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Correlations between brain electrical activities of two spatially separated human subjects

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Abstract

Six channels electroencephalogram (EEG) were recorded simultaneously from pairs of separated human subjects in two acoustically and electromagnetically shielded rooms. While brain electric responses to visual pattern-reversal stimuli were elicited in one subject, the other subject relaxed without stimulation. EEGs of both subjects were averaged at times of stimulus onset, effective voltage of the averaged signals was computed within a running window, and expressed as ratio (Q) to the effective voltage of averaged EEG signal from non-stimulation periods. These ratios in non-stimulated subjects at the latency of the maximum response in stimulated subjects were analysed. Significant departures of Q ratios from reference distributions, based on baseline EEG in non-stimulation periods, were found in most non-stimulated subjects. The results indicate that correlations between brain activities of two separated subjects may occur, although no biophysical mechanism is known.

Keywords: Brain electrical activity; Brain-state correlations; Electroencephalogram; Visual evoked potentials

From the point of view of contemporary science, living organisms are open systems exhibiting continuous exchange of matter, energy and information with their physical environments, and the variety of the organism-environment interactions is well-known. In this paradigm, interactions or state-correlations between organisms are conceived via their physical environment exclusively.

During the last decade, however, observations suggestive of possible state-correlations between human subjects under conditions excluding sensory communication via their environment were reported. Grinberg-Zylberbaum et al. [3] reported experiments in which one subject of an 'empathically bound pair' was stimulated by light flashes, and claimed they had found wave-forms similar to visual evoked potentials (VEP) in the other, non-stimulated subject. This 'transferred potential' (their term) reportedly occurred only in a state of 'connectedness', with an empathic relationship between the subjects established by meditation *à deux*. In spite of speculative statements of the paper and obvious methodological and formal flaws, this report should not be too easily dismissed. Similar experiments were reported, partially with positive outcomes [2], although later replication attempts failed [4].

The aim of the present study was to examine the alleged correlations between brain electrical activities of two spatially and sensorily isolated subjects, one of them being exposed to simple sensory stimulation. Hereinafter, by 'correlations' we understand any detectable, stimulus-related correlations between brain functional states, and not cross-correlations between two spontaneous electroencephalograms (EEGs). The experimental set-up involved basic components of the earlier experiments, including the 'empathic bond' established by a 'tuning-in' procedure.

The experiments were carried out in two acoustically and electromagnetically shielded, air-conditioned, video-monitored experimental rooms of dimensions about $3.3 \times 2.3 \times 2.95$ m, separated from each other by ≈ 0.5 m empty space and from the adjacent control room by double doors. (Acoustic damping was -64 dB between the two chambers, -52 and -57 dB between the chambers and the control room. Electromagnetic shielding by a conduc-

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tive layer in the walls, ceilings and floors of the chambers, grounded by copper-band collectors.) C.S. served as the principal experimenter, collecting data for his diploma thesis, with H.K.'s assistance and under H.W.'s and J.W.'s supervision.

A total of 38 subjects, 17 pairs and four single persons, paid volunteers recruited by local newspaper advertisements, participated in the study (Table 1). Two experimental groups, each comprising seven pairs, were defined by their 'relatedness': one group (E_1) consisted of related, self-reportedly emotionally connected pairs (spouses, relatives, friends); the other group (E_2) was composed of unrelated individuals. The control group consisted of three related pairs (K_1) and four single subjects (K_2).

Before the experimental sessions, a 'tuning-in' period was arranged with pairs from the E_1 subgroup. They had to spend about 20 ± 5 min together in one of the experimental rooms to establish, non-verbally, an empathic bond defined as 'intense feeling of the presence of the other' and maintain it throughout the experimental session. They were otherwise given full freedom as to the choice of the method: by physical contact, by watching each other, or by meditating with each other. Thereafter, one member of the pair moved to the other room, plugged-in a connector to the EEG amplifiers, and gave a signal by an electric bell to the experimenter waiting in another room.

Interactions between the participants from the E_2 subgroup were avoided; they did not know of presence of another person in the other room during the experimental session. To balance the time schedule between E_1 and E_2, the unrelated subjects were given a comparable period of time, ≈ 20 min, to relax before the experiment started.

During the experiments, subjects were seated in comfortable reclining chairs, and the rooms were illuminated by diffuse dim light. The non-stimulated subject was merely relaxing. The stimulated subject was observing a $19''$ CRT monitor from ≈ 175 cm distance, focusing on a fixation cross in the screen centre. A checkerboard pattern, an array of 16×12 black and white squares (brightness ratio $\approx 1{:}100$, angular size $\approx 0.75 \times 0.75°$, seen from the subject's seat) was used to elicit visual evoked responses. Each 1-s stimulus consisted of a presentation of the pattern and three pattern reversals, following each other after 250 ms. The screen was blank during the inter-stimulus intervals, duration of which varied randomly in the range 3.5–4.5 s. Digital markers generated by the stimulation program at the onset of stimuli were recorded synchronously with the subjects' EEGs. Seventy-two stimuli were presented during each session, in a time period of about 6 min. (These stimuli were followed by a series of ten 10 Hz stimuli, 10 s duration each. The latter data were not analysed because of a high eye-blink artefact rate.)

The experiments with the group K_1 were carried out by the same experimental protocol as with the group E_1: EEG was recorded simultaneously from both subjects, the stimulation program was running, the monitor displayed the checkerboard pattern, but the front of the monitor was hermetically covered by an opaque, black cardboard shield. Thus the only difference from E_1 was that the subjects were *not* stimulated. The control group K_2 consisted of single non-stimulated subjects, from which the EEG was recorded; there was no subject in the stimulation room while the stimulation program was running.

EEG data were derived from six scalp electrodes attached at $C_{3,4}$, $P_{3,4}$, and $O_{1,2}$, against a reference electrode at C_z, using Grass 8 mm GoldCup electrodes with Grass EC2 paste, and keeping skin/electrode impedances <5 kOhms. Vertical electrooculogram was recorded from the right eye. Two EEG systems of the same type, BrainScope EADC220 by M&I Ltd., with software EASYS2, version 2.21, by Neuroscience Technology Research Ltd., were used. EEG was band-passed to 0.15–70 Hz, sampled at frequency 1024/second/channel, A/D-converted, and transmitted over serial lines to the computers in the control room, where the data were down-sampled and stored at frequency 256/second/channel. Off-line, the EEGs were digitally band-pass filtered

Table 1
Age and gender of participants in experimental groups $E_{1,2}$ (where one member of the pair was visually stimulated) and control groups $K_{1,2}$ (without visual stimulation)[a]

Group	Number of subjects			Age (years)			Pair relationship	
	Female	Male	Total	Range	Mean	S.D.	Related	'Tuning-in'
E_1	11	3	14	24–57	36.8	12.1	Yes	Yes
E_2	10	4	14	25–56	36.1	12.3	No	No
E total	21	7	28	24–57	36.4	12.2		
K_1	5	1	6	23–36	30.7	4.4	Yes	No
K_2	4	0	4	28–41	35.5	4.8	(single subjects only)	
K total	9	1	10	23–41	32.6	5.1		

[a] S.D. = standard deviation. The 'tuning-in' sub-column refers to the procedure described in text.

to 1.5–30 Hz, visually checked for artefacts, and artefact-free 1-s epochs (256 samples) were marked for subsequent data analyses.

The EEG epochs following the stimulus onset were centred and averaged:

$$x_t = \frac{1}{N} \sum_{n=1}^{N} (u_{n,t} - \bar{u}_n) \qquad (t = 0, \ldots, T-1) \tag{1}$$

Here t is the time index of the data sample, T is the entire epoch length (256 samples $\equiv$ 1 s), n is the index of the data epoch, N is the number of available data epochs, and $\bar{u}_n = (1/T) \sum_{t=0}^{T-1} u_{n,t}$ is the average baseline for the n-th epoch. This was done independently for both subjects of each pair; in the non-stimulated subject, the beginnings of data epochs to be averaged were determined by stimulus onset markers for the stimulated subject.

The effective voltage of the averaged signal was computed by integrating the squared voltage values within a running fixed-length box-car window,

$$V_{\mathrm{eff}}(t) = \sqrt{\frac{1}{l} \sum_{\tau=-l/2}^{+l/2} x_{t+\tau}^2} \qquad (t = l/2, \ldots, T - l/2), \tag{2}$$

where l is the length of the integration window (35 samples $\equiv$ 137 ms). The window length was set to the reciprocal value of the dominant frequency of the averaged EEG data, determined by the most frequent peak in Fourier spectra of the averaged signal (1), to filter out most of the residual oscillatory activity.

Expectedly, the $V_{\mathrm{eff}}(t)$ curves showed usually early local peaks (Fig. 1 right, A) corresponding to the VEP components in stimulated subjects, (Fig. 1 left, A), in some cases even later in response to cumulative action of the complex pattern-reversal stimulus. No such peaks were seen in the $V_{\mathrm{eff}}(t)$ curves for the non-stimulated subjects (Fig. 1 right, B).

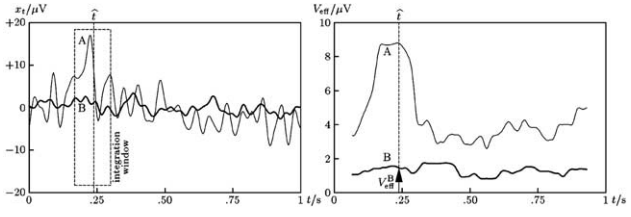

Fig. 1. Quantification of the EEG response in the stimulated subject A (thin lines) and its time-bound 'correlate' in the non-stimulated subject B (thick lines). – Left: Averaged EEG data (30 epochs) recorded from O_2 against C_z. Abscissae: time t in seconds; $t = 0$ indicates stimulus onset in the stimulated subject A. Ordinates: average amplitude in microvolts. The dashbox shows the integration window, centred at the maximum latency time $\hat{t}$ determined by the $V_{\mathrm{eff}}^{A}(t)$ response curve. – Right: Effective voltage curves $V_{\mathrm{eff}}(t)$ computed from the averaged EEG data. Abscissae: time t at the midpoint of the integration window. Ordinates: effective voltage in microvolts. Dashed vertical line marks the position of the maximal $V_{\mathrm{eff}}^{A}(\hat{t})$ response in the stimulated subject at time $\hat{t}$, at which the respective effective voltage $V_{\mathrm{eff}}^{B}(\hat{t})$ in the non-stimulated subject is read (vertical arrow).

Due to the null hypothesis there should be no time synchronous changes in the EEG of the non-stimulated subject; consequently, $V_{\mathrm{eff}}^{B}(t)$ should be randomly fluctuating. The alternative hypothesis is that a non-random variation, correlated with the response in $V_{\mathrm{eff}}^{A}(t)$, could be hidden in $V_{\mathrm{eff}}^{B}(t)$. To decide between the two hypotheses, we had to take into account the inter-individual variability of the baseline EEGs and thus to operate entirely on an intra-individual basis. For this purpose, N 1-s epochs (i.e. the same number as stimulation-bound epochs) were randomly drawn from a pool of artefact-free EEG recorded during the inter-stimulus intervals (IS). These data were centred, averaged (1), and their effective voltage

$$V_{\mathrm{ref}} = \sqrt{\frac{1}{T} \sum_{t=0}^{T-1} x_t^2} \tag{3}$$

was used to normalise, on an individual basis, $V_{\mathrm{eff}}(t)$,

$$\tilde{V}_{\mathrm{eff}}(t) = \frac{V_{\mathrm{eff}}(t)}{V_{\mathrm{ref}}} \,. \tag{4}$$

Latencies $\hat{t}$ at which $\tilde{V}_{\mathrm{eff}}^{A}(t)$ was maximal for stimulated subjects were determined separately for each EEG channel. The corresponding values $V_{\mathrm{eff}}^{B}(\hat{t})$ at the same latencies and channels, normalised with respect to V_{ref},

$$Q \equiv \tilde{V}_{\mathrm{eff}}^{B}(\hat{t}) = \frac{V_{\mathrm{eff}}^{B}(\hat{t})}{V_{\mathrm{ref}}^{B}} \,, \tag{5}$$

were taken for 'response correlates' in non-stimulated subjects. If the null hypothesis holds, Q is a random variable with mean expectancy $\langle Q \rangle = 1$, but other parameters of its distribution are unknown.

We used a non-parametric, randomisation statistics to test directly for deviations of Q values observed in data recorded synchronously with stimulation, from reference distributions based on IS EEG data for the non-stimulated subjects. N 1-s epochs were again randomly drawn from another pool of artefact-free EEG data (different from that used to calculate V_{ref}. IS data were averaged (1) and $\tilde{V}_{\mathrm{eff}}$) (t) calculated (2, 4) for all possible positions of the integration window $t = l/2, \ldots, T - l/2$. The minimum and maximum values of these $T - l = 222$ $\tilde{V}_{\mathrm{eff}}$ values defined a 'reference interval' $[\tilde{V}_{\mathrm{Min}}, \tilde{V}_{\mathrm{Max}}]$. The Q-values (5) were checked against the reference interval, marked as 'outliers' if $Q \notin [\tilde{V}_{\mathrm{min}}, \tilde{V}_{\mathrm{max}}]$, and the outliers counted. The procedure was repeated for twenty independent random selections of EEG epochs from the IS pools, yielding total counts C of outliers for each subject and electrode site.

The probability of an outlier to occur by chance (null hypothesis) is a small yet unknown number. (It is not exactly 1/222, because $\tilde{V}_{\mathrm{eff}}(t)$ were calculated for overlapping integration window positions $t = 0, \ldots, 221$ and thus the subsequent values were not independent.) Anyway, the total outlier counts C should approximately obey the Pois-

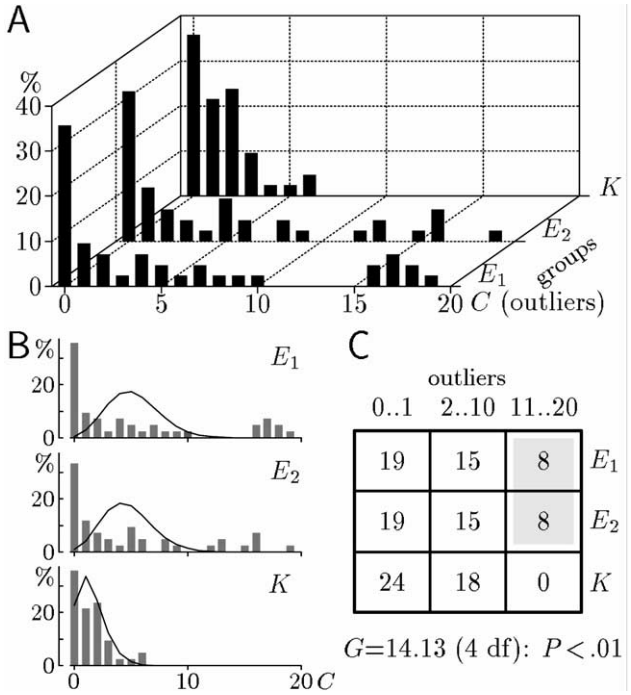

Fig. 2. Summary statistics of response correlates ('outliers') in non-stimulated subjects across all scalp locations. C = total count of outliers observed in 20 randomisation runs for each subject and location. – A: Histograms of outlier counts by classes E_1, E_2 and K. Abscissae: number of observed outliers. Ordinates: percentage out of seven subjects × six scalp locations = 42 observations. – B: Poisson probability function fits (Eq. (6)) of the outlier count distributions C. Same data as in section A (vertical bars), shown separately by classes E_1, E_2 and K with their respective Poisson function fits (curves). Only the control group K shows a good fit while the experimental groups $E_{1,2}$ deviate from the Poisson curve. – C: Contingency table of outlier counts collapsed into three bins ($0 \le C \le 1$, $2 \le C \le 10$, $11 \le C \le 20$) versus experimental groups $E_{1,2}$ and control group K. The sums across rows are $42 = 7 \times 6$ for each group. The contingency test criterion G indicates a strong dependence of the outlier count distributions on the group, i.e. the experimental condition.

son distribution

$$P\{C = n\} = \frac{\lambda^n}{n!}\, \mathrm{e}^{-\lambda}, \tag{6}$$

where the arithmetic mean $\bar{C}$ estimates the parameter λ.

Fig. 2A shows histograms of outlier counts C for the experimental groups E_1, E_2, and the combined control group K. The control group K displays a descending distribution, which can be roughly approximated by (6) with estimated $\lambda \approx 1.476$. The experimental groups E_1 and E_2 exhibit distributions similar to each other but strongly deviating from the Poisson curve fits ($\lambda \approx 5.238$ for E_1 and 4.714 for E_2), obviously due to unexpectedly high outlier counts $C > 10$ (Fig. 2B). For the final statistical test, the distributions of outliers have been collapsed to three bins (Fig. 2C), to reduce degrees of freedom. These numbers reveal no difference between the experimental groups E_1 and E_2, but strong deviation from the control group K: $G = 14.13$, 4 d.f., $P < 0.01$. (The G-test is more robust against small numbers in table cells [5] and thus preferable to Pearson's χ^2-test.)

These results indicate a high co-incidence of variations of the brain electrical activity in the *non-stimulated* subjects with brain electrical responses of the stimulated subjects. Since the data analyses relied on *individual* reference distributions based on EEG data for the same subject during the non-stimulation periods, the variations cannot be explained-out trivially by inter-individual differences between EEGs.

The results should not be interpreted as a successful replication of the 'transferred potential' [2,3]. We did not see any VEP-like wave-forms in the averaged EEG of the non-stimulated subjects: a more sophisticated data-analytic technique was necessary to detect an effect opposing the null hypothesis. There was no preferred direction of the effect; both increasing ($Q > \tilde{V}_{\max}$) and decreasing ($Q < \tilde{V}_{\min}$) effective EEG voltages were observed in non-stimulated subjects. Neither was there any 'locus of maximal effect'; the outliers occurred not only at occipital locations, i.e. homotopic to the areas primarily affected in the stimulated subject, but also in parietal and central regions. We thus should avoid the naïve and misleading term 'transferred potential'; whatever the nature of the effect is, it is *not* a transferred copy of the VEP in the stimulated subject.

The lack of directional and topographical consistency of the effect is physiologically counter-intuitive and suggestive of an erratic artefact. We were unable to identify any mechanism that could account for such an artefact. The experiments took place in separated, shielded rooms. Visual stimuli, unlike acoustic stimuli used in [2], eliminate the risk of sub-liminal sensory leakage. The pattern-reversal technique operates with much lower physical energies than flash-light tube used by [3]. Data were digitised in situ and error-free data transfer was verified by checking parity sum for each data sample. The final statistics was based on straightforward comparisons between the experimental and control groups, with only one essential factor varied, i.e. the presence or absence of a perceivable visual stimulus or of a perceiving subject.

It is thus possible that some of earlier findings [2,3] may indicate a real phenomenon. Note that our results concern brain state correlations under external stimulation of one of two subjects. Our experimental set-up and analysis methods do not allow any conclusions about EEG correlations occurring 'spontaneously' without stimulation. Neither can we account for possible 'experimenter effects'; interactions between experimenters and subjects were not varied but just minimised to not affect the empathic bond. However, the presence of the effects in both experimental groups E_1 and E_2 contradicts the assumption [2–4] about the crucial role of the subjects' 'connectedness'. If any kind of mental preparation of the subjects plays a role at all, then probably as a modulating, not the essential factor.

In conclusion, we are facing a phenomenon which is

neither easy to dismiss as a methodical failure or a technical artefact nor understood as to its nature. No biophysical mechanism is presently known that could be responsible for the observed correlations between EEGs of two separated subjects.

Nothing in our results substantiates the hypothesis [3] of a direct quantum physical origin of correlations between EEGs of separated subjects. However, another theoretical approach [1] might be considered, which generalises the notion of quantum entanglement as it is understood in ordinary quantum physics. It provides a formal framework for entanglement-like correlations in arbitrary systems, in all areas of science, as long as their properties do not commute with each other. The key condition for this generalised type of entanglement is *non-commutativity*: i.e. the sequence, in which two properties of the system are measured, matters for the measurement results. Whether or not this approach can be sensibly related to our findings, remains to be explored. Before that, more experimental work will be needed to secure the existence of the effect under strictly controlled and systematically varied conditions.

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