

Event-Related Electroencephalographic Correlations Between Isolated Human Subjects

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ABSTRACT

Objective: To examine electroencephalograms (EEG) in pairs of people to see if event-related potentials evoked in one person's brain are correlated with concurrent responses in the brain of a distant, isolated person.

Design: Simultaneously record EEGs using independent physiologic monitoring systems. One person relaxes in a double steel-walled, electromagnetically and acoustically shielded room while a second, located in a dimly lit room 20 meters away, is stimulated at random times by the live video image of the first person.

Subjects: Thirteen (13) pairs of volunteers. Eleven (11) pairs of adult friends and 2 mother–daughter pairs.

Outcome measures: Epochs of interest were the moments of stimulus onset and offset, ± 5 seconds, in both participants' EEGs. A positive correlation was postulated to appear between the ensemble variance of the stimulated subjects' EEGs versus an identical measure in the nonstimulated subjects. Control data using the same equipment and test conditions, but without humans present, was collected to check for equipment and analytical artifacts. Nonparametric bootstrap methods were used to assess statistical significance of the observed correlations.

Results: The control test resulted in a correlation of $r = -0.03$, $p = 0.61$; the experimental test resulted in $r = 0.20$, $p = 0.0005$. Three (3) of the 13 pairs of participants showed independently significant correlations. Examination of the stimulated subjects' event-related potentials showed that the stronger their responses, the larger the corresponding responses in the nonstimulated subjects ($p = 0.0008$).

Conclusion: Under certain conditions, the EEG of a sensorially isolated human subject can become correlated with event-related potentials in a distant person's EEG. This suggests the presence of an unknown form of energetic or informational interaction.

INTRODUCTION

Distant healing, including prayer, is one of the most popular alternative healing modalities and perhaps the most contentious from a scientific perspective (Wallis, 1996). Diverse cultural origins and philosophical assumptions have produced a panoply of different distant healing techniques, but all share the assumption that one person's mind can affect another person's body–mind at a distance (Schlitz et al., 2003). Controversy arises because the word “distant” in this context means shielded from all known energetic or informational interactions. This distinguishes distant healing from say, “energy” or “biofield” healing, which require a healer to be in close proximity to a patient.

Laboratory experiments and randomized clinical trials have provided replicable statistical evidence suggesting that mental intention can weakly influence distant living systems (Astin et al., 2000; Schlitz and Braud, 1997; Schmidt et al., 2004). Some of the early laboratory evidence has been criticized on methodological grounds (Schmidt and Walach, 2000), but many criticisms, especially those directed at clinical studies of distant healing, appear to be based on either *ad hominem* arguments or the belief that the underlying phenomena are theoretically impossible (Atwood, 2003; Park, 1997).

Because the topic of distant healing tends to evoke emotions that impede rational discourse, a more palliative approach might be to avoid tackling basic neuroscience assumptions head-on, and instead finesse the controversy by

adopting the assumption that all mental activity is correlated with brain activity (Crick, 1994). From this position, to study “mental influence at a distance” we conduct an experiment in which we ask two brain–minds to keep each other in mind, then stimulate one brain and see if the other shows correlated activity under conditions that exclude ordinary influences, expectations, and sensory cues.

The relevant literature can be traced to the origins of human neurophysiology. As a young man in the late nineteenth century, German scientist Hans Berger experienced a dramatic telepathic experience. In the early twentieth century, he was studying the electrophysiology of the brain in search of objective measures of subjective experience; in 1929, he published the first recordings of the human electroencephalogram (EEG) (Berger, 1940). Four decades later, two experiments were published reporting suggestive evidence for EEG correlations in isolated pairs, including student–teacher relationships (Tart, 1963) and identical twins (Duane and Behrendt, 1965), the latter funded by the National Institutes of Health and published in *Science*.^{*} Those two articles generated a number of conceptual replications, many of which were positive (Kelly and Lenz, 1976; Lloyd, 1973; May et al., 1979/2002; Millar, 1976; Millay, 1999; Orme-Johnson et al., 1982; Rebert and Turner, 1974; Targ and Puthoff, 1974). A decade later, Grinberg-Zylberbaum and colleagues reported detection of what they called transferred potentials in EEGs between isolated subjects (Grinberg-Zylberbaum and Ramos, 1987; Grinberg-Zylberbaum et al., 1993; Grinberg-Zylberbaum et al., 1994). Those publications stimulated a new series of replications, including those of Fenwick et al.,[†] Sabell et al. (2001), Standish et al. (2001), and Wackermann et al. (2003).

Overall, this body of research provides evidence that roughly 15% of pairs of people show nonchance, positive EEG correlations between isolated pairs of people. To be clear, the observed relationships are not independent EEGs that exhibit spontaneous, occasional correlations but relationships evoked as a result of stimulating one of the pair. Similar effects continue to be observed in each new generation of experiments, suggesting that the results are not because of measurement artifacts or obvious design flaws. The present study was conducted as a proof-of-principle replication using a new method of visual stimulation.

METHOD

Procedure

Pairs of friends were recruited for this study. No special relationships were required beyond sharing a mutual interest for participating in the experiment and a willingness to sign

an informed consent. Each person was asked to maintain a “feeling of connectedness” with the other. To encourage this focus, each person was also asked to exchange a personal item, like a ring or watch, and to hold it in the right hand for the duration of the experiment. Then the pair mutually decided who would be the sender (S) and receiver (R). For ease of exposition, sender means the visually stimulated person and receiver means the nonstimulated person, but these labels should not be taken to imply underlying mechanisms.

The experimenter used OmniPrep gel (Weaver & Co., Aurora, CO) to prepare participants’ skin at C_z according to the 10–20 electrode placement system (Jasper, 1958), and also the left and right mastoid processes (which acted as reference and ground, respectively). Then EEG electrodes (Biopac Systems, Goleta, CA, type EL258S, 8-mm diameter Ag/AgCl) were applied to the three sites using Microlyte electrode gel (Coulbourn Instruments, Allentown, PA). Electrode impedance was measured with a UFI (Morro Bay, CA) Model 1089e “Checktrode” to achieve minimum impedance. The C_z electrode was secured with a flexible band from under the chin to the vertex, and the other two electrodes by double-sided adhesive collars.

Available equipment made it possible to record only one EEG site on each participant; for the sake of symmetry the same site was used for both individuals. C_z was selected because previous studies have yet to demonstrate that any specific region is maximal for detecting the reported EEG correlations, and C_z is a convenient site for whole-brain monitoring. For exploratory purposes, electrodermal activity (EDA) was also monitored using electrodes attached to both participants’ left hand index and ring fingers (Biopac GSR-100C EDA amplifier, set to the 0–2 μ S range, with Biopac Systems type TSD203, 8-mm Ag/AgCl electrodes and Biopac GEL101 isotonic electrode gel).

R sat in a reclining chair located inside a solid steel-walled, electromagnetically and acoustically shielded chamber (Lindgren/ETS, Cedar Park, TX). All electrodes were connected to a Biopac Systems M150 physiologic recording system set to a 125-Hz sampling rate. The Biopac EEG-100C amplifier was set to 50,000 amplification, and filters were set to pass signals from 1–35 Hz. The interior of the shielded room was illuminated with a 25-watt incandescent lamp, and an infrared-sensitive video camera (Sony Model DCR-TRV140) was focused on R’s face. The Biopac and video signals were routed outside the shielded room via optical fiber (models 2809/2010 and model 2550; SI Tech, Batavia, IL) to computers that monitored the EEG signals and controlled the experiment.

R was asked to simply relax for approximately 30 minutes while maintaining a mental connection with S. After R was secured in the shielded room, S. was led through two closed doors to a dimly lit room 20 meters away (Fig. 1), and asked to sit in a chair approximately a half-meter in front of a video monitor. S’s electrodes were connected to the same model Biopac system as R’s, using the same amplifier settings and data sampling rate.

^{*}It was recently learned through the Freedom of Information Act that this study was actually funded by the Central Intelligence Agency (Playfair, 2003).

[†]Fenwick BCP, Vigus N, Sanders S. The transferred potential (unpublished manuscript).

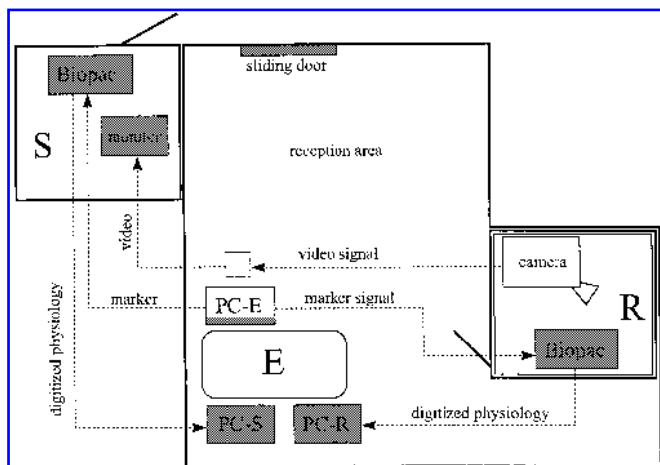


FIG. 1. Laboratory layout and infrastructure. The experimenter's workstation (E) consisted of three computers. Two recorded the physiologic data from the sender (PC-S) and receiver (PC-R) Biopac systems (Goleta, CA); the third (PC-E) controlled the random timing of the stimuli and a video switch. The receiver (R) was in an electromagnetically and acoustically shielded room; the sender (S) was in a distant room behind two doors and a double wall.

The digitized outputs from the Biopac systems were transmitted over a local area network and streamed to two Windows 2000 PCs, each running Biopac's Acknowledge 3.7.1 data collection software. The experiment was controlled by a third Windows 2000 PC running a program written by the author in Microsoft Visual Basic 6.0 (VB6). When this program was launched, it created a random timing schedule for 17–25 fifteen-second epochs, with randomness based on VB6's pseudorandom algorithm seeded by the PC's clock at the beginning of each session. Each of these epochs was separated from the next by a randomly determined 5–25-second interepoch period. At the beginning of each such epoch, the computer switched the video signal from R's chamber to the video monitor in front of S and simultaneously sent on-set marker signals to both Biopac systems using TTL signals generated by Ontrak Control Systems (Sudbury, Ontario, Canada) model ADR-100. At the end of each epoch, the computer switched the video signal to black and sent off-set marker signals to the Biopac systems.

The randomly timed appearance and disappearance of R's live video image was used to generate an event-related potential (ERP) in S's brain (Hillyard and Anllo-Vento, 1998). Unlike stimuli commonly used to optimize visual evoked potentials, such as stroboscope or reversing checkerboard patterns, the present study employed a less vibrant stimulus for two reasons: First, S was asked to sustain a sense of connection with R at all times. To avoid overwhelming or distracting S with flashing lights while trying to maintain this subjective state, R's image was used as the stimulus. Second, the live video image served as a novel, repeated reminder of the "mental connection" task. Real-time video was used rather than a static picture because video provides a stronger sense of presence and immediacy.

The epochs of interest were the EEG signals during the stimulus transition periods at the beginning and end of each epoch (i.e., those moments when the computer switched the image on the video monitor on or off) as these would be associated with ERPs in S. Neither S nor R were informed in advance about the number of transitions or the precise length of a session, R was blind to the length of the video epochs, and both participants and experimenter were blind to when the epochs would take place. In this latter sense, the experiment was conducted triple-blinded.

After both participants were secured in their respective rooms, the experimenter verified that the Biopac systems were recording EEG, EDA, and marker signals properly. If everything appeared to be in working order, the experimenter started the controlling program and attended to other tasks while waiting for the session to end.

Control test

A spurious positive correlation may arise between two sets of simultaneously recorded EEGs if the method of generating the visual stimulus causes electromagnetic pulses that are detectable in both EEG amplifiers at the same time. (In this test the video monitor's power remained on at all times; the display was switched from R's image to a blank screen.) Another artifact might arise if the marker signals (0- or 5-V DC signals) sent from the controlling computer to the S and R Biopac systems influenced the EEG amplifiers in those systems. The experimental setup was designed to attenuate such artifacts through use of an electromagnetically shielded chamber, fiber optics, and independent, battery-based uninterruptible power supplies for each Biopac system. As an additional safeguard, the experimental system was run under conditions identical to those in the experiment, but without humans present. The EEG and EDA electrodes were connected to the Biopac amplifiers, ungrounded, thus acting as antennas to provide maximal sensitivity for detecting potential electromagnetic pulses. Data from these sessions were analyzed in the same way as the experimental data.

To check for possible sensory leakage paths from S to R locations, a series of audio tests were conducted. These demonstrated that 1-sec 110-dB tones at 1000 Hz sounded in S's room could not be detected by a person secured in R's shielded chamber. Quantitative testing (Digital Sound Level Meter Model 840028, Sper Scientific, Scottsdale, AZ) confirmed that such tones were indistinguishable from background noise inside the chamber.

Hypotheses

Hypothesis 1 postulated that when S was exposed to visual stimuli, thereby creating ERPs, then an isolated R thinking about S would show corresponding ERPs at about the same time. These concurrent ERPs would be detected as a positive sender–receiver (S–R) EEG correlation.

Hypothesis 2 postulated that the magnitude of R's ERPs

would be related to the magnitude of S's ERPs. This goes beyond the postulate of a mere correlation and suggests that the S–R relationship is causally modulated by S's response to the stimulus.

Analytical procedures

Hypothesis 1 was tested by examining S and R EEGs at each stimulus transitional moment (stimulus onset and offset) ± 5 seconds. While the ERPs of interest were postulated to appear close in time to the stimuli, 10-second epochs were used to provide a broader context in which to study the hypothesized effects. Samples from each 10-second epoch (1250 samples) were normalized as $z_{ij} = (x_{ij} - m_j)/s_j$, where i ranged from 1 to 1250, j ranged from 1 to the number of epochs in the experiment, x_{ij} was the raw EEG sample i in epoch j , and m_j was the mean and s_j the standard deviation of all EEG samples in epoch j . This normalization step created an epoch with a shape identical to that of the original EEG signal, but transformed into a uniform scale to allow epochs to be easily combined and analyzed across subjects.

Head movements and eye blinks can introduce artifacts into EEG measurements, thus it is common practice to reject such outliers before conducting additional analyses. The standard way of doing this is to visually inspect EEG traces and to reject epochs with identified artifacts (Stern et al., 2001). However, even if performed blindly this introduces an undesirable subjective element into the analysis, thus given the controversial nature of the hypotheses a more objective method was deemed desirable. The approach adopted was objective and applied uniformly to all data: all normalized samples beyond ± 3 standard normal deviations (i.e., $|z_{ij}| > 3$) were excluded from further analysis. This excludes only extreme artifacts, and as a result it does not minimize EEG signal noise. Of course, not minimizing noise also means that this method is conservative. That is, unless R systematically blinked or moved precisely when S was looking at R over closed-circuit video, then the presence of randomly distributed artifacts in Ss and Rs would make it more difficult to detect positive S–R correlations at the pre-defined moments of interest. On balance, it was judged to be more important to eliminate subjective assessments of the data than to minimize potential signal artifacts, so this simple rejection method was used.

The next step was to determine the ensemble variance (v) per sample, independently for S and R, where v was defined as:

$$v_i = \left(\sum_{ij} z_{ij}^2 / N_i \right) - m_i^2$$

and where

$$m_i = \sum_{ij} s_{ij} / N_i,$$

z_{ij} was the normalized EEG sample i , ranging from 1–1250 in each epoch j , and N_i was the number of samples at array position i across all epochs. Recall that the artifact rejection algorithm was designed to remove $|z_{ij}| > 3$, thus N_i was not necessarily identical to the total number of epochs in the ensemble.

The same calculations were then applied to the S and R data to produce two independent v arrays, each 1250 samples in length. Then the Pearson correlation between these v arrays was determined (call this the S–R correlation). To determine the statistical likelihood of the S–R correlation, a nonparametric bootstrap method was used (Blair and Karninski, 1993; Davison and Hinkley, 1997).

This method began by creating an array x , where each element x_j was selected uniformly at random (with replacement) from the range 1–1250, and where j ranged from 1 to the number of R EEG epochs. Each x_j was used to randomly time-shift the corresponding R's epoch j by shifting that array such that the original sample z_{ij} was reassigned as the first element of a epoch time-shifted by x_j samples. For example, say epoch $j = 3$ and the random starting point for $x_j = x_3 = 50$. Then the time-shifted sample say, $z_{t50,3}$, would point to the first original sample $z_{1,3}$, and likewise, $z_{t51,3} \rightarrow z_{2,3}$, $z_{t52,3} \rightarrow z_{3,3}$, et cetera. At the end of the array the remaining samples would be rotated accordingly, thus the sample $z_{t1250,3} \rightarrow z_{1201,3}$ is followed by $z_{t1,3} \rightarrow z_{1202,3}$, $z_{t2,3} \rightarrow z_{1203,3}$, and so on.

After creating j randomly time-shifts epochs, the R time-shifted ensemble variance array, say v_t , was formed as described above, and then the S–R correlation was recalculated. This process was repeated 10,000 times, each time applying new random time-shifts to each of the R EEG epochs. Then the statistic $z_t = (r - m_t)/s_t$ was formed, where t indicates time-shifted, r was the original S–R correlation, m_t was the mean of the time-shifted correlations, and s_t the standard deviation. This z_t score, transformed into a one-tailed p value, represented the probability of observing an S–R correlation as large or larger than the one observed in the experiment. The accuracy of this p value depends on the normality of the distribution of the time-shifted correlations, thus as a secondary check the 10,000 time-shifted correlations were rank-ordered by magnitude and the position of the original correlation was located within this ranking. Then the p value was simply that position divided by 10,000.

This bootstrap procedure took into account the autocorrelated structure of R's original EEG data. Under the null hypothesis, there should be no synchronization between S's and R's EEG signals, thus there should be no difference between a time-synchronized S-R correlation and the bootstrapped time-shifted S–R correlations.

Hypothesis 2 was tested by examining the peak variance in R's EEGs within 200 ms of the peak response observed across all S EEGs. Two subsets of R data were examined: those epochs in which S showed the largest ERPs, and those with the smallest ERPs. This hypothesis proposed that R's

ERPs were causally linked to the magnitude of S's ERPs. A nine-step analytical procedure was followed:

1. Determine for each S epoch j the maximum z_{ij} value from the onset or offset of each stimulus up to one second post-stimulus; call these maximum values z_{max} .
2. Identify those S epochs where z_{max} values were larger than a maximum threshold value selected to identify at least 50 such epochs; call this subset of epochs $\{max\}$.
3. Find the peak value of the R ensemble variance array v , that is, variance across all Rs, from each stimulus up to 1 second afterwards; call the time where this peak occurred p and the associated variance value v_p .
4. Determine the R ensemble variance array v for the subset of $\{max\}$ epochs identified in step 2; call this array $v_{\{max\}}$.
5. Determine $\rho_{max} = \sum_{j=p-12}^{j=p+12} v_j$; this is the sum of 25 successive values (equivalent to 200 ms) in the R variance array $v_{\{max\}}$, centered around and including the sample v_p .
6. Evaluate the probability of ρ_{max} using the time-shifted bootstrap method, as described above; this results in the normalized score z_{max} .
7. Identify those S epochs with z_{ij} values smaller than a threshold designed to select out at least 50 epochs; call this subset of epochs $\{min\}$.
8. As in steps 4–7, calculate $\rho_{min} = \sum_{j=p-12}^{j=p+12} v_j$; based on the R array $v_{\{min\}}$, ultimately resulting in z_{min} .
9. Calculate $z_{\Delta} = (z_{max} - z_{min})/\sqrt{2}$. The hypothesis predicts that z_{Δ} would be significantly positive.

RESULTS

Participants

Participants included 13 pairs of volunteers, 17 females and 9 males (mean age, 36; range, 11–65). They included friends, mother–daughter pairs, and staff members of the Institute of Noetic Sciences (IONS). Nine pairs were fourth-year medical students participating in a month-long residential rotation on alternative and complementary medicine at IONS.

Sessions

The initial three sessions consisted of 17, 20, and 24 epochs; the number of epochs were varied to experiment with different session lengths. The last 10 sessions were set to a uniform 25 epochs. This produced 311 stimulus onset and offset transitions, for a total of 622 epochs. The artifact rejection algorithm rejected 10,829 of 1,555,000 samples (i.e., 1250 samples \times 622 epochs \times 2 participants per session), of which 5748 were R samples and 5081 were S sam-

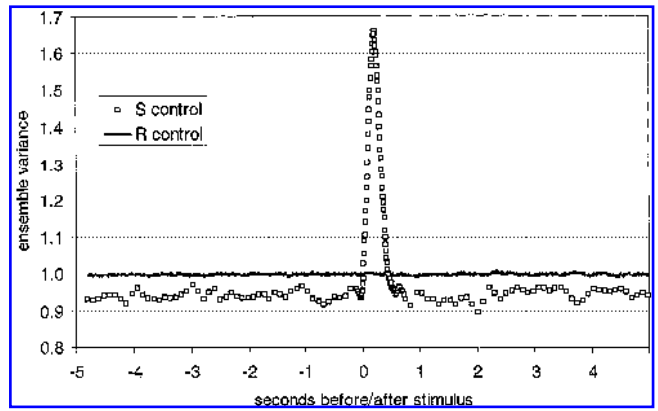


FIG. 2. Smoothed normalized variances for the electroencephalographic (EEG) amplifiers in the sender and receiver rooms, without humans present, based on $n = 300$ epochs. The sender's EEG amplifier signal was substantially over the baseline within 16 ms from the stimulus, which corresponds to the amount of time a standard video monitor takes to display an image. The signal peaked 104 ms after the stimulus. No comparable change was observed in the receiver's EEG amplifier signal.

ples. Thus, subsequent analyses were based on 99.3% of the original data.

Analysis of the distribution of rejected samples indicated that on average 4.6 out of 1250 samples were rejected from each R EEG epoch. A χ^2 test indicated that the rejected samples were uniformly distributed within the R epochs, χ^2 (1249 df) = 1158, $p = 0.97$. On average, 4.1 samples were rejected from the S EEG epochs. The χ^2 test showed that the rejected epochs were not uniformly distributed, χ^2 (1250 df) = 4819, $p \ll 0.001$, because of the vast majority of outliers clustering around the stimuli. This was to be expected because S's were exposed to explicit stimuli and R's were not.

Control test

Figure 2 shows the ensemble S and R normalized variance arrays in the control condition (i.e., no humans present), smoothed with a 200-ms sliding average to enhance clarity. The graph shows that the S's EEG amplifier detected the electromagnetic pulses associated with switching an image on and off in a nearby video monitor. It also shows that the R EEG

TABLE 1. RESULTS OF BOOTSTRAP TEST ON EXPERIMENTAL AND CONTROL S–R CORRELATIONS

	Experimental	Control
Observed S–R r	0.20	–0.03
Bootstrap mean r	0.00004	0.03
Bootstrap sd	0.06	0.23
z	3.32	–0.28
p	0.0005	0.61

S, sender; R, receiver.

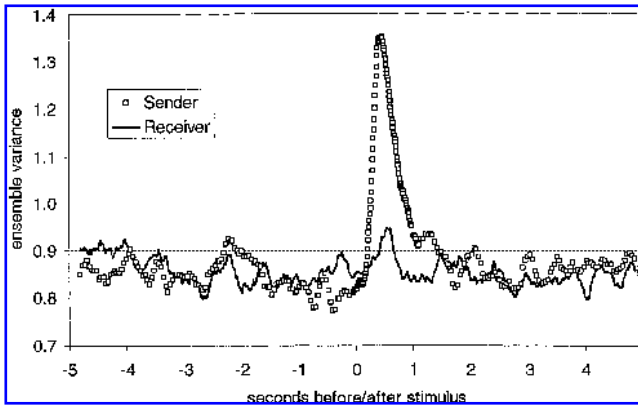


FIG. 3. Smoothed normalized variances for sender and receiver electroencephalographs (EEGs), based on $n = 622$ epochs. The senders' EEG peaks at 368 ms poststimulus; the receivers' EEG peaks 64 ms later at 456 ms poststimulus.

amplifier was adequately shielded from this pulse. As shown in Table 1, the S-R correlation for these curves was not significant, $r = -0.03$, $p = 0.61$ (one-tailed).

Sender-receiver relationships

Figure 3 shows the same analysis applied to the experimental data. The S EEG variance peaked in response to the video stimuli at 368-ms poststimulus and the R EEGs peaked 64 ms later. Table 1 shows that the S-R correlation postulated in Hypothesis 1 was confirmed, $r = 0.20$, $p = 0.0005$.

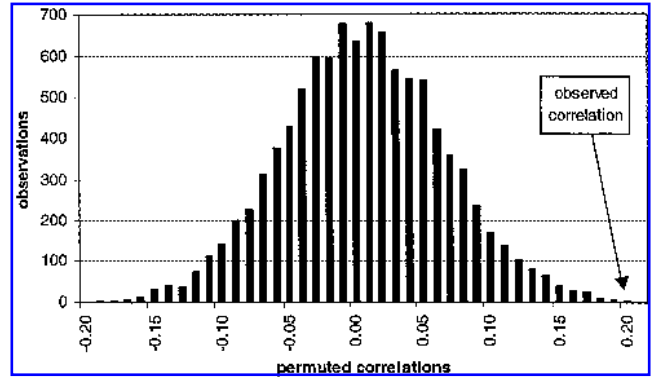


FIG. 4. Histogram of time-shifted correlations.

Figure 4 shows the distribution of randomly time-shifted correlations in comparison to the observed correlation. These correlations indicate what could have happened in this experiment if the timing of each stimulus had randomly differed from the original timing by up to ± 5 seconds. In this particular case, out of 10,000 repetitions, two time-shifted correlations were greater than the observed $r = 0.198$, thus $p = 2/10,000 = 0.0002$. The estimated $p = 0.0005$, based on the mean and standard deviation of this distribution, was therefore somewhat conservative.

The cross-correlation in Figure 5 indicates that the correlation was closely time-synchronized to the stimuli.

Hypothesis 2 is supported as shown in Figure 6. As subsets of increasingly stronger sender ERPs were selected, the

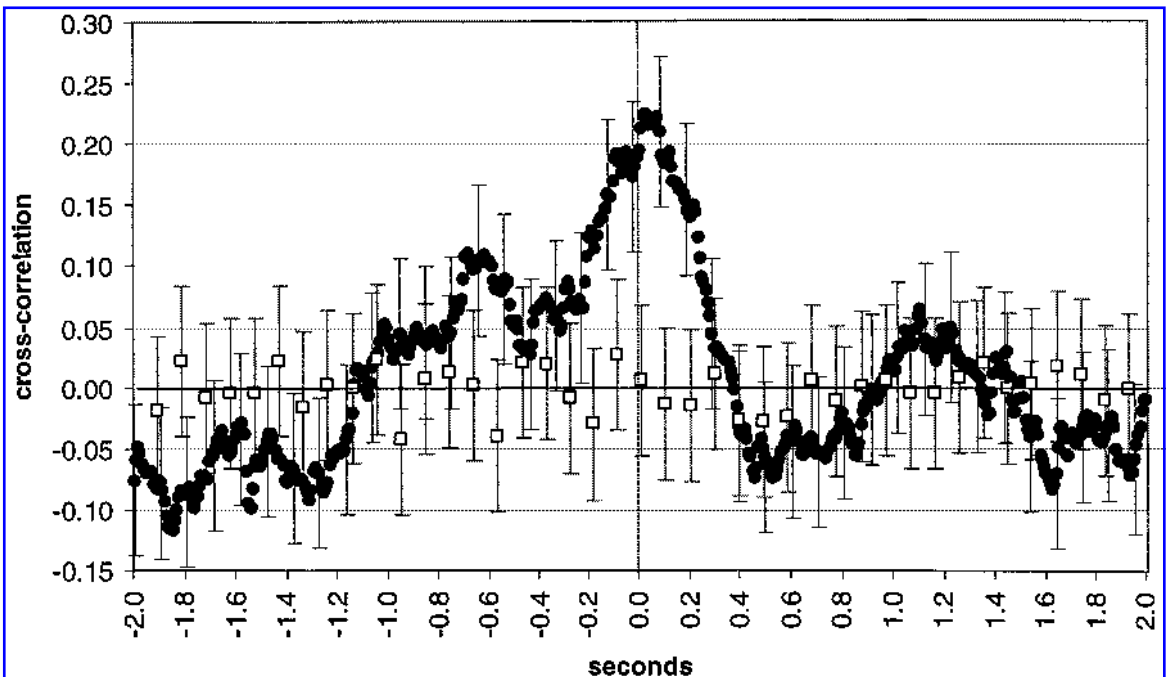


FIG. 5. Cross-correlations for experimental (black circles) and control data (white boxes), with one standard error bars. At the stimulus (time 0), the cross-correlation is 3.3 standard deviations over chance; 64 ms later (i.e., receiver [R] lagged sender [S] in time), the cross-correlation peaked at 3.5 standard deviations over chance.

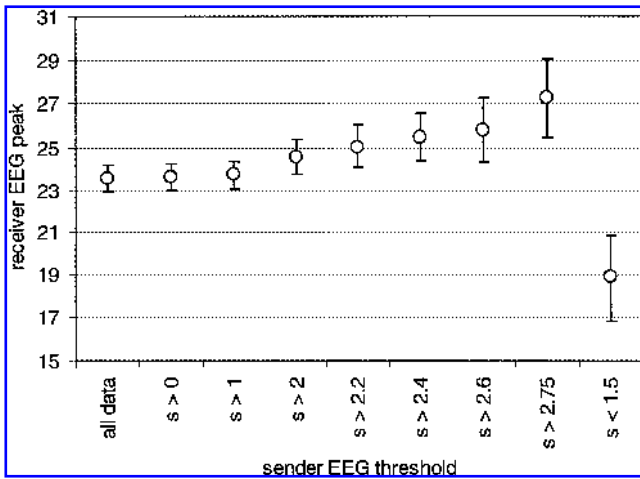


FIG. 6. Sum of receiver electroencephalographic (EEG) variance for 1 second with one standard error bars determined by bootstrap statistics, with different thresholds for the senders’ EEG responses to the stimuli.

magnitude of the receivers’ peak variance also increased. And with a subset of weaker S responses, the R peak variance declined. As predicted, the difference between two approximately equal subsets (strong S responses, shown as “s > 2.75” in Fig. 6, $n = 65$ epochs, versus weak responses, “s < 1.5,” $n = 71$ epochs) was in the predicted direction ($z = 3.15, p = 0.0008$).

To investigate whether the overall results examined in the above three hypotheses might have been the result of one exceptional session, or perhaps to a few epochs with coincidental ERPs, the S–R correlations and peak R responses were calculated separately for each pair of participants. Figure 7 shows that 3 of 13 pairs of participants independently achieved significant S–R correlations (exact binomial $p =$

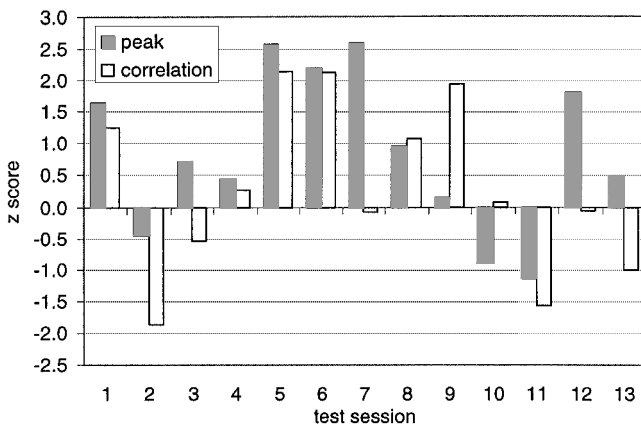


FIG. 7. Results for each of the 13 sessions expressed in terms of standard normal deviates (z scores), derived from the p values produced by bootstrap statistics. “Peak” refers to the peak receiver electroencephalograph as evaluated in Hypothesis 3; “correlation” refers to the S–R correlations. S, sender; R, receiver.

0.02), 5 Rs showed significant EEG peaks ($p = 0.001$), and 10 of the 13 Rs showed positive EEG peaks. Thus the ensemble results appear to reflect a generalized S–R relationship.

DISCUSSION

The central hypothesis in this experiment is that there is some form of unknown informational or energetic connection between isolated people. To test this idea, it is necessary to exclude all known sensory linkages. This was accomplished through the use of a heavily shielded chamber, and through controls for artifacts. We therefore know that the observed correlation was not caused by EEG amplifier cross-talk, or to participants’ anticipatory responses, or to spontaneous EEG correlations that may arise between isolated autocorrelated sequences, or to biases introduced through subjective identification of EEG artifacts.

If not because of known facts or artifacts, how else may we understand this correlation? One approach is to look for physical principles that might provide theoretical support for the observed connections. In that spirit, the relationship observed in this study is reminiscent of quantum entanglement (QE). This refers to a class of properties in which isolated physical systems display correlated behavior indicating that they are not as separate as they appear to be (Kwiat et al., 2001; Pan et al., 2000; Rowe et al., 2001). If macroscopic physical objects, including the brain, can exhibit QE properties for even short periods of time, as suggested by Hagan et al., (2002), Josephson and Pallikari-Viras (1991), Stapp (1988, 1997) and others, then it is conceivable that entangled brains might support correlations like those observed in this experiment.

Objections to this speculation include the observation that QE as presently understood is an exceedingly fragile state that requires conditions quite unlike the hot, wet environment of the human brain (Stenger, 1995). However, recent advancements in QE have shown that generalization of the underlying mathematics to higher dimensions results in robust forms of QE that are highly resistance to noise (Juls-gaard et al., 2001), that concepts such as “quantum repeaters” and “entanglement purification” are being pursued as practical means of extending the robustness and lifetimes of isolated QE systems (Duan et al., 2001), and that it is now possible to sustain entanglement of macroscopic objects (gas clouds with trillions of atoms) at room temperature for milliseconds, an extremely long lifetime in this context (Collins et al., 2002).

As advancements in QE continue, it seems increasingly plausible that some form of entanglement may be discovered that is sustainable in living tissue. If such a development does occur, by itself it would not explain the present EEG correlations, but it would provide a physical foundation on which to build such explanations, and it would fur-

ther deflate assertions that such phenomena are *ipse dixit* impossible.

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