MyCardioAdvocate™

Stroke Prevention

Stroke & Brain Attack Prevention

When cardiovascular disease hides in the brain—and no one takes ownership.

Updated March 2026

Why This Matters

Stroke is the 5th leading cause of death in the US and the #1 cause of long-term disability. Most strokes are ischemic (85%), caused by blockage of a brain artery. This blockage is usually atherosclerotic—the same process that causes heart attacks. Paradoxically, many stroke patients are managed by neurologists, many heart attack survivors by cardiologists, and the two don't talk. Yet a person who has survived a stroke IS a cardiovascular patient and requires aggressive secondary prevention.

The 2026 guideline change is subtle but critical: **Ischemic stroke is now classified as clinical ASCVD.** This means post-stroke patients qualify for the same aggressive lipid targets and therapies as post-MI patients. Yet many aren't on statins, and even fewer are on high-intensity therapy.

Why Stroke Flies Under the Radar

Stroke prevention is fragmented. Primary prevention (preventing first stroke) falls to primary care. Secondary prevention (preventing recurrent stroke) falls to neurology. Neither may own the cardiovascular component. Many stroke patients aren't anticoagulated properly if AFib is present. Many aren't on high-intensity statins. Blood pressure targets vary by specialty. And dual antiplatelet therapy—aspirin + clopidogrel—is often stopped too early or not initiated at all.

- Stroke is treated as a neurologic event, not a cardiovascular one.
- AFib-related strokes are underprevented due to underuse of anticoagulation.
- Lipid management post-stroke is often inadequate.
- Carotid disease screening (ultrasound, CT angiography) is sporadic.

What Changed in 2026: Stroke as ASCVD

2026 ATP IIIb Update:

- **Ischemic stroke = clinical ASCVD.** Post-stroke patients now qualify for LDL-C <55 mg/dL and non-HDL-C <85 mg/dL targets (secondary prevention). High-intensity statin ± ezetimibe ± PCSK9i is appropriate.
- **Dual antiplatelet therapy (DAPT):** Aspirin + clopidogrel for 21 days post-minor stroke/TIA (CHANCE, POINT trials) is evidence-based. After 21 days, clopidogrel can be discontinued; long-term aspirin continues.
- **Anticoagulation in AFib-related stroke:** Initiated after 2 weeks (if no contraindications) to prevent recurrent cardioembolic stroke. DOAs preferred.
- **Blood pressure targets:** <130/80 mmHg post-stroke to reduce recurrence (ACCORD BP, SPS3 trials support tighter control).
- **Carotid revascularization:** Carotid endarterectomy or stenting if ipsilateral carotid stenosis ≥70% and within 6 months of stroke.

MyCardioAdvocate™ Checklist: Stroke Prevention & Secondary Prevention

1. Do I know my stroke risk factors?

Age, hypertension, diabetes, smoking, AFib, prior TIA/stroke, carotid disease, LDL cholesterol, and family history all increase stroke risk. Ask your doctor to review your risk profile and what can be modified.

2. If I have AFib, am I on appropriate anticoagulation?

AFib raises stroke risk 5-fold. Calculate CHA2DS2-VASc score (even score 1 in men, 2 in women warrants anticoagulation discussion). DOAs (apixaban, dabigatran, edoxaban, rivaroxaban) are preferred over warfarin. Ask: 'What is my stroke risk? Am I on the right anticoagulant?'

3. Am I on aggressive lipid management post-stroke?

If you've had a stroke, you need high-intensity statin. LDL-C target is <55 mg/dL. If not achieved, ezetimibe and possibly PCSK9i should be added. Ask: 'What is my current LDL? Am I on high-intensity statin therapy?'

4. Is my blood pressure well-controlled?

Post-stroke, aim for <130/80 mmHg. Hypertension is the #1 modifiable stroke risk factor. Regular BP checks and medication adjustment are critical. Ask: 'What is my BP target, and are we at goal?'

5. Have I been screened for carotid disease?

Ultrasound or CT angiography can identify carotid narrowing. If a carotid artery is significantly stenosed (70%+) on the same side as your stroke, intervention may reduce recurrent stroke risk. Ask: 'Should I have carotid imaging?'

CPR Opportunity: Dual Antiplatelet Duration Post-Minor Stroke

The Gray Zone: CHANCE and POINT trials support aspirin + clopidogrel for 21 days after minor stroke or TIA. After 21 days, benefit plateaus and bleeding risk increases. Most guidelines recommend stopping clopidogrel at 21 days. However, some neurologists extend DAPT to 90 days, especially in high-risk cases. When is longer DAPT justified?

> **Shared Decision-Making Frame:** Standard practice is 21 days DAPT for minor stroke/TIA, then aspirin monotherapy. If you have recurrent TIAs despite DAPT or very high-risk features (severe carotid stenosis, multiple recurrent TIAs), longer DAPT may be considered. Discuss bleeding vs. ischemic risk with your neurologist and cardiologist.

Key Takeaways

- Ischemic stroke is cardiovascular disease. Secondary prevention targets are as aggressive as post-MI.
- LDL-C <55 mg/dL post-stroke. High-intensity statin ± ezetimibe ± PCSK9i.
- AFib-related strokes require anticoagulation (DOA preferred).
- Dual antiplatelet therapy (aspirin + clopidogrel) for 21 days after minor stroke/TIA.
- Blood pressure target <130/80 mmHg; screen for carotid disease.

Next Steps & Related Content

- If you've had a stroke, schedule a cardiology follow-up to discuss secondary prevention goals.
- Ask: 'What is my LDL cholesterol? Am I on high-intensity statin? Should I be on ezetimibe or PCSK9i?'
- Get recent blood pressure readings. Discuss target and medication adjustments.
- Ask if carotid ultrasound or CT angiography is appropriate for you.
- Review: MyCardioAdvocate™ After a Heart Attack, AFib, Lipid Guidelines, Statins, Hypertension.