

Does Complex Care Have to be That Complex? Perhaps More Information is Needed?

by

Pato M, DePace N, Plotnikoff G, Weintraub MI, Turner D, Manno R,
Munoz R, Colombo J

ABSTRACT

Here we review Complex Care, a multi-system, multi-discipline approach, from the point of view of autonomic, specifically, Parasympathetic and Sympathetic (P&S) nervous system monitoring. As per the National Institutes of Health [1996], the definition of health has evolved over time from an emphasis on morbidity and mortality to a multidimensional perspective that includes quality of life and productivity. Given that the P&S systems control or coordinate all other systems and organs of the body, the more information from P&S Monitoring simplifies the multidimensional perspective, promotes wellness thereby improving quality of life and productivity, while still addressing morbidity and mortality.

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INTRODUCTION

Complex care, by definition, seeks to improve health and well-being for people with complex health and social needs by coordinating and reshaping care delivery at the individual, community, and system levels. As per the National Institutes of Health, the definition of health has evolved over time from an emphasis on morbidity and mortality to a multidimensional perspective that includes quality of life and productivity ... “an emphasis on positive themes of happiness, social and emotional well-being” [1]. Similarly, the World Health Organization definition emphasizes the role of health as a resource for living that applies to individuals, groups, communities, or whole populations [2].

Complex medical conditions are frequently persistent in nature and require intermittent interventions to proactively identify and

resolve emerging problems and resolve acute exacerbations of illness or disability [3]. There is also a need for coordination of medical and other services to support and sustain an individual’s quality of life and productivity. Currently, a complex and comprehensive array of medical and other services is required to manage the multidimensional sequelae of populations with serious or complex medical conditions. Interventions are required not only to respond to the most immediate and acute threats to physical well-being, but also to prevent further complications and to initiate and sustain improvements in overall quality of life. Efforts to accomplish the latter require systems and services to address the long-term physical, social, and mental dimensions included in currently accepted definitions of health.

The latter has proven difficult, largely due to assumption and approximations based on

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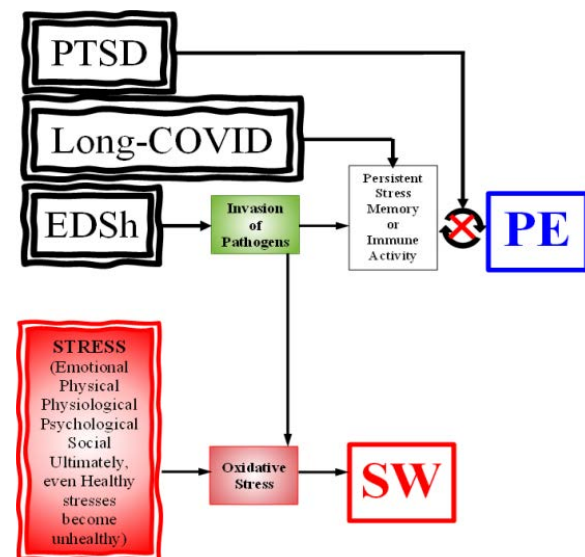
large population studies that still do not address the individual (*e.g.*, “personalized care”). As a result, the former has become focused on treating symptoms from a narrow view of the specialist, ignoring other symptoms that are not within the specialist’s purview. As a result, healthcare costs have continued to increase, especially as patients are perceived to grow more and more intolerant to medications, including due to (1) polypharmacy and all of its complications, and (2) complex patient access to healthcare due to current established protocols and regimens.

Our recent work with Long-COVID [4,5], Ehlers-Danlos Syndrome/Hypermobility Spectrum Disorder (EDSh) [6,7,8], as well as other conditions involving multiple systems, including: diabetes, hypertension, heart disease, infectious diseases, PTSD, Depression/Anxiety, etc. [9] has shown that more information is needed; especially in these cases where the patient seems resistant to treatment or is difficult to manage and is on or near maximum therapy. This information has proven to be available through monitoring of the activity of the Parasympathetic and Sympathetic (P&S) nervous systems [9,10] (see Figure 1 [10]). This is NOT the same as other available forms of Autonomic Nervous System monitoring which measure total ANS function, forcing assumption and approximation to hypothesize the activity of the two underlying P&S branches based on assumption and approximation. P&S monitoring provides mathematically independent and simultaneous measures of P&S activity, eliminating the assumptions and approximations. Given that the P&S together coordinate to control and coordinate all systems and functions of the body, one or two P&S dysfunctions often

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explain many symptoms. Further, treating these P&S dysfunctions often relieves the associated symptoms, reducing medication-load, the risks of polypharmacy and improving quality of life and productivity, improving patient outcomes and reducing healthcare costs to the individual and the nation [11,12,13,14].

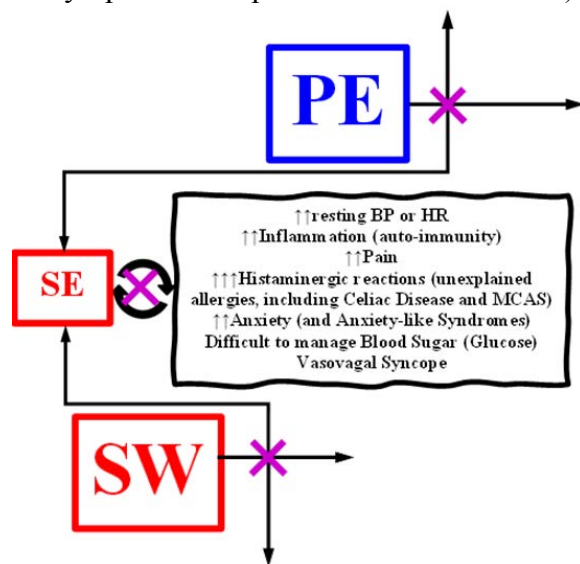
We know that Figure 1 is complicated, but this is how complicated your patients may be. Let us break it down. Ultimately, what this Figure states is that the symptoms of complex patients arise from only one or two P&S dysfunctions. In these cases, two (see insert below): 1) the Parasympathetic dysfunction Parasympathetic Excess (PE, a cholinergic response) and 2) the Sympathetic dysfunction Sympathetic Withdrawal (SW, an alpha-adrenergic or Sympathetic response). By treating these P&S dysfunction, low-and-slow, all other dysfunctions and symptoms are relieved. However, low-and-slow treatment takes time. Therefore, in the meantime there are treatments for the symptoms and intermediary causes of symptoms that help to promote compliance and perseverance.



Long-COVID, Post-Traumatic Stress Disorder (PTSD), and Ehlers-Danlos

Syndrome/Hypermobility Spectrum Disorder (EDSh) are selected as exemplar complex disorders from diverse areas of medicine, including Psychology. Whether through (1) persistent trauma or stress (*e.g.*, PTSD); (2) severe infection, trauma or stress (*e.g.*, Long-COVID); or (3) chronic disease, disorder, trauma or stress (*e.g.*, EDSh), PE may result, for it is a protective mechanism, but abnormal in a chronic state. PE may also lead to Vasovagal Syncope (which is known to be co-morbid with POTS in $\frac{1}{3}$ to $\frac{1}{2}$ of patients, depending on disease or disorder). Often, concurrently, whether directly due to whole body stressors or indirectly through disorders or diseases, Oxidative Stress (stress at the cellular level) presents and by damaging those Mitochondria in nerves, leads to SW which may lead to Orthostatic dysfunction (including POTS). Relieving PE & SW at this point relieves or even prevents most other symptoms. Again, PE & SW are treatable.

Central to Figure 1 and to the problem is Sympathetic Excess (SE, a beta-adrenergic or Sympathetic response – see insert below).



SE is what most providers see as the primary dysfunction when testing or analyzing these

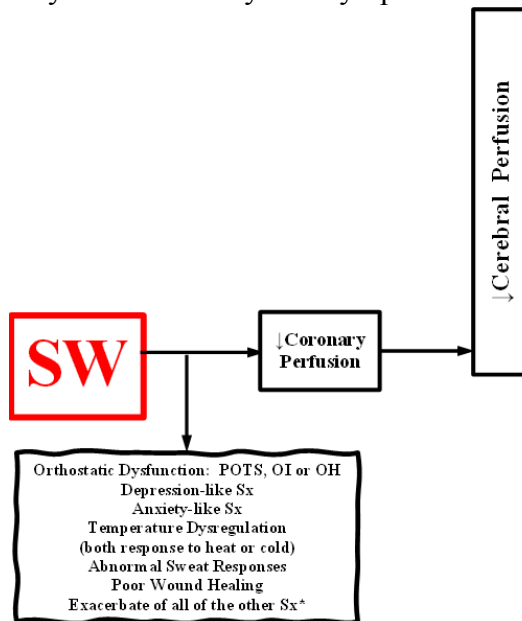
complex patients. SE may lead to high HR & BP, inflated and often chronic inflammation and histaminergic reactions, amplified pain and Anxiety-like symptoms, increased blood glucose, and Syncope including that it contributes to Vasovagal Syncope.

Granted SE is noteworthy as it will lead to Major Adverse Cardiovascular Events (MACE), including heart attack and stroke. However, if treated as a primary dysfunction when it is a secondary dysfunction or worse when it is a compensatory mechanism, then the treatment only adds to the problem and may accelerate the onset of MACE. The reason for this is that as a compensatory mechanism, the body uses SE to compensate for poor cerebral and possibly coronary perfusion due to SW & PE. With treatment for SE on board, attempting to reduce SE, the body then has to defeat the treatment as well to restore perfusion; adding more stress. Relieving PE & SW will ultimately relieve SE, and in the meantime (remember relieving PE & SW requires low-and-slow treatment) gently treating the symptoms of SE is often protective as the nervous system is brought into proper balance, both at rest and in response to challenge.

The remainder of Figure 1 lists symptoms, symptoms of P&S dysfunction, symptoms affecting the whole body, and all systems thereof. When a single trauma or chronic disease is affecting multiple systems, the P&S nervous system is implicated and a multi-system assessment should be considered. This not only helps to diagnose and develop proper treatment plans (given how the various systems are affected and the individual patient's history) but helps the provider to educate the patient as to how on trauma could cause so many, apparently disparate, disorders.

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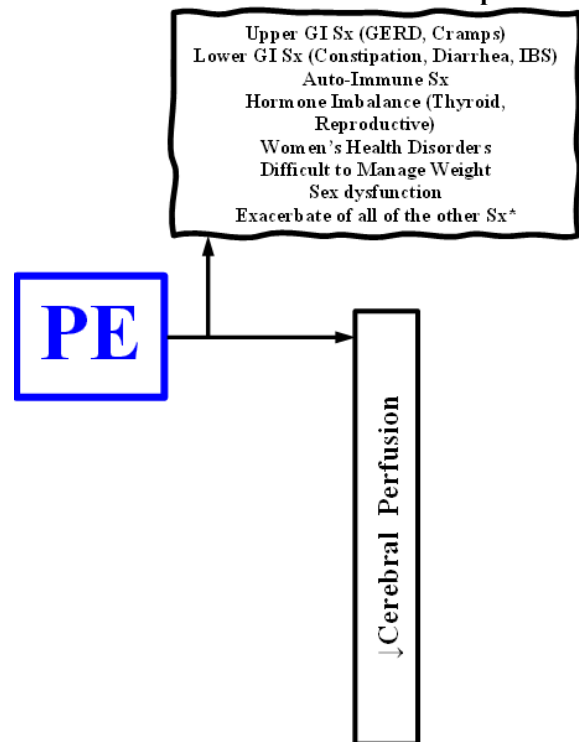
SW causes blood to pool in the lower extremities. As a result, SW leads to its own list of unique symptoms as shown in the insert below. It also contributes to poor coronary perfusion and poor cerebral perfusion. Blood pooling causes Orthostatic dysfunction, including POTS, Orthostatic Intolerance or Orthostatic Hypotension, depending on the HR or BP response. SW also contributes to depression-like symptoms, which with a further drop in BP may lead to anxiety-like symptoms. Blood



pooling also leads to poor peripheral circulation which may lead to poor wound healing and abnormal temperature control, including abnormal sweating. Of course, poor coronary perfusion will exacerbate all other symptoms as well.

PE also leads to its own unique set of symptoms (see insert below) and

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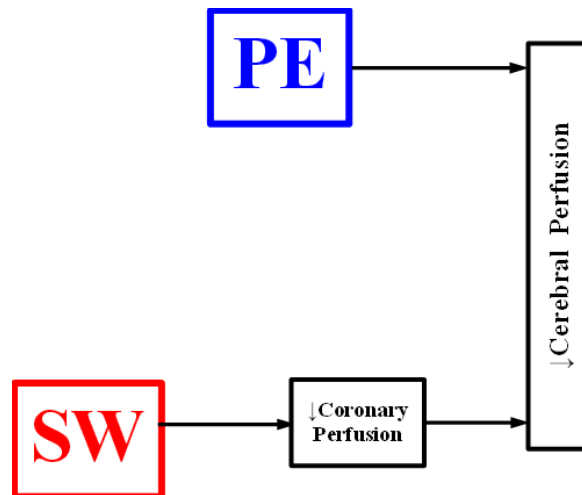
contributes to poor cerebral perfusion. PE may cause vasodilation and inefficient cardiac function, both contributing to poor cerebral perfusion. PE is also a component of Vasovagal Syncope. When this is co-morbid with POTS, patients are at risk for fainting. POTS is not the fainting risk, the Tachycardia with POTS prevents fainting.

The Parasympathetics also uniquely control or coordinate immune (including Mast Cell) activity, hormone function, and GI motility. As a result, GI upset, both upper and lower is a possible symptom. Auto-immune-like disorders are possible when PE maintains immune activity longer than is needed. Hormone dysfunction, including Thyroid and reproductive hormones. PE contributing to both hormone dysfunction and Mast Cell Activation Syndrome (MCAS) and given that the uterus is replete with Mast Cells, contributes to heavy and prolonged flows, which may cause cramping and Dysmenorrhea. The prolonged and heavy

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flows may cause Endometriosis which in turn may cause PCOS.

This is the connection between P&S dysfunction and these constellations of symptoms and poor cerebral perfusion (see insert below). Poor cerebral perfusion in turn (see following insert below) leads to

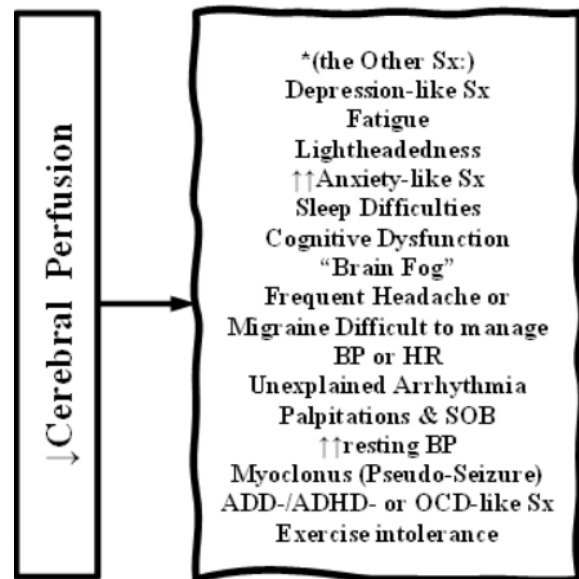


Fatigue, lightheadedness, cognitive dysfunction, frequent headache or migraine, brain-fog, sleep difficulties, sensory abnormalities (due to poor perfusion of the cranial nerves), Myoclonus, and apparent psychological disorders, including anxiety-like symptoms and symptoms of ADD/ADHD, OCD, and perhaps high-functioning Autism. These last five symptoms may be ways of the brain “calling for more blood” episodically via “adrenalin storms” that cycle the Anxiety-like symptoms, or continuously by causing persistent overactivity.

As PE & SW persist, (1) Oxidative Stress and (2) poor cerebral with or without poor coronary perfusion continue to cause more symptoms in different systems as the various systems begin to fail. Stress at the system level causes Oxidative Stress at the cellular level. Appropriate amounts of healthy stress cause healthy amounts of Oxidative Stress. However, like most things, excessive

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amounts of stress, even healthy stresses, are not healthy and lead to symptoms of Oxidative Stress, including and perhaps ultimately P&S dysfunction (see Figure 2



[15]). Again, with the involvement of the P&S nervous systems many systems of the body may be involved, compounded by the fact that the cells of the body may suffer mitochondrial dysfunction due to the Oxidative Stress, including the P&S nervous systems.

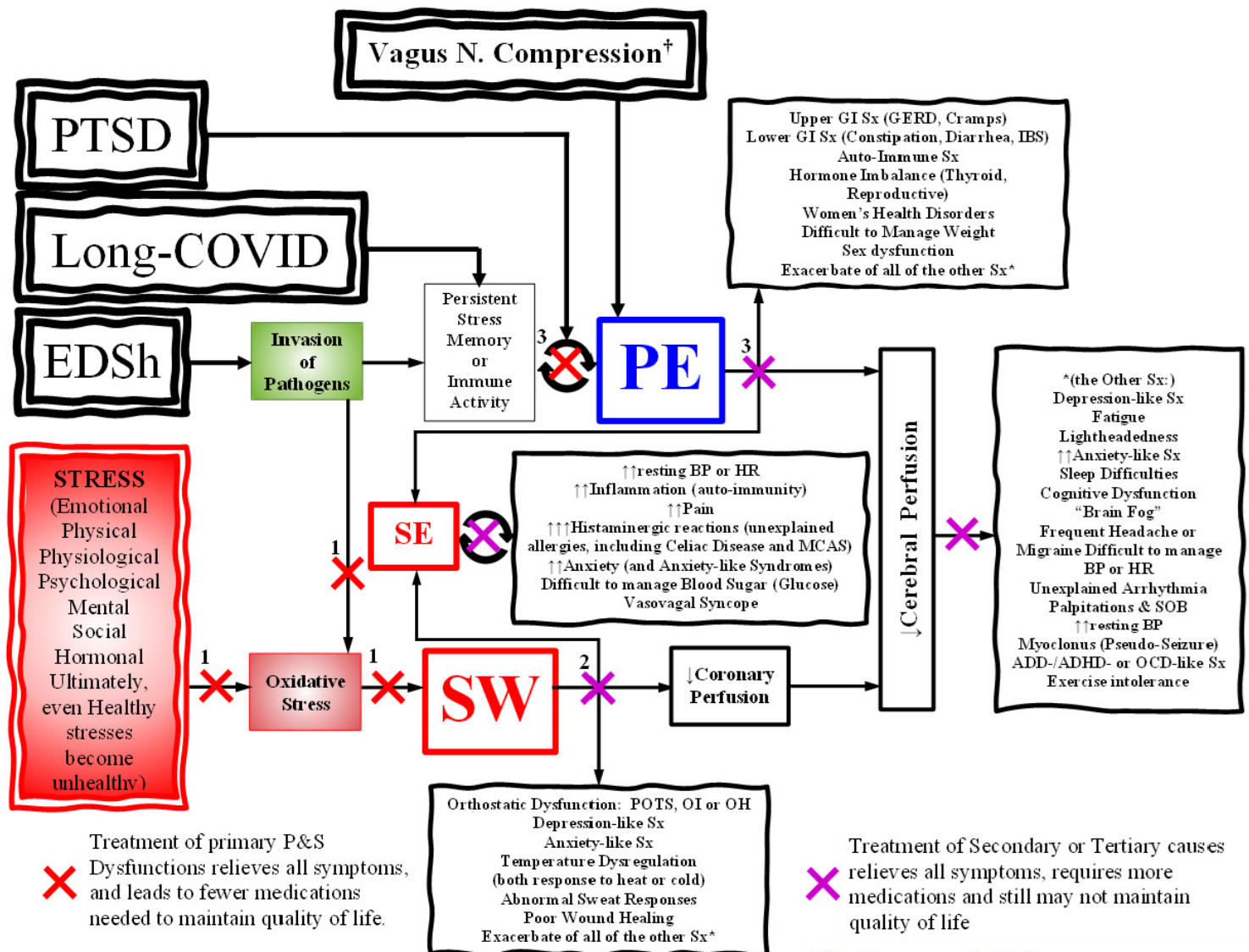
P&S dysfunction also leads to poor cerebral (including Cranial Nerves) and coronary perfusion, also contributing to the constellation of symptoms, again compounded by the fact that portions of the P&S are also not properly perfused, including the Vagus N. (Cranial N. X) and the command-and-control centers of the P&S system in the brain, mid-brain, and brain stem [16,17].

POTS

The Orthostatic dysfunction, causing blood to pool in the lower extremities, is the causal condition. The Tachycardia is a compensatory mechanism. It is the extra work needed to move more blood to the

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brain, typically due to hearts that have smaller stroke volumes. This is more common in younger females, especially before first pregnancy when the extra fluid-load naturally enlarges the heart in a normal



1. Anti-Oxidants (*i.e.*, r-Alpha Lipoic Acid (ALA) & CoQ-10)
2. Fluids, Electrolytes, & Vasopressors (*i.e.*, low dose Midodrine, *e.g.*, 2.5mg tid, titrated slowly)
3. Fluids, Electrolytes, & Anti-Cholinergics (*i.e.*, very low dose Nortriptyline, *e.g.*, rarely more than 10mg qd)

PE Parasympathetic Excess
SE βSympathetic Excess (*e.g.*, Syncope)
SW αSympathetic Withdrawal (*e.g.*, Orthostatic Dysfunction)

Sx: Symptoms

* The right hand most list of "the Other" Sx

† Compressed Discs, Cervicocranial Instability, Eagle Syndrome, Tissue laxity, Vagal N. Stimulation, etc.

Figure 1 depicts the causes of P&S dysfunction, including the two primary dysfunctions, PE & SW, and the secondary dysfunction, Sympathetic Excess (SE). At the bottom, possible therapy options are indicated. Rather than treating multiple symptoms (purple 'Xs') with multiple medications, potentially for life, the goal is to treat the primary P&S dysfunctions and once P&S balance is restored reduce medication-load as much as possible, except where end-organ defects present. [EDSh Book]

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manner. Ultimately, higher dose or prolonged beta-blocker treatment will exacerbate the condition as the body will either find ways to defeat the treatment or the patient will become more fatigued and exercise intolerant. The current issue with the Orthostatic dysfunction is the diagnosis paradigm. It is well known that $\frac{1}{3}$ to $\frac{1}{2}$ of all POTS patients have co-morbid Vasovagal Syncope (VVS). Note, fainting (overt Syncope) is not due to POTS, the Tachycardia prevents fainting, it is due to VVS. The feeling of fainting, *aka.*, near-Syncope, is possible with both. The problem is with most current monitoring techniques. Orthostatic dysfunction (*e.g.*, POTS) is an α -Sympathetic dysfunction and VVS (the Vagal component) is a Cholinergic dysfunction. Mathematically independent and simultaneous measures of P&S activity are required to differentiate P from S without assumption of approximation [9]. Current autonomic testing techniques are not able to do so. They only measure total autonomic activity and the current assumptions and approximations used to hypothesize underlying P&S activity are not appropriate for most POTS cases.

POTS is relieved by treating the Orthostatic dysfunction, the α -Sympathetic dysfunction. Once relieved, the Tachycardia is relieved, organically. As this may be a several month-long process, due to the fact that P&S treatment must be low-and-slow, the Tachycardia is treated as needed to protect the cardiovascular system and to help provide the patient a quality of life in the interim. If needed, the Parasympathetic dysfunction underlying VVS is also treated, concurrently, with low-dose anti-cholinergics. Once the P&S dysfunctions are relieved and the Autonomic Nervous System restored to proper (dynamic) balance, treatment may no longer be

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required (given the patient's history) given that the nervous system has a memory and is capable of maintain this new balance independent of treatment or therapy.

MCAS

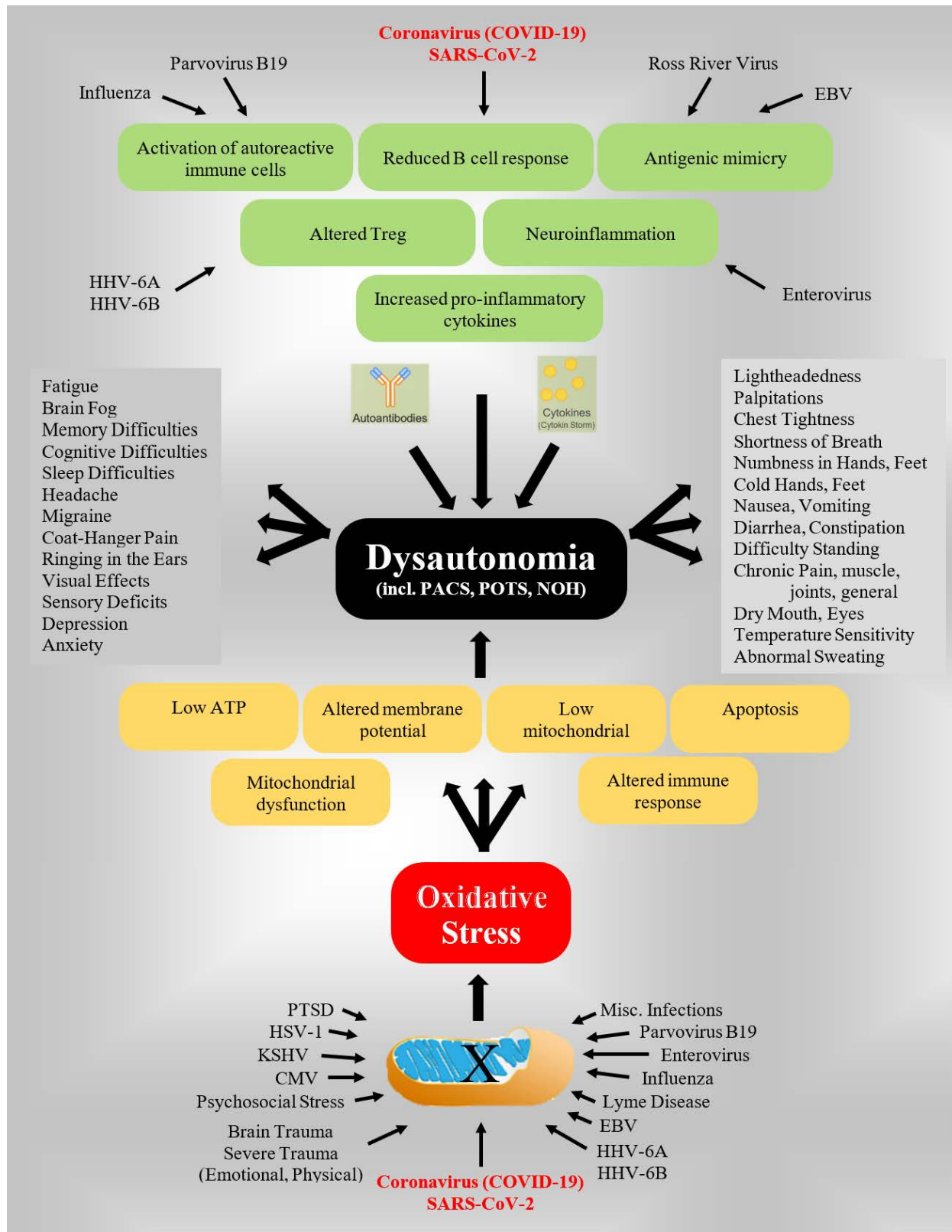
Mast Cells are part of the Immune System. It is well known that the Parasympathetic nervous system coordinates and controls the Immune System and provides it its memory (*e.g.*, this is how vaccines work). MCAS, whether it leads to (1) food allergies, including Celiac Disease; or (2) allergic reactions in general, including rashes, hives, or red-blotches on the skin especially after a hot shower; or (3) Women's health issues, including heavy or prolonged periods, blood clots, Dysmenorrhea, Endometriosis, or in some cases PCOS; etc., is at least exacerbated, if not caused by an inappropriately over-active Parasympathetic nervous system labeled as Parasympathetic Excess (PE).

The Parasympathetics release the Mast Cells. Then Sympathetics mediate Histamine release from the Mast Cells. With PE too many Mast Cells are released. PE also causes secondary Sympathetic Excess (SE). With SE (secondary to PE), too much Histamine is released from each individual Mast Cell. The result is an order of magnitude increase in the Histamine release. This is where unexplained rashes come from, or food allergies (which may be exacerbated by PE causing too much motility in the GI system), or women's health issues (which may be exacerbated by PE causing abnormal hormone levels, as the Parasympathetics also help to control and coordinate the Endocrine system).

Relieving PE with anti-cholinergic therapy prevents the cascade of reactions that lead to the symptoms of MCAS. Again, as this is a

low-and-slow process, treating MCAS in the interim as needed to protect the system and

to help provide the patient a quality of life



while PE, and therefore, MCAS is being relieved.

AUTO-IMMUNE DISORDERS

Remember, up to 60% of the immune system are not your own cells. These cells need to be coordinated with the body's own immune cells. Again, this is a function of the Parasympathetic nervous system. These cells that are not your own must be coordinated with the cells that are your own, so that they all live in a "peaceful" symbiotic relationship. PE helps to maintain an active immune system state. This may be protective, but it may be "over-protective." If immune cells remain active they remain "hungry." When they run out of the things they're supposed to "chew" on, the only thing left for them to "chew" on is ourselves. This then is the base definition of 'autoimmunity,' where our own immune system turns against our healthy cells. This is one of the possible results of PE.

In cases of hypothetical inflammatory autoimmune disorders without known or identified auto-antibodies, the true cause of these hypothetical conditions may simply be PE. In these cases, PE causes (1) an over- or prolonged-activation of the immune system and (2) a secondary SE. Since the Sympathetics mediate inflammation, SE may lead to excessive inflammation. Therefore, an overactive immune system that turns on the healthy cells of the body due to PE, and excessive inflammation due to SE, provides another possible mechanism for inflammatory autoimmunity without known or identified auto-antibodies. Anecdotally, many of our patients suspected of inflammatory autoimmunity without known or identified auto antibodies have been relieved of their autoimmunity once PE is relieved.

HORMONE DYSREGULATION

It is well known that P&S dysfunction in Hypothyroidism is common. Most of the cardiovascular and neurological manifestations of Hypothyroidism are due to P&S dysfunction. The published major findings are that Sympathetic dysfunction was observed more as compared to Parasympathetic dysfunction [18]. While we see this with most standard forms of autonomic monitoring, it is well known that the Parasympathetics control the hormones. With P&S monitoring we find that PE in response to Sympathetic challenges, causing secondary SE [19] is highly correlated with hormone abnormalities, including Hypothyroidism. Furthermore, our clinical evidence has shown that a significant reduction in Thyroid-hormone replacement therapy is associated in most patients with relief of PE. Therefore, PE promotes Thyroid dysfunction. In turn, Thyroid dysfunction promotes dysfunction in other hormone function, especially reproductive hormones, especially in females.

The connection between the P&S, Mast Cells, Histamine and hormones is that P&S controls and coordinates the other three, through feedback mechanisms. Estrogen stimulates Mast Cell degranulation (Histamine release from Mast Cells) and down-regulates the Diamine Oxidase²⁰ enzyme that clears Histamine. At the same time, Histamine stimulates the Ovaries to make more Estrogen. The net result can be a vicious cycle (see insert, right [21]). This may be exacerbated by PE causing more Estrogen to be released and more Mast Cells to be released, as well as secondary SE causing more Histamine release from each Mast Cell. This leads to an excessive inflammatory response which increases and

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prolongs blood flow. In addition, Estrogen dilates the vasculature enabling greater blood flow. Excessive Histamine release may also promote digestive issues, fatigue, acid reflux, weight gain, hair loss, trouble sleeping, and anxiety.

Women tend to have more Histamine than men because women have more Estrogen. Estrogen, Progesterone, and Histamine are closely linked in the body. Estrogen stimulates mast cells to make more Histamine. This may be why women may experience more Histamine issues at certain times in their cycle – likely when Estrogen levels are higher than Progesterone levels. Women with PE and SE tend toward the more symptomatic end of the spectrum due to Mast Cell over production due also to PE exacerbated by overly excessive Histamine release due also to SE caused by the PE; and all being further exacerbated by PE affecting Thyroid function and reproductive hormone production. Low thyroid levels can also contribute to Histamine issues [22]. This is likely due to Thyroid hormones that help regulate Mast Cells and reduce Histamine production. If reduced levels of Thyroid hormone, Histamine levels may be much higher.

SLEEP DIFFICULTIES

While it is true that the P&S also coordinates sleep, the typical P&S dysfunction that leads to sleep difficulty are those that cause poor cerebral perfusion: PE & SW. With poor cerebral perfusion, the brain “is half a sleep all day.” As soon as the patient lays down to sleep, the brain is properly perfused and “wakes up and wants to play.” The patient wants to sleep. Our patients report that they are more alert while trying to sleep and typically make lists in the heads of the things they want or wanted to do. However, those lists disappear as soon

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as they sit up upon waking. Or they go to the bathroom more than two or three times a night, but the additional trips produce nothing. Again, the brain “is playing, and wants to go for a ride.” To do so it makes the patient feel like they have to use the bathroom. Restoring proper brain perfusion helps to naturally stimulate the brain during the day and promotes normal brain function at night promoting restful and restorative sleep.

AMPLIFIED PAIN

Pain is mediated by the Sympathetics. SE amplifies the pain perception. SE may be a primary dysfunction or secondary to PE. If the latter, such as in CRPS, PTSD, Hypermobility etc., PE further amplifies the pain perception by exacerbating SE. Neuropathic pain transmission is a complex phenomenon which involves profound changes in the peripheral and central neural processing of afferent input. It arises from a pathological lesion or damage to the somatosensory system. Nociceptive neurons also change their responsiveness as a result of injury or disease. They may display a lowering of the activation threshold to normal and even sub-threshold inputs. The complexity of pain transmission circuitry means that pain conditions that are currently poorly understood will ultimately be explained without resorting to psychopathological etiology. Pain that is non-anatomical in distribution, pain that spreads to non-injured territory, pain that is said to be out of proportion to the degree of injury, and pain in the absence of injury have all, at one time or another, been used as evidence to support the idea that psychological disturbance underlies the pain. Notably, each of these features once thought to be caused by psychopathology can now be explained by peripheral and

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central neurophysiological mechanisms that have gone awry, including P&S dysfunction involving PE.

MYOCLONUS

Patients with poor cerebral perfusion often present with Epilepsy-like symptoms, including essential tremor. P&S dysfunction may include high or low blood sugar (Hyperglycemia or Hypoglycemia, respectively) which may also cause tremors or other involuntary movements. Stress, anxiety, or fatigue, which may also involve P&S dysfunction and may also be associated with tremors. Without positive evidence of Epileptic seizure activity on EEG or ambulatory EEG these cases should be investigated for P&S dysfunction as cases with Myoclonus.

SYMPTOMS OF PSYCHOLOGICAL DISORDERS

Depression-like symptoms may be caused by either poor cerebral perfusion or PE. Anxiety, ADD, ADHD, OCD, and possibly PTSD and Autism may all be compensatory mechanisms resulting in hyper-active brain function as a means for increasing cerebral perfusion. As a reaction to poor cerebral perfusion, to prevent lightheadedness, near-Syncope and Syncope, the brain may cause adrenaline storms as an attempt to increase cerebral perfusion. These storms cycle anxiety-like symptoms for the duration of the storm, including shortness of breath, palpitations, and chest tightness. They may also lead to panic attacks. All as increased brain activity responses as a means of temporarily demanding more perfusion. Higher, prolonged, or continuous increases in brain activity responses may mimic other brain syndromes that involve higher, prolonged, or continuous hyper-activity to constantly work to increase cerebral

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perfusion, including ADD, ADHD, OCD, and possibly PTSD and Autism. Diagnoses of Depression, Anxiety, ADD, ADHD, OCD, PTSD, and Autism, and related syndromes, should first include P&S monitoring to rule out any contribution from poor cerebral perfusion [12,23].

DISCUSSION

It is important to know and convey to patients the good news that all P&S dysfunctions are treatable [10,24]. Moreover, the patient must be confirmed and affirmed that P&S Dysfunction (*aka.*, Autonomic Dysfunction or Dysautonomia) is NOT a psychological condition. It is an organic condition. While most symptoms a patient with P&S dysfunction experiences may be traced to the P&S nervous systems, it is still highly recommended that other causes of symptoms are ruled out. To this end, multi-disciplinary consulting is recommended. In addition to frequent P&S monitoring, the multi-disciplinary approach may involve: (1) testing for pathogenic insults, including COVID (causing long-COVID), Lymes, other bacterial, mold, or mildew insults, and the like, and serious or prolonged stresses, including PTSD (see Figure 2); (2) Rheumatology to rule out EDSh and other collagen and inflammatory disorders or other Rheumatologic disorders that may masquerade as auto-immune diseases (an example of a chronic condition that will require P&S maintenance therapy for life to help maintain balance against the disorder); (3) EKGs, echocardiogram and vascular ultrasound monitoring from Cardiology, as well as blood work, arrhythmia monitoring to ensure anxiety-like symptoms are not more, and remote BP monitoring for Orthostatic dysfunction or Syncope; (4) Gastroenterology for upper and lower GI disorders; (5) Urology,

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Urogynecology, Gynecology, and Obstetrics for bladder and Women's Health issues; (6) Neurology to rule out central nervous system disorders; (7) Endocrinology to ensure Thyroid and other hormone balance and health; (8) Pulmonology to rule out any respiratory disorders underlying shortness of breath; (9) Psychiatry for more severe affective disorders; (10) Allergy Specialist for more severe drug or food reactions; (11) Low-and-Slow Physical Therapy is usually helpful in pain management. In addition, stress reduction is important, whether that is in the form of Yoga or meditation. Some patients also look to alternative medical approaches, including Nutritionists, Natural Medicine, Holistic and Herbal Medicine, Metabolic Medicine, etc.

IN SUMMARY

P&S Management starts with a comprehensive personal and family history including clinical assessment for history of chronic disease or trauma of all sorts: mental, emotional, physical, physiological or psychological. The personal history should investigate all bodily systems to obtain a complete picture. Remember these are complex cases and the P&S nervous systems affect all systems and organs of the

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body. An investigation of the initial trigger for symptoms may help to understand what stresses and triggers to avoid or treat if possible. It may also help in demonstrating the cumulative effect of stresses and triggers in the presentation of symptoms (like concussions or multiple viral infections). This level of detail helps the patient and patient's family and loved ones, and even their providers, to understand the complexity of their disorder.

FINAL PEARLS

With direct measures of the P&S nervous systems complex care is less complex. The many conditions, heretofore thought to be independent, are mostly if not all symptoms of P&S dysfunction (see Figure 1) – and all P&S dysfunctions are treatable. The underlying PE & SW leading to poor coronary and cerebral perfusion, with Oxidative Stress leading to Mitochondria dysfunction explains most if not all symptoms of complex care patients. Again, and it cannot be stressed enough, all P&S dysfunctions are treatable, assuming the individual P&S branches of the autonomic nervous system are measured independently and simultaneously.

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