Commentary

Psychopathology Applications of Event Perception Basic Research: Anticipating the Road Ahead using Posttraumatic Stress Disorder as an Example☆

Andrew M. Sherrill*
Emory University, United States

Joseph P. Magliano
Northern Illinois University, United States

Richmond, Gold, and Zacks (2017) provide a compelling argument to explore clinical applications of theories and methods used in basic research on event perception, particularly with respect to event segmentation. As the authors state, the ambitious goal of transporting strategies from the laboratory to the clinic will not be an "easy road." Their contribution is a first mile-marker of many that must precede the actual dissemination of segmentation-informed clinical tools.

Researchers have argued that event segmentation is relevant to functional outcomes for a range of clinical conditions including schizophrenia (Richmond et al., 2017), obsessive-compulsive disorder (OCD; Zacks & Sargent, 2010), traumatic brain injury (Zacks, Kurby, Landazabal, Krueger, & Grafman, 2016), and posttraumatic stress disorder (PTSD; Eisenberg, Sargent, & Zacks, 2016). Within the domain of psychopathology, PTSD is of particular interest (Eisenberg et al., 2016; Sherrill, Lilly, & Magliano, 2016). Richmond et al. (2017) address wide-ranging translational issues regarding applications of event segmentation to clinical contexts and thus do not provide a level of detail required for specific clinical applications. This commentary uses PTSD as an example to specify important research questions that need to be addressed along the road toward applications in the domain of psychopathology, particularly in the areas of etiology, symptomatology, assessment, and intervention.

Posttraumatic Stress Disorder

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–5*) defines PTSD as a constellation of the following symptoms presenting for at least one month after exposure to a traumatic event: (a) re-experiencing trauma memories and trauma reactions, (b) avoidance of trauma-related memories and trauma reminders, (c) alterations in mood and cognitions such as a disorganized and incomplete trauma memory, and (d) hyperarousal and hypervigilance (APA; American Psychiatric Association, 2013). In the United States, approximately 90% of adults experience at least one *DSM–5*-defined traumatic event during their lifetime, yet only about 8% of adults will ever meet diagnostic criteria for PTSD (Kilpatrick et al., 2013). Given the considerable difference in rates between trauma exposure and PTSD diagnoses, mediating mechanisms likely explain the link between experiencing trauma and developing psychopathology. Prominent theories argue that PTSD symptoms are causally related to maladaptive encoding, storage, and retrieval of the trauma memory (Brewin, Dalgleish, &

☆ Author Note.
* Correspondence concerning this article should be addressed to Andrew M. Sherrill, Emory University, School of Medicine, Department of Psychiatry and Behavioral Sciences, Veterans Program, 12 Executive Park Drive Northeast, Suite 300, Atlanta, GA 30329, United States. Contact: andrew.m.sherrill@emory.edu.

Joseph, 1996; Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989).

Etiology and Symptomatology Applications

Etiology

There has been considerable effort in assessing the causal role of memory encoding and retrieval processes in the development of PTSD (Brewin, 2011; Brewin, 2014). One perspective is that trauma encoding is affected negatively by an attentional bias toward perceptual information (e.g., salient details of the event) and away from conceptual information (e.g., event schemas; Brewin et al., 1996; Ehlers & Clark, 2000). One basis for this hypothesis is that high levels of stress affect one's ability to maintain attentional control (Eysenck, Derakshan, Santos, & Calvo, 2007). However, little evidence using moment-to-moment assessments of cognitive processes supports the assumption that peritraumatic distress disrupts the encoding stage of memory formation.

Sherrill et al. (2016) recently used the event segmentation task (Newtson, 1973) to explore (a) if segmentation is different for stress-inducing films than non-stress-inducing films and (b) if stress impacts the empirically established positive relationship between segmentation performance and memory (e.g., Sargent et al., 2013). In this study, undergraduate students without PTSD completed the event segmentation task on a non-stress-inducing film (making breakfast) and a stress-inducing film (violent sexual assault). It was anticipated that event segmentation would become less systematic during the stress-inducing film (i.e., low agreement scores) because stress impacts attentional systems (Eysenck et al., 2007) and any factor that negatively affects attentional engagement in an experience was expected to decrease the systematicity of segmentation. However, contrary to expectations, the stress-inducing film resulted in more systematic segmentation than the non-stress-inducing film. Further, self-reported anxiety and dissociation during the stress-inducing film positively predicted segmentation systematicity. And, most surprisingly, segmentation agreement scores during the stress-inducing film negatively predicted performance on a recognition task.

How do we explain these results? Event segmentation theory assumes that people perceive event boundaries when they detect unpredicted perceptual changes in the event (e.g., Zacks, Swallow, Vettel, & McAvoy, 2006). Under stress, viewers may have disengaged from processing the conceptual nature of the sexual assault scene and therefore engaged in shallow event processing. Such an encoding strategy could have increased the salience of perceptual changes, especially in the context of the segmentation task. This interpretation, in context with the negative correlation between segmentation and memory performance, is consistent with one PTSD etiological argument that trauma-memory disturbance is the result of a stress-enhanced processing of sensory impressions rather than the event's underlying meaning (i.e., "data-driven processing"; Ehlers & Clark, 2000). The assumed result of this encoding shift is a memory representation containing rich perceptual information that has a weak internal structure (i.e., disorganized and incoherent information) and is not adequately integrated into autobiographical memory, which makes it difficult for one to voluntarily search and retrieve information (e.g., Sündermann, Hauschildt, & Ehlers, 2013). This interpretation of the results of Sherrill et al. (2016) are tentative and should be considered with caution until further research is conducted.

Symptomatology

While understanding peritraumatic event perception may improve our understanding of how PTSD develops, another possibility to explore is that PTSD symptoms co-occur with, or even contribute to, deficits in everyday event perception. Eisenberg et al. (2016) argue that PTSD-related hypervigilance (i.e., sustained alertness for potential threats) may increase detection of unpredicted and non-threatening, yet meaningless, novel stimuli (e.g., hearing footsteps in an adjacent aisle while grocery shopping). They argue that distraction by meaningless perceptual changes reduces one's ability to identify meaningful event boundaries within the environment and, thus, interfere with comprehending the larger unfolding event. They suggest a link between PTSD symptoms and diminished segmentation may be rooted in hyperactivation in the dorsal anterior cingulate context (dACC), which has been linked to PTSD symptoms (Bryant et al., 2005; Felmingham et al., 2009) and cognitive processes that support segmentation (Cohen, Botvinick, & Carter, 2000). They posit PTSD or dACC hyperactivation could potentially affect several segmentation processes including making perceptual predictions, monitoring prediction errors, orienting, and updating memory.

Eisenberg et al. (2016) investigated the relationships between PTSD symptom severity, segmentation agreement scores using everyday event stimuli (e.g., gardening), and event memory of the same stimuli. Participants were recruited from a volunteer registry at a clinical setting, did not share any particular clinical complaint (e.g., a formal PTSD diagnosis), and were not selected based on trauma exposure (e.g., military veterans). Consistent with expectations, results indicated PTSD symptom severity had a negative relationship with segmentation performance and explained unique variance in segmentation even after controlling for general cognitive function. Further, hyperarousal and re-experiencing PTSD symptom clusters predicted unique variance in segmentation. Contrary to expectations, PTSD symptom severity did not explain unique variance in event memory.

The association between PTSD symptom severity and diminished segmentation provides impetus to explore a wide range of empirical questions. First, additional variables should be explored that could explain the current correlational data (Eisenberg et al., 2016). One possibility is that a vulnerability to develop PTSD is premorbid low segmentation-ability or factors contributing to low segmentation-ability such as trait dACC hyperactivation (Shin et al., 2009) or deficits in cognitive functioning (Brewin, 2011; Buckley, Blanchard, & Neill, 2000).

Second, segmentation abilities should be explored using a sample of adults diagnosed with PTSD and common