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Topography of Clonidine-induced Electroencephalographic Changes Evaluated by Principal Component Analysis

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Background: **Principal component analysis is a multivariate statistical technique to facilitate the evaluation of complex data dimensions. In this study, principle component analysis was used to reduce the large number of variables from multichannel electroencephalographic recordings to a few components describing changes of spatial brain electric activity after intravenous clonidine.**

Methods: **Seven healthy volunteers (age, 26 ± 3 [SD] yr) were included in a double-blind crossover study with intravenous clonidine (1.5 and 3.0 μg/kg). A spontaneous electroencephalogram was recorded by 26 leads and quantified by standard fast Fourier transformation in the δ, θ, α, and β bands. Principle component analysis derived from a correlation matrix calculated between all electroencephalographic leads (26 × 26 leads) separately within each classic frequency band. The basic application level of principle component analysis resulted in components representing clusters of electrodes positions that were differently affected by clonidine. Subjective criteria of drowsiness and anxiety were rated by visual analog scales.**

Results: **Topography of clonidine-induced electroencephalographic changes could be attributed to two independent spatial components in each classic frequency band, explaining at least 85% of total variance. The most prominent effects of clonidine were increases in the delta band over centroparietooiccipital areas and decreases in the alpha band over parietooccipital regions. Clonidine administration resulted in subjective drowsiness.**

Conclusions: **Data from the current study supported the fact that spatial principle component analysis is a useful multivariate statistical procedure to evaluate significant signal changes from multichannel electroencephalographic recordings and to describe the topography of the effects. The clonidine-related changes seen here were most probably results of its sedative effects. (Key words: Clonidine; electroencephalogram; pharmacodynamic; sedation; spatial analysis.)**

ELECTROENCEPHALOGRAPHY (EEG) has been described as one of the most sensitive methods to detect pharmacodynamic effects in brain electric activity.[1-3] Multichannel EEG recordings in combination with power spectrum analysis have been proposed for quantitation of changes in the topographic distribution of EEG patterns related to different states of alertness, sleepiness, and anesthesia.[4-8] The EEG "mapping" technique is a useful tool to visualize the topographic changes in brain activity.[9-11] For the evaluation of the effects seen in the color-coded brain maps, however, we need statistical methods. The statistical analysis of EEG data is complicated because of the limited number of subjects usually involved in pharmacologic studies. Because of the large number of tests necessary, the probability of incorrectly declaring an effect as significant increases. A convenient procedure to avoid this type of error is adjustment of the corresponding significance level (α level normally set at $P = 0.05$) downward by Bonferroni correction to ensure that the overall risk remains at $P = 0.05$. This means that effects had to be unrealistically strong to reach the level of significance. A possible strategy to overcome this problem is the reduction of tested variables.

Principal component analysis (PCA) often is used in data reduction to identify a small number of components that explain most of the variance observed in a much larger number of variables. PCA attempts to identify underlying variables, so-called principal components, that explain the pattern of correlations within a set of observed variables.[12] In electrophysiologic research, PCA already has been used to describe time series and wave-

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form patterns.[13-15] In multichannel recordings of electric potentials or magnetic fields, this technique has been used to estimate the number of sources within the brain that may explain the data measured over the scalp, and to separate signal from noise.[16,17] Spatial PCA multichannel EEGs may provide specific information about the spatial distribution of changes in spectral power densities at the surface of the brain.[13,18]

Clonidine has been shown to induce sedative effects that can be monitored by spontaneous EEG.[19-21] Recent investigations suggest that the effects may be most pronounced at parietooccipital sites,[22] but detailed evaluations are not available. The goal of the current study was to analyze the topographic distributions of pharmacodynamic effects of clonidine in spontaneous EEG by a multivariate statistical procedure.

Materials and Methods

After institutional approval (University of Hamburg, Hamburg, Germany) and written informed consent was obtained, seven adult healthy men (classified as American Society of Anesthesiologists physical status I; age, 26 ± 3 [SD] yr; weight, 76 ± 5 kg) participated in the study. All subjects were free from neurologic diseases and had not taken any centrally acting drugs. Clonidine was administered in a random double-blind crossover design on two different days with two different dosages intravenously (1.5 or 3.0 μg/kg) and an intercession interval of 1 week. The following variables were recorded: spontaneous EEG; subjective drowsiness and anxiety, as measured by visual analog scale; heart rate; oxygen saturation; and mean arterial blood pressure.

Experimental Sessions

Each session included eight experimental periods (habituation, baseline, and six posttreatment periods: 0-10 min, 15-25 min, 30-40 min, 45-55 min, 90-100 min, and 105-115 min). The habituation period was included to familiarize the subjects with the experimental setup and was excluded from evaluation. After baseline recordings, clonidine was given intravenously (2 min infusion time) after the start of the first period (0-10 min). To monitor the clonidine effects during a longer postmedication period (115 min), an interval of 35 min was performed between the 45-55 and 90-100 min periods. During this interval subjects were allowed to move.

EEG

The EEG was recorded by 30 Ag-AgCl electrodes placed according to the International 10-20 System referenced to linked earlobes.[23] For artifact control, an electrooculogram also was recorded from supra- and infraorbital leads. Bandpass filtering was set at 0.5 and 30 Hz (3-dB cutoff points, 24 dB/octave). The electrode impedance was kept less than 5 kΩ. EEG signals were inspected on-line to monitor the stability of the recording conditions. Spontaneous EEG was recorded continuously. Every 10 s, an EEG epoch 2.56 s in duration was digitized on-line with a sampling rate of 200 Hz and 12-bit resolution and stored on disk for later evaluation (SynAmps; Neuroscan, Herndon, VA). The EEG was evaluated off-line, and recordings contaminated by electrooculogram, myogenic noise, or other artifacts were eliminated before further data analysis. Power spectral density of each EEG epoch was calculated by fast Fourier transformation after tapering the time series with a Kaiser window[24] (40-dB side-lobe depression). Spectra were averaged over six successive EEG epochs, resulting in one mean spectra per minute. These spectra were quantified by computing the power within the classic frequency bands: delta (0.5-4.0 Hz), theta (4.0-8.0 Hz), alpha (8.0-12.0 Hz), and beta (12.0-30.0 Hz), using the trapezoidal rule as a linear interpolation technique.

Subjective Perceptions

To measure subjective perceptions of drowsiness and anxiety, visual analog scales (0-100) were used. The subjects were instructed to describe their perceptions before and 1, 2, and 4 h after drug administration.

Control Variables

Oxygen saturation and heart rate were monitored continuously (Jenacor, Jena, Germany) during the observation period and quantified every minute. To minimize artifacts resulting from measurements, blood pressure was measured intermittently before and after each experimental block noninvasively (Dinamap; Critikon, Tampa, FL).

Statistical Analysis

The one-sample Kolmogorov-Smirnov test was used to evaluate whether the observed values confirm to normal distribution. The spectral powers in the delta, theta, alpha, and beta bands were calculated for data from 26 EEG channels. The spatial distribution in each of these frequency bands was analyzed by PCA.[12] The data sets subjected to PCA consisted of 25,480 values in each frequency band (26 EEG channels × 7 subjects × 7

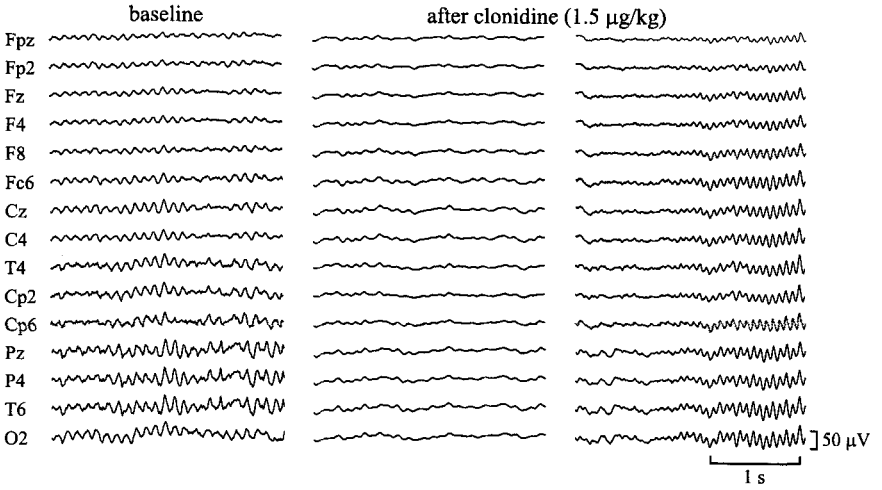

Fig. 1. Original tracings (15 electroencephalogram trails selected from 26 channels) from one subject during baseline measurements ([*left*] mixed electroencephalographic activity interspersed with alpha waves) and 25 min after administration of 1.5 μg/kg clonidine ([*middle*] slow wave activity with low amplitude; [*right*] sleeping spindles with frequencies of 12–14 Hz).

blocks × 10 values [sampled periods] in each block × 2 dosages). Spatial principal components were extracted from the correlation matrix between the EEG leads (26 × 26). Components with eigenvalues greater than 1.0 were extracted and transformed by varimax rotation[25] to principal rotated components (rPCs). To facilitate the interpretation of the spatial component, only component values greater than 0.7 were regarded as substantial. To describe the dosage effects with respect to the extracted spatial rPCs, the component scores were calculated. These scores quantify the manifestation of the corresponding principal component during the experimental conditions.

Data inhomogeneity in baseline values between runs was eliminated by subtracting differences between each postmedication value and the respective baseline value. These data were subjected to two-way analyses of variance using within-subjects factors (6 posttreatment blocks, 2 dosages). The Greenhouse–Geisser correction was used to take into account inhomogeneities of covariances. In case of significant effects in the analysis of variance, dosage effects were evaluated in detail by Bonferroni corrected paired *t* test comparison. The nonnormal distributed data from visual analog scale scores were tested by the Friedmann and Wilcoxon tests. $P < 0.05$ was considered to be significant. Statistical analysis was performed using the SPSS package (SPSS Inc., Chicago, IL).[26]

Results

Electroencephalography

Approximately 5% of total recordings were contaminated by electrooculogram, myogenics, or other artifacts and were eliminated from data analysis.

EEG baseline recordings were dominated by alpha waves. Clonidine administration resulted in an attenuation of the dominant alpha rhythms and an induction of slow wave activity and was associated with an intermittent appearance of sleeping spindles, characterized by 12- to 14-Hz wave activity (fig. 1). The EEG changes were dose-dependent, with longer-lasting effects after 3.0 μg/kg clonidine (over 115 min) than after 1.5 μg/kg (over 60 min).

Mapping of Multichannel Electroencephalography Activity. In a first attempt to describe the spatial effects of clonidine on the spontaneous EEG, the mean power values for each block before and after drug application were plotted separately for the chosen frequency bands as color-coded maps (fig. 2). The most prominent alterations in EEG were seen in the delta and alpha ranges. The delta power increases with bilateral symmetric distribution over the scalp. Alpha rhythms dominating during baseline, with maximal spectral power over parietooccipital areas that were attenuated after clonidine application. The two different doses of clonidine resulted in similar EEG changes, with slightly longer-lasting effects for the higher dosage. In the theta and beta bands, no visible drug-induced changes were registered.

Principal Component Analysis. To identify significant signal changes after clonidine from the color-coded EEG maps (fig. 2), within each frequency band the spectral power values of all EEG leads were subjected to spatial PCA. To analyze the dose-dependency of clonidine effects, data from both clonidine applications (1.5 and 3.0 μg/kg) were evaluated within the same PCA procedure.

The first step of the PCA was to analyze similarities of

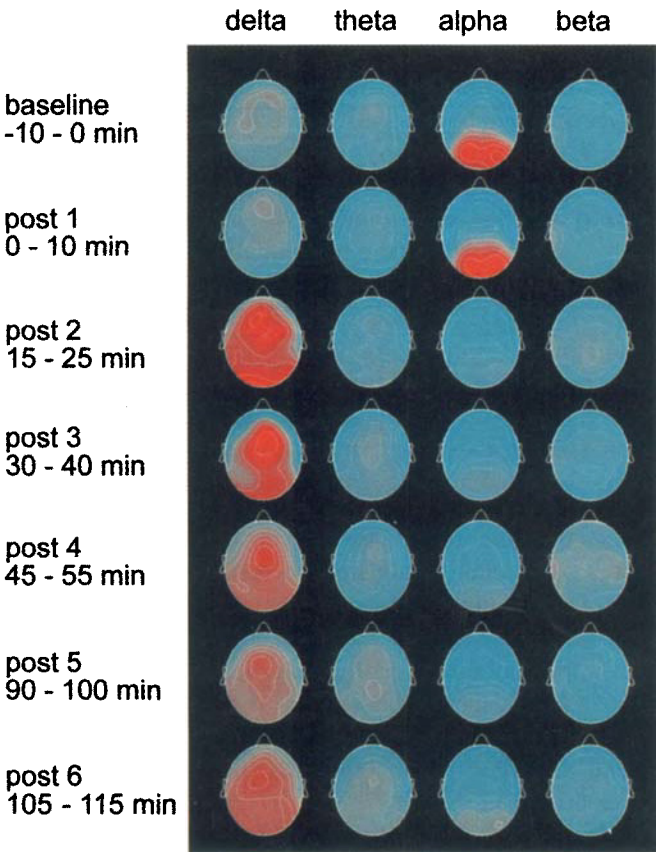

Fig. 2. Electroencephalographic maps representing spectral power of delta, theta, alpha, and beta bands. Data from seven subjects (after 3.0 μg/kg clonidine) were calculated from 26 electrode positions. Clonidine-related signal changes resulted in alpha suppression (between 15 and 115 min after administration) associated with increases in slow wave activity. Delta power was maximal between 15 and 25 min and 30 and 40 min after administration. Color codes range from no activity (blue) to maximal activity (red) for delta (30 μV^2), theta (30 μV^2), alpha (50 μV^2), and beta (15 μV^2) bands.

the data from the 26 EEG electrode positions, in order to detect patterns of high correlation. In each frequency band two uncorrelated spatial rPCs were calculated, explaining more than 85% of the variance. The spatial distribution of these rotated principal components is illustrated in figure 3, indicating selected electrode positions that are representative of the corresponding spatial components. The exact loadings of the spatial components for all electrode positions are given in table 1.

The second step of the PCA was to identify the dosage effects of clonidine on the defined spatial rPCs by computing the principal component scores. These rPC scores reflect the degree to which each spatial rPC is affected by both clonidine dosages during the observation period. The two-way analysis of variance (2 dosages, 6 postmedication blocks) indicates that only two spatial components (δrPC_2 and αrPC_2) were affected significantly by clonidine (table 2). The time course of the mean values of the principal component scores in the delta and alpha bands is plotted in figure 4. A more detailed statistical evaluation of drug-induced changes during the postmedication periods within dosages is given in table 3.

Clonidine-associated increases in delta over centroparietooccipital areas were indicated by δrPC_2. The appearance of delta was accompanied with a suppression of the most prominent activity (alpha) during baseline recordings. Alpha changes were distributed over parietooccipital regions represented by αrPC_2. After both clonidine applications (1.5 and 3.0 μg/kg), EEG changes in delta and alpha were significant during the postmedication periods between 15 and 25, 30 and 40, and 45 and 55 min). Patients regained baseline values between 90 and

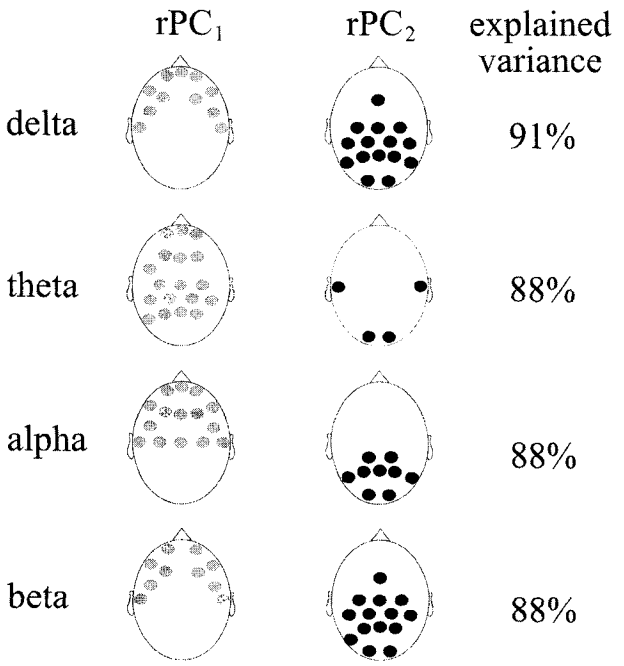

Fig. 3. Results from decomposing the electroencephalogram (EEG) spectral power into spatial components by principal component analysis. After each dosage (1.5 and 3.0 μg/kg) of clonidine, in each frequency band two independent topographic patterns of EEG activity were identified. Points denote electrode positions with high loading (≤ 0.7; table 1) in spatial rotated components 1 (rPC_1) and 2 (rPC_2). EEG activity in the selected frequency bands is topographically and not homogenously distributed but exhibits different spatial patterns. For example, the EEG in the alpha band could be divided in distinct frontal and occipital activity, which do not correlate with each other.

Table 1. Factor Loadings for Each Electrode Position* Calculated for the Components of the Selected Frequency Bands

	Electrode Sites																								
Frequency	Fp1	Fpz	Fp2	F7	F3	FZ	F4	F8	FC5	FC6	C3	CZ	C4	CP1	CP5	CP2	CP6	T3	T4	T5	T6	P3	PZ	P4	O1	O2
δ rPC_1	**0.87**	**0.79**	**0.89**	**0.83**	**0.74**	0.57	**0.75**	**0.86**	**0.73**	**0.72**	0.53	0.47	0.54	0.42	0.56	0.42	0.57	0.70	**0.74**	0.58	0.55	0.45	0.40	0.45	0.52	0.50
δ rPC_2	0.38	0.57	0.38	0.47	0.62	**0.74**	0.59	0.42	0.62	0.62	**0.79**	**0.80**	**0.80**	**0.88**	**0.79**	**0.89**	**0.78**	0.63	0.50	**0.72**	**0.75**	**0.87**	**0.90**	**0.86**	**0.76**	**0.79**
θ rPC_1	0.71	**0.78**	**0.71**	0.66	**0.77**	*0.83*	**0.75**	0.61	**0.76**	0.58	**0.87**	**0.90**	**0.83**	**0.91**	**0.85**	**0.89**	**0.78**	0.45	0.27	**0.75**	0.64	**0.79**	**0.87**	**0.72**	0.42	0.37
θ rPC_2	0.60	0.55	0.60	0.66	0.57	0.46	0.56	0.69	0.58	0.69	0.43	0.39	0.49	0.37	0.46	0.40	0.56	**0.76**	**0.80**	0.55	0.68	0.51	0.42	0.59	**0.80**	0.81
α rPC_1	**0.92**	**0.86**	**0.90**	**0.79**	**0.88**	**0.79**	**0.85**	**0.85**	**0.83**	**0.87**	**0.73**	**0.70**	**0.77**	0.57	0.63	0.58	0.60	**0.76**	**0.74**	0.36	0.53	0.36	0.46	0.34	0.27	0.35
α rPC_2	0.24	0.45	0.30	0.37	0.43	0.55	0.48	0.39	0.49	0.41	0.62	0.64	0.56	**0.77**	0.69	**0.76**	0.69	0.47	0.32	**0.86**	**0.74**	**0.91**	**0.86**	**0.90**	**0.89**	**0.88**
β rPC_1	**0.86**	0.69	**0.85**	**0.83**	**0.73**	0.56	**0.70**	**0.81**	**0.79**	**0.75**	0.54	0.39	0.49	0.29	0.61	0.28	0.51	**0.84**	**0.86**	0.61	0.54	0.30	0.22	0.28	0.50	0.49
β rPC_2	0.27	0.63	0.38	0.43	0.59	**0.75**	0.62	0.45	0.53	0.58	**0.80**	**0.87**	**0.83**	**0.94**	**0.75**	**0.94**	**0.80**	0.20	0.15	**0.70**	0.68	**0.93**	**0.96**	**0.93**	**0.79**	**0.78**

High loadings (> 0.7) are in bold.

* n = 26.

100 or 105 and 115 min after the 1.5-μg/kg clonidine dosage; 3.0 μg/kg resulted in longer-lasting drug effects still present at the end of the observation period.

Concomitant Variables

Subjective Perceptions According to the Kologorov–Smirnov test data, subjective perceptions were nonnormally distributed, and they were evaluated by nonparametric Friedmann and Wilcoxon tests. Clonidine administration resulted in maximal subjective drowsiness 1 h after clonidine application. This effect was more pronounced after 3.0 (+260% *vs.* baseline) than after 1.5 μg/kg (+144% *vs.* baseline) clonidine and lasted until 4 h after drug infusion; criteria of anxiety were unaffected.

Cardiovascular and Respiratory Parameters Oxygen saturation and mean arterial blood pressure did not change from baseline values during the observation periods. The heart rate, however, was statistically affected by the dosages ($P < 0.05$ *vs.* baseline), exhibiting a slight initial increase and a slight decrease at the end of the observation period (table 3).

Discussion

The EEG has been described as a suitable technique to detect pharmacodynamic effects on brain electric activity.[1,2] This may explain why electrophysiologic variables are being used more commonly in clinical practice to monitor the target organ of anesthesia. Recently, clinical interest in the processed EEG in particular has increased, to detect inadequate and adequate states of depth of anesthesia.[27] The most common variables of the processed EEG are the spectral edge frequency, median frequency, power in the classic frequency bands, and bispectral index.[28] Correlations between changes in spontaneous brain electric activity and alterations in the state of consciousness have allowed development of closed feedback loops to control the doses of anesthetics needed to maintain anesthesia successfully.[29,30]

The interpretation of drug-induced EEG alterations is difficult because the EEG activity is not distributed homogeneously over the scalp. To our knowledge, there is no processed EEG variable available that takes into ac-

Table 2. Two-way Analysis of Variance for Clonidine-induced Changes from Baseline

	Dosage			Block			Dosage × Block		
	F	DF	p(F)	F	DF	p(F)	F	DF	p(F)
δ rPC_1	0.03	1,6	NS	1.73	1.5,8.8	NS	0.82	1.9,11.4	NS
δ rPC_2	0.07	1,6	NS	7.39	2.7,16.4	<0.01	2.27	2.7,15.9	NS
θ rPC_1	1.05	1,6	NS	1.62	1.8,10.6	NS	0.18	2.3,13.5	NS
θ rPC_2	0.03	1,6	NS	2.44	1.5,9.2	NS	1.09	1.4,8.6	NS
α rPC_1	1.75	1,6	NS	1.08	2.5,15.2	NS	2.09	2.8,16.6	NS
α rPC_2	2.39	1,6	NS	7.70	2.6,15.4	<0.01	1.40	1.7,10.5	NS
β rPC_1	1.93	1,6	NS	2.47	2.5,15.1	NS	0.14	2.7,16.2	NS
β rPC_2	0.43	1,6	NS	1.28	1.8,10.8	NS	1.23	2.7,15.9	NS
Heart rate	0.08	1,6	NS	8.73	1.9,11.6	<0.01	1.38	2.8,16.9	NS
SaO_2	1.02	1,6	NS	2.17	2.4,11.2	NS	1.82	2.8,16.8	NS
MAP	2.06	1,6	NS	1.81	1.2,7.4	NS	0.82	1.1,6.6	NS

NS = not significant; MAP = mean arterial pressure.

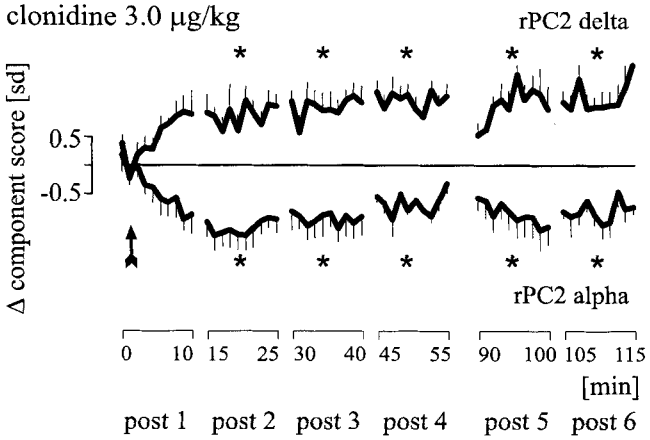

Fig. 4. Time-related changes of spatial EEG components (fig. 3) in the delta and alpha bands after treatment with 3.0 μg/kg clonidine. Given are mean principal component scores for 1-min intervals and their standard errors. The scores describe changes in the size of spectral power spatial patterns from baseline to postmedication periods. Clonidine exhibits topographically different effects, statistically significant (*) in the alpha and delta frequencies (table 2). For example, the spatial rotated component (rPC_2) of alpha (black line), representing parietooccipital activity, was decreased after 3.0 μg/kg clonidine below baseline values during the whole observation period.

count information about topographic distribution of EEG signal changes. The dynamics of topographic distributions of brain electric activity can be visualized using EEG mapping techniques. Unfortunately, this technique indicates signal changes only on visual inspection. One important problem of the evaluation of EEG maps recorded from multichannel EEG recordings is statistical handling of the large amount of data with respect to electrode position, power in frequency bands, and different time points. One technique commonly used to reduce the complexity of multichannel EEG data is to evaluate *a posteriori* only a subset of variables with an accidental selection or one region of interest. For example, in a previous clonidine EEG study[22] data along one frontoocciptial line (F4, C4, P4, and O2) from 16-channel recordings were calculated because of the interest in anteroposterior distributions of signal changes. This procedure may miss significant information regarding possible lateralization; alternative statistical procedures using the information within total data sets would be desirable.

One possible method to analyze all data from multichannel recordings is a reduction of the large number of variables by PCA, resulting in smaller numbers of uncorrelated "variables," so-called principal components.[12,31] PCA already has been used in electrophysiologic research to describe main waveform patterns of the EEG and evoked potentials.[13,32,33] PCA also has been used to analyze the spatial distribution of brain activity from different electrode positions.[18,34] Tanaka and coworkers,[18] for example, investigated the principal component structure of different sleep stages in 10 healthy men. This study demonstrated that PCA-extracted brain areas were topographically in concordance with visualized results from EEG mapping techniques. PCA is a possible strategy for analysis of complex signal information from multichannel recordings. There is evidence from a study of concordance between PCA-analyzed multichannel maps evoked by lateral visual half-field stimuli and their respective intracerebral electric field distributions that PCA may reflect a functional organization of brain electric activity.[34]

In the current study, PCA was used to evaluate multichannel EEG recordings after administration of two

Table 3. Significance of Clonidine Effects within Runs. Clonidine-induced Changes from Baseline during the Six Posttreatment Blocks

	Post 1		Post 2		Post 3		Post 4		Post 5		Post 6	
	p (F)		p (F)		p (F)		p (F)		p (F)		p (F)	
δ rPC_2												
clon *versus* baseline	NS		<0.05	+	<0.001	+	<0.001	+	NS		NS	
CLON *versus* baseline	NS		<0.01	+	<0.01	+	<0.01	+	<0.01	+	<0.001	+
α rPC_2												
clon *versus* baseline	NS		<0.001	−	<0.05	−	<0.05	−	NS		NS	
CLON *versus* baseline	NS		<0.001	−	<0.001	−	<0.01	−	<0.001	−	<0.001	−
Heart rate												
clon *versus* baseline	<0.001	+	NS		NS		NS		<0.001	−	<0.001	−
CLON *versus* baseline	<0.05	+	<0.001	−	NS		NS		<0.001	−	<0.001	−

Only in case of significant treatment effects of the corresponding analysis of variance (see table 2) the Bonferroni corrected post hoc *t* test was performed. clon = 1.5 μg/kg; CLON = 3.0 μg/kg; NS = not significant; + = increases above baseline; − = decreases below baseline.

different dosages of clonidine. One main result was the decomposition of the spatial distributions of spectral power values in different frequency bands. EEG registered from 26 electrodes resulted into only two major spatial rPCs within each frequency band, which accounted for more than 85% of variance. The most prominent EEG effect of clonidine was an attenuation of the dominant alpha rhythms associated with increases of power in the delta frequency range. These effects were not distributed homogeneously over the entire scalp. The PCA-related attenuation of alpha was significant only at parietooccipital sites represented by the spatial αrPC_2; slow wave activity increased significantly over centroparietooccipital leads represented by δrPC_2. In a controlled previous study with a comparable study design, no significant changes were observed after placebo.[35] We conclude that the EEG changes in the current study were not affected, for example, by spontaneous sleep behavior and most probably result from a clonidine effect.

In light of findings from several studies,[4,36-38] the clonidine-related EEG findings most probably represent sedative effects. The electrophysiologic correlates of sedation or decreases in vigilance already had been described in terms of EEG slowing and an attenuation of the dominant alpha rhythm.[1,39,40] In the current study, the findings in visual analog scale scores would support these interpretation.

In this study it was shown that PCA is an elegant multivariate statistical technique to replace the information from multichannel EEG recordings with a few independent principal components, minimizing loss of information. The restriction to using only the most important spatial components acts as a variation-reducing technique, relegating most of the random variation of the data to those components not extracted and collecting the most important information about spatial organization in the extracted ones. The most prominent principal components did not necessarily carry information about the experimental effects. The PCA technique does not clarify physiologic relations but only exposes statistical correlations between data. PCA is data-driven and dependent on the selected variables. The selection of variables is the most important decision.

In the current study PCA was applied at its most widely used basic level: computing the eigenvectors of the intercorrelation between EEG leads, extracting only eigenvectors with eigenvalues more than 1, with a final varimax rotation of the extracted principal components. Further variations of PCA are possible; *e.g.*, the varimax rotation could be replaced by another kind of rotation or no rotation at all. In the current study the varimax rotated solution exhibited a clearer spatial distribution than the unrotated one. Further studies are needed to gain more experience in the specific way to apply PCA to complex EEG data dimensions from multichannel recordings. At this time, spatial PCA should not replace visual signal inspection and mapping techniques, but it is a useful multivariate statistical tool to analyze statistically brain areas with significant signal changes. Further studies should evaluate whether PCA can provide a processed EEG variable desirable to monitor in clinical practice.

In conclusion, PCA is a multivariate analysis technique to support the evaluation of changes of topographic brain activity. In this clonidine study, data from 26 electrode positions were reduced by spatial PCA to only two variables quantifying two differently behaving cortical areas per EEG band, which explained more than 85% of total variance. One major advantage of PCA is that data derived from cortical areas may provide more realistic information about the functional state of the brain than data chosen by accidental selection. Qualitatively, the EEG changes seen here, represented by an attenuation of the dominant alpha rhythms (parietooccipital) and increases in slow wave activity (centroparietooccipital), most probably were caused by clonidine-related sedative effects.

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