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Detailed Studies of Ontogenetic Changes in EEG Parameters in Men and Women during the Reproductive Period

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Changes in 272 EEG parameters were studied in 252 women and 97 men during the period of life from age 16 years to age 45 years with a step of one month. Correlation and approximation analysis showed more progressive age-related decreases in the amplitude and power of α, θ, and δ but not β oscillations in women than men, particularly in the right posterior leads. A general ontogenetic tendency to nonlinear decreases in the amplitude and power of EEG rhythms with age was seen in people of both genders. The frequencies of rhythms generally showed compensatory increases during ontogeny. Thus, neurologically healthy men and women showed different means of achieving the same end – adaptive responding of the brain to age-related changes.

Keywords: electroencephalogram, amplitude, gender-related differences, age-related regression, ontogeny.

The traditional interest in studies of age-related electrophysiological characteristics in childhood [3, 18, 20] continue to develop, with expansion of age-related EEG dynamics in humans to cover increased detail over the age range from three to 82 years. Thus, detailed assessment of age – not in terms of periods of ontogeny or in periods measured in years, but rather in periods measured in months – have led to stepwise documentation of nonlinear flattening of cerebral rhythms through adulthood to older age [7–9]. In particular, a negative correlation between the amplitudes of EEG rhythms with age in humans has been seen in the order β → α → θ → δ, i.e., in the direction from the convex surface of the brain to its depths [7, 8]. In other words, growth to adulthood and older age is associated with decreases in the amplitude of EEG oscillations (we note that brain death is seen on the EEG as a flat line, i.e., the amplitude decreases to zero). The deeper rhythms (θ and especially δ) flatten more clearly than the relatively superficial rhythms (α and β). The pattern whereby "the slower the EEG rhythm, the more closely its amplitude is linked with age" has been recorded in studies of people of both genders. This naturally raises the question: do gender-related differences exist? And how do EEG parameters (other than amplitude) change month to month in men and women during ontogeny?

With the aim of addressing these questions, and considering the gender-related directions of our further interests, there is value in initiating studies to detail events during reproductive age. Thus, the aim of the present work was to study gender-related differences in ontogenetic changes in EEG parameters in humans aged from 16 to 45 years with a step of one month.

Methods

EEG investigations were performed of 349 healthy adult volunteers (97 men and 252 women) with no objective neurological impairments or weather-related complaints, aged 16–45 years. Exclusion criteria were the presence of neuropathology and the complete absence of an α rhythm. EEG recordings were made using a Neiron-Spektr-4-/VP digital 21-channel electroencephalograph (Neirosoft, Ivanovo). Electrodes were positioned using the international 10–20 scheme with a correction for 21 leads, and the reference electrodes were on the earlobes. For baseline recording (at rest with the eyes closed), amplitude, power, frequency, and periodometric EEG parameters were determined and

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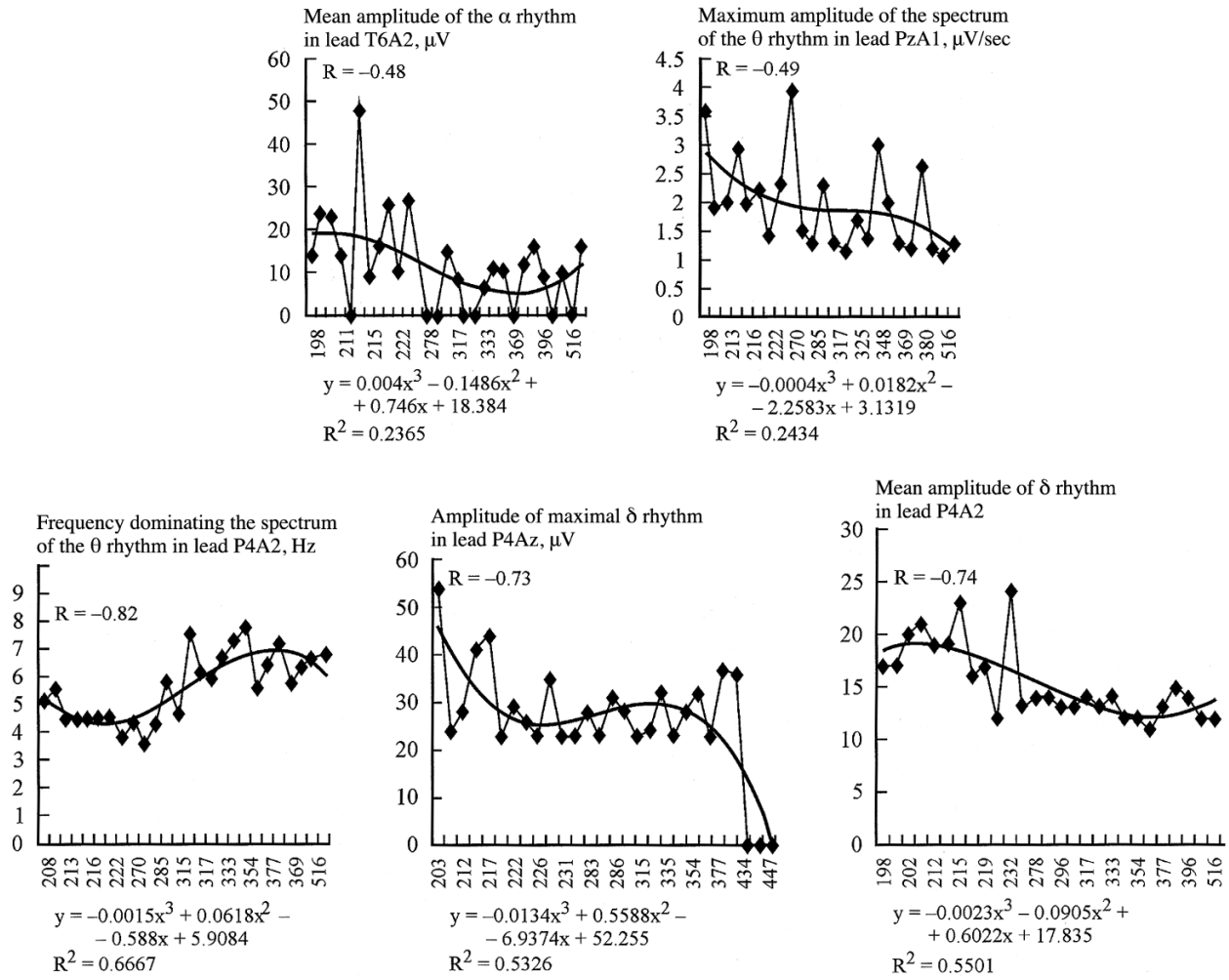

Fig. 1. Example plots of functions approximating the relationships between the values of neurodynamic parameters of EEG rhythms in women and age. Abscissas show age in months. Points show EEG measures in individual subjects. Equations are for polynomial functions, coefficients of determinacy (R^2), and correlation coefficients (R).

individual neuromaps were analyzed [19]. The calibration signal was 50 μV, the trace rate (EEG scanning rate) was 30 mm/sec, and the sensitivity (EEG scale) was 7 μV/mm. EEG parameters: monopolar, 21 leads. Spectral analysis: palette step 1; spectrum plot scale 0.5 μV/pixel, asymmetry assessment starting from 15%. The time delay for correlation analysis was 400 msec. EEG parameters were calculated – data averaging, assessment of mean and maximum EEG oscillation amplitude in each of the 21 leads in each of the rhythm ranges δ, θ, α, and two β (low- and high-frequency) after preliminary narrow-band filtration of the raw EEG, use of an EEG spectral power and amplitude analysis algorithm, measurement of spectral power indexes – in a statistics package included in the digital electroencephalogram software (Neiron-Spektr, from Neirosoft). The terms "low-frequency and high-frequency beta rhythms" (widely accepted in clinical neurophysiology) are also taken from the instrument interface and were identical to the standard division of β oscillations into β1 and β2 ranges used in electroencephalography, i.e., 14–25 and 25–40 Hz, respectively.

As the size (duration) of each of the epochs analyzed was 10.24 sec, i.e., 2048 sampling cycles, comparative analysis of, for example, two EEG epochs from one subject, involves an enormous number of degrees of freedom (n = = 2048), as computer electroencephalography compares values at each sampling point in the epoch. Digital analysis used artifact-free EEG traces of six epochs, which were subjected to fast Fourier transformation using a Hann window.

During investigation of each subject, 272 neurodynamic parameters were assessed: amplitude without consideration of individual rhythms and by lead (21 leads, maximum and mean amplitudes), μV; the amplitudes of the δ, θ, α, and two β (low- and high-frequency) rhythms for each lead (maximum, mean), μV; spectral power by lead (maximum, mean, full), $\mu V^2/sec^2$; spectral power of EEG

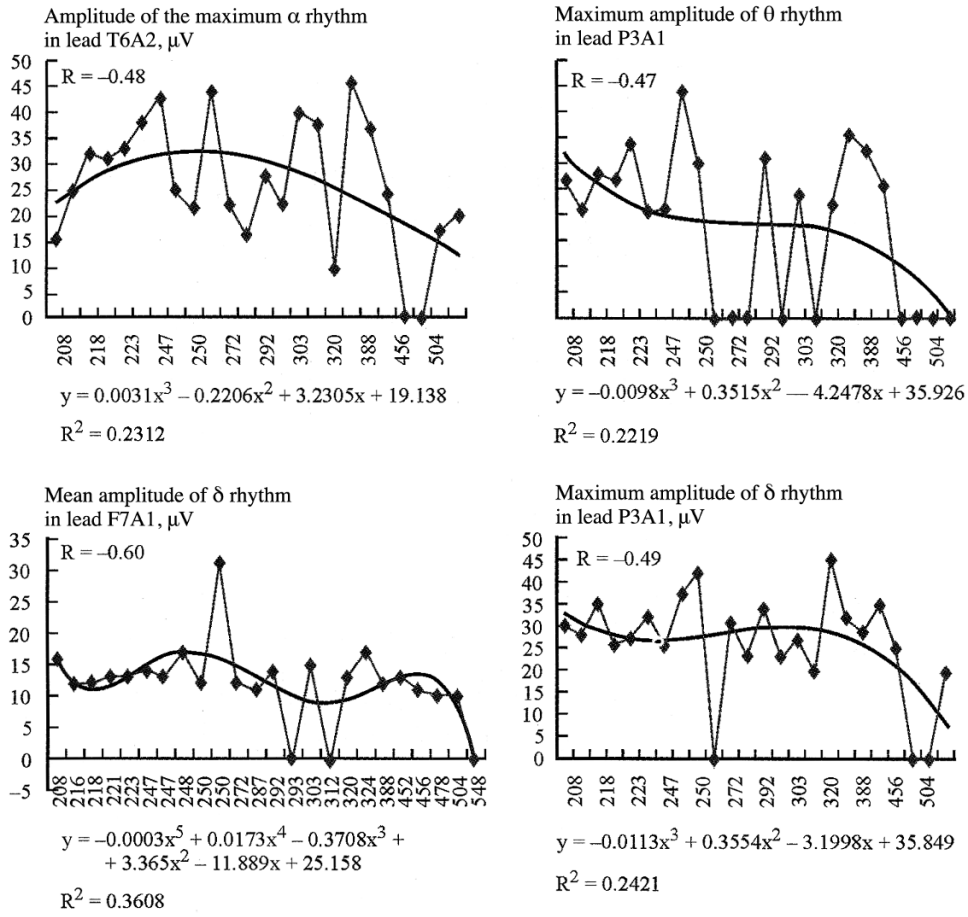

Fig. 2. Examples of plots of functions approximating relationships between the magnitudes of neurodynamic parameters and EEG rhythms in males and age. For further details see caption to Fig. 1.

oscillations in the δ, θ, α, and two β rhythms (maximum, mean, full), $\mu V^2/sec^2$; spectral frequency by lead (dominant, mean), Hz; spectral frequencies of the δ, θ, α, and two β (low- and high-frequency) rhythms (dominant, mean), Hz; spectral amplitudes by lead (maximum, mean, full), μV/sec; spectral amplitudes of the δ, θ, α, and two β rhythms (maximum, mean, full), μV/sec; power spectrum index of EEG oscillations in each study range; asymmetry, % (neuromapping results); distribution of amplitudes in five ranges in 21 leads for assessment of the periods of EEG rhythms, %. In the hierarchy of neurodynamic parameters analyzed here, those of leading physiological significance (on the basis of previously reported details [6]) were the measures of the expression of functions – rhythm amplitudes and spectral amplitudes. The data obtained here were subjected to correlation analysis and relationships between the values of each measure and age (in months) were approximated using the least squares method. Correlation analysis was used to evaluate the extents of linear relationships between each of the 272 neurodynamic parameters with subjects' ages, expressed in months rather than years. In particular, pairwise correlations were performed: a neurodynamic parameter, such as the amplitude of mean EEG oscillations in the δ rhythm in lead F7A1 (first variation series) and subject's age in months (from 208 to 504 months), each of which corresponded to an amplitude determination. R values were thus calculated. The least squares method was then used to assess the extent of the nonlinear relationship between all these pairs of characteristics, where the function in each case was one of the 272 neurodynamic parameters and the argument was the person's age in months. Values for each parameter in the two genders were also compared. Furthermore, Student's *t* test was used within each age range (16–45 years) to compare two groups of adult subjects: 1) early adulthood, 198 female subjects and 63 male subjects aged 16–35 years and 2) late adulthood, 54 female subjects and 34 male subjects aged 36–45 years.

We emphasize that our studies recorded and compared EEG data (components of variation series) in different neurologically healthy subjects whose ages at the moment of investigation (in months) were used as the argument in further analysis. It is important to note that the investigations were

TABLE 1. Comparison of EEG Spectral Power ($M \pm m$; $\mu V^2/sec^2$) in Women of Two Age Groups – Early (*1*) and Late (*2*) Adulthood

Lead	Maximum power			Full power			Mean power		
	1, $n = 198$	2, $n = 54$	t	1	2	t	1	2	t
O2A2	73.99 ± 33.93	33.96 ± 9.65	1.23937	345.92 ± 277.66	271.1 ± 245.54	0.770187	1 ± 0.21	0.75 ± 0.17	0.926635
O1A1	48.95 ± 20.54	27.79 ± 6.9	1.05936	294 ± 287.42	204.06 ± 170.59	1.047412	0.85 ± 0.22	0.56 ± 0.12	1.214995
P4A2	22.09 ± 3.65	26.89 ± 5.53	–0.68755	248.08 ± 108.93	213 ± 96.12	0.920862	0.75 ± 0.1	0.59 ± 0.07	1.318370
P3A1	22.74 ± 3.9	28.58 ± 8.21	–0.59437	248 ± 111.08	228.28 ± 157.43	0.378777	0.75 ± 0.12	0.64 ± 0.11	0.697105
C4A2	29.19 ± 4.81	17.6 ± 5.38	1.56888	250.85 ± 70.49*	163.75 ± 77.77*	3.125797	0.76 ± 0.08*	0.46 ± 0.05*	3.160720
C3A1	20.07 ± 2.69	19.97 ± 4.24	0.01895	195.15 ± 59.39	186.13 ± 71.3	0.364855	0.61 ± 0.11	0.52 ± 0.05	0.840725
F8A2	22.7 ± 6.38	15.24 ± 3.45	1.08156	173.85 ± 88.58*	117.94 ± 51.89*	2.121159	0.52 ± 0.13*	0.33 ± 0.04*	2.356739
F7A1	18.24 ± 5.32	17.38 ± 6.24	0.10170	167.08 ± 123.32	133.63 ± 82.75	0.871674	0.52 ± 0.16	0.37 ± 0.06	1.174880
F4A2	39.72 ± 12.94	20.91 ± 6.22	1.39127	255.62 ± 153.56	168.63 ± 79.22	1.971262	0.77 ± 0.14*	0.47 ± 0.05*	2.209161
F3A1	46.31 ± 21.84	26.06 ± 7.65	0.94728	262.92 ± 194.83	202.63 ± 123.99	1.013031	0.79 ± 0.14	0.57 ± 0.09	1.273798
Fp2A2	60.46 ± 12.75	44.81 ± 10.92	0.93713	349.31 ± 183.12	269 ± 116.09	1.428354	1.03 ± 0.14	0.75 ± 0.08	1.800573
Fp1A1	57.92 ± 16.4	46.1 ± 14.82	0.53458	338.54 ± 60.26	283.38 ± 197.53	0.812124	1.01 ± 0.13	0.79 ± 0.14	1.129741
T6A2	40 ± 12.39*	14.84 ± 3.29*	2.14841	252.08 ± 174.72*	117.56 ± 53.85*	2.924139	0.75 ± 0.14*	0.33 ± 0.04*	3.196420
T5A1	36.79 ± 15.45	18.71 ± 7.29	1.12503	199.23 ± 126.96	134.44 ± 91.84	1.594050	0.62 ± 0.126	0.38 ± 0.07	1.797377
T4A2	22.05 ± 5.3	60.21 ± 43.59	–0.78279	179.46 ± 86.72	314.63 ± 725.43	–0.665675	0.54 ± 0.08	0.87 ± 0.5	–0.584844
T3A1	12.68 ± 1.84	25.13 ± 8.04	–1.36787	144.46 ± 52.95	160.81 ± 104.12	–0.513647	0.45 ± 0.067	0.45 ± 0.07	–0.013541
FpzA2	53.54 ± 12.22	41.33 ± 11.9	0.70993	306.85 ± 146.16	274.5 ± 186.23	0.510801	0.91 ± 0.12	0.76 ± 0.13	0.805495
FzA1	25.11 ± 2.74	19.41 ± 5.28	0.89440	233.38 ± 63.52	196.88 ± 81.34	1.322086	0.71 ± 0.08	0.55 ± 0.05	1.682680
CzA2	23.67 ± 4.54	20.98 ± 3.15	0.50107	262.23 ± 109.4	206.69 ± 78.69	1.589377	0.78 ± 0.09	0.58 ± 0.05	1.974223
PzA1	41.25 ± 16.52	22.32 ± 4.31	1.21511	299.15 ± 171.58	221.75 ± 99.72	1.519658	0.91 ± 0.15	0.62 ± 0.07	1.825605
OzA2	40.39 ± 23.02	22.17 ± 4.5	0.85736	326.62 ± 581.88	159.88 ± 65.9	1.142024	0.94 ± 0.45	0.45 ± 0.05	1.221730

*$p < 0.05$.

not performed on individual subjects investigated at different periods of their lives. That is, the study was not longitudinal.

Data were processed in the statistical package Microsoft Excel 2010.

Studies were performed in compliance with the requirements of the Convention of the European Council "Human Rights and Biomedicine" (1997) and the supplementary protocol of this Convention applicable to certain biomedical investigations (2005); adult subjects provided informed consent to take part, while written content was provided by the parents of young people. The degree of risk imposed by this study was minimal.

Results

These experiments showed that there were gender-related differences in the dynamics of the main EEG parameters during the reproductive period in humans: women showed more progressive age-related regression of amplitude and power values than men.

TABLE 2. Comparison of EEG Spectral Power ($M \pm m$; $\mu V^2/sec^2$) in Men of Two Age Groups – Early (1) and Late (2) Adulthood

Lead	Mean power			Full power		
	1, $n = 63$	2, $n = 34$	t	1	2	t
O2A2	1.08 ± 0.34	0.82 ± 0.31	0.56652	366.5 ± 128.74	192.0 ± 46.27	1.34581
O1A1	1.56 ± 0.86	0.94 ± 0.28	0.76816	522.17 ± 314.57	248.43 ± 52.2	0.92875
P4A2	1.73 ± 0.56	0.69 ± 0.16	2.03687	521.33 ± 163.89	241.29 ± 67.87	1.67013
P3A1	1.35 ± 0.43	0.59 ± 0.12	1.92284	383.5 ± 67.2*	191.57 ± 46.27*	2.40990
C4A2	1.88 ± 0.53*	0.74 ± 0.13*	2.41061	583.3 ± 172.22	257.86 ± 52.2	1.93618
C3A1	1.02 ± 0.18	0.75 ± 0.17	1.09837	331.3 ± 68.62	248.71 ± 65.86	0.86607
F8A2	1.68 ± 0.76	0.38 ± 0.07	1.99828	582.17 ± 281.44	112.71 ± 17.09	1.81085
F7A1	0.89 ± 0.27	0.47 ± 0.09	1.61307	282.33 ± 97.61	170.29 ± 40.35	1.12234
F4A2	1.63 ± 0.49	0.67 ± 0.14	2.12576	554.0 ± 186.92	234.71 ± 55.96	1.75242
F3A1	0.99 ± 0.2	0.74 ± 0.13	1.07712	314.0 ± 71.73	254.71 ± 54.06	0.67140
Fp2A2	4.22 ± 1.83	1.01 ± 0.19	2.03111	1353.17 ± 669.03	365.71 ± 78.99	1.59098
Fp1A1	4.22 ± 2.96	0.96 ± 0.17	1.28594	1466.33 ± 1077.24	309.0 ± 59.21	1.16686
T6A2	1.58 ± 0.71	0.91 ± 0.46	0.83112	542.67 ± 256.84	171.14 ± 67.66	1.50308
T5A1	0.66 ± 0.16	0.41 ± 0.06	1.57864	209.5 ± 57.83	132.43 ± 21.17	1.33101
T4A2	1.19 ± 0.42	3.05 ± 2.71	–0.58354	405.5 ± 159.11	122.14 ± 21.54	1.91391
T3A1	0.98 ± 0.27	0.8 ± 0.18	0.57546	327.83 ± 103.69	294.29 ± 71.03	0.27356
FpzA2	1.81 ± 0.6	1.93 ± 1.08	–0.08586	592.5 ± 217.79	306.71 ± 63.29	1.35055
FzA1	2.61 ± 1.31	0.8 ± 0.14	1.59328	894.67 ± 484.15	287.71 ± 58.89	1.35055
CzA2	1.5 ± 0.52	0.85 ± 0.22	1.26050	511.3 ± 198.6	290.86 ± 88.79	1.06790
PzA1	1.16 ± 0.17	1.19 ± 0.47	–0.06263	371.0 ± 56.3	271.86 ± 75.98	1.01731
OzA2	1.17 ± 0.34	0.76 ± 0.31	0.88151	376.17 ± 125.05	165.57 ± 41.33	1.70714

*$p < 0.05$.

Overall, the general ontogenetic tendency to decreases in the amplitude and power of cerebral rhythms with age were seen in members of both genders over the age range 15–45 years (Figs. 1 and 2). The frequencies of rhythms during ontogeny generally showed compensatory increases, as the appropriate and optimal level of metabolic support for the brain in normal conditions is provided for by the level of expression of the cerebral function (adequate amplitude and power of the rhythmic process) or the time argument (increase in oscillation frequency) [6]. The decrease in EEG rhythm amplitude in neurologically healthy people (with rare specific exceptions) is therefore accompanied, including during ontogeny, by an increase in rhythm frequency which is not necessarily confined to the same range [7–10, 14] and vice versa. This applied to all EEG ranges except the β range.

However, the clearest regressive nature of amplitude and power changes was seen in women. Thus, investigation of female subjects revealed significant ($p < 0.001$; from 0.40 to 0.88 R and R^2) flattening of the amplitude and weakening of the power of the δ, θ, and α (but not β) rhythms during the reproductive period (Fig. 1), which is also supported by traditional intergroup comparison mean absolute values from women and men (Tables 1 and 2). Of the 272 neurodynamic parameters recorded and analyzed from each subject in each investigation, the illustrative material for the present report shows the individual parameters and leads for which typical patterns were recorded, though a larger set is covered where possible: for example, the Tables show power and the Figures show amplitude and frequency values. Even in terms of the quite crude Student's test (Tables 1 and 2, along with the approximation curves, Figs. 1 and 2), age-related differences were seen as additional support for the regressive ontogenetic tendency. Thus, averaging of the parameters from women (Table 1) demonstrated weakening of EEG power in the posterior frontal, posterior central, and posterior temporal leads on the right. Quantitatively, age-related differences were four times greater than in men (8 vs. 2, Tables 1 and 2). It is interesting to note, for example, that the amplitude of EEG oscillations in lead T6A2 in the state of waking (used in calculation of power values) generally started lower than in other scalp leads. However, in this case, the value important for interpretation was less the absolute value of this parameter at the study time point than its dynamics with peple's aging: the amplitude, which was initially low, nonetheless continued to decrease with age from the "initial" value (though to a lesser extent in the α rhythm than in other ranges), which we believe points to the existence of a common tendency to regression even in those leads which were very atypical in terms of the location of maximum amplitudes.

This regression could in some cases be followed continuously, while in others it was nonlinear in nature, reflecting the involvement mainly of high-frequency EEG components at the early stages of the ontogenetic period of interest and low-frequency components at the late stages of this period. In spatial terms, these features in women dominated over the posterior half of the scalp and were also recorded in individual parietal, central, and frontal leads. As for the slow rhythms (θ and δ), regression was seen mainly in the right hemisphere. As already noted, this tendency does not apply to the β range. In fact, females showed, as an exception, a gradual increase in the spectral power index for

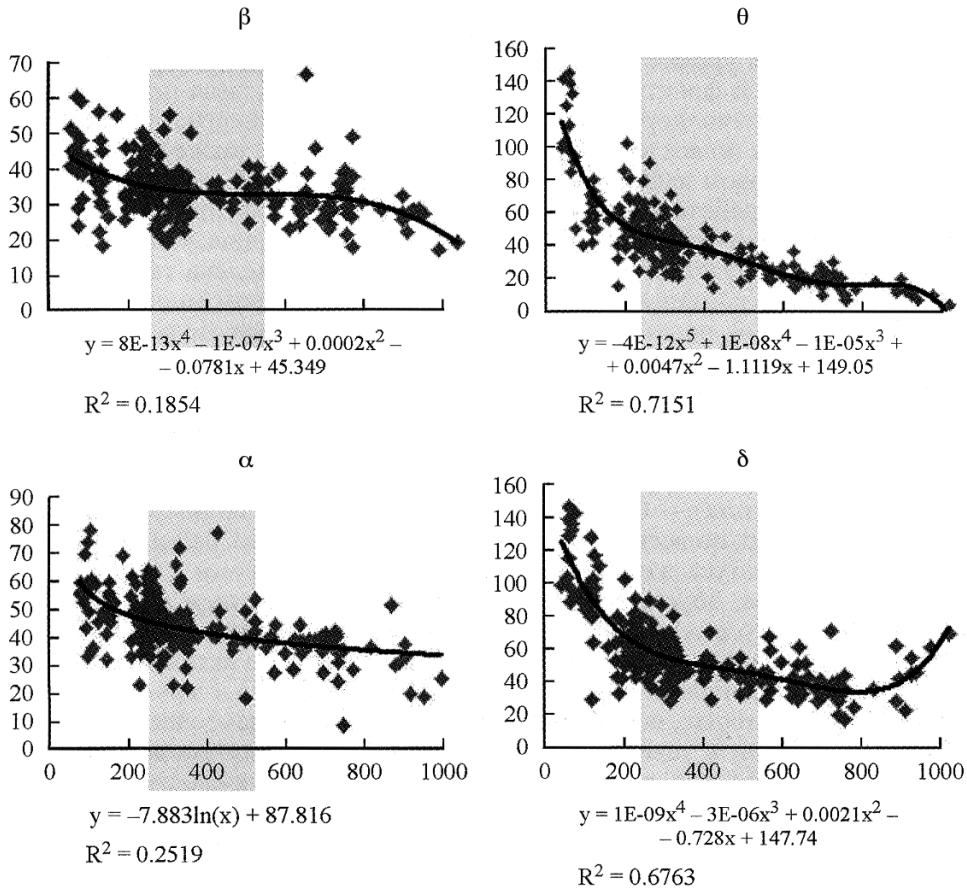

Fig. 3. Plots of functions approximating relationships between the mean amplitudes of EEG oscillation spectra in the δ, θ, α, and β rhythms (ordinates, μV/sec, mean for 21 leads) and age (abscissas, months). Results from investigations of 417 subjects of both genders aged 3–82 years. Points show measures of the amplitudes of the whole EEG spectrum in individual subjects. Equations for functions and coefficients of determinacy (R^2) are given. Gray bars show age range in which gender-related differences were studied: 16–45 years (208–504 months).

β-range EEG oscillations, mainly in the left central and parietal leads.

In contrast to this, assessment of male EEG recordings did not show such clear flattening of the amplitude and power of the main EEG rhythm from age 15 years to age 45 years (Table 2). Conversely, as shown in Fig. 2, the amplitude of the α (and β) rhythms from the moment at which the youth period was complete to the early adulthood stage showed some increase of the "short-term surge" type. It was only by the late adulthood period (and later) that this acquired an actual regressive character. As a result, the plot of the polynomial function approximating the relationship between α-rhythm amplitude and age in months was reminiscent of a parabola with a rise phase at an argument value of 250 months, corresponding to the 21st year of life. The gradient-type decrease during the whole of the reproductive period of interest in males applied only to the amplitudes of the slow-wave components of the spectrum – the δ and θ rhythms. This was accompanied by decreases in the frequencies of the slow waves too. In contrast to the EEG in women, age-related differences in the EEG of men affected a greater area of the left hemisphere, i.e., its frontal, temporal, and parietal areas.

Discussion

Despite known gender-related heterochrony in relation to the expression of the various rhythms in mature adult humans overall, the general ontogenetic tendency to flattening of the cerebral rhythms with age, as already noted, is evident in people of both genders, though it is more clear in women than men. The factor initially combining the genders in this situation consisted of the deep waves – δ and θ. Their amplitudes steadily decreased from age 16 years to age 45 years, and the link with age was significantly stronger than in the case of the β and α waves, which has in principle been established in humans without gender differentiation in a wider ontogenetic series – form three to 82 years (Fig. 3). As shown in Table 3, investigations of different humans of both genders (n = 417) aged from 36 to 990 months at the point of investigation showed that significant ontogenetic regression of the full amplitude of the EEG spectrum increased in the

series β → α → θ, δ, judging from the predominantly descending nature of the plot and the increase in the coefficient of approximation (R^2): 0.19, 0.25, and 0.72. In the δ range, the relationship barely showed a weakening (as compared with the θ range), had the specific feature of a late age-related dynamic, and had a value of 0.68, which nonetheless corresponded to the very tight and statistically significant correlation coefficient of –0.82 ($p < 0.001$).

Gender differences in the age-related dynamics of the EEG components studied here (measured in the life segment 208–504 months) affected mainly the superficial rhythms, not the fastest, i.e., the main (α) and low-frequency β rhythms. For example, in the interval from 200 to 276 months, i.e., from 17 to 23 years (the juvenile period of ontogeny), the amplitude and power of EEG oscillations increased in the α range in men but decreased in women. The frequency of the main rhythm in men decreased at this time, while that in women, conversely, increased. These changes, considering the ontogenetic significance of the juvenile period, (final establishment of reproductive functions per se and generating gender differences), are in essence "mirror images:" an "increase" vs. a "decrease" in the absolute levels of expression of the function. In terms of current concepts, the α rhythm supports integration of the operation of the brain at the level of corticocortical interactions, internalization of attention (imagination, creative activity, retention of information in operative or extraction from long-term memory), and the mechanisms of selecting descending (top-down) control, i.e., an increase in the useful signal to noise ratio due to inhibition of uninvolved cortical and subcortical areas [21, 23]. The spectral power of EEG oscillations in the α range, used in the present study to document gender divergence, judging from the results of multimodal neuroimaging [1], has an inverse relationship with the activity of the stem sections, midbrain, hypothalamus, amygdala, and the insular, prefrontal, and dorsal premotor areas of the cortex, while changes in β activity are increasingly linked with the cognitive regulation of the emotional domain and neuroautonomic status [12, 22].

It can be suggested that the δ rhythm is also not involved in the direct on-line regulation of autonomic functions, reflecting mostly the homeostatic changes involving the common brainstem system present in both genders [24]. Along with this, the θ rhythm is predominantly of septohippocampal and, probably, amygdalar origin (as can be determined with some degree of caution), is partly induced by cholinergic processes, and is related to the complex functional state of the brain [25, 26], such as, for example, autochronometry [3, 4, 7], ontogenetic establishment of which also depends on cognitive, emotional, and mnestic components heterochronously exerting their influences during the course of individual development. Ontogenetic regression of EEG rhythms in the θ range may be linked with weakening of cholinergic function as a result of the gradual decrease in circulating estrogen [13]. There are reports [17] that this process occurs as a result of suppression of the activity of the enzyme acetylcholine transferase and decreases in the level of expression of acetylcholine transferase mRNA in the hippocampus, as well as in the cortex and neocortex. Weakening of noradrenergic function (this system being a synergist of the dopaminergic system in relation to this parameter), which is also seen in estrogen deficiency [13], cannot be excluded as a cause.

The present report describes an attempt to use multiparametric recording and analysis of spontaneous total bioelectrical activity in women and men of fertile age to evaluate the chronophysiological components of the EEG with a natural set of multiple variants. This approach, despite the relative (and unavoidable in this case) heterogeneity in the dataset, identified a significant tendency to a decrease in amplitude and a weakening of the power of EEG rhythms in the period of ontogeny from age 16 years to age 45 years, which was demonstrated by statistical and mathematical analysis results. These facts provide evidence that this regression in women is clearer than in men and clearer in the slow-wave rhythms (θ and δ) than the short-period rhythms (α, and β). The cause of this is in all probability the greater variability of hormonal status in women than men during the ontogenetic period of interest, due to cyclic alternation of the phases of the ovarian-menstrual cycle, and also preclimacteric changes. The significant influences of steroids on bioelectrical activity are known [13, 17], in particular on EEG components in the θ and δ ranges, whose amplitudes and power levels are indicated by our data to show the greatest age-related decreases during the period of ontogeny of interest in women, predominating to the later adulthood period. There is probably a weakening of the effects of hormones on the female brain by the end of the reproductive period of ontogeny, linked on the one hand with age-related changes in the sensitivity of brain target structures (hypothalamic and hippocampal centers) and, on the other, with a natural decrease in their concentrations. This latter starts before the preclimacteric changes due to gradual depletion of the pool of follicles [15, 16].

The mirror nature of neurodynamic parameters ("men-women") is often seen both in the temporal and the spatial aspects. The value of this opposition (also repeatedly established in terms of other parameters and functions, e.g., rheoencephalography [10] and thoroughly reviewed by other authors [11]), in essence a dialectical unity, is explained by the evolutionary requirement for differentiation of the human population. This does not exclude a quite beneficial functional interchangeability.

Thus, judging from EEG data, ontogenetic changes in electrophysiological characteristics in adult men and women are not identical. Attention is drawn to the almost complete and almost mirroring coincidence of the individual components of the functioning of the α, β, θ, and δ neuronal networks: those indicators whose absolute values in members of the female gender were lower correlated more weakly with other age-related parameters in men and more strongly

in women. For example, in lead T6A2 the initially decreased amplitude and power of EEG rhythms nonetheless continued to show significant and objective decreases in women, while in men it remained unaltered on averaged age-based comparison using Student's test (Tables 1 and 2; Fig. 1). The more sensitive least squares method showed the age-related dynamics of the α rhythm to be nonlinear: there was a smooth increase, then a drop after the 21st year of life (250 months; Fig. 2). At the level of gender-related differences, such an obvious qualitative "deficiency," expressed as relatively low (but within the normal range) absolute values of parameters, is compensated for by a quantitative advantage. This type of compensation is expressed in the greater linkage of the components of individual chronophysiological processes with an irreversible and quite strong endogenous factor – age – the mechanisms of interaction with which are strong, various, adaptively important, and therefore in a state to provide an appropriate redistribution of intracerebral energy to the relevant parts of the brain. In other words, in neurologically healthy men and women, judging from EEG measures, a single aim – adequate adaptive responding of the brain to natural age-related changes, including those supporting regulation of normal reproductive functions – is achieved in different ways. Tactical intergender antagonism apparent at certain stages of development ultimately produces functional synergism at the level of the whole living system. The ontogenetic pattern of changes in EEG parameters in members of both genders is reproduced in detail both in long (the step being adulthood) and short (the step being one month) segments of individual development, probably supporting the harmonic interaction of the genders on the scale of human populations.

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