# Near death experiences (NDEs): The dying brain

**Towards a Cognitive Neuroscience of the Dying Brain. An in-depth analysis and critique of the survivalists' neuroscience of near-death experiences.**

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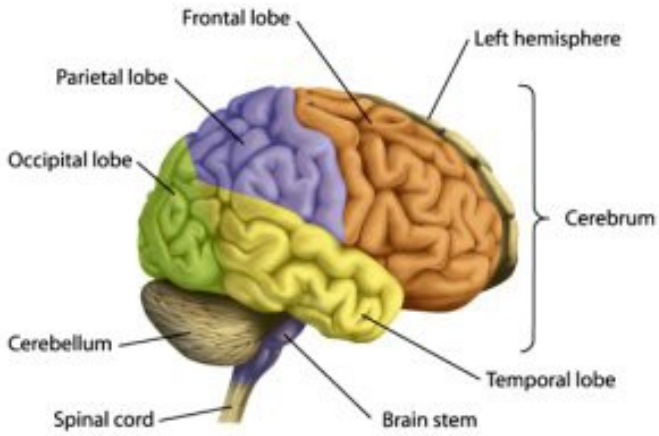
## Introduction

There is a growing perception that the existence of near-death experiences (NDEs) poses a serious challenge to current scientific understandings of the brain, mind and consciousness (Braude, 2003; Fenwick & Fenwick, 1995; Parnia & Fenwick, 2002; Parnia, Spearpoint & Fenwick, 2007; Parnia, Waller, Yeates & Fenwick, 2001; Ring, 1980; Sabom, 1998, 1982). This was reaffirmed recently in a high-impact publication which received world wide attention (van Lommel, van Wees, Meyers, & Elfferich, 2001). This is in some friction with the dominant view from mainstream neuroscience; that the mind is, what the brain does (for comprehensive reviews, see Gray, 2004; van Hemmen & Sejnowski, 2006; Kanwisher & Duncan, 2004). According to the current scientific view, consciousness is an emergent property of the human brain in action. Within mainstream science, this is hardly a controversial or indeed unsupported viewpoint.

Like a number of researchers before them (Fenwick & Fenwick, 1995; Parnia & Fenwick, 2002; Parnia, et al., 2001; Ring, 1980; Sabom, 1998), van Lommel et al. (2001) argued that their Near Death Experience research findings support the need for a radical revision of mainstream views concerning the relationship between the brain and consciousness. The implication is that the mind may be separable from the brain and hence we may all survive bodily death (known as the survivalist position). In contrast, other researchers have suggested that these experiences are hallucinations: the final visions produced by a massively disinhibited and dying brain (Blackmore, 1996, 1993, 1992, 1990; Braithwaite, 1998; Carr, 1982, 1981; Jansen, 1996, 1990; Saavedra- Aguilar & Gomez-Jeria, 1989). Although the various dying-brain accounts may concentrate on contributions from different mechanisms, none assume that mind is separate from brain.




The nature of the claim being made by the survivalists should not be underestimated. If true, it would require a truly radical revision of current neuroscience and the known laws of physics. To support such a radical view one would ideally require radical evidence of high quality. Did van Lommel et al. (2001) furnish their interpretations with such evidence? No. Despite its impact in Near Death Experience circles, the van Lommel et al. study provides no evidence

that human consciousness survives bodily death. This paper briefly examines the factual and logical errors present in the analysis proposed in the van Lommel et al. study. It should be noted that the criticisms outlined here for that study also apply to the other studies promoting the survivalist position which are based on similar arguments.

**The study of Pim van Lommel et al. (2001)**

Methodologically speaking the van Lommel et al. (2001) study makes a useful contribution. They carried out a prospective study of 344 successfully resuscitated cardiac patients, 18% of whom reported Near Death Experiences (12% reported core NDEs). They investigated a host of factors, including demographic variables, age and medical history, and also interviewed patients a number of times over an eight-year period. There is certainly a wealth of useful data gathered by this study and researchers interested in the Near Death Experience would do well to consult this work. However, the real problems with the van Lommel et al. study are not so much related to their methods, but their interpretations and conclusions.

> "Survivalists have repeatedly misunderstood and misrepresented the dying-brain hypothesis when trying to argue against it"

Based on their findings, van Lommel et al. (2001) concluded that we now require a new approach to consciousness – one that gives provision for non-irreducibility of the mind to the brain. In other words, the mind is not what the brain does and may indeed be independent of it. This neo-dualism is worrying. It is worrying as it appears to be primarily based on a potent combination of both factual and logical errors concerning the role of the brain in mental experience. The present paper will argue that the conclusions van Lommel et al. propose are at least premature and are at most unfounded. As such, the van Lommel study poses no serious challenge at all to current neuroscientific accounts of the Near Death Experience.

**Misunderstandings over the role of anoxia: The 18% claim does not support survival**

Survivalists have repeatedly misunderstood and misrepresented the dying-brain hypothesis when trying to argue against it (see for example: Fenwick, 1995; Fontana, 1992; Parnia & Fenwick, 2001; Parnia et al., 2001; Smythies, 1992). The van Lommel et al. study was no exception. Fundamental to van Lommel et al.'s argument against the dying-brain hypothesis was the observation that only 18% of patients actually reported an NDE. Apparently (according to van Lommel et al.), this supports the case for a whole new approach to consciousness (see also Fenwick & Fenwick, 1995; Fontana, 1992). I disagree. Their reasoning was as follows. They argued that if cerebral anoxia was crucial for causing these experiences, and these patients experienced the same level of anoxia, then all should have reported Near Death Experiences. They state (van Lommel et al., 2001, p. 2039):

> "With a purely physiological explanation such as cerebral anoxia for the experience, most patients who have been clinically dead should report one."

Subsequently, they claim (van Lommel et al., 2001, p. 2043):

> "Our results show that medical factors cannot account for occurrence of NDE; although all patients had been clinically dead, most did not have NDE. Furthermore, seriousness of the crisis was not related to occurrence or depth of the experience. If purely physiological factors resulting from cerebral anoxia caused NDE, most of our patients should have had this experience."

From this, van Lommel et al. argued that, as only 18% reported NDE, this is clear evidence against the idea that the experiences are due to a dying brain. If this was the case, then all comparable patients should have reported an NDE. From this point, it appears to have been a small and 'logical' progression to directly infer that these experiences must be of paranormal origin and that these experiences index some form of survival of consciousness. This analysis is unsupported, illogical, and academically speaking, misleading.

Before going any further, it is important to be clear that van Lommel et al. (2001) provided no direct measures of anoxia for anyone in their sample. The presence and level of anoxia was indirectly inferred via experiential components provided in questionnaire responses and medical information regarding the nature and duration of the cardiac arrest. While one can accept the general essence of this reasoning, the method is certainly indirect and highly problematic. As a consequence, the claims of the study go far beyond what the data were capable of showing. No hard claims over the levels of anoxia should have been made when there was little or no attempt to measure it directly. This is problematic for van Lommel et al., as their fundamental claim rests on the assumption that patients had comparable levels of anoxia (something which was never shown to be the case). There was no direct evidence, and hence no real reason to assume, that this was the case. As such, the whole rationale of this claim is undermined. While we can accept that those patients who suffered longer periods of cardiac insufficiency are more likely to have

received greater levels of anoxia, we have no idea what those levels were in each case, nor indeed that they were comparable. Comparing patients who have undergone a similar duration of cardiac arrest is also not a direct metric at all of the balance of blood gases in the brain as resuscitation methods, their duration, and their efficiency will have varied considerably (not to mention the physical differences across patients).

*Rene Descartes' name will forever be associated with the concept of dualism but it has yet to be proven that the mind can be separated from the brain*

Secondly, and perhaps more worryingly, the dying brain hypothesis makes no such direct claims about the level of anoxia per se. Blackmore (1996, 1993, 1992, 1990) is quite clear on the matter that it is the rate of change or rate of anoxia onset that is important, not the overall level reached (see also Woerlee, 2003, for further evidence). If the onset of anoxia is too fast, patients simply lose consciousness and black out. Here, no conscious experience or memory would occur. With more prolonged rates of onset, the patient can seem somewhat confused and dazed. However, an intermediate level of change seems more conducive to intense altered states and NDEs (Appleby, 1989; Blackmore, 1996, 1993, 1990; Woerlee, 2003). This was a clear point made explicit by Blackmore, who also outlined the many different types of anoxia and their experiential consequences yet this is either completely missed or misunderstood by van Lommel et al. Therefore, the whole logic of this position is based on a false and vastly oversimplified premise concerning the dying brain hypothesis.

Thirdly, van Lommel et al. also totally ignored the degrees of within-brain and between-brain heterogeneity which would have implications for the degree of anoxia present, its rate of onset, and how it could impact on human experience. For example, in terms of within-brain differences, Blackmore (1993) noted that as well as there being many different forms of anoxia (that have diverse neurophysiological consequences), any given rate of anoxia can impact on different brain areas disproportionately due to cell proximity to arteries and capillaries, to localised cell density, connectivity, and indeed the current levels of demand and activity in the specific neural systems being affected (Blackmore, 1993, 1990; Woerlee, 2003). In a structural sense, differing brain regions have differing numbers of neurons with diverse connections and characteristics; all of which have differing oxygen demands. In a functional sense, levels of activity across neural systems within and between brain regions will not be matched – and so certain areas will be more susceptible to anoxia than others – based on the current processing demands taking place. In terms of between-brain variability, one illustrative line of evidence is that air-force pilots have been shown to have different thresholds of G-LOC and can tolerate (within a certain degree) a different level of stress and anoxia before losing consciousness (Whinnery, 1997, 1990; see Blackmore, 1993, for a discussion). Under these circumstances the amount of G-force can be controlled, yet clear differences across individuals exist. These differences reflect important physiological characteristics which clearly interact with external stressors. So a given level of anoxia can impact on experience differently across individuals. The van Lommel et al. study ignores these well known and well documented aspects of the dying-brain account.

Finally, a further logical problem is that it is not at all clear how an afterlife hypothesis actually explains the 18% rate of NDE. Surely, if an afterlife existence were real, all those in a position to glimpse it would do so? In other words, if the afterlife existed in some real sense, the real question is why did only 18% glimpse it? Indeed, is it not more of a problem for the afterlife hypothesis that only 18% have reported such experiences? Van Lommel et al. say nothing about this and as such no viable survivalist case was ever made for why only 18% of patients reported NDEs. At the very least, this seems to be an opportunity lost by the authors.

**Misplaced confidence in EEG measurements**

Within Near Death Experience research, a number of investigators have argued that a flat electroencephalogram (EEG) reading can be taken as evidence of total brain inactivity (and van Lommel et al. recruit this argument into their interpretation; Fenwick & Fenwick, 1995; Parnia & Fenwick, 2001; Parnia et al., 2001; Sabom, 1998). This claim is totally incorrect. It is certainly the case that a flat cortical EEG would be indicative of a brain that is in some

trouble. Assuming no technical error or problems with electrode contact, a flat EEG is far from desirable. However, the assumption that a flat EEG can be taken as strong evidence of global and total brain inactivity is unfounded. (It is also noteworthy that the studies making large claims about flat EEGs provide no information regarding the level of gain employed on the EEG device, assuming they were digital-QEEG devices. This would seem important as any EEG can become almost flat with the gain turned to a minimum. A flat EEG at maximum gain would be more indicative of neocortical inactivity, though again, not full-brain inactivity).

Unless surgically implanted into the brain directly, the EEG principally measures surface cortical activity. The waveforms seen in cortical EEG are largely regarded to come from the synchronistic firing of cortical pyramidal neurons. As such, it is entirely conceivable that deep sub-cortical brain structures could be firing, and even in seizure, in the absence of any cortical signs of this activity (for evidence based on electrical stimulation and seizure propagation, see Gloor, 1986; Gloor, Olivier, Quesney, Andermann, & Horowitz, 1982). Indeed, evidence reviewed by Gloor (1986) argued that inter-ictal discharges in the hippocampus or amygdala alone were more than sufficient to produce complex meaningful hallucinations – no involvement from the cortex was necessary!

A related idea is that seizure-based hallucinatory EEG patterns have been absent from the background EEG in some instances of NDE, even when the EEG itself was not flat (Fenwick & Fenwick, 1995). By this account, if the NDE was a hallucinatory process based in disinhibition, then the logic is that such disinhibition should be clearly visible in the EEG at that time.

However, the emerging evidence is somewhat unhelpful for the survivalist. Tao, Ray, Hawes-Ebersole, and Ebersole (2005) compared EEG activity from surgically implanted electrodes placed in or around deep sub-cortical regions of epileptic patients with cortical EEG electrodes placed on the scalp of the same patients. The results were quite surprising. Tao et al. showed that for 90% of cases, large amplitude paroxysmal firing needed to recruit 10 cm2 of brain tissue in order to show up against background cortical EEG traces. In other words, large seizure-based activity was being recorded by the surgically implanted electrodes (indexing clear and widespread brain-seizure activity) which was completely absent from scalp-based EEG traces until it propagated through and excited 10 cm2 of brain volume. This is a considerable amount of brain tissue.

Furthermore, a recent study that employed both EEG and brain-imaging (fMRI) techniques to explore seizure processes found significant increases in localised cortical neural activity (indicative of a seizure) in the fMRI BOLD (blood-oxygen-level dependant) response, which was completely absent from the EEG data (Kobayashi, Hawco, Grova, Dubeau, & Gotman, 2006). This is particularly striking in that this occurred despite the fact that the intense seizure activity occurred in a region where EEG electrodes were closely spaced. Kobayashi et al. note that this is striking as the EEG completely missed the most intensely discharging region despite the fact that this region was also located at the cortical level.

> "... differing brain regions have differing numbers of neurons, with diverse connections and characteristics – all of which have differing oxygen demands"

The implication for NDE research is, of course, that the EEG does not provide a highly reliable measure of complete neural activity. Even high-amplitude seizure activity can fail to manifest itself in the background EEG if it does not recruit enough neural landscape. To summarise: confidence in previous claims that flat EEG represents total neural inactivity appears severely misplaced. These cases may represent instances of 'false positives' (positive from the perspective of the survivalist wanting to recruit such instances as evidence of a dead brain). In addition, even in the presence of a background EEG, seizure-based activity (which is sufficient to support hallucinatory imagery and aura) could be considerable and yet may not become manifest in the cortical scalp-based EEG. Note also that the above empirical estimates were based on epileptic brains which produce large-amplitude brain activity. These estimates themselves may need to be increased even further for the normal non-epileptic brain which does not typically produce such high-amplitude synchronistic characteristics.

**What the dying brain hypothesis really says: The importance of neural disinhibition**

When one considers the dying-brain account in its full context it is clear to see that the emphasis placed on cerebral anoxia misses the true essence of the account. As a consequence, many of the criticisms against the dying-brain hypothesis border on the irrelevant. For the dying-brain account, the central assumption does not revolve around the presence or absence of anoxia per se, but of neural disinhibition. So the dying-brain hypothesis is perhaps more accurately characterised as one that models NDEs as an experiential consequence of a disinhibited brain (Blackmore, 1996, 1993, 1992, 1990; Braithwaite, 1998; Carr, 1982, 1981; Jansen, 1996, 1990; Saavedra-Aguilar & Gomez-Jeria, 1989; Woerlee, 2003). Of course, such neural disinhibition can be induced by anoxia, and it is likely that under more prolonged near-death situations it is likely to be present; but as a process, disinhibition can actually be triggered by many psychological and neurological factors such as: confusion, trauma, sensory deprivation, illness, pathology,

epilepsy, migraine, drug use and brain stimulation (for comprehensive reviews see: Appleby, 1989; Baldwin, 1970; Blackmore, 1993; Sacks, 1995; Siegal, 1980). Without exception, all these instances that induce neural disinhibition and seizure-type activity can all be associated with aura and hallucination.

In principle then, anoxia does not need to be present at all to produce hallucinatory imagery. However, under cases where people are 'near death' or suffer cardiac insufficiency for any prolonged period of time, it is likely (i.e. reasonable to assume) a degree of anoxia would be present. Therefore, while anoxia is one route via which disinhibition can occur, it is by no means the only route. In addition, the dying-brain hypothesis predicts that more vivid, profound, and meaningful NDEs are likely to be associated with greater degrees of disinhibition. Thus, NDEs reported when people truly are nearer to death (and hence the level of disinhibition would conceivably be greater), should be more vivid, profound, detailed and meaningful, relative to those reported when people only believed themselves to be so. This is exactly what has been found (Drab, 1981; Gabbard & Twemlow, 1984; Gabbard, Twemlow, & Jones, 1981; Owens, Cook, & Stevenson, 1990).


*The soul of a good old man leaves him at the moment of death and is borne away by angels to Paradise.*

The idea that disinhibition underlies these striking perceptions is further evidenced by the brain's very limited scope for tolerating abnormal states and how it typically responds when it does encounter them. By far the most common reaction from the brain to such states is disinhibition and seizure. Very small changes in the neural environment have been shown to be more than sufficient to impact on the fine balance maintained in the brain. For example, a 10-15% reduction in GABA inhibition is sufficient to significantly increase seizure propagation in cortical tissue, and changes of a few millimoles in extracellular potassium levels can turn a stable neural population into an epileptogenic one (Chagnac-Amitai & Connors, 1989; Haglund & Schwartzkroin, 1990; Korn, Giacchino, Chamberlin, & Dingledine, 1987). The ranges of these values are well within those encountered under normal brain functioning. The real question then becomes not one of whether disinhibition or seizure could be involved in contexts conducive to NDE, but as Schwartzkroin (1997) states, more one of why seizures are not indeed far more common and why are we are not all having seizures constantly!

There is a further conundrum for the survivalist: in order for any experience to be remembered (assuming some form of perceptual experience occurred) memory must have encoded and represented the experience in the first place. Applied to the NDE, this means that there must have been sufficient neural activity to encode the experience, to represent the experience, and to store the experience (even a glimpse of an afterlife would require this). As far as current science is concerned, it is not at all clear how a memory of an experience can occur without the use of memory itself. The very fact that these experiences were 'remembered' in the first place suggests that memory itself was functioning and encoding at the time of the experience (meaning there was neural activity in those brain regions during the experience - which may indeed have been responsible for the experience).

Of course false-memories show that we can remember the palpably untrue as a real memory, but these false memories are often based on illusory conjunctions between other encoded information represented in our memory systems (see Brainerd & Reyna, 2005). A false memory still requires an intact memory system; or at the very least, a partially intact one. In addition to this, other survivalists have argued that a brain near-death is too unstable to support vivid hallucination and so cannot be an explanation for NDE (Fenwick & Fenwick, 1995; Parnia & Fenwick, 2002; Parnia, et al., 2001). The logical problem, however, for these researchers is: if the brain is too unstable to support hallucination, how is it possible for it to be stable enough to 'remember' mystical experience? A further problem is that it is factually incorrect; all disinhibitory models of brain function have provision for stable vivid hallucination (for examples, see Blackmore, 1993; Cowan, 1982; Sacks, 1995). Indeed, a disinhibited brain could produce an experience that is 'more vivid' and stable than even veridical perception as that experience would be endowed with ferocious neural activity, at least for a given time period. In addition, the survivalists assume that neural stability and cognitive stability are one and the same thing, which is certainly not the case.

This is the crucial logical fallacy of this whole field of research: how can one memorise an event in the absence of a working and functioning memory system? If, as the survivalists claim, the brain is dead then surely, so is memory. If memory is dead, then how can individuals remember anything – even if the original experience was mystical? The only way around this for the survivalist is to add some more untested assumptions and degrees of freedom that are

tailored to allow for some paranormal mechanism in the first place. However, this again is a folly. Firstly, it violates the principles of Occam's razor by adding assumptions that are clearly unjustified. Secondly, it begs the question: assuming to be true that which it seeks to argue is true in the first place. It thus represents a hopeless case of circular reasoning. The survivalists can only make their arguments work here by assuming further untested, supernatural ideas to be true. This is a serious error of reasoning, and one that undermines the argument to the level of uselessness.

Finally, the inescapable fact for the survivalist is that the brain is constantly trying to make sense of the ambiguous information it is given to arrive at a stable and coherent interpretation. If the context and information provided to the senses are unfamiliar, odd and bizarre, then one should not be surprised if the resulting conscious experience is somewhat unfamiliar, odd and bizarre (Cooney & Gazzaniga, 2003). This fits neatly with developments in cognitive psychology, cognitive science, and neuroscience that view neurocognition as an active model-building process. According to recent emerging scientific frameworks, even stable conscious experience is something of a fiction, but a far lesser fiction than other possible alternative realities. By this account, stable perception and indeed consciousness itself can be viewed as a form of controlled hallucination (Bentall, 1990; Claxton, 2005; Morgan 2003). Once it is realised that normal perception itself can be viewed, to some degree, as a stable and successful hallucination, it is hardly a leap to view Near Death Experiences as an extension of this natural process. The Near Death Experience then is merely a greater fiction that serves a temporary purpose for consciousness in that, for a short while, it represents reality in the absence of the more usual and stable reality provided by the senses (Blackmore, 1993; Braithwaite, 1998; Claxton, 2005; Morgan, 2003).

**Other common misunderstandings**

For neuroscientists, the fact that many components of the NDE are very similar to experiences associated with pathology, disease, illness, neurological conditions (e.g., schizophrenia, autoscopy, Charles-Bonnet syndrome, migraine aura, epilepsy aura) and direct forms of brain stimulation is a strong indication that such experiences have an underlying neural correlate (Bentall, 2003; ffytche, 2000, 1999, 1998; Gloor, 1986; Gloor et al., 1982; Bear, 1979; Halgren, Walter, Cherlow, & Crandall, 1978; Sacks, 1995; Siegal, 1980, 1977). There is no component of the NDE that is unique to being 'near-death'.

Ignoring such strong similarities, survivalists like to highlight the marginal differences and van Lommel et al. (2001) did not miss their opportunity to further add to this confusion. When discussing the experiences associated with direct electrical brain stimulation they stated (van Lommel et al., 2001, p. 2044):

> "These recollections, however, consist of fragmented and random memories unlike the panoramic life-review that can occur in NDE. Further, transformational processes with changing life-insight and disappearance of fear of death are rarely reported after induced experiences. Thus, induced experiences are not identical to NDE…"

Firstly, this claim is not entirely correct. Vivid and meaningful experiences are reported by patients undergoing brain stimulation (see Gloor, 1986; Gloor et al., 1982; Bear, 1979; Halgren et al., 1978). Secondly, what the analysis of van Lommel et al. ignores is the crucial role of context. Patients undergoing electrical brain stimulation are typically conscious, know what to expect, are relaxed and enjoy a constant controlled interaction with the surgeon (Gloor, 1986; Gloor et al., 1982; Halgren et al., 1978; Penfield, 1955; Penfield & Perot, 1963). They also receive constant feedback from the surgical team. This is nothing like the experiential context of the typical NDE where the patient is only semi-conscious (at best), and possibly undergoing some form of trauma, confusion, disorientation and dissociation from their surroundings. It is certainly not unreasonable to assume that the small experiential differences between NDE and brain stimulation studies can be explained, to some degree, by these large differences in context. This is certainly a far more probable conclusion than that of mind-brain dualism.

Furthermore, the reason the experiences under artificial circumstances are perhaps more brief and fragmented has nothing to do with a special status for the Near Death Experience, but more to do with the fact that the surgeon temporarily stimulates specific neuronal cell assemblies in an attempt to hone in on the type of aura experiences that the patients report as part of their epileptic condition. Under these circumstances the stimulation is meant to be brief, localised and controlled, which again is totally unlike a large intense seizure that would likely propagate through more tissue. The surgeon is trying merely to induce aura, not a massive seizure. It is certainly not the aim of the surgeon to induce deep, meaningful and long lasting spiritual experiences. It is usually the case that many experiences are elicited before the sought after aura is induced. Once the region associated with a particular sensation/aura has been identified then the surgery can begin.

To ignore these crucial differences in context is to do more than a disservice to both the relevance of these brain-stimulation studies and the way the dying-brain hypothesis recruits them into a theoretical framework. The dying-

brain hypothesis states that the fact that highly similar experiences occur through direct interaction with neural tissue strongly implicates the role of the brain in the Near Death Experience. It never claimed that the experiences under both contexts should be identical – simply because both contexts are not identical! To illustrate this further, imagine you become stranded in a busy city centre and need to find your way home. The feeling associated with being stranded would be totally different if that city centre was familiar to you versus being completely unfamiliar and foreign to you. This is despite the fact that the same process, that of being stranded, underlies both experiences.

In their discussion and interpretation, van Lommel et al. (2001) perpetuate a further common misunderstanding in NDE research regarding the co-occurrence of an NDE and the presence of flat EEG profiles. They ask (van Lommel et al., 2001, p. 2044):

> "How could a clear consciousness outside one's body be experienced at the moment that the brain no longer functions during a period of clinical death with flat EEG?"

This question is loaded and flawed. It is flawed because there are no documented cases that clearly show that NDE occurred at the precise time that the EEG was flat. This appears to be merely assumed. In any given case a flat EEG may occur and a patient may report an NDE, but there is no evidence that these two events occur at the same time. Although some have tried to make the argument for a link (Fenwick & Fenwick, 1995; Parnia & Fenwick, 2002; Parnia et al., 2001; Sabom, 1998), others have questioned this and shown it to be untrue, at least for the cases so far investigated and followed up by independent researchers (Blackmore, 1993; Braithwaite, 1998; French, 2001). It thus becomes loaded as it assumes something to be true, which has never indeed been reliably shown to be true. As such, the question is pointless in this context. To be fair, the van Lommel group are not the only ones to make this error, but their study represents one of the latest and highest impacting studies to make the mistake. Furthermore, as already explained, because flat EEG profiles do not necessarily index complete brain inactivity, even if such cases did exist it would not provide strong or convincing evidence that these experiences are taking place when the brain was dead. Instead, these arguments seem to reflect little more than a combination of poor understandings of brain science, selective evidence, and an uncritical acceptance of anecdotal reports.

Like others before them, van Lommel et al. (2001, p. 2044) imply that the NDE is severely problematic for contemporary cognitive neuroscience:

> "NDE pushes at the limits of medical ideas about the range of human consciousness and the mind-brain relation."

However, the evidence recruited in support of this statement is deeply unconvincing. Although the dying-brain hypothesis is far from complete, it is much further from being obsolete. All scientific accounts are in constant need of revision or refutation and the dying-brain hypothesis is no exception. Indeed, this is the process of science itself. As contemporary cognitive neuroscience progresses, the dying brain hypothesis should expect serious revision – though it is unlikely this will be to the benefit of the survivalist. In addition, the Near Death Experience should be considered a legitimate area of research for neuroscience and scientists could certainly learn a great deal about the brain and cognitive function by studying such instances. However, it is difficult to see what one could learn from the paranormal survivalist position which sets out assuming the truth of that which it seeks to establish, makes additional and unnecessary assumptions, misrepresents the current state of knowledge from mainstream science, and appears less than comprehensive in its analysis of the available facts. Scientifically speaking, confidence in the survivalist position would seem, at least at present, to be misplaced.

## Conclusion

The van Lommel study was a major investigation published in a high-impact medical journal that received worldwide coverage. While methodologically speaking the study was well carried out and is a valuable contribution to the field, the interpretations of the findings offered by the authors seem fanciful at best. The logical and factual mistakes in the interpretation of the study seem common to this field of research and show no sign of dissipating. Like many before it, the van Lommel et al. study has served to do little more than propagate poor understanding of brain science - which seems common to the survivalist approach. I know of no arguments proposed by the survivalists against the dying brain hypothesis which actually characterise the dying-brain hypothesis accurately. The van Lommel et al. study was no exception. Such arguments are at least disingenuous, and at most, active attempts to avoid crucial information. If, at the very least, future survivalists attempted to characterise and represent the dying-brain hypothesis appropriately before arguing against it, they would certainly be making a unique contribution to the literature from that perspective.

It is important to be clear that van Lommel et al. provided no evidence at all that the mind or consciousness is separate from brain processes. In addition, there were no direct measures of anoxia, and no measures of neuroelectrical brain activity from their patients. Their findings are entirely consistent with contemporary neuroscience and are in line with the general dying-brain account of Near Death Experiences. As such, this study poses no challenge at all to either psychological or neuroscientific accounts for the Near Death Experience. From this we can see that their claim of the need for a new science of consciousness (which makes provision for some form of dualism) is unfounded and unnecessary. In the absence of strong evidence for survival, it appears that the position of the survivalist is still one based on faith.

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