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KERNOHAN'S NOTCH PHENOMENON: REVIEW OF LITERATURE AND CLINICAL LESSONS

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Abstract
Kernohan's notch phenomenon is a rare and paradoxical clinical entity characterized by ipsilateral hemiparesis in the presence of a supratentorial space-occupying lesion. This false localizing sign occurs when an expanding mass causes a midline shift and transtentorial herniation, resulting in the mechanical compression of the contralateral cerebral peduncle against the rigid, free edge of the tentorium cerebelli (Kernohan and Woltman, 1929; Neki et al., 2017). Historically, this phenomenon posed significant diagnostic challenges, often leading to surgical interventions on the wrong side of the cranium (Wolf et al., 1995; Murhega et al., 2023). However, advancements in neuroimaging, particularly magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI), have elucidated the pathophysiological substrate of the condition, revealing structural disruptions and signal changes within the crus cerebri (Yoo et al., 2008; Jang and Pyun, 2013). This review synthesizes current knowledge on the historical background, neuroanatomical basis, pathophysiology, and clinical significance of Kernohan's notch phenomenon. It explores various etiologies including traumatic brain injury, primary neoplasms, and rare presentations such as spontaneous intracranial hypotension and sickle cell disease (Sasikala et al., 2014; Ozyigit, 2023). Ultimately, prompt radiological identification and surgical decompression are critical for mitigating permanent motor deficits and improving functional outcomes (Beucler et al., 2022; Chauhan et al., 2024).

Keywords: Kernohan's notch phenomenon, false localizing sign, ipsilateral hemiparesis, transtentorial herniation, cerebral peduncle compression, neuroimaging (MRI/DTI), surgical decompression

Introduction

Clinical localization in neurology typically relies on the established principle that cerebral motor output descends through the corticospinal tract and decussates at the medullary pyramids, meaning a lesion in one hemisphere manifests as motor deficits on the contralateral side (Tataranu, 2025;

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Murhega et al., 2023). Kernohan's notch phenomenon (KWNP) represents one of the most significant exceptions to this rule. It is defined as an ipsilateral motor deficit caused by the compression of the contralateral cerebral peduncle against the tentorial edge due to a mass-occupying lesion (Kernohan and Woltman, 1929; Jang and Seo, 2019). Because the clinical findings misguide the examiner regarding the topographic site of the primary pathology, KWNP is classified as a false localizing sign (McKenna et al., 2009; Neki et al., 2017).

While relatively rare, its recognition is paramount in neurosurgical practice to prevent catastrophic errors, such as performing a craniotomy on the side opposite the true lesion (Wolf et al., 1995; Murhega et al., 2023). The phenomenon is most frequently associated with acute or chronic subdural hematomas and primary brain tumors, but it has also been documented in cases of epidural hematomas, arteriovenous malformations (AVMs), and following neurosurgical complications such as sinking bone flap syndrome (Goyal et al., 2024; Dibble et al., 2015; Lee et al., 2019). Understanding the neuroanatomical and biomechanical factors that predispose certain individuals to this phenomenon is essential for accurate diagnosis and management in neurocritical care (Tataranu, 2025; Beucler et al., 2022).

Historical Background

The early 20th century marked the beginning of modern understanding regarding brain herniation and false localizing signs. James Collier is credited with providing one of the first comprehensive accounts of cerebellar tonsillar herniation as a terminal event in intracranial tumors in 1904, observing that 12.4% of consecutive tumor cases examined clinically and pathologically exhibited false localizing signs (Pearce, 2006). Shortly thereafter, Adolf Meyer in 1920 confirmed the pathological mechanisms of brain herniation, noting that the tentorium constitutes a vital protection between brain compartments but also provides a «rigid opportunity for trouble» during displacement (Pearce, 2006; Neki et al., 2017). Meyer was also among the first to describe hemianopia as a false localizing sign of uncal herniation caused by the strangulation of the posterior cerebral artery (Pearce, 2006).

The specific mechanism of ipsilateral hemiparesis was elucidated in 1929 by James Watson Kernohan and Henry William Woltman at the Mayo Clinic. Through postmortem examinations of 297 patients, they demonstrated that an expanding brain lesion could displace the entire midbrain toward the opposite side, causing the contralateral crus cerebri to be notched by the free margin of the tentorium (Kernohan and Woltman, 1929; Pearce, 2006; Neki et al., 2017). They concluded that this «grooving of the crus» explains the homolateral pyramidal tract signs noted in their cases (Pearce, 2006). In 1938, Sir Geoffrey Jefferson further refined the clinical picture by introducing the term «tentorial pressure cone,» emphasizing the downward herniation of the temporal lobes into the posterior fossa and its effect on the midbrain and posterior cerebral arteries (Pearce, 2006).

Historically, the inability to verify this mechanism in vivo meant that neurologists and surgeons often doubted radiological indexes, assuming scans were mislabeled when motor deficits appeared ipsilateral to the hematoma (McKenna et al., 2009; Murhega et al., 2023). It was not until the advent of MRI in 1990 that Cohen and Wilson provided the first radiographic verification of Kernohan's notch, paving the way for the use of advanced imaging in contemporary diagnosis (Lin et al., 2022; Neki et al., 2017).

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Neuroanatomical Basis

A thorough understanding of the corticospinal tract (CST) and the anatomy of the tentorial hiatus is required to interpret KWNP. The motor pathway originates in the primary motor and premotor cortex, with axons descending through the corona radiata and the internal capsule to reach the cerebral peduncles (Tataranu, 2025; Neki et al., 2017). Within the midbrain, these motor fibers are located in the crus cerebri. The critical anatomical juncture occurs at the lower end of the medulla, where approximately 75% to 90% of CST fibers decussate to form the lateral corticospinal tract, which controls the voluntary movement of the limbs on the contralateral side (Tataranu, 2025; Murhega et al., 2023).

The tentorium cerebelli is a rigid dural structure that separates the occipital lobes from the cerebellum. The tentorial notch, or incisura, is the only opening through which the brainstem can pass to connect the supratentorial and infratentorial compartments (Tataranu, 2025; Pereira, 2019). Variations in the anthropometry of this notch play a significant role in susceptibility to KWNP. Adler and Milhorat's morphometric analysis of 100 human autopsies classified tentorial notches into narrow (24.5–27 mm) and wide (32–39 mm) types (Tataranu, 2025; Lee et al., 2019). It has been speculated that individuals with narrow tentorial incisura are more prone to KWNP, as even a minor lateral displacement of the midbrain brings the contralateral crus cerebri into contact with the sharp, rigid dural edge (Tataranu, 2025; Lee et al., 2019). When the midbrain is pushed against this edge at a level *above* the medullary decussation, the resulting motor deficit is manifested on the same side as the original lesion (Kernohan and Woltman, 1929; Pereira, 2019; Murhega et al., 2023).

Pathophysiology

The pathophysiology of KWNP is best understood through the Monro-Kellie doctrine, which states that the total volume of brain tissue, blood, and cerebrospinal fluid (CSF) within the rigid skull remains constant (Rahimi-Movaghar et al., 2025; Ozyigit, 2023). When a space-occupying mass, such as a traumatic epidural or subdural hematoma, develops, the initial compensatory phase involves the displacement of CSF and reduction in venous volume (Rahimi-Movaghar et al., 2025). As the hematoma grows and autoregulation fails, intracranial pressure (ICP) rises linearly, leading to the physical displacement of brain tissue (Rahimi-Movaghar et al., 2025; Chauhan et al., 2024).

The classic progression involves uncal herniation, where the medial temporal lobe shifts downward and inward over the tentorial edge (Rahimi-Movaghar et al., 2025; Pereira, 2019). This shift typically results in ipsilateral oculomotor nerve compression and contralateral hemiparesis. However, in KWNP, the lateral force is sufficient to displace the entire diencephalic-mesencephalic axis, twisting the brainstem and widening the ipsilateral space of Bichat (Pearce, 2006; Tataranu, 2025). The contralateral cerebral peduncle is then squeezed against the tentorial edge, causing mechanical injury, cytotoxic edema, and sometimes permanent necrosis of the motor fibers (Jang and Seo, 2019; Ozyigit, 2023).

Secondary vascular compromise often accompanies this mechanical compression. Compression of the posterior cerebral artery (PCA) can lead to ipsilateral occipital lobe infarction and homonymous hemianopia (Rahimi-Movaghar et al., 2025; Leever, 2020). Furthermore, more severe variants of KWNP have been described where unilateral herniation results in combined contralateral superior cerebellar artery (SCA) territory infarction and mesencephalic injury (Leever, 2020). If herniation

remains untreated, the anteroposterior elongation of the midbrain can cause the tearing of paramedian perforating vessels, resulting in Duret hemorrhages, which are severe prognostic indicators (Dibble et al., 2015; Rahimi-Movaghar et al., 2025).

Clinical Manifestations

The hallmark clinical presentation of Kernohan's notch phenomenon is the paradoxical motor deficit—hemiparesis or hemiplegia—ipsilateral to the side of the primary intracranial lesion (Neki et al., 2017; Pereira, 2019; Murhega et al., 2023). This deficit is often accompanied by an altered level of consciousness, ranging from lethargy and drowsiness to deep coma, depending on the severity of midbrain compression and its impact on the ascending reticular activating system (Lee et al., 2019; Rahimi-Movaghar et al., 2025).

Pupillary abnormalities are frequently present and serve as vital diagnostic clues. The most common finding is an ipsilateral fixed and dilated pupil (mydriasis) caused by the compression of the oculomotor nerve on the same side as the lesion (Rahimi-Movaghar et al., 2025; Neki et al., 2017; Murhega et al., 2023). However, because the third nerve can also be compressed at the level of the tentorium on the contralateral side, practitioners may occasionally observe contralateral mydriasis, further complicating the clinical picture (Pellegrini et al., 2022). Other cranial nerve involvements, such as ipsilateral central facial palsy due to the compression of corticobulbar fibers, have also been reported (Murhega et al., 2023).

Atypical manifestations of KWNP include movement disorders. Cases have been documented where patients presented with intention tremors or parkinsonism (Sasikala et al., 2014; Evans et al., 2004). Hemiparkinsonism secondary to KWNP is thought to result from nigrostriatal degeneration caused by the mechanical «notching» of the midbrain, sometimes manifesting as a delayed complication months after the initial injury (Ueda et al., 2021; Evans et al., 2004). Additionally, in the setting of traumatic epidural hematomas, the «talk and die» syndrome may occur, where a patient initially appears alert before suddenly deteriorating due to rapid expansion and herniation (Rahimi-Movaghar et al., 2025).

Neuroimaging Findings

Neuroimaging is essential for confirming the clinical suspicion of KWNP and guiding surgical management. Computed tomography (CT) is typically the first-line modality in emergency settings, capable of identifying the primary lesion (e.g., a crescent-shaped subdural hematoma or biconvex epidural hematoma), significant midline shift, and effacement of the basal cisterns (Pereira, 2019; Murhega et al., 2023; Chauhan et al., 2024). While CT can demonstrate the mass effect, it often fails to clearly visualize the «notch» or intrinsic brainstem damage unless the injury is severe (Pereira, 2019; Lee et al., 2019).

Magnetic resonance imaging (MRI) is the gold standard for diagnosing KWNP, providing superior resolution of the brainstem structures. The characteristic radiological finding is a T2 and FLAIR hyperintense signal within the contralateral cerebral peduncle, often exhibiting a peripheral triangular morphology (Pereira, 2019; Tataranu, 2025; Beucler et al., 2022). In the acute stage, T1-weighted images may show hypointensity with variable diffusion restriction on diffusion-weighted imaging (DWI), reflecting cytotoxic edema (Beucler et al., 2022; Leever, 2020). Chronic cases may

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demonstrate leukomalacic changes or residual signal abnormalities (Yoo et al., 2008; Jang and Seo, 2019).

Advanced techniques such as DTI and fiber tractography (FT) allow for the in vivo evaluation of the integrity of white matter tracts. Studies have shown that KWNP is characterized by a drastic narrowing or interruption of the CST fibers at the level of the pons and midbrain (Jang and Seo, 2019; Yoo et al., 2008). DTI parameters, specifically a decrease in fractional anisotropy (FA) and an increase in the apparent diffusion coefficient (ADC), serve as objective measures of axonal damage in these patients (Yoo et al., 2008; Lim et al., 2021). Furthermore, recent reports have highlighted the utility of 18F-FDG PET/CT, which can demonstrate increased glucose metabolism and neuronal function alteration in the affected cerebral peduncle, offering microstructural confirmation of the injury (Lin et al., 2022).

Differential Diagnosis

The primary challenge in managing KWNP is differentiating it from other causes of ipsilateral motor impairment. A key differential is a «true» localization, where a lesion might exist in the contralateral hemisphere that was not initially identified, such as an undiagnosed stroke or secondary contusion (Neki et al., 2017; McKenna et al., 2009). For instance, Pangaribuan et al. (2024) reported a case mimicking KWNP where a patient with a left epidural hematoma and left hemiparesis was found to have a concomitant malignant right middle cerebral artery (MCA) infarction. Similarly, anatomical variations must be considered. While extremely rare, a complete lack of pyramidal decussation can result in a «true» ipsilateral motor deficit from a supratentorial lesion, which can only be confirmed via DTI or motor-evoked potential studies (Tataranu, 2025; Lim et al., 2021).

Other theories for ipsilateral hemiparesis include the concept of diaschisis, where a focal injury causes a distant, sudden decline in neuronal synaptic function due to a loss of afferent connections (Tataranu, 2025). Interhemispheric diaschisis, involving the corpus callosum, could potentially explain some cases of paradoxical weakness (Tataranu, 2025; Neki et al., 2017). Furthermore, Léon Ectors described the «syndrome of the third frontal convolution,» specifically associated with large pterional meningiomas. Ectors distinguished «early» or "precocious" ipsilateral hemiparesis—which involves the leg first and resolves completely after surgery—from later manifestations, suggesting that early cases may result from elastic deformation rather than permanent structural damage (Tataranu, 2025).

Clinical and Surgical Implications

The primary surgical implication of Kernohan's notch phenomenon is the risk of «wrong-side» surgery. In the pre-imaging era and in contemporary emergency scenarios where records are incomplete, a clinician relying solely on the physical examination might conclude that the lesion is contralateral to the hemiparesis (Wolf et al., 1995; McKenna et al., 2009). There are documented cases where surgeons, assuming a scan was mislabeled, performed craniotomies on the side opposite the motor deficit, only to find a healthy hemisphere (Wolf et al., 1995; Murhega et al., 2023). Therefore, clinicians must maintain a high suspicion for KWNP whenever a paradoxical deficit is encountered, and urgent neuroimaging is mandatory to prevent surgical errors (Murhega et al., 2023; Chauhan et al., 2024).

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Management of KWNP is centered on the prompt evacuation of the primary space-occupying mass to relieve midbrain compression (Rahimi-Movaghar et al., 2025; Murhega et al., 2023). Surgical options include craniotomy for complete evacuation or emergency burr holes when rapid intervention is needed (Rahimi-Movaghar et al., 2025; Alhassan et al., 2018). For cases involving epidural hematomas, surgery is indicated if the hematoma volume exceeds 30 cm^3 or if there is a progressive decline in GCS and anisocoria (Rahimi-Movaghar et al., 2025). In patients with spontaneous intracranial hypotension (SIH) complicated by SDH, the management is more complex, as hematoma evacuation prior to addressing the underlying CSF leak can sometimes exacerbate downward brain sagging (Ozyigit, 2023). In such cases, Trendelenburg positioning and epidural blood patches may be necessary adjuncts (Ozyigit, 2023). Similarly, in patients with uremic syndrome from chronic kidney disease, surgical intervention must be balanced with the risks of uremic bleeding and rigorous blood pressure control (Negrão et al., 2025).

Prognosis

The functional prognosis for patients with Kernohan's notch phenomenon is highly variable and depends on the duration and severity of midbrain compression. Literature reviews indicate that approximately 77% of patients show improvement in motor function after prompt surgical evacuation, with roughly 27% achieving full neurological resolution (Beucler et al., 2022; Lin et al., 2022). Good outcomes have been reported in many case series where patients recovered full strength within weeks to months post-surgery (Murhega et al., 2023; Alhassan et al., 2018; Chauhan et al., 2024).

However, some patients suffer from persistent neurological dysfunction. The presence of significant signal changes on MRI, such as T2 hyperintensity in the cerebral peduncle, often correlates with a lower probability of full functional recovery, as it indicates permanent reactive gliosis and demyelination within the CST (Yoo et al., 2008; Lin et al., 2022). Jang and Pyun (2013) demonstrated that clinical recovery patterns can differ drastically; while some patients achieve excellent improvement due to the alleviation of tissue swelling, others with substantial neuronal loss show limited recovery even after 15 months. In rare instances, the mechanical injury can lead to long-term sequelae like hemiparkinsonism, which may respond to anticholinergic therapy even if response to L-dopa is poor (Ueda et al., 2021; Evans et al., 2004). Early detection through advanced modalities like PET/CT may serve as an important reference for predicting the probability of functional recovery by identifying neuronal activity and neuroinflammation (Lin et al., 2022).

Conclusion

Kernohan's notch phenomenon remains a critical and fascinating clinical entity in neurosurgery and neurology. As a rare false localizing sign, it challenges the traditional axioms of neuroanatomical localization and emphasizes the necessity of integrating thorough physical examinations with advanced neuroimaging. The pathophysiology centers on the mechanical compression of the contralateral crus cerebri against the tentorial edge, a process driven by massive supratentorial mass effect and individual anatomical susceptibility. While historically elusive, the development of MRI and DTI has provided definitive evidence of the structural and functional disruptions underlying this paradoxical sign.

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The clinical significance of the phenomenon lies in its potential to lead to misdiagnosis and wrong-side surgery, particularly in emergency settings. Practitioners must recognize that ipsilateral hemiparesis, often coupled with ipsilateral mydriasis, should trigger immediate investigation into the possibility of a contralateral mass. Prompt surgical decompression remains the only effective treatment, with the speed of intervention being a primary determinant of functional prognosis. Despite the potential for permanent motor deficits and rare movement disorders, early identification and management offer a substantial chance for meaningful neurological recovery.

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