

TREATMENT OF **PANIC ATTACK** WITH VERGENCE THERAPY

AN UNEXPECTED VISUAL-VAGUS CONNECTION

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ABSTRACT

Panic attacks are a fact of life in today's culture. As much as 10% of the healthy population can suffer a panic attack within a given year. Various methods of treatment have been described in the literature to counteract these panic attacks. It has been noted that it is possible to alleviate panic disorder anxiety by performing convergence therapy. This somatic intervention functions as a vagal maneuver, activating the oculocardiac reflex (OCR) by medial recti traction. It results in bradycardia and other parasympathetic responses. I have found it possible to alleviate panic attack, non-cardiac chest pain and other vagally mediated symptoms by using convergence activity with patients who suffer from panic attacks. I have extended this technique to address non-cardiac chest pain and it may be further extended to patients with other anginal-like pains. It may be possible to alleviate panic attacks, non-cardiac chest pains, and other vagally-mediated symptoms with this technique. The risk-to-benefit ratio is nil. Research is needed to further elaborate the full spectrum of benefits of this novel technique.

Key Words

angina, extraocular muscles, non-cardiac chest pain, oculocardiac reflex, panic attack, somatic interventions, visual convergence therapy, vagus nerve.

INTRODUCTION

Panic Attack

Panic attacks are sudden episodes of multiple sensations that come upon a person who is under stress. The primary cognitive symptom of a panic attack is extreme, anticipatory anxiety. The sufferer can have the perception of a real or imagined threat to themselves or others, fear of dying, "going crazy," and the most frequent worry is the fear of having another panic attack.¹ The emotional stress causes excessive activation of the autonomic nervous system. This is manifested by a parasympathetic overcompensation for the strong response of the sympathetic nervous system associated with the stress. They generally last no longer than about 30 minutes and the somatic symptoms can include increased heart rate/palpitation, non-cardiac chest pain, tremor, breathing difficulty, difficulty with or an inability to speak, nausea or stomach irritability, and severe perspiration.² The inherent response can also lead to temporary loss of bladder control under moments of extreme fear. The vagal portion of this reaction can cause fainting (syncope) because of a sudden drop in blood pressure and heart rate. Vasovagal syncope affects young children and women more frequently than adult males, with the female to male ratio about 2:1. Up to 10% of otherwise healthy individuals may experience an isolated panic attack per year.²

Mechanism For Panic Attack

The vagus nerve is the major source of parasympathetic stimulation to the viscera. It supplies sensory parasympathetic fibers to all the organs, except the suprarenal glands, from the neck down to the second segment of the transverse colon.

Patients who are susceptible to panic disorder may have inherited brainstem loci that are relatively more hyper-excitabile or anomalous. Stress then appears to have an undue effect and excites the brainstem loci.³

Parasympathetic innervation of the heart is via the vagus nerve. The vagus has also been shown to affect the EEG in a frequency and intensity-dependent fashion when electronic vagal nerve stimulation is used. The precise mechanism for this effect however, remains uncertain.^{4,5} The vagus nerve controls a few skeletal muscles, as well. This means that the vagus nerve is responsible for such varied tasks as heart rate, gastrointestinal peristalsis, perspiring, and a fair number of motor movements in the mouth, including speech, and keeping the larynx open for breathing.⁶ The vagus nerve also receives some sensation from the outer ear. Physiological responses with vagal stimulation are known to be almost instantaneous, and this aspect seems to support, at least in part, the clinical observations being reported here.

The parasympathetic innervation to the heart comes from cardiac branches of the vagus nerves. Vagal stimulation slows the rhythm of the sinus node of the heart while simultaneously decreasing the excitability of the internodal pathways.⁷ The net result is a slowing of the heart rate and some decrease in the power of heart muscle contraction. Very strong stimulation can cause cardiac arrest for ten seconds or more.

Treatment For Panic Attack

Somatic interventions have long been used to control the body's responses to stress.⁶ They are often, but not always,

non-invasive strategies for altering physiological and psychological processes by working directly with tissue systems. Mild degrees of intermittent vagal nerve stimulation by daily performance of certain breathing exercises, over a period of several weeks, has been known to lower blood pressure and heart rate in persons with elevated blood pressure or heart rate. The same breathing technique may also stabilize mood and affect. Another effective method used is to take a deep breath, and forcefully blow out through a small hole in your mouth, puffing one's cheeks.⁶ The Valsalva maneuver (attempt to exhale against a closed glottis, or to bear down as if having a bowel movement) also activates the vagus nerve.⁸ These techniques stimulate the vagus nerve, telling it to reset. Other examples of somatic interventions are: electroconvulsive therapy, transcranial electrical stimulation, transcranial magnetic stimulation, deep brain stimulation, and electronic vagal nerve stimulation.⁹

Optometric Therapy Effects On The Central Nervous System

Central nervous system effects of vision therapy (VT) have been reported for about three decades.¹⁰ In the family of binocular dysfunctions, both vergence and accommodative problems can be treated by VT.^{11,12} Recently, a study of convergence insufficiency (CI) has reported that various symptoms associated with CI were resolved with VT and statistically demonstrated the superiority of in-office VT to other forms of treatment.¹³

Ludlam used convergence therapy to restore proper alpha blocking in several patients indicating a reduction in stress and an increase in attention.¹⁰ He empirically introduced convergence techniques to as many patients as possible. He used convergence techniques with those patients who showed attention deficit disorder or attention deficit hyperactivity disorder (ADD/ADHD) and where normal patterns of electroencephalogram (EEG) rhythms are often disrupted.^{14,15}

Based on the experiences with the patient in this report, I have since found that convergence techniques are particularly useful with individuals who have anxiety, panic attacks or panic symptoms. Convergence innervation has consistently shown good results with these patients. These clinical experiences might seem spurious without describing a unifying neuro-physiological mechanism to explain how convergence

therapy might specifically decrease a sensation of panic. There must be plausible reasoning for why the observations might be associated, a rationale to support the responses. It is my belief that the most reasonable explanation is the oculocardiac reflex (OCR). OCR stimulation of the vagus nerve through convergence therapy offers another, perhaps more practical, type of somatic intervention.

Oculocardiac Reflex

To understand the probable mechanism of the OCR, we should consider the neurology of the eye. The ophthalmic division of the trigeminal nerve is the afferent limb of the OCR. The major pathway mediating the OCR⁶ consists of an afferent link through the ophthalmic portion of the trigeminal nerve to the vagus nuclei and an efferent link through the vagus nerve to the heart. Impulses pass through the reticular formation to the vagus nerve's visceral motor nuclei. The efferent limb message is then carried by the vagus nerve to the heart and stomach.¹⁶ The OCR is usually understood to refer to a decrease in pulse rate (bradycardia) upon ocular stimulation—even to the point of actually stopping the heart (asystole). This phenomenon is associated with traction applied to extraocular muscles (EOMs) and/or compression of the eyeball. This reflex is especially sensitive in newborn and children and cardiac arrest may result as a major consequence. The mechanism may come from stimulation of the nerve endings of the EOMs.¹⁷ There are many nerve endings in the EOM insertions including Golgi tendon organs, palisade endings, stretch receptors, muscle spindles, trigeminal nerve terminals and other afferents.¹⁸ These sensors are thought to offer positional information to the EOM nerve nuclei and to higher processes.^{18,19} Veterinarians have used the OCR for some time, compressing the globes of the eye as a vagal maneuver to reduce tachycardia in their patients.²⁰ Strabismus surgeons and anesthesiologists are quite aware of the risks of OCR stimulation. Medicine has devised tests to predict which patients are particularly sensitive to the OCR.^{21,22} Anesthesia is then adjusted based upon the test results to reduce the possibility of an untoward event.^{23,24} Other areas of the head and face can result in vagal changes: pressing on the mandible, maxilla, eye lid or other facial bony structures can produce bradycardia. One may demonstrate OCR by pressing on the muscle mass remain-

ing after enucleation.^{7,16} This is believed to be mediated by the trigeminocardiac reflex.¹⁸

OCULOCARDIAC CONVERGENCE TECHNIQUE

Mechanism

Via the relationships described above, Oculocardiac Convergence Therapy (OCT), as a somatic intervention, appears to result in the stimulation of the primary parasympathetic vagus nerve, resulting in bradycardia (Table 1). The somatic interaction of the OCR offers a credible mechanism for the reduction of stress with its associated panic symptoms, including non-cardiac chest pain. The palliation may also embrace the diverse symptoms of pulmonary and gastric distress that can accompany congestive heart failure patients. Voluntary convergence stimulates the EOM insertions, especially those of the medial recti, plausibly resulting in vago-depressive responses. The site of origin for this phenomenon, most likely, is the muscle insertion into the globe, since topical anesthesia will greatly reduce the OCR.²⁵ OCT as reported here has been seen clinically to result in amelioration of non-cardiac chest pain and also the symptoms of panic attacks and anxiety disorder in humans. The strategy is employed for a minute or less when any distress is sensed in either its prodromal stages or during an active attack. It does not replace medications, but can be employed in conjunction with or, if it is possible to intervene, prior to, the use of pharmacologicals. It is strictly an intervention technique to be used spontaneously, at any needful moment. To this point, there have been no reports of adverse reactions while using this technique, though it theoretically might be possible to create transient asystole if performed too vigorously, or for a prolonged period of time. This potential transient asystole has not been reported, however.

CASE STUDY

A 62-year-old white female, with a history of panic attacks over a period of over six years or more has remained essentially panic attack-free after about half a dozen episodes and OCT interventions over a year's time. The activity was performed strictly as necessary when the symptoms were experienced. The patient later began to suffer classic effects of what were then thought to be congestive heart failure, including what were initially thought to be angina attacks. The attacks were not clas-

Table 1.

OCULOCARDIAC CONVERGENCE THERAPY

METHOD: This technique may be done in any posture but is probably best performed while seated. Any target, a finger, a pen, or a printed card of any sort, is centered before the face at a distance of about four to six inches. A downward gaze position is not a preferred posture, since that angle reduces the role of the medial recti in convergence and they are the muscles that are most stimulatory to the OCR.

The patient is to converge to the near-point target and hold the fixation for about two seconds, then look to a distant target ten or more feet away, for about two seconds. This is one cycle. (A “tromboning” movement will not be as effective, because of the reduced intensity level.)

These near-to-far cycles are repeated for 20 to 60 seconds. If there has been no relief of the panic attack or chest pain in a minute or less, then the usual medications should be taken. The procedure may be repeated as necessary. The response appears to be further enhanced by having the patient attempting to visualize, be mentally aware of, the distance between the near and distance targets (stereoscopic depth becomes involved).

ple procedure has the potential to reduce the distress from both physical and emotional factors. It has the potential to reduce not only tachycardia, but also non-cardiac chest pain and similar cardiac responses in perhaps a majority of sufferers.

Many of the medications used in ameliorating the conditions mentioned above have varying side effects. An effective somatic intervention like the one described here should be welcome to those who experience side effects while taking those medications. The risk to benefit ratio of this technique is virtually not a factor.

CONCLUSIONS

This new and novel method of employing convergence therapy in panic disorder attacks may be extended to non-cardiac chest pain sufferers. It may also be of merit to make an attempt to apply the technique as a palliative to those who suffer gastric disturbances, situational breathing distress, and perhaps even with the prodromal stages of migraine. The response in panic disorder and non-cardiac chest pain has been seen to be swift and effective. Panic attacks and pain began to subside within 20 to 60 seconds. The results lasted from hours to days and upon continued practice, weeks and months.

Alterations in vagal tone have been proposed as a factor in the origin of panic attacks.³⁰ This technique for stimulation of the vagus, and its tentative hypothesis, is based upon an initial set of observations and empirical clinical applications in a limited population. One case is reported above. Any stronger assertion will necessitate rigorous trials. Research in the form of controlled or cohort studies and/or additional case studies can be designed to explore and verify the precise mechanism of this intervention technique and the degree of amelioration that occurs. In doing so, it is difficult to conceive of how a true placebo control group might be designed. The patient is always aware that the treatment and traction upon all of the EOMs can stimulate the OCR, though to lesser degrees than the medial recti.¹⁹ It would also be of great interest to verify if there is—indeed—an accumulative result.

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sic angina, in part because the pains did not come on with or after exertion, nor did resting relieve them substantially. Nitroglycerine sublingual tablets were moderately effective in relieving the pains.

A Cardiolute Stress test and, subsequently, a heart catheterization revealed a healthy heart with minimal to no obstructive disease, so she continued to do OCT with the onset of any angina-like pain or even the prodromal signs of pressure or tingling, as she described it. The results were gratifying in that within four to six cycles, the somatic awareness dissipated. This technique rarely, if ever, failed to work for her and continues to do so for the rare occasions that require intervention. She no longer needs nitroglycerine tablets. The patient has even begun to use this somatic intervention with mild respiratory and gastric distresses that she more rarely experiences, with comparable results. Relief is experienced within a few cycles of OCT. This would be consistent with a generalized vagal response, in all likelihood. This has been clinically replicated in our office with a number of similar sufferers.

DISCUSSION

“Noncardiac” chest pain is present in about 20-30% of patients complaining of chest pain but who have no or insignificant obstruction upon cardiac catheterization.^{26, 27} Bradycardia, regulation of heart rate variability and increased beat-to-beat coherence, are indicators of increased vagal tonus. That is to say, they indicate increased parasympathetic activity. We have been able to clinically observe the impact of OCT upon heart rate regularity

patterns using heart rhythm biofeedback equipment (HeartMath FreezeFramer® 2.0).²⁸ This is especially true when the medial recti are engaged by appropriate head posture (thus isolating their action field) while the heart rate is being monitored. Regulation of heart rate variability has been called cardiac coherence training and is seen when the intervals between beats become more consistently regular through entrainment exercises (activities that can influence neural frequencies to adjust themselves to other functional frequencies, such as respiration).²⁸

The use of oculocardiac convergence therapy may be used as a possible treatment, a somatic intervention, in the event of non-cardiac chest pain, panic disorders and vagally-mediated stomach and breathing problems. And, because of the efferent fibers to the striated muscle of the pharynx and larynx, OCT may even treat globus hystericus, a lump-in-the-throat sensation that causes difficulty in swallowing in the absence of a physical cause. This maneuver is an interesting and novel use of OCR activation, because it appears to manage symptoms of autonomic hyperactivity.

OCT appears capable of providing a quick palliation to these distressing symptoms in many situations. It is quite likely that there may be a conditioning—an accumulative effect, with longer and longer refractory periods—since it has been noted that the vagus nerve response is somewhat trainable, as has been reported in the popular literature on pulse generated vagus nerve stimulation.²⁹ Whether that occurs in this instance or not, over the last five years, this patient believes that it does. This sim-

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