CME

# Recognizing postural orthostatic tachycardia syndrome in primary care

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## ABSTRACT

Postural orthostatic tachycardia syndrome (POTS) is a complex autonomic disorder characterized by abnormal tachycardia on standing. This disorder predominantly affects young women, with a peak incidence between ages 20 and 30 years. POTS often is associated with a variety of symptoms, including dizziness, palpitations, fatigue, generalized weakness, anxiety, and exercise intolerance, which can significantly impair patient quality of life. Primary care providers (PCPs) often are the first healthcare professionals to whom patients present. This article provides an overview of POTS, including diagnostic criteria and the importance of a thorough clinical evaluation to rule out other causes of symptoms. PCPs play a critical role in the comprehensive approach to care, involving patient education, lifestyle modifications, and treatment to improve outcomes and enhance patient quality of life.


**Keywords:** postural orthostatic tachycardia syndrome, primary care, autonomic, dizziness, palpitations, Ehlers Danlos syndrome

## Learning objectives

- Define POTS and its clinical manifestations.
- Explain the various tests and assessments used to diagnose POTS.
- Describe treatment options for patients with POTS.
- Discuss the importance of patient education and empowerment in the management of POTS and describe strategies to promote self-management and active participation in care.

Postural orthostatic tachycardia syndrome (POTS) is a "clinically heterogeneous disorder with multiple contributing pathophysiologic mechanisms manifesting as symptoms of orthostatic intolerance in the setting of orthostatic tachycardia without orthostatic hypotension."[1] POTS previously has been known as neurocirculatory asthenia, mitral valve prolapse syndrome, hyperdynamic beta-adrenergic state, irritable heart, soldier's heart, and Da Costa syndrome.[2] Management typically involves lifestyle modifications, such as increasing fluid and salt intake, as well as pharmacologic therapy with beta-blockers or other medications that affect autonomic function. In addition, patient education and support are crucial, given the effect of POTS on patients' daily functioning and quality of life. Because of the nonspecific nature of the symptoms and lack of awareness of this condition among healthcare providers, POTS often is misdiagnosed or overlooked. Delay in diagnosis or misdiagnosis can lead to unnecessary medical workups and treatments and can result in prolonged reduction in quality of life for patients.

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## EPIDEMIOLOGY

POTS is about four to five times more common among women than men, and typically affects patients between ages 15 and 25 years. The prevalence of POTS is about 0.2% to 1% of the US population, or 1 to 3 million people. The syndrome also has been associated with other conditions, such as chronic fatigue syndrome, Ehlers-Danlos syndrome (EDS), vasovagal syncope, fibromyalgia, and migraine.[3,4]

## PATHOPHYSIOLOGY

Standing upright requires the involvement of the neurologic, cardiovascular, and musculoskeletal systems. When a patient changes position, the resulting changes in blood

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**Key points**

- Symptoms of POTS include orthostatic intolerance, tachycardia, dizziness, and fatigue.
- POTS can mimic other medical conditions such as anxiety, cardiac conditions, syncope, and anxiety.
- PCPs should be knowledgeable about initial management strategies for patients with POTS, including lifestyle modifications, fluid intake, salt supplementation, and medications.

flow require the autonomic nervous system to increase sympathetic tone and decrease parasympathetic response. Increased sympathetic activity and the reduction of parasympathetic innervation lead to vasoconstriction, increased heart rate, and increased inotropy. Lastly, the musculoskeletal system assists in venous return to the heart, allowing cardiac output to perfuse the rest of the body.

Normally, standing results in an increase in heart rate of 10 to 30 beats/minute with a negligible change in systolic BP and a small increase (about 5 mm Hg) in diastolic BP. However, patients with POTS have an inappropriate autonomic response to changes in position or activity. POTS is a syndrome or a phenotype, not a disease, and is best described as a clinically heterogenous disorder with numerous contributing pathophysiologic mechanisms. Although the cause of POTS remains unknown, three major pathophysiologic mechanisms or endophenotypes have been described:

• *Partial autonomic neuropathy* or *neuropathic POTS* is characterized by autonomic neuropathy in vascular beds leading to arteriolar dysregulation and blood stasis that primarily affects the pelvic, splanchnic, and lower extremities regions.[1]

• *Persistent predilection for hypovolemia* or *hypovolemic POTS* is characterized by persistent hypovolemia, which may be caused by low blood volume, low plasma renin, and low aldosterone levels leading to lower levels of sodium and water retention and increased vasoconstriction.[1] Many patients with this phenotype are deconditioned, which contributes to a decrease in venous return and reactive tachycardia. This is further exacerbated by patients' lack of physical activity as they seek to avoid the symptoms that occur with activity. This cycle further worsens deconditioning, hypovolemia, and the syndrome itself.

• *Central hyperadrenergic state* or *hyperadrenergic POTS* is characterized by elevated plasma norepinephrine leading to an overactive sympathetic nervous system.[1]

The onset of POTS may be triggered by immunologic stressors such as viral infections, vaccinations, trauma, pregnancy, surgery, or intense psychosocial stress.[5] Most patients will not be able to identify a primary event that precipitated onset.

In 2020, postacute COVID-19 syndrome or *long COVID* was recognized and associated with a wide array of varying signs and symptoms, such as fatigue, disability, heart palpitations, orthostasis and exercise intolerance, and shortness of breath. Clinicians and researchers alike noted that some of the symptoms of long COVID were the same as in POTS, and patient responses to cardiovascular autonomic tests were similar. Other presentations of cardiovascular autonomic dysfunction (such as inappropriate sinus tachycardia) also were recognized.[6] As a result of subsequent research and surveillance, POTS now is considered a major phenotype in postacute COVID-19 syndrome.[7] The exact link between COVID-19 and POTS is not well understood. The increase in post-COVID-19 POTS could be a specific phenomenon and a unique mechanism of the SARS-CoV-2 virus, or could be because COVID-19 is a commonly diagnosed virus that triggers a more general postviral response.[8] Theories of how COVID-19 can cause POTS include overactivation of the sympathetic nervous system secondary to proinflammatory cytokines cross-reaction of antibodies with the autonomic nervous system.[9,10] As research into the association of POTS and COVID-19 continues, clinicians must recognize the risk of POTS in patients who had COVID-19. Test patients with postacute COVID-19 syndrome for POTS, which appears in about 30% of highly symptomatic patients.[11] Explain that not everyone who contracts COVID-19 will develop POTS and that POTS has many causes unrelated to the virus. By providing clear and accurate information, clinicians can address concerns appropriately and provide support to patients who are experiencing symptoms related to POTS.

## CLINICAL FEATURES

POTS is characterized by an increase in heart rate of 30 beats/minute or more within 10 minutes of assuming an upright posture from a supine position, without orthostatic hypotension.[12] Symptoms associated with this orthostatic intolerance vary in quality and severity, and include:

• *Cardiovascular signs and symptoms* such as tachycardia, palpitations, weakness, and chest pain.

• *Noncardiac signs and symptoms* such as flushing, dizziness, presyncope, syncope, tremulousness, depression, anxiety, nausea, bowel changes, abdominal pain, hypermobility, arthralgia, fatigue, and sleep disturbance.[5]

The signs and symptoms of POTS can be disabling, and clinicians should remember to include an evaluation of patient daily function, mental health, and behavioral symptoms. Ask patients if they are aware of any symptom triggers. Common symptom triggers include standing still (for example, waiting in line at a store or standing in front of a stove while cooking), bedrest, heat exposure, food or alcohol ingestion, pregnancy, or certain medications.[12,13] Most patients report at least 10 different symptoms, which makes diagnosis cumbersome.[5]

**TABLE 1.** Differential diagnosis for POTS[15,16]

| Differential diagnosis | Presentation |
|---|---|
| POTS | Continuous tachycardia, lightheadedness, palpitations in response to upright posture without hypotension |
| Vasovagal syncope | • Episodic symptoms<br>• Syncope with fall in BP and heart rate during tilt-table test |
| Orthostatic hypotension | • Postural tachycardia with hypotension due to volume depletion or medications<br>• Immediate fall in BP with or without increase in heart rate in upright posture during tilt-table test |
| Pseudosyncope | Loss of consciousness or inability to maintain posture without fall in BP or heart rate during tilt-table test |
| Dehydration | Compensatory tachycardia and orthostatic hypotension due to intravascular hypovolemia |
| Pharmacologic syndromes | Heart rate can be influenced by sympathomimetics, serotonin-norepinephrine reuptake inhibitors, tricyclic antidepressants, atomoxetine, and anticholinergics |
| Inappropriate sinus tachycardia | Unexplained tachycardia at rest |
| Anxiety disorder | Spontaneous tachycardia caused by anxiety or panic attacks |

## DIAGNOSIS

A thorough physical examination with orthostatic vital signs is an important step in diagnosing POTS. Diagnostic criteria are a heart rate increased by 30 beats/minute or more (40 beats/minute in patients ages 12 to 20 years) within 10 minutes of changing from a supine to an upright posture, absence of orthostatic hypotension, and presence of symptoms for 3 months (more than 6 months preferred).[14] As many as half of patients with POTS may experience acrocyanosis (discoloration of the legs from the feet to above the knees) with standing.[13] Diagnostic testing for POTS is another important step in evaluating patients with suspected autonomic dysfunction. Testing includes orthostatic vital signs, tilt-table testing, autonomic function tests, and hematologic and thyroid tests to evaluate for anemia and assess thyroid function. Exclude other causes by assessing electrolytes, complete blood cell (CBC) count, thyroid function testing, cortisol, and ECG. Obtain an orthostatic BP, portable ECG monitoring test, and perform the Dix-Hallpike maneuver to rule out vestibular and cardiovascular pathology.

Obtain a thorough past medical history, asking about hypermobility or EDS. EDS is a group of inherited connective tissue disorders characterized by hypermobility and hyperextensibility. Of the 13 subtypes, all have identified genetic defects except for hypermobile EDS, the subtype most associated with POTS.[14] However, the relationship between EDS and POTS is still unclear because of a lack of scientific validity, despite the overlap of symptoms such as orthostatic intolerance, dizziness, weakness, fatigue, and palpitations.[14]

A patient presenting with symptoms and physical examination findings suggestive of POTS should be referred to neurology for a tilt-table test, which increases the diagnostic yield for diagnosis of POTS. During this test, the patient is secured supine on a table and gradually tilted to an almost upright position for 20 to 45 minutes, while ECG, BP, heart rate, and symptoms are monitored. A normal response is no change or a slight increase in BP and heart rate until the patient is returned to the supine position. A positive POTS test is indicated by lightheadedness, palpitations, and a sustained increase in heart rate of more than 30 beats/minute (40 beats/minute in adolescents) within 10 minutes of starting the head-up tilt, without any accompanying drop in BP.[15]

The differential diagnosis list to be considered in patients with symptoms of POTS is long (**Table 1**). To narrow the list, first consider if structural heart disease needs to be excluded. Next, consider if the patient is experiencing isolated orthostatic intolerance, because not all patients with orthostatic intolerance have POTS but all patients with POTS have orthostatic intolerance. Then, informed by the history and physical examination, consider relevant conditions, such as those that cause orthostatic intolerance or tachycardia. Finally, assess for aggravating medications (diuretics, vasodilators, amphetamines, beta-agonists) or recently withdrawn medications (beta-blockers, clonidine) as well as conditions such as prolonged bedrest that may be contributing to presenting symptoms.

Anxiety disorders are a common differential; however, if the patient has tachycardia or palpitations with position change and not spontaneously, this excludes anxiety as a primary diagnosis. The diagnosis of POTS often is a clinical one, based on a thorough history and physical examination, and diagnostic testing is meant to support rather than replace the clinical diagnosis. PCPs should work closely with neurology specialists in autonomic disorders to ensure appropriate testing and management of patients with POTS. By doing so, they can help improve patient outcomes and quality of life.

## TREATMENT

Treatment for POTS involves a multifaceted approach that targets the underlying pathophysiologic mechanisms and aims to alleviate symptoms and improve patient quality of life.[5] Treatment is individualized based on the patient's symptoms, comorbidities, and response to previous treatment, but generally begins with nonpharmacologic options,

then integrates pharmacologic treatments if necessary. Referral to a cardiologist or autonomic specialist may be necessary for patients with persistent or severe symptoms after initial treatment.

PCPs should collaborate with specialists and the patient to develop a treatment plan that considers the patient's goals. Patients with POTS require ongoing follow-up and monitoring to assess treatment response and adjust the treatment plan as needed. Initial nonpharmacologic approaches include increased fluid and salt intake to increase blood volume, exercise for reconditioning, compression garments to minimize venous pooling, and avoidance of symptom triggers. The fluid intake goal is about 3 L/day and salt intake should be 8 to 12 g/day.[1] Suggested sources are table salt, sports drinks, broth, soups, and chips. PCPs also can provide IV hydration for up to 2 days as needed if a patient is unable to tolerate oral treatment.[16] Encourage a slow introduction of aerobic exercise and leg resistance training to prevent deconditioning, which can lead to worsening of POTS.[1]

Other lifestyle management recommendations include behavioral and environmental changes, such as encouraging patients to be upright as much as possible, counseling, cognitive behavioral therapy, physical therapy, improved sleep hygiene, and awareness of triggers. If lifestyle modifications have not helped patients, or if patients develop more severe symptoms, encourage them to use compression stockings or abdominal binders to reduce venous pooling.[5]

Pharmacologic interventions (**Table 2**), including medications to increase blood volume and improve heart rate control, also may be used in conjunction with nonpharmacologic interventions to achieve optimal symptom control. No medications have been approved by the FDA to treat POTS; thus, pharmacologic treatments are prescribed off-label. Clinicians can refer to consensus recommendations to support diagnosis and treatment of POTS, including off-label prescribing.[3]

If nonpharmacologic approaches fail to relieve symptoms, pharmacologic therapies can be targeted to treat the cause of the patient's specific symptoms and subset of POTS.

**TABLE 2.** Treatment recommendations for POTS subtypes[1,20]

| Treatment | Dosing | Adverse reactions | Precautions |
|---|---|---|---|
| ***Neuropathic*** | | | |
| Compression stockings | 30-40 mm Hg, waist-high preferred | Discomfort | |
| Midodrine | 2.5-15 mg orally three times per day | Hypertension, paresthesia, visual disturbance | Use with caution in patients with severe cardiac disease or renal impairment. |
| ***Hypovolemic*** | | | |
| Exercise: Aerobic + leg resistance | 30 minutes/day for 4 days per week | Patients will feel worse for a few weeks | |
| Hydration | 3 L/day | Polyuria | Use with caution in patients with hypertension, heart failure, or hyponatremia. |
| Salt intake | 8-12 g/day | Hypertension, edema | Use with caution in patients with hypertension, heart failure, or hypernatremia. |
| Fludrocortisone | 0.1-0.2 mg once per day | Hypokalemia, Cushing disease, weight gain, edema, hypernatremia | Taper patients off medication as needed. |
| Desmopressin | 0.1-0.2 mg once per day | Hyponatremia, edema, seizures | Avoid in patients with hyponatremia, polydipsia, uncontrolled hypertension, or heart failure. |
| ***Hyperadrenergic*** | | | |
| Propranolol | 10-20 mg orally four times per day | Hypotension, bradycardia, bronchospasm, fatigue, exercise intolerance | Avoid in patients with obstructive lung disease or bradycardia. |
| Pyridostigmine | 30-60 mg three times per day | Flatulence, diarrhea, cramping | Avoid in patients with obstructive lung disease. |
| Droxidopa | 100-600 mg three times per day | Headache, dizziness, hypertension | Use with caution in patients with cardiovascular disease. |
| Ivabradine | 2.5 mg once or twice per day | Bradycardia, hypertension, visual disturbance | Use with caution in patients with cardiac conduction defects. |
| ***All types*** | | | |
| Methylphenidate | 10 mg two to three times per day | Tachycardia, insomnia, headache, dizziness | Reserve for patients with refractory POTS. |

Consider the medication's mechanism of action in relation to POTS when prescribing therapy. Treatment can be guided by the Heart Rhythm Society recommendations based on benefit versus risk and levels of evidence.[3] The following medications are recommended:

- *Nonselective or selective beta-blockers* (benefit equivalent to or exceeding risk, evidence variable) are beneficial in patients with hyperadrenergic POTS because they suppress the sympathetic nervous system, reducing heart rate fluctuations and palpitations.[1,3]
- *Midodrine* (benefit equivalent to or exceeding risk, evidence variable) is an alpha-adrenergic agonist that increases vascular tone, aiding in arterial and venous return.[1,3] This drug is the mainstay pharmacologic treatment for patients with neuropathic POTS.[1,3]
- *Fludrocortisone* (benefit equivalent to or exceeding risk, evidence from weaker studies) is a mineralocorticoid that aids in intravascular volume expansion and sodium retention, and primarily is used in patients with hypovolemic POTS.[1,3]
- *Pyridostigmine* (benefit equivalent to or exceeding risk, evidence from weaker studies) increases parasympathetic nervous system tone, helping to reduce heart rate fluctuations in patients with hyperadrenergic POTS.[1,3]
- *Clonidine* and *methyldopa* (benefit equivalent to or exceeding risk, consensus opinion) are central-acting sympathetic agents that reduce sympathetic activity.[1,3] These drugs primarily are used in patients with hyperadrenergic POTS.[1,3]
- *Droxidopa* is a prodrug of norepinephrine that aids in peripheral vasoconstriction and is used in patients with neuropathic POTS.[1,3]
- *Ivabradine*, a hyperpolarization-activated cyclic nucleotide-gated channel blocker, has been shown to improve symptoms of orthostatic intolerance in patients with hyperadrenergic POTS by reducing heart rate without affecting the sympathetic nervous system.[1,3]
- *Modafinil* is used to help with fatigue in patients with POTS by binding to dopamine transporter and inhibiting dopamine reuptake.[1,3]
- *Methylphenidate* is a postsynaptic alpha receptor agonist that has been shown to improve fatigue and presyncope/syncope in patients with POTS.[1,3,17]

The length of treatment for POTS varies depending on the patient and the underlying cause of the condition. Some patients may require ongoing treatment for an extended period; others may see improvement and discontinue treatment after a year.[18] Patients must work closely with their PCP and specialist (cardiology, neurology) to individualize their treatment plan and regularly monitor symptoms and therapy effectiveness.

Treatment of POTS benefits from a multidisciplinary approach that addresses the underlying pathophysiologic mechanisms and aims to alleviate symptoms and improve quality of life. PCPs play a crucial role in the diagnosis, management, and ongoing care of patients with POTS. PCPs should provide patients with accurate and up-to-date information about their condition, encourage self-management strategies, and promote active patient participation in care.[5] Explain POTS to patients and encourage them to avoid immobility, prolonged recumbency, prolonged standing, and physical deconditioning. Advise patients to rise slowly from supine or sitting positions, especially in the morning, after meals, and during micturition and defecation. Patients should eat small and frequent meals, and avoid high ambient temperatures and high humidity.[5] Teach patients physical countermaneuvers such as squatting, leg crossing, and muscle tensing during standing and onset of symptoms.[5]

PCPs and patients must understand which medications to avoid. Medications that inhibit norepinephrine transporters, such as reboxetine and atomoxetine, can worsen symptoms in patients with POTS.[1] Selective serotonin reuptake inhibitors and serotonin-norepinephrine reuptake inhibitors also can worsen symptoms because of the nonselective inhibition of norepinephrine.[1]

In summary, PCPs should be aware of the various treatment options available for patients with POTS, collaborate closely with other specialists, and provide comprehensive, patient-centered care to improve outcomes and enhance quality of life.

## PROGNOSIS

POTS is a chronic condition that can significantly impair patient quality of life. Although POTS can significantly reduce patient quality life, it is not associated with increased mortality.[3,13] Some patients experience mild symptoms that can be managed effectively with lifestyle modifications; others may have severe and disabling symptoms that require more aggressive treatment. Studies have shown that symptoms improve at 1-year follow-up in 70% of patients and more than 33% no longer meet criteria for a tilt-table test.[19] Nonpharmacologic interventions such as lifestyle modifications, physical therapy, and use of compression garments can be effective strategies in reducing symptoms and improving deconditioning. Pharmacologic interventions, including medications that influence blood volume, peripheral resistance, and heart rate, may be necessary for some patients. The long-term prognosis for patients with POTS generally is good, and most patients can maintain their quality of life with appropriate management. Some patients may experience relapses or fluctuation in symptoms that may require adjusting the treatment plan.

Although patients with POTS are at no increased mortality risk because of the syndrome, they should be closely monitored for potential complications associated with POTS, such as syncope, falls, and functional impairment. Clinicians should address any underlying patient comorbidities that may contribute to the development or worsening of POTS.

Close monitoring and ongoing follow-up are important to ensure optimal outcomes for patients with this condition.

## CONCLUSION

Recognizing the various interventions available for patients with POTS is essential for managing this condition effectively. Patient education and empowerment also are crucial. By taking a comprehensive and patient-centered approach to the management of POTS, PCPs can help to improve outcomes and enhance quality of life for patients with this condition. Ongoing education and training in the diagnosis and management of POTS also can help PCPs stay up to date with the latest advances in this field and provide the best possible care to their patients. **JAAPA**

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