Cerebellar infarction is an important cause of stroke that often presents with common and non-specific symptoms such as dizziness, nausea and vomiting, unsteady gait, and headache. Accurate diagnosis frequently relies on careful attention to patients’ coordination, gait, and eye movements—components of the neurological physical examination that are sometimes omitted or abridged if cerebellar stroke is not specifically being considered. The differential diagnosis is broad, and includes many common and benign causes. Furthermore, early-stage posterior fossa ischaemia is rarely seen with brain CT—the most commonly available initial imaging test that is used for stroke. Insufficient examination and imaging can result in misdiagnosis. However, early correct diagnosis is crucial to help prevent treatable but potentially fatal complications, such as brainstem compression and obstructive hydrocephalus. The identification and treatment of the underlying vascular lesions at an early stage can also prevent subsequent occurrences of stroke and improve patients’ outcomes. Here, we review the clinical presentation of cerebellar infarction, from diagnosis and misdiagnosis to patients’ monitoring, treatment, and potential complications.

Introduction
Cerebellar infarction has not received the same level of attention in medical publications as the well-defined anterior circulation and brainstem stroke syndromes. This paucity of data might be partly because the clinical presentation of cerebellar infarction is diverse, and can resemble many other disorders. The main symptoms—dizziness, nausea and vomiting, gait instability, and headache—are non-specific, and are usually caused by more common and benign disorders. The important components of the neurological examination that help to identify cerebellar stroke—coordination, gait, and eye movements—are commonly omitted or abridged in primary care, particularly when symptoms might not suggest a CNS cause. Additionally, brain CT, which for decades has been the most readily available brain imaging study to identify strokes and is still the most commonly used, rarely identifies early-stage cerebellar infarction.

Accurate diagnosis of cerebellar infarction is important. Early oedema from infarction in the posterior fossa can result in potentially fatal—yet treatable—complications, such as brainstem compression and obstructive hydrocephalus. The identification and treatment of the underlying vascular lesion(s) might also prevent a second and more devastating stroke. Medical therapy (eg, thrombolytic, antiplatelet, and anticoagulant drugs), or vascular procedures (eg, stenting) can result in improved patients’ outcomes. The increasing availability of MRI has improved the ability of clinicians to diagnose this disorder definitively, and there have been important advances in bedside examination techniques to identify those patients who are at the highest risk. Physicians must familiarise themselves with the diagnosis and initial management of these patients. Here, we review cerebellar infarction, from diagnosis (and misdiagnosis) to the monitoring, treatment, and potential complications of patients.

Epidemiology
In nine studies of consecutive ischaemic strokes, cerebellar infarction accounted for almost 3% (660 of 23426) of strokes. This proportion, combined with the annual stroke incidence rate, suggests that nearly 20000 new cerebellar infarctions occur each year in the USA, not including patients whose infarcts simultaneously include the cerebellum and other cerebrovascular territories. The average age of patients is 65 years and two-thirds of patients are men. However, some patients present with cerebellar stroke at much younger ages, which could contribute to misdiagnosis because stroke might not be considered by the physician.

General risk factors for ischaemic stroke—hypertension, diabetes, cigarette smoking, hyperlipidaemia, atrial fibrillation, and history of stroke or transient ischaemic attack (TIA)—also apply to cerebellar stroke. As the population of developed countries ages, the incidence of stroke is expected to rise.

Relevant anatomy
The cerebellum, which is composed of two lateral hemispheres and a midline vermis, is important for movement; it modulates the functions of the motor system and corrects for differences between intended and actual movement. Damage to the cerebellum generally leads to inaccurate, erratic, or uncoordinated movements and difficulty with motor learning and adaptation. Many of these functions are regionally specified: the superior parts of the cerebellum are primarily concerned with limb (lateral hemispheres) and trunk (midline vermis) movements and motor control of speech articulation (paravermal area), whereas the inferior areas are primarily associated with oculomotor control and vestibular adaptation. Because projections between the limbs and cerebellum are either uncrossed or doubly crossed, appendicular motor deficits related to unilateral cerebellar lesions tend to be ipsilesional. Other deficits are often bilateral or of poor lateralising value owing to partially redundant, bilateral projections to that control midline muscles of the head, neck, and trunk.

The cerebellum is highly interconnected with other CNS structures through three cerebellar peduncles. The main inputs from the spinal cord and vestibular system enter
the cerebellum by the inferior peduncle at the level of the medulla, whereas the main inputs from the cerebral hemispheres enter by the middle peduncle at the level of the pons. The main outputs from the deep cerebellar nuclei exit predominantly by the superior peduncles near the pontomesencephalic junction.

The three peduncles and the cerebellum form the roof of the fourth ventricle and are situated over part of the aqueduct of Sylvius. The cerebellum and brainstem sit within the tightly constrained posterior fossa, and are bounded above by a rigid dural reflection (tentorium cerebelli) and below by the base of the skull. This anatomical knowledge is important to understand the most serious complications of cerebellar stroke—direct brainstem compression and secondary obstructive hydrocephalus with consequent herniation (figure 1).

Vascular anatomy

The cerebellum receives its blood supply from three paired arteries—the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA), and the superior cerebellar artery (SCA)—which are all branches of the posterior circulation, also known as the vertebrobasilar system (figure 2 and table 1).

Figure 1: Anatomy of the posterior fossa: normal appearance compared with appearance after a stroke

The cerebellum is dorsal to the brainstem and forms the roof of the fourth ventricle. Because of this location and the space constraints of the posterior fossa, swelling from oedema after cerebellar infarction can press on the aqueduct of Sylvius or on the fourth ventricle, which impedes the flow of cerebrospinal fluid and might result in an obstructive hydrocephalus.

The vertebral arteries originate from subclavian vessels, ascend in the neck through the transverse foramina of the cervical spine and exit at the C2 vertebrae to penetrate the dura and enter the cranium. Before fusing into the basilar artery, each vertebral artery usually gives rise to an anterior spinal artery and a PICA. Several small penetrating vessels that directly supply most of the brainstem branch off from the basilar artery; the basilar artery also typically gives rise to the AICA (proximally) and the SCA (distally). After the SCA branches off, the basilar artery splits into the right and left posterior cerebral arteries, which supply the thalamus, medial temporal and occipital lobes. The proximal posterior cerebral arteries are connected to the anterior cerebral circulation by the posterior communicating arteries. Thus, brainstem or hemispheric ischaemia often accompanies cerebellar infarction.

Three other anatomical facts are important to understand cerebellar infarctions. First, proximal branches of the three main cerebellar arteries typically supply the lateral part of...
artery embolism are also important causes. 20–22 Vertebral artery atherosclerosis can be intracranial, extracranial, or communicating arteries.18

In one study of patients less than 40 years old and who had had particularly in younger patients. In one study of patients with cerebellar infarction, both.23–25 The relative occurrence of these different pathological study of 88 posterior circulation infarcts, cerebral and distal structures supplied by major branches.20–22 Vertebral, proximal posterior cerebral, or posterior communicating arteries.18

Pathogenesis

As is the case with ischaemic stroke in the anterior circulation, the two most common causes of cerebellar infarction are cardioembolism and large vessel atherosclerosis.4,5,12,18 Small artery disease and artery-to-artery embolism are also important causes.20–22 Vertebral artery atherosclerosis can be intracranial, extracranial, or both.21,22 The relative occurrence of these different aetiologies and the distribution of large vessel atherosclerosis varies with ethnic origin and sex.6 Because underlying cardiovascular and cerebrovascular pathology is common in stroke patients, cardiac and cerebrovascular imaging is usually recommended for patients with stroke in the posterior circulation as for those with stroke in the anterior circulation.23 Patent foramen ovale is an important consideration for patients with cerebellar infarction, particularly in younger patients. In one study of patients who were less than 40 years old and who had had cerebellar stroke, half of all cardioembolic cerebellar infarcts were caused by patent foramen ovale.12

Vertebral artery dissection is another important cause of cerebellar infarction, particularly in younger patients.28,29 In a series of 169 patients with vertebral dissections, the median age was 43 years and 50% of the patients were men.28 Nearly 80% (n=131) had brain ischaemia, mostly from stroke in the posterior circulation.28 In another study of 37 patients who were aged less than 40 years and had cerebellar stroke, vertebral artery dissection accounted for 27% of cases (n=10), all of which were infarcts in the PICA territory.27 Vertebral artery dissection can also occur in children.28 Although some patients develop dissections after recognised major or minor head or neck trauma, including chiropractic manipulations,31,32 such a history is identified in fewer than half of cases.33,34 Dissection may also be responsible for some cases of so-called “beauty parlour stroke” due to prolonged neck hyperextension,5 because dissection might go unrecognised if it is not investigated specifically.5

Less common disorders that have been associated with cerebellar infarction in isolated cases include hypercoagulable states,12,13 vasculitis (eg, giant cell arteritis39 and meningovascular syphilis40), venous sinus thrombosis,5,41 acute marijuana42 or cocaine use,9 and migraine.5,6,43

Overall, PICA strokes are more common than SCA strokes, and AICA strokes are the least common.24,43,46,47 In the largest series of 293 patients with cerebellar infarction studied so far, 258 (88%) of these infarctions were unilateral, although unilateral infarctions sometimes include more than one vascular area (eg, combined AICA and PICA).28 In one small series

<table>
<thead>
<tr>
<th>Posterior inferior cerebellar artery</th>
<th>Anterior inferior cerebellar artery</th>
<th>Superior cerebellar artery</th>
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<tbody>
<tr>
<td>Typical origin</td>
<td>Typical origin</td>
<td>Typical origin</td>
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<tr>
<td>Vertebral artery</td>
<td>Proximal or mid-basilar artery</td>
<td>Distal basilar artery</td>
</tr>
<tr>
<td>Major branches</td>
<td>Major branches</td>
<td>Major branches</td>
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<tr>
<td>Medial branch, lateral branch</td>
<td>Cerebral branches, internal auditory tract</td>
<td>Medial branch, lateral branch</td>
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<tr>
<td>Key brainstem structures supplied by</td>
<td>Key brainstem structures supplied by</td>
<td>Key brainstem structures supplied by</td>
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<td>proximal branches</td>
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<tr>
<td>Posterior lateral medulla: cranial nerve nuclei (V, VIII) (vestibular, IX, X) and fascicles (IX, X), sympathetic tract, spinohalamic tract, inferior cerebellar peduncle</td>
<td>Posterior lateral pons: cranial nerve nuclei (V, VIII) (vestibular, cochlear) and fascicles (VII, VIII), sympathetic tract, spinohalamic tract, middle cerebellar peduncle</td>
<td>Posterior lateral medulla (and upper lateral pons): cranial nerve nuclei (IV, VI) and fascicle (IV); sympathetic tract, spinohalamic tract, middle medullary truncus, superior cerebellar peduncle</td>
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<tr>
<td>Cerebral and distal structures supplied by</td>
<td>Cerebral and distal structures supplied by</td>
<td>Cerebral and distal structures supplied by</td>
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<td>major branches</td>
<td>major branches</td>
<td>major branches</td>
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<tr>
<td>Posterioroinferior cerebellum, including: inferior vermis (including uvula, nodulus), parafl occulus</td>
<td>Anterioroinferior cerebellum, including: flocculus. Inner ear: vestibular labyrinth; cochlea</td>
<td>Superior cerebellum, including: superior vermis, dentate nucleus</td>
</tr>
<tr>
<td>Core cerebellar syndrome</td>
<td>Core cerebellar syndrome</td>
<td>Core cerebellar syndrome</td>
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<tr>
<td>Isolated acute vestibular syndrome without auditory symptoms (pseudo-vestibular neumitits)</td>
<td>Isolated acute vestibular syndrome without auditory symptoms (pseudo-vestibular neumitits)</td>
<td>Acute gait or trunk instability with associated dysaesthesia (pseudo-intoxication); nausea or vomiting (pseudo-gastroenteritis)</td>
</tr>
<tr>
<td>Indicative neurological signs</td>
<td>Indicative neurological signs</td>
<td>Indicative neurological signs</td>
</tr>
<tr>
<td>Lateral medullary syndrome: hemifacial analgesia†, unilateral absent gag reflex; palatal palsy; vocal cord palsy; Homer’s syndrome, body hemianalgesia†, limb hemiataxia; dysmetria</td>
<td>Lateral pontine syndrome: hemifacial sensory loss; facial palsy (lower motor neuron type); Homer’s syndrome, body hemianalgesia†, limb hemiataxia; dysmetria</td>
<td>Lateral midbrain syndrome: fourth nerve palsy†, hemifacial sensory loss; Homer’s syndrome, body hemisensory loss; limb hemiataxia; dysmetria</td>
</tr>
<tr>
<td>Vertebral artery syndrome: 12th nerve palsy; body hemisensory loss; hemiplegia or quadriplegia</td>
<td>Mid-basilar syndrome: impaired arousal or coma; sixth nerve palsy or internuclear ophthalmoplegia; horizontal gaze palsy; body hemisensory loss; hemiplegia or quadriplegia</td>
<td>Top of the basilar syndrome: impaired memory or attention; visual field cut; ptosis; third nerve palsy; vertical gaze palsy, hemiplegia or quadriplegia</td>
</tr>
</tbody>
</table>

Data from 1,4,5–10. *Note that because the fourth nerve fascicle crosses before exiting the brainstem posteriorly, a superior oblique palsy can occur either ipsilateral (fascicular post-decussation) or contralateral (nuclear, fascicular pre-decussation) to the cerebellar infarction. †Analgesia (loss of sharp sensitivity) and thermotaxis (loss of temperature sensitivity) typically cluster together.
Cardiac embolism might be a more common cause when infarctions are bilateral, but multiple infarcts are still frequently attributed to atherothrombotic disease. Haemorrhagic conversion is probably more common with embolic cerebellar infarctions, as is the case with strokes in the anterior circulation. Patients who have strokes that undergo haemorrhagic transformation are at higher risk for later complications due to the mass effect of the blood.

In addition to typical territorial infarcts (PICA, AICA, and SCA), non-territorial areas that lie at the border between major vascular areas can be selectively affected. Whether these small infarcts (less than 2 cm) are superficial or deep, they are sometimes called “borderzone infarctions” and occur in 23–31% of cases. The vascular risk factors in these patients are similar to those with territorial infarcts. Although deep cerebellar infarcts do occur in patients with chronic hypertension without obvious embolic or large vessel aetiology, there is no evidence that they result from lipohyalinosis of small penetrating vessels; therefore, the term “lacunar stroke”, which is used occasionally in publications about cerebellar infarction, is probably not the preferred term.

**Clinical manifestations**

When infarctions are restricted to the cerebellum, patients typically only experience non-specific symptoms (ie, dizziness, nausea, vomiting, unsteady gait, and headache) and show neurological signs (ie, dysarthria, ataxia, and nystagmus) that might be absent, subtle, or difficult to distinguish from benign disorders of the peripheral vestibular system. Clinical presentations of isolated cerebellar infarction are similar across the three main cerebellar vascular areas.

Kase and co-workers found that vertigo and headache were less common in patients with SCA infarcts (n=30) than in patients with PICA lesions (n=36). However, in the largest published case series so far (n=293), Tohgi and co-workers showed no difference in the frequency of these symptoms on the basis of arterial distribution. Even if measurable clinical differences are apparent among different vascular area presentations, they are mostly unimportant from a diagnostic perspective, as the physician’s first priority is to ascertain and then confirm that a cerebellar infarction has occurred. Because neuroimaging is generally needed to establish a diagnosis of cerebellar infarction, the specific cerebellar area implicated will thus be identified. Therefore, in table 2 we summarise the clinical manifestations of cerebellar infarcts as a group, rather than by specific vascular territory.

Although the presentation of cerebellar infarction, particularly in association with incipient basilar occlusion, can be severe (eg, coma and quadriplegia), such extremes are not usually the case. At initial presentation, altered mental status occurs in only a third of patients; about 26% are confused, and 3% are comatose. Classic “crossed” signs (ie, ipsilesional cranial nerve and contralesional long tract) are indicative of brainstem involvement, but these signs are often absent. Those with lateral medullary (PICA) strokes can present with acute vestibular symptoms in association with subtle, easily missed signs such as Horner’s syndrome or facial hemianalgiesia. Those with lateral pontine (AICA) strokes might present with acute vestibular symptoms in association with sixth or seventh nerve palsies, which mimics a peripheral cranial polyneuropathy. The simultaneous presence of tinnitus or hearing loss, which is suggestive of inner ear ischaemia (typical of AICA-territory strokes), might further blur the distinction between a central and peripheral localisation.

Similar to other ischaemic strokes, the symptoms of cerebellar infarction typically begin abruptly, and if multiple body parts are included, the symptoms often begin simultaneously. Posterior circulation transient ischaemic attacks (TIAs) occur before infarction in about 22% of patients with cerebellar stroke. These patients with TIA have the same, if not higher, short-term stroke risk as those patients with ischaemia in the carotid distribution. As with the anterior circulation, a crescendo pattern of posterior circulation TIAs might suggest incipient large vessel (eg, basilar) occlusion. However, unlike anterior circulation TIAs, the main symptoms of posterior circulation ischaemia are less obviously linked to stroke: dizziness, nausea, vomiting, unsteady gait, and headache.

These non-specific manifestations frequently occur in combination, enabling a more focused differential diagnosis; however, each can occur in relative isolation, which increases the challenge for an accurate diagnosis. Symptoms such as dizziness or headache are common, and most individuals have benign, self-limited problems, rather than cerebellar stroke. This emphasises the importance of the recognising the often subtle indicators of a potentially life-threatening condition. Therefore, a thorough review of systems and detailed physical examination that focuses on the nervous system, head, and neck are important to identify other neurological manifestations that might clarify the need for further testing or specialist consultation.

**Dizziness**

Dizziness (with or without vertigo) occurs in nearly three-quarters of patients with cerebellar infarction and need not imply direct involvement of the inner ear. Dizziness is also an exceptionally common symptom in general practice: the 1-year population prevalence of vestibular vertigo is 5%; dizziness accounts for 5% of all outpatient visits; and more than half of all patients who visit an emergency department report having dizziness in the previous 7 days. Physicians are taught to classify dizziness into one of four categories (vertigo, presyncope, imbalance, and non-specific dizziness) on the basis of the description of symptom quality, and then...
to proceed diagnostically on the basis of the category of dizziness.\textsuperscript{15} However, recent research suggests that this approach might not be optimal.\textsuperscript{16,17} In a review of 1666 patients aged over 44 years who were seen in the emergency department for symptoms of dizziness (ie, dizziness, vertigo, or imbalance), strokes were found at the same rate among those who used the non-specific term of dizziness and in those who used the more specific term of vertigo.\textsuperscript{18} In another prospective study of 316 patients who presented consecutively at the emergency department with dizziness, 287 of 316 (91%) endorsed symptoms when requestioned minutes later.\textsuperscript{19} These same patients were far more consistent and reliable in their responses to questions about episode duration and triggers might be more useful than one that emphasises symptom type.\textsuperscript{58}

Dizziness with cerebellar infarction is occasionally short-lived\textsuperscript{20} but usually lasts for days and is accompanied by nausea (often with vomiting), gait instability, head motion intolerance, and nystagmus, known together as the acute vestibular syndrome.\textsuperscript{21} Although stroke is the most serious cause of this syndrome, benign peripheral disorders, such as vestibular neuritis (without hearing loss) or labyrinthitis (with hearing loss), are more often the cause. Distinguishing central from peripheral causes can be simple when clear neurological signs (eg, hemiplegia, dysarthria, limb ataxia) are apparent, but these symptoms are present in fewer than half of strokes identified among unselected acute vestibular presentations.\textsuperscript{22} In one study of 240 patients with cerebellar infarction, 25 presented with isolated acute vestibular syndrome without auditory symptoms that resembled vestibular neuritis;\textsuperscript{23} of those 25 patients, 24 had PICA infarcts and one had an AICA stroke. Because AICA strokes are frequently accompanied by hearing loss,\textsuperscript{24} they usually mimic labyrinthitis instead.\textsuperscript{25} Clues to the underlying stroke aetiology are most likely to be found in the severity of gait or hearing abnormalities and detailed assessment of eye movements (panel).\textsuperscript{26,27}

### Nausea and vomiting

Nausea and vomiting occur in over half of cerebellar strokes.\textsuperscript{28} In some cases, nausea and vomiting might be the predominant presenting symptoms or might be disproportionate to any associated dizziness.\textsuperscript{29} In one study, seven of 12 patients with SCA infarcts had vomiting without vertigo at onset of stroke.\textsuperscript{30} However, nausea and vomiting are also extremely common symptoms seen in general practice, and most cases are due to gastrointestinal or other medical causes. If neurological signs are not obvious, the absence of associated medical symptoms (eg, abdominal or chest pain, diarrhoea, fever) or context (eg, new medication, postoperative state) should prompt a central cause to be considered.

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### Table 2: Frequency of common published clinical findings of cerebellar infarction listed in order of descending frequency

<table>
<thead>
<tr>
<th>Signs</th>
<th>n (%)</th>
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<tbody>
<tr>
<td>Headache</td>
<td>207 of 557 (37)</td>
</tr>
<tr>
<td>Nausea or vomiting</td>
<td>205 of 557 (37)</td>
</tr>
<tr>
<td>TIA</td>
<td>204 of 557 (37)</td>
</tr>
<tr>
<td>Confusion</td>
<td>203 of 557 (37)</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>202 of 557 (37)</td>
</tr>
<tr>
<td>Confusion or somnolence</td>
<td>201 of 557 (37)</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>200 of 557 (37)</td>
</tr>
<tr>
<td>Dysarthria</td>
<td>199 of 557 (37)</td>
</tr>
<tr>
<td>Coma</td>
<td>198 of 557 (37)</td>
</tr>
</tbody>
</table>

Numbers were tabulated from several studies.\textsuperscript{2,4,6,7,19,52} Patients’ data were pooled independent of infarct areas. When data were provided in sufficient detail that enabled the distinction of patients with pure cerebellar stroke from those with brainstem association, the latter patients were excluded. Therefore, the denominators are not the same for each category. However, some of these numbers might have included some patients who had associated brainstem infarction. TIA=transient ischaemic attack.
Panel: Factors that could help physicians to select which patients with non-specific symptoms ought to undergo further investigation for cerebellar infarction

**Epidemiological context**
- Age over 50 years
- Prior history of stroke or TIA
- Stroke risk factors such as cigarette smoking, hypertension, diabetes, hyperlipidaemia, atrial fibrillation, known coronary or peripheral vascular disease
- Recent head or neck injury (including chiropractic manipulation or motor vehicle collision) or known collagen-vascular disorder, predisposing to vertebral artery dissection

**History**
- Abrupt onset of symptoms
- Nausea and vomiting in the absence of other localising symptoms (eg, diarrhoea, abdominal or chest pain, fever) or disproportionate to amount of dizziness or vertigo
- Headache (sudden, severe, or sustained), particularly with stroke risk factors, or in association with sudden hearing loss at onset (whether transient or persistent)
- Symptoms of cranial nerve dysfunction (particularly diplopia, dysarthria, dysphagia, dysphonia, or facial dysesthesia)

**Physical examination**
- Normal vestibular-ocular reflex by head impulse test (absence of a corrective saccade)
- Spontaneous nystagmus that is direction-changing or dominantly vertical or torsional
- Dizziness that persists more than 24 h, particularly with stroke risk factors, or in association with sudden hearing loss at onset (whether transient or persistent)
- Symptoms of cranial nerve dysfunction (particularly diplopia, dysarthria, dysphagia, dysphonia, or facial dysesthesia)

**Other signs and symptoms**
Dysarthria, ataxia, and nystagmus are common signs among patients with cerebellar stroke. Dysarthria, ataxia, and nystagmus are common signs among patients with cerebellar stroke. Dysarthria in patients with cerebellar infarction has a distinctive presentation, typically involving a combination of slurred speech, ataxia, and nystagmus. This combination helps to distinguish cerebellar infarction from other causes of cerebellar dysfunction.

**Differential diagnosis and misdiagnosis**
Because the differential diagnosis of dizziness, vomiting, and headache encompasses many disorders, the differential diagnosis of cerebellar infarction is vast and includes many common and benign conditions. For each of these symptoms, cerebellar infarction is a rare cause and some instances of misdiagnosis are probably inevitable. There are two primary consequences of misdiagnosis. The first is that if stroke is not considered,
no search for the underlying mechanism will ensue, leaving patients at risk for subsequent, more devastating ischaemic events that might otherwise be averted. The second is that misdiagnosis leaves the patient vulnerable to otherwise preventable complications, such as brainstem compression or hydrocephalus. Therefore, a better understanding of misdiagnosis should minimise the risk to patients with stroke.

Misdiagnosis
There are no systematic studies of misdiagnosis of cerebellar infarction, but the results of one small case series indicate that serious misdiagnosis can—and does—occur. All although limited by case selection bias, the study found that, among 15 patients with misdiagnosed cerebellar infarction, six died and five were left with permanent deficits. The patients who were misdiagnosed were mostly young (<50 years), and presented with dizziness, vomiting, or headache. Many of the patients in this study had negative CT scans, suggesting that the physician was unfamiliar with the limitations of CT to identify acute cerebellar infarction, which might have contributed to misdiagnosis. On the basis of the results of a multicentre survey of more than 400 emergency physicians, this particular misconception is common, particularly among junior physicians. The authors of these two studies have identified several potential pitfalls in the diagnosis of cerebellar infarction that span the spectrum of possible cognitive errors (table 3).

Dizziness
Peripheral vestibular and toxic–metabolic disorders are important misdiagnoses when cerebellar stroke patients present with dizziness. The overall likelihood of TIA or stroke among patients who report dizziness is low. In a retrospective study of 1666 patients who were more than 44 years old reporting a main complaint of dizziness with or without other symptoms and who presented at the emergency department or were directly admitted to the hospital, only 53 (3%) had had a stroke or TIA, and of the 1297 patients with isolated dizziness, only nine (<1%) had a stroke or TIA. Of the 46 validated cases of stroke seen in the emergency department, the emergency physician did not make the correct diagnosis in 16 cases. Patients with TIA or stroke tended to be older, more likely to be men, and to have two or more risk factors for stroke. The results of two small, prospective studies of patients with isolated, acute vestibular syndrome suggest that increasing age and presence of typical stroke risk factors conferred a particularly high likelihood of stroke. In one study of isolated vestibular presentations to the emergency department, six of 24 patients (older than 50 years) had cerebellar infarction. In a similar study, 34 of 42 patients with at least one cerebrovascular risk factor (of any age) had cerebellar or brainstem strokes.

Nausea and vomiting
Gastritis or gastroenteritis are typical misdiagnoses when cerebellar stroke patients present with nausea and vomiting. Some patients with vomiting do not have associated symptoms (ie, diarrhoea, fever, chest or abdominal pain) that suggest a systemic disease. In such cases, a CNS cause for the vomiting should be taken into consideration. Therefore, patients with vomiting should be examined for cerebellar signs that might otherwise be overlooked in a sick patient lying in bed with an apparent systemic illness. Such signs include gaze-evoked nystagmus, limb ataxia or dysmetria, and truncal or gait instability. If the patient is also dizzy, the dizziness might be falsely attributed to orthostatic hypotension from

<table>
<thead>
<tr>
<th>Type of cognitive error in diagnosis</th>
<th>Examples for missed cerebellar infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faulty hypothesis generation (diagnostic possibility not considered)</td>
<td>Failure to appreciate all cerebellar stroke presentations (eg, isolated dizziness or nausea)</td>
</tr>
<tr>
<td>Faulty context formation (over-reliance on simple clinical role of thumb)</td>
<td>Overfixation on specific historical attributes (eg, type of dizziness as a predictor of aetiology)</td>
</tr>
<tr>
<td>Faulty information gathering (failure to gather relevant clinical data)</td>
<td>Inadequate or improperly conducted neurological examination</td>
</tr>
<tr>
<td>Faulty information processing (failure to correctly interpret clinical findings)</td>
<td>Failure to do brain imaging</td>
</tr>
<tr>
<td>Faulty estimation of disease prevalence</td>
<td>Failure to do tests to define the underlying vascular cause of the stroke and stratify imminent risk</td>
</tr>
<tr>
<td>Anchoring (fixation on a prior disease label)</td>
<td>Overfixation on prior neurological or medical disorders</td>
</tr>
<tr>
<td>Faulty verification (failure to confirm suspected diagnosis)</td>
<td>Patient comes with prior diagnosis for same symptoms; failure to consider that prior diagnosis is wrong</td>
</tr>
</tbody>
</table>

*Categories from two papers. Errors based on two papers.

Table 3: Potential pitfalls in the diagnosis of cerebellar infarction by type of cognitive error
dehydration, particularly if the patient notes a postural increase in their symptoms on standing, which is sometimes seen with cerebellar strokes. In cases where the patient is unwilling or unable to stand, nausea or dizziness can sometimes be brought on by shaking the head from side to side in the bed—a finding that would be unlikely to occur with gastrointestinal disorders.

Headache
Migraine is the typical misdiagnosis when cerebellar stroke patients present with headache. To avoid misdiagnosis of migraine, physicians should consider investigating all patients with new, abrupt-onset, persistent, or unusual headaches, particularly if located posteriorly or in the neck. Although migraine is a common cause of episodic dizziness and headache, transient dizziness with persistent headache should always prompt consideration of cerebellar TIA or stroke. Migraine headaches do not generally last longer than 3 days; therefore, a longer duration should prompt concern for vertebral artery dissection or another cause.

Elements of the neurological examination
A risk factor-oriented approach often misses infarction as a cause for symptoms in younger patients (<50 years of age), and misdiagnosis can have tragic consequences, including death and permanent disability. Bedside techniques can help to identify patients with cerebrovascular causes. Those patients with lateralising or otherwise obvious neurological signs are easily identified, but this group might be the minority. Oculomotor assessment (including visual smooth pursuit, vertical ocular alignment, nystagmus direction, and vestibulo–ocular reflex [VOR] responses) can potentially achieve 92% sensitivity and specificity in predicting central versus peripheral causes.

Although no single bedside test can confidently distinguish all strokes from vestibular neuritis, the single best predictor is probably the head impulse (or head thrust) manoeuvre (figure 3 and webmovie). A test of VOR function that can be done by non-experts at the bedside. Patients with cerebellar infarction typically have a normal test result, whereas the test result will generally be abnormal in patients with acute vestibular neuritis or labyrinthitis. A normal test is a strong predictor of a final stroke diagnosis, even when contradicted by an initially—and falsely—negative diffusion-weighted imaging (DWI) MRI. Care should be taken, however, when interpreting an abnormal test because, although a peripheral vestibular lesion is generally identified, exceptions do occur. In cases of suspected vestibular neuritis, the oculomotor triad of abnormal head impulse, direction-fixed nystagmus, and absent skew deviation (vertical ocular misalignment) excludes 91% of acute strokes and identifies 78% of peripheral vestibular lesions (C Cnyrim, University of Munich, personal communication). This bedside triad therefore has comparable sensitivity to MRI for identifying stroke in the first 24 h.

If hemiplegia, altered mental status, or other obvious clinical findings are present, the problem is likely to be neurological and will require urgent brain imaging. In cases in which the presenting symptoms or comorbid findings are more ambiguous, a review of systems should focus on ascertaining the presence of diplopia, dysarthria, dysphagia, dysphonia, or facial dysesthesia. Although a thorough neurological examination is generally indicated, special emphasis should be placed on assessment of limb coordination, trunk and gait stability, and cranial nerve function with primary focus on ocular motility.

Diagnosis of cerebellar infarction can be difficult. The first step in making the diagnosis is to consider cerebellar infarction as a possibility. Clinicians cannot extensively assess every patient with headache, dizziness, gait instability, or vomiting for cerebellar stroke, and they must develop some strategy to decide which patients to test. We believe that a combination of risk factor stratification and features of the history and physical examination can help physicians to decide which patients require further testing and which do not (panel), although this strategy has not been prospectively tested as yet.

Diagnosing and defining the vascular lesion
Brain imaging
The most commonly used emergent brain imaging test for stroke is CT, which is widely available, acquires images quickly, and accurately excludes acute haemorrhage. Unfortunately, CT is usually negative in the first hours after acute ischaemic stroke. Because of artifacts that are caused by the bone of the skull base, CT has even lower sensitivity in the posterior fossa. Clinicians without access to MRI must understand this intrinsic limitation of CT.

MRI is the preferred test and is far more sensitive than CT in diagnosis of acute ischaemic stroke, with study results indicating about 80% to 95% sensitivity in the first 24 h when DWI is used. Because DWI MRI is more sensitive than conventional MRI, the clinician should communicate any clinical concern for stroke to the radiologist so that DWI sequences are acquired. However even DWI MRI can be falsely negative in some cases of acute ischaemic stroke. Similar to CT, MRI might function less well with posterior fossa lesions than with those in the anterior circulation. As always, the results of brain imaging must be interpreted in the clinical context of each patient (figures 4 and 5).

Cerebrovascular imaging
When cerebellar infarction is diagnosed, the definition of the underlying vascular pathology is the next step. The four most commonly used methods for cerebrovascular imaging are Doppler ultrasound, CT angiography (CTA), MRI angiography (MRA), and conventional catheter angiograms. Each technique has advantages and disadvantages. The advantages of Doppler ultrasound and transcranial Doppler include their rapid speed, portability, and low cost; however, as with any ultrasound
examination, these techniques are operator dependent. Furthermore, the vascular flow in the intracranial posterior circulation is less well imaged than in the anterior circulation. A new method called power motion-mode Doppler accurately identified vascular pathology in the posterior circulation (compared with CTA or MRA); however, its sensitivity was only 73%. In some cases, power motion-mode Doppler showed results that were complementary to those obtained with CTA or MRA; therefore, this new technology deserves further study.

Some form of angiography (catheter, CTA, or MRA) is commonly used for diagnosis. CTA was better than colour Doppler ultrasound in one study of patients with vertebral artery pathology. Despite CTA being widely available and extremely quick to do, a potentially toxic dye load and ionising radiation is required. Although non-contrast MRA avoids the problems of the toxic dye and ionising radiation, this technique is far less available to physicians and more time is required to acquire images compared with CT. Furthermore, CTA might be more sensitive than MRA in the posterior circulation. Catheter angiography is the gold standard but this technique has serious limitations, including all the disadvantages of CTA, in addition to the need for highly trained personnel. Furthermore, this method cannot obtain structural images of the brain simultaneously with the vascular images, as CTA and MRA can. With improvements in hardware and software, there has been a considerable shift away from conventional catheter angiography towards non-invasive CT and MRI studies.

Other testing
As with other ischaemic strokes, tests such as electrocardiography and some form of echocardiography (either transthoracic or transoesophageal) are often useful to identify a cardioembolic source. Blood tests to identify causes of thrombophilia and vasculitis can be done in some patients, and other tests such as serum lipids might help with risk stratification for secondary stroke prevention.

Course, treatment, and complications
General therapy and blood pressure control
There are no data from randomised trials that are specific for patients with cerebellar infarction; therefore, treatment guidelines are derived from information on acute ischaemic stroke in general. The first priorities are the standard airway, breathing, and circulation. Hypoxic patients should receive supplemental oxygen. Patients who present with altered mental status and who have lost protective airway reflexes might need endotracheal intubation, not only to prevent aspiration but also to prevent secondary brain injury from hypoxia or hypercapnoea. Abnormal vital signs should be investigated and clinically managed based on their potential effects on brain ischaemia. For example, sources of fever (eg, endocarditis or aspiration pneumonia) should be determined, and treatment with antipyretics started. Furthermore, haemodynamics and oxygen saturation should be closely monitored.

As with anterior circulation stroke, current American Heart Association (AHA) guidelines suggest different threshold pressures to treat arterial hypertension depending on whether or not thrombolytic therapy will be given. In general, patients with blood pressures that are persistently higher than 185 mm Hg systolic or 110 mm Hg diastolic should not be treated with thrombolytic drugs. Any patient whose blood pressure is greater than 220 mm Hg systolic or 120 mm Hg diastolic...
should be treated with antihypertensive drugs. However, as with any given patient, it is important to try to understand the individual haemodynamic profile. In the occasional patient who presents with fluctuating symptoms in the face of labile or low blood pressure, pharmacologically induced hypertension to augment cerebral blood flow should be considered.

The natural history of cerebellar infarction, against which any treatment effects must be measured, ranges from complete recovery to death. From the pooled data from six series of consecutive, unselected patients, the mortality rate was 7% (38 of 546). In the largest series, 69% of patients (194 of 282) were classified as independent by the investigators at 3 months. Patients who presented with isolated vertigo or vertigo with headache, vomiting, and ataxia, but without other neurological symptoms or signs, have better long-term outcomes. Patients who have had stroke have better outcomes when treated in specialist stroke centres: a benefit that would presumably apply to the subset of stroke patients with cerebellar infarction.

**Specific treatments**

Some patients with cerebellar infarction can be treated with intravenous alteplase, provided that standard guidelines are followed. Local institutional protocols can exclude some of these patients owing to low National Institutes of Health stroke scores. Some authors have
reported their experience of treating patients with posterior circulation strokes with intravenous101,102 or intraarterial119 alteplase, but most of these patients had obvious brainstem involvement seen on their initial examination.

Although experienced vascular surgeons can reconstruct vertebral artery stenosis with good technical results,109,110 this technique is not a common procedure, and endovascular alternatives are being increasingly sought. These interventions, including angioplasty, stenting, and stent-assisted coiling, have been used in patients with vertebral artery dissections,106–108 and extracranial109,111 and intracranial110,114,115 vertebral artery atherosclerosis, sometimes in conjunction with intra-arterial thrombolysis.116 Most of these patients had vertebrobasilar ischaemia that affected more than the cerebellum area, and all of these studies were non-randomised trials. Nonetheless, this is an area of active investigation that might lead to proven treatment options in the future.

Complications
Whatever initial treatment is selected, subsequent oedema from the initial infarction can resemble a mass lesion in the posterior fossa, leading to brainstem compression and obstructive hydrocephalus. This process, which occurs in about 10–20% of patients, peaks on the third day after the infarction, although it can occur any time within the first week.117–120 Gaze palsy and a progressive decline in level of consciousness are common clinical manifestations,118,121,122 although only half of patients who develop radiographical evidence of mass effect deteriorate clinically.119 CT scans can show displacement of the fourth ventricle, obstructive hydrocephalus, and obliteration of the basal cisterns,118,122,123 but the initial CT scan in patients who go on to develop mass effect is normal in 25% of cases.118 Haemorrhagic transformation increases the likelihood of mass effect.119 When there is clinical deterioration, such deterioration usually occurs over the following 24 h,124 and seems to be independent of the affected vascular area.120,125 Of the patients who progress to coma, 85% die without surgical intervention.126

In the absence of randomised trials, the type and timing of surgical treatments have been debated in the literature.118,119,121,122,124–127 Procedures include external ventricular drainage as a first measure, suboccipital craniectomy with debridement of the infarcted tissue, or some staged or concurrent combination of the two. These decisions are often based on clinical algorithms that take into account the level of consciousness of the patient, as well as other clinical and brain imaging findings.22,53,114,115 Theoretically, ventricular drainage in the presence of posterior fossa oedema could lead to upward transtentorial herniation; however, this event seems to be uncommon.122 About half of the patients who progress to coma and who are treated with craniectomy have good outcomes (modified Rankin scale score ≤2).122,123 Elevation of the head of the bed by 30° can improve venous drainage. Corticosteroids are ineffective and the effects of hyperventilation or osmotic diuretics are transient.129 These measures should not delay a surgical approach when otherwise indicated; however, these actions might be the only ones that are available in hospitals that do not have immediate access to a neurosurgeon.

Monitoring
Wherever the patient is admitted, close clinical monitoring for signs of deterioration is crucial. If deterioration occurs, primary brainstem ischaemia or infarction from the original vascular lesion must be distinguished from secondary brainstem compression or hydrocephalus, or both, because the treatments for each differ. MRI helps to make this distinction. Therefore, patients with acute cerebellar infarction should ideally be managed in a stroke centre with a neurological intensive care unit where close clinical monitoring, rapid access to brain imaging, and on-call neurosurgical expertise are always readily available.129

Prevention
Patients with completed stroke ought to be admitted, but patients with posterior circulation TIA might also benefit from admission for rapid assessment and observation. Although the number of patients with symptoms attributable to the posterior circulation were not reported, two studies have shown that rapid diagnostic assessment in patients with TIA reduced the incidence of stroke.128,129
Because the risk factors for cerebellar infarction are the same as those for ischaemic stroke in the anterior circulation, the methods of primary prevention are identical. AHA guidelines emphasise treating risk factors that can be modified, such as hyperlipidaemia, diabetes, hypertension, atrial fibrillation, and others.130 Secondary stroke prevention with antiplatelet agents, statins, and other treatments are outlined in the AHA prevention guidelines.131,132 Recognising posterior circulation TIA, and then defining and treating the underlying vascular lesion(s) is another important method of secondary stroke prevention.

Conclusions
Cerebellar infarction can be difficult to diagnose because the dominant clinical manifestations are common symptoms that do not necessarily raise concern for stroke. Clinicians must develop improved strategies to identify cerebellar infarction. One approach is simply better recognition that younger patients can develop cerebellar stroke, often as a result of vertebral artery dissection. Another potential strategy is to develop clinical algorithms for the approach to dizziness that focus more on timing and triggers rather than the type of dizziness (ie, vertigo). Further research is needed to help clinicians decide which patients with dizziness, headache, and vomiting need brain imaging to diagnose cerebellar infarction. Clinicians must also better understand the limitations of CT scanning for identifying ischaemic cerebellar stroke. Because of
these limitations, physicians should choose MRI scanning when this option is available. Finally, early identification of which patients are at high risk for subsequent deterioration might allow better resource allocation in settings where cost containment is a priority.

References


