

Stroke prevention

Contents at a glance

Significance and status	.23
Stroke prevention strategies	.23
Health promotion	.23
Primary prevention	.23
Secondary prevention	.24
Best practice guidelines for stroke prevention	.24
Stroke risk factors	.24
Risk factor reduction	.24
Lifestyle modification	.24
Physical activity	.24
Diet	.24
Smoking and substance abuse	.25
Theoretical models of behavioural change	.25
Developing behavioural change strategies	.25
Management of hypertension	.26
Antihyperlipidemic therapy	.27
Specific stroke preventive therapy	.27
Agents acting on the renin-angiotensin system	.27
ACE inhibitors	.27
Angiotensin II receptor blockers	.27
Anticoagulant therapy	.28
Antiplatelet therapy	.28
ASA	.28
Dipyridamole plus ASA	.29
Clopidogrel	.29
Surgical interventions for carotid artery stenosis	.29
Carotid endarterectomy	.29
Carotid artery stenting	.29
Stroke prevention clinics	.29
Management of TIAs	.30
References	.31
Selected readings: behaviour change	.32

Stroke prevention

Significance and status

Tremendous advances have been made in the treatment of stroke, but it is through improving stroke prevention that the most significant reductions in stroke morbidity and mortality will be achieved. Increasing stroke prevention efforts is of paramount importance, because of the high prevalence of risk factors in the Canadian population: 75% of Canadian adults have at least one stroke risk factor.

The identification of the major stroke risk factors allows an individual's risk of stroke to be estimated and risk reduction strategies to be developed. Detection and effective management of predisposing medical conditions, such as hypertension, and modification of behavioural risk factors, such as inactivity and smoking, could prevent many strokes. Stroke prevention clinics are now being established to assist individuals with uncontrolled risk factors and those who have experienced a stroke or TIA. These clinics can play an important role in diagnosis, assessment, and access to appropriate interventions.

Strategies are also needed to inform the general public about stroke risk factors, as many individuals are still unaware of their own risk of stroke and of ways to reduce risk.

Stroke prevention strategies

Stroke prevention encompasses risk factor reduction, both at a population and an individual level. Health promotion addresses stroke prevention at a population level, whereas primary and secondary prevention address an individual's risk of stroke. Implementing optimal stroke prevention, using consistent and effective preventive strategies, has the potential to significantly reduce the incidence of stroke in Ontario, perhaps by as much as 50%.

Towards An Integrated Stroke Strategy for Ontario recommends a significant investment in health promotion and in primary and secondary prevention. Ontario public health units will play an important role in implementing preventive measures.¹ Existing public

health initiatives that target important lifestyle-related stroke risk factors — smoking, obesity, hypertension, and physical inactivity — are being enhanced, and additional targeted initiatives are being developed.

Effective stroke prevention can occur at the various levels of health promotion, primary prevention, and secondary prevention. Although health promotion and primary prevention are briefly described here, this manual focuses on secondary stroke prevention.

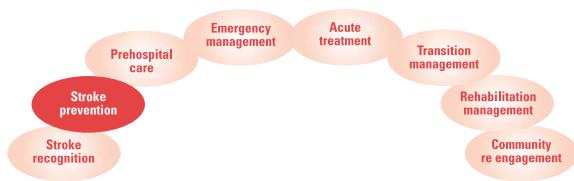
Health promotion

Health promotion is a population-based awareness and educational strategy designed to inform the public about stroke risk factors, to motivate individuals with modifiable risk factors to address them, and to convince those individuals without

behavioural risk factors how important it is to avoid them. Health promotion encourages members of the public to follow a healthy lifestyle and to take control of their health. Examples of health promotion programs that are relevant to stroke prevention include the Ontario Heart Health Program, the Ontario Tobacco Strategy, and the Ontario Physical Activity Strategy.

Primary prevention

Primary prevention is an individually based clinical approach to disease prevention in otherwise healthy individuals with modifiable risk factors. Primary prevention is usually implemented in the primary care setting, and the physician, advanced practice nurse, or the patient may initiate a discussion of stroke risk



Best practice guidelines for stroke prevention

2. Develop an optimal stroke prevention strategy in a timely manner for all individuals at high risk of stroke, all individuals with TIA, and all stroke survivors. The strategy should include the optimal use of stroke prevention services, a plan for lifestyle modification, pharmacotherapy, and a plan for effective communication between the client, primary care physician, and stroke prevention services.
3. Educate individuals at high risk of stroke, those who have experienced a TIA or stroke, family members, and healthcare providers about stroke prevention, relevant information resources, and how to gain access to them.

reduction. Common topics for these discussions include the importance of managing risk factors using strategies, such as smoking cessation, an active lifestyle, a healthy low-fat diet, and the appropriateness of specific pharmacotherapeutic agents in reducing risks.

Secondary prevention

Secondary prevention is an individually based clinical approach to reducing the risk of recurrent events in individuals who have already experienced an event, and in those who are experiencing symptoms that place them at high risk of an event. Thus, individuals who have experienced a TIA or a stroke are candidates for secondary prevention. Secondary prevention strategies encompass pharmacotherapy, surgical interventions, and support in making lifestyle modifications.

Stroke risk factors

Several risk factors significantly increase the risk of stroke. Virtually all individuals who experience a stroke have at least one risk factor, and the prevalence of stroke risk factors in the Canadian population is high.

- Some 75% of all Canadian adults have at least one lifestyle-related risk factor.²

- Some 25% of men and 18% of women have hypertension, but 42% of hypertensive Canadians are unaware of their condition.²

It is important to remember that individuals who have experienced an ischemic event have a significantly increased risk of subsequent ischemic events, both in the same and in other vascular beds, as atherosclerotic disease is usually progressive *and* generalized.³

Virtually all individuals who experience a stroke have at least one risk factor, and the prevalence of stroke risk factors in the Canadian population is high.

Risk factor reduction

The foundation of stroke prevention for all individuals is a healthy lifestyle, including smoking cessation, maintaining a healthy body weight, and living a physically active life. Managing key stroke risk factors, including diabetes mellitus, hypertension, and hypercholesterolemia is essential, as is treating predisposing conditions, such as atherosclerosis, ischemic heart disease, and atrial fibrillation.²

Optimal management of stroke risk factors has been conclusively demonstrated to significantly reduce an individual's risk of a first stroke, and it is critical to reduce the risk of recurrent stroke and other ischemic vascular events.⁴

Lifestyle modification

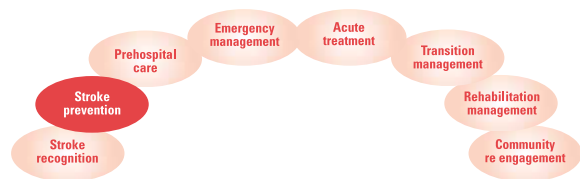
Level 2 evidence supports lifestyle modification, including weight loss, regular exercise, and low alcohol consumption to manage hypertension.⁵ In addition, level 2 evidence supports smoking cessation to reduce stroke risk.⁶ Strict glycemic control in individuals with diabetes has not yet been demonstrated to reduce stroke risk (level 3 evidence), but it does significantly reduce the risk of diabetic complications.⁶

Physical activity

Strong evidence supports the benefit of physical activity in reducing the risk of coronary heart disease, hypertension, diabetes mellitus, and obesity. As these conditions are also risk factors for stroke, a strong rationale exists for encouraging an active lifestyle to reduce the risk of stroke. Evidence too is now beginning to link physical activity to reduction in stroke risk. Analysis of data from the Nurses' Health Study indicates that even moderate-intensity exercise, such as walking, substantially reduces the risk of total and ischemic stroke, and that greater exercise intensity is associated with greater risk reduction.⁷

Diet

The importance of dietary modification has been vastly underestimated as a preventive strategy to reduce the risk of stroke. In addition, clinicians tend to focus more on lipid values than on diet, because effective pharmacological agents to treat hyperlipidemia are now available and also because people find it difficult to change longstanding



Stroke risk factors⁶

Modifiable risk factors

- Behavioural
 - physical inactivity
 - smoking
- Predisposing conditions
 - stroke
 - TIA
 - obesity
 - acute myocardial infarction (MI)
 - hypertension
 - hyperlipidemia
 - atrial fibrillation
 - diabetes mellitus
 - atherosclerosis
 - coronary heart disease
 - asymptomatic carotid stenosis
 - peripheral vascular disease
 - other cardiac disease
 - coagulation disorders
 - estrogen/progestin replacement therapy.⁸

Unmodifiable risk factors

- increasing age
- genetic factors
- male sex
- race.

Probable risk factors

- migraine
- oral contraceptive use
- alcohol abuse
- stress
- sleep apnea
- sympathomimetic agents
- illicit drug use
- congenital cardiac anomalies
- other.

dietary habits. Studies have demonstrated that postprandial lipemia, which is determined by diet, is more predictive of atherosclerotic risk than fasting lipemia,^{9,10} but the complex mixture of oxidized fats, trans fatty acids, and other compounds, including free radicals, circulating in the bloodstream after a high-fat meal, confuses the evaluation of postprandial lipids.

Recent studies have clearly demonstrated both the importance of diet as a risk factor for stroke and the benefits of dietary modification (level 1 evidence). The Lyon Diet Heart Study evaluated a Cretan-style Mediterranean diet low in harmful substances, such as cholesterol and fat, and high in beneficial substances, such as lycopene, alpha-linolenic acid, phytoestrogens, and antioxidants.¹¹ This diet was similar to a National Cholesterol Education Program (NCEP) Step II low-fat diet (20% of calories from fat), but contained more alpha-linolenic acid, more fruit and vegetables, and less red meat. The Lyon Diet Heart Study found that this Mediterranean diet reduced the incidence of MI and death by 60% over 4 years, compared with a typical North American diet.

Hypertension, an important stroke risk factor, can also be influenced by diet. A diet high in fruit and vegetables and low in meat, fish, and poultry was evaluated in the Dietary Approaches to Stop Hypertension (DASH) trial.¹² This diet substantially reduced blood pressure in hypertensive subjects, reducing systolic pressure by 11.4 mmHg and diastolic pressure by 5.5 mmHg. Restricting sodium in conjunction with the DASH diet further reduced blood pressure.¹³

Smoking and substance abuse

Smoking cessation and management of substance abuse are central to effective reduction of stroke risk. An extensive literature reviewing these topics exists, but discussion of smoking cessation and treatment of substance abuse is outside the scope of this manual.

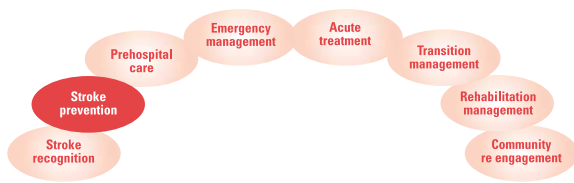
Theoretical models of behavioural change

Several theoretical models of behavioural change have been developed, including Social Learning Theory, Theory of Reasoned Action, Health Belief Model, and Stages of Change, the best-known model. These theoretical models provide a preliminary framework for understanding the mechanisms underlying poor lifestyle choices and the factors limiting behavioural change in the stroke population. Behavioural change strategies based on these models have been studied in various populations, such as heart disease, diabetes, HIV, substance abuse, and eating disorders, but evidence to support their use is lacking in stroke survivors.

Developing behavioural change strategies

Many stroke risk factors are related to unhealthy lifestyle choices, and psychological factors, such as anxiety and depression, play a well-established role in substance abuse and in non-compliance with healthy eating. As a result, developing effective behavioural change strategies may play an important role in risk factor reduction in stroke survivors and others at high risk of a stroke.

Behavioural change strategies have been well studied in the treatment of behavioural disorders, such as eating disorders and alcohol or substance abuse, and in risk factor modification in coronary artery disease (CAD),



diabetes, and HIV. However, little evidence exists to guide the development of specific behavioural change strategies in stroke. Also, advanced age and specific comorbidities in stroke survivors, such as reduced mobility and cognitive deficits, may complicate the implementation and evaluation of behavioural change strategies in these populations.

Nevertheless, behavioural changes promoting risk factor modification may have a beneficial impact on the incidence of stroke and possibly on related conditions, such as heart disease and diabetes. Moreover, the effectiveness of efforts to promote healthy behaviours — such as active lifestyles, healthy eating, smoking cessation, moderating alcohol intake, and managing stress — among individuals at risk of stroke may be increased by incorporating appropriate behaviour modification strategies.

On the basis of other populations and the lack of data to guide the development of behaviour modification strategies to change unhealthy behaviours in stroke populations, the following guiding principles are advocated:

- Clinical skills in health education and in psychology and psychiatry, including psychotherapeutic skills, are central to the design and implementation of effective programs. It is therefore crucial to include a psychologist as a member of, or consultant to, the clinical team designing and implementing stroke prevention strategies that target behavioural change. It is also important to maintain a commitment to the continuing education of clinicians working in this area.
- An understanding of the mechanisms mediating maladaptive healthcare choices, the factors hampering behavioural change, and the literature about behavioural change in various

populations is critical to developing effective strategies. Interventions should be theoretically driven, but there is insufficient supporting evidence for any specific theory to guide the development of behavioural interventions in stroke prevention.

- Interventions required for behavioural change may differ among subgroups within the stroke prevention population. While some patients may require education, others may also require additional interventions, such as psychotherapeutic strategies. Although knowledge, motivation, and readiness for change are necessary, they are generally insufficient to create enduring behavioural change. It is therefore important to tailor the behavioural change strategy to the individual.
- Studies of other clinical populations have demonstrated that neurocognitive difficulties, stress, health-related attitudes, readiness for change, and psychopathology can all influence maladaptive choices and the efficacy of any interventions. Also, studies have demonstrated that several programs, such as PACE (Program of All-inclusive Care for the Elderly) and CHAMPS (Controlled High-risk subjects Avonex MS Prevention Study), facilitate behavioural changes relating to activity levels and healthy eating. Therefore, it is important to use patient assessment information, theoretical frameworks, and the clinical literature on behavioural change to directly guide treatment.
- Factors that promote the initiation of change may not be the same across target behaviours, such as healthy eating, smoking cessation, and increasing activity. They may also differ from factors involved

in maintaining behavioural change across different target behaviours.

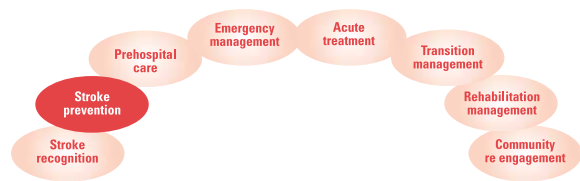
- It is important that programs provide a prescription for changing behaviour, use goal setting and employ clinical strategies to help the individual move through behavioural change, maintenance, and relapse avoidance. Studies in several populations have demonstrated the efficacy of cognitive-behavioural psychotherapeutic strategies.
- Finally, it is important to measure changes in behaviour, not simply self-reports of behavioural change, as self-reported behavioural change may not correlate with actual behavioural change.

A list of selected readings on behavioural change is provided on page 38.

Management of hypertension

Strong, level 1 evidence supports pharmacological management of hypertension.⁶ Hypertension triples or quadruples stroke risk, and it is also a risk factor for MI. The Systolic Hypertension in the Elderly Program (SHEP) indicated that effective management of hypertension can reduce the incidence of stroke by 36%.¹⁴

Many hypertensive individuals are either unaware of their condition, inadequately treated, or not treated at all. In fact, only 16% of Canadian hypertensives are adequately controlled.¹⁵ Patient factors contributing to this poor performance include poor awareness of hypertension as a stroke risk factor, poor knowledge about how to get blood pressure measured, and uncertainty about how to react if hypertension is confirmed. In addition, the shortage of family physicians makes it problematic for some patients



to gain access to health care. This shortage may also lead physicians to adopt a therapeutic rather than a preventive approach. Finally, poor compliance with prescribed medications undermines efforts to manage blood pressure, possibly because reinforcement of the need for long-term therapy is lacking. Improving the management of hypertension has the potential to reduce considerably the incidence of both stroke and MI. To this end, guidelines have been developed to assist physicians to manage this risk factor optimally, but they have not yet been consistently adopted.¹⁵

Improving the awareness and management of hypertension is an important part of the overall stroke strategy in Ontario. Therefore, the Heart and Stroke Foundation of Ontario, in partnership with the Ontario College of Family Physicians, supported by a grant from the Ontario Ministry of Health and Long-Term Care, has developed a public education strategy on hypertension, the Blood Pressure Action Plan™.

This program, which was developed using an evidence-based medical model, includes a personalized assessment of hypertension risk and health action plans to help people identify and reduce their risks. Health action plans assist in prioritizing lifestyle-modification goals, and they offer realistic strategies for healthier living, while supporting compliance with medical management.

Antihyperlipidemic therapy

Management of dyslipidemia is an important aspect of stroke prevention, and recommendations for target cholesterol values, based on calculating individual risk levels, are available.¹⁶ Studies that evaluate pharmacological management of hyperlipidemia, using the statin class of antihyperlipidemic

agents in individuals with CAD, have generally shown a reduction in stroke risk (level 1 evidence).⁶ Pravastatin 40 mg/day provides a relative risk reduction (RRR) of 32% compared to placebo, even in individuals with serum cholesterol in the normal range.⁶ Simvastatin is effective for the secondary prevention of stroke in individuals with coronary heart disease and elevated cholesterol, and the recent Heart Protection Study (HPS) found that simvastatin can protect and benefit a far wider range of people at risk of heart attacks and strokes than had previously been thought.¹⁷ Studies that evaluate the effectiveness of statins in preventing recurrent stroke in individuals without clinical evidence of CAD are nearing completion.

Specific stroke preventive therapy

Evidence also supports the use of specific stroke preventive therapy, including agents acting on the renin-angiotensin system, and antithrombotic and anticoagulant therapy in individuals at risk of stroke. For example, anticoagulation effectively reduces the risk of stroke in individuals with atrial fibrillation and previous MI, and antiplatelet therapy reduces the risk of stroke in high-risk individuals. Surgical interventions, such as carotid endarterectomy, are beneficial in some symptomatic patients.

Agents acting on the renin-angiotensin system

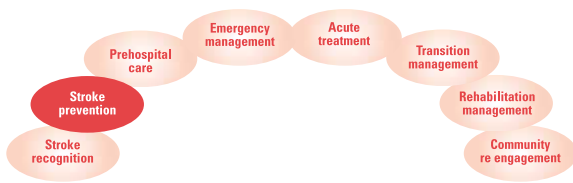
Angiotensin-converting enzyme (ACE) inhibitors. ACE inhibitors are frequently used to manage hypertension. They also protect the vascular endothelium from damage, a critical first step in the development of atherosclerosis. The enhanced risk factor reduction seen with ACE inhibitors may be related to endothelial protection.

The Heart Outcomes Prevention Evaluation (HOPE) study estimated cardiovascular risk reduction achieved by adding ramipril 10 mg/day to standard medical therapy, including an antiplatelet agent.¹⁸ The addition of ramipril resulted in RRR of 32% in stroke, death, or MI, compared to placebo (level 1 evidence). Only 40% of ramipril's efficacy could be attributed to its antihypertensive effects, and the remainder of its effect on risk might be related to endothelial protection.

A large randomized trial, the perindopril protection against recurrent stroke study (PROGRESS), evaluated the impact of 4 years of perindopril-based therapy on cardiovascular risk in more than 6,000 hypertensive and nonhypertensive patients with a history of stroke or TIA, compared with placebo. PROGRESS found that the combination of perindopril plus indapamide, a diuretic, significantly reduced stroke risk by 43%.¹⁹ The findings suggest that combination therapy is beneficial in patients with a history of stroke or TIA, irrespective of initial blood pressure. In the HOPE and PROGRESS trials, nonhypertensive individuals also benefited from therapy with an ACE inhibitor.

Angiotensin II receptor blockers.

Residual elevations of stroke risk in treated hypertensive patients may be related to incomplete control of hypertension, target organ damage, such as left ventricular hypertrophy, or both. However, angiotensin II receptor blockade appears to be associated with reversal of left ventricular hypertrophy. The Losartan Intervention For Endpoint reduction (LIFE) study, was conducted to determine if managing hypertension with an angiotensin II receptor blocker, such as losartan, rather than with a beta-blocker, would reduce cardiovascular morbidity and mortality



Pharmacotherapy

All patients at risk of stroke should receive appropriate pharmacotherapy, including medications to manage any risk factors that are present, and specific stroke preventive therapy.

Table 1. Pharmacotherapy in stroke prevention.

Risk factor reduction

Antihypertensive agents	various classes
Antihyperlipidemic agents	statins and other classes

Specific stroke preventive therapy

Angiotensin II receptor blockers	losartan
ACE inhibitors	ramipril, perindopril/indapamide
Anticoagulant therapy	warfarin
Antiplatelet therapy	ASA, clopidogrel, dipyridamole plus ASA

beyond that associated with control of hypertension.²⁰

The LIFE trial, which randomized more than 9,000 patients to either losartan or atenolol, found that both agents produced similar blood pressure reductions, but that significantly fewer fatal or nonfatal strokes occurred in the losartan group ($p=0.001$).

Anticoagulant therapy

Level 1 evidence supports anticoagulation in individuals with atrial fibrillation or previous MI.⁶ For patients with atrial fibrillation, the American College of Chest Physicians (ACCP) makes the following recommendations concerning anticoagulant therapy²¹:

- All high-risk individuals, and all individuals older than 75 years of age, regardless of risk level, should be treated with warfarin.
- Low-risk individuals and individuals younger than 65 years of age should be treated with ASA.
- Individuals 65–75 years of age without risk factors may receive warfarin or ASA, based on the clinical situation and the physician's judgment.

For individuals with atrial fibrillation, warfarin should be administered to reach a target international normalized ratio (INR) of 2–3. A target INR of 2.5–3.5 appears beneficial in patients with a vascular event and in lupus anticoagulant syndrome. For secondary prevention, warfarin therapy provides RRR of 70% compared with placebo: the annual incidence of stroke with warfarin, ASA, or placebo is 4%, 10%, and 12% respectively.²²

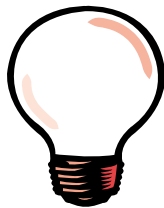
Antiplatelet therapy

Strong level 1 evidence supports the use of antiplatelet therapy (ASA, sustained-release dipyridamole plus ASA, or clopidogrel) in high-risk individuals.^{23,24} Ticlopidine, an inhibitor

of platelet aggregation that is related to clopidogrel, is associated with significant side effects, including neutropenia and thrombocytopenic purpura, and guidelines now recommend that individuals not be started on this agent. These idiosyncratic adverse events occur early in therapy, so individuals already on ticlopidine may safely be continued on this antiplatelet agent.

ASA. The worldwide Antiplatelet Trialists' Collaboration reviewed 145 randomized trials of antiplatelet therapy.²³ Trials comparing antiplatelet therapy with control included 30,000 low-risk patients and 70,000 high-risk patients, and trials comparing different antiplatelet agents included 10,000 high-risk patients. Antiplatelet therapy significantly reduced the risk of ischemic events in all high-risk secondary prevention categories by approximately 25%. The reduction was statistically significant irrespective of age, sex, blood pressure, or history of diabetes. Specifically, antiplatelet therapy reduced nonfatal MI by $1/3$, nonfatal stroke by $1/3$, and vascular death by $1/6$. The most widely tested regimen was medium-dose ASA 75–325 mg/day. Increased benefit was noted with increased duration of therapy.

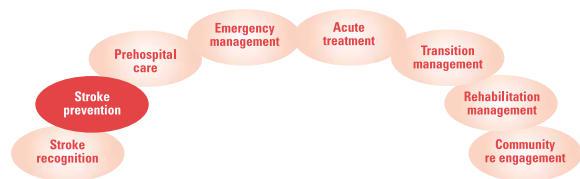
The recent Antithrombotic Trialists' Collaboration meta-analysis of randomized trials of preventive antiplatelet



Practical tip

Action plans are available online at the Heart and Stroke Foundation Web site <www.heartandstroke.ca/bloodpressure> or by calling the Foundation's toll-free health information line at 1-888-HSF-INFO (473-4636).

You can refer your patients to these resources or they can access the information directly. This information is provided at no cost and contacts are completely confidential.



therapy in high-risk patients reviewed 287 studies.²⁴ More than 77,000 patients were included in trials comparing antiplatelet regimens, and 135,000 patients were included in trials comparing active therapy with control. Antiplatelet therapy reduced nonfatal MI by $1/3$, nonfatal stroke by $1/4$, and vascular death by $1/6$.

ASA, the most studied agent, was found to be as effective at doses of 75–150 mg/day as at higher doses.²⁴ There is no evidence that increasing the ASA dose increases efficacy. The usual dose of ASA is 325 mg/day, but doses as low as 40 mg/day may be effective.

Dipyridamole plus ASA. One randomized trial of 6,600 individuals has shown sustained-release dipyridamole plus ASA to reduce the risk of stroke by 37% and the risk of stroke or death by 24% compared to placebo.²⁵ The combination of low dose ASA 25 mg and sustained-release dipyridamole 200 mg given twice daily reduced the relative risk of stroke by 19% over ASA. Sustained-release dipyridamole plus ASA is usually given BID.

Clopidogrel. Clopidogrel is an inhibitor of platelet aggregation. The Clopidogrel vs Aspirin in Patients at Risk of Ischemic Events (CAPRIE) study, a large randomized, controlled trial, found that clopidogrel reduces the incidence of stroke, MI, and death by 9%, compared with ASA in patients with a history of ischemic vascular disease.²⁶

The Clopidogrel in Unstable angina to prevent Recurrent Events (CURE) study compared the combination of clopidogrel and ASA with ASA in patients with unstable angina and non-Q-wave MI.²⁷ The CURE trial demonstrated RRR with clopidogrel plus ASA compared with placebo plus ASA of 20%, for the primary efficacy

outcome of cardiovascular death, nonfatal MI, or nonfatal stroke ($p < 0.001$), and 14% for the primary efficacy outcome of cardiovascular death, nonfatal MI, nonfatal stroke, or refractory ischemia ($p < 0.001$). The dose of clopidogrel is 75 mg/day.

The Management of Atherothrombosis with Clopidogrel in High-Risk Patients with Recent Transient Ischemic Attack or Ischemic Stroke (MATCH) study, which is currently underway, is being conducted to determine whether clopidogrel plus ASA is more effective than clopidogrel alone in preventing new ischemic events in patients after recent TIA or ischemic stroke and at high risk of recurrent ischemic events. It will also evaluate the safety of long-term clopidogrel plus ASA compared to clopidogrel alone.

Surgical interventions for carotid artery stenosis

Surgical interventions to treat symptomatic carotid artery stenosis may be beneficial in specific patient types, significantly reducing the risk of recurrent stroke.

Carotid endarterectomy. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) evaluated the benefit of carotid endarterectomy in individuals with symptomatic low-moderate (<50%), moderate (50–69%), or severe (70–99%) carotid stenosis.²⁸ The study found that individuals with less than 50% stenosis did not benefit from endarterectomy and that those with stenosis of 50–69% received a modest but statistically significant benefit. However, participants with 70–99% stenosis derived a substantial and durable benefit from the procedure, measured at the 8-year follow-up (level 1 evidence).²⁸ Carotid endarterectomy is a high-risk

procedure that is best carried out by surgeons with expertise in this procedure. The use of endarterectomy for asymptomatic carotid stenosis is still controversial.^{6,28} The Canadian consensus opinion does not recommend carotid endarterectomy for asymptomatic carotid stenosis.²⁹ In appropriate candidates, carotid endarterectomy should be performed within 1 month of the occurrence of TIA.

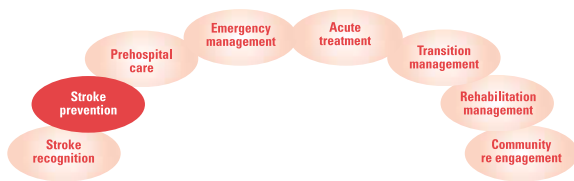
Carotid artery stenting. The benefits of coronary artery stenting in patients with symptomatic ischemic CAD prompted investigators to evaluate the potential of stenting in patients with symptomatic carotid artery disease. The incidence of procedure-related complications in trials conducted to date is high.^{30,33} Nevertheless, using new stents, antiplatelet agents and neuroprotective devices may significantly improve the safety and efficacy of carotid stenting procedures.

Several clinical trials are currently evaluating the role of carotid stenting, including the Stenting and Angioplasty with Protection in Patients and High Risk for Endarterectomy (SAPPHIRE) study and the Carotid Revascularization: Endarterectomy versus Stent Trial (CREST).³⁴

Stroke prevention clinics

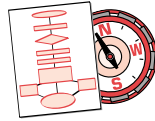
Regional stroke centres are establishing hospital-based stroke prevention clinics to provide an individualized, comprehensive, interdisciplinary approach to stroke prevention for high-risk individuals and stroke survivors. The Joint Stroke Strategy Working Group defined the role of stroke prevention clinics as follows:

Working closely with the acute care sector, primary care, and rehabilitation, regional stroke prevention clinics will be responsible for regional organization of



secondary stroke prevention services. Stroke prevention clinics will reduce delays and inefficiencies in risk factor management of high-risk patients, as well as facilitate access to carotid endarterectomy. Whenever possible, such clinics should be linked and share resources with cardiac prevention services and/or community-based prevention clinics.

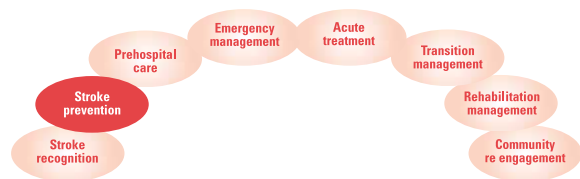
Additional information about stroke prevention clinics is contained in *A Guide to Establishing Stroke Prevention Clinics*, a 2001 Heart and Stroke Foundation of Ontario manual in the Coordinated Stroke Strategy series. This manual is available online at www.heartandstroke.ca/profed/.



Management of TIAs

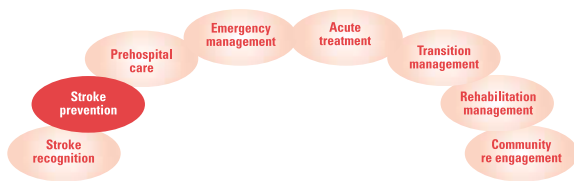
TIAs are associated with a high short-term risk of stroke.³⁵ Therefore, all individuals with TIA presenting to a hospital emergency department or a family physician's office should be referred to stroke prevention services, if possible, or to a neurologist or physician trained in vascular disease prevention for urgent investigation and development of a care plan. At that time, the emergency or primary care physician should initiate a TIA protocol to facilitate appropriate urgent assessment, medical therapy and possibly surgical intervention. It is critical that wait times for diagnosis, treatment, and risk factor management be reduced, so that patients are evaluated within 1 week of the TIA.

In appropriate candidates, carotid endarterectomy should be performed within 1 month of the occurrence of TIA.

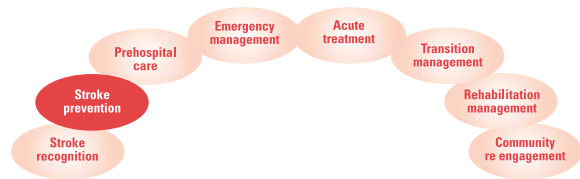


References

1. Ministry of Health and Long-Term Care and the Heart and Stroke Foundation of Ontario. *Towards An Integrated Stroke Strategy for Ontario*. Report of the Joint Stroke Strategy Working Group, June 2000.
2. Wilson E, Taylor G, Phillips S, et al. Creating a Canadian stroke system. *CMAJ* 2001;164:1853-1855.
3. Kannel WB. Risk factors for atherosclerotic cardiovascular outcomes in different arterial territories. *J Cardiovasc Risk* 1994;1:333-339.
4. Gorelick PB. Stroke prevention therapy beyond antithrombotics: unifying mechanisms in ischemic stroke pathogenesis and implications for therapy: an invited review. *Stroke* 2002;33:862-875.
5. Campbell NR, Burgess E, Choi BC, et al. Lifestyle modifications to prevent and control hypertension. 1. Methods and an overview of the Canadian recommendations. Canadian Hypertension Society, Canadian Coalition for High Blood Pressure Prevention and Control, Laboratory Centre for Disease Control at Health Canada, Heart and Stroke Foundation of Canada. *CMAJ* 1999;160(9 suppl):S1-6.
6. Ingall TJ. Preventing ischemic stroke: current approaches to primary and secondary prevention. *Postgrad Med* 2000;107:34-36, 39-42, 47-50.
7. Hu FB, Stampfer MJ, Colditz GA, et al. Physical activity and risk of stroke in women. *JAMA* 2000;283:2961-2967.
8. Writing group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA* 2002;288:321-333.
9. Kirchmair R, Ebenbichler CF, Patsch JR. Post-prandial lipaemia. *Baillieres Clin Endocrinol Metab* 1995;9:705-719.
10. Ebenbichler CF, Kirchmair R, Egger C, Patsch JR. Postprandial state and atherosclerosis. *Curr Opin Lipidol* 1995;6:286-290.
11. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation* 1999;99:779-785.
12. Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med* 1997;336:1117-1124.
13. Sacks FM, Svetkey LP, Vollmer WM, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med* 2001;344:3-10.
14. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). SHEP Cooperative Research Group. *JAMA* 1991;265:3255-3264.
15. Feldman RD, Campbell N, Larochelle P, et al. 1999 Canadian recommendations for the management of hypertension. Task Force for the Development of the 1999 Canadian Recommendations for the Management of Hypertension. *CMAJ* 1999;161(12 suppl):S1-17.
16. Fodor JG, Frohlich JJ, Genest JJ Jr, McPherson PR. Recommendations for the management and treatment of dyslipidemia. Report of the Working Group on Hypercholesterolemia and Other Dyslipidemias. *CMAJ* 2000;162:1441-1447.
17. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised, placebo-controlled trial. *Lancet* 2002;360:7-22.
18. Bosch J, Yusuf S, Pogue J, et al. Use of ramipril in preventing stroke: double blind randomised trial. *BMJ* 2002;324:699-702.
19. Randomised trial of perindopril-based blood-pressure lowering regimen among 6,105 individuals with previous stroke or transient ischaemic attack. *Lancet* 2001;358:1033-1041.
20. Dahlöf B, Devereux RB, Kjeldsen SE, et al. Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): a randomised trial against atenolol. *Lancet* 2002;359:995-1003.
21. Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P. Antithrombotic and thrombolytic therapy for ischemic stroke. *Chest* 2001;119 (1 suppl):300S-320S.
22. Albers GW, Dalen JE, Laupacis A, Manning WJ, Petersen P, Singer DE. Antithrombotic therapy in atrial fibrillation. *Chest* 2001;119:194S-206S.
23. Collaborative overview of randomised trials of antiplatelet therapy — I: Prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of patients. Antiplatelet Trialists' Collaboration. *BMJ* 1994;308:81-106.
24. Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high-risk patients. *BMJ* 2002;324:71-86.



25. Diener HC, Cunha L, Forbes C, Sivenius J, Smets P, Lowenthal A. European Stroke Prevention Study. 2. Dipyridamole and acetylsalicylic acid in the secondary prevention of stroke. *J Neurol Sci* 1996;**143**:1-13.
26. A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). CAPRIE Steering Committee. *Lancet* 1996;**348**:1329-1339.
27. Yusuf S, Zhao F, Mehta SR, Chrolavicius S, Tognoni G, Fox KK. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation. *N Engl J Med* 2001;**345**:494-502.
28. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. *N Engl J Med* 1998;**339**:1415-1425.
29. Perry JR, Szalai JP, Norris JW, for the Canadian Stroke Consortium. Consensus against both endarterectomy and routine screening for asymptomatic carotid stenosis. *Arch Neurol* 1997;**54**:25-28.
30. Spence D, Eliasziw M. Endarterectomy or angioplasty for treatment of carotid stenosis. *Lancet* 2001;**357**:1722-1723.
31. Chaturvedi S, Fessler R. Angioplasty and stenting for stroke prevention: good questions that need answers. *Neurology* 2002;**59**:664-668.
32. Theron JG, Payelle GG, Coskun O, Huet HF, Guimaraens L. Carotid artery stenosis: treatment with protected balloon angioplasty and stent placement. *Radiology* 1996;**201**:627-636.
33. Ohki T, Roubin GS, Veith FJ, Iyer SS, Brady E. Efficacy of a filter device in the prevention of embolic events during carotid angioplasty and stenting: an ex vivo analysis. *J Vasc Surg* 1999;**30**:1034-1044.
34. Bhatt DL, Kapadia SR, Bajzer CT, et al. Dual antiplatelet therapy with clopidogrel and aspirin after carotid artery stenting. *J Invasive Cardiol* 2001;**13**:767-771.
35. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. *JAMA* 2000;**284**:2901-2906.
- General**
- Pearson TA, Blair SN, Daniels SR, et al. AHA Guidelines for Primary Prevention of Cardiovascular Disease and Stroke: 2002 Update: Consensus Panel Guide to Comprehensive Risk Reduction for Adult Patients Without Coronary or Other Atherosclerotic Vascular Diseases. American Heart Association Science Advisory Committee. *Circulation* 2002;**106**:388-391.
- Selected readings: behaviour change**
- Ajzen I, Fishbein M. *Understanding Attitudes and Predicting Social Behavior*. Englewood Cliffs, NJ: Prentice Hall, 1997.
- Bandura A. Self-efficacy: toward a unifying theory of behavioral change. *Psychol Rev* 1977;**84**:191-215.
- Becker MH. The health belief model and personal health behavior. *Health Education Monographs* 1974;**2**:324-473.
- Bock BC, Marcus BH, Pinto BM, Forsyth LH. Maintenance of physical activity following an individualized motivationally tailored intervention. *Ann Behav Med* 2001;**23**:79-87.
- Burbank PM, Padula CA, Nigg CR. Changing health behaviors of older adults. *J Gerontol Nurs* 2000;**26**:26-33; quiz 52-53.
- Burton LC, Shapiro S, German PS. Determinants of physical activity initiation and maintenance among community-dwelling older persons. *Prev Med* 1999;**29**:422-430.
- Calfas KJ, Sallis JE, Zabinski MF, et al. Preliminary evaluation of a multi-component program for nutrition and physical activity change in primary care: PACE+ for adults. *Prev Med* 2002;**34**:153-161.
- Calfas, KJ, Sallis, JE, Oldenburg B, Ffrench M. Mediators of change in physical activity following an intervention in primary care: PACE. *Prev Med* 1997; **6**:297-304.
- Cameron R, Meichenbaum D. Cognition and behaviour change. Aust NZ *J Psychiatry* 1980;**14**:121-125.
- Clark M, Hampson SE. Implementing a psychological intervention to improve lifestyle self-management in patients with type 2 diabetes. *Patient Educ Couns* 2001;**42**:247-256.
- Conn VS. Older women's beliefs about physical activity. *Public Health Nurs* 1998;**15**:370-378.
- Deforche B, De Bourdeaudhuij I. Differences in psychosocial determinants of physical activity in older adults participating in organised versus non-organised activities. *J Sports Med Phys Fitness* 2000;**40**:362-372.
- Krummel DA, Koffman DM, Bronner Y, et al. Cardiovascular health interventions in women: What works? *J Womens Health Gen Based Med* 2001;**10**:117-136.
- Marcus BH, Banspach SW, Lefebvre RC, Rossi JS, Carleton RA, Abrams DB. Using the stages of change model to increase the adoption of physical activity among community participants. *Am J Health Promot* 1992;**6**:424-429.
- Nolan RP. How can we help patients to initiate change? *Can J Cardiol* 1995;**11** (suppl A):16A-19A.



Prochaska JO, DiClemente CC. Stages of change in the modification of problem behaviors. *Prog Behav Modif* 1992;**28**:183-218.

Ronda G, Van Assema P, Brug J. Stages of change, psychological factors and awareness of physical activity levels in The Netherlands. *Health Promot Internation* 2001;**16**:305-314.

Scharff DP, Homan S, Kreuter M, Brennan L. Factors associated with physical activity in women across the life span: implications for program development. *Women Health* 1999;**29**:115-134.

Sherwood NE, Jeffery RW. The behavioral determinants of exercise: implications for physical activity interventions. *Annu Rev Nutr* 2000;**20**:21-44.

Stevens W, Hillsdon M, Thorogood M, McArdle D. Cost-effectiveness of a primary care based physical activity intervention in 45-74 year old men and women: a randomised controlled trial. *Br J Sports Med* 1998;**32**:236-241.

Stewart AL, Verboncoeur CJ, McLellan BY, et al. Physical activity outcomes of CHAMPS II: a physical activity promotion program for older adults. *J Gerontol A Biol Sci Med Sci* 2001;**56**:M465-470.

van der Bij AK, Laurant MG, Wensing M. Effectiveness of physical activity interventions for older adults: a review. *Am J Prev Med* 2002;**22**:120-133.