Art Therapy, an effective outreach intervention with traumatized children with suspected acquired brain injury

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Introduction

This paper reviews Posttraumatic Stress Disorder (PTSD) with a focus on its impact on children, highlighting the use of Art Therapy as a healing psychotherapeutic counseling method. A case study will demonstrate the use of Art Therapy with a child in whom a sudden event produced suspected Acquired Brain Injury (ABI), concurrently with PTSD symptomology.

It is common after physical injury for healthcare professionals to work on physical recovery, leaving secondary psychological issues including stress to be assessed later, or when symptomology is evident, rather than examining risk factors at the time.

PTSD symptomology can occur immediately after a sudden incident, hindering mind-body recovery. Emotional sequelae can be present long after resolution of physical sequelae, affecting the continuum of complete recovery. It is crucial to resolve emotional difficulties in order to recover completely. Seeing an individual in community outreach allows the traumatic experience to be processed within familiar, non-medical settings, and also acknowledges re-adjustment difficulties in these same settings.

Art Therapy allows the use of creative materials to describe feelings and situations. Exercises are goal-oriented, require no artistic skill, and help to process feelings and concepts without having to ‘say’ all the words. Art Therapy is ideal when sensory, verbal or cognitive abilities are affected because it does not rely on verbal output.

Perspectives on PTSD in children

Malchiodi (1998) explains:

... For traumatized children, such as those who have been abused or have witnessed violence, art is a way to express themselves when talking seems unsafe or when words are not available to describe their fears, anxieties and other feelings... through creation of art there is a natural experience of wholeness, and this... is emotionally healing (p. 136).

PTSD is a relatively new diagnosis for children, and research into effects and effective interventions is ongoing. Although the Diagnostic and Statistical Manual of Mental Disorders (DSM) acknowledged stress in its diagnosis of a gross stress reaction in its first edition in 1952, it was not until the DSM-III (1980) that the American Psychological Association (APA) coined the term “Posttraumatic Stress Disorder,” and in its revised third edition (1987) that psychiatric symptoms specific to children were added (Keppel-Benson & Ollendick, 1993, p. 29).

Current criteria for PTSD require the experience of an event be outside the range of usual experience. Quoting from the APA, Keppel-Benson and Ollendick (1993) state “events so severe that preceding psychopathology is not necessary to experience significant psychological sequelae” (p. 29). The event is so extreme that almost anyone, including a child, would experience significant psychopathology and subsequent problems in adjustment (Terr, 1979 in Eth & Pynoos, 1985).

Why did it take so long for the adult medical community to acknowledge that trauma affects children too? Keppel-Benson and Ollendick (1993) conclude, in examining this historical lapse, that the
noradrenergic system and the HPA axis, accounting for symptom patterns (Terr, 1996). She cites the work of Yehuda, Southwick, and Perry (1990), noting excitatory responses (symptoms like posttraumatic repetitions and startle responses) might come from stimulating effects of corticosteroids on the noradrenergic functions of the brain cells. Inhibitory responses (symptoms like numbing and avoidances) might come from the effects of corticosteroids on brain activities.

It appears the brain reacts on several levels during traumatic response. Memory, learning and emotive responses are affected: the limbic system (important for memory, learning and emotion); endocrine system (hormonal, responsible for metabolic responses, blood pressure); autonomic nervous system (automatic responses including gastrointestinal) and the immune system, with components the thymus, spleen, lymph nodes and skin (Jay, 1999).

The following information has been proposed as a sequential model of response to PTSD-stimulating events, with effects on the victim in this sequence (Jay, 1999): (a) terror alters the brain chemical system, producing a limbic system response that can be a permanent, i.e., heartbeat may always beat faster after a traumatic experience; (b) altered brain chemical function heightens (victim develops an extreme sensitivity to stressors and triggers of event); (c) stressors and triggers stimulate noradrenaline surges; (d) noradrenaline surges stimulate hyperreactive state; and (e) hyperreactive state promotes survival response, or fight/flight reaction (Jay, 1999).

Perry and Azad (2000) suggests individual adaptive responses during traumatic stress are heterogeneous, varying with the nature, duration, pattern of the stress along with disposition and environmental support.

Therefore, neural systems, reacting to stress by altering their neurochemical organization, will affect the behavior of children experiencing a stressor. According to Perry and Azad (2000), following trauma, children will experience some persisting emotional, behavioral, cognitive and physiological signs and symptoms related to temporary shifts in their homeostasis. In general, the longer the activation of the stress–response system, the more likely there will be change in the neural system, so that a clinical disorder can occur if the stress response does not return to pre-event homeostasis.

The American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (1994) lists the following criteria for PTSD, six of which must be met for diagnosis: (a) exposure to traumatic event (witnessed, confronted by, actual/threatened death/serious injury to self/others), response involved intense fear, helplessness or horror; (b) re-experiencing through recurrent recollections as images/thoughts, dreams; acting as if

### PTSD symptomology, risk factors and diagnosis

PTSD elicits many responses in children. O’Donahue (1992) notes common ones are depression, anxiety, fearfulness, sleep-eating disturbances and cognitions such as self-blame and feeling damaged.

Keppel-Benson and Ollendick (1993) state that a key variable in determining impact of on children is the subjective interpretation of the trauma. As a result, some are unaffected with no symptomology. Others develop only some, or full symptomology only in the acute phase, with diminution over time (p. 30).

Terr (1996) reviews environmental impact, stating this can mean that once traumatized, a child may be modified biologically. Organic-style visual hallucinations have been noted to occur while traumatic events are proceeding, implying an immediate release of neurotransmitters in the brain upon traumatic impact (Terr, 1979).

Terr (1996) notes three brain systems implicated in the biology of PTSD: (a) the internal opiate system; (b) the noradrenergic system; and (c) the hypothalamic-pituitary/adrenal system. She cites work by Pitman, Van der Kolk and Orr (1990), who postulate that brain endorphins might cause the psychic numbing observed in trauma. The internal opiate system hypothetically causes the victim to fail to respond, to avoid and shut off feelings. Also, when the pituitary releases adrenocorticotropic hormone (ACTH) in order to regulate the levels of cortisol in the body, B-endorphins are released in the brain, and may stimulate trauma behaviors.

Studies that review changes observable in childhood trauma from the catecholaminergic system influence, including the startle response indicate that although startle responses become muted as development progresses, a traumatized child can also regress (Ornitz & Pynoos, 1989). Terr (1996) notes a brain trauma action site is the hypothalamic–pituitary–adrenal (HPA) axis. Investigations of trauma biology suggest an ongoing changed relationship between the noradrenergic system and the HPA axis, accounting for symptom patterns (Terr, 1996). She cites the work of Yehuda, Southwick, and Perry (1990), noting excitatory responses (symptoms like posttraumatic repetitions and startle responses) might come from stimulating effects of corticosteroids on the noradrenergic functions of the brain cells. Inhibitory responses (symptoms like numbing and avoidances) might come from the effects of corticosteroids on brain activities.
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