

# The Breath-emotion Loop: Neural Mechanisms of Dyspnea, Anxiety and Chronic Hyperventilation

Baumann Kohlman\*

Department of Pulmonology, Chang Gung Memorial Hospital, Taoyuan 333, Taiwan

## Introduction

Breathing isn't just a mechanical process—it's a window into our emotional state. The sensation of breathlessness (dyspnea), the heightened arousal of anxiety and the pattern of chronic hyperventilation form a tightly coupled physiological–neural circuit. In this Breath-Emotion Loop, disruptions in respiration amplify emotional distress, while emotional states, in turn, distort breathing dynamics. This cyclical interaction underlies common clinical phenomena—panic attacks, functional dyspnea in anxiety disorders and chronic hyperventilation syndrome. Achieving a clearer understanding of this loop is critical to refining therapeutic strategies such as breathing retraining and cognitive–behavioral interventions [1].

At the core of respiration lies the preBötzinger complex (preBötC)—a rhythm-generating network in the brainstem driving inspiratory cycles. This basal rhythm is modulated by higher-order brain regions such as the insula, Anterior Cingulate Cortex (ACC) and amygdala—key players in emotional evaluation and interoception. The locus coeruleus, a key arousal center, links respiratory dynamics to sympathetic tone [2].

## Description

Dyspnea—subjective air hunger—arises from mismatch signals between respiratory effort and afferent feedback. Even without lung pathology, anxious individuals may hyper-focus on breathing, triggering catastrophizing. For example, elevated ACC activation correlates strongly with perceived dyspnea severity during induced breath restriction, suggesting the ACC amplifies breath-related emotional distress. Anxiety activates the sympathetic nervous system via the fight-or-flight response, increasing respiratory rate—which can spiral into hyperventilation with resultant hypocapnia and respiratory alkalosis. Chronic Hyperventilation Syndrome (HVS) is characterized by persistent fast, shallow, or sighing breathing patterns causing over-inflation and hypocapnic alkalosis. Physiological symptoms from alkalosis reinforce anxiety, maintaining the cycle. Many individuals with panic disorder also meet criteria for HVS. Sensory feedback from mechanosensors in the lungs and chest wall is relayed by the insula and ACC, integrating bodily signals with emotional processing. The ACC assesses threat salience, often exaggerating harmless respiratory changes into perceived crises. The amygdala, central to fear, may show hyperactivation and reduced inhibition in panic-prone individuals. Neurophysiological research indicates that controlled slow breathing—5–6 breaths per minute—can enhance parasympathetic tone, increase Heart Rate Variability (HRV) and modulate brain oscillations, helping to calm affective circuits [3].

**\*Address for Correspondence:** Baumann Kohlman, Department of Pulmonology, Chang Gung Memorial Hospital, Taoyuan 333, Taiwan, E-mail: kohlmanbaumann@ahn.tw

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**Received:** 02 April, 2025, Manuscript No. jprm-25-167382; **Editor assigned:** 04 April, 2025, PreQC No. P-167382; **Reviewed:** 18 April, 2025, QC No. Q-167382; **Revised:** 23 April, 2025, Manuscript No. R-167382; **Published:** 30 April, 2025, DOI: 10.37421/2161-105X.2025.15.729

Dyspnea is not merely a mechanical or chemical sensation but an intensely emotional experience. It can occur in the absence of pulmonary pathology, especially in anxiety disorders. Research shows that patients with anxiety often report dyspnea during stress or panic attacks despite normal respiratory function. Neuroimaging studies highlight the activation of the insula and ACC during experimentally induced dyspnea. These regions interpret respiratory effort and discomfort, modulating the emotional impact of breathlessness. For instance, while a mild elevation in CO<sub>2</sub> may go unnoticed by one individual, it may trigger a panic episode in another, depending on their cortical appraisal and prior experiences. These altered patterns are not merely symptoms but causal contributors to sustained anxiety. Hyperventilation reduces arterial CO<sub>2</sub> levels (hypocapnia), leading to respiratory alkalosis. This biochemical imbalance causes symptoms like dizziness, chest tightness and paresthesia, which are then misinterpreted as signs of danger, fueling more anxiety [4].

Functional MRI studies in HVS patients reveal exaggerated responses in the insular cortex and reduced gray matter in emotion-regulatory areas. The syndrome often coexists with panic disorder and may be exacerbated by stress, caffeine and lack of sleep. Long-term hyperventilation desensitizes chemoreceptors to CO<sub>2</sub> levels, leading to a lowered threshold for panic and breathlessness. This vicious cycle is perpetuated by neural feedback loops. The ACC and insula not only respond to but also modulate breathing effort. Repeated exposure to such sensations wires the brain for heightened threat perception regarding bodily sensations—a hallmark of anxiety disorders [5].

## Conclusion

The Breath–Emotion Loop offers a compelling framework to understand the intertwined nature of respiratory and emotional dysregulation. Rather than viewing symptoms like dyspnea or panic in isolation, this model underscores their reciprocal relationship. Central neural circuits including the insula, ACC and amygdala serve as integration hubs where breathing and emotion converge. Therapeutic strategies that engage both sides of the loop—correcting breathing mechanics and restructuring emotional interpretations—are essential. As neuroscience continues to unravel the brain–body interface, integrative approaches promise not only symptom relief but long-term resilience. In future clinical paradigms, breathing may no longer be a mere vital sign but a vital key to emotional healing.

## Acknowledgement

None.

## Conflict of Interest

None.

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**How to cite this article:** Kohlman, Baumann. "The Breath-emotion Loop: Neural Mechanisms of Dyspnea, Anxiety and Chronic Hyperventilation." *J Pulm Respir Med* 15 (2025): 729.