Enhanced Brain and Concussion Recovery

Round 1

Knock Out Brain Injury – An Evolving Paradigm in Healing

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Board Certified Hyperbaric and Undersea Medicine

Board Certified Lobster Catcher

I Do Not have any relevant financial or other relationship or affiliation with a commercial vendor or manufacturer of medically-related products, whether referenced in this lecture or not.

Which way do you go?


GIANT

Severe Head Injury

- Cerebral contusion
- Intracranial hemorrhage
- Epidural hematoma
- Subdural hematoma
- Intra cerebral hematoma
- Diffuse axonal injury

We live in a world where all individuals face the risk of head injuries on a daily basis. | Articles and news reports appear regularly discussing the recognition of traumatic brain injuries and the preventative measures that can be used to lessen impact. | Newer modalities are emerging which will allow us to better identify injury. | Multiple techniques exist to assist with brain retraining.

Hyperbaric oxygen therapy is now emerging as the key management tool for brain repair when integrated into a comprehensive concussion recovery program.

Reason

- The paradigm of concussion care is changing rapidly leaving many unaware of the current shift toward early hyperbaric oxygen therapy.

- Traditional brain imaging has been insufficient in detecting a mild traumatic brain injury. Lesser known newer techniques such as QEEG and tensor brain imaging have evolved allowing easier identification- hence easier entry into a brain injury recovery program.

Enhanced Brain and Concussion Recovery: Round 1

Knock Out Brain Injury – An Evolving Paradigm in Healing

- Learning Objectives
  - Upon completion of this lecture, participants should be better able to recognize, investigate, and treat mild traumatic brain injury.
  - All will more fully appreciate the integration of Hyperbaric Oxygen Therapy into a comprehensive brain recovery program.

Concussion

Latin verb concutere….. To shake violently

TBI Statistics

1.7 million brain injuries per year

75% in form of concussions or mTBI

230,000 to 500,000 Hospitalizations per year

52,000 deaths

Economic toll of TBI exceeds $60 Billion per year

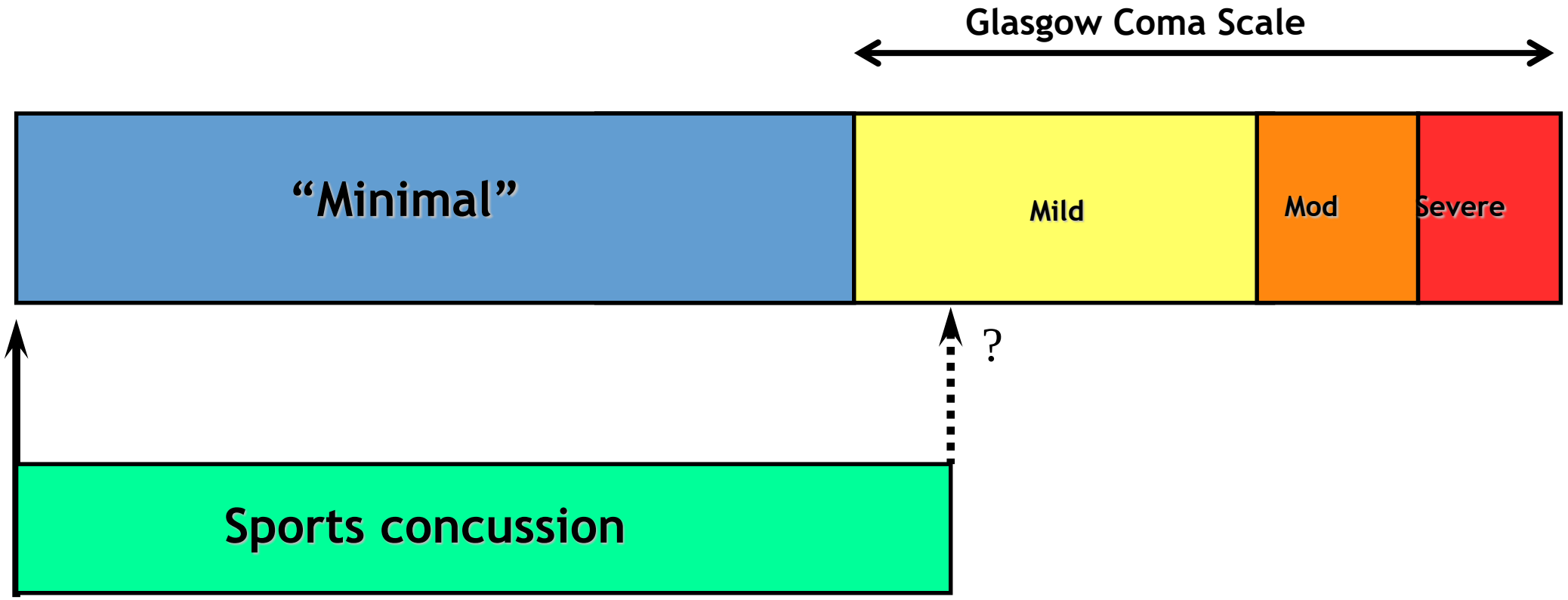
TBI is the LEADING cause of morbidity and mortality in individuals < 45 years of age in the WORLD

Incidence

- Overall, the activities associated with the greatest number of TBI-related ED visits included bicycling, football, playground activities, basketball, and soccer.
- 71% of all sports/recreation-related TBI ED visits were *males.*
- 70% of sports/recreation-related TBI ED visits were *persons aged 10-19 years*.
- For males aged 10-19 years, sports- and recreation-related TBIs occurred most often while playing *football* or *bicycling*.
- Females aged 10-19 years sustained sports- and recreation-related TBIs most often while playing *soccer* or *basketball* or while *bicycling*.

Source: www.cdc.gov

Traumatic Brain Injury



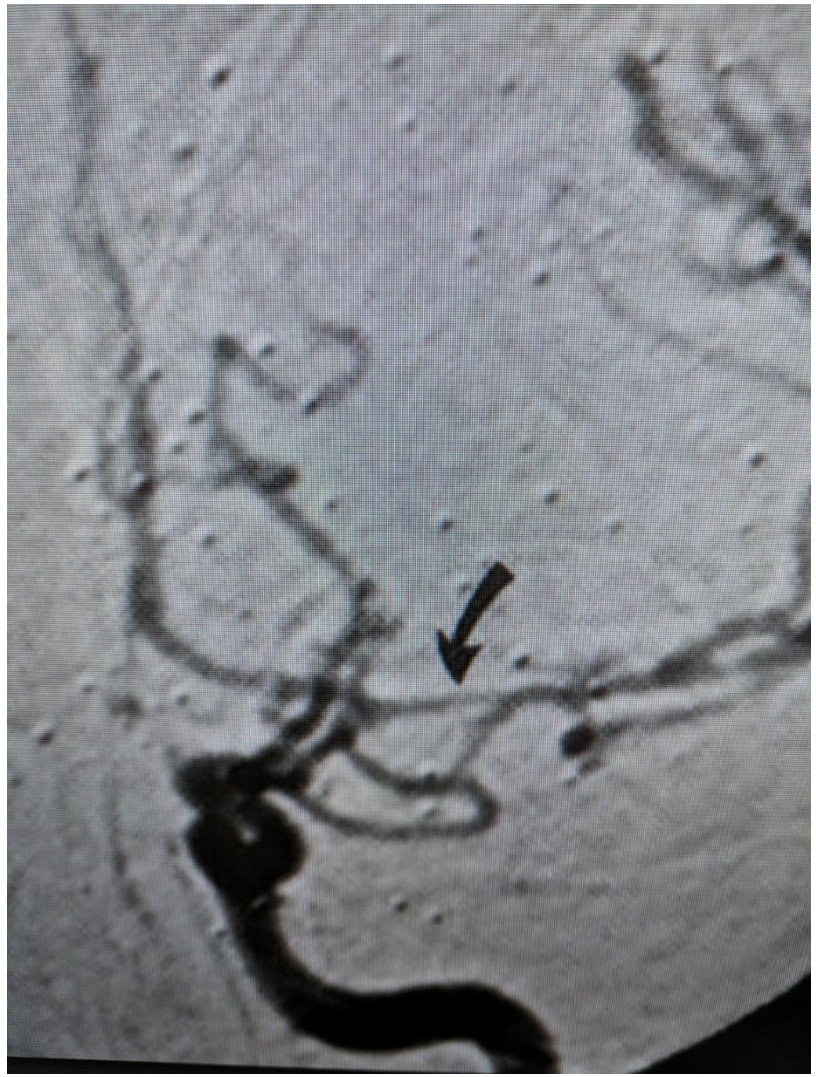
Mild TBI (mTBI)

- Any period of loss of or a decreased level of consciousness lasting less than 30 minutes
- Any loss of memory for events immediately before or after the injury lasting less than 24 hours after the event
- Any alteration in mental state at the time of the injury
- Transient neurological deficits
- >3 months: Post Concussion Syndrome
- 25% of patients continue to have symptoms after 6 months
- 70 to 90% of of all TBI in the US is mTBI

TBI Pathophysiology

- Focal Ischemia
- Cerebral Vasospasm
- Vasospasm found in 25-40% of severe TBI patients

  -Journal of Neurosurgery, Martin, 1992



TBI Pathophysiology

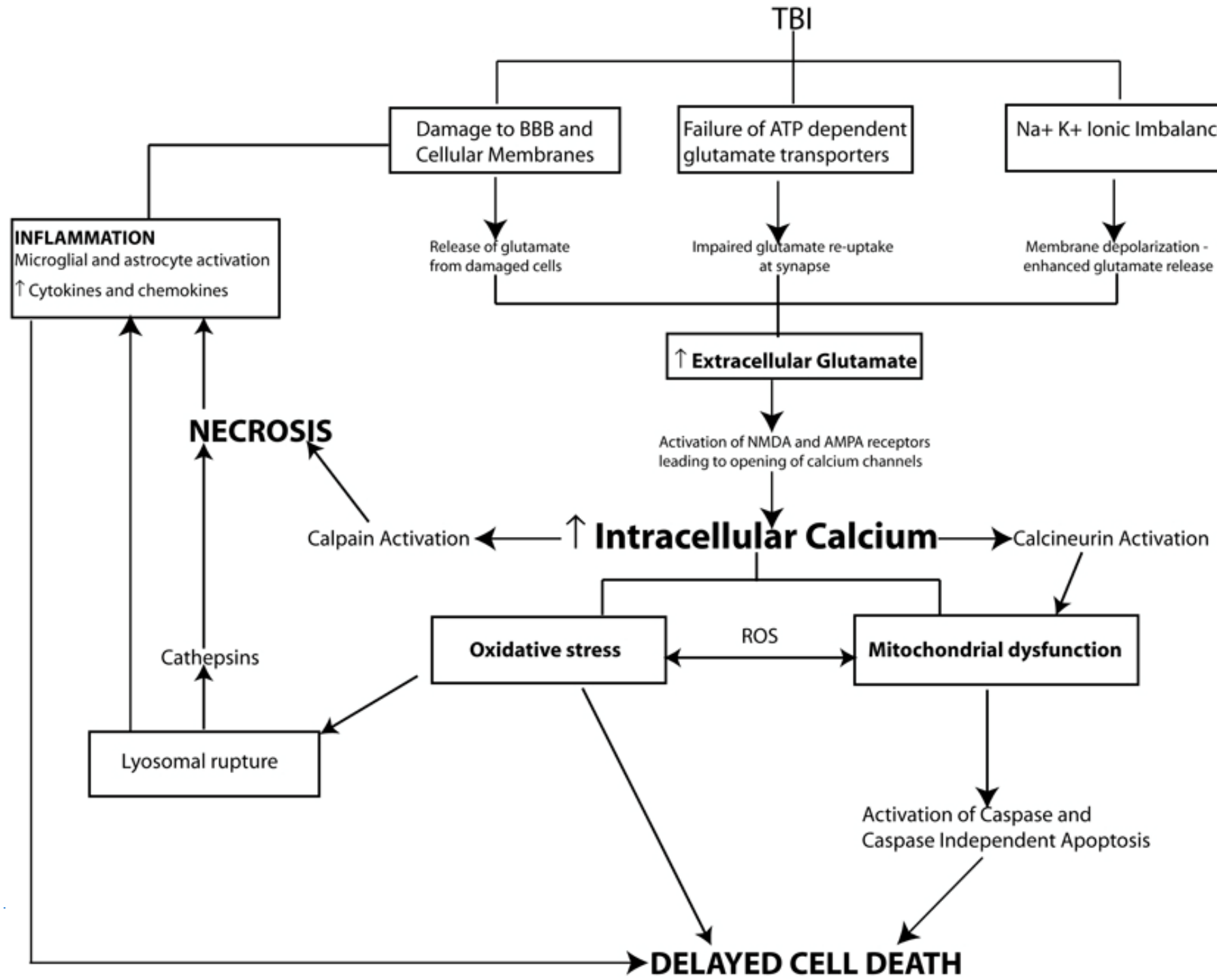
Hypoxia

Multiple studies 1980 – 1999

- Adams, Van den Brink, Zhi

LOW PO_2 seen within first 24 hours

Lower → worse outcome


TBI
Damage to BBB and Cellular Membranes
Failure of ATP dependent glutamate transporters
Na+ K+ Ionic Imbalances
INFLAMMATION
Microglial and astrocyte activation
↑ Cytokines and chemokines
Release of glutamate from damaged cells
Impaired glutamate re-uptake at synapse
Membrane depolarization - enhanced glutamate release
↑ Extracellular Glutamate
NECROSIS
Activation of NMDA and AMPA receptors leading to opening of calcium channels
Calpain Activation
↑ Intracellular Calcium
Calcineurin Activation
Oxidative stress
ROS
Mitochondrial dysfunction
Cathepsins
Lyosomal rupture
Activation of Caspase and Caspase Independent Apoptosis
DELAYED CELL DEATH

Neurometabolic Cascade

increased energy demand
+
decreased CBF (50%)
&
impaired
cellular efficiency

↓

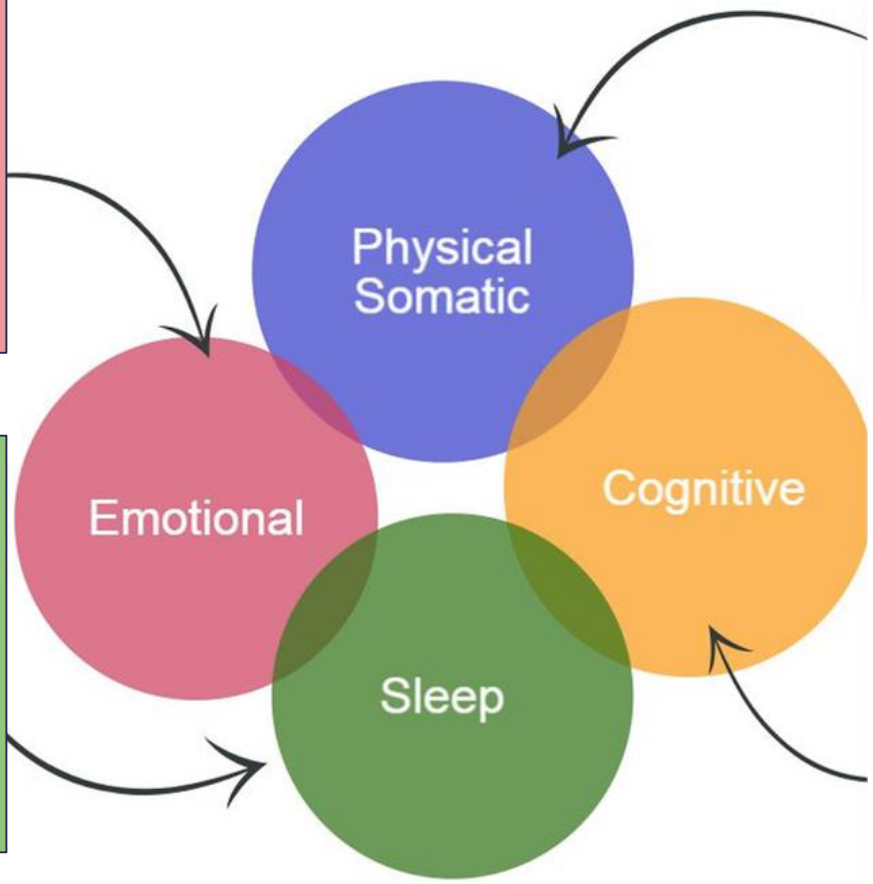
ENERGY CRISIS

J Athvl Train. 2001 Jul-Sep; 36(3): 228–235.

Signs and Symptoms of Concussion

Irritability
Sadness
More emotional
Nervousness

Drowsiness
Sleeping more than usual
Sleeping less than usual
Difficulty falling asleep



Headache
Nausea
Vomiting
Dazed
Stunned
Fatigue
Light Sensitivity
Sound Sensitivity
Balance problems
Vision Problems

Feeling "foggy"
Feeling slowed down
Difficulty concentrating
Difficulty remembering
Confused about recent events
Answers questions slowly
Repeats Questions

PEDIATRICS Vol. 126 No. 3 September 1, 2010

Brain injury recovery: It's not easy

A healthy brain utilizes *ALL* energy supplied to it

15% of the Cardiac Output

20% of the Total Body Oxygen Consumption

25% of the Total Body Glucose

Brain injury recovery: It's not easy

Healing from a brain injury requires additional energy (ATP)

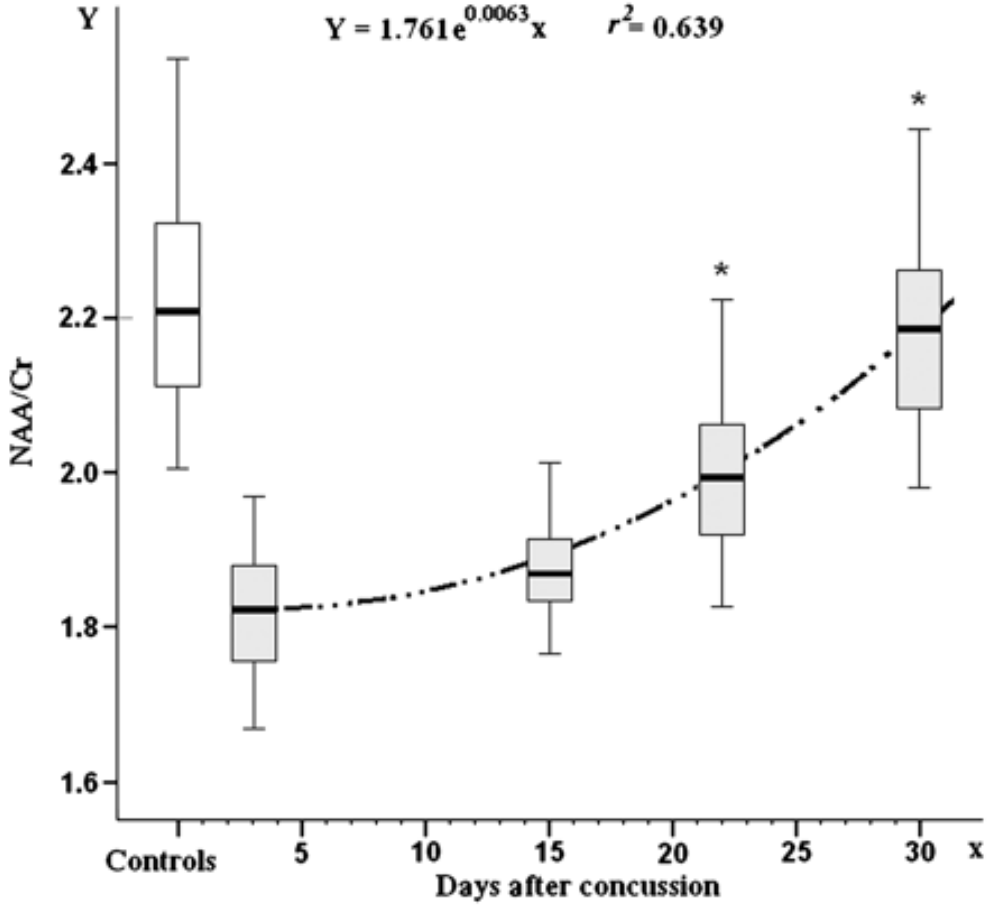
Energy is not in ready supply given high demands of brain tissue

In 80-90% Of mTBI Cases, The Symptoms Fade In 7–10 Days

McCrory, P., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J., et al. (2005). Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br. J. Sports Med. 39, 196–204. doi: 10.1136/bjsm.2005.018614

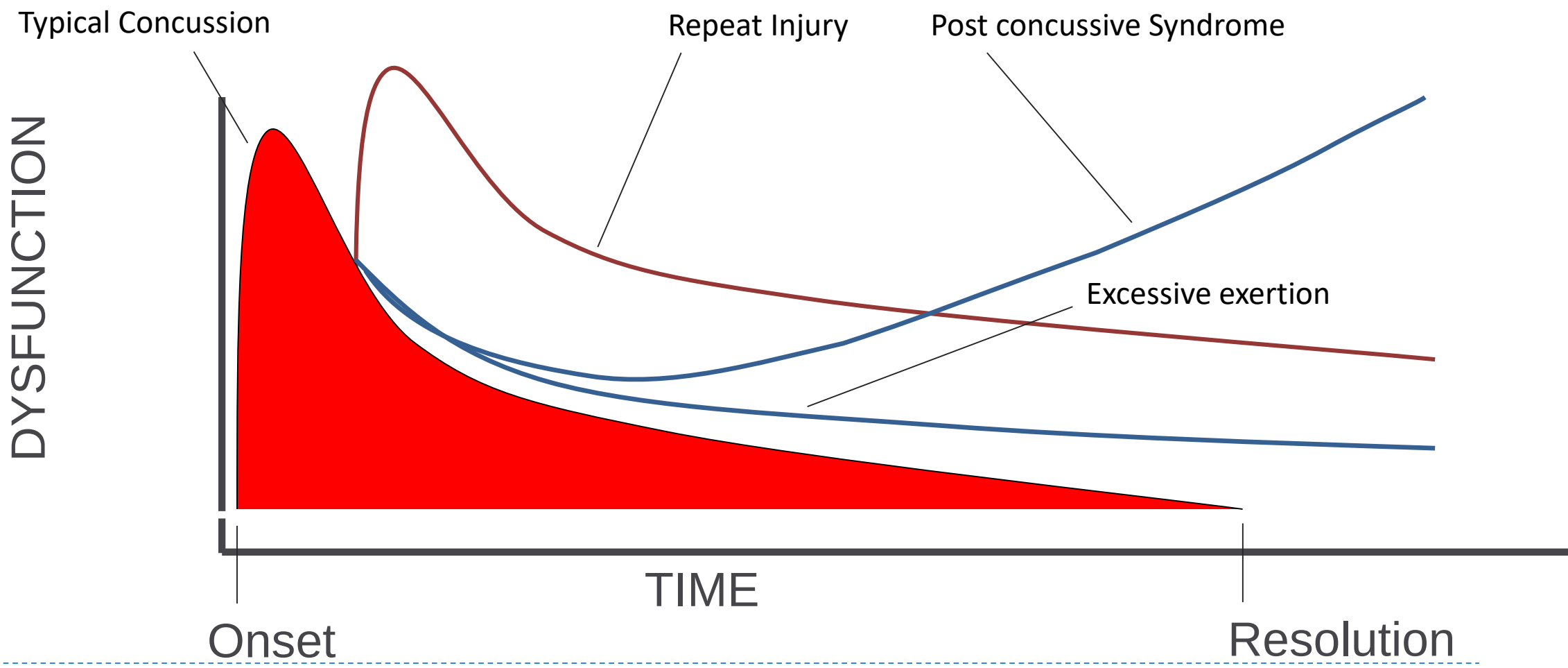
TBI Pathophysiology
Brain Metabolism following mTBI

- Proton magnetic resonance spectroscopy
- Recovery of neuronal metabolism marker in 40 athletes following concussion
- N-acetylaspartate/creatine-containing compounds ratio
- Concussive head injury→ window of brain vulnerability from cellular energetic metabolism impairment
- Symptom recovering 3-15 days
- Normalized metabolism by 30 days



Brain 2010; 133(11): 3232-3242.

Concussion Recovery Timeline



SECOND IMPACT SYNDROME

- Occurs in athletes with prior concussion following relatively minor second impact (controversial and based upon single case studies)
- Catastrophic increase in intracranial pressure due to dysfunction of autoregulation of cerebral circulation
- Most often occurs in athletes < 24 years old

LOW INCIDENCE – HIGH POTENTIAL ACUTE IMPACT

- Schneider, 1973; Saunders; 1984; Cantu, 1998.
  Evidence Level 4

Facts on Helmets

- Bicycle helmets are 85% effective in reducing traumatic brain injuries
- Only 40% of cyclists wear helmets

Wear the helmet flat atop your head, not tilted back at an angle!


Make sure the helmet fits snugly and does not obstruct your field of vision. Make sure the chin strap fits securely and that the buckle stays fastened.

Source: www.thinkfirst.org

Use of a helmet while biking could prevent 1 injury every 4 minutes in the U.S.

2010 NFL Concussion Policy

"Once removed for the duration of a practice or game, the player should not be considered for return-to-football activities until he is fully asymptomatic, both at rest and after ex… normal neurological examinatio… neuropsychological testing a… …eared to return by both his tea… …nd the independent neur… …ant."

WHERE'S THE HBOT ?!?

National Football League Concussion Guideline Policy, The Associated Press, 2009. http://www.nfl.com/news/story?confirm=true&id=0900 0d5d814a9ecd&template=with-video-with-comments

2018 Additional NFL Directives

- Neuropsychological testing has been expanded for all NFL players who have been removed from a game due to a [illegible] be re-tested during the season as part of the medical [illegible] of the player and to assist in determining when players [illegible] actice and play. Each club will select the neuropsych[illegible] provider of its choice.

- Player safety rules relati[illegible] the helmet will continue to be closely enforced. This wil[illegible] nforcement of the requirement that chin straps on he[illegible] ely and properly buckled so that the helmet provid[illegible] protection.

- The [illegible] will continue to research and study all elements of concussions with a particular focus on long-term effects.

WHERE'S THE HBOT ?!?

National Football League , November 18, 2008
http://www.nfl.com/news/story/09000d5d8017cc67/printable/nfl-outlines-for-players-steps-taken-to-address-concussions

Retired Players Get It

Otis Jerome Anderson “O.J.”

Bart Oates

Return to Play:
Consensus Statements and Evidence Based Guidelines

Zurich Consensus Conference on Sports Concussion (2013)

- One and Done: No return until symptom free with exertion
- Individually based decisions (one size does not fit all)
- Short period of rest and slow return to activity/school

WHERE'S THE HBOT ?!?

American Academy of Neuro Evidence Based Return to Play Guidelines (2013)

- One and Done: No return until symptom free with exertion
- Individually based decisions (no set timeline for safe RTP)
- HS age and younger should be managed more conservativ

WHERE'S THE HBOT ?!?

Graduated RTP Protocol (Zurich 2012)

WHERE'S THE HBOT ?!?

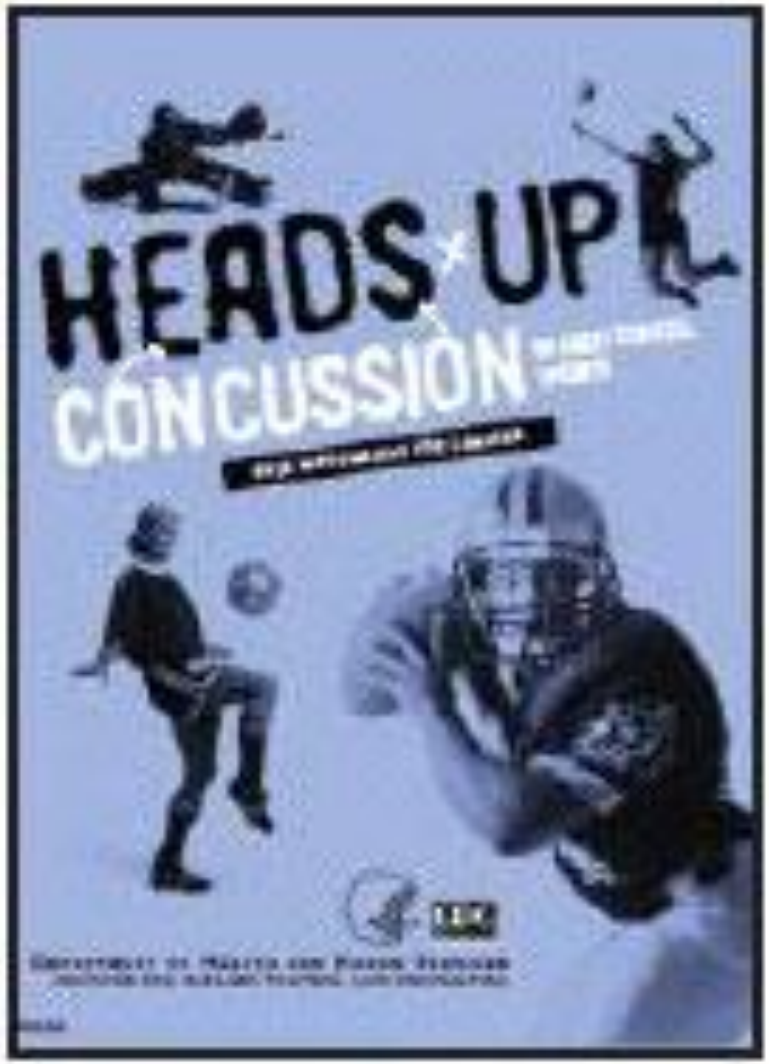
Rehabilitation Stage	Functional Exercise Each Stage	Objective of Each Stage
No Activity	Physical and Cognitive Rest	Recovery
Light Aerobic Exercise	Walking, swimming, stationary bike, <70% maximum MR. No resistance training.	Increase HR
Sports Specific Exercise	Skating drills in ice hockey, running drills, No head impact activities	Add Movement
Non-contact training drills	Progression to more complex training drills → May start progressive resistance training	Exercise, coordination and cognitive load
Full-contact practice	Full practice (following medical clearance)	Restore confidence and assess functional skills by coaching staff
Return to Play	Normal game play	

Heads Up: Concussion in High School Sports

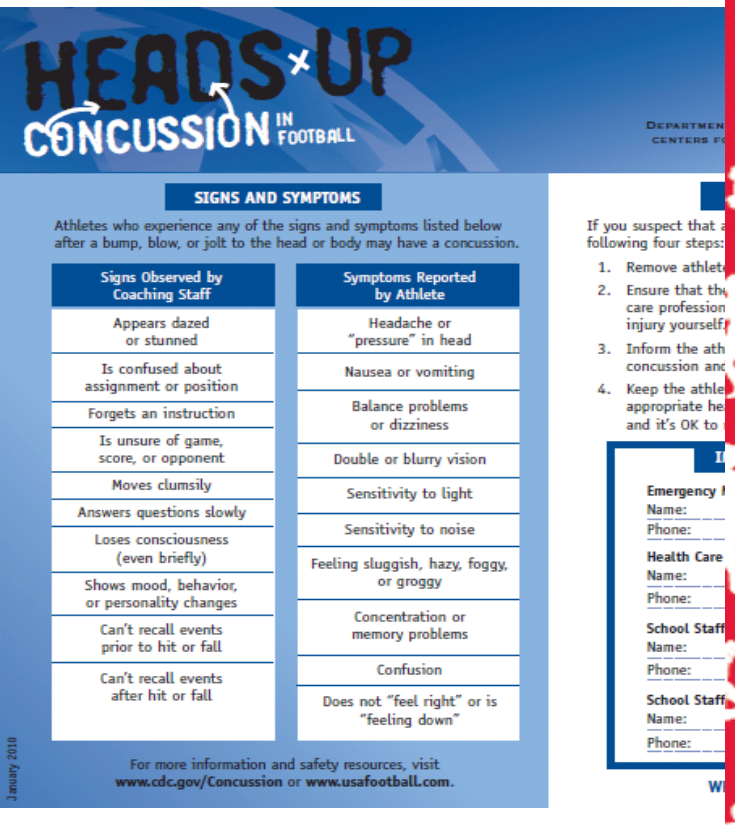
- CDC program
  - Online training
  - Fact sheets
  - Symptom posters
  - Wallet cards

- Four Step Action Plan

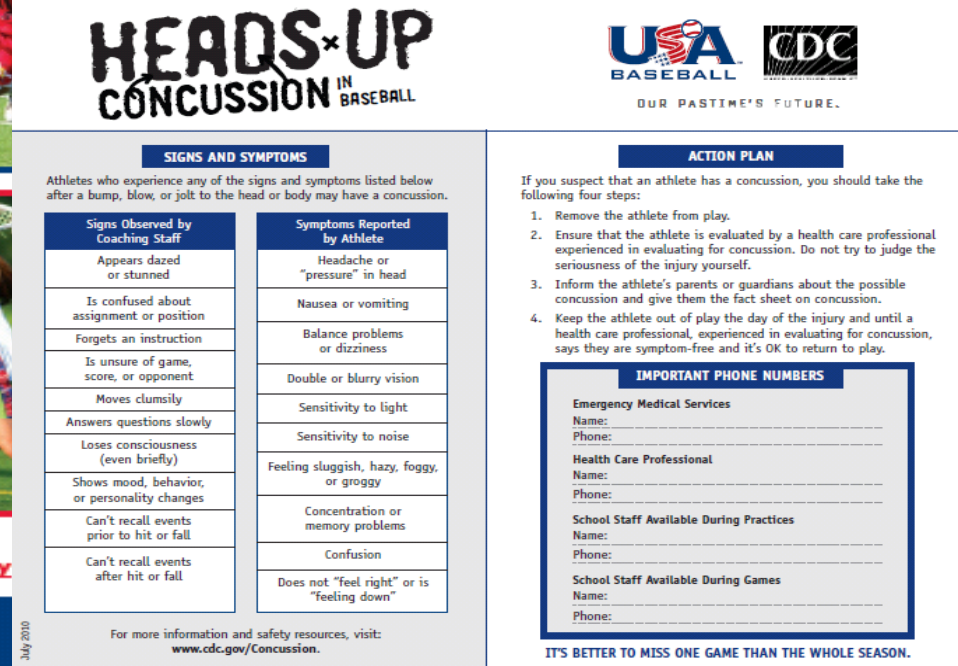
WHERE'S THE HBOT ?!?



Use Available Tools



WHERE'S THE HBOT ?!?



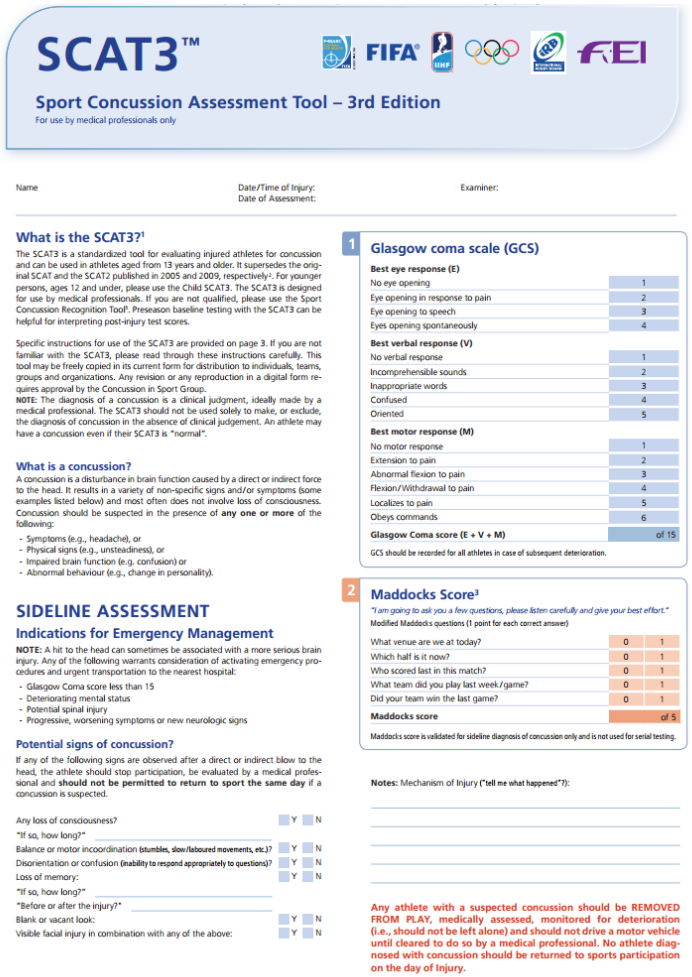
Sports Concussion Assessment Tool 2 (SCAT2)

Standardized method of evaluating injured athletes for concussion

- Used in athletes aged from 10 years and older
- Assessment Includes:
  - Symptoms: 22 possible
  - Cognitive & Physical Assessment
    - LOC?
    - Glasgow coma scale: eye, verbal, motor
    - Orientation: Month, Date, Day of the week, year, time?
    - Immediate Memory recall: 5 word recall: (elbow, apple, carpet, saddle, bubble)
    - Concentration:
      - Repeat Digits Backwards: 3-9-7, 4-6-1-0, 2-9-6-1-4
      - Months of the year in reverse order
  - Balance
  - Coordination

Resources

SCAT3



SCAT3™

Sport Concussion Assessment Tool – 3rd Edition

For use by medical professionals only

Name | Date/Time of Injury: Date of Assessment: | Examiner:

What is the SCAT3?[1]

The SCAT3 is a standardized tool for evaluating injured athletes for concussion and can be used in athletes aged from 13 years and older. It supersedes the original SCAT and the SCAT2 published in 2005 and 2009, respectively[2]. For younger persons, ages 12 and under, please use the Child SCAT3. The SCAT3 is designed for use by medical professionals. If you are not qualified, please use the Sport Concussion Recognition Tool[3]. Preseason baseline testing with the SCAT3 can be helpful for interpreting post-injury test scores.

Specific instructions for use of the SCAT3 are provided on page 3. If you are not familiar with the SCAT3, please read through these instructions carefully. This tool may be freely copied in its current form for distribution to individuals, teams, groups and organizations. Any revision or any reproduction in a digital form requires approval by the Concussion in Sport Group.

NOTE: The diagnosis of a concussion is a clinical judgment, ideally made by a medical professional. The SCAT3 should not be used solely to make, or exclude, the diagnosis of concussion in the absence of clinical judgement. An athlete may have a concussion even if their SCAT3 is "normal".

What is a concussion?

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific signs and/or symptoms (some examples listed below) and most often does not involve loss of consciousness. Concussion should be suspected in the presence of **any one or more** of the following:

- Symptoms (e.g., headache), or
- Physical signs (e.g., unsteadiness), or
- Impaired brain function (e.g. confusion) or
- Abnormal behaviour (e.g., change in personality).

SIDELINE ASSESSMENT

Indications for Emergency Management

NOTE: A hit to the head can sometimes be associated with a more serious brain injury. Any of the following warrants consideration of activating emergency procedures and urgent transportation to the nearest hospital:

- Glasgow Coma score less than 15
- Deteriorating mental status
- Potential spinal injury
- Progressive, worsening symptoms or new neurologic signs

Potential signs of concussion?

If any of the following signs are observed after a direct or indirect blow to the head, the athlete should stop participation, be evaluated by a medical professional and **should not be permitted to return to sport the same day** if a concussion is suspected.

Any loss of consciousness?	Y N
"If so, how long?"	
Balance or motor incoordination (stumbles, slow/laboured movements, etc.)?	Y N
Disorientation or confusion (inability to respond appropriately to questions)?	Y N
Loss of memory:	Y N
"If so, how long?"	
"Before or after the injury?"	
Blank or vacant look:	Y N
Visible facial injury in combination with any of the above:	Y N

1 Glasgow coma scale (GCS)

Best eye response (E)	
No eye opening	1
Eye opening in response to pain	2
Eye opening to speech	3
Eyes opening spontaneously	4
Best verbal response (V)	
No verbal response	1
Incomprehensible sounds	2
Inappropriate words	3
Confused	4
Oriented	5
Best motor response (M)	
No motor response	1
Extension to pain	2
Abnormal flexion to pain	3
Flexion/Withdrawal to pain	4
Localizes to pain	5
Obeys commands	6
Glasgow Coma score (E + V + M)	of 15

GCS should be recorded for all athletes in case of subsequent deterioration.

2 Maddocks Score[3]

"I am going to ask you a few questions, please listen carefully and give your best effort."

Modified Maddocks questions (1 point for each correct answer)

What venue are we at today?	0	1
Which half is it now?	0	1
Who scored last in this match?	0	1
What team did you play last week / game?	0	1
Did your team win the last game?	0	1
Maddocks score		of 5

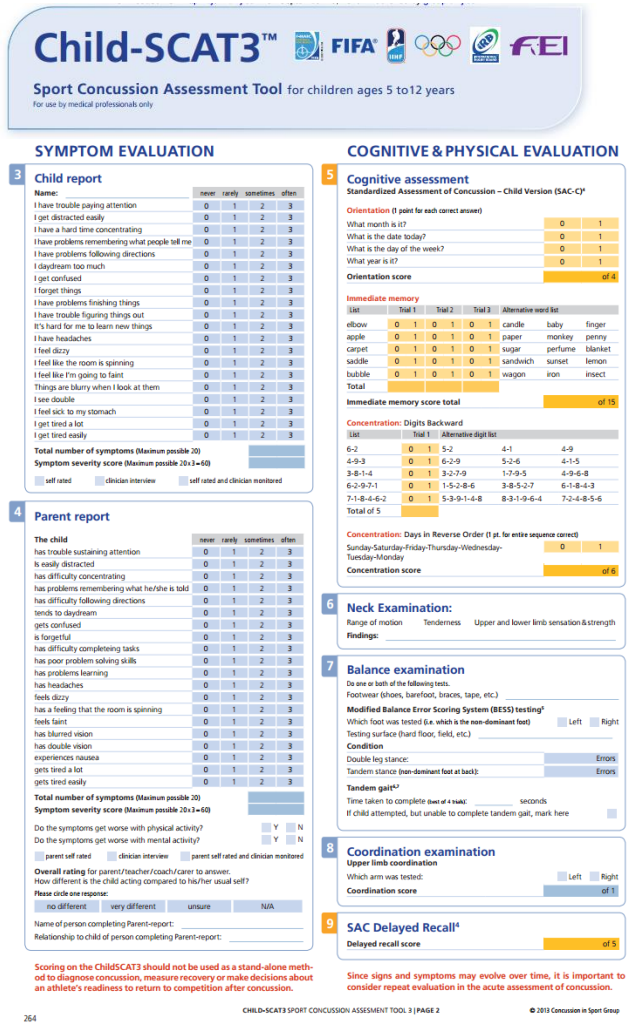
Maddocks score is validated for sideline diagnosis of concussion only and is not used for serial testing.

Notes: Mechanism of Injury (**"tell me what happened"?**):

Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle until cleared to do so by a medical professional. No athlete diagnosed with concussion should be returned to sports participation on the day of Injury.

http://bjsm.bmj.com/content/47/5/259.full.pdf

Child SCAT3



Child-SCAT3™

Sport Concussion Assessment Tool for children ages 5 to12 years

For use by medical professionals only

SYMPTOM EVALUATION

3 Child report

Name:

	never	rarely	sometimes	often
I have trouble paying attention	0	1	2	3
I get distracted easily	0	1	2	3
I have a hard time concentrating	0	1	2	3
I have problems remembering what people tell me	0	1	2	3
I have problems following directions	0	1	2	3
I daydream too much	0	1	2	3
I get confused	0	1	2	3
I forget things	0	1	2	3
I have problems finishing things	0	1	2	3
I have trouble figuring things out	0	1	2	3
It's hard for me to learn new things	0	1	2	3
I have headaches	0	1	2	3
I feel dizzy	0	1	2	3
I feel like the room is spinning	0	1	2	3
I feel like I'm going to faint	0	1	2	3
Things are blurry when I look at them	0	1	2	3
I see double	0	1	2	3
I feel sick to my stomach	0	1	2	3
I get tired a lot	0	1	2	3
I get tired easily	0	1	2	3
Total number of symptoms (Maximum possible 20)				
Symptom severity score (Maximum possible 20 x 3 = 60)				

self rated | clinician interview | self rated and clinician monitored

4 Parent report

The child	never	rarely	sometimes	often
has trouble sustaining attention	0	1	2	3
is easily distracted	0	1	2	3
has difficulty concentrating	0	1	2	3
has problems remembering what he/she is told	0	1	2	3
has difficulty following directions	0	1	2	3
tends to daydream	0	1	2	3
gets confused	0	1	2	3
is forgetful	0	1	2	3
has difficulty completeing tasks	0	1	2	3
has poor problem solving skills	0	1	2	3
has problems learning	0	1	2	3
has headaches	0	1	2	3
feels dizzy	0	1	2	3
has a feeling that the room is spinning	0	1	2	3
feels faint	0	1	2	3
has blurred vision	0	1	2	3
has double vision	0	1	2	3
experiences nausea	0	1	2	3
gets tired a lot	0	1	2	3
gets tired easily	0	1	2	3
Total number of symptoms (Maximum possible 20)				
Symptom severity score (Maximum possible 20 x 3 = 60)				

Do the symptoms get worse with physical activity? Y N

Do the symptoms get worse with mental activity? Y N

parent self rated | clinician interview | parent self rated and clinician monitored

Overall rating for parent/teacher/coach/carer to answer.

How different is the child acting compared to his/her usual self?

Please circle one response:

no different | very different | unsure | N/A

Name of person completing Parent-report:

Relationship to child of person completing Parent-report:

Scoring on the ChildSCAT3 should not be used as a stand-alone method to diagnose concussion, measure recovery or make decisions about an athlete's readiness to return to competition after concussion.

COGNITIVE & PHYSICAL EVALUATION

5 Cognitive assessment

Standardized Assessment of Concussion – Child Version (SAC-C)[4]

Orientation (1 point for each correct answer)

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
Orientation score		of 4

Immediate memory

List	Trial 1		Trial 2		Trial 3		Alternative word list		
elbow	0	1	0	1	0	1	candle	baby	finger
apple	0	1	0	1	0	1	paper	monkey	penny
carpet	0	1	0	1	0	1	sugar	perfume	blanket
saddle	0	1	0	1	0	1	sandwich	sunset	lemon
bubble	0	1	0	1	0	1	wagon	iron	insect
Total									

Immediate memory score total of 15

Concentration: Digits Backward

List	Trial 1	Alternative digit list		
6-2	0 1	5-2	4-1	4-9
4-9-3	0 1	6-2-9	5-2-6	4-1-5
3-8-1-4	0 1	3-2-7-9	1-7-9-5	4-9-6-8
6-2-9-7-1	0 1	1-5-2-8-6	3-8-5-2-7	6-1-8-4-3
7-1-8-4-6-2	0 1	5-3-9-1-6-8	8-3-1-9-6-4	7-2-4-8-5-6
Total of 5				

Concentration: Days in Reverse Order (1 pt. for entire sequence correct)

Sunday-Saturday-Friday-Thursday-Wednesday-Tuesday-Monday 0 1

Concentration score of 6

6 Neck Examination:

Range of motion | Tenderness | Upper and lower limb sensation & strength

Findings:

7 Balance examination

Do one or both of the following tests.

Footwear (shoes, barefoot, braces, tape, etc.)

Modified Balance Error Scoring System (BESS) testing[5]

Which foot was tested (i.e. which is the non-dominant foot) Left Right

Testing surface (hard floor, field, etc.)

Condition

Double leg stance: Errors

Tandem stance (non-dominant foot at back): Errors

Tandem gait[4,5]

Time taken to complete (best of 4 trials): seconds

If child attempted, but unable to complete tandem gait, mark here

8 Coordination examination

Upper limb coordination

Which arm was tested: Left Right

Coordination score of 1

9 SAC Delayed Recall[4]

Delayed recall score of 5

Since signs and symptoms may evolve over time, it is important to consider repeat evaluation in the acute assessment of concussion.

264

CHILD-SCAT3 SPORT CONCUSSION ASSESSMENT TOOL 3 | PAGE 2

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http://bjsm.bmj.com/content/47/5/263.full.pdf

Specific treatments

- Cognitive/fatigue
  - Sleep aids – melatonin
  - Possible stimulants – short term
- Migraine
  - Abortive and preventive
  - SSRIs, triptans
  -
- Cervical
  - PT, traction
- Anxiety/Mood
  - SSRI, sleep treatment as above
  - Behavioral therapy

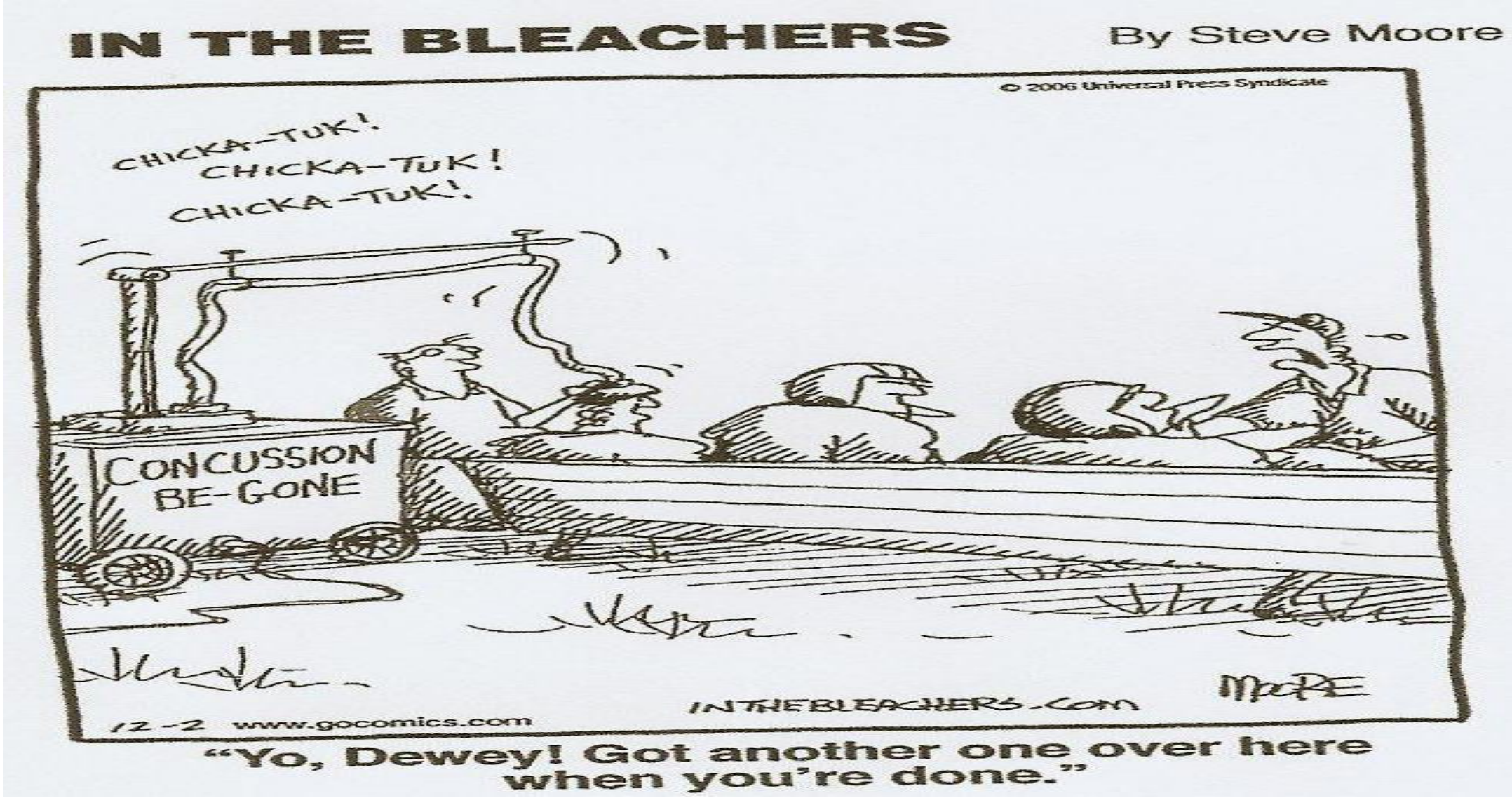
WHERE'S THE HBOT ?!?

Specific treatments

- Vestibular
  - Vestibular rehab - head coordination, balance, and gait-related exercises

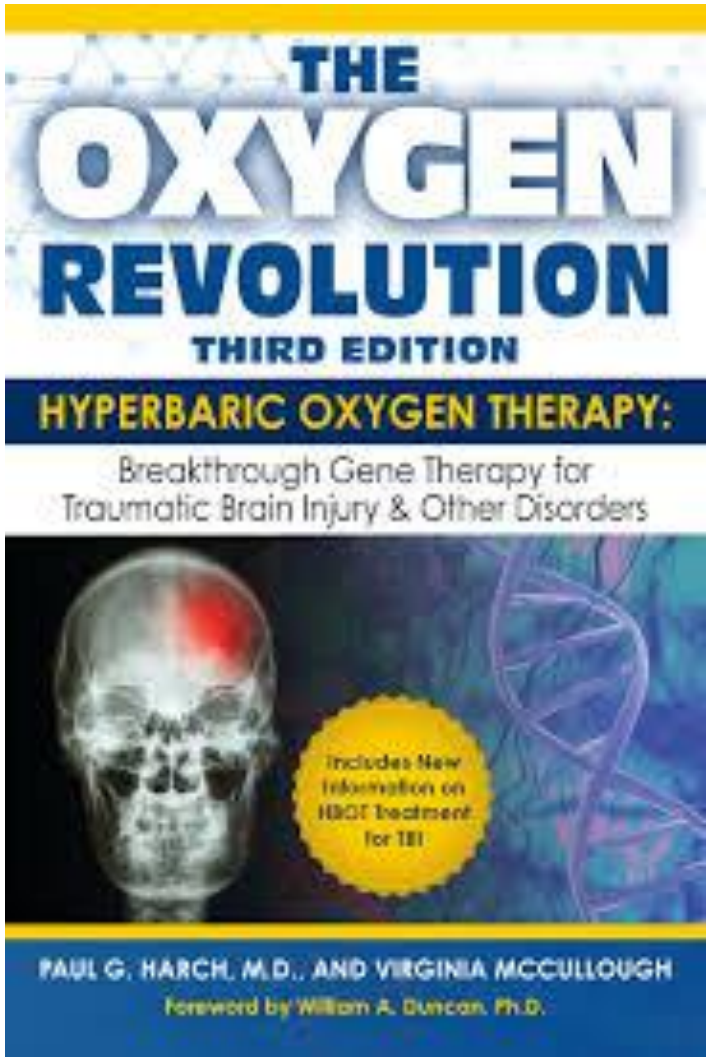
- Ocular
  - Eye exercises involving lenses, prisms, penlights, and cover-ups designed to improve ocular function
  - Vision therapy in treating convergence and accommodative insufficiency
  - Behavioral ophthalmologist or optometrist

WHERE'S THE HBOT ?!?


IN THE BLEACHERS
By Steve Moore
© 2006 Universal Press Syndicate
CHICKA-TUK!
CHICKA-TUK!
CHICKA-TUK!
CONCUSSION BE-GONE
12-2 www.gocomics.com
INTHEBLEACHERS.COM
MOORE
"Yo, Dewey! Got another one over here when you're done."

HBOT !

HBOT Starts Here



Disabilty and Rehabilitation, December 2006

Improving neuropsychological function after chronic brain injury with hyperbaric oxygen.

Golden Z[1], Golden CJ, Neubauer RA

PURPOSE:

One suggested treatment for chronic brain injury (CBI) is the use of hyperbaric oxygen therapy (HBOT). The present study was an evaluation of neuropsychological improvement after HBOT in CBI patients.

METHOD:

Study 1 compared test - retest results of 21 CBI children treated with HBOT against test - retest results of 42 untreated brain injured and normal children. Study 2 compared 21 CBI adults treated with HBOT against 42 untreated normal and brain injured adults. In each study, subjects received pre and post assessments to evaluate neuropsychological function. ? pressure

RESULTS:

The HBOT-treated children showed significant improvement when compared with the two control groups on measures of daily living, socialization, communication, and motor skills. The treated adults made significant gains in all neuropsychological areas tested as compared to controls.

CONCLUSION:

The studies were strongly supportive of HBOT as a treatment for lessening the neurological impact of CBI. These studies indicate that HBOT can be an effective aid in ameliorating the neuropsychological and physiological effects of CBI. The absence of a clear sham HBOT treatment group is an issue as it could be that there was a placebo effect, but it should be noted that the controls were receiving more traditional interventions during the study.

“There is a need to study the degree to which the factors of higher pressure and higher oxygen levels are responsible for the effects seen in this study. The initial emphasis has been to show that the therapy can work. Now we must determine if it is the higher oxygen levels or the higher pressure or a combination of the two is responsible for the effects seen here. It may be that a less expensive but effective treatment can be derived which relies on lower oxygen levels or lower pressure. The researcher is working on the design and implementation of such studies at present.”

Undersea Hyperb Med. **2009** Nov-Dec;36(6):391-9.

Case report: Treatment of mild traumatic brain injury with hyperbaric oxygen.

Wright JK[1], Zant E, Groom K, Schlegel RE, Gilliland K.

Abstract

Two United States Air Force Airmen were injured in a roadside improvised explosive device (IED) blast in Iraq in January 2008. Both airmen suffered concussive injuries and developed irritability, sleep disturbances, headaches, memory difficulties and cognitive difficulties as symptoms of mild traumatic brain injury (mTBI). Six months after injury, repeat Automated Neuropsychological Assessment Metrics (ANAM) testing showed deterioration, when compared to pre-injury baseline ANAM assessment, in all measured areas (simple reaction time, procedural reaction time, code substitution learning, code substitution delayed, mathematical processing, and matching to sample). The airmen were treated with hyperbaric oxygen in treatments of 100% oxygen for one hour at **1.5 atmospheres** absolute, resulting in rapid improvement of headaches and sleep disturbances, improvement in all symptoms and resolution of most symptoms. Repeat ANAM testing after completion of the hyperbaric treatments - nine months after initial injury - showed improvement in all areas, with most measures improving to pre-injury baseline levels. The airmen received no other treatment besides medical monitoring. Repeat neuropsychologic testing confirmed the improvement. We conclude that the improvement in symptoms and ANAM performance is most likely attributable to HBO treatment.

J Neurotrauma. **2012** Nov 20;29(17):2606-12. doi: 10.1089/neu.2012.2549. Epub 2012 Nov 9.

The effect of hyperbaric oxygen on symptoms after mild traumatic brain injury.

Wolf G[1], Cifu D, Baugh L, Carne W, Profenna L.

Abstract

In this single-center, double-blind, randomized, sham-controlled, prospective trial at the U.S. Air Force School of Aerospace Medicine, the effects of **2.4 atmospheres absolute** (ATA) hyperbaric oxygen (HBO_2) on post-concussion symptoms in 50 military service members with at least one combat-related, mild traumatic brain injury were examined. Each subject received 30 sessions of either a sham compression (room air at 1.3 ATA) or HBO_2 treatments at 2.4 ATA over an 8-week period. Individual and total symptoms scores on Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT®) and composite scores on Post-traumatic Disorder Check List-Military Version (PCL-M) were measured just prior to intervention and 6 weeks after completion of intervention. Difference testing of post-intervention means between the sham-control and HBO_2 group revealed no significant differences on the PCL-M composite score ($t=-0.205$, $p=0.84$) or on the ImPACT total score ($t=-0.943$, $p=0.35$), demonstrating no significant effect for HBO_2 at 2.4 ATA. PCL-M composite scores and ImPACT total scores for sham-control and HBO(2) groups revealed significant improvement over the course of the study for both the sham-control group ($t=3.76$, $p=0.001$) and the HBO_2 group ($t=3.90$, $p=0.001$), demonstrating no significant HBO_2 effect. Paired t-test results revealed 10 ImPACT scale scores in the **sham-control group** improved from pre- to post-testing, whereas two scale scores significantly improved in the HBO_2 group. One PCL-M measure improved from pre- to post-testing in both groups. This study showed that HBO_2 at 2.4 ATA pressure had no effect on post-concussive symptoms after mild TBI.

HBO & mTBI: Harch

A Phase I Study of Low-Pressure Hyperbaric Oxygen Therapy for Blast-Induced Post-Concussion Syndrome and Post-Traumatic Stress Disorder: Harch et al January 2012 Journal of Neurotrauma

- 15 patients symptomatic veterans w/blast injury
- Average 2.6 years post injury
- 40 treatments **1.5 ATA** in 30 days
- SPECT scanning done pretreatment, after 1 treatment, and after all 40 treatments

HBO & mTBI: Harch

- Harch Phase I Study (continued)
  - Significant symptomatic, cognitive, and affective improvements on an array of testing
  - 30% reduction in PTSD symptoms
  - IQ increased an average of 15 points
  - **64% of patients able to decrease or eliminate narcotic and psychoactive medications**
  - SPECT scanning documented improvement in blood flow

J Neurosurg 118:1317–1328, 2013

A prospective, randomized Phase II clinical trial to evaluate the effect of combined hyperbaric and normobaric hyperoxia on cerebral metabolism, intracranial pressure, oxygen toxicity, and clinical outcome in severe traumatic brain injury

Clinical article

***Sarah B. Rockswold, M.D.,[1–3] Gaylan L. Rockswold, M.D., Ph.D.,[3,4] David A. Zaun, M.S.,[5] and Jiannong Liu, Ph.D.[5]**

[1]Department of Physical Medicine and Rehabilitation and [2]Division of Neurosurgery, Department of Surgery, Hennepin County Medical Center; Departments of [3]Medicine and Rehabilitation and [4]Neurosurgery, University of Minnesota, Minneapolis, Minnesota; and [5]Analytical Services, Chronic Disease Research Group, Minneapolis Medical Research Foundation, Minneapolis, Minnesota

Acute, Severe Tbi: Rockswald et al

- 42 patients with mean GCS of 5.7 were prospectively randomized into two groups
- Group #1: HBOT (100% Oxygen) @1.5 ATA X 60 minutes followed by 3 hours of 100% face mask oxygen at 1.0 ATA for 3 treatments on 3 successive days
- Group #2: Standard severe TBI care (no HBOT chamber)
- Sham not needed: sedated and paralyzed

Acute, Severe Tbi: Rockswald et al

- Brain tissue O2 levels increased 600% during combined treatment
- Decreased intracranial pressure that lasted until next HBOT session
- In post treatment period brain tissue oxygen levels remained higher by 30% vs. controls
  - Peri-contusional brain with most impressive response.
- Significantly improved markers of cerebral function especially in peri-contusional brain

Acute, Severe Tbi: Rockswald et al

- An absolute 26% reduction in mortality for the combined HBO2/NBH group (p = 0.048)
  - 16% of patients died in HBOT group
  - 42% died in the control group
- An absolute 36% improvement in favorable outcome
- Phase III study recommended for confirmation of results (It was only 3 HBOT sessions!!)

Effrati - 2013: HBOT Improves Post Concussion Syndrome

OPEN ACCESS Freely available online

PLOS ONE

Hyperbaric Oxygen Therapy Can Improve Post Concussion Syndrome Years after Mild Traumatic Brain Injury - Randomized Prospective Trial

Rahav Boussi-Gross[1,3], Haim Golan[3,4,5], Gregori Fishlev[1], Yair Bechor[1], Olga Volkov[3,4], Jacob Bergan[1], Mony Friedman[1], Dan Hoofien[6,7], Nathan Shlamkovitch[8], Eshel Ben-Jacob[2,5,9,10]*, Shai Efrati[1,2,3,10]*

1 The Institute of Hyperbaric Medicine, Assaf Harofeh Medical Center, Zerifin, Israel, 2 Research and Development Unit, Assaf Harofeh Medical Center, Zerifin, Israel, 3 Sackler School of Medicine, Tel-Aviv University, Tel-Aviv, Israel, 4 Nuclear Medicine Institute, Assaf Harofeh Medical Center, Zerifin, Israel, 5 The Raymond and Beverly Sackler Faculty of Exact Sciences, School of Physics and Astronomy, Tel-Aviv University, Tel-Aviv, Israel, 6 Department of Psychology, The Hebrew University of Jerusalem, Jerusalem, Israel, 7 The National Institute for the Rehabilitation of the Brain injured, Tel-Aviv, Israel, 8 Otolaryngology, Head & Neck Surgery, Assaf-Harofeh Medical Center, Zerifin, Israel, 9 Center for Theoretical Biological Physics, Rice University, Houston, Texas, United States of America, 10 Sagol School of Neuroscience, Tel-Aviv University, Tel-Aviv, Israel

Abstract

Background: Traumatic brain injury (TBI) is the leading cause of death and disability in the US. Approximately 70-90% of the TBI cases are classified as mild, and up to 25% of them will not recover and suffer chronic neurocognitive impairments. The main pathology in these cases involves diffuse brain injuries, which are hard to detect by anatomical imaging yet noticeable in metabolic imaging. The current study tested the effectiveness of Hyperbaric Oxygen Therapy (HBOT) in improving brain function and quality of life in mTBI patients suffering chronic neurocognitive impairments.

Methods and Findings: The trial population included 56 mTBI patients 1–5 years after injury with prolonged post-concussion syndrome (PCS). The HBOT effect was evaluated by means of prospective, randomized, crossover controlled trial: the patients were randomly assigned to treated or crossover groups. Patients in the treated group were evaluated at baseline and following 40 HBOT sessions; patients in the crossover group were evaluated three times: at baseline, following a 2-month control period of no treatment, and following subsequent 2-months of 40 HBOT sessions. The HBOT protocol included 40 treatment sessions (5 days/week), 60 minutes each, with 100% oxygen at 1.5 ATA. "Mindstreams" was used for cognitive evaluations, quality of life (QOL) was evaluated by the EQ-5D, and changes in brain activity were assessed by SPECT imaging. Significant improvements were demonstrated in cognitive function and QOL in both groups following HBOT but no significant improvement was observed following the control period. SPECT imaging revealed elevated brain activity in good agreement with the cognitive improvements.

Conclusions: HBOT can induce neuroplasticity leading to repair of chronically impaired brain functions and improved quality of life in mTBI patients with prolonged PCS at late chronic stage.

Trial Registration: ClinicalTrials.gov NCT00715052

Plots One. 2013 Nov 15;8(11):e79995. doi: 10.1371/journal.pone.0079995. eCollection 2013.

Hyperbaric oxygen therapy can improve post concussion syndrome years after mild traumatic brain injury - randomized prospective trial.

Boussi-Gross R[1], Golan H, Fishlev G, Bechor Y, Volkov O, Bergan J, Friedman M, Hoofien D, Shlamkovitch N, Ben-Jacob E, Efrati S.

METHODS AND FINDINGS:

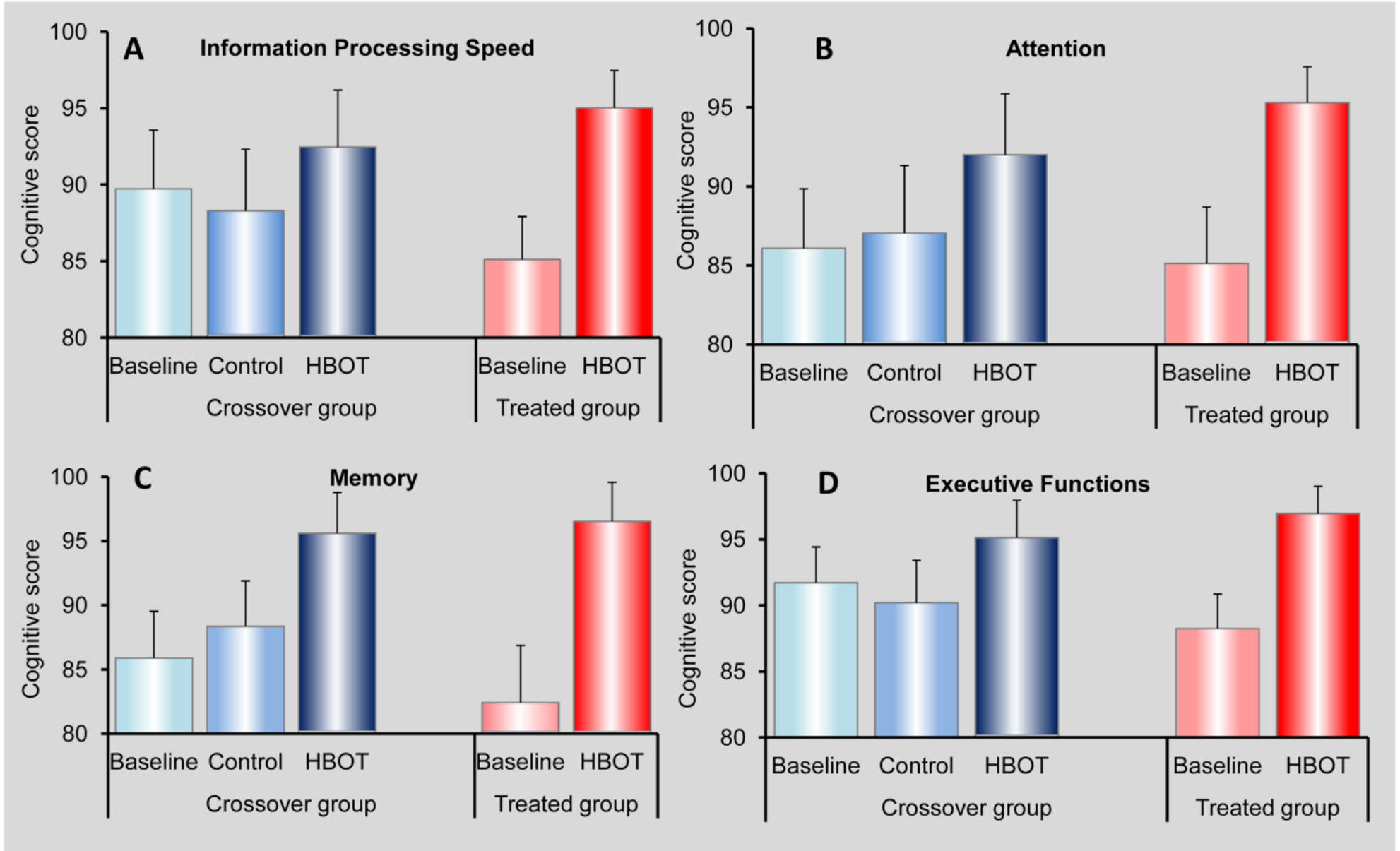
The trial population included 56 mTBI patients 1-5 years after injury with prolonged post-concussion syndrome (PCS). The HBOT effect was evaluated by means of prospective, randomized, crossover controlled trial: the patients were randomly assigned to treated or crossover groups. Patients in the treated group were evaluated at baseline and following 40 HBOT sessions; patients in the crossover group were evaluated three times: at baseline, following a 2-month control period of no treatment, and following subsequent 2-months of 40 HBOT sessions. The HBOT protocol included 40 treatment sessions (5 days/week), 60 minutes each, with 100% oxygen at **1.5 ATA**. "Mindstreams" was used for cognitive evaluations, quality of life (QOL) was evaluated by the EQ-5D, and changes in brain activity were assessed by SPECT imaging. Significant improvements were demonstrated in cognitive function and QOL in both groups following HBOT but no significant improvement was observed following the control period. SPECT imaging revealed elevated brain activity in good agreement with the cognitive improvements.

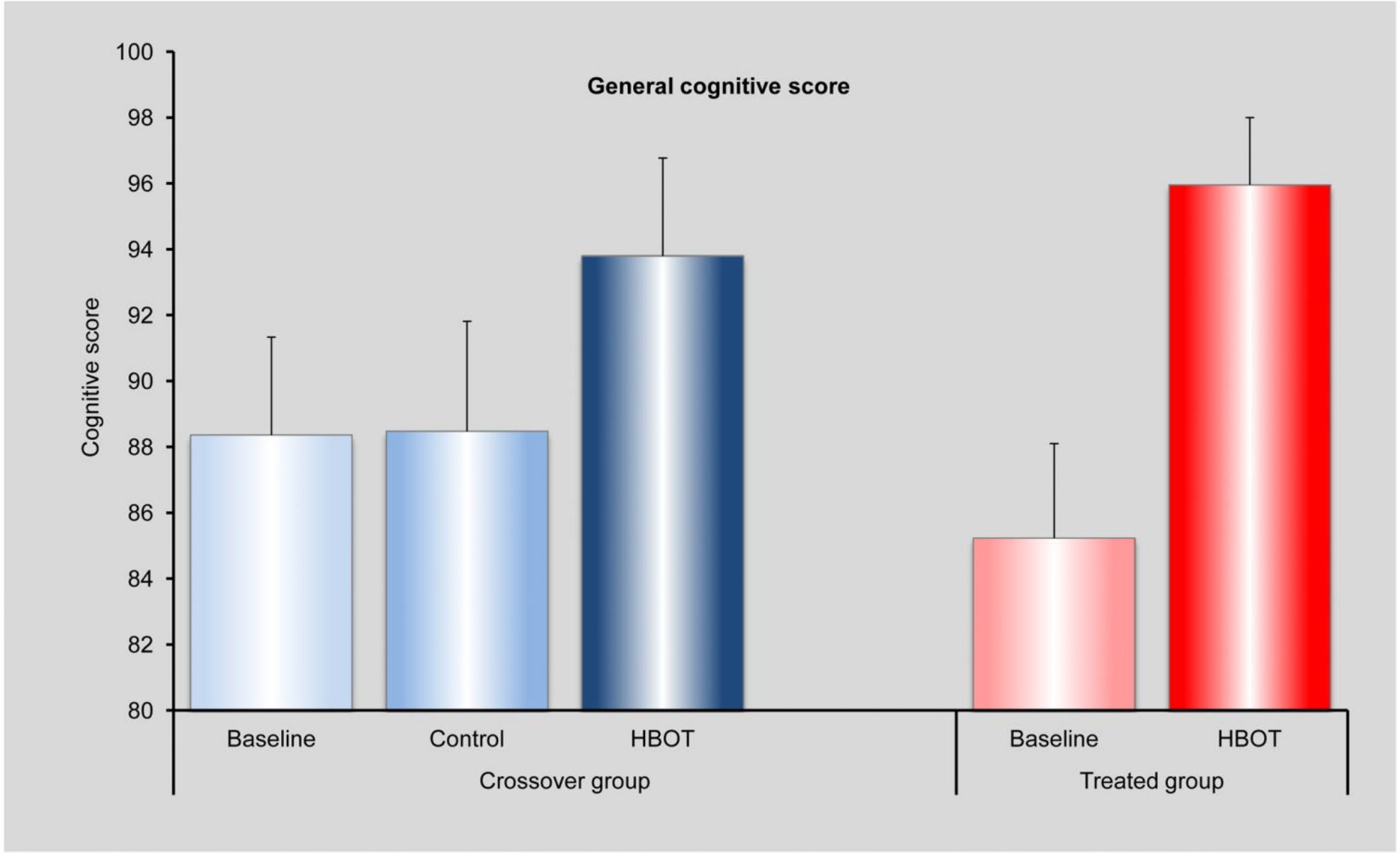
CONCLUSIONS:

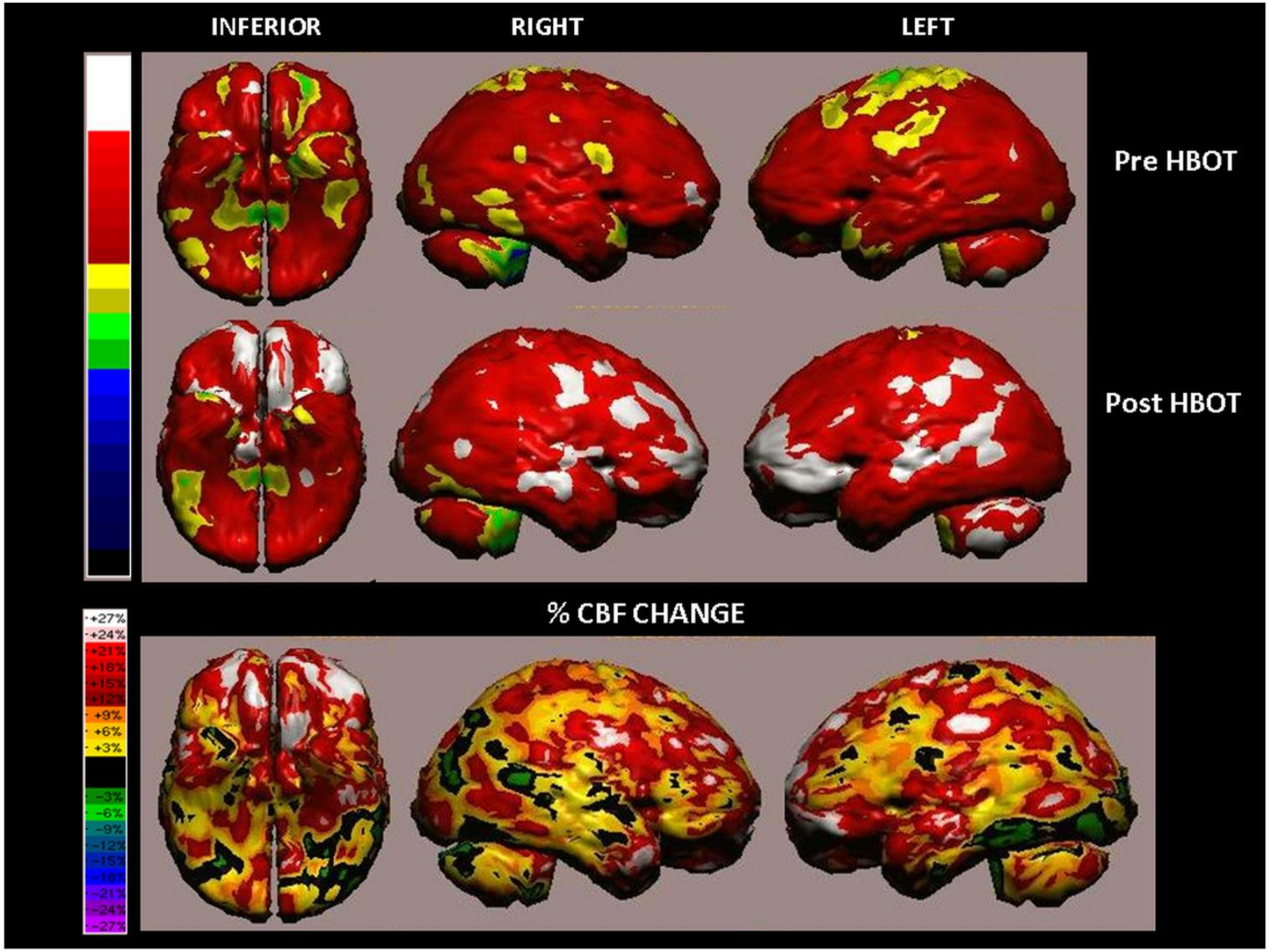
HBOT can induce neuroplasticity leading to repair of chronically impaired brain functions and improved quality of life in mTBI patients with prolonged PCS at late chronic stage.

Hbot & mTBI: Boussi-Gross

- Randomized Cross Over Controlled Study
- 56 patients with Post Concussive Syndrome (mTBI)
- 1 to 6 years post traumatic event (civilian)
- 40 sessions of HBOT
- 100% Oxygen at 1.5 ATA x 60 minutes
- SPECT Scans before & 1 to 3 weeks after treatment (blinded)


A
Information Processing Speed
Cognitive score
100
95
90
85
80
Baseline
Control
HBOT
Crossover group
Baseline
HBOT
Treated group
B
Attention
C
Memory
D
Executive Functions


General cognitive score
Cognitive score
100
98
96
94
92
90
88
86
84
82
80
Baseline
Control
HBOT
Crossover group
Baseline
HBOT
Treated group


INFERIOR
RIGHT
LEFT
Pre HBOT
Post HBOT
% CBF CHANGE
+27%
+24%
+21%
+18%
+15%
+12%
+9%
+6%
+3%
-3%
-6%
-9%
-12%
-15%
-18%
-21%
-24%
-27%

Crestor Neurol Neurosci. **2015**;33(6):943-51. doi: 10.3233/RNN-150585.
Hyperbaric oxygen may induce angiogenesis in patients suffering from prolonged post-concussion syndrome due to traumatic brain injury.
Tal S[1,2], Hadanny A[3,1], Berkovitz N[1,2], Sasson E[4], Ben-Jacob E[5,6,7], Efrati S[3,1,5,6].

PURPOSE:
Recent clinical studies present convincing evidence that hyperbaric oxygen therapy (HBOT) may be the coveted neurotherapeutic method for brain repair. One of the most interesting ways in which HBOT can induce neuroplasticity is angiogenesis. The objective in this study was to assess the neurotherapeutic effect of HBOT in post TBI patients using brain perfusion imaging and clinical cognitive functions.
METHODS:
Retrospective analysis of patients suffering from chronic neuro-cognitive impairment from TBI treated with HBOT. The HBOT protocol included 60 daily HBOT sessions, 5 days per week. All patients had pre and post HBOT objective computerized cognitive tests (NeuroTrax) and brain perfusion MRI.
RESULTS:
Ten post-TBI patients were treated with HBOT with mean of 10.3±3.2 years after their injury. After HBOT, whole-brain perfusion analysis showed significantly increased cerebral blood flow and cerebral blood volume. Clinically, HBOT induced significant improvement in the global cognitive scores ($p=0.007$). The most prominent improvements were seen in information processing speed, visual spatial processing and motor skills indices.

CONCLUSION:
HBOT may induce cerebral angiogenesis, which improves perfusion to the chronic damage brain tissue even months to years after the injury.

TBI: No Need to Die!

- HBOT2017
  11th International Symposium

- A review of HBOT in Acute Severe Traumatic Brain Injury with an Extension to Acute Concussion, and an Update on Chronic Mild TBI

  -Paul G. Harch, MD

BIMA: Weaver - A Randomized trial of hyperbaric oxygen in U.S. Service Members with post-concussive symptoms

- RTC trial -71 patients received **1.5 ATA** 100% vs 1.2ATA 21%
- Followed symptom's, quality of life, neuropsych testing, EEG, sleep, audiology, visual, lab testing, and brain imaging, at 13 weeks
- And symptom questionnaire at 6mo and 12 month.
- Found improvement in symptoms, processing speed, sleep quality, and vestibular symptoms.....with greater response in the PTSD patients.
- All regressed at 6 and 12 months.

Front. Hum. Neurosci, October 2017

Hyperbaric Oxygen Therapy Can Induce Angiogenesis and Regeneration of Nerve Fibers in Traumatic Brain Injury Patients

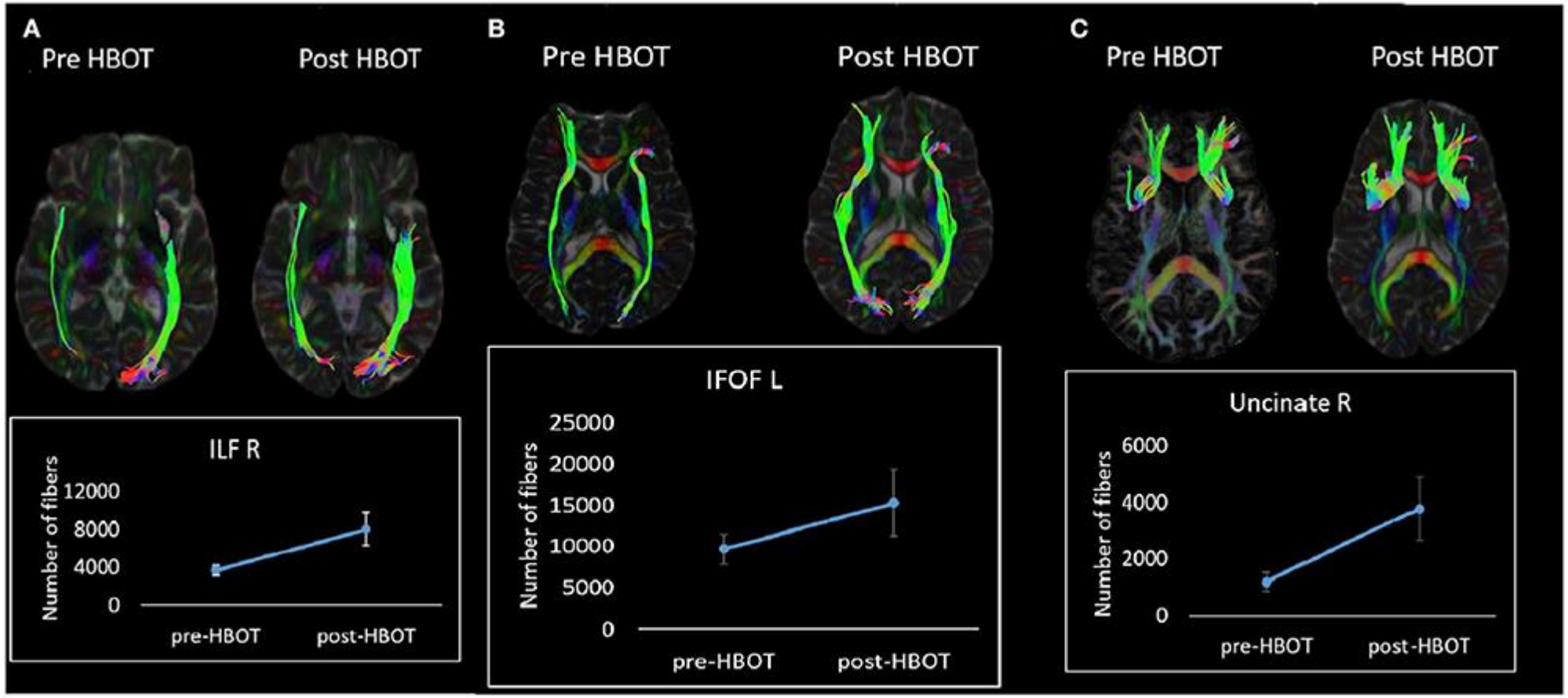
Sigal Tal, Amir Hadanny, Efrat Sasson, Gill Suzin and Shai Efrati

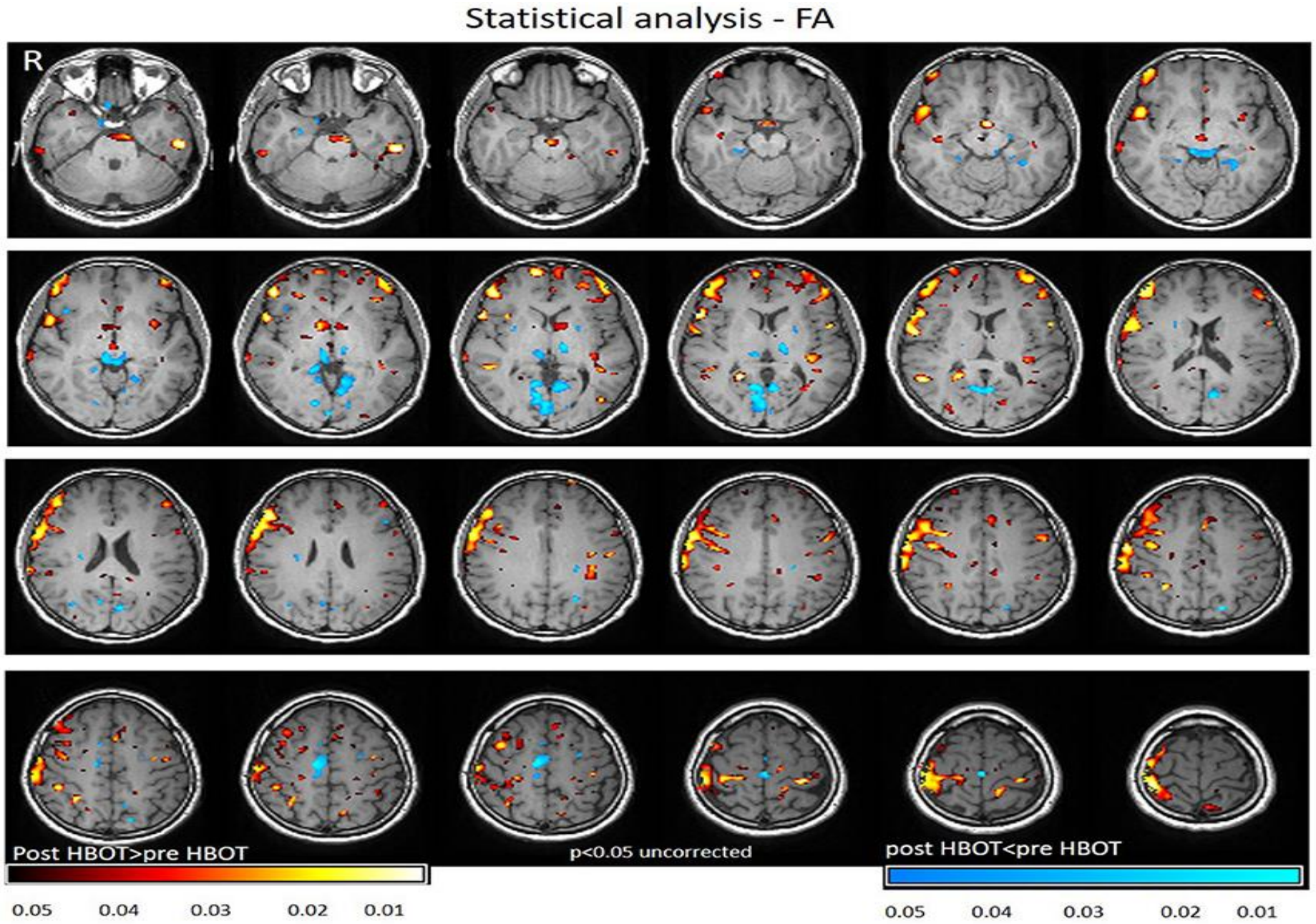
- Sackler School of Medicine, Tel-Aviv University, Tel-Aviv, Israel
- Radiology Department, Assaf Harofeh Medical Center, Zerifin, Israel
- Sagol Center for Hyperbaric Medicine and Research, Assaf Harofeh Medical Center, Zerifin, Israel
- Faculty of Medicine, Bar-Ilan University, Ramat Gan, Israel
- WiseImage, Hod Hasharon, Israel
- Research and Development Unit, Assaf Harofeh Medical Center, Zerifin, Israel
- Sagol School of Neuroscience, Tel-Aviv University, Tel-Aviv, Israel

15 patients treated x60 sessions at 2.0 ATA x 90min

DTI analysis

Conclusions: The mechanisms by which HBOT induces brain neuroplasticity can be demonstrated by highly sensitive MRI techniques of DSC and DTI. HBOT can induce cerebral angiogenesis and improve both white and gray microstructures indicating regeneration of nerve fibers. The micro structural changes correlate with the neurocognitive improvements





Yellow/red show increase FA

Conclusion

HBOT can induce cerebral angiogenesis and recovery of brain microstructure in patients with chronic cognitive impairments due to TBI months to years after the acute injury. The increased integrity of brain fibers correlates with the functional cognitive improvement. The mechanism by which HBOT can induce brain neuroplasticity can be demonstrated by highly sensitive perfusion MRI and DTI. Further studies, using DTI - MRI, are needed in order to gain better understanding of the neuroplasticity effect of HBOT in a larger cohort of patients with different types of brain injuries.

What About Dose?

- Reviewed multiple trials ranging from 1.3 to 2.4

- Dr. Daphne Denham, reports that 98% of her patients [51 out of 52] treated within ten days of suffering a concussion, completely resolved their symptoms in five treatments or less when treated with escalating dose.

The Evaluation

History

Red Flags!

- Go to emergency department right away if:
  - Look very drowsy or cannot be awakened
  - One pupil (the black part in the middle of the eye) is larger than the other
  - Have convulsions or seizures
  - Cannot recognize people or places
  - Increasingly confused, restless, or agitated; rapidly worsening headache
  - Exhibits unusual behavior
  - Lose consciousness
    (a brief loss of consciousness should be taken seriously and the person should be carefully monitored)
  - Weakness, numbness or decreased coordination
  - Repeated vomiting or nausea
  - Slurred speech

90 % mTBI have oculomotor dysfunction

39% mTBI have visual field deficits

43-46% mTBI have convergence insaufficiency

Neurologic Examination

- Cognition/ Mental Status
- Orientation (day, date, time, month, year)
- Immediate memory (5 items, 3 trials)
- Delayed recall (5 items after 5 minutes)
- Concentration (3, 4, and 5 digits backwards, months/WORLD backwards, serial sevens)
- Affect
- Coordination
- Finger-nose-finger/ Finger-to-nose
- Heel-to-shin
- Rapid finger movements
- CN testing:
- EOM evaluation (nystagmus, convergence)
- Speech
- Visual Fields
- Pupils
- VOMS (Vestibular/Ocular-Motor Screening)
- Balance assessment
- Modified BESS/ single leg stance
- Tandem gait
- Rhomberg test

PERRLA

Physical Examination

Dysfunction of Visual Accommodation/Convergence

Visual Accommodation

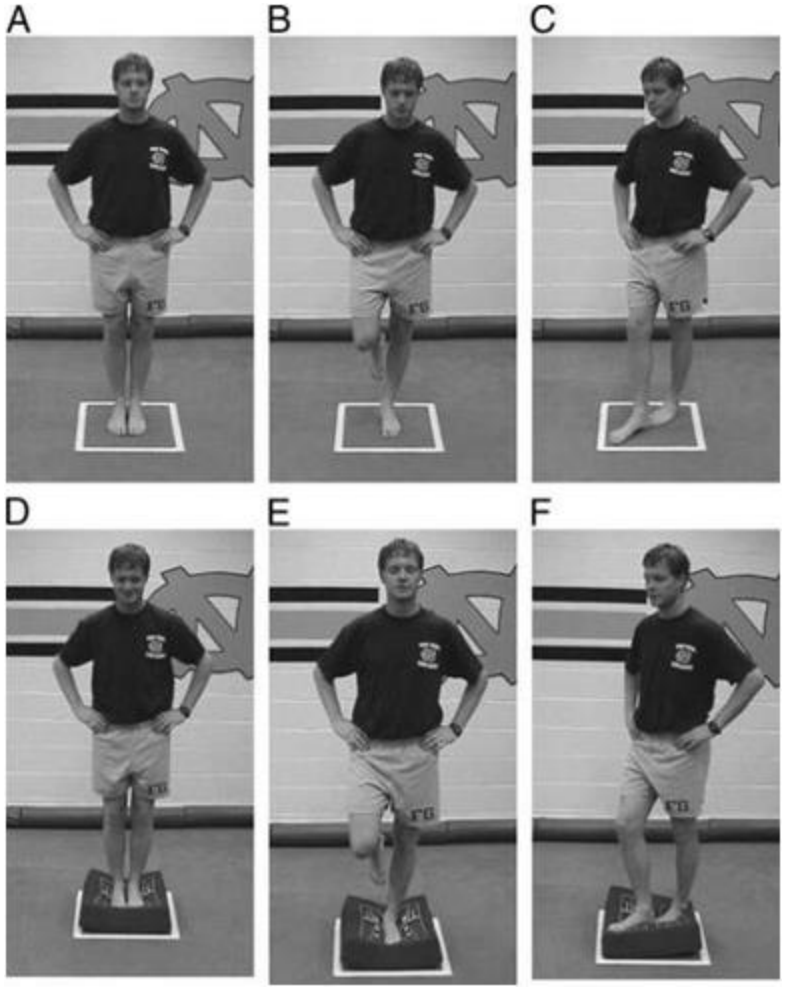
- **NPA** (Near point of accommodation)
  - Slowly move letters closer to the eye until they become blurry. Measure the distance the letters became blurry. This is the near point of accommodation.
  - Increased with dysfunction and age. Normal NPA of approximately 7cm from the bridge of the nose.

- **NPC** (Near point of convergence)
  - Note when patients lose ability maintain binocular vision
  - >6cm abnormal

BESS
Balance Error Scoring System

Screening for postural stability

- Double Leg, non-dominant leg, tandem stance
- Eyes closed
- Hard floor surface & foam surface
- Count “errors” or out of position moments:
  - Lifting hands off hips
  - Opening eyes
  - Step, stumble, or fall
  - More than 30 degrees abduction or flexion
  - Lifting foot or heel



http://www.csmfoundation.org/Concussion_Balance_Testing.html (Collegiate Sports Medicine Foundations)

NAMASTE

Vestibular/Oculo-Motor Screen

- Smooth Pursuit: follows a moving target while seated (3 ft from pt)
- Saccades: quickly follow a target between two points (3ft away, 1.5 ft to right/left OR above/below eye level)
- Convergence: view a near target without double vision (target at arms length moving toward nose, >6cm is abnormal)
- Vestibulo-ocular reflex*: ability to stabilize vision as the head moves (focus on object 3 ft away while moving head)
- Visual Motion Sensitivity*: ability to inhibit vestibular –induced eye movements using vision (rotate head and arm focus on thumb)

VOMS SCORING SHEET

Symptoms on a 0-10 point scale

Vestibular/ Oculomotor	Type	Not Tested	Headache	Dizziness	Nausea	Fogginess	Comments
Baseline Symptoms							
Smooth Pursuit							
Saccades (Horizontal)							
Saccades (Vertical)							
Convergence (Near Point)							Score#1_____cm Score#2_____cm Score#3_____cm
VOR Horizontal							
VOR Vertical							
Visual Motion Sensitivity							

(Mucha, Collins et al. 2014)

Gaze Stability Exercises

Fixed/Moving targets

Variable distances from targets

Simple to complex visual backgrounds

Simple to complex surfaces during:

- Sitting
- Standing
- Gait

What Structures Are Injured to the Vestibular System After Concussion?

- Actual sensors (otolith) or entire end organ gets damaged
  - Baro-trauma, blunt injury, blast/shockwave from hit
- Traction/tethering of CNVIII (vestibular-cochlear nerve)
  - From the origin of the sensor
  - In the axons of the nerve itself
  - From the insertion in the brainstem

The Vestibular – Cognition Connection

Journal of Vestibular Research 16 (2006) 75–91
IOS Press

Cognitive-vestibular interactions: A review of patient difficulties and possible mechanisms

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Neuro-otology Department, Legacy Research Center, Portland, OR, USA

Received 1 December 2005
Accepted 5 October 2006

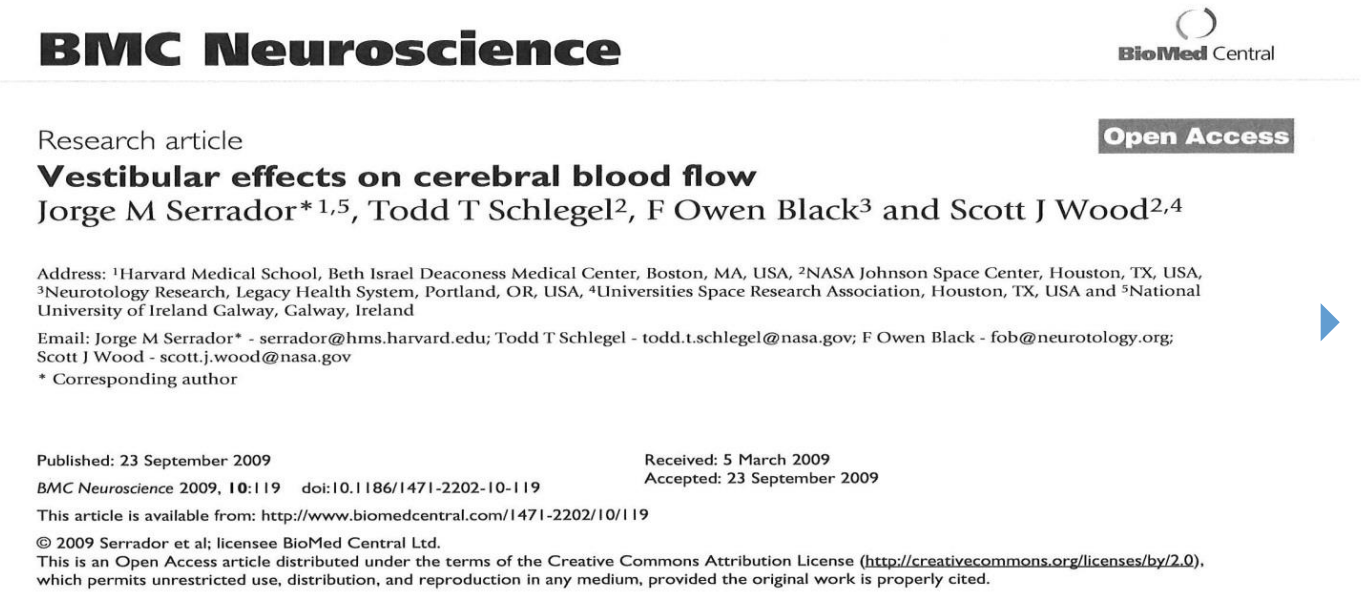
Abstract. Cognitive deficits such as poor concentration and short-term memory loss are known by clinicians to occur frequently among patients with vestibular abnormalities. Although direct scientific study of such deficits has been limited, several types of investigations do lend weight to the existence of vestibular-cognitive effects. In this article we review a wide range of studies indicating a vestibular influence on the ability to perform certain cognitive functions. In addition to tests of vestibular patient abilities, these studies include dual-task studies of cognitive and balance functions, studies of vestibular contribution to spatial perception and memory, and works demonstrating a vestibular influence on oculomotor and motor coordination abilities that are involved in the performance of everyday cognitive tasks. A growing literature on the physiology of the vestibular system has demonstrated the existence of projections from the vestibular nuclei to the cerebral cortex. The goals of this review are to both raise awareness of the cognitive effects of vestibular disease and to focus scientific attention on aspects of cognitive-vestibular interactions indicated by a wide range of results in the literature.

Keywords: Vestibular, cognitive, balance, spatial

- Damage to the vestibular system can directly create cognitive deficits
  - Spatial navigation
  - Object recognition memory
- You don't have to have symptoms of dizziness to have the cognitive symptoms

(Smith et al, 2005, Hanes, 2006 – Journal of Vestibular Research)

The Vestibular-Blood Flow Connection –



BMC Neuroscience

BioMed Central

Research article

Open Access

Vestibular effects on cerebral blood flow

Jorge M Serrador*[1,5], Todd T Schlegel[2], F Owen Black[3] and Scott J Wood[2,4]

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Email: Jorge M Serrador* - serrador@hms.harvard.edu; Todd T Schlegel - todd.t.schlegel@nasa.gov; F Owen Black - fob@neurotology.org; Scott J Wood - scott.j.wood@nasa.gov

* Corresponding author

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Accepted: 23 September 2009

This article is available from: http://www.biomedcentral.com/1471-2202/10/119

- The purpose of the otolith organ of the inner ear is assist in auto-regulation of blood flow to the head.
- Injury to this organ can lead to symptoms that commonly are thought to be cerebral deficits.

The Vestibular-Autonomic Nervous System Connection

- Vestibular system lesions produce a number of injurious effects, including:
  - Disruption in the ability to rapidly adjust blood pressure
  - Respiratory muscle activity during movement and changes in posture
- These perturbations in autonomic regulation are transient, and largely dissipate over time.
  - Could we be seeing a disruption of the vestibular system as the cause of the symptoms of concussion?

Journal of Vestibular Research 15 (2005) 119–129
IOS Press

The effects of vestibular system lesions on autonomic regulation: Observations, mechanisms, and clinical implications

Bill J. Yates[a,b,*] and Adolfo M. Bronstein[c]
[a]*Department of Otolaryngology, University of Pittsburgh, Pittsburgh, PA, USA*
[b]*Department of Neuroscience, University of Pittsburgh, Pittsburgh, PA, USA*
[c]*Division of Neurosciences and Psychological Medicine, Imperial College, London, UK*

Received 14 March 2005
Accepted 20 May 2005

Abstract. The loss of labyrinthine inputs in patients or animal models has been demonstrated to affect autonomic regulation. Considerable evidence suggests that vestibular-autonomic responses serve to adjust blood pressure and respiratory activity during movement and postural alterations. However, following peripheral vestibular lesions, compensation rapidly occurs, such that autonomic disturbances are not readily evident in patients with chronic labyrinthine dysfunction. This manuscript summarizes the evidence suggesting that vestibular inputs influence autonomic regulation, but that cardiovascular and respiratory responses linked to movement recover quickly subsequent to the loss of labyrinthine signals. In addition, the clinical implications of dysfunction of vestibulo-autonomic reflexes are described. Furthermore, the mechanisms potentially responsible for the return of the ability to produce posturally-related adjustments in blood pressure and respiration following vestibular lesions are discussed. In particular, evidence that somatosensory signals can replace labyrinthine inputs to vestibular nucleus neurons that participate in autonomic regulation is provided.

Keywords: Respiration, blood pressure, labyrinthectomy, inner ear disease, 8th cranial nerve

Treatment of Vestibular Dysfunction

- Principle I
  - Dizziness is an error message – avoidance of the symptom actually prolongs disorder.
- Principle II
  - Use the same treatment techniques you would use to treat an ankle sprain/strain
    - Repetitions and Sets
    - Small movements to dynamic movements
    - Symptoms as your guide

Neurocognitive Testing

- Objective measure for subtle cognitive impairments
- More sensitive than office examination
- Should NOT be used in isolation
- Helpful in the post concussion management of patients with persistent symptoms a and/or a more complicated course.
- Computerized testing compares to individual's pre-season baseline

Neurocognitive Testing

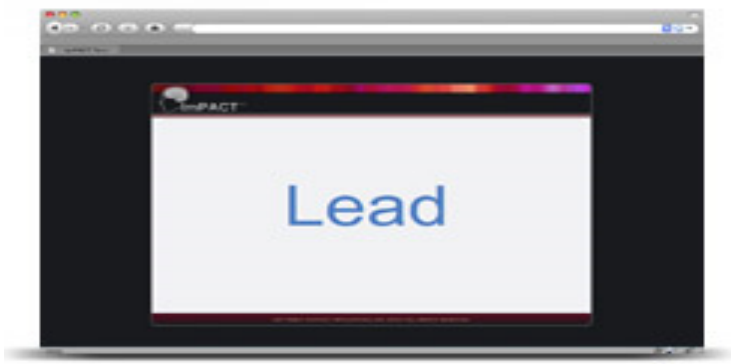
Used to provide a sensitive index of higher brain functioning by measuring:

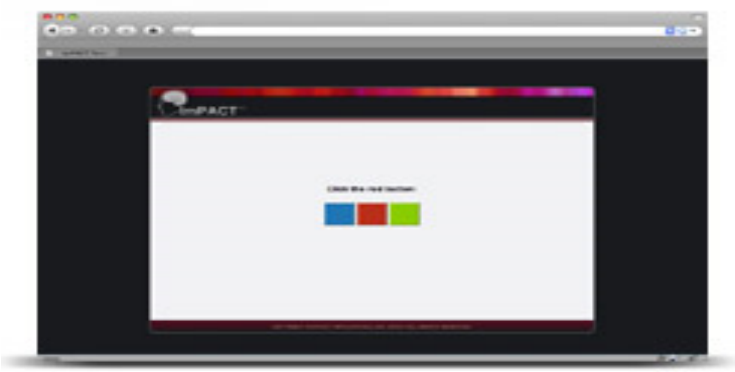
- Memory
- Attention
- Executive function
- Speed and flexibility of cognitive processing

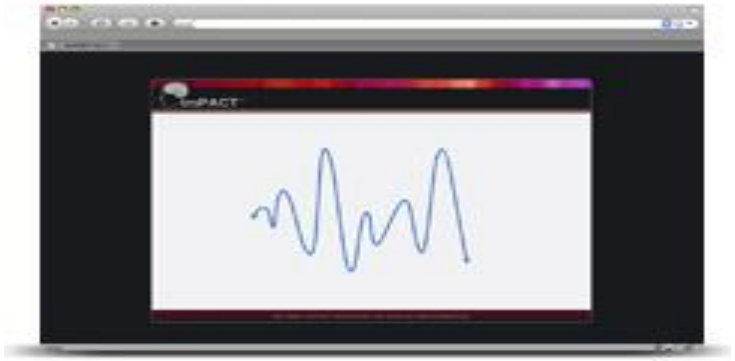
Immediate Post-Concussion Assessment and Cognitive Testing

- ImPACT testing is a 30-45 minute neurocognitive test battery that has been scientifically validated to measure the effects of sports related concussion.
- ImPACT is available for athletes age 11 to 65
- All athletes should have a baseline test prior to playing sports:
- Middle School & High School: test every two years
- College: test once
- Professional athletes: test once
- Athletes with no Baseline test: Athletes should be retested within 24-72 hours of sustaining a possible concussion.
- Athletes with a Baseline test: Athletes should be retested when they are completely symptom free.

ImPACT test:







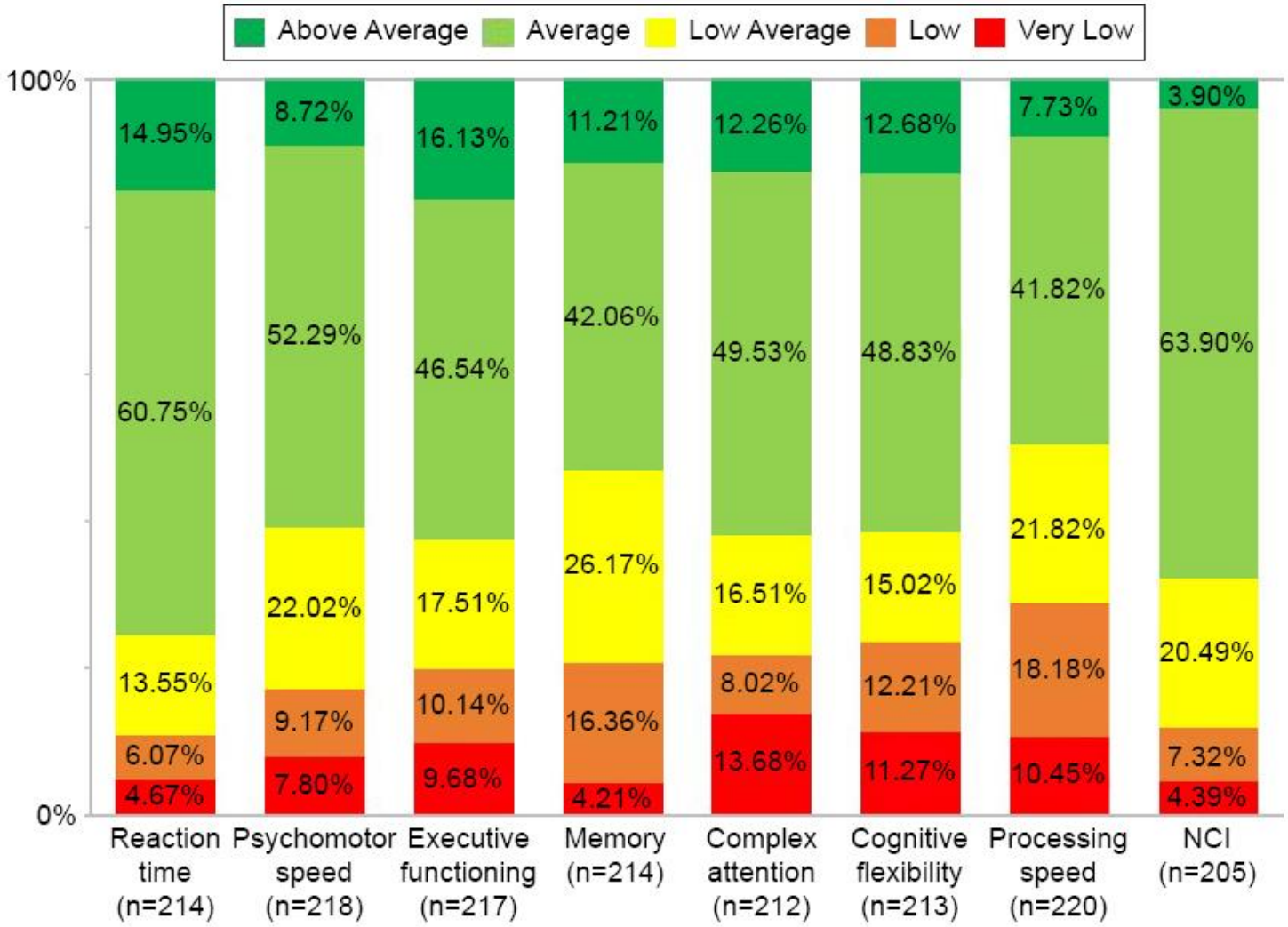


Figure 2 Percentages of patients per score category per CNSVS domain.
Abbreviations: CNSVS, central nervous system vital signs; NCI, neurocognition index.

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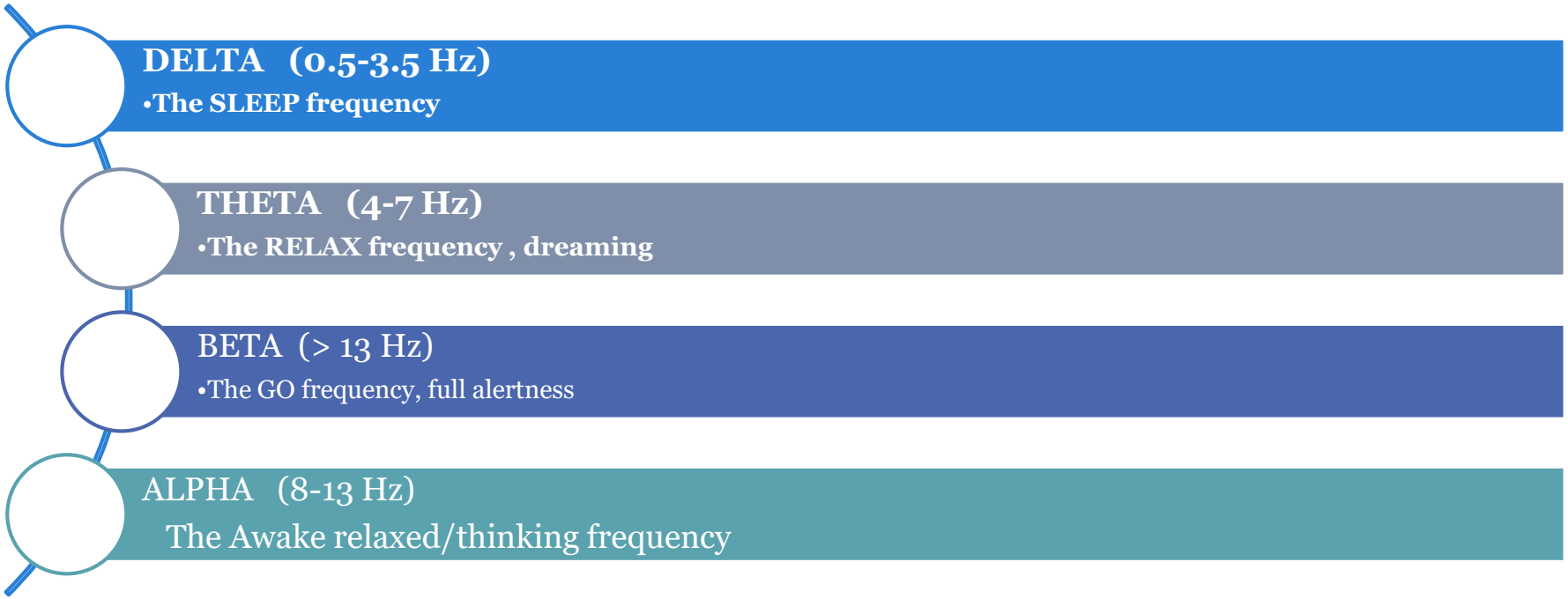
Clinical Advantages of Quantitative Electroencephalogram (QEEG)–Electrical Neuroimaging Application in General Neurology Practice

J. Lucas Koberda[1], Andrew Moses[1,2], Paula Koberda[1,2] and Laura Koberda[1,2]

Abstract

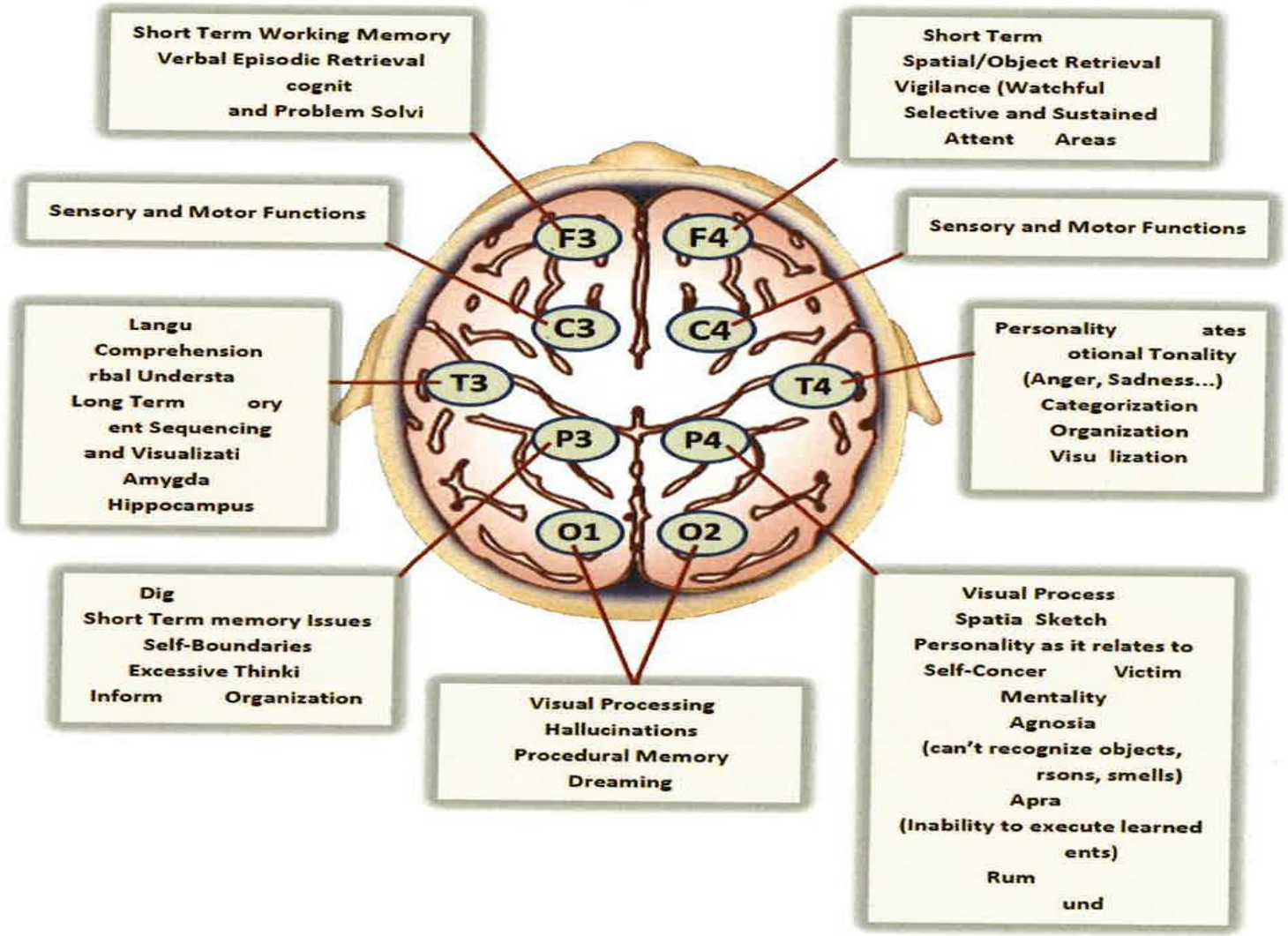
QEEG-electrical neuroimaging has been underutilized in general neurology practice for uncertain reasons. Recent advances in computer technology have made this electrophysiological testing relatively inexpensive. Therefore, this study was conducted to evaluate the clinical usefulness of QEEG/electrical neuroimaging in neurological practice. Over the period of approximately 6 months, 100 consecutive QEEG recordings were analyzed for potential clinical benefits. The patients who completed QEEG were divided into 5 groups based on their initial clinical presentation. The main groups included patients with seizures, headaches, post-concussion syndrome, cognitive problems, and behavioral dysfunctions. Subsequently, cases were reviewed and a decision was made as to whether QEEG analysis contributed to the diagnosis and/or furthered patient's treatment. Selected and representative cases from each group are presented in more detail, including electrical neuroimaging with addi

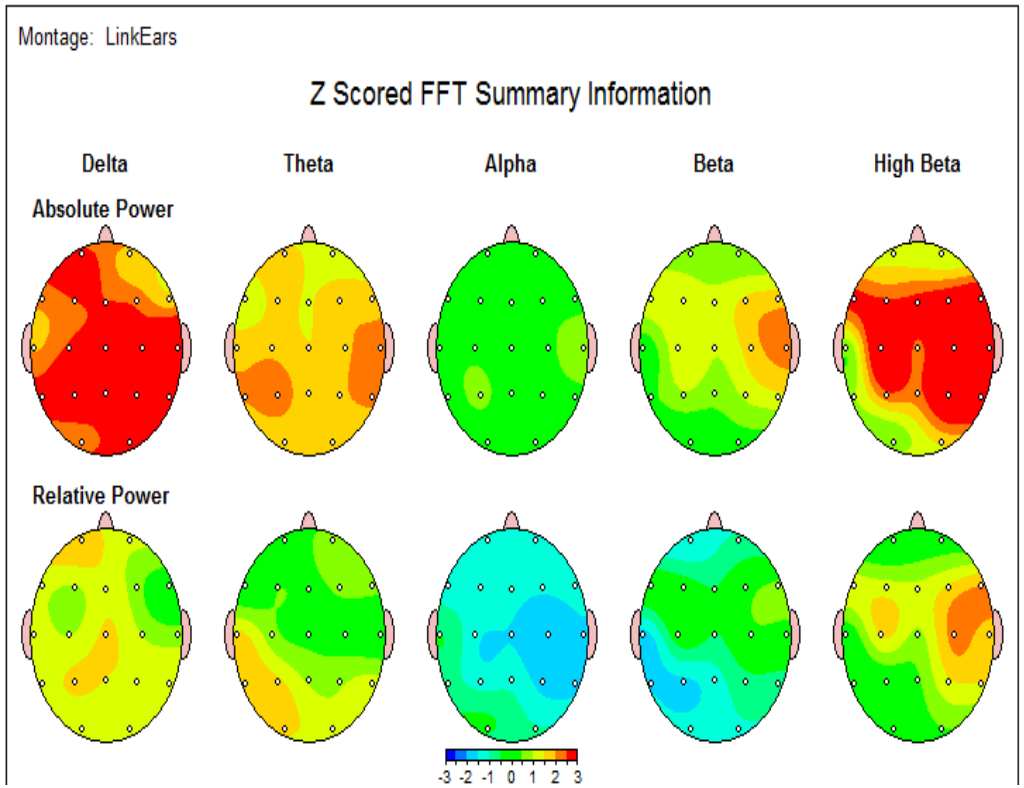


Neurotypical

Over active / Under active

QEEG SITE CORRELATIONS

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Delta
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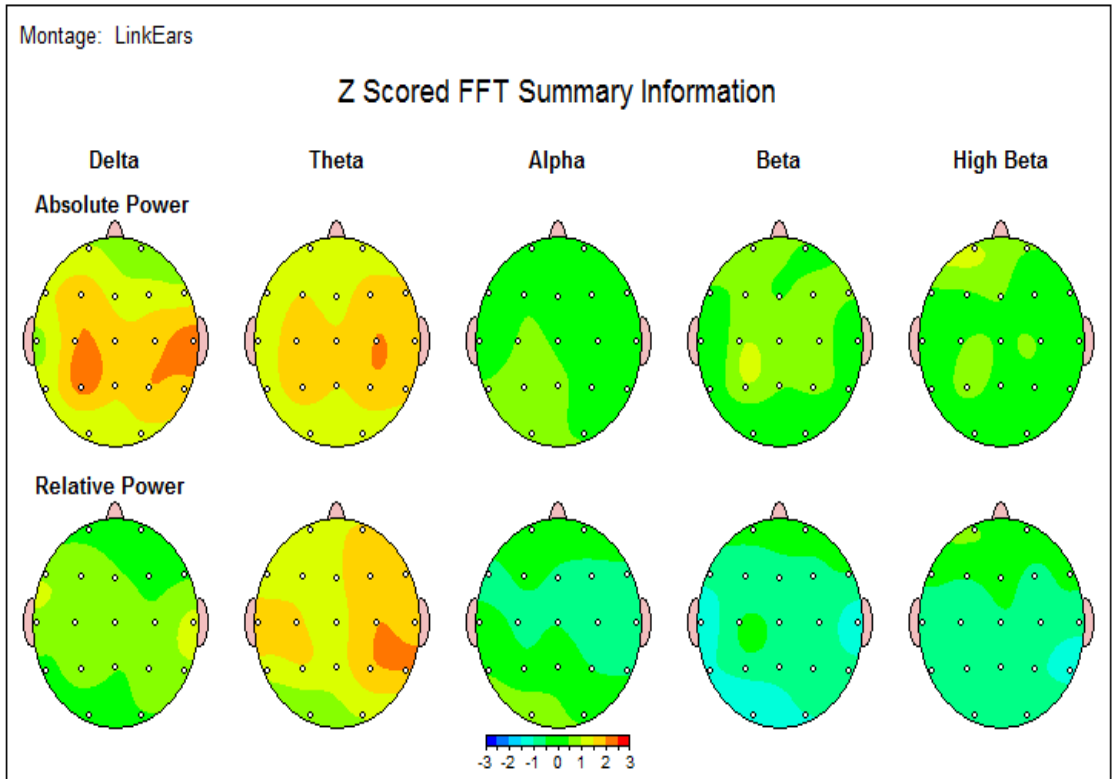
High Beta
Absolute Power
Relative Power
-3 -2 -1 0 1 2 3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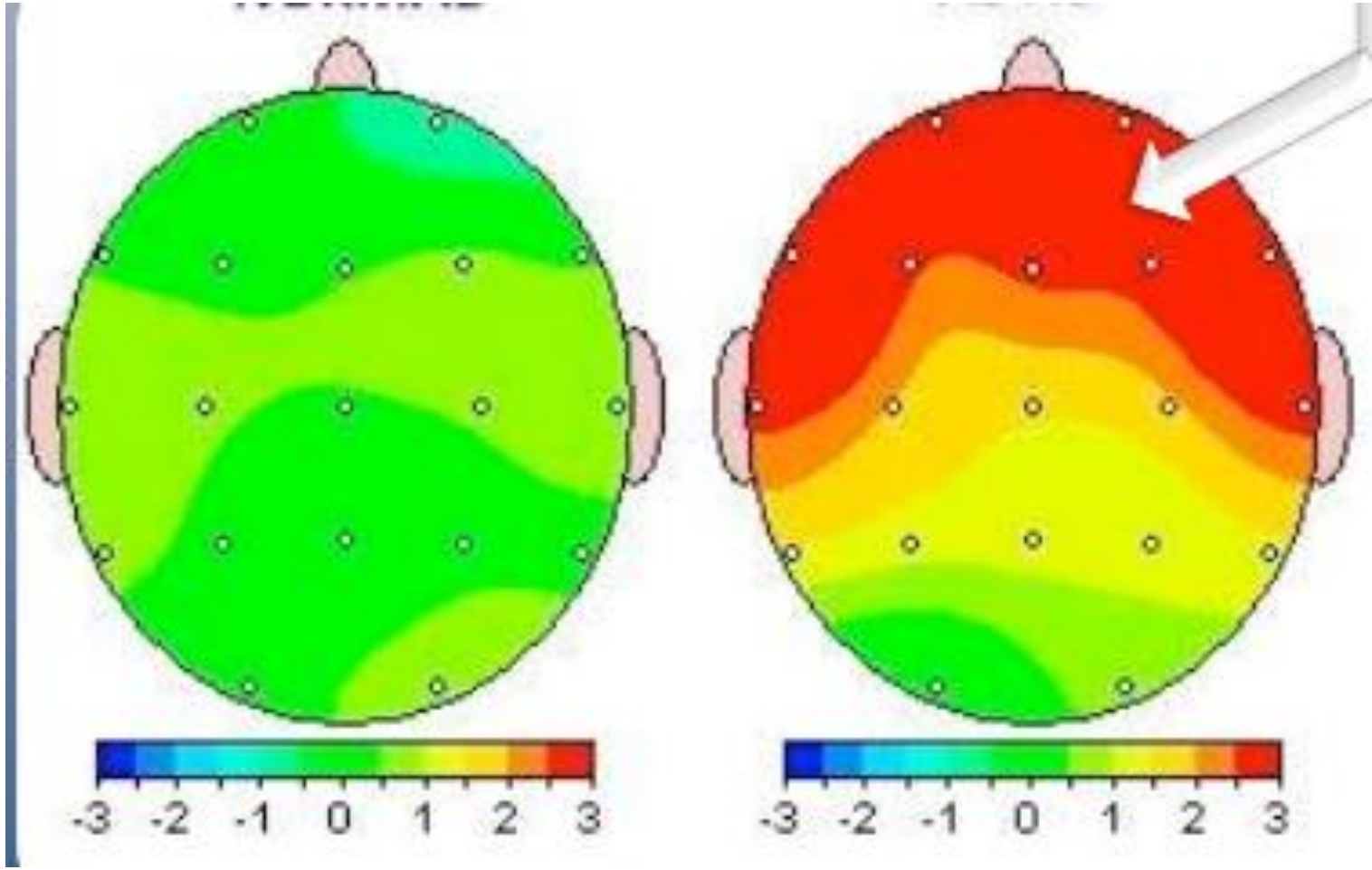
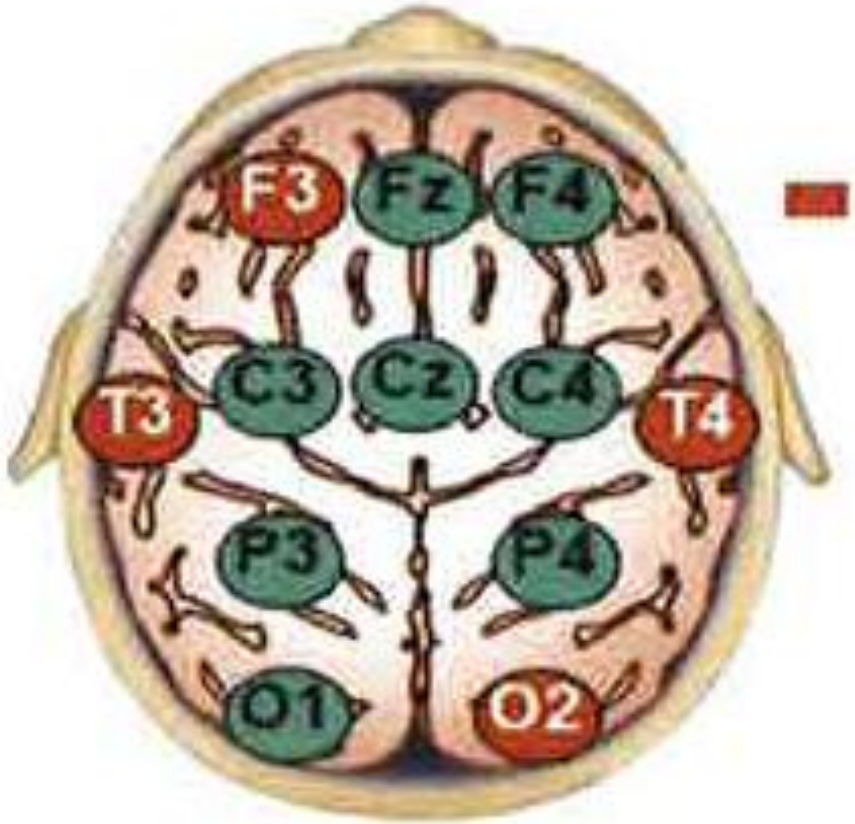
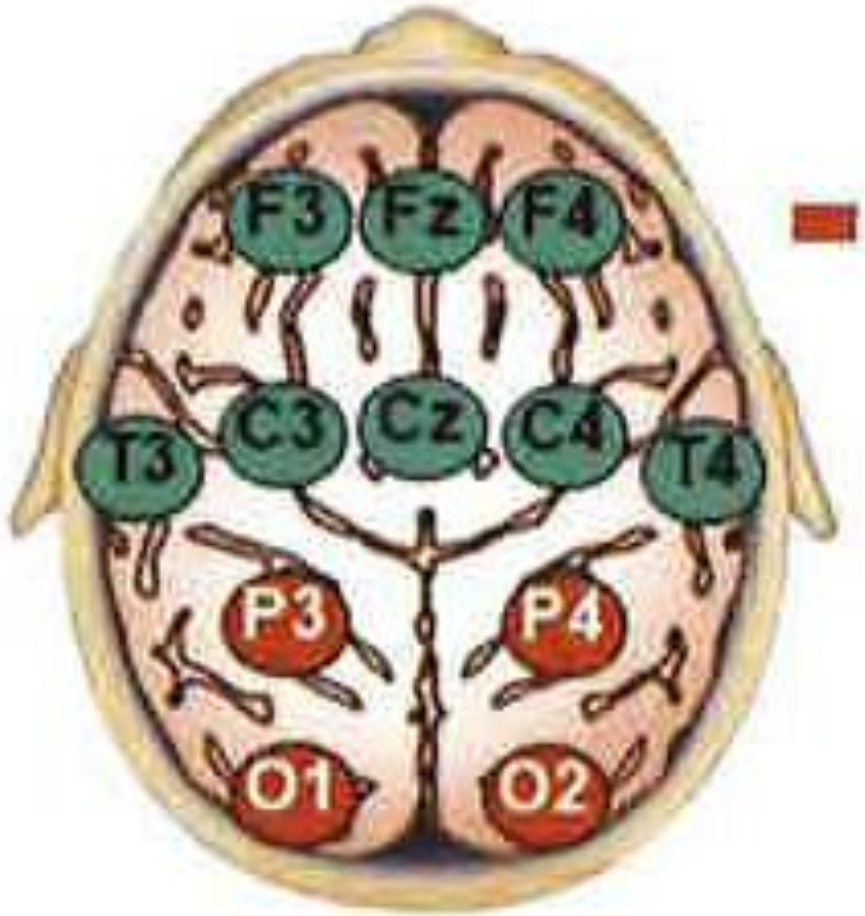


High Theta Activity

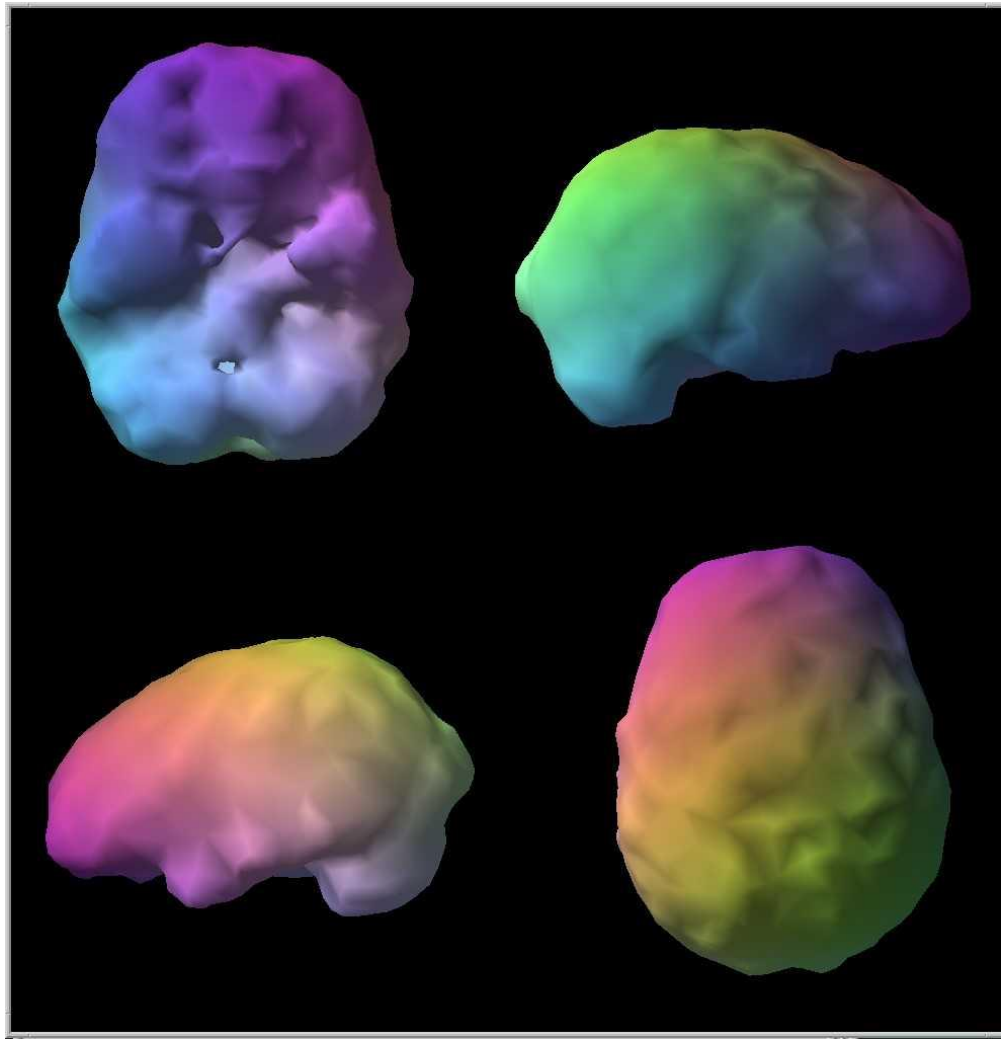


J.R. 12/5/2016



S.P.

SPECT Scan



Investigations

- Neuroimaging (CT, MRI)
  - Use when suspicion of intracerebral structural lesion exists:
    - prolonged loss of consciousness
    - focal neurologic deficit
    - worsening symptoms
    - Deterioration in conscious state

NeuroImaging

Computed Tomography (CT)

- Not recommended for routine concussion evaluation
- Sensitive for skull fracture and intracranial hemorrhage
- Test of choice in first 24-48 hours after injury
- Will not rule out chronic subdural or neurobehavioral dysfunction

Magnetic Resonance Imaging (MRI)

- Not recommended for routine concussion evaluation
- More sensitive for cerebral contusion, petechial hemorrhage, white matter injury, posterior fossa abnormalities
- Diffusion tensor imaging may detect white matter injury better

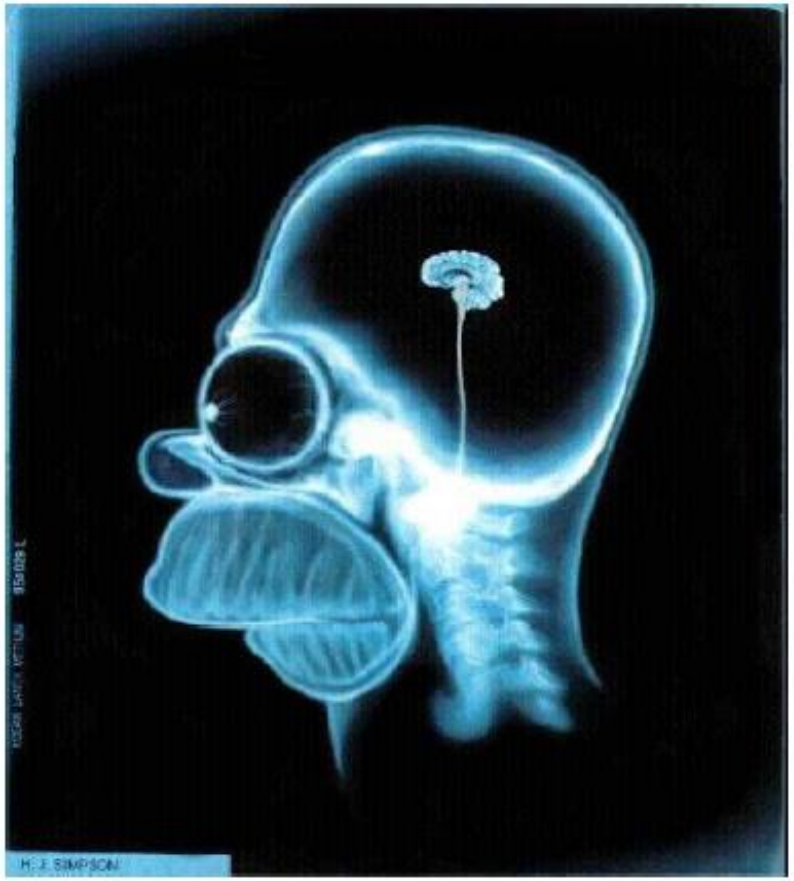
FMRI is…

Technique used for measuring metabolic correlates of neuronal activity

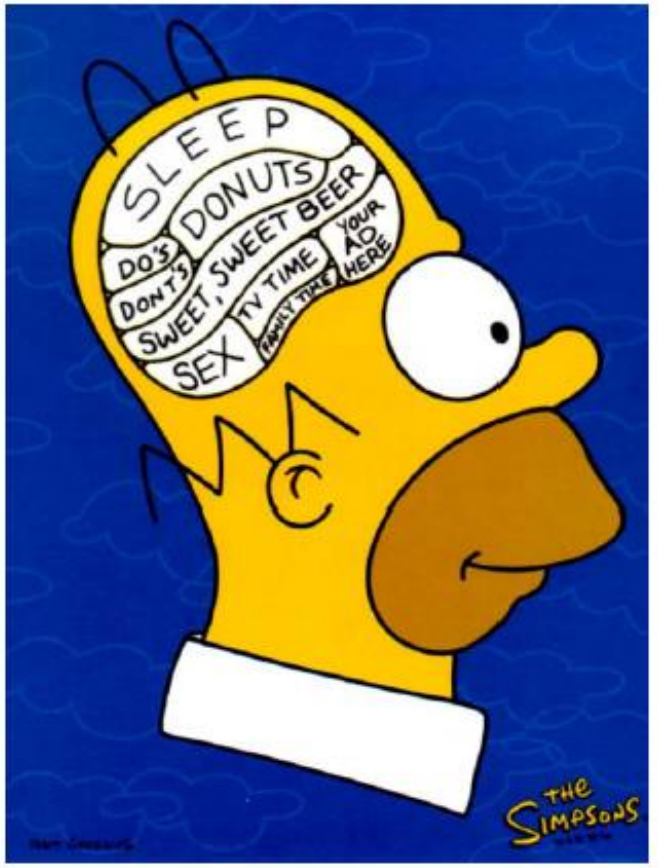
- Uses a standard MRI scanner
- Acquires a series of images
- Measures changes in blood oxygenation
- Use non-invasive, non-ionizing radiation
- Can be repeated many times; can be used for a wide range of subjects
- Combines good spatial and reasonable temporal resolution

Difference Between MRI & fMRI

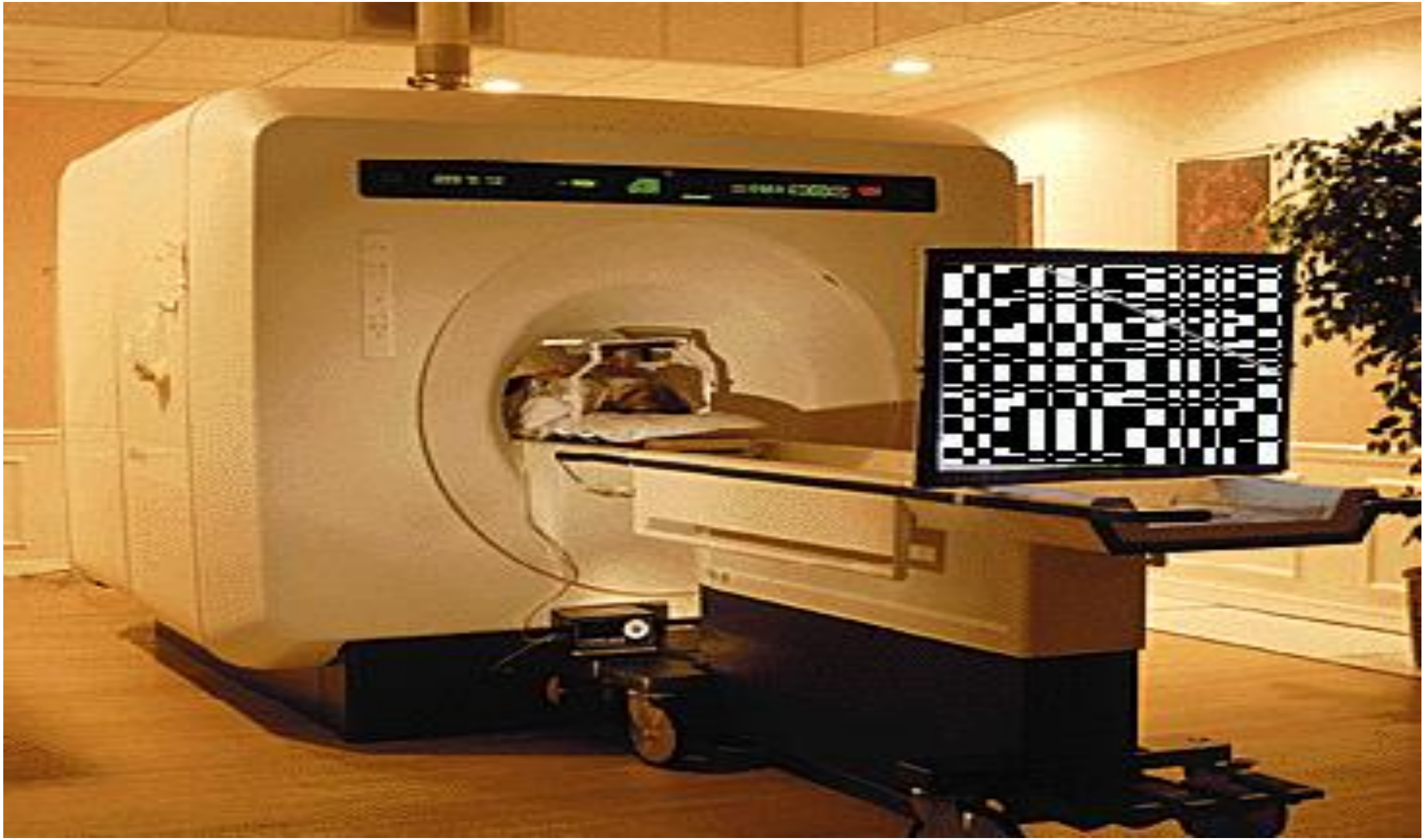
MRI studies brain anatomy.



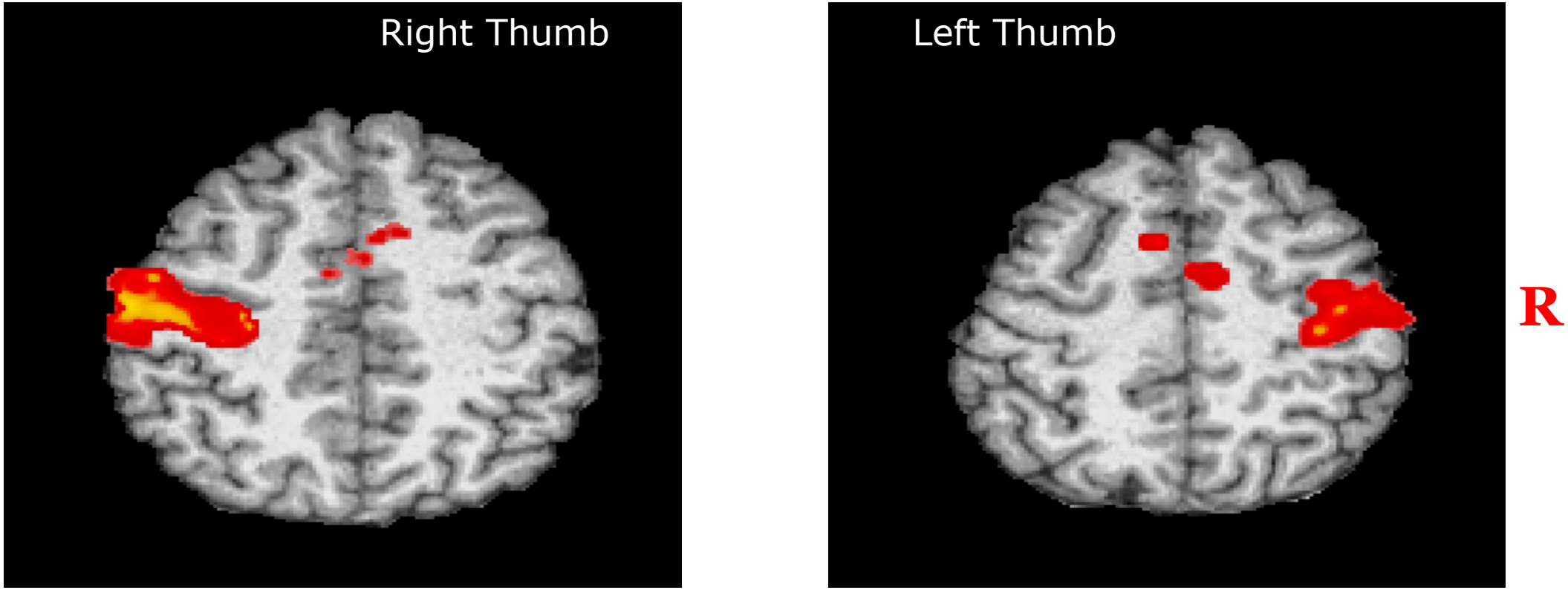
Functional MRI (fMRI) studies brain function.



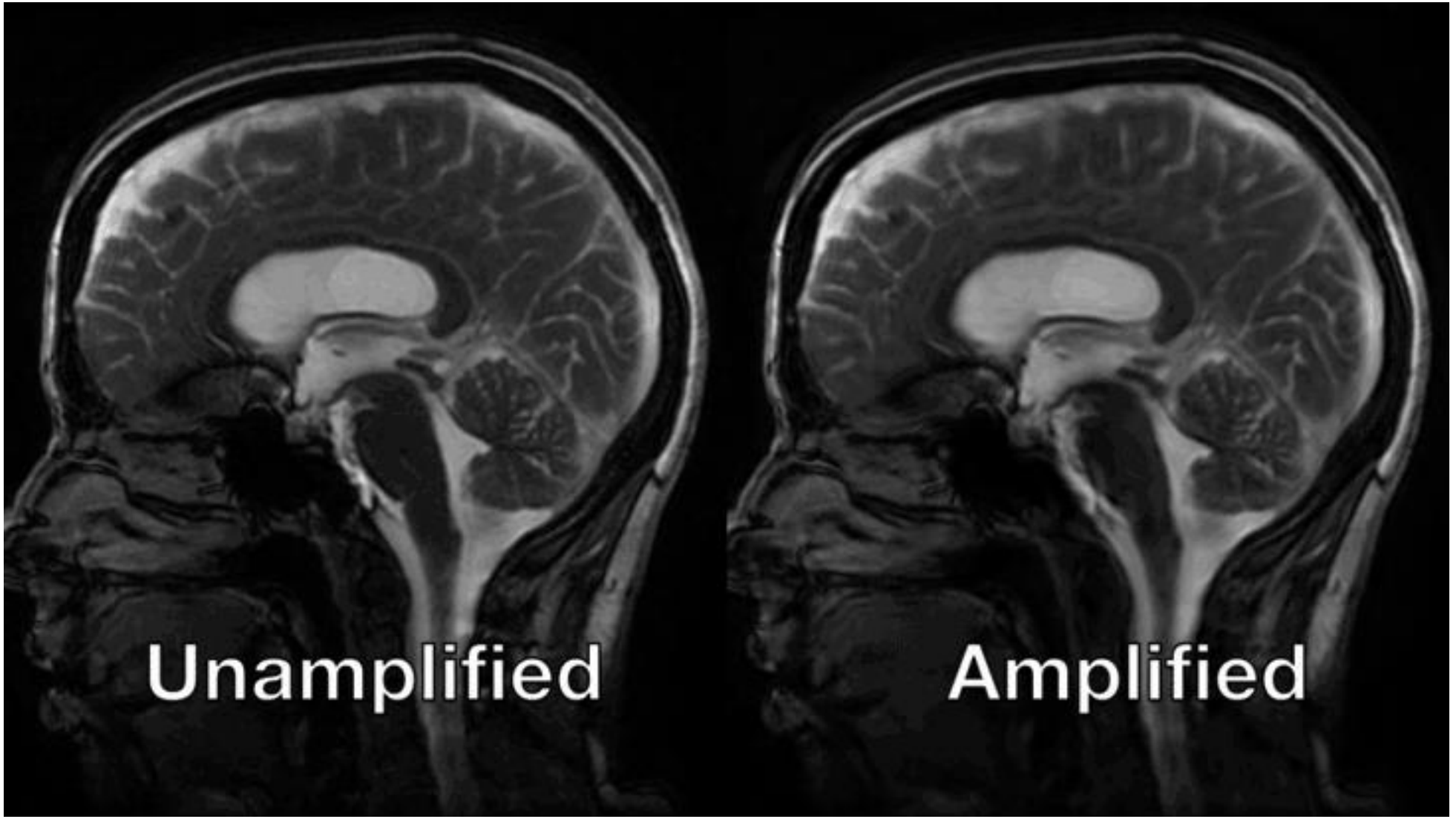
From: Daniel Bulte
Centre for Functional MRI of the Brain
University of Oxford



Unimanual Thumb Flexion



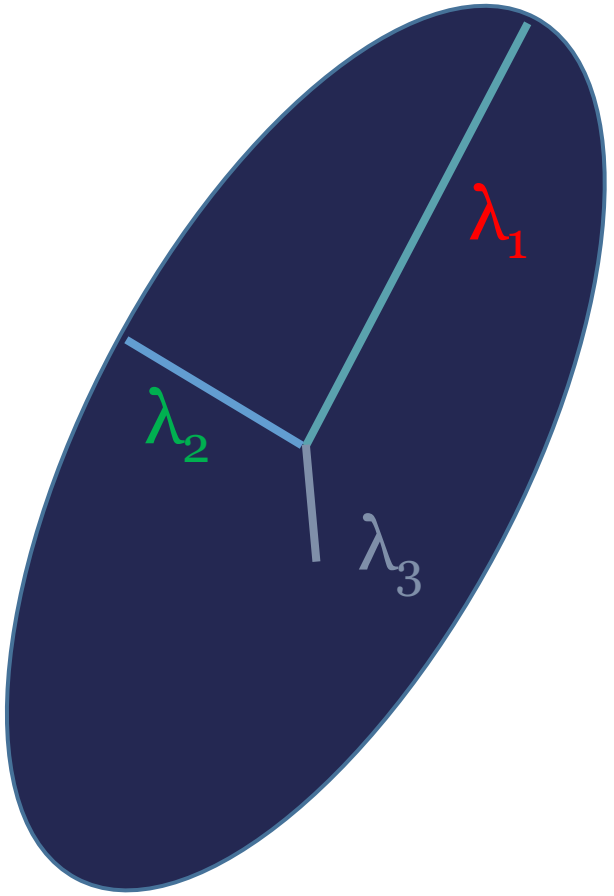
Amplified MRI



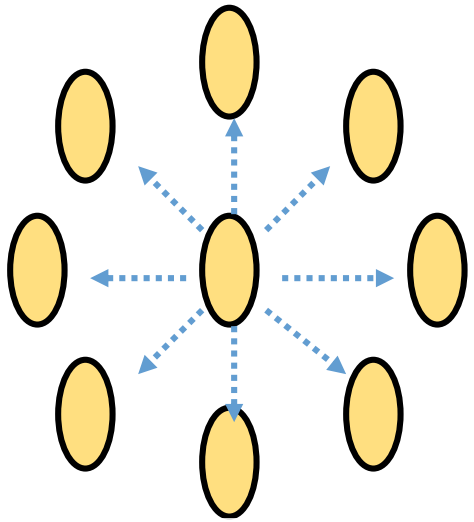
Diffusion Tensor Imaging

- Tensor is a mathematical model of directional anisotropy of diffusion

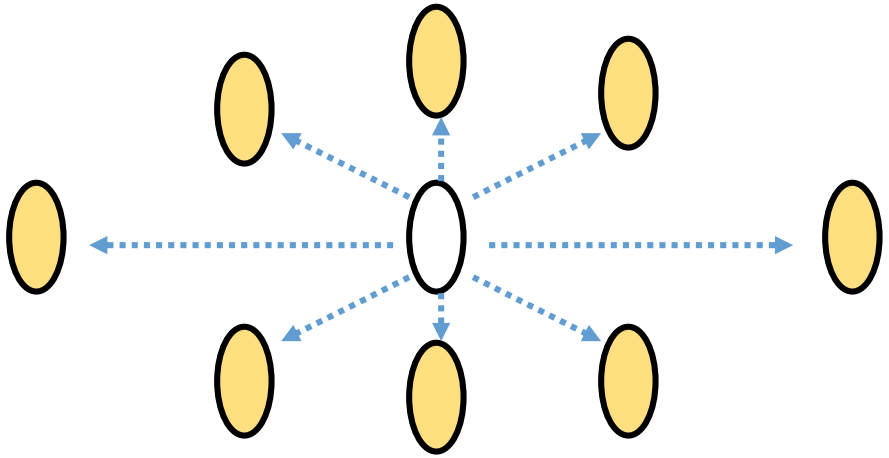
- From the tensor, we can calculate:
  - Direction of greatest diffusion
  - Degree of anisotropy
  - Diffusion constant in any direction



Diffusion Tensor Imaging



ISOTROPIC- Equal diffusion in all directions



ANISOTROPIC – Diffusion preferentially increased in some directions

Fractional Anisotropy - FA

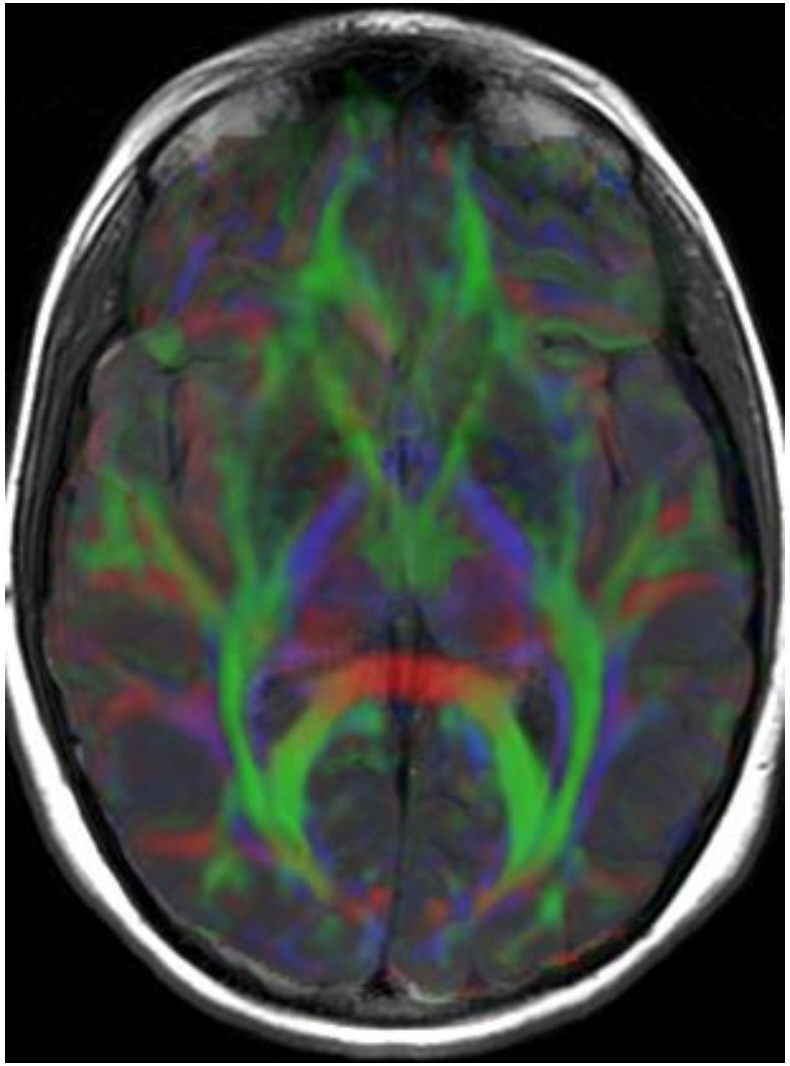
- Measures the degree of anisotropic (unequal) diffusion in a voxel
- Ranges from 0 to 1 (no units)
- 0 – isotropic (sphere-like)
- 1 – Purely anisotropic (straight line)
- Can characterize demyelinating lesions, e.g., breakdown of myelin and axonal loss can reduce FA and remyelination can increase FA
- FA value of CSF = 0

Fiber Tractography / DTI - Tractography

- Technique to assess direction of white matter tracts within the brain
- Directional information from neighboring voxels is combined to estimate 3D structure of major white-matter pathways
- Voxels are connected together taking into consideration both the direction of principle vector and FA value

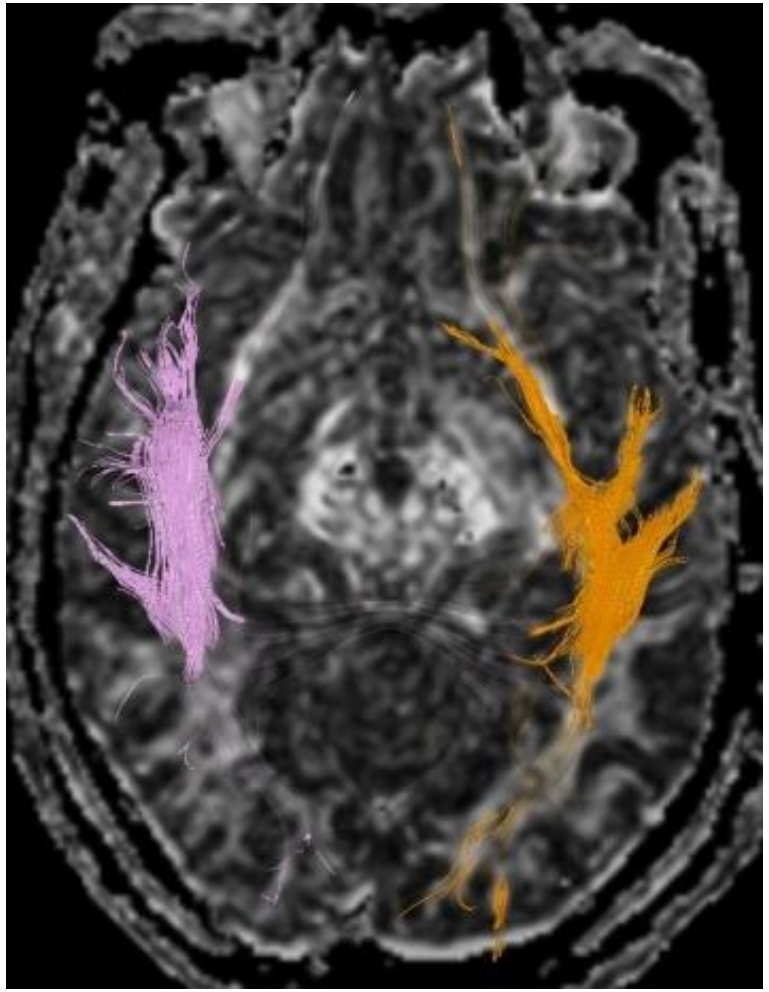
Clinical Applications – Normal Brain

- Fiber tracking provides critical information about white matter anatomy and connections
- Regions with similar tractographic features tend to be functionally co-activated - “neurons that fire together, wire together”
- IQ has been positively correlated with anisotropy in white matter association areas
- Reading ability has been correlated with anisotropy of left temporoparietal areas
- In the visual pathway, DTI has shown the retinotopic organization of fibers



Clinical Applications – Traumatic Brain Injury

- DTI is a useful technique to evaluate microstructural injury to the white matter fiber tracts in patients with TBI
- Decreased FA is seen in areas afflicted by TBI, that are occult on conventional MRI
- Studies suggest some correlation between findings on DTI with EEG and neuropsychological testing
- In the future, DTI may serve as a surrogate marker for closed head injury



Temporal White Matter

The Puzzle:

- Public Education – “Google”
- Physician Education
  - Recognition- evaluation
  - Good History
  - Neurologic exam
  - Neurocognitive screening
  - Visual-oculo-motor exam
  - Vestibular assessment (balance)
  - Neuropsychological testing
  - Quantitative EEG testing
  - SPECT scan
  - CT/MRI
  - Difussion Tensor Imaging – DTI scan
  - Access for HBOT

Real World Example

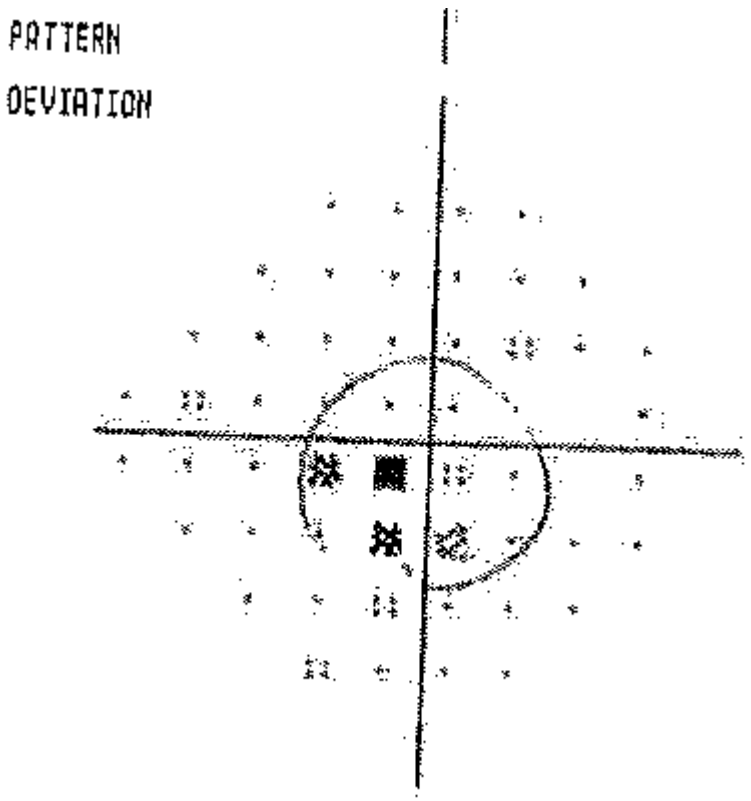
Hyperbaric Medical Solutions

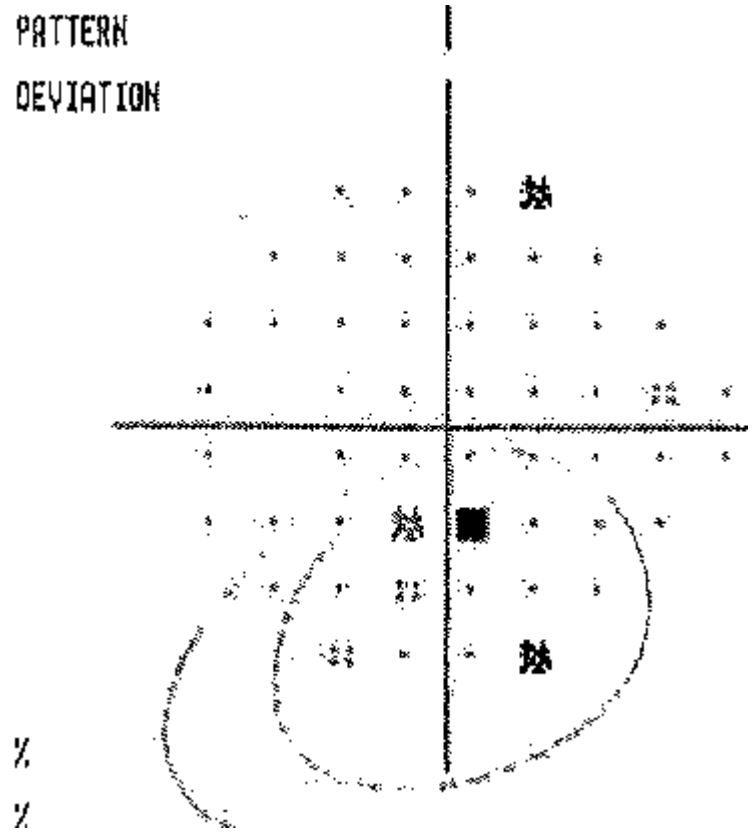
- Since 2016 we have completed 5160 dives on a total of 120 patients for mTBI (Avg 43/pt)
  - Additionally 30 Veteran patients with TBI / PTSD, totaling approximately 1,000 dives (Avg 33/pt)
- **59 percent were self referral / “Google”**

Our Experience

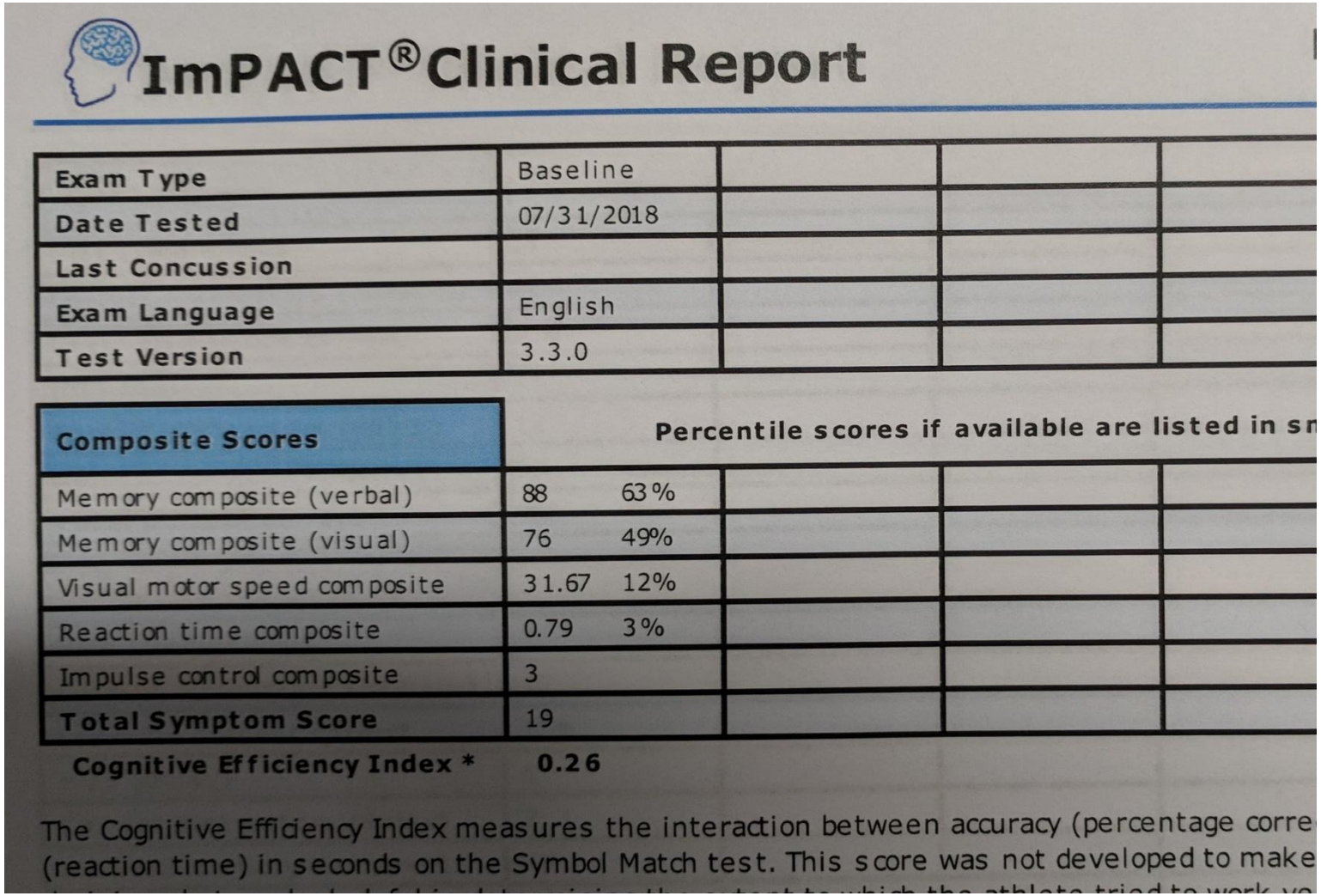
- Patient Example
  - M.G.
  - Male
  - 17 yo

M.G.





M.G.



ImPACT® Clinical Report

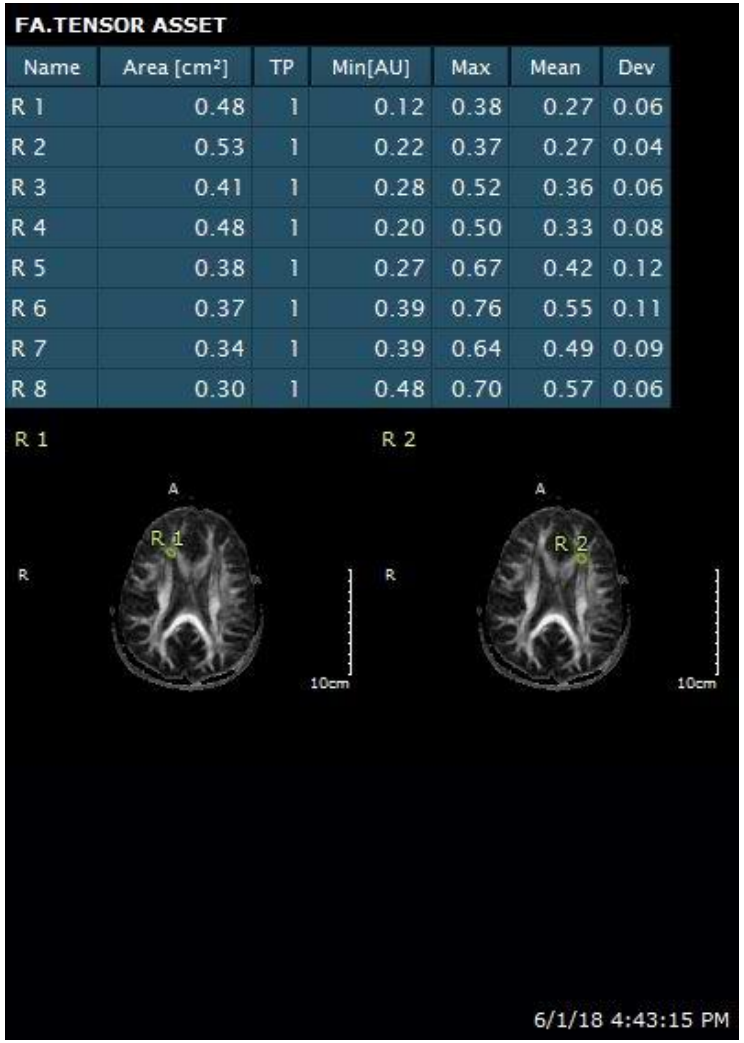
Exam Type	Baseline			
Date Tested	07/31/2018			
Last Concussion				
Exam Language	English			
Test Version	3.3.0			

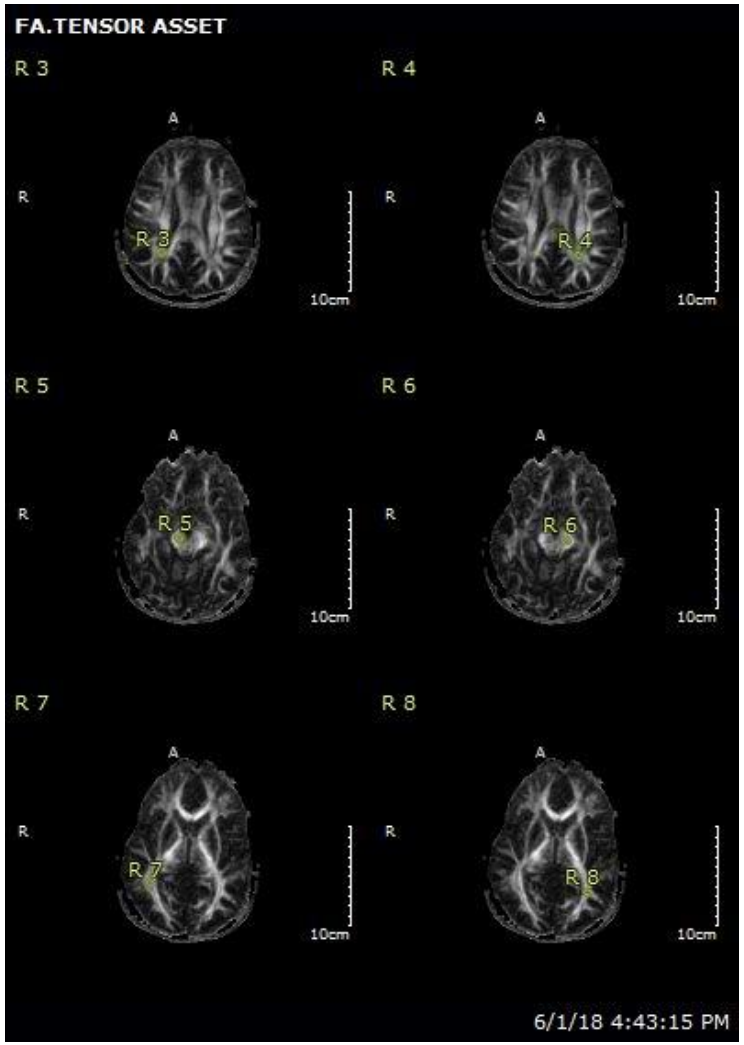
Composite Scores	**Percentile scores if available are listed in sm**			
Memory composite (verbal)	88 63%			
Memory composite (visual)	76 49%			
Visual motor speed composite	31.67 12%			
Reaction time composite	0.79 3%			
Impulse control composite	3			
Total Symptom Score	19			

Cognitive Efficiency Index * **0.26**

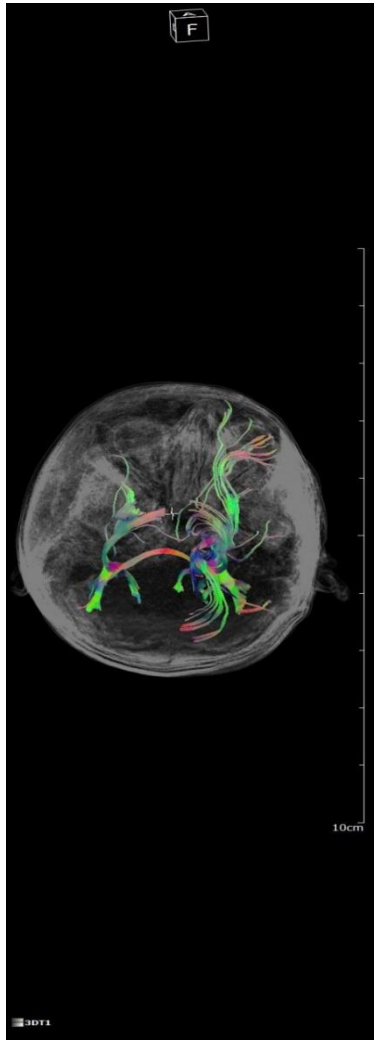
The Cognitive Efficiency Index measures the interaction between accuracy (percentage corre
(reaction time) in seconds on the Symbol Match test. This score was not developed to make

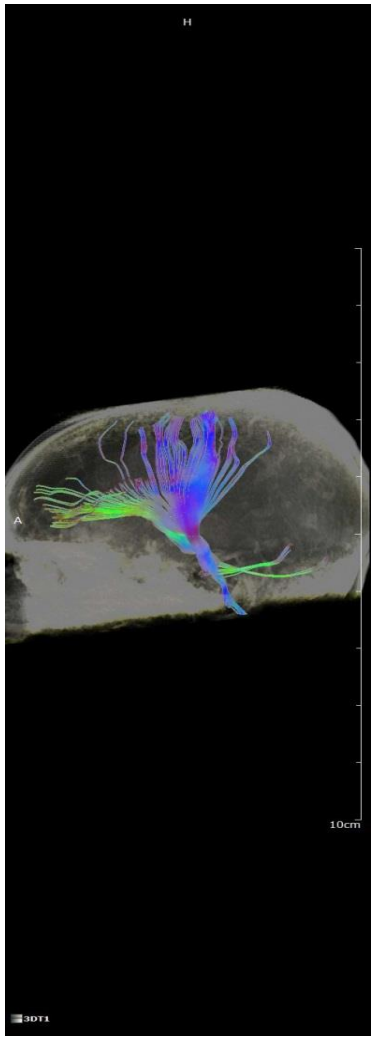
M.G.


FA.TENSOR ASSET
Name | Area [cm²] | TP | Min[AU] | Max | Mean | Dev
R 1 | 0.48 | 1 | 0.12 | 0.38 | 0.27 | 0.06
R 2 | 0.53 | 1 | 0.22 | 0.37 | 0.27 | 0.04
R 3 | 0.41 | 1 | 0.28 | 0.52 | 0.36 | 0.06
R 4 | 0.48 | 1 | 0.20 | 0.50 | 0.33 | 0.08
R 5 | 0.38 | 1 | 0.27 | 0.67 | 0.42 | 0.12
R 6 | 0.37 | 1 | 0.39 | 0.76 | 0.55 | 0.11
R 7 | 0.34 | 1 | 0.39 | 0.64 | 0.49 | 0.09
R 8 | 0.30 | 1 | 0.48 | 0.70 | 0.57 | 0.06
R 1
R 2
A
R
10cm
6/1/18 4:43:15 PM


FA.TENSOR ASSET
R 3
R 4
R 5
R 6
R 7
R 8
A
R
10cm
6/1/18 4:43:15 PM

M.G.





Another Case Study

Former high functioning executive, fell and hit her head on a door frame, knocking her unconscious

Could not walk after the event. Work up in the ER was "normal" and she was sent home.

Symptom subsequently worsened: difficulty with short term memory, concentration, attention, executive function and migraine headaches – getting lost

Impulsivity followed and secondary depression, which lead to 2 suicide attempts.

Inpatient psych hospital for 35 days

Case Study (cont.)

60 sessions of HBOT @1.5 ATA X 60 minutes

Significant subjective improvements in all these categories and marked objective improvement on both ImPACT testing and neuroimaging.

No further headaches

Does not get lost any more

Psychiatrist is amazed

Her life is back

Treatment with HBOT in my practice:

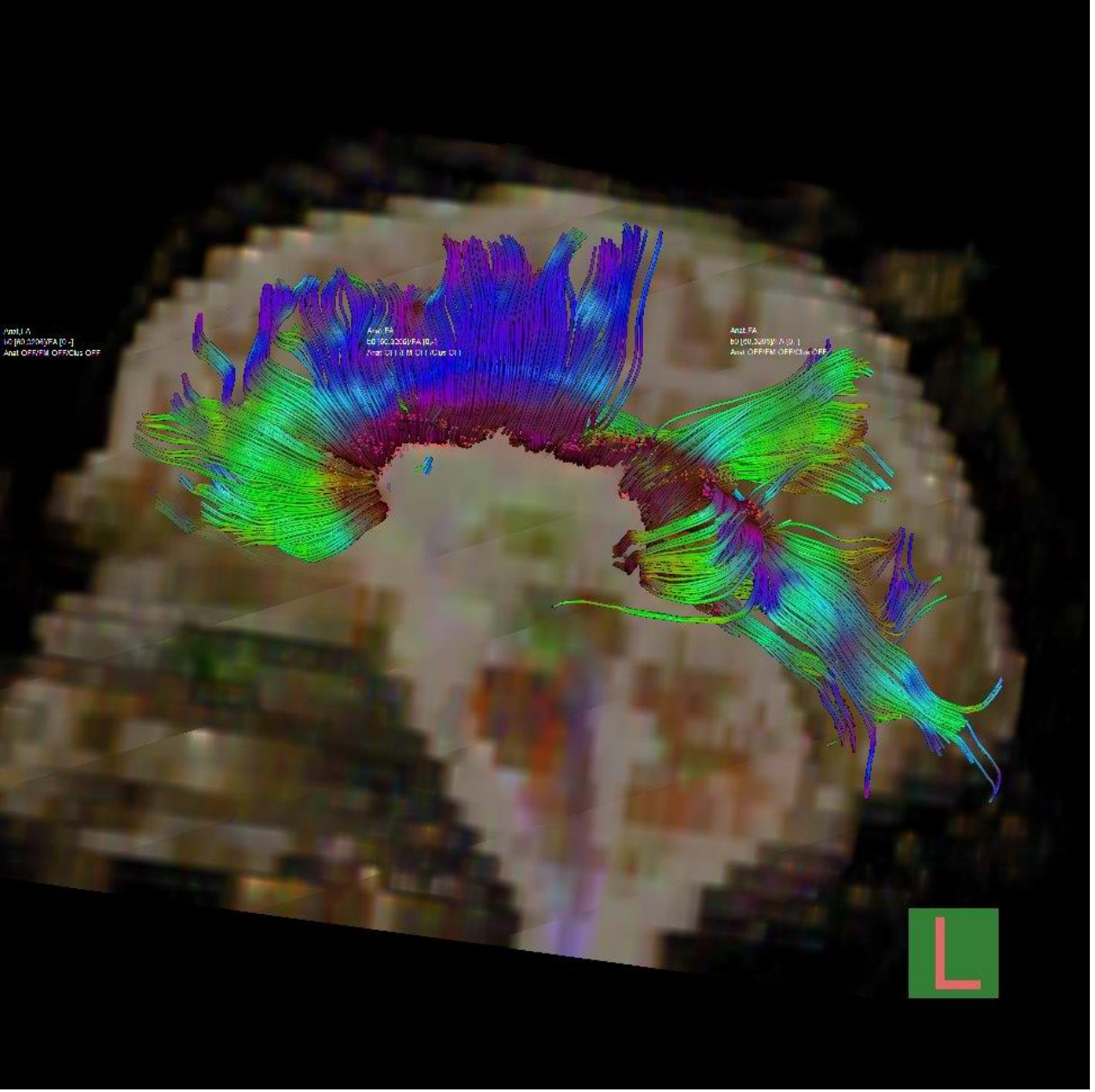
- Start ASAP using 1.5 ATA 100% O2 x 60
- Tx # 5-60

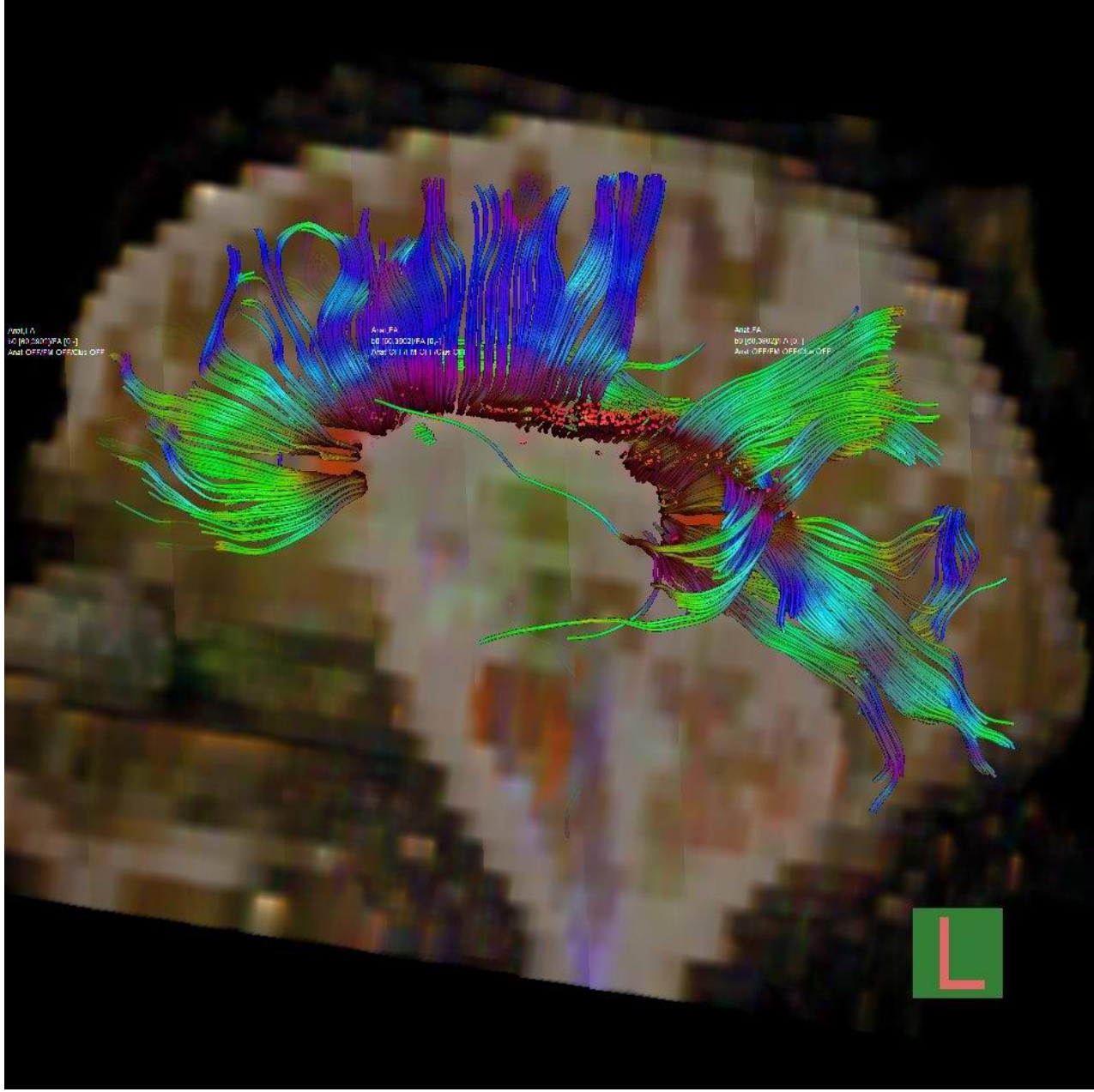
- Our Acute patients (<1 month) avg 5 sessions
  - Typically 1.5 ATA, however some 1.75 / 2.0 **Dr Denehem to review

- Our Sub Acute (1 month to 6 months) avg 30 sessions plus adjunctive care (Neurofeedback, visual training etc)

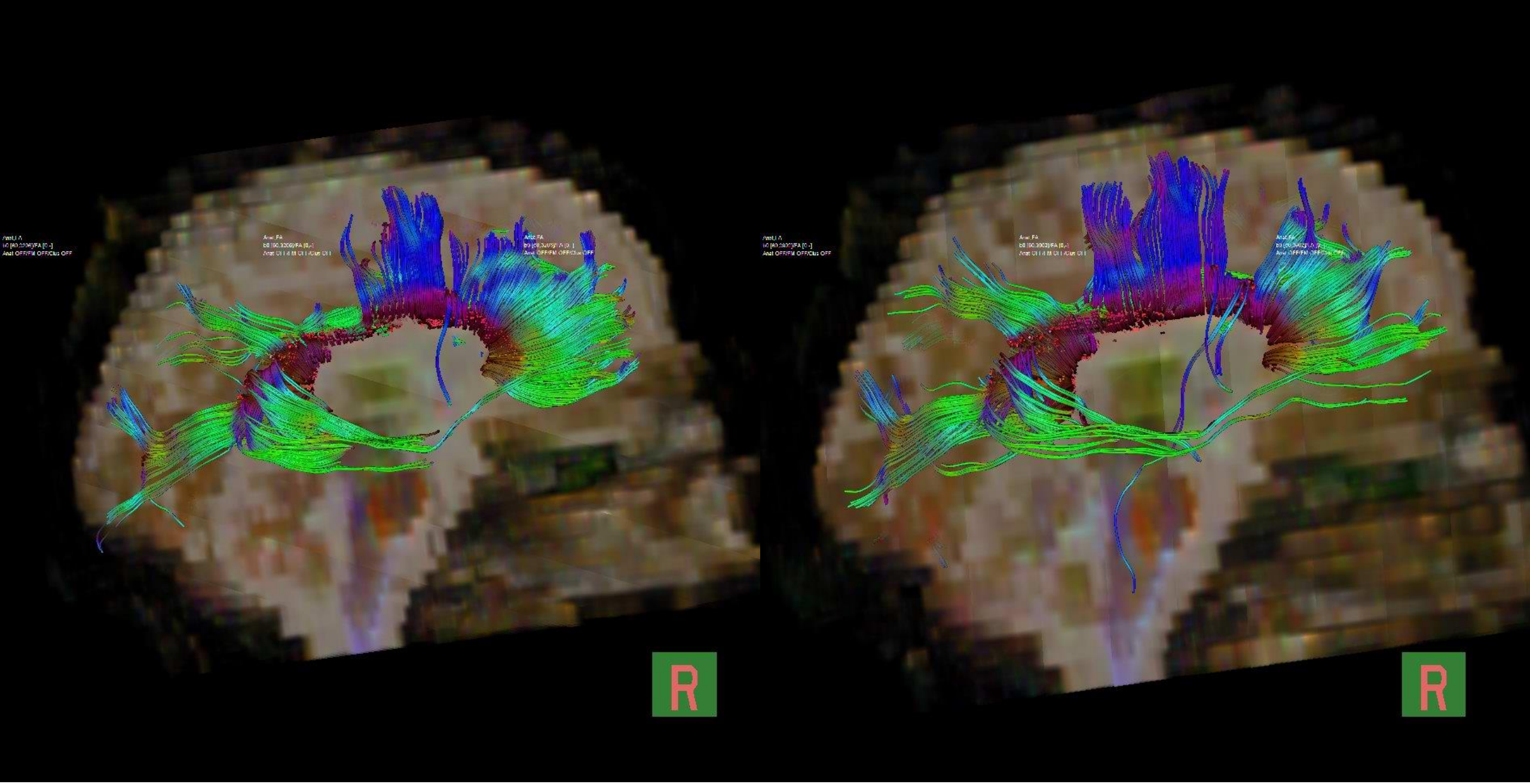
- Our Chronic (6 months to many years) avg 60 sessions plus adjunctive care

Evander Holyfield





S.B. 34 yo 4/24/18 vs 4/28/18 looses fight in 5th round – TKO – fights a Southpaw



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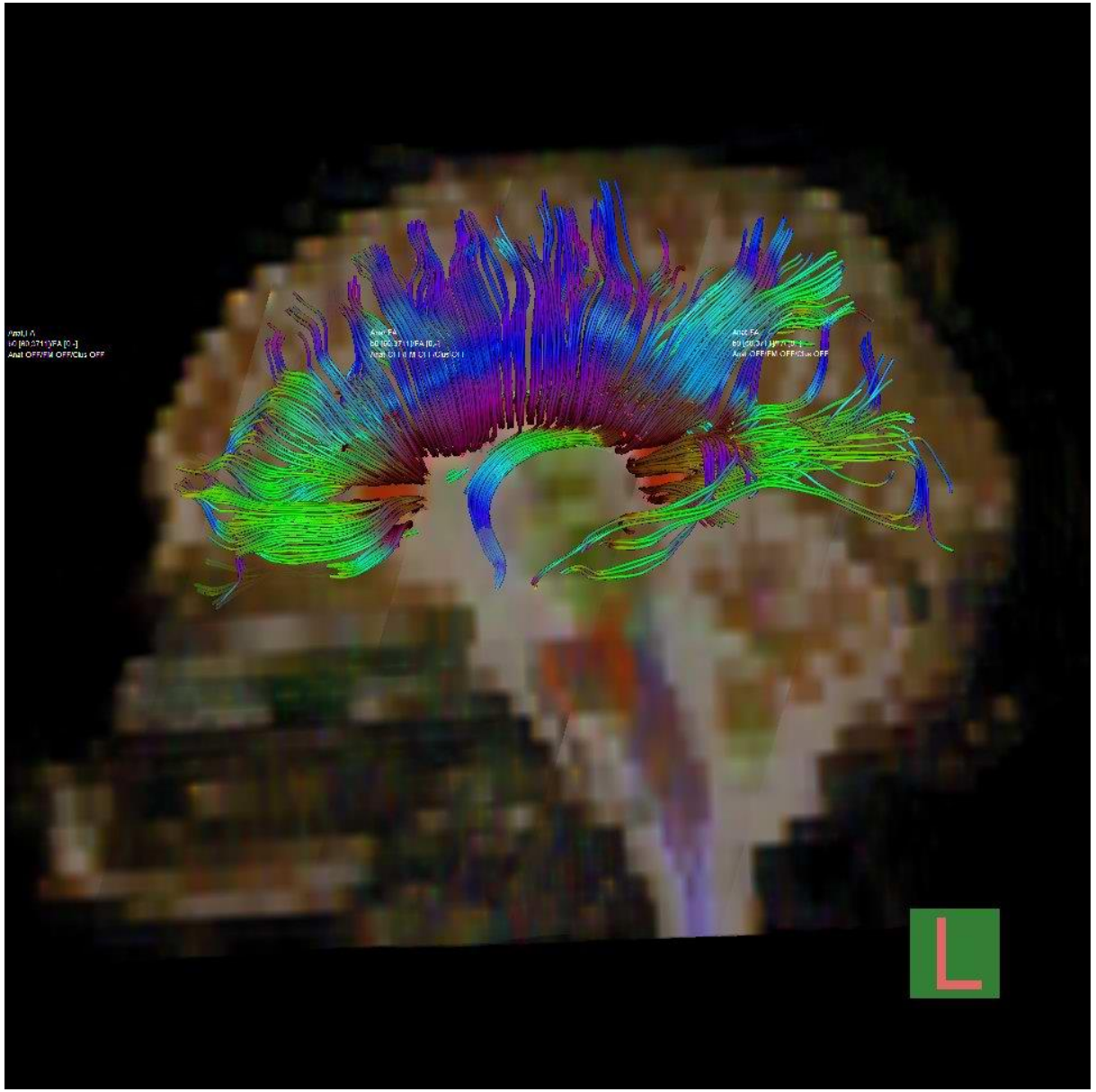
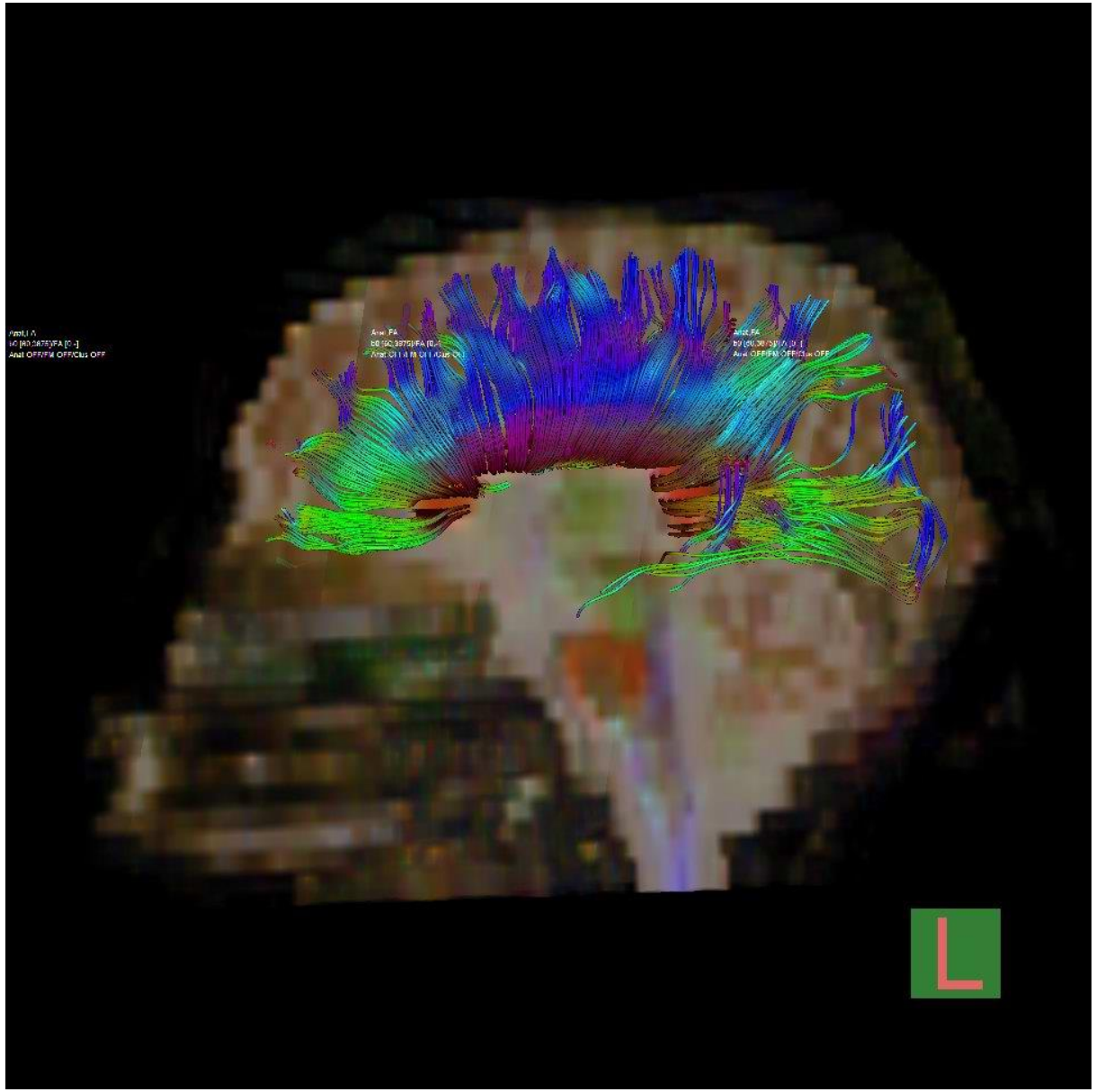
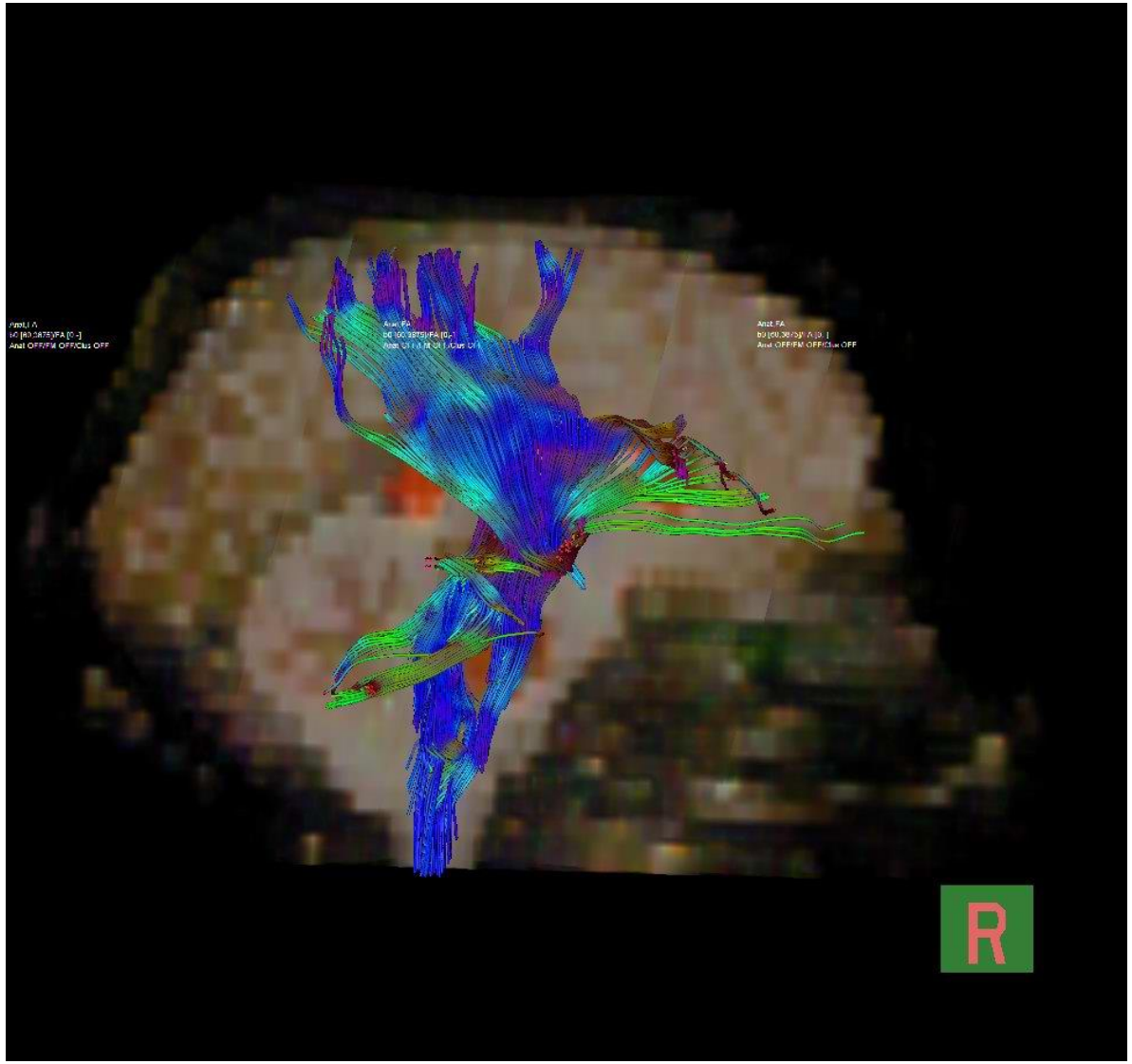
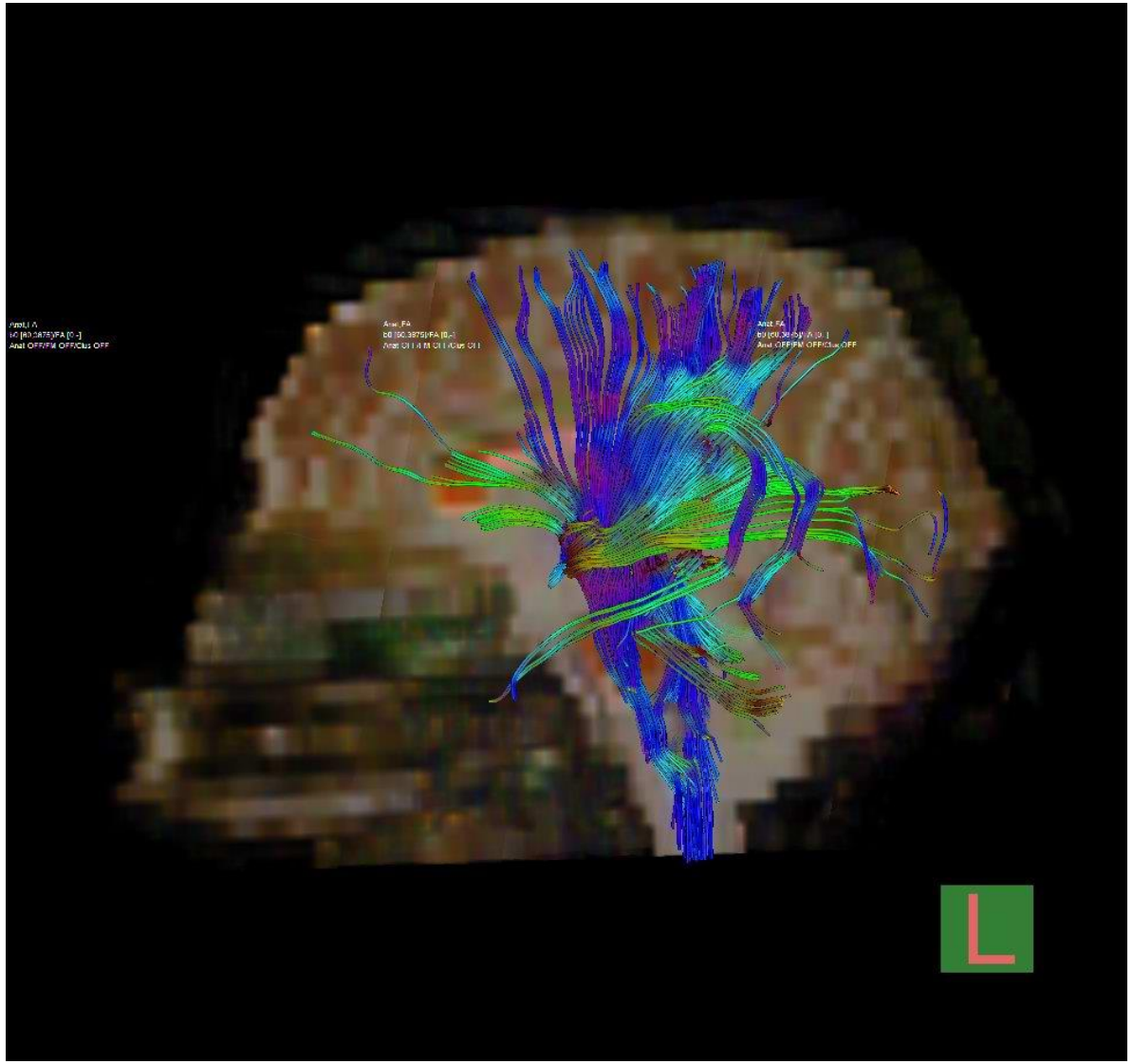




D.C. 4/12/18 vs 4/28/18 Easy fight - wins in 2nd round


F





Older (“less skilled “) fighter

Take Home:

1. A combined history, neurocognitive evaluation, and neurologic exam inclusive of vestibular and ocular evaluation in conjunction with advanced imaging and/ or electrophysiological methods are required in a modern brain injury detection and recovery program.

2. Hyperbaric oxygen serves as the hub treatment for brain repair

3. The best protocol for Hyperbaric Oxygen Therapy in brain repair has yet to be determined. What is clear is that hyperbaric oxygen therapy will serve a pivotal role.

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