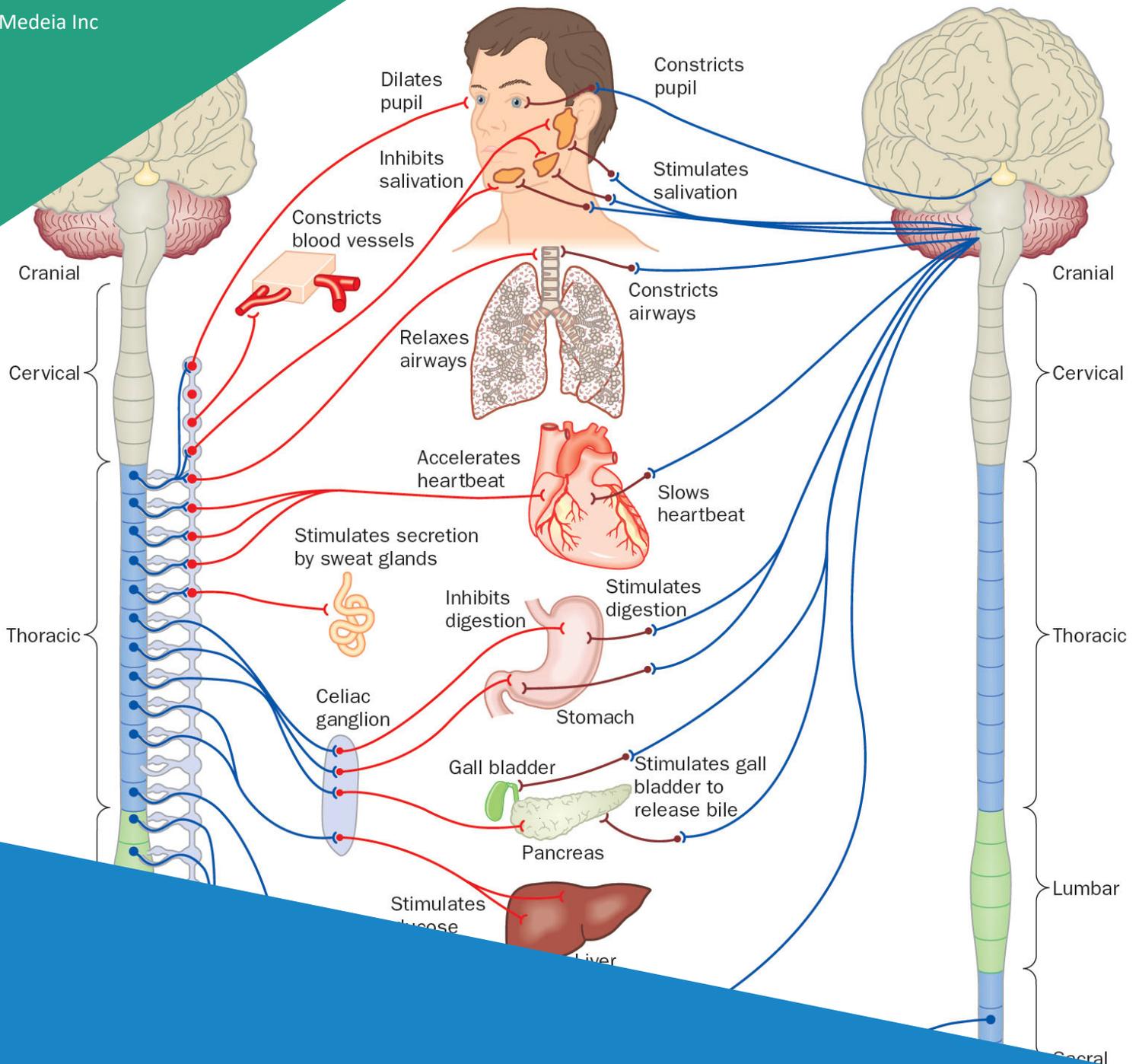


VitalScan Plus

Medeia Inc



Autonomic Dysfunction Assessment

In 15 Minutes Identify Patients at Risk

The VitalScan diagnostic test provides next generation non-invasive patient diagnostics within minutes. The FDA cleared equipment is used by physicians to help identify diseases that may be associated with Autonomic Nervous System, Vascular and Sdomotor disorders.

VitalScan Plus Benefits

VitalScan performs non-invasive tests to assess the patient's risk of autonomic nervous system & arterial dysfunction related to disorders and disease complications.

ANS Risk Assessment

- Sudomotor Function
- Autonomic Regulation
- Cardiac Autonomic Neuropathy

Arterial Risk Assessment

- Arterial Stiffness
- Peripheral Artery Disease

Sudomotor (or sweat motor) Function is related to the nerve fibers controlling the activity of the sweat glands (the post sympathetic cholinergic nerve fibers or C-fibers). Sudomotor dysfunction (sweat dysfunction) is an early indicator of small fiber neuropathy. Traditional neurophysiologic measurements of sudomotor function include invasive testing such as thermoregulatory sweat testing (TST), silicone impressions. VitalScan Sudomotor+ uses quantitative sudomotor axon reflex testing (QSART) and Galvanic Skin Response (GSR) to test Sudomotor Function, which are non-invasive tests.

Autonomic Regulation is the body's ability to maintain homeostasis (stability and balance) during internal and external stimuli. Autonomic Regulation is always functioning, and we are often unaware of the important tasks it is performing. When the nerves that control Autonomic Regulation are damaged, Autonomic Dysfunction can develop. Autonomic Dysfunction can be temporary or chronic. Diabetes and Parkinson's disease are two examples of chronic conditions that can lead to Autonomic Dysfunction. VitalScan ANS+ tests Autonomic Regulation through a combination of Heart Rate Variability (HRV) Assessment and Cardiac Autonomic Reflex Tests (CARTs).

Cardiovascular Autonomic Neuropathy (CAN) is a common form of autonomic neuropathy, causing abnormalities in heart rate control and central/peripheral vascular dynamics. CAN has a strong link to diabetes and can contribute to the development of a variety of severe conditions resulting in a multitude of complications and higher mortality risk. Like the Autonomic Regulation Assessment, VitalScan ANS+ evaluates the Sympathetic and Parasympathetic Nervous Systems using CARTs to identify CAN risk factors.

Arterial Stiffness occurs as a consequence of biological aging and Arteriosclerosis, which occurs when arteries become thick and stiff, sometimes restricting blood flow to your organs and tissues. Arterial Stiffness is associated with an increased risk of cardiovascular events such as heart attack and stroke, the two leading causes of death in the developed world. Depending on the cause, Arterial Stiffness may be treated and prevented. VitalScan Vascular+ evaluates Arterial Stiffness using Pulse Wave Velocity (PWV), Peripheral Augmentation Index (AI), and Central Aortic Systolic Pressure (CASP).

Peripheral Artery Disease (PAD) is a common manifestation of atherosclerotic vascular disease where the arteries in your legs or arms are narrowed or blocked. Its incidence increases with age and in the presence of known cardiovascular risk factors (e.g., smoking and diabetes). People with PAD are at an increased risk of heart attack, stroke, poor circulation and leg pain. VitalScan Vascular+ evaluates PAD using the Ankle Brachial Index (ABI). The ABI compares your systolic blood pressure measured at ankle with systolic blood pressure measured at the arm. A low ABI can be a strong indicator of PAD and risk of circulatory problems.

The VitalScan Measures 8 Risk Factors

The VitalScan device is a powerful analysis tool in managing your patients' health. Scientifically validated and FDA cleared, this medical device performs a range of tests covered and reimbursed by most insurance companies. This system is fast, non-invasive and takes less than 11 minutes to complete an assessment.

The one-page Physician Dashboard provides a comprehensive overview of a patient's health at-a-glance. The analysis system provides patient insights covering 8 key risk factors that are described in the pages that follow. Depending on the risk score for each factor, you will be able to determine the best course of action to resolve the patient's condition as well as motivate your patient to immediate action.

AUTONOMIC NERVOUS SYSTEM DYSFUNCTION RISK

Problems with the ANS can range from mild to life threatening. Sometimes only one part of the nervous system is affected. In other cases, the entire ANS is affected. Some conditions are temporary and can be reversed, while others are chronic and will continue to worsen over time. Chronic diseases such as Diabetes or Parkinson's disease can cause irregularities with the ANS. Problems with ANS regulation often involve organ failure, or the failure of the nerves to transmit a necessary signal.

ENDOTHELIAL DYSFUNCTION RISK

Current evidence suggests that endothelial function is an integrative marker of the net effects of damage from traditional and emerging risk factors on the arterial wall and its intrinsic capacity for repair. Endothelial dysfunction, detected as the presence of reduced vasodilating response to endothelial stimuli, has been observed to be associated with major cardiovascular risk factors, such as aging, hyperhomocysteinemia, post menopause state, smoking, diabetes, hypercholesterolemia, and hypertension.

SUDOMOTOR DYSFUNCTION RISK

Sudomotor dysfunction testing may indicate to physicians of a patient's peripheral nerve and cardiac sympathetic dysfunction. Neuropathy is a common complication in diabetes mellitus (DM), with 60%–70% of patients affected over lifetime. Symptoms of neuropathy are very common, and subclinical neuropathy is more common than clinical neuropathy. Neuropathy may remain undetected, and progress over time leading to serious complications. The most common associated clinical condition is peripheral neuropathy, affecting the feet. Autonomic nerve involvement is common but probably the most undiagnosed. Low scores in the sudomotor may lead a medical provider to look at clinical neuropathy.

INSULIN RESISTANCE RISK

Insulin resistance is defined clinically as the inability of a known quantity of exogenous or endogenous insulin to increase glucose uptake and utilization in an individual as much as it does in a normal population. Insulin resistance occurs as part of a cluster of cardiovascular-metabolic abnormalities commonly referred to as "The Insulin Resistance Syndrome" or "The Metabolic Syndrome". This cluster of abnormalities may lead to the development of type 2 diabetes, accelerated atherosclerosis, hypertension or polycystic ovarian syndrome depending on the genetic background of the individual developing the insulin resistance.

PLETHYSMOGRAPHY CARDIOVASCULAR DISEASE RISK

The PTG CVD risk factor is the combined total of the other seven risk factors assessments. It takes into consideration the cardiovascular, as well as, the autonomic nervous system (ANS) measurements.

SMALL FIBER NEUROPATHY RISK

A small fiber neuropathy occurs when damage to the peripheral nerves predominantly or entirely affects the small myelinated fibers or Unmyelinated C fibers. The specific fiber types involved in this process include both small somatic and autonomic fibers. The sensory functions of these fibers include thermal perception and nociception. These fibers are involved in many autonomic and enteric functions.

CARDIOMETABOLIC RISK

The specific factors that can cause this increased risk include: obesity (particularly central), hyperglycemia, hypertension, insulin resistance and dyslipoproteinemia. When patients have one or more risk factors and are physically inactive or smoke, the cardiometabolic risk is increased even more. Medical conditions that often share the above characteristics, such as type 2 diabetes, can also increase cardiometabolic risk. The primary focus of cardiometabolic risk treatment is management of each high-risk factor, including dyslipoproteinemia, hypertension, and diabetes. The management of these subjects is based principally on lifestyle measures, but various antihypertensive, lipid-lowering, insulin sensitizing, anti-obesity and antiplatelet drugs could be helpful in reducing cardiometabolic risk.

CARDIAC AUTONOMIC NEUROPATHY RISK

High chronic condition over a period of years may cause a cardiac autonomic neuropathy. This is damage to the nerves that control the regulation of involuntary function. When the nerve damage affects the heart, it is called cardiac autonomic neuropathy (CAN). CAN encompasses damage to the autonomic nerve fibers that innervate the heart and blood vessels, resulting in abnormalities in heart rate control, vascular dynamics and the body's ability to adjust blood pressure. CAN is a significant cause of morbidity and mortality associated with a high risk of cardiac arrhythmias and sudden death.

Clinical Benefits of VitalScan Testing

Peripheral Small Fiber Neuropathy

Frequently associated with Diabetes, Pre-Diabetes and Metabolic Syndrome. Other conditions may include: HIV, Guillain-Barre Syndrome, Celiac Disease, Hepatitis C, Restless Leg Syndrome, Abnormal Thyroid Function.

Cardiac Autonomic Neuropathy (CAN)

High blood glucose levels over a period of years may cause CAN. Nerves that signal the heart to beat faster are called Autonomic Nerves. When nerve damage affects the heart, it is referred to as CAN. CAN is not a disease of the heart; it is a problem with the nerves that control heart rate.

Cardiovascular and Peripheral Artery Disease

Atherosclerosis is the most common cause of CVD. Plaque buildup thickens and stiffens artery walls which inhibit blood flow through arteries to organs and tissues. In the heart it is known as coronary artery disease and in the legs it is known as peripheral arterial disease (PAD). CVD and PAD can be caused by correctable problems, such as an unhealthy diet, lack of exercise, being overweight and smoking.

Early detection for Endothelial Dysfunction

Endothelial dysfunction is a well-established response to cardiovascular risk factors and precedes the development of atherosclerosis. When cardiovascular risk factors are treated the endothelial dysfunction may be reversed. It is an independent predictor of cardiac events.

Cardio-Metabolic Risk Markers (CMR)

The combination of VitalScan tests provides important Cardiometabolic Risk Measurements (CMR): a set of results used to assess the cluster of risk factors which are good indicators of a patient's overall risk for type 2 diabetes and cardiovascular disease (CVD). CMR focuses clinical attention on the value of systematic evaluation, education, lifestyle and behavior changes, disease prevention, and treatment. Assessing CMR provides a more comprehensive picture of a patient's health and potential risk for future disease.

Adjunct in Diabetes Treatment

Early detection of Peripheral Small Fiber Neuropathy and Cardiac Autonomic Neuropathy.

ANS Diagnostic Applications

CARDIOLOGY, ENDOCRINOLOGY, INTERNAL MEDICINE, NEUROLOGY, ONCOLOGY AND PULMONOLOGY

CARDIOLOGY

Hypertension: ANS monitoring resolves at least two subpopulations of hypertensives: 1) primary hypertensives and 2) patients with Paradoxical Parasympathetic Syndrome (PPS) and secondary hypertension. If detected early, while there is still power or tone in the sympathetic nervous system (SNS), hypertension is foreshadowed (before blood pressure (BP) increase) or indicated with elevated or high BP by a hypersympathetic condition.

However, there are two reasons for the SNS to be hyper: 1) it is hyper on its own, or 2) it is driven high by the parasympathetic nervous system (PSNS) due to PPS. The first, primary hypertension or primary hypersympathetics (prior to BP elevation), is treated as usual. In the case of primary hypersympathetics, however, lower dosing and short term therapy should be considered.

Treating the second, PPS with secondary hypertension or hypersympathetics like the first will only serve to strengthen the PPS (by further suppressing the sympathetics) and further destabilize the patient. PPS with secondary hypertension or hypersympathetics must consider the PSNS first and then, if hypertension or hypersympathetics remain, follow with primary hypertension therapy as above. To treat the PSNS first there are two approaches: 1) Amytriptaline or Nortriptaline (10 to 12.5 mg at dinner titrated up to 25 mg bid) which have centrally acting anti-cholinergic side effects, and they reduce the limbic input to the medullary autonomic nuclei (these are useful if the patient also presents with sleep difficulties, pain, anxiety or depression), or 2) for cardiac patients or patients without sleep difficulties, pain, anxiety or depression, Coreg (Carvedolol, 3.125mg bid for non-cardiac patients and 6.25 mg bid for cardiac patients, titrated to 12.5 mg bid as needed).

Congestive Heart Failure: According to the ValHeft study published in 1999, 35% of the later stage CHF patients are over blocked (they are on a triple adrenergic blockade). The study showed that for these 35%, removing the patient from one of the sympathetic blockers, the patient's reported feeling better. Ans repeated this study at NYU Einstein Hospital under Dr. Ed Sonnenblick (the father of modern CHF therapy) [Lachmann, AHA, 2002].

Our study showed that the patients that qualify under the ValHeft criterion had PSNS levels (HFa's) that were abnormally high (as compared to the non-qualifiers) with normal to low SNS levels (LFA's). By removing an adrenergic blocker (the ARB in this case) the patient's PSNS levels dropped dramatically, the SNS levels were still low and remained blocked even in the face of a Valsalva, and the patients reported being able to climb a flight of stairs without pausing or spend an hour with the grandchildren without fatiguing.

Post-MI's: Here, titrating medications based on the patient's own numbers (customized medicine) to resting baseline; maintain balance; and keep the patient out of the high risk area is the key. Once this is accomplished, providing more appropriate ANS health through working with the challenge responses can be considered.

Unexplained Arrhythmias: Especially arrhythmias in young, healthy, fit women who, for example, frequently get palpitations during or after exercise. Our Cardiologists have found that, as you might expect, their hearts are healthy and all other cardiologic tests are negative except for an imbalance in their ANS. Readjusting their balance seems to relieve their arrhythmia, they have been rechecked on all of the usual cardiological tests and the tests results are negative. The ANS imbalances in these cases tend to be corrected using short term, low dose therapy. If the ANS imbalance is PSNS dominant, then an anti-cholinergic like digoxen (atrial fibrillation therapy) may help. If the imbalance is SNS dominant, then one of the anti-adrenergic may work. Remember, if PPS exists, then a central (Coreg) versus a peripheral agent is recommended.

Varicose Veins: We have over 5 dozen women in our database, all in their 40's and 50's who were scheduled for vein stripping surgery, but never had it. Their Doppler's showed functioning valves, but "lazy walls." There are two reasons why the walls are "lazy." First the muscles themselves could be malfunctioning, in which case surgery is more than likely. However, the nerves that control the muscles could be what are malfunctioning, and it only seems like "lazy walls." In these 5 dozen women, 6 months of Midodrine (ProAmatine: 2.5mg at bedtime titrated up to 5mg bid and d/c after 6 months based on correction of sympathetic withdrawal and relief of varicosities) corrected the "lazy walls" by reversing the sympathetic incompetence and forcing the vein's valves back together again. The sympathetic incompetence was determined by ANS testing in response to Valsalva and postural change and presented as primary sympathetic withdrawal or PPS masking sympathetic withdrawal.

Orthostasis: The physiologic definition of orthostasis is sympathetic withdrawal. Based on a 303 patient retrospective study performed by the University of Medicine and Dentistry of New Jersey, Newark, Cardiology Division [AHA, 2003 Abstract], the LFa parameters is 78.13% more accurate in identifying orthostatic patients than the classical heart rate (HR) variability ratio (LF/HF) parameter. Furthermore, the VitalScan was found to be 43% more accurate in identifying orthostatic patients than the patient's own physician. Sympathetic withdrawal (any decrease in the LFa from initial baseline to standing) defines the continuum of Orthostasis. If there is **sympathetic withdrawal** (SW) and the blood pressure (BP) decreases at all then pre-clinical **orthostatic hypotension** (OH) is indicated. If the BP drops by more than 20 mmHg systolic or 10 mmHg diastolic then clinical OH is indicated. If SW is present and the BP increases by between 1 and about 20 mmHg systolic then **orthostatic intolerance** is indicated. If SW is present and the BP increases by between 20 and 30 mmHg systolic then pre-clinical **orthostatic hypertension** is indicated. If BP increases by more than 30 mmHg systolic then clinical orthostatic hypertension is indicated. If SW is present and the HR increases by more than 30 bpm or exceeds 120 bpm then **postural orthostatic tachycardia syndrome** (POTS) or postural tachycardia syndrome is indicated.

Syncope: Syncope can be indicated in two ways, mostly depending on the ("physiologic") age of the patient. If the patient's ANS is depleted (e.g., "older" - the Valsalva and stand peak responses on the Trends plot are both below 5 bpm²), then (**Vasovagal**) syncope is indicated if 3 or more of the following 5 issues present: 1) the initial baseline HFa (parasympathetic parameter) is greater than the LFa (sympathetic parameter), 2) the deep breathing HFa is more than an order of magnitude greater than the rest of the HFa responses for the whole test, 3) either or both of the intervening baseline HFa responses are greater than the respective LFa's, 4) the HFa response to Valsalva is greater than twice that at baseline, or 5) the HFa response to stand is greater (by any amount) than that at baseline.

For ("physiologic") younger patients, the Trends plot seems to be the primary indicator of syncope. If the initial LFa peak at stand is approximately equal to or greater than the LFa peak during Valsalva, then syncope is indicated.

Physiologically this suggests that it takes more sympathetic drive to stand than it does to respond to a more powerful stressor such as a series of Valsalva maneuvers. If syncope presents, the HR can differentiate cardiogenic from neurogenic. If syncope presents and the HR does increase then the nerves seem to be working, so **cardiogenic syncope** is indicated. If HR does not increase then the nerves seem to not be working, so **neurogenic syncope** is indicated.

ONCOLOGY

It is known that chemo-therapy drugs are neurotoxic. Trying to protect the ANS and maintain balance will help to preserve patients' quality of life. Also, a significant problem with chemo- therapy is the fact that a significant subpopulation of patients tend to vomit often, leading to many complications and interfering with the therapy.

Determining those patients who are Vagally dominant and therefore may tend to vomit can help oncologists guide therapy.

NEUROLOGY

These assessments provide comprehensive assessment for neurological disorders, simple and complex. In addition to diagnosis it assists with the personalized long-term management of chronic neurological diseases. Disorders of the nervous system can affect any system within your body and can originate in the peripheral nervous system or the central nervous system and may be primary or secondary to other disorders.

Parkinsons', Multiple Sclerosis, etc., including Multiple System Atrophy: Most chronic progressive diseases eventually involve, among other things, GI tract motility and Urogenital function. These are, at least in part, ANS controlled quality of life functions. In some cases there is evidence that the combination of the disease and the therapy are accelerating the demise of the ANS.

Maintaining a better ANS balance throughout the life of a Parkinsons' patient will help to protect quality of life and possibly help to preserve length of life. Regardless of the condition, earlier detection of ANS decline to put off CAN as long as possible and better ANS balance to help preserve quality of life are the two main goals of ANS monitoring.

Sleep Disorders and Sexual Dysfunction: Again, ANS monitoring can help to detect earlier whether there is ANS involvement and determine ANS balance. Correcting ANS balance will help to maintain proper sleep and sexual function.

Unexplained Seizures: With all other exams negative, if seizures persist, there may be an ANS involvement. Our work with other physicians has discovered that in these cases, correcting the ANS imbalance seemed to resolve the seizure disorder. The patient's follow-up neurological and other testing where all negative, further suggesting ANS involvement.

Pain: ANS monitoring helps to differentiate between psychosomatic pain, somatosensory pain and complex regional pain syndrome (CRPS). Pain as a stressor will cause the sympathetics to be high. In psychosomatic pain patients the perception of pain may be very real, but the autonomic "perception" at rest (initial baseline) is normal or high normal. However, if the initial baseline sympathetics are abnormally high and the

parasympathetics are normal to low, in the un-medicated state, then the pain is affecting the autonomics and it may be **somatosensory pain**. In this case, in the medicated state, proper ANS balance will show a well maintained individual and help minimize the onset of secondary symptoms.

For **CRPS (specifically plexus damage)**, where there is nerve and vascular injury, both the sympathetics and the parasympathetics will be abnormally high in the un-medicated state. The sympathetics due to the stress of the pain and the parasympathetics due to the reduction in blood perfusion to the affected area of the body. Like somatosensory pain, in the medicated state, proper ANS balance will show a well maintained individual and help minimize the onset of secondary symptoms (e.g., ADD, bladder dysfunction, GI upset, sexual dysfunction, and depression). ANS monitoring helps to 1) differentiate the pain diagnosis and detect CRPS earlier, 2) objectively quantify pain levels (based on sympathetic levels); 3) titrated, typically, addicting medications specifically to the patient's individual needs based on their own objective, testable, and repeatable numbers; and 4) document rehabilitation (reduced pain and increased blood flow, if needed, will cause ANS levels to normalize) by serial monitoring with quantitative results.

PAIN MANAGEMENT

Scientific studies have consistently shown that nervous system function is disturbed in chronic pain patients (Bruehl and Chung, 2004). Acute pain impacts the nervous system in predictable and measureable ways as well (Koenig, 2014). This relationship between both chronic and acute pain has important implications for the treatment and monitoring of chronic pain patients.

The following studies have specifically shown reduced HRV in patients suffering from:

- Complex Regional Pain Syndrome (Terkelson, et al., 2012)
- Fibromyalgia (Mork et al., 2013)
- Chronic Neck Pain (Kang et al., 2012)
- Irritable Bowel Syndrome (Mazurak et al., 2012)
- Headache (Micieli et al., 1993)

INTERNAL MEDICINE

ADD/ADHD, Fibromyalgia, and Chronic Fatigue Syndrome (CFS): All involve PPS to greater and greater degrees (in order of listing). In fact, for ADD, physicians using ANS monitoring have removed ADD patients from long-term sedatives, and developmentally stunting medications and replaced them with Amytriptaline or Nortriptaline to deal with the limbic issues as well as the excess cholinergic issues. After 9 to 15 months patients have stabilized and have been weaned over a 3 month period and are reporting feeling normal. Fibromyalgia and CFS resolution may not be total. This is due to the diffuse nature of the syndromes, but they will be significantly reduced, and in some cases relieved.

Morbid Obesity, AIDS, and other chronic progressive diseases: As above, maintaining ANS balance permits the physician to work with a more stable patient response (to disease and therapy) so they can be more aggressive, and helps to diagnose the different stages of autonomic decline: acute, sub-acute, chronic and critical.

PULMONOLOGY

Patients with asthma have exaggerated bronchoconstriction of their airways in response to certain indirect (e.g. cold air, allergens, dust, exercise) or direct (e.g. inhaled methacholine) stimuli. This 'hyper-reactivity' usually co-exists with airway inflammation, although the pathophysiological mechanisms underlying these changes are not fully understood. It is likely that this hyper-reactivity is associated with abnormal parasympathetic nervous system control.

Sleep Apnea: It is known that with sleep apnea the sympathetics are exceedingly high and the parasympathetics are low normal to low. So much so that the natural inversion that should take place in the evening fails. The high sympathetics (due to the stresses of sleep apnea, prevent the inversion and thereby prevent proper sleep. CPAP helps to remove some of the stressors that are associated with sleep apnea and over time the sympathetics decrease and the parasympathetics normalize.

Asthma or COPD or Other Respiratory Disorders: Many of the medications utilized by respiratory disorder patients are now self-administered. ANS monitoring can help the pulmonologist maintain a better ANS balance to prevent potential cardiac event.

ENDOCRINOLOGY

Diabetes Mellitus: DAN is well known as the risk indicator for cardiac disease in diabetics.

However, earlier detection of ANS decline, far in advance of DAN is also known to help preserve quality of life, extend overall survival, and reduce the demand for costly intervention procedures.

From a report presented to the ADA in April 2004, the onset of ANS decline can be detected earlier by ANS monitoring. In the acute (early) stages, the nerves can be better protected (sugar and insulin regulation or anti-oxidants) to possibly reverse the trend of ANS decline (in this stage there is no structural damage only functional damage to the nerves). Returning ANS balance and better disease management can slow ANS decline in the sub-acute and chronic stages and still preserve some of the quality of life functions remaining to the patient.

DAN is a disease that requires customized medicine, and ANS monitoring facilitates customized medicine.

Hypothyroidism: Excess parasympathetic activity (including PPS) seems to contribute to the shut down of the Thyroid. About 3 dozen women in our database have hypothyroidism, are on high to very high levels of thyroid hormone replacement therapy (e.g., 0.1mg Synthroid) and still report not being totally themselves. The excess parasympathetic activity was reduced (e.g., Elavil or Coreg) and after a series of thyroid hormone blood tests, Synthroid levels were drastically reduced (as low as 0.01mg) or in a few cases even eliminated and follow-up thyroid levels are normal.

Menopause: We have noted that most, if not all, women that have had more than 3 pregnancies also have PPS. PPS seems to also be the common factor in women in their 40's presenting with menopause-like symptoms, as well as the common factor in women who are unstable on hormone replacement therapy once in their 50's. Resolving PPS seems to relieve the 40 y/os' symptoms and dampen if not mute the 50 y/os' symptoms.

What Types of Patients Will Benefit from Autonomic Testing?

EXAMPLES OF THE MANY SITUATIONS WHERE AUTONOMIC TESTING IS OF CLINICAL UTILITY INCLUDE:

1. Patients with syncope: Autonomic testing is necessary to differentiate neurally mediated syncope from neurogenic orthostatic hypotension and other causes of syncope.

2. Patients with diabetes mellitus: All patients with diabetes are recommended to have autonomic testing (sudomotor, cardiovagal and adrenergic) at diagnosis (type 2 diabetes) or five years after diagnosis (type 1 diabetes). There is a high prevalence of cardiovascular autonomic neuropathy in the diabetic population. The relationship between autonomic dysfunction and cardiovascular risk has been well documented and is important to monitor for patients planning major surgical procedures or considering moderate to high intensity physical exercise. This is the reason that the ADA recommends autonomic testing for all patients with type 2 diabetes at the time of diagnosis, and all patients with type 1 diabetes five years after diagnosis. The increased perioperative mortality in cardiovascular autonomic neuropathy is linked to greater blood pressure instability and hypothermia. This information may prompt high-risk patients to forgo an elective procedure or allow the anesthesiologist to prepare for potential hemodynamic changes, thereby reducing the risks of morbidity and mortality.

3. Patients with orthostatic dizziness: Patients with recurrent dizziness when standing may have autonomic dysfunction, postural tachycardia syndrome, or other autonomic neuropathy that can be treated if a diagnosis

is made. All autonomic tests (sudomotor, cardiovagal, and adrenergic) are appropriate to use in forming a differential diagnosis, defining the physiology of orthostatic intolerance in the individual patient, grading the severity of impairment, and directing appropriate therapy.

4. Patients with disorders of sweating: Autonomic testing can provide a diagnosis which can lead to treatment of the underlying disorder and improvements in clinical outcomes. Patients found to have global anhidrosis may be at risk for heat exhaustion or heat stroke and can benefit from interventions to restore sweating, when a reversible cause is diagnosed, or otherwise from management strategies to avoid heat stress. Although sudomotor testing will provide specific information about the problem with sweating, cardiovagal, and adrenergic testing will narrow the differential diagnosis and are therefore integral parts of the autonomic test (i.e., does the patient have an autonomic ganglionopathy, an isolated autonomic neuropathy such as Ross syndrome, a peripheral neuropathy causing distal anhidrosis and proximal hyperhidrosis, how severe or anatomically widespread is the deficit, etc.).

5. Patients with peripheral neuropathy from a number of different causes such as (but not limited to) amyloidosis, Fabry's disease, Sjögren's syndrome, and autoimmune neuropathies.



VitalScan

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