Activating or Stabilizing the Vagus Nerve

Role of the Vagus Nerve

- Vagus nerve controls:
  - Sensation of hunger
  - Expansion, fullness and emptying of stomach
  - Digestive enzyme secretion

- Severing the vagus nerve (vagotomy) causes:
  - Reduced appetite
  - Delayed stomach emptying
  - Prevention of weight gain

- The effects of vagotony are not sustainable
  - The problem — accommodation, or "work around", of permanent interruption
  - The solution — EnteroMedics’ proprietary intermittent block

20% of vagus nerve fibers send instructions from the brain to the stomach

These signals control:
- Gastric acid secretion
- Digestive enzyme secretion
- Gastric capacity
- Blood glucose

80% of vagus nerve fibers send instructions from the stomach to the brain

These signals control:
- Satiety (Hunger)
- Satiation (Fullness)
- Energy Metabolism
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Using scientific frequency stimulation
we can balance the Vagus Nerve

The Eductor has been designed with an interactive, auto-focused, energetic medicine until to balance the vega nerve.
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External Innervation

The vagus nerve (parasympathetic) decreases heart rate.

Cardioinhibitory center
Medulla oblongata

Cardio-acceleratory center

Thoracic spinal cord
Sympathetic trunk

Sympathetic cardiac nerves increase heart rate and force of contraction.

SA node
AV node

Parasympathetic fibers
Sympathetic fibers
Interneurons

http://indavideo.hu/video/Neck_and_Vagus_Nerve_massage

http://www.downloads.imune.net/medicalbooks/VASO-VAGAL%20Reaction%20what%20you%20need%20to%20know%20to%20operate%20the%20SCIO.pdf
Activating or Stabilizing the Vagus Nerve

[A novel transcortaneous vagus nerve stimulation leads to brainstem and cerebral activations measured by functional MRI].

Author information

Abstract

BACKGROUND:
Left cervical vagus nerve stimulation (VNS) using the implanted NeuroCybernetic Prosthesis (NCP) can reduce epileptic seizures and has recently been shown to give promising results for treating therapy-resistant depression. To address a disadvantage of this state-of-the-art VNS device, the use of an alternative transcortaneous electrical nerve stimulation technique, designed for muscular stimulation, was studied. Functional magnetic resonance imaging (fMRI) has been used to test non-invasively access nerve structures associated with the vagus nerve system. The results and their impact are unsatisfying due to missing brainstem activations. These activations, however, are mandatory for reasoning, higher subcortical and cortical activations of vagus nerve structures. The objective of this study was to test a new parameter setting and a novel device for performing specific (well-controlled) transcortaneous VNS (tVNS) at the inner side of the tragus. This paper shows the feasibility of these and their potential for brainstem and cerebral activations as measured by blood oxygenation level dependent functional MRI (BOLD fMRI).

MATERIALS AND METHODS:
In total, four healthy male adults were scanned inside a 1.5-Tesla MRI scanner while undergoing tVNS at the left tragus. We ensured that our newly developed tVNS stimulator was adapted to be an MR-safe stimulation device. In the experiment, cortical and brainstem representations during tVNS were compared to a baseline.

RESULTS:
A positive BOLD response was detected during stimulation in brain areas associated with higher order relay nuclei of vagal afferent pathways, respectively the left locus coeruleus, the thalamus (left >> right), the left prefrontal cortex, the right and the left postcentral gyrus, the left posterior cingulated gyrus and the left insula. Deactivations were found in the right nucleus accumbens and the right cerebellar hemisphere.

CONCLUSION:
The method and device are feasible and appropriate for accessing cerebral vagus nerve structures, respectively. As functional patterns share features with fMRI BOLD, the effects previously studied with the NCP are discussed and new possibilities of tVNS are hypothesized.
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.

STRESS & THE VAGUS NERVE

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 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!
Activating or Stabilizing the Vagus Nerve

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.
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
Activating or Stabilizing the Vagus Nerve

* 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!
LOVE

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.

references:
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http://tkcollier.wordpress.com/2006/10/05/how-the-dalai-lama-can-help-you-live-to-120/
http://www.subtleyoga.com/220/11
http://www.scientificamerican.com/article.cfm?id=forget-survival-of-the-fittest
How often do you have to deal with anxiety in your everyday life? If you find yourself worrying too much or getting caught into non-stopping irrational thoughts or even feeling nausea, chest pain and heart palpitations then this article is for you.

You are about to learn a simple yet very effective technique to deal with anxiety naturally by stimulating your vagus nerve. This powerful technique can be used to relieve stress and anxiety anywhere and anytime; at home, when commuting and of course at those horrible work meetings.

Did you know that the FDA approved a surgically implanted device that is successfully treating depression by periodically stimulating the vagus nerve?

But hopefully you won’t need surgery. You can enjoy the benefits of vagus nerve stimulation by adopting some simple breathing techniques.

So what is that vagus nerve?
Activating or Stabilizing the Vagus Nerve

The vagus nerve is the most important element of the **parasympathetic** nervous system (the one that calms you down by controlling your relaxation response).

It originates from the brainstem and it is “wandering” all the way down, into the belly, spreading fibers to the tongue, pharynx, vocal chords, lungs, heart, stomach, intestines and glands that produce anti-stress enzymes and hormones (like Acetylcholine, Prolactin, Vasopressin, Oxytocin), influencing digestion, metabolism and of course the relaxation response.

Vagus nerve acts as the **mind-body connection**, and it is the cabling behind your heart’s emotions and gut instincts. The key to manage your mind state and your **anxiety levels** lies on being able to **activate the calming nervous pathways of your parasympathetic system**.

You cannot control this part of the nervous system on demand, but you can indirectly stimulate your vagus nerve by:

- Immersing your face in cold water (diving reflex)
- Attempting to exhale against a closed airway (Valsalva maneuver).
- This can be done by keeping the mouth closed and pinching the nose while trying to breathe out. This greatly increases pressures inside the chest cavity stimulating the vagus nerve and increasing vagal tone
- Singing
- And of course, diaphragmatic breathing techniques

Strengthening this living nervous system can pay great dividends, and the best tool to achieve that is by training your breath.

**Breathe with your diaphragm**

Now it’s time to put this concept into practice. The first thing you need to do is breathe using your diaphragm (**abdominal breathing**). This is the foundation of proper breathing and anxiety relief.

The diaphragm is your primary breathing muscle. It is belled shaped and when you inhale it patterns out (or should flatten out), acting as piston and creating vacuum on you thoracic cavity, so your lungs can expand and air gets in.
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On the other side it creates pressure, pushing the viscera down and out, expanding your belly. That’s why good breathing practice is described as abdominal breathing or belly breathing.

**Breathe with the glottis partially closed**

Glottis is at the back of your tongue and it is closed when you are holding your breath. Here we want have it partially closed. It is that feeling you have in your throat while you exhale and make a “Hhhhh” sound in order to clean your glasses, but without actually making the sound.

It also resembles the way you breathe when you are in the verge of sleep and you are about to snore a little bit. By controlling the glottis you are:

- Controlling the air flow, both during inhale and during exhale
- Stimulating your vagus nerve.

**Try it right now**

Now it’s time to put all this theory into action by practicing this 7 – 11 diaphragmatic breathing technique.

- Inhale diaphragmatically through your nose, with your glottis partially closed, like almost making a “Hhhhh” sound for a count of 7
- Hold your breath for a moment
- Exhale through your nose (or you mouth), with your glottis partially closed, like almost making a “Hhhhh” sound for a count of 11

This is one breath cycle; go for 6 – 12 cycles and observe the results.

**Practice, Practice, Practice**

The more you practice the more effective this technique will be.
**Activating or Stabilizing the Vagus Nerve**

Eventually, when your newly acquired breathing skill is established and abdominal breathing becomes a habit, you’ll find your body constantly operating at a much lower stress level.

You will also notice (or sometimes you will not even notice it) how your breath responds to stressful situations; your body will be conditioned to automatically control your breath and by this, your stress and anxiety.

**Summary**

One of the keys to deal with anxiety is to learn how to stimulate your vagus nerve through proper breathing. The vagus nerve acts as the mind-body connection and controls your relaxation response. You can stimulate your vagus nerve by practicing diaphragmatic breathing with the glottis partially closed. Use your dead time to practice this technique consistently, turn it to a habit and you’ll be amazed by the results.

Bill Walker is an article writer and key founder of the AntiAnxietyWaves project. AntiAnxietyWaves offers information, guidance and techniques to deal with anxiety while involved into dead time activities (e.g. commuting time, waiting time etc). Help yourself to deal with anxiety by downloading this (free) pdf guide along with some unique anti-anxiety relaxation recordings at antianxietywaves.com/deal-with-anxiety

P.S.
Don’t procrastinate with your anxiety, please take action now; if you don’t take even a small step
Some patients have a vaso-vagal reaction when their 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 vaso-vagal episode or vasovagal response or vasovagal attack[1] (also called neurocardiogenic syncope) is a malaise mediated by the vagus nerve. When it leads to
Activating or Stabilizing the Vagus Nerve

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:[21]

- 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
Activating or Stabilizing the Vagus Nerve

- **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[^1]

---

**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

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]
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- Because vasovagal syncope causes a decrease in blood pressure, relaxing the entire body as a mode of avoidance isn't favorable. A patient can cross his/her legs and tighten leg muscles to keep blood pressure from dropping so drastically before an injection.
- 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.
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Acupuncture emergency spot, press for 10 - 15 sec in case of fainting
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Prevention of vasovagal syncope

A bottle of water can be as effective as a DDD pacemaker!

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Water Ingestion as Prophylaxis Against Syncope

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Received March 18, 2003; de novo received May 22, 2003; revision received August 4, 2003, accepted August 7, 2003.
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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

Parasympathetic stimulation slows heart rate
Sympathetic stimulation speeds heart rate
Sinoatrial node sets heart rate

Nucleus solitarius tract
Vagus nerve (Parasympathetic)
Cardiac nerves (Sympathetic)

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Exhibit # 399046-01X
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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
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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
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Polyvagal Theory

The Biology of Kundalini

The researchers at Howard Hughes medical Institute, led by Bruce Lahn have found evidence that the pressure of natural selection has lead to dramatic changes in two genes known to control brain size in humans. Brain size or intelligence is naturally selected for in evolution for obvious survival reasons, and larger brains require more oxygen. Although the brain represents only 2% of the body weight, it receives 15% of the cardiac output, 20% of total body oxygen consumption, and 25% of total body glucose utilization. The larger the brain, the greater the demand of oxygen and hence the more sophisticated the nervous system needed to provide that oxygen...the evolutionary payoff for larger brain size of course being survival. As a natural extension of mammalian evolution we can see that the human neocortex was an inevitable consequence of evolutionary pressure.

According to the Poly-Vagal Theory during evolution the mammalian nervous system developed two vagal systems. Built onto the relic of amphibians and reptiles is an evolutionary modification unique to mammals. Looking at the history of evolution Poly-Vagal Theory notes the importance of the need for oxygen in evolving the mammalian nervous system. During evolution as the mammalian nervous system got more complex than its amphibian and reptilian brothers, there was a greater demand for oxygen. Porges says that it was this need for extra oxygen that may have provided the evolutionary pressure leading to the development of the highly adaptive and sophisticated autonomic nervous system found in mammals; and that behaviors such as orienting, attention, emotion and stress are by-products of the evolutionary pressure to optimize oxygen resources. The Polyvagal Theory addresses the relative roles of the vagus nerve in energy conservation and survival.
Activating or Stabilizing the Vagus Nerve

POLYVAGAL THEORY

By Ravi Dykema

Events trigger you to react. If your first reaction doesn’t make you feel safe, you revert to the second, then the third:

<table>
<thead>
<tr>
<th>Evolved in humans</th>
<th>Feels like</th>
<th>The part of your nervous system used and what it helps you accomplish</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recently</td>
<td>Safety</td>
<td>Myelinated vagus nerve</td>
</tr>
<tr>
<td>Long Ago</td>
<td>Moderate to extreme danger</td>
<td>Sympathetic nervous system</td>
</tr>
<tr>
<td>Very Long Ago</td>
<td>Life Threatening</td>
<td>Normyelinated vagus nerve</td>
</tr>
</tbody>
</table>

Note: The polyvagal theory places great importance on social engagement as a component of staying healthy, physically and psychologically.
Activating or Stabilizing the Vagus Nerve

In Stephen Porges’s Polyvagal Theory he uses the term Polyvagal to distinguish between the two main branches of the vagus nerve:

1: The Vegetative Vagus—originates in the dorsal motor nucleus (DMNX), descends visceral efferent fibers regulating smooth and cardiac muscle and is associated with passive reflexive regulation of visceral functions: peristalsis of the GI tract, sweating, lungs, diaphragm, stomach. At the heart it is connected to stretch receptors of the aortic arch and chemoreceptors of the aortic bodies and is responsible for heart rate, dilation of blood vessels and blood pressure. The output from the dorsal motor nucleus does not convey a respiratory rhythm. The most primitive function of the vagal complex is the freeze response, which is dependent on the unmyelinated vagus which is part of the reptilian system.

2: The Smart Vagus—which originates in the medullary source of the nucleus ambiguus (NA), serving efferent fibers regulating the somatic muscles of speech and eating: the larynx, pharynx, and esophagus. The ventral vagal complex (including NA) is related to processes associated with attention, motion, emotion and communication. The functional output of the NA-vagus on the heart is part of a common neuronal network producing a cardiorespiratory rhythm. The most evolutionary recent component—the communication system functions through the new-mammalian or myelinated vagus that regulates the heart and the bronchi to promote calm and self-soothing states.

In mammals the two vagal systems are neuroanatomically distinct, have different origins, and are programmed with different response strategies and may respond in a contradictory manner. Thus Porges attributes various medical disorders to competition between DMNX and NA originating fibers. The different vagi may have oppositional outputs to the same target organ. The vagus is a complex of neural pathways originating in several areas of the brainstem. The vagus nerve consists of afferent and efferent parasympathetic (acetylcholine) fibers that run from the brainstem (medulla oblongata) down to the traverse colon and urinary organs; providing both motor and sensory parasympathetic activation for everything from the neck to the G spot. Efferent fibers originate primarily in two medullary nuclei (NA, DMNX). The vagus is not solely an efferent or motor pathway, at least 80% of the vagal fibers are afferent; that is they conduct impulses from the periphery of the body to the brainstem.

According to the Polyvagal Theory the growth of the autonomic nervous system evolves through three stages:

1. Freeze—First a primitive unmyelinated visceral vagus that fosters digestion and responds to threat by depressing metabolic activity eg: freeze response.
2. Flight/Fight—The mobilization or flight/flight is dependent on the functioning of the sympathetic nervous system; increasing metabolic output and inhibiting the visceral vagus to foster mobilization behaviors necessary for fight or flight.
3. **Communication**—The third stage, the **mammalian myelinated vagus**, can rapidly regulate cardiac output to align with the environment and is associated with cranial nerves that regulate sociability via facial expression and vocalization.

Stephen Porges points out the phylogenetic hierarchy of response to challenge: “The hierarchy emphasizes that the newer “circuits” inhibit the older ones. We use the newest circuit to promote calm states, to self-soothe and engage. When this doesn’t work, we use the sympathetic-adrenal system to mobilize for flight and flight behaviors. And when that doesn’t work, we use a very old vagal system, the freeze or shutdown system.”

Stephen Porges suggests that the true freeze response is dangerous to mammals. For example, high tone in the dorsal motor nucleus vagal system may be lethal in mammals through an overdose of the immobility response overdose. Whereas high tone from the NA-vagal system may be beneficial in adaptive significance of mammalian affective processes including courting, sexual arousal, copulation, and the establishment of enduring social bonds. In the development of enduring pair-bounds the mammalian vagus communicates safety and trust, via oxytocin and vasopressin, between the hypothalamus and the medullary source nuclei of the viscera vagus.

Porges suggests that we use our higher cognitive processes to calm the stress response and establish effective connections with others by using our facial muscles, making eye contact, modulating our voice and listening to others. In this way we increase the influence of the myelinated vagus, which calms us and turns off the stress response and makes us more metabolically efficient. He says the social neural circuit supports our health through its calming influences on the heart and lungs and its reduction of HPA axis activation.

The vagus is asymmetrical with the left and right sides performing different tasks, with the right vagus most active in the regulation of the heart. Primary emotions are related to autonomic functioning since they are often survival related, they must be integrated into the regulation of the heart and lungs. Emotions have a right limbic bias, as does the brainstem medullary structures controlling visceral function. Only when the environment is perceived as “safe” is there cortical regulation of the visceral pathways, because while under threat, cortical control of brainstem structures would compromise the individual’s ability to mobilize. Therefore when stressed or in danger, cortical control of brainstem is “inhibited” and the brainstem structures are “disinhibited” to allow the sympathetic nervous system to efficiently increase metabolic output.

Stimulation of the ascending fibers of the vagus releases **norepinephrine** into the amygdala strengthening memory storage in regions of the brain that regulate arousal, memory and feeling responses to emotionally laden stimuli. These ascending fibers is how the peripheral epinephrine from the adrenals released into the blood during the fight-flight response activates the release of norepinephrine in the limbic system sharpening memory of the events. Since the adrenal hormone epinephrine cannot cross the blood brain barrier it activates the vagus nerve, which in turn stimulates
neurons in the brainstem known as the “Nucleus of the Solitary Tract (NTS). This third medullary
nucleus, located near DMNX, is the terminus of many of the afferent pathways travelling through the
vagus from peripheral organs. Vagus afferent sensory fibers carrying information to the brain from the
head, neck, thorax, and abdomen relay information to the NTS. These NTS neurons release
norephinephrine into the memory processing areas such as the amygdala and hippocampus to activate
long term memory storage of emotionally laden events. This explains why vagus nerve stimulation was
found to improve memory consolidation of recent events. Researchers found that by microinjecting the
NTS with either GABA agonists or glutamate antagonists, they thereby increased GABA or decreased
 glutamate in the NTS and this blocked seizures.

Stephen W. Porges, Ph.D. found that he could improve autism by stimulating the newer structures and
prompting the social engagement system with the use of acoustic sessions using frequencies associated
with the human voice. Check out Stephen Porges's fabulous papers on the web:


Vaso-Vagal FAINTING: WHAT WE CAN DO

All types of fainting hark back to a problem. The problem may be minimal and require a minor adjustment in life style, diet, working conditions, etc. Or fainting might be a sign of things buried deeper in the body signaling dangerous conditions.

So it’s vitally important to consult with a health care professional. And it’s vitally important to do so if your workplace involves machinery, heights and other potentially dangerous situations.

Some things you can do if you’re starting to feel faint –

1. Lie down slowly and carefully if you can or sit down and place your head between your knees. If you can, raise your feet 12” above the level of your heart. Have someone call for an ambulance. If someone near you exhibits signs of fainting, do the same for her or him.

2. If you can, loosen any tight areas of clothing – collar, necktie, shoes.

Source: www.nhs.uk

B 23 and B 47 - on both sides of the area of the lower back at the level of the waist, two and four finger widths out from the spine

3. Direct some cool air toward you or toward the other person.

4. Drink some liquids slowly. Make sure your throat is clear first.
Activating or Stabilizing the Vagus Nerve

To Stop Fainting and Release Vaso-Vagal Distress, Try these Tips

Soft Gentle Pressure on the Eyes can Calm Vaso Vagal Distress

Splashing cold water onto your face activates the dive reflex, which gradually reduce your heart rate.

Here are some acupressure points that can provide quick release of Vaso--Vagal Distress.

This point is in between the anus and perineum

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Water Ingestion as Prophylaxis Against Syncope

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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
- water
- test, tilt table
- hypotension, orthostatic
- norepinephrine

Received March 18, 2003; de novo received May 22, 2003; revision received August 4, 2003; accepted August 7, 2003.
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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.13 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.13−15 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 0.05.13 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.17 Assumptions of proportional hazards were assessed by use of Schoenfeld’s residuals.18 The log-rank test was used to compare survival curves.22 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)**

**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).
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Figure 3. Heart rate (HR) during head-up tilt. Water ingestion blunts increasing heart rate response during upright tilt. $P<0.05$.

![Heart rate (HR) during head-up tilt](image)

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 $16.7\pm4.8\%$ increase in hematocrit without water and $13.8\pm4.2\%$ 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 dihydroxylphenylglycol (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).

Figure 5. Changes in plasma concentration of plasma dopa comparing tilt with water vs tilt without water. Water ingestion attenuated decrease in dopa at 15 minutes after tilt. $P<0.05$. 

![Changes in plasma concentration of plasma dopa](image)
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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 of water ingestion in other conditions and indeed has been applied to reduce the orthostatic tachycardia in patients with orthostatic intolerance. 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 and in patients with recurrent vasovagal syncope. 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. They found a biphasic change in plasma volume. Initially, there was early hemoconcentration, which they ascribed to sympathetic activation. This was followed by hemodilution, presumably because of a postsorptive 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 or increased muscle sympathetic activity 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, ≈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. 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
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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. 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.

Acknowledgments

This study was supported in part by National Institutes of Health grants HL-56693 and RR-00095. We acknowledge the technical expertise of Velma Watkins, RN, and Bonnie Black, RN, and the editorial assistance of Dorothea Boemer.

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**WHAT TO DO IF**

**Vasovagal Syncope**

Cold Towel over eyes with slight pressure
Rest Comfortably
Breathe into a bag if there is hyperventilation

**IMUNE**

International Medical University for Natural Education
Evidence Based Natural Energetic Medicine Education
Vagus nerve stimulation (VNS) is an adjunctive treatment for certain types of intractable epilepsy and treatment-resistant depression.

Vagus nerve action

Vagus, the tenth cranial nerve, arises from the medulla and carries both afferent and efferent fibers. The afferent vagal fibers connect to the nucleus of the solitary tract which in turn projects connections to other locations in the central nervous system. Little is understood about exactly how vagal nerve stimulation modulates mood and seizure control but proposed mechanisms include alteration of norepinephrine release by projections of solitary tract to the locus coeruleus, elevated levels of inhibitory GABA related to vagal stimulation and inhibition of aberrant cortical activity by reticular activation system.¹

Approval and endorsement

In 1997, the United States Food and Drug Administration (FDA) approved the use of VNS as an adjunctive therapy for partial-onset epilepsy. In 2005, the FDA approved the use of VNS for treatment-resistant depression (TRD).²
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Although the use of VNS for TRD has been endorsed by the American Psychiatric Association, the FDA's approval of VNS for TRD remains controversial. According to Dr. A. John Rush, vice chairman for research in the Department of Psychiatry at the University of Texas Southwestern Medical Center at Dallas, results of the VNS pilot study showed that 40 percent of the treated patients displayed at least a 50 percent or greater improvement in their condition, according to the Hamilton Depression Rating Scale. Many other studies concur that VNS is indeed efficacious in treating depression. However, these findings do not take into account improvements over time in patients without the device. In the only randomized controlled trial VNS failed to perform any better when turned on than in otherwise similar implanted patients whose device was not turned on.

Patients

Charles E. Donovan, a study subject in the investigational trial of vagus nerve stimulation therapy for treatment-resistant depression, wrote Out of the Black Hole: The Patient's Guide to Vagus Nerve Stimulation and Depression.

Other uses

Because the vagus nerve is associated with many different functions and brain regions, research is being done to determine its usefulness in treating other illnesses, including various anxiety disorders, Alzheimer's disease, migraines, fibromyalgia, obesity, and tinnitus.

- Alcohol addiction
- Atrial fibrillation
- Autism
- Bulimia nervosa
- Burn-induced organ dysfunction
- Chronic heart failure
- Chronic intractable hiccups
- Comorbid personality disorders
- Coronary artery disease
- Dravet syndrome
- Drop-attacks
- Heatstroke
- Inhibits heroin seeking behavior in rats
- Intestinal epithelial barrier breakdown
- Lennox-Gastaut syndrome
- Memory
- Mood disorders in elderly population
- Myocarditis
- Multiple sclerosis
- Obsessive compulsive disorder
- Peripheral arterial occlusion disease
- Postoperative cognitive dysfunction in elderly patients
- Rasmussen's encephalitis
- Severe mental diseases
- Sepsis
- Spinal trigeminal neuronal
- Transient focal cerebral ischemia
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- Trauma-hemorrhagic shock
- Traumatic brain injury
- Vaginal-Cervical self-stimulation in women with complete spinal cord injury
- Visceral pain-related affective memory

Other brain stimulation techniques used to treat depression include Electroconvulsive therapy (ECT) and Cranial electrotherapy stimulation (CES). Deep brain stimulation is currently under study as a treatment for depression. Transcranial magnetic stimulation (TMS) is under study as a therapy for both depression and epilepsy. Trigeminal Nerve Stimulation (TNS) is being researched at UCLA as a treatment for epilepsy.

Adverse effects

Cardiac

Cardiac arrhythmia has been reported in lead tests performed during implantation of the device along with late onset cardiovascular events.

Sleep apnea

Intermittent decrease in respiratory flow during sleep has consistently been demonstrated in patients with VNS implants. This seems to be due to an increase in vagal tone, a measure of the control the vagus nerve has over the heartbeat. Clinically significant sleep disordered breathing associated with VNS has been described in pediatric and adult patient populations. Most patients undergoing VNS treatment experience an increase of apnoea hypopnoea index (AHI) post treatment, up to approximately one third develop mild obstructive sleep apnoea post treatment, and a minority of patients develop severe obstructive sleep apnoea related to VNS therapy. These obstructive events can be alleviated by decreasing the frequency or intensity of VNS stimulation, by having the patient sleep in non-supine position or by applying positive airway pressure.

Screening for obstructive sleep apnoea (OSA) in patients with a seizure disorder who are undergoing a VNS implant is also important because adequate treatment of previously undiagnosed and untreated OSA is likely to result in better seizure control in these patients.
Patients undergoing vagal nerve stimulator placement are at risk for developing OSA related to the VNS and should therefore be screened clinically for the presence of OSA after the procedure. Continuous Positive Airway Pressure (CPAP) is a viable therapeutic option for patients who develop OSA related to the VNS. Other options include increasing the cycle length or stimulation frequency of the device. With increasing number of indications and the number of patients undergoing the procedure, awareness of this causation is important for appropriate diagnosis and treatment of OSA related to vagal nerve stimulators. \[citation needed\]

Symptoms such as loud snoring or intermittent cessation of breathing during the night or daytime symptoms as behavioral changes, fatigue and sleepiness may alert the patient or parent to the presence of obstructive sleep apnoea, but these symptoms are generally insensitive and a sleep study (diagnostic polysomnography) is generally required to diagnose the presence of obstructive sleep apnoea. The fact that many of these patients are children and may have associated cognitive deficits makes diagnosing the problem even more difficult without a sleep study. \[citation needed\]

**Other**

VNS causes stimulation of the superior and recurrent laryngeal nerves and is associated with problems ranging from alteration of voice (66%), coughing (45%), pharyngitis (35%) and throat pain (28%) and hoarseness (very common) to frank laryngeal muscle spasm and upper airway obstruction (rare). \[66\] "Increased muscle tension," presumably in the upper body, may be experienced during the stimulation period. \[67\] The left vagus has proportionally lesser number of cardiac efferent fibers and placing the stimulator on this side potentially limits the arrhythmogenic effects of vagal stimulation but reversible bradyarrhythmias associated with vagal nerve stimulators have been well described. \[68\] Other nonspecific symptoms include headache, nausea, vomiting, dyspepsia, dyspnea and paresthesia. \[69\]

In the treatment of epilepsy, randomized control trials conducted in the United States indicated that one-third of the patients using a particular vagus nerve stimulation device had some type of an increase in seizures, with 17 percent having greater than a 25 percent increase. In each of the studies, there were patients who had greater than a 100 percent increase. In the E05 study, the range went up to a 234 percent increase, while in the E04 study, it went even higher, to a 680 percent maximum range. \[63\] \[64\]

**Anti-inflammatory activities of vagus nerve stimulation**

The discovery by Kevin J. Tracey that vagus nerve stimulation inhibits inflammation by suppressing pro-inflammatory cytokine production has led to significant interest in the potential to use this approach for treating inflammatory diseases ranging from arthritis to colitis, ischemia, myocardial infarction, and congestive heart failure. \[63\] Action potentials transmitted in the vagus nerve activate the efferent arm of the Inflammatory Reflex, the neural circuit that converges on the spleen to inhibit the production of TNF and other pro-inflammatory cytokines by macrophages there. \[63\] This efferent arc is also known as the Cholinergic anti-inflammatory pathway. \[63\] Because this strategy targets the release of TNF and other pro-inflammatory cytokines, it may be possible to use vagus nerve stimulation instead of anti-inflammatory antibodies (e.g., Remicade or Enbrel) to treat inflammation.

A recent study published in Science (Sept 15, 2011 DOI: 10.1126/science.1209985) demonstrated the existence of acetylcholine-synthesizing T-cells in the spleen that respond to vagal stimulation, resulting in suppression of inflammatory response / TNF-alpha via macrophages.
Methods of stimulation

Direct vagus nerve stimulation

This is currently the only widely used method of therapeutic VNS. It requires the surgical implantation of a stimulator device.

The Cyberonics VNS devices consist of a titanium-encased generator about the size of a pocket watch with a lithium battery to fuel the generator, a lead wire system with electrodes, and an anchor tether to secure leads to the vagus nerve. The battery life for the pulse generator is "between 1 [and] 16 years, depending on the settings [ie how strong the signal being sent is, the length of time the device stimulates the nerve each time, and how frequently the device stimulates the nerve]."[68]

Implantation of the Cyberonics VNS device is usually done as an out-patient procedure. The procedure goes as follows: an incision is made in the upper left chest and the generator is implanted into a little "pouch" on the left chest under the clavicle. A second incision is made in the neck, so that the surgeon can access the vagus nerve. The surgeon then wraps the leads around the left branch of the vagus nerve, and connects the electrodes to the generator. Once successfully implanted, the generator sends electric impulses to the vagus nerve at regular intervals. The left vagus nerve is stimulated rather than the right because the right plays a role in cardiac function such that stimulating it could have negative cardiac effects.[44]

The device is currently only made by Cyberonics, Inc. However, other "wearable" devices are being tested and developed by other companies that involve transcutaneous stimulation and do not require surgery. These devices are similar to TENS (Transcutaneous Electrical Nerve Stimulation) devices that are often used for pain management.[citation needed]

Transcutaneous vagus nerve stimulation (t-VNS)[edit]

This method allows for the stimulation of the vagus nerve without surgical procedure. Electrical impulses are targeted at the aurical (ear), at points where branches of the vagus nerve have cutaneous representation. Specifically the concha has been target for t-VNS.[citation needed]

See also

- Electrotherapy
- Electrical brain stimulation
- Deep brain stimulation
- Transcranial Magnetic Stimulation
- Cranial Electrotherapy Stimulation

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Activating or Stabilizing the Vagus Nerve


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