The History of the Kernohan Notch Revisited

The paradoxical phenomenon of ipsilateral hemiplegia accompanying expanding brain lesions is commonly known as the Kernohan notch.1 James Watson Kernohan (1896-1981) and his coauthor, the neurologist Henry W. Woltman (1889-1964), both working at the Department of Anatomic Pathology at the Mayo Clinic in the United States, published their work on this remarkable phenomenon in 1929 in the Archives of Neurology and Psychiatry.1 Since then, this publication has been considered a classic article. Kernohan and Woltman mentioned that “since we completed our review of the literature in preparation for this report, a recent and noteworthy contribution came to our notice,” referring to an 18-page case report by the Dutch neurologist Arnold Groeneveld (1895-1962) and the German neurologist Georges Schaltenbrand (1897-1979), published 2 years earlier in 1927 in the Deutsche Zeitschrift für Nervenheilkunde.2 This article by Groeneveld and Schaltenbrand has never received full credit for providing the first actual detailed description of the Kernohan-Woltman phenomenon, currently better known as the Kernohan notch, most likely because it was written in German.

HISTORICAL CONTEXT

The research by Kernohan and Woltman in the 1920s was performed when neurological examination was the key to unlocking the location of the neurological pathology because advanced radiology was still in its infancy. At that time, only skull roentgenography, ventriculography, and pneumoencephalography were available for investigating intracranial pathology. Carotid angiography had been introduced by Moniz3 but was not widely used because of its invasiveness and high rate of significant complications at that time.

Neurological examination was notoriously difficult, as beautifully stated by Cushing in his 1904 address before the Academy of Medicine of...
Cleveland on the Special Field of Neurological Surgery: “...neurology was the *pons asinorum* (pons asinorum: Latin—asses’ or fools bridge; a critical test of ability or understanding; stumbling block) of the medical curriculum. ... Special training is necessary before one can obtain a working knowledge of the underlying pathological processes, without which the clinical superstructure is built upon sand.”

Interestingly, it was the famous Brown-Séquard some 3 decades earlier who openly questioned the dogma that a lesion on one side of the brain always results in symptoms on the other side.5-7

In the early days of neurosurgery, it was therefore not uncommon that a surgeon engaging in brain surgery would be guided by a neurologist to note the planned trephination. Nevertheless, negative explorations were not uncommon, to some extent as a result of false localizing signs.6 Percentages as high as 12% to 40% had been reported, which must have had a significant impact on the diagnosis and outcome of those cases.8,9 Oppenheim10 had noted that collateral hemiplegia had led to many failures of surgical therapy but offered no explanation or suggestion as to what the origin of this phenomenon could be. In his 1879 seminal work, Nothnagel11 also warned the reader of “diagnostische irrthümer in der localisirung” (misdiagnosis as a result of localization). Then, Bramwell12 wrote in 1899, “I may say that as my experience of intracranial tumors increases, I become more and more cautious in drawing conclusions from clinical data as to the exact position of the new growth.”

**KERNOHAN AND WOLTMAN’S 1929 ARTICLE**

Kernohan and Woltman studied 276 patients with intracranial tumors and found ipsilateral pyramidal signs in 24 cases (8.7%), of which only 3 actually led to false localization. They described their findings in their 1929 publication.1 Collier9 had already shown that false localizing signs were observed in 20 of 161 patients (12.4%). In his classic 1904 paper, he had actually described the Kernohan notch as “alterations in the position of the brain stem may also be demonstrated post mortem by the deep indentation of the crus cerebri by the free edge of the tentorium.”

There were, however, no figures of autopsy findings to substantiate his conclusion. Although better understood and imaged with diffusion tensor magnetic resonance imaging, for example, the Kernohan notch continues to lead to confusion and misdiagnosis, despite modern imaging technology.13-16

**GROENEVELD AND SCHALTENBRAND’S 1927 ARTICLE**

In recent historical notes,17,18 the very detailed article by Groeneveld and Schaltenbrand is inaccurately described as “another early, brief case report.” This description gives almost no credit to the work by Groeneveld and Schaltenbrand,2 although the case is well and exhaustively documented from the initiation of neurological symptoms to the patient’s death. Furthermore, a precise anatomical and histological description of the lesion to the cerebral peduncle is provided, together with a rational discussion on the relationship to the clinical consequences. Recently, the article of Groeneveld and Schaltenbrand has received increasing attention in a historical article on the neurological contributions of Kernohan,19 but its importance was still not fully appreciated.

In short, Groeneveld and Schaltenbrand2 described a case of an otherwise healthy 43-year-old man who presented with right-sided Jacksonian seizures over a period of 10 years. For 3 years, he complained of continuous headaches. Four months before the neurological consultations, he developed progressive left-sided spastic hemiparesis, and bilateral papillary edema was noted. In a subsequent period of 12 months, during which time the neurological examination was noted on many occasions, a clear deterioration was observed; only in the last months before his death did the patient also develop right-sided spastic hemiparesis.

At autopsy, “an endothelioma the size of a little apple” was found near the left sylvian fissure (Figure 1), involving “Brodmann-areas 1 to 4, 6, 9, 22, and 40 to 43.” Figure 1 shows the left-sided view of the patient’s brain, including a meningoia with a remnant of dura. Several coronal sections were obtained (I-VI in Figure 1). Section V just behind the mammillary bodies and just before the posterior end of the tumor indicated that the edema as a result of this tumor was such that it had displaced the brain toward the right, resulting in the stretching of the internal capsule on the left side and the production of a pressure notch in the pes pedunculi on the right side by the free tentorial edge (Figure 2).

Nissl staining of a necrotic part of the cerebral peduncle at the crus cerebri showed necrosis, and old and recent tissue reorganization with migration of fibroblasts was observed. The authors concluded that this pressure caused by the knifelike...
mechanism (“Messerähnlichen Wirkung”) of the tentorial edge was responsible for the ipsilateral hemiparesis. They also state that the posterior face of the right-sided petrosal part of the temporal bone, to which the brainstem could have been compressed, might also have contributed to the situation.

In 1936, in his doctoral thesis on meningioma, the Dutch neurosurgeon Arnoud C. De Vet (1904-2001) described a patient of Groeneveld and Schaltenbrand, mentioning that their report is so good that repeating all of their findings is not necessary. De Vet ends the section by explaining that Kernohan and Woltman had seriously and thoroughly researched the problem and had proven the mechanism described by Groeneveld and Schaltenbrand in a larger series, giving full credit for the first detailed description of the phenomenon to Groeneveld and Schaltenbrand (Figure 3).

DISCUSSION

The first report on the paradoxical phenomenon of ipsilateral hemiplegia accompanying expanding brain lesions commonly known as Kernohan notch was published by Magnan in 1878 to 1879 in Brain. Magnan described a patient with a tumor in the left rolandic fissure, which resulted in left-sided convulsions. Later, in 1917, Tucker described a patient with a left temporal tumor resulting in left-sided hemiplegia as part of a study of 15 patients with brain tumors of obscure localization. Three years later, Meyer published a well-illustrated pathological article on brain herniation, describing a patient with a supratentorial tumor illustrating the phenomenon. In all of these publications, the authors failed to provide a satisfactory explanation of the phenomenon, although Meyer has to be credited for confirming falcine, tentorial, and foramen magnum herniation. The first to explain the paradoxical phenomenon of ipsilateral hemiplegia were Groeneveld and Schaltenbrand in their above-mentioned publication from 1927. They deserve proper credit for their research and creative thinking.

Kernohan and Woltman’s subsequent report shed more light on the phenomenon of ipsilateral paresis as a result of an intracranial lesion but failed to include descriptions of pathological specimens. Furthermore, none of the mentioned authors explain the phenomenon. Is it a direct lesion with a loss of substance? Is it due to compression resulting in vascular ischemia? Is it a contusion in the mesencephalon? In his thesis, de Vet mentions that the posterior cerebral arteries were intact, thus making the vascular injury theory somewhat less credible. The lesion, as concluded by all authors before Groeneveld and Schaltenbrand, seemed to be related to local injury and compression. Macroscopically, it more strongly resembled a contusion. This could be due to actual mechanical injury of the structures as a result of long-acting compression or injury to the perforating arteries caused by compression and a brainstem infarction of a very small area, that of the corticospinal tract running ventrally in the brainstem at the level of the pedunculi cerebri.

CONCLUSION

The credit for the first accurate, thorough, logical explanation of the phenomenon observed by several scholars but never completely elucidated goes to Groeneveld and Schaltenbrand. Their publication takes us through a structured, detailed train of thought and produces hard evidence, which was lacking at that time. Kernohan and Woltman confirmed Groeneveld and Schaltenbrand’s findings and validated the phenomenon in a larger series.
To speculate, it might not even have been their intention to “prove” the mechanism but rather to confirm it in a larger series. Indeed, in the reviewed literature, they mention the “recent and noteworthy” contribution of Groeneveld and Schaltenbrand. Furthermore, Kernohan and Wolman themselves bestow the recognition and appreciation on this article that it has unjustly not received in the more recent era.

This will not change the way we see and name this phenomenon, but recognition is the least credit that the article can receive, given that it has spent the time in the limelight but has been obscured from view, always there but never acknowledged for what it truly is.

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

REFERENCES


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COMMENTS

U ncovering the explanation for false localizing signs has played a crucial role in the evolution of the neurosurgical and neurologic understanding of neuroanatomy, an effort that culminated at the end of the 19th century. In this important historical article, the authors study the history of our understanding of the Kernohan notch, a classic case of a landmark explanation of a simple process that baffled neurologists throughout the 19th and the beginning of the 20th century. The authors of the present work recall that Groeneveld and Schaltenbrand had already offered the explanation for this phenomenon 2 years before Kernohan in an exhaustive article. Rectification of the attribution of named phenomenon is an important function of historic articles like this, and the authors are to be commended for the thoroughness with which they investigated the history of this false localizing sign.

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