

On the Nature of Artifacting the qEEG

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An analysis of the effect of possible muscle artifact of the qEEG upon the relative power, coherence, phase, pkamp, and pkfreq figures is presented in terms of four issues and with three subjects. The study involves the: 1) analysis of muscle activity and its relationship to other muscle and cortical leads, 2) analysis of how muscle activity might be manifested in other cortical leads, 3) effects of possible cortical muscle artifacts on qEEG variables. In Experiment 1 two artifact leads were placed on the neck muscles and cortical positions T3 and F7 to conduct the first analysis. Experiment 2 was part of a larger experiment in which one of the subjects had four artifact leads placed on the heart, under the right and left ear, on the neck, and below the right eye. In this experiment all the epochs were selected which appeared to be eye movement type artifact or gross artifact and labeled as such in a statistical program.

Introduction

The artifacting of qEEG data is a crucial step in the analysis of qEEG data prior to submitting the resulting unartifacted data to a normative comparison. The traditional problematic areas include eye movement (affecting frontal activity predominantly), drowsiness (affecting central superior leads, Fz, Cz, and Pz), heart activity, and muscle activity generating from the neck (affecting the T3/T4 and O1/O2 positions) and facial muscles or from the temporal muscle itself. There is no complete agreement as to what constitutes the hertz ranges for the muscle artifact. Some (Davidson, 1988; O'Donnell, Berkhout, & Adey, 1974) have argued that the 13-20 hertz range is where muscle artifact manifests itself. O'Donnell et al. (1974) reported high degrees of relationship between frontal EEG and EMG frontal sites from the 8-19 Hz range as well as large increases in absolute power of EEG alpha and power during frontalis contraction. Other researchers have found facial muscle activity manifesting itself in the lower frequencies. (Nunez, 1981; Van Boxtel, Goudswaard, Vander Mollen, & VanDer Bosch, 1984). Davidson et al. (1990) argued that studies involving emotional expression might be contaminated by facial muscle activity. Duffy, Jones, Bartels, McAnuity, and Albert (1992) conducted a principal components analysis of 2,944 evoked potential variables. Of the 80 factors studied, almost half (38) were considered artifactual by the author's criteria of their loading patterns and familiar pattern. The subjects had four bipolar artifact leads strategically placed to monitor eye movements and muscle tension from the face, jaw, or scalp. Data above 32 Hz were considered artifactual and discarded. The analysis of the loading pattern included the artifact leads and factors were considered artifactual if the factor loaded predominantly on the artifact leads. The other criteria involved the loading being predominantly on a single electrode, especially bilateral mid-temporal or posterior temporal electrodes and if the activity was in the "higher spectral beta range," i.e. activity maximal around 25.5 Hz, but present in the 13-32 Hz range.

Friedman and Thayer (1991) employed a redundancy analysis to avoid the problem of correlational analysis and causation. They employed the 1- to 30-Hz range and placed artifact leads on 4 facial sites (2-cheek, 2brows) according to the placement guidelines of Cacioppo et al. (1990). They

employed 4 EEG leads (F3, F4, P3, P4).

They concluded that redundancy analysis indicated very little of the variance in the EEG data which could be explained by the brow EMG leads (8.22%), and frontal brow EMG could account for more of the alpha (4.38%) band of the EEG than the beta band (2.97%). In addition, they argued that the shared variance may also reflect "cortical interactions involved in the affective process."

Subjects

The data from three subjects were analyzed. The first subject (S1) was a 49-year old, white, right-handed male collected for comparison purposes as part of a larger experiment. Subject 2 (S2) was a 28-year old, white, left-handed male who had recently (within 3 months) suffered a head trauma in an auto accident. He did not recall the accident and was experiencing severe problems in neuropsychological functioning (as evidenced on subsequent neuropsychological testing). Subject 3 (S3) was a 23-year old, white, right-handed female with no history of head trauma. None of the subjects were on medication at the time of the testing.

Method

For Experiment 1, S1 had two artifact leads placed on the left neck area and two cortical positions recorded (T3, F7) to allow correction for eye movement artifact and to observe the relationship between the artifact leads and the T3/F7 positions. The two neck artifact leads were placed below the left ear about 1-1/2" apart, with A1 being lower. For Experiment 2, the data of three subjects who were part of a larger experiment were collected employing the Lexicor Medical technologies hardware. S1 in this experiment had artifact leads placed on the heart, under the ears on the neck and under the right eye. The number of epochs ranged from 4,000 for two subjects and about 8,000 epochs for a third. The Biolex/Neurosearch software generated relative power, coherence, phase, peak amplitude, and peak frequency data for all the epochs under consideration. Coherence and phase values were generated for all interhemispheric leads, homologous positions across hemispheres, and frontal and posterior nonhomologous relationships between the left frontal (F1, F7, F3) and right posterior (T6, P4, O2), and vice versa (right frontal to left posterior). Phase and coherence relationships to the central positions (Fz, Cz, and Pz) were not evaluated, nor other nonhomologous relationships not mentioned. The bandwidths were defined according the factor analysis conducted by Herman, Fichte, and Kubicki (1978) and Wieneke as reported in EEG by Niedermeyer and DaSilva (1993). However, as the factors previously reported only extend to the 18-Hz range and the study involved a sampling rate of 256 (resulting in a 64-Hz range of data), the 18- to 64-Hz range was divided into two ranges: Beta3 (18.5 to 38 Hz) and Beta4 (38 to 64 Hz). The other ranges were defined as follows: Delta (0-6 Hz), Theta (6-9 Hz), Alpha1 (9-10.5 Hz), Alpha2 (10.5-13.5 Hz), Beta1 (13.5-15.5 Hz) and Beta2 (15.5-18.5 Hz). None of the records were artifacted prior to the generation of the data. The data was transferred to a statistical software program (CSS Version 5). All the individual epochs were analyzed for artifact and labeled in the statistical spreadsheet accordingly. The predominant method of artifacting was focused on eye movement activity and gross disturbances of the EEG record as shown in Figures 1 and 2. Figure 3 represents the typical T3 high beta activity that is being examined for possible muscle activity.

There was no attempt to artifact the epochs which may have included muscle activity from the neck or beta activity in the frontal areas and which did not include eye movement artifacts. Epochs that included both types of contaminants were labeled as artifacts.

Results

Relationship between Muscle Artifact leads and qEEG measures in muscle contraction and non muscle contraction epochs.

Experiment 1 was directed towards understanding how muscle activity in a muscle contraction and resting condition would affect another muscle group and cortical leads. S1 engaged in the task under an eyes closed, resting condition, and in a conscious neck tensing condition. T-tests of significance comparing the muscle contraction epochs from the eyes closed resting condition showed significant reductions in the Delta to Beta2 bands (0-18.5) for all four positions and significant increases in relative power of Beta3, Beta4, and total microvolts for all positions, thus confirming the idea that conscious muscle contraction will result in increases in the upper bands. However, it does not necessarily follow that in a resting condition, we must consider high beta activity as unconscious muscle activity.

To evaluate the difference, a principal components factor analysis of the data in the muscle contraction condition (N=60, Eigenvalues set to 1) did not indicate a factor (of 11 factors) which related the artifact leads (high beta, A1 or A2) and the T3 activity in the Beta3 or Beta4 condition, but did relate the lower of the artifact leads (A1) to F7 Beta4 activity (employing a .40 or higher loading on the factors). Two other factors interrelated the cortical and muscle activity in a non-patterned manner [i.e. F7 Theta (minus loading) and A2 Alpha1 and Alpha2 (positive loadings)]. In this muscle contraction condition, there was also no factor of high beta activity between the A1 and A2 leads. The relevant factors of the leads included A2 (Alpha2 to Beta4, 6.9 % of variance), A1 (Beta1 to Beta4 activity, 3.9% of variance) and F7 (Beta1 to Beta4, 28.8% of variance), T3 (Beta1 and Beta2 and F7 Total microvolt, 6.2% of variance) and T3 [Beta3 (positive) and Beta4 (negative loading), 3.9% of variance].

Figure 1
Gross Distortion Artifact

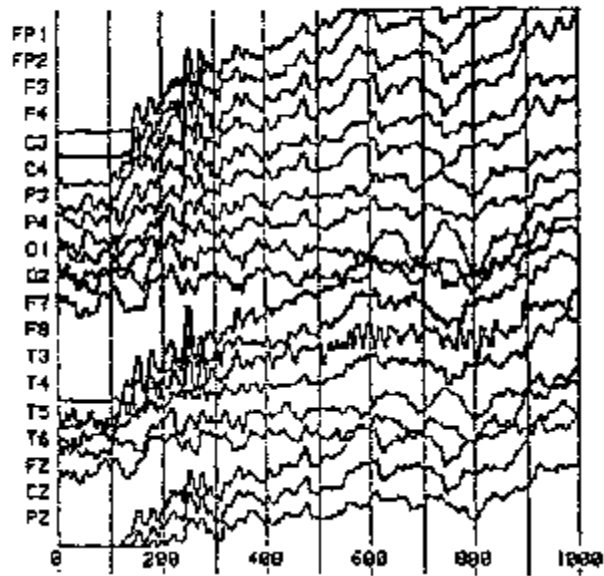
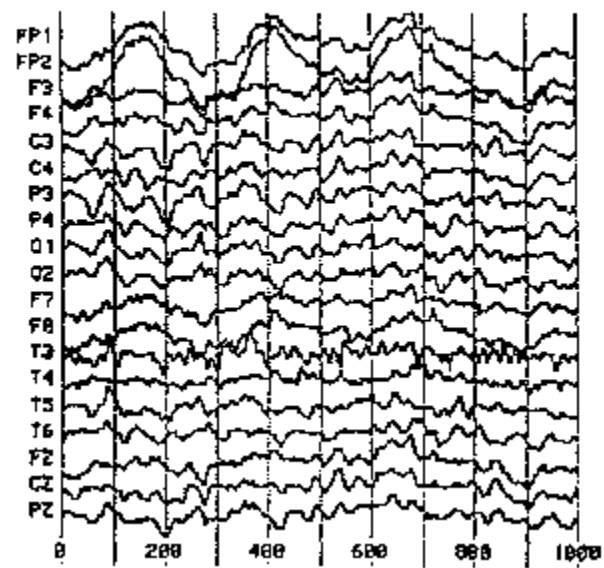
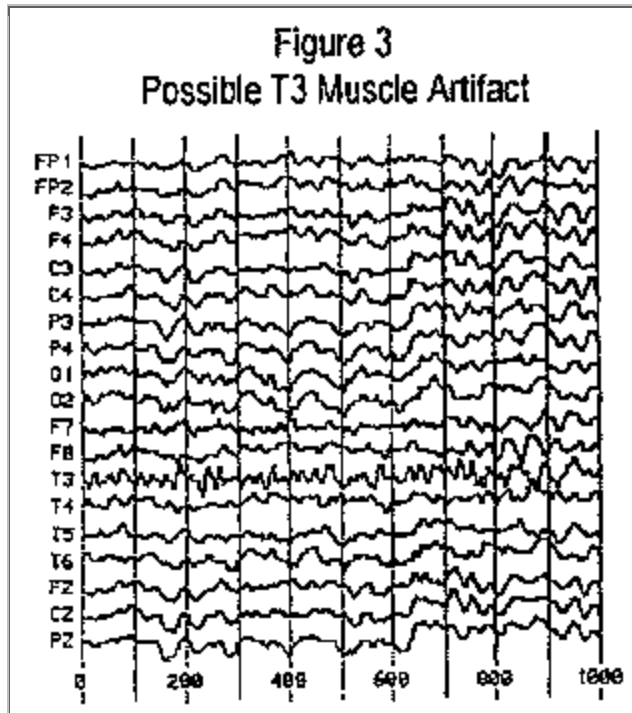


Figure 2
Eye Movement Artifact





A principal components factor analysis of the no muscle contraction data ($n=242$, Eigenvalues greater than 1) yielded 10 factors. The difference in factor structure between the muscle contraction and resting condition indicated that in the muscle contraction condition, there was a factor at F7 that related all the beta ranges, while in the resting condition the factor only included Beta1 and Beta2. The high beta activity at T3 did not inverse its loading, but had Beta3 and Beta4 both loading in a positive direction. In terms of the artifact leads, the A1 (lower) lead massed the beta activity in the muscle contraction condition and separated Beta1 and Beta2 into one factor, and Beta3 and Beta4 into another factor in the relaxed condition. The A2 lead (higher lead) kept a similar factor in that all the ranges from Alpha2 to Beta4 loaded on one factor. In addition there was a factor in the resting condition which did not emerge in the muscle contraction condition. A factor emerged for the F7 and T3 positions in terms of the Alpha1 and Alpha2 ranges, which is similar to the factor that emerged under the full experiment (2) condition. Thus, although the gross structure of the factors was similar, the pattern was different for conscious muscle contraction condition versus resting condition. It is of some interest to note that real muscle activity in the artifact leads tends to group most of the ranges into a factor, a phenomenon that also occurs at F7 in the muscle contraction condition. Only the A2 lead maintained this pattern in the relaxed condition. Thus, this may give us an indication of what can be considered muscle activity.

A factor analysis of the eyes closed, resting condition in Experiment 2, S1 ($n=449$, factor loadings set at .40) of the A3 position (Oust below left ear), and T3, F7, and T5 positions resulted in a similar pattern; there were separate factors for the cortical leads and A3 position which did not interrelate between the cortical leads and the artifact leads, but did interrelate along similar bandwidths for the cortical leads. For example, the A3 lead had 2 factors: 1) Alpha1 to Beta1 (2.8% of variance), and 2) Beta3 and Beta4 (minus loading of Delta; 2.2% of variance). This is similar to experiment 1 in the A1 position where the high and low ranges separated into separate factors, whereas they had not

separated for the similar position (M). A factor emerged for the Beta4 range relating almost all the cortical leads, but not including T3, T4, or F8 (9.7% of variance). A factor emerged for F8 Beta3 and Beta4 that all simultaneously loaded in a negative direction on T3 Beta3 and Beta4 (1.8% of variance). A factor also emerged for F7 Beta3 and Beta4 which had minor loadings (under .40) for the Theta to Beta2 ranges (1.11% of variance), reflecting the pattern seen in the muscle artifact leads in Experiment 1, where a large spectrum of ranges loads on the factor. Of particular interest is that the F7 Beta4 range loaded on two separate factors, one in a negative manner and the other in a positive loading. It may be, therefore, that Beta4 activity can be reflective of both muscle activity and cortical activity depending upon how it patterns itself. There was also a factor at T5 which related Beta2, Beta3, and Beta4 (1.5% of variance), possibly reflecting a muscle activity factor. For statistical reasons, a similar analysis could not be performed on S2 or S3 for the eyes closed, resting condition. The pattern of activation of the entire beta range is similar to the results of Duffy et al. (1992).

If we examine the factor pattern (principal components) of the three subjects in task conditions, there are factors that emerge which are reflective of this muscle factor. Presumably, task conditions elicit a greater propensity for muscle activity to occur. For S1 a factor emerged at F8 which loaded predominantly (greater than .50) on Beta3 and Beta4, but also had lower loadings on the Theta to Beta2 bands, and also included low loadings (under .30) on T4 Beta3 and Beta4, and F4 Beta3 and Beta4, indicating some spreading effect. There was a factor at F1 and F2 that related Beta3 and Beta4, and also had minor loadings on Theta to Beta2. There was a factor at T4 that related Beta3 and Beta4, and to lesser degree Beta1 and Beta2. This factor also loaded to a lesser degree with T3 Beta3 and Beta4. However, it should also be noted that there was a factor at F7 (with loadings under .50) which had minor loadings (under .40) of Beta3 and Beta4 at T3. An additional factor at F7 of Beta4 did not show this pattern and loaded with F3 Beta4, thus indicating again that Beta4 activity by itself is indicative of other processes than muscle activity. Thus, these three factors (of the 32 obtained) appear to exhibit the pattern of muscle activity and all generating from the frontal lateral positions. These factors also indicate that high beta activity by itself cannot necessarily be considered muscle artifact, as evidenced in several other factors which loaded on high beta activity in the more medially located positions (C3, Cz, C4, etc.), generally with a negative loading. There was no relationship between the heart lead factors (A1) and the lead placed below the right eye (A4) with the above factors. But these artifact leads did show the grouping effect for the entire range (except Delta) that was shown in Experiment 1.

For S2 there were factors at F7 and F8 which had major loadings (greater than .40) on the Beta2 to Beta4 bands with lesser loadings on the Beta1 band. Some spread effect was evident at F4, F3, T3, and T4. Additional factors with similar loadings occurred at F1-F2 and T4 (negative loadings). Again the same pattern emerged of additional factors in the upper bands evident on the medially located positions (positive and negative loadings).

For S3 there was an F1-F2 factor (loadings greater than .50) tied to Delta and Total Microvolts and another F1-F2 factor tied to negative loadings of Beta3 and Beta4, and positive loadings of Delta, both more reflective of residual eye movement artifacts. Additional factors were an F8 Beta3 factor with other lesser loadings on the Theta to Beta2 bands and Beta4 band. Another factor included an F7 factor in which all the beta loadings were under .50 and negative loadings with Delta activity. There were minor spread effects evident in terms of low loadings on these two factors to the F3,

F4, T3, and T4 positions. Again there were factors which loaded on the upper bands which were not reflective of this global activation pattern. In addition, for this subject there was a factor of Beta3 and Beta4 activation at T3 and T4, which did not show this characteristic pattern of global activation.

One of the problems of this approach is reflected in the nature of the loadings. The muscle artifact loadings generally loaded all the ranges or the beta ranges with significant loadings above .50 in most cases. None of the loadings obtained in the cortical leads approached this type of loading. There would be two or three beta ranges which loaded heavily on the factor (above .70) and the others would be in the lower ranges and sometimes there would be a large difference in the loading structure.

Relationships between temporal high beta activity and qEEG measures

To examine how these presumed artifact factors would affect qEEG measures, factor scores were generated for S1 in terms of relative power. The factor scores were then merged with the entire data set for the subject and correlational analysis conducted to assess the effect of the presumed muscle artifact located at F1-F2, F7, F8, and T4 on the coherence, phase, and other values. The correlation matrix (N=3234 epochs) indicated many significant correlations, but all within the range of +.11 to -.11. There was larger correlation with the Peak Amplitude data and could go as high as .25, generally reflected an increase in the higher beta range amplitude with increases in the weighting of the factor. For example, at T4 there was significant positive relationship between the T4 muscle factor score and peak amplitude of Beta3 and Beta4 (.14 and .19 respectively). There was also significant correlation with the T4 muscle factor and T3 Beta3 (.18) and Beta4 (.21). In terms of peak frequency there was smaller correlation than in the coherence and phase analysis. In terms of the relative power there was significant and larger correlation of the factors with their respective positions and the bands at that position. For example, the F8 muscle factor would correlate with all the F8 bandwidths from .17 to .62 (BetaQ and -.61 (Delta)). As the location moved away from its origins, its value dropped. For example, the F12 muscle factor correlated with F7 Beta3 and Beta4 about .12 to .14, and .25 with F8 Beta3, etc. As the distance decreased, the correlation would drop to .10 and below. Overall it doesn't appear that these muscle factors are affecting the qEEG in a significantly meaningful fashion, as the normal variation of activity appears to wash out the major effect that the muscle activity might be having.

The clinician, however, is faced sometimes with a situation in which a particular lead is replete with this type of activity throughout the data. To address this question with this subject, the muscle factor scores were coded to reflect a cut-off of +1 or -1 standard deviation from the mean. In this manner only the outliers were evaluated and t-tests of significance between those lying more than 1 standard deviation below the mean to those lying more than 1 standard deviation above the mean were conducted. This analysis revealed highly significant differences in the raw scores. For example, with an N of 950 Table 1 represents one set of differences for activation/deactivation of the frontal polar beta activity.

Thus if there is a subject who demonstrates excessively high and continual amounts of activity which can be classified in the manner described in the frontal to temporal lateral areas, then there will be a significant effect on the other qEEG measures.

Table 1				
	Lo FP	Hi FP	T-Value	P
F3T3PB1	72.712	79.198	-4.27362	.000021
F3T3PB2	63.568	74.099	-6.14988	.000000
F3T3PB3	64.643	72.749	-4.75564	.000002
F3T3PB4	57.844	66.219	-4.49735	.000008
FP = Frontal Polar Muscle Factor Scores, F3T3PBx = Phase Beta1-4 between F3 and T3				

Discussion

There are several conclusions that can be rendered as a result of the analysis. The pattern of "real" muscle activity in the neck and other artifact leads presents a different pattern than the pattern of alleged muscle activity originating from the frontal to temporal lateral positions. The pattern of muscle artifact resides more in its high activation of all the ranges (except Delta), rather than a specific range. Even though some of the factors generated by principal components analysis resemble the pattern of real muscle activity, the pattern isn't exactly the same. It is not self evident that high beta activity in the temporal leads or lateral frontal areas is necessarily artifact, as it can show a different pattern of interrelationship with the other bands in the same position and with neighboring positions, and can clearly represent underlying electrophysiological processes. Activation of specific bandwidths correlating with neighboring positions along similar bandwidths more probably represents valid qEEG data, especially in the more medially located positions. In a sample of data artifacted for gross eye movements, the effect of these muscle activity factors appears to have minimal effect on other qEEG data, if sufficient epochs are collected. However, in a subject where there is a high continuous level of this type of activity, the effects on the other qEEG variables will be highly significant and dispersed throughout the measures. This analysis only underscores the need to collect several samples of data and of a sufficient number so that any improper artifacting of the possible muscle activity will have a tendency to balance itself out in the final analysis.

Several cautionary notes are in order at this point. There was a tendency for the lower beta bands and upper bands to separate into different factors in relaxed conditions on the neck artifact leads in both experiments. The muscle factor analysis was conducted on data during which the subject was engaged in a task, presumably increasing the likelihood of muscle artifact occurring. This was the reasoning behind searching for an activation pattern which appears to be more characteristic of muscle activation, rather than the activity patterns of a muscle in a more relaxed condition. Based on this analysis, if the subject maintains a relaxed posture while high beta activity is observed in the lateral more frontally located leads, and this activity does not fall into the pattern of activation across all the ranges, then the activity can be considered cortical and not muscles. The above analysis was conducted on one subject predominantly. Other subjects may present more compelling evidence for the effect of muscle activity, or less evidence. In addition, correlation is not causation. The working assumption has been that the muscle activity causes the above changes. A third variable could be at work.

It is, of course, problematic to conduct factor analysis on the qEEG data given the other demands of a busy schedule. However, Lexicor's Neurosearch software allows for analysis of the breakdown of individual epochs which could be analyzed in this fashion.

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