Journal of Family Medicine and Disease Prevention

RESEARCH ARTICLE

Neurological Localisation in Clinical Practice

Roy G Beran[1,2,3,4,5*] (iD)

[1]*Neurology Department, Liverpool Hospital, Australia*

[2]*Conjoint Professor, South Western Clinical School, University of NSW, Australia*

[3]*Ingham Institute for Medical Research, South Western Sydney Area Health Service, Australia*

[4]*Professor, Griffith University, Southport, Australia*

[5]*Professor, Chair, Sechenov Moscow First State University, Russia*

*Corresponding author: *Roy G Beran, Neurology Department, Liverpool Hospital, South Western Clinical School, University of NSW, Ingham Institute for Medical Research, PO Box 598, Northbridge, NSW 1560, Australia*

Abstract

Neurological localisation is dependent on history and physical examination and demands that the clinician is aware of those features, encompassed within the clinical setting, that define the neuroanatomy and neurophysiology that defines the focus of any neurological lesion. The paper to follow provides an approach to the neurological evaluation of patients that employs the traditional methods of history, stylised physical examination, incorporating: Higher centres; cranial nerves; and peripheral neurological evaluation, and offers important features that define the focus and potential nature of pathology and relies on traditional clinical assessment rather than being dependent on adjunctive investigations. It offers insight into techniques that not only discuss novel approaches, to such areas as higher centre function testing, but also provides explanations as to the interpretation of the findings from an anatomical perspective and offers normative data to help with interpretation of the findings. It explores the anatomy of cranial nerve abnormalities and differentiates some of the findings from peripheral neurological examination to discern upper motor neurone pathology from lower motor pathology and extrapyramidal features from cranial nerve dysfunction. The paper also provides signs of unequivocal features of non-organic presentations to enable the clinician to determine a provisional diagnosis, of non-organic pathology, rather than relying on a diagnosis that is dependent on investigations that showed no abnormality thus leaving non-organic illness as a diagnosis of exclusion, which tends to undermine patient confidence in a situation where the patient is already vulnerable from functional illness.

Keywords

Neurological localisation, Positive signs, Non-organic illness, Diagnostic criteria

Introduction

Neurology is the one area in which there appears a growing chasm between many family physicians and neurologists, possibly because many general practitioners feel that they have inadequate training in the field [1,2]. Nicholls and Appleton stated that, "*many doctors have limited neurological exposure and are hence neurophobic.*"[2]. This seems to be a sad situation and one that should be remedied easily, if neurologists provide appropriate educational material [3].

One of the most important aspect of the neurological evaluation is the localisation of the possible lesion(s) within the nervous system, be it the Central Nervous (CNS) or Peripheral Nervous (PNS) system. These reveal the site of any neurological pathology which presents to the doctor, especially the general practitioner. The paper to follow will take the reader through the process of localisation of a neurological presentation.

Neurological localisation is a little like completing a cryptic cross word or a sudoku. One needs to pay special attention to the clues being offered and then to formulate a strategy with which to address them. It is like playing an intellectual game in which the clinician has been given a special privilege and an advantage, based on a proper understanding of the neuroanatomy and neurophysiology. This paper will not revisit the actual undertaking of a neurological consultation but will provide a road map as to how to evaluate the findings, consequent to that appraisal.

Beran. J Fam Med Dis Prev 2022, 8:144

• Page 1 of 9 •

The most important consideration, when evaluating the neurological patient, is to appreciate that the neurological presentation is no different to any other patient's presentation. The process is the same, namely the taking of a comprehensive history, performing a physical examination that encompasses both a focused neurological evaluation and a more generalised assessment, ordering appropriate tests and providing necessary treatment. What is included in the history, examination and investigations may be somewhat different, within the neurological domain, but the sequence of events remains very similar, irrespective of the system being interrogated. The aim of this paper is to provide a pathway through which to interpret the localising value that the findings may reveal, offering easy to follow suggestions and procedures that should assist the clinician to value-add to his/her existing skill set.

History

The history is the most important component of the neurological consultation and offers the introduction to the localisation of the presenting features. There are very important lessons to be learnt, when taking a neurological history. The first lesson is to ensure that message sent is also message received. There are many words that mean different things to different people and hence constitute 'non-words' [3]. These include such words as: 'dizzy', 'giddy', 'blackout', 'numbness' or 'vertigo'. Many patients interchange 'dizzy' with 'giddy' or with 'vertigo', without truly appreciating what any of them mean. It is imperative to interpret their meaning to reflect that which the patient is attempting to impart, rather than the traditional meaning that is accepted within the medical profession. For this reason, it is vital to insist that the patient defines what (s)he means when using such language. Most often, the patient who complains of 'vertigo' does not have what the clinician means when referring to 'vertigo'.

Vertigo represents the perception of an abnormal sensation of motion [4] and can occur without there being any motion or when a motion is sensed inaccurately [4]. Patients often refer to vertigo as "spinning", because they cannot think of a more appropriate descriptor, but, when cross examined, the feeling that the patient is trying to convey does not include motion but rather the patient is trying to impart a feeling of imbalance or disequilibrium which is something quite different to true vertigo and usually has a different aetiology. Spinning is usually generated from an inner ear origin [4], whereas disequilibrium is a sensation of impending falling, loss of balance or a need to obtain external assistance for proper locomotion and may have a variety of causes. This sensation can originate in the inner ear or may be generated from other motion sensors or elsewhere in the CNS [4]. It is important to discern the nature of what is being felt by the patient and then to seek its cause.

Having confirmed that the patient is truly reporting vertigo it is then imperative to focus attention on the possible causes thereof and how best to deal with these [5,6].

The reverse is equally important, namely when patient use jargon, such as 'convulsion' or 'neuralgia' which are not vague when used in their correct medical meaning. These technical terms may be inappropriately used by patients who are less familiar with their true meaning. It is imperative to always question the use of jargon by patients and ensure that what is interpreted by the physician equates to that which the patient is intending to impart.

Another perfect example in which history offers localising value is when a patient presents complaining of "loss of vision in one eye". Amaurosis fugax is far less common than is homonymous hemianopia. As stated by Pula, et al., *"A proper, well-performed history and physical exam after an episode of Transient Vision Loss (TVL) improves the chance of finding its cause."* [7]. Patients are not trained in neuroanatomy and may be under the misconception that left sided vision comes from the left eye and right sided vision comes from the right eye. It follows that the patient should be asked why (s)he reported that (s)he lost vision in one or other eye, claiming monocular vision loss [3]. The patient who truly has lost vision in one eye will often volunteer that (s)he covered one eye and everything went black but, when covering the other eye, vision was normal [3]. Monocular loss of vision implies a lesion that is distal or rostral to the optic chiasm, as may occur with temporal (giant cell) arteritis, while hemianopia, namely loss of vision in one half of the visual field, results from a lesion more proximal or caudal to the chiasm, as may occur with a stoke [3]. This highlights how important the history can be to the localisation of the neurological lesion.

History remains the single most valuable feature of the neurological assessment, especially within general practice. It is imperative to question the use of any jargon, being offered by a patient, as it may be used inappropriately and lead to misinterpretation by the unsuspecting doctor, resulting in an incorrect diagnosis and possibly incorrect therapy. The examples provided have not been exhaustive but were chosen to make the point that the history needs to accurately reflect the nature of that which the patient is experiencing. This requires practice and questioning that which is being offered, rather than accepting things at 'face value'. Localising the source of the problem, especially within an environment where there is limited access to sophisticated investigation, is heavily reliant on the history obtained [8,9].

In this review of neurological localisation, there is insufficient space to fully review the taking of a neurological history but it should reflect the same

process as the taking of any other history. It starts with clear definition of the presenting complaint, followed by exploring the evolution of the current illness, seeking as much information as is possible, and taking nothing for granted. Should the patient adopt any ambiguous language, it behoves the clinician to question what is being imparted and to be certain that what is being interpreted, by the doctor, matches that which the patient is trying to convey. The remainder of the history follows the standard pattern, including past medical history, social history, family history and a system review.

Nicholls and Appleton [2] encapsulated the importance of a good history by stating, "*Different from all other medical specialties, save perhaps psychiatry, the neurologist is heavily dependent on listening to and interpreting what the patient tells us… If you don't know what is happening by the time you get to the feet you are in real trouble*".

Physical Examination

The neurological physical examination follows a stylised format which is best adopted to obviate missing any vital signs. The format follows three levels of assessment, namely higher centres, cranial nerves and peripheral neurological evaluation. Space precludes an exhaustive discussion of each of these procedures and, what follows, will focus on how they contribute to the localisation of the neurological deficit.

Higher centre examination

The role of higher centre function testing is to discern those patients in whom a firm clinical diagnosis can be made from those for whom a more comprehensive neuro-psychological evaluation is required [10]. It is accepted that a patient presenting with Alzheimer's disease may have focal language problems rather than the more widely accepted autobiographical memory disturbance, despite having identical pathology [10]. As stated by Kipps and Hodges [10], "*The history forms part of the examination, and the ability to respond to conversational cues is as much part of the examination as any formal assessment. In addition, the perspective of a reliable informant is essential, as memory disturbance and impaired insight are common.*" It is important to direct attention to the relevant cognitive domain(s) which require additional attention [10].

Higher centre function testing starts at the beginning of the consultation, as soon as the patient is called to come into the room or when the consultation begins and is an integral part of patient assessment. It includes evaluation of language, ability to recall, in a chronological fashion, and the relevance of responses to the questions being asked [11]. Over time, each clinician will develop his/her own higher centre tools [3] which should provide an overview of the patient's performance. Memory loss and learning impairment which is out of

proportion to other cognitive disturbances is called an amnesic syndrome. Prograde amnesia is a feature of hippocampal damage, as may be encountered in herpes simplex encephalitis, focal temporal lobe tumours or infarction [11]. Confabulation, in which the patient produces memories that do not really exist and which are largely 'made up', suggests Korsakoff's syndrome, which is encountered in alcoholism or with dietary deficiencies. These might be grandiose or delusional stories but are often generated from rearranging and/ or fusing of real memories which end up being retrieved out of context [11]. A transient amnesic syndrome, with pronounced anterograde and variable retrograde amnesia, is seen in transient global amnesia, with focal temporal lobe features on an MRI, while "memory lacunes" and repeated brief episodes of memory loss suggest possible transient epileptic amnesia [11].

Other localising phenomena, within the cognitive domain, include: Problems with working memory in which the patient loses the train of thought of what is happening, as seen in older individuals, but which also occurs with basal ganglia diseases and white matter issues; semantic memory disturbances, such as word finding difficulties, occur with aging but, if they progress to anomia, which occurs in semantic dementia, and are typical of atrophy of the temporal lobe (usually left sided) [11]. Executive function, such as planning, judgement, reasoning, impulse control and problem solving are thought to emanate from the dorsolateral frontal lobe, although they may represent wider implications [11]. Much of the frontal lobe is occupied by white matter and hence a variety of conditions, such as traumatic brain injuries, multiple sclerosis, leukodystrophies and vascular pathology may affect executive function [11].

Visuospatial disorientation suggests non-dominant hemisphere abnormality. It comprises a multi-faceted set of functions, predominantly within the right-hemisphere network, involving widely distributed brain regions including: The parietal lobes; lateral prefrontal cortex; medial temporal lobes; inferior temporal cortex; occipital cortex; basal ganglia; and white matter tracts [12]. Visual neglect, as occurs in non-dominant, usually right hemisphere, stokes, results in the patient ignoring that which is on the left side of his/her environment, (s) he may reject his/her left upper limb as not belonging to him/her or fail to acknowledge that which is occurring on the left side of his/her environment [13].

The Austrian neurologist, Josef Gerstmann reported patients with difficulty discriminating their own fingers (finger agnosia), inability to write (dysgraphia), incapacity to properly distinguish right from left (right/ left dissociation) and to properly perform calculation (dyscalculia), subsequently identified as Gerstmann's syndrome, resulting from a lesion of the dominant parietal lobe [14]. This tetrad has been debated and questioned but Rusconi and colleagues [14] have

confirmed its legitimacy as a Syndrome which involves the tetrad of symptoms and signs but they have proposed that it may relate to a disconnection, due to a lesion to separate but co-localised fibre tracts, in the dominant hemisphere subcortical parietal white matter, based on imaging of functional and structural organisation in the healthy brain [14]. Gerstmann did not include dysphasia, within the original description, but dysphasia, namely difficulty with language, is often associated with the features of Gerstmann's syndrome and represents a dominant hemisphere lesion [3,11].

'Serial 7's' are used to evaluate a range of cognitive functions with few clinicians actually able to describe what they test. Subtraction, namely 100 - 7 = 93 - 7 = 86 - 7 = 79... is a simple task which is used by most doctors who test higher centre function [3]. This reflects dominant hemisphere function (usually left hemisphere), requiring ability to calculate and perform subtraction, but it also requires an active visuospatial capacity which is a non-dominant hemisphere activity, when moving from the 90's, to the 80's to the 70's along the calculation pathway [3]. For a single test to include both the dominant and non-dominant hemispheres, to work together, it requires that the connection, between the two hemispheres, is functionally intact, thus requiring a functional corpus callosum [3]. The test also assesses concentration, memory and attention span. Personal experience suggests that the average person loses concentration not at 65 (as is suggested in the Mini Mental Scale Examination (MMSE)) but at 44, on the subtraction pathway [3]. This indicates the crudity of the MMSE.

Other dyspraxias also evolve from damage to the non-dominant hemisphere and provide some localising value, emanating from the higher centre function testing. Dyspraxia reflects an impairment of, or difficulties with, the organisation, planning and execution of physical movement, with a developmental rather than acquired origin. Most individuals with dyspraxia manifest a combination of both ideational or planning dyspraxia and ideomotor or executive dyspraxia; ideational or planning dyspraxia affects planning and coordination; and ideomotor or executive dyspraxia affects the fluency and speed of motor activities [15]. There has been report of dyspraxia representing a disconnect between the right hemisphere and the left, involving the right superior parietal lobule being disconnected from the left which is dominant for volitional control of movement in most right-handed subjects [16].

The final aspect of the higher centre function testing, to be considered within this review of neurological localisation, is perseveration which may include three different categories, namely: (1) Repetition of a previous response to a subsequent stimulus (recurrent); (2) Inappropriate maintenance of a category of activity (stuck-in-set); and (3) Abnormal prolongation of a current activity (continuous) [17]. Patients with dysphasia were shown to have significantly more recurrent perseveration than did patients with right hemisphere damage or healthy controls [17]. Stuck-in-set perseveration was associated with dopamine system dysfunction (Dopamine producing neurons are located in the midbrain nuclei; mainly Ventral Tegmental Area (VTA) and substantia nigra pars compacta), and continuous perseveration with right hemisphere damage [17]. Sanson and Albert [17] proposed a theory of perseveration dependent on anatomic, neuropsychological, and pharmacologic factors related to cerebral dominance. According to this theory, disruption of specific anatomic and pharmacologic systems produces different forms of perseveration. The finding of perseveration, as part of the cognitive function testing, indicates the presence of organic disease and demands further investigation [3].

Cranial nerve examination

The localisation of lesions, based on cranial nerve examination, is considerably easier than is the case in relation to higher centre function testing. There are twelve cranial nerves, nine of which emanate from the brainstem, with the first and second cranial nerves exiting from the cerebrum and the eleventh cranial nerve which comprises fibres that enter the calvarium, through the foramen magnum, from the cervical nerves, and exits, together with further fibres, derived from brainstem origin, the eleventh cranial nerve, via the jugular foramen. Abnormality in cranial nerve function helps localise lesions to a specific part of the brain or brainstem [11]. Cranial nerves have motor, sensory and autonomic functions [11]. Involvement of a single, isolated cranial nerve usually suggests a lesion of the peripheral nerve component of that cranial nerve while abrainstem lesion usually involves multiple cranial nerves, often involving both motor and sensory tracts to the extremities, acknowledging that the brainstem is a confined organ with the cranial nerves arising from nuclei in close proximity to each other [11].

The first cranial nerve, the olfactory nerve, provides the sense of smell andany dysfunction indicates a need for radiological exploration of the nasal cavity and the anterior base of the skull, in particular the frontal and temporal lobes [18]. In children developmental anomalies or endocrine pathology need consideration [18]. In adults, one needs to consider sinusitis or meningiomas of the olfactory groove [18]. Frontal or temporal impairment: Tumoral or vascular and neurodegenerative disorders (Parkinson's disease) may be accompanied by a loss of olfaction [18], as may simple infections, such as influenza.

There are four components of the second, optic, cranial nerve examination, namely fields, fundi, acuity and pupils [3]. The history will reveal whether it is a monocular or binocular problem, the localisation of

which was discussed above. The pupillary light reflex is tested by shining a light directly into one eye which should result in constriction of the pupils of both eyes due to direct and concentual response to the light stimulation in which the second cranial nerve perceives the light and reacts directly while the contralateral pupil constricts due to effect of the innervation following the light stimulation affecting the oculomotor nerve. Failure of the pupils to constrict could indicate either an optic nerve lesion, a lesion of the efferent limb (oculomotor nerve), or any lesion along the pathway [11]. A space occupying lesion, pressing on the chiasm, may result in constriction of one pupil, when light is shone into it, while the contralateral eye may dilate when stimulated by a light source [19]. This results from atrophy of the optic nerve on one side, obstructing the efferent pathway from that eye, resulting in dilation when that pupillary response is tested, following the testing of the contralateral eye which caused its constriction, due to intact efferent pathway, while the other eye, in which the optic nerve is still functioning, will constrict in response to the light and will lead to both eyes constricting, allowing for a third nerve effect. This is called the Foster Kennedy syndrome, in which an anterior intracranial mass directly compresses the ipsilateral optic nerve, causing the atrophy, while increasing intracranial pressure, which results in contralateral papilledema [18] which is not apparent in the eye with optic atrophy.

Foster Kennedy syndrome is one of the causes of what appears to be unilateral papilloedema, when examining the fundi with an ophthalmoscope, but retrobulbar neuritis is another, reflecting inflammation of the optic nerve, called papillitis [3]. In general terms, papilloedema is bilateral, as it reflects raised intracranial pressure which is not unilateral. Retrobulbar neuritis, also called optic neuritis, as may occur in multiple sclerosis, inflammation, autoimmune diseases and infection, looks like unilateral papilloedema but reflects a lesion of that optic nerve [20]. The difference is that with papilloedema there is little in the way of visual field defect nor pain on eye movement whereas with papillitis there is direct involvement of the optic nerve and hence there is a central scotoma and pain on eye movement and possible obscuration of vision, not usually encountered with papilloedema, other than enlargement of the blind spot which is usually inadequately tested.

Other possible causes of unilateral papilloedema include very uncommon cases of idiopathic intracranial hypertension, although this is usually associated with bilateral papilloedema, and it has been reported with venous sinus thrombosis [21].

Field defects have localising value, such as bitemporal hemianopia, which indicates a lesion affecting the optic chiasm with pressure on the crossing over fibres [22]. Should imaging fail to demonstrate such a lesion, there has been a report of retinal damage, causing this phenomenon, but this is exceedingly rare [22].

Examination of the third (oculomotor), fourth (trochlear) and sixth (abducens) cranial nerves are conducted in conjunction with each other, testing extraocular muscle movement [11]. Abnormal findings present as deconjugate gaze or diplopia. The involvement of the third cranial nerve by compression (aneurysm of the posterior communicating artery) may lead to dilated pupil, ptosis and the eye deviated outward and downward. The fourth nerve innervates the superior oblique muscle and a palsy results in the eye looking down and inwards, often associated with a head tilt. Lateral rectus palsy is due to the involvement of the sixth cranial nerve with failure of abduction; it can be a false localizing sign, due to it having the longest intracranial passage, with increased intracranial pressure, which may cause bilateral lateral rectus palsies. The involvement of the pathways in the brainstem (with such conditions as brainstem lacunar infarct or multiple sclerosis) can lead to Internuclear Ophthalmoplegia (INO). This condition occurs when the medial longitudinal fasciculus, a heavily myelinated pathway that allows for coordinated horizontal gaze, is damaged [23]. There are two levels of INO, namely at the midbrain where there is difficulty with adduction of the affected eye and pontine where there is difficulty with abducting the affected eye.

The Trigeminal nerve serves both sensation and muscles of mastication but sensation is tested more often than is muscle strength. It is less useful as a localising sign and more valuable for testing for non-organic disease when the patient complains of sensory deficits on the face but the distribution thereof fails to respect the anatomical demarcation of the nerve in its three branches [3].

The facial nerve (seventh cranial nerve) supplies the muscles of facial expression, the stapedius muscle in the ear and taste to the anterior two- of the tongue via the chorda tympani branch. The location of weakness in facial muscles can differentiate between peripheral or central involvement [11]. A weakness, with the movement of the entire side of the face, is indicative of either a peripheral lesion involving the nerve or damage to the facial nucleus, in the pons, on the ipsilateral side. Bell's palsy results from an allergic reaction within the facial canal causing swelling and a neuropraxia of the nerve [24]. A weakness of the lower half of the face, with sparing of the forehead, is suggestive of a lesion above and contralateral to the facial nerve (stroke involving the motor cortex). This is because the forehead has innervation from both the left and right sides of the motor cortex [11]. There is an old saying, "Upper is lower and lower is upper", meaning if the forehead is involved it is most likely a Lower Motor Neurone (LMN) lesion and if the forehead is spared it is probably due

to Upper Motor Neurone (UMN) pathology. Damage to the facial nerve can also present with hyperacusis and loss of taste to the anterior 2/3 of the tongue.

A brainstem lesion, affecting the brainstem below the pons and the level of the facial nucleus, may result in pyramidal weakness affecting either side of the body but spare the facial muscles. This will still reflect an UMN lesion with weakness and hyperreflexia but will exclude involvement of the face.

The vestibulocochlear nerve (eighth cranial nerve) deals with hearing and equilibrium control. If air conduction is better than bone conduction, yet there is hearing loss, it represents a neurosensory deafness and, if bone conduction is better than air conduction, it reflects middle ear pathology [3]. When considering the localisation potential of the vestibulocochlear nerve, it is important to consider nystagmus which involves ocular movements with alternating fast and slow components, suggestive of the eye "beating' in the direction of the 'fast' phase. Nystagmus usually reflects an imbalance within the vestibular system, either central or peripheral [11]. Spontaneous, unidirectional, horizontal nystagmus (namely left-beating nystagmus that gets worse in left gaze and never changes to right beating even on right gaze) is highly characteristic of an acute vestibular nerve lesion, such as vestibular neuritis [25]. Benign Paroxysmal Positional Vertigo is diagnosed following a burst of short-lived, upbeat-torsional nystagmus, triggered by a positional test (Dix-Hallpike manoeuvre) [26]. Some nystagmus patterns suggest central lesions, including spontaneous vertical nystagmus, gaze-evoked direction-changing nystagmus (namely, left beating nystagmus on left gaze and right beating nystagmus on right gaze), and positional-triggered down beating nystagmus [27].

The glossopharyngeal nerve (nineth cranial nerve), the vagus nerve (tenth cranial nerve) and the brainstem component of the accessory nerve (eleventh cranial nerve) innervate the pharynx and posterior one third of the tongue [3]. Palatal, phonic speech, sounding as if air is escaping while speaking, is typical of a bulbar palsy, producing a hoarse, nasal quality to speech [3,28]. An UMN deficit will result in pseudobulbar palsy with a spastic, tight, high-pitched speech, together with an exaggerated gag reflex [3]. Pseudobulbar palsy may also be accompanied by emotional incontinence in which there is an over expression of what would otherwise be appropriate emotions, as compared with emotional lability in which there are inappropriate mood fluctuations [3]. The cervical root components of the accessory nerve innervate the sternocleidomastoid and trapezius muscles andthat component is not truly a cranial nerve, while the brainstem component which forms part of the pharyngeal plexus is still a cranial nerve [3].

The hypoglossal nerve (the twelfth cranial nerve)

provides the motor innervation of the tongue [29]. The tongue is the only site in the body where fibrillation can be viewed by the naked eye because the tongue is made up of masses on single muscle fibres and a LMN deficit in the tongue is reflected by fibrillations, often confused with fasciculations which represent spontaneous firing of single motor units, rather than single motor fibres [3]. When asking the patient to protrude the tongue, it will deviate to the side of the lesion [29]. Lesions of the lower cranial nerves, namely nineth through to twelfth cranial nerves suggest a lesion of the medullar oblongata in the brain stem.

It is worth noting that the tongue is the only place in the body where fibrillations can be seen with the naked eye, without use of electromyography (EMG) which is needed to demonstrate fibrillations anywhere else in the body.

Peripheral neurological examination

In the same way that there is a stylised methodology to examine the cranial nerves, so too is there a stylised approach to the peripheral neurological examination, namely: tone; power; reflexes; sensation; co-ordination; and gait [3]. It must be restated that the method of examination is not the focus of this presentation which is particularly focused on the localisation of lesion(s), consequent to the findings identified from the physical examination.

Observation may reveal wasting of muscles which is more prominent in LMN deficits. This is especially so if it is accompanied by fasciculations, namely spontaneous muscle twitching resulting from the spontaneous firing of a single motor unit [3]. Where the LMN deficit is due to a nerve root lesion, the weakness will be restricted to the muscles innervated by those roots [30]. Deep tendon reflexes, innervated by either a specific nerve or affected nerve roots, will be either reduced or absent in LMN deficits [30]. The presence of spasticity or flaccidity can help differentiate an UMN from a LMN cause of weakness, while the presence of lead-pipe or cogwheel rigidity points to a specific disease, like Parkinsonism [11]. The location of the weakness, related to other neurological deficits, can help differentiate a cortical lesion (hemiparesis from a stroke), from a brainstem lesion (crossed deficits from an Multiple Sclerotic plaque), from a spinal cord lesion (presence of dermatomal distribution), from a peripheral nerve lesion (neuropathy or radiculopathy) and a muscular disease (myopathy) or neuromuscular junction (myasthenia gravis) [11].

Weakness associated with an UMN deficitaffects the 'antigravity muscles', namely the abductors and extensors in the upper limbs and theflexors in the lower limbs [3]. While reflexes are depressed with LMN lesions, they are increased and brisk with UMN lesions, together with 'up going' planter responses [3]. Motor

Neurone Disease (MND), also called Amyotrophic Lateral Sclerosis (ALS) or Lou Gehrig's disease, presents with absent reflexes, up going plantar responses, wasting of muscles and fasciculations [31]. There are various forms of MND, accepting that ALS is the most common type, which include primary lateral sclerosis, progressive muscular atrophy and progressive bulbar palsy and each has its own presentation [31].

Testing sensation also has localising value. Cortical lesions present with altered sensation on the contralateral side of the body; spinal cord lesions present with a unilateral sensory level which, on bodily testing, is often one or two segments below the level of the lesion in the cord, respecting the midline; radiculopathies involve the specific dermatome relevant to that nerve root; and peripheral neuropathies can have a glove and stocking distribution [3,11]. Joint Position Sense (JPS) and Vibration Sense (VS) travel in the posterior columns of the spinal cord, on the same side of the body, until reaching the brainstem, where they cross over in the medullar oblongata, while pain, temperature and light touch sensation cross over one or two segments above where they enter the spinal cord. Loss of JPS and VS, on one side of the body, and loss of pain, light touch and temperature, on the other side of the body, indicates a Brown-Sequard syndrome, with a hemi cord lesion on the same side of the body as is the loss of JPS and VS [32]. This type of lesion may also produce features of an UMN lesion, below the level of damage in the cord, together with a segmental LMN deficit at the level of the lesion. This is indicative of a specific hemi-spinal cord damage.

Cerebellar dysfunction presents with incoordination resulting in a wide based gait which should be apparent as the patient enters the room, at the outset of the consultation [3,33]. Other features include horizontal nystagmus, fast beating to the side of the lesion, dysarthria with slurred speech, ataxia of finger-nose testing, heel-shin ataxia and difficulty with repetitive movements with dysdiadochokinesis [3,33]. Unlike cerebral UMN lesions which are apparent on the contralateral side of the body, cerebellar signs appear on the same side of the body as is the lesion within the cerebellum [3,33]. Other features of cerebellar impairment include: Hypotonia; reflex change; fatigue; movement disorder; and disturbances of coordination [33].

The final step in the stylised assessment of the peripheral motor system focuses on the evaluation of gait and gait disturbance. Gait and balance are no longer regarded as purely motor tasks but are considered as complex sensorimotor behaviours that are heavily affected by cognitive and affective aspects [34]. There has evolved a new appreciation of gait and how to manage gait disturbances [35] with the evolution of a new classification of gait and gait disturbances [34]. The proposed classification differentiates between continuous and episodic gait disturbances because it is postulated that this has important ramifications from the functional, prognostic and mechanistic perspectives [34]. Continuous gait disturbances may be the result of chronic neuronal or peripheral dysfunction [34]. When abnormal gait is the result of isolated spinal cord, cortico-spinal tract, cerebellum or extra-pyramidal system dysfunction, it is, with adequate clinical experience, relatively easy to characterize [35].

Within clinical practice, the gait has already been partially evaluated, by the time the patient has entered the examination room, by observing how the patient rose from the seated position, the ease with which (s)he walked into the room, the stability of gait and any problems encountered. Observations will include such gait disturbances as: An antalgic gait due to pain; an ataxic, wide based gait due to cerebellar disequilibrium; a short stepping shuffling gait with stooped posture, devoid of arm swing, as seen in Parkinsonism; a high stepping gait as may be seen with foot drop; a circumduction gait accompanying spastic paraparesis; or a failure of arm swing as may be seen in UMN, pyramidal lesions and extra pyramidal diseases, such as in Parkinsonism, due to increased tone [3].

Non-Organic Illness

As part of the determination of localising symptoms and signs, it is important also to be aware of those features which make the presentation unlikely to represent an organic pathology and thus the localisation is within the psyche [3]. This is somewhat tangential to a treatise on neurological localisation but is very important from the perspective of excluding physical localisation of the underlying pathology. Within this context, just a few important findings will be discussed to introduce the topic, as space precludes a detailed discussion which would represent a paper in its own right.

Consideration of non-organic origin, within any clinical presentation, should start with the taking of the history [3]. A good, well taken history should lead to a provisional diagnosis with alternative differential diagnoses and, if the history fails to lead in any given direction, leaving the clinician sceptical as to the origin of the complaint, non-organic, functional illness must become part of the differential diagnoses. It is imperative, within this context, that the differential diagnosis, of 'functional illness', should not supplant other more serious diagnoses but it should be added to the potential list for consideration. There are some hard-core signs which are pathognomonic of non-organic presentations and these will be touched upon as an introduction to the consideration of localisation [36-38].

Beran. J Fam Med Dis Prev 2022, 8:144

• Page 7 of 9 •

The earlier material demonstrated the localising value of a comprehensive clinical assessment. Where there is incongruity, between the clinical findings and the relevant anatomical parameters, there is high suspicion of non-organicity [3]. Monocular diplopia, in the absence of nystagmus, lenticular dislocation, as occurs in Marfan's syndrome, or significant cataracts, should raise the spectrum of 'functional illness'. This does not negate the need for thorough evaluation but should act as a red flag for the clinician [3]. This is just one example of ocular symptoms which herald a non-organic, functional basis, to a clinical presentation, others include diplopia in which the distant object, when obscuring the vision of one of the eyes, results in the loss of the distant object, irrespective of which eye is obscures, namely the distant object comes from both eyes which defeats the maxim that 'the distant object always comes from the affected eye', thereby excluding it coming from both eyes, during the physical examination [3,39]. Binocular visual acuity and visual field defects are present in up to 80% of non-organic causes [40].

The Trigeminal nerve has a well defined demarcation, namely at the midline of the face, in the binaural plane (between the ears) and along an imaginary line between the tragus of the ear to either the ipsilateral angle of the eye, ipsilateral angle of the lips and just below the midline, below the chin, avoiding the area innervated by high cervical roots, at the angle of the jaw, in the 'beard line', namely that area, at the angle of the jaw, where the bead would grow [3]. If the patient reports a change in sensory perception at the hairline, passing well beyond the midline or following the angle of the jaw, this is non-anatomical and hence a red flag for non-organic presentation [36-38]. If the side of sensory loss changes from one side to the other this is clearly not organic [40].

Sensory testing, anywhere in the body, must respect the anatomical constraints, such as changing in the midline on the torso or respecting the relevant dermatome, and if it fails to do that, be it on the face or the body, it is a red flag for the diagnosis of non-organic illness [36-38]. The observation of a "drift without pronation" sign is specific for Conversion Disorder and can be of help in making a quick distinction between organic and functional paresis at the bedside [41]. When testing maximal power, of any given muscle group, there should not be activation of the antagonistic muscle groups and if this is demonstrated that constitutes evidence of non-organicity [3].

Hoover sign, based on Newton's third law that for every action there is an equal and opposite reaction, provides convincing evidence on non-organic illness [36-38]. If one leg is lifted, by the patient, off the bed, the other leg must push down into the bed. If this does not happen it is apparent that there is incomplete effort in lifting the leg off the bed [42]. A clinical trial of the applicability of Hoover's sign failed to reveal any false positives [42]. There is also an upper limb equivalent of Hoover's sign, such that if one elbow is extended against force, the other, contralateral elbow, will flex and vice versa [36-38]. Should this not occur it is due to the patient not exerting the necessary power and hence confirming 'functional' status [3,36-38].

The above discussion of non-organic illness, also referred to a 'functional illness' or 'supra-tentorial illness' is not designed to be exhaustive but rather to demonstrate that this domain also has localising features which take it beyond the neurological domain and place it into the psychiatry/psychological domain. It should be accepted that this does not translate to the patient voluntarily presenting with these complaints but reflects a different aetiology which is still troubling to the patient and still requires appropriate intervention and treatment, assuming it is not an act of malingering.

It must be emphasised and reiterated that the finding of non-organic features does not, of itself, exclude there being concurrent organic pathology and it is for this reason that it is imperative to look for organic causes for the patient's presentation. As with all medicine, there is the provisional diagnosis and the differential diagnosis. The differential diagnosis takes on additional meaning in the light of non-organic features as it is very easy to ignore other possibilities, once a non-organic diagnosis has been made. This, in no way diminishes the need to explain the non-organic nature to the patient but must encourage the clinician to also have an open mind for possible alternatives, to look for these and to exclude them where appropriate.

Conclusion

Each stage of the neurological examination has the potential to reveal definitive symptoms and signs which can serve to localise the lesion causing the presenting problem. The clinician needs to familiarise him/herself with these features to expedite correct evaluation which leads to the right diagnosis and appropriately directed treatment or intervention.

Conflict of Interest

There is nothing to declare.

Funding

References

1. Morrish P (2009) The changing relationship between neurology and general practice in the UK Br J Gen Pract 59: 599-604.

2. Nicholl DJ, Appleton JP (2015) Clinical neurology: Why this still matters in the 21st century. J Neurol Neurosurg Psychiatry 86: 229-233.

3. Beran RG (2012) Neurology for General Practitioners Churchill Livingstone/ Elsevier, Sydney.

4. Konrad HR (1990) Vertigo and Associated Symptoms (Chpt 123). In: Walker HK, Hall WD, Hurst JW, Clinical Methods: The History, Physical and LaboratoryExaminations. (3rd edn), Butterworths, Boston.

5. Kuo CH, Pang L, Chang R (2008) Vertigo: Part 1– Assessment in general practice. Aust Fam Physician 37: 341-347.

6. Kuo CH, Pang L, Chang R (2008) Vertigo: Part 2 – Management in general practice. Aust Fam Physician 37: 409-413.

7. Pula JH, Kwan K, Yuen CA, Kattah JC (2016) Update on the evaluation of transient vision loss. Clin Ophthalmol 10: 297-303.

8. Paley L, Zornitzki T, Cohen J, Friedman J, Kozak N, et al. (2011) Utility of clinical examination in the diagnosis of emergency department patients admitted to the department of medicine of an academic hospital. Arch Intern Med 171: 1394-1396.

9. Redberg RF (2011) The value of history taking in diagnosis: comment on Utility of clinical examination in the diagnosis of emergency department patients admitted to the department of medicine of an academic hospital. Arch Intern Med 171: 1396.

10. Kipps CM, Hodges JR (2005) Cognitive assessment for clinicians. J Neurol Neurosurg Psychiatry 76: 22-30.

11. Shahrokhi M, Asuncion RMD (2021) Neurologic Exam. Stat Pearls.

12. Possin KL (2010) Visual Spatial Cognition in Neurodegenerative Disease Neurocase 16: 466-487.

13. Parton A, Malhotra P, Husain M (2004) Hemispatial neglect. J Neurol Neurosurg Psychiatry 75: 13-21.

14. Rusconi E, Pinel P, Dehaene S, Kleinschmidt A (2010) The enigma of Gerstmann's syndrome revisited: A telling tale of the vicissitudes of neuropsychology. Brain 133: 320-332.

15. Gibbs J, Appleton J, Appleton R (2007) Dyspraxia or developmental coordination disorder? Unravelling the enigma. Arch Dis Child 92: 534-539.

16. Tanaka Y, Yoshida A, Kawahata N, Hashimoto R, Obayashi T (1996) Diagnostic dyspraxia Clinical characteristics, responsible lesion and possible underlying mechanism. Brain 119: 859-873.

17. Sanson J, Albert ML (1987) Perseveration in behavioral neurology. Neurology 37: 1736-1741.

18. Leboucq N, Menjot de Chamfleur N, Menjot de Chamfleur S, Bonafe A (2013) The olfactory system. Diagn Interv Imaging 94: 985-991.

19. Pastora-Salvador N, Peralta-Calvo J (2011) Foster Kennedy syndrome: papilledema in one eye with optic atrophy in the other eye. CMAJ 183: 2135.

20. Hoorbakht H, Bagherkashi F (2012) Optic Neuritis, its Differential Diagnosis and Management. Open Ophthalmol J 6: 65-72.

21. Kulkarni GB, Singh RJ, Gadad V, Ramakrishnan S, Mustare V (2017) Unilateral Papilledema in Cerebral Venous Sinus Thrombosis. J Neurosci Rural Pract 8: S106-S110.

22. Salgado CM, Dagnelie G, Miller NR (2009) Bitemporal Hemianopia Caused by Retinal Disease. Arch Ophthalmol 127: 1686-1693.

23. Virgo JD, Plant GT (2017) Internuclear ophthalmoplegia. Pract Neurol 17: 149-153.

24. Tiemstra JD, Khatkhate N (2007) Bell's Palsy: Diagnosis and Management. Am Fam Physician 76: 997-1002.

25. Baloh RW (2003) Clinical practice. Vestibular neuritis. N Engl J Med 348: 1027-1032.

26. Furman JM, Cass SP (1999) Benign paroxysmal positional vertigo. N Engl J Med 341: 1590-1596.

27. Kerber KA (2009) Vertigo and dizziness in the emergency department. Emerg Med Clin North Am 27: 39-50.

28. Erman AB, Kejner AE, Hogikyan ND, Feldman EL (2009) Disorders of cranial nerves IX and X. Semin Neurol 29: 85-92.

29. Finsterer J, Grisold W (2015) Disorders of the lower cranial nerves. J Neurosci Rural Pract 6: 377-391.

30. Patten JP (1996) Peripheral Neuropathy and Diseases of the Lower Motor Neuron. In: Neurological Differential Diagnosis. Springer, Berlin, Heidelberg, 333-356.

31. Lau FS, Frank P, Brennan FP, Gardiner MD (2018) Multidisciplinary management of motor neurone disease. AJGP 47: 593-597.

32. Tattersall R, Turner B (2000) Brown-Sequard and his syndrome. The Lancet 356: 61-63.

33. Cole JD, Philip HI, Sedgwick EM (1988) Stability and tremor in the fingers associated with cerebellar hemisphere and cerebellar tract lesions in man. J Neurol Neurosurg Psychiatry 51: 1558-1568.

34. Giladi N, Horak FB, Hausdorff JM (2013) Classification of gait disturbances: Distinguishing between continuous and episodic changes. Mov Disord 28: 1469-1473.

35. Nutt JG, Horak FB, Bloem BR (2011) Milestones in gait, balance, and falling. Mov Disord 26: 1166-1174.

36. Beran RG (2011) "Functional Neurological Symptoms". J Roy Coll Phys Edinb 41: 238-241.

37. Stone J (2011) Functional neurological symptoms. J Roy Coll Physicians Edinb 41: 38-42.

38. Stone J, Davenport RJ (2013) Functional neurological symptoms. Clin Med (Lond) 13: 80-83.

39. Beatty S (1999) Non-organic visual loss. Postgrad Med J 75: 201-207.

40. Enslin JMN, Taylor A (2013) Distinguishing neurological from non-organic conditions. CME 31: 80-84.

41. Daum C, Aybek S (2013) Validity of the "Drift without pronation" sign in conversion disorder. BMC Neurol 13: 31.

42. Popkirov S, Stone J, Buchan AM (2020) Functional neurological disorder a common and treatable stroke mimic. Stroke 51: 1629-1635.

Beran. J Fam Med Dis Prev 2022, 8:144

• Page 9 of 9 •