INTELLECTUAL DISABILITY

- Poverty - Children in poor families may become intellectually disabled because of malnutrition, disease-producing conditions, inadequate medical care, and environmental health hazards. Also, children in disadvantaged areas may be deprived of many common cultural and day-to-day experiences provided to other young children. Research suggests that such under-stimulation can result in irreversible damage and can serve as a cause of intellectual disabilities.

HBOT in the U.S.

1. Air or Gas Embolism — Acute Wd
2. CO Poisoning/Smoke Inhalation — Acute Wd
3. Crush Injury, Compartment Syndrome, and other acute Traumatic Ischemias — Acute Wd
4. Decompression Sickness — Acute Wd
5. Selected Problem Wounds (diabetic, arterial insufficiency, venous stasis, etc.) — Chronic Wd
6. Exceptional Blood Loss (Anemia) — Acute Wd
7. Radiation Tissue Damage (Osteoradionecrosis and Soft Tissue) — Chronic Wd

UHMS HBOT Committee Report, 2014

HBOT in the U.S.

8. Skin Grafts and Flaps (Compromised)	Acute Wd
9. Thermal Burns	Acute Wd
10. Central Retinal Artery Occlusion	Acute Wd
11. ISSHL (Sudden hearing loss)	Acute/Subacute Wd
12. Clostridial Myonecrosis (gas gangrene)	Acute Wd
13. Necrotizing Soft Tissue Infections (Flesh-eating Bacteria)	Acute Wd
14. Osteomyelitis (Refractory) or (Acute) in Compromised Hosts (Bone Infections)	Chronic, Acute Wd
15. Intracranial Abscess (Brain)	Acute Wd

UHMS HBOT Committee Report, 20014

HBOT internationally

Russia: 70 diseases[1]

China: 49 diseases[1]

Japan: 33 diseases[1]

U.S: 15 diseases[1]

Gabb/Robin article: 132 diseases[2]

- Vast majority are wounding and inflammatory conditions
- My experience: 90-100 different conditions, 80% of which are neurological.

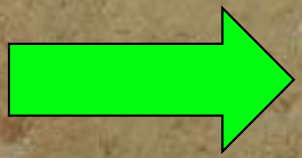
1. Textbook of Hyperbaric Medicine, 5th and 6th Editions, K.K. Jain, editor. Springer, Switzerland, 2009, 2017.
2. Gabb G. Chest. 1987;92(6):1074-82.

HOW DO WE HEAL ACUTE AND CHRONIC WOUNDS WITH HBOT?

WE REPAIR TISSUE & GROW NEW TISSUE

INPUT:

Daily intermittent exposure to hyper-oxia and increased pressure



OUTPUT:

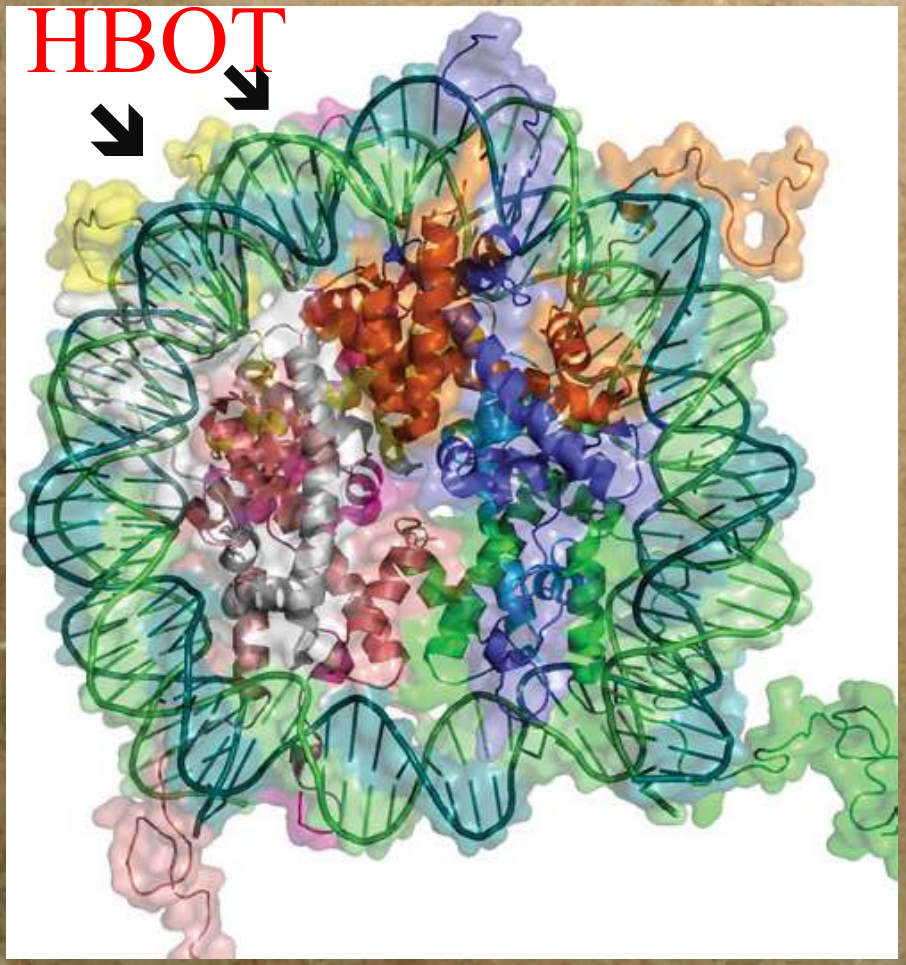
Reversal of pathophysiology and repair/growth of new tissue

What is the Mechanism of Action for growth of new tissue?

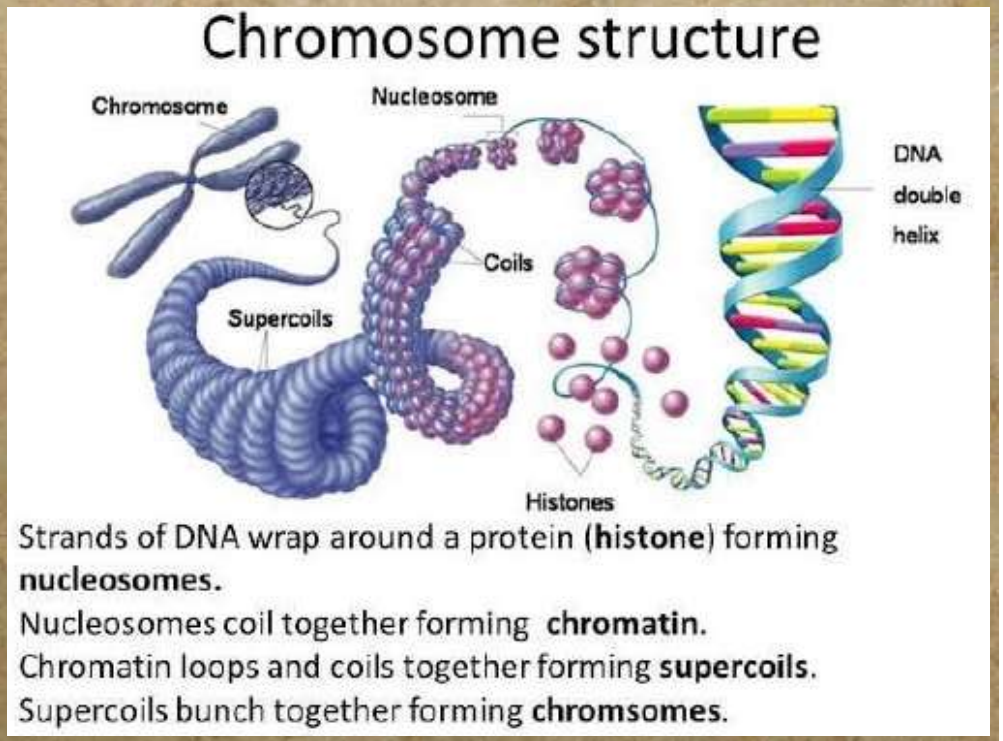
Gene Signaling

HBOT and Epigenetic Effects

DNA coil with histone protein interior.



http://www.genengnews.com/gen-articles/awaken-dormant-dna-epigenetically/5818



Strands of DNA wrap around a protein (**histone**) forming **nucleosomes.**
Nucleosomes coil together forming **chromatin.**
Chromatin loops and coils together forming **supercoils.**
Supercoils bunch together forming **chromsomes.**

https://www.facebook.com/108014754067239/photos/a.110468583821856/111089703759744/

HBOT and Gene Effects

Godman CA. Cell Stress and Chaperones, DOI 10.1007/s12192-009-0159-0 (Courtesy Dr. [illegible])

Human microvascular endothelial cells, in vitro

1st HBOT: 2.4 ATA/60 mins.

Continuous mass gene analysis for 48h

2nd HBOT at 24h

Results:

1. At 24h 8,101 of >19,000 protein-coding genes up or down regulated compared to control
2. Genes upregulated: anti-inflammatory and growth/repair hormones
3. Genes downregulated: pro-inflammatory and cell death.

At 48h:

1. Cells formed microtubules (blood vessels) in a petri dish

What else is HBOT Treating besides wounded tissue?

Wounded Cells: "Idling Neurons"

Neubauer, RA. The Lancet, 3/3/1990: 542

Concept of Idling Neurons
Other Examples

AWAKENINGS:

1. Donald Herbert, 43 y.o. fireman. 1995, anoxia→blind, mute, poor memory, nursing home. 4/2005-sudden intelligent conversation with wife and 4 sons over 14 hours. Family refuses to discuss followup.
2. Arkansas man, severe TBI, severe disability and "largely silent x 19 years," begins limited speech in 2003. Brain function remains limited.
3. Gary Dockery, police officer, GSW 1988→paralyzed, mute. 8 years later begins to speak and tell jokes x 18 hours. No further speech, but more alert.

Concept of Idling Neurons
Other Examples

AWAKENINGS:

What these cases imply is living, yet dormant, suppressed, or inhibited brain tissue that can exist in a non-functional state for years, only to be induced or allowed to spontaneously reactivate under the proper conditions.

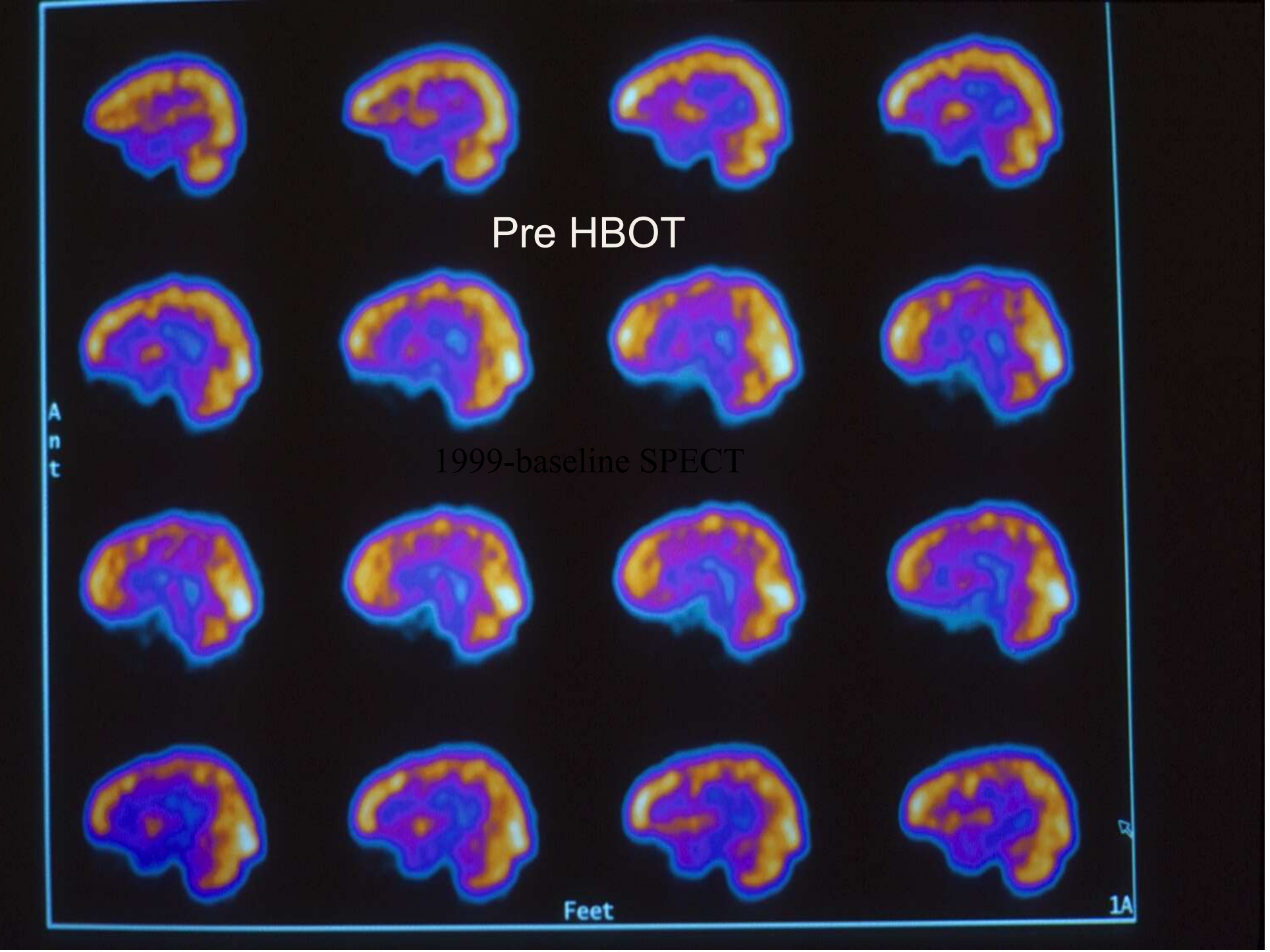
EVIDENCE for HBOT in chronic coma (17 patients):

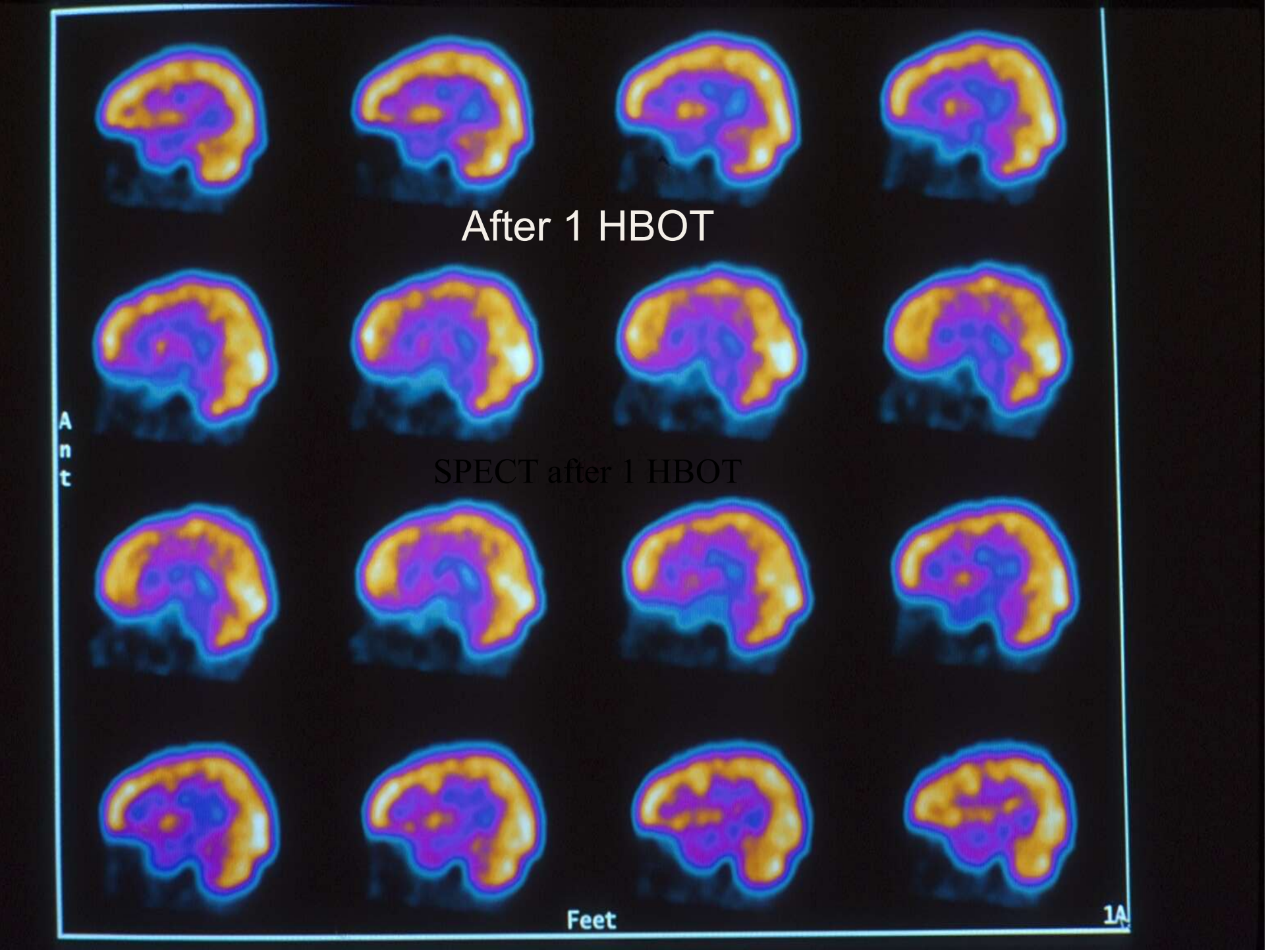
Neubauer RA. The effect of hyperbaric oxygen in prolonged coma. Possible identification of marginally functioning brain zones. Medicina Subacquea ed Iperbarica, 1985;5(3):75-79.

Concept of Idling Neurons

If Idling Neurons can exist in adults for 19 years why aren't they present in children?

They Are!


Pre HBOT
1999-baseline SPECT
Ant
Feet
1A


After 1 HBOT
Ant
SPECT after 1 HBOT
Feet
1A

HBOT in Pediatric Neurology-

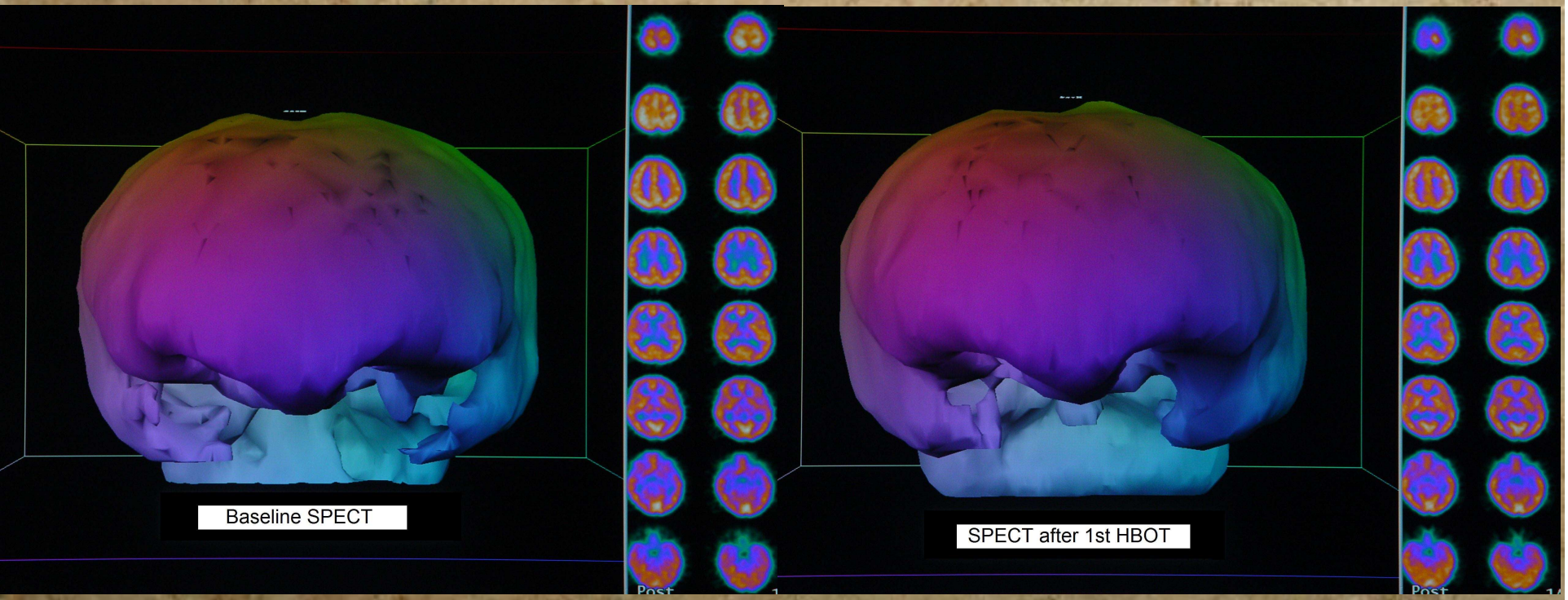
JoEllen Smith Medical Center Perfusion-Metabolism Encephalopathies Study, 1994-1999

Any chronic neurological condition, Neubauer test: SPECT, single HBOT, repeat SPECT.

40 HBOTs, repeat SPECT, 40 HBOTs, repeat SPECT, etc.

SPECT (1989-2002)

5 y.o. boy, CARS score 42-45,
Before and after 1 HBOT at 1.5 ATA



LSU New Orleans Chronic Pediatric Brain Injury Study

IRB Approved Protocol:

Exam, SPECT, 1 HBOT,
SPECT, 39 HBOT's,
Break, 40 HBOT's,
SPECT, Exam, SPM
Analysis SPECT.

Pixel p value cutoff < .01

Harch/Devous Analysis

First 18 patients, ages 1 - 15

15 patients:	1 - 5 y.o.
1 patient:	8 y.o.
1 patient:	12 y.o.
1 patient	15 y.o.

16 patients: CP

1 patient:	PDD/borderline autism/CP
1 patient	air embolism/stroke (14½ y.o. injury)

Harch, Devous, 2001

Harch/Devous Analysis
HBOT's/SPECT

Patient	1 vs. Base	80 vs base	80 vs 1
1	X	X	X
2	X	X	X
3	X		
4	X	X	X
5	X		
6	X		
7	X	X	X
8	X	X	X
9	X	X	X
10	X	X	X
11	X		
12	X	X	X
13	X	X	X
14	X		
15	X	X	
16	X	X	
17	X	X	
18	X	X	

Harch, Devous, 2001

LSU New Orleans Chronic Pediatric Brain Injury Study

Results:

Significant Similar Changes in Brain Blood Flow after 1 & 80 HBOT's with Concomitant Clinical Improvement

Areas Involved: thalamus, caudate, amygdala, medial temporal lobe, visual cortex

Harch, Devous, 2001

LSU New Orleans Chronic Pediatric Brain Injury Study

Pattern of SPECT Changes:

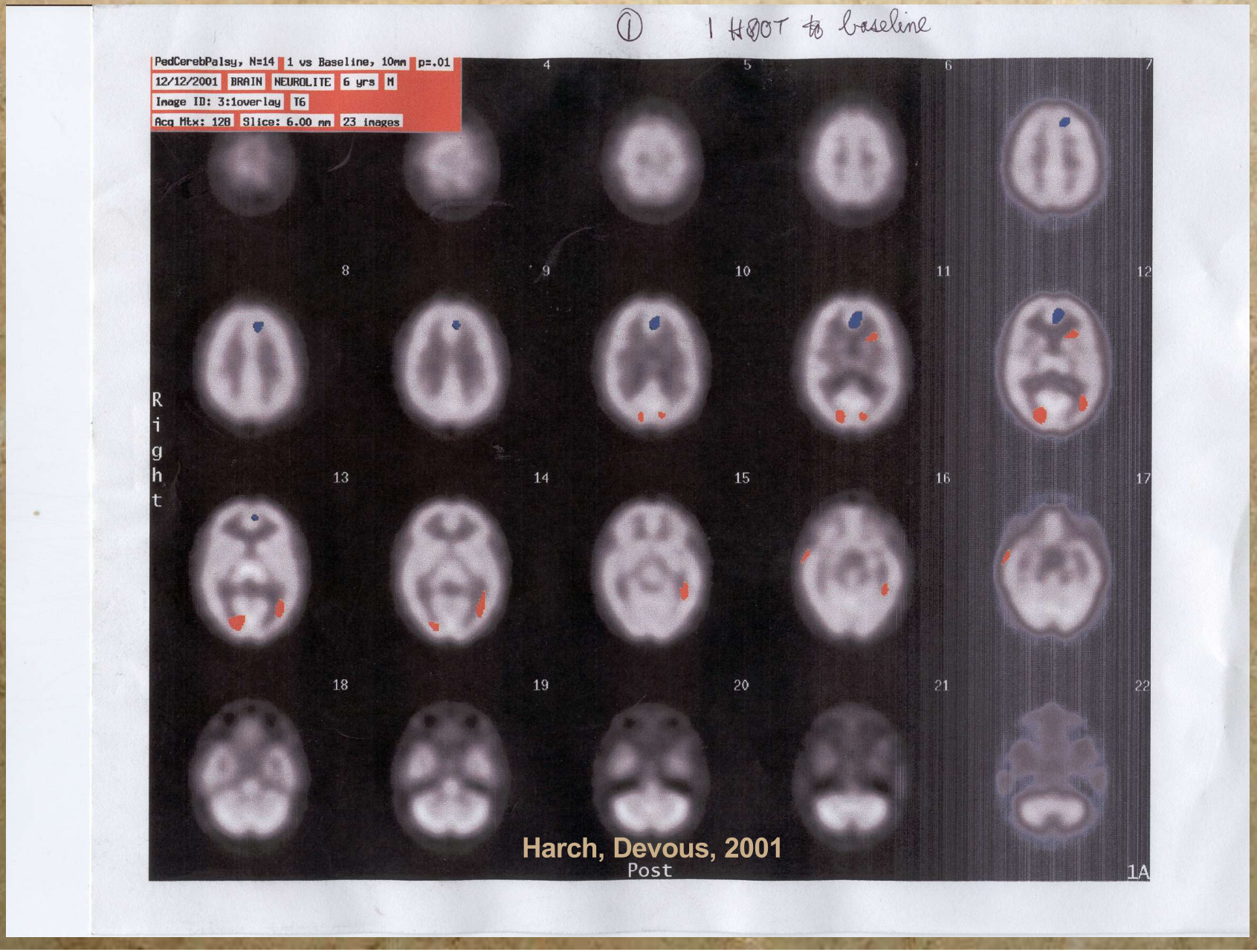
Some Areas Increase (low blood flow areas),

Some Areas Decrease (some areas of high blood flow).

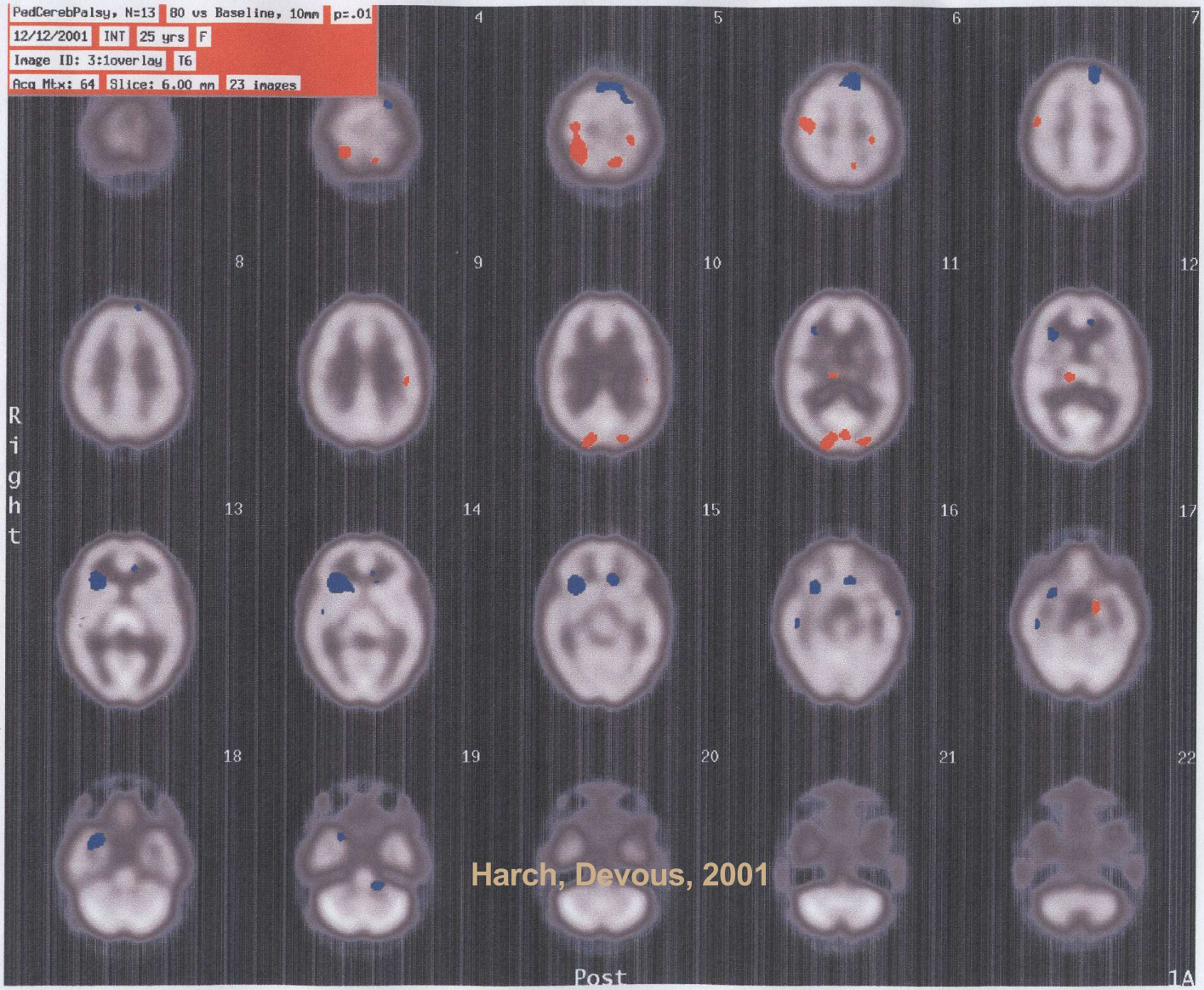
Similar to "Smoothing" seen qualitatively

Harch, Devous, 2001

One HBOT vs. Baseline



80 HBOTs vs. Baseline



80 HBOTs vs. 1 HBOT

Conclusions of the Analysis

The areas that improved after one HBOT were thesame as those after 80 HBOTs and thus could predict the areas that could improve with repetitive HBOT.

In other words, Neubauer's test for HBOT worked at least for CP

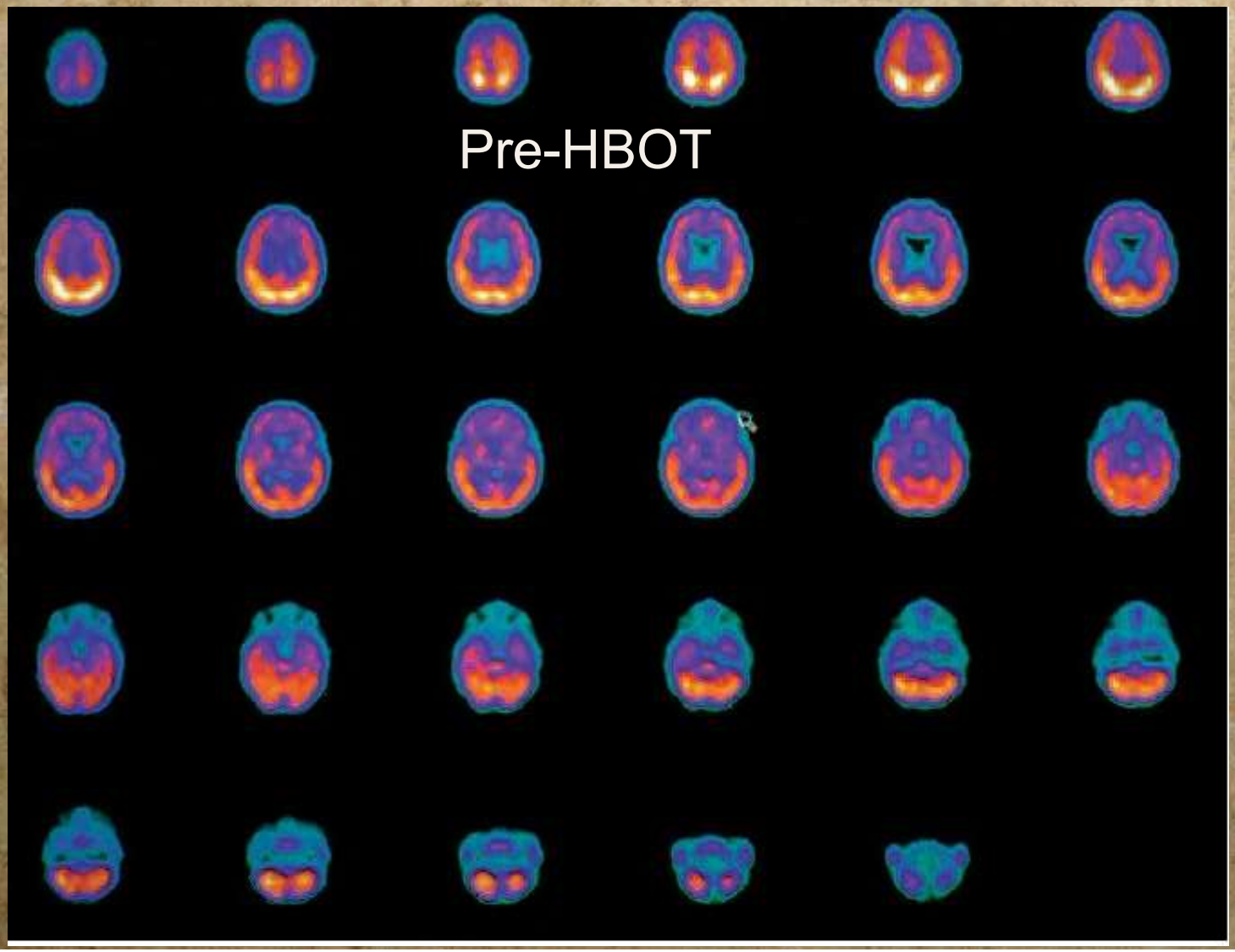
Harch, Devous, 2001

Examples of HBOT in Pediatric Neurology

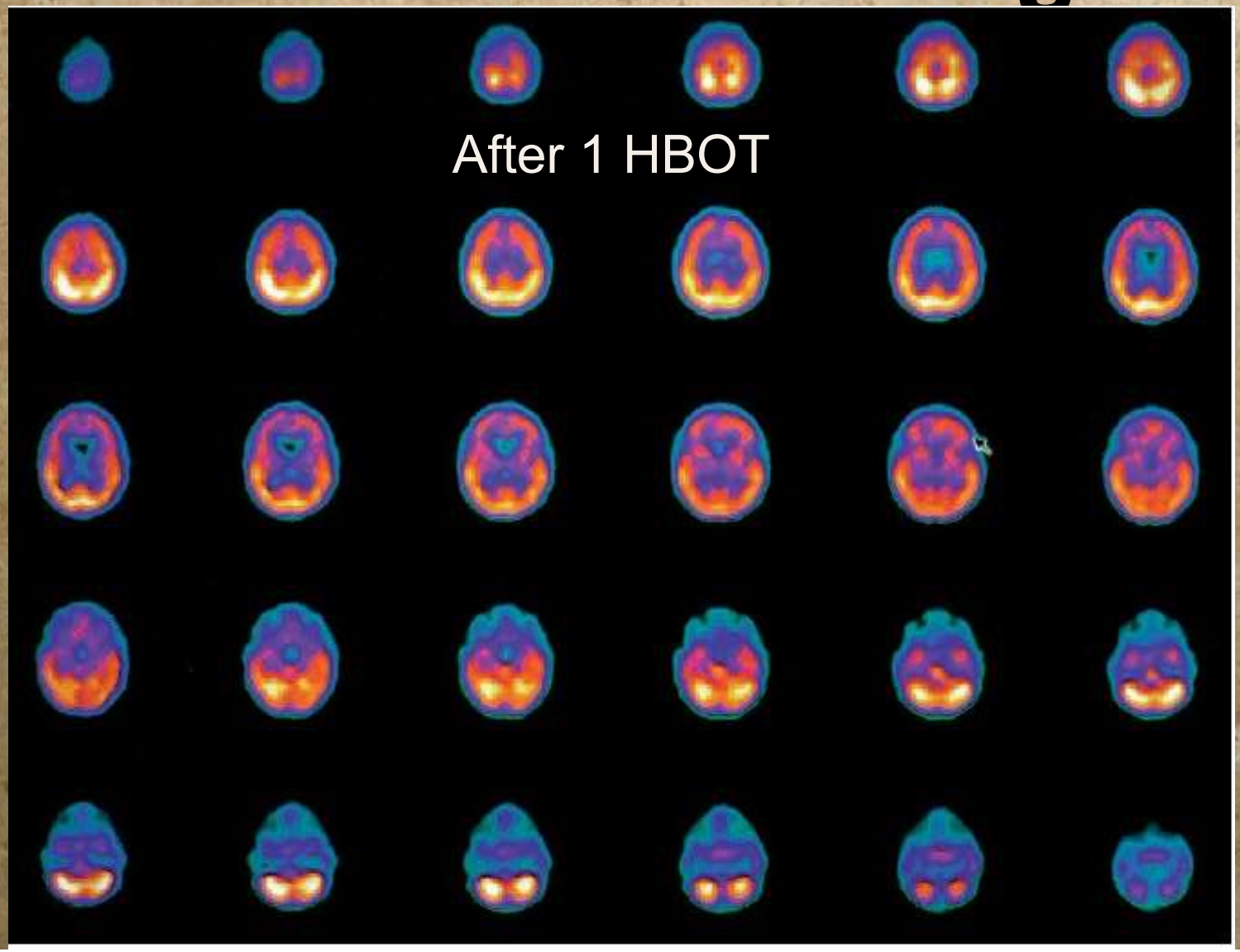
Chronic Drowning

- 4 y.o. boy, 5 min. submersion at bottom of pool
- ROSC: 45 minutes
- 2 years later: wheelchair, severe motor deficits, inability to speak/communicate, drooling, decreased attention, can't swallow.
- New Orleans: SPECT, single HBOT, SPECT.
- 40 HBOTs, 3 week break, 40 HBOTs.
- Generalized decrease spasticity, movement of all 4 extremities, increased head and trunk control, improved swallowing, awareness, non-verbal communication, attention span.
- Repeat SPECT

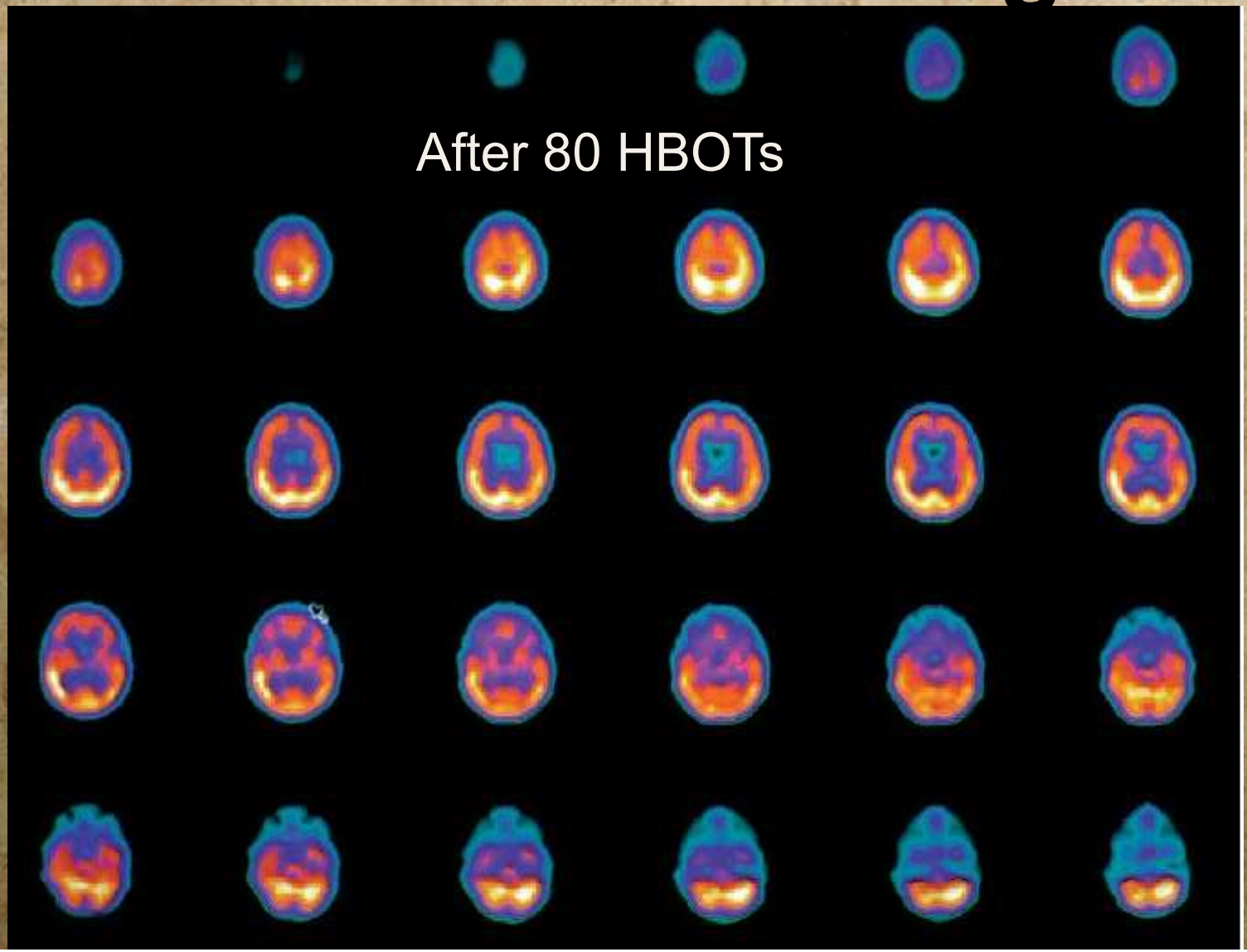
HBOT in Pediatric Neurology: Chronic Drowning



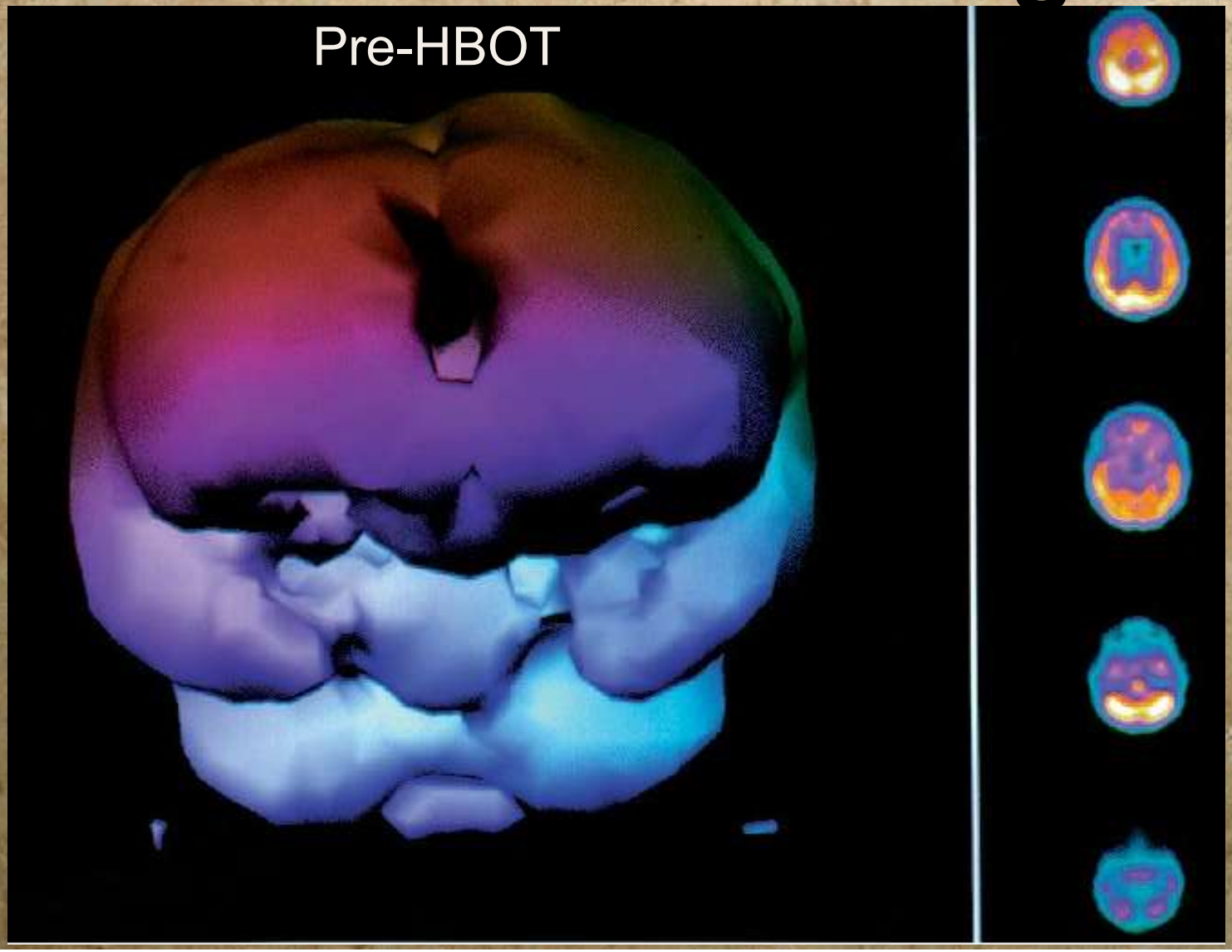
HBOT in Pediatric Neurology: Chronic Drowning



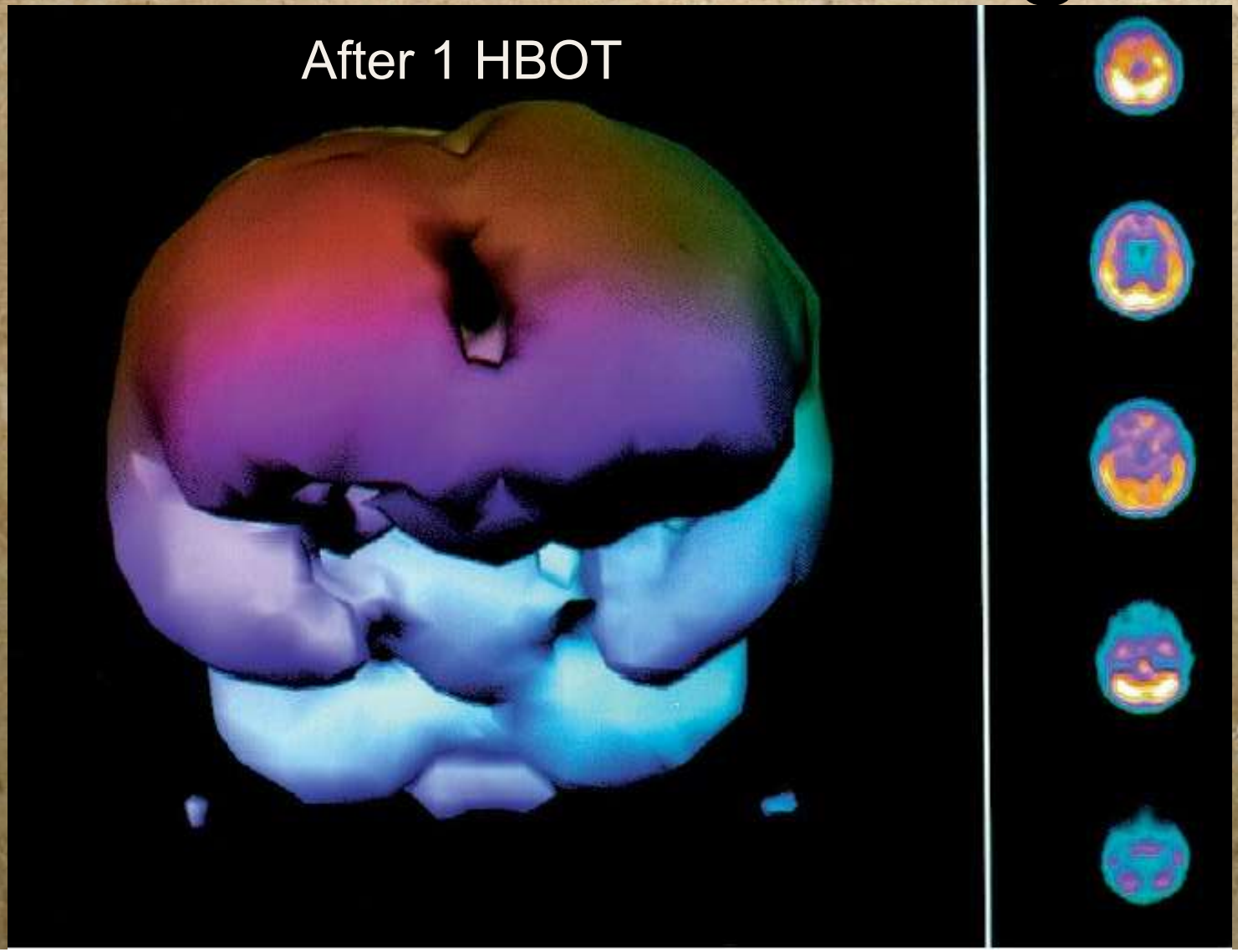
HBOT in Pediatric Neurology: Chronic Drowning



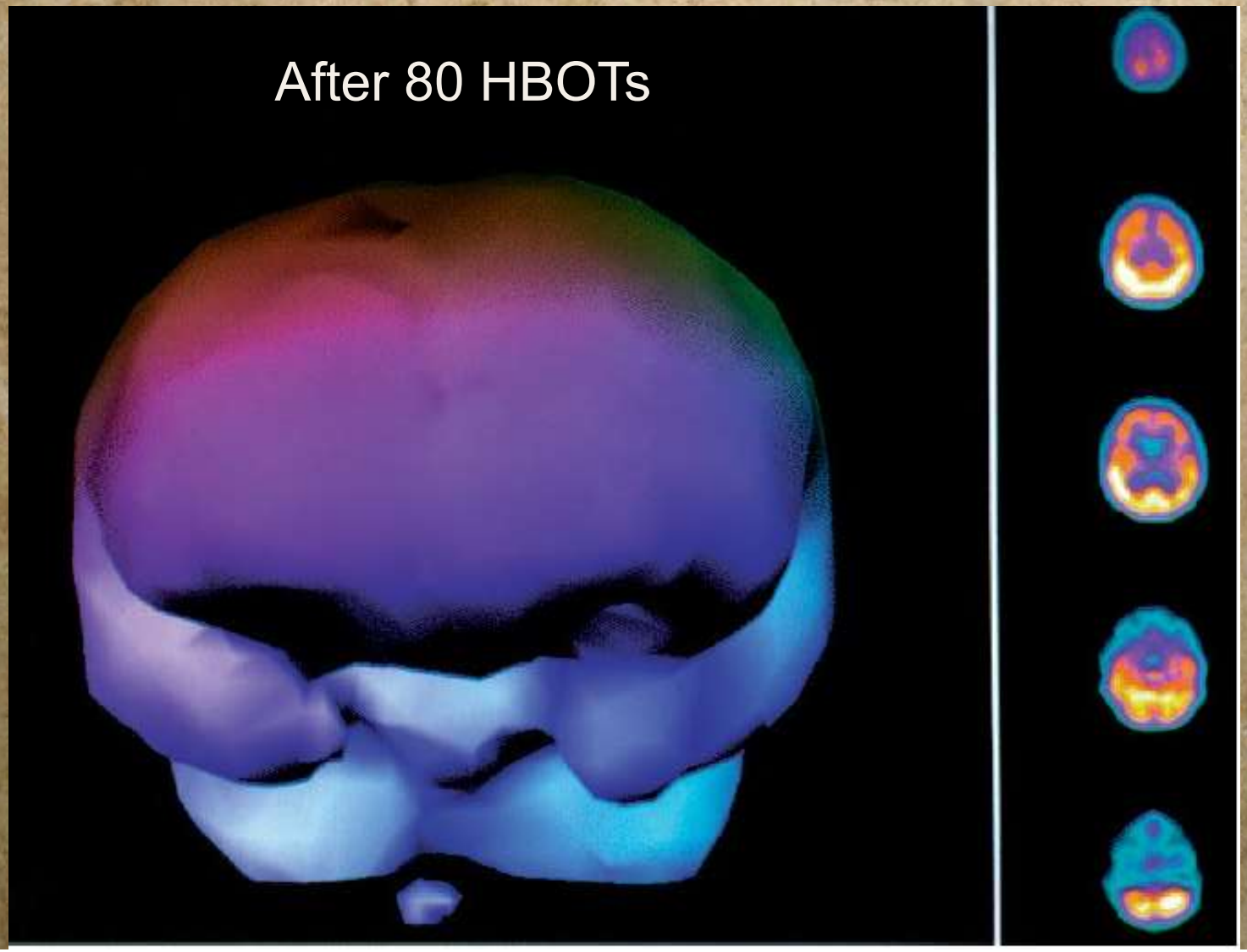
HBOT in Pediatric Neurology: Chronic Drowning



HBOT in Pediatric Neurology: Chronic Drowning



HBOT in Pediatric Neurology: Chronic Drowning



HBOT in Pediatric Neurology: PANS

- 18 y.o male c/o depression, 2/14/2012
  - High school, A's and B's, well-adjusted socially
  - 9/2009: swine flu syndrome.
  - One week later, fall, hit back of head during soccer, dizzy, kept playing-trouble, severe HA, abnormal eyes, couldn't drive home, gait disturbance, mild photoph.
    - ED eval with CT-negative. Dx: concussion.
    - Resolution of symptoms by next day.
    - Returns to soccer.
    - Within 2 weeks sore throat, fever, HA, URI, fatigue that persists for 3 months
    - Multiple courses of antibiotics; steroids, extensive W/U, ID evaluation, multiple serologies, Echovirus 9 positive. Cognitive difficulties, irritability.
    - 12/2009: Missed significant amount of school, depression. Anti-depressant. MRI brain-nl 1/2010.
    - Waxing/waning Sx, but persistent HA, fatigue.
    - 5/2010: Trip to New Orleans. Post return: change in behavior, agitation, short-tempered, anhedonia, suicidal ideation, sleep disruption.
    - 7/2010: Trip to New Orleans. HA, sound/light sensitivity. Tremors. Returns home.
    - 7-8/2010: Worsened behavior, withdrawal, paranoia of cameras, flat affect, no eye contact, on and off anti-depressant.

HBOT in Pediatric Neurology: PANS

- 18 y.o male c/o depression, 2/14/2012
  - 8/2010: further cognitive decline, anxiety, constant fight or flight agitation, auditory hallucinations.
  - Extensive neurological workup. Dx: Schizophrenia. Lamictal helps.
  - Sleep deprived EEG-"low level seizure activity."
  - Neurotransmitter evaluation: low NE, DA, serotonin, elevated taurine and GABA. Heavy metals negative. Mycotoxins (one positive urine test).
  - Multiple consultants, evaluations, medication changes, diagnoses (none fit: atypical psychiatric syndrome, unresponsive to meds).
  - By early 2011 slow improvement begins.
  - Still hears voices, paranoid, feels weird, out of place. Increased symptoms post flights.
  - PMH: transient depression 13 y.o,
  - Sx: depression, fatigue, lack of motivation/apathy, sleep disruption, mood swings, irritability, photophobia, agoraphobia, cognitive Sx (processing speed, attention, word finding, change in personality, thinking)

HBOT in Pediatric Neurology: PANS

- 18 y.o male c/o depression, 2/14/2012
  - Meds: levothyr., liothyronine (T3), Vibryd, lamictal, Latuda.
  - FH: depression on maternal side (2 individuals).
  - P& SH: no substance abuse.
  - PEx: "Spooked appearance", anxious
    - Saccadic eye movements, tremor, hyperreflexia lower extremities, 2 beats of clonus, Romberg (precession), positive elbow flexion-check left UE, incoordination RUE.
  - Lab: mild vitamin D deficiency and hypothyroidism, both treated.
  - LP: negative typical tests. Ray Biotech Lab, Georgia cytokine Ab tests: elevated inflammatory chemokines: IL-8, MCP-1, IP-10, GRO; growth factors: osteopontin, angiogenin.
  - SPECT brain: diffusely heterogeneous pattern of blood flow.
  - Repeat SPECT post 1 HBOT: dramatic improvement in brain blood flow. (HMPAO uptake high in astrocytes).

HBOT in Pediatric Neurology: PANS

- HBOT: 3/2012: 40 HBOTs, 1/2014: 32 HBOTs, Increased energy, alertness, stayed up later, felt good post 1st Rx.
  - Post 40 HBOTs: global improvement according to parents; patient (No change). PEx: Improved appearance, improvement in most exam abnormalities.
  - Progressive improvement over 21 months. Eye Therapy helpful.
  - Re-Exam 12/2013: passed GED, goes out in public, driving, global improvement. Markedly different person in appearance. Residual anxiety, sleep problems, and some paranoia.
  - Repeat SPECT: Improved.
  - Restart HBOT 1/2014: improved through 20 Rxs., vacation, URI, deterioration, steroids, sleep disruption, mixed results.

HBOT in Pediatric Neurology: PANS

- Stopped treatment due to some increase in anxiety , irritability, decreased energy (past-peak).
- 17 months later: continued improvement, part-time then full-time community college classes, doing well, exercise 3x/week.
- "The old T...is back".
- Documentary made of patient's case by LAMMICO.
- 12/2016: graduated from community college, all A's in computer science. Started sophomore year at 4-year university, some exacerbation of fatigue, social anxiety, depression. Mild OSA, using CPAP. Overall, doing well.
- 5/7/2021: Mom drops into FPC to give followup and express desire to promote HBOT: 5/2018: graduated from four year university with honors, in the top % of his class of computer science majors. Employed by Fortune 500 company doing IT work in a western state. Living independently.

HBOT in Pediatric Neurology: PANS