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Temporal Lobes and Their Importance in Neurofeedback

D. Corydon Hammond PhD [a]

[a] University of Utah School of Medicine , PM&R, 30 North 1900 East, Salt Lake City, UT, 84132
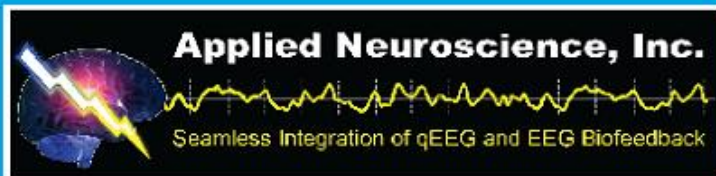E-mail:
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Temporal Lobes and Their Importance in Neurofeedback

D. Corydon Hammond, PhD

ABSTRACT. A review is presented on temporal lobe function and various conditions that are associated with the temporal lobes that have implications for both careful assessment and neurofeedback.

Margaret Ayers (1999) usually begins neurofeedback work by doing sequential (bipolar) training at T3-C3 and T4-C4, only turning to training elsewhere when the EEG appears normalized in these areas. However, in general in our field, it is my opinion that the temporal lobes often do not receive the attention they deserve from many neurofeedback practitioners. This may particularly be the case because most neurofeedback practitioners use ear references during neurofeedback and our QEEG databases have also used linked ear references. These are not the best montages for identifying temporal lobe problems, but there is a database (Thatcher, Walker, Biver, North, & Curtin, 2003) that provides Z-score norms for a Laplacian montage and for an average reference montage. This allows QEEG assessment to provide better localization and a much better evaluation of potential temporal lobe pathology. The Nx Link (NYU) database (John, Prichep, Fridman, & Easton, 1988) also provides us with valuable normative data on the mean frequency at various electrode sites, including temporal sites. So that we may address these con-

D. Corydon Hammond is affiliated with the University of Utah School of Medicine, PM&R, 30 North 1900 East, Salt Lake City, UT 84132 (E-mail: D.C.Hammond@ m.cc.utah.edu).

cerns, in the remainder of this article I will review some basic information about temporal lobe function and some of the problems associated at least in part with the temporal lobes.

The dominant temporal lobe (which is the left hemisphere in 95% of right-handed patients and 70% of left-handed patients) is associated with language comprehension, verbal memory, and word perception. The temporal lobe in the nondominant hemisphere is associated with sound modulation, music perception, loudness, tones, recognizing and remembering spatial patterns, faces, and nonverbal material (Joseph, 1996). The temporal lobes are richly connected with all the other cortical areas and are involved in integrating information associated with emotion and motivation.

The T3 electrode site is in the middle and more superior part of the temporal lobe, around Brodmann areas 22 and 21. In a right-handed person (and left-handed individuals when this is still their dominant hemisphere), this is an area involved with memory for verbal material and initial memory storage. As you will see in studies cited later, we can anticipate it being very involved in reading performance. The T5 electrode site falls in Brodmann area 37. It lies just behind Wernicke's area and near the bottom edge of the angular gyrus-both of which are involved with reading and expressive speech. Wernicke's area is at the back end of the superior temporal area and is associated with memories for sequences of sounds that make up words. The angular gyrus is more associated with attaching meaning to words (Pliszka, 2003). Appendix 1 illustrates the estimated location of common electrode sites in relation to areas of the brain.

In order to help further orient readers, if you think about the placement of the T3 and T5 electrode sites on the illustration in Appendix 1, and go inward a little deeper toward the center of the brain and just a little lower, this is where you find the hippocampus and amygdala. The anterior tip of the temporal lobe is associated with the meaning of words for people, while nouns related to animals are associated with the mid-temporal area, and the posterior temporal lobe is associated with words for tools. Performance of a working memory task with novel complex pictures activated medial temporal lobe structures more than familiar complex pictures (Stern, Sherman, Kirchhoff, & Hasselmo, 2001). In contrast, a working memory task with highly familiar pictures resulted in greater prefrontal activation. These results are consistent with the hypothesis that the medial temporal lobe is involved in the short-term maintenance of information that has no prior representation in the brain, whereas the prefrontal

cortex is more important in monitoring familiar stimuli that have a high degree of interference.

Thus, problems with word finding for only a particular category (e.g., the name of a person versus an object) may be associated with a lesion in a particular area. Wernicke's area sends out information to language areas behind and above it and also through the arcuate fasciculus to Broca's expressive speech area, which is also involved in understanding more complex sentences (Pliszka, 2003). The anterior temporal pole is considered part of the limbic association cortex and is involved with emotional behavior and memory storage. In the right temporal area, Arruda, Walker, Weiler, and Valentino (1999) found that T4 and T6 are importantly involved with vigilance and sustained attention. In their research, when there was less beta activity at T4 and T6, lower performance was found on continuous performance tests.

When we look at quantitative EEG and neuroimaging research, we find that the temporal lobes are often involved in many of the problems that we work with in neurofeedback, although certainly many, if not most conditions (e.g., Alzheimer's, autism, schizophrenia) clearly involve more than just one area of the brain. Although a comprehensive review of this voluminous literature is certainly prohibitive, in the remainder of this paper I will provide a brief overview.

AGING AND DEMENTIA

Many of us who conduct neurofeedback training are middle-aged or, to perhaps put it more delicately, post-middle-aged. The comments in the earlier article in this issue by Hughes about deterioration in the temporal lobes are particularly relevant to us and our older patients. Temporal slow waves are common in the elderly (Busse, Barnes, Friedman, & Kelty, 1956; Torres, Faoro, Loewenson, & Johnson, 1983; Visser, Hooijer, & Jonker, 1987). Correlations have also been found between temporal slow waves and cerebrovascular disease (Asokan, Pareja, & Niedermeyer, 1987; Maynard & Hughes, 1984). Most recently, Inui, Motomura, Kaige, and Nomura (2001) examined the EEGs of healthy volunteers and patients with clinical histories. They particularly looked at the presence of temporal low voltage irregular delta waves (TLID), temporal minor slow and sharp activity (TMSSA), and bursts of rhythmical temporal theta (BORTT). These patterns were found to be significantly more common in patients with mild cerebrovascular diseases, particularly mild ischemic cerebrovascular disorders, and with aging. They also shared a

left-sided predominance (as previously found by Asokan et al., 1987; Maynard & Hughes, 1984), were correlated with each other, were not continuous, and often just lasted for one to two seconds. TLID has also been found to be associated with findings on the MRI of silent small multiple infarctions (Inui, Kawamoto, & Kawakita, 1994).

With regard to the Inui et al. (2001) study, I want to underscore that these slow waves were not continuous, but only brief and of low voltage. Thus, a QEEG may not display such an abnormality and they may only be picked up by careful examination of the raw waves, particularly utilizing remontaging. These authors explained:

> One of the reasons why examination of temporal slow waves has not received enough attention in spite of the clinical usefulness of this methodology seems to be a technical problem. Because these EEG findings propagate to the ear lobes and are often low in voltage, it is difficult to detect such changes with the standard monopolar recording techniques. For a clear demonstration of these EEG findings, the electrodes have to connect to the common average reference. (p. 530)

This underscores the importance of carefully examining the raw EEG of our patients and not relying solely on averaged QEEG data. An illustration of these points is seen in Figure 1 where you may examine how the raw waves for the exact same epochs, viewed side-by-side, appear quite different at mid-temporal sites (T3, T4) when they are viewed with a linked-ears montage versus a Laplacian current source density montage.

The temporal lobes are an area of the brain affected by Alzheimer's and also by frontotemporal dementia. In a recent prospective study, Nobili et al. (1999) found that in mild dementia patients the loss of activities of daily living was predicted by delta power on the left side at central-posterior temporal electrode sites, and incontinence was predicted by both alpha and delta on the right side. Even more recently, Nobili et al. (2001) found that regional cerebral blood flow in the posterior temporal-inferior parietal areas of both hemispheres provided prognostic information that was predictive (an average of two years later) of loss of activities of daily living, incontinence, and death due to end-stage Alzheimer's disease. Other studies have found reduced temporal lobe volume to be associated with Alzheimer's (Morys et al., 2002).

EPILEPSY

Often the temporal lobes are the site of epileptogenic activity (Hughes, 1994). This may involve not only spike-and-wave activity, but also focal sharp waves or spikes, and infrequently even temporal intermittent rhythmic delta activity (TIRDA; Normand, Wszolek, & Klass, 1995).

HEAD INJURY AND HYPOXIA

Patients with mild TBI and persistent postconcussive symptoms have a high incidence of medial temporal lobe injury (Umile, Sandel, Alavi, Terry, & Plotkin, 2002) and severe brain injuries have been found to most often involve the frontal and temporal lobes (Pierallini et al., 2000). Even mild hypoxia causes temporal lobe damage (Yonelinas et al., 2002).

MIGRAINES AND HEADACHES

Posterior temporal and occipital areas seem to frequently be involved with problems of migraine or headaches (e.g., Donati et al., 1990; Valdizan, Andreu, Almarcegui, & Olivito, 1994), often involving slow wave activity with a posterior alpha asymmetry.

CHRONIC FATIGUE SYNDROME

The temporal lobes are one of the areas implicated in chronic fatigue syndrome in multiple studies (Goldstein, Mena, Jouanne, & Lesser, 1995; Ichese, Salit, & Abbey, 1992; Schwartz et al., 1994).

MANIA

A temporal asymmetry seems to often be involved with bipolar disorder. Recent evidence (Small et al., 1998) with manic patients suggested that in the left temporal area (T1, T3), amplitudes (power) will be low in delta, theta, slow alpha, and in beta 1 bands. Similarly, Al-Mousawi et al. (1996), Gyulai et al. (1997), and O'Connell et al. (1995) found in PET and SPECT studies of bipolar disorder that there was increased

FIGURE 1. Temporal Activity in: (A) a Linked-Ears Montage, versus: (B) a Laplacian Current Source Density Montage
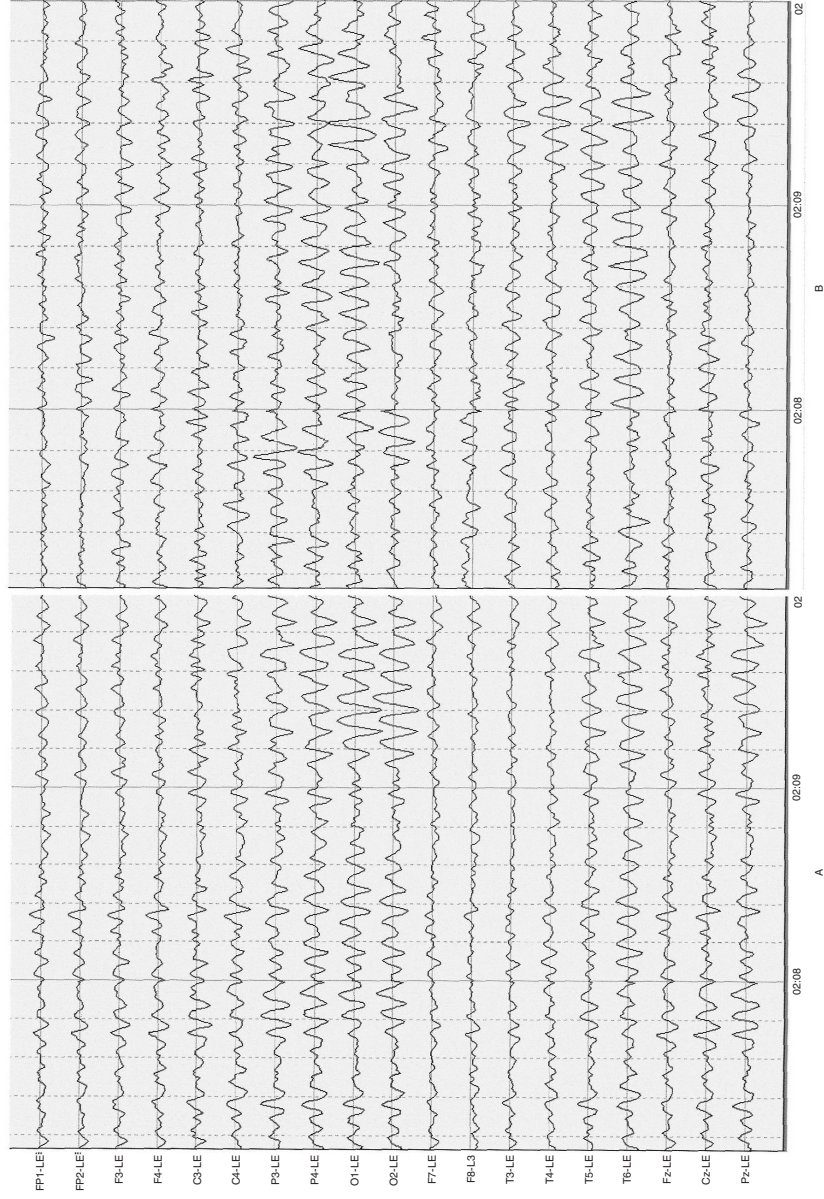
right temporal activity, and these increases correlated with mania scores (O'Connell et al., 1995).

Koek et al. (1999) used 12 bipolar patients as their own controls, performing repeated QEEGs over two years on them in different mood states. They discovered frontotemporal (F3-F4, T3-T4) asymmetry in slow wave activity, which became smaller as the speed increased from delta up through alpha. The asymmetry appeared in opposite directions in depression compared to mania/hypomania, but the nature of the change score did not permit determination of which hemisphere changed in which direction between mood states. Partially in contrast to the neuro-imaging studies cited above, Koles, Link, and Flor-Henry (1994) utilized a Cz reference and found that both schizophrenic and manic groups were characterized by hyperactivity in the left temporal region, while depressed patients displayed hyperactivity in the right temporal area, compared to control subjects. However, confirming Flor-Henry's earlier work (Flor-Henry and Koles, 1984), during verbal comparisons, the manic group displayed hyperactivity at T6.

PANIC DISORDER AND ANXIETY

It appears important to distinguish two types of anxiety (Heller, Etienne, & Miller, 1995; Heller, Nitschke, Etienne, & Miller, 1997). First, there is anxious arousal which is associated with physiological hyperarousal symptoms and somatic tension. This type of anxiety is associated with panic attacks, phobias, and evoked in high stress situations. Second, there is anxious apprehension which involves worrying and verbal rumination about the future. This type of anxiety is more associated with generalized anxiety disorder, obsessive compulsiveness, and trait anxiety as measured in questionnaires.

With panic disorder patients (with no depressive symptoms) compared to normal subjects, Wiedemann et al. (1999) found right frontal activation (reduced right frontal and increased left frontal alpha power) during eyes-open and eyes-closed conditions, and when exposed to anxiety-relevant stimuli. While Davidson, Ekman, Saron, Senulis, and Friesen's (1990) research showed that in depression the frontal asymmetry is more associated with decreased left frontal activation, the Wiedemann et al. study suggested that in panic disorder, the asymmetry is caused by increased right frontal hemisphere activation, which they perceived as an avoidance-withdrawal problem.

However, this does not appear to just involve the right frontal area. In the first study to apply quantitative volumetric methods to evaluate brain changes in panic disorder, Vythilingam et al. (2000) examined the volume of the temporal lobe and the hippocampus in patients with panic disorder and healthy control subjects using quantitative MRI measures. The mean volume of the left and right temporal lobes was significantly smaller in panic disorder compared to healthy subjects, despite the fact that they had normal hippocampal volume. More recently, Galderisi et al. (2001) examined the electrical microstates in panic disorder patients versus control subjects. Their findings indicated an overactivation of right temporal and right hemisphere networks involved in early visual processing and a hypoactivation of the right hemisphere circuits involved in the Late Positive Complex (which is an event-related potential component). The latter was correlated with a higher number of panic attacks. It was their conclusion that there was a pathogenetic role involving right temporo-hippocampal dysfunction in panic disorder.

Right temporal activation (Brodmann area 22; T4 electrode) has also been found in a study of memory-driven arousal of anxiety (Liotti et al., 2000). Liotti et al. (2000) further noted deactivation in Brodmann areas 20 (below T3), 37 (which is overlapped in part by electrode site T6), 7 (around electrode P4), 10 (Fp2), and 40 (near electrode site CP5). Concerning general anxiety, Isotani et al. (2001) used LORETA (low resolution electromagnetic tomography) for source localization of EEG activity during hypnotically elicited anxiety and relaxation in normal subjects. They found that during anxiety 18.5-21 Hz beta (and to a lesser extent 21.5-30 Hz beta) increased in Brodmann area 10 in the right hemisphere–the vicinity of electrode site Fp2. During relaxation, 8.5-10 Hz alpha increased over the right hemisphere. Congruent with these findings, PET and MRI studies of induced anxiety (Canli, Desmond, Zhao, Glover, & Gabrieli, 1998; Chua, Krams, Toni, Passingham, & Dolan, 1999; Dolan et al., 1996; George et al., 1995; Reivich, Alavi, & Gur, 1984) have found more right than left fronto-temporal activity. Results of neuroimaging studies (Naveteur, Roy, Ovelac, & Steinling, 1992; Stapleton, Morgan, & Liu, 1997) comparing individuals with high and low anxiety levels and a transcranial Doppler ultrasound study (Troisi et al., 1999) also support these findings.

STUTTERING

Individuals with stuttering problems have been found to display beta band hyper-reactivity, with the right temporal-parietal lobe region showing the greatest activity (Rastatter, Stuart, & Kalinowski, 1998). When they are under conditions of delayed auditory feedback and frequency-altered auditory feedback that improves the stuttering, they show a strong decrease in beta activity at posterior temporal-parietal electrode sites. Similarly, Fox et al. (2000) found that stuttering was associated with problems in the non-dominant hemisphere in the superior and middle temporal gyrus.

LANGUAGE IMPAIRMENT

Shafer et al. (2001) evaluated children with specific language impairment, which is a condition where the child lags behind peers in language acquisition, which contributes to learning and reading disabilities. The condition is present in 4 to 8 % of children. They examined event-related potentials time-locked to grammatical contexts. The left temporal cortex (T3) was found to be involved, with increased neural activity at T4 that seemed to be an effort to compensate for the reduced left temporal activity.

DYSLEXIA AND READING PROBLEMS

Ahissar et al. (2001) used magnetic EEG (MEG) to study speech comprehension, which was in the auditory cortex (Heschl's gyrus; Brodmann areas 41 and 42, just posterior to T3). This may be located by referring to Appendix 1. They found that the strongest response locking was at frequencies below 14 Hz. They further discovered that the average comprehension level of people correlated with the similarity that existed between the frequencies of the speech stimulus and the subject's cortical activity. "The frequency tuning of cortical neurons determines the upper limit of speech modulation rate than can be followed by an individual" (Ahissar et al., 2001, p. 13372). Their review concluded that the "decodable ranges" of speech modulations vary from one listener to another, and that poor readers have a slower and narrower range than good readers. Harmony et al. (1990) found that children with a reading disability had more absolute power delta (especially at T6) and theta (at P4 and T6), and more

relative power delta at F3, F7 and T3 electrodes, as well as lower alpha relative power.

Seki et al. (2001) used fMRI to study dyslexic readers compared with normal controls while they were reading and in comparison with a control task. All control subjects showed activation of the left middle temporal gyrus when they were reading, but in the dyslexic children the activation was rather vague. However, other distinctive areas were activated in the dyslexics, such as the occipital cortex, inferior frontal or precentral gyrus. These results suggested that the dyslexics were using compensatory processes to make up for their unskilled reading ability. The authors concluded that the results were similar to previous results in a Russian study, ". . . and suggest that brain malfunction in dyslexia during the task of reading must be common despite differences in languages" (p. 312).

Simos et al. (2002) have likewise implicated the left temporal area in reading problems. They found that children who, at the end of kindergarten, were found to be at risk for developing reading problems displayed markedly different activation profiles than children who had, at that stage, already mastered important pre-reading skills. The aberrant profile was characterized by the lack of engagement of the left-hemisphere superior temporal region (e.g., T3), an area normally involved in converting print into sound, and an increase in activation in the corresponding right-hemisphere region. The findings were found to be consistent with current cognitive models of reading acquisition and dyslexia, and with the Seki et al. (2001) study, pointing to the critical role of phonologic awareness skills in learning to read.

Dyslexics have also been shown (Temple & Hall, 2002) to have a disruption in white matter connectivity between posterior and frontal regions. Monitoring one's thoughts verbally has been thought to be dependent on an interaction between areas that generate and perceive inner speech in the frontal and temporal cortex, respectively. Shergill et al. (2002) used fMRI to examine the relationship between activity in these areas and the rate of inner speech that was being generated. Faster inner speech was associated with activation of the left inferior frontal gyrus, the right pre- and post-central gyri, and both superior temporal gyri. Thus, they found that activation of the superior temporal areas was associated with increasing the rate of inner speech, such as occurs with reading, suggesting that there is effective fronto-temporal connectivity. Regions responsible for verbal perception may be modulated by activity in areas that generate inner speech.

MARKSMANSHIP

Haufler, Spalding, Santa Maria, and Hatfield (2000) found that during aiming, when marksmen were compared with novice shooters, marksmen exhibited less activation (increased alpha with less beta and gamma activity) at all electrode sites, but the most pronounced differences were in the left central-temporal-parietal areas. In relation to the findings regarding anxiety and verbal processing, it is interesting that there have been reports of elevated left temporal alpha power, as we will review shortly, in marksmen during response preparation. This has been interpreted to indicate the suppression of verbal-analytic processes. However, lower-order motor processes had not been excluded as a possible explanation; therefore, Kerick et al. (2001) examined event-related alpha power (11-13 Hz) at sites electrode T3, T4, C3, and C4 in eight skilled marksmen during shooting and during two control tasks varying in perceptual-motor complexity. Over an eight-second period preceding the trigger pull, the marksmen exhibited higher power and slope at T3 than at all other sites during shooting compared with the control conditions. These differences were not found at C3 and C4, which means that these temporal findings cannot be primarily accounted for by "lower-order" motor processes.

Hatfield, Landers, and Ray (1987) discovered that alpha power progressively increased in the left temporal area during the last 7.5 seconds of aiming, while there was no change in the right temporal area. These results are congruent with previous research in the sports psychology literature that has shown less left hemisphere activation (more left hemisphere alpha power) in the preparatory period before golf putting (Crews & Landers, 1993) and archery (Landers et al., 1994; Salazar, Landers, Petruzzello, & Han, 1990). In archery, Landers (1991) found that alpha synchrony training at T3 during aiming significantly increased performance in pre-elite archers compared with a group trained to increase alpha at T4, which resulted in a decline in performance. In a longitudinal study over 14 weeks of archery training, Landers et al. (1994) discovered that left temporal alpha power increased and correlated with improved performance.

AUTISM

Most neuroimaging studies have found that autism is associated with anterior and medial temporal lobe dysfunction (e.g., Boddaert & Zilbovicius,

2002; Zilbovicius et al., 2000). For example, Zilbovicius et al. used PET scans to examine autistic children and non-autistic children with idiopathic mental retardation, but who were similar in average age and developmental quotients. The first autistic group studied had a highly significant hypoperfusion in both temporal lobes centered in associative auditory and adjacent multimodal cortex. This was detected in 76% of the autistic children, and virtually identical results were found in an extension of the study with a second autistic group.

NOCTURNAL ENURESIS

Hallioglu et al. (2001) evaluated primary enuresis patients and reported a decrease in alpha activity in the dominant temporal lobe and bilaterally in the frontal lobes. They also found an increase in delta activity in the right temporal area and differences from control subjects in the EEG after hyperventilation. Their results led them to conclude that, "Insufficient cerebral maturation is an important factor in the pathogenesis of primary nocturnal enuresis" (p. 714).

MORAL JUDGEMENT, PERSONALITY DISORDERS, AND MURDER

The frontal lobes are often implicated in violent and criminal behavior (e.g., Brower & Price, 2001). However, Gatzke-Kopp, Raine, Buchsbaum, and LaCasse (2001) found increases in slow wave activity in the temporal, but not frontal lobes, of murderers. Fascinatingly, they found that activation PET scans and resting EEG provided different information concerning brain dysfunction in murderers. Their study "… provides evidence that EEG is sufficiently sensitive to detect group differences, even in relatively small sample sizes" (p. 490). This is a very valuable finding given the practicality of doing EEG/QEEG evaluations in a prison setting as compared with PET scans, and the direct applicability of EEG evaluations to neurofeedback training. These temporal lobe findings are all the more interesting in light of the Raine, Buchsbaum, and LaCasse (1997) findings of parietal and medial temporal abnormalities in murderers and their conclusion that "… reduced prefrontal activity does not seem to be specific to severe violence, as this finding has been observed in a variety of psychiatric conditions" (p. 504).

Dolan, Deakin, Roberts, and Anderson (2002) used quantitative measures of frontal and temporal lobe volume as computed on the MRI in 19 control subjects and 18 personality disorder criminal offender patients who were characterized by impulsive-aggressive traits, but who did not show any evidence of brain pathology on diagnostic MRI scans. Interestingly, temporal lobe volumes were found to be 20% smaller in personality disorder patients than in control subjects, but their predicted reductions in frontal lobe volume did not occur, despite evidence of impairments in executive function. There was no evidence of differences in asymmetry of brain structures. This study further implicates the temporal lobes in the pathogenesis of severe personality disorder.

ALCOHOLISM AND CHILDREN OF ALCOHOLICS

Alcoholics have generally been found to have increased beta relative power (Costa & Bauer, 1997, John et al., 1988), particularly in frontal-temporal areas (Coger, Dymond, Seranfetinidea, Lowenstam, & Pearson, 1978; Coger, Dymond, & Seranfetinidea, 1979; John et al., 1988).

SCHIZOPHRENIA

Frontal and temporal pathology seem to be relatively consistent factors throughout schizophrenia research (e.g., Guenther et al., 1988; Halliday, 2001; Huang et al., 2003; Jin, Chen, Sandman, & Potkin, 1993; Lawrie & Abukmeil, 1998; Merrin & Floyd, 1996; Miyauchi et al., 1990; Notardonato, Gonzalez-Avilez, & Vanhhertum, 1989; Saletu et al., 1994; Woodruff et al., 1997), although there is tremendous heterogeneity in schizophrenia, and it certainly involves asymmetries and thalamocortical dysregulation (Gruzelier, 1999a, b) as well as coherence problems (e.g., John et al., 1994; Peled et al., 2001; Winterer et al., 2001). In the five subtypes that were found through cluster analysis by John et al. (1994), one subtype had beta hypercoherence in temporal regions (T3, T4). Another subtype showed a diffuse, moderate beta excess, particularly in temporal regions (T3, T4). A third subtype displayed moderate hypercoherence in alpha and beta temporally. Kircher et al. (2001) found that reduced activity in Wernicke's area in schizophrenics, which they associated with incoherent speech and it was correlated with formal thought disorder. Even ". . . when patients were articulating thought-disordered

speech, they showed a negative correlation with activity in the left superior temporal region–opposite to what occurs during production of coherent speech" (p. 772).

JET LAG

Now for a final, fascinating finding about the temporal lobes. There is evidence (Cho, 2001) that right-handed flight attendants who have been employed for five years on international flights, and who only have short recovery times (less than five days) between flights (as compared with those who have more than 14 days between flights) have significantly smaller right temporal structures. It is hypothesized that this temporal lobe atrophy is due to the effects of higher cortisol levels in these individuals, and it is associated with spatial memory impairment.

SUMMARY AND CONCLUSIONS

The temporal lobes are a site of frequent damage in concussions and head injuries, are associated with cognitive decline in aging, and are the location of the most common kinds of EEG abnormalities. The temporal lobes are centrally important in memory and verbal processes, and are areas associated with a variety of abnormal conditions, including language and reading disabilities, stuttering, epilepsy, bipolar disorder, schizophrenia, autism, panic disorder, headaches, and migraine. It is vitally important that the temporal lobes not be neglected in an EEG/QEEG evaluation and in neurofeedback training. However, thorough assessment of temporal lobe function requires evaluating the raw EEG with more than a linked-ears montage.

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APPENDIX 1. Electrode Sites in Relation to Brodmann Areas and Brain Structures

The information in this appendix has been compiled with the assistance of printed sources (Homan et al., 1987; Joseph, 1996; Nolte, 1999; Tranel, 2002) and from data supplied at my request by Roberto Pascual-Marqui at the Key Institute for Brain-Mind Research (Zurich). The numbers on the graphic refer to Brodmann areas and the circles to the vicinities of estimated electrode sites, which can vary slightly due to cranial differences and asymmetries. For instance, T3 might actually be located slightly lower in Brodmann area 21 in some individuals.

The frontal row of electrodes (from bottom to top) are estimates for electrode sites F9, F7, F5, F3, and Fz. The F7 electrode is at the front of Broca's area, in Brodmann area 45. The F5 electrode is at the front of Exner's writing area.

The central row of electrodes (from bottom to top) are T3, C5, C3, and Cz. On the homunculus, C3 is located over the area from approximately the wrist through the forearm. The C5 electrode is estimated to be over the area ranging from the fingers to the hand. About half-way between C5 and F7, near electrode site FC5, is the estimated middle of Broca's area.

The posterior row of electrodes are T5, P5, P3, and Pz. The P5 electrode is estimated to be in the middle of the Angular Gyrus.

The approximate location of certain functional areas in the brain is identified in the following legend.

Pink = Exner's Writing Area (In Brodmann areas 46, 8, and 6).
Orange = Broca's Expressive Speech Area (Brodmann area 44, and parts of areas 45, 6, and 4) (FC5 would be in the middle).
Yellow = Wernicke's Auditory Processing & Speech Area (Brodmann area 22 and part of 42), which is just behind Heschl's gyrus (Brodmann areas 41 and 42).
Blue = Angular Gyrus (Speech-Auditory Processing; Brodmann area 39).

APPENDIX 1 (continued)