Overview of the Autonomic Nervous System

The nervous system comprises the central nervous system (CNS) and the peripheral nervous system (PNS) components. The peripheral nervous system (PNS) is composted of three parts: the somatic, autonomic and enteric. The somatic portion functions to control voluntary movement, while the autonomic and enteric function involuntarily to control various bodily functions. The autonomic nervous system (ANS) controls homeostasis of the entire body, which includes the cardiovascular activity, digestion, urination, sexual function, sweating, sleep, and ability to maintain upright position. Many physicians, including ourselves, consider the enteric nervous system (ENS) as actually a part of the ANS, and it is the most independent functioning of the components of the ANS. It controls gastrointestinal function (secretion and peristalsis).

The ANS is separated into a central and peripheral part, even though it is considered part of the peripheral nervous system (PNS). The ANS is regulated by the hypothalamus and the central autonomic network (CAN) and is located predominately in the forebrain and brain stem. Therefore, the "mind to body" connection of the ANS operates from central to peripheral. The hypothalamus is the highest level of ANS integration under the control of the limbic (emotional influence) and cortical (voluntary influences) structures. The ANS controls respiration, cardiovascular heart rate regulation, blood pressure regulation (vasomotor) and reflexes such as swallowing, sneezing, vomiting and coughing. There are two major branches (three if we include the ENS): the sympathetic nervous system (SNS) and parasympathetic nervous system(PNS). The SNS is the "fight or flight system" or the "Accelerator of the body." The PNS acts as the "Brakes of the body," or the "rest and digest" portion. Some have coined it the "feed and breed" system.

It is simplistic to state that the SNS excites and the PNS inhibits bodily functions since there are exceptions. The "Accelerator" and "Brakes" concept of SNS and PNS help the patient understand better the symptoms they are experiencing when an imbalance between the SNS and PNS occurs (dysautonomia). Dysautonomia is a condition in which the ANS is not functioning properly, that is it is not in balance. The two arms, the PNS and the SNS can be equated to being on a seesaw where both ends of the seesaw should be level. There are various entities in which one is higher then the other on the seesaw, or at times the seesaw gets broken where both ends are up or both ends are broken.

Dysautonomia affects the function of the heart, intestine, bladder, pupils, sweat glands, blood vessels and virtually all organs of the body as the ANS supplies innervation of the smooth muscles and glands throughout the body.

The ANS is the regulator of cardiovascular function, arterial baroreceptor reflex, standing response, sweating, gastrointestinal tract motility and sleep-cycle. Imbalance, or malfunction of the ANS may cause hyperhidrosis and hand hidrosis, dizziness and orthostatic intolerance, and even syncope, high or low heart rates, blood pressure fluctuations, urinary incontinence and retention, insomnia, and swallowing difficulties.

Common symptoms of dysautonomia cause exercise intolerance, chronic fatigue, muscle aches and pains, diffuse sleep, tunnel vision, weakness, blurred vision, constipation, diarrhea, and brain fog. Word-finding difficulties and short-term memory lapses can occur. Gastroparesis is also a common and disabling problem.

Most common symptoms are due to orthostatic intolerance. Standing is a major challenge for a healthy ANS. Gravity causes venous pooling in the lower extremities and lowers central pressures and venous return to the heart. The normal functioning ANS activates the SNS and withdraws the PNS on immediate stand to increase venous return to the heart and avoid venous pooling. SNS constricts the lower extremity vessels and promotes blood flow centrally to the heart. PNS (Parasympathetic) take out) withdraw will increase the heart rate on the initial stand response. In orthostatic intolerant (OI) individuals, due to dysautonomia, this mechanism is defective. Sympathetic withdraw inappropriately occurs, blood pools in the legs, and cardiac output and venous return are impaired. The patient may have a drop in blood pressure or orthostatic hypotension and an inappropriate increase in heart rate, overcompensating as in postural orthostatic tachycardia (POTs), or simply brain fog, fatigue, and exercise intolerance without significant changes in blood pressure or heart rate. The latter is more commonly seen in patients, especially younger patients at presentation. It is estimated that 40 million people in the United States have OI, and this is a major cause of disability and may be responsible for many of the symptoms that we classify as fibromyalgia-type and many of the symptoms we classify as chronic fatigue. Orthostatic intolerance and chronic fatigue occur simultaneously.

To treat various forms of dysautonomia including OI, pharmacology along with lifestyle changes, diet and salt intake, physical maneuvers and compression garments, fluids, stress reduction techniques and neural feedback may be indicated depending on the patient's symptoms and type of dysautonomia. Experimental pacing and stimulating devices have often been used in various circumstances. Surgical procedures occasionally are indicated, for example, bilateral sympathectomies and various hyperhidrosis states. Patients with hypermobility syndrome, such as Ehlers-Danlos syndrome (EDS) and other related disorders are often a significant challenge and occasionally are referred for orthopedic or neurosurgical interventions. Gastroparesis is particularly troublesome, as there may be mixed components of imbalance in the autonomic nervous system affecting the GI tract as compared to other organs, and one pharmacological agent instituted to treat one organ disorder may adversely affect the other. Therefore, pharmacologic treatment of dysautonomia is very complex, and not always successful. However, one must get at the "root of the problem." Increased oxidative stress is often the real trigger event for causes of mechanism at the cellular level. Deficiency of ATP production and mitochondrial dysfunction leads to significant impairment of neuron function and abnormalities of myelin sheath (neurons are very rich in mitochondrial density). Also, small fibers (the type C fibers, which are sensory in ANS fibers) are susceptible to oxidative stress. Nitric oxide deficiency also contributes to many of the neurological and vascular features of autonomic function and dysfunction. A "cocktail" of antioxidants, antiinflammatories and nitric oxide-enhancing agents have been advocated and may be useful empirically, especially at appropriate titrated doses.

When a patient presents to our autonomic center with symptoms consistent with dysautonomia, they have usually seen a minimum of 14 physicians and a diagnosis has not been made. In fact, most patients arrive at the diagnosis by doing their own independent research on the Internet. Many patients are referred to us via Internet research and not by direct physician referral. One patient, an Indian/Asian young male stated that he had seen 99 physicians prior to seeing us. These patients with dysautonomia have various presentations. They have oftentimes been labeled with the diagnosis of anxiety and have not had any types of autonomic testing tests performed, such as sweat testing (sudomotor) or heart rate variability (HRV) testing, which are easy noninvasive tests to do in the outpatient center. Most have not had a Tilt Test performed. There is an obvious problem with identifying these patients due to physicians not being educated in this area sufficiently, both in medical school and within the medical literature. While autonomic dysfunction has in the past been the domain of the neurologist, the cardiologist is also beginning to learn more about it, as many of the diagnostic tests have a cardiac basis, and many of the patients present to a cardiologist with shortness of breath, chest pain and palpitations, which are a manifestation of autonomic dysfunction and not intrinsic cardiac disease. In addition, endocrinologists are beginning to have more awareness of autonomic dysfunction, especially in the diabetic population.

In this text, we detail the symptoms and diagnosis of dysautonomia and the universal mechanism at the cellular level that causes or contributes to autonomic dysfunction. We propose a six-prong treatment approach that has been useful empirically in clinical practice. This involves not simply pharmacology and physical measures and maneuvers, but also diet, an antiinflammatory approach an antioxidant approach, and a stress reduction approach. An integrative approach to the treatment of dysautonomia should be holistic. It is our goal to stimulate further research in this area and further education and recognition of this disease entity. Not just physicians and healthcare workers, but the public needs to be educated as to the existence and prevalence of dysautonomia and the morbility and treatment entailed.

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