Cerebrovascular Crises Leading to Major Neurological Disabilities and Rehabilitation Implications for the Occupational Therapist

by

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ABSTRACT:

A Cerebrovascular Accident (CVA) is the most common label ascribed to people who have experienced strokes. Ischemic Stroke, Hemorrhagic Stroke, and Transient Ischemic Attacks (TIA) are the most common causes for neurological disability that impairs quality of life. CVA's can be the result of a variety of morbidities, including thrombotic occlusions, atherosclerotic calcification, ruptured aneurysms, abnormal blood pressure, and irregular blood flow, associated with irregular dynamics. Understanding underlying CVA etiologies is essential for Medical and Allied Health professionals and their clients to affect prevention and treatment. Occupational Therapy assessment, interventions, and outcome evaluations are vital in the overall rehabilitation process. The focus of Occupational Therapy on Activities of Daily Living (ADL) and Functional Mobility can allow a patient to gain the skills needed to live as independently as possible after the occurrence of a CVA. This project explored the epidemiology, pathogenesis, brain anatomy, symptoms depending on location, risk factors, prevention, emergent treatment, and Occupational Therapy rehabilitation implication for multifarious types of CVA.

Key Words: Cerebrovascular Accident (CVA), Ischemic, Hemorrhagic, Transient Ischemic Attack (TIA), Thrombus, Atherosclerosis, Aneurysm, Arteriovenous Malformations (AVM), Meninges, Cerebrospinal Fluid (CSF), Cranial Nerves, Hemiplegia, Hemiparesis, Aphasia, Penumbra, Occupational Therapy, Activities of Daily Living (ADL), Instrumental Activities of Daily Living (IADL), Functional Mobility

Introduction/Background

The term Cerebrovascular Accident (CVA) is the most common label that is attached to people who have suffered from strokes. CVA is divided into different categories to promote diagnostic precision, as well as identify causes for certain symptoms after a stroke has occurred.[15] The types of CVA, pathogenesis, brain anatomy, symptoms depending on location of the CVA, risk factors, prevention, emergent treatment, and rehabilitation are documented here.

Different Types of Cerebrovascular Accidents

A stroke is defined as an acute disturbance of cerebral function or vascular origin causing disability lasting more than, or death within 24 hours.[15] Strokes are classified according to etiology and are either classified as ischemic or hemorrhagic. Ischemic strokes are caused by sudden interruptions of blood flow to the brain due to acute occlusions of cerebral blood vessels or systematic hypoperfusion. Hemorrhagic strokes occur when a cerebral vessel ruptures and bleeds within the brain.[22] A transient ischemic attack (TIA) is defined as an acute disturbance of cerebral function of vascular origin lasting less than 24 hours. TIA's can signal an impeding stroke.[48]

Ischemic strokes are further classified as either thrombotic or embolic. Thrombotic strokes occur as a result of atherosclerotic plaque formation, which can augment the development of a thrombus resulting in the blockage of blood vessels leading to hypoxia or anoxia of the brain. Embolic strokes occur when a thrombus originating in a distant part of the body, (often within the heart or the large arteries of the chest) breaking off, traveling through the bloodstream into cerebral circulation, and blocking small blood vessels in the brain.[22] Ischemic strokes can result in cerebral infarctions, areas in the brain that becomes necrotic due to lack of

oxygen delivery from occluded blood vessels. The term cerebral infarction includes all thromboembolic disease and accounts for 75% of all CVA's.[15]

Hemorrhagic Strokes are classified as either intracerebral or subarachnoid. Intracerebral hemorrhages follow the rupture of a diseased blood vessel or aneurysm inside the brain leading to the formation of a hematoma in a localized area of the brain. Other causes of intracerebral hemorrhages include chronic hypertension, arteriovenous malformation, and bleeding into or around a brain tumor.[15,22] A subarachnoid hemorrhage means that there is blood in the area between the brain and the tissue that covers it, also known as the subarachnoid space. The primary cause of a subarachnoid hemorrhage is the rupture of aneurysms located at the base of the brain.

Transient ischemic attacks (TIA) have similar symptoms as an ischemic CVA, but the signs and symptoms disappear within 24 hours. They are produced by temporary reductions or loss of blood supply to regions of the brain either due to partial or total obstruction of an artery in the carotid or vertebrobasilar system, or a critical fall in blood pressure. According to the North Carolina Comprehensive Stroke Program, six percent of 1,300 stroke patients experienced at least one TIA before manifesting a frank stroke. It is estimated that 35 percent of patients with a history of TIA will experience a major stroke in a five year time period.[48]

Etiology and Pathogenesis

Ischemic CVA's:

The most common cause of ischemic stroke, known as atherosclerosis is the blockage of blood vessels preventing sufficient oxygen delivery to the brain. Arteriosclerosis is the thickening and hardening of the walls of the arteries that typically develops over time.

Atherosclerosis is a type of arteriosclerosis that is characterized by chronic inflammation and buildup of lipid plaques on the inside of arterial walls.[40]

The first step leading to atherosclerosis is endothelial dysfunction. Low Density Lipoproteins (LDL) pass through the endothelial cells by using receptor mediated endocytosis, however when these endothelial cells become damaged there is an increase in the permeability of the arterial wall.[89] Etiological factors of endothelial cell damage include hypertension, smoking, high blood glucose, and lack of exercise. When it comes to exercise, a sedentary lifestyle can really speed up the process of atherosclerosis. Lack of exercise leads to a decrease in the production of nitric oxide (NO) due to improper function of an enzyme called endothelial nitric oxide synthase (eNOS). When the concentration of NO decreases, blood vessels are less able to vasodilate. A decreased amount in NO, results in adhesive molecules on the endothelial cells, which present to capture monocytes. When the monocytes bind to these adhesive molecules, they tend to flatten and squeeze between the endothelial cells, making their way into blood vessels.[89] Once inside the blood vessels, these monocytes produce free radicals that make contact with LDLs and oxidize them. The oxidized LDL molecules cause an inflammatory response, attracting more monocytes. This cascade results in production of more free radicals that combine with LDL, creating an abundance of oxidized LDL in the blood vessels. This inflammatory response leads to the laying down of collagen and accumulation of smooth muscle cells. [18,40] If monocytes make their way into the blood vessels, they become macrophages and engulf the oxidized LDL particles, leading to the production of foam cells. Foam cells are saturated with LDL particles, and the excessive amount of lipid in the cells gives the cytoplasm a foamy appearance. These foam cells eventually die, release their contents (LDL cholesterol), and are engulfed by other nearby white blood cells. Smooth muscle cells present in the blood vessels also

take up LDL.[18] Eventually, the accumulating lipid, fragments of dead macrophages, and collagen tissue produce an area with the lipid core, that forms a plaque. T endothelial cells then cover the plaque and as the plaque accumulates calcium salts and more dead cells over time, this plaque hardens. This plaque diminishes blood flow to that area of the blood vessel, leading to more cellular necrosis and endothelium pathology. Rupture of the endothelium is facilitated in the presence of Matrix Metalloproteinases (MMPs), which weaken the vascular wall by degrading the extracellular matrix or more angiogenesis of plaque, mediated by the cellular adhesion molecule, integrin. If the endothelial cells over the plaque are compromised or ruptured, a thrombus may form.[18,40]

The mechanism for the formation of a thrombus includes an endothelium formation over the hard plaque which ruptures, exposing the collagen. Platelets come into contact with the collagen, become highly adherent (sticky) resulting in the formation of a platelet plug. Thromboplastin is then released, which stimulates the conversion of a blood clotting protein called prothrombin to the enzyme thrombin. Thrombin then stimulates the conversion of soluble blood clotting protein, fibrinogen, to insoluble fibrin. This fibrin forms a tangled mesh in which red blood cells are entrapped, eventually forming a blood clot, which narrows the artery.[18.40]

Atherosclerosis can ultimately lead to a cerebral infarction when fragments called emboli, break off the original thrombus, travel through the blood stream, and cause an occlusion of the blood vessels leading to the brain. When a blood vessel is blocked, little oxygen is being delivered to the brain and may lead to an ischemic stroke if not removed. If the carotid and vertebral arteries are among the blood vessels blocked, a person is at a very high risk of suffering from a TIA or a stroke because these vessels are the sources for almost all of the blood delivered to and throughout the brain.[73,87]

Other conditions leading to Ischemic Stroke include hypotension (low blood pressure) and arterial spasms (contraction) which cause inadequate flow of blood to the organs. A cerebral vasospasm often occurs 4-10 days after an operation for a subarachnoid hemorrhage, increasing the risk for an ischemic stroke.[73] Pathologies of the heart muscle affecting blood flow throughout the body, can lead to irregular blood flow. An abnormal heart beat such as atrial fibrillation or atrial flutter, recent myocardial infarction, dilated cardiomyopathy, or left atrial or ventricular thrombus can all be causes of Ischemic Stroke.[22]

Hemorrhagic Stroke:

Intracerebral and Subarachnoid Hemorrhages can occur as a result of a variety of exogenic incidents. The three most common causes include ruptured aneurysms, chronic hypertension, and arteriovenous malformations. Substance abuse of cocaine, amphetamine, and alcohol can be etiological factors as well.[22]

Aneurysms are sac- like dilations of blood vessel walls that vary in size. These range in size from as small as a pea to the size of an orange.[56] The cause of an aneurysm is the weakening of a given spot of a blood vessel, and the unremitting force on the wall by the blood pressure. The artery can be weakened by congenital collagenic defects, and blunt or sharp trauma to the external layer or the whole wall of the blood vessel (including internal layers). This can cause blood deposition between the two layers, causing the blood vessel to become distended. Increased inflammation and cardiac irregularity can also contribute to the weakening of an artery. Abnormal amounts of blood passing through the blood vessel due to cardiac problems may cause erosions mediated by friction associated with inflammation as blood is surges through the vessel. This wear and tear is more severe if there are no structures surrounding the blood vessel to protect it. Inflammation can affect the structures surrounding and supporting the blood

vessel, causing it to be more vulnerable to harmful conditions.[45] Most intracranial aneurysms are located near the basilar surface of the skull and half of them arise from the internal carotid or the middle cerebral arteries. Aneurysms are usually found at the junction of main vessels and 85 percent of them are located in the Circle of Willis. Larger aneurysms can erode skull bones, but the smaller aneurysms can also be very severe. Most aneurysms that rupture are located in the subarachnoid space and cause subarachnoid hemorrhages.[56]

Arteriovenous malformations (AVM) can also be causes of hemorrhagic stroke. An AVM occurs when a tangle of blood vessels in the brain or on its surfaces bypass normal brain tissue and directly diverts blood from the arteries to the veins. Most AVM's are congenital and can occur in any of the four lobes of the brain, cerebellum, brainstem, or ventricles. AVM's begin to bleed when weakened blood vessels direct blood away from normal brain tissue. These weakened vessels dilate over time, and may eventually burst from chronic hypertension or increased blood flow. Brain AVM's occur in less than one percent of the general population, but more than 50 percent of patients with an AVM suffer from an intracranial hemorrhage.[29,80] Chason et al[9] reported that out of the 3,000 post mortem examinations of AVM victims, 5% manifested intracranial aneurysms. Their conclusion was that aneurysms and AVM are both co-morbidities in hemorrhagic stroke.[29]

Lifestyles which include use of cocaine, amphetamine, and alcohol can increase the risk of suffering from a hemorrhagic stroke. Cocaine and amphetamine can produce acute intracranial hypertension and both have been reported to be associated with cerebral vasculitis, defined as the inflammation of the walls of the blood vessels in the brain and spinal cord. Additionally, cocaine causes cardiac arrhythmias and cardiac myopathy, which are associated with brain infarction through cardiac embolism. Cerebral vasoconstriction can also occur if a person uses cocaine

consistently.[72] Chronic alcohol consumption and binge drinking pose a risk of ischemic and hemorrhagic stroke at a rate 1.6 to 1.8 times higher than typical. Alcohol contributes to high levels of triglycerides, produce cardiac arrhythmias, and can cause heart failure, all of which increase possibility of having a stroke.[81]

Transient Ischemic Attack

Causes for TIA's are almost the same as the risk factors for both Ischemic and Hemorrhagic strokes. Blocked blood vessels from atherosclerosis and emboli being carried through the blood stream are the main causes. However if these occlusions of the blood vessels are loosened and dismantled within 24 hours, a CVA may be averted. Carotid Artery Disease and temporarily uncontrolled hypertension can also be a cause for TIA's.[48]

Epidemiology

Stroke is the third leading cause of death and the most common cause of permanent disability in adults worldwide.[35,81] About 700,000 people in the United States experience a stroke annually, 500,000 being first time strokes and 200,000 recurrent. Stroke is the cause of mortality for about 140,000 Americans each year, which is the equivalent of 1 out of every 20 deaths. In the United States it is said that every 40 seconds someone suffers from a stroke and every 4 minutes someone dies of a stroke. Ischemic strokes account for 87% of these events while the balance are hemorrhagic.[81] Figure 1 shows that some areas in the United States are much more affected than others. As you can see, most of the deaths due to stroke occur in the southern

region of the country. Stroke risk factors fall into three categories: nonmodifiable factors, treatable medical factors, and alterable lifestyle factors.[81]
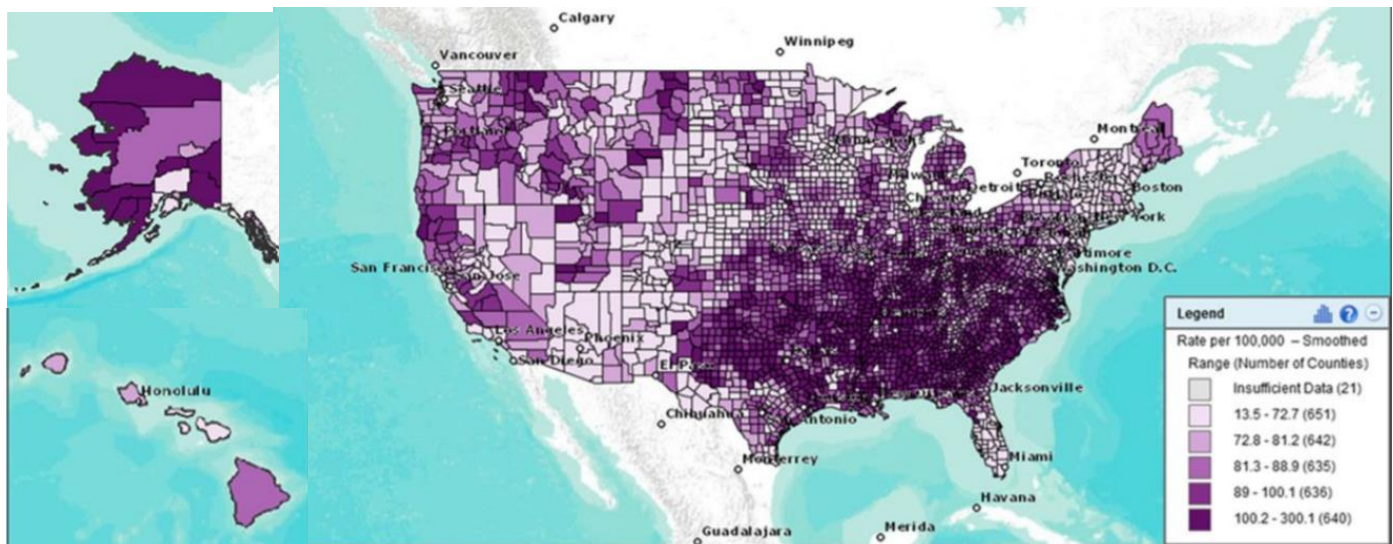
Stroke death rates, 2008 through 2010.

Figure 1. Stroke death rates throughout the United States 2008-2010 [62]

Nonmodifiable Risk Factors

Age

Age is the single most important risk factor for stroke. The incidence of stroke increases significantly with age in both men and women. The risk doubles every ten years after the age of 55 for both genders.[81] Half of all strokes occur in people over the age of 75 and one third in people above the age of 85.[28] The risk of stroke most likely increases with age, because as people get older they are more prone to develop other medical conditions such as hypertension or heart disease that are also risk factors for stroke. Although the risk of stroke increases with age, they can occur at any age. In 2009, 34% of the people that were hospitalized for stroke were under the age of 65.[28] In general, younger stroke patients have better outcomes (lower mortality and higher

functioning level) than older patients because their bodies simply aren't as worn due to the passage of time.[68]

Gender

Although the entire older population is at risk for strokes, there are gender differences in the incidence by age subgroups. On a demographic level, women have a lower risk of stroke than men when matched by age, but a woman's risk is significantly higher after 85 years of age.[35] Stroke incidence rates are 1.25 times greater in men, however more women than men die each year from stroke.[81] It is calculated that the lifetime risk for stroke in women aged 55 was 21% while it was only 17% in men of the same age. This higher lifetime risk of stroke among women is most likely attributed to the longer lifespan of women.[28] Men suffer more often from atherothrombotic strokes, while cardioembolic strokes are more frequent in women. Women are less likely to develop risk factors like ischemic heart disease, vascular peripheral disease, carotid artery stenosis, or aortic atherosclerosis. However, women seem to present more diabetes, hypertension, and dyslipidemia.[35] Figure 2 clearly shows how much the frequency of stroke occurrence really increases with age.
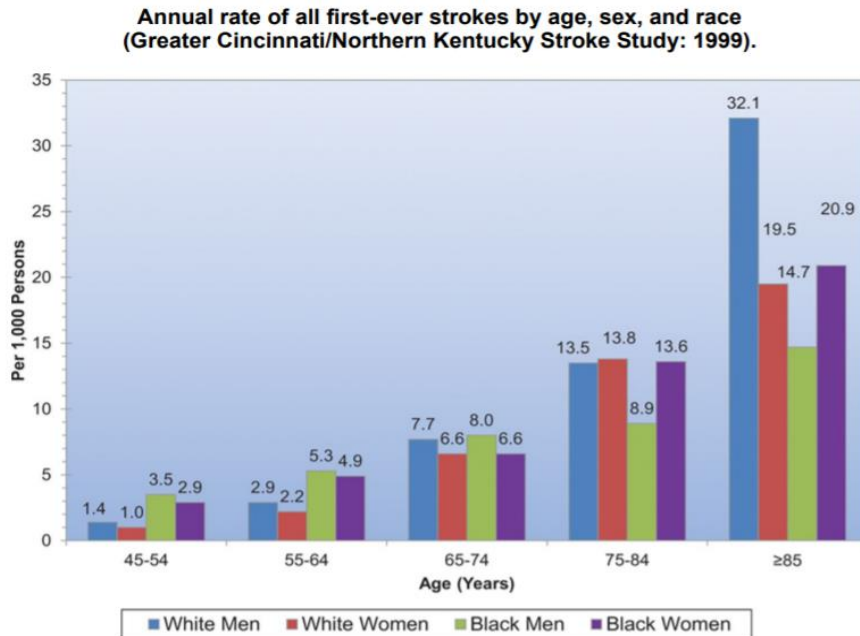
**Annual rate of all first-ever strokes by age, sex, and race
(Greater Cincinnati/Northern Kentucky Stroke Study: 1999).**

Figure 2. Annual occurrence of stroke by age, sex, and race [62]

Ethnicity

Stroke incidence and mortality rates vary widely between racial groups. Blacks between the ages of 18 and 64 are more than twice as likely to die of strokes than whites of the same age. Between the ages of 45-55, mortality rates are four to five times greater for African Americans than for whites. This difference however, decreases with increasing age. Figures 3 and 4 show how the incidence of strokes is much higher in Blacks than other ethnicities. In 1997, stroke death rates for Hispanics and Whites under the age of 65 were very similar, but were lower for Hispanics over the age of 65.[79] Since then, cerebrovascular disease mortality rates for Hispanics have been climbing. As of 2006, Mexican Americans, who make up the largest subgroup of Hispanics, were 20% more likely to have a stroke than non-Hispanic whites. Specifically, they have a much higher incidence of hemorrhagic stroke, which could be due to a higher incidence of diabetes as well as other socioeconomic factors. When it comes to death rates, they are still

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not as high for Hispanics as it is for either Blacks or Whites.[39,81] We see the same trend as Hispanics in the Native American population. Asians, specifically Chinese and Japanese, have high stroke incidence rates. Rates were extremely high all the way from 1900 to 2000, but dramatically dropped after World War II. In recent years, incidence rates have dropped below the rates of all of the other ethnicities. All of these comparisons can be seen in Figure 4.
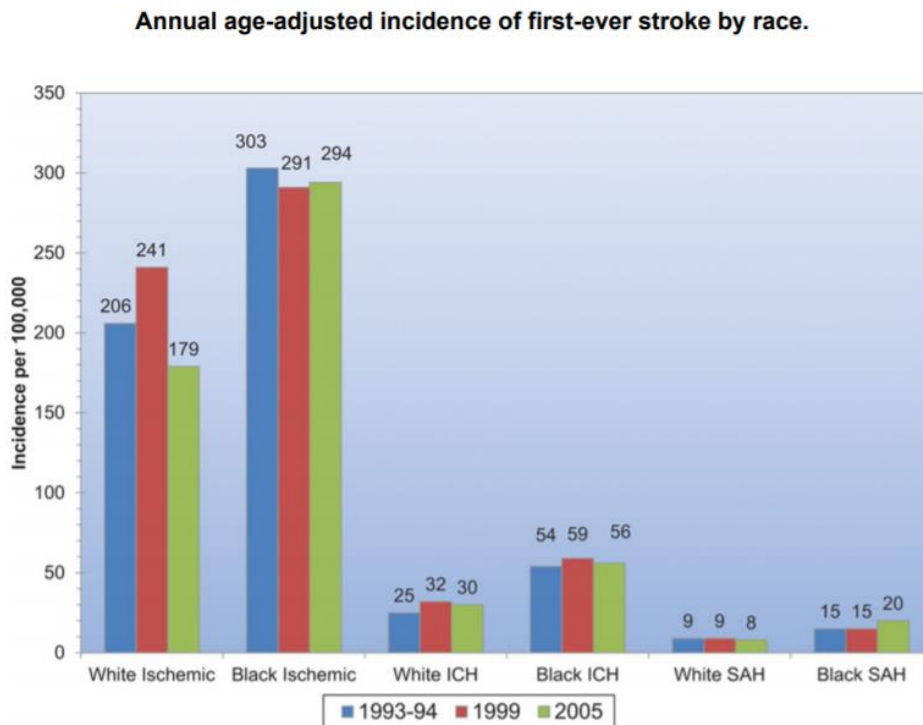
Annual age-adjusted incidence of first-ever stroke by race.

Figure 3. Annual Occurrence of stroke by race [62]

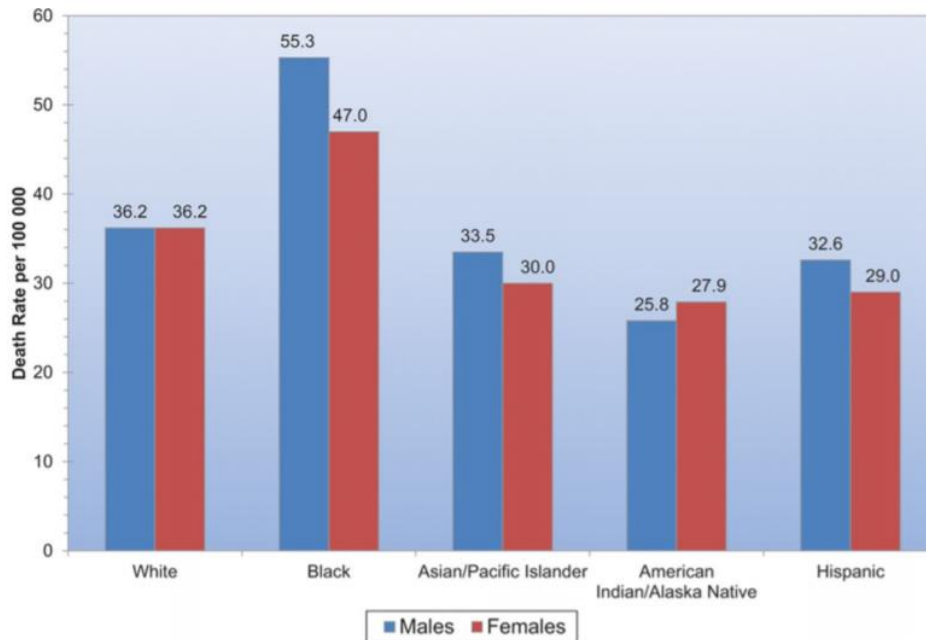**Age-adjusted death rates for stroke by sex and race/ethnicity, 2011.**

Figure 4. Annual Occurrence of stroke by sex and ethnicity [62]

Treatable Medical Risk Factors

Hypertension

Hypertension is the most important modifiable risk factor for stroke. It is defined as a blood pressure of 140/90mmHg or higher for an extended period of time. It is estimated that 77% of all primary strokes are related to chronic hypertension.[79,81] Continuous high blood pressures in the blood vessels can result in damaged endothelium and an increase in atherosclerotic plaque. This increases the likelihood of a thrombus forming and causing an occlusion in the blood vessels. An increased blood pressure also increases the risk of a weakened arterial wall from giving way, producing an intracerebral hemorrhage.[81] The risk of stroke continues to grow with increased hypertension. With age however, the impact of hypertension may decrease. There are much greater odds of having a stroke due to high hypertension at age 50 than at age 90.[79] While

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the impact of hypertension may decrease with age, the prevalence of hypertension does the opposite. For example, the prevalence is about 20% at age 50, but by age 90 the prevalence is 60%. The American Heart Association shows that blacks develop hypertension earlier in life than whites do. In fact, their average blood pressures are much higher than most other populations and may even be the highest in the world.[79,81] After various treatment trials, it has been shown that treatment of hypertension reduces the percentage of all strokes by 38% and fatal strokes by 40%. The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure recommends lifestyle modifications as well as the use of thiazide- type diuretics combined with antihypertensives as a treatment for high blood pressure. These treatments have been effective for all ages and all ethnicities. [79,81]

Cardiac Disease

Cardiac diseases have been shown to increase risk of stroke. Within 6 years of having a myocardial infarction, 8% of men and 11% of women will have a stroke.[81] A myocardial infarction can lead to increased stroke morbidity, including atrial fibrillation. Atrial Fibrillation is the most powerful and treatable cardiac precursor of a cardioembolic stroke. The incidence and prevalence of atrial fibrillation increases with age. Each decade after the age of 55, the incidence of atrial fibrillation doubles. It is estimated that 2.2 million Americans have intermittent or sustained atrial fibrillation.[79] A person with atrial fibrillation has a risk of stroke five times greater than the average individual. This condition is associated with 20% of all ischemic strokes. Studies have shown that anticoagulation therapy with Warfarin is effective in lowering the risk of a stroke occurring among patients who have atrial fibrillation. Warfarin anticoagulation has been shown to reduce the risk of stroke by 68%.[79,81]

Cardiomyopathy is a disease of the heart muscle in which the ability of the heart to pump blood to the rest of the body is severely reduced. Ischemic Cardiomyopathy is characterized by insufficient blood flow to and through the heart muscle due to constriction of blood vessels often caused by hypertension. The heart muscle tissue is weakened do to ischemia which may lead to myocardial infarction. When the heart is ischemic it is possible that the brain is hypoxic as well.[79,81]

Left Atrial Enlargement (LAE) may be a risk factor for stroke. In the Framingham Study, a 10mm enlargement of the left atrium doubled the risk of stroke for both men and women. Even after adjusting for other risk factors and the use of hypertensive medications the excess risk for cardiovascular mortality persisted in men, appearing mostly to be due to a large LA size.[79]

Valvular Heart Disease can also be a risk factor for strokes. Valvular disease manifests as mitral stenosis, mitral annular calcification, and valvular strands. Mitral stenosis is the narrowing of the mitral valve that blocks blood flow from the left atrium to the left ventricle. If the mitral valve fails to function, blood regurgitates from the left ventricle to the left atrium. This can contribute to a 4-5% increase in the chance of a stroke. Mitral Annular Calcification is a chronic process in which there is deposition of calcium in the mitral valve annulus. The annulus is the structure that separates the superior and inferior chambers of the left side of the heart. Usually, it is very flexible and provides support to the mitral valve, but as it becomes hardened by calcium it puts increased stress on the heart. This disease occurs in approximately 10% of men and 16% of women. Valvular Strands are filamentous, threadlike processes that are attached to the mitral and aortic valves. Two studies report some type of linkage between the strands and ischemic stroke, but more research is required.[79]

Some treatments for all of these different cardiac diseases include cardiac catheterization and angioplasty. Surgery greatly reduces the risk of stroke occurrence, however intracardiac devices may be complicated by a thrombus or an infection resulting in an embolism. Methods such as radiofrequency and pacing have also been known to cause complications leading to an embolism.[79,81]

Diabetes Mellitus, Hyperlipidemia, and Carotid Stenosis

A person with diabetes is 1.8 to 6 times more likely to have an ischemic stroke than a person without diabetes. Among those with diabetes, there is an increased incidence of ischemic strokes, but not hemorrhagic strokes. Women with diabetes requiring insulin therapy have been shown to be at greater risk than female diabetics not using insulin.[84] Diabetes and hyperlipidemia contribute to endothelial cell dysfunction, which accelerates atherosclerotic plaque formation. Plaques are much more common in the smaller branches of cerebral arteries in patients who have diabetes. Hyperlipidemia contributes to large buildup of cholesterol (LDL) and plaque in these arteries, contributing to a higher risk for carotid stenosis and atherosclerosis. When a carotid artery is narrowed by 70% or more due to a buildup of plaque, a person's risk for stroke is doubled.[81]

Transient Ischemic Attack

TIA's are often significant indicators of stroke risk. Approximately 10% to 20% of patients diagnosed with a transient ischemic attack have a stroke within 90 days. Fifteen percent of patients with stroke have had a TIA in the past.[81] Recent studies have shown that prompt assessment and treatment after TIA can substantially reduce the risk of early recurrent stroke.

The National Institute for Health and Clinical Excellence (NICE) state that it is best for high risk patients to be seen within 24 hours of symptom onset.[52]

Lifestyle Risk Factors and Prevention of Stroke

There are several different lifestyles that can definitely increase the chance of a person having a stroke. Eliminating these practices, can help with the overall prevention.

Smoking

Of the potentially modifiable lifestyle risk factors, smoking is associated with the greatest excess incidence of coronary heart disease and stroke. People with severe diabetes requiring insulin therapy are most vulnerable to morbidity.[84] Cigarette smoking has been shown to approximately double the risk of stroke and other risk factors. The toxic compounds in cigarette smoke, including carbon monoxide and nicotine, accelerate atherosclerosis. Smoking increases the adhesiveness and clustering of platelets, faster clotting time, and greater viscosity. After a person stops smoking, the risk of stroke steadily drops over time. After smoking cessation, a person can reach the level of risk that a non-smoker has after about 5 to15 years.[81]
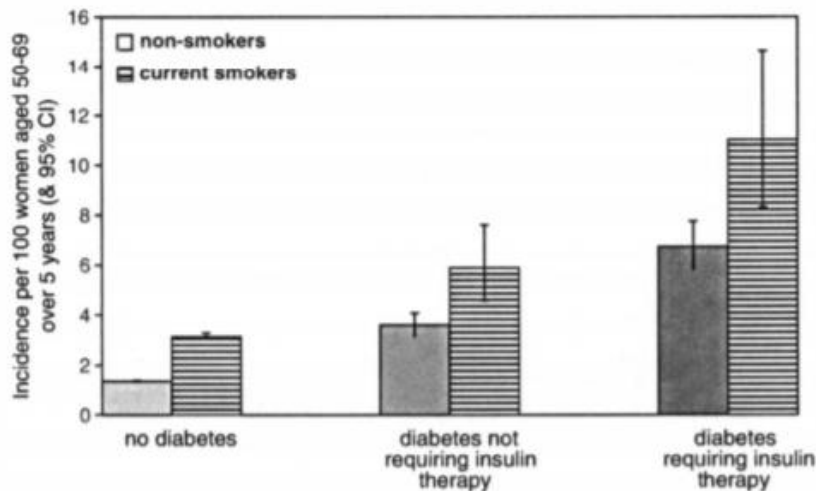

Figure. 5 The relationship between smoking and diabetes [84]

Alcohol Abuse

It has been shown that the incidence of ischemic stroke in those who consume small amounts of alcohol (1-2 drinks per day for men and 1 per day for nonpregnant women) is lower than people who don't consume any alcohol at all. This is because moderate alcohol consumption increases HDL cholesterol levels and produces endogenous tissue plasminogen activator, both of which reduce the risk of coronary artery disease and atherosclerosis. However, if alcohol consumption is excessive, it may lead to a higher incidence of stroke. People who chronically drink too much or binge drink have a risk of ischemic stroke 1.6 to 1.8 times higher than those who don't.[81] Increased alcohol consumption increased the mean red cell volume, gamma glutamyl transferase activities, high levels of triglycerides. Increased alcohol consumption not only increases the risk for strokes, but can lead to other conditions such as cardiac arrhythmias and heart failure.[33,81]

Obesity and Physical Inactivity

When it comes to obesity, if a person has a BMI of 30 or greater this can be connected to several risk factors for stroke, including hypertension, diabetes, and dyslipidemia.[81] Studies have shown that women with diabetes tend to have a higher BMI than women without diabetes (46% vs 17% obese). If diabetic females are inactive, they will increase in fat accumulation, which results in higher BMI.[84] The American Stroke Association recommends a BMI between 18.5 and 24.9 in order to reduce the overall risk of stroke. Obesity can be reduced with an increase of physical activity. Increasing the level of moderate to vigorous exercise to an average of 30 minutes per day can reduce stroke risk.[81] Exercise is something that is very beneficial to keeping our bodies as healthy as possible, no matter the age. Controlling obesity, diabetes, and Atherosclerosis reduces the risk of stroke. In the case of Atherosclerosis, if a person is physically

active, then there is less cholesterol (LDL) for the macrophages to engulf, decreasing the production of foam cells, in turn decreasing the amount of plaque that builds up on the walls of the blood vessels. Exercise decreases a protein called tissue factor, as well as integrin, which both increases the likelihood of a blood vessel getting blocked. There is less hypertension in the blood vessels due to a lower amount of plaque buildup and MMPs behaving in a beneficial way by consuming scar tissue. This low blood pressure can last for up to 8 hours after exercising. If there is less plaque accumulation and a lower blood pressure then blood clots are less likely to form.[18.40,89] Decreased physical activity is more likely to be found in women, blacks, Hispanics, older adults, and people of low socioeconomic status, which is why these groups have a higher chance for suffering from a stroke.[81]

Brain and Cranial Cavity Anatomy with Gross Anatomical Structures

Cranial Meninges

The Meninges are membranous coverings of the brain and spinal cord. The Cranial Meninges lie deep to the cranium and the scalp. The purpose of these meninges is to protect the brain, form the supporting framework for arteries, veins, and venous sinuses, and to enclose a fluid-filled cavity that is vital to the normal function of the brain. The meninges are composes of three membranous connective tissue layers: The Dura Mater, Arachnoid Mater, and the Pia Mater.[16,60]

Dura Mater

The outermost membrane of the meninges. This is a tough, thick, fibrous layer with a bilaminar membrane. It can also be called the pachymeninx. Most of the blood that flows

through the arteries of the Dura goes to the calvaria, the skull cap, then to the dura itself. The Dura Mater has two layers: the external periosteal layer and the internal meningeal layer.

Periosteal Layer

Adheres to the internal surface of the cranium along the suture lines and in the cranial base. This external layer is continuous with the periosteum on the external surface of the calvaria, but is not continuous with the dura mater of the spinal cord.

Meningeal Layer

The supporting layer that reflects away from the external periosteal layer of the Dura Mater to form dural infoldings. These dural infoldings divide the cranial cavity into compartments, forming partial partitions between certain parts of the brain and support for other parts. The Dural Infoldings include the Falx Cerebri that separates the left and right cerebral hemispheres, the Tentorium Cerebelli that separates the occipital lobes from the cerebral hemispheres of the cerebellum, the Falx Cerebelli that lies inferior to the tentorium cerebelli that partially separates the cerebellar hemispheres, and the Diaphrgma Sellae that forms a partial roof over the hypophysial fossa in the sphenoid and covers the Pituitary gland.[60]
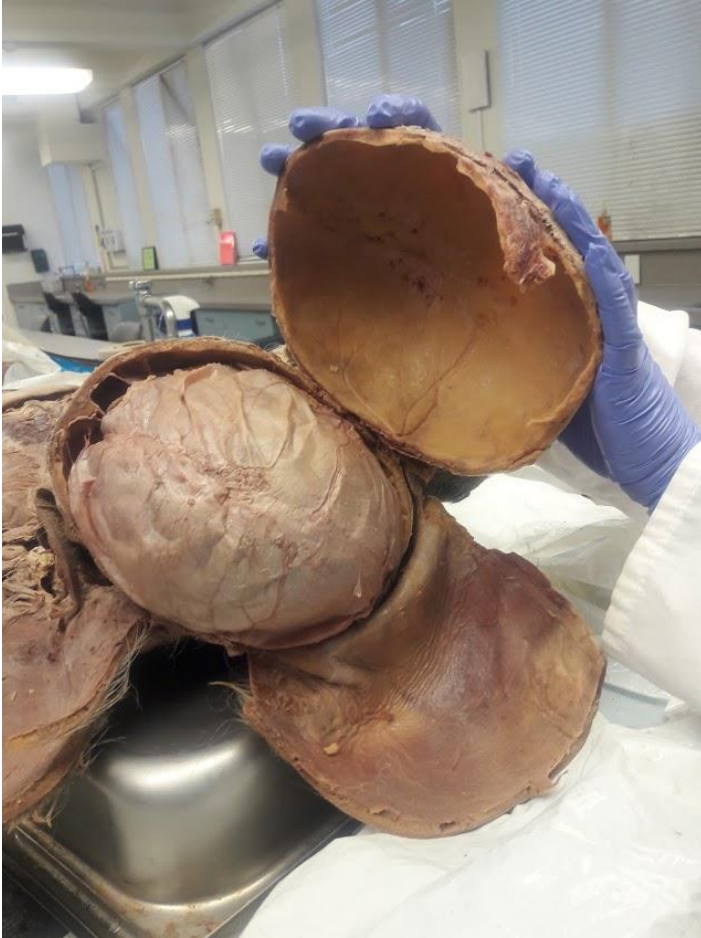
Figure. 6 Cadaveric Dissection of the Dura Mater

Arachnoid and Pia Mater Layers

The Arachnoid Mater is a thin, transparent membrane named for its spiderweb- like appearance. It is closely applied to the deep surface of the dura mater, held by the pressure of the cerebrospinal fluid in the subarachnoid space, but is not directly attached. The arachnoid is composed of collagen and elastic fibers and has variable thickness due to several different cell layers. The superficial layer of the arachnoid consists of closely packed electron-lucent cells that are impermeable to cerebrospinal fluid due to tight intercellular junctions. This is why there is no cerebrospinal fluid directly inferior the dura mater. The collagenous innermost portion of the of the arachnoid mater is comprised of loosely arranged cells anchored by desmosomes. Below this

is the subarachnoid space in which the cerebrospinal fluid is located.[46,60] The cerebrospinal fluid

is clear and colorless, and contains lymphocytes, sugar, and proteins. This fluid flows through

the ventricles of the brain and protects the brain and spinal cord by acting as a cushion in the

case of blunt trauma.[16] Deep to the subarachnoid space is the pia mater. This is a very thin

membrane that is highly vascularized by a network of fine blood vessels. This layer is difficult to

see, but it gives the surface of the brain a glossy appearance. The Arachnoid and Pia Mater layers

are known as the leptomeninges and most physicians refer to them together as the pia-
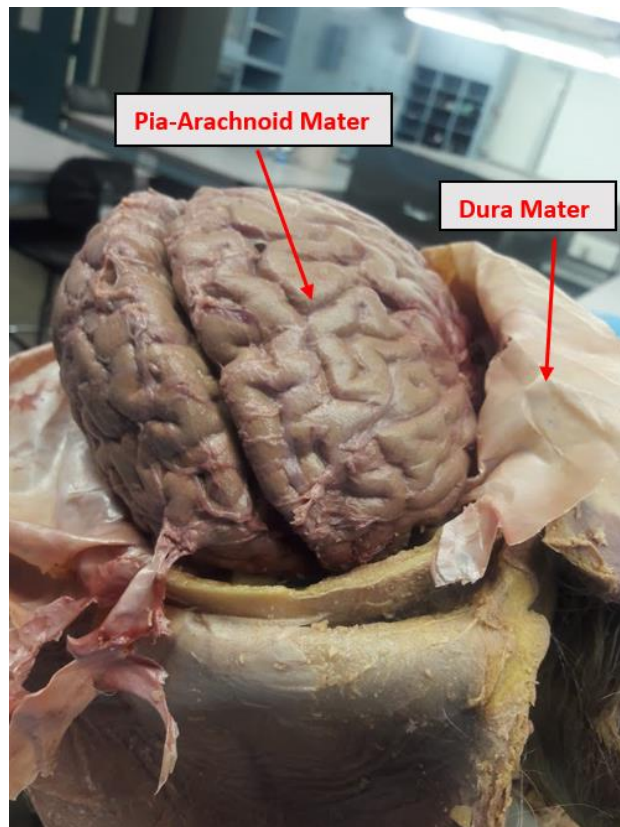
arachnoid.[16,60]

Figure. 7 Cadaveric Dissection of the Meninges

Exterior Anatomy of the Brain

In the human adult, the brain weighs approximately three pounds and has diverse subdivisions that control the various aspects of mind-body functions. The three main parts of the brain are the cerebrum, the cerebellum, and the brainstem.[16]

Cerebrum

The largest part of the brain that is known as the "thinking area". On the surface of the cerebrum, nerve cells lie in sheets and make up the cerebral cortex. These sheets are arranged in folds called gyri, which are separated from each other by grooves called sulci. Larger clefts in the brain are called fissures. When the calvaria and the dura mater are removed, the gyri, fissures, and sulci are visible through the delicate arachnoid-pia layer. The brain is divided into left and right halves called cerebral hemispheres.[16] The left and right hemispheres are separated by the falx cerebri within the longitudinal cerebral fissure. Each hemisphere is also subdivided into four major lobes: frontal (I), parietal (II), occipital (III), and temporal (IV), named for the cranial bones that overlie them. The frontal lobes are separated from the parietal lobes by the central sulcus, the parietal lobes are separated from the temporal lobes by the lateral sulcus, and the occipital lobes are separated from the temporal and parietal lobes by the parieto-occipital sulcus.[60] Each lobe of the brain has unique and individual functions. For example, the frontal lobe is the area of the brain that is responsible for thought processes, behavior, personality, emotion, language expression and body movement. The Parietal lobe is responsible for body sensations as well as visual and spatial perception. The Temporal lobe is responsible for hearing, understanding, speech, language, and language comprehension. Finally, the occipital lobe is responsible for vision.[16]

Brain Stem

The brainstem is divided into three different sections: the pons, midbrain, and medulla oblongata. The pons functions as a bridge. It contains nerve fibers that connect the cerebellum and cerebrum to the rest of the brain. The midbrain is located superior to the pons and serves important functions for the motor movements of the eye, as well as auditory and visual processing. It is composed of two sections called the tectum and the tegmentum. The final part of the brainstem is the Medulla Oblongata, which is the section that is continuous with the spinal cord. This is where nerve tracts cross from the left and right, causing movements and actions of one side of the body to be controlled by the opposite hemisphere of the brain. The medulla oblongata contains three vital centers that regulate the internal activities of the body: respiratory center (breathing), cardiac center (heartbeat), and the vasomotor center (blood pressure).[16,60]

Cerebellum

Located posterior to the pons and medulla oblongata and inferior to the posterior part to the cerebrum. It functions to coordinate voluntary movements and to maintain balance and posture. It consists of two lateral hemispheres that are united by a narrow middle portion called the vermis.[16,60]
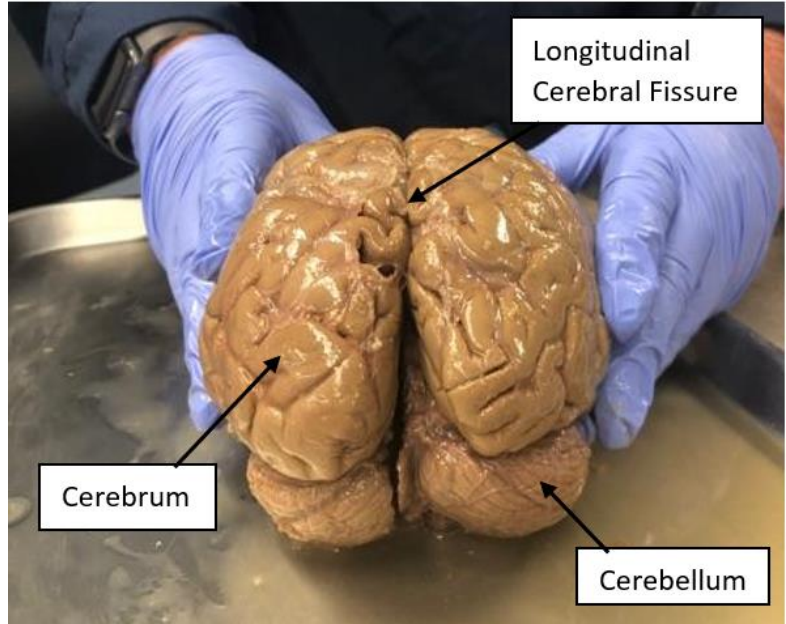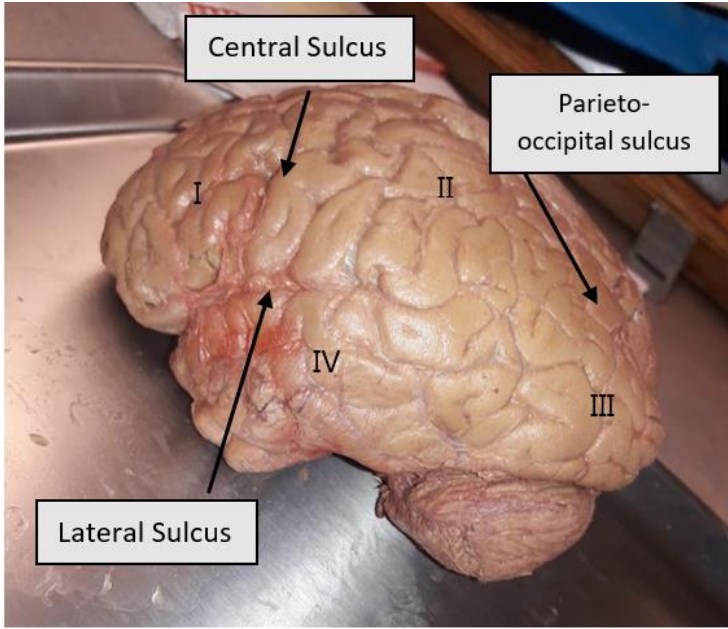
Figure. 8 Cadaveric Dissection of External Brain Anatomy

Figure. 9 Cadaveric Dissection of the Brain Stem

Interior Anatomy of the Brain

Thalamus and Massa Intermedia

See Fig. 10 below. A subcortical structure that sits within the brain at the level of the left temporal lobe. The thalamus is comprised of three different parts: two thalamic bodies and a tissue called the Massa Intermedia that connects them. The two thalamic bodies are separated by a space called the third ventricle, which is filled with cerebrospinal fluid. This is where the Massa Intermedia is located. The Thalamus is responsible for organizing sensory information from the periphery. Messages from all different sensory modalities, except for smell, pass through the thalamus on their way to cortical centers and other structures for further processing.[16,57]

Hypothalamus

Mainly responsible for motivational behavior which allows the human body to satisfy its physiological needs . It is responsible for behaviors such as hunger and thirst, as well as the maintenance of body temperature. This part of the brain also controls our pituitary gland, which is the gland that controls all of the other endocrine glands in the body. The hypothalamus helps to connect the endocrine system with the nervous system.[16,60]

Pineal Body

A small organ located near the center of the brain that is responsible for the secretion of the hormone, melatonin. This hormone's secretion is dictated by light, so melatonin secretion is low during daylight hours and high during the night. This aids in maintaining circadian rhythm, the 24 hour biological cycle of sleep-wake patterns.[16]

Choroid Plexus

A network of blood vessels and cells in the ventricles of the brain. The choroid plexus has two main functions, necessary for proper brain development and protection against harmful substances and microbes. First, choroid plexus ependymal cells produce cerebrospinal fluid (CSF), which fills the cerebral ventricles, the central canal of the spinal cord, and the subarachnoid space of the meninges. The CSF provides cushion and support for the brain and spinal cord, circulates nutrients, and removes waste from the central nervous system. Underproduction of CSF may stunt brain growth, and overproduction can lead to an excess of CSF accumulation in the ventricles, a condition known as hydrocephalus. The second main function of the choroid plexus is that it forms a barrier, along with the arachnoid membrane of the meninges between blood and the CSF. This barrier serves to prevent harmful substances in the blood from entering the CSF and causing damage to structures of the central nervous system.[8]

The Limbic Structures Inside the Brain

These structures make up the oldest and most primitive part of the brain. This system mainly focuses on emotions, memories, the creation of new memories, and has an area dedicated to the processing of olfactory stimuli.[57]

Corpus Callosum

A large C-shaped nerve fiber bundle that is found beneath the cerebral cortex. It stretches across the midline of the brain, connecting the left and right cerebral hemispheres. It helps to carry information received from one hemisphere of the brain over to the other for total brain

functioning as a whole. It also makes up the largest collection of white matter tissue in the brain.[32]

Fornix

A bundle of white matter fibers that arches around the thalamus and towards the front of the brain. It originates in the hippocampus, emerging from fibers called fimbria. When it reaches the anterior commissure, it branches downwards. Many of the fibers terminate after this, but some enter the hypothalamus and form connections with mamillary bodies. The fornix acts as the primary outgoing pathway of the hippocampus, so its' most important function is involvement in memory. Damage to the fornix can lead to memory deficits. In fact, neurodegeneration of the fornix has been associated with the cognitive impairment seen in Alzheimer's Disease.[32]

Septum Pellucidum

It is located in the midline of the brain between the two cerebral hemispheres. It is attached superiorly to the corpus collosum and is attached inferiorly to the fornix. Absence of the Septum Pellucidum can result in an abnormal development of the optic disk and pituitary deficiencies, also known as septo-optic dysplasia. Some symptoms of this are low muscle tone, hormonal imbalance, and seizures.[16]

Amygdala

The amygdala is responsible for the response and memory of emotions, especially fear. The amygdala is the reason that people are afraid of things outside of their control. It controls the way a person reacts to certain stimuli or an event that can be perceived as threatening or dangerous.[16,60]

Hippocampus

This structure assists with the storage of long term memories, creating new memories, and is responsible for the memory of the location of objects or people. Alzheimer's disease often affects the Hippocampus initially and severely before other parts of the cortex, so this is why memory is one of the first things to be influenced as the disease initiates.[16]
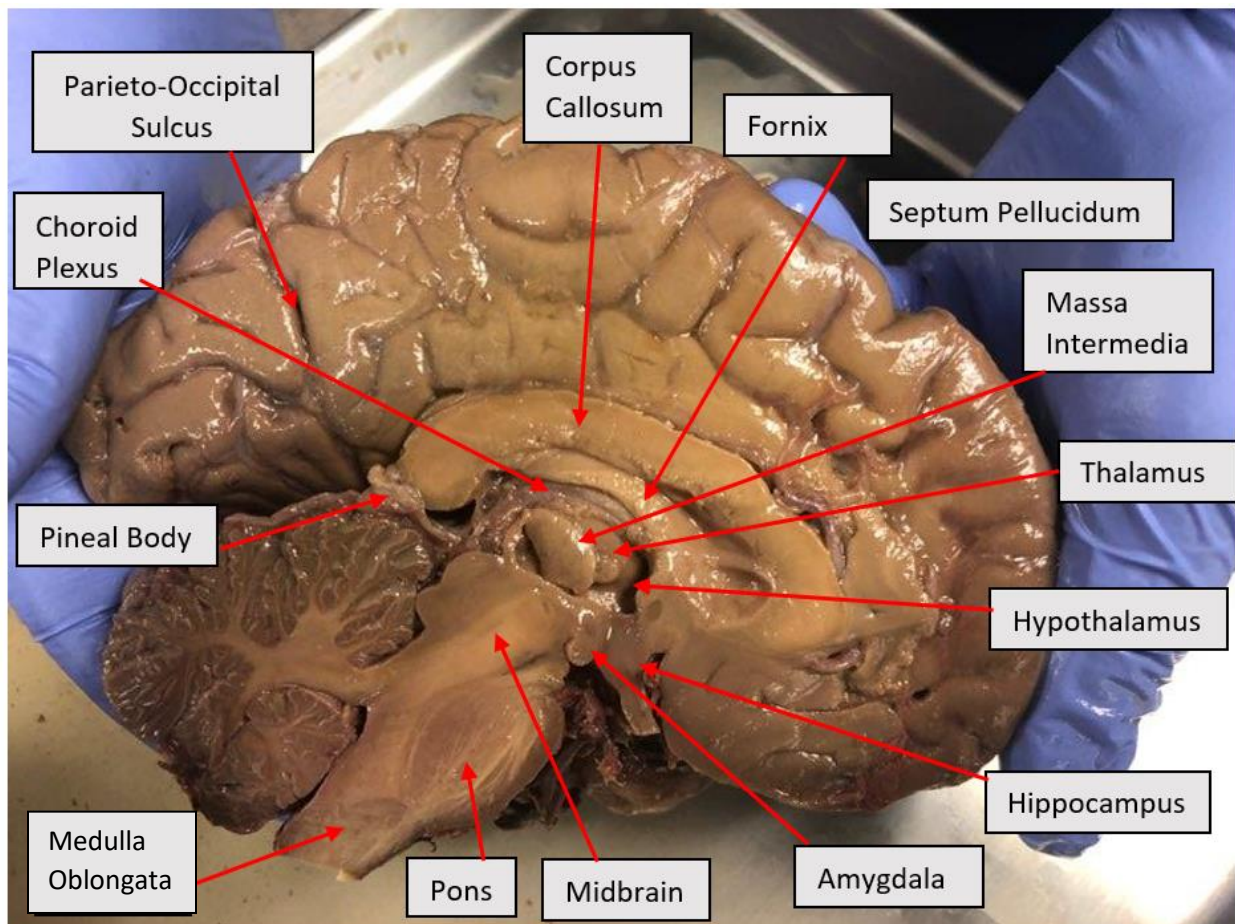

Figure. 10 Cadaveric Dissection of the Interior of the Brain

Cranial Nerves

Cranial nerves are bundles of sensory and motor fibers that innervate muscles or glands, carry impulses from sensory receptors, or have a combination of motor and sensory fibers. These nerves emerge through foramina or fissures in the cranium and exit from the cranial cavity. They

are covered by tubular sheaths that are derived from the cranial meninges. There are 12 pairs of cranial nerves, numbered rostral to caudal and named for their general distribution and function. The functions of the cranial nerves either come from motor (efferent) fibers or sensory (afferent) fibers When it comes to the efferent fibers, these can help control voluntary or striated muscle as well as innervate I involuntary or smooth muscles and glands. Afferent fibers can help to transmit general sensations from skin and mucus membranes, can convey sensation from the viscera, and transmit unique sensations like taste and smell.[2,60] See Fig. 11 below.

Cranial Nerve I: Olfactory Nerve

The olfactory nerve is a special sensory (afferent) nerve that is responsible for the detection of the sensation of odors that results from the detection of odorous substances aerosolized from the environment. This is more commonly referred to the special sense of smell. This is the shortest cranial nerve, and it doesn't join with the brainstem. Additionally, it is unmyelinated and covered by Schwann Cells. The olfactory receptor neurons are in the olfactory epithelium, also known as the olfactory mucosa, which is located on the roof of the nasal cavity. The central processes of the olfactory receptor neurons ascend through the foramina in the cribriform plate of the ethmoid to reach the olfactory bulbs in the anterior cranial fossa. These nerves synapse with the dendrites of large mitral cells and their axons in the olfactory bulbs to then create an olfactory tract. These tracts pass this sensation along to the primary olfactory cortex within the temporal lobe to then be interpreted into smell.[2,60]

Cranial Nerve II: Optic Nerve

The optic nerve is a special somatic afferent nerve that is responsible for the special sense of vision. This nerve carries sensory nerve impulses from more than one million ganglion cells of

the retina towards the visual centers of the brain. The optic nerve begins at the optic disk that forms through the convergence of ganglion cell axons as they pass out of the eye. When the nerve emerges from the back of the eye, it passes through the eye socket, through the optic canal, and emerges intracranially on the underside of the front of the brain. After this, the optic nerve from each eye comes together and forms an X shaped structure called the optic chiasm. In the chiasm, approximately one half of the nerve fibers in each eye continue on the same side of the brain. The remaining nerve fibers cross over at the chiasm to join fibers from the opposite eye on the other side of the brain. This is essential for producing binocular vision.[4]

Cranial Nerve III: Oculomotor Nerve

The Oculomotor nerve is a somatic motor and visceral motor nerve. The somatic motor component plays a major role in controlling the muscles responsible for the precise movements of the eyes for visual tracking and fixation on an object. The somatic component innervated four different extraocular muscles for this purpose. The four muscles are the Ipsilateral Inferior Rectus Muscle, which is responsible for the depression of the eye, the Ipsilateral Inferior Oblique Muscle, responsible for the extortion of the eye, the Ipsilateral Medial Rectus Muscle, responsible for the adduction of the eye, and the Contralateral Superior Rectus Muscle, responsible for elevation of the eye. The visceral motor component is involved in the pupillary light and accommodation reflexes. To do this, presynaptic parasympathetic fibers are sent to the ciliary ganglion, leading to the innervation of the ciliary body and sphincter pupillae. These nerves originate in the brainstem and run in the lateral wall of the cavernous sinus. They then enter the orbit through the superior orbital fissures and divide into superior and inferior branches which reach different parts of the eye.[2,60]

Cranial Nerve IV: Trochlear Nerve

The trochlear nerve is a somatic motor nerve and the proprioceptive to one extraocular muscle: the Superior Oblique Muscle, which is responsible for the depression, abduction, and inward rotation of the eye. The trochlear nerves emerge for the posterior aspect of the brainstem. They then run a long intracranial course, passing around the brainstem, entering the dura mater in the free edge of the tentorium cerebelli. The nerves then run in the lateral wall of the cavernous sinus, making contact with the superior oblique through superior orbital fissures.[2,16,60]

Cranial Nerve V: Trigeminal Nerve

The Trigeminal nerve is the largest and most complex cranial nerve and has motor, sensory, and parasympathetic functions of its terminal branches. This nerve has three terminal branches that supply sensations to the skin, mucous membranes, and sinuses of the face. The mandibular branch of the nerve innervates the muscles of mastication and contains proprioceptive fibers. Post- ganglionic neurons of parasympathetic ganglia travel with the branches of the Trigeminal Nerve, which is why this nerve is closely connected with the parasympathetic nervous system. The trigeminal nerve originates from three sensory nuclei and one motor nucleus, extending from the midbrain to the medulla. At the level of the pons in the brain, these sensory nuclei merge to form a sensory root. The motor nucleus continues to form a motor root. These roots are analogous to the dorsal and ventral roots of the spinal cord. After this, the sensory root expands into the trigeminal ganglion, located in the cavernous sinus. The trigeminal ganglion then gives rise to three different divisions: the Ophthalmic Nerve, Maxillary Nerve, and Mandibular Nerve.[2,60,71]

Ophthalmic Nerve (V₁)

Gives rise to three terminal branches: frontal, lacrimal, and nasociliary. These terminal branches innervate the skin and mucous membranes in the areas of the forehead, scalp, frontal sinus, ethmoid sinus, upper eyelids, conjunctiva, cornea, and dorsum of the nose.[60,71]

Maxillary Nerve (V₂)

The Maxillary Nerve gives rise to 14 terminal branches. These branches innervate the derivatives of the maxillary prominence of the 1ˢᵗ pharyngeal arch. These areas include the lower eyelid, conjunctiva, cheeks, maxillary sinus, nasal cavity, lateral nose, upper lip, upper teeth and gums, and the superior palate. This division of Cranial Nerve V helps parasympathetic supply when the zygomatic branch joins with the lacrimal branch of the Ophthalmic Nerve (V₁) to help to stimulate the lacrimal gland. Nasal glands can also be stimulated when parasympathetic fibers are carried to the mucous glands of the nasal mucosa.[60,71]

Mandibular Nerve (V₃)

The Mandibular Nerve gives rise to four terminal branches in the infra-temporal fossa. These four branches are the Buccal, Inferior Alveolar, Auriculotemporal, and Lingual Nerves. V₃ innervates the derivatives of the mandibular prominence of the 1ˢᵗ pharyngeal arch. These areas include the floor of the oral cavity, external ear, lower lip, chin, anterior 2/3 of the tongue, and lower teeth and gums. This is the only division of Cranial Neve V that conveys somatic motor fibers, distributed to the striated muscle derived from the mandibular prominence mesoderm, primarily the muscles of mastication.[60,71]

Cranial Nerve VI: The Abducens Nerve

The Abducent Nerve has one somatic motor (general somatic efferent) component. This component innervates the lateral rectus muscle, which is one of the six extraocular muscles responsible for the precise movement of the eye for visual tracking or fixation on an object. The lateral rectus muscle is responsible for the abduction of the eyeball. The abducens nerves originate from the pons, pierce the dura mater, go across the cavernous sinus, and superior orbital fissures, to enter the orbits.[2,60]

Cranial Nerve VII: The Facial Nerve

The facial nerve has both sensory and motor functions. It is responsible for both general somatic sensory functions, as well as special sensory functions, such as taste. There are also somatic motor and visceral motor aspects in which the nerve carries proprioceptive fibers from the muscles it innervates. The facial nerve emerges from the junction of the pons and the medulla as two divisions: the motor root and the intermediate nerve. The motor root innervates the muscles of facial expression and the intermediate root carries taste, parasympathetic, and somatic sensory fibers. Cranial Nerve VII travels to the internal acoustic meatus, and proceeds anteriorly towards the temporal bone. After this, it turns abruptly along the medial wall of the tympanic cavity. This sharp turn is the site of the sensory ganglion for CN VII, the geniculate ganglion. After this, the cranial nerve emerges from the cranium through the stylomastoid foramen and enters the parotid gland that forms the parotid plexus. The parotid plexus gives rise to five terminal motor branches: temporal, zygomatic, buccal, marginal mandibular, and cervical.[2,60]

Cranial Nerve VIII: The Vestibulocochlear Nerve

The Vestibulocochlear Nerve is a special sensory nerve that is comprised of two parts: vestibular fibers and cochlear fibers. These fibers are concerned with the special senses of hearing, equilibrium, and motion. They arise from the vestibular nuclei complex in the pons and medulla while the cochlear fibers arise from the ventral and dorsal cochlear nuclei in the inferior cerebellar peduncle. These fibers run through the internal acoustic meatus and divide into the cochlear and vestibular nerves. The cochlear nerve is sensory to the spiral organ, for the sense of hearing. Inner hair cells of the spiral organ activate ion channels in response to vibrations of the basilar membrane. Action potentials travel from the spiral organ to the cell bodies of the neurons of the cochlear nerve. The vestibular nerve is sensory to the cristae of the ampullae of the semicircular ducts and the maculae of the saccule and utricle. Vestibular hair cells, located on the otolith organs (utricle and saccule) detect linear movements of the head and the three semicircular canals detect rotational movements of the head. Information about the position of the head is used to coordinate balance and equilibrium.[2,60]

Cranial Nerve IX: The Glossopharyngeal Nerve

The Glossopharyngeal Nerve is a somatic, special, and visceral sensory nerve and originates in the Medulla Oblongata of the brain. It emerges from the anterior aspect of the medulla, moving laterally to the posterior cranial fossa. The nerve leaves the cranium through the jugular foramen. The somatic sensory fibers are sent to the stylopharyngeus, which is a muscle in the head that reaches the oropharynx and the tongue. The visceral motor fibers reach the otic ganglion for innervation of the parotid gland. CN IX also sends sensory fibers to the posterior third of the tongue, pharynx, tympanic cavity, pharyngotympanic cavity, carotid body, and sinus.[2,60]

Cranial Nerve X: The Vagus Nerve

The Vagus Nerve is a functionally diverse nerve that offers many different modalities of innervation. This nerve originates from the medulla of the brainstem and exits the cranium through the jugular foramen, along with the Glossopharyngeal and Accessory Nerves. The Vagus Nerve supplies motor fibers to the voluntary muscles of the larynx and the superior esophagus. Also, visceral motor fibers innervate the involuntary muscles and glands of the tracheobronchial tree and the esophagus, to the heart, and to the alimentary tract. The Vagus Nerve also sends sensory fibers to the pharynx, larynx and reflex afferents from the same areas. The Vagus Nerve also relays parasympathetic fibers to the heart, which can slow down the overall heartbeat. Because of the relationship to the heart, disorders of the Vagus Nerve can be very dangerous. On the other hand, stimulation of the Vagus Nerve has been shown to be potentially useful in different disorders, including epilepsy.[2,60]

Cranial Nerve XI: The Spinal Accessory Nerve

The Spinal Accessory Nerve has a somatic motor function, innervating the striated sternocleidomastoid and trapezius muscles. These nerves arise as rootlets from the sides of the spinal cord in the superior cervical segments. They then ascend into the cranial cavity through the foramen magnum and exit through the jugular foramina. Once the nerve is outside of the cranium, the spinal nerve descends along the internal carotid artery to reach the sternocleidomastoid muscle, which is then innervated. The nerve then goes across the posterior triangle of the neck to supply the motor fibers of the trapezius.[2,60]

Cranial Nerve XII: The Hypoglossal Nerve

The Hypoglossal Nerve has a somatic motor function, innervating the intrinsic and extrinsic muscles of the tongue. These muscles include the styloglossus, the hyoglossus, and the genioglossus, but not the palatoglossus, which is a palatine muscle. The hypoglossal nerve arises in the medulla oblongata, passes laterally across the posterior cranial fossa, and exits the cranium through the hypoglossal canal. There are three branches of the hypoglossal nerve: the meningeal branch, the superior root of the ansa cervicalis, and the linguinal branch. The meningeal branch innervates the dura mater on the floor and the posterior wall of the posterior cranial fossa. The superior root of the ansa cervicalis only conveys fibers from the cervical plexus that join the nerve outside of the cranial cavity. The lingual branch actually supplies the styloglossus, hyoglossus, genioglossus, and intrinsic muscles of the tongue.[2,60]
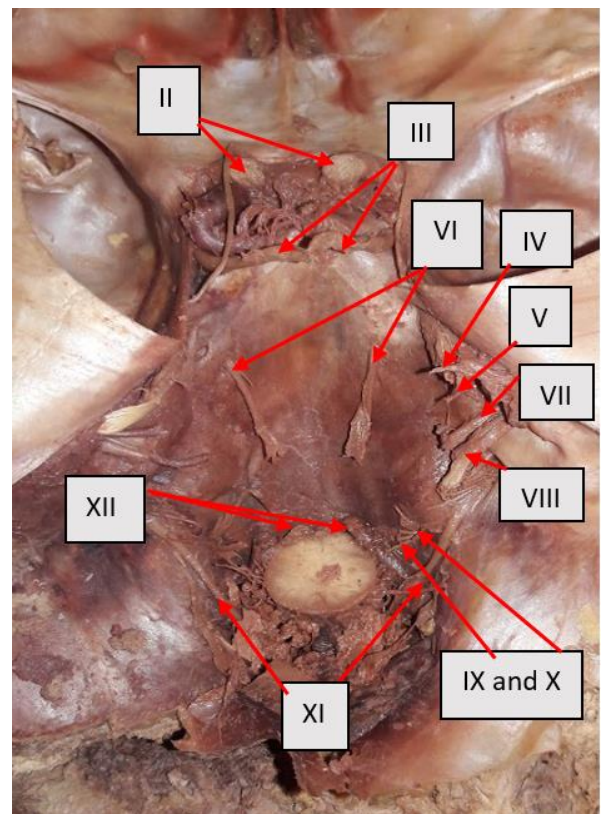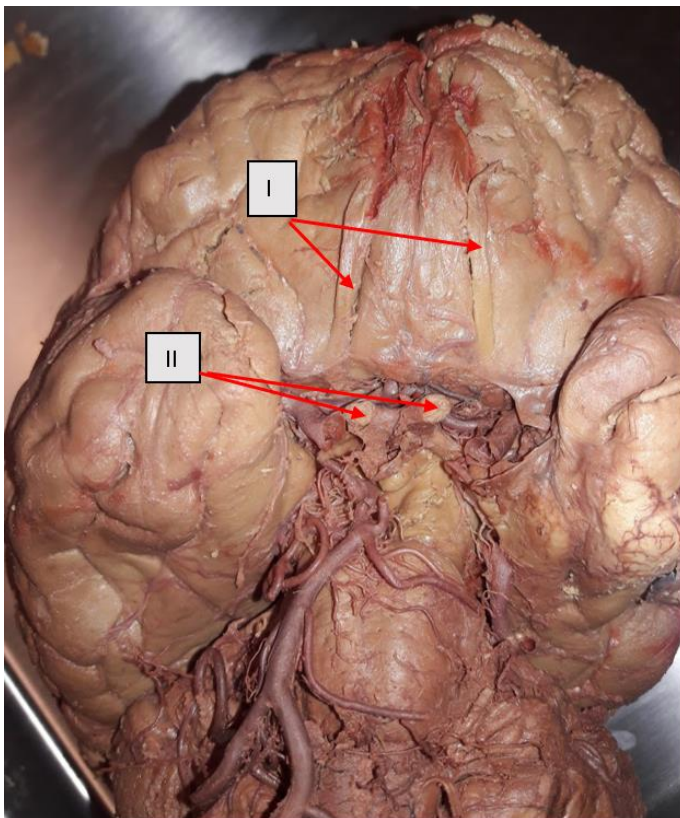

Figure. 11 Cadaveric Dissection of the Cranial Nerves

Typical and Aberrant Vascularity of the Encephalon

The brain receives approximately one-sixth of cardiac output and one fifth of the oxygen consumed by the body at rest. The blood supply to the brain is derived from the internal carotid and vertebral arteries and venous drainage occurs via cerebral and cerebellar veins shown in Figure 12.[60]

The internal carotid arteries arise at the point in the neck where the common carotid arteries bifurcate. They are found bilaterally and enter the cranial cavity through the cranial canal in the petrous part of the temporal bone, move superiorly, along the neck, passing posterior to the ear into the subarachnoid space near the temporal lobe. The cervical part of the artery ascends vertically through the neck without branching to the cranial base. The terminal branches of the internal carotid arteries are the anterior and middle cerebral arteries. The Anterior Cerebral artery supplies most of the medial and superior surfaces of the brain and the frontal pole while the middle cerebral artery supplies the lateral surface of the brain and the temporal pole.[58,60,74] Clinically, the internal carotid arteries and their branches are referred to as the anterior circulation of the brain, which supply deep structures, such as basal ganglia, thalamus, and the internal capsule.[60,74]

Figure 12 demonstrates how the left and right vertebral arteries intercept at the level of the pons , on the ventral surface of the brainstem to form the midline basilar artery, from which the posterior cerebral arteries arise. These vessels comprise the posterior circulation of the brainstem. The branches of the basilar artery supply most of the blood to the cerebellum and the pons. The Posterior Cerebral Arteries supply blood to the medial aspects of the Occipital Lobes, the inferior portions of the Temporal Lobes , the brainstem and the cerebellum.[60,74]

Near their termination, the internal carotid arteries are joined to the posterior cerebral arteries by the anterior and posterior communicating arteries, connecting the anterior and posterior circulations of the brain. The connection of the two circulations of the brain comprise the Cerebral Arterial Circle, or the Circle of Willis, shown in Figure 13. The two internal carotid arteries and the basilar arteries feed into the Circle of Willis and blood is then redistributed to the different parts of the brain by the anterior, middle, and posterior cerebral arteries.[60,74] If none of these arteries are blocked, there will be an equal distribution of blood to the different areas of the brain, because of equal amounts of pressure in opposite sides of the Circle. The connections made by the communicating arteries make collateral circulation , which is a redundancy mechanism in the event of pathological hypo blood flow in a portion of the circle. The Circle of Willis safeguards the brain in the event of blockage. As long as the Circle of Willis can maintain blood pressure at fifty percent of its normal level , no infarction or death of tissue will occur in an area where a blockage exists. If the collateral circulation is good, then the blockage will have no permanent effects.[58] Sometimes, an adjustment period is required before collateral circulation can reach a level that supports normal functioning. During this time, the Communicating Arteries may enlarge as increased amounts of blood flows through them. The communicating arteries might not expand in phase with flow requirements, which may result in a TIA as the brain is deprived of oxygen temporarily.[58,60,74]
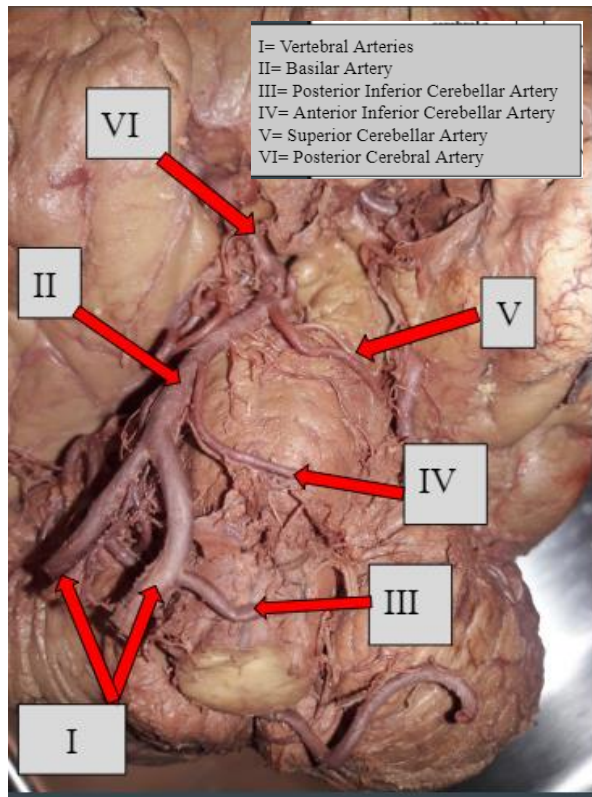
I= Vertebral Arteries
II= Basilar Artery
III= Posterior Inferior Cerebellar Artery
IV= Anterior Inferior Cerebellar Artery
V= Superior Cerebellar Artery
VI= Posterior Cerebral Artery

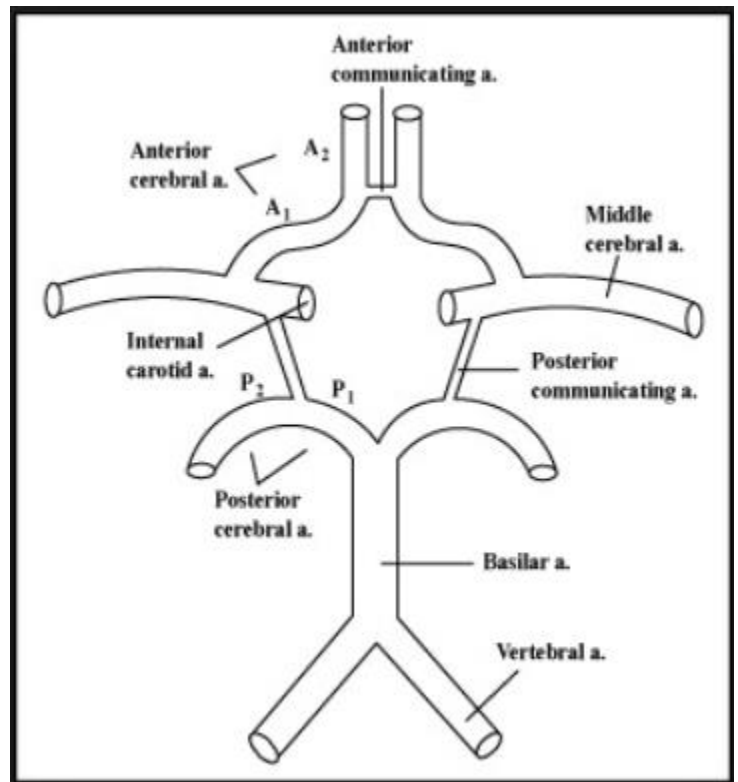Figure. 12 Cadaveric Dissection of Vascularity of the Encephalon

Figure. 13 Circle of Willis

Stroke Symptoms That Can Arise From Blockage of These Arteries

Occlusion of the Internal Carotid Arteries

The Left Internal Carotid Artery supplies blood to much of the left side of the brain, while the Right Internal Carotid Artery supplies blood to the right side of the brain. If these blood vessels become occluded, there can be very serious neurological deficits that affect the client's medical status and ultimately their quality of life.

If there is an occlusion that affects the left side of the brain, a person will have symptoms on the right side of their body. This would include right body muscle weakness, numbness, loss of vision on a portion in a portion of the right side of space, and trouble with academic and

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logical thinking. An occlusion on the right side of the brain is the opposite and affects the left side of the body. Some symptoms of this may include left body weakness, numbness, loss of vision on a portion on the left side of space, and trouble with artistic and creative activities.

The reason the left side of the brain controls the movements of the right side of the body and vice versa is the decussation of the pyramidal tract. The axons of the neurons in the brain are very long, and as they get closer and closer to the spinal cord they must bifurcate or split in two. The two axons cross over (decussation) just before the junction between the Medulla Oblongata and the Spinal Cord.[85,91]

Occlusion of the Middle Cerebral Arteries

The Middle Cerebral Artery (MCA) is the largest vessel branching from the internal carotid artery and is the most common cerebral occlusion site. The MCA feeds blood into the frontal, temporal, and parietal lobes, as well as the brain's deep structures: the basal ganglia and internal capsule. The MCA has a main stem and several branches emanating from it. If the main stem becomes occluded, all of distal structures will be negatively affected. The distribution of blood from the MCA is so large, that if the main stem becomes occluded, it can put a person at risk for severe disability or death. In contrast, if a small branch that comes off of the MCA becomes occluded, a smaller territory of the brain is damaged and there is a less severe disability.[88]

A complete occlusion of the MCA stem, blocking blood flow on both the left and right sides often lead to contralateral hemiplegia or hemiparesis as well as contralateral homonymous hemianopia. Hemiplegia is the complete paralysis of one half of the body and hemiparesis is weakening of one half of the body.[76,88] Hemiplegia can lead to something called "Frozen Joints".

This is when there is not enough movement in a joint to keep constant lubrication, which allows a joint to have full range of motion and move around. If a joint is not constantly moving, then it can become stiff, which can lead to calcium buildup, causing the joint to become frozen and unable to move at all.[60] Contralateral homonymous hemianopia is the name for a visual field deficit in the eyes. A total MCA stroke affects the arms and the face much more than the legs, so these areas can be used for tests to see if a person has suffered from a complete occlusion of the MCA. On assessment, the patient is asked to smile. If the patient is not able to do this, a harsh smell can be introduced, causing the patient to grimace. The assessor should be looking for asymmetry of the face, which can indicate paralysis of the facial muscles on one side of the face. Another test is to assess arm and hand strength. The assessor should be looking for spontaneous movement, differences between the left and right sides, or palmar drift and weakness. Even though both sides of the MCA may become occluded, the likelihood of both sides remaining occluded for a period of time that is detrimental to both sides of the body is unlikely. Often, blood flow is able to proceed on one side after a while, but the other side remains occluded. This is why hemiplegia is fairly common after an MCA occlusion. Paraplegia is not as likely to occur because an MCA stroke affects the arms and face the most. Paraplegia occurs when there is damage to the lower half of the spinal cord. Therefore, it more often occurs with occlusion of the vertebral arteries, which supply most of the blood to the spinal cord, rather than the occlusion of the MCA.[76,88]

Laterality of a MCA stroke determines additional signs and symptoms. If a stroke affects the left brain hemisphere, a patient may experience Aphasia, which is the partial or total loss of the ability to communicate.[88]

The left MCA provides Broca's area in the frontal lobe, and Wernicke's Area in the Temporal lobe with blood. If Broca's area becomes damaged, Expressive Aphasia (Broca's Aphasia) may occur. This is when a person has trouble expressing themselves through speech, writing, and gestures.[38,58,60] A person with Broca's Aphasia knows what they want to say, but cannot get the words out. Their speech may be difficult to understand and may include slurred speech, trouble pronouncing words correctly, using made up jargon, and the repetition of phrases. Overall, their voice may seem weak and hoarse. A person with expressive Aphasia may also have trouble with written communication and the naming of objects even though they may know how to use them. The other type of Aphasia that can occur from the blockage of the left MCA is known as Receptive Aphasia or Wernicke's Aphasia. When Wernicke's area becomes damaged, a person can speak just fine, but has trouble with comprehension of what is being heard. A person with Receptive Aphasia may speak in long sentences that do not make much sense, with made up words and non-sequiturs. They are able to hear what is being said but are unable to understand the message. They may also have trouble identifying letters and understanding written words.[38,58,60] The most severe type of Aphasia is called Global Aphasia, which occurs if there is damage to both Broca's and Wernicke's Areas or the fibers that connect them called the Arcuate Fasciculus (AF). If a person has Global Aphasia, they may lose the ability to understand language, speak, read, and write.[60]

Most people are left hemisphere dominant, where the speech and language center is located. A patient with right sided weakness may have Aphasia. However, there is a small percentage of left handed people who are right hemisphere dominant, meaning that if they have left sided weakness, Aphasia may occur due to the speech and language center being on the right hemisphere of the brain. When assessing a patient, it is important to notice what side any

weakness may be on, as well as hand dominance. This aids the examiner's diagnosis in order to properly treat.[88]

With stroke affecting the right MCA (or non-dominant hemisphere) patients may show signs of unilateral neglect. This is an impairment of the ability to recognize and respond to stimuli on one half of the body. For example, if a person's right MCA becomes occluded, their symptoms may include decreased awareness of, failure to attend to, and lack of concern for the left side of the body.[88] Neglect is often associated with Hemianopsia, which is the loss of sight in one half of the visual field. A blockage of the right MCA may cause left hemianopsia, or blindness in the left visual field of both eyes. This can occur if the left MCA is blocked, affecting the right visual field as well. The reason neglect is more common in a person with hemianopsia, is because if they are not able to see anything in one half of their body. They tend to forget it is there and ignore it.[58,88] Some tests that clinicians can use to test for neglect include the Bells Target Cancellation Task and the Line Bisection Task. The Bells Cancellation Task consists of seven columns on a piece of paper. Three of the columns are on the left side of the paper, three are on the right, and there is one in the middle. The person being tested is asked to cross out these columns. If the person misses crossing out all of the columns on one side of the paper, this can indicate Hemianopsia. The Line bisection task asks a person to divide different sets of lines in half. If a person fails to bisect the lines correctly this can also indicate impairment of one half of their visual field.[24,61] It is important for a therapist to recognize any type of hemianopsia or neglect, because in the rehabilitation process for stroke, the goal is for the patient to be able to use both sides of their body in order to perform everyday tasks.

Occlusion of the Anterior Cerebral Arteries

The Anterior Cerebral Artery (ACA) is least affected by strokes, so this type of stroke is very easily misdiagnosed. Classic signs of ACA strokes include contralateral leg weakness and sensory loss. Apraxia of gait may also occur if an ACA fails to supply an adequate amount of blood to the medial cortex. The ACA supplies blood to the anterior aspects of the frontal lobes, which are involved in higher level cognition, such as reasoning and judgement. If these areas of the brain fail to receive enough blood, conditions like cerebral dementia may result. Confused language or language indicative of cognitive impairment may also occur.[58,88]

Occlusion of the Posterior Cerebral Arteries

The Posterior Cerebral Arteries (PCA) supply blood to the occipital lobe and inferior and medial temporal lobes. Vision is the primary function of the occipital lobe, so occlusions of the PCA commonly cause visual deficits. Contralateral Homonymous Hemianopia (discussed above) can also be caused by damage to the occipital lobe. Hemianopia may occur as a result of occlusion of the MCA when the visual deficit stems from the visual pathway. In the case of an occlusion of the PCA, hemianopia may occur simply from direct occipital lobe injury. Similarly, to effects of an MCA stroke, neglect can occur due to the loss of parts of the visual field. Larger PCA strokes may also result in Aphasia.[88]

Occlusion of the Vertebral Basilar System

A stroke affecting the vertebral basilar circulation can affect the cerebellum, brain stem, or both. If the cerebellum is affected, a patient may suffer from impairment of balance and coordination. One test that can be done to assess for this is to have the patient extend their index finger and touch their nose, alternating hands each time to see if they have trouble moving their

fingers in a straight line. This tests for ataxia or incoordination. Another test that can be done is to have a patient slide their heel up and down the shin of the other leg, alternating legs to see if they have trouble moving in a straight line. A test for gait is also good to see if a person is losing their balance as they walk. Cerebellar strokes may also cause vertigo, nausea, nystagmus, and slurred speech.[58,60,88]

If the brain stem is affected, this can be devastating. Signs and symptoms differ with different stroke locations, but may include hemiparesis, quadriplegia, sensory loss or disturbances (Paresthesia or Neuropathic Pain), double vision, slurred speech, impaired swallowing (dysphagia or paralysis of the throat muscles), decreased levels of consciousness, or abnormal respiration. Patients with brain stem strokes are likely to be critically ill and may require emergency intubation and mechanical ventilation.[60,88]

Emotional Disturbances as a Result of Stroke

Emotion is a multi componential process in response to external and internal stimuli, which implicates perceptive, motivational, autonomic, motor response, and cognitive evaluation. Emotional changes can affect short-lasting processing like emotional reaction, or long standing states like mood. After stroke, emotional disturbances include mood disorders, emotional dyscontrol, and the alteration of emotional reactions.[5] Two of the most common emotional disturbances after stroke include Post Stroke Depression and Post Stroke Fatigue.

Post Stroke Depression

Post-Stroke depression (PSD) is the most common psychological sequel of stroke and occurs in approximately 40% of stroke victims. The characteristic features are sadness, feelings of hopelessness, and reduction of interest. Physically, PSD has been associated with left anterior

region in the brain affecting the left frontal lobe and left basal ganglia. In addition to this, depression from a stroke may stem from biological changes in the brain. Emotionally, PSD can come from the feelings that a person experiences after a stroke.[5,90] They may have emotions relating to physical losses or the overwhelming reality of needing to go through therapy to recover. The rehabilitation process can be very challenging and discouraging for someone who was once able to act as an independent individual, but now needs to depend on others for everyday living. Individuals may feel like they may never recover from their stroke, and may go into a depressive spiral.[5] Stroke patients who are depressed experience poorer outcomes from rehabilitation, decreased quality of life, and a substantially increased risk of suicide. Anxiety often accompanies PSD.A patient's mind can be filled with fear and worry about experiencing another stroke or TIA and they may often worry about things like being able to communicate in an emergency, returning to work, falling down, or embarrassing themselves in social situations. These anxious thoughts and feelings can bring about behavioral changes in the way a stroke patient acts and responds to others. They may feel irritable, have trouble concentrating, or get tired very easily. They might also avoid things that they would have normally done before the stroke, like going to see friends, or going shopping at the mall.[90]

Post Stroke Fatigue

Post-stroke Fatigue (PSF) is a symptom that affects between 38% to 75% of the stroke population. It has been described as the most distressing symptom after stroke because it is unpredictable in nature. PSF is recognized as a symptom independent of PSD, but it is not addressed as much despite its impact on the rehabilitation process.[63] PSF is a multidimensional motor perceptive , emotional, and cognitive experience and makes a person feel unwell and not in control of the recovery process. Fatigue can be classified as either objective or subjective.

Objective Fatigue is defined as observable and measurable decrement in performance occurring with the repetition of a physical or a mental task. Subjective Fatigue is a feeling of early exhaustion, weariness, and aversion to effort.[1,63] A person with PSF may lack energy and require frequent breaks throughout their day. This can make the whole rehabilitation process very challenging. PSF is often confused with "feeling tired", however it has its differences. Some of these differences include de-conditioning, physical impairment, disuse, and sleep disorders. PSF often arrives without warning, and rest does not always make it better. It affects every patient differently, but often, patients say it feels like they are hitting a wall physically, emotionally, and/or mentally.[1]

The Basis of Rehabilitation: Neuroplasticity

Neuroplasticity is the ability of the Central Nervous System to remodel itself. The brain is constantly changing and neuroplasticity is constantly occurring. Neuroplasticity is how humans adapt to changing conditions, learn new facts, and develop new skills. If the brain is injured, it tries to repair itself through these normal mechanisms. Neuroplasticity and the remodeling of brain pathways can be understood at several different levels, including changes in individual neurons and changes in how different neurons interact with one another. Some of the processes of change are quick and can allow for more rapid adaptations, while others may take longer, but may be more permanent. Generally speaking, the more persistent and early a change is, the more likely it will be permanent. This is where the efficacy of rehabilitation comes into play.[23,41]

Innate physiological and anatomical plasticity are important processes that underlie substantial gains in motor function after stroke. The key standard for poststroke rehabilitation is the combination of task-specific training and general aerobic exercise. Task-specific training,

which comes with both physical and occupational therapy focus on the rehabilitation of motor functions while the general aerobic exercise allows a person to regain the physical endurance needed to perform these specific tasks to the best of their ability and to attain specific functional gains. When it comes to the overall idea of adapting to new poststroke situations, rehabilitation implications that take place earlier and are used quite frequently have the best results.[23]

In the process of neuroplasticity, the brain has to find and create new pathways that can allow a person to maintain motor control, even if some pathways are inhibited by anoxia due to stroke and also in the case of other brain injuries. If one part of the brain is inhibited, other parts of the brain can be trained to take over affected areas. There are several different ways in which the brain can do this.[23,41]

First, body parts can compete for representation in the brain. Use of a body part can increase this representation. When it comes to rehabilitation, different therapies encouraged repetitive motions in order to enhance neuroplasticity. In the case of a hemiplegic patient, they practice motions with their paralyzed size in attempt to increase representation in the primary motor cortex. Plasticity permits the reorganization that may occur in this situation.

Second, the premotor cortex can substitute for the motor cortex to control motion. While the primary motor cortex has the largest and most powerful contribution to the function of the corticospinal tract, the premotor cortex also contributes. Therefore, if anything were to affect the motor cortex in the brain, the premotor cortex has the ability to contribute to an individual's ability to maintain motor functions.[41]

Third, the contralesional hemisphere can take over motor control if all else fails. In the human body, there are ipsilateral corticospinal neural pathways that connect both sides of the

brain. These pathways tend to be weak, however they can be helpful in the case of a hemiplegic patient, allowing the innervation of proximal muscles and gaining some type of movement. In a hemiplegic stroke patient, the connections between the undamaged and damaged hemispheres are essential for rehabilitation. Functional magnetic resonance imaging studies show that the damaged hemisphere has increased blood flow when bilateral movements are made. Use of the undamaged hemisphere might help support the damaged one. Also, the ipsilateral hemisphere helps with the activity of the premotor cortex rather than the motor cortex itself, going back to the idea that the premotor cortex can act as a substitute.[23,41]

Finally, Neuroplastic mechanisms can be facilitated through therapy and is a good basis for intervention. Constraint induced (CI) movement therapy forces stroke patients to use the hemiplegic limb by constraining the good limb. In several clinical trials, patients have shown improvement with the use of CI. Bilateral symmetrical arm movement training has also been shown to be helpful due to the facilitation of the contralateral hemisphere. These theories and evidence based practice can be built into the exercises that a patient might undergo through physical and occupational therapies.[41]

The Penumbra

The Penumbra is an area of brain tissue that is damaged but not yet dead after focal ischemia. These regions are usually peripheral in location and occur in regions where blood flow is sufficiently reduced to cause hypoxia. This hypoxia can be severe enough to arrest physiological function, but not so complete as to cause irreversible failure of energy metabolism and cellular necrosis. Tissue tolerance to ischemic damage is dependent on residual flow and duration of flow disturbance, so the ischemic penumbra is a dynamic imperative. The existence of a penumbra implies that therapeutic salvage is theoretically possible after a stroke. However,

the penumbra exists for a short period of time, even in the center of ischemia, where irreversible

necrosis propagates to the neighboring tissue over time. This renders the time window of

therapeutic opportunity. The necrosis can extend to the penumbra in a matter of hours, so that is

why it is imperative that quick action is taken to salvage the surviving tissue.[55,67]

When looking at the pathophysiologic mechanism of ischemic brain damage on MRI's,

or PET scans, it is important to understand how to identify the penumbra of the affected area.

Fig. 14 shows the "four tissue compartments concept". The compartments can be distinguished

by the various physiological imaging modalities during acute ischemic stroke. Compartment 1

represents the area of unaffected tissue, Compartment 2 is the area of mildly hypoperfused tissue,

Compartment 3 is the tissue at risk (the ischemic penumbra), and Compartment 4 is the tissue

already irreversibly damaged (the ischemic core). Action must be taken rather abruptly after the

occurrence of an acute stroke in order to prevent the ischemic core from spreading to the

ischemic penumbra and other areas of unaffected tissue, causing irreversible damage in a larger

region of the brain. Fig. 15, the image on the left shows a brain scan obtained 2 hours after

symptom onset of a 70-year-old female who presented with Aphasia and Right Sided weakness.

She responded to intravenous thrombolytic treatment that was initiated immediately after the

imaging. The image on the right shows the decreased extent of damage after treatment. The

difference in the extent between the two images can be interpreted as a treated ischemic

penumbra. If timely recanalization was not performed, the dark area in the center of the left

image (ischemic core) would have affected the surrounding white region (ischemic penumbra)

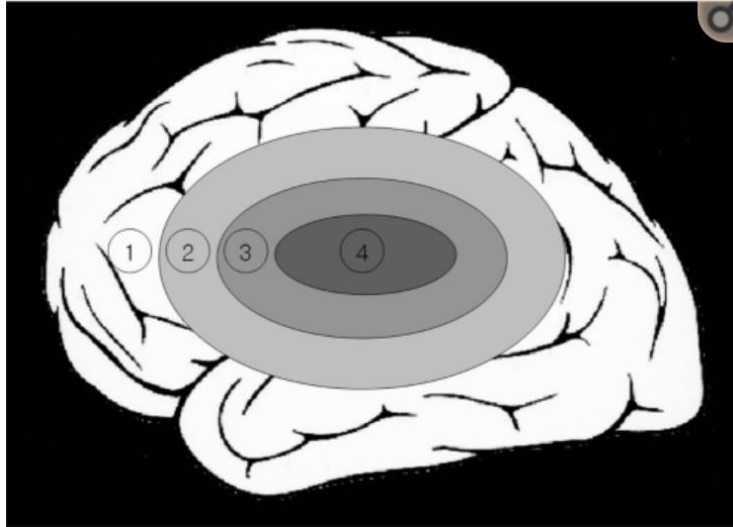leading to infarction in the entire region.

Figure. 14 Four compartments of an Ischemic Penumbra[53]
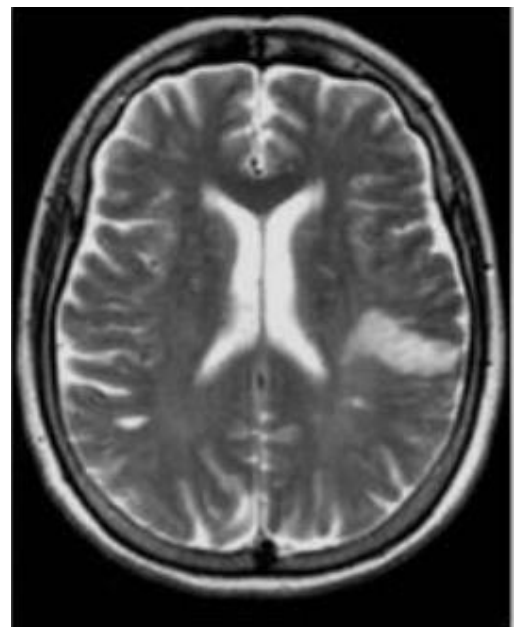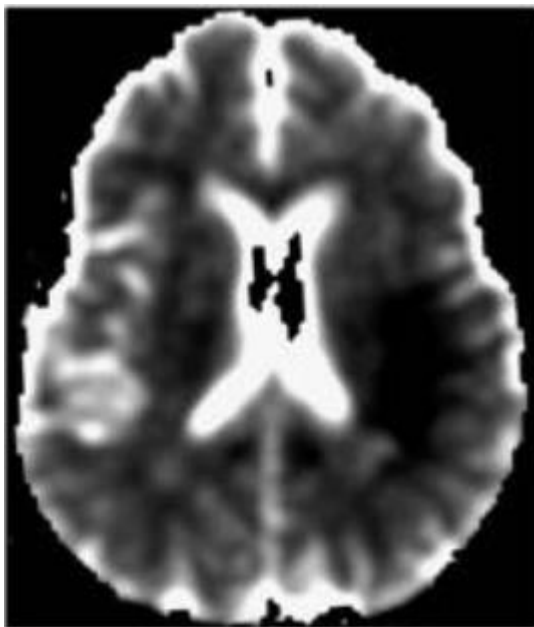
Figure. 15 Before and after treatment of ischemic penumbra in 70 year old female [53]

Rehabilitation Implications for the Occupational Therapist

Occupational Therapy is the only profession that helps people across the lifespan to do

the things they want and need to do through the therapeutic use of daily activities. Occupational

Therapy practitioners enable people of all ages to live life to the fullest by helping them promote

health and prevent (or live better with) injury, illness, or disability. In the specific case of a

stroke patient, an Occupational Therapist focuses on helping the recovery process through the regaining of skills and providing support for people experiencing physical and cognitive changes that they did not have previously, before the stroke. Occupational Therapy practitioners focus on adapting the environment and certain tasks to fit a person and their condition.[3]

The Occupational Therapy process includes an individualized evaluation in which assessments are made and goals are set, customized intervention to improve a person's ability to perform daily activities and reach these goals, and an outcome evaluation to ensure that goals are being met and/or changes being made to the intervention plan.[3,65]

Unlike the more well-known Physical Therapy, Occupational Therapy focuses on Functional Therapy, as well as Psychological Therapy. Functional Therapy focuses on the functions and dysfunctions of the motor and nervous systems, and how planned activity can best help develop or establish sensorimotor and perceptual abilities. This is especially true when physical disability limits a person' activities in terms of daily care, leisure activity, and working. Both Physical Therapy and Occupational Therapy have the functional therapy aspect, which is individualized to maximize developing and reestablishing nervous or muscular coordination, extending the motion of joints and strengthening muscles within the limits of the patient's physical tolerance. Functional Therapy must also take into account the patient's motivation to use the activities given in a therapeutic manner.[25,65]

The component of the Occupational Therapy rehabilitation process that physical therapy does not include is the Psychological Therapy aspect. Occupational Therapy acknowledges the fact that disability can stem from either physical handicap, or mental illness and creates interventions based on the understanding that psychological rehabilitation is equally as important as physical rehabilitation. Planned occupational therapy interventions and activities can be

helpful in overcoming lack of self-confidence, low self-esteem, difficulty coping with stress, and depression. Occupational Therapy focuses on balancing work, play, and rest, focusing on maximizing a patient's independent function, and ultimately viewing a patient as a functioning individual, as opposed to a patient with a handicap.[25,65]

The Occupational Therapy Process

Step 1: Assessments are made and goals are set

Assessment requires the therapist to draw on their theoretical knowledge and clinical experience to make a clinical judgement of what is to be assessed and how. It often includes a series of standardized and non standardized measures that inform the occupational therapist about the extent of impairment, activity, and participation limitations, while considering environmental and social contexts. Once this is done, goals can be set in order to see what rehabilitation interventions would be best for the situation at hand.[25]

Goal Setting is a collaborative process between the therapist, patient, and in some situations, the family. This process involves both education and negotiation. The occupational therapist should first ascertain the patient's and their family's long-term goals, to see where they see themselves at the end of therapy. Long term goals are aspirational, giving patients hope and motivation to engage in the therapeutic process. It is important that the therapist makes sure that these long- term goals are realistic and that the patients know they may take time to reach. In order for a person to reach these long term goals, patient's must take things step by step. Short term-goals are the small steps needed to actually obtain the long-term goals. This is where specific occupational therapy interventions come into play. It is the job of the therapist to make sure these short term goals fall in the SMART guideline setting approach. SMART stands for:

specific, measurable, achievable, realistic, and timely. While the process of rehabilitation may take an extended period of time, progress must be made in a timely manner, or the patient may start to lose hope and not want to engage in therapy at all.[25,54]

Stroke Impact Scale (SIS)

The American Occupational Therapy Association (AOTA) lists several different assessment tools for therapists to utilize when working with their patients. Among them is the Stroke Impact Scale (SIS), which is designed to assess multidimensional stroke outcomes, including strength, hand-function, Activities of Daily Living (ADL), Instrumental Activities of Daily Living (IADL), mobility, communication, emotion, participation, and memory and thinking. The SIS assessment is a stroke specific, self-report health status measure in which patients are asked several questions that evaluate how their stroke has impacted their health and life. The purpose of this assessment is for the therapist to get an idea of how the patient feels about their stroke without feeling any sort of pressure or interference from anyone else. This assessments gets this information solely from the patients point of view. The assessment includes questions about impairments and disabilities specific to a person's stroke, how they feel their stroke has affected their quality of life, and a rating of how much a person thinks they have recovered from their stroke on a scale of 1-100.[77]

Initial Occupational Therapy Assessment by the Therapist

The Occupational Therapy standards for Stroke states that the initial occupational therapy assessment should include an assessment of cognitive, motor, and functional abilities. Assessment in these three areas can help a therapist to understand exactly what areas the stroke may have affected and how to target these specific regions through rehabilitation interventions.[6]

Cognitive and Perceptual Screening

Cognitive and Perceptual screening is a key component of the occupational therapist's role in stroke care. It gives therapists more information on a patient's function and can allow them to gain an idea of any impairments patients might be experiencing. The aim of these assessments is to provide an indication of the patient's attention, memory, safety awareness, and judgement, as well as visual attention, spatial relations, and visual recognition. Cognitive and perceptual impairments may impact other occupational therapy assessments and interventions, so it is important to recognize them right away. Often, high-level cognitive and perceptual impairments are not as apparent in a person's everyday routine, so assessments by therapists are sometimes able to identify concerns that could otherwise be missed.[26,49]

Psychosocial Screening

It is important for an Occupational Therapist to be aware of and screen for psychosocial issues including mood and fatigue. The aim is for the therapist to recognize the patient's emotional state and motivation for rehabilitation. As part of the multidisciplinary team, if an occupational therapist notices that a patient appears unmotivated, tired, depressed, or unwilling to engage in therapy they may consult other members of the team, including a psychologist. It is in the patient's best interest that professionals in all areas are aware of the patient's emotional state post-stroke.[25,26]

Neurophysical and Motor Screening

These screenings can help to set the scene for further assessment and intervention and to build up a picture of a patient as a whole, and how they move. This can also help to provide information for setting goals in conjunction with the patient and other members of the

multidisciplinary team. Neurophysical screening is best used in whatever position the patient is the most comfortable in. Often, an occupational therapist will ask a patient to put on or take off a jacket or bend down to take off one of their shoes. This allows therapists to identify how a patient moves, attempts to move and what aspects of their movement is abnormal. This provides the information in which intervention can be formed. Motor Screening focuses more on specific movements such as how a patient moves when transferring from a wheelchair to a bed, how they sit on a chair, how they stand up, and what kind of gait they have. During these movements, therapists often look for favorability of one side of the body over the other. For instance, if a therapist notices that a patient is using their right leg to support all of their body weight when standing up, they could figure out that the left side of the brain was likely affected by the stroke.[25,26,43]

Functional Screening

Functional Assessments focus on obtaining information on a patients's residual skills, their impairments, and task performance. Different functional assessments like washing, dressing, or kitchen assessments can provide vital information on how a patient's impairments impact their functional ability and allow the opportunity to identify how a patient is using their residual skills to perform a familiar task. After a stroke, a person might have great difficulty physically approaching different tasks and may not have enough stamina and endurance to keep up with everyday tasks. Those affected by Aphasia may have trouble communicating with those around them slowing down their efficiency with everyday activities. Functional Screening looks out for these occurrences.[25,31,59,69]

Assessing Functional Mobility

Functional Mobility describes a person's ability to move around in his or her environment. Examples of this include walking, scooting along a bed, and rising from a chair. A person recovering from a stroke may have a difficult time maintaining normal functional mobility. A stroke patient in a hospital setting may be evaluated by a therapist to see if their condition is safe enough to return home. If a person is not able to leave their home due to their condition, home-health therapists may visit the home for evaluation and can also provide ideas to help adapt the patient's home environment to make it safer for the patient. Occupational Therapists can also help to train caregivers on how to help post stroke patients. The main focus of home-health Occupational Therapists is to help patients and caregivers work with bed mobility (scooting, rolling, moving, lying, and sitting), transfers (chair to bed or chair to shower etc.), and ambulation with assistive devices such as canes and walkers. . Home-Health OT's allow for a patient to be able to stay in an environment that they are comfortable in, while still receiving the knowledge of a rehabilitation professional.[59,82]

ADL and IADL

The main focus of Occupational Therapists is to assist their clients in engaging in meaningful and purposeful daily tasks. OT's mainly focus on interventions that help a patient to reach the highest level of independence possible. In order to do this, OT's focus on Activities of Daily Living (ADL) and Instrumental Activities of Daily Living (IADL). ADL's are activities in which people engage in on a day-to-day basis. These are everyday personal care activities that are fundamental to caring for oneself and maintaining independence. ADL's include bathing, dressing, grooming, mouth care, toileting, transferring, walking, climbing stairs, and eating. IADL's are activities related to independent living and that determine a person's ability to care

for himself or herself. IADL's include shopping, cooking, managing medications, using a phone, doing housework, doing laundry, managing finances, and using public transportation.[36,86]

Step 2: Customized intervention to reach set goals

Interventions enable patients to meet their goals and ultimately aim to reduce activity and participation limitations. Rehabilitation is a problem-solving and educational process aimed at maximizing recovery by using restorative approaches to reduce impairments and adaptive approaches to prevent impairments from translating into functional disability. A minimum of 45 minutes per session of occupational therapy is recommended in acute phases with opportunities for repeated practice and generalization across tasks.[25]

Main Approaches used by Occupational Therapists

Restorative Approach

The Restorative or Remedial Approach relies on the theories of neuroplasticity and the ability of the brain to reorganize itself. Neurophysiological activities, like normal movements and motor relearning are included within this approach. Within this, Occupational Therapists provide controlled stimulation to promote normal CNS processing of sensory information. This normal sensory processing helps to make patients more aware of their movements and help with the motor responses required for the performance of functional tasks. This approach aims to reduce impairment and to improve activity and participation with everyday tasks. This approach is designed to challenge patients and encourage the brain to adaptively reorganize itself for successful behaviors. Restoration of Impairments tends to be more successful for people with motor impairments alone because people with cognitive impairments may have more trouble transferring learned skills.[78,83]

Adaptive (Compensatory/Functional) Approach

The Adaptive Approach focuses on the repetition of particular skills, which are normally associated with ADL. Adaptive approaches are traditionally used when restoration is unlikely and assumes that certain functions will not recover. The compensatory side of the adaptive approach is when loss of function is compensated by changing the environment or daily routine of a stroke patient. The more functional side of this approach includes modifying and practicing a task that is meaningful and purposeful to the patient. With these aids, different ADL's become easier in a variety of environments. This approach is patient-centered, easy to explain, uses problem solving, , meets short-term needs, and gives quick results.[9,50]

Cognitive Rehabilitation

Cognitive functions that may be impaired after a stroke include things like attention, memory, language, praxis, and executive functions. These deficits typically occur from strokes resulting from the occlusion of the left MCA or the anterior or posterior Cerebral Arteries, which affect the frontal and temporal lobes. The principles of cognitive rehabilitation are goal oriented, individualized, educate and include family members, significant others, or caregivers, focus on functional improvement, and include psychological or emotional support. Occupational Therapists use both the Restorative and Adaptive approaches, but tend to favor the functional Adaptive approach because the activities are more meaningful and familiar to their patients.[25,70]

Intervention Strategies for Attention

Attention is required for most other cognitive functions to take place. It is dependent on an adequate degree of arousal and alertness and helps us to process a large amount of information on a daily basis. Attention is commonly affected after stroke, especially in the early

stages of recovery. When an Occupational Therapist is assessing a patient who might have attention deficits, they may ask the patient to count backwards from 20 or recite the days of the week backwards, making sure the patient has an organized approach, rather than a random, erratic approach. The Occupational Therapist may also observe a patient carrying out functional tasks, looking for things like the patient becoming easily distracted as complexity of the task increases, the patient stopping their task to talk, or if the patient can walk and talk at the same time. If the therapist notices the patient missing details, or appear impulsive or easily agitated while performing certain tasks, this may be a sign that a stroke may have affected the patient's attention.[25,34]

Once the Occupational Therapist determines what level a patient's attention problems are at and how it affects their occupational performance and behavior, they are able to implement different interventions. The most common OT interventions for cognitive rehabilitation using the functional adaptive approach is Task Specific Training, which focuses on the improvement of performance in functional tasks through goal-directed practice and repetition. It stresses the value of the use for specific and relevant functional tasks instead of impairment reduction exercises. Activities that are meaningful and interesting to the patient and are able to be easily modified by the OT should be chosen. An example of a task-specific exercise would be in a kitchen setting, where the OT asks the patient to make a cup of hot chocolate. The first time, the therapist might put a cup of hot water, the packet of chocolate powder, a spoon, and marshmallows out on the counter. The patient would just have to put it all together. The next time the patient comes in, the therapist could make the task more challenging, like leaving the marshmallows in a cupboard, or not filling the cup with water. The patient would not just have to put all of the items together, but would have to figure out to get water from the sink and put it in

the cup, and to also grab the bag of marshmallows from the cupboard. As the patient's attention improves, the challenge and complexity of the task can be increased to work on higher levels of attention. Repetitive tabletop activities such as word searches and jigsaw puzzles can also be used to help improve a person's attention, if used in the same manner, providing an appropriate challenge as the person's attention improves.[78,83]

Compensatory Adaptive Strategies can also be used for attention, but it is not as preferred by Occupational Therapists. This approach focuses on adapting the environment around a patient, rather than rewiring their attention through various activities. Strategies can include providing structure in a patient's day by keeping a diary, ensuring that a patient has a quiet place they can go if they feel overstimulated, and using verbal and visual prompting to maintain a person's attention during tasks. These strategies can be useful because they can help alleviate emotional stress between stroke patients and their caregivers.[9,20,50]

Intervention Strategies for Memory

An Occupational Therapist might discover memory difficulties by observing a patient shopping or recalling the events of their day. Once the therapist has a good idea of where the patient's memory processes might be breaking down, they are able to implement different interventions. In the case of memory problems, the restorative approach has limited effect. Instead, the use of adaptive approaches and assisted devices within the context of functional activities tends to be more successful. Fig. 16 shows the five main steps in order to get a patient recall specific things. It shows that adapting the environment is a very effective approach. Eliminating distractions, leaving cues and instructions for the patient to follow, and constant repetition in therapy has proven to be an effective approach for a stroke patient to remember how to perform certain tasks and remember important details.[25,34]
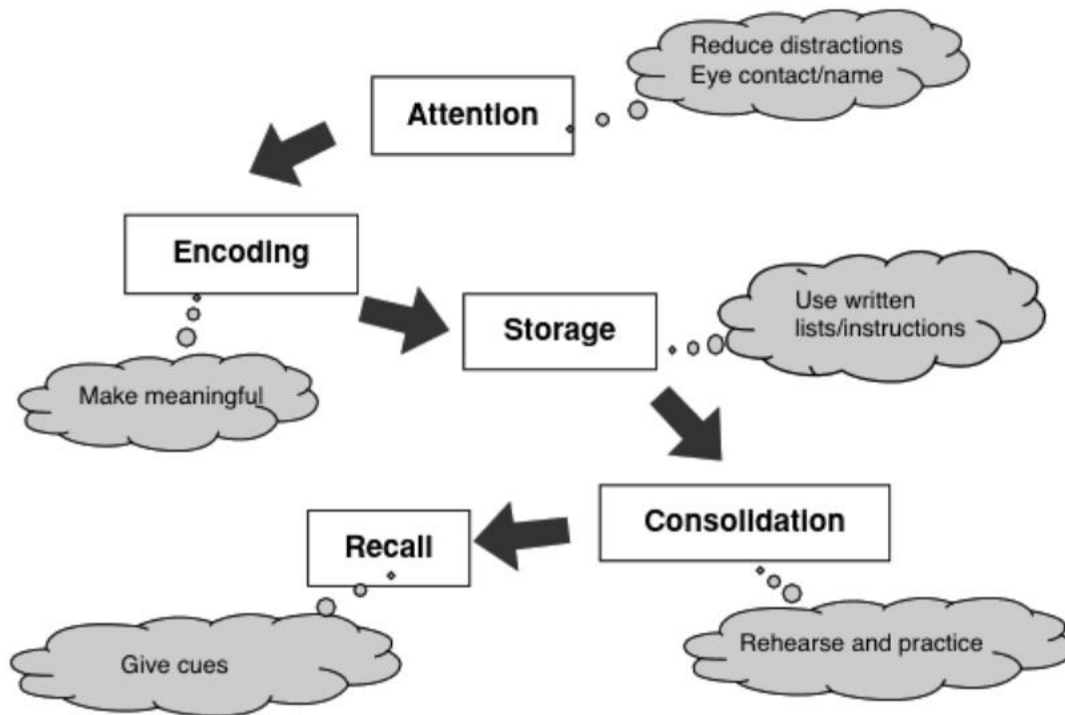
Figure. 16 Five step OT intervention strategy for recall [25]

Intervention Strategies for Apraxia

Praxis is the ability to plan and perform purposeful movement and is a cognitive process that relies on the interaction with other cognitive, perceptual, motor, and sensory systems. Praxis is based on the whole idea of motor planning in which actions are required to be performed in a specific sequence in order to complete a task successfully. In a person with unaffected praxis and motor planning, they may at times perform tasks incorrectly, but are able to recognize errors and correct them. In a person who develops Apraxia (a cognitive motor planning disorder) after a stroke, they may not be able to recognize their mistakes. Apraxia is one of the more disabling effects of stroke or brain injury and impacts a person's ability to carry out skilled voluntary movement.[42]

Ideational Apraxia is a disturbance in the conceptual organization of actions, so a person with this type of Apraxia may use a single action object appropriately, but may have more

difficulty when a sequence of actions for object use is required. An example of this would be a person buttering their toast before putting in the toaster, or putting on their shoes before their socks.

Ideomotor Apraxia is a disorder in the initiation and execution of planned sequences of movement. The concept of a task is understood, but the movements lack the correct direction and timing to reach a motor goal. For example, a person with Ideomotor Apraxia may be able to pick up a comb and comb their hair spontaneously, but may not be able to do it on command.

Occupational Therapists look to see how apraxia affects a person's ability to engage in meaningful occupations in the area of self-care, work, domestic and leisure. After observation and assessment, an OT might be able to identify which type of apraxia a person is experiencing and more importantly, the impact it has on independent living.[14,17]

The functional Adaptive approach is often used with Apraxia because studies have shown that people with Apraxia can improve their functional performance over time on their own, and the adaptive approach does not impede the recovery of the impairment. Task specific training through Occupational Therapy is often used to help restore independence for trained activities. Structured functional tasks in familiar environments, like a kitchen setting practiced over and over help to break down activities into manageable steps in order to help a patient relearn certain tasks. The tasks could not be generalized and should be specific to and incorporated into a patient's daily routine.[20,42,50]

Intervention Strategies for Aphasia

As discussed previously, Aphasia may occur following a stroke, and this may affect a person's understanding of the spoken word, verbal expression, reading, or writing. Aphasia is

often accompanied by physical deficits (for example, a person with a stroke on the left hemisphere of the brain may have difficulty with language accompanies by right sided weakness), so this is where the work of an OT comes into play. The OT typically focuses on the other cognitive problems that interfere with performance of functional activities, while the Speech and Language Therapist focuses on the actual speaking and comprehension of the patient. Joint sessions between the OT and a speech and language are beneficial because it helps to ascertain what elements of a person's performance are due to language difficulties or other cognitive problems.

Something that is important for OT's to remember when working with a patient with Aphasia is that they need to be 100% mindful of how they are communicating with a patient, and how the patient is attempting to communicate back. For instance, sometimes verbal instruction of tasks is affirmed by patients, but other times, it can be frustrating for them. Sometimes for an OT, gesturing, silence, and visual cues might be a more effective approach than verbal cues. It is also important that an OT lets family, caregivers, and other staff know what communication strategies are working and what types to avoid. This can help other people to understand how to best understand and work with an Aphasia patient, no matter their role in the rehabilitation process.[19,75]

<u>Perceptual Rehabilitation</u>

Perceptual Impairments after a stroke can have a major impact on occupational performance. With these impairments, a person may have trouble "getting a sense of their senses". Perceptual problems are common following both right and left hemiplegic stroke usually as a result of an occlusion of the MCA. Symptoms following damage to the right hemisphere are more common because it plays a greater role in processing visual and spatial

information than the left hemisphere. Specifically damage to the right parietal lobe and the occipital lobes causes the most problems when it comes to perception. Perceptual impairments can be classified into three categories: body scheme, visual discrimination, and Agnosia. Once the severity and types of perceptual impairments are clarified, Occupational Therapists are able to explain to their patients and caregivers what impact these impairments are likely to have on the patient's everyday activities and what strategies to take in the rehabilitation process.[25,64]

Body Scheme Interventions

Body scheme impairments are a lack of understanding of the relationship of the body and its parts. This includes the patient confusing body parts and sides of the body (Autopagnosia), a lack of recognition of the presence or severity of paralysis (Anosognosia), unilateral neglect, and difficultly understanding the concept of left and right. These problems are often related to hemianopia, because if there is impairment in a person's field of vision this may affect the way they function while performing everyday tasks.[64]

The focus of body scheme interventions is for patients to be aware of parts of their body and their relationship to each other and how they are used within function. Some restorative approaches include things as simple as asking a patient to verbally identify the parts and positions of their body. Tactile stimulation can also be used, like rubbing a cloth on a person's arm as they name it or identifying parts of the body before washing or dressing them. A lot of times, when a patient is paralyzed on one half of their body, they use their unaffected side for everything. If this becomes severe enough, it can lead to unilateral neglect. This is why it is important to incorporate bilateral activities to facilitate normal movement and improve body scheme. The adaptive approach tends to provide more cues for the patient to follow. For

instance, a therapist might say "move the part of your body that you use to nod" instead of saying "move your head".[9,20,50]

Midline Awareness Interventions

The term "Pusher Syndrome" is when patients have a severe misconception of their own upright orientation and present by pushing themselves towards their affected side of the body. Different interventions for this aim at the patient regaining awareness of their midline. Some restorative strategies include visual feedback, in which patients stand in front of a mirror and are told to self-correct themselves back to midline as well as the patient moving from different postures (standing to sitting) and maintaining their balance. Some adaptive strategies include placing pillows on a chair on the unaffected side to provide extra supporting surfaces to enhance the patient's feeling of security. With this approach, Occupational Therapists might also tell patients to refer to vertical structures like window frames and doors to adjust their balance.[7]

Unilateral Neglect Interventions

Intervention Strategies used for unilateral neglect are aimed at helping a patient to become aware of both sides of their environment. Restorative Strategies include using activities that cross the midline. For example if a patient is brushing their hair with their right hand, they would brush both sides of their head. During ADL sessions, OT's place stimuli on the patient's affected side, prompting the patients to look over to that side. For example, a therapist might place a line of colored beads from the patient's midline to their affected side. They would then ask the patient to tell them the color of the beads in order. This promotes spatial scanning and shifting attention to the side that has been ignored. Activities like word searches, jigsaw puzzles, and mazes also promote visual scanning from left to right. Tactile stimulation onto the neglected

side of the body, using vibration, or mildly hot or cold stimuli can bring more awareness that that side of the body is still there. Some Adaptive Strategies could include removing clutter on the affected side, approaching patients from their midline, placing objects in the midline, and gradually moving them further into the patient's affected side, and encouraging patients to turn their heads to become more aware of the affected side.[37,66]

Constraint-Induced Movement Therapy (CIMT) is an intervention that improves upper extremity function in stroke and other CNS damage patients by increasing the use of their affected side. Fig. 17 shows that in this therapy, a sling or mitt is placed on the unaffected arm to keep it from taking over. This therapy is an attempt to reverse the learnt non-use of the affected arm. This type of therapy can only be used if the patient is physically able to use their affected arm, and have simply forgotten about it due to unilateral neglect.[51]

Figure. 17 Constraint-Induced Movement Therapy (CIMT)[51]

Visual Discrimination Interventions

Visual Discrimination is the ability to distinguish one object from another. This includes form discrimination (having trouble differentiating between objects that are subtly different, like a water jug, or glass cup), depth perception impairment, trouble distinguishing between background and foreground, and difficulty perceiving the positions of two objects in relation to one another or oneself.

The aim of visual discrimination interventions is for the patient to become aware of the relationship of objects or self, identify foreground from background, and understand an object's position in space, depth, and distance. A good restorative strategy is to teach a patient to follow verbal instructions with spatial concepts. For example, the OT might say "get the remote on top of the TV". Others include teaching patients to place different items in different parts of the room and to encourage patients to verbalize the position of the parts of their body to improve awareness of where they are in relation to objects around the room. Some adaptive strategies include organizing objects so that they are in the same place, marking drawers where items are kept, removing clutter, and placing objects on contrasting surfaces.[11,25]

Interventions for Visual Agnosia

Visual Agnosia is the inability to recognize visual stimuli despite adequate primary visual function. A person with Visual Agnosia might have trouble identifying objects, as well as their colors and sizes and may also have trouble distinguishing between the faces of different individuals. Apperceptive Agnosia is where recognition fails because of impairment to visual perception. Patients do not see objects normally and cannot therefore respond to them. Associative Agnosia is when a person's perception is intact to allow recognition, but may have

trouble with naming the object. Patients with Apperceptive agnosia are unable to copy drawings or match objects, and patients with Associative Agnosia are able to copy drawings, but cannot describe the name or functions of objects.[14]

The goal of Occupational Therapy Interventions are to get patients to be able to identify objects through vision. Some strategies using the restorative approach include presenting objects to a patient in a straight position, rather than a different orientation and encouraging patients to verbalize similarities and differences of objects. Tome strategies using the adaptive approach include providing labels for different objects, adding texture or edge orientation to objects, using other senses like touch, smell, or sound to identify objects, and encouraging the patient to verbally describe perceptual and functional characteristics of objects to aid retrieval of the object name.[14,25]

Interventions for Tactile Agnosia

Tactile Agnosia is when a person is able to perceive weight and texture of an object, but can neither describe it by name nor comprehend its significance nor meaning.

The aim of Occupational Therapy Interventions for Tactile Agnosia is to identify objects through touch. The restorative strategy used by OT's is exploratory hand movements for object identification. This includes touching the surfaces and edges of objects and holding objects in the hands to obtain information on weight, size, and shape. The Adaptive Strategy includes using other senses, like vision, and touch from the unaffected hand, using familiar objects within daily functional tasks, and teaching patients to focus on specific properties of objects rather than all of the different properties.[27]

Motor Control Rehabilitation

Motor control impairments include things like hemiplegia, hemiparesis, and trouble maintaining balance, which are usually the result of an occlusion of the complete MCA or the Vertebral Basilar System. Because the impact of motor impairments after a stroke can be so devastating to a patient, the role that an Occupational Therapist plays in managing these problems is vital for learning to live as independently as possible. After assessing the different movements of a patient after a stroke, including standing, sitting, walking, bed mobility, transfers, and ADL's, the OT can see how these patients are moving, why they are moving that way. This helps them to know exactly what to target through rehabilitation interventions. The different interventions for motor impairments offered should include three different things. First, the opportunity to practice activities in the most natural, home-like setting possible. Second, training in the use of equipment and adaptations that create safe independence. Third, the training of family members and caregivers in helping the patient. In addition to this, task specific training in things like standing up, sitting down, gait speed, and gait endurance should be used to improve mobility and ADL's.

The main aims of Occupational Therapy intervention regarding motor problems is to increase functional independence by practicing graded activities of daily living, to prevent secondary complications, and to train caregivers in safe techniques for handling patients based on functional level and equipment needs.[25]

Positioning the Early Stroke Patient

While working with a patient after they have had a stroke, it is important to consider the functional activities an individual wishes to achieve, in any given posture. In the early stages of

recovery, when movements are restricted by the effects of their stroke individuals are unlikely to make the postural adjustments required to maintain symmetrical posture on their own. Those with sensory loss on the hemiplegic side may fear laying on that side, but if they lay on their unaffected side, this restricts movements. Correct positioning for sleeping and the early development of functional mobility should be focused on by the OT. It is not good to have a hemiplegic patient laying on their back all of the time, so it is imperative that patients are moved into different positions frequently, and are supported in all of the right areas, depending on which position they are in. Support with a pillow should be offered where required to enable the individual to maintain their position. If a person is laying on their back, it is important to make sure their affected side is supported by placing the pillow in a position that supports both the shoulder and the hip. If a person is lying on their side, this includes support along the back to prevent rolling. The affected side of the patients should always be straight, with the arm in a protracted position and the leg unbent. Sometimes, OT's will position patients lying on their unaffected side, if that side is overactive. This restricts that side from fully taking over. The correct positioning for a left hemiplegic patient, laying on their side can be seen in Fig. 18. The Hemiplegic side is shown in grey. When sitting up in a bed, it is important for a hemiplegic patient to have an even distribution of weight on both sides, a protracted shoulder away from the side and forward on a pillow, and straight legs.[25,47]
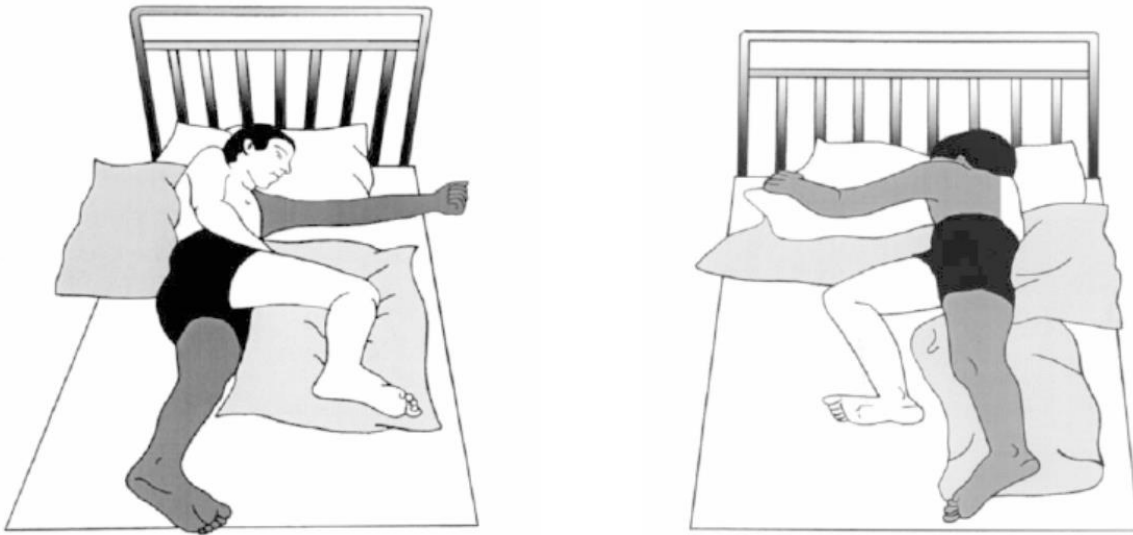
Figure. 18 Positioning of hemiplegic stroke patient while laying in bed [25]

More independence is offered to the early stroke patient when they are sitting in a chair. They have a more normal visual perspective and there is scope for the unaffected arm to be used in a range of functional activities. The trunk muscles begin to be used actively and the lower limbs begin to form a stable base of support. Support from pillows should still be used, because the affected side of a patient may not be strong enough to support full body weight. Armchairs tend to also provide more support. If a patient is able to sit in a wheelchair, this is helpful for OT's because this makes transportation from one place to another much easier. A correctly fitted wheelchair provides a more active sitting posture, encouraging greater freedom for upper limb movements. Over time, as patients begin to get stronger, transfers from the bed to a wheelchair become easier.[47]

Interventions for ADL's

Self-Care Activities

All self-care activities should be graded depending on a patient's level of functioning. After the OT figures out what the goal of each sessions should be, they should spend time preparing the environment and gathering the necessary items needed in order to make a comfortable environment, but still challenge the patient. The focus is on the restorative approach, but it is common to teach some adaptive techniques early to maximize early dependence.[30,78]

Personal Washing

In the early stages post-stroke, a patient may have trouble balancing while sitting. The emphasis for this stage is to make sure that the patient has good positioning and support from the chair. Low attentional demands should be practiced, such as shaving, combing hair, or washing the face. As the patient regains more stability and is able to maintain their position on the chair better, the emphasis can be moved on washing from the waist up. The patient can be positioned in front of a sink of the appropriate height and can practice reaching for things and using them for washing. During this stage, the OT is focused on making sure the patient has correct alignment while sitting and maintains balance while reaching for what they need. As time goes on, the patient's balance on a chair will have improved tremendously, making transfers much easier, both for the OT and the patient. Instead of self-washing at a sink, the patient may be able to transfer from a wheelchair to a shower chair with assistance. Eventually, standing in front of the sink or in the shower may be incorporated into the practice sessions. The goal for this progression of graded activities would be for the patient to be as independent as possible, washing in the manner of their choice.[25,30,78]

Dressing and Undressing

Hemiplegic patients early post stroke can be taught one handed dressing techniques while seated in a wheelchair or armchair. These sessions would also focus on sitting, balance, position, trunk control, and movements of the unaffected upper limb. Patients with improving trunk control can be taught dressing techniques on a bench-like structure called a plinth. When a patient is sitting on a plinth, this is a good time for the OT to work with them on lower body dressing techniques and to practice standing. The goal of practicing these techniques during Occupational Therapy sessions is to allow a patient to be able to do this on their own in a normal environment, like sitting on their bed at home or standing in their own bedroom or bathroom. Adaptations such as Velcro or elastic shoelaces, or teaching the patient a one-handed shoe tying technique can be used to make things easier on the patient as they are regaining their skills. Also, patients might initially wear leisure wear that is easy to slip on until they become proficient at dressing techniques or their motor and cognitive problems improve. They can then practice dressing with the clothing of their choice. The OT needs to be sure to discuss any changes in clothing style with the patient to make sure they are not trying to change the individual's autonomy of self-image.[25,30,78]

Instrumental Activities

Graded Kitchen and Household Tasks

When using interventions in a kitchen setting, an OT needs to be creative and consider the patient's preferences, goals, culture, religion, and previous roles. Medical status regarding level of exertion should also be considered. Before a patient goes into a kitchen rehabilitation setting, an OT might have them engage in therapeutic tabletop exercises like playing cards,

dominos, Connect 4, writing exercises, turning pages in a book, or using the mouse on a computer to help with upper limb movements. The patient's goals and interests should be considered when choosing these activities. At the beginning of being in a kitchen setting, a patient with poor sitting balance could be set up at a table and asked to complete a task, such as making toast, cereal, or a hot drink. As the patient improves, standing and reaching for items in cupboards can be incorporated into the sessions. For mobile patients, walking should be assisted as necessary and tasks should involve moving around the kitchen and transporting items between surfaces. Specialized kitchen equipment, such as non-slip mats, pan holders on the stove, adapted knives, electric can openers, and rolling trays for transporting items can be used to help make the process easier for accomplishing these kitchen tasks. A patient with dysphagia can also go through exercises that help them to eat and drink more easily. Adaptations like using straws when drinking and taking smaller bites at a slower rate can help these patients. Similar graded activities should be used for practice with other household tasks, such as laundry, vacuuming and cleaning around the house. These tasks can either be practiced in a rehabilitation setting, or in the patient's own home with a home health OT.[44,78]

Interventions for Emotional Disturbances

If an OT has any concerns regarding a patient's emotional state, such as severe mood swings, fatigue, or depression, further in-depth assessment by a psychologist might be required. If the OT notices these disturbances, it is their responsibility to notify the consultant and other members of the multidisciplinary team who are more equipped to deal with these types of problems.[5,25,90]

Step 3: Outcome Evaluation

Evaluation of Occupational Therapy effectiveness is an ethical and professional imperative. Ongoing evaluation enables both the patient and the therapist to monitor the appropriateness of intervention and allow opportunities for adjustment. Evaluation of patient outcomes determines if the therapy has been successful, whether or not goals have been met, and if the patients are satisfied with the intervention. Evaluations allow therapists to evaluate their practice against both profession-specific and stroke-specific standards and guidelines. By doing this, they are also able to evaluate their practice as well as consider intervention efficiency and cost effectiveness.[25]

<u>Importance of a Team</u>

Post stroke neurorehabilitation is evolving into a field dominated by multidisciplinary interactions and collaborations, that allow professionals to work together towards the goal of improving the quality of life of patients with stroke.[23] A multidisciplinary team is a collection of professionals from different disciplines who share a common idea of working practice. Interdisciplinary teamwork (IDT) is the key to success when dealing with a stroke patient. Team members not only perform activities towards a common goal, but they also accept the added responsibility of group effort on behalf of patients. In IDT, members contribute different professional perspectives, but goal setting, care planning, and decision making are collaborative activities. This collaboration can occur weekly or more frequently through ongoing, patient-focused dialogue. Team members must develop understanding of each other's roles and recognize where overlap occurs. This understanding and acceptance of blurring of role boundaries facilitates rapid information exchange, enables early interventions, and underpins

effective rehabilitation in secondary care, early supported discharge, and in longer stroke care in community settings.[21]

Some professionals included in this IDT would be physicians. physician assistants (PA), physical therapists, occupational therapists, clinical social workers, nurses, psychologists, dieticians, and speech and language pathologists. Physicians, assisted by PA's orchestrate the entire rehabilitation program and manage medical needs, by prescribing medications and ordering tests that may be beneficial to the patient. When it comes to PT's and OT's, there is definitely professional skill overlap but each has a specific emphasis in the rehabilitation of a stroke patient. Physical Therapists focus on the prevention of joint and tissue injury and retraining lost motor skills. The Occupational Therapist considers the total patient in his or her environment and assists the patient in regaining or improving function in all areas of daily living. Although these are very different areas of focus, both are equally important. Clinical Social Workers concentrate on psychosocial assessment and intervention with the patient and their family/caregiver, aiding them in the adjustment process and planning for discharge. Nurses help to educate patients on the big picture of life after stroke, including how to take medications, what to expect during the recovery process, and how to prevent another stroke from occurring. Dieticians help stroke patients to take the steps to avoid a second stroke, by promoting healthy eating that combat stroke risk factors like high blood pressure, excess weight, and unhealthy cholesterol levels. Psychologists step in when emotional disturbances require attention, and speech and language pathologists assist with improving communication and working with dysphagia patients to improve their ability to swallow a greater variety of foods and liquids.[12]

While an Occupational Therapist plays a vital role in the rehabilitation of a stroke patient, there is no possible way that the overall rehabilitation process would be successful if they work

alone. An Occupational therapist needs to be patient focused and be a team player. If all members in the IDT have this mindset, their overall success rate will be higher and well-being of the patient post-stroke will be the best possible scenario and outcome.

Conclusion

This in-depth research regarding the different types of cerebrovascular accidents, their causes, risk factors, and the epidemiology of who they affect. Deep research of the brain was performed through cadaver dissections, identifying the different structures and their functions. Knowledge of this anatomy and how the blood supplies the brain helps to identify signs and symptoms that may occur and correlated those for diagnosis and treatment in the stages of recovery. The Occupational Therapist is a key player in assisting post stroke rehabilitation. Occupational Therapy focuses on how to best help patients participate as independently as possible when performing everyday Activities of Daily Living (ADL) The overall process is broken down into three steps: assessment, intervention strategies, and outcome evaluations. These three steps are individualized for each patient. Overall, the Occupational Therapist is part of an Interdisciplinary team of professionals who all have different focuses, but have a common goal of helping the stroke patient recover from their injuries.

While this literature review mostly focused on acute occupational therapy rehabilitation strategies, my future studies will take me into detail understanding and practice of long term strategies to maintain status after being released from acute rehabilitation centers. More detail about how home healthcare Occupational Therapists help patients with resettlement in their homes and how patients are able to eventually get back to bigger picture activities like working or driving cars.

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