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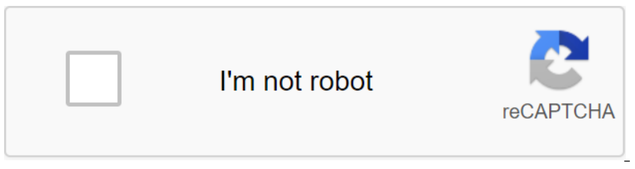

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March 12, 2019 Thomas K. Crawford, MD, FACC Authors: Bryarly M, Phillips LT, Fu, Vernon S, Levine BD. Citation: Postural Orthostatic Tachycardia Syndrome: JACC Focus Workshop. J Am Coll Cardiol 2019;73:1207-1228. Below are the key points to remember from this review article on postural orthostatic tachycardia syndrome (POTS): POTS is an orthostatic intolerance syndrome characterized by an increase in heart rate ≥30 bpm, often with a standing ambulance of 120 bpm, within 10 minutes of standing or tilting the head upwards, and in the absence of orthostatic hypotension (reducing systolic blood pressure (BP) ≥20 mmHghg and/or reducing diastolic BP ≥10 mmhg). It is the most common form of orthostatic intolerance in young people (mostly women in premenopausal). Presyncop is much more common than syncope in POTS, but it is not uncommon that POTS coexist with episodes of nervously mediated (reflex) syncope. Cardiovascular decondation is a universal feature in all POTS. Overlapping pathophysiological variants that may contribute to a person's susceptibility to the development of POTS include peripheral vegetative neuropathy, excessive venous association, hypovolemia in volume dysregulation, hyperaderergic conditions, mast cell activation disorders and autoimmune diseases. Chronic symptoms and comorbidities that cannot be physiologically explained by orthostatic intolerance or tachycardia, but common in patients with POTS include chronic fatigue, dizziness, syncop, migraine, functional gastrointestinal disorders, chronic nausea, fibromyalgia, and joint hypermobility. POTS should be distinguished from neurogenic orthostatic hypotension (NOH), which can occur in disorders such as multiple system atrophy, Parkinson's disease, Levi's body dementia, pure vegetative insufficiency, autoimmune vegetative ganglionoption and other vegetative neuropathy. In orthostatic hypotension, there should be at least a 20-point drop in systolic BP for 3 minutes of inclination and usually the increase in heart rate is minimal. Inappropriate sinus tachycardia (IST) is sometimes confused with POTS, especially because both occur in young women, but IST occurs regardless of body position. The key to IST diagnosis is outpatient monitoring; IST patients, unlike patients with POTS, exhibit a relative night tachycardia on the back. Formal testing of vegetative function is useful to evaluate for other types of vegetative disorders and to differentiate among POTS subtypes. Non-pharmacological treatment is the basis of therapy for POTS and includes: Exercise conditioning with a recumbent bike, rowing machine, or swimming. This approach allows exercise while avoiding the upright position and improves tolerance to the program. (Medications can be considered in patients with severe symptoms like bridge to help minimize some symptoms and allow patients to patients initiate an exercise program. Such drugs include propranolol, midodrin, pyridostigmin, fluodroncortisone.) An increase in blood volume can be achieved by drinking 3 liters of water per day and liberalizing salt intake by taking 5-10 grams of sodium per day. Avoiding large and heavy meals, alcohol and heat exposure. Wearing compression stockings up to the top of the thighs or above and abdominal binders, and they should spread at least at the top of the thighs and preferably into the abdomen. Sleeping with the head of the bed elevated and performing physical counter maneuvers such as crossing the legs and squats. Behavioral and cognitive therapy can be used to obtain long-term symptom control, especially when anxiety, hyperviolence, or catastrophic behavior are present. Clinical topics: Arrhythmia and clinical EP, Diabetes and cardiometabolic diseases, prevention, sports and exercise cardiology, vascular medicine, implantable devices, SCD / Ventricular arrhythmia, Atrial fibrillation / Supraventricular arrhythmias, exercise, sleep apnea Keywords: Arrhythmia, cardiovascular, autoimmune, cardiovascular hypotonia, orthostatic, hypovolemia, body disease Levi, Midodrin, Migraine Disorders, Monitoring, Ambulatory, Multiple atrophy system, orthostatic intolerance, Parkinson's Disease, Postural Orthostatic Tachycardia Syndrome, Protransolol, Secondary Prevention, Swimming, Syncope, Tachycardia, Sinus, Zolth; Return to the lists of Olshansky B, Cannom D, Fedorovsky A, Stuart J, Gibbons C, Sutton R, Shen WK, Maldowney J, Chung TH, Thung, Th. Olshansky B, et al. Prog Cardiovask Dis. 2020 May-June;63(3):263-270. doi: 10.1016/j.pcad.2020.03.010. Epub 2020 March 25. Prog Cardiowask Dis. 2020. PMID: 32222376 Review. Editor's note: Please note that this author will consider using non-label in the next article. Diagnostic Criteria - The general clinical features of POTS POTS are defined as the presence of chronic symptoms of orthostatic intolerance (≥6 months) accompanied by an increase in heart rate (HR) ≥30 bpm within 10 minutes of taking vertical posture (Figure 1) and in the absence of orthostatic hypotension (blood pressure (BP) in the fall of the 20/10 mm mouth). a higher threshold (≥40 bpm) should be used as they have a greater physiological orthostatic tachycardia.3 Morning postural assessments optimize diagnostic sensitivity (due to specificity) for POTS.4 Orthostatic tachycardia should occur in the absence of other clear causes of orthostatic tachycardia (e.g., orthostatic tachycardia) (e.g., orthostatic tachycardia) (e.g. acute blood loss), medications that impair autonomic regulation, or other chronic debilitating disorders that can cause tachycardia (e.g.. . diabetes with known vegetative neuropathy, systemic infectious or inflammatory conditions, hyperthyroidism). Often there are both cardiac symptoms (rapid heartbeat, lightheadedness, shortness of breath and discomfort in the chest), and not cardiovascular symptoms (headache (often migraine), tremration, nausea, difficulty sleeping,5 mental clouding (probably due to reduced attention and not necessarily memory problems), intolerance of exercise and chronic fatigue)5. Activities of everyday life, such as bathing or housework, can significantly exacerbate symptoms and, as a result, fatigue. Breast pain syndrome is rarely associated with epicardial coronary artery obstruction, but may be associated with low lead electrocardiographic changes, especially when upright.7 While before syncopation and lightheaded are universal in these patients, only a minority of patients experience frank syncope. The vast majority of patients with POTS are women (80-85%) and women (80-85%). Orthostatic tolerance also decreases in healthy women10, which varies depending on the menstrual cycle,11 especially in patients with POTS.12 Patients often report that their symptoms started after acute stressors (e.g. suspected viral disease, major operations, injuries or pregnancies), but symptoms can also develop more insidious. Acutely, patients are often bedridden during a variable period of time, which can quickly cause hypovolemia and cardiac atrophy at a rate of 1%/week,13.14 the physiological effects of bed treatment induced orthostatic intolerance,15 similar to that 16 Even 20 hours of decondation of bed can cause vertical tachycardia and orthostatic intolerance in previously energetically active people17, which can lead to a downward spiral of orthostatic intolerance, and further decoding of bedlessness. Ultimately, regardless of the cause of deposition, in a chronic condition, the physiology of cardiovascular decondation can dominate the clinical picture, making a significant contribution to nobility and disability. Numerous studies5.18 recorded poor quality of life of patients with POTS associated with health, with scores comparable to those in patients with congestive heart failure. Many patients have bowel disorders and have been jointly diagnosed with irritable bowel syndrome, and some have abnormalities.19 About 80% of patients report worsening symptoms around menstruation.20 Patients with POTS can often seem anxious in the clinic. However, misinterpretation of physical symptoms such as tachycardia and may explain some of this apparent anxiety. In a formal assessment, POTS patients did not have a higher incidence of underlying depressive disorders, anxiety disorders, or substance abuse than the general population.6 Using the anxiety sensitivity index, it was to less anxiety in POTS patients than the general population,6 and the heights in POTS correspond to blood-eaters in the lower extremities rather than to anticipate anxiety.21 The POTS Study Assessing Patient Evaluation with POTS begins with a detailed history and physical examination looking for commonalities outlined above. Phehromocytoma may mimic POTS (or vice versa) due to paroxysms of hyper-adrenergic symptoms, including rapid heartbeat, although patients with pheohromocytoma are more likely to have these symptoms while on the back than POTS patients. Plasma or urinary methanephrine22 can screen for pheohromocytoma. Conventional CBC and electrolyte panel can eliminate severe anemia or gross electrolyte disturbances. Tachycardia in POTS patients should come from the sinus node, and should develop and be resolved relatively gradually with changes in posture. The electrocardiogram should be performed regularly to eliminate the presence of in-wheel-drive bypass surgery or other cardiac conduction disorders. If the patient describes paroxysmal tachycardia with sudden onset and displacement, especially in a position on the back or sitting, then a Holter monitor or event recorder may be required to rule out a new tachycardia. It is important that monitoring continues as soon as possible to capture clinically significant events. The function of the left ventricle should be normal for the diagnosis of POTS. Cardiomyopathy (e.g. peripartum) can mimic the presentation of POTS. Other tests may be reserved for referral centers. With formal vegetative nervous system testing, POTS patients often retained the Vagal function and the energetic pressor response to the Valsalva maneuver, with an exaggerated drop in blood pressure, Recovery and excess of both before and after release.23 Vertical plasma noradrenaline (after at least 5-10 minutes of standing or tilting) is often elevated (600 pg/ml) in POTS patients that is often present in formal cardiopulmonary exercise testing can be useful for objective documentation of exercise capabilities, as well as for consistent quantitative evaluation of functionality over time. Since blood volume is low in many patients with POTS,24.25 official evaluation with nuclear medicine trials can help focus the treatment plan. Treatment of POTS Treatment Efforts should begin with correcting reversible causes and optimizing chronic disease management. Educating patients is important. If there has been an attack of prolonged bed rest, symptoms should gradually improve as patients regain themselves vertically posture. POTS patients should avoid aggravating factors such as dehydration and extreme heat. In order to optimize hydration, we are asked by patients to consume 8-10 glasses of water per day and increase their sodium intake to 8-10 g/day. In the it has to be by dietary modification. We recommend panties hose (waist high) style compression stockings with 30-40 mm Hg. counter pressure to minimize peripheral venous association and to increase venous return. Raising the head of the bed on blocks of 4-6 inches may also be useful for facilitating the expansion of plasma volume.26 Radiofrequency ablation may be required to treat reantent supraventricular tachyarrhythmia, but radiofrequency modification of sinus nodes for sinus tachycardia IS is not recommended, as it often makes the patient's symptoms worse (and sometimes). Exercise Patients with POTS have a small mass of the left ventricle (LV), LV end diastolic volume, and low vertical stroke volume compared to normal sex control (Figure 2);25.27 plasma volume and total blood volume are also low. Taken together, these cardiovascular characteristics are similar to those seen after bed restile,13.14 and are the opposite of what is observed in athletes28. high vertical HR is proportional to this low vertical SV assuming that orthostatic tachycardia is a normal vegetative reaction to the hemodynamics of vertical posture25, As seen in astronauts.29 When this cardiovascular deconduction from bed rest is prevented by flexible or semi-tracing exercises combined with a replete volume, Orthostatic intolerance is completely preventable.30,31 So exercise is a sensible first-line therapy for many patients with POTS. Exercise has long been recommended in general for POTS patients. Unfortunately, most POTS patients may not be able to tolerate vertical exercises like a treadmill or elliptical machine, and report feeling weakened for a few days after stress, limiting adherence to their exercise regimen. Anecdote, patients who did the exercise seemed to have a better long-term prognosis, but he wasn't sure if it was due to exercising themselves or because of the selection bias based on their ability to exercise. Fu et al.25 recently introduced a structured three-month exercise program for 19 patients with POTS. It was a structured program that included primarily aerobic cardiovascular workouts, but also some resistance workouts involving primarily leg muscles. The exercise program was detailed with individual training calendars designed for each patient and, where possible, it was done in a controlled environment. A key feature of this program was that patients were initially advised to perform all exercises in a sitting position, which is disassociated from exercise-induced tachycardia from the severity of induced tachycardia, which is in these patients. Recommended exercises included the use of a rowing machine (which causes the most energetic cardiac hypertrophy among all sports32 due to its unique combination of static and dynamic exercise;33 recumbent cycling or swimming are also effective. intervention in exercise reduced orthostatic tachycardia and improved quality of life despite a relatively short duration. Physiological parameters such as blood volume, stroke volume and MASS LV have all improved over the course of 3 months, both to show tolerance, and the hemodynamic response to exercise.34 This study elegantly showed that exercise is an important intervention in this population, not just the ability to exercise. Fu's team is nearing completion of a much larger international registry of 250 patients for whom intervention has been applied in the community instead of a carefully monitored study. Preliminary results have been presented recently and are very encouraging with the 73% treatment rate meaning no longer meet the criteria for POTS after intervention.35 Pharmacological Treatment POTS Initial Pharmacological Approach is to remove medications that may be predisposing to tachycardia (e.g. diuretics, vasodilators, and norepinenaline transporter blockers). Some oral contraceptives include drosperinone as progestin, which is an analogue of spironolal. The use of pharmacological agents should not be seen as a replacement for the exercise program, but as an addition to the exercise program. Beta-adrenergic blockers are commonly used in cardiac clinics to control tachycardia, but tolerance can be a problem in many patients with POTS. While reducing HR in POTS would be helpful if tachycardia was excessively compensated for physiological stimuli (i.e. really hyperdynamic circulation), but could be counterproductive if the HR increase in POTS were purely compensatory (e.g. low stroke). We found that a low dose of propranolol (10-20 mg PO TID-SID) was very effective in reducing standing HR and improving symptoms acutely in POTS,36 patients while a more complete beta blockade was less well tolerated.36 Long-acting propranolol in a chronic environment was just as effective, as an exercise in reducing standing HR, but not improving the quality of life in patients POTS.37 Not a selective beta blocker as propranolol can be more effective than a selective beta blocker like metoprolol, since it will also block beta-2 adrenoreceptor mediated vasodilation. Patients who have hypovolemia are either known or highly suspected, fludrocortisone (similar to aldosterone) is often used. Thanks to increased sodium retention, it should expand the volume of plasma, although clinical data is lacking. Adverse effects may include hypokalemia (which can be profound, especially when combined with the load of Naz), worsening headaches, acne, and fluid retention with swelling. Other medications for POTS include midodrine, pyridostigmin and central sympatolitics. Midodrin is a peripheral alpha-1 adrenargic agonist who is a vasoconstrictor and venoconstrator. Midodrin can cause balding scalp, goose or headaches that may limit his tolerance. Pyristosigmine is a peripheral acetylcholinesterase inhibitor that can increase levels of synaptic acetylcholine in both vegetative ganglia and peripheral muscaritic parasympathetic receptors. Pyridosminetig holds back HR significantly in response to standing in POTS patients,38 and 30-60 mg of PO TID has led to chronic improvement in symptoms in 50% of POTS.39 Pyridostigmine patients can increase intestinal mobility, and this can lead to discontinuation of the drug in 20%.39 Central sympatholic agents can be beneficial to patients with very hyperadrergic with their POTS. Clonidine is an alpha-2 adrenergic agonist that acts centrally to reduce the sympathetic nervous system churn. Clonidine 0.1-0.2 mg PO BID-TID (eventually switched to a long patch) can stabilize HR and BP, although α-methyldop 125-250 mg PO BID (false neurotransmitter) may be better tolerated due to its longer periods of semi-seed. Unfortunately, both drugs can cause drowsiness, fatigue and worsen the mental clouding of some patients.40 Pots findings can lead to significant disability among previously healthy people. Patients with POTS show an increase in HR at ≥30 bpm (≥40 bpm in children) for 10 minutes standing up, often hyper-adrenergic, and quite symptomatic. Many patients suffer from low upright stroke, and in a chronic condition, disability can be dominated by the decondation of the phenotype. The focus of therapy should be a recovery exercise program, including both aerobic and resistance training, with a focus on non-vertical exercises such as rowing machines, recumbent cycles and swimming. Pharmacological therapy targeting hypovolemia and excess sympathetic activation of the nervous system can help relieve symptoms. Links Schondorf R, Low PA. Idiopathic postural orthostatic tachycardia syndrome: a faded form of acute pandizaautoma? Neurology 1993;43:132-137. Raj SR. Postural tachycardia syndrome (POTS): pathophysiology, diagnosis and management. Indian Pacing Electrophysiol J 2006;6:84-99. Singer W, Sletten DM, Opfer-Gehrking TL, Brands CK, Fisher PR, Low PA. 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