Title: A jugular vein compression collar prevents alterations of endogenous electrocortical dynamics following blast exposure during special weapons and tactical (SWAT) breacher training

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Abstract

Exposure to explosive blasts places one at risk for traumatic brain injury, especially for special weapons and tactics (SWAT) and military personnel, who may be repeatedly exposed to blasts. In the current study, the effectiveness of a jugular vein compression collar to prevent alterations in resting state electrocortical activity following a single SWAT breacher training session was investigated. SWAT team personnel were randomly assigned to wear a compression collar during breacher training and resting state electroencephalography (EEG) was measured within two days prior to and two after breacher training. It was hypothesized that significant changes in brain dynamics—indicative of possible underlying neurodegenerative processes—would follow blast exposure for those who did not wear the collar, with ameliorated changes for the collar-wearing group. Using recurrence quantification analysis (RQA) it was found that participants who did not wear the collar displayed longer periods of laminar electrocortical behavior (as indexed by RQA's vertical max line measure) after breacher training. It is proposed that the blast wave exposure for the no-collar group may have reduced the number of pathways—via axonal disruption—for electrical transmission, resulting in the EEG signals becoming trapped in laminar states for longer periods of time. Longer laminar states have been associated with other electrocortical pathologies, such as seizure, and may be important for understanding head trauma and recovery.

1. Introduction

Individuals who are exposed to explosive blasts, such as military or police special weapons and tactics (SWAT) personnel, are at increased risk for traumatic brain injury (TBI)(Hicks et al. 2010). TBI is highly prevalent in service members deployed in recent conflicts (Center 2018b; Hayward 2008; Okie 2005; Rosenfeld et al. 2013; Warden 2006). More than 375,000 service members have been diagnosed with TBI since 2000 (Center 2018a), and one prospective study revealed that 9% of approximately 8,000 US marines displayed signs and symptoms for TBI on clinical questionnaires when returning from combat (Drake et al. 2010). Blast-related TBI (bTBI) from exposure to improvised explosive devices (IED) is the most commonly reported source of TBI in military personnel, accounting for approximately 30% to 52% of all cases (Drake et al. 2010; Galarneau et al. 2008). Similar effects of bTBI have also been documented in law enforcement specialists, such as SWAT team personnel (Carr et al. 2015; Littlefield et al. 2016).

The mechanism underlying bTBI is more complicated than the more typical blunt force trauma often associated with TBI. This is because bTBI involves the interactive effects of several different sources of blast injuries beyond the primary blast pressure wave (Nakagawa et al. 2011; Rosenfeld et al. 2013). The primary damage, caused by the initial blast pressure wave, may be a result of kinetic energy passing through soft tissues and fluids of different densities and acoustic impedances within the brain. This mechanical disruption can result in the formation and collapse of miniature vapor pockets (Goeller et al. 2012). This transient cavitation produces differentials in intracranial pressure that may cause damage to surrounding brain tissue, axonal pathways, and capillaries (Delius 2002). Additional primary damage may result from a "blood surge" in which blood from the torso is forced into the brain, causing damage to the circulatory system within and around the brain (Chen and Huang 2011). The remaining sources of damage, which are classified

as secondary (projectiles such as shrapnel), tertiary (contact with environmental objects such as the ground), and quaternary (all remaining causes of injuries) injury mechanisms, also contribute to bTBI and can have serious effects. The lasting complications and associated sequelae of bTBI resulting from any of these mechanisms can have a substantial negative effect on both short- and long-term health outcomes (Heltemes et al. 2012; Reid et al. 2014). However, the mechanical energy imparted by blast waves (i.e., the primary injury mechanism) is the least understood of the injury mechanisms (Chen et al. 2009b), and is our focus here.

SWAT personnel who regularly practice breaching—the action of tactically entering a secured area through a controlled, planned detonation of explosive charges—are exposed to low-level blast charges that may increase their risk of bTBI (Carr et al. 2015; Littlefield et al. 2016). While there is no prescribed "safe" level of blast exposure for the central nervous system (CNS), the level of pressure experienced during breach training does not commonly exceed 4 psi for a single blast (Carr et al. 2016). The breach blast pressure level is much lower than experienced during a combat event where an IED blast can exceed 60 psi (Chandler 2006). However, recent studies have reported evidence of neurological symptoms, such as headaches and memory impairments, after repeated blast training/exposure that were similar to TBI symptoms experienced after larger, single blasts (Carr et al. 2015; Littlefield et al. 2016). Thus, it is paramount for the health and safety of SWAT and military personnel to understand the effects of blast training and repeated blast exposure (even frequent, low level exposure) on the brain and to determine if any such effects can be mitigated by protective devices.

1.1 Protective Potential of a Jugular Vein Compression Collar

Various protective devices, such as helmets, are worn by personnel to limit the effects of blast exposure (Rodríguez-Millán et al. 2017). Helmets add an additional protective layer (in

addition to the skull) in order to protect the brain from blunt force trauma and prevent open head injury. However, when the cranium experiences an external force, even in the absence of blunt force trauma, the brain is exposed to differential accelerations that result in the brain "sloshing" *within* the skull (Benson et al. 2009). Helmets are thus largely ineffective with regard to the primary injury mechanisms of injury associated with explosive blast exposures.

A jugular vein compression collar is an alternative technology for protecting the brain, and it does so from within the brain. The collar applies gentle pressure to the internal jugular vein, which slows venous outflow and thus increases intracranial blood volume. The increased blood volume is hypothesized to surround the brain and provide cushioning which minimizes the intracranial space for rapid changes in brain positioning (i.e., "slosh"). Reductions in slosh are hypothesized to prevent brain deformation—a proposed mechanism of brain injury and concussion (Bayly et al. 2005). Thus, when the cranium is exposed to external forces, such as blast waves, the collar is hypothesized to minimize the extent to which the brain can slosh within the cranium. The efficacy of the collar was initially tested in animal models (Smith et al. 2011; Turner et al. 2012), Neuroimaging studies in humans have revealed that young contact-sport athletes who wore the collar during competitive sport exhibited significantly reduced pre- to post-season functional and structural changes in the brain compared to those who did not wear the collar (Myer et al. 2016a; Myer et al. 2016b; Yuan et al. 2017). For example, youth hockey players who wore the collar over the course of a competitive season did not show changes in white matter microstructure (a known neuroimaging marker of brain injury (Kraus et al. 2007; Niogi et al. 2008)), whereas athletes who did not wear the collar showed significant change in mean and radial diffusivity from pre- to mid-season, despite comparable head impact exposure (Myer et al. 2016a). Similar contrasts were observed over the course of a competitive football season in which changes in white matter

microstructure were significantly greater for athletes who did not wear the collar relative to collar-wearing athletes (Myer et al. 2016b; Yuan et al. 2018). No studies to date have determined whether the collar can mitigate electrical brain changes resulting from blast exposure.

The mechanical energy imparted by blast waves (i.e., the primary mechanism of bTBI) is similar to that which is generated from impacts experienced by contact sport athletes (e.g., football, hockey, and soccer) that can lead to mild TBI (mTBI), or concussion. Since both types of injury are linked to the damaging effects from the transfer of mechanical energy to (and through) the body, we hypothesized that a mechanical-based slosh mitigation jugular vein compression collar could also mitigate the effects of blast exposure on the brain. While the results previously obtained on contact-sport athletes indicate a possible protective mechanism of the collar on neural functioning, previous investigations have not explored whether the collar tempered any changes in endogenous cortical electrical activity. Changes in electrocortical behavior may provide an indication of neural injury at finer timescales than are observable using techniques such as functional magnetic resonance imaging (fMRI). Accordingly, the specific focus of this investigation was on the ability of the collar to prevent changes in the electrocortical behavior—as measured by electroencephalography (EEG)—of SWAT members following breacher training.

1.2 Brain Health, Structure, and Injury

While initial pathological damage from mTBI may be minimal, chronic neurodegenerative effects of mTBI can persist for months and may lead to psychological dysfunction (DeKosky et al. 2010). Synaptic changes following TBI have been linked to functional behavioral changes (Kolb 1999) and, thus, synaptic function relative to recovery is considered an important factor for TBI rehabilitation (Kleim and Jones 2008). In addition, changes in white matter integrity have been observed following TBI (e.g., (Oni et al. 2010; Wozniak et al. 2007) and have been

accompanied by behavioral changes measured by event-related potentials (ERP) using EEG (Dennis et al. 2015). Changes in white matter integrity are often accompanied by axonal injuries, as neurofilament damage induces swelling, which may disrupt axonal transport processes (Liu et al. 2014) and contribute to diffuse axonal injury (DAI). DAI is a common outcome found across all severities of brain injury (Johnson et al. 2013), including bTBI (Mac Donald et al. 2011). Axonal injury, which is generally recognized as a progressive degeneration of an axon's transportation capacity to a final complete disconnection (Johnson et al. 2013), peaks within 24 hours post injury (Gultekin and Smith 1994) and can potentially last for multiple years (Blumbergs et al. 1989; Chen et al. 2009a).

Although the purpose of this study was to measure changes in electrocortical activity using EEG, the abundance of literature demonstrating axonal damage resulting from brain injury provides a foundation for a cautious neural degenerative mechanistic hypothesis. We emphasize that EEG cannot directly measure neural degeneration, but blast related mTBI is frequently associated with axonal injury (Elder et al. 2014), and EEG may contribute to an indirect understanding of how blast-related head injuries may affect axonal integrity. Considering that DAI affects the axons responsible for generating the electrical signals measured by EEG, we hypothesized that potential axonal injuries resulting from breacher training may be detected as changes in the electrocortical behavior of the brain, independent of diagnosed injury.

1.3 Dynamical Characterization of Endogenous EEG Signals

Endogenous bioelectrical activity of the brain in the absence of external stimulation, once thought to be meaningless noise, has gained considerable attention as a way to index fundamental properties of brain structure and function (Buzsaki 2006; Sporns 2011; Stam 2005). For example, a recent systematic review (Gurau et al. 2017) revealed that approximately half of the 40 included

studies observed differences in resting-state EEG measurements between patients with autism spectrum disorder and typically developing participants. Differences in resting-state electrocortical behavior are also highlighted in a review of EEG analyses for assessing the brain condition of patients (in terms of diagnosis, prognosis, and evaluation) with disorders of consciousness (Bai et al. 2017). Consequently, there is a growing focus on quantifying the time-varying (i.e., dynamic) patterns of endogenous EEG activity (Rapp et al. 2015), in particular through the use of analytical tools developed in dynamical systems theory (Bassingthwaighte et al. 1994; Freeman 1994; Kaplan and Glass 1995; Kelso 1995).

Recurrence quantification analysis (RQA) (Marwan et al. 2007; Webber Jr and Zbilut 1994; Zbilut and Webber Jr 1992) is one such tool that has been shown to reveal information concerning the dynamics, transitions, and synchronization of EEG signals while overcoming many real-world signal issues (e.g., short, noisy, and nonstationary time series) (Romano et al. 2005). Essentially, RQA allows one to quantify the time evolution of a system's trajectory through a reconstructed phase space. RQA has been used to study a wide range of EEG phenomena including mild cognitive impairment (Timothy et al. 2017), multiple sclerosis (Carrubba et al. 2012), epilepsy (Thomasson et al. 2001; Zhang et al. 2008), anesthesia (Nicolaou and Georgiou 2014), sleep stages (Song et al. 2004), states of consciousness (Becker et al. 2010), and transitions into and out of epileptogenesis (Rizzi et al. 2016b). The use of RQA in these studies has revealed how EEG dynamics change in response to different experimental manipulations, diseases, and changing body states. Accordingly, RQA's ability to provide information about the underlying dynamics of electrocortical behavior may allow investigators to begin indexing the brain's functional "health."

The ability of RQA to index and quantitatively describe a type of dynamical behavior—*intermittency*—is of particular interest for the current study. Intermittency is a behavior in which

a system displays periods of approximately constant values (i.e., a laminar state) and, in simple terms, irregularly occurring bursts of variation. An optimal mix of laminar and transient states may provide system stability (i.e., the system is able to maintain its own internal state) while preserving the flexibility to alter its behavior as necessary (Kiefer and Myer 2015). Increases in the amount and/or duration of laminar states may indicate that a system is becoming less adaptive or "stuck," while decreases may indicate that a system is unable to maintain a stable behavior state. While this sort of "optimal variability" argument is not new (Stergiou and Decker 2011; Stergiou et al. 2006), its application in the context of laminar indices is relatively novel. Thus, the utility of RQA is that it provides a method to quantify the distribution of laminar and transient states of EEG signals, the changes in that distribution which might arise due to blast-related neuropathologies, and identify EEG-based dynamics that may indicate potentially neuroprotective effects of the jugular compression collar.

1.4 Present Study

The purpose of this study was to utilize RQA to measure changes in endogenous cortical behavior following a one-day SWAT blast training session and compare the difference in personnel who did and did not wear a jugular vein compression collar during the training. Based on the data from our recent neuroimaging studies of sub-concussive head impact in high school athletes (Myer et al. 2016a; Myer et al. 2016b; Yuan et al. 2017), and evidence that internal jugular vein compression prevents neural degeneration in animal models (Smith et al. 2011; Turner et al. 2012), we hypothesized significant changes in EEG brain dynamics—indicative of possible underlying neurodegenerative processes—would follow blast exposure for those who did not wear the collar compared to no changes for collar-wearing personnel.

2. Method

2.1 Participants

Twenty-three male participants (age range: 31 to 68 years, M ± SD = 43.53 ± 9.48 years) from a local police SWAT team were enrolled for this study (one participant did not complete EEG testing and as a result was not included in the analysis). Participants were allocated to one of two study groups based on tactical position[1] to ensure equal distribution of collar (n = 11) vs. no-collar (n = 11) at each position within breacher training line-up. Primary exclusion criteria included history of neurological deficits, previous cerebral infarction, previous severe head trauma, known increased intracranial pressure, metabolic acidosis or alkalosis, glaucoma (narrow angle or normal tension), hydrocephalus, penetrating brain trauma (within 6 months), known carotid hypersensitivity, central vein thrombosis, known airway obstruction, or seizure disorder. The institutional ethics committee approved the project and informed consent was obtained from all participants prior to the study.

2.2 Breacher Training

All participants completed a morning and afternoon session of SWAT breacher training that exposed each participant to blasts. The morning session involved exposures to three C4 blasts with a large stand-off distance of approximately 23m in an open field (a police shooting range). The afternoon session involved a more complex environment at an abandoned bank with shorter stand-off distances with many walls and irregular surfaces interspersed between the officers. Eleven C4 charges were placed on the doors and walls throughout the bank and were intermittently detonated over the course of approximately 3 hours, ranging between 1 and 40 minutes between blasts. Further, following the C4 blasts, approximately 25 flashbangs were detonated (at an

[1] Each SWAT group consisted of a mix of tactical, precision rifle, K-9, medical, explosive and command personnel.

approximate distance of 2.5-5.0 m) during the afternoon session. These blasts occurred within a duration of approximately 30 minutes. See Table 1 for details concerning the blasts, including the number of blasts, mean peak pressure, and mean total impulses each group received.

2.3 Instrumentation and Procedures

Pre- and post-breacher training EEG testing sessions were completed within 48 hours of blast exposure. On the day of the breacher training, subjects were outfitted with pressure gauges (Section 2.5) and those in the collar group were fitted with the appropriately sized collar device (Section 2.4).

2.4 Neck Ultrasound Evaluation and Collar Fitting

At the initial fitting of the collar, which took place prior to the breacher training session, a registered vascular technologist utilized ultrasound to ensure that the proper collar and internal jugular vein responses (IJV) (e.*g*., visual evidence of IJV dilation superior to collar). All measurements, images, and video clips were acquired using a LOGIQ e-unit (General Electric Inc., Fairfield Connecticut) with an ultra-high frequency L8-18i-RS linear transducer.

2.5 Blast wave monitoring and quantification

Each participant was outfitted with a Blast Gauge System (BlackBox Biometrics, Inc., Rochester, New York) to monitor blast exposure. The Blast Gauge System consisted of three wireless pressure gauges that were mounted on the participants' chest, shoulder, and helmet to capture the peak pressure and impulse (i.e., area under the pressure time series) of explosive blasts. The primary reason for using this system was to verify that there were no systematic differences in blast exposure between the collar and no-collar groups during the breaching sessions.

2.6 EEG Procedure and Data Processing

Resting state EEG data were collected while participants sat quietly with their eyes closed for two minutes. Participants wore a 64 electrode EEG cap (Electrical Geodesics Inc., OR, USA). Data from ten commonly utilized EEG channels—Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, and O2—was recorded at 200 Hz (see Figure 1). As this analysis is relatively new in its application to EEG signals, these ten channels were selected due to their commonality in EEG systems and comprehensive coverage of the frontal, temporal, parietal, and occipital lobes of the right and left hemispheres. This process yielded ten times series: one time series of 24,000 data points per channel.

Before performing RQA, each EEG time series was inspected using EEGLAB (Delorme and Makeig 2004) and custom written MATLAB scripts (The MathWorks, Inc.; Natick, MA) to ensure no measurement artifacts existed within the data. If artifacts were present (e.g., eye blink, chewing, talking, head movement, or electrode displacement), the data for that channel was removed. After inspection, the time series were filtered using a 1st-order Butterworth bandpass filter with cutoff frequencies of 0.10 Hz and 40.0 Hz, and the filtered data were then down sampled from 200 Hz to 100 Hz. Only the middle portion of each time series was retained for analysis—the first and last 30 s of data were removed—as this not only reduced computational demands but maintained the largest portion of artifact free data (artifacts were most commonly present during the beginning and end of the EEG recordings). The final pre-processed time series consisted of 6,000 data points and were submitted to RQA.

2.7 Recurrence Plots and Quantification Analysis

RQA (Marwan et al. 2002; Webber Jr and Zbilut 1994; Zbilut and Webber Jr 1992) is the process of objectively quantifying recurrence plots (Eckmann et al. 1987), which visually represent the time-dependent behavior of a system. Recurrence plots depict how a measured trajectory (i.e.,

the measured behavior of a system over time) revisits previous states (i.e., how data values recur in a time series) and, by quantifying the nature with which the trajectory either repeats or does not repeat, it provides insight into important dynamical properties of the system.

Essentially, a recurrence plot is the presentation of an $N \times N$ distance matrix transformed by a Heaviside step function into a logical (binary) matrix. In other words, a distance matrix specifying the proximity of each value of the system's trajectory in a reconstructed state space (Takens 1981) to every other value of the system in the reconstructed state space is turned into a matrix of 0s and 1s. This process entails comparing the values of the distance matrix to a cutoff value called the *radius* that defines a tolerance for what counts as the system revisiting a previous state (i.e., to account for the effects of measurement noise). If a distance defined in the distance matrix is less than the radius, it is assigned a value of 1 in the corresponding logical matrix (which maps directly to the recurrence plot), meaning that a previous state has reoccurred. If the distance matrix value is greater than the radius value, then a value of 0 is assigned meaning the state has not reoccurred.

The entire process of recurrence plot construction depends on several parameters which influence the organization and structure of the plot (Webber Jr and Zbilut 2005). In the current analysis, a variable radius value was selected to ensure each recurrence plot maintained a fixed recurrence rate of 5.0%. This approach is in line with a past investigation of EEG signals (Rizzi et al. 2016a; Rizzi et al. 2016b) and is commonly utilized to prevent the oversaturation of recurrence plots with recurrent points, which may influence the dependent measures derived from the plot (Marwan et al. 2007). A delay (τ) of 125 samples was chosen using the mutual information approach (Roulston 1999) for the purpose of creating time-delayed copies of the original EEG signals to serve as surrogate dimensions for reconstructing the original system's state space to

remove projection errors in the data (Takens 1981). False nearest neighbors analysis (Kennel et al. 1992) was used to select an embedding dimension (the dimensionality of the reconstructed state space) of 6 for the reconstructed state space, and the distance matrix was rescaled using the maximum normalized distance.

Following the construction of a recurrence plot, it is possible to quantify the total number of recurrent points and how they are organized within the plot. Several different measures are used to describe various properties of a recurrence plot and how they relate to the underlying process being measured (see (Webber Jr and Zbilut 2005) for a brief review; also see(Marwan et al. 2007). The organization of the vertical line structures—indicative of laminar states—are of special interest in EEG signals as the periodic dynamics of electrical brain activity can be classified using these measures (Rizzi et al. 2016b). Specifically, *vertical maximum line length* (vMAX) describes the longest period of observed laminar behavior, defined as

$$vMAX = max(\{v_i, i = 1, \dots N_v\}), \quad (1)$$

where N is the number of vertical lines identified in the recurrence plot. Vertical line structures indicate that an observed variable (e.g., a time series of electrocortical activity) is not changing its state for a given period of time, and the number and length of these lines allows investigators to identify features such as intermittency (Marwan et al. 2007; Marwan et al. 2002). In the current study we were specifically interested in vMAX as an indication of the level of intermittency within the electrocortical signal following blast exposure.

2.8 Data Analyses

To determine if there were any systematic differences in electrocortical dynamics due to blast exposure between the collar and no-collar groups, separate independent *t*-tests were conducted for the dependent variables peak pressure, total psi impulse, and number of blasts

obtained using the Blast Gauge System.

For the vMAX measure from RQA, percent change scores were calculated from the pre- to post-blast data for each EEG channel (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, and O2). Negative percent change values indicate that the vMAX measure decreased from pre- to post-blast while a positive percent change value signaled the opposite. After calculating percent change scores, two series of *t*-tests were performed to first investigate if there were significant differences between the groups in the amount of change of the vMAX, and second to determine if the level of percent change in each EEG channel was significantly different than zero (zero indicates no change) across both groups. The first series consisted of independent paired-sample *t*-tests comparing the percent change scores of the two groups (collar versus no-collar). The second series of one-sample *t*-tests were performed to assess if the groups displayed a change from their pre-blast recordings (i.e., are the percent change scores significantly different than 0%). An alpha level of .05 was selected *a priori* to assess significance for all analyses.

3. Results

3.1 Exposure to the Blast Waves During Training

The number of blast explosive waves recorded for each participant ranged from 1 to 12. As shown in Table 1, no significant differences were observed for the number of blast explosions, the average peak pressure, or the total impulse between the two groups (all $p > .05$).

3.3 Recurrence Plots of EEG

As Figure 2 A and B illustrate, for SWAT personnel who wore the collar the overall structure of the recurrence plot from pre- to posttest was similar. In contrast, for those who did not wear the collar (Figure 2 C and D), the recurrence plots appear to lose the distinct structure (i.e., the distinct black and white banded areas) present in the pretest, and the resulting posttest

recurrence plot is, qualitatively, without distinct structure. RQA provides a means for quantitatively capturing these otherwise qualitative and potentially subjective visual features of recurrence plots.

3.3 RQA of EEG

The no-collar group showed percent change scores in vMAX that were significantly greater than 0% in six channels: F3, $t(10) = 3.35$, $p = .007$, $d = 1.01$; C3, $t(10) = 2.66$, $p = .023$, $d = 0.80$; P3, $t(10) = 2.27$, $p = .046$, $d = 0.68$; O1, $t(10) = 2.41$, $p = .036$, $d = 0.73$; P4 $t(10) = 2.27$, $p = .046$, $d = 0.69$; and C4, $t(10) = 3.22$, $p = .009$, $d = 0.97$. All other channels were not significantly different than 0% (all $p > .05$). Alternatively for the collar group, one-sample *t*-tests revealed that none of the percent change scores in vMAX in any EEG channels were significantly different than 0% (all $p > .05$).

Percent change scores for vMAX were significantly different between the collar and no-collar groups for six of the ten EEG channels (see Figure 3). Specifically, vMAX significantly increased from the pre- to post-blast for the no collar group compared to the collar group for EEG channels: C3, $t(9) = 2.44$, $p = .037$, $d = 0.95$; P3, $t(9) = 2.40$, $p = .040$, $d = 0.94$; O1, $t(9) = 3.64$, $p = .005$, $d = 1.33$; O2, $t(9) = 2.50$, $p = .034$, $d = 0.97$; P4, $t(9) = 2.41$, $p = .040$, $d = 0.94$; and C4, $t(9) = 2.14$, $p = .020$, $d = 1.11$. Channels Fp1, Fp2, F3, and F4 showed no significant differences between the groups (all $p > 0.5$). Group means are displayed in Table 2.

4. Discussion

The purpose of this study was to evaluate the efficacy of a jugular vein compression collar in mitigating changes in endogenous EEG activity as measured by RQA. We hypothesized blast-induced changes in the EEG signal, as measured by RQA, from pre- to post-blast for those who did not wear the collar, without similar changes for personnel wearing the protective collar. RQA

was sensitive to distinguishing changes in intermittent electrocortical activity from pre- to post-blast exposure, providing further evidence of RQA's ability to classify endogenous EEG phenomena (Becker et al. 2010; Carrubba et al. 2012; Rizzi et al. 2016a; Rizzi et al. 2016b; Schinkel et al. 2007; Schinkel et al. 2009). Congruent with our hypotheses, SWAT members who did not wear the collar showed altered endogenous EEG activity from pre- to post-blast for six of the ten examined electrodes, whereas no changes were observed in any electrodes for the collar-wearing personnel.

Specifically, in the no-collar-wearing group, participants displayed longer periods of laminar electrocortical behavior after breacher training: vMAX increased significantly from pre- to post-blast for no-collar wearing personnel in six of the 10 examined EEG channels. This indicates that following blast exposure EEG signals from those channels were less likely to vary continuously and instead more likely to exhibit patterns of diminished variation—electrocortical activity returned to and maintained a similar dynamic state (i.e., participants' EEG activity became "stuck")—intermittently interrupted by occasional bursts of variation.

Considering the documented axonal and synaptic damage following blast exposure (Przekwas et al. 2016) and other mTBI (Barkhoudarian et al. 2011), we cautiously propose that the potential damage in our no-collar participants may have been a result of a reduced number of pathways—through neural degeneration—for electrical transmission, resulting in the EEG signals becoming trapped in laminar states for longer periods of time. This may have occurred as a result of either a reduction in the total number of electrocortical behavioral states or from the hindered ability to efficiently switch from one behavioral state to another. In contrast, the collar, which gently backfills the back of the cranium to act as an "airbag for the brain" (Smith et al. 2011; Turner et al. 2012), may have prevented this sort of damage as evidenced by no significant changes

from pre- to post-breacher training. However, this proposed mechanistic explanation in terms of axonal and synaptic damage must be interpreted with caution at this time, as altered electrocortical activity alone is an insufficient basis for identifying the underlying physical consequences of blast exposure.

Our finding that the collar mitigated changes in dynamic electrocortical activity is consistent with past research demonstrating the protective effects of the collar (Myer et al. 2016a; Myer et al. 2016b; Yuan et al. 2017). While previous investigations have shown the beneficial effects of the collar in minimizing microstructural damage to white matter (Myer et al. 2016a; Myer et al. 2016b) and changes in the blood oxygen level dependent (BOLD) signal during a working memory task as measured by fMRI (Yuan et al. 2017), our data indicates that the collar also prevents changes in electocortical activity. This is an important finding, as EEG captures the higher frequency temporal dynamics of the brain than fMRI, which is contingent on the relatively slow hemodynamic response. Further, RQA was successful in differentiating the groups with just two separate, two-minute, noninvasive EEG recordings. Thus, this method holds significant potential as a tool for monitoring changes in CNS activity relative to blast exposure, due to its cost and testing time efficiency compared to other methods, such as fMRI.

There are several limitations to this study. First, in their training exercise the participants were exposed to relatively low levels of blast exposure which may not cause lasting damage; however, the ability of RQA to detect changes in response to relatively low blast exposure attests to the sensitivity of the method. Further, although the current study is unable to test this, RQA may be able to identify subtle dynamical changes in electrocortical activity before a participant has elicited overt symptomology. Second, we did not complete any long-term follow-up examinations to assess whether the changes in vMAX were present after 48 hours, nor did we associate any

changes in electrocortical activity with a behavioral outcome. Thus, it is unknown what effect these changes may have on functional performance required by SWAT personnel and how long these changes may be present. Third, our sample size ($n = 22$) was relatively small and our findings may not extend to other populations. Future studies should include a larger sample size and monitor electrocortical behavior for longer time periods after breacher training to more completely understand the time evolution of electrocortical change following blast exposure.

Despite these limitations, this study makes multiple contributions. First, we have demonstrated the ability of RQA to measure changes in electrocortical activity following blast exposure. Second, we revealed that the collar can mitigate these changes, potentially by protecting the brain from synaptic and axonal damage. Future research should consider complementing our findings with techniques that provide higher spatial resolution, such as diffusion tensor imaging, to test our hypothesized neural degeneration mechanism. Although we are presently unable to conclusively identify the underlying mechanisms associated with blast-related changes in electrocortical activity, in this study we have shown that RQA is a viable method for detecting pathologies resulting from head trauma. Future research should also begin testing the collar with military personnel who are exposed to blasts over weeks or months in which structural and functional brain damage is more pronounced (Han et al. 2014; Vakhtin et al. 2013). The results from the present study could also be used to develop *a priori* regions of interest (ROI) analyses for fMRI, based on the functional relevance of significant EEG changes measured using RQA. For example, electrode channel C3 is functionally related to sensory and motor function (Zaepffel et al. 2013), thus an ROI such as the supplementary motor area could be used in seed-based analyses for fMRI. This would provide important insight into brain regions affected by TBI that may have previously been unexplored.

5. Conclusion

As indexed by vMAX, SWAT personnel wearing the collar during breacher training involving repeated blast exposures did not display changes in electrocortical behavior from pre- to post-breacher training. However, as revealed by RQA, the no-collar wearing SWAT group experienced increases in vMAX. Increases in vMAX are indicative of lengthened laminar states, which we hypothesize may be a result of neural damage, but requires further investigation with other techniques such as MRI. Although the no-collar group experienced changes in endogenous neural behavior, future studies need to investigate the possible connection of changes in intermittent electrocortical behavior and functional behavior. That is, understanding how changes in endogenous electrocortical behavior relate to changes in functional behavior is crucial in a holistic understanding of damage resulting from repeated low level blasts.

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