Depression Following Traumatic Brain Injury: 
A Model for Care and Treatment

Massachusetts Brain Injury Association, 2019

Frank D. Lewis, Ph.D.
Medical College of Georgia
NeuroRestorative Research Institute
Organization of this talk

I. Prevalence of Depression
II. Understanding Depression
III. Impact on Outcomes
IV. Neurophysiological Mechanism of Depression
V. Model for Treatment
I. PREVALENCE/INCIDENCE OF DEPRESSION FOLLOWING TBI
Magnitude of the Problem

• This disorder has always captured people’s interest
  – Millions of people have depressive disorders
  – Economic costs amount to more than $80 billion each year
  – Human suffering is incalculable
Magnitude to the Problem

• Incidence in the United States 5.4% for ages 12 and older (Center for Disease Control, 2014)
• Five to six times greater for TBI survivors
• Prevalence rate ranges from 30% to over 50% two years post TBI.
Magnitude of the Problem

“At some point, to some degree, for some length of time, depression is experienced by many” (APA).

Over ones lifetime, 20% of the U.S. population will be affected.
Magnitude of the Problem

“In this sad world of ours sorrow comes to all and it often comes with bitter agony. Perfect relief is not possible except with time. You cannot now believe that you will ever feel better. But this is not true. You are sure to be happy again. Knowing this, truly believing it will make you less miserable now. I have had enough experience to make this statement”. *Abraham Lincoln*

“Depression, my *Black Dog*, my constant companion”. *Winston Churchill*
II. Understanding Depression
Major Depressive Episode
Period of 2 weeks with at least 4 symptoms

- Decreased interest or pleasure in most or all activities.
- Weight loss or gain (5% of body weight)
- Insomnia or hypersomnia
- Psychomotor agitation or retardation
- Fatigue
- Diminished ability to concentrate or think
- Feelings of worthlessness or excessive guilt
- Recurrent thoughts of death or suicidal ideation.
Depression vs TBI Neurogenic Symptoms

Neurogenic cause by injury (frontal lobes) but not reflective of mood disorder:

• Difficulty concentrating
• Flat affect
• Lack of motivation
• Irritability
• Lack of initiation
• Fatigue
Depression vs TBI Neurogenic Symptoms

• Mayo Portland Adaptability Inventory Version 4 (MPAI-4)
• 29-items well defined symptoms of TBI ranging from vision to return to work potential.
MPAI-4 Neurogenic Symptoms

• Fatigue
• Attention/concentration
• Irritability
• Initiation
<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
<th>Details</th>
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<tbody>
<tr>
<td><strong>3 Moderate</strong>&lt;br&gt;Problem: interferes with activities 25-75% of the time</td>
<td>Depression is sufficiently severe to interfere with many activities including vocational activities or school attendance. As for level 2, adults, children and adolescents at this level usually appropriately receive a psychiatric diagnosis.</td>
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<tr>
<td><strong>4 Severe</strong>&lt;br&gt;Problem: Interferes with activities more than 75% of the time</td>
<td>Depression is disabling and those at this level may require hospitalization. Examples at this severe level would be an inability to work, attend school, or almost complete social isolation because of depression. Individuals who are actively suicidal would be rated at this level.</td>
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III. IMPACT OF DEPRESSION ON REHABILITATION OUTCOME
Depression and TBI
Lewis and Horn, 2017

Study of 820 outcomes for TBI survivors along the depression continuum.

As determined by MPAI-4 assessment:
• 261 not depressed
• 235 mildly depressed
• 223 moderately depressed
• 101 Depressed
Impact of Depression

Participation T-scores Admission to Discharge by Depression Group

Level of Disability

Not Depressed | Mildly Depressed | Moderately Depressed | Severely Depressed
---|---|---|---
54.46 | 55.67 | 57.26 | 61.94
47.18 | 49.29 | 50.28 | 53.82

Admission | Discharge
Depression and TBI
Lewis and Horn, 2017

Conclusions:

1. Depressed participants experienced greater disability at admission and discharge than those not depressed.

2. Remediation of depression symptoms greatly improved outcomes.

3. Assessment and treatment of depression is a critical component of TBI rehabilitation.
IV. Neurophysiological Mechanism of Depression
Neural Activity and Depression

• Pet scans show depressed people have less neural activity in the prefrontal cortex but more neural activity in the limbic system.
• Prefrontal cortex helps to inhibit negative emotions generated by the limbic system.
• Hippocampus is vulnerable to the effects of stress. The volume of the hippocampus is decreased in depressed people.
Neurotransmitters and Depression

Low levels associated with depression.

- Serotonin: Sleep, mood, aggression, eating, sexual behavior.
- Norepinephrine (NE): stress, fight/flight, focus, sustained alertness.
- Dopamine: ability to sense pleasure, focus.
- Gamma-aminobutyric acid (GABA): low levels result in an increase firing of neurons that trigger anxiety.
TREATMENT FOR DEPRESSION FOLLOWING TBI
Antidepressants

- Antidepressants can be an important part of the treatment program.
- Antidepressants act by increasing neurotransmission (NT function)
- Protect against “Bad Plasticity” loss of dendritic spines and loss of synapses in the hippocampus and prefrontal cortex.
- Different medications work on different neural pathways, trial and error often necessary to find most effect medication.
- Talk with physician about side effects.
Treatment: Antidepressants

Tricyclic or Selective Serotonin Reuptake Inhibitors
SSRIs = Prozac, Zoloft, Paxil, Wellbutrin.
Cognitive Behavior Therapy with TBI

Key elements include:

• Focus on past schema – how one perceives themselves interacting with their world.
• Educational – enhancing skill set to deal with circumstances
• Directive – restructuring how we think “how we think is everything”
• Structured – focused dealing with important issues in the “here and now”.
• Problem-oriented – clarify the problem.
• Solution-oriented – coping strategies for each problem.
Cognitive Behavior Therapy with TBI

Determining who may benefit.

• Evaluation of comprehension skills
• Compensation for short term memory deficits okay.
• Motivation vs initiation skills.
Cognitive Behavior Therapy
Case Example - Henry

• 24 year-old male w/ history chaotic childhood and drug and alcohol abuse.
• High on drugs drove into a tree at 80 mph.
• One year of acute rehabilitation.
• Residual deficits: 1) mobility, 2) dysarthria, 3) impulsivity, 4) short-term memory, 5) social interactions.
• Strengths: 1) resilient 2) family= grand father aunt, 3) faith
Case Example: The Process

• Patient history: Finding the positives.
• Reframing from negative to positive “in spite of all that you were still able to ______.”
• Creating life time line – events that “added to or detracted from” since of well being.
• Questioning to define the problem – focus on first addressable problem.
Case Example

- Building the case for strength and resilience.
- What does success look like – reframing.
- Memory compensation techniques.
- Real world performance – evidence that negative appraisals are inaccurate.
- Diet and exercise.
- Scaffolding – building on successes.
- Faith – courage to take risks.
- Generalization and maintenance – home work assignments.
Exercise and Diet

Exercise

• Common manifestation of depression is low energy.
• Exercise has been shown to improve energy levels and reduce symptoms of sadness, anxiety, irritability and hopelessness. (Berger & Motl, 2000, Scully et al. 1998).
• Start slow - set-up for success.
• Regular moderate aerobic exercise for 20 – 30 minutes 5 days a week. Lifestyle.
• Neurophysiological effects: Balancing levels of NT resulting in: reduced muscle tension, improved sleep, reduced levels of stress hormone cortisol (Acevedo & Ekkekakis, 2006)
Exercise and Diet

Brain Derived Neurotrophic Factor (BDNF)

• Chemical in the nucleus of neurons important for learning and memory.
• Facilitates neuroplasticity - specifically dendritic spreading.
• Stress/Pain decreases BDNF.
• Decrease in BDNF results in neurons shriveling, synapses get disconnected, in turn leads to depression.
• Exercise increases the levels of BDNF.
• Protective against depression.
Exercise and Diet

Diet

• Appetite disturbances common = weight gain (medication side effects) or weight loss.
• Food avoidance
• Eating for comfort
  – Carbohydrates and sugar (junk foods) temporarily increase levels of dopamine and serotonin, followed by crash.
Foods that promote good brain chemistry (GABA, Serotonin, Norepinephrine)

- Avocado
- Eggs
- Peaches
- Granola
- Grape juice
- Peas
- Sunflower seeds
- Almonds
- Cottage cheese
- Milk
- Shredded wheat
- Soybeans
- Turkey
- Lima beans
- Yogurt
Vitamin D

- Some evidence that Vitamin D supports cognitive function. High concentrations in the hippocampus an area of the brain important for short term memory. (Eyles et al. 2005, Levenson & Figueiroa, 2008).
- 400 IU per day
Omega-3 fatty acids

- More than 160 studies about food's affect on the brain were analyzed.
- Omega-3 fatty acids -- found in salmon, walnuts and kiwi fruit -- provided benefits, including improving learning and memory and helping to fight against such mental disorders as depression and mood disorders, schizophrenia, and dementia.
- Omega-3 fatty acids support synaptic plasticity and seem to positively affect the expression of several molecules related to learning and memory.
Putting it together

Research tells us:

1. Depression may respond to several approaches including cognitive, interpersonal and biological/pharmacological therapy.

2. Cognitive Behavioral Therapy (CBT) combined with pharmacological therapy (when necessary) biological therapies have been shown to be more effective than either alone (Hollon et al., 2002).

3. CBT important to prevent relapse and has no side effects (Gilson et al. 2009).

4. Proper diet and exercise add to well being and give the individual control.
“Everybody is a genius. But if you judge a fish by its ability to climb a tree, it will live its whole life believing that it is stupid.”

– Albert Einstein
The End

Questions?

Frank.lewis@neurorestorative.com