



Brain wave disturbance and cognitive impairment after CPR



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ABSTRACT

Cognitive impairment (CI) is a common morbidity after cardio-pulmonary resuscitation (CPR) with long time persistence. Brain hypoxia is believed to be the main but not the single etiology of post CPR cognitive impairment. Theta and lower theta waves of the EEG have essential role in proper functioning of the memory performance. Both endotracheal intubation and atropine administration in CPR process can abolish these waves. We hypothesize that CI in CPR survivors can be caused by disturbance in aforementioned waves due to endotracheal intubation and atropine administration.

Neurological dysfunctions (ND) are the most common morbidity in survivors of cardio-pulmonary resuscitation (CPR) [1,2]. In these patients cognitive impairment (CI) is a common and long lasting complication of CPR. Hypothermia reduces body oxygen consumption which results to a decrease in brain hypoxia and the followed reduction in CI. On the other hand, there is controversy on the role of brain hypoxia, due to insufficiency of blood supply, in cognitive impairment. Tiainen et al have shown that cardiac arrest survivors who are treated with therapeutic hypothermia had similar rate of cognitive impairment in comparison with control group [3]. Elapsed time to start a CPR and to return of spontaneous circulation is shorter in the in-hospital cardiac arrest and it is expected that cognitive impairment will be lower in the in-hospital cardiac arrest due to emergency brain oxygenation and shorter time of brain hypoxia. But evidence have shown that in both in-hospital and out-hospital cardiac arrests, cognitive impairments are the most common morbidity [4,5]. In another study it has been shown that elapsed time to start a CPR, the use of defibrillator, and return of spontaneous circulation have weak correlations with cognitive deficit [6]. Based on the above findings, one may conclude that brain hypoxia is not the sole responsible for CI in CPR survivors.

Previous studies have shown that hippocampus modulates neurons in medial prefrontal cortex (PFC) to produce theta-frequency (4–8 Hz) oscillations, especially type 1 theta waves, to keep a proper memory function [7]. Moreover, the theta waves are known to have important role in cognitive processes via synchronization between PFC and Hippocampus and dis-synchronization between these two areas can lead to cognitive deficit [8–10].

Atropine, a competitive antagonist of the muscarinic acetylcholine receptors, is frequently used in CPR cases when the heart rate is below 60/min. Previous studies have shown that after reaching the brain, atropine can abolish type 1 EEG theta waves [11,12]. One may conclude that the use of atropine via abolishment of theta waves might be a reason for increased cognitive impairment in CPR survivors. If this is the case, it is advisable to use a muscarinic acetylcholine receptor antagonist without the ability to cross blood brain barrier, instead of atropine.

Growing body of evidences has shown that mammalian olfactory sensory neurons (OSNs) have a role not only in smell but also in cognitive function via receiving nasal airflow mechanical stimulation. These studies

have shown that nasal airflow leads to a mechanical stimulation of nasal epithelium and produces low frequency waves in brain that couple with nasal respiration. Tracheotomy abolished hippocampal respiration-coupled rhythm, which was restored by rhythmic delivery of air puffs into the nasal cavity [13]. Previous studies have shown that these low frequency waves synchronized Hippocampus and other area of the brain such as PFC and these waves are responsible for the role of olfactory bulb in cognitive function. Also, Arshamian et al in recent study have confirmed effects of respiratory-related oscillations in memory [14].

It has been shown that in the case of nasal airflow disruption, cognitive impairment will be happened. One of the primary steps in CPR is establishing secure way to breath with endotracheal intubation. By using endotracheal tube, the air tends to pass through it instead of nostrils, due to the less resistance. In this situation, nasal air flow will be disrupted followed by disappearance of low wave oscillations that are produced by olfactory bulb. Based on the above reported sequence of events, one may hypothesized that disruption of nasal airflow is the second reason for cognitive impairment in CPR survivors.

Based on the above findings, our hypothesis is that disruption of nasal air flow, independently or concomitant with administration of atropine, will lead to cognitive impairments in the CPR survivors. We suggest that the impairment is due to abolishment of slow wave oscillation generated by olfactory bulb and EEG theta waves. To examine the present hypothesis, we suggest designing a device that pushes the air to nostrils in CPR subjects concomitant with the use of a cholinergic antagonist which cannot pass blood brain barrier e.g. glycopyrrolate.

Conflict of interest

The authors and co-authors have no personal conflict of interests.

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