NAVIGATING PEDIATRIC BRAIN INJURY MODERN SOLUTIONS FOR A DATED SYSTEM

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TLH4Minds Conference 2021

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The AUST
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HOPE FOR MINDS


1/2

This is what Casyn thinks of pediatric brain injury

Goals for today

1. **Community Development**
2. **Provide Answers - please come talk to me**
3. **Provide Hope**
4. **Lecture Discussion:**
    a. **Anoxic brain injury overview**
    b. **Give therapy strategies you can start now**
    c. **Discribe overall healing process**
    d. **Apply to ALL TYPES OF BRAIN INJURY**

Etiology

Anoxic Brain Injury

MONICA VERDUZCO-GUTIERREZ, MD, AND SIMA A. DESAI, MD

Anoxia is defined by the total absence of oxygen to the tissues or a particular organ such as the brain. The term *anoxic brain injury* (ABI) is frequently used synonymously in the literature with terms *hypoxic-ischemic*, *anoxic-ischemic*, *hypoxic*, or *cerebral anoxia*.

One of the earliest documented cases of ABI was in 1945 with eight cases of poisoning from nitrous oxide anesthesia that resulted in persistent deficits in judgment, attention, and memory; loss of insight; apathy; indifference, and restlessness.[1]

Etiology

Anoxic Brain Injury

MONICA VERDUZCO-GUTIERREZ, MD, AND SIMA A. DESAI, MD

Etiology

The etiology of ABI can be related to any event that deprives the brain of sufficient oxygenation. In addition to cardiac arrest and respiratory failure, a variety of disorders such as carbon monoxide poisoning, asphyxiation because of hanging, near drowning, obstructive sleep apnea, complications from anesthesia, metabolic conditions, and pulmonary disease can be attributable causes.[2]

Etiology

Most common ways to acquire an ABI

1. **Cariac Arrest - Majority**
2. Respiratory Failure
3. Carbon Monoxide Poisoning
4. Asphyxiation; hanging
5. **Near Drowning / Non Fatal Drowning**
6. Obstructive Sleep Apnea
7. Complications from anesthesia
8. Metabolic Conditions
9. Plumonary Disease

Etiology

Epidemiology

The majority of data for ABIs are in the setting of cardiac arrest. The majority of cardiac arrest events occur outside of the hospital in a private residence. The incidence of emergency medical services (EMS)-assessed cardiac arrest in the adult population is 347,322 per year, based on the most recent data by the American Heart Association (AHA).[3]

Survival with good functional outcomes was present for a majority of these patients, as defined by independence with

Pathophysiology

Primary Mechanisms

There are two primary mechanisms of injury with ABI: primary and secondary. During the primary injury, there is first ischemia and then subsequent reperfusion. During the process of the brain ischemia, there is anoxic depolarization, adenosine triphosphate (ATP) depletion, glutamate release, free radical formation, and nitric oxide production. The primary injury causes considerable neuronal damage, but the successive reperfusion accounts for substantial cerebral ischemia and cell death. Despite the return of spontaneous circulation during cardiac arrest, there is a brief period of hyperemia that is quickly replaced by a longer period of global and multifocal hypoperfusion known as the no reflow phenomenon

Anoxia Tolerant Brains

Göran E. Nilsson, Peter L. Lutz

First Published May 1, 2004 | Research Article | Find in PubMed
https://doi.org/10.1097/00004647-200405000-00001

Anoxic depolarization is a progressive and uncontrollable depolarization of neurons during stroke or brain ischemia in which there is an inadequate supply of blood to the brain. Anoxic depolarization is induced by the loss of neuronal selective membrane permeability and the ion gradients across the membrane that are needed to support neuronal activity. Normally, the Na+/K+-ATPase pump maintains the transmembrane gradients of K+ and Na+ ions, but with anoxic brain injury, the supply of energy to drive this pump is lost.[2] The hallmarks of anoxic depolarization are increased concentrations of extracellular K+ ions, intracellular Na+ and Ca2+ ions, and extracellular glutamate and aspartate. Glutamate and aspartate are normally present as the brain's primary excitatory neurotransmitters, but high concentrations activate a number of downstream apoptotic and necrotic pathways. This results in neuronal dysfunction and death.

Discussion on Seizures in Brain Injury

Discussion with Asher Bevens parents

Pathophysiology

Primary Mechanisms

Ischemia:

- Anoxic Depolarization
- ATP Depletion
- Glutamate Release
- Free Radical Formation
- Nitric Oxide Production
- Succesive Reperfusion - hyperemia quickly replaced by longer periods of global and multifocal hypoperfusion
- No reflow phenomenon - microvascular damage / autonomic dysfunction

Pathophysiology

Primary Mechanisms

Secondary injury occurs immediately after return of spontaneous circulation and is the result of the additive cerebral injury caused by an imbalance of postresuscitation cerebral oxygen delivery. Secondary injury consists of ongoing ischemia, autoregulatory failure, cerebral hypoperfusion, blood-brain barrier breakdown, seizures, oxidative injury, and hyperpyrexia

Pathophysiology

Secondary Mechanisms

Ischemia / Reperfusion:

- Imbalance in cerebral oxygen with resuscitation
- Ongoing ischemia
- Autoregulatory failure
- Ongoing Cerebral hypoperfusion
- Blood Brain Barrier Dysfunction
- Seizures
- Oxidative Injury
- Hyperpyrexia - dysfunction to hypothalamus, body temp above 106.7 deg F / 41.5 deg C

TABLE 55.1 Factors Associated with Poor Outcome

Symptom	Time Frame
Anoxia duration	>8–10 min
Duration of cardiopulmonary resuscitation	>30 min
Myoclonic status epilepticus	Day 1
Absent pupillary or corneal reflexes	Days 1–3
Serum neuron-specific enolase (NSE) >33 μg/L	Days 1–3 for nontherapeutic hypothermia patients
Absent N20 responses on somatosensory evoked potentials (SSEP) bilaterally	Days 1–3
Motor response extensor or none	Day 3 for nontherapeutic hypothermia patients; possibly longer for therapeutic hypothermia patients
Electroencephalogram (EEG) with nonreactive background	
EEG with burst suppression and generalized epileptiform activity	
Loss of gray-white matter differentiation on head computed tomography	
Widespread cortical restricted diffusion on brain magnetic resonance imaging	

Data from Fugate, JE, Wijdicks, EFM. Anoxic-Ischemic Encephalopathy. In: Flemming KD, Jones LK, eds *Mayo Clinic Neurology Board Review: Clinical Neurology for Initial Certification and MOC*. New York: Oxford University Press; 2015. p. 35–8; Wijdicks EFM, Hijdra A, Young GB, Bassetti CL, Wiebe S. Practice Parameter: Prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology.* 2006 Jul 25;67(2):203–10; Bouwes A, Binnekade JM, Kuiper MA, Bosch FH, Zandstra DF, Toornvliet AC, et al. Prognosis of coma after therapeutic hypothermia: A prospective cohort study. *Ann Neurol.* 2012 Feb;71(2):206–12.

Pathophysiology

Most Commmon Areas of Damage

There are specific areas of the brain that are more vulnerable to damage than others: the **hippocampus** (CA1 pyramidal neurons); Purkinje cells of the **cerebellum**; pyramidal neurons in layers three, five, and six of the **neocortex**; reticular neurons of the **thalamus**; neurons of the **striatum**; and **vascular border zone areas**

Pathophysiology

Most Commmon Areas of Damage

- Hippocampus (CA1 pyramidal neurons)
- Purkinje cells of the Cerebellum
- Pyramidal Neurons in layers 3, 5 & 6 of the Neocortex
- Reticular neurons of the thalamus
- Neurons of the striatum
- Vascular border zone areas

Pathophysiology

Attention • Processing speed • Memory impairments • Executive dysfunction • Language impairments • Calculation impairments • Apraxia • Agnosia• Visuospatial impairments • Balint syndrome (simultagnosia, optic ataxia, ocular apraxia) • Anton syndrome (anosognosia for visual impairment) • Alterations in personality and behavior • Affective dysregulation

Pathophysiology

Additionally, one should also assess for neurological impairments such as motor deficits, parkinsonism, dystonia, chorea, tremor, tics, athetosis, seizures, and myoclonic syndromes

Pathophysiology

Man-in-a-barrel syndrome (bilateral proximal upper limb paresis with preservation of lower limb function caused by injury between the anterior cerebral artery and middle cerebral artery watershed zone) • Paraparesis and quadriparesis in the upper and lower thoracic and lumbar regions of the spinal cord • Cortical blindness and Balint syndrome are examples of disorders of sensory function • Akinetic-rigid syndrome• Amnestic syndrome caused by hippocampal damage• Lance-Adams syndrome (significant action myoclonus associated with ataxia)

Pediatric Non Fatal Drowning

- Most common ages 1-4 years old
- 2 out of 3 survive drowning
- Most common location: swimming pool for children / natural bodies of water for teens
- Males have twice the mortality rate than females: some report 80% of drownings are male

Functional Integrity in Children With Anoxic Brain Injury From Drowning

Mariam Ishaque,[1,2]* Janessa H. Manning,[3] Mary D. Woolsey,[1] Crystal G. Franklin,[1] Elizabeth W. Tullis,[4] Christian F. Beckmann,[5,6,7] and Peter T. Fox [1,2,8,9]*

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[7]*Centre for Functional MRI of the Brain, University of Oxford, Oxford, United Kingdom*
[8]*South Texas Veterans Healthcare System, San Antonio, Texas*
[9]*Shenzhen University School of Medicine, Shenzhen, People's Republic of China*

Abstract: Drowning is a leading cause of accidental injury and death in young children. Anoxic brain injury (ABI) is a common consequence of drowning and can cause severe neurological morbidity in survivors. Assessment of functional status and prognostication in drowning victims can be extremely challenging, both acutely and chronically. Structural neuroimaging modalities (CT and MRI) have been of limited clinical value. Here, we tested the utility of resting-state functional MRI (rs-fMRI) for assessing brain functional integrity in this population. Eleven children with chronic, spastic quadriplegia due to drowning-induced ABI were investigated. All were comatose immediately after the injury and gradually regained consciousness, but with varying ability to communicate their cognitive state. Eleven neurotypical children matched for age and gender formed the control group. Resting-state fMRI and co-registered T1-weighted anatomical MRI were acquired at night during drug-aided sleep. Network integrity was quantified by independent components analysis (ICA), at both group- and per-subject levels. Functional-status assessments based on in-home observations were provided by families and caregivers. Motor ICNs were grossly compromised in ABI patients both group-wise and individually, concordant with their prominent motor deficits. Striking preservations of perceptual and cognitive ICNs were observed, and the degree of network preservation correlated ($\rho = 0.74$) with the per-subject functional status assessments. Collectively, our findings indicate that rs-fMRI has promise for assessing

Drowning is the second most prevalent cause of unintentional injury death in children 1–4 years of age. Nonfatal drowning (i.e., cardiopulmonary resuscitation is successful) is prevalent in this age group, with an estimated 2 out of 3 drowned children surviving [Borse and Sleet, 2009; Kriel et al., 1994; Topjian et al., 2012]. Neurological morbidity from anoxic brain injury (ABI) is a frequent outcome, as the brain is exquisitely sensitive to oxygen deprivation [Topjian et al. 2012].

Pathophysiology

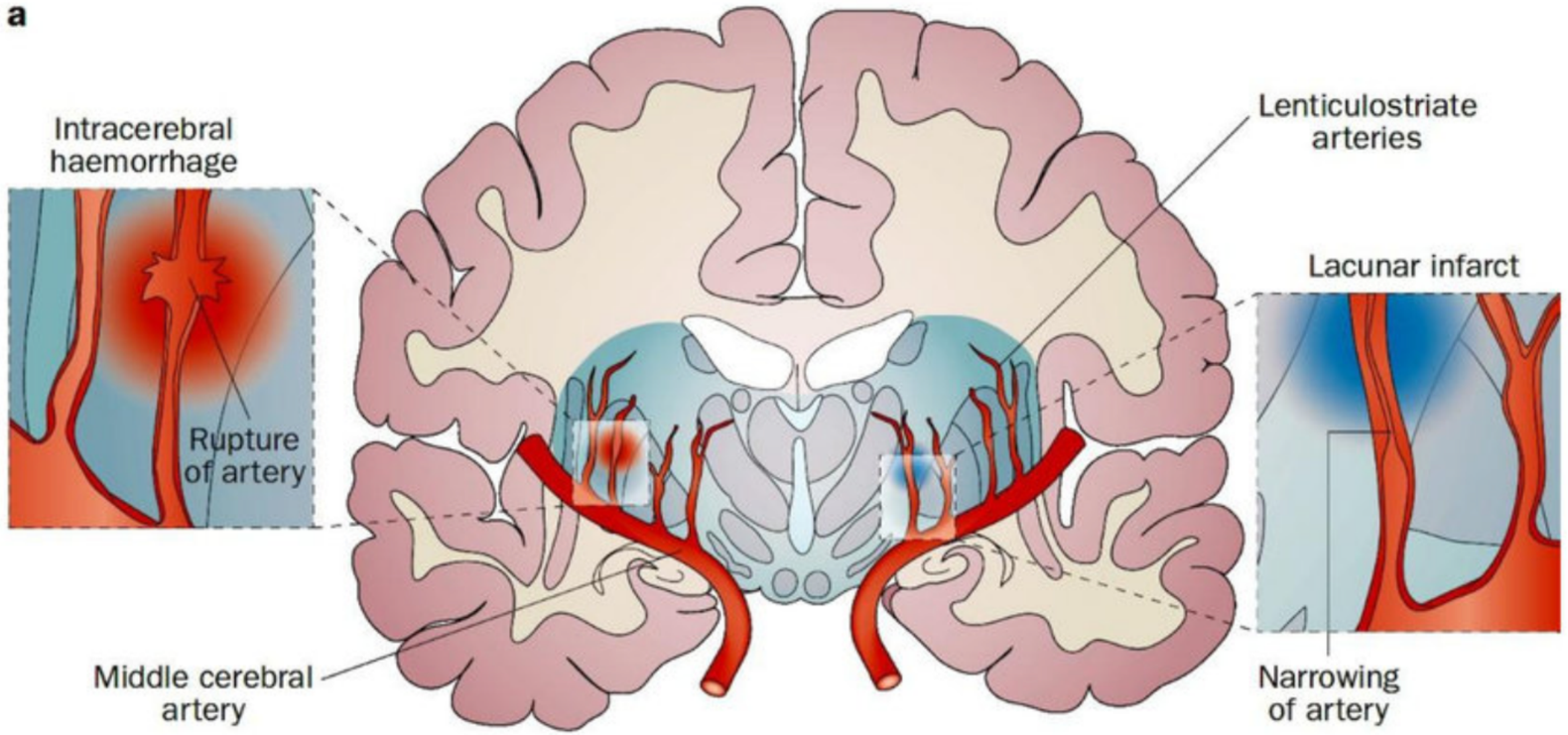
The neuropathology of pediatric, nonfatal ABI is not well established. Postdrowning anoxic injury has been described as diffusely **affecting grey matter more than white matter**, reflecting the respective metabolic demands of the two tissue types [Huang and Castillo, 2008; Rabin- stein and Resnick, 2009]. However, the possibility of a **more selective injury must be considered** in view of the predominant motor-system disability observed in survivors.

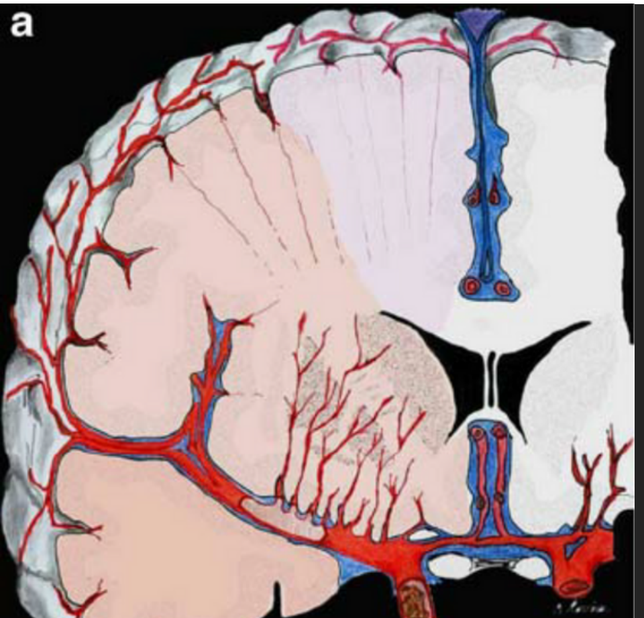
Pathophysiology

Grey matter loss was largely restricted to the **basal ganglia** and **thalamus**; white matter loss predominately affected the **posterior limb of the internal capsule (PLIC)**. Tract-based spatial statistics (TBSS) was applied to diffusion-weighted MRI to quantify fractional anisotropy and mean diffusivity [Ishaque et al., 2017]. TBSS indepen- dently confirmed our VBM white-matter findings, also showing **highly focal, deep subcortical lesions**.

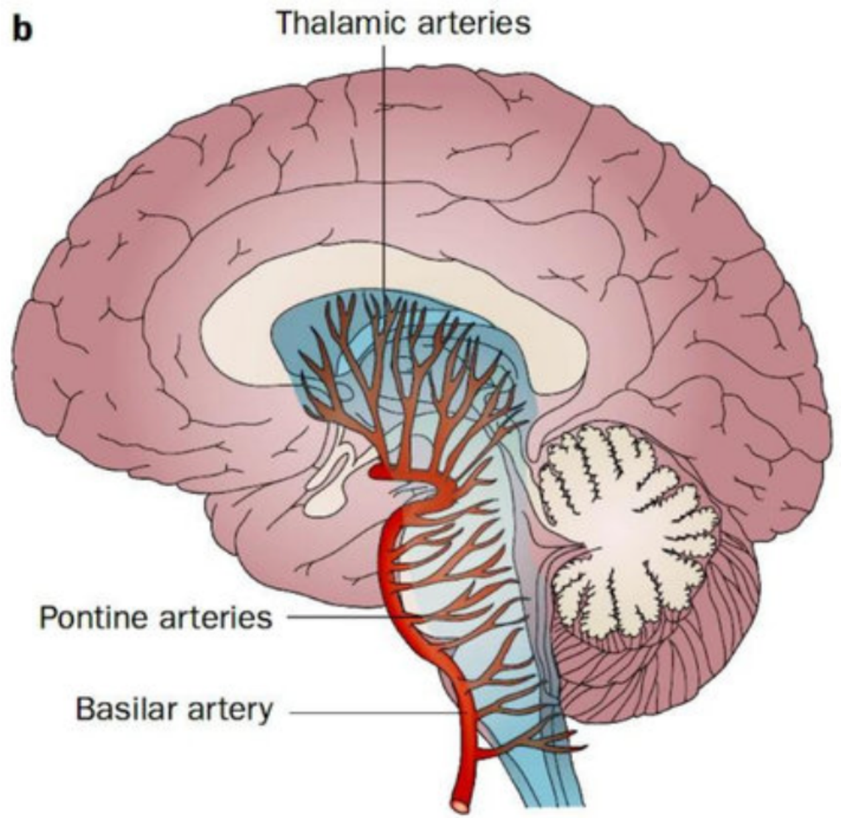
Pathophysiology

For both modalities, the lesion was largely confined to the **lenticulostriate vascular distribution**, suggesting that this end-arterial watershed zone may be uniquely susceptible in young children. A similar distribution has been reported in **perinatal asphyxia** using diffusion tensor imaging (DTI) [Barkovitch et al., 2001] and magnetic resonance spectroscopy [Pu et al., 2000], lending further credence to this hypothesis.





Lenticulostriate Watershed
&
Border Zones

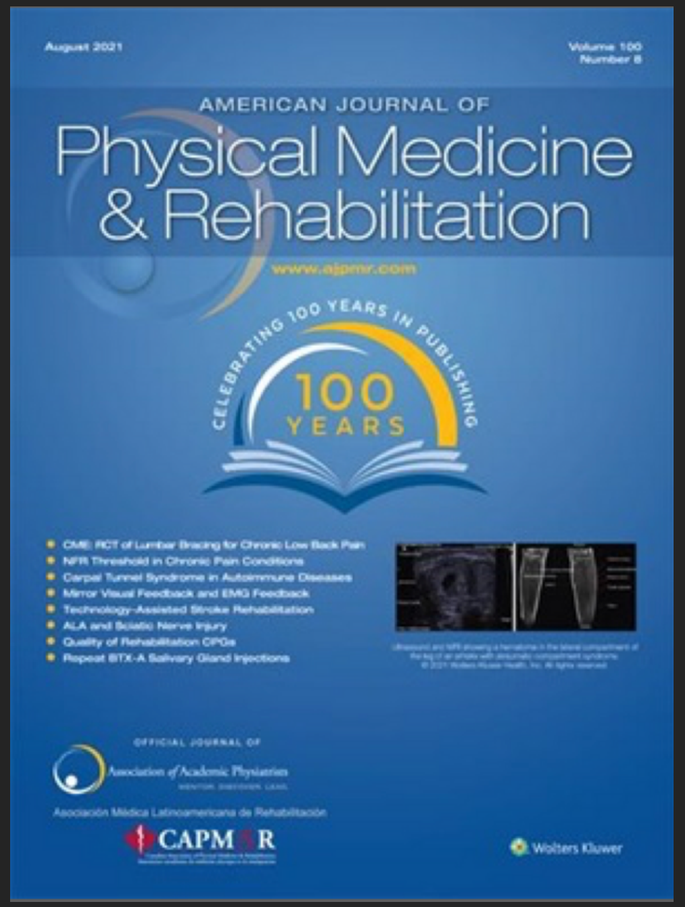


Pathophysiology

Descending **corticospinal and corticobulbar fibers** (pyramidal tract) pass through the **PLIC**, the **cerebral peduncles (midbrain)** and **anterior pons** en route to the pyramidal decussation **(medulla)**. Collectively, these observations suggest that pediatric drowning victims may be an at-risk population for a **selective deefferentation syndrome**, with ***disproportionate motor impairment and relatively preserved perceptual and cognitive function.***

Deefferentation Syndrome
Locked In Syndrome

...selective motor-system impairment with relative preservation of perceptual and cognitive function in children with post drowning ABI. Collectively, these observations suggest that the substantial motor deficits in these children—all are quadriplegic and most (7/10) are aphonic—arising from structural damage to the basal ganglia and posterior limb of the internal capsule, and from functional compromise of the Basal Ganglia and Cerebellar networks, effectively prohibit full expression of their level of awareness and cognition.



RESEARCH ARTICLES

AUTONOMIC DYSFUNCTION ASSOCIATED WITH LOCKED-IN SYNDROME IN A CHILD[1]

Scott, Jane; Ockey, Robin; Holmes, Grace[2]; Varghese, George

Author Information

American Journal of Physical Medicine & Rehabilitation: May 1997 - Volume 76 - Issue 3 - p 200-203

The autonomic dysfunction syndrome is a state characterized by hyperthermia, tachycardia, hypertension, tachypnea, pupillary dilation, increased extensor posturing, and diaphoresis.[1] In three reported cases of autonomic dysfunction syndrome,[2] the neurologic examinations and computed tomographic head scans indicated hypothalamic-mesencephalic dysfunction. This state is also referred to as autonomic or hypothalamic storming.

Pathophysiology

Regression of these intrinsic connectivity networks (ICNs) to group-specific maps demonstrated **substantial preservation of the Sensorimotor, Visual 1–3, Auditory, and Default Mode networks** in the patient group. **Less preservation was observed in the Left Frontoparietal, Right Frontoparietal, and Executive Function networks. The least preservation was observed in the Basal Ganglia and Cerebellar networks** ($P < 0.05$; Fig. 2, bottom row and Fig. 3).

Pathophysiology

Significant between-group differences were measured in the **Basal Ganglia, Cerebellar, and Left Frontoparietal networks** (P<0.05, corrected; Fig. 2, top row and Table II). Peak areas of decreased ICN connectivity in the ABI patient group were identified in ***bilateral caudate bodies (Basal Ganglia), posterior cerebellar lobe (Cerebellum), and left inferior parietal lobule (Left Frontoparietal)***.

Pathophysiology

TABLE II. Between-group network differences

Anatomical location	Hemisphere	Talairach coordinates of global maxima			Extent (voxels)
		x	*y*	*z*	
Control > ABI					
Basal ganglia					
Caudate body	L	−14	1	19	3,885
Caudate body	R	15	2	12	1,273
Cerebellum					
Posterior lobe	L	−27	−71	−24	37,901
Left frontoparietal					
Inferior parietal lobule	L	−36	−46	54	2,503

Locations, coordinates, and extents of peaks of significant between-group differences in intrinsic connectivity network (ICN) connectivity (from group ICA and dual regression analysis) are shown. Significant differences were identified in three ICNs: basal ganglia, cerebellum, and left frontoparietal (Controls > Patients; $P < 0.05$, corrected for multiple comparisons).

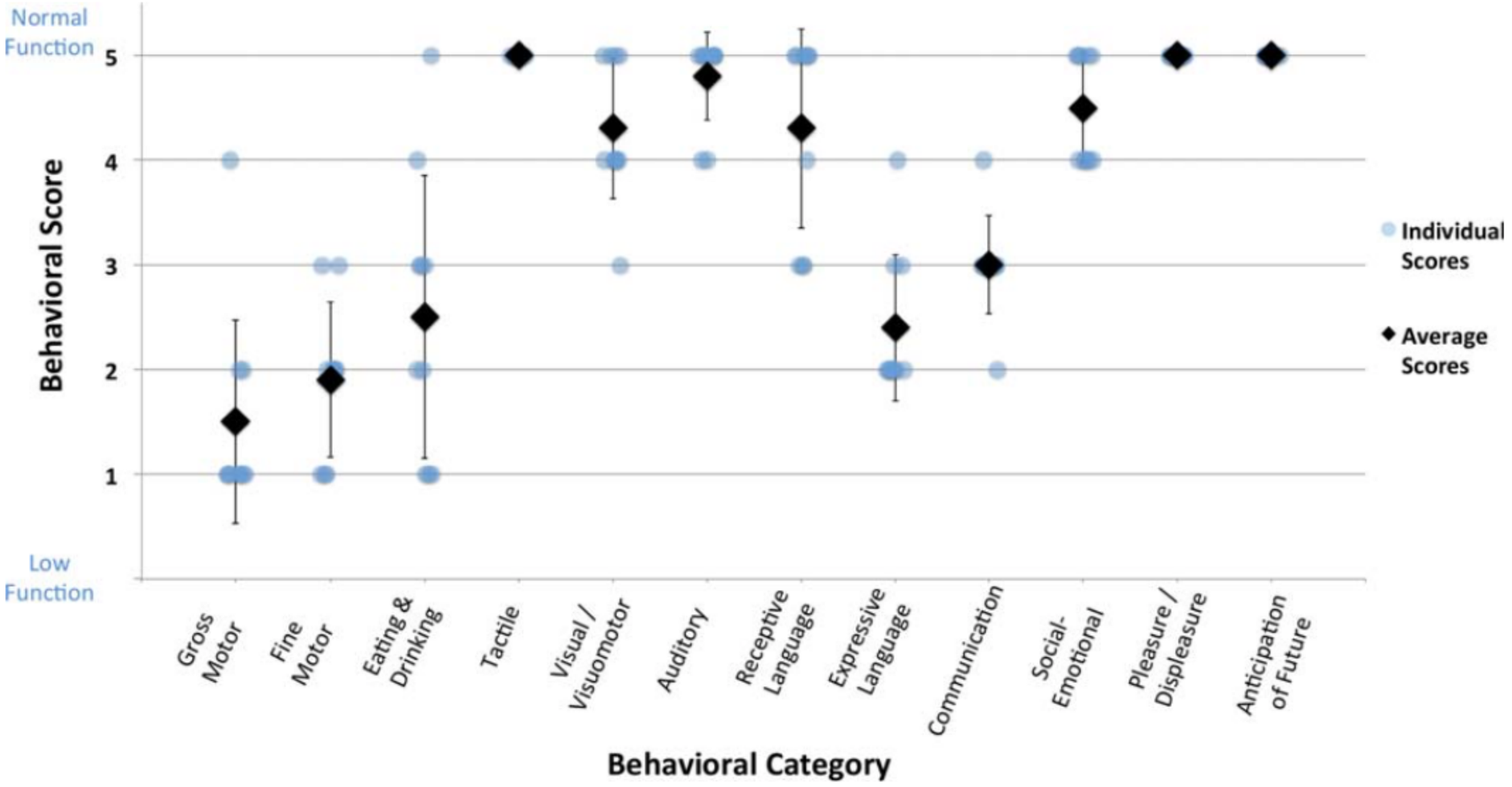


Figure 4.

Per-subject ABI behavioral data. Per-subject behavioral scores (with averages and standard deviations) for ABI patients are shown for 12 behavioral categories. The first 10 categories were assessed with a 1–5 scoring system (low–normal function); the last 2 categories were assessed with a Yes/No response (Yes = 5, No = 1). See Supporting Information for complete behavioral assessment form contents.

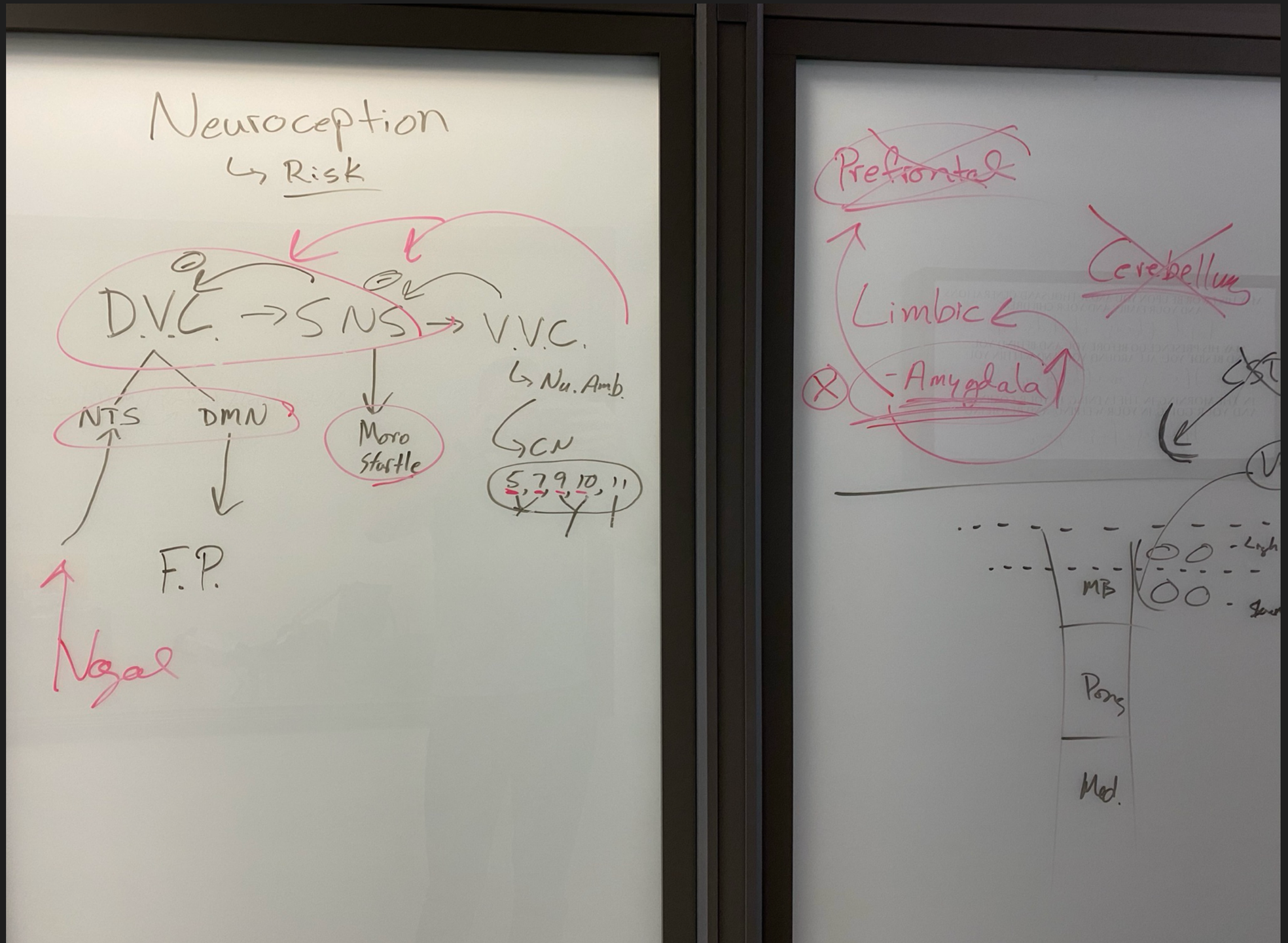
Pathophysiology

Motor ICNs (Basal Ganglia, Cerebellum) were grossly compromised in ABI patients both group-wise and individually, concordant with their prominent motor deficits. **Striking preservations of perceptual (Visual, Auditory, Sensorimotor) and cognitive ICNs (Default Mode, Frontoparietal, Executive Function) were observed**, and the degree of network preservation correlated (q50.74) with per-subject functional status assessments.

Pathophysiology

Collectively, our findings indicate that the severe motor deficits observed in this population can mask relatively intact perceptual and cognitive capabilities.

Compounding Factors for Locked in Syndrome
Poly Vagal Theory applications

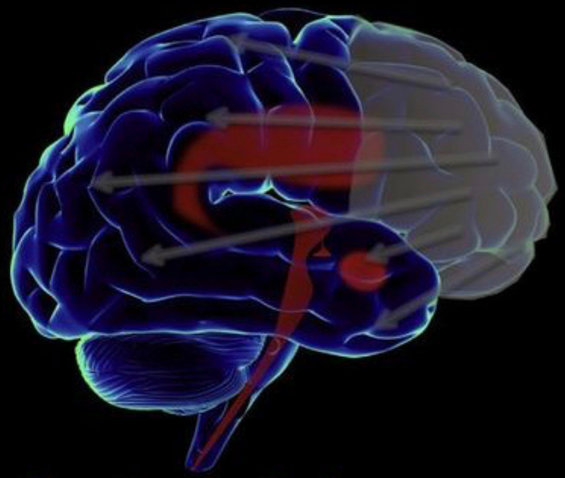

Neuroception
↳ Risk
D.V.C. → SNS → V.V.C.
↳ Nu. Amb.
↳ CN
5, 7, 9, 10, 11
NTS
DMN
Moro Startle
F.P.
Vagal
Prefrontal
Cerebellum
Limbic
Amygdala
MB
Pons
Med.

brainchat

The higher cognitive functions of the prefrontal cortex (PFC) are impaired by fatigue or uncontrollable stress

ALERT
Strong PFC Top-Down Control

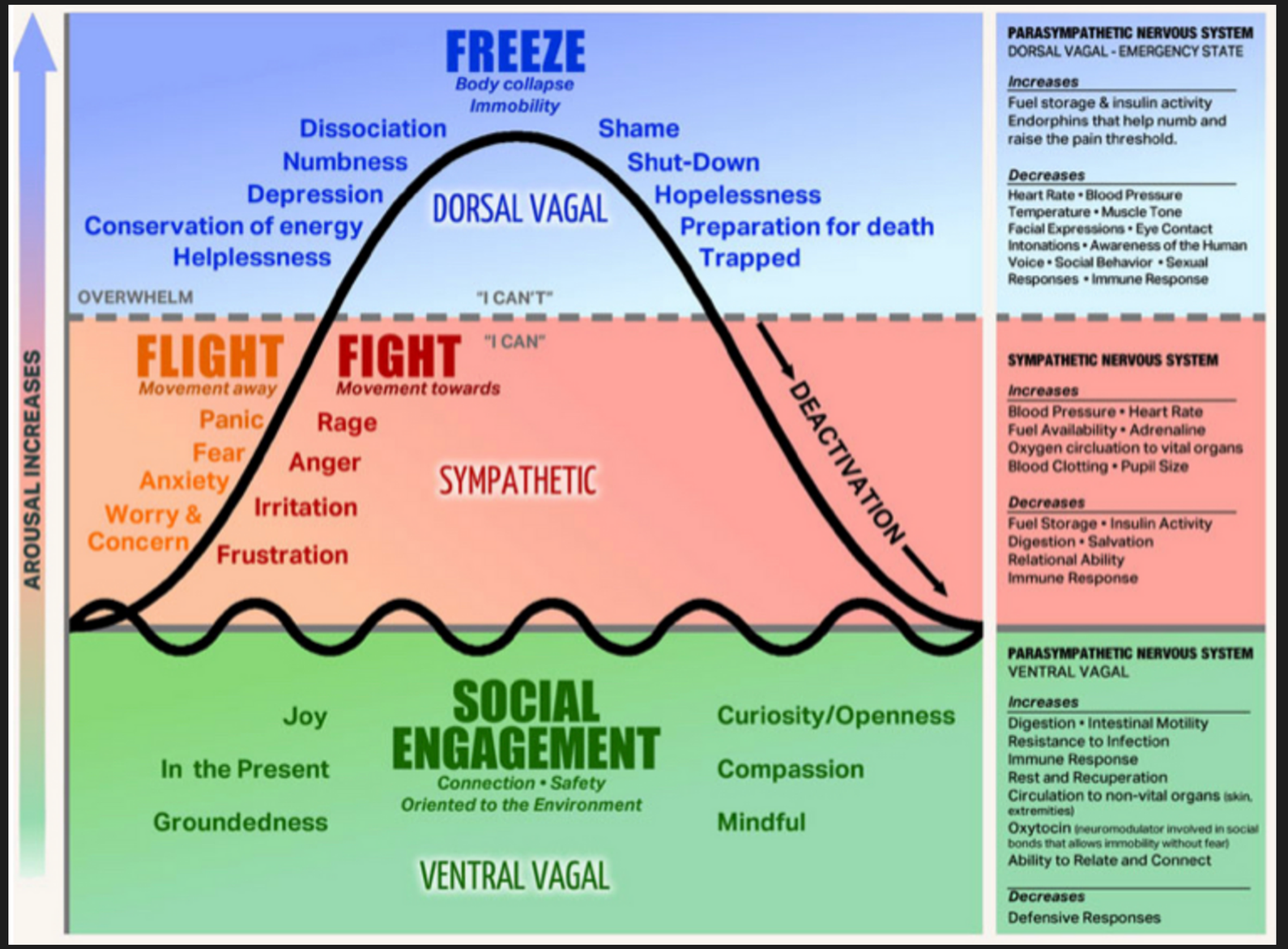
FATIGUE/STRESS
Weak PFC Top-Down Control



Examples of PFC dysfunction include:

- Forgetful thinking
- Difficulty concentrating
- Impaired decision making
- Reduced insight and judgment
- Decreased empathy
- Decreased optimism
- Decreased compassion
- Decreased self-regulation
- Decreased inhibition control
- Disorganized

https://www.mayoclinicproceedings.org/article/S0025-6196(20)31500-7/pdf

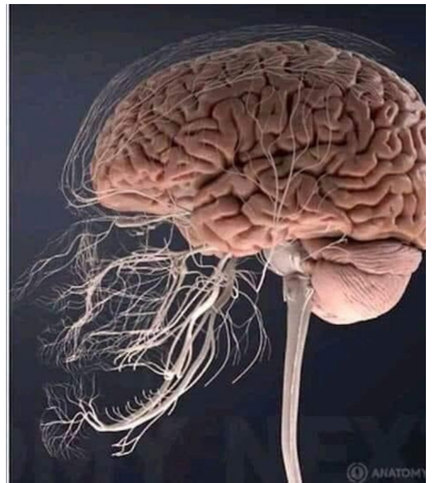

FREEZE
Body collapse
Immobility
Dissociation
Numbness
Depression
Conservation of energy
Helplessness
DORSAL VAGAL
Shame
Shut-Down
Hopelessness
Preparation for death
Trapped
OVERWHELM
"I CAN'T"
"I CAN"
FLIGHT
Movement away
Panic
Fear
Anxiety
Worry &
Concern
FIGHT
Movement towards
Rage
Anger
Irritation
Frustration
SYMPATHETIC
DEACTIVATION
AROUSAL INCREASES
SOCIAL
ENGAGEMENT
Connection • Safety
Oriented to the Environment
Joy
In the Present
Groundedness
Curiosity/Openness
Compassion
Mindful
VENTRAL VAGAL
PARASYMPATHETIC NERVOUS SYSTEM
DORSAL VAGAL - EMERGENCY STATE
Increases
Fuel storage & insulin activity
Endorphins that help numb and raise the pain threshold.
Decreases
Heart Rate • Blood Pressure
Temperature • Muscle Tone
Facial Expressions • Eye Contact
Intonations • Awareness of the Human Voice • Social Behavior • Sexual Responses • Immune Response
SYMPATHETIC NERVOUS SYSTEM
Increases
Blood Pressure • Heart Rate
Fuel Availability • Adrenaline
Oxygen circluation to vital organs
Blood Clotting • Pupil Size
Decreases
Fuel Storage • Insulin Activity
Digestion • Salvation
Relational Ability
Immune Response
PARASYMPATHETIC NERVOUS SYSTEM
VENTRAL VAGAL
Increases
Digestion • Intestinal Motility
Resistance to Infection
Immune Response
Rest and Recuperation
Circulation to non-vital organs (skin, extremities)
Oxytocin (neuromodulator involved in social bonds that allows immobility without fear)
Ability to Relate and Connect
Decreases
Defensive Responses

NeuroSolution

CHANGING LIVES WITH THE POWER OF LIGHT

Vagal System Rehab

The most powerful vagal rehab procedures



NeuroSolution

Vagal System Rehab

The most powerful vagal rehab procedures

Nu Tract Solitarius

Vibrate the Abdomen
Heat

Moro to inhibit F.P.

Light, Sound, Moro
Tectospinal Reflex

CN 5, 7

Facial stimulus
Facial mm contraction
Taste

CN 9 & 10

Stim the tongue, gag/gargle
Auricular branch of vagus
Stim / vibration / acoustics

CN 11

Stim SCMs and Traps

Social Engagement

Talk and engage with others while treating
Looking at family photos

Spinal Cord Considerations

Central Pattern Generators

Published: 03 August 2021

Corticospinal Motor Circuit Plasticity After Spinal Cord Injury: Harnessing Neuroplasticity to Improve Functional Outcomes

Syed Faraz Kazim, Christian A. Bowers, Chad D. Cole, Samantha Varela, Zafar Karimov, Erick Martinez, Jonathan V. Ogulnick & Meic H. Schmidt

Molecular Neurobiology (2021) | Cite this article

Abstract

Spinal cord injury (SCI) is a devastating condition that affects approximately 294,000 people in the USA and several millions worldwide. The corticospinal motor circuitry plays a major role in controlling skilled movements and in planning and coordinating movements in mammals and can be damaged by SCI. While axonal regeneration of injured fibers over long distances is scarce in the adult CNS, substantial spontaneous neural reorganization and plasticity in the spared corticospinal motor circuitry has been shown in experimental SCI models, associated with functional recovery. Beneficially harnessing this neuroplasticity of the corticospinal motor circuitry represents a highly promising therapeutic approach for improving locomotor outcomes after SCI. Several different strategies have been used to date for this purpose including neuromodulation (spinal cord/brain stimulation strategies and brain-machine interfaces), rehabilitative training (targeting activity-dependent plasticity), stem cells and biological scaffolds, neuroregenerative/neuroprotective pharmacotherapies, and light-based therapies like photodynamic therapy (PDT) and photobiomodulation (PMBT). This review provides an overview of the spontaneous reorganization and neuroplasticity in the corticospinal motor circuitry after SCI and summarizes the various therapeutic approaches used to beneficially harness this neuroplasticity for functional recovery after SCI in preclinical animal model and clinical human patients' studies.

NeuroSolution

Conditioning = Network Efficiency

Positive Neuroplasticity - We have to create more + plasticity vs - plasticiy to create & maintain sustainable change.

Negative Neuroplasticity

Neuropsychol Rev (2014) 24:409–427
DOI 10.1007/s11065-014-9273-6

REVIEW

Negative Neuroplasticity in Chronic Traumatic Brain Injury and Implications for Neurorehabilitation

Jennifer C. Tomaszczyk · Nathaniel L. Green · Diana Frasca · Brenda Colella · Gary R. Turner · Bruce K. Christensen · Robin E. A. Green

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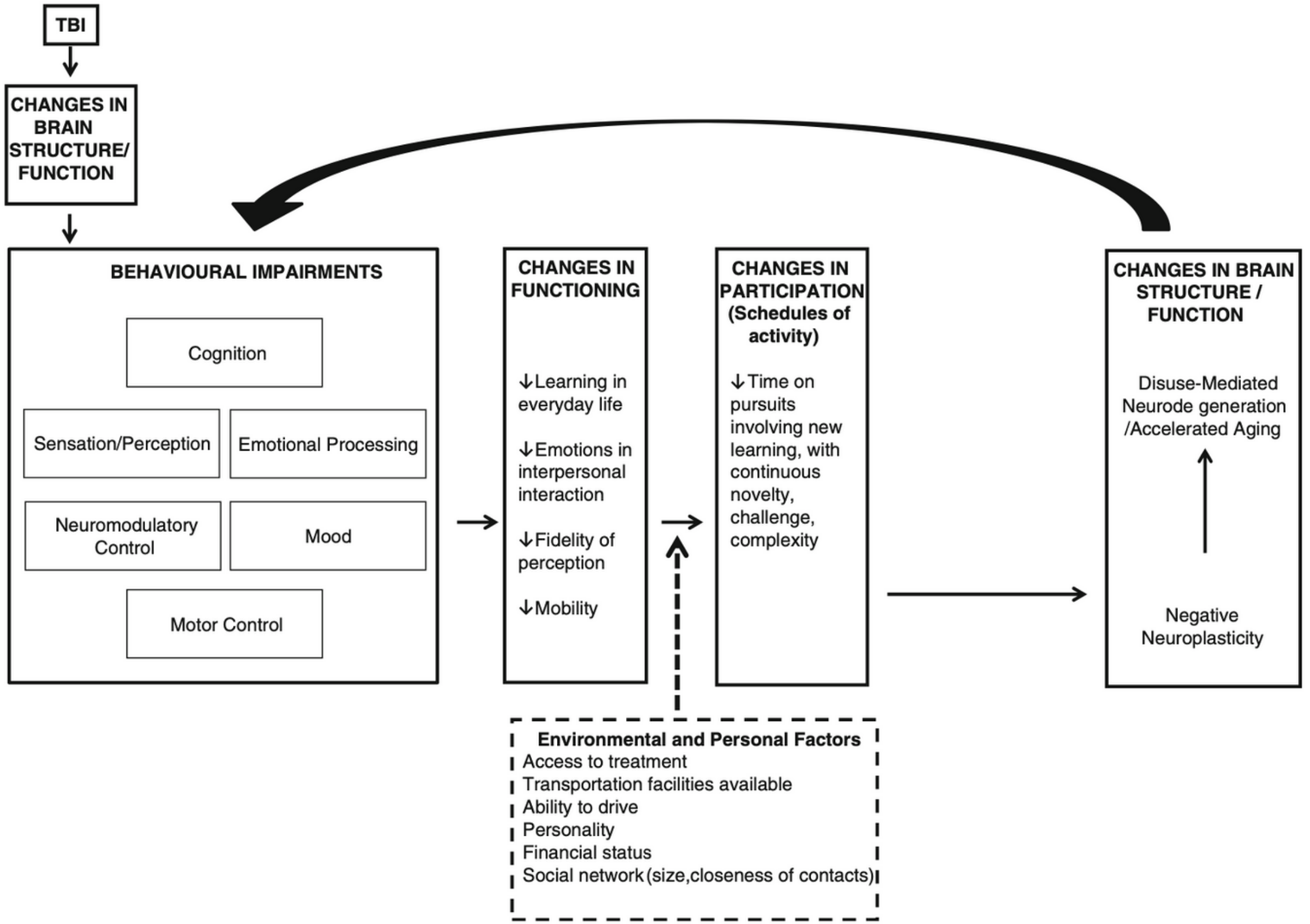

Fig. 1 Depiction of framework in context of TBI, showing neurodegeneration in chronic TBI secondary to negative learning/neuroplasticity. Figure adapted from Mahncke et al. (2006a), employing elements of the International Classification of Functioning, Disability and Health framework (World Health Organization 2013). *Arrows* between boxes (and within the "Changes in Brain Structure/Function" box) indicate postulated direction of causal influence, with *solid arrows* indicating direct influence and *dashed arrow* indicating modulator variables. In this adapted framework, various behavioural impairments caused by TBI result in deleterious changes in functioning and participation. As a result, negative learning creates negative neuroplastic brain changes resulting in neurodegeneration, which feed back to worsen behavioural impairments. Environmental and personal factors are posited to modulate the impact of changes in functioning on participation

To this end, we extrapolate from the "negative plasticity" framework of Mahncke et al. (2006a) that was proposed to explain cognitive decline in older adults. As described in greater detail later, their framework describes a self-reinforcing, downward spiral of negative brain plasticity whereby declining brain function is attributable to a combination of disuse (called "reduced schedules of activity"), reduced quality of sensory-perceptual processing, and weakened neuromodulatory control. In combination, these factors increase reliance on simplified cognitive processing at the expense of more complex processing capacity (called "negative learning"). These processing changes result in brain changes, which in turn result in further disuse, perceptual compromise and reduced neuromodulatory control.

Developmental Functional Neuroscience Melillo Method

Examination

The clinical manifestations of ABIs are varied and complex. Neurologists are often asked to evaluate patients after cardiopulmonary resuscitation and to help provide prognostication. The clinical examination is focused on brainstem reflexes, the presence of generalized myoclonus, and motor responses to noxious stimuli.[8] Additionally, it is important to assess for range of motion, skin breakdown, and muscle tone. In the acute care setting, physicians and families are interested in long term prognostication so that goals of care discussions can be established early.

We offer the most comprehensive functional exam possible for this population

1. Sensory Systems
2. Primitive Reflexes
3. Postural Reflexes
4. Core Stability
5. Vestibular System
6. Eye Movements
7. Cognition

Developmental Functional Neuroscience Melillo Method

Therapy

Therapy is defined by your exam

1. Sensory Systems
2. Primitive Reflexes
3. Postural Reflexes
4. Core Stability
5. Vestibular System
6. Eye Movements
7. Cognition

NeuroSolution

Developmental Blueprint

Cognition

Core Stability / Vestibular / Eye Movements

Sensory Systems / Primitive Reflexes / Postural Reflexes

Finding the Right Team is Key

Multidisciplinary Approach

Team Approach

1. HBOT - hyper acute and acute care especially. Dr. Harch, MD in New Orleans
2. Laser Therapy - www.neuro-solution.com
3. Developmental Functional Neuroscience / Chiropractic Care - Austin Center for Developing Minds
4. Stem Cells - Dr. Kenneth Proefrock, NMD in Surprise Arizona
5. Trauma Therapy for family & loved ones - Dr. Jeri LaVigne EdD, PhD in Atlanta
6. Functional Medicine / Nutrition

Kristin Hughes, OTR

Dr. Jazmin Isdale, DC

NeuroSolution

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SCAN ME

THANK YOU

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