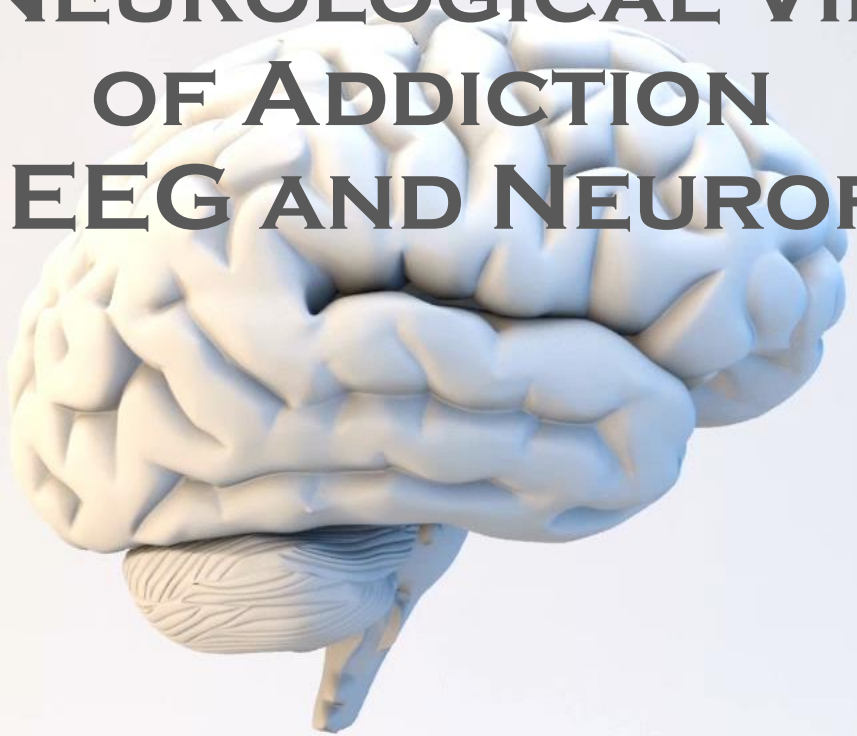
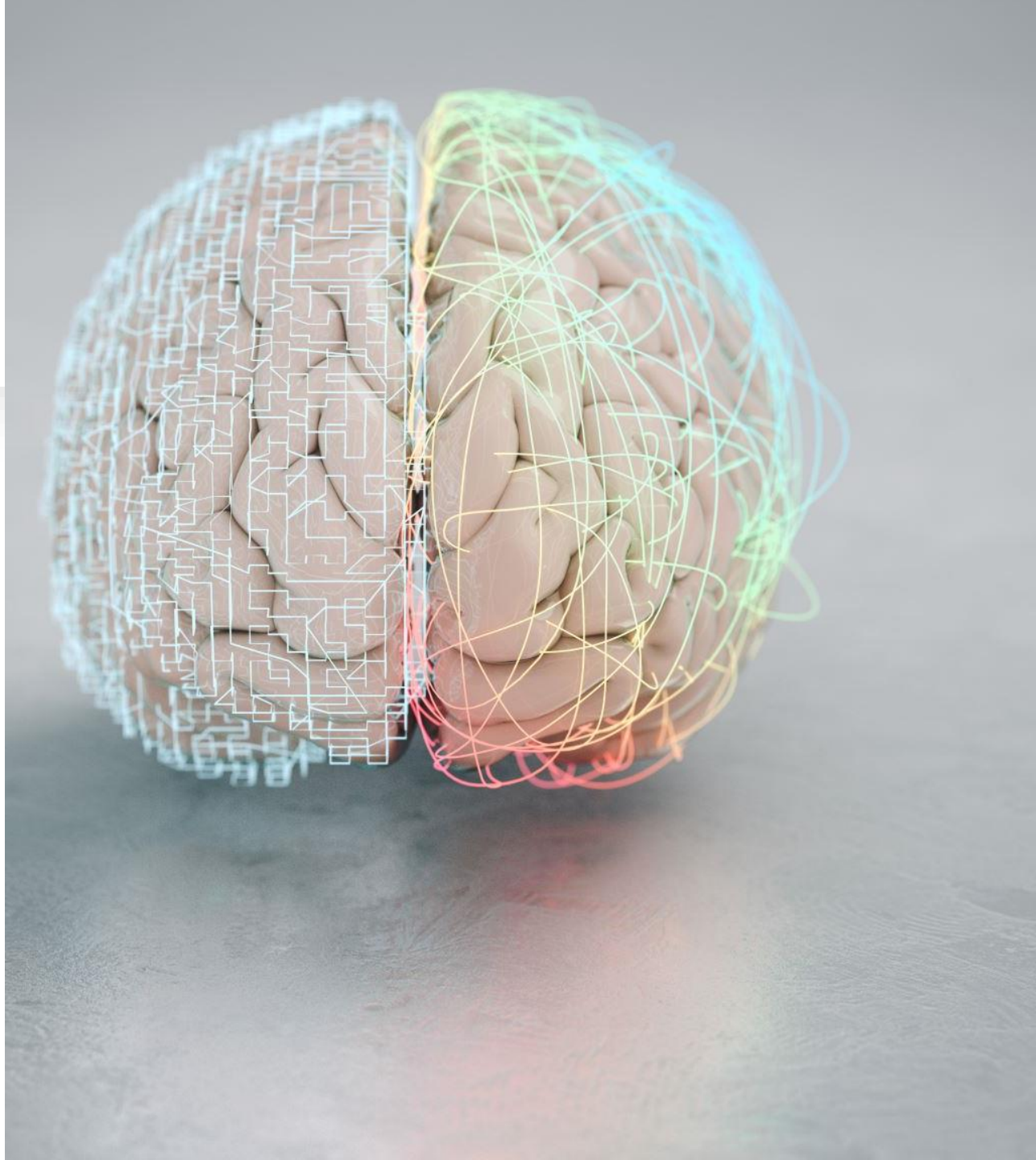
A Neurological View of Addiction through EEG and Neurofeedback

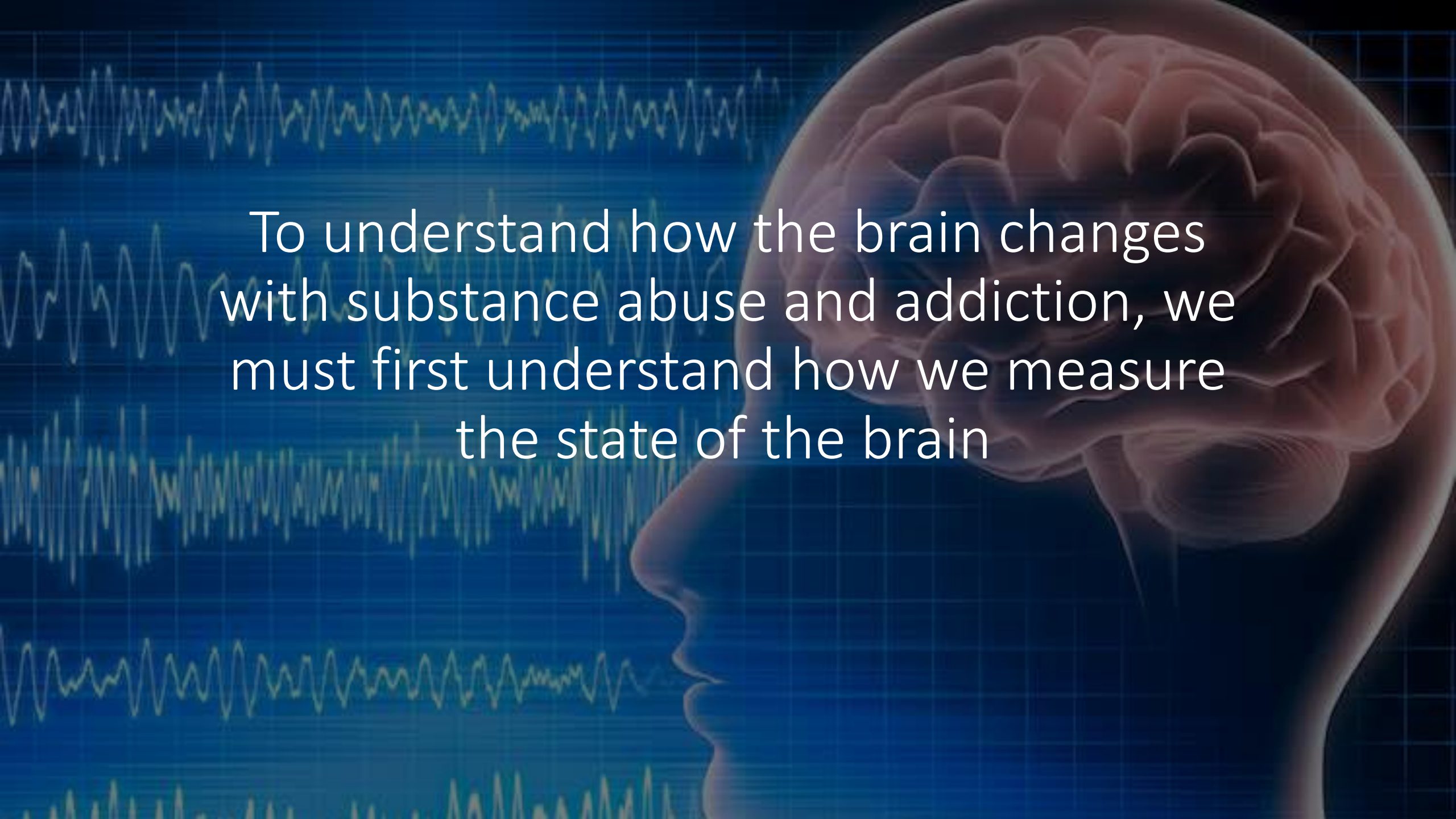


Bethany Brenes, MPA, LPTA

Overview

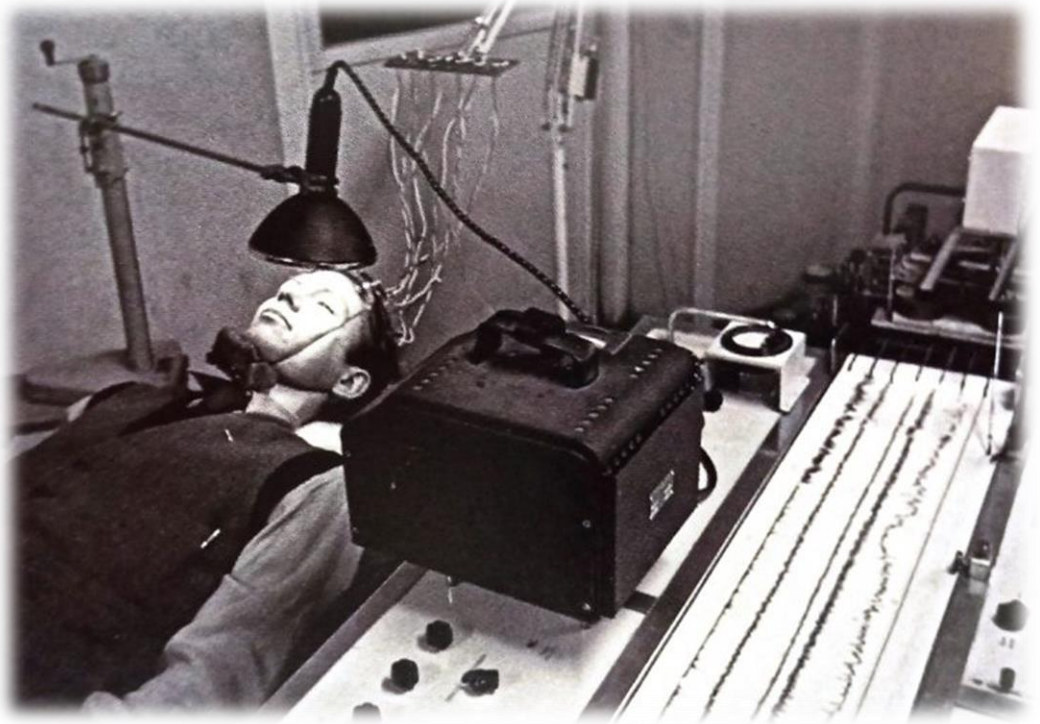
- History of EEG and Neurofeedback
- EEG and Neurofeedback process
- Anatomy of addiction centers in the brain
- Addiction prone EEG profiles
- How different substances activate the brain
- Neuromodulation for addiction profiles




To understand how the brain changes with substance abuse and addiction, we must first understand how we measure the state of the brain

History of EEG

- Hans Berger
- Following the work of Richard Caton, who worked with animals to determine brain activity, Berger made the first EEG recording of human brain activity in **1924**, a procedure he called ‘Elektrenkephalogramm’.
- EEG or Electroencephalogram is the measurement of the electrical activity created when neurons communicate with each other



Neurofeedback

- Neurofeedback was pioneered in the late 1950's and 60's by Joe Kamiya at the University of Chicago and Barry Sternman at UCLA .
- Kamiya found that using a reward system people could control their brain waves. This was the first real time feedback given to humans based on EEG monitoring

Neurofeedback

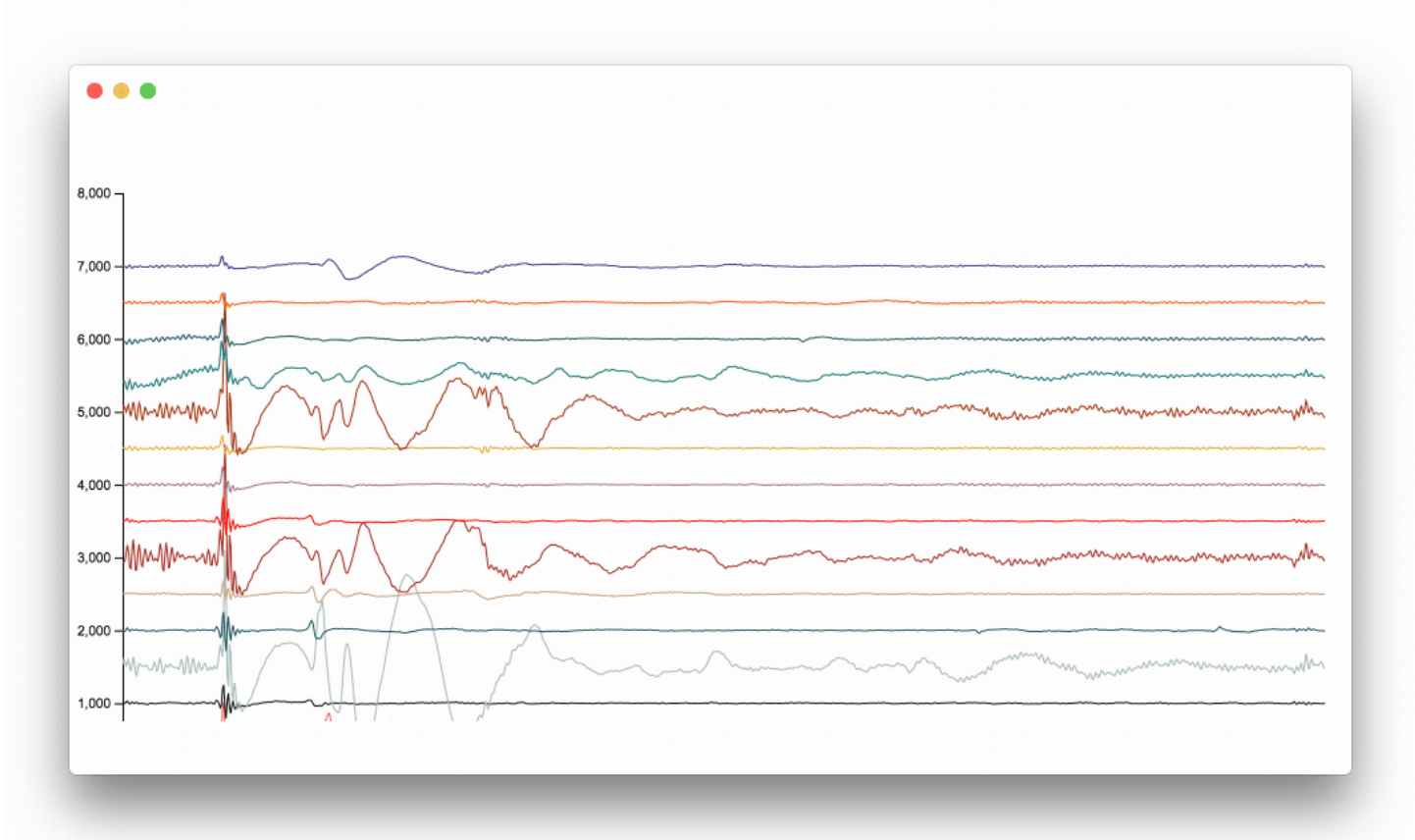
- While performing experiments for NASA Sternman discovered he was able to stabilize seizure activity by training brain waves into an SMR rhythm.
- Sterman moved on to train SMR in humans to control their epilepsy; 60% of his subjects reduced their seizure level by 20-100%, and the results lasted.
- Sternmans neurofeedback training is still part of NASAs astronaut program

Neurofeedback

- In the 1970's neurofeedback bridged between science and religion as it caught the attention of meditators as an aid in spiritual development.
- In the 1980's Joe Lubar helped bring neurofeedback back into the realm of science with his studies on ADHD and the proven long term effects.
- The 1990's started the push to bring neurofeedback into mainstream clinical practice to address a disorders ranging from depression, anxiety, PTSD, and addition to mental enhancement and optimization.

What is EEG?

- EEG neurofeedback training use EEG to monitor and provide real time feedback on brain wave activity. It's a form of biofeedback aimed to help individuals learn to self regulate their brain activity and improve cognitive functioning.
- EEGs are interpreted by the type of brain wave activity found in specific locations.



Delta 0-3hz

Best state-of-mind for deep sleep and full body repair.

Theta 4-7hz

Deep state meditation to allow for creativity.

Alpha 8-12hz

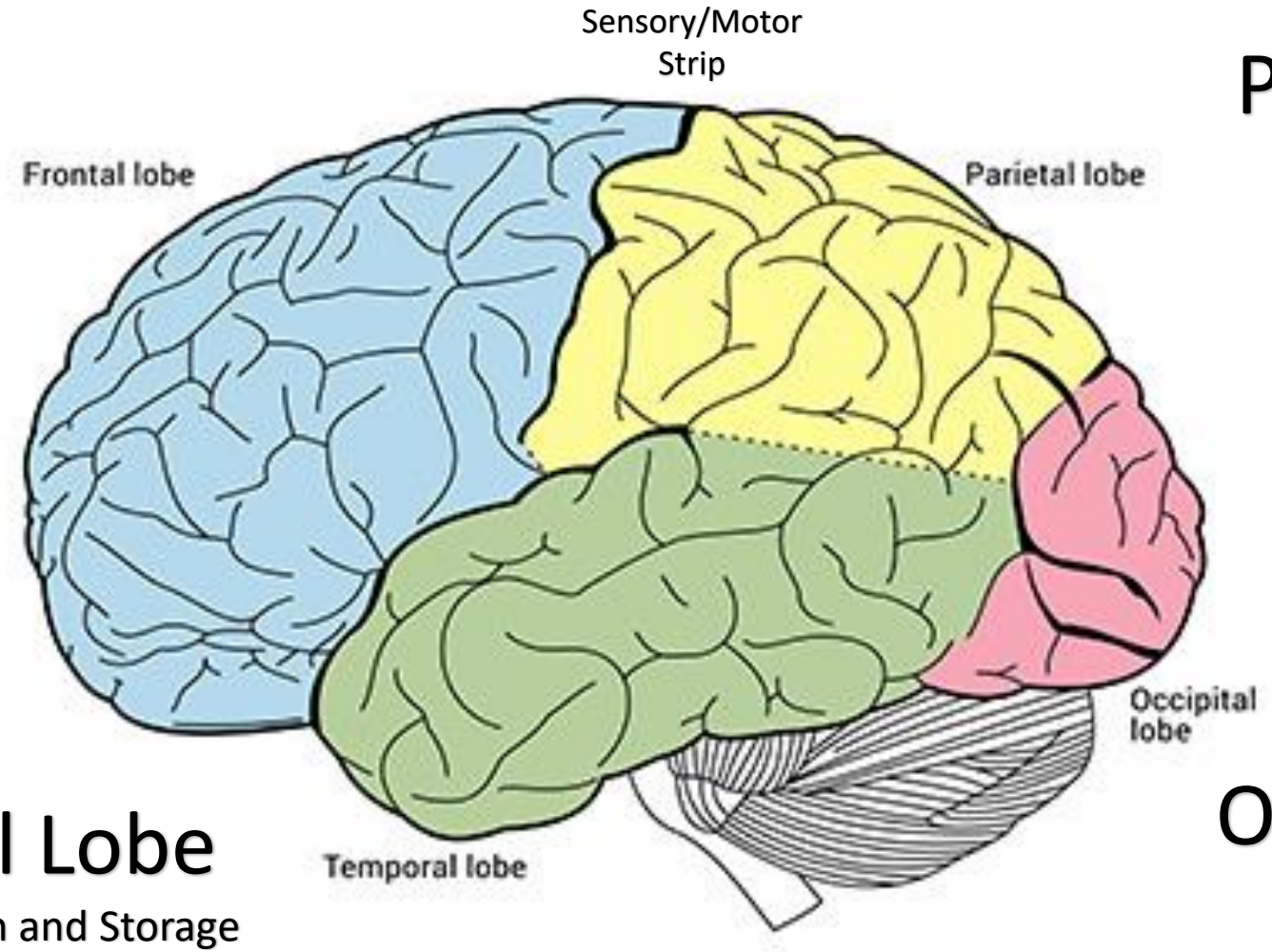
You are physically and mentally relaxed.

Beta 13-28hz

State-of-mind while completing daily tasks.

Frontal

Executive Processing
Emotional Processing
Memory Processing



Parietal Lobe

Spatial Sequencing
Attention Shifting
Calculations
Language

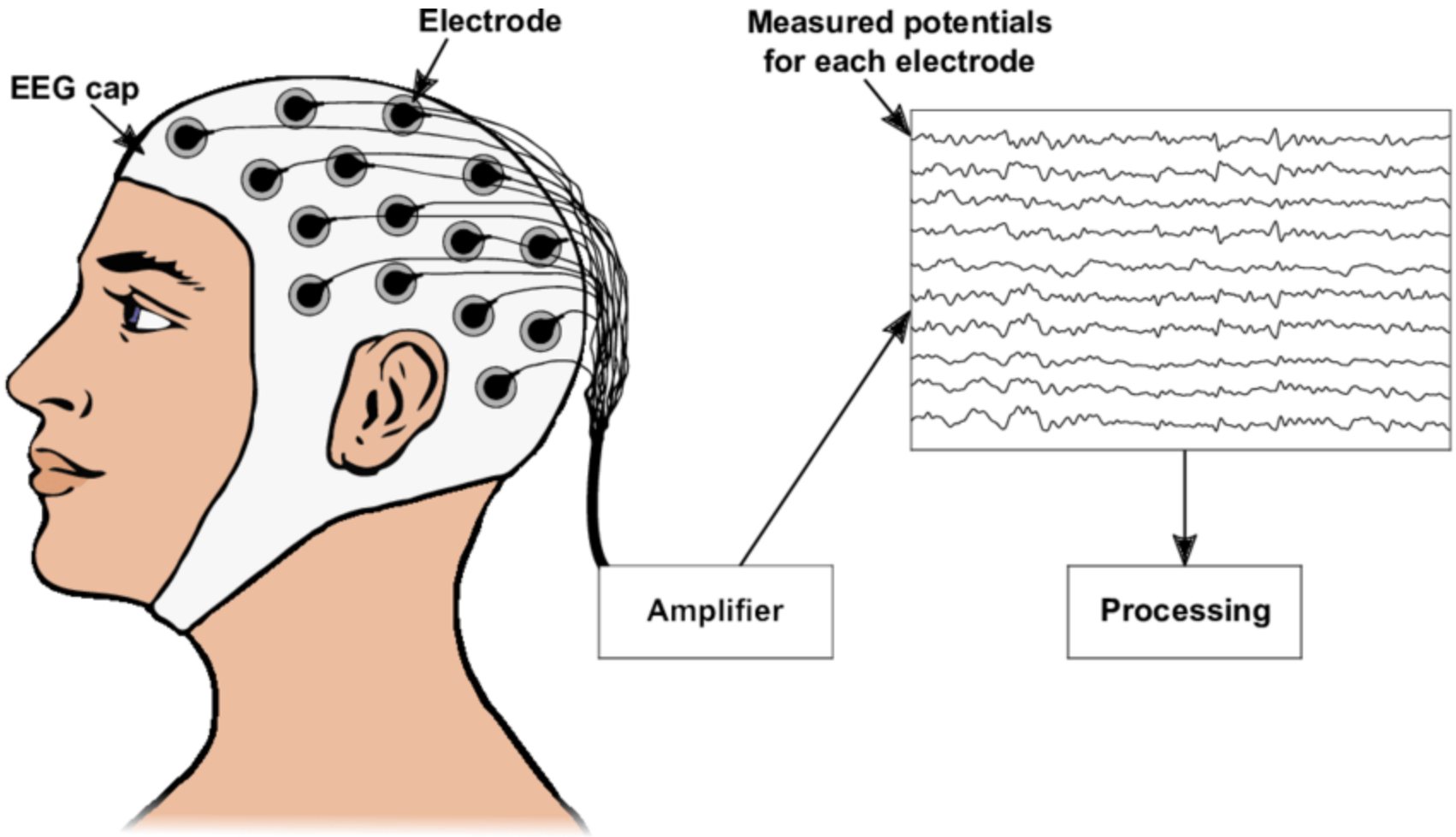
Temporal Lobe

Memory Formation and Storage
Verbal and Auditory Processing
Emotions

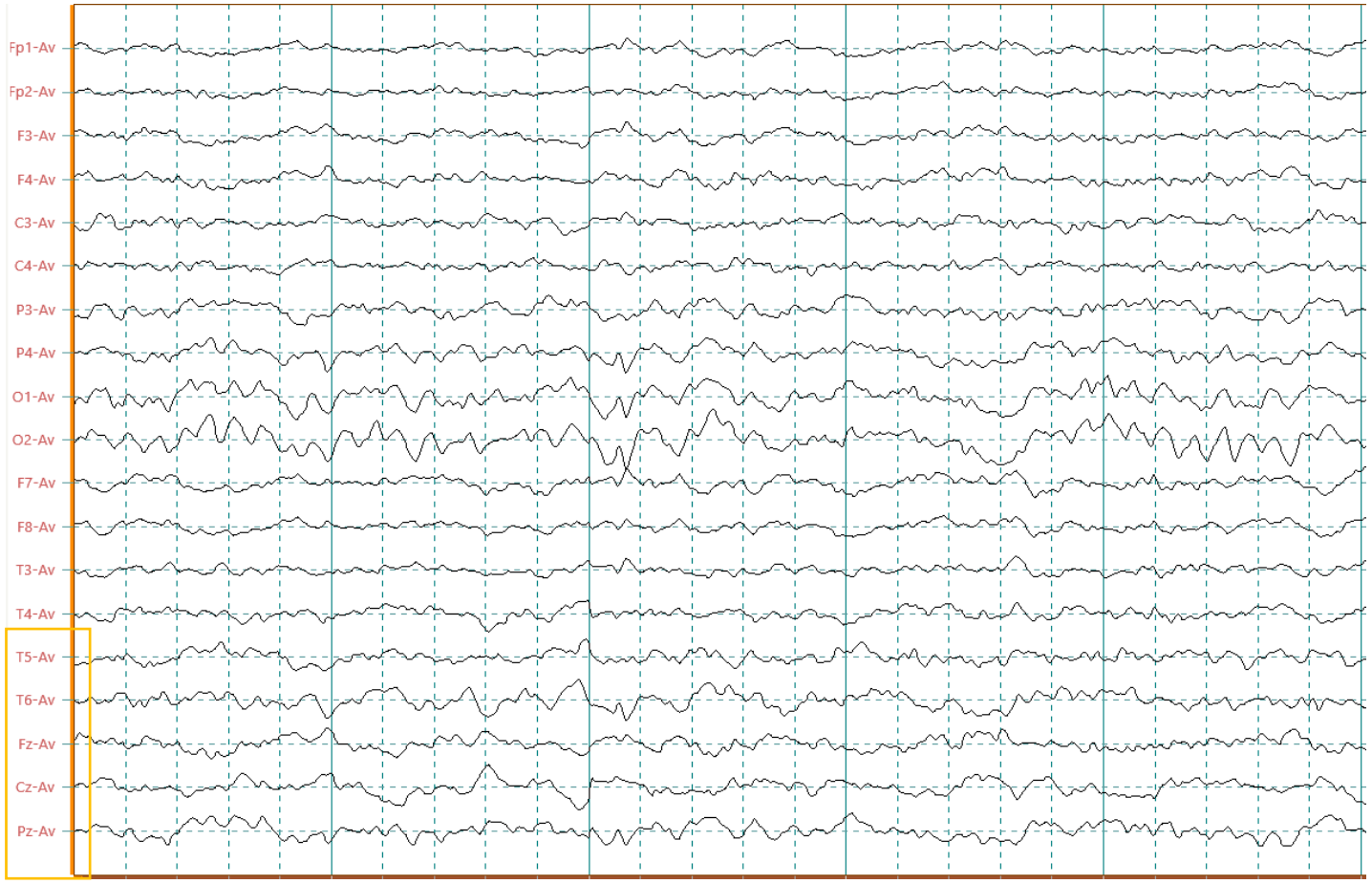
Occipital Lobe

Visual Processing
Perception

Mapping and Analysis

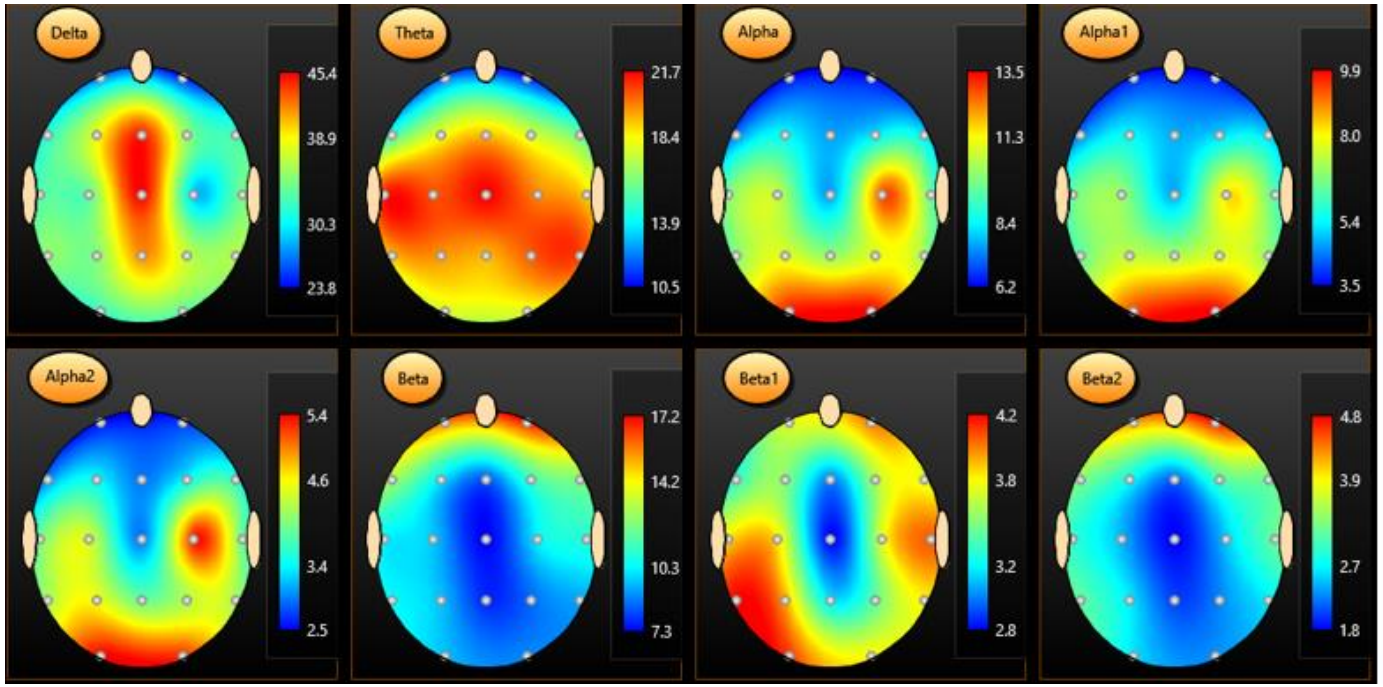


EEG



Slow delta waves at midline
Slow theta waves global and posterior

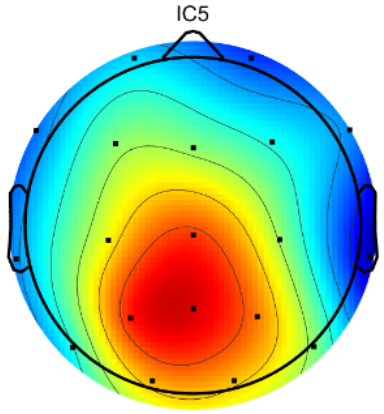
Head Maps

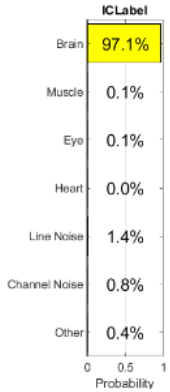


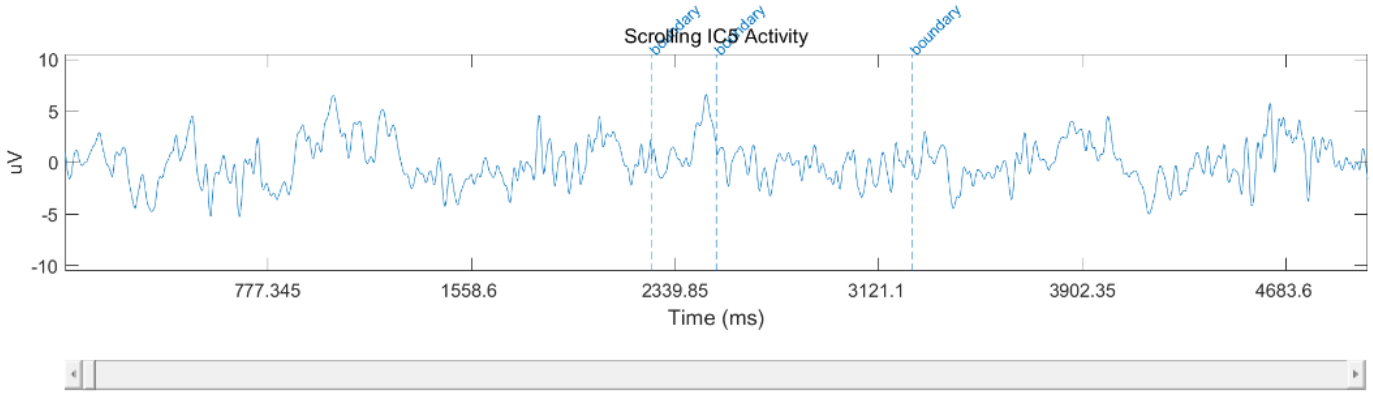
Slow Delta at midline
Slow theta global and posterior

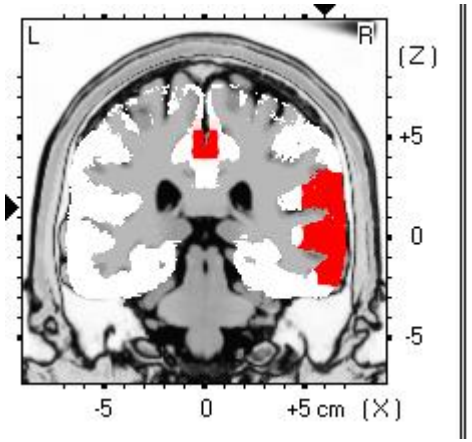
- attention
-verbal and auditory processing
-focus
-memory

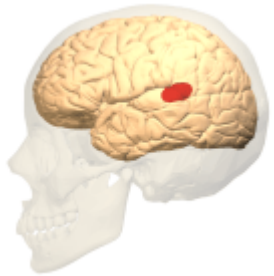
ICA Analysis







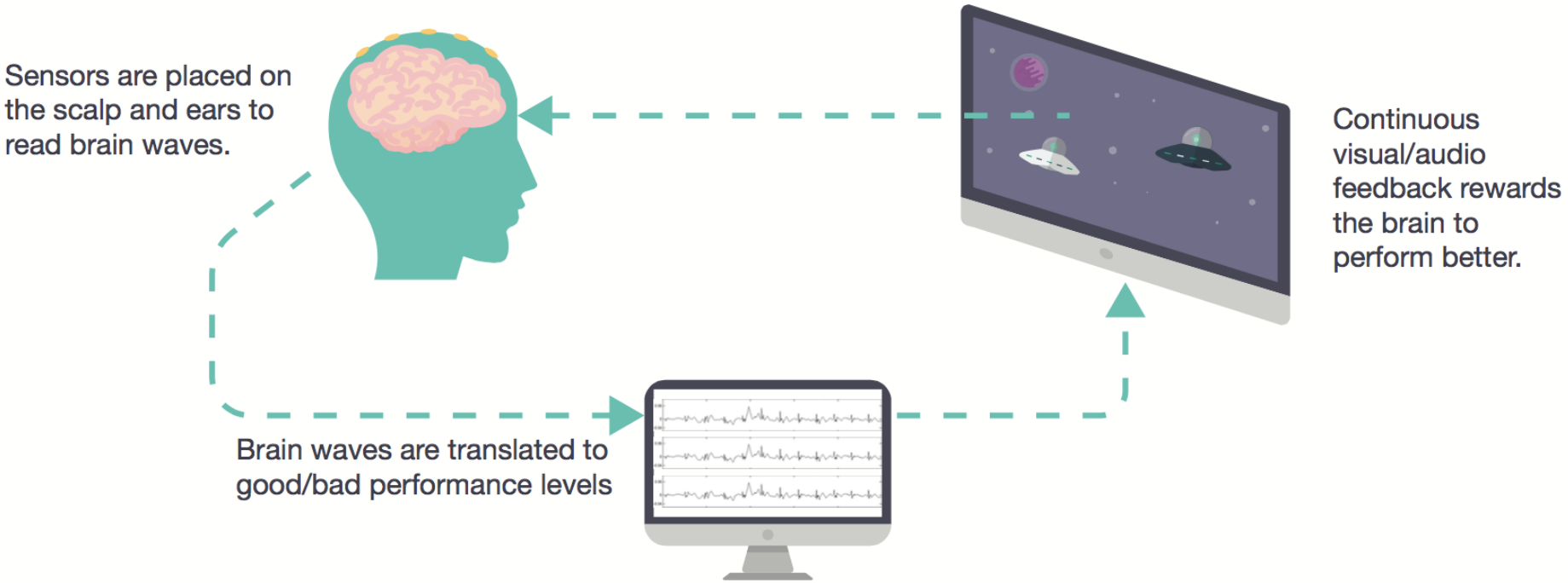




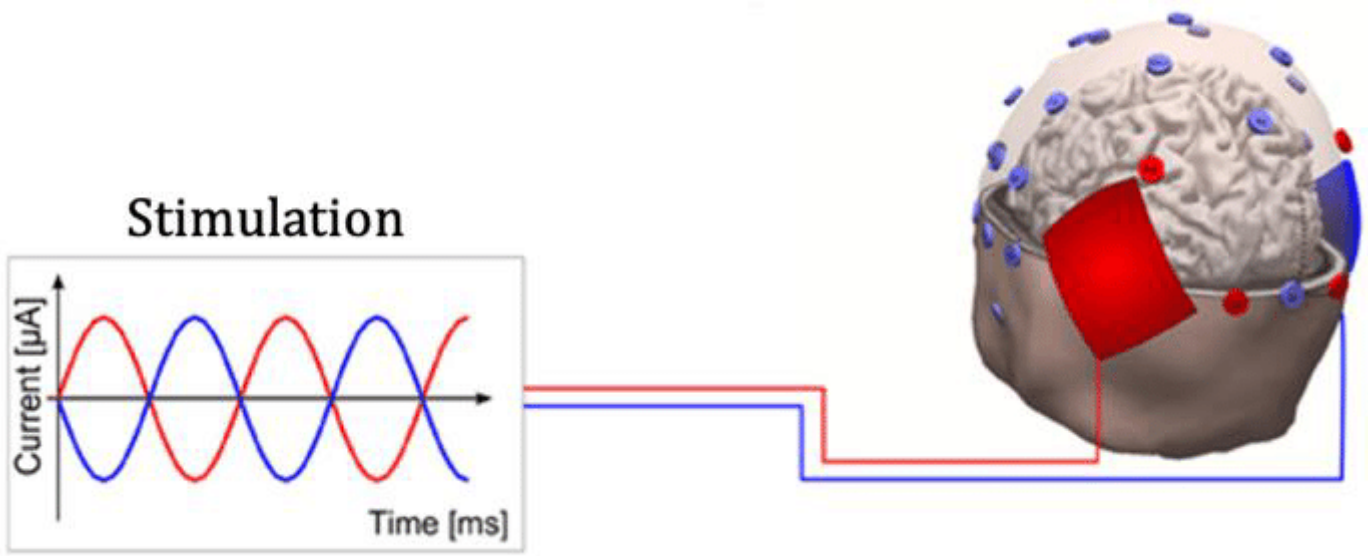
Brodmanns area 42- auditory cortex

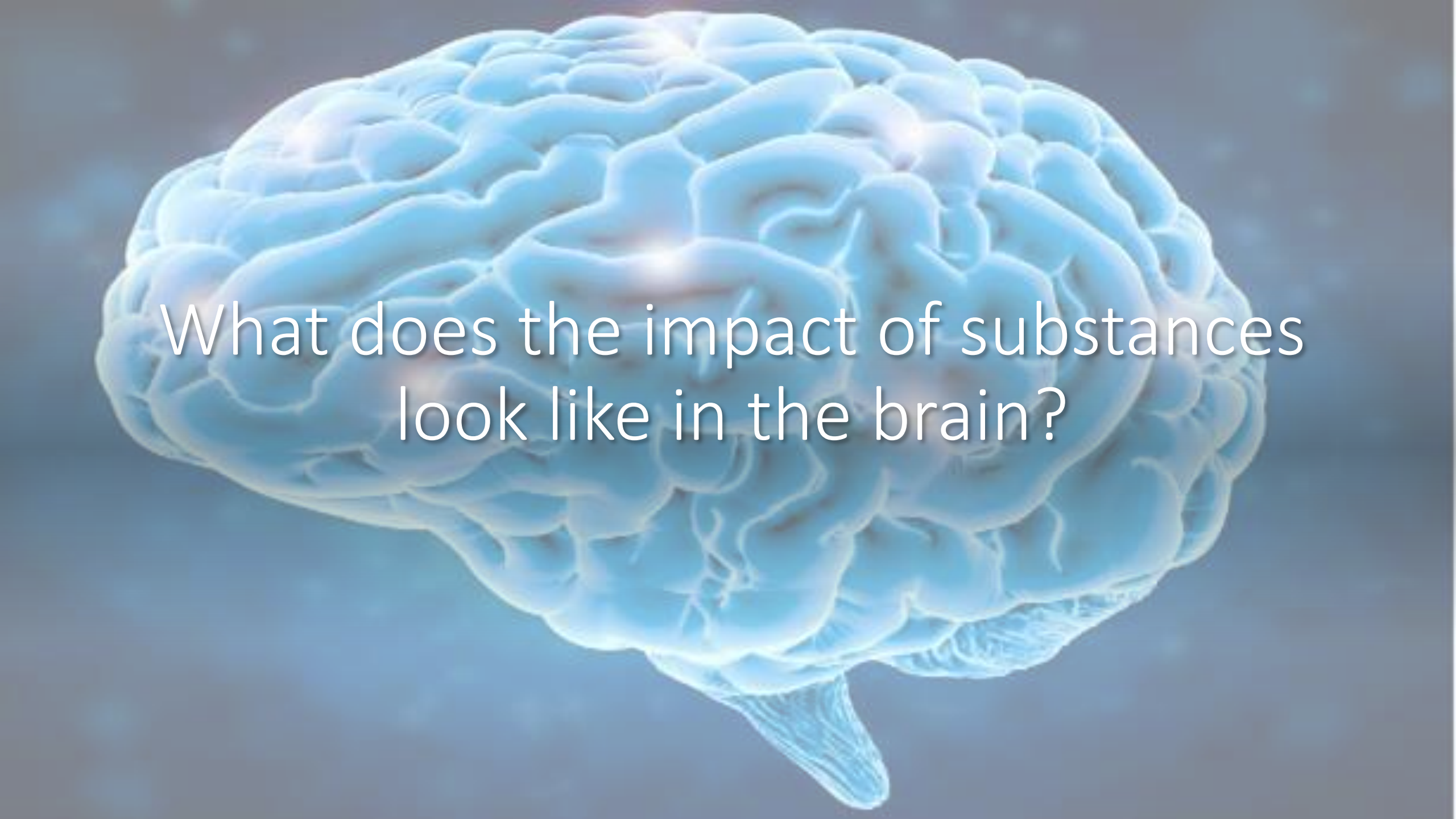
Neurofeedback

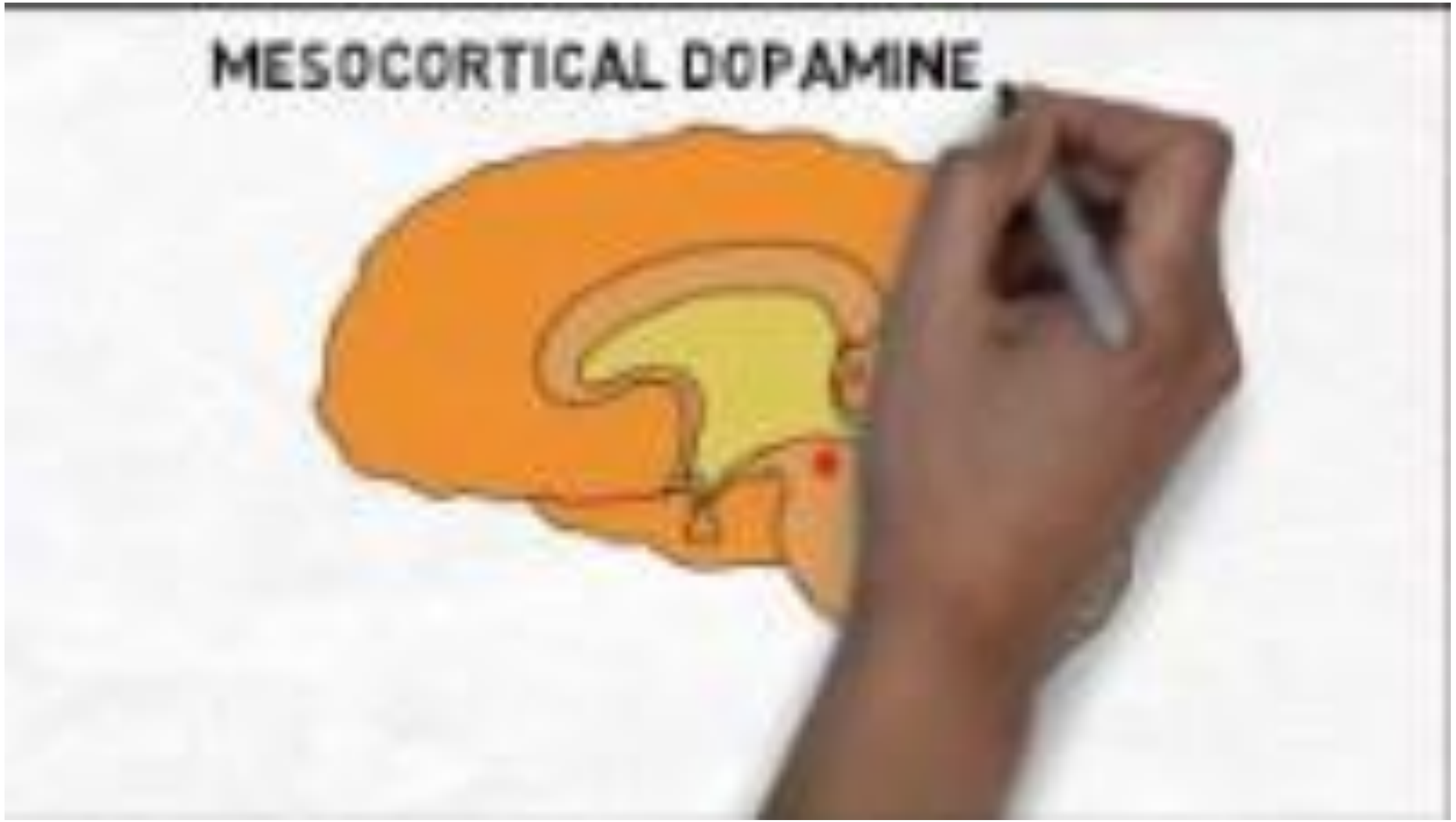
operant conditioning



Neurostimulation




What does the impact of substances look like in the brain?

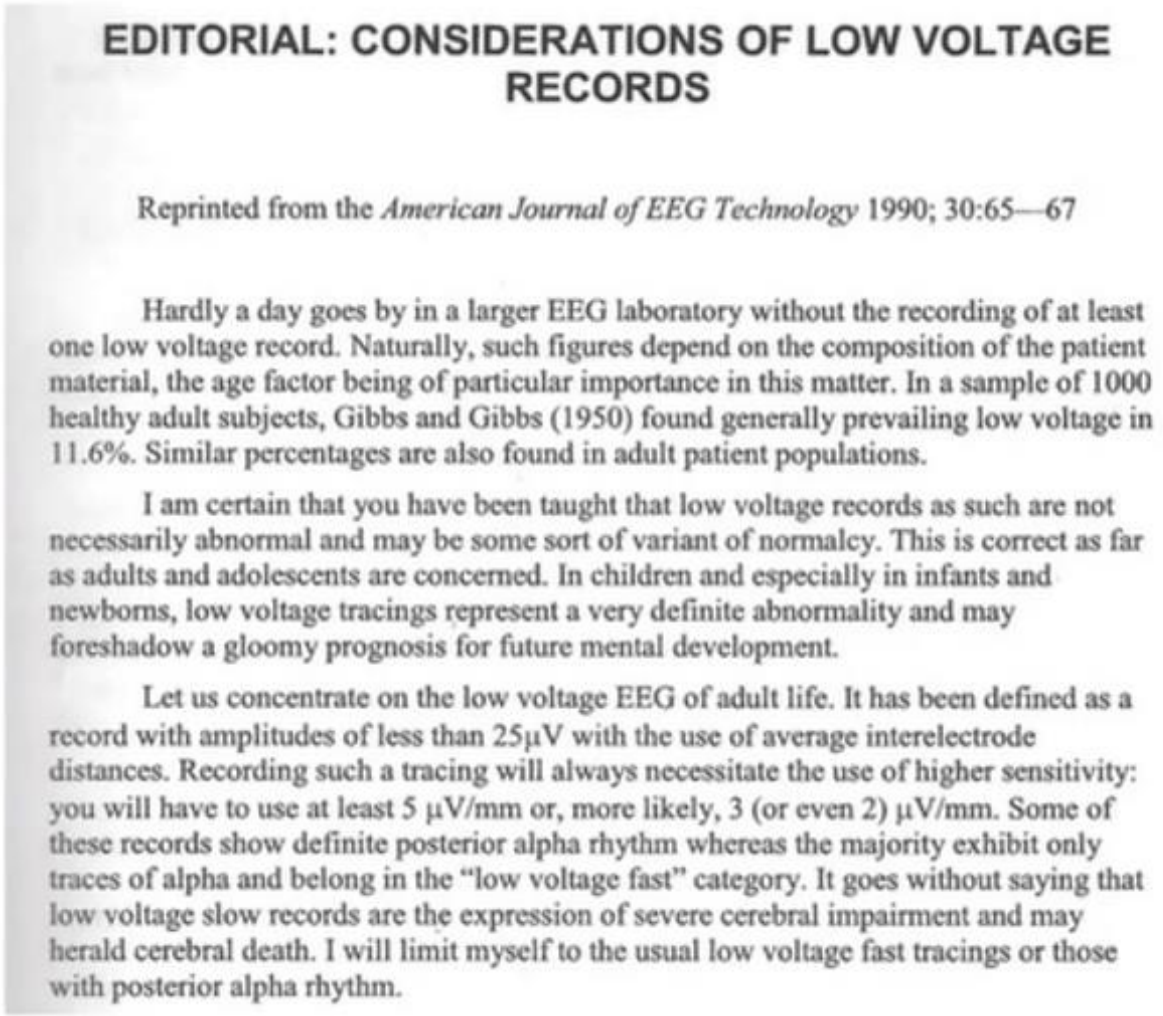

MESOCORTICAL DOPAMINE

To Summarize

- The release of dopamine from the mesolimbic pathway into the nucleus accumbens regulates incentive salience (motivation and desire for rewarding stimuli and reward related motor function learning
- The dysregulation of the mesolimbic pathway and its output neurons in the nucleus accumbens plays a significant role in the development and maintenance of addiction

Common addiction prone profiles

- Gibbs and Gibbs (1950) found 11.6% of 1,000 EEGs to be low voltage EEG
  - Records of less than 25mV
  - Defined as very tense persons “emotional tension”
  - Head injury
  - Chronic alcoholism



EDITORIAL: CONSIDERATIONS OF LOW VOLTAGE RECORDS

Reprinted from the *American Journal of EEG Technology* 1990; 30:65—67

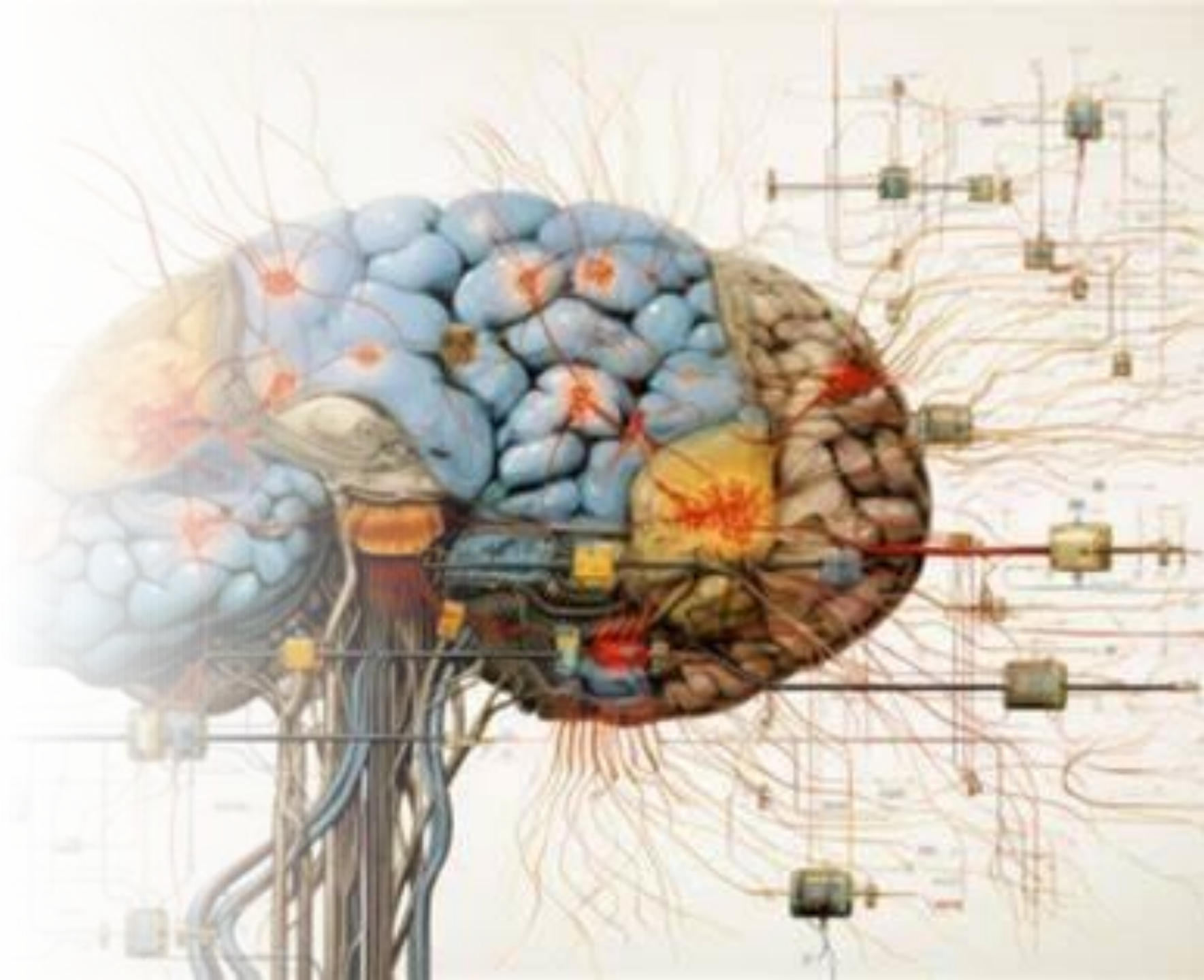
Hardly a day goes by in a larger EEG laboratory without the recording of at least one low voltage record. Naturally, such figures depend on the composition of the patient material, the age factor being of particular importance in this matter. In a sample of 1000 healthy adult subjects, Gibbs and Gibbs (1950) found generally prevailing low voltage in 11.6%. Similar percentages are also found in adult patient populations.

I am certain that you have been taught that low voltage records as such are not necessarily abnormal and may be some sort of variant of normalcy. This is correct as far as adults and adolescents are concerned. In children and especially in infants and newborns, low voltage tracings represent a very definite abnormality and may foreshadow a gloomy prognosis for future mental development.

Let us concentrate on the low voltage EEG of adult life. It has been defined as a record with amplitudes of less than 25μV with the use of average interelectrode distances. Recording such a tracing will always necessitate the use of higher sensitivity: you will have to use at least 5 μV/mm or, more likely, 3 (or even 2) μV/mm. Some of these records show definite posterior alpha rhythm whereas the majority exhibit only traces of alpha and belong in the “low voltage fast” category. It goes without saying that low voltage slow records are the expression of severe cerebral impairment and may herald cerebral death. I will limit myself to the usual low voltage fast tracings or those with posterior alpha rhythm.

Substances

- Stimulants
- Alcohol
- THC
- Benzodiazepines
- Opiates
- OTC medications



Stimulants – ADHD meds

- Effects in the EEG
  - The stimulants may normalize the EEG by taking out the excessive slow seen in ADHD profiles
  - Dependent on the baseline before the stimulants were onboard
  - Stimulants will shrink high amplitude slow and increase beta

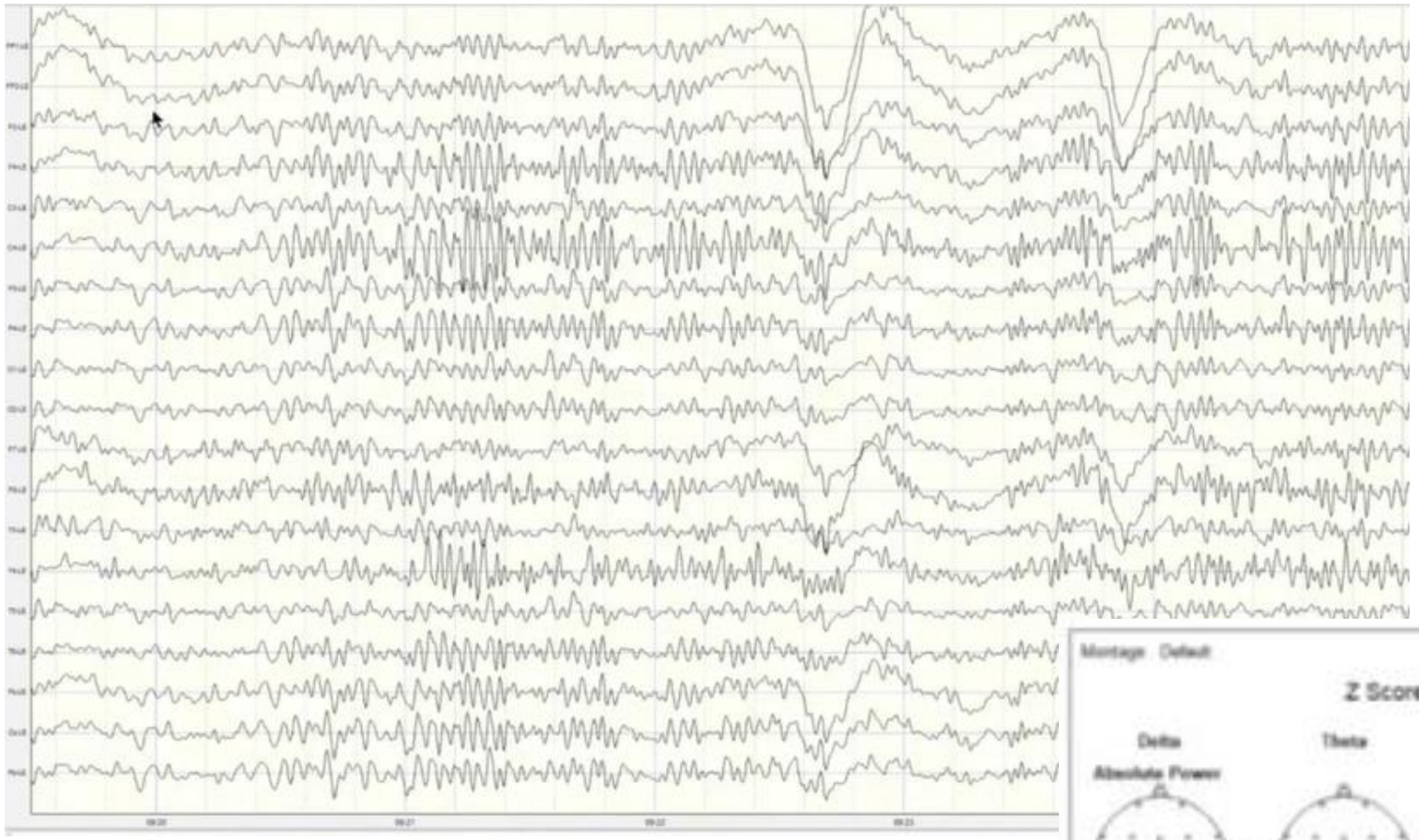
- Effect of the substances
  - With the increase of dopamine and norepinephrine, stimulants lead to heightened alertness and feelings of euphoria
  - Increased alertness, attention, and energy while also elevating BP, HR and breathing which can lead to heart failure and seizure.

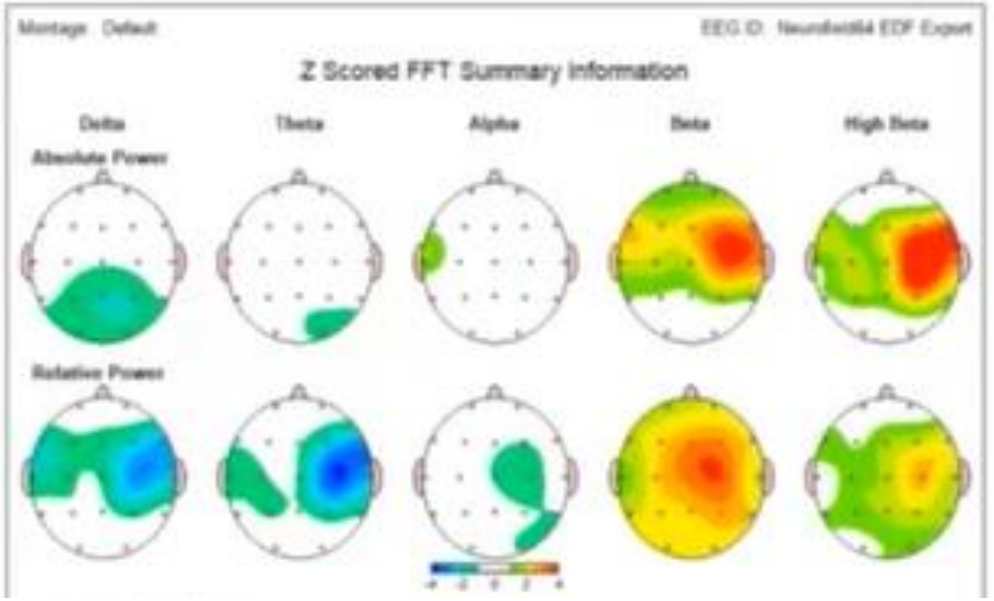
Adderall vs. Ritalin

- Amphetamines: both Dextrostat and Adderal are similar with decreased slower activity and increased beta (12-26hz)
- Methylphenidate: Ritalin produces decrease in delta and theta and more pronounced posterior alpha increase and increase in low beta. These effects can be delayed up to 6 hrs compared to amphetamines

Methamphedamine

- Due to the acceleration and lack of restoration, long term users present with a reduced ability to produce dopamine



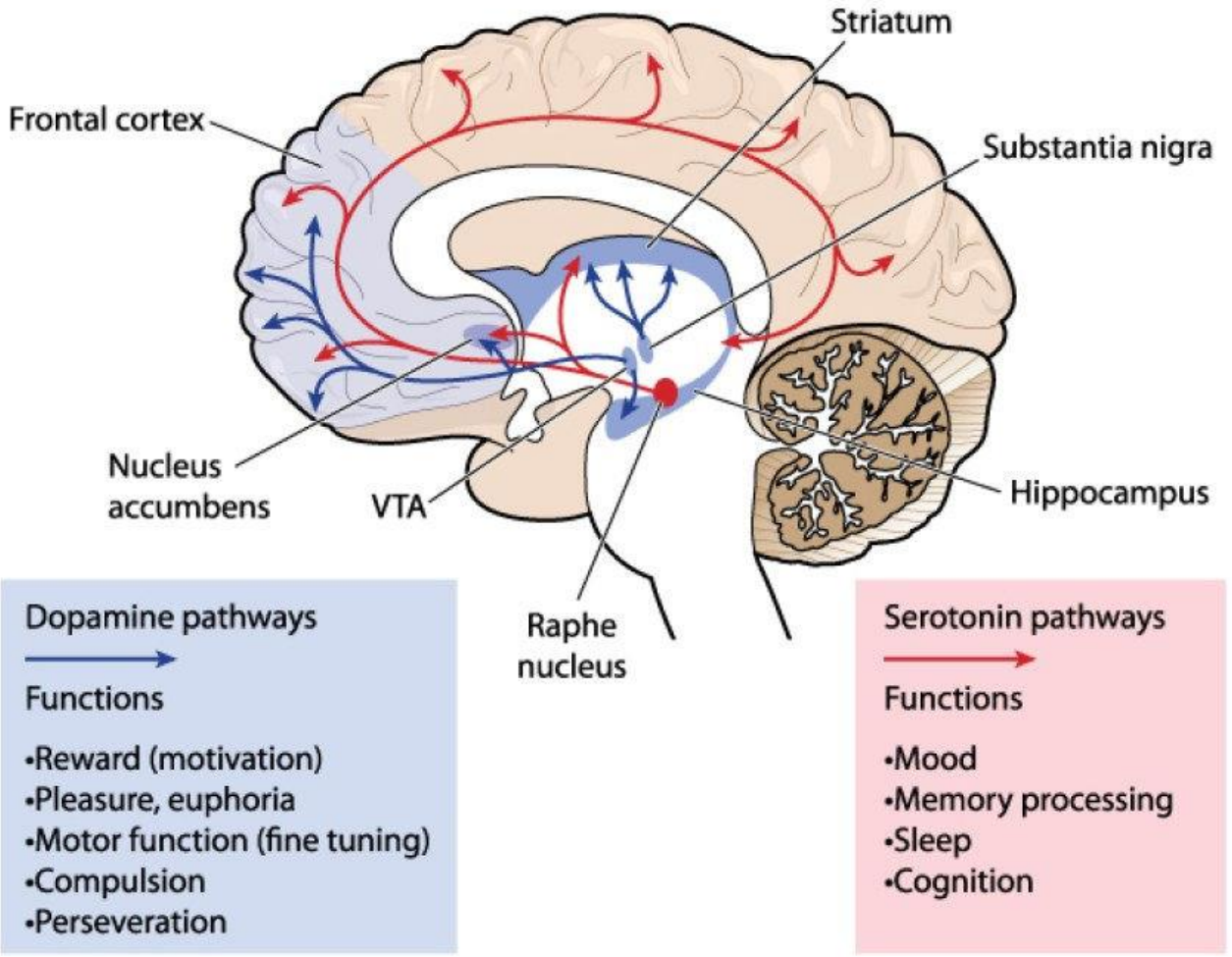


- Hi amplitude beta

Alcohol

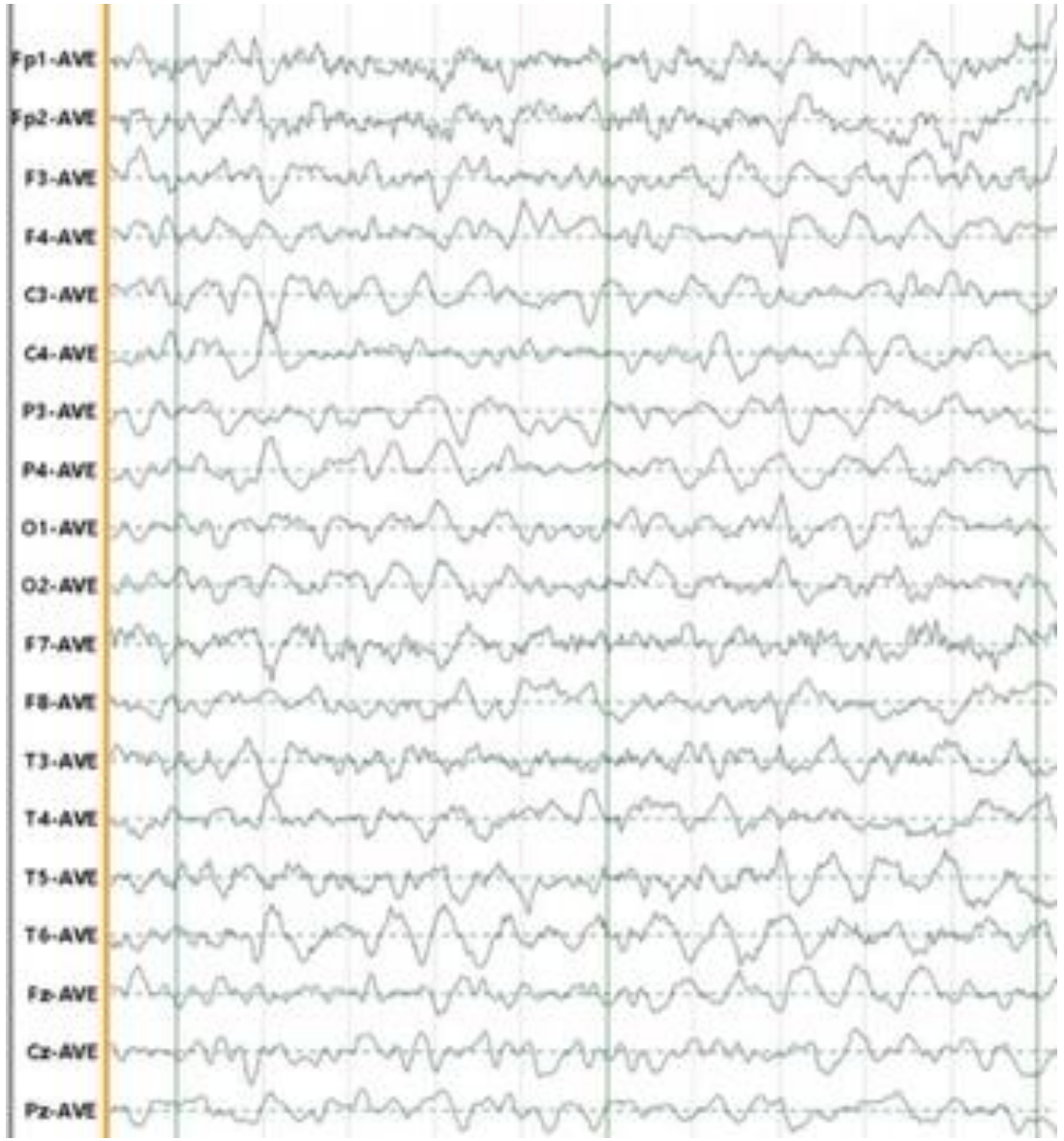
- Delta productionis tied into the release of dopamine
- Most delta is midline and anterior which is in line with the mesotelencephalic dopamine system.
- There is a strong tie between the production of delta and the production of dopamine
- Alcohol increases dopamine across the stiatum, but as tolerance delvelops demand increases.
- Over time there becomes a permanent dynamic homeostatic change which alters opiod signaling and dysfunction

Mesotelencephalic Dopamine System



THC

- Marijuana and its sister drugs will increase frontal alpha
- The effects are seen in dampened reaction time in evoked potentials
- Frontal alpha may also mimic an ADHD profile
- It also may reduce signs of an anxious profile
- The alpha seen is diffuse and slow

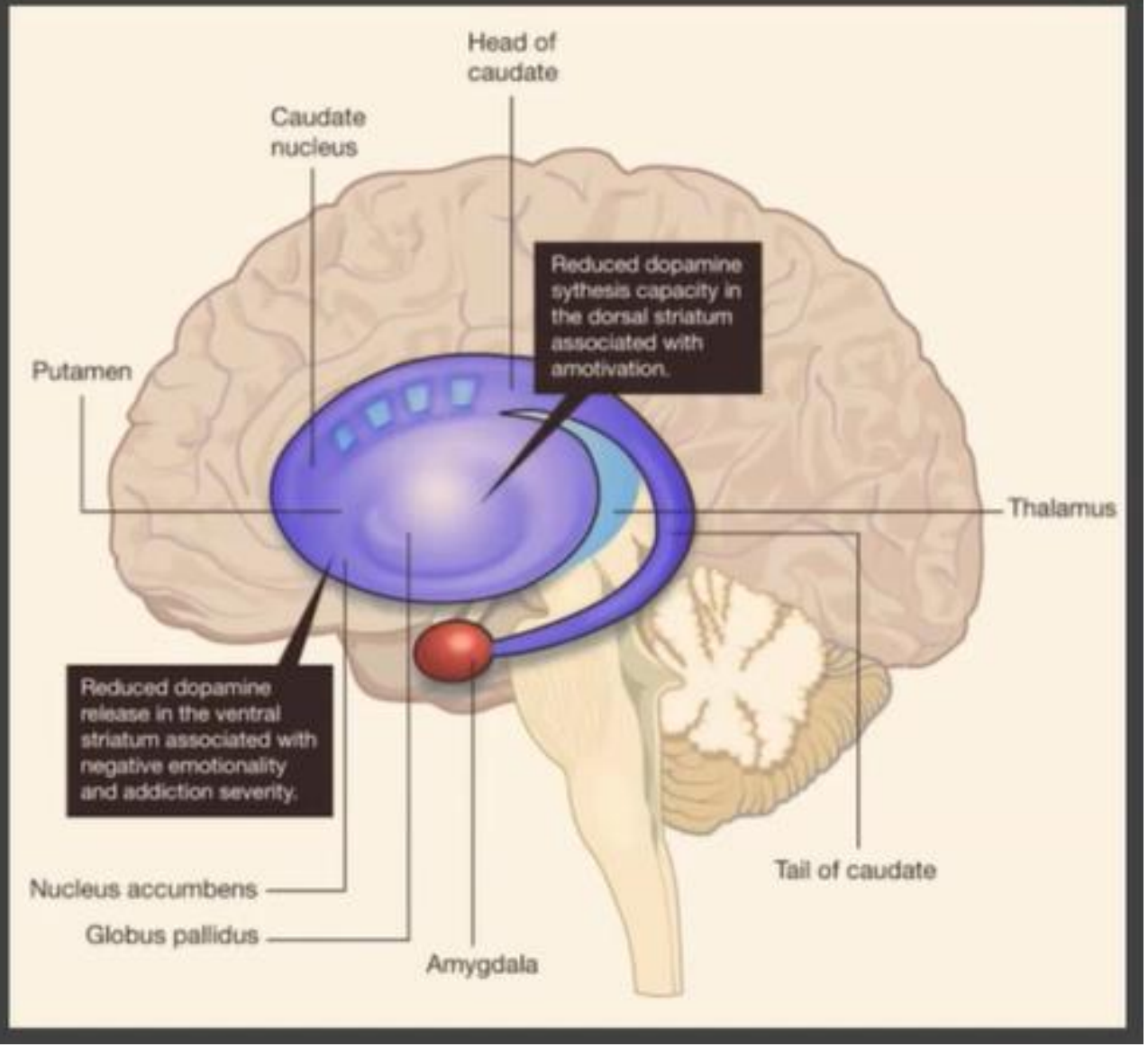


LONG- TERM EFFECTS

REGULAR USE OF CANNABIS OVER MANY YEARS LOWERS LEVELS OF DOPAMINE, A KEY CHEMICAL IN THE BRAIN'S REWARD CENTER.

"THE DOPAMINE SYSTEM IS CENTRAL TO LEARNING AND MOTIVATION. WE CONCLUDE THAT LONG-TERM CANNABIS USE IN PEOPLE IS LINKED TO BLUNTING OF THE DOPAMINE SYSTEM."

MICHAEL A. P. BLOOMFIELD ET AL. 2016. THE EFFECTS OF Δ9-TETRAHYDROCANNABINOL ON THE DOPAMINE SYSTEM. NATURE 539: 369-377; DOI: 10.1038/NATURE20153

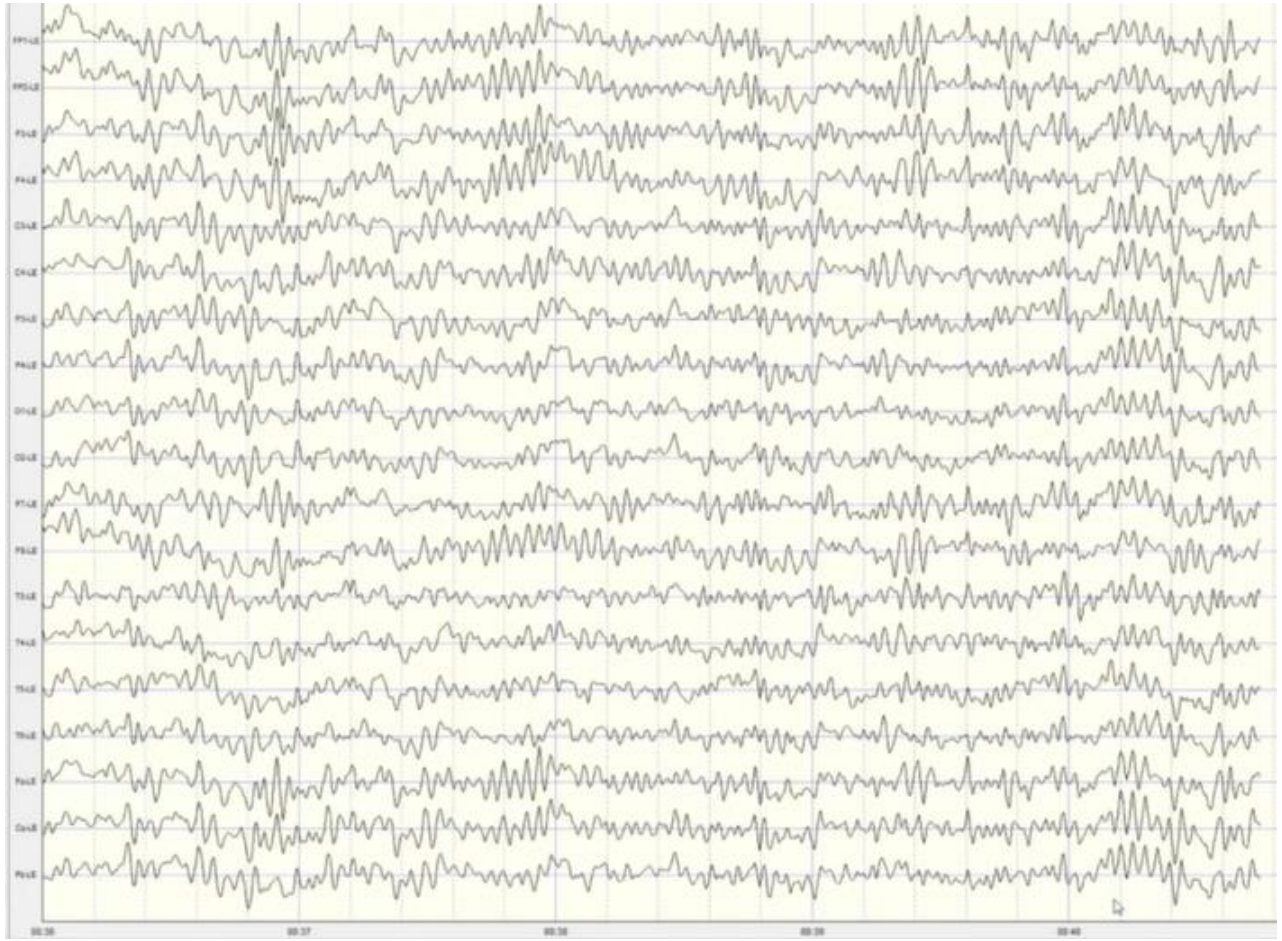


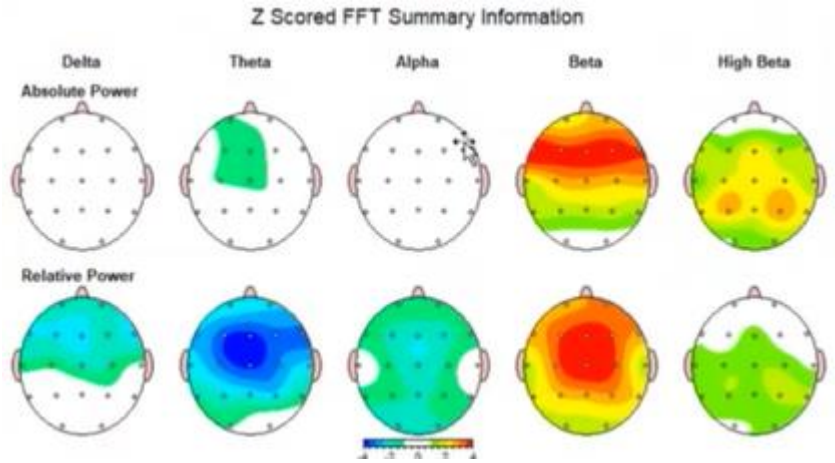
Benzodiazepine

Benzo Toxicity

- Benzodiazepines cause mild to severe depression of the nerves within the brain (CNS) and sedation (drowsiness).
- Benzodiazepines act on specific receptors in the brain, called gamma-aminobutyric acid-A (GABA-A) receptors. Benzos attach to these receptors and make the nerves in the brain less sensitive to stimulation, which has a calming effect.
- Benzodiazepines produce a spindling 18-25hz pattern which is sometimes more anteriorly pronounced. (How the drug will appear is based on dosing and systemic vascularity).

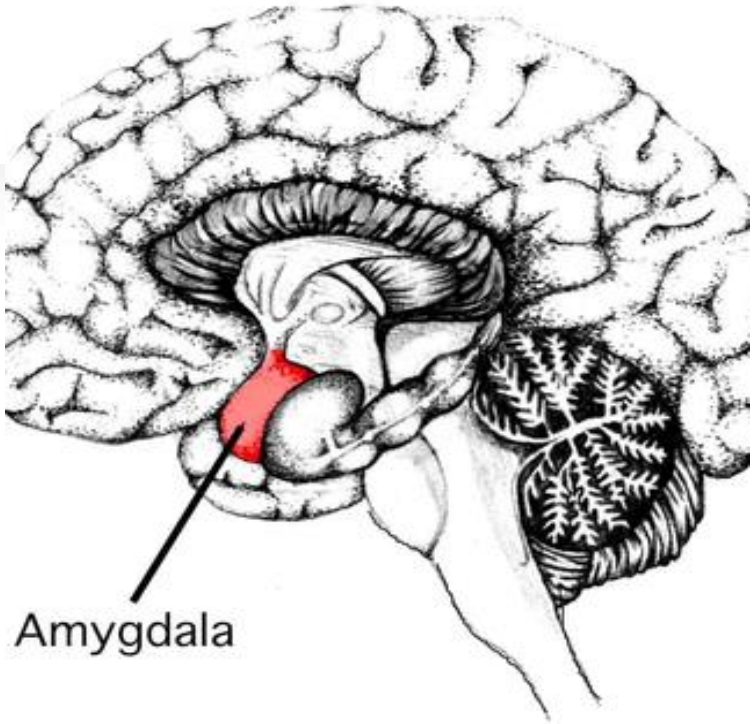
Beta Spindles





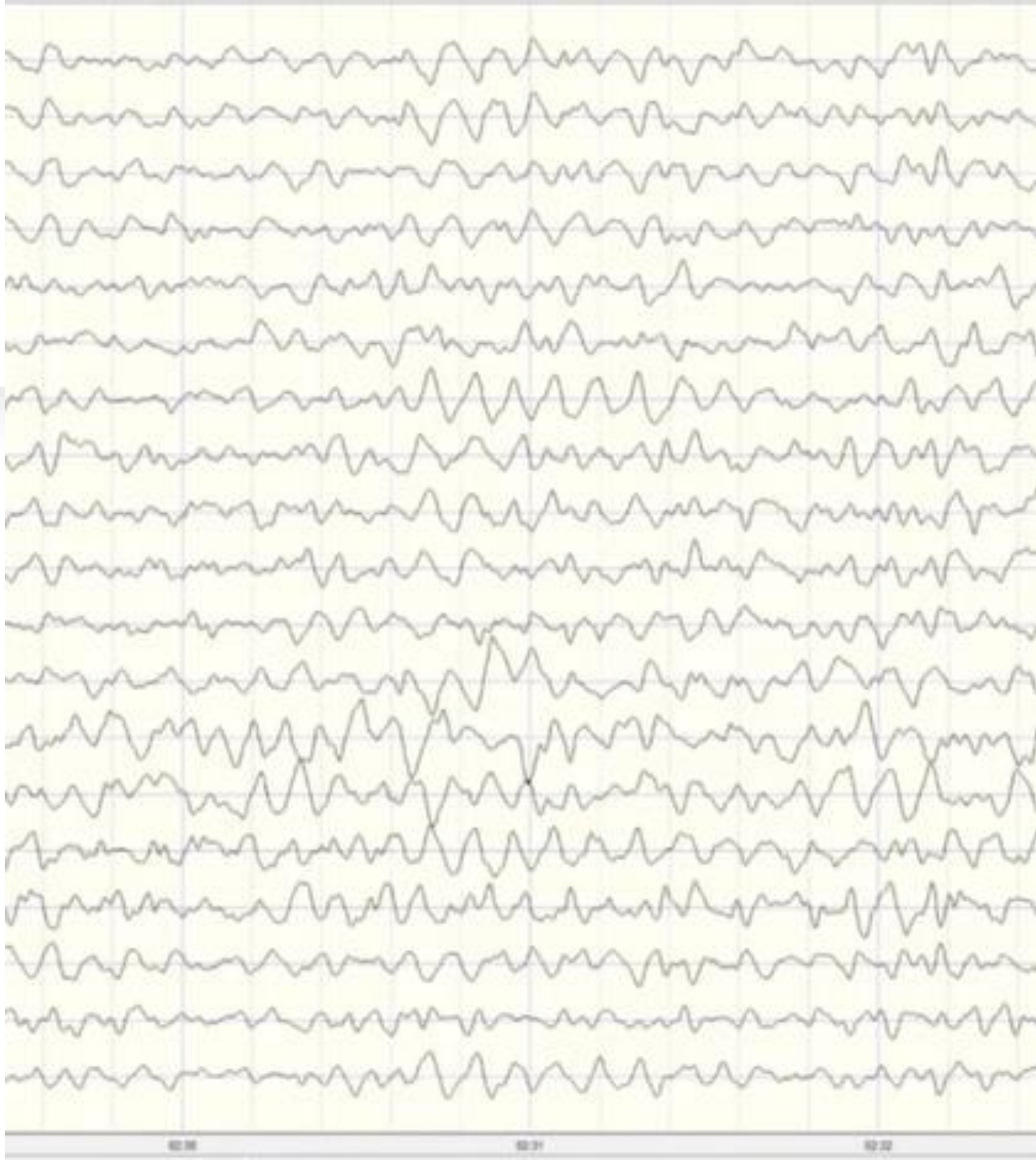
Opioids

- Prolonged daily use of opioids has been associated with a significant loss of brain cells in the amygdala.
- Opioid abuse can disrupt the balance between inhibitory and excitatory potentials in cortical pyramidal neurons affecting electrical synchronization.
- Amygdala controls fight or flight response



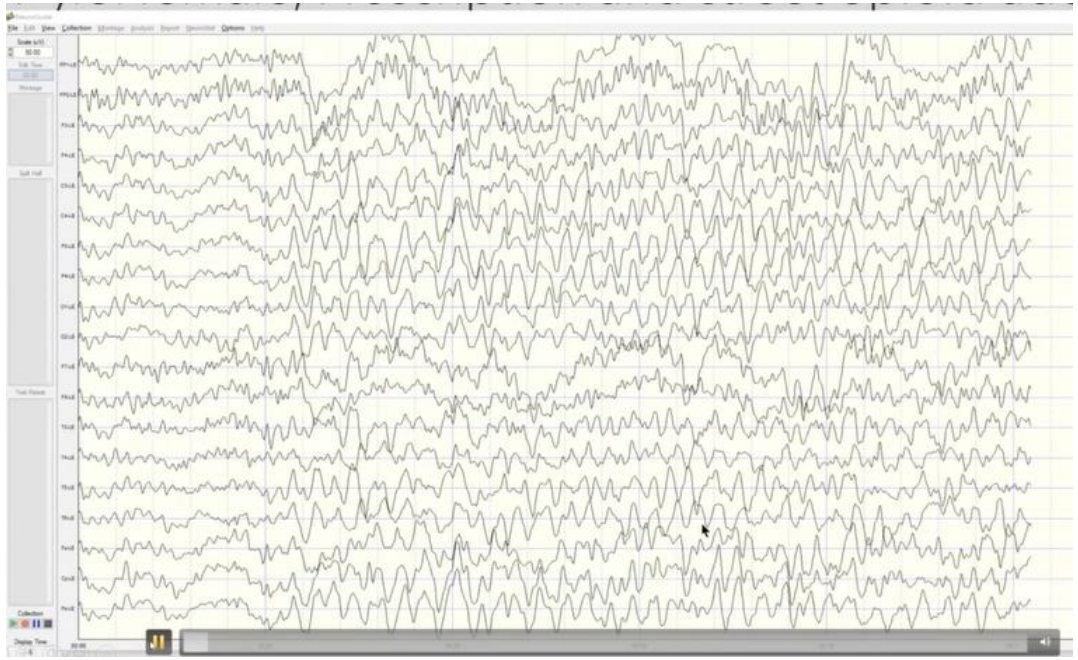
Opioids

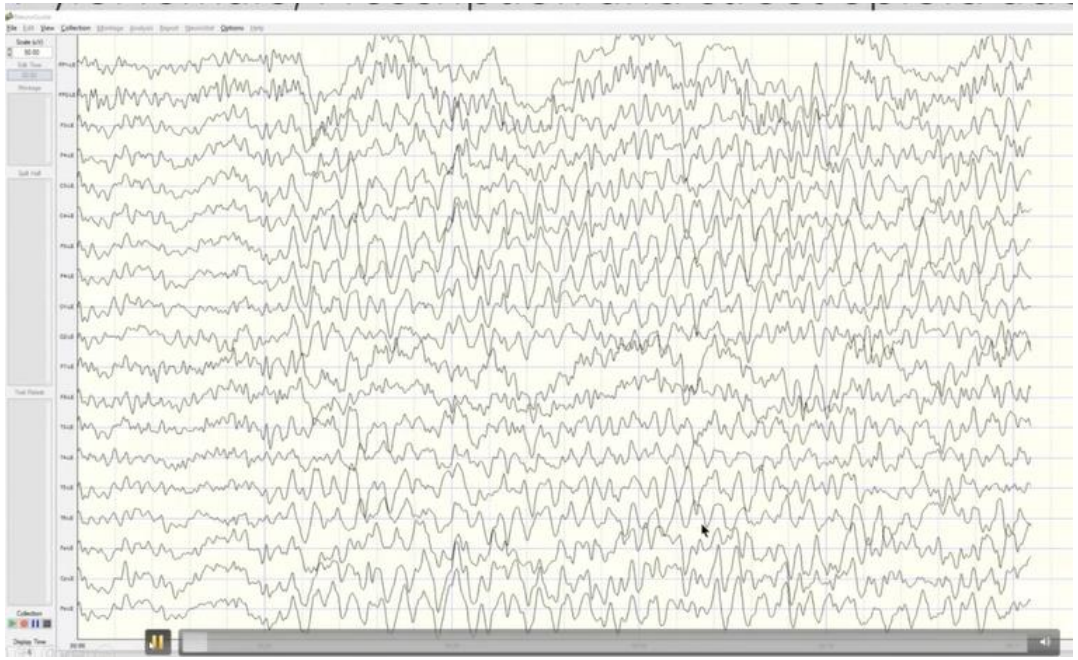
- Diffuse slowing- feeling “out of it”
- Memory compromise
- These effects result in long term damage to the brain



Case Study

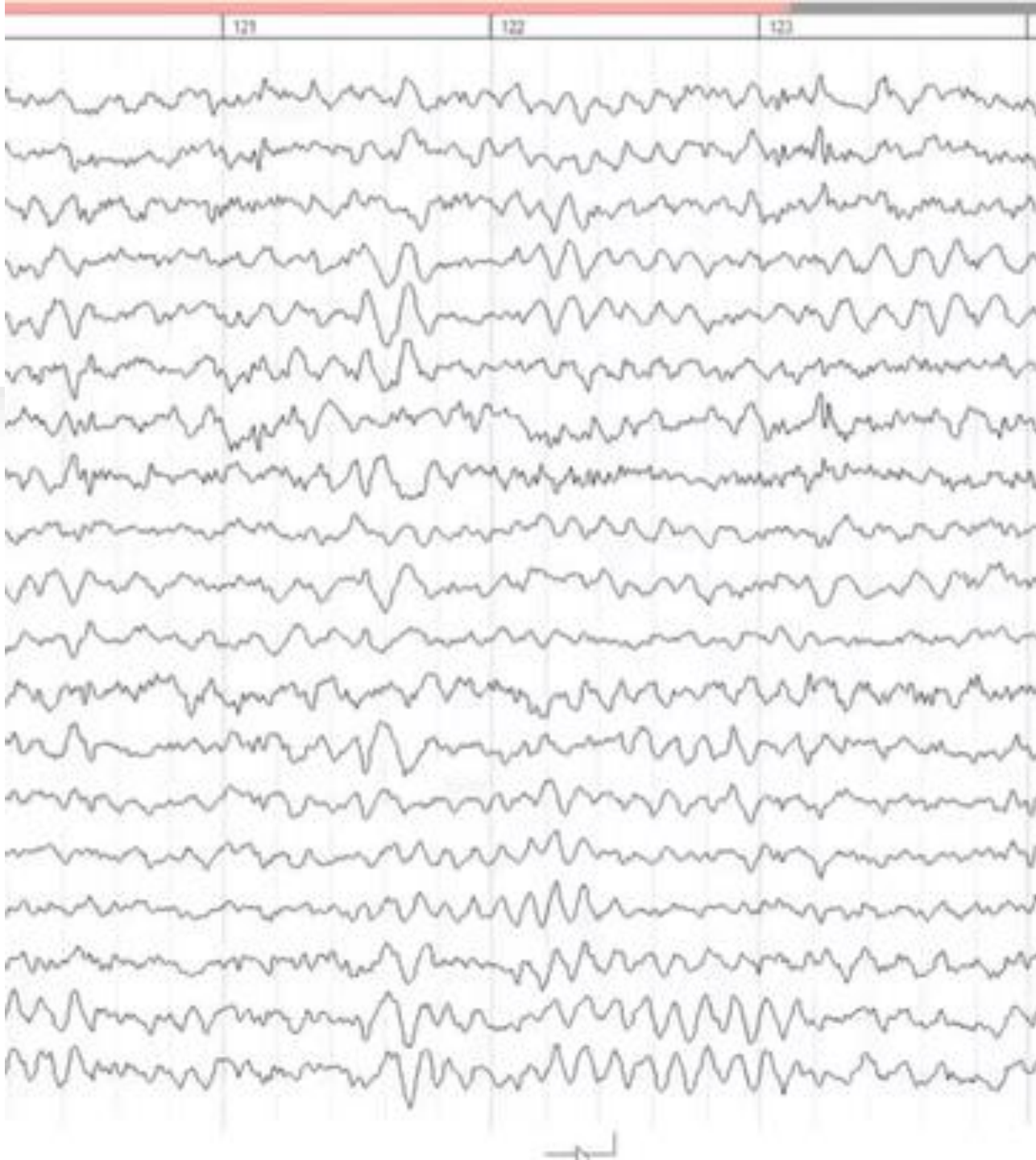
- 19 yr old female with a prescription and street opioid addiction
- Excessive delta slowing transitioning to low voltage followed by another large event
- Issues in vigilance



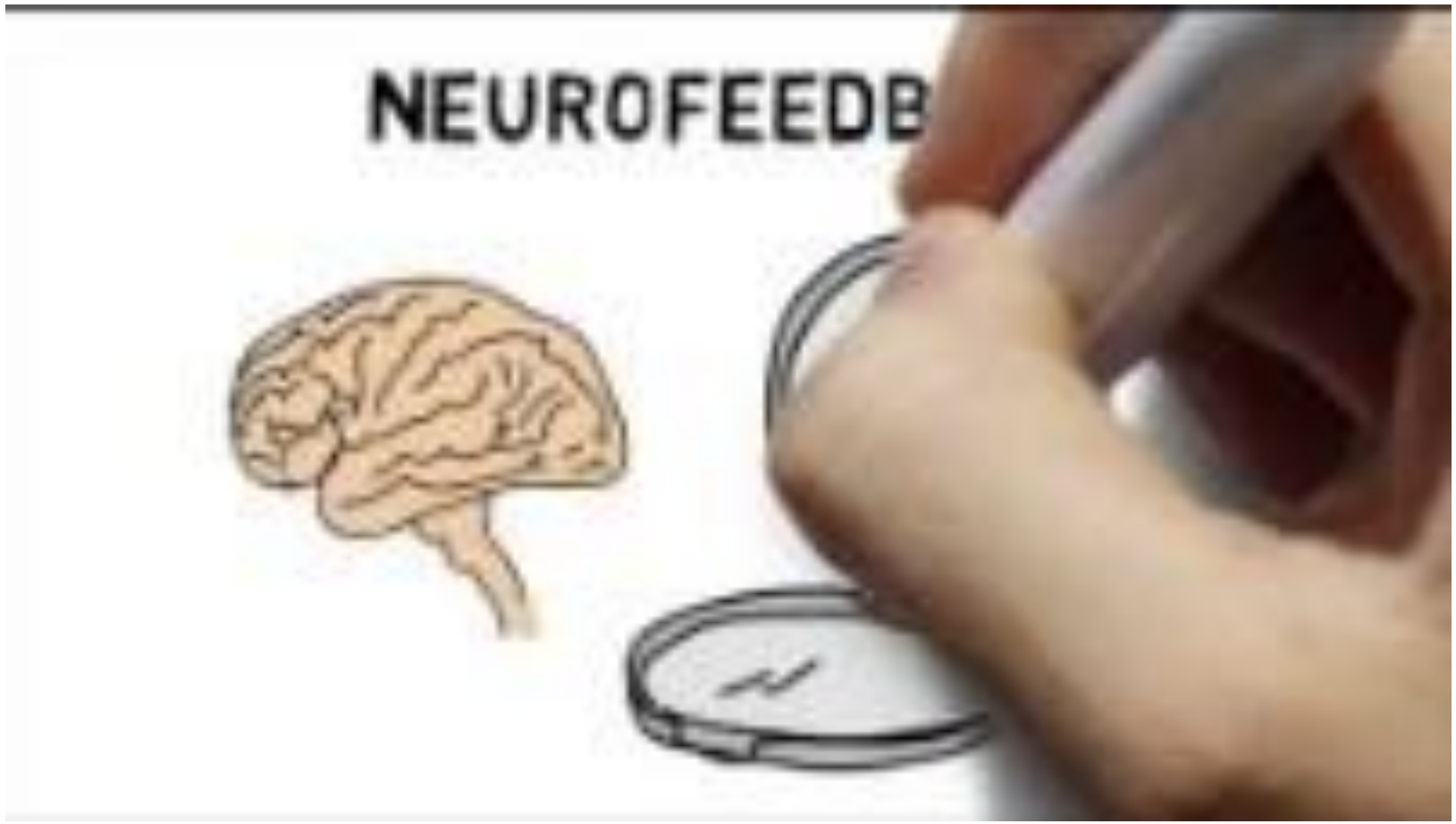


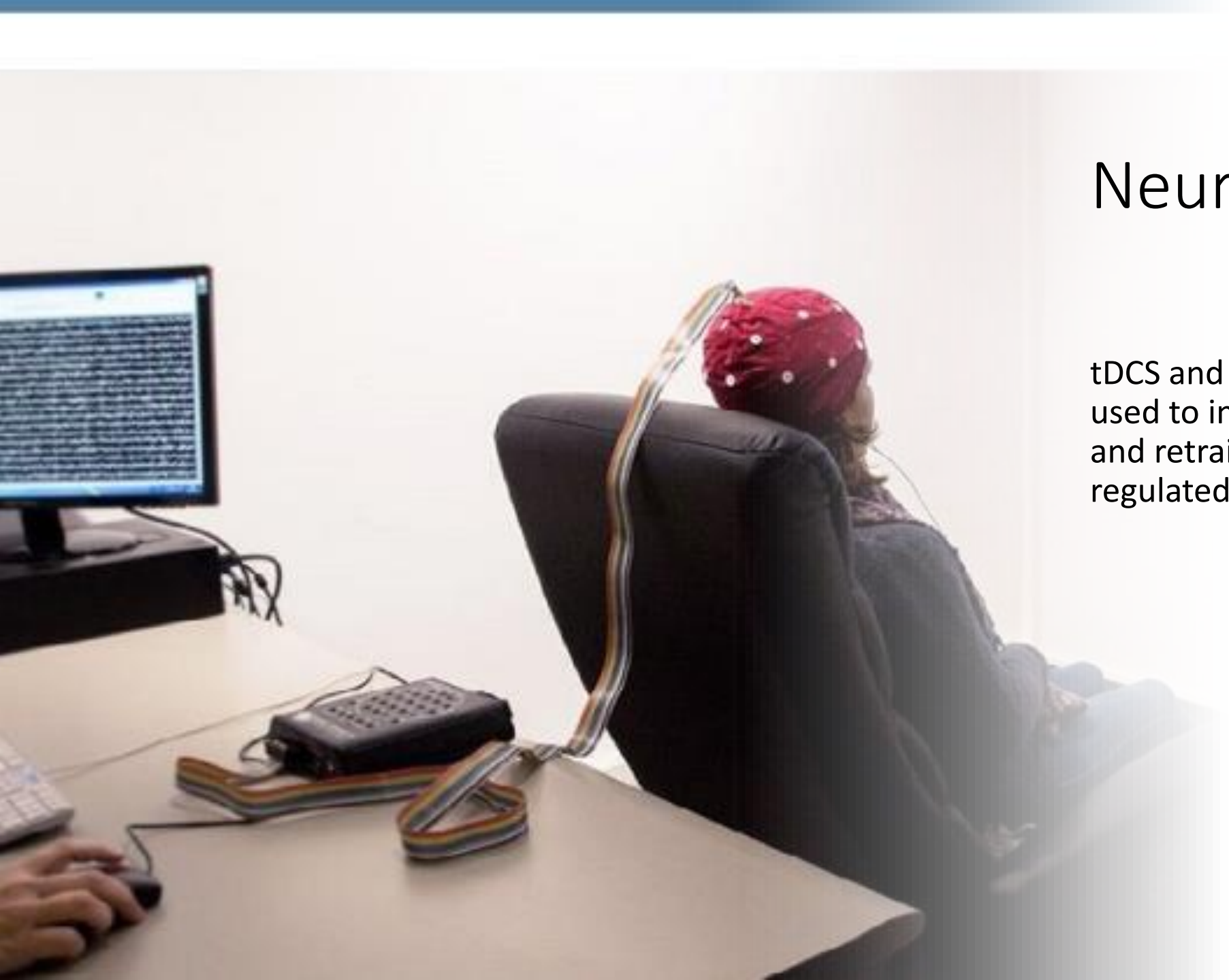
OTC Medications

- Dextromethorphan (DXM)
  - Delsym 12hr, Robitussin, Robafen Cough
  - Dissociative effects “living in another reality”
  - Creates a psychological rather than physical dependance



Treatment





Neurostimulation

tDCS and tACS along with noise are used to increase neuroplasticity and retrain the brain into more regulated rhythms