



Review article

Near death experiences in cardiac arrest: visions of a dying brain
or visions of a new science of consciousnessSam Parnia^{a,*}, Peter Fenwick^b^a Department of Medical Specialities, Southampton General Hospital, Tremona Road, Southampton SO 16 6YD, UK^b The Institute of Psychiatry, London, UK

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Abstract

Very little is known about the dying process and in particular the state of the human mind at the end of life. Cardiac arrest is the final step in the dying process irrespective of cause, and is also the closest physiological model of the dying process. Recent studies in cardiac arrest survivors have indicated that although the majority of cardiac arrest survivors have no memory recall from the event, nevertheless approximately 10% develop memories that are consistent with typical near death experiences. These include an ability to 'see' and recall specific detailed descriptions of the resuscitation, as verified by resuscitation staff. Many studies in humans and animals have indicated that brain function ceases during cardiac arrest, thus raising the question of how such lucid, well-structured thought processes with reasoning and memory formation can occur at such a time. This has led to much interest as regards the potential implications for the study of consciousness and its relationship with the brain, which still remains an enigma. In this article, we will review published research examining brain physiology and function during cardiac arrest as well as its potential relationship with near death experiences during this time. Finally, we will explore the contribution that near death experiences during cardiac arrest may make to the wider understanding of human consciousness. © 2002 Elsevier Science Ireland Ltd. All rights reserved.

Keywords: Cardiac arrest; Near death experiences; Dying brain; Visions of a new science

1. Introduction

The dying process, and in particular the subjective experience of dying is an area of which very little is known and, until now, has been largely ignored by scientific investigation. However, recent studies in cardiac arrest patients have begun to shed some light on the likely experiences that we may have at the end of our lives. Certainly from a clinical point of view, the question of what our patients' experience when they face death, is a very real and important issue with everyday practical relevance. Knowledge of this process may help doctors and other carers deal with the anxieties of patients who face death.

In this review we will examine the evidence related to the state of the human mind at the point of death, as well as its potential contribution in the wider attempts to understand the nature of human consciousness.

The advent of modern resuscitation techniques has enabled many critically ill patients who would otherwise have died, to be resuscitated successfully. Some of these survivors have recalled unusual experiences from their period of critical illness. In 1975 an American doctor, Raymond Moody, published a best selling book in which he collected the experiences of 150 people who had been close to death [1]. Recurring features in their accounts included seeing a tunnel, a bright light, deceased relatives, a mystical being, entering a new domain, reaching a point of no return, a review of their lives as well as 'out of body experiences' in which people described a feeling of separation from their

* Corresponding author. Tel.: +44-2380-777222x3308; fax: +44-2380-701771.

E-mail address: parnis@soton.ac.uk (S. Parnia).

bodies and being able to watch themselves as if from a vantage point above. These recurring features have been termed near death experiences (NDEs). Although there have been some reports of unpleasant, ‘nightmarish’ experiences, the majority who experience NDEs have described them as very pleasant and say that they are left with no fear of death and a more spiritual view of life [1]. The experiences are not confined to adults, and have also been reported in children, who have often been too young to have any real concept of death or an afterlife [2–5]. Although reported in most cultures, independent of any religious faith or after life, there are some cultural differences in the content of the experience [6–8], and the interpretation of the experience may reflect religious belief [9]. The phenomenon is relatively widespread; a Gallup survey in the US in the early 1980s showed that NDEs have been reported in approximately 4% of people who have been close to death [10].

2. The proposed mechanisms for the causation of NDEs

The most widely accepted scientific view regarding the causation of NDEs, is that they are either due to a disturbance of brain chemistry occurring during the dying process or are a psychological response to the perceived threat of death [11,12]. Many physiological and pharmacological cerebral mediators have been proposed to account for NDEs. These include endorphins [13,14], cerebral hypoxia [15], hypercarbia [11,16], various drugs, in particular hallucinogenic agents such as ketamine and phencyclidine [17], the NMDA receptor [17], serotonin pathways [18], activation of the limbic system [19,20], and temporal lobe anoxic seizures [21]. Thus NDEs are proposed to arise as a form of complex hallucinatory process in response to either an alteration in cerebral mediators or even possibly as a psychological form of ‘wish fulfilment’ in response to the perceived threat of death [22].

3. Cardiac arrest: the closest model to the dying process

Most of the collected data on NDEs have been obtained from retrospective cases, that have been reported by patients, often many years after a critical illness or a close encounter with death such as an accident. The authors have examined more than 750 [23] retrospective NDE cases, and have found that in most cases it is difficult to correlate the physiological state of the patients with their experiences to ascertain exactly how close to death they had been. Physiologically and clinically, a cardiac arrest is the closest state

to the dying process [24], and in objective terms therefore, experiences arising during a cardiac arrest shed most light on the state of the human mind at the point of death. Cardiac arrest is also the most appropriate condition to study the dying process as, irrespective of cause, it is the inevitable final stage before death. During a cardiac arrest the clinical criteria of death are always reached for a variable length of time ranging from a few seconds to tens of minutes. By definition, patients have at least two out of the three criteria of clinical death (no cardiac output, no respiration) and also usually develop the third (fixed dilated pupils) rapidly with the loss of brainstem function¹.

4. Experimental studies of NDE in cardiac arrest

Recently, a number of independent studies have confirmed reports of NDEs occurring in cardiac arrests. A 1-year prospective study of cardiac arrest survivors carried out by the authors showed a 6% incidence of NDEs [24]. This study, which was based upon interviewed accounts obtained from in hospital cardiac arrest survivors within 1 week of the arrest did not find any evidence to support the role of drugs, hypoxia, hypercarbia or electrolyte disturbances in the causation of NDE. This was a small study with a total of four out of 63 cardiac arrest survivors reporting NDEs. In a much larger study just completed in Holland, 344 cardiac arrest survivors from 10 hospitals were interviewed over a 2-year period, and 41 or 12% reported a core NDE [25]. Patients were followed up for a further 8 years following the arrest and reported less fear of death and a more spiritual outlook on life. The authors of both studies have raised questions regarding how lucid, well structured thought processes, together with long term memory formation that is characteristic of NDEs can arise during a cardiac arrest when cerebral function is impaired and would not be expected to support lucid thought processes and memory formation. This can be better understood by examining cerebral physiology during and after cardiac arrest.

5. Cerebral physiology during cardiac arrest

There is extensive literature available on the physiology of cardiac arrest obtained from both human and

¹ It must be pointed that atropine and epinephrine (adrenaline) which are often administered in cardiac arrests may cause dilated pupils that appear to be fixed. However in a cardiac arrest, brainstem activity is rapidly lost which would lead to the loss of pupillary reflexes irrespective of any administered drugs. The administration of morphine in patients with myocardial infarction may make the interpretation of pupil size more difficult.

animal studies. Immediately following a cardiac arrest due to the cessation of the heartbeat, the blood pressure drops to immeasurable levels. During properly performed chest compressions, the systolic pressure may rise to 60–80 mmHg, but despite this, the diastolic values and hence the mean arterial pressure still remains low. [26] In one animal study the mean arterial pressure (MAP) following cardiac arrest was recorded to fall to less than 30 mmHg (lowest measurable value in this study) and rose only marginally to approximately 30–40 mmHg during chest compressions [27]. The use of vasopressors such as epinephrine and vasopressin has been shown to increase blood pressure, and cardiac and cerebral perfusion pressure compared with chest compressions alone. In one study carried out in ten humans with prehospital cardiac arrest it was shown that during CPR epinephrine led to an increase in systolic arterial blood pressure from 47 (± 5) before its administration, to 69 (± 7), 74 (± 8) and 85 (± 8) mmHg with increasing doses of 1, 3 and 5 mg. The diastolic pressure also increased from 18 (± 2) to 27(± 3), 25 (± 4) and 36(± 6) mmHg respectively but still remained relatively low compared with pre cardiac arrest levels [28]. Coronary and cerebral perfusion rely on adequate diastolic and mean pressures and therefore the pressures generated during cardiac arrest with chest compressions combined with epinephrine, although better than no intervention, are still generally too low for adequate perfusion. Other studies in humans have shown similar changes in blood pressure during the administration of epinephrine and chest compressions [29]. Cerebral perfusion pressure (CPP) is determined by the difference between the mean arterial pressure and the intracranial pressure (MAP–ICP). It has been shown that the more prolonged the cardiac arrest, the higher the ICP rises and hence a higher MAP is needed to maintain CPP. In one animal study, cardiac arrest was induced by ventricular fibrillation in 14 cats. Following 15 min of cardiac arrest they were then resuscitated using chest compressions together with the administration of high dose (0.2 mg/kg) epinephrine for 4 min prior to defibrillation. After restoration of spontaneous circulation (ROSC) mean arterial pressure stabilised at 80–100 mmHg with epinephrine. Prior to the induction of cardiac arrest the mean arterial pressure was 107 (± 26). Following 2 and 4 min of chest compressions and the administration of high dose epinephrine the mean arterial pressure was 77 (± 20) and 65 (± 18) mmHg, respectively. There was also a corresponding cerebral perfusion pressure drop from a prearrest value of 101 (± 26), to 37 (± 20) and 31(± 20) mmHg, respectively [30]. In clinical practice it is generally accepted that a CPP of 70 mmHg is needed for adequate cerebral perfusion. These relatively low mean arterial blood pressures are maintained until the resumption of cardiac output despite conventional cardiopulmonary resuscitation [27–29,31].

In clinical practice the electroencephalogram is often used to assess cerebral ischaemia during procedures such as cardiac and neurosurgery. EEG has also been used in assessing cerebral function during cardiac arrest in animals and humans. The data from humans is largely limited to those obtained during defibrillation threshold testing at defibrillator implantation or individual case reports from patients, who were connected to an EEG during their cardiac arrest. Concurrent EEG monitoring during a cardiac arrest has shown an initial slowing of the EEG waves which then progress to an isoelectric (flat) line within approximately 10–20 s and remain flat during the cardiac arrest until the resumption of cardiac output in patients after early defibrillation [31]. In cases of prolonged cardiac arrest EEG activity may not return for many hours after cardiac output has been returned. In one study of cardiac arrest in dogs, 15 min of VF cardiac arrest was induced in three groups of dogs. At the end of the 15 min dogs in the first group were then treated with conventional cardiopulmonary resuscitation including closed chest compressions, epinephrine, sodium bicarbonate, and defibrillation for 3 min before being switched to femoral vein–femoral artery bypass (F–F bypass). Following the initial 15 min of VF cardiac arrest dogs in group 2 were given 8 min of conventional cardiopulmonary resuscitation including defibrillation and epinephrine, before being connected to F–F bypass. In this group cerebral and myocardial blood flow was measured during the resuscitation period. In group 3, F–F bypass was commenced after the initial 15 min cardiac arrest period together with the other measures including defibrillation and epinephrine.

In group 1 and 3 EEG measurements were made throughout the resuscitation period. In group 1 the EEG flattened 21.1 ± 5.7 s ($n = 10$) after VF and remained flat until 90.0 ± 24.7 min after the start of resuscitation when bursts of slow waves (burst and suppression) appeared. During the observation period, interrupted slow waves became continuous waves at 130.7 ± 28.1 min after beginning resuscitation. In group 3, EEG flattened after 17.4 ± 3.0 s after the occurrence of VF. Burst suppression appeared 62.8 ± 11.6 min and continuous waves appeared at 145.6 ± 27.5 min after the beginning of bypass. In group 2, local cerebral blood flow (CBF) decreased rapidly to zero after VF. Myocardial blood flow also decreased but took approximately 1–2 min to reach zero. After 15 min of cardiac arrest no appreciable local CBF could be restored in the following 8 min by cardiopulmonary resuscitation including closed chest compression, ventilation and the administration of resuscitative drugs. Blood flow returned to pre VF levels following the initiation of F–F bypass [32]. Therefore, during cardiac arrest cerebral blood flow is severely impaired which leads to a lack of electrophysiological activity in the cortex. This is made

worse as the period from initial ischaemia to adequate resuscitation is increased. Studies in animals have demonstrated that an absence of cortical activity as measured by EEG correlates with an absence or reduction in activity of the deep brain structures as measured by in-dwelling electrodes [33,34]. As a result of these processes during a cardiac arrest cerebral function and therefore consciousness are lost.

6. Cerebral physiology after cardiac arrest

Under normal circumstances the brain receives 15% of the cardiac output (750 ml/min). The grey matter flow is 60–70 ml/min and the white matter flow is 25 ml/min. Oxygen and glucose are needed to maintain cellular integrity (40–50% of total CBF) and electrophysiological activity (50–60% of CBF) [35]. Although, there is minimal blood flow to the brain during a cardiac arrest, it has also been shown that local cerebral blood perfusion is also severely impaired after the restoration of an adequate blood pressure and gross cerebral blood flow rate. This is due to local increases in vasoconstriction possibly brought about by an imbalance in the local production of vasoconstrictors and vasodilators, explaining the observed lack of EEG electrical activity despite the maintenance of adequate blood pressure during the recovery phase of cardiac arrest. In the above experiment EEG activity did not begin to recover until approximately 63 min after beginning F–F bypass. Immediately after resuscitation there is a period of multifocal no-reflow (phase 1). This is followed by transient global hyperaemia lasting 15–30 min (phase 2). Thereafter cerebral blood flow becomes severely reduced while cerebral metabolic rate of oxygen gradually recovers. This is termed the delayed hypoperfusion phase (phase 3). After 24 h, cerebral blood flow and metabolism may be restored, remain low or there may be secondary hyperaemia (phase 4). Multifocal no-reflow is a phenomenon observed in animals following recovery from cardiac arrest, in which, despite the restoration of adequate blood pressure, multiple areas of the brain develop perfusion defects that range from a pin hole to up to 95% of the brain. The cerebral hypoperfusion phase is characterised by a decrease in cerebral blood flow to about 50% or less of normal, but the reduction in flow is not homogenous in the brain with certain areas having more reduction in flow compared with other areas. In one animal study in rats it was shown that the flow rates in the frontal cortex were 10–15% of control compared with 70–90% in the cerebellum. The main factor responsible for this reduced cerebral blood flow following a cardiac arrest is the initial period of ischaemia prior to adequate resuscitation [35].

From a clinical point of view these observations are supported by the loss of brain stem reflexes such as the gag reflex that allows patients to be intubated easily. These also indicate a loss of brainstem function and perfusion which may result from depression of the reticular activating system (RAS) that normally drives cortical function via the thalamus.

7. Interpreting NDE in cardiac arrest

The occurrence of lucid, well structured thought processes together with reasoning, attention and memory recall of specific events during a cardiac arrest (NDE) raise a number of interesting and perplexing questions regarding how such experiences could arise. These experiences appear to be occurring at a time when cerebral function can be described at best as severely impaired, and at worst absent. Although, under other clinical circumstances in which the brain is still functioning, it may be possible to argue that the experiences may arise as a hallucination in response to various chemical changes in the brain, this becomes far more difficult during a cardiac arrest. NDE in cardiac arrest appear different to hallucinations arising from metabolic or physiological alterations, in that they appear to occur in a non-functioning cortex, whereas hallucinations occur in a functioning cortex. Therefore, it is difficult to apply the same arguments for their occurrence. In addition cerebral localisation studies have indicated that thought processes are mediated through a number of different cortical areas, rather than single areas of the brain. Therefore a globally disordered brain would not be expected to produce lucid thought processes. From a clinical point of view any acute alteration in cerebral physiology such as occurring in hypoxia, hypercarbia, metabolic, and drug induced disturbances and seizures leads to disorganised and compromised cerebral function [36]. Furthermore, as already described, any reduction in cerebral blood flow leads to impaired attention and higher cerebral function. A recent study by Marshall and co workers has demonstrated that deterioration in higher cerebral function correlates with reduction in the levels of cerebral blood flow, and that even relatively minor reductions in blood flow leads to impaired attention [37]. NDEs in cardiac arrest are clearly not confusional and in fact indicate heightened awareness, attention and consciousness at a time when consciousness and memory formation would not be expected to occur.

An alternative explanation is that NDEs reported from cardiac arrests, may actually be arising at a time when consciousness is either being lost, or regained, rather than during the actual cardiac arrest period itself. Experiments during simple fainting episodes have shown that, experiences arising during loss of con-

sciousness occur in conjunction with mental experiences at the beginning of the episode [38]. This is not seen classically in NDEs. The EEG during fainting show a gradual slowing of the cerebral rhythms with the appearance of delta activity before finally, in a minority of cases, the EEG becoming flat [39]. In cardiac arrest, the process is accelerated, with the EEG showing changes within a few seconds [40].

Any cerebral insult leads to a period of both anterograde and retrograde amnesia [41,42]. In fact memory is a very sensitive indicator of brain injury and the length of amnesia before and after unconsciousness is an indicator of the severity of the injury [43]. Therefore, events that occur just prior to or just after loss of consciousness would not be expected to be recalled. Recovery following a cerebral insult is confusional [41,42]. As has been described above, cerebral function as indicated by EEG has, in many cases been shown not to return until many minutes or even a few hours after successful resuscitation. Despite these observations it can still be argued that the occurrence of some of the features of an NDE such as seeing a light or a tunnel potentially may occur during the recovery phase following a cardiac arrest, with the patient thinking that the experiences had occurred during the actual period itself. However, anecdotal reports of patients being able to ‘see’ and recall detailed events occurring during the actual cardiac arrest, such as specific details relating to the resuscitation period verified by hospital staff, simply cannot be explained in this way. For this memory to take place, a form of consciousness would need to be present during the actual cardiac arrest itself.

8. NDE in cardiac arrest: clinical conclusions and the potential contribution to the wider debate on the nature of human consciousness

Patient’s experiences from cardiac arrests have begun to shed some light on the probable state of the human mind at the end of life. Although, as might be expected from studies of cerebral function during cardiac arrest, the majority of people who survive cardiac arrest recall no experiences from the period of unconsciousness, there is a significant proportion who recall unusual experiences that are characteristic of NDEs. These experiences are generally pleasant and have positive life changing effects on the individual [25]. The majority of patients with NDEs find it difficult to discuss their experiences with caregivers as well as family and close friends. Physicians and other caregivers therefore should be aware of these phenomena and advise patients accordingly. Interestingly, there are a small proportion of cardiac arrest survivors who have reported being conscious and aware of events during resuscita-

tion and have recalled ‘seeing’ specific details that would not have been known to them. These experiences have been recalled, while cerebral function has, through many studies, been shown to be severely compromised and electrical activity in both the cerebral cortex and the deeper structures of the brain have been shown to be absent. From a scientific point of view, the occurrence of these experiences would therefore seem highly improbable and paradoxical. However, the fact that they do occur, raises some questions regarding our current views on the nature of human consciousness and its relationship with the brain. Editorials in recent years, including some in ‘*Scientific American*’ and ‘*Nature Neuroscience*’ have highlighted the difficulties faced by cognitive neuroscience in attempting to answer questions regarding the nature and the mechanism by which subjective experiences and sense of consciousness may arise through cellular processes [44–46]. Traditionally, it has been argued that thoughts or consciousness; are produced by the interaction of large groups of neurones or neural networks [47]. Evidence for this view has come from the clinical observation that specific changes in function such as personality or memory are associated with specific cerebral lesions such as those that occur after head injury. This is further supported by the results of cerebral localisation studies using functional MRI and PET scanning, in which specific areas of the brain have been shown to become metabolically active in response to a thought or feeling [48]. However, those studies, although providing evidence for the role of neuronal networks as an intermediary for the manifestation of thoughts, do not necessarily imply that those cells also produce the thoughts [49]. Although, undoubtedly complex these networks nevertheless are composed of individual neurones connected via synapses and various neurotransmitters that lead to the generation of action potentials across the cell membrane. With our current scientific understanding a neurobiological mechanism to explain how cerebral chemical and electrical processes may lead to subjective experiences has yet to be discovered [44,45]. Direct evidence of how neurones or neural circuits can produce the subjective essence of the mind and thoughts is currently lacking and provides one of the biggest challenges to neuroscience [50].

Alternative scientific views for the causation of consciousness and subjective phenomenon have, therefore, been proposed. These range from the view that consciousness may arise from ‘quantum’ processes within neuronal microtubules [51], to consciousness being a form of ‘morphic resonance’ [52] or the possibility that the mind or consciousness may actually be a fundamental scientific entity in its own right irreducible to anything more basic [50,53]. This concept has been proposed to be similar to the discovery of electromagnetic phenomenon in the 19th century, or quantum mechanics in the 20th century, both of which were

inexplicable in terms of previously known principles and were introduced as fundamental entities in their own right [50]. An extension of this has been the view that contrary to popular perception, what has traditionally been perceived as spirituality, is therefore also an objective branch of knowledge with its own laws, theorems and axioms [53].

If the occurrence of NDEs during a cardiac arrest, when the mind (the collection of all our thoughts, feelings and emotions) and consciousness (self awareness) appear to continue at a time when the brain is non-functional and clinical criteria of death have been reached, can be proven objectively through large studies, then this will lend some support to this view. Although at present, this remains a mere possibility, if investigated through appropriate studies it may have significant implications not only for medicine but also for society as a whole. Such studies are currently possible, and it has been proposed to test the claims of 'consciousness' and being able to 'see' during cardiac arrest objectively by use of hidden targets that are only visible from a vantage point above. Although, at first these suggestions may sound rather unconventional, the study of consciousness has itself for many years been thought of as unconventional, but has now become a significant point of debate in neuroscience. Therefore, a new way of thinking may be needed to provide an insight into understanding this intriguing, yet largely undiscovered area of science.

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Portuguese Abstract and Keywords

Sabe-se pouco acerca do processo de morte e em particular sobre o estado da mente humana no fim da vida. A paragem cardíaca é o passo final no processo de morte independentemente da causa e é também o modelo fisiológico mais próximo do processo de morte. Estudos recentes em sobreviventes de paragem cardíaca indicam que embora a maioria dos sobreviventes de paragem cardíaca não tenham nenhuma memória do evento, contudo cerca de 10% desenvolvem memórias que são consistentes com experiências típicas de “quase morte”. Estas incluem a capacidade de “ver” e lembrar descrições detalhadas específicas da reanimação, que estão de acordo com o que foi verificado pela equipa de reanimação. Muitos estudos em humanos e animais têm indicado que a função cerebral cessa durante a paragem cardíaca, levantando a questão de como é que processos tão lúcidos e bem estruturados, com compreensão e formação de memória, podem ocorrer nesta altura. Estas situações despertam grande interesse já que têm implicações sobre a relação da consciência com o cérebro, que ainda permanecem um enigma. Neste artigo os autores revêm pesquisas publicadas examinando a fisiologia do cérebro e a sua função durante a paragem cardíaca, bem como a sua potencial relação com experiências de “quase morte” durante este período. Finalmente, exploramos a contribuição que as experiências de “quase morte”, durante a paragem cardíaca, podem ter para a melhor compreensão da consciência humana.

Palavras chave: Paragem cardíaca; Experiências de “quase morte”; Cérebro a morrer; Visões de uma nova ciência

Spanish Abstract and Keywords

Se sabe muy poco acerca del proceso de muerte, y en particular del estado de la mente al final de la vida. El paro cardíaco, sin importar su causa, es el paso final y el modelo fisiológico más cercano del proceso de morir. Estudios recientes en sobrevivientes de paro cardíaco, han indicado que aunque la mayoría de ellos no tiene recuerdos del evento, cerca del 10% desarrolla recuerdos que son consistentes con las experiencias ‘cercanas a la muerte’. Estos incluyen una capacidad para ver y recordar ciertos detalles de su resucitación, coincidentes con lo ocurrido. Muchos estudios en humanos y en animales han indicado que la función cerebral se detiene durante el paro cardíaco, haciendo que nos preguntemos cómo pueden ocurrir en ese momento procesos de pensamiento tan lúcidos, bien estructurados, con razonamiento y formación de memoria. Esto ha llevado a un creciente interés por las implicancias potenciales del estudio de la conciencia y su relación con el cerebro, hecho que sigue siendo un enigma. En este artículo revisaremos la investigación publicada examinando la fisiología y función cerebral durante el paro cardíaco y su potencial relación con experiencias ‘cercanas a la muerte’ durante este evento. Finalmente, exploraremos la contribución que las experiencias ‘cercanas a la muerte’ pueden hacer para mejorar la comprensión de la conciencia humana durante el paro cardíaco.

Palabras clave: Experiencias cercanas a la muerte; Paro cardíaco; Cérebro agónico; Visiones de una ciencia