A Dangerous Myth: Does Speaking Imply Breathing?

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On 25 May 2020, George Floyd pleaded at least 16 times, "I can't breathe." One officer in attendance nonetheless told bystanders, "He's talking. He's fine" (1, 2). Mayor Hal Marx of Petal, Mississippi, posted on Twitter the following day, "If you can say you can't breathe, you're breathing." Similar arguments were put forth by New York Representative Peter King and police officials in the wake of Eric Garner's death in 2014 (3, 4). The belief that a person's ability to speak precludes the possibility of suffocation is not true and can have fatal consequences. Although the medical community may suspect that vocalization does not guarantee adequate respiration, they may not be sufficiently familiar with the relevant physiology to allow them to speak with authority. Here, we review basic respiratory physiology and highlight our role as clinicians and scientists in educating the public against relying on speech as a sign of adequate respiration—especially when this medical misconception is used to propagate injustice or violence.

The volume of an ordinary breath is approximately 400 to 600 mL. When each breath is inhaled, air first fills the upper airway, trachea, and bronchi; speech is generated here, but no gas exchange takes place in this anatomical dead space. Only air that exceeds the volume of this dead space is conducted to the alveoli for gas exchange. Normal speech only requires approximately 50 mL of gas per syllable—thus, stating "I can't breathe" would require 150 mL of gas (5). Anatomical dead space is typically one third the volume of an ordinary breath. George Floyd could have uttered those syllables repeatedly with small breaths that filled only the trachea and bronchi but brought no air to the alveoli, where actual gas exchange happens.

Phonation can occur with exhalation alone in the complete absence of inhalation by using the expiratory reserve volume that remains after a normal tidal breath is exhaled. In contrast, adequate gas exchange to support life requires inhalation, as well. A 70-kg adult requires 4 to 5 L of air per minute (at rest) to reach the alveoli, where oxygen and carbon dioxide are exchanged with the blood; light muscular activity requires double that, and a person in extremis may require more than 100 L of air per minute (6). If less air reaches the alveoli, there may be devastating cardiopulmonary consequences, including death.

The origin of the pernicious myth that speaking signals adequate breathing is unclear but may be an extrapolation from first aid training for choking. Multiple training sources cite the inability to talk as a sign of choking along with other late signs of respiratory compromise, including dusky skin or loss of consciousness (7). It is true that if you cannot move any air you cannot speak. However, the reverse is not true: You can move enough air to produce sound but not be able to breathe enough to sustain the gas exchange needed to prevent organ damage from hypoxemia. It is therefore not surprising that such a person as George Floyd may have been able to both generate the sentence "I can't breathe" and still experience severe air hunger (that is, dyspnea) and decompensate into a state of respiratory failure.

This apparent paradox is also consonant with our experiences as pulmonary and critical care clinicians. We all have taken medical histories and even discussed intubation with patients who had rapidly increasing carbon dioxide levels or decreasing oxygen levels and were clearly in respiratory failure. A colleague recalled her child having the ability to scream, "I can't breathe," before losing consciousness from respiratory failure during an asthma exacerbation. As care providers, we are trained to prioritize addressing impairments to breathing and circulation above all else. Doing so includes recognizing earlier signs of respiratory failure, such as an increased respiratory rate, tripoding, or using accessory muscles to breathe. Waiting until a person loses the ability to speak may be too late to prevent catastrophic cardiopulmonary collapse.

Air hunger is the most uncomfortable and emotionally distressing quality of dyspnea. It directly activates the insular cortex, a primal sensory area of the brain that responds to such basic survival threats as pain, hunger, and thirst (8). Data from studies of war and torture victims show that the sensation of suffocation is the single strongest predictor of posttraumatic stress disorder and can cause more persistent psychological damage than mock execution with a pistol (9). This finding suggests that clinicians have a fundamental responsibility to serve as advocates for persons who report respiratory distress.

The use of incorrect physiologic statements to justify the force leading to the deaths of Eric Garner and George Floyd is unacceptable. According to our oath as clinicians, it is our responsibility to the public to aggressively correct such misconceptions to prevent further deaths. However, as human beings, we emphasize that it does not take medical training to inherently understand the profound danger and inhumanity behind forcibly inducing respiratory distress in another person. The persistent use of malignant platitudes in the face of another person's suffering is disturbing. At best, it represents the thoughtless use of heuristic shortcuts; at worst, it indicates deep gaps in empathy, toxic cognitive biases, or malicious intent. We hope that everyone will join us in advocating that all persons who describe

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respiratory distress receive immediate, serious attention and treatment.

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