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Postural Orthostatic Tachycardia Syndrome (POTS) Education, Awareness, and Support

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## Chapter 1 INTRODUCTION

### **Description of Problem**

Over the past two decades, dysautonomia, or conditions marked by dysfunction of the autonomic nervous system, have been gaining more attention (Grubb, 2008; Grubb, Kanjwal, & Kosinski, 2006). Postural orthostatic tachycardia syndrome (POTS) is one form of dysautonomia that has been gaining such attention. Differing opinions on exact diagnostic criteria and other features of the syndrome exist in the medical community, making the need for further research more evident (Abed, Ball, & Wang, 2012). Because of the incomplete understanding of POTS in the medical community and the complex and heterogeneous nature of the syndrome, POTS patients must advocate for themselves and continue to fight against dysautonomia. Therefore, there is a need for increased dysautonomia, and specifically POTS, education, awareness, and support for the patient population, loved ones of those who live with POTS, healthcare providers, and the general population.

### **Background**

Several authors have estimated that approximately 500,000 to 1,000,000 individuals in the United States, alone, have POTS, however there are varying estimates including statements of the prevalence being unknown (Low, Sandroni, Joyner, & Shen, 2009; Abed, Ball, & Wang, 2012; Grubb, 2008). Women of childbearing age, between approximately 13 and 50 years old, are affected more often than men (Abed et al., 2012; Benarroch, 2012; Raj & Levine, 2013). The female-to-male ratio is about 5:1 (Abed et al., 2012; Benarroch, 2012).

As previously stated, POTS is one type of dysautonomia. The autonomic nervous system is responsible for the involuntary functions in the body such as constriction and dilation of blood

vessels as well as heart beat (ANS Disorders, n.d.). POTS is marked by an inappropriate and inadequate autonomic response to changes in posture from supine to standing.

When an individual with an appropriate and healthy autonomic nervous system stands up, gravity causes a shift in the blood volume toward the lower body resulting in a slight increase in heart rate to pump the blood back up to the heart and other organs (Abed et al., 2012). The fully functional autonomic system compensates very quickly for the change in posture and heart rate remains stable with only minimal changes. The individual remains unconscious of the compensatory activity occurring and continues feeling well as homeostasis and balance are maintained.

Individuals with POTS, on the other hand, have a dysfunctional autonomic nervous system that does not react properly. When individuals with POTS stand up gravity pulls much of the blood volume into the legs and lower body and away from the vital organs such as the heart and brain (Grubb, 2008). Since these individuals do not have an autonomic nervous system that is working properly an imbalance occurs as the system attempts to compensate for this shift in blood volume by significantly increasing the heart rate to try and keep the vital organs perfused. As the body senses this decrease in blood flow to vital organs, signs and symptoms suggestive of hypoperfusion occur, such as lightheadedness, syncope or near syncope, tachycardia, palpitations, nausea, and diminished concentration. Once the individual sits or lies down and orthostatic stress is removed, or at least diminished, symptoms usually improve. It is important to note that many POTS patients report other varying symptoms that are not related to orthostatic stress which cause a more complex and difficult clinical picture and often affect diagnosis and management (Benarroch, 2012).

### **Categorization of POTS**

Researchers and authors have proposed several ways to classify and describe POTS by sorting cases into subtypes or groups. Because there is not yet one single consistent method of identifying subtypes, and many patients present with features of more than one “type”, it seems best to simply describe some of the common features that exist within each group and leave some classification open to self-interpretation. There are cases, however, in which even the categorization that is described here is not entirely consistent. That will be discussed briefly later in this paper. The bottom line is that continued research is needed on POTS patients in order to gain a deeper understanding of the condition and the relationships between the many variables involved and in order to establish more standardized concepts and definitions.

**Primary.** POTS can be categorized as being either a primary or secondary form of the condition (Grubb, 2008; Grubb et al., 2006). The primary form of POTS is not associated with any other known disease but instead due to a dysautonomic process, whereas the secondary form of POTS is secondary to some other known underlying illness or disease and results in the syndrome of signs and symptoms characteristic of POTS. The most common primary form of POTS is due to a type of peripheral autonomic neuropathy causing the vessels in the periphery not to constrict appropriately in response to orthostatic stress (Grubb, 2008; Grubb et al., 2006; Benarroch, 2012). This neuropathic, or partial dysautonomic, process is the most common process found and often has an abrupt onset after a viral illness or some other precipitating event such as pregnancy, surgery, trauma, sepsis, immunizations, or some other traumatic or stressful event (Conner, Sheikh, & Grubb, 2012; Grubb et al., 2006). At the current level of understanding and research, the neuropathic partial dysautonomic process causing POTS is thought to be caused by an immune-mediated response.

Another group of patients who are thought to have a partial dysautonomia process responsible for POTS is a developmentally presenting group. These cases are adolescents who develop POTS during the rapid growth of the teen years. It is thought to be caused by a temporary imbalance in the autonomic system due to the rapid adolescent changes (Conner et al., 2012; Grubb et al., 2006). A majority, around 80%, of these developmentally based cases seem to outgrow the syndrome and make a full or near-full recovery by young adulthood, usually around the 19-24 year old range.

A rarer primary form is the hyperadrenergic POTS. In the truest and purest sense of the term, hyperadrenergic POTS is a much less common form accounting for only about 10% of cases (Raj, 2006). These patients experience an overabundance of sympathetic activity and norepinephrine levels  $>600$  pg/ml, often even  $>1000-2000$  pg/ml (Raj, 2006; Grubb et al., 2006; Conner et al., 2012; Grubb, 2008; Benarroch, 2012). Patients often develop the hyperadrenergic form gradually and progressively, and it is thought to be caused by a genetic mutation causing an inappropriate overabundance of norepinephrine due to dysfunction of the reuptake transporter protein. The body is unable to clear that norepinephrine from the intrasynaptic cleft leaving the patient with elevated serum catecholamine levels and symptoms of excessive sympathetic activity.

Many POTS patients experience multiple pathophysiological processes and may fit the criteria for multiple processes. For instance, a patient may fit the neuropathic criteria but also have a standing norepinephrine level  $>600$ , or even greater than 1,000, but without known evidence of the genetic mutation. Many POTS patients do experience elevated norepinephrine levels, and some authors and researchers even argue that standing norepinephrine levels must be  $>600$  pg/ml for any diagnosis of POTS. However, other authors believe that this finding is

consistent only in hyperadrenergic POTS (Conner et al., 2012; Grubb, 2008; Benarroch, 2012; Grubb, 2006). Thus, what one physician might call a hyperadrenergic case of POTS, another might simply call a neuropathic case.

**Secondary.** Patients that have the secondary form of POTS have some other underlying illness or disease, such as diabetes, Sjogren's syndrome, or Lupus (Grubb et al., 2006). Chronic diabetes mellitus, for instance, can cause peripheral neuropathy resulting in improper or absent peripheral autonomic function, causing loss of proper peripheral vasoconstriction in response to orthostatic stress, thus causing the signs and symptoms.

Mast cell activation disorders such as mast cell activation syndrome (MCAS) have also been linked to POTS, most specifically to a secondary form of hyperadrenergic POTS (Benarroch, 2012). Mast cell disorders are either proliferative (too many mast cells accumulating in tissue or organs, as in mastocytosis) or activation based (the correct number, but overactive mast cells, as in MCAS) (Shibao et al., 2005). The histamine and other mast cell mediators are released in response to orthostatic or other stressors, causing the vessels to dilate and peripheral blood to pool, as in other types of POTS. In addition, the body then experiences an increased sympathetic discharge. There has been discussion on whether the mast cell response initiates the increase in sympathetic activity or whether the increase in sympathetic activity initiates the mast cell response. Either way, the end result is the same: POTS symptoms in addition to flushing episodes, marked with increased urine methylhistamine levels, and other symptoms such as shortness of breath, nausea and vomiting, diarrhea, excessive diuresis, headache, and lightheadedness.

### **Purpose**

As already discussed, several pathological processes may be coexistent, and thus it is very difficult to describe universal comprehensive subtypes. This confusion, lack of consistent phrasing and description, and need for further research has caused a mixed response from the medical and patient communities. Many medical providers are unknowledgeable about POTS, not to mention proper treatment and discussion of various “types”. Patients are left frustrated, confused, and feeling hopeless. Patients are often misdiagnosed as having anxiety or panic disorders (Grubb et al., 2006).

Through personal experience with POTS, this author has become aware of the need for further awareness, education, and support for patients, families, and healthcare providers dealing with POTS. The remainder of this paper will discuss current research literature and describe the project which is aimed at educating, increasing awareness, and providing support for those impacted or affected, either personally or indirectly, by POTS.

## Chapter 2 LITERATURE REVIEW

### Research Literature

#### Sleep and Quality of Life

Bagai et al. (2011), of Vanderbilt University, assessed (a) health-related quality of life, (b) fatigue, and (c) sleep quality in POTS patients, in comparison to healthy control subjects, by using a series of scales and questionnaires. A total of 90 participants were included in the study: 44 POTS patients who met the criteria for POTS diagnosis and 46 healthy control subjects recruited through a research volunteer database and advertisements within the local campus community.

Several tools were utilized during data collection. Bagai et al. (2011) used the Medical Outcomes Study Sleep Scale comprised of 12 questions in order to assess difficulties within several areas of sleep: snoring, sleep adequacy, sleep disturbance, headache, somnolence, and respiratory impairment. Each area is scored on a scale of 0-100, with a higher score for sleep adequacy indicating improved and better sleep, and a higher score on any of the other areas indicating increased dysfunction. The Epworth Sleepiness Scale ranging from 0 to 3, with a total possible score of 24, assesses subjective daytime sleepiness. Eight common situations of daily living are presented and respondents must indicate the likeliness of sleeping in each situation. Responses >10 are usually seen in individuals with diagnosed sleep disorders affecting daytime sleepiness, whereas responses <10 are considered normal. The Fatigue Visual Analogue Scale is a one question assessment of fatigue severity and uses a scale of 0-10, the higher responses indicating higher levels of fatigue. The RAND-36 is a more general questionnaire assessing quality of life in 8 domains: “physical functioning, role limitations caused by physical health problems, bodily pain, general health, emotional well-being, role limitations due to emotional

problems, social functioning, and energy/fatigue” (Bagai et al., 2011, p. 205). A 0-100 scale is used, asking participants to rate in each area over the last four weeks. Poorer quality of life is indicated by lower scores. Lastly, the EuroQol was used to collect subjective rating for level of problem within 5 areas relating to health: mobility, anxiety/depression, usual activities, self-care, and pain/discomfort. Participants rated each as either “no problem, some/moderate problems, or extreme problems” but results “were analyzed as dichotomous variables (no problems vs. some/moderate/extreme problems)” (p. 205).

The Medical Outcomes Sleep Scale found significantly poorer sleep adequacy and increased sleep difficulties in POTS patients than were found in the control subjects (Bagai et al., 2011). The Epworth Sleepiness Scale found a significant difference between POTS patients and control subjects. Fifty-one percent of POTS patients scored >10, indicating excessive daytime sleepiness, in comparison with 16% of control subjects scoring above the same mark. The Fatigue Visual Analogue Scale found that a significantly higher number of POTS patients, in comparison to healthy controls, indicated fatigue being a major issue within the past seven days. The RAND-36 and EuroQol both indicated significantly poorer “health-related quality of life” in POTS patients than in the control subjects. The physical health scores were lower than were the mental health related scores (Bagai et al., 2011, p. 206).

Overall, Bagai et al. (2011) found that POTS patients experience more fatigue and sleep related difficulties than do healthy control subjects. Furthermore, these sleepiness and fatigue related difficulties are related to poorer quality of life. Interestingly, results showed that physical health was affected more than mental health, and because the control group scored worse in the snoring category, the authors have assumed that the self-reporting nature of the study, viewed as a possible limitation, did not in fact cause over-reporting of symptoms in the POTS group. Bagai

et al. (2011) recommend further studies on this topic and involvement of more formal and objective methods to further understand the findings.

### **Inattention and Psychiatric Disorders**

Vidya Raj et al. (2009) used a cross-sectional controlled method to assess anxiety, attention, and mood in POTS patients (N=21) compared to ADHD participants (N=18) and normal control subjects (N=20) with no history of psychiatric or attention disorders. Previously validated and commonly used questionnaires were used. POTS patients met the diagnostic criteria for POTS, were tested while in the sitting position, were not currently taking any medications affecting psychiatric or autonomic function, and were asymptomatic during testing. Comparisons were done between two matched groups: POTS patients with ADHD participants, and POTS patients with normal control subjects with no history of attention or psychiatric diagnoses. Significance was set at the  $P < 0.05$  level.

Results showed that POTS patients did not experience significantly higher rates of anxiety disorders in comparison to the normal control subjects or those with ADHD (Vidya Raj et al., 2009). Similarly, POTS patients did not show a greater “lifetime prevalence of major depressive disorder” in comparison with the normal control subjects (p. 4). The ADHD participants, however, did show higher incidence of depressive disorders than both the normal control group and the POTS patients. POTS patients also showed no significant differences in substance dependence and abuse in comparison to the normal control group.

Significant differences were found among the groups for scores on the second edition of the Beck Depression Inventory for degree of depression symptoms (Vidya Raj et al., 2009). Both POTS patients and ADHD participants scored higher in comparison with the control group for “magnitude of current depression symptoms” (p. 4). The difference between POTS and

ADHD scores, however, were not significant. The Beck Anxiety Inventory showed significantly higher scores in POTS patients in comparison with both the control subjects and those with ADHD. The Anxiety Sensitivity Index showed significantly higher fear of anxiety related symptoms in the ADHD and POTS groups in comparison with the normal controls, however in comparison with the general population, POTS patients actually scored lower and ADHD subjects still scored higher.

The Connors Adult ADHD Rating Scale showed that POTS patients experienced significantly higher scores in comparison with the control subjects in three areas: inattention/memory problems, ADHD score, and ADHD index (Raj Vidya et al., 2009). However, those participants with ADHD scored significantly higher than POTS patients and the control subjects in all of the subscales measured. The ADHD Behavior Checklist for Adults measured symptoms both in childhood and in the past six months. Significant results showed that POTS patients experienced less symptoms in childhood in comparison to those with ADHD but no significant difference in comparison to the control group. However, for symptoms within the past 6 months, POTS patients showed significantly greater symptoms of inattention and hyperactivity in comparison with the control group but still less than those with ADHD.

Overall, Vidya Raj et al. (2009) found POTS patients to have no increased likelihood of developing a depression or anxiety related disorder. However, POTS patients did display a score indicating mild depression. Vidya Raj et al. (2009) discussed the possibility that this finding could be due to the difficulties associated with living with chronic illness. It was noted, that the quality of life that POTS patients experience has been compared with other chronic diseases including heart failure and chronic obstructive pulmonary disease. Similarly, POTS patients did display scores indicating higher levels of anxiety on one scale, however the discussion on this

was that POTS patients experience physical symptoms that could be attributed to anxiety but instead is due to the pathology of the postural orthostatic tachycardia syndrome. Therefore, it was suggested that the anxiety symptoms are physically rather than psychologically based. Further studies using objective measures were recommended due to subjective nature of data collected and small sample size.

### **Diurnal Variability in Heart Rate and Blood Pressure**

Brewster et al. (2012) conducted a study to examine diurnal variability within two groups, a POTS group (N=54) and a healthy control group (N=26). Those participants in the POTS group had not taken any medications affecting hemodynamics for at least five half-lives of the medication. Fludrocortisone was discontinued, specifically, for at least five days prior to beginning the study. The healthy control group also did not take any medications affecting hemodynamics. Significance was identified as  $P < 0.05$ .

An automatic BP monitor was used to obtain orthostatic BP and HR measurements the night of admission to the research center and then again the next morning (Brewster et al., 2012). Orthostatic measurements in both instances were taken supine and then after standing for five minutes. Both POTS patients and healthy controls experienced significantly greater orthostatic tachycardia and higher standing heart rates in the morning than in the evening. Supine HR measurements were unchanged between the evening and morning in the POTS patients while the healthy controls had a slight decrease in supine HR for the morning check. The only significant difference in BP was a slight decline in SBP for both POTS and control subjects in the morning measurement in comparison with the evening.

Interestingly, a higher percentage of participants, both POTS and healthy controls, met the orthostatic HR criteria ( $\geq 30$  bpm) of POTS in the morning than did in the evening (Brewster

et al., 2012). This finding suggests that this phenomenon is a normal finding that is simply increased in POTS patients. Brewster et al. (2012) discussed that consideration should be given to standardizing time of day for data collection in diagnosing POTS. More patients could possibly be accurately diagnosed if measurement time was standardized to morning.

### **Propranolol for Symptom Improvement**

Satish Raj et al. (2009) conducted a study utilizing a randomized crossover design to examine the efficacy of propranolol versus placebo in improving symptom load in POTS patients. Two protocols were used, some patients being eligible to complete both protocols. In the 1<sup>st</sup> protocol (N=54), propranolol 20 mg was compared with placebo, given on separate days within a randomized single-blind fashion. The 2<sup>nd</sup> protocol (N=18) involved comparison of propranolol 20 mg versus 80 mg, again single-blind, randomized, and given on separate days. For both protocols, vital signs were monitored immediately before and hourly for four hours after administration of the study drug. Each vital sign measurement was taken with the patient seated and after standing for 10 minutes and was collected via a Dinamap that automatically and digitally input the measurements into the computer database. Nine symptoms were assessed immediately before study drug administration and after administration of drug at the two and four hour marks. Patients rated the severity of each of the nine symptoms on a scale of 0-10, with 0 being the absence of symptom. Significant results were defined as  $P < 0.05$ .

In protocol #1, significantly lower heart rate was found with propranolol 20 mg versus placebo at the one hour post drug administration for both the sitting and standing measurement and for the remainder of the four hour study period (Satish Raj et al., 2009). Similarly, the SBP was significantly lowered after propranolol 20 mg versus placebo at the one hour check for standing, and continuing for three hours, and at the two hour mark for sitting, and continuing for

the full four hours. The sitting and standing DBP were lowered as well. Overall, there was a significant change (decrease) in HR following administration of 20 mg propranolol versus placebo. In protocol #2, the standing heart rate and SBP were significantly lower with the 80 mg versus the 20 mg starting at the one hour check and continuing for the remainder of the four hour collection time. There was not any significant difference in sitting HR, SBP, or DBP readings between the 20 mg and 80 mg groups. In overall change for HR, SBP, or DBP, the only significant change was seen in HR decrease at the one hour check, with a greater change following 80 mg administration versus the 20 mg, and continuing for the full four hours.

Based on the symptom severity rating scores, propranolol 20 mg was found to decrease total symptom load in comparison with placebo (Satish Raj et al., 2009). Interestingly, the low dose propranolol (20 mg) was also found to decrease total symptom load, overall, more than the high dose (80 mg). This finding is significant because it implies that lower dose propranolol is more effective at decreasing both HR and symptom load versus a higher dose. Satish Raj et al. (2009) discussed the theory that POTS patients may need some elevation in HR in order to preserve optimal cardiac output and perfusion. It was suggested that the commonly reported failure of beta-blocker trial in POTS patients may be due to commonly overprescribed dose. There were some reported limitations. The principle investigator was blind to the study drug until after the data collection was finished, but the nurse administering drug and collecting data was not. Therefore, it was single-blind only. Satish Raj et al. (2009) stated, however, that this possible limitation was corrected by use of automatic collection devices that input data into the database directly. Furthermore, the symptoms were reported by the patient who was blind to study drug. Further research could be done with larger sample size for protocol #2 and a longer duration for follow-up after drug administration.

## Chapter 3 DISCUSSION OF PROJECT

### **Website**

My personal experience with POTS was my inspiration and motivation for developing this project. Having had POTS for over 10 years, and only recently getting progressively worse, I feel driven to help educate and build awareness in the public and help provide answers and support to other POTS patients. This is an ongoing project, meaning that content and layout may change and develop over the coming weeks and months. I am committed to keeping my website (lifegonetopots.com) active for a minimum of one year, at which time I will analyze the effectiveness and helpfulness it has proven and then reevaluate at that point. The following headings and content is what can be found currently on my website:

### **Home**

Dysautonomia is the medical word used to describe various conditions in which the autonomic nervous system malfunctions. The autonomic nervous system is the branch of the nervous system that controls all of the functions in the body that are not under our conscious control: digestion, sweating, heart rate, blood pressure, etc. Postural Orthostatic Tachycardia Syndrome (POTS) is one type of dysautonomia which affects heart rate and the body's ability to adapt when a person moves around and changes position. Many POTS patients experience other symptoms in addition to abnormally high heart rate when standing. Some patients have difficulty even completing routine activities of daily living. I have heard several POTS patients, including myself, make the comparison of living with POTS being like the waves of the ocean or a roller coaster. Each day is different and it is difficult not knowing what to expect day-to-day and even hour-to-hour or minute-to-minute. Whether you or someone you love has POTS, or you are a healthcare provider or other interested individual, please explore the rest of my site for

more information about POTS, helpful resources, and to take a glimpse into my own experience of living with POTS. I hope you will find this website helpful and encouraging!

### **All About POTS**

**Who, What, and Why.** This section currently has an abbreviated version of the information covered in Chapter 1. Because it was already covered previously in this paper, please see Appendix A for the full version that is currently displayed on the website. The expanded version found earlier in this paper may eventually be the content that is included on the website. There is often a fine line between providing deep enough information to be helpful and providing so much that it just muddles the understanding. That line will eventually be found on the website.

**Diagnosis.** Diagnosis of POTS is done either through a tilt table test or, at times, bedside orthostatic vital signs. The heart rate must increase at least 30 beats per minute within the first 10 minutes of standing after the patient changes position from lying to standing (Conner, Sheikh, & Grubb, 2012; Grubb et al., 2006; Grubb, 2008; Raj, 2006). Diagnosis is made on somewhat of an exclusionary basis, meaning that all factors must be considered before diagnosing POTS and it cannot be diagnosed on a single episode of orthostatic tachycardia. There must not be any acute and reversible cause of symptoms such as medications disrupting normal autonomic function, acute dehydration, or active bleeding (Raj, 2006). Also, it is important for clinicians to consider and rule out alternate diagnoses that could be to blame before diagnosing POTS. Twenty-four hour holter monitor, lab (blood) tests, echocardiogram, and other testing may be necessary. Symptoms must be present for greater than three months (Grubb, 2008). One other interesting point is that by considering so narrowly the heart rate in the diagnostic criteria, other

symptoms can be downplayed or forgotten (Grubb et al., 2006; Grubb, 2008). This can leave the patient frustrated and feeling like answers are lacking.

**Symptoms.** Symptoms include light-headedness, fatigue, tremor, exercise intolerance, syncope and near syncope, in addition to many more (Abed et al., 2012). Due to the number of symptoms and expanded reference list included on the website, as well as the layout used, please see Appendix B for this information in its entirety.

**Treatment/Management.** POTS is difficult to manage, since each patient lives a unique experience with POTS and needs an individualized plan of care. POTS patients need to increase water and salt intake, aiming for 8-10 cups per day as hydration maintenance alone (Raj, 2006). Caution should be taken in drinking too much water, however, since the imbalance can swing in the opposite direction causing electrolyte imbalance and possible heart rhythm problems (Abed et al., 2012). Healthcare providers can help monitor electrolyte levels and find the correct intake balance. Salt should be liberalized, making sure to get at least 2-4 g per day (Conner et al., 2012; Grubb et al., 2006). Some healthcare providers recommend even more. Salt tablets are available if dietary intake is insufficient.

Use of medication such propranolol, a beta blocker, is another good treatment option. Research has found low dose propranolol to be helpful in reducing HR and symptoms in POTS patients, but care should be taken since many patients experience hypovolemia and thus issues with hypotension (Satish Raj et al., 2009; Abed et al., 2012). Beta-blockers can worsen this problem and thus patients must be monitored for efficacy in improvement as well as exacerbation of signs and symptoms. Normal saline (0.9% sodium chloride) infusions are a useful treatment option for providing temporary relief to patients. This improvement is due to an acute load of sodium and increase in overall blood volume helping to alleviate some of the very

issues that cause POTS patients' problems. Similarly, Fludrocortisone pills can be prescribed to aid the body in retaining sodium and water. The improvement in symptoms is through the same principal of increasing blood volume.

Compression stockings can be helpful in helping reduce the blood pooling. Waist high compression stockings with a pressure of 30-40 mmHg are recommended (Grubb et al., 2006; Raj, 2006). Exercise is also helpful. Both aerobic and resistance training are recommended, and patients should aim for about 20-30 minutes of exercise at least three days per week (Grubb et al., 2006; Raj, 2006; Abed et al., 2012). Exercise should be started slow and increased as tolerated, using target heart rate and personal tolerance as a guide. Persistence is crucial, however.

Overall, management of this condition is very individualized with many possible treatment plans, not all of which were discussed here. POTS cannot be cured, as such, but it can be managed. Some patients respond better than others to management, but all patients deserve informed choices of the available and appropriate options. Not all management options are appropriate for every case, so healthcare provider direction and prescription is important.

### **Current Research**

This page displays the information from the studies reviewed in Chapter 2 of this paper. Additional studies may be added at some point in the future. The layout and website view of this information can be found in Appendix C but is identical, with the exceptions of the first paragraph and hyperlinks to the articles, to what is displayed in Chapter 2 of this paper.

### **My Personal Experience**

This will be a blog style section with thoughts, ideas, and experiences from my personal journey with POTS. The purpose of this section is to help encourage others and let them know that they are not alone and that someone else is experiencing similar difficulties. I will also share

suggestions and advice, both from my personal journey and from any questions or comments that I may receive as a result of this website. Please see Appendix D for the current content of this page, with the exception of different background coloring and an embedded video. It will expand and develop over the coming weeks and months as it progresses further.

### **Resources**

This section has a hyperlinked list of helpful resources. Resources include: Dysautonomia International, Dysautonomia Information Network (DINET), National Dysautonomia Research Foundation, Vanderbilt Autonomic Dysfunction Center, PMC US National Library of Medicine, National Institutes of Health to search for current articles and research, a link to find compression stockings from BrightLifeGo, and a link ([www.nuun.com](http://www.nuun.com)) to a product called “nuun electrolyte enhanced drink tabs” that can be added to water as an alternative to other sports drinks. Other resources will be added as I communicate with other patients I know and learn about various products or suggestions that work well for them.

### **Future Plans**

As already discussed throughout Chapter 3, the website will continue to develop and change over the coming months as I get a feel for what is helpful and what might be lacking. In the past I have kept up a YouTube channel with videos I have recorded commenting on my experiences with POTS. I got a good number of views as well as comments and questions with that and am considering incorporating something like that again in connection with the website. Also, I have designed and printed postcard sized cards that can be given or mailed to patients, healthcare providers, family members, and others that might be interested in learning more. The design is a picture description of POTS that I designed using Microsoft Word. On the back are a small quick view calendar, a short description of who might be interested in visiting my website,

and a small logo including the URL to my website (see Appendix E). My intention is that my website can be a solid starting point toward learning more, and visitors can utilize the Resources page to visit other links that have additional and different information and resources. I also was asked to present my project during grand rounds at the inpatient Clinical Research Center at Vanderbilt in Nashville, TN, where I have also been a patient for several studies, and will hopefully get an opportunity to do that in the future. I also hope to attend the 3<sup>rd</sup> annual Dysautonomia International conference if possible. In the coming months and years, I hope to only expand my work as advocate, educator, and resource/support person for those affected by POTS.

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## Appendix A

# Who, What, and Why

Postural orthostatic tachycardia syndrome (POTS) is thought to affect at least 500,000 people in the United States alone.<sup>1</sup> Women are affected by POTS more often than men with an estimate of a 4 or 5:1 female-to-male ratio.<sup>1,4,5</sup> In order to understand the symptoms and treatment options for POTS, it is important to understand what is causing the signs and symptoms of the condition.

## What is POTS?

Simply stated, POTS patients have a faulty autonomic nervous system that does not react correctly when the patient is upright. Imagine when you stand up: a healthy person without POTS has an autonomic nervous system that is working properly. When that person stands up, gravity causes a shift in the blood volume toward the lower body, and a slight increase in heart rate occurs to help push the blood back up.<sup>2</sup> The autonomic nervous system is working well, so the body compensates very quickly and pumps the blood back up into the rest of the body where it belongs. The heart rate remains stable with only minimal changes, and the person continues feeling well as stability and balance is maintained in the body. People with POTS have an autonomic nervous system that is not working properly. When they stand up and blood rushes into the legs, the body is unable to compensate correctly. This imbalance causes prolonged blood pooling in the legs and a significantly increased heart rate. Since the autonomic system does not react properly and does not pump the blood back up to the brain effectively, the brain and other organs soon feel the decrease in blood flow and many unpleasant symptoms can occur: such as lightheadedness, palpitations (feeling like your heart is beating really hard or fast), and feeling faint. The body continues to try and compensate for the faulty autonomic system and the prolonged shift in blood volume by making the heart beat faster. Once the patient sits and/or lies down again symptoms usually decrease or subside, but many POTS patients do report other varying symptoms such as fatigue and difficulty sleeping.<sup>3</sup> A more comprehensive list of symptoms can be found on the symptom page. But why does the autonomic nervous system not function properly?

## Categorizing POTS

### Primary

POTS can be categorized as either the primary form or the secondary form.<sup>1</sup> Primary POTS is not associated with any other known disease, whereas the secondary form of POTS is caused by some other known underlying illness or disease.<sup>4</sup> The primary form of POTS is usually caused by a peripheral autonomic neuropathy, causing the vessels in the periphery not to contract appropriately when the patient is upright. This “partial dysautonomic” form is the most common form of POTS and often occurs abruptly after a viral illness or some other precipitating event such as pregnancy, surgery or trauma, sepsis, or immunizations. The abrupt onset after these types of events has lead researchers to believe that this form of POTS is immune-mediated. The other type of “partial dysautonomic” POTS is developmentally based. Some teens seem to develop POTS in the adolescent years, and often times (around 80% of cases) these patients outgrow or make a near full recovery by the early to mid-20s. The least common form of primary POTS is the hyperadrenergic form. Hyperadrenergic POTS is associated with significantly elevated levels of norepinephrine (>600 ng/mL) while upright. These patients often exhibit orthostatic hypertension in addition to tachycardia. Patients often develop the hyperadrenergic form gradually and progressively, and this form is believed, at this point, to be caused by a genetic mutation causing an inappropriate overabundance of norepinephrine due to dysfunction of the reuptake transporter protein. The body is unable to clear that norepinephrine from the intrasynaptic cleft leaving the patient experiencing high levels of circulating catecholamines (stress hormones such as norepinephrine) and resulting in symptoms of excessive sympathetic (fight-or-flight) activity. Imagine if you were suddenly chased by a huge and hungry grizzly bear. How would your body react? What symptoms would you experience when that “fight-or-flight” reaction kicked in? Hyperadrenergic POTS patients experience that same type of reaction from simple and everyday precipitating factors such as standing up or other various triggers that set off that “fight-or-flight” cascade.

### Secondary

Patients that have the secondary form of POTS have some other underlying illness or disease, such as diabetes, Sjogren’s syndrome, or Lupus.<sup>4</sup> For instance, chronic

diabetes mellitus can cause peripheral neuropathy resulting in improper or absent peripheral autonomic “communication” thus causing the vessels to react improperly and the symptoms of POTS to result.<sup>4</sup> Other conditions such as mastocytosis and mast cell activation syndrome (MCAS) have also been linked to POTS.<sup>6</sup> Mast cell disorders are either proliferative (too many mast cells accumulating in tissue or organs, as in mastocytosis) or activation based (the correct number but overactive mast cells, as in MCAS).<sup>7</sup> The histamine and other substances being released cause the vessels to dilate causing blood pooling as in other types of POTS and the body experiences a hyperadrenergic type response as it deals with this perceived threat.<sup>6,7</sup> There is discussion on whether the “fight-or-flight” response initiates the mast cell response or whether the mast cell response causes the “fight-or-flight” response. Either way, the result is the same. It is also important to recognize that patients experiencing the secondary form of POTS will experience a different symptom range than those with one of the primary forms. Those with the secondary forms of POTS have symptoms and difficulties associated with the primary or associated condition as well as POTS. Further reading of the references provided here, as well as other materials and the “My Personal Experience” page, will provide further insight into the additional symptoms and issues that patients can experience in addition to POTS, based on whatever underlying condition is present.

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## Appendix B

# Symptoms

In order to understand why POTS patients experience the symptoms they do, it's important to understand what POTS is. Check out the "All About POTS" tab to learn more. Not every person has the same combination or severity of symptoms, but the following is a list of possible symptoms that POTS patients experience:

- Chronic dizziness<sup>1</sup>
- Light-headedness<sup>1-3</sup>
- Chest discomfort or pain<sup>1-3</sup>
- Palpitations<sup>1-3</sup>
- Dyspnea (difficulty breathing)<sup>3</sup>
- Shortness of breath<sup>1</sup>
- Tremor/tremulousness<sup>1-3</sup>
- Nausea<sup>2-3</sup>
- Headaches<sup>2-3</sup>
- Sleep difficulties<sup>3</sup>
- Mental clouding (brain fog)<sup>1-3</sup>
- Exercise intolerance<sup>2-3</sup>
- Fatigue<sup>2-3</sup>
- Pre-syncope (feeling like passing out)<sup>2-3</sup>
- Syncope (actual fainting)<sup>2-3</sup>
- Diaphoresis (increased amount of sweating)<sup>2</sup>
- Blurred vision<sup>2</sup>
- Early satiety (feeling full after only a small amount of food)<sup>2</sup>
- Anxiety<sup>2</sup>
- Flushing<sup>2</sup>
- Postprandial hypotension (low blood pressure after eating)<sup>2</sup>
- Lower back pain<sup>2</sup>
- Aching neck and shoulders<sup>2</sup>
- Cold hands<sup>2</sup>
- Hypovolemia (decreased blood volume)<sup>2</sup>
- Generalized weakness<sup>1</sup>

- Inappropriate sinus tachycardia<sup>1</sup>
- Gastrointestinal or bladder disorders<sup>1</sup>

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## Appendix C

# Current Research

Research is a very important element toward patients receiving appropriate care and treatment. If you are a POTS patient and are interested in getting involved by participating in some studies, follow [this link for more information on how you can get involved](#). The following are descriptions and implications of some of the recent research studies that have been done regarding POTS.

## Sleep and Quality of Life

[Bagai et al. \(2011\)](#), of Vanderbilt University, assessed (a) health-related quality of life, (b) fatigue, and (c) sleep quality in POTS patients, in comparison to healthy control subjects, by using a series of scales and questionnaires. A total of 90 participants were included in the study: 44 POTS patients who met the criteria for POTS diagnosis and 46 healthy control subjects recruited through a research volunteer database and advertisements within the local campus community.

Several tools were utilized during data collection. [Bagai et al. \(2011\)](#) used the Medical Outcomes Study Sleep Scale comprised of 12 questions in order to assess difficulties within several areas of sleep: snoring, sleep adequacy, sleep disturbance, headache, somnolence, and respiratory impairment. Each area is scored on a scale of 0-100, with a higher score for sleep adequacy indicating improved and better sleep, and a higher score on any of the other areas indicating increased dysfunction. The Epworth Sleepiness Scale ranging from 0 to 3, with a total possible score of 24, assesses subjective daytime sleepiness. Eight common situations of daily living are presented and respondents must indicate the likeliness of sleeping in each situation. Responses >10 are usually seen in individuals with diagnosed sleep disorders affecting daytime sleepiness, whereas responses <10 are considered normal. The Fatigue Visual Analogue Scale is a one question assessment of fatigue severity and uses a scale of 0-10, the higher responses indicating higher levels of fatigue. The RAND-36 is a more general questionnaire assessing quality of life in 8 domains: “physical functioning, role limitations caused by physical health problems, bodily pain, general health, emotional well-being, role limitations due to emotional problems, social functioning, and

energy/fatigue” (Bagai et al., 2011, p. 205). A 0-100 scale is used, asking participants to rate in each area over the last four weeks. Poorer quality of life is indicated by lower scores. Lastly, the EuroQol was used to collect subjective rating for level of problem within 5 areas relating to health: mobility, anxiety/depression, usual activities, self-care, and pain/discomfort. Participants rated each as either “no problem, some/moderate problems, or extreme problems” but results “were analyzed as dichotomous variables (no problems vs. some/moderate/extreme problems)” (p. 205).

The Medical Outcomes Sleep Scale found significantly poorer sleep adequacy and increased sleep difficulties in POTS patients than were found in the control subjects (Bagai et al., 2011). The Epworth Sleepiness Scale found a significant difference between POTS patients and control subjects. 51% of POTS patients scored >10, indicating excessive daytime sleepiness, in comparison with 16% of control subjects scoring above the same mark. The Fatigue Visual Analogue Scale found that a significantly higher number of POTS patients, in comparison to healthy controls, indicated fatigue being a major issue within the past seven days. The RAND-36 and EuroQol both indicated significantly poorer “health-related quality of life” in POTS patients than in the control subjects. The physical health scores were lower than were the mental health related scores (Bagai et al., 2011, p. 206).

Overall, Bagai et al. (2011) found that POTS patients experience more fatigue and sleep related difficulties than do healthy control subjects. Furthermore, these sleepiness and fatigue related difficulties are related to poorer quality of life. Interestingly, results showed that physical health was affected more than mental health, and because the control group scored worse in the snoring category, the authors have assumed that the self-reporting nature of the study, viewed as a possible limitation, did not in fact cause over-reporting of symptoms in the POTS group. Bagai et al. (2011) recommend further studies on this topic and involvement of more formal and objective methods to further understand the findings.

Bagai, K., Song, Y., Ling, J., Malow, B., Black, B., Biaggioni, I.,...Raj, S. (2011). Sleep disturbances and diminished quality of life in postural tachycardia syndrome. *Journal of Clinical Sleep Medicine*, 7(2), 204-210.

### **Inattention and Psychiatric Disorders**

[Raj et al. \(2009\)](#) used a cross-sectional controlled method to assess anxiety, attention, and mood in POTS patients (N=21) compared to ADHD participants (N=18) and normal control subjects (N=20) with no history of psychiatric or attention disorders. Previously validated and commonly used questionnaires were used. POTS patients met the diagnostic criteria for POTS, were tested while in the sitting position, were not currently taking any medications affecting psychiatric or autonomic function, and were asymptomatic during testing. Comparisons were done between two matched groups: POTS patients with ADHD participants, and POTS patients with normal control subjects with no history of attention or psychiatric diagnoses. Significance was set at the  $P < 0.05$  level.

Results showed that POTS patients did not experience significantly higher rates of anxiety disorders in comparison to the normal control subjects or those with ADHD ([Raj et al., 2009](#)). Similarly, POTS patients did not show a greater “lifetime prevalence of major depressive disorder” in comparison with the normal control subjects (p. 4). The ADHD participants, however, did show higher incidence of depressive disorders than both the normal control group and the POTS patients. POTS patients also showed no significant differences in substance dependence and abuse in comparison to the normal control group.

Significant differences were found among scores on the second edition of the Beck Depression Inventory for degree of depression symptoms between the groups for ([Raj et al., 2009](#)). Both POTS patients and ADHD participants scored higher in comparison with the control group for “magnitude of current depression symptoms” (p. 4). The difference between POTS and ADHD scores, however, were not significantly different. The Beck Anxiety Inventory showed significantly higher scores in POTS patients in comparison with both the control subjects and those with ADHD. The Anxiety Sensitivity Index showed significantly higher fear of anxiety related symptoms in the ADHD and POTS groups in comparison with the normal controls, however in comparison with the general population, POTS patients actually scored lower and ADHD subjects still scored higher.

The Connors Adult ADHD Rating Scale showed that POTS patients experienced significantly higher scores in comparison with the control subjects in three areas: inattention/memory problems, ADHD score, and ADHD index ([Raj et al., 2009](#)). However, those participants with ADHD scored significantly higher than POTS patients and the control subjects in all of the subscales measured. The ADHD Behavior Checklist for Adults measured symptoms both in childhood and in the past six months. Significant results showed that POTS patients experienced less symptoms in childhood in comparison to those with ADHD but no significant difference in comparison to the control group. However, for symptoms within the past 6 months, POTS patients showed significantly greater symptoms of inattention and hyperactivity in comparison with the control group but still less than those with ADHD.

Overall, [Raj et al. \(2009\)](#) found POTS patients to have no increased likelihood of developing a depression or anxiety related disorder. However, POTS patients did display a score indicating mild depression. [Raj et al. \(2009\)](#) discussed the possibility that this finding could be due to the difficulties associated with living with chronic illness. It was noted, that the quality of life that POTS patients experience has been compared with other chronic diseases including heart failure and chronic obstructive pulmonary disease. Similarly, POTS patients did display scores indicating higher levels of anxiety on one scale, however the discussion on this was that POTS patients experience physiological physical symptoms that could be attributed to anxiety but instead is due to the pathology of the postural orthostatic tachycardia syndrome. Therefore, it was suggested that the anxiety symptoms are physically rather than psychologically based. Further studies using objective measures were recommended due to subjective nature of data collected and small sample size.

[Raj, V., Haman, K., Raj, S., Byrne, D., Blakely, R., Biaggioni, I.,...Shelton, R. \(2009\). Psychiatric profile and attention deficits in postural tachycardia syndrome. \*Journal of neurology, neurosurgery, and psychiatry\*, 80\(3\), 339-344. doi: 10.1136/jnnp.2008.144360](#)

### **Diurnal Variability in Heart Rate and Blood Pressure**

[Brewster et al. \(2012\)](#) conducted a study to examine diurnal variability within two groups, a POTS group (N=54) and a healthy control group (N=26). Those participants

in the POTS group had not taken any medications affecting hemodynamics for at least five half-lives of the medication. Fludrocortisone was discontinued, specifically, for at least five days prior to beginning the study. The healthy control group also did not take any medications affecting hemodynamics. Significance was identified as  $P < 0.05$ .

An automatic BP monitor was used to obtain orthostatic BP and HR measurements the night of admission to the research center and then again the next morning ([Brewster et al., 2012](#)). Orthostatic measurements in both instances were taken supine and then after standing for five minutes. Both POTS patients and healthy controls experienced significantly greater orthostatic tachycardia and higher standing heart rates in the morning than in the evening. Supine HR measurements were unchanged between the evening and morning in the POTS patients while the healthy controls had a slight decrease in supine HR for the morning check. The only significant difference in BP was a slight decline in SBP for both POTS and control subjects in the morning measurement in comparison with the evening.

Interestingly, a higher percentage of participants, both POTS and healthy controls, met the orthostatic HR criteria ( $\geq 30$  bpm) of POTS in the morning than did in the evening ([Brewster et al., 2012](#)). This finding suggests that this phenomenon is a normal finding that is simply increased in POTS patients. Brewster et al. (2012) discussed that consideration should be given to standardizing time of day for data collection in diagnosing POTS. More patients could possibly be accurately diagnosed if measurement time was standardized to morning.

[Brewster, J., Garland, E., Biaggioni, I., Black, B., Ling, J., Shibao, C.,...Raj, S. \(2012\). Diurnal variability in orthostatic tachycardia: Implications for postural tachycardia syndrome. \*Clinical science\*, 122\(1\), 25-31. doi: 10.1042/CS20110077](#)

### **Propranolol for Symptom Improvement**

[Raj et al. \(2009\)](#) conducted a study utilizing a randomized crossover design to examine the efficacy of propranolol versus placebo in improving symptom load in POTS patients. Two protocols were used, some patients being eligible to complete both protocols. In the 1<sup>st</sup> protocol (N=54), propranolol 20 mg was compared with placebo, given on separate days within a randomized single-blind fashion. The 2<sup>nd</sup> protocol

(N=18) involved comparison of propranolol 20 mg versus 80 mg, again single-blind, randomized, and given on separate days. For both protocols, vital signs were monitored immediately before and hourly for four hours after administration of the study drug. Each vital sign measurement was taken with the patient seated and after standing for 10 minutes and was collected via a Dinamap that automatically and digitally input the measurements into the computer database. Nine symptoms were assessed immediately before study drug administration and after administration of drug at the two and four hour marks. Patients rated the severity of each of the nine symptoms on a scale of 0-10, with 0 being the absence of symptom. Significant results were defined as  $P < 0.05$ .

In protocol #1, significantly lower heart rate was found with propranolol 20 mg versus placebo at the one hour post drug administration for both the sitting and standing measurement and for the remainder of the four hour study period ([Raj et al., 2009](#)). Similarly, the SBP was significantly lowered after propranolol 20 mg versus placebo at the one hour check for standing, and continuing for three hours, and at the two hour mark for sitting, and continuing for the full four hours. The sitting and standing DBP were lowered as well. Overall, there was a significant overall change (decrease) in HR following administration of 20 mg propranolol versus placebo. In protocol #2, the standing heart rate and SBP was significantly lower with the 80 mg versus the 20 mg starting at the one hour check and continuing for the remainder of the four hour collection time. There was not any significant difference in sitting HR, SBP, or DBP readings between the 20 mg and 80 mg groups. In overall change for HR, SBP, or DBP, the only significant change was seen in HR decrease at the one hour check, with a greater change following 80 mg administration versus the 20 mg, and continuing for the full four hours.

Based on the symptom severity rating scores, propranolol 20 mg was found to decrease total symptom load in comparison with placebo ([Raj et al., 2009](#)). Interestingly, the low dose propranolol (20 mg) was also found to decrease total symptom load, overall, more than the high dose (80 mg). This finding is significant because it implies that lower dose propranolol is more effective at decreasing both HR and symptom load versus a higher dose. [Raj et al. \(2009\)](#) discussed the theory that POTS patients may need some elevation in HR in order to preserve optimal cardiac output and perfusion. It was suggested that the commonly reported failure of beta-blocker trial in POTS patients may

be due to commonly overprescribed dose. There were some reported limitations. The principle investigator was blind to the study drug until after the data collection was finished, but the nurse administering drug and collecting data was not. Therefore, it was single-blind only. [Raj et al. \(2009\)](#) stated, however, that this possible limitation was corrected by use of automatic collection devices that input data into the database directly. Furthermore, the symptoms were reported by the patient who was blind to study drug. Further research could be done with larger sample size for protocol #2 and a longer duration for follow-up after drug administration.

[Raj, S., Black, B., Biaggioni, I., Paranjape, S., Ramirez, M., Dupont, W., & Robertson, D. \(2009\). Propranolol decreases tachycardia and improves symptoms in the postural tachycardia syndrome \(POTS\): Less is more. \*Circulation\*, 120\(9\), 725-734. doi: 10.1161/CIRCULATIONAHA.108.846501](#)

## Appendix D

# Tips for Living with POTS

Enjoy this video I recently created. More posts to come soon, so check back in!

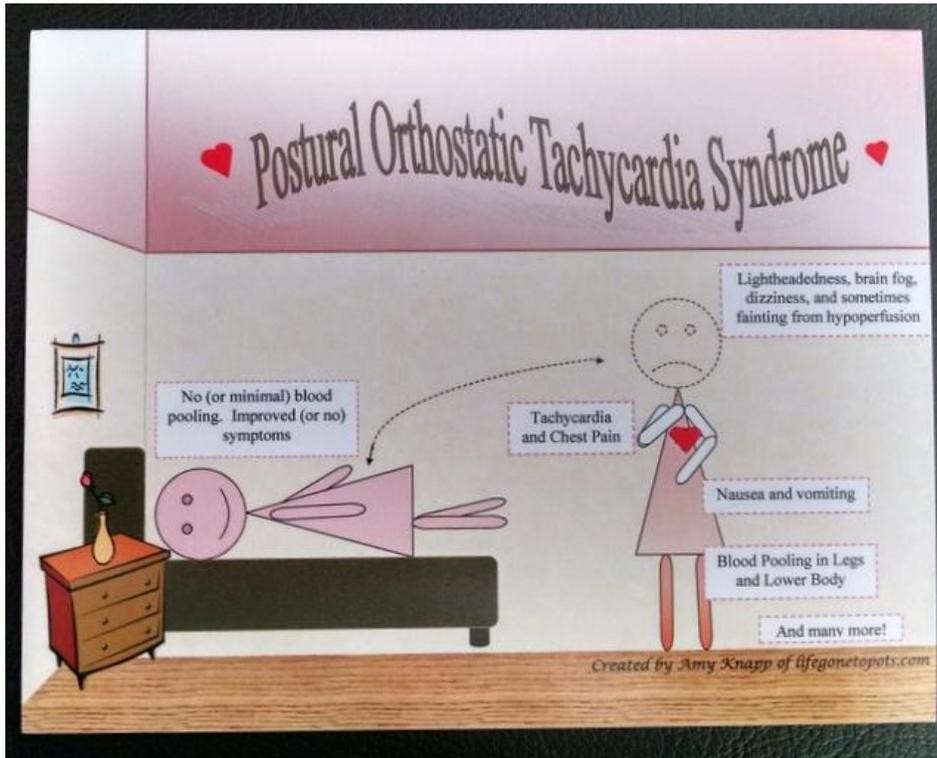
This video is embedded at this point: <http://youtu.be/AM652i2redM>

## Welcome!

October 24, 2014 My Personal Experience Edit

Welcome! I look forward to adding posts as time goes along and do plan on giving you more details about my experience with POTS. For now, know that I have lived with POTS for nearly 15 years and have experienced “good” days and very bad days. I want to share my experiences, thoughts, struggles, and advice with you so hopefully it will benefit at least one other person out there that may be having similar problems. Know that you are not alone! Don’t hesitate to contact me if you have any questions or thoughts you’d like to share.

Appendix E



### Postural Orthostatic Tachycardia Syndrome (POTS)

## 2015

<p><b>JANUARY</b></p> <p>S M T W Th F S</p> <p>1 2 3</p> <p>4 5 6 7 8 9 10</p> <p>11 12 13 14 15 16 17</p> <p>18 19 20 21 22 23 24</p> <p>25 26 27 28 29 30 31</p>	<p><b>FEBRUARY</b></p> <p>S M T W Th F S</p> <p>1 2 3 4 5 6 7</p> <p>8 9 10 11 12 13 14</p> <p>15 16 17 18 19 20 21</p> <p>22 23 24 25 26 27 28</p>	<p><b>MARCH</b></p> <p>S M T W Th F S</p> <p>1 2 3 4 5 6 7</p> <p>8 9 10 11 12 13 14</p> <p>15 16 17 18 19 20 21</p> <p>22 23 24 25 26 27 28</p> <p>29 30 31</p>
<p><b>APRIL</b></p> <p>S M T W Th F S</p> <p>1 2 3 4</p> <p>5 6 7 8 9 10 11</p> <p>12 13 14 15 16 17 18</p> <p>19 20 21 22 23 24 25</p> <p>26 27 28 29 30</p>	<p><b>MAY</b></p> <p>S M T W Th F S</p> <p>1 2</p> <p>3 4 5 6 7 8 9</p> <p>10 11 12 13 14 15 16</p> <p>17 18 19 20 21 22 23</p> <p>24 25 26 27 28 29 30</p> <p>31</p>	<p><b>JUNE</b></p> <p>S M T W Th F S</p> <p>1 2 3 4 5 6</p> <p>7 8 9 10 11 12 13</p> <p>14 15 16 17 18 19 20</p> <p>21 22 23 24 25 26 27</p> <p>28 29 30</p>
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<p><b>OCTOBER</b></p> <p>S M T W Th F S</p> <p>1 2 3</p> <p>4 5 6 7 8 9 10</p> <p>11 12 13 14 15 16 17</p> <p>18 19 20 21 22 23 24</p> <p>25 26 27 28 29 30 31</p>	<p><b>NOVEMBER</b></p> <p>S M T W Th F S</p> <p>1 2 3 4 5 6 7</p> <p>8 9 10 11 12 13 14</p> <p>15 16 17 18 19 20 21</p> <p>22 23 24 25 26 27 28</p> <p>29 30</p>	<p><b>DECEMBER</b></p> <p>S M T W Th F S</p> <p>1 2 3 4 5</p> <p>6 7 8 9 10 11 12</p> <p>13 14 15 16 17 18 19</p> <p>20 21 22 23 24 25 26</p> <p>27 28 29 30 31</p>

Whether you have POTS, know someone with POTS, or are a healthcare provider wanting more information about POTS: Visit my website for more information and resources, and to read about my personal experience with POTS.

[lifegonetopots.com](http://lifegonetopots.com)