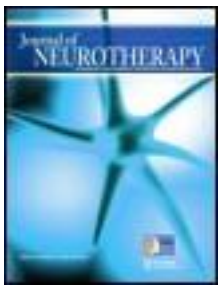


Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

Rethinking Standard Bands

David A. Kaiser PhD [a]

[a] Sterman-Kaiser Imaging Laboratory , Bel Air, CA

Published online: 08 Sep 2008.

To cite this article: David A. Kaiser PhD (2001) Rethinking Standard Bands, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 5:1-2, 87-96, DOI: 10.1300/J184v05n01_08

To link to this article: http://dx.doi.org/10.1300/J184v05n01_08

TECHNICAL NOTES

Rethinking Standard Bands

David A. Kaiser, PhD

The world is becoming more customized each day, yet most of us continue to use off-the-shelf principles when we assess and train individuals. In his first report Hans Berger (1929) identified large rhythmic sinusoidal waveforms in the human EEG, which he designated as "waves of the first order," or simply "alpha waves" for brevity's sake. Alpha waves appeared in bursts of 10 cycles per second, were pronounced in posterior regions when the eyes were closed, and abated when the eyes opened.

This is how the alpha rhythm was defined 70 years ago. Today we include a quantitative dimension to our definition. The alpha rhythm is designated as sinusoidal waves that dominate the spectral density distribution during eyes closed rest, but a standard frequency band of 8 to 13 Hz now measures its presence. Or 8 to 12 Hz. Or 7 to 13 Hz. Or 8 to 15 Hz (e.g., Etevenon, Eustache, Mitermite & Lepaisant, 1990; Ray & Cole, 1985) . . . you get the idea. What is more disturbing than bandwidths varying between laboratories are the boundaries being used. Brain activity rarely falls in step with our system of natural numbers, but you would never know this by perusing the scientific litera-

David A. Kaiser is affiliated with Sterman-Kaiser Imaging Laboratory, Bel Air, CA.
Address correspondence to: David A. Kaiser, PhD, Box 49196, Los Angeles, CA 90049 (E-mail: dakaiser@skiltopo.com).

ture. A frequency band, as it turns out, is a compromise between ease of communication and the limits of one's analytical techniques. This, despite the fact that communication has never been easier and our computational power more advanced.

INDIVIDUAL ALPHA FREQUENCY (IAF)

Klimesch and his colleagues (Klimesch, Schimke & Pfurtscheller, 1993) developed a simple, common sense strategy for analyzing dominant frequency in an EEG recording. He identifies the dominant peak frequency for each subject, and then derives alpha and theta bands relative to this peak. The alpha band is formulated as the individual (peak) alpha frequency (IAF), plus and minus 2.5 Hz. Alpha and theta bands may also be estimated as a percentage of IAF (Doppelmayr, Klimesch, Pachinger & Ripper, 1998). The concern I have for this approach is the width of each band, the plus and minus 2.5 Hz; although it may have some empirical basis, it is also artificial. Some subjects may fit neatly inside a 5-Hz-wide band, others may require a narrower band. Obviously, this part of the formula can be refined. Nonetheless, the use of individualized frequency bands might shake some of the noise out of our analyses and improve the reliability and validity of our conclusions.

As shown in Figure 1, the dominant frequency extends from 7 to 15 Hz for this sample, although the vast majority of peak frequencies (>95%) are captured between 8 and 12 Hz. But what is not captured by an 8-12 Hz may account for more than 5% of the information, namely individual differences. The alpha rhythm is not homogeneous, but varies in peak from individual to individual (Doppelmayr et al., 1998), and the shape or width may be just as diverse. In the present sample the distribution (width) of alpha activity across the frequency spectrum varied by a factor of two: anywhere from 20 to 40% of alpha magnitude was contained in the single peak frequency (mean 27.3% ± 3.0%) (see examples in Figure 2).

As shown in Figure 3, the dominant peak frequency varies widely between 8 to 13 Hz, with a mean of 10.0 Hz ± 1.0 Hz. (I guess natural numbers can reflect brain behavior, at least in its aggregate.) This sample consists entirely of normal adults, and yet it demonstrates much variance in peak and distribution. So this begs the question: why do most of us resort to standard bands to evaluate "nonstandard" people, those with clinical conditions? The calculation of IAF is trivial, and can be performed using a one-channel EEG system, but with little topo-

FIGURE 1. Distribution of Peak Dominant Frequency for 124 Adults at Five Posterior Sites (P3, Pz, P4, O1, and O2) During Eyes Closed Rest. Each Subject Provided Two to Six Replications.

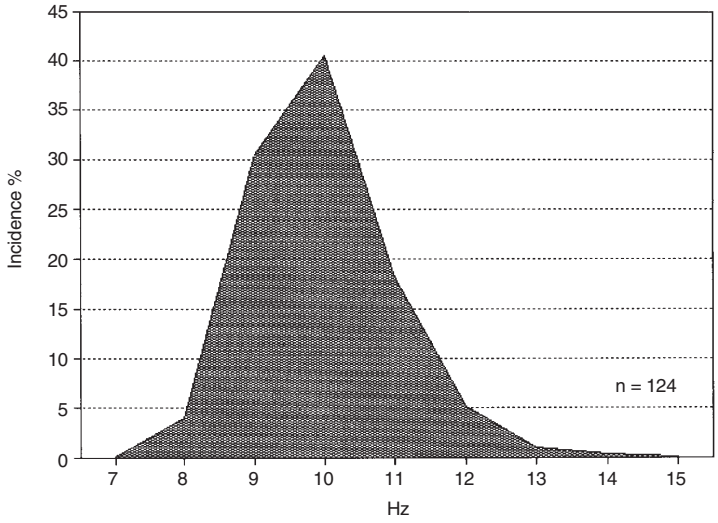
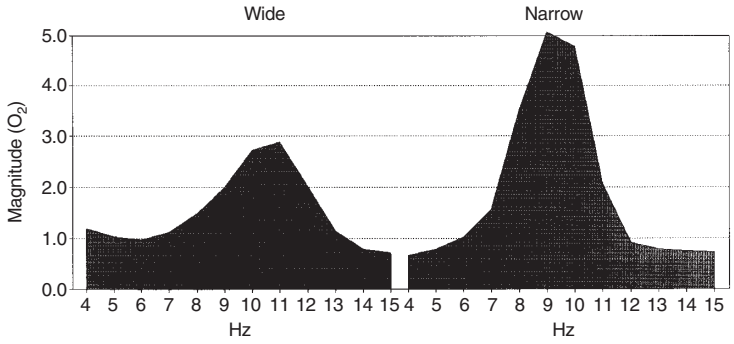
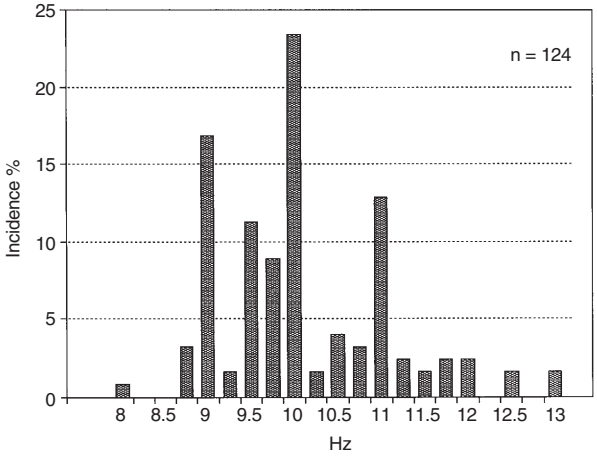


FIGURE 2. Alpha frequency Distributions During Eyes Closed Rest for Two Subjects.



graphic sensitivity and much repetitious work. For some individuals dominant frequency may vary somewhat across the scalp, but simply calculating a posterior IAF would be a good first step toward capturing individual differences in psychophysiology, and it should increase the reproducibility of our clinical findings.

FIGURE 3. Incidence of Peak Frequency Band During Eyes Closed Rest for 124 Adults. An Individual's Peak Frequency Band Is the Average of All Posterior Peak Frequencies.

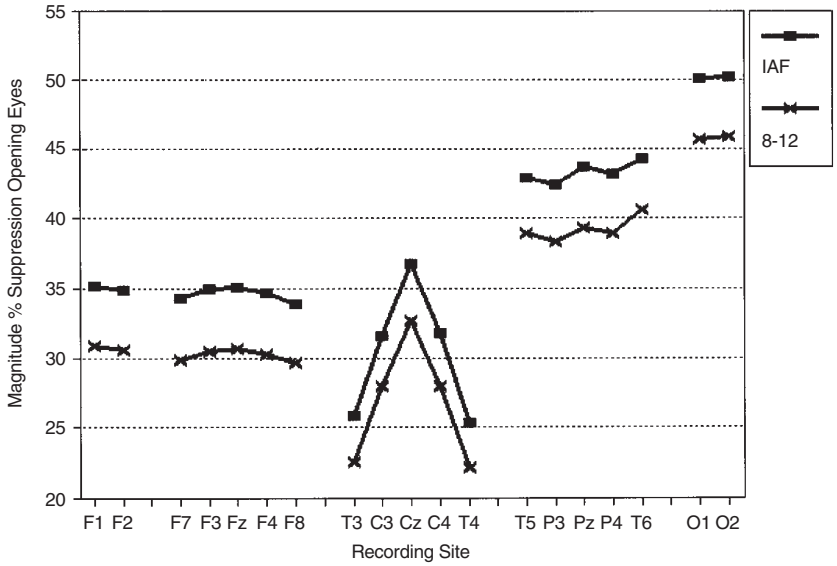


EVALUATING BAND MODELS

Which model for estimating alpha activity, IAF or standard bands, should we use? This question may be answered empirically. We expect an alpha band to capture accurately the behavior of the alpha rhythm. We know that the alpha rhythm attenuates from eyes closed to eyes open conditions, so one might compare the two models on how each responds to attenuation. As shown in Figure 4, IAF demonstrates slightly greater attenuation to eyes opening than a standard band of 8-12 Hz. IAF beats the standard band at the eyes open suppression test, in spite of the fact that IAF was calculated crudely in the present data set, with only 1-Hz bands at my disposal. Further tests will eliminate the inferior model, but already band customization shows promise.

One suggestion before we proceed: Although IAF, as proposed by Klimesch et al. (1993), captures individual differences in peak frequency, it ignores differences in spectral "morphology" or distribution. It relies on a predetermined distribution (± 2.5 Hz) for all individuals in generating each band. Further calibration might improve our estimations, taking into effect the general shape and width of an individual's dominant frequency. Some individuals may be served well by a 3-Hz-

FIGURE 4. Two Methods of Estimating Alpha Activity (IAF and Standard 8-12 Hz Band) Are Evaluated in Terms of Suppression in Response to Eyes Opening, in 104 Subjects.



wide alpha band, others by a 5.6 Hz-wide band. Of course, estimation of individual alpha distribution (IAD) is trickier than calculating peak frequency. IAD might be formulated as the interval centered around the peak frequency that captures 95% to 99% (two or three standard deviations) of the *rise in magnitude* across the middle frequency spectrum during eyes closed rest. Unfortunately, rise can be quantified in innumerable manners: in reference to a baseline, surely, but what constitutes the non-alpha baseline? Using magnitude in neighboring bands (e.g., 5-7 Hz and 13-15 Hz) for the baseline resulted in an IAD of 8.0-12.6 Hz (wide example) and 8.4-11.3 Hz (narrow example) for 99% rise (see Figure 2). To calibrate IAD consistently and reliably will require extremely rigorous and systematic recording techniques. Perhaps IAD is "a bridge too far," and essentially impractical under current circumstances and technologies. And we do not know what advantage, if any, such exceptional customization may provide. However, utilizing the IAF model to generate alpha bands is probably worthwhile and well within our reach today.

MATURATION OF THE ALPHA RHYTHM

The discussion to date has referred to the alpha rhythm for a normal adult population. When any of these three criteria are not met, the use of fixed standard bands becomes even more questionable. When we evaluate children with quantitative EEG, we need to take into account the degree of neurological maturation. The alpha rhythm does not spring forth at 10 Hz at birth; instead it emerges as a slow 4 Hz rhythm in infancy, and it takes ten years or so of development before the familiar 10 Hz rhythm establishes itself (Niedermeyer, 1999) (see Table 1). As some have suggested, the frequency of the alpha rhythm may reflect the general maturity or immaturity of a child's nervous system (e.g., Lazzaro et al., 1998). Thus, prominent 4-7 Hz activity in young clinical populations, notably attention deficit disorder, probably reflects an immature manifestation of the dominant thalamocortical rhythm (Harmony et al., 1995); theta is actually alpha.

Figure 5 shows two individuals excluded from the previous sample of 124 adults due to age and/or clinical condition. One is a 3-year-old child and the other at 65 is possibly entering second childhood, at least electrophysiologically. One alpha distribution is normal, the other a response to chronic alcoholism, and both would be poorly served by an 8-12 Hz alpha band.

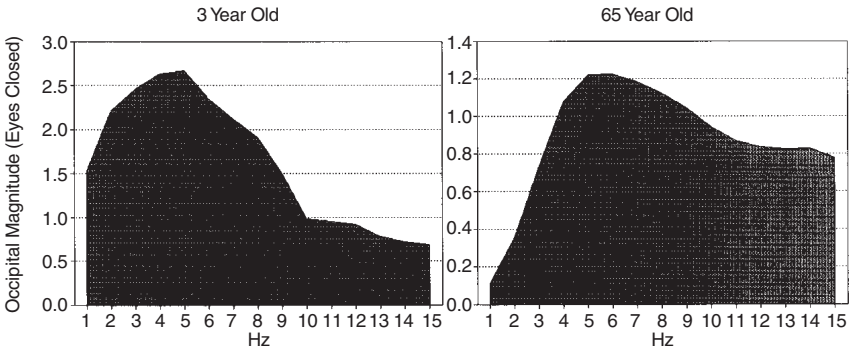
SENSORIMOTOR RHYTHM

The sensorimotor rhythm (SMR) is of obvious interest to neurofeedback clinicians, as much of the training revolves around this band. Unfortunately, SMR in humans rarely dominates the raw signal during wakefulness; therefore, it is very difficult to isolate this rhythm for spectral analysis. Fortunately for our purposes, the mechanism responsible for rhythmic activity of sensorimotor cortex during wakefulness (SMR) also generates the spindle burst pattern during sleep (Sterman &

TABLE 1. Maturation of the Alpha Rhythm: Minumum, Maximum Alpha Frequency Per Age Group

Age	Newborn	Infant	Toddler	Preschooler	Preteen
Alpha Rhythm	Not present	4 Hz, 6 Hz	5 Hz, 8 Hz	7 Hz, 9 Hz	9 Hz, 10 Hz

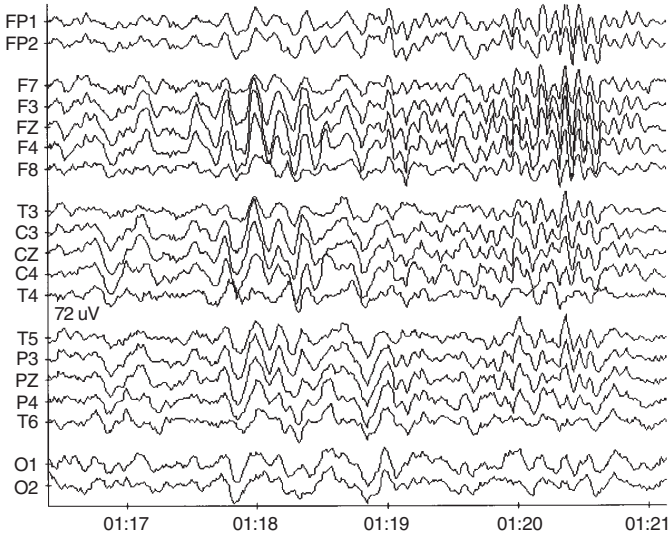
FIGURE 5. Occipital Alpha Distribution During Eyes Closed Rest in a 3-Year-Old Child and 65-Year-Old Chronic Alcoholic.



Bowersox, 1981). Put simply, the sleep spindle rhythm is the nighttime version of SMR. And it is easy to isolate and analyze sleep spindles in human EEG. Unlike the alpha rhythm, sleep spindles emerge fully formed in terms of frequency during early infancy. At three to four months of age sleep spindles are already at their adult speed, 12 to 15 Hz. A slower spindle (10 Hz) is rarely observed in healthy adults (Jankel & Niedermeyer, 1985) and may be indicative of cerebral pathology. That a band (spindle/SMR) should mature so rapidly and play such a significant role in neuroregulatory function is intriguing. (It makes me wonder whether there is a larger story here, especially in the context of our evolutionary history. Does the sensorimotor rhythm mature so quickly in all species, or only in primates, or only in humans?)

A drowsy child and a single active electrode positioned at recording site Cz or Fz is all one needs to accurately identify spindle frequency. Figure 6 shows sleep spindles recorded from a child diagnosed with autistic disorder. The spindle bursts range from 10 to 12 Hz, which is slower than normal. It may be the case that this slowing reflects the behavior of a dysfunctional mechanism, one that underlies his condition. In other words, a slow spindle frequency may be indicative of autism, or his specific subtype of autism, or an idiopathic process in this child, or even a poorly documented normal variant in children. There is little to no direct research on sleep spindles in autism so it's not clear whether this finding is relevant. Some researchers have observed a slower sleep spindle, but rarely in healthy adults (Jankel & Niedermeyer, 1985). Years ago some thought hyperactivity and sleep spindles incidence

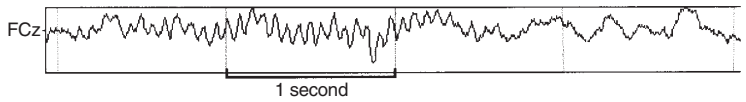
FIGURE 6. Five Seconds of EEG During Stage 2 Sleep in a Three-Year Old Boy with Autism. Note the 10-12 Hz Spindle Activity in Anterior Channels, Along with Earlier Slow Theta-Range Activity.



were associated, but this fell apart (Kiesow & Surwillo, 1987). Spindle frequency itself has not been thoroughly investigated in its relationship to clinical diagnoses. Slow spindle frequency may signify cerebral disorganization well before any behavioral symptoms emerge. Such a marker would be especially helpful for autistic children as this disorder can go undiagnosed for years. Obtaining a diagnosis at three or four months would allow treatment to begin incredibly early compared to current practices.

These speculations are derived from a single subject, but in physiology sometimes a single case makes your case (e.g., Morruzzi & Magoun, 1949). If autistics do manifest slower spindle frequencies, I foresee a parade of concerned parents taking their three- and four-month-old infants to neurofeedback clinics throughout the country for a neuroregulatory checkup. At the very least, it may verify whether regulatory features of an infant's motor system are intact. And unlike most sleep recordings, which require a night of adjustment, what we are looking for (the sleep spindle rhythm) can be acquired probably from the first hour of the first night. Figure 7 shows this idea put into practice: the

FIGURE 7. Single-Channel Recording During Sleep of 4-Month-Old Infant at Site FCz Shows Normal Spindle Frequency.



clinical assessment of my four-month-old son's spindle rhythm. As I was happy to see, it is well within the normal range. Only more research will determine whether this proves that he is not autistic, will not develop autism, or whether I simply wasted my time. But I do know at this point that his cerebral organization differs significantly from the three-year old autistic in Figure 6.

CUSTOMIZING THETA AND BEYOND

Alpha and sensorimotor rhythms dominate the raw EEG during specific conditions (eyes closed rest, stage two sleep, respectively), but can we say the same for theta and beta and other frequencies of interest? Sterman, Kaiser, and Veigel (1996) observed systematic bursts of theta in frontal sites during a continuous performance task at distinct times during a response. In fact theta synchronization may be necessary for some cognitive performance (Doppelmayr, Klimesch, Schwaiger, Auinger & Winkler, 1998b), but it may not be easy to elicit theta bursts consistently across subjects. Yet it's a start at identifying an ITF (individual theta frequency).

Statistical descriptions are effective tools, but rarely are they as powerful and as accurate as an individual's data. The use of customized frequency bands may optimize QEEG evaluation and neurofeedback training or it may add little to the picture. Only more research will tell.

REFERENCES

Berger, H. (1929). On the electroencephalogram of man. *Journal fur Psychologie und Neurologie*, *40*, 160-179.

Doppelmayr, M., Klimesch, W., Pachinger, T., & Ripper, B. (1998). Individual differences in brain dynamics: Important implications for the calculation of event-related band power. *Biological Cybernetics*, *79*, 49-57.

Doppelmayr, M., Klimesch, W., Schwaiger, J., Auinger, P., & Winkler, T. (1998). Theta synchronization in the human EEG and episodic retrieval. *Neuroscience Letters*, *257*, 41-44.

Etevenon, P., Eustache, F., Mitermite, F., & Lepaisant, J. (1990). EEG cartography before and after visual stimulation: A group study with descriptive non-parametric analysis of EEG data. *Brain Topography*, *3*, 13-19.

Harmony, T., Marosi, E., Becker, J., Rodriguez, M., Reyes, A., Fernandez, T., Silva, J., & Bernal, J., (1995). Longitudinal quantitative EEG study of children with different performances on a reading-writing test. *Electroencephalography and Clinical Neurophysiology*, *95*, 426-433.

Jankel, W. R., & Niedermeyer, E. (1985). Sleep spindles. *Journal of Clinical Neurophysiology*, *2*, 1-35.

Kiesow, N. A. & Surwillo, W. W. (1987). Sleep spindles in the EEGs of hyperactive children. *Psychological Reports*, *60*, 139-144.

Klimesch, W., Schimke, H., & Pfurtscheller, G. (1993). Alpha frequency, cognitive load and memory performance. *Brain Topography*, *5*, 241-251.

Lazzaro, I., Gordon, E., Whitmont, S., Plahn, M., Li, W., Clarke, S., Dosen, A., & Meares, R. (1998). Quantified EEG activity in adolescent attention deficit hyperactivity disorder. *Clinical Electroencephalography*, *29*, 37-42.

Morruzzi, G. & Magoun, H. W. (1949). Brain stem reticular formation and activation of the EEG. *Electroencephalography and Clinical Neurophysiology*, *1*, 455-473.

Niedermeyer, E. (1999). Maturation of the EEG: Development of waking and sleep patterns. In E. Niedermeyer and F. Lopes Da Silva (Eds.), *Electroencephalography: Basic principles, clinical applications, and related fields* (4th Ed.) New York: Lippincott, Williams & Wilkins.

Ray, W. J., & Cole, H. W. (1985). EEG alpha activity reflects attentional demands, and beta activity reflects emotional and cognitive demands. *Science*, *228,* 750- 752.

Sterman, M. B., Kaiser, D. A., & Veigel, B. (1996). Spectral analysis of event-related EEG responses during short-term memory performance. *Brain Topography*, *9*, 21-30.

Sterman, M. B., & Bowersox, S. S. (1981). Sensorimotor electroencephalogram rhythmic activity: A functional gate mechanism. *Sleep*, *4*, 408-22.