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Maturational Changes in Automated EEG Spectral Power Analysis in Preterm Infants

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ABSTRACT: Our study aimed at automated power spectral analysis of the EEG in preterm infants to identify changes of spectral measures with maturation. Weekly (10–20 montage) 4-h EEG recordings were performed in 18 preterm infants with GA <32 wk and normal neurological follow-up at 2 y, resulting in 79 recordings studied from 27^{+4} to 36^{+3} wk of postmenstrual age (PMA, GA + postnatal age). Automated spectral analysis was performed on 4-h EEG recordings. The frequency spectrum was divided in delta 1 (0.5–1 Hz), delta 2 (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz) band. Absolute and relative power of each frequency band and spectral edge frequency were calculated. Maturational changes in spectral measures were observed most clearly in the centrotemporal channels. With advancing PMA, absolute powers of delta 1 to 2 and theta decreased. With advancing PMA, relative power of delta 1 decreased and relative powers of alpha and beta increased, respectively. In conclusion, with maturation, spectral analysis of the EEG showed a significant shift from the lower to the higher frequencies. Computer analysis of EEG will allow an objective and reproducible analysis for long-term prognosis and/or stratification of clinical treatment. **(*Pediatr Res* 70: 529–534, 2011)**

Advances in the care of very preterm infants have led to an increased survival (1). However, a considerable number of these infants experience neurological deficits later in life, even in the absence of neuroimaging abnormalities (2,3). The exact etiology of these developmental deficits remains to be clarified, but it is suggested that medical, environmental, and iatrogenic conditions may interfere with white matter development of the vulnerable preterm brain (4). Therefore, brain function monitoring in preterm infants during their stay in the NICU may be valuable in detecting conditions that interfere with brain development (5). It is a challenge to develop effective monitoring and therapeutic strategies to protect the preterm brain.

The EEG is regarded as the gold standard in the assessment of cerebral function. Assessing changes in EEG are useful in the prediction of long-term outcome (6). Although the acute and chronic EEG changes are mainly nonspecific regarding type of damage, they correlate with later neurological and cognitive function (7). In preterm infants developing white matter damage, acute EEG findings include decreased continuity, lower amplitude of background activity, and epileptic seizure activity (8). The chronic EEG changes associated with white matter injury and abnormal neurological development include delayed maturation and disorganized pattern with the presence of abundant positive Rolandic sharp waves (9,10). In addition, EEG patterns of preterm infants change with postmenstrual age (PMA) (11,12). In the very preterm infant, the EEG background activity is characterized by discontinuity, instability, and fragmentation (13). The greater the prematurity, the more marked are these EEG aspects. These characteristics make the interpretation of the tracings very complex.

As EEG has reproducible patterns in normal very preterm infants, this constitutes a basis for a quantitative analysis of EEG for future neurological prognosis in very preterm infants (14). Today, digital EEG recorders provide the opportunity to analyze the EEG quantitatively. Scher *et al.* (15) calculated quantitatively several specific EEG sleep measures with the objective to combine these measures into a single "dysmaturity index," *e.g.* which best expresses an infant's EEG sleep behavior relative to the average full-term infant. Among other sleep EEG measures, spectral power analysis of the EEG has the potential to discriminate sleep state between preterm and term infants. By using automated spectral analysis, Victor *et al.* (16) studied 75-min EEG recordings performed in 53 preterm infants ≤30 wk of gestation (no follow-up available) during the first 4 d of life. West *et al.* (17) analyzed changes in continuity, amplitude, and spectral edge frequency (SEF) with an automated algorithm in 60-min EEG recordings, performed daily in 63 preterm infants <32 wk of gestation (no follow-up available) during the first week of life. These studies suggest that spectral power analysis of the EEG is useful for brain monitoring and show consistent changes in several quantitative EEG measures over time in preterm in-

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Abbreviations: PMA, postmenstrual age (GA + postnatal age); **SEF,** spectral edge frequency

fants, indicating functional cerebral maturation. Among limitations of the last two studies are that relatively short recordings were performed during the first days of life with a small range in PMA and no data on long-term follow-up available. As the recordings of these studies were performed during the first week of life, the results may merely reflect the perinatal transition phase rather than postnatal maturation.

The aim of this study was to investigate if neurophysiologic maturation in very preterm infants can be described by automated spectral power analysis of whole 4-h EEG recordings and to identify specific changes of spectral power measures with maturation. We performed serial (weekly) 4-h EEG recordings in healthy preterm infants with a GA <32 wk and a normal neurodevelopmental follow-up at 2 y of corrected age.

METHODS

Study subjects. This study was part of a comprehensive EEG research program of Máxima Medical Center in Veldhoven, The Netherlands, and conducted in the NICU from May 2006 to July 2007. The hospital's ethics committee approved the study. Infants were enrolled after written informed consent from both parents. For details of the study group, we refer to earlier articles (18,19).

During the study period, 95 preterm infants were eligible for the study, on the basis of "first-day" inclusion criteria: preterm infants with a GA <32 wk, birth weight appropriate for GA, Apgar score ≥6 at 5 min, arterial umbilical pH >7.00, normal cerebral ultrasonography < 48 h after birth, and infants who were expected to stay longer than 4 wk in our hospital. Of the eligible population, 67 subjects were subsequently excluded at the end of the first week because of cardiovascular instability (requiring volume expansion and/or inotropic drugs), abnormalities on cranial ultrasound (intraventricular hemorrhage II–IV and all grades of periventricular leucomalacia) (20), and use of any sedative or antiepileptic medication, subjects with recurrent apnea treated with doxapram or no parental consent.

Thus, 28 infants were enrolled in the study. Of these, 10 subjects were excluded from analysis because they met exclusion criteria after the first week ($n = 8$) or showed delayed development at corrected age of 24 mo ($n = 2$). Finally, 18 clinically stable preterm infants with at least 4 weekly EEG recordings and a normal neurological follow-up at corrected age of 24 mo (Bayley Scales of Infant Developmental II for mental and motor function) were analyzed in this article. The characteristics of these subjects are shown in Table 1.

Data acquisition. Starting at the end of the first week of life, weekly digital EEG recordings (NicoletOne; Viasys Healthcare, Conshohocken, PA) were performed. After skin preparation (Nuprep Gel, D.O. Weaver, Aurora, CO), Ag/AgCl cup electrodes, filled with a conductive paste (Ten20, D.O. Weaver), were placed according to the international 10 to 20 reduced montage system (Fig. 1) (21). The digital EEG signal was sampled at 256 Hz and stored on a hard disk. Artifacts related to repositioning the infant or replacement of an electrode were removed when impedance value was >10 k[Omega] or total spectral power value was above empirical threshold value of $10 \times 10^3\ \mu V^2$ (<5% of data). All recordings took place from 0800 to 1200 h. During the 4-h recording, the infants were in prone or side position. Feeding and care for the infant was performed according to the normal routine of the NICU.

Data analysis. Spectral analysis was performed using Nicolette EEG analyzer software. Data were filtered using a 0.5 Hz high-pass filter and 30 Hz low-pass filter. Subsequently, data were divided into 4-s half overlapping segments. Each segment was multiplied by a Hamming window before fast Fourier transform. The frequency spectrum and frequency band powers were determined for each segment and averaged over four consecutive segments, yielding a 10-s time resolution. Finally, the frequency information was averaged over the complete 4-h recording, including periods of continuity and discontinuity, to obtain mean EEG frequency parameters per EEG recording.

Five channels were used for analysis (Fig. 1): frontal channel, Fp_1-Fp_2; central channel, C_3-C_4; occipital channel, O_1-O_2, and the left and right centrotemporal channels, C_3-T_3 and C_4-T_4. The frequency spectrum was divided into the following bands: delta 1 (0.5–1 Hz); delta 2 (1–4 Hz); theta (4–8 Hz); alpha (8–13 Hz); and beta (13–30 Hz).

Three aspects of the transformed signal were calculated automatically: 1) absolute power (defined as the integral of all powers within the frequency band, expressed in μV^2), 2) relative power (defined as the ratio of absolute band power to total power of all bands, expressed in percentage); and 3) SEF (defined as the frequency that delimits 95% of the power between 0.5 and 30 Hz).

Table 1. *Characteristics of study population (n = 18)*

Clinical characteristics	
GA (wk)	29.0 ± 0.3
Birth weight (g)	1297 ± 58
Female rate (%)	39
5-min Apgar score	8.5 ± 1.3
Umbilical arterial pH	7.28 ± 0.09
Tocolysis (%)	83
Antenatal corticoids (%)	94
Caesarean section (%)	28
Lowest pH, first week	7.26 + 0.04
Lowest glucose (mmol/l), first week	3.0 ± 1.1
MDI at 24 mo	106 ± 12
PDI at 24 mo	91 ± 11

Values expressed as mean ± SD, percentage, or median (range). During the first week of life, the lowest pH and serum glucose value of each subject were noted and expressed as mean ± SD.

MDI, Mental Psychomotor Developmental Index; PDI, Psychomotor Developmental Index.

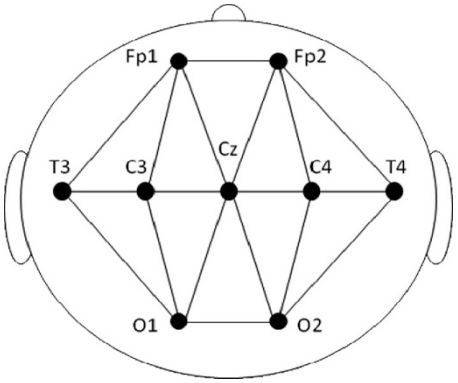

Figure 1. The international 10 to 20 reduced EEG montage system for neonates. From the EEG montage, the following channels were derived: frontal channel, Fp_1-Fp_2; central channel, C_3-C_4; occipital channel, O_1-O_2; left centrotemporal, C_3-T_3; and right centrotemporal, C_4-T_4.

Statistical analysis. Data with a normal distribution were expressed as mean ± SD, otherwise data were expressed as median and interquartile range (IQR). Data were statistically analyzed with SPSS 13.1 (SPSS Inc, Chicago, IL). The relations between PMA and EEG spectral measures were evaluated using multilevel analysis. Linear regression was estimated using the random effects model, which extends the generalized linear model to allow for analysis of repeated measurements between and within subjects. Regression analysis results are shown with the a- and b-coefficient (a, intercept; b, slope of regression line). A p value <0.05 was considered statistically significant.

RESULTS

Eighteen clinically stable infants (GA, 29.0 ± 0.3 wk; birth weight, 1297 ± 58 g) were studied weekly and underwent four to six EEG recordings (four recordings in $n = 13$ infants; five recordings in $n = 3$ infants; and six recordings in $n = 2$ infants). All 79 EEG recordings (representing a large variability in PMA, 27^{+4} to 36^{+3} weeks) were pooled into one dataset and analyzed with the generalized linear model. All EEG recordings showed the background patterns generally found in preterm infants: *tracé continue* (continuous EEG activity of similar voltage and frequency) and *tracé discontinue* (alternating segments of activity and quiescence). None of the EEG recordings showed evidence of neonatal seizures.

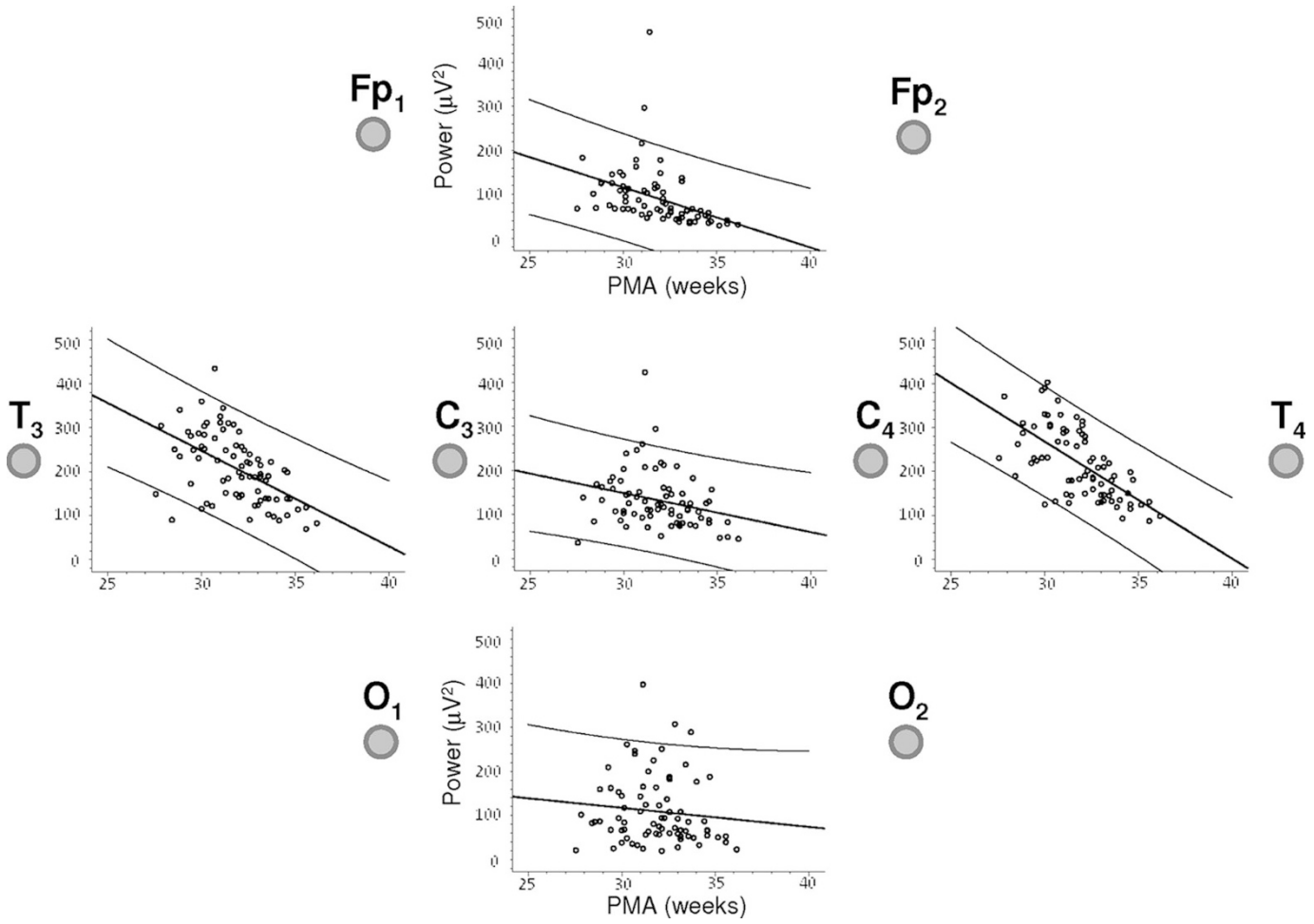

Figure 2. Regression plots for total spectral power as function of PMA for the different channels. The relationship between PMA (*x* axis, wk) and total spectral power (*y* axis, μV^2) is shown for five channels. The electrode positions are indicated by dots and accessory channel identification (frontal channel, Fp_1-Fp_2; central channel, C_3-C_4; occipital channel, O_1-O_2, and the left and right centrotemporal channels, C_3-T_3 and C_4-T_4). Linear regression lines are shown with 95% CI. Except for the occipital channel, the other channels showed significant slopes of the regression lines (b-coefficient ± SD): Fp_1-Fp_2, -13 ± 4 ($p < 0.01$); C_3-C_4, -9 ± 4 ($p < 0.05$); C_3-T_3, -21 ± 4 ($p < 0.01$); and C_4-T_4, -25 ± 4 μV^2/wk ($p < 0.01$).

Table 2. *Correlation between PMA and absolute spectral power measures in the different channels*

	Delta 1		Delta 2		Theta		Alpha		Beta	
	a ± SD (μV^2)	b ± SD (μV^2/wk)	a ± SD (μV^2)	b ± SD (μV^2/wk)	a ± SD (μV^2)	b ± SD (μV^2/wk)	a ± SD (μV^2)	b ± SD (μV^2/wk)	a ± SD (μV^2)	b ± SD (μV^2/wk)
Frontal	230 ± 70*	−6 ± 2*	250 ± 40*	−7 ± 1*	23 ± 7*	−0.5 ± 0.2†	9 ± 3*	NS	NS	NS
Central	280 ± 80*	−6 ± 2*	130 ± 40*	−3 ± 1†	8 ± 4†	NS	NS	NS	NS	NS
Occipital	NS	NS	100 ± 50†	NS	NS	NS	NS	NS	NS	NS
Left centrotemporal	620 ± 90*	−16 ± 3*	210 ± 40*	−5 ± 1*	50 ± 10*	−1.2 ± 0.3*	NS	NS	NS	0.2 ± 0.1†
Right centrotemporal	720 ± 80*	−18 ± 3*	270 ± 40*	−6 ± 1*	54 ± 7*	−1.4 ± 0.2*	NS	NS	NS	0.3 ± 0.1†

The frequency spectrum was divided in the following bands: delta 1 (0.5–1 Hz); delta 2 (1–4 Hz); theta (4–8 Hz); alpha (8–13 Hz); and beta (13–30 Hz). Linear regression was estimated using the random effects model, which extends the generalized linear model to allow for analysis of repeated measurements within subjects. The analysis was performed between PMA and absolute spectral power to assess the intercept (a-coefficient, μV^2) and spectral power change per week (b-coefficient, μV^2/wk).

* $p<0.01$; † $p<0.05$.

NS, not significant.

Absolute power. Figure 2 illustrates the changes of total spectral power with PMA for the different channels. In the frontal (Fp_1-Fp_2) channel, absolute spectral power measures in the delta and theta band decreased with PMA (Table 2). In the central channel (C_3-C_4), absolute delta spectral powers decreased, while theta, alpha, and beta powers remained unchanged. In the occipital channel, no changes in absolute spectral power measures were observed. In both centrotemporal channels (C_3-T_3 and C_4-T_4), absolute delta and theta spectral powers decreased, while beta power increased. The

Table 3. *Correlation between PMA and relative spectral power measures in the different channels*

	Delta 1		Delta 2		Theta		Alpha		Beta	
	a ± SD (%)	b ± SD (%/wk)	a ± SD (%)	b ± SD (%/wk)	a ± SD (%)	b ± SD (%/wk)	a ± SD (%)	b ± SD (%/wk)	a ± SD (%)	b ± SD (%/wk)
Frontal	65 ± 9*	−0.7 ± 0.3†	60 ± 7*	−0.6 ± 0.2*	−6 ± 2*	0.43 ± 0.07*	−7 ± 2*	0.31 ± 0.05*	NS	0.3 ± 0.1†
Central	88 ± 7*	−1.0 ± 0.2*	26 ± 5*	NS	NS	0.23 ± 0.07*	−5 ± 1*	0.23 ± 0.03*	−4 ± 1*	0.20 ± 0.03*
Occipital	57 ± 6*	NS	38 ± 4*	NS	4 ± 1*	NS	NS	NS	NS	0.12 ± 0.04*
Left centrotemporal	103 ± 8*	−1.5 ± 0.2*	NS	0.9 ± 0.2*	NS	NS	−6 ± 1*	0.25 ± 0.04*	−6 ± 1*	0.26 ± 0.04*
Right centrotemporal	98 ± 7*	−1.3 ± 0.2*	NS	0.8 ± 0.2*	5 ± 2†	NS	−6 ± 1*	0.25 ± 0.03*	−7 ± 2*	0.29 ± 0.05*

The frequency spectrum was divided in the following bands: delta 1 (0.5–1 Hz); delta 2 (1–4 Hz); theta (4–8 Hz); alpha (8–13 Hz); and beta (13–30 Hz). Linear regression was estimated using the random effects model, which extends the generalized linear model to allow for analysis of repeated measurements within subjects. The analysis was performed between PMA and relative spectral power to assess the intercept (a-coefficient, %) and relative spectral power change per week (b-coefficient, %/wk).

* $p<0.01$; † $p<0.05$.

NS, not significant.

greatest spectral power change per week was observed for the delta 1 spectral power measure in the centrotemporal channels. In general, absolute spectral power in the lower frequency range (<8 Hz) decreased with PMA.

Relative power. In the frontal channel, relative delta power decreased, while theta, alpha, and beta powers increased with PMA (Table 3). In the central channel, relative delta 1 power decreased, while theta, alpha, and beta relative spectral power measures increased with PMA, respectively. Except for a slight increase in relative beta power, other relative spectral powers remained unchanged in the occipital channel. In both centrotemporal channels, relative delta 1 power decreased, while delta 2, alpha, and beta increased with PMA. In general, relative spectral power measures showed a shift toward the higher frequency ranges with increasing PMA.

Spectral edge frequency. SEF increased with PMA in all channels, with the exception of the occipital channel (Table 4).

Table 4. *Correlation between PMA and spectral edge frequency in the different channels*

	Spectral edge frequency	
	a ± SD (Hz)	b ± SD (Hz/wk)
Frontal	NS	0.4 ± 0.1*
Central	−7 ± 2*	0.37 ± 0.07*
Occipital	NS	NS
Left centrotemporal	−7 ± 2*	0.37 ± 0.07*
Right centrotemporal	−7 ± 2*	0.36 ± 0.07*

Linear regression was estimated using the random effects model, which extends the generalized linear model to allow for analysis of repeated measurements within subjects. Analysis was performed to assess the intercept (a-coefficient, Hz) and frequency change per week (b-coefficient, Hz/wk).

* $p<0.01$.

NS, not significant.

DISCUSSION

We investigated spectral power changes with an automated algorithm based on fast Fourier transform in serial whole 4-h EEG recordings performed during the first 4 to 6 wk after birth. The preterm infants were clinically stable at time of the recording, and assessment at 24 mo showed a normal neurodevelopmental outcome. The main findings are that in normal preterm infants with increasing PMA, absolute spectral power in the lower frequency range decreases and relative spectral power measures show a shift toward the higher frequency ranges and SEF increases.

In general, our observation of decrease in absolute spectral power in the delta 1 (0.5–1 Hz) and delta 2 (1–4 Hz) band is consistent with others. Okumura *et al.* (22) demonstrated in 10 clinical stable preterm infants (29–34 wk of gestation, normal follow-up at 18 mo) a decrease of delta 1 spectral power in all channels with advancing age. Bell *et al.* (23) studied 20 healthy preterm infants (26–32 wk of gestation, normal follow-up at 18 mo) and demonstrated a decrease of absolute delta 1 and 2 spectral power with advancing age in the frontal (F_3-C_3 and F_4-C_4) and parietal (C_3-P_3 and C_4-P_4) channels. Scher *et al.* (24) studied maturational trends of EEG sleep measures in 56 healthy preterm infants (26–33 wk of gestation, normal follow-up at 24 mo) and demonstrated a decrease in absolute delta power during active sleep with advancing age. In the very preterm, EEG burst of slow delta waves are characteristic features which progressively disappear at 33 to 34 wk of gestation (13,14,25). The persistence of slow delta waves after 33 to 34 wk of gestation is related to adverse outcome (26). These studies and the present work suggest that maturation may be assessed by changes in the lower spectral frequency band (0.5–4 Hz) of the EEG.

Similar to Okumura *et al.* (27), we observed a decrease in absolute theta power with PMA in the frontal and centrotemporal channels. This observation is in agreement with the visual interpretation of the preterm EEG. High-voltage rhythmic temporal theta bursts appear frequently at 27 to 28 wk of PMA and diminish at 34 wk of PMA or older (12–14). These high-amplitude theta rhythms are characteristic of the temporal localization for very premature infants and are considered physiological (28). A decrease in theta spectral power with increase of PMA is considered an indicator of normality, while persistence of theta spectral power after 32 wk is associated with unfavorable neurological outcome (29).

Consistent with the article of Okumura *et al.* (27), we did not find significant differences in absolute spectral power changes in the alpha or beta frequency band. In contrast, others found absolute spectral power changes in the higher frequencies (8–30 Hz) with increase of PMA (23). The dif-

ferences in findings may be related to differences in EEG montage or selecting procedure of subsegments (23,27).

As total spectral power between subjects may vary considerably, spectral values are frequently normalized for total power and expressed as relative spectral power measures. We observed a decrease in relative delta 1 power and an increase in relative delta 2 power with increase of PMA in centrotemporal channels, respectively. The relative theta power increased in the central and frontal channels. The relative alpha and beta power increased in nearly all channels. Only the relative alpha spectral power remained unchanged in the occipital channel. In general, the changes in relative spectral power are in accordance with the study of Bell *et al.* (23). Other studies did not calculate relative spectral powers (22,24,27).

Our observations of spectral power analysis correspond with the EEG pattern of maturing preterm infants in whom EEG change from high-amplitude low-frequency waves to low-amplitude high-frequency waves (11,12,30). Total spectral power decreases with age with a shift from the lower to the higher frequency content of the EEG. The absolute and relative delta spectral power measures decrease, with increase of relative alpha and beta spectral power. As alpha and beta waves are complex EEG waveforms, these might be related to formation of connection between the cortex and subcortical nuclei and reticular formation, which mature postnatally (31).

In contrast to the majority of articles (22,23,27,32–36), we studied maturational changes of spectral power analysis quantitatively and automated on whole 4-h recordings, including several sleep-wake cycles. From a previous article using the same subjects, we observed periodic variation in background activity with alternating periods of discontinuity and continuity, suggestive for two to four sleep-wake cycles per 4-h recording (19). Bell *et al.* (23,33) performed spectral power analysis on eight 4-s epochs in each recording. Okumura *et al.* (22,27) analyzed six 10-s epochs in each recording. Okumura *et al.* selected EEG epochs where continuous patterns with maximal expression of high-voltage slow waves of preterm infants were continuously observed, whereas Bell *et al.* analyzed the EEG epochs during periods of relative continuity. Paul *et al.* (34,36) selected 5-min periods during active and quiet sleep. Thus, analyzed epochs are small and selected by qualitative (visual) criteria that differ between studies. Remarkably, despite these differences in analysis, we found comparable spectral power data. We consider the automated method of spectral analysis on whole 4-h EEG recordings as performed in this study as highly objective, less time consuming to perform in daily clinical practice, and generating comparable data.

In general, the amount of changes in total spectral power measures with increasing PMA were higher in the centrotemporal channels than in the other channels (Fig. 2; Table 2). This may be in agreement with imaging observations of the preterm brain, demonstrating that gyral development in parietotemporal area precedes that of other regions (37). A correlation between some aspects of EEG maturation and gyration of the preterm brain is suggested (38). With increasing PMA, the interburst interval shortened and cortical folding increased. For other spectral power measures, the correlation was less clear. Topographic mapping of changes in spectral power measures is an interesting technique to localize brain activity but is beyond the scope of this article (39).

Integrating spectral power analysis of the EEG during bedside continuous amplitude-integrated EEG monitoring in an NICU is an interesting option to assess brain maturation. For amplitude-integrated EEG, most advocate lead placement at the biparietal areas (P_3-P_4 channel), which overlies a vascular watershed (7). The closest corresponding electrode pair in neonatal EEG using the 10 to 20 montage is the adjacent bicentral area, represented by C_3-C_4 (23). As the spectral changes were most clearly seen in the centrotemporal channels, bilateral centrotemporal or parietotemporal channels may be valuable in continuous (spectral power) monitoring. Automated spectral power analysis of the EEG in preterm infants may provide easily accessible bedside information about brain function to the neonatologist and help to identify possible factors that interfere with normal brain development. Spectral power of low frequencies differs significantly between the burst episodes of healthy and asphyxiated infants (35). Lower SEF measures are shown in infants with later cerebral white matter injury on MRI (40). Less spectral beta energy is demonstrated in preterm infants who received skin-to-skin contact compared with a control group of preterm infants (41). In preterm infants, a correlation between increased power in the higher frequency range and normal behavioral outcome at 5 y of age is demonstrated (32). Recently, a relation between increased delta activity and chorioamnionitis-related brain injury is demonstrated in preterm lambs (42).

There are several methodological limitations of the study. First, the number of very preterm infants is relatively small. Of 95 eligible preterm infants, 28 enrolled the study and after later exclusion of 10 (sick or delayed neurodevelopment at 2 y of age), 18 (20%) were actually analyzed in this study. The aim of this study was to assess automated-derived spectral power EEG measures in healthy preterm infants without any additional pathologic conditions that might interfere with normal cerebral development. We consider a strength of the selection procedure that all included preterm infants showed normal neurodevelopment at 2 y of age and that spectral power values are obtained for "normal" preterm infants. Second, for this study, signal artifacts related to repositioning of electrodes or nursing care for the infants were removed based on an empirical impedance and spectral power threshold value. An acceptable percentage of remaining artifacts was confirmed visually. Third, our study did not address sleep state of preterm infants, and between-state differences were not analyzed. To maximize robustness of the method, we refrained from performing analysis on subsegments, to reduce bias based on user information and to avoid possible errors in sample selection. Finally, evaluating the complex and subtle maturation aspects of the neonatal EEG requires specific neurophysiological expertise. Automated power spectral analysis must be seen as an aid in estimating neurophysiological maturation in preterm infants.

In conclusion, we performed automated spectral analysis of serial 4-h EEG recordings in 18 healthy very preterm infants with a normal follow-up at 2 y of age. Neurophysiological

maturation of preterm infants can be demonstrated by automated spectral analysis. Maturational EEG changes assessed by amplitude spectral analysis are most prominent in the centrotemporal channels with a decrease of absolute delta frequencies (0.5–4 Hz). Relative spectral power measures show a shift from the lower toward the higher frequency ranges. The potential of computer-assisted analyses of serial EEG samples may be that using signal analytic features not readily identified visually and may provide an indicator of good prognosis or poor developmental outcome.

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