LETTER TO THE EDITOR

Response to “Some Basic Problems with the Term ‘Near-Death Experience’”

To the Editor:

In his Letter to the Editor of this Journal (this issue), Birk Engmann noted that the criteria for the term “near-death experience” (NDE) popularly include both experiential features, such as an out-of-body experience, as well as a connection to clinical death. He wrote that the experiential criteria are problematic because (1) it is unclear how many of them must be present, (2) it is unclear which (if any) are most important, (3) some of these features are in fact uncommon in NDEs, and (4) others of these features may be common in experiences other than NDEs. He also found the connection to clinical death problematic because (1) the experiential features can occur without clinical death, (2) the experiential features usually do not accompany clinical death, and (3) healthy people report more NDEs than do people who were clinically dead.

Pointing out semantic problems, as Engmann did, can be useful to a developing field in refining scholars’ and researchers’ questions and understanding. However, rather than suggest some helpful clarification of these criteria, Engmann instead implied that these problems render the concept of NDEs scientifically meaningless. To the contrary, the shortcomings in the criteria for NDE that Engmann mentioned—and, indeed, many other semantic problems—have been productively discussed for the past three decades in an extensive peer-reviewed literature examining the implications of varying definitions of and criteria for NDEs (e.g., Bates & Stanley, 1985; Greyson, 1998; Hobson, 1978; Smith, 1991).

Engmann further confused the definition of an NDE with the criteria for identifying an NDE. The general, abstract definition and the specific operational criteria are two different constructs that serve very different purposes. I have previously reviewed the uses and varying utility of definitions of NDEs, criteria for NDEs, and empirical tests of NDEs in studying these phenomena (Greyson, 1999). I believe that by failing to differentiate between definitions and criteria, Engmann has clouded rather than clarified the issues.

Engmann further noted that different parts of the brain may have
varying susceptibility to malfunction than others, suggesting that NDEs are caused by brain malfunction. But the likelihood that some parts of the brain may have varying susceptibility to malfunction reveals nothing about possible neurological causes of NDEs. It would be surprising indeed if NDEs were not associated with brain malfunction, because the brain necessarily malfunctions as the body approaches death; but there is no scientific basis for attributing the cause of NDEs to such brain malfunction. Although Engmann was correct in noting that many NDEs occur in people without documented clinical death, a large number of well-substantiated cases involve patients who were indeed clinically dead. In fact, the professional literature contains hundreds of published cases of NDEs occurring under conditions such as cardiac arrest and deep anesthesia in which standard neurophysiology models of the brain rule out conscious experience of any sort, let alone the vivid and complex thinking, perceptions, and memory typical of NDEs (Kelly, Greyson, & Kelly, 2007).

Engmann cited a study by Zalika Klemenc-Ketis and colleagues (Klemenc-Ketis, Kersnik, & Grmec, 2010) as supporting a link between NDEs and brain alterations. That small study found NDEs to be associated with high carbon dioxide levels—which was surprising, because larger studies both by Michael Sabom (1982) and by Sam Parania and colleagues (2001) found no association of NDEs with carbon dioxide levels. However, Klemenc-Ketis and colleagues tested several physiological variables, of which two were associated with NDEs. If they had corrected their statistics for multiple simultaneous univariate tests, as is usually done in medical research, then neither of those differences would have been significant. Thus, the odds that the link between carbon dioxide and NDEs in their study occurred just by chance were greater than most medical journals require for reporting results. Moreover, the meaning of this possible association, if in fact it exists, is far from clear. High carbon dioxide levels result from better cardiac output and perfusion pressure, which would reduce the amnesia usually seen in cardiac arrest. Therefore, any association between carbon dioxide levels and reports of NDEs might show only that patients who can remember more of what happened during their cardiac arrests also can better recall, and consequently report more, NDEs.

Engmann concluded that NDEs are “caused” not only by neuropsychology factors but also by cultural and religious variables. There is certainly evidence that neuropsychology, culture, and religion influence experiencers’ perception and understanding of their NDEs, but
there is no evidence that those factors cause the experience. Engmann claimed that out-of-body experiences (OBEs) are caused by drug abuse or temporal lobe seizures. Temporal lobe seizures also cause hallucinations of music, but that fact does not substantiate the claim that everyone who hears music is having a seizure-induced hallucination. Likewise, the fact that drugs or seizures can induce hallucinations of being out of the body does not imply that drugs or seizures are the cause of all OBEs. In fact, the induced hallucinations of OBEs are quite different from NDEs and other spontaneous OBEs in many ways (Greyson, Parnia, & Fenwick, 2008), not least of which being that near-death-related OBEs include accurate perceptions from an extracorporeal visual perspective in more than 90% of documented cases (Holden, 2009), whereas induced hallucinations do not.

Engmann included a parenthetical comment that the idea of a mind existing independent of a brain should be dismissed because it runs contrary to standard explanations for brain disorders like Alzheimer’s disease. However, it is now clear that the “standard” brain-mind identity model does not in fact explain disorders like Alzheimer’s disease in which patients can paradoxically recover mental function as the brain deteriorates, a phenomenon known as “terminal lucidity” (Nahm & Greyson, 2009).

Engmann is correct that the criteria for NDEs are imprecise. However, if the field of near-death studies is to advance, that shortcoming is not a reason to dismiss the phenomenon but, rather, is a justification for further research to refine the criteria.

References


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