Electrogastrographic and Autonomic Responses During Oculovestibular Recoupling in Flight Simulation

Michael J. Cevette, Gaurav N. Pradhan, Daniela Cocco, Michael D. Crowell, Anna M. Galea, Jennifer Bartlett, and Jan Stepanek

Electrogastrography (EGG) has been advocated as a diagnostic test for clinical evaluation of patients with unexplained nausea, vomiting, and other dyspeptic symptoms. In aerospace medicine, EGG has been a reliable and objective measurement of abnormal gastric activity related to motion sickness (MS) and simulator sickness (SS), a visually generated or computer-generated form of MS (25). Changes in gastric myoelectric activity were also demonstrated during the first few days of spaceflights, when gastric dysrhythmia was concomitant with episodes of nausea and vomiting (8).

Gastric myoelectrical activity can be recorded noninvasively by EGG, enabling accurate measurement of gastric waves, which are typically identified by their slow frequency and low amplitude (100 μV to 500 μV). The dominant frequency (DF) of EGG at 0.05 Hz or 3 cycles per minute (cpm) corresponds to the basic electrical rhythm (BER). The BER reflects the myoelectric activity of the stomach smooth muscle that originates from the pacemaker region in the upper half of the gastric body along the greater curvature. This pacemaker consists of interstitial cells of Cajal that are regulated by autonomic nerves. Increased vagal (parasympathetic) activity augments gastric motility, whereas increased sympathetic activity or vagal withdrawal decreases gastric motility (8). Disturbances of the BER (i.e., gastric dysrhythmia) have been described in disorders of delayed gastric emptying, postoperative nausea or vomiting, MS and other gastrointestinal disorders, and failure of autonomic neural regulation (12). Gastric dysrhythmia has three different patterns: 1) tachygastria, which is increased DF (3 cpm to 4-9 cpm regular activity); 2) bradygastria, which is decreased DF (3 cpm to 1-2 cpm regular activity); and 3) tachyarrhythmia, which consists of a series of premature control potentials followed first by a compensatory pause and then by irregular activity (4-9 cpm) (4).

The first use of EGG evaluation of circularvection-induced MS (onset, time-course, and symptoms) was reported by Stern and colleagues (27), who induced circularvection by rotating an optokinetic drum around the subject’s spinal axis. They reported a shift in DF from 3 cpm to one of 5 cpm to 8 cpm (tachygastria) in all symptomatic subjects (67%). In 64% of these subjects, the tachygastria preceded MS symptoms by 1 min, and returned to the 3-cpm BER within 1 to 9 min after discontinuation of the visual stimulus.

EGG has also been used to evaluate the efficacy of certain pharmacologic interventions. Stern et al. (28) used...
METHODS

Subjects

There were 29 subjects (18 men and 11 women; median age, 27.1 yr [range, 18-38 yr]) who enrolled in a randomized trial approved by the Mayo Clinic Institutional Review Board. Recruitment focused on subjects with no history of MS, vestibular disorders, or gastrointestinal disease. A negative urine pregnancy test was required from all subjects prior to enrollment.

Equipment

Illusory self-motion (vection) was created by exposing the subjects to 20 min of a prerecorded session of flight simulation (X-Plane; Laminar Research). The video consisted of a first-person perspective view of an F-22 Raptor flight over mountains, with balanced combination of pitch, yaw, and roll performed by an experienced fighter pilot. The altitude of the simulated flight was maintained between 1000-2000 ft to create a strong vection from the natural surroundings, including trees, mountain peaks, and lakes.

GVS was integrated in the simulator program to combine visual and vestibular stimulation (OVR simulation) [for technical details, see Cevette et al. (2)]. Visual input was then projected onto a 180° cylindrical screen in front of the subject. Electrogastric data were recorded with a portable EGG recorder (Medical Measurement Systems, Dover, NH) with low and high cutoff frequencies of 1 and 15 cpm, respectively. The signals were amplified, digitized at a rate of 1 Hz, and transferred to a personal computer for further analysis by a commercially available software program (MATLAB R2011a, Natick, MA). IBIs were continuously measured from peak-to-peak blood pressure recordings using a portable, noninvasive monitor (BMEYE Nexfin, Amsterdam, The Netherlands) through a sensor cuff around the finger.

Procedures

Subjects were asked to consume a light breakfast at least 1 h before the study was started. The experiment was carried out in a quiet, temperature-controlled, low-ambient light room in the Aerospace Medicine and Vestibular Research Laboratory, Mayo Clinic; Scottsdale, AZ. Before the simulation session was started, the subjects were randomly assigned to two parallel arms: 1) the OVR group; and 2) the control group. Each subject was tested in only one arm. For the OVR group, a maximum stimulation of 2.5 mA per electrode pair was administered synchronously, along with visual stimulation throughout the test. For the control group, a tactor device was applied to the mastoid electrodes to produce a mild cutaneous sensation of vibration without any resulting motion perception. For the OVR group, four active 20-mm diameter disposable silver/silver chloride gel electrodes (Visys; CareFusion Corp., San Diego, CA) were placed on the upper mastoid (electrode 1 on the left; electrode 3 on the right), forehead (electrode 2), and nape of the neck (electrode 4). A fifth electrode was affixed to the lower nape of the neck as a reference electrode. For the control group, only two electrodes were positioned on the mastoid and connected with the tactor devices.

All subjects had six cutaneous electrodes (Ambu Blue Sensor N; Ambu A/S, Ballerup, Denmark) positioned on the abdomen to record gastric myoelectric signals. The first electrode was positioned below the left rib margin 2 cm from the xiphoid process; the third electrode was placed equidistant between the xiphoid process and the umbilicus; the second and fourth electrodes
were placed, respectively, along the left and the right midclavicular lines, 3 cm below the rib margin and equidistant from the midline; the fifth and sixth electrodes were placed, respectively, along the left and the right midclavicular lines, equidistant from the midline and 3 cm below the line of the second and fourth electrodes. A seventh electrode served as a reference electrode and was placed on the center of the left clavicle. Before each electrode was attached, the skin beneath it was abraded gently to decrease electrical impedance. An elastic band was secured around the chest to detect the respiratory rate.

After the electrodes were positioned, each subject was seated in the flight simulator and instructed to limit body movements. The experiment was then started and included the following three consecutive periods: 1) a 10-min baseline period during which the subject sat quietly in the simulator chair with eyes closed; 2) a 20-min flight simulation with or without OVR; and 3) a 10-min post-simulation period (recovery time) during which the subject remained seated quietly in the simulator chair with eyes closed. The total duration of the experiment was 40 min.

A modified standardized simulator sickness questionnaire was administered to all subjects immediately after completion of the session (13). The questionnaire included a self-report checklist of 15 symptoms (5 gastrointestinal: salivation, nausea, stomach awareness, burping, and vomiting; 7 central: drowsiness, dizziness, difficulty concentrating; and 3 peripheral: warmth, sweating, and general discomfort) for subjective rating on a 10-point Likert scale, with higher numbers indicating greater symptom intensity.

**EGG data analysis:** The differential bipolar signals were derived from the combinations of the six unipolar electrode channels. From the independent combinations, the differential signal between electrodes 1 and 4 (EGG1-EGG4) was selected for further analysis because of their optimum location on the abdomen and the quality of the data they generated compared to that generated by the other electrode pairs. These signals were digitally filtered by applying high-pass and low-pass second-order Butterworth filters at 0.01 Hz and 0.16 Hz, respectively, to eliminate artifacts occurring as a consequence of respiration and movement. A fast Fourier transform was then applied on consecutive 64-s IBI segments with a 32-s (50%) overlap with the previous one in order to estimate spectral power in low frequency (LF, 0.04-0.15 Hz) and high frequency (HF, 0.15-0.4 Hz) spectral components. Given our analysis parameters, the resulting spectral resolution was 0.015 Hz.

**Statistical Analysis**

The EGG parameter (DPIC) and IBI parameter were compared across both groups (OVR vs. control) during baseline, simulation, and post-simulation periods using a two-way mixed model ANOVA followed by the point estimate analysis. DF% in normogastric, bradygastric, and tachygastric ranges during each period were compared between two groups using one-way analysis of variance (ANOVA) followed by the point estimate analysis.

The percentage distribution of EGG spectral power (PSP) in normogastric, bradygastric, and tachygastric ranges was analyzed only through point estimates between periods. Furthermore, IBI and the power of the high frequency (HF) spectral components of IBI were compared between periods in each group by using repeated-measures ANOVA. The correlation of reported symptoms during simulation to the DPIC as well as HF components of IBI was also analyzed. Statistical significance was set at $P < 0.05$. The results are represented as mean (SD).

**RESULTS**

All 29 subjects participating in the study completed the experiment. However, data from two subjects (one from each group) were discarded because of low signal-to-noise ratio in the EGG observed during analysis. There were no significant differences in incidence and severity of SS symptoms between the OVR and control groups. The
mean score for all symptoms was less than 2 on a 10-point Likert scale. The highest rating score was for drowsiness (2.9 in the OVR group vs. 3.5 in the control group).

Visual Analysis of EGG Signals

The gastric activity of the subjects during the baseline, simulation, and post-simulation periods can be represented concisely through running spectral analysis, a classical method based on the fast Fourier transform. As shown in Fig. 1, running spectral analysis represents the EGG data in terms of frequency and its power over the course of the entire experimental time. The relative power change in the three frequency ranges (normogastric, bradygastric, and tachygastric) can be observed visually as the subject goes through the simulation. Fig. 1 compares the gastric activity in two individuals, one in

![A] Control Subject

![B] OVR Subject

**Fig. 1.** The running spectra of the electrogastrography for the entire simulation experiment: A) control subject, and B) oculovestibular recoupling (OVR) subject during the baseline, simulation, and post-simulation periods.
each group (control and OVR) during simulation. Fig. 1A (control subject) shows an increase in the spectral power in the bradygastric frequency range (1–2 cpm) during the simulation period (10th minute to 30th minute) that illustrates the gastric dysrhythmia associated with the nausea that accompanies SS. In contrast, Fig. 1B (OVR subject) shows the effect of GVS on mitigating SS during the simulation period with minimal change in the bradygastric frequency range and the dominance of normogastric activity (2–4 cpm) throughout the experiment.

Percentage of Recording Time with the Dominant EGG Frequency

Fig. 2 shows the DF% in the normogastric (part A) and bradygastric (part B) frequency ranges in the baseline, simulation, and post-simulation periods. As shown in Fig. 2A, the DF% in the normogastric range during the simulation period had a statistically significant difference between the OVR and Control groups \( (F_{(1,25)} = 5.02, P = 0.03, \text{one-way ANOVA}) \). Based on the point estimate analysis, the control group DF% in the normogastric range decreased 22% from the baseline to simulation compared to the 4% increase (i.e., almost constant) in the OVR group. In the OVR group, the DF% in the normogastric range during the simulation and post-simulation periods rarely deviated from the baseline. Conversely, in the control group, the DF% in the normogastric range remained 17% below baseline at the end of the post-simulation recovery period. In addition, as shown in Fig. 2B, the DF% in the bradygastric range during the simulation period also had a statistically significant difference between the OVR and Control groups \( (F_{(1,25)} = 6.31, P = 0.02, \text{one-way ANOVA}) \). Based on the point estimate analysis, a significant 20% increase was observed in the DF% in the bradygastric range from baseline to simulation in the control group compared to a 3% increase in the OVR group. No group differences in DF% were noted in the tachygastric range.

Percentage Distribution of EGG Spectral Power

Fig. 3 shows the PSP in each of the EGG frequency ranges in the baseline, simulation, and post-simulation periods. In the control group, PSP in the normogastric range decreased from baseline by 16% during simulation around the 14th or 15th minute of simulation. However, in the OVR group, PSP in the normogastric range never decreased more than 4% from baseline through simulation (Fig. 3A).

In the control group, the onset of dysrhythmia (indicated by increased PSP in the bradygastric range) began approximately 3 min into the simulation period and continued through minute 12. In the OVR group, the onset of dysrhythmia was delayed until approximately minute 9 of the simulation period. In both groups, the PSP in the bradygastric range decreased after the 12th minute of simulation and returned to baseline levels by the end of the simulation period (20th minute) (Fig. 3B). PSP in the tachygastric range increased in both groups after the 12th minute of simulation (Fig. 3C). By the end of the 20-min simulation period, PSP in the tachygastric range in the control group remained elevated over baseline by 15% compared to a minimal change of 1.5% in the OVR group.

Instability Factors for the Dominant Power

The Generalized Estimating Equations approach introduced by Liang and Zeger (16) was used to evaluate DPIC using a two-way, mixed-model repeated measures design (2 Group × 3 Periods) with a first order autoregressive within-subject correlation matrix and gamma distribution with log a link function. The main effect for Group was not statistically significant between OVR and Control \[ \text{mean (95% CI)} = 0.430 (0.367, 0.504) \text{ vs. } 0.512 (0.427, 0.613) \text{ } P = 0.158 \]. The main effect for Periods showed a statistically significant linear increase from the baseline period to the simulation period \( (P < 0.001) \). A significant interaction effect was found for Group x Periods \[ \text{Wald Chi-Square (2 df)} = 6.179; P = 0.045 \]. The DPIC was similar between OVR and Control groups at baseline and post-simulation, but DPIC increased
substantially more in the Control group compared with the OVR group during the simulation period (mean difference = -0.269, 95% CI -0.541, 0.004); however, the observed difference failed to reach statistical significance (P = 0.053). Furthermore, based on the point estimate analysis, the percentage change of DPIC values from baseline to simulation was 108% (i.e., just over double) in the control group compared to 26% in the OVR group (Fig. 4). No significant correlation was found between DPIC and sum of the scores of all reported symptoms during simulation (control: R = 0.1, P = 0.73; OVR: R = 0.2, P = 0.5).

**IBI Analysis**

Fig. 5A presents the mean IBI values determined during the baseline, simulation, and post-simulation periods. In the OVR group, 20 min of simulation did not result in significant changes in IBI. Conversely, in the control group, there was gradual shortening of IBI, and the difference was significant from the first 5 min to the last 5 min of simulation (F(1,12) = 4.72, P = 0.05; repeated-measures ANOVA). In both groups, IBI returned to baseline during the post-simulation period. The Generalized Estimating Equations (16) approach was used to evaluate IBI using a two-way, mixed-model repeated measures design (2 Group x 6 Periods) with a first order autoregressive within-subject correlation matrix and linear distribution with an identity link function. The main effect for Group was statistically significant between OVR and Control [mean (95% CI) = 0.843 (0.789, 0.898) vs. 0.752 (0.708, 0.797) P = 0.011]. A significant main effect was observed for Period [Wald Chi-Square (5 df) = 45.56, P < 0.001]. A substantial decrease in IBI was seen from baseline through the simulation period, with recovery in the post-simulation period. No significant interaction effect was found for Group x Periods [Wald Chi-Square (5 df) = 3.021; P = 0.697]. With regards to the respiratory rate, the main effect for Group was not statistically significant between OVR and Control [mean (95% CI) = 13.023 (12.423, 13.623) vs.
13.349 (12.741, 13.956) \( P = 0.741 \) and also no significant
effect was observed for Period between the baseline and
simulation periods [mean (95% CI) = 13.22 (12.191
14.244) vs. 13.38 (12.358, 14.407); \( P = 0.567 \)].

**Fig. 5B** presents the power of the high frequency (HF)
spectral components of IBI expressed as absolute \((s^2)\)
units in the plot and as normalized units as the text in
mean (SD) format. These data should be reported in both
types of units for ease of interpretation because a shift in
sympathovagal balance can occur in SS without significa-
cnt changes in total power. This balance can be assessed
by the relation between HF and LF components of nor-
malized units (i.e., relative value of each power compo-
nent in proportion to the total power; e.g., normalized
unit of HF component is represented as HF/(LF+HF)*100)
(18). In the control group, the absolute power of the HF
components was significantly decreased from baseline to
minute 5 of simulation \( (F_{[1,12]} = 5.89, P = 0.03; \) repeated
measures ANOVA); to minute 15 of simulation \( (F_{[1,12]} =
8.62, P = 0.01) \); and to minute 20 of simulation \( (F_{[1,12]} =
7.07, P = 0.02) \). There was no significant difference in the
absolute power of HF components between the baseline
values and the post-simulation values in the control
group; however, a significant difference was observed
when normalized units were evaluated \( (F_{[1,12]} = 4.51, P =
0.05) \). In the OVR group, the 20-min simulation did not
result in any significant changes in HF components of IBI.
This observation suggests that the simulation without
OVR suppressed parasympathetic activity (i.e., decreased
the power of HF components), with a resultant increase
in the dominance of LF components from 47.2\% at base-
line to 51.8\% at the end of the 20-min simulation \( (F_{[1,12]} =
7.27, P = 0.02) \). No significant correlation was found be-
tween the HF components of IBI and sum of the scores
of all reported symptoms during simulation (Control: \( R =
0.17, P = 0.56; \) OVR: \( R = 0.01, P = 0.96 \)).

**DISCUSSION**

Despite the misleading name, SS is a normal psycho-
physiological response of healthy individuals to apparent
motion simulation of significant intensity and/or dura-
tion (19). SS is better defined as a multidimensional sub-
jective syndrome that includes heterogeneous symptoms
[gastrointestinal, central, and peripheral (7)] and whose
measures are complicated by intersubject variability, se-
verity ratings, and differences in incidence. Therefore,
the quantification and comparative analysis of SS re-
sults are often arduous. In this scenario, EGG and car-
diac autonomic responses have proven to be promising
tools for objectively quantifying changes related to
simulation.

Tachygastria has been considered synonymous with
MS and SS ever since Stern and colleagues (27,28) first
reported a shift in the DF of gastric myoelectric activity
from 3 cpm to 5 to 8 cpm during circularvection. How-
ever, the association between tachygastria and symp-
toms evoked by motion and vection stimuli has been
controversial. Tachygastria does not consistently occur
with symptoms of MS, and more importantly, episodes
of tachygastria have been observed in asymptomatic
subjects during simulation (4). Tian et al. (29) reported
that bradygastria and normogastria were related to nau-
sea in 81 pilots exposed to Coriolis stimulation. For this
reason, other authors prefer to use the term ‘dysrhyth-
mia,’ which is defined as the absence of normal (2–4
cpm) slow waves during the observation period, as a
more comprehensive index of gastric myoelectric activity
disruption resulting from real or induced motion percep-
tion (17).

Our findings did not show a predominance of tachy-
 gastria DF during simulation but did indicate a disrup-
tion of gastric activity toward the bradygastric level for
the control group (Fig. 1A). These results were indeed
expected given the low incidence of major symptoms.
Moreover, the incidence and severity of SS symptoms
between the OVR and control groups did not differ sig-
ificantly. These results may have several explanations.
First, we induced vection by exposing subjects to a vir-
tual reality video (prerecorded flight simulation),
whereas in earlier studies, MS was elicited by a rotating
optokinetic drum. Second, technical factors, such as in-
adequate width of field of view, length and/or speed of
simulation, fidelity of virtual scenes, and adaptation
may have been influential (11,14). Third, the length of
exposure to the stimulus might have influenced symp-
toms. Fourth, as previously reported (20), the time and
type of meal consumed before exposure to the simul-
ation might have influenced the susceptibility of subjects
to develop SS.

Norfleet et al. (21) found similar incongruity while
comparing the effects of scopolamine and placebo in
subjects exposed to MS. Although the scopolamine-
treated subjects had significantly fewer episodes of
vomiting than did placebo-treated subjects, the differ-
ence in subjectively reported symptoms was not signifi-
cantly different. Norfleet et al. (21) speculated that the
subjective reporting of MS symptoms was biased both
by subjects being reluctant to admit to having any symp-
toms and by subjects neglecting to mention less distress-
ing symptoms.

**Fig. 4.** Instability factor for the dominant power during the baseline,
simulation, and post-simulation periods.
Despite the absence of severe symptoms between the two groups (OVR and control) in the present study, the significant differences in EGG recordings might be the result of an early presymptomatic phase of gastric arrhythmia that precedes the onset of the tachygastria commonly associated with MS symptoms. The onset of tachygastric PSP illustrated in Fig. 3C was triggered at minute 12 of simulation and continued to increase.

Fig. 5. Comparing A) interbeat interval (IBI)/respiratory rate and B) power spectrum values in high frequency components between 2 groups (OVR vs. Control) during the baseline, simulation (consecutive 5-min epochs), and post-simulation periods. Note: Numbers in part A represent respiratory rate [mean (SD)] and numbers in part B are normalized values of high frequency; high frequency/(low frequency+high frequency)∗100.
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gradually from 25 to 30% at the end of the 20-min simulation. This observation might suggest that although tachygastric FSP increased in the last 8 min of simulation, it might not have been enough to generate severe nausea (even in the control group). Himi et al. (9) also reported gastric motility changes preceding the sensation of nausea in subjects exposed to a movie with oscillation pictures. Based on animal studies, Fukuda and colleagues (6) first proposed a model where the activation of a prodromal sign center for vomiting may elicit gastric changes before the sensation of nausea. If the center is strongly activated, the sensation of nausea may be produced in the cortex.

Our results also showed a statistically significant difference in the DF% of the normogastric (P = 0.03) and bradygastric (P = 0.02) domains between the OVR and the control groups during the simulation period. In the control group, DF% in normogastria decreased by 22% from baseline to simulation and DF% in bradygastria increased by 20% from baseline to simulation. Conversely, in the OVR group, a predominance of normal (2–4 cpm) gastric slow waves (normal gastric myoelectric activity) was observed during the entire simulation period. These results show that the combined visual-vestibular stimulation in the OVR group may have led to a minimization of the intersensory conflict that can provoke SS. These findings are consistent with previous studies where the potential role of transcutaneous electrical stimulation in mitigating MS was investigated. Park et al. (23) found that cathodal galvanic stimulation applied at the mastoid ipsilateral to the irrigated vestibular system during caloric stimulation was able to restore a normal level of gastric motility by reducing the tachygastria induced by the caloric test. Moreover, with the recovery of gastric motility, galvanic stimulation could ameliorate symptoms such as nausea, vomiting, dizziness, and nystagmus (23). Chu et al. (5) reported that applying transcutaneous electrical nerve stimulation to the midline posterior nuchal region and the Zusanli acupoint (anterior tibia) significantly reduced MS symptoms induced by Coriolis stimulation. They also reported that transcutaneous electrical nerve stimulation affected the vestibulo-autonomic components of HRV during MS, resulting in elevated sympathetic activity and suppressed parasympathetic activity. Similar results have been reported by Lacount et al. (15), who investigated the response of the HF components of the HRV to MS. Their results showed a gradual decrease in HF power in response to nausea, as a result of decreased vagal tone (parasympathetic activity) and increased sympathetic activity during MS. Ohyama et al. (22) focused on the LF components of the HRV during visually induced MS and reported an increase in LF without significant changes in HF. Authors concluded that since LF is influenced by both sympathetic and parasympathetic activity, whereas HF is solely influenced by parasympathetic activity, an increase in LF without a decrease in HF is the result of sympathetic arousal during MS (22).

Our results show that the absolute HF power did not change significantly during the 20-min simulation in the OVR group, whereas in the control group it was decreased from baseline at simulation minute 5, minute 15, and minute 20. This observation suggests that simulation without OVR suppresses parasympathetic activity (i.e., results in decreased HF power), with a resultant increase in the dominance of LF power (from 47.2% at baseline to 51.8% at the end of the simulation).

Our findings indicate that electrogastrography and heart rate variability measures are sensitive indices of autonomic changes in subjects exposed to flight simulation that may not correlate directly with subjective reports of SS. These findings suggest that autonomic changes might have their onset in the presymptomatic phase of SS. Therefore, it might be appropriate to monitor them to identify early signs of symptom onset in SS or MS. In addition to more subjective measurements, the quantification of objective autonomic changes could play a major role in the assessment of SS and MS. This study has also shown that OVR technology may have the potential to stabilize the gastric myoelectrical activity and the cardiac autonomic changes commonly associated with SS and MS. More studies involving subjects highly sensitive to MS exposed to more provocative stimuli are required in order to further investigate OVR technology in preventing and/or mitigating SS and MS.

ACKNOWLEDGMENT

This work was supported by Grants N68335-08-C-0294 and N68335-07-C-0043 by the U.S. Department of Defense.

Authors and affiliations: Michael J. Cevette, Ph.D., Gaurav N. Pradhan, Ph.D., Daniele Cocco, M.D., Jan Stepaneck, M.D., and Jennifer Bartlett, B.S., Department of Otolaryngology—Head and Neck Surgery/Audioiology, Mayo Clinic, Scottsdale, AZ, Drs. Cevette and Stepaneck, Division of Preventive, Occupational, and Aerospace Medicine, Mayo Clinic, Scottsdale, AZ, and Michael D. Crowell, Division of Gastroenterology, Mayo Clinic, Scottsdale, AZ; and Anna M. Galea, Ph.D., Vivotonics, Waltham, MA.

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