

The Deleterious Effect of Ocular Artefacts on the Quantitative EEG, and a Remedy

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Summary. The effect of ocular artefacts on spectral EEG parameters is assessed statistically. These artefacts are caused by movements of the eyeball and/or of the lid. Further, methods for correcting ocular artefacts are presented and evaluated. This methodological study is based on data from an investigation comparing the EEG of schizophrenic patients ($n = 17$) with healthy controls ($n = 15$). Ocular artefacts are monitored by the bipolar vertical and the bipolar horizontal electro-oculogram (EOG). It is shown that the influence of ocular artefacts on the measured electrical activity in the frontal region is larger than the cerebral potentials which the EEG is ideally intended to record. The more frequent occurrence of blinks and eye movements in schizophrenic patients may lead to an artificial enhancement of slow frequency EEG power for schizophrenics and eventually "false significances". In contrast to this, we found more significant group differences when correcting for EOG artefacts than without it. This can be attributed to a very much inflated sample variability of the uncorrected EEG, due to the individually varying EOG power. We conclude that it may not be sufficient to select visually epochs for analysis that are considered artefact-free. Rather, one should monitor EOG artefacts and apply an appropriate correction.

Key words: EEG analysis – Ocular artefacts – Statistical methods

Introduction

Eye movements and blinks of the eye-lid generate large electric potentials which are transferred in attenuated form to the EEG electrodes. Their influence is particularly strong in the slow bands delta and theta, and at anterior sites. In fact, the size of ocular EEG artefacts decreases in the anterior-posterior direction for vertical artefacts and increases with increasing distance from the midline for horizontal artefacts. Since the extent of ocular artefacts may vary systematically between different

experimental conditions, psychophysicists have tried for a long time to cope with this problem (see Brunia et al. 1989 for an extensive report). Psychiatrists have usually not paid sufficient attention to this problem, with few exceptions (Karson et al. 1988), despite an increasing interest in quantitative EEG analysis. The literature shows that slow bands and anterior brain sites are often of central interest in psychiatric research. Ocular artefacts can thus not be discussed as an academic problem, given the expectation that psychiatric patients may have a higher rate of eye movements and blinks.

The purpose of this paper is two-fold:

1. We wish to quantify the extent and the effects of ocular artefacts on quantitative EEG analysis for a controlled study in schizophrenia. This should alert psychiatrists, as well as neurologists, to paying sufficient attention to this problem.
2. Methods for correcting vertical and horizontal electro-oculogram (EOG) artefacts together are described. The subsequent changes in the distribution of EEG power across frequency bands and across topography are assessed and examples of the potential advantages for psychiatric research are given.

These methods for EOG correction, based on principles of signal analysis, have been previously developed and validated in the neurophysiological literature (Gasser et al. 1985, 1986; Brunia et al. 1989, pp 21–26). The data are based on an investigation on schizophrenia to be described in a companion paper (Gattaz et al. 1992). As a consequence, the evaluation is realistic, since it is not based on data selected for methodological purposes.

Subjects and Methods

Subjects. Samples of 17 schizophrenic patients and of 15 healthy controls entered into the evaluation; they are described in detail in Gattaz et al. (1992).

Recordings. The monopolar EEG was recorded against linked shunted earlobes at 14 locations of the 10–20 system ($F_7, F_3, F_4, F_8, C_3, C_z, C_4, T_3, T_4, T_5, P_z, T_6, O_1, O_2$). In order to monitor movements of the eyes and of the lid, the vertical and the horizontal EOG was recorded bipolarly above and below the right eye and at

the left and right outer canthi, respectively. Beckmann Ag/AgCl electrodes and Grass ECII self-adhesive cream were used. Data were amplified by a Nihon Kohden 4321 G with a time constant of 1 s and a lowpass filter at 70 Hz. After a period of accommodation, and when the paper recording showed a good quality, 120 s of EEG at rest, eyes closed, were recorded. By a visual judgement good to excellent recordings resulted from this procedure. After digitizing online with a frequency of 200 Hz, data were processed on an IBM 3090 at the University of Heidelberg.

Statistical Processing. A major problem in quantitative EEG analysis is the selection of epochs of good data, reflecting brain activity in a relaxed, awake state ("EEG at rest"), with minimal influence by artefacts. The length of epochs has also been open to debate. While a long epoch is desirable for statistical reasons, this aim may conflict with the aim of having roughly the same relaxed, awake state for the whole epoch, and with the aim of minimizing artefacts. This is particularly true for psychiatric patients. Our procedure follows a previous evaluation by Möcks and Gasser (1984). The 120 s of data available were subdivided into 11 overlapping epochs of 20 s. The one epoch with minimum ocular artefacts was chosen for further analysis, operationalized as minimum EOG power in the band 1.5–7.5 Hz. This is an operational procedure to obtain at the same time the "EEG at rest" and a minimization of EOG artefacts, and other artefacts of slow frequency type. This is an alternative to the selection usually done by eye. However, in this as in other studies the automatic choice of epoch was checked visually, with good agreement between the objective and the subjective choice. Inclusion of a quantitative criterion reflecting the extent of high frequency contamination by muscle activity did not offer advantages in the previous evaluation. However, it might be useful to include it in some studies, for example as power in a frequency band ranging from 35 to 70 Hz, excluding the 50 Hz line.

The length of epoch of 20 s is smaller than the practice adopted in many empirical investigations. However, longer epochs did not improve the quality of the statistical analysis (Möcks and Gasser 1984), for reasons outlined above. It should also be noted that the individual power in a frequency band of 2 Hz has a variability proportional to a χ^2 variable with 40 degrees of freedom for 20 s epochs. This guarantees rather stable intraindividual values for broad band power. The choice of epochs of rather short duration is also supported by work studying how stationary are EEG time series (Isaksson and Wennberg 1976; Kawabata 1976). These authors found that the EEG can be considered stationary for periods up to about 40 s.

Spectral band power was then computed for all derivations in the following bands: delta 1.5–3.5 Hz, theta 3.5–7.5 Hz, alpha₁ 7.5–9.5 Hz, alpha₂ 9.5–12.5 Hz, beta₁ 12.5–17.5 Hz, beta₂ 17.5–25.0 Hz, beta₃ 25.0–49.0 Hz. These band power values were computed for further comparison before EOG correction, after correcting for the vertical EOG, and after correcting for the vertical and the horizontal EOG.

EOG Correction. This evaluation is based on our algorithm presented in Brunia et al. (1989, pp 21–26) and also uses our previous experience with vertical EOG correction (Gasser et al. 1985, 1986). The validity and the effectiveness of the method relies on general statistical principles and was also checked via data-based comparisons in our previous papers. The following simple model assumes that – because of attenuation – a fraction of the EOG amplitude is present at an EEG derivation:

$$\text{MEEG}(t) = \text{TEEG}(t) + \beta_V \text{VEOG}(t) + \beta_H \text{HEOG}(t)$$

where MEEG and TEEG denote the measured EEG and the true cerebral EEG respectively, and VEOG and HEOG the vertical (and horizontal) EOG at time t . This model is assumed to hold for each individual with individually differing coefficients β_V and β_H . It is a simplification in so far as a frequency-independent transfer from the EOG to the EEG electrode is assumed, but the effect of the simplification on the results of the correction is not dramatic (Gasser et al. 1985, 1986). This equation represents a multiple linear

regression with the EOG as the predictor (or the design) and the TEEG as the noise term. The β_V and β_H are the individual transmission coefficients to be estimated. The vertical and horizontal transmission coefficients β_V and β_H represent the fraction of EOG amplitude still present at some EEG derivation. Owing to the electrical resistance of the head the VEOG, for example, is attenuated more and more from anterior to posterior. Thus EOG correction consists of the following steps:

1. Low-pass filtering of the EOG derivations to account partially for the frequency dependence of the EOG-EEG transfer: a smooth low-pass filter, descending between 7.5 and 9.5 Hz like a cosine bell from 1 to 0 has been used.
2. Multiple linear regression analysis to estimate β_V and β_H for each EEG derivation and each subject: all data of 120 s are used for statistical reasons (note that transfer cannot be well estimated in epochs with no EOG power). This is done in a segmented way to facilitate data management.
3. EOG correction is then performed by subtracting a fraction of VEOG and HEOG from the MEEG.

$$\text{MEEG}(t) - \beta_V \cdot \text{VEOG}(t) - \beta_H \cdot \text{HEOG}(t)$$

Three different methods are evaluated which differ in the application of coefficients β_V , β_H :

1. The sample average coefficient β_V , β_H are used for each subject.
2. The individual coefficients are used.
3. "Winsorized" coefficients are used, where for the coefficients close to the median, the individual coefficients are used. The 25% lowest and the 25% highest coefficients are put to the lower and upper quartile.

As shown in previous investigations, transmission coefficients vary systematically from individual to individual owing to anatomical and physical differences of the heads (Elbert et al. 1985). On the other hand these transmission coefficients are statistically not well determined and show a large random variability. Thus, the use of the more stable average coefficients was superior to the use of individual coefficients in our previous investigations. The use of "winsorized" coefficients presents a compromise between these two possibilities.

Results

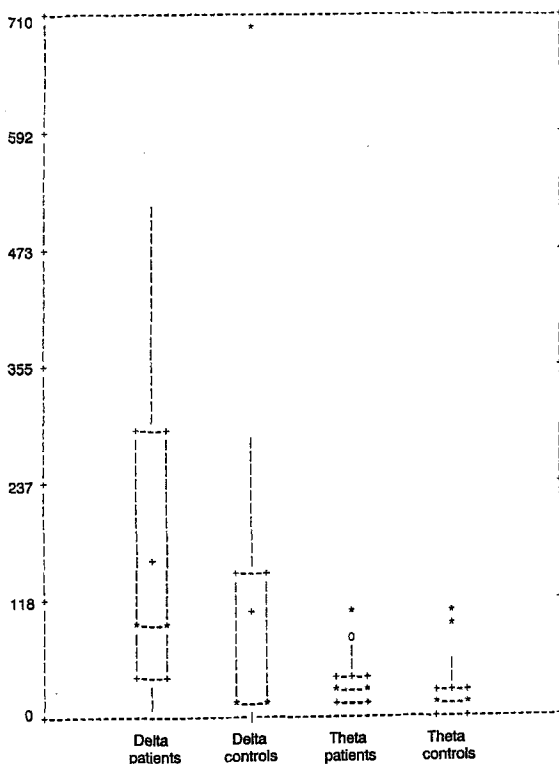
Table 1 shows means and standard deviations of the individual estimated transmission coefficients β_V , β_H . Statistics are given separately for patients and controls. The vertical coefficients decreased from anterior to posterior sites and the horizontal ones with increasing distance from the midline, as to be expected biophysically.

The coefficients showed roughly the same average size in both groups. Further evaluation will be restricted to the derivations F₇ to T₄.

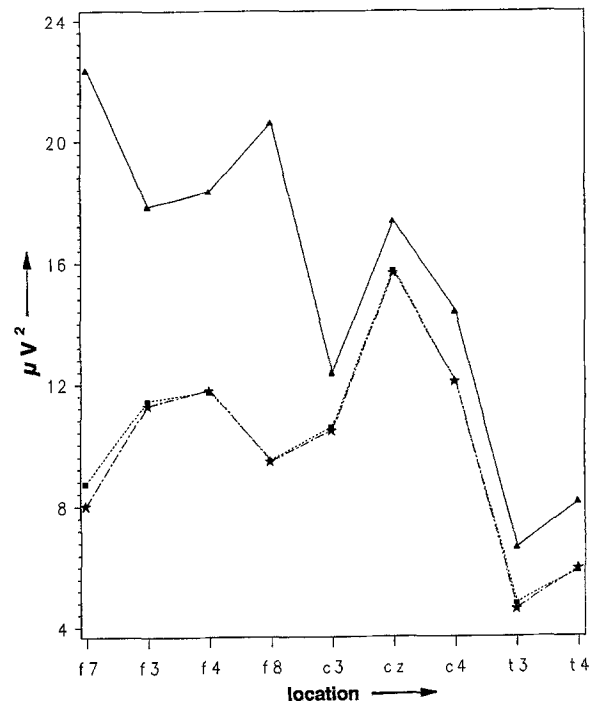
Next, movements of the eye and of the lid are quantified, reflecting the size and frequency of ocular artefacts. Schizophrenic patients had higher mean and median vertical and horizontal EOG power in the delta and theta bands. This difference was statistically significant in the theta band ($P \leq 0.05$) and showed a trend ($P \leq 0.10$) in the delta band. A box plot (Fig. 1) illustrates the effects. Ocular artefacts have a large influence on activity recorded at EEG sites. Figure 2 shows delta band EEG power for the schizophrenic group before and after correcting for EOG ocular artefacts. Large reductions in mean band power took place, in particular at the frontal line. There, the topographic pattern changed completely. This is due to the heavier corruption by horizontal

Table 1. Transmission coefficients for vertical (VEOG) and horizontal (HEOG) EOG for 17 schizophrenic patients and 15 normal subjects: tabulated are means and standard deviations

	Schizophrenic patients				Control group			
	Vertical		Horizontal		Vertical		Horizontal	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
F7	0.21	0.13	-0.27	0.23	0.21	0.06	-0.29	0.08
F3	0.17	0.07	-0.08	0.04	0.20	0.04	-0.09	0.06
F4	0.17	0.06	0.07	0.03	0.21	0.05	0.08	0.04
F8	0.19	0.09	0.17	0.23	0.24	0.06	0.25	0.14
C3	0.10	0.07	-0.05	0.04	0.11	0.03	-0.07	0.04
Cz	0.09	0.05	-0.00	0.04	0.11	0.04	-0.00	0.03
C4	0.10	0.06	0.04	0.03	0.11	0.03	0.06	0.02
T3	0.05	0.05	-0.11	0.09	0.07	0.02	-0.12	0.04
T4	0.06	0.04	0.10	0.05	0.08	0.02	0.14	0.03
T5	0.06	0.05	-0.06	0.10	0.06	0.02	-0.04	0.04
T6	0.08	0.10	0.05	0.06	0.08	0.05	0.06	0.03
Pz	0.07	0.05	0.00	0.03	0.08	0.03	-0.00	0.05
O1	0.05	0.08	-0.01	0.04	0.05	0.02	-0.01	0.03
O2	0.06	0.07	0.00	0.05	0.06	0.05	0.03	0.04

**Fig. 1.** Box plots of delta (left) and theta (right) power of vertical electro-oculogram (EOG) for schizophrenic patients ($n = 17$) and controls ($n = 15$) (box = lower and upper quartile, *--* = median, + = mean; dashes and stars outside boxes indicate distribution outside quartiles)

eye movements at F₇ F₈. The differences before and after "treatment" (i.e. EOG correction) were of course highly significant. The quantitative effects were even more dramatic in terms of reduction in variability (Fig. 3). Impressive reductions in mean and standard deviation of delta band power were also found for the normal group.

**Fig. 2.** Topographic profile of delta power: plotted is mean for schizophrenic patients ($n = 17$) without EOG correction (solid line and \blacktriangle), with EOG correction based on sample transmission coefficients (dotted lines and \blacksquare) and based on winsorized individual coefficients (dotted line and $*$)

These effects are particularly remarkable since the epochs have been selected for their low EOG power (see Methods). Using average or winsorized EOG transmission coefficients made hardly any difference (Figs. 2, 3). Individual coefficients led to somewhat less plausible results in the evaluation. The evaluation was also based on rank correlations between EOG and EEG power and on considering variability before EOG correction and when ap-

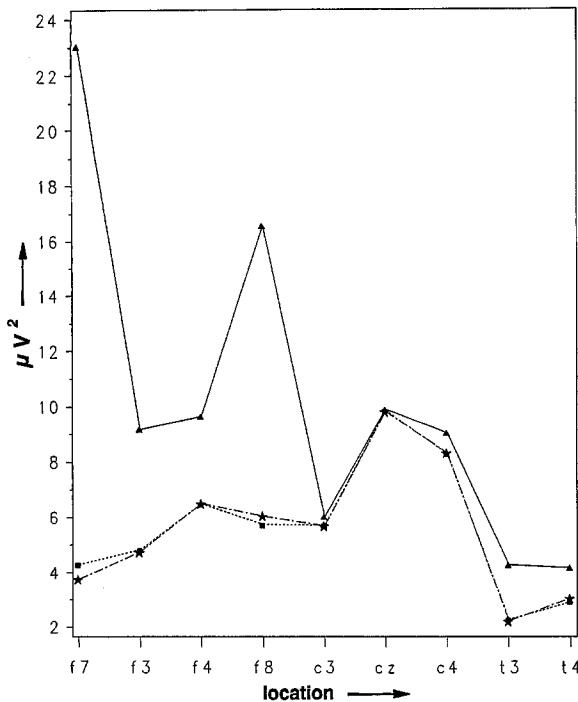


Fig. 3. Topographic profile of delta power: plotted is standard deviation for schizophrenic patients as for means in Fig. 2.

plying EOG correction based on average, individual or windorized coefficients. Both the correlations and variability should be reduced to a maximum extent by a good EOG correction method.

The question then arises how EOG correction affects the statistical comparison of schizophrenic and normal subjects, and in particular whether "false significances" due to bias from ocular artefacts are found without EOG correction. Table 2 gives a comparison of the two group in terms of Wilcoxon tests for the bands delta and theta. It is based on EEG data before EOG correction, with vertical, and with vertical and horizontal EOG correction, using average coefficients.

When using data without EOG correction just one significant difference ($P \leq 0.05$) emerged at F₇. After ap-

plying vertical EOG correction, some more differences became statistically significant or a trend ($P \leq 0.10$). With vertical and horizontal correction, statistical results became most pronounced.

Discussion

A topographically differentiated brain dysfunction has often been postulated for schizophrenia, in particular a frontal hypofunction (Weinberger 1988). Whether EEG analysis supports such claims has been a matter of debate. This is also due to the confounding influence of ocular artefacts (Karson et al. 1988). In fact, higher EOG power in the relevant delta and theta bands was found for schizophrenic patients in our investigations, and this leads to artificially increased power at anterior EEG sites by biophysical principles. This might in turn lead to "wrong significances". However, we found more and more pronounced significant differences after correcting for artefacts (for their discussion see Gattaz et al). Why is this so? There is in fact a large decrease in mean power in the slow bands due to EOG correction, and this decrease is larger for schizophrenic patients. However, the reduction in variability is often even more impressive. This can be explained by the fact that some subjects keep their eyes motionless during the "quiet" 20 s analysed, while others still have a substantial amount of eye movements and/or blinks. This factor inevitably increases variability of EEG power much beyond the interindividual variability expected for pure cerebral activity. The amount of variability present influences the statistical significance of group differences negatively. As a consequence, ocular artefacts may be responsible for postulating falsely significant differences in the EEG but may, as here, also be responsible for missing true differences due to variability associated with ocular artefacts. Correcting for ocular artefacts seems, therefore, indispensable in any quantitative EEG analysis, when assessing group differences or experimental differences within groups.

This evaluation has also some methodological implications. It is surprising to what extent EEG band power

Table 2. Results of applying Wilcoxon tests for group differences before EOG correction, after vertical and after vertical and horizontal correction

	Delta			Theta		
	Before correction	Vertical correction	Vertical + horizontal correction	Before correction	Vertical correction	Vertical + horizontal correction
F7	*	*	**		*	*
F3		*	*			
F4		°	°			
F8						
C3						
Cz						
C4		°	°			
T3		°	*			
T4		°	*			

** $P < 0.01$, * $P < 0.05$, ° $P < 0.10$

is reduced by EOG correction, despite analysing the one epoch selected for its lowest EOG power. While the direction of change conforms to expectation, the size of change is beyond our expectation. It is also interesting to see that the topographic pattern is affected by the artefacts owing to the different impact of horizontal and vertical artefacts. Necessarily, brain mapping may also be severely affected by ocular artefacts. These findings have several consequences:

1. The analysis of visually selected epochs for analysis without EOG corrections is shaky (but customary).
2. When analysing experimental data where a selection is usually not possible, effects may become worse.

EOG correction of vertical artefacts alone is common, but our analysis shows that horizontal artefacts need to be corrected off the midline as well (see, for example, the pattern of Fig. 2). The pattern of EOG transmission coefficients as estimated corresponds to biophysical expectations. Group differences in coefficients are small as to be expected. Those of normal subjects may be somewhat more reliable, probably owing to some other artefacts which are more frequent in the data of schizophrenic patients. Using average transmission coefficients rather than individual ones seems advisable, as in our earlier work (Gasser et al. 1986). Such a solution is also attractive in practical terms: coefficients may be computed for a sample of normal subjects once, and used for correction in further studies for different samples.

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