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Psychophysiological indices of cerebral maturation

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Maturation (1-21 yr) trajectories for quantitative electroencephalographic (QEEG) frequency spectra are presented for four regions of the human brain. The results show that all four regions exhibited discontinuous maturation rates: five stages were identified. The stages were synchronous across regions during the first $10\frac{1}{2}$ years of life. Thereafter, the four maturation trajectories became independent of one another. Interestingly, a major maturational advance was recorded from the frontal regions, during late adolescence. The relationships between these findings to maturation rates in skull volume, cortical thickness, cortical volume and nerve cell density measurements was discussed. These converging results indicate that the observed QEEG stages can reliably be interpreted as landmarks in cerebral maturation.

INTRODUCTION

Psychological maturation in humans depends upon a complex interaction between biological and experiential development which extends throughout the human lifespan. On the one hand, biological competence determines the psychological experience; on the other, psychological experience alters and updates biological competence. The cerebral substrates coordinate with psychological functions thus become a unified structural and functional product of such interaction.

Much of this interaction is specific to particular psychological processes and therefore to specific substrates. However, during infancy and childhood, so many new experiences are encountered and so many skills are developed that coordinate structures based on synchronous correlations among maturation patterns, can be discerned. Furthermore, during infancy and childhood, brain competencies, the biological substrates upon which experiences have their impact, show plasticity unmatched after puberty is completed. As a result, during infancy, childhood, and even through puberty, changes in the indicators of maturation reflect biological variables as much as they reflect experiential ones.

The characteristics of maturation in infancy and childhood give rise to uneven changes in rate. Relatively rapid development alternates with slower rates, even plateaus, which delimit 'stages'. McGuinness, Pribram and Pirnazar (1990) have reviewed the behavioral evidence regarding a stage interpretation of maturation and the role of such evidence in the appearance of stages. Here, we critically review the evidence pertaining to the biological component(s) of maturation.

A number of recent studies have suggested that cerebral and cognitive maturation are intimately correlated. Rates of cerebral development have been estimated from cross-sectional studies of skull size, the electroencephalogram, cortical thickness, cortical volume and nerve cell densities

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(Epstein 1974a, 1974b, 1980, 1986; Hudspeth 1985, 1987; Hudspeth & Pribram, 1990; Thatcher 1987a, 1987b, 1990; Thatcher, Hudspeth and Hannelon, 1991). This diverse set of measurements, overall, provides consistent evidence that cerebral maturation proceeds in a discontinuous manner, characterized by spurts and plateaus.

The question to be addressed in this review is whether electrophysiological maturation *in specific brain regions* is sufficiently distinct to permit correlations to be made concerning specific psychological (i.e., perceptual, cognitive, affective and psychosocial) maturation trajectories. The electroencephalogram (EEG) has been a useful instrument in assessing the biological contribution to such correlations. The early studies of Lindsley (1936, 1939) demonstrated a gradual increase in EEG alpha frequency power during maturation. This pioneering work has led a number of workers to quantify features of the EEG (QEEG). Such measures have been shown to have high reliability and validity as indices for normal brain function (Dongier, et al., 1976; Gianitrapani, 1969, 1985; Hudspeth, 1985, 1987; Hudspeth & Pribram, 1990; John, 1977; John et al., 1982; Matousek & Petersén, 1973a, 1973b;



Fig. 1. System of-cerebral structures which are coordinate with cognitive functions. The hierarchical organization is arranged in a top-down manner and designated by the indicated textures for: Executive (E), Cross-Modal (X), Categorical (C), Object (O) and Image (I) functions. The image, object and categorical functions apply to the sensory and motor processes such as Vision (V), Audition (A), Gustation (G), and the Somatic divisions. The Somatic division is partitioned into predominantly Motor (SM) and Somaesthetic (SS) compartments. Intrinsic cross-modal processes are represented by regions within which the principal sensory systems interact: Visuospatial (VS), Visuoauditory (VA) and Acousticosomatic (AS). The frontal executive functions are interconnected to all other systems in a manner that the sensory and motor systems can be regulated by Priorities (EI), Practicalities (E2) and Proprieties (E3).

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Thatcher, 1990; Thatcher, Walker & Giudice, 1987a, 1987b; Thatcher, Hudspeth & Hannelon, 1991; Vogel & Boverman, 1964).

The brain processes involved in psychological functions are composed by large anatomical systems (Fig. 1) which, at the cortex, are organized hierarchically into extrinsic, intrinsic and executive functions. A working model of cortical function can be derived from systematic discussions by Luria (1973, 1980), Thatcher and John (1977) and especially Pribram (1954, 1971 & 1991). The extrinsic cortical systems are centered on the sensory and motor projections which serve as mapping regions for images (I) (e.g., auditory, visual somatic), from which invariant objects (O) can be extracted from the sensory-motor imaging systems. Intrinsic modality-specific areas provide product spaces within which object category prototypes (C) are obtained by iterated computations upon extrinsic object representations. The relations among category prototypes (i.e., cross-modal functions) (X) are obtained by cooperative computations in adjacent brain systems. The executive systems (E) take over when possible solutions to task demands are insufficiently specified by current categories of experience and action. The criteria for potential solutions are defined by its priority (E1), practicality (E2), and propriety (E3) (Pribram, 1991). The neuropsychological significance of the QEEG can, therefore, be inferred from the standard (Jasper, 1958; Homan, 1988) locations for EEG recording electrodes (Fig. 1).

METHODS

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Subjects and procedures

The data reviewed in this paper were published by Matousek and Petersen (1973a, 1973b) and are reproduced here with the authors' gracious permission. All details concerning the nature and selection of subjects, recording and data analysis may be found in their original work. EEG records were obtained from 561 normal 1 to 21 year old Swedish subjects, using eight bilateral scalp locations (e.g., F7-T3, F8-T4, T3-T5, T4-T6, Cz-C3, Cz-C4, P3-O1, P4-O2). They used analog filtering to estimate the power in six EEG frequency bands (δ , Θ , α 1, α 2, β 1 & β 2). Since 21

spheric differences, they pooled spectral data for homologous pairs of electrodes, and their final data were composed of 21 annual frequency spectra for each of four cortical regions. Finally, they calculated multiple regression equations between chronological age and several QEEG indices from each frequency band. The single highest correlation with chronological age was the ratio of alpha to theta activity. This ratio, the age dependent quotient (ADO = $(\Theta/\alpha) + 8$), predicted chronological age with an accuracy of $R^2 = 52\%$. Since • these workers did not calculate the average velocity, or first derivative, functions for the obtained trajectories, evidence for periodic maturation rates could not have been observed, and consequently, were never discussed.

John (1977) recalculated and tabulated the Matousek and Petersen data in four frequency bands ($\delta = 1.5 - 3.5$, $\Theta = 3.5 - 7.5$, $\alpha = 7.5 - 12.5$, β = 12.5-25.0; all in c/s). These tables provided 21, four-band, EEG spectra for the analyses presented in this paper. Further, 20 additional spectra, computed as averages for adjacent years, were interpolated between successive pairs of annual spectra to attain 6-month resolution in the final maturation curves (i.e., 41 spectra/region). Fig. 2a presents the EEG spectra, for the four regions in compressed spectral arrays (Bickford, et al., 1971, 1973) which describe the proportion of power in four frequency bands, between 1 and 21 years of age. The spectral arrays presented in Fig. 2a show the gradual transition of EEG frequencies, from slower to faster rates, the accepted operational definition of EEG maturation.

The electrode designations provide significant information concerning the functional significance of QEEG data obtained from each brain location, as follows: PO: The parieto-occipital derivation spans the area between the occipital (O1-O2) and parietal (P3-P4) regions, and is most likely to reflect influences from visual and visuospatial functions; TT: The temporo-tem- . poral derivation spans the area between the anterior (T3-T4) and posterior temporal (T5-T6) regions, and is most likely to reflect influences from auditory and visuoauditory functions; CC: The centro-central derivation spans the area between



GEEG FREQUENCY SPECTRUM

the dorsal (Cz) and lateral central (C3-P4) regions, and is most likely to reflect influences from somatic functions; FT: The fronto-temporal derivation spans the area between the anterior temporal (T3-T4) and orbital frontal (F7-F8) regions, and is most likely to reflect influences from frontal executive and auditory functions.

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Statistical analysis

As a general rule, most workers have presented separate maturation trajectories for each frequency band in the QEEG. This practice serves to fragment inferences about regional cerebral maturation into multiple and conflicting trajectories that both decrease (i.e., δ and Θ) and increase (i.e., α and β) with maturity. As a preliminary solution, the current work presents two methods for calculating a composite index for maturational trends, using all frequency bands. The statistical analyses presented in this paper were carried out in SYSTAT (V.5.0), using the CORR, FACTOR MGLH and STATS modules (Wilkenson, 1990).

We assumed that maturation within any region of the brain was likely to be relatively independent of that in other brain regions, therefore, all of the analyses presented in this paper were focused upon observed changes within individual recording locations. The relationships between different brain regions were restricted to regional similarities and differences in the final QEEG maturation curves. Further, we assumed that the entire envelope of the QEEG power spectrum provided a complete description for QEEG maturational state (in the frequency domain). Semian-

Fig. 2(a-d). QEEG compressed spectral arrays obtained from the Parieto-occipital (PO), Temporo-temporal (TT), Centrocentral (CC) and the Fronto-temporal (FT) regions of the brains of children from 1-21 years of life. The figure presents: (a) the original QEEG data obtained by Matousek and Petersen (1973); (b) Factor scores for the Mature (M) varimax components; (c) Factor scores for the Transitional (T) varimax components; and (d) Factor scores for the Immature (I) varimax components. The arrays for each brain region (a-d) are indexed by semiannual age increments (1-21 yr), and by four QEEG frequency bands: Delta (δ) = 1.5-3.5; Theta (Θ) = 3.5-7.5; Alpha (a) = 7.5-12.5; and Beta (β) = 12.5-25.0 (all in c/s). nual changes in the QEEG spectrum (i.e., profile of four frequency bands) would, therefore, constitute a complete description of increments in QEEG maturation state.

Two analytic methods were used to quantify incremental (i.e., maturational) changes in the QEEG spectrum profiles: (a) The direct (D)method calculated QEEG growth as the sum of squared deviations in δ , Θ , α and β relative power as an index of the distance (i.e., vector length) between successive pairs of QEEG spectra; and (b) The PCA-Varimax (V) method used rotated principal components analysis to identify and quantify orthogonal components (i.e., spectrum profiles) within the developmental QEEG spectra obtained at each electrode location. Age dependent increments in the magnitude of the component loadings were used to estimate the distance (vector length) between successive pairs of QEEG spectra. In either case, we estimated age dependent changes in total QEEG spectra, rather than age dependent changes in the individual frequency bands from which the spectra were composed. The extent to which the D and V methods provided parallel information about QEEG maturation was evaluated, and the results are discussed below.

We chose the Direct (D) and Varimax (V)incremental methods because the Age Dependent Quotient (Matousek and Petersen, 1973a, 1973b) accounts for only 52% of the variance with chronological age. Similarly, the John, et al. (1982) report used linear regression equations, computed for individual QEEG frequency bands, as a function of chronological age. While these workers did not report the predictabilities (R^2) between the linear QEEG indices and chronological age, Thatcher (Personal communication) reports that the linear regressions for QEEG indices he contributed to the John, et al. (1980) data base had only moderate correlations with chronological age ($R^2 \approx 60\%$). There are, therefore, significant levels of error $(100 - R^2 \approx 40\%)$ in correlating chronological age with indices which are assumed to be linear or monotonic across age.

Each QEEG spectral array was processed separately to assure that regional specificities were preserved. Incremental maturation trajectories for each region were obtained from the following calculations.

A. Direct semiannual increment curves (D)

(a) First-order differences (d = average velocity) were obtained for the δ , Θ , α and β frequency bands, where x = 2-41 and:

$$d_{\delta}(x) = (\delta(x) - \delta(x-1)),$$

$$d_{\Theta}(x) = (\Theta(x) - \Theta(x-1)),$$

$$d_{\alpha}(x) = (\alpha(x) - \alpha(x-1)),$$

$$d_{\alpha}(x) = (\beta(x) - \beta(x-1)).$$

(b) The semiannual increment vector (D) for each 6-month period was calculated as:

$$D(x) = \left(d_{\delta}(x)^{2} + d_{\Theta}(x)^{2} + d_{\alpha}(x)^{2} + d_{\beta}(x)^{2}\right).$$

(c) Proportional increments were calculated as ratios:

$$D(x) = D(x) / \sum D_{x-4i}$$

(d) The five increment curves, D_{PO} , D_{TT} , D_{CC} , D_{FT} and the group average D_{MN} , were smoothed with a three-point running average.

B. PCA-Varimax increment curves (V)

Varimax components analysis was used to obtain the quantitative indices for the maturational changes in the QEEG spectrum profiles presented in Figs. 2A-D. Individual spectral arrays were placed in computer memory so that the spectra for 41 age periods were located in the columns, and each frequency band was located in the rows of a rectangular matrix. A correlation matrix was computed between all column combinations (i.e., age periods). The resulting correlation matrices were then submitted to principal components analysis (PCA), and then rotated to the varimax criterion (Kaiser, 1958).

Each of the four regional analyses extracted three components which accounted for 99.9% of the variance within each spectral array. The three components were identified with the age periods in which they accounted for the highest propor-



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tions of variance. Accordingly, these were called: immature (1), transitional (T) and mature (M). The squared component loadings for the I and M components had a perfect reciprocal relationship over age, and they accounted for 84-89% of variance in the four regional spectral arrays. The T component accounted for 10-15% of the variance in the four arrays. Thus, all three orthogonal components accounted for significant proportions of variance, and therefore, all were essential to reproduce the original data with accuracy. Component scores were computed to graphically depict the QEEG spectral profiles associated with the I, T and M components which are shown in Figs. 2B-D.

Varimax increment curves (V) were calculated with the following algorithm:

(a) First-order differences (v = average velocity) were obtained for the *I*, *T* and *M* component loadings;

Where x = 2-41 and:

$$v_1(x) = (I(x) - I(x-1)),$$

$$v_2(x) = (T(x) - T(x-1)),$$

$$v_M(x) = (M(x) - M(x-1)).$$

(b) The semiannual increment vector (V) for each 6-month period was calculated as:

$$V(x) = (v_1(x)^2 + v_T(x)^2 + v_M(x)^2).$$

(c) Proportional increments were calculated as ratios:

$$V(x) = V(x) / \sum V_{1-41}.$$

(d) The five increment curves, V_{PO} , V_{TT} , V_{CC} , V_{FT} and average V_{MN} , were smoothed with a three-point running average.

A $2 \times 5 \times 41$ matrix ((Methods: D, V), (Electrodes: PO, TT, CC, FT and the mean, MN), (Semiannual period: 1-41)) of maturation increments was prepared for analysis. Repeated measurements ANOVA was used to evaluate the statistical significance of differences: (a) between D and V methods for calculating increment curves; and (b) between developmental rates obtained from different electrodes. Greenhouse-Geiser and Huynh-Feldt corrections were obtained for all repeated measurements comparisons, and the adjusted probabilities are reported here as, $P_{\rm HF}$.

RESULTS

Figs. 2b-d presents spectral arrays of the varimax component scores (i.e., spectral profile components) for the I, T and M components which were obtained for the four brain regions. The component scores were scaled with the component loadings to reflect the proportion of variance each profile contributed to the original spectral arrays shown in Fig. 2a. In all four brain regions, the algebraic sum of the I, T and Mspectral arrays (i.e., Fig. 2b-d) reproduced the original spectral arrays (Fig. 2a) with an error of $R^2 \leq 0.1\%$. These results clearly demonstrate that regional QEEG maturation trajectories are strongly periodic rather than linear. Fig. 3a-e presents five pairs of semiannual maturation trajectories for the direct (D_{PO} , D_{TT} , D_{CC} , D_{FT} and $D_{\rm MN}$) and varimax components ($V_{\rm PO}$, $V_{\rm TT}$, $V_{\rm CC}$, $V_{\rm FT}$ and $V_{\rm MN}$) methods used here. Based upon average maturation rates across the scalp, the repeated measurements ANOVA showed that there were significant changes in maturation rates over the 21 year life-span ($F_{(39,156)} = 15.94$; $P_{HF} =$ 0.0009). Orthogonal polynomial (Orders: 1-10; df = 1) contrasts showed that the 10th order polynomial (i.e., reflecting five maturation periods) was highly significant ($F_{(1,4)} = 44.17$; P = 0.0009). In addition, the 2nd-order polynomial contrast showed that there was significantly higher rates of maturation during the early and late years than there was in the middle years $(F_{(1,4)} = 40.48; P =$ 0.003). None of the remaining orthogonal contrasts were significant.

The D and V methods, used to calculate maturation rates, produced significantly different estimates during two periods ($F_{(39,156)} = 3.15$; $P_{HF} = 0.008$). These differences were obtained during the first $3\frac{1}{2}$ years (Average over period: $F_{(1,4)} = 68.17$; P = 0.001), and at $20\frac{1}{2}$ years of age ($F_{(1,4)} = 41.46$; P = 0.003). The reasons for these differences are not clear at present; however, their \propto 26

presence increased the error variance for comparisons among electrodes, and consequently, some contrasts were more conservative.

The average maturation trajectory (Fig. 2e) provided evidence for five global stages of brain maturation, and these would seem to be the most likely correlate for behavioral measurements (i.e., final common path) that obviously reflect the concerted work of many specialized brain functions. The present findings provide a detailed account of the regional brain changes that under-lie global maturation, and we would expect that knowledge of these changes could enhance the accuracy of behavioral methods used in developmental and neuropsychological studies.

Univariate F-tests were used to evaluate the observed differences among brain regions during each of the global stages. The first global stage (1-6 yr) was composed of overlapping changes in regional maturation that differed in acceleration rates and duration. These differences were observed between $1\frac{1}{2}$ -5 yr of age (average over period: $F_{(4,4)} = 24.50$; P = 0.004). During the second global stage $(6-10\frac{1}{2} \text{ yr})$ regional brain maturation was sufficiently synchronized that no significant differences between regions were obtained until the somatic (CC: $10-10\frac{1}{2} \text{ yr})$ region



Fig. 4. Semiannual increment curves computed by the Direct (D) algorithm. This figure presents a 2-dimensional representation of Fig. 3, so that the timing of growth events can be aligned. Growth is scaled in percentage increments for each 6 mo. period (1-41), in each of four brain regions (e.g., PO, TT, CC, FT).

departed from the global trajectory ($F_{(4,4)} = 12.80$; P = 0.02) with a small growth spurt. In the third global stage, most regions showed little or no advancement, except for the visuospatial region (PO: $11\frac{1}{2}-12$ yr) region which exhibited a moderate but significant maturation spurt ($F_{(4,4)} = 6.42$; P = 0.05). The fourth global stage was composed of significant maturation spurts in the somatic (CC: $13\frac{1}{2}-14\frac{1}{7}$ yr; average over period: $F_{(4,4)} =$ 63.45; P = 0.001), visuospatial (PO: 15 yr; $F_{(4,4)} =$ 13.40; P = 0.01) and visuoacoustic (TT: $15\frac{1}{2}-16\frac{1}{2}$ yr; average over period: $F_{(4,4)} = 11.63; P = 0.02)$ regions. Finally, the fifth global stage was exclusively due to rapid advancement within the frontal executive region (FT: $18\frac{1}{2}-20\frac{1}{2}$; average over period: $F_{(4,4)} = 47.85$; P = 0.002). Due to the limited number of degrees-of-freedom, the methods \times electrodes interaction term could not be evaluated. Fig. 4 presents the regional maturation trajectories (using the D estimates) which provides the bases for our detailed discussion below.

DISCUSSION

The analyses presented here provide evidence for the existence of stages in QEEG maturation. As can be seen in Fig. 4a-e and Table I, maturation patterns over the first decade of life $(1-10\frac{1}{2})$ yr) are synchronized across all brain regions. Beginning with the stirrings of puberty (roughly), maturation of the QEEG recorded from the temporal, central and frontal regions of the brain show different onsets and offsets of rapid change. This difference appears despite the fact that some synchrony in maturation during this period was observed in the parieto-occipital, temporo-temporal, and central regions between 13 and 17 years of age. However, the synchrony is incomplete: the peaks of maximum change are separated by approx. I year intervals. By contrast, the fifth stage (i.e., 17-21 yr) was recorded almost exclusively from the frontal electrodes. During puberty, cerebral maturation was found to proceed from the posterior to the frontal areas of the brain.

Thatcher, Walker and Giudice (1987a, 1987b) obtained QEEG coherence indices from an independent sample of children (birth to 16 years of , age) which also revealed discontinuous development patterns. Since coherence and phase measurements were not available in the Matousek and Petersen (1973a, 1973b) QEEG data, there is a need to compare the findings presented here, and those reported by Thatcher et al. (1987b). Efforts are currently underway to obtain quantitative comparisons between these two data sets. Meanwhile, preliminary findings for equivalent recording locations and age ranges, reveal highly significant correspondences between the two data sets, and these findings will be presented elsewhere (Thatcher, Hudspeth & Hannelon, 1991).

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Thatcher's (in press) review of neuroanatomical studies provides further evidence that the rate of brain maturation is uneven. Thatcher notes that the thickness of cortical gray matter (Rabinovicz, 1979; Conel, 1955, 1959, 1963, 1967), density of cortical volume (Blinkov & Glezer, 1968; Shade & Groeningen, 1961), and especially cortical neuron packing densities, reveal maturational peaks and plateaus which are consistent with the QEEG data presented here, and with his own findings.

It is reasonable to conclude, therefore, that maturation of the human brain occurs in 'stages' as defined here. The patterns of brain maturation suggest that the temporal and regional development of the brain is fundamental and, perhaps, the observed stages reflect major landmarks in cerebral maturation. If this hypothesis is correct, the QEEG maturation curves presented here lead to provocative implications and predictions concerning the functional organization of the human brain during maturation. A maturation period has three phases: (a) an accelerating edge; (b) a peak; and (c) a decelerating edge. These phases imply that the brain region under study: (a) is changing state; (b) has attained a new state; and (c) has established a stable equilibrium within the state. If these state changes can be considered landmarks in cerebral development, then nearly all behavioral indices (i.e., perceptual, cognitive or emotional) should be influenced by the changes.

The specific contribution reported in this paper was that QEEG data were analysed with respect to local cortical regions that encompass visuospatial, visuoauditory, somatic and executive functions. In the following discussion, the average (over all electrodes) maturation trajectory, with five stages, provides the reference for the detailed changes observed in local brain areas, as shown in Fig. 4.

As noted, the first stage of maturation which spans the period between 1 to 6 years of age is characterized by rapid maturation in all brain regions. The frontal executive (FT), visuospatial (PO), somatic (CC) and visuoauditory (TT) functions reach their respective peaks almost simultaneously (i.e., within 6-month intervals of each other).

In a second stage of maturation which spans the period between 6 to $10\frac{1}{2}$ years of age, rates are synchronized across the sensory and motor systems (PO, TT, CC), with all reaching a peak at $7\frac{1}{2}$ yrs of age. Accelerated development in the frontal executive region (FT) starts only then $(7\frac{1}{2}$ yr), but then terminates synchronously with that of the rest of the brain at $10\frac{1}{3}$ yrs. The early part of this period is, thus, a continuation of the maturation of sensory-motor functions, whereas, the latter part, beginning at age $7\frac{1}{3}$, heralds the maturation of executive control.

The third stage of maturation spans the period between $10\frac{1}{2}$ to 13 years of age. The third stage appears to be primarily devoted to the elaboration of visuospatial (PO) functions. At the same time, however, maturation of the visuoauditory (TI) function starts and slowly accelerates into the next stage.

The fourth stage of maturation spans the period between 13 to 17 years of age. This stage shows successive maturation of the visuoauditory (TT), visuospatial (PO) and somatic (CC) systems, with each region reaching its maturational peak within 1-year intervals, i.e., at 14, 15 & 16 yr, respectively. The frontal executive region remains unchanged at this time.

The fifth stage of maturation spans a period between 17 to 21 years of age. Maturation in this period was confined to the frontal executive (FT) functions. This observation was surprising as there was no previous evidence that maturation of the frontal (FT) executive system would appear at such an advanced age. The Thatcher, Walker ~ 28

& Giudice (1987) QEEG coherence measurements span only the first 16 years of life, and therefore, do not address this later stage of maturation. Our findings suggest that QEEG maturation studies could profitably be extended into middle and late adulthood.

In summary, the current findings have shown that multichannel QEEG spectral indices provided evidence for five stages of cerebral maturation. Regional development curves showed that brain development was closely synchronized during the first 10 years of life, and that regional differentiation occurs during later stages of human development. Based upon these findings, we suggest that major changes in human psychological functioning might be expected during those ages when the brain (QEEG) enters a new stage of maturity (i.e., Onset Age: 1,6, $10\frac{1}{2}$ 13 & 17 yr; Peak Age: $2\frac{1}{2}$, $7\frac{1}{2}$, 12, 15 & $18\frac{1}{2}$ yr).

We (Hudspeth & Pribram, 1990) have determined that the global QEEG stage boundaries described here, are in close correspondence with the stages delimited by Piagetian models of cognitive maturation (Piaget & Inhelder, 1966; Riegel, 1973 & 1975; Kramer, 1984). Further, other stage-based theories for the maturation of emotion (Freud, 1933), moral judgement (Kohlberg, 1969) and psychosocial adaptation (Erikson, 1963) delineate roughly the same age boundaries as those described in this paper. These theories are based on a wide range of observed behaviors. Thus, a considerable amount of variation is to be expected when attempts are made to delineate maturational stages with behavioral criteria alone. The fact that stages could be gleaned from such evidence at all, strongly suggests that biological factors are entailed in the processes denoted by these behavioral indices. Further refinements in these biobehavioral relationships would be expected when maturation in specific brain systems can be linked to maturation in specific behavioral systems.

In conclusion, a final caveat: Recall that we introduced this analysis by stating that brain and psychological maturation have a reciprocal influence on one another. Not only biological variables produce accelerations and decelerations in brain maturation as this is reflected in the QEEG. As well, psychological variables can produce such changes – or perhaps alter their time-course. Nonetheless, the evidence reviewed here, despite of, or perhaps because of the still-crude indices used, suggests that some very basic biological processes are equally critical (with experience) in determining the neurobehavioral coordinate space which shows accelerations and decelerations in maturation.

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