

# Autonomic Dysfunction and POTS

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27 February 2022

AAP 33rd Annual Advances in Pediatrics Symposium

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# Pearls

- POTS is multifactorial in etiology and may be accompanied by multi-system comorbidities
- Autonomic function testing can help us to identify the type of autonomic dysfunction
- POTS should be treated using a multidisciplinary approach





# Objectives

- POTS background and definition
- Comorbidities
- Diagnosis
- Treatment





# Objectives

- POTS background and definition
  - History
  - Definition
  - Pathophysiology
  - Types
- Comorbidities
- Diagnosis
- Treatment





# History of POTS

- Described in mid-1800s as neurasthenia
- First described in 1993, first reported in teenagers in 1999
- Estimated to affect 0.2-1% of US population
- Age of presentation 15-50yrs
- Other autonomic dysfunctions
  - Neurocardiogenic syncope
  - Pure autonomic failure
  - Orthostatic hypotension
  - Inappropriate sinus tachycardia
- Pathophysiology is heterogeneous
  - Impaired sympathetically mediated vasoconstriction in LE (neuropathic)
  - Excessive cardiac sympathoexcitatory responses (hyperadrenergic)
  - Volume dysregulation
  - Physical deconditioning





# POTS features

- Typically begins within 1-2 years of onset of puberty
- More common in Caucasians?
- 15% have affected relative (parent or sibling)
  - Some genetic syndromes have findings including POTS
    - Norepinephrine transporter deficiency
    - Dopamine beta-hydroxylase deficiency
- Hyperextensibility – EDS?
  - Elastic soft tissues → POTS vs vasodilation → venous pooling
- Psychological features – “high achievers” ?due to hyperstimulated nervous system
- Triggered in 1/2 by febrile illness, injury-induced inactivity, concussion
- Female > male (adults 90%, adolescents 2/3)
- Common associated symptoms: dizziness, headache, nausea, brain fog, altered hot-cold sensation, abdominal symptoms, dependent acrocyanosis





# Postural Orthostatic Tachycardia Syndrome

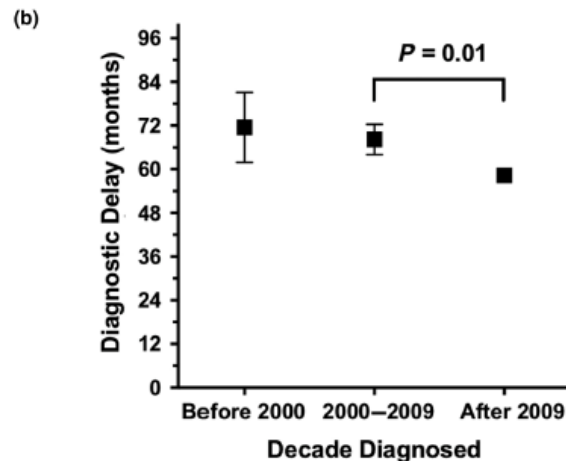
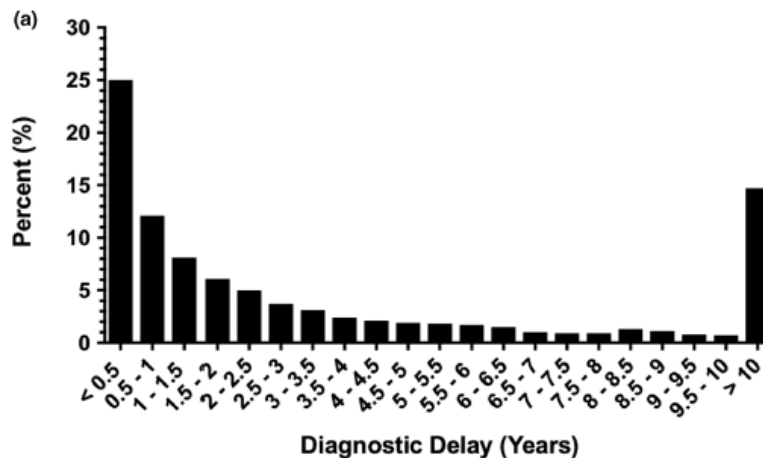
- “Complex, multi-system, chronic disorder of the autonomic nervous system characterized by orthostatic intolerance with excessive heart rate (HR) increase and symptoms on standing while blood pressure is maintained. Orthostatic symptoms improve rapidly after return to a supine position.”
- **Criteria**
  1. A sustained HR increment of not less than 30 bpm within 10min of standing or HUT. For 12-19yrs, required HR increment is  $\geq 40$ bpm
  2. An absence of orthostatic hypotension (no sustained SBP drop  $\geq 20$ mmHg)
  3. Frequent symptoms of orthostatic intolerance during standing, with rapid improvement upon return to a supine position (lightheadedness, palpitations, tremulousness, generalized weakness, blurred vision, fatigue)
  4. Duration of symptoms for at least 3mos
  5. Absence of other conditions explaining sinus tachycardia (anorexia, anxiety, HIV, anemia, fever, pain, infection, dehydration, hyperthyroidism, pheochromocytoma, cardioactive drugs, severe deconditioning)





**Table 3** *Diagnostic journey in POTS patients*

	Number (%) or mean (SD)
Misdiagnosed prior to POTS diagnosis	3421 (75%)
POTS diagnosis suggested by patient	1557 (34%)
Number of physicians seen prior to diagnosis	7 (11)
Number of ED visits prior to diagnosis	9 (16)
Specialty of physician who made diagnosis	
Cardiologist	1973 (41%)
Cardiac electrophysiologist	696 (15%)
Neurologist	889 (19%)
Family physician	392 (8%)
Emergency room physician	79 (2%)
Rheumatologist	74 (2%)
Other	711 (15%)





# Signs/symptoms

- **Orthostatic intolerance/cerebral hypoperfusion:** lightheadedness, palpitations, tremor, generalized weakness, blurred vision, sleep disturbances
- **Sympathoexcitation:** palpitations, chest pain, SOB, tremulousness, nausea, diarrhea, pallor, sweating, extremity coldness
- Vasovagal syncope 20-30%, but many with presyncope
- Symptoms exacerbated by heat, physical exertion, heavy meals, prolonged recumbency, menses, medications (diuretics, vasodilators, sympathomimetics)
- May be cyclical





# Role of anxiety/panic/hypervigilance in POTS

- Poorly understood roles
  - Abnormal processing of viscerosensory information, conditioning, behavioral amplification
    - Persists despite adequate HR. control, fails to readapt
  - Somatic hypervigilance and behavioral amplification → persistence
    - Poor sleep, anxiety, depression → relative sympathetic vs vagal predominance of HR
- Common clinical features
  - Fear/discomfort at symptom onset
  - Involvement of sympathetic noradrenergic system





# Symptoms in Adult Patients

- Mayo Clinic: 152 adult patients with POTS
- Non-orthostatic symptoms: chronic fatigue, sleep disturbances, migraines, fibromyalgia, functional GI and functional bladder disorders
- Exclusion of other causes of sinus tachycardia: acute physiological stimuli, diet, medications, anemia, dehydration, hyperthyroidism, inappropriate sinus tachycardia

Cutsforth-Gregory J, Handb Clin Neurol 2019;161:429-445  
Thieben MJ, Mayo Clin Proc 2007;82:308-313.

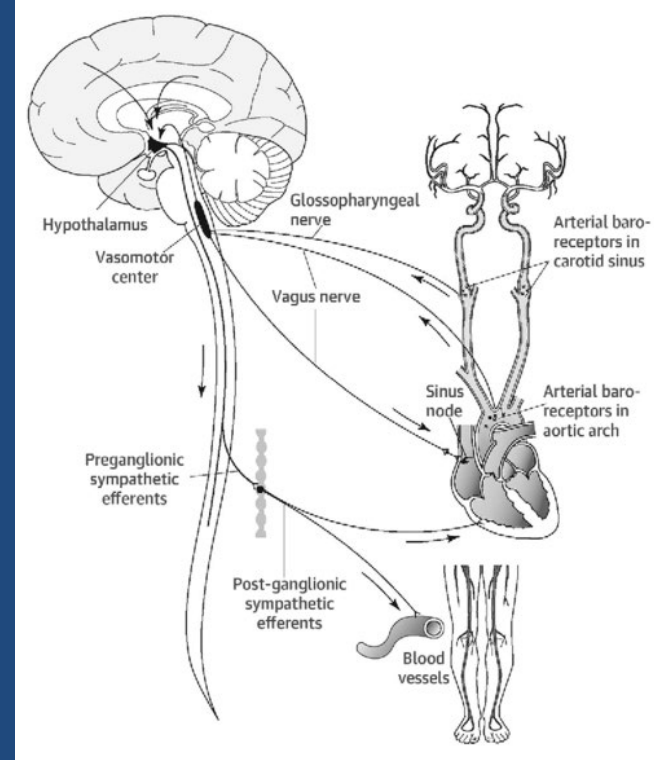
## Symptoms in patients with POTS

	Frequency (%) <sup>a</sup>
Orthostatic symptoms	
Lightheadedness or dizziness	78
Palpitations	75
Presyncope	61
Sense of weakness	50
Tremulousness	38
Shortness of breath	28
Chest pain	24
Hyperhidrosis	9
Anhidrosis	5
Nonorthostatic symptoms	
Fatigue	48
Nausea	39
Sleep disturbance	32
Migraine	28
Bloating	24
Diarrhea	18
Myofascial pain	16
Constipation	15
Abdominal pain	15
Bladder symptoms	9
Vomiting	9
Pupillary symptoms	3
Neuropathic pain	2



# Physiology of standing

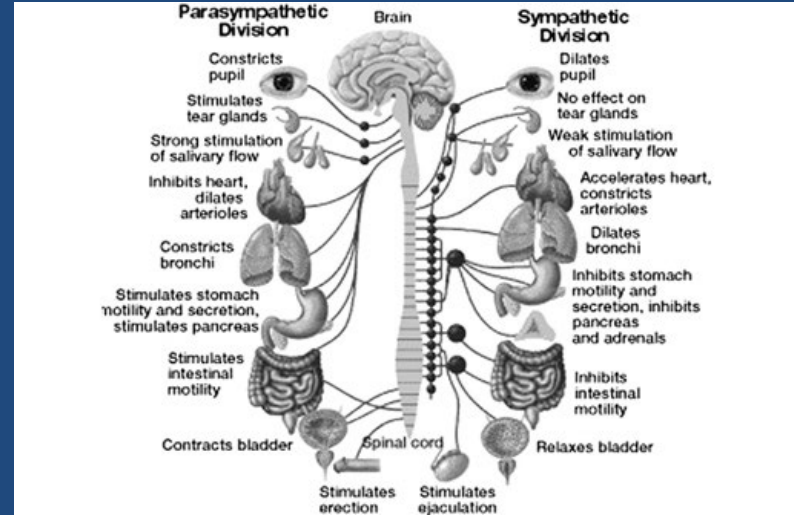
- $\frac{1}{4}$  blood volume in thorax when supine
- Upright  $\rightarrow$  500-1000mL shifts to LE and splanchnic circulation and 10-25% of plasma volume to interstitial space
- $\rightarrow$  impaired venous return to heart to reduce cardiac filling, stroke volume, BP
  - High-pressure arterial baroreceptors in carotid sinus and aortic arch  $\rightarrow$  stimulate sympathetic efferent nerve activity, suppress parasympathetic activity to heart and vessels
  - $\rightarrow$  cardioacceleration, increases systemic vascular resistance to enhance venous return
  - $\rightarrow$  engaged skeletal muscle pump system, activation of renin-angiotensin and endothelin systems



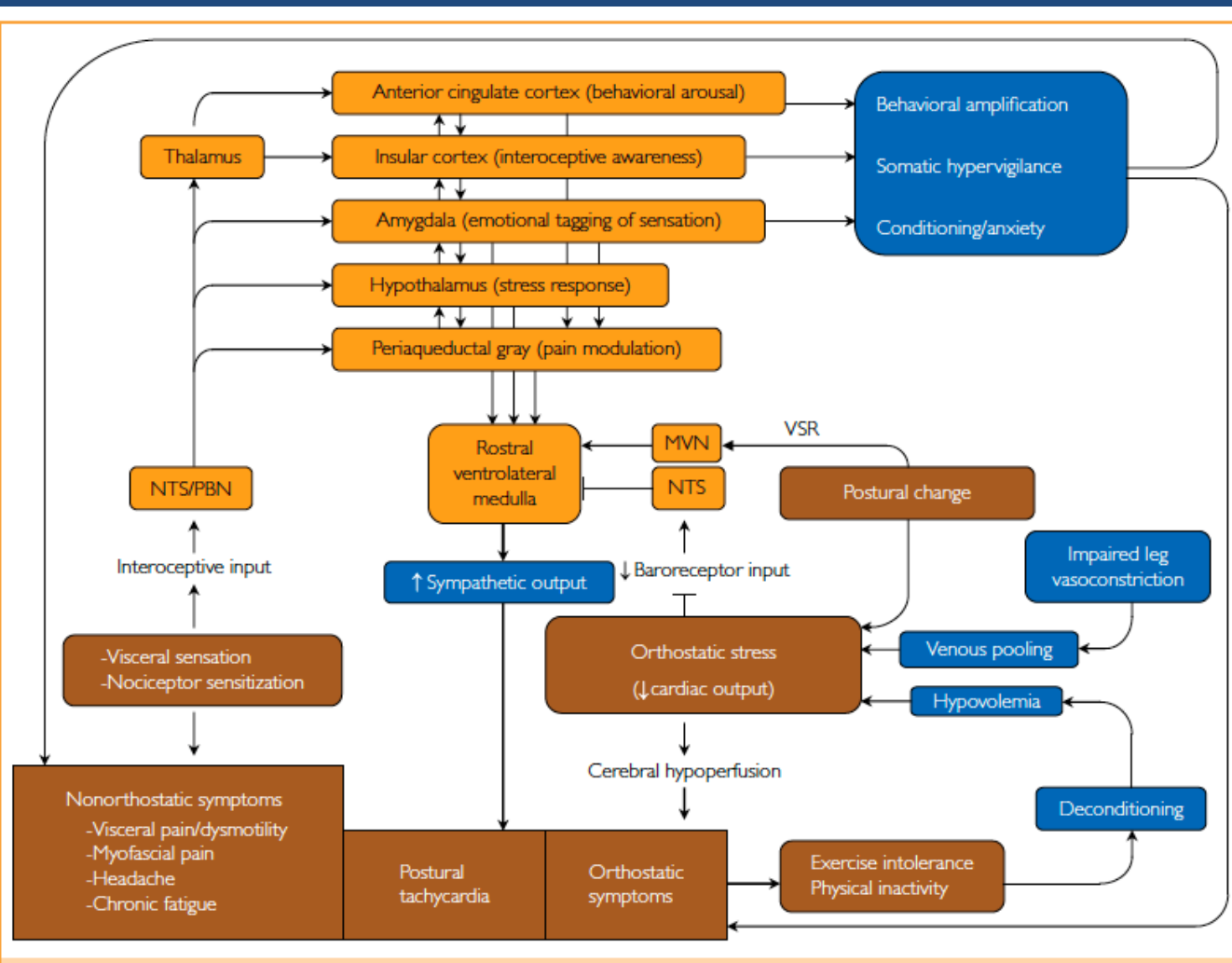


# Pathophysiology

- Orthostatic intolerance
  - Defective cerebral autoregulation – lightheadedness, reduced neurocognitive function, mental fatigue, headache
  - Sympathetic activation and parasympathetic withdrawal – sweating, pallor, exercise intolerance, GI symptoms, hypertension
- Tachycardia
  - Sinoatrial node abnormalities
  - Hypovagal responses
  - Hypersensitivity to norepinephrine
  - $\beta$ -receptor-sensitive inappropriate sinus tachycardia





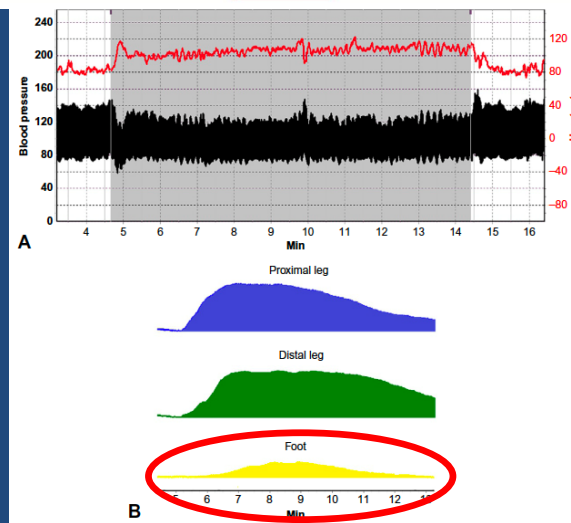
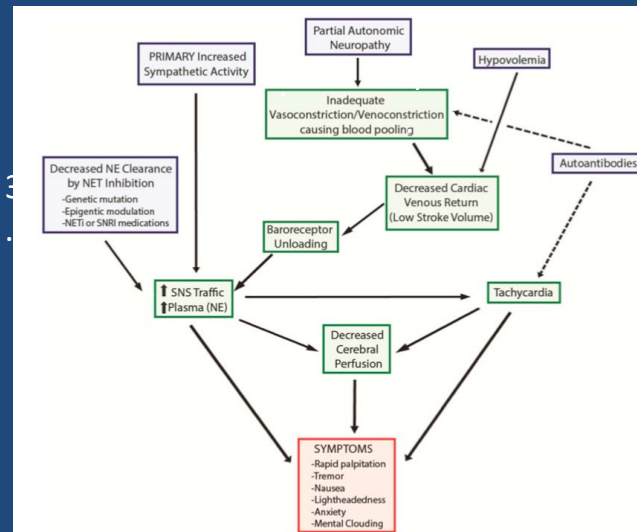


Benarroch EE,  
Mayo Clin Proc,  
2012;87(12):1214-  
1225



# POTS types

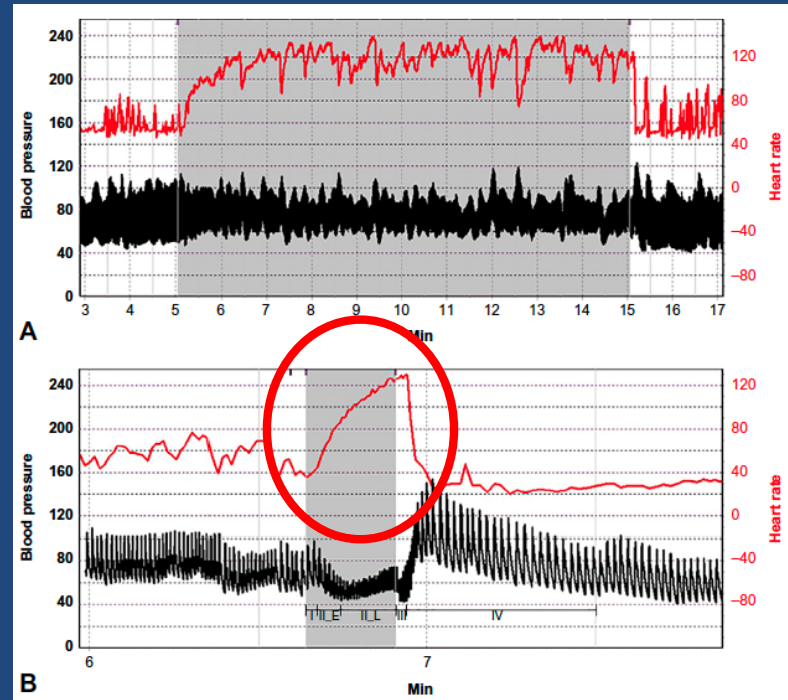
- Neuropathic “high flow” POTS – regional partial sympathetic denervation/adrenergic hypoactivity
  - Impaired sympathetic vasoconstriction in legs – **distal small fiber neuropathy**
  - Decreased NE release in LE - adrenergic impairment
  - Rarely autoimmune autonomic neuropathy
    - Organ-specific autoantibodies in 1/3 (thyroid)
  - Reduced stroke volume → reflex tachycardia and vasoconstriction
  - Sudomotor impairment
  - GI complaints common





# POTS types

- Hyperadrenergic POTS – “low-flow” upright adrenergic overactivity dominates
  - Pre- or post-synaptic adrenergic potentiation → **excessive sympathetic tone**
  - Initial decreases of CBF when upright → hyperpnea, hypocapnia, extreme tachycardia, HTN, induced anxiety
  - Primary (high plasma NE, 5-10%) vs secondary (heterogeneous)
    - Increased synaptic NE in genetic condition of deficient NE transporter protein
    - Associated with MCAS - mast cell activation syndrome – flushing, diarrhea, nausea, vomiting
  - Supine vasoconstriction, supine tachycardia, pale and cold skin, increased supine muscle sympathetic nerve activity



- BP overshoot during phase IV of Valsalva, can be normalized by IV propranolol
- Fluctuating BP or hypertensive response during HUTT





# POTS types

- Hypovolemic POTS
  - Impaired renin-angiotensin-aldosterone system → sodium retention, **decreased blood volume**
    - → reduced preload
  - 24hr urine sodium – 100mEq/24hr
  - Low plasma renin, aldosterone
  - May be result of poor oral intake due to nausea or excess loss due to diarrhea
- Bedrest POTS/**deconditioning**
  - orthostatic intolerance
    - Persistent tachycardia, reduced peak oxygen uptake when upright, and during/after exercise
  - Decreased blood volume and cardiac size, redistribution of blood, osteoporosis, skeletal muscle pump atrophy, impaired vasoconstriction





# POTS types

- Impaired cerebral autoregulation – paradoxical cerebral arteriolar vasoconstriction → cerebrovascular resistance, caused by hypocapnia
  - Abnormal orthostatic drop in CBFV during HUT in the absence of OH, arrhythmia, vascular abnormality
    - Reduction in CBFV proportional to angle of HUT
  - Can be reversed by CO<sub>2</sub> rebreathing
- Genetic predisposition likely modest
  - $\beta_2$ -AR
  - $\alpha_2$ -AR
- Orthostatic hypertension resulting from reduced cardiac preload from hypovolemia or venous pooling → decreased venous return → fall in cardiac output → increased sympathetic stimulation



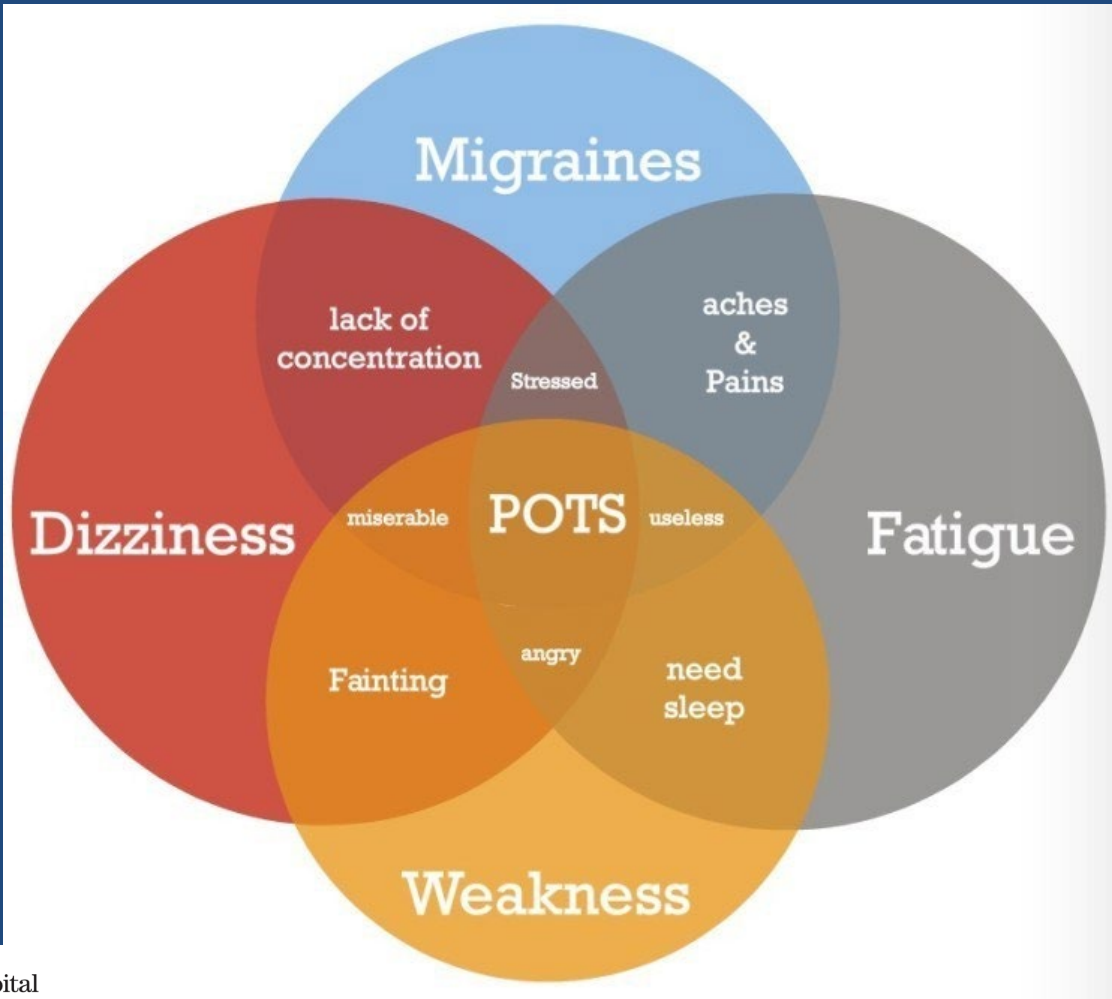


# Objectives

- POTS background and definition
- Comorbidities
  - Headaches/chronic pain
  - Chronic fatigue syndrome/myalgic encephalomyelitis
  - Functional GI disorders
  - Mast Cell Activation Syndrome (MCAS)
- Diagnosis
- Treatment









# Frequent comorbidities

**Table 2** Common comorbidities in POTS patients

Comorbidity	Number (%) (of 3933 respondents)
Migraine headaches	1557 (40%)
Irritable bowel syndrome	1192 (30%)
Ehlers-Danlos syndrome	994 (25%)
Chronic fatigue syndrome	809 (21%)
Asthma	798 (20%)
Fibromyalgia	786 (20%)
Raynaud's phenomena	610 (16%)
Iron deficiency anaemia	628 (16%)
Gastroparesis	548 (14%)
Vasovagal syncope	499 (13%)
Inappropriate sinus tachycardia	448 (11%)
Mast cell activation disorder	353 (9%)
Autoimmune disease	616 (16%)
Hashimoto's thyroiditis	228 (6%)
Coeliac disease	133 (3%)
Sjögren's syndrome	112 (3%)
Rheumatoid arthritis	93 (2%)
Lupus	81 (2%)
Other	160 (4%)

Other autoimmune conditions included the following: idiopathic thrombocytopenic purpura, Addison's disease, Grave's disease, Behcet's disease, autoimmune pancreatitis, autoimmune hepatitis, vasculitis, multiple sclerosis, myasthenia gravis and type 1 diabetes mellitus.

Shaw BH, J Int Med 2019;286:438-448

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# Headaches and chronic pain

- 2/3 complain of chronic daily headache with dizziness
- Symptoms similar to migraines
- Headaches aggravated by standing, relieved by rest
- Pediatric chronic pain estimated at 20-35% prevalence
  - Disruption of peripheral nerve activity
  - Central sensitization
- Joint hypermobility – EDS III – mutations in genes encoding fibrillar proteins or collagen-processing enzymes → reduced structural integrity of connective tissue ? → impaired venous return and venous pooling
  - Chronic pain of shoulders, hands, knees → deconditioning
  - Associated anxiety, depression, somatosensory amplification state





# Chronic fatigue syndrome

- Unexplained, persistent or relapsing chronic fatigue that is new or definite onset, not result of ongoing exertion, not substantially alleviated by rest, and results in substantial reduction in previous baseline function
  - Accompanied by 4 or more: self-reported short-term memory impairment, sore throat, tender cervical or axillary lymphadenopathy, msk pain, non-restorative sleep, post-exertion malaise
    - May occur post-infectious
    - Risk factors: female, older age, physical inactivity, depression, anxiety
    - Cluster with anxiety and depression
    - Associated with higher resting HR and BP
    - Stress → sympathetic overactivity
    - During sleep – ↑HR, ↓HRV, ↑NE, ↓aldosterone – sympathetic predominance
    - ↓exercise tolerance – slow HR acceleration, fatigue before peak HR





# Lack of sleep vs fatigue

- Why sleep?
  - Memory consolidation/pruning
  - Immune regulation
  - brain plasticity
  - Restorative therapy
- Sleep deprivation
  - → learning and behavioral difficulties
  - → inattentiveness
  - → impulsivity
  - → mood disorders
  - → weight problems
  - → increased risk-taking behaviors
- Look for sleep disorders and poor hygiene
  - Adolescents should be sleeping 9.25hrs
    - Circadian rhythm shift → delayed bedtime during puberty
    - Stressors
    - Lifestyle
    - Screen time





# Cardiac comorbidities

- Mitral valve prolapse (4%)
  - High urinary epinephrine, norepinephrine excretion
  - High plasma catecholamine
  - Elevated resting HR
- Inappropriate sinus tachycardia
  - Resting HR > 100bpm with mean 24hr HR > 90 bpm
  - Distressing symptoms of palpitations
  - Excessive sympathetic tone





# Functional gastrointestinal disorders

- Sympathetic denervation  $\pm$  parasympathetic dysfunction
- Symptoms: abdominal pain (15%), nausea (39%), diarrhea (18%), constipation (15%), bloating (24%)
- Electrogastrography shows decreases in normal gastric activity
  - Splanchnic hypervolemia and increased splanchnic venous pooling

*Thieben MJ, Mayo Clin Proc, 2007;82:308-313.*





# Mast Cell Activation Syndrome

- Symptoms: episodes of flushing, urticaria, dyspnea, headache, excessive diuresis, GI symptoms
- Elevated urine methylhistamine or 11- $\beta$ -Prostaglandin F2 excretion or elevation of other mast cell mediators



# Objectives

- POTS background and definition
- Comorbidities
- **Diagnosis**
  - History and Physical Exam
  - Autonomic Testing
    - Tilt table test vs active standing test
    - Heart rate response to deep breathing
    - QSWEAT
    - Valsalva
- Treatment





# Physical Examination

- Postural tachycardia + upright dizziness  
± nausea ± heaviness of extremities
- Mottling/duskiness/bluishness of  
extremities
- Lowish BP
- Dilated pupils





# Diagnosis

- Passive tilted challenge
  - Adapted to supine for 10mins, then gently tilted to 70'
  - HR changes recorded
  - Maximum sustained rate during tilting compared to baseline over 10mins
  - False + due to anxiety, dehydration, deconditioning
  - BP may
    - Oscillate – poor vasomotor tone → neuropathic POTS
    - Increase significantly → hyperadrenergic POTS
    - Remain stable
- EKG
- Cardiovagal and sudomotor function (1/2-2/3 abnormal) tests
- Maximal exercise test with peak oxygen uptake measurement
- Gastric motility if GI symptoms





# Diagnostic testing rationales

## Laboratory evaluation for suspected POTS

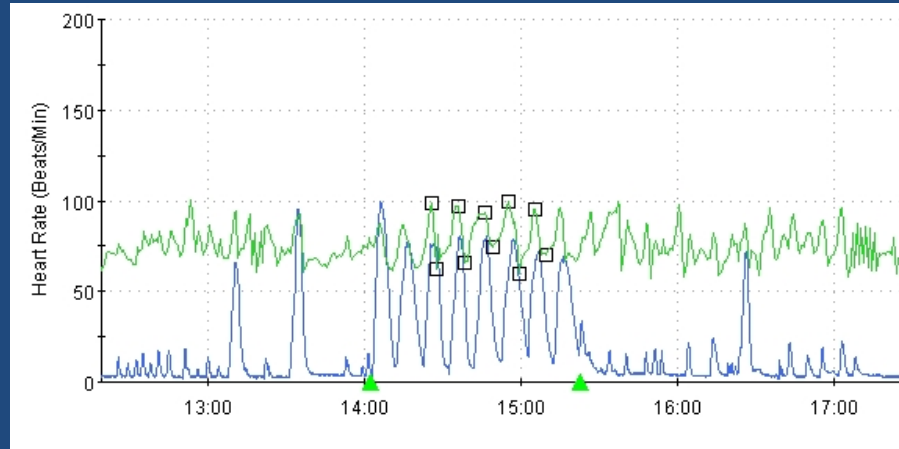
Investigations	Rationale
ECG, echocardiogram, Holter monitoring	Exclude primary cardiac cause of tachycardia
Head-up tilt (at 60–70 degree angle for 10 min)	Distinguish POTS from other forms of OI
Exercise testing with VO <sub>2</sub> max	Quantify physical deconditioning
24-h urinary sodium excretion	Assess for hypovolemia
Sudomotor and cardiovascular function tests	Detect neuropathic subtype of POTS and, if found, its cause
Fasting glucose, hemoglobin A1c	
Serum and urine protein electrophoresis	
Ganglionic AChR autoantibodies	
Supine and standing plasma catecholamines	Detect hyperadrenergic subtype of POTS and, if found, its cause
AM and PM cortisol	
Thyroid function tests	
Plasma and urine metanephrines	
Serum tryptase, urinary methylhistamine	
Scintigraphic motility studies, urologic evaluation	Investigate suspected functional visceral dysmotility syndromes
MRI of the head with gadolinium	Exclude CSF leak in patients with orthostatic headache





# Heart rate response to deep breathing (HRDB)

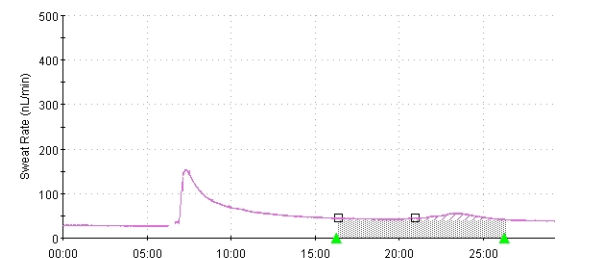
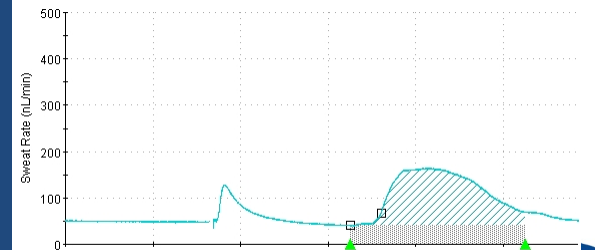
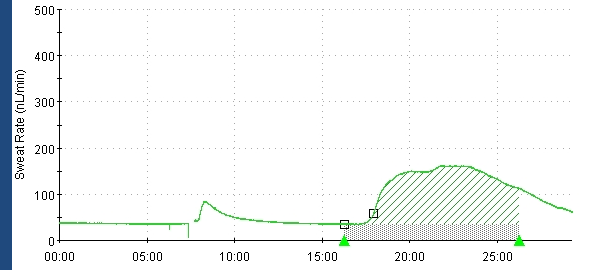
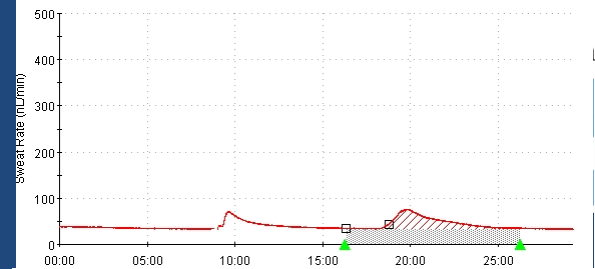
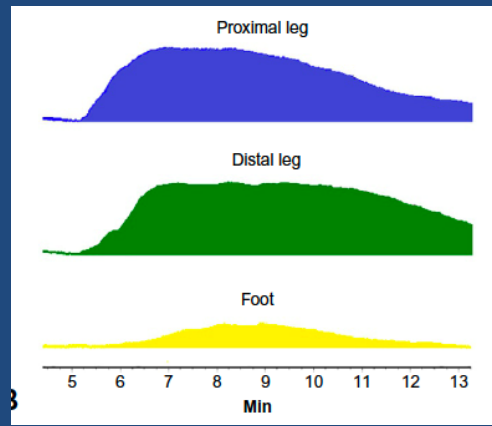
- Mean heart rate difference of 30.2bpm within expected normative range (14-41bpm)





# QSWEAT

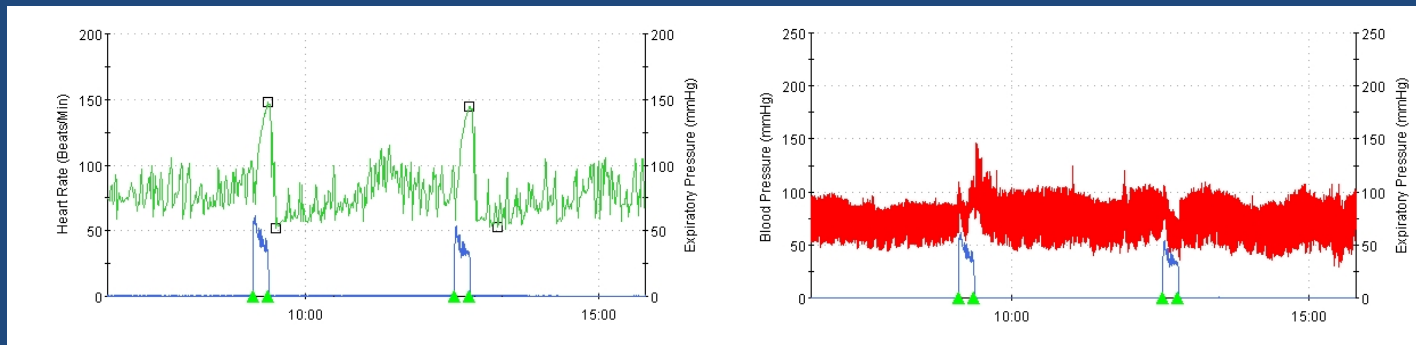
- Normal sweat responses in the forearm, proximal and distal leg, and foot
- No abnormalities in sudomotor axon reflex





# Valsalva Analysis

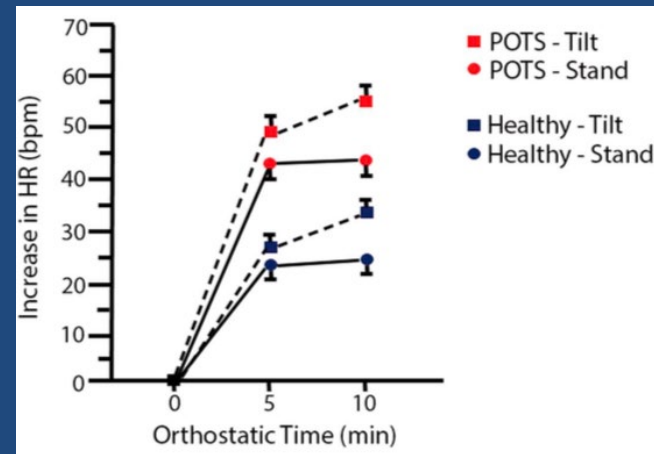
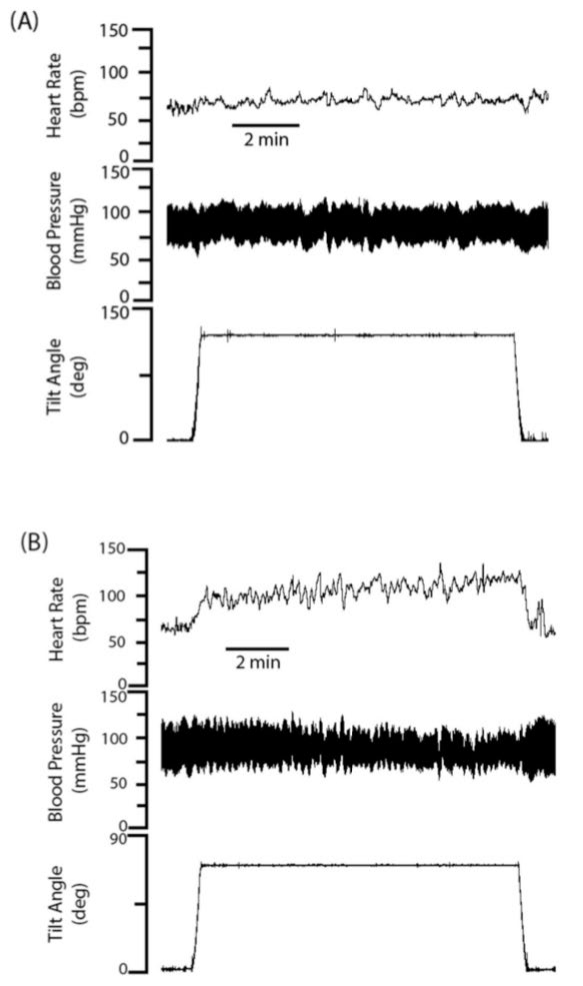
- Valsalva ratio 2.86 is above expected age-normative range (1.46-2.73)



- Adrenergic sensitivity analysis notable – adrenergic impairment
  - late phase 2 not returning to baseline
  - Prolonged pressure recovery time







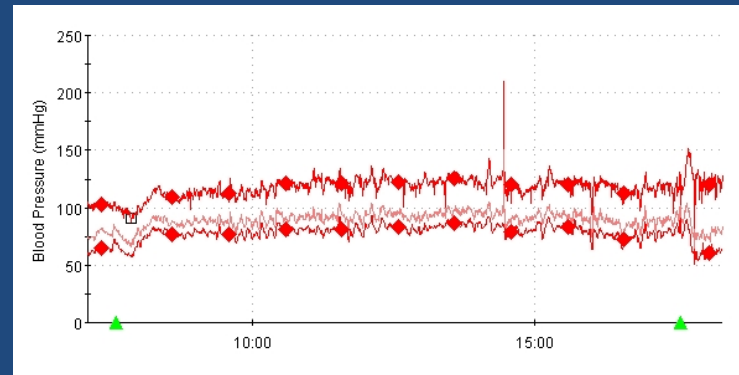
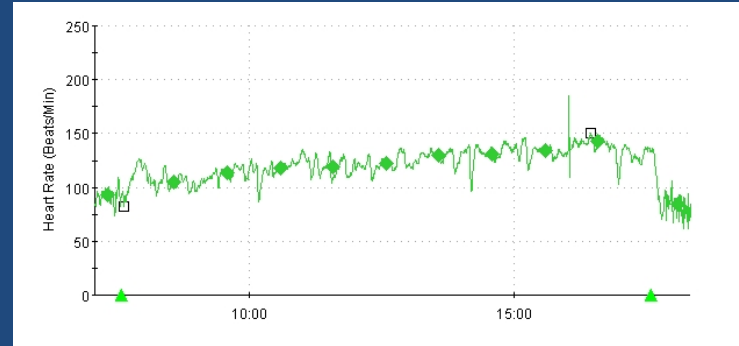


# Tilt table test

- 0: Really dizzy and seeing spots. 2: Same as above plus a headache and chest tightness. 4: Legs hurt. Dizzy as above. Light headed. Nausea. 6: Same as above. 8: Presyncopal. Joint pain. 10: Feels a bit better after tilt down. Sinus with atrial bigem
- Maximal SBP drop of 11.6 mmHG (normal<30mmHG) with tachycardia response of 67.8 bpm (normal<40bpm) indicates significant orthostatic tachycardia

Composite autonomic symptom score = 1, mild GAI

- Cardiovagal
- Adrenergic
- Sudomotor





# Objectives

- POTS background and definition
- Comorbidities
- Diagnosis
- Treatment
  - Nonpharmacologic
    - Behavioral modifications
    - Exercise
    - Therapies
  - Pharmacologic





# Nonpharmacologic treatments

- 2015 HRS consensus: non-pharmacologic interventions first
  - Discontinue meds that may worsen
  - Fluid 2-3L: caffeine-free fluids (measure by clear, non-yellow urine)
  - Salt intake 10-12g - 24hr urine – 170mmol/day
  - Compression stockings
  - Regular exercise - aerobic and resistance training → increases effective circulatory volume
- Avoid symptom exacerbation: sudden postural changes, prolonged recumbency, high temperatures, large meals, vasodilators, sympathomimetic drugs







Fludrocortisone (synthetic aldosterone) – fluid and salt retention (0.05-0.1mg q-bid)

- May worsen headaches, hypokalemia, caution in renal disease

Lower HR

- Beta-blockers – reduce blood pooling → ↑ venous return
  - propranolol sometimes increases fatigue in adolescents, hypotension
- Ivabradine – reduces HR by blocking funny channel – which modulates sinus node pacemaker rate, bradycardia

Pyridostigmine → acetylcholinesterase inhibitor → Ach → ↓ HR

- Diarrhea, abdominal cramps, bladder symptoms

Midodrine –  $\alpha$ -1 adrenergic agonist vasoconstrictor → ↑ venous return (2.5-10mg tid)

- may increase headache, HTN when supine, scalp paresthesias, supine urinary retention

Ocreotide – small bowel motility

Vasoconstrictors

- Inhibits antral contraction → erythromycin premedication

- Diarrhea, abdominal pain

Droxidopa – prodrug to NE → binds to  $\alpha$ -1 adrenergic R → ↑ vasoconstriction, BP

- Supine HTN

Stimulants?

Clonidine? – central sympatholytic – good for \prominent hyperadrenergic features

- Fatigue, hypotension, mental clouding

SSRI – increase GI flow, blood flow

Erythromycin – promotility

- Prolonged QT

Cyproheptadine – reduce mast cell activation

# Medications

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## Clinical Feature

## Drug Treatments

Tachycardia ++  
Palpitation

Beta- Blockers  
(Low Dose)

Blood Pooling  
HR not too High

Midodrine

Orthostatic  
HYPERtension

Methyldopa;  
Clonidine

Hypovolemia

Fludrocortisone  
DDAVP

Hypotension  
Freq Syncope

Pyridostigmine

Constipation

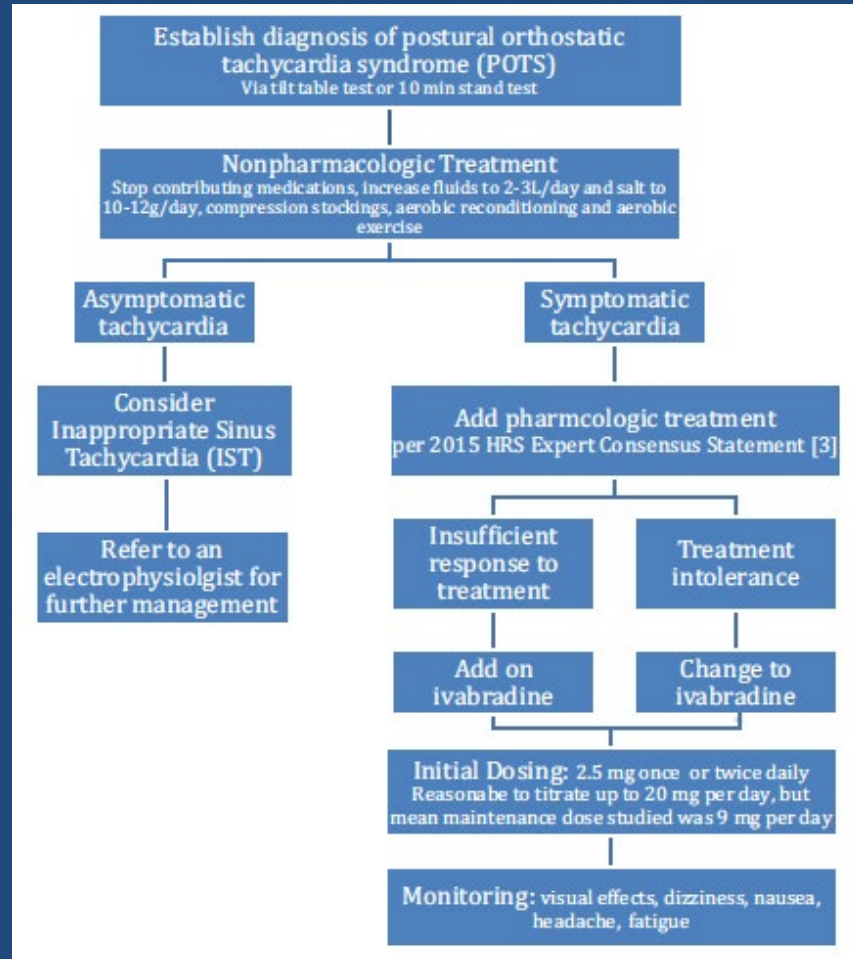
“Brain Fog”  
Cognitive Issues

Modafinil  
Stimulants (?)



# Ivabradine

- Selectively inhibits  $I_f$  current in a dose—dependent manner
- Blocks inward-directed hyperpolarization-activated cyclic nucleotide-gated channels in sinoatrial node cells
  - Slowed diastolic depolarization → reduced HR
  - Slows HR without affecting BP
    - No effect on myocardial contractility





# POTS treatment requires a multidisciplinary approach

- Goal is to return patient to fully functional
  - Symptom management
  - Sleep hygiene
  - Exercise
    - Decrease in upright HR
    - Effect on chronic pain through activation of central inhibitory pathways → analgesic effect mediated by opioid receptors in the rostral ventromedial medulla
    - Release of metenkephalin in brainstem nuclei
  - CBT
- If necessary – intensive pain rehabilitation program
  - Increase pain management
  - Increase QoL, emotional well-being
  - Improve school attendance

A structured endurance training program.

Workout zone	RPE	Target HR	Month 1	Month 2	Month 3
Base pace	13–15	~75% of maximal HR	10@25–30 min	6@30 min 3@35–40 min	5@35 min 4@45–60 min
Maximal steady state	16–18	(220–age) ± 5 bpm	1@20 min 1@25 min	1@25 min 1@30 min 1@35 min	1@30 min 1@35 min 1@40 min
Recovery	6–12	< lowest base pace HR	2@30 min	3@35 min	3@40 min
Cardio modes			Rowing Swimming Recumbent bike	Month 1 modes plus upright bike	Month 1 and 2 modes plus elliptical and treadmill walking

RPE, rating of perceived exertion (subjective rating of the entire cardio workout on a scale of 6–20: 6 is very, very easy; 11 is fairly easy; 13 is somewhat hard; 15 is hard; 17 is very hard; 19 is very, very hard). HR, heart rate.





# Parent component

- Increase sick role and pain behaviors
- Solicitous behaviors increase anxiety
  - Attention to pain
  - Reduced expectation for chores, schoolwork
  - Reduced expectation for emotional control
- → pay attention to child's well behavior and coping attempts
- → minimize attention to pain behaviors
- → behavior management strategies such as praise, rewards, consequences to motivate child to utilize pain management strategies
- → make privileges contingent on wellness activities





# Missing school causes a cascade of disability

- Poor social adjustment
- Poor social skills
- Isolation
- Lack of education
- Lack of future employment opportunities
- → despair





# Mayo Clinic Pediatric Pain Rehabilitation Center

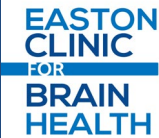
- Fifteen 8-hour days of a group pediatric outpatient interdisciplinary chronic pain rehabilitation program
  - 10-15 patients
  - Parents included
  - Goals related to restoring function and learning how to self-manage chronic pain, POTS symptoms
  - Include physician, psychologists, nurses, PT, OT, recreational therapists
  - Biobehavioral relaxation, PT, OT, recreational therapy, family group, 3h of CBT groups
  - 2 biofeedback sessions/week
  - Operant learning strategies
    - Elimination of pain and parent behaviors that secondarily reinforce pain behavior
  - Treatment of comorbid psychiatric illnesses
  - Tapering addictive medications (ie opiates, muscle relaxants, benzos)
  - Discontinuation of further medical workup and interventional procedures





# Treatment begins with establishing the diagnosis

- Assure the patient and family that the problem is real and physical
- Be positive and optimistic about recovery
- Validate their symptoms
- Give a specific plan
  - Focus on consistency, moderation, “just do it” approach
- Empower the patient and family with knowledge
- Focus on gradually working towards exercise goal





# Cognitive behavioral group therapy

- Pain management coping skills
  - Distraction
  - Positive self-talk
- Stress management
- Wellness instruction
  - Sleep hygiene
  - Healthy diet
- Chemical health education
- Activity pacing





# Relaxation strategies is a core element

- Diaphragmatic breathing
- Progressive muscle relaxation
- Imagery
- Self-hypnosis
- Biofeedback
  - Receive immediate feedback regarding level relaxation through awareness of muscle tension, HR, breathing, sweating

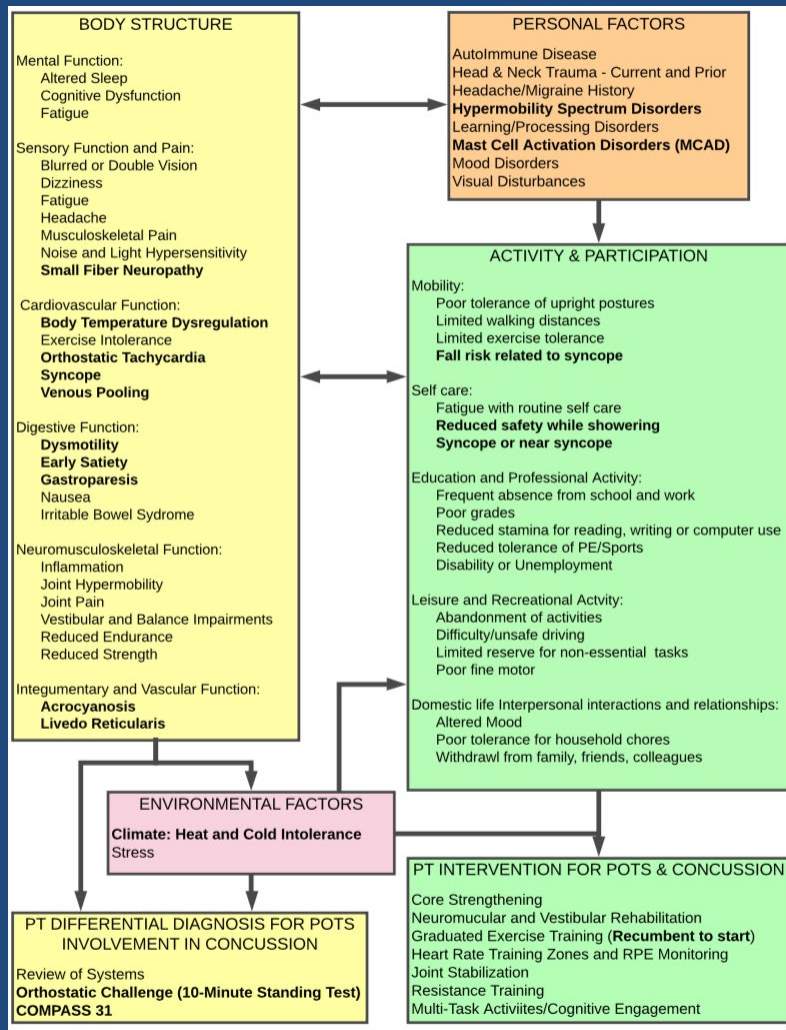


# Goal setting is important

- Remove the focus on symptoms
- Utilize a team including primary care
- Gradually incorporate changes so that plan does not feel overwhelming
- Encourage celebration of success and accomplishments
- Provide backup plan options



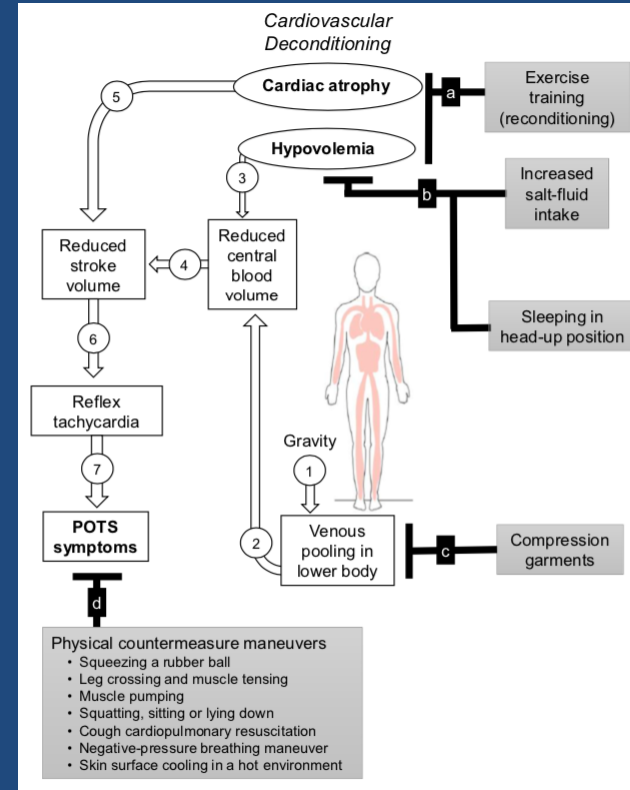






# Physical deconditioning and the role of exercise

- Normal response to exercise – heart receives more venous return, increase stroke volume (pumps more blood per beat), blood pressure rises, blood redistributed to perfuse exercising muscles
- Deconditioning diagnosed by maximal exercise test to measure peak oxygen uptake, which is reduced, low SV, relative tachycardia, abnormal vasomotor control, exercise intolerance





# Subthreshold Exercise

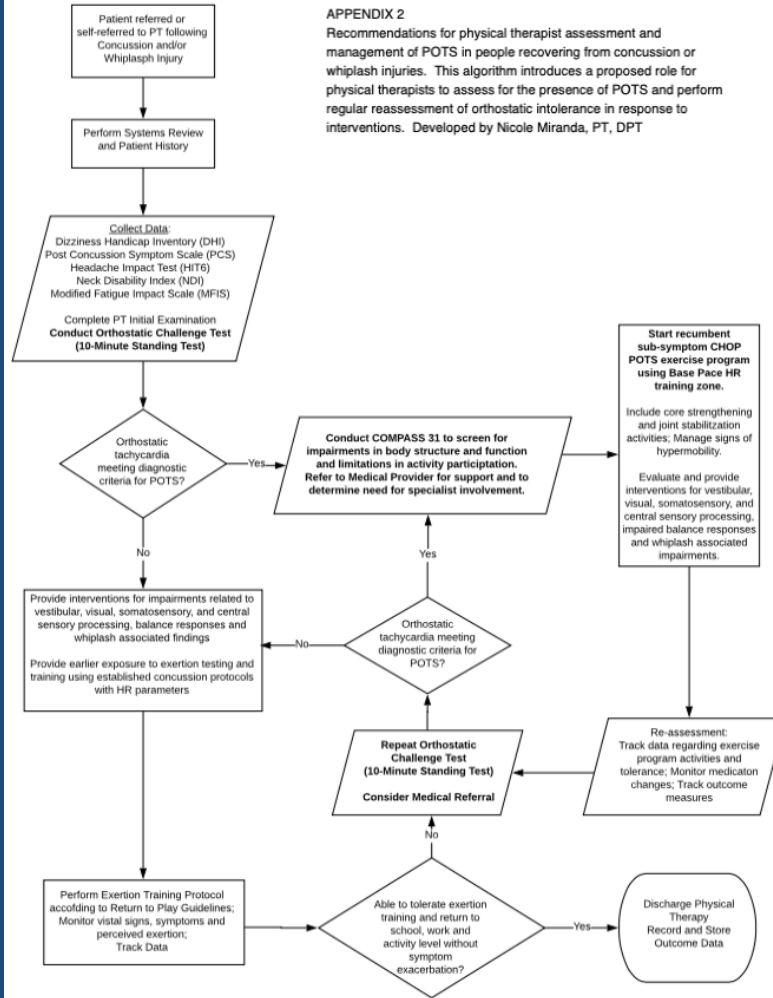
- Progressive exercise training → gradual increase
  - Blood volume
  - Exercise capacity
  - Stroke volume
  - Improved LV diastolic function





## APPENDIX 2

Recommendations for physical therapist assessment and management of POTS in people recovering from concussion or whiplash injuries. This algorithm introduces a proposed role for physical therapists to assess for the presence of POTS and perform regular reassessment of orthostatic intolerance in response to interventions. Developed by Nicole Miranda, PT, DPT





# Modify heart rate training zone calculations for POTS

- Resting HR determined by supine rest to determine heart rate reserve
  - $MHR - RHR - HRR$
- Maximal steady state
  - MSS training zone =  $0.75(HRR) + RHR \pm 5\text{bpm}$
- Begin exercise at 75-85% of MSS training zone
- Strengthening exercises to target postural control, core strength and joint stabilization are equally important to overcome decondition and MSK pain
- Incorporate balance and sensory processing activities

**Table 5. Progressive Exercise Training Modes<sup>51,62</sup>**

Mode 1	Any recumbent activity (rowing, cycle, stepper, kicking in a pool)
Mode 2	Upright stationary cycle
Mode 3	Treadmill walking 0% grade; elliptical trainer (arms stationary)
Mode 4	Treadmill walking with incline; elliptical trainer (arms moving); jogging 0% grade
Interval training	Initiate early interval training on recumbent equipment and gradually progress to upright postures





# Pearls

- POTS is multifactorial in etiology and may be accompanied by multi-system comorbidities
  - There may be overlapping autonomic dysfunctions in a patient, but orthostatic intolerance is the key in presentation
  - Common comorbidities include functional GI complaints, headaches/migraines and chronic pain, chronic fatigue, and MCAS
- Autonomic function testing can help us to identify the type of autonomic dysfunction
- POTS requires a multidisciplinary approach to treatment
  - Health care providers
  - Family members
  - Patient





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