**VASO-VAGAL Reaction**  
*What you need to know to operate the SCIO*

by *Desire’ Dubounet*

Conventional biofeedback systems using skin resistance all use stimulation of electricity. The Indigo is no different.

Some patients have a vaso-vagal reaction when her system tried to shift from sympathetic nervous dominance to a more relaxing para-sympathetic dominance. If this happens too fast there can be a vaso-vagal episode. I have been lecturing about the dangers of the vaso-vagal shift for years but no one seems to be listening to the problem.

A **vasovagal episode** or **vasovagal response** or **vasovagal attack**[^1] (also called neurocardiogenic syncope) is a [**malaise**](https://www.merriam-webster.com/dictionary/malaise) mediated by the [vagus nerve](https://en.wikipedia.org/wiki/Vagus_nerve). When it leads to
"syncope" or "fainting", it is called a **vasovagal syncope**, which is the most common type of fainting.

There are a number of different **syncope** syndromes which all fall under the umbrella of vasovagal syncope. The common element among these conditions is the central mechanism leading to loss of consciousness. The differences among them are in the factors that trigger this mechanism.

Typical triggers for vasovagal episodes include:[2]

- Prolonged standing or upright sitting, particularly when standing with legs in a locked position for long periods of time—avoidance of long-term locking of one's legs in the standing position is taught in the military as well as in marching bands and drill teams.
- Standing up very quickly
- **Stress**
- Any painful or unpleasant stimuli, such as:
  - Venepuncture
  - Experiencing intense pain
  - Experiencing medical procedures with local anesthesia
  - Giving or receiving a needle immunization
  - Watching someone give blood
  - Watching someone experience pain
  - Watching or experiencing medical procedures
  - Sight of blood
  - Occasions of slight discomfort, such as dental and eye examinations
  - **Hyperthermia**, a prolonged exposure to heat
  - High temperature, either in the environment or due to exercise
  - High pressure on or around the chest area after heavy exercise
- Arousal or stimulants e.g. sex
- Sudden onset of extreme emotions
- Hunger
- Nausea or vomiting
- **Dehydration**
Urination (‘micturition syncope’) or defecation, having a bowel movement (‘defecation syncope’)
Abdominal straining or 'bearing down' trying to pass a large stool as in the Scrubs episode with JD
Swallowing (‘swallowing syncope’) or coughing (‘cough syncope’)
Random onsets due to nerve malfunctions
Switching from sympathetic dominance(Adrenergic) to para-sympathetic (Cholinergic)
Pressing upon certain places on the throat, sinuses, anus, anal perium and eyes, also known as vagal reflex stimulation when performed clinically
Water colder than 10 Celsius (50° F), or ice that comes in contact with the face, that stimulates the mammalian diving reflex and can correct the episode
High altitude
Use of certain drugs that affect blood pressure, such as amphetamine
Intense laughter

Features

In people with vasovagal episodes, the episodes are typically recurrent, usually happening when the person is exposed to a specific trigger. The initial episode often occurs when the person is a teenager, then recurs in clusters throughout his or her life. Prior to losing consciousness, the individual frequently experiences a prodrome of symptoms such as lightheadedness, nausea, sweating, ringing in the ears (tinnitus), uncomfortable feeling in the heart, weakness and visual disturbances such as lights seeming too bright, fuzzy or tunnel vision. These last for at least a few seconds before consciousness is lost (if it is lost), which typically happens when the person is sitting up or standing. When sufferers pass out,
they fall down (unless this is impeded); and when in this position, effective blood flow to the brain is immediately restored, allowing the person to wake up.

The autonomic nervous system's physiologic state (see below) leading to loss of consciousness may persist for several minutes, so:

1. If sufferers try to sit or stand when they wake up, they may pass out again;
2. The person may be nauseated, pale, and sweaty for several minutes.

Vasovagal syncope is rarely life-threatening in itself, but is mostly associated with injuries from falling while having an episode.
Treatment for vasovagal syncope focuses on avoidance of triggers, restoring blood flow to the brain during an impending episode, and measures that interrupt or prevent the pathophysiologic mechanism described above.

- The cornerstone of treatment is avoidance of triggers known to cause syncope in that person. However, new development in psychological research has shown that patients show great reductions in vasovagal syncope through exposure-based exercises with therapists. [4]
• Because vasovagal syncope causes a decrease in blood pressure, relaxing the entire body as a mode of avoidance isn't favorable.[4] A patient can cross his/her legs and tighten leg muscles to keep blood pressure from dropping so drastically before an injection.[5]

• Before known triggering events, the patient may increase consumption of salt and fluids to increase blood volume. Sports and energy drinks may be particularly helpful.

• Discontinuation of medications known to lower blood pressure may be helpful, but stopping antihypertensive drugs can also be dangerous. This process should be managed by an expert.

• Patients should be educated on how to respond to further episodes of syncope, especially if they experience prodromal warning signs: they should lie down and raise their legs; or at least lower their head to increase blood flow to the brain. If the individual has lost consciousness, he or she should be laid down with his or her head turned to the side. Tight clothing should be loosened. If the inciting factor is known, it should be removed if possible (for instance, the cause of pain).

• Wearing graded compression stockings may be helpful.

• There are certain orthostatic training exercises which have been proven to improve symptoms in people with recurrent vaso-vagal syncope.

For our devices therapist must be warned to not use maximum settings for too long and stay at the safe calibrated levels. This will prohibit a vaso-vagal episode. Always ask if there is a history of vaso-vagal episodes most often fainting or cold sweats with heart palpitations. If so always use low calibrated settings. Do not try to push therapy after therapy on patients with such history. Please always watch a patient who is getting a therapy on electrical stimulation. Be prepared to respond. Respond by cold water on the face gently. Gentle pressure over the eyes with a cold rag. Push on the acupuncture emergency spot above their upper lip in the cleft under their nose. Lie the patient down but watch out if they stand up to fast. Reduce stress and wait for ten minutes it will most likely pass. Do not let them leave till they are better. Call 911 if they pass out for more than a min. have pain or vomiting. Report fainting spell. I was working in a doctor’s office where a woman passed out in the waiting room. She did not respond to the emergency acupuncture spot. The head doctor said for her to sleep it off. I went thru her hand bag and found sleeping pills. She was trying to commit suicide, but she did it in an office where they might save her. Most suicides do it where someone could save them. Luckily I was there to save her and called 911. She lived to tell the story.
Acupuncture emergency spot, press for 10 - 15 seconds in case of fainting.
Prevention of vasovagal syncope

A bottle of water can be as effective as a DDD pacemaker!
Water Ingestion as Prophylaxis Against Syncope

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VASOVAGAL RESPONSE

1. Blood vessel enlarges with increased volume
2. Brain senses increased blood pressure
3. Vagus nerve slows heart rate

Nucleus solitarius (tract)
Vagus nerve (Parasympathetic)
Cardiac nerves (Sympathetic)
Parasympathetic stimulation slows heart rate
Sympathetic stimulation speeds heart rate
Sinoatrial node sets heart rate
Syncope Classifications

→ Neurally-Mediated
  - VVS
  - CSS
  - Situational

→ Orthostatic
  - Autonomic Failure
  - Drug Effects
  - Volume Depletion

→ Cardiac Arrhythmias

→ Structural Cardiopulmonary Disease

→ Cerebrovascular
  - Vascular Steal

Source: Pacing Clin Electrophysiol © 2006 Blackwell Publishing
The Vagus Nerves & Gut Function

- Sensations of hunger, fullness and satisfaction
- Emptying of the stomach contents into the small intestine
- Relaxation of the stomach to prepare for entry of food
- Pancreatic secretion of digestive enzymes to enable calorie absorption
- Stomach contractions to reduce particle size
WHAT IS THE VAGUS NERVE?

The 10th of the cranial nerves, it is often called the “Nerve of compassion” because when it’s active, it helps create the “warm-fuzzies” that we feel in our chest when we get a hug or are moved by something…

The vagus nerve is a bundle of nerves that originates in the top of the spinal cord. It activates different organs throughout the body (such as the heart, lungs, liver and digestive organs). When active, it is likely to produce that feeling of warm expansion in the chest—for example, when we are moved by someone’s goodness or when we appreciate a beautiful piece of music.

Neuroscientist Stephen W. Porges of the University of Illinois at Chicago long ago argued that the vagus nerve is [the nerve of compassion] (of course, it serves many other functions as well). Several reasons justify this claim. The vagus nerve is thought to stimulate certain muscles in the vocal chamber, enabling communication. It reduces heart rate. Very new science suggests that it may be closely connected to receptor networks for oxytocin, a neurotransmitter involved in trust and maternal bonding.

Our research and that of other scientists suggest that activation of the vagus nerve is associated with feelings of caretaking and the ethical intuition that humans from different social groups (even adversarial ones) share a common humanity. People who have high vagus nerve activation in a resting state, we have found, are prone to feeling emotions that promote altruism—compassion, gratitude, love and happiness.
Arizona State University psychologist Nancy Eisenberg has found that children with high-baseline vagus nerve activity are more cooperative and likely to give. This area of study is the beginning of a fascinating new argument about altruism: that a branch of our nervous system evolved to support such behavior.

Your body’s levels of stress hormones are regulated by the autonomic nervous system (ANS) [3]. The ANS has two components that balance each other, the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS).

The SNS turns up your nervous system. It helps us handle what we perceive to be emergencies and is in charge of the flight-or-fight response.

The PNS turns down the nervous system and helps us to be calm. It promotes relaxation, rest, sleep, and drowsiness by slowing our heart rate, slowing our breathing, constricts the pupils of our eyes, increases the production of saliva in our mouth, and so forth.

The vagus nerve is the nerve that comes from the brain and controls the parasympathetic nervous system, which controls your relaxation response. And this nervous system uses the neurotransmitter,
acetylcholine. If your brain cannot communicate with your diaphragm via the release of acetylcholine from the vagus nerve (for example, impaired by botulinum toxin), then you will stop breathing and die[6].

Acetylcholine is responsible for learning and memory. It is also calming and relaxing, which is used by vagus nerve to send messages of peace and relaxation throughout your body. New research has found that acetylcholine is a major brake on inflammation in the body [4]. In other words, stimulating your vagus nerve sends acetylcholine throughout your body, not only relaxing you but also turning down the fires of inflammation which is related to the negative effects from stress[1].

Exciting new research has also linked the vagus nerve to improved neurogenesis, increased BDNF output (brain-derived neurotrophic factor is like super fertilizer for your brain cells) and repair of brain tissue, and to actual regeneration throughout the body.

HEALTH, LONGEVITY & AGING

As you get older, your immune system produces more inflammatory molecules, and your nervous system turns on the stress response, promoting system breakdown and aging.

That’s not just talk. It’s backed by scientific studies.

For example, Kevin Tracey, the director of the Feinstein Institute for Medical Research, discovered how the brain controls the immune system through a direct nerve-based connection.

He describes this as the inflammatory reflex (i). Simply put, it is the way the immune system responds to the mind.

Let me explain.

You immune system is controlled by a nerve call the vagus nerve.

But this isn’t just any nerve.

It is the most important nerve coming from the brain and travels to all the major organs.

And you can activate this nerve — through relaxation, meditation, and other ancient practices, such as the Mayan system of Light Language, combined with Vagus Nerve Activation Techniques given recently by the Group & Steve Rother, the Vagus Nerve can be activated and worked with energetically through geometry, frequency, color, and light.

What’s the benefit of that?

Well, by activating the vagus nerve, you can control your immune cells, reduce inflammation, and even prevent disease and aging!
It’s true. By creating positive brain states — as meditation masters have done for centuries — you can switch on the vagus nerve and control inflammation.

You can actually control your gene function by this method. Activate the vagus nerve, and you can switch on the genes that help control inflammation. Inflammation is one of the central factors of disease and aging.

**CELLULAR REGENERATION**

Even more fascinating was the discovery that our bodies can regenerate at any age.

Diane Krause, MD, PhD, from Yale University discovered that our own innate adult stem cells (cells that can turn into any cell in the body from our bone marrow) could be transformed into liver, bowel, lung, and skin cells. (ii)

This is a phenomenal breakthrough.

Here’s why.

It means that we have the power to create new cells and renew our own organs and tissues at any age.

And how are these stem cells controlled?

You guessed it: the vagus nerve.

For example, Theise et al. [5] have found that stems cells are directly connected to the vagus nerve. Activating the vagus nerve can stimulate stem cells to produce new cells and repair and rebuild your own organs.

So relaxation — a state of calm, peace, and stillness – can activate the vagus nerve.

And the vagus nerve, in turn, activates your stem cells to regenerate and renew your tissues and organs.
RELAXATION & MEDITATION

Scientists have even shown how meditation makes the brain bigger and better.

They’ve mapped out the brain function of “professional meditators” by bringing Tibetan lamas trained in concentration and mental control into the laboratory.

The result? They found higher levels of gamma brain waves and thicker brain cortexes (the areas associated with higher brain function) in meditators. (iii)

Relaxation can have other powerful effects on our biology.

In biology, being a complex system that can adapt to its environment and that is resilient and flexible is critical to health.

The same is true for us.

The more complex and resilient we are, the healthier we are.

Take, for example, our heartbeat.

Its complexity is called heart rate variability (HRV) or beat-to-beat variability. The more complex your HRV, the healthier you are. The least complex heart rate is the worst — a flat line.

So what does this have to do with relaxation?

The HRV is also controlled by the vagus nerve.

As you can see, turning on the relaxation response and activating that vagus nerve is critical to health.

Activating the Vagus Nerve Will:

* Reduce inflammation

* Help regenerate your organs and cells by activating stem cells

* Increase your heart rate variability

* Thicken your brain (which normally shrinks with aging).

* Boost immune function

* Modulate your nervous system

* Reduce depression and stress
* Enhance performance

* Improve your quality of life

Not bad for just learning to chill out!

**COMPASSION & DNA**

Elizabeth Blackburn, PhD, who discovered telomeres, explained that, ultimately, they become so short that the end of our DNA unravels and we can no longer replicate our cells, so they die.

Remarkably, mental stress produces a more rapid shortening of the telomeres — and leads to faster aging.

What’s even more remarkable?

In a study of caregivers of sick patients, the health of the caregivers' telomeres was determined by their attitude!

It sounds impossible, but it's true.

The caregivers who felt the care to be a burden had shorter telomeres, while those who saw their work as an opportunity to be compassionate had no shortening. (iv)

The Dalai Lama said that the seat of compassion is actually biological and — necessary for survival.

Perhaps the development of compassion and wisdom in coping with unfavorable life conditions is the true key to longevity.

It just may be that working to understand our true nature through the cultivation of our minds and hearts with positive practices like meditation or similar techniques is critical to health and longevity.

The ways we can change our bodies through changing our minds is not longer a theory.

There is a new scientific language to understand how the qualities of the mind control the body through effects on the vagus nerve, immune cells, stem cells, telomeres, DNA, and more.

Remember, your body has all the resources and infinitely adaptable systems to self-regulate, repair, regenerate, and thrive.

You simply have to learn how to work with your body, rather than against it. Then you can have a healthy, thriving life — and live out your full lifespan, which can be as high as 120+ years!
But here’s something even cooler – the research that Dacher Keltner, director of the Social Interaction Laboratory at the University of California, Berkeley is doing shows that stimulating that vagus nerve is not only good for you – it’s good for the planet!

“Our research and that of other scientists suggest that activation of the vagus nerve is associated with feelings of caretaking and the ethical intuition that humans from different social groups (even adversarial ones) share a common humanity. People who have high vagus nerve activation in a resting state, we have found, are prone to feeling emotions that promote altruism – compassion, gratitude, love and happiness.”

There you go. Do it for love.

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Vasovagal response
Vasovagal episode

Classification and external resources

Vagus nerve

ICD-10  R55

ICD-9  780.2

DiseasesDB  13777
A vasovagal episode or vasovagal response or vasovagal attack (also called neurocardiogenic syncope) is a malaise mediated by the vagus nerve. When it leads to syncope or “fainting”, it is called a vasovagal syncope (vay-zo-VAY-gul SING-cuh-pee), which is the most common type of fainting.

There are different syncope syndromes which all fall under the umbrella of vasovagal syncope. The common element among these conditions is the central mechanism leading to loss of consciousness. The differences among them are in the factors that trigger this mechanism.

**Signs and symptoms**

Among people with vasovagal episodes, the episodes are typically recurrent, usually happening when the person is exposed to a specific trigger. Prior to losing consciousness, the individual frequently experiences a prodrome of symptoms such as lightheadedness, nausea, the feeling of being extremely hot (accompanied by sweating), ringing in the ears (tinnitus), uncomfortable feeling in the heart, fuzzy thoughts, a slight inability to speak/form words (sometimes combined with mild stuttering), weakness and visual disturbances such as lights seeming too bright, fuzzy or tunnel vision, and sometimes a feeling of nervousness can occur as well. These last for at least a few seconds before consciousness is lost (if it is lost), which typically happens when the person is sitting up or standing. When sufferers pass out, they fall down (unless this is impeded); and when in this position, effective blood flow to the brain is immediately restored, allowing the person to regain consciousness. Short of fainting a person may experience an almost indescribable weak and tired feeling resulting from a lack of oxygen to the brain due to a sudden drop in blood pressure. Taber's Cyclopedic Medical Dictionary describes this as the “feeling of impending death” caused by expansion of the aorta, drawing blood from the head and upper body.

The autonomic nervous system’s physiologic state (see below) leading to loss of consciousness may persist for several minutes, so:

- If sufferers try to sit or stand when they wake up, they may pass out again
- The person may be nauseated, pale, and sweaty for several minutes or hours

**Cause**

Vasovagal syncope occurs in response to a trigger, with a corresponding malfunction in the parts of the nervous system that regulate heart rate and blood pressure. When heart rate slows, blood pressure drops, and the resulting lack of blood to the brain causes fainting.

Typical triggers for vasovagal episodes include:
- Prolonged standing or upright sitting
- Standing up very quickly (Orthostatic hypotension)
- Stress directly related to trauma[^6]

**Stress**

- P.O.T.S. (Postural Orthostatic Tachycardia Syndrome) Multiple chronic episodes are experienced daily by many patients diagnosed with this syndrome. Episodes are most commonly manifested upon standing up.
- Any painful or unpleasant stimuli, such as:
  - Trauma (such as hitting one's funny bone)
  - Watching or experiencing medical procedures (such as Venipuncture)
  - High pressure on or around the chest area after heavy exercise
- Myocardial infarction, severe valvular (sic) disease, and disturbances in rhythm which reduce the output of blood may be responsible[^7]
- Severe menstrual cramps
- Arousal or stimulants, e.g. sex, tickling or adrenaline
- Sudden onset of extreme emotions
- Lack of Sleep

**Dehydration**

- Hunger
- Being exposed to high temperatures
- In health care, such as nursing care, digital rectal procedures (e.g., digital disimpaction)
- Random onsets due to nerve malfunctions
- Pressing upon certain places on the throat, sinuses, and eyes (also known as vagal reflex stimulation when performed clinically)
- Use of certain drugs that affect blood pressure, such as cocaine, alcohol, marijuana, inhalants and opiates[^8]
- The sight of blood[^9] or blood drawing[^10]
- Violent coughing
- Serotonin level / SSRI[^11]
- Swallowing[^12][^13]
- (Less commonly) Low Blood Sugar[^7]

**Pathophysiology**

Regardless of the trigger, the mechanism of syncope is similar in the various vasovagal syncope syndromes. In it, the nucleus tractus solitarius of the brainstem is activated directly or indirectly by the triggering stimulus,
resulting in simultaneous enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone.

This results in a spectrum of hemodynamic responses:

1. On one end of the spectrum is the cardioinhibitory response, characterized by a drop in heart rate (negative chronotropic effect) and in contractility (negative inotropic effect) leading to a decrease in cardiac output that is significant enough to result in a loss of consciousness. It is thought that this response results primarily from enhancement in parasympathetic tone.

2. On the other end of the spectrum is the vasodepressor response, caused by a drop in blood pressure (to as low as 80/20) without much change in heart rate. This phenomenon occurs due to vasodilation, probably as a result of withdrawal of sympathetic nervous system tone.

3. The majority of people with vasovagal syncope have a mixed response somewhere between these two ends of the spectrum.

One account for these physiological responses is the Bezold-Jarisch reflex.

Diagnosis

In addition to the mechanism described above, a number of other medical conditions may cause syncope. Making the correct diagnosis for loss of consciousness is one of the most difficult challenges that a physician can face. The core of the diagnosis of vasovagal syncope rests upon a clear description by the patient of a typical pattern of triggers, symptoms, and time course. It is also pertinent to differentiate lightheadedness, seizures, vertigo, and hypoglycemia as other causes.

In patients with recurrent vasovagal syncope, diagnostic accuracy can often be improved with one of the following diagnostic tests:

1. A tilt table test (results should be interpreted in the context of patients' clinical presentations and with an understanding of the sensitivity and specificity of the test)
2. Implantation of an insertable loop recorder
3. A Holter monitor or event monitor
4. An echocardiogram
5. An electrophysiology study

Treatment

Treatment for vasovagal syncope focuses on avoidance of triggers, restoring blood flow to the brain during an impending episode, and measures that interrupt or prevent the pathophysiologic mechanism described above.
The cornerstone of treatment is avoidance of triggers known to cause syncope in that person. However, a new development in psychological research has shown that patients show great reductions in vasovagal syncope through exposure-based exercises with therapists if the trigger is mental or emotional, e.g. sight of blood.\(^{[19]}\) However, if the trigger is a specific drug, then avoidance is the only treatment.

Because vasovagal syncope causes a decrease in blood pressure, relaxing the entire body as a mode of avoidance isn't favorable.\(^{[19]}\) A patient can move or cross his/her legs and tighten leg muscles to keep blood pressure from dropping so drastically before an injection.\(^{[19]}\)

Before known triggering events, the patient may increase consumption of salt and fluids to increase blood volume. Sports and energy drinks may be particularly helpful.

Discontinuation of medications known to lower blood pressure may be helpful, but stopping antihypertensive drugs can also be dangerous in some people. Taking antihypertensive drugs may worsen the syncope, as the hypertension may have been the body's way to compensate for the low blood pressure.

Patients should be educated on how to respond to further episodes of syncope, especially if they experience prodromal warning signs: they should lie down and raise their legs, or at least lower their head to increase blood flow to the brain. If the individual has lost consciousness, he or she should be laid down with his or her head turned to the side. Tight clothing should be loosened. If the inciting factor is known, it should be removed if possible (for instance, the cause of pain).

Wearing graded compression stockings may be helpful.

There are certain orthostatic training exercises which have been proven to improve symptoms in people with recurrent vasovagal syncope. A technique called "Applied Tension" which involves learning to tense the muscles in the torso, arms, and legs is effective for vasovagal syncope.

Certain medications may also be helpful:
- Beta blockers (\(\beta\)-adrenergic antagonists) were once the most common medication given; however, they have been shown to be ineffective in a variety of studies and are thus no longer prescribed. In addition, they may cause the syncope by lowering the blood pressure and heart rate.\(^{[17][19]}\)
- Other medications which may be effective include: CNS stimulants\(^{[19]}\) fludrocortisone, midodrine, SSRIs such as paroxetine or sertraline, disopyramide, and, in health-care settings where a syncope is anticipated, atropine epinephrine (adrenaline).\(^{[20]}\)
- For people with the cardioinhibitory form of vasovagal syncope, implantation of a permanent pacemaker may be beneficial or even curative.\(^{[21]}\)

Types of Long-Term Therapy for Vasovagal Syncope include:\(^{[14]}\)
- Preload agents
- Vasoconstrictors
- Anticholinergic agents
Negative cardiac inotropes
Central agents
Mechanical device

Prognosis

Brief periods of unconsciousness do no harm and are seldom symptoms of disease. The main danger of vasovagal syncope (or dizzy spells from vertigo) is the risk of injury by falling while unconscious. Medication therapy could possibly prevent future vasovagal responses; however, for some individuals medication is ineffective and they will continue to have fainting episodes.

See also

- Vagus reflex
- Vagovagal reflex
- Roemheld Syndrome

References

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Clinical Investigation and Reports

Water Ingestion as Prophylaxis Against Syncope

1. Chih-Cherng Lu, MD, MS;
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Abstract

Background—Water ingestion raises blood pressure substantially in patients with perturbed autonomic control and more modestly in older subjects. It is unclear whether prophylactic water drinking improves orthostatic tolerance in normal healthy adults.

Methods and Results—Twenty-two healthy subjects, 18 to 42 years of age, with no history of syncope underwent head-up tilt-table testing at 60° for 45 minutes or until presyncope or syncope occurred. In their initial test, participants were randomized to either 16 oz (473 mL) of water drinking 5 minutes before tilt-table testing or tilt-table testing alone, with the alternative in a second test on a different day. During the first 30 minutes of tilt, 8 of 22 subjects without water experienced presyncope but only 1 of 22 who had ingested water (P=0.016). Water drinking attenuated the heart rate increase associated with tilt (P<0.001) while accentuating the increase in total peripheral resistance (P=0.012). The average time study participants tolerated head-up tilt was 26% longer after water (41.1±8.1 versus 32.6±14.3 minutes, mean±SD), with a pairwise mean difference of 8.5±14.0 minutes (95% CI, 2.3 to 14.7 minutes; P=0.011).

Conclusions—Water enhances tolerance of upright posture. The effect of water is mediated by increased peripheral vascular resistance. Water ingestion may constitute a simple and effective prophylaxis against vasovagal reactions in healthy subjects, such as those associated with blood donation.

Key Words:
• syncope
Syncope, the sudden brief loss of consciousness caused by diminished cerebral blood flow, occurs at least once in almost 22% of the population, and 9% have recurrent syncope. It occurs in both children and adults and is responsible for ≈6% of emergency room visits and 3% of hospitalizations. Most syncopal events are triggered by standing or emotion and are often referred to as vasovagal reactions. Under certain circumstances, such as blood donation, syncope has important medical and societal significance. More than 150 000 people experience syncope or near-syncope each year at the time of blood donation to the American Red Cross. Reducing such syncopal reactions could have a beneficial impact on donor convenience, safety, and desire to donate again. Currently, in blood donation facilities, the major preventive strategies against syncope focus on postdonation food and beverage, with little emphasis on predonation factors, such as water drinking. When vasovagal syncope occurs frequently, pharmacological agents and pacemakers are used, but therapy is expensive, efficacy is questionable, and adverse effects are common. We have shown previously that water ingestion raises blood pressure ≈30 mm Hg in patients with abnormal autonomic control and more modestly in older normal subjects. Tilt-table testing provides a means of calibrating orthostatic tolerance and assessing factors that influence it. We tested the hypothesis that water ingestion would enhance orthostatic tolerance. We also aimed to address the underlying physiology of this effect.

Methods

Subjects

The study was approved by the Institutional Review Board of Vanderbilt University and conducted in the General Clinical Research Center. We studied 22 healthy, normal adults (11 male and 11 female) with no history of syncope and not currently using any prescription or over-the-counter medication except for oral contraceptives.

Study Protocol

We used a randomized, crossover study design. Each subject underwent the study protocol twice on separate days. Subjects received either tilt test with water or tilt test without water in their initial study, with the alternative in their second test. Room-temperature tap water (16 oz [473 mL]) was ingested 5 minutes before head-up tilt. Analysis of the water demonstrated 5.1 mg/L sodium, 7.6 mg/L chloride, and 76 mg/L calcium, with a pH of 7.2. Caffeine-containing beverages, nicotine, and alcohol were prohibited for 1 week before the study. Subjects were placed on a calculated diet containing 150 mmol sodium and 70 mmol potassium for ≥3 days before testing. The volunteers took no food or beverage from midnight until the testing session on the subsequent morning. Study sessions took place in a quiet, dimly lit room at a comfortable ambient temperature (70°F to 75°F; 21°C to 24°C).

Instrumentation

An antecubital venous catheter for blood sampling was inserted ≥15 minutes before the beginning of the test, with the patient in the supine position. Heart rate and blood pressure were monitored by finger volume-clamp method (Finapres, 2300, Ohmeda), which provided continuous, noninvasive heart rate and pressure measurements. Baseline Finapres blood pressures were calibrated against cuff pressure from a Dinamap vital signs monitor (Critikon Company LLC) before data collection. Thoracic bioimpedance was monitored continuously for cardiac output, respiration, and peripheral vascular resistance (Thrim model 2994D, UFI).

Head-Up Tilt-Table Test

All studies began at 8:30 AM and finished by 10:30 AM. Data acquisition began after a 30-minute supine adaptation. After a further resting period of 10 minutes in the supine position, subjects were tilted at angles chosen to graduate
orthostatic stress. Head-up tilt was stepwise (0°, 15°, 30°, 45°, 60°) at 3-minute intervals (Tilt Table, βETA plus, Berne Manufacturing Co). Subjects then remained tilted for 45 minutes or until presyncope symptoms were observed. Syncope was defined as a systolic pressure <70 mm Hg and heart rate <50 bpm. Presyncope was defined as a fall in blood pressure of ≥30 mm Hg with a concomitant fall in heart rate of ≥10 bpm, or a fall in heart rate of ≥30 bpm with a concomitant fall in blood pressure of ≥10 mm Hg. These hemodynamic end points were assessed from tracings by 2 independent evaluators not involved in the protocol itself and blinded to the intervention group.

**Analytical Methods**

Blood samples for catecholamines were obtained and assayed as previously described. Samples were collected at −5, 0, 10, 15, 30, and 45 minutes of the study. In addition, blood was taken for assay 1 minute after the onset of presyncope or syncope for estimation of plasma volume change. Signals for blood pressure, the ECG, and bioimpedance were sampled at 500 Hz using Dataq model DI-220 and visualized using Windaq Pro+ software (Dataq Instruments Inc). Complete recordings of R-R interval, finger blood pressure values, and respiration were analyzed offline with a program based on PV-wave software (Visual Numerics Inc). Total peripheral resistance was calculated from the mean brachial blood pressure and cardiac output.

**Statistics**

Our primary end point was time until presyncope. The null hypothesis was that this time would not be statistically different between the tilt-table study after water ingestion and the study without water. A sample size of 22 was estimated to have 80% power to detect an effect size (difference between the means divided by the SD of the difference) of 0.6 with a paired t test with a 2-sided significance level of 0.05. A Pearson χ² test or Fisher’s exact test was used to assess categorical baseline comparisons. McNemar’s test was used to compare the pairwise presyncope concordance during the water and no-water phases. Differences between group means for continuous measurements were tested by Student’s t test or the Mann-Whitney U test. Before-and-after comparisons were analyzed with the paired t test or the Wilcoxon signed-rank test. A general linear model repeated-measures ANOVA was used to assess changes from baseline between the 2 phases of the study while adjusting for and assessing covariates such as the day order of the study. Cox proportional-hazards analysis was used to determine the effect of water on time to presyncope. Assumptions of proportional hazards were assessed by use of Schoenfeld’s residuals. The log-rank test was used to compare survival curves. Values are reported as means and SDs unless otherwise noted. Probability values of P=0.05 were considered statistically significant, and all tests were 2-tailed. Statistical analyses were performed with SPSS (version 11.0, SPSS).

**Results**

**Water Ingestion and Orthostatic Tolerance**

Resting baseline control data for study subjects are shown in Table 1. There were no significant baseline pairwise differences. Subjects receiving water tolerated head-up tilt-table testing 26% longer (41±8 versus 33±14 minutes, P=0.011). Water increased the time by an average of 8.5±14.0 (SD) minutes (95% CI, 2.3 to 14.7 minutes). Twelve participants tolerated tilt longer during the water phase, whereas only 3 had a longer tilt duration without water. Seven completed 45 minutes of tilt during both phases. During the first 30 minutes of tilt, 64% (14 of 22) of those without water tolerated the study. This increased to 96% (21 of 22) tolerating the tilting after water ingestion (Figure 1). Water ingestion increased the cumulative proportion tolerating the tilt test significantly, from 45% to 68% (P=0.036; Figure 2). Three subjects experienced presyncope both with and without water, but in all 3, water ingestion increased the duration of head-up tilt (11 versus 7, 35 versus 25, and 34 versus 12 minutes; Table 2).

**TABLE 1. Characteristics of the 22 Subjects Studied at Baseline (Time 0)**

<table>
<thead>
<tr>
<th>No Water</th>
<th>Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration (min)</td>
<td>0</td>
</tr>
</tbody>
</table>

**Figure 1.** Duration of head-up tilt after drinking 16 oz (473 mL) of water vs no water. Mean improvement of duration of head-up tilt was 8.5±14.0 minutes. P=0.011 by Wilcoxon signed-rank test.
Figure 2. Kaplan-Meier curves of cumulative proportion with orthostatic tolerance (remaining free of presyncopal episodes) with and without water. At 30 minutes, 95% of those with water compared with 63% of those without water were able to tolerate tilt. At completion of study (45 minutes), 69% with water compared with 45% without water were able to tolerate tilt. \( P=0.036 \) by log-rank test.

TABLE 2. Individual Hemodynamic Responses to Head-Up Tilt

We also examined the order effect of the 2 studies on duration of head-up tilt. Duration of tilt on the first day was 29.9±14.7 minutes without water and 38.2±10.4 minutes with water. On day 2, the durations were 35.3±14.2 minutes without water and 44.0±3.3 minutes with water. The effect of water ingestion was significant (\( P=0.006 \)), the effect order had on water (the order–water interaction) was borderline (\( P=0.059 \)), and the order itself was nonsignificant (\( P=0.964 \)).

**Effects of Water Ingestion on Hemodynamic Variables**

Systolic blood pressure, heart rate, cardiac output, and peripheral vascular resistance for head-up tilt with and without water were all altered significantly by tilt. Water-by-time interactions were significant for attenuation of heart rate increase associated with tilt (\( P<0.001 \)). Heart rate rose from 65.0±10.0 bpm at baseline to 87.2±11.2 bpm in subjects not receiving water and from 65.7±10.9 bpm to 80.1±9.8 bpm in the same subjects 20 minutes after ingestion of water (Figure 3). Peripheral vascular resistance rose sharply with tilt. It then gradually declined in subjects who ingested no water but remained elevated in those who ingested water (\( P<0.001 \), Figure 4).

Figure 3. Heart rate (HR) during head-up tilt. Water ingestion blunts increasing heart rate response during upright tilt. \( P<0.05 \).
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**Figure 4.** Total peripheral resistance (TPR) during head-up tilt. Water ingestion accentuated increasing TPR during tilt-table testing. \( P<0.001 \).

With upright posture or head-up tilt, hemoconcentration occurs as plasma volume enters the extravascular space in a gravity-dependent manner. Thus, under many circumstances, hematocrit may reflect this acute change in plasma volume. In this study, hematocrit increased significantly with head-up tilt. After 45 minutes of head-up tilt, there was a 16.7±4.8% increase in hematocrit without water and 13.8±4.2% increase in hematocrit with water (\( P=0.065 \)).

**Plasma Catecholamines**

In the supine position, concentrations of plasma norepinephrine and epinephrine were within normal range. With gradual tilting, both increased and were significantly raised between 15 and 45 minutes (\( P<0.001 \)). No significant difference in norepinephrine concentration with water versus without water was noted 15 minutes after tilt (305±108 versus 275±130 pg/mL; \( P=0.211 \)). Plasma epinephrine rose significantly in response to head-up tilt (\( P<0.01 \)), with increases in epinephrine of 43±4.5 pg/mL without water and 41.3±4.1 pg/mL with water at the 30-minute time point. Plasma dihydroxyphenylglycol (DHPG) rose significantly with upright posture. In the supine position, the average plasma dopa levels were similar in subjects receiving water or tilt alone. With water ingestion and tilt, however, there was a smaller decrease in dopa at 10 and 15 minutes after head-up tilt (\( P=0.041 \), **Figure 5**).

**Discussion**

The most important new finding in this study is that water ingestion significantly improves orthostatic tolerance during head-up tilt in normal healthy adults. Whereas 8 of 22 subjects experienced hypotension/bradycardia in the first 30 minutes with tilt alone, only 1 of 22 subjects experienced these symptoms in the first 30 minutes with water ingestion and tilt. The protection afforded by water was strongly correlated with an increase in peripheral vascular resistance. Water increased tolerance time to head-up tilt by 26%. Among subjects who experienced hypotension/bradycardia, both with water and without water, all had better orthostatic tolerance with water than without.

Water ingestion itself has a large pressor effect in autonomic failure. This effect is also present in older normal subjects, is antagonized by ganglionic blockade, and is associated with increases in plasma norepinephrine and in muscle sympathetic nerve activity. Water ingestion also benefits orthostatic intolerance. We hypothesized that this effect of water ingestion might also provide a margin of protection against syncope during orthostatic stress in normal subjects.

Water ingestion interacted with the cardiovascular response to orthostatic stress by attenuating the heart rate increase induced by head-up tilt testing in our study. Although the mechanism of the lesser heart rate increase after water ingestion is unclear, a trend toward reduced heart rate after water ingestion has been observed in our previous studies.
studies of water ingestion in other conditions and indeed has been applied to reduce the orthostatic tachycardia in patients with orthostatic intolerance.\textsuperscript{24} Our understanding of this heart rate reduction with water is limited. Vasovagal syncope is associated with a vasodilatation and a reduction in muscle sympathetic nerve activity together with an increase in plasma epinephrine in patients who faint\textsuperscript{25,26} and in patients with recurrent vasovagal syncope.\textsuperscript{27} The potentiation of the total peripheral resistance increase in our study subjects in response to water might suggest that water somehow abrogates these vasodilator responses. The effect of water is especially remarkable in the subgroup that did not have presyncope during either trial, in that the resistance-enhancing effect of water was greater in them.

An important factor contributing to interindividual variation in response to upright posture is orthostatic loss of plasma volume. In this study, hematocrit changes during tilt were substantial. The magnitude of this response may affect autonomic and cardiovascular mechanisms involved in the maintenance of homeostasis during upright posture. There was a trend toward less tilt-induced plasma loss among the 7 subjects who did not experience presyncope during the study. This may contribute to the stability of hemodynamics in subjects given water before the orthostatic challenge. Hematocrit slopes with tilt were steeper early in the study and less steep 30 minutes after head-up tilt. Similar observations of blood density and hematocrit measurement after water were noted by Endo et al.\textsuperscript{28} They found a biphasic change in plasma volume. Initially, there was early hemococoncentration, which they ascribed to sympathetic activation. This was followed by hemodilution, presumably because of a postabsorptive effect of the water. Together with these observations, our study results suggest that volume effects of water might become important, especially late in our head-up tilt protocol.

Previous studies have shown evidence for increased plasma norepinephrine\textsuperscript{29} or increased muscle sympathetic activity\textsuperscript{20} in response to water ingestion. We assessed plasma catecholamines and their metabolites in an effort to address the concomitant effects of water and upright posture intertwined. Plasma norepinephrine and epinephrine both rose with tilt, as did plasma DHPG. DHPG is produced predominantly intraneuronally by the action of monoamine oxidase on cytoplasmic norepinephrine. Because the DHPG is then available for release into plasma, plasma levels of this metabolite often reflect sympathetic activation and norepinephrine transporter function. The half-life of catecholamines in plasma is very short, \( \approx 60 \) to 120 seconds. Although plasma norepinephrine rose significantly with tilt, the presence or absence of water in the protocol did not seem to alter the plasma levels significantly. Similarly, plasma epinephrine levels, which rose even more dramatically, especially with presyncope, did not show significant differences with water. Dopa levels were significantly greater in patients who had received water than in those who had not. Plasma dopa levels often indicate level of activation of the enzyme tyrosine hydroxylase, which converts tyrosine to dopa in neurons. In addition, the gastrointestinal circulation is a major source of dopa production.\textsuperscript{21} The difference in dopa in this study may reflect an enhanced sympathetic activity.

The fact that heart rate was lower after water ingestion in the setting of upright tilt raises the possibility of a readjustment of baroreflex modulation of heart rate in our subjects. Such an effect could lead to lower cardiac sympathetic drive. Such a targeted decrease in sympathetic activity to the ventricles might improve tilt tolerance, in keeping with the so-called “ventricular theory” of the pathophysiology of syncope. However, the increase in plasma norepinephrine and the increase in muscle sympathetic nerve traffic in the peroneal nerve after water ingestion would be at variance with this unless the sympathetic suppression were targeted specifically to the heart.

The thorniest problem we faced in the design, conduct, and interpretation of this study was the placebo effect. All our previous studies of therapeutic interventions in autonomic disorders over the past 20 years have included a placebo. In preparation to include a placebo in this study, we undertook ancillary studies in autonomic failure patients and were able to demonstrate that 50 mL of water did not significantly raise pressure. We considered using 50 mL of water as a sort of placebo control for the 500 mL of water. Ultimately, we rejected this because, although it would technically be a placebo, in reality American study subjects would not accept 3 tablespoons of water as a true placebo because they “know” from their health classes in school that the dose of water is an 8-oz glass. However, our colleagues in Berlin and Leeds did undertake a somewhat analogous study using 50 mL of water as placebo and obtained nearly identical results in a small number of subjects.\textsuperscript{22} This strengthens the view that our results are not a result of placebo alone. However, the fact that it is perhaps impossible to provide an adequate placebo arm in our study certainly does not imply that no placebo response could occur.

Ascertainment of hemodynamic criteria assignment was validated by 2 individuals blinded to the intervention. Thus, observer bias is unlikely to be a factor in our findings. In studies of autonomic cardiovascular regulation, meticulous control of study variables is crucial. Only healthy subjects abstaining from caffeine, alcohol, and nicotine were
included. We randomized the order of interventions to avoid the confounding effect of a training effect and to minimize other potential biases.

It seems remarkable that a measure as simple as water ingestion could have such a large effect on orthostatic tolerance. The important role of sodium in blood pressure control mechanisms and orthostatic intolerance is firmly entrenched in the physiological literature and is of unquestioned importance in the chronic control of blood pressure. An acute effect of water on blood pressure in human subjects, however, is not mentioned in modern texts of human physiology. Yet our previous studies in older normal subjects showed that systolic pressure rose as much as 11 mm Hg in response to water; such a change means that water ingestion most likely represents a major unrecognized source of blood pressure fluctuation from visit to visit in older subjects. Furthermore, the blunted increase in heart rate during tilt and the increase of total peripheral resistance after water ingestion will need to be taken into account in future clinical research whenever drugs are ingested with water because of the potential confounding effects of water on human hemodynamics.

The fact that acute ingestion of water exerts such profound effects may be exploited in situations in which prophylaxis against syncope is possible. In blood donation programs, a period of enhanced vulnerability to syncope occurs during and immediately after phlebotomy. Water prophylaxis against syncope might benefit blood donors. In our studies of the effect of water on blood pressure in autonomic failure, the large (30 mm Hg) increases in blood pressure observed after water ingestion were not replicated by the intravenous infusion of comparable volumes of dextrose solution. Thus, the caloric fluids and food usually available at blood donation centers might paradoxically be less prophylactic against syncope in such circumstances than the administration of water alone. Another situation in which acute administration of water might be helpful is in astronauts on return from the microgravity environment, because it might attenuate their orthostatic intolerance on return to earth.

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References


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