UNDERSTANDING THE NERVOUS SYSTEM

Why the sympathetic system stays switched on — even in sleep

Educational material from Anna Elitzur, Medical Advisor · Welltory ELC Community

A QUESTION FROM THE COMMUNITY

"I run sympathetic-dominant almost all the time. The only way I reach meaningful parasympathetic activation is through prolonged, intentional effort — removing sensory input, lying flat, breathwork — and even then it does not always work. The moment I stop, sympathetic dominance returns, even during sleep. Is there a meaningful path toward the parasympathetic system engaging on its own, when it is needed, and staying engaged without constant deliberate effort?"

This is one of the most common experiences people bring to the community, and it deserves a real physiological explanation. When the sympathetic side stays active around the clock, even at 1 AM in deep sleep, that is not a matter of insufficient effort or the wrong relaxation technique. It has biological roots we can actually point to, and understanding the mechanism is the first step toward working with it.

A helpful way to picture it

Think about everything that keeps a mind on edge: money worries, work pressure, relationship strain, the news cycle. Several different stressors, all pulling at once, from different directions. The body has its own version of this. There are several separate physical sources that each push the sympathetic system toward "on," and most people in our group carry more than one of them at the same time. That is why a single calming tool rarely settles the whole picture — one source is quieted while the others keep signaling underneath.

The main physical sources, kept short

1. **Blood volume and pooling.** Many people with orthostatic intolerance run on a lower circulating blood volume, often around 13% below typical. The heart and brain read low volume as a perfusion problem and call up the sympathetic system to keep pressure and flow going. Standing makes this sharper as blood pools in the legs and belly.

2. **Small fiber nerves.** The tiny nerves that tell blood vessels to tighten are damaged in roughly half of people with ME/CFS and POTS. When vessels cannot constrict properly, blood pools, perfusion drops, and the body compensates centrally by raising sympathetic tone. Standard nerve tests miss this; it takes a skin biopsy or a QSART test to see it.

3. **Adrenergic autoantibodies.** Many people carry functional antibodies that sit on the receptors controlling heart rate and vessel tone and keep stimulating them. This runs in the background continuously and is completely independent of any breathing practice or relaxation.

4. **A hyperadrenergic pattern.** In a sizable share of people, the central sympathetic centers themselves are set too high. This shows up as standing norepinephrine over 600 pg/mL, sometimes blood pressure rising on standing, and migraines. With this pattern, calming the system from the bottom up meets a steady push from the top down.

5. **Neuroinflammation.** Imaging shows activated immune cells in the brainstem and other regions that govern the autonomic system. One of these regions is a major hub for vagal control, so inflammation there directly dampens the parasympathetic side. Inflammatory signals like IL-6 also turn up sympathetic input system-wide.

6. **The stress-hormone axis.** Across many studies, people with ME/CFS show a lower morning cortisol and a flattened daily rhythm. Low cortisol and low vagal tone reinforce each other: the body clears inflammation less well and stays in a low-grade “on” state to keep functioning at all.

7. **Mast cells.** Mast cell activation is a common companion to these conditions. Mast cells sit right next to autonomic nerves, and the histamine and inflammatory mediators they release destabilize vessel tone and crank up sympathetic reactivity. Histamine is also a wakefulness signal, so a nighttime release can trigger arousal and a racing heart without a full waking.

Why nights are often the worst

In a healthy night, the autonomic system shifts into parasympathetic mode: heart rate drops, blood pressure dips by 10 to 20%, vessels relax. Several of the sources above interfere with that shift. Lying flat does not refill low blood volume, so the compensation continues. Mast cell flares release histamine that fragments sleep. A disrupted circadian rhythm lowers nighttime vagal tone. And the normal nighttime blood pressure dip often disappears entirely. So a reading that shows the sympathetic side near its maximum and the parasympathetic side near its floor in the middle of deep sleep reflects a body that never got the signal to stand down.

Why breathwork helps in the moment and then fades

This part matters. Cyclical sighing, slow breathing, or lying flat produce a real physiological shift through the baroreflex and the vagus nerve. That is genuine biology, not imagination. The reason it does not hold is simple: it works on one layer while the deeper sources keep running. The antibodies keep stimulating receptors. Low volume restarts the compensation the moment a person stands. Damaged nerves still cannot do their job. Inflammation keeps the central system primed. Breathwork is a real and useful layer — it is one layer among several.

There is also a difference worth knowing: in research, slow breathing partially normalized autonomic measures in post-COVID but did much less in ME/CFS, where the baroreflex disruption runs deeper. So when breathing tools seem to do less than the internet promises, that finding may be part of why.

Is there a path toward the parasympathetic engaging on its own?

Honest answer: the steady shifts usually come from working on the upper sources, often several at once, with a clinician who can identify which ones are driving an individual picture. The general directions people and their doctors work with:

- **Volume support.** Higher sodium and fluids, and compression with abdominal coverage, raise circulating volume and reduce the standing tachycardia that keeps the sympathetic system busy. A 500 mL water bolus also triggers a pressor reflex within 15 to 30 minutes.
- **Pacing and PEM protection.** For this group this is the single most important non-drug lever. Every push past the energy envelope provokes a fresh wave of sympathetic activation and immune signaling, which feeds the cycle. Staying inside the envelope breaks that loop.
- **Addressing the immune layer** where it is present, since the antibody and inflammation sources are often what keep the system primed.
- **Vagus nerve stimulation (tVNS)** is a promising direction under study. In early trials it lowered adrenergic antibody levels and inflammatory signals and reduced standing heart rate, so it appears to reach some of the upstream sources.
- **Breathwork and inspiratory muscle training** stay valuable as a regular layer, with effects building over about four weeks of consistent practice.

A note on medication classes

Because the driving source differs from person to person, the medication options differ too. Broadly, clinicians may consider volume agents (such as fludrocortisone), heart-rate agents (such as ivabradine or beta-blockers), central sympatholytics for a hyperadrenergic pattern (such as guanfacine), mast cell stabilizers and antihistamines where mast cell activation is

involved, and low-dose naltrexone for the inflammatory layer. The right choice depends entirely on the individual phenotype, which is why this is a conversation for a person and their own doctor rather than something to self-select.

ONE SAFETY PRINCIPLE

In these conditions the same substance can produce opposite reactions in different people. It is wise never to start at a full dose, and never to add more than one new variable at a time. What is one person's miracle can be another person's setback.

Closing

For anyone carrying several of these sources at once, and many people do, a 10-minute breathing session was always going to be a necessary piece that could not do the whole job on its own. That is information about physiology, not a personal failing. The most useful approach is to keep watching one's own patterns over time, bring them to a clinician who works with dysautonomia, and treat the data as an investment in understanding one's own body better. Seeing the system clearly is worth a great deal.

— Anna Elitzur, Medical Advisor