EEG BASED PHENOTYPES: TRANSLATING RESEARCH METHODOLOGIES TO ACHIEVE PERSONALIZED INTERVENTIONS

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- Introduction to EEG Phenotypes.
- Phenotypes and DSM Grouping.
- Difference between EEG Phenotypes and Subtypes.
- Recognized Types of EEG Phenotypes.
- Neurofeedback Therapy based on EEG Phenotypes.
- EEG Phenotypes in Autism.
- Autism and Epilepsy.
- Connectivity and Coherence as an EEG subtype in Autism.
- References.
INTRODUCTION TO EEG PHENOTYPES

- Prior studies using statistical analysis of electroencephalography (EEG) have documented clusters of EEG/quantitative EEG (QEEG) features within psychiatric populations (John, Prichep, & Almas, 1992). Experience over the past 30-plus years with a large number of clinical EEGs and, more recently, decades worth of clinical and research experience with QEEG, as well as a review of the field’s literature, have shown that a limited set of EEG patterns can characterize the majority of EEG variance.

- The phenotypical approach acknowledges the overlap of symptoms in many psychological disorders, and, as opposed to DSM-IV labels, does not require a diagnosis as a precondition for effective treatment. Phenotypes provide classification of the abnormal EEG record only and any EEG record that does not contain the traits of a specific phenotype is often classified as “normal.”

- Phenotypes can be described as a bridge between genetics and behaviour.

EEG pattern are proposed to reflect semistable states of neurophysiological function EEG phenotypes. EEG phenotypes are clusters of commonly occurring EEG patterns found in the general population that are believed to be the result of underlying genetics. These phenotypes are purported to play an intermediate role between genetics and behavior (Gunkelman, 2006).

- Research and clinical experience in the field have shown that a limited set of EEG patterns can characterize the majority of EEG variance. This means that similar EEG patterns recur in persons with different psychological disorders.
EEG PHENOTYPES AND DSM GROUPING

- One phenotype may be seen in a wide variety of DSM groupings and this implicate a lack of specific overlapping with the DSM grouping.

- For example, theta-beta ratio abnormal values are supposed to be distinctive and sensitive to attention deficit hyperactivity disorder (ADHD), but similar increase in this ratio also may be seen in a wide variety of other clinical conditions as well as in the absence of ADHD.

PHENOTYPES HAVE POWERFUL IMPLICATIONS FOR BOTH MEDICATION, AND NEUROFEEDBACK.


DIFFERENCES BETWEEN PHENOTYPES AND SUBTYPES

- The DSM does not yield optimal treatment efficacy following diagnosis.

- Diagnoses have multiple “EEG subtypes”, but they ARE NOT SPECIFIC TO THE DIAGNOSIS:
  - Frontal alpha in ADD
  - Frontal alpha in depression
  - Frontal alpha in early dementia
  - Frontal alpha in anxiety
  - Frontal alpha in OCD
11 RECOGNIZED TYPES OF EEG PHENOTYPES

Diffuse slow activity, with or without low frequency alpha.
- Increased delta and theta (1-7 Hz) with or without slow posterior dominant rhythm. Stimulant inhibit midline frontocentral activity below 10 Hz, add reward anterior beta frequencies for increased effect.

Focal abnormalities, not epileptiform.
- Focal slow activity or focal lack of activity.
- Inhibit slow activity (<10 Hz) and reward higher frequencies (>12 Hz).

Mixed fast and slow
- Increased activity below 8 Hz, lack of alpha, increased beta frequency activity. Combine across classes, e.g. stimulant + anticonvulsant.
- Inhibit slow frequencies, reward SMR.

Frontally dominant excess theta or alpha frequency activity
- Antidepressant, stimulant.
- Inhibit midline frontocentral activity below 10 Hz, add reward anterior beta frequencies for increased effect.

Frontal asymmetries
- Variable asymmetry L>R or R>L, primarily at F3, F4.
- Antidepressant
- Reward F3 beta, inhibit F3 theta and alpha frequencies.

Excess temporal lobe alpha
- Increased alpha activity generated in temporal lobe
- Stimulant
- Inhibit 9-12 Hz activity over affected temporal region(s), + inhibit frontal slow activity.

Epileptiform
- Transient spike/wave, sharp waves, paroxysmal EEG
- Anticonvulsant medication
- Inhibit low and high frequencies over affected regions, reward SCP and/or SMR.

Generally low magnitudes (fast or slow)
- Metabolic support (LVS), nutraceuticals
- Reward alpha activity posteriorly. (Penniston protocol for LVF)

Faster alpha variants, not low voltage
- Alpha frequency greater than 12 Hz over posterior cortex.
- GABA related medication (slightly slows the EEG frequencies)
Reward 9-10Hz alpha at Pz, shift alpha frequency lower with alpha/theta protocol.

Spindling excessive beta
- High frequency beta with a spindle morphology, often with an anterior emphasis.
- Anticonvulsants
- Inhibit beta frequencies, wide band inhibit, possibly Penniston if alpha levels are depressed.

Persistent alpha with eyes open
- Lack of appreciable alpha blocking with eye opening, generally this is slower alpha.
- SNRI or amphetamine.
- Reward beta frequencies, inhibit alpha. Reward higher frequency alpha.
## NEUROFEEDBACK THERAPY BASED ON EEG PHENOTYPES

<table>
<thead>
<tr>
<th>Candidate Phenotype</th>
<th>EEG Findings</th>
<th>Associated Neurofeedback Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low-voltage fast</td>
<td>Low-voltage EEG overall</td>
<td>Reward alpha activity posteriorly</td>
</tr>
<tr>
<td>Epileptiform</td>
<td>Transient spike/wave, sharp waves, paroxysmal EEG</td>
<td>Inhibit low and high frequencies; sensorimotor rhythm training; also consider slow cortical potential control</td>
</tr>
<tr>
<td>Diffuse slow activity (with or without lower alpha)</td>
<td>Increased delta and theta (1-7 Hz) with or without slower posterior alpha</td>
<td>Inhibit midline fronto-central activity slower than 10 Hz, add reward for anterior beta for increased stimulating effect</td>
</tr>
<tr>
<td>Focal abnormalities (not epileptiform)</td>
<td>Focal slow activity or focal lack of activity</td>
<td>Inhibit slower activity and reward higher frequencies (consider medical referral)</td>
</tr>
<tr>
<td>Mixed fast and slow</td>
<td>Increased slower activity, lack of organized alpha, increased beta</td>
<td>Inhibit slow frequencies, reward alpha and SMR, inhibit faster beta</td>
</tr>
<tr>
<td>Frontal lobe hypoperfusion disturbances</td>
<td>Frontally dominant excess theta or alpha frequency activity</td>
<td>Inhibit midline fronto-central activity below 10Hz, reward anterior beta for increased effect</td>
</tr>
<tr>
<td>Frontal asymmetries</td>
<td>Frontal asymmetry primarily measured at F3, F4</td>
<td>Adjust frontal symmetry with alpha, theta, and beta</td>
</tr>
<tr>
<td>Excess temporal lobe alpha</td>
<td>Increased alpha activity generated in temporal lobe</td>
<td>Inhibit alpha over affected temporal region(s), and inhibit frontal slow activity</td>
</tr>
<tr>
<td>Faster alpha variants, not low voltage</td>
<td>Alpha peak frequency greater than 12 Hz over posterior and parietal cortex</td>
<td>Reward 8-10 Hz alpha at Pz, shift alpha frequency slower with alpha/theta protocol</td>
</tr>
<tr>
<td>Spindling excessive beta</td>
<td>Rhythmic beta with a spindle morphology, often with an anterior prominence</td>
<td>Inhibit beta's spindle frequencies, wide band inhibit; alpha-theta training may help</td>
</tr>
<tr>
<td>Persistent eyes-open alpha</td>
<td>Alpha doesn’t attenuate by at least 50% with eyes open; it is generally slower alpha</td>
<td>Reward beta frequencies, inhibit alpha; reward higher frequency alpha</td>
</tr>
</tbody>
</table>
EEG PHENOTYPES IN AUTISM

Autism comprises a heterogeneous clinical and neurophysiological group of symptoms and phenotypes.

A single biomarker in ASD is no longer supported.

Research and clinical data have reported different phenotypes with also specific incidence.

This information is essential to guide the choice of the treatment in the light of response, outcomes and effectiveness.
EEG phenotypes are further described as highly heritable and reliable measures of brain functioning that carry treatment planning implications (Johnstone, Gunkelman, & Lunt, 2005).

- Measuring brain activity with EEG has great potential for use in the diagnosis of autism
- The distribution of EEG spectral disturbances often overlap the clinical observed behaviour.
- Brain activity is also linked to severity of symptoms

“EEG coherence presents a unique target for treatment of these and other populations, in that the ability to modulate connectivity via EEG neurofeedback has been shown to be of significant clinical utility (Decker et al, 2017)”

<p>| Table 1 |</p>
<table>
<thead>
<tr>
<th>Assumptions regarding brain connectivity and cognitive functions.</th>
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</thead>
<tbody>
<tr>
<td>1. For any given cognitive function, there are multiple brain structures or sets of structures that are primarily involved in performing that function.</td>
</tr>
<tr>
<td>2. In most cases, these areas are functionally (and often structurally) connected, forming a specific network.</td>
</tr>
<tr>
<td>3. Dysfunction of a network via over- or under-connectivity will result in reduced proficiency in cognitive functions reliant upon connectivity of the involved brain regions.</td>
</tr>
</tbody>
</table>
# COMMONLY REPORTED EEG PHENOTYPES IN AUTISM

| THE PRESENCE OF SLOW EEG ACTIVITY, FREQUENTLY DELTA | White matter disturbance also present  
DTI provides more details on white matter disturbances and high delta activity very often reflecting active, impulsive and aggressive behaviours in ASD. |
| EPILEPTIFORM PAROXYSM | Frontal discharges disturb higher functions of attentional and affective regulation  
Posterior and parietal discharges involving disturbances in sensory processing (Casanova et al., 2013). |
| BETA SPINDLES | Mu rhythm is disproportionally present in autism suggested to be the effect of fronto-central disconnections associated with mirror neurons. |
| BOTH HYPOCOHERENCE AND HYPERCOHERENCE, ABNORMAL COHERENCE AND NETWORK DISTRIBUTION | Functional connectivity alterations in theta band (Sperdin et al, 2017) |
COMMONLY REPORTED EEG PHENOTYPES IN AUTISM

Neurotypical

ASD
AUTISM AND EPILEPSY

- Comorbid epilepsy estimated in about 30% of Autism.
- It is possible to observe an age-related pattern for the onset of seizures that may be presumably related to critical changes in the brain during neural development.
- Bimodal incidence:
  - Infancy to 5 yrs.
  - Adolescence (< 10 yrs.)
- These phases mostly reflect hyper excitability and aberrant brain coherence that also may likely account for the often-observed hyperactivity.
- Probability increase with severe symptoms and presence of other disorders as mental retardation.
A study by Murias et al. (2007) explored the fact that theoretical conceptions of autism spectrum disorder (ASD) and experimental studies of cerebral blood flow suggest abnormalities in connections among distributed neural systems in ASD. Functional connectivity was assessed with electroencephalographic coherence between pairs of electrodes in a high-density electrode array in narrow frequency bands among 18 adults with ASD and 18 control adults in an eyes closed resting state.

In the theta (3-6 Hz) frequency range, locally elevated coherence was evident for the ASD group, especially within left hemisphere frontal and temporal regions.

Topography of significantly elevated ASD group coherences in the 3–6 Hz band. Lines are drawn between channel pairs at which ASD group coherence exceeded control with p-values below .025. Lines are coloured according to distance (cm) between electrode pairs, along the scalp surface.
ABNORMALITIES IN CONNECTIVITY & COHERENCE

Robust patterns of over- and under-connectivity are apparent at distinct spatial and temporal scales in ASD subjects in the eyes closed resting state.

In the lower alpha range (8-10 Hz), globally reduced coherence was evident for the ASD group within frontal regions and between frontal and all other scalp regions. The ASD group exhibited significantly greater relative power between 3 and 6 Hz and 13-17 Hz and significantly less relative power between 9 and 10 Hz.

Robust patterns of over- and under-connectivity are apparent at distinct spatial and temporal scales in ASD subjects in the eyes closed resting state.

(Murias et al. 2007)
A study conducted by Coben et al. in 2008 explored the difference between a control group and a group with ASD in terms of connectivity and coherence. There were group differences in power, intrahemispheric and interhemispheric coherences. Findings included excessive theta, primarily in right posterior regions, in autistics. There was also a pattern of deficient delta over the frontal cortex and excessive midline beta. More significantly, there was a pattern of underconnectivity in autistics compared to controls. This included decreased intrahemispheric delta and theta coherences across short to medium and long inter-electrode distances. Interhemispherically, delta and theta coherences were low across the frontal region. Delta, theta and alpha hypocoherence was also evident over the temporal regions. Lastly, there were low delta, theta and beta coherence measurements across posterior regions.

These results suggest dysfunctional integration of frontal and posterior brain regions in autistics along with a pattern of neural underconnectivity. This is consistent with other EEG, MRI and fMRI research suggesting that neural connectivity anomalies are a major deficit leading to autistic symptomatology.
ASD AND DELTA AND THETA COHERENCE

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Intrahemispheric and interhemispheric coherences for Autistic and Control groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Delta</td>
</tr>
<tr>
<td>Intrahemispheric coherences</td>
<td></td>
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<tr>
<td>Short-medium</td>
<td></td>
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<tr>
<td>L vs. R</td>
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<tr>
<td>Autistic vs. Controls</td>
<td>****</td>
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<tr>
<td>Autistic vs. Controls X</td>
<td>-</td>
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<tr>
<td>L vs. R</td>
<td></td>
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<tr>
<td>Long</td>
<td></td>
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<tr>
<td>L vs. R</td>
<td></td>
</tr>
<tr>
<td>Autistic vs. Controls</td>
<td>****</td>
</tr>
<tr>
<td>Autistic vs. Controls X</td>
<td>-</td>
</tr>
<tr>
<td>Interhemispheric coherences</td>
<td></td>
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<tr>
<td>Frontal</td>
<td></td>
</tr>
<tr>
<td>Autistic vs. Controls</td>
<td>***</td>
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<tr>
<td>Temporal</td>
<td></td>
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<tr>
<td>Autistic vs. Controls</td>
<td>*</td>
</tr>
<tr>
<td>Central/posterior</td>
<td></td>
</tr>
<tr>
<td>Autistic vs. Controls</td>
<td>*</td>
</tr>
</tbody>
</table>

\(* p < .05.\)  
\(* * p < .01.\)  
\(* * * p < .005.\)  
\(* * * * p < .001.\)


*EEG power and coherence in autistic spectrum disorder.*

*Clinical Neurophysiology, 119(5), 1002–1009.*


*A big-world network in ASD: Dynamical connectivity analysis reflects a deficit in long-range connections and an excess of short-range connections.*

*Neuropsychologia, 49(2), 254–263.*


*A stable pattern of EEG spectral coherence distinguishes children with autism from neuro-typical controls - a large case control study.*

*BMC Medicine, 10(1), 64.*
REFERENCES

IMPROVING COGNITION AND MOTIVATION IN PATIENTS WITH NEUROPSYCHIATRIC DISORDERS

Barbara J. Sahakian
University of Cambridge, Department of Psychiatry and Behavioural and Clinical Neuroscience Institute, Cambridge, UK

While many people monitor their physical health using mobile devices and wearable technology to preserve their physical health throughout their life course, they rarely consider improving and monitoring their brain health. This is strange, when we consider the regrettable statistics that one in four of us will suffer a mental health disorder at some point in our lives and that 75% of mental health disorders start before 24 years of age and 50% before 18 years of age. If we are going to have good mental capital and wellbeing throughout our lives, it is imperative that we consider mental health as being every bit as important as physical health and move to game-changing initiatives which include early detection and early effective treatment of neuropsychiatric disorders (Narayan & Manji 2016, Beddington et al. 2008, Sahakian 2014). Major approaches will include biomarkers, including cognitive ones, for early detection, but also will utilise novel pharmacological and also technological approaches to treatment, including neuroprotective drugs for Alzheimer's disease, fast acting antidepressant drugs for depression, cognitive enhancing drugs and game apps for delivering cognitive training on mobile phones or tablets in schizophrenia and other neuropsychiatric disorders (Savulich et al 2017, Sahakian et al. 2015, National Academies of Sciences Engineering and Medicine 2015, Insel et al. 2013, Bruhl & Sahakian 2016). In changing the framework by moving to early detection and early effective treatment, we can stop these mental health disorders becoming debilitating, chronic and relapsing. Using these novel pharmacological and non-pharmacological treatment approaches, we can ensure that patients with neuropsychiatric disorders have better quality of life, functionality and wellbeing. Not only can innovation and technology promote a flourishing society, but could also reduce the cost and burden of neuropsychiatric disorders for governments.

EEG BASED PHENOTYPES: TRANSLATING RESEARCH METHODOLOGIES TO ACHIEVE PERSONALIZED INTERVENTIONS

Adrian Attard Trevisan
Neurotech International Ltd, Australia

A number of QEEG and EEG studies suggest that it is possible to identify a number of traits and power values directly related to specific physiological and psychological states and conditions.

Several studies already grouped these clusters alongside other types of “phenotypes” with a very high level of success. These studies varied from achieving repeatability of EEG phenotypes in baboons with epilepsy (Szabao et al. 2005) to phenotypes directly related to a number of sections of Autism Spectrum in humans (Duffy & Als 2012).

Among neurodevelopmental disorders, Autism comprises a heterogeneous clinical and neurophysiological group of symptoms and phenotypes, as well as remarkable comorbidities with other neurological disorders as epilepsy. In fact, the majority of studies suggest that approximately one-third of children with ASD develop epilepsy. Furthermore, in Autism spectrum disorders it is possible to observe an age-related pattern for the onset of seizures that may be presumably related to critical changes in the brain during neural development. These phases mostly reflect hyperexcitability and aberrant brain coherence that also may likely account for the often-observed hyperactivity.

Together with these observations, research and clinical practice are moving forward the utilization of comparative studies between the subtypes and subcategories of Autism spectrum since the expression of different phenotypes may have an impact not only on the variety of symptoms but also on the degree of severity with which these symptoms are manifested. This latter discrimination meets the need to effectively address specific neural functioning within the ‘spectrum’ and to potentially predict the expression of related comorbidities.

Integrating these EEG based phenotypes are thus considered to be an easy, yet effective way of having remarkable clinical information and distinct biomarkers into the clinician’s toolbox, both for diagnosis and monitoring.
Advances in HD-tDCS stimulation: 
a new pipeline to build anisotropic head models

Marco Rotonda, PhD

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From tDCS to HD-tDCS

From tDCS to HD-tDCS

Optimisation into HD-tDCS

Figure 7. Optimized electrode stimulus patterns for targeting (a) M1 ROI in cortical surface normal direction, using (b) Dmochowski et al.’s, (c) Ruffini et al.’s, and (d) Guler et al.’s optimization formulation.

## Models into HD-tDCS

<table>
<thead>
<tr>
<th>Method</th>
<th>Description</th>
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<tbody>
<tr>
<td>BEM</td>
<td>Solves the surface integral equations instead of the volume partial equation. Only isotropic.</td>
</tr>
<tr>
<td>FDM</td>
<td>Digitizing the whole volume into small volumetric elements. Inhomogeneity and anisotropy can be handled. The regular cubed images in FDM map directly to the computational grid.</td>
</tr>
<tr>
<td>FEM</td>
<td>Digitizing the whole volume into small volumetric elements. Inhomogeneity and anisotropy can be handled. FEM is computationally more efficient than FDM. Shown to achieve high numerical accuracies. Constructing a FEM mesh from MRI image is difficult and can be inaccurate because of the complex head geometry.</td>
</tr>
</tbody>
</table>

**Finite Element Method (FEM)**

- **Boundary Element Method (BEM)**
- **Finite Difference Method (FDM)**
Pipeline proposed

MRI + DTI → Segmentation of skin, skull, white matter, gray matter and cerebrospinal fluid → Electrodes placement

NeuroBattery/ANTs

Huang & Parra (2015)

Guler et al. (2016) → Montage optimization

Discontinuous Galerkin (DG) solver

PABLO: PArallel Balanced Linear Octree

Conductivity assignment → Volume Meshing
Segmentation and image handling

NiBabel: Read / write access to some common neuroimaging file formats

ANTs - Advanced Normalization Tools is, at the moment, the state of the art tool to automatically segment and label MRI images

NeuroBattery: ANTs script for handling different images of single subject MR processing


Electrode montage

For the creation of the individualised electrodes montage we followed the method proposed by Huang & Parra (2015)

Volume meshing

We automatically generate a discretized computational domain, including the electrodes, using an octree-based approach, which produces a non-conforming mesh of hexahedral elements which are adaptively refined according to the geometry as well as the rate of change in conductivity properties, particularly within the brain.

In this example ~6.5Millions of elements

PABLO: PArallel Balanced Linear Octree (https://github.com/optimad/PABLO)


Conductivity assignment

Conductivity is assumed anisotropic in the white matter and gray matter, with conductivity tensors estimated from DTI MR sequences. The anisotropic Laplace equation is efficiently resolved using non-conforming Discontinuous Galerkin finite elements, using a stable numerical scheme to obtain potential field and electrical currents throughout the domain for a unitary stimulation per electrode.

SpaFEDTe: C++ library for discontinuous element spaces


Optimal montage

The mix of electrode currents to optimize the stimulation at target is then optimized by solving a convex optimization problem, following the method of Guler et al. (2016). This approach try to maximise the current over the ROI having the constrain chosen imposed.

Models adhere to reality?

A recent study provide important assurance that current flow models indeed do a good job in estimating how much current is delivered to the brain of an individual by tES, and where to.

Why models are important?

For a given stimulator output, current in a cortical target region may vary by up to 100% across individuals.

Thank you

I would like to thank Neurotech International for sponsoring this research and dissemination
ABSTRACTS

The Following abstracts represent other papers read in the meeting. They are only published in abstract form as they only represent work in progress or are to be published in full elsewhere.

RECENT ADVANCES IN THE NEUROBIOLOGY
OF OBSESSIVE-COMPULSIVE RELATED DISORDERS

Samuel R. Chamberlain
Department of Psychiatry, University of Cambridge & Cambridge & Peterborough NHS Foundation Trust, Cambridge, UK

Several mental disorders share phenomenological and comorbid overlap with obsessive-compulsive disorder, including trichotillomania (hair pulling disorder), gambling disorder, and compulsive Internet use. These disorders merit clinical and research scrutiny due to their high prevalence and untoward functional consequences. They are under-studied, under-recognized, and under-treated.

As considered elsewhere in the symposium, several neuroscientific concepts are useful for understanding obsessive-compulsive related disorders, including ‘impulsivity’, ‘compulsivity’, and ‘behavioural addiction’. Impulsivity refers to premature, unduly hasty acts, poorly thought out, leading to negative untoward consequences. Compulsivity refers to repetitive acts undertaken in a stereotyped or rigid manner, leading to negative functional consequences. Behavioural addiction draws analogies with substance addiction: some behaviours, as with certain substances (e.g. cocaine), are highly rewarding and (for vulnerable individuals) and thus are prone to repetition.

This talk will focus on impulsivity, compulsivity, and behavioural addiction, as these terms apply to the obsessive-compulsive related disorders. Current diagnostic criteria and categories will be examined. Focusing on cognitive tasks of inhibitory control by means of example, I shall identify overlapping cognitive profiles between these disorders, along with implicated neural abnormalities. This will include presentation of findings from an international mega-analysis of structural brain findings in trichotillomania, which found excess cortical thickness in a key inhibitory node (the right inferior frontal gyrus), coupled with morphometric abnormalities of the nucleus accumbens reward centre. Using a rich population-based dataset, I will then consider the relationship between impulsivity, compulsivity, and addiction in the dimensional rather than categorical sense.

ADVANCES IN HD-TDCS STIMULATION:
A NEW PIPELINE TO BUILD ANISOTROPIC HEAD MODELS

Marco Rotonda
AAT Research, San Gwann, Malta marco.rotonda@mentetech.com

The transcranial Direct Current Stimulation (tDCS) is emerging as a promising technique in rehabilitation, treatment of conditions or peak performance.

Starting with only two big sponges (one anode and one cathode), in the last years is affirminga new approach using multiple little sponges: High Density tDCS (HD-tDCS). With this novel approach is essential to model the brain in order to maximise the effect in the region of interest. There are different approaches in the modelisation of the brain. One of the most promising is the Finite Element Model (FEM) that could take in consideration the anisotropic conductivity properties of the different structures. For the creation of a FEM, a fast and not so explored approach is the octree one.

We utilised this octree-based approach to produce a non-conforming mesh of hexahedral elements which are adaptively refined according to the geometry of the different structures (skin, skull, white matter, grey matter and cerebrospinal fluid) as well as the rate of change in conductivity properties, assumed anisotropic.

The anisotropic Laplace equation is efficiently resolved using non-conforming Discontinuous Galerkin finite elements, using a stable numerical scheme to obtain potential field and electrical currents throughout the domain for a unitary stimulation per electrode. The mix of electrode currents to optimize the stimulation at target (identified by absolute location or through the specification of a particular region of interest) is then optimized by solving a convex optimization problem, following the method proposed by Guler et al. (2016).
A home-based neurofeedback therapy for autism spectrum disorders: A case study of a young adult patient

EMANUELA RUSSO, PhD
THE CASE STUDY

The subject
- Patient is a young adult woman of age 19 at the time of intake.
- She has a diagnosis of Autism.
- EEG pattern: Excessive slow waves as well as exaggerate amount and distribution of Beta waves as compared to neurotypical pattern.

The intervention
- Neurofeedback sessions provided by means of a home-based device.
- Daily sessions were collected on a web server.
- Feedback consisted in an auditory stimulation and therapy protocol aimed to normalize abnormal patterns as observed in the qEEG.
MEASUREMENTS AND DATA ANALYSIS

Study Design

- A–B design (with follow-up)

  The Baseline (B) phase comprised 5 observations per week, the Intervention (I) phase comprised 8 observations per week. The Follow-Up (F) phase consisted of 8 observations every 2 weeks.

  Each observation consisted of an EEG recording with Eyes closed on 19 channels 10-20 system.

Data Analysis

- Data analysis of the measurements was conducted using Simulation Modeling Analysis (SMA) for Time-Series (Borckardt, 2006).

  SMA evaluates the statistical significance of between phase changes in data streams and also accounts for the presence of autocorrelation.

  A phase-effect size (Pearson’s r) is produced for each phase comparison. The data stream of each phase is then compared to a distribution of random data streams, resulting in an empirical estimate of the probability of the observed effect occurring by chance.
RESULTS AND OUTCOMES

**Baseline Phase**

- Relative Power
  - Total
  - Delta
  - Theta
  - Alpha
  - Beta
  - Beta2

**Intervention Phase**

- Relative Power
  - Total
  - Delta
  - Theta
  - Alpha
  - Beta
  - Beta2

**Graph**

- **Delta Power Frontal sites**
- **Weeks**
  - 1 2 3 4 5 6 7 8 9 10 11 12
- **Baseline** to **Intervention**

**Statistics**

- $R = -0.539$, $p = 0.0824$
RESULTS AND OUTCOMES

R = -0.660, p = 0.0436
RESULTS AND OUTCOMES

Theta Power

Weeks

Baseline Intervention

1st Observation
Baseline Phase

Last Observation
Intervention Phase

Absolute Power
Total Delta Theta Alpha Beta Beta2

Relative Power
Total Delta Theta Alpha Beta Beta2

Frontal Central Posterior
RESULTS AND OUTCOMES

Alpha Power

Weeks

Frontal ➔ Central ➔ Posterior

1st Observation
Baseline Phase

Last Observation
Intervention Phase

Absolute Power

Relative Power

Total
Fp1, Fp2, F7, F3, Fz, F4, F8,
T3, C3, Cz, C4, T4,
T5, P3, Pz, P4, T6,
O1, O2.

Delta

Theta

Alpha

Beta

Beta2

Ist Observation
Baseline Phase

Last Observation
Intervention Phase

4 Z

2 Z

0 Z

-2 Z

-4 Z
RESULTS AND OUTCOMES

\[
R = -0.639, p = 0.0424
\]
RESULTS AND OUTCOMES

Ist Observation
Baseline Phase

Last Observation
Intervention Phase
DISCUSSIONS AND REMARKS

- Results of the SMA phase effect analyses indicate a number of trends in expected directions and some significant effects.
  - Delta decrease in Frontal and Centro-temporal areas toward normalization
  - Beta decrease in Frontal and Centro-temporal areas toward normalization
  - Alpha towards normalization
Thank you for your attention!

I would like to thank Neurotech International for sponsoring this research and dissemination.
PSYCHIATRIC EDUCATION ON THE EUROPEAN LEVEL: CHALLENGES AND OPPORTUNITIES

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Over the last decade, junior and senior psychiatrists have been working more closely throughout Europe in order to improve the quality of training. Numerous challenges have been identified such as a lack of training in psychotherapy, trainees’ burnout, stigma against mental illness and psychiatrists, migration and brain drain, and access and exposure to research. More generally, the diversity of training schemes and significant variations in their quality has been emphasised. The European Psychiatric Association (EPA), the UEMS (European Union of Specialist Doctors), the European Federation of Psychiatric Trainees (EFPT), and the World Psychiatric Association (WPA), among others, have produced numerous initiatives in order to support Early Career Psychiatrists to improve the quality of their training. The Task Force on European Education in Psychiatry, composed of members of EPA, WHO, UEMS and EFPT, is aiming at harmonising postgraduate training in psychiatry throughout Europe and develop a European training curriculum in psychiatry.

A HOME-BASED NEUROFEEDBACK THERAPY FOR AUTISM SPECTRUM DISORDERS: A CASE STUDY OF A YOUNG ADULT PATIENT

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Several neuroanatomical findings highlighted that the neuropathology of autism may represent an ongoing process with age-related changes in the brain. On the other hand, a relatively consistent and unique profile of electrophysiological abnormalities emerged from resting-state EEG studies, which appears to be present across diverse patient populations. Specifically, excessive power at low-frequency and high-frequency bands, as well as reduced power in the middle-range frequency band, has been found in ASD individuals. This pattern presumably underlies specific core symptoms within the spectrum in ASD.

The case study we are presenting deals with treatment outcomes of a home-based neurofeedback therapy in a young adult woman with ASD. The home-based system delivers tailored binaural beats as an auditory feedback aimed to entrain the brain into a more relaxed and focused state. The patient was 19 years old at the intervention phase with a clinical diagnosis of ASD. A quantitative electroencephalographic (QEEG) analysis revealed increased delta and high beta activity as well as high bipolar relative power in delta and theta frequencies on frontal areas.

A single case pre- and post-intervention design was adopted. The neuropsychological profile of the patient was compared pre and post the neurofeedback treatment. The patient completed an 8-week intervention period during which she utilized daily the home-based device which delivers a tailored neurofeedback therapy using binaural beats. Further data have been collected for a follow-up period of 16-weeks. Pre-post evaluation of the treatment efficacy has also been provided from the Autism Treatment Evaluation Checklist (ATEC).

Analysis of baseline, intervention phase, and follow-up are presented and implications of these findings are discussed. The present study puts forward the feasibility of treating young adult patients with the present home-based neurofeedback therapy.