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False localizing signs in traumatic brain injury

CRISTIN MCKENNA, JONATHAN FELLUS, and ANNA M. BARRETT

Kessler Institute for Rehabilitation, West Orange, NJ, USA

Abstract

Background—Hemiparesis ipsilateral to a mass-occupying lesion can be due to Kernohan-Woltman Notch Phenomenon (KWNP). This syndrome implies a false-localizing sign because clinical findings lead the examiner to an incorrect neuroanatomical diagnosis. The contralateral crus cerebri (pyramidal tract) is pressed against the tentorial incisum and a resultant hemiparesis is found on the same side of the lesion.

Review—A detailed literature search of false-localizing signs is presented.

Conclusions—Not infrequently, patients presenting to a physiatrist may have incomplete records. The existence of false localizing signs may point the physician towards the wrong underlying pathology.

Keywords

Rehabilitation; traumatic brain injury; hemiparesis; subdural haematoma

False localizing lesions

The traditional methods of teaching medical neurology rely heavily on textbook neuroanatomy and academic testing determines students' ability to relate the neuroanatomy to loss of function. Signs observed or elicited during the neurologic examination may be thought to be specific localizing signs if their presence indicates a lesion at a specific area in the brain or nervous system [1]. It is true that clinical signs often provide a clear idea of the site and likely nature of a pathologic process. This is a valuable tenet but not clinically infallible.

A false localizing sign implies a situation where the physical exam does not point the investigator towards the appropriate location of the lesion. A more nuanced view of the term accepts that the 'falseness' of the sign may be removed as more knowledge is gained and the mechanism producing the sign is better understood, thus the correct interpretation and localization is attached to it. The falseness enters with the interpretation of the findings, not that the finding does not exist [2]. One of the best-known false-localizing sign is that of the non-specific nature of sixth nerve palsy which is widely associated with elevated intracranial pressure from any cause [3].

Ocular false localizing signs

Ocular signs can be false localizing. Papilloedema cannot be reliably used as an indication of elevated intracranial pressure because the leading cause of optic disk swelling in late life is not

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Correspondence: Cristin McKenna, Kessler Institute for Rehabilitation and Kessler Foundation, 1199 Pleasant Valley Way, West Orange, NJ 07052, USA. Tel: 973 324 3552. Fax: 973 243 6861. cmckenna@kmrrec.org.

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elevated intracranial pressure but ischemic optic neuropathy [3]. Even signs which have attained status as neurological axioms, for example, that unilateral fixed dilation of the pupil occurs ipsilateral to a supratentorial mass, have been demonstrated in published cases to be, on occasion, false localizing [3,4]. Therefore, unilateral fixed pupillary dilation is not a reliable measure of uncal herniation [3]. The actual incidence of ocular motor disturbance is unknown because the poor condition of patients who present with serious intracranial pathology frequently limits the neuroophthalmological examination [5].

Intracranial tumours and false localizing signs

Intracranial tumours and their false localizing signs are the subject of both the initial work in KWNP and much subsequent investigation. In a review of 250 cases of meningioma, there were 101 cases which had false localizing signs. These false localizing signs included visual field deficits, cranial nerve palsies of the first, third, fifth, sixth, seventh and eighth cranial nerve and many others including cerebellar signs, pyramidal signs and neck stiffness [6]. In some cases, several false localizing signs can be seen at once, such as a left cerebellar hemangioblastoma presenting with right fifth, seventh, eighth, ninth and tenth cranial nerve palsies and right-sided cerebellar signs [7].

Recognizing the signs and symptoms of intracranial pathologies can have significant impact on diagnosis and treatment of patients. The ability to recognize impending brain herniation by observation of false localizing signs and detection of hyperaemia by measuring tagged red blood cells has been investigated [8]. The study by Gelmers and Beks [8] underscored the possible mechanism for some false-localizing lesions. Namely, a mass lesion may create remote vascular strangulation which then can elicit a cycle of hypoxemia-lactic acidosis-hyperaemia which can cause damage during post-hypoxic perfusion. This post-hypoxic perfusion is also the basis for the remote hyperaemia they measured. Ehni [2], in 1950, outlined the main mechanisms by which a single intracranial tumour may give rise to false localizing signs. He names many mechanisms including hydrocephalus, meningitis, shifts of cerebral tissue distorting distant nerves and blood vessels and entrapment of neural tissue by distant brain herniation.

Cranial nerve false localizing signs

Commonly reported false localizing cranial nerve palsies are abducens, trigeminal (either palsy or neuralgia) and oculomotor. Most commonly, a single cranial nerve is affected, though several can be affected simultaneously [9]. False localizing lesions of the fifth cranial nerves can present as palsy or neuralgia [2,10–12].

Kernohan's notch

Hemiparesis contralateral to a lesion is contrary to the classic picture of transtentorial herniation, which results from the herniated medial temporal lobe descending into the tentorial hiatus. The classic clinical picture, from a neurologic viewpoint, is ipsilateral pupil mydriasis, contralateral hemiparesis (sometimes bilateral) and abnormal extensor posturing [13].

James Watson Kernohan, born in Ireland and educated at both Queen's University of Belfast in Northern Ireland and Crichton Royal Institute in Scotland [14], published extensively in both pathology and neuropathology at the Mayo Clinic [15]. In 1929 he published a now-classic work with Henry William Woltman [16], a fellow professor at the Mayo Clinic. Through post-mortem examination of patients with tumours, they proposed that in patients with hemiparesis homolateral to a cerebral hemispheric mass, the downward pressure caused compression of the contralateral incisura of the crus cerebri by the tentorial edge [17]. This incisura is now dubbed Kernohan's Notch (sometimes Kernohan-Woltman Notch). The resulting clinical

picture is dubbed Kernohan's Notch Syndrome, sometimes called Kernohan-Woltman Notch Phenomenon (KWNP).

The original data for KWNP was obtained from post-mortem examination of patient's brains [17]. Until relatively recently, the mechanism was unable to be verified by imaging. Since Kernohan and Woltman's original publication, KWNP has been observed and in many clinical settings [18,19] and has been examined using new imaging techniques such as computerized tomography (CT) [20], magnetic resonance imaging (MRI) and electrophysiologically, using transcranial electrical motor evoked potentials [21]. These studies have largely supported Kernohan's original proposed mechanism. The reason why some patients present with a 'classic' transtentorial herniation clinical picture and some with KWNP is not precisely understood but may be due to variations in the structure of the tentorial notch [22]. There is some speculation that KWNP is due to the component of the uncrossed corticospinal tract (anterior corticospinal tract) and anatomic variation amongst individuals determines those susceptible to KWNP.

Older literature examining intracranial pathology emphasized the need for neuroradiological investigations [23]. Imaging of Kernohan's notch by MRI has been reported [24,25]. Both Cohen and Wilson [24] and Mastronardi et al. [25] discuss traumatic right-sided intracranial haemorrhages leading to right-sided hemiparesis. Mastronardi et al. report that an MRI showed a small area of abnormal signal intensity in the left cerebral peduncle. Cohen and Wilson report that MRI showed an increased signal in the right cerebral peduncle. MRI has also been used to support at least one case where new-onset Parkinsonism was proposed to be due to KWNP, causing isolated nigrostriatal dysfunction [26].

Challenges in localizing brain lesions

Early methods of location involved direct cortical stimulation of human or animal models. However, these had well-recognized limits as the physical or electrical stimulation of a region of the brain is not the same as physiologic activation of a brain structure or function [27]. Newer methods of functional imaging which include positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) attempt to produce functional images which can be combined with detailed anatomical images to better understand both normal and injured brain function [28]. The limits, well recognized in the field, include but are not limited to the difficulty in transposing the functional images of individual subjects into a meaningful template so that intersubject comparisons can be made. Integration of anatomic brain atlases with functional and electronic brain atlases with generation of a patient specific atlas is of great importance to the field of intracranial surgery.

There's a conjectural leap when determining the location of a lesion, which produces a similar dysfunction in different patients (i.e. a lesion in Broca's or Wernicke's areas) and stating that this area is therefore the site of that function. That particular locations in the brain are crucial to functions is generally well accepted. However, backtracking from lesions to normal function is not scientifically sound (tempting though it certainly is). For many well-documented lesions causing dysfunction, there are exceptions, which may represent anatomic or physiologic variations in individuals. Other exceptions likely stem from other neuronal pathways serving to replace the injured brain tissue [29]. Updated methods of measurement attempt to recognize that differences in motivation for actions may have significantly different brain mechanisms. Thus, new methods of measurement include activity contemplation (where the subject contemplates an action but does not move or speak), command activity (the subject moves or speaks as directed by an examiner) and amputated limb activity (amputees are asked to attempt to 'move' an amputated limb) [27].

Discussion

Importance of false localizing lesions to the clinician has a different role in the rehabilitation setting as opposed to the acute care setting where, in at least two published cases after the advent of head CT, the failure to include KWNP in the differential diagnosis led to a patient receiving a craniotomy on the skull contralateral to a haematoma. In this case, the CT was accurate but was believed to be mislabelled because the site of the haematoma was apparently 'not fitting the clinical picture' of hemiparesis ipsilateral to the haematoma [20]. Another case outlined a brain surgery in which it was assumed that the scan had been incorrectly labelled because multiple false localizing signs led surgeons to suspect strongly a specific side for a given clinical presentation [30]. It should be noted that in both of these published reports, the patient did eventually receive appropriate-sided surgical intervention after the initial surgery on the unaffected side.

Localization of function and better understanding of loss of function has meaningful clinical implications. The ability to integrate physical exam findings with diagnostic test data can be crucial to therapy decisions in the rehabilitation setting. Knowing if an injured part of the brain represents an area of functional specialization or an area of equipotential representation [31] may have a profound effect on therapy, as it may lead the physiatrist to direct attempts towards regaining the lost function vs. teaching compensation for the lost function. Acute spinal cord injury patients represent one real world example of this dilemma. In an acutely injured patient with paraplegia and in the current medical setting where length of stay is curtailed, the physician needs to know whether to focus on transfers and wheelchair mobility vs body weight supported tread-mill training with an eye towards walking.

Other investigations into the specificity of response to therapy and the possibility of false-localizing interventions may have significant impact on the emerging evidence-based medicine portion of Physical Medicine and Rehabilitation (PM&R). For example one prospective, randomized study of sciatica found that local anaesthetic blocks are of extremely limited predictive value in either diagnosis or predicting patients' response to therapy or surgery [32]. Other false localizing lesions of the spinal cord of particular importance to the rehabilitation physician include patients with cervical compressive myelopathy and without thoracic spinal cord involvement, presenting with mid-trunk unpleasant sensations [33]. These patients have been described with lesions above cervical level four presenting with false-localizing signs such as finger and hand dyesthesias and hand atrophy [34,35]. These represent lower motor neuron signs as a false localizing sign of upper cervical spinal cord lesions. Misdiagnosis can include brachial plexopathy, shoulder dysfunction and viral syndromes [34]. Putative mechanisms include ischemia in the watershed area of the anterior spinal artery in the thoracic cord [33,34].

Besides the neuroanatomical correlations, the neurophysiological aspects may also be important. Ipsilateral motor signs may be a diaschitic phenomenon (deafferentiation), especially in cases without cerebral herniation [36]. Interhemispheric diaschisis may cause weakness in patients, particularly those with a left hemisphere lesion, as there is evidence that left-sided cortical regions play a critical role in mediating motor function in both the ipsilateral and contralateral body, for both fundamental and skilled learned movements [37,38].

Conclusions

Physical examination at admission to a rehabilitation facility seldom establishes the aetiologic diagnosis. Detailed knowledge of false localizing signs helps the rehabilitation team avoid misdiagnosis and better care for the patient. Physical examination at admission guides treatment and may illuminate subtle defects not previously noted. It may be appropriate to

adjust the established aetiological diagnosis and elaborate or establish the functional diagnosis to guide treatment and to project prognosis that could not be appreciated or addressed in the acute care setting. No studies describe prognosis of recovery from KWNP or other false-localizing signs but long-term recovery from moderate to severe traumatic brain injury, of types similar in severity to most KWNP patients, indicate persisting impairments both 6–18 months after injury and many years later [39].

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