



The autonomic nervous system controls many of the functions vital to life—like blood flow, breathing, heart rate and digestion. What is research uncovering about disturbances to this system in people with CFS?

Autonomically Speaking

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When it comes to body systems, the central nervous system, with its higher brain functions, tends to get all the press. But the autonomic nervous system (ANS) is just as important. Turn the central nervous system off and the autonomic nervous system can keep your heart beating and blood flowing for decades. Turn the ANS off and you need highly sophisticated machines to keep you alive.

While we're using our central nervous system to come up with our ideas and dreams, our ANS is quietly humming away, ensuring we have the resources to carry out them out. When we encounter any kind of challenge, from standing to moving to digesting food, the ANS directs our system to meet that challenge.

Understanding the autonomic nervous system

The ANS is the part of the nervous system that maintains homeostasis (stability) in the body. Through its control of the smooth muscles lining our blood vessels and organs, the ANS affects many of the functions that support daily life. Most of these functions—such as heart rate, blood pressure, respiratory rate, digestion, salivation, perspiration, urination and diameter of the pupils—are performed without conscious thought.

For example, the ANS is constantly opening and narrowing blood vessels across the body in order to energize some tissues and de-energize others. Without the ANS narrowing the blood vessels in our legs every time we stand, we would quickly get dizzy and faint. Without it opening our blood vessels when we exercise, our muscles couldn't get the blood they need. Without it adjusting the blood flow to our skin, we would quickly overheat every time it gets hot or get chilled every time it gets cold.

AT-A-GLANCE ▶

- The autonomic nervous system maintains homeostasis (stability) in the body, governing both the “fight or flight” response and the “rest and digest” mechanism.
- CFS research into blood flow, breathing, heart rate variability, neuroimmune dynamics and sleep suggest disturbances in the autonomic system.
- People with CFS may have an overactive “fight or flight” response and an underactive “rest and digest” mechanism.

There are two parts to the ANS. When we encounter a stressor, the sympathetic nervous system (SNS), or the “fight or flight” system, jumps in. When we’re at rest or asleep, the parasympathetic nervous system (PSNS), or the “rest and digest” system, is dominant. These two systems work in sync; when one is turned on the other is generally turned off. Their finely tuned operations allow us to rise to the physical challenges life presents, recover from them and then rise to face them again.

Exploring the connection to CFS

Accumulating research indicates that something may be awry in the ANS of CFS patients. Specifically studies suggest that the “fight or flight” branch of the ANS (the SNS) may be overactivated and that the “rest and digest” (the PSNS) branch may be underactivated. This could impose two broad stresses on CFS patients: the stress of being “on” all the time and the stress of never being able to really relax and rejuvenate. This concept of an overactive SNS and underactive PSNS can be seen in various facets of CFS research on the autonomic system.

Follow the blood. An excellent way to see the ANS in action is to follow the blood, and a good place to start is the blood’s driver, the heart. The ANS continually adjusts the heart rate and the force of the heart’s contraction as it responds to our need for energy. Three abnormalities found in people with CFS and generalized chronic fatigue—overly rapid heart rates, reduced filling (diastole) speed and increased contraction speed (systole)—suggest the SNS may be working too hard and the PSNS may not be working hard enough.

Other studies suggest that the process of moving blood to where it’s most needed has gone off-kilter. The SNS narrows and the PSNS opens blood vessels across the body by controlling the activity of the smooth muscles lining the arterioles. Several studies suggest that in people with CFS, the SNS has clamped down on those smooth muscles, narrowing the blood vessels and perhaps reducing blood flow to the muscles and organs.

But that’s not all. Because our blood volume, in part, reflects the amount of blood vessel volume available, those narrowed blood vessels could contribute to the low blood

volume found in many CFS patients as well. Vanderbilt University researcher Italo Biaggioni, MD, is currently examining the blood volume/SNS connection in CFS.

Breathing. The ANS doesn’t just help move the blood around, it’s also responsible for ensuring that the blood is healthy. Autonomic sensors are continually measuring blood oxygen and carbon dioxide (CO₂) levels. CO₂, a waste product, is released into the air when we exhale, but several studies suggest that when some CFS patients stand, they unconsciously begin to hyperventilate—a breathing pattern that leads to abnormally low blood levels of CO₂. Intriguingly this condition, called hypocapnia, induces some of the symptoms associated with CFS, including fatigue, stiff muscles and pain. Hyperventilation is also known to increase SNS activity.

Heart rate variability. Perhaps the best window into the operation of the ANS comes from an examination of the complex electrical signals in the heart. Many studies examining these signals, which researchers call “heart rate variability” (HRV), suggest increased SNS and decreased PSNS activity is present in CFS.

Researchers are just beginning to understand what these electrical signals mean but similar HRV readings in fibromyalgia patients and in highly fatigued multiple sclerosis, liver disease and post-cancer patients may reflect processes associated with the production of fatigue. Low HRVs also appear to be associated with poorer coping during stress, poor mental endurance and more difficulty controlling emotions.

Sleep. If the “fight or flight” branch of the ANS is overactivated, does this mean CFS patients don’t get enough “rest and digest” time? Sleep is where we spend most of our rejuvenation time, and two recent sleep studies suggest that the ANS abnormalities seen during the day also occur during sleep. One recent study, done by researchers at the Centers for Disease Control and Prevention (CDC), found three indications of increased SNS activity in CFS patients during sleep—low HRVs, increased heart rates and increased norepinephrine levels.

The neuroimmune connection. The connection between the ANS and immune function has hardly begun to be addressed in CFS, but the possibilities

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are fascinating. Chronic SNS activity is known to down regulate the proinflammatory (Th1) immune response and to decrease natural killer cell activity, two dynamics that are present in CFS. University of Miami researcher Mary Ann Fletcher, PhD, is currently examining whether immune abnormalities in CFS first affect the SNS and then boomerang to damage natural killer cells.

On cause and response

Why the SNS might get locked into the “on” position in CFS is unclear, but some researchers are focusing on a part of the brain called the central autonomic network. This region not only affects our autonomic functioning but also our thinking and emotions. It contains three parts of the brain: the basal ganglia, anterior cingulate and hypothalamus. CFS studies using functional magnetic resonance imaging (fMRI) have found anomalies in these areas. These are also the areas of the brain with the highest levels of the neurotransmitter serotonin—a substance Japanese CFS researchers and others are investigating as a potential factor in the cause of CFS (see page 4).

Fighting back. If the ANS is damaged in people with CFS, there may be a very simple way of restoring it to some degree. Breathing is one of the few processes that is automatic and yet under some voluntary control. A good deal of evidence suggests that we may be able to at least partially return balance to the ANS by using controlled breathing techniques. Slow, deep breathing has been shown to cause a generalized decrease in the excitatory pathways of the brain.

This concept was recently borne out in fibromyalgia, an illness with the same HRV patterns as CFS. After using a biofeedback machine to identify a breathing rate associated with improved HRV levels, FM patients were able to moderately improve their functioning, mood and sleep quality and to reduce their pain simply by instituting this breathing pattern a few times a day.

Indeed many CFS physicians including Drs. Paul Cheney, Charles Lapp, Richard Podell and Fred Friedberg, advocate using techniques that slow down the breathing rate. Lapp notes that CFS patients often exhibit postural and muscle problems (hunched shoulders, shallow chests, neck and head pain) that can contribute to rapid breath rates and impede the kind of slower, deeper breathing associated with optimal CO₂ release, relaxation and good health. He recommends neck and shoulder stretches to loosen up these areas.

In some ways the effectiveness of these techniques may not be surprising. Studies now suggest that both of the

biologic controls of the stress response mechanism—the HPA axis and the sympathetic nervous system—may be affected in CFS. If this is so, then increasing the relaxation response and downgrading the stress response should be beneficial. In his book *Fibromyalgia and Chronic Fatigue Syndrome*, Friedberg suggests that doing 30 minutes or more of relaxation exercises such as focused breathing, progressive muscle relaxation or guided imagery before sleeping can profoundly improve a CFS patient’s sleep and enhance energy.

Much left to be done

Clearly there’s much left to learn about the role the autonomic nervous system plays in CFS. Are CFS patients locked into a kind of chronic stress response? Do the “wired but tired” feelings so often reported by CFS patients come from their inability to fully enter the “rest and digest” mode? Could the immune problems in CFS reflect SNS abnormalities or vice versa? Is SNS activation causing the low blood volume in CFS?

There’s also the problem of patient subsets. Researcher Julian Stewart, MD, PhD, believes one set of CFS patients (what he terms “high-flow”) has trouble opening the blood vessels while another (“low flow”) has trouble narrowing them. He and University of Medicine and Dentistry of New Jersey researcher Benjamin Natelson, MD, found that CFS patients, as a group, displayed four responses to a specific blood flow/blood pressure test called a tilt test, each of which could have been caused by a different dysfunction in the ANS.

It’s also important to note that some of the ANS abnormalities in CFS patients are fairly subtle. The CO₂ levels, for instance, tend to fall in the low-normal range, and researchers still aren’t completely sure what to make of the complex HRV signal activity.

The ANS may or may not play a major role in CFS, but its long reach and the many intriguing connections between it and the abnormalities found in the illness make it a part of the body that researchers are sure to keep examining in the future. ■

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