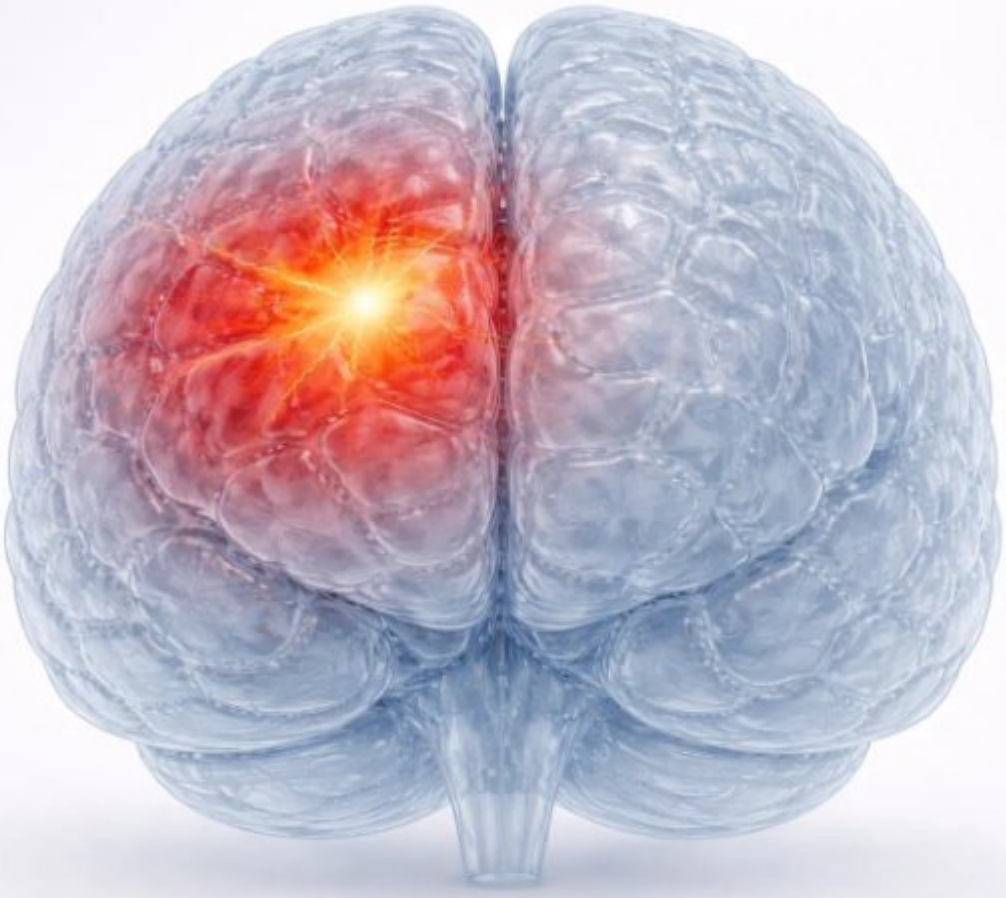
STROKE

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

JFPR
Japan Fund for Prosperous and Resilient Asia and the Pacific

From the People of Japan

World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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STROKE

STROKE

QUICK REFERENCE GUIDE

Stroke is a major global health challenge with prevalence continuing to rise worldwide. It affects people across all age groups, not just the elderly, and carries a high risk of death and disability, especially in hemorrhagic cases and where timely care is limited. Early recognition using tools like FAST and rapid imaging is essential for initiating treatment. Prompt management with standardized protocols such as thrombolysis, blood pressure control, and secondary prevention, greatly improves outcomes by reducing mortality, disability, and recurrence.

Definition

- **Stroke:** Rapid-onset focal (or global) neurologic dysfunction due to vascular cause, ischemic or hemorrhagic.
- **Ischemic stroke (IS):** Brain infarction from arterial occlusion (thrombotic/ embolic).
- **Transient ischemic attack (TIA)**: Focal ischemia without acute infarction (DWI-negative).
- **Intracerebral hemorrhage (ICH):** Non-traumatic bleed into brain parenchyma.
- **Subarachnoid hemorrhage (SAH):** Bleed into subarachnoid space, often aneurysmal.

Causes, Risk factors & Triggers

- **Causes (ischemic):** large-artery atherosclerosis, cardioembolism (AF, valves), small-vessel (lacunar), watershed; hemorrhagic: hypertensive rupture, cerebral amyloid angiopathy, vascular malformation, coagulopathy.
- **Risk factors (modifiable):** hypertension, diabetes, dyslipidemia, smoking, AF, carotid stenosis, obesity/ inactivity, OSA, alcohol excess, diet. Non-modifiable: age, prior stroke/TIA, family history.
- **Triggers:** infection, uncontrolled BP, arrhythmia, dehydration, stimulant use, perioperative stress, stopping antithrombotics.

Evaluation for Diagnosis

- **Clinical features:** sudden focal deficit (weakness, aphasia, neglect, visual loss, ataxia), severe headache (SAH), decreased level of consciousness (large stroke/ICH).
- **Physical exam:** vitals, glucose; focused neuro exam (cranial nerves, motor, sensory, speech, gaze), signs of raised ICP; cardiac and carotid exam.
- **Labs:** CBC, electrolytes, renal function, glucose, coagulation (if bleeding risk/ anticoagulants).

- **Confirmation (imaging):**
  - **NCCT head ≤20 min** to exclude hemorrhage and assess early ischemia (ASPECTS).
  - **CTA ± CTP** for LVO and penumbra (thrombectomy selection).
  - **MRI DWI/FLAIR/GRE/SWI/MRA** where available (small/posterior infarcts, TIA vs minor stroke, microbleeds).

Classification / severity assessment criteria

- **NIHSS (0–42):** quantifies ischemic stroke severity; ≥4-point change is meaningful.
  - 0: none; 1–4: minor; 5–15: moderate; 16–20: mod-severe; >20: severe.
- **ICH Score (0–6):** predicts 30-day mortality (GCS, volume >30 mL, IVH, infratentorial, age ≥80).

Differential Diagnosis

Hypoglycemia, seizure with Todd's paresis, migraine aura, brain tumor, peripheral facial palsy, conversion disorder, metabolic derangements, chronic subdural hematoma, CNS infection.

Management Goals & principles

- Ischemic: restore/preserve perfusion; prevent recurrence.
- Hemorrhagic: limit hematoma expansion; control ICP.
- Universal: minimize time-to-imaging/treatment, optimize BP/O_2/glucose, prevent complications, start early rehab, coordinate multidisciplinary care.

Approach to management

- ABCs, O_2 ≥94%, glucose check, IV access, labs, NIHSS.
- Immediate imaging: NCCT ± CTA/CTP (or MRI-first protocol).
- Branch: ischemic vs hemorrhagic.
- Parallel bundle: swallow screen, DVT prevention, temperature and glucose control, early rehab, risk-factor plan.

Stroke Care Bundle (0–24 hours)

1. Pre-hospital

- FAST positive → EMS pre-notify stroke team; record last-known-well (LKW).
- Check capillary glucose, vitals; give O_2 only if SpO_2 <94%.
- Triage to thrombectomy-capable center if suspected LVO and transport times allow.
- For transfer hospitals, aim door-in-door-out ≤60 min. (www.heart.org, American Heart Association Journals)

2. ED arrival: first 10–20 minutes

- ABCs, two IV lines, labs (CBC, CMP, PT/INR, troponin if indicated), ECG; NIHSS.
- Non-contrast CT head ≤20 min from arrival; add CTA ± CTP if available.
- Keep BP permissive until imaging (unless extremes or tPA planned), treat hypoglycemia, maintain SpO_2 ≥94%. (www.heart.org)

3. Ischemic stroke pathway (no hemorrhage on CT/MRI)

- IV thrombolysis: evaluate eligibility; door-to-needle ≤60 min.
  - Lower BP to ≤185/110 before thrombolysis; maintain ≤180/105 for 24 h after.
  - No antiplatelet/anticoagulant for 24 h; repeat brain imaging at 24 h. (www.heart.org, American Heart Association Journals)
- Endovascular thrombectomy (EVT): for LVO within 0–6 h, and 6–24 h in selected patients using perfusion/ clinical mismatch. Expedite vascular imaging and team activation.
- If not receiving reperfusion: start aspirin when safe (typically after hemorrhage excluded), start high-intensity statin, manage BP and risk factors per protocol. (American Heart Association Journals)

4. Hemorrhagic stroke pathway (ICH/ SAH)

- Rapid BP lowering with protocolized IV agents; typical ICH target SBP ~140–160 early (per guideline and local protocol).
- Reverse anticoagulation/antiplatelet effects as indicated.
- Neurosurgery/endovascular consult; consider CTA to evaluate aneurysm/ AVM in SAH.
- ICP measures, head-of-bed elevation, temperature and glucose control. (American Heart Association Journals, www.heart.org, cpr.heart.org)

5. Universal nursing/medical bundle (applies to all stroke)

- Swallow screen before any oral intake; keep NPO until passed.
- Temperature: treat fever; Glucose: target moderate control (avoid <67 mg/dL / 3.7 mmol/L).
- VTE prophylaxis per pathway; early mobilization and rehab once safe.
- Dysphagia, fever, hyperglycemia care are core nurse-led elements (QASC). (professional.heart.org, European Stroke Organisation)

6. Secondary prevention (start during admission)

- Determine etiology (TOAST): cardioembolic vs large-artery vs small-vessel.
- Anticoagulation for atrial fibrillation (timing per infarct size/hemorrhagic risk).
- Carotid revascularization if indicated; control BP, lipids, diabetes, stop tobacco; sleep apnea screening. (American Heart Association Journals)

7. Time targets summary

- Door-to-imaging (NCCT) ≤ 20 min.
- Door-to-needle (IV thrombolysis) ≤ 60 min.
- Transfer hospitals (Door In Door Out): DIDO ≤ 60 min.
- **Endovascular treatment (EVT):** within window (0–6 h; 6–24 h when imaging shows mismatch). (www.heart.org, American Heart Association Journals, NEJM)

Non-Pharmacological interventions

- Swallow screen before any PO; NPO if failed; texture/posture strategies; SLT referral (tele-SLT if needed).
- Early mobilization when stable (24–48 h); PT/OT for gait/ADLs; caregiver training.
- DVT prevention: intermittent pneumatic compression; mobilize early.
- Nutrition/hydration: dietitian input; enteral feeding if unsafe oral intake; monitor electrolytes and refeeding risk.
- Low-resource setting: prioritize NCCT + tele-consult, validated checklists (FAST, NIHSS prompts), protocol posters, and transfer pathways to thrombectomy-capable centers.

Pharmacological therapy (indications, dose, route, duration, cautions)

Indication	Drug (route)	Dose / Duration	Key cautions
IV thrombolysis (≤4.5 h)	**Alteplase (IV)**	0.9 mg/kg (max 90 mg): 10% bolus, rest over 60 min	Image-confirmed no hemorrhage; BP ≤185/110 before, <180/105 for 24 h; no antithrombotics for 24 h; 24-h CT/MRI first
	Tenecteplase (IV)	0.25 mg/kg (max 25 mg) single bolus (select protocols)	Cost implication; Off-label in many regions; confirm local protocol
Thrombectomy	—	0–6 h (to 24 h in mismatch)	Requires LVO on CTA/MRA; activate team early
Antiplatelet (non-cardioembolic)	**Aspirin or Clopidogrel**	75–100 mg daily **or** 75 mg daily long-term	Start after hemorrhage excluded; if IVT given, wait 24 h and re-image
DAPT (high-risk TIA/minor IS)	**Aspirin + Clopidogrel**	Load clopidogrel 300 mg (some use 600 mg) → 75 mg daily + aspirin 75–100 mg daily for **21 days**, then single agent	Not for long-term use
Anticoagulation (AF)	**Apixaban**	5 mg BID (2.5 mg BID if ≥2 of: age ≥80, wt ≤60 kg, Cr ≥1.5 mg/dL)	Start timing by infarct size/bleed risk (e.g., 1–3–6–12 day rule; earlier per ELAN selection)
	Rivaroxaban	20 mg QPM with food (CrCl ≤50 → 15 mg QPM)	
	Warfarin	INR 2.0–3.0	Bridging usually not needed with DOAC-eligible patients
BP control (ischemic)	**Labetalol/Nicardipine/ Clevidipine**	Reperfusion candidate: ≤185/110 pre-treatment; <180/105 for 24 h after. No reperfusion: treat if SBP >220 or comorbidity; reduce ~15%/24 h	Avoid rapid drops—protect penumbra
ICH/SAH essentials	**Labetalol/Nicardipine; Mannitol/HTS;** reversal agents (vit K + PCC; idarucizumab; andexanet)	SBP often 140–160 early; ICP control; reverse anticoagulation promptly	Neurosurgery/endo consult early; EVD for hydrocephalus; avoid hypotonic fluid and maintain normothermia/normoglycemia.

Post-thrombolysis monitoring timeline with the non-pharmacological stroke care bundle

Time window / Bundle element	What to do	Start / Withhold	Who	Targets
0–24 h (acute phase)	Stabilize; confirm subtype; prevent complications. Neuro checks q15 min ×1 h → q30 min ×6 h → q1h until 24 h. BP per pathway, SpO_2 ≥94%, glucose 140–180 mg/dL, temp control. NPO until swallow screen.	If IVT: BP ≤180/105; no antiplatelet/anticoagulant for 24 h; avoid invasive lines. If no IVT: aspirin once hemorrhage excluded. Start DVT prevention (mechanical).	ED/ICU team, stroke nurse	NCCT/MRI at 24 h (or earlier if deterioration). Activate rehab after swallow cleared.
Swallow assessment	Formal bedside swallow screen before any oral intake. Failed screen → keep NPO, SLT review, alternative nutrition.	At admission, before PO intake, and after neuro changes.	SLT, trained nurse	Prevent aspiration pneumonia. Document safe consistencies; re-screen as needed.
Early rehabilitation	Physiotherapy (PT): motor recovery, gait, tone, balance. Occupational therapy (OT): ADLs, upper-limb function, adaptive techniques. SLT: aphasia/dysarthria, communication, swallowing training.	Start once stable; aim 24–48 h. Withhold if raised ICP, active hemorrhage expansion, uncontrolled BP/O_2/glucose.	PT, OT, SLT, nurse, physician	Reduce deconditioning, pneumonia, DVT. Daily functional goals and reassessment.
24–48 h (subacute)	Begin secondary prevention. Continue neuro checks, BP/glucose control. Watch for hemorrhagic transformation.	Antiplatelet: aspirin 150–300 mg load → 75–100 mg daily. If minor stroke/high-risk TIA: short DAPT (aspirin + clopidogrel) for 21 days. Start statin. VTE prophylaxis (LMWH/UFH) if safe.	Physician, nurse	Review 24-h scan before starting antithrombotics. Cardiac monitoring, echo, vascular imaging. Escalate rehab; caregiver education.
DVT prophylaxis	IPC stockings or devices unless contraindicated; mobilization ladder (bed↔chair, stand, ambulate).	Mechanical from day 0. Pharmacologic (LMWH/UFH) only when bleed risk acceptable.	Nursing, PT, physician	Daily mobility documentation; IPC hours tracked.
Nutritional support & hydration	Nutrition screen, calorie/protein needs, dietitian input. Unsafe swallow → NG/PEG feeds. Monitor fluids/electrolytes; watch for refeeding in malnourished patients.	Within 24 h of admission. Withhold PO until swallow cleared.	Dietitian, SLT, nurse, physician	Adequate caloric/protein intake, maintain euvolemia. Prevent refeeding syndrome. Track weight & intake/output.
1–2 weeks (early recovery)	Finalize etiology, escalate rehab, screen for complications (depression, spasticity, aspiration risk).	Anticoagulation for AF: start per infarct size/timing (e.g., 1–3–6–12 days). Carotid endarterectomy/stenting if indicated (ideally ≤2 weeks). Continue antiplatelet/statin/lifestyle measures.	Stroke team, cardiology, rehab, vascular surgery	Outpatient follow-ups arranged (stroke, rehab, cardiology). Screen sleep apnea, driving/work counseling.

Assessment of response, review & follow-up (step-up/step-down)

- Neurologic: NIHSS trend; ≥4-point rise → urgent CT/MRI.
- Imaging: 24-h scan after IVT/EVT; earlier if deterioration.
- BP/glucose/temp: on-target vs adjust drips/insulin/antipyretics.
- Swallow/nutrition: pass screen vs NPO/NG; caloric/protein targets.
- Antithrombotic plan: start/hold dates; DAPT stop at 21 days; anticoag timing by size/bleed risk.
- Rehab milestones: bed→sit→stand→walk; activities of daily living (ADLs); caregiver training.
- Step-up if: worsening neuro status, uncontrolled BP, aspiration, expanding hemorrhage, uncontrolled ICP, recurrent events.
- Step-down if: stable 24–48 h, safe swallow, controlled vitals, clear secondary prevention plan.

Referral (tiered)

- Primary/community: FAST recognition; glucose; NCCT if available; immediate transfer if LVO suspected/ICH; tele-stroke consult.
- Secondary/district: CT/CTA; IVT; stabilize and door-in–door-out ≤60 min if EVT center needed.
- Tertiary: EVT, neurosurgery, stroke unit, advanced imaging, multidisciplinary rehab.

Complications

Hemorrhagic transformation, malignant edema, aspiration pneumonia, DVT/PE, seizures, depression/anxiety, pressure injuries, recurrent stroke.

Objectives of patient education & caregiver instructions

- Know FAST signs; call EMS immediately.
- Medication adherence: antithrombotic, statins, BP/diabetes medicines; clear start/stop dates.
- Risk-factor control: BP, lipids, glucose, smoking cessation, diet (Mediterranean/DASH), exercise 150 min/week, weight targets, limit alcohol; screen/treat sleep apnea.
- Swallow safety: follow texture/posture guidance; re-screen after changes.
- Rehabilitation: home exercises, gait safety, fall prevention, pressure-area care.
- Follow-up: stroke clinic and therapy appointments booked before discharge; bring a med list; know red flags (worsening neuro signs, SOB, chest pain).

INTRODUCTION

Stroke is an acute neurologic syndrome caused by interrupted blood flow (ischemic) or bleeding (hemorrhagic). It is a leading cause of death and disability worldwide. Each year, over 12 million people suffer a first stroke and about 6.5 million die. Since 1990, incident strokes have risen by ~70%, deaths by ~44%, and prevalence by ~86%, with the heaviest burden in low- and middle-income countries.

In Southeast Asia, stroke prevalence and mortality are high, with increasing cases in younger adults. Hypertension and diabetes are the main modifiable risks; large artery atherosclerosis and small vessel disease dominate ischemic mechanisms.

Globally, more than 60% of strokes occur under age 70 and ~16% under 50. Mortality is higher in hemorrhagic strokes and where timely care is lacking. Standardized acute management - early recognition, thrombolysis protocols, blood pressure control, and secondary prevention, reduces death, disability, and recurrence, while inconsistent care leads to delays and missed opportunities for recovery.

SCOPE OF THE GUIDELINES

Covers acute, subacute, and secondary prevention phases of stroke (ischemic and hemorrhagic), including transient ischemic attacks (TIA). Includes triage, diagnosis, initial management, risk factor identification, rehabilitation referral, and prevention strategies across care levels.

Intended users

Primary care physicians, nurses, emergency providers, general practitioners, internal medicine physicians, neurologists, radiologists, rehabilitation therapists, and health program managers involved in stroke care delivery at primary, secondary, and tertiary facilities.

Applicability by level of care

- Primary/community: Recognize stroke early (FAST), stabilize ABCs, check glucose and BP, initiate transfer protocols, begin secondary prevention in known cases, risk factor screening, patient/caregiver education. Limited imaging—use clinical scales; teleconsult if available.
- Secondary (district/regional): Perform CT and labs if available, start acute stroke pathways (thrombolysis if within window), identify hemorrhage, initiate BP control, begin rehabilitation, manage complications. May lack advanced intervention; follow algorithms.

- Tertiary (specialist centers): Implement full stroke protocol with advanced imaging, assessing eligibility for thrombolysis or thrombectomy, neurosurgical evaluation, intensive monitoring in stroke units, multidisciplinary rehab, comprehensive etiologic workup, and tailored secondary prevention.

DEFINITION

- **Stroke:** Rapid onset of focal (or at times global) neurological dysfunction caused by a vascular event (ischemia or hemorrhage), persisting >24 hours or leading to death, with no apparent nonvascular cause. (AHA/ASA, ESO)
- **Transient Ischemic Attack (TIA):** A brief episode of neurological dysfunction due to focal brain, spinal cord, or retinal ischemia **without evidence of acute infarction.** Symptoms typically resolve within minutes to <1 hour; by older definitions within 24 hours. (AHA/ASA, ESO)
- **Ischemic Stroke (IS):** Acute brain infarction resulting from obstruction of blood flow in an artery, commonly due to thrombus or embolus. (AHA/ASA, ESO)
- **Intracerebral Hemorrhage (ICH):** Spontaneous, non-traumatic bleeding into the brain parenchyma, often from small vessel disease, hypertension, or vascular malformations. (AHA/ASA, ESO)
- **Subarachnoid Hemorrhage (SAH):** Bleeding into the subarachnoid space, usually from rupture of an intracranial aneurysm or other vascular lesion. (AHA/ASA, ESO)
- **Penumbra:** Hypo-perfused but structurally viable brain tissue surrounding the infarct core, potentially salvageable with timely reperfusion. (AHA/ASA, ESO)

CAUSES, RISK FACTORS, AND TRIGGERS

Category	Details
Causes	Thromboembolism – Cardioembolic (atrial fibrillation, valvular disease) • Large artery atherosclerosis • Small vessel disease (lacunar stroke) • Hemorrhagic – Hypertension-induced rupture, cerebral amyloid angiopathy, vascular malformations, coagulopathy • Other/Uncommon – Vasculitis, hypercoagulable states, reversible cerebral vasoconstriction syndrome (RCVS), infections, drug-induced
Risk Factors	Modifiable: Hypertension, diabetes mellitus, dyslipidemia, smoking/tobacco, physical inactivity, obesity, atrial fibrillation, carotid stenosis, excessive alcohol, unhealthy diet, sleep apnea Non-modifiable: Age, male sex (variable), family history, previous stroke/TIA, ethnicity (e.g., higher risk in South Asian populations)
Triggers	Acute infection, uncontrolled blood pressure, arrhythmia, dehydration, sudden cessation of antithrombotic therapy, stimulant use (e.g., cocaine, amphetamines), perioperative stress

EVALUATION FOR DIAGNOSIS

Early recognition of stroke is critical. The FAST acronym is a quick bedside screen to triage stroke.

Letter	Sign	Description	Action
F	Face drooping	Sudden asymmetry when smiling; one side may sag or fail to move.	Check facial movement; note side affected.
A	Arm weakness	Inability to raise one or both arms evenly; drift or weakness, usually on one side.	Ask the person to raise both arms; observe for weakness or drift.
S	Speech difficulty	Slurred, garbled, or incoherent speech; inability to repeat simple phrases (aphasia/dysarthria).	Ask to repeat a simple phrase; listen for errors or slurring.
T	Time to act	Presence of any above signs requires urgent medical evaluation.	Call emergency services immediately; rapid recognition is critical for treatment.

Beyond FAST, other important clinical features include:

- Sudden onset of focal neurological deficits:
  - Weakness or numbness in the face, arm, or leg, especially if unilateral
  - Visual disturbances such as field cuts or double vision
  - Gait disturbance, imbalance, or ataxia
  - Sudden severe headache, particularly "the worst headache of life" (raises concern for subarachnoid hemorrhage or other hemorrhagic stroke)
  - Altered level of consciousness, more common in large infarcts or hemorrhages

Note: FAST is sensitive for anterior circulation strokes but may miss posterior circulation events (e.g., isolated vertigo, nausea, diplopia, or bilateral limb ataxia). A high index of suspicion should be maintained when sudden neurological symptoms occur even if FAST is negative.

Assessment	Key Components	Clinical Purpose
Vital Signs	Blood pressure, heart rate & rhythm, oxygen saturation, bedside blood glucose	Detects mimics (hypoglycemia), identifies triggers (arrhythmia, hypertension), guides acute management
Neurological Exam	Cranial nerves, motor strength/tone, sensory deficits, reflexes, coordination, speech, gaze deviation	Localizes lesion, gauges severity, baseline for NIHSS
Raised ICP Signs	Papilledema (if not hyperacute), reduced consciousness, headache, vomiting, abnormal posturing	Suggests intracranial hypertension or large stroke/bleed; informs urgency of neuroimaging
Systemic Exam	Cardiac auscultation (arrhythmia, murmurs), carotid bruit	Identifies embolic sources (AF, valvular disease, carotid stenosis)
Point-of-Care Tests	Blood glucose, SpO_2, ECG, CBC, electrolytes, renal function, coagulation profile, platelet count	Rules out mimics (hypoglycemia, hypoxia), detects cardiac sources (AF, ischemia), screens for bleeding risk & treatment eligibility

CONFIRMATION OF DIAGNOSIS

Accurate and timely confirmation distinguishes ischemic from hemorrhagic stroke, identifies candidates for reperfusion, rules out mimics, and directs secondary prevention. The cornerstone is immediate neuroimaging **to rule out hemorrhagic stroke and assess for large vessel occlusion (ASPECT score)** combined with targeted clinical evaluation and ancillary testing.

Imaging Modality	Role & Key Features	Practical Points / Contrast Use
Non-Contrast CT (NCCT) Head	First-line in almost all acute cases. Rules out intracranial hemorrhage, detects early ischemic signs (loss of gray-white differentiation, hyperdense artery), screens for large infarcts (ASPECTS scoring).	**Target:** "Door-to-imaging" ≤20 min after arrival. Limitations: insensitive for very early ischemia, small posterior or lacunar infarcts.
CT Angiography (CTA)	Evaluates intracranial/extracranial large vessel occlusions, stenosis, collateral status. Key for thrombectomy eligibility.	Use non-ionic iodinated contrast (150–1100 mOsm/kg). Inject with power injector at ~3 mL/sec via large forearm vein.
CT Perfusion (CTP), if available	Differentiates infarct core from salvageable penumbra. Extends treatment window up to 24h in selected patients (when mismatch present).	Often combined with NCCT + CTA in "code stroke" protocols.
MRI (Acute Stroke Protocol)	**Diffusion-weighted imaging (DWI):** Most sensitive for early ischemia; detects cytotoxic edema within minutes. **FLAIR/T2:** Lesion aging; DWI-positive but FLAIR-negative pattern suggests hyperacute stroke, useful in unknown onset. **Gradient echo / susceptibility (T2):*GRE/SWI:** Detects microbleeds or hemorrhagic transformation. **MR Angiography (MRA):** Noninvasive vascular imaging. **Perfusion MRI (PWI):** Perfusion mismatch assessment (penumbra vs core).	MRI contrast (Class II agents) used cautiously; rare NSF risk. Contrast-enhanced MRA may be indicated if non-contrast MRA suboptimal.

Note: Up to ~20–30% of clinically confirmed mild/non-disabling strokes may have negative acute DWI; absence of a lesion does not rule out true ischemic stroke, and secondary prevention should still be applied when clinical suspicion is high.

MRI-first paradigms are increasingly feasible; ultrafast protocols (some as short as 3 minutes) are being evaluated to allow MRI as initial imaging in select centers, improving detection without delaying therapy in systems where MRI access and workflow permit.

Differentiating Ischemic from Hemorrhagic Stroke

- NCCT: High sensitivity for acute hemorrhage; first-line to rule out bleeding. If hemorrhage is seen, evaluate for underlying cause (CTA for aneurysm/AVM in SAH) and consider anticoagulant reversal.

- MRI (GRE/T2*): Further characterizes hemorrhagic components and chronic blood products; useful when etiology or hemorrhagic transformation is uncertain.

Vascular Etiology Workup

- Large vessel: CTA, MRA, or carotid Doppler to detect stenosis, atherosclerosis, dissection, or occlusion.
- Cardioembolic: ECG for atrial fibrillation; echocardiography (TTE/TEE) for cardiac thrombi or valvular disease; prolonged monitoring if initial ECG non-diagnostic.
- Perfusion imaging: CTP or MR perfusion to identify infarct core vs salvageable penumbra, guiding reperfusion decisions (especially in extended/wake-up strokes).

TIA vs Minor Stroke

- MRI-DWI within 1 week differentiates TIA (no infarct) from minor stroke (acute infarct seen). Alters risk stratification and secondary prevention planning.

Stroke Mimics

- Common mimics: hypoglycemia, seizures with Todd paralysis, migraine with aura, functional/psychogenic disorders, tumors, metabolic derangements, neuropathies.
- Workup: Imaging (especially DWI MRI) and labs help exclude mimics. Thrombolysis in mimics carries relatively low risk, but confirmation avoids unnecessary treatment.

Timing & Workflow

- Door-to-imaging target: ≤20 minutes (NCCT first in most systems).
- Imaging-to-decision: Minimize delays for thrombolysis/thrombectomy.
- MRI-first centers: Use ultrafast protocols to avoid reperfusion delays.

Integration into Acute Pathway

- Clinical suspicion → ABC stabilization → rapid NCCT (or MRI in MRI-first systems) → ischemic vs hemorrhagic determination →
- If ischemic: assess reperfusion eligibility (vascular imaging, contraindications).
- If hemorrhagic: manage bleeding, reversal, and neurosurgical referral.
- Begin etiological workup (cardiac, vascular, metabolic) in parallel with acute decision-making.

CLASSIFICATION

The etiology of ischemic stroke is categorized Ischemic strokes are classified into three main subtypes per the TOAST system from the multicenter Trial of ORG 10172 in Acute Stroke. The TOAST classification Treatment which guides secondary prevention:

Subtype	Key Features & Pathophysiology	Typical Imaging Findings	Secondary Prevention Implications
Large Vessel Occlusive Disease	Occlusion of major intracranial/extracranial arteries (often carotid). Causes large territorial infarcts, usually embolic from atherosclerotic plaque.	NCCT: large hypodensity, mass effect, sulcal effacement. CTA/MRA: stenosis or occlusion. DWI MRI: acute infarction.	Antiplatelet therapy (aspirin, dual therapy short-term in select), statins, revascularization (CEA/CAS) if indicated.
Lacunar Infarction (Small Vessel Disease)	Occlusion of deep penetrating arteries due to lipohyalinosis or small-vessel atherosclerosis (HTN, DM). Lesions ≤15 mm in internal capsule, basal ganglia, thalamus, corona radiata.	CT/MRI: small, deep hypodensities. DWI: restricted diffusion in deep structures, low ADC.	Single antiplatelet, strict BP and glycemic control, statins, lifestyle modification.
Cardioembolic Infarction	Emboli from atrial fibrillation, mural thrombus, valvular disease, prosthetic valves, recent cardiac surgery. High early mortality.	DWI: multifocal infarcts in different vascular territories, scattered or bilateral lesions.	Anticoagulation (DOACs/ warfarin depending on context), risk factor management, cardiac source treatment.
Watershed (Border-Zone) Infarction	Hypoperfusion in distal border zones between cerebral arteries (e.g., ACA-MCA). Caused by severe carotid stenosis or systemic hypotension; may also involve micro emboli.	MRI: linear/patchy lesions in border zones, diffusion restriction if acute.	Optimize perfusion (BP management, revascularization if carotid disease), antiplatelet/statins, address precipitating factors (hypotension, arrhythmia).
Hemorrhagic Transformation	Ischemic infarct converts to hemorrhage (2–14 days, often after reperfusion). Risk higher in large infarcts, cardioembolic sources, or post-thrombolysis.	CT: new hyper-densities within prior infarcted area. MRI GRE/SWI: blood products.	Careful BP control, delay or adjust anticoagulation, supportive neurocritical care.

Rarely structural/genetic disorders, and undetermined/cryptogenic when no single cause is clear or workup is incomplete.

SEVERITY ASSESSMENT

Severity and prognosis are quantified with standard scales.

Tool	Score Range	Components	Interpretation / Prognosis	Clinical Use
NIH Stroke Scale (NIHSS)	0–42	■ 15 items: LOC, gaze, vision, facial palsy, motor (arm/leg), limb ataxia, sensory, language, dysarthria, extinction/neglect	■ 0 = No deficit ■ 1–4 = Minor stroke ■ 5–15 = Moderate stroke ■ 16–20 = Moderate–severe ■ >20 = Severe stroke ■ **Change ≥4 points is clinically significant**	Guides reperfusion eligibility (thrombolysis/ thrombectomy), outcome prediction, serial monitoring
ICH Score	0–6	■ Glasgow Coma Scale (GCS) ■ Hemorrhage volume (>30 mL) ■ Intraventricular hemorrhage (IVH) extension ■ Infratentorial origin ■ - Age ≥80 years	■ Higher scores = worse outcome. Mortality rises steeply: ■ 0 = low risk, ■ 5–6 = near 100% 30-day mortality	Prognostication, triage to ICU/stroke unit, neurosurgical referral, family counseling

Diagnostic Flowchart

1. Suspected acute neurologic deficit →
2. Immediate ABCs, vitals, glucose, ECG →
3. Non-contrast CT head within 20 minutes of arrival →
   - If hemorrhage → manage per hemorrhagic protocol
   - If no hemorrhage and within thrombolysis window → assess eligibility for reperfusion
4. If ischemic and candidate → vascular imaging + decision on thrombolysis/ endovascular
4. Etiologic workup initiated in parallel (cardiac, carotid, metabolic)

DIFFERENTIAL DIAGNOSIS

Stroke can be confused with several other conditions. Clinical context, basic labs (including glucose), imaging, and careful neurologic exam help distinguish these from true vascular stroke.

Scenario	Red flags for true vascular stroke (act now)	Clues favoring a mimic	Bedside action
Universal check	Sudden, maximal-at-onset focal deficit; persistent >60 min; cortical signs (aphasia, neglect, gaze deviation); hemianopia; dense hemiparesis; new AF on ECG	Fluctuating symptoms; non-anatomic pattern; inconsistent exam	ABCs → glucose → SpO_2 → **NCCT ≤20 min;** ECG; labs
Hypoglycemia	Deficit persists after glucose correction	Rapid resolution with IV/PO glucose; low capillary glucose	Check glucose immediately; treat and reassess
Electrolyte/ metabolic	Clear focal cortical signs; normal labs	Global confusion, asterixis, myoclonus; abnormal Na/Ca, uremia/hepatic failure	Basic labs; correct derangement; re-evaluate neuro exam
Seizure → Todd's paralysis	No seizure witnessed; persistent fixed deficit; cortical signs without post-ictal state	Witnessed seizure; post-ictal confusion; deficit resolves <24 h	Glucose, ECG, **NCCT;** consider EEG if uncertain
Migraine with aura	Negative symptoms (loss) sudden at onset; no prior migraine history; high vascular risk	Gradual "march" of positive symptoms (scintillations, tingling), headache follows	**NCCT** if first/atypical aura; treat migraine; counsel
Brain tumor	Hyperacute onset; papilledema uncommon in hyperacute stroke	Weeks–months progression, seizures, raised ICP symptoms	**NCCT/MRI;** urgent neuro referral if mass suspected
Bell's palsy (peripheral VII)	Lower face weakness **sparing** forehead (UMN pattern); limb/ cortical signs	Complete unilateral facial weakness **including** forehead; no limb signs	Eye care, steroids ± antivirals per protocol; safety-net
Conversion/ functional	Consistent neuroanatomical deficits; objective imaging findings	Inconsistent effort (e.g., Hoover sign), non-dermatomal sensory loss, midline splitting	Exclude stroke (glucose, **NCCT**); reassure, document
Chronic subdural hematoma	Truly sudden focal deficit with trauma absent	Older age/anticoagulant use; subacute headache; fluctuating deficits	**NCCT** (crescent collection); reverse anticoagulation if needed
CNS infection (encephalitis/ meningitis)	Pure focal deficit without fever/meningism	Fever, neck stiffness, altered sensorium, seizures, diffuse deficits	Sepsis bundle; **NCCT** if focal signs → LP when safe; start empiric antimicrobials

MANAGEMENT PRINCIPLES

Management of stroke rests on a few core principles.

- **Time is brain**: Minimize door-to-imaging and door-to-treatment intervals; delays cause irreversible neuronal loss.
- **Confirm stroke type before treatment:** Differentiate ischemic vs hemorrhagic stroke on imaging before giving antithrombotics.
- **Optimize vital parameters:** Maintain oxygenation, correct hypoglycemia, and carefully manage blood pressure—avoid aggressive overcorrection.
- **Prevent early complications:** Begin aspiration precautions, DVT prophylaxis, pressure sore prevention, and infection control; encourage safe early mobilization.
- **Multidisciplinary coordination:** Neurology, emergency, cardiology, rehabilitation, nursing, and allied teams should work together from the outset to align acute care, secondary prevention, and recovery.

MANAGEMENT GOALS

- **In ischemic stroke:** restore or preserve cerebral perfusion (e.g., thrombolysis, thrombectomy).
- **In hemorrhagic stroke:** limit hematoma expansion and manage intracranial pressure.
- In all cases: prevent recurrence, reduce mortality, preserve neurologic function, and maximize independence through rehabilitation and tailored secondary prevention.

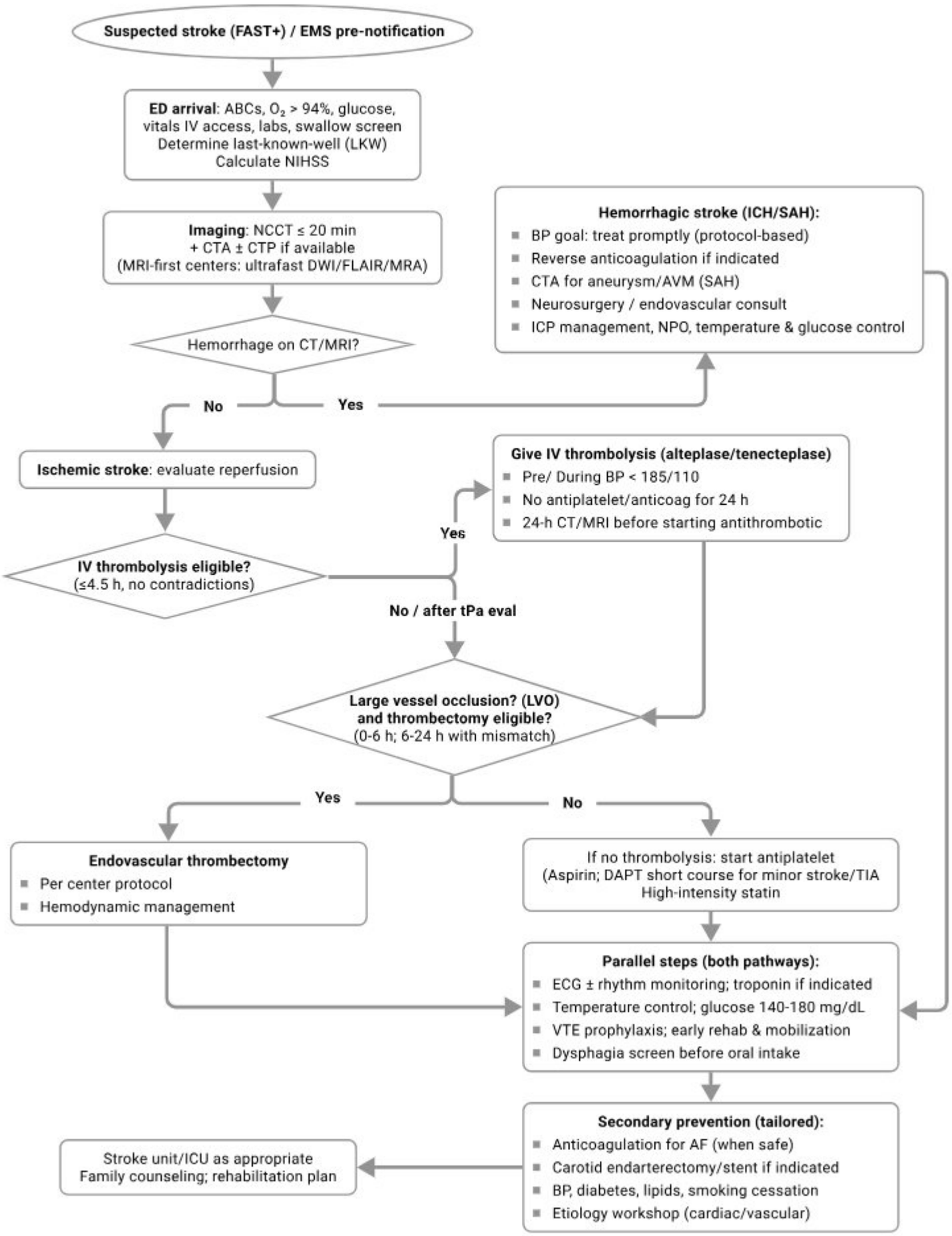
SEVERITY ASSESSMENT – NIH STROKE SCALE (NIHSS)

Standardized tool (0–42) assessing consciousness, gaze, vision, motor strength, coordination, speech, language, and sensation.

Early NIHSS scoring supports:

- Confirming severity
- Guiding reperfusion eligibility (IV thrombolysis, thrombectomy)
- Predicting prognosis and complications (e.g., hemorrhagic transformation)
- Establishing a baseline for monitoring progression or recovery.

Management algorithm for stroke management



Preferred facility/type for management:

- Center with immediate access to non-contrast CT head.
- Established acute stroke protocol (including rapid triage, thrombolysis capability).
- Stroke-trained team or primary stroke center for initial care and stabilization.
- Clear transfer pathway to comprehensive/tertiary stroke center for:
  - Endovascular therapy (thrombectomy)
  - Neurosurgical intervention
  - Multidisciplinary stroke unit care
  - Advanced etiologic workup and rehabilitation planning

STROKE CARE BUNDLE (0–24 HOURS)

1. Pre-hospital

- FAST positive → EMS pre-notify stroke team; record last-known-well (LKW).
- Check capillary glucose, vitals; give O_2 only if SpO_2 <94%.
- Triage to thrombectomy-capable center if suspected LVO and transport times allow.
- For transfer hospitals, aim door-in-door-out ≤60 min. (www.heart.org, American Heart Association Journals)

2. ED arrival: first 10–20 minutes

- ABCs, two IV lines, labs (CBC, CMP, PT/INR, troponin if indicated), ECG; NIHSS.
- Non-contrast CT head ≤20 min from arrival; add CTA ± CTP if available.
- Keep BP permissive until imaging (unless extremes or tPA planned), treat hypoglycemia, maintain SpO_2 ≥94%. (www.heart.org)

3. Ischemic stroke pathway (no hemorrhage on CT/MRI)

- IV thrombolysis: evaluate eligibility; door-to-needle ≤60 min.
  - Lower BP to ≤185/110 before thrombolysis; maintain ≤180/105 for 24 h after.
  - No antiplatelet/anticoagulant for 24 h; repeat brain imaging at 24 h. (www.heart.org, American Heart Association Journals)

- Endovascular thrombectomy (EVT): for LVO within 0–6 h, and 6–24 h in selected patients using perfusion/clinical mismatch. Expedite vascular imaging and team activation.
- If not receiving reperfusion: start aspirin when safe (typically after hemorrhage excluded), start high-intensity statin, manage BP and risk factors per protocol. (American Heart Association Journals)

4. Hemorrhagic stroke pathway (ICH/SAH)

- Rapid BP lowering with protocolized IV agents; typical ICH target SBP ~140–160 early (per guideline and local protocol).
- Reverse anticoagulation/antiplatelet effects as indicated.
- Neurosurgery/endovascular consult; consider CTA to evaluate aneurysm/AVM in SAH.
- ICP measures head-of-bed elevation, temperature and glucose control. (American Heart Association Journals, www.heart.org, cpr.heart.org)

5. Universal nursing/medical bundle (applies to all stroke)

- Swallow screen before any oral intake; keep NPO until passed.
- Temperature: treat fever; Glucose: target moderate control (avoid <67 mg/dL / 3.7 mmol/L).
- VTE prophylaxis per pathway; early mobilization and rehab once safe.
- Dysphagia, fever, hyperglycemia care are core nurse-led elements (QASC). (professional.heart.org, PMC, European Stroke Organisation)

6. Secondary prevention (start during admission)

- Determine etiology (TOAST): cardioembolic vs large-artery vs small-vessel.
- Anticoagulation for atrial fibrillation (timing per infarct size/hemorrhagic risk).
- Carotid revascularization if indicated; control BP, lipids, diabetes, stop tobacco; sleep apnea screening. (American Heart Association Journals)

7. Time targets summary

- **Door-to-imaging (NCCT) ≤ 20 min.**
- **Door-to-needle (IV thrombolysis) ≤ 60 min.**
- **Transfer hospitals (Door In Door Out): DIDO ≤ 60 min.**

- **Endovascular treatment (EVT):** within window (0–6 h; **6–24 h** when imaging shows mismatch). (www.heart.org, American Heart Association Journals, NEJM)

See section on referral criteria and transport of patients

ICU/STROKE UNIT CARE FOR ACUTE STROKE

Vital parameter targets

- Temperature: Aim normothermia (36.5–37.5°C).
- Treat fever (>37.8°C) promptly—prefer acetaminophen and investigate infection source. Fever worsens ischemic injury.

Blood glucose:

- Maintain 140–180 mg/dL in most acute stroke patients.
- Avoid hypoglycemia (<70 mg/dL) and rapid swings; use insulin infusion protocols if persistent hyperglycemia.
- Monitor every 1–2 hours initially.

Blood pressure:

- Ischemic stroke without reperfusion therapy: tolerate SBP up to 220 mmHg unless other indications; only treat if extremely high or comorbid (e.g., aortic dissection, myocardial ischemia).
- Ischemic stroke with thrombolysis or thrombectomy candidate: lower to <185/110 mmHg before therapy and maintain <180/105 mmHg afterward.
- Hemorrhagic stroke: target SBP generally <140 mmHg (individualize), using titratable IV agents (nicardipine, labetalol).
- Continuous or frequent BP monitoring.

Do	Don't
Establish and secure airway early if consciousness is reduced or airway protection is compromised.	Do not aggressively lower BP in acute ischemic stroke unless clearly indicated (e.g., >220/120 mmHg or >185/110 mmHg if thrombolysis/thrombectomy candidate).
Use standardized stroke scales (e.g., NIHSS) serially to monitor changes.	Do not give antiplatelets or anticoagulants before excluding hemorrhage on imaging.
Initiate DVT prophylaxis (mechanical early; pharmacologic when safe).	Do not delay reperfusion therapy for nonessential imaging or lab tests.
Conduct a swallow assessment before any oral intake.	Do not start enteral feeds before confirming safe swallowing.
Maintain euvolemia with appropriate fluids.	Avoid aggressive fluid overload or depletion.
Provide oxygen if SpO_2 <94%.	Do not give routine oxygen if SpO_2 ≥94%.
Monitor and control glucose (target 140–180 mg/dL).	Do not rapidly overcorrect glucose levels (risk of injury).
Implement early mobilization protocols once clinically stable (often 24–48 h).	Avoid very early/aggressive mobilization in severe/ unstable stroke.
Start secondary prevention workup and management promptly.	—

General ICU vigilance

- Continuous cardiac monitoring for arrhythmias, especially new atrial fibrillation.
- Regular neurologic checks and sedation minimization to allow assessment.
- Prevent and treat infections early (UTI, pneumonia).
- Pressure injury prevention: repositioning, skin care.

PHARMACOLOGICAL THERAPY

Intravenous Thrombolysis in Acute Ischemic Stroke

Aspect	Key Points
Indications	■ Clinical diagnosis of acute ischemic stroke with measurable deficit ■ Onset/last known well ≤4.5 hours ■ No hemorrhage on NCCT ■ Blood glucose >50 mg/dL ■ BP ≤185/110 mmHg before treatment

Exclusions	■ Active internal bleeding or recent (<3 months) intracranial hemorrhage ■ Major surgery/serious trauma within 14 days ■ Platelet <100,000, INR >1.7, or known coagulopathy ■ Intracranial surgery in past 3 months ■ Uncontrolled severe hypertension despite treatment ■ GI/urinary bleeding within 21 days (context-specific) ■ Known intracranial neoplasm, AVM, or aneurysm (unless excluded)
Time Targets	■ Door-to-needle ≤60 min ■ Best outcomes with earlier treatment ■ Window: ≤4.5 hours (extended criteria only under trial/protocols)
Drug & Dosing	Alteplase (tPA): 0.9 mg/kg (max 90 mg); 10% IV bolus over 1 min, rest over 60 min Or Tenecteplase (emerging/selected protocols): 0.25 mg/kg IV single bolus (some studies 0.4 mg/kg); follow local guidance (off-label in many regions)
Monitoring	■ Continuous neurologic checks & BP monitoring (<180/105 mmHg for at least 24 h) during and post-infusion - Watch for hemorrhagic transformation or angioedema, reperfusion arrhythmias, maintain normothermia, normoglycemia and euvolemia ■ Repeat CT/MRI at 24 h before starting antiplatelet/anticoagulant

POST STROKE MONITORING:

Cautions and Post-Stroke Monitoring after thrombolysis:

Domain	Key Points
Cautions (Pre/Post Thrombolysis)	■ Recent DOAC use (<48 h) without reliable assay may preclude use ■ Mild/rapidly improving symptoms: treat only if deficit disabling ■ Control BP before and for ≥24 h post-thrombolysis (≤180/105 mmHg) ■ Watch for hemorrhagic transformation → sudden neurologic worsening → immediate CT/MRI ■ Avoid invasive procedures (NG tube, catheterization) during infusion unless essential
Post-Thrombolysis Monitoring	■ Neuro checks: every 15 min in first hour, then per protocol (e.g., q30 min for 6h, then hourly for 16h) ■ Strict BP monitoring and control Immediate imaging if deterioration occurs ■ Repeat CT/MRI at 24 h before starting antithrombotic therapy
Antiplatelet Therapy	■ If **no thrombolysis:** Aspirin 150–300 mg loading within 24–48 h, then 75–100 mg daily long term **If minor stroke/high-risk TIA**: Dual therapy (Aspirin + Clopidogrel) for 21 days, then single agent

Anticoagulation	■ For cardioembolic source (e.g., AF) after stabilization Timing depends on infarct size and bleeding risk (commonly 1–2 weeks after large infarct)

Acute ischemic stroke blood pressure management

In acute ischemic stroke, blood pressure is managed differently based on treatment pathway. If the patient is a candidate for reperfusion (thrombolysis or thrombectomy), lower BP to ≤185/110 mmHg before treatment and maintain <180/105 mmHg for at least 24 hours afterward.

In patients not receiving reperfusion, permissive hypertension is allowed, avoid treating unless systolic BP exceeds ~220 mmHg or there are other indications (e.g., acute coronary syndrome, aortic dissection).

Blood pressure should be lowered cautiously when treatment is needed: target a modest reduction (e.g., 15% in the first 24 hours) using titratable IV agents such as labetalol or nicardipine.

Clinical Scenario	BP Targets	Approach	IV Agents & Dosing	Cautions
Candidate for reperfusion (IV thrombolysis or thrombectomy)	Lower to ≤185/110 mmHg before treatment; maintain <180/105 mmHg for ≥24 h after	Active BP lowering with IV agents before reperfusion therapy	■ Labetalol: 10–20 mg IV over 1–2 min; may repeat q10–20 min (max 150 mg). Infusion 0.5–2 mg/min if needed ■ Nicardipine infusion: Start 5 mg/h; ↑ by 2.5 mg/h q5–15 min; usual 5–15 mg/h ■ Clevidipine (If available): Start 1–2 mg/h; double q90 sec; usual 4–6 mg/h; max ~21 mg/h	Avoid overshoot; monitor q5–15 min; maintain perfusion
Not receiving reperfusion therapy	Permissive hypertension allowed. Do not lower unless SBP >220 mmHg or comorbidity present (ACS, aortic dissection, hypertensive encephalopathy)	Cautious BP lowering only if indicated	■ Same IV options as above ■ Hydralazine (less preferred): 5–10 mg IV q4–6h; variable effect	Reduce BP gradually (≤15% in first 24 h); monitor closely
Special considerations	Large hemispheric or cerebellar infarcts → neurosurgical referral for decompression	—	—	—

Cautions: avoid rapid or excessive lowering; aim for controlled reduction. Monitor BP closely (every 15 minutes during active lowering) and adjust infusion rates to avoid compromising penumbral perfusion. Alert neurosurgical referral for large and cerebellar infarct for decompression.

HEMORRHAGIC STROKE

Acute management of hemorrhagic stroke focuses on limiting hematoma expansion and stabilizing physiology.

Intracranial hemorrhage supportive care bundle

Domain	Key Measures	Drugs / Protocols	Cautions
Intracranial Pressure (ICP) Management	■ Head elevation (30°) ■ Sedation for agitation ■ Osmotherapy: mannitol or hypertonic saline ■ Avoid hypotonic fluids	■ Mannitol: 0.25–1 g/kg IV bolus (repeat per ICP/serum osmolality) ■ Hypertonic saline: titrate to serum Na target (per protocol)	Invasive ICP monitoring in select cases (large ICH, low GCS). Monitor Na/osmolality to prevent complications
Blood Pressure Control	■ Target SBP <140 mmHg if presenting with elevated pressure (unless contraindicated)	■ Labetalol: 10–20 mg IV bolus; repeat q10–20 min (max ~150 mg); or infusion 0.5–2 mg/min ■ Nicardipine infusion: Start 5 mg/h, ↑ by 2.5 mg/h q5–15 min (usual 5–15 mg/h)	Avoid rapid excessive lowering → risk of hypoperfusion; monitor BP q5–15 min during titration
Coagulopathy Correction	■ Reverse anticoagulant effect rapidly; maintain hemostasis	■ Warfarin: Vitamin K 5–10 mg IV + PCC 25–50 IU/kg (dose by INR/weight) ■ FFP if PCC unavailable ■ Dabigatran: Idarucizumab 5 g IV (two 2.5 g doses) ■ Factor Xa inhibitors: Andexanet alfa per protocol; if unavailable, PCC (less effective)	Coordinate with hematology/ critical care; verify INR or DOAC use if assays unavailable
Neurosurgical Intervention	■ Evacuate accessible lobar hematomas with mass effect or deteriorating status ■ Decompressive craniectomy for refractory ICP or large cerebellar bleed with brainstem compression ■ External ventricular drain (EVD) for hydrocephalus due to intraventricular extension	—	Decision individualized; early neurosurgery consult essential

Vascular Lesion Treatment	■ SAH: urgent aneurysm repair (clipping or endovascular coiling) ■ AVM/vascular malformation: definitive treatment via embolization, surgery, or radiosurgery depending on size/location	—	Timing and modality based on stability and local expertise
Supportive Care	■ Maintain normothermia ■ Moderate glucose control (avoid extremes) ■ Seizure treatment if occurs ■ Prevent complications (DVT, aspiration, infections) ■ Early rehabilitation initiation	■ Anticonvulsants if seizure: Levetiracetam or phenytoin	Routine seizure prophylaxis not recommended; treat only if seizures occur
Etiologic Workup	■ Identify cause and recurrence risk	■ Hypertension control ■ Vascular imaging for aneurysm/AVM ■ Consider amyloid angiopathy in lobar hemorrhage	Guides long-term prevention and counseling

TRANSIENT ISCHEMIC ATTACK (TIA)

Transient ischemic attack (TIA) is a transient episode of neurological dysfunction caused by focal cerebral, spinal cord, or retinal ischemia without acute infarction on imaging. Symptoms usually resolve within minutes to a few hours; by definition, they last less than 24 hours.

It signals high short-term risk of stroke—up to 10–20% within 90 days, with the highest concentration in the first 48 hours. Early diagnosis, rapid risk stratification, targeted dual antiplatelet therapy in high-risk cases, timely vascular and cardiac evaluation, aggressive risk factor modification, and appropriate disposition reduce the likelihood of progression to major ischemic stroke.

Risk stratification:

Tool	Components	Scoring / Cut-offs	Interpretation	Remarks
ABCD2 Score	■ Age ≥60 (1) ■ Blood pressure ≥140/90 (1) ■ Clinical features: unilateral weakness (2), speech disturbance w/o weakness (1) ■ Duration: ≥60 min (2), 10–59 min (1) ■ Diabetes (1)	0–7 points	0–3 = low risk 4–5 = moderate risk 6–7 = high risk of stroke within 2–7 days	Widely used; simple bedside tool; higher scores (≥4) → admit or urgent workup
ABCD3 Score	■ ABCD2 + Dual TIA (recurrent within 7 days) (2 points)	0–9 points	Higher score = greater early recurrence risk	Improves prediction over ABCD2, esp. for crescendo TIAs
ABCD3-I Score	ABCD3 + Imaging findings: ■ Acute DWI lesion (2) ■ Carotid stenosis ≥50% (2)	0–13 points	≥8 = high risk of early stroke	Most accurate; requires MRI-DWI and carotid imaging
Imaging Integration	■ MRI-DWI, carotid Doppler/ CTA/MRA	-	Clinical score + imaging = best predictive accuracy	Supports early intervention (antiplatelet, revascularization, admission decisions)

1. Immediate actions (treat as stroke alert):

2. Early medical therapy

Risk Category	Criteria	Recommended Therapy	Duration
High-Risk TIA / Minor Ischemic Stroke	■ ABCD2 ≥4 ■ Imaging evidence of infarction ■ Crescendo/recurrent TIAs	Dual Antiplatelet Therapy (DAPT) to start immediately after symptom onset (ideally within 12-24 h): ■ Aspirin 75–100 mg daily ■ Clopidogrel: 300 mg loading dose (some protocols allow 600 mg if very early/high risk), then 75 mg daily	Continue DAPT for 21 days, then switch to single antiplatelet (usually clopidogrel 75 mg daily or aspirin 75–100 mg daily)
Low-Risk TIA	■ ABCD2 ≤3 ■ No acute infarct on imaging ■ No crescendo attacks	Single Antiplatelet: ■ Aspirin 75–100 mg daily OR ■ Clopidogrel 75 mg daily	Long-term monotherapy; confirm no contraindication on imaging (e.g., hemorrhage)

Cardioembolic evaluation:

- Continuous or extended rhythm monitoring if initial ECG is nondiagnostic to detect paroxysmal atrial fibrillation.
- Echocardiography when indicated to identify sources like left ventricular thrombus, valvular disease, or patent foramen ovale.
- If a cardioembolic source is confirmed (e.g., atrial fibrillation), initiate anticoagulation per risk-benefit assessment after excluding contraindications.

Vascular causes:

- Evaluate for carotid disease in anterior circulation TIAs. Symptomatic high-grade carotid stenosis (typically ≥70%) warrants expedited referral for carotid endarterectomy or stenting, ideally within two weeks if surgical risk acceptable.

Risk factor control:

- Start or optimize control of hypertension, diabetes, and dyslipidemia (high-intensity statin irrespective of baseline LDL in atherosclerotic risk).
- Counsel and support tobacco cessation, healthy diet, physical activity, and weight management.

Disposition:

- Admit or urgent observation for high-risk patients: ABCD2 ≥4, crescendo TIAs (multiple in short interval), atrial fibrillation, significant carotid stenosis, evidence of infarction on imaging, or diagnostic uncertainty.
- Expedited outpatient workup (within 24–48 hours) may be acceptable for low-risk patients with reliable follow-up and no concerning features.

Secondary Prevention

- Blood pressure: target <130/80 mmHg (at least <140/90 if tighter control not tolerated). Use agents like ACE inhibitors/ARBs (e.g., lisinopril 10–20 mg once daily or losartan 50–100 mg once daily), thiazide diuretics (e.g., hydrochlorothiazide 12.5–25 mg daily), or calcium-channel blockers. Strict control reduces both ischemic and hemorrhagic recurrence.
- Lipids: high-intensity statin for atherosclerotic ischemic stroke regardless of baseline LDL. Example: atorvastatin 40–80 mg daily or rosuvastatin 20–40 mg daily.

ANTITHROMBOTIC THERAPY

Scenario	What to start	When to start	Key doses / notes
Non-cardioembolic ischemic stroke / TIA	Single antiplatelet: aspirin 75–100 mg daily OR clopidogrel 75 mg daily	After imaging excludes hemorrhage. If IV alteplase was given, wait ≥24 h and a clear CT/MRI before any antithrombotic. (AHA Journals)	Long-term monotherapy for most. (AHA Journals)
High-risk TIA / minor stroke (e.g., ABCD2 ≥4, DWI+ lesion, crescendo TIAs)	Short-term DAPT: aspirin 75–100 mg daily + clopidogrel 300 mg load (some use 600 mg if very early/high risk) → 75 mg daily	Start as early as possible once hemorrhage excluded (and not within 24 h after IVT). Stop at 21 days, then continue single agent. (AHA Journals, PMC)	DAPT not for long-term secondary prevention. (AHA Journals)
Cardioembolic stroke (atrial fibrillation, etc.)	Oral anticoagulant: DOAC preferred or warfarin (INR 2.0–3.0)	Timing by infarct size/ severity. Common practice: "1–3–6–12 days" (TIA/small/ moderate/large). ELAN supports starting earlier (0–2–6–12 days) in imaging-selected patients. Avoid if hemorrhagic transformation or uncontrolled BP. (New England Journal of Medicine, American College of Cardiology)	Apixaban 5 mg BID (reduce to 2.5 mg BID if ≥2: age ≥80, weight ≤60 kg, creatinine ≥1.5 mg/dL). Rivaroxaban 20 mg QPM with food (CrCl ≤50 → 15 mg QPM). Warfarin per INR.
After IV alteplase (IVT)	—	No antiplatelet/anticoagulant for the first 24 h; obtain repeat CT/MRI before starting. (AHA Journals, www.stroke.org)	Maintain BP per IVT protocol.
After EVT without IVT	Usually aspirin once post-procedure imaging excludes hemorrhage (center protocol)	Often by 24 h post-EVT if scans are clean	Combine with anticoagulation later only if indicated (e.g., AF).
Post-hemorrhagic stroke (ICH/SAH)	Avoid antithrombotics acutely unless a compelling indication exists	Restart is individualized after hematoma stability, etiology/ addressing bleed risk, and specialty input	Multidisciplinary decision (stroke, neurosurgery, hematology).
Diabetes (risk factor)	Glycemic control (target ~7% A1c, individualized); metformin first-line if not contraindicated	Start/adjust during admission and at discharge	Part of secondary prevention bundle. (AHA Journals)
Lifestyle	Stop smoking; Mediterranean/ DASH diet; 150 min/week moderate activity; weight/BMI goals; limit alcohol	Begin in-hospital; reinforce at discharge	Core to all secondary prevention. (AHA Journals)
Other interventions	Carotid stenosis (symptomatic ≥70%): CEA/ CAS; AF detection (longer rhythm monitoring); sleep apnea screen; PFO assessment in select patients	As soon as feasible (CEA ideally within 2 weeks of symptoms when indicated)	Use DSA when noninvasive vascular imaging is inconclusive or to plan endo(AHA Journals)

Other interventions: Digital Subtraction Angiography (DSA) and stenting, indication

Intervention	When / Indication	Key points & Timing	Peri-procedure meds / Follow-up
DSA (Digital Subtraction Angiography)	Noninvasive imaging (CTA = CT angiography, MRA = MR angiography, Doppler) is discordant/indeterminate; need high-resolution vessel detail for therapy planning (aneurysm, arteriovenous malformation [AVM], carotid/ intracranial stenosis); suspected dissection/ vasculitis/RCVS not confirmed on CTA/MRA	Gold standard lumen imaging; allows diagnosis and treatment in one setting (coiling, embolization, stenting)	Contrast exposure—optimize hydration and renal protection; hold metformin if AKI risk per local policy
Carotid revascularization (symptomatic stenosis)	≥70% NASCET symptomatic stenosis: carotid endarterectomy (CEA) preferred if surgical risk acceptable; carotid artery stenting (CAS) if high surgical/anatomical risk for CEA (hostile neck, prior CEA restenosis, radiation, tracheostomy), or unfavorable anesthesia risk	Aim within 14 days of TIA/ minor stroke when feasible; consider CEA for 50–69% case-by-case; older patients (>70) tend to benefit more from CEA than CAS	CEA: single antiplatelet. CAS: dual antiplatelet (aspirin + clopidogrel) before and ≥1–3 months after, then single agent; continue high-intensity statin and risk-factor control
Intracranial atherosclerotic disease	Symptomatic 70–99% intracranial stenosis	First-line: aggressive medical therapy (antiplatelet, statin, BP, lifestyle). Stenting only in highly selected cases or trials after failure of best medical therapy	Close follow-up for recurrence; avoid routine stenting given procedural stroke risk
Atrial fibrillation (AF) screening	Ischemic stroke/TIA without known AF (especially embolic pattern)	Telemetry 24–72 h in-hospital; extended ambulatory monitoring (patch 2–4 weeks) or ILR (implantable loop recorder) if cryptogenic	If AF detected → anticoagulation plan (timing by infarct size/bleed risk)
Sleep apnea (OSA) evaluation	Snoring, witnessed apneas, daytime sleepiness, resistant hypertension, obesity	Screen (e.g., STOP-BANG), confirm with home sleep apnea test or polysomnography; start CPAP if indicated	CPAP improves BP/sleep quality; reinforce adherence and weight management
PFO (Patent Foramen Ovale) management	Cryptogenic embolic stroke, age 18–60, no alternative cause, high-risk PFO features (large shunt/atrial septal aneurysm)	Multidisciplinary decision (stroke + cardiology). Closure favored in suitable patients; otherwise antiplatelet/ anticoagulation as indicated	Post-closure antiplatelet per protocol; endocarditis prophylaxis only if specified by local guidance
General risk-factor control & adherence	Applies to all ischemic mechanisms	Tight BP, lipids, diabetes, smoking cessation; Mediterranean/DASH diet; exercise. Pharmacy reconciliation, pill organizers, reminders, caregiver engagement	Set review dates; monitor adherence and side effects; escalate therapy if targets unmet

NON-PHARMACOLOGICAL INTERVENTIONS

Non-pharmacological care begins early—often within the acute or subacute phase—and directly influences recovery, complication prevention, and long-term function.

Bundle Element	What to do	Start / Withhold	Who	Targets
Early rehabilitation	Start physiotherapy (PT) for motor recovery, balance, tone, gait with task-specific practice and progressive resistance. Occupational therapy (OT) for ADLs, upper-limb function, adaptive techniques, energy conservation. Speech & language therapy (SLT) for aphasia/dysarthria; communication strategies; swallowing training.	Begin once medically stable; aim within 24–48 h. Withhold if unstable intracranial pressure, active hemorrhage expansion, or uncontrolled BP/O_2/ glucose.	PT, OT, SLT, stroke nurse, physician	Early rehab reduces deconditioning, pneumonia, and deep vein thrombosis (DVT). Set short, functional goals and reassess daily.
Swallow assessment	Formal bedside screen before any oral intake (food, fluids, meds). If failed → nil per os (NPO), SLT assessment, alternative nutrition. Use texture modification, postural adjustments, and swallowing exercises when appropriate.	On admission/ arrival to ward and after any neurological change.	SLT, trained nurse	Prevents aspiration pneumonia. Re-screen after interventions; document safe consistencies.
DVT prophylaxis (mechanical first)	Intermittent pneumatic compression (IPC) or graduated compression stockings unless contraindicated. Early mobilization plan (bed ↔ chair, standing, ambulation).	Start immediately unless limb contraindications. Pharmacologic prophylaxis (LMWH/UFH) only when imaging and clinical judgment deem hemorrhagic risk acceptable (often delayed in large infarcts or recent bleed).	Nursing, PT, physician	Document mobility level daily; track IPC hours/day. *(LMWH = low-molecular-weight heparin; UFH = unfractionated heparin).*
Nutritional support & hydration	Screen nutrition early; calculate protein/calorie needs. If unsafe oral intake → enteral feeding (NG/PEG) per plan. Monitor fluids closely; avoid over/under-resuscitation. Watch for refeeding syndrome in malnourished patients; add micronutrients as needed.	Within 24 h of	-	-

These interventions should be coordinated through a multidisciplinary stroke team, with regular reassessment to adjust intensity, prevent complications, and set incremental functional goals.

ASSESSMENT OF RESPONSE

Assess response with structured neurologic and functional monitoring.

Area	Measure	When	Response/Target	Action if Off-Target	Level of Care
Neurologic status	NIHSS change; consciousness; new deficits	0–24 h (q15–60 min as per protocol), 24–48 h, daily	Stable or improving; ≥4-point rise = red flag	Urgent CT/MRI; check glucose, BP; consider hemorrhagic transformation or edema	All (tele-consult if needed)
Imaging response	24-h CT/MRI after IV thrombolysis/ endovascular therapy; stability after hemorrhage	24 h; earlier if deterioration	No new bleed; recanalization if treated; stable hematoma size	Escalate to stroke/ neurosurgery; adjust antithrombotic plan	Secondary/ Tertiary
Reperfusion therapy	Door-to-needle; door-to-groin times; protocol adherence	Acute phase	Met time targets; no major complications	Case review; workflow fixes; re-train team	Secondary/ Tertiary
Blood pressure (BP)	SBP/DBP trends	Hourly initially; then q4–6 h	Ischemic: ≤185/110 before IVT, <180/105 ×24 h after; No IVT: treat if SBP >220 mmHg or comorbidity. ICH (intracerebral hemorrhage): SBP ~140–160	Titrate IV labetalol/ nicardipine; avoid rapid drops; review fluids/analgesia	All
Glucose	Capillary glucose	0–24 h q4–6 h; then per status	140–180 mg/dL; no hypoglycemia	Adjust insulin protocol; review feeds/renal function	All
Oxygenation/ temperature	SpO_2; temperature	Continuous SpO_2; temp q4–6 h	SpO_2 ≥94%; normothermia	Oxygen if <94%; antipyretics; search infection	All
Swallow & nutrition	Bedside screen; SLT (Speech & Language Therapy) assessment; intake	On arrival to ward; re-screen after change	Safe oral consistency confirmed; adequate kcal/ protein	Keep NPO; start NG feeds; SLT plan; prevent aspiration	All (SLT via tele-consult in atolls)
DVT (deep vein thrombosis) prevention	Intermittent pneumatic compression use; chemoprophylaxis timing	Day 0–1; daily	IPC used; LMWH/UFH when safe	Start IPC; start chemoprophylaxis when bleed risk acceptable	All
Mobility/rehab	PT/OT (Physio/ Occupational Therapy) milestones: bed→sit→stand →walk	Daily	Stepwise progression; no falls	Adjust plan; address orthostasis, vision, balance; add aids	All

Antithrombotic plan (ischemic)	Antiplatelet start time; DAPT (dual antiplatelet therapy) for minor stroke/ high-risk TIA; anticoagulation timing if AF (atrial fibrillation)	24–48 h; at discharge	Started after hemorrhage excluded; DAPT 21 days when indicated; anticoagulation per size/timing	Delay/withhold if bleed risk; re-image; set firm start/stop dates	All
Anticoagulation (cardioembolic)	Start date; agent/dose; adherence	1–2 weeks (large infarct may be later)	Started per protocol; no bleed	Reassess imaging/BP; bridge plan; educate patient	Secondary/ Tertiary
ICH care bundle	SBP control; reversal complete; ICP (intracranial pressure) measures; EVD (external ventricular drain) function	Hourly early; daily	Targets met; stable hematoma; EVD patent	Re-bolus reversal; adjust drips; neurosurgery review	Secondary/ Tertiary
Vascular workup	Carotid imaging; intracranial stenosis; dissection; AF monitoring	Inpatient or ≤2 weeks	Etiology defined; plan set (CEA/CAS if symptomatic ≥70%)	Escalate imaging (CTA/ MRA/DSA); schedule surgery	Secondary/ Tertiary
Complication surveillance	Aspiration pneumonia, UTI, pressure injuries, seizures, depression	Daily	None, or early detection	Treat promptly; preventives reinforced	All
Risk-factor control	BP, LDL-C, HbA1c, smoking/ alcohol, diet/ activity	At discharge; 4–12 weeks	Targets set: statin, BP meds, diabetes plan, lifestyle	Intensify therapy; refer to NCD clinic; community follow-up	All
Education & adherence	Teach meds, warning signs, follow-ups; caregiver training	Before discharge	Teach-back successful; clear plan	Re-educate; written plan in Dhivehi/English; phone reminders	All
Follow-up & referrals	Stroke clinic/ review dates; rehab slots; community health link	Book before discharge			

- In the acute phase, perform serial NIHSS (or bedside equivalent) assessments - every 1–2 hours for the first 6 hours, then every 4–6 hours until stable—to detect improvement or deterioration; any drop of ≥4 points warrant urgent reevaluation and imaging.

- Assess functionality using modified Rankin Scale, Barthel index: modified Rankin Scale (mRS) measures global disability on a 0–6 scale (0 no symptoms, 6 dead); it's used to categorize independence and recovery trajectory. Barthel Index quantifies activities of daily living (0–100) across feeding, mobility, toileting, grooming, etc.; higher scores mean greater independence. Assess both at discharge to establish baseline and again at scheduled follow-ups (typically 1 month, 3–6 months) to gauge recovery, rehabilitation effectiveness, and need for support or adjustment in care plans. Significant change guides goal setting and referral intensity.
- Reimage if there is clinical worsening. Track vital sign trends (BP target <130/80 long term, acute thresholds per protocol; glucose 140–180 mg/dL in hospital) and confirm adherence to secondary prevention medications.
- Initial follow-up occurs within 1–4 weeks to reassess risk factor control, functional status, medication compliance, screen for depression, and perform cognitive evaluation if indicated.
- Ongoing visit: Revisit every 3–6 months for optimization, re-evaluate anticoagulation if new arrhythmias appear, and consider repeat vascular imaging for recurrent or unexplained symptoms.
- Before modifying therapy (step-up or step-down), ensure blood pressure and glucose are controlled, rule out new deficits or evolving infarcts, reassess swallowing and mobility support, and screen for common post-stroke complications such as infection, seizures, or mood disturbances.

PROGNOSIS

Stroke outcome varies widely and depends on subtype, initial severity, patient factors, and timeliness/quality of care.

Predictors of Poor Outcome

High baseline NIHSS, older age, large infarct/hematoma, reduced consciousness, comorbidities (HTN, diabetes, AF, cardiac/renal disease), and complications (pneumonia, DVT, recurrent stroke).

Ischemic vs Hemorrhagic Stroke

- Ischemic: Prognosis improves with early reperfusion (thrombolysis, thrombectomy).
- Hemorrhagic: Outcome depends on hematoma size, infratentorial site, intraventricular extension, and raised ICP (ICH score).

Clinical Course

- Early deterioration from infarct growth, edema, hemorrhagic transformation, recurrent embolism, or metabolic derangements.
- Cerebral edema peaks at 3–5 days in large infarcts; hemorrhage often expands in the first hours.

Recovery Phases

- **Acute (days):** stabilization and complication prevention.
- **Subacute (weeks):** active neuroplasticity; rehab yields rapid gains.
- **Chronic (months+):** slower improvements, compensation strategies.

Determinants of Recovery

Lesion site, early/intense rehab, pre-stroke function, and psychosocial support.

Long-Term Risks

Recurrent stroke (highest in 3–6 months), vascular cognitive impairment, depression/anxiety, mobility decline.

Secondary Prevention

BP and lipid control, tailored antithrombotic therapy, diabetes management, lifestyle change, and treatment of cardiac/vascular sources reduce recurrence and improve survival.

REFERRAL CRITERIA (TIERED APPROACH)

Primary care:

- Suspected stroke → stabilize airway/breathing/circulation, check glucose and vital signs, note time of onset, and urgently refer to nearest facility with neuroimaging.
- Immediate transfer if consciousness drops, neurologic status worsens, or local triage is uncertain.

Secondary care:

Refer to tertiary center if any of the following: candidate for thrombolysis or thrombectomy (needs advanced decision support), large territorial or rapidly evolving stroke, unclear etiology requiring comprehensive imaging, worsening hemorrhage, signs suggesting need for neurosurgical intervention, or failure to improve/stabilize.

Tertiary care:

Handle endovascular therapy, surgical decompression, complex etiologic workup (cryptogenic/recurrent strokes), and coordinate multidisciplinary rehabilitation and secondary prevention planning.

Emergency transfer protocol:

- Pre-notify receiving facility with patient identity, time of symptom onset/last known well, stroke type suspicion, current neurologic status (NIHSS if available), vital signs, imaging done (attach/report), interventions given (e.g., thrombolysis), and estimated arrival time.
- Use dedicated transport with monitoring (vitals, neurologic checks) and a concise transfer summary.
- Ensure receiving team has a clear point of contact and stroke pathway activated before arrival.
- Prehospital Stroke Management and Systems of Care Hospital Stroke Teams Timeline & Action
- 10 minutes from arrival or sooner ≤ 15 minutes ≤ 20 minutes ≤ 45 minutes or sooner Evaluation by physician Stroke or neurologic expertise contacted NCCT or MRI Interpretation of neuroimaging ≤ 60 minutes 16 Initiation of IV alteplase

First aid and care during transport:

- Use FAST to recognize stroke and note exact time of symptom onset.
- Keep airway patent; position supine with head midline (slightly elevated if vomiting or signs of raised ICP).
- Do not give food, drink, or oral medications.
- Check and correct blood glucose if hypoglycemia is suspected.

- Maintain oxygen saturation ≥94%; supplement only if below target.
- Monitor vital signs and neurologic status en route; note any deterioration.
- Pre-notify receiving facility with suspected stroke details and time of onset.
- Avoid unnecessary delays or interventions that do not affect immediate stroke care.

COMPLICATIONS

Complication	Key Features	Monitoring/Prevention	Management
Cerebral edema & increased intracranial pressure	Peaks within first days after large infarct/hemorrhage; can cause herniation	Frequent neuro checks, head elevation	Osmotherapy, consider decompressive surgery
Hemorrhagic transformation	Risk after reperfusion or large infarcts; sudden neurologic worsening	Early imaging, BP control, risk stratification	Supportive care, adjust antithrombotics
Seizures	Early (acute injury) or late (gliotic scarring)	Clinical observation, EEG if needed	Treat only if seizures occur; anticonvulsants as indicated
DVT/PE	Immobility increases risk	Early mobilization, compression devices	Mechanical prophylaxis first, pharmacologic once safe
Aspiration pneumonia	Due to dysphagia, impaired consciousness	Swallow screen, oral care, upright positioning	NPO if unsafe, modify diet, antibiotics if infected
Pressure injuries	From immobility	Regular repositioning, skin checks, support surfaces	Local wound care, mobilization
Depression & cognitive impairment	Common, impairs rehab outcomes	Screen early, caregiver input	Psychosocial support, antidepressants, cognitive rehab
Secondary stroke (recurrence)	Highest risk in first year	Control risk factors, adherence to therapy	Antithrombotics, statins, lifestyle modification

PREVENTION AND HEALTH PROMOTION

Prevention and health promotion are the strongest tools to reduce stroke incidence, mortality, and disability.

- **Primary prevention:** Control hypertension, diabetes, dyslipidemia, tobacco, diet, inactivity, and detect atrial fibrillation.
- **Secondary prevention:** Sustain risk factor control, use appropriate antithrombotics, and address underlying causes.
- **Public education** (e.g., FAST) speeds presentation and treatment, reducing complications.

- **System impact:** Prevention lowers healthcare costs, reduces demand for acute/rehab services, and preserves independence.

National strategy (Maldives NCD Action Plan 2023–2031): Integrates BP and diabetes control, tobacco cessation, healthy diet, physical activity, lipid management, and AF screening. Anchored in primary care, community awareness, and equitable access.

Embedding stroke prevention into the NCD framework shifts care from reactive to proactive, reduces recurrence, supports adherence, and makes outcomes measurable at the population level.

PATIENT EDUCATION

Patient education aims to empower survivors and caregivers to improve outcomes. Together, education and caregiver involvement create a safety net that catches deterioration early and sustains long-term prevention.

- Key objectives are early recognition of stroke warning signs and rapid action, understanding why prescribed medications matter and ensuring adherence, and knowing which lifestyle changes (blood pressure control, diet, exercise, smoking cessation) lower recurrence risk.
- Emphasize TIA as a warning sign; any recurrent symptoms require immediate presentation.
- Reinforce medication adherence, lifestyle modification, and awareness of risk factor targets.
- Education also clarifies the follow-up schedule and highlights red flags—new weakness, worsening speech, altered consciousness, fever, or swallowing difficulty—that need prompt attention.
- Engaging caregivers ensure consistent support for rehabilitation, medication routines, mobility assistance, and emotional recovery.

Instructions to Patient/Caregiver

Do's	Don'ts
Learn and act on **FAST** signs (Face droop, Arm weakness, Speech changes, Time to call emergency services).	Do not wait to "see if symptoms improve" — every minute counts.
Call the designated stroke response number immediately if sudden weakness, speech difficulty, vision changes, or severe headache occur.	Don't skip or stop medicines without your doctor's advice.
Follow all prescribed medications exactly (antiplatelets, anticoagulants, blood pressure drugs, statins).	Don't delay or miss rehab — recovery potential is highest in the early months.
Attend all follow-up visits and rehabilitation sessions (physiotherapy, occupational therapy, speech therapy).	Avoid high-salt, high-fat, and highly processed foods that worsen vascular risk.
Keep blood pressure, blood sugar, and cholesterol under control through diet, exercise, and medication.	Don't ignore fall hazards, especially after stroke-related weakness or balance problems.
Maintain a safe home environment — remove trip hazards, use grab bars, ensure good lighting.	Don't give food or drink before a swallow screen if there is any risk of aspiration.
Support good nutrition and hydration; follow swallowing precautions if advised.	Avoid overexertion or unsupervised strenuous activities that could cause injury.
Encourage and assist with daily exercises as guided by therapists.	Don't dismiss emotional changes; post-stroke depression is common and treatable.
Manage mood and emotional health — seek help for depression or anxiety.	Don't assume everyone around knows what to do in an emergency — train family and caregivers.
Keep a list of emergency contacts and medical history handy.	Don't restart smoking or drink excessively — both increase risk of recurrence.
Promote smoke-free living and limit alcohol intake.	-

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