Near-Death Experiences: A Neurophysiologic Explanatory Model

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ABSTRACT: Prior hypotheses as to the etiology of near-death experiences (NDEs) have been limited to psychiatric explanations or brief discussions of endorphins as causative agents. We present a neurophysiological explanation for NDEs based on their similarities with lysergic acid-, ketamine-, and hypercapnia-induced hallucinations. We believe the core NDE is genetically imprinted and triggered by serotonergic mechanisms.

Near-death experiences (NDEs) are profound transcendental events experienced on the threshold of death. They have been reported by people who have been seriously injured or ill but recovered (Greyson & Stevenson, 1980), and by people who have had anticipated imminent death in a potentially fatal situation but escaped unharmed (Comer, Madow, & Dixon, 1967). They have been documented as occurring primarily in survivors of cardiac arrests and profound comas, as opposed to seriously ill patients who are treated with mechanical ventilation, narcotics, and anesthetic agents, but who are not critically ill and at risk of dying (Morse, Castillo, Venecia, Milstein, & Tyler, 1986).

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Such NDEs commonly include a positive affect, a conviction that one did indeed die, an impression of being outside the physical body, an impression of passing through a dark enclosed space, apparent extrasensory phenomena, apparent encounters with persons not physically present, an impression of entering some unearthly realm, and a review of past life events (Moody, 1976; Sabom, 1982; Green & Friedman, 1983).

For example, a 6-year-old male was admitted to Children's Orthopedic Hospital for a tonsillectomy. There was no significant past medical history. After induction with halothane, there was a cardiac arrest requiring 3 min of chest compressions. While in the recovery room, he had a second and third cardiopulmonary arrest necessitating cardioversion and cardiac medications. Later the same day, he was described as being alert, eating, and with no apparent complications.

When interviewed 2 years later, he had very clear memories of his experience while comatose. He had never confided them to his mother, but had told a grandparent about his experience 1 year earlier. He stated that he had left his body and hovered above the operating table. He said he had only hazy images of the doctors, but noted, "I had a tube in my mouth."

His next memory was that of traveling in a long tunnel that was lined with brightly colored lights of every hue, similar to "airplane landing lights." He felt very peaceful. He specifically stated that he did not know where the tunnel went, why he was in it, how he came to return to his body, or any other details. His experience embodies the core NDE, as described by Kenneth Ring (1980).

The medical literature includes many psychological analyses of NDEs (Greyson, 1983; Noyes, 1979; Enhrewald, 1974), but to our knowledge few neurophysiological models.

It has been proposed (Carr, 1982) that NDEs are the result of stimulation of the hippocampus by endogenous endorphins. The stresses of dying certainly generate "natural opiates," such as enkephalins and endorphins, and enkephalins have been documented to inhibit neuronal discharge within the hippocampus. This theory proposes that increased endorphin levels disinhibit the hippocampus, as well as lowering the seizure threshold within the temporal lobe, and that NDEs are the result of limbic lobe and temporal lobe seizures.

One problem with this hypothesis is that patients with narcotic overdoses or narcotic-induced hallucinations do not report the characteristic features of NDEs; in particular, they do not report vivid out-of-body experiences (OBEs). Our study of NDEs used a control group treated with narcotics, as well as being hypoxic and hypercapnic, and

none of those patients had NDEs (Morse, Castillo, Venecia, Milstein, & Tyler, 1986).

We are proposing a similar model, but based on serotonin rather than opioid peptides. The NDE may be the result of activation of neuronal connections in the temporal lobes that code for NDE-like memories. Activation of these neuronal connections is mediated via serotonergic pathways. Alteration of central serotonergic activity through extreme emotional or physiological stress, or via certain psychoactive drugs, could lead to activation of these neuronal connections resulting in an NDE. Imbalances in monoamine transmitter systems at the level of the hippocampus would lead to disinhibition of "hard wired" neurons with axons to the temporal lobe, leading to activation of areas of the temporal lobe that have been documented to cause mystical visions, out-of-body sensations, panoramic memories, and vivid hallucinations.

Support for this model begins with the work of Wilder Penfield, who identified areas of the temporal lobe associated with psychical hallucinations, memories, heavenly music, and religious visions through direct electrical cortical stimulation during neurosurgical procedures. For example, a 33-year-old man suffered from temporal lobe seizures that included vertigo and autoscopic hallucinations. On stimulation at a depth of 2 cm along the superior surface of the temporal lobe within the fissure of Sylvius, the patient exclaimed, "Oh God, I am leaving my body" (Penfield, 1955; see Figure 1).

Another patient whose seizures were never psychical reported that when points 0, 1, and 3 were stimulated, she "had a queer sensation as if I am not here. As though I were half and half here." Many of the elements of NDEs described by Raymond Moody (1975) are localized to the temporal lobe by electrical stimulation experiments. Penfield concluded that when complex hallucinations are induced by stimulation of the temporal cortex, they are perceived as memories by the patients (Penfield & Rasmussen, 1950).

The sensation of floating or otherwise being out of the physical body is the most commonly reported element of NDEs (Greyson & Stevenson, 1980; Moody, 1975; Sabom, 1982; Green & Friedman, 1983; Ring, 1980; Morse, Castillo, Venecia, Milstein, & Tyler, 1986). Ring (1980) wrote that there is a sequence of events in the NDE, and he listed leaving the body as stage 2 of 5, following feelings of peace. Although no precise definition of NDEs is commonly agreed upon, most researchers describe the experience as beginning with an OBE.

Furthermore, while Glen Gabbard and Stuart Twemlow (1984) were able to identify differences between OBEs and other diverse ex-

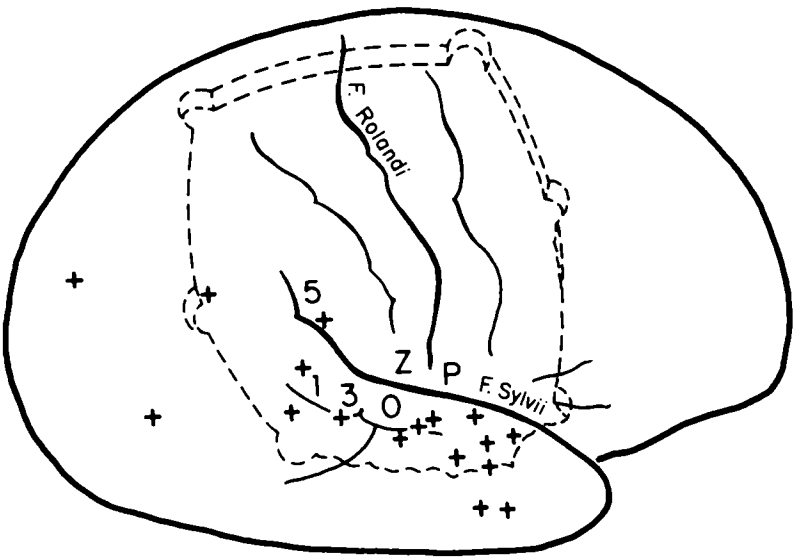

Figure 1. Numbers and letters indicate areas that produced the sensation of leaving the physical body, by direct electrical stimulation. "+" indicates areas that, when stimulated, created related phychical events, such as hearing heavenly music and other hallucinations. Reprinted from Penfield and Rasmussen (1950), with permission.

periences, such as schizophrenia, hypnagogic states, depersonalization, vivid dreams, daydreams, flying dreams, and autoscopic hallucinations, they could not conclude "that NDEs are characterized by exclusive features that are not present in other forms of OBE" (1984, p. 138).

OBEs and NDEs do not occur only in near-fatal situations. Ring (1980) developed a Weighted Core Experience Index to analyze the subjective accounts of persons reporting psychological experiences. When we applied this scale retrospectively to five case histories of patients treated with LSD-25 (Grof & Halifax, 1977), three scored as having moderate NDEs, and two as having deep NDEs.

Similarly, the anesthetic agent ketamine also produces perceptions that typically accompany NDEs, such as leaving the physical body and meeting beings with higher powers (Collier, 1972; Rogo, 1984). Finally, early work by Laszlo von Meduna (1950), using inhaled carbon dioxide as a psychotherapeutic agent, demonstrated that all the reported elements of NDEs can be reproduced in the office setting.

It is well established that the hippocampus, amygdala, and related limbic structures play a central role in processing and distributing neocortical information. This area of the brain is responsible for coordinating and processing perceptions, memories, emotions, early development, dreams, and the massive waking activities of the neocortex (Winson, 1986). This is the area described by Penfield (1975) as "the man in the machine." It is directly linked to the temporal lobe, which explains why temporal lobe seizures can result in activation of complex activities, such as walking, eating, or riding a bus to distant cities, whereas other brain seizures result in disorganized motor movements or complete loss of function. Temporal lobe seizures are transmitted to the hippocampus and related structures, which then trigger purposeful motor movements in the neocortex.

Monoamine transmitter systems, especially serotonin, have now been documented to have neurons in the brainstem with axons throughout the brain. The hippocampus is an important gateway for these neurotransmitters, and these monoamines play an important part in the limbic system's role as the central processing unit in the brain (Winson & Abzug, 1977, 1978). As we will show, the documented agents that cause OBEs and NDEs act to create imbalances in serotonin and other monoamines.

The effects of LSD-25 have been directly attributed to alterations in the functioning of central monoamine neurotransmitters, especially serotonin (White & Appel, 1982). Serotonergic cell bodies have been detected in the midbrain dorsal raphe nucleus, axons of which innervate forebrain and temporal lobe areas (Strahlendorf, Goldstein, Rossi, & Malseed, 1982). Reduction of raphe-mediated forebrain and temporal lobe inhibition, via LSD-induced depression of raphe activity, could result in the disinhibition of raphe target neurons in the temporal lobes, resulting in an OBE or other related psychical phenomena that have been electrocortically mapped in the same area.

The mechanisms of action of ketamine have not been as clearly defined. It is known that ketamine affects central nervous system neurotransmitter systems, and brain acetylcholine turnover has also been found to be reduced in the rat caudate nucleus and hippocampus during ketamine anesthesia (Domino, Chodoff, & Corssen, 1965). It could be speculated that ketamine may also act like LSD on serotonin turnover, resulting in a similar disinhibition of raphe target neurons.

Serotonin has been implicated in a wide variety of mammalian physiologic behaviors, including sleeping–waking cycles, pain perception, and cardiovascular control. An example of its modulatory action was demonstrated in studies of stress-induced antinociception (SIA).

SIA is an intriguing phenomenon in which exposure to stressful events decreases an animal's response to painful stimuli. In this study, norepinephrine, dopamine, and serotonin were altered in the brain, and SIA measured. Only changes in serotonin affected responses to pain perception (Snow, Tucker, & Dewey, 1982), supporting our hypothesis that responses to stress involve serotonergic systems. It is by this mechanism that stress-related NDEs, such as those experienced by mountain climbers who have survived falls or coal miners trapped in mines, may be explained.

The only other agent we know of that has been clinically documented to create NDE-like experiences is carbon dioxide (Meduna, 1950). Hypercapnia could result in secondary changes in central serotonin activity, or could produce epileptiform activity in the temporal lobe, resulting in the direct discharge of neuronal connections coding for NDE-like memories. The only clinical study comparing carbon dioxide levels in patients with or without NDEs documented that both groups had similar carbon dioxide levels in the bloodstream (Morse, Connor, & Tyler, 1985).

We searched the medical literature without success for other agents or psychological processes that could cause NDEs or OBEs. For example, studies of hypoxia, other hallucinogens, psychotic conditions, or intensive care unit stress syndromes do not produce NDEs. One study described a complex hallucination in a patient on narcotics, reversed by naloxone (Judson & Wiltshaw, 1983). Careful review of that patient's experience revealed that it lacked most of the descriptive elements of NDEs, including an OBE, and that the patient was semiconscious during the experience. Although that case has been cited as clinical evidence that perhaps endorphin systems are involved with NDEs, it is unlikely that that patient had an NDE. Naloxone, which the patient was treated with, has been well documented to cause very similar hallucinations, including a sense of soullessness and paranoid ideation. These elements, which are seen in drug-induced hallucinations, are not seen in NDEs (Cahal, 1957).

Our neurophysiological model for near-death experiences is presented in Figure 2. There is clear evidence that within the temporal lobe are neuronal connections that, when electrically stimulated, produce OBEs. This is the most common element in NDEs, and is the first element cited by most researchers. The temporal lobe is connected by serotonergic neurons to the midbrain dorsal raphe, and especially the hippocampus. The hippocampus is the central processing area of the brain, and is the area of the brain most associated with a sense of consciousness or soul. Psychological stresses and psychoactive agents

have a neurochemical effect in this region of the brain, mediated by serotonin.

Our model hypothesizes that near-fatal events, psychoactive agents, or stress could trigger OBEs and psychical hallucinations by disinhibiting target axons in the temporal lobe, from the level of the hippocampus. Hypercapnia, which would be present in any near-fatal event, even if only on the cellular level, could act either through the hippocampus or by directly stimulating the temporal lobe.

Although much of our work is speculative, it is well documented that neuronal connections specific for creating out-of-body states exist. Such genetically determined areas in our brains may well serve as a natural defense mechanism against stressful situations, such as childbirth or trauma, both of which have been reported to cause OBEs. However, it is just as likely that such an area represents the seat of the soul, the area of our brain that serves as a trigger point for the release of the soul at death. Such an area could serve equally well for stresses during life. This would also explain how certain religions use control of the autonomic nervous system, which is integrally enmeshed with the

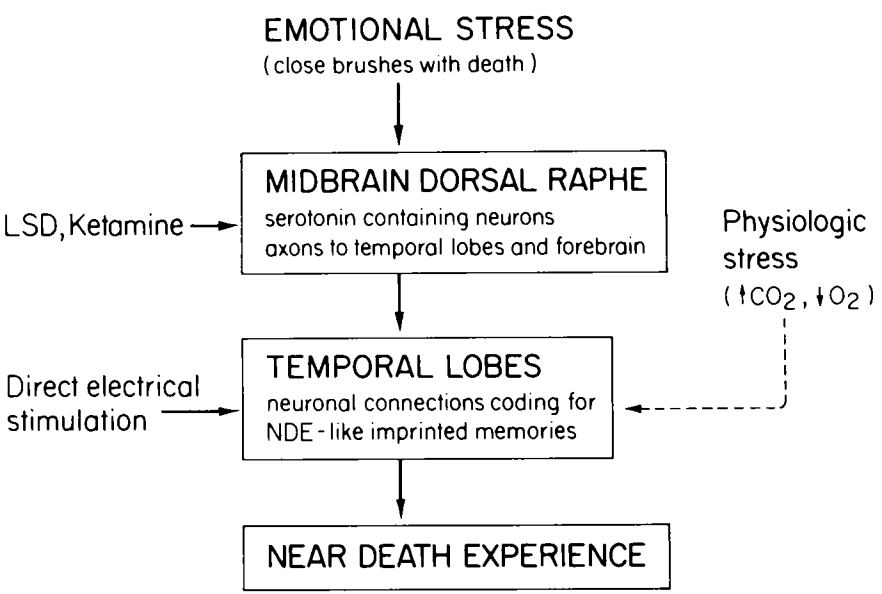

Figure 2. A cohesive model linking all known causative agents of NDEs and OBEs.

hippocampus and limbic structures, to produce out-of-body states and religious ecstasy. This area of our brain is the first genetically imprinted correlate to a Jungian archetype that we have seen described, and we hope it will serve as a bridge between psychological and neurochemical descriptions of brain activity.

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