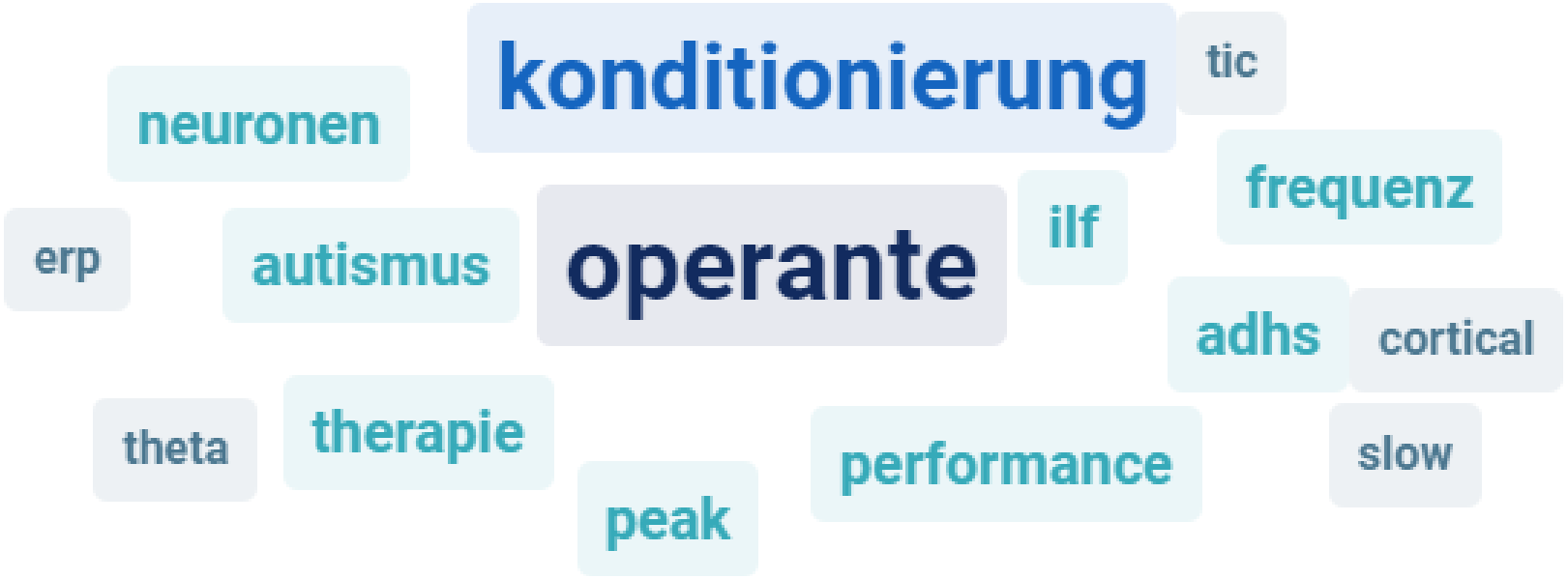
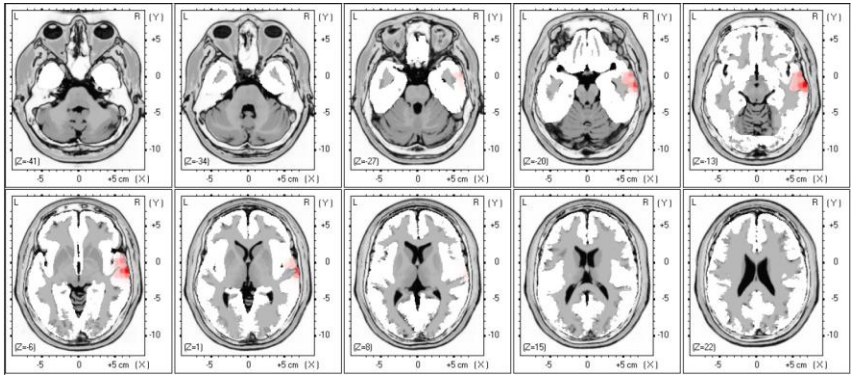
NEUROFEEDBACK UND QEEG BEI ZWANGSSTÖRUNGEN


konditionierung
tic
neuronen
frequenz
erp
autismus
operante
ilf
adhs
cortical
theta
therapie
performance
slow
peak

qEEG – Quantitative Elektroenzepkalographie



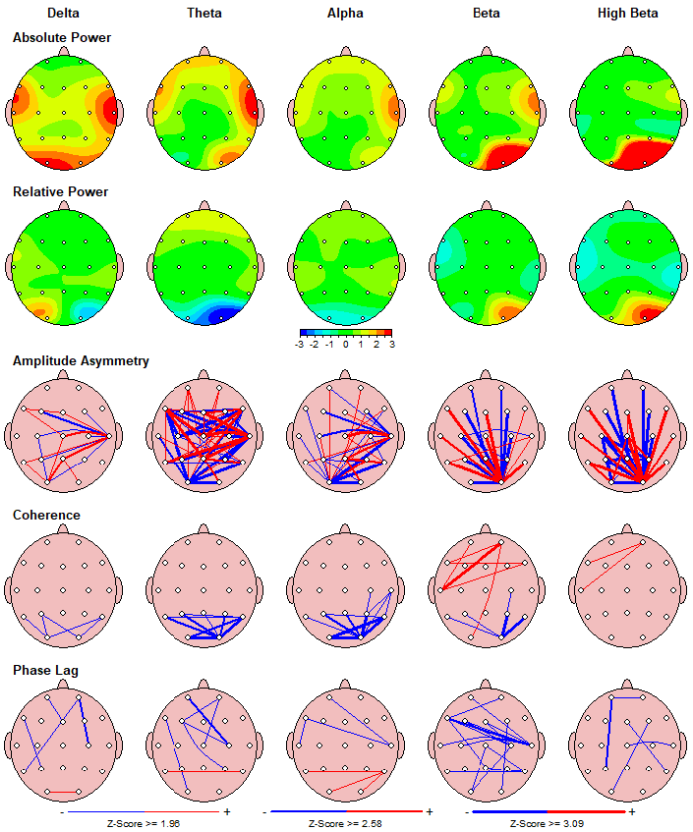
auch "Topometric Brain Mapping" genannt

die Daten werden statistisch-mathematischen Analysen unterzogen

die Messungen erfolgen digital

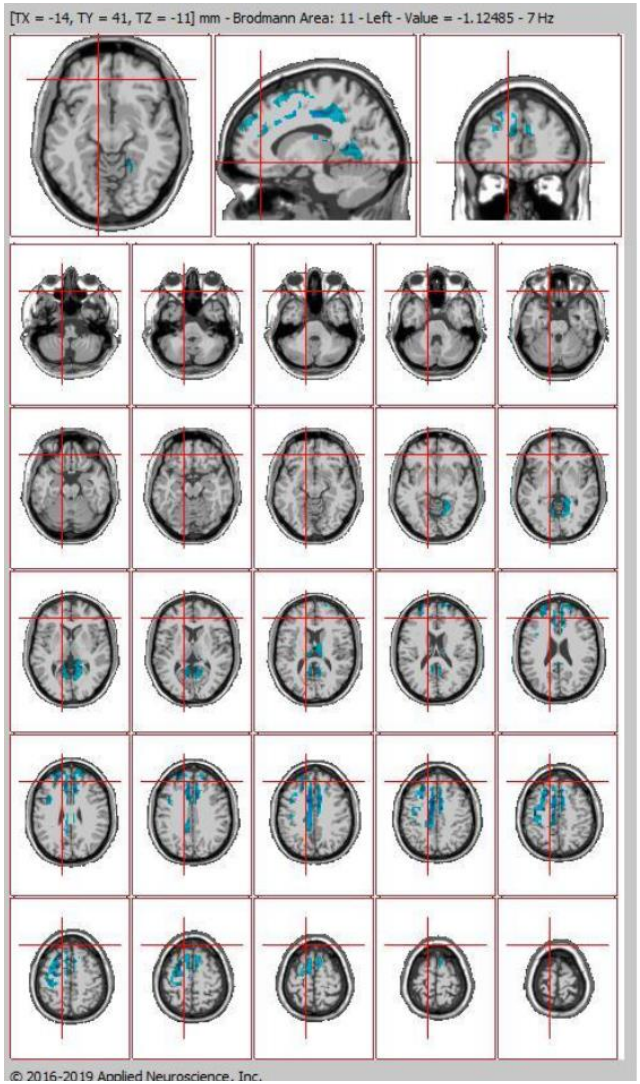
die Analysen ergeben unterschiedliche Parameter

qEEG Maße



Absolute Power und relative Power in allen typischen EEG-Frequenzbänder	Power Ist ein Maß für die Energie, kann berechnet werden für jedes einzelne Frequenzband im EEG
Kohärenz	Kohärenzmaße quantifizieren die Konnektivität neuronaler Schaltkreise, zeigen das Ausmaß der Kommunikation zwischen Arealen
Asymmetrie	zeigt die Verteilung der Power an der Schädeloberfläche
Dominante Frequenz	dadurch kann herausgefunden werden, welche Frequenz innerhalb eines Frequenzbandes oder des gesamten Frequenzspektrums die meiste Power hat

qEEG Maße und EEG-Tomographie

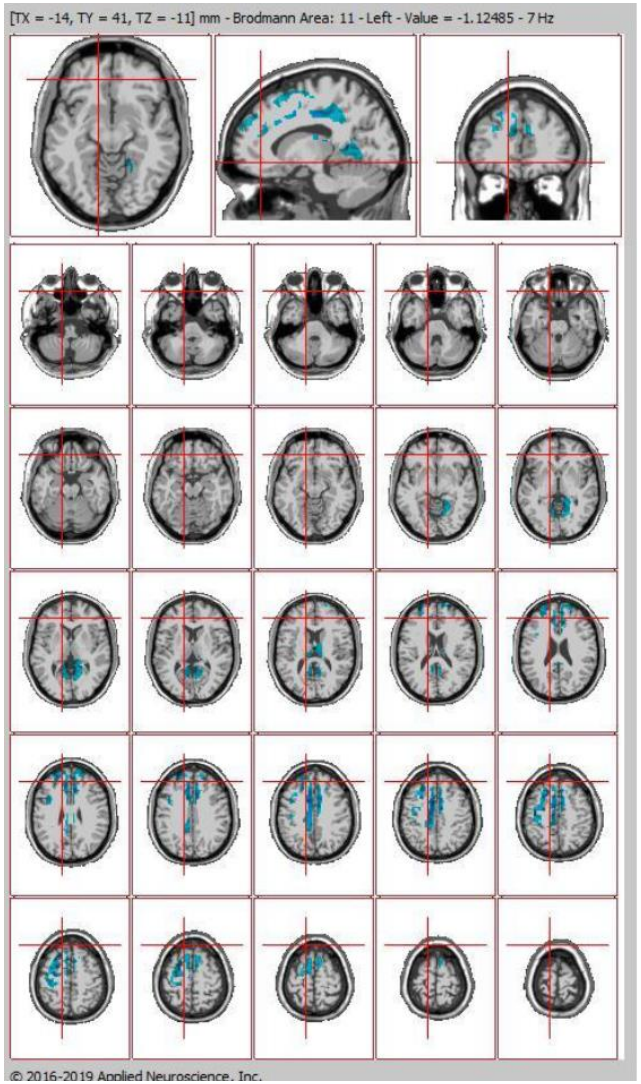


EEG Tomographie

- auch bekannt als tEEG, elekrisches Imaging (Michel et al, 2009) oder „Brain Elektromagnetic Tomography“ (BET) (Valdes-Sosa et al, 1994; Hernandez-Gonzales et al, 2011)
- basiert auf sogenannte „inverse Solutions“
- Loreta, sLoreta, eLoreta, swLoreta werden zur Zeit als „Inverse Solutions“ in der eeg - Forschung benützt
- zur Zeit 795 LORETA Publikationen
- http://www.uzh.ch/keyinst/NewLORETA/QuoteLORETA/PapersThatQuoteLORETA05.htm

EEG Tomographie

qEEG Maße und EEG-Tomographie



- swLoreta
- berechnet wird die Stromdichte

Stromdichte

Formel zur Berechnung

$$Stromdichte\ J = \frac{Elektrischer\ Strom\ I}{Leiterquerschnitt\ A} \qquad J = \frac{I}{A}$$

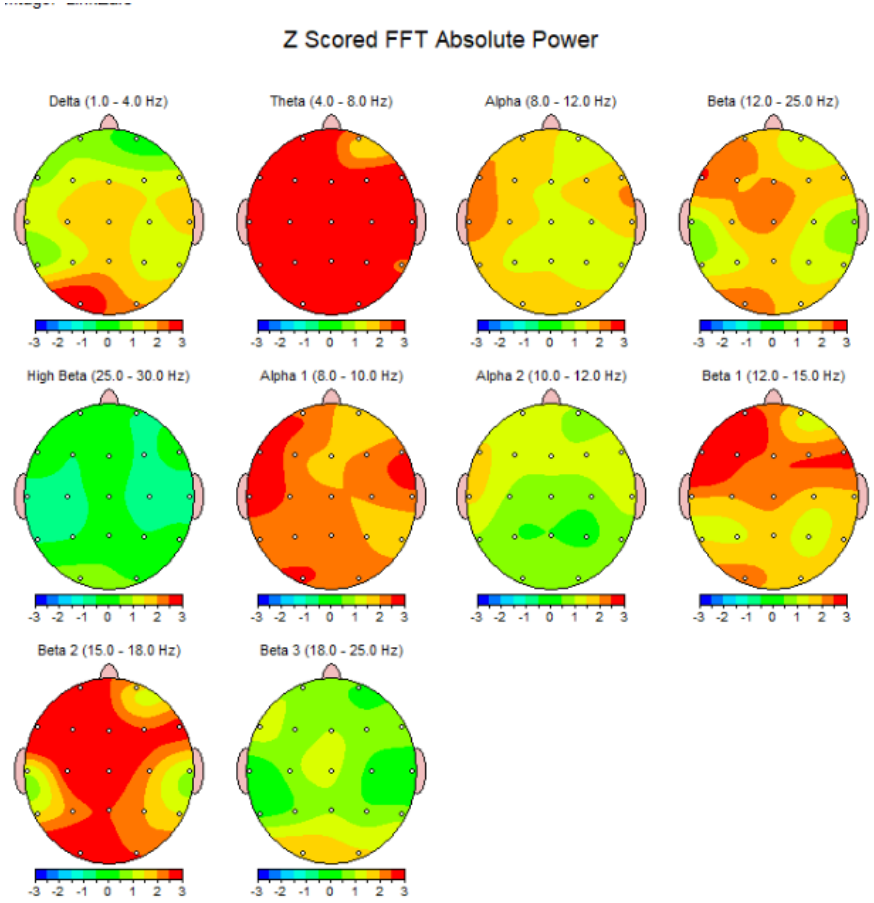
Warum qEEG vor NF-Therapiebeginn

- Störungen haben Heterogene Erscheinungsbilder (qEEG Maps, tEEG, fMRI. MRI)

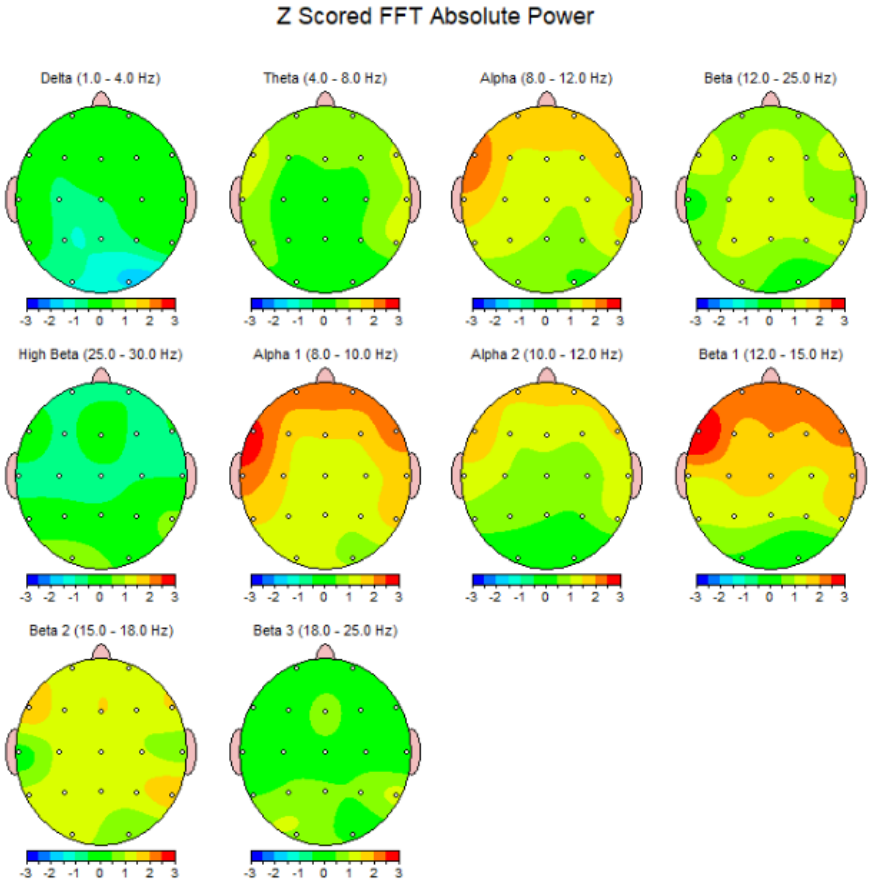
Fallvignette Herr Z.

- ein mittlerweile 31-järiger Mann mit psychiatrischer Vorgeschichte, seit 1999 wegen Zwangssymptomatik in Behandlung
- Zwangssymptomatik hat sich nach Erstmanifestation mehrfach verändert, er hat nahezu das ganze Spektrum von Zwangsgedanken und Handlungen erlebt
- Vor Beginn der NF Behandlung rufen fremde Gesichter Zwangsgedanken und Handlungen hervor
- Vor Beginn der NF Therapie 150 mg Sertralin und Aripiprazol 5 mg

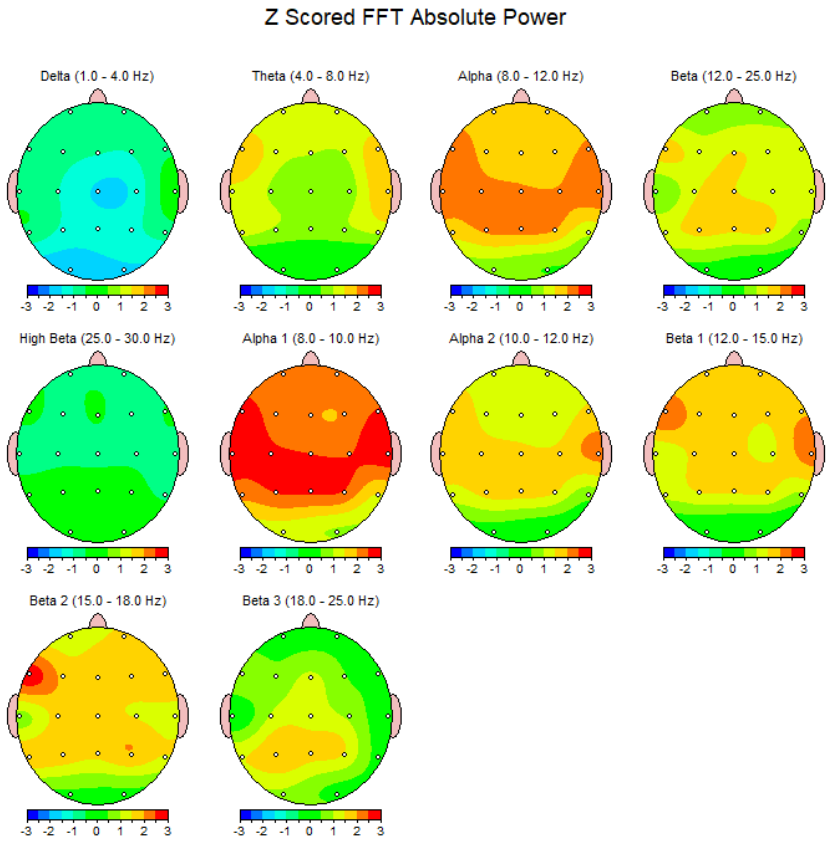
T1-vor Vor NF-Therapie

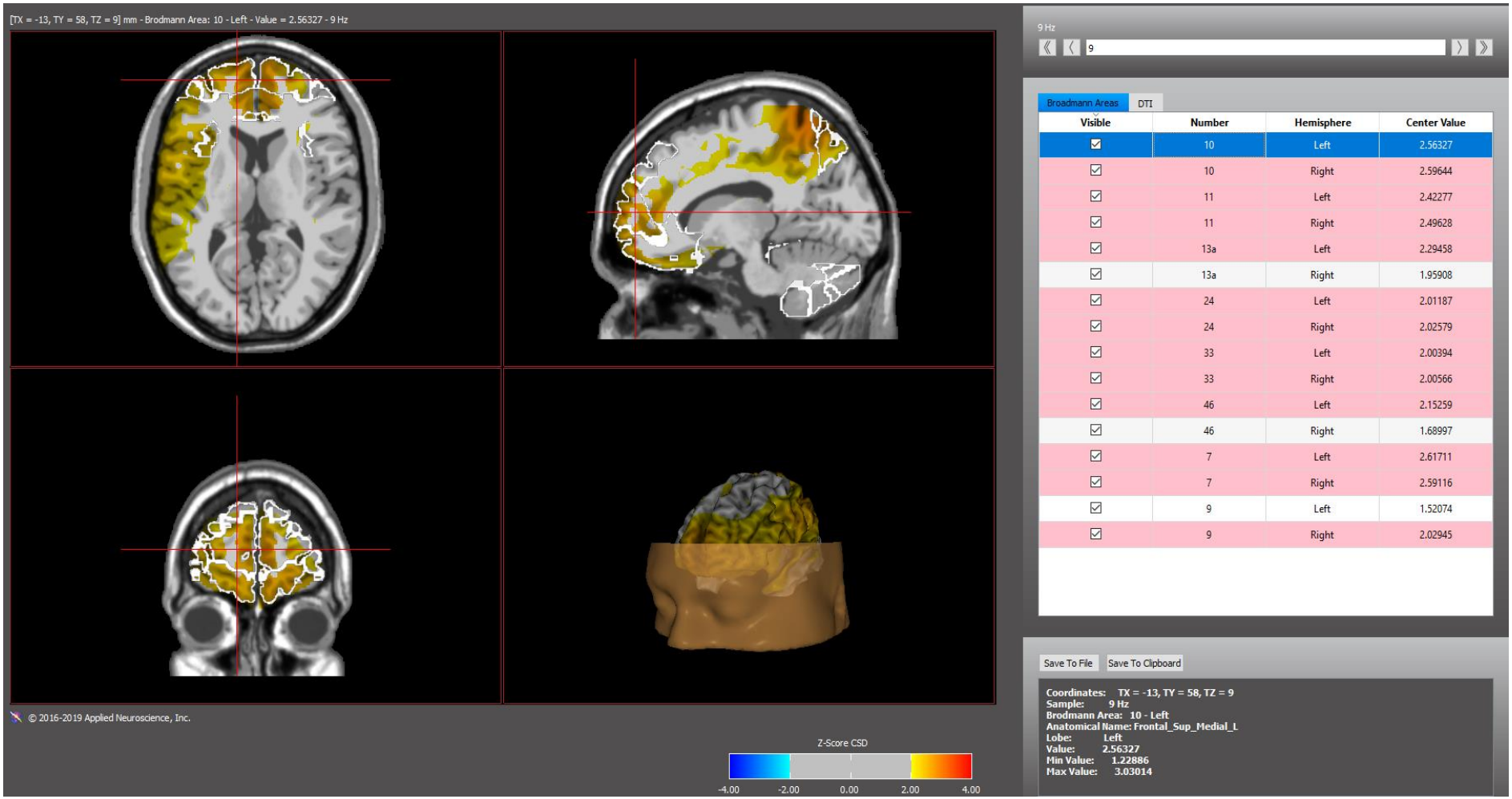


T2- nach 26 Sitzungen (Frequenzband)



T3-nach weiteren 30 NF-Sitzungen (Frequenzband) und Aripiprazol Absetzung





9 Hz

9

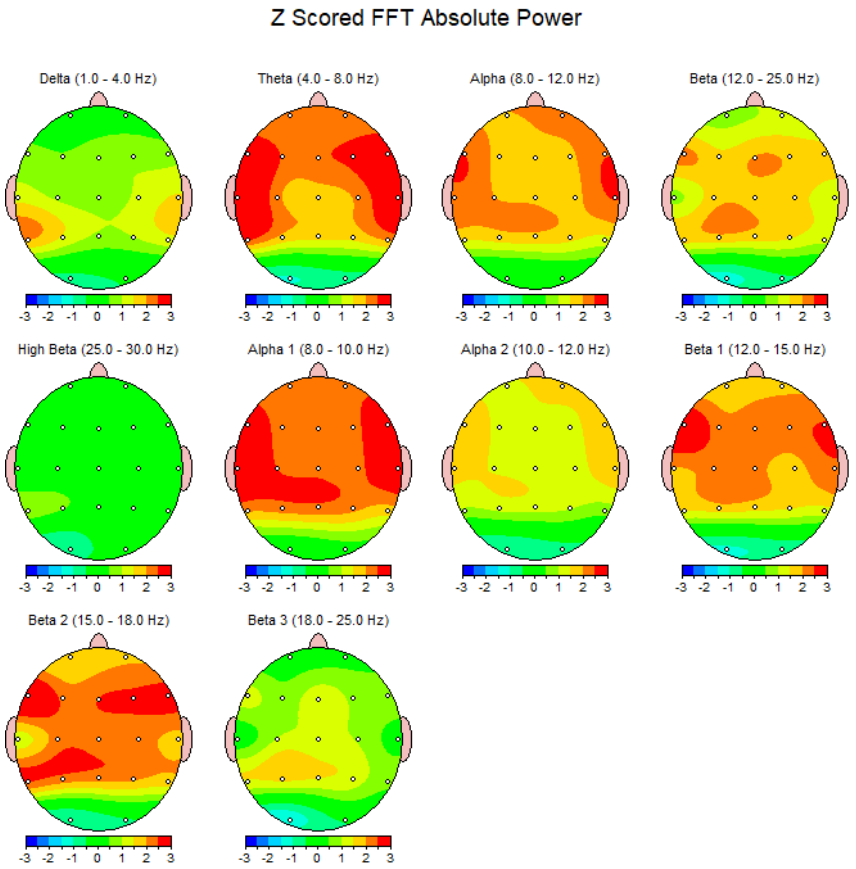
Broadmann Areas | DTI

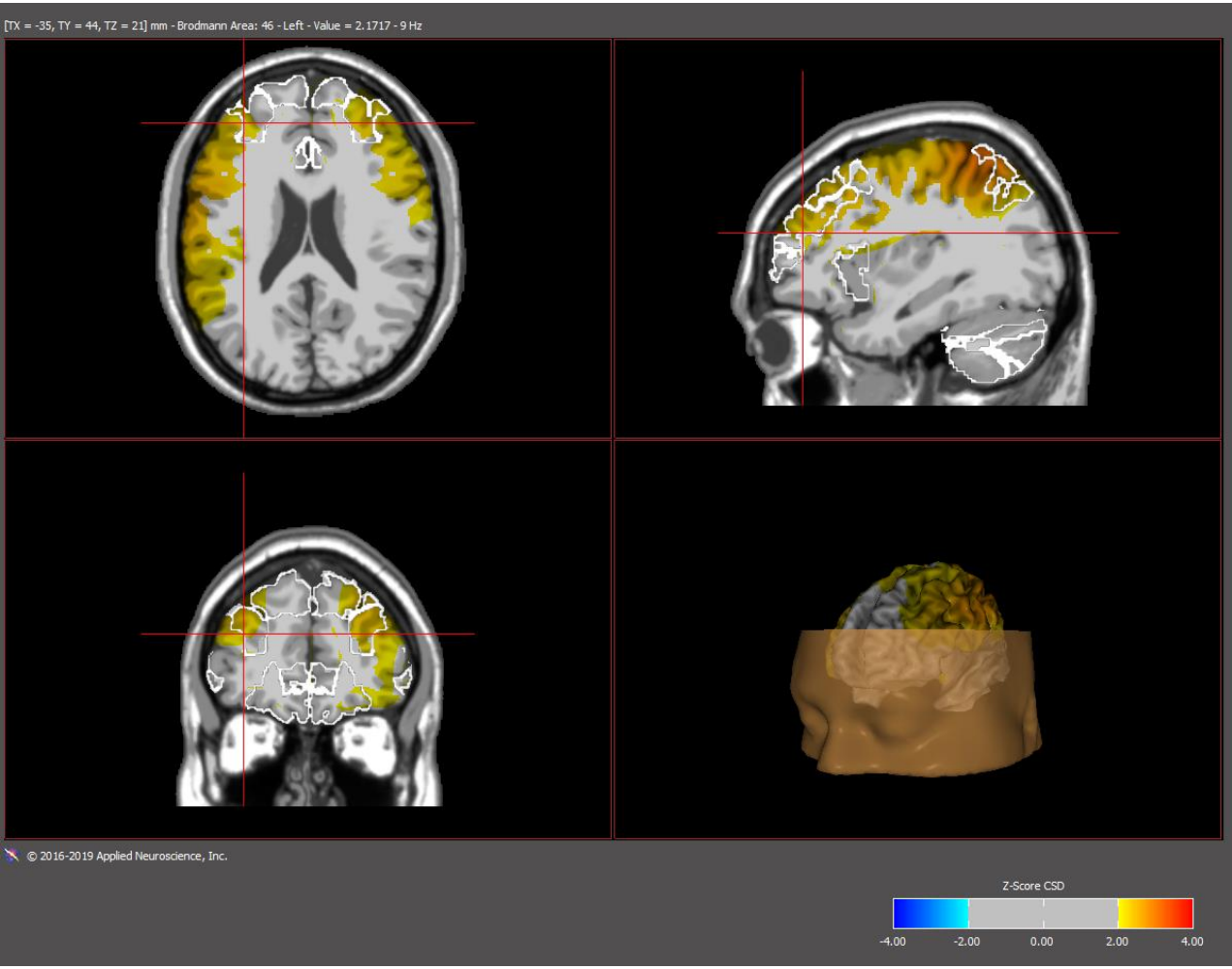
Visible	Number	Hemisphere	Center Value
☑	10	Left	2.56327
☑	10	Right	2.59644
☑	11	Left	2.42277
☑	11	Right	2.49628
☑	13a	Left	2.29458
☑	13a	Right	1.95908
☑	24	Left	2.01187
☑	24	Right	2.02579
☑	33	Left	2.00394
☑	33	Right	2.00566
☑	46	Left	2.15259
☑	46	Right	1.68997
☑	7	Left	2.61711
☑	7	Right	2.59116
☑	9	Left	1.52074
☑	9	Right	2.02945

Save To File | Save To Clipboard

Coordinates: TX = -13, TY = 58, TZ = 9
Sample: 9 Hz
Brodmann Area: 10 - Left
Anatomical Name: Frontal_Sup_Medial_L
Lobe: Left
Value: 2.56327
Min Value: 1.22886
Max Value: 3.03014

T4 – nach 30 sl-NF Sitzungen und mit nur noch 50mg Sertralin





9 Hz

9

Broadmann Areas | DTI

Visible	Number	Hemisphere	Center Value
☑	10	Left	1.69074
☑	10	Right	1.89246
☑	11	Left	1.79504
☑	11	Right	1.85675
☑	13a	Left	1.85919
☑	13a	Right	1.79185
☑	24	Left	1.81059
☑	24	Right	1.83898
☑	33	Left	1.81693
☑	33	Right	1.83647
☑	46	Left	2.1717
☑	46	Right	2.27822
☑	7	Left	2.5786
☑	7	Right	2.44015
☑	9	Left	2.09049
☑	9	Right	2.2879

Save To File | Save To Clipboard

Coordinates: TX = -35, TY = 44, TZ = 21
Sample: 9 Hz
Brodmann Area: 46 - Left
Anatomical Name: Frontal_Mid_L
Lobe: Left
Value: 2.1717
Min Value: 1.11116
Max Value: 3.12817

Vielen Dank für die Aufmerksamkeit!

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The Value of Quantitative Electroencephalography in Clinical Psychiatry: A Report by the Committee on Research of the American Neuropsychiatric Association

Kerry L. Coburn, Ph.D.
Edward C. Lauterbach, M.D.
Nash N. Boutros, M.D.
Kevin J. Black, M.D.
David B. Arciniegas, M.D.
C. Edward Coffey, M.D.

The authors evaulate quantitative electroencephalography EEG (qEEG) as a laboratory test in clinical psychiatry and describe specific techniques, including visual analysis, spectral analysis, univariate comparisons to normative healthy databases, multivariate comparisons to normative healthy and clinical databases, and advanced techniques that hold clinical promise. Controversial aspects of each technique are discussed, as are broader areas of criticism, such as commercial interests and standards of evidence. The published literature is selectively reviewed, and qEEG's applicability is assessed for disorders of childhood (learning and attentional disorders), dementia, mood disorders, anxiety, panic, obsessive-compulsive disorder, and schizophrenia. Emphasis is placed primarily on studies that use qEEG to aid in clinical diagnosis, and secondarily on studies that use qEEG to predict medication response or clinical course. Methodological problems are highlighted, the availability of large databases is discussed, and specific recommendations are made for further research and development. As a clinical laboratory test, qEEG's cautious use is recommended in attentional and learning disabilities of childhood, and in mood and dementing disorders of adulthood.

(The Journal of Neuropsychiatry and Clinical Neurosciences 2006; 18:460–500)

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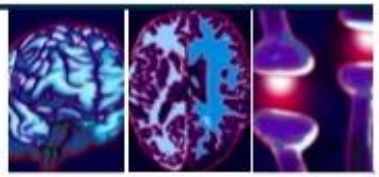
REVIEW



Neuropsychiatry and quantitative EEG in the 21st Century

Robert W Thatcher[1]

Practice points

- Use conventional clinical evaluation to derive a diagnosis and identify patient symptoms.
- Measure eyes open and eyes closed artifact-free quantitative EEG.
- Calculate auto- and cross-spectra to identify scalp locations and network deviations from normal.
- Use EEG tomography to link the patient's symptoms and complaints to functional systems in the brain.
- Identify and separate the 'weak' systems from compensatory systems.
- Use Z-score biofeedback to target the deregulated brain subsystems to reinforce optimal and homeostatic states of function while the clinician monitors the patient's symptom reduction.
- Use quantitative EEG to evaluate pre- versus post-treatment and follow-up evaluations to determine treatment efficacy (e.g., medications, repetitive transcranial magnetic stimulation, electroconvulsive therapy, brain–computer interfaces and biofeedback, among others).

SUMMARY The human brain weighs approximately 3 lbs and consumes 40–60% of blood glucose. This disproportionate amount of energy is used to create electricity in approximately 100 billion interconnected neurons. Quantitative EEG is a real-time movie of the electrical activity of the preconscious and conscious mind at frequencies of approximately 1–300 Hz. Numerous studies have cross-validated electrical neuroimaging by structural MRI, functional MRI and diffusion spectral imaging and thereby demonstrated how quantitative EEG can aid in linking a patient's symptoms and complaints to functional specialization in the brain. Electrical neuroimaging provides an inexpensive millisecond measure of functional modules, including the animation of structures through phase shift and phase lock. Today, neuropsychiatrists use these methods to link a patient's symptoms and complaints to functional specialization in the brain and use this information to implement treatment via brain–computer interfaces and neurofeedback technology.

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part of future medicine fsg

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comment

The golden age of computational psychiatry is within sight

Sophia Vinogradov

Clinically useful tools to identify the aberrant neural circuitry in individuals with psychiatric illness are lacking, as are treatments that do more than just address symptoms. Neuroplasticity-based treatments and computational neuroscience may hold some of the keys to unlocking the golden age of psychiatry.

There's a secret that we psychiatrists do not like to talk about: the abysmally primitive state of how we assess, understand, and treat mental illness.

A 19-year-old student — let's call him Tom — comes to my office. He has dropped out of his engineering programme because he believes an audio receiver has been implanted behind his ear by his professor, and he has begun to hear whispers telling him to set himself on fire.

Here is how I will evaluate Tom clinically, as we sit together in my office: "Tom, can you tell me if people have been singling you out in some way? Communicating with you against your will?" And a few minutes later: "Tell me about the frightening thoughts you have of harming yourself." These questions are the only 'lab test' I currently have at my disposal: the mental status examination, a structured way of observing and describing a patient's state of mind. Based on this examination and the patient's history (and assuming I have ruled out a medical disorder), I will make a diagnosis and an educated guess about Tom's prognosis, choosing a few descriptive labels such as 'schizophrenia and depression, with moderate risk of self-harm'.

There is no standardized brain imaging study I can use to understand the nature and severity of the impairments in Tom's brain. There are no easily available digitalized cognitive or socio-affective assessment measures that Tom can complete so we can understand how exactly his brain is failing to process critical information. There are no decision tools that allow me to use this kind of neuroscience-based information to make informed predictions about Tom's clinical trajectory. Even more sobering, my treatment offerings will rely heavily on just a few medications that can provide Tom with symptom control, but at the cost of weight gain, sexual dysfunction, and mental blunting. In other words, despite the astonishing neuroscience breakthroughs of recent decades, we still do not have assessment tools, a nosology, or a set of treatments that directly relate to the underlying brain system dysfunctions of mental illness and that I can use to help patients.

> Well-designed cognitive training approaches have real promise for harnessing adaptive plasticity in the brain.

The situation is about to change. I have spent the past 10 years investigating cognitive training and neuroplasticity in impaired neural system functioning in schizophrenia[1,2]; this work, along with exciting developments in neuroimaging, neuromodulation, and computational neuroscience, has radically altered how I and others think about the clinical practice of psychiatry and how I want to approach patients like Tom. The reification of the mental status examination as a diagnostic tool is the equivalent of prodding a swollen leg and making a diagnosis of oedema without trying to understand whether the swelling was caused by diabetes or by an insect bite. And while the use of medications for psychiatric symptom control is immensely helpful, drugs alone will not promote long-term psychosocial recovery nor correct, in a robust and enduring manner, the underlying distortions in brain representational systems that characterize mental illness. Well-designed cognitive training approaches have real promise for harnessing adaptive plasticity in the brain and improving neural system dysfunction in a number of mental illnesses, with positive real-world benefits[3]. This line of work, along with neuromodulation research and sophisticated multivariate analyses of behavioural, imaging, and electrophysiological data that seek to characterize the nature and predictive value of brain network function and dysfunction, has created several paradigm shifts that are inexorably leading to an entirely new way of approaching patients[4,5].

New approaches to assessment

Psychiatry has long been plagued by the fact that, until now, we did not know what the physiologic 'lesion' was that gave rise to troubling symptoms. Without knowledge of the lesion, we had no lab test to aid us in diagnosis and decision-making, let alone to inform us about treatment development. Interestingly, the emerging picture in psychiatry is that there is no lesion, there is only a computational probability. The brain processes information through its neural circuits — a cellular wiring diagram for the multiple levels of information processing steps (computations) that ultimately give rise to our thoughts, feelings, and behaviour.

As an analogy, consider what must happen to create a symphony: each musician must play specific notes on a specific instrument, and each section of musicians must interact harmoniously, and every section must integrate together under the direction of the conductor to create the final symphonic experience. Every component of a symphony performance (instruments, musicians, sections, conductor and orchestra hall) can and does show interesting and important variations around a modal output (a probability distribution) that ultimately result in a range of outcomes. Beethoven's Symphony No. 3 as played by the New York Philharmonic is different when played by the San Francisco Symphony — but in both cases is still recognizably the Eroica.

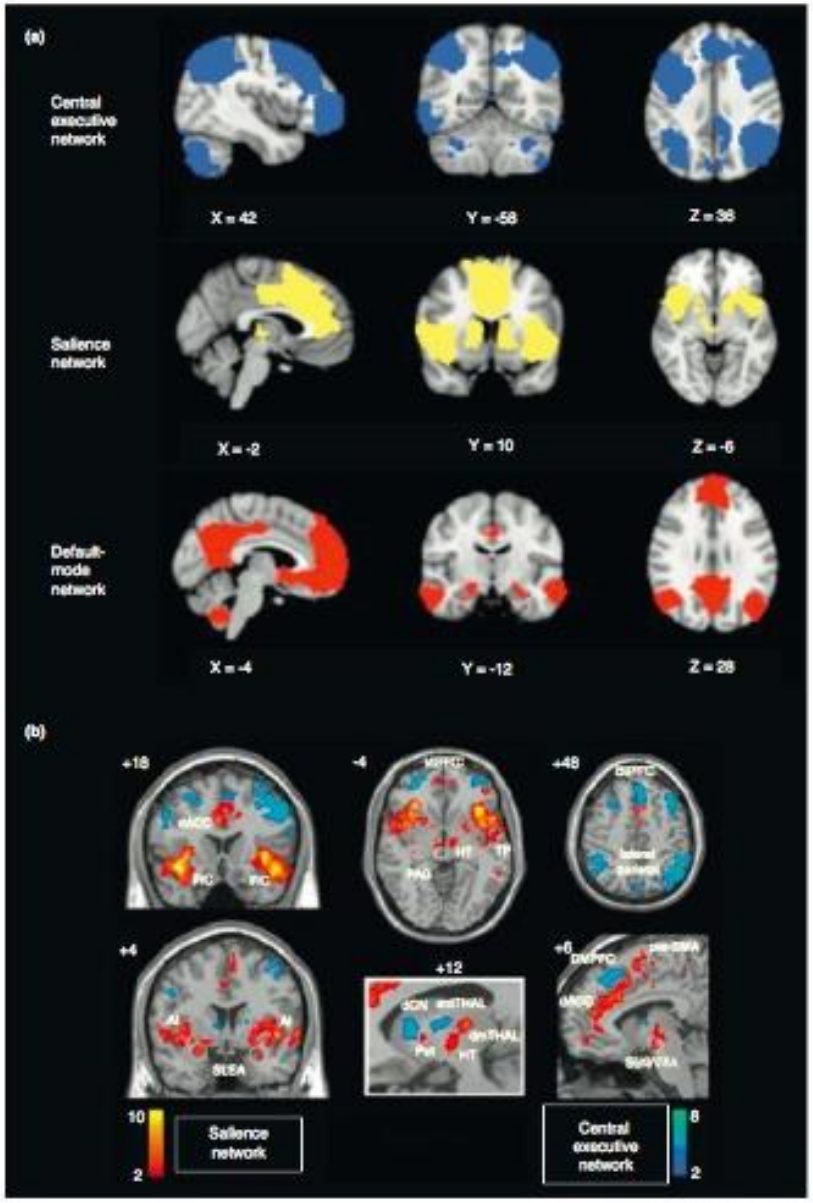
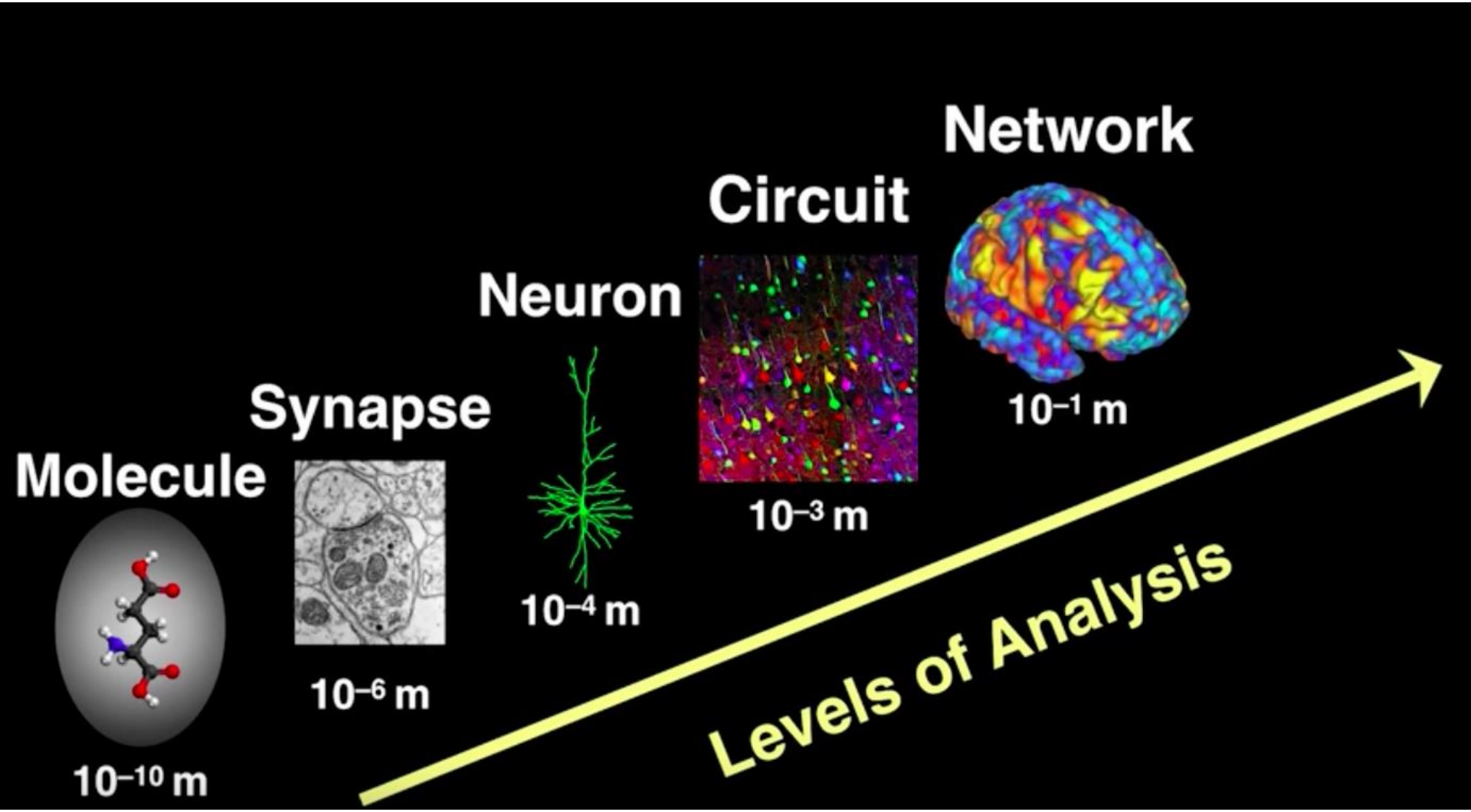
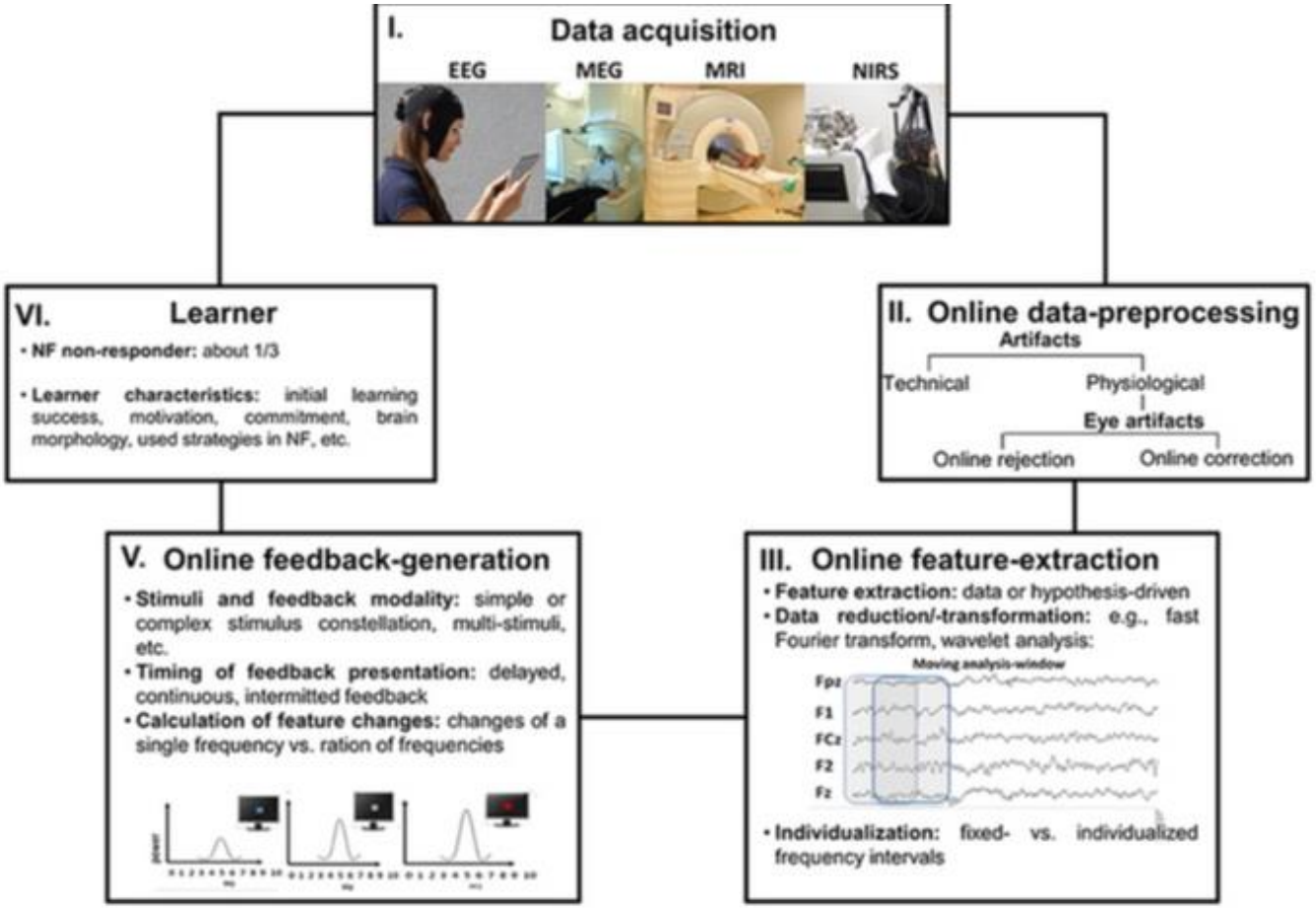


Figure 7. Three core neurocognitive networks. (a) The CEN, SN and DMN. The frontoparietal CEN (shown in blue), anchored in the dlPFC and the PPC, plays an important role in working memory and attention. The SN, shown in yellow, is important for detection and mapping of salient external inputs and internal brain events. The SN is anchored in the PIC and dorsal dACC and features extensive connectivity with subcortical and limbic structures involved in reward and motivation. The DMN (shown in red), anchored in the PCC and medial PFC, is important for self-referential mental activity. Adapted from [27,28,36] (b) The CEN and SN are both coactivated during a wide range of cognitive tasks but have distinct patterns of intrinsic cortical connectivity in the dorsomedial prefrontal cortex (DMPFC) dACC, dlPFC, vlPFC and lateral parietal cortex and subcortical connectivity in the anterior thalamus (anTHAL), dorsal caudate nucleus (dCN), dorsomedial thalamus (dmTHAL), hypothalamus (HT), periaqueductal gray (PAG), putamen (Put), sublenticular extended amygdala (SLEA), SuN/VTA and the temporal pole (TP). Adapted from [27].



Quelle: https://www.youtube.com/watch?v=8G83r_SUDXE

Neurofeedback als Methode zur Verhaltensänderung?



Quelle: Enriquez-Geppert et al.; http://journal.frontiersin.org/article/10.3389/fnhum.2017.00051 doi: 10.3389/fnhum.2017.00051