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Review Paper

Stroke Prevention: A Comprehensive Review of Risk Factors, Lifestyle Measures, Pharmacological Strategies and Long-Term Care

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ABSTRACT

Stroke remains a major cause of death, disability and loss of independence worldwide, yet a large proportion of strokes can be prevented by identifying modifiable risks early and managing them consistently. Stroke occurs when cerebral blood flow is interrupted by vascular occlusion or when a cerebral vessel ruptures. Prevention therefore requires more than one intervention: blood pressure control, lipid management, diabetes care, smoking cessation, healthy diet, regular physical activity, weight management, appropriate treatment of atrial fibrillation and other cardiac disorders, and adherence to evidence-based antithrombotic therapy after a previous ischemic event all contribute to risk reduction. This review summarizes the basic concepts of stroke, major modifiable and non-modifiable risk factors, primary and secondary prevention, diagnostic assessment of risk, lifestyle measures, pharmacological approaches and the role of rehabilitation and patient education. Particular attention is given to hypertension, dyslipidemia, diabetes, atrial fibrillation, tobacco exposure, alcohol use, diet, physical inactivity and medication adherence. The review also highlights the importance of individualized care, because the best prevention strategy depends on stroke mechanism, comorbidities, bleeding risk, age and social circumstances. Prevention is most effective when patients, physicians, pharmacists, nurses and rehabilitation professionals work together and when risk-factor control continues over the long term

INTRODUCTION

Stroke is a sudden neurological disorder caused by an interruption of blood supply to part of the brain or by bleeding within the brain. The resulting injury may lead to weakness, speech disturbance, visual impairment, cognitive problems or death. Although stroke is often described as an acute event, the factors that produce it usually develop over many years. For this reason, prevention

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begins well before the first symptoms appear and continues after a stroke or transient ischemic attack (TIA). [1–4]

Recent global estimates underline the continuing public-health importance of stroke. The World Health Organization reports that stroke was among the leading causes of death and disability globally in 2021, with about 11.9 million new strokes and an estimated 93.8 million people living with stroke. A large share of the burden is linked to modifiable exposures such as high blood pressure, unhealthy diet, tobacco use, high LDL cholesterol, high blood glucose, excess body weight, physical inactivity, kidney dysfunction, air pollution and harmful alcohol use. [1,2]

The encouraging message is that stroke risk is not fixed. Regular screening, healthy behavior and appropriate treatment of vascular risk factors can substantially lower risk. The 2024 American Heart Association/American Stroke Association (AHA/ASA) guideline emphasizes prevention across the life course and places greater attention on healthy behaviors, social determinants of health, sex-specific risks and integrated cardiovascular risk reduction. [3] For people who have already experienced stroke or TIA, secondary prevention is equally important because recurrence risk remains clinically significant. [4,5]

2. Types of Stroke

Stroke is broadly classified into ischemic and hemorrhagic stroke. A TIA is not a completed stroke but is an important warning event and should be treated as an emergency because it identifies a period of increased risk. [1,4]

2.1 Ischemic stroke

Ischemic stroke occurs when a cerebral artery becomes blocked, reducing oxygen and glucose delivery to brain tissue. Common mechanisms include large-artery atherosclerosis, small-vessel disease and cardioembolism, particularly in patients with atrial fibrillation. Other causes include arterial dissection, uncommon vasculopathies, thrombophilic states and strokes of undetermined cause. Prevention is therefore mechanism-specific: antiplatelet therapy is generally used for non-cardioembolic ischemic stroke, whereas anticoagulation is used when a suitable cardioembolic source such as atrial fibrillation is identified. [4,6,7]

2.2 Hemorrhagic stroke

Hemorrhagic stroke results from rupture of a cerebral blood vessel. Intracerebral hemorrhage and subarachnoid hemorrhage are the major forms. Long-standing or severe hypertension is a major modifiable contributor to intracerebral hemorrhage, while aneurysms and vascular malformations are important causes of subarachnoid hemorrhage. Prevention therefore focuses strongly on blood-pressure control, avoidance of unnecessary exposure to bleeding-promoting drugs and identification of structural vascular disorders when clinically indicated. [8,9]

2.3 Transient ischemic attack

TIA produces temporary focal neurological symptoms without persistent infarction detectable by standard tissue-based definitions. A TIA should never be dismissed simply because symptoms resolve. It provides an opportunity to identify the underlying vascular mechanism and rapidly address blood pressure, lipids, diabetes, atrial fibrillation, carotid disease, smoking and other risks. [4,10]

3. Pathophysiological Basis of Stroke Prevention

The vascular processes leading to stroke are interconnected. Hypertension promotes endothelial injury, arterial remodeling and small-

vessel damage. Atherosclerosis causes plaque formation and can lead to artery-to-artery embolism or local thrombosis. Dyslipidemia accelerates atherosclerotic plaque development, while diabetes contributes through endothelial dysfunction, inflammation, oxidative stress and accelerated vascular disease. Atrial fibrillation favors formation of thrombi in the atria, which may embolize to cerebral arteries. Smoking increases oxidative stress, platelet activation and vascular injury. [2,7,11]

These mechanisms explain why prevention works best as a combined strategy. Treating only one factor may leave substantial residual risk. For example, a patient with hypertension and atrial fibrillation needs both effective blood-pressure management and appropriate stroke-preventive anticoagulation. Similarly, lipid lowering is more effective when accompanied by smoking cessation, physical activity and dietary improvement. Prevention should therefore be individualized rather than reduced to a single drug or lifestyle recommendation. [3,4,7]

4. Stroke Risk Factors

Stroke risk factors can be divided into non-modifiable and modifiable factors. Age, sex, genetic susceptibility and a previous stroke or TIA cannot be changed, but they help determine the intensity of prevention. Modifiable factors are the main targets for intervention. [1–4]

4.1 Non-modifiable risk factors

Increasing age is strongly associated with stroke because vascular and cardiac disease accumulate over time.

Previous stroke or TIA is a major marker of recurrent risk and requires long-term secondary prevention.

Family history and genetic susceptibility can contribute to vascular risk.

Some sex-specific and reproductive factors influence risk, including hypertensive disorders of pregnancy and premature menopause. [3,4]

4.2 Modifiable risk factors

Hypertension is the most important modifiable risk factor for both ischemic and hemorrhagic stroke. Other major factors include cigarette smoking, dyslipidemia, diabetes mellitus, obesity, physical inactivity, unhealthy diet, atrial fibrillation and other heart disease, excessive alcohol use, kidney disease and certain drug exposures. Air pollution is increasingly recognized as an important population-level contributor. [1–3,11]

Table 1. Major stroke risk factors and prevention approach

Risk factor	Why it matters	Key prevention strategy
Hypertension	Promotes small-vessel disease, atherosclerosis and hemorrhage	Regular BP measurement; lifestyle measures; antihypertensive therapy when indicated
Smoking	Causes endothelial injury, thrombosis and accelerated atherosclerosis	Complete cessation; behavioral and pharmacological support
Dyslipidemia	Increases atherosclerotic vascular disease	Dietary improvement; statin or other lipid-lowering therapy when indicated
Diabetes	Accelerates vascular injury and atherosclerosis	Glycemic control, weight management and cardiovascular risk reduction

Atrial fibrillation	Promotes cardioembolic cerebral events	Risk assessment and anticoagulation when indicated
Obesity/inactivity	Associated with hypertension, diabetes and dyslipidemia	Regular activity, healthy diet and sustainable weight reduction
Excess alcohol	Raises BP and can increase arrhythmia and hemorrhage risk	Limit or avoid harmful alcohol use
Unhealthy diet	Contributes to hypertension, obesity and dyslipidemia	Mediterranean-style or other heart-healthy dietary pattern
Air pollution	Associated with vascular and cerebrovascular risk	Reduce exposure where feasible; population-level clean-air measures

5. Screening and Assessment for Stroke Risk

Prevention begins with identifying risk before symptoms occur. A routine cardiovascular assessment should include blood pressure, smoking and tobacco exposure, body weight or waist-related measures, physical activity, dietary pattern, lipid profile, glucose status and relevant medical history. Assessment should also consider atrial fibrillation, sleep disorders, kidney disease, pregnancy-related vascular risk and family history when clinically appropriate. [3,11,12]

For patients with suspected acute stroke, prevention is not a substitute for emergency care. Sudden facial weakness, arm or leg weakness, speech difficulty, vision loss, severe imbalance or a sudden unexplained severe headache should prompt immediate emergency evaluation. Brain imaging with CT or MRI helps distinguish ischemic from hemorrhagic disease, while vascular imaging, cardiac evaluation and laboratory testing may be used to identify the mechanism and guide secondary prevention. [1,5,8]

6. Primary Prevention of Stroke

7. Primary Prevention of Stroke

6.1 Blood pressure control

Blood pressure control is the central component of stroke prevention. Persistent hypertension damages both large and small cerebral vessels and is strongly associated with ischemic stroke and intracerebral hemorrhage. Regular measurement is important because hypertension is often asymptomatic. Treatment combines lifestyle modification with antihypertensive medication when clinically indicated. [1,3,11,12]

11. Lifestyle Modification

6.2 Lipid management

Elevated LDL cholesterol contributes to atherosclerotic disease and therefore increases vascular risk. Diet, physical activity and weight management are useful foundations, but many high-risk patients also require statin therapy. Evidence from the SPARCL trial showed that intensive atorvastatin therapy reduced recurrent stroke and major vascular events in selected patients after recent stroke or TIA. [14,15]

6.3 Diabetes management

Diabetes increases stroke risk through a combination of metabolic, endothelial and inflammatory mechanisms. Prevention requires sustained glycemic management together with attention to blood pressure, lipids, weight, kidney function and cardiovascular risk. The aim is not

simply to normalize a single laboratory value; rather, diabetes care should reduce the patient's overall vascular risk. [3,4,16]

6.4 Smoking cessation

Tobacco exposure is a major preventable cause of vascular disease. Smoking promotes endothelial dysfunction, platelet activation and atherosclerosis. Complete cessation is preferable to simply reducing the number of cigarettes. Counseling, behavioral support and pharmacological aids can improve the likelihood of successful quitting. Avoiding second-hand smoke is also desirable. [1–3,17]

6.5 Healthy diet

Diet influences stroke risk through several pathways, particularly blood pressure, lipid levels, body weight and glycemic control. A Mediterranean-style dietary pattern emphasizing vegetables, fruits, whole grains, legumes, nuts, fish and unsaturated fats has supportive evidence for cardiovascular and stroke prevention. Reducing sodium, processed foods, excess saturated fat and added sugars can further support risk reduction. [3,18]

6.6 Physical activity

Regular physical activity improves blood pressure, insulin sensitivity, lipid profile, body composition and cardiorespiratory fitness. For most adults, a practical goal is at least 150 minutes of moderate-intensity aerobic activity per week, or an equivalent combination of moderate and vigorous activity, with gradual progression according to fitness and medical status. Breaking up prolonged sitting and adding muscle-strengthening activities can complement aerobic exercise. [3,19]

6.7 Weight management

Overweight and obesity are linked to stroke through hypertension, diabetes, dyslipidemia, inflammation and sleep-disordered breathing. Sustainable weight management should focus on dietary quality, regular activity, adequate sleep and realistic behavioral goals rather than rapid weight loss. In selected patients with obesity and diabetes or high cardiovascular risk, modern weight-management and glucose-lowering therapies may have a role under medical supervision. [3,16]

6.8 Alcohol and substance use

Harmful alcohol use can increase blood pressure and may contribute to atrial fibrillation and hemorrhagic stroke. Prevention should focus on reducing harmful consumption and recognizing patients who need structured support. Illicit stimulants such as cocaine and amphetamines can also increase vascular and stroke risk and require appropriate counseling and treatment. [1,2,20]

6.9 Sleep and obstructive sleep apnea

Sleep disorders, particularly obstructive sleep apnea, are associated with hypertension and vascular risk. Patients with loud snoring, witnessed apneas, excessive daytime sleepiness or resistant hypertension may benefit from clinical assessment for sleep-disordered breathing. Addressing sleep health is increasingly considered part of comprehensive cardiovascular prevention. [3,21]

6.10 Atrial fibrillation and cardiac disease

10. Stroke Prevention in Atrial Fibrillation

7. Secondary Prevention After Stroke or TIA

8. Secondary Prevention of Stroke

7.1 Antiplatelet therapy

For most patients with non-cardioembolic ischemic stroke or TIA, antiplatelet therapy is a major component of secondary prevention. Aspirin, clopidogrel or aspirin combined with

extended-release dipyridamole may be used depending on the clinical situation. Short-term dual antiplatelet therapy can be beneficial after selected minor ischemic strokes or high-risk TIAs, but prolonged dual therapy increases bleeding risk and is not routinely used for long-term prevention. [4,23,24]

7.2 Anticoagulation for atrial fibrillation

When atrial fibrillation is responsible for cardioembolic risk, anticoagulation rather than antiplatelet therapy is generally the preventive strategy, unless contraindicated. Direct oral anticoagulants are commonly preferred for many patients with non-valvular atrial fibrillation, while warfarin remains important in selected situations such as mechanical heart valves. Choice and timing depend on stroke severity, bleeding risk, kidney function, age, interactions and the specific cardiac condition. [4,22,25–27]

7.3 Lipid lowering after stroke

High-intensity statin therapy is widely used in secondary prevention when appropriate. The purpose is not only to lower LDL cholesterol but also to reduce future atherosclerotic vascular events. Treatment intensity and LDL goals should be individualized according to stroke mechanism and overall cardiovascular risk. [4,14,15]

7.4 Blood pressure after stroke

Once the acute phase is over and the patient is clinically stable, long-term blood-pressure reduction is a central element of secondary prevention. A commonly recommended target for many patients is below 130/80 mmHg, although the target should be adapted when orthostatic symptoms, frailty or other conditions make aggressive lowering unsafe. [4,10,12]

7.5 Carotid and vascular disease

Patients with non-cardioembolic ischemic stroke or TIA should be assessed for relevant extracranial or intracranial arterial disease when the clinical picture suggests it. Symptomatic severe carotid stenosis may require revascularization in appropriately selected patients, while all patients benefit from intensive medical risk-factor management. [4,28]

8. Pharmacological Strategies for Stroke Prevention

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Table 2. Common pharmacological approaches

Drug group	Examples	Main preventive role	Important considerations
Antihypertensives	ACE inhibitors, ARBs, thiazide-type diuretics, calcium-channel blockers, others	Reduce vascular and stroke risk by controlling BP	Individualize target; monitor adherence, electrolytes and adverse effects
Statins	Atorvastatin, rosuvastatin	Reduce LDL and recurrent vascular events	Monitor tolerance; consider intensity and LDL response
Antiplatelets	Aspirin, clopidogrel	Prevent platelet-mediated thrombosis in non-cardioembolic disease	Assess bleeding risk; avoid unnecessary long-term dual therapy
Anticoagulants	Apixaban, rivaroxaban, dabigatran, edoxaban, warfarin	Prevent cardioembolic stroke in appropriate AF or other indications	Assess renal function, interactions and bleeding risk

Glucose-lowering therapy	Metformin and selected newer agents according to indication	Reduce metabolic and cardiovascular risk in diabetes	Individualize by comorbidity, kidney function and hypoglycemia risk

9. Role of Lifestyle Modification in Long-Term Prevention

11. Lifestyle Modification

Family involvement can be particularly useful after stroke because dietary preparation, medication organization, transport to appointments and rehabilitation often depend on caregivers. Prevention should also consider health literacy, financial constraints, food availability and access to healthcare. These social factors can influence whether a theoretically effective prevention plan is actually followed. [3,29]

10. Rehabilitation and Prevention of Recurrent Disability

Prevention does not end when the acute stroke is treated. Rehabilitation helps survivors regain function, adapt to disability and return to daily activities. Physical therapy addresses strength, mobility and balance; occupational therapy supports independence in daily tasks; speech and language therapy addresses communication and swallowing; and cognitive and psychological interventions may be required for memory, attention, mood and emotional adjustment. [1,30]
Depression, anxiety, cognitive impairment, spasticity, dysphagia and falls can interfere with recovery and indirectly reduce adherence to prevention. Regular follow-up should therefore assess both vascular risk factors and functional needs. The multidisciplinary team should communicate clearly so that medication changes, dietary recommendations and rehabilitation goals are mutually consistent. [4,30]

11. Medication Adherence and the Role of the Pharmacist

Medication adherence is a practical determinant of prevention. Patients may miss medicines because of side effects, complex schedules, cost, forgetfulness, misunderstanding or the belief that treatment is unnecessary when they feel well. Pharmacists can help by reviewing the medication list, identifying interactions, simplifying regimens when possible, counseling about adverse effects and reinforcing the importance of continued therapy. [4,29]
For anticoagulants and antiplatelets, counseling should include bleeding precautions and the importance of informing healthcare professionals before procedures. For statins and antihypertensives, patients should understand that benefits depend on continued use rather than short-term symptom relief. Medication reconciliation after hospitalization is particularly valuable because stroke admissions often involve multiple medication changes. [4,22]

12. Special Considerations in Stroke Prevention

12.1 Women and pregnancy-related risk

Some stroke risks are specific to women or occur more often in particular reproductive settings. Hypertensive disorders of pregnancy, premature menopause and certain hormonal exposures can contribute to later vascular risk. The 2024 AHA/ASA primary prevention guideline therefore recommends attention to sex-specific and reproductive factors when assessing stroke risk. [3]

12.2 Older adults

Older adults often have several competing risks, including hypertension, atrial fibrillation, kidney disease, falls and polypharmacy. Prevention should therefore balance stroke reduction with treatment burden, bleeding risk and functional status. Blood-pressure targets and antithrombotic decisions should be individualized rather than based on age alone. [3,4,22]

12.3 Chronic kidney disease

Kidney dysfunction is associated with increased cardiovascular and stroke risk. Risk-factor control may be more difficult because patients can have altered drug clearance, anemia, electrolyte abnormalities and multiple medications. Kidney function should therefore be considered when selecting and dosing antihypertensive and antithrombotic therapies. [1,4]

12.4 Health inequalities and access to care

A prevention plan can fail when a patient cannot afford medicines, reach a clinic, obtain healthy foods or understand health information. Social determinants of health should be considered as part of risk assessment. Community-based screening, affordable medicines, culturally appropriate education and coordinated primary care can make prevention more achievable. [3,29]

13. Emerging and Future Directions

Stroke prevention is increasingly moving toward integrated cardiovascular and brain health rather than isolated treatment of single risk factors. Digital blood-pressure monitoring, electronic reminders, telepharmacy, remote follow-up and wearable rhythm monitoring may help identify uncontrolled risk or intermittent atrial fibrillation. Their value depends on accessibility, data quality and appropriate clinical follow-up. [3,22]

Newer metabolic therapies are also changing cardiovascular prevention. Selected glucose-lowering and weight-management medicines may reduce cardiovascular events in people with diabetes or obesity and high cardiovascular risk. However, these medicines should be used according to approved indications and individual clinical assessment rather than as universal stroke-prevention drugs. [3,16]

Population-level strategies remain essential. Reducing tobacco exposure, improving access to healthy food, reducing dietary sodium, improving air quality and strengthening primary healthcare can influence risk across entire communities. The growing contribution of environmental factors, including air pollution and high ambient temperature, suggests that future stroke prevention will require both clinical and public-health action. [1,2]

14. Practical Stroke Prevention Checklist

Measure and manage blood pressure regularly.

Do not smoke; provide support for complete tobacco cessation.

Maintain a healthy dietary pattern with adequate fruits, vegetables, whole grains and fiber and lower sodium intake.

Be physically active and reduce prolonged sedentary time.

Maintain a healthy weight and address obesity when present.

Check and treat dyslipidemia according to overall cardiovascular risk.

Screen for and manage diabetes and other metabolic disorders.

Recognize and appropriately treat atrial fibrillation and other relevant cardiac conditions.

Limit harmful alcohol use and avoid illicit stimulant drugs.

After stroke or TIA, identify the mechanism and use appropriate antiplatelet or anticoagulant therapy.

Continue statin and blood-pressure therapy when prescribed and monitor adherence.

Attend follow-up and rehabilitation appointments. Educate patients and caregivers about warning symptoms and the need for emergency care.
Address practical barriers such as medication cost, health literacy and access to healthy food and healthcare.

CONCLUSION

Stroke prevention is a continuous process rather than a single intervention. The strongest approach combines early identification of vascular risk with sustained lifestyle improvement and evidence-based pharmacological treatment. Blood-pressure control remains central, while lipid management, diabetes care, smoking cessation, physical activity, healthy diet, weight management and appropriate management of atrial fibrillation address other major contributors to risk. After stroke or TIA, prevention must become more individualized because treatment depends on the mechanism of the event and the patient's bleeding and cardiovascular risks. [1–4]
The practical goal is to make prevention achievable in everyday life. Patients need clear advice, affordable treatment, regular follow-up and support from healthcare professionals and family members. Pharmacists can strengthen adherence and medication safety, while rehabilitation professionals help reduce disability and improve independence. When these measures are combined and maintained over time, many first and recurrent strokes can be prevented or their impact substantially reduced. [3,4,30]

REFERENCES

1. World Health Organization. Stroke. Fact sheet. 19 December 2025. Geneva: WHO; 2025.
2. GBD 2021 Stroke Risk Factor Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. 2024;23(10):973–1003. doi:10.1016/S1474-4422(24)00369-7.
3. Bushnell C, Kernan WN, Sharrief AZ, et al. 2024 Guideline for the Primary Prevention of Stroke: A Guideline From the American Heart Association/American Stroke Association. Stroke. 2024. doi:10.1161/STR.0000000000000475.
4. Kleindorfer DO, Towfighi A, Chaturvedi S, et al. 2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack. Stroke. 2021;52:e364–e467. doi:10.1161/STR.0000000000000375.
5. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update. Stroke. 2019;50:e344–e418. doi:10.1161/STR.0000000000000211.
6. Klijn CJM, Paciaroni M, Berge E, et al. Antithrombotic treatment for secondary prevention of ischaemic stroke and transient ischaemic attack. Eur Stroke J. 2019;4(3):198–223.
7. Hankey GJ. Stroke. Lancet. 2017;389:641–654. doi:10.1016/S0140-6736(16)30962-X.
8. Greenberg SM, Ziai WC, Cordonnier C, et al. 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage. Stroke. 2022;53:e282–e361. doi:10.1161/STR.0000000000000407.
9. Hoh BL, Ko NU, Amin-Hanjani S, et al. 2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage. Stroke. 2023;54:e314–e370. doi:10.1161/STR.0000000000000436.
10. Kleindorfer DO, Bernstein R, Dorsch M. Primary prevention of stroke: a review of guidelines and recommendations. Curr Atheroscler Rep. 2017;19:39.

11. O'Donnell MJ, Chin SL, Rangarajan S, et al. Global and regional effects of potentially modifiable risk factors associated with acute stroke in 32 countries (INTERSTROKE): a case-control study. Lancet. 2016;388:761–775. doi:10.1016/S0140-6736(16)30506-2.
12. Whelton PK, Carey RM, Aronow WS, et al. 2017 ACC/AHA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults. Hypertension. 2018;71:e13–e115.
13. Wright JT Jr, Williamson JD, Whelton PK, et al. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med. 2015;373:2103–2116. doi:10.1056/NEJMoa1511939.
14. Amarenco P, Bogousslavsky J, Callahan A 3rd, et al. High-dose atorvastatin after stroke or transient ischemic attack. N Engl J Med. 2006;355:549–559. doi:10.1056/NEJMoa061894.
15. Szarek M, Amarenco P, Callahan A 3rd, et al. Atorvastatin reduces first and subsequent vascular events across vascular territories: the SPARCL trial. J Am Coll Cardiol. 2020;75:2110–2118. doi:10.1016/j.jacc.2020.03.015.
16. American Diabetes Association Professional Practice Committee. Standards of Care in Diabetes—2026. Diabetes Care. 2026;49(Suppl 1).
17. Hackam DG, Spence JD. Combining multiple approaches for the secondary prevention of vascular events after stroke: a quantitative modeling study. Stroke. 2007;38:1881–1885.
18. Estruch R, Ros E, Salas-Salvadó J, et al. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. N Engl J Med. 2018;378:e34.
19. Lee CD, Folsom AR, Blair SN. Physical activity and stroke risk: a meta-analysis. Stroke. 2003;34:2475–2481.
20. Mostofsky E, Chahal HS, Mukamal KJ, Rimm EB, Mittleman MA. Alcohol and immediate risk of cardiovascular events: a systematic review and dose-response meta-analysis. Circulation. 2016;133:979–987.
21. McNicholas WT, Bonsignore MR. Sleep apnoea as an independent risk factor for cardiovascular disease: current evidence, basic mechanisms and research priorities. Eur Respir J. 2007;29:156–178.
22. Van Gelder IC, Rienstra M, Bunting KV, et al. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Eur Heart J. 2024;45:3314–3414.
23. Wang Y, Wang Y, Zhao X, et al. Clopidogrel with aspirin in acute minor stroke or transient ischemic attack. N Engl J Med. 2013;369:11–19. doi:10.1056/NEJMoa1215340.
24. Johnston SC, Easton JD, Farrant M, et al. Clopidogrel and aspirin in acute ischemic stroke and high-risk TIA. N Engl J Med. 2018;379:215–225. doi:10.1056/NEJMoa1800410.
25. Granger CB, Alexander JH, McMurray JJV, et al. Apixaban versus warfarin in patients with atrial fibrillation. N Engl J Med. 2011;365:981–992.
26. Connolly SJ, Ezekowitz MD, Yusuf S, et al. Dabigatran versus warfarin in patients with atrial fibrillation. N Engl J Med. 2009;361:1139–1151.
27. Patel MR, Mahaffey KW, Garg J, et al. Rivaroxaban versus warfarin in nonvalvular atrial fibrillation. N Engl J Med. 2011;365:883–891.
28. Naylor AR, Ricotta JJ, de Borst GJ, et al. Management of asymptomatic and symptomatic carotid disease:

recommendations and evidence. Eur J Vasc Endovasc Surg. 2018;55:3–81.

29. World Health Organization. Integrating stroke services in health-care systems: a practical approach. Geneva: WHO; 2025.
30. Winstein CJ, Stein J, Arena R, et al. Guidelines for Adult Stroke Rehabilitation and Recovery. Stroke. 2016;47:e98–e169.

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