

**\*\*\*Pre-Print Publication\*\*\***

**INVITED COMMENTARY**

**Critique of Recent Report of Electrical Activity in the Dying Human Brain**

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On May 1, the journal *Proceedings of the National Academy of Sciences (PNAS)* published an article (Xu et al., 2023) about an increase in EEG-detected electrical activity in two dying patients' brains. Because many of the media reports of this study distorted the significance and implications of these findings, we would like to offer a more reasoned perspective on this report, as we did with a comparably misinterpreted study last year (Greyson et al., 2022a, 2022b; Vicente et al., 2022).

In the present study, Xu et al. (2023) reviewed the cases of four comatose patients who died in the University of Michigan academic medical center's neuro-intensive care unit since 2014. In two of those patients, both known with epilepsy, electroencephalographic (EEG) recordings revealed a sharp burst of gamma waves in one part of the brain and interconnected electrical activity across both hemispheres. These findings have been trumpeted in respected media as explaining near-death experiences (NDEs) and continuation of consciousness after the heart stops (e.g., Reardon, 2013).

However, the findings of this study must be very carefully interpreted because the researchers reported *no evidence whatsoever* that these brain activities were correlated with conscious experiences in those two patients—and no reason to compare these results with prospective NDE studies in patients who have survived a cardiac arrest.

Even though the authors wrote that they demonstrated activity in the human brain during cardiac arrest, they in fact had *not* studied patients in cardiac arrest but, rather, had studied patients in coma as mechanical ventilation had been withdrawn. These patients had decreased oxygen, initially even with increased heart rate, although the authors did not mention cardiac electrical activity during the later dying process. They did not report on *any* brain electrical

activity when the patients had definitely died, that is, when the EKG showed terminal cardiac arrest.

In other words, the EEG changes reported in this study were associated with a *decrease in oxygen* after the withdrawal of patients' oxygen support but *not with a total lack of oxygen* as is the case in acute cardiac arrest. The surge in electrical activity was seen only in the two patients whose heart rates actually increased after mechanical ventilation was stopped.

The authors themselves acknowledged that, although they believed their findings *suggested* elevated conscious processing in these patients, no one actually observed any indication of any return of consciousness in these comatose patients. They therefore further acknowledged that the measured electrical activity may have been unrelated to conscious processes.

As we have pointed out elsewhere (e.g., Greyson, 2021a; Van Lommel, 2010), brain function has been shown in many studies with induced cardiac arrest in both human and animal models to be severely compromised during cardiac arrest: Immediately following ventricular fibrillation, cerebral blood flow ceases completely (Gopalan et al., 1999), and the resulting loss of function of the cortex results in the sudden loss of consciousness and of all body reflexes; the abolition of brainstem activity, including all brainstem reflexes such as the gag reflex and the corneal reflex resulting in fixed and dilated pupils (Van Lommel, 2010); and failure of the function of the respiratory center, located close to the brainstem, resulting in apnea—no breathing.

Under normal circumstances, a patient in cardiac arrest needs to be successfully resuscitated and defibrillated as soon as possible, so no attempt is made to measure EEG because the necessary preparations for this assessment takes far too much time. In some cases of cardiac

arrest, however, such as during surgery, EEG was part of the surgical protocol, so electrical activity of the brain was measured: Following the cardiac arrest ('no-[blood]flow'), the EEG flatlined after an average of 15 seconds and remained flat despite external resuscitation ('low [blood]flow'; Clute et al., 1990; Hossmann et al., 1973; Losasso et al., 1992; Moss et al., 1980). A persistent flatline EEG during external CPR has also been shown in animal studies (Birchner et al., 1980). During induced cardiac arrest in humans, EEG monitoring of the electrical activity of the cortex has shown that from the onset of cardiac arrest, the first ischemic changes—reduction in activity—are detected in 6.5 seconds on average, and this reduction always progresses to a flatline EEG within 10–20 seconds—15 seconds on average (Clute et al., 1990; De Vries et al., 1998; Losasso et al., 1992; Parnia et al., 2002). As long as cardiac arrest continues—that is, until cardiac function has been restored by defibrillation—the EEG remains flat (Fisher et al., 1996; Marshall et al., 2001). In tests on animals during induced cardiac arrest, auditory evoked potentials can no longer be induced, meaning that sound stimulation that normally would result in brainstem activity no longer results in any measurable activity (Brantson et al., 1984; Gua et al., 1995).

In acute heart attacks, the duration of cardiac arrest in coronary care units is always longer than 20 seconds, usually at least 60-120 seconds, and in a hospital ward or in the case of an out-of-hospital arrest it takes even much longer. Therefore, all 562 survivors of cardiac arrest in the four published prospective studies so far (Greyson, 2003; Parnia et al., 2001; Sartori, 2006; Van Lommel et al., 2001) must have had a flatline EEG. However, between 10-20% of those patients nevertheless reported NDEs, and because of the timing of occasional verifiable aspects of their experiences, it is clear that their NDEs and the accurate perceptions they included must

have happen during the period of unconsciousness rather than in the first or last seconds of cardiac arrest (Van Lommel, 2013).

We have previously published critiques of studies purporting to demonstrate that NDEs are produced by changes in brain physiology (e.g., Greyson et al., 2008; Greyson et al., 2012; Greyson et al., 2013; Greyson et al., 2022a, 2022b; Greyson & Long, 2006). However, these critiques of the details of brain physiology and their overinterpretation have missed a vital point: Whereas such studies may contribute to an understanding of the *mechanisms* through which the brain processes phenomena such as NDEs, they do not address the *cause* of NDEs.

For example, as you, the reader, read the words on this page, nerve cells in your eyes send electrical signals to the vision center of the occipital lobe of your brain and to the language center of your temporal lobe. But the electrical activity in your nerve cells did not cause the words to appear on this page; they merely enabled you to see and understand them.

Likewise, regarding the well-documented phenomena of NDEs, understanding electrical processes in the brain might elucidate the mechanisms whereby experiencers process their memories and interpretations of their NDEs. However, electrical processes do not and cannot explain what enables unconscious patients to see unexpected things in the material world accurately from an out-of-body visual perspective (Holden, 2009; Rivas et al., 2023 in press); to recognize and interact with deceased persons who, in the material world, were not yet known to have died (Greyson, 2010a); or to experience greatly enhanced cognition and perception during cardiac arrest or general anesthesia when neuroscientific models deem such complex consciousness to be impossible (Greyson et al., 2009; Greyson, 2021b; Kelly et al., 2007).

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