

Taking a neurological history

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Abstract

Obtaining a detailed neurological history will allow the physician to determine where the lesion is in the nervous system, what the nature of the pathological process is and which physical signs to seek on examination. The ability to take a neurological history depends on basic knowledge of the hierarchical organization of the nervous system and the principles of functional localization. Characterizing the pattern of neurological disease over time is important for management. Episodic, fluctuating and progressive courses of symptoms are the most common. For an episodic condition one needs to determine clearly the events before, during and after an episode. Obtaining a history from an eyewitness may be necessary. Different symptom complexes may point the clinician towards cortical, extrapyramidal, spinal, radicular, peripheral nerve and neuromuscular pathologies. These presentations are discussed.

Keywords assessment; examination; history; neurological symptoms; neurology

What is different about a neurological history?

Whether in the general practice clinic or on a busy hospital take, the neurological history should be a focused, goal-directed exercise which seeks to answer the following questions.

- Where in the nervous system is the lesion?
- What is the pathological process (e.g. inflammatory, vascular, infectious)?
- Is this a purely neurological problem or a neurological manifestation of a systemic disease?

Unlike some specialties, a full neurological examination for every possible sign is rarely practical, and a clear history allows a more appropriate physical examination of the relevant part of the nervous system. There is almost always value in a collateral history, from a relative or care-giver, in order to describe an event the patient cannot recall due to loss of consciousness, or to objectively inform the physician of changes in behaviour or cognitive

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ability. You should remember to seek permission where possible from the patient before obtaining collateral history. Finally, whilst one should always beware of ascribing symptoms to the mind rather than the brain without full investigation, there is overlap of neurology with psychiatry and one must always consider the patient's affect, mood and other psychiatric aspects during the encounter.

Approach to the neurological history

You should adopt a friendly and relaxed interview style which provides sufficient opportunity for the individual to describe in their own words their symptoms. You should balance where possible the use of open and closed questions and not try to put words in to their mouth. You should explore each symptom carefully describing the extent and time course as precisely as possible. You should clarify what the individual actually means by their symptoms. (No two people necessarily mean the same thing when they say they have a numb foot!) You should try to understand how the individuals work, social life and emotions are affected by their problems. If the history is complex (as is often the case) a brief initial overview can be helpful to outline the 'shape' of the history.

Basic information

Age – for example, a particular history in a 70-year-old might raise the suspicion of cerebrovascular pathology whereas the same story from a 17-year-old might suggest migraine or an inflammatory or inherited disease.

Handedness – almost all right-handed individuals and at least three-quarters of left-handed are left-hemisphere dominant for language and this information is important when localizing a lesion. Furthermore, the more left-handed the patient is, the more likely they are to be right-hemisphere dominant.¹

Presenting complaint – what is the patient's most troubling problem (e.g. headache, loss of consciousness)? This is sometimes difficult for the patient to identify and may depend on their insight into their difficulties. It is useful to consider where in the evolution of this problem they are at this point in time. Sometimes formulating a list of problems can be helpful. One can then clarify the importance and sequence of individual problems on the list. If they have a known diagnosis such as multiple sclerosis (MS), what is the symptom that has brought them to hospital on this occasion and can it be explained by their disease process or not?

How do the patient's symptoms and disease change over time?

There are a number of patterns of neurological disease and it is helpful to characterize the patient's symptoms then consider the line of questioning and differential diagnoses for each category.

Discrete episodes

If a patient has episodes such as seizures or blackouts, for example, one needs to start by getting a clear history of the most recent event. Structure the history clearly under the headings 'Before', 'During' and 'After'. Then try drawing a simple chart of the episodes in time.

Describe each episode

- What was happening immediately before the episode? (the circumstances sometimes help to determine the aetiology)
- Were there palpitations, a subjective awareness of their own heartbeat, or did they become pale with clouding of vision or hearing (suggesting vasogenic syncope)?
- What position was the patient in?
- For epilepsy, what factors may have lowered the seizure threshold – was there sleep deprivation or alcohol excess?
- For loss of consciousness, was the patient sitting, standing or lying when they blacked out?
- Does the event always happen upon standing (suggesting a postural hypotension)?
- For vertigo, a recurrence with turning of the head or sitting up from lying flat may indicate benign positional vertigo.
- With thrombolysis increasingly available for acute stroke, time of onset of symptoms needs to be precisely noted.
- What happened during the episode? Clarify the sequence of events as demonstrated in Figure 1. Here an eyewitness is invaluable, particularly if the patient loses consciousness. You should familiarize yourself with a description of a generalized tonic/clonic seizure and contrast this with a more short-lived syncopal event. Jerking movements of the limbs may occur in both syncope and in a generalized seizure.
- Was there movement? Of which limbs?
- Was there loss of continence?
- For non-organic epileptiform attacks, consider if the patient was talking or kept their eyes open, for example. How long did this episode last?
- What was the course of the episode, did it reach a crescendo, suddenly come and go or fluctuate?
- What happened thereafter?
- How long did it take to return to normality?
- For a migraine or seizure, was there focal limb weakness?
- Was the patient generally tired, vague or confused and for how long a time? A prolonged period of confusion or sleepiness is usual after a seizure and a highly useful discriminator.²

A line chart showing a patient's description of the events surrounding their most recent loss of consciousness

Visualizing the pattern of an individual episode helps the clinician distinguish this problem as a probable seizure disorder and sets the scene for a more focused, organized history.

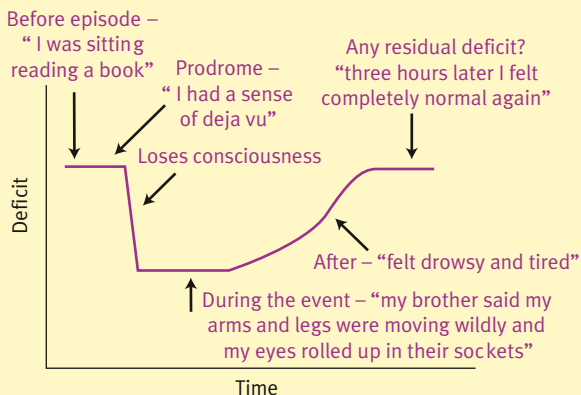


Figure 1

Find out about the pattern of episodes over time

- Have they ever had such an episode before? If so when?
- When did they first have such an episode?
- Since then have they come daily, weekly, monthly?
- Has there been a long interval without problems, for example since adolescence?

What is the severity of each episode? It is helpful to record these in a methodical, objective way as pictured in Figure 2. If the individual has had multiple episodes of altered consciousness, ten or more, one can usually obtain a history of the first episode, the worst episode and the most recent episode.

For headache, one could very quickly distinguish, in this way, between migraine and cluster headache, for example. Likewise, for a patient with chronic epilepsy, one could see that seizures have returned or suddenly started again after a stable period and begin to question what has changed (e.g. a new medication interacting with existing therapy or change of formulation). The relationship to physiological events, such as menstrual cycle, may be important.^{3,4}

Fluctuating levels of severity

Pathological processes such as inflammatory or autoimmune disease (common examples being MS or myasthenia gravis) do not usually present as brief discrete episodes, but more prolonged episodes of symptoms. These may reach a steady level of disease and then fluctuate in severity over time with the patient asymptomatic between each. In such circumstances one needs to be objective about the level of function so as to compare different time-points, especially before and after therapies. Examples include time taken to perform a certain task, how far the patient can walk unaided or how many of the *activities of daily living* such as washing and dressing they can perform alone. These factors are often formalized in scoring systems such as the expanded disability status scale for MS⁵ or the more general Barthel index,⁶ but a good history should encompass such considerations anyway. In particular, consider when a patient has deteriorated what they can or cannot do now which they could previously

Occurrence and severity of episodes for a patient with migraine headaches

When presented in this way it is clear that these episodes were at one stage entirely absent but then returned. Once this had been established the clinician was able to go on and clarify what had specifically changed at that point; in this case eating chocolate – a migraine trigger – whereas the patient was previously on a diet .

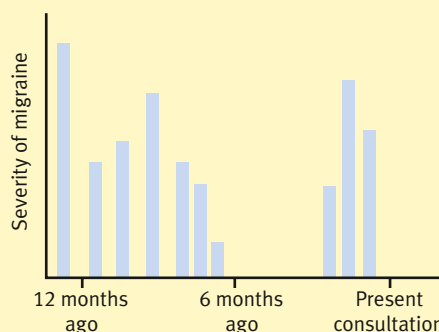


Figure 2

(e.g. get up stairs to bedroom at the end of the day for a myasthenia patient).

Consider when in the day, month, year the symptoms are worst – myasthenic symptoms may worsen over the day as muscles fatigue, and MS symptoms may be more pronounced in summer or after a hot bath due to Uhthoff's phenomenon. Does a change in disease activity correlate with any other change in medications or lifestyle?

For MS, the pattern of symptoms tells you what sort of disease it is⁷ – primary progressive relapsing–remitting (as demonstrated in Figure 3) or secondary progressive. This directly affects choice of therapies that are available.

Progressive disease – rapid and slow

Other neurological diseases are, by nature, progressive, and symptoms never remit nor are they episodic. Common examples include Alzheimer's disease in which the patient gradually deteriorates cognitively, or motor neurone disease (MND) in which the destruction of motor neurones increasingly causes worsening motor symptoms. Some degenerative conditions may be susceptible to treatment which attempts to delay progression (Figure 4), but they rarely reverse the underlying disease process, making decisions about licensing therapies complex and controversial.⁸ A clear history is needed of what the level of function was before assessment and what it is now. One needs to determine what the speed of symptom progression is over time in order to plan management. When did symptoms first appear? How quickly has function been lost? In MND, for example, how many different parts of the motor system are involved? Upper and lower motor neuron? Spinal and brainstem? Likewise for a condition like dementia, what domains of memory are affected – just short-term or longer-term as well?

Patterns you need to be familiar with

Having looked at how symptoms may change over time it is necessary to consider the different patterns associated with each

Line graph of disability against time for relapsing–remitting multiple sclerosis (MS)

Each episode may completely resolve or leave the patient with a baseline level of disability from which their next relapse will occur. Establishing what the baseline level of function was before the current relapse is an important part of the history in such cases.

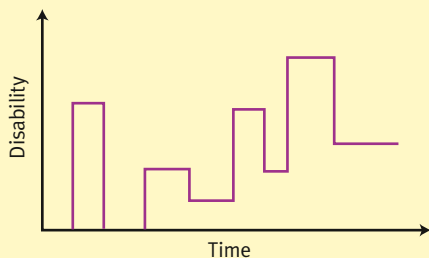


Figure 3

Alzheimer's dementia typifies a progressive neurological disease

There is an unrelenting decrease in cognitive ability over time, measured by mini-mental test scores for example. Treatments, as shown by the arrow, may alter the disease course but will not reverse it.

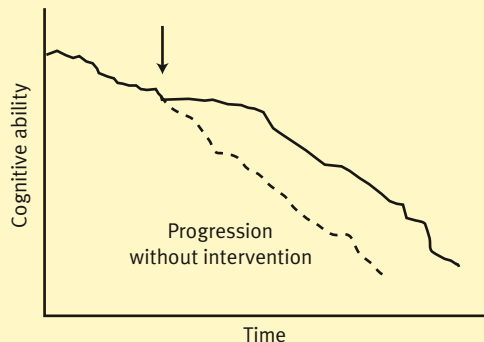


Figure 4

part of the nervous system in order to recognise the common presentations.

Cortical

Syndromes which may suggest a localizing pathology in the cortex include:

- epileptic attacks
- disturbances of consciousness
- cognitive and psychiatric symptoms, e.g. dementia
- organic psychoses
- gaze paralysis
- hemiparesis (unilateral weakness)
- hemisensory disturbance (unilateral sensory disturbances)
- visual field deficits (homonymous hemianopia).

Different areas of the cortex subserve different functions and you should develop a set of questions for localizing deficits to a particular lobe. Pathological processes which commonly affect the cortex include vascular, tumour, abscess and trauma, as well as atrophy/infection for the temporal lobe. Remember that vascular pathologies may not obey anatomical, lobar boundaries but rather have distinct distributions relating to the cerebral arteries. Recall also that the motor and sensory cortices obey somatotopic organization and one may localize motor paralysis, for example, to a particular territory depending on the relative weakness of upper and lower limbs.

Frontal lobes are responsible for planning and executive function; a patient with frontal lobe damage may show an overly familiar or flippant affect. Enquire about odd statements or beliefs. Simple questions such as 'how tall do you think I am?' 'how many patients are there in this ward do you think?' may detect deficits. Is their attention span or recall of phone numbers poor? Here there is overlap with psychiatry as a patient with significant frontal lobe damage may lack capacity and need a formal assessment of this before consenting to treatments and investigations. A wide range of symptoms can suggest frontal

lobe dysfunction including personality, mood, urinary incontinence and insight.

Parietal lesions: cortical sensory deficits can be difficult to identify. Central sensory deficits need to be distinguished from peripheral ones, which are more common. Simple loss of functions, such as temperature or proprioception, suggests a lesion lower down the chain from receptor to cortex. Disorders such as agnosia (can you feel what things you are holding if you close your eyes?) or apraxia (where the individual has an inability to perform gestures and complex motor skills) or inattention suggest a cortical pathology.

Temporal lobe: symptoms that suggest a temporal lobe disorder may, if the left temporal lobe is affected, affect speech and language as well as memory, particularly episodic memory. The hippocampus and the temporal lobe are needed to form new memories and are exquisitely sensitive to anoxia. Patients who have seizures which originate in the temporal lobe origin may experience a strange smell or sense of déjà vu that precedes their seizure. The medial temporal lobe is sensitive to infectious pathology and if a young patient presents with headache and cognitive impairment one must suspect encephalitis. An eye witness account from their family is important in this circumstance.

Occipital lobe: Cortical pathology is an uncommon cause of visual deficits. Because the macula is over-represented in the cortex, in the event of occipital lobe pathology the patient may be left with a black spot in their visual field (a scotoma) where the macula, rich in photoreceptors, is located. Enquire about whether symptoms are bilateral and, in particular, if there is any history of trauma; contre-coup injury with sudden deceleration may damage both occipital poles simultaneously.

Extra-pyramidal

In order to consider whether a patient has extra-pyramidal symptoms, one needs to consider whether the patient fits into a syndrome of too much or too little movement. Syndromes which cause too little movement, such as Parkinson's disease, cause a slowing of movement characterized by stiffness and difficulties with activity, such as turning over in bed or turning round. Extra-pyramidal diseases may also cause involuntary movements including tremor. Hypokinetic-hypertonic syndromes are due to pallidum and substantia nigra lesions such as in parkinsonism. Putamen and caudate lesions, however, lead to a hyperkinetic syndrome in which tone tends to decrease. Within this group, choreoathetosis describes rapid changes in movements often with a writhing or dancing quality. Ballismus, although rare, is a more brief, violent and less smooth involuntary movement. This suggests a localized pathology in the subthalamic nucleus. The differential should include genetic disease and degenerative pathology.

The spinal cord

The spinal cord is symmetrically arranged into ascending and descending columns which are themselves somatotopically organized. With this functional organization in mind we can simply take a history for any cord lesion by asking ourselves what level(s) it involves, whether the whole cross section of the cord

is affected and whether the pathology is *intrinsic* or *extrinsic* to the cord.

For example, when considering level, a high transection of the cord in the cervical spine (due to a traumatic accident) may result in spastic paralysis of all four limbs (tetraplegia) whereas the same pathology lower down, in the thoracic or lumbar spine, would affect just the lower limbs (paraplegia). In trying to establish the level of spinal pathology, sensory symptoms are also useful indicators. Involvement of the perineum with or without associated autonomic dysfunction – such as urinary retention – may indicate that there is a conus or cauda equina pathology. One should try to establish if there is a specific 'sensory level' above which sensation is intact which could then be localized on examination.

Having established the level of the lesion, next ask yourself if the whole cross-section of the cord is affected. Symptoms may be unilateral such as in the Brown-Sequard syndrome and this has a limited number of causes, namely trauma, vascular insult or tumour compression. Likewise symptoms may spare the posterior cord columns (preserved touch, vibration and proprioception) if there is an anterior cord infarction due to spinal artery occlusion. Even if symptoms seem to involve all four quadrants of the cord, the lesion may not be complete – a central pathology such as an expanding syrinx would press on central columns but not peripheral ones causing a cape-like sensory disturbance and upper limb but not lower limb weakness (the so-called 'central cord syndrome').

Finally consider whether the pathological process is from within the spine or from outside it. When considering extrinsic compression it is important to ask about neck or back pain, malignancy which might have metastasized to vertebrae and recent procedures such as epidural anaesthesia or facet injection which could have caused a haematoma or abscess. For intrinsic disease of the cord, the myelinated dorsal and lateral columns are characteristically affected by dietary deficit of vitamin B12 so one should ask if the patient eats well – 'what is a typical meal for you?' – and if there is excess alcohol. Is there a history of syphilis? Untreated this may lead to dorsal column degeneration and a high stepping gait as the patient has no proprioceptive feedback.

Root

A basic understanding of a dermatomal distribution of sensation and muscle innervation is required. Root disturbance usually causes pain which radiates into the muscles that are innervated by that particular nerve root. For instance, in a C5 radiculopathy, the pain would radiate into C5 innervated muscles in the arm, there would be weakness in the deltoid and the other muscles innervated by C5, as well as sensory disturbance in the C5 dermal distributions. The causes include disc herniation, herpes zoster, or tumours of the nerve root. Metastasis can sometimes cause a focal radiculopathy.

Peripheral nerve lesions

Peripheral nerve lesions give rise to weakness, wasting and sensory disturbances. The sensory loss can either give rise to positive symptoms, such as tingling and dysaesthesiae, or negative symptoms, such as numbness or lack of sensation. Symmetrical peripheral polyneuropathy usually affects the feet more than the hands because these nerves are longer. The rate of onset of

symptoms is important to determine aetiology. For example, in Guillain–Barré there is a rapid onset of symptoms over one to four weeks, whereas in diabetic peripheral neuropathy there is a more slowly progressive onset.

Myopathy

Muscle disease is a purely motor condition and it would be important to exclude any sensory symptoms. Motor neurone disease is also a pure motor condition. Proximal myopathy manifests itself as difficulty ascending stairs, rising from a chair or reaching up for things on a high shelf. Myasthenia gravis leads to fatigueability where there is a progressive, increasing weakness during the day. If there is associated pain and soreness of the muscles this may suggest an inflammatory process or inflammatory myelopathy. It is important to enquire whether the patient is taking steroids as this is a common iatrogenic cause of proximal myopathy. Also note if anyone else in the patient's family suffers similar symptoms and if so which gender – muscle disease is often hereditary and the important Duchenne's muscular dystrophy is X-linked.

Further aspects to complete the history

Personal history

A patient's educational background, personal development and IQ may help you to assess whether cognitive symptoms are significant or not. It is also worth enquiring about a patient's birth history and whether they had a normal delivery. Recent foreign travel is simple to ask about, but may completely alter the differential diagnosis. Alcohol is an important neurological toxin and a realistic estimate of use should be sought.

Past medical history

It is important to enquire whether there is a known systemic disease such as sarcoidosis or tuberculosis which could have neurological manifestations. A history of cerebral, neck or back trauma are also relevant. Is there a known malignancy in the past, treated or otherwise, that could now have recurred in the CNS or as a paraneoplastic phenomenon? Does the patient have ischaemic heart disease, risk factors for which would also apply to stroke? Ask when they last had a brain scan, if ever. HIV status should be sought regardless of gender or sexual orientation.

Drug history

A complete list of medications is essential because a number of commonly prescribed medications have neurological side effects. Peripheral neuropathy is a particularly common problem and Table 1 lists some of the possible agents. Ask when each medication was initiated because effects – for example, parkinsonism as a result of atypical antipsychotics – may be related to the extent of use. Substances of abuse are always significant and for intravenous drug users this may be both as a route for infection and as a direct cause of neuropsychiatric symptoms. It should be noted that for epilepsy or suspected epilepsy, drugs may lower the seizure threshold *per se* (e.g. bupropion, aminophylline) or interact with anti-epileptic medications to increase or decrease their metabolism.⁹

Systems enquiry

- Psychological – many patients who get neurological diseases become depressed. It is therefore worth while asking about

Commonly prescribed drugs which may cause peripheral neuropathy as a side-effect

- Amiodarone
- Amytriptyline
- Dapsone
- Disulfiram
- Gold
- Hydralazine
- Isoniazid
- Lithium
- Metronidazole
- Nitrofurantoin
- Phenytoin
- Pyridoxine
- Reverse transcriptase inhibitors (as part of antiretroviral therapy)
- Thalidomide
- Vinblastine
- Vincristine

(More detailed information may be found in the British National Formulary.)

Table 1

depressive symptoms. The patient may not always necessarily volunteer that they are depressed but may feel down, sad, tearful or otherwise low.

- Autonomic nervous system – is there normal bladder, bowel and sexual function? Does the patient complain of light-headedness or falls upon standing? Autonomic disturbance along with parkinsonism, for example, may suggest multiple system atrophy rather than Parkinson's disease as the diagnosis.¹⁰
- Infectious – does the patient have features of sepsis? Low-grade fever? Is there a history of recent infection? Sore throat, myocarditis or diarrhoeal illness might point to a post-infectious phenomenon such as Sydenham's chorea or Guillain–Barré.
- Rash or joint problems are suggestive of a vasculitis.
- Cardiac symptoms are important when considering, for example, loss of consciousness.
- Sleep disturbance is suggestive of a brainstem lesion.
- Weight loss or poor appetite should be enquired after if malignancy is suspected.
- A useful closing question is to ask whether the individual feels there is any symptoms they have omitted to ask about.

Family history

A proportion of neurological disorders are inherited and a clear family tree should be constructed, paying attention to likely patterns of transmission (e.g. X-linked vs. autosomal dominant). One should enquire about age of onset as trinucleotide repeat diseases, such as Huntington's, may show anticipation.

Social history

It is important to try to assess the individual's home, work and other activities to understand how their neurological symptoms affect their ability to care for themselves in terms of walking, shopping, dressing and bathing. An appreciation of their typical

daily routine and expectation of outcomes is helpful. This information helps determine decisions about management, as treatment choice for many common chronic diseases depends on quality of life and its alteration by the disease process. ♦

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