ORIGINAL ARTICLE

Pseudobulbar Palsy in Artery of Percheron Infarction: A Narrative Mechanistic Review of Structural Versus Network Disconnection

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ABSTRACT

Background: Pseudobulbar palsy (PBP) is an upper motor neuron syndrome that typically results from bilateral CBT injury due to vascular, demyelinating, or degenerative processes. Infarction of the Artery of Percheron (AOP)—a rare single arterial variant supplying both paramedian thalami and sometimes the rostral midbrain—can produce bilateral motor deficits from a unilateral vascular lesion.

Objective: To describe anatomical and functional mechanisms by which AOP infarction can result in pseudobulbar palsy and to summarize reported clinical and imaging findings.

Methods: A narrative literature review was conducted using Google Scholar and related databases to identify case reports and series of AOP infarction published from 2019 onward. Studies were included if they reported clinical and radiological features relevant to corticobulbar pathway or thalamic-mesencephalic network involvement. No formal quality assessment or quantitative synthesis was performed.

Results: Across reviewed publications, PBP occurred in association with both isolated bilateral thalamic and combined thalamic–midbrain infarcts. Cases with midbrain involvement demonstrated imaging evidence of direct CBT injury, whereas purely thalamic infarctions were linked to disruption of motor relay pathways causing functional disconnection of brainstem motor nuclei. Lesion distribution within the AOP territory appeared to determine the mechanism of pseudobulbar presentation.

Conclusion: AOP infarction may produce pseudobulbar palsy through either structural corticobulbar damage or thalamic network disconnection. This pattern illustrates how unilateral vascular lesions can generate bilateral motor syndromes and highlights the need for further neuroimaging studies to define corticobulbar network organization in the thalamic–mesencephalic region.

INTRODUCTION

Pseudobulbar palsy (PBP) is a distinct upper motor neuron syndrome characterized by dysarthria, dysphagia, lingual and facial weakness, and the pathological emotional expression known as pseudobulbar affect.[1] It typically arises from

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bilateral corticobulbar tract disruption secondary to multifocal vascular, demyelinating, or degenerative pathology.[1,2] The occurrence of PBP following an Artery of Percheron (AOP) infarction presents an anatomical paradox: a unilateral vascular lesion producing bilateral motor deficits.[3]

The AOP is an uncommon anatomical variant, found in approximately 0.1–0.7% of angiographic or autopsy studies, and is estimated to cause less than 0.5% of ischemic strokes.[5] Because of its small caliber and deep location, AOP occlusion is frequently missed or misdiagnosed, often confused with metabolic encephalopathy or top-of-the-basilar syndrome.[5] Clinically, AOP infarction most often produces the classic triad of altered mental status, vertical gaze palsy, and memory impairment defining the paramedian thalamic syndrome. Yet presentation is highly variable: symptoms may include fluctuating alertness, cranial nerve deficits, or pseudobulbar features when the infarct extends into the rostral midbrain. A recent systematic review emphasizes both this heterogeneity and the frequent diagnostic delay. This review investigates how a single-vessel AOP infarction can lead to bilateral pseudobulbar palsy. It integrates vascular anatomy, corticobulbar organization, and thalamic–mesencephalic network function to clarify mechanisms underlying this rare phenomenon.[5]

To clarify this relationship, the paper first reviews the neurovascular anatomy of the AOP and its clinical variants. It then outlines the architecture and bilateral organization of the corticobulbar system, emphasizing the compensatory mechanisms that typically preserve bulbar function following unilateral lesions. Next, it examines the radiological and clinical spectrum of AOP infarction, including the broader framework of paramedian thalamic syndrome. The analysis then synthesizes clinical and anatomical evidence to propose a mechanistic model linking AOP occlusion to the development of pseudobulbar palsy, culminating in a unified interpretation that situates this phenomenon at the intersection of vascular anomaly and neuroanatomical vulnerability.

Ultimately, pseudobulbar palsy secondary to AOP infarction represents a rare but revealing convergence of vascular and neural systems, wherein a single vascular event exposes the bilateral dependency and limited redundancy of supranuclear motor control.[5,6,7] This unique lesion model underscores the intricate balance between cerebral vascular architecture and the distributed organization of human motor pathways.

This study makes several original contributions to the understanding of how vascular anomalies can precipitate complex neurological syndromes, specifically elucidating the mechanistic basis of pseudobulbar palsy (PBP) secondary to Artery of Percheron (AOP) infarction. By bridging the domains of cerebral vascular variation, descending motor system anatomy, and functional neurocircuitry, this work advances a unified framework that extends beyond descriptive case analysis. It proposes a dual-mechanism model integrating both direct bilateral ischemic disruption of CBT within the rostral midbrain and functional cortico-thalamo-bulbar disconnection within the paramedian thalami, demonstrating how a single vascular event can simultaneously cause structural and network-level dysfunction in the supranuclear motor system. This perspective redefines the concept of bilateral motor vulnerability, revealing that the AOP territory represents a unique neurovascular “critical point” where minimal redundancy in the corticobulbar system allows a unilateral lesion to generate a bilateral upper motor neuron syndrome. Furthermore, the study introduces the novel concept of functional de-efferentation, in which bilateral thalamic injury disrupts higher-order cortico-thalamic integration, effectively silencing corticobulbar output despite anatomical tract preservation—thereby reframing certain thalamic strokes as network-level disconnection syndromes. Through synthesis of clinical, radiological, and anatomical evidence, the paper establishes that PBP secondary to AOP infarction exists along a pathophysiological continuum ranging from structural tract damage to

functional disconnection, depending on infarct epicenter and vascular variability. This unified model provides a conceptual scaffold for interpreting heterogeneous clinical presentations and enhances clinicoradiological correlation. More broadly, this research positions vascular anomalies such as AOP occlusion as natural experiments in neural network failure, offering a powerful paradigm for exploring how vascular architecture constrains neural redundancy, resilience, and recovery.

METHODS

This study is a narrative literature review aimed at elucidating the mechanistic relationship between Artery of Percheron (AOP) infarction and pseudobulbar palsy (PBP). The review focused on how a single-vessel vascular anomaly can result in bilateral supranuclear motor dysfunction, emphasizing neuroanatomical and neuroimaging correlates rather than the broader spectrum of posterior circulation strokes. The goal was to integrate clinical, radiological, and anatomical evidence to explain how vascular configuration, corticobulbar tract (CBT) vulnerability, and thalamic–midbrain network interactions converge to produce PBP.

Because available data are derived primarily from individual case reports and small case series, this review is subject to inherent selection bias favoring unusual or clinically striking presentations. Additional limitations include heterogeneity of imaging protocols—particularly in diffusion-based modalities—and potential publication bias toward positive or illustrative findings. Consequently, interpretations are intended as hypothesis-generating rather than definitive, and conclusions should be viewed within the context of descriptive, rather than quantitative, synthesis.

The literature search emphasized studies published between 2020 and 2025, with preference for reports using advanced neuroimaging methods such as magnetic resonance imaging (MRI), diffusion tensor imaging (DTI), diffusion-weighted imaging (DWI), and fluid-attenuated inversion recovery (FLAIR). Relevant publications were identified through Google Scholar, using combinations of the search terms “Artery of Percheron infarction,” “pseudobulbar palsy,” “paramedian thalamic infarction,” “corticobulbar tract,” “thalamopeduncular stroke,” and “mesencephalothalamic syndrome.” Reference lists from included articles were also manually reviewed to capture additional pertinent reports.

No formal inclusion criteria, quantitative synthesis, or risk-of-bias assessment was applied. Instead, evidence was qualitatively integrated to develop a conceptual framework describing structural and functional pathways by which AOP infarction may produce bilateral corticobulbar dysfunction. The synthesis emphasizes anatomical plausibility and network-level inference rather than statistical generalization.

Data synthesis followed a narrative and integrative framework emphasizing neuroanatomical mapping of AOP variants and CBT topography, radiological characterization of bilateral thalamic and midbrain ischemic patterns, and clinical correlations between pseudobulbar features and lesion distribution. Mechanistic hypotheses were comparatively analyzed, focusing on direct CBT disruption versus functional cortico-thalamo-bulbar disconnection. Where available, quantitative neuroimaging metrics—such as fractional anisotropy, mean diffusivity, and lesion volume—were descriptively reviewed to identify convergent trends in white matter injury and network disconnection. The synthesis aimed to construct a unified mechanistic model integrating vascular anatomy, motor pathway integrity, and clinical outcome. Because this research involved secondary analysis of previously published data without direct involvement of human subjects, ethical approval was not required or applicable. All data were derived from publicly available, peer-reviewed scientific publications.

LITERATURE REVIEW

Role of Artery of Percheron (AOP)

Despite growing recognition of the Artery of Percheron (AOP) as a clinically important vascular variant, the mechanistic relationship between AOP infarction and pseudobulbar palsy (PBP) remains poorly delineated.[8] Most available studies consist of isolated case reports and small series that document bilateral thalamic or mesencephalic infarctions with variable neurological outcomes, focusing primarily on descriptive clinical features or imaging patterns while neglecting the underlying neuroanatomical and network-level mechanisms responsible for specific syndromes such as PBP. Three principal gaps persist within the literature: first, there is an absence of an integrative mechanistic framework explaining how a single unilateral vascular occlusion can produce a bilateral upper motor neuron syndrome, as most analyses treat AOP infarction as an anatomical rarity rather than a model for supranuclear motor dysfunction. Second, there is limited recognition of functional network disruption, with the concept of functional de-efferentation—in which bilateral thalamic injury interrupts cortico-thalamo-bulbar integration despite intact CBT—remaining underexplored.[9] Third, current research lacks a spectrum-based interpretation of AOP-related syndromes, often categorizing outcomes as either thalamic or midbrain in origin without acknowledging the continuum of ischemic injury dictated by microvascular variability.[10] The present analysis addresses these deficiencies by proposing a dual-mechanism model that integrates direct CBT disruption with functional cortico-thalamo-bulbar disconnection, thereby providing a unified explanation for the emergence of bilateral pseudobulbar symptoms from a single midline lesion. It reframes AOP infarction as a natural experiment in network vulnerability, expanding interpretation beyond focal lesion localization to include dynamic interactions between vascular architecture, neural connectivity, and compensatory failure.[11] By conceptualizing PBP secondary to AOP infarction along a pathophysiological continuum of structural and functional compromise, this work synthesizes fragmented case-based evidence into a coherent explanatory framework that bridges descriptive vascular neurology with mechanistic neuroanatomy, advancing understanding of how focal vascular anomalies can yield complex bilateral neurological deficits.

DISCUSSION

Neuroanatomy of the Corticobulbar System

The corticobulbar tract (CBT) originates in the inferior precentral gyrus and lower primary motor cortex, descending through the genus of the internal capsule and entering the middle third of the cerebral peduncle within the rostral midbrain.[12] These fibers provide bilateral cortical input to cranial motor nuclei, especially those of cranial nerves V, IX, X, XI, and the upper division of VII, creating redundancy that typically preserves bulbar function after unilateral injury.

Within the AOP perfusion territory, both CBTs converge in a compact zone of the paramedian midbrain tegmentum supplied by small perforators from the posterior cerebral and superior cerebellar arteries, including the AOP. Thus, occlusion of this single artery may compromise both corticobulbar tracts simultaneously, creating the neuroanatomical basis for pseudobulbar palsy.

Neuroimaging Spectrum of AOP Infarction

Four principal magnetic resonance imaging (MRI) patterns of Artery of Percheron (AOP) infarction have been described in the literature.[13] These include bilateral paramedian thalamic infarction with midbrain involvement (observed in approximately 35–43% of cases), bilateral paramedian thalamic infarction without midbrain involvement (35–38%), bilateral thalamic infarction with anterior thalamic extension, and bilateral thalamic infarction involving the mammillothalamic tract. On diffusion-weighted imaging (DWI), these lesions typically appear as symmetric hyperintensities within the paramedian thalami, occasionally

extending into the rostral midbrain, while fluid-attenuated inversion recovery (FLAIR) sequences emphasize the subacute components of the infarct. A subset of patients—approximately 30–40%—exhibit the characteristic midbrain "V-sign," a V-shaped hyperintensity along the interpeduncular fossa seen on axial DWI or FLAIR sequences.[14] Although not pathognomonic, this imaging feature is highly suggestive of midbrain involvement and correlates with direct corticobulbar tract injury. For clarity, a schematic figure illustrating AOP vascular anatomy, perfusion territory, and representative imaging patterns is recommended to accompany this section.

Mechanistic Pathways

Structural Disruption of Bilateral CBTs

Infarctions involving the rostral midbrain may directly damage descending corticobulbar fibers as they traverse the central portion of the cerebral peduncles, effectively abolishing bilateral supranuclear input to cranial motor nuclei.[14] This structural disruption compromises the brain's intrinsic redundancy within the corticobulbar system, in which bilateral cortical projections normally ensure preserved motor control following unilateral injury. When both tracts are simultaneously affected, the result is a profound loss of voluntary control over bulbar musculature.

Clinically, these patients often exhibit dysarthria, dysphagia, facial weakness, and pseudobulbar affect, frequently accompanied by hemiplegia, oculomotor disturbances, and vertical gaze palsy due to neighboring involvement of corticospinal and oculomotor pathways. Neuroimaging typically reveals midbrain hyperintensity forming the characteristic "V-sign" along the interpeduncular fossa on DWI or FLAIR, a finding that reflects bilateral tegmental ischemia and correlates closely with pseudobulbar manifestations. Such lesions exemplify the structural pole of the dual-mechanism model, in which direct ischemic injury to both corticobulbar tracts leads to overt and symmetrical pseudobulbar symptoms.

Functional Cortico-Thalamo-Bulbar Disconnection

In contrast, cases of PBP without radiologic midbrain involvement—those limited to bilateral paramedian thalamic infarction—require an alternative explanation.[15] Here, the CBT are anatomically preserved, yet the syndrome manifests fully. This suggests a mechanism of functional disconnection, wherein ischemic injury to key thalamic nuclei (notably the dorsomedial and intralaminar groups) disrupts cortico-thalamo-bulbar relays essential for the modulation and execution of bulbar motor commands.[16]

This phenomenon, termed "functional de-efferentation," denotes a higher-order network failure in which motor commands from the cortex are prevented from reaching brainstem nuclei, not by tract destruction but by loss of integration within thalamic relay loops.[17] This mechanism aligns with neuroimaging findings showing normal midbrain architecture but altered thalamic connectivity on diffusion and functional imaging. It also explains the occurrence of pseudobulbar effect—a symptom more closely tied to network disinhibition than to focal motor tract injury.

Spectrum of Injury and Combined Mechanisms

These two mechanisms—structural and functional—represent poles of a pathophysiological spectrum rather than discrete categories. Many AOP infarctions likely involve elements of both: direct injury to descending fibers and simultaneous disruption of thalamic relays. The relative contribution of each depends on the infarct's epicenter and the individual's microvascular anatomy.[18]

This spectrum concept explains the variability of clinical presentations seen across reported AOP infarction cases. Small shifts in lesion location—mere millimeters—can transform a presentation dominated by altered consciousness into one featuring profound bulbar dysfunction.[19] Hence, PBP secondary to AOP infarction exemplifies how a single strategic lesion can

selectively disable bilateral supranuclear control through overlapping vascular–neural interactions.

Clinical-Radiological Correlation and Mechanistic Validation

Comparative review of published case evidence reinforces the dual-mechanism model. In one report, a 70-year-old woman developed isolated PBP—manifesting as severe dysarthria, dysphagia, and a hypernasal voice—following a confirmed AOP infarction on CT, but notably without gaze palsy or cognitive disturbance.[20] The absence of midbrain findings indicated a selective thalamic lesion, supporting the functional disconnection hypothesis.

Conversely, cases with coma, vertical gaze paresis, and pseudobulbar affect demonstrated MRI evidence of bilateral thalamic and midbrain infarction, confirming that direct tract involvement can reproduce the full PBP phenotype. Still other reports describe mixed patterns, suggesting a combined mechanism at the thalamo-mesencephalic junction. Collectively, these findings validate the proposed continuum model of AOP-related pseudobulbar palsy, bridging the structural and functional domains.

Management and Rehabilitation

Diagnosis of Artery of Percheron (AOP) infarction relies on urgent magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR), which remain the gold-standard modalities for detecting bilateral thalamic or thalamo-midbrain lesions. Complementary vascular imaging using magnetic resonance angiography (MRA) or computed tomography angiography (CTA) may help confirm the ischemic pattern, although the AOP itself is rarely visualized directly. Early recognition is essential, as eligible patients may benefit from thrombolysis or mechanical thrombectomy within standard therapeutic windows. Following the acute phase, rehabilitation focuses on restoring speech and swallowing function and addressing dysarthria, dysphagia, and emotional lability through structured therapy programs. Pharmacologic treatment of pseudobulbar affect, such as with selective serotonin reuptake inhibitors or dextromethorphan–quinidine, may be considered when appropriate.[21] Advanced neuroimaging techniques, including diffusion tensor imaging (DTI) and functional MRI, can be valuable in research or complex diagnostic scenarios for assessing corticobulbar and thalamo-cortical integrity, though they are not yet recommended for routine clinical use.[22] Ultimately, analysis of AOP-related pseudobulbar palsy transforms a rare vascular event into a mechanistic framework for understanding the bilateral dependencies and limitations of motor control in the human brain.

Policy and Systems Considerations

Recent reviews highlight that delayed diagnosis—often exceeding 24 hours—is common in AOP infarction due to its variable clinical profile and subtle imaging findings.[22] In resource-limited settings such as Zambia and comparable health systems, Artery of Percheron (AOP) infarction is often underrecognized due to limited access to advanced neuroimaging. A pragmatic diagnostic protocol may help mitigate this challenge by prioritizing magnetic resonance imaging (MRI) with diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) sequences in cases of suspected bilateral thalamic stroke. When MRI is unavailable, clinical recognition—particularly the triad of altered consciousness, vertical gaze palsy, and pseudobulbar features—should prompt expedited referral to a tertiary center for further evaluation.[23] This approach seeks to balance diagnostic accuracy with resource constraints, thereby reducing delays in recognition and management of this rare but clinically significant vascular syndrome.

Study Limitations: This study, while offering a comprehensive and mechanistic synthesis of pseudobulbar palsy (PBP) secondary to Artery of Percheron (AOP) infarction, has several inherent limitations that should be acknowledged. First, because the analysis is based primarily on published case reports and small clinical series, the limited

sample size constrains the generalizability of findings and may not fully capture the variability in AOP vascular anatomy or clinical outcomes. The potential for selection bias also exists, as published reports often emphasize atypical or severe cases, thereby underrepresenting subclinical or mild presentations. Second, heterogeneity in neuroimaging methodologies across studies—particularly variations in MRI resolution, diffusion tensor imaging (DTI) protocols, and analytic parameters—introduces a degree of measurement variability that may affect the consistency of observed lesions–symptom correlations. Third, the analysis is limited by clinical heterogeneity, as patients with AOP infarction can exhibit diverse neurological deficits, infarct topographies, and comorbidities that complicate efforts to isolate corticobulbar pathway involvement as a singular mechanism. Additionally, publication bias may have influenced the literature pool, favoring reports of positive or striking cases while excluding unpublished or negative findings that could balance mechanistic interpretations. Finally, the interpretation of DTI and other imaging-derived metrics must be approached cautiously, as parameters such as fractional anisotropy (FA) and mean diffusivity (MD) are indirect markers of white matter integrity and cannot alone distinguish between demyelination, axonal injury, or functional disconnection. Integration of standardized imaging protocols, multimodal neuroimaging, and histopathological correlation in future studies would enhance the robustness and translational relevance of mechanistic conclusions regarding corticobulbar pathway disruption in AOP infarction.

Future research should aim to overcome these limitations through prospective, multicenter investigations employing standardized neuroimaging protocols and larger patient cohorts to validate the proposed mechanistic model of pseudobulbar palsy secondary to Artery of Percheron infarction. Incorporating longitudinal diffusion tensor imaging (DTI) and functional MRI (fMRI) could elucidate temporal changes in corticobulbar and thalamo-cortical connectivity, offering insight into both acute injury and neuroplastic recovery processes. Additionally, computational modeling and connectome-based analyses may further clarify the structural and functional thresholds at which unilateral vascular lesions produce bilateral motor network failure. Integration of clinical, imaging, and neurophysiological data within collaborative, multidisciplinary frameworks will be crucial for refining diagnostic criteria, guiding individualized rehabilitation strategies, and advancing understanding of how focal vascular anomalies precipitate complex supranuclear motor syndromes.

In light of these findings, continued investment in the research and development of neuroimaging technologies is essential to advance our understanding of neurological disorders and to facilitate innovation in diagnostic and therapeutic approaches. Overall, this paper underscores the transformative potential of advanced neuroimaging techniques in revolutionizing the diagnosis, treatment, and management of neurological conditions, ultimately improving the quality of life for patients affected by AOP infarction and other related disorders.

CONCLUSION

This narrative review examines the mechanistic relationship between Artery of Percheron (AOP) infarction and pseudobulbar palsy (PBP), clarifying how a single vascular occlusion can produce bilateral supranuclear motor dysfunction through both structural and functional mechanisms. Synthesis of neuroanatomical, clinical, and radiological evidence supports a dual-mechanism model encompassing (1) direct corticobulbar tract involvement within the rostral midbrain and (2) functional cortico–thalamo–bulbar disconnection within the paramedian thalami. This framework positions AOP infarction as a natural model for network-level motor failure, emphasizing the interdependence of vascular architecture and bilateral motor control. Advanced neuroimaging modalities such as diffusion tensor imaging (DTI) and functional MRI (fMRI) may enhance detection

of microstructural injury and network disconnection, contributing to a more comprehensive understanding of stroke-induced pseudobulbar syndromes. Collectively, these findings link vascular topology, corticobulbar pathway integrity, and thalamic network function, providing a coherent explanation for PBP secondary to AOP infarction.

Actionable Recommendations: DTI and related connectivity analyses may be considered in research or complex diagnostic scenarios to evaluate corticobulbar and thalamo-cortical integrity when standard imaging is inconclusive. Correlating diffusion metrics such as fractional anisotropy and mean diffusivity with clinical recovery could help refine prognostic models and validate the dual-mechanism hypothesis. Future studies integrating multimodal imaging, longitudinal follow-up, and connectome-based tractography are needed to delineate dynamic changes in supranuclear motor networks. Collaboration among neurologists, neuroimaging specialists, and rehabilitation teams will be important to translate mechanistic insights into clinically meaningful interventions. A patient-centered approach grounded in individualized neuroimaging profiles may ultimately inform more precise rehabilitation strategies and improve outcomes in supranuclear motor disorders.

Ethics approval and consent to participate

This article is a narrative literature review and does not involve new studies with human participants or animals conducted by the author. Therefore, ethics committee approval was not required. No individual patient data are included.

Consent for publication

Not applicable, as this review contains no individual person's data in any form.

Availability of data and materials

All data discussed in this review are derived from previously published studies. No new datasets were generated or analyzed. The list of reviewed articles is available from the author upon reasonable request.

Competing interests

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Author's contributions

RK conceived the study design, conducted the literature search, synthesized the data, and drafted the manuscript. The author reviewed and approved the final version and agreed to be accountable for its content.

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