Research targets long-term brain deficits in cardiac arrest survivors

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Research conducted by Jason Middleton, PhD, Assistant Professor of Cell Biology and Anatomy, and Neuroscience at LSU Health New Orleans School of Medicine, and colleagues may lead to a treatment to prevent long-term sensory problems by restoring normal brain function in survivors of cardiac arrest. The study, done in a rodent model and using modeling data, is published online in *eNeuro*, an open-access journal of the Society for Neuroscience.

Cardiac arrest is a common cause of brain injury. When the brain is deprived of oxygen, not only can cells die, but surviving cells can suffer damage resulting in long-term sensory dysfuntion in the cortex. The cerebral cortex is the outer covering of the brain—the gray matter that covers hemispheres of the brain like a helmet. This is the part of the brain that receives sensory input, such as vision, hearing and touch, and areas of the cortex are also involved in more complex functions, such as memory, language, creativity, judgement and emotion.

The research team studied the long-term impact of cardiac arrest on the cortex in a rat model. They measured sensory response and found that after oxygen deprivation, the sensory circuits in the cortex are less responsive with behavioral deficits. Their data suggest that cardiac arrest and resuscitation permanently affect cortical circuit function in survivors.

"Our work characterizes the changes that occur in the sensory cortex after a form of global hypoxic injury in juvenile rats," notes Dr.

Middleton. "The injury did not result in widespread cell death as occurs in other forms of acute, focal ischemic injury; the deficits uncovered were subtler and reflected decreased ability of the cortex to discriminate sensory stimuli. We used computer modeling of the neural network to implicate changes in the balance of excitatory and inhibitory synaptic transmission in the cortex."

According to the American Heart Association, more than 350,000 Americans experienced out-of-hospital cardiac arrest last year. With bystander CPR, 46.1% survived.

"These findings lay the groundwork for further studies to pinpoint therapeutic targets to restore excitatory/inhibitory balance in the injured brain and mitigate sensory deficits later in life," concludes Middleton.

More information: Jason W. Middleton et al, Long-Term Deficits in Cortical Circuit Function after Asphyxial Cardiac Arrest and Resuscitation in Developing Rats, *eneuro* (2017). DOI: 10.1523/ENEURO.0319-16.2017

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