This is the second in a series of featured articles, brief and controversial, on medical, psychological, or related issues. I hope to stimulate discussion, letters, and interaction in Telicom and also possibly on outside forums, such as ISPE-net. I focus on the areas where the mythology may need to be broken and where limitations may not necessarily be recognized.

Brain, Reductionism, Neuroscience, Controversy and Data.

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Abstract *Recent findings in the neurosciences have attempted to correlate subjective experiences with specific brain findings. This paper reviews the most important research in this regard involving stimulating areas of the brain to produce out of body experiences, attempted correlations of near-death experiences with REM intrusion and other phenomena, the links of the temporal lobe of the brain with subjective paranormal experience and the use of functional magnetic resonance imaging to clarify extrasensory perception. Correlations should not be regarded as causal and the author proposes a new bidirectional approach for causality. Also, results of brain findings neither confirm nor deny the veridicality of the subjective event, which may have origins in the brain or outside, but may reflect a link in the event with that specific area of the brain. The author applies a baseball analogy metaphor, written in farce, to illustrate the double-standards sometimes applied to such research.*

Key words baseball analogy, bidirectional causality model, brain, extra-sensory perception (ESP), functional MRI (fMRI), hallucination, INSET, near-death experience (NDE), out-of-body experience (OBE), neurosciences, phenomenology, Possible Temporal Lobe Symptoms (PTLSs), pseudoparsimony, pseudoskeptics, psi, REM intrusion, reductionism, subjective paranormal experience (SPE), temporal lobe, Temporal Lobe Questionnaire (TLQ).

"Since its inception in the early 20th century, neuroscience has taught us a tremendous amount about the brain. Our sensations have been reduced to a set of specific circuits. The mind has been imaged as it thinks about itself, with every thought traced back to its cortical source. The most ineffable of emotions have been translated into the terms of chemistry, so that the feeling of love is just a little too much dopamine. Fear is an excited amygdala. Even our sense of consciousness is explained away with references to some obscure property of the frontal cortex. It turns out that there is nothing inherently mysterious about those 3 pounds of

wrinkled flesh inside the skull. There is no ghost in the machine.

The success of modern neuroscience represents the triumph of a method: reductionism. The premise of reductionism is that the best way to solve a complex problem—and the brain is the most complicated object in the known universe—is to study its most basic parts. The mind, in other words, is just a particular trick of matter, reducible to the callous laws of physics. But the reductionist method, although undeniably successful, has very real limitations."[1]

We live in exciting times.

Are so-called "out of body experiences" simply artifacts of distorted brain functioning? Can we demonstrate that "out of the body" is actually "in the body"?

Can we show that the "near death experience" is nothing more than a particular brain wave patterning producing bizarre experiences?

Can we reduce all our "psychic", "paranormal", "intuitive" subjective experiences just to a dysfunction of a part of the brain?

And can we actually photograph the changes in our brain finally destroying that pesky mythology that surrounds extra-sensory perception?

Let's examine briefly our late twentieth century and early twenty first century perspective. It would certainly be gratifying to neuroscientists if we could just become a mass of microtubules, and a controlled but amorphous protoplasmic mess. We would be able to tame the ineffable concepts of "life", "consciousness", "reality", "identity", "self" and even "creativity", "genius" and "intuition". We would once and for all pack that ghost into the machine and heave a sigh of relief that we need not unthink anything we've thought before.

Brain Stimulation and the Out of Body Experience:

The problem:

The best way to examine such ideas is to examine the fabric of the subjective experiences that are amongst the most threatening to our current world view, namely subjective paranormal experiences (SPEs).[2] I developed this as a non-prejudicial term to examine such phenomena. This way, I could evaluate ostensibly anomalous or psychic or intuitive experiences with a similar face validity approach that I would use in my examination of auditory hallucinations.[3]

The approach then neither confirms nor denies the validity of the SPE itself, and does not label it as pathological or "normal". Instead, SPE allows for analyzing links with areas of the brain so as to understand the ultimate expression of the experience. Charley Tart became the first modern investigator of EEG correlates of out-of-body experiences (OBEs), a term he developed in the 1960s[4], and pointed out that naturally occurring OBEs are psychologically important as they are a primary cause of a belief in souls.[5] Finding some EMG correlates of restless leg syndrome is interesting, e.g., but we wouldn't call it the higher level phenomena of walking.[6] Whether the specific findings here will eventually be shown to be part of the causal

mechanism of OBEs is unknown, but future research should study the full-scale OBE complex, not only possible component phenomena.[6]

The controversy:

This area has become particularly controversial since the year 2002, when a stimulation of a particular area of the brain, the angular gyrus, in a patient with a right temporal seizure disorder produced the subjective report in the patient of the patient saying she was out of her body.[7] This was replicated on further stimulation. Subsequently, the same researchers[8-12] and others[13] have reported stimulation of similar, but slightly different areas of the brain (e.g. right hemisphere[14] producing these out of body experiences and a case of persistent tinnitus (ear-ringing) also was linked with such experiences. In fact, such reports are not new.

Wilder Penfield in the 1950s already reported how he induced such an experience in the brain ("*Oh God! I am leaving my body!")*[15] as did Munro[16]. And the latest stimulation case producing "out of body experiences" (OBEs) has also been replicated in a patient with intractable tinnitus (ear ringing) who described a "sense of disembodiment"[17].

The perspective:

How do we approach such subjective out-of-body-experience (OBE) reports? With each, enormous interest is evoked as a brain site for the OBE is postulated.

First when one analyzes such OBEs induced by brain stimulation they are atypical enough to debate whether they in fact are phenomenologically OBEs—do they have the correct properties? They certainly are quite different from spontaneous, non-induced OBEs described by Subjective Paranormal Experients (SPEs). They are incomplete in that not the whole body is experienced as outside the body, and they may continue to perceive the environment from the visual perspective of the physical body.

They variably produced trivial illusion phenomena[18], distorted body-image, depersonalization and derealization, visual perceptions of specific fixed location, and associated other parieto-temporal state or trait features. These descriptions differ markedly from thousands of spontaneously reported OBEs in ostensibly "normal" individuals. These frequently involve subjectively extracorporeal consciousness with locality dependent perceptual experiences; clear imagery; polymodal perceptions and profound cognitive awareness; the environment (including the physical body) is accurately perceived from an extracorporeal perspective and this disembodied center of consciousness may move about independent of the physical body.[19, 20]

These descriptions are generally in individuals with brain dysfunctions—seizure foci or tinnitus. Generalization to other people without foci is not warranted. Additionally, when analyzing OBEs and for that matter comparable phenomena such as déjà vu and memory, no single localization can be found[21-23]. We can learn from other experiences: At least four distinct nosological subtypes of déjà vu exist.[24] Similar research on OBEs needs to be performed to demonstrate the likely subtypes that exist.[25]

The following method is logical to ensure the phenomenological (symptom, descriptive) purity of the data and correlate it with diagnostic groups or special

research groups.[26]

- Analyze these subjective experiences (SPEs) in as much detail as possible and compare them with the typical features of SPEs of those who do not give any history of brain dysfunction.
- Realize that any brain pathology the subject has a specific pathophysiological context.
- Do not generalize single cases to other humans and compare the literature; similarly, encourage detailed research learning from past knowledge.
- We should search for sources of single localization for specific phenomena but recognize the existence of nosological subtypes (e.g. as found in déjà vu)
- Even when findings are referable to specific anomalous brain functioning, they neither confirm nor deny the actual origins within the brain: one explanation for events may be endogenous origins within the brain like pathological hallucinations; or a particular brain function pattern may allow experience of an outside, usually covert, reality.
- Methodologically, associative links do not imply causality. To consolidate the causality hypothesis, one should analyze both normal populations for specific brain changes and also the converse e.g. find patients with those brain changes and analyze if they have the same SPEs.

Thus, these dichotomous epiphenomena of subjectively interpreted "out-of-body experiences" require careful phenomenological differentiation—the induced OBE apparently greatly differs from the spontaneous OBE. Using one term—OBE—for both endpoint expressions could produce incorrect clustering of entirely different phenomena (e.g., spontaneous OBE versus complex partial symptoms) or subtypes of OBE: Different origins and etiologies would be inappropriately interpreted as one.[26, 27]

The near-death experience:

'Let me show in allegory how far our nature is enlightened or unenlightened. … Human beings living in a underground den, which has a mouth open towards the light and reaching all along the den… they are strange prisoners… they see only the … shadows, …on the opposite wall of the cave… Would they not suppose that they were naming what was actually before them?

If told this were an illusion, would Man not fancy that the shadows he formerly saw were truer than the objects now shown to him? He will take refuge in the shadows which are clearer to him than the truth. … The truth may be nothing but the shadows of images.' Plato (condensed).[28] Is not possible that the shadow Man sees is his physical reality alone?[29]

Plato's famous cave allegory may be an important warning that not all brain waves reflect what we think they do.

The problem:

The OBE research on brain stimulation is closely linked with some work reducing so-called Near Death Experiences (NDEs), in which individuals report strange experiences during states near death (e.g. coma, or cardiac resuscitation). These vary but have some consistency: seeing non-physical beings, a tunnel and light experience, or being out of their body.[30, 31] Since its initial reports, these experiences have been debated in origin. The neurophysiologic basis of near death experience (NDE) is unknown. Are they from endorphins[32], or from imbalances of neurotransmitters[33], or from the temporal lobe[34]? How can it be that these experiences can occur when memories are lost during coma?[30, 31, 35-38]

The controversy:

The most recent controversy relates to linking Near Death Experiences (NDEs) to a state of sleep called REM intrusion, which is a kind of dream sleep with sleep paralysis or may occur during wakefulness or other clinical conditions, like narcolepsy.[39] Again, neuroscience has ostensibly explained this strange experience as purely brain linked. Is this an entirely brain related phenomenon linked with abnormal neurochemistry or wave forms.

The Nelson REM intrusion NDE article[39] has become so significant, that it is critical to see its limitations, and yet also use it as a jumping ground for research. They argue that NDE elements can be explained by REM intrusion; this is evoked by cardiorespiratory afferents in an arousal system predisposing to REM intrusion. They showed that the life-time prevalence of REM intrusion in 55 NDE subjects compared with an age/gender-matched control group and sleep paralysis as well as sleep-related visual and auditory hallucinations were substantially more common in subjects with an NDE. These findings argue that under circumstances of peril, an NDE would be more likely in those with previous REM intrusion which could promote subjective aspects of NDE and often associated syncope. Suppression of an activated locus ceruleus (involved with norepinephrine) could be central to an arousal system predisposed to REM intrusion and NDE.

There were methodological problems, however. This study had a retrospective design with distorted selection of subjects with NDE (only those 64 patients of 446 patients from the large internet site that recruited subjects responded to the authors). The NDE-ers chosen were also people who had contributed their experiences to a website, so might be likely to over-endorse questions about unusual experiences, thinking that the investigators were looking for them. They are therefore a selected subpopulation of NDE-ers, but this may be a methodological limitation that cannot be solved. Moreover, the NDE-ers REM symptoms were compared to a sample of hospital workers, who may have been particularly reluctant to admit they had bizarre symptoms. This may be the dissimilar group and conceivably, the NDE-ers may be similar to the general public in REM intrusions, though that group is also not a good one. A proper control group would be people who had been near death but did not have an NDE.

But let us rightly or wrongly assume the sample was adequate for illustrative purposes. We then have our neuroscience questions: Is the NDE simply an endogenous physiological phenomenon? Does it alternatively have a greater implication for survival of consciousness?

The perspective:

The important challenge to such NDE research is Occam's razor: We should explain anomalous phenomenon in a parsimonious way. However, we must be aware of what Stent has aptly called *premature parsimony*—a premature conceptual reduction by investigators that can distort our understanding. The object is to find appropriate explanations, not just the simplest. The explanation must be fruitful explaining all, not just some aspects.

In the Nelson research, the data is post-hoc[39]: they did not compare the NDE-ers' REM intrusions before and after the NDE. Could the NDE-ers as a consequence of their NDEs have experienced neurological insults that increased their rates of a variety of symptoms? Was the REM state intrusion the result of experiencing a NDE, not the explanation for the cause of NDE? Personally, I believe this *legitimate argument to be* unlikely. This is so as the classic REM advanced/ REM onset state is narcolepsy. The narcoleptic syndrome has sleep paralysis as a major feature. REM intrusion could also be a way to possibly describe the hypnagogic and hypnopompic states before and after sleep, possibly the cataplexic and diplopic states we commonly see in narcolepsy and certainly the overwhelming short day-time sleepiness in narcolepsy. The reason is *the proportions of REM intrusion, as in narcoleptic syndromes[40], seem to have a solid HLA linkage so there is precedent[41], and I suspect this predisposition is constitutionally more likely based as I have never encountered brain injury producing such changes—other changes yes but not specifically this one. But we also see a variety of other bizarre symptoms such as hallucinatory experiences.[42]* In fact, my working hypothesis is the narcoleptic like condition correlates with subjective paranormal experiences just as anomalous temporal lobe functioning does.

However, there is a terminological aspect here. The term REM intrusion is not commonly used. I suspect it should be limited to demonstrable REM occurring on polysomnography not based on historical interview or questionnaire data of symptoms. This in itself creates a further level of error. If the authors had used the less sexy term "sleep paralysis" correlating with NDE, then we might have said "so what"? Yet sleep paralysis is a major epiphenomenal expression of REM intrusion and this would therefore not be the correct dressing for newsworthiness. The parallel is an important one: If we were to find a cohort of patients with NDEs had a more rapid pulse than a control group, would that make news?

But let's now look at the other side:

- First, we need to re-examine fundamentals. What may be more relevant is that NDE-ers change spiritual attitude. Yet this is regarded as too psychologically theological for the conventional medical journals.
- Second, *the phenomena described are subjective sleep paralysis or subjective REM intrusion. In other words, we must be careful to read about experiences as proven using an EEG or sleep wake brain wave study. In this instance, the experiences are simply subjective anomalous or brain experiences. They are not objectively validated on such measures as sleep polysomnography.*
- Next, the presence of so-called REM intrusion in say a state of coma is

physiologically very dubious and unproven. This is a key. Because REM intrusion or sleep paralysis correlate with NDEs does not make them causal and they are unlikely to be so in the vast majority of NDEs.

There is no immediate reason why we need spend thousands on complex equipment when the medical approach has always been primarily on history and examination. History includes the eliciting of symptoms of both state kind—those that are occurring at the same time as the feature being examined—as well as trait signs, in which any symptoms or features that occur over a lifetime or extended period are examined but they are not occurring for example with the sleep paralysis or near-death experience. In the Nelson research only trait features were examined, limiting interpretations of the links with medical conditions or changes in specific foci of the brain. Additionally, like almost all models, the unidirectional approach was used—near death experients were evaluated for their REM intrusion symptoms by medical history. The bidirectional approach would be examining, for example, those presenting with REM intrusion features and looking at NDE correlates.

This approach is examined further in the next section.

The temporal lobe and subjective paranormal experience.

''But it is a miserable thing for a question of truth to be confined to mere presumption and counter-presumption, with no decisive thunderbolt of fact to clear the baffling darkness.'' William James[43]

The problem:

Reducing a system to its parts and discarding the critical synergism and gestalt relationships may lose perspective. Reductionism in science is an appropriate methodology, but that must include complex and possibly, at this stage, ineffable concepts such as "reality", "self and identity" and "consciousness" and "life" itself. We must continue to study these empirically and in controlled laboratory settings being careful to recognize our interpretations as limited without prematurely applying theoretical paradigms to the results available. Sometimes that lab setting is simply the medical clinical evaluation.

The clinical symptomatology approach has been a fruitful direction of interest for the neuroscience researcher. It is much cheaper than apparatus such as Functional Imaging, which can cost thousands per subject.

This is particularly important because this approach emphasizes the phenomenological aspect of neuroscience examining symptoms and clinical features so they can correlate with the particular subgroup being analyzed, for example, detailed information on olfactory hallucinations and correlations with subgroups, for example, temporal lobe epilepsy or subjective paranormal experients.[44]

This approach also illustrates *the bidirectional approach to causality. It applies what has been an unrecognized but standard medical model for centuries. It allows statements about correlations to reflect likely* causality.

The controversy:

Are such subjective paranormal experiences just a manifestation of the brain playing tricks on us? If so, is there an area of the brain where these tricks occur?

Our understanding of subjective experience and specifically SPEs from a physiological point of view would be greatly enhanced if we could pinpoint a section of the brain in which psi mediation occurs, or at least an area that plays a primary role. Such knowledge would provide at least three concrete benefits. First, by considering the functions performed by this part of brain, we could develop more incisive insights about how psi manifests. For instance, if the area plays a crucial role in the activation of memories, credence would be lent to the hypothesis that psi occurs by activating stored memories. Second, if momentary brain states could be found to correlate with the accuracy of discrete psi responses, progress could be made in predicting which particular psi responses (e.g., guesses on a card test) will prove to be correct. Third, attempts could be made through biofeedback, drugs, or other means to alter the functioning of this part of the brain to enhance psi performance.[45, 46]

The perspective:

We can apply these same analogies directly to psi and brain. Can we use a methodology based on the clinical medical model to assist our studies here? Let me whet your appetite by briefly discussing my own research on the temporal lobe, which with respect, pioneered this approach in the context of SPEs.[47, 48]

This research required the development of a measuring instrument, the Neppe Temporal Lobe Questionnaire (TLQ), so as to elicit symptoms that could be attributed to the temporal lobe of the brain. These were called Possible Temporal Lobe Symptoms (PTLSs).

Our later work used an upgrade of this instrument, the INSET[45, 46] (Inventory of Neppe of Symptoms of Epilepsy and the Temporal Lobe).

Similarly the research required development of measures to screen for so-called "psychic experiences" and thereafter to go into great detail to elicit whether subjects had had spontaneous subjective paranormal experiences and to use criteria for levels of subjective validation of the experiences. Again, in no way, does this imply the experiences objectively occurred as interpreted. The essence of such phenomenological research is to be non-prejudicial and approach experience in the same way as one would approach subjective reports of pain, or of dizziness or of hallucinations. There are ways of quantitating, and this way one is able to develop experimental and control (comparative) groups. Again, in our later work, the INSET incorporated such data.

The original work was my retrospective pilot study in South Africa. This examined whether more possible temporal lobe symptoms (PTLSs) are associated with ostensibly normal subjects claiming a large number of SPEs (Subjective paranormal experients) than with subjects claiming none (Nonexperients) and if so, whether any specific PTLSs stand out. Both state and trait symptoms were examined—if PTLSs occurred during or just before or after the SPEs then this would be a Temporal Lobe / SPE state; conversely, if they occurred at other times (as with the Nelson research on REM intrusion) these would be trait phenomena which may imply correlations of symptoms.

Initially, all Witwatersrand-based members of the South African Society for Psychical Research were studied. The research was conducted in 3 successive phases designed to exclude all but the final participants. Phase 3 also involved administration of the major research instrument, which was developed to elicit clinical features of epilepsy and temporal lobe functioning, including PTLSs. Despite a small endpoint sample size, SPE Experients exhibited significantly more PTLSs than SPE Nonexperients ($p<0.001$), with olfactory hallucinations being particularly common. These findings suggest an anomalous kind of temporal lobe functioning among the experients, but neither confirm nor deny the veridicality of their SPEs. This work is theoretically very important: For the first time since Descartes misrepresented the pineal gland, an anatomical area could reliably be regarded as a kind of interaction area for mind and brain—whether or not that "mind" may still be brain or allow an appreciation of such experience.

Subsequently, this work was replicated in a Canadian different population by Michael Persinger again in normal populations[49]. I did further work on Experients demonstrating a detailed qualitative kind of olfactory hallucination which overlapped with PTLSs but were also different[44]: the particular type of olfactory hallucination associated with SPEs is pleasant and perfumed; but it commonly co-exists with different unpleasant burning, or foul temporal lobe type hallucinations. Similarly, I extended this work to the déjà vu phenomenon demonstrating that there were at least four qualitatively distinct, homogeneous categories of déjà vu[21]: Subjective paranormal déjà vu experience was characterized by its time distortions and specific predictions component and occurred in Experients. Associative déjà vu occurred in "Normals" and essentially was vague with a lack of memorable and outstanding features. In the neuropsychiatric group, déjà vu experienced by temporal lobe epileptics was characterized by post-ictal (post-seizure) features and associated PTLSs. This type of experience did not occur in schizophrenics, whose déjà vu experiences were characterized by psychotic intrusions.

These kinds of findings reflect *correlations* only. To establish a higher probability of *causality* one needs to be bidirectional. This means using a novel methodology for the first time by applying the two converse rules: After examining subjective paranormal experients (and a suitable comparative non-experient group) for temporal lobe state and trait phenomena; my colleague, John Palmer, and I then did the converse, looking at patients with temporal lobe dysfunction and their SPEs. Again, we were able to demonstrate that this population has more SPEs than a comparative population.[45, 46] This produces what I call the "*bidirectional medical model of causality*". Effectively, physicians have been applying this through the ages. For example, in the model of malaria, the condition can be diagnosed clinically and then confirmed by isolating Plasmodium. Conversely, a malaria diagnosis may be made based on Plasmodium in the blood and then finding correlates with clinical malaria. This moves correlations closer to causality. Even though this is subjective here, the subjective experience model in no way also diminishes trying to correlate it with objective experience.

This temporal lobe work has phenomenological implications but was a critical milestone: for the first time, an area of the brain was demonstrated to be linked with SPE. Moreover, an extra component of these research projects has been that context of phraseology is important for neuroscience: Commonly when one key question is

asked, the consequent interpretations based on that one key question may be interpreted as relevant for the whole scientific area. The interpretations made are actually valid only for the key question, and if that may not represent the key area so that it may ultimately distort knowledge of that key area.

In conclusion, one can say that the temporal lobe, the great integrator of higher brain function, not surprisingly is associated with a variety of subjective experiences including at times, déjà vu and subjective paranormal experiences. There is more than a correlation. There are causal elements but that reflects only one component: The epiphenomenal expression of these features/ symptoms. They neither confirm nor deny the veridicality of these experiences: These may have origins in the brain or a particular anomalous pattern of brain function may allow the temporal lobe to integrate exogenous reality based outside experiences more easily.[50]

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Functional MRI scanning: visualizing extra-sensory perception

Years ago, Woody Allen used to joke that he'd been thrown out of college as a freshman for cheating on his metaphysics final. 'I looked within the soul of the boy sitting next to me,' he confessed.[51] Saletan's anecdote if true as attributed, may reflect purely Allen's humor. But it introduces the new phase of functional MRI scanners.

Does our very being stop with a mass of microtubules?

The problem:

Finally, I discuss the most recent trigger for this paper…the attempted visualization of the sublime: Research has become even more sophisticated, with a report out of Harvard that a functional Magnetic Resonance Imaging study attempted to demonstrate whether or not extrasensory perception existed.[52]

In functional MRI (fMRI) studies, one attempts in the neurosciences to demonstrate the functional, as opposed to anatomical, correlates of certain physiological changes that are occurring. One can study thinking or hallucinations or delusions or changes in emotion. We can experimentally study the brain's expression of what happens when people laugh or cry. The technology is truly remarkable, but is at its early stage and the resolution of functional change will no doubt increase enormously over the next decade.

The Harvard researchers have argued that their special methodology was the first to actually demonstrate that extrasensory perception does not occur as it would have to be processed by the brain, and they could find no difference between their model attempting to elicit telepathy, clairvoyance and precognition compared with a control group. Again, the research has caused enormous reaction from the press; in fact, Harvard had a whole press release on it.[53] This research looks persuasive until one re-examines the significant literature on positive results elsewhere which has not been mentioned by the conventional press, and one re-examines the "new method" and the conclusions drawn.

The controversy:

In the Harvard study, functional magnetic resonance imaging (fMRI) was used in an effort to document the existence of extrasensory perception. The researchers believed that fMRI would be more sensitive than using indirect behavioral methods and designed an experiment which they felt would increase the sensitivity of producing positive results for telepathy, clairvoyance and precognition. They concluded that there was no difference with extrasensory perception compared with not. Does this conclusively prove that ESP does not exist? "No," says Moulton. "You cannot affirm the null hypothesis. But at the same time, some null results are stronger than others. This is the best evidence to date against the existence of ESP. *Perhaps most important, this study offers scientists a new way to study ESP that avoids the pitfalls of past approaches.*"[53]

The perspective:

There are major problems with this Moulton study, however. The researchers did not find extrasensory perception in fifteen of sixteen pairs they were testing. That the home run was not hit (absence of ESP means they could not study the phenomenon), and during that absence of a home run, there was no physiological change. Even if there were no physiological change and a home run was hit, this would not mean disproof either, it would just mean that we had not found the endpoint anatomical correlates. (I amplify on this home run metaphor later).

However, it appeared that they did have a home run. In the sixteenth pair, they found significant results, which they tried to explain away as an artifact, and they concluded no such results occur. In reality, one such finding may be highly relevant because extrasensory perception is elusive even in a laboratory where statistical results are based on a low but relevant level of hits.

Effectively results should be taken in the complete context: Was objective ESP demonstrated? We do not know. The Harvard researchers possibly legitimately explained away their one positive result which therefore makes the rest of their methodology questionable, because if one positive can be explained, why not explain sixteen positives if necessary? The study design is flawed.[54]
Additionally, were their other correlations like high statistical elements against chance? No, and this requires a very large sample in experimental settings. And if the subjects did not demonstrate ESP then no comments can be made about the state of ESP as opposed to examining "gifted" subjects who may have the trait potential for ESP.

Moreover, if you ask subjects to think about an apple and an orange, the fMRI scans may find no differences between the two types of events. But they are still different thoughts, just not demonstrable.
In fact, there is no precedent justifying why the Harvard experimental procedure which is complex and untested might work at all.

Even if so-called ESP demonstrably occurred, we may not be measuring it with the correct instruments. For example should we be measuring changes in episodic brain waves, or specific anatomical loci?
Would it be appropriate to assume such changes are different from ordinary stimuli? If a visual ESP stimulus were given, would we see the same changes as a regular

visual stimulus? Would we see no changes because we are measuring the wrong phenomenon? Is the sensitivity of the instrument e.g. the fMRI adequate to detect such changes?
And if we found a change, would that change reflect all of ESP or just one specific kind? For example, in the above, there was a different test for so-called contemporaneous telepathy, contemporaneous clairvoyance and precognition. Also how specific are such changes? Can we characterize them in terms of correlations only? What would be required to demonstrate cause and effect?

It turned out the Harvard researchers did not cite the literature looking at extrasensory perception and fMRI, and in a later blog, Sam Moulton asked esteemed parapsychological researcher, Dean Radin, to delineate the literature they had missed.[55]

There are in fact several positive fMRI studies relating to ESP. Most come out of Bastyr University and the University of Washington in Seattle, WA, USA from the Standish, Johnson, Kozak, Achterberg, Richards group, where one positive result led to replications.[56-58] Moreover, Dick Bierman in Amsterdam has also done some important work on fMRI.[59, 60]

For example, in the Achterberg study[58], distant healing was evaluated. Significant differences between experimental (send) and control (no send) procedures were found (p= 0. 000127). Areas activated during the experimental procedures included the anterior and middle cingulate area, precuneus, and frontal area.

These studies are in their infancy. Clearly, there are even many different areas of approach still to discover.

Baseball metaphor

I cynically use the following metaphor using a baseball analogy. I am deliberately being farcical because it illustrates well the problems of both methodology, need for detail, the complexities of interpretations and the ostensible double standards. I'm a great admirer of Griffey whose career began in Seattle, so my choosing him in this farce is meant to reflect my admiration for this baseball icon.

Let us suppose, that we want to examine the brain correlates of Ken Griffey Jr's home-run swing. Griffey is appropriately tested with the most sophisticated apparatus around. But he does not hit a home run. Do we conclude that there is no change in his brain during his home run swing? Of course not.

But the skeptics do this with ESP.

Now, we test him again. This time he hits a home run. Our apparatus shows there is some change but we find that this same change occurs during other states that others have exhibited. Do we conclude that he hasn't really hit a home run because others who don't hit home runs also show that change? Of course not. Or do we say we will study a large sample of home runs and compare it with a large sample of non-home run swings in which he made an out? Fair enough. We find Griffey shows no demonstrable change.

Do we conclude that his home run swing does not really exist? After all, we

cannot demonstrate it in the brain. Of course not. We know it exists. We may say that our specific apparatus is not sensitive enough to differentiate his home run swing from his non-home run swing.

But the skeptics do all this with psi.

Now Griffey hits a home run again. This time he exhibits some unique change. The researchers notice it is one specific kind of home run he hits, only the higher ones to the left side. They are proud that they are realizing there is a need to subtype the kind of home run.

This is excellent data. Researchers realize that they need to qualitatively differentiate subtypes just like with research in SPEs.

They conclude that they need to study this specific feature more because how do they know these specific home run findings really are linked with the brain changes? This seems legitimate but time consuming and difficult. But they have millions in funding to do so they go ahead. So they allocate some complex mathematical analyses (e.g. multidimensional scaling, correspondence analyses) to ensure that they understand the differences between the subtypes they have analyzed.

And in fact, I used multidimensional scaling in 22 dimensions to examine the déjà vu phenomenon in great deal and to demonstrate correlations of particular kinds of déjà vu with specific subtypes.[22-24]

They think, "Wow, if we studied psi we might have to subclassify some events as well." But they quickly put that out of their minds because they know psi phenomena don't exist …and if they did, their mention is as politically correct as mentioning the earth was flat …and in any event, if psi existed, they may have to unthink all the thoughts they had had before.

The logic of unthinking much of what one has thought before is not very appealing. On the other hand, we look at the most parsimonious, fruitful explanations and these are often good solid physicalistic explanations. However, we must not be premature in our interpretations and use pseudo parsimony, finding something easy but inappropriate.

Now the researchers want to replicate their study. They examine a new series of 60 Griffey home runs and they find there are, indeed, specific subtypes. The same unique pattern appears in many of them, different from the other home runs and the non-home runs.

Replication is a key in research. The difficulties often relate to exact conditions being replicated. The data may be similar but the hypotheses often have been refined.

Do they now conclude that his home runs are simply an artifact of his brain function? Of course not: They know there is a complex system of neurobiology going on and maybe even a dogged determination which they cannot measure. They realize this is correlative not causal.

But if these were SPEs would they conclude that the brain changes are the causal links for the specific SPE features they have? They look at the research cited on stimulating the brain and producing OBEs, on REM intrusion causing NDEs, on fMRI showing up or not showing up any kind of ESP, and wonder why causality not correlations were emphasized. They also examined the temporal lobe and its links to SPEs and noted the care taken to emphasize correlations not causality.

The researchers would of course not conclude that Griffey's home runs actually do not really exist and are instead imaginings. They know there are independent spectators who saw these home runs and they are not just mass hallucinating them.

Yet, bizarrely, the skeptics do all this with psi, even when statistical data for methodologies achieve unbelievable levels of 1 in 28. 10^{15} against chance as in a specific kind of ESP research, called Ganzfeld. Rejecting all that evidence for their conclusions would be ridiculous as saying Griffey's home runs were not hit, instead of saying "we don't quite know in what way".

Perspective

In the above examples, open minded researchers seek confounding factors to explain any of their unexpected results.

They would perceive the many principles they need to understand when dealing with analyses such as these:

1. Is the apparatus appropriate? Is it able to measure what it purports to be able to measure? If so, can it provide some kind of quantitation? Can this quantitation be adequately controlled?
2. Now let us say that these researchers have studied the remarkable contributions to careful methodology of such phenomenological analyses. They are even more careful in dealing with confounding factors as they realize such information can contaminate their work. They try to understand for example the attitudes to "home run" hitting of our experimenters because they know there may be experimenter effects. They try to control all parts of their research and they know this includes the large population of spectators: So they focus on home games thinking that they may show similar attitudinal aspects or intuitions or determination or motivation or other conduciveness to "home-runs". They realize these may be different from away games (but they preliminarily study this home-away hypothesis first in order to confirm this is true by comparing home and away games). They want to know more about the real brain correlates of the special Griffey home run swing and they know this is a great deal of work.

But they discover it's even more complex. Those same home spectators get fed up when Griffey does not hit home runs and goes into a slump, so they try to control for this factor as far as they can separating the home runs by phase of spectator attitude by doing large randomized attitudinal samples through the year and correlating them with Griffey's individual successes.

By this stage they're realizing that they're in the real world and have to just guesstimate confounding factors hoping many of the large numbers of confounding factors will randomize out between control and experimental groups.

Meanwhile poor Griffey has been allowed only to have the same consistent meals before games and the same workout patterns. Moreover, the experiments become even more detailed. The researchers realize their experimental subject, Griffey, may be manifesting some other state different from the "home run state" so they study his moods and motivation so we can plot that. They ensure the same coach and coaching staff and the same manager and they are distressed when one of them is fired because they wonder how this will impact on poor Griffey. They persuade the team that science is so important that they must not trade any players

because they don't want any variation in their experiment and team chemistry, batting order and several other psychological factors may be diminished. They now realize that perhaps the groundsmen may even be influencing the team, so they arrange with the owner that that staff be kept constant, with the actual field constant. And unfortunately, one of the groundsmen becomes ill and needs to be replaced. They realize here is another confounding factor. Then they realize the weather may be a variable so they decide that if there is a wind above 5 miles per hour in any direction, they will eliminate that data as wind assisted or wind impaired. They also eliminate days it rains as the air density needs to be controlled and keep careful barometric records so their analyses can ensure there are no differences. They keep the study to those days in which the weather is between 50 and 70 degrees F.

But suddenly they realize they have so many controls *they cannot get an adequate sample, so they abandon the whole study or do not control for any of these*. "After all", they argue, "our intent is to publish and who would know the difference anyway? And who really cares that Griffey has this unique manifestation in his brain anyway when he hits a home run?" In fact, they realize that it may be more comfortable showing nothing changed as is their null hypothesis.

The researchers draw a parallel with neuroscience research on intuition and ESP. They realize how complex this all is, and how pat conclusions just are insufficient.

But when one of the researchers dares draw this analogy of this research with research on ESP he is ridiculed. We all know ESP does not exist and even if it did, no reputable journal would dare to publish it. After all, the chances of it occurring are impossible so we will set a P value of less than one in ten million! But *that researcher sticks to his ground and actually notes that meta-analyses and sometimes individual research data in psi, even then are highly significant.*

He points out the limitations of a recent ostensibly sophisticated study out of Harvard, and several others ignoring the phenomenological differences of brain-stimulated out of body experiences and the spontaneous ones. He even tries to throw in the near-death experience as an extra, …and discovers to his distress that one of his co-researchers is so upset, he has a heart attack. This does not get him promoted.

…And so extrasensory perception remains a dirty word.

But back to Griffey. The researchers go in another direction. They are excited by their original results and they want to generalize them to others to make it worthwhile. They consider what broader factors are linked with this research? Have they gone into sufficient detail to ensure like is phenomenologically equivalent to like? For example, how representative is that special pattern of the home run swing of Griffey with that of Bonds, McGwire and Sosa (and they then try to take into account, confounders like non-prescribed substances so they have appropriate extra controls introducing lab test controls)? So they study Bonds, McGwire and Sosa, as well. They use the data they have preliminarily learnt from Griffey, so they're able to hone in on many factors they believe they had controlled for unnecessarily before. They find conflicting information on two of their major independent variables they're studying.

Even on the one finding where there is consistency they have a new conflict:

Do they then take a run-of-the-mill Premier leaguer and study him for seven seasons till he hits 60 "special factor" home runs too? This can extend, and eventually they can study even little leaguers.

Let us imagine that the researchers now find a real consistency. "Special" home run swings are different based on a very specific brain measure from non-home run swings and also from the other home-run swings. The study has now gone on for fifty varied players and is really consistent in its findings.

They publish!!

What can we conclude? Can we say that a certain pattern of brain function allows individuals to hit home runs? No we cannot even say that after all this.

All we can say is that there appears to be a *correlate* of a distinct pattern of brain functioning when individuals hit the ball hard enough to hit a "special" home run. This neither *proves nor denies the causal link of the underlying subjective experience of the hitter or the objective brain experience that the individuals concerned may have when they hit a home run. Indeed, when a home run is hit far more than cerebral cortical mechanisms are involved. A whole, possibly perfectly synchronous sensorimotor loop is involved in the correctly trained individual. That one tiny finding in the brain in this extensive positive study may be simply an epiphenomenon of something far more complex.*

And so the researchers learn about the utter complexity of the real world, and the difficulties of doing such research and the focus they needed to only measure home-runs.

Concluding Perspective

But incidentally, let's just substitute one word here. Let's instead of home runs, apply the same story to ESP. The researchers know the answer here: They dare not have definitive positive results. If they do, they may lose all the rest of their funding for the next year, or their opportunity for tenure, or even their job. It's much safer being a cynic and a skeptic.

The paradox about it is only in the discipline such at this that I call dimensional biopsychophysics would pseudoskeptics dare to write without assiduous examination of the appropriate literature …and succeed with approbation. In what other discipline do second graders have the sheer chutzpah to pontificate?

Arthur Koestler summarized the situation[61]:

'Innovation is a two-fold threat to academic mediocrities: it endangers their oracular authority; and it evokes the deeper fear that their whole laboriously constructed intellectual edifice may collapse.'

Moreover, most scientists apply only the empirical 'physicalistic presupposition' involving the notion that all knowledge has its basis in what is physically perceived, and only physically: therefore, it is of course, deceit and illusion to speak of knowledge based on non-physical perception and therefore, it follows that the paradigm of anything but reductionistic neuroscience is dealing with deceit and illusion[29]. There is however, limited theoretical backing for the physicalistic presupposition and certainly it has no truly empirical support.[29]

References

1. Lehrer, J. Misreading the mind. If neuroscientists want to understand the mystery of consciousness, they'll need new methods., in *Los Angeles Times*. Los Angeles, 2008,

2. Neppe, VM. Subjective paranormal experience. *Psi*. 1980, 2:3, 2-3.

3. Neppe, VM. Psychiatric interpretations of subjective paranormal perception. *Ppsych J of South Africa*. 1982, 3:1, 6-17.

4. Tart, C. A psychophysiological study of out-of-the-body experiences in a selected subject. *International Journal of Parapsychology*. 1967, 9, 251-258.

5. Tart, C. Six studies of out-of-body experiences. *J Near-Death Studies*. 1998, 17:2, 73-99.

6. Tart, C. OBE Email: Private communication to a group of experts, 4 November, 2007,

7. Blanke, O, Ortigue, S, Landis, T, Seeck, M. Stimulating illusory own-body perceptions. *Nature*. 2002, 419:6904, 269-270.

8. Blanke, O, Landis, T, Spinelli, L, Seeck, M. Out-of-body experience and autoscopy of neurological origin. *Brain*. 2004, 127:Pt 2, 243-258.

9. Bunning, S, Blanke, O. The out-of body experience: precipitating factors and neural correlates. *Prog Brain Res*. 2005, 150, 331-350.

10. Blanke, O, Mohr, C. Out-of-body experience, heautoscopy, and autoscopic hallucination of neurological origin Implications for neurocognitive mechanisms of corporeal awareness and self-consciousness. *Brain Res Brain Res Rev*. 2005, 50:1, 184-199.

11. Blanke, O, Mohr, C, Michel, CM, Pascual-Leone, A, Brugger, P, et al. Linking out-of-body experience and self processing to mental own-body imagery at the temporoparietal junction. *J Neurosci*. 2005, 25:3, 550-557.

12. Blanke, O, Arzy, S. The out-of-body experience: disturbed self-processing at the temporo-parietal junction. *Neuroscientist*. 2005, 11:1, 16-24.

13. Tong, F. Out-of-body experiences: from Penfield to present. *Trends Cogn Sci*. 2003, 7:3, 104-106.

14. Booth, JN, Koren, SA, Persinger, MA. Increased feelings of the sensed presence and increased geomagnetic activity at the time of the experience during exposures to transcerebral weak complex magnetic fields. *Int J Neurosci*. 2005, 115:7, 1053-1079.

15. Penfield, W. Functional localization in temporal and deep Sylvian areas. *Research Publications Association for Research in Nervous and Mental Disease*. 1958, 36:1, 210-226.

16. Munro, C, Persinger, MA. Relative right temporal-lobe theta activity correlates with Vingiano's hemispheric quotient and the "sensed presence". *Percept Mot Skills*. 1992, 75:3 Pt 1, 899-903.

17. De Ridder, D, Van Laere, K, Dupont, P, Menovsky, T, Van de Heyning, P. Visualizing out-of-body experience in the brain. *N Engl J Med*. 2007, 357:18, 1829-1833.

18. Greyson, B. Visualizing out-of-body experience in the brain. *New England J. of Medicine*. 2008 (In press),

19. Gabbard, GO, Twemlow, SW. *With the eyes of the mind: An empirical analysis of out-of-body states*. New York: Praeger. 1984.

20. Gabbard, GO, Twemlow, SW, Jones, FC. Differential diagnosis of altered mind/body perception. *Psychiatry*. 1982, 45, 361-369.

21. Neppe, VM. *The Psychology of Déjà Vu: Have I been Here Before?* Johannesburg: Witwatersrand University Press. 1983.

22. Neppe, VM, Funkhouser, ATS-E (eds). *Déjà Vu: A Second Look*. Seattle, WA., Brainvoyage.com:, 2006.

23. Neppe, VM. *Déjà Vu Revisited*. Seattle, WA: Brainvoyage.com. 2006.

24. Neppe, VM, Bradu, D. Déjà vu subtypes: four challenges for researchers, in *Déjà Vu: A Second Look* Edited by Neppe VM, Sub-Ed. Funkhouser AT. Seattle, Brainvoyage.com, 2006, Chapter 6. 52-67.

25. Neppe, VM. Parapsychological approaches to interpreting anomalous brain function and subjective paranormal experience: The out-of-body experience as an example., in *Proceedings, 46th Annual Parapsychological Association Convention.* Edited by Wilson S. Vancouver, B.C., Canada, PA., 2003, 132 – 140.

26. Neppe, VM. "Out-of-body experiences" (OBEs) and brain localisation: A perspective. *Australian Journal of Parapsychology*. 2002, 2:2, 85-96.

27. Alvarado, CS. Out-of-body experiences, in *Varieties of anomalous experience: Examining the scientific evidence* Edited by Cardeña E, Lynn SJ, Krippner S. Washington, DC, Amer Psychol Assn, 2000, 183-218.

28. Plato. *Book VII: Plato's cave*. Boston: Internet Classics Archive MIT Media. Circa 360 BCE.

29. Neppe, VM. Aspects of psychical phenomena. *The Leech*. 1973, 43, 27-35.

30. Morse, M, Castillo, P, Venecia, D, Milstein, J, Tyler, DC. Childhood near-death experiences. *Am J Dis Child*. 1986, 140:11, 1110-1114.

31. Sabom, MB. The near-death experience. *JAMA*. 1980, 244:1, 29-30.

32. Jansen, KL. Neuroscience and the near-death experience: roles for the NMSA-PCP receptor, the sigma receptor and the endopsychosins. *Med Hypotheses*. 1990, 31:1, 25-29.

33. Bonta, IL. Schizophrenia, dissociative anaesthesia and near-death experience; three events meeting at the NMDA receptor. *Med Hypotheses*. 2004, 62:1, 23-28.

34. Neppe, VM. Near-death experiences: A new challenge in temporal lobe phenomenology? Comments on "A neurobiological model for near-death experiences." *Journal of Near Death Studies*. 1989, 7:4, 243-248.

35. Sacks, O. A neurologist's notebook: a bolt from the blue: where do sudden passions come from? *New Yorker*. 2007, 38-42.

36. French, CC. Near-death experiences in cardiac arrest survivors. *Prog Brain Res*. 2005, 150, 351-367.

37. Morse, ML, Neppe, V. Near-death experiences. *Lancet*. 1991, 337:8745, 858.

38. Sabom, MB. *Light and Death*. Grand Rapids, MI: Zondervan. 1998.

39. Nelson, KR, Mattingly, M, Lee, SA, Schmitt, FA. Does the arousal system contribute to near death experience? *Neurology*. 2006, 66:7, 1003-1009.

40. Mignot, E, Lin, L, Finn, L, Lopes, C, Pluff, K, et al. Correlates of sleep-onset REM periods during the Multiple Sleep Latency Test in community adults. *Brain*. 2006, 129:Pt 6, 1609-1623.

41. Planelles, D, Puig, N, Beneto, A, Gomez, E, Rubio, P, et al. HLA-DQA, -DQB and -DRB allele contribution to narcolepsy susceptibility. *Eur J Immunogenet*. 1997, 24:6, 409-421.

42. Douglass, AB, Hays, P, Pazderka, F, Russell, JM. Florid refractory schizophrenias that turn out to be treatable variants of HLA-associated narcolepsy. *J Nerv Ment Dis*. 1991, 179:1, 12-17; discussion 18.

43. James, W. Address of the President before the Society for Psychical Research. *Science: New Series*. 1896, 3, 881–888.

44. Neppe, VM. Anomalies of smell in the subjective paranormal experient. *Psychoenergetics: J. of Psychophysical Systems*. 1983, 5:1, 11-27.

45. Palmer, J, Neppe, VM. Exploratory Analyses of Refined Predictors of Subjective ESP Experiences and Temporal Lobe Dysfunction in a Neuropsychiatric Population. *European J. of Parapsychology*. 2004, 19:1, 44-65.

46. Palmer, J, Neppe, VM. Subjective paranormal experiences and temporal lobe dysfunction in a neuropsychiatric population: analyses of refined predictors. *Journal of Parapsychology*. 2003, 67:1, 75-98.

47. Neppe, VM. *The Psychology of Déjà Vu: Have I been Here Before?* Johannesburg: Witwatersrand University Press. 1983.

48. Neppe, VM. Subjective paranormal experience and temporal lobe symptomatology. *Ppsych J of South Africa*. 1980, 1:2, 78-98.

49. Persinger, MA, Valliant, PM. Temporal lobe signs and reports of subjective paranormal experiences in a normal population: a replication. *Percept Mot Skills*. 1985, 60:3, 903-909.

50. Neppe, VM. Temporal lobe symptomatology in subjective paranormal experients. *J Amer Soc for Psych Research*. 1983, 77:1, 1-30.

51. Saletan, W. Full-Mental Nudity. The arrival of mind-reading machines. 2007.

52. Moulton, ST, Kosslyn, SM. Using Neuroimaging to Resolve the Psi Debate. *Journal of Cognitive Neuroscience*. 2008, 20, 182-192.

53. Lavoie, A. Researchers use neuroimaging to study ESP. Cambridge, Mass., Harvard University, 2008,

54. Radin, D. Abandoning professional peril as taboo (Radin, Science and Taboo). 2008.

55. Radin, D. Blog http://deanradin.blogspot.com/2007/12/japanese-translation-of-entangled-minds.html. January 2008.

56. Standish, LJ, Johnson, LC, Kozak, L, Richards, T. Evidence of correlated functional magnetic resonance imaging signals between distant human brains. *Altern Ther Health Med*. 2003, 9:1, 122-125.

57. Richards, TL, Kozak, L, Johnson, LC, Standish, LJ. Replicable Functional Magnetic Resonance Imaging Evidence of Correlated Brain Signals Between Physically and Sensory Isolated Subjects. *Journal of Alternative and Complementary Medicine*. 2005, 11, 955-963.

58. Achterberg, J, Cooke, K, Richards, T, Standish, LJ, Kozak, L, et al. Evidence for Correlations Between Distant Intentionality and Brain Function in Recipients: A Functional Magnetic Resonance Imaging Analysis. *Journal of Alternative and Complementary Medicine*. 2005, 11, 965-971.

59. Radin, D. Bierman's Brain, in *Entangled Minds*. New York, Paraview, 2006, 176-179.

60. Bierman, D. Anomalous baseline effects in mainstream emotion research using psychophysiological variables, in *Proceedings of the PA, 43rd Convention*, 2000, 34-37.

61. Koestler, A. *The Sleepwalkers*. London: Hutchinson. 1959.

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