



Daffodil
International
University

Project on
A Review on Post-Stroke Management:
Non-pharmacological and Pharmacological Aspects

[In the partial fulfillment of the requirements for the degree of Bachelor of
Pharmacy]

Submitted To
Department of Pharmacy
Faculty of Allied Health Sciences
Daffodil International University

Submitted By
ID: 191-29-176
Batch: 21 DSC-B
Department of Pharmacy
Faculty of Allied Health Sciences
Daffodil International University

April 2023

APPROVAL

This project paper, A Review on Post-Stroke Management: Non-pharmacological and Pharmacological Aspects, submitted to the Department of Pharmacy, Faculty of Allied Health Sciences, Daffodil International University, has been accepted as satisfactory for the partial fulfillment of the requirements for the degree of Bachelor of Pharmacy and approved as to its style and contents.

BOARD OF EXAMINERS

.....

Dr. Muniruddin Ahmed
Professor and Head,
Department of Pharmacy,
Faculty of Allied Health Sciences,
Daffodil International University

.....

Internal Examiner 1

.....

Internal Examiner 2

.....

External Examiner 3

DECLARATION

I hereby declare that this project report, “A review on Post Stroke management: Non-pharmacological and Pharmacological Aspects”, is done by me under the supervision of Md. A.K. Azad, Assistant Professor & Coordinator M.Pharm, Department of Pharmacy, Faculty of Allied Health Sciences, Daffodil International University. I am declaring that this Project is my original work. I also declare that neither this project nor any part thereof has been submitted elsewhere for the award of a Bachelor’s or any degree.

Supervised By



.....

Md. A.K. Azad

Assistant Professor & Coordinator M.Pharm

Department of Pharmacy

Faculty of Allied Health Sciences

Daffodil International University

Submitted By



.....

Sumitro Das

ID: 191-29-176

Department of Pharmacy

Faculty of Allied Health Sciences

Daffodil International University

ACKNOWLEDGEMENT

I might want to communicate my profound applause to the All-powerful Creator who has given me the capacity to finish my undertaking work and the chance to concentrate on this subject.

I'm grateful to my honorable project supervisor Md. A. K. Azad, Assistant Professor, Department of Pharmacy, Daffodil International University for his brilliant direction and steady oversight just as for giving essential data regarding the task and for his help in finishing the project.

I would like to express my humble regards to Dr. Muniruddin Ahmed, Professor and Head of, Department of Pharmacy, Daffodil International University.

I also wish to offer my respect to all of the teachers of Pharmacy Department, Daffodil International University, and thankful to other members for their excellent cooperation with us. Finally, I would like to express my gratitude towards my parents and other family members for their kind cooperation and encouragement which helped me in the completion of this project.

Dedication

My Parents

The persons who always encourage me in every sphere of my life.

Abstract

Post-stroke management is a crucial component of stroke care, as it can help improve outcomes, prevent complications, and promote recovery. Both non-pharmacological and pharmacological approaches are used in post-stroke management. This study was carried out to increase the awareness of the people and to gather versatile information about post-stroke management. The work was done based on different articles and journals.

Non-pharmacological management includes lifestyle modifications, such as exercise programs, dietary changes, and mental support. Studies have shown that non-pharmacological post-stroke management is commonly used in many countries. It is difficult to provide an exact percentage of non-pharmacological management used by people worldwide after stroke as this can vary depending on factors such as availability of resources, healthcare systems, cultural beliefs and practices, and individual preferences. For example, a study published in the International Journal of Stroke in 2015 reported that rehabilitation therapy was used in more than 80% of stroke patients in hospitals in India. Another study published in the Journal of Stroke and Cerebrovascular Diseases in 2018 reported that non-pharmacological management was used in the majority of stroke patients in a hospital setting in Saudi Arabia.

Similarly, the use of pharmacological management after stroke is a common practice worldwide and is often used in combination with non-pharmacological interventions. However, the specific percentage of pharmacological management used after stroke is difficult to estimate as it can vary widely depending on individual patient characteristics and healthcare practices in different countries.

The extensive knowledge regarding post-stroke management that has been summarized in this review article may be useful to readers all over the world.

Contents

Chapter one	1
Introduction	1
1. Introduction	2
1.1 Types of Stroke	3
1.2 Pathophysiology of Stroke	5
1.3 Supervision of Stroke	8
Chapter two	10
Purpose of the Study	10
2.1 Purpose of the Study	11
Chapter three	12
Methodology	12
3.1 Methodology	13
Chapter four	14
Results & Discussion	14
4.1 Non-pharmacological Management	15
4.1.1 Lifestyle Modification	16
4.1.1.1 Physical Activities	16
4.1.1.2 Habituation	18
4.1.2 Dietary Habit	19
4.1.3 Mental Health	24
4.2 Pharmacological Management	26
4.2.1 Tissue Plasminogen Activators (TPA)	26

4.2.2 Blood Thinners.....	27
4.2.2.1 Anticoagulants.....	27
4.2.2.2 Antiplatelet Agents.....	28
4.2.3 Blood Pressure Lowering Medication.....	30
4.2.4 Cholesterol Lowering Agents.....	32
4.2.5 Routine Follow-up.....	33
Chapter five	34
Conclusion.....	34
5.1 Conclusion.....	35
Chapter six	36
References	37

Chapter one

Introduction

1. Introduction

The prevalence of stroke is increasing in developing countries, where it is the second greatest cause of death and disability. [1] The World Health Organization claims that the "incoming epidemic of the 21st century" is stroke. Recent studies indicate that 85% of all strokes may be avoided, therefore prevention strategies are now taking center stage in stroke care. [2] Stroke incidence rates overall in low- and middle-income countries were 20% higher than those in high-income countries between 2000 and 2008, marking the first time this has happened. It is no longer necessary to debate whether or not stroke should be a concern for governments in low- to middle-income nations. The time to act is right now. Time lost is brain lost. Every minute counts. [3]

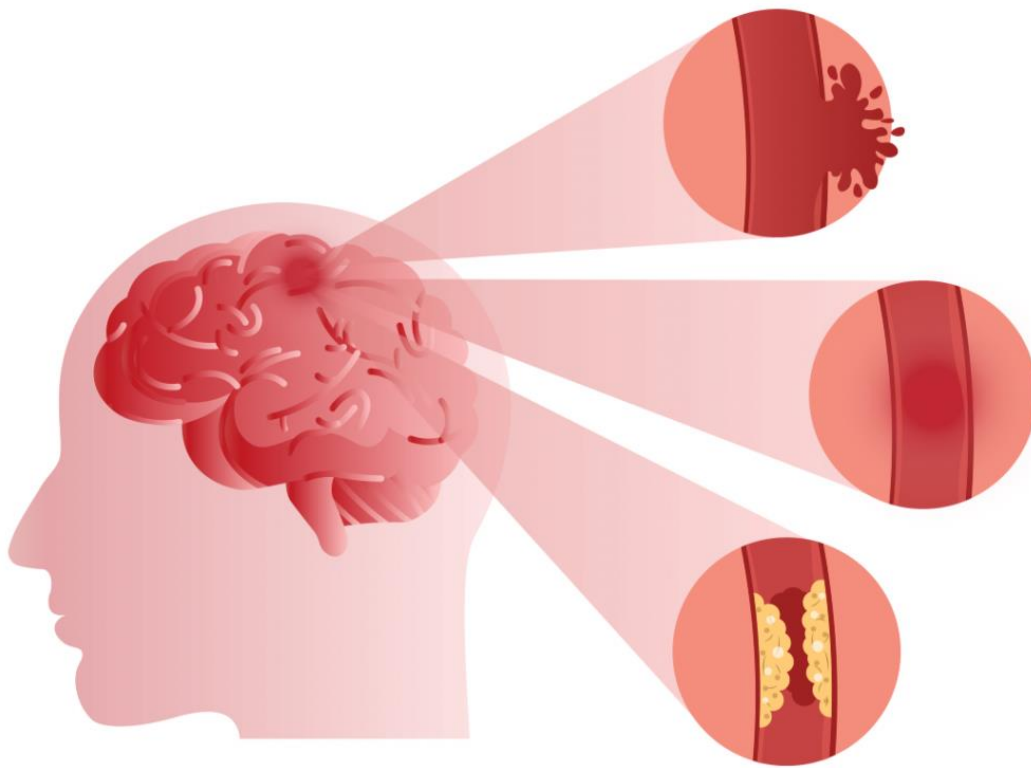


Fig.1: Stroke [136]

1.1 Types of Stroke

There are two major forms of stroke: ischemic and hemorrhagic. When a brain vessel is blocked by an obstruction that blocks 80% or more of the vessel, an ischemic stroke happens. On the other side, a vascular rupture results in a hemorrhagic stroke. [4] A transient ischemic attack (TIA), is also known as a "mini-stroke". Because the blockage of blood flow to the brain occurs for a much shorter period typically no longer than five minutes than with other major forms of stroke, it is distinct from them. [5]

Ischemic strokes are the most common type. When blood clots or other objects obstruct the brain's blood arteries, an ischemic stroke ensues. Blockages may also be caused by the buildup of plaque, or fatty deposits, in blood arteries. [6] An ischemic stroke has three main causes: thrombosis, hypoperfusion, and embolism. Most frequently, thrombosis is thought to be the culprit. The clinical signs and symptoms of ischemic strokes differ in severity from hemorrhagic strokes in that they develop more gradually (over hours). Paralysis, paresis, ataxia, vomiting, and ocular gazing can all be signs of an ischemic stroke. The precise lesion site will decide the symptoms and indicators the patient may experience. [7]

Myocardial infarctions, hypertension, and usage of thrombolytics are predisposing variables that dramatically raise the likelihood of experiencing a hemorrhagic stroke. [8] Hemorrhagic strokes can appear differently clinically from case to case. Patients typically arrive with intense headaches, vomiting, and extremely high blood pressure. Focal neurological symptoms that appear within minutes go along with these clinical presentations. Although they can occasionally happen with any other form of stroke, these acute symptoms have been determined to predominantly be associated with hemorrhagic strokes. [9]

A "mini-stroke" or TIA is a type of ischemic stroke (transient ischemic attack). This is a temporary obstruction in the blood supply to your brain. The symptoms typically disappear within a few minutes or sometimes within a day. TIAs are often referred to as "warning strokes" at times. It is crucial to understand that a TIA can be an indication of an upcoming stroke. Similar to a serious stroke, a TIA is a medical emergency. Emergency care is required for strokes and TIAs. It is impossible to tell at first whether symptoms are caused by a TIA or a serious type of stroke. Blood clots frequently induce TIAs, much like ischemic strokes do.

A Review on Post-Stroke Management

Within a year, a massive stroke occurs in more than one-third of TIA patients who do not receive medical attention. Within three months of a TIA, 10% to 15% of patients will experience a severe stroke. Major stroke risk can be reduced by identifying and treating TIAs. If you experience a TIA, your medical team can determine the cause and take action to stop a catastrophic stroke. [10] Early stroke diagnosis, combined with the identification of the type and etiology of the stroke, is the primary prognostic factor that will reduce the rate of complications and persistent morbidities. The kind of stroke can be determined with the aid of a detailed medical history and a complete physical examination. [11] Imaging is however still necessary to make a diagnosis. The preferred imaging technique to distinguish between a hemorrhagic and an ischemic stroke is a non-contrast CT scan. In hospitals and emergency rooms, it isn't always accessible, though. Because of this, researchers have tried to develop protocols for differentiating between the two categories based simply on clinical signs. The kind can be distinguished by factors including localized versus diffuse symptoms, positive versus negative symptoms, and gradual versus rapid onset of symptoms. Additionally, several research claimed that eye gazes and pupil size can aid differentiate between kinds. [12]

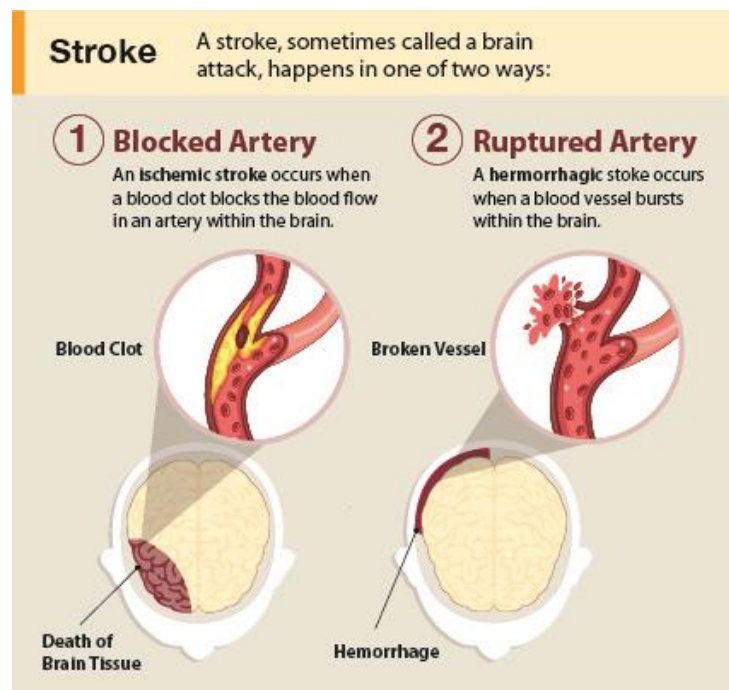


Fig.2: Types of Stroke [137]

1.2 Pathophysiology of Stroke

The term "stroke" refers to a sudden neurological attack caused by blocked blood arteries in the brain. To research the clinical manifestation of the stroke, it is crucial to comprehend the neurovascular anatomy. [13] Two internal carotid arteries in the front and two vertebral arteries in the back control the blood flow to the brain (the circle of Willis). [14] Hemorrhagic strokes are brought on by bleeding or blood vessel leaks, whereas ischemic strokes are brought on by insufficient blood and oxygen reaching the brain. [15] Both vascular risk factors and other general predisposing factors that may function as a trigger have an impact on a person's likelihood of having a stroke. In certain circumstances, the stroke might be directly linked to a valid cause. The internal carotid artery being dissected and blocked as a result of a neck injury is an illustration of this. [16] In contrast, this reliable cause is absent in the majority of other instances. Furthermore, the exact mechanisms by which conditions including pregnancy, systemic illnesses, and psychological stress lead to a stroke remain unknown. [17] According to some hypotheses, this may be due to vascular inflammation and activation of the coagulation cascade. These hypotheses predict that coagulopathies and vascular abnormalities will interact with one other and with other risk factors to accelerate the development of stroke (either hemorrhagic or ischemic). [18] This notion is supported by the observation that acute stroke patients have leukocytosis and elevated inflammatory markers, which are poor prognostic indicators. [19] Overall, because of the gaps in our understanding, more research is still required to fully understand the causes and risk factors of strokes as well as to detect high-risk patients early. [20]

Comparing the brain to other body organs, it is commonly believed that the brain is more susceptible to hypoxia injury. [21] This is mostly due to the high quantities of glutamate (a neurotransmitter) and the relatively high metabolic activity. [22] Thus, cerebral vascular blockage from emboli or an in-situ thrombus can result in hypoxic damage. [23] Distant emboli typically come from the carotid artery, left atrium, or left ventricle (specifically from an atherosclerotic plaque). Immediately following this blockage, neurological symptoms will appear. [24] However, depending on the injury site, the cellular makeup, the vulnerability, and the amount of the lesion, lasting damage, and cell death will manifest over a period of minutes to hours (or even days). [25]

A Review on Post-Stroke Management

Vascular reaction to injury is a significant element that also affects the outcome. An illustration of this is the absence of a vasodilator, which will cause a bigger stroke. [26] On the other side, less damage will result from the clot being dissolved early in the flow recovery process. [27] To accomplish this, recombinant tissue plasminogen activator (tPA) is occasionally administered; in fact, it is the only medication approved for this purpose by the FDA. [28] Due to the extensive list of side effects associated with tPA, however, less than 10% of stroke patients are eligible for its usage. [29] It is linked to a high risk of bleeding as well as other undesirable side effects such as harm to the blood-brain barrier (BBB). Combination medicines are used in some regimens to reduce the likelihood of side effects and increase the drug's therapeutic window. [30] Whether the tissue could be saved or not in situations where the wounded vessels were unable to regain blood flow was not widely known until recently. In most cases, the brain tissue could continue to function for hours after a vascular occlusion occurred. To safeguard these tissues and possibly keep them viable, enough vascular blood flow must be restored as quickly as feasible. [31]

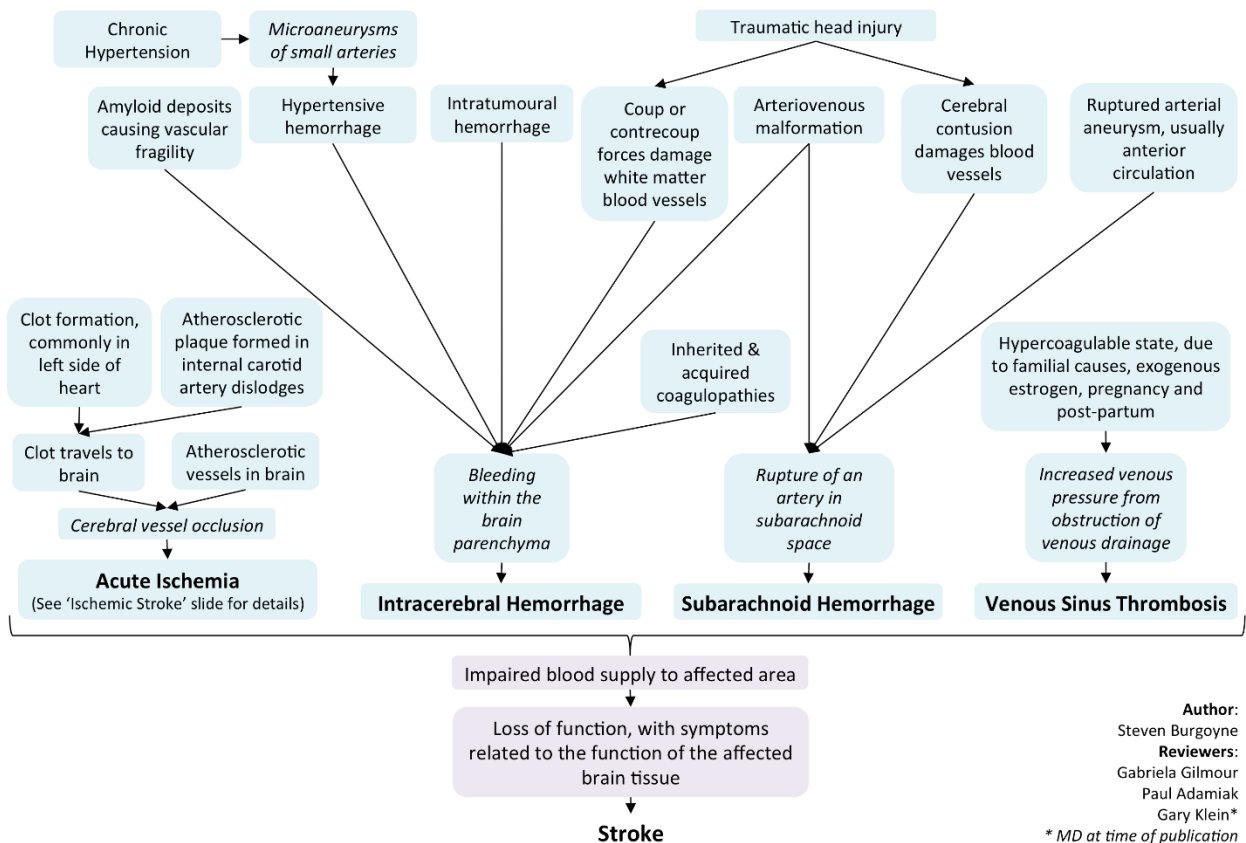
Around 85% of stroke patients lose their lives as a result of ischemic occlusions, with intracerebral hemorrhage accounting for the remaining 15%. Ischemic occlusion in the brain leads to thrombotic and embolic circumstances. [32] Vascular constriction brought on by atherosclerosis in thrombosis interferes with blood flow. Plaque accumulation will eventually narrow the vascular chamber and create clots, leading to thrombotic stroke. In an embolic stroke, the reduced blood supply to the brain region causes an embolism; the reduced blood flow to the brain causes extreme stress and premature cell death (necrosis). Following necrosis, the plasma membrane is disrupted, organelles enlarge, cellular contents seep into extracellular space [33], and diminished neural activity. Inflammation, energy failure, loss of homeostasis, acidosis, elevated intracellular calcium levels, excitotoxicity, free radical-mediated toxicity, cytokine-mediated cytotoxicity, complement activation, deterioration of the blood-brain barrier, activation of glial cells, oxidative stress, leukocyte infiltration are additional significant events that contribute to stroke pathology. [34]-[38]

Hemorrhagic strokes have a significant fatality rate and make up about 10% to 15% of all strokes. Blood vessels rupture as a result of internal damage and stress on the brain tissue in this illness. It causes circulatory system toxicity and infarction as a result. [39] It is divided into a

A Review on Post-Stroke Management

subarachnoid and intracerebral hemorrhage. Blood arteries burst in ICH, which results in an abnormal blood buildup inside the brain. Hypertension, disturbed vasculature, excessive anticoagulant, thrombolytic drug use, and ICH are the main causes. Due to a brain injury or cerebral aneurysm, subarachnoid hemorrhage is a condition in which blood builds up in the subarachnoid space of the brain. [40][41]

Stroke: Pathogenesis



Legend: Pathophysiology Mechanism Sign/Symptom/Lab Finding Complications Published October 28, 2015 on calgaryguide.ucalgary.ca/



Fig.3: Pathophysiology of stroke [138]

1.3 Supervision of Stroke

Post-stroke management refers to the treatments given to stroke survivors after a stroke. Non-pharmacological management and pharmacological management are the two basic categories of management. Non-pharmacological management includes lifestyle modifications, dietary habits, and mental health conditions. On the other hand, the core of pharmacological management is drug management. Non-pharmacological management control pain soothes stress, promotes mood improvement, lessens depression, and heightens awareness of oneself and the surroundings.

It encompasses both non-pharmacological and pharmacological treatments. "Prevention is better than cure" According to WHO research, non-pharmacological management is preferred over emergency care for stroke. [42] This type of management includes patient education, adequate hydration, exercise, dietary modification, smoking cessation, reduction in salt intake, decrease in body mass index and regular checking, control of blood pressure and blood glucose level, [43], and meetings with consultant neurologist, ophthalmologist, and physiotherapist, aspirin should be used for long periods for paralysis.[44]

Stroke prevention entails altering risk factors within a group or among specific individuals, while stroke management relies on treating its pathophysiology. Despite extensive studies into stroke over the past 20 years, there is still no easy way to treat or prevent all of the clinical causes of stroke. The main goal of modern stroke research is to develop innovative treatments that alter risk factors for both major and minor strokes. [45]

A Review on Post-Stroke Management

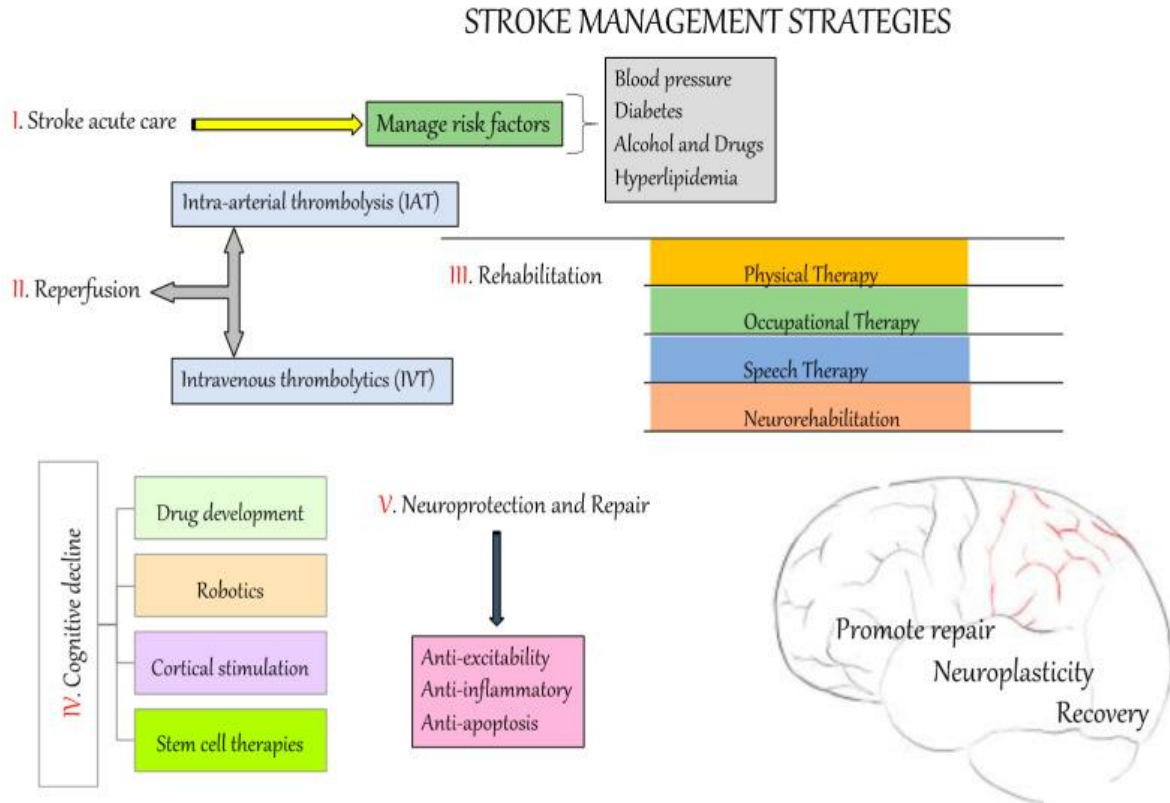


Fig.4: Stroke management strategies [139]

Chapter two

Purpose of the study

2.1 Purpose of the study

- To have a deeper understanding of the various diagnostic techniques utilized to identify this illness.
- To know treatment & management of stroke.
- To make the awareness of the people as well as to gather information regarding this.

Chapter three

Methodology

3.1 Methodology

A methodological evaluation **from whole data** provides a framework for exploration approaches in addition to techniques for data collection and analysis. The methods employed in the investigation are covered in this chapter. Key phrases with "stroke," "stroke pathophysiology," "stroke management," and "post-stroke management" were explored by utilizing web-based search engines, PubMed, Google Scholar, Research Gate, and Springer as well as from many websites. The learning environment is described in it. This is a summary of earlier research on the display of post-stroke management. All research on the types, pathophysiology, and management of the stroke ailment. A portion of the data was gathered by reading the original study articles, while another portion was found by searching the internet for relevant material. Many management activities were documented. All of the data acquired from earlier study articles were introduced and arithmetically coded.

Paper search link:

<https://scholar.google.com/>

<https://www.researchgate.net/>

<https://pubmed.ncbi.nlm.nih.gov/>

<https://link.springer.com/>

Chapter four

Results & Discussion

4.1 Non-pharmacological Management

The value of non-pharmacological stroke management for the general population must be emphasized, as well as the fact that it should continue whether or not pharmacotherapy is used to prevent strokes. A healthy diet, frequent exercise, a low-normal body mass index, quitting smoking, and moderate alcohol consumption are all essential components of a healthy lifestyle that can prevent strokes without the use of medications. It is crucial to educate patients about the significance, worth, and advantages of non-pharmacological stroke management, especially when it is the only available treatment choice if pharmacotherapy prevention has negative side effects. Many studies showed that even little lifestyle changes could drastically lower the risk of stroke. As a result, to avoid both primary and secondary strokes, physicians must advocate for a moderate and healthy lifestyle. [46]

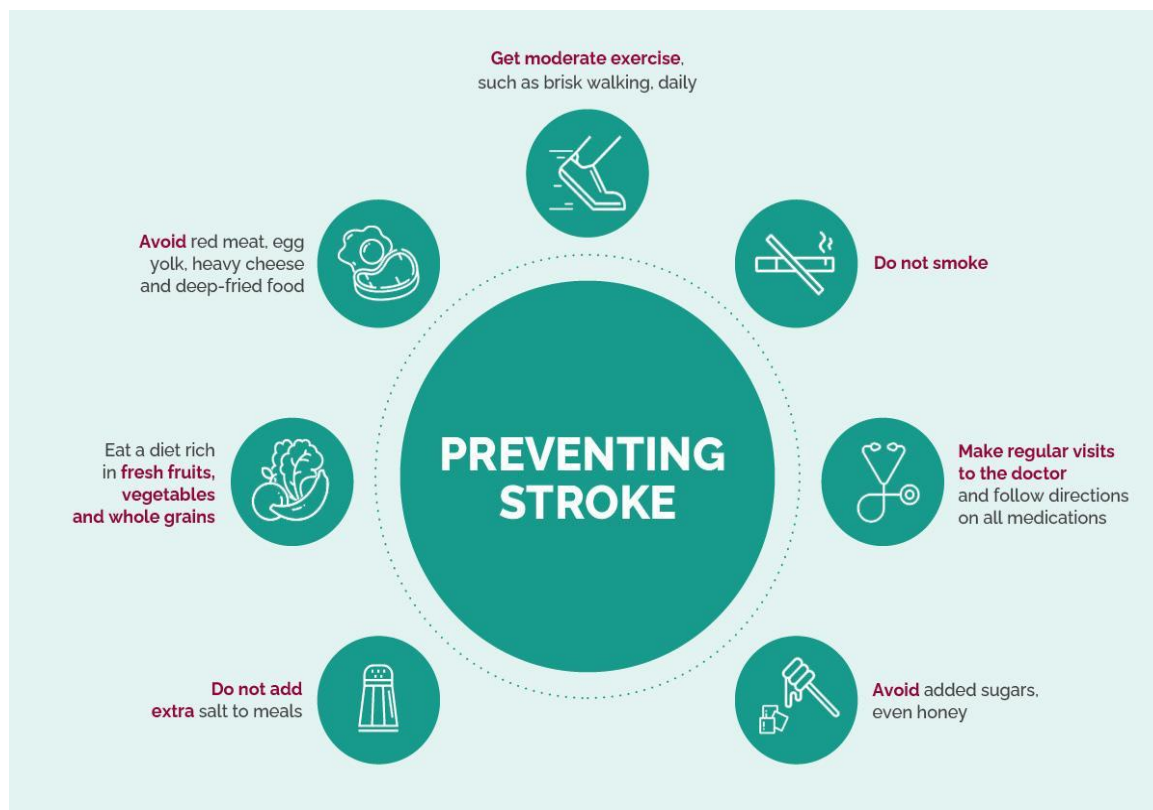


Fig.5: Non pharmacological management of stroke [140]

4.1.1 Lifestyle Modifications

4.1.1.1 Physical activities

Stroke patients need to collaborate with their medical professionals to create an exercise regimen that reflects their distinct side effects and fitness levels. The diverse set of aerobic workouts to help avoid another stroke and neuromuscular training to aid recovery will likely be encouraged by doctors and therapists. [47]

Maintaining a healthy weight and lowering your cholesterol and blood pressure are all benefits of physical activity. The surgeon general advises 2 hours and 30 minutes of moderate-intensity aerobic exercise per week for adults, such as a brisk stroll. Children and teens should get 1 hour of physical activity every day. [48]

Following a stroke, doctors advise aerobic activity for 20 to 60 minutes per day, three to seven days a week. The dosage should be adjusted based on the patient's level of fitness. Muscle atrophy, which typically takes place during the hospital stay and in the days following, can be reversed with strength-training exercises. Lightweights that provide at least one set of 10 to 15 repetitions should be used in strength-training regimens. Two to three days per week should be dedicated to strength training, with eight to ten exercises targeting the main muscle groups. Setting your post-stroke fitness goals in collaboration with your medical team is essential. For instance, to establish the motivation for regular aerobic exercise, certain patients might need to prioritize gait training. Gait training and other forms of aerobic exercise might be acceptable for other patients with moderate secondary effects. [49] Stroke patients should avoid vigorous exercises.

For muscles that may have been lost or weakened after a stroke, stroke survivors should also use light weights and apply minimal resistance. You should perform two to three sessions of this kind of exercise. [50] Stroke victims should stretch frequently to improve flexibility in addition to aerobic exercise and strength training. Your legs and arms may be challenging for you to raise. To make your movements easier to complete, try stretching before or after your other workouts. [51]

Since stroke survivors frequently lose their stability, you might also want to include balancing training. Stroke survivors are advised to include a couple of sessions of balance or cognitive exercises in their daily regimen. [52]

A Review on Post-Stroke Management

Several healthcare professionals also advise stroke survivors to participate in group exercise activities as a source of encouragement for them both physically and emotionally. [53] They can include exercises like Pilates, Zumba, spin, or water aerobics. You might also look into participating in enjoyable group activities like biking or walking. As you bring fitness into your life during this difficult period, having that communal element in your fitness journey may provide you with a lot of strength, enthusiasm, and legitimacy. [54]

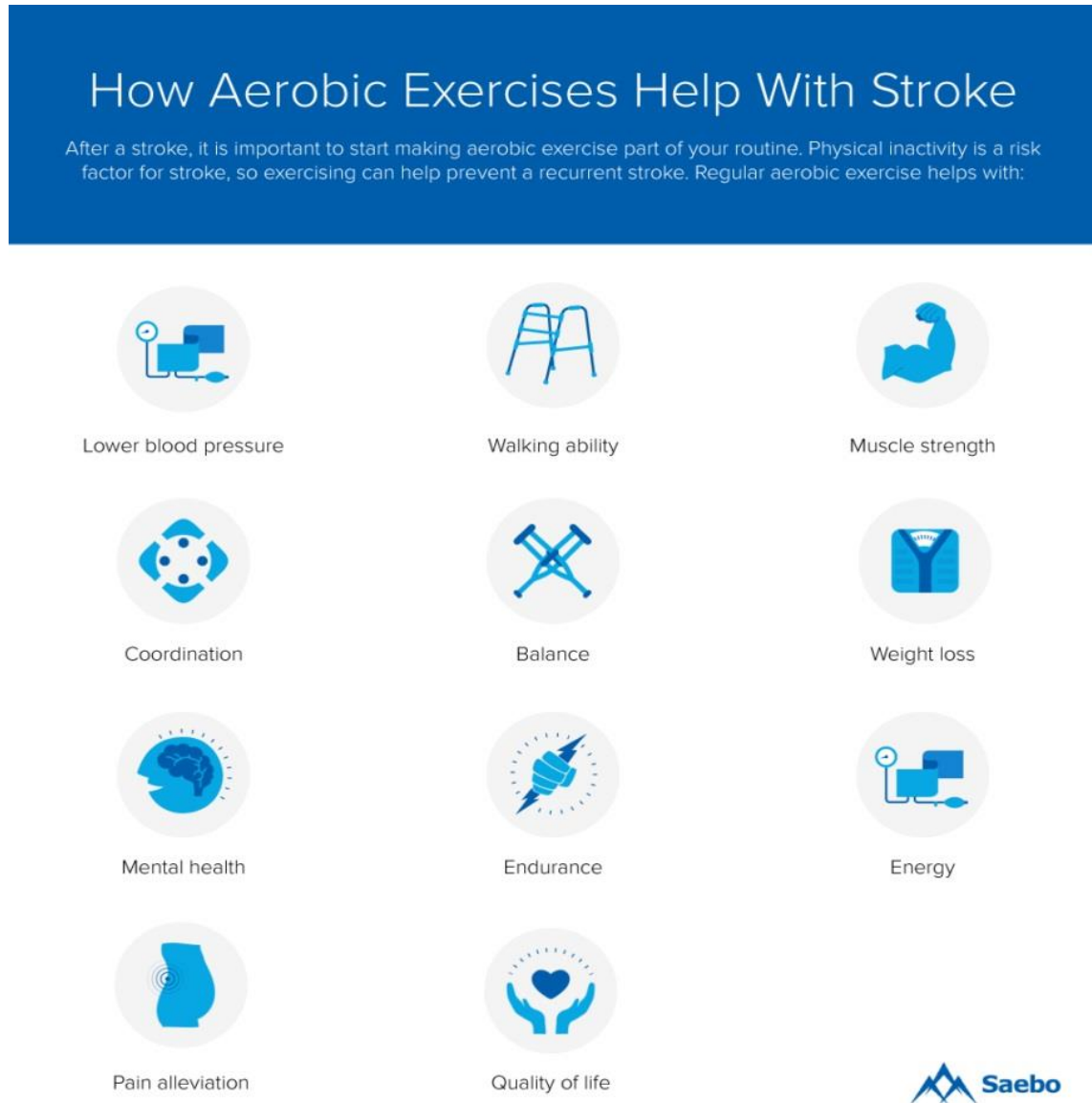


Fig.6: Physical Exercise after Stroke [141]

4.1.1.2 Habituation

It is widely acknowledged that **alcohol** misuse has both short-term and long-term impacts on the brain. [55] Recently, it was mentioned that another risk for alcohol drinkers is stroke. [56] Overindulging in alcohol might increase the risk of stroke and blood pressure. Moreover, it raises blood levels of triglycerides, a type of fat that might harden your arteries. [57] Alcohol consumption regularly is linked to hypertension, [58] intracranial hemorrhage; both lethal and not, [59] cerebral infarction, [60] as well as an elevated risk of stroke-related mortality. [61] Newer, less rigorously controlled research indicates that alcohol use is a risk factor for cerebral infarction [62] in young adults who occasionally drink alcohol [63] and in middle-aged women and young men who occasionally drink alcohol and regularly consume large amounts of alcohol. [64] Alcohol use also may potentially increase the risk of subarachnoid hemorrhage. [65] Alcohol can raise your risk of getting another stroke if you've already had one. It may worsen the effects of speech, cognitive, visual, and balance difficulties brought on by your stroke. [66] So, after a stroke alcohol intake should be avoided.

Stroke risk is also increased by tobacco usage. **Smoking** cigarettes can harm your heart and blood vessels, which raises your risk of stroke. [67] Cigarette smoke contains carbon monoxide, which lowers your blood's ability to carry oxygen, and nicotine, which elevates blood pressure. Consuming other people's secondhand smoke can increase your chance of having a stroke even if you don't smoke. [68] Continued smoking raises the chance of another stroke after the first stroke. The risk increases with the amount of smoking. Smoking after a stroke raises the risk of dying from a heart attack or stroke. You must stop smoking right away if you have ever experienced a stroke or TIA. [69] According to estimates, up to two years after a stroke, more than 75% of people who smoked before having a stroke continued to smoke, and their smoking habits didn't change much in the years after. [70] Persistent smoking has been linked to several negative outcomes for stroke survivors, including an increased risk of subsequent stroke and other cardiovascular diseases and an increased mortality risk. [71] After a stroke, quitting smoking is typically advised to avoid myocardial infarction and subsequent strokes. [72] There is still little information about changing one's smoking habits after having a stroke. [73]

4.1.2 Dietary Habit

Eating healthily has numerous advantages. A healthy food plan can help you manage your weight, increase your immunity, build strong bones, and reduce your risk of stroke. It also helps you stay active and offers the right fuel and energy for that. Food, or the foods we eat, is a modifiable risk factor, which means that changes in your diet or behavior can have a direct impact on how well or how poorly your health turns out. [74] It should come as no surprise that some diets can alter the risk of stroke. The foods or diets that have been linked to an increased risk of stroke are included in the table below, along with potential explanations: [75]

Table 1:

Risky foods	Reasons
Prepacked meals and snacks	Trans fats are abundant, raising LDL (bad cholesterol) and lowering HDL (good cholesterol)
Processed meats	Blood arteries may be harmed by preservatives (such as sodium nitrite and nitrate).
Cakes, cookies, and pastries	Contains a lot of presumably hydrogenated oils and trans fats, which diminish HDL and elevate LDL.
Spreads and processed foods	Trans fats are abundant, raising LDL and lowering HDL
Soda pop (regular & diet)	▪ Regular: High in sugar with no discernible nutritional advantages ▪ Diet: Artificial sweeteners and the risk of stroke: a source of worry

A Review on Post-Stroke Management

Fried foods	Trans fats are abundant, raising LDL and lowering HDL
-------------	---

Another food group that has a negative reputation is **red meat**, but there is still much to learn about this subject. A report in the journal Stroke revealed that stroke incidence was 42% greater in women who habitually ate large quantities of red meat. [76] Unprocessed red meat consumption and stroke are associated, according to a recent study published in October 2022, but the data is poor. [77]

Before reviewing a healthy post-stroke diet, remember that occasionally consuming one of these food groups won't significantly impact your performance or short- or long-term health, lowering your chance of recurrent stroke. That is a combination of both portion management and a long-term, balanced diet is responsible. [78]

A Review on Post-Stroke Management

Table 2: Healthy foods that decrease the risk of stroke: [75]

Foods decreased risk	Reasons
Salmon and Fish Oils	Omega-3 fatty acids and polyunsaturated fats reduce inflammation, lessen the plague, halt blood clots and lower triglycerides, and increase HDL (the good cholesterol)
Whole Nuts	Rich in dietary protein and "healthy fats" Some contain a lot of amino acid arginine, which maintains the health of blood vessels
Fruits and Vegetables	Very good source of minerals and vitamins, rich in fiber, and disease-prevention tools including stroke.
Berries	High antioxidant content and supports a healthy gut
Olive Oil	It is believed that apolipoprotein prevents the accumulation of lipids, cholesterol, and other chemicals on the walls of arteries.
Whole Grains	Reduces cholesterol levels, provides fiber, and nutrients, and is believed to minimize the chance of developing heart disease and other illnesses.

A Review on Post-Stroke Management

Randomized controlled trials haven't looked into how frequently drinking **tea or coffee** affects cardiovascular risk. Drinking coffee and tea lowers the risk of myocardial infarction, according to relatively strong evidence, although stroke is barely mentioned in the same body of literature. A reduced risk of myocardial infarction would presumably result in decreased risk of stroke. [79] There are two things to take into account for both tea and coffee: the effects of caffeine and the effects of antioxidants. In research, slow metabolizers of caffeine seemed to be associated with an increased risk of myocardial infarction, providing some indication that caffeine in coffee may be hazardous. [80] According to a recent meta-analysis, drinking three to five cups of coffee daily lowers the risk of cardiovascular disease. [81] Green tea and other types of tea both lower the risk of cardiovascular disease. Blood pressure and LDL cholesterol are both reduced by green tea [82] as well as enhancing endothelial performance [83] Both tea and coffee consumption seems to have advantages. It is unclear whether drinking both tea and coffee would be more advantageous. It seems doubtful that doctors would recommend one or the other given that drinking these beverages is a matter of personal preference and is influenced by cultural differences between nations. [84] Thus, individuals who are at risk for stroke should limit their consumption of animal flesh and stay away from red meat and egg yolk. They should consume a lot of whole grains, vegetables, fruits, and legumes, as well as healthful oils like olive and canola. Because egg whites are a good source of protein, meals that often contain meat can be replaced with egg white-based dishes such as omelets, frittatas, and egg salad sandwiches. [85] Patients must approach their meatless day as a learning opportunity rather than a punishment to achieve this.

Dietary Changes After a Stroke

Not to Eat		Eat	
 mayonnaise	 sausage	 tofu	 high-fiber cereal
 fatty fried foods	 chips	 nuts	 milk
 sweets	 saturated fat	 greek yogurt	 olive oil
 margarine	 fizzy drinks	 fish	 high fiber bread
 red meat	 fast food	 vegetables	 fruits

Fig.7: Foods after stroke [142]

4.1.3Mental Health

A stroke is a significant life-changing event. It may alter your self-perception and cause you to worry about the future. Stress and unhappiness can be brought on by changes in obligations, relationships, jobs, and finances. Changes in personality, mood, and emotions can also result from a stroke's effects on the brain. All of this suggests that there is a close connection between anxiety, depression, and stroke. In the five years following a stroke, one in three persons experience depression. Depression can strike at any time, although it is most frequent in the first year following a stroke. Moreover, anxiety may manifest, either on its own or in tandem with depression. [86]

The most common psychiatric side effect of stroke is post-stroke **depression** (PSD). In comparison to stroke patients who are not depressed, stroke patients with PSD have higher fatality rates and exhibit little improvement in rehabilitation programs. [87] The quality of life for individuals following a stroke is greatly decreased by the co-occurrence of depression and stroke. [88] Early detection and appropriate care are essential to achieve better outcomes in PSD patients. [89]

25% of stroke survivors experience **anxiety**. There are no reliable cures. Post-stroke anxiety may be influenced by several variables, including post-stroke depression, pre-stroke anxiety and depression, locus of control, coping mechanisms, confidence, exhaustion, and sleep. These variables may be the subject of therapy. [90] During the first year after a stroke, anxiety is frequent. It may be worthwhile to conduct further routine screening after a stroke because anxiety has a major impact on quality of life and is a predictor of depression. [91]

Anger after a stroke is a regular occurrence and negatively impacts functionality and quality of life. [92] After a stroke, it's a common neuropsychiatric symptom that can interfere with treatment and recovery. This is especially true because anger affects how well patients adhere to their treatment and how well they learn about their health conditions. [93] There are numerous causes of anger after a stroke. Some are caused by the biological effects of the stroke, while others are caused by unwelcome lifestyle adjustments, such as losing a job because of limitations associated with the stroke. As rage problems following a stroke can upset both survivors and their loved ones, it's critical to treat them. The frequency and severity of post-

A Review on Post-Stroke Management

stroke anger and aggressive behavior episodes frequently decrease over time. Survivors might naturally regain control of their emotions as their brains recover and as they become used to their new surroundings. Yet rather than waiting for things to go better on their own, controlling emotions is best done proactively. [94]

Despite being widespread, depression and anxiety are also very treatable. Numerous factors can support recovery, and it is achievable. You will be able to heal more quickly the earlier you seek assistance. Some psychological therapy may help if you have mild or moderate mental issues. [95]

- **Behavior modification:** The aim of behavior therapy is on engaging in rewarding, enjoyable, or satisfying activities. It seeks to reacquaint you with life.
- **Cognitive behavior treatment (CBT):** Creates a more positive and problem-solving approach by assisting you in recognizing and changing problematic thought patterns. It is among the best therapies for depression.
- **Interpersonal counseling:** Assists you in identifying interpersonal patterns that increase your risk of depression. You put your attention on developing relationships, dealing with grief and sadness, and learning new methods to get along with people.
- **Mindfulness Cognitive treatment:** Meditation-based group treatment. You learn to concentrate on the current moment without attempting to change it through mindfulness meditation. Stopping your thoughts from straying toward the past or the future can be helpful. It enables you to catch sad or negative feelings before they fester.
- **Art Therapy:** One type of psychological therapy that can help stroke victims with sadness and anxiety is art therapy. Stroke sufferers can use art to communicate their internal conflicts, feelings, and psychological status, which will aid in the healing of their mental issues. [96]

Positive social interaction can significantly impact stroke victims' physical and mental health. There is evidence to support the idea that family members, especially spouses, offer significant social support. This **family support** could affect how well stroke victims recover after therapy. [97]

4.2 Pharmacological Management

Pharmacological management is also known as drug management. Stroke-specific treatment and stroke prevention include the two main categories of pharmacological therapy. [98] The type of stroke you have, the afflicted area of the brain, and the underlying reason all determine how you are treated. Medications are frequently used to treat strokes. These include drugs that lower cholesterol, lower blood pressure, and prevent and dissolve blood clots. Procedures could be necessary in some circumstances to get rid of blood clots. If your stroke was brought on by brain swelling, surgery might also be required to correct it and lower the risk of more bleeding. [99]

You have a 25% to 35% probability of getting another stroke after having one. Your doctor will prescribe medicine to reduce those chances. [100] Depending on whether a stroke is ischemic, hemorrhagic, or transient ischemic attack (TIA), different pharmacological management is used to treat it. [101] A stroke patient may get or be prescribed a variety of treatments, including the clot-busting agent tPA, blood thinners, and medications to control cholesterol and high blood pressure. [102]

4.2.1 Tissue Plasminogen Activator (tPA)

The only stroke medication that truly dissolves a blood clot is tissue plasminogen activator (tPA). It is frequently applied as an emergency stroke therapy. [103] With improved results and minimal risk of bleeding when treatment is started promptly, tPA was shown to be effective in animal models of stroke in multiple trials conducted between 1984 and 1991. [104]-[109] A drug called **alteplase** injections, which break blood clots and restore blood flow to the brain, are frequently used to treat ischemic strokes. Thrombolysis is the term for this application of "clot-busting" medication. Starting alteplase as soon as feasible after the stroke occurs, and most definitely within 4.5 hours, will maximize its effectiveness. It's not typically advised if more than 4.5 hours have gone because it's unclear how useful it is after this period. It's critical to confirm the diagnosis of an ischemic stroke with a brain scan before alteplase can be administered. This is because the medication has the potential to worsen hemorrhagic stroke-related bleeding. [110] For tPA to effectively treat the blood clot, it is injected into a vein. Not everybody uses tPA. tPA is not administered to people who have a significant risk of a brain hemorrhage. [111] Getting tPA can lessen the effects of a stroke and even help you recover

A Review on Post-Stroke Management

more rapidly by reversing some of its effects. Other therapies are necessary for some situations where tPA cannot be employed. [112]

4.2.2 Blood Thinners

Blood thinners are frequently used in post-stroke management to help avoid blood clots and additional strokes. The choice of blood thinner is based on several variables, including the type and severity of the stroke, the patient's age, medical history, and other medications being used. Blood thinners come in two varieties: **anticoagulants** and **antiplatelet** medications.

4.2.2.1 Anticoagulants

Anticoagulants have been utilized in the emergency care of individuals with acute ischemic stroke for a long time. Anticoagulants are administered to patients, particularly those who have cardioembolism brought on by arterial fibrillation and large-artery atherosclerotic disease, to avoid a first or subsequent stroke. [113] To assist lower their chance of forming further blood clots in the future, some persons could be recommended an anticoagulant. Anticoagulants work by altering the blood's chemical structure in a way that prevents blood clots from forming.

Anticoagulants for long-term use include **warfarin**, **apixaban**, **dabigatran**, **edoxaban**, and **rivaroxaban**, among others.

Heparins, a class of anticoagulants that can only be administered by injection and are only used temporarily, are another class of anticoagulants. [114]

Anticoagulants are potent medications. You often take them if your risk of stroke is high or if you have atrial fibrillation, a disorder marked by irregular pulse. [115]

Moreover, life-threatening, severe bleeding has been connected to **warfarin**. If you have a bleeding issue or have had heavy bleeding, let your doctor know. Your doctor will probably think about using another medication. [116]

4.2.2.2 Antiplatelet agents

When platelets become activated in sufferers of acute ischemic stroke, they can form blood clots that block arteries in the brain and cause damage to some of the brain tissue. The effects of such damage include stroke symptoms. Antiplatelet therapy may lessen the amount of brain tissue that has been destroyed by ischemia as well as lower the risk of having an early recurrent ischemic stroke, which would lower the likelihood of early death and enhance long-term results for survivors. [117]

ASA (acetylsalicylic acid, Aspirin) is the antiplatelet medication that is most frequently utilized. [118] After an ischemic stroke, aspirin is often administered to the majority of patients. Aspirin is a pain reliever as well as an antiplatelet, which lowers the possibility of another clot forming. [119] The only oral antiplatelet medication that has been studied for the management of acute ischemic stroke is aspirin. Between 24 to 48 hours of an ischemic stroke, but not sooner than 24 hours after alteplase medication is finished, aspirin therapy should start. [120] As an alternative, it is advised that patients who cannot receive thrombolytic therapy start taking aspirin as soon as possible. Aspirin has a favorable effect when given during an acute ischemic stroke, even though it increases the risk of bleeding. [121]

Afterward, other antiplatelet medications including **clopidogrel** and **dipyridamole** might be utilized. [122] Blood clots can be avoided by using antiplatelets like clopidogrel. They function by making it more difficult for your blood's platelets to clump together, which is how blood clots are formed. [123]

The most frequently observed side effect of blood thinners is bleeding. The majority of bleeding is mild, such as gum bleeding, light bruises, or cuts that take longer to clot. While serious bleeding is uncommon, it can be fatal. Blood in your vomit or bloody, extremely dark, or black feces may indicate stomach or intestinal hemorrhage. A head injury or sudden headaches could indicate internal bleeding. [124]

A Review on Post-Stroke Management

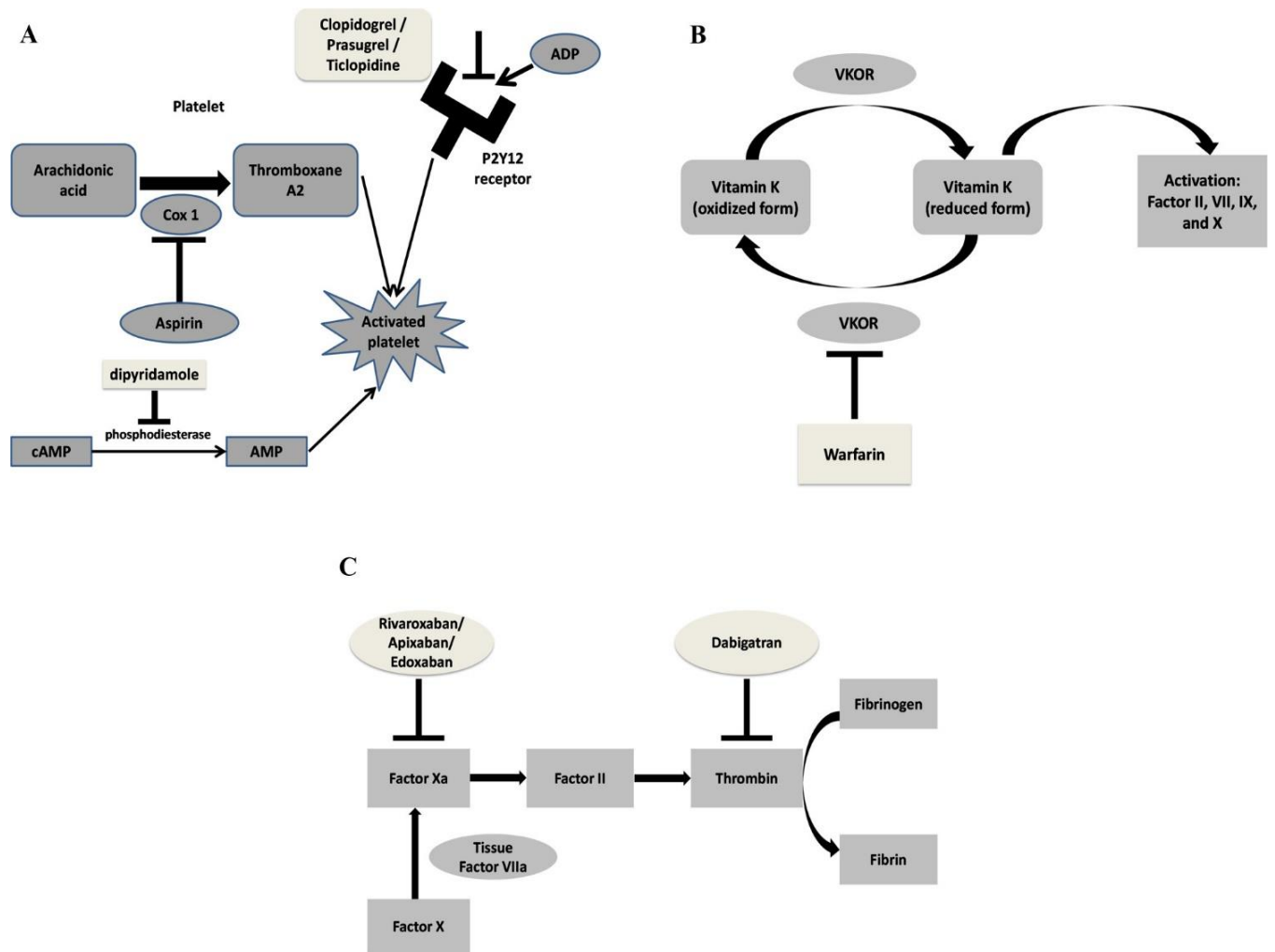


Fig.8: Blood Thinners Mechanism [143]

4.2.3 Blood Pressure Lowering Medication

Also known as antihypertensive drugs. Blood pressure and stroke are continuously correlated and unaffected by other risk variables. The substantial correlation between high blood pressure and stroke, including both ischemic stroke and ICH, has been demonstrated by persuasive evidence for more than 30 years. [125] When a hemorrhagic stroke is in its acute stage, blood pressure often rises. This increase in blood pressure has been associated with a higher risk of mortality, disability, and the formation of hematomas. Reducing the patient's risk of having another stroke is also accomplished by controlling blood pressure. [126] It is now plainly obvious that hypertension management, including blood pressure screening, is crucial to preventing strokes. [127] **Thiazide diuretic agents, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and calcium-channel blockers** are only a few kinds of antihypertensive drugs that have been demonstrated to reduce the incidence of cardiovascular events, including stroke. For the prevention of stroke, the use of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may have a specific benefit. [128] According to a significant meta-analysis of cardiovascular outcomes, beta-blockers may not be as effective at preventing strokes as other antihypertensive medications. Each patient's choices must be personalized specifically to them. [129] Antihypertensive medications should be used to treat hypertensive patients so that their target blood pressure is less than 140/90 mmHg. The successful reduction of BP is more crucial in lowering the risk of stroke than the selection of a particular agent, and treatment should be tailored to the patient's other characteristics and tolerance to the medicine. Antihypertensive drug treatment candidates and the desired level of blood pressure management are chosen for hypertensive stroke patients based on the clinical disease type, time since onset, severity, age, and antithrombotic medication use. [130] A Cochrane review of randomized controlled trials examining the use of blood pressure medication to treat individuals with a history of stroke or TIA in the prevention of major vascular events, dementia, and recurrent stroke. At least 48 hours after a stroke or TIA, blood pressure-lowering medication was started. The findings, according to the authors, support the use of BP-lowering medications in stroke or TIA patients to lower the risk of having another stroke. They also noted that the majority of the current evidence comes from trials examining

A Review on Post-Stroke Management

ACE inhibitors or diuretics and that there is currently insufficient data to draw firm conclusions about the ideal systolic BP target following stroke or TIA. [131]

Table 3: Blood pressure lowering drugs: [118]

Classes	Examples	Function
ACE Inhibitors	Lisinopril, Perindopril and Ramipril etc.	Widen blood vessels by relaxing them. This facilitates easier blood flow.
Angiotensin Receptor Blockers	Losartan, Valsartan and Telmisartan etc.	By preventing a substance that narrows blood arteries, you can make it simpler for your heart to pump blood.
Beta Blockers	Propranolol, Metoprolol, Labetolol, etc.	Stop the effects of the hormone adrenaline to reduce the rate and pressure of heartbeat.
Calcium Channel Blockers	Amlodipine, Nicardipine and Nimodipine etc.	By preventing calcium from entering the cells in your heart and blood vessels, you can aid in the relaxation and opening up of your blood vessels. They can also reduce blood pressure by slowing your heart rate.
Thiazide Diuretics	Hydrochlorothiazide, Chlorthalidone, and Indapamide etc.	These medicines treat heart failure and excessive blood pressure. Assist your body in eliminating surplus salt and liquids. These may be taken along with other blood pressure medications.

4.2.4 Cholesterol-Lowering Drugs

Your physician will most likely prescribe you a statin, a drug that lowers cholesterol, after a stroke. Considering that statins appear to reduce the chance of having a second stroke. [132] Statins aid in reducing elevated cholesterol levels. Cholesterol might begin to accumulate along the artery walls when your cholesterol levels are too high. The accumulation is known as plaque. The HMG-CoA reductase enzyme, which your body needs to generate cholesterol, is blocked by these medications. Your body produces less of it as a result. By doing so, plaque risk is decreased and ministrokes are avoided. [133] An increased risk of ischemic stroke is linked to elevated LDL and total cholesterol. The use of niacin, fibrates, and cholesterol-absorption inhibitors is also an option for individuals who cannot take statin medication, even though statin therapy has the most evidence to support it in the literature for the prevention of recurrent ischemic stroke. [134] One of the most significant causes of stroke is carotid atherosclerosis. Several clinical trials looked explored the advantages of statins for stroke patients. 4731 participants participated in the SPARCL experiment (Stroke Prevention by Aggressive Reduction of Cholesterol Levels), which was followed up for five years. [135]

Table 4: Cholesterol-lowering drugs: [118]

Classes	Examples
Statins	Simvastatin, Atorvastatin & Rosuvastatin etc.
Cholesterol Absorption Inhibitors	Ezetimibe
Fibrates (Folic Acid Derivatives)	Bezafibrate, Fenofibrate & Gemfibrozil etc.
Niacin	Vitamin B
Resins	Cholestyramine, Colestipol

4.2.5 Routine Follow-up

A post-stroke routine checkup is an important part of stroke care, as it allows healthcare professionals to monitor the patient's progress, identify any potential complications or long-term effects of the stroke, and adjust their care plan as needed. During a routine checkup, the healthcare professional will perform a physical exam to assess the patient's strength, sensation, coordination, balance, and reflexes. They may also perform a neurological assessment to evaluate the patient's cognitive function, speech, and vision.

Imaging tests such as CT scans, MRIs, or ultrasounds may be ordered to check for any changes in the brain since the stroke. The healthcare professional will also review the patient's medications to ensure they are taking the correct doses and adjust any medications as needed. If the patient is still in rehabilitation, the healthcare professional will evaluate their progress and adjust their therapy plan as needed.

Additionally, the healthcare professional may discuss any lifestyle modifications the patient needs to make, such as changes to their diet or exercise routine, to reduce their risk of future strokes. They may also address any emotional or psychological concerns the patient may have and provide resources or referrals for additional support. Finally, the healthcare professional will discuss the frequency of future checkups and any additional tests or evaluations that may be needed to ensure the patient's ongoing care and recovery.

Chapter five

Conclusion

5.1 Conclusion

Post-stroke management is critical for long-term recovery and quality of life. Non-pharmacological and pharmacological both are possible ways for post-stroke management.

Non-pharmacological and pharmacological approaches are both important aspects of post-stroke management. Rehabilitation, exercise, diet, and stress management are non-pharmacological approaches that can improve recovery and prevent recurrent strokes.

Anticoagulants, antiplatelet agents, blood pressure-lowering medications, and cholesterol-lowering medications are pharmacological approaches that can improve outcomes and quality of life in stroke survivors.

A multidisciplinary approach that incorporates both non-pharmacological and pharmacological interventions can provide comprehensive care and support for stroke survivors and their families. It's important to note that post-stroke management should be individualized based on the patient's specific needs, and it should involve a multidisciplinary team approach including physicians, nurses, therapists, and other healthcare professionals.

Chapter six

Reference

References

1. Campbell, B. C. V., De Silva, D. A., Macleod, M. R., Coutts, S. B., Schwamm, L. H., Davis, S. M., & Donnan, G. A. (2019). Ischaemic stroke. *Nature reviews. Disease primers*, 5(1), 70. <https://doi.org/10.1038/s41572-019-0118-8> [PubMed] [Google Scholar]
2. Sarikaya, H., Ferro, J., & Arnold, M. (2015). Stroke prevention--medical and lifestyle measures. *European neurology*, 73(3-4), 150–157. <https://doi.org/10.1159/000367652> [PubMed] [Google Scholar]
3. Feigin, V. L., Lawes, C. M., Bennett, D. A., Barker-Collo, S. L., & Parag, V. (2009). Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review. *The Lancet. Neurology*, 8(4), 355–369. [https://doi.org/10.1016/S1474-4422\(09\)70025-0](https://doi.org/10.1016/S1474-4422(09)70025-0) [PubMed] [Google Scholar]
4. Ojaghihaghighi, S., Vahdati, S. S., Mikaeilpour, A., & Ramouz, A. (2017). Comparison of neurological clinical manifestation in patients with hemorrhagic and ischemic stroke. *World journal of emergency medicine*, 8(1), 34–38. <https://doi.org/10.5847/wjem.j.1920-8642.2017.01.006> [PubMed] [Google Scholar]
5. American Heart Association/American Stroke Association. TIA (Transient Ischemic Attack). Accessed October 6, 2016.
6. Tsao, C. W., Aday, A. W., Almarzooq, Z. I., Alonso, A., Beaton, A. Z., Bittencourt, M. S., Boehme, A. K., Buxton, A. E., Carson, A. P., Commodore-Mensah, Y., Elkind, M. S. V., Evenson, K. R., Eze-Nliam, C., Ferguson, J. F., Generoso, G., Ho, J. E., Kalani, R., Khan, S. S., Kissela, B. M., Knutson, K. L., ... Martin, S. S. (2022). Heart Disease and Stroke Statistics-2022 Update: A Report From the American Heart Association. *Circulation*, 145(8), e153–e639. <https://doi.org/10.1161/CIR.0000000000001052> [PubMed] [Google Scholar]
7. Alrabghi, L., Alnemari, R., Aloteebi, R., Alshammari, H., Ayyad, M., Al Ibrahim, M., Alotayfi, M., Bugshan, T., Alfaifi, A., & Aljuwayd, H. (2018). Stroke types and management. *International Journal Of Community Medicine And Public Health*, 5(9), 3715–3719. <https://doi.org/10.18203/2394-6040.ijcmph20183439>
8. Rymer M. M. (2011). Hemorrhagic stroke: intracerebral hemorrhage. *Missouri medicine*,

A Review on Post-Stroke Management

108(1), 50–54. [PubMed] [Google Scholar]

9. An, S. J., Kim, T. J., & Yoon, B. W. (2017). Epidemiology, Risk Factors, and Clinical Features of Intracerebral Hemorrhage: An Update. *Journal of stroke*, 19(1), 3–10. <https://doi.org/10.5853/jos.2016.00864> [PubMed] [Google Scholar]

10. American Heart Association/American Stroke Association. TIA (Transient Ischemic Attack). Accessed October 6, 2016.

11. Kolominsky-Rabas, P. L., Sarti, C., Heuschmann, P. U., Graf, C., Siemonsen, S., Neundoerfer, B., Katalinic, A., Lang, E., Gassmann, K. G., & von Stockert, T. R. (1998). A prospective community-based study of stroke in Germany--the Erlangen Stroke Project (ESPro): incidence and case fatality at 1, 3, and 12 months. *Stroke*, 29(12), 2501–2506. <https://doi.org/10.1161/01.str.29.12.2501> [PubMed] [Google Scholar]

12. Brott, T., Adams, H. P., Jr, Olinger, C. P., Marler, J. R., Barsan, W. G., Biller, J., Spilker, J., Holleran, R., Eberle, R., & Hertzberg, V. (1989). Measurements of acute cerebral infarction: a clinical examination scale. *Stroke*, 20(7), 864–870. <https://doi.org/10.1161/01.str.20.7.864> [PubMed] [Google Scholar]

13. Kuriakose, D., & Xiao, Z. (2020). Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. *International journal of molecular sciences*, 21(20), 7609. <https://doi.org/10.3390/ijms21207609> [PubMed] [Google Scholar]

14. Farb, A., Burke, A. P., Tang, A. L., Liang, T. Y., Mannan, P., Smialek, J., & Virmani, R. (1996). Coronary plaque erosion without rupture into a lipid core. A frequent cause of coronary thrombosis in sudden coronary death. *Circulation*, 93(7), 1354–1363. <https://doi.org/10.1161/01.cir.93.7.1354> [PubMed] [Google Scholar]

15. Falk E. (1985). Unstable angina with fatal outcome: dynamic coronary thrombosis leading to infarction and/or sudden death. Autopsy evidence of recurrent mural thrombosis with peripheral embolization culminating in total vascular occlusion. *Circulation*, 71(4), 699–708. <https://doi.org/10.1161/01.cir.71.4.699> [PubMed] [Google Scholar]

A Review on Post-Stroke Management

16. Kim, Y. K., & Schulman, S. (2009). Cervical artery dissection: pathology, epidemiology and management. *Thrombosis research*, 123(6), 810–821. <https://doi.org/10.1016/j.thromres.2009.01.013> [PubMed] [Google Scholar]
17. Elkind M. S. (2007). Why now? Moving from stroke risk factors to stroke triggers. *Current opinion in neurology*, 20(1), 51–57. <https://doi.org/10.1097/WCO.0b013e328012da75> [PubMed] [Google Scholar]
18. Wolf, P. A., D'Agostino, R. B., O'Neal, M. A., Sytkowski, P., Kase, C. S., Belanger, A. J., & Kannel, W. B. (1992). Secular trends in stroke incidence and mortality. The Framingham Study. *Stroke*, 23(11), 1551–1555. <https://doi.org/10.1161/01.str.23.11.1551> [PubMed] [Google Scholar]
19. Welsh, P., Barber, M., Langhorne, P., Rumley, A., Lowe, G. D., & Stott, D. J. (2009). Associations of inflammatory and haemostatic biomarkers with poor outcome in acute ischaemic stroke. *Cerebrovascular diseases (Basel, Switzerland)*, 27(3), 247–253. <https://doi.org/10.1159/000196823> [PubMed] [Google Scholar]
20. Lowe G. D. (2005). Circulating inflammatory markers and risks of cardiovascular and non-cardiovascular disease. *Journal of thrombosis and haemostasis : JTH*, 3(8), 1618–1627. <https://doi.org/10.1111/j.1538-7836.2005.01416.x> [PubMed] [Google Scholar]
21. Rothman, S. M., & Olney, J. W. (1986). Glutamate and the pathophysiology of hypoxic--ischemic brain damage. *Annals of neurology*, 19(2), 105–111. <https://doi.org/10.1002/ana.410190202> [PubMed] [Google Scholar]
22. Choi, D. W., & Rothman, S. M. (1990). The role of glutamate neurotoxicity in hypoxic-ischemic neuronal death. *Annual review of neuroscience*, 13, 171–182. <https://doi.org/10.1146/annurev.ne.13.030190.001131> [PubMed] [Google Scholar]
23. Drejer, J., Benveniste, H., Diemer, N. H., & Schousboe, A. (1985). Cellular origin of ischemia-induced glutamate release from brain tissue in vivo and in vitro. *Journal of neurochemistry*, 45(1), 145–151. <https://doi.org/10.1111/j.1471-4159.1985.tb05486.x> [PubMed] [Google Scholar]
24. Church, J., Zeman, S., & Lodge, D. (1988). The neuroprotective action of ketamine and

A Review on Post-Stroke Management

MK-801 after transient cerebral ischemia in rats. *Anesthesiology*, 69(5), 702–709. <https://doi.org/10.1097/00000542-198811000-00011> [PubMed] [Google Scholar]

25. Wieloch, T., Koide, T., Westerberg, E. 1986. Inhibitory neurotransmitters and neuromodulators as protective agents against ischemic brain damage. In *Pharmacology of Cerebral Ischemia*, ed. 1. Krieglestein, pp. 191-97. New York: Elsevier

26. Division of Chronic Disease Control and Community Intervention. Cardiovascular disease surveillance: stroke, 1980-1989. Atlanta: Centers for Disease Control and Prevention, 1994.

27. Fieschi, C., Argentino, C., Lenzi, G. L., Sacchetti, M. L., Toni, D., & Bozzao, L. (1989). Clinical and instrumental evaluation of patients with ischemic stroke within the first six hours. *Journal of the neurological sciences*, 91(3), 311–321. [https://doi.org/10.1016/0022-510x\(89\)90060-9](https://doi.org/10.1016/0022-510x(89)90060-9) [PubMed] [Google Scholar]

28. del Zoppo, G. J., Poeck, K., Pessin, M. S., Wolpert, S. M., Furlan, A. J., Ferbert, A., Alberts, M. J., Zivin, J. A., Wechsler, L., & Busse, O. (1992). Recombinant tissue plasminogen activator in acute thrombotic and embolic stroke. *Annals of neurology*, 32(1), 78–86. <https://doi.org/10.1002/ana.410320113> [PubMed] [Google Scholar]

29. National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group (1995). Tissue plasminogen activator for acute ischemic stroke. *The New England journal of medicine*, 333(24), 1581–1587. <https://doi.org/10.1056/NEJM199512143332401> [PubMed] [Google Scholar]

30. National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group (1995). Tissue plasminogen activator for acute ischemic stroke. *The New England journal of medicine*, 333(24), 1581–1587. <https://doi.org/10.1056/NEJM199512143332401> [PubMed] [Google Scholar]

31. Murata, Y., Rosell, A., Scannevin, R. H., Rhodes, K. J., Wang, X., & Lo, E. H. (2008). Extension of the thrombolytic time window with minocycline in experimental stroke. *Stroke*, 39(12), 3372–3377. <https://doi.org/10.1161/STROKEAHA.108.514026> [PubMed] [Google Scholar]

32. Musuka, T. D., Wilton, S. B., Traboulsi, M., & Hill, M. D. (2015). Diagnosis and

A Review on Post-Stroke Management

management of acute ischemic stroke: speed is critical. CMAJ : Canadian Medical Association journal = journal de l'Association medicale canadienne, 187(12), 887–893. <https://doi.org/10.1503/cmaj.140355> [PubMed] [Google Scholar]

33. Broughton, B. R., Reutens, D. C., & Sobey, C. G. (2009). Apoptotic mechanisms after cerebral ischemia. Stroke, 40(5), e331–e339. <https://doi.org/10.1161/STROKEAHA.108.531632> [PubMed] [Google Scholar]

34. Woodruff, T. M., Thundiyil, J., Tang, S. C., Sobey, C. G., Taylor, S. M., & Arumugam, T. V. (2011). Pathophysiology, treatment, and animal and cellular models of human ischemic stroke. Molecular neurodegeneration, 6(1), 11. <https://doi.org/10.1186/1750-1326-6-11> [PubMed] [Google Scholar]

35. Gelderblom, M., Leypoldt, F., Steinbach, K., Behrens, D., Choe, C. U., Siler, D. A., Arumugam, T. V., Orthey, E., Gerloff, C., Tolosa, E., & Magnus, T. (2009). Temporal and spatial dynamics of cerebral immune cell accumulation in stroke. Stroke, 40(5), 1849–1857. <https://doi.org/10.1161/STROKEAHA.108.534503> [PubMed] [Google Scholar]

36. Suh, S. W., Shin, B. S., Ma, H., Van Hoecke, M., Brennan, A. M., Yenari, M. A., & Swanson, R. A. (2008). Glucose and NADPH oxidase drive neuronal superoxide formation in stroke. Annals of neurology, 64(6), 654–663. <https://doi.org/10.1002/ana.21511> [PubMed] [Google Scholar]

37. Qureshi, A. I., Ali, Z., Suri, M. F., Shuaib, A., Baker, G., Todd, K., Guterman, L. R., & Hopkins, L. N. (2003). Extracellular glutamate and other amino acids in experimental intracerebral hemorrhage: an in vivo microdialysis study. Critical care medicine, 31(5), 1482–1489. <https://doi.org/10.1097/01.CCM.0000063047.63862.99> [PubMed] [Google Scholar]

38. Wang, J., Fields, J., Zhao, C., Langer, J., Thimmulappa, R. K., Kensler, T. W., Yamamoto, M., Biswal, S., & Doré, S. (2007). Role of Nrf2 in protection against intracerebral hemorrhage injury in mice. Free radical biology & medicine, 43(3), 408–414. <https://doi.org/10.1016/j.freeradbiomed.2007.04.020> [PubMed] [Google Scholar]

39. Flaherty, M. L., Woo, D., Haverbusch, M., Sekar, P., Khoury, J., Sauerbeck, L., Moomaw, C. J., Schneider, A., Kissela, B., Kleindorfer, D., & Broderick, J. P. (2005). Racial variations in

A Review on Post-Stroke Management

location and risk of intracerebral hemorrhage. *Stroke*, 36(5), 934–937. <https://doi.org/10.1161/01.STR.0000160756.72109.95> [PubMed] [Google Scholar]

40. Testai, F. D., & Aiyagari, V. (2008). Acute hemorrhagic stroke pathophysiology and medical interventions: blood pressure control, management of anticoagulant-associated brain hemorrhage and general management principles. *Neurologic clinics*, 26(4), 963–ix. <https://doi.org/10.1016/j.ncl.2008.06.001> [PubMed] [Google Scholar]

41. Aronowski, J., & Zhao, X. (2011). Molecular pathophysiology of cerebral hemorrhage: secondary brain injury. *Stroke*, 42(6), 1781–1786. <https://doi.org/10.1161/STROKEAHA.110.596718> [PubMed] [Google Scholar]

42. WHO (2010), "Republic of Ghana, Standard treatment guidelines", Ministry of health, 6th edition,

43. Sarikaya H, Ferro J, Arnold M: Stroke Prevention - Medical and Lifestyle Measures. *Eur Neurol* 2015;73:150-157. doi: 10.1159/000367652

44. Slark, J., Bentley, P., Majeed, A., & Sharma, P. (2012). Awareness of Stroke Symptomatology and Cardiovascular Risk Factors Amongst Stroke Survivors. *Journal of Stroke and Cerebrovascular Diseases*, 21(5), 358-362. <https://doi.org/10.1016/j.jstrokecerebrovasdis.2010.09.010> [Google Scholar]

45. Kuriakose, D., & Xiao, Z. (2020). Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. *International Journal of Molecular Sciences*, 21(20). <https://doi.org/10.3390/ijms21207609> [PubMed] [Google Scholar]

46. Planjar-Prvan M. (2010). Nefarmakoloske metode prevencije mozdanog udara [Non-pharmacological methods of stroke prevention]. *Acta medica Croatica : casopis Hrvatske akademije medicinskih znanosti*, 64(1), 3–8. [PubMed] [Google Scholar]

47. <https://www.flintrehab.com/exercise-after-stroke-guidelines/#:~:text=Experts%20recommend%20aerobic%20exercise%20after,hospital%20stay%20and%20days%20thereafter.>

48. <https://www.cdc.gov/stroke/prevention.htm>

A Review on Post-Stroke Management

49. <https://www.flintrehab.com/exercise-after-stroke-guidelines/#:~:text=Experts%20recommend%20aerobic%20exercise%20after,hospital%20stay%20and%20days%20thereafter.>
50. Circulation: “Physical Activity and Exercise Recommendations for Stroke Survivors.”
51. <https://www.webmd.com/stroke/features/how-to-exercise-after-stroke>
52. Stroke Association: “Exercising after stroke.”
53. Stroke Foundation: “Mobility and exercise after stroke fact sheet.”
54. <https://www.webmd.com/stroke/features/how-to-exercise-after-stroke>
55. Victor M: Neurologic aspects of alcoholism, in Feldman RG (ed): Neurology: The Physician's Guide. New York, ThiemeStratton, Inc, 1984, pp 178-190
56. Hillbom, M., & Kaste, M. (1981). Ethanol intoxication: a risk factor for ischemic brain infarction in adolescents and young adults. Stroke, 12(4), 422–425. <https://doi.org/10.1161/01.str.12.4.422> [PubMed] [Google Scholar]
57. <https://www.india.com/health/these-7-lifestyle-are-increasing-your-risk-of-brain-stroke-world-stroke-day-5074535/>
58. Stokes G. S. (1982). Hypertension and alcohol: is ther a link?. Journal of chronic diseases, 35(10), 759–762. [https://doi.org/10.1016/0021-9681\(82\)90086-8](https://doi.org/10.1016/0021-9681(82)90086-8) [PubMed] [Google Scholar]
59. Kagan, A., Yano, K., Rhoads, G. G., & McGee, D. L. (1981). Alcohol and cardiovascular disease: the Hawaiian experience. Circulation, 64(3 Pt 2), III–31. [PubMed] [Google Scholar]
60. Walbran, B. B., Nelson, J. S., & Taylor, J. R. (1981). Association of cerebral infarction and chronic alcoholism: an autopsy study. Alcoholism, clinical and experimental research, 5(4), 531–535. <https://doi.org/10.1111/j.1530-0277.1981.tb05355.x> [PubMed] [Google Scholar]
61. Kozararevic, D., McGee, D., Vojvodic, N., Racic, Z., Dawber, T., Gordon, T., & Zukel, W. (1980). Frequency of alcohol consumption and morbidity and mortality: The Yugoslavia Cardiovascular Disease Study. Lancet (London, England), 1(8169), 613–616. [https://doi.org/10.1016/s0140-6736\(80\)91116-2](https://doi.org/10.1016/s0140-6736(80)91116-2) [PubMed] [Google Scholar]

A Review on Post-Stroke Management

62. Hillbom, M., & Kaste, M. (1978). Does ethanol intoxication promote brain infarction in young adults?. *Lancet* (London, England), 2(8101), 1181–1183. [https://doi.org/10.1016/s0140-6736\(78\)92160-8](https://doi.org/10.1016/s0140-6736(78)92160-8) [PubMed] [Google Scholar]
63. Hillbom, M., & Kaste, M. (1981). Ethanol intoxication: a risk factor for ischemic brain infarction in adolescents and young adults. *Stroke*, 12(4), 422–425. <https://doi.org/10.1161/01.str.12.4.422> [PubMed] [Google Scholar]
64. Hillbom, M., & Kaste, M. (1983). Ethanol intoxication: a risk factor for ischemic brain infarction. *Stroke*, 14(5), 694–699. <https://doi.org/10.1161/01.str.14.5.694> [PubMed] [Google Scholar]
65. Hillbom, M., & Kaste, M. (1981). Does alcohol intoxication precipitate aneurysmal subarachnoid haemorrhage?. *Journal of neurology, neurosurgery, and psychiatry*, 44(6), 523–526. <https://doi.org/10.1136/jnnp.44.6.523> [PubMed] [Google Scholar]
66. <https://strokefoundation.org.au/about-stroke/prevent-stroke/alcohol#:~:text=If%20you%20have%20had%20a,if%20you%20are%20taking%20warfarin.>
67. <https://www.hindustantimes.com/lifestyle/health/world-stroke-day-5-everyday-habits-that-could-increase-risk-of-stroke-101635479208269.html>
68. <https://www.india.com/health/these-7-lifestyle-are-increasing-your-risk-of-brain-stroke-world-stroke-day-5074535/>
69. <https://strokefoundation.org.au/about-stroke/prevent-stroke/smoking#:~:text=After%20an%20initial%20stroke%2C%20continued,need%20to%20stop%20smoking%20immediately.>
70. Newsom, J. T., Huguet, N., McCarthy, M. J., Ramage-Morin, P., Kaplan, M. S., Bernier, J., McFarland, B. H., & Oderkirk, J. (2012). Health behavior change following chronic illness in middle and later life. *The journals of gerontology. Series B, Psychological sciences and social sciences*, 67(3), 279–288. <https://doi.org/10.1093/geronb/gbr103> [PubMed] [Google Scholar]
71. Kammersgaard, Lars & Olsen, Tom. (2006). Cardiovascular Risk Factors and 5-Year Mortality in the Copenhagen Stroke Study. *Cerebrovascular diseases* (Basel, Switzerland). 21.

A Review on Post-Stroke Management

187-93. 10.1159/000090531.

72. Hankey, G. J., & Warlow, C. P. (1999). Treatment and secondary prevention of stroke: evidence, costs, and effects on individuals and populations. *Lancet* (London, England), 354(9188), 1457–1463. [https://doi.org/10.1016/S0140-6736\(99\)04407-4](https://doi.org/10.1016/S0140-6736(99)04407-4) [PubMed] [Google Scholar]

73. Redfern, J., McKevitt, C., Dundas, R., Rudd, A. G., & Wolfe, C. D. (2000). Behavioral risk factor prevalence and lifestyle change after stroke: a prospective study. *Stroke*, 31(8), 1877–1881. <https://doi.org/10.1161/01.str.31.8.1877> [PubMed] [Google Scholar]

74. Heart Disease and Stroke Statistics-2022 Update: A Report From the American Heart Association (Nutrition, Chapter 5)

75. <https://www.neuroolutions.com/post/post-stroke-nutrition-and-how-it-improves-stroke-recovery>

76. Kaluza, J., Wolk, A., & Larsson, S. C. (2012). Red meat consumption and risk of stroke: a meta-analysis of prospective studies. *Stroke*, 43(10), 2556–2560. <https://doi.org/10.1161/STROKEAHA.112.663286> [PubMed] [Google Scholar]

77. Lescinsky, H., Afshin, A., Ashbaugh, C., Bisignano, C., Brauer, M., Ferrara, G., Hay, S. I., He, J., Iannucci, V., Marczak, L. B., McLaughlin, S. A., Mullany, E. C., Parent, M. C., Serfes, A. L., Sorensen, R. J. D., Aravkin, A. Y., Zheng, P., & Murray, C. J. L. (2022). Health effects associated with consumption of unprocessed red meat: a Burden of Proof study. *Nature medicine*, 28(10), 2075–2082. <https://doi.org/10.1038/s41591-022-01968-z> [PubMed] [Google Scholar]

78. Tsao, C. W., Aday, A. W., Almarzooq, Z. I., Alonso, A., Beaton, A. Z., Bittencourt, M. S., Boehme, A. K., Buxton, A. E., Carson, A. P., Commodore-Mensah, Y., Elkind, M. S. V., Evenson, K. R., Eze-Nliam, C., Ferguson, J. F., Generoso, G., Ho, J. E., Kalani, R., Khan, S. S., Kissela, B. M., Knutson, K. L., ... Martin, S. S. (2022). Heart Disease and Stroke Statistics-2022 Update: A Report From the American Heart Association. *Circulation*, 145(8), e153–e639. <https://doi.org/10.1161/CIR.0000000000001052> [PubMed] [Google Scholar]

79. Spence J. D. (2018). Diet for stroke prevention. *Stroke and vascular neurology*, 3(2), 44–50.

A Review on Post-Stroke Management

<https://doi.org/10.1136/svn-2017-000130> [PubMed] [Google Scholar]

80. Cornelis, Marilyn & El-Sohemy, Ahmed & Kabagambe, Edmond & Campos, Hannia. (2006). Coffee, CYP1A2 Genotype, and Risk of Myocardial Infarction. *JAMA : the journal of the American Medical Association*. 295. 1135-41. 10.1001/jama.295.10.1135. [PubMed] [Google Scholar]

81. Poole, R., Kennedy, O. J., Roderick, P., Fallowfield, J. A., Hayes, P. C., & Parkes, J. (2017). Coffee consumption and health: umbrella review of meta-analyses of multiple health outcomes. *BMJ (Clinical research ed.)*, 359, j5024. <https://doi.org/10.1136/bmj.j5024> [PubMed] [Google Scholar]

82. Onakpoya, I., Spencer, E., Heneghan, C., & Thompson, M. (2014). The effect of green tea on blood pressure and lipid profile: a systematic review and meta-analysis of randomized clinical trials. *Nutrition, metabolism, and cardiovascular diseases : NMCD*, 24(8), 823–836. <https://doi.org/10.1016/j.numecd.2014.01.016> [PubMed] [Google Scholar]

83. Park, C. S., Kim, W., Woo, J. S., Ha, S. J., Kang, W. Y., Hwang, S. H., Park, Y. W., Kim, Y. S., Ahn, Y. K., Jeong, M. H., & Kim, W. (2010). Green tea consumption improves endothelial function but not circulating endothelial progenitor cells in patients with chronic renal failure. *International journal of cardiology*, 145(2), 261–262. <https://doi.org/10.1016/j.ijcard.2009.09.471> [PubMed] [Google Scholar]

84. Spence J. D. (2018). Diet for stroke prevention. *Stroke and vascular neurology*, 3(2), 44–50. <https://doi.org/10.1136/svn-2017-000130> [PubMed] [Google Scholar]

85. Spence JD. How to prevent your stroke. Nashville, TN: Vanderbilt University Press, 2006.

86. <https://strokefoundation.org.au/what-we-do/for-survivors-and-carers/after-stroke-factsheets/depression-and-anxiety-after-stroke-fact-sheet#:~:text=Depression%20and%20anxiety%20are%20common,or%20sleep%20more%20than%20usual>.

87. Lenzi, G. L., Altieri, M., & Maestrini, I. (2008). Post-stroke depression. *Revue neurologique*, 164(10), 837–840. <https://doi.org/10.1016/j.neurol.2008.07.010> [PubMed] [Google Scholar]

A Review on Post-Stroke Management

88. Alajbegovic, A., Djelilovic-Vranic, J., Nakicevic, A., Todorovic, L., & Tiric-Campara, M. (2014). Post stroke depression. *Medical archives (Sarajevo, Bosnia and Herzegovina)*, 68(1), 47–50. <https://doi.org/10.5455/medarh.2014.68.47-50> [PubMed] [Google Scholar]
89. Medeiros, G. C., Roy, D., Kontos, N., & Beach, S. R. (2020). Post-stroke depression: A 2020 updated review. *General hospital psychiatry*, 66, 70–80. <https://doi.org/10.1016/j.genhosppsy.2020.06.011> [PubMed] [Google Scholar]
90. Wright, F., Wu, S., Chun, H. Y., & Mead, G. (2017). Factors Associated with Poststroke Anxiety: A Systematic Review and Meta-Analysis. *Stroke research and treatment*, 2017, 2124743. <https://doi.org/10.1155/2017/2124743> [PubMed] [Google Scholar]
91. Rafsten, L., Danielsson, A., & Sunnerhagen, K. S. (2018). Anxiety after stroke: A systematic review and meta-analysis. *Journal of rehabilitation medicine*, 50(9), 769–778. <https://doi.org/10.2340/16501977-2384> [PubMed] [Google Scholar]
92. Ramos-Perdigués, S., Mané-Santacana, A., & Pintor-Pérez, L. (2015). Revisión sistemática de la prevalencia y factores asociados a la ira tras un ictus [Prevalence and associated factors of anger post stroke: a systematic review]. *Revista de neurologia*, 60(11), 481–489. [PubMed] [Google Scholar]
93. Santos, A. C., & Ferro, J. M. (2017). The impact of anger in adherence to treatment and beliefs about disease 1 year after stroke. *Journal of neurology*, 264(9), 1929–1938. <https://doi.org/10.1007/s00415-017-8577-x> [PubMed] [Google Scholar]
94. <https://www.flintrehab.com/anger-after-stroke/>
95. <https://strokefoundation.org.au/what-we-do/for-survivors-and-carers/after-stroke-factsheets/depression-and-anxiety-after-stroke-fact-sheet#:~:text=Depression%20and%20anxiety%20are%20common,or%20sleep%20more%20than%20usual.>
96. Eum, Y., & Yim, J. (2015). Literature and art therapy in post-stroke psychological disorders. *The Tohoku journal of experimental medicine*, 235(1), 17–23. <https://doi.org/10.1620/tjem.235.17> [PubMed] [Google Scholar]
97. Tsouna-Hadjis, E., Vemmos, K. N., Zakopoulos, N., & Stamatelopoulos, S. (2000). First-

A Review on Post-Stroke Management

stroke recovery process: the role of family social support. Archives of physical medicine and rehabilitation, 81(7), 881–887. <https://doi.org/10.1053/apmr.2000.4435> [PubMed] [Google Scholar]

98. Piechowski-Józwiak B, Bogousslavsky J. Pharmacotherapy of stroke. Eur Neurol Dis. 2006;1:52-62.

99. <https://www.nhs.uk/conditions/stroke/>

100. <https://www.webmd.com/stroke/meds-after-stroke>

101. Shari N. Allen, P. D. A. P. of P. P. P. C. of O. M. S. of P. S. (2012, February 17). Pharmacologic management of stroke. U.S. Pharmacist – The Leading Journal in Pharmacy. Retrieved April 10, 2023, from <https://www.uspharmacist.com/article/pharmacologic-management-of-stroke>

102. <https://www.heartandstroke.ca/stroke/treatments/medications#:~:text=How%20stroke%20medications%20work,high%20blood%20pressure%20and%20cholesterol.>

103. Arnett, D. K., Blumenthal, R. S., Albert, M. A., Buroker, A. B., Goldberger, Z. D., Hahn, E. J., Himmelfarb, C. D., Khera, A., Lloyd-Jones, D., McEvoy, J. W., Michos, E. D., Miedema, M. D., Muñoz, D., Smith, S. C., Jr, Virani, S. S., Williams, K. A., Sr, Yeboah, J., & Ziaeian, B. (2019). 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation, 140(11), e596–e646. <https://doi.org/10.1161/CIR.0000000000000678> [PubMed] [Google Scholar]

104. Zivin, J. A., Fisher, M., DeGirolami, U., Hemenway, C. C., & Stashak, J. A. (1985). Tissue plasminogen activator reduces neurological damage after cerebral embolism. Science (New York, N.Y.), 230(4731), 1289–1292. <https://doi.org/10.1126/science.3934754> [PubMed] [Google Scholar]

105. Penar, P. L., & Greer, C. A. (1987). The effect of intravenous tissue-type plasminogen activator in a rat model of embolic cerebral ischemia. The Yale journal of biology and medicine, 60(3), 233–243. [PubMed] [Google Scholar]

106. Zivin, J. A., Lyden, P. D., DeGirolami, U., Kochhar, A., Mazzearella, V., Hemenway, C. C., & Johnston, P. (1988). Tissue plasminogen activator. Reduction of neurologic damage after

A Review on Post-Stroke Management

- experimental embolic stroke. Archives of neurology, 45(4), 387–391.
<https://doi.org/10.1001/archneur.1988.00520280033012> [PubMed] [Google Scholar]
107. Phillips, D. A., Fisher, M., Smith, T. W., & Davis, M. A. (1988). The safety and angiographic efficacy of tissue plasminogen activator in a cerebral embolization model. *Annals of neurology*, 23(4), 391–394. <https://doi.org/10.1002/ana.410230414> [PubMed] [Google Scholar]
108. Lyden, P. D., Zivin, J. A., Clark, W. A., Madden, K., Sasse, K. C., Mazzearella, V. A., Terry, R. D., & Press, G. A. (1989). Tissue plasminogen activator-mediated thrombolysis of cerebral emboli and its effect on hemorrhagic infarction in rabbits. *Neurology*, 39(5), 703–708. <https://doi.org/10.1212/wnl.39.5.703> [PubMed] [Google Scholar]
109. Overgaard, K., Sereghy, T., Boysen, G., Pedersen, H., & Diemer, N. H. (1992). Reduction of infarct volume and mortality by thrombolysis in a rat embolic stroke model. *Stroke*, 23(8), 1167–1174. <https://doi.org/10.1161/01.str.23.8.1167> [PubMed] [Google Scholar]
110. <https://www.nhs.uk/conditions/stroke/treatment/#:~:text=Most%20people%20will%20be%20given,such%20as%20clopidogrel%20and%20dipyridamole.>
111. Ischemic stroke treatments. (2018). strokeassociation.org/en/about-stroke/treatment/ischemic-stroke-treatment
112. <https://www.heartandstroke.ca/stroke/treatments/medications/#:~:text=How%20stroke%20medications%20work,high%20blood%20pressure%20and%20cholesterol.>
113. Shahpouri, M. M., Mousavi, S., Khorvash, F., Mousavi, S. M., & Hoseini, T. (2012). Anticoagulant therapy for ischemic stroke: A review of literature. *Journal of research in medical sciences : the official journal of Isfahan University of Medical Sciences*, 17(4), 396–401. [PubMed] [Google Scholar]
114. <https://www.nhs.uk/conditions/stroke/treatment/#:~:text=Most%20people%20will%20be%20given,such%20as%20clopidogrel%20and%20dipyridamole.>
115. Stroke: "Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack," "Guidelines for Adult Stroke Rehabilitation and Recovery, A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association."
116. <https://www.healthline.com/health/stroke/drugs>

A Review on Post-Stroke Management

117. Minhas, J. S., Chithiramohan, T., Wang, X., Barnes, S. C., Clough, R. H., Kadicheeni, M., Beishon, L. C., & Robinson, T. (2022). Oral antiplatelet therapy for acute ischaemic stroke. The Cochrane database of systematic reviews, 1(1), CD000029. <https://doi.org/10.1002/14651858.CD000029.pub4> [PubMed] [Google Scholar]
118. <https://www.heartandstroke.ca/stroke/treatments/medications#:~:text=How%20stroke%20medications%20work,high%20blood%20pressure%20and%20cholesterol.>
119. <https://www.nhs.uk/conditions/stroke/treatment/#:~:text=Most%20people%20will%20be%20given,such%20as%20clopidogrel%20and%20dipyridamole.>
120. Adams, H. P., Jr, del Zoppo, G., Alberts, M. J., Bhatt, D. L., Brass, L., Furlan, A., Grubb, R. L., Higashida, R. T., Jauch, E. C., Kidwell, C., Lyden, P. D., Morgenstern, L. B., Qureshi, A. I., Rosenwasser, R. H., Scott, P. A., Wijedicks, E. F., American Heart Association/American Stroke Association Stroke Council, American Heart Association/American Stroke Association Clinical Cardiology Council, American Heart Association/American Stroke Association Cardiovascular Radiology and Intervention Council, Atherosclerotic Peripheral Vascular Disease Working Group, ... Quality of Care Outcomes in Research Interdisciplinary Working Group (2007). Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Circulation*, 115(20), e478–e534. <https://doi.org/10.1161/CIRCULATIONAHA.107.181486> [PubMed] [Google Scholar]
121. Hirsh, J., Guyatt, G., Albers, G. W., Harrington, R., & Schünemann, H. J. (2008). Antithrombotic and thrombolytic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest*, 133(6 Suppl), 110S–112S. <https://doi.org/10.1378/chest.08-0652> [PubMed] [Google Scholar]
122. <https://www.nhs.uk/conditions/stroke/treatment/#:~:text=Most%20people%20will%20be%20given,such%20as%20clopidogrel%20and%20dipyridamole.>
123. <https://www.healthline.com/health/stroke/drugs#antiplatelets>

A Review on Post-Stroke Management

124. Pundi, K., Baykaner, T., True Hills, M., Lin, B., Morin, D. P., Sears, S. F., Wang, P. J., & Stafford, R. S. (2021). Blood Thinners for Atrial Fibrillation Stroke Prevention. *Circulation. Arrhythmia and electrophysiology*, 14(6), e009389. <https://doi.org/10.1161/CIRCEP.120.009389> [PubMed] [Google Scholar]
125. Goldstein, L. B., Adams, R., Alberts, M. J., Appel, L. J., Brass, L. M., Bushnell, C. D., Culebras, A., Degra, T. J., Gorelick, P. B., Guyton, J. R., Hart, R. G., Howard, G., Kelly-Hayes, M., Nixon, J. V., Sacco, R. L., American Heart Association/American Stroke Association Stroke Council, Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group, Cardiovascular Nursing Council, Clinical Cardiology Council, Nutrition, Physical Activity, and Metabolism Council, ... American Academy of Neurology (2006). Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council: cosponsored by the Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; and the Quality of Care and Outcomes Research Interdisciplinary Working Group: the American Academy of Neurology affirms the value of this guideline. *Stroke*, 37(6), 1583–1633. <https://doi.org/10.1161/01.STR.0000223048.70103.F1> [PubMed] [Google Scholar]
126. Marsh, J. D., & Keyrouz, S. G. (2010). Stroke prevention and treatment. *Journal of the American College of Cardiology*, 56(9), 683–691. <https://doi.org/10.1016/j.jacc.2009.12.072> [PubMed] [Google Scholar]
127. ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (2002). Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic: The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). *JAMA*, 288(23), 2981–2997. <https://doi.org/10.1001/jama.288.23.2981> [PubMed] [Google Scholar]
128. Neal, B., MacMahon, S., Chapman, N., & Blood Pressure Lowering Treatment Trialists' Collaboration (2000). Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: results of prospectively designed overviews of randomised trials. Blood Pressure Lowering Treatment Trialists' Collaboration. *Lancet (London, England)*, 356(9246),

A Review on Post-Stroke Management

- 1955–1964. [https://doi.org/10.1016/s0140-6736\(00\)03307-9](https://doi.org/10.1016/s0140-6736(00)03307-9) [PubMed] [Google Scholar]
129. Mukete, B. N., Cassidy, M., Ferdinand, K. C., & Le Jemtel, T. H. (2015). Long-Term Anti-Hypertensive Therapy and Stroke Prevention: A Meta-Analysis. *American journal of cardiovascular drugs : drugs, devices, and other interventions*, 15(4), 243–257. <https://doi.org/10.1007/s40256-015-0129-0> [PubMed] [Google Scholar]
130. Tanahashi N. (2016). *Nihon rinsho. Japanese journal of clinical medicine*, 74(4), 681–689. [PubMed] [Google Scholar]
131. Zonneveld, T. P., Richard, E., Vergouwen, M. D., Nederkoorn, P. J., de Haan, R., Roos, Y. B., & Kruyt, N. D. (2018). Blood pressure-lowering treatment for preventing recurrent stroke, major vascular events, and dementia in patients with a history of stroke or transient ischaemic attack. *The Cochrane database of systematic reviews*, 7(7), CD007858. <https://doi.org/10.1002/14651858.CD007858.pub2> [PubMed] [Google Scholar]
132. Stroke: "Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack," "Guidelines for Adult Stroke Rehabilitation and Recovery, A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association."
133. <https://www.healthline.com/health/stroke/drugs>
134. Furie, K. L., Kasner, S. E., Adams, R. J., Albers, G. W., Bush, R. L., Fagan, S. C., Halperin, J. L., Johnston, S. C., Katzan, I., Kernan, W. N., Mitchell, P. H., Ovbiagele, B., Palesch, Y. Y., Sacco, R. L., Schwamm, L. H., Wassertheil-Smoller, S., Turan, T. N., Wentworth, D., & American Heart Association Stroke Council, Council on Cardiovascular Nursing, Council on Clinical Cardiology, and Interdisciplinary Council on Quality of Care and Outcomes Research (2011). Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the american heart association/american stroke association. *Stroke*, 42(1), 227–276. <https://doi.org/10.1161/STR.0b013e3181f7d043> [PubMed] [Google Scholar]
135. Amarenco, P., Bogousslavsky, J., Callahan, A., 3rd, Goldstein, L. B., Hennerici, M., Rudolph, A. E., Sillesen, H., Simunovic, L., Szarek, M., Welch, K. M., Zivin, J. A., & Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) Investigators (2006).

A Review on Post-Stroke Management

High-dose atorvastatin after stroke or transient ischemic attack. The New England journal of medicine, 355(6), 549–559. <https://doi.org/10.1056/NEJMoa061894> [PubMed] [Google Scholar]

136. <https://beaumonteh.com/first-aid-for-stroke/>
137. <https://www.cdc.gov/stroke/about.htm>
138. <https://calgaryguide.ucalgary.ca/stroke-pathogenesis/>
139. Kuriakose, D., & Xiao, Z. (2020). Pathophysiology and Treatment of Stroke: Present Status and Future Perspectives. International journal of molecular sciences, 21(20), 7609. <https://doi.org/10.3390/ijms21207609> [PubMed] [Google Scholar]
140. <https://news.lau.edu.lb/2021/world-stroke-day-2021-act-fast-treat-and-prevent.php>
141. <https://www.saebo.com/blog/why-you-should-exercise-after-stroke/>
142. <https://www.shutterstock.com/image-vector/infographics-stroke-dietary-changes-after-vector-735171775>
143. Kapil, N., Datta, Y. H., Alakbarova, N., Bershad, E., Selim, M., Liebeskind, D. S., Bachour, O., Rao, G. H. R., & Divani, A. A. (2017). Antiplatelet and Anticoagulant Therapies for Prevention of Ischemic Stroke. Clinical and applied thrombosis/hemostasis : official journal of the International Academy of Clinical and Applied Thrombosis/Hemostasis, 23(4), 301–318. <https://doi.org/10.1177/1076029616660762> [PubMed] [Google Scholar]