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Reading Task and Lambda EEG Activity

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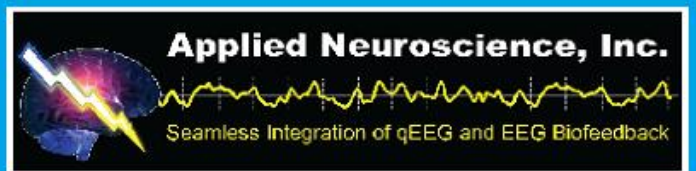
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Technical Notes:

Reading Task and Lambda EEG Activity

Jay Gunkelmen, QEEGT

Since 1994, when qEEG was declared ready for clinical application by the American Medical EEG Association, many Neurologists, Psychiatrists and Psychologists have begun using the qEEG when evaluating brain function. Most evaluations are recorded during the eyes closed waking state. Evaluating the brain under task, such as mental math or reading, adds significantly to the evaluation. Task dependent slowing reflects the strain from the stress of the task and the higher alpha to lower alpha ratio signals level of vigilance shifts (F. Schober et al., *Neuropsychobiology* 1995;31:98-112).

Understanding activity in qEEG mapping requires a strong background in EEG as well as technical skill selecting clean data. The sampling of transients and state changes violates the fast fourier transform (FFT) assumptions. Even the cleanest recording still requires a depth of experience with EEG. Not all slowing represents the strain from a task, the slower frequency normal variants are not strain.

There are normal variants in EEG which are commonly seen in testing (Mu, psychomotor variants, low voltage fast variant, frontal midline theta, hypnogogic hypersynchrony, fusiform alpha, etc. The proper interpretation of qEEG requires knowledge of these findings and their appearance in the mapping. Lambda, a common finding, is one such normal variant.

Lambda was originally described in 1951 by Y. Gastaut, and perhaps earlier by Evans (1949). Lambda was extensively reviewed by Chartrain (1976). It is most prevalent in children, waning following puberty, eventually seen in only 36% of the 31-50 year age range (Tsai and Liu, 1965). There is a variant of lambda seen in 1-3 year olds associated with eye blinks as well (Westmoreland and Sharbrough, 1975).

Lambda's morphology is biphasic and occasionally triphasic, and has a prominent positive

phase at 100 milliseconds. The period of the entire waveform is 200-300 milliseconds and it is commonly seen in repetitive trains with a period of 200-500 milliseconds and an occipital "sharp" positivity. The marked similarity morphologically to the VEP has been remarked on by many (Remond et al., 1965; Lesevre, 1967).

The proper interpretation of lambda's mapped image will depend on the identification of lambda in the raw EEG. The discrimination between lambda and other slow activity based on the mapping is not possible. This differentiation will require visual discrimination of the raw waveform based on pattern recognition.

The appended images are examples of the morphology and distribution of the waveforms as well as the mapping of these various presentations.

The morphology and mapping is displayed with a variety of referencing techniques to show the various appearances seen clinically with different montages.

When seen as an intermittent isolated waveform and mapped, lambda is seen as "theta" (5-6 Hz.) focal to the occipital poles. It may appear unilaterally or bisynchronously as isolated waveforms, or in periodic trains. This periodic repetition of the waveform is noted during saccadic scanning of the visual field, when looking at objects or text.

Unlike the common 5-6 Hz occipital focus seen with intermittent lambda, the periodic repetition of the lambda causes a much broader frequency range of activity, from 2-6 or 7 Hz., occipitally focal findings when mapped.

Lambda is elicited when an image is focused on the retina during visual scanning, eyes open. The task of reading most frequently will elicit lambda, as would any visual scanning task. There is a 67-85 millisecond delay (mean = 78) between the ocular fixation and the occurrence of the

lambda reported by Neidermeyer (see any edition of *Electroencephalography: Basic Principles, Clinical Applications and Related Fields*). The novelty of the stimuli increases the amplitude of the lambda.

Lambda may represent the occurrence of a singular visual evoked potential (VEP), as the location, morphology and periodicity would attest. The depth electrode studies by Perez-Borja et al. (1962) showed multifocal lambda in the calcarian fissure area which bisects the occipital lobe laterally. This area receives the primary visual path input cortically from the lateral geniculate of the thalamus.

This same waveform may be seen with eyes closed during visual imagery as "lambdoid" activity. The lambdoid activity would not be expected to have a retinal genesis, though the lateral geniculate of the thalamus is likely involved.

Lambdoid activity is seen in creative adults who use imagery with eyes closed (Julian Isaacs, Ph.D., personal communication, 1985), though it was not seen during hypnotic viewing of a picture in the subjects "mind's eye", as reported in Neidermeyer.

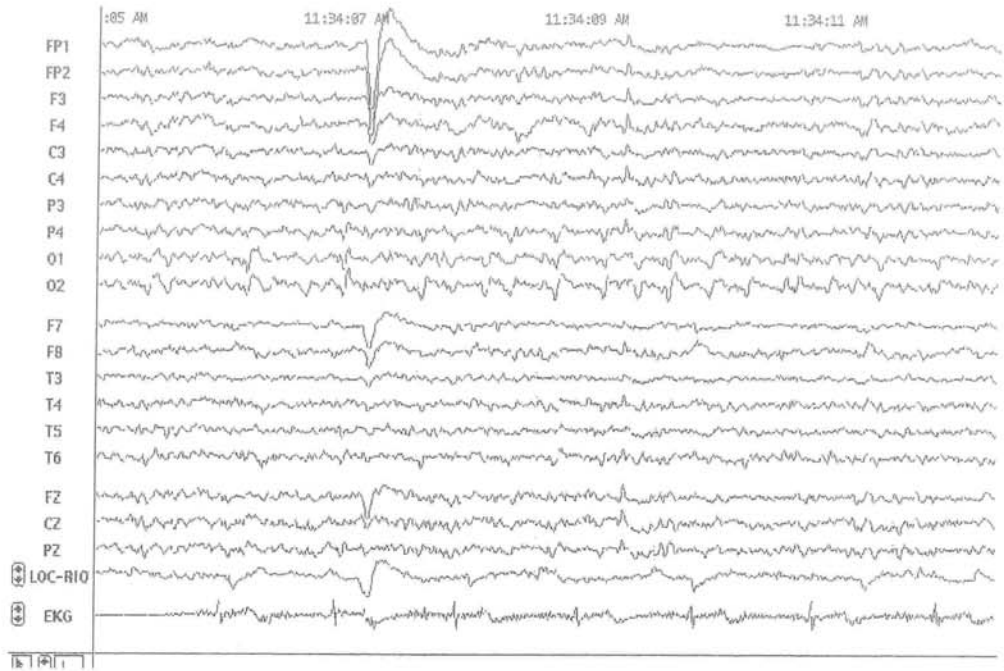
Positive Occipital Sharp Transients of Sleep (POSTS) is yet another name for activity with lambda's same morphology, periodicity, polarity and distribution. The true singular nature of these various terms is discussed by Neidermeyer.

The qEEG containing lambda mapping with posterior focal slowing in the theta (isolated) or theta and delta (repetitive periodic) presentations are normal. The same frequencies may also indicate pathology or dysfunction if the source is not lambda.

The images used in most texts on EEG are too small to give the real image of lambda, but the following images and maps may assist in the pattern recognition required for documentation of lambda in a clinical or research setting.

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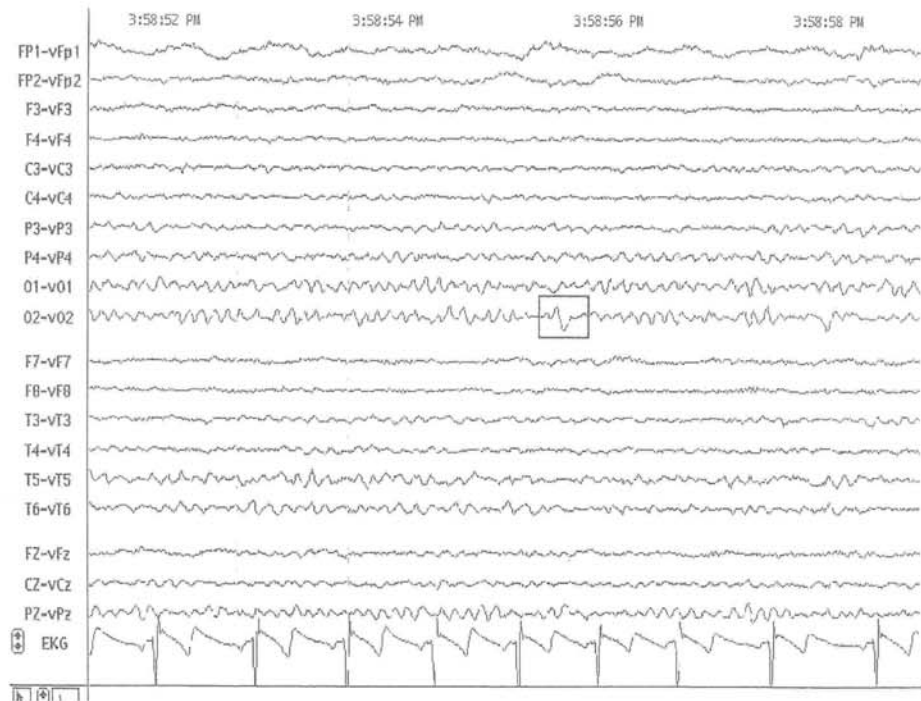
Figure 1: Lambda in an adult EEG during reading



Note the occipital positive (downward) sharp deflection seen repetitively

Figure 4: Lambdoid activity, Eyes Closed EEG

Hjorth montage, also called laplacian, source derivation, local average and 'virtual' reference



Lambda in an adult reading

Figure 2: Topographic Mapping of Spectral Analysis (Reading)

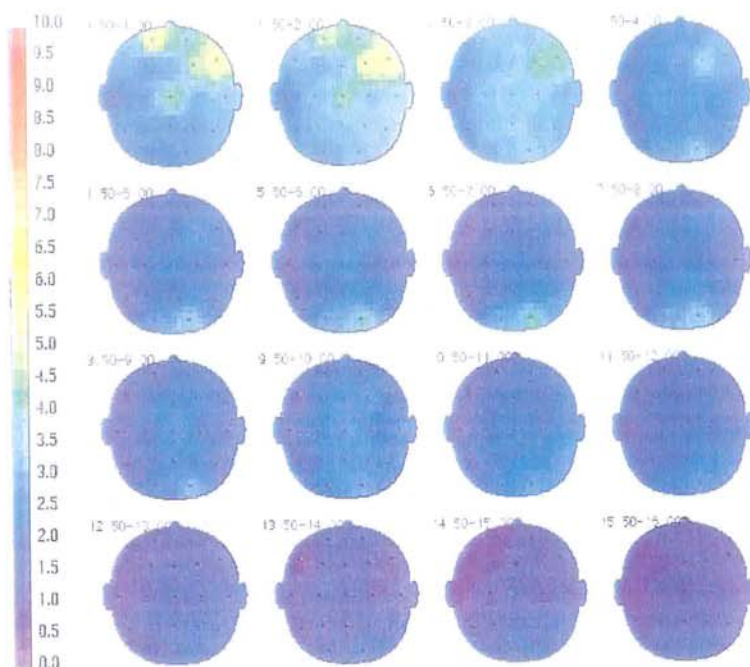
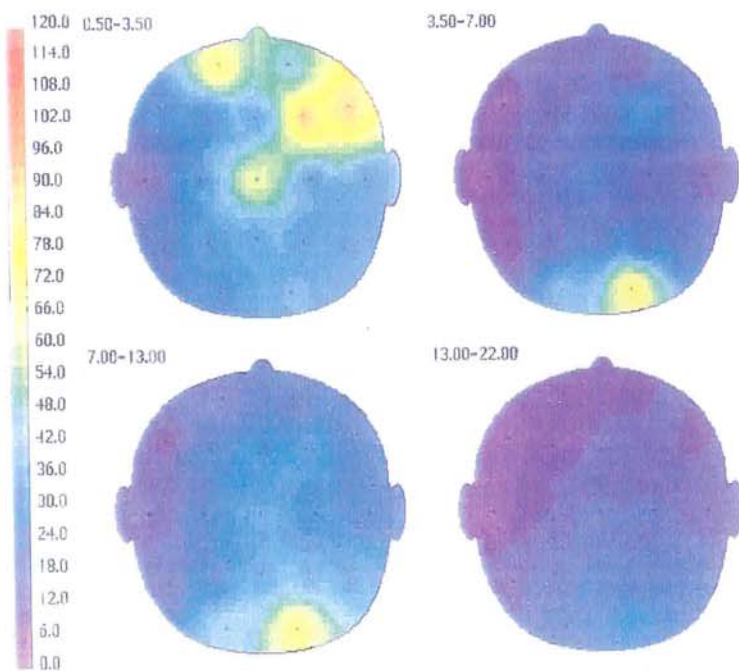


Figure 3: Topographic Mapping of Spectral Analysis (Reading)



Lambda in QEEG mapping
Figure 7: Hjorth maps

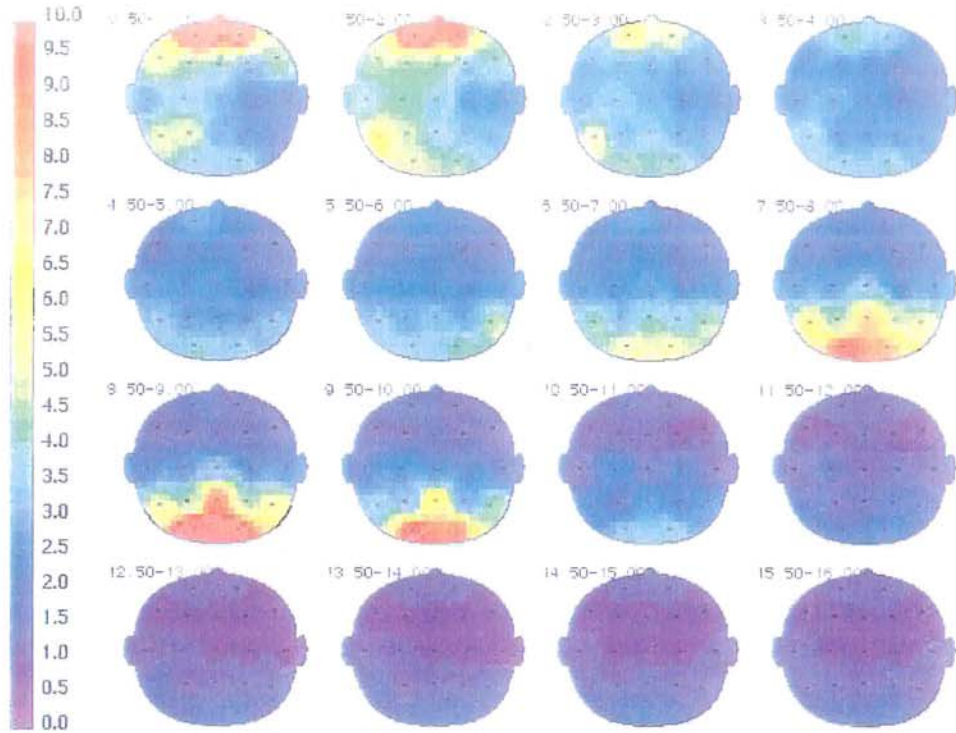
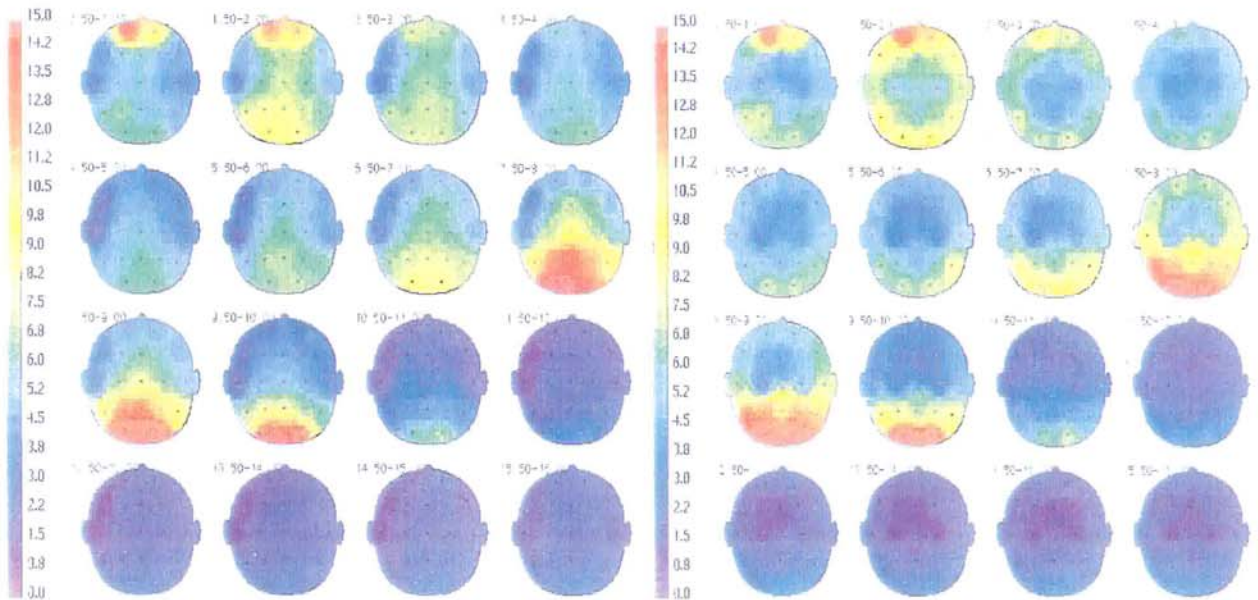


Figure 8: linked car (left) and Cz (right) maps



Lambda in QEEG mapping
Figure 5: linked ears (EYES CLOSED)

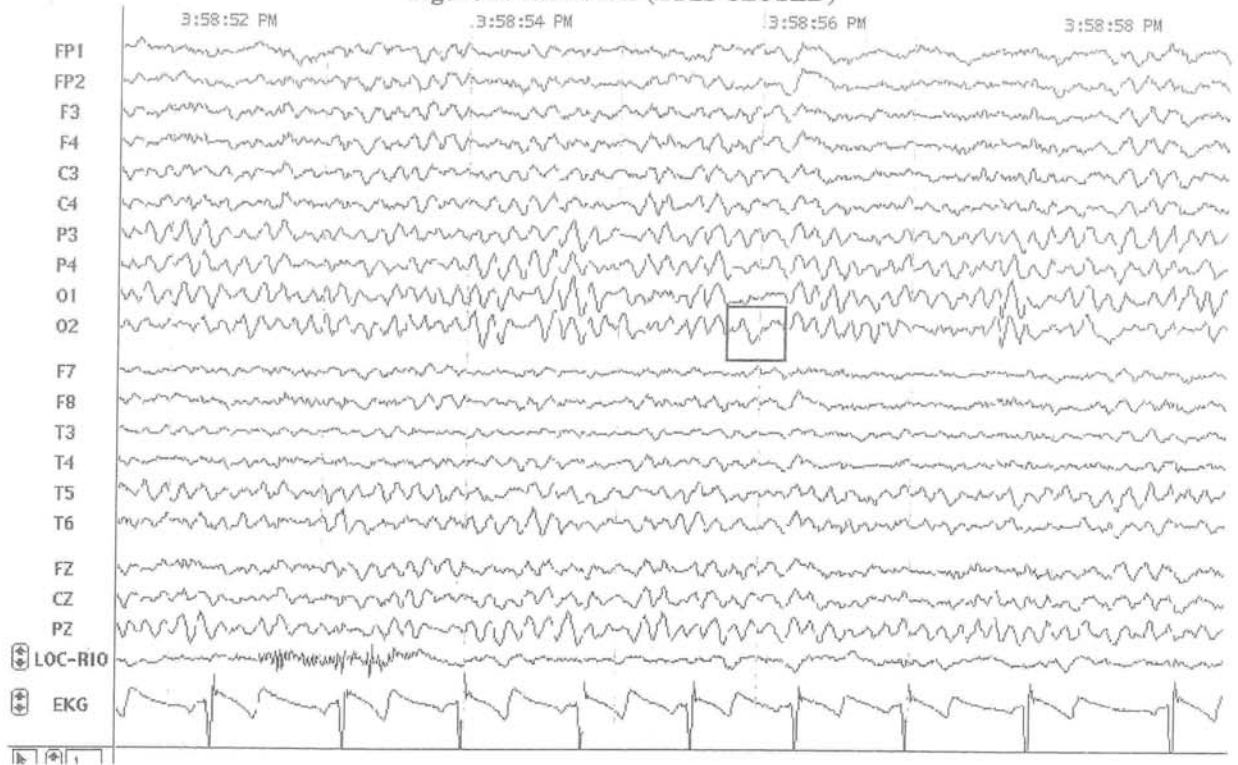
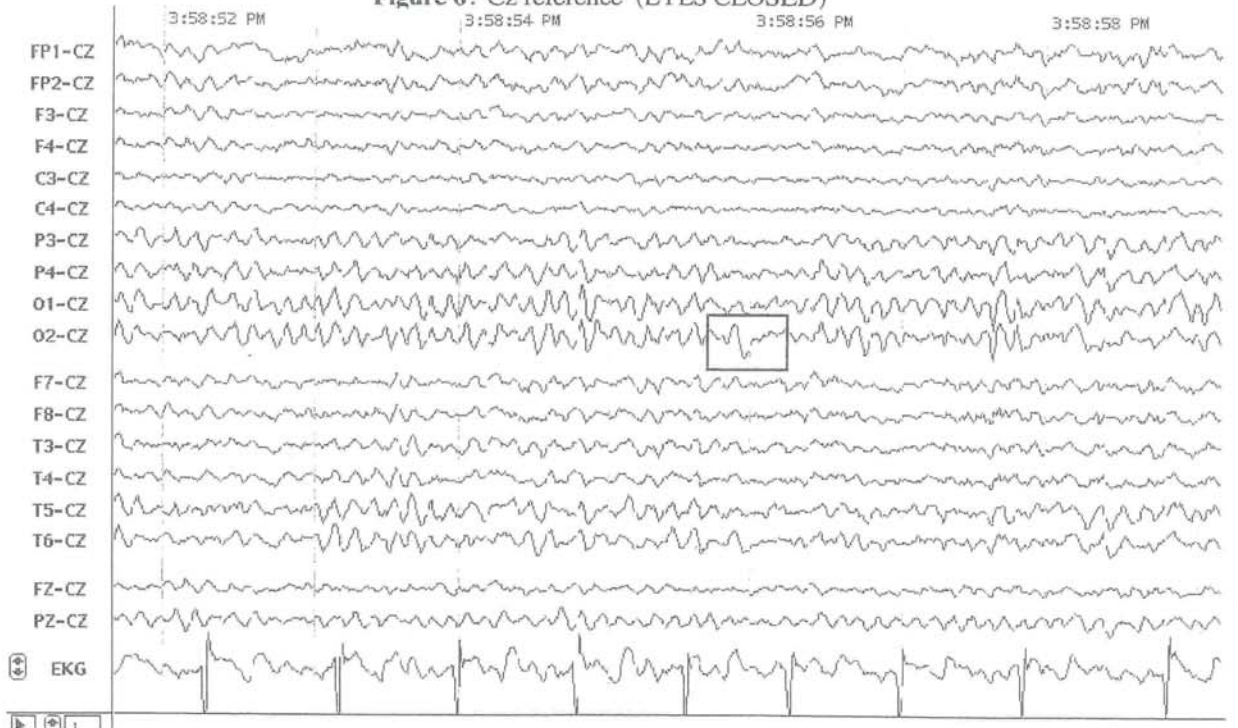


Figure 6: Cz reference (EYES CLOSED)



News From Other Journals and Websites:
(Continued from page 34).

problems.” Thatcher and his colleagues counter: “ ‘Others’ were not identified, and there were no citations by AAN/ACNS to scientific evidence that refutes or contradicts the findings of Thatcher et al. or Tebano et al. It would appear that the AAN/ACNS paper arbitrarily discounted, without scientific justification and only by reference to anonymous ‘others,’ at least three well-controlled studies including one that involved 608 mild TBI patients and 103 age-matched controls with independent cross-validations. This conclusion was supported by sworn statements that the chief author of the AAN/ACNS report made in a 1998 civil deposition in which he was unable to identify or recall under oath who the alleged ‘others’ were or whether they told him this verbally or in writing” (p. 95). Due to the seriousness of the statements about anonymous “others,” the court held that the AAN/ACNS statement cited at the beginning of this paragraph and all similar references to anonymous “others” had to be omitted from the trial.

Thatcher et al. (1999) provide a helpful review of the QEEG literature relevant to TBI, citing a sensitivity of 96.59%, a specificity of 89.15%, a positive predictive value of 93.6%, and a negative predictive value of 97.4%—sensitivities and specificities comparable to MRI’s, sonograms, blood tests, and other diagnostic procedures in medicine. Evidence is cited for the test-retest reliability of QEEG: 82% for 20 second samples, 90% for 40 second samples, 92% for 60 second samples, and that QEEG data analyzed by three different individuals has been found to still be highly reliable. It is noted that the Nuwer paper did not cite a single study supporting their stance of questioning the reliability of QEEG. The scientific literature is judged to be sufficient for QEEG data to meet Frye and Daubert rules on admissibility in the courtroom and to be useful in evaluating the one to two million Americans who experience a traumatic brain injury yearly.

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(Review by D. Corydon Hammond, Ph.D.)

Hoffman, D.A., Lubar, J.F., Thatcher, R.W., Sterman, W.B., Rosenfeld, P.J., Striefel, S., Trudeau, D.L., Stockdale, S. Limitations of the American Academy of Neurology and American Clinical Neurophysiology Society paper on QEEG. *Journal of Neuropsychiatry and Clinical Neurosciences*. 1999; 11: 401-407.

Three significant papers have appeared in the last several months affirming the role of QEEG in behavioral science and medicine. Elsewhere in this section, Cory Hammond Ph.D. comments on the report of the EEG and Clinical Neuroscience Society¹, and Ted LaVaque Ph.D. reports on the review of conventional and quantitative electroencephalography by Hughes and John². This most recent report, by a committee of experts from various disciplines and societies, was primarily authored by Daniel Hoffman, M.D. Once again, the application of the rating system proposed by the American Academy of Neurology and American Clinical Neurophysiology Society (AAN/ACNS)³ is taken to task in terms of the ratings the society gives to certain clinical applications of QEEG. (This rating system is detailed in LaVaque’s summary and review of the Hughes and John paper.)

The lack of scientific foundation in the AAN/ACNS paper is noted for excluding: (1) traumatic brain injury, (2) psychiatric disorders, including learning disabilities, and (3) medico-legal uses of QEEG.

The AAN/ACNS position is criticized as wrought with problems of bias and misrepresentation. For example, the demonstrated cross correlation of EEG findings with MRI findings and the demonstrated sensitivity and specificity of discriminant analysis of QEEG are not cited in the AAN/ACNS report, which is seen as skewed and misrepresentational in omitting these well documented phenomena. Other important scientific data omitted from the initial AAN/ACNS report are cited in regards to the established utility of QEEG in seizure disorder, mild traumatic brain injury, and attention deficit disorders. “QEEG allows one to see attributes of brain function that cannot be seen in the raw EEG signal,” in

a highly standardized and reproducible manner in these conditions.

While this paper finds the AAN/ACNS report to be biased, and certainly not the definitive opinion to be applied in health care decisions, there is still some common ground. Both the authors and the AAN/ACNS report agree on numerous quality issues related to certification and utilization standards. Both agree that substantially more than QEEG is needed for diagnosis and that "both neurologists and non neurologists who use QEEG need to be trained and competent in its inherent complexities."⁴ The report stresses that adequately trained non-physicians have made the most extensive and accurate descriptions of normal human QEEG, and are often the teachers of neurologists and other physicians.

In summary, this paper finds that "the AAN/ACNS report is misleadingly negative regarding the current status of quantitative EEG and tends to discourage its development and use with other related clinical problems." The AAN/ACNS position should be considered one side of an opinionated debate, not the final word. "Too many implications for health care are at stake. The debate and research may continue without withholding valuable help from the public."

The paper, copyrighted by American Psychiatric Press, Inc., can be found online at neuropsychiatry.online.org. Correspondence should be addressed to Dr. Hoffman, Neuro-Therapy Clinic, P.C., 8200 Belleview Avenue, Suite 600E, Englewood, CO 80111.

(Review by David L. Trudeau, M.D.)

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Websites:

David Kaiser website offers weekly e-literature surveys:
<http://www.thegrid.net/dakaiser/> or
<http://www.eegspectrum.com>

David Kaiser, Ph.D. is doing an outstanding job of reviewing the electronic literature that may be of interest to neurotherapists in general, and specifically those therapists working with addictive disorders and attention deficit disorders. His websites are to be found at <http://www.thegrid.net/dakaiser/> and <http://www.eegspectrum.com>. Complete directions for signing on to focal e-mail lists can be found here. Neurofeedback news alert offers top stories and book reviews, with lists of web resources for articles and features. A monthly feature, What's 3New in Neurofeedback, is a newsletter sponsored by EEG Spectrum, Inc., available at www.eegspectrum.com/newsletter/. CD alert covers the electronic resources for chemical dependency on a regular basis.

(Review by David L. Trudeau, M.D.)

Camp, Bonnie W. "Studies of QEEG and Attention Deficit Hyperactivity Disorder," June 24, 1999. (University of Colorado Website. <http://www.uchsc.edu/sm/jfk/bwcweb.htm>) With the advent of the World Wide Web, publishing of new scientific reports on

private or university websites prior to peer review and publication in traditional journals is increasing. The Web surfer should be very careful evaluating non-peer reviewed material. The Bonnie Camp article is an interesting example of a report that includes some very nice design elements, provides some further positive results of neurotherapy for ADHD, but unfortunately includes statements about quantitative EEG that are inaccurate and potentially misleading to technically unsophisticated readers.

Camp discusses the fact that she was unable to find consistent EEG differences between ADHD and a "normal" group of children. This is not surprising. The recording site Camp used is not by itself capable of such discriminations. She was using a single channel commercial neurofeedback instrument to perform EEG amplitude measurements at the "central sulcus" (presumably at or near Cz). Although Camp mentions at the beginning of her article that "QEEG evaluations commonly include total brain mapping," she then goes on to call what she did QEEG. Quantitative electroencephalography really must include a minimum of 16 scalp channels and, more commonly today, 19 channels plus ground, linked ear references and one or two artifact channels. Electroencephalographers are also recognizing the need for using more than one montage, e.g., referential, averaged reference, etc. to examine data. To call single channel recording QEEG just because it digitizes a signal is to risk misrepresenting facts to a public that may or may not understand the technical issues.

Camp also takes issue with E. Roy John's normative database, claiming it has too few participants at particular ages. The sample size issue is a mathematical one and deserves further discussion, which I will defer to Dr. John. Camp cites John et al. articles from 1977 and 1980. Recent articles from John's group replicate excellent discrimination of normals versus ADHD. Chabot and Serfontein (1996a), for example, compared the QEEGs of 407 ADHD and 310 normal children and reported high sensitivity (93.7%) and specificity (88%). Those numbers empirically make the sample size discussion moot. ADHD is character-

ized by excess frontal absolute and relative theta power, smaller elevations in alpha relative power and decreases in alpha and beta mean frequency. Further, ADHD is distinguished from normal by a range of coherence abnormalities. Lower IQ was associated with greater excesses of theta and alpha and lower coherence. Excess theta or alpha and other QEEG variables have been used successfully to discriminate ADHD from normal by Mann et al. (1992), Valdizan & Andreu (1993), Matsuura et al. (1993) and Lazarro et al. (1998). Camp's comment that "...QEEG may not be that useful in differential diagnosis..." is a serious misstatement of scientific fact.

Camp also refers to "the debate over whether there is a difference between children with learning disability and attention deficit" suggesting further research is needed. I refer the interested reader to the work of Chabot (1996b) at NYU differentiating LD from ADHD and defining multiple subtypes of ADHD. This group and the research of Suffin and Emory (1995) has shown that there is differential responsiveness to specific medications as a function of QEEG subtype. Research indicates one can discriminate between children who will respond best to Ritalin versus Dexedrine versus SSRIs and avoid the negative side effects that occur when the wrong medicine is prescribed.

The author's desire to simplify QEEG diagnostics appears well-intentioned (to bring the technology into schools at the lowest cost), but this is not a simple technology. If QEEG is to be used effectively to aid diagnosis, it must be used at the highest standards. Further, QEEG offers the possibility of guiding the choice of training sites and protocols in a rational way by demonstrating what regions of brain actually exhibit abnormalities of amplitude, coherence or phase.

On the positive side, the treatment phase of Camp's work appears to be a well-designed replication of positive results from protocol-guided neurotherapy for ADHD. The study is interesting and important since it uses a cognitive behavior modification control group and two experimental groups comparing referential and bipolar recording. As far as I am aware this is the first comparison between the two montage types.

Apparently there were some differences between the effects of referential and bipolar montages. The sample sizes in the groups are small (N=16 approx.) and produced some significant results with the EEG groups showing more improvement than the CBM group on behavior checklists, the TOVA and some other cognitive tests.

Importantly, the CBM group did show improvements on most measures and did better than the EEG group on some. This is confirmation for the concept many of us have been using (following Lubar) of integrating cognitive tasks with neurofeedback training. The statement that the CBM group showed more beta and lower T/B ratios "in the learning analysis" is unclear, but very interesting and deserves elaboration. The author mentions her impression that many of the children in each group might have done better with a combination of treatments and an individualized approach not possible in a research paradigm. That is a much appreciated acknowledgement of the limitations of formal research and the potential for neurotherapy and cognitive behavior modifications in clinic settings. I hope Dr. Camp will quickly submit her research and thoughts on neurotherapy for peer review and publication, as there are significant findings here.

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(Review by John K. Nash, Ph.D.)

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