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Judith Lauter
Stephen F Austin State University

Elizabeth Mathukutty
Kids Developmental Clinic

Brandon Scott
University of New Orleans

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2aPP7. How can a video game cause panic attacks? 1. Effects of an auditory stressor on the human brainstem

Judith Lauter*, Elizabeth Mathukutty and Brandon Scott

***Corresponding author's address: Stephen F. Austin State University, Nacogdoches, TX 75965, jlauter@sfasu.edu**

The auditory brainstem response (ABR) was recorded during simultaneous binaural presentation of two types of sounds: 1) condensation clicks presented through in-the-ear earphones at 43.1/sec, 60dB nHL; and 2) recordings of breathing sounds, presented through supra-aural headphones, at levels adjusted by participants to be equivalent to the clicks. In alternate blocks, the breathing sounds were either: 1) a recording of quiet breathing (blocks 1, 3, 5); or 2) a recording of erratic (stressed) breathing (blocks 2, 4). The erratic breathing was modeled on a video game soundtrack in which the character was represented as running, wounded, and frightened. Four 2048-sweep ABR waveforms were collected in each of the five blocks, and the mean amplitude of ABR peak V was calculated over each set of four waveforms. Results indicate a significant decrease in the amplitude of ABR peak V during erratic breathing vs. quiet breathing. Implications include: 1) new evidence of the effect of selective attention on the ABR; 2) the potential for using auditory stressors to study the central physiology of emotional responses in humans; and 3) clues to physiological correlates of the effects of certain video games known to evoke panic attacks in susceptible players.

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INTRODUCTION

As reviewed by Anderson and Dill (2000), concern has been voiced for many years regarding the possible negative psychological, social, and even physiological effects of violent movies, television, and video games. Violent video games have received particular attention because they go beyond the passive viewing of movies and television to engage the player actively in the vicarious, "virtual" violence of the game.

There are video games that are not violent (referred to as Casual Video Games, CVG), and some of these have been shown to reduce rather than increase stress signs in players – cf. Russoniello et al., 2009). However, the genre known as "action" games (fighter, shooter, and sport games such as boxing, racing, etc.) has seen a dramatic escalation in violence since such games were first introduced during the 1990s, represented by Mortal Kombat, which according to Anderson and Dill (2000) was the most popular game of 1993. In 1998, Dietz found that of 33 games surveyed, 80% involved homicidal violence, and 21% included violence against women.

Action games have been shown to activate the body's stress system, in particular the primitive "fight/flight" sympathetic component of the autonomic nervous system (Segal & Dietz, 1991; Calvert & Tan, 1994; Anderson et al., 1995; Ballard & Weist, 1996; Erb et al. 1998; Anderson & Dill, 2000; Hebert et al., 2005; Wang & Perry, 2006; Vella & Friedman, 2007; Sharma et al., 2006; Ravaia et al., 2008; Mellecker & McManus, 2008; Borusiak et al., 2008; Raudenbush et al., 2009). Games have also been reported to induce seizures in certain at-risk individuals (for recent reviews of this substantial literature that extends back into the 1980s, cf. Kastelijn-Nolst Trenite et al., 2002; Chuang, 2006; and Shoia et al., 2007).

The evidence that violent games can also cause feelings of anxiety, and even lead to full-scale panic attacks, is to date anecdotal rather than formal. However, the extensive literature on *peripheral signs* of sympathetic arousal cited above, combined with findings from a few reports demonstrating *central* activation involving the limbic system (cf. fMRI studies such as Mathiak & Weber, 2006; and Hoeft et al., 2008; and EEG results such as Thalemann et al. 2007, and Salminen & Raviai, 2008) certainly suggest that, from a neurological standpoint, feelings of anxiety and panic are entirely consistent with the physiological effects that have been demonstrated.

The implications of children and adults spending many hours per week (or even many hours per day, in the case of "frequent" gamers or even "addicted" ones -- cf. Griffiths & Hunt, 1998; Tejero Selguero & Morgan, 2002; Sun et al., 2008) in a state of high-stress autonomic activation raise a variety of serious issues. These involve not only effects on *concurrent behavior* (e.g., increased aggression [Cooper & Mackie, 1986; Dill & Dill, 1998; Kirsh, 1998;], changes in academic performance [Creasy & Myers, 1986], increased "social anxiety" [Lo et al., 2005]; sleep disturbance [Higuchi et al., 2005, Dworak et al, 2007], and extremes of psychopathology such as the Columbine murders carried out by two violent-game aficionados [details outlined in Anderson & Dill, 2000]), but also the possibility of negative impacts on *future health* as the result of years of chronic sympathetic arousal ("elevated sympathetic tone"), with direct consequences for multiple body functions influenced by autonomic activation, including the cardiac, gastrointestinal, reproductive, and immune systems.

Although such grave possibilities merit attention from scientists, a complete objective evaluation of game effects has been hampered by a number of factors. These

include: 1) the lack of a good theoretical construct regarding the dynamic nature of brain/body relations that could make it possible to connect physiological changes in cortex, subcortex, and body periphery with each other and with behavior, all of which are clearly important for guiding experimental design as well as interpretation of results; 2) the lack of a useful theoretical construct of individual differences, necessary for understanding the cause of the dramatic differences from person to person (including gender differences) that have been observed formally (e.g., Winkel et al, 1987; Yambe et al., 2003; Thalemann et al., 2007; Vella & Friedman, 2007; and Ravaia et al., 2008) as well as informally, in both the attraction and reactions to violent games; and 3) the enormous source of variation presented by the games themselves in terms of the sensory, motor, emotional, and cognitive environments they incorporate, which can make it difficult to generalize conclusions across different games.

The current study is designed to demonstrate the application of a set of theoretical and procedural methods as a first step toward addressing these three factors, and thus offer suggestions for improving and expanding the study of video-game effects on brain, body, and behavior.

(1) A dynamic view of brain/body relations: The Handshaking Model of Brain Function. The Handshaking Model (Lauter 1998a, 1999b, 2004, 2008) is an extension of ideas first advanced by J. Hughlings Jackson in the19th century (cf. Lauter 1998a for details) regarding the effects of neurological damage. Jackson suggested that neural injury resulted in one of three general conditions: loss, irritability, or release. The term *release* invoked neural connectivity, and was Jackson's interpretation of clinical states such as spastic paralysis, which he saw as due to hyper-responsivity (via hypersynchronicity) in lower motor neurons (LMNs) in the cord that resulted from damage to upper motor neurons (UMNs) in motor cortex, which normally exerted top-down control on LMNs in the resting state as well as during motion.

Jackson's idea of release was limited to the obligatory top-down control situation found in the somatic motor system, but the Handshaking Model re-interprets the concept as a more general model of dynamic physiological relations, that is, 1) applicable to physiological relations of all types (not only top-down control), 2) involving all three body/brain axes (not just rostro-caudal), and 3) active in one way or another in all neural sub-systems (not only somatic motor).

Guidelines for experimental design and interpretation derived from the Handshaking Model make it possible to study activity at any level of the nervous system, the body periphery, or even in behavior, and draw meaningful conclusions about how all of these are related to each other. These methodological ideas have been operationalized in the form of the Auditory Cross-Section (AXS) Test Battery (Lauter, 2000, 2002, 2004). The AXS Battery is a coordinated test battery, that is, one in which individual assessments are chosen so that the results on each of them can be interpreted in the context of results on all the others.

Test design and interpretation of results using the AXS Battery are based on considering the auditory system as a microcosm of the 'neural style' of an individual, such that a 'neurological fingerprint' derived from the battery's 'cross-sectional' look at the auditory system yields insights into the way that a person's nervous system works in general, specified in terms of the dynamic relations along all three brain/body axes: rostro-caudal, right-left, and dorso-ventral.

This makes it possible to describe links between: a) brain, b) physics and physiology at the body surface, and c) behavior, and thus offers a neurobiological, whole-system means for studying the physiological correlates of individual differences of all kinds, including those related to video games.

(2) A new approach to individual differences: The Trimodal Model of Brain Organization. Understanding the nature and origin of individual differences in the attraction and reactions to violent video games may be extremely important not only for designing and evaluating the results of individual experiments, but also for addressing the sociological implications of games.

A new theory of individual differences that may be helpful in this enterprise is the Trimodal Model of Brain Organization (cf. Lauter, 1998a, 1999a, 2002, 2004, 2007, 2008) that incorporates both the Handshaking Model reviewed above, and the EPIC Model of Functional Asymmetries (Lauter, 1999a, 2002, 2004, 2007, 2008) which itself offers a new neurobiological schematic accounting for a broad spectrum of right-brain/left brain specializations.

The Trimodal Model identifies three principal "brain types" created as the result of a continuum of different levels of prenatal hormone (testosterone) exposure: polytropic (zero to very low exposure), middle (moderate exposure), and focal (high exposure). The three types are distinguished from each other in terms of a broad spectrum of anatomical, physiological, and psychological features that include some that are important for judging reactions to violent games, such as differences in the level of resting sympathetic tone, and also in the "set-point" of rostro-caudal relations, a feature that, according to the Handshaking Model, is associated with dramatic differences in response to external stimulation.

For example, some brains are characterized by a "quiet cortex" that requires high levels of external stimulation to achieve a "normal" state of rostro-caudal relations (reminiscent of the "extravert," stimulus-seeking personality type described by Eysenck, 1967). Other brain types in which cortical power is already at a "normal" level may be very resistant to video games, and find them irritating and repulsive. Still other brains may be susceptible to being "captured" by the sensory stimulation of games, but find their violent aspects unnatural and disturbing. These last brains may be the ones most prone to panic attacks, brains in which internal arousal is already at normal levels, and the overstimulation of games, combined with their strict time demands for rapid reactions and violent action (both of which may be difficult in certain ways for these brain types) drives them into a state of anxiety and/or panic.

(3) An analytic approach to game effects based on "unpacking" their multivariate nature. Violent video games employ "immersive stimulus environments" (cf. Allison & Polich, 2008) based on the manipulation of a large number of variables – sensory, motor, emotional, and cognitive – that affect players in various ways, all of which should be interpretable in objective, neurologically-relevant terms.

For instance, the multivariate, multidimensional nature of games can be seen as targeting: 1) *whole-system sensory activation* (i.e., demanding rapid two-way [caudal-rostro-caudal] communication linking bilateral receptor surfaces with bilateral association cortex in all three somatic sensory systems [visual, auditory, somatosensory]); 2) *whole-system motor activation* (two-way communication between bilateral association cortex and bilateral effectors [and kinesthetic receptors] in the somatic motor system); 3) *rapid*

sensorimotor (dorso-ventral) coordination and response, required for the fast reaction-time responses that are de rigeur in most violent games; 4) *autonomic (sympathetic-only) activation* (central limbic arousal implicating prefrontal cortex - diencephalic limbic connections and their communication with sympathetic ganglia and terminals affecting many body organs, including heart, digestive tract, urinary and anal sphincters, and genitals); and 5) *prefrontal cortical activation* coordinating cortico-cortical connections throughout neocortex (supporting processes for goal-setting, planning, selective attention, etc.)

One analytic approach to understanding the effects of such a multivariate environment would be to: 1) isolate the different aspects of game presentation (the nature of the particular sensory stimuli, required motor gestures, emotional vectors such as time pressures, etc.) and then 2) examine their effects both alone and in combination on brain, body, and behavior.

There is a substantial literature on the effects of the rapidly fluctuating types of *visual* stimuli used in violent games, apparently motivated by the fairly frequent phenomenon of "video-game epilepsy (VGE)" that has been recognized for more than two decades (cf. the reviews cited earlier). Researchers have not only been interested in identifying which game features evoke seizures (for instance, not all cases are due to simple photosensitivity – cf. Takahashi et al., 1995; Funatsuka et al., 2001), but also in establishing which individuals are most susceptible (cf. Yambe et al., 2003).

This obviously includes at least some individuals with a history of epilepsy; for instance, some researchers suggest that all individuals with photosensitive epilepsy may be at risk for VGE (cf. Fylan et al., 1999). However, others note that for many if not the majority of VGE cases, there is no previous seizure history (cf. Ferrie et al., 1994; Kastelijn-Nolst Trenite et al., 2002), and often brain imaging and EEG reveal no neural abnormalities in these brains (Graf et al., 1994; McAbee & Wark, 2000). Concerned with susceptibility questions, some have called for better epileptic-seizure warnings to be included with games; for instance, in a report on seizures associated with MMORPGs (Massively Multiplayer Online Role-Playing Games), Chuang called for "an appropriate health warning to MMORPG players" (Chuang, 2006).

Researchers have identified several *visual* triggers of VGE: flashing lights, special figure patterns, scene-changing (cf. Maeda et al., 1990); high temporal frequency in general (Krolak-Salmon et al., 2003); rapid hand manipulation and spatial processing (Inoue et al., 1999), and "steady maximal brightness" (SMB – Ricci & Vigevano, 1999). VGE-inducing variables have even included screen flicker rates (e.g., 50 vs. 100 Hz – cf. Fylan & Harding, 1997), and the way this may interact with visual components specific to the game itself.

In contrast, although there are also many *auditory* components of violent games (gunshots and other sounds of weapons or explosions; vocal "human" sounds such as shouts, groans, screams, heavy breathing; screeching and percussive sounds associated with vehicle crashes, walls collapsing, etc.), very little has been done to study the effects of auditory game features. The only studies we have been able to identify to date that focused on the effects of any type of auditory stimuli from commercial games were reported by Loba et al. (2001) and Hebert et al. (2005).

The first of these (Loba et al., 2001) was concerned with identifying which features in a video lottery terminal (VLT) game might be changed to make the game less

attractive to pathological gamblers. Along with two non-auditory features, the researchers manipulated the presence/absence of sound, which they allowed to co-vary with game speed (fast/slow). Results suggested that by making the game both slower and silent significantly reduced the attractiveness of the game to pathological gamblers (as judged by self-rating responses from participants). While this finding suggests the general importance of some types of sound as an arousing stimulus, it does not provide specific information as to *which* acoustic parameters may be most important for causing such an effect.

The second study (Hebert et al., 2005) addressed the music employed in a violent game. The music was described as being written in a "techno-pop style" and consisting of synthesized bass sounds repeating a single note or simple two- and three-note patterns in a "steady and relatively fast rhythmic pattern." Subjects played the game under one of two conditions, either Music On (at maximal available levels) or Music Off; all other sound effects were turned off for all players. At intervals throughout the gaming session, salivary levels of cortisol were collected to index physiological arousal. Results indicated the Music On group showed higher cortisol levels than did the Music Off group, and the authors concluded this indicated that "the built-in sound environment of video games… entails a measurable physiological response in the organism" (p. 2377). Although since only a single aspect of 'the built-in sound environment' was examined, the terms of this conclusion are somewhat more generic than merited by the experiment, the findings are suggestive and certainly support additional examination of this and other auditory features employed in games.

Design of the current study. The current study was suggested by the information that certain breathing sounds used in a violent video game had recently been revised due to reports that players were experiencing anxiety and panic attacks at a rate that was greater than with previous games. We obtained a copy of the original game and identified audio segments of the game in which erratic breathing sounds were heard while the character was represented as frightened, wounded and running.

Initial personal observations by the experimenters indicated that these erratic breathing sounds were indeed disturbing, and informal testing of a number of peripheral sympathetic indices (heart rate, blood pressure, galvanic skin response, respiratory rate) suggested that the erratic breathing sounds might be effective as sympathetic stressors. That is, they might serve as a "pressor" (sympathetic activator) analogous to a "cold pressor," where cold is used to stimulate the sympathetic system, such as placing one hand in ice water.

This study is the first in a planned series designed to examine the effects of this type of breathing sound on the human nervous system, from body periphery to cortex, using the cross-sectional methodology of the AXS Test Battery as a guide for series design. Of all the choices made available under AXS-Battery testing, the brainstem was chosen for the first experiment because of its role as a "fulcrum" linking cortex (presumably the source of top-down physiological vectors related to attention) with peripheral auditory physiology. The brainstem is also clearly important as the location of centers for control of heart and respiration, which are related to many observed video-game effects. Future experiments will extend our examination of the effects of these sounds to other aspects of brainstem response, the cortex above, and to physiological

functions peripheral to the brainstem such as those affecting ear, eye, hand, heart, voice, and respiration.

METHODS

Stimuli. Because the erratic breathing sounds in the original game were mixed with other sound effects (gunshots, rustling leaves, etc.), it was not possible to simply excerpt portions from the soundtrack for use in the experiment. One experimenter (B.S.) who was very familiar with the soundtrack listened to the game's erratic breathing as a model, and recorded 5 min of his own erratic breathing onto a Macintosh MacBook Pro using GarageBand software and a headset microphone. A Kay Elemetrics CSL 4400 speech-analysis system was used to compare this digital recording to the game's breathing sounds, and the recording was judged both by ear and by eye to be a good facsimile of the original. The same speaker also recorded 5 min of calm breathing for use in the study.

Sample waveforms of the calm and erratic-breathing recordings are displayed in Fig. 1; note that the time windows are of different lengths (8 sec for Calm, 4 sec for Erratic), to better compare the fine temporal structure of the two types of breathing. Breath-group (inhale + exhale) timing for calm breathing was approximately 20 breaths per minute, while the erratic-breathing rate was more than three times as fast (approximately 70 per minute). The long-term average spectrum (LTAS) of the calm and erratic breathing sounds as measured on the Kay 4400 revealed very similar spectral envelopes, with somewhat more energy above 3 kHz for the erratic as opposed to calm breathing. However, up to and including the spectral region of the ABR clicks (1-2 kHz), the spectra of the two breathing sounds were virtually identical.

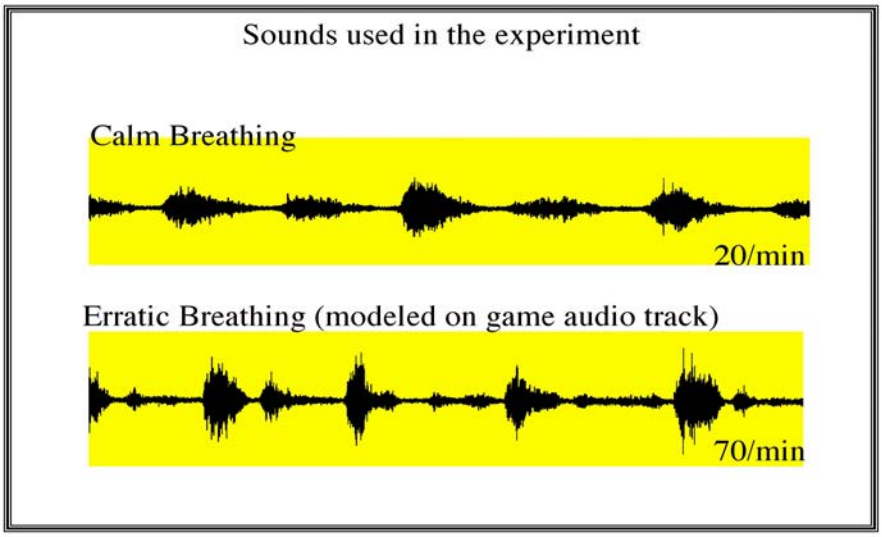

Fig. 1. Representative waveform segments from the Calm sounds (top panel, 8-sec window) and Erratic sounds (lower panel, 4-sec window).

Subjects. Participants were 12 students recruited from undergraduate and graduate students at Stephen F. Austin State University (SFA). Candidates were told they were being recruited for an experiment testing the effects of different types of breathing sounds on the human brain. Eleven were female, one was male, and ages ranged from 20 - 36, with a mean of 23 years. All were screened to have normal hearing between 250 and 6000 Hz (thresholds no greater than 15 dB nHL). Procedures for the experiment were explained to each participant individually, and informed consent was obtained.

Design. The independent variable was the type of breathing, either erratic or calm. The dependent variable was the amplitude of peak V of the auditory brainstem response (ABR), the variable most often used in audiological applications of the ABR. The ABR stimuli were binaural condensation clicks, presented at 43.1/sec at a level of 60 dB nHL. Other ABR test parameters (filter settings, window length, etc.) were standard for ABR data collection.

The design was within-subject, repeated-measures, with blocks arranged in an off-on-off sequence: blocks 1, 3, and 5 were based on responses to ABR clicks + calm breathing, while blocks 2 and 4 utilized ABR clicks + erratic breathing. Statistical analysis consisted of a 2-tailed t-test comparing group ABR peak V amplitudes calculated for calm vs. erratic blocks.

Procedures. For testing, each participant was seated in a comfortable recliner in a sound-treated, electrically-shielded test room in the Human Neuroscience Laboratory at SFA. Four nine-mm silver-silver disk electrodes were placed at: Cz (vertex – active), A1A2 (earlobes – reference) and Fpz (forehead – ground), and impedances were checked using a Biologic Traveler evoked-potential system and maintained below 5 kOhms at all sites throughout testing.

As electrodes were being placed, the participant was told about the sequence of testing: 1) first, they would be asked to help make some adjustments in the loudness of test sounds, to make sure all sounds were presented at the same level; 2) second, they would be tested in several 4-min blocks with combinations of simple clicks plus some breathing sounds. In some blocks, breathing sounds would be slower; on others, they would be faster. Once the levels were adjusted and testing started, lights would be turned down in the room, and they were to remain quiet and relaxed until the end of the testing. They were encouraged to relax all muscles in the head and neck, so that recordings of brain waves would not be affected by movement. (This instruction was included not only to ensure artifact-free recording, but also to establish an attitudinal state of relaxation and calm.)

Once electrodes were in place, the participant was told they were going to be asked to make a loudness comparison of two types of sounds heard in both ears – simple clicks (used to collect the ABR) and breathing sounds. The first comparison would involve a slower breathing and the second a faster breathing. They were told that in each case, the level of the clicks could not be changed, but the level of the breathing sounds could, and that once the sounds started, they were to tell the experimenter whether to turn the breathing sound louder or softer, until it matched the loudness of the clicks.

Insert earphones (for ABR testing) were placed in the external ear canal bilaterally, and stereo supra-aural earphones (for delivery of the breathing sounds) were placed over the ear canals. Binaural clicks were initiated using the Biologic, and the

breathing sounds were presented binaurally using GarageBand on the MacBook Pro. The participant's judgments of breathing-sound level were used to adjust the presentation level of first the calm breathing and then the erratic-breathing sound via GarageBand's level adjustment routine. The adjustment values in dB were read from the GarageBand screen, recorded on the session log sheet, and used in subsequent test blocks for that participant, to ensure that each breathing sound was presented at the level judged by that individual to be equivalent to the level of the ABR clicks.

Once adjustments were complete, electrode impedances were checked again, the participant was reminded to remain quiet and relaxed, the room was darkened and the door closed. Clicks were initiated first, then the calm breathing sound, and four 2048-sweep ABR waveforms were collected while the 5-min calm-breathing recording was played (Calm block #1). Following this, the erratic breathing sound was loaded, its adjustment level set, and another four ABR waveforms were collected during the 5 min of erratic breathing (Erratic block #1). This sequence of calm breathing/erratic breathing was repeated once more (Calm 2 then Erratic 2), followed by a final calm-breathing block (Calm 3) to end the session.

Electrodes were removed, and the participant excused.

Data analysis employed the Biologic cursor and screen to retrieve amplitude values for ABR peak V for all waveforms. The selection of the exact location of peak V on each waveform was guided by methods developed for the AXS Test Battery, based on our Repeated-Evoked Potentials (REPs) protocol (e.g., Lauter & Loomis, 1988; Lauter & Karzon, 1990, Lauter & Oyler, 1992, Lauter & Wood, 1993, Lauter, 2000). The values were entered into an Excel spreadsheet and mean values were calculated over the four waveforms per block for each participant. These mean values were then tested in Excel using a 2-tailed t-test, to compare peak-V amplitudes for Calm vs. Erratic conditions. The comparison to be reported here used Calm blocks 1 and 2, and Erratic blocks 1 and 2. (Essentially the same results were found when data from Calm block 2 were replaced by values for Calm block 3.)

RESULTS

Because this is a study about within-subject changes in ABR amplitudes in response to different test conditions, it may be of interest to compare current data with data from a previous experiment from our laboratory that involved no changes in test conditions.

The inset of Fig. 2 presents data for five subjects from an experiment designed to document the nature of *spontaneous* within-subject changes in peak amplitude of ABR peak V, that is, in a session where test conditions (rate and level of clicks, etc.) were held constant across blocks. No sounds other than the condensation clicks were presented. Although, as indicated by the ordinate, there are definite individual differences in peak-V amplitude, there is *minimal within-subject variation* across the five blocks (there is no block-to-block change larger than .08 uV).

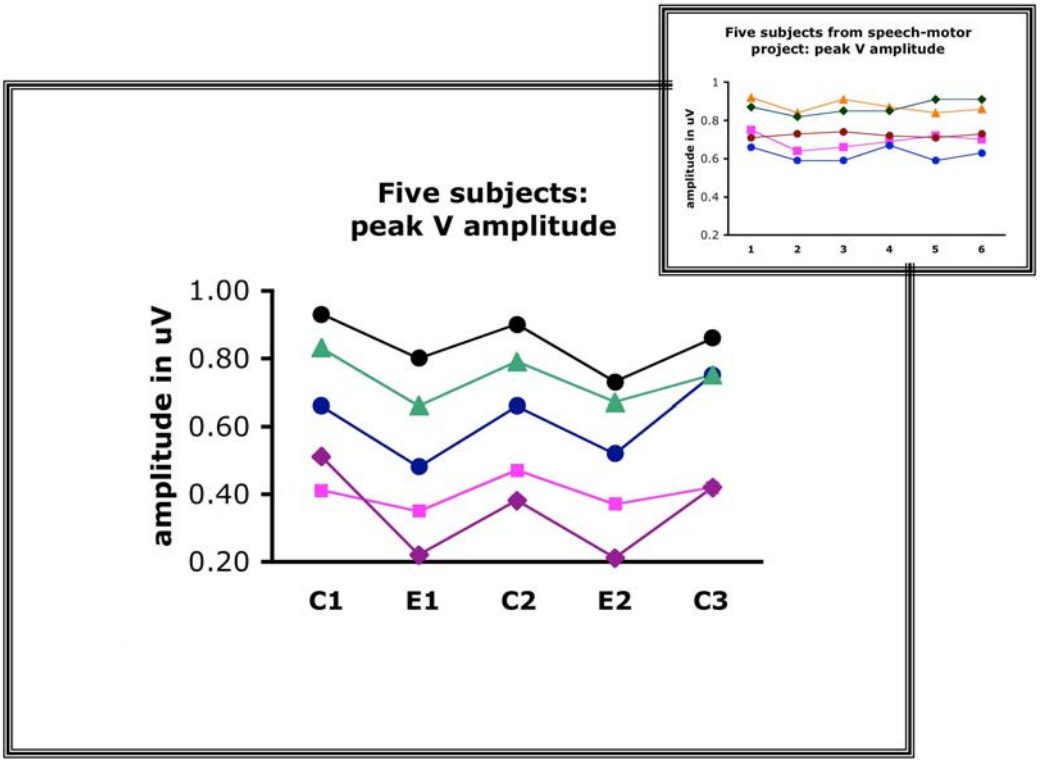

Fig. 2. Inset: plot of ABR peak-V amplitudes for five subjects, simultaneous fluctuations over six test blocks (stimuli = ABR clicks only). Main graph: ABR peak-V amplitudes for five subjects from current study; block conditions are noted on the abscissa (C = ABR clicks + Calm breathing; E = ABR clicks + Erratic breathing).

In contrast, the main graph of Fig. 2 presents data for five participants from the current study. Again, individual differences in peak-V amplitude are in evidence, but in addition to this there are *marked differences in amplitude* associated with the two breathing-sound conditions. Specifically, for all subjects, peak-V amplitudes are lower during Erratic-breathing blocks, compared with the values observed during Calm-breathing blocks. Block-to-block changes as large as .21 uV and .29 uV – three to four times the spontaneous fluctuations shown in the inset -- occur.

The range of individual differences in similar data for all twelve subjects indicated that for statistical testing of these changes, it would be advisable to convert raw values to z-scores, so as to more fairly assess Calm/Erratic differences in terms of the range of within-subject change in each participant.

Figure 3 presents a sample of such a z-score profile for one of the subjects from the main graph of Fig. 2; and Figure 4 provides an overview of the z-score values for all twelve participants for the first four blocks (C1, E1, C2, E2). As noted in the title of Fig. 4, a 2-tailed t-test on the z-score values, comparing Calm (C1, C2) with Erratic (E1, E2) blocks indicated a significant difference ($p < .00001$), specifically, a significant *decrease* in peak- V amplitude during presentation of the Erratic breathing sounds.

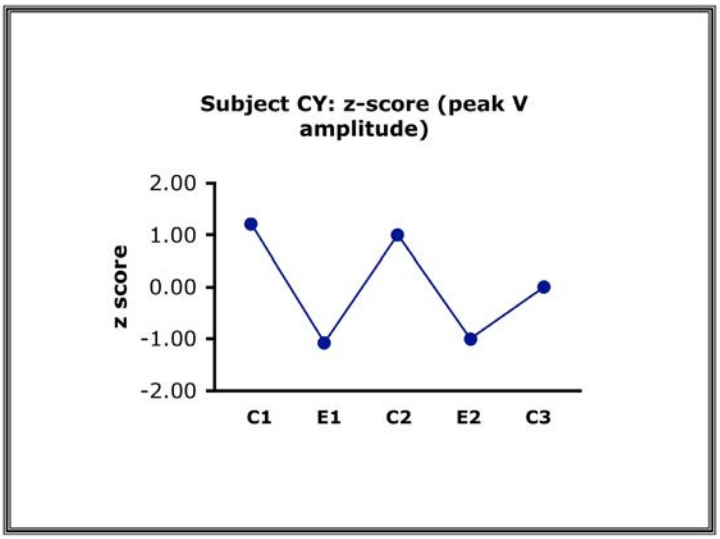

Fig. 3. Z-score profile for one subject from current study.

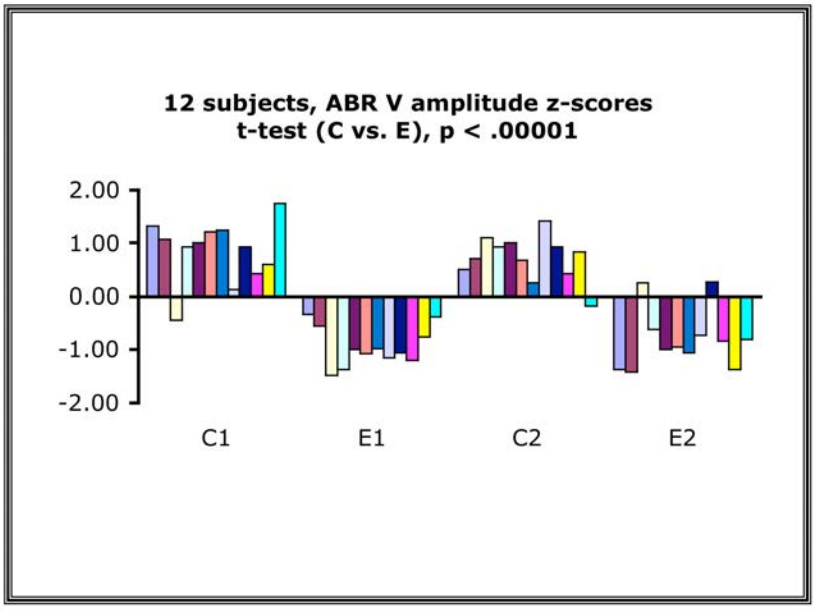

Fig. 4. Z-score values for all 12 subjects.

DISCUSSION

One possible explanation of this result is that it represents a peripheral masking effect, that is, the Erratic breathing sounds somehow interfered more with generation of responses to the condensation clicks in the inner ear and lower brainstem, than did the Calm breathing sounds.

Of course, adding any other sound to the ear at the same time as the ABR clicks does present the possibility of creating at least some interference. However, we do not believe that the significantly lower peak-V amplitudes during Erratic vs. Calm effect were only due to masking.

There are four reasons for this conclusion. First, neither the amplitudes (as suggested in Fig. 2) nor the latencies observed during either breathing condition differed significantly from values observed in other ABR series from our laboratory when clicks were presented at the same rate (43.1/sec) and level (60 dB nHL), and *no* additional sounds were present.

Second, we were careful to present the clicks and breathing sounds to each listener at levels that were pre-judged by that listener to be equivalent. Since perceptual equivalence presumably equates to physiological equivalence, this indicates to us that the effect of both sounds on the cochlea (and presumably the rest of the auditory system) was essentially the same when judged in terms of loudness – which should be a reasonably accurate perceptual estimate of "degree of masking."

Third, if it were only a case of masking, the significant *decrease* in peak-V amplitude should be accompanied by an *equally* significant *prolongation* of peak-V latency. The criterion of "equally significant' was not supported in these data. A 2-tailed t-test comparing raw latencies for the two conditions showed no significant difference ($p = .458$), in contrast with the result cited earlier for raw amplitudes ($p = .015$). The latency z-scores did in fact indicate a significant difference ($p = .001$) between Calm and Erratic blocks, but the size of the significance was not equivalent to the extremely clear result cited above for amplitude z-scores ($p = 2.8 \times 10^{-16}$). We believe the fact that peak-V amplitudes seemed to be affected more than latency is an indication that the change was due to a top-down (attention) rather than a bottom-up (interference) effect. If so, these results suggest that ABR peak-V amplitude may be more manipulable via attentional suppression than is latency.

Fourth, if the observed results for peak-V amplitude were due to physiological masking, one would expect that similar if not larger significant differences should be seen at lower levels (e.g., ABR peaks I and III). However, this was not the case. There were *no significant differences* observed under the two conditions in peak amplitude for either peak I or III. Related to this, it is notable that the amplitude of the peak-I response of the single male participant (the one who showed the largest effect at peak V) actually *changed in the opposite direction* during Erratic breathing – that is, his peak-I amplitudes were *larger* during Erratic blocks than during Calm blocks.

In conclusion, we believe that these results:1) provide new evidence of the effect of selective attention on the ABR; 2) suggest the potential for using auditory stressors to study the central physiology of emotional responses in humans; and 3) offer clues to physiological correlates of the effects of certain video games known to evoke panic attacks in susceptible players.

The findings are definitely encouraging with regard to future research on the physiological effects of individual components of video games, whether sensory, motor, emotional, or cognitive. Specifically with regard to the stimuli used here, we are designing a series of AXS-Battery experiments to examine the effects of these two types of sounds on several other aspects of physiology, including: otoacoustic emissions, eye-movement coordination, heart rate, blood pressure, respiratory rate, voice production, ear differences in ABR recordings, and power asymmetries in auditory cortex.

In addition to the outcomes regarding the particular sounds used here, we expect that the methods used in these projects will provide guidelines for an even wider examination of the effects of games, extending to other sensory stimuli (auditory, visual, somatosensory), motor activation, emotional components, and cognitive demands. Knowing more about exactly how violent video games affect human physiology may help us make more informed decisions regarding the nature of the risk they may present to the mental and physical health of players.

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