

# 15 ASSESSMENT AND MANAGEMENT OF NEUROLOGICAL PROBLEMS (2)

C M Gavin, J T Gray

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**P**atients with neurological conditions present with a wide variety of symptoms and signs. These may sometimes be non-specific or vague and often present a diagnostic challenge to the practitioner. Therefore it is important that all patients are assessed using a robust system that will identify those who require urgent treatment or hospital referral. Box 1 describes the objectives of this article.

## Box 1 Article objectives

This article will consider patients presenting with:

- ▶ Collapse
- ▶ Dizziness
- ▶ Visual disturbance
- ▶ Focal neurological deficit
- ▶ Generalised weakness

## THE PRIMARY SURVEY POSITIVE PATIENT

The patient should be assessed according to ABC principles (box 2).

## Box 2 Primary survey

**If any of the observations below are present treat immediately and transfer to hospital**

- ▶ Airway obstruction
- ▶ Respiratory rate <10 or >29 per minute
- ▶ Oxygen saturation <93%
- ▶ Pulse <50 or >120 per minute
- ▶ Systolic blood pressure <90 mm Hg
- ▶ Glasgow Coma Scale score <12

The main categories of primary survey positive patients are covered in neurological problems (1),<sup>1</sup> however, occasionally patients will be found to have an ABC problem not related to unconsciousness or fitting that will require immediate transfer to hospital.

## Tip

- ▶ **A** airway obstruction/compromise—loss of protective airway reflexes or aspiration secondary to brainstem damage or dysfunction
- ▶ **B** breathing inadequacy due to respiratory muscle involvement—for example, Guillain-Barré syndrome, myasthenia gravis, motor neurone disease
- ▶ **C** circulatory compromise—hypotension due to arrhythmias may produce symptoms such as dizziness or collapse

See end of article for authors' affiliations

Correspondence to:  
Dr C Gavin, Consultant in  
Emergency Medicine, Hope  
Hospital, Salford, M6 8HD;  
carole.gavin@srht.nhs.uk

The patient's primary symptom should suggest the differential diagnosis. It is important that a thorough history is taken to elicit this further. A focused neurological examination will then enable the practitioner to decide if the patient can be managed at home or if referral is needed. An important clue to the possible aetiology is the speed of onset of symptoms (table 1).

**Table 1** Speed of onset as a clue to possible aetiology

Speed of onset	Possible aetiology
Sudden	Stroke, hypoglycaemia
Hours to days	Infection, Guillain-Barré syndrome, botulism
Days to weeks	Infection, inflammation—for example, temporal arteritis, polymyalgia
Weeks to months	Cerebral tumours—primary or secondary
>Months	Degenerative conditions, motor neurone disease

## THE PATIENT PRESENTING WITH COLLAPSE/ SYNCOPE

The management of the patient who is unconscious has been discussed in a previous article.<sup>1</sup> They should be transported to hospital immediately by ambulance following treatment of any ABC problem and after excluding hypoglycaemia. A more common problem is the patient who has had an episode of collapse with or without a period of unconsciousness, but who is fully alert when help arrives.

Syncope is the abrupt and transient loss of consciousness associated with absence of postural tone, followed by a rapid and usually complete recovery. This symptom is alarming for the individual, witnesses, family, and clinicians. Although syncope can be a harbinger of a multitude of disease processes and can mimic the appearance of a cardiac arrest, it is most often benign and self-limiting. There are many causes of syncope and collapse. However, despite the multiple aetiologies the cause may remain unknown. One prospective study was unable to establish the cause in 18% of patients with syncope.<sup>3</sup> The practitioner will need to rely on a careful

history and examination to establish the most likely cause for each individual patient.

### Tip

► A mnemonic that can help remember all the possible differential diagnoses is: HEAD, HEART, VESSELS, DRUGS (see table 2)<sup>2</sup>

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### Subjective assessment—history

Some types of syncope are more sinister. Sudden and complete loss of consciousness, often causing the patient to fall and injure themselves, may indicate a potentially serious cause, as does syncope that occurs with exertion. It is helpful to get a witness account that may verify the loss of consciousness, any associated limb movements, and the presence or absence of pallor or sweating. Information regarding the presence or absence of a pulse and the pulse rate during the episode is also of diagnostic significance.

### Tip

Establish the number of episodes that have occurred. In general, benign causes of syncope are usually associated with a single syncopal episode or with multiple episodes over many years. The patient with multiple episodes occurring over a short period of time is more likely to have a serious underlying disorder.

**Table 2** Causes of collapse<sup>2</sup>

Differential diagnosis	Clinical clues
Hypoxia, hypoglycaemia	Should be picked up in primary survey Do not forget the glucose
Epilepsy* Affective (psychological) Dysfunction of brain stem—for example, vertebrobasilar transient ischaemic attack, basilar migraine	Previous history, postictal period History of anxiety or panic disorder, hyperventilation Cerebellar signs on neurological examination
Heart—for example, ischaemic heart disease Emboli—pulmonary embolism Aortic obstruction—for example, stenosis, hypertrophic obstructive cardiomyopathy (HOCM)† Rhythm disorders—for example, sick sinus syndrome, complete heart block Tachydysrhythmias—for example, SVT, VT, long QT syndrome	Recent chest pain, history of myocardial infarction Pleuritic chest pain, dyspnoea, calf pain, or swelling Precipitated by exertion, cardiac murmur on auscultation May be picked up on primary survey if heart rate <50, history of ischaemic heart disease History of palpitations, may be picked up on primary survey if heart rate >100, <5 s prodromal period
Vasovagal* ENT—for example, Ménière's disease, acute labyrinthitis, benign paroxysmal positional vertigo Situational—for example, fright, micturition, deglutition, defaecation Sensitive carotid sinus Ectopic pregnancy**	Prodrome of nausea, dizziness, yawning, sweaty History of vertigo, deafness, tinnitus, nystagmus on neurological examination May be apparent from history
Low vascular tone Subclavian steal**	Precipitated by head movement History of abdominal pain, amenorrhoea, PV bleeding, positive pregnancy test
DRUGS—for example, antihypertensives, sympathetic blockers causing postural hypotension*	Precipitated by upper arm exertion
	Elderly patient on multiple drugs Postural fall in blood pressure

\*Common causes

†Rare causes

**Table 3** Conditions causing dizziness

Type	Definition	Causes
Vertigo	Sensation that the individual or environment is rotating	Benign paroxysmal positional vertigo Acute labyrinthitis Vestibular neuronitis Ménière's disease Brainstem or cerebellar disorder Drug toxicity
Presyncope	Sensation that one is about to black out	Postural hypotension Dysrhythmia Cardiac outflow obstruction Other causes of syncope (see table 2)
Hypoglycaemia		Diabetes mellitus Alcohol
Dysequilibrium	Imbalance when standing or walking without the sensation of movement or impending loss of consciousness	Insulin secreting tumours Non-specific symptom—may be from many different causes. Usually in proprioceptive or vestibular systems
Drug effects		Salicylates, tranquillisers, aminoglycosides, anticonvulsants, antihypertensives, antimalarials, alcohol
Psycho-physiological		Panic attacks Anxiety Phobias

In some cases it may be difficult to distinguish between seizures and syncope. Seizures are the probable cause of 5–15% of apparent syncopal episodes.<sup>4</sup> They can mimic syncope when the seizure is atypical and not associated with tonic-clonic movements, the seizure is not observed, or a complete history cannot be obtained. In addition, some patients with syncope present with abnormal movements that are suggestive of a seizure but are actually due to cerebral hypoxia. One distinguishing feature is that patients with seizures rarely have an abrupt and complete recovery. Instead, the postictal state is characterised by slow and complete recovery. Another important clue, if present, is evidence of soft tissue injury at multiple sites due to tonic-clonic movements during the seizure.

### Objective information

Perform a good general examination including lying and standing blood pressure, cardiovascular and neurological examinations. Other examinations including abdomen may be indicated. Check blood sugar (BM) and if possible take an electrocardiogram (ECG).

### Analysis and plan

After taking a thorough history and performing a thorough examination the practitioner will need to make the decision whether the patient can be safely managed at home or if they need referral for a medical assessment. Certain features may indicate that the patient is at higher risk of complications (box 3).<sup>5</sup>

#### Box 3 Worrying features in the history of a patient who had collapsed

- ▶ Symptoms and signs of heart disease or arrhythmia
- ▶ Abnormal ECG
- ▶ Multiple episodes
- ▶ Neurological deficit
- ▶ Age over 70
- ▶ Sudden loss of consciousness with injury, marked tachycardia or exertional syncope
- ▶ Moderate to severe postural hypotension

If none of these features are present then the patient is at low risk of serious problems. If the history suggests the possibility of a cardiac cause, or if the patient has abnormal examination findings, they should be referred immediately to hospital for further investigation. Those patients who have a single episode with a history suggestive of benign cause—for example, vasovagal syncope in a young, otherwise fit person—may be left at home if they have fully recovered. They should be advised to seek medical attention if the episode recurs or if they develop any new symptoms. If the patient has had multiple episodes over a relatively short period of time, but the history is unhelpful and there are no abnormal findings, they should be referred to their general practitioner (GP) for assessment and further referral as necessary.

### THE PATIENT PRESENTING WITH DIZZINESS

The term dizziness is used to describe a variety of vague symptoms including floating, swimming, light headedness, and giddiness. Vertigo on the other hand is the specific sensation that the environment or individual is rotating or undergoing angle motion. Equilibrium and spatial orientation depends upon the interaction between the visual system (eye and eye muscles), proprioceptive system (posterior columns, muscles, joints and tendons), and the vestibular system (ear, eighth cranial nerve, brainstem and cerebellum). Disturbance of any part, or the interaction between them, may lead to the symptom of dizziness. Vertigo is usually due to defects in the vestibular system alone. The various conditions that may be described as dizziness by the patient are summarised in table 3.

The history and neurological examination could suggest the possible cause of the symptoms. Particular attention should be paid to the presence of nystagmus, cerebellar signs, and ataxia. Patients who have a history of sudden onset of symptoms and who have signs of brainstem or cerebellar dysfunction may have had a posterior circulation stroke and should be referred to hospital for assessment. If a vestibular cause is suspected then referral to the GP or ENT clinic may be appropriate. Vertigo is covered in more detail in the ABC article on the assessment and care of ENT problems.<sup>6</sup> Care



**Figure 1** Third nerve palsy right eye: ptosis, eye deviated down and out in primary position, dilated pupil. Courtesy of Medical-on-Line/Mediscan.

should be taken in elderly people in whom the symptom of dizziness may be due to a combination of disorders and may be compounded by poor general mobility.

**THE PATIENT PRESENTING WITH VISUAL DISTURBANCE**

The practitioner may be called to see a patient whose main symptom is altered vision. To form a differential diagnosis and management plan it is important to establish the precise nature of this including the mode of onset (sudden or gradual), duration, and any associated symptoms—for example, pain. The examination should include inspection of the eye, visual acuity, pupil reaction, visual fields, and eye movements. The commonest types of visual disturbance the practitioner will encounter are blurred or double vision (diplopia), visual loss, and migrainous aura (table 4).

**Diplopia or blurred vision**

It is important to establish whether the patient has true diplopia or blurred vision. Ask specific questions such as “Do you see two of everything or are objects blurred around the edges?”. Try to determine whether it is present with the eyes in a resting position or only in certain directions of gaze. Establish whether the onset was sudden or gradual. Gradual onset of blurred vision may be due to refractory problems or diabetes. If there are no other abnormal findings on examination and the blood sugar is normal these patients may be managed at home with



**Figure 2** Fourth nerve palsy right eye: failure of abduction. Courtesy of Dr P Marazzi/SPL model released.

advice to see their optometrist or GP. In contrast, sudden onset of blurred vision or diplopia should always be taken seriously as it may be a symptom of an ocular or neurological condition requiring urgent investigation and/or treatment. Diplopia may be due to a defect at any level of the brainstem, cranial nerves, neuromuscular junction, ocular muscles, or orbit. The commonest causes of diplopia are cranial nerve palsies (third, fourth, sixth cranial nerves) (figs 1 and 2), and conditions causing weakness of the ocular muscles.

**Third cranial nerve palsy**

Weakness of eye movement whereby the eye is deviated “down and out” in the resting position is only one feature of a third nerve palsy which may also cause ptosis and dilatation of the pupil (see fig 1). Causes include posterior communicating artery aneurysm (which is usually painful), and pathology in the cavernous sinus, superior orbital fissure, or orbit.

**Fourth cranial nerve palsy**

This causes incomplete depression of the eye in the adducted position. The patient may try to compensate by tilting their head towards the opposite shoulder. It is uncommon but may be caused by trauma affecting the orbit.

**Table 4** Common visual symptoms

Main visual symptom	Causes	Clinical clues
Double vision (diplopia)	Cranial nerve palsy Weakness of ocular muscles—for example, multiple sclerosis, myasthenia gravis, Guillain-Barré syndrome	Abnormal eye movements Often associated with other neurological symptoms and signs
Sudden visual loss	Central retinal vein occlusion Retinal artery occlusion Retinal detachment Optic neuritis Stroke	Abnormal funduscopy
Complete		Flashing lights Associated with pain
Partial	Multiple sclerosis	Associated with other neurological signs May be other neurological symptoms/signs
Transient	Glaucoma Transient ischaemic attack, amaurosis fugax	Painful red eye, vomiting Risk factors for cerebrovascular disease. May be other neurological symptoms
Migrainous aura	Occur in approximately 80% of people with migraine	Usually precede onset of headache Typically flickering zigzag lines starting in centre of vision and gradually proceeding towards the periphery of one visual field often leaving a scotoma. Usually resolves in less than an hour

### Sixth cranial nerve palsy

This may cause some in-turning of the eye and double vision in the primary position (fig 2). The patient may try to compensate by turning their head to the side of the eye affected to obtain single vision by looking forward. Causes include multiple sclerosis and raised intracranial pressure.

Conditions causing weakness of the ocular muscles include myasthenia gravis, multiple sclerosis and the Guillain-Barré syndrome. Onset may be acute or gradual. Usually these conditions will be associated with other neurological symptoms and signs not confined to the eyes. In general, any cranial nerve palsy or sudden onset of diplopia requires a neurological assessment, and these patients should be assessed in hospital.

### Sudden visual loss

Acute loss of vision always merits urgent hospital assessment.

#### Tip

Key points in the history that will aid in making the diagnosis are the presence of pain, whether the loss of vision is in one eye or both, and whether it is partial, complete, or transient.

Acute painless loss of vision in one eye is most commonly due to retinal arterial or venous occlusion or temporal arteritis. Central retinal artery or vein occlusion causes complete loss of vision in one eye whereas branch occlusions may result in partial visual field loss. Patients should be asked about vascular risk factors and previous history of stroke or myocardial infarction. Symptoms such as headache, malaise, myalgia, and weight loss in a patient aged over 50 years should raise the suspicion of temporal arteritis. Little can be done in the prehospital setting to affect outcome, however, these conditions should be treated as medical emergencies for two reasons.

- ▶ In the case of central retinal artery occlusion the prognosis for visual recovery is directly related to the promptness of treatment, and urgent referral to ophthalmology is indicated.
- ▶ If a branch occlusion has occurred, or if the visual loss is a result of temporal arteritis, the rest of the retina or the other eye is at risk. If the underlying conditions are not treated the patient may go on to develop complete blindness.

Other causes of painless loss of vision requiring urgent assessment include retinal detachment and vitreous haemorrhage. Retinal detachment is usually associated with “floaters” or flashing lights at the onset, and the patient may describe the loss of vision “like a curtain falling”. It is more common in shortsighted people. Vitreous haemorrhage may occur with central retinal vein occlusion or retinal detachment and may also occur spontaneously in patients with proliferative diabetic retinopathy. Transient painless loss of vision with or without other neurological symptoms is most likely to be a transient ischaemic attack (TIA). Patients should be referred for investigation as discussed later in this article.

The commonest cause of a painful loss of vision is optic neuritis. The onset may be subacute with visual blurring

gradually getting worse over a few days until the patient can see very little. The pain is typically worse on eye movement. A common underlying condition is multiple sclerosis and this diagnosis should be suspected in young patients, particularly if they have a previous history of other neurological symptoms.

Reduction in visual acuity associated with pain, headache and vomiting may be due to glaucoma (see Assessment and management of neurological problems (1)<sup>1</sup>). If there is any suggestion of this the patient must be referred immediately for an ophthalmological assessment.

### THE PATIENT WITH FOCAL NEUROLOGICAL DEFICIT

It is not uncommon for the community practitioner to encounter a patient who has developed a focal neurological deficit. The spectrum of severity is wide, ranging from the patient with dense unilateral signs (for example, stroke, hemiplegic migraine, Todd's paresis) to the patient with isolated symptoms in one muscle or dermatome (for example, peripheral nerve palsy or mononeuropathy). The cause will usually be apparent from the history and examination and the practitioner must formulate a management plan based on the underlying condition.

#### Box 4 World Health Organization definition of stroke<sup>7</sup>

Clinical syndrome characterised by “rapidly developed clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin”.

### Stroke and TIA

Stroke is the commonest cause of a unilateral motor and/or sensory deficit. A TIA by definition will resolve completely within 24 hours of onset, although it is often not possible to make this distinction if the patient is seen shortly after the onset of symptoms. Approximately 80% of strokes are due to cerebral ischaemia, with the remainder being due to haemorrhage (15%) or unknown cause (5%). However, in practice it is impossible to make the distinction between an ischaemic stroke and a haemorrhagic stroke based on the history and examination alone. The neurological examination will usually indicate which vascular territory is affected. For ischaemic strokes a commonly used classification is the Oxford Community Stroke Project (Bamford *et al*) classification (table 5), which gives an indication of stroke severity and prognosis.<sup>8</sup>

Stroke is a medical emergency. With active management in the initial hours after stroke, vulnerable areas of the brain around the infarct may be saved from infarction and morbidity and mortality may be improved. The Royal College of Physicians has issued comprehensive guidelines on the acute management of stroke and TIA.<sup>9</sup> Some patients with stroke will be primary survey positive—for example, reduced conscious level, fast atrial fibrillation—and should be managed according to ABC principles. All other patients with persistent symptoms that may be due to stroke should be referred urgently to hospital for specialist assessment and brain imaging. Some units may be offering thrombolysis for

**Table 5** Oxford Community Stroke Project classification of stroke<sup>8</sup>

Stroke subtype	Clinical features
Total anterior circulation syndrome (TACS)	Unilateral motor and/or sensory deficit of at least two areas of the face, arm, and leg Homonymous hemianopia Higher cerebral dysfunction—for example, aphasia, neglect
Partial anterior circulation syndrome (PACS)	Any two of: unilateral motor and/or sensory deficit ipsilateral hemianopia higher cerebral dysfunction
Posterior circulation syndrome (POCS)	Any of: ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficit bilateral motor and/or sensory deficit disorder of conjugate eye movement cerebellar dysfunction without ipsilateral long tract deficit (that is, ataxic hemiparesis) isolated homonymous visual field defect
Lacunar syndromes (LACS)	
Pure motor stroke (PMS)	Unilateral pure motor deficit clearly involving two of three areas—face, arm, and leg—with the whole of any limb being involved
Pure sensory stroke (PSS)	Unilateral purely sensory symptoms ± signs involving at least two of the three areas with the whole of any limb being involved
Ataxic hemiparesis (AH)	Ipsilateral cerebellar and corticospinal tract signs with or without dysarthria in the absence of higher cerebral dysfunction or visual field deficit
Sensory motor stroke (SMS)	PMS and PSS combined—that is, unilateral motor and sensory signs in the absence of higher cerebral dysfunction or visual field deficit

**Box 5 Investigation and management of TIA<sup>9</sup>**

- ▶ Patients first seen in the community should be assessed and investigated in a specialist service—for example, neurovascular clinic—as soon as possible within seven days of the incident
- ▶ Patients likely to have a diagnosis of TIA should be prescribed an alternative antiplatelet regimen immediately
- ▶ Patients with more than one TIA in a week should be investigated in hospital immediately
- ▶ Risk factors for cerebrovascular disease such as severe hypertension should be treated appropriately or the patient referred for specialist management

eligible patients presenting within three hours of stroke onset in which case time is of the essence.

Patients who have had symptoms of stroke that have completely resolved by the time they are seen by the practitioner—that is, have a possible diagnosis of TIA—should be treated with the same urgency as stroke, as the risk of developing a stroke after a TIA can be as high as 20% in the first month, with the greatest risk being in the first 72 hours.

When making a diagnosis of TIA it is important to establish that the symptoms were *focal*, came on *suddenly* and were *maximal at onset*. Vague symptoms of dizziness not associated with focal neurological deficit and symptoms associated with loss of consciousness are rarely due to TIA and other causes should be considered (see tables 2 and 3). Whether patients thought to have had a TIA are referred immediately to hospital or to a direct access clinic will depend on local protocols, however the National Clinical Guidelines<sup>9</sup> should be followed (box 5). If there are no contraindications the patient should be started on aspirin or an alternative antiplatelet agent—for example, dipyridamole. Patients who

**Box 6 International Headache Society classification of prolonged auras**

- ▶ Migraine with prolonged aura—if the aura lasts more than one hour but less than one week with normal brain imaging
- ▶ Persistent aura without infarction—if the aura lasts more than one week with no evidence of infarction
- ▶ Migrainous infarction (if the deficit lasts more than one hour and the neuroimaging shows an infarction)

have had a TIA and are in atrial fibrillation should be referred to hospital as anticoagulation may help to prevent a stroke.

**Hemiplegic migraine**

Migraine may be associated with sensory, motor, or aphasic aura. Hemiplegic migraine is characterised by unilateral sensory and/or motor signs. One population study found that 37% of patients with migraine had sensory aura and 6% had motor aura, usually affecting the hand and arm.<sup>10</sup> In general the motor aura tended to be most prolonged, lasting more than an hour in 67% of cases. In 93% of cases the aura preceded the onset of headache. If the patient has a history of hemiplegic migraine and the focal symptoms resolve in less than an hour the patient may be managed at home. Patients in whom the aura has lasted for longer than an hour should be referred for hospital assessment as they may be developing complicated migraine (box 6) and will need neuroimaging to exclude migrainous infarction. Caution must be exercised in diagnosing migraine, especially if it is associated with neurological signs, when the patient has no previous history of migraine, or if the history differs from their usual migraine symptoms. In such cases alternative diagnoses—for example, subarachnoid haemorrhage—must be excluded.<sup>1</sup>

**Table 6** Conditions that may present with generalised weakness

Condition	Clinical clues
Guillain-Barré syndrome	Rapidly progressive ascending weakness May involve respiratory muscles Loss of reflexes May have severe back pain Respiratory or gastrointestinal illness two to four weeks prior to onset
Botulism	Symmetric descending weakness
Wound botulism	Cranial nerve weakness—for example, diplopia
Food borne botulism	No sensory symptoms Prodromal nausea, vomiting, diarrhoea Suspect in intravenous drug users
Spinal cord disease	Back pain Sensory level Sphincter disturbance
Motor neurone disease	Progressive weakness over months Fasciculation of muscles Exaggerated reflexes May have bulbar involvement
Vasculitic neuropathy	Painful Multiple peripheral nerves affected (mononeuritis multiplex) Raised inflammatory markers May be associated with rash, arthropathy, renal disease
Myasthenia gravis	Bulbar, ocular, and respiratory muscles affected Ptosis Fatigability
Viral myositis	Painful muscles Raised creatine kinase
Drug induced myopathy	Progressive proximal weakness over weeks Myalgia Drugs history—for example, statins
Metabolic disorders	Alcoholism
Hypokalaemia	Anorexia
Hypomagnesaemia	Drugs—for example, laxatives, diuretics
Hypophosphataemia	
Polymyalgia rheumatica	Elderly patient Proximal pain and weakness Malaise May be associated with weight loss, headache, Raised ESR

### Todd's paralysis

Todd's paralysis (also called postictal paresis), is a transient neurological deficit following an epileptic seizure. As the name implies, the classic deficit is weakness of a hand, arm, or leg that appears following focal motor seizure activity involving one limb or side of the body. The diagnosis may be apparent from the history and should be considered in a patient with a history of epilepsy. One study of patients with intractable focal epilepsy found the incidence of Todd's paralysis to be 13.4%.<sup>11</sup> The signs were always unilateral and ranged in duration from 11 seconds to 22 minutes. If the patient has a history of epilepsy and the neurological symptoms have completely resolved then they may be safely managed at home. The practitioner must be aware however that acute stroke may be associated with fitting, therefore if the patient has any persistent neurological signs or does not have a history of epilepsy, they should be referred to hospital for further investigation.

### Mononeuropathy

Making the diagnosis of a mononeuropathy can be difficult. It depends on recognising that the neurological deficit is confined to the distribution of a single nerve, and it requires good knowledge of peripheral nerve anatomy and of the

motor and sensory territories of each nerve. A good history may give a clue to the underlying cause. Common causes include trauma, external compression (for example, after a period of unconsciousness or sleep), internal compression (for example, median nerve entrapment in the carpal tunnel), or intrinsic lesions of the nerve (for example, arising as a focal manifestation of a more generalised process such as vasculitis). A full neurological examination should be performed to determine the extent of the deficit. A general examination should also be done to exclude any underlying condition. Patients with diabetes have an increased risk of nerve injury therefore blood sugar should be checked. If the signs are confined to the distribution of a single nerve and the patient is otherwise well then they can be managed at home, however, arrangements will need to be made for further investigation that may include nerve conduction studies.

### THE PATIENT WITH GENERALISED WEAKNESS

Patients may present with less specific symptoms of generalised weakness. This can present particular diagnostic difficulty as it may be due to a wide range of conditions affecting the spinal cord, nerves or muscles, some of which may be serious and require urgent investigation and treatment. Once again a thorough history and examination are the key to determining the likely differential diagnosis. The patient should be asked specifically about the speed of onset and about any associated symptoms such as difficulty with speech or swallowing (bulbar symptoms), breathing difficulties, visual disturbance, pain, and twitching of the muscles. Conditions to consider are listed in table 6. Any patient with bulbar symptoms or shortness of breath should be referred to hospital immediately. All patients will require some investigation either as an inpatient or as an urgent outpatient to determine the cause.

### SUMMARY

The community practitioner may be asked to see patients with a wide range of neurological symptoms that can be difficult to diagnose with certainty in the community. A structured history and examination is vital in formulating a differential diagnosis and in deciding which patients should be referred for urgent investigation and treatment. In general symptoms of acute onset require immediate referral whereas those of subacute (over days to weeks) or chronic onset may be more appropriately investigated in an outpatient setting if the patient is otherwise well.

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C M Gavin, J T Gray, Hope Hospital, Salford, UK

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## CORRECTIONS

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In the short report titled, Not all cases of neck pain with/without torticollis are benign: unusual presentations in a paediatric accident and emergency department (*Emerg Med J* 2005; 22:645–8) two errors have occurred. The corresponding address for A Natarajan is incorrect and should be Consultant Paediatrician, anatarajan@hotmail.com. The second error is in the legend for figure 1. It should read 'T1-weighted MRI scan of the cervical spine showing a large intramedullary tumour in the cervical and upper thoracic region C1-T2.' The journal apologises for these errors.

doi: 10.1136/emj.2003.11403corr1

An author's error occurred in the paper titled Hazardous drinkers in the accident and emergency department—Who accepts advice? (*Emerg Med J* 2004;21:491–2). Incorrect proportions for 'Believed initial AED attendance related to drinking' appear in table 1 (A). The figures were calculated using a denominator based on the number of responses to that item, rather than the number of patients who were offered advice. The correct proportions are 49.9 for 'Accepted advice' and 45.9 for 'Did not accept advice' (not 70.1 and 69.8 as stated in the text). The difference in proportion (95% CI) should read 4.0 (-2.1 to 10.1).

doi: 10.1136/emj.2003.10247corr1

In the paper titled, Comparison of the effectiveness of intravenous diltiazem and metoprolol in the management of rapid ventricular rate in atrial fibrillation (*Emerg Med J* 2005;22:411–4) an error has occurred in table 4. At 20 minutes, places of systolic and diastolic pressures were exchanged. The author apologises for this error.

doi: 10.1136/emj.2005.002005

In part 15 of the ABC of community emergency care (*Emerg Med J* 2005;22: 564–71) the legend for figure 2 is incorrect. It should read 'Sixth nerve palsy right eye: failure of abduction. Courtesy of Dr P Marazzi/SPL model released'.

## Clinical Evidence—Call for contributors

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