

***Commentary on Using Client-Centered Psychotherapy Embedded Within
A Pluralistic Integrative Approach to Help a Client With Executive
Dysfunction: The Case of "Judith"***

The Case of "Judith": A Neuropsychologist's Perspective

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ABSTRACT

Executive Functioning (EF) deficits may be the most catastrophic form of brain injury, as they alter one's personality and sense of self while impeding the ability to make use of rehabilitation, compensatory strategies, psychotherapy and social support. The injury itself can produce a loss of appreciation for the deficits, as well as depression, apathy and a lack of empathy. Although EF deficits can be nuanced, individuals with EF deficits are often presumed incapable of benefiting from higher order interventions. Ward and Hogan's (2015) case study of Judith effectively contradicts this assumption. While this case does not offer traditional neuropsychological assessment results, it highlights how the value of such measures rests in their ecological validity. The years of psychotherapy and intervention that Judith underwent offer ample ecological data and an opportunity to comment on the neurobiological aspects of Judith's injury and recovery beyond the assessment setting.

Ke words: TBI; brain injury; social neuroscience; endocrine; intervention; psychology; neuropsychology; clinical case study; case study

While brain injuries are inherently debilitating, intervention strategies and accommodations are most effective when patients can appreciate their strengths and limits, are capable of anticipating their need for assistance, and are able to recognize when to use a compensatory tool or technique, such as the "Plan-Do-Review" (Ylvisaker and Feeney, 2002). Classically, Executive Functioning (EF) deficits are defined by the lack of these qualities, along with impulsivity, poor risk perception, poor error monitoring, inflexibility, social "stickiness," apathy, delay-of-gratification difficulties, emotional volatility, and failure to appreciate social cues. These individuals are well described by the saying, "The problem with bad judgment is that you lack the judgment to know you have bad judgment." The EF deficit also places a significant burden on the family, friends and medical providers of the afflicted individual, often while the patient remains oblivious to these transferred hardships. It is rare that all of these impairments co-exist in the same individual, unless they have severe brain injury, such as was the case of Judith described by Ward and Hogan (2015).

Judith's case involved transient failures of insight—e.g., thinking she had attended to her paperwork, only to find that she had made a trail of papers—accompanied by an appreciation of her limitations and the implications of such, but not always at the same time. It was reported that Judith would burst into tears upon realizing how little she had accomplished but not until she had spent a day's worth of effort laying papers out on her floor. In clinical practice, EF deficits often present in this seemingly chaotic fashion, but there is logic to them. Specific EF deficits correspond to specific anatomical regions and circuits, and often manifest symptoms with identifiable nuances. These deficits can be aggravated by social context, and frequently present as part of a constellation of medical and psychological complaints (for neurobiological reasons). Understanding these factors offers another perspective on Judith's challenges, as well as how and why her therapy was successful.

UNDERSTANDING EXECUTIVE FUNCTIONING (EF) FROM A NEUROPSYCHOLOGICAL PERSPECTIVE

Lezak et al. (2004) described EF as the ability to adapt when confronted with novel or unfamiliar situations and tasks. It is not a constant from the perspective of an outside observer. Once a routine is established, there is no need to develop a new strategy; therefore, impairments may not be evident when performing well-learned tasks. Inconsistent attention and performance are hallmarks, although this profile creates the illusion that the person with deficits is choosing to perform in a substandard fashion on some occasions (which often becomes a source of acrimony).

Classically, EF is understood to have four components: volition or motivation, planning, initiation and maintenance of purposeful action, and effective performance (Lezak et al., 2004). These components often have known neuroanatomical correlates and neuropsychological indicators that are useful in making distinctions. Each problem also requires slightly different interventions because they cause different outcomes. The recent expansion of the classical model of brain functioning has included the union of emotion and reason (Bechara et al., 2000, 2005). Judgment can be impaired by the loss of "gut instinct," and the underlying mechanism involves severing of the orbitofrontal cortex circuits connecting logical reasoning with the physiological signs of emotion, such as sweaty palms or a lump in the throat.

Feelings are one of the most vital components of complex decision-making. Normally, bad decisions trigger the warning of unpleasant emotions long before we can articulate why a decision is poor (Bechara et al., 2005). Without this warning system, error monitoring is slow and relies on a conscious decision and follow-through to review one's approach to solving a problem. Inefficient error monitoring was one of the hallmarks of Judith's case, exemplified by the paperwork handling failure described above, as was her need to use external supports and systems to prevent mistakes with her mail. Judith also combined insight and intrapersonal curiosity with social blindness, which interfered with making good use of her therapeutic hour (e.g., repeating herself and being longwinded).

Damage to the orbitofrontal cortex circuits is implicated in the social awkwardness that may accompany EF deficits. Empathy requires that we feel another's emotional state by feeling

the resonance of that emotion in our own body; for example, we feel another's embarrassment because our own face flushes (Cozolino, 2014; Grattan et al., 1994). Chillingly, it is important to realize that without the inclusion of this aspect of judgment, a motivated, well organized and effective serial killer would meet the classic standard for good EF.

JUDITH'S INJURIES, STRENGTHS AND CHALLENGES

For an individual with EF deficits, the threat of changing homes, habits and daily routines is exceptionally challenging. Ward and Hogan's intervention with Judith was a noteworthy accomplishment because Judith was able to manage these transitions successfully, despite a history of suicide attempts, reliance on others, avoidance and self-isolation.

A neuropsychologist like myself would have found the findings on neuroimaging, neurological and neuropsychological examinations, and the medical records related to Judith's traumatic brain injury (TBI) advantageous for augmenting her psychosocial history and helpful in characterizing the structural and functional parameters of her injury. Those findings and records might also have helped to identify other comorbid factors that could have been contributing to her psychological symptoms (e.g., anemia, deficiencies in B12, hypothyroidism, and/or medication effects). The brain-behavior relationship training that neuropsychologists complete teaches them to query patients about information that other psychologists may find irrelevant (e.g., handedness), as such information can provide insights into functional brain specializations and/or response to intervention.

Brain injury produced by a fall is typically the product of focal damage, although there can be damage from centrifugal force as the person rotates through the air and the motion stops abruptly upon impact. Access to the additional neuromedical data described above might have clarified this etiology in Judith's case and helped to illuminate the basis for her symptomology. Although there is much to extrapolate from the ecological data that is richly documented in Judith's case study, the lack of neuromedical information, for example, precludes my ability to fully agree with the etiology of fall-induced TBI proposed for Judith's EF deficits. A particular concern in Judith's case is that an inability to recognize familiar individuals is a highly atypical symptom of a TBI, while difficulty retrieving names and words on demand is common. Ward and Hogan clearly state that Judith experienced difficulty in recognizing neighbors, and from my neuropsychological perspective, this distinction might well lead not only the diagnosis but also the intervention down different clinical paths.

Acquired face blindness is almost always produced by a stroke and requires a relatively complete destruction of the right fusiform face area, which is located on the underside of the temporal lobe (Busigny et al., 2010). Face blindness produced by trauma is exceptionally rare, particularly from a TBI with a brief loss of consciousness. In individuals with acquired prosopagnosia from right temporal lobe injury, the condition is also accompanied by severe behavioral disturbance, such as disinhibition and aggression (Chan et al., 2009). Further, the damage producing face blindness does not correspond to the brain regions and circuits associated with EF. Inability to recognize familiar faces after a TBI may be the product of a somatoform or dissociative disorder, which can co-exist with a TBI.

Unfortunately, the suggestion of a somatoform disorder is sometimes interpreted as an implication that the patient's symptoms are of lesser importance or validity, which in Judith's case would be incorrect. Impairment from a somatoform disorder increases with the addition of anxiety and depression symptoms (De Waal et al., 2004). Somatoform disorders are often a response to significant stress, such as trauma, profound dislocation, the threat of losing a key relationship, or a sense of personal failure with no evident way to remedy it. All of these risk factors are documented in Judith's case history. Should the face blindness reflect a somatoform component, the distress remains legitimate, acute, and worthy of respect.

Psychological pain induced by a social problem, such as social rejection, produces the same activation pattern in the brain as physical pain (Eisenberger et al., 2003). A TBI is a psychological and social injury, as well as a biological one, and TBIs rarely exist without disturbances in all spheres. Similarly, acute stress has been shown to produce transient, functional EF deficits in neurologically intact individuals (Keinan, 1987). Increases in the level of stress are accompanied by diminished quality of thinking; moreover, this phenomenon occurs regardless of whether the distress is from psychological, social or physical causes. Disinhibition, poor planning, impulsiveness and unproductive repetition are also characteristics of maladaptive responses to stress. The combination of Judith's injuries and the degree of psychosocial stress would be expected to aggravate her cognitive impairments.

WHEN A TRAUMATIC BRAIN INJURY (TBI) COLLIDES WITH LIFE

A TBI is not an event as much as an unspooling of a series of intertwined biological, social and psychological changes. While the physical impact of a fall may produce damage to and death of neurons, it also launches a series of neurological changes with measureable effects that can persist up to months and years after the fall event. Injured neurons become less able to produce sufficient energy to maintain their structural integrity and often die in the months subsequent to the fall event (Giza and Hovda, 2001). This energy-deficit state has been documented across all forms of acquired brain injury, including simple concussion (Giza & Hovda, 2001). The neurons die through a form of cellular suicide (apoptosis) that can be triggered by oxidative stress signals from the endocrine, neurotransmitter and enzyme systems. Moreover, there is a limited window of time in which injured neurons survive or die (Liou et al., 2003).

Relevant to Judith's case, apoptosis can also be induced through "psychological" and social processes, such as pain, loss of control, social isolation, fear, depression, loneliness, posttraumatic stress disorder, and membership in a stigmatized group (Eisenberger et al., 2003; Cacioppo et al., 2002; Cole, 2009, 2010; Copeland et al., 2012; O'Donovan et al., 2012; Sapolsky, 1997; Webb et al., 2014). These risk factors and their shared biomarkers have been documented across human cultures and among primates (Platt et al., 1999; Smith & Elwood, 2004). Judith's TBI contributed to a personal and social disruption. In particular, her marriage was characterized by dependence, fear, an external locus of control, depression, and finally panic attacks. Her attempt to avoid bothering her husband by her crying when he returned home in the middle of the night following a sexual liaison with another woman is disturbing on many levels,

and suggests that she had been living in a marriage that forced her to live a life that was emotionally incongruent and full of double-binds.

Ward and Hogan's reporting of Judith's descriptions of her household, and her responses to it, seems consistent with repeated exposure to verbal abuse while being isolated and psychologically dominated by an individual on whom she depends (Herman, 1992). Even without the added misery of verbal abuse, the subjective experience of loneliness may be one of the most well researched psychological risk factors for ill health and depression (Cacioppo et al., 2002; Cole et al., 1996, Cole et al., 2007).

When Judith was injured and her social context changed, she accumulated several social risk factors supportive of further neuronal and endocrine injury while losing some known protective factors (e.g., social stability and high social rank). Judith left a professional community in which she had been a member for a decade; upon ceasing to be a professional, she lost some of her financial autonomy, prestige and sense of control that accompany a professional position. When her therapist addressed these social and psychological issues, he was at the same time addressing pathological neuronal processes.

Distress can interfere with neuronal health directly through alterations in the neuroendocrine system (Masel et al., 2010; Rothman et al., 2007), but distress can also interfere with physical health and sleep. The increased stress burden can diminish the resources a person can bring to problem solving. As such, Judith would be more prone to making poor judgments and absentminded errors, further increasing her stress. In this vicious cycle the source of each stress represents a potential intervention point, by which one may improve the quality of decision making, social support, coping mechanism, attributions, and interpretations to return the person to a healthier equilibrium (Uchino et al., 1996).

In about one-half of the TBI cases studied by Popovic et al. (2004), the TBI was found to be accompanied by mild to moderate levels of clinical depression, and typically presented within a constellation of symptoms. Depression appears to be both a cause and a consequence of distress. At the time Judith's case report was written, there were 192 peer-reviewed articles in the publicly available literature documenting the role of stress and the associated endocrine changes as a cause of depression (Irwin & Cole, 2011; Mills et al., 2013). In short, these collective studies show that the experiences we perceive, and our interpretation of them, change us, and that the psychological is, inevitably, biological.

High levels of circulating stress hormones (glucocorticoids) compromise neurons in the regions of the brain rich in glucocorticoid receptors; this pathological mechanism involves induction of cell death (apoptosis), decreasing the connectivity within the neuron (dendritic branching) and altering synapses (Bremner, 1999). Exposure to circulating stress hormones at high levels and/or for extended periods damages the areas of the brain richest in stress hormone receptors (e.g., amygdala, hippocampus, prefrontal cortex, and cerebellum), as well as the brain regions responsible for inhibiting the stress response (e.g., the amygdala). This process extends the stress damage to those structures that would have otherwise turned off the stress response, thereby creating a biological vicious cycle (Cole, 2009; Herman & Cullinan, 1997). (Note that

the glucocorticoid system is complex, including at least two receptor subtypes. There are also receptors on neurons and on glia. For a comprehensive review, see de Kloet, Joels, & Holsboer, [2005].)

PSYCHOTHERAPY AS A BIOLOGICAL INTERVENTION

Research by Sapolsky and colleagues indicates that experiencing change in one's position in the social hierarchy affects levels of circulating stress hormones; specifically, rising in the social hierarchy is associated with a decrease in circulating stress hormones and *vice versa* (Sapolsky, Albert, & Altmann, 1997). This phenomenon has been observed across species. Much of the early research relied on *in vivo* monitoring of olive baboons (Sapolsky et al., 1997), with later studies replicating the findings in humans (Abbott et al., 2003, Sapolsky, 2004). Like other primates, we are a communal species. Injuries that damage the ability to form and maintain social ties and social status, therefore, increase our own levels of stress, both from psychological and physiological perspectives. Stigma, particularly when it is internalized, adds a second layer of risk (Cole, 2010; Eisenberger et al., 2003).

Ultimately, a brain injury produces damage to the organ responsible for interpreting and responding to complex social cues, for inhibiting impulses, for monitoring self-presentation, and for adapting our behaviors to novel situations. These socially unappealing qualities have been noted in other classic works on TBI (Lezak, 2004), as well as in the case study of Judith by Ward and Hogan. The biological benefits of Rogerian therapy is supported by the research findings published in the collective neuroscience literature. In particular, studies have demonstrated that attending a weekly peer support group enhanced the viability of individual cells (e.g., lengthening of telomeres), improved recovery of brain tissue (plasticity), and improved immune functioning (Chen et al., 2010; Cole et al., 2010; Miller et al., 2008, 2009; Okereke, et al., 2012).

Anotoni et al. (2012) studied the impact of a 10-week cognitive-behavioral stress management intervention with early-stage breast cancer patients and found that the intervention reversed anxiety-related upregulation of pro-inflammatory gene expression in circulating leukocytes. They concluded that "these findings clarify the molecular signaling pathways by which behavioral interventions can influence physical health and alter peripheral inflammatory processes that may reciprocally affect brain affective and cognitive processes" (p. 366). In short, Anotoni et al.'s findings demonstrate how psychological change is intertwined with physical change.

It seems clear that Judith benefitted from individualized psychotherapy that was conducted while her therapist monitored indicators of improvement and modified his approach to match Judith's unique pattern of strengths and weaknesses. The outcomes of this type of intervention strategy should match or exceed the benefits noted in the type of peer support group studies mentioned above, particularly since the psychotherapy intervention also included teaching Judith new methods for circumventing some of her deficits.

Learning can produce larger morphological changes, as evidenced by increases in hippocampal volume corresponding to use (Maguire et al., 2000). Placing demands on the

hippocampus through spatial challenge exercises has been shown to increase cellular survival, as well as the density and size of the hippocampal nucleus. In these exercises, stem cells integrated into the existing cell matrix, improving both the structure and function of the hippocampus upon use (Song, Stevens, & Gage, 2002). Interestingly, TBI appears to facilitate certain types of learning, particularly fear learning, as well as the activity level of brain regions that monitor for threat cues (Reger, et al., 2012). Given the energy-deficient state that follows a TBI, the challenge to clinical intervention is gauging the right proportions of neurocognitive demand (for enhancing learning) to rest (for enhancing recovery) (Langlois, Rutland-Brown, & Thomas, 2004; Masters et al., 2012).

OTHER POTENTIAL AVENUES OF INTERVENTION

Judith's case study did not mention any trials of pharmacological agents as adjuncts to therapy, although there are several classes of medication that might directly or indirectly improve her EF. When used with skill and delicacy, pharmacotherapy may provide the necessary neurobiological scaffolding, potentially normalizing specific neurocircuitry and enhancing the benefits of psychotherapy. As discussed above, certain brain regions are more vulnerable to injury. In many cases, specific symptoms are associated with specific circuits and neurotransmitters. There are currently no U.S. Federal Drug Administration (FDA) approved medications for use with EF deficits or neurobehavioral impairments post-TBI. However, it is still possible to conduct an effective, thoughtful application of psychotropics through "off-label" prescribing, that is using a medication approved by the FDA generally but not for the specific targeted condition or age group. (Note that this practice is "entirely legal and very common," used in "more than one in five outpatient prescriptions in the U[nited] S[tates]" (Miller, 2015). However, knowledge and an appreciation of the functional neuroanatomy associated with EF deficits are necessary to guide the drug selection. A "rational pharmacology" approach, which uses medications to target specific neurotransmitter subtypes associated with the symptoms presentation, may be most beneficial.

Perhaps the greatest potential benefit of psychotropic use is the measurable improvement in quality of life (Danckaerts et al., 2010; Silver et al., 2011). The best outcomes are achieved with the combination of psychotherapy and medication, with the medication serving as a support to the psychotherapy.

Psychotherapy requires learning, which is more efficient when a client such as Judith is able to attend to the therapy, discern what is salient, tolerate the frustration that accompanies learning, remember what she learned, and apply her new skills reliably in her daily life. Psychotherapy and rehabilitation confront patients with their deficits repeatedly. At early points in Judith's intervention, when she was facing her lost abilities, her ability to cope with the evoked anxiety and helplessness led her to suicide attempt. If pharmacotherapy had been able to ease her anxiety and depression symptoms, so that she could have better tolerated the tasks ahead of her, or had improved her ability to attend to these challenges or her memory, even a small difference would have represented a cumulative change.

The range of potential pharmacological agents for these purposes spans a range of classes, including agents targeting serotonin and dopamine receptors and transport pathways or targeting norepinephrine α -2-adrenoceptors; agents for sleep; atypical stimulants acting on the histamine system; anticholinergic drugs used for dementing disorders; and blockers of NMDA receptors (e.g., memantine). A medication review during Judith's intervention may also have included identifying certain medications that should be stopped; for example, self-prescribed medications, such as Benadryl® taken as a remedy for sleep, can be associated with cognitive dulling (Benadryl® remains bounded to the receptor for 2-3 weeks [Stahl, 2013]). Similarly, prescribed medications, such as the tricyclic antidepressants and Clonidine (used to treat hypertension and attention deficit hyperactivity disorder), can produce cognitive clouding.

HEALTH INTERVENTIONS

Since psychological and social interventions are inherently neurobiological interventions, they serve to enhance the integrity and functioning of the body to improve mental health (Cole, et al., 2003; Miller et al., 2009). Depression symptoms are accompanied by elevated inflammatory markers and have been shown to repeatedly respond to anti-inflammatory drugs in double-blind trials (Raison et al., 2011). Some of these same inflammatory markers were found to increase in the six months after periods of interpersonal conflict, increasing the risk of depression, heart disease and other infectious diseases able to take advantage of a slightly compromised immune system (Miller et al., 2009).

Depressed patients may complain of ill health more frequently because they may actually be experiencing more ill health. The well-known purpose of physical exercise to improve one's waistline is not the only benefit to one's health, as it also increases compounds in the brain that enhance neuronal survival (trophic factors). As a result, our traditional attempts to differentiate body and mind have become increasingly futile. The surgeon Richard Selzer (1996) wrote, "You cannot separate passion from pathology any more than you can separate a person's spirit from his body." Ironically, in my view, this statement brings forth another important question: "Why would you want to try?"

CONCLUSION

In Judith's case study, Rogerian psychotherapy and the adjunct treatments increased Judith's autonomy, dignity, and social support, while helping her cope with her distress without resorting to self-destruction. As described above, these psychosocial treatments were also profound neurobiological interventions. When Judith perceived herself as lonely, helpless, worthless or in danger, this could produce these pathological endocrine changes, altering the lifespan of cells by shortening telomere length and contributing to neuronal apoptosis and oxidative stress. Psychosocial stressors threaten neurohormonal integrity, and vice versa at a cellular and structural level (Lutgendorf et al., 2009; Miller et al., 1997, 2009; Piazza et al., 2013). These psychosocial risk factors also damage some of the same neurohormonal systems as does a traumatic brain injury, creating a greater degree of damage.

Traumatic brain injury damages neurohormonal systems, typically in proportion to the severity of brain injury damage (Masel et al., 2010). Clinical symptoms of endocrine damage are diffuse. These symptoms overlap with symptoms of TBI and depressive disorders as they include: fatigue, poor concentration, irritability, depressed mood, and a decline in overall cognitive functioning (Lynch et al., 1994). Attempting to parse out the etiology and assigning certain symptoms to the mental category and others to the physical category becomes futile; however, treatment is not. Addressing physical, psychological and social health can produce positive change within each domain. In the endocrine system, there is no clear primacy of one source of interventions over the other as the psychosocial factors are well documented and their effect size is substantial (Sapolsky, 2004). Losing social stability and status within a community produce pathological alterations in endocrine functioning (Abbott et al., 2003). Unfortunately, these losses are almost an inevitable consequence of serious illness in the United States (Himmelstein et al., 2009).

Judith's history was consistent with the symptom presentation associated with the endocrine changes associated with TBI and with psychosocial stress, which would have aggravated her executive functioning deficits. She also sustained many of the social and economic harms that may accompany a TBI, although her access to quality care over several years and the financial support provided by her settlement provided a buffer.

Judith's case study demonstrates the empirical value of "higher order" therapies for individuals with neurocognitive impairments, as well as the importance of an integrative, context-sensitive approach to treatment. Her case study also offered me the chance to highlight the value of psychotherapy from a neurobiological perspective, and to share some of the ample literature from social neuroscience with fellow clinicians, for which I am grateful.

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