

Postural Orthostatic Tachycardia Syndrome (POTS)

Author: Frank Gargano DPT, OCS

The clinical presentation of a patient with Postural Orthostatic Tachycardia Syndrome (POTS) can not only be challenging but at times frustrating. For Physical Therapists POTS is a relatively new diagnosis. It is commonly diagnosed in conjunction with other pathology such as: Migraine, cervical pain, dizziness, imbalance/dysequilibrium and Chronic Fatigue Syndrome (CFS). There are several reports suggesting that there may be a great deal of overlap between POTS and CFS.¹ The purpose of the paper is to introduce the pathophysiology of the condition and basic management principles.

To better understand the pathophysiology of POTS, a brief discussion of the Autonomic Nervous System (ANS) is necessary. The ANS is divided into three subsystems: the Sympathetic Nervous System (SNS), the Parasympathetic Nervous System (PNS), and the Enteric Nervous System (ENS). The SNS is active for emergency situations such as the “fight or flight” response. The SNS is responsible for producing bodily responses such as pupil dilation, increase in heart rate, reduction in digestive function and increase in conversion of glycogen to glucose. The PNS is involved in non-emergency situations that allow us to “rest and digest.” The PNS produces bodily responses such as pupil constriction, reduction in heart rate, increased saliva production and digestion. The ENS assists in control of the viscera (gastrointestinal tract, pancreas, and gallbladder.)

POTS by definition is the failure of the peripheral vascular system to appropriately vasoconstrict under orthostatic stress. The body then attempts to compensate for this by increasing the heart rate (tachycardia).² The increase in heart rate is clinically defined as: a heart rate increase of 30 beats per minute (bpm) or more from supine to the standing position within ten minutes or less. Several authors believe that POTS represents the earliest sign of autonomic dysfunction and that some patients (approximately 10%) later progress into having autonomic system failure.³ Many of these patients also can be misdiagnosed as suffering from Chronic Fatigue Syndrome. There are several reports suggesting that there may be a great deal of overlap between these two disorders.³

There are many suspected causes of POTS⁴ such as:

- Viral and/or bacterial infections
- Changes in blood volume (hypovolemia)
- Exposure to toxic chemicals
- Genetically inherited
- Damage to the vagus nerve (cranial nerve ten) or spinal cord
- Spinal canal stenosis particularly in the upper cervical spine
- Diabetes
- Alcoholism
- Chiari I malformation

The hallmark of this syndrome is persistent/recurrent tachycardia while upright. This can be associated with severe fatigue, near syncope or syncope, exercise intolerance, lightheadedness or dizziness.⁴ Patients may complain of always feeling cold, while at

the same time they are unable to tolerate extreme heat. Stomach discomfort is common because digestion can be effected. When the brain does not receive adequate blood circulation it will shunt blood away from the stomach creating digestive difficulties. Shortness of breath, memory disturbances, blurred vision, lower extremity paresthesia, sweating and vertiginous episodes when transitioning from lying to standing and eye pain with a feeling of pressure behind the eyes.

Another important characteristic of POTS is low blood pressure (although this is not a requirement, some patients can have hypertension). The presence of low blood pressure does not impact intelligence, but does impact a person's ability to think clearly and concentrate. The effect on concentration is usually intermittent and brief and is related to the amount of blood getting to the brain. The reason that people have dramatic changes in their heart rate is their body is attempting to compensate for the drop in their blood pressure by increasing the heart rate. The increased stress on the heart is one of the reasons why POTS patients complain of significant fatigue and a general feeling of being "washed out." The severity of a patient's symptoms will vary. Some patients with POTS can feel perfectly normal and have few symptoms.⁵ At other times symptoms can be so severe patients are forced to lie down for the entire day unable to function. POTS is a chronic disease that often waxes and wanes but is always present to some extent on a daily basis.

The pathophysiology of POTS can be described in simpler terms. Consider the human body as a fluid filled bag, when lying supine the fluid is evenly distributed throughout. Upon rising from supine to standing there is a gravity induced downward displacement of approximately 25-30% of the blood volume (fluid) to the abdomen and dependent extremities.⁶ Up to 50% of this displacement occurs within the first few seconds of standing.⁶ This rapid redistribution of central blood volume (fluid) reduces the venous return to the heart (cardiac filling pressure). The heart can not pump what it does not receive, so the stroke volume (amount of blood the heart pumps) declines approximately 40%.⁷ The result is a reduced amount of blood volume to the brain and production of the numerous symptoms previously listed. The SNS attempts to respond immediately to these changes by:

1. Increasing the heart rate (tachycardia)
2. Increasing the force of the heart's contractions.
3. Dilation of blood vessels in the lower extremities.

The medical management of POTS is pharmacologically designed medication intended to regulate blood pressure and normalize blood volume. Diagnostics such as head upright tilt (HUT) table testing may be performed prior to prescribing these medications. The HUT is a passive orthostatic stress test used to evoke autonomic reflexes and vascular responses. The patient is secured supine on a table that slowly elevates to an angle between 60-90 degrees from horizontal for 10-45 minutes. The physiological response to this positional change is closely monitored. Clinically, the HUT table test is not particularly accurate due to a significant number of false positives (symptoms produced during the test but are not true POTS) produced with this test.⁸

There are a number of non-pharmacological treatments that have low risk and considerable potential benefits. Increasing salt intake is often the safest first method to try to improve orthostatic tolerance. (*A high salt diet should only be tried under the recommendation and supervision of a Physician*). Adding extra salt to your diet will increase your blood volume and blood pressure by increasing fluid retention (benefits are variable and may only be transient). Patients suffering from POTS often have a measurably reduced blood volume (hypovolemia). Increasing fluid intake is necessary for the salt to expand the blood volume. This usually occurs spontaneously as salt increases thirst. The use of supportive stockings may help reduce the pooling of blood into the lower extremities while standing. Increasing lower extremity muscle tone has a similar effect on reducing blood pooling and venous dilation. A patient with severe symptoms may find any amount of exercise difficult however; slow progressive increases over time can improve exercise tolerance. A Physical Therapist can design a program to meet the specific needs of this patient population.

The following suggestions/tips may assist in managing symptoms related to POTS:

1. Avoid eating heavy meals. Overloading the stomach decreases orthostatic tolerance by drawing blood to the digestive tract and away from the main arteries that feed the brain.
2. During the day it is better to rest in a recliner chair rather than lying flat in bed since. Constant bedrest decreases your orthostatic tolerance. An example of this can be seen in the astronauts. Astronauts often develop a temporary form of orthostatic tachycardia (rapid heart rate) upon returning to earth due to the deconditioning effect of weightlessness, which is quite similar to prolonged bedrest. Exercising can be done even when lying in bed by isometrically contracting, and then relaxing the muscles in the arms and legs. It is very difficult to regain muscle mass once it is lost therefore, it is very important to avoid becoming deconditioned.
3. Avoid working with your arms over your head, lifting heavy objects, and climbing stairs. Take frequent rest breaks and/or ask for assistance.
4. Warm temperatures can have a negative effect on their exercise tolerance. Heat dilates blood vessels and diverts blood to the skin, thus reducing blood flow in key arteries that feed the brain. Air conditioning in warm weather is essential, especially when exercising.

Many POTS patients have little success and considerable frustration in attempting to get relief for their symptoms. Dr. Blair Grubb⁹ from the Medical College of Ohio has an interesting way of explaining the frustration of treatment for this condition. “Think of your brain like the thermostat in your home, once it is set, it automatically maintains that temperature and you no longer have to think about it. But if the furnace was not putting out the heat needed to keep the home at that set temperature you would think something was wrong with the furnace because all you know is that you are cold. If the repairman came in and checked your furnace he would find that it is working normally. The reality is the repair person (or medical specialist) has to understand the heating system well enough to know to also check the “thermostat” (control center). Most importantly he

would then have to know how to fix the “thermostat”. This is an interesting analogy because most physicians just want to try to fix the furnace; that is all they have been trained to do. They may never give much thought to the “thermostat” (control center). It can be very difficult to find healthcare providers trained to understand and manage the whole problem. This is why many patients with POTS are frustrated and never truly experience a therapeutic resolution.

POTS is an entity of varied etiologies and until a better understanding of the pathophysiology is achieved, treatments are essentially empirical. Fortunately, ongoing research is being conducted and treatment protocols continue to develop.

References

1. Bou-Holaigh I, Rowe P, Kan J, Calkins H. The relationship between neurally mediated hypotension and chronic fatigue syndrome. *JAMA*. 1995; 274: 961-967.
2. Hoeldtke RD, Danis KM. The orthostatic tachycardia syndrome: Evaluation of autonomic function. *J Clin Endocrinol Metab*. 1991; 73:132-139.
3. Schondorf R, Low P. Idiopathic postural orthostatic tachycardia syndrome: An attenuated form of postural orthostatic tachycardia syndrome? *Neurology*. 1993; 43:132-137.
4. Low P, Opfer-Gehrking T, Textor S, et al. Postural Tachycardia syndrome (POTS). *Neurology* 1995; 45:519-525.
5. Steward J. Orthostatic Intolerance: An Overview. *Emedicine*. (www.emedicine.com/ped/topic2860.htm). 2003; 1-18.
6. Shepherd JT, Sheperd RFJ. Control of the blood pressure and circulation in man in Bannister R, Mathias C (eds). *Autonomic Failure: A textbook of Clinical Disorders of the Autonomic Nervous System*. Oxford Medical Publishers, Oxford UK 1992; 78-93.
7. Hainsworth R. Physiology and pathophysiology of syncope in Kenny R (ed). *Syncope in the older patient*. Chapman and Hall Medical Publishers. London UK 1996, pp 15-31.
8. Leonelli FM, Wang K, Evans JM: False positive head-up tilt: hemodynamic and neurohumoral profile. *J Am Coll Cardiol* 2000 Jan; 35(1): 188-93 {Medline}.
9. Grubb BP. Associate Professor of Medicine and Pediatrics; Division of Cardiology and Neurology. The Medical College of Ohio.