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Excessive parasympathetic responses to sympathetic challenges: a treatable, hidden, dynamic autonomic imbalance

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Abstract *Background:* A common assumption with autonomic assessment is that one branch opposes the other. With independent measures of parasympathetic (P) and sympathetic (S) activity, based on concurrent time-frequency analysis of respiratory activity and heart rate variability, this assumption has been challenged. Clinical observations of unprovoked P-excess during S-stimulation have been associated with treatable, abnormal responses.
Method: Serial autonomic profiling of 12,967 patients was performed using the P&S method (ANX-3.0 Autonomic Monitor by ANSAR Medical Technologies, Inc., Philadelphia, PA) over a five-year period. Treatment protocols are very low-dose and depend on patient history. For cardiovascular disease patients, Carvedilol was prescribed. For non-CVD patients, Nortriptyline was prescribed. In some cases where end-organ effects were not yet presented or relieved, patients were weaned of therapy once PE was relieved. Alternative therapies included Specific Chiropractic Adjustment, better known in the literature as Chiropractic Manipulative Therapy and intensive zero-impact, cardiovascular exercise.
Results: PE patients present with normal HR and BP and no other apparent symptoms at rest. However, they reported symptoms of: sleep difficulties, palpitations, poor peripheral circulation, general malaise, depression (often with anxiety or ADD-like symptoms), frequent headache or migraines, menopause difficulties in women, hypothyroidism, cognitive difficulties,

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gastrointestinal upset, persistent weight-gain, and dizziness after standing.
Conclusion: Normalizing PE, regardless of method, stabilizes the patient, relieves symptoms, improves quality of life, and improves patient outcomes.
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Abbreviations

ANS	autonomic nervous system
BP	blood pressure
CVD	cardiovascular disease
CMT	Chiropractic Manipulative Therapy
HR	heart rate
HRV	Heart Rate Variability
P	parasympathetic
PE	parasympathetic excess
RA	respiratory activity
RSA	respiratory sinus arrhythmia
S	sympathetic
SB	Sympathovagal Balance
SCA	Specific Chiropractic Adjustment
SE	sympathetic excess
SW	sympathetic withdrawal

Introduction

Clinical observations of unprovoked parasympathetic excess (PE) are associated with abnormal clinical and pathophysiologic responses. PE may occur with or without abnormal sympathetic (S) activity. Chronic conditions such as diabetes,[1–5] thyroid disease,[6] kidney disease,[7] cardiovascular disease,[11,12] demyelinating and inflammatory neurological diseases,[8] some dementia,[9] depression, and altered psychological states[9] may cause autonomic imbalance and associated P-dysfunction. Severe acute conditions may precipitate PE, including trauma, injury, infection, surgery, cancer, and myocardial infarction. Preliminary evidence suggests that severe or chronic exposure to chemicals, cold, and allergens may induce PE as well as multiple pregnancies for women. Stress, excess caffeine, nicotine, and other chemical[10,11] and environmental exposures affect autonomic balance. History assessment indicates that genetically mediated, or congenital, PE may be evidenced as colic in infants followed by intermittent (between developmental cycles during the childhood and teenage years) sleep difficulties, or depression with anxiety, or ADD. PE is associated with difficult to manage patients including those with difficult to control blood pressure (BP), blood glucose, and hormone levels (*i.e.*, thyroid, estrogen, or growth hormones), and persistent weight-gain.

Establishing PE may help clarify a diagnosis when patients present with multiple, confounding or conflicting symptoms (*e.g.*, hypertension with depression, depression with anxiety, hypotension with nighttime sleeplessness, and CRPS) and provide a more integrated approach to therapy. Disease may cause P&S imbalance, for example pain causes S-excess (SE) which may lead to early hypertension. Symptoms appearing as disease may be caused by P&S imbalances, such as when dizziness upon standing (orthostasis or syncope) is caused by PE with secondary S-dysfunction. Therefore, it seems reasonable to hypothesize that P&S imbalances such as PE or SE are separate and distinct dysfunctions. A single agent, however, may often address both the primary disease and the P or S disorder, and PE may be treated directly. Once PE is relieved, often the patient is more stable and the primary disease(s) may be treated more aggressively.

Often PE is not detected at rest, and therefore not detected with the standard office physical. Furthermore, it is often associated with (secondary) SE and associated with symptoms (high BP, palpitations, anxiety). Remember, P-activity established the threshold around which S-activity responds. As a result, SE is treated and often the patients are (seemingly) unresponsive or become worse. Clinical evidence has shown that detecting, and thereby treating, PE will lead to normalized SE which will lead to relief of both SE-related symptoms and PE-related symptoms.

P&S Monitoring has documented failures in the reactive push—pull dynamics within the ANS.[12–15] Measures based solely on Heart Rate Variability (HRV) or beat-to-beat BP often fail to isolate P- from S-activity.[13–15,20,21] Independent, simultaneous P&S measures are critical to understanding the true nature of autonomic dysfunction and its clinical implications.[16–21] Documentation of P&S activity has provided more insight into many commonly observed clinical conditions. PE is often the primary autonomic disorder and S-abnormalities appear to be secondary.

Methods

Serial P&S profiling of 12 967 patients (7424 females, 57.25%) was performed (ANX-3.0 Autonomic Monitor by ANSAR Medical Technologies, Inc., Philadelphia, PA). Patients ranged in age from 6 to 100 years. Patients with high quality ectopy were omitted from this analysis. Data were collected over a five-year period at 19 primary care and ambulatory clinics. Patients were followed for at least 18 months as a matter of routine, based on their primary diagnosis. EKG and respiratory data were collected concurrently, and analyzed to compute independent, simultaneous P&S activity measures (the P&S Method).[20,21] P&S assessment was based on a clinical study that included (in order): 1) five-minute of rest (initial baseline); 2) one

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