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****
THE UNIVERSITY OF YAOUNDE I
****
FACULTY OF MEDICINE AND BIOMEDICAL SCIENCES (FMBS)
****

DEPARTMENT OF INTERNAL MEDICINE AND SPECIALTIES

POST-STROKE PSYCHIATRIC DISORDERS: PREVALENCE AND EFFECTS ON FUNCTIONAL RECOVERY OF STROKE SURVIVORS AT THE YAOUNDE CENTRAL HOSPITAL

Thesis presented and publicly defended in partial fulfilment of the requirements for the award of Doctorate Degree in General Medicine

by

THOM NKUIDJEU CLAUDE

MATRICULE: 17M066

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ACADEMIC YEAR: 2023-2024

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Publicly defended on

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President of Jury

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Examiner:

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ACADEMIC YEAR: 2023-2024

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DEDICATION

To

My beloved parents, Mr. Thomfeum Zephirin and Mrs. Tchuenkam Rolande

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KEY

HOD = Head of Department

P= Professor

AP= Associate Professor

SL= Senior Lecturer

L= Lecturer

HIPPOCRATIC OATH

Declaration of Geneva adopted by the 2nd General Assembly of the World Medical Association, Geneva, Switzerland, September 1948 and amended by the 22nd World Medical Association General Assembly, Sydney, Australia (August 1968).

On admission into the medical profession;

I will solemnly pledge myself to consecrate my life to the service of humanity

I will give my teachers the respect and gratitude which is their due

I will practice my profession with conscience and dignity

The health of my patients will be my first consideration

I will respect secrets confided in me even after the patient has died

I will maintain by all means in my power the honour and noble tradition of the medical profession

My colleagues will be my brothers

I will not permit considerations of religion, nationality, race, political party or social standing to intervene between my duty and my patient

I will maintain the utmost respect for human life from the time of conception

Even under threat I will not use my medical knowledge contrarily to the laws of humanity

I make these promises solemnly, freely and upon my honour

ABSTRACT

Background: Stroke is the second leading cause of death worldwide and a leading cause of acquired physical disability in adults. Stroke survivors experience various psychiatric disorders which may impact recovery and lead to dependence on caretakers.

Objective: To determine the prevalence of psychiatric disorders post-stroke and their effect on the functional recovery of stroke survivors.

Methodology: We conducted a 7-month hospital-based analytical cross-sectional study at the Yaoundé Central Hospital. We included all adult stroke patients between two weeks and twelve months post-stroke, followed at the Neurology service of the hospital. Patients with severe cognitive impairment and communication difficulties were excluded. Relevant socio-demographic and clinical data was collected from participants, standardized tools were used for the assessment of various post-stroke psychiatric disorders. Data was compiled and analysed using the SPSS software and results presented.

Results: A total of 96 participants were included in our study and their data analysed. There were more females (52.1%) in our population and the mean age was 61.1±13.0 years. Ischemic stroke was more represented (68.8%), the median duration of stroke was 1.8[0.8-4.5] months, the median NIHSS score was 1[0-3.75] and 59.4% of participants had a good functional outcome (mRS<3) upon recruitment. Up to 36 people (37.5%) had a psychiatric disorder post-stroke. Psychiatric disorders found were distributed as follows: Post-Stroke Depression (PSD) in 28.1%, Post-Stroke Anxiety (PSAn) in 5.2%, Post-Stroke Apathy (PSA) in 24%, Post-Stroke Fatigue (PSF) in 15.6% of our study population. Post-stroke mania and psychosis were not present in the population. Low level of education (p=0.04), low BMI (p=0.016) and sedentary lifestyle (p=0.002) were associated with PSD; sedentary lifestyle (p=0.016), side of brain lesion (p=0.02), stroke severity (p=0.006) and fatigue (p<0.001) were associated with PSA, while older age (p=0.03) male sex (p=0.032), PSD (p<0.001) and antidepressant use (p=0.02) were associated with PSF. We found no factors associated with PSAn in our study. PSD and PSA were significantly associated with a poor functional outcome (p=0.001, p<0.001 respectively), PSF was only close to significance.

Conclusion: Two out of five stroke patients in our context will likely develop a psychiatric disorder following their stroke. A sedentary lifestyle is strongly correlated to the development of these post-stroke psychiatric disorders, and they in turn are often associated with poor functional recovery in stroke survivors.

Keywords: Post-stroke; psychiatric disorders; functional recovery; Yaoundé

RESUME

Contexte : L'Accident Vasculaire Cerebral (AVC) est la deuxième cause de décès dans le monde et la principale cause d'handicap acquis chez l'adulte. Les survivants d'AVC pourraient être atteint de divers troubles psychiatriques qui peuvent avoir un impact sur le rétablissement et conduire à une dépendance vis-à-vis des gardes malades.

Objectif : Déterminer la prévalence des troubles psychiatriques post-AVC et leur effet sur la récupération fonctionnelle des survivants d'AVC.

Méthodologie : Nous avons mené une étude transversale analytique pendant 7 mois à l'Hôpital Central de Yaoundé. Nous avons inclus tous les patients adultes victimes d'AVC, entre deux semaines et douze mois après l'AVC, suivis au service de neurologie de l'hôpital. Les patients présentant des troubles cognitifs sévères et des difficultés de communication ont été exclus. Les données sociodémographiques et cliniques pertinentes ont été collectées à l'aide d'outils standardisés. Les données ont été compilées et analysées à l'aide du logiciel SPSS et les résultats présentés.

Résultats : Au total, 96 participants ont été inclus dans notre étude et leurs données ont été analysées. Il y avait plus de femmes (52,1 %) dans notre population et l'âge moyen était de 61,1±13,0 ans. L'AVC ischémique était le plus représenté (68,8 %), la durée médiane de l'AVC était de 1,8 [0,8-4,5] mois, le score de NIHSS médian était de 1 [0-3,75] et 59,4 % d'entre eux avaient un bon résultat fonctionnel lors du recrutement. Environ 36 personnes (37,5 %) souffraient d'un trouble psychiatrique post-AVC. Les troubles psychiatriques post-AVC retrouvés étaient repartis comme suit : Depression Post AVC (DPAVC) chez 28,1 %, Anxiété Post AVC (AnPAVC) chez 5,2 %, Apathie Post AVC (APAVC) chez 24 %, Fatigue Post AVC (FPAVC) chez 15,6 % de notre population d'étude. La manie et la psychose post-AVC n'ont pas été retrouvées dans la population. Le bas niveau d'éducation ($p=0.04$), un Indice de Masse Corporelle (IMC) bas ($p=0.016$) et le mode de vie sédentaire ($p=0.002$) étaient associés à la DPAVC ; le mode de vie sédentaire ($p=0.016$), le côté de la lésion ($p=0.02$), la sévérité de l'AVC ($p=0.006$) et la fatigue ($p<0.001$) étaient associés à l'APAVC, tandis que l'age > 65 ans ($p=0.03$), le sexe masculin ($p=0.032$), la DPAVC ($p<0.001$) et l'utilisation d'antidépresseurs ($p=0.02$) étaient associés à la FPAVC. Nous n'avons retrouvé aucun facteur associé à l'AnPAVC dans notre étude. La DPAVC et l'APAVC étaient associées à un mauvais résultat fonctionnel ($p=0.001$, $p<0.001$ respectivement), la FPAVC n'était que proche de la significativité.

Conclusion : Deux patients sur cinq victimes d'AVC dans notre contexte peuvent avoir un trouble psychiatrique suite à l'AVC. Le mode de vie sédentaire est fortement corrélé au développement de ces troubles psychiatriques post-AVC, et ils sont à leur tour souvent associés à une mauvaise récupération fonctionnelle chez les survivants d'AVC.

Mots-clés: Post-AVC; Troubles psychiatriques; Récupération fonctionnelle; Yaoundé

LIST OF ABBREVIATIONS

ABC	:	Airway Breathing Circulation
ACA	:	Anterior Cerebral Artery
ACEI	:	Angiotensin-Converting Enzyme Inhibitor
ADL	:	Activities of Daily Living
AF	:	Atrial Fibrillation
AHA	:	American Heart Association
AHA/ASA	:	American Heart Association/American Stroke Association
AnPAVC	:	Anxiété Post Accident Vasculaire Cerebral
APAVC	:	Apathie Post Accident Vasculaire Cerebral
ART	:	Antiretroviral Therapy
ASA	:	American Stroke Association
ATP	:	Adenosine Triphosphate
AVC	:	Accident Vasculaire Cerebral
BI	:	Barthel Index
BMI	:	Body Mass Index
BP	:	Blood Pressure
CAA	:	Cerebral Amyloid Angiopathy
CENAME	:	National Central of Essential Medicine
CSVD	:	Cerebral Small Vessel Disease
CT	:	Computed Tomography
DBP	:	Diastolic Blood Pressure
DPAVC	:	Depression Post Accident Vasculaire Cerebral
FBG	:	Fasting Blood Glucose
FMBS	:	Faculty of Medicine and Biomedical Sciences
FPAVC	:	Fatigue Post Accident Vasculaire Cerebral
FSS	:	Fatigue Severity Scale
GV	:	Galen Vein
HADS	:	Hospital Anxiety and Depression Scale
HIV/AIDS	:	
HTN	:	Hypertension
IBM SPSS	:	International Business Machines Statistical Package for Social Sciences

ICA	:	Internal Carotid Artery
ICH	:	Intracerebral Hemorrhage
ICP	:	Intracranial Pressure
IJV	:	Internal Jugular Vein
INR	:	International Normalised Ratio
LMICs	:	Low and Middle Income Countries
LOC	:	Loss of Consciousness
LSD	:	Lysergic acid Diethylamide
MAOI	:	Monoamine Oxidase Inhibitors
MCA	:	Middle Cerebral Artery
MD	:	Medicinae Doctor
MRI	:	Magnetic Resonance Imaging
MRS	:	Modified Rankin Scale
NIHSS	:	National Institute of Health Stroke Scale
NMDA	:	N-Methyl-D-Aspartate
OAD	:	Oral Anti-diabetic agent
PCA	:	Posterior Cerebral Artery
PCP	:	Phencyclidine
PHQ-9	:	Patient Health Questionnaire-9
PSA	:	Post Stroke Apathy
PSAn	:	Post Stroke Anxiety
PSD	:	Post Stroke Depression
PSF	:	Post Stroke Fatigue
PSM	:	Post Stroke Mania
PSP	:	Post Stroke Psychosis
RAS	:	Reticular Activating System
Rt-PA	:	Recombinant tissue-type plasminogen activator
SAH	:	Subarachnoid Hemorrhage
SAS	:	Starkstein Apathy Scale
SBP	:	Systolic Blood Pressure
SNRI	:	Serotonin and Norepinephrine Reuptake Inhibitors
SSA	:	Sub-Saharan Africa
SSRI	:	Selective Serotonin Reuptake Inhibitors
TIA	:	Transient Ischemic Attack

USB	:	Universal Serial Bus
VVP	:	Vertebral Venous Plexus
WHO	:	World Health Organization
WMA	:	World Medical Association
YCH	:	Yaoundé Central Hospital

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CHAPTER I : INTRODUCTION

I.1. BACKGROUND AND RATIONALE

Stroke or cerebrovascular accident is the second leading cause of death, and the leading cause of acquired physical disability in adults worldwide[1]. It is defined by the World Health Organization (WHO) as rapidly developing clinical signs of focal or global disturbance of cerebral function lasting 24hours or longer or leading to death, with no apparent cause other than of vascular origin[2,3]. Stroke is a huge and increasing global health problem, affecting roughly 13.7 million people per year, responsible for about 5.5 million deaths with about 5 million who are left disabled[4]. Globally 70% of strokes and 87% of stroke-related deaths and disability adjusted life years occur in low and middle income countries (LMICs) in Africa and other continents. The incidence of stroke in LMICs has almost doubled in the last four decades meanwhile it has decreased by 42% in high income countries over this same period[4,5]. In Cameroon, stroke has a prevalence of 7.3% with a mortality rate of 26.7% during the first month and 31.7% within the first three months[6].

Stroke survivors may experience complications such as stroke recurrence, cognitive impairment and neuropsychiatric disorders among others. Neuropsychiatric disturbances are a common but challenging consequence of stroke as they arise at the intersection of lesion-related brain dysfunction and psychological distress due to the event and its aftermath[7]. These disorders can be briefly divided into post-stroke neurological diseases (including mainly post-stroke pain, post-stroke epilepsy, post-stroke dementia) and post-stroke psychiatric disorders (including mainly post-stroke depression, post-stroke anxiety, post-stroke apathy and post-stroke fatigue) [8]. Post-stroke psychosis, mania and obsessive compulsive disorders are less commonly encountered [7]. Neuropsychiatric sequelae are disabling, have a negative impact on recovery, reduce quality of life and lead to exhaustion of patients' caretakers[9].

Post-stroke depression (PSD) is the most frequent and most studied post-stroke psychiatric complication, with a global prevalence that ranges from 18 to 33%[10]. PSD has as main risk factors stroke severity, large left hemisphere lesion and lack of social support. Patients who suffer from PSD are more vulnerable to cognitive disorders, suicidal ideas and long-term disability[11]. Post-stroke anxiety (PSAn) is also a frequent post-stroke psychiatric complication, with a pooled prevalence of 29.3%[12], mostly seen amongst youths and in females. The main risk factors concerned with PSAn are post-stroke depression, pre-stroke anxiety or depression, lack of sleep, and coping[11]. Post-stroke apathy (PSA) has an estimated prevalence of 25%[11] and is usually misdiagnosed as depression since both would manifest as inactive mood[8]. It is associated with a higher risk of depression and poor functional outcome[13]. Post-stroke psychosis previously considered rare was found to have a prevalence

of 4.87%, mostly in elderly males with an average age of 66 years[14]. A review done by Cumming *et al* reported a pooled prevalence for post-stroke fatigue of up to 50% among stroke survivors[15]. Post-stroke mania is rare, with a prevalence of < 2%[13]. It's onset is usually between the first days until 24 months after the stroke with the typical patient described as being a male with high cardiovascular risk factors and having a lesion in the right hemisphere[13].

In Africa, few studies have been published on the prevalence of psychiatric complications after stroke. Most of the studies highlighted PSD and a few others mentioned PSAn. In 2017, Ojagbemi *et al* reported a pooled frequency of 31% for PSD in Sub-Saharan Africa (SSA)[16] while Wubshet *et al* found a point prevalence of 27% for PSD in Ethiopia[17]. In another study, Ojagbemi *et al* reported a prevalence of 19.7% for PSAn in SSA with over 70% of patients suffering from depression as a comorbidity[18].

In 2021, Angwafor *et al* found a prevalence of 45.6% for PSD in Cameroon [19]. There is paucity of data on the psychiatric complications of stroke in our context and on their effect on stroke survivors' recovery. We therefore sought to carry out this study to investigate and highlight the occurrence of various psychiatric complications among stroke survivors, in order to inform management strategies and guidelines, thus contributing to improve the clinical outcome of stroke survivors in our setting.

I.2. RESEARCH QUESTIONS

1. What is the prevalence of psychiatric disorders following stroke among stroke survivors in the Yaoundé Central Hospital?

2. How does the occurrence of post-stroke psychiatric disorders affect the functional recovery of stroke survivors in the Yaoundé Central Hospital?

I.3. OBJECTIVES

I.3.1. General Objective

To investigate psychiatric disorders following stroke, their prevalence and their effect on functional recovery amongst stroke patients in the Yaoundé Central Hospital

I.3.2. Specific Objectives

1. To determine the prevalence of psychiatric disorders occurring after stroke, in stroke patients followed at the Yaoundé Central Hospital (YCH)
2. To describe the factors associated with post-stroke psychiatric disorders among stroke patients followed at the YCH

3. To evaluate the effect of post-stroke psychiatric disorders on the functional recovery of stroke patients followed at the YCH.

I.4. OPERATIONAL DEFINITION OF TERMS

1. **Stroke:** WHO defines stroke as rapidly developing clinical signs of focal (or global) disturbance of cerebral function with symptoms lasting 24hours or more or leading to death, with no apparent cause other than of vascular origin[2]. In our study we consider any patient who suffered such sudden neurological deficit for which the diagnosis of stroke was confirmed by a neurologist and/or imaging.
2. **Stroke survivor:** A patient who has had a stroke attack and is still alive. For this study we particularly considered stroke survivors at 2 weeks post-event at least.
3. **Psychiatric disorders post-stroke:** These are psychiatric disorders occurring for the first time in a stroke survivor after the stroke event. For this study, these disorders will comprise depression, anxiety, apathy, fatigue, psychosis and mania.
4. **Functional recovery:** This is the degree to which a stroke survivor can return to his/her abilities, activities or functions as they were prior to the stroke event. It is also the ability of the patient to re-adapt into the society in his new condition following the stroke attack.

CHAPTER II : LITERATURE REVIEW

II.1. STROKE

II.1.1. Introduction

II.1.1.1. Definition

Stroke is defined by WHO as rapidly developing clinical signs of focal (or global) disturbance of cerebral function with symptoms lasting 24hours or more or leading to death, with no apparent cause other than of vascular origin[20].

II.1.1.2. Epidemiology

Stroke is an increasing global health challenge. It is the third leading cause of death in the world after coronary heart disease and cancer, and a leading cause of acquired physical disability in adults[1]. In the last few decades, the incidence of stroke has declined by about 42% in High income countries, while it has doubled in LMICs[4]. About 85% of all stroke deaths in the world occur in LMICs in Africa and other continents, accounting for over 87% of disability-adjusted life years[1].

In Cameroon, the prevalence of stroke has been found to be 7.3% with a mortality rate of 26.7% at 1 month and 31.7% at 3 months after stroke[19].

Stroke is generally classified into two main types; Ischemic stroke (interruption of blood supply to a part of the brain due to a clot formation, resulting in sudden loss of cerebral function) and Hemorrhagic stroke (interruption of blood supply to a part of the brain due to rupture of a blood vessel supplying the area or an abnormal vasculature)[3]. Hemorrhagic stroke again is of two types; intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Ischemic strokes account for about 80% of all strokes while hemorrhagic strokes account for 20% of strokes. ICH accounts for 10-15% 0f all strokes, that is 80% of hemorrhagic strokes[3].

Transient Ischemic Attack (TIA) is another type of stroke. It is defined as a transient episode of focal neurological dysfunction caused by focal brain, spinal cord or retinal ischemia without acute infarction[21]. This definition seems more accurate as opposed to the previous one, which talked of focal neurological deficit lasting less than 24 hours, as it included patients who suffered minor stokes (defined as a stroke that leaves a patient without significant physical disability). Patients with TIA clinically present with sudden onset of neurological symptoms lasting less than an hour. Studies showed that misdiagnosis of TIA is high (60%), the incidence rate of stroke within the next 48hours after TIA is 4.8% and secondary prevention of stroke in TIA cases reduces the outcome of stroke by 80%[21].

II.1.1.3. Risk Factors

The risk factors for stroke can be classified into Non-modifiable and Modifiable risk factors

- **Non-Modifiable Risk Factors**

- **Age**; the incidence of stroke increases with age and doubles for each decade after 55years[1,4]

- **Sex**; the occurrence of stroke is higher in younger ages in women, probably due to factors related to pregnancy, hormone therapy, oral contraceptive use, pre-eclampsia[1]. At older ages, stroke rates are higher in men than in women.

- **Race**; the incidence of stroke is doubled in young black adults compared with age-matched white counterparts.

- **Genetics**; added to the genetic disorders that are associated with stroke (Fabry's disease, homocystinuria, sickle cell disease, connective tissue disorders etc.) about 22 new gene loci have been identified, some of which are linked to specific stroke mechanisms while others are associated to vascular pathologies.

- **Modifiable Risk Factors[4,22]**

They are of great importance because appropriate and timely medical intervention upon these can reduce the risk of stroke for individuals who are susceptible.

- **Hypertension**; it is the most important modifiable risk factor for stroke and is defined by WHO as a blood pressure (BP) greater than or equal to 140/90 mmHg. Over half of stroke patients have a history of hypertension and even among non-hypertensive patients, the higher the BP, the higher the risk of stroke. This is why diagnosis and control of hypertension is key in the prevention of stroke.
- **Diabetes Mellitus**; (FBG level > 1.26g/l OR random blood glucose level >2g/l OR a known diabetic patient) it is an independent risk factor for stroke, yielding a 2-fold increased risk with an increased mortality of about 20%. The prognosis for stroke in diabetic patients is worse than in non-diabetic patients as they exhibit more severe disability and slower recovery.
- **Cardiac Factors**; cardioembolic infarction (mainly from atrial fibrillation (AF)) is the most severe ischaemic stroke subtype, with high disability and mortality, contributing close to 15% of all strokes. It increases the risk of stroke 2 to 5 folds.
- **Hyperlipidemia**; There is an increased risk of ischaemic stroke with increased total cholesterol (>2g/l), and a decreased risk of ischaemic stroke with elevated high-density

lipoprotein-cholesterol (>0.40g/l). The use of statins in secondary prevention appears to reduce the risk of ischaemic stroke as well as mortality.

- **Smoking**: the risk of stroke doubles with smoking.
- **Alcohol**; daily alcohol intake >40g/l increases the risk of stroke
- **Substance abuse**; Illicit use of drugs such as cocaine, heroin, phencyclidine (PCP), lysergic acid diethylamide (LSD), cannabis/marijuana or amphetamines is a common predisposing factor for stroke among individuals aged below 35 years.
- **Obesity and sedentary behaviour:** the effect of body mass index on stroke risk is mediated by blood pressure, cholesterol and glucose concentrations thus people who are physically inactive have a higher risk of stroke than those with considerable daily exercise.
- **Inflammation**: raised inflammatory biomarkers have a modest association with increased risk of arteriosclerosis and stroke[4].

II.1.2. RECALL

II.1.2.1. Anatomy Of The Brain[23–27]

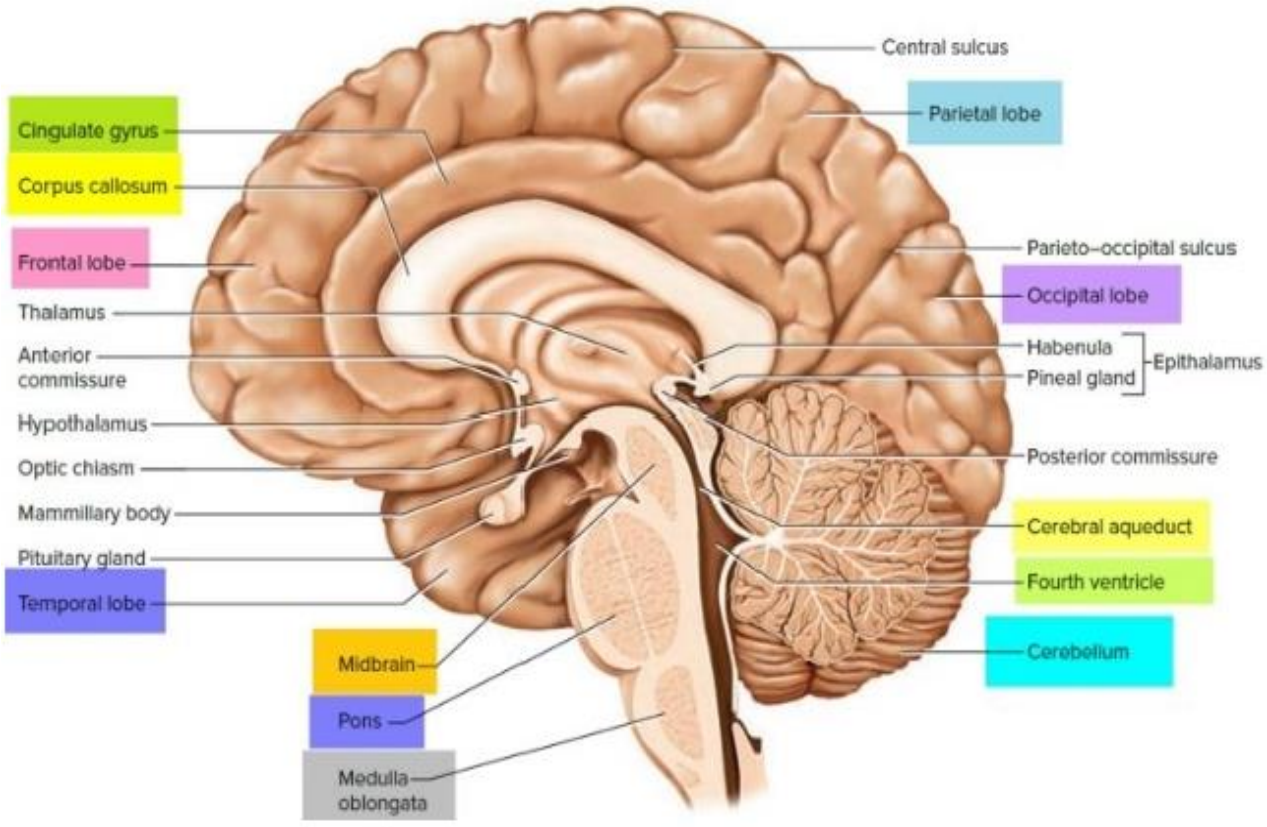

Figure 1: Sagittal section through the brain [28]

The brain is an impressive organ of the human body with remarkable abilities. It is the largest and most complex mass of nervous tissue in the body, weighing about 1.5Kg and located in the skull. It is usually divided into four main regions; the cerebral hemispheres or cerebrum, the diencephalon, the cerebellum and the brain stem.

- **The Cerebrum**

It is the most superior part of the brain, made of both a right and a left hemisphere separated by a longitudinal fissure. The surface of each cerebral hemisphere exhibits elevated ridges of tissue called gyri separated by shallow grooves called sulci. There are also deeper grooves called fissures, which separate the hemispheres into four different lobes. The two hemispheres communicate with each other through the corpus callosum. Each cerebral hemisphere has 3 main regions; the superficial cortex or gray matter (named as such because of its unmyelinated axons that appear gray) interprets received signals from and sends respond signals to the body, the inner region of white matter (named as such because of its myelinated axons that appear white) mainly constitutes fiber tracts for interconnection and communication among various parts of the brain and the basal nuclei, islands of gray matter buried deep in white matter that mainly help regulate the initiation and stopping of voluntary motor activities. Each cerebral hemisphere is divided into 4 lobes namely; the frontal, parietal, temporal and occipital lobes.

- **The Frontal Lobe**

The frontal lobe is anterior to the central sulcus, which separates the frontal and parietal lobes. It divides into a superior, middle, and inferior frontal gyrus, primary motor cortex, and orbital area. These areas combine to control our executive and motor functions. It controls judgment, problem-solving, planning, behavior, personality, speech, writing, speaking, concentration, self-awareness, and intelligence. The primary motor cortex is present in the precentral gyrus of the frontal lobe and is positioned immediately anterior to the central sulcus. The premotor cortex is anterior to the primary motor cortex. This area controls contralateral body and limb movement. The medial region controls the lower limb while the supero-lateral region controls the upper limb and hand. The lateral region controls the face. The majority of the primary motor cortex is used to finely control the muscle of the hands, face, and lips, which is well represented by the homunculus model. Within the middle frontal gyrus is the frontal eye field area and it is mostly responsible for the contralateral eye abduction and ipsilateral eye adduction. Broca's area is responsible for speech and is present only within the inferior frontal gyrus of the dominant hemisphere which is the left hemisphere for most people.

- **The Parietal Lobe**

It is found between the central sulcus and the parieto-occipital sulcus, it controls perception and sensation. The primary somatosensory cortex is in the postcentral gyrus and is positioned immediately posterior to the central sulcus, in the parietal lobe. The primary somatosensory cortex controls the sense of touch, temperature, and pain of the contralateral body. Mirroring the primary motor cortex, the medial region senses the lower limb, the supero-lateral region senses the upper limb and hand, and the lateral region senses the face. Similar to the primary motor area, the hands, face, and lips take up the majority of the somatosensory area as shown by the homunculus model. Damage to the parietal lobe can present with a lack of these sensations as well as other symptoms depending on whether the dominant or nondominant hemisphere is more affected.

- **The Occipital Lobe**

It is found behind the parieto-occipital sulcus and is responsible for interpreting vision, distance, depth, color, and facial recognition. The occipital lobe receives its information from the contralateral vision field of both eyes (i.e., the left occipital lobe receives and interprets information from the right visual field from both the left and right eye).

- **The Temporal Lobe**

It is below the lateral fissure, that separates the parietal and temporal lobes, and further divides into a superior, middle, and inferior temporal gyrus. This lobe controls language comprehension, hearing, and memory. Wernicke's area is responsible for language comprehension, and it is not found in both hemispheres. Similar to Broca's area, Wernicke's area is in the superior temporal gyrus of the dominant hemisphere, usually the left hemisphere. Therefore, the location of Wernicke's area is most commonly in the left superior temporal gyrus. The primary auditory cortex is in the superior temporal gyrus and processes most auditory information from the contralateral ear and some from the ipsilateral ear. The temporal lobe communicates with the hippocampus and amygdala to form memories.

- **The Diencephalon**

It is found above the brain stem, enclosed in the cerebral hemispheres and composed of the thalamus, hypothalamus and epithalamus.

- **The Thalamus**

It encloses the third ventricle of the brain. It is a relay station for sensory impulses passing upward to the sensory cortex. As impulses surge through the thalamus, we could

recognize whether the sensation we are about to have is pleasant or not, while the neurons of the sensory cortex actually localize and interpret the sensation.

- **The Hypothalamus**

It constitutes the floor of the diencephalon. It is an important autonomic center because it plays a role in regulating body temperature, water balance and metabolism. The hypothalamus is also a center for many drives and emotions and is a part of the limbic system. It contains centers for thirst, appetite, sex, pain and pleasure and also regulates the pituitary gland, the endocrine gland responsible for regulating secretions of most hormones in the body. The hypothalamus also produces two hormones of its own. Mamillary bodies which are reflex centers for olfaction also originate from the floor of the hypothalamus.

- **The Epithalamus**

It forms the roof of the third ventricle, with two important parts, the pineal gland and the choroid plexus, which is a network of capillaries within each of the four ventricles that form and reabsorb cerebrospinal fluid.

- **The Cerebellum**

It projects dorsally from below the occipital lobe of the cerebrum. Like the cerebrum, it has two hemispheres, with an outer gray matter cortex and an inner white matter as well as a convoluted surface. It controls our balance and coordinates and smoothens our movements. Fibers from the equilibrium apparatus in the inner ear, the eye, the proprioceptors of skeletal muscles and other areas reach the cerebellum. The cerebellum continuously compares the brain's intentions with actual body performance by monitoring the body's position and tension in various body parts and sends messages to initiate the appropriate corrective measures.

- **The Brain Stem**

It is made of the mid-brain, pons and medulla oblongata. It is not only the pathway for ascending and descending tracts to and from the brain, it contains many small areas of gray matter that program rigid autonomic behaviors necessary for survival.

- **The Mid-Brain**

A relatively small part of the brainstem extending from the mamillary bodies to the pons inferiorly. The cerebral aqueduct passes through it, connecting the third ventricle of the diencephalon to the fourth ventricle below. Anteriorly there are cerebral peduncles that convey ascending and descending impulses. Posteriorly are the corpora quadrigemina, reflex centers involved with vision and hearing.

- **The Pons**

This is the rounded structure of the brain stem just below the midbrain. It mainly consists of fiber tracts, bundles of nerve fibers in the central nervous system and important nuclei involved in the control of breathing.

- **The Medulla Oblongata**

This is the most inferior part of the brain stem that merges into the spinal cord below. Just like the pons, it is an important fiber tract area. It is the area where pyramidal tract (motor fibers) cross over to the opposite side. It contains many nuclei that regulate vital visceral activities such as heart rate, blood pressure, breathing, swallowing, vomiting among others.

- **The Reticular Formation**

This is a diffuse mass of gray matter extending the entire length of the brain stem, containing neurons that regulate motor activities of visceral organs for example the smooth muscle of the digestive tract. A special group of reticular formation neuron called the reticular activating system (RAS) plays a role in consciousness and the awake/sleep cycle. The RAS filters the daily flood of impulses into the brain and spinal cord. Damage to this area could result in coma.

II.1.2.2. Vascularization of the Brain[29,30]

The development of the cerebral circulation occurs even before the heart starts beating, in two main stages: vasculogenesis (the de novo creation of blood vessels where previously none existed) and angiogenesis (the production of new capillaries from existing blood vessels, driven by hypoxia within the fetus). The circulatory system develops from the six pairs of branchial arch arteries. The third pair of branchial arch arteries and the distal segment of the paired dorsal aortae become the internal carotid arteries (ICAs). The anterior division of the ICA goes on to form the primitive olfactory artery, which becomes the middle (MCA) and anterior cerebral arteries (ACA), whilst a posterior division eventually becomes the posterior cerebral artery (PCA). Initially, the posterior circulation relies on anastomoses from the anterior circulation to the basilar artery before the development of the vertebral arteries and the loss of the anastomoses.

- **Arterial Circulation**

Blood supply to the brain is through the two ICAs and the vertebrobasilar system, which provide 70% and 30% of the flow, respectively. The vertebral arteries originate from the subclavian arteries and join to form the basilar artery. This basilar artery then divides again to

form the PCAs that anastomose with the ICAs to form a system at the base of the brain called the circle of Willis. The ICAs also continue on to form the MCAs and join anteriorly to form two ACAs. The anterior cerebral circulation, made up of the ACAs and MCAs, also includes the anterior choroidal artery, which can be significant in disease. It usually arises from proximal to the bifurcation of the ACAs and MCAs, although there are a number of variations. The ACAs supply most of the medial part of the cerebral hemispheres; the MCAs supply the lateral sides of the hemispheres, and the PCAs supply the occipital and inferior parts of the temporal lobes.

- **Anterior Cerebral Arteries**

The ACAs supply all of the medial surfaces of the frontal and parietal lobe, the majority of the corpus callosum and the fronto-basal cerebral cortex. These areas include the frontal, prefrontal, primary motor, primary sensory and supplemental motor cortices (which contain Broca's speech area). The main motor and sensory functions relate to the lower limbs and speech motor production. The ACA is split into five segments: A1-5.

- **Middle Cerebral Arteries**

The MCA is the largest of the intracerebral vessels, arising from the ICA and continuing into the lateral sulcus where it divides further and supplies the cerebral cortex of the lateral frontal, parietal and temporal lobes; part of the basal ganglia; and the internal capsule. The basal ganglia are involved in motor control, learning and executive function along with emotions. The motor and sensory areas supplied are mainly those of the face and upper limbs. The MCA is split into segments M1-4.

- **Posterior Cerebral Arteries**

The PCAs are the terminal branches of the basilar artery, arising from the basilar artery and projecting towards the occiput and over the tentorium cerebelli to the occipital lobe. They supply the occipital lobe, which includes the visual areas and the lower portion of the temporal lobe. It also supplies the thalamus, which relays sensory and motor signals, deep structures of the brain and part of the internal capsule, which contains the descending parts of the lateral and anterior corticospinal tracts. The PCA is split into four segments P1-4.

- **Venous Circulation**

The cerebral venous circulation has a wide variability between people and even between the two hemispheres compared with the arterial system. They differ from other veins in the body, as they do not follow the pathway of the associated cerebral arteries and do not have

valves. This makes the venous circulation bidirectional, which is essential in intracranial pressure (ICP) regulation in relation to posture and cerebral venous outflow. These qualities make the cerebral venous circulation unique and protect against several clinical conditions of the brain. Venous circulation of the cerebrum consists of deep and superficial cerebral veins, which drain into the dural venous sinuses located in between the periosteal and meningeal layers of the dura mater, and eventually drain into the internal jugular vein (IJV).

- **Deep Cerebral Veins**

The deep cerebral veins are closely associated with the thalamus originating at the foramen of Munro, and run posteriorly within the roof of the third ventricle. The two veins anastomose to form the great vein of Galen (GV). The basal vein of Rosenthal drains the midbrain structures and into the GV, which drains into the straight sinus.

- **Superficial Cerebral Veins**

These veins comprise the Superior cerebral veins and the Sylvian vein. The superior cerebral veins extend on the lateral surface of the brain superiorly to drain into the superior sagittal sinus, which is within the falx cerebri. The Sylvian vein lies in the Sylvian fissure and drains into three different sinuses. It drains in the superior sagittal sinus via the superior anastomotic vein of Trolard, transverse sinus via the inferior anastomotic vein of Labbe and anteriorly into the cavernous sinus. The cavernous sinus is located in the middle cranial fossa next to the sella turcica and pituitary gland, and is the only place in the body that an artery passes inside a venous structure. This sinus contains important structures, such as the ICA, carotid plexus and cranial nerves (oculomotor, trochlear, ophthalmic, maxillary and abducens nerves). The falx cerebri contains the superior sagittal, inferior sagittal and straight sinus. These anastomose at the confluence of sinuses located at the internal occipital protuberance. This becomes the transverse sinus, which emerges as the sigmoid sinus and drains into the IJV together with the cavernous sinus carrying deoxygenated blood back to the heart. The IJV is able to drain 100% of the cerebral venous outflow, but there is a second venous system; the vertebral venous plexus (VVP) which can drain up to 30% of the venous outflow from the brain. The majority of veins in the posterior fossa drain into the inferior petrosal sinus. This sinus is a connection between IJV and VVP.

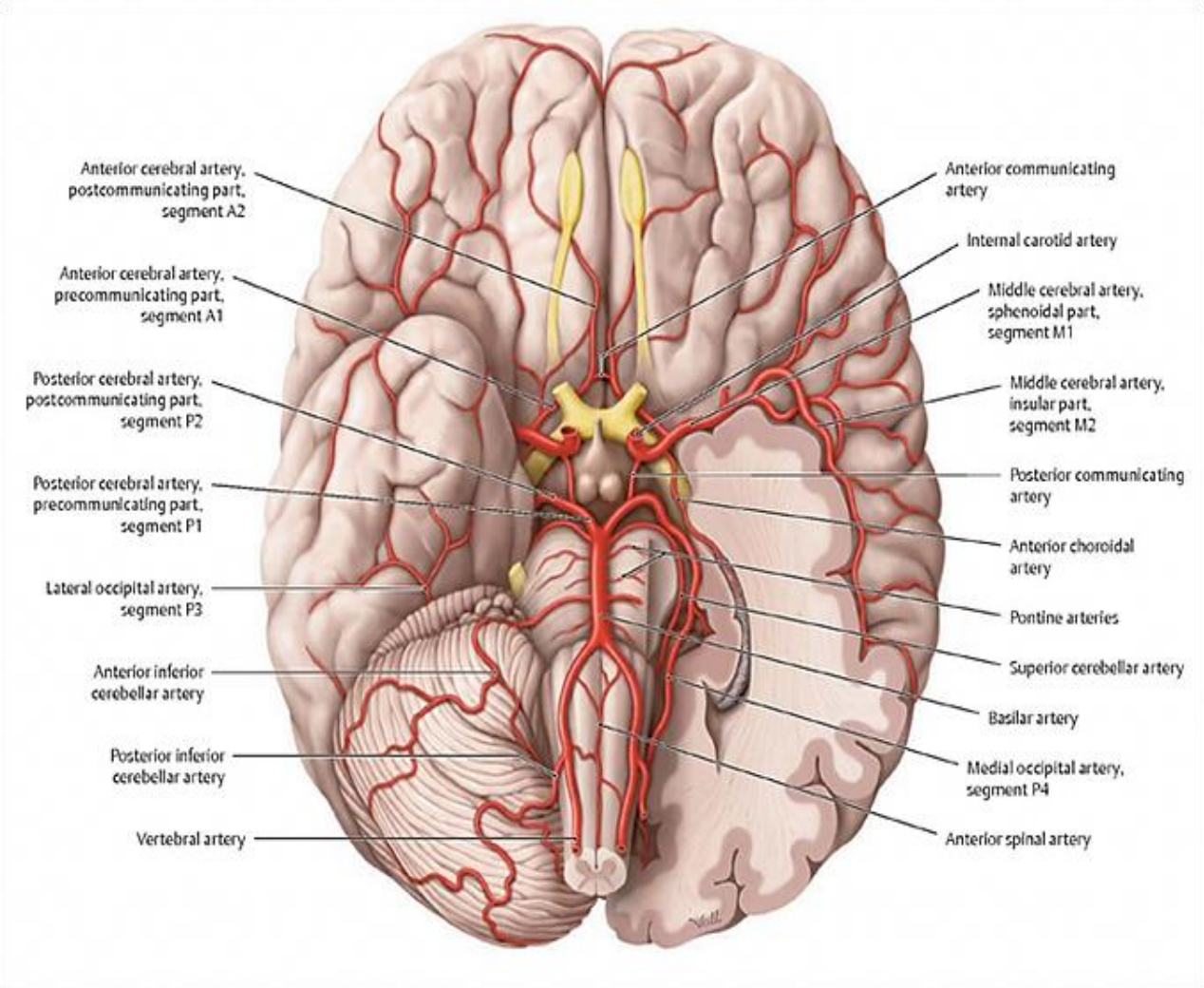

Figure 2: Brain Arterial Circulation - Atlas of Anatomy. Head and Neuroanatomy. Micheal Schuenke[31]

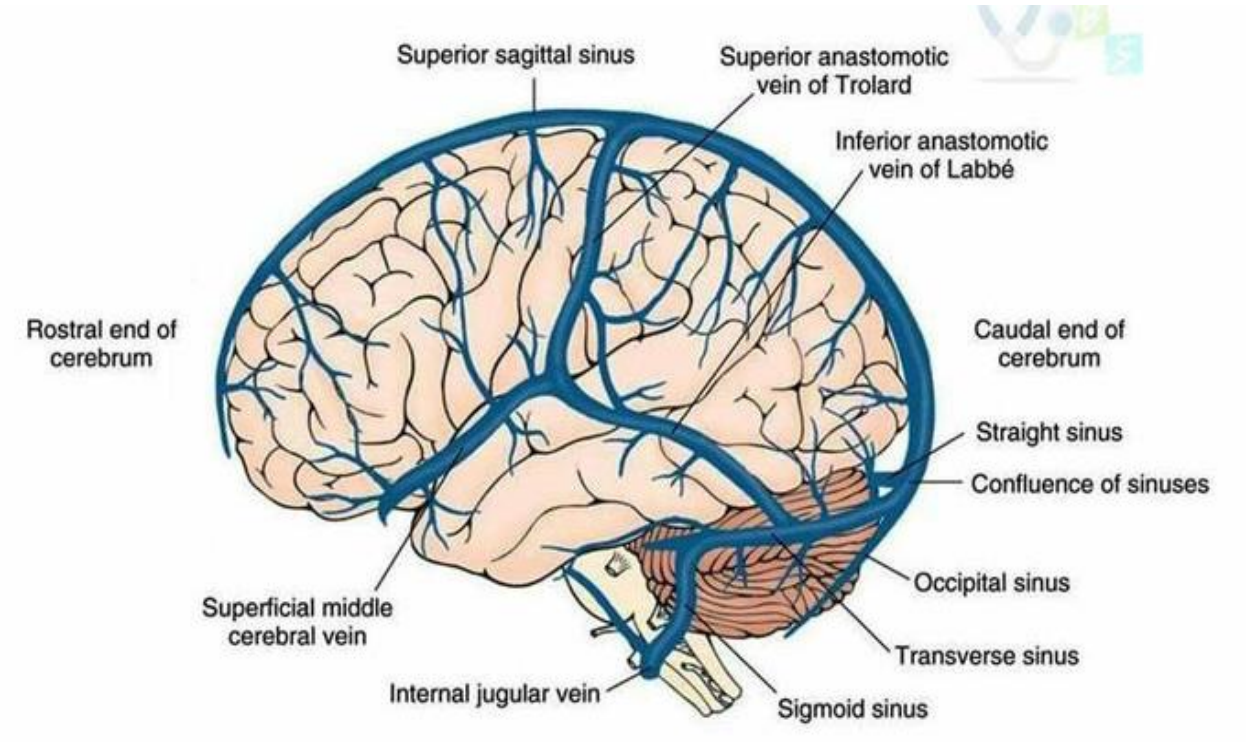

Figure 3: Veins of the Brain[32]

II.1.3. PATHOPHYSIOLOGY [1,4,8,33]

II.1.3.1. Ischemic Stroke

Acute ischemic stroke results from vascular occlusion secondary to thromboembolic disease. Ischemia causes cell hypoxia and depletion of cellular adenosine triphosphate (ATP). Without ATP there is no longer energy to maintain ionic gradients across the cell membrane and cell depolarization. Influx of sodium and calcium ions and passive inflow of water into the cell lead to cytotoxic edema. An acute vascular occlusion produces heterogenous regions of ischemia in the affected vascular territory and local blood flow is limited to any residual flow in the major arterial source and collateral supply if present. Affected regions with a cerebral blood flow of less than 10mL/100g of tissue/min are referred to as the core while those with a flow of less than 25mL/100g of tissue/min (zone of decreased or marginal flow) are known as the ischemic penumbra. Cerebral ischemia disrupts normal sodium-calcium exchange protein also found on the plasma membrane, the influx of calcium leads to the release of glutamate in large quantities, which activates N-methyl-D-aspartate (NMDA) and other excitatory receptors on other neurons which become depolarized causing further calcium influx, increasing the initial ischemic insult. The massive calcium influx also activates various degradative enzymes which lead to the destruction of the cell and essential neuronal structures. Free radicals, arachidonic acid and nitric oxide generated by this process also lead to further neuronal damage.

Ischemia also leads to breakdown of the blood brain barrier within 4 to 6 hours after infarction. Proteins and water flood the extracellular space, leading to vasogenic edema, greater swelling and mass effect that peak at day 3-5 and resolve over the next weeks. Cytokines and other inflammatory factors will be released within days following stroke. Infarction results in the death of supporting glial cells which will eventually undergo liquefaction necrosis and be removed by macrophages with a resultant loss of parenchymal volume.

Approximately 85% of strokes are ischemic, predominantly the result of cerebral small vessel disease (CSVD), cardioembolism and large artery disease (atherosclerosis).

- Cerebral Small Vessel Disease (CSVD);

Includes deep perforator arteriopathy (also termed arteriolosclerosis or hypertensive arteriopathy) and cerebral amyloid angiopathy (CAA). Deep perforator arteriopathy affects the structure and function of small vessels supplying the basal ganglia and brainstem; it causes approximately 25% of ischemic strokes. CSVD is diagnosed on the basis of radiological markers, including: recent small subcortical infarcts, white matter hyperintensities, lacunes, cerebral microbleeds, enlarged perivascular spaces and cerebral atrophy on MRI; or white matter hypodensities and lacunes on computed tomography (CT). Its prevalence increases with age with no differences between sexes, and can be higher in Asian populations. The most important risk factor for CSVD is hypertension, more rarely genetic disorders, radiation exposure and immune-mediated vasculitis can cause CSVD.

- Cardioembolic Stroke:

A further 25% of ischemic strokes are caused by cardioembolic disease (mainly AF), the risk increasing with age. In stroke patients, paroxysmal AF is more prevalent than persistent AF.

- Large Artery Disease:

Stenosis or occlusion of the large cerebral arteries (predominantly the extracranial carotid) is the cause of about 20% of ischemic strokes. Rupture of arteriosclerotic plaques leads to in situ thrombus formation and distal embolization. In addition, ruptured carotid plaques lead to widespread platelet activation, and recurrent events are very common, particularly in the first few weeks. Less commonly, stenosis of the vertebrobasilar or intracranial arteries causes ischemic strokes. Hemodynamic strokes can occur when systemic blood pressure drops in the context of arterial stenosis, leading to infarction of border zone territories. Extracranial dissections of cervico-cephalic arteries (sometimes traumatic) account for about 1 in 5 ischemic strokes in patients <50 years old.

- **Cryptogenic Stroke**:

In 20-30% of patients with ischemic stroke, no cause is found. These strokes may relate to undiagnosed cardioembolic disease, hypercoagulable states, paradoxical emboli via a patent foramen ovale, sub-stenotic atheromatous disease, non-atherosclerotic arteriopathies, occult recreational drug use or undiagnosed genetic conditions or risks.

II.1.3.2. Hemorrhagic Stroke

In intracerebral hemorrhage (ICH), bleeding occurs directly into the brain parenchyma. The usual mechanism is thought to be leakage from small intracerebral arteries, damaged by chronic hypertension. Other mechanisms include bleeding diatheses, iatrogenic anticoagulation, cerebral amyloidosis and cocaine abuse. The thalamus, putamen, cerebellum and brainstem are more prone to ICH. In addition to the area of the brain injured by the hemorrhage, the surrounding brain can be damaged by pressure produced by the mass effect of the hematoma. A general increase in intracranial pressure may occur.

Subarachnoid hemorrhage (SAH) has multifocal effects on the brain. It results in raised intracranial pressure and impaired cerebral autoregulation. These effects can occur in combination with acute vasoconstriction, microvascular platelet aggregation and loss of microvascular perfusion, resulting in profound reduction of blood flow and ischemia.

- **Intracerebral hemorrhage**:

Spontaneous (non-traumatic) ICH can be anatomically divided into deep and lobar. Deep hemorrhages account for approximately two-thirds of ICH cases, and occur in the basal ganglia and internal capsule (35-70%) or brainstem (5-10%). About 5-10% of ICHs are in the cerebellum. The remainder are lobar hemorrhages located in cortico-subcortical areas in the cerebral lobes, often near or reaching the cerebral convexities. Hypertensive (deep perforator) arteriopathy (CSVD) is the most important cause of deep ICH, although it also contributes to lobar ICH. Detection of the neuro-imaging markers of CSVD mentioned above (including cerebral microbleeds) can help increase confidence that an ICH is due to CSVD. CAA, a CSVD characterized by the presence of amyloid-b protein within the cortical and leptomeningeal blood vessel walls is an important cause of lobar (but not deep) ICH in older people. CAA is also associated with cognitive impairment, transient focal neurological episodes (usually recurrent stereotyped attacks of spreading paresthesias affecting the arm and face, often related to small convexity subarachnoid hemorrhages). CAA can be diagnosed by brain imaging showing hemorrhage restricted to the lobar brain regions. After CSVD (which causes about 80% of all ICH), the next most common cause of ICH is macrovascular abnormalities (arteriovenous malformation, dural arteriovenous fistula); these are more common in (but are not limited to)

younger people and can only be identified by imaging of the brain vessels (e.g. using CT angiography, MR angiography etc.).

Rarer causes of ICH include hemorrhagic transformation of ischaemic infarcts, venous sinus thrombosis, brain tumours, reversible cerebral vasoconstriction syndrome and endocarditis. Recreational drug use (especially cocaine) increasingly contributes to ICH in younger people.

II.1.4. DIAGNOSIS [1,34–37]

II.1.4.1. Signs and Symptoms

Stroke should be considered in any patient presenting with an acute neurologic deficit (focal or global) or altered level of consciousness. No historical features clearly distinguish ischemic from hemorrhagic stroke, although nausea, vomiting, headache and a sudden change in the patient's level of consciousness are more common in hemorrhagic strokes.

Common signs and symptoms of stroke include the abrupt onset of any of the following;

- Hemiparesis/ monoparesis/ quadriparesis
- Hemisensory deficits
- Monocular or binocular visual loss, diplopia, visual field deficits
- Dysarthria
- Facial droop
- Aphasia
- Ataxia
- Vertigo
- Decrease in the level of consciousness

These symptoms most likely occur in combination. It is important to establish the time at which the patient was last seen without stroke symptoms or last seen to be normal, as this is critical when fibrinolytic therapy is an option.

A focused medical history for patients with stroke is mainly to identify risk factors for atherosclerotic and cardiac disease as mentioned above. In younger patients we also need to elicit a history of recent trauma, coagulopathies, illicit drug use, migraines and oral contraceptive use.

II.1.4.2. Physical Examination

The goals of physical examination are to;

- Detect extracranial causes of stroke symptoms
- Distinguish stroke from stroke mimics

- Determine and document for future comparison the degree of neurological deficit
- Localize the lesion
- Identify comorbidities
- Identify conditions that may influence treatment decisions for example recent surgery or trauma, active bleeding, active infection etc.

The physical examination must encompass all of the major organ systems, starting with airway, breathing, circulation (ABCs) and vital signs. Patients with a decreased level of consciousness should be assessed to ensure their airway is patent. Patients with stroke, especially hemorrhagic stroke, can suffer quick deterioration and so must be reassessed regularly. Vital signs can point to impending clinical deterioration and may assist in narrowing the differential diagnosis.

The physical examination always includes a careful head and neck examination for signs of trauma (contusions, lacerations, deformities), meningeal irritation. Auscultation of the neck may elicit a bruit, suggesting carotid disease as the cause of the stroke. Cardiac arrythmias such as atrial fibrillation are a common finding among stroke patients. Acute cardiac conditions such as myocardial infarction and acute heart failure could occur concurrently with stroke, auscultation is thus recommended in search of murmurs and gallops. Unequal pulses or blood pressures in the extremities may reflect the presence of aortic dissections, a potential cause.

The neurological examination is performed in order to confirm the presence of a stroke syndrome, rule out a stroke mimic, establish a neurologic baseline should the patient's condition improve or deteriorate, establishing severity to assist in prognosis and therapeutic selection. The components of the neurological examination include

- Mental status and level of consciousness
- Superior functions; language (expressive and receptive capabilities)
- Cranial nerves
- Meningeal signs
- Motor function
- Sensory function
- Gait

The National Institute of Health Stroke Scale (NIHSS)[38], a 42-point scale, enables the health care provider to rapidly determine the severity and possible location of the stroke. NIHSS scores are strongly associated with outcome and can help identify those patients who are at higher risk of developing complications from the stroke itself and those to benefit from reperfusion therapies. It focuses on 6 major areas of the neurologic examination; level of consciousness, visual function, motor function, sensation and neglect, cerebellar function,

language. It is worth noting that the presenting signs and symptoms in stroke vary according to the arterial territory affected.

- **Anterior Cerebral Artery Stroke**

ACA occlusions primarily affect frontal lobe function. Findings in ACA stroke may include

- Disinhibition and speech perseveration
- Primitive reflexes e.g grasping, sucking reflexes
- Altered mental status
- Impaired judgement
- Contralateral weakness (greater in legs than arms)
- Contralateral cortical sensory deficits
- Gait apraxia
- Urinary incontinence

- **Middle Cerebral Artery Stroke**

MCA occlusions commonly produce the following

- Contralateral hemiparesis
- Contralateral hypesthesia
- Ipsilateral hemianopsia
- Gaze preference towards the side of the lesion
- Agnosia
- Receptive or expressive aphasia depending on whether the lesion occurs in the dominant hemisphere
- Neglect, inattention and extinction of double simultaneous stimulation with some non-dominant hemisphere lesions.

  The MCA supplies the upper extremity motor strip, consequently, weakness of the arm and face is usually worse than that of the lower limb.

- **Posterior Cerebral Artery Stroke**

PCA occlusions affect vision and thought and can manifest as follows

- Contralateral homonymous hemianopsia
- Cortical blindness
- Visual agnosia
- Altered mental status
- Impaired memory

- **Vertebrobasilar artery** occlusions are difficult to localize because they cause a wide variety of cranial nerve, cerebellar and brainstem deficits. They include

- Vertigo
- Nystagmus
- Diplopia
- Visual field deficits
- Dysphagia
- Dysarthria
- Facial hypesthesia
- Syncope
- Ataxia

Anterior circulation stroke generally produces only unilateral findings while posterior circulation stroke could exhibit crossed findings, that is ipsilateral cranial nerve deficits and contralateral motor deficits.

Lacunar strokes result from the occlusion of small perforating arteries of the deep subcortical areas of the brain. The most common lacunar syndromes include pure motor, pure sensory and ataxic hemiparetic strokes. Due to their small size and well defined location, lacunar infarcts do not lead to impairments in cognition, memory, speech or level of consciousness.

II.1.4.3. Work-Up [35,37]

Emergent brain imaging is required for excluding stroke mimics and potentially confirming the diagnosis of stroke. The current standard is **non contrast computed tomography (CT)** of the head because it is fast and widely available[37]. When interpreted by an expert, head CT can rule in a diagnosis of hemorrhagic stroke (intracerebral or subarachnoid hemorrhage) with over 95% accuracy. Head CT can also rule in a diagnosis of major stroke in about two-thirds of cases in which ischemic changes are evident, but it is highly insensitive to the diagnosis of minor stroke and TIA because small-volume ischemic change is beyond the resolution of CT; therefore, a “normal” scan in the scenario of minor stroke neither confirms nor excludes ischemia.

Magnetic resonance imaging (MRI) has greater spatial resolution to detect brain ischemia in minor ischemic stroke and is the modality of choice to make an inclusive imaging diagnosis of minor stroke in cases where the deficits are very mild. For all presentations of acute stroke syndrome, **CT angiography** immediately following non-contrast head CT is recommended for identification of the occluded intracranial vessel and evaluation of the

extracranial carotid, extracranial vertebral, aortic arch and proximal great vessels, if not immediately then over the next several days. In cases of hemorrhagic stroke, intracranial CT angiography will identify intracranial aneurysm as the cause of subarachnoid hemorrhage or show the source of bleeding in intracerebral hemorrhage as a "spot" sign. Although MRI has greater sensitivity for the small-volume ischemia observed in transient ischemic attack or minor stroke, it is used only in situations where there is no time pressure to offer treatment, typically as follow-up imaging[37].

Carotid duplex scanning is also useful not only to detect the cause of stroke but also to stratify patients for either medical management or carotid intervention in case of stenoses.

Digital subtraction angiography is considered the definitive method for demonstrating vascular lesions, including occlusions, stenoses, dissections and aneurysms.

Additional laboratory tests are tailored according to the individual patient and may include the following;

- Complete blood count (for baseline studies and may reveal a cause of stroke e.g. polycythemia, thrombocytosis, thrombocytopenia, leukemia, evidence of concurrent illness, issues that may affect reperfusion strategies)
- Coagulation studies (may reveal a coagulopathy, useful for monitoring use of anticoagulants)
- Echocardiography (suspicion of a cardioembolic origin)
- Cardiac biomarkers (association with coronary artery disease)
- Fasting lipid profile (investigating dyslipidemia)
- Blood glucose (rule out hypoglycemia which can mimic stroke, investigate diabetes)
- Toxicology screening (investigating intoxication with drugs that mimic stroke syndrome)
- Urine pregnancy test (women of childbearing age)

II.1.5. MANAGEMENT [4,36]

II.1.5.1. Ischemic Stroke

The central goal of therapy in acute ischemic stroke is to preserve tissue in the ischemic penumbra. Guidelines for early management of stroke are available from the AHA/ASA. Patients with acute ischemic strokes are usually hospitalized.

Supportive measures needed during initial evaluation and stabilization include;

- **Airway support** and ventilatory assistance if decreased consciousness or airway compromise

- **Oxygen supplementation**; used in case of documented oxygen requirement (oxygen saturation <95%)
- Correction of hyperthermia by antipyretics and/or identifying and treating the cause
- Hypoglycemia must be identified and treated early in the evaluation. Hyperglycemia should be corrected as well while monitoring.
- **Blood pressure control**; according to the 2013 ASA guidelines, threshold for antihypertensive treatment in ischemic stroke is a SBP > 220mmHg and DBP > 120mmHg. A reasonable goal is to lower blood pressure by 15% within the first 24hours. Care must be taken to not lower blood pressure too quickly or aggressively, since this can worsen perfusion in the penumbra.

  For patients who are eligible for acute reperfusion therapy but have a BP>185/110mmHg, it can be treated with either labetalol 10-20mg IV bolus over 1 to 2 minutes which may be repeated once, OR Nicardipine 5mg/hour IV infusion initially, the dose can be increased by 2.5mg/hour every 5 to 15 minutes to a maximum of 15mg/hour
- **Cerebral edema control**; proclive position, hyperventilation, hyperosmolar therapy and rarely barbiturate coma may be used as in patients with raised intracranial pressure secondary to head trauma. Hemicraniectomy has been shown to decrease mortality and disability among patients with life threatening edema secondary to large hemispheric infarctions.
- **Seizure control**; seizures occur in 2-23% of patients within the first days after stroke. Primary prophylaxis is not recommended but secondary prevention with standard antiepileptic therapy is recommended.

  Treatment options for ischemic stroke comprise;
- **Fibrinolytic therapy**; recombinant tissue-type plasminogen activator (rt-PA) or alteplase, administered within 4.5hours of stroke onset, restores cerebral blood flow in some patients with acute ischemic stroke and may lead to improvement or resolution of neurologic deficits. Unfortunately, it is susceptible to cause symptomatic intracranial hemorrhage, extracranial hemorrhage, angioedema or allergic reactions. An age greater than 80 years, use of oral anticoagulants regardless of the INR, Baseline NIHSS score >25 and a history of stroke and diabetes, are some conditions that make patients ineligible for such therapy.
- **Mechanical thrombectomy**; this is a procedure that involves intra-arterial removal of a thrombus or embolus by a stent retriever device directed angiographically. It is an

alternative for patients who can't undergo fibrinolysis. It is recommended in eligible patients within 6-16 hours after stroke

- **Thrombolysis-in-situ;** can be done for patients who are not eligible for treatment with IV recombinant tPA, if stroke symptoms have lasted less than 6 hours.
- **Anticoagulation therapy**; oral anticoagulation is the therapy of choice for primary and secondary stroke prevention in patients with AF and any known additional risks factors. Asymptomatic patients younger than 65 years with AF and no other risk factors are at low risk and should either be treated with aspirin or not treated at all.
- **Stroke prevention**; preventative measures may include the use of antihypertensives, antiplatelet agents, statins, anticoagulants and encouraging life-style interventions such as smoking cessation, diabetes control, low-fat low-salt low-sugar diet and regular exercise.

II.1.5.2. Hemorrhagic Stroke

The treatment and management of patients with acute ICH depends on the cause and severity of the bleeding. It begins with stabilization of vital signs.

- Intubate patients with poor airway protection and low level of consciousness
- Hyperventilate if ICP is elevated and initiate mannitol use for further control
- Glucose levels should be monitored, normoglycemia is recommended
- **Blood pressure control**; greatly elevated BP is thought to lead to rebleeding and hematoma expansion. Intensive BP reduction (target MAP<110mmHg) early lessens hematoma growth. Suggested agents for use in the acute phase include beta blockers, angiotensin-converting enzyme inhibitors (ACEIs), nicardipine and hydralazine can be used for more refractory hypertension.
- **Seizures**; occurs in about 4-28% of patients with ICH and are often non-convulsive. Seizure activity accompanied by altered mental state should be treated with anti-epileptic drugs
- **ICP Control**; elevate the head of the bed at 30, this improves jugular venous outflow and lowers ICP. More aggressive therapy such as osmotic therapy, barbiturate anesthesia and neuromuscular blockage generally require monitoring of BP and ICP to maintain adequate cerebral perfusion pressure >70mmHg.
- **Analgesia** and sedation should be provided as needed
- **Antacids** are used to prevent gastric ulcers associated with ICH
- **Hemostatic therapy**; with recombinant factor VIIa

- **Evacuation of hematoma** either by open craniotomy or endoscopy is life-saving especially if the hematoma is greater than 3cm in the cerebellum.

Rehabilitative interventions (physiotherapy, speech therapy) should be initiated as soon as the patient has been stabilized and should be continued even as outpatient rehabilitation. Contraindications to the commencement of rehabilitation include: early deterioration, immediate surgery, another serious medical illness or unstable coronary condition, SBP < 110 mm Hg or > 220 mm Hg, Oxygen saturation <92% with supplementation, resting heart rate < 40 beats per min or >110 beats per min, temperature > 38·5°C and more than 30mmHg drop in BP on achievement of an upright position [39].

II.2. POST-STROKE PSYCHIATRIC DISORDERS

Stroke survivors are susceptible to many complications. These individuals commonly have comorbidities such as hypertension, diabetes, heart disease, or other ailments that increase the risks of systemic medical complications during stroke recovery. However, several complications can arise as a direct consequence of the brain injury itself, from the ensuing disabilities or immobility, or from stroke-related treatments. These events have a substantial effect on the final outcome of patients with stroke and often impede recovery. Cardiac complications, pneumonia, venous thromboembolism, fever, pain, dysphagia, incontinence, and neuropsychiatric disorders are particularly common after a stroke. Stroke survivors are often affected by psychological distress and neuropsychiatric disturbances. About one-third of stroke survivors experience depression, anxiety or apathy, which are the most common neuropsychiatric sequelae of stroke. Neuropsychiatric sequelae are disabling, and can have a negative influence on recovery, reduce quality of life and lead to exhaustion of the caregiver.

This review focuses on the most common non-cognitive neuropsychiatric consequences of stroke namely depression, anxiety, apathy, fatigue, psychosis and mania. For each consequence, we discuss definition and identification, prevalence, associations and treatment.

II.2.1. Post-Stroke Depression

II.2.1.1. Definition

The US Diagnostic and Statistical Manual of Mental Disorders-5, DSM-5 requires ‘depressed mood’ or ‘anhedonia/loss of interest or pleasure’ for at least two weeks, plus at least four other symptoms which are persistent and interfere with daily life; significant weight loss/gain; insomnia or hypersomnia; psychomotor agitation or retardation; fatigue or loss of

energy; worthlessness or inappropriate guilt; diminished concentration or indecisiveness[13,40].

II.2.1.2. Epidemiology

Depression is the most frequent post-stroke psychiatric disorder, with an estimated prevalence range from 18% to 33%[8]. In Africa, PSD prevalence ranges from 22.9 to 53.6%, pooled prevalence estimate in SSA was 31%[17] while Angwafor et al reported the prevalence of PSD in Cameroon to be 45.6% [19].

II.2.1.3. Risk Factors [8,11,40]

- **Genetic factors**; Common genetic variations might confer vulnerability or resilience to develop psychiatric illness when an individual faces an unusual stressful challenge. The 5-HTTLPR and the STin2 VNTR, polymorphisms of the serotonin transporter gene (SERT) have been associated with PSD in stroke survivors. Also, higher brain-derived neurotrophic factor (BDNF) gene methylation status was associated with incident PSD and more severe symptoms at 12 months follow-up.

- **Female gender**;

- **Medical and psychiatric history**; patients with PSD might be more likely to have a history of diabetes mellitus. A personal history of depression or anxiety or both was also consistently identified as a risk factor for PSD. A family history of depression was also associated with PSD.

- **Stroke characteristics and lesion location**; two studies led by Robinson reported that acute stroke patients with left frontal or left basal ganglia lesions had a significantly higher frequency of major or minor depression than patients with other lesion locations. A significant association has been shown between stroke severity and PSD.

- **Functional and cognitive impairment**

- **Lack of social support**

II.2.1.4. Pathophysiology [40]

A few studies have been done to investigate the mechanisms of PSD. It has been hypothesized that ischemic lesions (single and/or multiple) of the neural circuits that connect the prefrontal cortex, basal ganglia, thalamus, and amygdala (independently of their lateralization) may disrupt mood regulation and executive function leading to such a clinical presentation. Furthermore, there appears to be a threshold by which the confluence of multiple etiological factors or further damage to specific white matter tracts, such as the cingulate

bundle, the uncinate fasciculus, and superior longitudinal fasciculus, triggers the onset of clinical depression. Also decreased CSF levels of serotonin or norepinephrine metabolites and serum BDNF were significantly associated with severity of PSD. Thus, acute ischemia can unveil the presence of vascular depression, or a strategically located stroke might produce depression independently of the presence of widespread cerebrovascular pathology. Additionally, HPA axis deregulation and increased levels of proinflammatory cytokines may inhibit neurogenesis in the hippocampus and decrease the neuroplasticity of the prefrontal cortex contributing to the onset and perpetuation of PSD. Thus, although there are numerous possible physiological mechanisms related to PSD, many investigators have concluded that this complex disorder, like most of the major psychiatric disorders not associated with stroke, may best be described as a bio-psycho-social disorder.

II.2.1.5. Diagnosis

There are many rating scales to measure depression which include Hamilton depression rating scale, Geriatric depression scale, Beck depression inventory scale, Montgomery–Asberg depression rating scale, Patient Health Questionnaire among others. The Patient Health Questionnaire is considered and used as an updated tool for diagnosing post-stroke depression, with a specificity of 90%[41].

II.2.1.6. Treatment

Pharmacological therapies such as selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants are effective in the treatment of post-stroke depression, however, associated with a greater risk of stroke (48%)[11]. Patients treated with Nortriptyline (50-100mg/ day) and Citalopram (10-20mg/day) showed significant improvement within 6 weeks of treatment. The AHA recommends antidepressants for PSD until at least 6months after recovery[40]. Though pharmacological therapy has been proven very efficient, non-pharmacological treatment, through therapies have been beneficial as well[11]. These include;

- Psychological therapy that helps in the prevention of the occurrence of post-stroke depression mostly in 5-HTTLPRR S allele carriers
- Cognitive therapy corrects the negative therapy and increases the daily activities, thereby facilitating problem-solving skills and cognitive restructuring skills to manage the ongoing tensions.
- Problem-solving therapy reduces the mental disability and reduces the mortality.
- Motivational therapy motivates medication adherence in patients with post-stroke depression.

- Verbal therapy includes literature therapy (expressive therapy) and story therapy (narrating a person's story)
- Music therapy helps in improving emotional aspects of life. Listening to music generates motions and emotions and thereby enhances the cognitive and emotional functions in patients. 64.3% of stroke patients in music therapy showed mood improvement. It involves exercises using musical instruments which accelerate the motor function by producing a change in cortical plasticity and rapid plastic adaptation.
- Art therapy forms a patient rapport with the therapist by free self-expression through painting or drawing.
- Electroconvulsive therapy involves the passage of electric current through the brain with the help of anesthetic and muscle relaxant that helps in the growth of nerve cells.
- Transcranial stimulation targets the distributed brain network responsible for depression and delivers magnetic stimulation in short time. Transcranial direct current stimulation along with cognitive control therapy as a combination is used to treat PSD, with a response rate of 25% and is beneficial in chronic stroke patients[11].

II.2.2. Post-Stroke Anxiety

II.2.2.1. Definition

The DSM-5 describes several anxiety disorders. Anxiety symptoms that are 'out of proportion to the actual threat or danger the situation poses', must be present for six months to meet diagnostic criteria for a Generalized Anxiety Disorder, plus at least three of the following symptoms: feeling wound-up, tense or restless; fatigued; difficulty concentrating; irritability; significant muscle tension; difficulty sleeping[13]. PSAn typically manifests as excessive concern on personal prognosis including recurrence of stroke, returning to work, falling, and ability to maintain independence, which could be considered normal responses to stroke, and are considerable challenges for the accurate diagnosis of PSAn[8].

II.2.2.2. Epidemiology

PSAn occurs in 18% to 34% of survivors during the first year after stroke, and does not change significantly up to 5 years after stroke[8]. It is more common in the young and in women[11]. A meta-analysis done by Ojagbemi et al in SSA in 2017, reported a prevalence of PSAn of 19.7%[18].

II.2.2.3. Risk Factors

The main predictive factors associated with post-stroke anxiety include post-stroke depression, pre-stroke anxiety or depression, coping, confidence, tiredness, and lack of sleep leading to decreased quality of life and physical disability[11]. Another study mentioned stroke severity, early anxiety and dementia or cognitive impairment following stroke as important risk factors for onset of anxiety following stroke.

II.2.2.4. Pathophysiology

Anxiety related neural circuits cover many brain structures including subcortical white matter and the limbic system. The lesion-location hypothesis for PSAn might be considered, as the infarction may damage anxiety associated brain structures leading to dysregulation and onset of anxiety following stroke. A clear understanding of the pathophysiological mechanisms underlying PSAn is still in process.

II.2.2.5. Diagnosis

A lot of scales have been developed for assessment of anxiety such as HADS-A, Hamilton Anxiety Scale-A, BAI, Irritability Depression and Anxiety Scale, among others. The HADS was developed for the assessment of emotional disturbances in non-psychiatric patients within a hospital setting and is the main anxiety-specific case-finding tool recognized for use in stroke[13]. It includes a total of 14 items, each with a score of between 0 and 3. One half of the items are related to anxiety while the other half is specific for depression. The HADS has been previously validated in Nigeria, where the HADS-A was found to have a sensitivity ranging 85.0–92.9% and a specificity of 86.5–90.0%.

II.2.2.6. Treatment

Serotonin–norepinephrine reuptake inhibitors (SNRIs) are superior over SSRIs because of their dual action (inhibiting) on serotonergic and adrenergic receptors. Treatment with duloxetine (SNRI) is more effective than citalopram and sertraline for anxiety symptoms such as decreased appetite, psychomotor retardation, and physical impairment. Zoplicone and zaleplon are the hypnotics used for treating insomnia in GAD and post-traumatic stress disorder[8,11].

Non-pharmacological treatment e.g. slow-stroke back massage can reduce pain levels, anxiety, and other physiological measures and is an effective treatment for older patients with stroke-related pain and anxiety. Autogenic relaxation training is an advantageous and acceptable treatment for reducing anxiety in stroke survivors[11].

II.2.3. Post-Stroke Apathy

II.2.3.1. Definition

Apathy is a behavioral syndrome characterized by a loss of motivation that occurs in one-third of patients after stroke. It can be defined as a quantitative reduction in goal-directed behaviors (GDB) occurring in the cognitive/behavioral, emotional, or social domains of an individual's life. Post-stroke patients with apathy suffer from greater functional impairment and demonstrate slower recovery times to normal functioning[8].

II.2.3.2. Epidemiology

Apathy could be grouped into classical, depressive, and combined. Classic or combined apathy is due to medium lesions and is seen mostly in men, whereas depressive apathy is seen more in women and is associated with small lesions. Approximately 1 of 3 patients will develop apathy but the incidence is low in patients who have no previous history of cerebrovascular diseases, and there is no difference between ischemic and hemorrhagic stroke concerning the incidence of apathy symptoms[11].

Post-stroke apathy (PSA) is often misdiagnosed as depression partially due to overlapping symptoms with depression, and the fact that both manifest as an inactive mood. PSA and depression differ in prevalence, pathogenesis, comorbidities, and outcomes. Emotional distress is the main distinguishing factor between apathy and depression: patients with PSD usually exhibit pessimism and hopeless emotion, while PSA patients often present with indifference and passive engagement with daily behaviors[8].

II.2.3.3. Risk Factors

Risk factors for post-stroke apathy mainly include older age, cognitive impairment, and functional decline.

II.2.3.4. Pathophysiology

Apathy is traditionally described as the result of damage to specific brain structures related to GDB such as the basal ganglia and prefrontal cortex. However, a clear relationship between lesion location and apathy has not been established, suggesting that the link between structural damage and functional deficit may be more complex. Secondary neurobiological changes can propagate through structural and functional connections in the brain and lead to apathy symptoms if GDB related networks are affected. This network-based conceptualization

of apathy explains a wide range of findings in patients with post-stroke apathy, although further testing of it is necessary. As a corollary, apathy may improve in response to restorative mechanisms such as adaptive plasticity and functional remapping[34].

II.2.3.5. Diagnosis

Apathy can be suspected in routine clinical practice during the history taking and examination from an observed loss of motivation. Informant histories may also reveal symptoms of apathy, such as loss of interest in previous activities and hobbies or doing little when left alone, which can be valuable as patients may underplay symptoms. Apathy assessments can be supplemented with semi-structured interviews or questionnaires like the Apathy Evaluation Scale, Lille Apathy Rating Scale, Dimensional Apathy Scale, Starkstein Apathy Scale among others.

II.2.3.6. Treatment

- Antidepressants such as selective serotonin reuptake inhibitors (SSRIs) may be prescribed for apathy in clinical practice due to shared symptomatology with depression. A daily dose of nefiracetam-900 mg given for 4 weeks or cilostazol for 6 months helps in reducing symptoms of apathy in post-stroke depression[11].

- Neuropsychological advice can be provided to patients with apathy in the context of more general rehabilitation procedures and can be delivered individually or in formal group settings. Patients can be engaged in goal setting with an emphasis on planning future goals and evaluating success to help re-establish GDB. Complimentary approaches include problem-solving, wherein a patient selects an activity and makes a plan to achieve it while self-monitoring the process and outcome. Behavioral activation can be combined with cognitive-behavioral therapy which explores psychological issues that may prevent GDB engagement. Approaches should foster a sense of the self, belonging, and respect and be tailored toward settings, where patients can derive enjoyment from exercises and activities such as planned outings consistent with personal backgrounds[34].

II.2.4. Post-Stroke Fatigue [42]

II.2.4.1. Definition

A proposed definition for PSF is a self-reported, perceived lack of physical or mental energy to perform daily activities. PSF can be of two types; exertion fatigue, which manifests in acute post-stroke period is experienced typically after intense physical exertion or use of mental effort, with a rapid onset, short duration, and short recovery. The other type of poststroke

fatigue is chronic fatigue, manifesting in the late phase after stroke, and characterized by mental and psychological symptoms, associated with a lack of interest or poor motivation.

II.2.4.2. Epidemiology

A systematic review and meta-analysis found marked variabilities in estimates of poststroke-fatigue prevalence, ranging from 25% to 85%. This wide range across studies may be due to the multifactorial features underlying fatigue, the varying times of assessment, and the methodological differences among studies including assessment techniques and diagnostic cut offs-used.

II.2.4.3. Risk Factors

PSF is affected by multiple risk factors, including cognitive impairment, lower level of social support, passive coping patterns, sleep problems such as insomnia, frequent wakening, and apnea. PSF has also been found to be associated with posterior circulation stroke as well as internal capsule lesions. A strong relationship has also been reported between PSF, PSD and poststroke pain, where pain promotes fatigue while fatigue and depression promote each other. Other associated factors include young age and female sex.

II.2.4.4. Pathophysiology

In a study with transcranial magnetic stimulation, it was reported that the overall excitability of cortical motor pathways seem to be diminished in patients with poststroke fatigue. Moreover, neural excitability was reported to be partly dependent on spontaneous neuronal firing rates, and reduced neuronal firing rates that were seen immediately after stroke. This suggests that poststroke fatigue was most likely triggered by reduced homeostatic rebalancing of spontaneous neuronal firing rates in the period after stroke, therein, leading to the lowered corticomotor excitability.

II.2.4.5. Diagnosis

Clinical characteristics of PSF include self-control and emotional instabilities, reduced mental capacity, and a reduction in energy needed for daily activities.

A recent study has concluded that the Fatigue Severity Scale can be reliably used for diagnosis of poststroke fatigue

II.2.4.6. Treatment

The strategy used to treat PSF comprises interventions on various predisposing, triggering and perpetrating factors involved with PSF. Modafinil, a neuroendocrine regulator and wakefulness promoting agent that stimulates monoaminergic pathways with neuroprotective properties, has been reported to significantly reduce poststroke fatigue and improve quality of life, without significant adverse events.

Regarding interventions on affective disorders, antidepressants that are commonly used to treat poststroke depression, such as fluoxetine, citalopram, could be used in case of concomitant emotional affect.

Graded physical activity programs have been suggested since exercise improves both physical and functional outcomes and therein reduces fatigue[42].

II.2.5. Post-Stroke Psychosis

II.2.5.1. Definition

Post-stroke psychosis is a neuropsychiatric disorder characterized by hallucination, psychomotor agitation, thought disorder, delusional beliefs, and euphoria.

II.2.5.2. Epidemiology

Poststroke psychosis (PP) was once considered a rare phenomenon; however, current prevalence estimates indicate around 4.87% of post-acute stroke patients exhibit either delusions or hallucinations with poor cognitive insight. The average age of presentation for PP is 66.6 years and it is more frequent in men. Psychosis may develop acutely in a matter of days following stroke; nonetheless, an average time to onset of 6 months after stroke has been reported.

II.2.5.3. Risk Factors

In a review done between 1975 and 2016, the main risk factors identified for PP included an older age, the male sex, stroke lesions in the right cerebral hemisphere, especially the frontal, temporal, parietal and caudate nucleus[43].

II.2.5.4. Pathophysiology

The pathophysiology involved in post-stroke psychosis is a dysfunction of subcortical basal ganglia limbic system interaction, involving abnormal dopaminergic neurotransmitters. The content of delusions can reflect the underlying function of the cortex

II.2.5.5. Diagnosis

The diagnosis of post-stroke psychosis is made upon the presence of delusions and/or hallucinations, occurring in full consciousness and not explained by any other non-specific cause for example delirium[43].

Delusions and hallucinations are the most prominent symptoms of post-stroke psychosis. Delusions are erroneous beliefs that usually involve the misinterpretation of perceptions and experiences while hallucinations are abnormal perceptions that are not experienced by others. The most common delusions are persecutory followed by jealousy delusions (Othello syndrome) while the most common hallucinations are auditory. According to Stangeland, the average time of onset of PP was 2 days following stroke, the average age of onset was 66.6years, the average duration of symptoms was 6.1months with men being most represented.

II.2.5.6. Treatment

For post-stroke psychosis treatment, antipsychotic drugs, namely haloperidol, risperidone, olanzapine, and quetiapine are empirically used, even though they might increase the risk of a new cerebrovascular event[14]. Psychological interventions, psychosocial measures, and neuromodulation therapies may also be useful in the management of PP. It is worth noting that the most common treatment outcome reported in the literature was complete resolution of poststroke psychosis, with the average time interval to complete resolution being about 3.5 months[43]

II.2.6. Post-Stroke Mania

II.2.6.1. Definition

Mania represents elevated or irritable mood along with increased disturbance in language, thoughts, and contents ordinarily caused by predisposing genetic factor, brain atrophy, and disruption in the right corticolimbic pathways. Secondary mania symbolizes elevated or irritable mood caused by neurological, metabolic, or toxic disorders. PSM therefore is a form of secondary mania manifesting after a stroke event (neurological disorder).

II.2.6.2. Epidemiology

The prevalence PSM is considered to be low (<2%) despite the fact that there is little or no reliable epidemiological data on the subject[13].

II.2.6.3. Risk Factors

A review of 49 individual cases described the typical patient as being male, having vascular risk factors and a lesion in the right hemisphere[13]

II.2.6.4. Pathophysiology

Lesions responsible for poststroke mania could be found in the thalamus, caudate nucleus, and temporal, parietal, and frontal lobes. Mania seems to be more associated with right-sided lesions[11,13] though a few reports show left-sided lesions. Onset of PSM could be from the first days following stroke until 2 years after the stroke event.

II.2.6.5. Diagnosis

The symptoms of secondary mania are hyperactivity, pressured speech and fight of ideas, reduced sleep, and lack of judging skills. According to the DSM-5, post-stroke mania can be diagnosed if the following criteria are met;

- Prominent and persistent disturbance in mood
- Evidence from history, physical examination or laboratory finding that the disturbance is directly linked to stroke
- The disturbance is not caused by delirium or another mental disorder
- It causes significant impairment in social, occupational and other important areas of life.

The Mood Disorder Questionnaire is commonly used to ascertain diagnosis[44].

II.2.6.6. Treatment

The pharmacological treatment suggests the use of mood stabilizers and typical and atypical antipsychotics. Lithium has favorable/ positive results, but its use is questionable in the case of cerebral lesions. Atypical antipsychotics are ordinarily preferred because of minor side effects. Benzodiazepines may be used as supporting therapy for hyperactivity and lack of sleep. Anticonvulsants such as divalproex sodium are effective and well-tolerated choice of treatment in the elderly and in secondary mania [11].

II.3. CURRENT KNOWLEDGE ON PSYCHIATRIC DISORDERS AND STROKE

A. IN THE WORLD

Stroke is a leading cause of death and a considerable economic burden owing to the cost of long-term rehabilitation. Most stroke survivors will suffer physical disability and/or

psychiatric disorders including depression, anxiety, apathy, fatigue, psychosis, mania, bipolar disorder and pseudobulbar affects (laughing and crying)[8,11]. Risk factors found to be highly related to these post-stroke neuropsychiatric complications include age, the most cited, gender, lifestyle, stroke type, medication, lesion location, and comorbidities. Recent studies have revealed several critical mechanisms underlying these complications, namely inflammatory response, dysregulation of the hypothalamic pituitary adrenal axis, cholinergic dysfunction, reduced level of 5-hydroxytryptamine, glutamate-mediated excitotoxicity and mitochondrial dysfunction. Although these disorders have been proven to cause poorer recovery in affected patients, recent studies have come up with both pharmacological and non-pharmacological treatment measures that altogether lead to remission for most stroke survivors and reset them on the right track for rehabilitation.

B. IN AFRICA

A few studies have been carried out in Africa on post-stroke psychiatric disorders. In Kenya, Wairoto *et al* found an overall prevalence of psychiatric morbidity of 32.85 % among 210 stroke patients who constituted the study population. Specific diagnoses recorded were depression (19%) and generalized anxiety disorder (9.5 %). Alcohol dependence (2.3%) and bulimia nervosa (1.9 %) were also described. Sociodemographic variables were not significantly associated with psychiatric morbidity in this study[45]. Atigossou *et al* found that depression and anxiety had a negative impact on activity and health related quality of life among chronic stroke survivors in Benin[46]. A metanalysis done by Ojagbemi *et al* in 2017, reported a pooled frequency of clinically diagnosed PSD of 31% versus 13.9% in healthy control pairs in SSA. This prevalence was mainly affected by methods of depression ascertainment in the various studies examined. PSD was significantly associated with low education, cognitive impairment, physical disability, poor quality of life, and divorced marital status[16]. In 2020, Ojagbemi found that over one-third of stroke survivors in SSA will develop anxiety within one year following their stroke, with the hemorrhagic stroke type being an important predictor of anxiety [47].

C. IN CAMEROON

In Cameroon, the prevalence rate of stroke is 7.3%, and its mortality rates are 26.7% during the first month and 31.7% in the first three months after the stroke event [6,48]. Angwafor *et al* found a prevalence of PSD of 45.6% among stroke survivors in 2 hospitals in Cameroon[19]. Little data was found concerning post-stroke neuropsychiatric complications in our setting.

CHAPTER III : METHODOLOGY

III.1. STUDY DESIGN

We carried out a hospital-based cross-sectional and analytical study.

III.2. STUDY PERIOD

This study lasted for 7 months, from 1st November 2023 to 28th May 2024.

III.3. STUDY AREA AND SETTING

This study was carried out in the Neurology service of the Yaoundé Central Hospital.

Yaoundé, the city spread over seven hills, is the capital city of Cameroon with a population of about 2.8 million inhabitants spread over 7 subdivisions. It is an administrative zone, with high level of urbanization, industrialization and education. Owing to its high-profile central structures, Yaoundé has a higher standard of living, healthcare and security than the rest of the country.

The Yaoundé Central Hospital (YCH)

The YCH is a reference hospital found in the capital city, Yaoundé. It has several specialized units including a Surgery and Specialties Unit, an Anesthesia and Intensive Care Unit, an Obstetrics/Gynecology Unit and a Medicine and Specialties Unit. It receives a daily influx of patients from Yaoundé and its environs, and from other health facilities. The Neurology service of the hospital includes a hospitalization section, a nurse station and 3 consultation offices. It receives patients with various neurological conditions of which stroke makes up a good number of cases.

III.4. STUDY POPULATION

Our study population consisted of all out-patient stroke survivors followed at the Neurology service of the YCH during our study period. Participants were further screened based on the criteria below.

III.4.1. Inclusion Criteria

We included in our study:

- Individuals with a confirmed diagnosis of stroke by neuroimaging (CT-Scan or MRI) and/or a neurologist
- Individuals aged 18years or older
- Individuals between 2 weeks and 12 months following their stroke event
- Individuals who gave an informed written or oral consent

III.4.2. Exclusion Criteria

- Individuals with major cognitive impairment hindering data collection
- Individuals with major hearing difficulties, severe aphasia
- Individuals who withdrew their consent at any time during the study

III.5 SAMPLING

III.5.1. Sampling Method

Sampling was done in a consecutive manner. All participants fulfilling the eligibility criteria were included in our study until our sample size was attained.

III.5.2. Sample Size Estimation

Based on our study design, we calculated the sample size using the formula for cross-sectional studies as follows;

$$\mathbf{N = (Z^2.pq)/e^2}$$

Z = Variance

p = Prevalence of stroke in Cameroon

q = 1- p

e = Precision

For a risk α of 5% and confidence interval of 95%;

Z = 1.96

p = 7.3% [6]

e = 5%

$N = [(1.96)^2(0.073X\ 0.927)]/\ (0.05)^2$

$= 103.98596544$

$= 104$

Therefore, with a prevalence of 7.3%[6], we needed to recruit at least 104 participants

III.6. PROCEDURE

III.6.1 Administrative Formalities

After writing and presenting the research protocol to our supervisors for approval, we applied for ethical clearance and research authorization from the Institutional Review Board of the Faculty of Medicine and Biomedical Sciences of the University of Yaoundé I and from the Director of the Yaoundé Central Hospital respectively.

III.6.2. Recruitment

Patients fulfilling eligibility criteria were recruited at the out-patient section of the Neurology service of the YCH over a period of 04 months. Participants were approached during follow up visits, their written/oral consent was obtained depending on their ability to read and/or write after a comprehensive explanation of the study. Relevant data was obtained from consented subjects with the use of well structured, pre-established questionnaires for the research.

III.6.3. Variables

The variables collected included:

i. **Sociodemographic data**: Name, Age, Sex, Marital Status, Occupation, Religion, Handedness, Socioeconomic status, Level of education

ii. **Past History**: Stroke, Cardiovascular risk factors

iii. **Stroke Clinical Parameters**: type, site, lesion, severity (NIHSS scale), time

iv. **Psychiatric Disorders:** these were assessed using the following tools

a. Depression was assessed using the PHQ-9 questionnaire

1. The Patient Health Questionnaire-9 (PHQ-9)[41]

The PHQ-9 is a version of the PRIME-MD tool (self-administered) for the diagnosis of mental disorders. It has 9 criteria scored from 0 (not at all) to 3(nearly every day). It is a useful clinical and research tool due to its brevity, reliability and validity in diagnosing and measuring severity of depression. It is interpreted as:

- A score < 5 indicates the absence of a depressive disorder
- A score between 5 and 9 indicates a mild depressive disorder
- A score between 10 and 14 shows moderate depression
- A score between 15 and 19 shows moderately severe depression
- A score greater than 20 shows severe depression

b. Anxiety was assessed using the HADS

2. The Hospital Anxiety and Depression Scale (HADS)[49]

It is a self-report rating scale of 14 items, used to diagnose depression and anxiety with 7 items in each subscale. Each item has scores ranging from 0 to 3, making a total score of 21 per subscale. It is interpreted for each subscale as follows;

- A score < 7 indicates a non-case
- A score between 8 and 10 is mild
- A score between 11 and 14 is moderate
- A score ≥ 15 is severe

c. Apathy was assessed using the AES

3. The Apathy Evaluation Scale (AES)[50]

It is a tool used to quantify and characterize apathy in adults, developed by Marin Roberts MD. It has 3 versions; self-rated, clinician-rated and informant versions. It consists of 18 items, each scored between 1 and 4, giving a total score range between 18 to 72, with higher scores indicating more apathy.

d. Fatigue was assessed using the FSS

4. The Fatigue Severity Scale (FSS)[51]

It is a short questionnaire which contains nine statements that rate the severity of fatigue symptoms. Each statement is rated between 1 to 7 where 1 indicates a strong disagreement and 7 indicates a strong agreement with the statement. The numbers chosen by the patient for each question are the summed up to give a total score.

- If the score is less that 36, it indicates the absence of fatigue
- If the score is greater than 36, it indicates the presence of fatigue.

e. Psychosis was assessed based on the presence/absence of delusions and/or hallucinations

f. Mania was assessed using the **YMRS**

5. The Young Mania Rating Scale (YMRS)[52]

It is frequently used to assess manic symptoms. It has 11 items and is based on the patient's subjective report of his or her clinical condition over the previous 48hours. Clinical observations made during the course of the clinical interview provide further information. Four items are graded on a 0 to 8 scale (irritability, speech, thought content, and disruptive/aggressive behavior), while the seven others are graded on a 0 to 4 scale.

v. **Functional Outcome** was measured using the BI and MRS

6. The Barthel Index (BI)[53]

It measures the extent to which a person can function independently and have mobility in their Activities of Daily Living (ADL) and indicates the need for assistance and care. It was developed for use in rehabilitation patients with stroke. The score is allotted as such; 0 or 5 points per item for bathing and grooming; 0,5, or 10 points for feeding, dressing, bowel control, bladder control, toilet use and stairs; 0,5, 10 or 15 points for transfers and mobility. The index yields a total score of 100, the higher the score the greater the degree of functional independence of the individual.

- A score <20 indicates a totally dependent person
- A score between 20 and 39 indicates a very dependent person
- A score between 40 and 59 indicates a partially dependent person

- A score between 60 and 79 shows a minimally dependent patient
- A score between 80 and 100 indicates that the patient can live independently

7. The Modified Rankin Scale (MRS)[54,55]

It is used to measure the degree of disability in patients who have had a stroke as follows

- 0; no symptoms at all
- 1; No significant disability despite symptoms; able to carry out all usual duties and activities
- 2; Slight disability; unable to carry out all previous activities but able to look after own affairs without assistance
- 3; Moderate disability; requiring some help but able to walk without assistance
- 4; Moderately severe disability; unable to walk without assistance, unable to attend to own bodily needs without assistance
- 5; Severe disability; bedridden, incontinent requiring constant nursing care and attention
- 6; Dead

A score from 0 to 2 indicates a good functional outcome while from 3 to 6 indicates a poor functional outcome.

III.7. DATA COLLECTION AND ANALYSIS

III.7.1. Material for Data Collection

- Pre-established consent forms
- Pre-established questionnaires (PHQ-9, HADS, AES, FSS, YMRS, BI, MRS)
- Patients and their medical records
- Pens, pencils, erasers
- Calculator
- Ream of A4 papers

III.7.2. Material for Patient Examination

- Weight balance
- Blood pressure machine
- Pulse oximeter
- Thermometer
- Stopwatch
- Reflex hammer
- Alcoholic solution

III.7.3. Statistical Analysis

Collected data was entered and analyzed using the IBM SPSS (Statistical Package for Social Sciences) version 25.0. Quantitative variables were presented as mean and standard deviation while qualitative variables were presented as frequencies and percentages. The association between two quantitative variables was established using the Student-t test while the association between two qualitative variables was established using the Chi square test. Factors associated with any of the psychiatric disorders studied were determined using a multivariate analysis after a bivariate analysis. Statistical significance was determined with a p value less than 0.05 and a confidence interval of 95%.

III.7.4. Material for Data Management

- Personal Computer
- Flash Drive
- IBM SPSS Software package
- Smart phone
- Data connection
- External hard drive

III.8. HUMAN RESOURCES

- Director and Co-directors
- Statistician
- Investigator

III.9. ETHICAL CONSIDERATIONS

Ethical clearance was sought and obtained from the Institutional Ethical Review Board of the Faculty of Medicine and Biomedical Sciences before data collection. We also obtained authorizations from the Director of the Yaoundé Central Hospital and from the Head of the Neurology service.

A written consent form was made and signed by participants/caretakers after detailed explanation of the study and allowance for clarifications where needed. Confidentiality was ensured by assigning randomly generated codes to every participant and these codes used at every step of documentation. All data collected was used only for the research.

CHAPTER IV : RESULTS

IV.1. RECRUITMENT SCHEME

In our study, 107 participants were assessed for eligibility, we excluded 11 participants; 5 participants were severely aphasic, 2 participants had severe cognitive impairment, and 4 participants withdrew their consent during the interview. We finally retained a total of 96 participants who were enrolled in the study and whose data was analyzed.

(**Figure 3**)

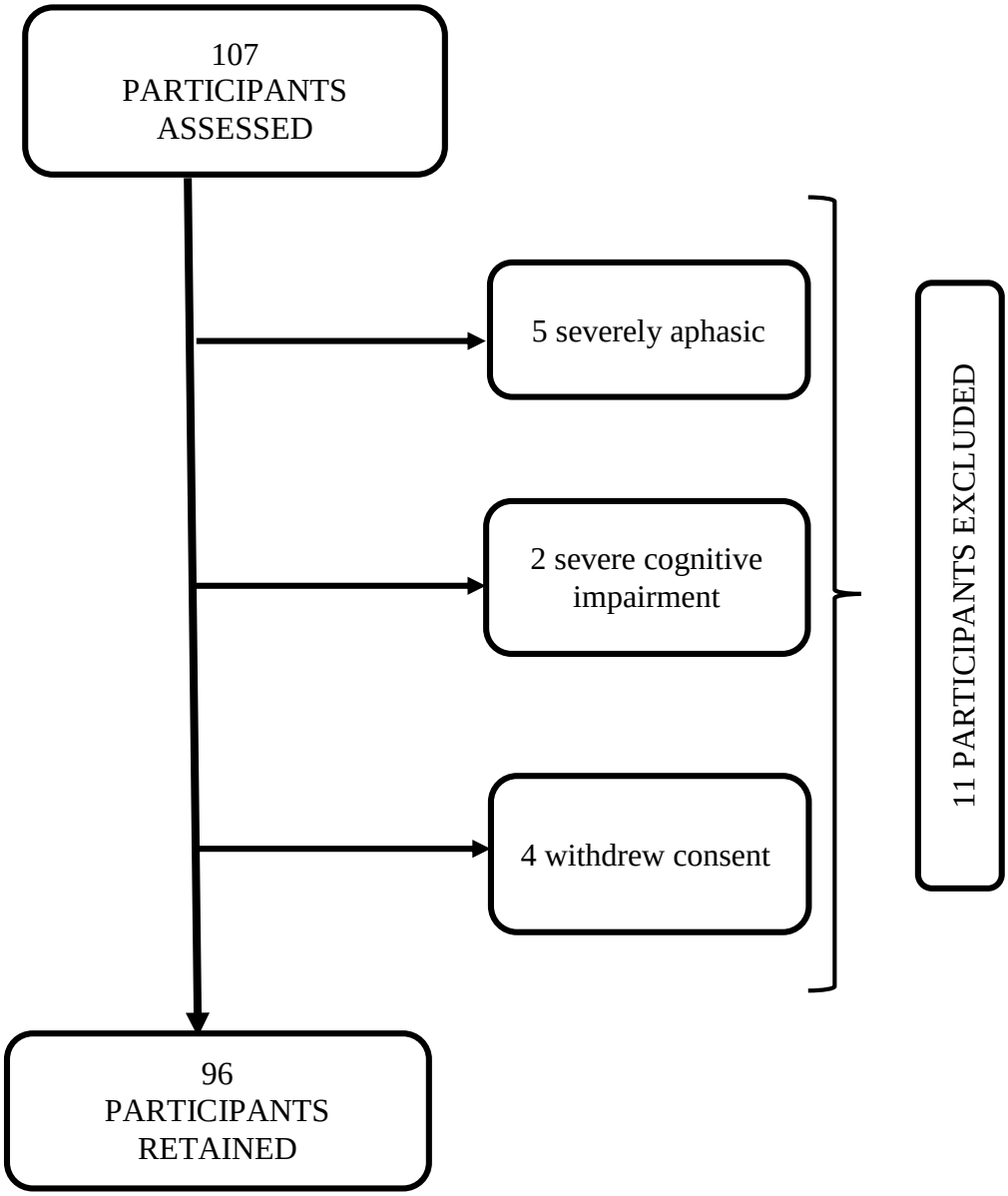

Figure 4: Triage of participants.

IV.2. GENERAL CHARACTERISTICS OF THE STUDY POPULATION

IV.2.1. Sociodemographic Characteristics

In our study, the ages of the participants ranged from 30-98years with a mean age of 61.1± 13.0years. Females (52.1%) were more represented than males, with a male to female sex ratio of 0.92. Among the participants, 42.7% had at most a primary level of education, 45.8% had secondary education and the remaining 11.5% had a higher level of education. Also, 66.7% of the participants in our study were married and 33.3% were not.

Table I: Socio-demographic characteristics of the study population

Variables	Frequency	Percentages (%)
Age (in years)		
< 65	62	64.6
≥65	34	35.4
Sex		
Male	46	47.9
Female	50	52.1
Level of Education		
None	17	17.7
Primary	24	25
Secondary	44	45.8
Higher	11	11.5
Marital Status		
Single	9	9.4
Married	64	66.7
Divorced	3	3.1
Widow(er)	20	20.8

IV.2.2. Cardio-Vascular Risk Factors

In our study, 67.7% of participants had a history of hypertension, 17.7% were diabetic, 6.3% had a heart condition, 2.1% were followed for dyslipidemia and 12.5% were experiencing at least a second episode of stroke. We had only 1 participant with HIV. Also 64.6% of our participants consumed alcohol regularly, 14.6% had been chronic smokers and 47.9% had a sedentary lifestyle.

Table II: Cardio-Vascular Risk Factors Observed in the Study Population

Risk factor	Frequency	Percentages (%)
Hypertension	65	67.7
Alcohol consumption	62	64.6
Sedentary lifestyle	46	47.9
Diabetes Mellitus	17	17.7
Smoking	14	14.6
Reccurent Stroke	12	12.5
Cardiopathy	6	6.3
Dyslipidemia	2	2.1
HIV	1	1

IV.2.3. Clinical Characteristics of Stroke

The duration of stroke in our population ranged from 2 weeks to 11 months, with a median duration of 1.8(0.8-4.5) months. The greatest proportion of participants were between 1 and 6 months following their stroke (47.9%). The BMI in our population varied from 13.9 to 37.6 Kg/m^2, with a median at 23.1[20.7-26.3] Kg/m^2 with 4.2% obese participants.

The NIHSS scores of participants in our study ranged from 0 to 13, with a median of 1[0 – 3.75], 79.2% of the participants presented with mild stroke.

Table III: Duration of Stroke and Clinical characteristics of Stroke in the Study Population

Variables	Frequency	Percentages (%)
Duration of Stroke (in months)		
< 1	34	35.4
[1- 6[	46	47.9
[6-12]	16	16.7
BMI (Kg/m2)		
Emaciated (<18.5)	8	8.3
Normal ([18.5-25[)	56	58.3
Overweight ([25-30[)	28	29.2
Obesity (≥30)	4	4.2
Hemiparesis		
Left hemiparesis	43	44.8
Right hemiparesis	53	55.2
NIHSS		
<5	76	79.2
[5-20]	20	20.8
>20		

IV.2.4. Paraclinical characteristics of Stroke in the study population

Ischemic stroke (68.8%) was more represented than hemorrhagic stroke in our population. The left hemisphere was affected in 54.2% of participants, and 3.1% of participants had a lesion in both hemispheres. The two main documented etiologies of ischemic stroke in our population were atherosclerosis (81%) and cardio-embolism (14.3%). All patients were placed on appropriate treatment measures for stroke depending on the type. We noted that 39.6% of participants were on treatment with antidepressants, 29.2% were on treatment with anxiolytic drugs and 57.3% currently followed physiotherapy.

Table IV: Paraclinical characteristics of Stroke in the study population

Variables	Frequency	Percentages (%)
Type of stroke		
Ischemic	66	68.8
Hemorrhagic	30	31.3
Affected Hemisphere		
Left	52	54.2
Right	41	42.7
Bilateral	3	3.1
Affected dominant hemisphere		
Yes	54	56.3
No	42	43.8
Affected Artery		
ACA Superficial	4	4.2
ACA Deep	2	2.1
MCA Superficial	27	28.1
MCA Deep	44	45.8
PCA Superficial	5	5.2
PCA Deep	4	4.2
Lacunar	10	10.5
Stroke Etiology (n=21)		
Atherosclerosis	17	81
Cardio-embolism	3	14.3
Small vessel disease		
Indeterminate	1	4.8

IV.2.5. Level of Dependence and Functional Status in Study Population

Among our participants, 66.7% were minimally dependent and could take care of their personal needs and 6.3% were independent. Most of the participants in our study (59.4%) had a good functional outcome according to the mRS scale. The rest had a poor functional outcome.

Table V: Level of Dependence and Functional Outcome in Study Population

Variables	Frequency	Percentages (%)
Barthel Index		
Totally dependent 0-19	6	6.3
Very dependent 20-39	12	12.5
Partially dependent 40-59	14	14.6
Minimally dependent 60-79	64	66.7
Independent 80-100	6	6.3
mRS		
Good functional outcome <3	57	59.4
Poor functional outcome ≥3	39	40.6

IV.3. PREVALENCE OF POST-STROKE PSYCHIATRIC DISORDERS IN THE POPULATION

Among our 96 participants, 36 (37.5%) of them had a psychiatric disorder. Post-stroke depression was present in 28.1% of our total population. Among depressive patients, 74.1% had mild depression, 18.5% had moderate depression and 7.4% were severely depressed. No participant was diagnosed with mania or psychosis in our population.

Concerning co-occuring psychiatric disorders, we had no participants with both depression and anxiety only, 4 participants (4.2%) had both depression, anxiety and apathy. Both depression and fatigue were present in 4.2% of participants, 8.3% of participants had both depression and apathy while 2.1% had both apathy and fatigue.

Table VI: Prevalence of post-stroke psychiatric disorders in the study population

Diseases	Frequency	Percentage (95% CI)
Depression		
Yes	27	28.1 (19.8-37.5)
No	69	71.9
Anxiety		
Yes	5	5.2 (1-10.4)
No	91	94.8
Apathy		
Yes	23	24 (15.6-32.3)
No	73	76
Fatigue		
Yes	15	15.6 (8.3-22.9)
No	81	84.4
Mania		
Yes	-	0
No	96	100
Psychosis		
Yes	-	0
No	96	100

IV.4. FACTORS ASSOCIATED WITH POST-STROKE PSYCHIATRIC DISORDERS IN THE POPULATION

IV.4.1. Factors Associated with Post-Stroke Depression

A low level of education, sedentary lifestyle and low BMI were the factors which showed significant statistical association with post-stroke depression. However, other factors such as a history of diabetes, history of cardiac disease, history of stroke and stroke severity were close to significance. The independent factors identified following multivariate analysis were a sedentary lifestyle and a history of stroke.

Table VII: Sociodemographic characteristics associated with Post-Stroke Depression

Variables	PSD		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Age				
< 65	14 (22.6)	48 (77.4)	0.471 (0.189 - 1.174)	0.103
≥65	13 (38.2)	21 (61.8)		
Sex				
Male	15 (32.6)	31 (67.4)	1.532 (0.626 - 3.75)	0.349
Female	12 (24)	38 (76)		
Level of education				
None/primary	16 (39)	25 (61)	2.56 (1.029 - 6.367)	**0.040**
Secondary/university	11 (20)	44 (80)		
Marital status				
Married	12 (37.5)	20 (62.5)	1.96 (0.781 - 4.919)	0.149
Not married	15 (23.4)	49 (76.6)		

Table VIII: Cardio-Vascular Risk Factors associated with Post-Stroke Depression

Variables	PSD		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Comorbidities				
Hypertension	18 (27.7)	47 (72.3)	0.936 (0.363 - 2.413)	0.891
Diabetes	8 (47.1)	9 (52.9)	2.807 (0.95 - 8.292)	**0.075**
Dyslipidemia	0 (0)	2 (100)	-	1
HIV	0 (0)	1 (100)	-	1
Cardiopathy	4 (66.7)	2 (33.3)	5.826 (1 - 33.939)	**0.051**
Recurrent Stroke	6 (50)	6 (50)	3.00 (0.873 - 10.312)	**0.09**
Alcoholism	14 (22.6)	48 (77.4)	0.471 (0.189 - 1.174)	0.103
Smoking	4 (28.6)	10 (71.4)	1.026 (0.292 - 3.602)	1
Sedentary lifestyle	6 (13)	40 (87)	0.207 (0.074 -0.578)	**0.002**

Table IX: Clinical characteristics associated with Post-Stroke Depression

Variables	PSD		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Duration of Stroke (in months)				
<6	23 (28.7)	57 (71.3)	1.211 (0.354 - 4.145)	1
≥6	4 (25)	12 (75)		
BMI (Kg/m2)				
Emaciated (<18.5)	6 (75)	2 (25)	7.50 (1.367 - 41.14)	**0.016**
Normal ([18.5-25[)	16 (28.6)	40 (71.4)	1	1
≥25	5 (15.6)	27 (84.4)	0.463 (0.152 - 1.414)	0.17

Table X: Paraclinical characteristics associated with Post-Stroke Depression

Variables	PSD		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Type of stroke				
Ischemic	20 (30.3)	46 (69.7)	1.429 (0.528 - 3.867)	0.481
Hemorrhagic	7 (23.3)	23 (76.7)		
Affected dominant hemisphere				
Yes	14 (25.9)	40 (74.1)	0.781 (0.319 - 1.908)	0.587
No	13 (31)	29 (69)		
Side of brain lesion				
Left	15 (28.8)	37 (71.2)	0.98 (0.398 - 2.413)	0.964
Right	12 (29.3)	29 (70.7)		
Stroke severity				
NIHSS 5-25	9 (45)	11 (55)	2.636 (0.944 - 7.366)	**0.059**
NIHSS <5	18 (23.7)	58 (76.3)		
Antidepressant				
Yes	8 (21.1)	30 (78.9)	0.547 (0.211 - 1.42)	0.212
No	19 (32.8)	39 (67.2)		
Physiotherapy				
Yes	16 (29.1)	39 (70.9)	1.119 (0.453 - 2.761)	0.807
No	11 (26.8)	30 (73.2)		

Table XI: Multivariate analysis of factors associated to Post-Stroke Depression

Variables	Adjusted OR (95% CI)	Adjusted p value
Level of education (Low/high)	2.3 (0.6 - 8.6)	0.210
Diabetes	2.1 (0.5 - 8.9)	0.333
Cardiopathy	1.3 (0.15 - 12.56)	0.777
Recurrent Stroke	10.7 (0.93 - 123.47)	**0.057**
Sedentary lifestyle	12.8 (1.48 - 110.60)	**0.02**
BMI (Emaciated/normal)	0.35 (0.05 - 2.24)	0.266

IV.4.2. Factors Associated With Post-Stroke Anxiety

We found no factors with a statistically significant association to post-stroke anxiety in our study population. However, having a moderately severe stroke and having a stroke in the dominant hemisphere were close to significance.

Table XII: Sociodemographic characteristics associated with PSAn

Variables	Anxiety		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Age				
< 65	4 (6.5)	58 (93.5)	2.276 (0.244 - 21.22)	0.653
≥65	1 (2.9)	33 (97.1)		
Sex				
Male	3 (6.5)	43 (93.5)	1.674 (0.267 - 10.50)	0.668
Female	2 (4)	48 (96)		
Level of education				
None/primary	1 (2.4)	40 (97.6)	0.319 (0.034 - 2.965)	0.389
Secondary/university	4 (7.3)	51 (92.7)		
Marital status				
Not married	0 (0)	32 (100)	-	0.166
Married	5 (7.8)	59 (92.2)		

Table XIII: Cardiovascular Risk Factors associated with PSAn

Risk Factor	Anxiety		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Hypertension	3 (4.6)	62 (95.4)	0.702 (0.111 - 4.43)	0.657
Diabetes	1 (5.9)	16 (94.1)	1.172 (0.123 - 11.194)	1
Dyslipidemia	0 (0)	2 (100)	-	1
HIV	0 (0)	1 (100)	-	1
Cardiopathy	0 (0)	6 (100)	-	1
Stroke	0 (0)	12 (100)	-	1
Alcoholism	2 (3.2)	60 (96.8)	0.344 (0.055 - 2.171)	0.343
Smoking	0 (0)	14 (100)	-	1
Sedentary lifestyle	2 (4.3)	44 (95.7)	0.712 (0.114 - 4.465)	1

Table XIV: Clinical and Paraclinical characteristics associated with Post-Stroke Anxiety

Variables	Anxiety		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Duration of Stroke (in months)				
<6	4 (5)	76 (95)	0.789 (0.082 - 7.567)	1
≥6	1 (6.3)	15 (93.8)		
Type of stroke				
Ischemic	3 (4.5)	63 (95.5)	0.667 (0.105 - 4.213)	0.646
Hemorrhagic	2 (6.7)	28 (93.3)		
Affected dominant hemisphere				
Yes	5 (9.3)	49 (90.7)	-	**0.066**
No	0 (0)	42 (100)		
Side of brain lesion				
Left	5 (9.6)	47 (90.4)	-	**0.064**
Right	0 (0)	41 (100)		
Stroke severity				
NIHSS 5-25	3 (15)	17 (85)	6.529 (1.011 - 42.163)	**0.059**
NIHSS <5	2 (2.6)	74 (97.4)		

IV.4.3. Factors associated with Post-Stroke Apathy

A sedentary lifestyle, moderately severe stroke, left-sided brain lesion and post-stroke fatigue were the factors significantly associated with post-stroke apathy in our study. Only the side of brain lesion was not an independent factor following multivariate analysis.

Table XV: Sociodemographic characteristics and Comorbidities associated with PSA

Variables	Apathy		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Age				
< 65	13 (21)	49 (79)	0.637 (0.244 - 1.66)	0.354
≥65	10 (29.4)	24 (70.6)		
Sex				
Male	13 (28.3)	33 (71.7)	1.576 (0.613 - 4.052)	0.343
Female	10 (20)	40 (80)		
Level of education				
None/primary	12 (29.3)	29 (70.7)	1.655 (0.645 - 4.25)	0.293
Secondary/university	11 (20)	44 (80)		
Marital status				
Married	10 (31.3)	22 (68.8)	1.783 (0.68 - 4.677)	0.237
Not maried	13 (20.3)	51 (79.7)		
Comorbidities				
Hypertension	14 (21.5)	51 (78.5)	0.671 (0.253 - 1.779)	0.421
Diabetes	5 (29.4)	12 (70.6)	1.412 (0.439 - 4.542)	0.561
Dyslipidemia	0 (0)	2 (100)	-	1
HIV	0 (0)	1 (100)	-	1
Cardiopathy	1 (16.7)	5 (83.3)	0.618 (0.068 - 5.58)	1
Stroke	4 (33.3)	8 (66.7)	1.711 (0.464 - 6.305)	0.473
Alcoholism	12 (19.4)	50 (80.6)	0.502 (0.193 - 1.305)	0.211
Smoking	3 (21.4)	11 (78.6)	0.845 (0.214 - 3.335)	1
Sedentary lifestyle	6 (13)	40 (87)	0.291 (0.103 - 0.823)	**0.016**

Table XVI: Clinical and Paraclinical characteristics associated with Post-Stroke Apathy

Variables	Apathy		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Duration of Stroke (in months)				
<6	21 (26.3)	59 (73.8)	2.492 (0.522 - 11.893)	0.343
≥6	2 (12.5)	14 (87.5)		
Type of stroke				
Ischemic	15 (22.7)	51 (77.3)	0.809 (0.3 - 2.183)	0.675
Hemorrhagic	8 (26.7)	22 (73.3)		
Affected dominant hemisphere				
Yes	16 (29.6)	38 (70.4)	2.105 (0.775 - 5.722)	0.14
No	7 (16.7)	35 (83.3)		
Side of brain lesion				
Left	17 (32.7)	35 (67.3)	3.497 (1.164 - 10.509)	**0.021**
Right	5 (12.2)	36 (87.8)		
Stroke severity				
NIHSS 5-25	10 (50)	10 (50)	4.846 (1.678 – 13.995)	**0.006**
NIHSS <5	13 (17.1)	63 (82.9)		
Post-stroke fatigue				
Yes	16 (59.3)	11 (40.7)	12.883 (4.308 - 38.528)	**<0.001**
No	7 (10.1)	62 (89.9)		

Table XVII: Multivariate analysis of factors associated to Post-Stroke Apathy

Variables	Adjusted OR (95% CI)	Adjusted p value
Sedentary lifestyle	0.036 (0.003 - 0.493)	**0.013**
Fatigue	19.418 (1.798 - 209.659)	**0.015**
Side of brain lesion	6.28 (0.862 - 45.775)	0.070
Stroke Severity	10.654 (1.359 - 83.559)	**0.024**

IV.4.4. Factors Associated with Post-Stroke Fatigue

The factors that were significantly associated to post-stroke fatigue in our study were age greater than 65years, male sex, having post-stroke depression and use of anti-depressants. The independent factors following multivariate analysis were age and antidepressant use.

Table XVIII: Sociodemographic characteristics associated with Post-Stroke Fatigue

Variables	Fatigue		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Age				
≥65	9 (26.5)	25 (73.5)	3.36 (1.079 - 10.459)	**0.03**
<65	6 (9.7)	56 (90.3)		
Sex				
Male	11 (23.9)	35 (76.1)	3.614 (1.061 - 12.315)	**0.032**
Female	4 (8)	46 (92)		
Level of education				
None/primary	6 (14.6)	35 (85.4)	0.876 (0.285 - 2.693)	0.817
Secondary/university	9 (16.4)	46 (83.6)		
Marital status				
Married	6 (18.8)	26 (81.3)	1.41 (0.454 - 4.381)	0.551
Not married	9 (14.1)	55 (85.9)		

Table XIX: Comorbidities and Risk Factors associated with Post-Stroke Fatigue

Variables	Fatigue		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Comorbidities				
Hypertension	9 (13.8)	56 (86.2)	0.67 (0.215 - 2.084)	0.552
Diabetes	4 (23.5)	13 (76.5)	1.902 (0.524 - 6.903)	0.459
Dyslipidemia	0 (0)	2 (100)	-	1
HIV	0 (0)	1 (100)	-	1
Cardiopathy	2 (33.3)	4 (66.7)	2.962 (0.491 - 17.849)	0.235
Stroke	2 (16.7)	10 (83.3)	1.092 (0.214- 5.571)	1
Alcoholism	10 (16.1)	52 (83.9)	1.115 (0.348 - 3.578)	0.854
Smoking	3 (21.4)	11 (78.6)	1.591 (0.386 - 6.555)	0.454
Sedentary lifestyle	6 (13)	40 (87)	0.683 (0.223 - 2.097)	0.504

Table XX: Clinical and Paraclinical characteristics associated with Post-Stroke Fatigue

Variables	Fatigue		OR (95%CI)	p value
	Yes n(%)	**No n(%)**		
Duration of Stroke (in months)				
<6	12 (15)	68 (85)	0.765 (0.189 - 3.093)	0.711
≥6	3 (18.8)	13 (81.3)		
Type of stroke				
Ischemic	11 (16.7)	55 (83.3)	1.30 (0.378 - 4.473)	0.77
Hemorrhagic	4 (13.3)	26 (86.7)		
Affected dominant hemisphere				
Yes	8 (14.8)	46 (85.2)	0.87 (0.288 - 2.627)	0.804
No	7 (16.7)	35 (83.3)		
Side of brain lesion				
Left	7 (13.5)	45 (86.5)	0.756 (0.242 - 2.359)	0.629
Right	7 (17.1)	34 (82.9)		
Stroke severity				
NIHSS 5-25	5 (25)	15 (75)	2.2 (0.655 - 7.386)	0.296
NIHSS <5	10 (13.2)	66 (86.8)		
Post-stroke depression				
Yes	11 (40.7)	16 (59.3)	11.172 (3.14 3- 39.717)	**<0.001**
No	4 (5.8)	65 (94.2)		
Antidepressant Use				
Yes	10 (26.3)	28 (73.7)	3.786 (1.178 - 12.162)	**0.02**
No	5 (8.6)	53 (91.4)		

Table XXI: Multivariate analysis of factors associated to Post-Stroke Fatigue

Variables	Adjusted OR (95% CI)	Adjusted p value
Age (≥65/<65)	5.956 (1.585-22.380)	**0.008**
Sex (male/female)	3.641 (0.974-13.609)	0.055
Antidepressant	5.280 (1.382-20.166)	**0.015**

IV.5. EFFECTS OF POST-STROKE PSYCHIATRIC DISORDERS ON FUNCTIONAL OUTCOME IN THE POPULATION

Post-stroke Depression and Post-stroke Apathy were significantly associated with a poor functional outcome in our study population. Post-stroke Fatigue however was close to significance. The lower the Barthel Index score (more dependence), the higher the PHQ-9 score (more depression), see figure 4.

Table XXII: Effects of having a Psychiatric disorder on Functional outcome in the population

Variables	Poor functional outcome (from mRS)		OR (95%CI)	p value
	Yes n(%)	No n(%)		
Post-Stroke Depression				
Yes	18 (66.7)	9 (33.3)	4.571 (1.767 - 11.825)	**0.001**
No	21 (30.4)	48 (69.6)		
Post-Stroke Anxiety				
Yes	4 (80)	1 (20)	6.4 (0.687 - 59.616)	0.155
No	35 (38.5)	56 (61.5)		
Post-Stroke Apathy				
Yes	18 (78.3)	5 (21.7)	8.914 (2.929 - 27.127)	<**0.001**
No	21 (28.8)	52 (71.2)		
Post-Stroke Fatigue				
Yes	9 (60)	6 (40)	2.55 (0.826 - 7.871)	0.096
No	30 (37)	51 (63)		

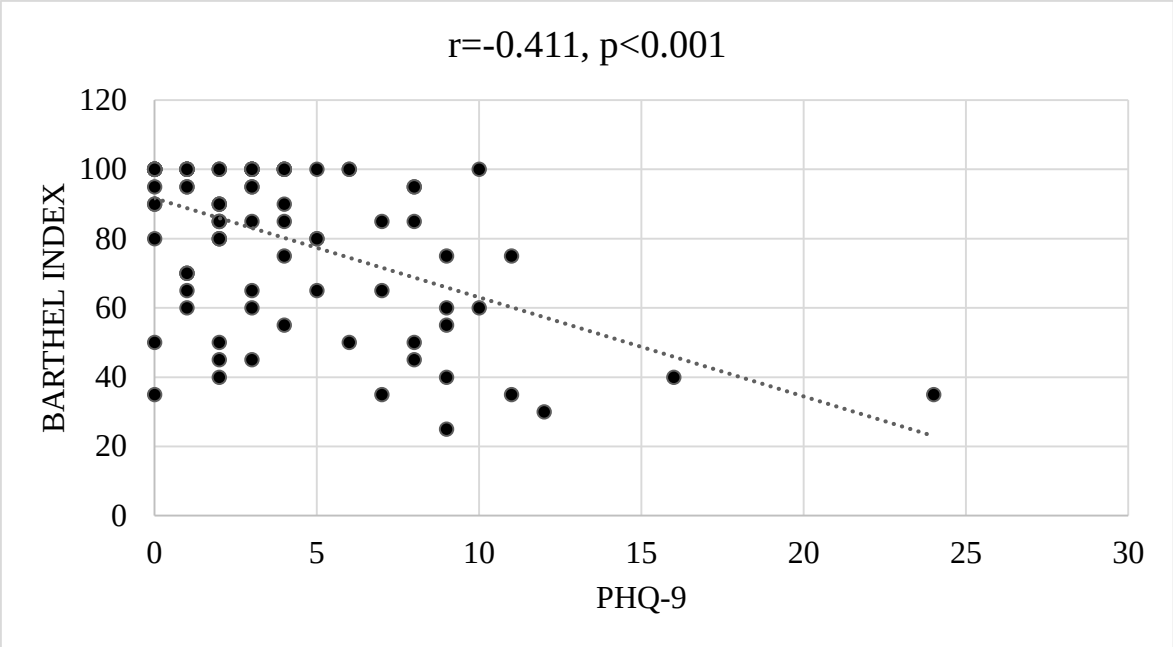

Figure 5 : Relationship between the Barthel Index and the PHQ-9 scale

CHAPTER V : DISCUSSIONS

Psychiatric disorders are a non-negligible complication following stroke. They are generally unrecognized and untreated, leading to significant retardation in the process of rehabilitation after stroke and increased morbidity in stroke survivors [56]. This study was aimed at assessing the prevalence of psychiatric disorders and their associated factors, as well as evaluating the effects of these disorders on the functional outcome of stroke survivors in our context. The results of this study show that various neuropsychiatric disorders often affect stroke survivors in our context and impede optimal recovery from the stroke event.

General Characteristics of the Study Population

In our study, the mean age of participants was 61±13.0 years. This was similar to the results obtained in a study assessing depression in stroke survivors in Ghana by Sarfo *et al* in 2017, who obtained a mean age of 62± 14.4 years in his study. Many studies seem to have such similarity in Africa and other parts of the world [17,56,57]. This is probably due to the fact that the risk for stroke doubles after every decade as from 55 years, thus most stroke patients are expected to be found around these ages. It is generally known that men are more at risk of stroke than women at all ages[4] and most studies have obtained a higher proportion of men than women in their studies, yet we had more female participants (52.1%), most of whom were overweight, suggesting that the sedentary behavior of these women increases their risk of having a stroke.

Hypertension, alcohol consumption and sedentary lifestyle were the most represented vascular risk factors in our population just as in a study published by Kamgang *et al*. on reccurent strokes in Cameroon in 2021 [6]. Ischemic stroke was the most represented stroke type in our study (68.8%), although the hemorrhagic stroke type was quite prevalent (31.3%). Sarfo *et al* also made similar findings in a study on PSD in Ghana in 2017 [57].

Prevalence of Post-Stroke Psychiatric Disorders in the Population

The prevalence of psychiatric disorders in general was found to be 37.5%. Ajiboye *et al* had a similar prevalence of psychiatric disorders of 36% among stroke survivors in Nigeria, regardless of stroke duration. However, Gabr *et al* had a prevalence of psychiatric disorders of 31% among stroke survivors in a study evaluating psychiatric disorders in acute stroke survivors in Egypt [58]. This difference was probably because of the smaller number of participants he had in his study compared to ours. These results show that almost two out of five stroke patients may be experiencing a psychiatric disorder, emphasizing the importance of psychiatric evaluation among stroke patients.

Post-stroke depression was present in 28.1% of participants in our study. This is similar to what Wubshet *et al* found in Ethiopia among first-time stroke patients, 27% of their study

population had PSD [17]. In a meta-analysis, Ojagbemi *et al* found a pooled prevalence of PSD of 31% in SSA [16], other studies done around the world have shown that the prevalence of PSD ranges between 18 to 33%. The prevalence we obtained falls within the expected range despite the slight differences in the estimates which could be attributed to the difference in the tools used in the assessment of clinically significant depression following stroke. On the other hand, the prevalence of post-stroke anxiety in our study was found to be 5.2%. This contrasts greatly with the estimated prevalence of PSAn, ranging from 18 to 34% in a recent review [8]. Ojagbemi *et al*. obtained a prevalence of post-stroke anxiety of 19.7% in Nigeria in 2017. Rafsten *et al* did a meta-analysis with studies mainly published in Europe, which revealed a pooled frequency of PSAn of 29.1%. The difference between the prevalence we obtained and those of other studies could be explained by the fact that some of our patients, 29%, were on treatment with benzodiazepines at the time of recruitment. We suppose that the anxiolytic properties exhibited by benzodiazepines certainly reduced considerably the description of anxiety symptoms by our patients. In that same study by Ojagbemi and colleagues, they also found that 71% of patients with anxiety had comorbid depression [18], whereas we had no patients with comorbid depression among those with anxiety in our population.

Many studies around the world agree that post-stroke apathy has a prevalence of about one out of three patients [8,11,59], in our study we found a prevalence of 24% for post-stroke apathy. In a review on PSA done by Willem and colleagues, it was shown that studies which used a clinician-rated specific scale for apathy and which were limited only to out-patients obtained a prevalence of 25.6% for PSA [60]. We used similar methods and obtained similar findings. The prevalence of post-stroke fatigue in our population was 15.6%. Studies around the world have estimated the prevalence of PSF to be between 25 and 85% [15,42] although a more recent global metanalysis revealed a pooled frequency of PSF of 47% [61]. Alghamdi *et al* had a prevalence of PSF of 48% among Indian stroke survivors with a longer duration of stroke than ours. Chen *et al* highlighted a great variation in the different prevalence rates obtained around the world as a result of the difference in the assessment tools used [62]. Also, knowing that post-stroke fatigue increases with increase in the duration of stroke [42], the fact that most of our participants had a duration of stroke less than 6months could explain why we obtained such a low prevalence as compared to others.

Post-stroke mania was not found in our population. This was to be expected since most studies agree that mania is a rare sequelae of stroke, occuring in less than 1% of stroke survivors [44]. Studies emphasized that it is relatively rare as compared to depression and other mood disorders[63]. In a review on post-stroke psychosis done by Stangeland *et al* in 2018, most studies seemed to agree that post-stroke psychosis had a 4.8% prevalence among stroke

survivors. In our population, there was no participant with post-stroke psychosis, probably because the mean time to onset of PSP following stroke was estimated to be about 6.1 months [43] and most of our participants had a duration of stroke of less than 6months. Also sample sizes in these studies were much larger than ours.

Factors Associated with Post-Stroke Psychiatric Disorders in our Population

In our study, we found that a low level of education, sedentary lifestyle and low BMI were associated with PSD. A review done by Ojagbemi et al in SSA showed that low level of education was significantly associated with PSD[16]. Various studies also found gender, marital status, stroke severity, and left hemisphere lesions as factors associated with PSD [8,16,57] but these were not strongly associated to PSD in our study. However, recurrent stroke, history of cardiac disease and diabetes were close to significance. We did not find another study which showed low BMI as an associated factor to PSD. We however considered this to be important since loss of appetite, being a key symptom in PSD, could explain this emaciation in PSD patients in our context.

There were no significantly associated factors to post-stroke anxiety in our study due to the low proportion of post-stroke anxiety in our population. However, having a stroke in the dominant hemisphere, stroke severity and side of lesion were close to significance. These factors were significantly associated with PSAn in other studies [8,18,47].

Sedentary lifestyle, side of brain lesion, stroke severity and presence of post-stroke fatigue were significantly associated with post-stroke apathy. A review done by Tay *et al* in 2021 on post-stroke apathy, showed that sedentary lifestyle was a significantly associated factor with PSA [34]. In another review, PSA was said to be associated with fatigue but independent of stroke severity [8]. Except for stroke severity, our results largely agreed with the findings of other researchers on PSA. Disability in our context, especially suddenly acquired disability from stroke, tends to weigh very heavily on the affected persons due to lack of efficient supportive mechanisms to help alleviate the need to fend for themselves. As such severely disabled patients following stroke tend to have a sense of worthlessness which leads them to lose interest in their previous activities and relationships.

Being a male, 65 years old and above, having PSD and using antidepressants were significantly associated with post-stroke fatigue in our study. This is similar to what Vitturi *et al* described from a cohort study with 60 patients who had had a minor stroke. In that study, there were more males, and the use of antidepressants was significantly associated with PSF[64]. Just like in their study, being a male was associated to PSF in the present study although many studies showed that females were more prone to PSF [65,66] as opposed to

males. We could not find an appropriate explanation to this finding, yet we hope that subsequent research on the matter will give us more insight to better understand the phenomenon.

Effect of Psychiatric Disorders on Functional Outcome

In our study, post-stroke depression and post-stroke apathy only were significantly associated with a poor functional outcome (mRS ≥ 3) while post-stroke fatigue was only close to significance. Post-stroke anxiety was not significantly associated with poor functional outcome according to the mRS scale in our study, we found only one study which emphasized that PSAn was associated with poor quality of life [47]. Many studies around the world agree that having a psychiatric disorder following stroke leads to a poor functional outcome. PSD was associated with a poor functional outcome and greater dependency in various studies [8,16]. Sarfo *et al* in Ghana actually showed that an increase in mRS score, that is, a poorer functional outcome, will lead to an increase in the frequency of depression [57]. Tay *et al* described post-stroke apathy to be strongly associated with a reduction in basic activities of daily living and a slower recovery since deficits in motivation lead to a reduced engagement in activities in general and rehabilitative programs [34]. PSF has also been found to be a predictor of poorer functional outcome and greater dependency in activities of daily living [15,42].

Limitations of the Study

There are quite a few limitations to our study. We excluded patients with severe aphasia and those with severe cognitive impairment, the difficulty communicating with them could not permit us to assess them efficiently although this disability could be a substrate to pull them into experiencing a psychiatric disorder. We did not exclude participants who used benzodiazepines due to their considerable number, thus we could not effectively assess post-stroke anxiety and its associated factors. Based on our study design, we cannot conclude on the causality of the observed relationship between psychiatric disorders and functional outcomes, longitudinal studies would be more adapted for this need.

CONCLUSION

The objective of our study was to assess psychiatric disorders following stroke, their prevalence and their effect on the functional recovery of stroke survivors. At the end of this study, we arrived at the following conclusions;

- The prevalence of post-stroke psychiatric disorders in general among stroke survivors in our context is 37.5%. Specifically, post-stroke depression has a prevalence of 28.1%, post-stroke anxiety has a prevalence rate of 5.2%, post-stroke apathy has a prevalence of 24% and post-stroke fatigue has a prevalence of 15.6%. Post-stroke mania and post-stroke psychosis were not found in our context.
- The main factors associated with post-stroke depression are a sedentary lifestyle, low level of education and being underweight, with the sedentary lifestyle being the most strongly associated factor. Sedentary lifestyle, stroke severity, side of lesion and fatigue are independently associated with post-stroke apathy. Older age (≥ 65years), gender, post-stroke depression and antidepressant use are factors associated with post-stroke fatigue, with age and antidepressant use being independent factors. We had no factors associated with post-stroke anxiety.
- Post-stroke depression and post-stroke apathy are strongly associated with a poor functional outcome in stroke survivors.

RECOMMENDATIONS

- **To Stroke survivors**

To freely and constantly report any symptoms of concern to their physicians to help improve their management and to engage in exercise and socialization habits despite disability, as this is important to quicken recovery from stroke.

- **To Clinicians**

To be aware that stroke survivors are often prone to psychiatric disorders which delay recovery from stroke and should therefore be considered and appropriately handled in the management of stroke patients.

- **To the Scientific committee**

To carry out larger longitudinal studies in order to firmly establish causality between psychiatric disorders and poor functional outcome in stroke survivors.

- **To the Government**

To design and implement in collaboration with medical personnel, sensitization campaigns on the risk factors for stroke so as to ensure a more efficient action against stroke in the general population.

To put in place well equipped neurovascular intensive care units, recruit and train personnel for better management of stroke in our context. Also to provide psychiatric establishments with adequate rehabilitative equipment for effective management of post-stroke psychiatric disorders in stroke patients, so as to quicken their recovery and reinsertion into the society.

To implement insurance policies or financial aid systems in order to help affected stroke patients cover up the cost of treatment and other essential life expenses for the period of their disability following stroke as this will considerably reduce the psychological burden on them and thus quicken their recovery.

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APPENDICES

APPENDIX I : QUESTIONNAIRE (ENGLISH)

QUESTIONNAIRE

TOPIC: Neurological And Psychiatric Disorders Post-Stroke: Prevalence And Effects On Functional Recovery Of Stroke Survivors In Yaoundé

Investigator: THOM NKUIDJEU CLAUDE

Date:..... Contact:.....

Code:.....

SECTION 1: SOCIO-DEMOGRAPHIC DATA

No/Q	VARIABLES	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
1.	Date of birth			_____
2.	Sex	1. Male 2. Female	1. ___ 2. ___	
3.	Address			
4.	Level of education	1. None 2. Primary 3. Secondary 4. Tertiary	1. ___ 2. ___ 3. ___ 4. ___	
5.	Profession	1. None 2. Student 3. Housewife 4. Trader 5. Farmer 6. Civil Servant 7. Private sector 8. Retired	1. ___ 2. ___ 3. ___ 4. ___ 5. ___ 6. ___ 7. ___ 8. ___	
6.	Marital status	1. Single 2. Married 3. Divorced 4. Widow(er)	1. ___ 2. ___ 3. ___ 4. ___	
7.	Religion	1. None 2. Christian 3. Muslim 4. Other	1. ___ 2. ___ 3. ___ 4. ___	
8.	Handedness	1. Left 2. Right	1. ___ 2. ___	

SECTION 2: PAST HISTORY

No/Q	VARIABLES	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
9.	Hypertension	1. Yes 2. No	1. ___ 2. ___	
10.	Diabetes	1. Yes 2. No	1. ___ 2. ___	
11.	Dyslipidemia	1. Yes 2. No	1. ___ 2. ___	
12.	Sickle cell anemia	1. Yes 2. No	1. ___ 2. ___	
13.	HIV	1. Yes 2. No	1. ___ 2. ___	
14.	Psychiatric disease	1. Yes 2. No	1. ___ 2. ___	
15.	Cardiac disease	1. Yes 2. No	1. ___ 2. ___	
16.	Stroke	1. Yes 2. No	1. ___ 2. ___	If yes Number:_____
17.	Genetic Disease	1. Yes 2. No	1. ___ 2. ___	
18.	Tobacco	1. Yes 2. No	1. ___ 2. ___	
19.	Alcohol	1. Yes 2. No	1. ___ 2. ___	
20.	Sedentary lifestyle	1. Yes 2. No	1. ___ 2. ___	

SECTION 3: CLINICAL FEATURES

No/Q	VARIABLES	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
21.	Date of onset of symptoms			
22.	Blood pressure	1. Normal 2. HTN Grade 1 3. HTN Grade 2 4. HTN Grade 3	1. ___ 2. ___ 3. ___ 4. ___	
23.	HR (beats per minute)			
24.	FR (cycles per minute)			
25.	Weight(Kg)/ Height(m)			
26.	BMI			
27.	AC(cm)			
28.	Glasgow score			
29.	Superior Functions	1. Intact 2. Altered	1. ___ 2. ___	

	If the above response is Altered, specify which function	1. Aphasia 2. Amnesia 3. Apraxia 4. Agnosia	1. ___ 2. ___ 3. ___ 4. ___	
30.	Cranial nerves	1. Intact 2. Altered	1. ___ 2. ___	
	If the above answer is Altered, name which nerve is impaired and its manifestation			
31.	Upper extremity muscle strength	1. Left 2. Right	1. ____ 2. ____	
32.	Muscle strength in the lower limbs	1. Left 2. Right	1. ____ 2. ____	
33.	Left Upper Extremity Reflexes	1. Normal 2. Hyperreflexia 3. Hyporeflexia	1. ___ 2. ___ 3. ___	
34.	Right upper extremity reflexes	1. Normal 2. Hyperreflexia 3. Hyporeflexia	1. ___ 2. ___ 3. ___	
35.	Left Lower Extremity Reflexes	1. Normal 2. Hyperreflexia 3. Hyporeflexia	1. ___ 2. ___ 3. ___	
36.	Right Lower Limb Reflexes	1. Normal 2. Hyperreflexia 3. Hyporeflexia	1. ___ 2. ___ 3. ___	
37.	Left Babinski Sign	1. Negative 2. Positive 3. Indifferent	1. ___ 2. ___ 3. ___	
38.	Right Babinski Sign	1. Negative 2. Positive 3. Indifferent	1. ___ 2. ___ 3. ___	
39.	NIHSS Score (Initial ; at stroke onset)	1. (0-5) Mild stroke 2. (6-10) Moderate Stroke 3. (11-20) Moderate to Severe Stroke 4. (> 20) Very severe stroke	1. ___ 2. ___ 3. ___ 4. ___	
40.	NIHSS Score (Current)	1. (0-4) Mild stroke 2. (6-10) Moderate Stroke 3. (11-20) Moderate to Severe Stroke 4. (> 20) Very severe stroke	1. ___ 2. ___ 3. ___ 4. ___	

THE NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS)

No	Category	Description	Score
1a	Level of Consciousness (LOC)	Alert Drowsy Stuporous Coma	0 1 2 3
1b	LOC questions (month, age)	Answers both correctly Answers 1 correctly Incorrect on both	0 1 2
1c	LOC commands (open and close eyes, grip and release non paretic hand)	Obeys both correctly Obeys 1 correctly Incorrect on both	0 1 2
2	Best gaze (follow finger)	Normal Partial gaze palsy Forced deviation	0 1 2
3	Best visual (visual fields)	No visual loss Partial hemianopia Complete hemianopia Bilateral hemianopia	0 1 2 3
4	Facial palsy (show teeth, raise brows, squeeze eyes shut)	Normal Minor Partial Complete	0 1 2 3
5	Motor arm left* (raise 90, hold 10 seconds) (preferably with palm facing up)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
6	Motor arm right* (raise 90, hold 10 seconds) (preferably with palm facing up)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
7	Motor leg left* (raise 30, hold 5 seconds)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
8	Motor leg right* (raise 30, hold 5 seconds)	No drift Drift Cannot resist gravity No effort against gravity No movement	0 1 2 3 4
9	Limb ataxia (finger-nose, heel-shin)	Absent Present in 1 limb Present in 2 limbs	0 1 2
10	Sensory (pinprick to face, arm, leg)	Normal Partial loss Severe loss	0 1 2
11	Extinction/neglect (double simultaneous testing)	No neglect Partial neglect	0 1

		Complete neglect	2
12	Dysarthria (speech clarity to "mama, baseball, huckleberry, tip-top, fifty-fifty")	Normal articulation Mild to moderate dysarthria Near to unintelligible or worse	0 1 2
13	Best language** (name items, describe pictures)	No aphasia Mild to moderate aphasia Severe aphasia Mute	0 1 2 3
	TOTAL		0-42

- For limbs with amputation, joint fusion etc, score 9 and explain
- For intubation or other physical barriers to speech, score 9 and explain. Do not add 9 to the total score

SECTION 4: PARACLINICAL FEATURES

No/Q	VARIABLES	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
41.	Type of stroke	1. Ischemic 2. Hemorrhagic	1. ___ 2. ___	
42.	Hemisphere	1. Left 2. Right 3. Bilateral	1. ___ 2. ___ 3. ___	
43.	Localization	1. Supratentorial 2. Infratentorial	1. ___ 2. ___	
	If the above answer is supratentorial, choose the exact location	1. Frontal lobe 2. Parietal lobe 3. Temporal lobe 4. Occipital lobe 5. Internal Capsule 6. Thalamus	1. ___ 2. ___ 3. ___ 4. ___ 5. ___ 6. ___	
	If the answer to 43 above is infratentorial, choose the exact location	1. Cerebellum 2. Brainstem 3.	1. ___ 2. ___ 3. ___	
44.	Affected artery	1. ACA Superficial 2. ACA Deep 3. MCA Superficial 4. MCA Deep 5. PCA Superficial 6. PCA Deep	1. ___ 2. ___ 3. ___ 4. ___ 5. ___ 6. ___	
45.	Fasting Blood Glucose (N < 1.26g/l)/ HbA1C(N<7mmol/l)	1. Low 2. Normal 3. High	1. ___ 2. ___ 3. ___	
46.	LDL level (N< 100mg/l or <1g/l)	1. Low 2. Normal 3. High	1. ___ 2. ___ 3. ___	
47.	HDL Levels	1. Low	1. ___	

	(N < 40mg/dl men N < 55mg/dl women)	2. Normal 3. High	2. ___ 3. ___	
48.	Triglyceride levels (N < 150mg/dl)	1. Low 2. Normal 3. High	1. ___ 2. ___ 3. ___	
49.	Urea (N:)	1. Normal 2. High	1. ___ 2. ___	
50.	Serum Creatinine (N:)	1. Normal 2. High	1. ___ 2. ___	
51.	Cardiac ultrasound	1. LVH 2. AF 3. Other	1. ___ 2. ___ 3. ___	
52.	Etiology of Stroke	1. Atherosclerosis of the large vessels 2. Cardioembolism 3. Small Vessel Disease 4. Indeterminate 5. Other etiology	1. ___ 2. ___ 3. ___ 4. ___ 5. ___	

SECTION 5: TREATMENT

No/Q	VARIABLES	POSSIBLE ANSWERS	ANSWER	EXACT ANSWER
53.	Antihypertensive	1. Yes 2. No	1. ___ 2. ___	
	If yes, choose the therapeutic class	1. CCB 2. ACEI 3. Diuretics 4. Beta-blockers 5. ARA II	1. ___ 2. ___ 3. ___ 4. ___ 5. ___	
54.	Anti-Diabetics	1. Yes 2. No	1. ___ 2. ___	
	If so, choose the therapeutic class	1. Insulin 2. OAD 3. Other	1. ___ 2. ___ 3. ___	Other: _____
55.	Anti-platelet aggregant	1. Yes 2. No	1. ___ 2. ___	
56.	Anti-Vitamin K	1. Yes 2. No	1. ___ 2. ___	
57.	Statins	1. Yes 2. No	1. ___ 2. ___	
58.	Anti-Depressant	1. Yes 2. No	1. ___ 2. ___	
59.	Anti-epileptic	1. Yes 2. No	1. ___ 2. ___	
60.	Osmotic Laxative	1. Yes 2. No	1. ___ 2. ___	
61.	Hypnotic or sedative	1. Yes 2. No	1. ___ 2. ___	

62.	Re-education	1. Yes 2. No	1. ___ 2. ___	
	If yes, choose the therapy(s)	1. Physiotherapy 2. Speech therapy	1. ___ 2. ___	Number of sessions:_____
63.	Psychotherapy	1. Yes 2. No	1. ____ 2. ____	
64.	Rehabilitation personnel	1. Qualified personnel 2. Family Member	1. ___ 2. ___	

SECTION 6: EVALUATION OF PSYCHIATRIC DISORDERS

A. THE PATIENT HEALTH QUESTIONNAIRE-9 (PHQ-9)

Over the last 2 weeks, how often have you been bothered by any of the following problems?

No	Symptom	Not at all	Several days	More than half the days	Nearly every day
1	Little interest or pleasure in doing things	0	1	2	3
2	Feeling down, depressed or hopeless	0	1	2	3
3	Trouble falling or staying asleep or sleeping too much	0	1	2	3
4	Feeling tired or having little energy	0	1	2	3
5	Poor appetite or over eating	0	1	2	3
6	Feeling bad about yourself -or that you are a failure or have let yourself or your family down	0	1	2	3
7	Trouble concentrating on things such as reading a book or watching television	0	1	2	3
8	Moving or speaking so slowly that other people could have noticed or the opposite -being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9	Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3
	SUM				
	TOTAL SCORE				

If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home or get along with other people?

Not difficult at all (__) Somewhat difficult (__) Very difficult (__) Extremely difficult(__)

B. THE HOSPITAL ANXIETY AND DEPRESSION SCALE (HADS)

No	A/D	Symptom/ Score	3	2	1	0
1.	A	I feel tense or 'wound' up ____	Most of the time	A lot of the time	From time to time occasionally	Not at all
2.	D	I still enjoy the things I used to enjoy ____	Hardly at all	Only a little	Not quite so much	Definitely as much
3.	A	I get a sort of frightened feeling as if something awful is about to happen _____	Very definitely and quite badly	Yes, but not too badly	A little but it doesn't worry me	Not at all
4.	D	I can laugh and see the funny side of things _____	Not at all	Definitely not so much now	Not quite so much now	As much as I always could
5.	A	Worrying thoughts go through my mind _____	A great deal of the time	A lot of the time	From time to time but not too often	Only occasionally
6.	D	I feel cheerful _____	Not at all	Not often	Sometimes	Most of the time
7.	A	I can sit at ease and feel relaxed _____	Not at all	Not often	Usually	Definitely
8.	D	I feel as if I am slowed down _____	Nearly all the time	Very often	Sometimes	Not at all
9.	A	I get a sort of frightened feeling like 'butterflies' in the stomach _____	Very often	Quite often	Occasionally	Not at all
10.	D	I have lost interest in my appearance _____	Definitely	I don't take so much care as I should	I may not take quite as much care	I take just as much care as ever
11.	A	I feel restless as if I have to be on the move _____	Very much indeed	Quite a lot	Not very much	Not at all
12.	D	I look forward with enjoyment to things _____	Hardly at all	Definitely less than I used to	Rather less than I used to	As much as I ever did
13.	A	I get sudden feelings of panic _____	Very often indeed	Quite often	Not very often	Not at all
14.	D	I can enjoy a good book or radio or TV program _____	Very seldom	Not often	Sometimes	Often

Now check that you have answered all questions

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D (8-10) = ……

A (8-10) = …….

C. THE APATHY EVALUATION SCALE (AES)

Rate each item based on an interview of the subject. The interview should begin with a description of the subject's interest, activities and daily routine. Base your ratings on both verbal and non-verbal information. Ratings should be based on the past 4 weeks. For each item ratings should be judged:

No	Questions	Not At All = 1	Slightly = 2	Somewhat = 3	A Lot = 4
1.	S/he is interested in things + C Q				
2.	S/he gets things done during the day + B Q				
3.	Getting things started on his/her own is important to him/her + C SE				
4.	S/he is interested in having new experiences + C Q				
5.	S/he is interested in learning new things + C Q				
6.	S/he puts little efforts into anything -B				
7.	S/he approaches life with intensity + E				
8.	Seeing a job through to the end is important to him/her + C SE				
9.	S/he spends time doing things that interest her/him + B				
10.	Someone has to tell him/her what to do each day - B				
11.	S/he is less concerned about her/his problems than s/he should be - C				
12.	S/he has friends + B Q				
13.	Getting together with friends is important to him/her + C SE				
14.	When something good happens, he/she gets excited + E				
15.	S/he has an accurate understanding of her/his problems + O				
16.	Getting things done during the day is important to him/her				

	+ C SE				
17.	S/he has initiative + O				
18.	S/he has motivation + O				

Note; Items that have positive versus negative syntax are identified by +/-. Type of item: C = cognitive; B = behavior; E = emotional; O = other. The definitions of self-evaluation (SE) and quantifiable (Q) items are discussed in the administration guidelines

D. THE FATIGUE SEVERITY SCALE (FSS)

Please circle the number between 1 and 7 which you feel best fits the following statements. This refers to your usual way of life within the last week. 1 indicates "strongly disagree" and 7 indicates "strongly agree."

No	Questions	1	2	3	4	5	6	7
1.	My motivation is lower when I am fatigued							
2.	Exercise brings on my fatigue							
3.	I am easily fatigued							
4.	Fatigue interferes with my physical functioning							
5.	Fatigue causes frequent problems for me							
6.	My fatigue prevents sustained physical functioning							
7.	Fatigue interferes with carrying out certain duties and responsibilities							
8.	Fatigue is among my three most disabling symptoms							
9.	Fatigue interferes with my work, family or social life							

TOTAL SCORE: _____

E. THE YOUNG MANIA RATING SCALE (YMRS)

No	Item	Score
1.	**Elevated Mood** 0 Absent 1 Mildly or possibly increased on questioning 2 Definite subjective elevation; optimistic, self-confident; cheerful; appropriate to content 3 Elevated; inappropriate to content; humorous 4 Euphoric; inappropriate laughter; singing	_____
2.	**Increased Motor Activity-Energy** 0 Absent 1 Subjectively increased 2 Animated; gestures increased 3 Excessive energy; hyperactive at times; restless (can be calmed) 4 Motor excitement; continuous hyperactivity (cannot be calmed)	_____
3.	**Sexual Interest** 0 Normal; not increased 1 Mildly or possibly increased	

	2 Definite subjective increase on questioning 3 Spontaneous sexual content; elaborates on sexual matters; hypersexual by self-report 4 Overt sexual acts (toward patients, staff, or interviewer)	_____
4.	**Sleep** 0 Reports no decrease in sleep 1 Sleeping less than normal amount by up to one hour 2 Sleeping less than normal by more than one hour 3 Reports decreased need for sleep 4 Denies need for sleep	_____
5.	**Irritability** 0 Absent 2 Subjectively increased 4 Irritable at times during interview; recent episodes of anger or annoyance on ward 6 Frequently irritable during interview; short, curt throughout 8 Hostile, uncooperative; interview impossible	_____
6.	**Speech (Rate and Amount)** 0 No increase 2 Feels talkative 4 Increased rate or amount at times, verbose at times 6 Push; consistently increased rate and amount; difficult to interrupt 8 Pressured; uninterruptible, continuous speech	_____
7.	**Language-Thought Disorder** 0 Absent 1 Circumstantial; mild distractibility; quick thoughts 2 Distractible, loses goal of thought; changes topics frequently; racing thoughts 3 Flight of ideas; tangentiality; difficult to follow; rhyming, echolalia 4 Incoherent; communication impossible	_____
8.	**Content** 0 Normal 2 Questionable plans, new interests 4 Special project(s); hyper-religious 6 Grandiose or paranoid ideas; ideas of reference 8 Delusions; hallucinations	_____
9.	**Disruptive-Aggressive Behavior** 0 Absent, cooperative 2 Sarcastic; loud at times, guarded 4 Demanding; threats on ward 6 Threatens interviewer; shouting; interview difficult 8 Assaultive; destructive; interview impossible	_____
10.	**Appearance** 0 Appropriate dress and grooming 1 Minimally unkempt 2 Poorly groomed; moderately disheveled; overdressed 3 Disheveled; partly clothed; garish make-up 4 Completely unkempt; decorated; bizarre garb	_____
11.	**Insight** 0 Present; admits illness; agrees with need for treatment 1 Possibly ill	

	2 Admits behavior change, but denies illness 3 Admits possible change in behavior, but denies illness 4 Denies any behavior change	_____

PSYCHOSIS: _____

SECTION 7: EVALUATION OF FUNCTIONAL OUTCOME

A. THE BARTHEL INDEX (BI)

No	ACTIVITY/ SCORE	0	5	10	15
1.	FEEDING _____	Unable	Needs help cutting, spreading butter etc., or requires modified diet	Independent	
2.	BATHING _____	Dependent	Independent (or in shower)		
3.	GROOMING _____	Needs help with personal care	Independent face/hair/teeth/shaving (implements provided)		
4.	DRESSING _____	Dependent	Needs help but can do about half unaided	Independent (including buttons, zips, laces etc.)	
5.	BOWELS _____	Incontinent (or needs to be given enemas)	Occasional accident	Continent	
6.	BLADDER _____	Incontinent or catheterized and unable to manage alone	Occasional accident	Continent	
7.	TOILET USE _____	Dependent	Needs some help but can do something alone	Independent (on and off, dressing, wiping)	
8.	TRANSFERS (BED TO CHAIR AND BACK) _____	Unable, no sitting balance	Major help (one or two people, physical) can sit	Minor help (verbal or physical)	Independent
9.	MOBILITY (ON LEVEL SURFACES) _____	Immobile or < 50 yards	Wheelchair independent, including comers, > 50 yards	Walks with help of one person, (verbal or physical) > 50 yards	Independent (but may use any aid; for example, stick) > 50 yards

10.	STAIRS _____	Unable	Needs help (verbal, physical, carrying aid)	Independent	
	TOTAL SCORE (/100)				_____

B. THE MODIFIED RANKIN SCALE (MRS)

GRADE	SYMPTOMS
0	None
1	No significant disability despite symptoms; able to carry out all usual duties and activities
2	Slight disability; unable to carry out all previous activities but able to look after own affairs without assistance
3	Moderate disability; requiring some help but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance, unable to attend to needs without assistance
5	Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6	Dead

C. **SCORE:** _____

0– 2: Good functional outcome

3 – 6: Poor functional outcome

APPENDIX II : QUESTIONNAIRE (FRENCH)

QUESTIONNAIRE

SUJET : Troubles neurologiques et psychiatriques post-AVC : prévalence et effets sur la récupération fonctionnelle des survivants d'AVC à Yaoundé

Enquêteur : THOM NKUIDJEU CLAUDE

Date:..... Contact:.....

Code:.....

SECTION 1 : DONNÉES SOCIODÉMOGRAPHIQUES

Non/Q	VARIABLES	RÉPONSES POSSIBLES	RÉPONDRE	RÉPONSE EXACTE
1.	Date de naissance			_____
2.	Sexe	3. Masculin 4. Feminin	3. ___ 4. ___	
3.	Adresse			
4.	Niveau d'éducation	1. Aucun 2. Primaire 3. Secondaire 4. Tertiaire	1. ___ 2. ___ 3. ___ 4. ___	
5.	Profession	1. Aucun 2. Étudiant 3. Ménagère 4. Commerçant 5. Cultivateur 6. Fonctionnaire 7. Secteur privé 8. Retraité	1. ___ 2. ___ 3. ___ 4. ___ 5. ___ 6. ___ 7. ___ 8. ___	
6.	Statut marital	1. Célibataire 2. Marié 3. Divorcé 4. Veuf(ve)	1. ___ 2. ___ 3. ___ 4. ___	
7.	Religion	1. Aucun 2. Chrétien 3. Musulman 4. Autre	1.___ 2___ 3. ___ 4. ___	
8.	Maniabilité	1. Gauche 2. Droite	1. ___ 2. ___	

SECTION 2 : ANTÉCÉDENTS

No/Q	VARIABLES	RÉPONSES POSSIBLES	RÉPONDRE	RÉPONSE EXACTE
9	Hypertension	1. Oui	1.___	

		2Non	2___	
10	Diabète	1.Oui 2Non	1___ 2___	
11	Dyslipidémie	1. Oui 2.Non	1___ 2___	
12	Drépanocytose	1. Oui 2. Non	1___ 2___	
13	VIH	1. Oui 2. Non	1___ 2___	
14	Maladie psychiatrique	1. Oui 2. Non	1___ 2___	
15.	Maladie cardiaque	1. Oui 2. Non	1___ 2___	
16	AVC	1. Oui 2. Non	1___ 2___	Si oui Nombre:_____
17	Maladie génétique	1. Oui 2. Non	1___ 2___	
18	Tabac	1. Oui 2. Non	1___ 2___	
19	Alcool	1. Oui 2. Non	1___ 2___	
20	Sédentarité	1. Oui 2. Non	1___ 2___	

SECTION 3 : CARACTÉRISTIQUES CLINIQUES

Non/Q	VARIABLES	RÉPONSES POSSIBLES	RÉPONDRE	RÉPONSE EXACTE
21	Date d'apparition des symptômes			
22	Tension artérielle	1. Normal 2. HTN Grade 1 3. HTN Grade 2 4. HTN Grade 3	1. ___ 2. ___ 3. ___ 4. ___	
23	FC (battements par minute)			
24	FR (cycles par minute)			
25	Poids (kg) / hauteur (m)			
26	IMC			
27	AC(cm)			
28.	Score de Glasgow			
29	Fonctions supérieures	1. Intact 2. Altéré	1. ___ 2. ___	
	Si la réponse ci-dessus est Modifié, spécifiez quelle fonction	1. Aphasie 2. Amnésie 3. Apraxie 4. Agnosie	1. ___ 2. ___ 3. ___ 4. ___	
30	Nerfs crâniens	1. Intact 2. Alteré	1. ___ 2. ___	

	Si la réponse ci-dessus est Altéré, nommez quel nerf est altéré et sa manifestation			
31	Force musculaire des membres supérieurs	1. Gauche 2. Droite	1. ____ 2. ____	
32	Force musculaire des membres inférieurs	1. Gauche 2. Droite	1. ____ 2. ____	
33	Réflexes du membre supérieur gauche	1. Normal 2. Hyperréflexie 3. Hyporéflexie	1. ___ 2. ___ 3. ___	
34	Réflexes du membre supérieur droit	1. Normal 2. Hyperréflexie 3. Hyporéflexie	1. ___ 2. ___ 3. ___	
35	Réflexes du membre inférieur gauche	1. Normal 2. Hyperréflexie 3. Hyporéflexie	1. ___ 2. ___ 3. ___	
36	Réflexes du membre inférieur droit	1. Normal 2. Hyperréflexie 3. Hyporéflexie	1. ___ 2. ___ 3. ___	
37	Panneau Babinski gauche	1. Négatif 2. Positif 3. Indifférent	1. ___ 2. ___ 3. ___	
38	Panneau Babinski droit	1. Négatif 2. Positif 3. Indifférent	1. ___ 2. ___ 3. ___	
39	Score NIHSS (initial ; au début de l'AVC)	1. (0-5) Accident vasculaire cérébral léger 2. (6-10) Accident vasculaire cérébral modéré 3. (11-20) Accident vasculaire cérébral modéré à grave 4. (> 20) Accident vasculaire cérébral très grave	1. ___ 2. ___ 3. ___ 4. ___	
40.	Score NIHSS (actuel)	1. (0-4) Accident vasculaire cérébral léger 2. (6-10) Accident vasculaire cérébral modéré 3. (11-20) Accident vasculaire	1. ___ 2. ___ 3. ___ 4. ___	

		cérébral modéré à grave 4. (> 20) Accident vasculaire cérébral très grave		

L'ÉCHELLE DE NIHSS

Non	Catégorie	Description	Score
1a	Niveau de conscience (LOC)	Alerte Somnolent Stuporeux Coma	0 1 2 3
1b	Questions sur la lettre de crédit (mois, âge)	Répond correctement aux deux Réponse 1 correcte Incorrect sur les deux	0 1 2
1c	Commandes LOC (ouvrir et fermer les yeux, saisir et relâcher la main non parétique)	Obéit correctement aux deux Obéit correctement à 1 Incorrect sur les deux	0 1 2
2	Meilleur regard (suivre le doigt)	Normal Paralysie partielle du regard Déviation forcée	0 1 2
3	Meilleur visuel (champs visuels)	Aucune perte visuelle Hémianopsie partielle Hémianopsie complète Hémianopsie bilatérale	0 1 2 3
4	Paralysie faciale (montrer les dents, lever les sourcils, fermer les yeux)	Normal Mineur Partiel Complet	0 1 2 3
5	Bras moteur gauche* (lever 90, maintenir 10 secondes) (de préférence paume vers le haut)	Pas de dérive Dériver Ne résiste pas à la gravité Pas d'effort contre la gravité Pas de mouvement	0 1 2 3 4
6	Bras moteur droit* (lever 90, maintenir 10 secondes) (de préférence paume vers le haut)	Pas de dérive Dériver Ne résiste pas à la gravité Pas d'effort contre la gravité Pas de mouvement	0 1 2 3 4
7	Jambe motrice gauche* (lever 30, maintenir 5 secondes)	Pas de dérive Dériver Ne résiste pas à la gravité Pas d'effort contre la gravité Pas de mouvement	0 1 2 3 4
8	Jambe motrice droite* (relancer 30, maintenir 5 secondes)	Pas de dérive Dériver Ne résiste pas à la gravité Pas d'effort contre la gravité Pas de mouvement	0 1 2 3 4

9	Ataxie des membres (doigt-nez, talon-tibia)	Absent Présent dans 1 branche Présent en 2 branches	0 1 2
10	Sensoriel (piqûre d'épingle au visage, au bras, à la jambe)	Normal Perte partielle Perte grave	0 1 2
11	Extinction/négligence (double test simultané)	Pas de négligence Négligence partielle Négligence totale	0 1 2
12	Dysarthrie (clarté de la parole à « maman, baseball, huckleberry, tip-top, fifty-fifty »)	Articulation normale Dysarthrie légère à modérée Presque inintelligible ou pire	0 1 2
13	Meilleure langue** (nommer les éléments, décrire les images)	Pas d'aphasie Aphasie légère à modérée Aphasie sévère Muet	0 1 2 3
	TOTAL		0-42

- Pour les membres amputés, fusionnés, etc., notez 9 et expliquez
- Pour l'intubation ou d'autres obstacles physiques à la parole, notez 9 et expliquez. N'ajoutez pas 9 au score total

SECTION 4 : CARACTÉRISTIQUES PARACLINIQUES

Non/Q	VARIABLES	RÉPONSES POSSIBLES	RÉPONDRE	RÉPONSE EXACTE
41	Type d'AVC	1. Ischémique 2. Hémorragique	1. ___ 2. ___	
42	Hémisphère	1. Gauche 2. Droite 3. Bilatéral	1. ___ 2. ___ 3. ___	
43	Localisation	1. Supratentorial 2. Infratentorial	1. ___ 2. ___	
	Si la réponse ci-dessus est supratentorielle, choisissez l'emplacement exact	1. Lobe frontal 2. Lobe pariétal 3. Lobe temporal 4. Lobe occipital 5. Capsule interne 6. Thalamus	1. ___ 2. ___ 3. ___ 4. ___ 5. ___ 6. ___	
	Si la réponse à 43 ci-dessus est infratentorielle, choisissez l'emplacement exact	1. Cervelet 2. Tronc cérébral 3.	1. ___ 2. ___ 3. ___	
44	Artere atteinte	1. ACA Superficiel 2. ACA Profond 3. MCA Superficiel 4. MCA Profond 5. PCA Superficiel	1. ___ 2. ___ 3. ___ 4. ___ 5. ___	

		6. ACP Profond	6. ___	
45	Glycémie à jeun (N < 1,26 g/l) / HbA1C (N<7 mmol/l)	1. Bas 2. Normal 3. Haut	1. ___ 2. ___ 3. ___	
46	Taux de LDL (N< 100mg/l ou <1g/l)	1. Bas 2. Normal 3. Haut	1. ___ 2. ___ 3. ___	
47	Taux de HDL (N < 40mg/dl hommes N < 55mg/dl femmes)	1. Bas 2. Normal 3. Haut	1. ___ 2. ___ 3. ___	
48	Taux de triglycérides (N < 150mg/dl)	1. Bas 2. Normal 3. Haut	1. ___ 2. ___ 3. ___	
49	Urée (N :)	1. Normal 2. Haut	1. ___ 2. ___	
50	Créatinine sérique (N :)	1. Normal 2. Haut	1. ___ 2. ___	
51	Échographie cardiaque	1. LVH 2. AF 3. Autre	1. ___ 2. ___ 3. ___	
52	Étiologie de l'AVC	1. Athérosclérose des gros vaisseaux 2. Cardioembolie 3. Maladie des petits vaisseaux 4. Indéterminé 5. Autres étiologies	1. ___ 2. ___ 3. ___ 4. ___ 5. ___	

SECTION 5 : TRAITEMENT

Non/Q	VARIABLES	RÉPONSES POSSIBLES	RÉPONDRE	RÉPONSE EXACTE
53	Antihypertenseur	1. Oui 2. Non	1. ___ 2. ___	
	Si oui, choisissez la classe thérapeutique	1. ICC 2. L'IEC 3. Diurétiques 4. Bêta-bloquants 5. ARA II	1. ___ 2. ___ 3. ___ 4. ___ 5. ___	
54	Antidiabétiques	1. Oui 2. Non	1. ___ 2. ___	
	Si oui, choisissez la classe thérapeutique	1. Insuline 2. OAD 3. Autre	1. ___ 2. ___ 3. ___	Autre: _____
55	Anti-aggregant plaquettaire	1.Oui 2Non	1. ___ 2. ___	
56	Anti-vitamine K	1. Oui 2. Non	1. ___ 2. ___	

57	Statines	1. Oui 2. Non	1. ___ 2. ___	
58	Antidépresseur	1. Oui 2. Non	1. ___ 2. ___	
59	Antiépileptique	1. Oui 2. Non	1___ 2___	
60	Laxatif osmotique	1. Oui 2. Non	1___ 2___	
61	Hypnotique ou sédatif	1. Oui 2. Non	1___ 2___	
62	Rééducation	1. Oui 2. Non	1___ 2___	
	Si oui, choisissez la ou les thérapies	1. Physiothérapie 2. Orthophonie	1___ 2___	Nombre de séances : _____
63	Psychothérapie	1. Oui 2. Non	1____ 2____	
64	Personnel de rééducation	1. Personnel qualifié 2. Membre de la famille	1. ___ 2. ___	

SECTION 6 : ÉVALUATION DES TROUBLES PSYCHIATRIQUES

A. THE PATIENT HEALTH QUESTIONNAIRE-9 (PHQ-9)

Au cours des 2 dernières semaines, à quelle fréquence avez-vous été dérangé par l'un des problèmes suivants ?

No	**Symptôme**	**Pas du tout**	**Plusieurs jours**	**Plus de la moitié des jours**	**Presque tous les jours**
1	Peu d'intérêt ou de plaisir à faire les choses	0	1	2	3
2	Se sentir déprimé, déprimé ou désespéré	0	1	2	3
3	Difficulté à s'endormir ou à rester endormi ou à dormir trop	0	1	2	3
4	Se sentir fatigué ou avoir peu d'énergie	0	1	2	3
5	Manque d'appétit ou suralimentation	0	1	2	3
6	Se sentir mal dans sa peau - ou avoir échoué ou s'être laissé tomber ou avoir laissé tomber sa famille	0	1	2	3
7	Difficulté à se concentrer sur des choses comme lire un livre ou regarder la télévision	0	1	2	3
8	Bouger ou parler si lentement que d'autres personnes auraient pu le remarquer ou le	0	1	2	3

	contraire - être si agité ou agité que vous avez bougé beaucoup plus que d'habitude				
9	Penser que vous seriez mieux mort ou de vous blesser d'une manière ou d'une autre	0	1	2	3
	SOMME				
	NOTE TOTALE				

Si vous avez coché des problèmes, à quel point ces problèmes vous ont-ils rendu difficile de faire votre travail, de vous occuper des choses à la maison ou de vous entendre avec d'autres personnes ?

Pas difficile du tout (__) Plutôt difficile (__) Très difficile (__) Extrêmement difficile (__)

B. THE HOSPITAL ANXIETY AND DEPRESSION SCALE (HADS)

No	**A/D**	**Symptôme / Score**	**3**	**2**	**1**	**0**
1.	Un	Je me sens tendu ou « énervé » ____	La plupart du temps	La plupart du temps	De temps en temps occasionnellement	Pas du tout
2.	D	J'aime toujours les choses que j'aimais ____	Presque pas du tout	Seulement un peu	Pas tant que ça	Certainement autant
3.	Un	J'ai une sorte de sentiment de peur comme si quelque chose d'horrible était sur le point de se produire ____	Très certainement et assez mal	Oui, mais pas trop mal	Un peu mais ça ne m'inquiète pas	Pas du tout
4.	D	Je peux rire et voir le côté drôle des choses _____	Pas du tout	Certainement pas tellement maintenant	Pas tout à fait maintenant	Autant que je le pouvais toujours
5.	Un	Des pensées inquiétantes me traversent l'esprit ____	La plupart du temps	La plupart du temps	De temps en temps mais pas trop souvent	Seulement occasionnellement

6.	D	Je me sens joyeux _____	Pas du tout	Pas souvent	Parfois	La plupart du temps
7.	Un	Je peux m'asseoir à l'aise et me sentir détendu _____	Pas du tout	Pas souvent	Habituellement	Sans faute
8.	D	J'ai l'impression d'être ralenti _____	Presque tout le temps	Très souvent	Parfois	Pas du tout
9.	Un	J'ai une sorte de sensation de peur comme des « papillons » dans l'estomac _____	Très souvent	Assez souvent	Parfois	Pas du tout
10.	D	J'ai perdu tout intérêt pour mon apparence _____	Sans faute	Je ne prends pas autant de précautions que je le devrais	Je ne prendrai peut-être pas autant de soin	Je prends autant de soin que jamais
11.	Un	Je me sens agité comme si je devais être en mouvement _____	Tout à fait en effet	Beaucoup	Pas grand-chose	Pas du tout
12.	D	J'attends les choses avec plaisir _____	Presque pas du tout	Certainement moins qu'avant	Un peu moins qu'avant	Autant que je l'ai jamais fait
13.	Un	J'ai des sentiments soudains de panique _____	Très souvent en effet	Assez souvent	Pas très souvent	Pas du tout
14.	D	Je peux profiter d'un bon livre ou d'une émission de radio ou de	Très rarement	Pas souvent	Parfois	Souvent

		télévision _____				

Vérifiez maintenant que vous avez répondu à toutes les questions

À USAGE HOSPITALIER UNIQUEMENT

D (8-10) = ...

A (8-10) = ...

C. THE APATHY EVALUATION SCALE (AES)

Évaluez chaque élément en fonction d'une interview du sujet. L'entretien doit commencer par une description de l'intérêt, des activités et de la routine quotidienne du sujet. Basez vos évaluations sur des informations verbales et non verbales. Les évaluations doivent être basées sur les 4 dernières semaines. Pour chaque article, les notes doivent être jugées :

No	Question	Pas du tout = 1	Légèrement = 2	Unpeu = 3	Beaucoup = 4
1.	Il s'intéresse aux choses + C Q				
2.	Il fait avancer les choses pendant la journée + B Q				
3.	Démarrer les choses par lui-même est important pour lui + C SE				
4.	Il est intéressé à vivre de nouvelles expériences + C Q				
5.	Il est intéressé à apprendre de nouvelles choses + C Q				
6.	Il fait peu d'efforts dans quoi que ce soit -B				
7.	Il aborde la vie avec intensité + E				
8.	Aller jusqu'au bout d'un travail est important pour lui + C SE				
9.	Il passe du temps à faire des choses qui l'intéressent + B				
10.	Quelqu'un doit lui dire quoi faire chaque jour - B				
11.	Il est moins préoccupé par ses problèmes qu'il ne devrait l'être - C				
12.	Il a des amis + B Q				

13.	Se réunir avec des amis est important pour lui + C SE				
14.	Quand quelque chose de bien se produit, il est excité + E				
15.	Il a une compréhension précise de ses problèmes + O				
16.	Faire avancer les choses pendant la journée est important pour lui + C SE				
17.	Il a de l'initiative + O				
18.	Il a de la motivation + O				

Note; Les éléments qui ont une syntaxe positive ou négative sont identifiés par +/-. Type d'élément : C = cognitif ; B = comportement ; E = émotionnel ; O = autre. Les définitions d'éléments d'auto-évaluation (ET) et quantifiables (Q) sont discutées dans les directives d'administration

D. THE FATIGUE SEVERITY SCALE (FSS)

Veuillez encercler le chiffre entre 1 et 7 qui, selon vous, correspond le mieux aux énoncés suivants. Il s'agit de votre mode de vie habituel au cours de la dernière semaine. 1 indique « fortement en désaccord » et 7 indique « tout à fait d'accord ».

No	Question	1	2	3	4	5	6	7
1.	Ma motivation est plus faible lorsque je suis fatigué							
2.	L'exercice provoque ma fatigue							
3.	Je suis facilement fatigué							
4.	La fatigue interfère avec mon fonctionnement physique							
5.	La fatigue me cause des problèmes fréquents							
6.	Ma fatigue empêche un fonctionnement physique soutenu							
7.	La fatigue nuit à l'exécution de certaines tâches et responsabilités							
8.	La fatigue est l'un de mes trois symptômes les plus invalidants							
9.	La fatigue interfère avec mon travail, ma famille ou ma vie sociale							

NOTE TOTALE : _____

E. THE YOUNG MANIA RATING SCALE (YMRS)

Non	Article	Score
1.	**Humeur élevée** 0 Absent 1 Légèrement ou peut-être augmenté au interrogatoire 2 Élévation subjective définie ; optimiste, sûr de lui ; gai; adapté au contenu 3 Élevé ; inapproprié au contenu ; humoristique 4 Euphorique ; rire inapproprié ; chant	_____

2.	**Augmentation de l'activité motrice-énergie** 0 Absent 1 Augmentation subjective 2 animés ; les gestes ont augmenté 3 Énergie excessive ; hyperactif par moments ; agité (peut être calmé) 4 Excitation motrice ; Hyperactivité continue (ne peut pas être calmée)	_____
3.	**Intérêt sexuel** 0 Normal ; pas augmenté 1 Légèrement ou peut-être augmenté 2 Augmentation subjective certaine lors de l'interrogatoire 3 Contenu sexuel spontané ; développe sur les questions sexuelles ; Hypersexuel par auto-déclaration 4 Actes sexuels manifestes (envers les patients, le personnel ou l'intervieweur)	_____
4.	**Dormir** 0 Ne signale aucune diminution du sommeil 1 Dormir moins que la normale jusqu'à une heure 2 Dormir moins que la normale de plus d'une heure 3 signalent une diminution du besoin de sommeil 4 Nie le besoin de sommeil	_____
5.	**Irritabilité** 0 Absent 2 Augmentation subjective 4 Irritable parfois pendant l'entrevue ; Épisodes récents de colère ou d'agacement dans le service 6 Irritable fréquemment pendant l'entrevue ; court, bref tout au long 8 Hostile, peu coopératif ; interview impossible	_____
6.	**Discours (débit et montant)** 0 Aucune augmentation 2 Se sent bavard 4 Taux ou montant augmenté parfois, parfois détaillé 6 Pousser ; augmentation constante du taux et du montant ; difficile à interrompre 8 Sous pression ; Parole ininterrompue et continue	_____
7.	**Trouble du langage et de la pensée** 0 Absent 1 Circonstanciel ; légère distraction ; Réflexions rapides 2 Distrait, perd le but de la pensée ; change fréquemment de sujet ; Pensées qui défilent 3 Fuite des idées ; tangentialité ; difficile à suivre ; rimes, écholalie 4 Incohérent ; communication impossible	_____
8.	**Contenu** 0 Normal 2 Plans douteux, nouveaux intérêts 4 Projet(s) spécial(s) ; hyper-religieux 6 Idées grandioses ou paranoïaques ; Idées de référence 8 Délires ; Hallucinations	_____
9.	**Comportement perturbateur-agressif** 0 Absent, coopératif 2 Sarcastique ; bruyant parfois, prudent 4 Exigeant ; Menaces sur le service	_____

	6 Menace l'intervieweur ; Cris; entretien difficile 8 Agressif ; destructeur; interview impossible	
10.	**Apparence** 0 Tenue vestimentaire et toilette appropriées 1 Minimalement négligé 2 Mal entretenu ; modérément échevelé ; trop habillé 3 échevelé ; partiellement vêtu ; maquillage criard 4 Complètement négligé ; décoré; Tenue bizarre	_____
11.	**Perspicacité** 0 présent ; admet une maladie ; est d'accord avec la nécessité d'un traitement 1 Possiblement malade 2 Admets un changement de comportement, mais nie la maladie 3 Admets un éventuel changement de comportement, mais nie la maladie 4 Nie tout changement de comportement	_____

PSYCHOSE:_____

SECTION 7 : ÉVALUATION DU RÉSULTAT FONCTIONNEL

F. THE BARTHEL INDEX (BI)

Non	**ACTIVITÉ/ SCORE**	**0**	**5**	**10**	**15**
1.	ALIMENTATION _____	Incapable	A besoin d'aide pour couper, étaler du beurre, etc., ou nécessite un régime alimentaire modifié	Indépendant	
2.	BAIGNADE _____	Dépendant	Indépendant (ou sous la douche)		
3.	PANSAGE _____	A besoin d'aide pour les soins personnels	Visage/cheveux/dents/rasage indépendants (outils fournis)		
4.	PANSEMENT _____	Dépendant	A besoin d'aide mais peut en faire environ la moitié sans aide	Indépendant (y compris boutons, fermetures éclair, lacets, etc.)	
5.	INTESTINS _____	Incontinent (ou doit recevoir des lavements)	Accident occasionnel	Continent	
6.	VESSIE _____	Incontinent ou cathétérisé et incapable de se	Accident occasionnel	Continent	

		débrouiller seul			
7.	UTILISATION DES TOILETTES _____	Dépendant	A besoin d'aide, mais peut faire quelque chose seul	Indépendant (marche et arrêt, habillage, essuyage)	
8.	TRANSFERTS (LIT À CHAISE ET DOSSIER) _____	Incapable, pas d'équilibre assis	Aide majeure (une ou deux personnes, physique) peut s'asseoir	Aide mineure (verbale ou physique)	Indépendant
9.	MOBILITÉ (SUR DES SURFACES PLANES) _____	Immobile ou < 50 mètres	Fauteuil roulant indépendant, y compris les arrivants, > 50 mètres	Promenades avec l'aide d'une personne, (verbale ou physique) > 50 mètres	Indépendant (mais peut utiliser n'importe quelle aide, par exemple, un bâton) > 50 verges
10.	ESCALIER _____	Incapable	A besoin d'aide (verbale, physique, aide au portage)	Indépendant	
	NOTE TOTALE (/100)				_____

G. THE MODIFIED RANKIN SCALE (MRS)

GRADE	**SYMPTÔMES**
0	Aucun
1	Pas d'invalidité significative malgré les symptômes ; capable d'effectuer toutes les tâches et activités habituelles
2	Handicap léger ; incapable de mener à bien toutes les activités antérieures mais capable de s'occuper de ses propres affaires sans aide
3	Handicap modéré ; nécessitant de l'aide, mais capable de marcher sans aide
4	Invalidité modérément sévère ; incapable de marcher sans aide, incapable de répondre aux besoins sans aide
5	Invalidité grave ; alitée, incontinent et nécessitant des soins infirmiers et une attention constants
6	Mort

H. **RÉSULTAT : _____**

0 à 2 : Bon résultat fonctionnel

3 – 6 : Mauvais résultat fonctionnel

APPENDIX III: INFORMED CONSENT FORM

INFORMED CONSENT FORM

TITLE: **Neurological And Psychiatric Disorders Post-Stroke: Prevalence And Effects On Functional Recovery Of Stroke Survivors In Yaounde**

I Mr./Mrs./Ms. _____, the undersigned, acknowledge that I have been informed and fully briefed by the final year medical student, THOM Claude, on the study entitled "Neurological And Psychiatric Disorders Post-Stroke: Prevalence And Effects On Functional Recovery Of Stroke Survivors In Yaounde" in view of her M.D. Thesis and have accepted to participate in the study. She clearly stated I was free to accept or deny the proposal. I have understood the aim of the study, the possible constraints and risks.

I accept that all data collected in relation to me will be subject to a code of strict confidentiality. Only the research personnel and eventually a health authority representative will have access to my data.

My participation can be interrupted any time if the principal investigator deems it necessary or if I wish without any prejudice. At any time, I can ask supplementary information or make corrections on my data.

I hereby accept to participate in the study under the said conditions

Read and approved; YES NO

Yaounde, ____/____/_____

Investigator's Signature Participant's Signature

APPENDIX IV: FICHE DE CONSENTEMENT ECLAIRE

FICHE DE CONSENTEMENT ECLAIRE

TITRE : **Troubles Neurologiques Et Psychiatriques Post-AVC : Prévalence Et Effet Sur La Récupération Fonctionnelle Des Patients A Yaoundé**

Je soussigné M/Mme/Mlle _____, reconnait avoir été pleinement informée et briefée par l'étudiante en 7e année de médecine, THOM Claude sur l'étude intitulée «Troubles Neurologiques Et Psychiatriques Post-AVC : Prévalence Et Effet Sur La Récupération Fonctionnelle Des Survivants A Yaoundé» pour sa thèse de fin de formation et avoir accepté de participer dans cette étude. Elle a clairement mentionnée que je suis libre d'accepter ou de refuser sa proposition. J'ai compris le but de l'étude ainsi que les possibles contraintes et risques.

J'accepte que toute donnée collectée en relation à moi sera soumis à un code de confidentialité stricte. Seul le personnel de recherche et éventuellement une autorité représentative de la sante pourront avoir accès à mes données.

Ma participation pourrait être interrompue a n'importe quel moment si l'investigateur principal le trouve nécessaire ou si cela est mon souhait. Je peux demander des informations supplémentaires ou faire des corrections sur mes données à tout moment.

J'accepte par la présente de participer à cette étude sous les conditions suscitées

Lu et approuvé : OUI NON

Yaoundé, le ___/____/____

Signature de l'investigateur Signature du Participant

APPENDIX V: ETHICAL CLEARANCE

UNIVERSITÉ DE YAOUNDÉ I

FACULTÉ DE MÉDECINE ET DES SCIENCES BIOMÉDICALES

COMITÉ INSTITUTIONNEL D'ÉTHIQUE DE LA RECHERCHE
Tel/ fax : 22 31-05-86 22 311224
Email: decanatfmsb@hotmail.com

THE UNIVERSITY OF YAOUNDE I

FACULTY OF MEDICINE AND BIOMEDICAL SCIENCES

INSTITUTIONAL ETHICAL REVIEW BOARD

Ref. : N° 1134/UY1/FMSB/VDRC/DAASR/CSD

CLAIRANCE ÉTHIQUE

19 JUIL 2024

Le COMITÉ INSTITUTIONNEL D'ÉTHIQUE DE LA RECHERCHE (CIER) de la FMSB a examiné

La demande de la clairance éthique soumise par :

M.Mme : THOM NKUIDJEU CLAUDE **Matricule: 17M066**

Travaillant sous la direction de :
- **Pr NJAMNSHI Alfred**
- **Dr NTONE ENYIME Félicien**
- **Dr NGARKA Leonard**

Concernant le projet de recherche intitulé : **Neurological and psychiatric disorders post stroke: prevalence and effects on functional recovery among stroke survivors in Yaoundé**

Les principales observations sont les suivantes

Evaluation scientifique	
Evaluation de la convenance institutionnelle/valeur sociale	
Equilibre des risques et des bénéfices	
Respect du consentement libre et éclairé	
Respect de la vie privée et des renseignements personnels (confidentialité) :	
Respect de la justice dans le choix des sujets	
Respect des personnes vulnérables :	
Réduction des inconvénients/optimalisation des avantages	
Gestion des compensations financières des sujets	
Gestion des conflits d'intérêt impliquant le chercheur	

Pour toutes ces raisons, le **CIER** émet un avis **favorable** sous réserve des modifications recommandées dans la grille d'évaluation scientifique.

L'équipe de recherche est responsable du respect du protocole approuvé et ne devra pas y apporter d'amendement sans avis favorable du **CIER**. Elle devra collaborer avec le **CIER** lorsque nécessaire, pour le suivi de la mise en œuvre dudit protocole.

La clairance éthique peut être retirée en cas de non - respect de la réglementation ou des recommandations sus évoquées.

En foi de quoi la présente clairance éthique est délivrée pour servir et valoir ce que de droit

LE PRESIDENT DU COMITE ETHIQUE

UNIVERSITE DE YAOUNDE I

Mme Abena Ondoa
Mme Obama Marie Thérèse

APPENDIX VI: RESEARCH AUTHORIZATION

REPUBLIQUE DU CAMEROUN
Paix-Travail-Patrie
MINISTERE DE LA SANTE PUBLIQUE
SECRETARIAT GENERAL
DIRECTION DE L' HOPITAL CENTRAL DE YAOUNDE
SECRETARIAT MEDICAL

N°210/24 / AP/MINSANTE/SG/DHCY/CM/SM

REPUBLIC OF CAMEROUN
Peace-Work-Fatherland
MINISTRY OF PUBLIC HEALTH
GENERAL SECRETARY
DIRECTORATE OF CENTRAL HOSPITAL OF YAOUNDE
MEDICAL SECRETARY

Yaoundé, le 14 FEV 2024

ACCORD DE PRINCIPE

Je soussigné **Professeur FOUDA Pierre Joseph**, Directeur de l'Hôpital Central de Yaoundé, marque mon Accord de Principe à Monsieur **THOM NKUIDJEU Claude**, étudiant de 7ème année de Médecine générale à la Faculté de Médecine et des Sciences Biomédicales de l'Université de Yaoundé I, sous le thème « NEUROLOGICAL AND PSYCHIATRIC DISORDERS POST-STROKE : PREVALENCE AND EFFECTS ON FUNCTIONAL RECOVERY AMONG STROKE SURVIVORS IN YAOUNDE » dans le service de Neurologie à l'Hôpital Central de Yaoundé, sous la codirection du docteur NGARKA Leonard.

Pour Le Directeur et par ordre
Le Conseiller Médical

Le Conseiller Médical
Pr. Agr. Pierre Angolo Fogo

Ampliations :

- Conseiller Médical ;
- Chef service concerné ;
- Intéressé ;
- Chrono/Archives.