STROKE, T.I.A. AND CERVICAL ARTERY DISSECTION

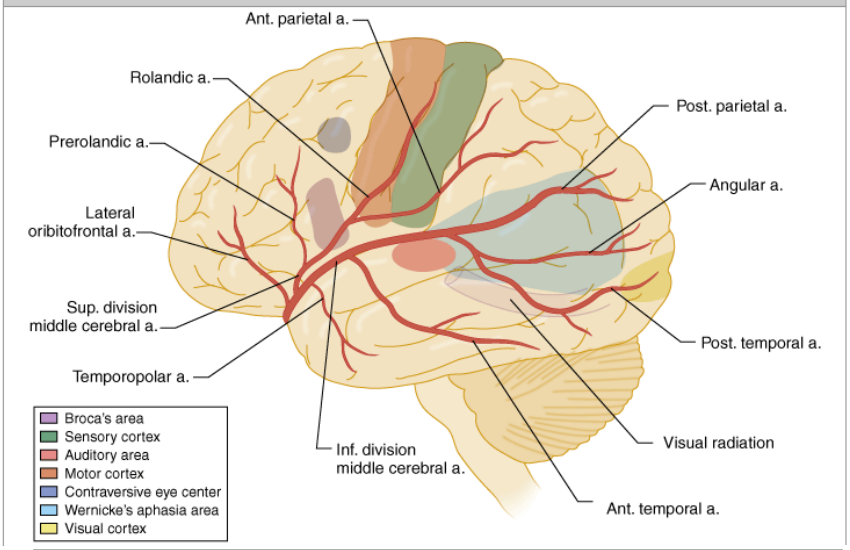
PATHOPHYSIOLOGY:

- STROKE → generally defined as any disease process that interrupts blood flow to the brain
- Injury is related to the loss of oxygen and glucose substrates necessary for high-energy phosphate production and presence of mediators of secondary cellular injury
- Subsequent factors → oedema and mass effect may exacerbate initial insult

VASCULAR SUPPLY:

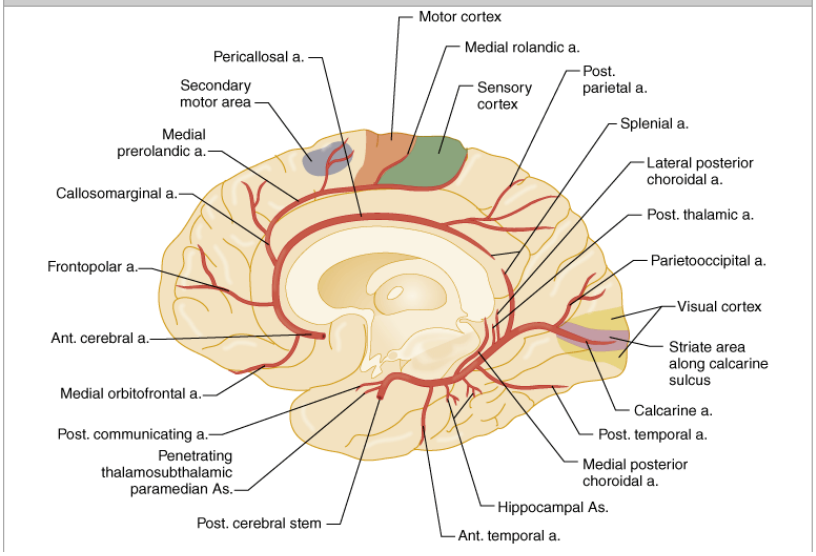
- Clinical findings in storke are determined by the location of the lesion(s), but the degree of collateral circulation may cause variations in the specific clinical symptoms

Figure 161-1.



Lateral cerebral hemisphere

Figure 161-2.



Medial aspect of cerebral hemisphere

Table 161-1 Anterior and Posterior Circulation of the Brain		
Circulation	**Major Arteries**	**Major Regions of Brain Supplied**
Anterior (internal carotid system)	Ophthalmic	Optic nerve and retina
	Anterior cerebral	Frontal pole
		Anteromedial cerebral cortex
		Anterior corpus callosum
	Middle cerebral	Frontoparietal lobe
		Anterotemporal lobe
Posterior (vertebral system)	Vertebral	Brainstem
	Posteroinferior cerebellar	Cerebellum
	Basilar	Thalamus
	Posterior cerebral	Auditory/vestibular structures
		Medial temporal lobe
		Visual occipital cortex

STROKE TYPE:

- TWO MAJOR MECHANISMS → ISCHAEMIA AND HAEMORRHAGE
- ISCHAEMIC STROKES:
  - Account for 87% → categorised as thrombotic, embolic, hypoperfusion-related
- HAEMORRHAGIC STROKES:
  - Subdivided → ICH, SAH (10% and 3% of strokes respectively)
- FINAL COMMON PATHWAY IS ALTERED NEURONAL PERFUSION AND CELL DEATH → neurons are exquisitely sensitive and may die within minutes of cessation of blood flow

Table 161-2 Stroke Classification			
Stroke Type	**Mechanism**	**Major Causes**	**Clinical Notes**
Ischemic			
Thrombotic	Narrowing of a damaged vascular lumen by an in situ process—usually clot formation	Atherosclerosis	Symptoms often have gradual onset and may wax and wane.
		Vasculitis	
		Arterial dissection	Common cause of transient ischemic attack.
		Polycythemia	
		Hypercoagulable state	
		Infection (human immunodeficiency virus infection, syphilis, trichinosis, tuberculosis, aspergillosis)	
Embolic	Obstruction of a normal vascular lumen by intravascular material from a remote source	Valvular vegetations	Typically sudden in onset.
		Mural thrombi	Account for 20% of ischemic strokes.
		Paradoxical emboli	
		Cardiac tumors (myxomas)	
		Arterial-arterial emboli from proximal source	
		Fat emboli	
		Particulate emboli (intravenous drug use)	
		Septic emboli	
Hypoperfusion	Low–blood flow state leading to hypoperfusion of the brain	Cardiac failure resulting in systemic hypotension	Diffuse injury pattern in watershed regions.
			Symptoms may wax and wane with hemodynamic factors.
Hemorrhagic			
Intracerebral	Intraparenchymal hemorrhage from previously weakened arterioles	Hypertension	Intracranial pressure rise causes local neuronal damage.
		Amyloidosis	Secondary vasoconstriction mediated by blood breakdown products or neuronal mechanisms (diaschisis) can cause remote perfusion changes.
		Iatrogenic anticoagulation	
		Vascular malformations	
		Cocaine use	Risks include advanced age, history of stroke, tobacco or alcohol use.
			More common in Asians and blacks.
Nontraumatic subarachnoid	Hemorrhage into subarachnoid space	Berry aneurysm rupture	May be preceded by a sentinel headache ("warning leak").
		Vascular malformation rupture	

PRE-HOSPITAL CONSIDERATIONS:

- **Early detection must begin with general public**
  - Awareness of risk factors and early warning signs
  - Time is a critical component in care of stroke patients → ascertain time of onset of symptoms ASAP

CLINICAL FEATURES:

- DIAGNOSIS RESTS ON FOCUSES HISTROY AND EXAM
- PRESENTATIONS RANGE FROM OBVIOUS (facial droop, arm drift, abnormal speech) to subtle → weakness, dizziness, sensory changes)
- Women report non-traditional symptoms more often!

HISTORY AND ASSOCIATED SYMPTOMS:

Table 161-4 Symptoms of Stroke	
Traditional symptoms	Sudden numbness or weakness of face, arm, or leg—especially unilateral
	Sudden confusion or aphasia
	Sudden memory deficit or spatial orientation or perception difficulties
	Sudden visual deficit or diplopia
	Sudden dizziness, gait disturbance, or ataxia
	Sudden severe headache with no known cause
Nontraditional symptoms	Loss of consciousness or syncope
	Shortness of breath
	Sudden pain in the face, chest, arms, or legs
	Seizure
	Falls or accidents
	Sudden hiccups
	Sudden nausea
	Sudden fatigue
	Sudden palpitations
	Altered mental status

- Onset suggests aetiology → sudden onset for embolic or haemorrhagic stroke, whereas a stuttering or waxing/waning course more suggestive of thrombotic or hypoperfusion-related stroke
- History of recent neck trauma or manipulation suggests cervical artery dissection
- RF for vessel thrombosis → HT, DM, IHD
- RF for embolism → AF, recent AMI, valvular disease or replacement
- ACCURATE DETERMINATION OF TIME OF ONSET OF THE PATIENT'S SYMPTOMS IS ESSENTIAL → for the purposes of thrombolysis in stroke, if the person AWAKES with the symptoms, the time of onset is considered to be the last known time when the patient's condition was at baseline
- ESTABLISH INCLUSION AND EXCLUSION CRITERIA FOR CONSIDERATION OF THROMBOLYTICS (SEE BELOW):

Table 161-11 American Heart Association/American Stroke Association 2007 Criteria for IV Recombinant Tissue Plasminogen Activator (rtPA) in Acute Ischemic Stroke	
Indications	
Measurable diagnosis of acute ischemic stroke	Use of NIHSS recommended. Stroke symptoms should not be clearing, minor, or isolated. Caution is advised before giving rtPA to persons with severe stroke (NIHSS score of >22), because they have increased risk of intracerebral hemorrhage; however, they are at high risk of death, regardless.
Age ≥18 y	No clear upper age limit.
Time of symptom onset ≤3 h	Must be *well established* [2009 AHA/ASA Scientific Advisory suggests time window may be extended to 3 to 4.5 h if ECASS criteria are met[71,72] (**Table 161-10**)].
Exclusion Criteria	
Symptoms consistent with subarachnoid hemorrhage	
Seizure with postictal residual neurologic impairments	
Previous head trauma or stroke within preceding 3 mo	
Previous myocardial infarction within preceding 3 mo*	
Previous GI or urinary tract hemorrhage within preceding 21 d	
Major surgery within preceding 14 d	
Prior intracranial hemorrhage	
Pretreatment systolic blood pressure >185 mm Hg *or* diastolic blood pressure >110 mm Hg despite therapy (**Table 161-8**)	
Evidence of active bleeding or acute major fracture	
Blood glucose level <50 milligrams/dL (2.7 mmol/L)	
International normalized ratio >1.7 (oral anticoagulant use in and of itself is not a contraindication to rtPA)	
Use of heparin within preceding 48 h *and* a prolonged activated partial thromboplastin time	
Platelet count <100,000/mm^3	
Head CT shows multilobar infarction (hypodensity of more than one third cerebral hemisphere) or hemorrhage or tumor	
Failure of the patient or responsible party to understand the risks and benefits of, and alternatives to, the proposed treatment after a full discussion	

- European society has time of onset inclusion up to 4.5 hours
- NEED TO EXCLUDE "STROKE MIMICS" :

Table 161-5 Differential Diagnoses of Consequence for Acute Stroke Symptoms	
Stroke Mimic	**Distinguishing Clinical Features**
Seizures/postictal paralysis (Todd paralysis)	Transient paralysis following a seizure, which typically disappears quickly; can be confused with transient ischemic attack. Seizures can be secondary to a cerebrovascular accident.
Syncope	No persistent or associated neurologic symptoms.
Brain neoplasm or abscess	Focal neurologic findings, signs of infection, detectable by imaging.
Epidural/subdural hematoma	History of trauma, alcoholism, anticoagulant use, bleeding disorder; detectable by imaging.
Subarachnoid hemorrhage	Sudden onset of severe headache.
Hypoglycemia	Can be detected by bedside glucose measurement, history of diabetes mellitus.
Hyponatremia	History of diuretic use, neoplasm, excessive free water intake.
Hypertensive encephalopathy	Gradual onset; global cerebral dysfunction, headache, delirium, hypertension, cerebral edema.
Meningitis/encephalitis	Fever, immunocompromise may be present, meningismus, detectable on lumbar puncture.
Hyperosmotic coma	Extremely high glucose levels, history of diabetes mellitus.
Wernicke encephalopathy	History of alcoholism or malnutrition; triad of ataxia, ophthalmoplegia, and confusion.
Labyrinthitis	Predominantly vestibular symptoms; patient should have no other focal findings; can be confused with cerebellar stroke.
Drug toxicity (lithium, phenytoin, carbamazepine)	Can be detected by particular toxidromes and elevated blood levels. Phenytoin and carbamazepine toxicity may present with ataxia, vertigo, nausea, and abnormal reflexes.
Bell's palsy	Neurologic deficit confined to isolated peripheral seventh nerve palsy; often associated with younger age.
Complicated migraine	History of similar episodes, preceding aura, headache.
Ménière disease	History of recurrent episodes dominated by vertigo symptoms, tinnitus, deafness.
Demyelinating disease (multiple sclerosis)	Gradual onset. Patient may have a history of multiple episodes of neurologic findings in multifocal anatomic distributions.
Conversion disorder	No cranial nerve findings, nonanatomic distribution of findings (e.g., midline sensory loss), inconsistent history or examination findings.

- MAJOR MIMICS TO REMEMBER → SEIZURES, CONFUSIONAL STATES, SYNCOPE, TOXINS, NEOPLASMS, SUBDURAL (DESCENDING ORDER OF FREQUENCY)

NEUROLOGIC EXAMINATION:

- Focus the examination on the level of consciousness and visual, motor, sensory, coordination and language functions → goal of confirming diagnosis of stroke and potentially localising the stroke lesion
- NIHSS → 11 category neurologic evaluation giving score between 0-42, that yields reproducible results with high interrater reliability and provides a score that correlates with INFARCT VOLUME:
    - Level of consciousness
        - Commands/questions
    - Best gaze
    - Visual fields
    - Facial palsy
    - Motor arm
    - Motor leg
    - Limb ataxia
    - Sensory
    - Best language → degree of aphasia
    - Dysarthria
    - Inattention
        - Widely accepted stroke thrombolytic therapy protocols incorporate its use in ED → excellent practical template for comprehensive, yet rapid assessment of the stroke patient
        - DISADVANTAGES → weighted towards ANTERIOR CIRCULATION STROKES as opposed to posterior circulation strokes
        - Favourable outcomes reported for patients with scores ≤5 for anterior strokes and ≤8 for posterior strokes

<u>CARDINAL FEATURES OF STROKE SYNDROMES:</u>

TRANSIENT ISCHAEMIC ATTACK:

- Defined as "a transient episode of neurological dysfunction caused by focal brain, spinal cord or retinal ischaemia WITHOUT ACUTE INFARCTION" → duration of symptoms is an unreliable discriminator between TIA and infarction
- Analogous to unstable angina
- Overall 90-day stroke risk after TIA is ≥9.5% → 50% occur within 2 days after presentation to ED
    - Increased risk of stroke → age ≥60, HT, DM, symptoms >10 minutes, weakness, speech impairment

ISCHAEMIC STROKE SYNDROMES:

- ANTERIOR CEREBRAL ARTERY INFARCTION:
    - Uncommon
    - Causes contralateral sensory and motor symptoms in the LOWER EXTREMITY → sparing hands and face
    - Left → akinetic mutism, right → confusion and motor hemineglect

- MIDDLE CEREBRAL ARTERY INFARCTION:
  - Most commonly involved in stroke, presentation quite variable → depending on exactly where the lesion is located and which hemisphere is dominant (right handed, and ~80% left-handed patients → left hemisphere is dominant)
  - Typical presentation → hemiparesis, facial plegia, sensory loss contralateral to affected cortex
    - Deficits variably affect face and upper extremity more than lower
    - If dominant hemisphere involved → APHASIA (receptive and/or expressive)
    - Non-dominant → inattention, neglect, structural apraxia
- POSTERIOR CEREBRAL ARTERY INFARCTION:
  - Visual field defects (contralateral homonymous hemianopia)
  - Light-touch and pinprick sensation deficits
  - Inability to name colours
  - Recent memory loss
  - Unilateral third nerve palsy
  - Hemiballismus
- VERTEBROBASILAR INFARCTION:
  - MULTIPLE SIMULTANEOUS SIGNS AND SYMPTOMS
    - MOST COMMON → VERTIGO (usually with other cerebellar or brainstem signs)
    - Headache
    - Nausea/vomiting
    - Visual disturbances
    - Oculomotor palsies
    - Oropharyngeal dysfunction
- **HALLMARK OF POSTERIOR CIRCULATION STROKE IS CROSSED NEUROLOGIC DEFICITS (I.E. IPSILATERAL CRANIAL NERVE DYSFUNCTION AND CONTRALATERAL MOTOR WEAKNESS).**
- BASILAR ARTERY OCCLUSION:
  - SEVERE QUADRIPLEGIA, coma and locked-in syndrome → complete muscle paralysis except for upward gaze
  - High risk of death and poor outcomes
- CEREBELLAR INFARCTION:
  - Frequently present with VERTIGO, GAIT INSTABILITY, LIMB ATAXIA, HEADACHE, DYSARTHRIA, NAUSEA/VOMITING AND CRANIAL NERVE ABNORMALITIES
  - On CT of posterior fossa, bone artifact can obscure imaging → EMERGENT MRI or MRA should be performed for diagnosis
  - Most important factor influencing outcome is PRESENCE OF OBSTRUCTING HYDROCEPHALUS
- LACUNAR INFARCTION:
  - Pure motor or sensory deficits caused by infarction of small penetrating arteries and are commonly associated with chronic HT

    - Presentation is variable depending on location and size of the lesion → many are subclinical
    - Prognosis more favourable
- CERVICAL ARTERY DISSECTION:
    - Uncommon overall, but is an important cause of stroke in YOUNG TO MIDDLE-AGED PATIENTS (10-25% cases
    - Can occur in both anterior and posterior circulations → peak incidence in fifth decade of life
    - RISK FACTORS:
        - Neck trauma (can be trivial, including manipulation of the neck)
        - Family history arterial disease
        - Recent respiratory infection
        - Connective tissue disease
        - History of migraine
    - Typical first symptoms of patients with ICA dissection is UNILATERAL HEAD PAIN (most often frontoparietal region), followed by facial pain or neck pain → precedes other symptoms by hours to days → pain is only symptom in cervical artery dissection in 8% cases
- VERTEBRAL ARTERY DISSECTION:
    - Posterior neck pain and headache (typically occipital) are most common
    - Other symptoms and signs → unilateral facial paraesthesiae, dizziness, vertigo, N+V, diplopia, ataxia, limb weakness, dysarthria
    - Median time from neck pain to neurological signs → 14 days
    - MRI/MRA/CTA have replaced formal DSA in diagnosis
    - TREATMENT OF DISSECTION TRADITIONALLY HAS BEEN WITH ANTICOAGULATION, DESPITE EVIDENCE SUPPORTING ITS USE

DIAGNOSIS:

- Many conditions can simulate a stroke, but most can be distinguished by careful history-taking and exam
- TIME IS BRAIN → hence an organised protocol for ED evaluation with suspected stroke is recommended → decide on treatment within 60 minutes → USE OF STROKE TEAMS NOW WIDESPREAD
    - Nonessential testing should not delay brain imaging ASAP (recommended within 25 minutes)

IMAGING:

- CT head is the most commonly used imaging modality in suspected acute stroke → most acute ischaemic strokes are not visualised by non-contrast stroke → utility exists with exclusion of ICH< abscess, tumour and other stroke mimics → within 48 hours, CT will identify almost all parenchymal haemorrhages >1cm in diamter and 95% SAH
    - The CT should be evaluated as soon as the study is completed by the most expert operator, especially if thrombolytic therapy is under consideration
- MRI more sensitive than CT in detection of acute stroke (46% vs 10%), but results similar when scan performed ≤3 hours

- MRI far better for posterior fossa strokes

TREATMENT OF T.I.A. AND ISCHAEMIC STROKE:

STANDARD TREATMENT:

- INITIAL STABILISATION
- NORMALISE PHYSIOLOGICAL PARAMETERS → based on consensus rather than RCT evidence
  - DEHYDRATION → contributes to poor outcome due to ↑d viscosity, hypotension, recurrent strokes, VTE → no definitive evidence other than perhaps in those with severe polycythaemia
  - HYPOXIA → routine oxygen administration DOES NOT IMPROVE OUTCOME IN MILD TO MODERATE STROKE → aim saturations ≥92%
  - HYPERPYREXIA → fever is associated with ↑d morbidity and mortality → but convincing data is lacking, but judicious control of temperature is reasonable
  - HYPERGLYCAEMIA → multifactorial in stroke, control is recommended
  - HYPERTENSION → several studies have shown a correlation between HT and poor outcomes, but poor outcomes have also been seen in those with active attempts to lower BP, perhaps due reduction in perfusion to ischaemic penumbra of damaged brain. AHA/ASA guidelines DICHOTOMISE PATIENTS ON BASIS OF POTENTIAL FOR ACUTE REPERFUSION THERAPY → in those not for TPA → PERMISSIVE HYPERTENSION, with no active attempts to lower BP unless BP >220/120. Published reduction targets of 10-25% over first day
    - Conversely → patients for TPA, BP control prior to, during and after thrombolysis is CRUCIAL. BP ≥185/110 is a contraindication for TPA, because ↑d BP is associated with haemorrhagic transformation.
    - Thus if a patient is a candidate for TPA → active lowering of BP

THROMBOLYSIS:

- **NINDS** study was an RCT/double blind trial compairing IV TPA with placebo, administered within 3 hours of symptom onset, with ~50% treated within 90 minutes
  - No difference between two groups at 24 hours, but favourable outcome recorded at 3 months (11-13% ARR)
  - Symptomatic ICH 6.4% in TPA with 45% mortality vs 0.6% in placebo group
  - Patients left severely disabled lower in those receiving TPA
- CONCERNS WITH TPA IN ACUTE STROKE:
  - Lack of subsequent RCT
  - Early treatment effect (i.e. those presenting <90 minutes)

    - NINDS trial only had 624 patients, with imbalance (based on chance) in stroke severity between two groups that favoured TPA group
- ECASS III (European Cooperative Acute Stroke Study) → expanded to 4.5 hours for inclusion:
    - Mortality similar in both groups
    - Higher rates of ICH in TPA group
- Use of intra-arterial thrombolyis is increasing and can occur to an expanded treatment window of ≥6 hours, with ability to specifically evaluate the occluded vascular territory and lower total dose of TPA with possibility of mechanical clot disruption → NEEDS INTERVENTIONAL RADIOLOGY TO BE AVAILABLE

Patient selection criteria for thrombolysis in stroke (Table 7.16)

Indications

- onset of ischaemic stroke within the preceding 4.5 hours
- measurable and clinically significant deficit on NIH Stroke Scale examination [NB1]
- patient's CT scan does not show haemorrhage or nonvascular cause of stroke
- patient's age is more than 18 years

Absolute contraindications

If any of the following apply, do NOT use alteplase:

- uncertainty about time of stroke onset (eg patients awaking from sleep)
- coma or severe obtundation with fixed eye deviation and complete hemiplegia
- minor stroke deficit that is rapidly improving
- seizure observed or known to have occurred at onset of stroke
- hypertension: systolic blood pressure 185 mm Hg or more, or diastolic blood pressure more than 110 mm Hg on repeated measures
- clinical presentation suggestive of subarachnoid haemorrhage even if the CT scan is normal
- presumed septic embolus
- patient has received heparin within the last 48 hours and has elevated APTT or has a known hereditary or acquired haemorrhagic diathesis (eg PT or APTT greater than normal)
- INR more than 1.5
- platelet count less than 100 x 10^9/L
- plasma glucose concentration less than 2.8 mmol/L or more than 22 mmol/L

Relative contraindications

If any of the following apply, use alteplase with caution. In each situation the balance of the potential risks and benefits must be carefully considered:

- severe neurological impairment with NIH Stroke Scale score more than 22
- age more than 80 years
- CT evidence of extensive MCA territory infarction (sulcal effacement or blurring of grey-white junction in greater than one-third of MCA territory)
- stroke or serious head trauma within the past 3 months where the risks of bleeding are considered to outweigh the benefits of therapy
- major surgery within the last 14 days
- patient has known history of intracranial haemorrhage, subarachnoid haemorrhage, known intracranial arteriovenous malformation or previously known intracranial neoplasm such that, in the opinion of the clinician, the increased risk of intracranial bleeding would outweigh the potential benefits of treatment
- suspected recent (within 30 days) myocardial infarction
- recent (within 30 days) biopsy of a parenchymal organ or surgery that, in the opinion of the clinician, would increase the risk of unmanageable (eg uncontrolled by local pressure) bleeding
- recent (within 30 days) trauma with internal injuries or ulcerative wounds
- gastrointestinal or urinary tract haemorrhage within the last 30 days or any active or recent haemorrhage that, in the opinion of the clinician, would increase the risk of unmanageable (eg by local pressure) bleeding
- arterial puncture at noncompressible site within the last 7 days
- concomitant serious, advanced or terminal illness or any other condition that, in the opinion of the clinician, would pose a risk to treatment

The regimen for thrombolytic therapy is:

alteplase 0.9 mg/kg up to 90 mg IV over 1 hour, with 10% of the dose given as an initial bolus. Withhold aspirin for 24 hours.

- AFTER ADMINISTRATION OF TPA → BP AND NEUROLOGICAL CHECKS SHOULD BE PERFORMED EVERY 15 MINUTES FOR 2 HOURS
- No antiplatelets or anticoagulants should be administered in the initial 24 hours
- ADMIT TO A SPECIALISED STROKE UNIT
- If post-TPA bleeding is suspected → EMERGENT CT BRAIN and cross match for packed red cells, FFP, cryoprecipitate and platelets.
    - If confirmed, emergent neurosurgery input

ANTIPLATELET THERAPY:

Aspirin has a modest benefit when administered within 48 hours of acute ischaemic stroke, and should be used routinely. It should not be given until brain imaging excludes intracranial haemorrhage. If the patient has received alteplase, withhold aspirin for 24 hours and commence after follow-up imaging excludes haemorrhage.

Use:

aspirin 150 to 300 mg orally or via nasogastric tube or rectally, on the first day (initial CT scan is required to exclude cerebral haemorrhage). Dose can be reduced to 75 to 150 mg daily thereafter [Note 1].

For rapid action, soluble aspirin 300 mg will achieve maximum effect within 30 minutes.

There are insufficient data to recommend routine use of other antiplatelet drugs for acute ischaemic stroke.

- After TIA, use of aspirin to prevent vascular events is well accepted → one study showed combined treatment with dipyrimadole was superior to aspirin alone
- Benefit of administration of aspirin within the first 48 hours seems to be related to REDUCTION OF RECURRENT STROKE
    - Aspirin is not thought to interfere with subsequent consideration of thrombolytics

ANTICOAGULATION:

- WARFARIN IS THE TREATMENT OF CHOICE FOR STROKE PREVENTION IN THOSE WITH NONVALVULAR ATRIAL FIBRILLATION AND TIA
    - Risk of recurrent stroke infirst 48 hours without anticoagulation is very low (<5%) and the risk of haemorrhagic transformation is greatest in this time period
- HOWEVER:
    - A Cochrane meta-analysis of eight RCT (22,125 patients) found NO NET BENEFIT OF ANTICOAGULANTS IN ACUTE STROKE
    - In addition, a recent meta-analysis found no benefit in cardiothrombotic stroke
    - HENCE → EVEN IN THE PRESENCE OF A.F. ANTICOAGULATION IS NOT RECOMMENDED ON THE AVAILABLE EVIDENCE AT PRESENT
- In setting of concurrent AMI, heparin does reduce extension or reinfarction, but the risk of mortality, revascularisation and major/minor bleeding, recurrent angina are similar in those treated with or without heparin

NEUROSURGICAL INTERVENTION:

- Required early in all those with cerebellar infarction → high risk of herniation, hence early consideration of posterior fossa decompression
- HEMICRANIECTOMY CAN BE LIFE-SAVING IN THOSE WITH LARGE AMOUNT OF HEMISPHERIC INFARCTION AND RELATED OEDEMA → can reduce mortality by 50%

DISPOSITION AND FOLLOW UP:

TRANSIENT ISCHAEMIC ATTACK:

- Should be viewed as an ominous sign of cerebral vascular disease
- Scoring system → ABCD2

Table 161-12 ABCD^2 Score to Predict Very Early Stroke Risk after Transient Ischemic Attack

Criteria	Points
Age ≥60 y	0 = Absent
	1= Present
Blood pressure ≥140/90 mm Hg	0 = Absent
	1= Present
Clinical features	0 = Absent
	1 = Speech impairment without unilateral weakness
	2 = Unilateral weakness (with or without speech impairment)
Duration	0 = Absent
	1 = 10–59 min
	2 = ≥60 min
Diabetes	0 = Absent
	1 = Present

- Johnson *et al* reported a 2 day risk of subsequent stroke as:
  - 1% if score 0-3, 4.1% if score 4-5, 8.1% if score 6-7
  - Allows risk stratification of patients with TIA that may allow outpatient work up
- OBVIOUSLY, ALL PATIENTS WITH ACUTE STROKE SHOULD BE ADMITTED TO MONITORED CARE UNITS, FAMILIAR WITH CARE OF STROKE PATIENTS
  - Use of stroke units are associated with decreased complications, length of stay, improved daily function and decreased rate of discharge to long-term care facilities→ ALL INDEPENDENT OF USE OF LYTICS

SPECIAL POPULATIONS:

- SICKLE CELL DISEASE:
  - Most common causes of ischaemic stroke in children

    - Stroke occurs by age 45 in 25% patients with homozygous disease → STOP study showed prophylactic transfusion inpatients with abnormal Doppler US reduced stroke by 90%
    - Cerebral aneurysms occur at higher rates in SCD
    - Initial management is similar, but care should be taken to treat underlying SCD with oxygen, adequate hydration and pain control if required
    - CONSIDER EXCHANGE TRANSFUSION IN PATIENTS WITH SICKLE CELL AND ACUTE STROKE
    - Not a contraindication to TPA
- YOUNG ADULTS:
    - Cervical artery dissection occurs in 20% of people under 50 with ischaemic stroke
    - Cardioembolic events → mitral prolapse, rheumatic heart disease, paradoxical embolism
- PREGNANT WOMEN:
    - Increased risk both during pregnancy and up to 6 weeks post-partum
    - Contributors → SCD, preeclampsia, HT, DM, drug abuse
    - Low dose aspirin has been used in the second trimester of those with preeclampsia without complications
    - TPA does not cross the placenta, but pregnancy is a relative contraindication