Brief review in cerebellar stroke

Diagnosis and Management of Acute Cerebellar Infarction

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Outline

- Introduction
- General anatomy
- Function
- Clinical manifestation
- Neurological examination
- Management
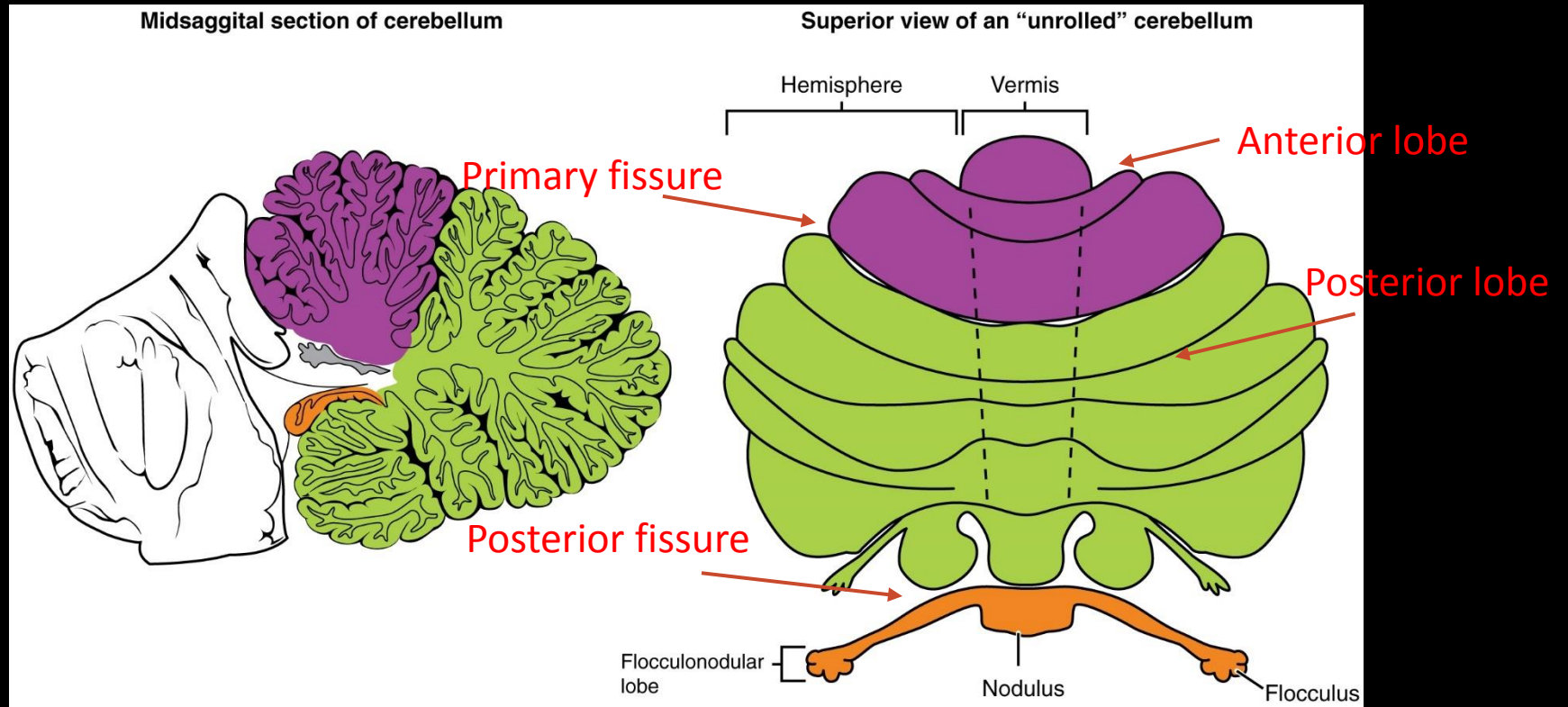- Take-home points

Introduction

- This paucity of data might be partly because the clinical presentation of cerebellar infarction is diverse

- The main symptoms were dizziness, nausea and vomiting, gait instability, headache

- Coordination, gait, and eye movements help to identify cerebellar stroke

- Early edema from infarction in the posterior fossa can result in potentially deaths

Epidemiology

- Ischemic > hemorrhagic

- In nine studies of consecutive ischemic strokes, cerebellar infarction accounted for almost 3% (660 of 23 426) of stroke

- The average age of patients is about 65 years

- Two-thirds of patients are men

- However, they may still be underdiagnosed by the CT scan cause of some limitations

Macdonell RA, Kalnins RM, Donnan GA. Cerebellar infarction: natural history, prognosis, and pathology. *Stroke* 1987; **18:** 849–55

General anatomy

- The largest structure in the posterior fossa

- Consists of a midline **vermis** and two large **cerebellar hemispheres**

- **Primary fissure** separates the cerebellum into an **anterior lobe** and a **posterior lobe**

- **Posterolateral fissure** separates the posterior lobe from the **flocculonodular** lobe, a region with important connections to the vestibular nuclei

- Gyri, the small ridges that run from medial to lateral on the surface of the cerebellum are called **folia**

Midsaggital section of cerebellum

Superior view of an "unrolled" cerebellum

Hemisphere Vermis

Primary fissure

Anterior lobe

Posterior lobe

Posterior fissure

Flocculonodular lobe

Nodulus

Flocculus

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Cerebellar stroke syndromes P.540

Function

- Three functional regions, from medial to lateral, based on their input and output connections

- The superior parts of the cerebellum are primarily concerned with limb (by lateral hemispheres)

- Trunk (midline vermis) movements and motor control of Speech articulation (by paravermal area)

- Inferior areas are primarily associated with oculomotor control and vestibular adaptation
- Appendicular motor deficits related to unilateral cerebellar lesions tend to be ipsilesional

TABLE 15.1 Functional Regions of the Cerebellum

REGION	FUNCTIONS	MOTOR PATHWAYS INFLUENCED
Lateral hemispheres	Motor planning for extremities	Lateral corticospinal tract
Intermediate hemispheres	Distal limb coordination	Lateral corticospinal tract, rubrospinal tract
Vermis and flocculonodular lobe	Proximal limb and trunk coordination	Anterior corticospinal tract, reticulospinal tract, vestibulospinal tract, tectospinal tract
	Balance and vestibulo-ocular reflexes	Medial longitudinal fasciculus
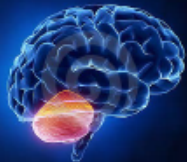
Cerebellar specific function

Table 18.2 • Subdivisions of the Cerebellum by Specific Function

Subdivision	Topography	Function	Clinical Manifestation
Motor	Anterior lobe Posterior lobe	Gait, posture Multijoint movement	Ataxia Dysarthria Limb dysmetria
Oculomotor	Flocculus, paraflocculus, and nodulus Dorsal vermis and fastigial nucleus	Control of vestibular ocular reflex and smooth pursuit Control of saccades	Impaired pursuit Gaze-evoked nystagmus Saccadic dysmetria Opsoclonus
Cognitive	Posterior lobe Dentate	Executive Visuospatial Language Memory	Impaired executive function, visuospatial disorientation, verbal fluency, and visual memory
Affective (limbic)	Posterior lobe (vermal) Fastigial nucleus	Emotion, affect Autonomic	Blunted or disinhibited behavior

Countersy of Dr.Pornsawan/Mintra
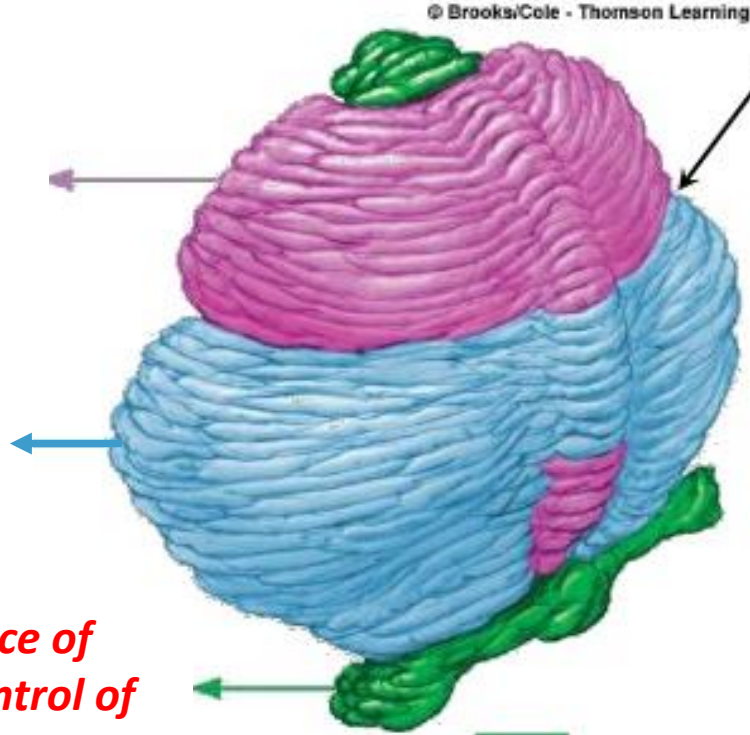
Blood supply

- Blood supply from three paired arteries
    - The posterior inferior cerebellar artery (PICA)
    - The anterior inferior cerebellar artery (AICA)
    - The superior cerebellar artery (SCA)
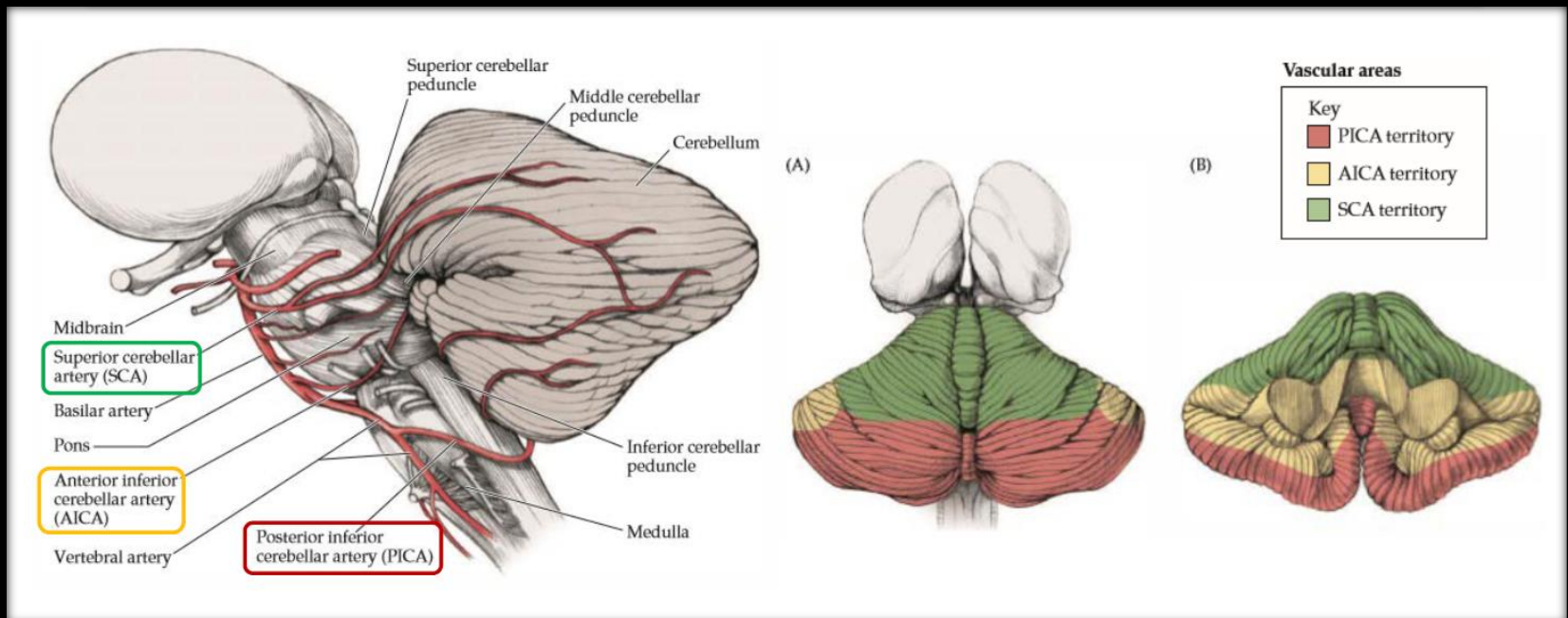
- The cerebellum and brainstem locate within the tightly constrained posterior fossa

- It bounded above by a rigid dural reflection brainstem

- Co-incident brainstem signs are common in patients with cerebellar stroke

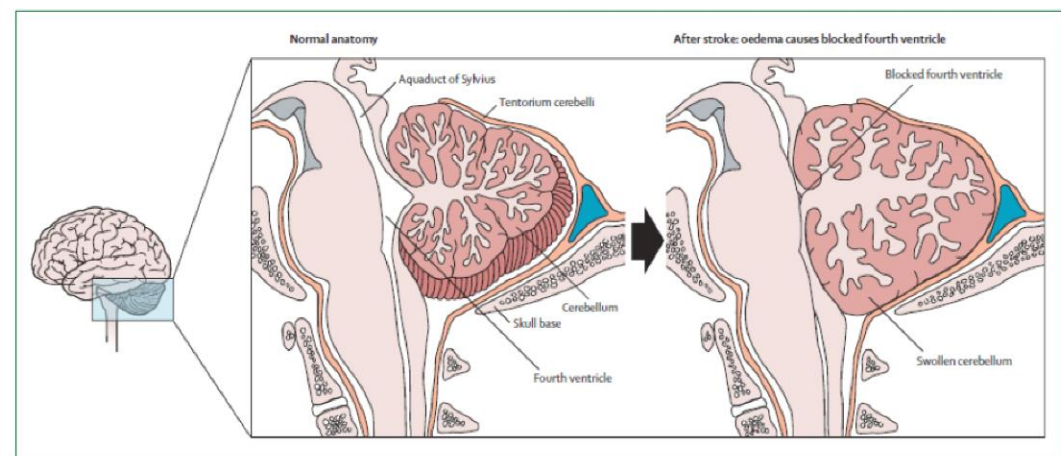- Variations of normal vascular anatomy are common
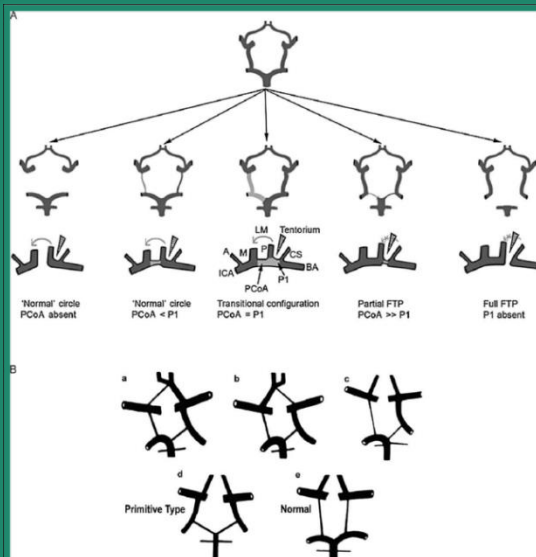
Figure 1: Anatomy of the posterior fossa: normal appearance compared with appearance after a stroke

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.

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

Data from [2-4,16,17]. *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 thermanaesthesia (loss of temperature sensitivity) typically cluster together.

Table 1: Typical arterial supply of the cerebellum and associated clinical vascular syndromes

Pathogenesis

- Two most common causes of cerebellar infarction are cardioembolism and large vessel atherosclerosis

- Who were aged less than 40 years and had cerebellar stroke, patent foramen ovale is an important consideration

- Vertebral artery dissection is another important cause

 of cerebellar infarction, particularly in younger patients

Barinagarrementeria F, Amaya LE, Cantu C. Causes and mechanisms of cerebellar infarction in young patients. *Stroke* 1997;
28: 2400–04.

- Don't forget to recognised major or minor head or neck trauma, including chiropractic manipulations

- "Beauty parlour stroke[1]" due to prolonged neck hyperextension

- Less common disorders include hypercoagulable states, vasculitis ,CVST ,acute marijuana or cocaine

- Overall, PICA strokes are more common than SCA , and AICA strokes are the least common

1. Weintraub MI. Beauty parlor stroke syndrome: report of five cases. *JAMA* 1993; **269:** 2085–86.

Clinical manifestations

- Clinical presentations of isolated cerebellar infarction are similar the three main cerebellar vascular areas

- Neurological signs might be absent, subtle, or difficult to distinguish from benign disorders of the

  peripheral vestibular system

- Patients typically only experience non-specific symptoms ie. dizziness, nausea, vomiting, unsteady gait, and headache

Clinical manifestations

- Ataxia
- Tremor
- Headache
- Dysmetria
- Dysdiadokokinesia
- Dysarthria/scanning speech
- Nystagmus
- Hypotonia
- Vertigo and unsteadiness

Clinical finding	n (%)	Comments
Prior event		
History of posterior circulation TIA	65 of 295 (22)	TIA suggests the need for rapid work-up and treatment, as with anterior circulation TIA
Symptoms		
Dizziness or vertigo	404 of 557 (73)	Whether the patient specifically describes vertigo rather than dizziness does not alter the likelihood of stroke
Nausea or vomiting	298 of 557 (54)	Nausea or vomiting can occur without dizziness and can sometimes be posturally provoked
Gait disturbance	186 of 389 (48)	The inability to walk independently suggests a central rather than a peripheral cause
Headache	207 of 557 (37)	Location and quality of headache are not diagnostic; abrupt onset can mimic haemorrhage; head or neck pain in younger patients should prompt consideration of vertebral dissection
Slurred speech	122 of 417 (29)	Slurred speech is more commonly the result of anterior circulation strokes than posterior circulation strokes; as a symptom, slurred speech must be distinguished from partial aphasia
Signs		
Limb ataxia	298 of 513 (58)	Limb ataxia (clumsy, wavering, dyssynergic movements) and dysmetria (pastpointing or mismeasured reaching) cluster together clinically and are coded together
Truncal ataxia	263 of 513 (51)	Truncal ataxia is typically assessed with the patient seated at the bedside (or in an armless chair) and arms folded
Dysarthria	204 of 447 (46)	Anterior circulation strokes might be more likely to produce labial (facial) dysarthria, and posterior circulation strokes more likely to produce lingual and guttural dysarthria; excludes a peripheral cause in a patient with dizziness
Nystagmus	226 of 513 (44)	Nystagmus that is direction-changing or vertical strongly suggests a central rather than a peripheral cause
Confusion or somnolence	116 of 447 (26)	Altered mental status is more common in strokes of the superior cerebellar artery, perhaps because of its association with top-of-the-basilar ischaemia that extends to the paramedian thalamus and medial temporal lobes
Coma	14 of 447 (3)	Frank coma typically suggests either mid-basilar occlusion or the onset of secondary complications (direct brainstem compression or obstructive hydrocephalus with herniation)

Numbers were tabulated from several studies.[2,4,6,7,11,19,52] 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.

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

Neurological examination

- Ataxia
  - Limb : cerebellar hemisphere lesion
  - Truncal : cerebellar vermis lesion
  - Gait : wide base gait

- Ocular symptom
  - Nystagmus, broken pursuit, hypometric saccade, ocular dysmetria

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Neurological examination

- Tremor
  - Kinetic with exacerbation at the end of the movement
- Rebound phenomenon : patient puts arm out and examiner pushes down onto wrist , observe on swaying
- Titubation : Head and body tremor (midline zone)
- Dysarthria : Scanning speech
- Hypotonia
- Normo,hyporeflexia

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Head impulse test

- Test of VOR function that can be done by non-experts at the bedside

- To distinguish with acute vestibular neuritis or labyrinthitis.

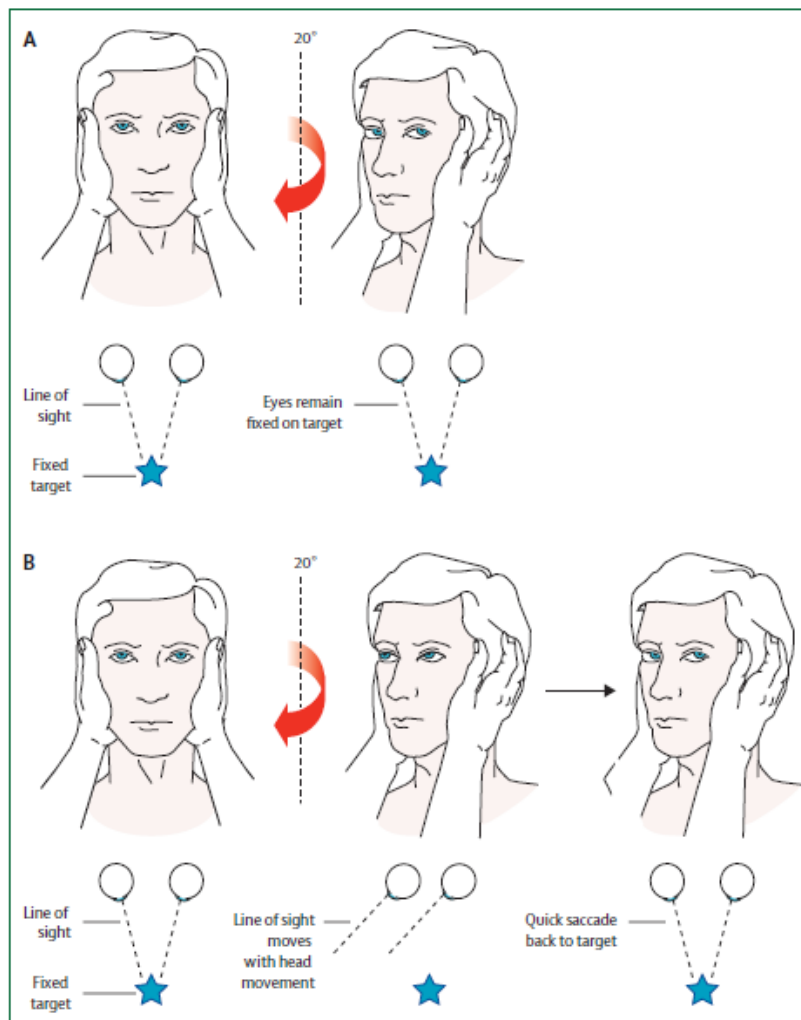- Patients with cerebellar infarction typically have a normal test.

Figure 3: The head impulse test

The head impulse, or head thrust, manoeuvre is a test of vestibular function that can be easily done during bedside examination. This manoeuvre tests the vestibulo–ocular reflex (VOR), and can help to distinguish a peripheral process (vestibular neuritis) from a central one (cerebellar stroke). With the patient sitting on the stretcher, the physician instructs him to maintain his gaze on the nose of the examiner. The physician holds the patient's head steady in the midline axis and then rapidly turns the head to about 20° off the midline. (A) The normal response (intact VOR), is for the eyes to stay locked on the examiner's nose. (B) An abnormal response (impaired VOR) is for the eyes to move with the head, and then to snap back in one corrective saccade to the examiner's nose. The test is usually "positive" (ie, corrective saccade is visible) with peripheral lesions (vestibular neuritis), and the test is usually normal in cerebellar stroke. This occurs because the primary VOR pathway bypasses the cerebellum.

Clinical hints to Ix for cerebellar stroke

- **Epidemiological context**
  - Age over 50 years
  - Prior history of stroke or TIA
  - Stroke risk factors (smoking, HT,DM,DLP,AF 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
  - N/V 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 other neurological symptoms (especially motor complaints such as limb weakness or abnormal speech)
  - 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 dysaesthesia)
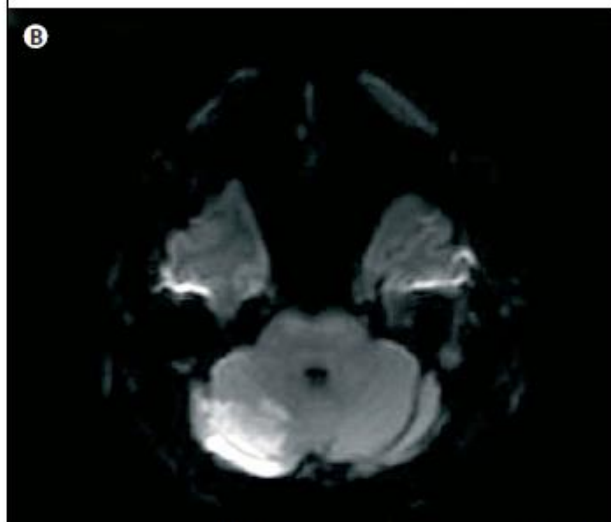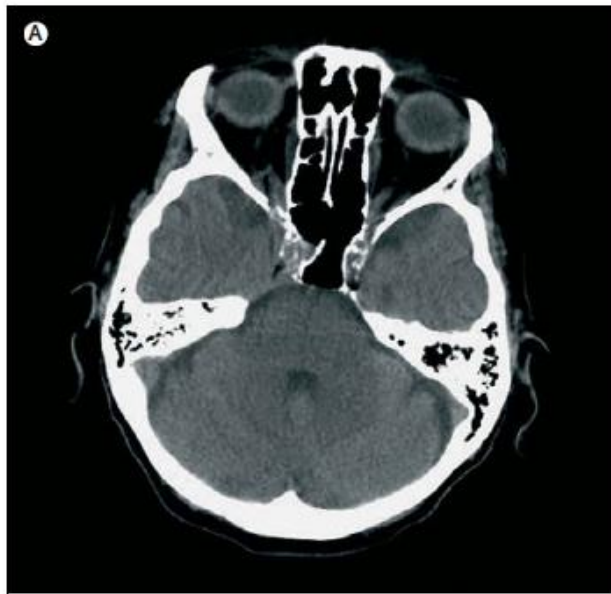
Figure 4: False negative brain CT in a patient with inferior cerebellar stroke
(A) Brain CT scan of the posterior fossa of a 77-year-old woman who presented to the emergency department with nausea, vomiting, and severe gait instability. This scan is normal with no indication of infarction. (B) Within about 1 h, the MRI was obtained. This diffusion-weighted image, taken at the same level, shows an infarction of the right posterior inferior cerebellum. Reproduced from Savitz SI et al,[18] with permission from Wiley-Blackwell.
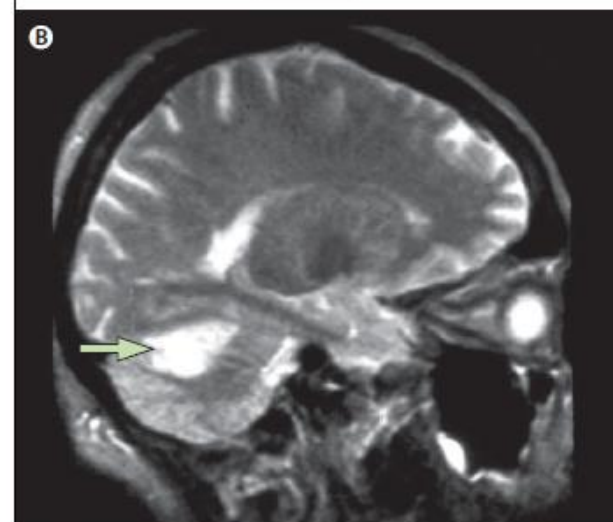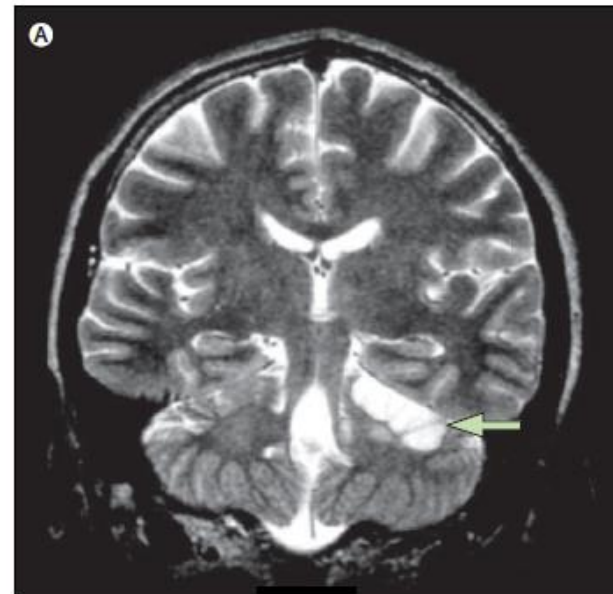

Figure 5: Infarction restricted to the superior cerebellar artery territory
Patients with infarction in the superior cerebellar artery territory typically present with the sudden onset of gait and truncal instability in association with dysarthria and limb ataxia or dysmetria. Dizziness, although frequently present, is typically a milder (non-vertiginous) sensation than the dizziness experienced by patients with infarcts of the inferior cerebellum. (A) T2 coronal MRI and (B) T2 sagittal MRI demonstrating superior cerebellar infarction. Arrows indicate the location of the infarction.

Clinical hints to Ix for cerebellar stroke

- ## 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

  - Severe difficulty or inability to stand or walk

  - Any other abnormal neurological finding, particularly cranial nerve dysfunction, Horner's syndrome, or long tract signs (hemimotor, hemisensory, limb ataxia, or dysmetria)

Diagnosing and defining the vascular lesion

Brain imaging

- MRI is the preferred test , 80% -95% sensitivity in the first 24 h with DWI

- For CT scan, widely available, acquires images quickly, and accurately excludes acute haemorrhage

- CT has lower sensitivity in the posterior fossa ; the bone of the skull base artifacts (sensitivity 40-50%)

General management

- Similar to infarcts of other areas of the brain

- Close neurological monitoring , clinically significant cerebellar edema typically occurs within 1 to 7 days, with a mean peak of 3 days

- Osmotic diuretics such as mannitol or hypertonic saline may be useful.

- Surgical treatment is the management of choice

- **Decompressive Surgery**
  - suboccipital craniectomy ; preventing and treating herniation and brain stem compression
- **External Ventricular Drainage**
  - in cerebellar infarction is still widely debated
  - In acute hydrocephalus, where transition to the operating room may be delayed
  - EVD placement will be rapid reduction in intracranial pressure may decrease mortality and prove lifesaving

Cerebellar strokes: a clinical outcome review of 79 cases

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- ***RESULTS*** A total of 79 patients with cerebellar stroke.

- 17.7% died and 31.6% had poor outcomes at six months after discharge.

- Patients with cerebellar haemorrhage have poor outcomes as compared to patients with cerebellar infarct(odds ratio [OR] 4.3, 95% CI (1.3–14.1)

Table II. Treatment and mean size of lesion of patients admitted for cerebellar strokes (n = 79).

Parameter	Cerebellar infarct (n = 43)	Cerebellar haemorrhage (n = 36)	All (n = 79)	p-value*
Treatment†				
Conservative management	35 (81.4)	24 (66.7)	59 (74.7)	0.32
Ventriculostomy	1 (2.3)	2 (5.6)	3 (3.8)	
Suboccipital decompression	7 (16.3)	10 (27.8)	17 (21.5)	
Size of lesion† (cm³)				
Patients conservatively managed	13.6 ± 12.9	14.6 ± 19.6	14.0 ± 15.8	0.81
Patients who underwent ventriculostomy	9.0 ± 0.0	10.3 ± 8.1	9.9 ± 5.7	0.92
Patients who underwent suboccipital decompresssion	34.5 ± 23.2	30.1 ± 15.9	31.9 ± 18.7	0.64

*95% confidence interval with unpaired student t-test. †Data presented as no. of patients (%). ‡Data presented as mean ± standard deviation.

- Six months after discharge (OR 5.2, 95% CI 1.6–17.2). When compared to small lesions (< 5 cm3), lesions > 20 cm3 were significantly associated with poorer outcomes and the development of hydrocephalus and brainstem compression

- A cerebellar stroke with a lesion is 3 cm in diameter on CT would give rise to a lesion with an estimated volume of 15 cm^3.

- These finding more likely to have a poor outcome

Table III. Comparison of the outcomes of patients with cerebellar infarct and patients with cerebellar haemorrhage, at discharge and at follow-up six months after discharge.

Outcome	No. of patients (%)		χ^2 (df)	p-value*	OR (95% CI)	p-value†
	Cerebellar infarct	Cerebellar haemorrhage				
Based on GOS score						
At discharge			6.15 (1)	0.013	4.3 (1.3–14.1)	0.017
Poor outcome	11 (25.6)	19 (52.8)				
Good outcome	32 (74.4)	17 (47.2)				
At follow-up†			8.67 (1)	0.003	5.2 (1.6–17.2)	0.007
Poor outcome	7 (17.1)	17 (48.6)				
Good outcome	34 (82.9)	18 (51.4)				
Based on mRS score						
At discharge			7.06 (1)	0.008	4.4 (1.3–15.3)	0.019
Poor outcome	27 (62.8)	32 (88.9)				
Good outcome	16 (37.2)	4 (11.1)				
At follow-up†			11.9 (1)	0.001	5.2 (1.8–14.4)	0.002
Poor outcome	13 (31.6)	25 (71.4)				
Good outcome	28 (68.3)	10 (28.6)				

5.1. Cerebellar and Cerebral Edema

5.1. Cerebellar and Cerebral Edema	COR	LOE	New, Revised, or Unchanged
1. Ventriculostomy is recommended in the treatment of obstructive hydrocephalus after a cerebellar infarct. Concomitant or subsequent decompressive craniectomy may or may not be necessary on the basis of factors such as infarct size, neurological condition, degree of brainstem compression, and effectiveness of medical management.	I	C-LD	Recommendation revised from 2014 Cerebral Edema.
Ventriculostomy is a well-recognized effective treatment for the management of acute obstructive hydrocephalus and is often effective in isolation in relieving symptoms, even among patients with acute ischemic cerebellar stroke.[244,245] Thus, in patients who develop symptoms of obstructive hydrocephalus from a cerebellar stroke, emergency ventriculostomy is a reasonable first step in the surgical management paradigm. If cerebrospinal			See Table LIX in online Data Supplement 1.

5.1. Cerebellar and Cerebral Edema (Continued)	COR	LOE	New, Revised, or Unchanged
2. Decompressive suboccipital craniectomy with dural expansion should be performed in patients with cerebellar infarction causing neurological deterioration from brainstem compression despite maximal medical therapy. When deemed safe and indicated, obstructive hydrocephalus should be treated concurrently with ventriculostomy.	I	B-NR	Recommendation revised from 2014 Cerebral Edema.
The data support decompressive cerebellar craniectomy for the management of acute ischemic cerebellar stroke with mass effect.[244–246] This surgery is indicated as a therapeutic intervention in cases of neurological deterioration caused by cerebral edema as a result of cerebellar infarction that cannot be otherwise managed with medical therapy or ventriculostomy in the setting of obstructive hydrocephalus.[244,245]			See Table LIX in online Data Supplement 1.
3. When considering decompressive suboccipital craniectomy for cerebellar infarction, it may be reasonable to inform family members that the outcome after cerebellar infarct can be good after sub-occipital craniectomy.	IIb	C-LD	Recommendation and Class unchanged from 2014 Cerebral Edema. Wording revised and LOE amended to conform with ACC/AHA 2015 Recommendation Classification System.
4. Patients with large territorial supratentorial infarctions are at high risk for complicating brain edema and increased intracranial pressure. Discussion of care options and possible outcomes should take place quickly with patients (if possible) and caregivers. Medical professionals and caregivers should ascertain and include patient-centered preferences in shared decision making, especially during prognosis formation and considering interventions or limitations in care.	I	C-EO	New recommendation.
Cerebral edema can cause serious and even life-threatening complications in patients with large territorial supratentorial infarctions. Although less severe edema can be managed medically, surgical treatment may be the only effective option for very severe cases; in such instances, timely decompressive surgery has been shown to reduce mortality.[247] Nevertheless, there is evidence that persistent morbidity is common and individual preexisting decisions about end-of-life and degree of treatment performed in the face of severe neurological injury must be considered.			

5.1. Cerebellar and Cerebral Edema (Continued)	COR	LOE	New, Revised, or Unchanged
8. Although the optimal trigger for decompressive craniectomy is unknown, it is reasonable to use a decrease in level of consciousness attributed to brain swelling as selection criteria.	IIa	A	Recommendation, Class, and LOE unchanged from 2014 Cerebral Edema.
9. Use of osmotic therapy for patients with clinical deterioration from cerebral swelling associated with cerebral infarction is reasonable.	IIa	C-LD	Recommendation reworded for clarity from 2014 Cerebral Edema. Class unchanged. LOE amended to conform with ACC/AHA 2015 Recommendation Classification System. See Table LXXXIII in online Data Supplement 1 for original wording.
10. Use of brief moderate hyperventilation (Pco_2 target 30–34 mm Hg) is a reasonable treatment for patients with acute severe neurological decline from brain swelling as a bridge to more definitive therapy.	IIa	C-EO	New recommendation.
Hyperventilation is a very effective treatment to rapidly improve brain swelling, but it works by inducing cerebral vasoconstriction, which can worsen ischemia if the hypocapnia is sustained or profound.[256] Thus, hyperventilation should be induced rapidly but should be used as briefly as possible and avoid excessive hypocapnia (<30 mm Hg).			
11. Hypothermia or barbiturates in the setting of ischemic cerebral or cerebellar swelling are not recommended.	III: No Benefit	B-R	Recommendation and LOE revised from 2014 Cerebral Edema. COR amended to conform with ACC/AHA 2015 Recommendation Classification System.
The data on the use of hypothermia and barbiturates for the management of AIS continue to be limited. Such data include only studies with small numbers of patients and unclear timing of intervention with respect to stroke onset. Hypothermia use has recently been shown to have no impact on stroke outcomes in a meta-analysis of 6 RCTs.[257] Further research is recommended.			See Tables LIX and LX in online Data Supplement 1.
12. Because of a lack of evidence of efficacy and the potential to increase the risk of infectious complications, corticosteroids (in conventional or large doses) should not be administered for the treatment of cerebral edema and increased intracranial pressure complicating ischemic stroke.	III: Harm	A	Recommendation wording modified from 2013 AIS Guidelines to match Class III stratifications. LOE unchanged. Class amended to conform with ACC/AHA 2015 Recommendation Classification System.

Take-Home Points

- Patients with cerebellar stroke may present with generalized symptoms.
- A high index of suspicion must be maintained in patients with significant risk factors.
- Head impulse test may help for distinguish pheripheral or central lesion
- CT has low sensitivity for infarctions of the posterior fossa. If suspicion of infarction is high, MRI/diffusion weighted imaging sequences should be obtained.
- Emergent placement of an external ventricular drain or posterior fossa decompression may be lifesaving in patients with hydrocephalous or brain stem compression.

- Thank you for your kindness