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CO₂-Related Vasoconstriction Imposed on Medullary Brain Ischemia During Sleep Apnea in Heart Failure by Adaptive Servo-Ventilation May Trigger Sudden Death

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Abstract

Schematically on a drawing board, the heart is an electric pump. The brain is a computerized electronic guidance system. The vagus nerve is an electric cable cord attached to the heart, utilized to plug the heart into the brain, just as an electric pump cord would be plugged into a wall socket. The cable holds 2 wires, each insulated from the other: an up-stream wire from the heart to the brain, and a down-stream wire from the brain to the heart. At the end of the cable cord are 2 metal prongs that fit into an electric socket in the brain. Each metal prong attaches to one of the 2 wires in the vagus nerve. The 2 metal prongs that are 'plugged into' the brain are thereby imbedded in the brain and receive their blood supply from the brain vessels, which are significantly influenced by carbon dioxide (CO₂) levels. The solitary tract nucleus is the up-stream wire prong. The dorsal motor nucleus of the vagus nerve is the down-stream wire prong.

When someone initially begins to develop heart failure, the myocardium begins to suffer from lack of oxygen. The heart then sends distress signals to the brain through the vagus nerve up-stream wire. The brain then makes adjustments in the heart rate and contractility through the down-stream wire. As heart failure becomes progressively more severe, the 2 metal prongs are unable to handle the high volume of signaling which occurs, and they begin to overheat, and require increased blood flow. Respiratory therapy given to improve myocardial oxygenation, or to manage concurrent sleep apnea, may inadvertently lower CO₂ levels, causing vasoconstriction in the brain, and reducing blood flow to the metal prongs. In the absence of a treatment modality which offers increased blood flow in the brain, an electrical short circuit may occur between the metal prongs, sending abnormal electrical discharges from the prongs through the down-stream wire to the heart – sometimes causing cardiac arrest.

Keywords: Sleep apnea; Heart failure; Vagus nerve; Solitary tract nucleus; Brain medulla; Brain ischemia; Cardiac arrhythmia; Cardiac arrest; Vasoconstriction; CPAP; Carbon dioxide

Editorial

Forensic investigators and others [1] remain confounded as to the cause of increased mortality associated with adaptive servo-ventilation when used in the management of central sleep apnea in systolic heart failure. I have a few observations.

During adaptive servo-ventilation, blood flow in the brain is probably reduced, causing adverse effects. Carbon dioxide is a potent dilator of brain vasculature, thereby increasing blood flow to the brain. When ventilation is increased, even if only to improve it back toward normal from a depressed steady-state level, the alveolar partial pressure of carbon dioxide is decreased, likely resulting in a converse relative vasoconstriction in the brain, thereby reducing blood flow in the brain, especially in pertinent watershed areas like the solitary tract nucleus of the medulla. In normal physiology this is demonstrated impressively by the ability of hyperventilation to induce loss of consciousness.

When blood flow is decreased to an area that is already ischemic like the solitary tract nucleus, the ischemia will likely worsen and cause secondary effects related to its normal function which includes regulation of heart rate, rhythmicity, and contractility through its efferent (downstream) output pathway via the dorsal motor nucleus of the vagus nerve.

In recent years sudden unexpected death has been associated with many types of small and pathologically benign medullary brain lesions like demyelination plaques — largely asymptomatic until they caused sudden death [2]. Many such medullary lesions, typically without hemorrhage or

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mass effect, have in themselves been previously considered relatively harmless — in cases where they have been known to be present. Some not known to be present during life were discovered at autopsy in cases where no other cause of death could be identified.

And significantly, in heart failure and in sleep apnea (which often co-exist), sudden death has also been associated with medullary autonomic ischemic lesions, especially involving the solitary tract nucleus [3-5]. Sensory afferent (upstream) vagus nerve stimulation terminates at the solitary tract nucleus in the medulla, where the increased metabolic requirements associated with intense neurotoxic stimulation converge with a limited watershed vasculature to contribute to the formation of focal ischemic lesions. This constellation of events is believed to trigger sudden death by a mechanism which is unknown, but may be efferent (downstream) arrhythmogenic through the dorsal motor nucleus of the vagus nerve.

Confounding all of this pathophysiology is the possibility of reperfusion injury to the solitary tract nuclei when adaptive servo-ventilation is stopped and vasoconstriction is stopped [6,7].

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