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PRACTICAL PEARL

Prolonged Retention of Awareness During Cardiopulmonary Resuscitation for Asystolic Cardiac Arrest

Shailesh Bihari · Venkatakrishna Rajajee

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Abstract
Objective To describe high level of awareness in a patient undergoing cardiopulmonary resuscitation for an asystolic cardiac arrest and review the literature regarding this phenomenon.
Methods This is a case report of a patient admitted to the Intensive Care Unit who suffered an asystolic cardiac arrest. We reviewed MEDLINE using the terms "awareness," "consciousness," "cerebral perfusion," "sedation," "analgesia," "termination," "cessation," and "cardiopulmonary resuscitation."
Results A 57-year-old man with renal failure suffered asystolic cardiac arrest. He was awake and alert during cardiopulmonary resuscitation (CPR). Cardiac arrest was confirmed by echocardiogram and invasive arterial monitoring. He briskly localized and consistently followed simple commands while chest compressions were in progress before becoming unresponsive and dying after a 3-h resuscitative effort. No sedation/analgesia was used. There are few reports in the literature describing similar events.
Conclusion It is possible to retain a high level of awareness following cardiac arrest, particularly with effective CPR. Recognition of this situation when it occurs allows appropriate decisions to be made regarding the use of sedation and the length of resuscitative efforts.

Keywords Cardiopulmonary resuscitation · Cardiac arrest · Awareness · Consciousness · Sedation

Introduction

Unresponsiveness is thought to almost universally occur at the time of cardiac arrest, as a consequence of a precipitous drop in cerebral perfusion. The first action that Basic Life Support (BLS) and Advanced Life Support (ALS) trainees are taught to perform is to check for unresponsiveness. It is theoretically possible, however, to maintain awareness following cardiac arrest if cerebral perfusion is maintained by the use of highly effective chest compressions and in the presence of adequate oxygenation. Reports of such retained awareness during cardiac arrest in the literature are sparse [1, 2].

Case Report

A 57-year-old man with no significant past medical history was admitted to the general medical ward with complaints of tingling in both hands, loss of weight, vomiting, and

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S. Bihari · V. Rajajee
Critical Care Unit, Apollo Hospitals, No. 21, Greams Lane, Chennai 600 006, Tamilnadu, India

Present Address:
S. Bihari
Critical Care Unit, Flinders Medical Center, Bedford PK, Adelaide, SA, Australia

Present Address:
V. Rajajee
Departments of Neurology and Neurosurgery, University of Michigan Medical Center, Ann Arbor, MI 48109-0338, USA

V. Rajajee (✉)
Department of Neurosurgery, 3552 Taubman Health Care Center, University of Michigan, 1500 E. Medical Center Drive, SPC 5338, Ann Arbor, MI 48109-0338, USA
e-mail: vrajajee@yahoo.com

joint pain for 30 days. He was discovered to have proteinuria and elevated blood urea nitrogen (BUN) (88 mg/dl), and Creatinine (3.6 mg/dl). He was thought to have an acute worsening of chronic renal failure and a series of investigations were planned to determine the etiology. On the second day of his hospital stay, a renal biopsy was performed. Approximately 10 h after the renal biopsy, he developed acute confusion and agitation and was moved to the ICU for better monitoring and further investigation. On arrival he was found to be awake, disoriented, and agitated, but able to communicate in short sentences. His BP was 120/70, his heart rate was 64, respiratory rate was 28, and the continuous ECG monitor revealed normal sinus rhythm with a rate of 64 per minute with tall T waves. Pulse oximetry revealed a SpO2 of 97%. Cardiac, respiratory, and abdominal examinations were unremarkable. Shortly after drawing of blood for investigations, the cardiac rythm was noticed to become slow, followed quickly by a broad complex bradycardia (20/mt) leading to asystole (confirmed in two leads). No carotid pulse was palpable. At this time he was briefly unresponsive to stimuli and CPR was initiated immediately as part of the standard management algorithm for asystolic cardiac arrest [3, 4]. Chest compressions were initiated alternating with manual bag-mask ventilation in a 30:2 ratio. The patient then appeared awake and alert and was moving both his arms very purposefully (brisk localization). Epinephrine 1 mg (repeated every 3 min) and Atropine 1 mg IV (repeated every 3 min to a total of 3 mg) were administered. The paddles of a defibrillator also registered asystole. Following three cycles of chest compressions and bag ventilation he was noticed to still be in asystole with the absence of pulse in both carotid and femoral arteries confirmed by three different physicians. At this time endotracheal intubation was performed. During direct laryngoscopy, the patient was noticed to react violently and attempted to pull the laryngoscope away as chest compressions were in progress. The team leader handling the arrest had never previously encountered this situation in the context of a cardiac arrest and was concerned about the potential hemodynamic/neurological consequences of administering sedation or neuromuscular blockade at this time. No sedative or analgesic drugs were administered and the patient's arms were manually immobilized while the tracheal tube was inserted. Resuscitation continued with concomitant chest compressions and manual ventilation following placement of the definitive airway and rhythm and pulse checks were performed every 2–3 min. At all times the rhythm was asystole and no pulse was palpable. Four different individuals were performing cardiac compressions, in cycles of 100 compressions each. A transcutaneous pacemaker was unable to elicit any effective contractions. During this time the patient continued to track the surrounding health care providers with his eyes and attempted to localize and resist chest compressions. Again, no sedation or analgesia was administered and the patient was manually immobilized. A femoral pulse was palpable during CPR. An arterial catheter was emergently placed in the right femoral artery and central venous access obtained with a triple lumen catheter in the right femoral vein. Efficacy of cardiac compressions was confirmed by the excellent waveform seen on the arterial line; at all times a mean arterial pressure (MAP) of greater than 50 mmHg was maintained during compressions. No arterial tracing was seen when compressions were held. Breaks in chest compressions lasted <10 s and no attempt at a neurological assessment was made specifically during these brief periods initially, it is unclear if he was just as alert during these breaks in compressions. The patient was given 50 mmol sodium bicarbonate, 30 cc 10% calcium gluconate, and 100 cc 50% dextrose with 10 units insulin IV for a presumed hyperkalemic cardiac arrest in view of his renal failure. The results of investigations drawn on arrival to the ICU (prior to cardiac arrest) were now available—he was found to have a potassium level of 7.2 meq/dl and Creatinine of 6.0 mg/dl. A 2D Echo was then performed using 20–30 s breaks in chest compressions. This revealed only minimal fluid in the pericardial sac and confirmed the complete absence of any spontaneous cardiac contractions while compressions were held. One and a half hours after initiating chest compressions he remained conscious, opening eyes to call and moving both hands appropriately to command. At this time he was noticed to become unresponsive during breaks in chest compressions. Following 2 h and 10 min of resuscitation efforts he became completely unresponsive and no further motor response or ocular movement was seen. The pupils were noticed to dilate to 6 mm bilaterally and become unreactive. Corneal and gag reflexes were also no longer present. The average MAP during compressions remained >50 mmHg. Three hours following initiation of resuscitation efforts he remained pulse less and asystolic. Resuscitation efforts were stopped and the patient declared dead 3 h and 15 min after initiation of CPR. His renal biopsy was later found to suggest early amyloidosis.

Discussion

A state of unresponsiveness usually rapidly follows cardiopulmonary arrest because of a precipitous fall in cerebral perfusion pressure (CPP). Perhaps as a consequence, traditional ALS and BLS teaching does not address the possibility of a victim of a cardiac arrest being awake and aware during the resuscitation [3, 4]. We have documented here our experience with this uncommon

phenomenon, and address some of the important issues that arise in the conduct of cardiopulmonary resuscitation (CPR) in such a patient. There are other reports in the literature of retained awareness during CPR [1, 2] (Table 1). One report describes a patient in whom awareness was maintained, presumably because of effective CPR while using a mechanical compression device, for a period of time following which, as happened in our case, the patient lapsed into unresponsiveness and could not be resuscitated [1]. The authors document a large systolic aortic to right atrial pressure gradient in this patient, suggesting that direct cardiac compression while using the mechanical device played a direct role in maintaining adequate perfusion. Small doses of morphine and diazepam were used during resuscitation. Another study describes the occurrence of agitation and purposeful movements following the initiation of Active Compression Decompression-CPR (ACD-CPR) in a patient who had suffered cardiac arrest [2]. The patient required physical restraints, sedation and paralysis to facilitate endotracheal intubation and subsequent management. The eventual loss of neurological response in these patients, despite continuing CPR, might conceivably be a result of ischemic neurons, barely functioning with the minimal cerebral perfusion provided by chest compressions, finally progressing to irreversible dysfunction and death in the absence of a full return of perfusion. Regions of the brain that receive blood flow between 10 and 20 ml/100 g/min are generally considered to be in a state of reversible ischemia, and are expected to progress to cell death (infarction) if normal blood flow is not restored [5]. While it is unclear why some patients retain greater neurological function than others during CPR, it is possible that the resuscitative efforts for individuals with retained consciousness during CPR were initiated much earlier by trained professionals (such as with many in-hospital cardiac arrests) and that the CPR they received was more effective, although no objective data exists to support this hypothesis. The efficacy of an individual patient's cerebral autoregulation in response to a decreased CPP may also be a factor in determining the level of cerebral oxygenation and therefore the level of awareness in an individual patient undergoing CPR [6]. In one study correlating CPPs with brain tissue oxygenation, the percentage of patients showing signs of cerebral hypoxia (PtiO2 $<$ 15 mmHg) was 50%, 25% and 10%, respectively, with CPPs of $<$60 mmHg, 60–70 mmHg, and $>$70 mmHg [7]. An individual with superior autoregulation may achieve better cerebral oxygenation, and therefore greater retention of neurological function, with a relatively low CPP compared to an individual with less effective autoregulation. It is also possible that the ischemic threshold varies somewhat between individuals [8].

The strength of our report lies in the fact that we had objective evidence of cardiac arrest, in the form of both echocardiography revealing a complete absence of cardiac contractions in the absence of chest compressions as well as adequately functioning invasive arterial monitoring that revealed no flow without compressions. This is significant as simple palpation for a pulse can easily be erroneous in definitively diagnosing a complete absence of systemic (and hence cerebral) perfusion [9, 10] and "asystole" on a cardiac monitor is sometimes the consequence of technical problems, such as misplaced or malfunctioning leads, rather than true asystole. Reports that base a diagnosis of cardiac arrest on the absence of a palpable pulse alone may therefore actually be describing patients with a greatly decreased, but present, spontaneous circulation and cerebral perfusion. Also, our patient had an unquestionably high level of awareness during compressions—he clearly lifted his arm to command and tracked to auditory stimuli. This is definitely retained awareness, as against reports of motor responses or versive eye movements that may represent "last gasp" cerebral activity after cardiac arrest.

The possibility of retained awareness during CPR raises several issues. The first is the suffering a person must almost certainly experience during resuscitative efforts such as aggressive chest compressions, endotracheal intubation, and manual bag ventilation. Awareness among healthcare providers of the possibility that patient awareness might be retained during CPR may facilitate the recognition of such a situation when it is encountered. This may result in the appropriate administration of analgesia and possibly sedation. Our experience demonstrates that experienced healthcare providers may be hesitant to administer sedation or analgesia under these circumstances, even when the patient is obviously in extreme discomfort (such as during endotracheal intubation). Other authors have described the use of sedation and analgesia during cardiac arrest [1, 2, 11]. While concerns about the adverse effects of sedative and analgesic medications in such a situation (hemodynamic depression, obscuration of neurological assessment) may have merit, it is worthwhile to consider that a patient in this situation is quite unlikely to survive despite maximal resuscitative efforts. To our knowledge no published report of awareness during CPR has documented patient survival. Alleviating pain and suffering at this likely terminal stage should outweigh concerns about potential side effects. A related issue would be when to stop resuscitative efforts in such a situation. It is clearly difficult for a resuscitation team to cease efforts when the patient appears to be awake and aware, although there is no objective evidence to date that such retained awareness increases the likelihood of survival after prolonged CPR. Our team chose to cease efforts once there was absence of any neurological response (including brainstem reflexes such as pupillary and corneal reflex) for

Table 1 Case reports of retained awareness during CPR

Case report	Type of arrest and etiology	Best neurological response seen	Mechanism of chest compression	Blood pressure generated by compressions	Duration of resuscitation efforts following cardiac arrest	Sedation/analgesia/paralytics used	Outcome
Lewinter et al. [1]	Pulseless ventricular tachycardia—Myocardial infarction	Not documented. Patient described only as "responsive" during compressions	Mechanical device. (Thumper®, Michigan Instruments, Grand Rapids, Michigan)	Aortic systolic 100–122 mmHg, Aortic diastolic 35–38 mmHg	2 h 42 min	Diazepam and morphine	Death
Quinn et al. [2]	Pulseless electrical activity—Myocardial infarction	Brisk localization during compressions	Active compression–decompression mechanical device	Not documented	36 min	Midazolam (0.1 mg/kg IV) and Succinylcholine (1.5 mg/kg IV)	Death

an hour. Administration of sedation and analgesia may obscure the neurological examination and thereby alter a decision on when to cease resuscitative efforts. In a retrospective study done to identify indices that might assist in deciding whether to continue CPR, it was suggested that for patients in asystole with gasping respiration CPR should continue for at least 30 min and that if pupillary reaction to light were present during CPR resuscitation efforts should continue for at least 45 min [12]. An early prediction score advocated by some authors [13, 14] uses five factors to decide when to cease resuscitative efforts, with longer efforts suggested in the presence of the following: witnessed cardiac arrest, ventricular fibrillation as the initial rhythm, presence of gasping respiration, and the presence of pupillary reaction.

The third issue illustrated by our report is the importance of effective chest compressions. The effective chest compressions performed on this patient very likely played an important role in preserving his cerebral perfusion. The value of chest compressions in maintaining adequate perfusion during CPR and thereby improving outcomes has been clearly shown [15–17]. Current resuscitation guidelines place an appropriate emphasis on effective compressions with minimal interruptions ("push hard, push fast, allow full chest recoil after each compression, and minimize interruptions in chest compressions") with the goal of maintaining a MAP that sustains some degree of coronary and cerebral perfusion [18]. Both reports of retained awareness during CPR that we identified in the literature were of patients resuscitated with mechanical compression devices, which, potentially, provide more effective perfusion pressures than manual compressions. Effective manual compressions in the context of in-hospital cardiac arrests have been shown in one study of an electromechanical compression device to result in peak aortic pressures of 115 ± 42 mmHg, with the electromechanical compression device being studied resulting in even higher peak aortic pressures (153 ± 28 mmHg, $p < 0.0001$) [19]. In the first case report involving a patient with retained awareness, the Thumper® device (Michigan Instruments, Grand Rapids, MI, USA) was used, generating 80 lb of force to used to compress the sternum 2 in [1]. The second report of retained awareness during CPR describes the use of an ACD-CPR device [2]. Some studies have reported better hemodynamic responses and outcomes with ACD-CPR [20–22], although others have found no major benefit [23–25].

In conclusion, retention of a high degree of awareness during resuscitative efforts is possible following cardiac arrest. Better awareness of this unusual and traumatic phenomenon may allow recognition of the situation when it occurs and appropriate decisions to be made regarding the use of sedation and analgesia and the length of resuscitative efforts.

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References

1. Lewinter JR, Carden DL, Nowak RM, Enriquez E, Martin GB. CPR-dependent consciousness: evidence for cardiac compression causing forward flow. Ann Emerg Med. 1989;18(10):1111–5.
2. Quinn JV, Hebert PC, Stiell IG. Need for sedation in a patient undergoing active compression–decompression cardiopulmonary resuscitation. Acad Emerg Med. 1994;1(5):463–6.
3. 2005 AHA Guidelines for CPR and ECC—management of cardiac arrest. Circulation. 2005;112:IV-58–IV-66.
4. Nolan JP, Deakin CD, Soar J, Böttiger BW, Smith G. European resuscitation council guidelines for resuscitation 2005 section 4. Adult Adv Life Support Resuscit. 2005;67S1:S39–86.
5. Latchaw RE, Yonas H, Hunter GJ, Yuh WT, Ueda T, Sorensen AG, Sunshine JL, Biller J, Wechsler L, Higashida R, Hademenos G. Guidelines and recommendations for perfusion imaging in cerebral ischemia: a scientific statement for healthcare professionals by the writing group on perfusion imaging, from the Council on Cardiovascular Radiology of the American Heart Association. Stroke. 2003;34:1084–104.
6. Moppett IK, Hardman JG. Modeling the causes of variation in brain tissue oxygenation. Anesth Analg. 2007;105(4):1104–12.
7. Marín-Caballos AJ, Murillo-Cabezas F, Cayuela-Domínguez A, Domínguez-Roldán JM, Rincón-Ferrari MD, Valencia-Anguita J, Flores-Cordero JM, Muñoz-Sánchez MA. Cerebral perfusion pressure and risk of brain hypoxia in severe head injury: a prospective observational study. Crit Care. 2005;9(6):R670–6. Epub 2005 Oct 14.
8. Bandera E, Botteri M, Minelli C, Sutton A, Abrams KR, Latronico N. Cerebral blood flow threshold of ischemic penumbra and infarct core in acute ischemic stroke: a systematic review. Stroke. 2006;37(5):1334–9. Epub 2006 Mar 30. Review.
9. Ochoa FJ, Ramalle-Gomara E, Carpintero JM, Garcia A, Saralegui I. Competence of health professionals to check the carotid pulse. Resuscitation. 1998;37(3):173–5.
10. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation. 1996;33(2):107–16.
11. Martens P, Mullie A. Sedation during and after CPR, is it worth a guideline? Resuscitation. 1995;29(3):223–4.
12. Van Hoeyweghen R, Mullie A, Bossaert L. Decision making in continuing or cease cardiopulmonary resuscitation (CPR), cerebral resuscitation study group. Resuscitation. 1989;17(Suppl): S137–47.
13. Delooz HH, Lewi PJ. Early prognostic indices after cardiopulmonary resuscitation (CPR), cerebral resuscitation study group. Resuscitation. 1989;17(Suppl):S149–55.
14. Martens PR, Mullie A, Buylaert W, Calle P, van Hoeyweghen R. Early prediction of non-survival for patients suffering cardiac arrest-a word of caution. The Belgian cerebral resuscitation study group. Intensive Care Med. 1992;18(1):11–4.
15. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. Circulation. 2002;105: 2270–3.
16. Yu T, Weil MH, Tang W, Sun S, Klouche K, Povoas H, Bisera J. Adverse outcomes of interrupted precordial compression during automated defibrillation. Circulation. 2002;106:368–72.
17. Abella BS, Sandbo N, Vassilatos P, Alvarado JP, O'Hearn N, Wigder HN, Hoffman P, Tynus K, Vanden Hoek TL, Becker LB. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation. 2005;111:428–34.
18. 2005 AHA Guidelines for CPR and ECC—overview of CPR. Circulation 2005;112:IV-12–IV-8.
19. Timerman S, Cardoso LF, Ramires JA, Halperin H. Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest. Resuscitation. 2004;61(3):273–80.
20. Plaisance P, Lurie KG, Vicaut E, Adnet F, Petit JL, Epain D, Ecollan P, Gruat R, Cavagna P, Biens J, Payen D. A comparison of standard cardiopulmonary resuscitation and active compression–decompression resuscitation for out-of-hospital cardiac arrest. French active compression-decompression cardiopulmonary resuscitation study group. N Engl J Med. 1999;341:569–75.
21. Cohen TJ, Goldner BG, Maccaro PC, Ardito AP, Trazzera S, Cohen MB, Dibs SR. A comparison of active compression–decompression cardiopulmonary resuscitation with standard cardiopulmonary resuscitation for cardiac arrests occurring in the hospital. N Engl J Med. 1993;329:1918–21.
22. Tucker KJ, Galli F, Savitt MA, Kahsai D, Bresnahan L, Redberg RF. Active compression–decompression resuscitation: effect on resuscitation success after in-hospital cardiac arrest. J Am Coll Cardiol. 1994;24:201–9.
23. Schwab TM, Callaham ML, Madsen CD, Utecht TA. A randomized clinical trial of active compression–decompression CPR vs standard CPR in out-of-hospital cardiac arrest in two cities. JAMA. 1995;273:1261–8.
24. Stiell I, H'ebert P, Well G, Laupacis A, Vandemheen K, Dreyer J, Eisenhauer M, Gibson J, Higginson L, Kirby A, Mahon J, Maloney J, Weitzman B. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. JAMA. 1996;275:1417–23.
25. Nolan J, Smith G, Evans R, McCusker K, Lubas P, Parr M, Baskett P. The United Kingdom pre-hospital study of active compression–decompression resuscitation. Resuscitation. 1998; 37:119–25.