Cerebral vascular accidents and therapeutic treatments

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CEREBRAL VASCULAR ACCIDENTS
AND THERAPEUTIC TREATMENTS

by ALLISON L. HARTMAN

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INTRODUCTION

A cerebral vascular accident (CVA) is commonly referred to as a stroke. Stroke is the third leading cause of death and the number one cause of serious disability in the United States. Approximately 550,000 people suffer a stroke each year; 150,000 of these result in death. Each stroke is unique. After a stroke, some victims will suffer severe deficits and/or permanent disability for the remainder of their lives. Yet other victims may suffer only mild, temporary deficits and may recover completely. Currently over three million Americans are living with the effects of stroke. Most of these individuals have been successfully rehabilitated through therapy, medical treatment, and lifestyle changes.

I have been exposed to stroke patients and their families in a medical setting throughout the past three years. It has been my observation that most people do not fully understand stroke, and they definitely do not comprehend their own ability to prevent stroke. Therefore, the purpose of this paper is to provide thorough information about stroke and its consequences, and to educate the reader about preventative measures which can decrease stroke risk. Every individual has some degree of stroke risk; however, many risk factors can be eliminated by mere changes in lifestyle. These conscious behaviors, in addition to major medical and diagnostic breakthroughs, provide hope for stroke prevention and treatment in the future.

As I enter the field of physical therapy, I will reference this paper often. I plan to share it with stroke survivors,
their families, and others affected by stroke so that it can serve as an educational tool for those frightened individuals with unending questions. However, it is my hope that this paper not only educates stroke survivors and their families, but also health conscious individuals who desire to reduce their risk of stroke and improve their overall quality of life.
BACKGROUND OF BRAIN ANATOMY & FUNCTION

Stroke occurs in the brain, the most complex organ of the human body. The brain weighs only an average of three pounds, but consists of billions of neurons. Neurons are the basic cellular unit of the nervous system; they are responsible for all communication links within the brain and links throughout the entire human body. For these responsibilities, the brain requires an immense amount of energy. Unlike other body organs, the brain is unable to store energy; therefore, the brain relies on a steady supply of oxygen and other nutrients. These necessities are continually provided by blood which is delivered from the heart to the brain via arteries. This blood flow to the brain is so vital that approximately one-fourth of all blood pumped by the heart reaches the brain (Caplan, Dyken, and Easton 3). A disruption of the blood supply to some part of the brain causes a stroke, and the nerve cells in the affected area of the brain are unable to function.

The brain is extremely specialized. Because each particular area of the brain has specific responsibilities, experts can determine the regions of the brain damaged from stroke by evaluating the patient’s symptoms. In order to comprehend how and why a stroke affects the various parts of the body, an understanding of the organization of the brain and its functions is necessary.

The brain and the spinal cord compose the central nervous system. The peripheral nervous system is composed of nerve
fibers which carry information between the central nervous system and the rest of the body. Thus, nerve cells, or neurons, are responsible for communication. Afferent neurons (sensory nerve fibers) carry information to the brain from the body. Efferent neurons (motor nerve fibers) send information from the brain to other parts of the body. In addition, the brain controls endocrine glands which release hormones into the bloodstream causing chemical changes in the body. It is obvious that in some way the brain controls all activity in the human body.

The brain can be divided into several regions. Although these regions interact, each region has its own responsibilities.

FIGURE 1: Structures of the Brain from Neurologic Disorders by Chipps, Clanin, and Campbell.
FOREBRAIN

TELENCEPHALON (CEREBRUM)

One portion of the forebrain is the telencephalon, more commonly referred to as the cerebrum. The cerebrum is the largest part of the brain and represents approximately two thirds of the brain's total weight (Sessler 6). The cerebrum is the most developed area of the brain, thus it is the center for the highest functions, such as: mental activities and thought processes, emotions, sensory activities, motor activities and voluntary acts, memory, learning, and other association activities (Sessler 8).

The cerebrum is divided into two similar halves called cerebral hemispheres. These hemispheres govern the opposite sides of the body. For example, the right hemisphere receives impulses from and sends impulses to the left side of the body, and the left hemisphere receives impulses from and sends impulses to the right side of the body. The right and left hemispheres are connected by the corpus callosum, a network of nerve fibers which promote communication between the two hemispheres. The hemispheres have distinguishing characteristics. The right hemisphere is often associated with visual and spatial awareness, the imagination, memory and recognition, musical and artistic abilities, and negative emotions (Caplan, Dyken, and Easton 10). The left hemisphere is related to language and verbal skills, math and logic and analytical skills, thought processes and problem solving, and positive emotions (Caplan, Dyken, and
Easton 11). In most individuals one hemisphere is more developed, it is referred to as the dominant hemisphere. The dominant hemisphere is most often the hemisphere responsible for organizing both written and spoken language. In 97 percent of the population the language center is located in the left hemisphere (Sessler 7); therefore, the majority of the population has a dominant left hemisphere.

The cerebrum is made of two layers. The inner layer is called the white matter of the brain, it is comprised of a network of axons which permit communication with all parts of the brain. (Axons are the portion of the neuron which sends impulses.) The outer layer of the cerebrum is the cerebral cortex, which is commonly referred to as gray matter. Gray matter is composed of neuron cell bodies, it has processing and integrating responsibilities which are unique to humans.

The cerebral cortex is wrinkled and folded. The deepest folds form fissures, or deep grooves, which divide the cerebrum into four distinct areas referred to as lobes. The medial longitudinal fissure is a natural division which extends from front to back down the center of the brain separating the right and left hemispheres. The lateral fissure, sometimes called the Sylvian fissure, extends along the side of each hemisphere separating the temporal lobe from the above frontal and parietal lobes. The central fissure begins at the medial longitudinal fissure and extends down to the lateral fissure. This fissure separates the frontal lobe and the parietal lobe. The most
posterior portion of the cerebrum is the occipital lobe.

Each lobe of the cortex is responsible for different functions. Because the cerebrum is a common site for stroke damage, it is important to understand the functions specific to each lobe.

**FIGURE 2: Divisions of the Cerebrum**
from *Family Guide* by Caplan, Dyken, and Easton.

**FRONTAL LOBE:** The frontal lobe lies in front of the central fissure and can be divided into two regions, the precentral region and the prefrontal region (Walsh 50).

The precentral region is the area of the frontal lobe which concentrates on motor function, it contains three major portions: the precentral gyrus, the premotor cortex, and Broca's area (Walsh 50). The precentral gyrus is a bulge which lies just anterior to the central fissure. This area is known as the primary motor cortex because it is responsible for initiating
voluntary movements on the opposite side of the body (Liebman 4). The motor cortex extends from the top of the frontal lobe down to the lateral fissure, and its organization is represented by a motor homunculus (Figure 3). In the motor homunculus, neurons are organized upside down and distorted (Sherwood 120). Neurons responsible for lower body movement are located near the top of the motor cortex, neurons responsible for mid-body movement are located in the middle of the motor cortex, and neurons responsible for facial movement are found near the base of the motor cortex (Sherwood 120). In addition, the amount of space allotted for areas in the motor cortex is proportional to the precision and complexity of the motor skills (Sherwood 120). For example, the large amount of space in the motor cortex devoted to the fine muscles related to speech is magnified compared to the small area devoted to the trunk and lower extremities.

FIGURE 3: The Motor Homunculus from Human Physiology by Sherwood.
The premotor cortex lies just anterior to the precentral gyrus (motor cortex) and it is responsible for the planning of sequential movements. This area allows individuals to concentrate, plan, and accomplish complex tasks (Caplan, Dyken, and Easton 14).

Broca’s area is located on the inferior frontal gyrus of the dominant (left) hemisphere. Broca’s area controls the planning and programming of speech and it is closely associated with the motor areas of the cortex responsible for speech (Walsh 54).

The prefrontal region is the area of the frontal lobe which lies anterior to the premotor cortex; it is considered association cortex -- a higher, more integrative type of cortex. The prefrontal region is closely related to personality and behavior. Emotions, attitude, insight and abstract thinking, initiative, and inhibition are all generated from this area (Caplan, Dyken, and Easton 15).

PARietal Lobe: The parietal lobe lies directly behind the central fissure, it is responsible for receiving and processing sensory input such as temperature, pain, touch, and pressure (Sessler 7). This lobe also regulates proprioception, which is perception and awareness of body position. Although the hemispheres of the parietal lobe possess specific functions, the lobe can be divided into two main regions, the somatosensory cortex and the posterior parietal cortex (Sherwood 119).

The somatosensory cortex consists of the postcentral gyrus, a bulge which lies just posterior to the central fissure. The
postcentral gyrus actually receives sensory input from areas of the body. Similar to the precentral gyrus in the frontal lobe, the postcentral gyrus extends from the top of the parietal lobe down to the lateral fissure, and its organization can also be represented by a homunculus (Figure 4). Similar to the motor homunculus, the sensory homunculus is organized upside down. The allotted amount of somatosensory cortex for each body part depends on the degree of sensory perception associated with the specific body part (Sherwood 119). Therefore, the areas in the somatosensory cortex delegated to the face or to the hands are exaggerated in comparison to the smaller areas delegated to the lower body extremities. The somatosensory cortex in each hemisphere usually receives sensory input from the opposite side of the brain.

FIGURE 4: The Sensory Homunculus from *Human Physiology* by Sherwood.
Just behind the postcentral gyrus in the parietal lobe lies the area of the somatosensory cortex responsible for recognizing the sensory input. In this area, the current sensation is matched to a previous memory of a similar sensation (Walsh 51). Thus, through this organization and perception, the current sensation is recognized.

The posterior parietal lobe is considered the association area of the parietal lobe. The function of the posterior part of the parietal lobe is to link different sensations and to integrate somatosensory, visual, and auditory input (Walsh 51).

**TEMPORAL LOBE:** The temporal lobe is located on the sides of the head directly below the lateral fissure. Similar to the parietal lobe, the hemispheres of the temporal lobe have slightly different tasks. Despite this asymmetry, the temporal lobe can be divided into three regions: the auditory association cortex, the complex association area, and the limbic cortex (Walsh 53).

The auditory association cortex is the superior temporal gyrus (bulge), which is located directly below and parallel to the lateral fissure. The auditory association cortex is responsible for sensing sound and decoding language (Walsh 53). The posterior region of the superior temporal gyrus on the dominant hemisphere (usually left) is known as Wernicke’s area. This is where the actual decoding and language comprehension occurs (Sherwood 122).

The complex association area is composed of the middle and inferior temporal gyri. This area controls long term memory,
selection attention, and complex visual and auditory processing (Walsh 53).

The limbic cortex is located in the medial (inner) temporal lobe. It pertains to personality, emotional state, sexual state, and the creation of new memories (Caplan, Dyken, and Easton 19).

OCCIPITAL LOBE: The occipital lobe is located at the low, posterior portion of the cerebrum, it is responsible for the processing of visual input. The left hemisphere of the occipital lobe controls the right visual field in both eyes, and the right hemisphere of the occipital lobe controls the left visual field in both eyes.

Although the lobes of the cerebrum are quite complex and have many specific functions, they are all interconnected and dependent on each other. The lobes of the cerebrum communicate with each other and work together to affect the activities of the human body.

Diencephalon

The diencephalon is the second component of the forebrain, it is located deep in the interior of the forebrain between the cerebral hemispheres (Liebman 6). The diencephalon consists of the thalamus and the hypothalamus.

The thalamus is the main sensory relay center of the brain, it partially processes all sensory input and then directs it to the proper portion of the cortex (Sherwood 126). The
hypothalamus lies beneath the thalamus and is very small in size; however, it is a vital portion of the brain because it influences many basic survival behaviors and homeostatic functions. The hypothalamus receives input from the cerebral hemispheres and serves as a link to both the autonomic nervous system and the endocrine system (Walsh 45). Some functions of the hypothalamus include: control of body temperature, control of hunger and thirst and urine output, production of hormones and control of hormone secretion, control of emotional and behavioral patterns, and coordination of much activity of the autonomic nervous system (Sherwood 126).

**BRAINSTEM**

The brainstem consists of the midbrain, pons, and medulla; and it serves as a link between the spinal cord and the rest of the brain. In fact, all incoming sensory neurons and all outgoing efferent commands must pass through the brainstem (Sherwood 136).

The mesencephalon, or midbrain, is the smallest portion of the brainstem. Located just above the pons between the cerebrum and the cerebellum, the midbrain is composed of fibers which link the cerebral cortex to the many reflex centers in the brain (Sessler 9).

The pons is located near the center of the brain between the midbrain and the medulla. The pons connects the two halves of the cerebellum; and it serves as a bridge permitting
communication between the various parts of the brain (Sessler 9). The pons also is responsible for regulating state of consciousness.

The medulla, or myelencephalon, is the lowest section of the brain, it is located at the base of the skull where it eventually becomes continuous with the spinal cord. All impulses traveling from the cerebral cortex to the spinal cord must pass through the medulla (Caplan, Dyken, and Easton 6). The medulla is primarily responsible for regulating involuntary, life sustaining functions such as breathing, blood pressure, and heart rate (Liebman 6).

**CEREBELLUM**

The cerebellum is located under the back portion of the cerebrum and is attached to the posterior, upper portion of the brainstem. This is the second largest area of the brain (Sessler 8). The cerebellum is very folded and consists of two hemispheres which are linked by the pons (Sessler 8). (Together the pons and the cerebellum constitute a portion of the brain known as the metencephalon.) The function of the cerebellum is to maintain a sense of balance and coordination. The cerebellum synthesizes smooth movement, regulates muscle tone, and monitors the state of equilibrium.
Circulation of blood to the brain delivers the constant supply of oxygen and nutrients which the brain requires for survival. The delivery of blood to the brain is more vital than the delivery of blood to any other part of the body (Sessler 10). Therefore, the brain contains a built-in mechanism, called collateral circulation, which serves as a backup system assuring a constant supply of blood (Sessler 10). The flow of blood to the brain is carefully regulated by the body because any reduction of blood deprives brain cells of oxygen, known as ischemia, and causes damage or possibly cell death. If an artery becomes partially blocked, collateral circulation may allow blood to pass around the blocked area in order to supply the deprived region of the brain with blood (Liebman 74). This is possible because often when an artery has been damaged, smaller surrounding arteries dilate and take over the functions of the damaged artery; thus, compensating for missing blood flow (Sessler 11). This process permits cells temporarily deprived of oxygen to recover. Therefore, the availability of collateral circulation helps to determine the extent of damage caused by blockage.

If the deprived area is not quickly supplied with oxygen, the tissue area dies, this is called an infarct. The dead brain cells in this area are not able to function and will never be replaced (Caplan, Dyken, and Easton 23). The areas and functions of the body which are affected depends on the region of the brain
where infarction occurs.

Two large pairs of arteries from the aortic arch are the only suppliers of blood to the brain. These two pairs are the carotid arteries and the vertebral arteries. Both pairs branch into smaller arteries and tiny capillaries in order to continuously supply every portion of the brain with blood (Caplan, Dyken, and Easton 22).

FIGURE 5: Arteries of the Brain (ventral view) from Neurologic Disorders by Chipps, Clanin, and Campbell.
CAROTID ARTERIES

The carotid arteries run up the sides of the neck and branch into the external carotid arteries, which supply blood to the neck and the face, and the internal carotid arteries, which supply blood to the front part of the brain (Sessler 10). An interruption in blood flow to the internal carotid arteries may impair memory, muscle movements and the sensation of temperature, pain, and touch (Sessler 10).

The internal carotid arteries further divide into the anterior cerebral arteries and the middle cerebral arteries (Liebman 74). The anterior cerebral arteries supply blood to the medial surface of the cerebral hemispheres. The middle cerebral arteries are a frequent sight of stroke because these arteries deliver blood to the lateral surface of the cerebral hemispheres, affecting much of the cerebral cortex (Sessler 13). A decrease in blood delivered to the middle cerebral arteries may result in paralysis, numbness on the nondominant side of the body, possible speech problems, and possible vision impairments (Sessler 13).

VERTEBRAL ARTERIES

The vertebral arteries run up the spinal column and enter at the base of the brain. These arteries and their branches provide blood to the brainstem and the rear portion of the brain. The two vertebral arteries join to form the basilar artery (Liebman 74). Damage to the basilar artery creates difficult swallowing and speaking conditions, facial numbness, extremity weakness and
unsteadiness (Sessler 12).

Near the pons, the basilar artery bifurcates into the posterior cerebral arteries (Liebman 74). These arteries supply blood to the visual control center; thus, blockage of this artery causes visual disturbance. However, reading and memory problems may also result (Sessler 13).

THE CIRCLE OF WILLIS

At the base of the brain a ring is formed between the internal carotid arteries and the vertebral arteries, this ring is referred to as the Circle of Willis (Liebman 74). The Circle of Willis is a backup system which effectively demonstrates collateral circulation in the brain. If one artery is blocked, blood can flow around the blockage via the Circle of Willis to reach the deprived area of the brain (Sessler 13).

This general introduction to the organization of the brain and its functions should supply the background needed to understand the different types of strokes and how they occur. This information is also vital in comprehending how and why specific functions of the body are impaired after stroke.
THE PROCESS OF STROKE

A stroke is caused by an interruption of blood flow to the brain which occurs when an artery leading to the brain becomes blocked or ruptures. A stroke has recently been labeled by the National Stroke Association (NSA) as a "brain attack" (NSA, Stroke is a Brain Attack 1). During this brain attack, brain cells are deprived of vital supplies of oxygen and nutrients which are normally provided from the blood. This lack of oxygen is called ischemia, and leads to rapid cell death; the initial dead area of tissue is referred to as an infarct (Caplan, Dyken, and Easton 23).

However, damage is not limited to the immediate blood-deprived area where the stroke occurs. Neural damage spreads and affects a large surrounding area, called the penumbra, due to a process known as excitotoxicity (NSA, Brain at Risk 7). Excitotoxicity can be described as a "glutamate cascade," or an electro-chemical chain reaction which results in cell death (NSA, Darkness into Light" 11). This process consists of three distinct stages: induction, amplification, and expression.

The toxic release of glutamate is the cause of excitotoxicity, also known as secondary injury. Glutamate is an excitatory neurotransmitter which is normally released in small amounts from neurons as a means of chemical communication between brain cells (Sherwood 115). Induction: Triggered by oxygen deprivation, brain cells at the initial site of stroke are stimulated and release excessive amounts of glutamate (Zivin and
Choi 61). Amplification: Excess glutamate changes the cell’s electrical balance and opens calcium channels resulting in calcium overload within the affected cell (Zivin and Choi 61). This calcium build up causes the release of more glutamate, which binds with and overexcites surrounding neurons in the penumbra (Sherwood 115). Thus, as a chain reaction, this process continues to spread to areas around the initial site of oxygen deprivation. Expression: The abundance of calcium in excited cells activates enzymes which break down cellular components (Zivin and Choi 61). These degraded components give rise to oxygen free radicals which destroy the structure of the brain cell and continue further destruction of cellular constituents (NSA, "Darkness into Light" 11). Finally, the brain cell dies.

The "glutamate cascade" is a rather recent discovery and it is under much investigation by scientists and researchers. A clear understanding of this process will undoubtedly introduce new methods of stroke prevention and treatment.
TYPES OF STROKE

Two general categories of stroke exist, ischemic and hemorrhagic, and each category contains several main types of stroke.

ISCHEMIC STROKE

An ischemic stroke occurs when an artery is blocked and blood is prevented from supplying oxygen and other nutrients to the brain. According to the National Stroke Association, ischemic strokes account for approximately 80 percent of all strokes (NSA, Brain at Risk 8). Symptoms of an ischemic stroke include paralysis on one side of the body and problems with speech and language (Caplan, Dyken, and Easton 26). These symptoms develop suddenly in a few minutes and may become worse over a period of hours or days. The two types of ischemic stroke are the thrombotic stroke and the embolic stroke.

THROMBOTIC STROKE

A thrombotic stroke is often called a cerebral thrombosis. It occurs when a blood clot forms along the wall of an artery, blocking the passage of blood to the brain. A thrombosis does not occur in healthy arteries. Atherosclerosis is often responsible for this clotting. Atherosclerosis is an abnormal condition in the arteries in which a thick, rough deposit forms on the inner wall of the arteries and gradually narrows the passageway so that blood flow is slowed (NSA, Road Ahead 1).
Atherosclerosis is commonly referred to as hardening of the arteries. It starts from a minor injury to the inner lining of the blood vessel, and progresses as cholesterol, fats, and other substances from the blood accumulate along the vessel walls near the rough, injured area (Caplan, Dyken, and Easton 27). From this accumulation, deposits called plaques develop. Because of these enlarging plaques, the passageway for blood flow to the brain narrows. When this type of obstruction of a cerebral artery is only partial, it is called arterial stenosis (Sessler 16). However, blood clots often form over these rough plaques in the artery and block the passageway completely. This complete blockage, or occlusion, of a cerebral artery by a blood clot stops blood flow to the brain and causes a thrombotic stroke (Caplan, Dyken, and Easton 28).

Aside from atherosclerosis, thrombotic stroke may occur where there is a history of hypertension (high blood pressure), cardiovascular disease, diabetes, smoking, or high alcohol intake (Sessler 15). Often preceded by mini-strokes, a thrombotic stroke tends to occur at times when blood pressure is low -- during times of inactivity, particularly at night during sleep (Weiner, Lee, and Bell 145). Thrombotic strokes are quite common; according to the American Heart Association, they account for almost 60 percent of all strokes.
An embolic stroke is often called a cerebral embolism. A cerebral embolism occurs when a blood clot or other undissolved material breaks loose and enters the bloodstream. This clot travels through the bloodstream to the brain. Because arteries in the brain eventually become smaller, the wandering clot eventually reaches an artery too narrow to pass through and lodges there, blocking the blood supply of the artery and causing stroke.

Often an embolus is a piece of a blood clot from a damaged heart or diseased carotid artery in the neck (Weiner, Lee, and Bell 145). Therefore, the middle cerebral arteries and the posterior cerebral arteries are the most common sites of an embolism because of their location as pathways for the flow of blood from the carotids (Chipps, Clanin, and Campbell 70). An embolism occurs suddenly and without warning, and usually takes place while the victim is awake and active (Sessler 17). The American Heart Association reports that about 20 percent of all strokes are caused by cerebral embolisms.
HEMORRHAGIC STROKE

A hemorrhage occurs when a cerebral blood vessel ruptures in the brain and spills blood into surrounding tissue. When this takes place, the supply of blood to some area of the brain is disrupted and tissue in that area dies because the cells are deprived of oxygen and other needed nutrients. An additional affect of a hemorrhage is that the blood leaking from the breakage site accumulates in the surrounding area, this accumulation displaces brain tissue and disrupts specific brain functions (Caplan, Dyken and Easton 31).

FIGURE 8: Hemorrhage from Family Guide by Caplan, Dyken, and Easton.
There are several factors which lead to hemorrhagic strokes. The most common cause of hemorrhage is hypertension, or high blood pressure. The stress of uncontrolled high blood pressure weakens blood vessel walls, leading to degeneration and eventual breakage (NSA, *High Blood Pressure* 2). Aneurysms are a source of hemorrhage which can not be controlled; in fact, aneurysms are believed to be congenital (Caplan, Dyken, and Easton 31). Weak spots in an artery wall are present at birth, and aneurysms develop in adulthood when the weak spots balloon out and fill with blood. Overtime, the aneurysm continues to balloon in size and stretch the thin walls of the artery until they finally burst causing hemorrhage (Sessler 18). High blood pressure can hasten the aneurysm process. Brain hemorrhage is also associated with head injury, acute leukemia, and drug abuse -- especially the use of cocaine, amphetamines, and alcohol (Caplan, Dyken, and Easton 32).

**FIGURE 9:** Aneurysm from *Family Guide* by Caplan, Dyken, and Easton.
According to the American Heart Association, hemorrhagic strokes represent approximately 20 percent of all strokes. When a hemorrhage occurs there is a sudden loss of consciousness and other reactions include headache, decreased alertness, stiff neck, and vomiting (Caplan, Dyken, and Easton 32). Damage to the brain depends on the size, location, and speed of the bleeding. The two main types of hemorrhagic stroke are the subarachnoid hemorrhage and the intracerebral hemorrhage.

SUBARACHNOID HEMORRHAGE

A subarachnoid hemorrhage occurs when there is uncontrolled bleeding into the area under the thin, delicate membrane surrounding the brain. This area is called the subarachnoid space. This type of hemorrhage is most often a result of a ruptured aneurysm (Sessler 21). However, subarachnoid hemorrhages can also be caused by a ruptured arteriovenous malformation, which is usually located on the surface of the brain and composed of many thin-walled vessels that easily burst and allow blood to leak (Caplan, Dyken, and Easton 33). In addition, a subarachnoid hemorrhage may be the result of bleeding from a head injury.

Despite the cause, a subarachnoid hemorrhage has no warning signs and the onset is sudden. Because of the bleeding into the subarachnoid space, this type of hemorrhage increases pressure around the brain instead of affecting the brain itself. Therefore, victims of a subarachnoid hemorrhage do not display
specific stroke symptoms. A person experiencing a subarachnoid hemorrhage may develop stiffness and pain in the neck, a severe headache, dizziness, or double vision (Caplan, Dyken, and Easton 33). In addition, the victim may display drowsiness and confusion, or even a state of unconsciousness (Sessler 21).

Subarachnoid hemorrhages account for 10 percent of all strokes. These hemorrhages are extremely serious because death can occur within minutes. In fact, at least 50 percent of those who suffer a subarachnoid hemorrhage die as a result of the initial bleeding. Of the survivors, 25 percent will have reoccurring bleeding within weeks; and with each episode of bleeding, the risk of death increases (Sessler 21).

**INTRACEREBRAL HEMORRHAGE**

An intracerebral hemorrhage is caused by bleeding into the tissue deep within the brain. This usually occurs because small penetrating blood vessels burst and the escaped blood collects, pressing on the brain and destroying brain tissue in the affected area (Caplan, Dyken, and Easton 35). Intracerebral hemorrhages are largely due to hypertension; however, other causes include recreational drug use, aneurysms, brain tumors, and arteriovenous malformations (Caplan, Dyken, and Easton 34). General symptoms caused by uncontrolled bleeding of an intracerebral hemorrhage include headache, vomiting, and an altered state of alertness. These symptoms are a result of increased pressure in the brain due to an increased volume of blood. Focal symptoms of an
intracerebral hemorrhage are found in specific parts of the body. Because each portion of the brain controls specific bodily functions, focal symptoms indicate the location in the brain where the hemorrhage occurred (Caplan, Dyken, and Easton 36).

An individual is usually awake and active when an intracerebral hemorrhage occurs; however, there are no warning signs and the onset of symptoms is usually quite rapid. An intracerebral hemorrhage accounts for 10 percent of strokes. Most victims of a massive intracerebral hemorrhage die within one to two weeks; however, those who survive this type of stroke usually make a remarkable recovery and recurrence is rare (Sessler 20).
WARNING SIGNS OF STROKE

Fortunately most strokes do give warning signs. By recognizing these symptoms and taking prompt action, a stroke may be averted or its severity reduced. A stroke is a medical emergency and demands immediate care. The sooner the victim receives medical attention, the better the chance of limiting the amount of damage to the brain. In the pamphlet Reducing Risk and Recognizing Symptoms, the National Stroke Association has provided a list of the warning signs of stroke, these include:

-- sudden weakness, numbness, or paralysis of face, arm, or leg (especially on one side of the body)

-- sudden blurred or decreased vision in one or both eyes

-- difficulty speaking or understanding simple statements

-- dizziness, loss of balance or coordination (especially when combined with another symptom)

-- sudden, unexplainable, intense headache (often described as "the worst headache ever")

-- sudden nausea, fever, and vomiting (distinguished from a viral illness by the speed of onset)

-- brief loss of consciousness or period of decreased consciousness (fainting, confusion, convulsion, or coma)

Additional symptoms of stroke include double vision, ringing in the ears or decreased hearing, difficulty swallowing, sudden personality changes (such as irritability or paranoia), emotional lability (uncontrolled crying or laughing), or cognitive changes (impaired memory, judgment, or orientation) (NSA, Road Ahead 5).
Warning signs are the same for persons who have had a previous stroke and for those who have not. These symptoms may occur alone or in combination; however, they are most often temporary -- a transient ischemic attack.

TRANSIENT ISCHEMIC ATTACK (TIA)

A transient ischemic attack is a sudden episode of one or more stroke warning signs which lasts from a few minutes to a few hours (never more than 24 hours), leaving no aftereffects (Sessler 28). A TIA is the most important signal preceding an actual stroke. Referred to as a mini-stroke, a TIA occurs when there is a temporary interruption of the blood supply to a localized area in the brain (NSA, Brain at Risk 5). Because a TIA represents a vascular problem, early recognition and treatment helps to prevent the development of stroke.

The American Heart Association estimates that 20 to 40 percent of all strokes are preceded by TIAs (Caplan, Dyken, and Easton 38). These temporary attacks may occur hours, days, weeks, or months before a full stroke. Some people may only experience one episode, and others may experience numerous TIAs. However, once a TIA has occurred, it tends to recur unless the underlying cause is eliminated (Sessler 29). TIAs are vital indicators of stroke, people who have had TIAs are almost ten times more likely to have a stroke soon thereafter than people of the same age and sex who have not experienced a TIA (Caplan, Dyken, and Easton 38).
The most common cause of a TIA is the presence of small emboli. A small blood clot or a piece of plaque on the walls of an artery breaks off and travels through the bloodstream until it temporarily lodges in a small artery. This brief blockage of blood flow and lack of oxygen affects vital functions of the brain and the TIA occurs (Sessler 30). The average TIA lasts approximately 18 minutes. When the emboli passes through the blood vessel and past the vital control centers of the brain, the episode ends, the symptoms disappear, and there is a return to normal function. This recovery may occur because the plaques break up or the plaques are dissolved by substances in the blood; this allows the flow of blood to return to normal (Sessler 30). Another possibility is that the collateral circulation is sufficient to maintain a flow of blood to all vital areas of the brain; therefore, brain cells are short of oxygen temporarily, but do not die (Sessler 31).

A TIA is a reliable predictor of strokes. Despite the fact that the symptoms of a TIA quickly disappear, they should not be ignored. Medical consultation should be sought immediately because only a professional can determine whether it was a stroke that occurred, a TIA, or some other medical problem with similar symptoms. Stroke-related brain damage gets worse the longer a stroke goes untreated. Therefore, immediate medical treatment for the warning signs reduces the risk of death and improves chances for successful rehabilitation and recovery after stroke. If a TIA has occurred, prompt medical attention will evaluate and
treat the underlying causes and problems. In turn, this knowledge of a TIA, lifestyle changes, prescribed medication, and medical treatment will reduce the risk of full stroke.
AFFECTS OF STROKE

The brain is a complex organ and each area within the brain is responsible for specific functions. A stroke does not affect all areas of the brain. The damage caused by stroke depends on the type and severity of stroke, and where it occurs in the brain. The location of a stroke determines which parts of the brain are damaged. These affected areas are no longer able to control their functions; therefore, stroke results in specific physical and mental impairments.

Usually a stroke occurs in one hemisphere of the brain. Although the hemispheres share responsibilities for some functions, each side of the brain also has its own specialties which control separate functions of the body. Therefore, a common method of characterizing stroke injury is by the particular side of the brain that is damaged.

LEFT HEMISPHERE BRAIN INJURY

RIGHT HEMIPLEGIA / HEMIPARESIS

The most obvious sign of stroke is paralysis on one side of the body. Damage to the left side of the brain often results in paralysis on the right side of the body, this is known as right hemiplegia. Hemiplegia is defined as the motor dysfunction of stroke which causes a paralysis of one side of the body (Ryerson 474). Right hemiparesis is weakness, instead of paralysis, on the right side of the body.
APHASIA

The left side of the brain predominantly controls the speech and language centers; damage to these areas can cause aphasia. The term aphasia applies to a wide range of communication deficits. It is a catch-all phrase used to describe a condition in which there is interference with the understanding of and use of speech and language (Sessler 206). The types of aphasia can be divided into three broad categories: Wernicke’s aphasia, Broca’s aphasia, and global aphasia.

WERNICKE’S APHASIA: Wernicke’s aphasia occurs when there is damage to the auditory association cortex of the left temporal lobe. When suffering from Wernicke’s aphasia, also known as receptive aphasia, a person is unable to understand language. The individual needs a prolonged reaction time in order to process verbal input. Wernicke’s aphasia is characterized by a decrease in the ability to isolate, recognize, and select phonemes; thus, impairing comprehension (Walsh 54). This confusion of the sounds may also impair reading and writing. A person suffering Wernicke’s aphasia can often speak fluently; however, their message lacks meaning because of the improper use of phonemes, the use of inappropriate words and made up words, and word finding problems (Walsh 54). Despite the fact that the speech is gibberish and confusing, a person suffering from Wernicke’s aphasia is unaware of his or her errors and does not notice that his or her message is not understood.

BROCA’S APHASIA: Broca’s aphasia, also known as motor or
expressive aphasia, occurs when there is damage to the inferior frontal gyrus of the left frontal lobe. In Broca’s aphasia, comprehension of language is fairly accurate, but the individual has difficulty with expression. Speech is slow, deliberate, and quite simple in structure as a result of apraxia of speech (Walsh 55). Apraxia is the disruption of a well learned movement; it is the inability to carry out purposeful, learned, voluntary acts although there is no paralysis present (Liebman 71). In Broca’s aphasia, the person is not able to initiate the process of speaking because he or she no longer "remembers" how it is programmed (Walsh 55). The individual knows what he or she wants to say, but is unable to communicate it in speech and often becomes frustrated.

A major characteristic of Broca’s aphasia is nonfluent output. A person suffering from Broca’s aphasia does not comprehend small grammatical words, grammatical word endings, or grammatical word order (Walsh 55). Therefore, their speech is often missing small, connecting words which provide proper sentence structure.

GLOBAL APHASIA: Global aphasia is a combination of Wernicke’s aphasia and Broca’s aphasia. A person with global aphasia has difficulty with both comprehension and expression (Caplan, Dyken, and Easton 98). The individual provides only utterances, there are usually no functional language skills present.
Another common aphasia is **anomic aphasia**, this is a condition in which the person has difficulty producing the names of people or objects (Caplan, Dyken, and Easton 99). Often people with aphasia display related problems with reading and writing. The loss of the ability to write is called **agraphia**. **Alexia**, or visual aphasia, is known as word blindness. Individuals with alexia can see printed words, but are unable to read them; thus, the words have no meaning (Liebman 71).

**DYSARTHRIA AND DYSPHASIA**

Dysarthria is another type of speech impairment common to left hemisphere injury. Dysarthria is a mechanical deficit in speaking caused by disturbances or injuries to the area of the brain which controls the muscles that regulate articulation (Sessler 208). This impairment in articulation often results from weakness, slowness, or incoordination of the mouth, tongue, and jaw. Signs of dysarthria include slurred speech, slow and imprecise speech, monotonous speech, and lack of fine control in the mechanics of speech (NSA, *Road Ahead 60*).

Dysphasia is often found in association with many speech and language impairments. Dysphasia is defined as an impaired ability to swallow (NSA, *Road Ahead 144*).
RIGHT HEMIANOPSIA

Hemianopsia is permanent damage to the optic nerve which results in blindness in one half of each eye. The same half of each eye is affected corresponding to the side of the body which is affected by the stroke (NSA, Road Ahead 146). When a stroke damages the left cerebral hemisphere, there is a loss of the right visual field. In right hemianopsia, a person can see only toward the left (Weiner, Lee, and Bell 173). The whole of any object cannot be seen, and the individual is unable to perceive the environment on the affected side. For example, a person suffering from right hemianopsia may see and eat food only on the left half of the plate, leaving the right side untouched. This problem is often referred to as one-sided neglect. Most often, the person is unaware of this change and must be taught to turn his or her head to compensate for this lack of vision (Sessler 215).

APRAXIA

Left brain injury often results in a condition of impaired voluntary motion known as apraxia. Apraxia is a perceptual and cognitive impairment. It is the inability to voluntarily perform certain skilled, purposeful movements despite presence of adequate strength, sensation, and coordination (NSA, Road Ahead 142). When experiencing apraxia, a person is able to perform the movements when not thinking about it, but cannot do so if asked.
IMPAIRED THOUGHT PROCESSES

Persons suffering a stroke to the left hemisphere may have difficulty processing information. These persons may not be able to remember all aspects of a task. They also may have difficulty understanding a whole idea, instead they may only grasp a small part of it. And because of their impaired retention, individuals who have damage to the left side of the brain require a significant amount of repetition during recovery (Sessler 212). In addition to these comprehension deficits, these individuals may also have problems with math, logic and analytical skills.

OTHERS

-- When approaching an unfamiliar situation or problem, right hemiplegics tend to be slow, cautious, and disorganized. These individuals also frequently display anxiety (AHA, How Stroke Affects Behavior 9).

-- Right hemiplegics often display compulsiveness and slowness, and they are easily frustrated (NSA, Road Ahead 3).

-- Right hemiplegics demonstrate increased lability. Lability is an impairment of emotional control after brain damage. It is displayed frequently as a brief episode of spontaneous crying or laughing with no apparent stimulus (NSA, Road Ahead 147).

-- Persons suffering from injury to the left hemisphere have impaired left-right discrimination (NSA, Road Ahead 3).
RIGHT HEMISPHERE BRAIN INJURY

LEFT HEMIPLEGIA / HEMIPARESIS

Damage to the right hemisphere often results in hemiplegia (paralysis) on the left side of the body, or hemiparesis (weakness) on the left side of the body.

SPATIAL AND PERCEPTUAL DEFICITS

Right brain injury often results in impaired spatial and perceptual skills; this refers to the inability to judge distance, size, position, speed, form, and the relation of parts to wholes (AHA, *How Stroke Affects Behavior* 11). This type of disability can be compared to the feeling one experiences when he or she misses the last step at the bottom of a flight of stairs and has a crash landing. Or when one misjudges the edge of the table when trying to set down a glass. Victims of stroke damage to the right hemisphere constantly struggle with these deficits. For example, many stroke patients are unable to get their wheelchair through a doorway without bumping into the walls; or they are not able to distinguish between the right and wrong side of their clothing; or perhaps they have the ability to read but cannot keep their place on the page. Obviously these deficits not only affect judgment, but also orientation, awareness, and visual memory (Weiner, Lee, and Bell 176).

Individuals suffering right side brain damage may have difficulty recognizing familiar objects, people, and information. These persons become lost easily, even in their immediate
environment; and they often have problems with the concept of time (Weiner, Lee, and Bell 176). These individuals frequently display dressing apraxia. They do not remember the concept of getting dressed; they can see what needs to be done, but they lack the spatial perspective to do it correctly. For example, they may put on their clothing in the wrong order and not recognize the error (Weiner, Lee, and Bell 176).

PRAGMATIC DEFICIT

A pragmatic deficit is a communication problem characterized by excessive verbalization, which is caused by damage to the right hemisphere. A person with a pragmatic deficit is usually fluent with good vocabulary and correct grammar; however, this person may talk too much, go off on tangents, stray from the topic of conversation, or discuss an inappropriate subject with the listener (NSA, Road Ahead 60). In addition, an individual with this deficit has difficulty recognizing and responding to facial expressions and tone of voice (NSA, Road Ahead 60).

LEFT HEMIANOPSIA

When a stroke damages the right cerebral hemisphere, the individual may be blind in the left half of each eye. Because an individual suffering from left hemianopsia has lost their left visual field, he or she can see only toward the right (Weiner, Lee, and Bell 175). The individual often does not acknowledge anything in the left side of his or her environment, the left
half of any object, or the left side of his or her body. For example, this person may bump into things on the left when attempting to steer a wheelchair; or this individual may only comb his or her hair, shave, or apply make-up on the right side. As with right hemiplegics, left hemiplegics must be instructed to compensate for this one-sided neglect. However, left hemiplegics have more problems with neglect than do right hemiplegics (AHA, How Stroke Affects Behavior 15).

**SHORT ATTENTION SPAN**

Many persons who suffer a stroke to the right side of the brain have high distractibility, which results in a short attention span (NSA, Road Ahead 2). These individuals are usually unable to do activities that require full and focused attention; and they often have problems completing a task.

**PERSONAL BEHAVIOR**

Aside from their problems judging spatial-perceptual relationships, left hemiplegics also display poor judgment in their behavior. These individuals tend to overestimate their abilities. A left hemiplegic is often unaware of his or her impairments and tends to be impulsive and act too fast (Sessler 217). Therefore, a left hemiplegic will frequently attempt tasks that are not within his or her abilities and are dangerously unsafe (NSA, Brain at Risk 11).
OTHERS

-- Left hemiplegics may experience short-term memory problems (NSA, Brain at Risk 11). For example, they may remember a family reunion that took place 20 years ago; however, they are unable to recall who visited just two hours earlier.

-- Left hemiplegics have impaired abstract thinking, they tend to display only concrete thinking (NSA, Road Ahead 2).

-- Individuals who experience a stroke in the right hemisphere often demonstrate a lack of interest, lethargy, and may appear emotionally flat. However, these periods will be suddenly interrupted by brief emotional highs and lows, such as an uncontrollable burst of swearing and angry words (Weiner, Lee, and Bell 177).

INJURY TO SPECIFIC AREAS OF THE BRAIN

The left hemisphere / right hemisphere general characterization is a common method used for describing and categorizing impairments which result from stroke. However, below is an outline in conjunction with the second section of this paper "Background of Brain Anatomy and Function." This outline displays stroke impairments which result from damage to the specific areas of the brain. (These areas of the brain have previously been described on pp. 3-18.)
FOREBRAIN
TELENCEPHALON (CEREBRUM)
FRONTAL LOBE

precentral region
- primary motor cortex -- problems initiating voluntary movements of the muscles on the opposite side of the body; particular skeletal muscles affected depends on the location of injury on the motor homunculus.
- premotor cortex -- difficulty planning sequential movements and orienting the body toward a specific target (Caplan, Dyken, and Easton 14).
- Broca’s area -- Broca’s aphasia; problems planning and programming speech.

prefrontal region
- behavioral and personality changes.
- uninhibited behaviors and lack of social conscience; may speak and/or act inappropriately and/or impulsively (Caplan, Dyken, and Easton 15).
- condition called abulia, a lack of initiative and apathy (Caplan, Dyken, and Easton 15).
- decreased memory for order of events,
- personal body orientation deficits,
- inability to adapt and change strategies (Walsh 50).

PARietal LOBE

somatosensory cortex
- difficulty receiving and recognizing sensory input.

posterior parietal lobe
- difficulty integrating somatosensory, visual, and auditory input,
- tactile agnosia (inability to recognize touch),
- visual agnosia (inability to recognize what is seen),
- inability to link information from different sensory systems (cross-modal matching),
- decreased complex sensory analysis (inability to use sensory information),
- decreased short term memory (Walsh 51).

LEFT PARIETAL LOBE
- language related problems such as anomia, agraphia, and alexia,
- poor grammar and math,
- impaired verbal and numerical short term memory,
- apraxia (impaired motor programming and motor memory),
- poor left-right discrimination (Walsh 51).
- sensory neglect of the right side.

RIGHT PARIETAL LOBE
- impaired use of spatial information.
- dressing apraxia (Weiner, Lee, and Bell 176).
- constructional apraxia (inability to put together),
- sensory neglect of the left side,
- impaired nonverbal short term memory,
- impaired recognition skills (Walsh 52).
TEMPORAL LOBE

auditory association cortex
- impaired auditory sensation and perception.
- (left) Wernicke’s aphasia; problems understanding speech and language.
- (right) impaired recognition of nonverbal sounds and sequencing (Walsh 53).
- (bilateral) deafness.

complex association area
- impaired selective attention,
- (left) impaired verbal long term memory,
- (right) impaired nonverbal long term memory (Walsh 53).

limbic cortex
- personality changes with a tendency toward aggression and hypersexuality.

OCCIPITAL LOBE
- (left) right hemianopsia (right side of each eye is blind).
- (right) left hemianopsia (left side of each eye is blind).
- possible visual agnosia.

DIENCEPHALON

THALAMUS
- profound sensory loss.

HYPOTHALAMUS
- damaged links to autonomic nervous system and endocrine system,
- impaired ability to regulate homeostatic functions (Sherwood 126).

BRAINSTEM
- unstable vital signs; possibly fatal.
- impaired survival functions such as breathing, blood pressure, and heart rate (Liebman 6).
- coma or low-level consciousness,
- loss of muscle tone and paralysis (one-sided or bilateral),
- nausea and vomiting (NSA, Road Ahead 3).
- impaired swallowing, hearing, speech, and eye movements (NSA, Brain at Risk 12).

CEREBELLUM
- ataxia, muscular incoordination, and balance problems,
- abnormal reflexes of head and trunk,
- dizziness, nausea, and vomiting (NSA, Road Ahead 3).

The specific abilities affected by stroke depend on the location in the brain where the stroke occurs, the type of stroke which occurs, and the severity of the stroke. It is important to note that no two strokes are exactly alike. Each stroke is
unique because the affects of a brain injury are individualized (Weiner, Lee, and Bell 146). Therefore, the impairments mentioned in this paper are considered to be generalizations. Recovery and rehabilitation efforts are also unique to each stroke survivor. Recovery from stroke depends on the amount and location of damage in the brain, the individual’s overall health, the individual’s personality and emotional state, the support of loved ones, medical treatment, therapy, and other rehabilitation efforts (Caplan, Dyken, and Easton 24).
RISK FACTORS OF STROKE

Risk factors of stroke are unfavorable conditions and behaviors which make a person more susceptible to stroke. Some of these factors are not controllable, such as age, race, gender, diabetes, previous stroke or TIA, or family history of stroke. However, most major stroke risk factors can be controlled by lifestyle changes and/or medical attention, these include: hypertension, heart disease, high cholesterol, smoking, alcohol intake, obesity, lack of exercise, stress, and use of oral contraceptives.

UNCONTROLLABLE STROKE RISK FACTORS

AGE

The risk of stroke steadily increases with age. According to the National Stroke Association, two thirds of all strokes affect people age sixty-five or older. And the risk of stroke doubles with each decade past age fifty-five (NSA, Brain at Risk 15).

RACE

Different ethnic groups display varying levels of all risk factors. Scientists believe these differences may be genetically inherited and also due to environmental and social factors. Thus, it is believed that a person's race is a specific indication of stroke risk (Caplan, Dyken, and Easton 48). For
example, African Americans have a higher stroke risk than other racial groups. In fact, according to the American Heart Association, African Americans are 60 percent more likely than Caucasians to suffer a stroke (Caplan, Dyken, and Easton 47).

GENDER

Men are more likely to have a stroke than women.

DIABETES

Diabetes is a disease in which the body does not produce or does not properly use insulin. Insulin is needed in order for the body to convert sugar and starch into energy (Caplan, Dyken, and Easton 300). Diabetes increases stroke risk; in fact, about 20 percent of the people who experience stroke also suffer from diabetes (Caplan, Dyken, and Easton 52). This is partially due to circulation problems caused by diabetes. Because this disease affects cholesterol and triglyceride levels in the blood, it increases the risk of cardiovascular disease, which can lead to thrombotic stroke (Caplan, Dyken, and Easton 52).

PREVIOUS STROKE OR TIA

After experiencing a prior stroke or TIA, a person's risk of stroke increases by ten times. The American Heart Association claims that 20 to 40 percent of all strokes are preceded by a TIA or TIAs (Caplan, Dyken, and Easton 38).
FAMILY HISTORY

Family history of stroke or TIA increases an individual's risk of stroke (NSA, *Brain at Risk* 15). Also, scientists are now investigating a possible genetic predisposition to stroke. Many of the risk factors related to stroke are partially hereditary, including: hypertension, heart disease, atherosclerosis, and obesity. Therefore, the genetic make-up of an individual may significantly elevate his or her risk of stroke (Caplan, Dyken, and Easton 47).

CONTROLLABLE STROKE RISK FACTORS

HYPERTENSION

Blood pressure is a measurement of the force blood exerts on blood vessel walls as it travels through the body's circulatory system. A blood pressure reading consists of two numbers expressed in a fraction format (s/d). The number on top (s), is the systolic blood pressure which measures the maximum force blood exerts on blood vessel walls as the heart pumps. The number on bottom (d), is the diastolic blood pressure which measures the minimum force blood exerts on blood vessel walls when the heart is at rest between beats (Caplan, Dyken, and Easton 57). Blood pressure will vary with stress or exercise; however, normal blood pressure is 140/90 or lower (NSA, "Stroke Screenings" 1).

The medical name for high blood pressure is hypertension and it is defined as blood pressure consistently higher than 140/90.
According to the National Stroke Association, approximately 50 million Americans have high blood pressure -- that is one in every four Americans (NSA, "High Blood Pressure" 7). High blood pressure is the single most important controllable risk factor of stroke, increasing the risk between four to six times (NSA, Brain at Risk 16). In the pamphlet High Blood Pressure and Stroke, the National Stroke Association lists the harmful effects of high blood pressure which may lead to stroke:

-- High blood pressure puts extra stress on blood vessel walls which may cause the walls to thicken, resulting in atherosclerosis and leading to a cerebral thrombosis.

-- Hypertension may weaken the walls of blood vessels in the brain, causing vessel breakage and cerebral hemorrhage.

-- Hypertension can forcefully stretch a weak spot in the wall of an artery so that an aneurysm develops. When this balloon-type structure bursts, a hemorrhagic stroke may result.

-- High blood pressure can cause clots and plaques to break off of vessel walls, enabling them to travel through the bloodstream and possibly cause an embolic stroke. (2)

High blood pressure is often referred to as "the silent killer" because it has no symptoms; yet it is a serious condition which can produce life-threatening medical problems. Therefore, it is important for people of all ages to have their blood pressure checked regularly. The National Stroke Association provides several additional suggestions for reducing the risk of hypertension, these include: take prescribed high blood pressure medication regularly, stop smoking, reduce salt, fat, and cholesterol intake, increase physical activity with an exercise program, reduce alcohol consumption, manage stress effectively,
and maintain a sufficient dietary intake of potassium, calcium, and magnesium ("High Blood Pressure" 7).

HEART DISEASE

The American Heart Association claims that persons with some form of heart disease are twice as likely to have a stroke than persons without it (Caplan, Dyken, and Easton 62). Heart disease is often linked to the accumulation of deposits on blood vessel walls and/or to hypertension (NSA, Brain at Risk 19). Also, a heart attack may damage the heart and create emboli which eventually cause cerebral infarction. Other heart ailments which increase the risk of stroke include: congestive heart failure, rheumatic heart disease, and atrial fibrillation or other cardiac arrhythmias (Caplan, Dyken, and Easton 62).

Atrial fibrillation affects over one million Americans, this type of heart disease increases stroke risk from four to six times (NSA, Atrial Fibrillation 1). Approximately 15 percent of all people who have a stroke also suffer from atrial fibrillation, or AF (NSA, Brain at Risk 19). In Af, the left atrium (left upper chamber) of the heart beats rapidly and unpredictably. These irregular contractions make it difficult for all of the blood in the left atrium to be emptied from the chamber. Therefore, blood pools in the left atrium and tends to form clots. These clots can break loose and travel through the bloodstream to the brain, where they may cause a cerebral embolism.
AF has varying outward symptoms which are often difficult to detect. An individual may experience heart palpitations, may feel dizzy or faint or light-headed, may experience mild or severe chest pain, or may display no symptoms at all (NSA, Atrial Fibrillation 2). Therefore, the only reliable method of determining AF is a medical test called an electrocardiogram (ECG or EKG) (NSA, Atrial Fibrillation 1). AF is commonly treated with prescribed medication. Other types of heart disease are often treated with a monitored diet and exercise program, or surgery and/or medication.

HIGH CHOLESTEROL

Cholesterol is a fatlike substance found in the blood that the body has manufactured and/or obtained from fat in the foods digested (Caplan, Dyken, and Easton 69). High cholesterol is an elevated level of blood lipids (fats), this increases the risk of stroke and heart disease because it promotes the development of atherosclerosis. A cholesterol level of more than 200 mg/dl is considered high (NSA, Brain at Risk 17). A person’s cholesterol level should be checked periodically because there are no outward symptoms of high cholesterol.

Cholesterol is carried in the bloodstream by lipoproteins. There are two types of lipoproteins, the high-density lipoprotein (HDL) and the low-density lipoprotein (LDL). HDLs carry cholesterol out of the arteries to the liver where it is eliminated (Caplan, Dyken, and Easton 70). LDLs retain
cholesterol and allow the excess to accumulate on artery walls, which leads to atherosclerosis (Caplan, Dyken, and Easton 70). Therefore, individuals want to raise their HDL levels and lower their LDL levels. This can usually be accomplished by following a low-fat diet, exercising regularly, and terminating any smoking habits. For those people who can not control their cholesterol levels, medication can be prescribed.

**SMOKING**

An estimated 55 million people in the United States smoke. This habit doubles a person’s risk of stroke (NSA, Brain at Risk 21) and also increases the chance of developing heart disease and atherosclerosis (Caplan, Dyken, and Easton 64). Cigarette smoke consists of may dangerous substances which have adverse affects on the body. The nicotine found in cigarette smoke causes blood vessels to constrict, which increases blood pressure and heart rate. Carbon monoxide from smoke which enters the bloodstream disrupts the available supply of oxygen and forces the heart to work harder. Other harmful substances from cigarette smoke damage blood vessel walls and leave behind plaque-like deposits which create prime conditions for atherosclerosis. The risk of stroke caused by smoking is most detrimental to women who also take high-level estrogen birth-control pills. These women are 22 times more likely to suffer a stroke than their peers who do not smoke and use other methods of birth control (Caplan, Dyken, and Easton 65).
If a person stops smoking, his or her risk of stroke will decrease within two years; and within five years, his or her stroke risk will be similar to someone who has never smoked (NSA, *Brain at Risk* 21). A physician can provide the best information concerning a program for smoking cessation.

**ALCOHOL INTAKE**

Excessive alcohol consumption (more than two drinks a day) and binge drinking have been linked to an increase in blood pressure -- the most vital risk factor associated with stroke. Therefore, the American Heart Association suggests drinking no more than one ounce of pure alcohol, or its equivalent, per day (Caplan, Dyken, and Easton 72).

**OBESITY**

Obesity is defined as being more than 30 percent overweight (Caplan, Dyken, and Easton 73). This excess weight puts unnecessary strain on an individual’s circulatory system. In addition, obese people are more likely to develop high blood pressure, heart disease, high cholesterol, and diabetes (Caplan, Dyken, and Easton 73). A low-fat diet together with a sensible exercise program can help control these stroke risk factors.

**EXERCISE**

Regular periods of physical activity can help prevent heart disease, hypertension, obesity, and heart attack. Also, this
increased exercise will positively influence cholesterol levels by raising the level of desirable HDLs. The American Heart Association recommends moderate exercise three times a week with each period lasting fifteen to thirty minutes (Caplan, Dyken, and Easton 73). However, before implementing an exercise program, a physician should be consulted.

STRESS

Stress influences several risk factors associated with stroke. Stress affects hypertension, contributes to atherosclerosis, and elevates the level of cholesterol in blood (Sessler 181). The National Stroke Association suggests for people at risk of stroke to avoid stressful situations; and to learn and practice relaxation techniques which aid in stress management (Road Ahead 6).

ORAL CONTRACEPTIVES

High-level estrogen birth-control pills of the past were known to raise a woman's blood pressure, blood cholesterol, and blood sugar levels. These versions of the Pill were also thought to contribute to the formation of blood clots (Caplan, Dyken, and Easton 74). However, recent versions of this oral contraceptive contain lower estrogen levels. Therefore, new studies are now in progress to determine whether a link still exists between oral contraceptives and stroke. Despite this controversy, if a woman smokes and is taking the estrogen birth-control pills, she is at
an extremely high risk for stroke (Caplan, Dyken, and Easton 65). Thus, a woman smoker must choose an alternative form of birth control.
MEDICAL DIAGNOSIS AND TREATMENT

Medical researchers are currently discovering more information and developing a better understanding of stroke. In addition to this knowledge, innovative technology has provided accurate and reliable diagnostic procedures for stroke, and also developed successful methods of stroke prevention and stroke treatment.

STROKE DIAGNOSTICS

EVALUATING BRAIN TISSUE AND STRUCTURE

COMPUTERIZED AXIAL TOMOGRAPHY (CAT or CT SCAN): Ct scans use an X-ray beam to take several cross-sectional pictures of the brain. A computer then calculates the x-ray penetration of each tissue, creating a three-dimensional view of the brain (Chipps, Clanin, and Campbell 37). A CT scan allows doctors to quickly determine the nature of the stroke -- ischemic or hemorrhagic. This discovery determines all further treatment of the stroke. However, this test does have some limitations. CT scans do not reveal a great amount of detail, they do not detect TIAs, and they have low-quality spatial resolution. Therefore, this picture is only able to display the general location of the stroke (NSA, "Something Old" 7). In addition, the CT scan is hindered by a 24 hour time lapse which exists before a full image of the tissue damage can be provided (NSA, "Something Old" 7).
MAGNETIC RESONANCE IMAGING (MRI): MRI is a procedure based on the magnetic behavior of protons in body tissues. When a strong magnetic field is applied, atoms in the body align themselves uniformly. Radiofrequency waves are then applied and these pulses affect the movement of the nuclei of these atoms. After the radiofrequency waves are terminated, the atoms generate a voltage within the magnetic field which is analyzed by a computer to generate a three-dimensional image of the brain (Chipps, Clanin, and Campbell 38). For an MRI scan, the patient must lie very still within the narrow confinements of the scanner for approximately an hour. This restraining procedure is often considered unpleasant by patients.

MRI images provide doctors with a sharp, detailed picture of the brain and its most intricate injuries (Caplan, Dyken, and Easton 106). This test furnishes a full image of stroke within 6 to 12 hours of its occurrence. Aside from determining the nature of stroke and discovering small injuries, MRI is also able to locate and measure strokes in the brainstem or cerebellum, and smaller strokes deep within the brain, and even TIAs (NSA, "Something Old" 7). This procedure can even distinguish between ischemic tissue (cells dying because of a lack of oxygen) and infarcted tissue (cells which are dead because of a lack of oxygen). In the future, this capability may help doctors save ischemic tissue; thus, limiting stroke damage and improving recovery efforts (Caplan, Dyken, and Easton 197).
DIFFUSION-WEIGHTED MRI: Diffusion-weighted MRI is a new technology which is in limited use throughout the country. "Diffusion-weighted MRI uses rapidly changing magnetic fields to detect the water that floods brain cells after injury" (NSA, "Something Old" 7). This procedure supplies vital information about the size, location, and severity of an ischemic stroke; and it can accurately detect damage within an hour of stroke onset. Thus, in the future this test may be performed when a stroke is in progress (NSA, "Something Old" 14).

EVALUATING VASCULAR CONDITIONS

ANGIOGRAPHY: In angiography, a tube is threaded into a blood vessel and special dye agents are injected into the vessels via this tube. Through a series of CT or MRI scans, this dye will show the size and shape of the blood vessels and also locate occlusions (blockages) or other irregularities (Chipps, Clanin, and Campbell 39). Although this invasive medical procedure has few complications, it is not the most efficient method of evaluating the conditions of blood vessels.

ULTRASONOGRAPHY: To get an ultrasound reading, a sensitive probe is placed over an artery in the neck or an artery at the base of the skull. This device sends sound waves through the skin; some of these sound waves are absorbed by the tissue and some are reflected (NSA, Road Ahead 11). The probe picks up echoes that bounce back and sends them to a computer. The computer records and analyzes this information and converts the
echoes into images which reflect the condition of the particular blood vessel (NSA, Road Ahead 11).

**POSITRON EMISSION TOMOGRAPHY (PET):** During PET, a patient is injected with or inhales some type of radioactive material that emits positrons. Once this radioactive substance is absorbed, its positron emitters react with a negative electron, which causes the release of gamma rays (Chipps, Clanin, and Campbell 40). The gamma rays are detected and analyzed by a computer, which then constructs color-coded images of the vessels. PET makes it possible for doctors to evaluate cerebral blood flow at the capillary level (NSA, "Something Old" 8).

Aside from diagnostic information concerning vascular structure, PET is also able to provide details about cerebral function, such as oxygen metabolism. With these capabilities, PET is a resource which helps physicians distinguish between dead and damaged brain cells (NSA, "Something Old" 8).

**PERFUSION MRI:** Perfusion MRI is the newest vascular imaging technique, it uses a series of ultra-fast MRIs to track the progress of a standard dye agent as it circulates through the vessels (NSA, "Something Old" 8). The MRI signal is lost in areas with sufficient blood supply, and the signal is transmitted in areas with an inadequate supply of blood (NSA, "Something Old" 8). Therefore, the signals immediately create a three-dimensional image which pinpoints regions damaged by stroke.
CAROTID ENDARTERECTOMY

The carotid arteries in the neck carry blood from the heart to the brain. Narrowing of these arteries due to atherosclerotic damage is called carotid stenosis. If a person’s carotid arteries demonstrate blockage of 70 percent or more, he or she may be a candidate for a surgical procedure known as carotid endarterectomy (NSA, "Major Trail" 4). The goal of carotid endarterectomy is to prevent an ischemic stroke. During this procedure, surgeons open an artery and remove fatty deposits and harmful plaques from the vessel walls (NSA, "Strides" 15). Thus, when blockage is cleared from the artery, the free flow of blood is restored. This surgery helps prevent stroke in individuals who have severely blocked carotid arteries; and it helps reduce the risk of recurrent stroke in individuals whose initial stroke was due to carotid stenosis (NSA, Recurrent Stroke 1).

SURGICAL TREATMENT OF HEMORRHAGIC STROKE

According to the National Stroke Association, a surgical method for treating subarachnoid hemorrhage is becoming quite common. This procedure involves the early clipping of aneurysms (NSA, "Strides" 15). In addition, scientists are researching three surgical methods for the treatment of intracerebral hemorrhage. Robert C. Heros, M.D. summarizes these methods for the National Stroke Association:
Stereotactic radiosurgery is a procedure in which surgeons use 3-D imaging to precisely locate the hemorrhage, and then operate with a laser.

In ultrasonic aspiration, the blood clots which form after an artery ruptures are dissolved with extremely high-frequency sound waves and then removed using suction.

The injection of thrombolytic, or clot-busting, drugs. ("Strides" 15)

DRUGS AND MEDICATION

ANTIHYpertensives

When blood pressure can not be controlled through diet and exercise, a physician may prescribe an antihypertensive medication. There are a wide range of these drugs available, and each drug may cause varying responses and/or side effects. A person may undergo several trial periods before deciding which drug is most effective and has the fewest side effects. The American Heart Association provides a list of the six main types of antihypertensive medications:

-- DIURETICS: This class of drugs eliminates excess salts and water from the body.

-- SYMPATHETIC NERVE INHIBITORS: This class of drugs blocks messages from the brain which would constrict blood vessels.

-- VASODILATORS: This type of drug relaxes muscles in the artery walls, causing them to dilate.

-- ANGIOTENSIN CONVERTING ENZYME INHIBITORS: These drugs interfere with this enzyme to keep arteries dilated and blood pressure down.

-- CALCIUM ANTAGONISTS: This class of drugs reduces a person’s heart rate and relaxes blood vessels.
BETA BLOCKERS: These agents are involved in many mechanisms which work to decrease blood pressure. (Caplan, Dyken, and Easton 54)

Any type of high blood pressure medication must be taken exactly as prescribed by the physician.

ANTICOAGULANT / ANTIPLATELET THERAPY

Anticoagulants, or blood thinners, are medications which interfere with the production of certain blood components that are necessary for the formation of clots (NSA, Brain at Risk 25). This type of drug therapy prevents recurrent embolic stroke; however, it also has a serious risk associated with it -- hemorrhage (Caplan, Dyken, and Easton 122). Anticoagulants are complex drugs; their use should be monitored and should be as brief as possible. Heparin and warfarin are two types of anticoagulant medication. Heparin therapy is given intravenously and it lasts only a few days; this treatment immediately prevents blood from clotting (Caplan, Dyken, and Easton 122). Warfarin, commercially known as Coumadin, is designed for long term use and is administered in the form of a pill. This drug prevents existing blood clots from growing larger and it also prevents new clots from forming (NSA, Brain at Risk 26).

Antiplatelet medications inhibit an enzyme which is responsible for the body's platelet formation (NSA, Brain at Risk 24). Platelets are the element of blood which adhere together to form a clot. Antiplatelet medications prevent these platelets from sticking together to begin the clotting process. These
drugs are used primarily for persons with atherosclerosis, for persons with increased clotting tendencies, or for persons who have previously experienced stroke or TIA (Caplan, Dyken, and Easton 123). Aspirin and ticlopidine (Ticlid) are two common forms of antiplatelet medication. Aspirin is the drug most commonly prescribed to prevent stroke. It has been shown to reduce stroke risk by about 20 percent, especially for persons with a history of atherosclerosis, hypertension, or atrial fibrillation (NSA, *Brain at Risk* 25). Ticlopidine is a new drug only prescribed for people who have had a previous stroke or TIA. In fact, ticlopidine has proven to be more effective than aspirin in preventing recurrent stroke or further stroke after TIA (NSA, *Brain at Risk* 25). Like aspirin, ticlopidine prevents clotting by keeping blood platelets from adhering together.

**THROMBOLYTIC DRUGS**

Thrombolytic drugs are experimental drugs which are now being tested for the treatment of ischemic stroke. These drugs are referred to as "clot-busters" because they are believed to dissolve blood clots in the arteries going to the brain (NSA, "Thrombolytic Therapy" 13). Trials are now being administered for several thrombolytic drugs such as tissue plasminogen activator (tPA), streptokinase, and urokinase (NSA, "Darkness into Light" 10).
NEUROPROTECTIVE AGENTS

Damage from a stroke on the cellular level is a result of a process called excitotoxicity. The "glutamate cascade" involved in this process is responsible for spreading stroke damage to cells in surrounding areas of the brain. This discovery of the cellular process of stroke has lead to research regarding new methods of stroke treatment. Scientists hope to interrupt the "glutamate cascade" or counteract the resultant damage (NSA, "Darkness into Light" 11). This interaction would protect healthy brain cells in the areas surrounding the stroke which are threatened by the cascade (NSA, Stroke is a Brain Attack 4). Drugs being developed and tested for this purpose are called neuroprotective agents, or glutamate-receptor blockers.
CONCLUSION

It is my hope that after reading this paper, individuals are more content with their knowledge of stroke. Stroke is a life-threatening experience which demands immediate medical attention. Therefore, it is important to be cautious of any stroke warning signs and stroke risk factors. We have a great deal of control over our health. By implementing preventive changes in lifestyle individuals can greatly reduce their risk of stroke. In addition, advanced technology has provided innovative diagnostic methods, successful surgical procedures, and effective medication for survivors of stroke. With the combination of health conscious preventive measures and medical breakthroughs, there is great hope for preventing and treating stroke effectively in the future.


