Hypoxic-Ischemic Brain Injury: Movement Disorders and Clinical Implications

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Disclosures

• Carolyn Tassini, PT, DPT, CBIS, NCS – Nothing to disclose
• Kimberly Miczak PT, NCS – Nothing to disclose
Following this session:

- Participants will appreciate mechanisms underlying motor, cognitive and visual impairments following hypoxic-ischemic brain injury.
- Participants will utilize current evidence from movement disorder literature in developing treatment plan for individuals following hypoxic-ischemic brain injury.
- Participants will demonstrate the use of a framework to evaluate movement systems and prioritize treatment following hypoxic-ischemic brain injury.
- Participants will recognize the importance of transdisciplinary team in maximizing treatment and outcomes for individuals recovering from hypoxic-ischemic brain injury.

Introduction

- What is a hypoxic or anoxic brain injury?
  - Hypoxic-Ischemic brain injury
- Common Causes
  - Cardiorespiratory arrest
  - Respiratory failure
  - Drug overdose
  - Carbon monoxide poisoning
  - Drowning/strangulation
- Primary vs secondary injury
Background Info

- No national data available on the prevalence of HI-BI
- Peak frequency in males aged 60-years (r/t cardiac) and females in their late 20’s r/t suicide and parasuicide/self-harm attempts (Fitzgerald 2010)
- Can range from mild to severe
- HI-BI vs TBI rehab course:
  - slower progress
  - poorer outcome
  - more likely to DC to residential facility vs home (Fitzgerald 2010)

Mechanism of Injury

- Cardiorespiratory- hypoxia with ischemia
  - Reperfusion injury
- Pure anoxic or hypoxic
- Carbon monoxide
- **Delayed Post-Anoxic Encephalopathy
Cerebral Vulnerability with HI-BI

- Hippocampus – CA1 pyramidal neurons
- Superior brainstem – reticular formation
- Cerebellum – Purkinje cells
- Cerebral white matter at “watershed areas”
- White matter and subcortical structures supplied by distal branches of deep and superficial penetrating vessels
- Striatum
- Pyramidal neurons in cortical layers 3, 4, 5, and 6

Common Impairments Associated with HI-BI

- Cognitive impairments: memory, concentration, attention, executive dysfunction
- Visual impairments: cortical blindness, partial visual loss, visual inattention, visuospatial impairments
- Motor/sensory impairments: quadriplegia w/ cranial nerve impairment, quadriplegia, mono/para/hemiplegia, cranial nerve, myoclonus, seizures
- Speech/swallowing impairments: dysphagia, dyspraxia, dysarthria, dyslexia, dysgraphia
Addressing today:

- Cognition
- Visual-perceptual
- Ataxia
- Post Hypoxic Parkinsonism
- Dystonias and abnormal posturing
- Myoclonus

Cognitive Impairments
Cognitive Impairments

- Arousal & Awareness
- Attention
- Higher level cognitive function
  - Memory
  - Processing speed
  - Executive function

(Anderson 2010)

<table>
<thead>
<tr>
<th>Attention</th>
<th>Problems manifest as.....</th>
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<tbody>
<tr>
<td>Sustained Attention</td>
<td>Inability to persist with a task, unable to maintain focus of conversation</td>
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<tr>
<td>Selective Attention</td>
<td>Inability to maintain focus when competing stimuli present; may stop one task when interrupted by sound/visual distractor.</td>
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<tr>
<td>Divided Attention</td>
<td>Inability to dual task- Walking &amp; talking, keeping count of reps while performing task</td>
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(Anderson 2010)
### Cognitive Impairments

#### Memory

<table>
<thead>
<tr>
<th>Type</th>
<th>Problems manifest as…</th>
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<tbody>
<tr>
<td>Declarative (Semantic, episodic, autobiographical)</td>
<td>Disorientation, can’t recall facts, short-term &amp; long-term recall (prior PT session, previous education provided)</td>
</tr>
<tr>
<td>Implicit (Procedural, sensorimotor, emotional)</td>
<td>Unable to perform or recall well learned tasks; difficulty learning new routines/skills</td>
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#### Executive Function

<table>
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<tr>
<th>Function</th>
<th>Problems manifest as……</th>
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<tbody>
<tr>
<td>Judgement</td>
<td>Behaviors exhibited are not congruent with situation (attempt to walk unassisted when need significant assist)</td>
</tr>
<tr>
<td>Insight &amp; self-awareness</td>
<td>Cannot detect deficits in performance or current functional status; believes abilities exceed capacity</td>
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<tr>
<td>Planning &amp; organization</td>
<td>Poor time management, late for appointments, arranging transportation, doesn’t get refill on medication before running out</td>
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Anderson 2010
Clinical Implication

• Cognitive deficits will impact therapeutic intervention
• You must consider cognition when structuring plan of care
• Treatment may require varied or combined approaches:
  • Environmental modification
  • Remediation
  • Compensation

Visual Deficits
Visual Impairments

• Due to susceptibility of grey matter in visual cortex, especially Lamina 3
• Highly complex system with damage to any one area influencing visual skills
  • Visual impairments can implicate motor and cognitive skills
• Anton’s syndrome (anosognosia): lack of awareness of visual disturbances

Cain & Watson, 2000

Wertz, 2015
### Common Visual Impairments

- Cortical Blindness
- Visual perceptual deficits
- Balint’s Syndrome

- Fitzgerald et al, 2010
- Wilson & Harpur, 2003

### Cortical Blindness

- Due to damage of parietal-occipital lobe
- Will present with inability to differentiate objects, may report seeing shadows or light
  - Moving target easier/earlier to see than stationary
- May have some remediation initially, though usually long term persisting deficits (Hoyt & Walsh, 1958)
- Ability to compensate (low vision rehabilitation) will depend largely on cognitive function
  - Community orientation and mobility specialist

- Kerkhoff, 2000
- Margolin et al, 2007
Visual Perceptual Deficits

• Due to damage hippocampus, parietal/temporal/occipital lobes
• Can include visual spatial, visual memory, pattern recognition, visual scanning, visual attention
• Difficulty interacting with environment
  • Object misuse, groping, over-reliance on tactile
  • Decreased topographical memory
  • Facial recognition

Kerkhoff, 2000

Visual Perceptual Deficits

• Interventions can be Adaptive or Remediative:
  • Adaptive: prisms, patching, large print
  • Remediative: Scanning, self-monitor, situational strategies
  • Consultation with OT, neuro-psychology, neuro-optometry

Kerkhoff, 2000
Raymond, 2006

Image from: Wikipedia Commons, visual perceptual gaze human FOV
Balint’s Syndrome

• Due to damage in parietal lobes
• Presents as a triad of symptoms:
  1. Simultanagnosia: attending to one thing/feature when more than one thing/feature presented
  2. Ocular-motor apraxia (“psychic paralysis of gaze”): decreased visual gaze/attention to motor task
  3. Optic ataxia: lack of spontaneous eye movements

Kerkhoff, 2000

Image from: Alvarez, 2016

Balint’s Syndrome

• Interventions include:
  • Consult to OT, neuro-optometry and/or Neuropsychology
  • Multi-disciplinary approach suggested
  • Compensatory training
    • Scanning
    • Widening of visual field

Kerkhoff, 2000
Movement/Motor Disorders

Ataxia
Parkinsonism
Myoclonus
Dystonia

Post-anoxic Ataxia

- Due to cerebellar damage, especially Purkinje cells
- Incoordination of movement (limb and/or trunk)
  - Inability to coordinate activity of multiple muscles and adjust movements for effects at other moving joints (interaction torques)
  - Decreased control/timing of agonist and antagonist musculature leads to hypermetria
  - Anticipatory reactions for postural control
  - Manifests as inconsistent movement patterns
  - Hypometria and/or volitional rigidity as a compensation

Bastian, 1997
Godani, 2015
Post-anoxic Ataxia

- Interventions can include
  - Prevent secondary complications
  - Errorless learning (cerebellum involved in motor learning)
  - Decomposition of movements or reducing degrees of freedom
  - Task specific practice (higher repetition needed than other populations)
  - Core/proximal stability
  - Compensations - closer proximity to target, use of bracing/adaptive equipment

Fonteyn, 2014
Gillen, 2002

Post-anoxic Parkinsonism

- Due to damage of Globus Pallidus
- Most common delayed onset movement disorder after HI-BI
- Presents as:
  - Akinetic-rigidity, +/- tremor
  - Bradykinesia, Retropulsion
  - Gait pattern: festinating or shuffling, freezing
- Does not respond well to dopaminergic agents

Mangaraj, 2014
Schramm, 2004
Cho, 2002
Post-anoxic Parkinsonism

- Treatment:
  - Largely depends on level of cognition
  - “Typical” PD strategies
    - Auditory or visual cues
    - External inputs to drive movement—Treadmill, guided assistive device
    - Wider turns, counting when freezing occurs
    - Postural re-ed “Backwards dysequilibrium”

  Avanzino, 2016
  Radder, 2017
  Scheets et al, 2015

Post Hypoxic Myoclonus (PHM)

- Characterized by rapid, jerking muscle contractions, arrhythmimic and irregular in amplitude
- Pathophysiology unknown
  - Thought to be related to abnormal neurochemicals (in particular Seratonin)
  - May be of cortical, subcortical, brainstem or spinal cord etiology (Levy 2016)
- 2 Types
  - Acute Post Hypoxic Myoclonus (PHM)
  - Chronic PHM*
Chronic PHM

• AKA Lance-Adams syndrome
  • LAS or chronic PHM = myoclonus associated w/ dysarthria, apraxia, seizures or cognitive deficits
• More favorable prognosis than acute PHM
• Typically quiet during sleep, mild at rest and worse w/ startle or action especially intention
• Can be contractions (positive) or muscle tone lapses (negative), brief, involuntary, shock-like (Gupta 2016)
• Impairs function – self care, mobility, transfers etc.
• Often associated w/ signs of incoordination

Chronic PHM

• Diverse clinical presentation
• Possible triggers include:
  • Intention to move
  • Voluntary contractions
  • sensory stimuli
  • Emotional state
Chronic PHM

Pharmacologic Treatment

- Trial and error often applied
- Typically a combination of:
  - Sodium valproate
  - Clonazepam
  - L-5-HTP (L-5 hydroxytryptophan)
  - Levetriacetam (Keppra)

PT Interventions for PHM

- *PT treatment is adjunct to pharmaceutical*
- Assessing change in impairments
- Functional training (Polesin 2006)
- Postural stability
- Slow the movement
- Handling techniques
- Seating and positioning
- Adaptive equipment
- Environment modifications
PT Interventions for PHM

- Self-management
  - Cognitive strategies
  - Relaxation
  - Augmented movement/posturing
- Biofeedback to address:
  - Postural awareness
  - Alter motor recruitment
  - Facilitate motor recruitment
  - Tone/reflex inhibition
- **Reduce Anxiety**

Chronic PHM

**Surgical Treatment**
- Deep brain stimulator- B GPi
- Baclofen pump

VIDEOS with permission from:
Dystonia

- Characterized by: involuntary, abnormal muscle contractions that result in sustained abnormal postures, twisting, or both, and repetitive movements of body parts
  - Dystonia following HI-BI is a “Secondary Dystonia”
  - Classified by location: focal, multi-focal, segmental, hemi-dystonia, and generalized dystonia
- Caused by disruption of the basal ganglia
  - Globus pallidus
  - Putamen (Hawker 1990)
  - Cortico-striatal-thalamo-cortical loop
  - Dopamine changes
  - Decreased reciprocal inhibition
  - Impaired cortical activity during motor planning (Gilio 2003)
Treatment for Dystonia

Pharmacologic Treatment
• Benzodiazepines
• Anticholinergics
• Chemodenervation
• Baclofen

Surgical Treatment
• Inconclusive but emerging evidence for DBS

PT Interventions for Dystonia

• Seating and positioning
• Environmental modifications
• Adaptive equipment (Jahansahi 1991, Krack 1998)
• Contracture management
  • Stretching of agonist
• Managing secondary pain (Zetterberg 2008)

• Antagonist strengthening (Zetterberg 2008, Hakkinen 2004)
• Motor control and endurance
• Postural Orientation (Zetterberg 2008)
• “slow down exercises” (Yoshie 2015)
• Self management strategies
Integrated Case Examples

Framework to exploring cases

- History
- Identification of “relevant” information
- Functional task analysis
- Hypothesis generation
- Prioritization of treatment
- Evidence for treatment
Meet MH

- 50 y/o man s/p cardiac arrest in 2012
  - 7-8 months “unresponsive” per family
- 8-9 month acute rehab stay then DC to SNF
  - Family reports “he mostly stayed in bed”
- DC to home w/ his wife approx 1.5 years prior to presenting to OP therapy
  - Per family had course of PT at home but no services in the past year
  - Pt transitioned from his wife’s care to family care approx 1 year ago when he became too much for her to care for at home

MH

- PMHx
  - Unremarkable
  - Old right shoulder injury from HS football
- Current Medical
  - Seizures
- Course of care from this injury
  - Pt noted decline over the past year:
    - Per pt he was able to do short walks at home w/ his wife but hasn’t been able to walk for “a long time”
    - Previously able to dress and feed himself
    - Myoclonus has increased over the past year
MH

• Medications
  • Keppra
  • Klonopin
  • Xanax
  • Valium
  • Lipitor

MH

• Social
  • Prior worked FT in maintenance at apartment complex
  • Married but separated, estranged daughter
  • Living with family and hired CNA – 24 hour care
  • Multi story home w/ stairs to enter. Bedroom and bathroom on 2nd floor.
  • DME includes commode, urinal and standard WC w/ sling back and seat
  • Unable to transfer to shower due to bathroom setup
  • Current activity pattern: out of bed in AM, spending day in chair (WC or recliner) all on 2nd floor. Meals brought to him.
  • Dependent for dressing and feeding, requires assist w/ all transfers, unable to walk
What stands out as “Relevant” information

- Cardiac arrest
- Time in coma and time in hospital
- Change in status

Observe - WC <-> Mat transfer
Task Analysis- Sit pivot transfer

• Deviations that vary from “normal”
  • Static sitting: (positive) myoclonus present
  • Sitting posture
    • post pelvic tilt, “slouched” posture, UEs not engaged, poor foot contact with floor
  • Decreased ability to attain initial condition for task
    • Neutral pelvis, neutral spine, arms to supportive position
    • Difficulty sustaining unsupported posture due to myoclonus
  • Execution unsuccessful due to myoclonic intrusions

Generation of Hypotheses

• Myoclonus interferes with ability to perform at optimal level
  • Doesn’t allow for isolated movement, which may or may not be intact
  • Limited ability to determine motor planning and motor execution
  • Potential of bradykinesia

• Would like to evaluate cognition, vision, emotional status
  • Executive dysfunction (externally/internally distracted, hypervigilant/responsive to environment)
  • Vision- intact
  • Emotional status- signs of depression, frustrated with situation
Prioritization of treatment

• Patient’s goal “to be able to walk”
• Barriers to goal attainment:
  • Myoclonus
  • Selective motor control
  • Cardiovascular fitness
  • ROM
• Prioritized interventions to address myoclonus management
• Secondarily
  • Selective motor control
  • Functional motor planning

Treatment Approach Utilized

• Education
• Self-management techniques
  • Deep breathing and relaxation
• Postural re-education and training
  • Supported training
  • Unsupported
  • Anticipatory vs reactive postural control activities
• Body weight support training
• Functional training
  • Closed chain work
• Caregiver training
• Referral: Occupational therapy, neuropsychology
Education

- Critical to inform patient that myoclonus is not unusual following his injury
- Educate role and likely outcomes with PT
  - Adjunct to medications
  - Expect long term deficits
- Educate treatment plan and factors impacting myoclonus
  - Anxiety
  - Mood
  - Triggers

Awareness & Engagement

- Patient engagement!
  - May increase activity in several cortical regions
  - Improve neuroplasticity
- Skills to improve engagement
  - Build rapport and trust
  - Use motivational interviewing
  - Client education process
  - Empowerment strategies

Danzl 2012
Self-Management Techniques: Relaxation & Deep Breathing

- Duckett & Kramer (1994) reported on case using biofeedback and relaxation techniques to manage myoclonus
  - Relaxation techniques listening to tape 3x/day
  - Relaxation techniques utilized prior to transfers and standing
  - EMG on temporalis for biofeedback
- Khoury et al. (2013) found mindfulness-based therapy (MBT) to be effective for treating psychological disorders including anxiety and depression
- Deep breathing techniques work by inhibiting the sympathetic nervous system and can be useful to decrease anxiety and stress

Treatment Concepts

- Slow the movement
  - Cuing the patient to perform actions in “slow motion” improved control and decreased myoclonus (Polesin & Stern 2006)
- Clinician awareness of touch and environment
  - Reflex myoclonus can be triggered by either or both of these stimuli
  - Firm touch and intentional contact
  - Manage environmental sounds
  - Manage environmental distractions
Postural Re-Ed & Body Awareness

- Postural and equilibrium control are required for balance (Massion & Woollacott 1996)
- Balance system requires adequate sensory input, efficient CNS processing, and strong effector system of muscles and joints (Horak 1989)
- Feedback vs feedforward loops

Figure 1. Determinants of functional balance.
Functional Interventions

• Functional training combined with slow movement training improved transfer and gait status (Polesin 2006)
• BWS training – allow for practice of task specific training
  • Allow for reciprocal gait pattern
  • Provides safe environment
  • Increased duration practice
  • Increased number of repetition

Treatment Videos
Meet AP

- 26 year old woman w/ history of asthma was found down in respiratory distress in May 2015
- PEA upon arrival of EMT’s
- Treated w/ hypothermia protocol
- Hospital course: vented for airway protection, seizures, dysautonomia, leukocytosis, sepsis, fevers
  - Rehab course: B adhesive capsulitis, B ulnar neuropathy
- Transferred to acute rehab and DC home Sept 2015
- Presented to OP in September 2015

AP

- PMHx: asthma, drug abuse
- Social
  - College senior- Neuroscience degree – 1 final before graduation
  - Independent prior to event
  - Current: living with mother in 2-story home with 1 step to enter
Examination Findings (Acute Rehab)

- Cognition/communication:
  - Non-verbal initially
  - Follows simple verbal command i.e. stick out tongue, move arm

- Functional status:
  - Total A for bed mobility and all transfers

- Motor exam:
  - Myoclonus noted in BUE at rest and w/ activity
  - Difficult to isolate movements BUE/LE

AP

- Medications
  - Keppra
  - Baclofen
  - Trazodone
  - Albuterol
  - Dextroamphetamine
  - Lacosamide
  - Protonix
What stands out as “Relevant” information

- Respiratory distress followed by PEA
- Central and peripheral nervous system involvement
- Young age
- Drug history
- Less than 6 months from injury
- High level of education and independence

Observe: Ambulation
Task Analysis - Ambulation

- Deviations that vary from “normal”
- Initial conditions
  - Postural deviations:
    - flexed trunk (notably T&C spine), immobile UE’s w/ atypical hand/finger posture,
    - posterior CoM,
    - + myoclonic jerks
  - Initiation of movement:
    - dependent for weight shift, swing limb en bloc
  - Execution:
    - limb advancement is dependent on weight shift vs hip flexion, limb advancement occurs in rotation plane vs flexion
    - lack of postural reactions
    - limited muscle activation to propel gait
    - slow speed

OP Gait (~2 mos rx)
Task Analysis - Ambulation

- Deviations that vary from “normal”
- Initial conditions
  - Postural deviations:
    - flexed trunk (notably T&C spine), immobile UE’s w/ atypical hand/finger posture
    - Right trunk shortening
  - Initiation of movement:
    - Decreased wt shift laterally, swing limb en bloc
  - Execution:
    - limb advancement is dependent on weight shift (pt wt shifting w/ PT providing balance), limb advancement progression from rotation plan to flexion of hip
    - Asymetrical stance time
    - lack of postural reactions
    - limited muscle activation to propel gait
    - slow speed

Generation of Hypotheses

- Co-contractions (dystonia) lead to stiffness and rigidity of movement
  - Decreased ability to dissociate lumbo-pelvic region
  - Potential difficulty with isolation of movements elsewhere due to movements en bloc
  - Spinal mobility (ROM) and postural dyscontrol major contributors to movement patterns
  - Potential underlying strength deficits (hip extensors, hip abductors)
- Would like to further assess ROM, presence of bradykinesia (as a differential of movement pattern)
Prioritization of treatment

- Patient’s priority: Myoclonus, Walk & move normally
- Barriers to goals:
  - Dystonia/rigidity – led to decreased balance and postural reactions
  - Range of motion – PF contracture led to impaired gait and balance
  - Strength – hips! Needed to help w/ dystonia and ROM
  - Anxiety- exacerbated myoclonus, fear!!

Treatment Approach Utilized

- Deep breathing, relaxation, mindfulness training
- Fall exposure and falls training
- Standing – weight bearing and stretching program
- PNF – to encourage new and multi-plane movement patterns
- Functional positions – quadruped > tall kneel > half kneel
- Coordination training – variable speeds and positions
- Family and patient education/training
- Referral: OT, SLP, Neuropsychology
Dystonia

- Scarce literature for PT intervention, nothing for generalized dystonia
  - PT targets secondary deficits r/t dystonia

- Biofeedback: limited evidence that biofeedback alone is effective treatment for cervical dystonia (CD) BUT can be beneficial combined with relaxation and exercise (De Pauw 2014)
- Combination of exercises and relaxation equally effective as biofeedback training in reducing tone of SCM, improve ROM and head position (Jahanshahi 1991)
- Self-relaxation techniques and “slow down” exercises in focal hand dystonia (van Vugt 2014)

Dystonia

- Quieroz 2012 examined rehab protocol of kinesiotherapy, motor learning exercises and FES and Botox compared to treatment of botox® alone for pts with CD
  - Ex/btx® group improved on severity, pain and disability subscales of TWSTRS vs improvement on severity subscale only for botox
  - Ex/btx® group improved on all 3 domains (physical functioning, role-physical, bodily pain) of SF-36 vs only role-physical and bodily pain with btx alone
  - Ex/btx® group improved mental health on SF-36 (vitality, social functioning and mental health) with no significant diff in the btx alone group
- Kinesio Taping improved pain and sensory discrimination in individuals with focal hand dystonia compared to sham taping (Pelosin 2015)
- “Sensory tricks” to alleviate spasms
**Falls exposure**

- Falls exposure- anxiety
  - A form of acquired inhibition that suppresses a fear response
    - Common treatment in treatment of anxiety disorders, panic disorders, PTSD
  - “In exposure therapy the patient is repeatedly exposed for prolonged periods to a fear of object or situation in the company of a supported therapist and hence in the absence of aversive consequences” Davis et al. (2006)
  - Lovibond (2004) notes that extinction training is an automatic, unconscious and low-level process

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**Weight bearing & Stretching**

- Standing- weight bearing & stretching
  - Long-term stretching may be recommended for clients at risk or with contractures of neurologic origin
  - Katalinic et al. (2011, 2010) and Harvey (2017) found that regular stretches do not produce clinically important changes in pain, joint mobility, spasticity or activity limitation
  - Stretching not ruled in or out for individuals with ABI
  - **Not followed for stretching >6 months or combined with other intervention i.e. motor training or botox**
Multi-plane movements

• PNF – based on maximal stimulation of proprioceptive and external (touch, verbal, visual) receptors aiming at activation of impaired structures of the nervous system
  • Principles: slow approximation, timing for emphasis, bilateral reciprocal patterns
  • Mostly closed chain
• Mirek et al (2015) found improvement in static and dynamic balance w/ PNF rx in individuals with HD (BBS, FRT, Pastor test)

Functional positions

• Quadruped, tall and ½ kneel
  • Allow for targeted stability
• Core stability training - necessary for stability of spinal column, resist perturbations, and provide stable base for balance (Wilson 2005)
Coordinated/Timing

• Bilateral movements improve inter-limb coordination and coupling of muscles to improve motor performance in hemiparesis (Stewart 2006, Cauraugh 2005)
• Post-stroke hemiparesis shows atrophy of type II muscle fibers (Patten 2004)
• Rehabilitation requires training of both resistance exercises and skill (motor control exercises)
• Patten (2013) compared functional task specific training (FTP) to hybrid (FTP and power training) and found greater improvement in function and 6-mos retention w/ hybrid compared to FTP

Treatment Videos
Meet RF

- 33 y/o man admitted to OSH 1/2014 after being brought in by EMS for SOB and change in MS. He was aggressive and not following commands. Nonverbal. Breathing decompensated to status asthmaticus and hypercapnic respiratory failure requiring intubation in ED
- BP on initial eval 224/122
- Shortly after intubation pulses dropped and progressed to PEA code requiring resuscitation
- UDS positive for cannaboids, Negative EtoH

RF

- > 1 1/2 month hospital course c/b VDRF w/ vent associated PNA and aspiration PNA both due to MRSA, rhabdomyolysis, AKI with progression to ATN
- Trach and PEG on 2/7.
- LUE celluitis and edema, RLE DVT, RUE SVT, agitation requiring 1:1, falls x2
- Note pt assumed to have anoxic encephalopathy without evidence on CTs or MRI of brain. Unclear as to timeline of PEA code or time before pt resuscitated
RF

• PMH: Asthma, ? HTN
• Social: Prior to injury was living with girlfriend, limited activity pattern
  Per sister, he “slept all day and played videogames at night…..he’s a tech guy.”
• D/C disposition: To mother’s house which has 3 stairs to enter

What stands out as “relevant” information

• Primarily respiratory injury followed by cardiac complication
  • In hospital care for cardiac condition, leading to quicker management
• Age
• Elevated BP
• Many medical complications
  • Pneumonia
  • Increased time in acute care prior to rehab
• Imaging negative
• Limited activity pattern
Observe: Ambulation

Task Analysis- Ambulation

• Deviations that vary from “normal”
• Initial conditions
  • Standing flexed at knees and hips, body tendency towards flexion, arms in mid-guard position, no rotation at body or head
• Execution
  • Inconsistent movement patterns, unsteadiness in stance, variable BOS, postural dyscontrol
  • Generalized slowness of movement
• Termination
  • Grading of amplitude
Generation of Hypothesis

- Dyscontrol of trunk and limbs leading to inefficient and inconsistent movement patterns
- Balance is greater concern than kinematics
- Proximal stability impaired influencing distal control
- Bradykinesia influences balance reactions

Prioritization of Treatment

- Ability for family to provide care at home
- Barriers:
  - Postural Control (Fall risk)
  - Selective motor control & coordination
  - Speed of movement
  - Cardiovascular fitness
Treatment approach utilized

- Postural control
  - Sitting with progression to standing balance
  - Static to dynamic tasks
  - Anticipatory to reactionary postural control
- Treadmill
  - External cuing for gait speed, repetition, endurance
- Selective motor control
  - Functional postures
  - Management of degrees of freedom
- Task specific training

Treadmill training

- Treadmill training for ataxia WITH or WITHOUT body weight support has demonstrated
  - Reduced assistance for ambulation
  - Increased walking distance
  - May be due to motor learning, balance improvements or other unknown reasons

Marquer, 2014
Vaz DV, 2008
Task Specific Training

- Assessment of performance to allow for collaborative problem solving of therapist and client
- Systems model of motor control, which considers
  - Person
  - Environment
  - Task

Gillen, 2002
Gillen, 1999

Case Summary

- Understand the pathology and recognize common clinical presentation to frame your evaluation
  - Motor, cognition, vision
- Use of task analysis combined with pathophysiology may help generate hypothesis and prioritization of treatment
  - Recognize that movement disorders often do not present in isolation
- Limited literature necessitates insights from comparable diagnoses/disorders
Take Home Messages

- HI-BI is a diffuse injury w/ diverse clinical presentation
- Incidence of HI-BI may be increasing due to improved emergency medicine abilities
- May be seen as secondary complication to another injury i.e. primary TBI, cardiac arrest, etc.
- Onset of movement disorders may be acute, subacute or chronic
- Unfortunately there lack of literature in this specific population must use complimentary literature
  - Not fully generalizable to persons with HI-BI
- Multisensory component to many of the movement disorders with HI-BI
- Transdisciplinary approach is beneficial to maximize outcomes

THANK YOU!

QUESTIONS?
References


References

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References


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