

Consider MRI for inapparent cause

Table 3.2. National Institutes of Health Stroke Scale (maximum = 42)

Response	(Score)	Response	(Score)
Level of consciousness	11047	Motor arm (left and right)	
alert	(0)	no drift	(0)
drowsy	(1)	drift before 10 seconds	(1)
stuporous	(2)	falls before 10 seconds	(2)
coma	(3)	no effort against gravity	(3)
		no movement	(4)
Response to level of		Motor leg (left and right)	
consciousness questions*		no drift	(0)
answers both correctly	(0)	drift before 5-10 seconds	(1)
answers one correctly	(1)	falls before 5-10 seconds	(2)
answers neither correctly	(2)	no effort against gravity	(3)
		no movement	(4)
Response to level of	03 (4) 4	Ataxia	
consciousness commands†		absent	(0)
obeys both correctly	(0)	one limb	(1)
obeys one correctly	(1)	two limbs	(2)
obeys neither	(2)		
Pupillary response	Programme and the second	Sensory	
both reactive	(0)	normal	(0)
one reactive	(1)	mild	(1)
neither reactive	(2)	severe loss	(2)
Gaze	auto adji s	Language	
normal	(0)	normal	(0)
partial gaze palsy	(1)	mild aphasia	(1)
total gaze palsy	(2)	severe aphasia	(2)
Carlotte and Carlotte and Carlotte		mute or global aphasia	(3)
Visual fields	Contractions	Facial palsy	
no visual loss	(0)	normal	(0)
partial hemianopsia	(1)	minor paralysis	(1)
complete hemianopsia	(2)	partial paralysis	(2)
bilateral hemianopsia	(3)	complete paralysis	(3)
Dysarthria	TO STATE	Extinction/inattention	September 1
normal	(0)	normal	(0)
mild	(1)	mild	(1)
severe	(2)	severe	(2)

^{*} Level of consciousness questions: "How old are you?" "What month is this?"

[†] Level of consciousness commands: "Squeeze my hand" (using nonparetic hand), "Close your eyes."

<4 = Good prognosis -- No tPA 4-20 = mild to moderate - ideal tPA >20 = severe deficit -- No tPA

Assessment Date Symptomonset Date	Time Time		
GCS E= M= V= BP *BM *BM *If BM <3.5 mmol/L treat urgently and reassess once blood glucose normal			
Has there been loss of consciousness or syncope? Has there been seizure activity? Is there a <u>NEW ACUTE</u> onset (or on awakening from	Y (−1) □ N (0) □		
I. Asymmetric facial weakness II. Asymmetric arm weakness III. Asymmetric leg weakness IV. Speech disturbance V. Visual field defect *Total	Y (+1)		
Provisional diagnosis □Stroke □ Non-stroke (specify) *Stroke is unlikely but not completely excluded if total scores are ≤0.			

Etiology subtypes:

- Large artery atherosclerosis Cardioaortic embolism Intracranial small vessel disease
- Dissection, hypercoagulable states, inherited, unidentified

Differential:

- Hyperglycemia/Hypoglycemia
- Structural brain lesion
- Demyelinating disease (MS)
- Psychogenic

- Seizure with Todd paralysis
- CNS infection
- PNS lesion
- Delirium

- Migraine
- acute vestibular syndrome
- Metabolic
- Syncope

Acute Vestibular Syndrome vs Stroke

Tamutzer AA et al. CMAJ. 2011; 183(9): E571-592.

Acute vestibular syndrome (AVS): Acute dizziness with N/V, unsteady gait, nystagmus, intolerance to head motion, and lasts ≥24 hrs; no focal neuro signs (hemiparesis, hemisensory loss, gaze palsy)

- Most common causes: Vestibular neuritis (labyrinthitis) and vertebrobasilar CVA
- Central causes: Vertebrobasilar CVA (83%), multiple sclerosis (11%), other (6%)
- Over 50% of vertebrobasilar CVA's have no focal neuro deficit.
- Excludes benign positional vertigo and Meniere's (< 24 hrs of continuous sx)

Which bedside tests can help differentiate peripheral from central causes of AVS?

NOT helpful

- Differentiating type of dizziness (vertigo, presyncope, unsteadiness)
- Onset of dizziness (sudden vs gradual)
- Provocative head movement (eg. Hallpike-Dix)
- Proportionality of sx such as severity of dizziness, vomiting, gait impairment (eg. severe gait impairment with mild dizziness does not mean central cause)
- Hearing loss
- · Patterns and vectors of nystagmus
- Noncontrast head CT has sensitivity of only 16% for acute ischemic CVA

HELPFUL

- Multiple prodromal episodes of dizziness Predictive of central cause (CVA)
- Headache or neck pain Predictive of central cause (CVA, vertebral artery dissection) with positive LR = 3.2. Absence of pain not as predictive.
- Any neurologic signs, esp. truncal ataxia (unable sit upright with arms crossed) and severe gait instability – Strongly predictive of central cause
- Horizontal head impulse test (vestibular-ocular reflex) If normal, predictive of central cause (positive LR 18.4, negative LR 0.16)
- Gaze-evoked nystagmus (right-beating nystagmus on right gaze and left-beating nystagmus on left gaze) = dysfunction of gaze-holding structures in brainstem and cerebellum – If abnormal, predictive of central cause (specificity 92%, sens 38%)
- Vertical ocular misalignment on alternate cover test If abnormal skew deviation, predictive of central cause (specificity 98%, sens 30%)
- Diffusion-weighted MRI is good but not perfect Sensitivity 83% for ischemic CVA
- Composite HINTS (Head Impulse test, Nystagmus, Test of Skew) exam Abnormal findings summarized using INFARCTs acronym:
 - Impulse Normal Fast-phase Alternating Refixation on Cover Test
 - * If any 1 of 3 abnormal, sensitivity 100% and specificity 96% for central cause (Kattah et al, Stroke, 2009)
 - * HINTS test seems just as good as diffusion-weighted MRI to r/o CVA in AVS.



ABCD² risk factor	Value	Score
Age	≥60 years	1
Blood pressure	Systolic >140 mmHg or diastolic >90 mmHg	1
Clinical symptoms	Unilateral weakness	2
	Speech disturbance without weakness	1
Duration of symptoms	>60 minutes	2
	10–59 minutes	1
Diabetes	Oral medication or insulin	1

Appendix E and F

Indications

Diagnosis of ischemic stroke causing a measurable neurological deficit Onset of symptoms <4.5 h before initiation of treatment

CONTRAINDICATIONS

Clinical

Sustained hypertension above 180/110 mm Hg

Symptoms suggestive of subarachnoid hemorrhage

Previous history of intracranial hemorrhage

ST elevation myocardial infarction within the previous 3 months

Major head trauma or stroke within the previous 3 months

Major surgery within the previous 14 days

Gastrointestinal or urinary tract hemorrhage within the previous 21 days

Arterial puncture at a non-compressible site within the previous 7 days

Active bleeding or acute traumatic fracture on examination

Seizure at onset with suspected postictal deficits

Minor or rapidly improving neurological deficits

Radiological

Head CT showing hemorrhage or multilobar infarction (i.e., hypodensity involving >1/3 of the cerebral hemisphere

Laboratory

Oral anticoagulation with INR >1.7*

Heparin within previous 48 h with elevated current aPTT

Platelet count <100,000 per mm³

Blood glucose level <50 mg/dL (2.7 mmol/L) at presentation with improving deficits following correction of hypoglycemia

Additional contraindications for treatment between 3 and 4.5 h

Age >80 years

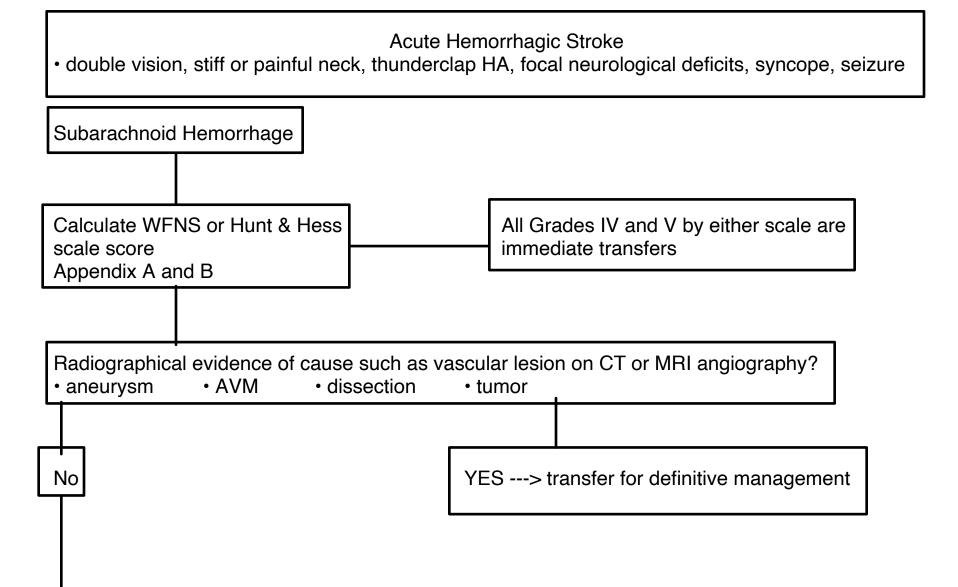
Very severe deficits at onset (NIHSS score >25)

Combination of previous stroke and diabetes mellitus

Table 13. Treatment Of Acute Ischemic Stroke: Intravenous Administration Of tPA

- Infuse 0.9 mg/kg (max dose 90 mg) over 60 min with 10% of the dose given as a bolus over 1 minute.
- Admit the patient to an intensive care or stroke unit for monitoring.
- Perform neurological assessments every 15 minutes during the infusion and every 30 minutes thereafter for the next 6 hours, then hourly until 24 hours after treatment.
- If the patient develops severe headache, acute hypertension, nausea, or vomiting, discontinue the infusion (if tPA is being administered) and obtain emergency CT scan.
- Measure blood pressure every 15 min for the first 2 hours and subsequently every 30 min for the next 6 hours, then hourly until 24 hours after treatment.
- Increase the frequency of blood pressure measurements if the systolic blood pressure is greater than 180 mm Hg or if the diastolic blood pressure is greater than 105 mm Hg; administer antihypertensive medications to maintain blood pressure at or below these levels.
- Delay placement of nasogastric tubes, indwelling bladder catheters, or intra-arterial pressure catheters.
- Obtain a follow-up CT scan at 24 hours before starting anticoagulants or antiplatelet agents.

^{*}Oral anticoagulation regardless of current INR should be considered a contraindication for treatment between 3 and 4.5 h.



Consider other causes

- HTN-induced: Look for LVH on EKG or retinal changes consistent with, protein in the urine
- Sympathomimetic drug use
- Smoking and heavy alcohol use increases risk
- Trauma, including head, neck, or spinal trauma
- Coagulopathy: anticoagulants, inherited connective tissue/bleeding disorders, herbal supplements (esp ginkgo biloba, appetite suppressants)
- Secure if cause is known and reversible
- Keep SBP < 160 or < 140 until secured.
 Nicardipine is best, may also use beta-blockers
- · Analgesia may be enough to control BP
- Consider anti-convulsants, seizure precautions for deficits out of proportion to injury
- Isotonic fluids with positive fluid balance. NS preferred over LR (less isotonic)
- Maintain strict normothermia
- Maintain normal glucose levels. Both hyper and hypoglycemia harmful
- Consider nimodipine 60 mg PO Q4H for 21 days, esp for aneurysmal Start within 96 hrs
- Reverse anticoagulation: Vit K, FFP, PCC (factor VII not useful for this)
- EKG and trop to evaluate for resulting acute cardiac injury; cardiac stunning
- At risk for arrhythmias due to neurohormonal response. Consider telemetry
- May develop Takusubu's cardiomyopathy or pulmonary edema.
 Monitor for these and echo if indicated.

Appendix A

Hunt and Hess classification of subarachnoid hemorrhage

- Grade 1: Mild headache, alert and oriented, minimal (if any) nuchal rigidity: 30% mortality
- Grade 2: Full nuchal rigidity, moderate severe headache, alert and oriented, no neurological deficit (besides CN palsy): 40% mortality
- Grade 3: Lethargy or confusion, mild focal neurological deficits: 50% mortality
- Grade 4: stuporous, more severe focal deficits: 80% mortality
- Grade 5: comatose, showing signs of severe neurological impairment i.e. posturing: 90% mortality

Appendix B

World Federation of Neurological Surgeons Grading System for Subarachnoid Hemorrhage - (WFNS) scale

Overview:

The clinical grading system proposed by the World Federation of Neurologic Surgeons is intended to be a simple, reliable and clinically valid way to grade a patient with subarachnoid hemorrhage. This system offers less interobserver variability than some of the earlier classification systems.

Glasgow Coma Score	Motor Deficit	Grade
15	absent	1
13 - 14	absent	2
13 - 14	present	3
7 - 12	present or absent	4
3 - 6	present or absent	5

^{*}Where a motor deficit refers to a major focal deficit.

Interpretation:

- · Maximum score of 15 has the best prognosis
- Minimum score of 3 has the worst prognosis
- Scores of 8 or above have a good chance for recovery
- Scores of 3-5 are potentially fatal, especially if accompanied by fixed pupils or absent oculovestibular responses
- · Young children may be nonverbal, requiring a modification of the coma scale for evaluation

In assessing outcome of subarachnoid hemorrhage, the Federation recommended use of the Glasgow Coma Scale:

Glasgow coma scale = (score for eye opening) + (score for best verbal response) + (score for best motor response)

Eye Opening	<u>Score</u>
Spontaneously	4
To verbal stimuli	3
To pain	2
Never	1

	<u>ore</u>
Oriented and converses 5 Disoriented and converses 4 Inappropriate words 3 Incomprehensible sounds 2 No response 1	

Best Motor Response	<u>Score</u>
Obeys commands	6

Localizes pain	5
Flexion withdrawl	4
Abnormal flexion (discorticate rigidity)	3
Extension (decerebrate rigidity)	2
No response	1