

CAROTID STENOSIS

Contemporary Management Strategies

Audience: APRN | Contact hrs: 1.5 hrs | Pharmacology hrs: 0.0 hr

Expected revision: October 3, 2028

Pharmacology hours are included in the total contact hours.

Why carotid stenosis still matters

Atherosclerotic narrowing of the extracranial internal carotid artery remains an important, preventable cause of ischemic stroke. Management has changed substantially because contemporary medical therapy is more effective than the aspirin-only regimens used in the landmark endarterectomy trials, endovascular techniques have evolved, and patient selection has become more nuanced. The clinical question is no longer simply whether a stenosis is present. It is whether the lesion is truly symptomatic, how severe it is by a validated measurement method, how soon the patient is at risk for another event, and whether revascularization adds net benefit to intensive medical management.

For APRNs, the practical priorities are to recognize carotid-territory symptoms, verify that the imaging result is comparable to modern NASCET-based thresholds, accelerate referral when revascularization is indicated, optimize vascular risk reduction, and identify situations in which a procedural strategy may cause more harm than benefit. The original source appropriately emphasized CEA, CAS, and best medical treatment; this update retains that focus but incorporates current guideline recommendations, transcatheter revascularization, and the 2025 CREST-2 randomized results for asymptomatic disease.

Purpose and learning outcomes

By the end of this activity, the learner should be able to: (1) distinguish symptomatic from asymptomatic carotid stenosis using clinically meaningful neurologic criteria; (2) interpret stenosis severity using NASCET-based thresholds and appropriate imaging confirmation; (3) identify patients with symptomatic stenosis who are likely to benefit from carotid endarterectomy and explain why timing matters; (4) compare CEA, transfemoral carotid artery stenting, transcatheter artery revascularization, and intensive

medical management; (5) construct a contemporary vascular risk-reduction plan; and (6) apply patient-specific factors such as age, anatomy, prior radiation, comorbidity, and life expectancy to shared decision-making.

Defining symptomatic disease correctly

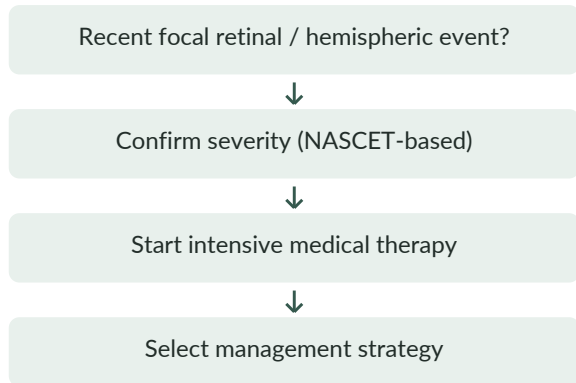
A carotid stenosis is considered symptomatic when an ipsilateral retinal or hemispheric ischemic event can reasonably be attributed to that artery. Typical events include amaurosis fugax, a transient ischemic attack with contralateral weakness or numbness, aphasia from dominant-hemisphere ischemia, or a nondisabling ischemic stroke. Current guidelines generally use a recent event within the preceding six months, but the absolute benefit of revascularization is greatest when the event is recent because early recurrent stroke risk is front-loaded (Kleindorfer et al., 2021; Naylor et al., 2023).

Dizziness, isolated syncope, generalized weakness, nonspecific visual blurring, or positive visual phenomena do not by themselves establish that the carotid lesion is symptomatic. Mislabeling an incidental stenosis as the cause of a nonspecific symptom can expose a patient to an unnecessary procedure while distracting from the actual diagnosis. When the neurologic syndrome is uncertain, the safest approach is prompt stroke or neurovascular evaluation rather than assuming causality from an imaging report.

Measuring stenosis: use a common language

Revascularization thresholds are derived from the North American Symptomatic Carotid Endarterectomy Trial (NASCET) method. In simplified terms, the narrowest residual lumen is compared with the normal distal internal carotid artery. The older European Carotid Surgery Trial method used an estimated original diameter at the diseased segment and therefore produced numerically higher percentages

Figure 1. Carotid stenosis: a practical management pathway



Symptomatic 70-99%: CEA usually preferred if surgical risk is acceptable

Symptomatic 50-69%: individualized benefit; urgent specialist review

Asymptomatic: intensive medical therapy first; revascularization selectively

Evidence basis: contemporary AHA/ASA, SVS, ESVS guidance and CREST-2 where applicable.

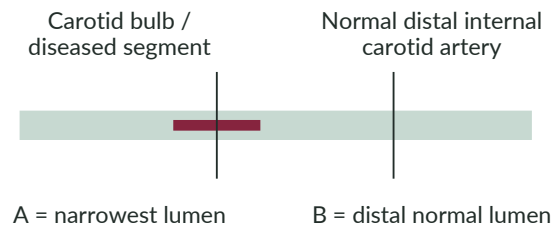
for the same lesion. Modern reports should state the method used or provide enough imaging detail to interpret the severity reliably.

Carotid duplex ultrasonography is usually the first-line noninvasive test in stable patients because it is inexpensive and provides both velocity and plaque information. CTA is rapid and is often favored in acute stroke pathways; MRA is useful when iodinated contrast is undesirable or when additional vascular characterization is needed. Catheter angiography is generally reserved for discordant noninvasive studies, procedural planning, or selected complex cases because it carries an invasive stroke risk. Calcification, tortuosity, tandem lesions, low-flow states, and laboratory-specific duplex criteria can all alter interpretation.

Atherosclerotic plaque and stroke mechanisms

Carotid atherosclerosis causes cerebral ischemia primarily through artery-to-artery embolism from an unstable plaque surface, not simply through gradual reduction in blood flow. Fibrous-cap disruption, ulceration, intraplaque hemorrhage, platelet activation, and superimposed thrombus can generate emboli to the retinal or cerebral circulation. Hemodynamic

Figure 2. NASCET method: the denominator matters



$$\text{NASCET stenosis} = (1 - A/B) \times 100\%$$

Use the same measurement method as the evidence thresholds.

Evidence basis: contemporary AHA/ASA, SVS, ESVS guidance and CREST-2 where applicable.

failure can contribute when stenosis is critical and collateral circulation is inadequate, but percentage narrowing alone does not fully describe plaque behavior. This explains why two patients with the same measured stenosis can have very different short-term stroke risk.

Plaque morphology is increasingly relevant to risk stratification. MRI can identify intraplaque hemorrhage; ultrasound can describe echolucency, surface irregularity, and progression; CTA can demonstrate ulceration and calcification. These features do not replace symptom status or stenosis severity, but they can help a vascular team decide whether an otherwise asymptomatic lesion appears to carry higher risk on medical therapy.

Symptomatic carotid stenosis: who benefits from revascularization?

For a patient with a recent nondisabling ischemic stroke or TIA and ipsilateral severe carotid stenosis, revascularization should be evaluated urgently. The strongest evidence supports CEA for 70% to 99% symptomatic stenosis when the patient is an acceptable surgical candidate and the center's perioperative stroke/death rate is low. AHA/ASA and SVS guidance also supports CEA for selected patients with 50% to 69% symptomatic stenosis, but the absolute benefit is smaller and depends on age, sex, comorbidity, anatomy, and the nature of the presenting event (Kleindorfer et al., 2021; AbuRahma et al., 2022).

Patients with less than 50% symptomatic stenosis do not benefit from CEA. Near-occlusion deserves separate consideration because the historical randomized data show less benefit than conventional severe stenosis; management should be individualized, particularly when symptoms recur despite intensive medical therapy. Complete carotid occlusion is not treated with CEA or routine stenting of the occluded segment.

The timing of CEA is a major determinant of benefit. In neurologically stable patients with nondisabling stroke or TIA, SVS recommends revascularization after 48 hours once clinically stable and within 14 days of symptom onset; AHA/ASA likewise emphasizes early treatment. Earlier surgery is not automatically appropriate after a large territorial infarction, major disability, intracranial hemorrhage, severe edema, or unstable neurologic status because hemorrhagic transformation and hyperperfusion risks may outweigh the immediate benefit.

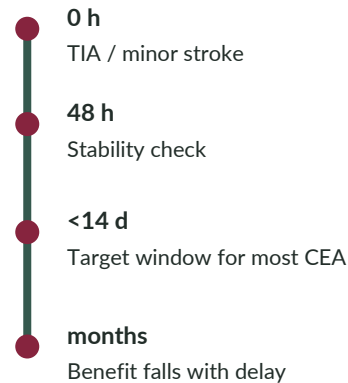
Case 1 - The clock matters

A 68-year-old man develops 20 minutes of right arm weakness and expressive aphasia that fully resolves. CTA shows 78% left internal carotid stenosis by NASCET criteria. MRI shows a small nondisabling cortical infarct without hemorrhage. He is neurologically stable 36 hours later. **Clinical interpretation:** This is a recent symptomatic severe ipsilateral stenosis. Intensive medical therapy should begin immediately, but it is not a substitute for urgent revascularization assessment. If he remains stable and operative risk is acceptable, CEA should generally be planned promptly after the first 48 hours and within 14 days rather than being deferred for months.

Diagnostic workflow and confirmation

When carotid disease is suspected after TIA or ischemic stroke, imaging should be obtained quickly enough that a treatment opportunity is not lost. Duplex ultrasound is a practical first test in many outpatient settings, but acute stroke programs often use CTA from the aortic arch through the intracranial circulation because it simultaneously identifies

Figure 5. Symptomatic stenosis: benefit is time-sensitive



Large infarct, hemorrhage, major disability, or unstable neurology may require a different timing strategy.

Evidence basis: contemporary AHA/ASA, SVS, ESVS guidance and CREST-2 where applicable.

extracranial stenosis, intracranial occlusion, tandem lesions, vascular variants, and arch anatomy. MRA is useful when iodinated contrast is undesirable and can also characterize plaque, but acquisition time, flow artifacts, and device compatibility must be considered.

A single discordant test should not automatically trigger an invasive procedure. If duplex suggests severe disease but CTA suggests only moderate narrowing, the clinician should review image quality, the laboratory's velocity criteria, cardiac output, contralateral occlusion, calcification, and whether the distal reference segment is adequate. Repeat high-quality noninvasive imaging or catheter angiography may be appropriate when the result would change management. The goal is not to obtain every possible study; it is to establish a defensible severity estimate before exposing a patient to procedural risk.

CEA versus carotid stenting

CEA remains the reference revascularization strategy for most standard-risk patients with symptomatic carotid stenosis. Compared with transfemoral CAS, CEA generally has a lower periprocedural stroke risk, whereas CAS may have a lower risk of myocardial infarction and avoids a cervical incision and cranial nerve injury. In CREST, the composite long-term outcome was similar, but the pattern of complications differed: stroke was more frequent with CAS and myocardial

infarction more frequent with CEA. Age modifies this tradeoff; outcomes with transfemoral CAS become less favorable as age increases, especially beyond approximately 70 years (Brott et al., 2010; Kleindorfer et al., 2021).

CAS can be reasonable when surgical risk is high because of prior neck radiation, restenosis after CEA, surgically inaccessible lesions, severe cardiopulmonary disease, contralateral laryngeal nerve palsy, or other unfavorable anatomy. Conversely, a heavily calcified aortic arch, marked arch tortuosity, or unfavorable plaque morphology can increase embolic risk with transfemoral access. Operator experience and center outcomes are central to the decision.

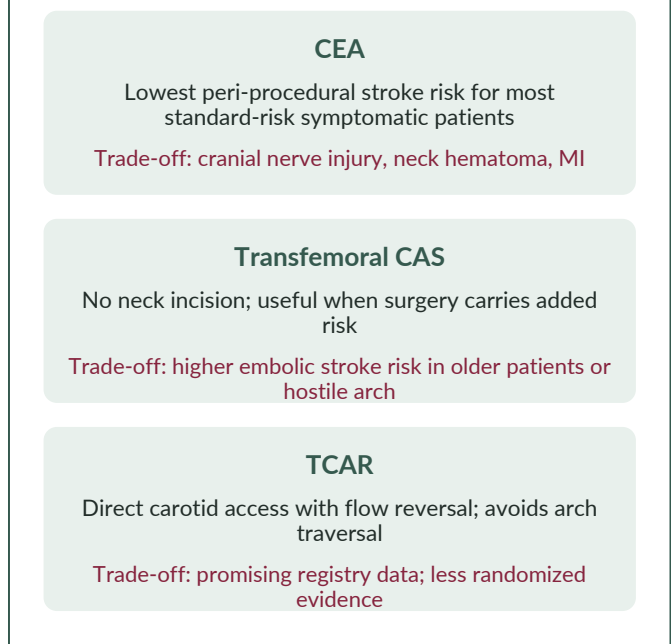
Where transcarotid artery revascularization fits

Transcarotid artery revascularization (TCAR) uses direct common carotid access and dynamic flow reversal to reduce embolization during stent deployment while avoiding navigation through the aortic arch. Registry data have suggested lower in-hospital stroke/death rates than transfemoral CAS in some matched analyses, including symptomatic populations, but the evidentiary base remains less mature than for CEA. Current decisions about TCAR should therefore be individualized and should account for anatomy, surgical risk, institutional expertise, and the fact that randomized comparisons with intensive medical therapy and CEA remain limited (Kleindorfer et al., 2021).

Case 3 - Choosing the modality

A 76-year-old man has a TIA from an 82% left ICA stenosis. He previously received high-dose neck radiation and has a hostile surgical field. CTA also shows a relatively favorable arch and access anatomy. **Clinical interpretation:** Standard CEA carries added local surgical risk. CAS or TCAR may be reasonable alternatives in an experienced center. The choice should be made jointly by vascular surgery, neurointerventional specialists, and the patient after discussing perioperative stroke, MI, cranial nerve injury, access anatomy, and center-specific outcomes.

Figure 3. Revascularization options: how the trade-offs differ



Evidence basis: contemporary AHA/ASA, SVS, ESVS guidance and CREST-2 where applicable.

Asymptomatic carotid stenosis: the evidence changed

Older trials such as ACAS and ACST-1 established a modest long-term stroke-prevention benefit from prophylactic CEA in selected patients with asymptomatic stenosis, but those studies predated current statin use, contemporary blood-pressure control, smoking cessation programs, and modern antithrombotic strategies. As medical therapy improved, the annual ipsilateral stroke risk in many asymptomatic cohorts fell substantially. That changed the risk-benefit equation because even a low procedural complication rate can offset a small absolute stroke reduction.

The 2025 CREST-2 publication materially updated this discussion. CREST-2 consisted of two parallel randomized trials in patients with high-grade asymptomatic stenosis who received intensive medical management. In the stenting trial, the 4-year primary-outcome rate was 2.8% with stenting plus medical therapy versus 6.0% with medical therapy alone. In the endarterectomy trial, the corresponding rates were 3.7% and 5.3%, a difference that was not statistically significant. These findings support a more individualized, evidence-aware discussion

rather than treating all asymptomatic stenoses above one numeric threshold the same (Brott et al., 2025). The results do not mean that every asymptomatic patient should undergo stenting. CREST-2 enrolled selected patients and was conducted in experienced centers; procedural events occurred early, whereas any stroke-prevention advantage accumulated over time. Life expectancy, procedural risk, anatomy, patient preferences, and the quality of ongoing medical therapy remain essential. A 2026 CREST-2 cognitive analysis also found no cognitive benefit from adding CEA or CAS to intensive medical therapy, so revascularization should not be recommended solely to improve cognition in otherwise asymptomatic patients.

Case 2 - Not every dizziness episode is carotid disease

A 74-year-old woman with hypertension has recurrent brief light-headed spells when standing. Duplex ultrasound obtained during an outpatient workup reports 72% right ICA stenosis. She has never had monocular visual loss, focal weakness, aphasia, or hemispheric sensory loss. **Clinical interpretation:** The stenosis is asymptomatic unless a true ipsilateral retinal or hemispheric ischemic event is identified. Her orthostatic symptoms need their own evaluation. Management should prioritize intensive medical therapy and a vascular consultation focused on individualized asymptomatic-risk assessment, not automatic intervention.

Identifying higher-risk asymptomatic disease

Guidelines continue to recognize that some asymptomatic plaques may carry greater future stroke risk. Features that can support specialist consideration include progression of stenosis, silent ipsilateral infarction, spontaneous microemboli on transcranial Doppler, impaired cerebrovascular reserve, echolucent or ulcerated plaque, and intraplaque hemorrhage on MRI. None of these findings should be interpreted in isolation; they refine risk rather than replace the overall clinical context. ESVS guidance emphasizes considering revascularization for selected patients with 60% to 99% asymptomatic stenosis when one or more higher-risk clinical or

imaging features are present and expected procedural risk and life expectancy are favorable (Naylor et al., 2023).

Routine population screening for asymptomatic carotid stenosis is not recommended. Selective testing may be reasonable in patients with multiple vascular risk factors when the result would change management, but screening should not become a pathway to intervention in patients who would not otherwise be candidates for treatment.

Case 4 - Asymptomatic disease after CREST-2

A 71-year-old woman is found to have 75% asymptomatic ICA stenosis during evaluation of a cervical bruit. She is independent, adherent to therapy, has no silent infarct on MRI, and has no high-risk plaque features. **Clinical interpretation:** The discovery of a high-grade stenosis no longer mandates prophylactic CEA. Intensive medical management is essential. The 2025 CREST-2 results should be part of shared decision-making; any intervention should depend on procedural risk, life expectancy, plaque-risk features, anatomy, and the patient's values rather than stenosis percentage alone.

What modern medical therapy actually means

Best or intensive medical therapy is an active treatment strategy rather than a passive alternative to intervention. It requires measurable targets and follow-up. Antiplatelet therapy is used for noncardioembolic disease unless contraindicated. Lipid management generally begins with a high-intensity or maximally tolerated statin, with ezetimibe or a PCSK9 inhibitor added when necessary to reach the selected LDL-C goal. Blood-pressure therapy should be titrated over time, with most secondary-prevention patients managed toward less than 130/80 mm Hg when tolerated. Smoking cessation should include counseling plus pharmacologic support when appropriate, and diabetes therapy should address both glycemic control and global cardiovascular risk.

Medication reconciliation is particularly important around revascularization. CEA and stenting do not use identical antiplatelet strategies, and local pro-

protocols may differ. ESVS recommends lower-dose aspirin rather than doses above 325 mg for patients undergoing CEA and provides specific options for combination therapy in recently symptomatic patients. CAS generally requires dual antiplatelet therapy around the procedure because of acute stent thrombosis risk. The exact regimen should be directed by the treating neurovascular or vascular team, with attention to bleeding risk, drug intolerance, urgent surgery, and concomitant anticoagulation.

Intensive medical management is foundational

Every patient with atherosclerotic carotid disease needs aggressive vascular risk reduction whether or not revascularization is performed. The plan generally includes antiplatelet therapy for noncardioembolic cerebrovascular disease, high-intensity or maximally tolerated lipid-lowering therapy, blood-pressure control, diabetes management, smoking cessation, physical activity, dietary improvement, and treatment of other vascular risk factors. For secondary prevention after ischemic stroke or TIA, AHA/ASA supports an LDL-cholesterol goal below 70 mg/dL for many patients with atherosclerotic disease and a blood-pressure target below 130/80 mm Hg for most patients when tolerated (Kleindorfer et al., 2021).

Short-term dual antiplatelet therapy is appropriate only in defined situations. For example, patients with early-arriving minor ischemic stroke or high-risk TIA may receive aspirin plus clopidogrel for a limited course under a stroke protocol, but long-term dual therapy is not routinely recommended because bleeding risk rises without a corresponding long-term benefit. ESVS provides specific perioperative antiplatelet strategies for patients proceeding to CEA or CAS; these should follow local neurovascular protocols and operator preference rather than being improvised at the bedside (Naylor et al., 2023).

Statins remain first-line lipid-lowering therapy. Ezetimibe and PCSK9 inhibitors may be added when LDL goals are not reached or when statin intolerance limits therapy. Antihypertensive therapy should avoid both chronic undertreatment and overly abrupt reductions in a patient with acute cerebral ischemia. Diabetes treatment should follow current

cardiovascular-risk reduction principles, including use of agents with proven cardiovascular benefit when otherwise appropriate.

Figure 4. Intensive medical management is the foundation

Antiplatelet

Noncardioembolic prevention

LDL lowering

High-intensity statin; add-on therapy as needed

BP control

Generally <130/80 mm Hg when tolerated

Smoking

Complete cessation support

Diabetes

Individualized vascular-risk strategy

Activity & diet

Sustainable lifestyle change

Evidence basis: contemporary AHA/ASA, SVS, ESVS guidance and CREST-2 where applicable.

Special situations

Prior neck radiation can produce long-segment, fibrotic carotid disease and may increase the technical difficulty of CEA. Both CEA and stenting can be reasonable, and ESVS recommends individualized selection in recently symptomatic patients with prior cervical radiation. Restenosis after prior CEA is another setting in which endovascular therapy may be attractive. Conversely, severe arch atheroma, complex tortuosity, and advanced age may favor CEA over transfemoral CAS when surgery is otherwise acceptable.

Patients who require coronary artery bypass surgery and also have carotid stenosis need multidisciplinary review rather than routine prophylactic carotid intervention. Stroke around CABG is often multifactorial, and asymptomatic unilateral carotid stenosis may be a marker of systemic atherosclerosis rather than the direct cause. Symptomatic high-grade stenosis, bilateral severe disease, or contralateral occlusion changes the discussion, but sequence and modality should be individualized.

Pregnancy, severe chronic kidney disease, active malignancy, and limited life expectancy alter the balance between imaging, medical therapy, and intervention. The expected time to benefit matters

especially in asymptomatic disease; a procedure with an immediate complication risk is difficult to justify when competing mortality risk is high.

Periprocedural complications and surveillance

After CEA, early complications include ipsilateral or contralateral stroke, myocardial infarction, cranial nerve injury, wound hematoma, blood-pressure instability, and hyperperfusion syndrome. After CAS or TCAR, embolic stroke, bradycardia, hypotension, access-site complications, contrast exposure, and acute or later in-stent restenosis require attention. New neurologic deficits should never be attributed to sedation or postoperative fatigue until stroke and other urgent causes have been excluded.

Duplex surveillance schedules vary by procedure, anatomy, and local practice. Surveillance is most useful when a detected change would alter management, such as restenosis after CEA or in-stent restenosis after CAS. Patients should also be reassessed for recurrent retinal or hemispheric symptoms at every visit. A normal postoperative course does not eliminate the need for long-term vascular prevention because coronary, peripheral, and cerebrovascular atherosclerosis share the same underlying biology.

Nursing and APRN priorities around carotid revascularization

Before revascularization, confirm the neurologic baseline, blood pressure, antithrombotic regimen, renal function when contrast is planned, allergy history, and the timing and nature of the qualifying event. After CEA or stenting, frequent neurologic and hemodynamic assessment is essential. New weakness, aphasia, visual loss, severe headache, seizure, reduced consciousness, or neck swelling requires immediate escalation.

Hyperperfusion syndrome is uncommon but potentially catastrophic. It may present with ipsilateral headache, seizures, focal deficits, or intracerebral hemorrhage, often in the setting of postoperative hypertension. Cranial nerve dysfunction, wound hematoma, bradycardia, hypotension, access-site bleeding, and contrast-related kidney injury are additional procedure-specific concerns. A rapidly

expanding neck hematoma after CEA is an airway emergency.

Patient education should make the long-term message explicit: revascularization treats one lesion; it does not cure systemic atherosclerosis. Antiplatelet adherence, lipid lowering, blood-pressure management, smoking cessation, physical activity, and follow-up imaging remain necessary after a technically successful procedure.

Shared decision-making: translating evidence into a patient choice

A high-quality carotid discussion should present absolute risks rather than only relative percentages. For symptomatic severe stenosis, the untreated early stroke risk is high enough that the benefit of timely CEA is often substantial. For asymptomatic disease, the annual stroke risk on contemporary therapy may be low, so a one-time procedural risk can consume much of the potential benefit. CREST-2 makes this contrast easier to explain: intensive medical therapy is not a control-group afterthought; it is a core treatment, and the incremental value of revascularization depends on the modality and the patient's baseline risk.

Patients should understand that center and operator outcomes matter. A procedure supported by trial data may not offer the same net benefit if local complication rates exceed accepted thresholds. The discussion should also include the types of complications that differ between procedures, expected recovery, need for antiplatelet therapy, imaging follow-up, and the possibility that a technically successful intervention may not improve unrelated symptoms such as chronic dizziness or cognitive complaints.

Clinical synthesis

Management of carotid stenosis is best framed as a sequence of decisions. First, determine whether the neurologic event is truly carotid-territory and recent. Second, confirm stenosis severity with a validated method. Third, start or optimize intensive medical therapy. Fourth, for symptomatic 50% to 99% disease, obtain rapid specialist evaluation, with CEA favored for most standard-risk patients and treatment

ideally within 14 days when clinically appropriate. Fifth, use CAS or TCAR selectively when patient factors or anatomy favor an endovascular strategy. Finally, approach asymptomatic disease with greater restraint: modern medical therapy is mandatory, and any revascularization decision should be individualized in light of CREST-2, procedural risk, life expectancy, plaque-risk features, and patient preferences.

Putting the plan into practice

For an APRN evaluating a newly identified carotid stenosis, the chart should clearly document the qualifying symptom, side, date, neurologic deficit, imaging modality, measurement method when known, and severity. A vague entry such as 'carotid disease - refer vascular' does not communicate the urgency of a recent symptomatic lesion. Conversely, documenting that a lesion is asymptomatic prevents nonspecific dizziness or syncope from being misinterpreted later as a carotid-territory event.

Urgent escalation is appropriate for new or recurrent focal neurologic symptoms, crescendo TIAs, amaurosis fugax with a known ipsilateral high-grade stenosis, or a nondisabling ischemic stroke with a revascularizable lesion. Stable asymptomatic disease can usually be addressed through deliberate outpatient risk-factor optimization and specialist review. In both settings, medication reconciliation, smoking status, blood-pressure trend, lipid values, diabetes control, renal function, and adherence barriers should be treated as core components of the carotid plan rather than separate problems.

At follow-up, the most useful question is not simply whether the artery has been treated. Ask whether the patient has had any new focal neurologic events, whether preventive targets are being achieved, whether antithrombotic therapy is appropriate for the current indication, and whether surveillance imaging is needed based on the procedure and local protocol. This longitudinal approach turns carotid care from a one-time procedural decision into sustained stroke prevention.

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