PSYCHOPHYSIOLOGY

Vol. 28, No. 1
Printed in U.S.A.

Methodology

Removal of the Ocular Artifact from the EEG: A Comparison of Time and Frequency Domain Methods with Simulated and Real Data

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ABSTRACT

Frequency-dependent transfer from EOG to EEG may be insufficiently accounted for by simple time domain regression methods (Gasser, Sroka, & Möcks, 1986; Woestenburg, Verbaten, & Slangen, 1983). In contrast, a multiple-lag time domain regression analysis, using lagged regression of EEG on EOG, must theoretically account for both frequency dependence and independence.

Two data sets were constructed, in which the transfer from EOG to EEG was either frequency-independent (constant gain) or frequency-dependent. Subsequently, three different correction methods were applied: 1) a simple regression analysis in the time domain; 2) a multiple-lag regression analysis in the time domain; and 3) a regression analysis in the frequency domain.

The major results were that, for data set 1, the three methods constructed the original EEG equally well. With data set 2, reconstruction of the original EEG was achieved reasonably well with the frequency domain method and the time domain multiple-lag method, but not with simple time domain regression. These three correction procedures were also applied to real data, consisting of concomitantly recorded EEG and high-variance EOG series. No appreciable differences in outcome of the three methods were observed, and estimated transfer parameters suggested that these data were marked by weak frequency dependence only, which can be accounted for by simple time domain regression (and also by the other two methods).

DESCRIPTORS: Ocular artifact, EOG-EEG transfer, Multiple-lag regression analysis, Frequency-dependent gain.

Correction of EEG epochs for transient perturbations due to eye activity can be accomplished by regression techniques in the time domain (Gratton, Coles, & Donchin, 1983; Verleger, Gasser, & Möcks, 1982) or the frequency domain (Gasser, Sroka, & Möcks, 1985; Woestenburg, Verbaten, & Slangen, 1983). There has recently been some debate regarding which technique (domain) is preferable. The results of Gasser, Sroka, and Möcks (1986), stemming from analysis of empirical data, suggest frequency-dependent transfer from EOG to EEG, and superiority of frequency domain methods over time domain methods. Their time domain technique, however, involves analysis of simple regression of EEG on EOG only, and therefore can-

The authors gratefully acknowledge the helpful comments of Drs. T. Elbert, G. Gratton, G.A. Miller, and J. Möcks on an earlier version of this manuscript.

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not account for frequency-dependent and delayed (phase-shifted) transfer from EOG to EEG. In contrast, a multiple-lag time domain regression model describes both transfer characteristics. It can be shown that the commonly applied correction techniques in either domain constitute particular instances of the same multiple-lag regression model.

We will present the defining equation of the time domain multiple-lag regression model and discuss its relationship with common time and frequency domain algorithms. Furthermore, we applied various instances of the general model to both simulated and real data. With respect to the frequency domain correction method, we closely followed the guidelines as outlined by Gasser et al. (1985). For simple time domain regression, we implemented a technique similar to Verleger et al. (1982). As will be shown, this technique constitutes a particular instance of the more general multiple-lag procedure. The purpose of the simulation part was to examine the extent to which the three methods adequately detect and correct constant-gain transfer (i.e., no frequency dependence) and pronounced frequency-dependent transfer, respectively. It was expected that the frequency domain and multiple-lag methods would perform equally well, and better than the simple time domain method, with respect to frequency-dependent transfer. With constant gain, no differences were expected among the three methods. As to real data then, we could evaluate the extent to which differences between outcomes of the three techniques resemble either the constant-gain or the frequency-dependence simulation.

A general multiple-lag regression model describing the effect of the EOG on the EEG at each sampling time t is given by:

$$eeg_{cont,i}(t) = eeg_{true,i}(t) + \Sigma(\beta_{iu} eog_i(t-u)) \quad (1)$$

where eeg_{cont} denotes the observed, contaminated EEG, and eeg_{true} the 'true' EEG (uncontaminated by eye activity artifacts); i denotes epoch or trial, t denotes sample point, and the summation runs from lag $u=0$ to $u=U$, where U is the maximum lag. The sequence of lagged regression coefficients $\beta_{i0}, \beta_{i1}, \ldots, \beta_{iU}$ describes the instantaneous and delayed effects of the EOG on the EEG, and can be conceived of as a filter of length U. The model can accommodate pure delays, pure amplitude modulation, as well as delayed amplitude modulation in the effects of EOG on EEG. Notice that the filter is strictly causal, i.e., future EOG values cannot affect the current value of the EEG.

If the length of the filter (the sequence of regression coefficients) is constrained at $U=0$, the standard regression model in the time domain is obtained (Gasser et al., 1986; Gratton et al., 1983; Verleger et al., 1982). In contrast, straightforward Fourier transformation of the filter yields a frequency-dependent transfer function (Molenaar & Roelofs, 1987), which underlies the usual regression technique in the frequency domain. Specifically, the transfer function of a filter of non-zero length ($U \neq 0$) has gain and phase functions that vary across frequencies, whereas the gain function is constant across frequencies if $U=0$ (Brillinger, 1975; Gasser et al., 1986). Consequently, delayed amplitude modulation can be seen to be the time domain equivalent of frequency-dependent transfer, and both time and frequency domain regression techniques are obtained as particular instances of the same multiple-lag regression model.

Time domain analysis will result in a strictly causal estimated transfer. Causality refers to exclusively relating EEG data point t to EOG data points $t-u$, $u \geq 0$, but not $u<0$. In contrast, the transfer function obtained by frequency domain techniques may be non-causal (Brillinger, 1975). That is, estimation of transfer in the frequency domain may lead to a time domain equivalent where the estimated contribution of EOG to EEG at time point t depends on both EOG values at time points t, $t-1, \ldots, t-U$, and EOG values at $t+1, t+2, t+3, \ldots$. As can be seen in (1), the time domain multiple-lag regression equation involves past and present values of the EOG only.

Generally, the convolutions involved in time domain multiple-lag regression require considerably more computation time than the equivalent multiplications in the frequency domain (fast Fourier transformations included; Beauchamp & Yuen, 1979). If this is considered a problem, the extremely fast time domain algorithms for Wiener filtering, as described by Goodwin and Sin (1984), may be of help. In the applications to be described below, we used a Wiener filter algorithm adapted from Robinson (1967), in which both EEG and EOG may be multichannel signals. Simulated data were constructed in such a manner as to provide a priori known frequency-dependent transfer from high-variance EOG to EEG. Because blinks and saccades were weighted equally, there was no need for separate estimation and correction of blink artifact. With real data, however, blinks and saccades were analyzed separately (Corby & Kopell, 1972).

Methods

General Recording Procedures

Frontal (F_Z) EEG and vertical EOG were recorded with standard Ag/AgCl electrodes. For the EEG, linked ear electrodes were used as inactive references. For the EOG, infra- and supra-orbital electrodes were attached in line with the pupil of the left eye. All signals were

amplified and filtered by an Elema universal filter, with a time constant of 5 s and a low-pass frequency of 30 Hz. Amplifier output was first entered into a 50 Hz notch filter with a bandwidth of 4–5 Hz, and subsequently into a 45 Hz passive low-pass network. On-line A/D-conversion and stimulus presentation were controlled by a PDP 11/23 computer. Each recording epoch lasted 1024 ms, with a sampling rate of 250 Hz, hence in each epoch 256 sample points were produced. Sampling started 100 ms before stimulus onset.

Simulations

Two simulated data sets were constructed. The raw data that were entered into the simulation were identical in the two cases. These data consisted of 36 epochs of frontal (F_Z) EEG and 36 epochs of vertical EOG.[1] The EEG epochs were recorded from one subject in a modified visual oddball paradigm (see Kenemans, Verbaten, Melis, & Slangen, 1989) on 36 trials of infrequent deviant stimuli. The 36 EOG epochs were recorded from a second subject during a categorization task (see Real Data section below), and mainly contained large saccades, as a consequence of the task. Selected epochs containing the EOG signal and true (uncontaminated) EEG signal are shown in Figure 1. Prior to each transfer simulation, in each epoch the mean of the first 25 samples was subtracted, and the first and last 25 samples were multiplied with a cosine-bell window.

1) Constant gain (usual time domain model). For all 36 trials, a contaminated EEG signal was constructed from the true EEG and the EOG in the following way:

$$eeg(t)_{cont} = eeg(t)_{true} + .2eog(t), \quad (2)$$

with t denoting 1, . . . ,256 samples. The resulting EEG is shown in Figure 1.

2) Frequency-dependent gain (general causal transmission). For all 36 trials, a frequency-dependent transfer function was constructed in the following way. A strictly causal time domain convolution was chosen according to

$$\beta(j) = .02^{j-1},\ j = 1, \ldots, 512.^2 \quad (3)$$

This series was fast-Fourier transformed, leading to a series of complex Fourier coefficients $\beta(\omega)$, $\omega = 0, \ldots, 512$, with a maximum transfer amplitude of .2 for the first elementary frequency component. The amplitude spectrum of the resulting transfer function is depicted in Figure 4 (right panel). The 36 EEG and EOG series

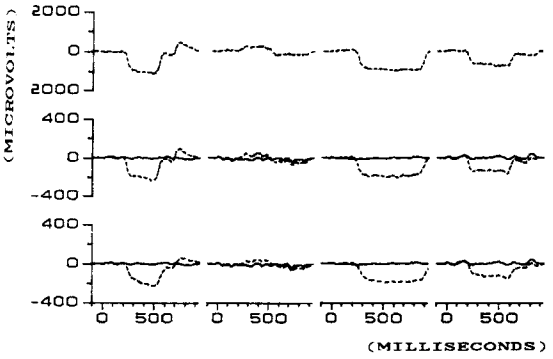

Figure 1. Top: Selected EOG epochs. **Middle** and **bottom:** EEG epochs (same trials as EOG) containing true (uncontaminated) frontal EEG (solid lines), and EEG contaminated (dashed lines) according to the constant gain model (middle), and the frequency-dependent gain model (bottom).

were padded with 256 zeroes and transformed to the frequency domain, leading to the complex coefficients $EEG(\omega)$ and $EOG(\omega)$, $\omega = 1, \ldots, 512$. The contaminated EEG signal was constructed in the frequency domain according to

$$EEG_{cont}(\omega) = EEG_{true}(\omega) + B(\omega)EOG(\omega) \quad (4)$$

and subsequently transformed back to the time domain (see Figure 1).

Real Data

Thirty-six epochs of frontal EEG and concomitant vertical EOG were recorded from 5 subjects during a categorization task. Stimuli consisted of either a '5' or a '2', and were presented at various unpredictable locations on a TV screen; subjects had to count '5's and were therefore required to make large saccadic eye movements in both vertical and horizontal directions (see Kenemans, Verbaten, Sjouw, & Slangen, 1991; Verbaten, Roelofs, Sjouw, & Slangen, 1986).

Techniques for Estimation and Correction

1) Time domain simple regression analysis. This technique is essentially the same as the one described by Verleger et al. (1982; see also Gratton et al., 1983, and Gasser et al., 1985). After baseline subtraction, for each trial i the coefficient β_i for regression of EEG on EOG is estimated as

$$\beta_i = cov(eeg_i, eog_i)/var(eog_i), \quad (5)$$

where the numerator contains the covariance between the EOG and the EEG series, and the denominator the variance of the EOG series. The B_i are averaged over trials, and for each trial the weighted EOG sample t is subtracted from the EEG sample t, t=1, . . . , 256.

2) Time domain multiple-lag regression analysis. For this technique, the model is given in equation (1). Note that the simple time domain model is a special case of the multiple-lag model, with U=0. The vector $b_i\ [\beta_{i0}, \beta_{i1}, \ldots, \beta_{iU}]$ is estimated according to

$$b_i = SS_i^{-1}sp_i, \quad (6)$$

[1]All analyses on simulated data were carried out with 84 trials as well, leading to similar results.

[2]Setting all phase angles to zero induces non-causality. That is, when a series of complex transfer coefficients $\beta(\omega)$ is created with zero imaginary parts (and hence zero phases) only, inverse Fourier transformation to the time domain will lead to a non-causal series of $\beta(j)$, with $\beta(1) = \beta(-1)$, $\beta(2) = \beta(-2)$, etc.

where sp_i is a vector of length U+1 containing the sum-of-products among eeg_t and eog_{t-u}, u=0, . . . , U, at trial i, and SS_i is the (U+1)×(U+1) sum-of-products matrix among the eog_{t-u}. Finally, the b_i are averaged over trials, and for each trial the weighted EOG is subtracted from the EEG according to

$$eeg_{i,true}(t) = eeg_{i,cont}(t) - \Sigma(\beta_u eog_i(t - u)). \quad (7)$$

The implementation of this method is based essentially on a description of multichannel Wiener filtering by Robinson (1967). The program allows variable input of U, and hence both simple and multiple-lag time domain analysis. For a certain U, the algorithm (Robinson, 1967) recursively yields U+1 filters, with respective lengths 1, 2, . . . , U+1. In addition, for each filter a normalized mean square error is computed, consisting of a ratio of the squared deviation between the observed EEG and the weighted contributions of the EOG at different lags (numerator), and the squared EEG values (denominator). Hence, for a filter of length U+1, a sequence of U+1 normalized mean square error values is computed, which can be inspected as to the extent to which it levels out toward the end of the filter. If there is no leveling out, appreciable decrease in normalized mean square error will be found with longer filters, and the regression model should be extended with further-lagged predictors. In the present study, all analyses were conducted with U=31 (samples).

3) Frequency domain regression analysis. This technique is essentially the same as the one described by Gasser et al. (1985), and differs from the method of Woestenburg et al. (1983) in only some minor respects. Among these differences are the facts that no *F*-tests for regression of EEG on EOG are conducted, and no upper limits are maintained with respect to levels of transfer amplitude[3]. For each trial i, the EEG and EOG series (n=256) are subjected to subtraction of the baseline (first 25 samples), and padded with 256 zeroes. Then both series are transformed to the frequency domain, leading to the Fourier coefficients $EEG(\omega)_i$ and $EOG(\omega)_i$, with ω denoting the multiplication factor for the basal frequency. The complex-valued regression coefficients are computed according to

$$\beta(\omega) = \frac{\Sigma(EEG(\omega)_i EOG^*(\omega)_i}{\Sigma(EOG(\omega)_i EOG^*(\omega)_i} \quad (8)$$

where summation runs from i=1 to i=36 (number of trials) and * denotes conjugation. In this expression the denominator contains the average EOG periodogram and the numerator contains the EEG-EOG cross-periodogram. Smoothing of periodogram and cross-periodogram before division, according to the procedure recommended by Gasser et al. (1985), appeared to improve estimation. In contrast, multiplying the time series with a cosine-bell window before Fourier transformation had a deteriorating effect. For a discussion of the respective advantages and disadvantages of linear (cosine-bell) versus quadratic (smoothing) windowing, the reader is referred to Beauchamp and Yuen (1979), Otnes and Enochson (1972), and Sloan (1967). Gasser et al. (1985) recommend the use of both cosine-bell and smoothing of the periodogram (but use of very few trials, i.e., 5), and Woestenburg et al. (1983) report on the use of cosine-bell only. In addition the regression formula of the latter authors contains subtraction of the (cross-)periodograms of the average EEG and EOG series. In our analysis, this appeared to have no appreciable effect. After estimation of the $B(\omega)$, for each trial and each frequency band the weighted EOG is subtracted from the EEG according to

$$EEG_{i,true}(\omega) = EEG_{i,cont}(\omega) - B(\omega)EOG_i(\omega), \quad (9)$$

and the corrected EEG series are transformed back to the time domain.

Results

Simulations

1) Constant gain (usual time domain model). Figure 2 (upper panels) shows selected epochs from the artifact-free EEG, as well as the EEG corrected according to the three methods. As can be seen, there are very few differences among the four time series, indicating that the three techniques performed equally well upon this data set. Mean correlations, over trials, between uncontaminated and corrected EEG series are listed in Table 1.

2) Frequency-dependent gain (general causal transmission). Figure 2 (lower panels) shows selected epochs from the artifact-free EEG, as well as the EEG corrected according to the three methods. In Table 1 the main results are listed. As can be seen in Figure 2, substantial over- and under-correction occurred with the simple time domain method, which was reflected in a correlation of .44 with the uncontaminated EEG series. Figure 2 also shows multiple-lag corrected EEG trials (U=31), confirming the high correlation with the uncontaminated EEG. The frequency domain corrected EEG can be seen to deviate from the true EEG at some sample ranges, but still resembles it much more than the simple time domain corrected EEG. In Figure 3 regression coefficients and normalized mean square errors are depicted for U=31 (multiple-lag) (for other values of U normalized mean square errors would be the same), for both constant-gain and frequency-dependent transfer. For illustrative purposes multiple-lag analysis was also applied to a constant-gain transfer data set with a pure delay of 6 samples (i.e., $eeg(t)_{cont} = eeg(t)_{true} +$

[3]Preliminary inspection of the main results revealed that, with respect to the first 40 frequency components, *F*-values always exceeded the critical *F*-value computed according to Woestenburg, Verbaten, and Slangen (1983), and transfer amplitude levels never exceeded the criterion level of Woestenburg et al. (i.e., 0.7).

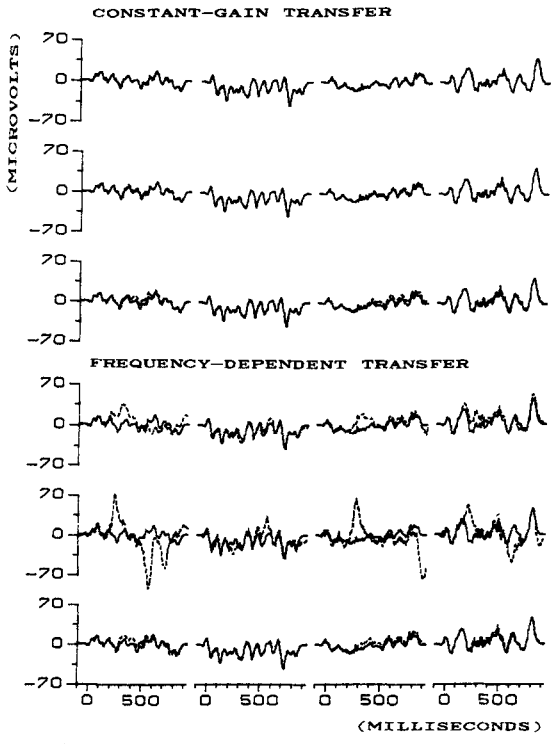

Figure 2. Selected epochs containing true frontal EEG (same trials as in Figure 1). Solid lines: true EEG; dashed lines: corrected EEG. **Upper panels:** Superimposed EEG series contaminated by transfer from EOG with constant gain, corrected according to frequency domain, simple time domain, and multiple-lag time domain methods. **Lower panels:** Same for superimposed EEG series contaminated by transfer from EOG with frequency-dependent gain.

.2eog(t − 6))[4]. As can be seen (upper panel), in the case of constant gain the regression coefficient beta peaks exclusively at u=0, at which the normalized mean square error levels out as well. With delayed constant gain, beta peaks and the normalized mean square error levels out, at u=6. With frequency-dependent gain (middle-panel), a much flatter beta function is obtained and the normalized mean square error levels out at about u=20. Figure 4 shows the FD-estimated gain functions for imposed constant gain and frequency dependence, which can both be seen to be approximated fairly well.

[4]There are no indications for pure phase delay with respect to EOG-EEG transfer (Gasser, Sroka, & Möcks, 1985; Elbert, personal communication, January, 1989; Woestenburg, personal communication, January, 1988). However, additional simulations, employing the same data set, showed that pure phase delay can be detected and corrected adequately by both frequency domain and multiple-lag methods, but not with the simple time domain technique.

Table 1

Mean (across 36 trials) correlations between true EEG, contaminated EEG, and EEG corrected according to the three methods, for both imposed constant-gain and imposed frequency-dependent transfer

	Correlations					
	Constant gain			Frequency dependent		
EEG Series[a]	True	FD	TDS	True	FD	TDS
Contaminated	.18			.17		
FD	.99			.80		
TDS	.99	.98		.49	.76	
TDM	.96	.95	.99	.95	.84	.46

[a]FD = frequency domain method, TDS = simple time domain regression, TDM = multiple-lag time domain regression.

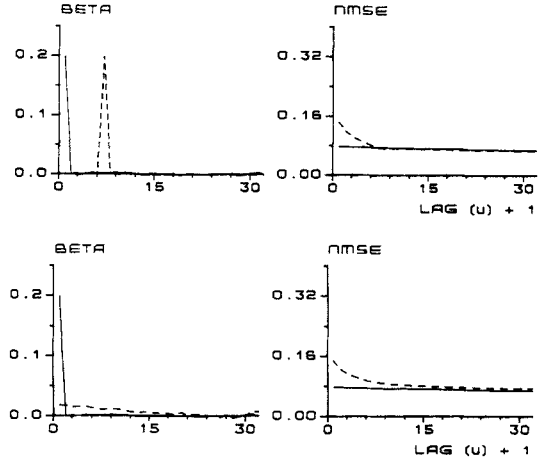

Figure 3. Left panels: Regression coefficients (beta) estimated with multiple-lag analysis as function of lag (u), for maximum lag (U) = 31. **Right panels:** Corresponding normalized mean square errors (NMSE; see text for explanation). NMSE without correction is always 1. **Upper panels:** Simulated constant-gain (solid) and delayed constant-gain (dashed) data. **Lower panels:** Simulated constant-gain (solid) and frequency-dependent transfer (dashed) data.

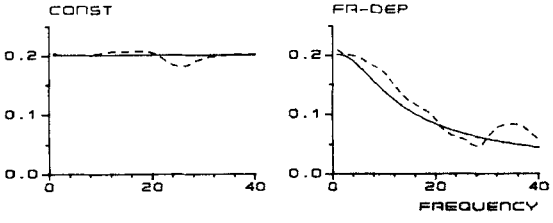

Figure 4. Imposed (solid) and frequency domain-estimated (dashed) gain functions for simulated constant-gain (left panel) and frequency-dependent transfer (right panel) data.

3) Causality and single-trial estimation. Frequency domain estimated transfer functions were inversely transformed to the time domain, both for the constant-gain and the frequency-dependent

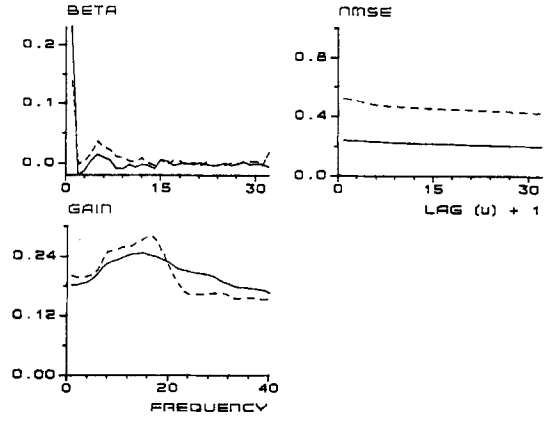

Figure 5. EOG-EEG transfer parameters, estimated from real data. Curves (solid for blink trials, dashed for saccades) represent averages across subjects and trials. **Left upper panel:** Regression coefficients (beta) as function of lag (u), for maximum lag (U) = 31. **Right upper:** Corresponding normalized mean square errors (NMSE; see text for explanation). NMSE without correction is always 1. **Lower:** Frequency domain-estimated gain functions.

transfer data. For constant gain β_{-1} (i.e., the coefficient for regression of EEG sample t on EOG sample t+1) amounted to .064, indicating substantial estimated contribution of future EOG values to EEG. As reported, however, this did not have a detrimental effect on correction performance. For frequency-dependent transfer, absolute values of β_u, $u<0$, did not exceed .003. The same value can be seen in Figure 3 (lower left panel, frequency-dependent transfer) for $u+1=18$. For multiple-lag analysis single-trial correction was probed with respect to the frequency-dependent data. Mean correlation with the true EEG series amounted to .78.

Real Data

Figure 5 shows estimated regression coefficients and normalized mean square errors for $U=31$ (multiple-lag) and gain function (frequency domain), averaged over the 5 subjects. Trials containing blinks were analyzed separately from those containing saccades only. No attempt was made to account for contamination of EOG by EEG, because only high-variance EOG records were available. As can be seen, for blinks, at $u=0$, mean β was relatively very large and the normalized mean square error leveled out, indicating that a filter length of 1 (as realized with simple time domain regression) would be sufficient to estimate blink transfer. This was confirmed by the rather flat gain function. For saccades, a similar drop in β was observed for $u=0$, although relatively smaller than for blinks. The normalized mean square error function also shows a slightly less radical leveling, compared to blinks, because there is a small crack at about $u=8$ as well. Similarly, the saccade gain function suggests more pronounced frequency dependence. Comparison with Figure 4, however, reveals that the observed frequency dependence was far less pronounced, relative to the simulated frequency dependence, suggesting that also for saccades a filter length of 1 would suffice. With simple time domain analysis, the mean obtained regression coefficients were .20 (blinks) and .22 (saccades). Table 2 shows mean correlations between EEG series corrected according to the three methods. As was to be expected, all correlations were invariably high, with no single-subject value below .91. Figure 6 shows selected EOG and EEG trials from one subject, confirming the high correlations. Note that increasing U, when $U=0$ would suffice, does not affect correction performance. Indeed, this was not expected from the results on the simulated constant gain function.

Table 2

Mean (over subjects and trials) correlations between EEG series, corrected for EOG according to three methods

	Correlations			
	Blinks		Saccades	
EEG Series[a]	Mean	SD	Mean	SD
FD, TDS	.94	.02	.95	.02
FD, TDM	.95	.01	.94	.03
TDS, TDM	.97	.03	.96	.03

[a]FD = frequency domain method, TDS = simple time domain regression, TDM = multiple-lag time domain regression.

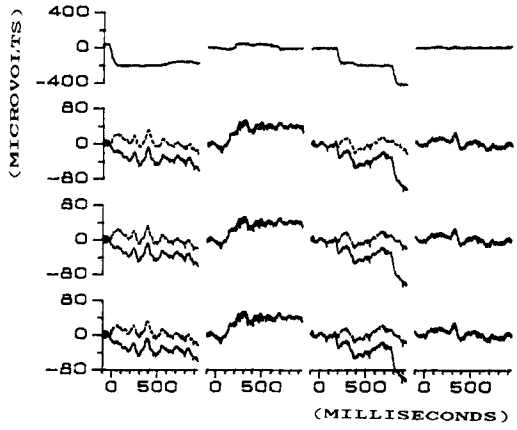

Figure 6. Selected epochs from one subject containing vertical EOG (upper traces), and contaminated and corrected F_Z-recorded EEG (lower traces). Corrections according to frequency domain, simple time domain, and multiple-lag time domain methods. Solid lines: true EEG; dashed lines: corrected EEG. Bold EEG traces reflect the superposition of similar corrected waveforms estimated with the three different techniques.

Discussion

The results of the present simulation study show that a time domain multiple-lag regression analysis can detect and correct both constant-gain and frequency-dependent transfer from EOG to EEG. The adequacy of the multiple-lag correction technique depends on the number of regression coefficients (U+1) included in the model. With U=0, the commonly used time domain simple regression technique is obtained. Under the conditions of frequency-dependent transfer imposed in the present study the simple time domain method did not perform adequately. With U>0, however, frequency-dependent transfer can be detected and corrected in the time domain at least equally well, compared to a frequency domain method. Furthermore, both multiple-lag and frequency domain techniques, as well as the simple time domain method, perform equally well in case of frequency-independent transfer (constant gain). That is, multiple-lag and frequency domain techniques estimate the transfer function correctly also when frequency-dependent transfer is not present. Here, correlations between corrected and true EEG series were above .95 for all three methods, and the same held for the correlations among the corrected EEG series (see Table 1).

This latter result was also obtained in the analysis of real data, suggesting that EOG-EEG transfer in this data set may be well approximated by a constant-gain model. This was confirmed by the behavior of several estimated transfer parameters (see Figure 5). Regression coefficients (beta) and normalized mean square errors (NMSE) showed a sharp drop immediately at lag 0 (compare with Figure 3) and gain functions were rather flat (compare with Figure 4). All these observations held equally for blinks and saccades, although transfer of the latter seemed to be slightly more dependent on frequency.

The absence of differences between outcomes of frequency domain versus simple time domain techniques is somewhat at odds with the results of Gasser et al. (1986), who found differential effects on certain evaluation parameters, suggesting frequency dependence of transfer and hence superiority of frequency domain over simple time domain analysis. It is possible that differences between frequency domain and simple time domain results also existed in the present data but were not reflected in the correlations between EEG series corrected with the two techniques. Hence, although simple time domain analysis seems to be adequate as far as the present analysis of real data is concerned, it may still be safer to routinely apply frequency domain methods for the correction of ocular artifact. For the same purpose, multiple-lag analysis appears to be an adequate time domain analog of frequency domain analysis. In addition, multiple-lag performance with simulated frequency-dependent transfer was somewhat better (see Table 1). This finding may be related to the use of windowing with frequency domain analysis, which in itself modulates the input series but is obligatory when using frequency domain methods (Beauchamp & Yuen, 1979; Bergland, 1969).

With respect to eventual estimation of noncausal transfer by the frequency domain method, we found that it either may be present but does not affect correction performance (constant-gain simulation), or is not present at all (frequency-dependence simulation). Single-trial correction, probed with multiple-lag analysis on simulated frequency-dependent transfer data, resulted in slightly less adequate correction. Apparently some random trial-to-trial fluctuation may occur with respect to estimated EEG-EOG transfer, which is reduced by averaging over trials. A similar problem was recognized by Gasser et al. (1985), who recommend additional averaging of transfer coefficients over subjects.

Many of the issues raised in this paper are also discussed in the recently published report on the 1987 Tilburg symposium (see Brunia, Möcks, & Van den Berg-Lenssen, 1989). Among others, they include the equivalence between frequency domain and multiple-lag methods and the apparent similarities in outcome between these two methods and the simple time domain technique. Problems not addressed in the present study pertain to transfer from EEG to EOG and to event-related activity occurring independently in EOG and EEG. To deal with these issues, procedures are given by Gasser et al. (1985, EOG-EEG transfer) and Gratton et al. (1983, event-related activity).

To conclude, it can be stated that conditions can be created in which standard, simple time domain regression techniques do not perform adequately in correcting eye activity artifacts. In contrast, a multiple-lag time domain regression technique detects and corrects the simulated frequency-dependent transfer from EOG to EEG at least as adequately as the frequency domain method, and both were better than the simple time domain technique, confirming the report by Gasser et al. (1986). In reality, however, frequency dependence does not seem to be as pronounced as in our simulated data. With respect to our empirical data, there were no differences in outcome between the three methods. As noted before, however, others have reported differently (Gasser et al., 1985), and therefore, as general procedures, frequency domain or multiple-lag time domain analysis may still be preferred. From these two methods, the latter may be more intuitively

comprehensible, and does not involve issues of windowing. Furthermore, the implementation after Robinson (1967) allows multiple input series, and therefore the possibility of including more than one EOG series in the regression analysis; dependences between different EOG series are accounted for as in classical multiple regression analysis (e.g., Harris, 1975).

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(Manuscript received October 25, 1988; accepted for publication December 22, 1989)

Announcement

Second International Conference on Biobehavioral Self-Regulation and Health

From 16–20 September, 1991, the German Research Society and the Association for Applied Psychophysiology and Biofeedback will sponsor the Second International Conference on Biobehavioral Self-Regulation and Health, entitled "Advances in Applied Psychophysiology," in Munich, Germany. The conference will include invited workshops, symposia, speakers, and posters on the psychophysiological treatment of cardiovascular disorders, central nervous system disorders, gastrointestinal disorders, pain, pediatric disorders, sleep disorders, stress-related disorders, and neuromuscular disorders. Submissions related to environmental hazards and ethical and philosophical issues are also welcome.

Please send poster and workshop submissions to: (Europe) Niels Birbaumer, Universität Tübingen, Behavioral Neuroscience Department, Gartenstr. 29, D-7400 Tübingen, FRG; or (USA) Ronald A. Seifert, Association for Applied Psychophysiology and Biofeedback (AAPB), 10200 West 44th Avenue, Suite 304, Wheat Ridge, CO 80033-2840 (303/422-8436). For general information about the conference, contact Francine Butler at AAPB (303/422-8436) or Rainer Schandry, Psychologisches Institut, Klinische Abteilung, Leopoldstraße 13, D-8000 München 19, FRG.