

## Time for a theory-driven approach to QEEG

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QEEG has been used for the last 30 years and has become more widely available with the increases in processing power of computers which enabled 'digital EEG'. Many clinicians nowadays are using QEEG in their practice, mainly for guiding neurofeedback protocols and for some to guide medication prescription.

In our field often findings from published group studies are used to speculate about the best treatment recommendation, such as the theta/beta ratio in ADHD and frontal (alpha) asymmetry in depression. However, this is mere speculation, since there is no theory nor adequate research backing this up for use in individual clients. Furthermore, current use of individualized or personalized QEEG is more technology-driven rather than theory-driven. These two issues will be further discussed below, along with a new theory-driven model which might change the way we interpret QEEG and generates testable hypothesis.

### Averaged group data vs. Individual client data

An often reported finding in ADHD is the theta/beta ratio. Indeed most group-averaged studies comparing ADHD with a healthy control groups will show ADHD kids have more 'theta' and less 'beta' EEG power (also see figure 1 below). However, when looking at this exact same data on an individual level a completely different picture emerges where only 25% of children with ADHD showed excess frontal slow. Furthermore, an additional 25% of children showed a slowed Alpha Peak Frequency (APF) which showed up in frontal sites as 'theta'. Also see Arns et al., 2008 for the full background on these data. Most importantly these two groups responded differentially to stimulant medication as well. So indeed the theta/beta ratio might often deviate, however what is the cause of that? Excess frontal slow or a slowed APF?

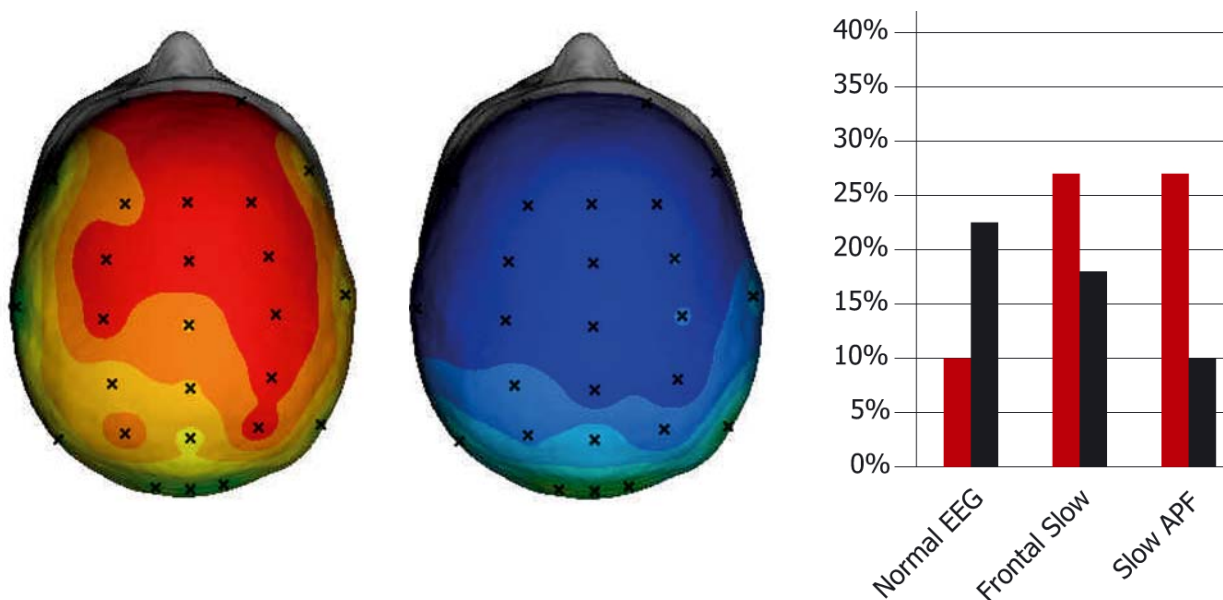


Figure 1: The head-maps on the left show the average Theta and Beta power for 250 children with ADHD compared to a control group (data from the Brain Resource International database). The graph on the left shows that when inspecting the individual data sets only 25% of children with ADHD (red) indeed exhibit Frontal slow or Frontal Theta. Finally, about the same percentage exhibit a Slow APF pattern, which after filtering will show up as 'Theta' but in fact is alpha.

In Depression research often the frontal alpha-asymmetry is mentioned and investigated based on Davidson's work. rTMS or magnetic brain stimulation is also partly based on this work with the assumption that Depression is characterized by left frontal hypoperfusion. After inspecting over 200 individual depressed clients from both our clients and data from a clinical trial, I can assure that a specific frontal asymmetry is not reliably found in individual depressed patients. In some previous studies employing group-averaged data we did find the expected frontal alpha asymmetry, demonstrating that our used methodology is fine. So how can this be, that findings are often found in group-averaged data but cannot be found reliably in individual data?

Take the hypothetical example of 100 people sitting in a room and we average their eye-color. The average will be black! How many people do you know with black eyes?

This demonstrates that by averaging group data we might end up with a construct which does not exist in reality. Since we are not treating an average group but an individual client this is very important to keep in mind!

### **Technology-driven QEEG**

Currently many new analysis tools and databases have become available to perform more sophisticated QEEG analysis. Given the large number of permutations some packages allow being done on EEG data will make some statisticians frown their eye-brows (think about alpha-correction for multiple statistical tests). Many of these new techniques have been hardly investigated or validated for clinical use and are often adapted from ECoG or depth recordings from multiple cells and directly applied to EEG. Take the simple example of coherence. There are many different ways to calculate coherence and two QEEG studies in dyslexia have shown completely opposite effects for EEG coherence data (Arns et al., 2007 study and Coben et al. Study presented at ISNR). This could even suggest that what we are concluding based on database X (coherence needs to be up-trained), but by performing the neurofeedback with software Y we might be feeding back something completely different (i.e. downtraining). Therefore the technology driven QEEG approach - although very interesting and exciting - should be complemented more by theory driven QEEG and be better validated and standardized. Ultimately it should be the interaction between theory-driven and technology-driven QEEG which will lead to real new discoveries in our field!

### **Theory-driven QEEG**

Currently no real theory for interpreting individual QEEG's exists. One of the first published papers on interpreting QEEG's was the pioneering paper of Jack Johnstone, Jay Gunkelman and Joy Lunt on EEG Phenotypes (2005). Although this paper did not represent a unifying theory, it at least summarized the different EEG patterns or proposed EEG Phenotypes and their proposed treatment recommendation for Neurofeedback and Medication. Most importantly, it provided a testable model!

Over the last couple of years Jay Gunkelman and myself started testing this model based on data generously provided by the Brain Resource Company. We 'EEG Phenotyped' 49 children with ADHD, 113 patients with Depression and a group of 170 healthy controls. The 'EEG Phenotype' construct proposed the EEG Phenotypical patterns to be stable - trait-like - patterns, hence the term 'Phenotype'.

Along these developments Werner van den Bergh exposed us via a newsgroup to the Vigilance model of Bente (1964) and at the ECNS conference last year I was also

exposed to a presentation from Ulrich Hegerl who presented data on this same model as well. This 'EEG Vigilance' model is originally based on the work by Bente, and nicely fits into the well investigated sleep-wake states also described by Dement & Kleitman (1957), Loomis et al (1937) and Roth (1961), also see figure 2 below. Vigilance in this sense refers to Henry Head's concept of vigilance: 'the organization and efficiency of the adaptive capabilities of the individual'.

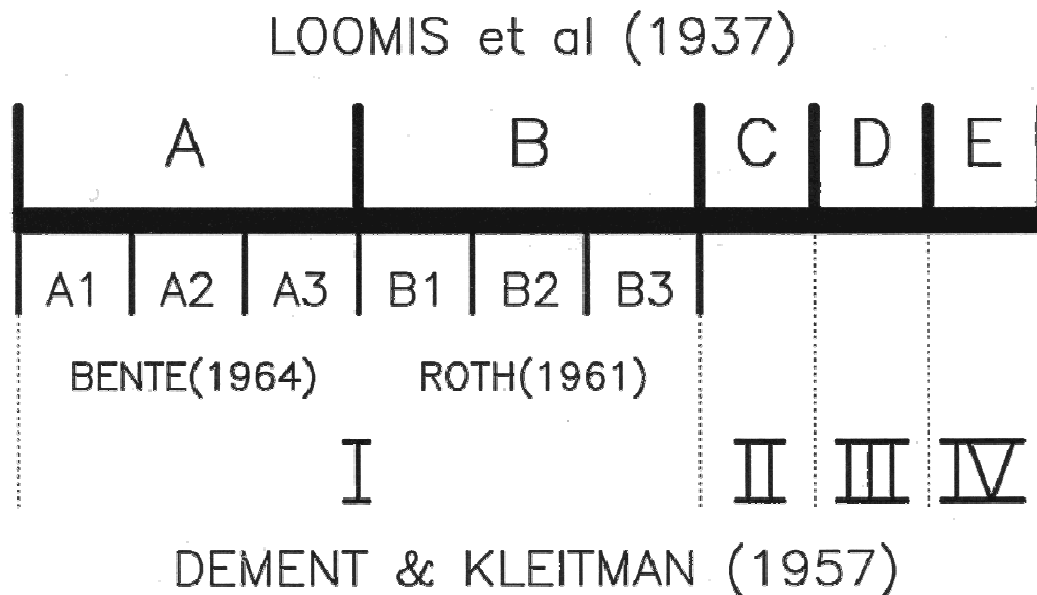


Figure 2: The well investigated transition from wakefulness (A) to passive waking (B) to the different sleep-stages (C, D and E). The in this article described vigilance model is related to the A and B stages, stage C is Slow Wave Sleep (SWS)

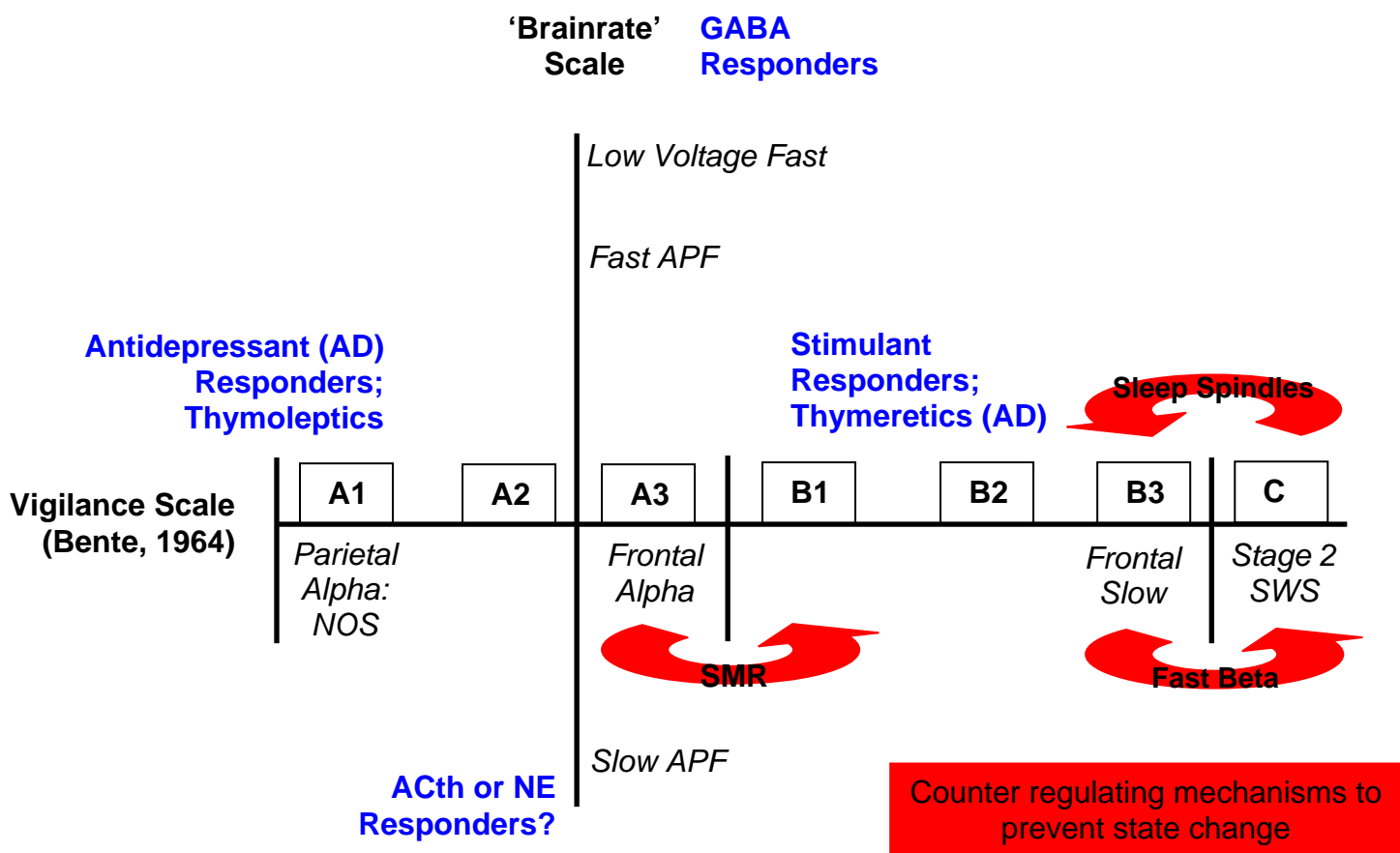
When reading the description of the A and B states one sees a lot of similarities between the EEG Phenotypes and these Vigilance stages, however now as *dynamic variants of the EEG* which can occur within the same subject as a function of time! Briefly, they observed that when people close their eyes for 10 minutes, their EEG will in most cases cycle through stages of parietal alpha (A1), frontal alpha (A3), an intermediate 'low-voltage like' state with some beta spindles (B2) and finally – before falling asleep – a frontal slow EEG.

This started challenging the view of stable EEG Phenotypes. This along with some inconsistencies we found from the original Johnstone et al. paper with respect to treatment outcomes, led us to organize a workshop last November with Jay Gunkelman, Werner van den Bergh and myself. This finally resulted in a new theoretical-model which incorporated both the EEG Phenotypes and the Vigilance model from Bente. Most importantly, the data from our EEG Phenotype experiments fit very well into the model. Details on this new model are currently being prepared for publication.

In this new model we view EEG phenotypes as the 'predominant vigilance state'.

## The 2-dimensional Vigilance-Brainrate model

The picture below shows a graphical display of the proposed 2-dimensional 'Vigilance-Brainrate' model. The X-axis displays the original Vigilance model by Bente (1964) with the different A, B and C stages, where the C stages refer to sleep. Below the different stages the EEG Phenotype name in *italics* indicates where the different EEG Phenotypes fit into the model. The Y-axis – which is the main expansion of the model – is called the Brainrate or 'Speed of Processing' scale. This scale indicates a very slow APF (bottom) to a very fast APF, finally resulting into a Low Voltage Fast EEG. In Blue we indicated the medication response for the different groups. Furthermore, in red the counter-regulating mechanisms are depicted which can be used to guide Neurofeedback treatment. The box below mentions a summary of remaining 'neurological' or 'localized' EEG Phenotypes.

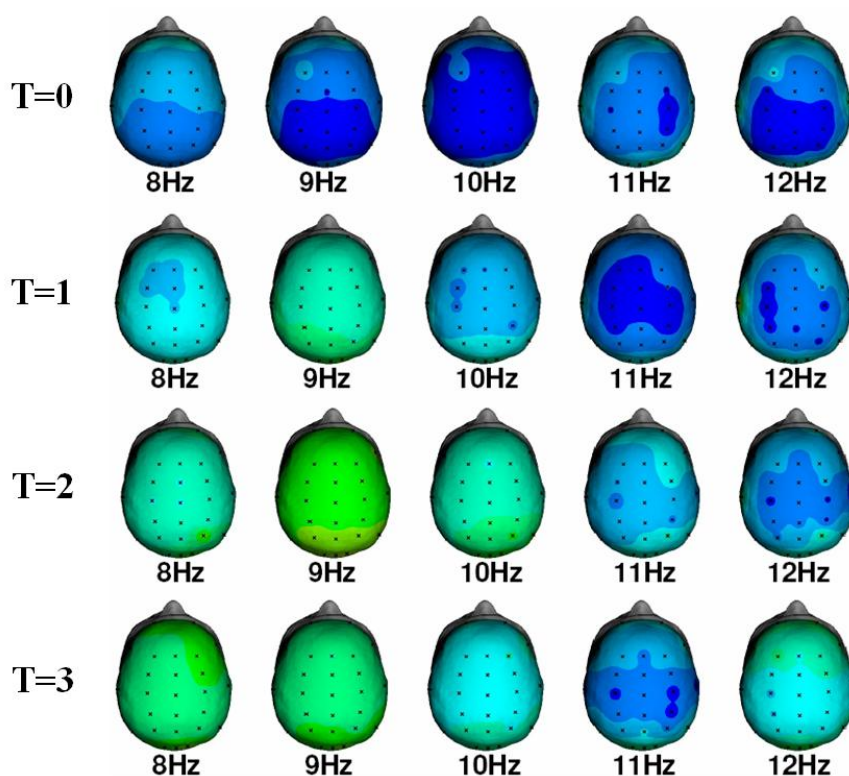


### "Neurological" or "Localized" EEG Phenotypes

- 1) Paroxysmal EEG
- 2) Beta Spindles
- 3) Cingulate dysfunctions (midline excess Alpha, excess Theta or excess Beta (spindles))
- 4) Persistent Mu rhythm (not attenuated by hand movement).
- 5) Temporal alpha

Werner's important addition of 'counter regulating mechanisms' is very important for Neurofeedback. It is well known for instance that Sleep Spindles serve to keep people asleep, so prevent the brain from going from stage C to B. Therefore Sleep Spindles can be considered a counter-regulating mechanism for stage C. In the same way fast beta is a counter regulating mechanism for preventing people to shift from stage B to C and SMR to prevent a transition from A to B. This might also explain why both beta/theta and SMR/theta training work in the treatment of ADHD, albeit both might work slightly better for different sub-groups of ADHD, i.e. SMR for frontal alpha and Fast Beta for the frontal slow subtype. In a similar fashion Stimulant medication helps people moving to A stages when they are in B stages, which also makes a lot of sense from a vigilance perspective.

The Brain-rate scale is more or less an alpha-peak frequency (APF) scale where going up this scale is associated with faster APF's until the EEG completely desynchronizes into a low-voltage EEG without any alpha. In a small N=1 study we replicated findings Yuri Kropotov has also reported before, where alcohol (indirectly increasing GABAergic activity) made alpha re-appear again with increasing doses of alcohol and eventually slowed down the alpha peak frequency as well. Also see figure 4 below. Hence this scale can also be regarded a continuum which is partly regulated through GABA. More Neurofeedback research is needed to demonstrate the effects on this scale, by up or down-training the APF. Given some studies and some preliminary results from our practice, we suspect rTMS or magnetic brain stimulation acts more efficiently on this scale. This might explain the differential efficacy of Neurofeedback on disorders such as ADHD (= evidence based) and rTMS on Depression (= evidence based).



*Figure 4: This figure shows EEG power in the alpha band with increasing doses of alcohol (T=0 is pre-alcohol and T=3 is after 0,5 Liter of Vodka). The EEG at T=0 shows a low-voltage fast EEG, and with increasing doses an increase in alpha is seen, eventually slowing down with the highest dose.*

In summary, the 2-dimensional Vigilance-Brain rate model is grounded in theory and also fits data. We will be writing up this model and refine it further through more literature and data, which should eventually result in a publication. Although this model is still very preliminary, it generates a lot of testable hypothesis and we welcome any further input from you on how to further improve, test and refine this model. We feel that it is time for a more theoretical underpinning of what we are doing!

Looking forward to your feedback and input!

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