

TransLingual NeuroStimulation (TLNS) for mild and moderate TBI

Tactile Communication & Neuromodulation Laboratory (TCNL)
Kinesiology Department, School of Education
UW- Madison, Madison, WI, USA

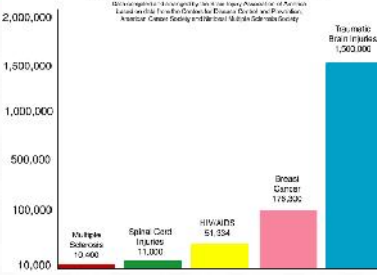
2017, September 14-15,
Annual BAAM Fall Conference



Traumatic brain injury

Comparison of Annual Incidence

Source: National Institute on Neurological Disorders and Stroke, National Institute of Mental Health, National Cancer Institute, National Institute on Deafness and Other Communication Disorders



Estimated annual costs from TBI's:
\$56.3 billion

Leading causes of TBI:


- Motor-vehicle crashes
- Falls & sporting accidents
- Violence & firearms
- Blasts #1 cause in military

Risk factors for TBI:

- Males 1.5-2 times as likely as females
- Ages 0-4 years, 15-19, & elderly (>75)
- African Americans have highest TBI death rate
- Military duties increase risk of TBI

From www.biausa.org

Army/Civilian Consequences of Concussive Traumatic Brain Injury (TBI)



Military

US: 2010-2012 - 100,000+ cases

28% of patients at Walter-Reed >\$100,000,000/year

Explosive blast injury, Overpressure, Penetrating injury, Diffuse axonal injury

Civilian

Blunt trauma
MVAs, sports, assaults

76.5B Cost to US

30% Chronic Disability

60,000 Active Duty Soldiers TBI

600,000 Retired Soldiers TBI

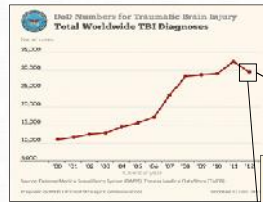
5.3M living with TBI related disability

1.750 TBI/year 30% chronic disability

Number of cases


DoD Numbers for Traumatic Brain Injury

Total Worldwide TBI Diagnoses

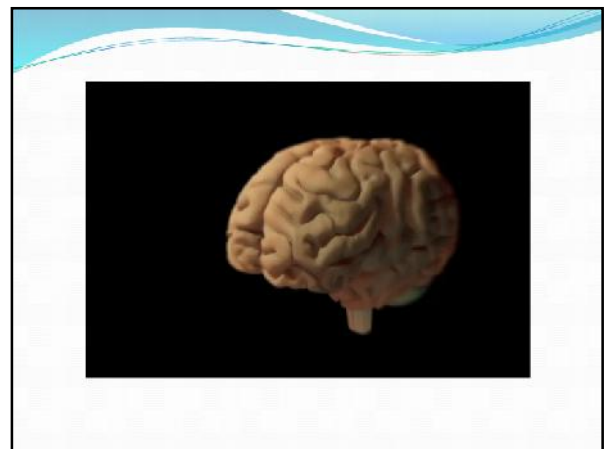


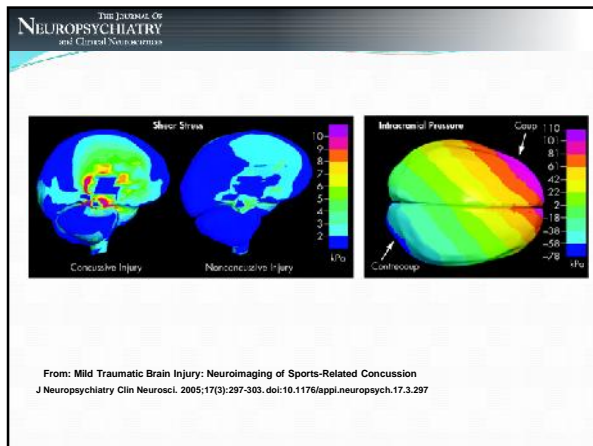
DoD Numbers for Traumatic Brain Injury Worldwide - Totals

Severity	Count
Severe	107
Moderate	208
Mild	1,028
Not Classifiable	2,601
Total - All Severities	30,406



*Worldwide Military TBI Incidence





Neuron: Cytoskeleton

The "bones" of cytoskeleton, form cellular shape
 "Neurofibrills" - within cytoplasm; responsible for **axoplasmic flow** (slow),
axonal transport (faster).

- **Microtubules** = straight hollow pipe, unbranched, ~25 nm diameter
 - associated protein – **tubulin** (tau);
 - **Microtubule-associated protein, or MAP**
- **Neurofilaments** =
 - ~ 10 nm
 - have fine branches
 - /like keratin, hair/
- **Mikrofilaments** = associated protein **actin**
 - ~ 5 nm
 - / like muscle contraction/

8/24/2017 Biological Processes of Aging, Anatomy 144-575 8

Neuron: axonal transport rate

No ribosomes in axon !!!

- **Slow axoplasmic transport rate**
 - 0.2-0.4 mm/day (actin, tubulin)
- **Intermediate**
 - 15-50 mm/day (mitochondrial protein)
- **Fast**
 - 200 - 1000 mm/day (peptides, glycolipids)

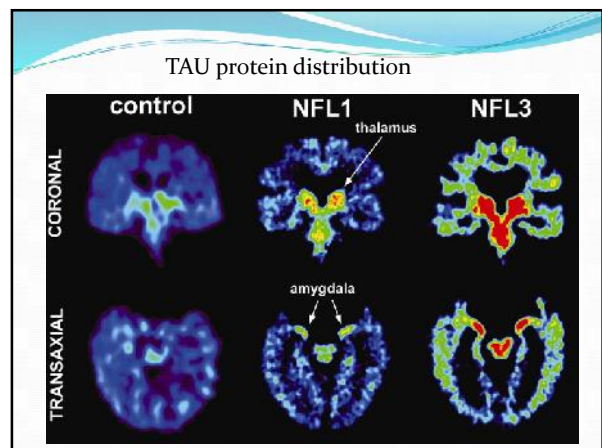
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Neuritic plaques

Neurofilament tangles


Stages of neurodegeneration

The dark globular mass in this stained image is a plaque of beta-amyloid protein in the brain of an Alzheimer patient. The plaque is surrounded by a halo of abnormal axons and dendrites and degenerating nerve cell bodies that appear darker than normal neurons.



STAGE 1
NO SYMPTOMS


In stage 1, isolated spots of tau build up, mostly around the frontal lobe, or the crown of the head.



CTE causes a protein known as tau (the brown spots in this stained slice above) to form around the brain's blood vessels, interrupting normal functioning and eventually killing nerve cells.

RAGE, IMPULSIVITY, DEPRESSION


In stage 2, symptoms begin to appear as defective tau protein affects more nerve cells in the brain's frontal (top) lobes.



(Source: Boston University Center for the Study of Traumatic Encephalopathy)

STAGE 3
CONFUSION, MEMORY LOSS

Tau deposits expand from the frontal (top) section to the temporal (side) section of the brain.

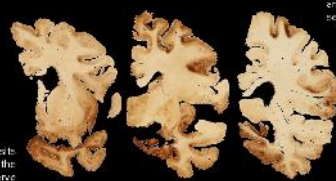
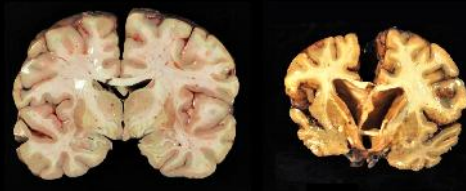


Condition begins to affect the amygdala and the hippocampus, which impairs emotion and memory.

STAGE 4
ADVANCED DEMENTIA

The brain becomes deformed and brittle, and cognitive function is severely limited.

By Stage 4, tau deposits have overwhelmed the brain, killing many nerve cells and shrinking it by roughly half its size.


Normal Brain Advanced CTE

(Source: Boston University Center for the Study of Traumatic Encephalopathy)

POST-TBI SYMPTOMS

Physical Symptoms

- Headache
- Neck pain
- Nausea
- Lack of energy
- Dizziness
- Sensitivity to light and sound
- Ringing in the ears
- Loss of taste and smell
- Change in sleep pattern



POST TBI SYMPTOMS

Cognitive Symptoms

- Feeling “dazed” or “foggy”
- Difficulty concentrating and paying attention
- Problems with word-finding and putting thoughts into words
- Easily confused and loses track of things
- Slower in thinking, acting, reading, and speaking
- Easily distracted
- Trouble doing more than one thing at a time
- Lack of organization in everyday tasks

POST TBI SYMPTOMS

Social and Emotional Symptoms

- Mood changes including irritability, anxiousness, and tearfulness
- Decrease motivation
- Easily overwhelmed
- More impulsive
- Withdrawn and wanting to avoid social situations

- Personality Changes
- Social behavior and Communication skills

mTBI

Functional System Approach

Simple

Complex

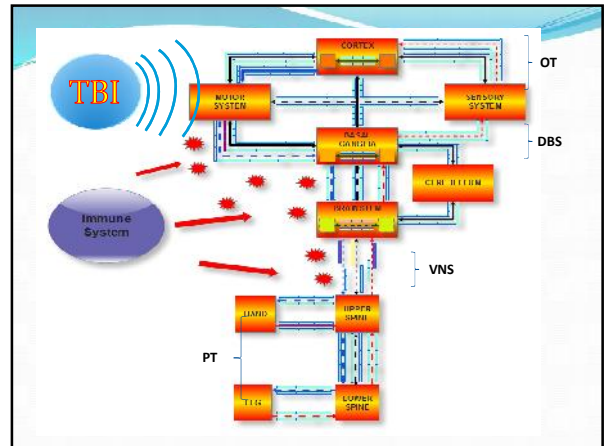
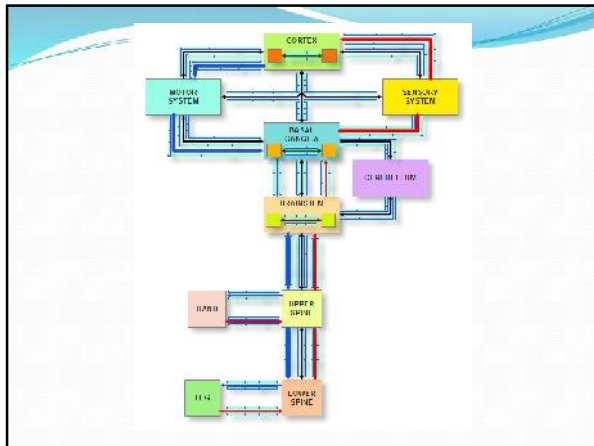
Parkinson's Disease

Super complex

...
Speech
Cognitive disorders
Memory loss
Attention deficit
Movement disorders
Balance
Posture
Gait
PTSD
Depression
...

TBI, Stroke, MS, Alzheimer Disease, Concussions, Brain tumor, ...

Solution 1?
Solution 2?
Solution 3 – Rehabilitation?



Tactile Communication and Neuromodulation Laboratory (TCNL)



Paul Bach-y-Rita
1934 - 2006

- Sensory Substitution
- Non Synaptic Transmission
- Late Brain Plasticity

Sensory Substitution

Tongue Display Unit (TDU)
Electrotactile Brain-Machine Interface


BrainPort

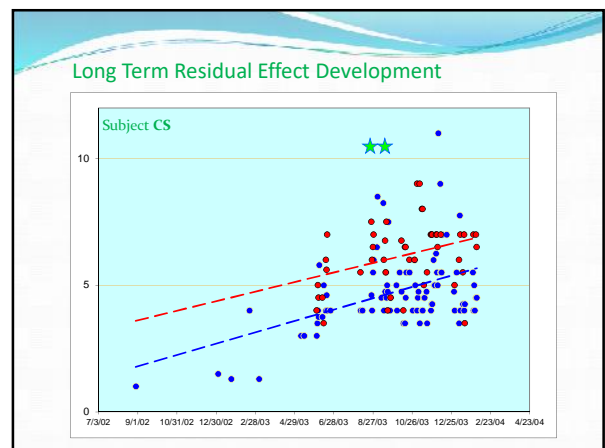
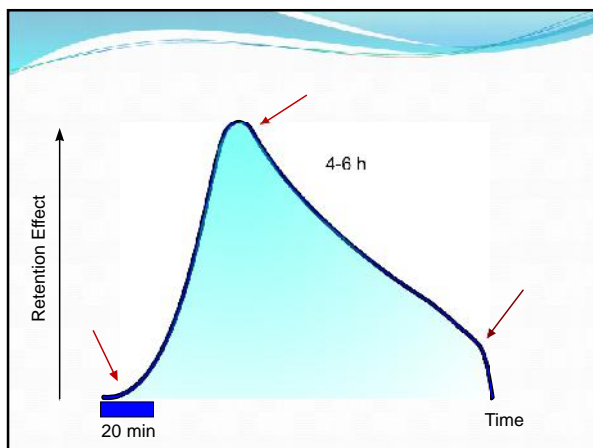
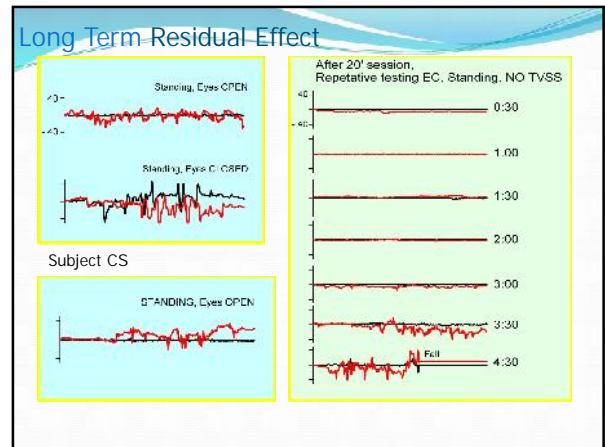
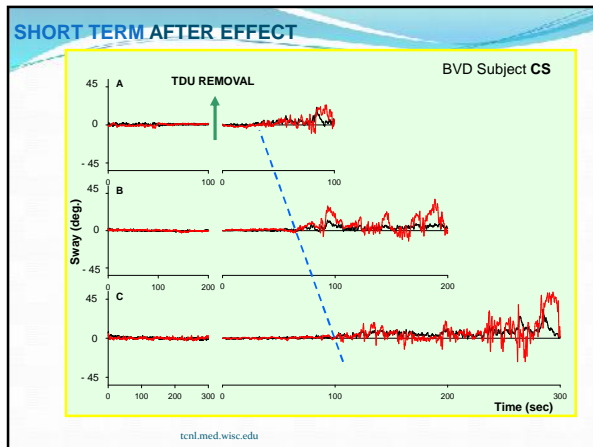
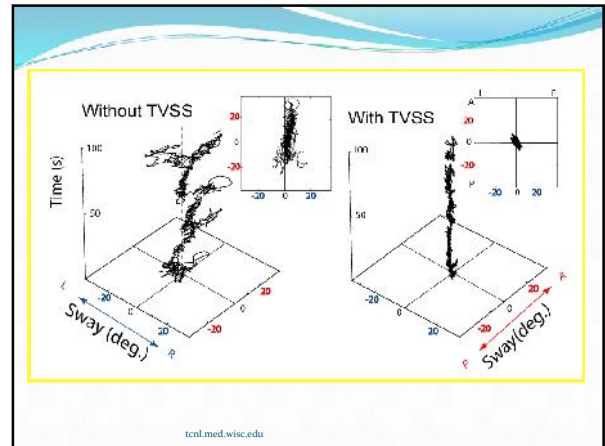
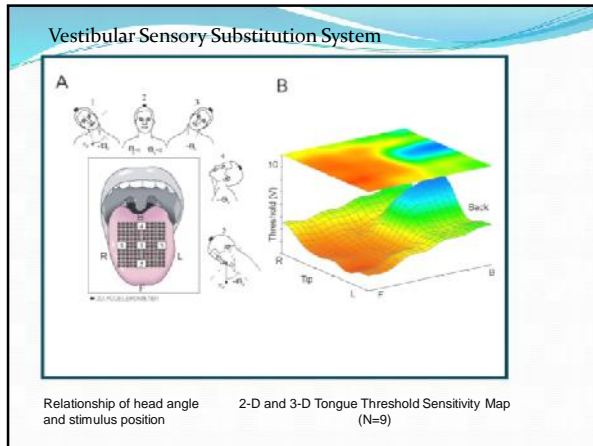
- BrainPort Vision Device
- BrainPort Underwater Navigation Device
- BrainPort Balance Device

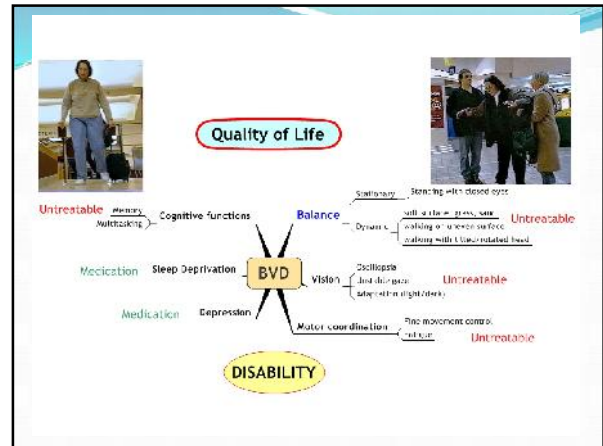
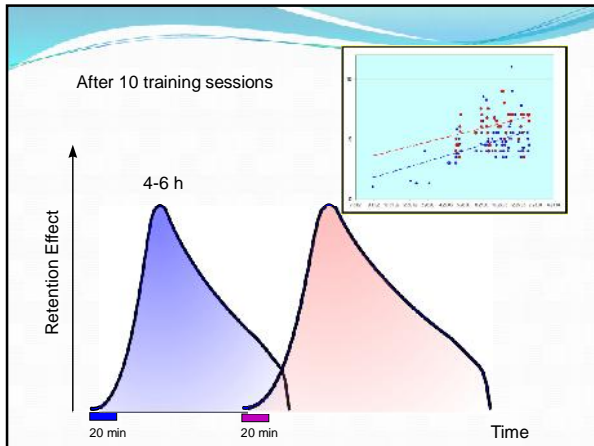
1998

Why the tongue?

- High density of sensory nerve endings
- Saliva is excellent electrolyte
- Tongue stays at constant **pH+ and t°**
- Electrode array is **non-invasive**
- Discreet **protected** environment
- Comfortable sensation



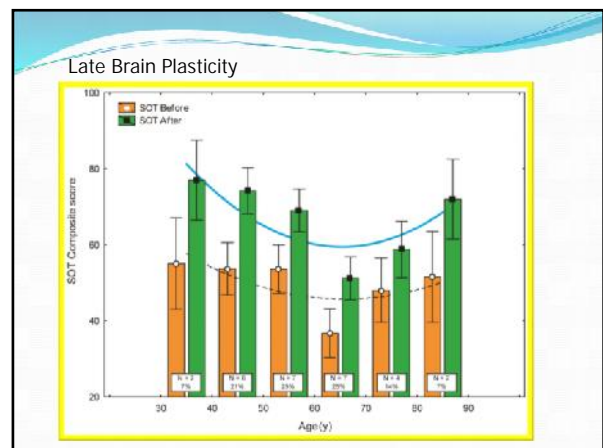


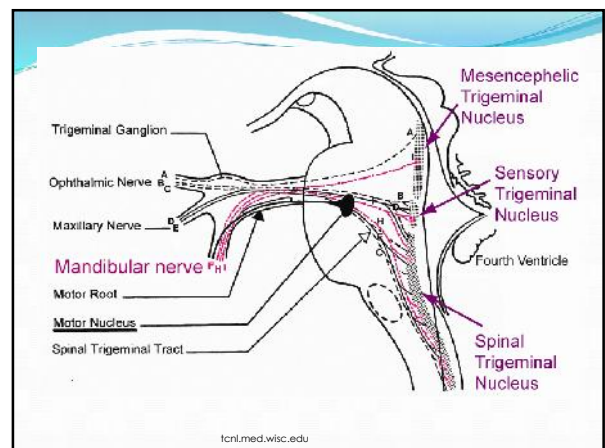
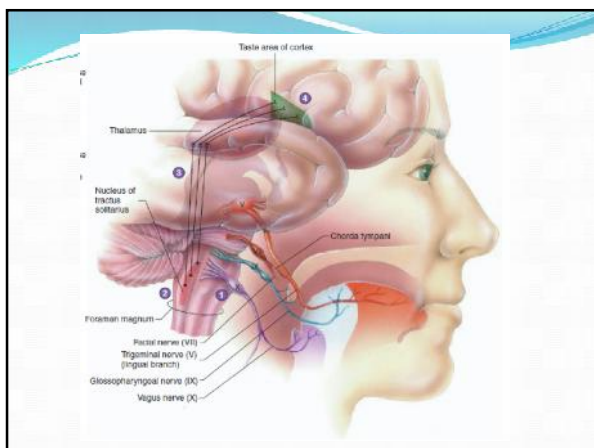
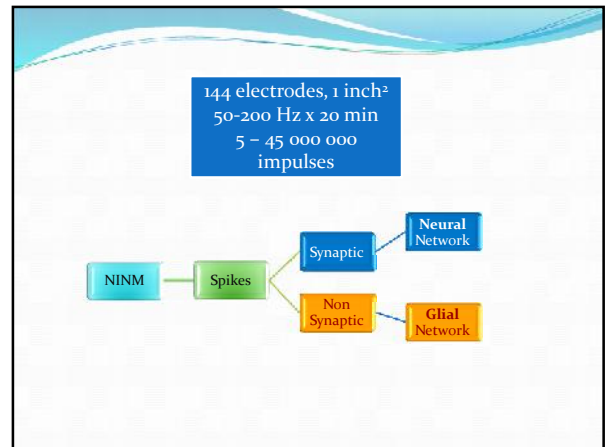
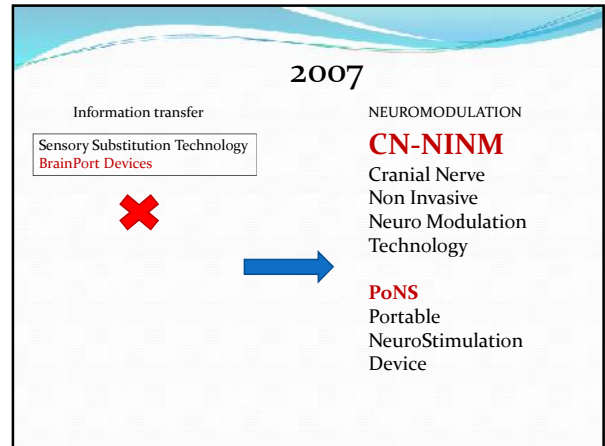
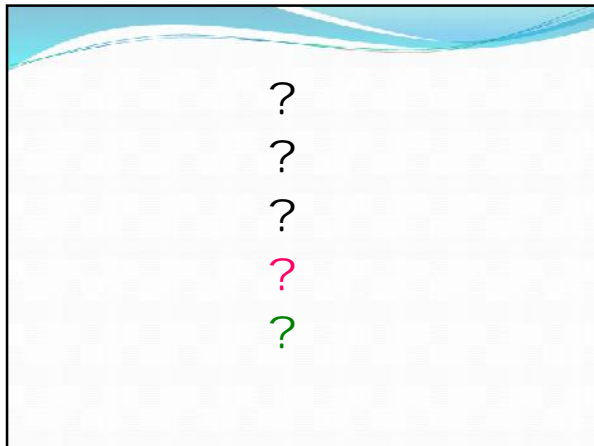


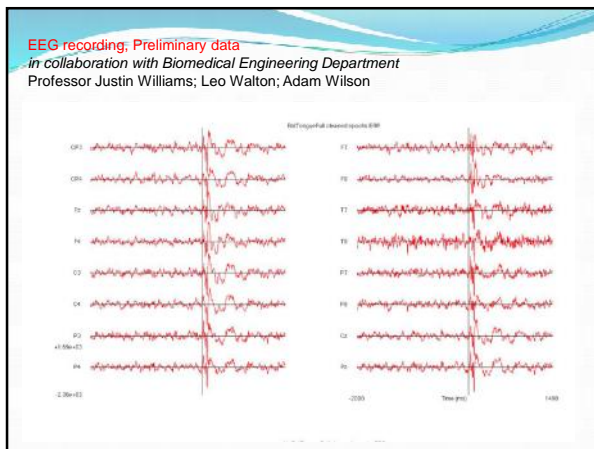
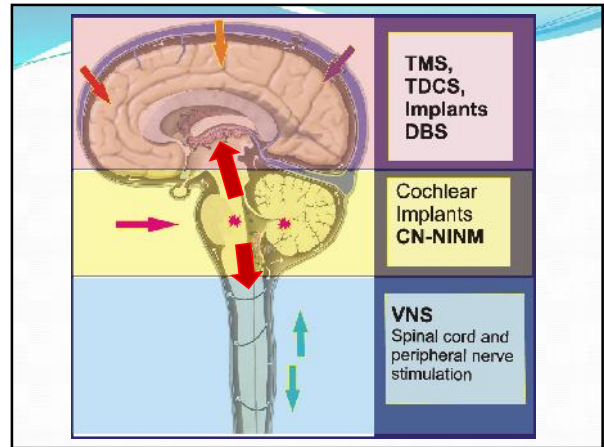
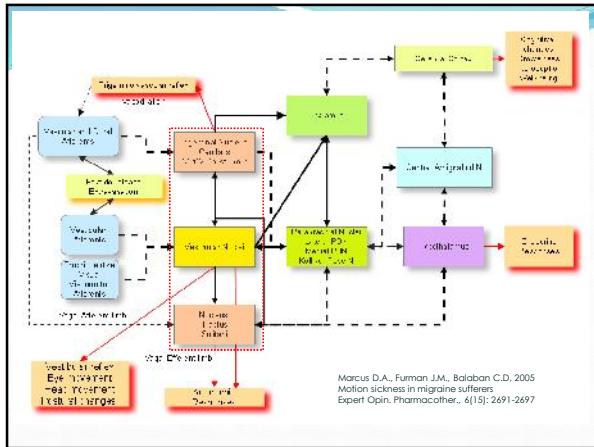
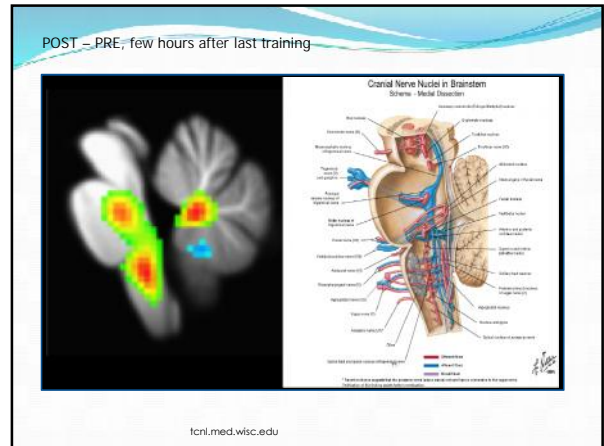
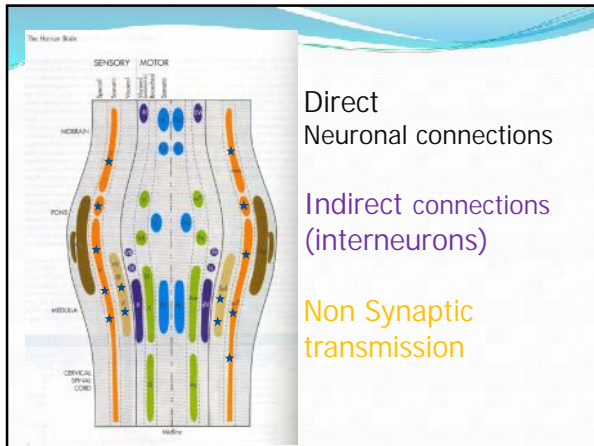
The Beginning:
Bilateral Vestibular Loss
Standing Eyes Closed

Scope of Patients Tested N=80

Nature of Deficit	Etiologies	Common Observations	
		Improved	Reduced
Peripheral	<ul style="list-style-type: none"> Ototoxicity Meniere's disease Acoustic Neuroma Perilymphatic Fistulas Endolymphatic Hydrops Vestibular neuritis 	Balance	Falls
Central	<ul style="list-style-type: none"> Migraine Auto Immune degeneration Mal de Debarquement Idiopathic origin 	Steadiness	Depression
Non-Vestibular	<ul style="list-style-type: none"> Cerebellar lesion, atrophy Cerebellar ataxia - stroke Traumatic Brain Injury Parkinson's Disease MS Neuropathy 	Posture	Migraine
		Muscle Tone	Rigidity
		Gait	Fatigue
		Speech	Oscillopsia
		Sleep	Tinnitus

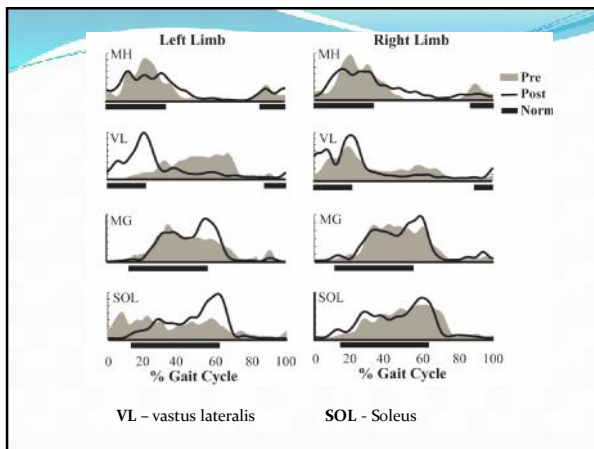
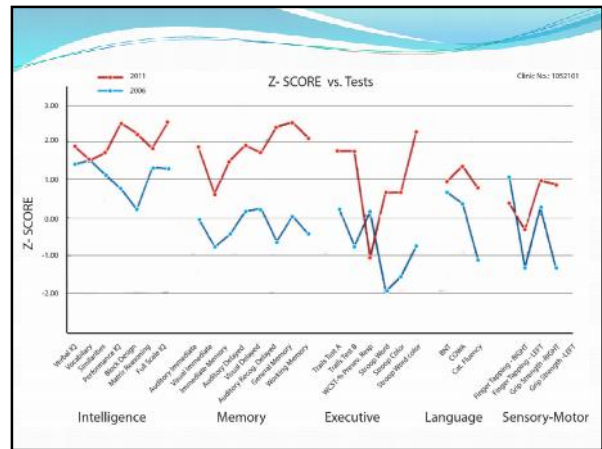
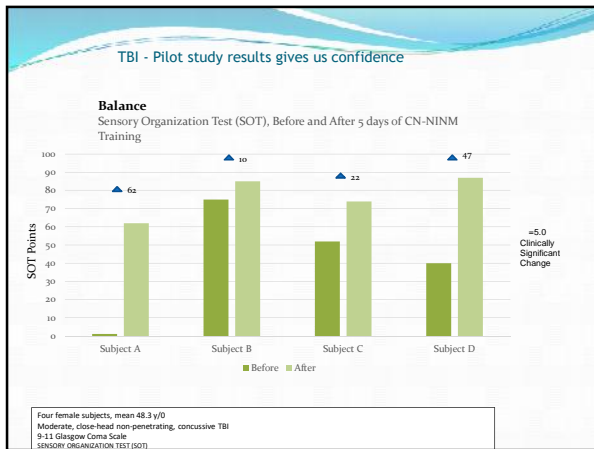
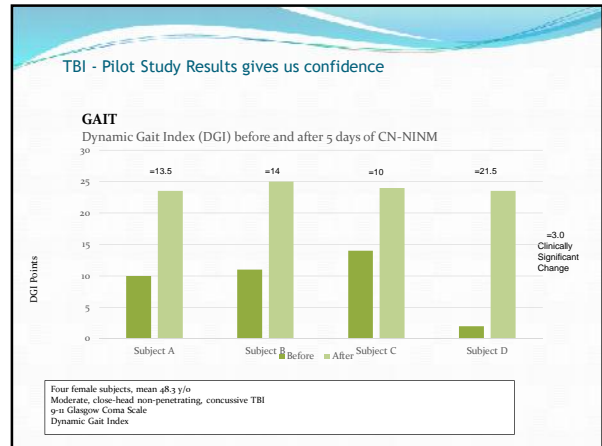
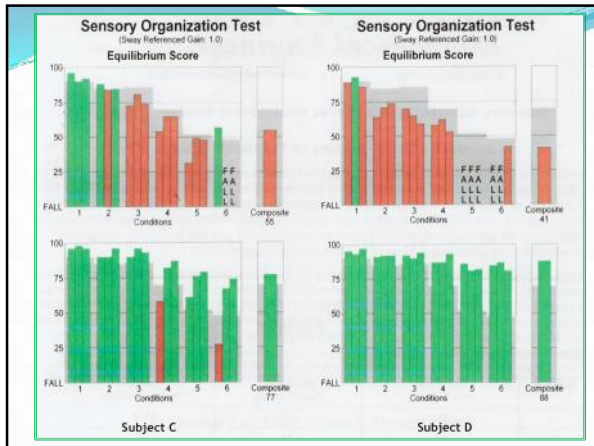




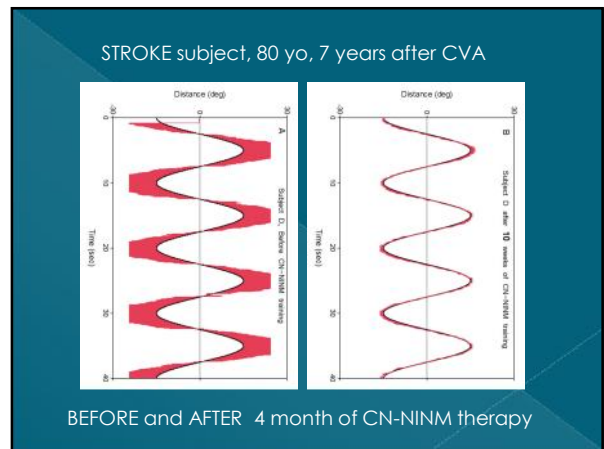
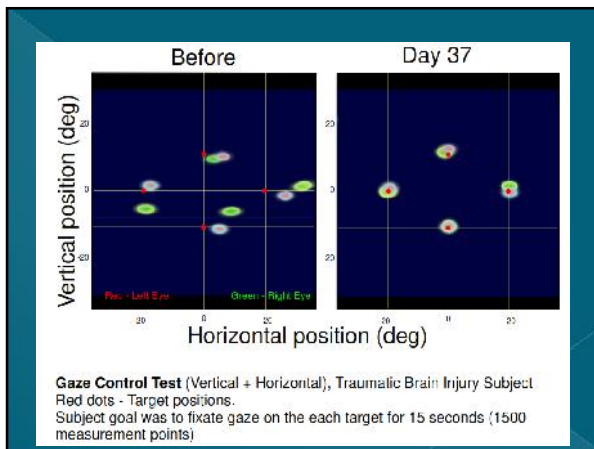
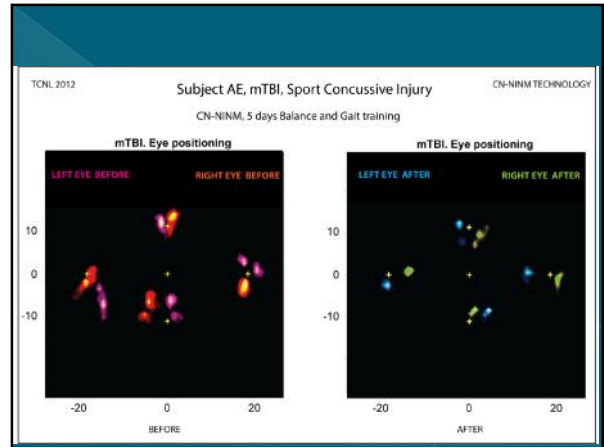
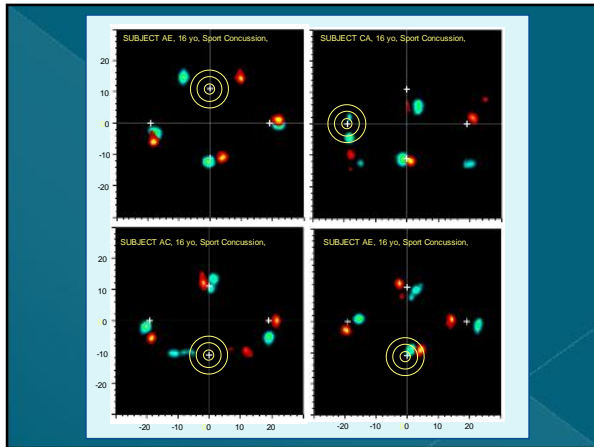
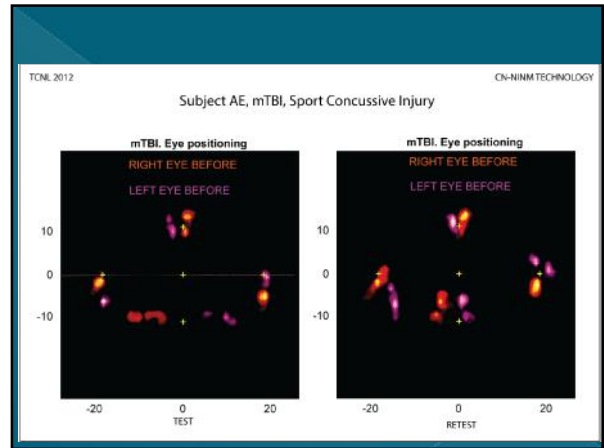
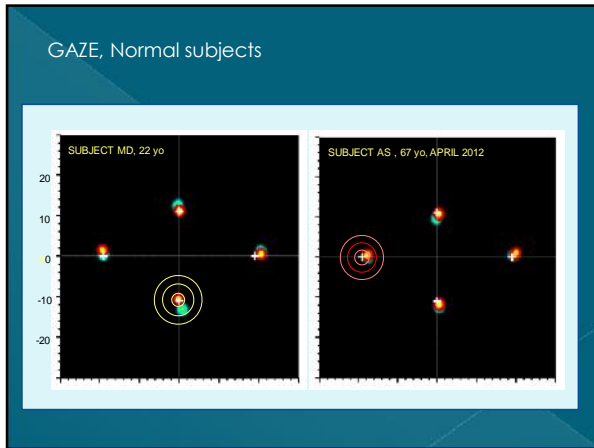


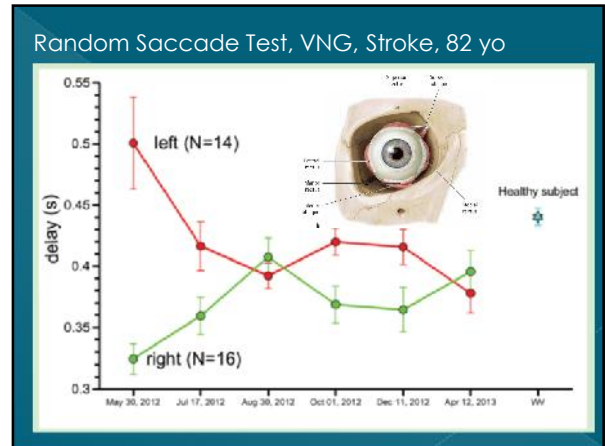
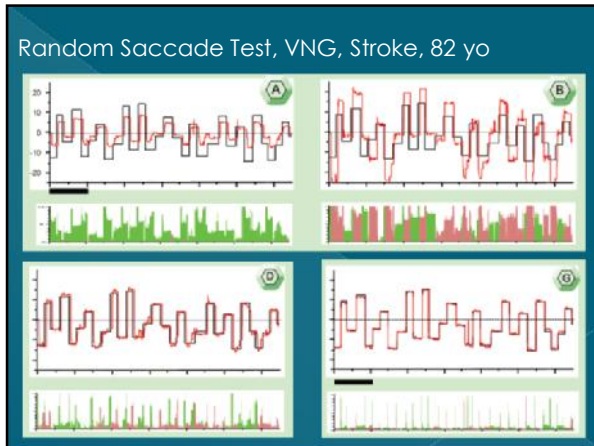
Traumatic brain injury

Results

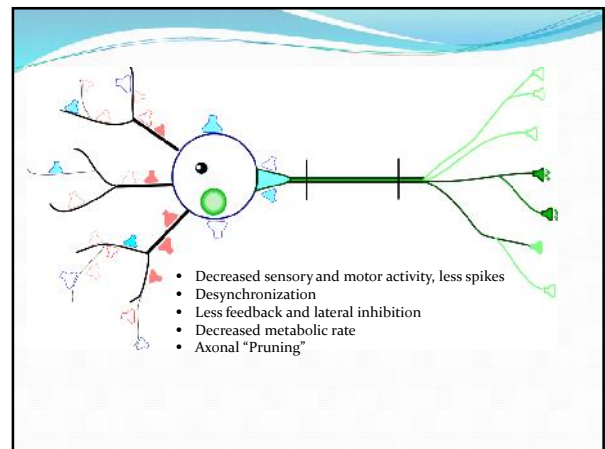
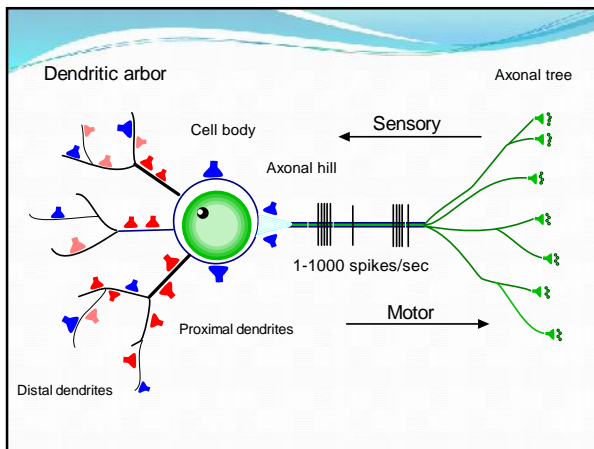
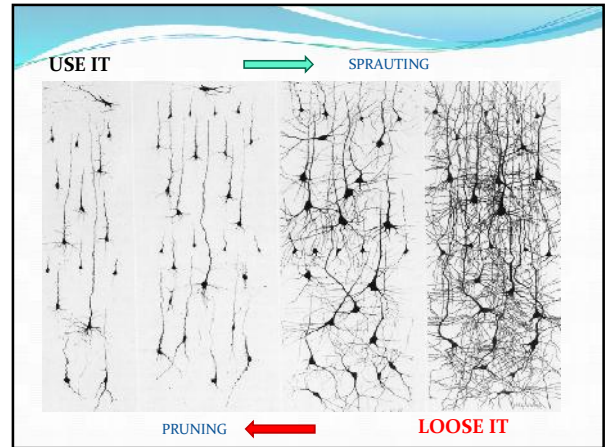


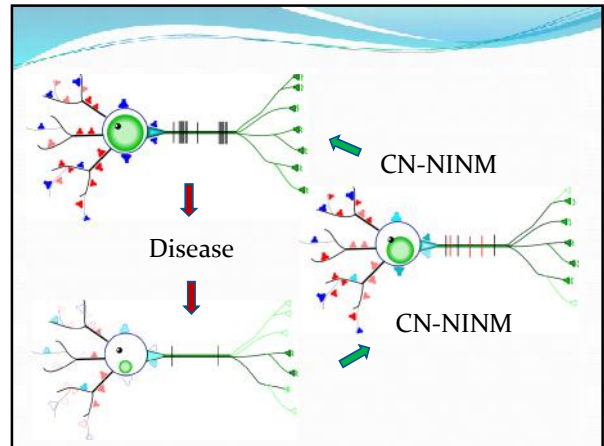
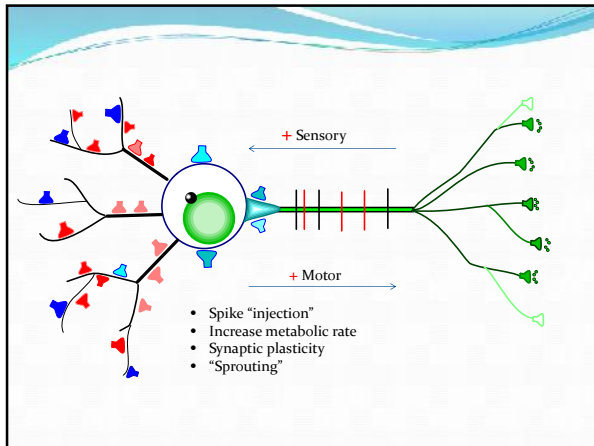
mTBI
 5+ years after injury





Activity Dependent Plasticity





CN-NINM = Enforced Physical Therapy
Brain Stimulation + Exercises (physical, cognitive)

CN-NINM = Rehabilitation Therapy Toolbox
PoNS, Face Stimulator, etc. + Training Protocol

CN-NINM is a Symptom Oriented, Multi targeted Therapy
Mind-Body Interaction, holistic approach

CN-NINM is a patient oriented therapy

CN-NINM is a non invasive and safe therapy

