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Acute Rehabilitation of Persons with Traumatic Brain Injury (TBI)

Jill Seale, PT, PhD, NCS

Physicaltherapy.com

August 31, 2016

Objectives

1) Describe the pathophysiology and at least two most common mechanisms of TBI.

2) Describe the common clinical presentations across the TBI spectrum from concussion/mild to severe TBI.

3) Examine the current medical management of acute TBI, and impact of medical management on rehabilitation intervention.

4) Identify at least three evidence based rehabilitation interventions for the most common impairments and activity/participation limitations in the acute phase of TBI.

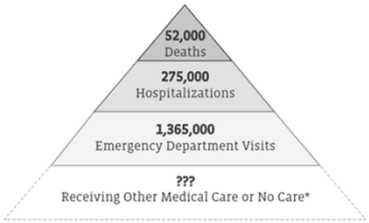
5) Differentiate the levels of care and appropriate referral across the continuum of care for persons with acute TBI.

TBI… what is it?

- "caused by a bump, blow, or jolt to the head or a penetrating head injury that disrupts the normal function of the brain"
- Severity ranges from mild to severe
- Most TBIs that occur each year are mild, commonly called concussions.
  - CDC, http://www.cdc.gov/traumaticbraininjury/get_the_facts.html

TBI Statistics

- 2.5 **million** sustain a TBI each year in US
- 52,000 die; 275,000 hospitalized, almost 1.4 million treated and released
- At risk for TBI
  - Males 1.5 times greater
  - 0-4 yr/old and 15-19 yr/old



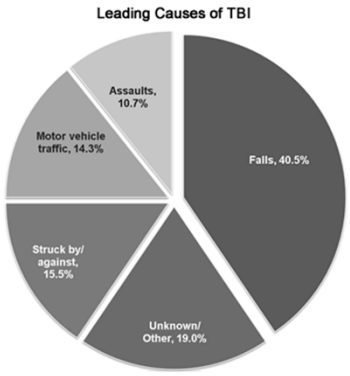
- Long Term
  - 5.3 million Americans with long term disability
  - Direct/indirect costs estimated at $60 million in US in 2000
    - CDC 2010

TBI by Age

- Children 0-4, adolescents 15-19, and adults aged ≥ 65 are most likely to have TBI
- Adults ≥ 75 have highest rates of TBI related hospitalizations and death

TBI by Gender and External Cause

- At every age, TBI rates higher in males
- Males 0-4 have highest rates of TBI ER visits, hospitalizations, and deaths



Mechanisms of Injury

- Direct Impact
- Severe acceleration/deceleration
- Blast injury
- Penetrating object

Primary v Secondary Damage

- Primary
  - Contusions
  - Hematomas
  - Diffuse axonal injuries
  - Penetrating injuries
  - Blast injuries
- Secondary
  - Increased intracranial pressure (ICP)
  - Hypoxia or ischemia
  - Seizures
  - Intracranial hemorrhage (ICH)
  - Electrolyte and acid-base imbalance

Neurological Rehabilitation, 2013

The Effects of TBI

- Neuromuscular/somatosensory
- Autonomic dysfunction
- Cognitive
- Psychological
- Behavioral
- Communication
- Visual/Perceptual
- Dysphagia
- Vision/Vestibular
- Cardiovascular

Neuromuscular/Somatosensory

- Paralysis/paresis
- Altered muscle tone and/or abnormal reflexes
- Poor coordination/ataxia
- Cranial nerve dysfunction
- Impaired balance
- Poor selective motor control
- Bowel and bladder dysfunction
- Dysphagia
- Loss of sensory function or hypersensitivity

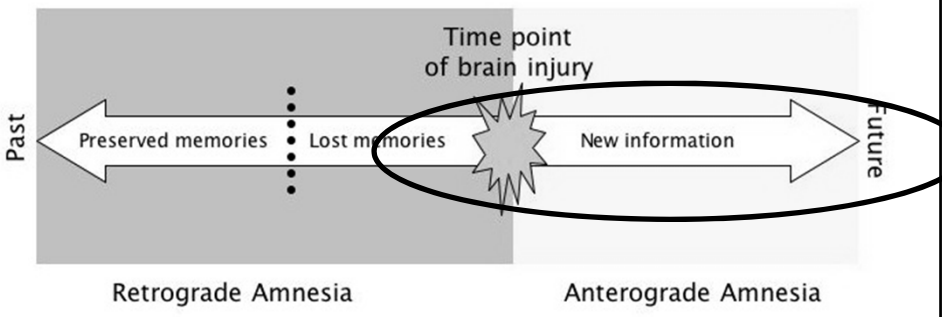
Autonomic Dysfunction

- Temperature elevations/Excessive sweating
- Hypertension
- Tachycardia/Tachypnea
- Pupillary dilation
- Extensor posturing
- Paroxysmal Autonomic Instability with Dystonia (PAID) and Paroxysmal Sympathetic Hyperactivity
- Management of symptoms pharmacologically
  - Meyer KS. *Surg Neurol Int*. 2014

Neuropsych

- Memory impairment
  - PTA
  - Short term memory
- Emotional changes
  - Behavior ranging from obtundity to hyperactivity
- Communication
  - Aphasias
  - Dysarthria
- Cognitive impairment
  - Attention/concentration deficits
  - Executive function loss
  - Loss of reasoning and/or abstract thinking
  - Poor problem solving

Posttraumatic Amnesia

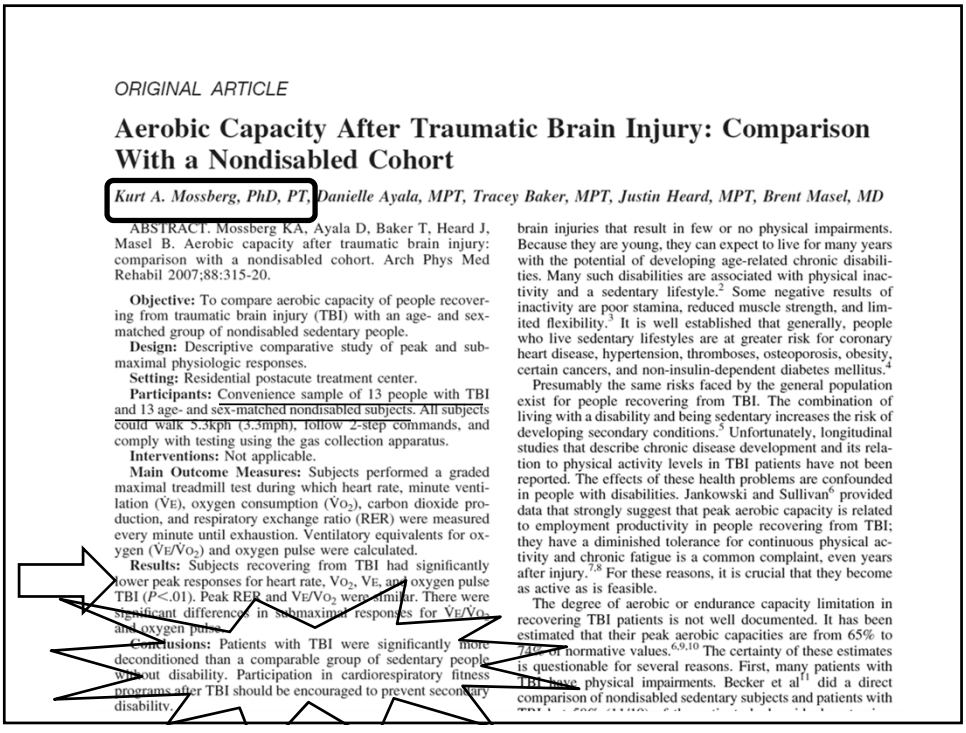


Visual, Perceptual, Vestibular

- Cranial nerve impairment that affects vision
- Visual field changes
- Visuospatial abnormalities
- Vestibular: peripheral or central
- Vestibulo-ocular: dizziness, vertigo, blurred/unstable vision, nausea, difficulty with busy environments
- Agnosia
- Apraxia

Cardiovascular

- Severe deconditioning
- VO2 Peak significantly decreased in all neuro dx
- NO RESERVE
- Most with neuro dx don't have the VO2 peak to meet demands for daily living of older adult
- Growth hormone insufficiency



ORIGINAL ARTICLE

Aerobic Capacity After Traumatic Brain Injury: Comparison With a Nondisabled Cohort

Kurt A. Mossberg, PhD, PT, Danielle Ayala, MPT, Tracey Baker, MPT, Justin Heard, MPT, Brent Masel, MD

ABSTRACT. Mossberg KA, Ayala D, Baker T, Heard J, Masel B. Aerobic capacity after traumatic brain injury: comparison with a nondisabled cohort. Arch Phys Med Rehabil 2007;88:315-20.

Objective: To compare aerobic capacity of people recovering from traumatic brain injury (TBI) with an age- and sex-matched group of nondisabled sedentary people.

Design: Descriptive comparative study of peak and submaximal physiologic responses.

Setting: Residential postacute treatment center.

Participants: Convenience sample of 13 people with TBI and 13 age- and sex-matched nondisabled subjects. All subjects could walk 5.3kph (3.3mph), follow 2-step commands, and comply with testing using the gas collection apparatus.

Interventions: Not applicable.

Main Outcome Measures: Subjects performed a graded maximal treadmill test during which heart rate, minute ventilation ($\dot{V}_E$), oxygen consumption ($\dot{V}O_2$), carbon dioxide production, and respiratory exchange ratio (RER) were measured every minute until exhaustion. Ventilatory equivalents for oxygen ($\dot{V}_E/\dot{V}O_2$) and oxygen pulse were calculated.

Results: Subjects recovering from TBI had significantly lower peak responses for heart rate, $\dot{V}O_2$, $\dot{V}_E$, and oxygen pulse TBI ($P<.01$). Peak RER and $\dot{V}_E/\dot{V}O_2$ were similar. There were significant differences in submaximal responses for $\dot{V}_E/\dot{V}O_2$ and oxygen pulse.

Conclusions: Patients with TBI were significantly more deconditioned than a comparable group of sedentary people without disability. Participation in cardiorespiratory fitness programs after TBI should be encouraged to prevent secondary disability.

brain injuries that result in few or no physical impairments. Because they are young, they can expect to live for many years with the potential of developing age-related chronic disabilities. Many such disabilities are associated with physical inactivity and a sedentary lifestyle.[2] Some negative results of inactivity are poor stamina, reduced muscle strength, and limited flexibility.[3] It is well established that generally, people who live sedentary lifestyles are at greater risk for coronary heart disease, hypertension, thromboses, osteoporosis, obesity, certain cancers, and non-insulin-dependent diabetes mellitus.[4]

Presumably the same risks faced by the general population exist for people recovering from TBI. The combination of living with a disability and being sedentary increases the risk of developing secondary conditions.[5] Unfortunately, longitudinal studies that describe chronic disease development and its relation to physical activity levels in TBI patients have not been reported. The effects of these health problems are confounded in people with disabilities. Jankowski and Sullivan[6] provided data that strongly suggest that peak aerobic capacity is related to employment productivity in people recovering from TBI; they have a diminished tolerance for continuous physical activity and chronic fatigue is a common complaint, even years after injury.[7,8] For these reasons, it is crucial that they become as active as is feasible.

The degree of aerobic or endurance capacity limitation in recovering TBI patients is not well documented. It has been estimated that their peak aerobic capacities are from 65% to 74% of normative values.[6,9,10] The certainty of these estimates is questionable for several reasons. First, many patients with TBI have physical impairments. Becker et al[11] did a direct comparison of nondisabled sedentary subjects and patients with

More issues

- For most with neuro dx, VO2 requirements increase secondary to gross motor insufficiencies
- Low level of fitness associated with increased mortality
- Decrease in available motor units → decrease in metabolically active tissue → decrease in oxidative potential

Classifications of TBI

- Most commonly determined by Glasgow Coma Scale (GCS)
  - 3 Domains: eye response, verbal response, motor response
- Mild: 13-15
- Moderate: 9-12
- Severe: 3-8
  - Adapted from: Advanced Trauma Life Support: Course for Physicians, American College of Surgeons, 1993

Glasgow Coma Scale		
Response	Scale	Score
Eye Opening Response	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	No eye opening	1 Point
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernible	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Point
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Point
Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points; **Severe Brain Injury** = 3-8 points		

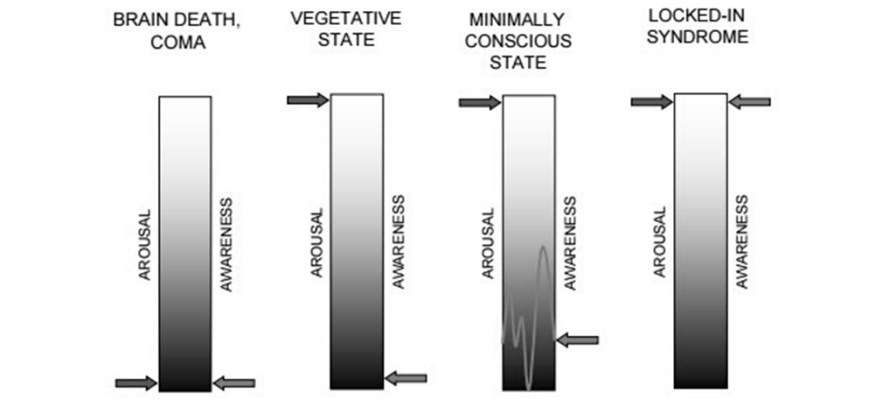


Fig. 1. Behavioral observation assesses two dimensions of consciousness: arousal and awareness. In brain death and coma, both dimensions are absent. In the vegetative state, arousal level is relatively preserved in the absence of signs of awareness. In the minimally conscious state, both dimensions are present although behavioral signs of awareness often fluctuate. In the locked-in syndrome, both dimensions are fully preserved despite complete loss of speech and motor functions.

Giacino JT, et al, *Progress in Brain Research*, 2009.

Level	Response
1	No Response
2	Generalized Response
3	Localized Response
4	Confused and Agitated
5	Confused-Inappropriate/ Non-agitated
6	Confused-Appropriate
7	Automatic-Appropriate
8	Purposeful – Appropriate, initiates and carries out with stand by assistance
9	Purposeful- Appropriate, initiates, requests assistance when needed
10	Purposeful –Appropriate, Modified Independent

Examination of Level of Consciousness

- Coma Recovery Scale Revised (CRS-R)
  - Score Range from 0-23
  - 6 subscales:
    - Auditory
    - Visual
    - Motor
    - Oromotor/verbal function
    - Communication
    - Arousal
  - Lower scores = reflex activity
  - Higher scores = cognitively mediated activity
    - Giacino J et al, 2004

Measurement	Mild	Moderate	Severe
GCS	13-15	9-12	3-8
Loss of Consciousness	< 30 min	30 min – 24 hours	> 24 hours
Posttraumatic Amnesia	0-1 day	>1 to ≤ 7 days	> 7 days

Neurological Rehabilitation, 2013

Mild TBI or Concussion

- Injury to head from blunt trauma or acceleration/deceleration forces
- Results in 1 or more of the following:
  - Confusion, disorientation, or impaired consciousness
  - Dysfunction of memory around time of injury
  - LOC < 30 minutes
  - Onset of observed signs or symptoms of neurological or neuropsychological dysfunction
    - CDC

Mild TBI or Concussion

Thinking/ Remembering	Physical	Emotional/ Mood	Sleep
Difficulty thinking clearly	Headache	Irritability	Sleeping more than usual
	Fuzzy or blurry vision		
Feeling slowed down	Nausea or vomiting (early on)	Sadness	Sleep less than usual
	Dizziness		
Difficulty concentrating	Sensitivity to noise or light	More emotional	Trouble falling asleep
	Balance problems		
Difficulty remembering new information	Feeling tired, having no energy	Nervousness or anxiety	

CDC

Trends and Patterns

- Impact of Income
- Mortality and outcomes improving?
  - Overall mortality rate in severe TBI not decreasing
  - Increased risk of death increased up to sevenfold
  - Increased risk for Alzheimer's disease
    - Roozenbeek B, Maas AIR, Menon D, Nat rev Neurol, 2013.
- TBI Model System Data at 5 yrs post injury
  - 21.7% dead
  - 32.3% need assistance
  - 29.1% dissatisfied with life
  - 55% unemployed
  - 57.8% with moderate to severe disability
  - 38.8% declined from earlier status to 5 yr outcome
    - Corrigan JD et al, 2014

Acute Management of TBI

Common Medical Complications

- Seizures (46%)
- Spasticity (57%)
- UTIs (47%)
- Hydrocephalus (with/without shunt) (38%)
  - Ganesh et al, *Arch Phys Med Rehabil*, 2013

Restrictions and Precautions

- Craniotomy/Bone flap
- Head of bed restrictions
- Mechanical ventilation/tracheostomy
- Weightbearing precautions
- Seizure precautions

Intracranial Pressure

- Edema, abnormal fluid dynamics or hematomas can result in increase ICP
- Normal ICP is 4-15 mmHg
- Severe increase in ICP can result in brain herniation
- Increased ICP associated with increased mortality and poorer outcomes

Unstable ICP Management

- Ventriculostomy open to drain
- Hyperventilation - decreases PCO_2 = vascular constriction = decreased space taken up in cranium = decreased pressure (ICP)
- Osmotherapy with Diuretics/Mannitol - decreases edema only in areas with intact cells and vasculature = decreased edematous pressure = decreased ICP

Drug Induced Paralysis

- Done in cases with increasing ICP
- Pentobarbitol: strong barbituate; acts on smooth and skeletal mm; need careful monitoring of cardio resp status
- Pavulon: blocks impulse at NMJ; affects skeletal mm only

Drug Interventions after TBI

Common BI Medications

- Anti-convulsants
- Neuro-stimulants
- Anti-spasmodics
- Anti-depressants/anti-anxiety
- Psychoactive or neuroleptic medications

RECOMMENDATIONS

- **Level 1**
  - Phenytoin is effective in decreasing the risk of early PTS in patients with severe TBI.
  - Valproate should not be used for early PTS prophylaxis.
  - Phenytoin, carbamazepine or valproate are ineffective in decreasing the risk of late PTS.
  - There are insufficient data to recommend routine PTS prophylaxis in patients with mild or moderate TBI.
- **Level 2**
  - Levetiracetam is an effective and safe alternative to phenytoin for early PTS prophylaxis.
  - Routine prophylaxis of late PTS is not recommended.
- **Level 3**
  - Levetiracetam should not replace phenytoin as a first-line agent for PTS prophylaxis based upon efficacy and economic analysis.
  - Carbamazepine is effective in decreasing the risk of early PTS.
  - The following CT scan findings may indicate the need for late PTS prophylaxis (anticonvulsant therapy for longer than 7 days post injury) (1):
    - Biparietal contusions (66%)
    - Dural penetration with bone and metal fragments (63.5%)
    - Multiple intracranial operations (36.5%)
    - Multiple subcortical contusions (33.4%)
    - Subdural hematoma with evacuation (27.8%)
    - Midline shift greater than 5mm (25.8%)
    - Multiple or bilateral cortical contusions (25%)
  - CT scan findings are superior to GCS score when used as a predictor for PTS development.
  - EEG does not currently have a role in evaluating the need for PTS prophylaxis.

Dept of Surgical Education, Orlando Regional Med Center, 2012

Neuro-stimulants

- Ritalin (Methylphenidate)
- Symmetrel (Amantadine)
- Other dopamine-enhancing agents
- Antidepressants
- Provigil (Modafinil)
- Ambien (Zolpidem)
- Revia (Naltrexone)

Antipsychotics or Neuroleptics

- Haldol
- Thorazine
- Zyprexa
  - Weight gain, diabetes, postural hypotension, sleepiness, hyperlipidemia, and dizziness
- Risperdal
  - Postural hypotension, insomnia, dizziness, weight gain
- Seroquel
  - Sleepiness, dizziness, postural hypotension
    - Hammond FM, Barrett RS, Shea T, et al. Arch Phys Med Rehabil. 2015.
    - Luaute J, Plantier D, Wiart L, et al Annal Phys Med Rehabil. 2016.

Acute PT Treatment

- Prevention of anticipated problems
- Patient/family education
- ONGOING Evaluation
- Early mobilization
- Appropriate environment
- Systems based approach

Start with Mental Status

- Increasing arousal and awareness
- Memory: orientation, implementation of memory strategies
- Cognition, speech, language: increased time for processing, cognitive rest, consistent approach to speech/language; graded cognitive exercise

Neuromuscular Interventions

- Increasing upright tolerance
  - Tilt table therapy
    - Krewer C, Luther M, Koenig E, Muller F., 2015
- Activities to maintain flexibility
- Management of altered tone
- Emphasize automatic tasks/movements
- Normalizing sensory input

Cardiovascular/Respiratory

- Increasing upright tolerance
  - Managing orthostatic hypotension
- Endurance training
- DVT prevention
- Positioning to optimize pulmonary function
- Activities to improve respiratory function

Integumentary System

- Thorough and ongoing examination
- Positioning to prevent pressure
- Pressure relieving beds and w/c systems
- Bowel and bladder management
- Protection against agitation/restlessness
- Family education

Exercise and TBI

Benefits above Improved Fitness

- Impact on cognition
- Impact on mood/behavior
- Impact on recovery

Exercise and Depressive Symptoms

- Increased prevalence of depression in persons with neurological disorders
- Exercise improves depression
  - Needed to be exercise that met PAGs
  - Moderate to vigorous intensity
  - 3-5 days per week
    - Adamson BC, Ensari I, Motl RW, Arch Phys Med Rehabil, 2015.

Exercise Maintenance after TBI

- Decreased score on BDI
  - Maintained improvement over time
- Increased physical activity
  - 48% with increased activity level at 6 months post
- Exercise greater than 90 minutes per week resulted in lower BDI and higher perceived QOL and mental health
- 52% of subjects were exercising greater than 90 minutes per week at 6 months
  - Wise EK et al, *Arch Phys Med Rehabil*, 2012

Cognition and Aerobic Exercise

- > 50% of TBI survivors still experiencing cognitive problems several years post TBI
- Vigorous training: 3 times/wk x 30 minutes on treadmill x 12 wks
  - supervised
- Improved cognitive function with aerobic training in TBI
  - Processing speed, executive function, overall cognition
- Aerobic exercises associated with physical adaptations and positive cortical functions like angiogenesis and neurogenesis
  - Chin LM et al, *Arch Phys Med Rehabil*, 2015

PM R. 2009 Jun;1(6):560-75.

Physical exercise and cognitive recovery in acquired brain injury: a review of the literature.

Devine JM, Zafonte RD.

Department of Physical Medicine & Rehabilitation, Spaulding Rehabilitation Hospital, Harvard Medical School, Boston, MA, USA.

Abstract

OBJECTIVE: Physical exercise has been shown to play an ever-broadening role in the maintenance of overall health and has been implicated in the preservation of cognitive function in both healthy elderly and demented populations. Animal and human studies of acquired brain injury (ABI) from trauma or vascular causes also suggest a possible role for physical exercise in enhancing cognitive recovery. DATA SOURCES: A review of the literature was conducted to explore the current understanding of how physical exercise impacts the molecular, functional, and neuroanatomic status of both intact and brain-injured animals and humans. STUDY SELECTION: Searches of the MEDLINE, CINHAL, and PsychInfo databases yielded an extensive collection of animal studies of physical exercise in ABI. Animal studies strongly tie physical exercise to the upregulation of multiple neural growth factor pathways in brain-injured animals, resulting in both hippocampal neurogenesis and functional improvements in memory. DATA EXTRACTION: A search of the same databases for publications involving physical exercise in human subjects with ABI yielded 24 prospective and retrospective studies. DATA SYNTHESIS: Four of these evaluated cognitive outcomes in persons with ABI who were involved in physical exercise. Three studies cited a positive association between exercise and improvements in cognitive function, whereas one observed no effect. Human exercise interventions varied greatly in duration, intensity, and level of subject supervision, and tools for assessing neurocognitive changes were inconsistent. CONCLUSIONS: There is strong evidence in animal ABI models that physical exercise facilitates neurocognitive recovery. Physical exercise interventions are safe in the subacute and rehabilitative phases of recovery for humans with ABI. In light of strong evidence of positive effects in animal studies, more controlled, prospective human interventions are warranted to better explore the neurocognitive effects of physical exercise on persons with ABI.

Dev Neurorehabil. 2008 Jul;11(3):236-40.

Exercise is brain food: the effects of physical activity on cognitive function.

Ploughman M.

Clinical Research, Rehabilitation Program, Eastern Health Authority, St John's, Newfoundland and Labrador, Canada. mploughm@mun.ca

Abstract

This commentary reviews selected biomedical and clinical research examining the relationship between physical exercise and cognitive function especially in youth with disability. Youth with physical disability may not benefit from the effects of exercise on cardiovascular fitness and brain health since they are less active than their non-disabled peers. In animal models, physical activity enhances memory and learning, promotes neurogenesis and protects the nervous system from injury and neurodegenerative disease. Neurotrophins, endogenous proteins that support brain plasticity likely mediate the beneficial effects of exercise on the brain. In clinical studies, exercise increases brain volume in areas implicated in executive processing, improves cognition in children with cerebral palsy and enhances phonemic skill in school children with reading difficulty. Studies examining the intensity of exercise required to optimize neurotrophins suggest that moderation is important. Sustained increases in neurotrophin levels occur with prolonged low intensity exercise, while higher intensity exercise, in a rat model of brain injury, elevates the stress hormone, corticosterone. Clearly, moderate physical activity is important for youth whose brains are highly plastic and perhaps even more critical for young people with physical disability.

Concussion and Exercise

- Rest versus exercise debate
- Neurometabolic cascade following mild TBI results in neurologic energy deficit
- Period of vulnerability to additional injury
- Led to extreme of absolute rest until all symptoms have resolved
- But is that the best strategy???
  - Wells EM, Godkin HP, Griesbach GS, *J Child Neurol*, 2016

Concussion Treatment and Recovery

- Physical Rest
  - Metabolic dysfunction post concussion
  - Risk of second injury
  - Unrestricted activity may worsen symptoms/delay recovery
  - But is complete rest best?
  - Unrestricted exercise in immediate acute phase may increase risk of subsequent injury and/or delay recovery
  - Some level of exercise may be beneficial once beyond acute injury stage
    - Broglio SP, Collins MW, Williams RM, Mucha A, Kontos A. *Clin Sports Med*. 2015

Concussion Treatment and Recovery

- Cognitive Rest
  - increased cognitive activities post concussion increase symptom recovery time and prolong recovery
  - Reduction in brain stimulating activity
  - "prolonged cognitive rest and reduction of school events have the potential to exacerbate symptoms or cause negative mental health issues"
  - Key is during acute phase; symptoms are guide
    - Broglio SP, Collins MW, Williams RM, Mucha A, Kontos A. Clin Sports Med. 2015

Concussion Treatment and Recovery

- Vestibular and Oculomotor Impairment
  - Occurs in approx 60% of athletes
  - Vestibular: peripheral or central
  - Vestibular issues: benign paroxysmal positional vertigo (BPPV), vestibulo-ocular reflex (VOR) impairment, visual motion sensitivity, balance dysfunction, cervicogenic dizziness, and exercise-induced dizziness.
  - Vestibulo-ocular: dizziness, vertigo, blurred/unstable vision, nausea, difficulty with busy environments
  - Vision Therapy
  - Pharmacological interventions
- May predict prolonged recovery
  - Broglio SP, Collins MW, Williams RM, Mucha A, Kontos A. Clin Sports Med. 2015

TBI Continuum of Care

- Acute Care
  - ICU
  - Neuro unit
- Inpatient Rehabilitation
- Post Acute Brain Injury Rehabilitation
  - Residential
  - Day program
- Outpatient Therapy
- SNF
- Long term care

Any Questions

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