



False Localizing Signs in Neurology – A reflection of True Pathology

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Abstract

False localizing signs in neurology have always fascinated the students and experts alike. Neurology has seen the days when only autopsy was the method to exactly define the lesion, and now, its flooded with the structural imaging techniques which can demonstrate “near live” anatomy. But still false localizing signs have not lost the significance, neither in guiding the physician, nor in misguiding too. This article is yet another attempt to review this fascinating phenomenon in neurology highlighting some historical points and clinically applicable points of false localizing signs.

Keywords: False Localizing Signs; Idiopathic intracranial hypertension; Intracranial Herniations; James Collier; Kernohn Notch.

To become comfortable with uncertainty is one of the primary goals in the training of a physician. -Sherwin B. Nuland

Introduction

Like other fields of sciences, medicine is also full of exceptions, uncertainties, contradictions and things that defy the logic of common sense. There are situations when two plus two does not make four. False localizing signs are such situations in clinical medicine, more specifically said, in neurology. They are false only in the sense that they don't take the clinician to the actual site, but

they are not at all useless signs, or only misguiding forces in the science of medicine. Only in the hands of a beginner or to a mind that is fed upon the dosages of certainty (and now a days the imaging techniques are overdosing the certainty!), these signs become a source of discomfort and make the diagnosis obscure or false. While, In experienced hands, they can alert

the physician of impending complication; can be the clue to the more serious pathology and even help to reach at a diagnosis. Only prerequisite to use them as a scientific tool, is to put them in the correct algorithm of the findings of history and physical examination. In the modern era, there are many advanced structural imaging modalities to help the physician in case of any doubt.

A neurological sign is said to be ‘false localizing sign’ if it reflects the site of dysfunction distant or remote from the actual site of the locus of pathology and hence leads to the breakdown of traditional viewpoint of one-to-one correlation of a sign to neuroanatomy that forms the real ground for localizing neurological lesions on the basis of clinical examination.¹ It is not that a clinical finding is misinterpreted. A bitemporal hemianopia associated with a posterior fossa tumor is a false localizing sign. The site of damage is clearly the optic chiasma on the basis of this clinical finding but the localization of the initiating lesion to the chiasma will prove incorrect. However, false localizing sign should be differentiated from “neighborhood” signs as these signs should be anticipated by the examiner during evaluation. For example, unilateral corneal hypesthesia associated with an ipsilateral homonymous hemianopia may originate from damage to the contralateral temporal isthmus. If the corneal finding is thought to reflect an ipsilateral trigeminal neuropathy, it is an error in evaluation and not a false localizing sign.

History

Every medical student when starts his bedside ward learnings, soon comes to know the legend of the ‘false localizing sign’ and it is the almost always the abducent nerve that tells this legend. The narrator of this legend of neurology is Dr. James Collier.

In 1904, in a landmark paper James Collier² described the cerebellar tonsillar herniation in study based on 161 cases of intracranial tumours which he observed during clinical examination and then during autopsy. He noted false localizing signs in 20 out of them and made first reference to cerebellar herniation as a cause of false localizing

signs. Among false localizing signs observed by him, most common was abducent nerve palsy.³ A year later, Alquier described cerebellar coning in intracranial tumours.² Groeneveld and Schaltenbrand in 1927 and Kernohan and Woltman in 1929 further elaborated the pathological mechanisms behind false localizing signs.⁴ Many more important works were done in following decades, most famous of them was by Gessel et al.⁵ It was only past the middle 20th century² when a formidable knowledge about the clinical signs and physical mechanisms underlying intracranial hypertension and brain swelling and false localizing signs accumulated to make the neurological examination less “embarrassing” for neurologists.

The false localizing signs were such a mystery that pioneer neurosurgeon Harvey Cushing once said: “... neurology was pons asinorum of medical curriculum.”⁶ (pons asinorum - Latin, “bridge of donkeys” in literal meaning, a paraphrase used in geometry.) Many big names of medicine found it difficult to deal with false localizing signs. Outstanding physician-surgeons (as were the things at that time when “specialization” had not “infected” the medicine!) like Jonathan Hutchison and William Macewan missed to correlate their finding to transtentorial herniation.⁷ William Macewan, in 1876, localized a frontal lobe abscess on the basis of clinical examination in young boy and offered to operate but parents of the boy refused. Later, the boy died and on autopsy, Macewan was proved right. But he overdid his observation as he was not able to point to false localizing signs. At the same time, in 1876 only, Brown-Sequard (famous for Brown-Sequard Syndrome) opposed the dogma that lesion on one side of brain will always cause paralysis on the opposite side of body.⁴ It was probably first allusion towards the Kernohan-Woltman phenomenon and towards false localizing signs as a whole.

How significant was the work of Collier and others, cannot be felt rightnow in the era of MRIs. At that time, neurosurgeon was dependent upon the neurologist for the localization of the site of lesion in nervous system. Negative explorations were common, percentage as high as 12-40%,

many were due to error in localization. operations were sometimes performed on, and treatments administered to, the wrong side based on these signs.^{3,5} Oppenheim noted this falseness of localization but offered no explanation for error in localization reached by the clinical signs.⁸ Similarly Nothnagel also issued warning regarding ‘misdiagnosis as result of localization.’⁹ In 1899 Bramwell said, ““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.”¹⁰ Then the legendary Collier came on the stage of neurology and rest is history as we have seen.

Pathogenesis

Pathogenesis depends upon the site and type of pathology. In cranial pathologies, increased intracranial pressure is the usual culprit that causes herniation syndromes. This leads to compression of cortical or subcortical areas, and/or mechanical distortion of cranial nerves leading to the genesis of false neurological signs. The causes of raised intracranial pressure may be intra or extraparenchymal space-occupying lesions like tumors, abscesses, haematomas, hemorrhages and rarely large infarcts, and idiopathic intracranial hypertension (IIH), formerly called as pseudotumour cerebri. The course of the disease can be acute as in cerebral hemorrhages and intracranial hematomas, or can be chronic as in tumors and IIH.¹ Vascular events¹¹ can also produce false localizing signs as discussed later.

Pathogenetic mechanisms of false localizing signs resulting from intracranial pathology are best enumerated by Ehni.¹² According to him, these include general compression of a nerve having a long course, meningitis, edema and gliosis, metastatic infiltration, dorsal column degeneration, infarction at a distance from the primary lesion caused by occlusion of a vessel by neoplasm or by cerebral herniation through a dural aperture, hydrocephalus, transmitted pressure, gross brain displacement with sagittal plane shifting involving the brain stem and causing traction on cranial nerves and kinking of

cranial nerves over vessels, and brain stem shifting to the opposite side, causing tentorial notching, pressure at the rim of the foramen magnum, pressure at points of emergence of cranial nerves, or damage to the corticospinal tract.

Pathogenesis of false localizing signs involving spinal cord is more complicated and contested than those of the intracranial site. Many mechanisms have been contemplated but none of them is undisputed yet. First mechanism suggested is compression of anterolateral spinothalamic tracts.¹³ In these tracts, the peripheral nerve fibres cross 2-3 spinal segments above the level of their entry in the cord to synapse with anterior horn cells and compression at higher level can produce signs suggestive of lower level but it does not explain the discrepancy of 9 segments, even upto 11 spinal segments between the sensory level and the actual level of lesion. Another explanation which involves again the spinothalamic tracts, is based on the lamination of the spinothalamic tract. Fibres originating in lower segments run laterally and posteriorly and those in the upper segments are located medially and anteriorly. But this explanation is again unsatisfactory as it can explain the lateral compression but not central lesions producing false localizing signs.¹⁴ Ischaemia due to occlusion of arterial supply has also been thought of by many but it has not been backed by pathological studies.¹⁵ But others think that ischaemia at watershed zone can explain some of spinal false localizing signs, like positive symptom of “mid-trunk girdle sensation” in cervical compression.¹⁶ But such cases¹⁷ were associated with low lying artery of Adamkiewicz and compensatory ancillary arterial branches¹⁸ were absent. But most commonly accepted view nowadays is the hypothesis of venous obstruction due to compression. It is suggested that venous obstruction from pressure effects of the lesion leads to stasis of blood, resultant hypoxia, and subsequent loss of anterior horn cells.¹⁹ Features of anterior horn cell damage rather than root compression at the level of the clinical signs have been shown by electromyography,²⁰ has been seen pathological examination,²¹ and even induced experimentally at sites distant from the cord

lesion.¹⁹ However, if raised venous pressure is the mechanism the site of the anterior horn cell damage would be expected to be where the arterial pressure—and hence tissue perfusion—in the cord is lowest, but there is no consistent falsely localising level described between cases which would support this hypothesis.¹⁴

Still, there is more to say on this. Mechanical stresses within the spinal cord, consequent perhaps on the conjunction of extrinsic compression with the anchoring of the spinal cord by the dentate ligaments, have also been suggested to account for remote signs.¹ Rise in intracranial pressure may cause raised cerebrospinal fluid pressure in subarchnoid space of spinal cord. This can cause nerve root compression. Thus it is yet another for false localizing signs of spinal cord. Radiculopathies in raised intracranial pressure conditions are best explained by this mechanism.²² Documented enlargement of spinal subarachnoid space and distended root pouches in a patient with radicular pain and areflexia due to IIH supports this view.²³ This is almost always seen with IIH or cerebral sinus thrombosis. Despite all these theories, it will be more wise to say at this stage that different cases may have different pathogenesis, and multiple mechanism may be operating in any one case.¹

False Localizing signs

False localizing signs can be cortical, motor system, sensory system, cranial nerves and spinal cord and roots depending upon the pathology.

Higher Cognitive Malfunction and Cortical Signs

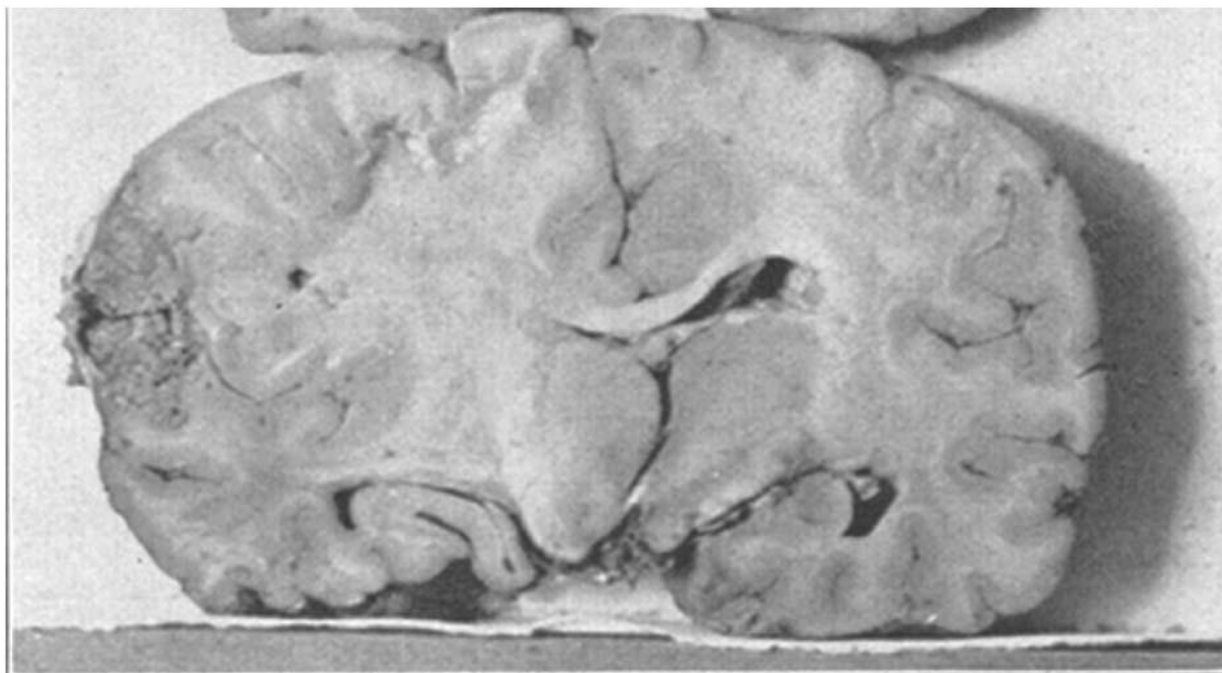
Hemineglect is much commoner with right rather than left parietal lobe lesions. False localizing ipsilateral hemineglect has been reported²⁴ in patients with posterior fossa tumors like meningioma causing left pontine compression, long tract signs and hydrocephalus despite normal structural imaging of the cerebral hemispheres. The neglect resolved promptly after shunting and did not recur despite progressive brainstem compression.

Signs traditionally thought to be of cortical origin, such as aphasia and inattention, may sometimes occur with exclusively subcortical pathology; conversely exclusively cortical lesions may result in dysarthria.¹ Dementia does not qualify as a false localizing sign if there is a frontal lobe tumor, substantially increased ICP, dysphasia, or confusion. But Gassel observed six patients, however, in whom dementia was falsely localizing.⁵ Pseudo(choreo)athetosis and pseudoastereognosis, which may occur with high cervical cord lesions rather than intracranial pathology.¹

Motor System Signs as False Localizing Signs

Kernohn-Woltman Phenomenon

It is also known as Kernohn notch syndrome. Usually a supratentorial lesion, such as acute subdural hematoma, may cause transtentorial herniation of the temporal lobe which leads to compression of the ipsilateral cerebral peduncle against the tentorial edge. Because it is above the pyramidal decussation, a contralateral hemiparesis results. Occasionally, however, the hemiparesis may be ipsilateral to the lesion, and hence false-localizing; this occurs when the contralateral cerebral peduncle is compressed (Pic 1) because of grooving of the crus cerebri on the side opposite to a tumour by the free edge of the tentorium as the brain is pushed by expanding space occupying lesion. This is the Kernohan-Woltman notch phenomenon, or Kernohan's notch syndrome.²⁵ There may be concurrent homolateral third nerve palsy, ipsilateral to the causative lesion.²⁶



Coronal section (V) just behind the mammillary bodies of the brain of the patient case presented by Groeneveld and Schaltenbrand² (adapted from original Fig.²⁷). Note the tumor, an endothelioma (equivalent to meningioma), covering the Sylvian fissure on the left side, the marked edema of the left white matter with displacement of the left hemisphere to the right (subfalcine herniation). The corticospinal tract at the site of the cerebral peduncle is smaller and necrotic at the right side. (For Pic & Caption Source see Ref 4)

Brainstem Compression: False-localizing Diaphragm Paralysis

Hemidiaphragmatic paralysis with ipsilateral brainstem (medullary) compression by an aberrant vertebral artery has been described, in the absence of pathology localized to the C3-C5 segments of the spinal cord where phrenic motor neurones originate, hence it a false-localizing sign.²⁸

Cerebellar Syndrome

Frontocerebellar pathway damage, for example, as a result of infarction in the territory of the anterior cerebral artery, may result in incoordination of the contralateral limbs, mimicking cerebellar dysfunction. Suboccipital

exploration to search for cerebellar tumors based on these clinical findings was known to occur before the advent of brain imaging.¹¹

“Frontal ataxia”, a cerebellar type of ataxia resulting from lesions of the contralateral frontal cortex in which fibres of the corticopontocerebellar pathway are said to be interrupted, is also a false localising sign, but its frequency is uncertain.¹ Round and Keane reported four patients in their series of 101 with IHH to have “ataxia”, meaning a transient postural giddiness with unsteadiness when beginning to walk²⁹; whether this was “frontal ataxia” is not clear. Although functional imaging studies have shown reduced metabolism in the cerebellar hemisphere contralateral to a middle cerebral artery territory infarct (“crossed cerebellar diaschisis”),³⁰ this is not generally accompanied by clinical signs. Cerebellar ataxia contralateral to a posterior fossa mass lesion has been reported.³¹

Pseudo-internuclear Ophthalmoplegia

To describe internuclear ophthalmoplegia, usually indicative of medial longitudinal fasciculus dysfunction, in patients with myasthenia gravis.³² This ‘pseudointernuclear ophthalmoplegia’ has also been observed in dermatomyositis.

Pseudoathetosis

Pseudoathetosis or abnormal writhing movements, usually of the fingers, is caused by a failure of joint position sense (proprioception). They indicate disruption of the proprioceptive pathway, from peripheral nerve to parietal cortex. It may be mistaken for choreoathetosis. However, these abnormal movements are relatively constant irrespective of whether the eyes are open or closed and occur in the absence of proprioceptive loss.³³

False Localizing signs involving Cranial Nerves

Sixth nerve palsy

Sixth nerve palsy, either unilateral or bilateral, is the classic example and most common of the false localizing signs observed by Collier (12 out of 20).³ It occurs in the context of raised intracranial pressure resulting from any cause, may be supratentorial or infratentorial space occupying lesion, idiopathic intracranial hypertension, cerebral venous sinus thrombosis. In a retrospective review of 101 cases of idiopathic intracranial hypertension (IIH), Round and Keane noted 14 cases (11 unilateral, three bilateral).²⁹ IIH with sixth nerve palsy in the absence of papilloedema has also been reported.³⁴ Stretching of the nerve in its long intracranial course or compression against the petrous ligament or ridge of the petrous temporal bone have been suggested as the mechanism for false-localizing sixth nerve palsy.¹ Collier, however, was “unable to accept this explanation”, his view being that since the sixth nerve emerges straight forward from the brain stem, whereas other cranial nerves emerge obliquely or transversely, it is more liable to the mechanical effects of backward brain stem displacement by intracranial space occupying lesions.³

It is also noteworthy that false localizing abducens nerve palsy also occurs in decreased intracranial pressure due to cervical spinal manipulation³⁵, Spontaneous intracranial hypotension syndrome in cervicothoracic cerebrospinal fluid leak³⁶, or following a lumbar puncture.³⁷ Another important thing regarding sixth nerve palsy is that it is not always false localizing, it can be “true localizing sign” also³⁸ when it is affected by lesions in the pons (nucleus and fascicle), subarachnoid space, clivus, cerebellopontine angle, cavernous sinus, superior orbital fissure, and orbit along the course of the nerve (“true-localizing palsy”).³⁹

Oculomotor Nerve

Unilateral fixed dilated pupil may result from an ipsilateral lesion such as an intracerebral hemorrhage, due to transtentorial herniation of the brain compressing the oculomotor nerve against the free edge of the tentorium, k/a Hutchinson’s pupil. Very occasionally, fixed pupil may occur on the opposite side leading to false localization of cranial pathology.⁴⁰ The exact mechanism for this clinical observation is not known. The mechanism for this third nerve palsy has traditionally been ascribed to extrinsic compression of the third nerve on the margin of the tentorium. An alternative explanation, possibly relevant to false localizing third nerve palsy, is that raised ICP causes kinking of the nerve over the clivus, just posterior to the clinoid.²⁹ Another suggestion is that a central mechanism might be responsible, supratentorial pressure causing the brainstem to buckle as it descends because of caudal tethering of the neuraxis at the first dentate ligament (“dynamic axial brainstem distortion”).¹

Trochlear Nerve

False localizing fourth nerve palsies, causing diplopia on downward and inward gaze, have occasionally been described in the context of IIH.^{41,42} Trochlear nerve palsy might be overlooked in cases in which other cranial nerves are affected (sixth, third) because the signs are subtle.

Trigeminal Nerve

Trigeminal nerve involvement in the trigeminal sensory neuropathy or trigeminal neuralgia can be false-localizing, either in association with IIIH⁴³ or with contralateral lesion, usually a tumor.⁴⁴ Reports of both trigeminal neuropathy^{31,45,46} and trigeminal neuralgia^{47,48} are there in literature. For example, trigeminal neuralgia has been described to be associated with a contralateral chronic calcified subdural hematoma, which caused rotational displacement of the pons. The neuralgia resolved after removal of the hematoma.⁴⁹

Trigeminal neuropathy in a patient with IIIH can present as absent blink reflex and absent jaw jerk, either ipsilaterally or contralaterally, in spite of normal motor function.⁴³ Gassel found motor involvement in only two of eight patients with false localizing fifth nerve involvement.⁵ Collier was of opinion that motor involvement preceded sensory features in trigeminal nerve palsy,³ but the later reports suggest sensory findings are more prominent.

The pathophysiology of trigeminal neuralgia associated with contralateral tumors is debated. Vascular compression of the nerve root⁴⁷, angulations and distortion of the nerve root entry/exit zone by displacement of brain tissue caused by an expanding mass lesion in the posterior fossa^{44,48} especially tumors arising from the cerebellopontine angle⁵⁰ are candidate pathogenetic mechanisms. It is also proposed that adherence of arachnoid membrane to the nerve is a contributing factor and its resection in order to straighten the nerve axis is beneficial.⁴⁴ In nutshell, a false localizing sign of trigeminal nerve dysfunction can occur rarely on the contralateral side in patients with large posterior fossa tumors and should be considered in the differential diagnosis of hemifacial sensory disturbance.

Facial Nerve

The intracranial portions of the motor fibres of the seventh nerve run a reasonably short course before they enter the petrous temporal bone of the skull, so it is uncommonly affected by raised intracranial pressure from any cause.⁵¹ False

localizing facial palsy when occurs, mostly takes the form of lower motor neuron type facial weakness, mainly in the context of IIIH.⁵² It occurs usually with concurrent sixth nerve palsy or palsies. It is thought that at the origin in the Pons, fibres of the seventh nerve wrap around the nucleus of the sixth nerve. Any damage at the sixth nerve nucleus, could involve the lower motor fibres of the seventh nerve.⁵¹ There is only single report of facial diplegia.⁵³ and hemifacial spasm is also rare.^{44,54} Trigeminal neuralgia and hemifacial spasm, are only reported in association with posterior fossa tumors.

Vestibulo Cochlear Nerve

Hearing loss has on occasion been reported as a complication of IIIH.⁵⁵

Multiple Lower Cranial Nerve involvement

Concurrent false-localizing involvement of multiple cranial nerves has been noted on occasion, for example, trigeminal, abducens and facial nerves with a contralateral acoustic neuroma,⁴⁶ and trigeminal, glossopharyngeal and vagus nerves with a contralateral laterally-placed posterior fossa meningioma.³¹

Spinal cord and root False localizing Signs

Foramen magnum and upper cervical cord lesions

Lesions at the level of the foramen magnum leads to suboccipital and neck pain and upper motor neuron long tract signs. In addition, these can result in false localising signs including paraesthesia in the hands and lower motor neuron signs in the upper limbs.⁵⁶ The wasting (“remote atrophy”), weakness, and areflexia may suggest the involvement of cervical cord segments well below the level of the foramen magnum lesion, hence are false localising. Similarly, a syndrome of “numb and clumsy hands” has been described with midline cervical disc protrusions at the C3/C4 level; concurrent with numbness of fingertips and palms, there may be a tightening sensation at mid-thoracic level.⁵⁷ Cervical spinal cord lesions (cervical spondylosis, herniated disc)

at or above the level of C4 in which finger and hand dysaesthesia with hand muscle atrophy preceded limb spasticity or gait disturbance have also been reported.⁵⁸ Other falsely localising symptoms or signs that may occur in spinal cord disease include low back and leg pain simulating lumbar disc disease but actually caused by cervical cord compression.^{59,60}

Benign extramedullary tumours at the foramen magnum are notorious for producing signs which do not accurately identify the anatomical site of the tumour, with sensory loss below C5 occurring in 15% of patients and atrophy localised to the hands in 13% of patients.⁶¹

Lower Cervical/Upper Thoracic Cord

Compressive lower cervical or upper thoracic myelopathy may produce spastic paraplegia with a mid-thoracic sensory level (or 'girdle sensation').^{16,62} For example, in one case a spastic paraplegia with a sensory level at T10 was associated with cervical compression from a herniated disc at C5/C6.⁶³

Radiculopathy

False-localizing radiculopathy may occur in the context of IHH and cerebral venous sinus thrombosis. It may manifest as acral paresthesias, backache and radicular pain, and less often with motor deficits.⁶⁴ Rarely it may be sufficiently extensive to mimic Guillain-Barré syndrome (flaccid-areflexic quadriplegia).²² The postulated mechanism for such radiculopathy is mechanical root compression due to elevated cerebrospinal fluid (CSF) pressure.

Other False Localizing Signs

Pseudosyringomyelia has been used to describe a selective loss of pain and temperature sensation with relative preservation of vibration and position sense seen in amyloid polyneuropathy and Tangier disease, a small fibre sensory neuropathy, in the absence of any spinal cord pathology, and hence false localising.¹

Conclusion

Recognition of the false localizing signs remains an important thing still in the present era of neuroimaging especially in spinal cord pathologies. A wrong spinal level of lesion suggested by the false neurological signs on examination can lead to imaging of wrong area which may be normal and delay in diagnosis. Golden rule in such situations is: Normal radiological examination at the clinical site of a cord lesion should prompt investigation at higher levels.¹⁴ Radiculopathy may actually a result of intracranial pathology, but its presence may falsely suggest cord pathology. Repeated search in cord may be negative and diagnosis may be missed altogether or much delayed if one doesn't think on the lines of false localizing signs.

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References

1. Lerner AJ. False localising signs. *J Neurol Neurosurg Psychiatry*. 2003 Apr; 74(4): 415–18.
2. Pearce JMS. James Collier (1870–1935) and uncal herniation. *J Neurol Neurosurg Psychiatry*. 2006 Jul; 77(7): 883–84.
3. Collier J. The false localising signs of intracranial tumour. *Brain*. 1904;27:490–508.
4. Dammers R, Volovicci V, Kampanje EJO. The History of the Kernohan Notch Revisited. *Neurosurgery*. 2016; 78:581–84.
5. Gassel MM. False localizing signs: a review of the concept and analysis of the occurrence in 250 cases of intracranial meningioma. *Arch Neurol*. 1961;4:526-54.
6. Cushing H. The special field of neurological surgery. *Cleveland Med J*. 1905;4: 1-25.
7. Kalyan B. Bhattacharyya. *Eminent Neuroscientists Their Lives and Works*. Academic Publishers. Kolkata. 2011; 265-66.

8. Oppenheim H. Allgemeine Symptomatologie der Hirnkrankheiten. In: Lehrbuch der Nervenkrankheiten für Ärzte und Studierende. 6th ed. Vol 2. Berlin, Germany: Karger; 1913:894-95.
9. Nothnagel H. Einleitung. In: Topische Diagnostik der Gehirnkrankheiten. Berlin, Germany: Verlag von August Hirschwald; 1879:9.
10. Bramwell B. On the localization of intracranial tumours. *Brain*. 1899;22:1.
11. Gado M, Hanaway J, Frank R. Functional anatomy of the cerebral cortex by computed tomography. *J Comput Assist Tomogr*. 1979;3(1):1-19.
12. Ehni, G. 'False' localising signs in intracranial tumor. *Archives of Neurology and Psychiatry*. 1950; 64:692-98.
13. Stookey B. Compression of the spinal cord due to ventral extradural cervical chordomas. *Arch Neurol Psychiatr Chicago*. 1928;20:275-90.
14. Jamieson DRS, Teasdale E, Willison HJ. Lesson of the Week: False localising signs in the spinal cord. *BMJ*. 1996;312:243.
15. Ono K, Ota H, Tada K, Yamamoto T. Cervical myelopathy secondary to multiple spondylitic protrusions: a clinicopathologic study. *Spine*. 1977;2:109-25.
16. Ochiai H, Yamakawa Y, Minato S, Nakahara K, Nakano S, Wakisaka S. Clinical features of the localized girdle sensation of mid-trunk (false-localizing sign) appeared in cervical compressive myelopathy patients. *J Neurol*. 2002; 29: 549-53.
17. Rousseff RT, Tzvetanov P. False localising levels in spinal cord compression. *NeuroRehabilitation*. 2006; 21: 219-22.
18. Schoenen J. Clinical anatomy of the spinal cord. *Neurol Clin North Am*. 1991;9: 503-33.
19. Taylor AR, Byrnes DP. Foramen magnum and high cervical cord compression. *Brain* 1974;97:473-80.
20. Stark RJ, Christopher K, Swash M. Hand wasting in spondylitic high cord compression. *Ann Neurol*. 1981;9:58-62.
21. Mair WGP, Druckman R. The pathology of spinal cord lesions and their relation to the clinical features in protrusion of cervical intervertebral discs. *Brain*. 1953;76:70-91.
22. Obeid T, Awada A, Mousali Y. Extensive radiculopathy: a manifestation of intracranial hypertension. *Eur J Neurol*. 2000;7:549-53.
23. Bortoluzzi M, Di Lauro L, Marini G. Benign intracranial hypertension with spinal and radicular pain. *J Neurosurg*. 1982;57:833-36.
24. Larner AJ. A topographical anatomy of false-localising signs. *Adv Clin Neurosci and Rehabil*. 2005; 5(1): 20-21.
25. Kernohan JW, Woltman HW. Incisura of the crus due to contralateral brain tumor. *Arch Neurol Psychiatry*. 1929;21:274-87.
26. Cohen AR, Wilson J. Magnetic resonance imaging of Kernohan's notch. *Neurosurgery* 1990;27(2):205-7.
27. Groeneveld A, Schaltenbrand G: Ein Fall von du raendothelium über der Grosshirnhemisphäre mit einer bemerkenswerten Komplikation: Läsion des gekreuzten pes pedunculi durch Druck auf den Rand des Tentoriums. *Dtsch Z Nervenheilkd*. 1927; 117: 32-50.
28. Schulz R, Fegbeutel C, Althoff A, Traupe H, Grimminger F, Seeger W. Central sleep apnoea and unilateral diaphragmatic paralysis associated with vertebral artery compression of the medulla oblongata. *J Neurol*. 2003;250:503-5.
29. Round R, Keane JR. The minor symptoms of increased intracranial pressure. 101 cases with benign intracranial hypertension. *Neurology*. 1988;38:1461-4
30. Baron JC, Bousser MG, Comar D, et al. Crossed cerebellar diaschisis in human supratentorial brain infarction. *Trans Am Neurol Assoc*. 1980;105:459-61.
31. Maurice-Williams RS. Multiple crossed false localizing signs in a posterior fossa tumour. *J Neurol Neurosurg Psychiatry*. 1978;38:1232-4.

32. Bakheit AM, Behan PO, Melville ID. Bilateral internuclear ophthalmoplegia as a false localizing sign. *J R Soc Med.* 1991;84(10):627.
33. Joseph Jv. False Localizing Signs in Neurology. *Indian Journal of Clinical Practice.* 2013 feb; 23 (9): 553-59.
34. Krishna R, Kosmorsky GS, Wright KW. Pseudotumor cerebri sine papilledema with unilateral sixth nerve palsy. *J Neuroophthalmol.* 1998;18:53-5.
35. Kurbanyan K, Lessell S. Intracranial hypotension and abducens palsy following upper spinal manipulation. *Br J Ophthalmol.* 2008;92:153-55.
36. Kuhl V, Andreas J, Müller-Forell W, Thömke F, Hopf HC. Spontaneous intracranial hypotension syndrome in cervicothoracic cerebrospinal fluid leak. *Nuklearmedizin.* 2000;39:N22-24.
37. Anwar S, Nalla S, Fernando DJ. Abducens nerve palsy as a complication of lumbar puncture. *Eur J Intern Med.* 2008;19:636-637.
38. Baldawa S. Bilateral Abducent Nerve Palsy: Not Always a False Localising Sign. *J Neurol Disord.* 2015;3:243.
39. David M Katz, Jonathan D Trobe. Isolated sixth nerve palsy. In: Greenamyre JT, editor-in-chief. *MedLink Neurology.* San Diego: MedLink Corporation. Available at www.medlink.com. Last updated: [December 30, 2014].
40. Marshman LA, Polkey CE, Penney CC. Unilateral fixed dilation of the pupil as a false-localizing sign with intracranial hemorrhage: case report and literature review. *Neurosurgery* 2001;49(5):1251-6.
41. Lee AG. Fourth nerve palsy in pseudotumor cerebri. *Strabismus.* 1995;3(2):57-9.
42. Speer C, Pearlman J, Phillips PH, Cooney M, Repka MX. Fourth cranial nerve palsy in pediatric patients with pseudotumor cerebri. *Am J Ophthalmol.* 1999;127(2):236-7.
43. Arsava EM, Uluc K, Nurlu G, Kansu T. Electrophysiological evidence of trigeminal neuropathy in pseudotumor cerebri. *J Neurol.* 2002;249(11):1601-2.
44. Matsuura N, Kondo A. Trigeminal neuralgia and hemifacial spasm as false localizing signs in patients with a contralateral mass of the posterior cranial fossa. Report of three cases. *J Neurosurg.* 1996;84(6):1067-71.
45. Davenport RJ, Will RG, Galloway PJ. Isolated intracranial hypertension presenting with trigeminal neuropathy. *J Neurol Neurosurg Psychiatry.* 1994;57(3):381.
46. Ro LS, Chen ST, Tang LM, Wei KC. Concurrent trigeminal, abducens, and facial nerve palsies presenting as false localizing signs: case report. *Neurosurgery.* 1995;37(2):322-4; discussion 324-5.
47. Michelucci R, Tassinari CA, Plasmati R, Rubboli G, Forti A, Tognetti F, et al. Trigeminal neuralgia associated with contralateral intracranial tumour: a false localising sign caused by vascular compression? Report of two cases. *J Neurol Neurosurg Psychiatry.* 1989;52(10):1202-3.
48. Grigoryan YA, Onopchenko CV. Persistent trigeminal neuralgia after removal of contralateral posterior cranial fossa tumor. Report of two cases. *Surg Neurol.* 1999;52(1):56-61.
49. Kondoh T, Tamaki N, Takeda N, Shirataki K, Mastumoto S. Contralateral trigeminal neuralgia as a false localizing sign in calcified chronic subdural hematoma: a case report. *Surg Neurol.* 1989;32(6):471-5.
50. Koenig M, Kalyan-Raman K, Sureka ON. Contralateral trigeminal nerve dysfunction as a false localizing sign in acoustic neuroma: a clinical and electrophysiological study. *Neurosurgery.* 1984 Mar;14(3):335-7.
51. Kearsy C, Fernando P, Benamer HT, Buch H. Seventh nerve palsy as a false localizing sign in benign intracranial hypertension. *Journal of the Royal Society of Medicine.* 2010;103(10):412-414. doi:10.1258/jrsm.2010.100049.

52. Davie C, Kennedy P, Katifi HA. Seventh nerve palsy as a false localising sign. *J Neurol Neurosurg Psychiatry*. 1992;55(6):510-1.
53. Kiwak KJ, Levine SE. Benign intracranial hypertension and facial diplegia. *Arch Neurol*. 1984;41(7):787-8.
54. Rapana A, Guida F, Conti C, et al. Ependymoma of the fourth ventricle presenting with hemifacial spasm. *Rev Neurol (Paris)*. 1999;155:309–11.
55. Dorman PJ, Campbell MJ, Maw AR. Hearing loss as a false localising sign in raised intracranial pressure. *J Neurol Neurosurg Psychiatry*. 1995;58:516.
56. Symonds CP, Meadows SP. Compression of the spinal cord in the neighbourhood of the foramen magnum. *Brain*. 1937;60:52–84.
57. Nakajima M, Hirayama K. Midcervical central cord syndrome: numb and clumsy hands due to midline cervical disc protrusion at the C3–4 intervertebral level. *J Neurol Neurosurg Psychiatry*. 1995;58:607–13.
58. Sonstein WJ, LaSala PA, Michelsen WJ, et al. False localizing signs in upper cervical spinal cord compression. *Neurosurgery*. 1996;38:445–8.
59. Langfitt TW, Elliott FA. Pain in the back and legs caused by cervical spinal cord compression. *JAMA*. 1967;200:382–5.
60. Chan CK, Lee HY, Choi WC, Cho JY, Lee SH. Cervical cord compression presenting with sciatica-like leg pain. *Eur Spine J*. 2011 Jun; 20: 217-21.
61. Yasuoka S, Okazaki H, Daube JR, MacCarty CS. Foramen magnum tumors: analysis of 57 cases of benign extramedullary tumors. *J Neurosurg*. 1978;49:828–38.
62. Adams KK, Jackson CE, Rauch RA, et al. Cervical myelopathy with false localizing sensory levels. *Arch Neurol*. 1996;53:1155–8.
63. Pego-Reigosa R, Trobajo de las Matas JE, Branäs F, Martínez-Vázquez F, Cortes-Laino JA. Dorsal sensory level as a false localizing sign in cervical myelopathy. *Rev Neurol*. 1998;27(155):86-8.
64. Moosa A, Joy MA, Kumar A. Extensive radiculopathy: another false localizing sign in intracranial hypertension. *J Neurol Neurosurg Psychiatry*. 2004;75(7):1080-1.

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