

# Disordered Displays of Emotions: An Exploration of Pseudobulbar Affect

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For many of us, our emotional responses to situations seem to almost follow a universal script. Different scenarios generally tend to elicit different emotional outputs based on the affective tone of the scenario itself to the severity of its emotional quality. When we recount a mildly funny situation to a coworker by the water-cooler, we expect them to politely chuckle for a brief moment. Meanwhile, while watching comedy specials of our favorite comedians, we would predictably allow ourselves to let out gut-busting laughs complete with a touch of knee-slapping and a single happy tear. Spilling coffee on a favorite shirt would not draw out anything more than a frown, but news of the sudden death of a loved one may send us into a sustained, hysterical, body-racking cry. These are the emotional norms we follow both implicitly and deliberately. 'X' emotional stimulus outputs 'y' emotional response, in which 'y' is both mood-congruent and lasts for an appropriate duration of time.

However, in some people with a rare neurological condition called Pseudobulbar Affect (PBA), something in this system gets disrupted. The bout of laughter that you and I would let out in response to a well-crafted stand-up joke may be elicited by a PBA patient even if they were not provoked. While I might feel a twinge of displeasure if I receive harsh feedback from an editor on this article, someone with PBA would probably exhibit a prolonged cry to something of a similar negative valence. This article explores the symptoms, underlying pathophysiology, and proposed treatment of Pseudobulbar Affect, a neurological condition marked by episodes of sudden, uncontrollable, mood-incongruent and inappropriate crying and / or laughing.

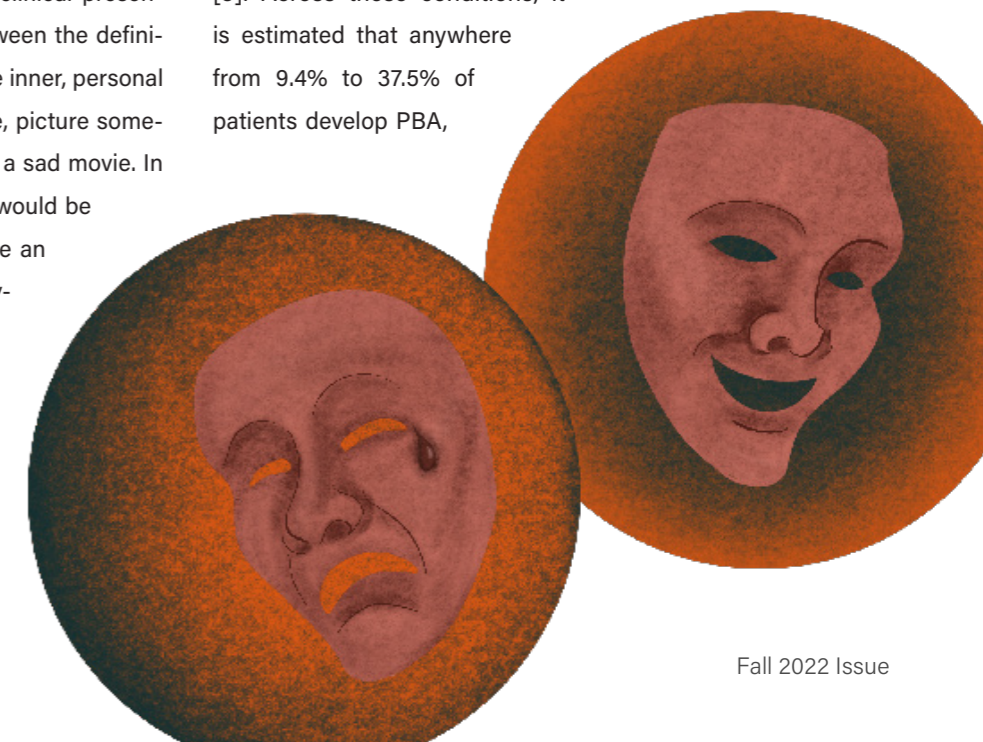
## A BREAKDOWN OF PSEUDOBULBAR AFFECT'S PRESENTATIONS

Before we delve into the symptomatology and clinical presentations of PBA, it's important to distinguish between the definitions of "mood" and "affect". "Mood" refers to the inner, personal experience of a certain emotion [1]. For example, picture somebody crying due to the sadness they feel during a sad movie. In this scenario, the internal sensation of sadness would be the "mood". However, the act of crying would be an instance of "affect", or the objective and observable display of an emotion [1]. Here, the affect (crying) is mood-congruent, meaning it matches the emotion conveyed by the mood (feeling sad). Though the concepts of "mood" and "affect" are very closely connected establishing their difference is necessary to grasp the mechanisms behind PBA.

PBA is an affective disorder, meaning that though the internal experiences of emotions are otherwise "normal", the abnormality lies within the way emotions are displayed [1]. When talking about the disordered way PBA patients demonstrate emotions, the presentations can take on more than one form. Some PBA patients may display affect that is congruent to the mood they feel, but their affect is exaggerated beyond the emotional severity of the situation [2]. For example, they might laugh hysterically for a long duration of time to something that is both objectively slightly amusing and internally experienced as just slightly amusing. On the other hand, some PBA patients express affect that is neither congruent to the mood they feel nor the emotional tone of the situation [2]. Crying while in a positive mood in response to a funny joke, or in the absence of an emotional stimulus, is an example of this. Furthermore, the affect expressed by PBA patients has been described as presenting in uncontrollable episodes [3]. In other words, not only are patients unable to regulate appropriate affect, they are also unable to put a stop to their prolonged display of emotion whilst in the midst of it. Like an implicit knee-jerk reaction, these various affective presentations can come on suddenly, and are impossible to control.

## PROPOSED NEUROLOGICAL CAUSES AND PATHOPHYSIOLOGY

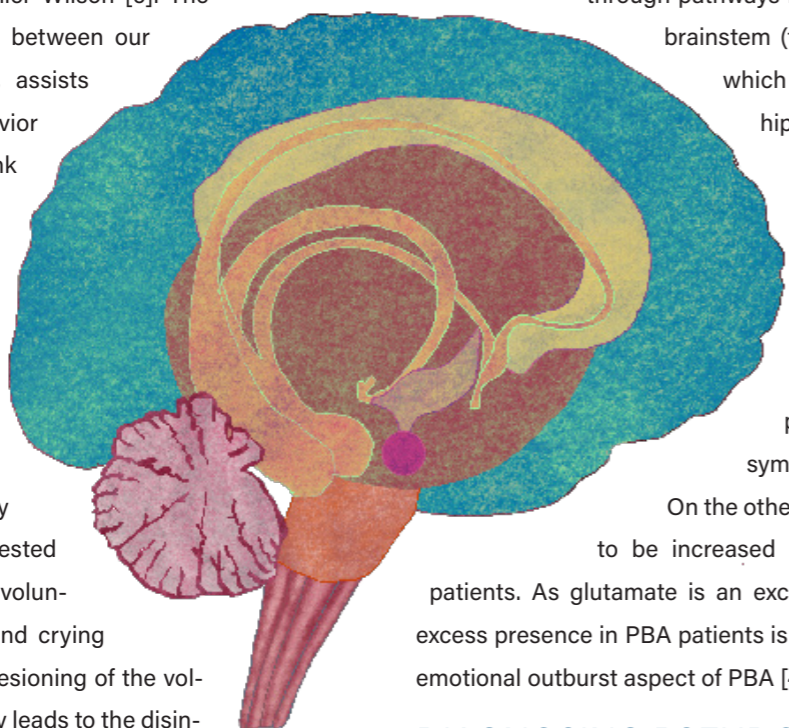
The uncontrollable symptoms of PBA are rooted in neurological dysfunction. Most patients with PBA developed the disorder as a result of a neurological ailment, or after sustaining a brain injury [4]. The neurological conditions most associated with the occurrence of PBA include Alzheimer's dementia, multiple sclerosis, Parkinson's disease, amyotrophic lateral sclerosis, stroke, and traumatic brain injury [5]. Across these conditions, it is estimated that anywhere from 9.4% to 37.5% of patients develop PBA,



depending on the disorder itself as well as individual physician scoring criteria [5]. As such, PBA is considered a relatively rare neurological condition.

Given its rarity, the exact pathophysiology of PBA is also rather ambiguous. In terms of neurocircuitry, there are two main hypotheses for PBA onset, both of which are rooted in the hindbrain.

The first is the brainstem hypothesis, which was originally proposed in 1923 by S. A. Kinnier Wilson [6]. The brainstem, which is located between our brain and our spinal cord, assists with the aspects of our behavior that we do not have to think about [7]. The basis of this theory is that the brainstem area that assists with the expression of laughing and crying contains two separate pathways: one for voluntary expression, and another for involuntary expression [8]. Wilson suggested that PBA occurs when the voluntary pathway for laughing and crying is somehow disrupted. The lesioning of the voluntary pathway consequently leads to the disinhibition of the involuntary pathway. This would explain the uncontrollable mood incongruity seen in PBA.



The second, more recent, neurocircuitry hypothesis centers around the role of the cerebellum in the modulation of emotion. In humans, the cerebellum, which has a host of responsibilities, is a structure that is nestled in between the back of the brain and the brainstem [7]. Under normal circumstances, the frontal cortex sends cognitive, emotional, and social context information about a situation to the cerebellum [8]. The cerebellum would then use this information to regulate both the intensity and duration of the facial muscle activity that produces emotional expression (affect). In PBA patients, according to this hypothesis, there is a disruption in the pathway between the frontal cortex and the cerebellum [9]. As the cerebellum is operating with less contextual information about the situation, it can not adequately adjust emotional expression to match the appropriate emotionality of the situation.

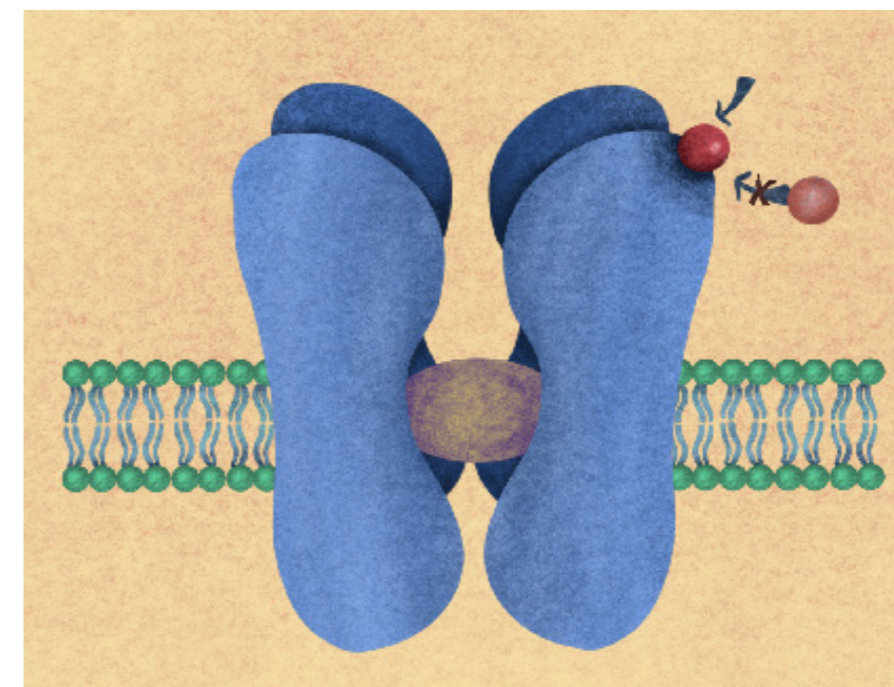
On a more cellular level, there are two main neurotransmitters that are thought to be involved in PBA's etiology: serotonin and glutamate [3]. Within the human body, serotonin is used in several biological processes including the modulation of emotional expression. Key brain structures involved in emotional regulation include the hippocampus, which aids in the processing of emotional memories [10], and the frontal cortex, which is involved in conscious cognitive control [11]. The striatum serves as an integration point that receives input from these areas [12]. Serotonin aids in the modulation of emotional expression through pathways from the raphe nuclei of the brainstem (the cluster of cell bodies in which serotonin is created) to the hippocampus, frontal cortex, and the striatum [4]. Since serotonin works to regulate how emotions are expressed, it is thought that a decrease in serotonin in these pathways is partially responsible for the symptoms displayed in PBA [5].

On the other hand, glutamate is thought to be increased in these pathways in PBA patients. As glutamate is an excitatory neurotransmitter, its excess presence in PBA patients is thought to contribute to the emotional outburst aspect of PBA [4].

## DIAGNOSING PSEUDOBULBAR AFFECT

Due to both the rarity and the complexity of PBA, a definitive diagnostic scale for PBA has not yet been established [13]. As such, clinicians have used patient history, structured interviews, neuroimaging results, and the Pathological Laughing and Crying Scale to aid in identifying PBA in patients [14]. However, there have been several proposed diagnostic criteria for determining the presence of PBA [2]:

1. The patient experiences episodes of involuntary and / or exaggerated emotional expressions as a result of a neurological disorder.
2. These episodes differ from the patient's typical emotional reactivity, are exaggerated or mood-incongruent, and occur either without a stimulus present or in excess to a stimulus.
3. These episodes cause significant distress or impairment in social and / or occupational settings to a clinical degree.



4. The above symptoms can not be attributed to another neurologic or psychiatric disorder, or to the effects of a substance.

Part of the difficulty in diagnosing PBA is due to its observable similarities to mood disorders, with depression being the most common misdiagnosis in PBA cases [2]. This confusion makes differential diagnosis, or the process used to identify the proper diagnosis from other possible diagnoses, all the more necessary [15]. Unlike PBA patients, those with depression can mostly control their crying, as it stops once their mood changes [14]. Furthermore, in patients with depression, crying is always mood-congruent, and the duration and start of the crying is defined by the negative mood they are experiencing [14]. Episodes of laughter are not present in patients with depression, but are one of the hallmarks of PBA [14]. Finally, while PBA occurs secondary to the development of a neurological disorder or condition, depression's etiology is multifactorial and does not specifically have neurological roots [14]. As PBA can initially be confused with severe depression, obtaining a comprehensive and detailed history of patients presenting with such symptoms is necessary to avoid a misdiagnosis.

## TREATMENT FOR PSEUDOBULBAR AFFECT

Currently, there is only one drug that has been approved by the U.S. Food and Drug Administration for treating PBA: dextromethorphan (DM)/quinidine (Q) [16]. The Q component solely works to make DM, a naturally weak substance, more bioavailable in the central nervous system [16]. DM is commonly known for its properties as an N-methyl-D-aspartate (NMDA) receptor

antagonist [17], meaning DM hinders the typical functions of NMDA receptors [18]. NMDA receptors are a form of glutamate receptors. Since excess glutamate is thought to be involved in PBA's etiology, it is DM's properties as an NMDA receptor antagonist that partially alleviates PBA symptoms [19].

DM also works at a variety of other receptors and transporters, including acting as a reuptake inhibitor at serotonin transporters [19]. Embedded on the membrane of neuron axon terminals, reuptake transporters remove their respective neurotransmitters from the synaptic cleft [20]. Therefore, inhibiting reuptake transporters results in

leaving more of a certain neurotransmitter to act on its receptors. DM's ability to inhibit serotonin reuptake transporters allows for more serotonin presence in the neural circuits associated with PBA, further reducing its symptoms [19]. To date, the use of DM/Q seems to be the most effective treatment for PBA [16]. While other pharmaceutical interventions, such as tricyclic antidepressants and selective serotonin reuptake inhibitors, as well as behavioral approaches, have been attempted to treat PBA, there is limited evidence for their efficacy [19]. Ultimately, it is recommended that treatment of the underlying neurological disorder, in addition to the use of DM/Q, is the best course of action to completely treat PBA [19].

## CONCLUSION

When considering the landscape of research on PBA, it is evident that further exploration is needed to fill in the gaps of knowledge. The epidemiology of PBA suggests that it is largely undiagnosed or misdiagnosed due to clinicians' lack of awareness about the disorder [4]. As such, a more robust presence in the medical literature would in turn help patients with PBA symptoms receive the proper care they need. This is especially crucial considering PBA's striking presentations carry severe social detriments. As their uncontrollable crying and / or laughing could be considered disruptive or uncomfortable in interpersonal and work functions, many individuals with PBA face social rejection, job loss, and significant relationship issues [21]. By receiving medication to ameliorate their symptoms, and therapy to talk through the psychological distress their illness causes them [21], PBA patients may be able to finally attain a sense of solace. 🧠

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