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

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The neurobiological model for near-death experiences (NDEs) put forward by Juan C. Saavedra-Aguilar and Juan S. Gómez-Jeria must surely come very near to being the most comprehensive, coherent, and up-to-date model so far available. Its five-fold basis in temporal lobe dysfunction, hypoxia/ischemia, stress, neuropeptide/neurotransmitter imbalance, and the overall role of the language system, which emphasizes how the later verbal reconstruction of the actual NDE is made to fit in with the personal beliefs and background of the individual, is certainly comprehensive enough to cover most aspects of the underlying physiological processes. The model restricts itself to the neurobiological level of explanation and, although it cannot escape from mentioning its psychological correlates, it refrains, probably wisely, from speculating at what locus in the model a paranormal psychological content might fit.

In their discussion of temporal lobe dysfunction, a most useful concept, the authors' summary of the phenomenology of temporal lobe epilepsy (TLE) is also comprehensive and relevant. Much clinical knowledge has accumulated about TLE since the seminal writings of J. Hughlings Jackson in the 1880s on this strange kind of epilepsy, which Jackson made as much his own as the "Jacksonian epilepsy" that took his name. Over the same period, however, some of Jackson's meticulous observations and formulations, which may be of relevance to the NDE, have tended to be forgotten. To mention two of these not

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discussed by Saavedra-Aguilar and Gómez-Jeria is not to criticize, but to point to the possibilities of expansion of their model, which is such a commendable feature of it.

One example I have in mind is Jackson's distinction, in discussing the hallucinatory manifestations of TLE, between "crude sensations" and "formed sensations," both of which occur. In this distinction, a "crude sensation" in the visual system would be a flash, or balls of fire, or colored lights, while a spectral face would be a "formed sensation." In their paper, Saavedra-Aguilar and Gómez-Jeria refer to the noises reported in the early stages of the NDE, and favor attributing these to discharges in the cells of the organ of Corti, resulting from a decreased blood flow there. But such noises could also be "crude sensations" resulting from discharges in the superior gyrus of the temporal lobe, where sound is represented linearly, according to pitch, after the manner of a piano keyboard. Similarly, the "hush" that is sometimes experienced in TLE and due, presumably, to transitory suppression of activity in that auditory area, may also occur in the NDE and thus play a part in the feelings of peace sometimes reported.

Another feature of TLE of possible relevance to NDEs, and which might be added to the authors' discussion of amnesia, is the occurrence of the "dysmnesic syndrome" sometimes lasting for only a brief interval, but which I have observed to continue for more than a week. In this state, the duration of the capacity for recall is cut down to less than a minute but, as the patient is otherwise in touch with the immediate environment and usually behaves in a manner that appears deceptively normal to the unsophisticated observer, the defect may only be brought out if specific tests are made. If such a dysmnesic state, as distinct from amnesia, should occur even briefly in a near-death setting, it could be of considerable relevance to moment-to-moment forgetting of the nearness of death, and to a calmness of mind in a person who, again to the unsophisticated observer, may appear to be in full possession of his or her faculties.

The authors correctly relate the impressive variability of the phenomenology of TLE to the complex anatomical connections of the temporal lobes and the limbic system, and to the equally complex biochemistry of their functional relationships. Here the baffling network of the anatomical connections that the authors so expertly describe could have perhaps been made still more clear by a diagram.

On the biochemical aspects of the authors' model, as distinct from the clinical aspects, I am less competent to comment. However, in their discussion of hypoxia/ischemia, the authors seem to deal well with the various possible mechanisms whereby this may bring about limbic

discharges, and with the evidence of the special sensitivity of the hippocampus to oxygen deprivation. They also seem to show well how the balance between excitatory and inhibitory synapses is altered during hypoxia, as well as how a number of other subtle mechanisms of relevance to NDEs may become disordered. Similarly, in discussing neuropeptide/neurotransmitter imbalance, they muster convincing evidence that during moderate "brain stress," a term whose meaning is not precisely clear, and brain trauma, there is a variety of different biochemical disturbances, including the liberation of endogenous peptides, that could lead to abnormal limbic discharges.

In conclusion, I have the impression that rather too many writers about NDEs and about out-of-body experiences have written in rather too obvious ignorance of the basic relevant clinical knowledge that has been accumulating in the fields of general medicine, neurology, and psychiatry since the later decades of the 19th century. One consequence has been that they have not had enough ballast, as it were, to take on board the further impressive developments that the neurosciences have undergone in the last decade. Saavedra-Aguilar, however, with his sound clinical training, and Gómez-Jeria, with his equally sound laboratory training, working together have had sufficient ballast to do just that. They say modestly that their neurobiological model for NDEs could be seen as a complement to other explanatory domains, but I think that, in some important respects, it may seriously challenge the speculations at other levels of explanation of some of these writers.