Atypical presentations of acute cerebrovascular syndromes

Jonathan A Edlow, Magdy H Selim

Lancet Neurol 2011; 10: 550–60

Harvard Medical School, Boston, MA, USA (J A Edlow MD, M H Selim MD); **and Department of Emergency Medicine** (J A Edlow) **and Department of Neurology** (M H Selim), **Beth Israel Deaconess Medical Center, Boston, MA, USA**

Correspondence to: Jonathan A Edlow, Department of Emergency Medicine, Beth Israel Deaconess Medical Center, West Clinical Center 2, One Deaconess Road West CC-2, Boston, MA 02215, USA
jedlow@bidmc.harvard.edu

Correct diagnosis of acute stroke is of paramount importance to clinicians to enable selection of correct treatments and to ensure prevention of acute complications, including recurrent stroke. Timely diagnosis can be difficult in some cases because patients with acute stroke can present with atypical or uncommon symptoms that suggest another cause altogether. Publications on these patients suggest that the following strategies could help to reduce misdiagnosis. First, clinicians should suspect stroke in any patient with abrupt onset of neurological symptoms. Second, clinicians should be aware that some patients will initially present with various uncommon and atypical stroke symptoms. Third, a complete and systematic neurological examination should be routinely done in patients presenting with acute neurological symptoms because this might shed light on the true nature of the problem. Finally, clinicians should be aware that even with the most sophisticated neuroimaging tests, stroke might be missed in the early hours after the event.

Introduction

Worldwide, about 15 million people have a stroke every year, of whom 5 million die and 5 million have a permanent deficit.[1] In high-income countries, stroke is the most common cause of disability and is the third most common cause of death. When patients present with acute neurological symptoms, physicians must distinguish between stroke and other causes that mimic it. Accurate and prompt diagnosis is crucial because implementation of time-dependent therapies, identification and treatment of the underlying vascular mechanism, and attention to the underlying risk factors can improve outcomes and prevent subsequent events.

Patients who present with acute neurological symptoms can be divided into four groups (table). The first group includes patients with obvious stroke—eg, an elderly individual with untreated atrial fibrillation who abruptly develops aphasia and hemiparesis. The second group includes patients whose cause of neurological dysfunction is clearly not stroke, such as a patient without vascular risk factors who develops unilateral weakness of the facial muscles in association with hyperacusis and altered taste consistent with a lower motor neuron facial nerve palsy. In the third group, patients seem to have a stroke, but in fact have a non-vascular cause such as conversion reactions, Todd's paralysis (a disorder characterised by a brief period of paralysis with or without aphasia after a seizure), or migraine. Hypoglycaemia is a particularly important stroke mimic because its treatment is simple and effective. In the era of thrombolysis for acute ischaemic stroke, these stroke mimics have received much attention.[2–5] The final group of patients includes those with actual strokes, but whose presentations are unusual or atypical, suggesting a non-vascular cause. Huff has referred to these patients as "stroke chameleons".[2] Compared with stroke mimics, this group of patients with missed or delayed diagnosis of stroke has received much less attention and is the focus of this Review.

Patients with stroke can present with atypical symptoms for various reasons. First, in the first minutes to hours after the event, all the diagnostic information might not be available to the initial health-care providers. Additionally, patients' symptoms can evolve with time. Second, there is substantial variability in the classic cerebrovascular territories that can also result in non-classic presentations. Patients with small strokes, early presentations, young age, posterior circulation location, and deficits that do not result in lateralising motor or speech findings might be more difficult to diagnose clinically.[6–9]

In this Review, we aim to help clinicians improve the accuracy and timeliness of their diagnosis of patients with acute stroke by reviewing causes of misdiagnosis and non-classic symptoms of acute stroke (panel 1). Although much of the focus is on acute ischaemic stroke, we also discuss some aspects of misdiagnoses of haemorrhagic strokes, including intracerebral haemorrhage (ICH), subarachnoid haemorrhage (SAH), and haemorrhage resulting from cerebral venous sinus thrombosis (CVST). Panel 2 provides a glossary of terms of atypical symptoms of stroke.

Non-localising symptoms

Stroke is usually characterised by the sudden onset of focal neurological deficits, such as hemiparesis, aphasia, or hemianopia, depending on the affected brain structures and vascular territory. However, some strokes can present in a non-localising manner without clear-cut focal deficits.

Neuropsychiatric symptoms

About 3% of patients with stroke can present with delirium, a delusional state, acute onset of dementia, or mania mimicking a psychiatric illness.[10] Neurological signs are often absent or mild and transient, and therefore might be easily missed. This presentation is usually seen in patients with right-sided (non-dominant) focal strokes in the frontal and parietal regions. Several stroke-related symptoms and signs, such as anosognosia, aphasia, akinetic mutism, abulia, and aprosodia, can be misinterpreted as manifestations of depression. For example, a patient with a right frontal or parietal stroke

	Stroke-like presentation	Atypical stroke-like presentation
True stroke	Stroke	Stroke "chameleons"[2]
Not a stroke	Stroke mimic	Non-stroke

Table: **Diagnosis of patients presenting with acute neurological symptoms**

might be unable to correctly perceive or express the appropriate emotional inflection owing to aprosodia and could have monotonous speech; therefore, these patients might be misdiagnosed as having an affective disorder.[11]

Caudate strokes in the territory of the anterior lenticulostriate arteries might present with only mild neuropsychological and behavioural symptoms, such as abulia, mental and affective stagnation, and impairments in initiative for action, speech, and usual daily activities.[12] Similar features have been reported in patients with isolated strokes in the frontal lobes and underlying subcortical structures, presumably due to interruption of the limbic-frontal connections, and connections to the thalamus. Mania-like presentation, with associated psychosis, might occur in patients with focal strokes in the right orbitofrontal cortex, thalamus, and temporoparietal region.[13] Partial complex seizures due to temporal lobe injury might account for the psychotic symptoms in many patients with temporoparietal strokes.[14]

Although pathological laughing and crying and uncontrollable fits of laughing and crying inappropriate to the context are common sequelae of stroke, these symptoms are relatively rare at stroke presentation. This disorder is most common with strokes that affect the supranuclear motor pathways, bilateral pontine, basal ganglia, or periventricular subcortical areas, and with focal strokes in the frontal or temporal regions.[15] Catastrophic reaction, a collection of symptoms indicative of a patient's desperation and frustration and that include anxiety, aggression, and refusal of treatment, are also not uncommon in patients with stroke, particularly those with left anterior subcortical strokes and premorbid depression.[16]

Acute confusional state

Delirium can be the presenting feature in a few patients with stroke,[17] particularly after a hemispheric stroke; this state is more frequent in haemorrhagic stroke than in ischaemic stroke (figure 1).[18] Strokes involving the right temporal gyrus, right inferior parietal lobe, or occipital lobe can present with acute confusional states, agitation, restlessness, and easy-to-miss neurological signs, and can be misdiagnosed as delirium. Rarely, vertebrobasilar ischaemia leading to involvement of the thalami, particularly the paramedian nuclei, can present as unexplained rapid onset of confusion with amnesia and minimal neurological deficits.[19] This form of amnesia is not to be confused with transient global amnesia, the acute onset of transient loss of memory for recent events and impaired ability to retain new information with an otherwise normal neurological examination.[20]

Patients with strokes of the corpus callosum can present with symptoms that are difficult to localise or neurological symptoms and signs attributable to associated interhemispheric disconnection syndromes and can be misdiagnosed as being confused.[21] Patients

Panel 1: **Non-classic presentations of acute stroke**

Non-localising symptoms
- Neuropsychiatric symptoms
- Acute confusional state
- Altered level of consciousness

Abnormal movements or seizures
- Abnormal movements
- Limb-shaking transient ischaemic attacks
- Seizures
- Alien hand syndrome
- Localised asterixis
- Isolated hemifacial spasms
- Disappearance of previous essential tremor

Peripheral nervous system symptoms
- Acute vestibular syndrome
- Other cranial nerve palsies (especially third and seventh cranial nerves)
- Acute monoparesis
  - Cortical hand syndrome
  - Cortical foot syndrome
- Isolated sensory symptoms

Atypical symptoms
- Isolated dysarthria
- Isolated dysarthria-facial paresis syndrome
- Isolated visual symptoms
  - Anton's syndrome (cortical blindness with denial of deficit)
  - Balint's syndrome
  - Isolated visual field disturbances
- Foreign accent syndrome
- Isolated dysphagia or stridor

Isolated headache
- Subarachnoid haemorrhage
- Cerebral venous sinus thrombosis
- Cervical artery dissections
- Cerebellar infarction

Acute neurological syndrome with negative brain imaging
- Negative non-contrast CT in subarachnoid haemorrhage, cerebral venous sinus thrombosis, arterial dissection, and acute ischaemic stroke
- Negative MRI in acute ischaemic stroke

Panel 2: Glossary

Abulia
Absence of initiative and diminished desire to undertake a task

Akinetic mutism
A state characterised by mutism and loss of volitional movements

Anosagnosia
Absence of awareness of neurological deficits

Apraxia
Inability to carry out skilled movements and gestures, despite having the desire and the physical ability to undertake them

Aprosodia
Inability to properly comprehend or express variations in tone of voice used to express linguistic and emotional information

Graphesthesia
Ability to identify characters that are written on the palm of the hands with eyes closed, purely based on the sense of touch

Hemianopia
Loss of vision in one half of the visual field

Prosopagnosia
Inability to recognise familiar faces

Stereognosis
Ability to recognise objects by the sense of touch, with eyes closed

Visual agnosia
Inability to recognise familiar objects

with stroke who have a predominantly receptive aphasia are also commonly mistaken as being confused. Difficulty with comprehension is often intermixed with some expressive difficulties, such as use of nonsensical words, and can often obscure assessment; a hemianopsia can be difficult to detect, especially if not specifically tested for. Detailed language and visual field examination and a clear sensorium provide clues to the correct diagnosis.

Similarly, patients with bilateral strokes involving the primary and visual association areas often present with visual agnosia, prosopagnosia, and anosognosia. These deficits can be difficult to detect by an inexperienced clinician and might be mistaken for a confusional state. Classic examples include Anton's syndrome, associated with bilateral occipital infarcts and cortical blindness and characterised by denial of being blind and confabulated responses, and Balint's syndrome, caused by bilateral occipitoparietal strokes and characterised by a visual perceptual deficit and an inability to recognise more than one object at a time.

Altered level of consciousness

Rapid deterioration of level of consciousness and unresponsiveness can be the presenting feature of large strokes, particularly haemorrhages associated with a rapid increase in intracranial pressure. This presentation can also be caused by ictal or post-ictal unresponsiveness owing to seizures at stroke onset. However, two unique posterior circulation stroke syndromes are worth mentioning. The first is embolic occlusion of the central artery of Percheron, a variant arterial supply where the bilateral medial thalamic and rostral midbrain perforators arise from a single trunk from the P1 segment of one posterior cerebral artery, resulting in infarcts of those areas. Such patients present with coma and few, if any, other neurological signs.[22] The second is the top-of-the-basilar syndrome, caused by embolic occlusion of the distal portion of the basilar artery where it branches into the posterior cerebral arteries. Patients with this syndrome present with unconsciousness, quadriplegia, and occasionally incontinence; pupillary abnormalities or oculomotor signs, often bilateral, are present in more than 40% of patients and can provide clues to the diagnosis.[23]

Abnormal movements or seizures

Stroke is usually characterised by loss of movement. However, in a small percentage of cases, patients can have various abnormal movements (hyperkinetic, hypokinetic, or seizure-like) at stroke onset.

Abnormal movements

Movement disorders are a well recognised delayed complication of stroke. However, several abnormal movements, such as dystonia, chorea, athetosis, tremors, myoclonus, convulsions, jerking movements, limb shaking, and asterixis, can occasionally manifest at stroke onset.[24,25] In the Lausanne Stroke Registry,[25] the prevalence of a movement disorder in 2500 patients with acute stroke was 1%; hemichorea, hemiballismus, and dystonia were the most common symptoms. Small deep strokes involving the basal ganglia, caused by presumed small-vessel disease, were most often associated with abnormal movements.[25] However, abnormal movements in patients with stroke do not show a specific predilection to arterial territory, stroke site, or stroke type or subtype; large-vessel atherosclerosis, cardioembolism, haemorrhages, and thalamic, cerebellar, and brainstem involvement can be associated with abnormal movements at stroke onset.[24–28]

Handley and colleagues[27] reviewed 2942 papers from 1966 to 2008 on movement disorders after stroke and concluded that dystonia, chorea, and hemiballismus most commonly result from basal ganglia strokes;

tremors most commonly result from strokes that involve the posterior thalamus or the dentatorubrothalamic pathway; and strokes that involve the striatum or lentiform nucleus can result in parkinsonism. Ghika-Schmid and colleagues[25] reported that a jerky dystonic unsteady hand syndrome is specifically associated with small strokes in the territory of the posterior choroidal artery. Myoclonus is particularly common in posterior circulation strokes.[24] Segmental myoclonus can be seen in midbrain and pontine strokes, and palatal myoclonus (regular and rhythmic contraction of the soft palate) can be the only manifestation of a small pontine stroke.

When patients present with involuntary repetitive hyperkinetic movements of the limbs, clinicians might diagnose focal motor seizures and not consider stroke or cerebral ischaemia. However, small strokes in the basis pontis can present with involuntary tonic spasms and hemiparesis of the contralateral limb.[29] Similarly, clonic, convulsive-like or posturing-like limb movements can be seen in patients with thalamic or brainstem strokes.[30] These abnormal movements might be associated with disturbance to the function of the corticospinal tract (the descending inhibitory fibres affecting the anterior horn cells of the involved limb). These movements can also be seen in patients presenting with the top-of-the-basilar syndrome and might be misdiagnosed as status epilepticus. However, the presence of oculomotor palsies, often bilateral, and absence of epileptiform discharges on electroencephalography during these movements provide additional clues to the diagnosis. Awareness of this presentation and a high degree of suspicion are necessary for early identification and urgent treatment of this life-threatening disorder.

Limb-shaking transient ischaemic attacks

Involuntary repetitive and stereotyped limb shaking might be the manifestation of diminished perfusion of the frontosubcortical motor pathways.[31] Fisher described repetitive involuntary transient trembling and shaking of limbs contralateral to a high-grade carotid occlusive lesion, and emphasised that these were motor manifestations of transient ischaemic attacks and not epileptic in nature.[32] These abnormal movements have been termed as limb-shaking transient ischaemic attacks. They are often brief and show postural dependence, being precipitated by abrupt standing up and relieved by lying down. Limb-shaking transient ischaemic attacks preferentially affect the upper limbs and spare facial muscles and are almost always contralateral to a tight carotid stenosis. Early recognition of limb-shaking transient ischaemic attacks is crucial as these patients are at high risk for stroke if steps to improve cerebral perfusion (augmentation of blood pressure, optimisation of intravascular volume, and carotid revascularisation) are not done expeditiously. The precipitation of these abnormal movements with orthostatic changes and the absence of a Jacksonian seizure help to distinguish them from focal motor seizures.[33]

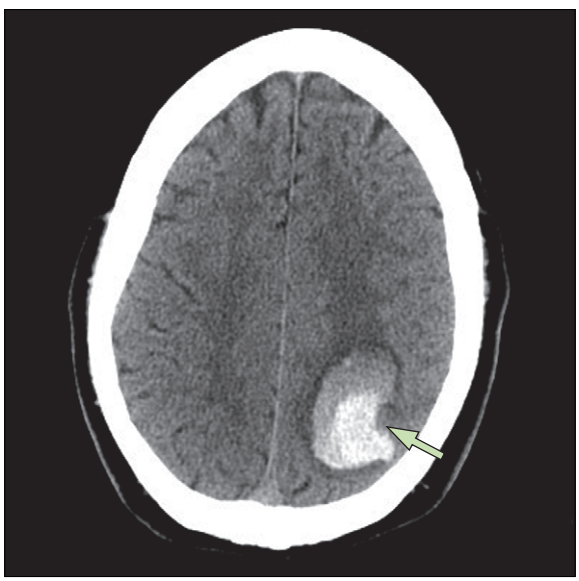

Figure 1: **CT of a patient with acute confusional state**
This patient aged 67 years presented with acute confusion and inattentiveness. He had driven himself to a routine primary-care doctor appointment 2 h earlier.The patient recalled signing in at the physician's office with his name at an angle on the page, but he did not recall being at the office or driving himself home. On returning home, his wife immediately noticed that he was confused and that he was "using incoherent words". She brought him to an emergency department, where this CT was obtained. At the emergency department, he complained of a mild "burning sensation" in his head, but he had no motor or sensory complaints or any abnormal findings on neurological examination. Arrow shows left parietal intracerebral haemorrhage.

Seizures

Seizures occurring in the setting of acute stroke are not uncommon, with a frequency that varies from 1·5% to 5·7% in various studies.[34–36] The prevalence of seizures at stroke presentation tends to be higher in younger patients and those with haemorrhagic strokes, infarcts involving the cerebral cortex, and watershed infarctions at the borders of the internal carotid artery territory.[34–36] It is important for clinicians to differentiate postictal Todd's paralysis from deficits attributable to a stroke with a seizure at onset. In the initial minutes to hours, such a distinction is often difficult on the basis of clinical examination alone. The use of advanced brain imaging techniques, such as perfusion and vascular imaging, is often needed to discriminate paralysis attributable to seizures alone from that caused by a stroke and these techniques could facilitate treatment decisions.[37,38]

The prevalence of presenting seizures is particularly high in patients with cerebral sinus thrombosis and venous infarcts. Nearly 40% of patients in the International Study on Cerebral Vein and Dural Sinus Thrombosis had

seizures at presentation.[39] Eliciting the history of ongoing headaches or symptoms and signs of elevated intracranial pressure, such as papilloedema, in these patients could provide clues to the correct diagnosis.

Other symptoms

One of the most interesting rare presentations of stroke is the so-called alien hand syndrome, in which one hand seems to have a mind of its own and acts independently of the patient's voluntary control. This syndrome can be seen in patients with strokes involving the corpus callosum, frontal lobe, or posterolateral parietal lobe.[40,41] Alien hand syndrome is thought to result from disconnection of the area of the primary motor cortex that controls the hand from the premotor cortex, while retaining its ability to execute hand movements. Physicians who are unaware of this unusual presentation might mistake this symptom for a psychiatric disorder.

Another interesting presentation of some strokes is unilateral asterixis, which might be mistaken for an underlying metabolic derangement. Asterixis can occur in focal mesodiencephalic strokes, resulting in impaired processing of proprioceptive input, and in cortical strokes that involve the primary motor cortex, with subsequent impairment of centrally generated motor-command signals that control the postural tone of the distal upper limb.[42–44]

Isolated hemifacial spasms might be the only presenting signs of an ipsilateral lacunar pontine stroke.[45] The hemifacial spasms are thought to result from irritation of the intra-pontine roots of the facial nerve or its nucleus by ischaemic oedema, leading to hyperexcitability of the facial motor neurons and interneurons that mediate the blink reflex.[46]

Conversely, disappearance of abnormal movements might be the presenting feature of a stroke. In a few reports, improvement of patients' essential tremors has been described after strokes that affect the cerebellum, frontal lobe, thalamus, and basis pontis.[47–49] These authors speculated that interruption of transcortical motor and cerebellar-thalamic-cortical loops by a stroke could result in disappearance of the tremors.

Peripheral nervous system symptoms

Although stroke is a CNS event, some cases present in ways that suggest a peripheral nervous system (PNS) cause.

Acute vestibular syndrome

One specific and relatively common clinical presentation is the acute vestibular syndrome. These patients have abrupt onset of dizziness, nausea, vomiting, headache, intolerance to head motion, nystagmus, and unsteady gait. Although PNS problems such as acute vestibular neuritis are the most common causes, some of these patients have posterior circulation stroke. In one series of 240 patients with cerebellar stroke, 25 (10%) presented with acute vestibular syndrome and no obvious CNS findings.[50] In another study, 76 of 101 (75%) patients with this syndrome had a cerebrovascular cause, but patients at higher risk for stroke were intentionally recruited in this study.[51] Pontine stroke can also present with an acute vestibular syndrome.[52]

Misdiagnosis is more common in patients with vertebrobasilar strokes than with other stroke types.[7] Symptoms can be minimal and non-specific and the patients themselves might not recognise the gravity of their symptoms.[53] Physicians might not consider a stroke diagnosis because of the relative youth of many of these patients.[7] Patients discharged from emergency departments in California, USA, with a diagnosis of vertigo or dizziness had an elevated stroke risk during the ensuing month.[54] The results from the neurological examination can be deceptively benign; some individuals will have a stroke with a National Institutes of Health stroke scale (NIHSS) score of zero.[55] If misdiagnosed, some of these patients can have poor outcomes.[56]

Isolated infarction of the vestibular nucleus has also been reported,[57,58] an event that leads to what seems to be a vestibular neuritis caused by a stroke. In these cases, careful bedside assessment might help to confirm the correct diagnosis. Patients with acute vestibular syndrome who have a stroke often have more serious gait problems and other findings on neurological examination compared with patients with the acute vestibular syndrome that has a peripheral cause.[50,59] Detailed oculomotor evaluation can also provide information on the true nature of the problem. In particular, the combination of negative results from a head impulse test and the presence of skew deviation and direction-changing or vertical nystagmus strongly suggests a CNS cause.[51]

Other cranial neuropathies

Because the cranial nerve nuclei reside in the brainstem, they are frequently affected in brainstem strokes. Most commonly, there are other obvious neurological findings such as dysarthria, ataxia, long tract signs, and abnormalities in consciousness. However, occasionally, cranial neuropathy will predominate. Isolated, or nearly isolated, cranial neuropathy from infarction of either the nucleus or fibres as the nerve exits the brainstem is rare but does occur, most commonly with the third and seventh cranial nerves.[60–62] This neuropathy is more commonly reported in patients who have risk factors for small-vessel disease, such as diabetes, hypertension, and hyperlipidaemia.

Although co-involvement of hearing and vertigo suggests a peripheral lesion, stroke of the anterior inferior cerebellar artery territory can affect both hearing and vestibular function.[63] Acute hearing loss can result from labyrinthine infarction due to stroke of

the labyrinthine artery. This event is not attributable to involvement of the cranial nerve nucleus but to infarction of the labyrinth and is most often reported in patients with diabetes.

Acute monoparesis and cortical hand syndrome

Acute monoparesis (isolated unilateral face, arm, or leg weakness) is another stroke presentation that can suggest a PNS disorder. In two large studies of 6805 patients, between 2·5% and 4·1% of patients presented with a monoparesis (which included isolated facial weakness in one study).[64,65] In some patients, the stroke was in a subcortical structure. In one of the studies,[64] 24% of patients had ICH and 40% of strokes were caused by small-vessel disease.

Cortical hand syndrome is a classic but uncommon stroke syndrome. Because the area known as the cortical "hand knob" is large (relative to the amount of anatomy it serves), a small stroke affecting this region of the precentral gyrus can lead to a very targeted deficit involving only the hand, several fingers, or even just the thumb (figure 2).[66–68] Because either the radial or the ulnar side can predominate, physicians might misdiagnose this as cervical disc disease or a radial or ulnar neuropathy. Cortical sensations, including stereognosis, graphesthesia, and point localisation, in the affected hand should be carefully assessed because they are often impaired and testing could assist the correct diagnosis. Strokes presenting with a cortical hand syndrome are often caused by an artery-to-artery embolism that results from ipsilateral carotid artery atherosclerosis or from cardioembolism.[67,69]

Although arm or hand monoparesis is more common, leg monoparesis accounts for about a third of monoparesis from stroke.[64] The classic situation is in the setting of anterior cerebral artery stroke, in which the medial surface of the precentral gyrus is affected.[70] Many of these patients will have subtle weakness of the ipsilateral deltoid muscle and some can also have sensory dysfunction in the paretic limb, identified by undertaking thorough sensory and motor examinations.[70] As with upper extremity monoparesis, isolated leg weakness can occur with subcortical strokes.[64,65] Predominant leg involvement can also occur with middle cerebral artery territory strokes, CVST, and haemorrhages.[71]

Finally, analogous to cortical hand syndrome, some patients with both ischaemic and haemorrhagic stroke can present with a cortical foot syndrome.[72,73] In these patients, isolated foot drop mimics a peroneal nerve lesion.

Isolated sensory symptoms

Patients with pure sensory strokes can be misidentified as having PNS or psychiatric disorders. Pure sensory strokes are uncommon; subtle motor or cognitive findings are commonly present, but isolated sensory symptoms do occur. The most common site of sensory strokes is the posterior thalamus (figure 3).[17,74,75] In the Lausanne Stroke Registry of more than 3600 patients, less than 1% had thalamic strokes that presented with pure sensory symptoms.[76] These patients usually present with abnormal sensation in more than one body region (face, arm, trunk, leg) rather than having peripheral causes of abnormal sensation in which only one region is typically involved. Pure sensory strokes can, however, occur anywhere along the sensory axis from the cortex to the brainstem.[77–79] Although sensory loss is the usual presentation, these strokes can occasionally present with positive sensory changes in the form of paraesthesias.

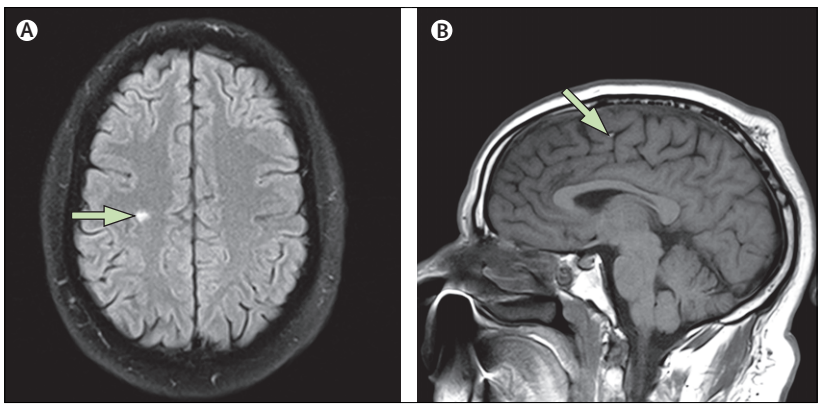

Figure 2: **MRI of a patient with cortical hand syndrome**
(A,B) This 51-year-old patient presented with weakness of three fingers of the left hand and was incorrectly diagnosed as having a peripheral nerve problem. The MRI, which was ordered 2 days later by a neurologist who was seeing the patient for follow-up, shows a tiny infarct at the hand knob on the motor cortex (arrows).

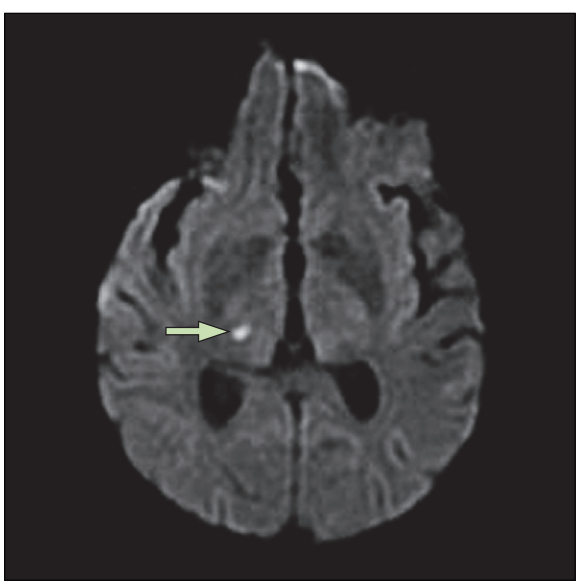

Figure 3: **DW-MRI of a patient with thalamic lacunar stroke**
This 68-year-old man presented with numbness of the left side of his body (face, arm, and leg) and had diminished sensation to pin prick. There were no other neurological symptoms or signs. This DW-MRI shows a small infarct in the right thalamus (arrow). DW-MRI=diffusion-weighted MRI.

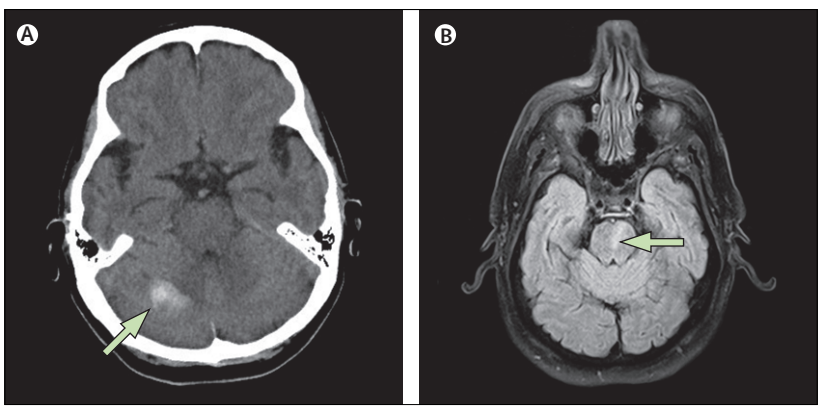

Figure 4: **CT and MRI scans of two different patients who presented with dysarthria**
(A) This 67-year-old patient presented with isolated dysarthria; she had ataxic speech. There was no headache and on examination, the patient had normal finger-to-nose and gait testing. The CT scan shows a right-sided cerebellar haemorrhage (arrow). (B) This 82-year-old patient awoke with slurred speech. Although there were no motor complaints, on examination, he had a very slight right-sided pronator drift, emphasising the importance of a complete neurological examination in patients who present with neurological symptoms. The fluid-attenuated inversion recovery MRI shows a left-sided paramedian pontine infarct (arrow).

Atypical symptoms

Rarely, patients with stroke present with atypical and unusual symptoms and signs. Increased awareness of these unusual presentations facilitates early recognition, minimises unnecessary tests, and facilitates prompt treatment.

Dysarthria

Dysarthria caused by stroke is often associated with other neurological deficits. Isolated dysarthria without associated sensorimotor deficits is poorly localising, difficult to interpret, and often attributed to toxic or metabolic derangements (figure 4). In a review of 68 patients with acute ischaemic stroke and dysarthria, the dysarthria was isolated in only two (3%) patients.[80] Discrete strokes in the anterior limb or superior part of the genu of the internal capsule, neighbouring corona radiata, or pons that selectively involve the corticobulbar fibres can present with the so-called isolated dysarthria-facial paresis syndrome.[81,82] The facial paresis is usually very subtle and resolves rapidly. Occasionally, lingual, palatal, pharyngeal, and laryngeal weakness is present, but might not be easily detectable on examination. Similarly, small strokes involving the dominant opercular and medial frontal cortices might present with only pure dysarthria.[83]

Visual symptoms

Visual disturbances can be the predominant, if not the only, presenting symptom in some strokes. These signs include blindness with denial of deficit and confabulation (Anton's syndrome) or a visual agnosia with the inability to perceive more than one object at a time, oculomotor apraxia, and optic ataxia (Balint's syndrome), as mentioned earlier. Identification of these peculiar deficits requires specific knowledge of these syndromes and a sophisticated examination. Isolated homonymous hemianopia occurs mainly in strokes that involve the occipital cortex and optic radiations.[84] Strokes involving the V1 or V2 areas of the visual cortex or optic radiation can present with positive spontaneous visual phenomena, making migraine or occipital seizures possible diagnoses.[85]

Other symptoms

One of the most intriguing presentations of stroke is the so-called foreign accent syndrome. This is predominantly characterised by a change in speech prosody and articulation resulting in altered phonetics, which is perceived as a foreign accent. This symptom has been described with strokes that involve the left (dominant) frontoparietal regions and subcortical structures including the basal ganglia, although a role for the cerebellum has also been suggested.[86–88] Unlike aphasia, the patient's speech is usually perfectly clear, and those meeting such patients for the first time assume that they have a different first language.

Rarely, isolated dysphagia can be the only presentation of a discrete brainstem or medullary stroke, which might or might not be visible on brain imaging.[89] The sudden onset, absence of other causes, and, rarely, presentation with mild nausea or vertigo are important diagnostic clues. A lateral medullary stroke can present with dysphonia, difficulty breathing, and stridor caused by vocal cord paralysis.[90] Other neurological signs including Horner's syndrome, ipsilateral facial anhidrosis, and contralateral appendicular sensory impairment, can be present, but can be subtle. This variability in presentation emphasises the importance of a detailed and thorough neurological examination.

Isolated headache

Another stroke presentation, in both ischaemic and haemorrhagic disorders, is the presence of a prominent headache that is either isolated or associated with non-specific symptoms that are not obviously attributable to a cerebrovascular cause.

Isolated headache can occur with arterial dissections, CVST, and SAH.[91–93] Although headache suggests ICH, patients with acute ischaemic stroke can also present with prominent headache.[94] Headache at onset (with or without concomitant dizziness, vomiting, ataxia, and dysarthria) is particularly common with cerebellar infarction.[95,96] These clinical events are especially important because non-contrast CT scans might be negative in such cases and clinicians must consider undertaking lumbar puncture (SAH) and CT with contrast (dissection and CVST) or MRI (dissection, CVST, and cerebellar infarction), depending on the differential diagnosis in an individual patient.[97] Unilateral headaches are common presenting symptoms in patients with posterior cerebral artery infarcts and can lead to misdiagnosis of complicated migraine.[98]

Headache with stroke can also be associated with a history of migraine, young age, and female sex.[96]

Limitations of brain imaging

Although stroke is a clinical diagnosis, nearly all patients undergo brain imaging. The purpose of brain imaging in patients with acute neurological symptoms includes establishing a stroke diagnosis and excluding stroke mimics. CT scanning can establish the diagnosis of stroke (ICH, SAH, and occasionally acute ischaemic stroke) and exclude stroke mimics (in patients with acute ischaemic stroke who are candidates for thrombolysis). MRI is being increasingly used to definitively confirm a diagnosis of acute ischaemic stroke.

As with any diagnostic test, clinicians must understand the limitations of brain imaging. Despite the remarkable advances in CT and MRI, neither test is perfect. CT scanning is very sensitive for blood. There is nearly always evidence of blood on non-contrast CT in patients with ICH.[99] However, patients with small or late-presenting SAHs can have false-negative CT scans. For this reason, if SAH is being considered, the clinician should do a lumbar puncture after a non-diagnostic CT.[93,100] In patients with CVST, a plain CT scan often shows non-specific findings or might be normal. MRI is better than CT for diagnosing CVST, especially when gadolinium is used. Regardless of the brain imaging technique, dedicated imaging of the sinuses and veins with magnetic resonance venography or CT venography is needed to confirm CVST. Communicating the differential diagnostic possibilities to radiologists before the scan might also help to make the correct diagnosis.

For acute ICH, MRI is as sensitive as CT.[99] For diagnosis of acute ischaemic stroke, diffusion-weighted MRI (DW-MRI) is better than CT.[101–103] DW-MRI is recommended as the imaging study of choice for patients with acute stroke,[104] and MRI is excellent for establishing a diagnosis of ICH or SAH and for excluding stroke mimics.

Although data from head-to-head studies indicate that DW-MRI is better than CT for diagnosis of stroke, DW-MRI is not perfect and clinicians must understand its limitations. When compared with the final clinical diagnosis, MRI had sensitivities that varied between 83% and 97%.[101–103] The lowest sensitivity (83%) was reported in the study that included patients who most accurately mirrored patients with stroke symptoms in clinical practice.[101] Patients with small lacunar strokes, brainstem location, and low NIHSS scores are more likely to have a false-negative DW-MRI scan.[101,105] Although interpretation error is less common with MRI than with CT, this factor is another potential cause of a false-negative study. Finally, some patients with seizures will have a false-positive DW-MRI scan. Thus, clinicians must know that even DW-MRI is imperfect for definitively ruling in the diagnosis of acute ischaemic stroke.

Conclusions

The topics covered in this Review might suggest that almost any neurological symptom could be indicative of a stroke; what practical conclusions can the clinician derive from this? By design, we assessed many case reports and case series, which tend to overestimate the atypical manifestations of stroke, but understanding this variability of presentation is key.

In various systematic studies, acute speech and lateralising motor findings consistently correlate with stroke diagnosis.[6,106] In these same studies, the absence of these symptoms, or the presence of seizures or altered mental status, suggests a non-stroke diagnosis. However, these studies were done to help distinguish strokes from stroke mimics and were not focused on diagnosing atypical stroke syndromes. Nevertheless, this highlights the tension between classic stroke symptoms (which suggest the diagnosis) and atypical ones (which, while consistent with stroke, might lead the doctor away from the correct diagnosis). The abruptness of symptom onset in a patient with risk factors for stroke is probably the most important feature in stroke diagnosis. Although some patients with stroke have fluctuating symptoms, most patients with stroke have an abrupt onset.

Another useful clinical feature is the concept of positive symptoms versus negative symptoms. A positive neurological symptom is the abnormal presence of a given function, whereas a negative symptom is its absence. Stroke-related symptoms are more often negative (ie, loss of movement, loss of sensation, blindness). For example, for a motor symptom, abnormal shaking movements would be positive whereas weakness (absence of movement) would be negative. For visual symptoms, flashing lights is a positive sign, whereas blindness is a negative sign. As with most rules in medicine, this one has exceptions, which we have presented, but it is still a useful concept.

In conclusion, the presentation of stroke is as heterogeneous as its causes. In the appropriate clinical circumstances, four tenets could help the clinician to make an accurate diagnosis. First, suspect stroke in any

Search strategy and selection criteria

We identified articles for this Review through searches of PubMed using the search terms "stroke", "misdiagnosis", and "atypical presentations" from 1980 to March, 2011. Additional articles were identified from the bibliographies of the initial articles and from the authors' own files. With one exception, we reviewed articles only published in English. The final reference list was generated on the basis of originality and relevance to the broad scope of this Review; therefore, we included many case reports and case series.

cases of abrupt onset of neurological symptoms. Second, be aware of these rare and atypical stroke presentations. Third, routinely undertake a complete neurological examination in patients who present with acute neurological symptoms because this step might shed light on the true nature of the problem. Finally, be aware that even the most sophisticated neuroimaging techniques might miss a stroke in the early hours.

Contributors

Both authors planned the structure of the Review, helped to identify articles, and prepared and edited the manuscript.

Conflicts of interest

We declare that we have no conflicts of interest.

References

1 WHO. Cardiovascular disease. The atlas of heart disease and stroke. http://www.who.int/cardiovascular_diseases/resources/atlas/en/ (accessed Dec 4, 2010).
2 Huff JS. Stroke mimics and chameleons. *Emerg Med Clin North Am* 2002; **20:** 583–95.
3 Hand PJ, Kwan J, Lindley RI, Dennis MS, Wardlaw JM. Distinguishing between stroke and mimic at the bedside: the brain attack study. *Stroke* 2006; **37:** 769–75.
4 Hemmen TM, Meyer BC, McClean TL, Lyden PD. Identification of nonischemic stroke mimics among 411 code strokes at the University of California, San Diego, Stroke Center. *J Stroke Cerebrovasc Dis* 2008; **17:** 23–25.
5 Kothari RU, Brott T, Broderick JP, Hamilton CA. Emergency physicians. Accuracy in the diagnosis of stroke. *Stroke* 1995; **26:** 2238–41.
6 Goldstein LB, Simel DL. Is this patient having a stroke? *JAMA* 2005; **293:** 2391–402.
7 Kuruvilla A, Bhattacharya P, Rajamani K, Chaturvedi S. Factors associated with misdiagnosis of acute stroke in young adults. *J Stroke Cerebrovasc Dis* 2010; published online Aug 17. DOI:10.1016/j.jstrokecerebrovasdis.2010.03.005.
8 Nakajima M, Hirano T, Uchino M. Patients with acute stroke admitted on the second visit. *J Stroke Cerebrovasc Dis* 2008; **17:** 382–87.
9 Nor AM, Ford GA. Misdiagnosis of stroke. *Expert Rev Neurother* 2007; **7:** 989–1001.
10 Dunne JW, Leedman PJ, Edis RH. Inobvious stroke: a cause of delirium and dementia. *Aust N Z J Med* 1986; **16:** 771–78.
11 Huffman J, Stern TA. Acute psychiatric manifestations of stroke: a clinical case conference. *Psychosomatics* 2003; **44:** 65–75.
12 Kumral E, Evyapan D, Balkir K. Acute caudate vascular lesions. *Stroke* 1999; **30:** 100–08.
13 Cummings JL, Mendez MF. Secondary mania with focal cerebrovascular lesions. *Am J Psychiatry* 1984; **141:** 1084–87.
14 Levine DN, Finklestein S. Delayed psychosis after right temporoparietal stroke or trauma: relation to epilepsy. *Neurology* 1982; **32:** 267–73.
15 Balakrishnan P, Rosen H. The causes and treatment of pseudobulbar affect in ischemic stroke. *Curr Treat Options Cardiovasc Med* 2008; **10:** 216–22.
16 Carota A, Rossetti AO, Karapanayiotides T, Bogousslavsky J. Catastrophic reaction in acute stroke: a reflex behavior in aphasic patients. *Neurology* 2001; **57:** 1902–05.
17 Carrera E, Michel P, Bogousslavsky J. Anteromedian, central, and posterolateral infarcts of the thalamus: three variant types. *Stroke* 2004; **35:** 2826–31.
18 Sinanovic O. Neuropsychology of acute stroke. *Psychiatr Danub* 2010; **22:** 278–81.
19 Salam A, Sanmuganathan P, Pycock C. Unusual presentation of basilar artery stroke secondary to patent foramen ovale: a case report. *J Med Case Reports* 2008; **2:** 75.
20 Toledo M, Pujadas F, Grive E, Alvarez-Sabin J, Quintana M, Rovira A. Lack of evidence for arterial ischemia in transient global amnesia. *Stroke* 2008; **39:** 476–79.
21 Kasow DL, Destian S, Braun C, Quintas JC, Kagetsu NJ, Johnson CE. Corpus callosum infarcts with atypical clinical and radiologic presentations. *AJNR Am J Neuroradiol* 2000; **21:** 1876–80.
22 Kumral E, Evyapan D, Balkir K, Kutluhan S. Bilateral thalamic infarction. Clinical, etiological and MRI correlates. *Acta Neurol Scand* 2001; **103:** 35–42.
23 Caplan LR. "Top of the basilar" syndrome. *Neurology* 1980; **30:** 72–79.
24 Alarcon F, Zijlmans JC, Duenas G, Cevallos N. Post-stroke movement disorders: report of 56 patients. *J Neurol Neurosurg Psychiatry* 2004; **75:** 1568–74.
25 Ghika-Schmid F, Ghika J, Regli F, Bogousslavsky J. Hyperkinetic movement disorders during and after acute stroke: the Lausanne Stroke Registry. *J Neurol Sci* 1997; **146:** 109–16.
26 Ferbert A, Gerwig M. Tremor due to stroke. *Mov Disord* 1993; **8:** 179–82.
27 Handley A, Medcalf P, Hellier K, Dutta D. Movement disorders after stroke. *Age Ageing* 2009; **38:** 260–66.
28 Kim JS. Delayed onset mixed involuntary movements after thalamic stroke: clinical, radiological and pathophysiological findings. *Brain* 2001; **124:** 299–309.
29 Kaufman DK, Brown RD Jr, Karnes WE. Involuntary tonic spasms of a limb due to a brain stem lacunar infarction. *Stroke* 1994; **25:** 217–19.
30 Ropper AH. 'Convulsions' in basilar artery occlusion. *Neurology* 1988; **38:** 1500–01.
31 Tatemichi TK, Young WL, Prohovnik I, Gitelman DR, Correll JW, Mohr JP. Perfusion insufficiency in limb-shaking transient ischemic attacks. *Stroke* 1990; **21:** 341–47.
32 Fisher CM. Concerning recurrent transient cerebral ischemic attacks. *Can Med Assoc J* 1962; **86:** 1091–99.
33 Maddula M, Keegan B. Limb shaking as a manifestation of low-flow TIA. *Int J Gerontol* 2010; **4:** 47–50.
34 Denier C, Masnou P, Mapoure Y, et al. Watershed infarctions are more prone than other cortical infarcts to cause early-onset seizures. *Arch Neurol* 2010; **67:** 1219–23.
35 Shinton RA, Gill JS, Melnick SC, Gupta AK, Beevers DG. The frequency, characteristics and prognosis of epileptic seizures at the onset of stroke. *J Neurol Neurosurg Psychiatry* 1988; **51:** 273–76.
36 Szaflarski JP, Rackley AY, Kleindorfer DO, et al. Incidence of seizures in the acute phase of stroke: a population-based study. *Epilepsia* 2008; **49:** 974–81.
37 Mehdiratta M, Selim M. Value of perfusion computed tomography in stroke mimics. *Arch Neurol* 2007; **64:** 1348–49.
38 Selim M, Kumar S, Fink J, Schlaug G, Caplan LR, Linfante I. Seizure at stroke onset: should it be an absolute contraindication to thrombolysis? *Cerebrovasc Dis* 2002; **14:** 54–57.
39 Ferro JM, Canhao P, Bousser MG, Stam J, Barinagarrementeria F. Early seizures in cerebral vein and dural sinus thrombosis: risk factors and role of antiepileptics. *Stroke* 2008; **39:** 1152–58.
40 Bejot Y, Caillier M, Osseby GV, et al. Involuntary masturbation and hemiballismus after bilateral anterior cerebral artery infarction. *Clin Neurol Neurosurg* 2008; **110:** 190–93.
41 Spector AR, Freeman WD, Cheshire WP. The stroke that struck back: an unusual alien hand presentation. *J Stroke Cerebrovasc Dis* 2009; **18:** 72–73.
42 Donat JR. Unilateral asterixis due to thalamic hemorrhage. *Neurology* 1980; **30:** 83–84.
43 Nighoghossian N, Trouillas P, Vial C, Froment JC. Unilateral upper limb asterixis related to primary motor cortex infarction. *Stroke* 1995; **26:** 326–28.
44 Peterson DI, Peterson GW. Unilateral asterixis due to ipsilateral lesions in the pons and medulla. *Ann Neurol* 1987; **22:** 661–63.
45 Kawakami M, Sato T, Tochigi S, Ito T. Lacunar pontine infarction with hemifacial spasm as the initial symptom. *Stroke* 1990; **21:** 1236.
46 Ambrosetto P, Forlani S. Lacunar pontine infarction presenting as isolated facial spasm. *Stroke* 1988; **19:** 784–85.
47 Constantino AE, Louis ED. Unilateral disappearance of essential tremor after cerebral hemispheric infarct. *J Neurol* 2003; **250:** 354–55.

48 Dupuis MJ, Evrard FL, Jacquerye PG, Picard GR, Lermen OG. Disappearance of essential tremor after stroke. *Mov Disord* 2010; **25:** 2884–87.

49 Kim JS, Park JW, Kim WJ, Kim HT, Kim YI, Lee KS. Disappearance of essential tremor after frontal cortical infarct. *Mov Disord* 2006; **21:** 1284–85.

50 Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. *Neurology* 2006; **67:** 1178–83.

51 Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke* 2009; **40:** 3504–10.

52 Thomke F, Hopf HC. Pontine lesions mimicking acute peripheral vestibulopathy. *J Neurol Neurosurg Psychiatry* 1999; **66:** 340–49.

53 Gargano JW, Wehner S, Reeves MJ. Presenting symptoms and onset-to-arrival time in patients with acute stroke and transient ischemic attack. *J Stroke Cerebrovasc Dis* 2010; published online Aug 17. DOI:10.1016/j.jstrokecerebrovasdis.2010.02.022.

54 Kim AS, Fullerton HJ, Johnston SC. Risk of vascular events in emergency department patients discharged home with diagnosis of dizziness or vertigo. *Ann Emerg Med* 2011; **57:** 34–41.

55 Martin-Schild S, Albright KC, Tanksley J, et al. Zero on the NIHSS does not equal the absence of stroke. *Ann Emerg Med* 2011; **57:** 42–45.

56 Savitz SI, Caplan LR, Edlow JA. Pitfalls in the diagnosis of cerebellar infarction. *Acad Emerg Med* 2007; **14:** 63–68.

57 Kim HA, Lee H. Isolated vestibular nucleus infarction mimicking acute peripheral vestibulopathy. *Stroke* 2010; **41:** 1558–60.

58 Hong CT, Lai YJ, Chan JL. Vertigo with an isolated lesion in brainstem vestibular nucleus and nerve. *Neurology* 2010; **74:** 437.

59 Kerber KA, Brown DL, Lisabeth LD, Smith MA, Morgenstern LB. Stroke among patients with dizziness, vertigo, and imbalance in the emergency department: a population-based study. *Stroke* 2006; **37:** 2484–87.

60 Kim JS, Kim J. Pure midbrain infarction: clinical, radiologic, and pathophysiologic findings. *Neurology* 2005; **64:** 1227–32.

61 Kumral E, Bayulkem G, Evyapan D. Clinical spectrum of pontine infarction. Clinical-MRI correlations. *J Neurol* 2002; **249:** 1659–70.

62 Keane JR. Third nerve palsy: analysis of 1400 personally-examined inpatients. *Can J Neurol Sci* 2010; **37:** 662–70.

63 Lee H, Kim JS, Chung EJ, et al. Infarction in the territory of anterior inferior cerebellar artery: spectrum of audiovestibular loss. *Stroke* 2009; **40:** 3745–51.

64 Paciaroni M, Caso V, Milia P, et al. Isolated monoparesis following stroke. *J Neurol Neurosurg Psychiatry* 2005; **76:** 805–07.

65 Maeder-Ingvar M, van Melle G, Bogousslavsky J. Pure monoparesis: a particular stroke subgroup? *Arch Neurol* 2005; **62:** 1221–24.

66 Gass A, Szabo K, Behrens S, Rossmanith C, Hennerici M. A diffusion-weighted MRI study of acute ischemic distal arm paresis. *Neurology* 2001; **57:** 1589–94.

67 Takahashi N, Kawamura M, Araki S. Isolated hand palsy due to cortical infarction: localization of the motor hand area. *Neurology* 2002; **58:** 1412–14.

68 Sudo K, Kishimoto R, Tajima Y, Matsumoto A, Tashiro K. A paralysed thumb. *Lancet* 2004; **363:** 1364.

69 Peters N, Muller-Schunk S, Freilinger T, During M, Pfefferkorn T, Dichgans M. Ischemic stroke of the cortical "hand knob" area: stroke mechanisms and prognosis. *J Neurol* 2009; **256:** 1146–51.

70 Kang SY, Kim JS. Anterior cerebral artery infarction: stroke mechanism and clinical-imaging study in 100 patients. *Neurology* 2008; **70:** 2386–93.

71 Schneider R, Gautier JC. Leg weakness due to stroke. Site of lesions, weakness patterns and causes. *Brain* 1994; **117:** 347–54.

72 Hiraga A, Kamitsukasa I. Isolated foot drop due to subcortical hemorrhage. *J Neurol* 2010; **257:** 1741–42.

73 Ku BD, Lee EJ, Kim H. Cerebral infarction producing sudden isolated foot drop. *J Clin Neurol* 2007; **3:** 67–69.

74 Schmahmann JD. Vascular syndromes of the thalamus. *Stroke* 2003; **34:** 2264–78.

75 Wang X, Fan YH, Lam WW, Leung TW, Wong KS. Clinical features, topographic patterns on DWI and etiology of thalamic infarcts. *J Neurol Sci* 2008; **267:** 147–53.

76 Paciaroni M, Bogousslavsky J. Pure sensory syndromes in thalamic stroke. *Eur Neurol* 1998; **39:** 211–17.

77 Derouesne C, Mas JL, Bolgert F, Castaigne P. Pure sensory stroke caused by a small cortical infarct in the middle cerebral artery territory. *Stroke* 1984; **15:** 660–62.

78 Kim JS. Pure sensory stroke. Clinical-radiological correlates of 21 cases. *Stroke* 1992; **23:** 983–87.

79 Shintani S, Tsuruoka S, Shiigai T. Pure sensory stroke caused by a pontine infarct. Clinical, radiological, and physiological features in four patients. *Stroke* 1994; **25:** 1512–15.

80 Urban PP, Wicht S, Vukurevic G, et al. Dysarthria in acute ischemic stroke: lesion topography, clinicoradiologic correlation, and etiology. *Neurology* 2001; **56:** 1021–27.

81 Celebisoy M, Tokucoglu F, Basoglu M. Isolated dysarthria-facial paresis syndrome: a rare clinical entity which is usually overlooked. *Neurol India* 2005; **53:** 183–85.

82 Kim JS. Pure dysarthria, isolated facial paresis, or dysarthria-facial paresis syndrome. *Stroke* 1994; **25:** 1994–98.

83 Urban PP, Wicht S, Hopf HC, Fleischer S, Nickel O. Isolated dysarthria due to extracerebellar lacunar stroke: a central monoparesis of the tongue. *J Neurol Neurosurg Psychiatry* 1999; **66:** 495–501.

84 Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V. Homonymous hemianopia in stroke. *J Neuroophthalmol* 2006; **26:** 180–83.

85 Baier B, de Haan B, Mueller N, et al. Anatomical correlate of positive spontaneous visual phenomena: a voxelwise lesion study. *Neurology* 2010; **74:** 218–22.

86 Cohen DA, Kurowski K, Steven MS, Blumstein SE, Pascual-Leone A. Paradoxical facilitation: the resolution of foreign accent syndrome after cerebellar stroke. *Neurology* 2009; **73:** 566–67.

87 Marien P, Verhoeven J, Engelborghs S, Rooker S, Pickut BA, De Deyn PP. A role for the cerebellum in motor speech planning: evidence from foreign accent syndrome. *Clin Neurol Neurosurg* 2006; **108:** 518–22.

88 Naidoo R, Warriner EM, Oczkowski WJ, Sevigny A, Humphreys KR. A case of foreign accent syndrome resulting in regional dialect. *Can J Neurol Sci* 2008; **35:** 360–65.

89 Lee BC, Hwang SH, Chang GY. Isolated dysphagia due to a medullary infarction: a new lacunar syndrome. *Eur Neurol* 1999; **41:** 53–54.

90 Vaidyanathan S, Capper R, Chadha D. Stridor: an unusual presentation of lateral medullary syndrome. *J Laryngol Otol* 2007; **121:** e9.

91 Arnold M, Cumurciuc R, Stapf C, Favrole P, Berthet K, Bousser MG. Pain as the only symptom of cervical artery dissection. *J Neurol Neurosurg Psychiatry* 2006; **77:** 1021–24.

92 de Bruijn SF, Stam J, Kappelle LJ. Thunderclap headache as first symptom of cerebral venous sinus thrombosis. CVST Study Group. *Lancet* 1996; **348:** 1623–25.

93 Edlow JA, Malek AM, Ogilvy CS. Aneurysmal subarachnoid hemorrhage: update for emergency physicians. *J Emerg Med* 2008; **34:** 237–51.

94 Runchey S, McGee S. Does this patient have a hemorrhagic stroke?: clinical findings distinguishing hemorrhagic stroke from ischemic stroke. *JAMA* 2010; **303:** 2280–86.

95 Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol* 2008; **7:** 951–64.

96 Tentschert S, Wimmer R, Greisenegger S, Lang W, Lalouschek W. Headache at stroke onset in 2196 patients with ischemic stroke or transient ischemic attack. *Stroke* 2005; **36:** e1–3.

97 Savitz SI, Edlow J. Thunderclap headache with normal CT and lumbar puncture: further investigations are unnecessary: for. *Stroke* 2008; **39:** 1392–93.

98 Brandt T, Thie A, Caplan LR, Hacke W. [Infarcts in the brain areas supplied by the posterior cerebral artery. Clinical aspects, pathogenesis and prognosis]. *Nervenarzt* 1995; **66:** 267–74 (in German).

99 Kidwell CS, Chalela JA, Saver JL, et al. Comparison of MRI and CT for detection of acute intracerebral hemorrhage. *JAMA* 2004; **292:** 1823–30.

100 Edlow JA, Panagos PD, Godwin SA, Thomas TL, Decker WW. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache. *Ann Emerg Med* 2008; **52:** 407–36.

101 Chalela JA, Kidwell CS, Nentwich LM, et al. Magnetic resonance imaging and computed tomography in emergency assessment of patients with suspected acute stroke: a prospective comparison. *Lancet* 2007; **369:** 293–98.

102 Fiebach JB, Schellinger PD, Jansen O, et al. CT and diffusion-weighted MR imaging in randomized order: diffusion-weighted imaging results in higher accuracy and lower interrater variability in the diagnosis of hyperacute ischemic stroke. *Stroke* 2002; **33:** 2206–10.

103 Mullins ME, Schaefer PW, Sorensen AG, et al. CT and conventional and diffusion-weighted MR imaging in acute stroke: study in 691 patients at presentation to the emergency department. *Radiology* 2002; **224:** 353–60.

104 Schellinger P, Bryan R, Caplan L, et al. Evidence-based guideline: the role of diffusion and perfusion MRI for the diagnosis of acute ischemic stroke: Report of the Therapeutics and Technology Subcommittee of the American Academy of Neurology. *Neurology* 2010; **75:** 177–85.

105 Doubal FN, Dennis MS, Wardlaw JM. Characteristics of patients with minor ischaemic strokes and negative MRI: a cross-sectional study. *J Neurol Neurosurg Psychiatry* 2010; published online Jun 27. DOI:10.1136/jnnp.2009.190298.

106 Nor AM, Davis J, Sen B, et al. The Recognition of Stroke in the Emergency Room (ROSIER) scale: development and validation of a stroke recognition instrument. *Lancet Neurol* 2005; **4:** 727–34.