ONE EYE INTEGRATION TECHNIQUES:
LIKELY MECHANISMS OF ACTION

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Introduction and Rationale

Many clinicians we have trained are excited by the results they get with these "One Eye Integration (OEI) Techniques". Some, however, continue to have difficulty learning and applying the tools because they cannot conceive of how, or why, these procedures *could be* effective. For this reason, I have initiated a *formal* research program to assess the mechanisms of action and effectiveness of the procedures. One of the first steps in that process has been to review relevant psychobiological research regarding:

(a) the neuropsychology of Posttraumatic Stress Disorder; and

(b) likely neuropsychological mechanisms of action for these techniques.

Neuropsychology of Posttraumatic Stress Disorder (PTSD)

There has been considerable research to date documenting neuropsychological concommitants of PTSD, and even of triggered flashbacks. Many of these findings involve observed differences in brain function between patients *with* PTSD and those with*out*.

<u>Lateralization of Hemispheric Function</u>

Before reviewing studies pertaining to lateralization of function in PTSD patients, it seems important to briefly review the normal tasks or functions of each brain hemisphere. Typically, the right hemisphere is more closely associated with amygdaloid (emotionally intense) arousal. It is particularly important for evaluating the emotional significance of incoming information and in regulating autonomic and hormonal responses to these incoming signals (van der Kolk, et. al., 1997). As Ramachandran (1998) notes, it is also more concerned with questions of "how" something happened (body image, spatial vision, intensity level, emotional valence, etc.) than with "what" happened (verbally mediated, linear, logical sequences of events).

Rauch et. al. (1996) measured changes in regional Cerebral Blood Flow (rCBF) in PTSD patients between resting (non-triggered) states, and states in which they were “triggered” by the reading of individualized scripts of previous traumas they had encountered. Script-driven scans revealed a marked lateralization of activity in the right hemisphere (limbic, paralimbic and visual cortex), along with decreases in rCBF in the left inferior frontal (Broca’s) area and the middle temporal cortex. Using EEG during recall of traumatic memories by PTSD patients, Schiffer and his associates (1997) also found a marked laterality in favour of the right hemisphere and concommitant diminished left hemispheric functioning.

Interestingly, when the same traumatized patients were given *neutral* memory tasks there was *enhanced* left cortical activity (patients in the control group showed no significant laterality during the recall of distressing memories). These results support the speculation that one of the roles of the left frontal lobe is to mediate or inhibit the activity of the right frontal lobe and right-sided limbic structures (particularly the amygdala). Schiffer and his colleagues suggested that the hemispheres function more automomously (i.e., in a less synchronized fashion) in patients with childhood abuse. The preponderance of right-sided brain findings during the activation of posttraumatic states is consistent with the literature supporting the preferential role of the right hemisphere in evaluating the emotional significance of incoming information. It is also consistent with the role of the right hemisphere in anxiety, panic and phobic disorders.

Reduced Activity in the Hippocampus

The hippocampus is thought to retain the “facts” of experiences (episodic memories), and seems to create cognitive “maps” to allow integration of new experiences with autobiographical information (van der Kolk, et. al., 1997). It appears that this brain structure is also responsible for placing memories in context, in terms of space (perceptual patterns) and time (“here & now” versus “then & there”). Proper functioning of the hippocampus is necessary for explicit or declarative memory (van der Kolk, 2001).

One of the most consistent psychobiological findings regarding PTSD is that there is decreased hippocampal volume (Stein, et. al., 1997). Activity in this area is decreased in response to stress-induced, corticotropin-releasing factor (CRF) production, which results in highly fragmented memories (van der Kolk, 1994). In other words, when an individual encounters an event that is sufficiently (a) negative, (b) unexpected, and (c) makes him or her feel overwhelmed, powerless and/or confused, the event gets “logged” into memory in present tense, at full intensity.

Van der Kolk (1994) noted that when distress interferes with “hippocampally mediated memory storage and categorization, a mental representation of the experience is probably laid down by means of a system that records affective experience but has no capacity for symbolic processing or placement in space and time" (p.261). The hippocampus is responsible for recognizing the significant difference between a lion at the zoo and one in your living room (Goleman, 1995).

These "sealed capsules" of experience can later be "triggered" in response to stimulation (through *any* of the senses) that reminds individuals of the original experiences. This "triggering" involves the secretion of epinephrine and norepinephrine which surges through the body. Amygdaloid arousal seems to imprint in memory the moments of highest emotional charge with an added degree of strength. The more intense the arousal, the stronger the imprint (LeDoux, 1986).

<u>Increased Activation of the Amygdala with Reduction of Activity in Broca's Area</u>

When PTSD patients are exposed to reminders of their traumas, the amygdala has been shown to be activated. This contributes to hyperaroused states which, when combined with simultaneous decrease in oxygen utilization in the inferior prefrontal cortex (Broca's area), results in "speechless terror" (van der Kolk, et. al., 2001). Broca's area is thought to be responsible for translating personal experiences into communicable language. If this process fails to occur, memories are stored in sensorimotor modalities, somatic sensations and visual images.

Studies by LeDoux (1986) have shown that the amygdala can act independently of the neocortex, such that emotional reactions and memories can be formed with no conscious cognitive participation. The amygdala retains the emotional flavour of memories, and seems to integrate multisensory information (sights, sounds, smells, tastes & proprioceptive or touch stimuli). The thalamus is the original reception site for multisensory stimulation, and it routes signals to both the amygdala and the neocortex.

Van der Kolk (2001) suggests that the failure of left hemisphere functioning during states of extreme arousal may be responsible for the derealization and depersonalization that acute PTSD patients report.

<u>Decreased Activity in the Anterior Cingulate Gyrus</u>

The anterior portion of the singulate gyrus "appears to be involved in conditioned emotional learning, assigning the emotional valence to internal & external stimuli and facilitating a more realistic differentiation between real and perceived threat" (Bergmann, 1998, p.2). This area helps individuals to distinguish between current dangers and traumatic reminders that are no longer relevant to current experiences. Since activation of this area of the brain is reduced during triggering of traumatic memories, PTSD patients experience hyperarousal in the face of benign stimuli (van der Kolk, et. al., 1997). The cingulate gyrus is thought to function as both an amplifier and filter, helping to integrate the emotional and cognitive components of the mind. Ruth Lanius and her associates used functional MRI to compare levels of brain activation in subjects with and without PTSD (Lanius, et. al., 2001). When comparing baseline levels to those associated with subjects' focusing on traumatic memories, it was evident that the control group showed greater activation than the PTSD group in the anterior cingulate gyrus, bilaterally. In other words, brain functioning in this area was reduced, even though heart rate was significantly *increased* for PTSD subjects.

Reduced Effectiveness of the Corpus Callosum

The corpus callosum is the band of nerve fibers connecting the hemispheres of the brain. It allows for the transfer of information by both hemispheres, integrating emotional and cognitive aspects of experience. This structure has been shown to be decreased in size in adults who experience the trauma of abuse as children (Teicher, et. al., 1997).

Additional Psychophysiological Correlates of PTSD

In addition to the above-referenced neuropsychological differences between PTSD patients and controls, other findings have been reported consistently in the literature. Shalev (1996) noted dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. This was suggested by results of neuroendocrine studies in which it was found that PTSD patients had increased dexamethasone suppression, elevated urinary catecholamines and an increased norepinephrine/ cortisol ratio. Marinko and his associates echoed this finding (Marinko, et. al., 2001).

Shalev also reported higher rises in heart rates, increased skin conductance and increased electromyographic responsiveness of PSTD patients compared to non-PTSD subjects when presented with loud noises, cues associated with specific traumas (visual or auditory), and narrative scripts of the unique traumas suffered by specific patients. In addition to higher levels of initial arousal, PTSD subjects' responses to stimuli extinguished less readily and over-generalized to neutral stimuli more easily than was the case with control subjects. He does note, however, that with appropriate psychotherapeutic treatment these elevated response patterns were significantly reduced.

The parietal lobes are thought to integrate information from different cortical association areas. These lobes and the temperoparietal regions of the brain have also been implicated in the spectrum of PTSD symptoms (van der Kolk, et. al., 1997; van der Kolk, 2001). A fascinating phenomenon in the parietal lobes was noted by Ramachandran (1998). Although he is not specifically speaking of PTSD patients, he described a small region of the right hemisphere that receives information from the right parietal lobe. The information pertains to the recognition of discrepancies between what appears to be happening and what is felt to be happening. By stimulating the left ear with an irrigation of cold water, Dr. Ramachandran was able to activate that center for recognition of discrepancies. As a result, stroke patients who had previously been denying paralysis in parts of their bodies suddenly recognized and reported these impairments. Since one of the most puzzling symptoms of PTSD is lack of ability to recognize the discrepancy between present experience and past trauma, the re-activation of this "discrepancy identification" center may be relevant for PTSD treatment.

Further evidence of relative abnormalities in the parietal and temporoparietal regions was a recent study of torture survivors. Single photon emission computed tomography (SPECT) technology was used to assess differences in regional cerebral blood flow between PTSD patients and control subjects (Mirzael, et. al., 2001). They assessed levels of hemispheric heterogeneity in nine regions of the brain in each of their subjects, and found that regional blood flow was markedly more heterogeneous in patients suffering from PTSD. This lateral variability was most pronounced in the parietal lobes and temporoparietal region in general. Level of asymmetry was found to be associated with severity and duration of torture (more severe and prolonged abuse was correlated with greater variability).

In a study using Transcranial Doppler Sonography, 62 percent of PTSD patients had signs of vasospasm (excessive mean speed of blood circulation) in an area of the brain known as "Willis's circle" (and in vertibrobasilar flow in general). Twenty-two percent of control subjects evidenced this same pattern (Marinko, et. al., 2001). Six months later, after psychiatric treatment, 22 percent of PTSD subjects still showed signs of vasospasm. At the same six-month post-test, 10 percent of untreated control subjects evidenced vasospasms.

Likely Neuropsychological Mechanisms of Action for OEI Techniques

The One Eye Integration (OEI) Techniques will not be described here. Rather, readers are referred to the manual for clinicians to review detailed explanations, photographs, case examples and flow charts (Cook & Bradshaw, 2002). As mentioned in the Introduction of this paper, both Audrey Cook and I had completed training in EMDR and had been using it a number of years before Audrey innovated most of the One Eye techniques. At the time we were releasing the first edition of our book in 1999, we did a brief Internet search for other similar trauma treatment techniques using one eye at a time. A Harvard University psychiatrist by the name of Fredric Schiffer had just released his new book on dual-brain theory and therapy (Schiffer, 1998). Although the techniques he was using involved alternately covering the eyes while thinking of disturbing events, people or affective states, it was clear that he had taken a different approach to the task.

Schiffer identified associations between the eyes and the hemispheres of the brain. Specifically, he found that the intensity of dysphoric affective states (fear, sadness, shame, anger or anxiety) differed significantly depending on which eye was blocked from light and which one was exposed (technically, he has clients cover all except the outer third of the retinal shield, using modified glasses). He posits that the hemispheres have considerable independence of function, and that the impact of this independence upon intensity of mood can be reduced through alternating which eye is exposed to light. Recently this lateralization has proven helpful in predicting which depressed subjects will benefit from the application of Transcranial Magnetic Stimulation to one of the hemispheres (Shiffer, et. al., 2002). Interestingly, almost half of the subjects in that study (20 out of 37) had greater improvement in mood while looking out of the right lateral visual field than the left. This clearly indicates that a considerable number of individuals are "reversed" from what would be expected in terms of affective intensity associated with hemispheric stimulation. In clinical work I have seen this "reversal" of hemispheric response pattern in 10 to 15 percent of my clients.

Neither Audrey nor I believe that the One Eye Integration techniques are simply a variation of EMDR. For one thing, EMDR can be performed effectively without the use of the eyes (applying alternating hand taps or audio tones), whereas at this time our clients must be capable of perceiving light and tracking objects across the visual fields of both eyes in order to benefit from OEI. This does not mean, however, that the mechanisms of action for these two sets of psychotherapeutic procedures have nothing in common. They both produce a constant shifting in the brain by alternating stimulation of one set of hemispherically related structures with stimulation of structures on the other side.

Stickgold (2002) notes that the primary mechanism of EMDR is likely:

> …a pattern of alternating, bilateral stimulation that forces subjects to shift their attention across the midline. It is this orienting response that we propose induces a REM-like state, facilitating cortical integration of traumatic memories…This reorienting of attention requires first a release of focus from one location in visual space, then its shift to a new location, and finally its refocusing on this new location (pp. 70-71).

He was referring to such stimulation in EMDR, but it also occurs in OEI therapy (particularly in the rapid "switch" and "sweep" techniques). He suggests that such repetitive reorientation of attention from one location to another produces shifts in regional brain activation and neuromodulation similar to REM sleep. Among the regions involved in that process are the anterior cingulate (discussed earlier, relative to assessment of threat), and the superior colliculus (which controls eye movement) that is activated by the pontogeniculooccipital (PGO) waves of REM sleep. He notes that this REM-like state "permits the integration of traumatic memories into associative cortical networks without interference from hippocampally mediated episodic recall" (p. 71). Further, he states that alternating stimulation therapy such as EMDR can work even better than REM sleep because norepinephrine arousal can be contained through careful application of the therapy. This is even more possible with OEI than with EMDR and was, in fact, the main reason for the development of the OEI techniques.

Levels of anxiety and fear can be kept low enough during processing to prevent full reactivation of posttraumatic states and dissociation, but engaged enough to produce the desired physiological and neurochemical shifts.

In support of that hypothesis, Levin and his associates saw increased activation of the anterior cingulate and left frontal cortex following the alternating stimulation of EMDR (Levin, et. al., 1999). As mentioned in previous sections of this paper, the effect of increased activation in the anterior cingulate will be more accurate appraisal of the present danger of incoming stimuli. Since clients are usually in safe, relatively quiet psychotherapy sessions when this is occurring, levels of limbic activation can be reduced as the anterior cingulate confirms the safety of the current environment. With increased activity in the left frontal cortex, most subjects will have a greater capacity for semantic memory processing and communication of their traumatic experiences to others (rather than the fragmented, inexpressible flashbacks of PTSD).

Nicosia (1994) posits that the phase relationship of the hemispheres is disrupted by the failure of norepinephrine suppression during exposure to trauma(s). Huge amounts of norepinephrine are released in the forebrain during trauma which, he theorizes, causes a localized decoupling of one area of the cortex from the pacemaker. This asynchrony, in turn, prevents integrative memory processing. He suggests that alternating stimulation (across the midline) mimics the activity of the pacemaker mechanism which was suppressed. In his QEEG study, a PTSD client started with an interhemispheric hypocoherence that was 5 standard deviations below what would be expected of non-PTSD individuals. After only 90 minutes of EMDR processing (alternating stimulation) the asynchrony was completely resolved, to normal levels.

In the previous section of this paper, it was mentioned that in PTSD patients who are re-triggered with trauma scripts, at least two brain structures were reduced in activation: The anterior cingulate gyrus (bilaterally), and the left prefrontal cortex (Broca’s area). Van der Kolk and his associates applied three sessions of alternating stimulation (via EMDR) to PTSD subjects in the study (van der Kolk, et. al., 1997). Using SPECT scans, they demonstrated increased activity in the anterior cingulate bilaterally and in the right prefrontal cortex. The latter is involved in problem solving, learning, and complex stimulus discrimination. Since both of these areas *increased* in activation, while PTSD symptom scores *fell* in half, the researchers suggested that recovery from PTSD may depend on the capacity of higher brain functions to override input from limbic structures (such as the amygdala and hippocampus). As mentioned by van der Kolk (2001), the anterior cingulate gyrus is also considered to be an amplifier and filter that helps integrate emotional and cognitive components of the mind. When activation in this area is increased, integrative processing of traumas is facilitated.

Both Bergmann (1998) and Servan-Schreiber (2000) have proposed models based on enhanced interhemispheric communication and synchronous neuronal firing patterns. Bergman's model is similar to that proposed by Stickgold (2002): First, the alternating stimulation brings about a constant alternating shifting of attention, which facilitates a constant cholinergic surge. That surge activates the REM sleep system, which activates areas of the anterior cingulate, facilitating the integration of traumatic memory into general semantic networks. He notes that bilateral activation of the anterior cingulate cortex facilitates a more realistic differentiation between real and perceived threat, and a concommitant reduction in hypervigilence. This leads to a reduction in intensity of both hippocampally mediated episodic memories and amygdaloid mediated negative affect. Bergmann also notes that the cerebellum is stimulated by the alternating stimulation. It constitutes another association area and information processing centre, allowing information exchange with the limbic area and directly activating areas in the dorsolateral and orbitofrontal cortices. Stimulation of these areas further facilitates the integration of traumatic memories into general semantic and other neocortical networks.

Dr. Carla Hannaford read the psychotherapist training manual for OEI (Cook & Bradshaw, 2002). She responded with an electronic mail message to Audrey:

> …the results are very profound from such a simple process. The eyes are tied directly into the vestibular system and also to all the muscles of the body via the vestibular system. So as we move our bodies and eyes together…there is a vestibular integration as well as a hemispheric integration in the areas of the brain that have to do with movement…the cerebellum, substantia niger, basal ganglion, and motor cortex of the frontal lobe…(The cerebellum) ties directly into the amygdala, so is affected by emotions and emotional events in our lives. The cerebellum helps to modulate our emotions, therefore movement helps to modulate our emotions via the cerebellum. The vestibular system ties directly into the cerebellum and the eyes----again the connection (Hannaford, 2001).

Carla refers specifically to page 90 in her book, for further information and a diagram illustrating the areas of the brain that have to do with movement (Hannaford, 1995).

Having clients focus on somatic sensations and affect likely targets dissociated mental representations of experiences that are not symbolically processed or placed in space and time. Switching the coverage & exposure of the eyes likely activates (stimulates) the cells in the left prefrontal lobe of the Neocortex. In other words, the process of Switching facilitates an integration of Neocortical and Amygdaloid activity. Continued and/or rapid Switching probably allows for the downregulation of the sensitized pontine and limbic areas, facilitating integration of higher cortical functioning. Once Noradrenergic firing is diminished, clients can generate more adaptive explanations of their traumatic experiences.

One unusual phenomenon that occurs during OEI processing of traumatic memories that doesn't seem to happen during EMDR is what we have come to call "dissociative (or neurological) artifacts". These include various physical and sensory dissociative symptoms as headaches, drowsiness, light-headed feelings and visual distortions. I was interested to see that van der Kolk (2001) observed a similar phenomenon with PTSD clients as they re-activated traumatic memories:

> We found that two decades after the original trauma, people with PTSD developed opioid mediated analgesia in response to a stimulus resembling traumatic stressor, which we correlated with a secretion of endogenous opioids equivalent to 8 mg of morphine. Self-reports of emotional responses suggested that endogenous opiods were responsible for a relative blunting of the emotional response to the traumatic stimulus (p. S53).

With OEI therapy, in most cases we find that continued switching of eye coverage and exposure results the dissolution of such symptoms, but the actual mechanism of this resolution remains to be further investigated with our QEEG studies.

Finally, it is worth noting that many clients who have successfully integrated traumatic memories with OEI express very spontaneous smiles, or laughter. Interestingly, Ramachandran (1998) notes that the source of such "spontaneous" (natural) smiles is the basal ganglia; whereas intentional smiles (e.g., photo opportunities) are generated by the neocortex. For this reason, the basal ganglia will be another area of the brain for further exploration regarding the integration of traumatic memories. Research aside, however, isn't that the kind of relief we're seeking for those who are brokenhearted and crushed in spirit?

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