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Review Article

Head Injury Treatment (HIT): Protocol and Physical Therapy

Patrick di Santo[1*], Casey R Toews[2], Anna Pope[3], Bruce Cappo[4], Merlin G Butler[5]

[1]*Research Scientist affiliated with the University of Kansas, Lawrence, KS, Union Center for Cultural and Environmental Research, Ulster Park, NY, USA*
[2]*Research Scientist affiliated with the University of Kansas, Lawrence, KS, Intern at Clinical Associates, Lenexa, KS, USA*
[3]*Department of Psychology, University of Kansas, Lawrence, KS, USA*
[4]*Clinical Associate PA; Lenexa, KS, USA*
[5]*Department of Psychiatry and Behavioral Sciences, University of Kansas Medical Center Kansas City, KS, USA*

Correspondence

Patrick di Santo

Research Scientist affiliated with the University of Kansas, Lawrence, KS; Union Center for Cultural and Environmental Research, Ulster Park, NY, USA

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Abstract

Physical manifestations vary from subject to subject as well as genetic risk factors when considering traumatic brain injury (TBI)/chronic traumatic encephalopathy (CTE) and recovery. Clinical and behavioral findings may resemble autism that appear with limited mobility and the physical experience may impact treatment. Ataxia, weakness, hemiplegia, hemiparesis are among them. Novel therapy uncovered physical correlations between subject's presentation and physicality with relief and progress when movement and sound are present and monitored during a case study of an adult male with acute head injury from blunt force trauma reported previously. Namely, the release of soft tissue trigger points relieves stress held in the regions affected through soft tissue trigger points stimuli. The occipital attachments for the extensor muscles and positive responses were noted with the stimulation of these regions with effleurage or cross fiber friction and stripping. Vertigo or nausea affecting equilibrium might present as physical displays of deviations of the ability to control balance. Benign Paroxysmal Positional Vertigo or BPPV, vestibular migraines or Broca's aphasia, are among other presentations affecting one's balance and need to be considered during treatment. Challenging desensitization or flooding and habituation with compensatory strategies are required to maintain safety and autonomy of daily living ADL while providing relief when other therapies fall short. One must not underestimate the commitment needed during pre-diagnosis or when diagnosing a disability of head injury by evaluating for other risk factors. This may include advanced genetic testing for potential genes that may contribute to brain injury and recovery. Brain imaging may also be required to assess the location and severity that may impact treatment approaches and recovery responses.

Introduction, Background, and Discussion

Avoiding extraneous stimuli auditory/visual/tactile stimuli following head injury such as overhead fans, excessive lighting, and frequency of interruptions is important. Presenting a tranquil space for assessment is imperative to develop a baseline to communicate with the physicality and presentations.

Genetic risk factors may contribute to head injury and recovery detectable by using next-generation sequencing (NGS) of disease specific genes including for autism, ADD, ADHD, and/or synaesthesia [1]. Specialized genetic testing, brain imaging with PET, CT, and MRS brain scans may allow for identification of other risk factors and the location of brain injury that impact TBI/CTE recovery [1,2]. Identification of risk factors and source triggers associated with head injury and response are often present as: PTSD, hypervigilance, acute savant presentation or synaesthesia, lapse in memory, MDD, and anxiety seen in subject(s) and their predisposing gene variants are important to characterize and understand. A prime target of our recent report and case study of an adult with brain injury and repair is neuroplasticity in the CNS impacted by potential neurodevelopmental genes. Developmental disorder research has uncovered that chromatin remodeling, cell proliferation, migration, synaptic networks, and long-term potentiation are targets of autism spectrum disorder where hundreds of genes have been identified as playing a role [1,3]. These processes are critical to plasticity and deleterious variants in neurodevelopmental genes, and therefore could greatly impact brain repair after TBI and outcome measures of recovery.

A potential intervention often effective is yoga and addressing emotional regulation with health related quality of life or HRQOL for individuals with head injury. Emotional regulation is an important factor affecting those with head injuries and TBI. Yoga and meditation focuses the mind body connection

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by relieving symptoms of vertigo, nausea, anxiety, and OCD as among the much studied other health benefits [4]. Skilled soft tissue therapy and understanding of trigger points to relieve stress in regions of the body are essential not only in pre-diagnosis and symptom relief, but also in the rehabilitation of regaining autonomy of functional independence. For example, the occipital attachments of the extensor muscles (semi spinalis cathetus, splenius pathadis, levator scapulae, upper fibers of trapezius) and positive responses by therapy can be noted with the stimulation of these regions with effleurage (broad strokes to warm the tissue, against the muscle grain) or cross fiber friction and stripping (manipulation with the grain of the muscle fiber). When one employs these novel techniques for treating presentation(s) of head injury in association with nutrition and genetic referencing including pharmacogenetics [5], we see the greatest progress made.

Disruption of processes in the area(s) of neuron plasticity are characteristic of childhood neurological disorders caused by mutations in neurodevelopment functional genes as proposed including: MECP2, FMR1, TSC1, TSC2, UBE3A, and NF1 as examples of genes that impact dendritic spines and synapse morphology and repair. When these genes are mutated then classical conditions such as Rett, fragile X, tuberous sclerosis 1 and 2, Angelman syndrome, and neurofibromatosis type 1 can present and all of these conditions are at risk for autism or a similar clinical presentation. Additionally, the TOR1A gene influences cerebellum synaptogenesis, while SHANK3 modulates the expression of receptors for both AMPA and NMDA, and in turn impact plasticity, long-term potentiation and autism [1,6,7]. In addition, other neurodevelopmental genes are responsible for neuron survival and migration by assisting growth cone formation.

Synaesthesia, a neurological condition, was introduced in our reported case study during childhood, particularly due to the subject having an identifiably different relationship with sensory input from the environment [3]. This neurological condition may follow post-traumatic brain injury as observed by several professionals and from the subject's own reports.

Those with synesthesia are not a different class of people but simply have more explicit experiences, as noted by Ward and Simner [8] with a more extreme manifestation of what all individuals experience. For example asking nonsynaesthetes, 'What color is A?' as being a question while synaesthetes have a window into perception and when pressed for an answer, can pair more common letters with brighter colours or higher pitched notes with lighter colours. Hence, the study documents an unusual case of developmental allophony, phoneme ordering in synesthesia, and sounds that induce an involuntary sensation specific to the individual.

Furthermore, our study proposes genetic markers that predispose the subject to autism spectrum disorder including features of synesthesia and may indicate who are more susceptible to TBI/CTE and variable outcomes. The role of neurogenesis is relevant to repair of injury, recovery and outcome via ischemic damage as neonates or in the pediatric context. It is agreed that quiescent stem cells exist early on that are called into action in the face of injury. Studies show that lab animals' aging brains have quiescent stem cells that are kept in a dormant state by inflammatory signals and antagonism of the Wnt pathway. In recovery, microglia cells in the brain play an active role in repairing damage in the CNS including TBI [7].

Immune cells are responsible for the clearance of debris during post injury, remodeling that occurs along with neurogenesis, angiogenesis and oligodendrogenesis, and remyelination. Furthermore, microglia can also play a detrimental role, through inflammation or neurotoxic cytokines as well documented polarization of microglia and M2-like cells aid in repair processes. An enhancing turnover of microglia either through genetic depletion and replacement or pharmacologic manipulation, considerably enhances recovery after TBI through increased neurogenesis mediated by an IL6 immune response requiring more studies in TBI [7,9].

Strain-dependent differences in the inherent capacity for functional recovery after central nervous system (CNS) injury are known with findings highlighting axon growth within the inflammatory response thereby mediating recovery processes. One study of four mice strains with induced contusion injury to the spinal cord showed better recovery of function in C57Bl/10, B10.Pl research mice, relative to C57Bl/6 or BALB/c44. Spinal cord injury with substantial increase in axonal growth via the lesion area observed in 129 × 1/SvJ animals showed an association decreased chronic inflammatory response relative to C57Bl/645 mice [10]. With fewer macrophages in the lesion of 129 × 1/SvJ animals, more neurons and astrocytes were generated, levels of laminin proved higher, and chondroitin sulfate proteoglycan (CSPG) was lower; all play a role in neuro-development and scar formation [9].

The third spinal cord injury reported in 129 × 1/SvJ mice displayed significant corticospinal axon extension relative to C57Bl/625. Axon generation regrowth was enhanced in both strains on a Nogo−/− background which was reflected in an in vitro study of dorsal root ganglia neurite outgrowth with more macrophages found in the lesions of the C57Bl/6 animals [11].

Two strains with differentially expressed genes were associated with neurite growth, synapse formation, inflammation, and immune response. This oxidative stress study on research animal neuronal cultures revealed strain differences in innate neuronal response reflecting the ability to adapt more efficiently to inflammation [12], but more animal research is needed and its impact on TBI.

In addition, those with autism spectrum disorder and synesthesia appear to have a highly structured, non-random relationship between particular combinations of phonemes rather than graphemes, influenced by a number of finegrained phonemic property orderings, allophone and phoneme [13]. These uncommon experiences have no modern frame of reference thus fall under the label of spectrum disorder. This misrepresentation of experiences inhibits the full understanding of these individuals as we reported on the adult subject with brain injury in our previous study [3].

Current protocols may take months to years to understand in their full form, affecting vocabulary acquisition initiated and guided by learned linguistic and conceptual education and rehabilitation. Trigger phonemes appear with corresponding semantic association between the word expression and thought. Innate connections from the perceptual system relay to another, if the other sense is damaged as in the cases of autism spectrum disorder and TBI/CTE, a novel association may be established to make communication of the subject's experience more timely. If this is the case, it is possible to influence symbolic/conceptual level of representation to interpret the highest level of communication acceptable to current societal norms. In other words, by addressing the damaged brain and allowing recovery space to be minimally

influenced by non-helpful stimuli, we can induce the best case scenario for treatment and recovery protocol in those with TBI [14-16].

Perspectives in cognitive neuroscience vary as do philosophies. What is most often agreed upon is the effects of priming and framing on individuals without the tools to navigate the complex experience. Sensations uncovered and raw form trauma can produce the best results by keeping the subject clear of distractions and unhelpful stimuli: i.e., media overload. A person with both TBI/CTE and autism spectrum or synesthesia may have a slower recovery time naturally. When synapse and neurons are over-working from excessive stimuli, or incorrect physcal therapy approaches can damage or slow rate of deciphering information in the framing and priming aspect of the scenario [15]. The reasoning is the same as in children under the age of 5 years needing mirroring and correction to appropriate the acceptable behavior of that which is in their environment. If you expose the child to unacceptable behaviors, they will mirror and connect these behaviors to become their current norm [17].

Involuntary, automatic, and highly consistent protocols will establish this mechanism to protect the person with a head injury from having to waste valuable time sorting through the harangue of media over stimuli. Crossing of the senses where a stimulus received in one sense gives rise to an experience in another and/or others is a common phenomenon. First, there are many apparent variants involving qualities within a single sense, inducing stimuli or inducers that are not restricted to conventional sensory input, meaningful units, numerals and words that can be used as part of rehabilitation. Meaningful quality for the subject being addressed in turn triggers the experience. Cognitive association, alphanumeric personification, letters and numerals are paired with consistent symbols that reflect the personality traits often associated with communication.

Perceptual quality is experienced individually per subject and needs to be observed and diagnosed by an educated practitioner [18,19]. Apparent prototypical hearing triggered by spoken language have recently been shown to be triggered by abstract cognitive representations, rather than purely perceptual processing. A unified notion of a condition is being argued that has disparate and isolated qualities. This area of research is in its infancy and many questions presently remain unanswered [18,19].

Triggering stimuli presented as spoken language, grapheme and phoneme are in fact distinct variants, each with their own underlying systems used to communicate is often lost in the translation of head injury, regardless of the input mode of the triggering stimulus. The range of viable terminology used is wide and simply reflects the lack of terminological agreement throughout the current literature [20].

This pre-identification and correct diagnosis remain essential in understanding rehabilitation in post-head injury protocol. A more precise diagnosis of an individual's novel presentation(s) is needed to address functional recovery in the CNS in both animals and human subjects that further impact on prognosis and treatment. One needs to employ a holistic plan of care, including traditional therapeutic activities: nutrition, meditation, yoga movement(s), tai chi, dynamic vestibular exercises, and soft tissue manipulation(s). In vitro modeling of cells and the use of organoid cultures along with complete-organism studies are needed to help identify gene presentation mode(s) and test their interaction(s), and networks that manifest individual variation in recovery from brain injuries. Research points in the direction and development of new individual therapeutic approach(s) [21, 22]. Homogeneous treatment plans do not work for all head injuries to obtain optimal recovery. Novel treatment plans may be needed based on the type of brain injury, severity, and location as well as other risk factors and pre-existing conditions that might affect treatment, further impacted by pharmacogenetics and medication management. Using our described protocol and physical therapy approaches should further demonstrate the importance of this mindset and validity of its employment in treating subjects with head injury, one subject at a time.

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