CHAPTER 11

SYMPATHETIC/PARASYMPATHETIC BALANCE EXPLAINED

Introduction

Sympathetic/Parasympathetic balance represents a dualistic, diphasic model of autonomic nervous system activity. The term diphasic refers to the alternate operation of opposing forces for the purpose of maintaining metabolic balance. Each of these opposing forces operates under the direction of one or more fundamental control systems. Sympathetic/Parasympathetic Balance is one of several fundamental control systems in human physiology.

Sympathetic and Parasympathetic Imbalances represent the two opposite abnormalities possible regarding the autonomic nervous system. It is often more appropriate to think of these imbalances as sympathetic or parasympathetic **stress**. The term stress, as it is used here, is intended to mean an over-sensitivity, or an over-reactivity of that branch of the autonomic nervous system. In a normal healthy individual, sympathetic forces are initiated in response to some stress demand, continuing in operation until the limits of homeostasis are reached. At this point, sympathetic activity is switched off and parasympathetic activity is initiated, moving physiological qualities such as temperature, blood sugar levels, etc. in the parasympathetic direction until the opposite homeostatic limit is reached, at which point parasympathetic forces are switched off and sympathetic forces reinitiated, and so on.

Many patients you see in your clinical practice are showing the effects of a Sympathetic/Parasympathetic system out of control. One of the two branches of the autonomic nervous system has been oversensitized to the point of reacting spontaneously, excessively, and inappropriately. In essence, this patient is "stuck" in either a state of sympathetic or parasympathetic over-reactivity. The clinical picture is often best described as unpredictable – with bizarre symptoms that come and go seemingly with a mind of their own.

The sympathetic and parasympathetic systems are antagonistic at the organic level of biological organization, i.e., organs stimulated by sympathetic activity are inhibited by parasympathetic, and vice-versa.

Sympathetic/Parasympathetic Nervous System Activity

The SNS and PNS stimulate or work in harmony with (1):

Sympathetic	Parasympathetic
Calcium; Phosphorous	Potassium; Sodium; Magnesium
Adrenal Medulla; Thyroid	Pancreas Tail; Pancreas Head; Adrenal Cortex
Hypothalamus; Pineal	Thymus; Parotid
Testosterone; Progesterone	Estrogen
G.I. sphincters	G.I. lumen motility; G.I. secretion & salivary glands
Sphincter of Vater	Gall Bladder & Bile Ducts
Liver glycogen mobilization; triglyceride	Liver glycogen storage; Fat storage mobilization
Respiratory center	Bronchial muscles & glands
Baroreceptors	Nasal & Pharyngeal secretion
Myocardium	
Vasomotor activity (constriction and dilation)	
Pupil dilators; muscles of Muller (exophthalmos)	Pupil constrictors; muscles of the iris; muscles of accommodation: levator palpebrae (enophthalmos)
	Lacrimal secretion
Prostate: Cowpers and Bartholins production	Prostate: Cowpers & Bartholins release of secretion
	Penis erection
Bladder trigonum and sphincters; uretha	Bladder wall
Pilomotor activity; sweat secretory activity	
	Histamine activity

Sympathetic Stress & Parasympathetic Stress

Sympathetic Stress or sympathetic nervous over-reactivity, is typified by excess activity in one or more of the listings under Sympathetic Nervous System Activity, above. In the various states of toxemia, the SNS is particularly sensitive to harmful stimulation. The calcium:potassium ratio determines the SNS:PNS effect on various organs. When there is Sympathetic stress there will be adrenergic and/or cholinergic nerve hyperexcitability.

Parasympathetic Stress is typified by excess activity in one or more of the listings found above under Parasympathetic Nervous System Activity. Again, the calcium:potassium ratio determines the SNS:PNS effect on various organs. Parasympathetic stress is associated with cholinergic nerve hyperexcitability.

Sympathetic or Parasympathetic reactivity is evidenced by changes in skin color and temperature. The skin color, red or pale, depends upon the quantity of blood in the capillaries and venules. Sargent's white line and a dermographic white line (see Clinical Findings, below) indicate constriction of capillaries and venules, and result from sympathetic alpha adrenergic over-reactivity. A dermographic red line bordered by white means the capillaries and venules in the central area are dilated, which results from sympathetic cholinergic stress. A dermographic red line reflects inhibition of alpha adrenergic activity by parasympathetic cholinergic over- reactivity.

Skin temperature depends both on the quantity of blood flowing, and its rate of flow. Warm, pale skin occurs when the arteries are dilated (SNS cholinergic), and the capillaries are constricted (SNS alpha adrenergic). Cold, blue skin means the arteries are constricted (SNS alpha adrenergic), while the capillaries and venules are dilated (SNS cholinergic). (SNS beta adrenergics do not affect the skin, as these are dilators to skeletal muscles and coronary arteries, and constrictors to the abdominal viscera.)

Warm, flushed skin indicates that the arteries are dilated (SNS cholinergic), as are the capillaries, reflecting PNS cholinergic inhibition of SNS alpha adrenergic activity. This vasodilation is particularly evident on the ears of patients with a tendency toward Parasympathetic Stress.

The sweat glands are stimulated by SNS cholinergic activity. This is the only SNS activity not stimulated by the adrenal medulla. Night sweats are frequently an indication of Sympathetic Stress.

Parasympathetic Stress involves an unstable equilibrium that can result in excessive sweating of the hands and feet. Otherwise, the patient with Parasympathetic Stress tends to have dry skin associated with SNS inhibition. However, acetylcholine excess can result in night sweats.

One point of interest about these imbalances is that they are **reactive** as often as causative. What do we mean by that? We mean that these nervous system over-reactivities have developed in response to an unrelenting stressor in the patient's life. The source of stress may be an emotional factor; it may be a chronic nutritional inadequacy; very often it is one of the other Nutri-Spec Fundamental Imbalances. Whatever the source of stress, the involved branch of the autonomic nervous system has become **habituated** to reacting in an attempt to meet the challenge of the stressor. Over time, the neurological activity becomes **facilitated** to the point where an inappropriate and excessive response is triggered by the slightest provocation.

Clinical Findings

Sympathetic	Parasympathetic
Pupil large	Pupil small
Pulse increased; marked orthostatic increase; arrhythmias	Pulse decreased; little orthostatic increase
Respiratory rate increased; bronchial dilation; respiratory depth increased	Respiratory rate decreased unless bronchial constriction
Systolic BP & Pulse Pressure increased, especially orthostatically	Orthostatic failure of systolic BP & Pulse Pressure
Pilomotor reflex increased	Pilomotor activity absent
Dermographic white line, or thin red line bordered by white; Sargent's Line positive	Dermographic red line; Sargent's line negative
Cough reflex decreased	Cough reflex increased
Oliguria; urine specific gravity high	Polyuria; urine specific gravity low
Cold sweat on hands, or, cold dry hands	Hands warm and dry
Nervous tension; insomnia; tremors	Nervous tension; depression; anxiety; somnolence or insomnia
Dry mouth	Saliva and tear quantity increased
Exophthalmos	Enophthalmos (unless Anaerobic Imbalance)
Glucose increased	Glucose decreased (or increased if insulin resistance)
WBC decreased	WBC increased
Poor circulation associated with vasoconstriction	Poor circulation associated with decreased pulse pressure
Indigestion; ulcers; gall bladder dysfunction	Indigestion; ulcers; mucus colitis
Food allergies	Allergies; asthma

Objective Testing vs. Clinical Symptoms

Many of the clinical findings listed above are symptoms that are often associated with Sympathetic or Parasympathetic Imbalances. Many of the symptoms frequently associated with Sympathetic or Parasympathetic tendencies can also be caused by other Nutri-Spec fundamental imbalances, and in fact are more likely associated with those imbalances than with the autonomic nervous system.

There are a number of common symptoms that can associated with **either** a Sympathetic or a Parasympathetic Imbalance. To illustrate, consider a patient suffering from constipation. A Sympathetic Imbalance is typified by spasms of the G.I. sphincters, a lack of tone in the lumen musculature, and an atonic constipation. On the other hand, a Parasympathetic patient tends to have spasms of the G.I. lumen, which can be accompanied by either diarrhea or by a spastic constipation. So, you see that two patients with the identical symptom, constipation, can have that condition associated with exactly opposite causes, and therefore need exactly opposite therapeutic intervention. From a nutrition standpoint, the dietary recommendations and supplementation that would benefit the constipation of one of these patients would actually make the other patient much worse.

Clearly, you need some objective means to determine the Sympathetic/ Parasympathetic Imbalances in your patients. The objective tests you need are, of course, provided by your Nutri-Spec testing procedure. The specific tests related to Sympathetic/Parasympathetic Imbalances are found in Chapter 8, and many are described below.

The 3-point quick scan for evaluating this imbalance includes the orthostatic blood pressure response, and the orthostatic pulse rate response. All three of these tests are primary indicators of autonomic nerve function.(2)

Note that you will never treat an asthmatic as Sympathetic, and, you will never treat a Type I diabetic as Parasympathetic. To make such a mistake will almost invariably precipitate a disastrous exacerbation of the patient's asthma or diabetes.

Other confirmatory findings that will clue you in to a Sympathetic or Parasympathetic Imbalance are the pupil size, dermographics red or white, high or low blood sugar, ear color, the saliva and tear quantity, and any tendency to insomnia or somnolence.

Consider the primary source of our Sympathetic/Parasympathetic paradigm. When Francis Pottenger wrote "Symptoms of Visceral Disease" more than 90 years ago, he was <u>not</u> an "alternative" or "natural" health care provider. He was a world-renowned medical doctor who served as an officer in several influential medical organizations of his day. In other words, he was as "establishment" as could be. Yet he was an astute clinician, and the first to make the observation that most disease symptoms were at least partly mediated through the autonomic nerves. Further (as the first practitioner of your own <u>patient-specific</u> emphasis in health care), he observed that two patients could be victimized by the same pathological stressor, yet respond with entirely different symptoms – either sympathetic or parasympathetic dominant. Controlling symptoms was achieved far more effectively by treating the sympathetic/parasympathetic component of the disease than by treating the disease symptoms per se.

Lowe's Clinical Autonomic Disorders was published in the mid 1990s. All the clinical phenomena defined by Pottenger were now quantified – to the nth degree. Using modern technology, the contributors to Lowe's were able to define the complex interplay between Sympathetic and Parasympathetic stimulation and inhibition that underlie the orthostatic blood pressure response and the orthostatic pulse rate response.

The orthostatic blood pressure response is not a simple matter of a sympathetic mediated increase in blood pressure, moving blood from the pool in the splanchnic vessels up to the brain to keep us from passing out when we stand up. The systolic blood pressure actually changes direction 4 times – going up, then down, then up, then down – in the 30 seconds beginning with initiation of the standing movement. Similarly, the diastolic blood pressure goes through the same 4 changes, but lags the systolic changes by a few seconds.

The orthostatic pulse response is just as complex. It is the initial contraction of the large muscles of the thighs, hips, pelvis, abdomen, and spine that sets off the orthostatic response. In fact, the most significant change in pulse occurs in the first 3 seconds, during most of which the patient is still recumbent.

In a Sympathetic Imbalance, the orthostatic rise in blood pressure and pulse is exaggerated, and sometimes prolonged, while in a Parasympathetic Imbalance the amplitude of the changes is it muted. There is also an interplay between Sympathetic/Parasympathetic balance and Electrolyte Balance in many patients' orthostatic response. All such considerations are programmed into your Nutri-Spec analysis, so you know exactly how to manage patients regardless of what wild gyrations in pulse and blood pressure they show upon orthostatic challenge.

Sympathetic/Parasympathetic Stress vs. Insufficiency

It is often appropriate to think of autonomic nerve imbalances as Sympathetic or Parasympathetic **Stress**. We all tend to react to acutely stressful events in our lives with some combination of Sympathetic and Parasympathetic activation, along with increased secretion of stress hormones. Some of us, however, tend to be predominantly Sympathetic reactors to stress, while others tend to be Parasympathetic reactors. Sympathetic reactors respond to a stress demand with Sympathetic nervous system activation (norepinephrine), along with adrenal medulla secretion of epinephrine – the classic catecholamine-mediated fight or flight response. Parasympathetic reactors tend to react to challenges with excess stimulation of the Parasympathetic system, along with increased secretion of corticosteroid stress hormones.

So, when faced with a crisis, a Sympathetic reactor will show a racing heart, an increased rate and depth of respiration, a pounding heart associated with an elevation in pulse pressure, a dry mouth, and trembling hands. In the same crisis situation, a Parasympathetic reactor will experience a stomach "tied in knots," may vomit, and perhaps experience diarrhea. The respiratory rate may increase, but the heart rate will increase far less. Blood pressure may rise, but the diastolic will increase as much as the systolic. A diversity of Parasympathetic-related symptom is possible, including tension headache, breaking out in a rash, or, in susceptible individuals, an asthma attack. In extreme cases, the person may faint ("Vaso-Vagal"). In chronic cases, the Parasympathetic dominant person will tend to withdraw – and eat.

What we have just described are the classic Sympathetic Stress and Parasympathetic Stress reactions. In other words, a Sympathetic Imbalance in which the Sympathetic system is strongly reactive while the Parasympathetic system is inhibited or weak, or conversely, when the Parasympathetic system totally dominates over a relatively weak Sympathetic system. These are the types of Sympathetic and Parasympathetic Imbalances you will tend find in younger adult patients. However, as we age, autonomic nervous system imbalances will tend to show up in association not with an extremely strong Sympathetic or Parasympathetic portion of the autonomic nervous system, but with an insufficiency or failure of one or the other. In other words, we can find a Sympathetic Imbalance associated not with a strong Sympathetic system, but rather with a weak Parasympathetic system. Similarly, we can find people who test as Parasympathetic because of an inability to produce Sympathetic responses rather than an abnormally reactive Parasympathetic system.

So, at about age 33 some of your patients who test Sympathetic dominant are as much showing Parasympathetic failure as Sympathetic Stress, and some of your Parasympathetic patients are as much Sympathetic insufficient as they are Parasympathetic dominant. At about age 53, failure of the Sympathetic or the Parasympathetic system is a component of most Sympathetic or Parasympathetic patients' imbalances. Increasingly with age, some degree of failure of both Sympathetic and Parasympathetic reactivity is common.

Let us consider the various possibilities that unfold through a person's lifetime as regards Sympathetic/Parasympathetic balance. First, there are those who have a normal, healthy reactivity of both the Sympathetic and Parasympathetic systems. They go through their entire lives never showing a Sympathetic or Parasympathetic Imbalance.

Next, consider the individuals who are just plain "hyper." They show an exaggerated response of both the Sympathetic and Parasympathetic system from childhood up through at least age 22. Testing these individuals as teenagers will yield an extreme combination of both Sympathetic and Parasympathetic indicators. If we test a hyper 17-year-old, we may find extreme orthostatic pulse response on the upside, yet orthostatic failure of the blood pressure. There may be sweaty palms and dilated pupils, yet a +3 dermographics response. These patients have neither a Sympathetic nor a Parasympathetic Imbalance, they are just extremely sensitive types. As these people progress through life, the reactivity of the Sympathetic and Parasympathetic systems gradually decrease. If they decrease at an equal rate, these patients will never show a Sympathetic/Parasympathetic Imbalance. If one system begins to fail before the other, then the system that does not fail will show up as the imbalance you must treat with Nutri- Spec.

Now, consider those who are your classic Sympathetic or Parasympathetic reactors. Upon Nutri-Spec testing, these individuals will show a Sympathetic or Parasympathetic Imbalance through adolescence, young adulthood, and into middle age. That imbalance may persist into old age, but in many of these patients their dominant system begins to fail at some point. As that previously dominant system becomes exhausted, the patient will no longer test as Sympathetic or Parasympathetic.

Regardless of what Sympathetic or Parasympathetic Imbalances exist or do not exist in a person through adolescence, early adulthood, and middle age, there comes a point in life when

both the Sympathetic and Parasympathetic reactive capacity begins to fail. Lowe's makes the point that by far the most common autonomic disorder is a failure (rather than an over-reactivity) of one of the autonomic systems, or both. Of course Lowe's is written from a pathologist's point of view; it is concerned with the treatment of frankly pathological conditions of the autonomic nervous system. Still, his point is well taken. Many of your patients, particularly those over age 42, will test as Sympathetic or Parasympathetic because of a failure of one system of the other. Many, many of your patients will test neither Sympathetic nor Parasympathetic because they are failing equally in the performance of both systems.

It is in this universal failure of both the Sympathetic and Parasympathetic system with age that we see the essential need for the Nutri-Spec SOLID DNP (Stage Of Life Inflam-Aging Defense Diphasic Nutrition Plan). Next to Nutri-Spec Metabolic Balancing, the most valuable service you offer your patients is maintaining their Vital Reserves with some combination of Complex S and/or Complex P. These powerful supplements will maintain a high amplitude diurnal cycle of Sympathetic and Parasympathetic vitality throughout middle age and beyond.

The 6 Types of Sympathetic/Parasympathetic Imbalance

Your Nutri-Spec Sympathetic/Parasympathetic Imbalance Analysis guides you toward ultra-specific recommendations for each patient's individualized needs. The 6 types of Autonomic Imbalance include:

- Parasympathetic Stress
- Sympathetic Noradrenergic Insufficiency
- Parasympathetic Insufficiency
- Sympathetic Adrenergic Stress
- Sympathetic Nor-Adrenergic Stress
- Sympathetic Nor-Adrenergic Stress Failure

Identifying which of these Imbalances applies to your patient allows you to consider expanding your nutrition recommendations beyond supplementing with Complex S or Complex P. The Table below gives you a nice summary of the most common clinical findings in each of the 6 autonomic nerve disturbances.

Parasympathetic Stress	**Sympathetic Noradrenergic Insufficiency**	**Parasympathetic Insufficiency**	**Sympathetic Adrenergic Stress**	**Sympathetic Noradrenergic Stress**	**Sympathetic Noradrenergic Stress Failure**
Pulse Rate - -	Orthostatic Pulse Pressure failure	Pulse Rate + Orthostatic Pulse Rate +	Pulse Rate +	Pulse Rate +	Orthostatic Pulse Pressure Failure
	Pulse Rate – Orthostatic Pulse Rate +		Breath Rate +	Systolic BP + Orthostatic BP +	Orthostatic Pulse Rate +
Pupils small, light reactivity –	Pupils small, light reactivity –	Pupils large, light reactivity +	Pupils large, light reactivity +	Pupils large, light reactivity +	
Saliva + Saliva pH +		Dry Mouth & Eyes		Saliva thick Saliva pH – Dry Mouth	Vertigo Nocturnal Diuresis
	Fatigue		Cutaneous Vasoconstriction; Muscle Vasodilation	Vasoconstriction Hands Cold Pallor	Fatigue
Rapid GI Transit, Diarrhea	Post-prandial hypotension	Atonic Constipation	Slow GI Transit, Constipation	Pilomotor +	
Bronchoconstriction	(Hypothyroid?)		Fine Hand Tremor	Fine Hand Tremor	
			Hyperglycemia	Perspiration +	

Parasympathetic Stress: You find this Imbalance in your patients with a very strong Parasympathetic reactivity. Their Sympathetic tone may not be particularly deficient, but is definitely dominated by Parasympathetic response to the challenges of daily living.

The pulse rate in these patients will be slow at rest, and will not spike when challenged by your orthostatic testing, or during stress-provoking life events. The pupils will tend to be small (--- and if you see a patient with large pupils you can totally rule out the need for Complex P or any other vagus-controlling nutrients).

In accord with the Parasympathetic activation of the gastrointestinal tract, the patient will produce much saliva (almost always of a somewhat elevated pH), will show rapid GI transit, even to the point of frequent diarrhea.

Since the Parasympathetic system inhibits the vasoconstriction of Sympathetic activity, there will tend to be red dermographics, and the fingers will be palpably as warm as the upper arm. The red dermographics will also be potentiated by the histamine reactions typical in those who are Parasympathetic dominant, and will also be exacerbated by any tendency to any form of Alkalosis Imbalance.

The vagus nerve is bronchoconstrictor, which explains the tendency to asthma and Eosinophilic Bronchitis in those who are Parasympathetic dominant.

A dominant theme emergent from the chaotic realm of alternative healthcare is "Strengthen the Vagus Nerve." Be careful what you wish for. Vagotonia is a direct cause in:

- asthma
- mucous colitis
- hypoglycemia
- some forms of chronic fatigue
- some forms of depression
- orthostatic intolerance (dizzy/weak/off-balance upon standing)
- vaso-vagal effects (with or without syncope) --- in response to such stressors as the sight of something ugly or frightening, exposure to airborne toxins or allergens, prolonged standing, or emotional stress.

Sympathetic Nor-Adrenergic Insufficiency: These are your patients with insufficient catecholamine response. That weakness may represent depletion occurring with the aging process. Just as likely, the decline in Sympathetic response capacity results from years of "whipping a tired horse," such that now the Sympathetic system is in a chronic state of exhaustion.

These individuals show the slow pulse rate typical of Parasympathetic dominance, but the big clinical sign is orthostatic blood pressure failure. In particular, the systolic pressure will drop upon standing, and will drop more than does the diastolic pressure, thus creating an extreme drop in Pulse Pressure. The ability to maintain normal pulse pressure under stress is often shown in these patients after nothing more than the stress of eating a meal. A substantial size meal pulls circulation into the gut, leaving the periphery somewhat hypovolemic, and so these patients will

often show low blood pressure post-prandially, and may in extreme cases, actually tend to dizziness or even syncope shortly after eating.

Fatigue is a major symptom, and very often these patients are also hypothyroid, and in fact, deficient thyroid function may be the primary cause of a patient's nor-adrenergic insufficiency.

Parasympathetic Insufficiency: These individuals are Sympathetic dominant in association with a very weak Parasympathetic reactivity. The pulse rate will be elevated, and the orthostatic pulse rate increase is often exaggerated far above normal. With insufficient Parasympathetic activity, there will be dry mouth and eyes, and atonic constipation.

Knowing that you can consider the Sympathetic (catecholamine) system as go-go "fight or flight", while the Parasympathetic system is "rest and digest" — If you are asked which system tends to decline most rapidly as part of the aging process, the "go-go" Sympathetic system "no-go" Parasympathetic system, which would be your guess? Surprisingly, it is the Parasympathetic system that fades noticeably beginning at age 43, and then precipitately at age 53 (in line with your Stage Of Life INFLAM-AGING Defense concept). Here, you have the element of truth in the alternative health care emphasis on "support the vagus". These patients critically need the most specific vagal support, your Complex S, along with a full complement of adjunctive supplements to improve Vagus tone.

Sympathetic Adrenergic Stress: This imbalance is an excess of the Adrenal Medulla component of the catecholamine stress response. It does not directly involve the Sympathetic nervous system, but activates in parallel with it, with adrenaline secretion to compliment the nor-adrenaline activity of the autonomic system.

In these individuals you will see not only the increased pulse rate of a Sympathetic Imbalance, but also an increased breath rate. These individuals tend to be "hyper" both in terms of physical and mental activity. There may be a fine hand tremor. The patient is often plagued by constipation, and rising blood sugar can become a problem even in early adulthood.

Sympathetic Nor-Adrenergic Stress: These patients are demonstrating the entirely neurogenic component of Sympathetic Stress. The pulse rate, diastolic blood pressure, and orthostatic blood pressure response are all on the high side. The pupil may be large, and the mouth dry. Palpation of the fingers simultaneously with the bicep area of the arm may show a big drop in cutaneous temperature as the circulation moves toward the periphery. There may be a fine hand tremor, an exaggerated Pilomotor reflex, and perspiration may be excessive.

As with the Adrenergic Stress patients, the chronic vasoconstriction plus the push-push-push of the myocardium can lead to cardiovascular patho-physiology , ultimately showing up as a Nutri-Spec Electrolyte Stress Imbalance.

Sympathetic Nor-Adrenergic Stress Failure: These are your most difficult autonomic imbalanced patients, and, they will make up a substantial portion of your patients age 43+. The mechanism here is a hypertonic Sympathetic system, but with impaired responsiveness.

These individuals show the orthostatic Pulse Pressure failure typical of those with either Parasympathetic Insufficiency or Sympathetic Nor-Adrenergic Insufficiency, and the orthostatic pulse rate increase will be particularly intense.

This Imbalance develops in those who are innately strong Sympathetically, but in whom Sympathetic function degenerates as a result of some other patho-physiological process. Type II diabetes, or even insulin resistance that has not yet progressed to the point Metabolic Syndrome is common. It is also common in Type I diabetics. It is very often associated with Metabolic-Associated Fatty Liver Disease, or, this condition can result from simply a chronically stressed catecholamine system that is now in a state of permanent depletion.

Explanation of clinical findings in Sympathetic Nor-Adrenergic Stress Failure: While individuals with orthostatic hypotension (OH) typically have impaired sympathetic nervous system tone, and therefore low levels of upright plasma norepinephrine, here is your category of patients who have orthostatic hypotension, but who paradoxically have elevated plasma norepinephrine.

One study divided OH patients whose mean age was mid-to-late 60s into two groups . One group was the typical OH, with decreased Sympathetic Tone associated with low plasma norepinephrine; the other group was the atypical OH with elevated plasma norepinephrine. Both groups had severe OH, but the elevated norepinephrine group had less severe adrenergic function deficiency as per a full battery of tests of autonomic imbalance. The orthostatic symptoms of patients in both groups were severe enough that the majority were medicated.

The patients with paradoxical orthostatic failure in the presence of elevated noradrenergic stress showed a less significant drop in orthostatic blood pressure, but with a relatively exaggerated increase in Pulse Rate from supine to standing. (The Pulse Pressure drop is consistent with an absence of Electrolyte/Water Imbalance such as hypovolemia or hypervolemia.) The underlying mechanism behind noradrenergic stress orthostatic failure may be partial autonomic neuropathy of the sympathetic nerves innervating the lower extremities. For reasons unknown, autonomic sympathetic denervation preferentially affects the lower limbs. Progressive impairment of these nerves results in progressive venous pooling of blood, contributing to orthostatic insufficiency.

Another mechanism at work in addition to or instead of autonomic neuropathy is the neuropathy associated with Diabetes. Diabetic neuropathy affects the legs because the nerves to the lower extremities are of longer length, and thus the first to show distal polyneuropathy. In either case, the neuropathy causes an exaggerated attempt at compensatory response by baroreceptors, and that attempted compensation involves stimulating excessive amounts of norepinephrine.

One other possible mechanism is that norepinephrine is elevated in diabetics and the elderly due to decreased renal clearance. The continuum of Insulin Resistance that eventually results in Type 2 diabetics involves excessive Insulin secretion to compensate for the increase in Insulin Resistance in an effort to maintain a normal blood glucose. At some point, a “burnout” is reached, when Insulin production fails to meet the increased demand. As the transition from high to low Insulin progresses, norepinephrine renal clearance steadily decreases, likely potentiating the elevated norepinephrine.

Regardless of the mechanism by which norepinephrine remains chronically elevated, the concept of "burnout" can be applied, not just upon orthostatic challenge, but in activities of daily living. Many of these noradrenergic stress individuals with orthostatic insufficiency have likely been strongly sympathetic-dominant throughout their lives. Now with age, the positional demands on autonomic control of circulation cannot be adequately met, so a sympathetic activation is continuously being stimulated, but without full effect. This circumstance is analogous to flooring a car's gas pedal when two engine cylinders are not firing. No matter how forcefully the gas pedal is depressed, the engine response is inadequate. These individuals may show many signs of physical, mental, and emotional norepinephrine stress, even while they are weak in response to postural orthostatic challenge.

There is one disturbing symptom you will often find in patients with this paradoxical combination of both Sympathetic Stress and Sympathetic Insufficiency. That symptom is vertigo. There are two mechanisms that can potentially underly that condition. The first is that the Sympathetic Stress response will cause fluid retention in a portion of the inner ear because of the vasoactive fluid dynamics in those very sensitive membranes.

The second mechanism that can underly vertigo in these patients is nocturnal diuresis. With the centralization of blood flow while the patient is sleeping, the kidneys eliminate much of this fluid, leaving the peripheral circulation somewhat hypovolemic. The patient needs to get up several times during the night to urinate. Meanwhile, the blood pressure drops in association with the hypovolemia, and circulation to the brain is compromised. These patients, even though they might be somewhat hypertensive during the day, will very often show low blood pressure in the a.m. upon arising, and can experience vertigo when they get up during the night, or first thing in the morning.

The Rationale Behind Your Sympathetic/Parasympathetic Testing

Pa-P1: Testing the change from the sitting position to the supine position is so valuable because it is ultra-specific for Sympathetic/Parasympathetic balance. It is an unwavering physiological fact that when humans assume a supine posture, the Parasympathetic nervous system activates, and the Sympathetic nervous system deactivates. (There is only one other metabolic dysregulation that can cause an excessive decline in supine heart rate, and that is low thyroid function.)

Clinical tests with this kind of specificity are rare, yet here is one that your staff can perform so very simply in just minutes. Your patients who show an exaggerated drop in heart rate after 30 seconds supine definitely need Complex P. These patients have an extremely reactive vagus (and Sacral Parasympathetic) reactivity, and/or a very weak adrenergic catecholamine stress defense capacity. All the nutrients in Complex P, by one or more mechanisms, boost adrenergic power, and/or control an over-sensitized vagus.

Orthostatic Blood Pressure Failure --- Systolic and/or Diastolic and/or Pulse Pressure Failure:

Neurogenic Orthostatic Hypotension (OH) is defined as a fall in supine to standing blood pressure of 20 or more systolic, and 10 or more diastolic. Orthostatic Intolerance (OI) is the same phenomenon of falling systolic and diastolic pressure upon orthostatic challenge, but of lower

magnitude. The symptoms of OI can be just as severe as the symptoms of OH. The criterion for defining OH or OI is that the orthostatic response be measured with a blood pressure taken within 3 minutes of standing.

Central control of blood pressure is regulated tightly through changes in both cardiac output and vascular tone. The Sympathetic Nervous System plays the predominant role in determining the blood pressure and the distribution of cardiac output. While the Parasympathetic Nervous System exerts significant control over the pulse rate, its contribution to the regulation of vascular tone is negligible. Short-term reflex control of the Sympathetic Vasomotor activity is regulated by homeostatic feedback mechanisms --- baroreceptor and chemoreceptor reflexes in particular.

Orthostatic Stress is a significant challenge to the Sympathetic System. Almost immediately upon transition from supine to standing, a gravitational shift of nearly 500 mL of blood away from the chest to the distensible venous capacitance system below the diaphragm (venous pooling) occurs. The result is a rapid decrease in central blood volume, causing a sudden drop in ventricular load, stroke volume, and mean blood pressure. Compensation requires contraction of lower limb muscles along with leg venous valve action, providing intermittent upward flow to help minimize venous pooling.

Sympathetic Noradrenergic Insufficiency severe enough to qualify as a classic medically diagnosable Orthostatic Hypotension shows a decrease in systolic blood pressure of 20 or more and/or a diastolic pressure of 10 or more within 30-180 seconds of standing. But clinically significant sympathetic weakness causing the symptoms listed above is indicated by any decrease in SBP upon standing. In healthy individuals, the SBP actually temporarily increases 8-12 mm Hg immediately upon standing, and that quick, temporary increase in blood pressure is accompanied by an increase in pulse rate of between 8-12 bpm. Any failure of the SBP to rise, or if it changes little, but that failure to rise is accompanied by an increased pulse rate of more than 12, indicates your patient is suffering from deficient sympathetic tone.

Maintaining an appropriate perfusion pressure to critical organs (brain and heart) requires an effective set of neural regulatory actions that must be promptly activated. The Sympathetic Nervous System is fast-acting, and primarily reacts via mechano-receptors, and to a smaller degree via chemoreceptors. Upon standing, the sudden drop in blood pressure in the carotid sinus and the aortic arch triggers baroreceptor-mediated compensatory mechanisms within seconds --- resulting in increased pulse rate, plus a surge in blood pressure driven by both myocardial contractility and peripheral vasoconstriction. Ideally, orthostatic stabilization is achieved in one minute or less.

The most critical consideration regarding your Nutri-Spec orthostatic test of Sympathetic/Parasympathetic Imbalance is its immediacy – checking the pulse rate and blood pressure within 1 minute of standing. During prolonged quiet standing, following the quick sympathetic response, there will be other factors such as gravitational effects, further reducing central blood volume and cardiac output. The influences of increased blood viscosity and arteriosclerotic vascular resistance also come into play, further stressing the cardiovascular challenge. There is activation of neuroendocrine mechanisms such as the renin-angiotensin-aldosterone system, which are also dependent on the blood viscosity and vascular resistance, and which influence the pulse rate and blood pressure over a period of 10-30 minutes after standing.

These factors are in the realm of Electrolyte/Water Balance more than Sympathetic/Parasympathetic Balance.

Clinics specializing in the testing of dysautonomia use the table-tilt test as their gold standard in evaluating sympathetic function. The table-tilt test is valuable in gathering information on the neuro-cardio-vascular system, but loses its specificity for Sympathetic/Parasympathetic Imbalance. To illustrate, the table-tilt test often reveals POTS (Postural Orthostatic Tachycardia Syndrome). That is a valuable diagnosis, but again, we are talking about a lack of testing specificity because there are many factors, such as Electrolyte Imbalance, other than autonomic function involved with POTS.

What are the common causes of Sympathetic Noradrenergic Insufficiency underlying orthostatic dysfunction? Consider these causes, but with the understanding that you are also seeing some overlap with other Nutri-Spec imbalances, with the autonomic nervous system reacting to, and/or failing, in response to Electrolyte/Water Imbalance, Anaerobic/Dysaerobic Imbalance, Glucogenic/Ketogenic Imbalance, and Acid/Alkaline Imbalance:

- Iatrogenic causes – inappropriate or excessive prescription of medications:
    - Antidepressant drugs
    - Vasodilators (particularly α-adrenergic blockers used for either blood pressure control or for BPH)
    - Diuretics
    - Beta-blockers &/or Calcium channel blockers
- Type 1 and Type 2 Diabetes
- Water/Electrolyte Imbalances associated with either hypo- or hyper-volemina
- Cardio-Vascular-Renal stress caused by Rouleaux formation of RBCs, or loss of Zeta Potential of the blood colloid.
- Fatty Liver Disease
- Hormone disorders such as Adrenal Insufficiency and either Hypo- or Hyper-Thyroid
- Autoimmune Diseases
- Heart Failure
- Renal Failure

The medical profession remains largely unaware of orthostatic dysfunction and its countless ramifications. Physicians only recognize the possibility of the condition when it is extreme enough to cause recurring lightheadedness, dizziness, visual blurring, and even syncope. The symptoms you will see in your patients are:

- fatigue
- accompanied by malaise and likely a lack of motivation
- an aversion to exercise
- weakness
- nausea or lightheadedness after a large meal accompanied by gastrointestinal distension.
- nocturnal polyurea, which results from redistribution of peripheral blood to the centrum while recumbent. That symptom is all the worse in patients who experience a decrease in blood pressure while sleeping. These individuals often feel lightheaded when they first get up in the morning because of the volume loss overnight.

Another result you will see in response to your orthostatic test is a failure of the SBP to rise, yet the DBP does not fall as is typical in orthostatic dysfunction specifically caused by deficient sympathetic strength. Rather, the DBP will often rise. The combination of SBP drop and DBP rise gives a dramatic decrease in pulse pressure, and that failing pulse pressure is often accompanied by an extreme increase in heart rate of 12 or more bpm. This response to your orthostatic testing indicates likely Adrenergic Insufficiency, but compounded by imbalances in Electrolyte/Water control. And in those cases, if you let the patient remain standing for 3 minutes and check the heart rate once more, you will likely find that the pulse rate continues to rise to higher than it was in the immediate response to standing. Such patients may have hypovolemia and/or be in the early stages of POTS.

The incidence of medically diagnosable Orthostatic Hypotension is from 6% in young healthy adults to 35% or more in older individuals, but with a much higher incidence in diabetics and those with hypertension, or in those with autoimmune diseases. But among your patients, you will find a much, much higher incidence of functional Sympathetic Noradrenergic Insufficiency.

Research shows growing evidence that deficient orthostatic control predicts all-cause morbidity and mortality, as well as the incidence of cardiovascular disease. That predisposition to progressing pathology of all types among those with inadequate orthostatic response is critical, because ordinary blood pressure monitoring, and even ambulatory blood pressure monitoring, totally fail as prognosticators of future cardiovascular or cerebrovascular events, let alone all-cause morbidity and mortality.

Interestingly, the association between Orthostatic Hypotension and mortality in the population less than age 65 is more significant than in geriatric individuals. In other words, when you find Orthostatic Dysfunction in your patient, you have discovered a major red flag – a warning sign that your patient needs to make a significant commitment to better health.

Nutrition supplementation is essential. Your patients with Sympathetic Noradrenergic Insufficiency or Sympathetic Nor-Adrenergic Stress Failure need supplements to boost sympathetic function, and control any excess reactivity of the Vagus nerve.

Orthostatic failure is a common cause of fatigue and lightheadedness, and is one of the earliest and most debilitating symptoms of Sympathetic Noradrenergic Insufficiency. OH incidence in the elderly is as much as 40%, although some degree of OI is evident in a much higher percentage of the elderly, and is common in young adults with compromised health. OH is associated with increased mortality in the elderly, nearly doubling the death rate of those afflicted. OH in diabetics is frequently an early manifestation of Cardiac Autonomic Neuropathy. And in nondiabetics among the elderly, OH increases the mortality of Cardiac Autonomic Neuropathy by 25 – 50% within 10 years, and the heart failure death or hospitalization rate is also nearly doubled. In summary, orthostatic failure is an early warning signal of progressive pathology.

The low sympathetic tone underlying orthostatic failure has many causes. The sympathetic system is extremely sensitive both peripherally and centrally to any pathophysiologies that over-stress endothelial cells, immune cells, the microvasculature, or the renal glomerular filtration capacity. In Nutri-Spec terms, anything that accelerates either the Exogenous INFLAM-AGING pathway or the Endogenous INFLAM-AGING pathway can impair the Sympathetic Noradrenergic Response.

Orthostatic failure is caused by a failure of the baroreceptor reflexes and/or a declining sympathetic tone. The pathology there is a result of neurologic intracellular α-synuclein production and aggregation, which again, can be a result of either Exogenous or Endogenous INFLAM-AGING. The incidence of orthostatic dysfunction is much higher in diabetics of all ages.

The Scope and Ramifications of Your Sympathetic/Parasympathetic Analysis

There are countless studies from the literature showing the intimate association between the Sympathetic/Parasympathetic System and Immune System activity. These studies show that the Autonomic Nervous System is part of the First-Responder reaction to any environmental stress that triggers an immune response. A strong Immune System, a well-controlled Immune System, a balanced Immune System --- all depend on Sympathetic/ Parasympathetic ADAPTATIVE CAPACITY.

Only with Nutri-Spec Complex S and Complex P can you directly intervene in these neuro-immune processes.

Sympathetic/Parasympathetic reactivity in tandem with immune response is critical because there is virtually no patho-physiology that does not have a viscero-motor component, a somato-viscero component, and/or a neurovascular component. (And for the many of you reading this who are Doctors of Chiropractic, achieving Sympathetic/Parasympathetic Balance is the perfect adjunct to your neurologically based profession.)

Nearly all your patients will need Complex S and/or Complex P at some point along their road to --- LIVE STRONGER LONGER**.**

Now that you see all the health implications of Sympathetic/Parasympathetic Imbalance, you may be surprised at how few patients test as clearly Sympathetic or Parasympathetic dominant. The reason is that in many individuals by age 43, and in almost all your patients age 53+, both divisions of the autonomic nervous system have begun to fade. That is why a key component of your Stage Of Life Inflam-Aging Defense plan emphasizes a diphasic approach to Complex P and Complex S supplementation. Your goal is to pump up the diphasic cycle to approach the amplitude of what may have been a healthy teenager years ago – with both Sympathetic and Parasympathetic reactivity in healthfully high gear.

Clinical Pearl #1: If, while you are checking the 30-second heart rates, you detect an arrhythmia --- whether a "skipped beat" (pre-ventricular contraction or pre-auricular contraction) or any other irregularity of the heart rhythm --- that patient *absolutely* needs Complex S along with Taurine (and also likely needs Oxy Tonic).

Clinical Pearl #2: All your patients with sleep apnea need Complex S. We are not suggesting you "treat" sleep apnea with Complex S (Nutri-Spec does not "treat" any diseases.) Complex S is not only *not* a treatment for sleep apnea, as it will not help sleep apnea in the least. BUT --- sleep apnea creates an extraordinary nutrition need that puts the Sympathetic System under chronic stress. Only Complex S will meet that need, such that your patient can maintain neuro-immune and neuro-vascular health.

Clinical Pearl #3: All your patients who show a double rotary scoliosis need Complex S. Giving Complex S will not do a thing for the scoliosis. But research shows that the genetic neurological tendency to scoliosis is always accompanied by a genetic tendency to excess Sympathetic reactivity.

Clinical Pearl # 4: Asthma in Your Parasympathetic Patients

A classic example of inappropriate autonomic nerve reactivity is your asthmatic patient. In this case you have an oversensitive, over-reactive parasympathetic nervous system. The vagus nerve is in a state of oversensitivity and is thus subject to over-reactivity with very minimal provocation. Given the increased parasympathetic tone of the asthmatic patient, something as seemingly innocuous as a mild allergic exposure, an emotional stress, or a sudden change in temperature can elicit an extreme, even life-threatening, response.

A special note is warranted here about asthma. With your Nutri-Spec tests and supplements, you have the ability to have a most favorable influence on people suffering from asthma. There are, however, factors typical of your asthma patient that make it very difficult to interpret Nutri-Spec testing.

How do you analyze asthma in Nutri-Spec terms? Easily. Asthma is an over-reactive parasympathetic response. In particular, it is an over-reactive vagus nerve firing impulses into the bronchial tubes, causing bronchial constriction and increased mucous secretion. In asthma, you have about as direct a cause-and-effect relationship between a particular Nutri-Spec

imbalance and a particular disease as you will ever find. Not every Parasympathetic patient has asthma, but every one of your asthma patients has a Parasympathetic Imbalance.

For patients to have asthma, they must have two things. They must first have the Parasympathetic Imbalance which keeps the vagus nerve in a continuous state of facilitation or over-reactivity. Second, they need a trigger. In other words there must be some second factor that further irritates the already over-sensitive vagus nerve into the bronchial tree, precipitating the actual asthma attack. The trigger may take several different forms. The trigger may be an allergy (which itself may be the result of the histamine excess associated with a Parasympathetic Imbalance). The trigger may be a second Nutri-Spec imbalance. It can be an upper respiratory tract infection. It can be the stress of exercise, or a sudden change in the weather. It can be an emotional stressor. The trigger may, at times, be a chiropractic subluxation maintaining a facilitated state in the parasympathetic system. There is almost always a Prostaglandin Imbalance involved. In any event, the key to helping your asthma patients is to correct the underlying cause, the Parasympathetic Imbalance.

Once an asthma attack has been triggered, here is the sequence of events. The bronchial tree constricts and increases both its serous and mucous secretions. This causes increasingly labored breathing and also triggers a respiratory inflammatory reaction, which brings various prostaglandins (especially leukotrienes) into the picture. The increased airway resistance, plus the presence of excess fluid, plus the activity of prostaglandins causes an inflammatory response and a swelling of the tissues, which has now progressed to the point of a positive feedback loop. In other words, the asthma irritates the bronchial tree, which feeds back afferently to the central nervous system, and then back over the vagus to the bronchial tree, causing more constriction and more secretion, and thus more inflammation and prostaglandin activity, and so on and so on.

To break this positive feedback loop we, as Nutri-Spec practitioners, must decrease the underlying parasympathetic tone (and increase the antagonistic sympathetic tone), plus do whatever we can to eliminate the trigger. This includes correcting any other Nutri-Spec fundamental imbalances plus the Prostaglandin Imbalance (NO VEGATABLE OILS!!!).

From a Nutri-Spec perspective, there is something else very interesting going on in these asthma patients. Because an asthma attack decreases functional respiratory capacity, we see excess carbon dioxide accumulating in the system. This is, by definition, a Respiratory Acidosis. It is not at all uncommon to find a respiratory acidosis pattern upon testing these patients with Nutri-Spec. You must understand, however, that this Respiratory Acidosis is the result of, **not** the cause of the asthma.

The problem with this Respiratory Acidosis, of which we must be aware, is that to compensate for the Respiratory Acidosis the patient dumps chlorides into the urine. This loss of chlorides is very significant in asthma patients because it can tend to create a Metabolic Alkalosis or a Dysaerobic Imbalance. It turns out that Metabolic Alkalosis and the Dysaerobic Imbalances resulting from the loss of chlorides can subsequently further stimulate the vagus nerve. This triggers the whole cycle all over again.

Since asthma is always associated with a parasympathetic bronchial tree, all your patients with true asthma should test as Parasympathetic on Nutri-Spec testing. Many do – however, some do not. We are about to explain the reasons why some asthma patients do not test as Parasympathetic – but up front, you must understand that you will treat virtually all your asthma patients as Parasympathetic. You will **never** treat an asthma patient as Sympathetic, no matter if their Nutri-Spec tests seem to indicate a Sympathetic Imbalance. Furthermore, since any Respiratory Acidosis imbalance is secondary to the asthma, and treating it can push a person into a rebound chloride-deficient stimulation of the vagus nerve – you will **never** treat an asthma patient as a Respiratory Acidosis.

Now, let us look at the reasons why many of your asthma patients with a Parasympathetic condition do not test with Nutri-Spec as a Parasympathetic Imbalance, and may occasionally test as a Sympathetic Imbalance.

Since the asthma patient is Parasympathetic, you expect a slow pulse. Unfortunately, however, the asthma patient is often so hypoxic that the heart must beat faster in a desperate attempt to deliver oxygen. Therefore, the patient often shows a pulse that appears to be in the Sympathetic range. Likewise, looking at the orthostatic pulse increase, you may see the pulse jump up and stay up upon the stress demand of rising to the standing position.

With several tests for this imbalance leaning toward the Sympathetic side, you might well end up deciding this patient has no Sympathetic/Parasympathetic imbalance, or, (horrors!) even making the mistake of treating this patient as a Sympathetic Imbalance.

The situation is complicated even further by the fact that most asthma patients are taking medication, which further distorts the clinical test picture. Many asthma medications are powerfully anti-parasympathetic (which explains why they are effective). With chronic use of these anti-parasympathetic medications, patients will begin to show an elevated blood pressure, an accelerated pulse, an exaggerated orthostatic pulse response, and an exaggerated orthostatic blood pressure response. They will often also show an enlarged pupil and a white dermographics line. In other words, they will show a classic Sympathetic test pattern. You must understand that this pattern is the result of the medication only, and that it is the pattern you **want** the patients to show. As long as they are testing somewhat Sympathetic their asthma symptoms are being controlled to some degree.

If you make the mistake of treating asthma patients as Sympathetic as per a drug-induced Sympathetic test pattern, you will push them directly back into an extreme state of Parasympathetic Imbalance and precipitate an asthma attack. Again, **never treat an asthma patient as Sympathetic**.

If you are going to treat your asthma patients as Parasympathetic despite a Sympathetic test pattern, how are you going to monitor their progress? Most often there is at least one test in these patients that will still show a Parasympathetic tendency. Focus on this test as a means to monitor the patient.

There is another reason why some of your asthmatic patients do not test Parasympathetic and may even test as Sympathetic. If you look at the history of asthma over the last 40 years you

find that two things have happened – first, the incidence of asthma has increased dramatically; second, asthma, a condition that once was principally a childhood affliction that decreased or disappeared by the time a person reached adulthood, is now persisting throughout life, and is even affecting many members of the adult population who did not suffer asthma as children.

What has happened over the last 40 years to create the increase in frequency and duration of asthma? This increase has been shown to be associated with the immunization and the indiscriminate use of antibiotics in children, plus the increased incidence of estrogen stress in young women, particularly those on birth control pills. Estrogen is the leading cause of allergies and asthma in adult women.

Historically, asthma was found in patients who had an inborn tendency to a Parasympathetic Metabolic Imbalance. Now, because of the influence of immunizations and antibiotics, we have patients who do not have a systemic parasympathetic tendency, but rather a **localized** parasympathetic tendency in the specific neurological control of the bronchial tree. There is a possibility that the neurotoxic effect of vaccines damages an autonomic nerve ganglion which causes the localized parasympathetic over-reactivity and thus the asthma. While this has not been proved conclusively, a study published in The Journal of Anthroposophic Medicine demonstrates that the recovery from childhood diseases plays a role in the maturation of the immune system and helps the individual develop resistance to disease, including helping to prevent the development of asthma and other chronic diseases.(3)

Another study published in Science shows that childhood infections paradoxically protect against asthma, and that allowing respiratory ailments to run their course is essential to developing natural immunity. Suppressing this immune response leads to a state of neuro-immunological deficiency in the upper respiratory tract and a predisposition to asthma.(4)

So, to summarize, many of our modern day asthma conditions are associated with a localized parasympathetic over-reactivity rather than a systemic Parasympathetic Imbalance. To control the asthma in these patients you must still treat them with Complex P, tyrosine, and very often with magnesium chloride as per your QRG. However, you must be cautious because these patients can very easily be pushed into a systemic state of Sympathetic Imbalance, even as their bronchial system is struggling with its own parasympathetic condition.

One other note on your asthmatic patients is that many of these people also tend to be Anaerobic. This imbalance can also be hidden by asthma medications, many of which push a patient more Dysaerobic. Look for an Anaerobic tendency in these patients and treat it with Oxygenic A, Oxy Tonic, and Diphasic A.M.

While you will be of tremendous help to your asthma patients, most of them will continue to need some pharmacological intervention, at least from time to time. Which medications are the most beneficial and which ones are damaging? The best medications for your asthma patients are those which are both anti-parasympathetic and anti-anaerobic. These are the epinephrine analogs. Most of them are provided in the form of inhalers. These constitute the most logical choice for asthma medication since they not only directly impact the symptom but are also addressing the underlying metabolic imbalances. Also beneficial are the leukotriene inhibitors.

Another common medication used for asthma is glucocorticoids. These steroids are a very poor choice in that while they may give short term relief, they actually exacerbate the Parasympathetic and the Anaerobic Imbalances that cause the asthmatic condition. We could condone the use of the medication for short-term crisis relief despite its side effects **if there were no other alternative**. However, since epinephrine is just as effective or more so at controlling an asthmatic crisis, there is no justification for using steroids.

In a crisis situation, such as when a severe asthma attack necessitates hospital emergency care, a shot of epinephrine (adrenalin) should be the first treatment choice. This used to be standard practice, but has been replaced by the use of prednisone. Prednisone "works" symptomatically by virtue of its anti-inflammatory and anti-prostaglandin effect, while actually exacerbating the patient's underlying Parasympathetic (and Anaerobic) tendency. Only asthmatics with a Dysaerobic tendency derive more short-term good than long-term harm from steroids.

Two additional comments on asthma medications are in order. First, many asthma medications and allergy medications contain sodium benzoate. It has been found that this common cough, cold, and allergy medication actually causes asthma in many patients. A study published in The Archives of Pediatrics showed that children with asthma had their condition clear completely as soon as they discontinued sodium benzoate containing medication.(5)

Finally, let us consider allergy shots, which are often used in the belief that a decreased sensitivity to allergens will decrease the frequency or severity of asthma attacks. We love it when establishment researchers set out to prove themselves right and end up stubbing their toe in the process. A study published in the New England Journal Of Medicine, which was designed to prove the efficacy of allergy shots in asthma patients, proved exactly the opposite. When the study showed absolutely no benefit from allergy shots, and likely harm, the researchers were dumbfounded, and in their state of disbelief urged caution in accepting their own study's conclusions.(6)

So – what will you do with your next asthma patient? First, identify all the Nutri-Spec fundamental imbalances. Second, treat all those imbalances plus a Parasympathetic Imbalance, regardless of whether the patient tests as Parasympathetic. To monitor that patient, use whatever tests lean toward the Parasympathetic side. Finally, put the patient on the **Prostaglandin Dietary Recommendations** in addition to the Nutri-Spec Fundamental Diet. This means the patient must strictly avoid salad dressings, margarine, mayonnaise, nuts and nut butters, and all fried foods. The patient should also be following the Parasympathetic Diet, which means a decrease in carbohydrate intake with particular attention to decreasing fruit and other forms of sugar, plus strict avoidance of juices.

Your only additional responsibility with these patients is to check their medications. Make sure they are taking no sodium benzoate containing medications. Also make sure that if they are using an inhaler it is an epinephrine analog and not a steroid.

Again, the key to asthma is correcting the Parasympathetic Imbalance and keeping the other Nutri-Spec imbalances under control as well. Most typically the other imbalances include a Metabolic Alkalosis, or an Anaerobic or Dysaerobic Imbalance.

Dietary Recommendations

The dietary recommendations for both Sympathetic and Parasympathetic Imbalances require that these patients strictly avoid concentrated sugars of any kind, particularly juice and other sugar drinks (fructose). This consideration is so important for Parasympathetic patients that they must include strict avoidance of fruit as well.

Blood sugar irregularities are typical of both Sympathetic and Parasympathetic Imbalances. But the problems associated with dietary sugars and starches are not limited to those directly related to energy production and blood and brain sugar levels. Water/Electrolyte balance is affected as well. Excess carbohydrate intake causes sodium retention in both your Sympathetic and your Parasympathetic patients (and to some extent in all people), and, carbohydrates cause loss of potassium in your Sympathetic patients.

Both these imbalances need to increase their intake of the high nutrient density foods, i.e., meat and vegetables. The Parasympathetic patient has a greater need for high adenine type meats such as beef, lamb, poultry dark meat, seafood, and organ meats. A high protein, low carb diet will increase the vitality of a Parasympathetic patient, while eliminating Parasympathetic symptoms, and assuring loss of excess body fat. The Sympathetic patient does better emphasizing fish, poultry, dairy and eggs as a source of protein. Carbohydrates are no problem for Sympathetic patients as long as sugar foods are avoided.

Recommended Supplementation

Your Sympathetic and Parasympathetic patients will often present you with an extraordinarily complex symptomatic picture.

Now let us consider just what it is in your Nutri-Spec supplements for these imbalances that enables you to expect improvement in both your objective indicators and in the symptomatic picture in such extreme patients.

First look at Complex S. Your Complex S contains a complete blend of nutrients which have a specific normalizing effect on the characteristic problems of a Sympathetic Imbalance:

- excess vaso-constriction
- excess renin activity
- elevated blood sugar
- nervous tension
- deficient immune response
- deficient GI secretion and motility

These nutrients include vitamins, minerals, trace minerals, and amino acids designed to push your patient powerfully from the Sympathetic side of the balance scale to the Parasympathetic side, **without** creating any other metabolic imbalances.

If you look at the label on your Complex S, one nutrient you should note is the amino acid **arginine**. We could easily devote several pages to this amazing nutrient – but here are the highlights:

Research published in both The Lancet and in The Journal of the American College of Cardiology shows that arginine is used in the body to produce nitric oxide.(7,8) Why is this important? Several studies have shown that the role of cholesterol in cardiovascular disease is not so much a matter of clogging the arteries, but rather of causing both spasms of the blood vessels and a fibrous thickening of the vessel walls. Nitric oxide, made from arginine, allows the blood vessels to relax and dilate, and also decreases the fibrous thickening that we know as atherosclerosis. (However, nitric oxide in even slight excess can cause a decrease in mitochondrial energy production in cells throughout the body, and will cause cellular death in the brain, heart, and blood vessels. It exacerbates all inflammatory diseases. It can accelerate the development of cancer. Clearly, Arginine must be supplemented responsibly.)

You will find that many of your Sympathetic patients have cardiovascular disease. Arginine is one of several nutrients in your Complex S that will reverse cardiovascular disease, and restore the blood pressures, pulses, and dermographics reflex to normal.

Some of the other benefits of arginine should be mentioned:

- It decreases angina pain
- It has anti-cancer activity
- It is beneficial in certain types of arthritis
- It promotes wound healing
- It restores sexual functioning to impotent men
- It helps detoxify the liver
- It assists in generating creatine phosphate, a compound required for muscle contractions and stability of the membrane that surrounds the heart
- It helps produce collagen required for strong joints and youthful skin
- It helps maintain normal growth hormone levels

HOWEVER, arginine is among the several amino acids that dramatically accelerates the Endogenous Inflam-Aging pathway. For that reason, we have significantly decreased the amount of arginine in Complex S, relying instead on the other nutrients to carry much of the load. Additionally, you should supplement all your patients age 43+ who need Complex S with at least 2 Rejuvenator first a.m.

Since the excess catecholamines typical of your Sympathetic patients have a catabolic effect, your Complex S is loaded with anti-catabolic nutrients. Many of the minerals and trace minerals in Complex S have an anti-catabolic effect, preserving the integrity of brain tissue as well as muscle and skin.

You will note that Complex S contains several forms of **magnesium**. Magnesium is vitally important to the Sympathetic patient because of its vaso-dilating effect, because of its effect on stimulating GI secretion and motility, because of its favorable impact on the heart muscle, and because of its importance in regulating electrolyte levels. Magnesium meets the extraordinary nutrition caused by diabetes, a condition commonly associated with Sympathetic Imbalance.

Diabetes is an important consideration for your Sympathetic patients. Most of your insulin-dependent diabetics will show a Sympathetic Imbalance upon Nutri-Spec testing – and virtually all your sympathetic patients will have a tendency to become diabetic during a lifetime of excess sugar consumption.

An amino acid beneficial for many of your Sympathetic patients is **taurine**. We have described taurine in previous chapters on Electrolyte Stress and Anaerobic imbalances. Its anti-sympathetic effect is associated primarily with two of its functions:

- It is a natural calcium channel blocker (thus keeping blood pressure and pulse under control).
- It facilitates the upper GI function of the liver and gall bladder.

Constipation is a problem that plagues many of your Sympathetic patients. Complex S is in itself very beneficial in that regard. However, there are some Sympathetic patients who have a strong Anaerobic component to their problem. In these cases, supplementation with **Oxy Tonic** will do the trick, if it is indicated by a tendency to elevated urine pH.

The neuro-vascular component of a Sympathetic Imbalance can often lead to rebound **migraine headaches**. In these cases, you will often find an indication for supplementation with **Oxygenic D-Plus.** Sympathetic migraines are also benefited by extra **Taurine**. Extra taurine is also indicated if your Sympathetic patient has **elevated cholesterol** or **cardiac arrhythmia**.

Oxy-Max will reverse the oxidative stress typical of many Sympathetic patients. It, along with Oxy D-Plus, will lower elevated cholesterol. Oxy-Max will also benefit sympathetic-related diabetes, allergies, and arthritis.

Turn your attention now to the Parasympathetic page of your QRG. Complex P contains the blend of nutrients which have a specific normalizing effect on the consequences of a Parasympathetic Metabolic Imbalance, which include:

- blood and brain sugar problems
- excess GI motility and secretion
- hypotension and poor circulation
- allergies and asthma
- low energy; nervous tension; and depression

Looking at the ingredients in your Complex P, you see the list headed by a few of our critically important amino acids – **phenylalanine** and **glutamine**. The benefits of these amino acids to **brain function**, to **energy levels**, and to **GI function** are critical.

Some other noteworthy items on the list of Complex P ingredients include two forms of **calcium** – glycerophosphate and orotate. Each form has its own distinct impact on the metabolic dysfunctions associated with Parasympathetic Imbalance.

You will also find **chromium as polynicotinate –** the most biologically active form of chromium – for its impact on maintaining normal **blood and brain sugar levels**.

Complex P also contains therapeutic doses of the bioflavonoid **rutin**. Bioflavonoids are powerfully protective nutrients. But there is something about the bioflavonoids added to almost all supplements that you must know. You will often find an ingredient in other companies' products listed as "bioflavonoids." This is a tell-tale sign that this product is cheap garbage. Do you know what constitutes this "bioflavonoids" ingredient that nearly all nutrition companies use in their products? It is nothing more than a crude concentrate of lemon peels. It contains approximately 2 milligrams of biologically active bioflavonoids per 100 milligrams of the so-called bioflavonoid substance. Remember that – only 2 milligrams of good stuff for every 100 milligrams on the label. Compare that with your Complex P, which has a clinically significant quantity.

Phos Drops will give a very nice boost to many of your Parasympathetic patients who show a tendency toward an A**lkalosis.**

Sodium Glycerophosphate will ease the anxiety associated with a Parasympathetic Imbalance, yet will give a boost if fatigue is a problem.

Oxy D+ will help control GI inflammation, correct the vascular dysfunction, and will lower cholesterol if need be.

The out-of-control **vaso-dilation** typical of a Parasympathetic Imbalance can give these patients migraine headaches. These migraines often have a Dysaerobic component as indicated by a low urine pH. When that is the case, you can use **Oxygenic D-Plus** for these Parasympathetic migraines.

Finally, we must consider asthma as a Parasympathetic condition. Many of your asthmatic patients will benefit from an additional dosage of magnesium chloride because of its impact as a broncho-dilator, plus its ability to replace the chlorides dumped by the kidneys during the Respiratory Acidosis phase of an asthma attack.

References

1. Pottenger, Francis M.: "Symptoms of Visceral Disease," 4th Ed., Mosby, St. Louis, Missouri, 1930

2. Lowe, Phillip, A., "Clinical Autonomic Disorders," Little Brown & Company Boston, 1993.

3. "The Immunization Campaign Against Measles, Mumps and Rubella, Coercion Leading to Uncertainty: Medical Objections To A Continued MMR Immunization Campaign In Switzerland," Journal Of Anthroposophic Medicine, Spring, 1992.

4. Science, Jan 1997.

5. Petrus, M., et al, "Asthma Induced By Benzoate Contained In Some Foods & Anti-allergic Drugs," Archives Of Pediatrics, 3:984-987, 1996.

6. New England Journal Of Medicine, Jan 30, 1997.

7. The Lancet, Vol. 337, 645-46, 1991.

8. Wang, Bing-Yin, et al, "Arginine Restores Nitric Oxide Activity and Inhibits Monocyte Accumulation After Vascular Injury In Hypercholesteremic Rabbits," Journal Of The American College Of Cardiology, 28:1573-1579, 1996.

Additional References:

Gary L Murray, et al. *(r)Alpha Lipoic Acid is a Safe, Effective Pharmacologic Therapy of Chronic Orthostatic Hypotension Associated with Low Sympathetic Tone*. Int J Angiol. 2019 Sep;28(3):188-193. https://pmc.ncbi.nlm.nih.gov/articles/PMC6754261/

Philip L. Mar, et al. *Neurogenic Hyperadrenergic Orthostatic Hypotension – A Newly-Recognized Variant of Orthostatic Hypotension in Older Adults with Elevated Norepinephrine*. Clin Sci (Lond). 2015 Jul;129(2):107-16. https://pubmed.ncbi.nlm.nih.gov/25706983/

Artur Fedorowski, et al. *Orthostatic Hypotension: Management of a Complex, but Common, Medical Problem*. Circ Arrhythm Electrophysiol. 2022 Mar;15(3):e010573. https://pubmed.ncbi.nlm.nih.gov/35212554/

William P. Cheshire, et al. *The Physical Examination as a Window into Autonomic Disorders*. Clin Auton Res. 2018 Jan 2;28(1):23–33. https://pmc.ncbi.nlm.nih.gov/articles/PMC8921977/

Fabarizio Ricci. *Orthostatic Hypotension: Epidemiology, Prognosis and Treatment.* J Am Coll Cardiol. 2015 Aug 18;66(7):848-860. https://pubmed.ncbi.nlm.nih.gov/26271068/.

Janice M Fritsch-Yelle, et al. Subnormal Norepinephrine Release Relates to Presyncope in Astronauts After Spaceflights. J Appl Physiol 1996 Nov;81(5):2134-41. https://pubmed.ncbi.nlm.nih.gov/8941538/.

Derek S, et al. Hypovolemia and Neurovascular Control During Orthostatic Stress. Am J Physiol Heart Circ Physiol. 2002 Feb;282(2):H645-55. https://pubmed.ncbi.nlm.nih.gov/11788414/

Damian J Dyckman, et al. Glycerol-Induced Fluid Shifts Attenuate the Vestibulosympathetic Reflex in Humans. Am J Physiol Regul Integr Comp Physiol. 2011 Mar;300(3):R630-4. https://pubmed.ncbi.nlm.nih.gov/21178123/