

Comments on "A Neurobiological Model for Near-Death Experiences"

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Juan C. Saavedra-Aguilar and Juan S. Gómez-Jeria have carefully reviewed some of the anatomic connections of the limbic system, as well as certain aspects of the literature on endogenous opiates and temporal lobe seizures, and have placed this information into a framework to explain near-death phenomena. It is their opinion that during life-threatening situations, brain stress leads to hypoxia, limbic seizure discharges, and endogenous opiate release, which produce, in turn, subjective sensations that upon recovery are put into language and reported as near-death experiences (NDEs). Although they regard their concepts as a "model" for this symptomatology, the term "hypothesis" would seem to be more appropriate because "model" implies a replication of the phenomena.

Since I have published a paper on this topic ten years ago (Rodin, 1980), I experienced a considerable degree of *deja vu*, presumably without accompanying seizure discharges in the temporal lobe, on reviewing this manuscript. Endogenous opiates were not yet fashionable at that time, but the effects of hypoxia coupled with a rise in carbon dioxide and nitrogen certainly were known, and these events are bound to occur during the dying process. On the other hand, it must be remembered that the mechanisms and resulting mental phenomena accompanying true and final death need not be the same as those occurring during "near-death" situations.

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The essence of the manuscript under discussion is that the authors are impressed with the similarity between NDE reports and temporal lobe seizure symptomatology. There are, however, several points the epileptologist needs to address lest the nonspecialist reader accept the model as closely resembling the truth of the situation. The hallmarks and nuclear components of NDEs are a sensation of peace or even bliss, the knowledge of having died, and, as a result, being no longer limited by the physical body. In spite of having seen hundreds of patients with temporal lobe seizures during three decades of professional life, I have never come across that symptomatology as part of a seizure.

Furthermore, electrical stimulation of neocortical structures (Penfield and Jasper, 1954) or of the amygdala or hippocampus (Gloor, Olivier, Quesney, Andermann, and Horowitz, 1982) also failed to induce those symptoms. On the contrary, stimulation of the amygdala, when accompanied by a change in emotional tone, led to a sensation of fear and never of pleasure (Gloor, Olivier, Quesney, Andermann, and Horowitz, 1982). There is one subjective report in the lay literature by Fyodor Dostoyevsky (1977, p. 258) of a feeling of bliss at the onset of a generalized seizure, but this is exceedingly uncommon in actual clinical practice.

The authors also suggest that the subjective sensation of a bright light is due to a discharge having propagated to the occipital areas. This assumption seems to be based on Kenneth Ring's five stages of NDEs, in which the light comes in relatively late (Ring, 1980), but there are reasons to believe that this may not be correct. Occipital lobe discharges produce crude visual phenomena of a flashing or zigzag-fortress nature that migraine sufferers are clearly familiar with; they do not produce a steady bright light to which the experiencer feels himself drawn.

The bright light is actually relatively infrequent during NDEs, reported by only 28% of Michael Sabom's cases (Sabom, 1982) and 16% of Ring's (1980). On the other hand, according to the Buddhist Chikhai Bardo, the experience of meeting the primordial light and abiding in it is the only true way to everlasting bliss at the moment of genuine death of the individual (Evans-Wentz, 1960). To miss that opportunity by lack of will power only leads to karmic illusions and eventual rebirth. Thus, instead of being the last event it should be the first, but since we have not yet faced ultimate death, at least in this incarnation, we have to admit to a lack of information that unfortunately is irremedial. I mention this only to point out the differences between NDEs and death experiences as related by Eastern mystics.

Saavedra-Aguilar and Gómez-Jeria also feel that phenomenological differences between NDEs and temporal lobe epilepsy may be due to the fact that temporal lobe epilepsy patients have mesial temporal sclerosis that would produce a different propagation pattern of seizure discharges than would occur in normal individuals. It needs to be pointed out that mesial temporal sclerosis is not invariably present in patients with temporal lobe epilepsy, and other patients who are found to have the characteristic changes of mesial temporal sclerosis on autopsy may never have had a temporal lobe seizure. Furthermore, there is no reason to believe that temporal-limbic dysfunction has to be accompanied by seizure discharges.

It is reasonable to assume that moderate stress and brain trauma liberate endogenous peptides, but an attempt to explain near-death phenomena on that basis appears rather tenuous. Specifically, Dennis Kelly's work, which the authors referred to, is quite peripheral to the question at hand, and a typical example of the leap of faith Saavedra-Aguilar and Gómez-Jeria have taken from scientific observation to philosophical speculation. Kelly (1982) reported on a series of rats who swam for 3.5 minutes in cold water and were subsequently examined in regard to the tail-flick and flinch-jump reflex, as well as a liminal escape test, which is not a reflex but requires volition. The study was based on a total of six rats. Volitional activity, that is, liminal escape threshold, returned to baseline by 70 minutes after the swim, while the reflex action thresholds remained elevated for three hours or more. These results were presented to demonstrate that duration of analgesia after a cold stressor depends on the type of test used. Whether or not the rats hallucinated or had an NDE is, of course, unknown.

Possibly more important for Saavedra-Aguilar and Gómez-Jeria's thesis is the point Kelly made in that same paper that the analgesic effect and the hormonal stress response were unrelated. Cold water swimming produced analgesia, as mentioned, and also elevation of three stress hormones: adrenocorticotrophic hormone (ACTH), cortisone, and β -endorphins. But when the rats swam in warm water, they showed the same hormonal response without experiencing analgesia. Kelly also pointed out that "the maximal β -endorphin responses induced by the most extreme stress (limb fracture) is manyfold lower than levels required to induce analgesia via systemic injections" (1982, p. 265).

In a similar vein, the paper by Crawford Clark, Joseph Yang, and Malvin Janal investigating prolactin, β -endorphin, ACTH, and growth hormone in three marathon runners reported increases in levels but

“neither of the pre-run pain measures nor post-run change scores correlated with either initial levels or post-run changes in levels of any of the plasma endocrines” (Clark, Yang, and Janal, 1986, p. 119). Although the euphoria and joy scales showed an increase in positive affect 30–40 minutes after the run—and that increase did not occur when the subjects had been given naloxone, an opiate inhibitor, previously—this was likewise not correlated with any of the plasma levels mentioned above.

The statement that decreased acetylcholine levels in the brain lead to such a variety of symptoms as hallucinations, delirium, dreams, amnesia, and analgesia is also open to doubt. The most common clinical condition in which decreased cerebral acetylcholine levels appear to be the prime etiology is Alzheimer’s dementia, and the main initial symptoms consist of memory loss and impaired judgment rather than a florid psychotic picture. As far as anoxia is concerned, it is of course known that the hippocampus is highly vulnerable, but so are the Purkinje cells of the cerebellum. Since they have a predominantly inhibitory function, their loss could be just as important as the hippocampal effects.

The authors note that the “model tries to answer the fundamental question of whether neurosciences and psychiatry are reconcilable.” In view of the fundamental gaps in our knowledge about the physical basis of mental processes, I am inclined to think that, although the roads are converging, the final fusion is still well in the distant future. Neurophysiology and neurochemistry can give isolated insights, but the demonstration of how neuronal firing patterns are transmuted into a thought or feeling is beyond our capabilities at this time. Until we know what a thought is and can produce one at will, until we know not the concomitants but the mechanisms of a dream and can produce it at will, we are reduced to speculation; and there appears to be a general truth that the less we know, the more we theorize and argue.

It is nice to build models, theories, and hypotheses, but to have utility they should be subjected to experimental verification, and that cannot be done in the present instance. We do not know what the neuropeptide changes in the brain are during NDEs, and we will not know in the foreseeable future. The brain has protected itself very well, and we simply cannot stick probes into numerous brain areas to take tissue samples from dying individuals, or from those who are in the process of resuscitation. Even magnetic resonance imaging (MRI) spectroscopy is still in its infancy, and could not be applied to the question at hand.

We must remember also that whatever goes on in the blood in regard to ACTH or endogenous opiates may bear very little relation to what happens in discrete areas of the brain. From a rat, swimming in ice water and having a degree of analgesia thereafter that may or may not be accompanied by a rise in endorphins, to heavenly bliss during an NDE is a rather large jump. No degree of model building will overcome that gap at this time.

Thus the most fitting conclusion would seem to be what Saavedra-Aguilar and Gómez-Jeria stated in regard to the verbal system that is undoubtedly at work, not only in reporting NDEs, but also in the writing of scientific articles: "One fundamental function of the verbal system appears to be the construction of logical, coherent, and explicative hypotheses based on the functioning of other cognitive and emotional systems. These hypotheses can sometimes be completely erroneous, but they are taken as absolute truth."

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