Near-Death Experiences: A New Challenge in Temporal Lobe Phenomenology? Comments on "A Neurobiological Model for Near-Death Experiences"

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The excellent framework for a neurobiological model for near-death experiences (NDEs) proposed by Juan C. Saavedra-Aguilar and Juan S. Gómez-Jeria attempts to link anomalous temporal lobe functioning with neuropeptide imbalances, particularly imbalances of the endogenous opioid peptides. Like all pioneering contributions, this paper has drawn on a wide variety of sources to make speculative jumps that are largely justified in the context of present-day knowledge, but will no doubt require numerous alterations in basic perspective over the next two decades.

I was particularly impressed with this neurophysiological NDE model of temporal lobe dysfunction, as it impinges very closely on my own work on anomalistics experiences in the temporal lobe, which has not been cited by these authors. These studies of anomalous experiences and the cerebral cortex have recently been reviewed, and the following is a capsule outline of current research.

First, any link between anomalous experiences and brain functioning requires a detailed description of each event. Not all subjective paranormal experiences may derive from, or be associated with, the

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same anatomical locus, or be predisposed to by the same kinds of psychophysiological conditions or states (Neppe 1984, 1989). This implies the need for the kind of detail outlines by Saavedra-Aguilar and Gómez-Jeria with regard to each specific NDE. Different NDEs may involve different mechanisms, and in fact cluster analyses using detailed phenomenological descriptions may demonstrate that the NDE is a heterogeneous function.

Also, the anatomical or physiological link between NDEs and any pattern of brain functioning does not in any way imply a direct etiologic link. NDEs may reflect genuine patterns of functioning outside the brain, modulated by a particular psychophysiologic mechanism. Alternatively, NDEs may be the epiphenomena of an experience purely within the brain, without any outside reality playing a role.

These comments imply the need for a multiaxial classification system that can unify anomalistic experiences of a variety of kinds relating to subjective paranormal experiences, psychopathological hallucinations, delusional phenomena, illusions, near-death experiences, and temporal lobe symptomatology. The only current classification of such events is the 16-axis Neppe Multiaxial Schema for Anomalous Events, which assists in the analysis of these experiences (Neppe, 1985, 1989). This classification system is reflected in Table 1.

Second, brain functioning may theoretically predispose to anomalous experience in two ways: either globally or focally. The global possibility is exemplified by the hypothesis of endogenous opiates in some way priming the brain, while the focal possibility is exemplified by the hypothesis that temporal lobe functioning is highly relevant. There is good theoretical evidence from the analysis of focal cerebral deficits that the area of focal coordination of anomalous experiences is the temporal lobe (Neppe, 1983c, 1984).

On the other hand, it is almost impossible to test a single hemisphere's functions, as the other hemisphere may compensate for or accentuate anomalous or dysfunctional elements. Consequently, it is dubious at this point whether specificity such as right or left temporal lobe, or right or left cerebral hemisphere, can be achieved.

The only available measure of temporal lobe symptomatology, as opposed to personality structure as reflected by an instrument such as the Bear-Fedio scale (Bear and Fedio, 1977), is the Neppe Temporal Lobe Questionnaire (NTLQ)(Neppe, 1983c). I applied the NTLQ to two contrasting groups, the first subjective paranormal experiencers, the second nonexperiencers. In essence, I was not attempting to prove or disprove individuals' subjective interpretations of anomalous events. Rather, I demonstrated that experiencers have both statistically

Table 1
Neppe's Multiaxial Schema for Anomalous Events

Axis A:	Anomaly level (e.g., subliminal)
Axis B:	Base level (e.g., afferent)
Axis C:	Content (includes perspective and concomitant symptoms)
Axis D:	Dimensions (e.g., retrocognitive, distant)
Axis E:	Ego-consciousness (e.g., meditation)
Axis F:	Forum (e.g., self-referential)
Axis G:	Gestalt (includes special characteristics and physical content)
Axis H:	Heuristic (e.g., waking ESP)
Axis I:	Intention (e.g., spontaneous)
Axis J:	Judgment (includes correspondence and prior knowledge)
Axis K:	Prior knowledge of events
Axis L:	Localization of the focus (e.g., experimenter)
Axis M:	Mental status examination
Axis N:	Neurophysiological correlate
Axis O:	Organizing environment (physical/psychological features of relevance)
Axis P:	Psychiatric diagnosis

and clinically far more possible temporal lobe symptoms than do nonexperiencers.

Most interesting, however, was the association of these symptoms with regard to the anomalistic experiences. They appeared to be related both anatomically and physiologically by virtue of their occurrence together, implying some kind of common temporal lobe state. However, possible temporal lobe symptoms also occurred separately, suggesting that the temporal lobe state may be a trait in people with large numbers of varied anomalistic experiences.

The third interesting finding was the possibility that, despite anomalistic experiences being broadly similar to possible temporal lobe symptoms, they may have a different anatomico-physiological origin. That was suggested by two separate studies involving specific symptoms.

The principal study was on the *deja vu* phenomenon. The *deja vu* phenomenon is particularly interesting in that it occurs in 70 percent of the normal population. It also occurs in patients with temporal lobe

epilepsy and schizophrenia and in subjective paranormal experiencers. I have demonstrated that there are qualitatively distinct nosological kinds of *deja vu* experiences (Neppe, 1983b); and specifically in this context, that *deja vu* in the temporal lobe epileptic is different from that in other epileptics and in patients with nonepileptic temporal lobe dysfunction; and moreover, that this experience is qualitatively quite different from *deja vu* in subjective paranormal experiencers.

These distinctions may imply the need for detailed qualitative analyses of each and every symptom that occurs. This interpretation may have special implications in the context of NDEs: Saavedra-Aguilar and Gómez-Jeria have emphasized the differences between NDEs at transcultural levels, and this association with environmental and cultural factors in no way precludes such experiences deriving from the same kind of focus within the temporal lobe. The consistency of the experience, although it may be pleomorphic in nature, and its episodic qualities suggest an organic link. The caution here, however, is the need for very substantial detail, which the framework of modern computers will allow us to perform.

The second of these specific symptom studies evokes particular interest in NDEs. That study looked at olfactory hallucinations, and found that the smell hallucination experience in subjective paranormal experiencers was phenomenologically distinct from that of temporal lobe epileptics (Neppe 1983a). Temporal lobe epileptics characteristically had unpleasant burning or rotting olfactory experiences. Subjective paranormal experiencers, while they may have that kind of unpleasant experience, usually have pleasant, perfumy, flowery smells.

Particularly interesting is the subjects' reported association of these pleasant smells with presences of what they perceive as dead individuals. The link with near-death experiences in this context is interesting. But even more thought provoking is the difference in quality of olfactory hallucinations: both types technically may derive from the temporal lobe, but pleasant olfactory hallucinations probably occur in less than 10 percent of temporal lobe epileptics (Neppe, 1989).

The hypothesis of a link between NDEs and temporal lobe activity raises the questions of a secondary hypothesis: that NDEs might be more common in temporal lobe epilepsy. NDEs among temporal lobe epileptics have not been sought out actively. Moreover, phenomenological subtyping of subjective paranormal experiences in temporal lobe epileptics is difficult, because these patients are prone to deny the occurrence of such experiences, lest their physicians interpret them as further epileptic symptoms and increase their dose of medication.

This problem has major implications for epidemiologic research in this area, and I leave open at this point the possibility that temporal lobe epileptics may have an increased incidence of subjective paranormal experiences. It is clear, however, that these symptoms respond to anticonvulsant medication when temporal lobe epileptics do indeed report them. At times patients are considerably distressed by the absence of so-called psychic experiences after they are medicated.

A link may be further shown in family studies, as reflected by the analysis of two families with spontaneous paranormal experiences and temporal lobe dysfunction (Hurst and Neppe, 1982). Other researchers, including Peter Fenwick (1983) and Michael Persinger (1989), are actively pursuing related areas, and their work, although not using measuring instruments in as great detail, supports the hypothesis of anomalous temporal lobe experience in this population. It is interesting that Wilder Penfield (1958) reported producing a subjective out-of-body experience by stimulating an area of the temporal lobe in a patient with epilepsy.

Where does the hypothesized psychophysiological opiate link fit in? I am extremely fascinated by this excellent linkage hypothesis, and feel that indeed, parts of the temporal lobe, being embryologically, developmentally, structurally, and functionally dissimilar and more primitive than the other parts of the higher brain, may be more predisposed to changes that may relate at neuropeptide levels (Neppe, 1981a, 1981b, 1981c, 1982).

It is important, however, to remember that neurotransmitter involvements are the biochemical expression of underlying physiologic functions. It is extremely difficult to demonstrate causal, as opposed to coincidental or correlative, relationships. This is particularly so in the context of the paper by Saavedra-Aguilar and Gómez-Jeria, as they present many statements as if they are definitely proven, when in fact the majority of these areas are extremely tentative. Nevertheless, their hypothesis is an outstanding workable model.

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