Identifying the sources of the pulse artefact in EEG recordings made inside an MR scanner.

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Introduction

Electroencephalography (EEG) data recorded during simultaneous functional magnetic resonance imaging (fMRI) are compromised by large artefact voltages. The pulse artefact (PA) is particularly challenging because of its persistence even after application of artefact correction algorithms (1-3). Several different causes of the PA have been proposed (1, 4-6) with the most likely sources believed to be cardiac-pulse-induced head rotation, scalp expansion or Hall voltages generated by blood flow. Previous analytical work (7) showed that bulk head rotation is likely to be the dominant source of the PA. Here we carried out experiments aimed at isolating the effects of the different putative sources of the PA and identifying their relative contributions to the amplitude and variance of the artefact.

Methods

EEG recordings were made on 6 subjects (Age=26±3 yrs, 4 Males,) positioned at the centre of a 3T MR system. EEG data were acquired with a 32-channel EEG cap and BrainAmp MR-plus EEG amplifier (Brain Products, Munich), along with simultaneous ECG recording. Head movement was monitored using two, 3-axis accelerometers, which were placed on the forehead (between Fp1&Fp2) and the right temple (above T8), and connected to a BrainAmp ExG-MR amplifier. Four minutes of data were acquired with four different subject conditions: i) relaxed, yielding a PA typical for EEG-fMRI; ii) head restrained using a bite-bar and vacuum cushion, so as to eliminate pulse-driven head rotation; iii) with an insulating layer placed between the scalp and the EEG cap, and an outer layer of conducting gel used to form connections between the electrodes, thus eliminating Hall voltages due to blood flow; iv) with head restraint and insulating layer, to eliminate head rotation and Hall voltages, leaving only the effect of scalp expansion.

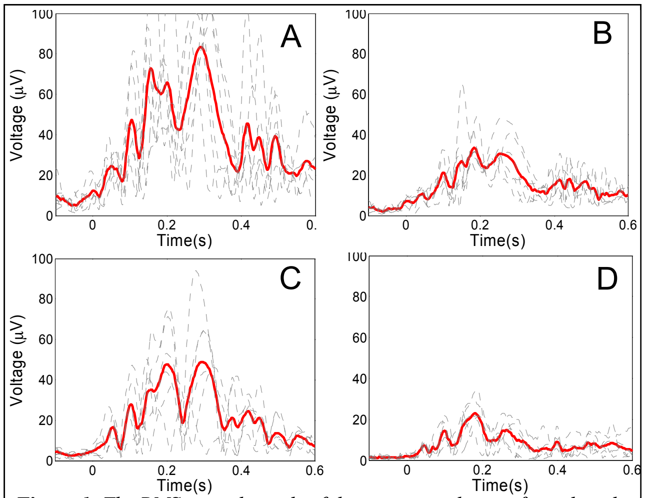

Figure 1: *The RMS over channels of the average pulse artefact when the subject's head is:* ***A)*** *relaxed;* ***B)*** *restrained;* ***C)*** *insulated from the EEG cap;* ***D)*** *restrained and insulated. Individual subject data (dashed lines) and the average over subjects (red line) are shown.*

Analysis

Initial analysis was carried out using Analyzer2 (v2.0.1, Brain Products) and involved detecting the R-peaks from the ECG trace using the peak detection method before filtering data to the 0.1-20 Hz frequency range. Data were then exported to Matlab, segmented using the R-peaks (-100ms to 600ms extent) and baseline-corrected based on the 100ms preceding each R-peak. The accelerometer data were integrated to give a measure of velocity. The mean PA on each channel was formed by averaging over 90 cardiac cycles and the variation of the root-mean-square (RMS) amplitude over channels was then calculated. The mean RMS amplitude over the cardiac cycle was formed and the average over subjects used as a measure of the PA magnitude in each condition. A similar measure of the velocity variation was formed. The standard deviation of the PA over the 90 cardiac cycles was also calculated and then averaged over channels and subjects. Wilcoxon signed-rank tests were used to assess the significance of differences in the amplitude and variance of the PA between the different conditions.

Results

Figure 1 shows the RMS of the pulse artefact for each of the different scanning conditions, while Figure 2 compares the mean RMS and standard deviation of the PA and velocity measures. Figure 1 demonstrates that although there is some variability, the temporal pattern of the PA is similar across subjects with the main peaks delayed by ~170ms and ~260ms relative to the R-peak (Fig 1A), in agreement with previous studies (6-7). A significant, 61% reduction in the mean RMS amplitude in the PA ($P<0.03$) was produced by restraining the head (Fig. 1B&2) while the RMS velocity recorded on the forehead was decreased by 74% ($P<0.03$) (Fig. 2B). However, the reduction in the standard deviation (std) of the PA was much smaller (16%, $P<0.05$). The insulating layer also produced a significant, 42% reduction ($P<0.05$) in the PA amplitude and, interestingly, also reduced the standard deviation of the PA by 37% ($P<0.05$). As would be expected, the measured head motion was unchanged (Fig. 2B) by the presence of the insulating layer. Use of both the restraint and insulating layer reduced the RMS and std of the PA by the greatest amounts (78%, $P<0.03$ and 52%, $P<0.05$ respectively) with the RMS and std of the velocity measured on the forehead also being reduced by comparable amounts (73%, $P<0.03$ and 42%, $P<0.05$ respectively).

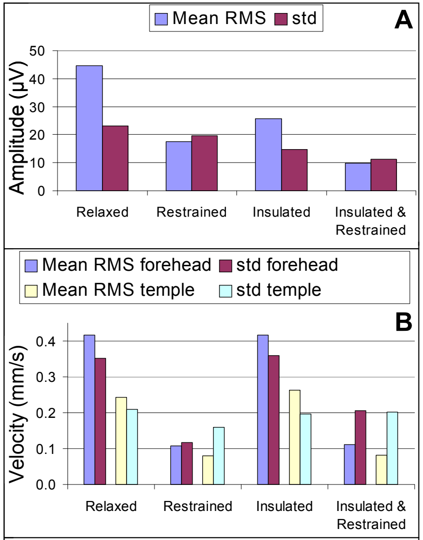

Figure 2: *The RMS and std averaged over subjects and cardiac cycle for:* ***A)*** *the EEG artefact;* ***B)*** *and head motion in each condition.*

Discussion

The larger reduction of the mean PA (and head velocity) which we measured when using the head restraint compared with the insulating layer confirms the hypothesis that cardiac-pulse-driven head rotation is the dominant source of the PA (7, 8). The additional reduction of the mean PA that was produced by combining the insulating layer and head restraint (44% reduction from restrained to restrained & insulated), with no corresponding reduction in the measured head velocity, could be explained by a contribution to the PA from flow-induced Hall voltages that is eliminated when the conducting path from vessels to electrodes is removed. The large reduction in the standard deviation of the PA (which reflects a reduced variation of the artefact across cardiac cycles) that was produced by the insulating layer, with no corresponding reduction in the standard deviation of the measured head velocity, also points towards a Hall voltage contribution which is more variable across heart beats than the pulse-induced head rotation. It is likely that any Hall voltage contribution arises from vessels in the scalp as previous modelling work indicated that arteries in the brain do not produce large enough voltages at the scalp to contribute significantly to the PA (7). The use of head restraint and the insulating layer still left a residual PA (Fig. 1D) of about 10μV in RMS amplitude which could be due to (i) residual pulse-driven head rotation or (ii) pulsatile scalp expansion. The larger RMS velocity measured at the forehead versus the temple is more consistent with (i). These findings indicate that restraining head movement by using a bite bar significantly reduces the magnitude of the PA, but does not produce a similar reduction in the variation of the artefact across cardiac cycles. It is therefore likely that such head restraint will not significantly improve the efficacy of PA correction when average template methods are used (1). Our findings also suggest that understanding the variation of flow-induced voltages across cardiac cycles will be important for the development of improved PA correction.

References **[1]** Allen *et al.* Neuroimage 8**:**229,1998, **[2]** Debener *et al* Neuroimage, 34:587, 2007 **[3]** Naizy *et al* Neuroimage 28:720, 2005 **[4]** Bonmassar *et al* Neuroimage 16:1127, 2002 **[5]** Nakamura *et al* IEEE Trans Biomed. Eng. 53:1294, 2006 **[6]** Debener *et al.* Int. J. Psychophysiol. 67:189, 2008 **[7]** Yan et al, HBM 31:604, 2010 **[8]** Anami, *et al*, Int Congress Series, 1232:427, 2002.