

Weill Cornell Brain and Spine Center

**Concussion Recovery and Prevention:
Neuroplasticity, Normal Concussion Course
vs. Complicated Course**

Roger Hartl, M.D.

Kenneth Perrine, Ph.D.

Weill Cornell Medical College

Financial Disclosures

- Dr. Hartl receives compensation as a consultant for the New York Giants
- Dr. Perrine receives compensation as a consultant for the New York Jets and the New York Islanders

© Original Artist
Reproduction rights obtainable from
www.CartoonStock.com

© Steve Moore Distributed by Universal Uclick via CartoonStock



search ID: smbo0121

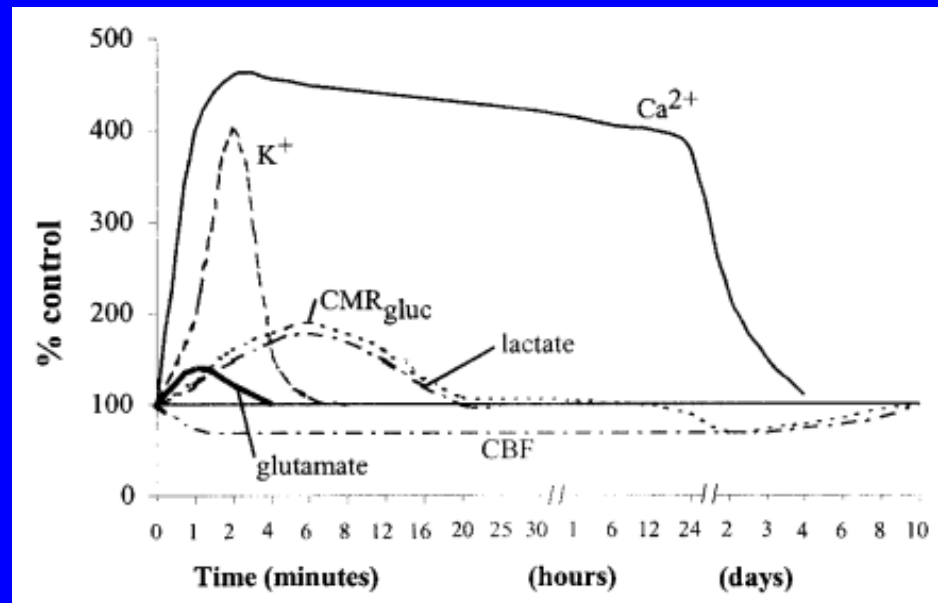
"Voila! ... Concussion-proof!"

Topics

- Neurological substrate of recovery
- Neuroplasticity concepts
- Normal concussion course
(recovery)
- Complicated concussion recovery

Neurologic Substrate of Recovery

- Biochemical cascade:
 1. Immediate release of neurotransmitters
 2. Acute hypermetabolism
 3. Refractory hypometabolism



Biochemical Cascade of Concussion: Stage 1

Immediately following concussion:

- Disruption of neuronal membranes
- Stretching of axons
- Wave of neural excitation
- Followed by wave of neuronal suppression
 - Could explain loss of consciousness, amnesia, being dazed
- Reduced blood flow to the brain

Biochemical Cascade of Concussion: Stage 2

Hypermetabolism follows Stage 1:

- The brain tries to restore balance, requiring use of energy (glucose)
- Use of energy results in lactate production
- Blood flow to the brain remains reduced
 - Problematic because blood supplies glucose to the brain

Biochemical Cascade of Concussion: Stage 3

- Glucose use is decreased by 1 day post injury
 - Low levels of glucose use (hypometabolism) can remain for days to weeks
 - Reduced blood flow may last for days
- * Post-injury hypometabolism not related to *
level of consciousness

Neuroplasticity

What is neuroplasticity?

- Structural or functional reorganization of nervous system
- Important in recovery from brain injury: stroke, surgery, TBI

Neuroplasticity & Concussion

- “Concussion” generally does not include major structural damage to the brain
- However, changes at the cellular level do occur (biochemical cascade)
- Recovery from these changes is considered neuroplasticity
- Neurons can generally recover after initial concussion
- BUT second concussion during the recovery period may lead to cell death

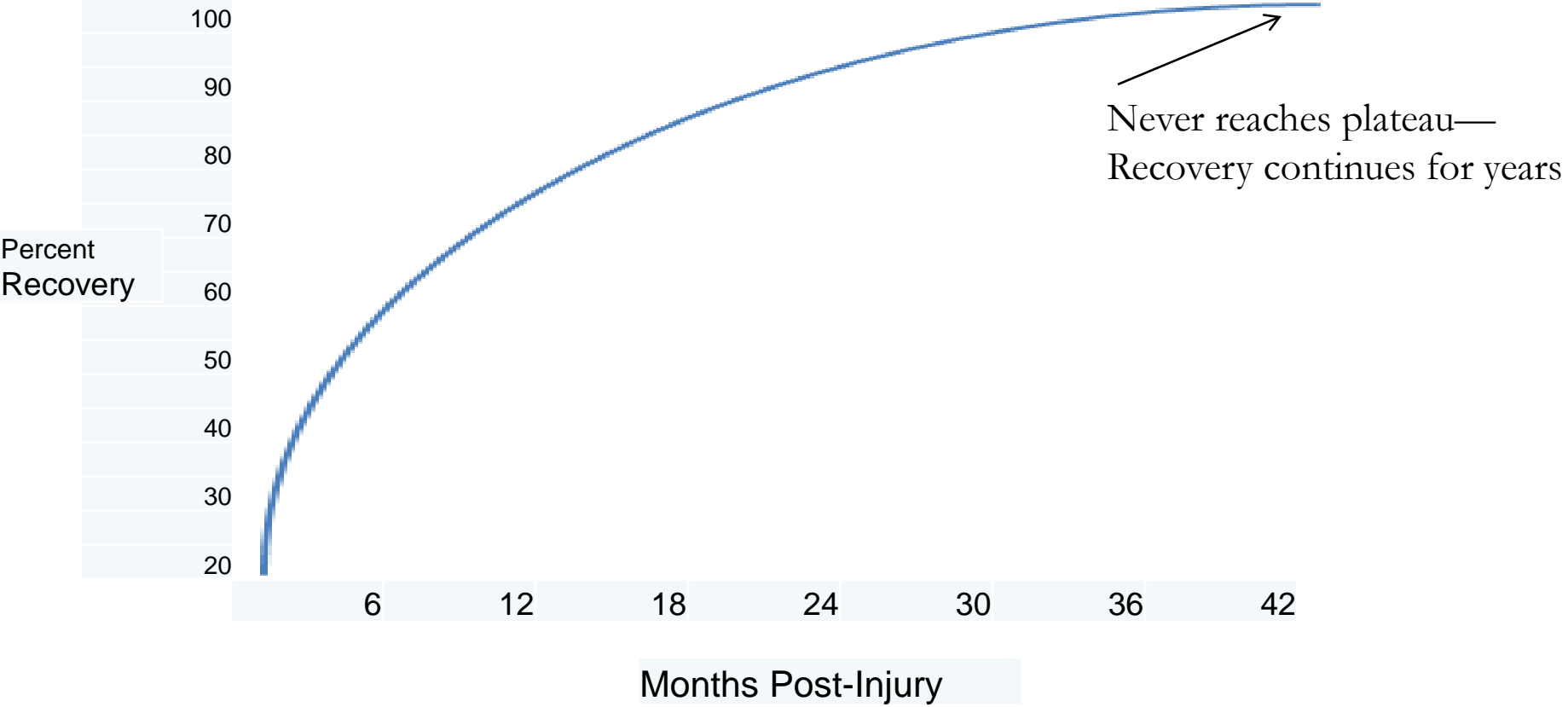
Neuroplasticity and Concussion

- Neurotransmitters, lactate, glucose, neuroinflammatory processes and other functions gradually return to normal states
- The brain has a natural response to disruption and spontaneously heals itself when damage is not severe
- Neural networks damaged by the biochemical cascade can be compensated for by new neural networks serving the same function

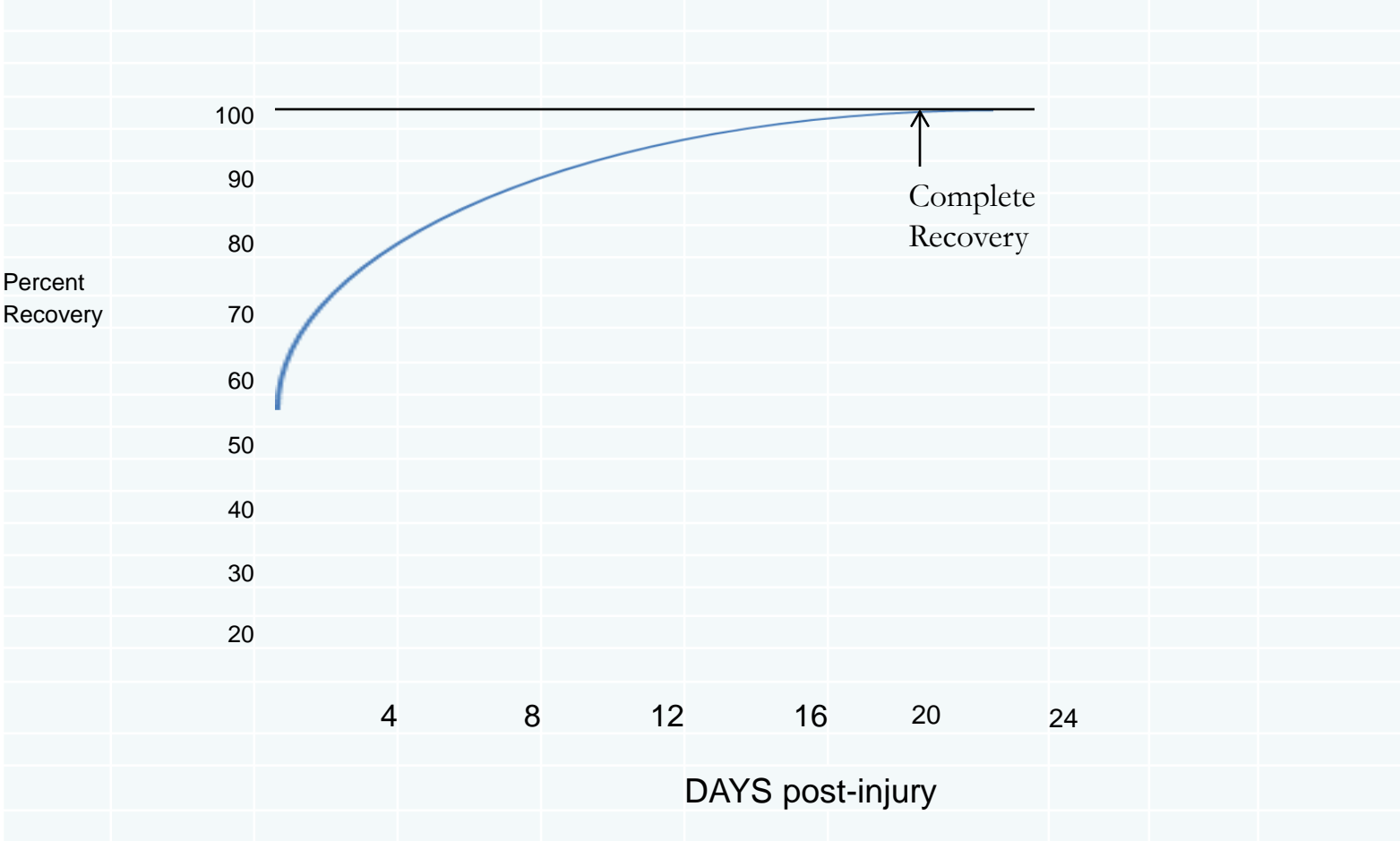
Recovery

- Most uncomplicated concussions resolve in 1-2 weeks
- If more than a few symptoms are present at 4-6 weeks, or some severe, consider evaluation at a concussion clinic
- COMPLETE cognitive and physical rest is no longer considered appropriate
- Tailor rest to symptoms— if activity produces/exacerbates symptoms, back off
- As recovery progresses, increase activity

Recovery after Moderate to Severe TBI



Recovery after Uncomplicated Concussion



Well-Intentioned Exacerbation

- Internist or other health care provider advises complete rest; no computer, cell, video, reading
- Advises no work for xx days/weeks
- No other medical disorder advises complete rest– surgery, back pain, etc.
- There is NO research showing that complete rest improves or speeds up recovery
- Can result in the injured person inadvertently assuming a “sick role”

Risk Factors for Extended Recovery

- History of:
 - Prior concussions (LOC not that predictive)
 - Learning disabilities
- Post-traumatic Amnesia
- Younger age– white matter tracts not fully myelinated yet
- Still “foggy” at one week
- Absence of practice effect on testing

Other Factors Affecting Cognition and Return to Normal Functioning

- Severity of concussion
 - Loss of consciousness
 - Retrograde Amnesia
 - Post-Traumatic Amnesia (most important!)
- Pre-morbid (injury) factors
 - Baseline cognitive functioning
 - Personality style
 - Occupation, responsibilities
 - Family (support, responsibilities)

Functional Disability Long after mTBI

- Neurological factors
 - Nerve injuries or other longer-lasting symptoms
- Physical factors
 - Orthopedic or other non-neurologic injuries
- Psychological factors
 - Strongest cause of prolonged/incomplete recovery
- Personality factors
 - Pre-morbid personality style
- Psychosocial factors
- Litigation

Psychological factors

- – Cognitive compromise (attention, mental efficiency, learning & memory)
- – Cognitive symptoms create frustration, emotional distress, & “shaken sense of self”
- – Anxiety & avoidance of anxiety provoking situations
- – May result in depression
- – Anxiety & depression cause further cognitive problems, which create more anxiety & depression
- – Psychological overlay accumulates & intensifies
- – May become more disabling than the injury

Other Psychological Factors

- Personality styles:
 - Overachiever
 - Dependent
 - Insecure
 - Grandiose
 - Borderline Personality characteristics
- Emotional significance of the injury
 - Sense of self
 - Setback to lifelong goal

Long-Term (> 6 weeks) Symptoms

- “Post-Concussion Syndrome”
- Psychosocial and psychiatric condition related to aforementioned features
- Patients often invested in believing “it’s my brain” rather than accepting psychosocial factors
- Cognitive symptoms can last for years, and are REAL for the patient
- Best treated by psychotherapy or, better yet, cognitive remediation
 - Helps remediate perceived deficits in attention, concentration, memory, executive functions
 - Teaches coping strategies for symptoms
 - Compensatory strategies
 - Relaxation training
- Referral to neurologist trained in concussion can help with residual headaches and sleep problems