustained, and laterally displaced apical impulse**
  + **Audible and occasionally palpable 3rd (S3) and 4th (S4) heart sounds**
  + **Accentuated pulmonic component (P2) of the 2nd heart sound (S2)**

**These abnormal heart sounds also can occur in HFpEF. A pansystolic murmur of mitral regurgitation at the apex may occur in either HFrEF or HFpEF.**

**Pulmonary findings include early inspiratory basilar crackles that do not clear with coughing and, if pleural effusion is present, dullness to percussion and diminished breath sounds at the lung base(s).**

**Signs of RV failure include**

* + **Nontender peripheral pitting edema (digital pressure leaves visible and palpable imprints, sometimes quite deep) in the feet and ankles**
  + **Enlarged and sometimes pulsatile liver palpable below the right costal margin**
  + **Abdominal swelling and ascites**
  + **Visible elevation of the jugular venous pressure, sometimes with large *a* or *v* waves that are visible even when the patient is seated or standing (see figure Normal jugular vein waves)**

**In severe cases of heart failure, peripheral edema can extend to the thighs or even the sacrum, scrotum, lower abdominal wall, and occasionally even higher. Severe edema in multiple areas is termed anasarca. Edema may be asymmetric if patients lie predominantly on one side.**

**Large V waves in the jugular veins are usually indicative of significant tricuspid regurgitation which is often present in RV failure. A paradoxical increase in the jugular venous pressure during inspiration (Kussmaul sign) is indicative of right-sided heart failure and can occur in RV failure, restrictive cardiomyopathy, constrictive pericarditis, and severe tricuspid regurgitation.**

**With hepatic congestion, the liver may be palpably enlarged or tender, and hepatojugular or abdominal-jugular reflux may be detected (see Approach to the Cardiac Patient). Precordial palpation may detect the left parasternal lift of RV enlargement, and auscultation may detect the murmur of tricuspid regurgitation or the RV S3 along the left sternal border; both findings are augmented upon inspiration.**

**Diagnosis of Heart Failure**

* + **Sometimes only clinical evaluation**
  + **Chest x-ray**
  + **Echocardiography, cardiac radionuclide scan, and/or MRI**
  + **BNP or N-terminal-pro-BNP (NT-pro-BNP) levels**
  + **ECG and other tests for etiology as needed**

**Clinical findings (eg, exertional dyspnea or fatigue, orthopnea, edema, tachycardia, pulmonary crackles, S3, jugular venous distention) suggest heart failure but are usually not apparent early. Some similar symptoms may result from COPD (chronic obstructive pulmonary disease) or recurrent pneumonia or may be erroneously attributed to obesity or old age. Suspicion for heart failure should be high in patients with a history of myocardial infarction, hypertension, or valvular disorders or murmurs and should be moderate in any patient who is older or has diabetes.**

**Chest x-ray, ECG, and an objective test of cardiac function, typically echocardiography, should be done (see figure Diagnosis of heart failure of acute onset). Blood tests, except for BNP levels, are not used for diagnosis but are useful for identifying cause and systemic effects (1, 2).**

**Diagnosis of heart failure of acute onset**

| Data from McDonagh TA, Metra M, Adamo M, et al: 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J* 42(36):3599-3726, 2021. doi: 10.1093/eurheartj/ehab368.  Diagnosis of heart failure of acute onset |
| --- |

**Chest x-ray**

**Chest x-ray findings suggesting heart failure include an enlarged cardiac silhouette, pleural effusion, fluid in the major fissure, and horizontal lines in the periphery of lower posterior lung fields (Kerley B lines). These findings reflect chronic elevation of left atrial pressure and chronic thickening of the intralobular septa due to edema. Upper lobe pulmonary venous congestion and interstitial or alveolar edema may also be present. Careful examination of the cardiac silhouette on a lateral projection can identify specific ventricular and atrial chamber enlargement. The x-ray may also suggest alternative diagnoses (eg, COPD, pneumonia, idiopathic pulmonary fibrosis, lung cancer).**

**Imaging in Patients With Heart Failure**

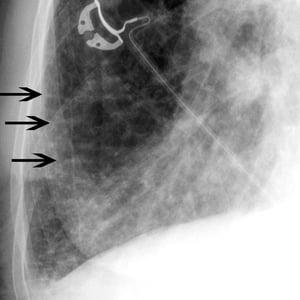
****

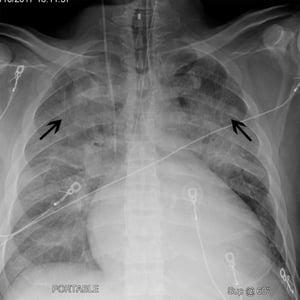
**Chest X-Ray of a Patient with Bilateral Pleural Effusions**

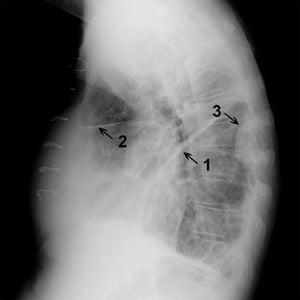
**This patient has bilateral pleural effusions (arrows). The normally sharp costophrenic angles are obscured by fluid in this patient.**

**... read more**

**© 2017 Elliot K. Fishman, MD.**

****

****

****

**ECG**

**ECG findings are not diagnostic, but an abnormal ECG, especially showing previous myocardial infarction, left ventricular hypertrophy, left bundle branch block, or tachyarrhythmia (eg, rapid atrial fibrillation), increases suspicion for HF and may help identify the cause. An entirely normal ECG is uncommon in chronic HF.**

**Imaging**

**Echocardiography can help evaluate chamber dimensions, valve function, LVEF, wall motion abnormalities, LV hypertrophy, diastolic function, pulmonary artery pressure, LV and RV filling pressures, RV function, and pericardial effusion. Intracardiac thrombi, tumors, and calcifications within the heart valves, mitral annulus, and aortic wall abnormalities can be detected. Localized or segmental wall motion abnormalities strongly suggest underlying coronary artery disease but can also be present with patchy myocarditis. Doppler or color Doppler echocardiography accurately detects valvular disorders and shunts. The combination of Doppler evaluation of mitral inflow with tissue Doppler imaging of the mitral annulus can help identify and quantify LV diastolic dysfunction and LV filling pressures. Measuring LVEF can distinguish between predominant HFpEF (EF ≥50%) and HFrEF (EF ≤ 40%). It is important to re-emphasize that heart failure can occur with a normal LVEF. Speckle-tracking echocardiography (which is useful in detecting subclinical systolic dysfunction and specific patterns of myocardial dysfunction) may become important but currently is routinely reported only in specialized centers.**

**Radionuclide imaging also can help assess systolic and diastolic function, previous MI (myocardial infarction), and inducible ischemia or myocardial hibernation. It is used most commonly to assess the presence and/or severity of ischemic heart disease and can also be used to quantify left ventricular ejection fraction.**

**Cardiac MRI provides accurate images of cardiac structures and is becoming more widely available. Cardiac MRI using late gadolinium enhancement imaging (LGE, also called fibrosis or scar imaging) is useful to evaluate the cause of myocardial disease and to detect focal and diffuse myocardial fibrosis. Cardiac amyloidosis, sarcoidosis, hemachromatosis, and myocarditis are causes of HF that can be detected with or suspected by cardiac MRI findings.**

**Blood tests**

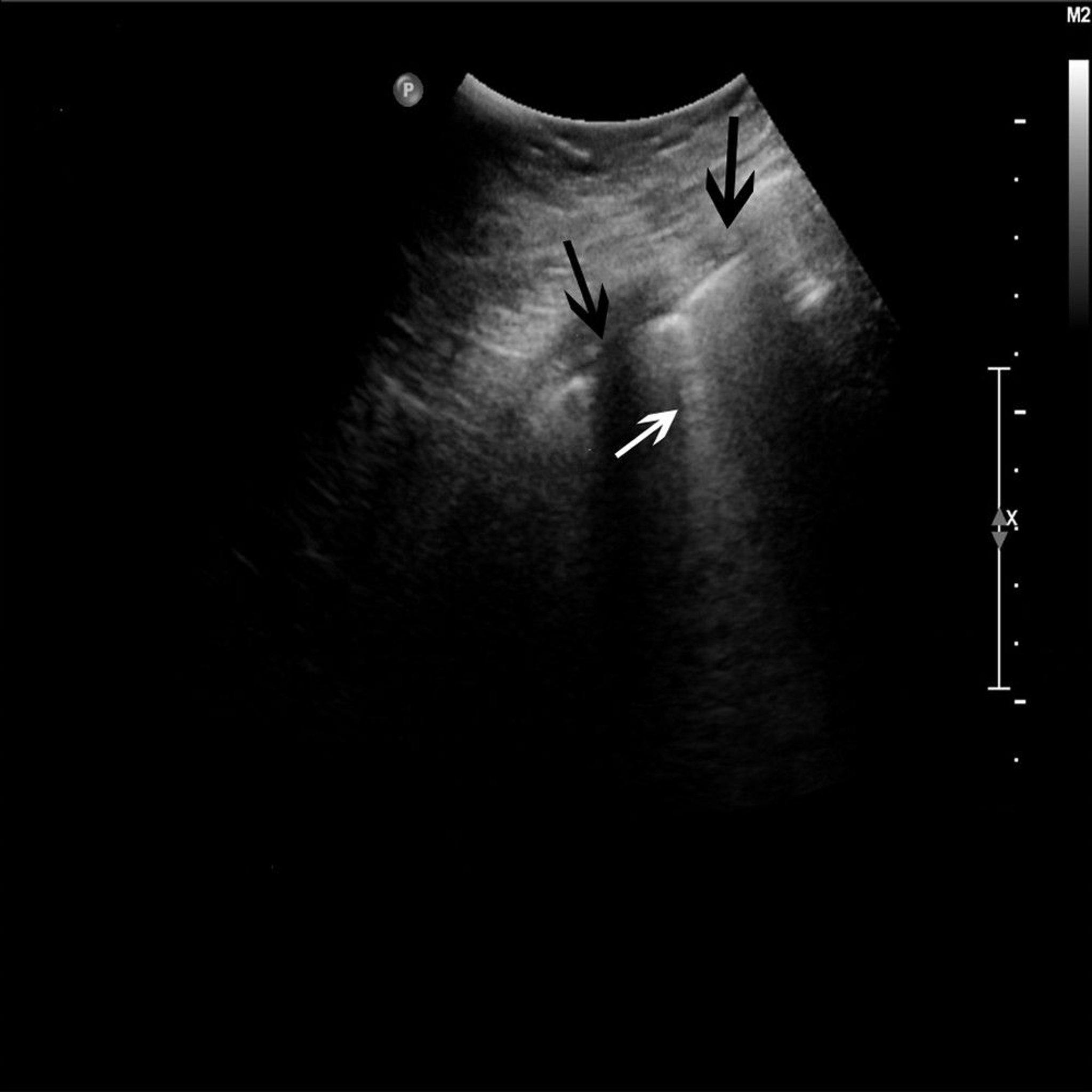
**Serum BNP levels are often high in heart failure; this finding may help when clinical findings are unclear or other diagnoses (eg, COPD) need to be excluded. It may be particularly useful for patients with a history of both pulmonary and cardiac disorders. NT-pro-BNP, an inactive moiety created when pro-BNP is cleaved, can be used similarly to BNP. However, *a normal BNP level does not exclude the diagnosis of heart failure,* particularly in patients with HFpEF and/or obesity. In HFpEF, BNP levels tend to be about 50% of those associated with HFrEF (at similar degree of symptoms), and up to 30% of patients with acute HFpEF have a BNP level below the commonly used threshold of 100 pg/mL (100 ng/L). Obesity, which is becoming an increasingly common comorbidity in HF, is associated with reduced BNP production and increased BNP clearance, resulting in lower levels.**

**Besides BNP, recommended blood tests include complete blood count, creatinine, BUN (blood urea nitrogen), electrolytes (including magnesium and calcium), glucose, albumin, ferritin, and liver tests. Thyroid function tests are recommended for patients with atrial fibrillation and for selected, especially older, patients.**

**Other tests**

**Thoracic ultrasonography is a noninvasive method of detecting pulmonary congestion in patients with heart failure. Sonographic "comet tail artifact" on thoracic ultrasonography corresponds to the x-ray finding of Kerley B lines.**

**Comet Tail Artifact**

****

**IMAGE**

**© 2017 ELLIOT K. FISHMAN, MD.**

**Coronary angiography or CT coronary angiography is indicated when coronary artery disease is suspected or the etiology of HF is uncertain.**

**Cardiac catheterization with intracardiac pressure measurements (invasive hemodynamics) may be helpful in the diagnosis of restrictive cardiomyopathies and constrictive pericarditis. Invasive hemodynamic measurements are also very helpful when the diagnosis of HF is equivocal, particularly in patients with HFpEF. In addition, perturbing the cardiovascular system (eg, exercise testing, volume challenge, drug challenges [eg, nitroglycerin, nitroprusside]) can be very helpful during invasive hemodynamic testing to help diagnose HF.**

**Endocardial biopsy is sometimes done when an infiltrative cardiomyopathy, or acute giant cell myocarditis is strongly suspected but cannot be confirmed with noninvasive imaging (eg, cardiac MRI).**

**Diagnosis references**

* 1. **1. McDonagh TA, Metra M, Adamo M, et al: 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J* 42(36):3599-3726, 2021. doi: 10.1093/eurheartj/ehab368**
  2. **2. Heidenreich PA, Bozkurt B, Aguilar D, et al: 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure: A report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*145:e876–e894, 2022, doi: 10.1161/CIR.0000000000001062**

**Prognosis for Heart Failure**

**Generally, patients with heart failure have a poor prognosis unless the cause is correctable. Overall combined 5 year survival is 35% for patients with HFpEF or HFrEF after an initial hospitalization for heart failure. In overt chronic HF, mortality depends on severity of symptoms and ventricular dysfunction and can range from 10 to 40%/year.**

**Specific factors that suggest a poor prognosis include hypotension, low ejection fraction, presence of coronary artery disease, troponin release, elevation of BUN, reduced GFR, hyponatremia, and poor functional capacity (eg, as tested by a 6-minute walk test).**

**BNP, NTproBNP, and risk scores such as the Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) Risk Score and the Seattle Heart Failure model, are helpful to predict prognosis in HF patients as an overall group, although there is significant variation in survival among individual patients.**

**HF usually involves gradual deterioration, interrupted by bouts of severe decompensation, and ultimately death, although the time course is being lengthened with modern therapies. However, death can also be sudden and unexpected, without prior worsening of symptoms.**

**End-of-life care**

**All patients and family members should be taught about disease progression and the risk of sudden cardiac death. For some patients, improving quality of life is as important as increasing quantity of life. Thus, it is important to determine patients’ wishes about resuscitation (eg, endotracheal intubation, CPR [cardiopulmonary respiration]) if their condition deteriorates, especially when HF is already severe.**

**All patients should be reassured that symptoms will be relieved, and they should be encouraged to seek medical attention early if their symptoms change significantly. Involvement of pharmacists, nurses, social workers, and clergy (when desired), who may be part of an interdisciplinary team or disease management program already in place, is particularly important in end-of-life care.**

**Treatment of Heart Failure**

* + **Diet and lifestyle changes**
  + **Treatment of cause**
  + **Drug therapy**
  + **Sometimes device therapy (eg, implantable cardioverter-defibrillator, cardiac resynchronization therapy, mechanical circulatory support)**
  + **Sometimes cardiac transplantation**
  + **Multidisciplinary care**

**Immediate inpatient treatment is required for patients with acute or worsening heart failure due to certain disorders (eg, acute myocardial infarction, atrial fibrillation with a very rapid ventricular rate, severe hypertension, acute valvular regurgitation), as well as for patients with pulmonary edema, severe symptoms, new-onset HF, or HF unresponsive to outpatient treatment. Patients with mild exacerbations of previously diagnosed HF can be treated at home.**

**The primary goal is to diagnose and to correct or treat the disorder that led to heart failure.**

**Short-term goals include relieving symptoms and improving hemodynamics; avoiding hypokalemia, renal dysfunction, and symptomatic hypotension; and correcting neurohumoral activation.**

**Long-term goals include correcting hypertension, preventing myocardial infarction and progression of atherosclerosis, improving cardiac function, reducing hospitalizations, and improving survival and quality of life.**

**Treatment involves dietary and lifestyle changes, drugs, devices, and sometimes percutaneous coronary interventions or surgery.**

**Treatment is tailored to the patient, considering causes, symptoms, and response to drugs, including adverse effects. There are currently several evidence-based therapies for chronic HFrEF . There are fewer evidence-based treatments for chronic HFpEF, HFmrEF, acute HF syndromes, and RV failure .**

**See Drugs for Heart Failure for detailed information on drug treatment and the specific drugs and classes.**

**Disease management**

**General measures, especially patient and caregiver education and diet and lifestyle modifications, are important for all patients with heart failure.**

* + **Education**
  + **Sodium restriction**
  + **Appropriate weight and fitness levels**
  + **Correction of underlying conditions**

**Patient and caregiver education are critical to long-term success. The patient and family should be involved in treatment choices. They should be taught the importance of drug adherence, warning signs of an exacerbation, and how to link cause with effect (eg, increased salt in the diet with weight gain or symptoms).**

**Many centers (eg, specialized outpatient clinics) have integrated health care practitioners from different disciplines (eg, HF nurses, pharmacists, social workers, rehabilitation specialists) into multidisciplinary teams or outpatient heart failure management programs. These approaches can improve outcomes and reduce hospitalizations and are most effective in the sickest patients.**

**Dietary sodium restriction helps limit fluid retention. All patients should eliminate salt in cooking and at the table and avoid salted foods; the most severely ill should limit sodium to < 2 g/day by consuming only low-sodium foods.**

**Monitoring daily morning weight helps detect sodium and water accumulation early. If weight increases > 2 kg over a few days, patients may be able to adjust their diuretic dose themselves, but if weight gain continues or symptoms occur, patients should seek medical attention.**

**Intensive case management, particularly by monitoring drug adherence and frequency of unscheduled visits to the physician or emergency department and hospitalizations, can identify when intervention is needed. Specialized HF nurses are valuable in education, follow-up, and dosage adjustment according to predefined protocols.**

**Patients with atherosclerosis or diabetes should strictly follow a diet appropriate for their disorder. Obesity may cause and always aggravates the symptoms of HF; patients should attain a body mass index (BMI) ≤ 30 kg/m2 (ideally 21 to 25 kg/m2).**

**Regular light activity (eg, walking), tailored to symptoms, is generally encouraged. Activity prevents skeletal muscle deconditioning, which worsens functional status; however, activity does not appear to improve survival or decrease hospitalizations. Rest is appropriate during acute exacerbations. Formal exercise cardiac rehabilitation is useful for chronic HFrEF and is likely helpful for patients with HFpEF.**

**Patients should have annual influenza vaccination because influenza can precipitate HF exacerbations, particularly in institutionalized or older patients. Patients should be vaccinated against SARS-CoV-2.**

**If causes such as hypertension, persistent tachyarrhythmia, severe anemia, hemochromatosis, uncontrolled diabetes, thyrotoxicosis, beriberi, alcohol use disorder, or toxoplasmosis are successfully treated, patients may dramatically improve. Significant myocardial ischemia should be treated aggressively; treatment may include revascularization by percutaneous coronary intervention or bypass surgery. Management of extensive ventricular infiltration (eg, in amyloidosis) has improved considerably. Newer treatments for amyloidosis have markedly improved prognosis for many of these patients.**

**Arrhythmias**

**Because arrhythmias can worsen heart failure, it is important to identify and treat the cause of any arrhythmia.**

* + **Electrolytes are normalized.**
  + **Atrial and ventricular rates are controlled.**
  + **Sometimes antiarrhythmic drugs are given.**

**Sinus tachycardia, a common compensatory change in heart failure, usually subsides when HF treatment is effective. If it does not, associated causes (eg, hyperthyroidism, pulmonary emboli, fever, anemia, pain) should be sought. If sinus tachycardia persists despite correction of causes, a beta-blocker, given in gradually increasing doses, may help selected patients. However, lowering heart rate with a beta-blocker can be detrimental to patients with advanced HFpEF (eg, restrictive cardiomyopathy), in whom stroke volume is fixed because of severe diastolic dysfunction. In these patients, CO is heart rate–dependent, and lowering heart rate can thus lower CO at rest and/or with exertion.**

**Atrial fibrillation with an uncontrolled ventricular rate must be treated; the target resting ventricular rate is typically < 80 beats/minute. Beta-blockers are the treatment of choice, although rate-limiting calcium channel blockers may be used cautiously if systolic function is preserved. Adding digoxin, low-dose amiodarone, or other rhythm and/or rate controlling drugs may help some patients. Routine conversion to and maintenance of sinus rhythm has not been shown to be superior to rate control alone in large clinical trials. However, it is best to make this determination on a case-by-case basis because some patients improve significantly with restoration of normal sinus rhythm. If rapid atrial fibrillation does not respond to drugs, permanent pacemaker insertion with complete or partial ablation of the atrioventricular node, or other atrial fibrillation ablation procedures, may be considered in selected patients to restore a sinus or regular rhythm.**

**Isolated ventricular premature beats, which are common in HF, do not require specific treatment, although rarely very frequent ventricular premature beats (> 15,000/day) have been shown to precipitate heart failure (that reverses with suppression). However, optimization of HF treatments and correction of electrolyte abnormalities (especially potassium and magnesium) reduce the risk of ventricular arrhythmias.**

**Sustained ventricular tachycardia that persists despite correction of cause (eg, low potassium or magnesium, ischemia) and optimal medical treatment of HF may require an antiarrhythmic drug. Amiodarone, beta-blockers, and dofetilide are the drugs of choice because other antiarrhythmics have adverse proarrhythmic effects when LV systolic dysfunction is present. Because amiodarone increases digoxin and warfarin levels, digoxin and/or warfarin doses should be decreased by half or stopped. Serum digoxin level and INR (international normalized ratio) level should be routinely monitored. However, drug toxicity can occur even at therapeutic levels. Because long-term use of amiodarone can cause adverse effects, a low dose (200 mg orally once a day) is used when possible; blood tests for liver function and thyroid-stimulating hormone are done every 6 months. If chest x-ray is abnormal or dyspnea worsens significantly, chest x-ray and pulmonary function tests are done yearly to check for pulmonary fibrosis. For sustained ventricular arrhythmias, amiodarone may be required; to reduce risk of sudden death, a loading dose of 400 to 800 mg orally twice a day is given for 1 to 3 weeks until rhythm control is adequate, then dose is decreased over 1 month to a maintenance dose of 200 mg orally once a day.**

**Device therapy**

**Use of an implantable cardioverter-defibrillator (ICD) or cardiac resynchronization therapy (CRT) is appropriate for some patients.**

**An ICD is recommended for patients with an otherwise good life expectancy if they have symptomatic sustained ventricular tachycardia or ventricular fibrillation or if they remain symptomatic and have an LVEF persistently < 35% while receiving guideline-directed medical therapy. The data for ICD use in HFrEF are stronger for ischemic cardiomyopathy than in nonischemic cardiomyopathy. A clinical trial that included HFrEF patients with nonischemic cardiomyopathy demonstrated no mortality benefit from prophylactic (primary prevention) ICD placement .**

**CRT is a mode of pacing that synchronizes contraction of the left ventricle by simultaneously pacing its opposing wall, thereby improving stroke volume. CRT may relieve symptoms and reduce heart failure hospitalizations for patients who have HF, LVEF <35%, and a widened QRS complex with a left bundle branch block pattern (> 0.15 second—the wider the QRS, the greater potential benefit). CRT devices are effective but expensive, and patients should be appropriately selected. Many CRT devices also incorporate an ICD in their mechanism.**

**An implantable device that remotely monitors invasive hemodynamics (eg, pulmonary artery pressure) may help guide heart failure management in highly selected patients. For example, drug (eg, diuretic) titration based on readings from one of these devices was associated with a marked reduction in HF hospitalization in one clinical trial that included patients with both HFrEF and HFpEF. The device uses the pulmonary artery diastolic pressure as a surrogate for pulmonary capillary wedge pressure (and hence left atrial pressure) in HF patients. However, it has been evaluated only in NYHA (New York Heart Association) class III patients who had recurrent HF exacerbations. Further evidence will help guide how this technology should be implemented.**

**Ultrafiltration (venovenous filtration) can be useful in selected hospitalized patients with severe cardiorenal syndrome and volume overload refractory to diuretics. However, ultrafiltration should not be used routinely because clinical trials do not show long-term clinical benefit.**

**An intra-aortic counterpulsation balloon pump (IABP) is helpful in selected patients with acute HF who have a good chance of recovery (eg, acute HF following myocardial infarction) or in those who need a bridge to a more permanent solution such as cardiac surgery (eg, to fix severe valvular disease or to revascularize multivessel coronary artery disease), an LV assist device, or heart transplantation. Other forms of temporary mechanical circulatory support for patients with acute HF and cardiogenic shock include surgically placed devices such as extracorporeal membrane oxygenation (ECMO, typically venoarterial cannulation) and centrifugal flow ventricular assist devices that can support either the LV, the RV, or both and can also be combined with an oxygenator to provide full cardiopulmonary support. Percutaneously placed devices such as intravascular microaxial ventricular assist devices are available for both LV and RV support. Selection of temporary mechanical circulatory support devices is based mainly on availability and local medical center experience.**

**Durable or ambulatory LV assist devices (LVADs) are longer-term implantable pumps that augment LV output. They are commonly used to maintain patients with severe HF who are awaiting transplantation and are also used as "destination therapy" (ie, as a long-term or permanent solution) in some patients who are not transplant candidates.**

**Surgery and percutaneous procedures**

**Surgery may be appropriate when certain underlying disorders are present. Surgery in patients with advanced HF should be done in a specialized center.**

**Surgical closure of congenital or acquired intracardiac shunts can be curative.**

**Coronary artery bypass grafting (CABG) for patients with LV systolic dysfunction secondary to coronary artery disease and evidence of myocardial viability may be beneficial; however, those patients with prior myocardial infarction and non-viable myocardium are less likely to benefit from CABG. Thus, the decision to revascularize a HF patient with multivessel coronary artery disease should be made on a case-by-case basis.**

**If HF is primarily due to a valvular disorder, valve repair or replacement should be considered. Patients with primary mitral regurgitation are more likely to benefit than patients with mitral regurgitation secondary to LV dilation, in whom poor myocardial function is likely to continue postoperatively. Surgery is preferably done before myocardial dilation and damage become irreversible. More recently, percutaneous mitral valve repair procedure (also called transcatheter end-to-end repair [TEER]), in which a clip is applied to approximate the anterior and posterior mitral leaflets, has been shown to reduce death and HF hospitalization in carefully selected patients with symptomatic HF despite optimal medical management and moderate to severe or severe mitral regurgitation with preserved LV size (end-systolic dimension ≤ 70 mm—5).**

**Heart transplantation is the treatment of choice for patients < 60 who have severe, refractory HF and no other life-threatening conditions and who are highly adherent to management recommendations. Some older patients (about 60 to 70 years) with otherwise good health are also typically considered if they meet other criteria for transplantation. Survival is 85 to 90% at 1 year, and annual mortality thereafter is about 4%/year; however, mortality rate while waiting for a donor is 12 to 15%. Human organ donation remains low.**

**Anemia and iron deficiency**

**Anemia is common among patients with chronic heart failure and is frequently multifactorial. Anemia is associated with worse symptoms and outcomes in HF and so reversible causes should be sought and treated. Iron deficiency is among the most common causes of anemia in HF, and iron replacement therapy should be considered once treatable causes such as blood loss (gastrointestinal or other) have been excluded. Oral iron replacement is often less effective due to poor absorption and other reasons, thus intravenous iron replacement is preferred.**

**Persistent heart failure**

**After treatment, symptoms often persist. Reasons include**

* + **Persistence of the underlying disorder (eg, hypertension, ischemia/infarction, valvular disease) despite treatment**
  + **Suboptimal treatment of heart failure**
  + **Drug nonadherence**
  + **Excess intake of dietary sodium or alcohol**
  + **Presence of an undiagnosed thyroid disorder, anemia, or supervening arrhythmia (eg, atrial fibrillation with rapid ventricular response, intermittent ventricular tachycardia)**

**Also, drugs used to treat other disorders may interfere with HF treatment. Nonsteroidal anti-inflammatory drugs (NSAIDs), thiazolidinediones (eg, pioglitazone) for diabetes, and short-acting dihydropyridine or nondihydropyridine calcium channel blockers can worsen heart failure and should be avoided unless no alternative exists; patients who must take such drugs should be followed closely.**

**Drugs for Heart Failure**

**VIEW PATIENT EDUCATION**

* **Classes of Drugs for Heart Failure**|
* **More Information**

**Heart failure (HF) is a syndrome of ventricular dysfunction (see Heart Failure).**

**Drug treatment of heart failure (HF) involves symptom relief with**

* + **Diuretics**
  + **Nitrates**
  + **Digoxin**

**Drug treatment for long-term management and improved survival is with**

**Angiotensin converting enzyme (ACE) inhibitors**

* + - **Beta-blockers**
    - **Aldosterone antagonists**
    - **Angiotensin II receptor blockers (ARBs)**

**Angiotensin receptor/neprilysin inhibitors (ARNIs)**

* + - **Sodium-glucose cotransporter-2 inhibitors (SGLT2i)**
    - **Sinus node inhibitors**

**All patients should be given clear and explicit information about their drugs, including**

* + - **The importance of timely prescription renewal**
    - **The importance of adherence to therapy**
    - **How to recognize adverse effects**
    - **When to contact their physician**

**Selection of drugs for heart failure**

**Choice of drug depends on the type of heart failure along with individual patient characteristics. The most common classification of heart failure currently in use stratifies patients into**

* + - **Heart failure with reduced ejection fraction ("systolic HF")**
    - **Heart failure with preserved ejection fraction ("diastolic HF")**
    - **Heart failure with mildly reduced ejection fraction**

**Heart failure with reduced ejection fraction (HFrEF)**

**In HFrEF standard of care includes the following four classes of therapies, considered to be 'foundational therapies' for HFrEF management:**

* + - **Beta-blocker**
    - **Renin-angiotensin-aldosterone system (RAAS) inhibitor (typically an ARNI, although an ACE inhibitor or ARB could also used if ARNI is not tolerated)**
    - **Aldosterone antagonist**
    - **SGLT2**

**These four drug classes have been studied and have shown benefit for long-term management of HFrEF. Therapy is typically titrated up to maximal tolerated doses. Patients are typically given a drug from each class. Because patients may already be taking one of these classes of drugs prior to developing heart failure, the order of therapy initiation and rate of up-titration are generally patient specific.**

**Addition of a sodium-glucose cotransporter-2 (SGLT2) inhibitor, either dapagliflozin or empagliflozin (1), has been shown to reduce morbidity and mortality when added to standard care in patients with elevated natriuretic peptide levels; benefit was similar in patients with and without diabetes.**

**Other therapies are used in patient-specific settings (eg, sinus node inhibitors for lowering heart rate if patients cannot tolerate beta blockers).**

**Heart failure with preserved ejection fraction (HFpEF)**

**In HFpEF fewer drugs have been adequately studied. However, ACE inhibitors, ARBs, or aldosterone antagonists (mineralocorticoid receptor antagonists) are often used to treat HFpEF and/or associated comorbidities (such as hypertension and renal dysfunction), although survival benefit has not been demonstrated in clinical trials and, therefore, are not considered a standard of care.**

**ARNIs may reduce hospitalizations for heart failure but do not improve other outcomes.**

**In a recent clinical trial, the addition of the SGLT2 inhibitor empagliflozin to usual therapy was shown to reduce mortality and hospitalizations for HFpEF (2).**

**Beta blockers should be used only when there is another existing indication (eg, control of heart rate during atrial fibrillation, angina, following myocardial infarction). In patients with severe HFpEF (in contrast to HFrEF), lowering the heart rate (eg, with a beta-blocker) can exacerbate symptoms because they have a relatively fixed stroke volume due to severe diastolic dysfunction. In these patients, cardiac output (CO) is heart rate dependent, and lowering heart rate can thus lower CO at rest and/or with exertion.**

**In patients with infiltrative, restrictive, orhypertrophic cardiomyopathy, digoxin is not effective and may be harmful. In addition, vasodilator therapy may also be poorly tolerated and has not shown benefit in these patients.**

**Heart failure with mildly reduced ejection fraction (HFmrEF)**

**In HFmrEF there may be a specific benefit from ARNIs, although this possibility requires confirmation.**

**Patients with HFmrEF also benefit from the addition of an SGLT2 inhibitor such as empagliflozin to standard care.**

**Drug selection references**

* + 1. **1. Packer M, Anker SD, Butler J, et al: Cardiovascular and renal outcomes with empagliflozin in heart failure. *N Engl J Med*383(15):1413-1424, 2020. doi: 10.1056/NEJMoa2022190. Epub 2020 Aug 28. PMID: 32865377.**
    2. **2. Anker SD, Butler J, Filippatos G, et al: Empagliflozin in heart failure with a preserved ejection fraction. *N Engl J Med*385(16):1451-1461, 2021. doi: 10.1056/NEJMoa2107038. Epub 2021 Aug 27. PMID: 34449189.**

**Classes of Drugs for Heart Failure**

**Aldosterone antagonists**

**Because aldosterone can be produced independently of the renin-angiotensin system, its adverse effects are not inhibited completely even by maximal use of ACE inhibitors and angiotensin II receptor blockers (ARBs). Thus, the aldosterone antagonists (also termed mineralocorticoid receptor antagonists) are often used, particularly for patients with moderate to severe symptomsor signs of heart failure.**

**Typical drugs include spironolactone 25 to 50 mg orally once a day and eplerenone 25 to 100 mg orally once a day (does not cause gynecomastia in males). Aldosterone antagonists can reduce mortality, including from sudden death, in patients with left ventricular ejection fraction (LVEF) < 30% and chronic HF, or acute HF complicating acute myocardial infarction.**

**Potassium supplements should be stopped. Serum potassium and creatinine should be checked every 1 to 2 weeks for the first 4 to 6 weeks and after dose changes. Dose is lowered if potassium is between 5.0 and 5.5 mEq/L ( 5.5 mmol/L) and stopped if potassium is > 5.5 mEq/L (5.5 mmol/L), if creatinine increases above 2.5 mg/dL (220 micromol/L), or if ECG changes of hyperkalemia are present. Aldosterone antagonists should not be used in patients receiving both an ACE inhibitor and an ARB because of the high risk of hyperkalemia and renal dysfunction.**

**In patients with HFrEF, an aldosterone antagonist plus either an ACE inhibitor or an ARB is preferred over the combination of an ACE inhibitor and ARB.**

**In patients with HFpEF, spironolactone reduces hospitalization for heart failure and likely reduces cardiovascular mortality (1). Thus, aldosterone antagonists should be used in patients with HFpEF, particularly if they are volume overloaded and/or have a history of HF hospitalization. Loop diuretics can be minimized if necessary to accommodate the use of aldosterone antagonists.**

**Angiotensin converting enzyme (ACE) inhibitors**

**All patients with HFrEF should be given oral ACE inhibitors unless contraindicated (eg, by plasma creatinine > 2.8 mg/dL [> 250 micromol/L], bilateral renal artery stenosis, renal artery stenosis in a solitary kidney, or previous angioedema due to ACE inhibitors).**

**ACE inhibitors reduce production of angiotensin II and breakdown of bradykinin, mediators that affect the sympathetic nervous system, endothelial function, vascular tone, and myocardial performance. Hemodynamic effects include**

* + 1. **Arterial and venous vasodilation**
    2. **Sustained decreases in LV filling pressure during rest and exercise**
    3. **Decreased systemic vascular resistance**
    4. **Favorable effects on ventricular remodeling**

**ACE inhibitors prolong survival and reduce HF hospitalizations. For patients with atherosclerosis and a vascular disorder, these drugs reduce the risk of myocardial infarction and stroke. For patients with diabetes, they delay onset of nephropathy. Thus, ACE inhibitors may be used in patients with diastolic dysfunction and any of these disorders.**

**The starting dose typically should be low (usually one fourth to one half of the target dose depending on blood pressure and renal function); the dose is gradually adjusted upward over 8 weeks as tolerated, then continued indefinitely. Usual target doses of representative drugs include enalapril 10 to 20 mg twice a day, lisinopril 20 to 30 mg once a day, and ramipril 5 mg twice a day; there are many others.**

**If the hypotensive effect (more marked in patients with hyponatremia or volume depletion) is troublesome, it can often be minimized by separating administration of other blood pressure–lowering drugs, reducing the dose of concomitant diuretics, using a longer acting ACE inhibitor (eg, perindopril), or giving the dose at bedtime. ACE inhibitors often cause mild to moderate reversible serum creatinine elevation due to vasodilation of the efferent glomerular arteriole. An initial 20 to 30% increase in creatinine is no reason to stop the drug but does require closer monitoring, slower increases in dose, reduction in diuretic dose, or avoidance of nonsteroidal anti-inflammatory drugs (NSAIDs). Because aldosterone’s effect is reduced, potassium retention (hyperkalemia) may result, especially in patients receiving potassium supplements. Cough occurs in 5 to 15% of patients, probably because bradykinin accumulates, but other causes of cough should also be considered. Occasionally, rash or dysgeusia occurs. Angioedema is rare but can be life threatening and is a contraindication to ACE inhibitors. Alternatively, ARBs can be used, although rarely cross-reactivity is reported. Both are contraindicated in pregnancy.**

**Serum electrolytes and renal function should be measured before an ACE inhibitor is started, at 1 month, and after each significant increase in dose or change in clinical condition. If dehydration or poor renal function due to acute illness develops, the ACE inhibitor dose may need to be reduced or the drug may be temporarily stopped.**

**In HFpEF, a randomized controlled trial of the ACE inhibitor perindopril demonstrated improved exercise capacity. It did not improve survival, although there was a high rate of crossover from placebo to ACE inhibitor in this trial (2). Given the very high prevalence of hypertension in HFpEF, it is reasonable to use an ACE inhibitor to control hypertension in these patients as these drugs may have secondary beneficial effects on exercise capacity in these patients.**

**Angiotensin II receptor blockers (ARBs)**

**These drugs are not demonstrably superior to ACE inhibitors but are less likely to cause cough and angioedema; they may be used when these adverse effects prohibit ACE inhibitor use.**

**In chronic HFrEF, ACE inhibitors and ARBs are likely equally effective. Usual oral target doses are valsartan 160 mg twice a day, candesartan 32 mg once a day, and losartan 50 to 100 mg once a day. Introduction, upward dose adjustment, and monitoring of ARBs and ACE inhibitors are similar. Like ACE inhibitors, ARBs can cause reversible renal dysfunction, and the dose may need to be reduced or stopped temporarily during an acute dehydrating illness.**

**Adding an ARB to a regimen of an ACE inhibitor, beta-blocker, and aldosterone antagonist is unlikely to be helpful and should be avoided given the risk of hyperkalemia. If a patient who is taking an ACE inhibitor or ARB is still symptomatic, an aldosterone antagonist should be started and/or an angiotensin receptor/neprilysin inhibitor (ARNI) should be used.**

**In HFpEF, a large randomized controlled trial of candesartan (3) demonstrated reduced number of hospitalizations for recurrent HF; however, hospitalization was a secondary endpoint. In another trial , irbesartan was not associated with any improvement in outcomes in HFpEF. Therefore, ARBs should be used in HFpEF only if they are already being used to treat hypertension, diabetic kidney disease, or microalbuminuria.**

**ARBs are contraindicated in pregnancy.**

**Angiotensin receptor/neprilysin inhibitors (ARNIs)**

**ARNIs are a new combination drug for the treatment of heart failure. They include an ARB and a newer class of drug, neprilysin inhibitors (eg, sacubitril). Neprilysin is an enzyme involved in the breakdown of vasoactive substances such as brain (B-type) natriuretic peptide (BNP) and other peptides. By inhibiting the breakdown of BNP and other beneficial vasoactive peptides, these drugs lower blood pressure, decrease afterload, and enhance natriuresis. Because neprilysin inhibitors increase BNP levels, NTproBNP levels (which are not increased by the drug) should be used instead to help diagnose and manage HF.**

**In HFrEF, a large randomized, controlled trial (5) compared sacubitril/valsartan to enalapril in patients with NYHA (New York Heart Association) class II through IV heart failure (see table NYHA Classification of Heart Failure). Sacubitril/valsartan reduced the primary endpoints of combined cardiovascular mortality or hospitalizations for HF; the number needed to treat was 21. Sacubitril/valsartan also reduced all-cause mortality. Thus, the ARNI sacubitril/valsartan should be considered in all patients with stable HFrEF, particularly those with NYHA class II or III symptoms on optimal guideline-directed medical therapy and who have elevated natriuretic peptide levels before starting treatment. Evidence supports early transition of patients from ACE/ARB to ARNI, even in the hospital setting where patients will experience less pulmonary congestion and may have fewer early readmissions.**

**There are 3 strengths of sacubitril/valsartan: 24/26 mg, 49/51 mg, and 97/103 mg, all are taken orally twice a day. The starting dose is 49/51 mg orally twice a day for patients previously taking an ACE inhibitor or ARB, and 24/26 mg for patients previously taking a low dose of an ACE inhibitor or ARB (eg, ≤ 10 mg enalapril daily) or in those patients who are ACE inhibitor/ARB naive or who have low/borderline blood pressure. ACE inhibitors must be discontinued 36 hours before initiation of sacubitril/valsartan. Patients previously taking an ARB can simply switch to sacubitril/valsartan without a washout period.**

**Complications associated with use of ARNI include hypotension, hyperkalemia, renal insufficiency, and angioedema. Sacubitril is coupled with valsartan (an ARB) because of the increased risk of angioedema with the use of sacubitril alone or in combination with an ACE inhibitor. For this reason, combined ACE/ARNI therapy is absolutely contraindicated.**

**In HFpEF, a phase 2 trial showed that the ARNI sacubitril/valsartan reduced NTproBNP levels at 12 weeks and left atrial volume at 36 weeks. The PARAGON HF study in a stable population of patients with HFpEF showed a non-significant reduction in death and hospitalization (6, 7). However, there may have been lower hospitalization rates—further study is needed.**

**Beta-blockers**

**In patients with HFrEF, beta-blockers, unless otherwise contraindicated (by asthma, 2nd- or 3rd-degree atrioventricular block, or previous significant intolerance), are critical for the treatment, and an important addition to ACE inhibitors in these patients. In HFrEF, beta-blockers are best started when the patient has no evidence of pulmonary congestion. Specific beta-blockers such as carvedilol and metoprolol succinate (ie, long-acting metoprolol) improve left ventricular ejection fraction, survival, and other major cardiovascular outcomes in patients with chronic HFrEF, including those with severe symptoms.**

**In patients with HFpEF, beta-blockers have not shown clear benefits in clinical trials. However, data from large registries have suggested that beta-blocker use is associated with improved outcomes in HFpEF despite the relatively high prevalence of chronotropic incompetence (ie, the inability to raise heart rate in response to increased exertional demand) in HFpEF. All major guidelines for heart failure recommend beta-blockade as first-line therapy for conditions where ventricular rate control is indicated (ie, control of ventricular rate with atrial fibrillation).**

**The starting dose should be low (one fourth of the target daily dose), then the dose is gradually increased over 8 weeks as tolerated. The acute negative inotropic effects of beta-blockade may initially cause cardiac depression and fluid retention. In such cases, a temporary increase in diuretic dose and slower upward titration of the beta-blocker dose is warranted. Tolerance may improve over time, and efforts should be made to reach target doses. Usual oral target doses are carvedilol 25 mg twice a day (50 mg twice a day for patients ≥ 85 kg), bisoprolol 10 mg once a day, and metoprolol 50 to 75 mg twice a day (tartrate) or 200 mg once a day (succinate extended-release). Carvedilol, a 3rd-generation nonselective beta-blocker, is also a vasodilator with alpha-blocking and antioxidant effects; it is the preferred and most widely studied beta-blocker but is more expensive in many countries. Some beta-blockers (eg, bucindolol, xamoterol) do not appear beneficial and may be harmful.**

**During a severe, acute decompensation, beta-blockers should not be started until patients are stabilized and have little evidence of fluid retention. For HFrEF patients with acute HF exacerbation already taking a beta-blocker, the dose should not be decreased or stopped unless absolutely necessary. Often the beta-blocker dose can be continued in patients with an acute HF exacerbation if the diuretic dose is temporarily increased.**

**In HFrEF, after initial treatment, heart rate and myocardial oxygen consumption decrease, and stroke volume and filling pressure are unchanged. With the slower heart rate, diastolic function improves. Ventricular filling returns to a more normal pattern (increasing in early diastole), which appears less restrictive. Improved myocardial function is measurable in some patients after 6 to 12 months but may take longer; ejection fraction (EF) and cardiac output (CO) increase, and LV filling pressure decreases. Exercise capacity improves.**

**Digoxin**

**Digoxin inhibits the sodium-potassium pump (Na+, K+-ATPase). As a result, it causes weak positive inotropy, reduces sympathetic activity, blocks the atrioventricular node (slowing the ventricular rate in atrial fibrillation or prolonging the PR interval in sinus rhythm), reduces vasoconstriction, and improves renal blood flow. Digoxin is excreted by the kidneys; elimination half-life is 36 to 40 hours in patients with normal renal function.**

**Digoxin has no proven survival benefit but, when used with diuretics and an ACE inhibitor, may help control symptoms and reduce the likelihood of hospitalization in patients with HFrEF. However, because of the availability of a large number of evidence-based treatments for HFrEF, digoxin use has dropped significantly and is reserved for patients with significant symptoms despite optimal treatment with other mortality lowering medications. Digoxin should not be used in HFpEF unless it is being used to control heart rate in concomitant atrial fibrillation or to augment RV function in patients with RV failure. Digoxin is most effective in patients with large LV end-diastolic volumes and a 3rd heart sound (S3). Acute withdrawal of digoxin may increase the hospitalization rate and worsen symptoms.**

**In patients with normal renal function, digoxin, 0.125 to 0.25 mg orally once a day depending on age, sex, and body size, achieves full digitalization in about 1 week (5 half-lives). More rapid digitalization can be achieved with digoxin 0.5 mg IV over 15 minutes followed by 0.25 mg IV at 8 and 16 hours or with 0.5 mg orally followed by 0.25 mg orally at 8, 16, and 24 hours. Prescription patterns vary widely by physician and by country, but in general, doses are lower than those used in the past, and a trough (8- to 12-hours post-dose) digoxin level of 0.8 to 1.2 ng/mL (1 to 1.5 nmol/L) is preferable. In addition, unlike in the treatment of atrial fibrillation, there is typically little reason to rapidly digitalize (ie, digoxin load) patients with HF. Thus, simply starting digoxin at 0.125 mg orally once a day (in patients with normal renal function) or digoxin 0.125 mg orally every Monday, Wednesday, and Friday (in patients with abnormal renal function) is sufficient in patients with heart failure.**

**Digoxin toxicity is a concern, especially in patients with renal dysfunction and perhaps in women. These patients may need a lower oral dose, as may older patients, patients with a low lean body mass, and patients also taking amiodarone. Digoxin has a narrow therapeutic window. The most important toxic effects are life-threatening arrhythmias (eg, ventricular fibrillation, ventricular tachycardia, complete atrioventricular block). Bidirectional ventricular tachycardia, nonparoxysmal junctional tachycardia in the presence of atrial fibrillation, and hyperkalemia are serious signs of digitalis toxicity. Nausea, vomiting, anorexia, diarrhea, confusion, amblyopia, and, rarely, xerophthalmia may occur. If hypokalemia or hypomagnesemia (often due to diuretic use) is present, lower doses and serum levels can still cause toxicity. Electrolyte levels should be monitored in patients taking diuretics and digoxin, so that abnormalities can be prevented if possible; potassium-sparing diuretics may be helpful.**

**When digoxin toxicity occurs, the drug should be stopped; electrolyte abnormalities should be corrected (IV if abnormalities are severe and toxicity is acute). Patients with severe toxicity are admitted to a monitored unit, and digoxin immune Fab (ovine antidigoxin antibody fragments) is given if arrhythmias are present or if significant overingestion is accompanied by a serum potassium of > 5 mEq/L (> 5 mmol/L). Digoxin immune Fab is also useful for glycoside toxicity due to plant ingestion. Dose is based on the steady-state serum digoxin level or total amount ingested. Ventricular arrhythmias are treated with lidocaine or phenytoin. Atrioventricular block with a slow ventricular rate may require a temporary transvenous pacemaker. Isoproterenol is contraindicated because it increases risk of ventricular arrhythmia.**

**Diuretics**

**Diuretics are given to all patients with HF (regardless of underlying ejection fraction) who have current or previous volume overload; dose is adjusted to the lowest dose that stabilizes weight and relieves symptoms.**

**Loop diuretics should be used initially for control of volume overload, but their dose should be reduced when possible in favor of aldosterone antagonists.**

**Commonly used loop diuretics include furosemide, bumetanide, and torsemide. The starting dose of these drugs depends on whether the patient has previously received loop diuretics. Common starting doses are: furosemide 20 to 40 mg orally once a day or twice a day, bumetanide 0.5 to 1.0 mg orally once a day, and torsemide 10 to 20 mg orally once a day. If needed, loop diuretics can be titrated up to doses of furosemide 120 mg orally twice a day, bumetanide 2 mg orally twice a day, and torsemide 40 mg orally twice a day based on response and renal function. Bumetanide and torsemide have better bioavailability than furosemide. If patients are switched between different loop diuretics, they should be placed on equivalent doses. Furosemide 40 mg is equivalent to bumetanide 1 mg, and both are equivalent to torsemide 20 mg.**

**In refractory cases, IV loop diuretics or metolazone 2.5 to 10 mg orally can be used for an additive effect. IV infusion of furosemide (5 to 10 mg/hour) or other loop diuretics may be helpful in selected patients with severe edema. A bolus dose of loop diuretic should be given before starting an IV infusion and before each increase in infusion rate.**

**Loop diuretics (particularly when used with metolazone) may cause hypovolemia with hypotension, hyponatremia, hypomagnesemia, and severe hypokalemia. The dose of diuretic required acutely can usually be gradually reduced; the target is the lowest dose that maintains stable weight and controls symptoms. When HF improves, the diuretic may be stopped if other drugs improve heart function and relieve HF symptoms. Using larger than required doses of diuretics lowers CO, impairs renal function, causes hypokalemia, and increases mortality. Serum electrolytes and renal function are monitored, initially daily (when diuretics are given IV) and subsequently as needed, particularly after a dose increase.**

**An aldosterone antagonist, either spironolactone or eplerenone, should be added early to offset the potassium-losing effects of higher-dose loop diuretics. Hyperkalemia may result, especially when ACE inhibitors or ARBs are also taken, so electrolytes must still be monitored, especially during a dehydrating illness that could cause renal dysfunction. Aldosterone antagonists may have particular benefit in chronic right ventricular failure, in which hepatic congestion results in elevated aldosterone levels as aldosterone metabolism is reduced. To reduce the risk of hyperkalemia, aldosterone antagonists should generally be given only to patients whose potassium level is < 5.0 mEq/L (< 5 mmol/L), serum creatinine is < 2.5 mg/dL (< 221 micromol/L), and GFR is > 30 mL/min/1.73 m2. Furthermore, it should be noted that the equivalent dose of eplerenone is twice that of spironolactone (ie, spironolactone 25 mg = eplerenone 50 mg).**

**Thiazide diuretics are not normally used alone unless being given as treatment of hypertension; however, a thiazide diuretic may be added to a loop diuretic for additional diuresis and to reduce the loop diuretic dose. Hydrochlorothiazide, metolazone, and chlorthalidone can be used in this manner.**

**Reliable patients are taught to take additional diuretic doses as needed when weight or peripheral edema increases. They should seek medical attention promptly if weight gain persists.**

**Vasopressin (antidiuretic hormone) receptor antagonists are not frequently used though they may be helpful in cases of severe refractory hyponatremia in patients with HF.**

**Sinus node inhibitors**

**There is an inward sodium/potassium current that travels through a certain gated channel (funny or "f" channel) in sinus node (cardiac pacemaker) cells located in the posterior right atrium. This current is sometimes referred to as the inward funny current (If). Inhibition of this current prolongs the time it takes to achieve critical spontaneous depolarization of pacemaker cells, and thus lowers the heart rate.**

**Ivabradine is an If channel blocker that acts at the sinus node to slow the heart rate. Since the receptors are present only in cardiac pacemaker cells, these drugs have no other cardiac effects (ie they do not directly affect contractility), and are not useful for treatment in patients who are not in sinus rhythm. Ivabradine is currently recommended for use in HFrEF patients who have symptomatic HF, normal sinus rhythm, and heart rate > 70 beats/minute despite guideline-directed medical therapy (which should include beta-blockers). Typically, patients who may benefit from ivabradine are those with HFrEF who have NYHA (New York Heart Association) class II or class III symptoms (see table NYHA Classification of Heart Failure) and heart rate > 70 beats/minute who are at target beta-blocker dose or cannot tolerate a further increase in beta-blocker dose (8).**

**Initial dose of ivabradine is 2.5 to 5 mg orally twice a day, titrated at 2-week intervals to a heart rate of 50 to 60 beats/minute; maximum dose is 7.5 mg twice a day.**

**Ivabradine is currently the only drug in this class.**

**Sodium-glucose cotransporter-2 inhibitors (SGLT2i)**

**SGLT2 inhibitors are used in treatment of diabetes to block glucose reabsorption, thus causing glycosuria and lowering plasma glucose. They may also have effects on the myocardium and vasculature. These drugs had previously been shown to prevent the onset of heart failure in patients with type 2 diabetes. One member of this class, dapagliflozin, was shown to improve symptoms and quality of life and decrease hospitalization and mortality in patients with HFrEF when added to standard care in patients with elevated natriuretic peptide levels; benefit was similar in patients with and without diabetes . In a recent clinical trial, the addition of the SGLT2 inhibitor empagliflozin to usual therapy was shown to reduce hospitalizations and death for patients with HFpEF, with or without diabetes (10).**

**Dapagliflozin and empagliflozin may be given 10 mg orally once a day. With treatment, there is a mild (10 to 15%) reduction in estimated glomerular filtration rate (eGFR) which does not progress, glucosuria, and a small reduction in body weight. Risks include genital fungal infection, and in patients with diabetes, a very small risk of hypoglycemia and diabetic ketoacidosis. These drugs are generally not indicated in patients with type I diabetes, low blood pressure, low eGFR (< 30 mL/min/1.73 m2), or rapidly worsening renal function.**

**Other SGLT2 inhibitors (eg, canagliflozin, ertugliflozin) have not been studied directly in HF, but secondary analysis of studies in diabetes suggest they may also be beneficial.**

**Vasodilators**

**Hydralazine plus isosorbide dinitrate may help patients truly intolerant of ACE inhibitors or ARBs (usually because of significant renal dysfunction), although limited studies show long-term benefit of this combination. However, in patients of African ancestry this combination, when added to standard therapy, has been shown to reduce mortality and hospitalization, and improve quality of life. As vasodilators, these drugs improve hemodynamics, reduce valvular regurgitation, and increase exercise capacity without causing significant renal impairment.**

**When used instead of ACE/ARB therapy, hydralazine is started at 25 mg orally 4 times a day and increased every 3 to 5 days to a target total dose of 300 mg/day, although many patients cannot tolerate > 200 mg/day because of hypotension. Isosorbide dinitrate is started at 20 mg orally 3 times a day (with a 12-hour nitrate-free interval) and increased to a target of 40 to 50 mg 3 times a day. Whether lower doses (frequently used in clinical practice) provide long-term benefit is unknown. In general, vasodilators have been replaced by ACE inhibitors, which are easier to use, are usually better tolerated, and have greater proven benefit.**

**When added to ACE/ARB therapy, hydralazine-nitrate therapy may benefit patients of African ancestry with HFrEF. In this case, the starting dose is hydralazine 37.5 mg and isosorbide dinitrate 20 mg orally three times a day, with the maximum dose 75 mg and 40 mg three times a day. These doses are also available as a fixed-dose combination. The decision to add or substitute hydralazine-nitrate therapy to an ACE/ARB in patients of African ancestry with HF is patient specific and frequently determined by drug tolerance and symptom burden. In general, RAAS inhibitor therapy (ACE, ARB, or ARNI) should be used in this population, if tolerated.**

**Nitrates alone can relieve HF symptoms in patients with HFrEF; patients can be taught to use sublingual nitroglycerin spray as needed for acute dyspnea and a transdermal patch for nocturnal or exertional dyspnea. In HFrEF, nitrates are safe, effective, and well tolerated and are particularly helpful in patients with HF and angina. Adverse effects include hypotension and headache. Isosorbide mononitrate has been tested in HFpEF (11), where it was shown to be associated with increased adverse effects (eg, headache) and reduced physical activity. Thus, routine use of long-acting nitrates should be avoided in HFpEF.**

**Other vasodilators such as calcium channel blockers are not used to treat LV systolic dysfunction. Short-acting dihydropyridines (eg, nifedipine) and nondihydropyridines (eg, diltiazem, verapamil) may be deleterious. However, amlodipine and felodipine are better tolerated and may be useful for patients with HF and associated angina or hypertension. Both drugs may cause peripheral edema; rarely, amlodipine causes pulmonary edema. Felodipine should not be taken with grapefruit juice, which significantly increases plasma levels and adverse effects by inhibiting cytochrome P-450 metabolism. In patients with HFpEF, dihydropyridine calcium channel blockers such as amlodipine may be used as needed to treat hypertension or ischemia; nondihydropyridines such as diltiazem or verapamil may be used to control ventricular rate in atrial fibrillation. Verapamil is often used in hypertrophic cardiomyopathy.**

**Other drugs**

**Various positive inotropic drugs have been evaluated in heart failure but, except for digoxin, they increase mortality risk. These drugs can be grouped as adrenergic mode of action (norepinephrine, epinephrine, dobutamine, dopamine) or nonadrenergic (enoximone, milrinone, levosimendan [calcium sensitizers]). Regular outpatient IV infusions of inotropes (eg, dobutamine) were previously tried but found to increase mortality and are not recommended. However, outpatient continuous infusions of inotropes such as dobutamine or milrinone can be used for palliative purposes in patients with severe HFrEF.**

**Vericiguat is an oral soluble guanylate cyclase stimulator which enhances the cyclic guanosine monophosphate (GMP) pathway and sensitizes soluble guanylate cyclase to endogenous nitric oxide, resulting in pulmonary vasodilation. A clinical trial in symptomatic chronic HFrEF patients with evidence of worsening HF demonstrated reduced cardiovascular mortality or HF hospitalizations for patients randomized to receive vericiguat (12). Vericiguat may therefore be an option to improve outcomes for HFrEF patients with worsening HF symptoms.**

**SOURCE**

Fine, N.M. (2022) *Heart Failure (HF)*. Available at: https://www.msdmanuals.com/professional/cardiovascular-disorders/heart-failure/heart-failure-hf#Treatment\_v936260

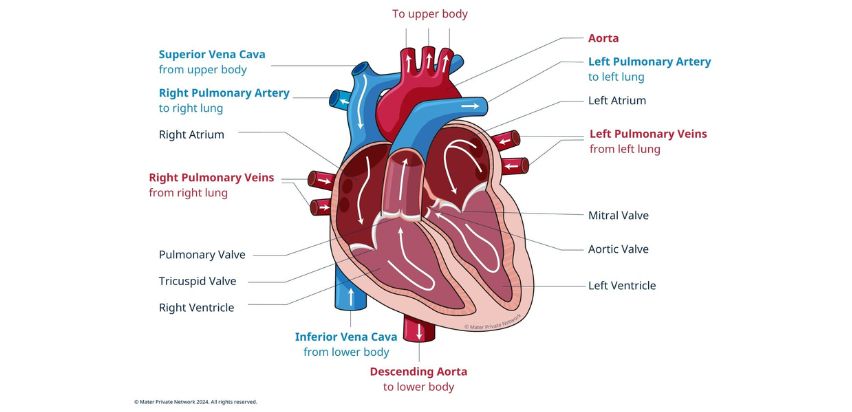
# Heart arrhythmias

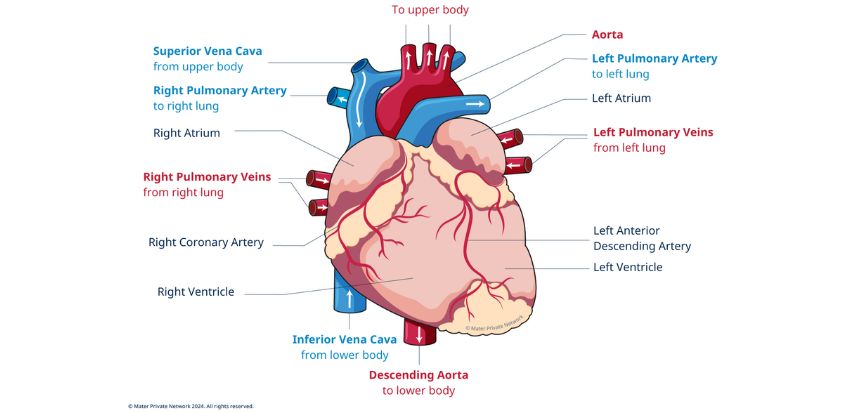
Heart arrhythmias, also known as cardiac rhythm disorders, involve abnormalities in the heart's electrical system, leading to irregular, too fast (tachycardia), or too slow (bradycardia) heartbeats. These disorders can impair blood flow, increasing the risk of complications such as stroke, heart failure, or sudden cardiac arrest. Below is a structured, evidence-based overview of arrhythmias, incorporating recent guidelines and epidemiological data. Arrhythmias are due to problems with your heart’s electrical pathways, affecting its rate or rhythm. Heart conditions, electrolyte imbalances, and certain medications are among the causes that can lead to these problems.

Arrhythmia occurs when your heart beats irregularly. It can beat too fast, too slow, or skip beats.

Arrhythmia can affect almost anyone but may more likely develop as you age. Researchers estimate that between 1.5% and 5%.

People in the United States have this condition. Atrial fibrillation, caused by your atria (two upper heart chambers) contracting irregularly, is the most common arrhythmia type.





### Sources:

* https://www.materprivate.ie/health-information/medical-conditions/article/heart-veins/arrhythmia
* https://www.healthline.com/health/arrhythmia/arrhythmia-causes
* https://www.heart.org/en/health-topics/arrhythmia/symptoms-diagnosis--monitoring-of-arrhythmia

## Definition and Types

Arrhythmias are classified based on origin and heart rate:

* Tachyarrhythmias: Heart rate >100 bpm.
  + *Supraventricular*: Atrial fibrillation (AFib), atrial flutter, paroxysmal supraventricular tachycardia (PSVT).
  + *Ventricular*: Ventricular tachycardia (VT), ventricular fibrillation (VFib).
* Bradyarrhythmias: Heart rate <60 bpm.
  + *Sinus node dysfunction*: sick sinus syndrome.
  + *Conduction blocks*: Atrioventricular (AV) block.

# Types of arrhythmia

Doctors classify arrhythmias into several general types:

* [bradycardia](https://www.healthline.com/health/slow-heart-rate), or a slow heart rate, typically fewer than 60 beats per minute
* tachycardia, a fast heart rate, typically more than 100 beats per minute
* supraventricular arrhythmias, which occur in your atria, for example:
  + [atrial fibrillation](https://www.healthline.com/health/atrial-fibrillation/types-of-atrial-fibrillation), or your upper heart chambers contracting irregularly
  + [atrial flutter](https://www.healthline.com/health/heart-disease/atrial-flutter), or your upper heart chambers contracting fast
* ventricular arrhythmias, which occur in your ventricles (lower heart chambers):
  + [ventricular tachycardia](https://www.healthline.com/health/ventricular-tachycardia), or your ventricles contracting fast
  + [ventricular fibrillation](https://www.healthline.com/health/ventricular-fibrillation), an emergency condition that makes your heart unable to pump blood due to irregular ventricle contractions
* premature atrial or [ventricular contractions](https://www.healthline.com/health/arrhythmia/when-to-worry-about-pvc), which happen when your atria or ventricles have early contractions.

Arrhythmia may be classified by rate (tachycardia, bradycardia), mechanism (automaticity, re-entry, triggered), or duration (isolated premature beats; couplets; runs, that is, 3 or more beats; non-sustained = less than 30 seconds or sustained = over 30 seconds).

Arrhythmias are also classified by site of origin:

**Atrial arrhythmia**

* Sinus bradycardia
* Sinus arrhythmia
* Sinus tachycardia
* Premature atrial contractions (PACs)
* Wandering atrial pacemaker
* Atrial tachycardia
* Multifocal atrial tachycardia
* Supraventricular tachycardia (SVT)
* Atrial flutter
* Atrial fibrillation (Afib)
* AV nodal reentrant tachycardia

**Junctional arrhythmia**

* AV nodal reentrant tachycardia
* Junctional rhythm
* Junctional tachycardia
* Premature junctional contraction

**Ventricular arrhythmia**

* Premature ventricular contractions (PVCs), sometimes called ventricular extra beats (VEBs)
  + Premature ventricular beats occurring after every normal beat are termed ventricular bigeminy
  + PVCs that occur at intervals of 2 normal beats to 1 PVC, or 1 normal beat to 2 PVCs, are termed "PVCs in trigeminy."
  + Groups of three premature ventricular beats are called triplets and are considered a brief run of nonsustained ventricular tachycardia (NSVT); if the grouping lasts for more than 30 seconds, it is considered sustained ventricular tachycardia (VT).
* Accelerated idioventricular rhythm
* Monomorphic ventricular tachycardia
* Polymorphic ventricular tachycardia
* Ventricular fibrillation
* Torsades de pointes
* Arrhythmogenic right ventricular dysplasia
* Re-entry ventricular arrhythmia

**Heart blocks**

These are also known as AV blocks because the vast majority of them arise from pathology at the atrioventricular node. They are the most common causes of bradycardia:

* First-degree heart block, which manifests as PR prolongation
* Second-degree heart block
  + Type 1 second-degree heart block, also known as Mobitz I or Wenckebach
  + Type 2 second-degree heart block, also known as Mobitz II
* Third-degree heart block, also known as complete heart block

First-, second-, and third-degree blocks also can occur at the level of the sinoatrial junction. This is referred to as a sinoatrial block, typically manifesting with various degrees and patterns of sinus bradycardia.

**Sudden arrhythmic death syndrome**

Sudden arrhythmic death syndrome (SADS), is a term used as part of *sudden unexpected death syndrome* to describe sudden death because of cardiac arrest occasioned by an arrhythmia in the presence or absence of any structural heart disease on autopsy. The most common cause of sudden death in the US is coronary artery disease, specifically because of poor oxygenation of the heart muscle, that is, myocardial ischemia or a heart attack. Approximately 180,000 to 250,000 people die suddenly of this cause every year in the US. SADS may occur from other causes. There are many inherited conditions and heart diseases that can affect young people, which can subsequently cause sudden death without advance symptoms.

Causes of SADS in young people include viral myocarditis, long QT syndrome, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, hypertrophic cardiomyopathy, and arrhythmogenic right ventricular dysplasia.

**Fetal arrhythmia**

Arrhythmias may also occur in the fetus. The normal heart rate of the fetus is between 110 and 160 beats per minute. Any rhythm beyond these limits is abnormal and classed as a fetal arrhythmia. These are mainly the result of premature atrial contractions, usually give no symptoms, and have little consequence. However, around one percent of these will be the result of significant structural damage to the heart.

**Signs and symptoms**

The term cardiac arrhythmia covers a very large number of very different conditions.

The most common symptom of arrhythmia is an awareness of an abnormal heartbeat, called palpitations. These may be infrequent, frequent, or continuous. Some of these arrhythmias are harmless (though distracting for patients) but some of them predispose to adverse outcomes. Arrhythmias also cause chest pain and shortness of breath.

Some arrhythmias do not cause symptoms and are not associated with increased mortality. However, some asymptomatic arrhythmias *are* associated with adverse events. Examples include a higher risk of blood clotting within the heart and a higher risk of insufficient blood being transported to the heart because of a weak heartbeat. Other increased risks are of embolization and stroke, heart failure, and sudden cardiac death.

If an arrhythmia results in a heartbeat that is too fast, too slow, or too weak to supply the body's needs, this manifests as lower blood pressure and may cause lightheadedness, dizziness, syncope, loss of consciousness, coma, persistent vegetative state, or brain death due to insufficient supply of blood and oxygen to the brain.

Some types of arrhythmia result in cardiac arrest, or sudden death.

Medical assessment of the abnormality using an electrocardiogram is one way to diagnose and assess the risk of any given arrhythmia.

**Mechanism**

Cardiac arrhythmia is caused by one of two major mechanisms. The first type of arrhythmia is a result of enhanced or abnormal impulse formation originating at the pacemaker or the His-Purkinje network. The second is due to re-entry conduction disturbances.

**Diagnostic**

Cardiac arrhythmia is often first detected by simple but nonspecific means: auscultation of the heartbeat with a stethoscope or feeling for peripheral pulses. These cannot usually diagnose specific arrhythmias but can give a general indication of the heart rate and whether it is regular or irregular. Not all the electrical impulses of the heart produce audible or palpable beats; in many cardiac arrhythmias, the premature or abnormal beats do not produce an effective pumping action and are experienced as "skipped" beats.

The simplest *specific* diagnostic test for assessment of heart rhythm is the electrocardiogram (abbreviated ECG or EKG). A Holter monitor is an EKG recorded over a 24-hour period, to detect arrhythmias that may happen briefly and unpredictably throughout the day.

A more advanced study of the heart's electrical activity can be performed to assess the source of the aberrant heart beats. This can be accomplished in an electrophysiology study, an endovascular procedure that uses a catheter to "listen" to the electrical activity from within the heart, additionally if the source of the arrhythmias is found, often the abnormal cells can be ablated and the arrhythmia can be permanently corrected. *Transesophageal atrial stimulation* (TAS) instead uses an electrode inserted through the esophagus to a part where the distance to the posterior wall of the left atrium is only approximately 5–6 mm (remaining constant in people of different age and weight).Transesophageal atrial stimulation can differentiate between atrial flutter, AV nodal reentrant tachycardia and orthodromic atrioventricular reentrant tachycardia. It can also evaluate the risk in people with Wolff–Parkinson–White syndrome, as well as terminate supraventricular tachycardia caused by re-entry.

### Sources: https://www.ncbi.nlm.nih.gov/books/NBK558923/

## Causes and Risk Factors

## Primary Causes

* Structural Heart Disease: Coronary artery disease, cardiomyopathy, congenital defects.
* Electrolyte Imbalances: Hypokalemia, hypercalcemia, or hypomagnesemia disrupting electrical signaling.
* Infections: Myocardial inflammation from COVID-19 or viral myocarditis.

## What happens in your body to cause arrhythmia?

To better understand arrhythmia, let’s discuss how a healthy heart works.

Your heart has an electrical system that controls your heartbeat. This system consists of electrical impulses that travel through your heart, prompting it to contract and pump blood.

These impulses usually follow precise pathways*,* causing a steady heart rhythm. However, a disruption in your heart’s electrical system can lead to arrhythmia.

These disruptions can present in a few ways. In some cases, your sinoatrial (SA) node, which starts your heartbeat, can slow down or stop producing electrical impulses completely. In others, extra signal pathways can develop, or a blockage can exist along the pathway.

What causes the disruption can vary. Possible causes include:

* heart damage
* a heart’s structural irregularities
* electrolyte imbalances
* obstructive sleep apnea (breathing disruptions during sleep)
* too much heart stress or strain
* certain heart medications or medical procedures
* alcohol consumption
* thyroid problems
* illegal drug use

## What conditions can cause arrhythmia?

Several health conditions can lead to arrhythmia by affecting your heart’s electrical system:

* heart attack
* coronary artery disease
* congenital (present at birth) heart disease
* electrolyte disorders, such as potassium, sodium, and calcium imbalances
* overactive thyroid
* viral infections like COVID-19

According to a 2024 research review, atrial fibrillation (the most common arrhythmia type) can also be due to:

* hypertension (high blood pressure)
* obesity
* high blood pressure
* diabetes
* chronic obstructive pulmonary disease (COPD)

Many things can lead to, or cause, an arrhythmia including:

* Coronary artery disease, other heart problems, and previous heart surgery: narrowed heart arteries, a heart attack, prior heart surgery, heart failure, and other heart damage.
* High blood pressure: this increases your risk of developing coronary artery disease. It may also cause the walls of your heart to become stiff and thick.
* Congenital heart disease: heart abnormality from birth may affect your heart's rhythm.
* Thyroid problems: an overactive or underactive thyroid gland.
* Drugs and supplements: some over-the-counter cough and cold medicines, and some prescription drugs.
* [Diabetes](https://www.materprivate.ie/health-information/medical-conditions/article/diabetes-medical-conditions/diabetes): your risk of developing coronary artery disease and high blood pressure greatly increases with uncontrolled diabetes.
* Sleep apnoea: this disorder, in which your breathing is interrupted during sleep, can increase your risk of a slow heart rate and other arrhythmias.
* Electrolyte imbalance: substances in your blood called electrolytes — such as potassium, sodium, calcium and magnesium — can affect your heart and contribute to arrhythmia if their levels are too high or too low.
* Dehydration: not drinking adequate amounts of fluids each day.
* Drinking too much alcohol.
* Caffeine or nicotine use: caffeine, nicotine and other stimulants can cause your heart to beat faster.
* Drugs: illegal drugs, such as amphetamines and cocaine, may seriously affect your heart, leading to arrhythmias and even sudden death.

### Sources:

### https://www.healthline.com/health/arrhythmia,

* https://www.thelancet.com/journals/lanepe/article/PIIS2666-7762(23)00203-X/fulltext
* https://www.materprivate.ie/health-information/medical-conditions/article/heart-veins/arrhythmia

## Risk Factors

* Lifestyle: Excessive alcohol, caffeine, stimulants, or smoking.
* Medical Conditions: Hypertension, obesity, diabetes, sleep apnea, hyperthyroidism.
* Genetic Factors: Family history of arrhythmias or sudden cardiac death.

## What factors increase my risk of arrhythmia?

In addition to health conditions, several other factors can increase your risk of arrhythmia. These include:

* limited physical activity
* alcohol and caffeine consumption
* tobacco use
* family history of arrhythmia
* older ages

**What can trigger arrhythmia?**

If you have the above risk factors or underlying conditions, the following can cause arrhythmia:

* stress and anxiety
* heavy caffeine consumption
* high or low blood sugar levels
* certain illegal drugs
* physical overexertion
* some medications, like antidepressants, blood pressure medications, and certain cancer drugs
* dehydration

### SOURCES:

https://www.healthline.com/health/arrhythmia/arrhythmia-causes#triggers

## Signs and Symptoms

* Common: Palpitations, dizziness, fatigue, shortness of breath.
* Red Flags:
  + Chest pain radiating to the arm/jaw.
  + Syncope (fainting) or near-syncope.
  + Sudden weakness, confusion, or cardiac arrest.

### What are the symptoms of arrhythmia?

The symptoms of a slow heartbeat include:

* Fatigue
* Dizziness
* Light-headedness
* Fainting or near-fainting spells

The symptoms of a rapid heartbeat include:

* Palpitations
* Dizziness
* Light-headedness
* Fainting or near fainting

## 

## Diagnosis Methods

1. Electrocardiogram (ECG/EKG): Detects rhythm abnormalities in real-time.
2. Holter Monitor/Event Recorder: Captures intermittent episodes over 24–48 hours.
3. Echocardiogram: Assesses structural abnormalities (e.g., valve disease).
4. Blood Tests: Checks electrolytes, thyroid function, and cardiac enzymes.
5. Electrophysiology Study (EPS): Maps aberrant electrical pathways via catheters.

### How is arrhythmia diagnosed?

A heart arrhythmia can be detected by regular pulse checks and will only be diagnosed following a check-up with your doctor.

If your doctor is concerned, you will be referred to a consultant cardiologist with expertise in arrhythmia, for further evaluation and testing. This specialist is known as an electrophysiologist.

The following tests may be carried out:

* [ECG (electrocardiogram)](https://www.materprivate.ie/our-services/heart-vascular/electrocardiogram): records the heart rhythm and activity on a moving strip of paper. This is often the first test you will have, but it only captures information at that specific moment in time, so you may need additional tests.
* 24/48-hour ECG monitor: You may be asked to wear a portable ECG monitor for one or two days to record information on your heart’s activity over an extended period of time. This may be because the initial ECG was inconclusive.
* [Echo (echocardiogram)](https://www.materprivate.ie/our-services/heart-vascular/echocardiogram): An echo is a test that uses an ultrasound scan to look at the structure of your heart.
* [Event monitor](https://www.materprivate.ie/our-services/heart-vascular/cardiac-event-recorder): This is a device that is used over a period of time to record the heart activity when you are experiencing an arrhythmia. Some event monitors are implanted under the skin for several months.
* Coronary angiogram: This is a procedure that examines the coronary arteries in your heart to see if there is any narrowing caused by heart disease. It uses dye and an x-ray to show whether your blood is flowing freely or if your arteries are narrowed or blocked. It can also show any problems in the chambers of your heart or heart valves. This test is also sometimes called cardiac catheterization.

If an irregular heartbeat is not found during those tests, a healthcare professional may suggest more tests to try to trigger the arrhythmia. These tests may include:

* **Stress test.** Some arrhythmias are triggered or worsened by exercise. During a stress test, the heart's activity is watched while you ride on a stationary bicycle or walk on a treadmill. If you can't exercise, you may be given medicine that affects the heart in a way that's similar to exercise. This test takes a recording of your heart rate and rhythm using an ECG while you are exercising on a treadmill. Exercise can raise your heart rate, which may help to show any arrhythmias.
* **Tilt table test.** This test may be done if you've had fainting spells. Your heart rate and blood pressure are checked as you lie flat on a table. The table is then tilted to put you in a standing position. A healthcare professional watches how your heart and nervous system respond to the change in angle.
* **Electrophysiological (EP) testing and mapping.** This test, also called an EP study, can confirm a diagnosis of tachycardia or find out where in the heart the faulty signaling occurs. An EP study is mostly used to diagnose isolated arrhythmias.  
  An EP study is done in the hospital. One or more thin, flexible tubes are guided through a blood vessel, usually in the groin, to various areas in the heart. Sensors on the tips of the tubes record the heart's electrical activity. An EP study shows how electrical signals spread through the heart during each heartbeat. An EP study is a procedure used to examine the electrical function of the heart and irregularities in your heart rhythm. It is performed to find out why your heart beats too quickly or too slowly, or why it does not beat in a regular rhythm. It helps identify the location of the abnormal electrical signals in your heart.

SOURCES:

* https://www.mayoclinic.org/diseases-conditions/heart-arrhythmia/diagnosis-treatment/drc-20350674
* https://www.materprivate.ie/health-information/medical-conditions/article/heart-veins/arrhythmia

## 

## Treatment Options

Treatment for a heart arrhythmia depends on whether the heart is beating too fast or too slow. Some heart arrhythmias do not need treatment. Your healthcare team may suggest regular checkups to watch your condition.

Heart arrhythmia treatment is usually only needed if the irregular heartbeat causes significant symptoms or puts you at risk of more-serious heart problems. Treatment for heart arrhythmias may include medicines, special actions called vagal maneuvers, procedures or surgery.

## Medications

* Rate Control: Beta-blockers (metoprolol), calcium channel blockers (diltiazem).
* Rhythm Control: Antiarrhythmics (amiodarone, flecainide).
* Anticoagulants: Apixaban or warfarin for stroke prevention in AFib.

Medicines used to treat heart arrhythmias depend on the type of irregular heartbeat and possible complications.

For example, most people with tachycardia are given medicine to control the heart rate and rhythm.

If you have atrial fibrillation, blood thinners may be given to prevent blood clots.

### Therapies

Other treatments for heart arrhythmias include:

* **Vagal maneuvers.** These are simple but specific actions that can slow the heart rate. They include coughing, bearing down as if having a bowel movement and putting an ice pack on the face. These actions affect the vagus nerve. The nerve helps control the heartbeat. Vagal maneuvers may be recommended if you have a very fast heartbeat due to supraventricular tachycardia. Vagal maneuvers don't work for all types of arrhythmias.
* **Cardioversion.** Paddles or patches on the chest are used to give an electrical shock to the heart and help reset the heart rhythm. Cardioversion is typically used when vagal maneuvers and medicines don't work. Your healthcare team may recommend this treatment if you have a certain type of arrhythmia, such as atrial fibrillation.

Treatment for heart arrhythmias also may involve a procedure or surgery to place a heart device in your body. Sometimes, open-heart surgery is needed to stop or prevent an irregular heartbeat.

Types of procedures and surgeries used to treat heart arrhythmias include:

* **Catheter ablation.** In this procedure, the doctor places one or more catheters into blood vessels to the heart. Sensors at the catheter tips use heat or cold energy to create tiny scars in your heart. The scars block irregular heart signals and restore the heartbeat.
* **Pacemaker.** If slow heartbeats don't have a cause that can be fixed, a pacemaker may be needed. A pacemaker is a small device that's placed in the chest to help control the heartbeat.
* **Implantable cardioverter-defibrillator (ICD).** This device is placed under the skin near the collarbone. It continuously checks the heart rhythm. If the device finds an irregular heartbeat, it sends out low- or high-energy shocks to reset the heart's rhythm.  
  You may need this device if you have a high risk of dangerously fast or irregular heartbeats in the lower heart chambers. Such conditions are called ventricular tachycardia or ventricular fibrillation. Other reasons for an ICD include a history of sudden cardiac arrest or conditions that increase its risk.
* **Maze procedure.** In the maze procedure, a surgeon makes tiny cuts in the upper half of the heart to create a pattern of scar tissue. The pattern is called a maze. The heart's signals can't pass through scar tissue. This treatment can block stray electrical heart signals that cause some types of fast heartbeats.  
  The maze procedure is usually only done if you don't get better with other treatments or if you're already having open-heart surgery for another reason.
* **Coronary bypass graft surgery.** If you have severe coronary artery disease with an irregular heartbeat, you may need this type of heart surgery. The surgery creates a new path for blood to flow around a blocked or partially blocked artery in the heart.

After treatment for irregular heartbeats, it's important to get regular health checkups. Take your medicines as directed. Tell your healthcare team if your symptoms get worse.

SOURCES:

* https://www.mayoclinic.org/diseases-conditions/heart-arrhythmia/diagnosis-treatment/drc-20350674

## Procedures and Devices

* Cardioversion: Electrical shocks restore sinus rhythm in AFib/VFib.
* Catheter Ablation: Destroys arrhythmia-causing tissue using radiofrequency/cryotherapy.
* Implantable Devices:
  + *Pacemakers*: Treat bradycardia.
  + *ICDs*: Terminate life-threatening VT/VFib.

## Lifestyle Modifications

* Limit alcohol/caffeine, manage stress, quit smoking.
* Regular exercise and a heart-healthy diet.

**Lifestyle and home remedies**

Making lifestyle changes can help keep your heart as healthy as possible.

Examples of heart-healthy lifestyle changes are

* **Eat heart-healthy foods.** Eat a healthy diet that's low in salt and solid fats and rich in fruits, vegetables and whole grains.
* **Get regular exercise.** Try to exercise for at least 30 minutes on most days.
* **Don't smoke.** If you smoke and can't quit on your own, talk to a healthcare professional about strategies or programs to help.
* **Maintain a healthy weight.** Being overweight increases the risk of heart disease. Talk with your care team to set realistic goals for body mass index (BMI) and weight.
* **Control blood pressure and cholesterol.** High blood pressure and high cholesterol increase the risk of heart disease. Make lifestyle changes and take medicines as directed to manage high blood pressure or high cholesterol.
* **Limit alcohol.** If you choose to drink alcohol, do so in moderation. For healthy adults, that means up to one drink a day for women of all ages and men older than age 65, and up to two drinks a day for men age 65 and younger.
* **Practice good sleep habits.** Poor sleep may increase the risk of heart disease and other chronic conditions. Adults should aim to get 7 to 9 hours of sleep daily. Go to bed and wake at the same time every day, including on weekends. If you have trouble sleeping, talk to a healthcare professional about strategies that might help.
* **Manage stress.** Managing stress is an important step in keeping the heart healthy. Getting more exercise, practicing mindfulness and connecting with others in support groups are some ways to reduce and manage stress.

**Preparing for your appointment**

Medical appointments can be brief. There's often a lot to discuss. So it's a good idea to be prepared for your appointment. Here's some information to help you prepare for your appointment.

### What you can do

* **Be aware of any pre-appointment restrictions.** When you make the appointment, ask if there's anything you need to do in advance. For example, you may be told not to eat or drink for a few hours before a cholesterol test.
* **Write down any symptoms you're having,** including any that may seem unrelated to heart arrhythmias.
* **Write down important personal information,** including a family history of heart disease, stroke, high blood pressure or diabetes, and any major stresses or recent life changes.
* **Make a list of all medicines,** including vitamins or supplements that you're taking. Include dosages.
* **Take someone with you,** if possible. Someone who goes with you can help you remember information you're given.
* **Write down questions to ask** your care team.

Prepare a list of questions from most important to least important in case time runs out. For heart arrhythmias, some basic questions to ask your healthcare team include:

* What's the most likely cause of my symptoms?
* Are there other possible causes for my symptoms?
* What kinds of tests will I need? Do I need to do anything to prepare for these tests?
* What's the most appropriate treatment?
* Are there any foods or drinks I should avoid? Is there anything I should add to my diet?
* What's an appropriate level of physical activity?
* How often should I be screened for heart disease or other complications of an arrhythmia?
* I have other health conditions. How can I best manage these conditions together?
* Is there a generic option to the medicine you're prescribing?
* Are there any brochures or other printed material that I can take home with me? What websites do you recommend visiting?

### What to expect from your doctor

Your healthcare team is likely to ask you questions, such as:

* When did you first begin having symptoms?
* Do you always have symptoms, or do they come and go?
* How severe are your symptoms?
* Does anything seem to improve your symptoms?
* What, if anything, makes your symptoms worse?
* Does anyone in your family have a heart arrhythmia?

SOURCES:

* https://www.mayoclinic.org/diseases-conditions/heart-arrhythmia/diagnosis-treatment/drc-20350674

## Prevention Tips

* Underlying Condition Management: Control hypertension, diabetes, and sleep apnea.
* Avoid triggers: over-the-counter decongestants (pseudoephedrine) and illicit drugs.

## Can I prevent arrhythmia?

While you can’t change some risk factors like age and genetics, you can reduce your risk of arrhythmia by:

* eating a balanced diet
* exercising regularly
* maintaining a moderate weight
* quitting smoking if you smoke
* limiting or avoiding alcohol and caffeine consumption
* avoiding illegal substances
* managing stress with techniques such as meditation, yoga, or deep breathing exercises
* scheduling regular checkups with a doctor, especially if you have conditions or risk factors associated with arrhythmia

## Takeaway

Several underlying conditions and risk factors can lead to arrhythmia. While some factors are beyond your control, adopting a healthy lifestyle and managing underlying health conditions can significantly reduce your risk and help you maintain a healthy heart.

If you experience arrhythmia symptoms, such as a fluttering in your chest or dizziness, schedule a prompt appointment with a healthcare professional for a proper diagnosis and treatment plan. Get emergency medical attention if you have sudden shortness of breath, chest pain, or fainting.

### SOURCES

https://www.healthline.com/health/arrhythmia/arrhythmia-causes#triggers

## Prognosis

* Benign Arrhythmias: Premature beats often require no treatment.
* Chronic AFib: Increases stroke risk 5-fold but is manageable with anticoagulation.
* VFib: Survival depends on immediate CPR/defibrillation.

## Possible Complications

* Stroke: AFib causes 15–20% of ischemic strokes.
* Heart Failure: Chronic tachycardia weakens myocardial contractility.
* Sudden Cardiac Death: VFib accounts for 50% of cardiac deaths.

**SOURCES:**

https://www.heart.org/en/health-topics/arrhythmia/symptoms-diagnosis--monitoring-of-arrhythmia

## When to See a Doctor

* Persistent palpitations with lightheadedness or chest discomfort.
* Family history of sudden cardiac death or congenital arrhythmia syndromes.

**SOURCES:**

https://www.heart.org/en/health-topics/arrhythmia/symptoms-diagnosis--monitoring-of-arrhythmia

## 

## 

## 

## 

## 

## Differential Diagnosis

| **Condition** | **Distinguishing Features** |
| --- | --- |
| Panic Attack | No ECG abnormalities; situational anxiety triggers. |
| Heart Attack | Elevated troponin, ST-segment changes on ECG. |
| Hyperthyroidism | Elevated TSH, weight loss, heat intolerance. |

**Normal electrical activity**

Each heartbeat originates as an electrical impulse from a small area of tissue in the right atrium of the heart called the sinus node or sinoatrial node (SA node). The impulse initially causes both atria to contract, then activates the atrioventricular node (AV node), which is normally the only electrical connection between the atria and the ventricles (main pumping chambers). The impulse then spreads through both ventricles via the bundle of His and the Purkinje fibers, causing a synchronized contraction of the heart muscle and, thus, the pulse.

In adults, the normal resting heart rate ranges from 60 to 90 beats per minute. The resting heart rate in children is much faster. In athletes, however, the resting heart rate can be as slow as 40 beats per minute and be considered normal. The term "sinus arrhythmia" refers to a normal phenomenon of alternating mild acceleration and slowing of the heart rate that occurs with breathing in and out, respectively. It is usually quite pronounced in children and steadily decreases with age. This can also be present during meditation breathing exercises that involve deep inhaling and breath-holding patterns.

**Bradycardias**

A slow rhythm (less than 60 beats/min) is labelled bradycardia. This may be caused by a slowed signal from the sinus node (sinus bradycardia), by a pause in the normal activity of the sinus node (sinus arrest), or by blocking of the electrical impulse on its way from the atria to the ventricles (AV block or heart block). Heart block comes in varying degrees and severity. It may be caused by reversible poisoning of the AV node (with drugs that impair conduction) or by irreversible damage to the node. Bradycardias may also be present in the normally functioning heart of endurance athletes or other well-conditioned persons. Bradycardia may also occur in some types of seizures.

**Tachycardias**

In adults and children over 15, a resting heart rate faster than 100 beats per minute is labeled tachycardia. Tachycardia may result in palpitations; however, tachycardia is not *necessarily* an arrhythmia. Increased heart rate is a normal response to physical exercise or emotional stress. This is mediated by the sympathetic nervous system on the sinus node and called sinus tachycardia. Other conditions that increase sympathetic nervous system activity in the heart include ingested or injected substances, such as caffeine or amphetamines, and an overactive thyroid gland (hyperthyroidism) or anemia.

Tachycardia that is not sinus tachycardia usually results from the addition of abnormal impulses to the normal cardiac cycle. Abnormal impulses can begin by one of three mechanisms: automaticity, re-entry, or triggered activity. A specialized form of re-entry which is both common and problematic is termed fibrillation.

Although the term "tachycardia" has been known for over 160 years, bases for the classification of arrhythmias are still being discussed.

**Heart defects**

Congenital heart defects are structural or electrical pathway problems in the heart that are present at birth. Anyone can be affected by this because overall health does not play a role in the problem. Problems with the electrical pathway of the heart can cause very fast or even deadly arrhythmias. Wolff–Parkinson–White syndrome is due to an extra pathway in the heart that is made up of electrical muscle tissue. This tissue allows the electrical impulse, which stimulates the heartbeat, to happen very rapidly. Right ventricular outflow tract tachycardia is the most common type of ventricular tachycardia in otherwise healthy individuals. This defect is due to an electrical node in the right ventricle just before the pulmonary artery. When the node is stimulated, the patient will go into ventricular tachycardia, which does not allow the heart to fill with blood before beating again. Long QT syndrome is another complex problem in the heart and has been labeled as an independent factor in mortality. There are multiple methods of treatment for these including cardiac ablations, medication treatment, or lifestyle changes to have less stress and exercise.

**Automaticity**

Automaticity refers to a cardiac muscle cell firing off an impulse on its own. All of the cells in the heart have the ability to initiate an action potential; however, only some of these cells are designed to routinely trigger heartbeats. These cells are found in the conduction system of the heart and include the SA node, AV node, Bundle of His, and Purkinje fibers. The sinoatrial node is a single specialized location in the atrium that has a higher automaticity (a faster pacemaker) than the rest of the heart and, therefore, is usually responsible for setting the heart rate and initiating each heartbeat.

Any part of the heart that initiates an impulse without waiting for the sinoatrial node is called an ectopic focus and is, by definition, a pathological phenomenon. This may cause a single premature beat now and then, or, if the ectopic focus fires more often than the sinoatrial node, it can produce a sustained abnormal rhythm. Rhythms produced by an ectopic focus in the atria or by the atrioventricular node are the least dangerous dysrhythmias, but they can still produce a decrease in the heart's pumping efficiency because the signal reaches the various parts of the heart muscle with different timing than usual and can be responsible for poorly coordinated contraction.

Conditions that increase automaticity include sympathetic nervous system stimulation and hypoxia. The resulting heart rhythm depends on where the first signal begins: if it is the sinoatrial node, the rhythm remains normal but rapid; if it is an ectopic focus, many types of dysrhythmia may ensue.

**Re-entry**

Re-entrant arrhythmias occur when an electrical impulse recurrently travels in a tight circle within the heart, rather than moving from one end of the heart to the other and then stopping.

Every cardiac cell can transmit impulses of excitation in every direction but will do so only once within a short time. Normally, the action potential impulse will spread through the heart quickly enough that each cell will respond only once. However, if there is some essential heterogeneity of refractory period or if conduction is abnormally slow in some areas (for example, in heart damage) so the myocardial cells are unable to activate the fast sodium channel, part of the impulse will arrive late and potentially be treated as a new impulse. Depending on the timing, this can produce a sustained abnormal circuit rhythm.

As a sort of *re-entry*, vortices of excitation in the myocardium (autowave vortices) are considered to be the main mechanism of life-threatening cardiac arrhythmias. In particular, the autowave reverberator is common in the thin walls of the atria, sometimes resulting in atrial flutter. Re-entry is also responsible for most paroxysmal supraventricular tachycardia and dangerous ventricular tachycardia. These types of re-entry circuits are different from WPW syndromes, which utilize abnormal conduction pathways.

Although omega-3 fatty acids from fish oil can be protective against arrhythmias, they can facilitate reentrant arrhythmias.

**Fibrillation**

When an entire chamber of the heart is involved in multiple micro-re-entry circuits and is, therefore, quivering with chaotic electrical impulses, it is said to be in fibrillation.

Fibrillation can affect the atrium (atrial fibrillation) or the ventricle (ventricular fibrillation): ventricular fibrillation is imminently life-threatening.

* Atrial fibrillation affects the upper chambers of the heart, known as the atria. Atrial fibrillation may be due to serious underlying medical conditions and should be evaluated by a physician. It is not typically a medical emergency.
* Ventricular fibrillation occurs in the ventricles (lower chambers) of the heart; it is always a medical emergency. If left untreated, ventricular fibrillation (VF, or V-fib) can lead to death within minutes. When a heart goes into V-fib, effective pumping of the blood stops. V-fib is considered a form of cardiac arrest. An affected individual will not survive unless cardiopulmonary resuscitation (CPR) and defibrillation are provided immediately.

CPR can prolong the survival of the brain in the lack of a normal pulse, but defibrillation is the only intervention that can restore a healthy heart rhythm. Defibrillation is performed by applying an electric shock to the heart, which resets the cells, permitting a normal beat to re-establish itself.

**Triggered beats**

Triggered beats occur when problems at the level of the ion channels in individual heart cells result in abnormal propagation of electrical activity and can lead to a sustained abnormal rhythm. They are relatively rare and can result from the action of antiarrhythmic drugs or after depolarizations.

### SOURCES

https://en.wikipedia.org/wiki/Arrhythmia

## Recent Guidelines and Epidemiology

* Prevalence: AFib affects 1–2% of the U.S. population, with a lifetime risk of 1 in 4 for adults >40.
* Racial Disparities: Lower AFib incidence in Black/Hispanic populations despite higher comorbidities.
* 2023 AHA/ACC Guidelines: Recommend early rhythm control for AFib and expanded ICD use in high-risk patients