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Acute neurology: a suggested approach

Authors: Kuven K Moodley,[A] Arani Nitkunan[B] and Anthony C Pereira[C]

ABSTRACT

Acute neurological problems are common, accounting for 10–20% of medical admissions. In the coming years, there will be increased neurology involvement in the acute care of these patients complementing traditional outpatient-based services. Models of acute neurology are reliant on close collaboration between the emergency department, acute medicine and neurology and should integrate with existing hyperacute stroke pathways. In this article the authors briefly describe the two models of acute neurology set up recently in our neuroscience group and suggest a clinical approach that may help non-neurologists involved in acute care settings. The authors emphasise some of the lessons learnt in delivering the service, particularly the importance of focusing on the acute problem and tailoring the examination and investigations to tackling it in the context of the patient's functional level and personal circumstances. Early neurology intervention can reduce admission and hospital length of stay.

Introduction

Acute neurological problems account for 10–20% of acute medical admissions.[1–3] While neurology has traditionally been an outpatient specialty (owing to the historic shortage of UK neurologists), only a few neurology units in the UK currently deliver an acute service.[4] However, it is clear that patients benefit from early neurology expertise, ideally working closely with emergency departments and acute medicine units to deliver specialist care at the front door.[5] The Association of British Neurologists (ABN) has produced quality standards for acute neurology[1,4] while commissioning guidance (for London) has focused on the long-term sustainability of service models in different settings.[6,7]

At teaching hospital level, St George's Hospital has an established Hyperacute Stroke Unit (since 2010) and was the first UK unit to provide a full 24/7 thrombectomy service. To complement these services, the Hyperacute Neurology Service (HANS) was established. This is a consultant-led (2.2 FTE) service that currently operates 9am–5pm, Monday to Friday. We recognise that neurology needs to evolve to provide full 24/7 care. However, to ensure sustainability, service growth has been staged with the intention being to incorporate weekends and then evenings and nights. The service covers all emergency neurology including stroke and is proactive in managing stroke reperfusion or mimics, avoiding unnecessary admissions and streamlining care of acute neurology cases. Patients can also be seen rapidly on the day unit or in 'hot' clinics. The ethos is for each patient to receive an appropriate specialist management plan and avoid over-investigation and admission if possible.[8] The service was recently formally commissioned. It managed 1,299 patients in its 1st year (603 stroke, 452 acute neurology and 244 'hot' clinic patients). Admission was avoided in 25% of cases. The median length of stay reduced from 2 days to 1 day (p=0.001) and the median thrombolysis door-to-needle time from 44 to 29 min (p=0.001).

At district general hospital level, Croydon University Hospital NHS Trust[7] Hyperacute Neurology Team comprises four consultant neurologists (3.2 WTE) available 5 days a week. The lynch pin of the team is an acute neurology nurse coordinator who sees all referrals including collating investigation results to present to the consultant. Two epilepsy nurses complement this service. The proportion of patients seen on the day of admission rose from 59% pre-onset of this service to currently 92%. For patients with epilepsy, length of stay reduced by 32% and admission rates by 12%.

Key Points

Establishing the time course of the presenting neurological symptom is key in diagnosis

Practice a neurological screening examination. When proficient, this should take less than 5 minutes. Extend the assessment appropriately if the history dictates so

Have a pre-test hypothesis to rationalise appropriate investigation/s

The management plan should include practical help such as pain relief, involvement of the multidisciplinary team eg physiotherapists

Trusts will need to establish acute neurology services that suit their particular circumstances, ensuring that these complement existing acute services in improving patient outcomes

KEYWORDS: hyperacute, neurology, hot clinics, localisation ■

Authors: [A]consultant neurologist, St George's University Hospitals NHS Foundation Trust, London, UK; [B]consultant neurologist, St George's University Hospitals NHS Foundation Trust, London, UK and Croydon University Hospital, Croydon, UK; [C]consultant neurologist, St George's University Hospitals NHS Foundation Trust, London, UK

Box 1. The principles of acute neurology

1 Use the history (direct and collateral) to establish a differential or working diagnosis. Be an active listener. Let the patient talk; they will often 'tell' you the diagnosis

2 Develop a routine to rapidly screen for neurological deficits.[9] Where is the problem: brain, spinal cord, peripheral nerve etc? This should only take 4 to 5 minutes

3 Target investigations to define aetiology. Decide on the general underlying pathological process rather than a diagnostic label (eg inflammatory problem affecting the cervical cord to guide investigation[10]

4 Ensure the management plan is helpful to the patient (eg pain relief)

5 Involve the multidisciplinary team

6 Decide whether patient needs admission

The main lessons learned from both models are that:

- sustainable, acute neurology services can be set up in different hospital environments, each very responsive to patients' needs and dramatically reduce admissions and hospital stay
- early, senior review improves patient confidence in the diagnostic plan and can facilitate a planned outpatient management.

The commonest conditions seen are stroke / transient ischemic attack (TIA), headache, seizures, functional neurological disorders and medical decompensation of known neurological injury. Here we concentrate on the principles involved in acute neurology (Box 1).

Acute medicine is under tremendous time pressure making it necessary to conduct an efficient neurological assessment.[11] Cutting corners is usually a false economy. However, in acute neurology, many problems are relatively simple to diagnose and solve. The key is to focus on the presenting problem.

History

The nervous system will often yield symptoms in parallel with pathophysiological development. Consider the time course of symptoms carefully (Table 1).

Most neurological diagnoses rest on the history. We find that focusing on the presenting problem until one understands it properly will usually yield the solution and management plan before the remaining traditional elements of history taking are undertaken. The past medical history may provide obvious clues (eg diabetes, known neurological diagnoses). Medication errors (eg forgetting antiepileptics or mistakenly taking the wrong dose) in our experience are common and may trigger an event. Family history, even for common conditions (eg migraine), is informative. Always ask about smoking, drinking and drugs. Constitutional symptoms may indicate a medical condition (eg renal failure) affecting the nervous system. Lastly, the social circumstances are essential not only in planning ambulatory care[8] or admission, but also determining any stressors that may be contributing to the presentation.

In many instances (eg blackouts), a reliable collateral account of events will make the diagnosis and establish the baseline premorbid neurological function. Sometimes, a written plan may be available via the hospital electronic system to guide management. Epilepsy specialists often provide this for frequent attenders. Remember to give safety advice and discuss occupational or driving implications relevant at the time.[12]

Table 1. Diagnostic modelling in neurology based on the temporal course of symptom onset

Time course	Onset time	Probable aetiology
Hyperacute	Seconds	> Vascular eg stroke > Epileptic seizure
Acute	Minutes	> Vascular > Toxic-metabolic > Compressive lesions > Vasovagal syncope
Subacute	Over 72 hours	> Toxic-metabolic eg B12 deficiency > Inflammation (Infective or immune-mediated) > Closed space pathology eg subdural haemorrhage > Infiltrative or neoplastic process > Myasthenia gravis > Medical decompensation of previous neurological insult (can occur with or without delirium)
Remitting – Relapsing	Back to baseline within a day	> transient ischemic attack (usually recovered in <1 hour) > Periodic disorders (migraine with aura, epilepsy, ocular myasthenia)
Remitting – Relapsing	Returning to baseline but taking days–weeks	> Multiple sclerosis > Toxic-metabolic eg drug toxicity
Stuttering	Usually acute or subacute in onset but patient developing new deficits rather than progression of existing ones	> Stroke-like syndromes due to vasculitis/endocarditis > Autoimmune encephalitis > Myasthenia and atypical forms of Guillain-Barré syndrome
Chronic, progressive	Weeks	> Guillain-Barré syndrome > Malignancy
Chronic, progressive	Months	> Degenerative diseases eg Alzheimer's disease, motor neurone disease > Malignancy

Common diseases may present in an atypical manner

Examination

Acutely, the problem may still be present and obvious. Otherwise, localise the lesion. Think of the body as a grid: left or right, with levels from higher cognitive function, cranial nerve levels, spinal cord, plexus, peripheral nerve to muscle (Box 2). Distinguish upper (brain and spinal cord) from lower

Box 2. A suggested approach for a screening neurological examination

- General features: make a note of dress, foetor, level of arousal (Glasgow Coma Scale), insight into presenting problem, ease of communication, recall of personal information, speech (slurred speech or dysarthria / expressive or receptive dysphasia / hypophonia) – if affected, perform a bedside swallow test to assess aspiration risk
- Cranial nerves: gross acuity (able to read your ID badge), fundi, fields, eye movements, pupil size and light responses, facial strength
- Tone: normal, flaccid, spastic or rigid
- Power:

  Pronator drift

  Characterise the pattern of weakness: pyramidal (brain/spinal cord), proximal>distal (muscle/radiculopathy), distal>proximal (neuropathy, rarer myopathies). Is it lateralising (ie hemiplegia) or localising (paraplegia as seen in cord lesions)
- Neck flexion: this is of particular importance in cases of suspected acute neuromuscular weakness (in extreme cases, patients may present with 'head-drop') and should be regarded as a harbinger of diaphragmatic failure
- Reflexes: absent, normal or brisk. Remember reflexes are frequently reduced in diabetic patients
- Sensory: distal light touch sensation, double simultaneous stimulation (the presence of sensory hemi-neglect indicating higher cortical sensory impairment, as may commonly occur with right parietal strokes). Characterise the pattern of light touch sensory loss where possible but bear in mind that the sinister causes of sensory impairment are usually associated with other signs. Organic patterns of isolated light touch sensory impairment include: unilateral (either mononeuropathy or monoradiculopathy), 'glove and stocking' (toxic-metabolic axonal polyneuropathies) and dense hemisensory (rare instances of lacunar stroke, more likely to indicate a functional neurological disorder). The initial stages of GBS and inflammatory transverse myelopathy may be predominantly sensory; the presence of a truncal level usually indicates cord pathology)
- Coordination: finger–nose, heel–shin
- Gait including walking on toes and tandem gait
- Blood pressure and pulse (including postural responses)

  Postural blood pressure measurements should be obtained after lying down for *at least* 5 minutes of rest and repeated after standing for 3 minutes. Postural hypotension is defined as a reduction of greater than 20 mmHg systolic or 10 mmHg diastolic. Any symptoms that develop during the standing phase of the assessment should be clearly documented

(cranial or peripheral nerve) motor neurone problems. Use the medical examination to complement the neurology (eg cardiorespiratory function, including spirometry, in Guillain-Barré Syndrome[9]).

In patients with suspected delirium, use an appropriate screening tool such as the 4AT[10] or the Confusion Assessment Method (CAM)[13]. In patients without delirium, consider a validated bedside screening tool of cognitive function such as the Montreal Cognitive Assessment.[14] *In either case, it is mandatory to establish baseline ability through collateral history taking.*

Establish a working diagnosis after history taking and examination so as to minimise unnecessary investigations, reassessing if necessary.[15,16]

Investigation

Investigations are less likely to give you the answer unless you ask the right question. Key neurological investigations include the following.

- Blood tests. These are particularly important where there is decompensation of pre-existing neurological conditions, as may occur with intercurrent infection or metabolic disturbances. These usually include full blood count (FBC), urea and electrolytes (U&E), liver function tests (LFT), bone profile, glucose, and C-reactive protein (CRP). Other tests should be problem-orientated, such as erythrocyte sedimentation rate (ESR – patients over 50 years with headache for possible temporal arteritis) or thyroid function tests (TFTs – for tremor).
- Imaging. Determine whether computed tomography (CT) or magnetic resonance imaging (MRI) is more appropriate. Identify where the problem lies (eg brain or spinal cord) and image accordingly. Scanning the entire neuroaxis is rarely acutely necessary and is time consuming and inefficient.[16]
- Neurophysiology. Electroencephalogram (EEG) is *not* a test of epilepsy. 10% can be 'positive' in the normal population and a 'negative' EEG does not have sufficient negative predictive value to 'rule out' epilepsy.[17] It is particularly helpful in the unconscious patient or those with suspected encephalitis or non-convulsive status.
- Lumbar puncture (LP). Always examine for papilloedema and review any imaging available before LP. Note that imaging is not a prerequisite for LP in immunocompetent adults, even if unable to view the fundus, unless there are focal neurological signs, continuous/uncontrolled seizures, or Glasgow Coma Scale (GCS) score <13.[18] *Take enough CSF* (about 5 mL, more if cytology is needed). Always document the opening pressure, bearing in mind that these are only diagnostically useful when LP is performed with the patient in the lateral position. Send for protein, absolute cell count, viral polymerase chain reaction (PCR), Gram stain, culture, glucose and, when appropriate, oligoclonal bands (OCBs). Paired blood samples should accompany requests for cerebrospinal fluid (CSF) glucose and OCBs. In a patient with suspected infectious meningoencephalitis and fever, remember additional tests such as blood cultures, throat swabs and urinalysis, particularly if LP is contraindicated or likely to be delayed.[18]

Patients with 'coma'

For medical patients with an unspecified (non-trauma related) cause of coma, the clinical assessment should not simply centre on the GCS. First check the blood glucose. Then the FOUR score[19] is particularly useful for identifying 'where' the problem may be. In cases where the CT is normal, a careful assessment of the pupils is mandatory particularly before opiates have been administered as part of any anaesthetic intervention. Small, symmetrical and reactive pupils may signify bilateral thalamic injury (toxic or stroke-related). Whereas bilaterally mid-position 'fixed' pupils may be seen in pontine injury (pupils small) or midbrain injuries (pupils

mid-size or dilated). In the absence of hypoglycaemia, meningism or other focal signs (eg paralysis unilateral bilateral or crossed, unilateral eye deviation or periodic respiration *inter alia)*, consider systemic metabolic causes for coma, including a toxin screen. Wijdicks addresses this area in greater detail.[20]

Management plan

You must put the patient in the centre and tackle the reason for their presenting. They may have a new neurological diagnosis which requires investigation and treatment but one cannot overstate the importance of simple practical advice such as pain relief or involving other professionals (eg physiotherapists, speech and language therapists or neurology nurse specialists). Rapid access clinics are the preferred route to assess ambulatory patients, including those with long-term neurological conditions (eg multiple sclerosis) experiencing sub-acute decline. For patients with long-term conditions, these should tackle common problems such as recurrent urinary tract infections, aspiration pneumonia and falls before they escalate and need admission. Multidisciplinary team involvement will be essential in some instances.

Conclusion

Acute neurology is not about knowing where the nucleus ambiguus is nor about treating very complex cases. Like other acute specialties, it is about thinking clearly and logically about an acute problem and focusing on it without losing sight of the patient's wider medical and social context and delivering appropriate advice and care when patients need it most. Like most skills, practising a focused screening examination is key to becoming competent and confident in the detection of clinical signs. When considered in the context of the history, this is critical for modelling diagnoses and management decisions.[15] Individual trusts need to consider different models dependent on the services already available.[6] ■

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Address for correspondence: Dr K K Moodley, Department of Neurology, Atkinson Morley Wing, St George's Hospital, Tooting, London SW17 0QT, UK.
Email: Kuven.moodley@nhs.net