Carotid Disease and CAD

Brain–Heart Risk and Safer Decisions

Audience: APRN | **Contact hrs:** 1.5 | **Pharmacology hrs:** 0.0
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Pharmacology hours are included in the total contact hours.

The artery is local; the risk is systemic

A carotid scan answers a question about one vascular territory. It does not, by itself, explain a patient's total cardiovascular risk or identify the source of every future stroke. A person with carotid atherosclerosis may also have coronary artery disease (CAD), aortic atheroma, peripheral artery disease, or atrial fibrillation. These conditions can coexist while requiring different diagnostic and treatment decisions. The useful clinical question is therefore not simply whether heart disease should be considered. It is how to assess the heart without overlooking an urgent neurologic event, ordering low-value screening, or exposing the patient to unnecessary procedures (Naylor et al., 2023; Virani et al., 2023).

Purpose and audience. This adult-care activity is designed for advanced practice registered nurses (APRNs). It develops integrated assessment, prevention, referral, and surveillance decisions for extracranial carotid stenosis and CAD, including patients undergoing coronary artery bypass grafting (CABG). Diagnostic and prescribing decisions depend on local authorization and privileges; nursing monitoring and escalation follow approved pathways. Recommendations identify U.S. and European guidance where their framing differs. Five fictional cases illustrate application rather than prescribing a universal protocol.

Learning outcomes. After completing this activity, the learner should be able to: distinguish systemic atherosclerotic risk from lesion-specific stroke mechanisms; recognize carotid-territory symptoms and select an appropriate diagnostic pathway; justify selective coronary and pre-CABG carotid assessment; individualize prevention and antithrombotic safety checks; interpret contemporary revascularization evidence without confusing populations or endpoints; and prioritize neurologic, cardiac, airway, and hemodynamic complications after intervention.

The practical starting point is a paired history: “What has changed in brain or retinal function?” and “What has changed in exercise tolerance or cardiac symptoms?” A scan showing severe stenosis is important, but symptom timing, functional status, competing illness, procedural risk, and treatment preferences determine what that scan means. A prevention plan should begin while the team establishes whether an intervention is appropriate—not only after the procedural decision is finished.

Shared biology, different routes to injury

Atherosclerosis is a systemic inflammatory process involving retention of atherogenic lipoproteins, endothelial dysfunction, inflammatory-cell recruitment, and remodeling of the arterial wall. The carotid bulb is particularly susceptible because local flow patterns create regions of disturbed shear. Plaque composition matters as well as lumen narrowing: a lipid-rich core, fibrous-cap disruption, surface thrombus, ulceration, and intraplaque hemorrhage can make an arterial lesion more likely to generate emboli. A tight but stable lesion and a less severe recently symptomatic lesion therefore do not necessarily carry the same immediate risk (Naylor et al., 2023).

Figure 1 separates two ideas that patients often combine. Carotid plaque can release material downstream into the ipsilateral retinal or cerebral circulation. Coronary plaque can impair myocardial perfusion or provoke a local coronary thrombotic event. Carotid plaque does not normally travel backward through the aorta into a coronary artery. Its importance for the heart is principally as evidence of shared atherosclerotic susceptibility, not as a direct coronary embolic source.

Cerebral injury can arise through artery-to-artery embolization, reduced perfusion distal to critical disease, or an interaction between the two. When distal arterioles have already dilated to preserve flow, reserve may be limited during hypotension or low cardiac

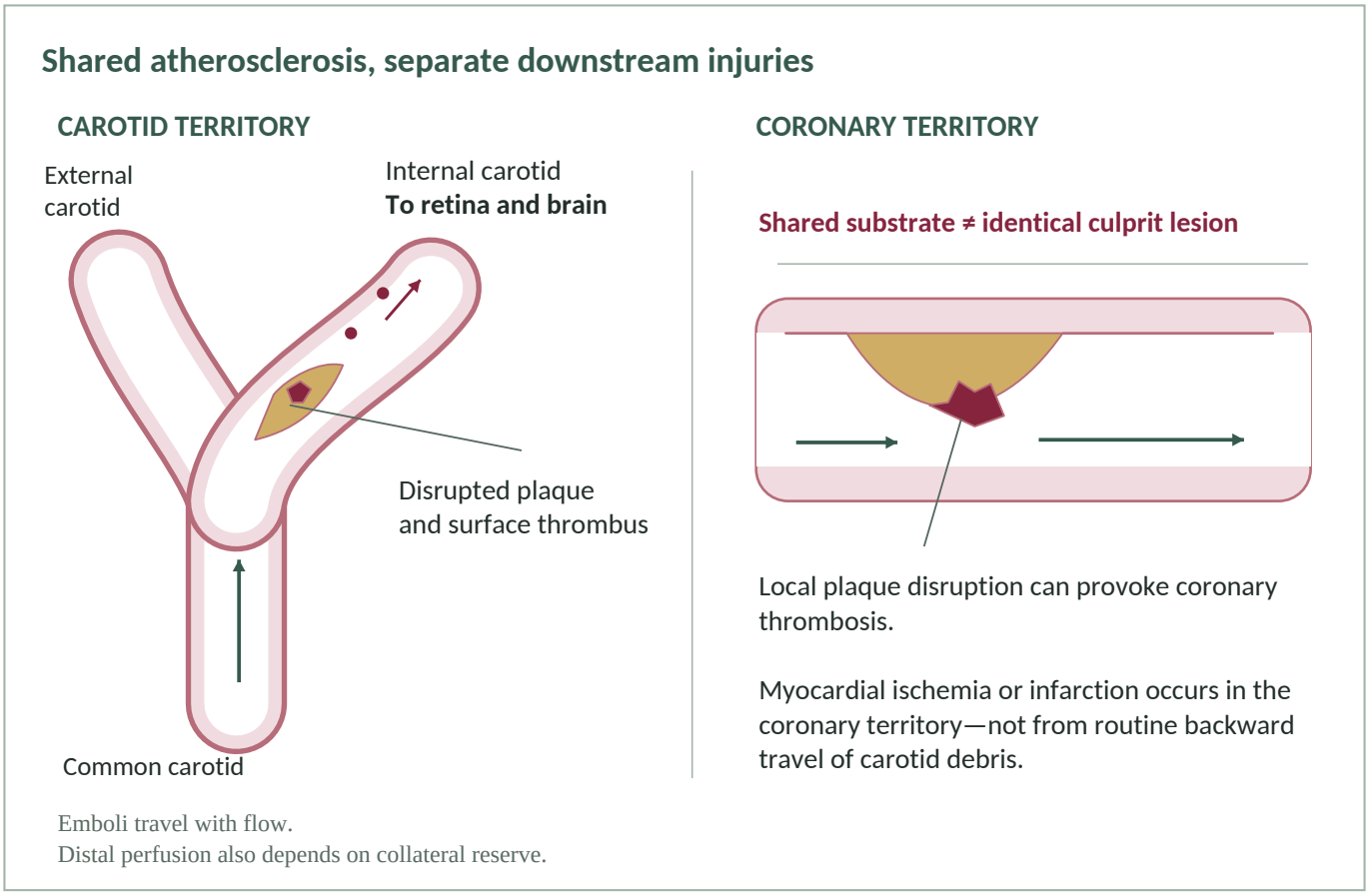


Figure 1. Shared disease, different local events. Original educational schematic, not to scale. Carotid embolization follows downstream flow; coronary atherosclerosis is a related but separate lesion. Evidence: Naylor et al. (2023); Virani et al. (2023).

output. Collateral circulation and the status of the opposite carotid and intracranial vessels influence tolerance. Severe bilateral disease or occlusion on one side may therefore require a different perioperative discussion from an isolated unilateral lesion.

The same distinction matters when assessing association and causation. A patient with carotid stenosis may be at increased overall vascular risk, yet a postoperative stroke might arise from the aortic arch, new atrial fibrillation, intracardiac thrombus, or another mechanism. Removing a carotid plaque cannot prevent every one of these events. Conversely, uncertainty about the cause of a possible future CABG-related stroke does not negate the established importance of evaluating a recently symptomatic carotid lesion.

Risk is a clinical profile—not a percentage alone

Document age, smoking exposure, blood pressure, lipid treatment, diabetes, kidney function, prior myocardial infarction, heart failure, prior stroke or transient ischemic attack (TIA), and functional limitations. Ask which vascular events actually occurred and when. “History of carotid disease” is less informative than “left retinal ischemic event ten days ago with a confirmed left internal carotid stenosis.” Frailty, advanced nonvascular illness, and limited life expectancy may reduce the opportunity to benefit from a preventive procedure, even when the stenosis looks impressive.

A patient with disease in several arterial beds needs coordinated prevention and a realistic discussion of absolute benefit and harm. It is not enough to assign a label such as “high risk.” Specify high risk of what: ipsilateral stroke without intervention, myocardial infarction during an operation, bleeding during antithrombotic treatment, or technical complications with a particular access route. These risks can point toward different choices.

Recognize symptoms before ordering surveillance

Carotid-territory ischemia commonly produces sudden monocular visual loss on the affected side or contralateral weakness or numbness. Aphasia can accompany dominant-hemisphere ischemia. Ask about a curtain or shade over one eye, inability to manipulate familiar objects, unexpected word-finding difficulty, and whether a witness observed facial asymmetry or abnormal speech. Isolated nonspecific dizziness or syncope should not automatically be attributed to an

incidental carotid stenosis. However, posterior-circulation symptoms and stroke mimics also require a competent assessment; an atypical presentation is not a reason to dismiss an acute neurologic change (Amin et al., 2023).

TIA is not defined simply by symptoms resolving within 24 hours. The contemporary tissue-based distinction is transient focal ischemic dysfunction without acute infarction. A person whose symptoms resolve but whose imaging demonstrates an acute infarct has had an ischemic stroke. Normal findings when the patient reaches the clinic do not erase a credible history. A low clinical risk score or a negative noncontrast head CT cannot, by itself, exclude a dangerous ischemic event (Amin et al., 2023).

New focal deficits call for the emergency stroke pathway: establish last-known-well time, check point-of-care glucose, assess airway and circulation, obtain a focused neurologic examination, and arrange urgent brain and vascular imaging according to the treating system. Do not wait for an outpatient carotid ultrasound before activating emergency evaluation. Resolved focal symptoms still require urgent TIA/stroke assessment, including consideration of cardiac rhythm and other embolic sources. The specific imaging sequence depends on local capability and the acute clinical question.

> **Case 1 | The symptom disappeared**
> A 69-year-old with diabetes and a prior coronary stent describes eight minutes of painless loss of vision in the left eye that occurred this morning. Vision and strength are now normal. An old record lists a left carotid bruit. The best next step is urgent evaluation for retinal or cerebral ischemia, not reassurance or a routine scan several months later. Document the affected eye, onset, duration, recurrence, medications, and last-known-well time. A bruit is neither proof of a culprit stenosis nor an explanation that replaces brain, vascular, and cardiac assessment. Symptom resolution makes the history more important, not less important (Amin et al., 2023).

Silent ischemia is not synonymous with infarction

Myocardial ischemia means inadequate oxygen delivery relative to myocardial demand. Myocardial infarction requires acute myocardial injury with evidence of ischemia; an elevated troponin alone is not sufficient to label every patient as having an infarction. Silent ischemia refers to objective ischemia without recognized anginal symptoms, whereas a silent or unrecognized infarction describes myocardial necrosis that was not identified clinically when it occurred. These terms should not be used interchangeably (Thygesen et al., 2018).

Ask about exertional breathlessness, reduced walking distance, pressure in the chest or upper body, unexplained diaphoresis, and a change in usual activities. Patients may have normalized symptoms by avoiding exertion, and neuropathy or other illness may alter symptom perception. Nevertheless, the possibility of silent disease does not establish that every asymptomatic person with carotid plaque benefits from a stress test. Symptoms, functional change, established CAD, and the consequence of a test result should drive the next decision.

Imaging: confirm the lesion and its meaning

Duplex ultrasonography is commonly the initial test for extracranial carotid disease. It combines plaque and lumen imaging with Doppler velocity measurements. A useful report identifies side, vessel segment, degree of stenosis, image limitations, and the interpretive criteria used. Do not transfer a velocity threshold from an old report into a different laboratory's classification without checking its validated standard. Technical quality and concordance among parameters are especially important near a treatment threshold (Intersocietal Accreditation Commission [IAC], 2023).

The IAC's updated interpretation recommendations raised the principal peak systolic velocity threshold for 50% stenosis to 180 cm/s while retaining a multiparameter approach. A peak systolic velocity of 125–180 cm/s with an internal-to-common carotid velocity ratio of at least 2 can also support 50–69% stenosis when the overall findings fit. This is why a single number should not be interpreted in isolation. Heavy calcification, acoustic shadowing, low cardiac output, contralateral disease, and near-occlusion can create misleading or discordant patterns (IAC, 2023).

Computed tomographic angiography (CTA) or magnetic resonance angiography (MRA) can define anatomy when duplex is uncertain or when revascularization is being planned. Selection considers kidney function, contrast issues, implanted-device compatibility, motion, claustrophobia, local expertise, and the urgency of the question. Noncontrast MRA may

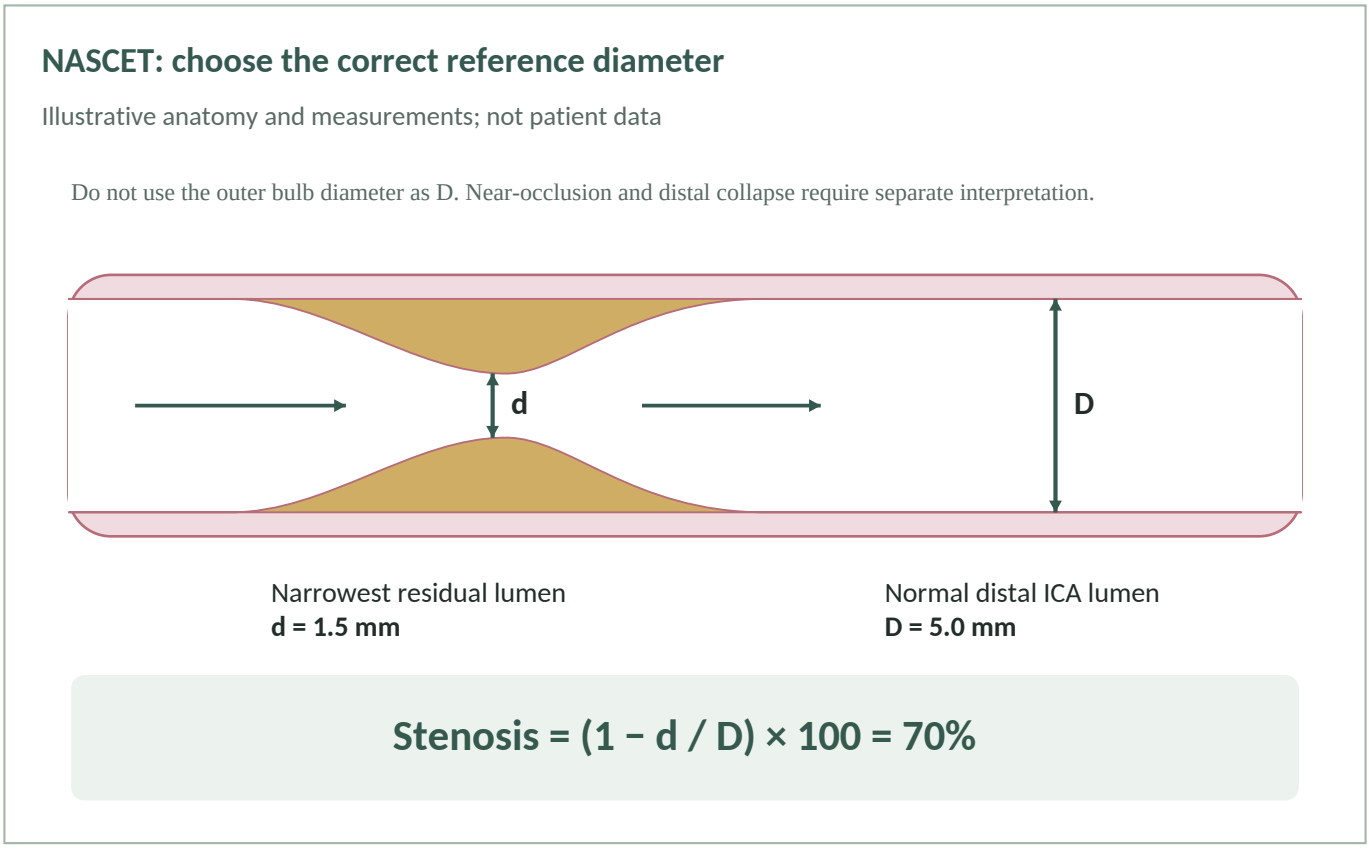


Figure 2. NASCET measurement. Original diagram using an illustrative 1.5 mm residual lumen and 5.0 mm normal distal lumen. Near-occlusion is not an ordinary percentage measurement. Evidence: Naylor et al. (2023).

be useful in selected patients, but flow-related artifacts can overestimate disease. CTA can also be limited by heavy calcification. Catheter angiography is not a reflex confirmatory test for every patient because an invasive study introduces its own risk.

The North American Symptomatic Carotid Endarterectomy Trial (NASCET) method compares the narrowest residual lumen with a normal distal internal carotid artery diameter. Figure 2 shows the denominator explicitly. Different measurement conventions can produce different numerical percentages for the same lesion. Near-occlusion, especially with distal vessel collapse, requires specialist interpretation rather than an ordinary percentage calculation; complete occlusion is a separate category. Confirm which condition is present before treating “almost closed” as an uncomplicated severe stenosis (Naylor et al., 2023).

Case 2 | One velocity does not make the diagnosis

A 73-year-old being evaluated after transient right-hand weakness has a left internal carotid peak systolic velocity of 165 cm/s, a velocity ratio of 2.7, visible plaque, and substantial acoustic shadowing. The report calls the examination technically limited. Declaring the lesion mild solely because the velocity is below 180 cm/s would be an error. The ratio and clinical context matter, and the anatomy may need clarification with another modality. The APRN should communicate the recent focal event, confirm the laboratory's interpretation, and expedite specialist review rather than making a procedural decision from one Doppler value (IAC, 2023).

Advanced plaque imaging can identify features associated with risk, including intraplaque hemorrhage, surface abnormalities, and progression. These findings may contribute to multidisciplinary selection, but an association is not an automatic treatment indication. The practical question remains whether identifying the feature changes an evidence-supported decision for this patient, at an institution with acceptable outcomes. Serial examinations should be compared using consistent methods whenever possible.

Assess the other vascular bed selectively

Screening, diagnosis, and surveillance are different activities. Screening tests a person without relevant symptoms or a known lesion. Diagnostic imaging investigates symptoms or a specific clinical concern. Surveillance follows established disease or a treated

Table 1. IAC duplex interpretation: selected updated parameters

Category	ICA PSV (cm/s)	ICA/CCA PSV ratio	ICA EDV (cm/s)	Interpretation safeguard
Normal / <50%	<180	<2	<40	Distinguish normal from plaque-bearing arteries using the image.
50–69%	180–230	2–4	40–100	PSV 125–180 with ratio ≥2 can also support this category.
>70%, below near-occlusion	>230	>4	>100	Integrate plaque/lumen findings and concordant parameters.
Near-occlusion	Variable	Variable	Variable	Distal collapse or very low flow requires specialist interpretation.
Complete occlusion	No detectable flow	Not applicable	Not applicable	Confirm absence of a patent lumen and distinguish very low flow.

Selected parameters, not a stand-alone diagnostic rule. ICA = internal carotid artery; CCA = common carotid artery; PSV = peak systolic velocity; EDV = end-diastolic velocity. Source: IAC (2023).

artery. Confusing these purposes can turn an appropriate test in one setting into a blanket policy in another.

The U.S. Preventive Services Task Force recommends against screening the general asymptomatic adult population for carotid stenosis. Its recommendation does not apply to the diagnostic evaluation of recent neurologic symptoms. A bruit has limited accuracy and does not reliably quantify stenosis; absence of a bruit does not exclude disease. CAD, smoking, or several vascular risk factors should prompt prevention and clinical assessment, but not an automatic general-population carotid screening program (U.S. Preventive Services Task Force [USPSTF], 2021).

Selected patients being considered for CABG constitute a different decision context. Prior stroke or TIA, a carotid bruit, older age, and severe coronary disease can prompt selective carotid duplex when the result could alter counseling or management. The goal is not to imply that the scan will detect every cause of operative stroke. Routine screening of every patient and selective assessment of a high-risk surgical patient should not be described as the same recommendation (Naylor et al., 2023).

A structured coronary history before another test

In a person with carotid disease, establish whether CAD is already documented and whether symptoms or function have changed. Review prior myocardial infarction, coronary anatomy when available, percutaneous coronary intervention (PCI) and stent dates, CABG history, ventricular function, angina treatment, and exercise tolerance. Assess the pulse and rhythm, signs of congestion, blood pressure, and medication adherence. A person who “has no chest pain” but has stopped walking to the mailbox has not necessarily demonstrated stable functional status.

For established chronic coronary disease, routine periodic anatomic or ischemic testing without a change in clinical or functional status is not recommended. In patients being evaluated for noncardiac surgery, testing and treatment should follow the indications that would apply outside the perioperative setting, with attention to avoiding unnecessary delay. The AHA's 2026 republication of its perioperative guideline retained the 2024 recommendations after review through March 2026. These principles support selective, management-changing investigation rather than obligatory stress imaging before every carotid procedure (Virani et al., 2023; American Heart Association [AHA], 2026b).

The APRN can make the reasoning explicit: What question will this test answer? What would change after a positive or negative result? Could it delay a time-sensitive carotid operation without improving care? New ischemic symptoms, decompensated heart failure, or an unstable arrhythmia require evaluation on their own merits. Stable disease should not be “cleared”

by accumulating normal tests that do not address the actual risk.

Coronary revascularization is not a general prerequisite for carotid revascularization. In stable patients, the decision to perform PCI or CABG should arise from the coronary indication, not simply from the desire to make a noncardiac operation appear safer. A new coronary stent can also introduce an antiplatelet requirement that complicates the timing of carotid surgery. The potential diagnostic benefit must therefore be considered together with the downstream consequences.

Prevention is treatment—not a waiting period

Intensive medical management is required whether the patient ultimately receives endarterectomy, stenting, or no carotid procedure. It addresses the shared substrate for stroke and myocardial infarction. A procedural success does not remove aortic or coronary atherosclerosis, and an uncomplicated recovery is not a reason to discontinue prevention. Medication reconciliation, smoking cessation support, nutrition, physical activity adapted to functional status, blood-pressure treatment, and diabetes management should be translated into a plan the patient can actually follow (Virani et al., 2023; Brott et al., 2026).

Lipid lowering with a treatment target and follow-through

Statins remain foundational treatment for clinical atherosclerotic cardiovascular disease (ASCVD). Use maximally tolerated therapy and address adherence before declaring treatment failure; add evidence-supported nonstatin therapy when needed for the patient's risk and goal. The 2026 U.S. dyslipidemia guidance recommends a low-density lipoprotein (LDL) cholesterol goal below 55 mg/dL for very-high-risk clinical ASCVD, with a non-HDL cholesterol goal below 85 mg/dL. Clinical ASCVD not meeting very-high-risk criteria generally has a less intensive LDL goal below 70 mg/dL. An incidental plaque finding should not be used to assign every patient automatically to the same risk category (AHA, 2026a).

Explain what the drug is intended to accomplish even when the patient feels well. Ask about the reason for missed doses, access, prior symptoms, and concurrent medicines. New severe muscle symptoms, weakness, dark urine, or symptoms suggesting hepatic injury require clinical assessment, not automatic dismissal as an expected effect. Conversely, a nonspecific ache should be evaluated rather than allowed to end lipid treatment permanently without discussion of alternatives. Document the follow-up lipid measurement and who will act on the result.

Antithrombotic treatment follows the indication

Antiplatelet treatment and anticoagulation are not interchangeable. Antiplatelets address platelet-mediated arterial thrombosis in many carotid and coronary settings. Oral anticoagulation may be indicated for atrial fibrillation or another cardioembolic condition. A carotid stenosis alone is not an indication to add therapeutic-dose anticoagulation. Likewise, finding carotid plaque in a patient already taking an anticoagulant is not an automatic reason to add aspirin. Balance the actual coronary, cerebral, stent-related, and bleeding indications together (Naylor et al., 2023; Virani et al., 2023).

Single antiplatelet treatment is common in stable atherosclerotic disease. Dual antiplatelet therapy (DAPT) is used for defined situations, including carotid stenting, selected recent TIA or minor stroke pathways, and coronary indications. The indication, start date, planned duration, and stop or review date should be visible in the record. DAPT for a carotid stent is not the same prescription as long-term secondary stroke prevention, and one specialist's routine instructions may conflict with another specialist's procedural plan.

For carotid stenting, aspirin plus clopidogrel is a commonly recommended regimen continued for at least four weeks after the procedure, followed by long-term single antiplatelet treatment unless another indication changes the plan. For a recent TIA or minor stroke, short-term DAPT eligibility and duration depend on the neurologic syndrome and planned intervention. In a patient proceeding to carotid endarterectomy, the stroke and vascular teams should use an agreed local antiplatelet pathway so that treatment uncertainty does not delay a beneficial operation. Long-term DAPT is not a default response to any carotid plaque (Naylor et al., 2023).

Before administering or changing antithrombotic therapy, establish the last dose, recent PCI or carotid stent, prior intracranial or gastrointestinal bleeding, anemia, kidney function, and all prescription and nonprescription medicines. Concurrent anticoagulants, additional antiplatelets, and nonsteroidal anti-inflammatory drugs can increase bleeding risk. Teach the patient not to stop a prescribed stent-protection regimen without contacting the treating team. Bleeding symptoms require assessment; a generic

Table 2. Medication decisions across carotid and coronary care

Treatment role	Useful clinical distinction	Monitoring and teaching priority
Lipid lowering	Maximally tolerated statin-based treatment; nonstatin additions according to risk and goal.	Check adherence, response, symptoms, interactions, and a named follow-up plan.
Single antiplatelet therapy	Common in stable atherosclerotic disease; selection depends on the indication and tolerance.	Identify bleeding history, concurrent drugs, and the actual reason for treatment.
Dual antiplatelet therapy	Defined stent or recent ischemic indications—not a universal lifelong prescription for plaque.	Record start, duration, review/stop date, and one agreed perioperative plan.
Oral anticoagulation	May be needed for atrial fibrillation or another embolic indication; not for carotid narrowing alone.	Review kidney function, bleeding risk, and whether an added antiplatelet is truly needed.
Pressure / cardiorenal therapy	Chronic prevention goals differ from immediate operative hemodynamic requirements.	Monitor symptoms, renal function and electrolytes as indicated; plan perioperative interruptions.

This comparison does not replace product labeling or an individualized prescription. Sources: Naylor et al. (2023); Virani et al. (2023); AHA (2026a, 2026b).

instruction to stop every “blood thinner” can be as unsafe as ignoring major bleeding.

Case 3 | Two specialists, one medication list

A 66-year-old with a recent coronary drug-eluting stent takes aspirin and clopidogrel. A symptomatic carotid lesion is now being evaluated for endarterectomy. A generic preoperative handout says to stop antiplatelets. The APRN should reconcile the actual PCI indication and date, notify cardiology and vascular surgery, and obtain one documented perioperative plan. Independently stopping both agents risks coronary stent thrombosis; independently continuing every agent without considering surgical bleeding is also unsafe. The needed intervention is coordinated prescribing, with the patient receiving a single clear set of instructions (Naylor et al., 2023; AHA, 2026b).

Blood pressure, diabetes, and practical adherence

Long-term blood-pressure control reduces vascular risk, but chronic targets and immediate perioperative limits are different decisions. CREST-2 intensive management targeted systolic pressure below 130 mmHg and LDL cholesterol below 70 mg/dL. Those trial targets describe the intervention tested; they do not supersede newer risk-specific lipid guidance. In a person with recent ischemia, orthostatic symptoms, bilateral severe disease, or poor cardiac output, avoid unmonitored rapid pressure changes. A blood-pressure number is meaningful only alongside symptoms, volume status, and the current phase of care (Brott et al., 2026).

When adjusting renin–angiotensin system therapy or diuretics, arrange renal function and electrolyte monitoring appropriate to the drug and clinical state. In patients with diabetes, heart failure, kidney disease, or obesity, choose treatments with demonstrated cardiovascular benefit when indicated rather than treating glycemic numbers in isolation. Perioperative instructions must account for the individual medicine: for example, sodium–glucose cotransporter 2 (SGLT2) inhibitors require advance interruption before planned surgery because of ketoacidosis risk, using the product-specific and perioperative protocol. The reaffirmed U.S. guideline describes a three- to four-day interval (AHA, 2026b).

Practical prevention includes asking whether prescriptions are affordable, whether the patient can read the labels, and whether there is a reliable refill plan. A simple schedule, teach-back, smoking-cessation support, and a named contact for side effects often matter more than adding another instruction sheet.

Record both the treatment goal and the barrier the plan is intended to overcome.

Recently symptomatic stenosis: time and selection

A carotid lesion is considered symptomatic when a recent ipsilateral retinal or cerebral ischemic event can reasonably be attributed to it. Nonspecific symptoms and a contralateral or posterior-circulation event should not automatically label the lesion symptomatic. Establish laterality, timing, brain imaging, and alternative mechanisms. The evidence for revascularization is strongest for appropriately selected patients with recent symptoms and severe stenosis, not simply for anyone whose ultrasound report contains a high percentage (AbuRahma et al., 2022; Naylor et al., 2023).

Carotid endarterectomy (CEA) is generally favored for suitable patients with symptomatic 70–99% stenosis when the center's expected 30-day stroke/death risk is below 6%. Selected patients with 50–69% stenosis can also benefit; age, sex, comorbidity, anatomy, symptom type, and time since the event influence the balance. Routine CEA is not indicated for stenosis below 50%, and a completely occluded internal carotid artery is not treated as an ordinary severe stenosis. Near-occlusion deserves a separate specialist assessment (Naylor et al., 2023).

Benefit from intervention declines with unnecessary delay. In neurologically stable patients after a nondisabling stroke, SVS guidance supports revascularization after the first 48 hours and within 14 days of symptom onset. A TIA without infarction and a large evolving infarct are not the same timing problem. Large infarction, hemorrhagic transformation, substantial disability, unstable neurology, or competing cardiac instability can alter the plan. Neither a favorable NIH Stroke Scale score alone nor a small historical surgical series establishes that every symptomatic patient should undergo immediate CEA (AbuRahma et al., 2022).

CEA, transfemoral stenting, and TCAR are distinct choices

CEA removes plaque through a neck incision. Transfemoral carotid artery stenting (CAS) treats the lesion from an arterial access route that traverses the aorta and supra-aortic vessels. In older patients, aortic arch atheroma, elongation, calcification, and vessel tortuosity can increase embolic and technical risk with transfemoral CAS. Age is therefore relevant to procedure selection, but it is not a substitute for assessment of anatomy and overall health. CEA is generally preferred over transfemoral CAS in suitable older symptomatic patients (Naylor et al., 2023).

The original CREST trial illustrates why a composite endpoint needs interpretation. Similar overall composite results did not mean identical complications: periprocedural stroke was more frequent with CAS, whereas myocardial infarction was more frequent with CEA. Patients may value these outcomes differently, and both deserve discussion. “Less invasive” should not be translated automatically into “lower total risk” (Brott et al., 2010).

Transcarotid artery revascularization (TCAR) uses direct carotid access and temporary flow reversal for embolic protection. It avoids some aortic arch manipulation but still requires patient selection, appropriate anatomy, and antiplatelet planning. It must not be treated as synonymous with transfemoral CAS when discussing trial results. In particular, TCAR was not included in CREST-2; that trial does not supply a randomized comparison of TCAR with intensive medical management alone (Naylor et al., 2023; Brott et al., 2026).

The decision conversation should identify the expected procedural stroke/death rate at the treating center, cardiac risk, cranial nerve or access complications, need for DAPT, and the patient's goals. Referral is not a commitment to a procedure. It is an opportunity to verify whether the individual resembles the populations in whom benefit has been demonstrated.

Asymptomatic disease: interpret the new trials carefully

Older asymptomatic trials established that CEA could reduce later stroke in selected patients, but medical therapy and procedural practice have changed. Contemporary decisions must therefore distinguish historical evidence, current trial results, and guidelines written before those results. The 2022 SVS and 2023 ESVS guidance preceded the main CREST-2 publication. Their recommendations should be interpreted alongside—not represented as already incorporating—the newer randomized evidence (AbuRahma et al., 2022; Naylor et al., 2023; Brott et al., 2026).

CREST-2 consisted of two parallel trials in patients with at least 70% stenosis and no ipsilateral stroke, TIA,

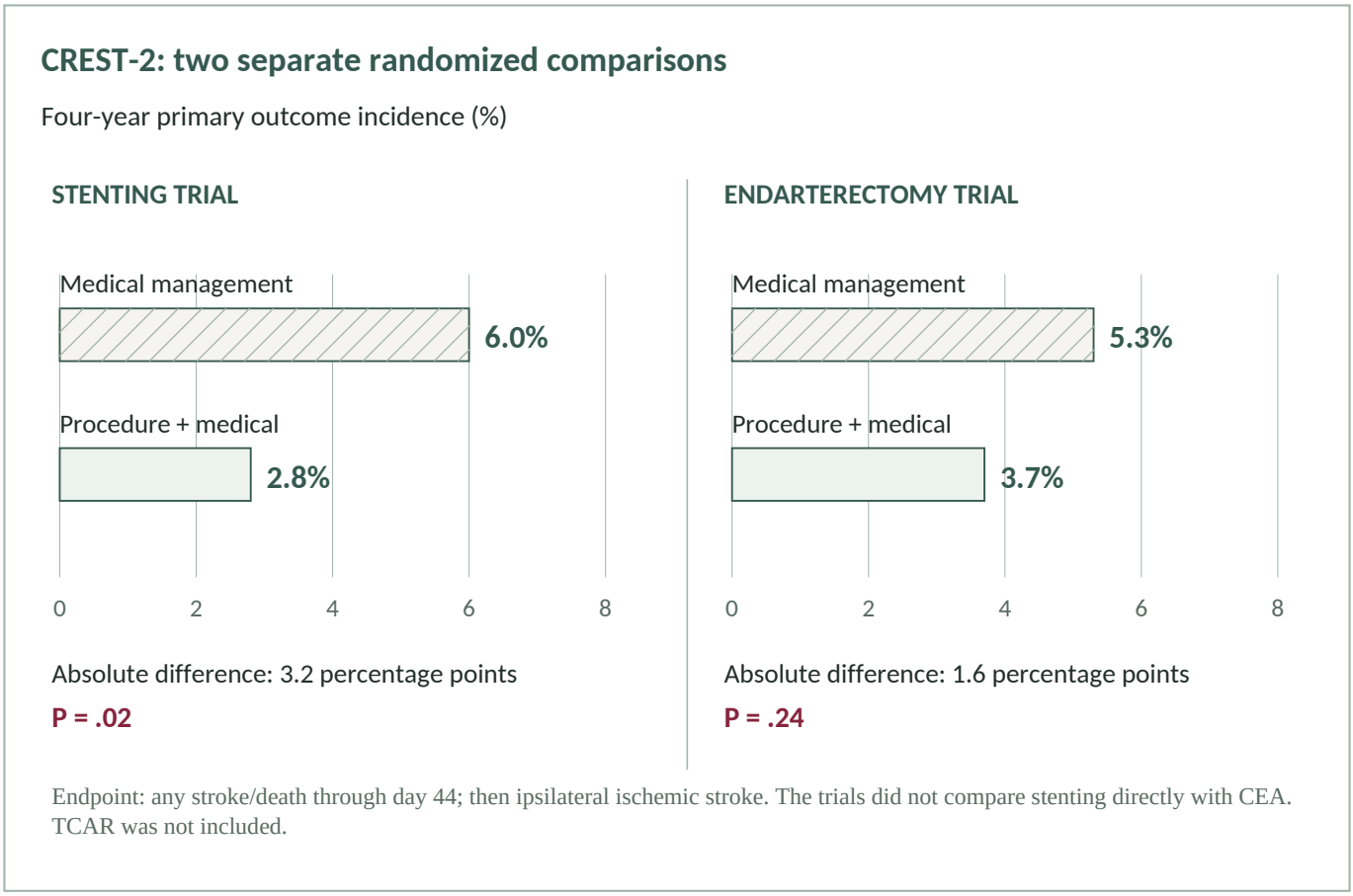


Figure 3. CREST-2 primary outcomes. Original display of published trial estimates, not a head-to-head procedural comparison. Each procedure group received intensive medical management. Evidence: Brott et al. (2026).

or amaurosis fugax in the preceding 180 days. One trial compared stenting plus intensive medical management with medical management alone; the other compared CEA plus intensive management with medical management alone. This design did not randomize patients directly between stenting and CEA. Certified operators and defined eligibility criteria are important to its interpretation (Brott et al., 2026).

At four years, the primary outcome occurred in 6.0% of the medical-only group and 2.8% of the stenting group in the stenting trial, an absolute difference of 3.2 percentage points (P = .02). In the CEA trial, the corresponding rates were 5.3% and 3.7%, an absolute difference of 1.6 percentage points (P = .24). The endpoint combined any stroke or death through day 44 with subsequent ipsilateral ischemic stroke. It was not a four-year composite of every cardiovascular event. Figure 3 keeps the two randomized comparisons separate.

The stenting trial demonstrated a statistically significant reduction in its primary outcome. The CEA trial did not demonstrate a statistically significant reduction at four years; this does not prove equivalence or absence of every possible benefit. Comparing the P values across separate trials cannot establish that CAS is superior to CEA. Nor should a trial excluding unstable angina and atrial fibrillation requiring anticoagulation be treated as a direct answer for every patient with complex cardiac disease (Brott et al., 2026).

ECST-2 offers a complementary but different perspective. Its two-year interim analysis involved 429 patients with asymptomatic or low-to-intermediate-risk symptomatic stenosis and found no evidence of benefit from adding revascularization to optimized medical therapy over that interval. Its risk-selected population, shorter follow-up, and mixed symptom status prevent direct substitution for CREST-2. It also does not overturn the established urgency of evaluating a high-risk, recently symptomatic severe lesion (Donners et al., 2025).

Translate evidence into a shared decision

An asymptomatic stenosis is not an emergency solely because it is severe. Discuss the person's expected survival and function, medical-treatment adherence, procedural anatomy, local complication rates, and willingness to accept an immediate procedural risk for a possible later reduction in stroke. Older guidelines

generally required very low perioperative risk and sufficient life expectancy for benefit to accrue. Patients with major competing illness may have little opportunity to realize a delayed advantage.

Use absolute numbers and clearly state the population and time horizon. A 3.2-percentage-point difference over four years in one randomized comparison is not a guarantee that a particular patient will benefit. Progression or concerning plaque features can contribute to the discussion, but they do not remove uncertainty. Equally, choosing medical treatment should mean active risk-factor management and follow-up, not discharge from care. The plan should explain what symptom or imaging change will trigger reassessment.

CABG: prevent stroke without assuming its source

Stroke around CABG is a serious complication with multiple pathways. Manipulation of an atherosclerotic ascending aorta can release embolic material. Cardiac thromboembolism, including postoperative atrial fibrillation, can contribute. Hypotension and low cardiac output may reduce cerebral perfusion, especially when reserve is limited. Carotid plaque can also be an embolic source. Figure 4 shows why a technically successful carotid intervention cannot eliminate all of these mechanisms (Naylor et al., 2023).

The distinction between an early deficit recognized on awakening and a later event after initially normal recovery can guide investigation, but timing alone does not prove a cause. Review intraoperative hemodynamics, aortic findings, rhythm, neurologic baseline, and the distribution of injury. A stroke in a patient with carotid stenosis should not be labeled “carotid-related” without evaluating whether the territory and clinical evidence support that conclusion.

Who needs a carotid procedure before or with CABG?

Recent carotid-territory symptoms with significant stenosis justify a different discussion from an asymptomatic unilateral lesion. SVS guidance supports considering CEA before or concomitant with CABG in patients with symptomatic 50–99% stenosis who require both procedures, with sequencing determined by clinical presentation and institutional experience. A patient with unstable coronary ischemia may not tolerate the same staging delay as someone with stable CAD and a recent TIA (AbuRahma et al., 2022).

For an asymptomatic unilateral 70–99% carotid stenosis, ESVS guidance does not recommend staged or synchronous carotid intervention solely to prevent postoperative stroke during CABG. Bilateral severe stenoses or a severe stenosis with contralateral occlusion may justify individualized consideration, but they are not automatic indications for a combined operation. Neurology, vascular surgery, cardiac surgery, cardiology, and anesthesia should identify which vascular threat is most immediate and what each proposed procedure can realistically prevent (Naylor et al., 2023).

The CABACS randomized trial directly examined synchronous CABG plus CEA versus CABG alone in patients with asymptomatic severe carotid stenosis. Recruitment stopped early, leaving 127 randomized patients. Five-year stroke or death was 40.6% with combined surgery and 35.0% with CABG alone (P = .58). The trial did not demonstrate benefit from adding CEA, but its size limits precision. These findings should not be converted into proof that all strategies are equivalent or that every symptomatic patient should avoid carotid treatment (Knipp et al., 2022).

Case 4 | An 80% stenosis before bypass

A 76-year-old with stable multivessel CAD is scheduled for CABG. Selective imaging shows an 80% right internal carotid stenosis; the left artery has mild disease. There is no history of retinal or hemispheric ischemia. The family asks for both arteries to be “fixed at once.” Explain that a unilateral asymptomatic carotid lesion does not establish the source of a possible operative stroke, and routinely adding CEA has not shown a clear benefit in this setting. The team should optimize medical therapy, evaluate the aorta and overall operative risk, and document an individualized plan. A newly disclosed focal neurologic event would change the assessment (Naylor et al., 2023; Knipp et al., 2022).

Staging creates competing hazards: carotid-first treatment can delay needed coronary care, CABG-first treatment can leave a recently symptomatic carotid lesion untreated, and synchronous treatment combines physiologic and procedural burdens. Stenting introduces a DAPT requirement that may be difficult to reconcile with an urgent cardiac operation. These are reasons for explicit multidisciplinary planning rather than a

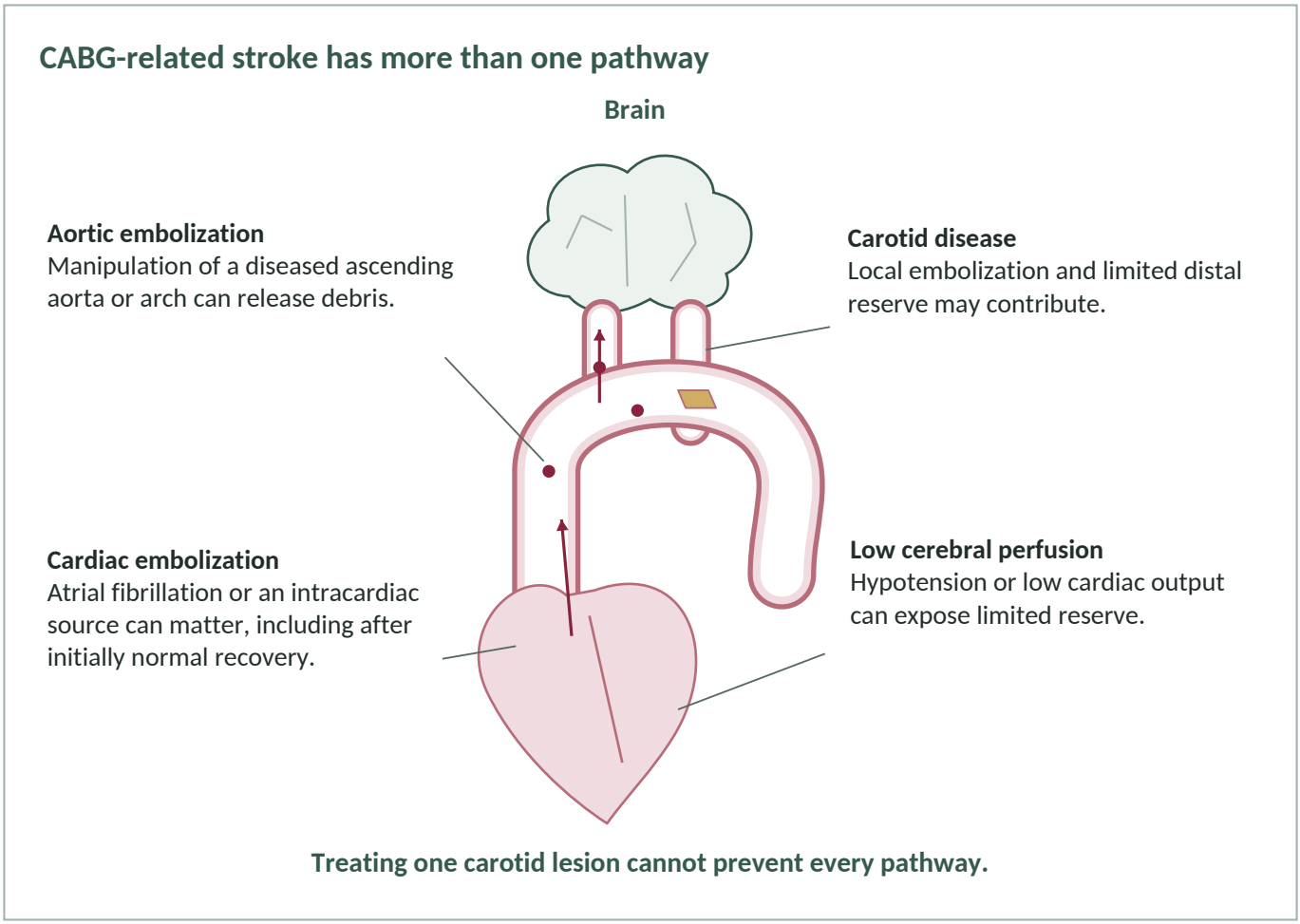


Figure 4. Mechanisms of stroke around CABG. Original simplified circulation schematic; connections illustrate mechanisms rather than precise anatomy or laterality. Evidence: Naylor et al. (2023); Knipp et al. (2022).

standing rule that one artery must always be treated first.

A broader operative prevention plan

A comprehensive plan includes review of aortic atheroma and strategies to reduce embolization when technically appropriate, preservation of oxygenation and cerebral perfusion, avoidance of marked hemodynamic instability, and postoperative rhythm surveillance. The cardiac surgical and anesthesia teams determine operative techniques and monitoring. No individual adjunct, including cerebral oximetry, should be presented as a guarantee of stroke prevention. For the APRN, the actionable contribution is reliable baseline documentation, prompt recognition of change, medication continuity when appropriate, and escalation across disciplines.

The preoperative handoff should state the neurologic baseline, laterality and timing of prior events, confirmed carotid anatomy, relevant coronary history, renal function, and antithrombotic plan. After surgery, carry this information forward rather than making the next team reconstruct it from separate specialty notes. A concise shared problem representation can prevent both missed deterioration and reflex attribution of every symptom to the known stenosis.

After intervention: protect brain, heart, and airway

Postoperative monitoring should begin with a documented baseline and explicit escalation parameters. Neurologic observations include consciousness, speech, facial movement, limb strength, vision when assessable, and comparison with preexisting deficits. A new change warrants immediate assessment rather than being attributed to anesthesia, fatigue, or the patient's age. Check glucose and oxygenation while activating the appropriate emergency pathway; these checks should not create avoidable delay in stroke evaluation (Amin et al., 2023; Naylor et al., 2023).

After CEA, a developing neck hematoma can threaten the airway. Increasing swelling, dysphagia, voice change, stridor, respiratory distress, or agitation requires urgent surgical and airway support. Do not rely on a normal oxygen saturation as proof that an evolving

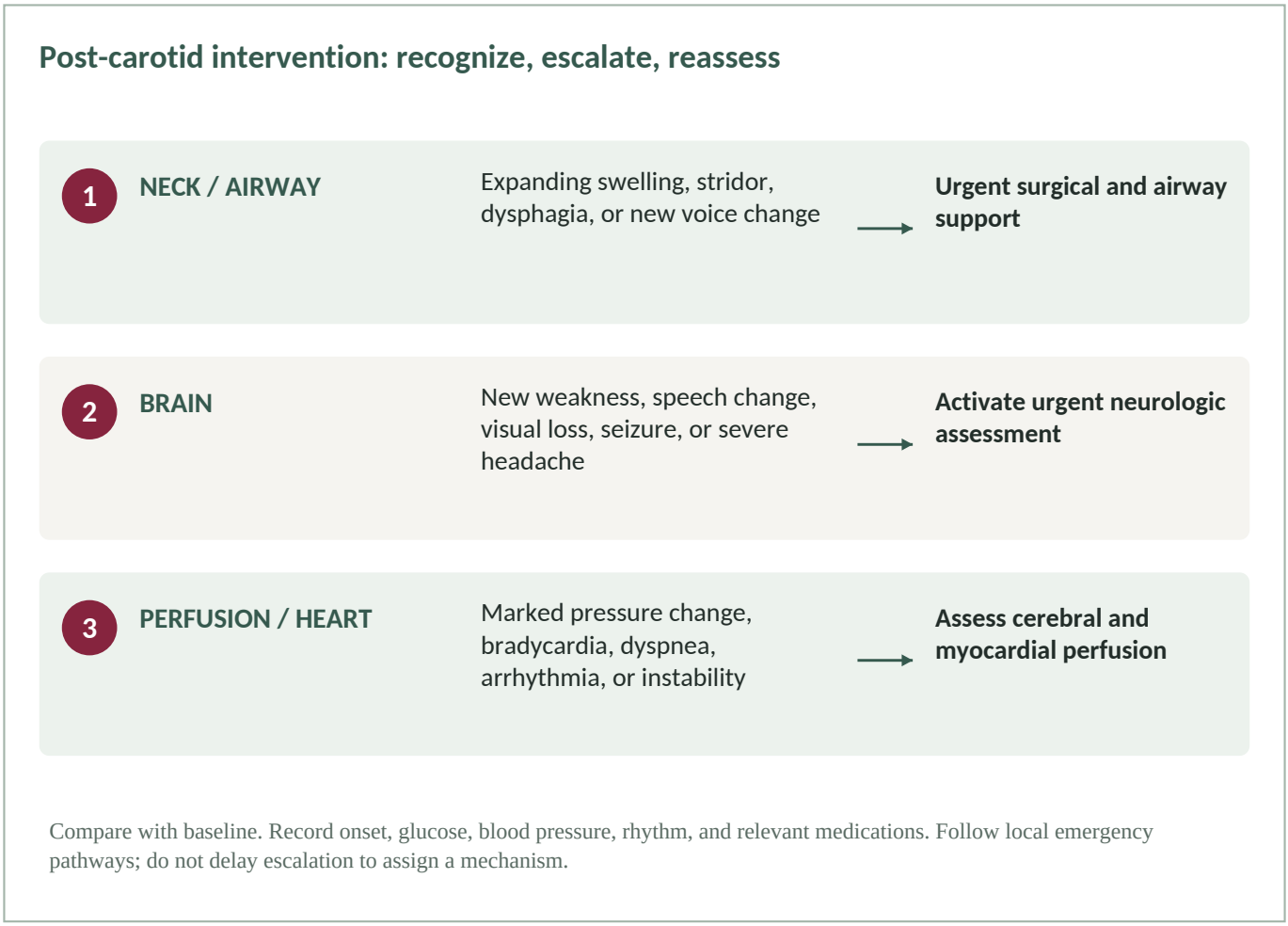


Figure 5. Early warning signs after carotid revascularization. This original safety display supports escalation, not bedside confirmation of a specific complication or a universal blood-pressure target. Evidence: Naylor et al. (2023); AHA (2026b).

mechanical airway problem is safe. Cranial nerve dysfunction can also produce voice or swallowing changes, but an urgent threat must be excluded before settling on a less immediately dangerous explanation.

After carotid stenting, baroreceptor stimulation can produce bradycardia and hypotension. Evaluate the rhythm, symptoms, volume status, access site, and perfusion, and follow the treating team's hemodynamic plan. In a patient with CAD, prolonged low pressure can threaten both cerebral and myocardial perfusion. Conversely, substantial postoperative hypertension deserves attention because cerebral hyperperfusion and hemorrhagic complications are possible after carotid revascularization. No single blood-pressure target is appropriate for every patient and procedure (Naylor et al., 2023).

New severe headache, seizure, or focal deficit after carotid treatment requires urgent assessment for hyperperfusion-related injury, ischemia, hemorrhage, or another cause. These symptoms are not an invitation to diagnose hyperperfusion at the bedside without imaging. Preventing an avoidable delay is more important than confidently choosing the wrong mechanism. Record symptom onset and trend, blood pressure, neurologic findings, antithrombotic exposure, and the actions already taken.

Case 5 | Headache is not a routine discharge symptom

A 71-year-old on the day after CEA develops a severe new ipsilateral headache and systolic pressure of 190 mmHg. A family member notices slowed speech. The APRN should activate urgent neurologic and surgical assessment, initiate prescribed blood-pressure management, and arrange appropriate imaging. Hyperperfusion-related injury is a concern, but ischemia and hemorrhage must also be considered. Giving an analgesic and proceeding with discharge would miss a potentially time-sensitive complication. The team needs the last normal examination, precise onset, medication history, and current neurologic findings (Naylor et al., 2023).

Do not miss perioperative myocardial injury

Chest pain may be absent or difficult to recognize after surgery. New dyspnea, hypotension, arrhythmia, pulmonary congestion, or otherwise unexplained deterioration should prompt cardiac assessment. Electrocardiography (ECG) and troponin testing follow the clinical presentation and perioperative risk pathway. Troponin interpretation requires the assay, serial change, symptoms, ECG, imaging, and competing causes of injury. An isolated elevated value does not by itself identify plaque rupture or mandate the same treatment as a spontaneous acute coronary syndrome (Thygesen et al., 2018; AHA, 2026b).

Postoperative atrial fibrillation raises questions about reversible triggers, rate or rhythm management, recurrence, and thromboembolic prevention. Document the episode and arrange follow-up; do not assume that conversion to sinus rhythm makes it irrelevant. Anticoagulation decisions must also account for recent surgery, intracranial findings, and the actual bleeding risk. In a patient who has both a stent-related antiplatelet requirement and an anticoagulation indication, the discharge plan should identify who owns the combined-regimen decision.

The safest surveillance plan is not simply “neuro checks and telemetry.” It specifies frequency, baseline, ordered pressure range, symptoms that require immediate escalation, and the contact pathway. It also accounts for communication barriers, residual aphasia, sensory impairment, and the patient's usual function. A family member's observation of an abrupt change can provide important information when the patient cannot describe it.

Plaque biomarkers: mechanism is not clinical utility

Inflammatory and immune pathways help explain why similarly narrowed arteries may behave differently. They are not yet a substitute for clinical assessment, vascular imaging, and established prevention. Before using any biomarker to make a treatment decision, ask whether the assay is standardized, whether it improves prediction beyond existing information, and whether acting on its result improves patient outcomes. Association, biological plausibility, and clinical utility are separate steps.

Intraplaque CRP is not the same as a blood hs-CRP result

Bonaventura and colleagues studied C-reactive protein within excised carotid plaques from 217 patients. Higher upstream plaque CRP content was associated with features of vulnerability and subsequent cardiovascular events; circulating high-sensitivity CRP did not show the same relationships in that cohort. The reported 2.9% tissue-content threshold was a study-specific histologic finding, not a serum laboratory cutoff or a validated rule for selecting CEA or coronary testing (Bonaventura et al., 2016).

This distinction prevents an important translational error. Tissue obtained during surgery is different from a screening blood test available before surgery. A finding after plaque removal cannot simply be assigned the role of a preoperative treatment-selection test. The study supports further investigation of local inflammation, but it does not justify promising that a CRP measurement can identify which asymptomatic patient needs carotid revascularization.

Anti-apolipoprotein A-I antibodies remain investigational

Research in patients undergoing CEA linked anti-apolipoprotein A-I IgG antibodies with a plaque phenotype characterized by inflammatory-cell infiltration, greater matrix metalloproteinase activity, and less collagen. Experimental studies supported possible proinflammatory mechanisms, including signaling through the toll-like receptor 2 (TLR2)/CD14 complex. These findings are relevant to biological understanding, but neither the original carotid cohort nor the mechanistic experiments established an outcome-improving clinical testing strategy (Montecucco et al., 2011; Pagano et al., 2012).

Do not prescribe a treatment because it modified an antibody-related response in cultured cells or an animal model. Do not use anti-apolipoprotein A-I IgG to determine whether a patient needs a stress test, PCI, CEA, or a mineralocorticoid receptor antagonist. These would be unsupported extensions of exploratory evidence. Research biomarkers can be discussed honestly with patients while making clear that routine care still rests on symptoms, anatomy, validated risk assessment, and treatments with clinical outcome evidence.

A durable follow-up plan and clinical synthesis

Follow-up should connect the treated or observed carotid lesion with the patient's broader cardiovascular care. Specify who will review symptoms, blood pressure, lipid response, medication tolerance, smoking status, and exercise capacity. Duplex surveillance intervals depend on the lesion, progression, procedure, symptoms, and local vascular protocol; an interval copied from another patient is not an individualized plan. New focal symptoms override the routine appointment schedule.

Teach patients and caregivers to seek emergency help for sudden weakness, speech change, or loss of vision—even when symptoms disappear. Explain separately which cardiac symptoms require urgent care. Ask for teach-back: "What would you do if vision disappeared in one eye for a few minutes?" and "Who will you contact before stopping a medicine prescribed for your stent?" These questions expose misunderstandings that an apparently successful explanation may leave hidden.

The final message is balanced. Carotid disease should prompt consideration of coronary disease because the atherosclerotic burden is systemic, but it does not mandate indiscriminate testing or automatic revascularization. Recently symptomatic carotid disease requires timely evaluation. Asymptomatic disease requires careful interpretation of contemporary evidence, including the distinct CREST-2 comparisons. CABG-related stroke has multiple mechanisms, so a carotid procedure cannot replace a broad perioperative prevention plan. The APRN's contribution is to connect symptoms, evidence, medication safety, patient preferences, and reliable follow-through into one coherent course of care.

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