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Bruce Greyson and Marieta Pehlivanova

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A Neuroscientific Model of Near-Death Experiences Reconsidered

Bruce Greyson and Marieta Pehlivanova

Department of Psychiatry and Neurobehavioral Sciences, Division of Perceptual Studies,
University of Virginia School of Medicine

A recent review article by a multinational team of researchers made a significant contribution to the literature on near-death experiences (NDEs) by collating the evidence supporting diverse physiological explanatory hypotheses for these experiences into one overarching theory (Martial et al., 2025). Given the variety of models proposed over the decades, combining these disparate neurophysiological and psychological hypotheses into a comprehensive model is an admirable effort. However, the authors acknowledged that their multifactorial mechanism for NDEs left some questions unanswered and did not cover all key features of NDEs. We cite important NDE elements that remain unexplained by that model, as well as empirical data that are incompatible with it. We respectfully suggest that, taken together, these points generate sufficient doubts to merit reconsideration of the completeness of that overarching model and suggest that a more comprehensive treatment of the literature would strengthen our understanding of NDEs and their etiology.


Keywords: near-death experiences, neurophysiological models, death-related phenomena, mind–body relationship, consciousness

Near-death experiences (NDEs) are profound, subjective experiences that can occur when an individual is physiologically—or sometimes psychologically—close to death, including in instances of clinical death followed by resuscitation (Holden et al., 2009; van Lommel et al., 2001). Often described as mystical or spiritual, these experiences may involve a sense of unity with the universe and others, ego dissolution, a sense of disembodiment, and encounters with a transcendent reality (Greyson, 1983). NDEs often permanently and dramatically alter the experiencers' attitudes, beliefs, and values (Noyes et al., 2009). Aftereffects most often reported include increases

in spiritual attitudes and interests, concern for others, and sense of purpose or meaning in life, as well as decreases in fear of death, materialism, and competitiveness (Noyes, 1980; Noyes et al., 2009; van Lommel et al., 2001).

Lack of awareness and understanding of NDEs within healthcare settings has led some near-death experiencers to prolonged distress and inhibited them from sharing their experiences (Duffy & Olson, 2007; Greyson, 1997, 2010b; Pehlivanova et al., 2025; Samoilov & Corcoran, 2020). Beyond their impact on experiencers, NDEs continue to intrigue scientists and academics, raising questions about our understanding of the mind and brain and

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Bruce Greyson  <https://orcid.org/0000-0002-2192-7898>

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Correspondence concerning this article should be addressed to Bruce Greyson, Department of Psychiatry and Neurobehavioral Sciences, Division of Perceptual Studies, University of Virginia School of Medicine, P.O. Box 800152, Charlottesville, VA 22908, United States. Email: cbg4d@uvahealth.org

suggesting that these and comparable phenomena require a reconsideration of our current neurophysiological models (Greyson, 2010b; Greyson et al., 2009). These clinical and theoretical issues highlight the importance of understanding the etiology and mechanisms of NDEs.

A recent review article in *Nature Reviews Neurology* by a multinational team of researchers attempted to pull together a variety of explanatory hypotheses into one coherent overarching theory to explain NDEs (Martial et al., 2025). That review article provided a new and helpful synthesis of existing data into one coherent model, which they called the Neurophysiological Evolutionary Psychological Theory Understanding Near-Death Experience (NEPTUNE). Combining these disparate neurophysiological and psychological hypotheses into a comprehensive model is an admirable strategy, particularly if the component parts are supported by adequate empirical bases. In this article, we respectfully suggest that there are additional empirical data from NDE research that counsel reconsideration of the comprehensiveness of the NEPTUNE model, including studies that contradict its premises and important NDE features that remain unexplained.

Components of the NEPTUNE Model

Changes in Brain Blood Gases

The first explanatory hypothesis Martial et al. (2025) discussed involved the role of altered blood gases in producing NDEs. They cited several studies suggesting that decreased oxygen (Annen et al., 2021; Lempert et al., 1994a, 1994b; Martial, Piarulli, et al., 2024; Whinnery & Whinnery, 1990) and increased carbon dioxide (Klemenc-Ketis et al., 2010) can produce some phenomena seen in NDEs, but studies of blood gases in people who report NDEs generally do not show hypoxia or hypercarbia. In fact, studies with large samples of near-death experiencers actually report increased or comparable oxygen and decreased or comparable carbon dioxide levels in near-death experiencers compared with comparison patients (e.g., Parnia et al., 2001; Sabom, 1982). Moreover, increased carbon dioxide indicates better cardiac output and perfusion pressure, which reduces postcardiac arrest amnesia, facilitating *recall* of NDEs, but not necessarily *occurrence* of NDEs (e.g., Greyson, 2010a).

Involvement of Temporoparietal Junction and Temporal Lobe

In regard to temporoparietal junction (TPJ) activation, Martial et al. (2025) cited two studies to support the claim that activation of the TPJ may be responsible for out-of-body experiences (OBEs; De Ridder et al., 2007; Arzy, Thut, et al., 2006). However, the purported OBEs in these two studies are quite unlike the OBEs reported in many NDEs: the center of consciousness remains inside the physical body in TPJ activation, and perception of the environment is from the normal visual perspective of inside the physical body, rather than from outside the body as in NDEs; there is a sense of disembodiment without actually seeing a disembodied image, or, if seen, the disembodied image is static and does not move, whereas in NDEs the disembodied center of consciousness moves about independently of the physical body; and patients having TPJ activation perceive the event as illusory, whereas patients with NDEs perceive the event as profoundly real (e.g., Greyson et al., 2008).

Electrical stimulation studies have produced not OBEs but rather optical illusions in which experiencers do not believe themselves to have left their bodies. Despite these experiences being labeled by some researchers as OBEs, they are not labeled as such by the experiencers. For example, in a recent report of the purported induction of an OBE by brain stimulation, the single subject reported only a sensation of “her body falling/drifted side wards and even out of the chair” (Schutter et al., 2006, p. 240). Electrical stimulation may produce a sense of perceiving things visible from the physical position of the individual’s eyes, but those perceptions disappear when the eyes are closed or the person loses consciousness. There is no evidence that electrical brain stimulation has ever produced accurate perception of anything not visible to the physical eyes, or that persists when the eyes are closed, or that is from an out-of-body perspective—all features observed in spontaneous OBEs (Geisler-Petersen, 2008; Holden et al., 2006).

Martial et al. (2025) also cited a study (Arzy, Seeck, et al., 2006) in which electrical stimulation of the TPJ “can trigger an own-body illusion of another person in the extrapersonal space, resembling the encounter of entities reported in NDEs” (Martial et al., 2025, p. 8). In fact, the illusion induced by such stimulation is nothing

like the visions of deceased persons reported in NDEs: in the single patient in the study by Arzy Seeck, et al. (2006) in whom this illusion was induced, “the illusory person closely ‘shadowed’ changes in the patient’s body position and posture” (p. 287). The illusory person was not seen or identified but was rather “the impression that somebody was behind her ... a ‘shadow’ who did not speak or move” (p. 287). This is not remotely comparable to the visions reported in many NDEs of identified deceased persons who are seen, heard, smelled, and touched, and interact meaningfully, conveying information to the experiencer (E. W. Kelly, 2001).

A more problematic distinction between electrically induced bodily illusions and spontaneous OBEs that challenges the claim that they are comparable is that some OBEs include corroborated reports of perception of events at a distance. In many of these cases, the events in question included unlikely and unexpected details that had been verified as having occurred, and the experience had been reported to someone else before that verification occurred (Holden, 2009; Parnia et al., 2014; Ring & Lawrence, 1993; Rivas & Smit, 2013; Rivas et al., 2023; Sabom, 1981, 1982, 1998; Sartori, 2008; Sartori et al., 2006; Smit, 2008; van Lommel et al., 2001).

Martial et al. (2025) claimed that multiple lines of evidence indicate that the temporal lobe is involved in the generation of NDEs, including electrical stimulation studies of the temporal lobe and observations of patients with temporal lobe seizures. In fact, most of that evidence suggests that experiences induced by TPJ activation do not involve coherent NDE-like experiences but rather fragmentary bits of music or singing, seeing isolated and repetitive scenes that seemed familiar, hearing voices, experiencing fear or other negative emotions, or seeing bizarre imagery that was often described as dream like, distorted experiences quite unlike NDEs (Gloor, 1990; Gloor et al., 1982; Penfield, 1955; Penfield & Perot, 1963, pp. 611–665). Neuropsychiatrist Peter Fenwick concluded that

abnormal discharges in the temporal lobe may produce confusional fragments of phenomena sometimes seen in NDEs. ... This is a very long way from arguing that seizure discharges in those areas, resulting from brain catastrophe, can give rise to the clearly remembered, highly structured NDE. (Fenwick, 1997, p. 48)

In a study of 100 consecutive epilepsy clinic patients with partial complex seizures, none of the

patients’ descriptions met standard criteria for a mystical experience. Although some features of mystical experience were reported, particularly those suggestive of introvertive mysticism, they were not associated with the TPJ nor with any particular lobe of the brain. Mysticism Scale scores were not significantly associated with seizure characteristics, including localization and type of seizure (Greyson et al., 2015).

In that same study, seven patients reported at least one experience associated with a seizure that involved a vague sense of being unaware of their bodies. Those patients did not differ from others in terms of type or location of seizure. Only one patient reported a sense of exiting her body and visualizing her body from a disembodied perspective; that patient had a periventricular lesion that led to epileptiform spikes in the parasagittal region and in both periventricular temporal lobes, but did not involve the TPJ (Greyson et al., 2014).

It is certainly not unheard of for patients to report some features of NDE during their seizures. However, the vast majority of patients with seizures do not experience any of these features. In fact, most patients with temporal lobe epilepsy have no memory afterward for what happened during their seizures (Fenwick, 1997). Epileptologist Ernst Rodin, who favored a neurological explanation of NDEs, acknowledged bluntly: “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 [of NDEs] as part of a seizure” (1989, p. 256). In a prospective study (Devinsky et al., 1991), only 6% of seizure patients described any body image anomalies, let alone OBEs. Furthermore, although these patients had suffered numerous seizures, often over a period of many years, the majority who described body image anomalies reported only one such experience. These findings suggest that localized abnormal activity in the brain is not only unnecessary but also insufficient to produce an OBE (E. W. Kelly, Greyson, & Kelly, 2007).

In supporting the role of the temporal lobe in NDEs, Martial et al. (2025) discussed an electroencephalographic (EEG) study of near-death experiencers by Britton and Bootzin (2004), which found no clinically significant temporal lobe seizure activity, either by EEG or tonic-clonic posturing. The study did find an indirect suggestion of *subclinical* temporal lobe activity that was not suggestive of seizures in 22% of near-

death experiencers and only 5% of comparison participants. But if this subclinical activity in the temporal lobe, which has absolutely no clinical significance, was involved in producing NDEs, why was it absent in three fourths of the near-death experiencers? The experiencers in this study had better positive coping skills than the comparison participants, but the groups did not differ on maladaptive stress responses, suggesting that whatever differentiated the experiencers from the comparison participants was an enhanced function rather than a dysfunction.

Although the near-death experiencers as a group scored higher on a measure of subclinical signs of temporal lobe activity, the concept of whether there really are signs typical of temporal lobe activity remains controversial after centuries of conflicting anecdotal observations (Shetty & Trimble, 1997). Studies of personality traits of epileptic patients have been plagued by methodological problems associated with diagnostic uncertainties and difficulties operationalizing some of the purported psychophysiological correlates of temporal lobe epilepsy. It appears likely that patients who have right temporal lobe seizures do differ from others in certain ways, and a list of the character traits included in standard measures of a “temporal lobe personality” includes many features common among near-death experiencers. Temporal lobe epileptics and near-death experiencers, for example, are both reported to have deeper emotions, elation, increased moral sensitivity, a sense of personal destiny, desire to write detailed notes and even books, deep religious beliefs, and interest in philosophical issues (Persinger & Makarec, 1987). But those similarities do not necessarily imply a similar etiology.

As an analogy, if one stays up all night, one is likely to be tired, mentally foggy, and confused the next day, with difficulty thinking and speaking clearly. The fact that those symptoms may also be associated with alcohol intoxication does not make us identify the sleep-deprived person as being intoxicated. Sleep deprivation itself is a sufficient and straightforward explanation. Similarly, the fact that temporal lobe dysfunction can mimic (a few of) the effects of NDEs does not imply that the experiencer has damaged temporal lobes. The experience itself is sufficient explanation of the effects. There is a tautological character to the argument that defines certain NDE features and aftereffects as epileptic signs (or other pathological symptoms, such as hallucinations or dissociation)

and then presents the same NDE features and aftereffects as evidence that experiencers are unreliable witnesses because they have epilepsy (or hallucinate or dissociate). In addition to relying on a questionable premise, this line of reasoning may also lead to dismissal of important aspects of these profound subjective experiences by forcing them into a pathological framework that may not be appropriate.

Even if the temporal lobe were shown to be implicated in NDEs, which is far from established, it is not plausible that part of the brain is *producing* them. This is because both seizure activity and direct electrical stimulation typically disrupt whatever patterns of neuroelectric activity would otherwise be going on in that part of the brain. Electrical stimulation and seizures are not like physiological electrical activity and do not result simply in a localized “activation” of the stimulated region. As Penfield (1975), the neurosurgeon who first mapped brain regions by stimulating the brain, clearly recognized, the predominant effects of electrical stimulation are disruption of electrical activity in the immediate vicinity of the electrode, accompanied by abnormal patterns of discharge into additional brain areas to which the stimulated cortex itself is linked. The net result of electrical stimulation and epileptic seizures is a poorly controlled, poorly characterized, and spatially widespread pattern of abnormal electrical activity.

In other words, seizure activity and direct electrical stimulation might explain the failure of normal perceptual integration, but not the production of coherent and highly vivid experiences such as NDEs (E. W. Kelly, Greyson, & Kelly, 2007). Electrical stimulation or abnormal electrical discharges in the brain cannot account for complex perception or thought because those disruptions in brain functioning would ordinarily abolish consciousness. This much was acknowledged by Devinsky, who is often cited in support of the temporal lobe model, but who called consciousness during seizures a “paradox” and “an unresolved problem” (Devinsky et al., 1991).

Ketamine and Other Psychedelic Substances

Concerning the role of ketamine in the etiology of NDEs, Martial et al. (2025) cited Jansen’s ketamine model of NDEs, originally developed as a possible neurochemical explanation for NDEs. However, they did not cite Jansen’s later conclusion that, after 12 years of studying ketamine effects on the brain, he viewed ketamine as “just another door” to NDEs and

not actually producing them (Jansen, 1997). Despite some apparent phenomenological similarities between NDEs and ketamine-induced experiences (e.g., dissociation, ego dissolution), as well as semantic parallels between narratives of each, as noted by Martial et al., they recognize that Jansen's model may be at odds with other important aspects of NDEs, such as the often-vivid recall of the experiences. Nonetheless, they use the proposed ketamine connection to suggest a possible mechanism of NDEs via the glutamatergic neurotransmitter system, which is targeted by ketamine.

In general, Martial et al. (2025) applied a similar argument with other substances whose effects may resemble certain aspects of NDE phenomenology to suggest theoretical mechanisms for NDEs based on the various neurotransmitter systems on which these substances act. They specifically singled out N,N-dimethyltryptamine as modeling NDEs in laboratory settings. However, a thorough comparison of the phenomenology of N,N-dimethyltryptamine-facilitated experiences and NDEs revealed far more differences between the two than similarities (Potts, 2012). While the ability of psychedelics to mimic some features of NDEs is an intriguing line of reasoning, it should be presented with caution, as the experiences—though showing similarities—are not equivalent. In a within-subjects comparison of the features of NDEs and mystical experiences induced by serotonergic psychedelic substances (including N,N-dimethyltryptamine) in individuals who have had both, NDEs were rated as involving a higher degree of disembodiment (Martial, Carhart-Harris, & Timmermann, 2024). This NDE feature, especially when combined with vivid perceptions of events going on elsewhere and not acquired via sensory input, presents a challenge for physiological and psychological NDE models.

Endorphins

With respect to the role of endorphins in NDEs, Martial et al. (2025) discussed the possible role of endorphin release under stress in producing NDEs, citing Blackmore's suggestion that endorphins could explain the euphoric or peaceful sensations during NDE (Blackmore, 1996). However, endorphin-associated euphoria lasts several minutes to several hours, whereas the euphoric sensations in NDE cease as soon as the NDE is over, stopping as soon as the patient is awake (e.g., van Lommel, 2010).

Timing of NDEs

Regarding when during the course of cardiac arrest an NDE occurs, Martial et al. (2025) wrote that the timing of NDEs cannot be known, implying that they might occur either before or after cardiac arrest, rather than during the arrest, when brain function is significantly impaired. However, some NDEs include "time anchors," corroborated reports of veridical perceptions during a complete loss of consciousness, permitting verification of the exact time of the NDE (e.g., Greyson, 2010b; E. W. Kelly, Greyson, & Kelly, 2007, p. 419; Khanna et al., 2018; Lake, 2017; Nahm & Weibel, 2020; van Lommel et al., 2001).

Rapid Eye Movement Intrusion

Martial et al. (2025) also discussed the hypothesis that rapid eye movement (REM) intrusion plays a role in NDEs, but they overlooked the contradictory evidence that near-death experiencers report REM-intrusion phenomena no more often than the general population (Greyson et al., 2009) and have longer REM latency and fewer REM periods than comparison groups (Britton & Bootzin, 2004), that REM intrusion is usually associated with distress (Balch et al., 2024) and fear, as in sleep paralysis (Sampat, 2021), and that NDEs often occur under general anesthesia and other drugs that inhibit REM (e.g., Cronin et al., 2001; Greyson & Long, 2006).

Electrical Activity in the Dying Brain

Finally, Martial et al. (2025) discussed relatively more recent explanatory models for NDEs, which concern an unexpected "death surge" in brain electrical activity during the near-death or dying process. They cited recent studies by Vicente et al. (2022) and by Xu et al. (2023), which they characterized as confirming an increase in functional activity across the brain in the human dying brain. But, in fact, neither of these studies involved patients whose hearts had stopped. Xu et al. monitored comatose patients via EEG, after mechanical ventilation had been withdrawn, but cardiac activity persisted for the entire duration they were monitored, and the patients showed no evidence of consciousness (van Lommel & Greyson, 2023).

Vicente et al. (2022) presented a single case study of EEG monitoring in an elderly, severely

brain-damaged patient in whom again cardiac activity persisted for the entire duration of the cerebral electrical activity. Notably, the patient had a number of confounding conditions that may have affected the EEG, including a recent traumatic brain injury and subdural hematoma, anesthesia-induced loss of consciousness, dissociative drugs given to the patient, continual seizures, anticonvulsant drugs given to try to control the seizures, and the patient's asphyxia and anoxia. In addition, the authors had no comparison recording of the patient's normal brain activity prior to his recent brain injury and unconsciousness. Most importantly, as noted above, despite the authors' claim that they were monitoring electrical activity "in the dying human brain," the patient's heart was still showing normal cardiac activity at the time the reported EEG changes occurred. Although such studies provide ample food for thought, they are far from providing evidence of coordinated brain activity after cardiac arrest (Greyson et al., 2022).

Martial et al. (2025) claimed that a marked decrease in power across all EEG frequency bands but an increase in functional connectivity between the bilateral frontal and visual cortices in recordings taken immediately before the cessation of EEG activity (Vicente et al., 2022; Xu et al., 2023) suggested that global hypoxia increased gamma power and gamma coupling with slower oscillations. They noted that this relative increase in gamma activity and connectivity in these two studies may be suggestive of conscious perception. However, no behavioral observations suggesting consciousness were noted in any of the patients in these two studies cited, nor were any verbal reports of conscious experience elicited. While Martial et al. acknowledged that "these findings should be interpreted cautiously as no interview was possible owing to the imminent death of the patients," this limitation cannot be overstated. To further complicate the interpretation of these findings, the gamma oscillations reported are also associated with a wide range of brain circumstances, including pain, and are typical of electrical activity produced by spasms of scalp muscles (especially in the frontal and temporal electrodes, which is where the reported activity was primarily found), which is a well-recognized problem in interpreting EEG spectral power and coherence (Greyson et al., 2022).

Martial et al. (2025) also cited a study by Chawla et al. (2009), suggesting that transient

electrical spikes in critically ill patients may play a role in producing NDEs. However, none of these studies purporting to show brain electrical activity in terminally ill patients have ever included any patient report of a subjective experience nor any behavioral observation suggestive of consciousness, let alone a near-death experience.

Finally, Martial et al. (2025) cited a recent innovative study by Parnia et al. (2023), in which patients undergoing cardiac arrest and manual cardiopulmonary resuscitation or electrical cardioversion had EEG measurements during the 3–5 s pauses for pulse checks between chest compressions or electrical shocks. The research team was able to obtain interpretable EEG activity from only 53 of the 567 patients studied, of whom about half had what appeared to be "near normal/physiological EEG activity ... suggestive of emergence of consciousness" (Parnia et al., 2023, p. 9), although none of them showed any observable signs of being conscious. Parnia et al. reported some level of cognitive awareness among 11 patients, including 6 who reported NDEs and concluded that "The emergence of normal EEG may reflect a resumption of a network-level of cognitive activity, and a biomarker of consciousness, lucidity and recalled experience of death (authentic 'near-death' experiences)" (p. 1).

However, none of the patients in Parnia et al.'s (2023) study who reported NDEs or other types of awareness also showed this EEG activity consistent with consciousness and none of the patients who showed this EEG activity consistent with consciousness reported NDEs or any other cognitive activity. This complete lack of overlap between the patients who had EEGs purportedly consistent with consciousness and the patients who reported having NDEs or being otherwise conscious is evidence *not* for the EEG activity being a biomarker of NDEs but rather the exact opposite, which the EEG activity was *not* a biomarker of NDEs (Greyson & van Lommel, 2024). Parnia (2024) subsequently acknowledged that "The question of consciousness and its relationship with the brain remains one of the biggest mysteries in science" (p. 8), citing "the absence of studies showing any causative mechanism between brain processes and consciousness" (p. 1).

As part of their discussion of abnormal brain activity at death, Martial et al. (2025) speculated that excitation of visual cells during a near-death state could trigger tunnel vision. But tunnel vision (defined as loss of peripheral vision) is not the

same as the perception of a structured tunnel in many NDEs, in which experiencers see not only inside the tunnel but also objects outside the tunnel, with no loss of peripheral vision. As Blackmore, whom Martial et al. cited as having developed this hypothesis, later clarified,

Tunnel vision refers to a narrowing of the visual field down to a small area in the center. There is simply no visual experience in the periphery, rather than a specific visual impression of darkness.

In a tunnel experience, by contrast, there is usually a bright light at the end of a dark, but often complex, tunnel. People describe spirals, colored webs, tunnels made from multiple images, spaces full of stars, or solid tunnels like subways or sewers, and many other complex images in tunnel form.... We should be careful to discriminate between tunnel vision and tunnel experiences during NDEs. (Blackmore, 1999, pp. 271–272)

Additional Considerations

Regarding NDEs as Hallucinations

Throughout their article, Martial et al. (2025) referred to NDEs as “hallucinations” caused by various neurological or neurochemical factors. However, neurologically generated hallucinations typically involve only one sensory modality (most often, vision or hearing) and are usually accompanied by fear and confusion, quite unlike the realistic, interactive visions of deceased persons on NDEs, which are usually welcomed by and comforting to the experiencer and often seen, heard, smelled, and touched (E. W. Kelly, 2001). In addition, unlike hallucinations, NDEs tend to result in memories that are retained for decades (Greyson, 2007), are perceived as “realer” than other memories around the same period (Moore & Greyson, 2017; Palmieri et al., 2014; Thonnard et al., 2013) and often have a meaningful and positive impact on experiencers that persists for decades (Greyson, 2022). Notably, some NDEs also feature veridicality of perceptions (Holden, 2009), which is not typical of neurological hallucinations.

Unexplained NDE Features

Martial et al. (2025) concluded their description of their multifactorial physiological mechanism for NDEs with appropriate caution that some questions remain unanswered, such as the relative importance of the various mechanisms described and that the model does not cover all

key NDE features. Of note, they did not discuss major NDE features that seem incompatible with their physicalist theory, such as veridical out-of-body perceptions during NDEs (Holden, 2009). Furthermore, some NDEs include encounters with deceased persons of whose death the experiencer had no knowledge, or whom the experiencer had never met; accurate information acquired about the deaths of these deceased persons challenges the interpretation of these visions as hallucinations (Greyson, 2010c; Khanna et al., 2018).

Philosophical Bias

In addition to the restricted range of empirical evidence reviewed in their article, Martial et al. (2025) acknowledged that, in developing their coherent overarching model, “We have excluded dualistic theories from our discussion owing to the lack of empirical neuroscientific evidence and the fact that a fundamental tenet of neuroscience asserts that human experience arises from the brain” (p. 2). However, two of the four sources they cited in support of this comprehensive dismissal of evidence for dualistic theories of mind-brain function were papers by neuropsychiatrist Giulio Tononi, who most recently disavowed that “fundamental tenet” of all human experience arising from brain function, writing,

Computations may successfully explain how the brain does what it does (its functions) just as they can explain why a computer does what it does; however, consciousness as such—its presence and quality (say, what it is like to see a face in the middle of the visual field)—remains unexplained, not to say magical.

Maybe it requires ... some mysterious biological ingredient (reminiscent of the *élan vital*). However, this means that, scientifically, we are back to magic, or at least to square one

Science benefits from imagination and experimentation coupled with seriousness of purpose and openness of mind. It will not benefit from a self-appointed tribunal—the academic equivalent of the congregation for the doctrine of the faith (the computational-functionalist faith, that is)—deciding which topics or approaches are permissible and which should be banned. Especially with something as hard as consciousness. (Tononi et al., 2025, p. 694, 701, 702)

Indeed, there are a number of physicalist models of consciousness that, while generally assuming that consciousness emerges from the brain, make differing assumptions and lead to differing implications (Seth & Bayne, 2022; Wahbeh et al., 2022). These varying physicalist

models can be grouped (Wahbeh et al., 2022) into higher order theories (e.g., Cleeremans et al., 2020), global work-space theories (e.g., Baars et al., 2021), integrated information theory (e.g., Tononi et al., 2016), and reentry and predictive processing theories (e.g., Lamme, 2010). What these physicalist models share with each other, and with the NEPTUNE model, is an assumption that consciousness is produced by, and can therefore be fully explained in terms of, brain activity.

However, there is a substantial body of empirical evidence, both from NDEs (e.g., Greyson et al., 2012; O'Grady & Varghese, 2025; Rivas et al., 2023) and from related phenomena (e.g., E. F. Kelly, Kelly, et al., 2007; Kelly & Marshall, 2021), suggesting that physicalist models fail to account adequately for these common experiences, in which conscious, vivid, and accurate perception has been demonstrated to occur in the absence of normal brain activity, and even under conditions of severe brain impairment. The hypothesis that all human experience arises solely from the brain is an assumption of physicalist philosophy and not "a fundamental tenet of neuroscience."

Just as there are a number of physicalist models of consciousness with differing assumptions and implications, so too are there a number of non-physicalist models of consciousness that do not assume that a physical substrate generates consciousness (Wahbeh et al., 2022). Such non-physicalist models, supported by empirical evidence, mathematical modeling, and philosophical reasoning, include operational probabilistic theories (e.g., Faggin, 2021), interface theories of perception (e.g., Hoffman et al., 2015), analytic idealism (e.g., Kastrup, 2021), the triadic dimensional vortical paradigm (e.g., Neppe & Close, 2020), zero-point field theories (e.g., Keppler, 2018), orchestrated objective reduction theory (e.g., Hameroff, 2021), the hypothesis of subjective time (e.g., Schooler, 2015), and the theory of double causality (e.g., Guillemant & Medale, 2019). It is beyond the scope of this article to provide an in-depth discussion of varying models of consciousness, both physicalist and nonphysicalist. However, our purpose in listing them here is to highlight the richness of serious scholarship on this topic.

In light of the empirical phenomena of NDEs and the viable philosophical alternative models of consciousness, as well as prior methodological objections to Martial et al.'s (2025) assumptions (Engmann, 2025; O'Grady & Varghese, 2025),

we submit that it may be premature to anoint the NEPTUNE model as a "comprehensive explanation for NDEs" (Martial et al., 2025, p. 1), until all the relevant data have been considered.

Conclusion

In this article, we review Martial et al.'s (2025) recently published theoretical explanatory model combining various neurophysiological and psychological hypotheses for the mechanisms that may generate NDEs. Developing such a framework is a reasonable approach, as NDEs are typically triggered by physical (e.g., illness or trauma) or psychological (e.g., psychological trauma or expectation of dying) factors. The authors drew on evidence and arguments from multiple levels of analysis to present a multi-factorial explanation of NDEs, including neurochemical influences via distinct neurotransmitter systems and specific brain regions whose altered functioning may contribute to the occurrence of NDEs. Integrating the various physiological and some psychological models that have been proposed over the decades to explain NDEs is a significant undertaking, and Martial et al. have done a monumental job of summarizing the major arguments in this field, relying on both theoretical and empirical contributions from the NDE literature and broader medical research.

Despite the upsides of this ambitious undertaking and the effort invested, we propose that the data synthesis underlying the NEPTUNE model raises some concerns that temper our enthusiasm for the model and our confidence that it can provide a comprehensive explanation for NDEs. A major critique centers on the omission of empirical data that either fails to support or directly contradicts speculation presented by Martial et al. (2025). In other instances, we raise concerns about arguments based on data from experiences that superficially resemble some NDE features, such as purported OBEs induced by TPJ stimulation and confusing "tunnel vision" with "tunnel experiences," but upon closer examination appear significantly different from typical reports from experiencers about these features. Regarding recent claims that brain electrical "surges" purportedly at the end of life may explain the occurrence of NDEs, we raise issues about the lack of any evidence directly linking such electrical activity to vivid conscious perceptions of NDEs specifically. Although Martial et al. acknowledged that the NEPTUNE

model does not account for all features of NDEs, we suggest that those salient NDE features that are difficult if not impossible for the model to explain are more critical than they proposed.

Raising these issues should not be interpreted as a categorical rejection or dismissal of any physiological causes or correlates associated with the occurrence of NDEs. Clearly, NDEs are typically triggered by physiological events. Rather, our position is that, based on all of the currently available data, these physiological correlates alone are insufficient to account fully for the occurrence and phenomenology of NDEs. Going forward, we believe that in addition to the empirical testing of various components of the NEPTUNE model, it will also be important to remain open to other potential causes, whether currently unknown or not yet fully understood.

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