Invited Commentary

Recent Report of Electroencephalogram of a Dying Human Brain

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ABSTRACT: A recent journal article described continuous brainwave recording leading up to and following cardiac arrest in an elderly patient with multiple brain abnormalities. The authors wrote that increased brainwaves in the gamma frequency after his heart stopped might indicate persistent mental activity and particularly memory recall, and they speculated that this brain electrical activity might explain the life review in near-death experiences (NDEs). This speculation was subsequently hailed in the popular media as establishing a neurological explanation for NDEs. We review alternative interpretations and sources of the electrical activity recorded from this one patient's head, question the timing of the reported cessation of cardiac activity, and contrast findings from other studies of brainwave activity at death. We conclude that this one intriguing case does not provide adequate evidence of continued memory processing after death.

KEYWORDS: near-death experience; end-of-life; life review; electroencephalogram; neurophysiology

In February 2022, Vicente et al. published an article in *Frontiers in Aging Neuroscience* that described continuous electroencephalography (EEG) recording in an 87-year-old patient who unexpectedly suffered a cardiac arrest after traumatic bilateral subdural hematoma (bleeding between the brain and the skull) and status epilepticus (a prolonged seizure or series of seizures without regaining brain function). They reported that brainwaves in the delta, beta, alpha, and gamma frequency ranges were decreased, but a higher percentage of relative gamma power was observed compared to the interictal interval (the period between seizures), and cross-frequency coupling (interactions between brainwaves at differences frequency bands) revealed modulation of left-hemispheric gamma activity by alpha and theta rhythms.

The authors wrote with appropriate caution: "Given that cross-coupling between alpha and gamma activity is involved in cognitive processes and memory recall in healthy subjects, it is intriguing to speculate that such activity could support a last 'recall of life' that may take place in the near-death state" (Vicente et al., 2022, p. 9). To their credit, Vicente et al. appropriately ended their report acknowledging that the lack of any normal brain electrical activity recorded from the patient that could serve as a baseline for comparison cast doubts on any interpretation of the findings (2022, p. 9). However, subsequent mainstream media reports went far beyond the authors' modest statement that "it is intriguing to speculate" and proclaimed that this single case proved that near-death experiences (NDEs) were explained entirely by brain electrical activity persisting for several minutes after cardiac arrest. Although we agree that this case report is intriguing, we believe the data from this patient do not suggest that brain electrical activity is responsible for the life review often reported to occur in NDEs.

Gamma Oscillations: Their Presence in the Dying Process and Their Source

Vicente et al. (2022) reported that the patient displayed a temporary increase in gamma power when bilateral hemispheric activity ceased but that it declined after what the authors described as cardiac arrest. In other words, the EEG recorded from this patient did *not* show the increase in absolute gamma activity after cardiac arrest that was speculated to be responsible for (at least) the life review but, rather, showed a *reduction* in absolute gamma waves after cardiac arrest. It was only the *relative* amount of gamma that was increased compared to alpha, beta, and delta. That is, all brain activity fell off after cardiac arrest, but because brainwaves in the alpha, beta, and delta bands decreased faster than gamma, the percent of residual activity that was in the gamma range was greater relative to the other frequencies.

Regarding the source of gamma oscillations, Vicente et al. noted that increased gamma power and long-range gamma synchronization have been identified in conscious perception—but increased gamma power and synchronization have also been found across the cortex in association with a wide variety of brain circumstances (Muthukumaraswamy, 2013) ranging from ongoing pain (Schulz et al., 2015) to preparation for and execution of movements (Ulloa, 2022). To their credit, Vicente et al. listed several reasons not to place too much importance on this one patient's EEG. They noted first that the patient's traumatic brain injury and subdural hematoma may have idiosyncratically influenced brain electrical activity, including gamma waves. Second, they recognized that the patient's anesthesia-induced loss of consciousness might have influenced his gamma and other electrical activity. Third, they acknowledged that the

dissociative drugs given to the patient may have likewise played a role in his brain electrical activity, including gamma oscillations. Fourth, they noted that the anticonvulsant drugs given to control the patient's seizures may have had similar effects on his EEG. And fifth, they recognized that the patient's asphyxia (lack of oxygen) and hypercapnia (excess of carbon dioxide) are also conditions known to influence brain electrical activity, including gamma waves. These confounding variables suggest the possibility that the relative increase in gamma oscillations seen following cardiac arrest in this patient might have been associated with one or more of the specific circumstances in this patient's case rather than having been generalizable to all dying patients.

Furthermore, there are questions about whether the gamma waves recorded in the EEG entirely reflected the patient's brain activity or were at least in part measuring his muscle contractions. Contamination of EEG recordings by muscle artifact is a well-recognized problem, as electromyographic activity can masquerade as electrical activity originating in the brain, especially in the high-frequency gamma range, leading to erroneous estimates of EEG spectral power and coherence (Fitzgibbon et al., 2013; Goncharova et al., 2003; Pope et al., 2009). The peak power of the gamma oscillations recorded from this patient between burst suppression and cardiac arrest was in the upper gamma range, between 40 and 120 Hz, which is typical of muscle activity rather than signals coming from the brain; and it occurred primarily on the frontal and temporal electrodes, where muscle artifact is most often found. These data suggest that the recorded gamma waves may not have been generated from the brain after all but, rather, from frontalis and temporalis muscle activity. Spatial maps of high-frequency activity are important in identifying muscle contamination in the EEG (Muthukumaraswamy, 2013). Vincente et al.'s use of the global EEG output to calculate the relative and absolute power of the frequency bands obscures any spatial information about the source of those electrical signals.

Determination of Cardiac Arrest

Vicente et al. (2022) described electroencephalographic changes at the moment of, and following, cardiac arrest, which they defined as "the abrupt loss of heart function measured by the inability to obtain pulse activity in the ECG [electrocardiogram]" (pp. 2-3). They wrote that the patient developed a ventricular tachycardia (rapid heart rate) with apneustic respirations (abnormal breathing caused by damage to the pons or upper medulla as a result of traumatic brain injury) and a clinical cardiorespiratory arrest. Indeed, the illustration in their paper showed the patient having ventricular tachycardia, but it also showed continued heart electrical activity in the electrocardiogram past the moment in the figure that the authors specified as the time of the cardiac arrest. The patient developed a very low blood pressure due to the ventricular tachycardia, which can cause hypoxia (low flow of oxygen) in the brain, but not anoxia (absence of oxygen), as in ventricular fibrillation. Thus, at the time of the changes in brain electrical activity, the patient had not in fact experienced cardiac arrest but was continuing to show some cardiac activity on the ECG.

Prior Studies of Brain Function in Cardiac Arrest

Vicente et al. (2022) wrote that systematic research about brain activity during the process of dying has never been conducted. However, decades of clinical experience and research have established clearly that after about 8 seconds following the onset of cardiac arrest, brain activity decreases, and after about 18 seconds, it shows a flatline EEG (Clute & Levy, 1990; de Vries et al., 1998; Losasso et al., 1992; van Lommel, 2011).

Medical professionals have previously reported cases in which the electrical activity of patients' brains was measured during cardiac arrest, for example during surgery with EEG monitoring. Following the cardiac arrest, the EEG flatlined after an average of 15 seconds and remained flat despite external resuscitation (Clute & Levy, 1990; Hossmann & Kleihues, 1973; Losasso et al., 1992; Moss & Rockoff, 1980).

Monitoring of the electrical activity of the cortex through the EEG has shown that the first changes due to ischemia (inadequate blood supply) during induced cardiac arrest in humans are detected an average of 6.5 seconds after circulatory arrest. When the absence of blood flow to the brain prevents the supply of glucose and oxygen, a neuron's first symptom will be the inability to maintain its membrane potential, resulting in the loss of neuronal function (van Dijk, 2004). The acute loss of electrical and synaptic activity in neurons can be seen as the cell's inbuilt defense and energy-saving response and is called a "pilot light state." When the electrical functions of neurons cease, the remaining energy sources can be very briefly deployed for the cell's survival. With prolongation of the cerebral ischemia, progression to a flatline EEG always occurs within 10 to 20 seconds from the onset of cardiac arrest (Clute & Levy, 1990; de Vries et al., 1998; Losasso et al., 1992; Parnia & Fenwick, 2002), and the EEG remains flat during the cardiac arrest until cardiac output has been restored by defibrillation (Fischer & Hossmann, 1996; Marshall et al., 2001).

Furthermore, in a prospective research study, Norton et al. (2017) monitored continuous EEG and cardiac function in four intensive care unit (ICU) patients after life-sustaining therapy was withdrawn. In three of those four patients, prior to the cessation of arterial blood pressure and ECG activity, their EEGs showed inactivity—defined as amplitude of less than 2μV, according to recommended guidelines for EEG testing in brain death. In the fourth patient, after ECG activity had stopped, the EEG showed infrequent single delta bursts for more than 10 minutes. The residual EEG activity reported in this study was insufficient to produce the kind of coherent, complex, conscious experience characteristic of NDEs (Colombo et al., 2019).

In summary, following what Vicente et al. (2022) described as cardiac arrest in this one patient, the EEG showed that brain activity decreased, with gamma activity decreasing more slowly than alpha, beta, and delta activity; the detected gamma activity may actually have been a reflection of muscular activity rather than brain activity; the point at which the authors had specified cardiac arrest actually was followed by further heart activity; and extensive research on numerous other patients has indicated that following the cessation of heart activity, brain activity—especially of a nature capable of generating the kind of coherent, complex, conscious experience characteristic of NDEs—discontinues within about 20 seconds. For all these reasons, the recent paper by Vicente et al. (2022) is, as the authors wrote, intriguing enough to stimulate speculation, but not evidential enough to suggest a neurological basis for NDEs.

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